A RANDOMIZED CONTROLLED CLINICAL TRIAL TO ASSESS THE ROLE OF MAHATIKTAKAM KWATHAM TABLET IN AMLAPITTA W.S.R TO FUNCTIONAL DYSPEPSIA

A thesis submitted to

TILAK MAHARASHTRA VIDYAPEETH

PUNE

For the Degree of

DOCTOR OF PHILOSOPHY (PhD)

in

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Under the Board of Ayurvedic Studies

Submitted By

JITHESH M.

Under the Guidance of

Dr. Renuka R Gayal

October 2015

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Subject	:	Kayachikitsa
Under the Faculty of	:	Ayurveda
Name of the Candidate	:	Jithesh M
Name of the guide	:	Dr Renuka R Gayal
Department	:	Ayurveda
Month and Year	:	October 2015

TILAK MAHARASHTRA VIDYAPEETH
Pune, Maharashtra, India

Certificate

This is to certify that the thesis entitled **"A RANDOMIZED CONTROLLED CLINICAL TRIAL TO ASSESS THE ROLE OF MAHATIKTAKAM KWATHAM TABLET IN AMLAPITTA W.S.R TO FUNCTIONAL DYSPEPSIA"** which is being submitted herewith for the award of the Degree of Vidya vachaspathi (PhD) in Kayachikitsa under the board of Ayurvedic studies of Tilak Maharashtra Vidyapeeth, Pune is the result of original research work completed by **Shri JITHESH M** under my supervision and guidance. To the best of my knowledge and belief the work incorporated in this thesis has not formed the basis for the award of any degree or similar title of this or any other university or examining body upon him.

Dr Renuka R Gayal
Research Guide

Pune

October 2015

TILAK MAHARASHTRA VIDYAPEETH
Pune, Maharashtra, India

Declaration

I hereby declare that the thesis entitled **"A Randomized Controlled Clinical trial to assess the role Of Mahatiktakam Kwatham tablet in Amlapitta W.S.R to Functional Dyspepsia"** completed and written by me has not previously been formed as the basis for the award of any Degree or other similar title upon me of this or any other Vidyapeeth or examining body. I understand that if my PhD Thesis or part of it is found duplicate at any point of time my research degree will be withdrawn.

JITHESH M

Pune

October 2015

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List of Abbreviations

FD	-	Functional Dyspepsia
WSR	-	With special Reference to
GI	-	Gastrointestinal
FGID	-	Functional Gastrointestinal Disorders
IBS	-	Irritable Bowel Syndrome
PDS	-	Postprandial Distress Syndrome
EPS	-	Epigastric Pain Syndrome
H Pylori	-	Helicobacter pylori
PPI	-	Proton-Pump Inhibitor
SSRI	-	Selective Serotonin-Reuptake Inhibitors
UD	-	Uninvestigated Dyspepsia
NUD	-	Non Ulcer Dyspepsia
NSAID	-	Non- Steroidal Anti-inflammatory drugs
PUD	-	Peptic Ulcer Disease
EGG	-	Electro gastrography
PET	-	Positron Emission Tomography
CCK	-	Cholecystokinin
RUT	-	Rapid Urease Test
UBT	-	Urea Breathe Test
HPSA	-	H Pylori Stool Antigen Test
FAT	-	Fecal Antigen Test
GERD	-	Gastro-esophagal Reflux Disease
EGD	-	Esophago gastro Duodenoscopy
CNS	-	Central Nervous System
CTZ	-	Chemoreceptor Trigger Zone
KS	-	Kasyapa samhitha
MN	-	Madhava nidana
BP	-	Bhavaprakasha

BR	-	Bhaishajyaratnavali
SN	-	Sidhanta nidana
HS	-	Hareetha Samhitha
YR	-	Yogaratnakara
NAFLD	-	Non-alcoholic Fatty Liver Disease
MIC	-	Minimum inhibitory concentration
MBC	-	Minimum bacteriocidal concentration
PECWE	-	Phyllanthus Emblica cold water extract
QOL	-	Quality of life
HIM	-	Hemidesmus Indicus
AIE	-	Azadirachta indica leaves
OMZ	-	Omeprazole
PCR	-	Polymerase Chain Reaction
NDI	-	Nepean Dyspepsia Index
ARM	-	Asparagus Racemosus
CRS	-	Cold restraint stress
SOD	-	Superoxide dismutase
MNZ	-	Metronidazole
TC	-	Total Carbohydrate
SCHAE	-	Hydro alcoholic extract of Swertia chirayita
PL	-	Pyloric Ligation
SD	-	Standard Deviation
MD	-	Mean Deviation
ANOVA	-	Analysis of Variance
HCL	-	Hydrochloric acid
RCT	-	Randomized Controlled Trial

ABSTRACT

A RANDOMIZED CONTROLLED TRIAL TO ASSESS THE ROLE OF MAHATIKTAKAM KWATHAM TABLET IN AMLAPITTA W S R TO FUNCTIONAL DYSPEPSIA

Nearly everyone has experienced a disorder associated with the digestion and the social impact of gastrointestinal illness is probably under appreciated by almost all those who are affected. A majority of the digestive infirmity presented to a clinician is of functional in its nature and Functional Dyspepsia is the dominant among them. A study reported from Mumbai, India had revealed that almost a third of the population suffered from dyspepsia overall, with 12% among them experiencing the symptoms, which are of noteworthy in nature. Considering the high prevalence of Functional Dyspepsia in the current Indian population, socio-economic burden of this disease in our community, is expected to be enormous and of serious in nature, as far as the nation's health status is concerned.

Patients with the chronic dyspeptic symptoms for the past 3 months prior to the consultation, with the onset, at least 6 months preceeding the diagnosis, is confirmed to have the condition of Functional Dyspepsia, as per the currently accepted Rome III criteria. The latest definition of the Functional Dyspepsia is the presence of one or more of the dyspeptic symptoms that originate from the gastroduodenal region, in the absence of any organic, systemic or metabolic basis, that could possibly explain the ensuing pathology. The suspected underlying pathophysiologic mechanism is so complex and varied among the affected, which includes the alteration in the gastric motility, visceral hypersensitivity, altered gastric accommodation, genetic susceptibility, dietary factors, psychosocial factors, and also the infestation by the organism, Helicobacter pylori in the gut. In these ample range of mechanisms, a single drug or approach is seldom providing the results as expected, as per an assortment of the accessible studies.

One of the paramount competent drugs in hand and used worldwide, with efficacy in the condition of Functional dyspepsia, is the PPI group of drugs including the Omeprazole and the reported results are also promising as well, in this regard. This drug was set aside as the control drug, for the study. But Omeprazole is one of the misused drugs as over the counter medication, resulting in quite a few adverse effects in the individuals and also the attained results are not withstanding much, as per reported trials.

One of the key areas resulting from the drug therapy that the Ayurvedic scholars have focused, is to restore the functional status towards the normality, so as to reverse the pathology of the condition. This makes the management of functional disorders a bit easy and fruitful, with this particular system of medicine. Many a functional digestive disorders are being managed effectively by the Ayurvedic practitioners, with proven clinical significance. Functional dyspepsia is being managed using several combinations based on the protocol mentioned, in the context of the condition, Amlapitta. Mahatiktaka yoga mentioned by Acharya Charaka, is one such proficient combination and also observed to be effective as its gritha form in Parinamasoola, as per the study conducted by CCRAS. This combination is being used as the gritha as well as kwatha form, in clinical practice. The kwatha transformed to the tablet form can be administered with ease, in several conditions.

In this study, the Mahatiktaka combination in the form of kwatham tablet was used as the study drug, in Functional Dyspepsia. The kwatham tablet form seems quite easy to administer as the dosage is very accurate, also palatable and comfortable. Besides the tablet format is devoid of the preservatives which is very much idyllic for a person, with the gastric disturbances. Moreover this multidrug combination seems to unravel the multifactorial pathology of the conditions like Functional Dyspepsia.

Aims and Objectives

1. To evaluate the role of Mahatiktakam Kwatham Tablet in Amlapitta with special reference to Functional Dyspepsia
2. To compare the efficacy of Mahtiktakam Kwatham Tablet with Omeprazole in Amlapitta with special reference to Functional Dyspepsia.

Methodology: A Randomized Controlled Trial with 100 subjects in each group, the allocation done using the computer generated random number table and after due written consent, those satisfying the Rome III diagnostic criteria, for Functional Dyspepsia as well as Amlapitta, were included.

Assessment: was done on the day of inclusion, the 15th day of intervention, the 30th day of intervention and also the 45th day, using the Gastrointestinal Symptom Rating Scale (GSRS) and also the Amlapitta rating scale.

Result: The recorded observations were analyzed with the respective statistical methods. Instagraph Pad 1.0 version and Microsoft Excel 07 were used for the same.

Efficacy of the therapy: The overall efficacy of the therapy was significant at 1% level after the intervention and 0.1% level after the follow-up, on the GSRS scale. On the Amlapitta rating scale there was no significant difference between the groups after the intervention, but on the follow-up, there was statistical significance at the 5% level.

Among the individual symptoms in the GSRS scale, those of abdominal pain, borborygymi, sucking sensation, increased flatus, decreased stool, loose stool, hard stool, urgency of defecation and the feeling of incomplete evacuation, there was statistically significant difference between the groups. There was no significant difference in the improvement attained in the symptoms of heart burn, abdominal distension, nausea and acid regurgitation after the intervention.

After the follow up period, there was significant difference between the groups in the symptom of heartburn, acid regurgitation, sucking sensation, increased flatus, decreased stool, loose stool, hard stool, urgency of defecation and the feeling of incomplete evacuation. There was no significant difference in the abdominal pain, nausea, borborygymi, abdominal distension and eructation between the groups, on follow-up.

In the Amlapitta rating scale, the individual symptoms of daha, amlodgara and soola were having high significance, avipaka was having minimal

significance, and the symptom of chardi was having no significant difference between the groups, after the intervention.

After the follow up period, the symptom of soola was having high significance, amlodgara was having significance at 1% level, and the symptoms of daha, chardi and avipaka were having no significant difference between the groups. But there was minimal significance on the total score on the Amlapitta scale, after the follow up. This indicates that the initial response was obtained in the control group, but at the end of the therapy the study drug was having better response, on comparison. The attained efficacy was maintained by the study drug in a better manner.

There was also efficacy on the H pylori for the study drug on the selected patients with a positive test on inclusion, when compared with the control drug, statistically.

Percentage of relief: In the study group, after the therapy, 3 were unchanged, 59 slightly improved, 37 moderately improved and 1 got marked improvement. After the follow-up period, 2 were unchanged, 9 showed slight improvement, 88 moderately improved and 1 attained marked improvement.

In the control group, after the therapy, 5 were unchanged, 89 slightly improved, 6 moderately improved and none markedly improved. After the follow-up period, none were unchanged, 56 showed slight improvement, 44 were moderately improved and none markedly improved.

The level of improvement seems superior in the study group when compared with the control. Even though the initial response was more in the control group, at the conclusion of the trial, the study drug revealed better percentage of relief.

Association between the various contributory factors

Excess use of amla, lavana and katu rasa were having association at 5% level in the GSRS score. Those having Vatha Pitta prakrithi among the subjects were having a significant association. The association between the irregular timing of the meals and the GSRS score was studied and found to be

highly significant. There was a minimal significance between the association of the GSRS score and those with the habit of having processed food, in a more frequent manner.

It was found that there was highly significant association with those who were having stale food and the score of the GSRS. On testing the association between the use of mixed diet in a regular manner and the GSRS, there was significance at 5% level, pointing out the importance.

In those who were reported to be with the history of mental exertion, there was significant association with the severity of presentation of Functional Dyspepsia. Similar significant association was observed in the case of continuous physical exertion as well. There was also association between those with the habit of regular and excessive travelling and the GSRS score in a significant manner.

There was also significant association between those with frequent NSAID administration and the dyspepsia score, indicating the contribution of the factor in the condition.

Conclusion

Ayurvedic management has an immense role to play in the forthcoming days, in the management of functional gastrointestinal disorders, including Functional Dyspepsia. Mahatiktakam kwatham tablet is statistically significant compared with the control drug, on its efficacy, in the management of Functional Dyspepsia. There was no reported incidence of adverse effects throughout the study. The combination is also able to deal with the psychological factors contributing to such conditions as well. The efficacy of this drug in the management of H pylori has to be studied in detail eventhough it was statistically significant, in the selected subjects. The Ayurvedic combinations like Mahatiktaka is a real choice for the management of the conditions like FD, where multifactorial causations are having role in the pathogenesis and also the individual drugs with much more specificity in action is not the exact choice in the management.

Key words: Functional Dyspepsia, Amlapitta, Mahatiktakam kwatham tablet, GSRS score, Omeprazole, H pylori

Chapter - 1

INTRODUCTION

The man and his evolution are being considered as the superlative creation in this universe. According to the Indian tradition, the ultimate goal of health is to accomplish the purusharthas ie. the dharma, artha, kama and moksha which are the prime achievement in one's life[1]. Hence an affection of the health was being considered as a very serious obsession, as it affects the fulfillment of the ultimate aim of one's life ie. the purusharthas during their entire life span. Many an attempts are being explained to maintain as well as preserve the health and any variation or disruption in the same, is approached with serious apprehension, as per the Indian system of medicine.

The modern era and the associated changes with it has affected the human being in the several areas of his life, not only regarding the activities and habits, but also the manner of food, sleep, thoughts and expressions as well, in the due course. The engagement of humans in the excessive travelling so as to fulfill his several deeds and needs, have altered the food habits immensely, in a negative manner, as far as the health status is concerned[2]. Faulty life style along with the unbalanced food habits has affected the digestive system a lot, along

with its function and the disorders of the system are ever on the rise, as reported from studies all over the world. Food is an integral part of the daily life, but we spend fewer point of time for preferring, preparing, the intake as well as digestion of the food[3]. Consequently many of us are not having food in an appropriate manner and form, as expected or as per the body's demand, which may result in major as well as minor gastro intestinal dysfunction or complaints.

Practicing high-quality dietary habits require a little more instance and scheduling than we can usually afford, in this fast as well as altering world and the lifestyle. However, its benefits will pay off at the long run, because of the sufficient scientific evidence that links most G I disorders to diets\dietary habits, in general[4]. Besides is the presence of a positive mind, that is considered necessary for the proper digestion, as well as assimilation of food.

Among the many a kind of the digestive disorders, the functional disorders are the dominating group, compared with the structural as well as the infectious disorders, affecting the tract. The Functional Gastrointestinal Disorders (FGIDs) can affect any part of the GI tract including the esophagus, stomach as well as the intestines. FGIDs account for about 40% of a gastroenterologist's practice and is affecting the quality of life of such individuals in a noteworthy manner[5].

FGIDs are characterized by recurrent symptoms (ie. abdominal pain or discomfort, bloating, nausea, vomiting, early satiety, constipation, or diarrhea) that indicate a dysfunctional GI tract despite that an organic or biochemical rationale for the generation of the symptom, on investigation. Many of the disorders are diagnosed by exclusion of the related structural disorders, along with the diagnostic criterias.

STATEMENT OF THE PROBLEM

As per the available studies, more than 50% of the people with FGID's symptom seldom consult a physician, although they may undertake over the counter medications, on the arousal of the symptoms for the relief, making the computing of the exact prevalence of the condition, as composite. Significant job absenteeism and disability are reported among these, than the people without these symptoms. The most reported or dominant FGID's in the society are the Functional Dyspepsia (FD) and the Irritable Bowel Syndrome (IBS). The prevalence of FD has noted to vary between 11-29.2% as per available studies[6].

The reported prevalence for dyspepsia varies widely among the different populations, possibly because, most of the studies have focused on the uninvestigated dyspepsia, rather than the FD[7]. The definition and the diagnostic approach also have also been modified several times. One of the other affective variable being the difference in the food habits as well as the mode of life, within the various societies, where the studies have been conducted. Further more, some of these studies must have the inclusion of patients with the reflux disease, misclassified as FD; this makes the interpretation of the true prevalence, intricate and final[8]. Variations in the definition of the dyspepsia and specifically the FD, is also affecting the prevalence, as per the experts in this field. The self limitation of the symptoms over a peculiar period of time and also over the counter treatment in some of the subjects is also considerably altering the statistics.

A recent study from Mumbai had shown that almost a third of the population suffered from dyspepsia among which, 12% of them experiencing the symptoms of the disease, considerably[9]. There is a balance in the actual number of people reporting the new symptoms and the number of people reporting the

disappearance of the same. Bearing in mind, the high prevalence of FD reported in the Indian population, socio-economic burden of this disease in the Indian community, is predicted to be considerable and enormous. No Indian study is available to show the health related quality of life in relation with this condition, as less number of studies are aiming such a direction. Studies from the other Asian countries indicate that the FD is associated with the substantial impairment in the quality of life, work absenteeism, decreased productivity and use of health care resources, with the resultant consequent economic burden[10].

FUNCTIONAL DYSPEPSIA – CURRENT SCENARIO

Patients having the chronic dyspeptic symptoms for the past 3 months with the onset at least 6 months prior to the diagnosis, in the absence of any structural abnormality on upper GI endoscopy and possible metabolic or systemic causes, so as to explain the existing symptoms, are classified as the Functional Dyspepsia. ROME III criteria is the most used worldwide, for its specific as well as constant diagnosis[11]. The 2006 Rome III criteria defined the FD and its two subgroups, Postprandial distress syndrome (PDS) and Epigastric pain syndrome (EPS), based on the clinical presentation[12].

Multiple mechanisms such as abnormal gastric emptying, visceral hypersensitivity, impaired gastric accommodation and central nervous system factors are likely involved in the pathogenesis of the disease entity. The other area of interest to be discussed is that of the H pylori and its eradication, in the role, as well as the management of FD. According to several authors, the combination of physiological, environmental, genetic and psychological factors definitely occupy their role in the disease[13].

Currently, the possibilities of available pharmacological agents in the management of the FD are still having its own limits; however, the experience of administering prokinetics, tricyclic antidepressants, Selective Serotonin Reuptake Inhibitors (SSRIs), Proton Pump Inhibitors (PPIs), and several alternative techniques has been accumulated and tried as well[14]. The diverse combination of the alterations in the physiologic functions of the gastrointestinal and central nervous system, result in the very heterogeneous nature of FD so that, the present available protocols, have their own limited areas in the studied efficacy. World is looking ahead to the alternative systems for their contribution to these patients, so as to provide them with a superior prospective.

Due to geographical, social, cultural, educational, and the psychological aspects, the universally pertinent guidelines on the diagnostic and therapeutical measures, are not in a perfect condition so as to implement[15]. The wide range of the available therapies reflect the uncertainty about the currently explained pathogenesis and the lack of satisfactory management of the condition. Many of the studies have concluded that, Omeprazole is observed to be more effective than placebo, in controlling the symptoms of Functional Dyspepsia[16]. The real comparison was with the placebo only, as far as such studies are concerned. The placebo studies are undergoing worldwide in almost all the functional conditions, without an expected yield or outcome. The world is in search of identifying new as well as efficient drugs in this regard.

THE AYURVEDIC APPROACH AND THEIR ROLE

Several Ayurvedic practitioners are dealing with conditions like FD in a satisfactory manner and the system of medicine has definitely a key role to play

in the FGID's, especially. The tridosha theory in Ayurveda, approaches any clinical condition in a functional manner, rather than the structural abnormalities or alterations. It is stressing a good deal on the functional attributes of the body[17]. It is of the view that the functional alterations have to be improved initially, before approaching the limitations in the structure. Once the functional status is normalized, the rest of the problems can be approached with ease. Encouraging perspectives have been recently performed by methodologically well designed interventional studies, with the herbal drug preparations. Herbal drugs, yielded their proven efficacy in clinical trials, offer a safe therapeutic alternative in the management of FD, which is often favored both by the patients and the physician.

Amlapitta is the commonest diagnosis in an Ayurvedic gastroenterology clinic. Many a clinical conditions like FD are managed on the luminosity of the protocol, mentioned for Amlapitta. The symptoms of the condition FD, resemble very much that of the Amlapitta. The drugs mentioned in the management of Amlapitta need special significance here, in the management of the condition. Mahatiktaka yoga is one such widely used combination, mentioned by Acharya Charaka,[18] with proven clinical efficacy in such a condition. This drug was selected for the clinical study due to the confidence attained from the efficacy of the drug, from the reported clinical practice. Several studies are available and contributory in this regard.

SELECTED PREVIOUS STUDIES

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SIGNIFICANCE OF THE CLINICAL STUDY

Eventhough many studies have been conducted with the Ayurvedic drugs since several years, still there are quite a few areas to be explored. Drugs like PPI's are studied extensively and shows positive response in the condition of the FD[19]. The subsequent level of exploration is the comparative study in this direction, with the allopathic counterparts. Such was the thinking behind such an initiation and the commonest PPI, Omeprazole was fixed as the control drug.

Mahatiktaka gritha is attaining efficacy in Non ulcer dyspepsia, as per the study conducted by CCRAS[20]. But there are difficulties in the administration of the drug in the gritha form in many a patient, with associated life style disorders and if there is further restriction, in the administration of sneha preparations. The

alternate option is the kwatha form of the combination[21]. But nowadays the prepared kwatha is on the use, which are having added preservatives. The use of such kwathas is adding on to the dyspepsia, if used in a persistent and repeated manner. That led to the thinking of administering the combination in the kwatham tablet format. This form also helps us to administer the drug in a more accurate dose and is expedient, without the affront of the preservatives. Hence the Mahatiktaka yoga was used in the form of the kwatham tablet, as the study drug.

In the contest of the FD, H pylorus is the most predisposed organism without much substantial evidence regarding the same[22]. This combination of Mahatiktaka was also tried for the action on H Pylori in selected subjects in those which, the Rapid Urease Test, was positive on inclusion[23]. It was a Randomized Controlled Clinical Trial with 100 subjects in each group of those attending the OPD of VPSV Ayurveda College, Kottakkal within the proposed inclusion criteria during the study period and submitted due written consent.

Aims and Objectives

1. To evaluate the role of Mahatiktakam Kwatham Tablet in Amlapitta with special reference to Functional Dyspepsia
2. To compare the efficacy of Mahtiktakam Kwatham Tablet with Omeprazole in Amlapitta with special reference to Functional Dyspepsia.

Null Hypothesis

There is no significant difference in the role of Mahatiktakam Kwatham Tablet when compared with Omeprazole in Amlapitta w.s.r. to Functional Dyspepsia.

Alternate Hypothesis

There is significant difference in the role of Mahatiktakam Kwatham tablet when compared with Omeprazole in Amlapitta w.s.r. to Functional Dyspepsia.

The recorded observations were analyzed and the drawn conclusions were compiled and prepared in the format of the proposed thesis, which consists of the following sections arranged accordingly.

The conceptual part consists of three sections

1. Review – Functional Dyspepsia
2. Review – Amlapitta
3. Drug review – Mahatiktaka yoga

Clinical study part consists of three sections

1. Methodology of clinical study
2. Observations, Analysis and Interpretation
3. Discussion, Summary and Conclusion.

Chapter - 2

FUNCTIONAL DYSPEPSIA

CONCEPTUAL STUDY

Many of the subjects attending a gastroenterology OPD is reported to have conditions which are of functional in nature ie. the pathology of the condition is resulting from the alteration in the normal functioning of the tract, so that there is variation in the expected physiology, resulting in the clinical presentation. Such conditions cause extreme dissatisfaction and loss of confidence in the affected, even though it doesn't result in harm to the life of the individual. Also one has to proceed for the costly investigations to rule out the structural lesions, with similar or resembling presentation.

Many of the factors including the diet, stress, habits such as smoking and alcohol, alterations in the motility of the GI tract, hyper sensitivity of the intestines etc. are being studied as the contributors in such conditions[1]. These may also result from certain diseases affecting the nervous or endocrine functions like diabetes, thyroid disorder etc.

Among the functional disorders of the GI tract, the foremost presentation is that of the Functional Dyspepsia (FD). Rome III criterion is being used worldwide, to make out the condition of FD. It includes the presence in the individual of one or more of the symptoms of

- Bothersome postprandial fullness
- Early satiation
- Epigastric pain
- Epigastric burning

for a minimum period of 3 months with the onset of the symptoms, atleast 6 months prior to the diagnosis. Also there may be no absolute evidence of a structural association on the performed investigations, including the upper GI endoscopy.[2]

The disease was better known in the prior years by the terminologies, Essential Dyspepsia or the Non Ulcer Dyspepsia, as the symptoms of the peptic ulcer was negative on investigation along with, the patient still presenting the symptoms.

In the scenario of management also, there is vast difference in opinion regarding the FD and its management, among the medical professionals, all over the world. Many studies are reported with the efficacy of diverse drugs, at dissimilar levels. A few studies conclude that placebo is as effective as the medical management of such a condition, as expected in a functional condition. Similar is the opinion and feedback about the efficacy of psychotherapy, dietary management, alternative therapy etc. Great scope is still left over in the research of this condition, as far as the medical science is concerned, as all the fraternities are having much more expectations, in the formulation of a gold standard therapy regarding the aspect of its management[3].

DYSPEPSIA - Historical Perspective

Dyspepsia is a term used to explain a group of symptoms referable to the upper GI tract of the individual. It is said to be affecting one-fourth of the population worldwide as per studies, in one or another stage of their life.[4] It is also affecting their quality of life as well as the productivity is concerned, at the young age itself. Even though the condition is being explained in the history of medicine, the advances in the knowledge of the anatomy and physiology of the GI tract, has shed a large amount of light, into the pathogenesis. The treatise of the ancient system of medicine, Ayurveda is explaining a similar condition, in a very respectable manner, including its management in the context of Amlapitta[5].

In the 19th century, the habit of smoking, alcohol and the excess use of the drugs like NSAID's and the steroids were being the only blamed, for the development of dyspeptic symptoms. Later, after the development as well as the explanation of the psychosomatic concept of the disease, the psychological stressors were also being considered as contributory to the same[6]. These all lead to the discussion in the history of medicine, whether the psychological events are having any role in the management of the so called physical illness. A strong emotion seems to alter the functioning of the GI tract, leading to the manifestation of dyspeptic symptoms. Tranquilizers were also tried with, but with little success, by many a practitioners worldwide.

Similar was the effect of the identification of the mucosal barrier with the bicarbonate ions, having the role of protecting the gastric mucosa from the influential acid attacks. That altered the management strategy towards the condition and the medical world felt that, the neutralization of the acid was not

the only option mandatory, to get rid of the same[7]. Previously it was even explained that excessive production of the acid, was the chief culprit in all the dyspepsia's. Later it was traced out that in many subjects with less or normal acid output also, similar presentations were reported.

One of the revolutions in the area of conditions like FD was the identification of the organism, the Helicobacter Pylori from the tract. It was traced out that this organism while able to survive in such an acid medium, can also release cytotoxins, which causes mild dyspepsia to even gastric carcinomas, in its due course in many of the affected[8]. That led to the Nobel Prize achievement of the two Australian physicians, Warren and Marshall. This pointed out to the eradication of the causative organism, as the advancement in the management. That was a real milestone in the management of FD, which still needs further clarifications about the contribution to the aetiology, before generalization.

The altered immune system is severely affecting the functioning of the mucosal barrier was a further explanation, in the pathology of the conditions like FD, this being contributed by the physical, psychological as well as the chronic drug administration. The food allergies are also suspected for the same. Immune enhancement was also being thought upon in the protocol, after this explanation[9]. The immune system and its alteration were discussed in the recent studies undergone, in the area of FD worldwide. The combination of the above said factors ie. when the psychological stress alters the immunological factors, the chance of infestation of the organisms like H pylori, is on the mount[10]. These inter relations altered the approach towards management of conditions like FD.

The improvement the science had achieved regarding investigations led to the tracing out of the pathology inside the gut and also to the mentioning

and distinction between the structural and functional group of diseases of the GIT, including that of FD. In one of the studies, it was observed that 1/3rd of the subjects were presenting with a normal endoscopy, in those having dyspepsia. After the same, the upper GI scopy is being considered, as the gold standard investigation in this regard[11]. Functional Dyspepsia can thus be identified from the organic conditions like gastric ulcer, by detailed history taking, along with the above said investigation, as explained in the Rome III criteria.

A genetic component was also being traced out in the manifestation of the conditions, especially functional in nature, including the FD, which resulted in the explanation of a polymorphism of the genes associated with the condition (GNBeta3). These points out to a similar approach, we have to develop in this regard, in the management of such a condition[12].

Dyspepsia – Prevalence

It is quite usual for the conditions like FD which depends on the food, life style as well as the psychological factors for its occurrence, to vary in prevalence across the world, as these factors quite vary, from place to place and from one country to another. It is being reported that it occurs in about 25% of the adults annually, of which about less than a half seeks, apt medical attention. It is being reported that dyspepsia as a whole before investigation occurs in 10 - 45% of the population, while the FD upto 30%.[13] As the symptoms vary from one place to another across the globe, it is a racket whether the standard definition seems applicable to all. It is in fact the presence of the upper abdominal pain or discomfort with or without other upper gastrointestinal symptoms, such as nausea, belching, vomiting, fullness etc.

Functional Dyspepsia - Evaluation

The economic impact as well as the alteration in the quality of life of the affected, is creating keen interest in the researchers regarding the FD. It is a clinical condition in which, the person presents with features of dyspepsia as mentioned earlier, with the fact that there is no organic disease or biochemical variation, that could satisfactorily explain the possibility of the symptoms. The symptoms have to be there for more than 3 months, to reach a diagnostic conclusion. The affected person approaches a practitioner due to the severity of the symptoms or scared of any grave disease. One has to go ahead with the Rome III diagnostic criteria for a conclusion and also has to exclude the organic cause for the same[14]. A list of gastro irritant drugs like NSAID's as well as corticosteroids also have to be considered contributory for the pathology.

ROME III Criteria

Must include one or more of

- Epigastric pain
- Epigastric burning
- Early satiation
- Bothersome post prandial fullness and

No evidence of structural disease at the upper GI scopy, that is likely to explain the symptoms.

Criteria fulfilled for the last 3 months with the symptom onset at least 6 months prior to the diagnosis[15]

Types of FD

In observing the symptoms of the FD for long, it came to the mind of the medical world that more than one category is there for the disease, as the symptoms are varied.

If the symptoms of the individual are mainly epigastric pain or discomfort that is relieved with food or antacids, it is called ulcer resembling type of FD. If the symptoms are dominating in the motility part with nausea, abdominal bloating, early satiety etc. it is being treated as dysmotility type FD. This classification puts forward a fragment to think, regarding the management rather than a unique approach for all.

As per the ROME III criteria the condition is of 2 subtypes[16]

a) Post prandial Distress Syndrome (PDS)

Here bothersome post prandial fullness happening after an ordinary meal and/ early satiety that prevents from completing a regular meal, existing for several times a week, is to be considered. Nausea after the food intake, belching and epigastric pain usually associates the same.

b) Epigastric Pain Syndrome (EPS)

In this variety, pain is the dominant component in the clinical presentation. Intermittent moderate pain or burning sensation localized to the epigastrium may be there, at least once per week and not localized or generalized to other abdominal or chest regions and must not be relieved by defecation or passage of flatus. The pain may occur while fasting, but induced or relieved by ingestion of a meal.

It is still a debate whether there can be a perfect distinction between the subgroups of FD, but many are of the opinion that even though slight overlap may be there, the bifurcation is positive for the thinking between diverse approaches of the management for the condition. There is scope for further studies or research in this regard so that the management can be much more effective.

Factors contributing to the pathology of FD

The condition of FD is being explained to be resulting from a cluster of alleged mechanisms in the individual. The precise mechanism of pathogenesis is yet to be explained by the medical community. The alterations of the physiology and its various contributors are blamed or being suspected in this regard. Many are to be studied at a clinical level by the upcoming studies.

The concerned mechanisms include[17]

- Altered gastric or duodenal motility
- Hypersensitivity to gastric or intestinal distension
- Defective gastric accommodation to a meal
- Gastrointestinal infections
- H pylori infestation
- Neurohormonal dysfunctions
- Psychological factors including stress
- Dietary mismanagement
- Genetic susceptibility etc.

Let us discuss in detail the role and mechanism of each and every factor in the pathogenesis of FD.

Alteration in the GI tract motility

Impaired control of the neuromuscular apparatus of the GI tract is the resultant aspect of the altered motility. The altered motility results in digestive disturbances and the resultant symptoms of dyspepsia. The motility can be affected in any part of the tract, leading to the upper or lower GI symptoms. Studies have postulated several alterations in FD such as slow emptying, abnormal distribution of the contents of food, alteration in the rhythm of gastric contraction, abnormal contraction of the fundus of the stomach, altered motility of the intestines etc. in the affected individuals. The slow emptying of the gastrum is contributing to the symptoms of abdominal bloating, fullness, nausea etc[18].

The accommodation of food has also been explained as altered in FD, as most of the food was seen at a lower part of the stomach, when compared to fundus in healthy volunteers, the reason being yet to be recognized. Alteration of motility has also been visualized and reported in FD, as per the Electro Gastro Graphic studies as well.[19]

Diet and Environmental factors

Many patients presenting with the FD explains to have specific food intolerances, but a satisfactory relation between the diet and the resultant condition, has to be traced out further. Many food items are being suspected including the spicy food, alcoholic drinks, wheat rich diet, caffeine etc. but the exact mechanism is yet to be known. Along with the intolerance, the factor of hypersensitivity is also coming to the act, as add on in the pathogenesis[20].

Eventhough the acid secretion seems to be normal in subjects with FD, studies propose that the gastric as well as the duodenal mucosa is hypersensitive, even to the normal level of the acid output, resulting the condition.

Disturbances in the Brain Gut Axis

Enteric nervous system is a part of the central nervous system supplying the GI tract and acts as a part and parcel of the same. Those seeking medical attention for FD are reported to be more vulnerable to anxiety and depression, than the normal subjects. Acute stress or similar psychological components result in reduced gastric contractility, which leads to the symptoms. A supressed vagal tone and a higher sympathetic tone, have been disclosed in FD as well. The alteration of the perception by the stomach is said to be resulting in the symptoms such as abdominal distension. Activation of the area responsible for the regulation of hunger and satiety was observed in the subjects with FD, at a lower level of stimulation, in PET studies, indicating the related hyper response[21].

Altered visceral sensation

The threshold of a sensation to a physiological or pathological stimulus seems to have lowered and there is a resultant brisk response, as seen in FD. The enhancement is recorded in both the perception and also the neural processing, by the CNS[22]. The excitability is aggravated in the afferent nerve cells of the viscera, present in the GI tract. This is the reason behind explanation of the condition as nervous dyspepsia, on preceding days. The threshold seems to have lowered, both for the perception as well as the adaptation of the pain, as proven by the balloon distension tests, in the tract.

PET studies prove that gastric distension activates the area of lateral pain system. Several chemicals are also suspected in causing the hypersensitivity of the GI tract, resulting in the altered function in FD eg. Capsaicin. The lipids in the diet is also proved to have affecting the gastric relaxation as well as the

distension. Similar is the case of the hypersensitivity to the acid, regardless of its level. Glutamate, the neurotransmitter is also being pointed as the culprit for the pseudo perception of the sensation, peculiarly from the gut.[23]

Genetic contributors

The existence of conditions like FD more in families have raised the suspicion of the medical world, regarding the role of the genetic factors or causes for the same and there are many supported studies. Genetic epidemiological studies are also pointing to the same conclusion. Genetic factors are also suspected to have in the role on exposure to the dietary or environmental factors in an individual[24]. It is believed that the genetic factors determine the response of the GI tract to an external stimuli, contributing to the pathology. Interleukin17, Migratory Inhibitory Factor and Serotonin Transporter Protein have all been studied in detail as contributory. More studies have to be conducted before the conclusion of their role in FD.

Immune system and its involvement

In a few individuals presenting with the FD, the symptoms are post to gastrointestinal affections, raising the suspicion where the immune system has been affected as well as involved, resulting in the manifestation. The delayed or late response of the immune system towards the normalcy, is being noticed by the medical fraternities in this regard as contributory[25]. Medical world is in constant exploration of a possible inflammatory response in other GI conditions like Ulcerative Colitis, which may revolutionize the strategy of management.

The inflammatory mechanism affecting the gastric functions like impaired accommodation and delayed emptying, is yet to be studied. The role of

raised cholecystokinin is also being a matter of research, in the pathology. Similarly the increased incidence of FD in those with the allergic manifestation including esnophilia and allergic asthma, is also being a centre of attraction, so as to study the association of the immune alteration in FD.

H pylori infection

The identification of the organism, H pylori in the GI tract have altered the outlook of the management strategy, of several abdominal conditions. This is supported by the information that the occurrence of FD is much more dominating in those affected with the helicobacter, than the normal population. In its due course, the affection of the organism seems to alter the functioning of the GI tract as per studies, the exact mechanism yet to be acquainted with[26]. On the other side, the eradication of the H pylori in those positive, is also contributing to the improvement, pointing to its importance. Here the concept of post infectious dyspepsia can also be discussed, in the case of H pylori.

Many are of the opinion that the condition of FD can be classified into two, based on the presence or absence of H Pylori, for the sake of management. But everyone with the H Pylori positive FD are not benefitting with the much emphasized eradication therapy as per studies and also is questioning the significance of the eradication[27]. The efficacy of the therapy differs as per reports from the all over the globe, in a considerable manner and the reported maximum, being less than 25%.

Testing for the *H. pylori*

If the individual is not responding to the usual protocol for the FD within 2-3 weeks, the tests for H Pylori, has to be kept in mind. It is also being

performed to identify those with dyspepsia having PUD and infection. About 80% of the subjects with PUD have an associated H Pylori infection, even though the relation has not been evidently established. Also the investigation is to be done, if one plans to perform an eradication therapy for it. In those who are opting for the upper GI scopy, invasive tests can be done and non invasive tests are preferred in those without GI scopy [28].

In the Rapid Urease Test, which is an invasive test, biopsy is performed and placed on a strip with urea and a Ph indicator. From the resultant colour due to conversion of urea to ammonia and bicarbonate, the presence of active infection with H pylori can be identified. The patient is being advised to stop the PPI for about 2 weeks before the test usually, so as to overcome a false result.

Non invasive tests are used in the identification of H Pylori infection at a primary level and also for confirmation of the eradication, after the completion of the therapy. The urea breathe test (UBT) and the stool antigen test (HPSA) are the most accurate noninvasive tests for *H. pylori* infection in the case of specificity and sensitivity. **.** In the presence of *H. pylori,* the ingestion of urea, labelled with the non-radioactive isotope 13C or 14C, results in the production of CO_2, which can be quantified in the expired breath, useful in both pre and post testing**.** The patient has to stop the antibiotics and PPI's for a minimum of 2 weeks before the test.

In the stool antigen test, the H Pylori bacterial antigen in the stool is being identified with the use of the antibody, by performing the immune assay. The test is effortless, cost effective and also used to confirm the eradication of the organism and seems effective, as early as 14 days after the therapy.

Serologic testing is used to detect the IgG or IgA antibodies to H Pylori in blood for detection of the same, which becomes detectable 3 weeks after the infection and cost effective as well. It can also be detected upto weeks after the eradication.

Differential diagnosis of FD

The functional disorders affecting the GI tract is having numerous symptoms in common or with much overlap and is also really confusing, especially for the general practitioner. But one desires to have a clear cut idea so as to perform an effective management[29]. The conditions to be considered here are

- Gastro-oesophageal reflux disease
- Peptic ulcer disease
- Drugs like NSAID's and Antibiotics
- Cholelithiasis
- Diabetic gastroparesis
- Irritable bowel syndrome
- Chronic pancreatitis
- Functional abdominal bloating

By performing the investigative techniques such as upper GI scopy as well as Ultra Sonography of the abdomen, the organic conditions can be ruled out to a better extent. One has to exclude many a diseases, as the diagnosis of FD can be reached by exclusion only. The physician should perform a detailed history as well as the abdominal examination at the initial sitting, tracing any clue that point to a diagnosis, other than the FD. Drug history has also been given its own

importance so as to rule out the drugs, causing the gastric insult. The psychological status also have to be assessed, to trace out their contribution.

Upper GI scopy

This is the investigation with gold standard for the diagnosis or exclusion of any type of dyspepsia[30]. It also helps to perform the invasive test for the confirmation of H Pylori infestation like RUT and can also able to reassure the patient with anxiety, regarding a serious illness and hence termed as cost effective. It also helps to rule out a structural lesion in the GI tract and aids in diagnosing the conditions like FD and has fully replaced the unfriendly investigations, like barium meal x-rays.

If a person is presenting with the condition like FD after the age of 55 years, certain features are to be observed for, as they may be a part of some serious illness which needs immediate intervention and we have to rule out these conditions, at the earliest. These include

- Abdominal mass
- Progressive dysphagia, Odynophagia
- Persistent vomiting
- Previous documented peptic ulcer
- Lymphadenopathy
- Early satiety, unexplained weight loss (>10% body weight)
- Bleeding, anemia
- Family history of gastrointestinal carcinoma

They have to undergo immediate endoscopy, as per the latest protocol[30]. It is better to perform an endoscopy in those with long term history of

NSAID or corticosteroid intake. It can also be done if the individual is not responding to a minimum 30 days of medical management, as per available studies. Once endoscopy is performed, no need of further investigation to be done, unless there is worsening of the symptoms or arousal of new symptoms. The benign nature of the lesions like peptic ulcer, can also be confirmed with GI scopy.

After the endoscopy and physical examination, if one suspects of any organ pathology or so, an ultrasound scan of the abdomen have to be performed, which provides with fruitful information.

Approach towards a patient with FD

As the condition of FD is resulting from numerous factors in the pathogenesis as described above, a common approach is not favorable always, in those affected. Besides, the presentation is also wide-ranging from one to another and there is a lack of a standard therapy and not all patients are responding, to the same pharmacologic approach. Along with it, many studies are reporting a placebo response in FD, in a considerable manner. The commonly used drug groups include the acid suppressants, mucosal protectants, prokinetics, H pylori eradicators, antidepressants and so on, individually or as a combination[31]

The percentage of therapeutic efficacy varies for the different groups of drugs in FD, as per the reported studies. Likewise is the case of dietary interventions, H Pylori eradication as well as the psychological therapies.[32] The expected reason behind may be that, each of the drugs are working on the corresponding pathology alone and neglecting or unaffecting the rest. As a result, the medical world is in search for a better option or the development of new drugs in the management of such condition.

FD – primary approach

The primary areas of concern in dyspepsia are whether the condition in functional or not, whether there is any need of an upper GI scopy, is H pylori involved in the pathology, what potentially may be causing the symptoms, what is the psychological background of the patient, what is the dietary status, what all drugs are being used causing the abdominal symptoms and is there any familial factors to be considered as supportive. These questions are to be answered all through history taking as well as physical examination, along with the possible investigations[33].

The major strategies in practice include a trial of acid suppressants, a test and treat approach for H pylori and also an early upper GI scopy. In the absence of any warning signs, the test and treat approach seems to be on a better side. It is the clinician's responsibility to decide about and perform the necessary investigation. The type of the drug as well as the dose and the duration has to be discussed and fixed. If the patient is not responding to the empirical therapy, the investigation has to be decided without hesitation. Immediate investigation along with biopsy need to be performed if there are warning signs, as mentioned earlier[34]. Let us discuss the mode of action of each of these group of drugs.

GASTRIC ACID SUPPRESSION

These groups of drugs are being in use extensively, with considerable improvement, in the conditions like FD worldwide. These reduce the acid production and the resultant effects, in a very noticeable manner. H_2 blockers were used in the initial trials being replaced by the Proton Pump Inhibitors in both, by the efficacy as well as safe and short term use. It is better to assess the

cost and safety profile of these drugs peculiarly, on long term use. As per studies, ulcer like dyspepsia is having more response than dysmotility like dyspepsia on prescribing this group of drugs. These drugs are reported to be functioning better after the eradication therapy, in H pylori positive individuals[35]. There is no generalized conclusion that basal acid output is affected, in those with FD. But there is clear cut evidence of dysregulation of the acid secretion, in those positive with H Pylori.

PROKINETICS

This group of drugs enhances the smooth muscle contractions that increase the gastric emptying and also the intestinal transit time. Improving the gastric motility is helpful in the symptoms such as abdominal bloating, early satiety, nausea etc. These drugs are having more potency in post prandial distress syndrome group of FD patients. These drugs are also having a property of increasing the lower esophageal sphincter pressure and also discourages the reverse peristalsis[36]. This enhanced motility is effective also in mild pain associated with this condition, as it potentiates the intestinal motility. Prokinetic agents are reported to have more efficacy than H_2 receptors, according to certain studies in FD. Herbal drugs containing peppermint is also reported to have efficacy in FD, with its action on the intestinal smooth muscles. The mode of action of the prokinetics in the symptomatic relief of these patients is yet to be explained in a satisfactory manner.

Drugs which causes fundic relaxation are also being tried with efficacy in FD as impaired fundic accommodation is blamed in its pathology and they enhances accommodation to a meal.[37]

Eradication of the H pylori

As the output of various reported trials are having difference in opinion regarding the efficacy of the management after the eradication of the H Pylori, it is still a matter of debate. The present approach is to eradicate the organism in all the subjects whom positive, from any of the tests. But the long term follow-up studies show results not satisfactory, as expected. It arises the doubt whether the eradication of the H Pylori is beneficial, for the management of FD, in the long run. Fewer studies have undergone in strictly H Pylori negative cases. The response with the H Pylori eradication also varies in different parts of the world.

Recently, a 4 drug combination with a PPI, Amoxicillin, Clarithromycin and Metronidazole is given sequentially for the purpose of eradication. The area of concern is the developing resistance by the organism and presently a post test is being advised in many cases, which are not responding as expected[38].

Psychological Approach

As the functioning of the GI tract is altered by any psychological disturbance, it points to the fact that such an approach is utmost essential for the management of functional conditions like FD, affecting the tract. The reason narrated being the affection of the enteric nervous system and its functions. Psychological factors affect the motility as well as the sensation of the GI tract, which is in fact a key area in FD. Studies point to the fact that a higher percentage of subjects with this condition have psychological distress or comorbid minor psychiatric illness.

If an individual is not responding to the medical treatment upto 6 months and there is exacerbation of the symptoms with stress or emotional factors, one may assume of a psychological intervention. Cognitive behavioural therapy,

relaxation therapy and the hypnotherapy are reported to be beneficial in this condition, according to various studies. The psychological approach varies from one disease to another in the gastroenterological conditions[39]. The results attained are also maintained for a longer period than the drug attained relief.

Because of the higher rate of comorbid conditions like anxiety and depression, many physicians are using antidepressants and anxiolytic agents in FD, with claimed positive response. The action of antidepressant can be explained as it reduces the associate psychological problem; the symptoms of FD definitely reduces. Promotion of sleep also alleviates the condition. Anxiolytic agents are said to have positive benefits in post prandial distress syndrome.[40]

Psychotherapy like CBT is reported to have greater efficacy when used with the other drugs for FD as they manage their response to the stressful situations. One drawback is that the methodology varies from person to person and from study after study, in the case of proposed psychotherapy.

Resistant FD

There are a group of FD subjects who doesn't respond to the above mentioned approaches and are termed as the resistant group. The various drugs are used as combination conditionally, in such subjects. Research is progressing with a few drugs that arises the threshold of pain arising from the gut. Here there attains an area of scope of research in newer drugs and approach in FD[41].

The better strategy in the management of FD is in the initial stages, go for the PPIs or H_2 Receptor Antagonistic group of drugs. If not responding, go for the investigations, including the upper GI scopy. Test the presence of H Pylori simultaneously and eradicate, if positive. Still no response, proceed for the

psychological pathway. If alarm signs present, go for endoscopy initially and then fix the treatment protocol accordingly. In H Pylori negative patients, if there are no alarm signs, go for the antisecretory therapy, for a period upto 8 weeks. The treatment options like psychotropic medication, antispasmodics, prokinetics etc. are used conditionally. Short duration of treatment intermittently is superior to the long duration of treatment, as per studies[42]. Physician is having the last word of decision in this regard.

Diet and life style modification

As in any other conditions affecting the GI tract, the diet and life style modification are having utmost importance, in the management of FD. The limitations in the pharmacologic management compels the people to go for the dietary adjustments, so as to achieve better results. The diets creating the disturbances vary from person to person, in the case of FD. Fatty food, spicy diet, milk and milk products, smoking, alcohol etc. have been blamed in this regard and observed to trigger the symptoms. These food products alter the hypersensitivity as well as the gastric accommodation after a meal. Better to prefer frequent small meals rather than a larger one and avoid lying down for 1-2 hrs after the meal seems ideal peculiarly, if the subject is having abdominal bloating[43].

Maintaining an ideal body weight and performing minimal exercises are very much essential, in those with FD. Elevating the head part of the bed upto six inches also seems working in this condition. The association of psychological factors alters the food habits, leading to the manifestation of the symptoms. Fewer studies have been reported evaluating the efficacy of diet and life style modifications in

FD. A generalized recommendation for FD is yet to evolve in this direction. Thus the gastroenterologists are hesitant for advising a general schedule and leave the same to the affected individual, to identify the triggering factors.

Conclusion

Eventhough the medicine has advanced a lot, still the pathogenic mechanisms of the conditions like FD are a mystery for all[44]. The various factors are contributory in an overlapping nature for the condition. The presentations are also varied from one to another, among the affected. Likewise is the response to the various management protocols. A combined approach with the pharmacological agents, H Pylori eradication, psychotherapy, life style modification etc. has to be adopted in this regard, as per the demand from the condition. There is scope of research of developing new drugs really working in this condition and with minimal adverse effects, taking into consideration, all the contributory factors.

Chapter - 3

AMLAPITTA
CONCEPTUAL STUDY

The life style of today is absolutely altered or modified, within the last two decades, when compared with the past. Likewise are the habits of food intake as well. Most of the people are not able to compromise much in their food habits. They have awful food habits like irregular intake, eating of smoked foods, excessive use of spices, chillies and masalas, intake on inappropriate time, using processed foods, having food with preservatives etc. Similar is the psychological imbalances observed in the present day community and the relation with food. The non proper living style and faulty dietary habits upset the digestion initially. It is reversible to some extend. If we are repeating the same contributory factors in a regular as well as constant manner, it may lead to conditions like dyspepsia, peptic ulcer and other major disorders of the GI tract, in the due course[1].

Due to these alterations in the food and life style, more than a few people are suffering from a number of disorders of digestion and the resultant clinical conditions. The most common abdominal condition experiencing in the primary health care, is dyspepsia[2]. Gastric dyspeptic disorders are mainly caused by the unfriendly dietetic pattern, mental stress and strain, mishandling of certain

drugs etc. which are directly or indirectly affecting our digestion or its mechanism[3].Ayurveda has given utmost importance to optimal dietary regimen and its variation as per the season, agni, prakrithi, type of the intaken food and the disease condition[4]. The unaccustomed variations in the same, may lead to the diseases of the koshta or the GI tract. Depending on the three doshas, which gets disturbed with the pathology, the condition or the disease varies and is named accordingly, in the classics.

Charaka and Kashyapa have clearly discussed that the Grahani dosha and Amlapitta occurs in the person, who could not verify the temptation of food in their life[5]. The nidana or the etiological components create agnimandya or depleted digestive mechanism and thus ajirna is developed and leads to the formation of amavisha or substances which are not supportive in nature or even harmful to the body. This amavisha mixed with the doshas, mainly the Pitta dosha and gets accumulated in the amashaya, then gradually resulting in the diseases like Amlapitta[6]. The alteration in the status of the doshas lead to various other clinical presentations.

In Bruhatrayi, diseases such as Amlapitta have not been explained as such, by the name. In many diseases, the symptoms resembling that of the disease can be traced out. It is also dealtwith among the indications mentioned in the context of a few medicines. Charaka has specified more than a few references about Amlapitta, in a scattered manner[7]. Acharya Kashyapa was the first to mention a separate chapter to deal with the Amlapitta, among the ancient Indian legends.[8] Acharya Madhava and the Bhavaprakasha have specified a detailed description on the symptoms and also the management of the Amlapitta.

The management is explained under mainly two categories. Hetuvipareetha and the vyadhi vipareetha are the core two types of the chikitsa[9]. The vyadhivipareetha chikita is being performed mainly at the OPD level in our regular practise. The drugs are being selected and prescribed as per the clinical condition of the patient, for the recovery. On the other side, Hethuvipareetha chikitsa, which is the ultimate and wholesome management, can be successfully performed, at the inpatient level. All the suitable sodhana procedures can be incorporated in an appropriate and required manner, on a conditional basis. Each of these branches has their own role to play, in an ultimate management protocol. Satvapachaya chikitsa is the third component which is to be explained, under the psychological aspect and is one of the key approaches in many diseases in the current social scenario, including the GI disorders.

Among the sodhanas, vamana is the most important as well as effective tool, as far as the Amlapitta is concerned[10]. We are using several drugs with success in the management of Amlapitta, for the purpose of samana. Along with the same, a few dietary corrections are also advised and are also having a very significant role, in the current scenario. Another area to be discussed here, is the emotional status of the affected individuals.[11]

Gastritis and Functional dyspepsia have been compared and discussed along with Amlapitta, by several experts of Ayurveda. Modern medicine is not having the exact as well as ultimate answer for gastric diseases, including the Functional Dyspepsia[12]. Ayurveda have a lot to offer in this regard. Ayurvedic physicians are providing cure for the patients of these category of chronic diseases, in a quite efficient manner, where a few more research works are expected in the near future.

HISTORICAL REVIEW

For the complete acquaintance of any subject, it is necessary to trace out its historical background, which gives a concrete picture in the development of the science from time to time. This seems more applicable to the most ancient health care system of the world, Ayurveda. The development this system has achieved in the ancient period itself, makes the study of its history interesting and informative as well. Here the references we are receiving about the disease Amlapitta, has been dealt with detail.

Vedic Period:

The review of Vedic literature points to no known suggestive references, regarding the description of Amlapitta.the But among the puranas, in the Agnipurana, the author is mentioning about Gastrointestinal conditions like atisara, aruchi, grahani, sula etc.[13]

SAMHITA KALA

The real references in Ayurveda about this clinical condition seems available, from the period of samhithas itself.

Charaka Samhitha

Eventhough Acharya Charaka has not mentioned Amlapitta as a separate entity in the samhitha; the treatise is contributing with many scattered references of Amlapitta by the name itself, which are explained below.

1) Among the indications of eight types of ksheera, Amlapitta is mentioned, for the exercise of the particular ksheera.[14]
2) While mentioning the ultimate dravyas, Kulattha is considered as chief etiological factor or the contributor for Amlapitta.[15]

3) The excessive use of lavana rasa is said as the causative factor for conditions including Amlapitta, while describing the function of various rasas. This is due to the resultant dushti happening to the Pitta as well as the Rakta.[16]

4) A list of diseases ensuing from the recurrent intake of virudhahara is mentioned by the Acharya, while explaining the consequences of virudha, which includes also Amlapitta.[17]

5) Rajamasha is mentioned to have the property of relieving the condition, Amlapitta and is also enhancing the taste.[18]

6) Mahatikta Ghrita has been indicated for the disease Amlapitta, with its administration.[19]

7) While describing Grahani roga, pathogenesis of Amlapitta has been clearly mentioned, while explaining the role of agnidushti and its role in the manifestation of diseases, by affecting the three doshas, mainly the Pitta.[20] A general samprapthi of a GI disorder can be traced out here.

8) The indications of Kamsaharitaki, mentioned along with the management of sopha, also includes Amlapitta.[21]

9) The list of Paittika nanatmaja disease includes dhumaka, amlaka and vidaha which are the symptoms seen in Amlapitta, eventhough the term as such, canot be traced out here.[22]

The references are sufficient to give a clear cut indication regarding the samprapti and management of Amlapitta, in the period of Charaka, eventhough the disease as such is not explained in the samhitha, as a separate disease entity.

Sushruta Samhita

Though Charaka has mentioned the word Amlapitta, but it is not traced

out in Sushruta Samhita. Susrutha is explaining the sthana of Pitta as madhya of amasaya and pakwasaya and Dalhanacharya is explaining the yakrit, pleeha, hridaya, drishti and twak as the pecuiliar sthanas of Pitta. Katu rasa is the natural rasa of Pitta says the Acharya and it becomes amla when vidagdha, which is observed here in the pathology[23].

Sushruta while describing the diseases resulting from excessive use of lavana has mentioned a condition "Amlika" which resembles one of the symptoms of Amlapitta[24]. Among the five components of the Vatha, Susrutha mentions that Prana, Samana and Apana controls and maintains the agni directly. While explaining the treatment of dosha according to the localisation, Acharya is of the opinion that, if Pitha is localizing in the seat of Kapha after dushti, the management to be done is for the Kapha, initially which is an important management protocol followed in the case of Amlapitta.

Ashtanga Sangraha and Ashtanga Hridaya

Vagbhata has also not described the Amlapitta as a separate disease entity. In many diseases like hridroga, grahani, chardi etc. particularly in the Paittika variant, many a symptoms observed in Amlapitta are explained[25]. In Paittika chardi, symptoms such as dhoomaka, amlaka and chardi with Amlapitta is being explained. In Paittika grahani, amlaka is described among the poorvaroopa. In the Paittika grahani amlodgara, hrit-kanta daha, aruchi and chardi are also mentioned. In vidagda ajeerna also, the symptoms similar to Amlapitta is explained, but as one among the classification of the ajeernas. The condition of soola in the amashaya is explained by the Acharya, after mentioning the hridrogas.

Kashyapa Samhita

Kashyapa is the former among the ancient scholars, who mentioned Amlapitta, as a separate disease entity. The acharya is explaining detailed causative factors including ahara, vihara, vegadharana, divaswapna, virudha etc. for the materialization of the Amlapitta. Not only the vivid description of the Amlapitta with its treatment, has been mentioned in it, but also the suggestion to change the place for the peace of mind in case where the medicine does not work out, has also been narrated[26]. These points to the importance of a psychological balance in alleviating the symptoms of Amlapitta and also in those affected. Samana vatha along with Udana is responsible for the normal digestion, opines Acharya Kasyapa. Vitbedha is also considered here among the symptoms of Amlapitta.

Kasyapa is explaining the variants of Amlapitta as per the doshas, with the pecuiliarity in the symptoms, so that it can be approached in the clinics, in an enhanced manner[27]. This is also a hint for considering the associative dosha in the management of Amlapitta. He is also explaining the relevance of vamana in its management, with its pecuiliarity and also the possible mode of action[28]. The dietary restriction has also been adviced with its magnitude, in the treatise. The utmost importance given to this disease by the Acharya has been esteemed.

Harita Samhita

A separate chapter for the disease of Amlapitta is available in this treatise[29]. Acharya explains the etiological factors as the guda sevana, excessive intake of substances with amla rasa and also intake of virudha aahara as the causative factors. The important symptoms mentioned are the burning sensation

in kanta pradesha as well as hridaya and also sirasoola. Udgara or hikka, with amla in rasa, is also being experienced by the patient[30].

The management is also advised where Vamana is mentioned for the urdhwaga Amlapitta and virechana for adhogata Amlapitta, as the protocol. A kwatha with patola, paatala, dhanyaka, vasa, nimba and nagara is also mentioned. Dhanyaka and naagara is advised as kalka form at night, for attaining pachana.

Madhava Nidana

Madhava nidana is the first text available which gives importance to Amlapitta and describes its aetiopathogenesis and symptoms in detail along with two clinical sub types viz. 1) Urdhvaga Amlapitta and 2) Adhoga Amlapitta[31]. This is one of the concepts we are using very effectively, in the clinical scenario of management. The involvement of the other doshas, Vatha and Kapha in the etiopathogenesis has aso been discussed. The prognosis has also been mentioned as well[32].

Chakradatta

Chakrapani in his commentary on the management of grahani has given the symptomatology of Amlapitta without mentioning the references[33]. He discussed its management in the manuscript Chakradatta, which is one of the latest available books on chikitsa in Ayurveda. A detailed management protocol has been explained with equal importance to sodhana as well as samana chikitsa. Sodhana as per the condition followed by the samana, is the advice. Several samana drugs like kwatha, choorna, gritha, rasa preparations etc. have been explained[34]. Laja saktu has been mentioned along with the dietary regimen which seems clinically very effective. The importance of tikta rasa in the diet, among all the rasas has been projected.

Sharngdhara Samhita

In the chapter dealing with the enumeration of diseases, Acharya has explained three types of Amlapitta, Vathika, Kaphaja and Vatha kaphaja[35]. Here Pitta dosha has been kept as a common and the associative doshas, Vatha and Kapha are given significance, in the classification. This also points out the significance of keeping the associative doshas in mind, while framing the management. Amla asyata is also mentioned as one of the condition among the nanatmaja roga of Pitta[36]. Sharangadhara has mentioned recipes for the condition of the Amlapitta like laja mandha, indicating the importance of dietary modification.

Yogaratnakara

Regarding Amlapitta, the author has followed the Madhava Nidana as a whole, in the aspect of nidana and lakshana.The oordwaga, adhoga types and also the doshika fractions have also been discussed along with the detailed management. Acharya commences the management by performing vamana with the drugs patola, arishta, madanaphala and saindhava, mixed with honey [37].

Virechana is to be performed next with the trivrit choorna mixed with the kwatha of triphala. If one does not get relief as expected with the sodhana procedures, raktamoksha is also advised, followed by the dietary restrictions for Amlapitta[38]. If the burning still persists, one have to advise samana drugs accordingly. Vamana followed by virechana is to be done, in praboothа dosha.

Anuvasana is to be done if necessary after snehana, following the sodhana therapies. Nirooha is also to be done if the condition is chronic. The author is also quoting the protocol mentioned by Acharya Vrinda, that Kapha Pittahara drugs are the ideal choice for Amlapitta. This statement is having extreme

significance as many of the drugs we use in an effective manner in Amlapitta, is of Pittakapha samana in nature.

Sarpi with guda, ksheera and kana and also Kamsahareethaki are advised, if Vathakopa associated with vibanda is present, in a person with Amlapitta.[39] Several yogas are also mentioned for the management. Kwathas like guduchyadi kwatha, lehyas like nalikera khanda, grithas like satavari gritha, rasa preparations like sootasekhara rasa, along with a selected diet also explained[40].

Gada nigraha

Acharya Shodala is here also unfolding the condition Amlapitta, with due consideration. The nidana, symptoms and the varieties resemble that of the Madhava nidana. Sleshma Pitta lakshana has also been narrated.The treatment pattern resembles that of Kashyapa to some extend. Several kwatha yogas are also mentioned.[41] A patient with severe burning sensation will be beneficiary on performing the sodhana therapy only, advises the Acharya. Vamana is ideal for both the chronic as well as the acute cases.

Vangasena samhitha

In this script also, Amlapitta is dealt with sincerely, as a separate chapter. The nidana portion resembles that of Madhava nidana and the treatment part that of Chakradatta, to a great extend. Acharya explains that the assessment of the dosha in Amlapitta may create moha or confusion in the physician. He also advises to perform a lepa on kanta with Kaphasamana drugs in Kaphaja symptoms like aruchi, asya vairasya etc.[42] Kabala with the Pitta samana drugs are also advised in the resultant arochaka.

Raktamoksha is also advised in a patient not responding to sodhana, followed by application of seeta lepa. A kwatha with yava, dhatri and trisugandha along with honey is mentioned for chardi, with Pitta of amla in rasa. Paittika grahani chikitsa is also advised for adhogata Amlapitta along with pachana and deepana drugs, as per the condition.[43] In a case of Amlapitta whether chira or achira, vamana is the ideal option, says the Acharya. Vangasena is advising tiktaka gritha, shadpala gritha, panchatiktaka gritha and guggulutiktaka gritha for Amlapitta. Besides narikela khanda and also a combination, brihat nalikera khanda is also explained. Avipatti karachoorna is also explained in the same context.

Vrinda Madhava (Sidhayoga)

Vrinda Madhava in his Kusumavali vyakhya mentions Amlapitta as a separate condition and also explains its management. He has mentioned vamana, virechana, anuvasana and asthapana as the main protocol for the condition[44]. The avasthika chikitsa has also been discussed. The treatment seems analogous with that of Kasyapa. Administration of several kwatha yogas and the use of yoosha are also adviced. Kusmandaka khanda is mentioned in the condition of associated raktasruthi. Grithas like vasa gritha, tiktaka gritha are also mentioned. Combinations like pippalikhanda, khandanalikera are also mentioned.[45]

Veera Sinhavaloka

This treatise has considered Amlapitta as a separate disease along with its varieties and the management. Vamana is the initial treatment, followed by virechana and samana therapy. A few yogas like guda kooshmanadaka has also been narrated.[46]

Basavarajiyam

This book has included Amlapitta among the twenty four nanatmaja

vyadhis of Pitta. Among the other symptoms amlodgara, trishna, aruchi and also the symptoms like swara heenata, jihva-vak paridaha has been attributed to Amlapitta, which has not been mentioned in other texts.[47] Rasoushadis including sudhanidhi rasa, lilavilasa rasa, use of abhraka etc. has also been explained.

Bhavaprakasha

Amlapitta has been dealt seriously in this particular samhitha with the entitlement of 'Amlapitta sleshmapitta adhikara'[48]. Etiopathological factors and classification has been described with similarity of Madhava nidana. The symptoms of Sleshma Pitta has been described which includes praseka, mukha madhurya and siroruja and also a choorna combination has been mentioned. Here there is an indication of identification of Kapha dominant Amlapitta. He has put forward many recipes along with khanda kushmanda avaleha, narikela khanda and brihat narikela khanda.[49] The importance of daily intake of amalaki swarasa is mentioned as effective in Amlapitta and also as a rasayana**.**

Vaidya Jivana and Vaidya Rahasya

A kwatha of bhoonimba, nimba, triphala, patola, vasa, amrita, parpata and bringaraja is the only mentioning in Vaidya jivana. In vaidya rahasya, the treatment of Amlapitta is explained, which resembles that of Chakradutta. Sodhana chikitsa followed by samana, is the proposed protocol with combinations that include avipattikara choorna, nalikera khanda and gudapippalyadi yoga.[50]

Bhaishajya Ratnavali

The treatise explains a number of recipes for Amlapitta and also the effective soubhagya sunthimodaka and sunthi khanda.[51] Along with the same,

pathya-apathya for the particular roga, has also being explained. The treatment protocol observed is very much similar to that of Chakradutta.

Sidhanta nidana

This treatise is visualizing many of the diseases through the gaze of modern symptomatology as well as the pathology. Gananathsen opines that the indulgence of the nidana mentioned for vidagdhajeerna at a superior or next level, leads to the manifestation of the condition, Amlapitta.[52] Anasana, akalabhojana, madyasevana and vishamasana has been included among the nidana, which may lead to the increased production of amlarasa srava. This amlarasa srava aggravates with the intake of the food. The excess amlarasa produces daha and also damages the amasaya abyantara kala.[53] The pathology seems to have been explained, as per the modern medical knowledge.

Raktapitta, amasaya kshata, atisara, siro peeta and soola are included among the symptoms. The types as per the dosha also have been explained. He describes some of the complications that of Amlapitta, which is not mentioned in the other former texts such as udarda, vicharchika, visphota, raktapitta, grahani etc.[54] Amlapitta which is chronic and associated with soola is said to be not good in prognosis, says Sidhanta nidana. Also while explaining the soola, it is said that, of all the soolas, those caused by Amlapitta, is the most vital, as well as severe one. If it is occurring in the amasaya, it is called annadravasoola and if in grahani, it is termed as the parinamasoola[55].

To conclude, it is crystal clear that, the various treatises in Ayurveda had approached the condition Amlapitta very seriously as well as systematically.

Definition and Etymology of Amlapitta

The 'Amlapitta' is constituted of the words 'Amla' and 'Pitta'.[56] The term Amla has been used as an appellation to Pitta. Though, the Amla has been said as a natural property of Pitta along with katu rasa, according to Charaka[57]. Sushruta has enlisted katu as its original rasa and mentioned that when the Pitta becomes vidagdha, it transforms to amla in rasa.[58].

By considering the disease as well as its symptoms, it seems that in Amlapitta, the Pitta seems distorted or is vidagdha, as mentioned by Acharya Susrutha. Shrikantha datta in his commentary here, has defined that Amlapitta is a condition where excessive secretion of Pitta with amla in guna takes place, resulting in the conditions like vidaha etc[59].

- "Amlagunodriktam Pittam Amlapittam"[60]
- "Vidaahyamla gunodriktam Pittam" [61]
- "Amlapitta samjnaam tu amlamamlaadhikam Pittam yateti Vyakhyaaya" [62]

Therefore, Amlapitta is a condition where amlarasa of the Pitta dosha gets amplified, in an unusual manner. For any dosha, the rise in a pecuiliar characteristic of it, eventhough it is its natural one, is considered as pathological. The normalcy of the various properties of a dosha is essential for its ideal functioning. In Amlapitta, the Pitta gets vitiated by one or all the gunas, causing various pathophysiological conditions of annavaha srotus and purishavaha srotus.

Madhava Nidana has given a clinical definition of Amlapitta that, the presence of avipaka, klama, utklesha, amlodgara, gaurava, hrit-kantha- daha and aruchi together should be coined as Amlapitta[63]. This is a disease mainly due to

vitiation of Pitta (Pachaka) but Kapha (Kledaka) and Vata (Samana) vitiation is there associated, as mentioned by Shrikanta datta. In samhitas, some other words have also been mentioned, in the reference of Amlapitta. These are amlaka, dhumaka and vidaha which are seen in conditions, with the disturbed Pitta[64].

The word Amalpitta is composed of the words Amla and Pitta, as mentioned earlier. Pitta is a dosha which is present in the living body and responsible for the digestion, formation of raktadhatu, colouration of the skin, vision, body temperature etc.[65] In brief, it is more or less responsible for all the biochemical transformations, at the cellular level as well as the supracellular level.

'Amla' is presented as a qualitative word, which is indicative or characteristic of the guna which is a rasa, implicating sour in the taste. The amla rasa is having its own natural physiological functions in the body including digestion, as mentioned under the karmas of the individual rasas[66]. The rise from the optimum level may cause alterations in the function of the body. That is the reason of a balanced diet being advised, constituting of all the rasas[67].

According to Dalhana, the commentator of Sushruta samhitha, there are two clinical stages of Pitta.[68]

(1) Sama Pitta (2) Nirama Pitta.

This is a condition not only for Pitta but also for all the other doshas. Acharya assumed that sama Pitta has amla rasa, while nirama Pitta has katu rasa. So the symptoms of Amlapitta are due to the Pitta, which is sama or associated with ama or a condition produced by sama Pitta.[69]

Chakrapani assumed that Amlapitta is a condition in which amla property of the Pitta is exaggerated. Madhukoshakara has also accepted this

definition. He has given the vidagdha status of Pitta as a causative factor, for aggravating the amlaguna of Pitta.

ANNAVAHA SROTUS

Anatomical and Physiological Aspects

The word Annavaha srotus narrates the channel through which food is transported, metabolised as well as absorbed. This is otherwise called the koshta or the mahasrotus. The koshtangas mentioned provides functional support for the entire mechanism.

The functions of Annavaha Srotus deals with

- Anna adana (ingestion)
- Anna pachana (digestion)
- Sara kitta vivechana (separation of nutrients and waste)
- Rasa soshana (absorption of nutrients)[70]

MOOLA STHANA

For every srotus, the moola sthanas are mentioned by the Acharyas which can be inferred as the key centres for controlling the functions, to be performed by the system, which is represented by the srotus. Like wise these areas are having too much significance in the management of pathologies affecting them. For the Annavaha srotus they are

- Amashya and vamaparshva[71]
- Aamashaya and annavaha dhamanis[72]

These are said to be the manipulative centres for the entire digestive mechanism, in the body.

Chakapani has given the two terminologies - urdhva and adha, in the case of amashaya[73]. Urdhva amashaya is the location of the Kapha, while the adho amashaya is the location of the Pitta. Amasaya can be considered as the sthana of both the doshas, Kapha and Pitta. Otherwise, a disease in which amasaya is the prime sthana, both of the doshas has to be considered in the pathogenesis as well as the management.

The ingestion and the deglutination process of food commences from mouth and in the upper part of the esophagus. The main digestive process starts from the stomach, even though salivary enzymes also has a minimum role to perform, in the initial stages. Digestive juices are secreted from the lower part of the stomach and the intestine. Bile and the pancreatic juice secreted from the liver and the pancreas then after, secretes to the small intestine.

Therefore we can include oesophagus and upper part of the stomach as urdhva amashaya and lower part of the stomach and small intestine as adho amashaya, for the matter of explanation. So both the functions of Kapha and the Pitta, can also be interpreted here. The term annavaha dhamanis are also, explained as the moola sthana, of the annavahasrotus which are the channels that transport the end products of ahara from the intestine, to the blood. Under the microscope, the mucous membrance of the small intestine contains villi, the purpose is to increase the surface area of absorption, within the available area. The villi are providing such an extensive area for the GI tract, for enhancing the absorption, as equal to surface area of a tennis court, as per published studies[74].

This villus is lined by a single layer of epithelial cells, small arteries, veins and lymphatic vessels. The villi act as a semi permeable membrane and

permits the course of digested food through the rasavaha srotus and raktavaha srotus. In other words, these microscopic parts of the membrane, carry out the transportation of the ahara rasa though the intestinal barrier, so that it is absorbed to the body and are transformed as the dhatus.

PITTADHARA KALA

Acharya Sushruta and Vagbhata have described the Pittadhara kala. Acharya Sushruta opines this as "the sixth kala situated in between the pakvashaya and the amashaya and it is better known as Grahani."[75] In his view, "the integrity of grahani depends upon the agni." As per Charaka, grahani is so called because of grahana ie. it receives and retains the food, upto the extent of its digestion. He observed that the food, which has reached the amashaya after under going the digestion, is absorbed, while the grahani holds the food for the time being, essential for the digestion. Such a minimal retension of the food or a timelag is crucial for its proper digestion[76]

Pittadhara kala endows with the digestive juices collectively and functions as jatharagni. These enzymes not only digest the food, but also aids the seperation of the saara and the kittabhaga.

The descriptions of Pittadhara kala illustrate that it is a macroscopic structure which not only serves as the protective lining of the small intestine, but also as a secreting and absorbing structure. The structure can be compared as the absorptive area of the small intestine responsible for absorption. Here the term 'grahana' is mentioned not for the sphincteric action, but holding the food so that, the absorptive action is properly undergone. So the area responsible for performing the digestion of food, that is localized in the intestine is the "grahani".

Samana Vatha

Vagbhata clarifies that the Samana Vatha is located in the vicinity of the agni and responsible for the reception, digestion, separation as well as propulsion of the food[77]. The seat of agni is said as grahani. The functions of Samana Vatha are similar to intrinsic nervous system of the stomach and intestine. Intrinsic nervous system is the part of the autonomic nervous system, supplying the gut. This system is related to the brain and the spinal cord and works as its integral part.

The peristaltic movements of the intestine are responsible for the mechanical breakdown of the intestinal contents. They are throughly mixed up with the enzymes of pancreas, liver and the small intestine and are absorbed through the intestinal wall. So the Vatha dosha definitely have an important role in the modus operandi of digestion. One such condition we are observing frequently in the clinics is the diabetic gastroparesis where, the gastic emptying is delayed due to the affection of the intrinsic nervous system, resulting from the slowening of the nerves, resulting from the diabetes[78]. It is a magnificient observation done by Vaghbata that, there is a chance of one with prameha, to have kroorakoshta, and has to be managed accordingly.

This has been known to be described as the role of Samana Vatha in the digestion of food, separation of nutrient fraction and also expulsion of the undigested food ie. pachana, vivechana and munchana.

We consider following organs & systems in annavaha srotus

(A) Amashaya:-

(i) Urdhva :- (a) Oesophagus (b) Upper part of the stomach

(ii) Adha: - (a) Lower part of the stomach (b) Small intestine

(B) Pittadhara kala: - Inner layer of mucous membrane of small intestine and lower part of stomach responsible for absorption.

(C) Annavaha dhamanis: - The channels that receive the end products of the food from the intestine.

(D) Samana Vayu: - Intrinsic nervous system of the GI tract.

AHARA PAKA KRIYA

The ahara undergoes two stages for the complete digestion says Chakrapani.[79]

(A) Avastha paka (B) Nishta paka or Vipaka

In Ayurveda, the digestion and metabolism is interrelated to agni. Mainly the Pachaka Pitta is responsible for the digestion of the food.[80] The Pachaka Pitta is situated in the grahani that directly participates, in the mechanism of digestion. Grahani is also considered as the Pittadhara kala, by the Acharyas. Avastha paka is the primary phase and vipaka is the subsequent phase of the digestion. Avasthapaka, the former phase of the digestion is completed by pachakagni in the annavaha srotus and vipaka, the subsequent phase of digestion is completed by bhutagni and dhatvagni. Vipaka commences after the avasthapaka and is also responsible for the metabolism at the cellular level.

(A) AVASTHAPAKA

There are three stage of the avasthapaka mentioned as such[81].

(i) madhura avasthapaka (ii) amla avasthapaka (iii) katu avasthapaka

(i) Madhura Avasthapaka

Four types of intaken ahara like the asita, peetha, leeda and khadita that reaches the amashaya turns in to madura bhava, initially. At this stage, salivary

digestion will be completed in the fundus of stomach, where the insoluble starch and polysaccharides are converted into soluble dextrin, under the influence of salivary amylase. The final rasa in the upper portion of the urdhva amashaya, is madhura. Prana Vatha is responsible for the entire movement of food from the mouth to the amashaya.[82] It is responsible for the mechanisms like shteevana and also controls the budhi or the higher control, in this regard. Any alteration in the functioning of Prana Vatha results in the disorders of deglutition or swallowing. Such presentations are seen in conditions like bulbar palsy, where the deglutition is affected, eventhough there is no disturbance in the organs concerned with the same.

The Bodhaka Kapha and Kledaka Kapha are also responsible for the madhura avasthapaka. Bodhaka Kapha is responsible for perception of the taste of the food in the mouth.[83] The Bodhaka Kapha is the analogue of the saliva which acts on substances including proteins, the enzyme content begins to act and it lubricates the food. Kledaka Kapha lubricates the food in the amashaya and also converts it into such a form, so that the action of the other enzyme is also enhanced. We can consider it as mucine like substances present in the stomach.

(ii) Amla avasthapaka

An amla type of srava occurs here, and after its completion, ahara is transformed to amla and hence termed as the amla avasthapaka.[84] Ahara gets converted from insoluble proteins to soluble one, under the influence of pepsin, in the presence of the hydrochloric acid. According to Charaka and Vagbhata, the final outcome of the entire gastric digestion is the acidified chyme. The term 'Vidagdha' has been interpreted by Chakrapani as 'pakva- apakvam' or 'kinchit pakvam - kinchit apkvam' i.e. partly or not fully digested[85]. At this phase, the

ahara pachana is due to the amla factor secreted within the urdhva amashaya. The ahara which is amla in nature passes on to the next lower portion of the annavaha srotus, where achapitta is secreted.

The acidified chyme passes down from the pylorus to the duodenum, acts as a stimulus for the duodeneal glands, to secrete the secretin, cholecystokinin, enterogastrone, pancreozymine etc. The presence of acid in the duodenum enhances the circulation of the secretin and stimulates the flow of the pancreatic juice. Secretin also enhances the secretion of the bile and the intestinal juice.

The pancreozymine and intestinal hormone also stimulates the secretions of enzymes from the pancreas, occuring in the intestinal mucosa. Cholecystokinin is also responsible for the contraction of the gallbladder and therefore the discharge of bile to the duodenum. All these hormones acts due to entering the acidified chyme in to duodenum and therefore pancreatic juice, bile and intestinal juice are secreted in the small intestine[86]. In Ayurveda, achhapitta can be considered as a combination of these enzymes, responsible for the mechanism of digestion. Due to these enzymes, all the fats and semidigested proteins are digested and converted to fattyacids, glycerol and the amino acids.

(iii) Katu Avasthapaka

The content transverses down the pakvashaya from the amashaya and are being dried up by the action of agni and are rendered in to lumps here ie. Paripindita pakva.[87] Already the main component of absorption has been completed from the small intestine itself. The sesha agni indicates that the digestive process going on here is continuos that of amasaya. During this process, Vatha and mala

are produced, as the outcome. The minerals and water are absorbed from the end products and the remaining materials are converted into feaces.

(B) VIPAKA

According to Charaka, the digestion of food by the jatharagni breaks down the food into parthiva, apya, agneya, vayavya and akashiya.[88] Activated agni bhuta is present in each of these bhautika groups. The bhutagni thus activated, digests the substance of the pecuiliar group, converts and also helps their absorption into the body.[89] Panchaboutika nature of the sareera is maintained with the food in this manner.

(i) Bhutagni Paka

Bhutagni paka follows jatharagni paka and it completes the process of intestinal digestion.[90] After the bhutagni paka, ahara rasa is completed and the rasa shoshana is possible. Thus the Agni constituents of the predominant parthiva molecule spoken as the parthivagni, digests the substances of the corresponding molecule. Similarly apyagni digests the substance of the molecules of apya and so on. The out come of this type of digestion according to Chakrapani, is the transformation of the characteristic qualities of each group and the assumption by them of vilakshana gunas or all together new qualities.This is infact the conversion of the food as a whole to its different basic components, being explained through the physico chemical parameters, according to this ancient science.

(ii) Dhatvagni Paka

After jatharagni paka and the bhutagni paka, the resultant ahara rasa is absorbed from the annavaha srotus and circulates of dhamanies throughout the body. This annarasa undergoes the process of dhatvagni metabolism and thus the

seven dhatus are formed and also stabilized.[91] Each of the seven dhatus has to be provided nutrition for their growth and maintenance. Seven dhatvagni corresponds to seven specific dhatus respectively: viz rasagni, raktagni etc. The rasagni does the digestion of the ahararasa, so rasadhatu and its mala ie. the malarupa Kapha is the result.

Similarly, every dhatvagni digests the same molecular particles of the ahararasa and the corresponding dhatu is developed. Charaka explained that the pakas act upon the seven dhatus giving rise to kitta and prasada bhaga[92]. The prasada part is related to the anabolic aspect and the kitta part to the catabolic one. Dhatvagni converts the ahara rasa in to the sthayi and asthayi dhatu. Prasada bhaga is being an asthayi dhatu. Asthayi dhatu is converted into sthayi dhatu by the particular dhatvagni by its accomplishment[93].

By the dhatvagni, the kitta portion like sveda, mutra, purisha, vatha, pitta, kapha, smashru, nakha, kesha etc are also developed, which are infact the dhatumalas.The significance of the dhatumala is that, they are the direct indicators or predictors of the ongoing proper dhatu metabolism. The alteration in the status of the malas indicates a derangement of the dhatu metabolism and has to be considered by the physician. So their level in the body is having its own significance, rather than the functions.

Jadaragni paka results only in the breakdown of complex substances into their elemental forms, which continue to be viajatiya in nature, says Chakrapani. Bhutagnipaka is required to process and convert them to pre-homologous substances. These are being worked upon by the seven corresponding dhatwagnis, leading to the contribution to the respected dhatus.

Ayurvedic scholars have given various hypotheses like Khalekapota nyaya, Ksheeradadhi nyaya, Kedara kulya nyaya etc. so as to narrate the transformation and metabolism of dhatus in the body, in an scientific manner[94]. These seem helpful in illustrating the dhatu transformations and the pattern of distribution to all dhatus, in an effective manner. These are also helpful for a critical and analogical reasoning of the pathologies, in the dhatu parinama as well. For eg. in conditions like muscular dystrophy, where the mamsa dhatu is affected much more, than either the rasa or rakta dhatus, the pathogenesis can be explained using the khale kapota nyaya as the nutrition of mamsa dhatu is impaired without much affecting the rasa or rakta dhatu, the contributory component being, the genetic or hereditary factors. Like wise is the case of the other nyayas. The various nyayas represent the different aspects or the areas of the dhatu metabolism.

PATHOPHYSIOLOGICAL ASPECTS OF ANNAVAHA SROTUS

Main Reasons for vitiation of the srotus

(1) Atimatra Bhojana (2) Akala Bhojana

(3) Ahita Bhojana (4) Agni Dusti [95]

Ati matra bhojana is the excessive intake of food not only in quantity, but also in quality. Nowadays the over or excess nutrition with the reduced expenditure of energy resulting from the sedentary life style, is the causative factor of many a lifestyle disease. Hence the santarpanajanya diseases are on the rise, nowadays all over and is also gaining importance.

Akala bhojana means the irregular pattern of food intake which includes the time factor as well. Timely intake of food and its importance is an

area that needs further discussion. Its role in the pathogenesis of many abdominal conditions has been studied in detail. Ahita bhojana means the food taken by the person that is not reliable or ideal for his health. The aspect may have a psychological component as well as it affects the proper digestion, of even an ideal food.[96] A food which is ideal to a person may not be suitable for another. Dietary habits also vary from place to place or country wise.

Agni dusti means the improper digestive power or mechanism which may be contributed by quite a few factors. First three causative factors mentioned above contribute to the agni dusti by the alteration in the customary functions of the doshas. The cause of agni dushti is also due to other diseases like rajayakshma, inhibition of vegas, excess intake of drava ahara or due to the psychologic components such as soka, krodha etc. affecting the digestion[97]. The violation in the rules regarding the sodhana procedures also results in the disparity of agni.

All the above causative factors create the platform for the manifestation of a disease of the annavaha srotus, including the Amlapitta. They generate the amadosha and abnormality in the functioning of the doshas, which is responsible for the aggravation of the mechanism of the diseases like ajirna, chardi, atisara, arsha, grahani, Amlapitta, alasaka, aruchi, visuchika etc.[98] These causative factors alter the functioning of the annavaha srotus and its components as well as the Pittadhara kala resulting in the manifestation.

DUSTI LAKSHANA

Four cardinal symptoms are mentioned resulting from the annavaha srotodusti[99]. They are the the primary and cardinal presentation of any diseases affecting the srotus including the GI tract. These features are seen as combinations

in several diseases and are also of the most informative.

(1) Arochaka (2) Avipaka

(3) Chardi (4) Anannabhilasha

(1) Arochaka

The loss of exact taste of the food is considered as Arochaka[100]. The loss of interest in the intake of food even though it is very excellent and delicious, is the case. Acharya Sushruta opined that arochaka is a condition in which one has absolute loss of interest in the food due to shoka, bhaya, krodha, lobha etc.[101] Vitiated tridoshas and manasika bhavas are mentioned as locating in jihva, hridaya and bhaktayana in the condition[102].

The different areas of sthanasamsraya point out to the various causes of arochaka like oral cause, psychological cause along with the GI cause. According to Sushruta, manasika bhavas including shoka, bhaya etc. are the dominant causative factors of the same. They generates the vitiation of the three doshas. Vitiated doshas situated in jihva, hridaya and bhaktayana causes the various types of arochaka according to the etiological factors. Arochaka is to be dealt with seriously because it may be the key presentation of an uncomplicated disease like Pandu to the most terminal conditions like gastric carcinoma[103].

(2) Avipaka

Avipaka points to the lack of proper digestion as well as absorption of the food. The paka of the ahara or biotransformation is affected due to disturbance in agni or alteration in the digestive mechanism and the avipaka is the resultant condition. It is also termed as ajeerna, ajaraka, apakti and paktinasa by various scholars[104]. Any sort of digestion can be included under avipaka, if it is

lacking in any sort, so that it is not resulting in effective absorbtion. It is one of the symptoms of koshtagata Kaphakopa says vridha Vaghbata.The paka of the food is affected by the altered functioning of any of the three doshas.

The causative factors may be either a psyche or a somatic one. Grahani is the key organ in the annavaha srotus, responsible for the digestion and the absorption of the ingredients. Any alteration of its functioning may lead to symptoms like avipaka. So the Pittadhara kala and the agni are also disturbed, because there is a reciprocal relationship between the agni and the grahani. Such is the explained pathogenesis and significance of the conditions like grahani[105].

The mucos membrane lining the stomach and small intestine is responsible for the secretion of enzymes. If there is some disturbance in it, the enzymes are not secreted properly, resulting in avipaka. Avipaka is also resulting from conditions with hypermotility of the intestines like IBS, diarrhea etc. Many GI conditions such as gastritis, peptic ulcer, malabsorption syndrome etc. have avipaka as the primary symptom. Many of the psychological factors such as fear, anxiety etc. also leads to avipaka. It is one of the presentation in many a psychiatric conditions like depression, indicating the psychological component.[106]

(3) Chardi

Chardi is the forceful expulsion of the gastric and / or duodenal contents through the mouth. Usually it may be the resultant item from avipaka and the alteration in agni. Mainly the components of Vatha dosha, Udana and Samana seem to be disturbed in Chardi, according to Susrutha[107]. Prana vayu is also a contributor factor, as the transfer of food from the mouth to the stomach, is well explained as its function.[108]

Chardi occurs when any part of the upper GI tract is excessively provoked or disturbed. Impulses are transmitted both by vagal and sympathetic afferent to the vomiting center of the Medulla, which lies near the tractus solitarious.[109] Motor impulses are transmitted though the 5th, 7th, 9th, 10th and 12th cranial nerves to the upper GI tract and through the spinal nerves, to the diaphragm and abdominal muscles.

This nervous mechanism is considered as a vitiation of Vatha in Ayurveda, as the sole responsible factor for the motility, is Vatha[110]. Mainly observation of anti peristalsis activity is responsible for the vomiting. Irritation of mucus membranes like gastritis, enteritis etc are also responsible. In psychological vomiting, there is direct stimulation of the chemoreceptor trigger zone (CTZ) which is causative behind vomiting, at once, without enough prodormal or warning symptoms.[111]

(4) Anannabhilasha

Anannabhilasha means the overall loss of desire or interest of food even though it is provided, as per the daily demand by the individual. Chakarapani has mentioned that the particular person can dig the food in to stomach through the mouth with the loss of interest of food, persisting [112]. The absolute loss of appetite may not be there, but the interest in the food intake is mislaid, accordingly. In Ayurveda, this is termed as abyavaharana sakthi and it has to be successfully differentiated from jarana sakthi, which is altered in several conditions.[113] Here, if the person is taking the food without considering the status of abyavaharana, it may not get properly digested.

Aharaparinama kara bhavas

Gastro intestinal digestion or change in the state or form of the food substances in amashaya and pakvashaya is the course of the digestive process. Two phases of the paaka ie. prapaaka and vipaaka have been envisaged. The prapaka opines Chakrapani, as the prathama paka or the primary stage[114]. These changes have been described in terms of the rasa or the taste of the end products of gastro-intestinal digestion viz. madhura, amla and katu[115].

Prapaka commences right from the time, when food is introduced into the mouth. This aspect of digestion ie. in the upper portion of urdhwa amaashaya are comprehended by the madhura bhava. When the food is introduced into the mouth, the perception of its rasa takes place which is stated to be enabled by Bodhaka Kapha[116] which is also agreed by Vaghbata.The next event which takes place is the categorisation of food by the tejas or agni element of the lala srava, also described in Ayurveda Sootra[117]. Taste perception, preparatory digestion and the beginning of the madhura bhava occur here. The movements are brought by Prana Vatha.[118]

The second phase i.e. amla avastha paaka involves the vidagdha stage of food. As the partly digested food which has attained amla bhaava is moved down, the achha Pitta is secreted.[119] The term amla refers to the production of Pitta under influence of the ahara, which has since assumed amla in nature.

The third aspect of avastha paka is the katu bhava. This aspect relates to the acrid and pungent nature of the reactions that occur in the pakvashaya. Charaka explains that the material passed down from the amashaya having reached the pakvashaya is dehydrated and converted into lumps by the ushma[120].

Chakrapanidatta has observed that the term soshana used by Charaka instead of Paachana, is very relevant.[121]

The former relates to the dehydration of the food residue, which has been brought to pakvashaya whereas, the later refers to the digestion of food in the amashaya by the agni. The term 'Paripindita pakva' refers to the process of formation of fecal lumps[122]. The term 'Vaayu syat katubhavatah' describes the production of acrid and pungent gas[123]. Pakvashaya is the seat of Vatha where all the fractions are formed and hence the pakwasaya is considered as the prime site of the Vatha dosha. The importance of pakwasaya in the management of Vatha vyadhi comes over here. That is the reason of vasthi being considered as the ultimate sodhana procedure for Vatha, the site of action of it, being the pakwasaya.

Sushruta opines that the separation of rasa, mala and mootra is brought about by the Pachaka Pitta[124]. Sharngdhara and Bhavamishra states that, the sara bhaga or useful portion is known as rasa, and the saraheena bhaga is the mala[125,126].

Shad ahara parinamakara bhavas or the six factors responsible for the proper digestion are mentioned by Acharya Charaka[127].

i.e. i) Ushma ii) Vayu
iii) Kleda iv) Sneha
v) Kala vi) Samyoga.

Let us discuss one after the other and its role in digestion.

i) Ushma: Ushma is a quality of agni mahabhuta, which is represented in the body in the form of Pitta. Here two related terms are to be considered i.e. Agni and Pitta. Sushruta explains that there is no agni except Pitta, in the body

and that agni or Pitta can be represented itself in the body, in the form of the ushma only[128]. Out of the five types of Pitta, Pachaka Pitta situated in the amashaya, performs all the favorable and unfavorable functions, described as agni. Various secretions of the GIT can be considered in the radiance of Pachaka Pitta. The Pitta which has lost the natural drava guna is said to function as the agni. So the release of these secretions in proper time and the quality of the same is crucial for proper digestion, interruption of any of them will lead to agni dusti and commences the background of the aetiopathogenesis, for a disease.

ii) Vayu: Samana Vayu is seated in amashaya and helps the Pachaka Pitta in digestion, explains Sushruta[129] There is a vicious relationship between the three components of Vatha responsible for the entire digestive mechanisms, Prana, Apana and Samana. The Prana and Apana Vayu balances or maintains the Agni. Prana Vayu directly takes place in the act of digestion by transporting the food upto the stomach and Samana moves in koshtha all around and performs the functions attributed to agni, grahani and Pachaka Pitta. Three phases of gastric acid secretion can be considered under the karma of Vayu ie. the cephalic phase, gastric phase and intestinal phase. The other two fractions of the Vatha, the Udana and Vyana is also having indirect role in maintaining the digestive mechanism.

The apakarshana, grahana and munchana karma of Vatha are essential for proper digestion and any exacerbation or cessation in these, lead to improper digestion. As definite time is required for proper digestion, delayed emptying will cause shuktapaka and formation of annavisha or ama, essential for the samprapti of abdominal disease. Now, it is clear that all the regulations of the secretions can be understood to be by Samana Vayu. Any disturbance of Samana causes agni vaishamya, which leads to ajeerna and the pathogenesis. The etiological factors

like krodha, shoka, bhaya, chinta and other stress factor work through the vagus chain, which is mediated by Vatha. Provocation of Vatha by any factor will result in hypo or hyper secretion leading to gastritis or similar manifestations.

iii) Kleda: This factor is necessary for the proper digestion. For a food to digest in an effective manner, several changes must occur inside the GI tract. Kleda looses and emulsifies the food substance, so that it may be easily digested[130]. This function is performed mainly by the liquid portion of the food where Kledaka and the Bodhaka Kapha is considered. Charaka has mentioned that the function of disintegration and softening of food substance in the Koshta is due to 'drava' and 'sneha'.

Though Kapha has not been explained having drava quality but Kapha is made up of 'Ap' dhatu and so that Kapha possess dravata, but it depends upon the ushna guna. So the task of Kledaka Kapha can be summarized as kledana, sithilikarana, mridukarana and samghata bheda. Also Bodhaka Kapha does moistening of mouth to help in speech and aids mastication. Dravata is also the quality of Pitta and kleda functions can also be attributed to dravata of Pachaka Pitta. The ideal level of dravata is ultimate for the proper digestion to take place, balanced by Pitta and Kapha.

The excessive klinnata hampers the agni directly as mentioned in the literature that, dravata ceases the agni the ideal example being Pittaja grahani. Eventhough this disease is Paittika in nature, the Pitta causes agnimandya due to increase in the drava guna, says Chakrapani, while clarifying the role of agnimandya in Paittika grahani[131].

Ingestion of any excessive ushna, tikshna and katu dravya causes too

much secretion of the mucosa, which interferes with digestion process and causes the vidagdha avastha in excess, leading to conditions such as ajirna. Similarly, increase in the Kapha dosha causes the mandagni. Hence, if the function of Kapha ceases or lessens, the insult can directly be produced due to action of agni on the mucosa, leading to conditions like gastritis and even that of gastric ulcer. Hence, a very delicate balance of the responsible factors is required for proper digestion.

iv) Sneha: Usually ahara consists of the property of sneha which is the most familiar or the guna which is most satmya to the human. Kapha is also having the property of Sneha, it also belongs to Ap mahabhuta which is been described, possessing a specific quality of ap. Pitta is also having sneha guna but not to the extent of Kapha[132]. Hence, it can be said that, sneha is also the quality of Kledaka Kapha and also the Pachaka Pitta. Sneha performs the function of mardava of ahara. Ultimately it helps in the proper mastication and churning by stomach musculature, so that proper digestion and the transformation takes place.

The decrease in the quality of sneha damages the intestinal mucosa due to ruksha guna of the various food materials. Hence, sneha guna also performs the protective effect to the stomach musculature during the mechanism of digestion, which is attributed to Kledaka. The similar thinking comes as the bicarbonate mucosal layer protecting the mucosa form the tides of acid secretion. Decrease of sneha in the stomach leads to provocation of Samana Vayu which causes imbalance of agni, infact leading to diseases. Nowadays people are having numerous food items which adds to rookshata of koshta affecting the functioning.

v) Kala: This is an important factor for every process to accomplish which starts with the time of ingestion of food. Kala means mainly, the time

required for the digestion of the ingested food. Time required for the proper secretion of all the digestive components and for proper digestion and absorption, is also a matter of concern here. But other consideration of the kala are also necessary for proper digestion and absorption of food i.e. the kala or time period of kshut, trishna, dosha, rithu, chinta and also charvana. The normal status of agni is having the diurnal variation and the seasonal variation.[133] That is the reason of the seasonal purification methods mentioned with rithucharaya. Like wise is the case of drug administration, the virechana drug is administered at the time after the Kaphakala, for achieving the optimum action. Like wise, the sodhana sneha should be administered before the feeling of appetite.

The subsequent food is to be taken after the proper digestion of the previous meal. The meals we had without proper digestion of the previous one is termed adhyasana and this untimely food leads to improper digestion and also mixing of the undigested and semidigested food causing amadosha, leading to agnidushti. Emptying of the stomach requires certain instance, liquids empty rapidly than the solids, in a usual manner. Timing of transit of food material in the intestine is regulated by Vatha. Any disturbance to Vatha disturbs the dharana and munchana period leading to improper digestion and absorption, leading further to provocation of the doshas and agni. Also the prakrithi, koshta status as well as the status of the psyche of the individual, is also having a role in the intestinal movements. That is the basic difference in the individuals by differing in koshta, prakrithi etc.[134] Excessive dharana of acidified anna causes damage to the duodenal mucosa, as the ahara turns vidahi. Adhyashana and ajirna-bhojana causes prakopa of all the three doshas simultaneously, leading to agnidushti[135].

vi) Samyoga

Equilibrium of all above factors are necessary for the proper digestion. Ashtavidha ahara ayatana should be considered and followed always, to avoid the aetiological factors for agnidushti. Charaka has specified a judgment on the various aspects of the qualities of food materials, which is obvious from the fact that, most of the diseases have a long list of etiological factors from the dietary habits and also the contents of the diet. Acharya has formulated guidelines for a healthy diet selection and at the same time framed the rules for healthy eating, for maintaining the health[136]. Therefore the ashtavidha ahara ayatana and the ahara vidhi vidhana should be considered in a serious nature, so that the agni vaishamya and vitiation of doshas may not take place.

vii) Ashtavidha Aharavidhi Visheshayatana

Acharya Charaka suggests a few factors to be considered or assessed while selecting the food. The hitatwa as well as the ahitatwa has been explained by Chakrapani in the explanation. Such factors are being explained in detail by the Acharya in the Vimanasthana indicating that, an ideal food selection is the prime factor supportive for the digestion which is the reason behind the variation in diet from place to place.

a. **Prakriti** (Natural Qualities) – Before ingestion, the natural properties of food must be considered so that these may not hamper the agni as well as the doshas. Here, prakrithi means the natural qualities of a substance prior to any samskara or processing. For example, among the dhanya, masha is guru, while the mudga is laghu by prakrithi, says Chakrapani[137]. The difference in the gunas of the food may have different impact on the the agni as well as the doshas, during the process of digestion.

b. **Karana** (Preparation) – Various preparation procedures increase, decrease or rather alter the properties of the food stuffs which is due to admixture of water, heating and predominance of time as well as the season. Also the other factors ie. desha, kala and bhajana must be considered, during the instant of preparation process. Chakrapani has specified several examples for the same. The dhanya such as saali becomes more laghu on samyoga with agni. Dadhi is usually sophajanana, but will act in reducing sopha, after mandhana. The karana factor has to be dealt with before the intake of a particular type of food, along with its prakrithi.

c. **Samyoga** (Combination) – The combinations of dravyas vary much in accomplishment from the individual properties, mentions Charaka samhitha. For eg. the madhua and sarpi which is very excellent and supportive to the body becomes harmful, as a combination. Acharya has discussed eighteen types of virudha in the diet, which is not compatible to the body[138]. These combinations in one way or other hampers the process of digestion, in the due course. If the same offence is repeated, it may lead to the manifestation of several disorders or diseases itself. The primary location may be in the digestive tract itself.

Acharya has also enlisted the various diseases manifested, including the Amlapitta and grahani roga, resulting from the ingestion of the virudhahara. So, for avoiding an insult for agni, one has to take care to avoid the combinations of virudha, mentioned in the classics. The mentioned list can be taken only as examples as the dietary habits have been changed, to a greater extend.

d. **Rasi** (Quantum) – It refers to the overall quality of food as well as that of the various constituents of the diet. Everbody must eat the required quality

of food, which may directly interfere with the gastric juice secretion and the digestion. Atimatra and heena matra bhojana leads to impaired digestion, resulting in vitiation of the doshas. Not only vitiation, but the heena matra bhojana may result in many diseases arising, as a result of undernutrition and the resultant depletion of dhatus, as well as the Vatha kopa. The ideal matra suitable for the body has to be promoted, so as to maintain the healthy status of the GI tract.

e. **Desha** (Habitat) – This denotes the place relating to growth as well distribution of a substance. The different desas vary in the dosha status as well. Really the quality or guna of the food stuff varies depending on the habitat, in which the plant grows. Such alteration in the climate and the soil seems to affect the chemical constituents of the plant. So the same food stuff collected from different habitat varies in the guna, slightly. This may also directly affect the status of the agni and hence digestion. So as the desa or sthana of those who are intaking the food is also included here and also has to be considered.

f. **Kala** (Time factor) – Here both nityaga and avasthika kala should be considered. Nityaga is the diurnal variation and avasthika is the variation as per the age of the the patient, according to Chakrapani.[139] Seasonal dietetic variation, age wise, diurnal and disease wise variation are all to be considered here. These all may have an impact on the digestive mechanism and functions. There is more chance for a child to have Kaphaja vikara as per the characteristics of his age and also an aged person to have a Vatha vyadhi. We have to consider these factors, while administering the routine food.

g. **Upayoga Sanstha** (rules of use) – Mainly two factors are under consideration here. One is the policy mentioned while having food like naatidrutam,

naati vilambitam, tanmana etc. ie. the nature of food intake, which also have to kept in our mind. The pace of intake, the method of chewing, the status of manas while administering the food, all these are having a role to play in the ongoing digestive process.The other factor is to be kept in mind, is the jeerna lakshana of ahara mentioned. These have to be taken into account while moving on for next food. Ajeernabhojana leads to excessive vridhi of all three doshas, says the Acharya.

h. **Upayokta** – means one who consumes the food ie. the user. Diet varies from person to person according to their physical compatibility and habits. One has to consider all the factors described above to have an ideal food habit. One who consumes the food keeping in mind, all the above said factors, is the real upayokta, opines Charaka[140]. He will have an appropriate digestion and is not much vulnerable to diseases of the GI tract. Such an importance has been given by the Acharyas, regarding the intake of food. The concept of Okasatmya is also discussed in the context as the pathya as well as apathya will become suitable for a pecuiliar person, on constant and continous use.

Aharavidhi vidhana

Charaka has also prescribed the code of healthy eating, after describing the basis for the selection of a healthy diet which is being discussed below[141].

i) **Ushnamashniyat** – It enhances the taste, favours agni, makes food easily digestible and does anulomana of Vatha and results in the decrease of the Kapha.

ii) **Snigdhamashniyat** - It diminishes the rookshata of ingested food and regulates the Vayu, makes digestion easy, enhances bala, varna, prasada and also stimulates the indriyas.

iii) **Matravadashniyat** – Quantity is related with the status of the agni of the individual. Such a diet digests and eliminates from the body without altering the three doshas, says Chakrapani. Decline and excess in the qualities of food create the disturbance in agni and results in impairing the normal digestive process.

iv) **Jirne ashniyat** – Ingestion of food before digestion of the previous meal causes the vitiation of agni and all the three doshas. The transforming ahara rasa gets mixed with the subsequent intaken food, leading to disturbance in functioning of the doshas. Hence a meal should be taken only after digestion of the previous meal. It results in the timely nutrition and maintenance of the dhatus. Many current gastric problems are due to the repeated intake of several types of food, without even considering the jeernata of the previous one.

v) **Viryaviruddham ashniyat** – Intake of virya viruddha dravyas cause tridosha prokopa and must be avoided.

vi–xi) **Ishta desha, Ishta sarvopakarana, Natidruta, Nativilambita, Tanmana.**

The food has to be taken from one's favourite place or atmosphere. We must satisfy all our requirements, during the intake. The food has to be properly chewed and swallowed ie. must be taken not too fast, neither too slow. We have to concentrate on the food and not on anything else. The involvement of mind in the food intake and its absorption in the body has to be taken very seriously. Today's fast life is not allowing a food intake with the involvement of mind, due to the technological advancements and all. The stress and similar components are

proved as affecting the metabolism and leading to conditions like dyslipidaemia, still they are having food with high nutritious value.

Bhunjita Ajalpannahasan – The habits of talking, laughing etc. are also not acceptable while having the food in a proper manner which is the key thing that allows proper chewing of food and mixing with the saliva.

xii) **Atmanam Abhisamiksha bhunjita** – Every user must consider his self well being of psyche as well as soma, while preferring his dietary habits, so that he may follow mentioned dietary rules.

The food administered in such an atmosphere provides some sort of positive energy to the body as well as the mind. In modern life, the least cared thing is the nature or the atmosphere of the food intake. We are eating, while we are doing something else ie. we are not concentrating on the food which is against the methodology, mentioned seriously by our acharyas[142].

All the above factors cause proper digestion of food by which there is no chance of vitiation of agni and hence lesser chance of getting diseases like Amlapitta. All the above factors are responsible for proper secretion of the gastric juice and normal digestion. If any factor causes vitiation of Pachaka Pitta or Samana Vayu, it leads to agni dushti.

Sushruta has also prescribed the code of behaviour after a meal. He advised walking at least a hundred steps, after the food intake. There after taking rest in sitting position for a while and then lying supine in left lateral posture, is ideal. This provides proper instance for digestion[143]. This seems very much practical nowadays, with the hectic life schedule of the majority of the individuals. The new style of occupation sitting infront of the computer, after taking hyper nutritious

food along with the lack of exercise, is the contributory source behind most of the lifestyle diseases.

Samanya nidana for the diseases due to agnidushti

Acharya Charaka is narrating the mechanism of digestion and absorption very effectively, while explaining the chikitsa of grahani. This is because grahani is such a condition where, the proper transformations of dhatu is affected. Chakrapani says that by the term Grahani, the doshas of agni located in the grahani, is being explained.[144] Here the transformation and metabolism of the dhatus are dealt with seriously also with the stage of metabolism of the food.The etiological factors for agnidushti are mentioned thereafter.

Table 1 Samanya nidana for Agnidushti

Dietary habits	Abhojana, ajeerna bhojana, atibhojana, vishamasana
Alteration in food quality	Excess of guru, seeta, rooksha bhojana, dushta bhojana
Unused	Asatmya bhojana
Iatrogenic	Snehana, sodhana vibrama or unideal paschat karma
Convalescent	Karsana resulting from other vyadhis
Seasonal	Alteration in the desa, kala, rithu
Vega	Dharana of the vegas

These all leads to alteration in the functioning of agni, altered digestive mechanisms and the resultant disorders like ajeerna or in the later stages, diseases such as Amlapitta.[145] The resulting ajeerna or amadosha is acting as a toxin or the causative factor, for the a choice of resultant diseases. If it does not gets subsided at that level, the pathogenesis progresses further. The ama or toxin when associated

with Pitta, results in conditions like daha, trishna, mukha roga, Amlapitta etc. If the associative doshavridhi is that of Kapha, it leads to peenasa, meha, rajayakshma etc. If there is associated Vathavridhi, the result is the manifestation of Vathavyadhis. If the amavisha associates with dhatu dhushti, it may result in rogas affecting that pecuiliar dhatu. The associative factor, is the deciding point in the pathogenesis of such diseases.[146]

Thus Acharya Charaka has described two important areas, along with this discussion. Primarily, all the diseases will be having a basic level of doshadushti, originating in the koshta. Secondly, the associative dosha dushti as well as the involved dhatu, is the deciding factor of the disease to be manifested. Also it is to be projected that the dietary factors will not only contribute to digestive disorders like Amlapitta, but also to systemic diseases like prameha or rajayakshma.The initial stage of many a diseases, commences from the koshta itself. For eg. in swasa, one of the initial things happening or the basic level of samprapthi is the Vatha kopa, in the amasaya.

Nidana of Amlapitta

Nidana is the sum total of all the causative factors of the disease. They include the contributory factors, to the development of the condition, at all levels. They may definitely aid the management of the disease, by preventing further progress and also the reversal of the pathogenesis. Acharya Sushruta has assumed that, nidana parivarjana is the first line of treatment of any disease[147]. For the same, a definite knowledge of the etiological factors has to be dealt with.

After a careful screening and analysis of the etiological factors of Amlapitta, discussed under the various texts, it may be better to classify the same

into groups, for the expediency of understanding. Various types of etiological factors have been described, in the reference of the diseases of annavaha srotus and purishavaha srotus.

Aharaja Hetu

The first and the foremost group of the etiological factors of Amlapitta may be considered as the dietary factors. Under this group, the intake of food against the code of dietetics i.e. ahara vidhi vidhana and ahara vidhi viseshayatana is included. Various types of incompatible substances, excess use of Pitta aggravating factors like katu, amla, vidahi etc. bharjitanna and untimely consumption are the factors against the dietetic code and they directly disturbs the equilibrium of all the doshas, mainly the Pitta dosha.

Intake of food which is processed by several methods is the custom of the modern community. Refrigerating and reusing of food materials and also the fast foods is also a routine habit now, and is the part of the lifestyle. The use of soft drinks is also creating some sort of mutilation, to the stomach. The increase in consumption of bakery items and smoked items is also not gracious to the stomach. The addictions like smoking and alcohol also definitely disturbs the stomach physiology. Kasyapa advises to consume food materials which are not processed in the dietary schedule of an Amlapitta person, as a part of its management[148].

Viharaja Hetu

To keep the health undisturbed, one is required to follow the code of healthy habits. An individual requires to have the regular habit of defecation, to eat properly and to sleep on time. One must not supress the natural urges, maintaining the equilibrium of the body constituents and by that obviously, he

would maintain good health and proper functioning of the body. If this is not followed regularly, the whole functioning of the body will be disturbed and in the long run, they will cause disturbance to the equilibrium of Pitta and digestion, which ultimately result in conditions including Amlapitta.

The modern life style has affected the above said stuff, very negatively. The habit of exercise is becoming less and less. Also the life style is increasing in the pace as well. Nobody is satisfied with their own pace. The primary affected by all these is the GI system along with its functions.

Manasika Hetu

Psychology also plays an immense role in maintaining the health and physiological activities. While one is with an imbalance in psyche, the primary function that gets impaired is the mechanism of digestion and its components.

Any abnormal psychology in terms of anger, anxiety, greed etc. affects the physiology of digestion and also the functions of Vatha, the regulator among the doshas. Either there would be a lesser secretion of the digestive juice or secreted at improper times and sometimes a hyper secretion. All these conditions lead to indigestion which ultimately gives rise to conditions like Amlapitta. Besides, the increased pace in the peristaltic movements also alters the absorptive aspects of the digestion.

The modern medicine has established that gastritis is resulting from the stress and strain which shows the imperative role played by the psychogenic factors, in the production of diseases like Amlapitta.[149] The psychic factors affects the abdomen in altering the release of chemicals and hormones and also affecting the motility of the gut. The enteric nervous system supplying the gut is having as

much neurons as in the brain and spinal cord, as per latest studies. The functioning of the gut is affected by the variation in the neurotransmitter function. This is the reason of the high influence of psychic factors in the functioning of the GI tract.

Agantuja hetu (Miscellaneous Factors)

Allied factors can be taken under this factor. Under this group constant and excessive consumption of alcohol, tobacco, beverages, soft drinks, smoking or other irritant substances are taken. These substances cause a local irritation in the stomach which in turn secretes further gastric juice. Also the excessive use of gastro irritant drugs like NSAID's, corticosteroids etc are also a real contributors.

Krimi

Krimi denotes the various microorganisms in the body. While explaining krimi, Chakrapani comments that of the several krimis present in the human body, many are helpful or supportive to the bodily functions. The mentioned names in the text are of the pathological krimis and the non pathological or physiological are not mentioned.[150] But the keen observation of the Ayurvedic ancestors at their period without microscopes is very much appreciable, from this information and at a very later stage was evolved, the concept of probiotics.

The twenty types of krimi mentioned are of the pathological ones, seen both externally and internally. One of the sites mentioned for the localization of krimi, is in the amasaya. The amasayagata krimi causes symptoms such as hrillasa, asyasravana, avipaka, aruchi, chardi, anaha etc. which resembles very much with the gastric conditions like dyspepsia which gives us an initial hint regarding the contribution of krimi in the same.[151]

Even though the microorganisms like Helicobacter pylori were not known to our Ayurvedic ancestors, they evolved the theory of localization of krimi in amasaya and the resultant symptoms, resembling the dyspepsic condition. We have to think of incorporating the drugs with krimihara in action in the management of conditions like Amlapitta, resulting from krimi, along with the much needed sodhana therapy, so as to eradicate the organisms like H pylori.

The Acharyas Kashyapa, Harita, Madhavakara have described the etiological factors of Amlapitta, but the list of Kashyapa is minute. Madhavakara has given few etiological factors which are mainly causing the Pitta prakopa. Kashyapa has mentioned such etiological factors of Amlapitta, which are Kapha Prakopaka in nature – this shows his inclination towards the role of Kapha as the chief causative factor.[152] The importance of amasaya as sthanas for both the Kapha and Pitta is also stressed on, by Kashyapa while explaining the Amlapitta. All scholars have mentioned viruddhahara as one of the causative factor of Amlapitta, which points to the importance of it in the pathology.

TABLE 2 : ETIOLOGICAL FACTORS OF AMLAPITTA

No.	Etiological factor	K S	M N	B P	B R	S N
1	Adhyasana	+	-	-	-	-
2	Abhishyandi aahara	+	-	-	-	-
3	Ajeerna	+	-	-	-	-
4	Aama	+	-	-	-	-
5	Aama pakwanna	+	-	-	-	-
6	Akaala bhojana	+	-	-	-	-
7	Amlasevana	+	+	+	+	+
8	Ajeerna Aahara	+	-	-	-	-
9	Antarodaka Pana	+	-	-	-	-
10	Atyushna aahara	+	+	+	+	+

No.	Etiological factor	K S	M N	B P	B R	S N
11	Ati snigdha ahara	+	-	-	-	-
12	Ati rooksha ahara	+	-	-	-	-
13	Ati drava sevana	+	-	-	-	-
14	Atisnana	+	-	-	-	-
15	Avagaha	+	-	-	-	-
16	Buktwa buktwa	+	-	-	-	-
17	Brishta dhanya	+	-	-	-	-
18	Dushtanna	-	+	+	+	+
19	Divaswapna	+	-	-	-	-
20	Gorasa	+	-	-	-	-
21	Guru bhojana	+	-	-	-	-
22	Ikshu vikara	+	-	-	-	-
23	Kulatha sevana	+	-	-	-	-
24	Madya	+	+	+	+	+
25	Paryushita anna	+	-	-	-	-
26	Pitta prakopi annapana	-	+	+	+	+
27	Pishta anna	+	-	-	-	-
28	Virudhasana	+	+	+	+	+

TABLE 3 Aharaja and Manasika Hetus

Aharaja Hetu[153]	Manasika Hetu [154]
Viruddha bhojana	Kama
Asandusta bhojana	Krodha
Ati ruksha bhojana	Lobha
Vidahi bhojana	Irshya
Shuska sevana	Moha
Guru sevana	Shoka
Atidrava sevana	Bhaya

TABLE 4 The involved nidana and the affected dosha

Guru ahara	Kapha
Ruksha ahara	Vatha Pitta
Seeta ahara	Vatha Pitta
Shushka ahara	Vatha Pitta
Apriya ahara	Tridosha
Vishtambhi ahara	Tridosha
Vidahi	Pitta
Viruddha ahara	Tridosha
Kama	Vatha
Krodha	Pitta
Moha	Pitta
Irshya	Vatha
Shoka, Bhaya	Vatha
Udvega	Vatha

Table – 5 Nidana and the affected dosha – a comparison

Nidana	Dosha	K S	M N	B P	S N	H S	Y R
Kulattha Sevana	Pitta	+	-	-	-	-	-
Pulaka sevana	Pitta	+	-	-	-	-	-
Pruthuka sevana	Pitta	+	-	-	-	-	-
Guru ahara	Kapha	+	-	-	-	-	-
Abishyandi ahara	Pitta Kapha	+	-	-	-	-	-
Atisnigdha ahara	Pitta Kapha	+	-	-	-	-	-
Ati rooksha ahara	Vatha	-	-	+	-	-	-
Vidahi annapana	Pitta	-	+	+	-	-	+
Pishtanna sevana	Pitta Kapha	+	-	-	-	-	-
Apakwaanna sevana	Pitta Vatha	+	-	-	-	-	-

Nidana	Dosha	K S	M N	B P	S N	H S	Y R
Phanita sevana	Kapha	+	-	-	-	-	-
Ikshuvikara	Kapha	+	-	-	-	-	-
Paryushita anna	VPK	+	-	-	-	-	-
Dushtanna	VPK	-	+	+	-	-	+
Ati ushna ahara	Pitta	+	-	-	-	-	-
Virudha ahara	VPK	+	+	+	+	+	+
Ati amla ahara	Pitta Kapha	+	+	+	+	+	+
Ati tikshna ahara	Vatha Pitta	-	-	-	+	-	-
Adhyasana	Pitta Kapha	-	-	-	-	-	-
Aaama poornata	VPK	+	-	-	-	-	-
Ajeernasana	Pitta kapha	+	-	-	-	-	-
Akaala bhojana	Kapha Pitta	-	-	+	-	-	-
Kale anashana	Vatha	+	-	-	+	-	-
Vishamasana	Vatha	+	-	+	-	-	-
Vega dharana	VPK	+	-	-	-	-	-
Bhukte divaswapna	Kapha	+	-	-	-	-	-
Bukte avagahana	Vatha Kapha	+	-	-	-	-	-
Bukte snanam	Vatha Kapha	+	-	-	-	-	-
Atimadya sevana	VPK	+	-	-	-	-	-

BHEDAS OF AMLAPITTA

Amlapitta is classified according to its associative dosha lakshanas by the ancient Acharyas. Kashyapa has given the classification of Amlapitta according to symptoms from the involved doshas. Madhavakara as well as Bhavamisra has also classified the Amlapitta into two types according to the location of dusti in the srotus or the main distribution of the symptoms, whether in the upper or lower part of the mahasrotus[155].

According to the vitiated Doshas

Acharya Kashyapa has mentioned three types.

1. Vataja 2. Pittaja 3. Kaphaja

Madhavakara has explained four types[156].

1. Vatadhika 2. Kaphadhika 3. Vatakaphadhika

4. Shleshma pittaja

According to dusti sthana of the srotus

Madhavakara and Bhavamishra[157] both have described mainly two types 1. Urdhavaga Amlapitta 2. Adhoga Amlapitta

This classification is mainly based on the presentation of the symptoms suggestive of the GI tract. This classification has its own importance in the management, as far as the clinical strategy is considered.

SAMPRAPTI

In Ayurveda, samprapthi is the gross of the various stages upto the manifestation of the disaease, right from the commencement of the dosha dushti. It really explains the progressive development of the disease, including the etiological factors. Even the quality of the doshas involved in the aetiopathogenesis is also being discussed ie. because of this the five types of samprapthi being discussed, in this ancient medical science. By this methodology, it is possible to explain each and every aspect of the disease.

As long as the etiopathogenesis of GI diseases are concerned, Charaka has described, it elaborately[158]. No other scholar has mentioned it so vividly. But Charaka has not mentioned Amlapitta, as separate disease entity. The samprapti

of grahani roga mentioned by Charaka is able to explain the pathogenesis of Amlapitta, eventhough the name not mentioned as such. Ayurveda gives the emphasis on the fabrication of the disease, mainly due to mandagni.

If we come across the samprapti of Grahani Roga, it seems that it is the progressive samprapti of different diseases. The samprapti of different diseases coincide at few stages, that being the reason behind the similarity between symptoms of a few diseases. The poorvaroopa of kushta resembles vathasonitha, opines Vaghbata, due to this similarity, the disease being different.There are so many conditions which resembles Amlapitta e.g. poorvarupa of gulma, udara, grahani, roopas of arsas, pittaja pandu, pittaja hridroga, Pittavrita Vatha etc.[159] There are two main conditions from which we must differentiate the disease Amlapitta, those are vidagdhajirna and sama Pitta.[160] These are the points supportive for its differential diagnosis.

Out of which sama Pitta is the stage of the Pitta, where ama is associated. As there is no specific dosha-dushya- sammurchana undergone at that level, it cannot be demonstrated as disease. But in Amlapitta, mainly the rasadhatu is involved as dushya and sthanasamsraya is in the amashaya.

Samprapti Ghataka

Dosha	-	Pitta (Pachaka)
	-	Vatha (Samana, Prana, Apana)
	-	Kapha (Kledaka, Bodhaka)
Agni	-	Mandagni
	-	Vishamagni
Srotus	-	i. Annavaha ii. Rasavaha
		iii. Purishavaha iv. Raktavaha

Srotodushti	-	Sanga, Vimarga gamana, Atipravriti
Adhisthana	-	Amashaya, Grahani
Dushya	-	Rasa, Rakta
Vyakti	-	Amashaya, Grahani
Marga	-	Abhyantara (Kostha)
Swaroopa	-	Chirakari
Prabhava	-	Daruna

Dosha- In the pathophysiology, the involved sites of lesion and disturbance are observed to be Pachaka Pitta, Samana Vayu and Kledaka Kapha.

Samana Vatha – The seat mentioned for samana is very near to the agni and is the causative factor for grahana, pachana, vivechana and munchana, almost all the functions expected to be performed, by the GI tract. It controls all the secreting and motility functions of the two ashayas and helps in the action of digestive enzymes, assimilation of end products and their separation into various tissue elements and when vitiated, causes indigestion, diarrhoea and defective assimilation.

Pachaka Pitta- The amlaguna and dravaguna of Pachaka Pitta gets vitiated. Kledaka Kapha situated in amashaya is to counteract the withering action of Pachaka Pitta. Due to the imbalance in Pitta, the pachana kriya is also disturbed. This leads to the formation of vidagdhajeerna then to suktapaka and later to ama.The ama deprives the body of its nutrition and causes sadana.It is the root cause of all the diseases and is also the first stage or phase of dosha dushti in any disease.

When this ama is combined or impregnated with tridosha or saptadhatus or with malas, they are called sama and the diseases produced as

such are the sama rogas. This is so crucial in the management of any disease. For eg. in the management of autism in children, the ama is to be considered seriously before providing snehana or similar treatments.

TABLE 6 Symptoms of the Sama Doshas [161]

Sama Vatha	Sama Pitta	Sama Kapha
Vibandha	Durgandha	Avila
Stamba	Haritha syava	Tantula
Antrakujana	Amla	Styana
Vedana	Sthira	Pralepi
Sopha	Guru	Pichila
Nisthoda	Amlika	Kantadeshe avatishtate
Adhmana	Kanta hritdahakara	Kshududgara vighatakara
Asanchara		

These symptoms are very much useful for a clinician approaching a disease and tailoring the management. It is ideal to manage the ama initially and correct the jadaragni, then only rest of the treatment can be performed effectively. The components of the samprapthi are dealt here with in detail.

Agni – Sushruta explains that the prana, samana and apana while enduring at their original sites, maintain the agni.[162] Charaka is explaining the various aspects and merits of agni along with grahani. Agni can be considered as normal or equal (samagni) if all the components controlling it such as Samana Vatha, Pachaka Pitta and Kledaka Kapha are on the normalcy. ie. samagni seems to be the sum total of these components of dosha. The vitiation of any one of these, leads to agni dushti and vice versa.

Amashaya and grahani are the sthana of jatharagni or infact, the Pachaka Pitta[163,164]. Any type of disturbance of agni as mentioned above, starts the pathogenesis ie. i) vishamagni, ii) tikshnagni and iii) mandagni, which are said to be the resultant of the disturbances in the three doshas, respectively.

As the diseases of the mahasrotus are caused from agni dushti and all the three doshas affects the agni, the manifestation may be according to the dominance of the relevant dosha. Kashyapa Samhita is the first treatise, to explain the symptoms of Amlapitta, as per involvement of predominant associative dosha.

Srotus – Amlapitta involves the amashaya, grahani and the pakvashaya. Hence the annavaha and the purishavaha srotus seem to be mainly affected but the rasavaha srotus which receives the ama resultant from agnidushti first, gets surely involved. Regarding the types of srotodushti, the three types of disturbance of annavaha and purishavaha can be observed ie.i) Sanga ii) Atipravritti and iii) Vimarga gamana.

Dushya – While reviewing the symptoms of Amlapitta, it seems that the main dushya is rasa, as the initial dhatu to receive the ama along with the ahara rasa. The aetiological factors and symptoms are also suggestive of the rakta dushti. There is also a chance of affection of the other dhatus, if it is not intervened in a proper time or manner.

Mala – One of the important cause of the diseases in Ayurveda is vegavarodha i.e. suppression of natural urges. eg. micturition, defaecation, hunger etc. This affects the sphincteric competence and the related physiology adversely. Natural contractility and motility of the smooth muscles of the viscera, the GI tract and the macro and micro channels of the body are also affected. Once the

function of srotus gets affected, it leads to abnormality in them, leading to accumulation and later the vitiation of doshas.

The etiological factors such as abhojana, atibhojana, veganigraha, vyapats of panchakarma, seasonal variation etc. causes the vitiation of doshas altering the agni[165]. Due to the resultant avipaka, even light and small quantity of food is not digested in a proper or expected manner. This ill digested food gets shuktatva, leading to ajirna.[166]

Acharya Charaka opines that the ajirna forms the annavisha resulting in ama. This ama, when mixed with the aggravated Pitta, resulting from its own etiological factors, develops to the disease Amlapitta. Usually dravaguna and amlaguna of Pitta gets altered, causing vidagdha ajirna at the intial stage. If neglected, as suggested by Gananat sen, this will progress causing the inflammation and erosion of sleshmadhara kala of the amashaya, leading to the manifestation of diseases like Amlapitta and Parinamasoola.[167]

Shadkriyakala

Shadkriya kala was explained by the Acharyas so that, the steps of the pathogenesis, be explained in a better manner. This also helps the physician to intervene in the pathology, at any level effectively. This is very much evident in many a clinical conditions. The concept has been much seriously dealt by Susrutha, while describing the condition of vrina[168]. If a vrina has to be properly managed, it is highly essential to analyse each and every stage of the pathogenesis and must be intervened within no time. Similarly these steps give significant information, in the stage of each and every disease which is very much crucial to the physician and also the management seems different in each stage.

(1) SANCHAYA

Anupa desha, varsha ritu, vidahi annapana, excessive amla and katu rasa, dusta anna sevana etc are the causative factors, resulting in the vridhi of Pitta. This stage is sanchaya where mainly, the Pitta is on the rise, at its own site. Pitta is the principal dosha which is necessarily vitiated but the associated Kapha and Vatha dosha are also disturbed, due to further nidanas. In this stage, the treatment principle is only the nidana parivarjana and also promotion of health.

(2) PRAKOPA

With the excessive and continous use of the etiological factors, Pitta is exaggerated more and more. So, sanchita Pitta becomes prakupita and this stage is called as prakopa. This stage includes the stages of vidagdha and shukta paka of anna leading to ajirna. Amlodgara, daha, trishna, avipaka, etc. are the resulting symptoms which is according to the main dosha. Due to involvement of Pitta and Kapha there may be amlodgara, pipasa, daha, annadvesha, utklesha and hrillasa[169]. In this stage, treatment is nidana parivarjana, deepana and pachana.

(3) PRASARA

If the prakopavastha is not managed properly, the prasara stage commences. In this stage, ahara which is shukta in nature due to the agnimandya and ajirna becomes ama, circulates the whole body with the ahara rasa and through the dhamanies. So the rasa dhatu is also vitiated in this stage. This annavisha gets mixed with the Pitta dosha and circulates throughout the whole body. This is called as Sama Pitta which is of amlarasa and the stage called as the prasaravastha.

This stage differentiates ajirna like – aama, vidagdha and vishtabdha due to involvement of three doshas with visharupa anna.[170] Also observed that

manovaha srotus gets vitiated due to various mental factors causing ajirna and Amlapitta. In this stage, the main symptoms are trishna, jvara and avipaka. The treatment desirable is the nidana parivarjana, ama pachana with tikta madhura rasa along with langhana.

(4) STHANASAMSHRAYA

This is the auxilliary stage and from here the specific pathogenesis of each disease commences as per the specificity of nidanas, quality of doshas by which they are vitiated and the site of alteration in the functioning of srotus. Mixture of annavisha and Pitta dosha is retained in annavaha srotus, mainly the amashaya vitiating the srotus. Amlarasa and dravaguna of the Pitta are increased further. According to Kashyapa, Kapha has an important role in Amlapitta[171]. This vitiated kapha also is staying in the Pittadhara kala of the amasaya and hence Pittadhara kala of the amashaya is vitiated. The symptoms manifested in this stage may be same as of the Amlapitta but with less in severity. In this stage, the management needed mainly is sodhana karma with ama pachana and agni deepana.

This is the stage from where the vidagdhajirna can be separated from the Amlapitta. Vidagdhajirna is an acute stage occurring due to the causative factors, directly. The nidana is sapeksha, which means after the mithyaahara vihara leading to Pitta kopa leads to vidagdhajirna, but here the doshas have not established their affinity with any organ on tissue and only langhana or kala will cure the condition, but the symptoms may be produced again and again whenever, the mithyaahara and vihara is administered. But due to the repeated provocation, the doshas establish their affinity in amashaya and grahani ie. sthanasamshraya. After this stage even the laghu and alpa bhojana causes shuktatva and vidagdhata

to the annapana, leading to the production of Amlapitta and hence the relapse.

Diagram 1
Samanya Samprapthi of Amlapitta

(5) VYAKTI

In the sthanasamsraya stage, pittadhara kala or amasaya gets vitiated by the sama Pitta. After this stage, if the vitiated Pitta gets the appropriate alteration of the functioning of srotus, process gets commenced. Pitta eventhough drava is acting as agni, when it is not atidrava and not atiamla. This type of Pitta acts as tyakta drava in the form of agni, digesting whatever the food is taken. But in this condition, Pittadhara kala is vitiated by sama Pitta and so the Pachaka Pitta is produced, which has elevated degree of amlata and dravatha. So the normal action of Pitta is diminished, i.e. it acts as vykta drava, giving rise to avipaka and stagnation of food leading to fermentation. Due to this condition, all the symptoms and signs of Amlapitta are created. In this stage, the treatment advisable is the sodhana karma,preferably vamana, ama pachana without ushna and teekshna drugs and also agni deepana[172].

(6) BHEDA

Bhedavastha is the final stage of any disease. If Amlapitta is not managed in its vyaktavastha, it proceeds to the bheda stage as well. Due to the high degree of amlata and dravata of Pitta, mandagni results. More and more vitiated rasa dhatu also leads to aggravate the agnimandya. Hence annavisha is produced more, which vitiates the Pittadhara kala accordingly. So the symptoms and signs are produced in a severe manner and also lead to the manifestation of other diseases of GI tract such as parinamasoola and gulma. The ahara, even taken with the proper quality and quantity cannot be digested in this stage, ultimately leading to dhatukshaya and jvara, sopha, pandu etc. develops as the upadravas[173]. The disease goes into an incurable state with the complications.

From the management point of view, we must differentiate these stages and the diseases, which amends the line of treatment.

Kashyapa has described samprapti of Amlapitta in detail and it seems similar to samprapti of grahani Roga, described by Charaka[174]. Chakrapani commented so as to describe the whole itinerary of action[175]. Madhava mentioned the involvement of only one dosha ie. Pitta, but Kashyapa has given involvement of three doshas by the word "Vatadyaha," with the dominance of Pitta.

Srikanthadatta has clarified that like the symptom of Kotha, the causative factors of Amlapitta are also Kapha and Pitta. Acharya advised that gourava, udgara and kampa are due to involvement of Kapha and Vatha respectively.[176] Hence from the above discussion, it is clear that Kapha and Pitta i.e. Kledaka Kapha and Pachaka Pitta are the main doshas. But at the same time, we know that inverse relation of Pachaka Pitta and Samana Vayu, which always

work in association with agni. Hence, it can be concluded that there is involvement of tridosha, but with the dominancy of Pitta and Kapha in Amlapitta[177].

In the pathogenesis of Amlapitta, it can be categorized as mainly Pitta on the foremost place, Kapha on subsequent place and the least involved is the Vatha, but the status can amend, with the chronicity of the condition. On becoming chronic, the condition from the Pitta Kapha stage becomes more and more Vatha Pittaja in nature.The condition Pittavrita Prana stay near to the symptoms of the Amlapitta. Hence it seems that the Amlapitta is a disease condition produced due to Pitta Kaphavrita Vatha, mainly Prana and Samana[178,179,180] In brief, some sort of avarana pathologies in the koshta results in the symptoms of Amlapitta. Like wise, with the samprapthi of parinamasoola, Madhava is explaining one of the reasons of the Vathakopa in the amasaya, as the avarana caused to the gati of Vatha, by the other two doshas ie. the Pitta and Kapha.[181] It seems that, the obstruction to the gati of Vatha not only in the sakha, but also in the koshta, is resulting the Vatha kopa leading to diseases. Similar pathology is explained by Acharya Vaghbata, while describing the samprapthi of gulma[182]. Kashyapa describes that this disease manifests mostly in the persons having the jihva laulya ie. lack of control over their dietary habits[183].

Patients generally are aware of the etiological factor of the diseases, but due to greed or lack of control over their mind, they go on with consuming and this progress to the krichraasadhya stage and it manifests as the upadravas, like Parinama soola. This also indicates the relation between the food and the manifestation. The drastic changes in the food habits, is providing more gravity to this matter, in the occurrence of such diseases.

Madhavakara has explained two types of Amlapitta i.e. adhoga and urdhvaga. Doshika varieties are also mentioned by the various Acharyas and the differentiation in this type, is a complex task. This is also supported by Madhavakara by mentioning this as bhishak mohakara vyadhi or confusing to the physician.[184] The adhoga type is very difficult to diagnose or differentiate, as it coincides with the Pitta atisara as well as Pitta grahani, in the presentation. Amlapitta is a disease with a direct relationship with the GI tract and mithya ahara and vihara are the chief causes. The etiological factors are further are classified into two groups. The first group includes those factors, which are responsible for agnimandya and the second group include the factors, those alters the dravata of Pitta and causes its pathological vridhi or variation in its quality.

Two additional components which aggravate the Pitta, anupa desha[185] and the varsha ritu, have specifically been observed and mentioned by Kashyapa and Madhavakara respectively[186]. Both these factors vitiate the Pitta as well as the jatharagni leading to agnimandya. As a result of above stated causes, any of the dosha vitiated leads to agnimandya. In this state of mandagni, whatsoever food material is consumed by an unwise person, become vidagdha and are converted to shukta form, This vidagdha and the vitiated Pitta later manifests in the form of the disease, Amlapitta[187].

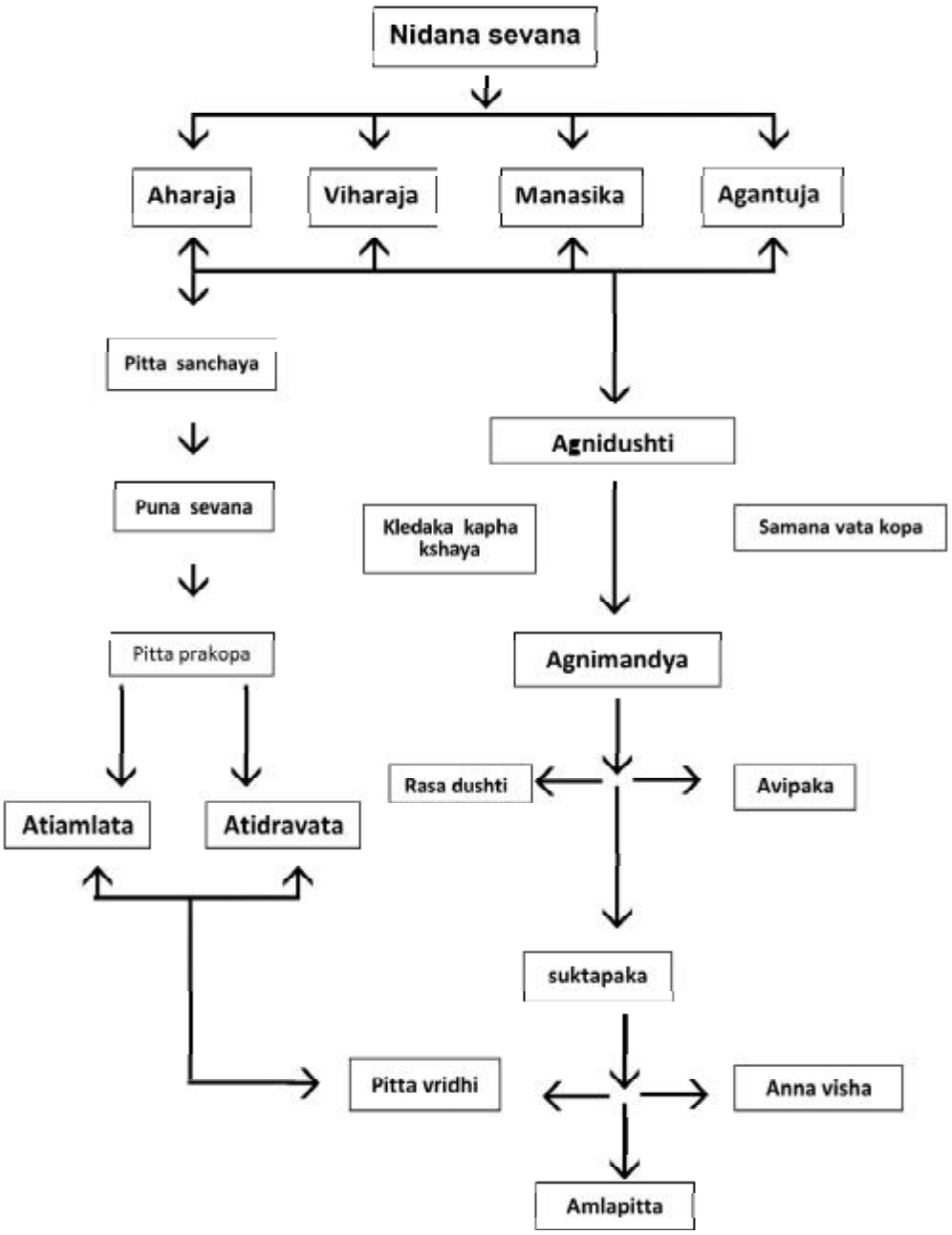


Diagram 2

Detailed Samprapthi of Amlapitta

SANKHYA SAMPRAPTI

A. Two types according to the Gati

1) Urdhvaga 2) Adhoga

B. Three types according to Kashyapa

1) Vatolbana 2) Pittolbana

3) Kapholbana

C. Fout types according to Madhavakara

1) Vatha 2) Vata-Kapha

3) Kapha 4) Sleshma Pitta[188]

VIDHI SAMPRAPTI

a) 1) Nija 2) Agantu

b) 1) Svatantra 2) Paratantra

According to the curability of the disease

Nava – Curable

Chirottha – Krichrasadhya or yapya

Purana – Asadhya

VIKALPA SAMPRAPTI

is the amsansha kalpana or categorisation of the aggravated dosha.

(1) Vata - Chala and rookhsa guna vriddhi

(2) Pitta - Drava, amla, usma, teekshna guna is increased

(3) Kapha - Guru guna vriddhi

PRADHANYA SAMPRAPTI

Independence or dependence of the aggravated dosha

(i) Pitta – Vriddhatama (ii) Kapha – Vriddhatara

(iii) Vata - Vriddha.

BALA AND KALA VISHESHA SAMPRAPTI

Bala samprapthi - cause of the disease, purvaroopa, bala of the roopa and the curative nature of the disease is acknowledged.

Kala samprapthi - By knowing the relation of the doshas with the ritukala, ahara, vihara etc. we can be acquainted with the chief caustive dosha.

Seasonal aggravation - In varsha and sarad ritu, the variation depending on the dosha.

Day / Night - Noon and mid night ie: the diurnal variation

Food time - Bhojanottara

Many conditions like gastro esophageal reflux disease, hyperacidity, gastritis, gastric atrophy, gastric ulcer, gastric carcinoma etc. can be included in its various stages under the condition of Amlapitta and managed accordingly.

PURVARUPA

Purvarupa is the manifestation observed, when the vitiated doshas gets localized in a pecuiliar organ, area or the srotus. It is interesting that some of the diseases like amavatha, visarpa, vidradhi, vatavyadhi etc. are mentioned without an appropriate poorvaroopa.[189] Purvarupa or premonitory symptoms of Amlapitta is not mentioned, in any of the classical texts. But while going through the patient history, the symptoms of ajeerna are observed. In those diseases, in which the exact purvarupa is not mentioned, the avyakta form of roopa is usually considered, says Charaka.[190] Chakrapani opines that, in such conditions, the atmaroopa of the involved doshas will be manifested, in the premature stage. This is mentioned in the diseases, Vatha vyadhi and kshata ksheena. The cardinal symptoms or roopa

mentioned at a milder level may be considered here, as the purvarupa of Amlapitta.

A few symptoms of Ajirna and especially the vidagdhajirna, may be appearing before the manifestation of the Amlapitta and is considered as the purvarupa, says Gananath Sen. Charaka has mentioned a few purvarupas of grahani like trishna, alasya, loss of bala, anna vidaha, and heaviness of body[191]. These symptoms are considered as the purvarupa of grahani as well as of Amlapitta, due to the similarity in findings. In some of the cases, the poorvaroopas mentioned for chardi such as utklesa, praseka, aruchi etc, are also being noticed.[192] Besides, the presentation varies due to the drastic variation, in the dietary habits.

RUPA

Rupa is manifested after the exact localization of the doshas has taken place and gives a clear indication of the doshas involved, in the corresponding pathology. Rupa is specific for each and every disease and helps the diagnosis of that particular condition.The analysis of the classical references related to Amlapitta will reveal that a big list of symptoms of Amlapitta can be prepared according to different sources. Rupa or lakshana are useful for the clinical knowledge of a disease as well as diagnosis. For having a uniform diagnosis of the disease allover, the diagnostic criteria have been framed as in ICD. The general symptoms of Amlapitta described by Madhavakara are as follows[193]. Kashyapa has given a few symptoms in addition to the same.

Table 7 Symptoms of Amlapitta

Madhavakara		Kasyapa
Avipaka	Tikta amla udgara	Antrakujana
Gourava	Hrit daha	Udara adhmana
Utklesha	Kanta daha	Vidbedha
Aruchi	Klama	Hritshula

Symptoms of Amlapitta have been described by Kashyapa, Madhavakara and Harita. There after all the scholars have followed the same.

Table 8 Symptoms of Amlapitta by various authors - comparison

Symptom	K S	H S	MN	B P	YR	S N
Avipaka	-	-	+	+	+	-
Amla utklesha	+	-	-	-	-	-
Amla udgara	-	-	+	+	+	+
Amla hikka	-	+	-	-	-	-
Angasada	+	-	-	-	-	-
Antrakujana	+	-	-	-	-	-
Aruchi	-	-	+	+	+	-
Dahayukta atisara	-	-	-	-	-	+
Gourava	-	-	+	+	+	-
Guru koshtata	+	-	-	-	-	-
Hritsula	+	-	-	-	-	-
Hrit daha	-	-	+	+	+	+

Symptom	K S	H S	MN	B P	YR	S N
Kanta daha	+	-	+	+	+	+
Klama	-	-	+	+	+	+
Romaharsha	+	-	-	-	-	-
Siroruja	+	-	-	-	-	+
Tiktodgara	-	-	+	+	+	-
Tiktasyata	-	-	-	-	-	+
Udara admana	+	-	-	-	-	-
Utklesa	-	-	+	+	+	-
Urovidaha	-	-	-	-	-	+
Vanti	+	-	-	-	-	-

Table 9 Symptoms of Ekadoshaja Amlapitta

Vathika	Paittika	Kaphaja
Angasada	Brama	Chardi
Jrimba	Seeta upasaya	Guruta
Shula	Swadu upasaya	Ruksha upasaya
Snigda upasaya	Vidaha	Ushna upasaya

Table 10 Symptoms of Vathika Amlapitta - comparison

Symptom	M N	B P	Y R	S N
Bhrama	+	+	+	+
Chimchimatwa	+	+	+	+
Gatrasada	+	+	+	+
Harsha	+	+	+	+
Kampa	+	+	+	+

Symptom	M N	B P	Y R	S N
Murcha	+	+	+	+
Pralapa	+	+	+	+
Shula	+	+	+	+
Tamo darsana	+	+	+	+
Moha	+	+	+	+

Table 11 Symptoms of Kaphaja Amlapitta - comparison

Symptom	M N	B P	Y R	S N
Aruchi	+	+	+	+
Agnimandya	-	-	-	+
Gourava	+	+	+	+
Jadyata	+	+	+	+
Kandu	-	-	-	+
Kapha nishtivana	+	+	+	+
Nidra	-	-	-	+
Sada	+	+	+	+
Sheeta	+	+	+	+
Vami	+	+	+	+

Table 12 Vishishta Lakshana of Urdhvaga Amlapitta

Symptoms	K S	H S	M N	B P	Y R
Abukte vami	-	-	+	+	+
Abukte va tikta vami	-	-	+	+	+
Abukte tikta udgara	-	-	+	+	+
Abukte amla vami	-	-	+	+	+
Bukte vidagdha tikta vami	-	-	+	+	+
Bukte vidagdha amla vami	-	-	+	+	+

Symptoms	K S	H S	M N	B P	Y R
Kara, Carana, hrit, kukshi, Kanta daha	-	-	+	+	+
Kandu	-	-	+	+	+
Mantala	-	-	+	+	+
Mahati aruchi	-	-	+	+	+
Pidaka	-	-	+	+	+
Siro ruja	-	-	+	+	+
Ushnata	-	-	+	+	+
Vanta harita, neela, peeta, krishna	-	-	+	+	+
Vanta arakta, raktabha	-	-	+	+	+
Vanta ativamla	-	-	+	+	+
Vanta atipichila, atiacha	-	-	+	+	+

Table 13 Vishishta lakshana of adhoga Amlapitta

Symptoms	K S	H S	B P	M N	S N
Anala sada	-	+	+	+	-
Anga pitata	-	+	+	+	-
Brama	-	+	+	+	-
Daha	-	+	+	+	-
Harsha	-	+	+	+	-
Hrillasa	-	+	+	+	-
Murcha	-	+	+	+	-
Moha	-	+	+	+	-
Trit	-	+	+	+	-
Sweda	-	+	+	+	-
Kotha	-	+	+	+	-

Analysis of the symptoms of Amlapitta

(1) AMLA / TIKTA UDGARA

The commonest symptom of Amlapitta is amlodgara, tiktodgara or both which is due to vitiated Pitta dosha. Normal pitta is mentioned to have katu rasa. But when it becomes vidagdha, katu rasa is transformed to amla. The amla guna and dravaguna of Pitta are also increased reulting in agnimandhya and ajirna. The ahara becomes vidagdha in amashaya and the amla/tikta udgara is the result.

(2) HRIT - KANTHA DAHA

The subject with the Amlapitta feels burning sensation in the throat, heart region as well as the abdomen. Sometimes the whole body, palms and soles are also affected which depends on the extent of the Pitta kopa. The vitiated Pitta leads to agnimandhya, ajirna and ama. This is one of the distressing symptoms and seen in gastritis and also in GERD. In the initial stages, the Pitta vridhi is in koshta but in the later stages, it also happens in the sakha as well, which results in the burning sensation of the extremities.

(3) AVIPAKA

Mandagni and ajirna are the core causes of Amlapitta. Charaka explains that annavisha is produced from the resultant ajirna because of agnimandya. It is mixed with the Pitta dosha and creates the disease Amlapitta. Avipaka is due to the reduction in the performance of agni. There is a feeling that the food after intake is not actually digested or transformed which causes discomfort to the person and may affect the subsequent food intake as well.

(4) KLAMA, ANGASADA AND GAURAVA

It means fatigue, lassitude and heaviness in the body respectively. They are much related terms and the person gets tired without any unusual exercise or strain and is termed as klama. One will have a decreased energy level as well as disinterest in their routine activities. All these symptoms occur due to the rise or accumulation of the amadosha in the body, which affects all the functions. Many GI conditions like gastritis, liver diseases etc. causes the feeling of fatigue in the individual. It is one of the early presentations of jaundice, before icterus. These symptoms are also observed in conditions like hypothyroidism, wherealso the BMR seems affected. These symptoms point to accumulation of ama in the body.

(5) UTKLESHA

It can not be clearly defined and considered as the pravritti of doshas in the upward direction, either Vatha or Kapha is the contributor. Nausea is the word which can be compared here. Utklesha is generated due to amadosha and vitiated Kapha as well as the Vatha. Praseka can be considered as its poorvaroopa. These two symptoms atogether are explained in the poorvaroopa of chardi[194]. Utklesha is considered as the sensation of vomit or retching. It is one of the most common symptom seen in dyspepsic patients and is really distressing as well.

(6) ARUCHI

Loss of taste of food and loss of interest of food intake, both are considered as aruchi, better known as anorexia. Sushruta believed that aruchi is a disease where one has absolute loss of interest in food due to shoka, krodha, bhaya like manasika bhavas and vitiated Vathadi doshas staying in jihva, hridaya and bhaktayana[195]. This indicates the somatic as well as the psychic components for the

causation of aruchi. This condition is explained by Vaghbata along with the rajayakshma, as one of the upadravas of the same. This is an indicator of the association of the symptom 'aruchi' along with the chronic conditions.

(7) GURUKOSHTATA

Heaviness in abdomen is termed as guru koshthata. It is due to delayed gastric emptying as well as digestive mechanisms. Mandagni in Amlapitta causes avipaka and vitiation of the dosha pecuiliarly, ama or Kapha. This avipaka and resultant dosha dusti are responsible for producing this symptom, gurukoshtata.

(8) VIDBHEDA

While explaining ajeerna, Vaghbata has mentioned that the resultant presentation due to ajeerna is of two ways. One is the atipravritti or vidbheda and the other is the apravrithi or malarodha[196]. In poorvaroopa of atisara also, malarodha is explained. So vidbheda is one of the conditions resulting from ajeerna. It is one of the symptoms of Amlapitta as well, explained by Acharya Kashyapa. The vegas regarding the malapravriti is increased, but the total quantity of pureesha may be normal. Vatha dusti and agnimandya are the main responsible factors for developing vidbheda. This is a more prominent feature of adhoga Amlapitta.

(9) UDARADHMANA

It is found in amashaya or in pachyamanashya. The pain or discomfort may be due to the movement of Vatha in the koshta. The function of the Vatha is deranged by the altered doshas or the ama. The excessive production of Vatha is due to the resulting fermentation or shuktata. Fermentation occurs due to the avipaka or chirakala or late paka. Excessive production of Vatha leads to the distension of amashaya and pachyamanashaya. Excess admana leads to discomfort

in breath as well leading to conditions like kshudra swasa. The sthanasamsraya of doshas are in the amashaya, in the samprapthi of Swasa roga.

(10) SHIRORUK

Acharya Kashyapa has explained this symptom along with the Amlapitta. Mainly it is associated with bhrama. Headache is produced due to vitiated Pitta and Vatha dosha. Ajirna and amadosha, which produces vibandha, are also responsible for the same. Most of the the patients of Amlapitta, have constipation, which also creates shiroruk, due to the pratiloma gathi of Vatha.

Urdhva Amlapitta and Adhogata Amlapitta

1) In this type, the upper GI symptoms predominate clinically. Vamana/ utklesha are the most common presentation. The vomitus may be of various colours. Also the daha of uras, kanta regions are also associated.
2) Tikta and amla udgara are the main symptoms of urdhvaga Amlapitta, amla rasa being characteristic rasa of vidagdha Pitta.
3) Kapha Pittaja urdhvaga Amlapitta – Here kara-charana daha, avipaka, utklesha, aruchi, jwara, kandu and mandala are present.
    a. Urdhvaga Amlapitta tends to affect the annavaha srotus predominantly. Urdhvaga Amlapitta exhibits symptoms of Pittaja and Kaphaja Amlapitta. (aruchi, vami, kandu, daha)
    b. Adhoga Amlapitta exhibits also the symptoms of Purishavaha srotodushti. Here the Vatha dosha is also having a significant role. This disease if continued for some time, involves many other srotuses of body.

c. Marked similarity can be observed between adhoga Amlapitta and grahani (bhrama, daha, murchha, moha, and harsha) and is the point for differential diagnosis.

UPASHAYA – ANUPASHAYA

Those factors which relieves and aggravates the signs and symptoms of the disease are known as upasaya and anupasaya of the specific disease. It provides a diagnostic aid for the disease, which are the otherwise difficult to diagnose. It includes not only the oushada, but also ahara and vihara. Really these three factors are causing any sort of disease in the body and also is contributing to the relief or cure as well. It also provides an idea about the initial line of management to the physician. The idea we are getting while assessing the upasaya, has to be effectively incorporated for further treatment.

Vatika	Snigdha as well as ushna is comfortable for the patient.
Paittika	Madhura and seeta dravyas relieve the symptoms.
Kaphaja	Ruksha and ushna dravya provides upashaya.

We are determining the dosha status of many a disease by using the appropriate gunas, in the form of oushada. In Vathika Amlapitta, we can use grithas as the medicine. While in Kaphaja, we can effectively use churnas like hinguvachadi or vaiswanara, eventhough rooksha and ushna, they may subside the symptoms of Amlapitta, due to the samana of Kapha. As the Amlapitta is having association of ama along with Pitta, initially the administration of gritha is noticed as anupasaya. Grithas are being given after getting rid of the ama stage. Aharas are also providing similar information, regarding the dosha status of the condition.

UPADRAVA

In the progressive stages, if the nidana sevana continues, samprapti spreads to other adhisthanas causing different diseases or symptoms, rather than pertaining to Amlapitta. The main samhithas explaining the upadravas of Amlapitta are the Kasyapa samhitha[197] and the Sidhanta nidana.[198]

Table 14 Upadravas of Amlapitta

Symptom	K S	S N	Symptom	K S	S N
Jwara	+	-	Seetapitta	-	+
Atisara	+	-	Udarda	-	+
Pandu	+	-	Kandu	-	+
Shula	+	-	Mandala	-	+
Shotha	+	-	Vicharchika	-	+
Bhrama	+	-	Pidaka	-	+
Amasaya kshata	-	+	Grahani kshata	-	+

DHATUGATATVA OF AMLAPITTA

Doshas vitiated due to same nidana can produce different diseases as per their lodging, with special reference to ashaya and dushya.[199] Though the pathology of Amlapitta seems simple, but the treatment given in some condition does not give relief, this exhibits another view to think about. Each and every dosha resides by the shelter of any of the related dhatu. But the vitiated doshas moves in different places, dhatus and leads to the vikruti of that particular dhatu, this condition is known as dhatugatatva or existence of roga in the dhatus. If doshas are following the dhatugatatva, then there should be difference in its treatment. In many a diseases, the chronicity leads to dhatugata condition, the

extent varies as per the pathogenesis of the disease. Eventhough each and every disease is having a dhatugata avastha, a few examples or the dhatugata stage of the major diseases are only explained in classics.

Dhatugata doshas are generally tiryakgata and are difficult to manage. Doshas might be sama or nirama, that is why we must think on the line of samadosha and tiryakgata doshas, in the management. They are chronic in nature and so the pachana or shamana should be done by observing the status of dosha, agnibala etc. Doshas should be brought into kostha and thereafter, they should be eliminated out of the body. "Dhatu vaishamya" is nothing, but the discordance of the corresponding dhatu. Dhatu samya is the ultimate aim of Ayurvedic treatment.

For a gata Vatha to occur, it needs two types or stages of nidana, the nidana for the Vatha kopa and the nidanas for the dhatukshaya. Such is the case of any disease affecting a pecuiliar dhatu.

Acharya Vagbhata in Ashtanga samgraha has described the dhatugatatva of Pitta and Kapha. Charaka has explained the Gatatva of Vatha[200]. In dhatugata avastha, dosha disturbs the sthayi dhatu along with poshaka dhatu. Generally, it leads to kshaya condition of that pecuiliar dhatu. At such a condition, treatment should be based on that particular dhatu.The drug should be doshasamana as well as doing poshana of that dhatu.

Dr Sadashiva Sharma have described that, in the Samhitas, Dhatugata stage of four diseases have been described namely ;

1) Jwara, 2) Kustha, 3) Vatavyadhi 4) Masoorika.

He explained that, the first three diseases are representatives of Dhatugatatva of Pitta, Kapha and Vatha diseases. He further explained that Jwara

should be taken as example for all the Pittolbana diseases, Kustha for Kapholbana and Vatavyadhi for Vatolbana diseases in the context of concept of dhatugata.

In Amlapitta, the rasagata symptoms observed are gourava, aruchi, chardi, praseka etc. The raktagata symptoms are hasta pada daha, hritdaha, manadala, pidaka, trishna etc. In the stage of Mamsagata, Mamsapaka can be observed which can be co-related with peptic ulcers, gastritis etc. The soola resultant from Amlapitta, is the most difficult to manage, says the great Gananath sen in his manuscript, the Sidhanta nidana[201].

VIKALPA SAMPRAPTHI

In the pathogenesis of Amlapitta, first there is production of suktapaka due to agni dushti and if it mixes with Pitta, leading to the disease. So whenever the patients complain about the symptoms of Amlapitta, a thorough examination as per Ayurvedic point of view, should be done. We must analyze by which of the properties, Pitta is vitiated and mixed with shuktapaka, as treatment differs in these situation. Mainly drava and amla guna is aggravated, in this disease. By observing the sign and symptoms of the patients, we can infer by which guna, Pitta is vitiated as described here.

Altered Guna - Lakshana

Drava	Hrillasa, Asyasrava, Chardi
Amla	Amlika, Amlasyata, Amlodgara, Amlarasayukta Chardi
Teekshna	Vedana, Vrina
Ushna	Ura-Udara-Kantha Daha, Jwarapratiti, Aaushnya, Sarvanga daha
Visra	Aasya daurgandhya, Loha-Ama Gandha, Utsahahani
Sara	Asamhata malapravritti

By analyzing these gunas, we can alter the line of management accordingly. If we are able to differentiate these properties, we can manage the patient easily and get enhanced results. Likewise, if these properties involve different dhatus then also we can change line of treatment and it should be according to progression of the pathogenesis.

PRINCIPLES OF CHIKITSA

Ayurveda has three basic categories of Chikitsa regarding the management of any disease. The combinations of these are used in the treatment of the condition, accordingly.

1) Nidana Parivarjana

Removal of all the alleviating factors of both ahara and vihara which are responsible for causation of the disease, is to be performed. It is to be advised to the patient to avoid such type of factors which are responsible for. Even in the days of Kashyapa, Acharya advises to avoid the food materials which are processed. While explaining the sadhyasadhyata, he adds that the disesases like Amlapitta becomes sadhya in those who are not greedy towards their food habits[202]. The ancient people were not as careless as we people, as far as the food habits are concerned. Still the restrictions are mentioned by them, to be followed. That points towards the seriousness of dietary restrictions, in GI conditions like Amlapitta, in their management.

2) Apakarshana

Apakarshana or sodhana chikitsa is the unique methodology of Ayurveda as far as the approaches of all the medical systems are considered[203]. Sodhana is the management protocol for the prabhoota dosha in any condition.

While selecting the concerned sodhana, mainly 3 factors are to be considered. ie. the status of dosha, localization of the doshas or the sthanasamsraya and bala of the patient. In Amlapitta, eventhough it is a Pitta dominant disorder, the sthanasamsraya is in the amasaya, which is the Kaphasthana. The primary route of sodhana mentioned in vyadhi affecting the amasaya is the oordhwa sodhana, vamana. The sodhana which eliminates the doshas through the easiest route is always considered as the ideal one[204].

Kashyapa has described vamana as the first line of treatment, followed by langhana and laghu bhojana.[205] ie. Eventhough the Amlapitta is a Paittika disease, the most effective sodhana is vamana. Kashyapa opines that just like a tree with its trunk and branches are destroyed by striking the blow at its root. He says that, pecuiliarly for the chronic conditions; vamana is to be done definitely. He also gives another example for the relevancy of sodhana. If we are pouring pure mik in a vessel in which we have already kept takra and not properly cleaned, the milk eventhough fresh, is having a chance to become fermented. In fact, the samana drugs applied after doing vamana, will have the expected effect.The amasaya with the doshas associated with the ama is defective in absorbing the drugs eventhough samana drugs are being adviced, without proper sodhana. Such a significance is there for sodhana, in the context.

As per the Chakradatta and Yogaratnakara, the second line of treatment is to perform mridu virechana.[206, 207] Drugs used for vamana are lavanambu, sukhosna, dugdha, ikshurasa, madhudaka or tiktadravyas and for the virechana triphala, trayamana, katuki along with trivrit is mentioned.[208] According to Bhavaprakasha, the decoction of patola, nimba, and madanaphala with saindhava lavana should be used for vamana in Amlapitta[209].

Nishotha churna and amalaki are prescribed for the virechana. Avipathi choorna is one of the most commonly used yogas for virechana, which is mentioned by Vaghbata.[210] The next regimen consists of administration of anuvasana followed by asthapana, in the chronically afflicted patients. The drugs such as tiktaka gritha, indukanta gritha, aragwadhadi gana gritha etc. are usually used for the purpose of anuvasana. Madhuyashtyadi taila and Ksheerabala taila are also used in this regard.[211] After anuvasana, in chronic cases, nirooha is mentioned. Usually ksheeravasthi is the ideal one to be administered here.[212] Instead of plain ksheera, ksheera kwathas with guduchyadi gana, tiktaka kwatha, indukanta kwatha, aragwadhadi gana is usually used. Plain gritha is observed to cause utklesha in these patients.

Yogaratnakara added raktamokshana as a tool if Amlapitta is not cured by both of the sodhana procedures, vamana and virechana.[213] Vaghbata explains the diseases occurring due to raktadushti. Eventhough, Amlapitta is not mentioned as such, the symptoms resembling the disease are mentioned as katu and amla udgara may happen in one with the vitiation of raktha[214]. Moreover in some of the patients with Amlapitta, symptoms of rakta dhatu dushti are also observed. So in such a condition, raktamoksha is having significant role, the technique being decided by considering all the related factors.

3) Prakritivighata

Prakritivighata refers to the use of drugs which suppress the elevated dosha, such treatment is also termed as the samana therapy[215]. Kashyapa opines that after vamana, if the doshas persist, the physician should resort to the samana chikitsa with the aid of laghu bhojana, samana and pachana. It is forbidden by Acharyas to give drava aushadhi if the doshas are in condition of utklesha, because

if vamana is not done, the drava aushadhi will not be metabolized. When the dosha utklesha has reduced with the help of ahara and vihara, physician can advise ama pachana and bhedana drugs. Once the doshas have been expelled and amashaya is devoid of vitiated doshas, the physician should direct the patient to take care of the agni. The doshas lodged in the pakvashaya, is removed with the help of sramsana drugs, which is selected as per the condition.

Mainly tikta rasa, laghu, snigdha guna, katu or madhura vipaka and seeta veerya drugs are advocated by all the Acharyas, for Amlapitta. Use of samana drugs opposite to quality of Pitta and to an extent Kapha, is beneficial for Amlapitta.

We have to consider the associative dosha, while prescribing the samana drug. We can use combinations like drakshadi kwatha in Vatha pitta condition, guloochyadi gana in Kapha Pitta condition and tiktaka kwatha for Pitta condition. Likewise we can select gritha yogas as well. Indukanta gritha is ideal for Vatha Kapha condition, Mahatiktaka gritha for Vatha Pitta condition and Aragwadhadi gritha for Pitta Kapha condition. The drug of any format is usually selected according to the dosha. Choornas like Avipatti can be used both as a samana as well as a sodhana drug.

The yogas can also be used in a different format, if the condition demands. The gritha yogas can be administered in the form of Kwatha, if we know that gritha must not be administered, but the combination is ideal for use[216]. For eg. Mahatiktaka yoga when administered in the gritha form is much more Vatha Pitta in action, while if it is being administered as kwatha, it is more Pitta Kapha samana in action.

Importance of tridoshas

From the above said factors, it is very much clear that Samana Vatha, Pachaka Pitta and Kledaka Kapha is directly involved in the functioning of agni. So the variation or insult in any of the three doshas affect the process of digestion and is the causative factor of conditions like dyspepsia or Amlapitta. So in the management of Amlapitta also, all the doshas are to be considered, while performing the management.

On the other side it is also to be said that, the medicines mentioned for the management of other diseases are being used successively in cases of Amlapitta. For eg. Gandarvahasthadi kwatha which is indicated in mandagni and malarodha is causing relief to the patients with Amlapitta.[217] The combination seems to create anulomya of Vatha which infact, improves the symptoms of the GI tract, eventhough it is Paittika in nature. Nayopaya kwatha mentioned for the swasa roga is also showing similar effects.[218] The kwathas like sukumaram, saptasaram is also reported to have clinical efficacy in this regard.

Similarly, if the dominating symptom is avipaka, utklesha etc. the condition seems much more Kaphaja in distribution. Here we can safely use combinations like shaddharana, vaiswanara etc. ensuring that the patient is not having vridhi of Pitta[219] Eventhough, these are not effective in Amlapitta condition directly, the pachana property of the drugs help the condition to become better, if it is more Kaphaja in nature. So an ideal drug for Amlapitta is to have the properties of Kapha Pitta samana, Vathanulomya and Pachana.

SADHYAASADHYATHA

The prognosis of the disease, Amlapitta is not uniformly favorable as

per the classics. Madhavakara has pointed out that, in case the patient has been suffering from Amlapitta recently or is nava and is treated properly, the prognosis is excellent.[220] Chronic cases may either improve a little or may be relieved completely, during the course of treatment. As soon as the patient deviates from the wholesome diet, the disease relapses. When the disease is of short duration, then it is sukhasadhya, it is yapya when chronic, kricchrasadhya when the duration of the disease is prolonged and cured with great obscurity and asadhya when the patient will be having different upadravas and symptoms of dhatu kshaya[221].

Kashyapa has indicated that in case of patients with Amlapitta gets complicated by jwara, pandu, soola, sopha, aruchi and bhrama with dhatu kshaya are incurable.[222] Age of maximum occurrence is in the youvana avastha which is Pittakala. Sharad ritu is also Pitta prakopaka kala and the reasons also are many in the causation of this disease i.e. ahara, vihara, manasika bhavas etc. These are the factors capable to convert this disease as krichhrasadhya.

The life style of the people has changed drastically, since the age of the samhithas. The stressful life situations happening nowadays is having a very serious role to play, in the pathogenesis of conditions like Amlapitta. A psychological approach is to be enhanced in the present day management protocol after assessing the situation. Yoga therapy and pranayama can be incorporated here in an effective manner.

PATHYA-APATHYA

Any disorder involving the GI tract is having a direct and definite relationship with the dietary habits and hence the dietary regulations are a must. This has to be considered seriously while managing diseases like Amlapitta, where the dietary factors are working along with several other diseases, in the

manifestation. Even in the period of samhithas, Kashyapa has stressed on the dietary restriction and its importance in the management of Amlapitta, with utmost concern. In the recent times the human beings have lost the control over sticking on to the healthy food habits. So besides enlisting the good or bad as per the diet, strict patient education is to be done in this regard.

The following list of Pathyapathya are observed in the disease Amlapitta as suggested by various Ayurvedic scholars.

Ahara

1) Anna Varga – Yava, godhuma, purana sali, mudgayusha, lajasaktu.
2) Saka Varga – Karavellaka, patola, kusmanda etc.
3) Phala Varga – Dadima, amalaki, kapittha etc.
4) Dugdha Varga – Godugdha
5) Mamsa Varga – Jangala, mamsarasa
6) Miscellaneous – Sarkara, madhu, narikelodaka.

Vihara Seetopachara, vishrama etc**.**

APATHYA

A) Ahara – Guru, vidahi, viruddha, ushna, katubhojana, kulatha, rasona, navanna, tila, and fermented foods like bread, pickles
B) Lavana, amla, katurasa, madya, arishta, preservatives in food as well as drugs
C) Adhyasana, samashana, vishamashana, virudhashana
D) Bakery items which are excess in salts, oil and maida and processed

E) Vihara - Vegadharana, atapasevana, ayasa after food, divaswapna

F) Psychological - Chinta, krodha, shoka, bhaya.

G) Vishama nidra

The interesting thing observed in clinics is that, one which is apathya to a person is not creating much disturbance or harm in another one. So a general list of apathyas are difficult to frame for all the patients attending with conditions like Functional Dyspepsia and the advise varies from person to person. One fact explained by Vaghbata in udara chikitsa needs mention here ie. One must not exercise the apathya mentioned for that disease, also he must not use the pathya mentioned also, beyond a certain extend.

Chapter - 4

DRUG REVIEW

MAHTIKTAKA

The formulations mentioned in the Ayurvedic parlance are aimed or designed not only to manage a particular condition, but also to restore the absolute harmony of the functions of the body as well as the mind. These are also formulated in such a way that the combinations are maintaining the adverse effect or unaccepted effect of the drug, to a bare minimum. It is also aimed at the reversal of the multifactorial contribution, regarding a disease. The same formulations are also being advised to administer in such a form, after visualizing the condition or status of the disease.

Mahatiktaka yoga is a combination mentioned in the form of gritha, in the context of Kushta by the Acharyas.[1,2,3] It is also being used in the form of kwatha and lepa effectively for the management of several conditions. Several kwathas had been converted to tablets in due course, the purpose being many and varied. In this peculiar study, Mahatiktakam kwatham tablet is being used as the trial drug. No studies seem to be reported with the kwatham tablets in this condition, as per the available knowledge. The published studies are the efficacy trials being conducted, by the pharmaceuticals at an early phase[4]. One of the studies conducted concludes that, there is no difference in the efficacy of

Rasnerandadi yoga as kwatha as well as tablet in symptoms of Vathasonitha.To conclude, there is no significant difference in the efficacy of the kwatham tablet when compared with the same kwatha and is also safe, as well as convenient.

There are several clinical conditions where the combination of the corresponding gritha is ideal, but the administration in the sneha form is not appreciated by the patient or his clinical condition. In such situations, the gritha yoga is used as kwatha or some other form, according to Vaghbata as well as Charaka.[5] Currently we are using several yogas in the form of different pharmaceutical preparations. Here in this study, the Mahatiktaka yoga is administered as kwatha in the tablet form and it is being compared for the efficacy with the allopathic counterpart, Omeprazole in Functional Dyspepsia.

Indications

As per Acharya Vaghbata, the indications of Mahatiktaka yoga are the same as the tiktaka gritha, but the efficacy is said to be superior to that combination, accordingly[6]. But Acharya Charaka is describing separate indications and conditions for both these combinations on its use[7].

Table 15 Indications of Mahatiktaka gritha

Raktapittaja kushta	Pandu	Jwara
Raktarsas	Visphota	Kandu
Visarpa	Pama	Hridroga
Amlapitta	Unmada	Gulma
Vatasonitha	Kamala	Pidaka
Asrigdhara	Gandamala	

If the drug is administered as per the bala and at a suitable kala, Charaka advices that it is capable of curing hundreds of diseases which cannot be subsided by the other similar as well as famous yogas[8]. Besides this, Acharya Susrutha is advising an effective use of the combination in apasmara, vishamajwara, sleepada and klaibya.

These points to the wide,varied and multi systemic use of the combination, Mahatiktaka. The tiktaka gritha is mentioned to have efficacy in the disorders of each and every system of the body in a combination of dosha, dominated by the Pitta. In the context of agnivisarpa, Vaghbata advises Mahatiktaka as the best combination in its management, both internally and externally, indicating that, the combination is Vatha Pitta samana in nature[9].

Kwatham tablet – Advantages

- Palatability
- Easy to handle and administer
- Comfortable
- Accurate dosage
- Lack of preservatives
- Long shelf-life

MAHATIKTAM KWATHAM TABLET

a. METHOD OF PREPARATION

The Kwatha is prepared from cleaned, chopped, weighed ingredients with 16 times their weight of water and reduced to 1/4th the volume. The decoction is filtered and concentrated to a thick paste using a vacuum evaporator at a temperature of 53^{0}C. The thick mass is further dried in a tray drier at a temperature

not exceeding than 60^0C. The dried mass is then pulverized to obtain the powder of the kwatham.

The powder (approx 40 kg) obtained is mixed with the required quantity of excipients and also the binding agents in a mass mixer (To the extract powder 3 Kg maize starch is added as powder and 2 Kg starch is added as paste. To this 3 Kg Gum Acacia is added in the powder form).

The wet mass is dried in a tray drier and granulated in a comminuting mill using No.10 mesh to obtain the granules. The granules are then lubricated with the required quantity of lubricating agents such as Magnesium stearate. The lubricated granules are compressed on a rotary tablet punching machine to tablets of 8 x 16 mm size. The tablets are packed in blister strips of 10 tablets each on an automatic blister packing machine. The blister strips are packed in cartons of 10 strips each[10].

Total quantity of drug in one tablet – 890 mg

Total weight of one tablet – 1g.

Table 16 Ingredients : each Mahatiktam Kwatham Tablet[11]

Official Name	Botanical Name	Part Used	Form	Quantity
Saptacchada	Alstonia scholaris	Stem bark	Ext.	0.532 g
Parpataka	Hedyotis corymbosa	Plant.	Ext.	0.266 g
Samyaka	Cassia fistula	Stem bark	Ext.	0.266 g
Katuka	Neopicrorhiza scrophulariiflora	Root	Ext.	0.266 g
Vacha	Acorus calamus	Rhizome	Ext.	0.266 g
Haritaki	Terminalia chebula	Fruit.	Ext.	0.266 g
Amalaki	Phyllanthus emblica	Fruit	Ext.	0.266 g
Vibhitaki	Terminalia bellirica	Fruit	Ext.	0.266 g
Padmaka	Prunus cerasoides	Root	Ext.	0.266 g

Official Name	Botanical Name	Part Used	Form	Quantity
Patha	Cyclea peltata	Root	Ext.	0.266 g
Rajani	Curcuma longa	Rhizome.	Ext.	0.266 g
Daruharidra	Berberis aristata	Root	Ext.	0.266 g
Sariba	Hemidesmus indicus	Root	Ext.	0.266 g
Krishnasariba	Ichnocarpus frutescens	Root.	Ext.	0.266 g
Kana	Piper longum	Fruit.	Ext.	0.266 g
Krishnamula	Piper longum (wild var.)	Root	Ext.	0.266 g
Nimba	Azadirachta indica	Stem bark.	Ext.	0.266 g
Chandana	Santalum album	Heart Wood	Ext.	0.266 g
Yashtyahwa	Glycyrrhiza glabra	Root	Ext.	0.266 g
Visala	Citrullus colocynthis	Plant	Ext.	0.266 g
Indrayava	Wrightia antidysenterica	Seed	Ext.	0.266 g
Amrita	Tinospora cordifolia	Stem.	Ext.	0.266 g
Kiratatikta	Swertia chirayita	Plant	Ext.	0.266 g
Sevya	Vetiveria zizanioides	Root.	Ext.	0.266 g
Vrisha	Justicia beddomei	Root	Ext.	0.266 g
Murva	Chonemorpha fragrans	Root	Ext.	0.266 g
Satavari	Asparagus racemosus	Tuber	Ext.	0.266 g
Patola	Trichosanthes lobata	Plant	Ext.	0.266 g
Ativisha	Aconitum heterophyllum	Root	Ext.	0.266 g
Musta	Cyperus rotundus	Tuber	Ext.	0.266 g
Trayanti	Gentiana kurroo	Plant	Ext.	0.266 g
Dhanvayasha	Tragia involucrata	Root	Ext.	0.266 g

METHOD OF ANALYSIS

Disintegration test

Place five tablets in the tube. Insert the guided disc above the tablets, in the tube and raise and lower the tube in such a manner that, the complete up and down movement is repeated to about thirty times per minute. The tablets are disintegrated when no particles remains above the gauze which will not readily pass through it. The time required for five tablets to disintegrate in the manner prescribed is, unless otherwise stated in monograph, not more than fifteen minutes.

Diagram 3 Preparation flow chart of the Kwatham

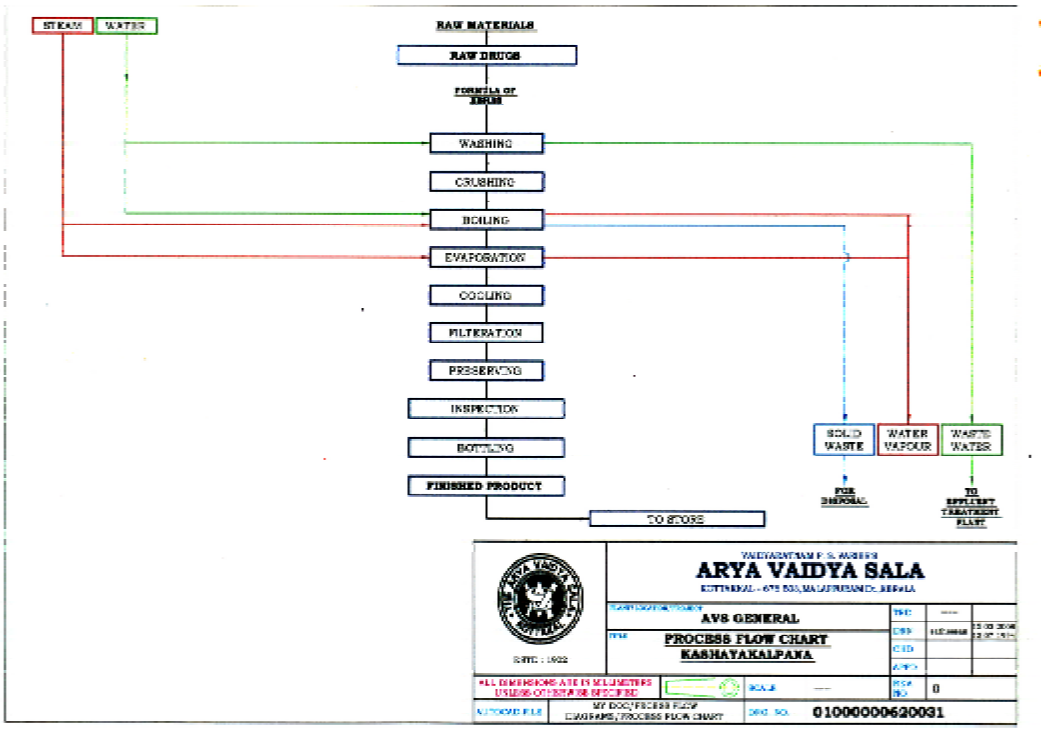
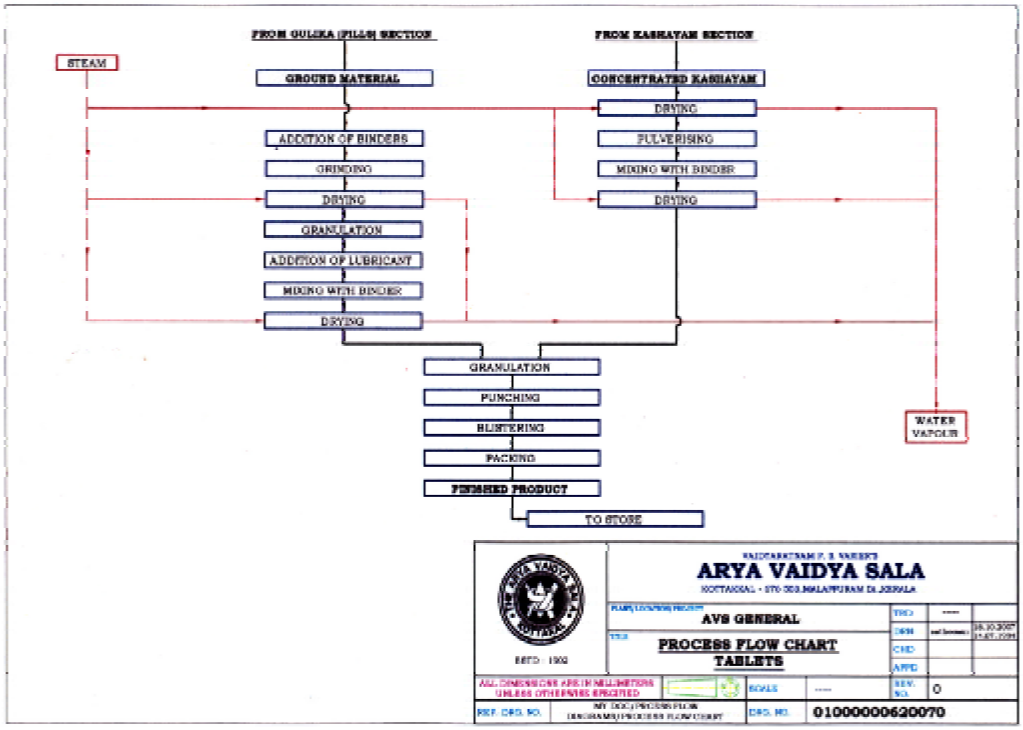


Diagram 4 Preparation flow chart of the Kwatham Tablet



Loss on Drying

Known weight of the tablet powder is dried at 105^0C for one hour and the loss of weight is calculated as a percentage.

Average weight

Average weight of the 20 randomly selected tablets is taken.

Friability

Friability of 10 tablets are measured by the Friability tester.

Hardness test

It is measured by the hardness tester.

Leak test

Sealing deficiency is measured with the leak test apparatus.

PRODUCT SPECIFICATIONS OF MAHATIKTAM KWATHAM

Table 17 PHYSICOCHEMICAL PARAMETERS - Kwatham

	Name of the test	Unit	Standard
01	Description	-	Brownish black colloidal liquid with bitter taste
02	pH	-	4.0 – 5.0
03	Density	g/ml	1.0 – 1.15
04	Brix value	-	18.0 – 30.0
05	Total Solids	%w/w	20.0 – 30.0

HEAVY METAL LIMITS

Pb = NMT 10 ppm; As = NMT 3 ppm; Hg = NMT 1 ppm; Cd = NMT 0.3 ppm

PRODUCT SPECIFICATION OF MAHATIKTAKAM KWATHAM TABLET

Table :18 Physicochemical parameters - Kwatham Tablet

	Name of the test	Unit	Standard
01	Description	-	Brownish black tablet with bitter taste
02	Average weight	g	1.15 – 1.25
03	API content	g	0.85 – 0.95
04	Hardness	kg/cm^2	NLT 6
05	Thickness	mm	6.3 – 6.5
06	Disintegration time	minutes	NMT 15
07	Weight variation	% w/w	$\pm$ 10 %
08	Friability	% w/w	NMT 1

HEAVY METAL LIMITS

Pb = NMT 10 ppm; As = NMT 3 ppm; Hg = NMT 1 ppm; Cd = NMT 0.3 ppm

HPTLC ANALYSIS OF MAHATIKTAM KWATHAM & TABLET

SAMPLE DETAILS

01. Mahatiktam Kwatham
02. Mahatiktam Kwatham Tablet

HPTLC CONDITIONS

01. 5 ml Mahatiktam Kwatham is evaporate to dryness and extracted with 10 ml methanol and spotted.
02. 1 Tablet of Mahatiktam kwatham is extracted with 10 ml methanol and spotted.

STATIONARY PHASE

Merk, 1.05554.0007, TLC Silica gel 60 $F_{254,}$ 10x10 cm Aluminium sheet.

MOBILE PHASE

Toluene: Ethyl acetate: Formic acid: Methanol (7:5:1:0.5)

CAMAG Linomat 5, CAMAG TLC Scanner 3, CAMAG Reprostar 3

Development

CAMAG 10x10 cm Twin trough chamber.

Table 19: Properties of the drugs in Mahatiktaka Yoga

Property of the drugs	Percentage
Madhura rasa	31
Katu rasa	31
Tikta rasa	84
Kashaya rasa	41
Ushna veerya	44
Seeta veerya	53
Snigdha guna	34
Rooksha guna	60
Laghu	81
Guru	19
Vatha kapha hara	19
Pitta Kaphahara	56
Tridoshahara	22
Madhura vipaka	38
Katu vipaka	66

HPTLC Instrumentation

I. Results

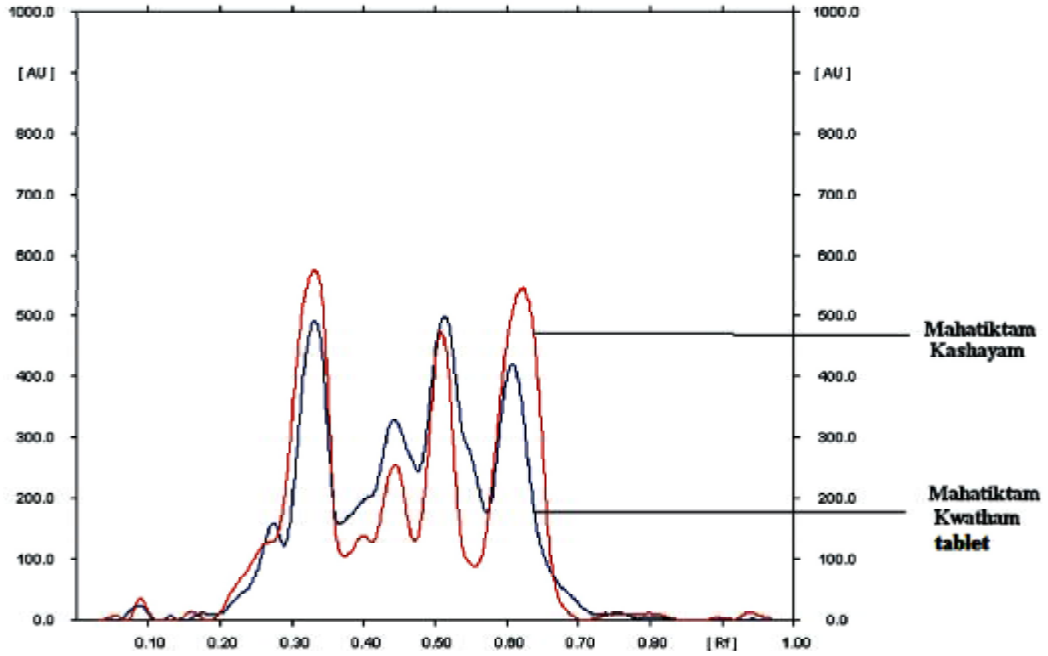


Diagram 5 Overview graph of samples at 254 Nm

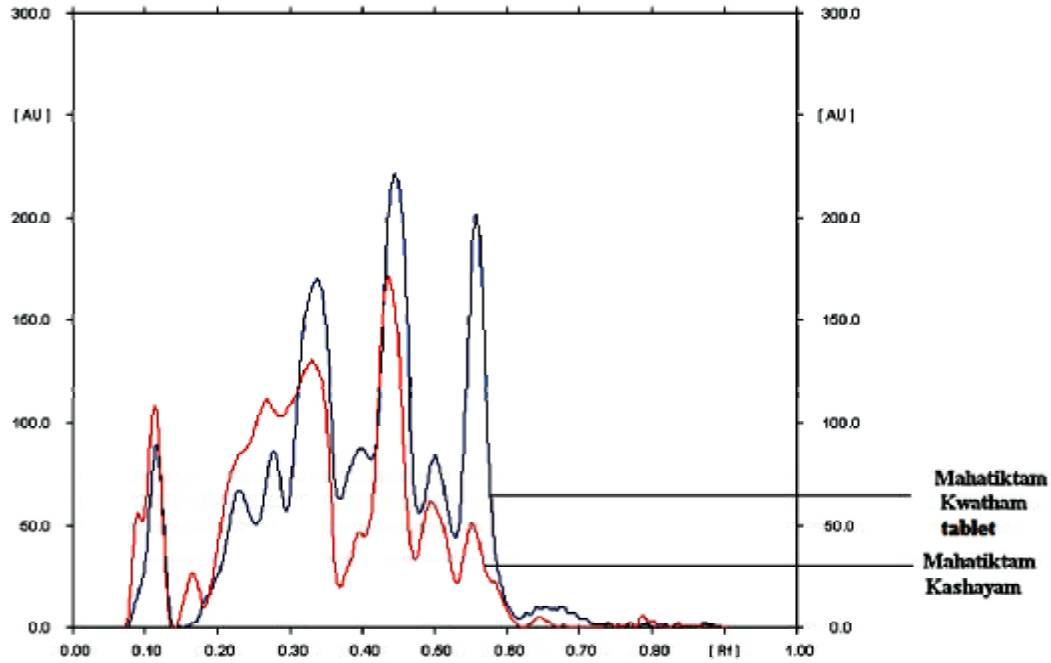
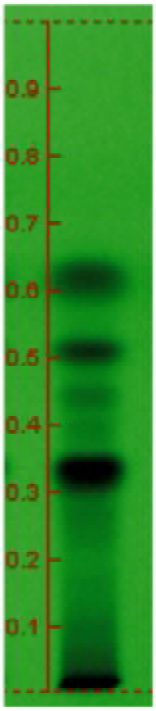
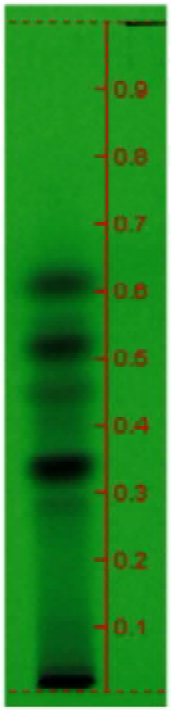


Diagram 6 Overview graph of samples at 366 nm



Mahatiktam Kashayam

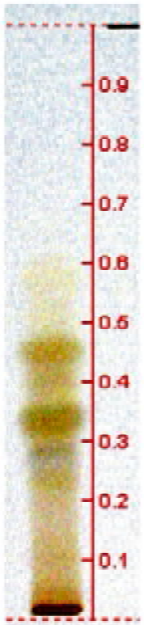


Mahatiktam Kwatham **tablet**

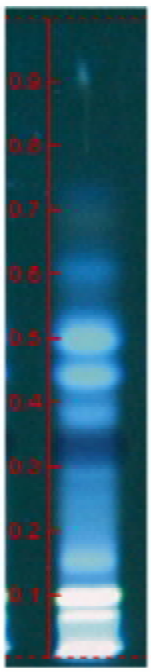
Diagram 7
TLC plate views samples at 254 nm

Diagram 8
TLC plate views samples at 366nm

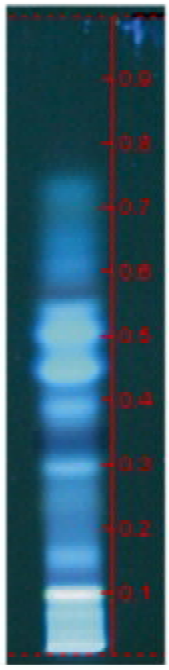
Mahatiktam Kashayam



Mahatiktam Kwatham **tablet**



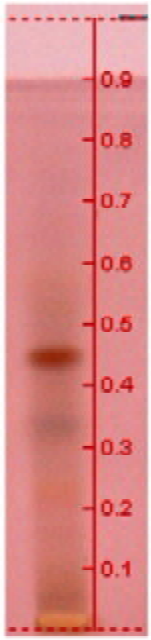
Mahatiktam Kashayam



Mahatiktam Kwatham tablet

Diagram 9
TLC plate views samples at white light

Mahatiktam Kashayam



Mahatiktam Kwatham tablet

Diagram 10
TLC plate views samples at derivatization

STUDIES OF THE INDIVIDUAL DRUGS OF MAHATIKTAKA

Several trials have been conducted overall both at the experimental and clinical level regarding the individual drugs of the Mahatiktaka yoga in dyspepsia and also on H Pylori. Even though the additive effects of the individual drugs cannot be expected as such in the combination, the efficacy of the individual drugs are a real pointer to the efficacy of the combination. Here a few observations on the studies conducted by the individual ingredients of the Mahatiktaka combination, are being discussed.

Alstonia scholaris was observed to be effective in gastritis, have anti ulcer activity and enhances the mechanism of digestion.[12] It is also having antidiarrhoeal property, as per studies. Aqueous extract of Cassia auriculata leaves possess protective activity against ethanol induced hepatotoxicity and also aids the improvement in the body weight and normalization of vitamin A and C level[13]. Methanolic extract of Cassia plant proved a reduction in the ulcer index and gastric acid output, in the experimental animals[14].

Alcoholic extract of Picrorhiza and kutkin possess hepatoprotective activity and is a stimulant of both the cell mediated and humoral immunity and cured Non-alcoholic Fatty Liver disease in rats and reduced the lipid content of liver as well[15].

Studies report that Phyllanthus emblica, Myristica malabarica, Terminalia chebula, Picrorhiza kurroa, and so forth, and their active constituents reduced gastric ulcer, which was induced by the NSAID's.[16]. These are proved to have anti oxidant activity, immunomodulation and gastro protective activity.

Urease inhibitor activity was identified in the plant extracts of Zingiber

officinale, Acorus calamus, Allium sativum, Curcuma longa etc. which in fact deactivates the infection of H pylori.[17] Terminalia Chebula extract reported significant improvement in NSAID induced and stress induced ulcers in the experimental study and also antisecretory activity, with increase in gastric pH[18]. Experimental studies conducted earlier have proved that Phyllanthus emblica, Plumbago zeylanica and Cyperus rotundus, plants from the medohara group mentioned by Charaka, possess the anti - atherosclerotic activity[19].

The drug Asparagus racemosus, along with Phyllanthus emblica prevents gastric damage by reducing edema, congestion and inflammation over there, caused by Methanol and Aspirin.[20] Anti - H Pylori activity was shown by the aqueous extract of Emblica officinalis. The alkaloid Cissampariene from Cissampelos pariera shows anti- diarrhoeal as well as depressant activity of the CNS.[21] The extract of Curcuma longa proved to reduce gastric acid secretion by H_2 receptor antagonistic action to an extent, reducing the dyspepia, but was not effective against the H Pylori[22]. The Curcuma longa extract was also significantly effective for Irritable Bowel Syndrome.

Berberine isolated from Berberis aristata pointed out the dose dependant growth inhibition, in the H Pylori culture[23]. Hemidesmus indicus roots proved to possess the antioxidant activity and are also antimicrobial.[24] The water extract of this drug can be used in ORS solution for accelerating its anti diarrhoeal action[25].The ethanolic extract of Piper longum was observed effective with amoebicidial action[26].

When the antisecretory as well as antiulcer activity of the aqueous extract of Azadirachta indica was studied, it was equal in potency against

Omeprazole in stress ulcer and also is an antioxidant[27]. There was significant reduction in the gastric acid secretion and also healed the duodenal ulcers, monitored by the Barium meal study, but don't showed any shift in the mucus secretion.

An extract of Glycyrrhiza glabra was reported effective in reducing the severity of the symptoms of FD, when assessed with the Nepean Dyspepsia Index with 30 days and was also safe.[28] Glabridin, the flavonoid present in the Glycyrrhiza glabra also exhibited superior activity against H pylori in dilution with agar, in the form of inhibition. Methanolic extracts of Punica granatum, Holarrhena antidysentrica and Triphala showed activity against the drug resistant Salmonella typhi infection[29].

The powder of Adathoda vasica leaf proved to be effective in ethanol as well as aspirin induced ulcers in an experimental study[30]. The extract of Asparagus racemocus was studied to have comparable efficacy with Ranitidine in reducing the ulcer index and also reduces the gastric secretion[31]. This drug also enhanced the mucosal defensive factors in the particular study and is also working as an antioxidant. The decoction of rhizomes of Cyperus rotundus is established as potent against the ethanol induced gastritis, in a protective mode[31].

The ethanolic extract of the drug Swertia chirata reduces the damage to the gastric mucosa due to the NSAID's in an experimental trial and also the mucus content was restored to normal.[32] Ethanolic extract of Citrullus colocynthis is having noteworthy role in reducing the ulcer index due to the flavonoids and alkaloids present in the extract comparable to the outcome of Ranitidine [33].

The acetone extract of Calotropis procera proved to have significant

urease inhibitor quality and effective against H Pylori, comparable with the triple therapy for it [34]. The ethanol extract of the leaves of Glycyrrhiza glabra and Fagonia Arabica is said to have efficacy against Escherichia coli and Staphylococcus aureus and is anti microbial.[35] Tinospora cordifolia in the form of ethanol extract proved the protective action in the case of stress induced ulcer[36]. This drug is also having hepatoprotective and also immune enhancing action and is also having a dose dependant anti diarrhoeal effect. The pharmacological effects of the various ingredients of the Mahatiktaka yoga has been studied according to the modern parameters and found effective in dyspepsia.

Indukanta Ghrita and Mahatiktaka Ghrita

The Ghrita preparations are given usually before sodhana for utklesha and also as samana therapy. Here the Indukanta gritha and the Mahatiktaka Ghrita are given in two separate groups of patients with one group as samana and in the other group after a small course of sodhana. The studies in the four group of patients have been conducted, on about 1200 subjects. The diagnosis and assessment of efficacy has been made on the basis of modern investigations, including endoscopy. It was noted that both the gritha preparations were effective in 75 to 80% of patients. The efficacy is further enhanced if a course of sodhana given before the administration of ghritas. The effect of Mahatiktaka Ghrita is relatively better, compared with the control.[37]

It was observed that Snehapana with both the Indukanta gritha and Mahatiktaka gritha are effective in Parinamasoola, the efficacy being higher in the Mahatiktaka group (80%). Mahatiktaka was found more effective in Vatha Paittika soola among the included. In another study, it was observed that healing

effect of Mahatiktaka gritha are high in the age group of 20-30 as 60% had their ulcers healed after the therapy, compared with the control[38]. The Indukanta gritha which is Vatha Kaphasamana is not having the efficacy compared to the Mahatiktaka gritha, in the healing of peptic ulcer. These point to the Vathahara as well as Pittahara action of the drug, Mahatiktaka and its efficacy here.

Ulcerative colitis

According to a study conducted by Simi Raveendran etal. the Mahatiktaka gritha was administered along with the classical combination of Pichavasthi in patients of Ulcerative colitis as Yoga vasthi, for 8 days. It was concluded that the vasthi protocol is effective in reducing the passage of blood with stool, mucus in stools, abdominal cramps, anorexia at 1% level. Also the protocol improved the Hemoglobin of the included subjects[39]. The Mahatiktaka yoga is very much effective in conditions of Pitta raktaja in nature, opines Charaka.

Psoriasis

In a study by Rajkala S Ramteke, it was observed that sodhananga snehapana with Mahatiktaka gritha was highly significant in reducing the symptoms of Psoriasis such as itching, exfoliation, erythema, pain and also attained the samyak snigdha lakshanas such as vatanulomana, deeptagni, snehodvega and klama in almost all the subjects in the trial[40].

Parinama soola

In a study by Ansy etal. it was observed that the samana snehapana with Mahatiktaka gritha was significant in the Pittadhika avastha of the parinama soola patients when performed, upto a duration of 30 days. It was also concluded

that in the same subjects, snehapana can be done with Indukanta gritha in Vatha Kapha stage and if the psychological factors dominate, the best option is the Kalyanaka gritha and the study was done with a single case design preferred by WHO for altrenative medicine.[41] The study pointed to the need of using different combinations for the same disease, if the dosha status changes through out the disease, after assessing the prakrithi, koshta and satwa.

Mahatiktakam Kwatham in Stress induced hyperglycemia

As per the study conducted by Snigdha Roy etal in stress induced hyperglycemia, it was observed that Mahatiktakam kwatham when administered in stress associated hyperglycemia in a Pitta predominant constitution was statistically significant as per the Stress Assessment Questionnaire and also the blood sugar evaluation among the included[42]. Mahatiktakam kwatham also possesses mild to moderate anxiolytic activity which was also proved in the animal model. This study is very much relevant as far as the management of the FD is concerned, as the psychological factors are having a leading role, in the exacerbation of the condition.

Mahtatiktaka gritha in Krodha WSR to Paittikonmada

As per a study by Preethy etal, it was concluded that Mahatikitaka gritha is significant in controlling the krodha and also reducing the agitation, destructive nature and self harm in those included, among the subjects with Paittikonmada[43].

From this it can be concluded that the combination, Mahatiktaka is much effective in the management of several conditions affecting the various systems of the body as well as the mind.

Omeprazole – Mode of action

Omeprazole is a proton pump inhibitor drug for the treatment of GI conditions like dyspepsia, peptic ulcer disease and gastroesophageal reflux disease. It is on the World Health Organization's List of Essential Medicines; the most important medications needed in any basic health care system and is of common use, among the practitioners[44].

Omeprazole is a drug with the property of selective and irreversible proton pump inhibition. The drug suppresses secretion of the gastric acid from the stomach by specific inhibition of the hydrogen-potassium adenosine triphosphatase (H^+, K^+, ATPase) enzyme system found at the secretory surface of the parietal cells of the stomach. It inhibits the final transport of hydrogen ions (via exchange with potassium ions) into the gastric lumen. Since the enzyme system is regarded as the acid (proton) pump of the gastric mucosa, it is known as a gastric acid pump inhibitor[45]. The inhibitory effect is dose-related. Omeprazole inhibits both the basal as well as the stimulated acid secretion irrespective of the corresponding stimulus. Omeprazole does not have anticholinergic or H_2-receptor antagonist properties, as per the current knowledge[46].

The inhibitory effect of Omeprazole occurs within 1 hour after oral administration. The maximum effect occurs within 2 hours with the duration of inhibition up to 72 hours. Although it has a very short plasma half-life, the anti-secretory effect lasts for a long time due to prolonged binding to parietal H^+, K^+, ATPase enzyme. When the drug is terminated, baseline stomach acid secretory activity returns after 3 to 5 days[47]. The inhibitory effect of Omeprazole on acid secretion will plateau after 4 days of repeated daily dosage.[48] This is completely metabolized by the cytochrome P450 system, mainly in the liver.

Omeprazole has demonstrated antimicrobial activity in vitro against Helicobacter pylori, by selective inhibition of *H* pylori urease, which is necessary for the gastric colonization of the organism.[49]

Studies regarding the adverse effects of the chronic PPI use

Eventhough the PPI's including the Omeprazole is one of the commonest as well as safer weapons of a gastroenterologist, the medical world is too anxious about its overuse and the reported harm or adverse effects of the therapy. There have been emerging concerns with reports and publications of potential adverse effects associated with the use of PPIs. The pathogenesis of these proposed associations is not clear in most of the cases and the evidence base to support a clear association for the harm, is extremely variable.

In the patients receiving chronic PPI therapy, there was a significant decrease in all hematologic indexes from the baseline data[50]. There have been concern among the medical personnel that the hypo-chlorhydric states, in particular those induced by the group of PPIs, may impair the absorption of calcium in the body[51]. Overall, the studies suggested that calcium absorption potentially was affected negatively only in the setting of reduced acid secretion.

There have been several reported cases of hypomagnesemia that were associated with the long-term PPI use in various conditions, the mechanism yet to be known[52]. One of the studies suggested that the health care providers should consider checking magnesium levels in patients who are anticipated to be on long-term PPI's.

Gastric acid is involved in the absorption of Vitamin B_{12} by facilitating its release from the dietary protein, such that B_{12} can bind to the same. Because

B_{12} absorption is dependent on gastric acid, theoretically, long-term PPI use impairs an individual's absorptive ability of the same[53]. Many studies point out to the drug interactory mechanisms with the drugs like clopidogrel in its antiplatelet action, when administered along with the PPI's[54].

Several studies have focused on assessing the risk between the continuous use of the PPI and the community-acquired and the hospital-acquired pneumonia in European countries.[55] We should be aware of the potential adverse relationship between short-term and high-dose PPI use and acquired pneumonia.

Recent studies conclude the thought raised from previous studies that of a higher risk of Clostridium difficile infection among the chronic PPI users[56]. PPI exposure as a risk factor for enteric infections in travellers has not been studied in a detailed manner.

A few studies have suggested small intestinal bacterial overgrowth that is associated with bloating, diarrhea, and malabsorption, associated with PPI use, although the significance of the association is yet to be clarified[57]. Recent reports have suggested that there is a relationship between the PPI use and development of spontaneous bacterial peritonitis in cirrhotic patients with ascites. A study on the co administration of the PPIs with the methotrexate estimated that there was a considerable reduction in the overall clearance of the drug.[58] Liver cirrhotic patients are at a higher risk of serious bacterial infections, as per a sreported study.[59]

One of the observational studies point to the possibility of the increased cardiovascular risk in the patients on non systematic use of the PPI's. It elevates the Plasma Assymmetrical Dimethyl Arginine (ADMA) which is an

inhibitor of nitric oxide synthase which leads to reduced ntric oxide levels and hence reduces the endothelium dependant vasodilatation[60].

Even though the drug is effective, the clinical risk/benefit of any medical intervention or therapy always should be evaluated for each patient and the appropriate use of therapy should be directed accordingly. The physician is having the last word regarding the same, in any system of medicine. Because PPIs are overprescribed in many patients, in particular for continuos long-term use, the clinical effects always should be reviewed and attempts should be justified to terminate any therapy that may not be ultimately necessary for the benefit of the affected ones. That can be considered as a universal drug prescription formula for any practitioner and his ultimate aim is to protect the body of the patient at all levels.

MASTER DRUG CHART										
No.	**Name**	**Botanical name**	**Family**	**Rasa**	**Guna**	**Veerya**	**Vipaka**	**Chemical composition**	**Pharmacological action**	**Doshaharatwa**
1	Saptachada	Alstonia scholaris	Apocynaeceae	Tikta, Kashaya	Laghu, snigdha	Ushna	Katu	Echitamine Echitamidine	Deepana ,Hridya	Tridoshahara
2	Parpata	Hedytois corymbosa	Fumariaceae	Tikta	Laghu	Seeta	Katu	Fumaric acid Fumatine	Grahi, trisnahara	Kapha pittahara
3	Shampaka	Cassia fistula	Caesalpinaceae	Madhura	Mridu, guru, Snigdha	Seeta	Madhura	Pectin, Hydroxy methyl anthraquinone	Sramsana	Kapha pittahara
4	Katuka	Neopicrorhiza scrophulariiflora	Scrophulariaceae	Tikta	Ruksha, laghu	Seeta	Katu	Picrorhizin, Picrorhizetin Cathartic acid	Bhedana, lekhana, deepana, hridya	Kapha pittahara
5	Vacha	Acorus calamus	Araceae	Katu, tikta	Laghu, Tikshna	Ushna	Katu	Acorin, acoritin	Lekhana, medhya	Kapha vathahara
6	Hareethaki	Terminalia chebula	Combretaceae	Kashaya,madhura, amla, katu, tikta	Laghu, rooksha	Ushna	Madhura	Chebulinic acid, tannic acid, myrobalanin	Anulomana, rasayana, lekhana	Tridoshahara
7	Vibheetaki	Terminalia bellerica	Combretaceae	Kashaya	Ruksha, laghu	Ushna	Madhura	Saponin	Bhedana, Kesya, Chakshushya	Kapha pittahara
8	Amalaki	Emblica officinalis	Euphorbiaceae	Amla, madhura, katu, tikta, kashaya	Snigdha	Seeta	Madhura	Phyllemblin, Linolic acid, Ellagic acid	Rasayana, vayashthapana, vrishya	Tridoshahara

9	Padmaka	Prunus cerasoides	Rosaceae	Kashaya, tikta	Laghu, snigdha	Seeta	Katu	Genistin, prunatin, Cerasin	Varnya, vrishya vedanasthapana,	Kaphapittahara
10	Paata	Cyclea peltata	Menispermaceae	Tikta, Kashaya	Laghu, tikshna	Ushna	Katu	Hyatine, Menismine, Cissamine	Vishagna, grahi, balya	Vatha kaphahara
11	Haridra	Curcuma longa	Scitaminae	Tikta, katu	Ruksha, laghu	Ushna	Katu	Curcumine, Cineole, β - siosterol	Lekhana, vishagna, varnya	Kapha vathahara
12	Daruharidra	Berberis aristata	Berberidaceae	Tikta, kashaya	Laghu, ruksha	Ushna	Ushna	yperosid	takalamine, oxycantine, berberine	Kapha pittahara
13	Sweta sariba	Hemidesmus indicus	Asclepidiaceae	Madhura, tikta	Guru, snigdha	Seeta	Madhura	Hyperoside, Desinine,β - sitosterol	Grahi, raktaprasadana	Tridoshahara
14	Krishna sariba	Ichnocarpus frutecens	Asclpdiaceae	Madhura, tikta	Guru, snigdha	Seeta	Madhura	Hyperoside, Desinine,β - sitosterol	Grahi, vrishya,raktaprasadana	Tridoshahara,
15	Pippali	Piper longum	Piperaceae	Katu	Laghu, snigdha, teekshna	Ushna	Madhura	Caryophyllen, piperin	Deepana, vrishya, rasayana	Kaphavatha hara
16	Gajapippali	Piper chaba	Piperaceae	Katu	Laghu, snigdha, teekshna	Ushna	Madhura	Caryophyllen, piperin	Deepana, vrishya, rasayana	Kaphavatha hara
17	Nimba	Azadirachta indica	Meliaceae	Tikta, kashaya	Laghu, ruksha	seeta	Katu	Nimbin, sitosterol, Azadirachtanin	Deepana, grahi,krimighna	Kaphapittahara
18	Chandana	Santalum album	Santalaceae	Tikta, madhura	Laghu, rooksha	Seeta	Katu	Santalic caid, Plamitone,	Varnya, Dahasamana	Kaphapittahara
19	Yashtimadhu	Glycyrrhiza glabra	leguminosae	Madhura,	Guru, snigdha	Seeta	Madhura	Glycerrhizin,Liquirtin, Glabrene	Rasayana, vrishya	Tridoshahara
20	Indrayava	Holarrhena antidysentrica	Apocynaceae	Tikta, Kashaya	Laghu, rooksha	Seeta	Katu	Canessidine, Holarrhimine, Holacetine	Grahi, deepana	Kapha pittahara
21	Vasa	Adathoda vasaca	Acanthacaea	Tikta, Kashaya	Laghu, ruksha	Seeta	Katu	Vascicine, Adathodic acid	Hridya, swarya	Kaphapittahara

22	Moorva	Chonemorpha fragrans	Asclepidiaceaea	Tikta, Kashaya	Guru, ruksha	Ushna	Katu	Marsedin, D- canar	Jwrahara	Kapha vathahara
23	Satavari	Asparagus racemocus	Liliaceaea	Madhura, tikta	Guru, snigdha	Seeta	Madhura	Spirsanolic, sarsapogenin, sitosterol	Rasayana, Vrishya	Vatapittahara
24	Patola	Trichosanthus diocia	Cucurbitaceae	Tikta, katu	Laghu, rooksha	Ushna	Katu	Colocynthin, Trichosanthin, Hentriacontane	Varnya, deepana, vrishya	Kaphapittahara
25	Musta	Cyperus rotundus	cyperaceae	Tikta, katu, kashaya	Laghu, rooksha	Seeta	Katu	Cyperolone, Rotundone	Lekhana, grahi, pachana	Kapha pittahara
26	Trayanti	Gentiana kurroo	Gentianaceae	Tikta, kashaya	Laghu, rooksha	Ushna	Katu	Catalpol, aucubin	Vishagna, rechaka	Pitta kaphahara
27	Kiratatikta	Swertia chirayata	Gentianaceae	Tikta	Laghu,roo ksha	Sheeta	Katu	Amarogentin, Gentiopicrine, Chiratol	Sodhaka, stanyajanaka	Kaphapittahara
28	Ushira	Vetiveria zizanioides	Graminae	Tikta, madhura	Laghu, rooksha	seeta	Katu	Allakhusiol, benzoic acid, eugenol	Pachana, stambana	Kapha pittahara
29	Vishala	Citrullus colocynthis	cucurbitaceae	Tikta	Sara, laghu	Ushna	Katu	A- Elaterin, Phytosteron, saponin	Vishagna, amadoshahara	Kapha pittahara
30	Dhanvayasha	Fagonia arabica	Zygophyllaceae	Tikta, madhura	laghu	seeta	Madhura	Melizitose	Dahasamana	Kapha pittahara
31	Amrutha	Tinospora cordifolia	Menispermaceae	Katu, tikta	laghu	Ushna	Madhura	Gilion, giliosterol	Samgrahi, deepana, rasayana	Tridoshahara
32	Ativisha	Aconitum heterophyllum	Ranunculaceae	Tikta, katu	Laghu	Ushna	Katu	Atisine, aconitic acid	Grahi, balya	Kapha pittahara

Chapter - 5

METHODOLOGY

From the time immemorial, man is in search of better and better options for improving the quality of his life. He was always very much conscious and eager about his own health and its care. The alterations in the lifestyle and also the working conditions contributed to the manifestation of many a disease. Not a few of them were not responding as expected, with the then available medicines. That turned his attention to research for greater piece of knowledge. Eventhough the modern research techniques are not fully athletic to the Ayurvedic system of medicine, evidence based studies are very much essential for the global acceptance of the science.

In any form of research, the methodology is the key component. In this study, the selected drug was tried for the efficacy with the commonest drug in practice, Omeprazole in Functional dyspepsia. The design used was a Randomized Controlled Clinical Trial. The selected patients were allocated into two groups using a system generated random number table after receiving proper written consent.

MATERIALS AND METHODS

Aims and Objectives

1. To evaluate the role of Mahatiktakam kwatham Tablet in Amlapitta with special reference to Functional Dyspepsia
2. To compare the efficacy of Mahtiktakam kwatham Tablet with Omeprazole in Amlapitta with special reference to Functional Dyspepsia.

Research Question

Is there any significant difference in the efficacy of Mahatiktakam kwatham tablet when compared with the Allopathic drug, Omeprazole in reducing the symptoms of Amlapitta with special reference to Functional Dyspepsia when administered continuously for 30 days.

Null Hypothesis

There is no significant difference in the role of Mahatiktakam kwatham tablet when compared with Omeprazole in Amlapitta with special reference to Functional dyspepsia.

Alternate Hypothesis

There is significant difference in the role of Mahatiktakam kwatham tablet when compared with Omeprazole in Amlapitta with special reference to Functional dyspepsia.

Materials

Patients/ Drugs/ Case Record Form / Written consent form

Drugs

Study drug

Mahatiktakam Kwatham tablet

prepared from Arya Vaidya Sala, Kottakkal with batch number 123/ 1/ 2013.

1 gm of the medicine of active ingredients of the respective kwatham was there in each of the tablets.

Dosage and duration

2 tablets thrice daily, to be administered, with warm water, 1 hour before food, continuously for 30 days.

Control drug

Omeprazole - 20 mg capsule, sufficient in quantity was purchased from Micro labs limited, Solan district, Himachal Pradesh with batch no. OMADOO5/ 06/ 2013

Dosage and duration

One capsule daily 1 hour before lunch, continuously for 30 days.

Study Design

Randomized Controlled Clinical Trial

Randomization was done by the random number table of 200 subjects which was created using the Random number generator software.

The protocol was submitted for clearance and approved by the research committee of the Tilak Maharashtra Vidyapeeth, Pune.

Sample Size

Calculation of sample size: The sample size was calculated assuming the prevalence rate of the disease as 15% of the population, with a probability for type I error fixed at 5% ($Z\alpha = 1.96$) and an effect size of 5%. The calculated sample size was 195. Expecting a dropout of 5%, the final sample size calculated was 200 which were equally divided, among the 2 groups. The samples in each group were allocated according to the computer generated random number table. (Appendix X)

Duration of the study

24 months from June 2012 onwards

Setting

OPD and IPD of Kayachikitsa Department and the Gastroenterology OPD, Vaidyaratnam P S Varier Ayurveda College, Kottakkal .

Diagnostic Criteria

I . Features of Amlapitta

1. Daha
2. Amlodgara
3. Chardi
4. Avipaka
5. Soola

Atleast 3 symptoms from the duration of more than 3 months included

II . Functional Dyspepsia

ROME III DIAGNOSTIC CRITERIA

Those with the following:

a. Bothersome postprandial fullness

b. Early satiation

c. Epigastric pain

d. Epigastric burning

AND

- No evidence of structural disease (including at upper endoscopy) that is likely to explain the symptoms
- Criteria fulfilled for the last 3 months with the symptom onset at least 6 months prior to the diagnosis

Inclusion Criteria

- ✓ Participants fulfilling the proposed diagnostic criteria
- ✓ Upper GI Endoscopy negative for structural changes
- ✓ Age : 20 - 50 years
- ✓ No discrimination of sex, caste, religion and economic status
- ✓ Participants from whom the written consent is obtained

Exclusion Criteria

- × Those with the endoscopic findings of peptic ulcer or gastritis
- × Endocrine disorders - DM, Thyroid disorders etc.
- × Other systemic disorders- CAD, HT, Bronchial asthma, Dyslipidaemia etc.
- × Those on NSAID's, antibiotics or other long-term and continuous medication
- × Pregnant women and lactating mothers

Assessment Criteria

- Changes in the Gastrointestinal Symptom Rating Scale (GSRS)
- Changes in the Amlapitta rating scale

Assessment

There were three assessments throughout the study after the baseline evaluation

- Before starting the medication
- 15 days after starting the medication
- After completion of 30 days of the medication
- After 45 days from the commencement of the medication

Those who were positive in the Rapid Urease Test for the H pylori were also given the respective medicine, as per the randomization. After full course of intervention and on the 60th day of the commencement, they were advised to perform the post test, ie. Immunocomb II Helicobacter pylori IgG kit and the results were observed and recorded.

Observations and analysis

The observations were recorded in both the groups and was compared at the baseline and at the end of 15 days, 30 days and 45 days of assessment for the GSRS score as well as the Amlapitta rating scale, using the selected statistical tests - unpaired t test between groups, one way ANOVA for comparing within groups, Tuckey Kramer test for multiple comparison, Chi square test for association between the various factors. Also the correlation and regression test were done accordingly. Insta Graph pad 1.0 version and Microsoft Excel 07 were used for performing the statistical tests.

Chapter - 6

OBSERVATION, ANALYSIS & INTERPRETATION

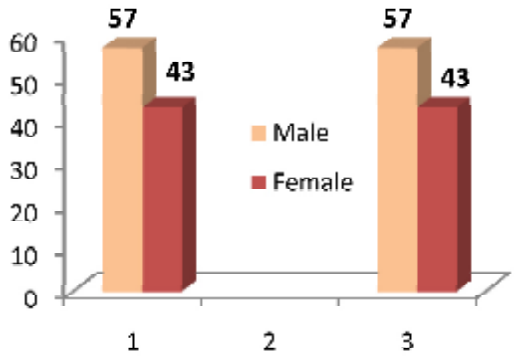
As per the calculated sample size, 200 subjects were recruited for the study, who were fulfilling the diagnostic as well as the inclusion criteria and provided with due written consent. They were randomized into two groups, the study group and control group as per the software generated random number table. Of the 200 subjects, there were 10 dropouts overall, before the completion of all the assessments, 5 each from both the groups and carry forward strategy was applied here, during the analysis. The observations recorded are being presented here for detailed inference.

Table 21 Demographic Data

Distribution	Criteria	Trial	Control
Sex	Male	57	57
	Female	43	43
Age group	20-30	24	26
	31-40	26	22
	41-50	50	52
Religion	Hindu	41	35
	Muslim	57	63
	Christian	2	2
Marital Status	Unmarried	22	23
	Married	75	72
	Divorced	2	5
	Widow	1	0
Economic Status	Poor	19	17
	Middle Class	78	78
	Rich	3	5
Education	Illiterate	1	0
	Primary	12	7
	Secondary	38	30
	Undergraduate	35	36
	Graduate	13	23
	Postgraduate	1	4
Occupation	Home manager	36	33
	Clerical	4	7
	Physical exertion	30	26
	Business	21	14
	Professional	6	12
	Student	3	8

Distribution according to Sex

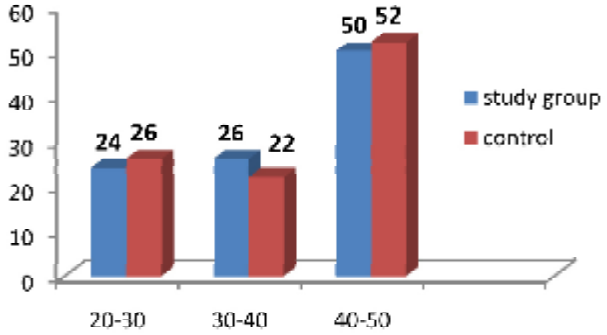
Among the 100 patients in the trial group, 57 were males and the rest 43, female subjects. In the control group also there were 57 males and 43 females included, among the subjects.



Graph 1 - Sex Distribution

Distribution according to Age group

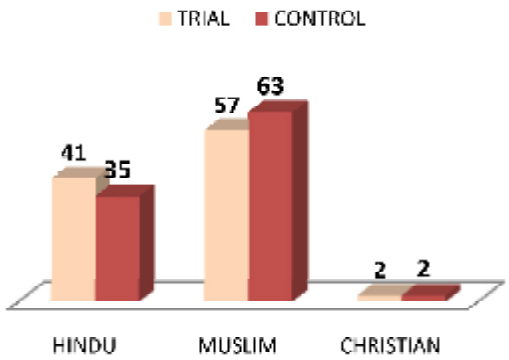
In the trial group, among the 100 patients, 24 were of the age group 20 - 30 years while in the control group there were 26 subjects. In the age group 31- 40, there were 26 subjects in the trial group and 22 from the control group. In the age group 41 – 50, 50 subjects were from the trial group while 52 from the control group.



Graph 2 - Age group

Distribution according to Religion

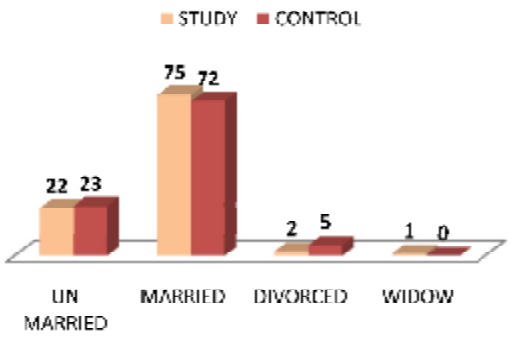
Among the 100 subjects in the trial group, 41 were Hindus, 57 were Muslims and there were 2 Christians. In the control group, there were 35 Hindus, 63 Muslims and 2 subjects were of Christian community.



Graph 3 - Religion

Distribution according to Marital status

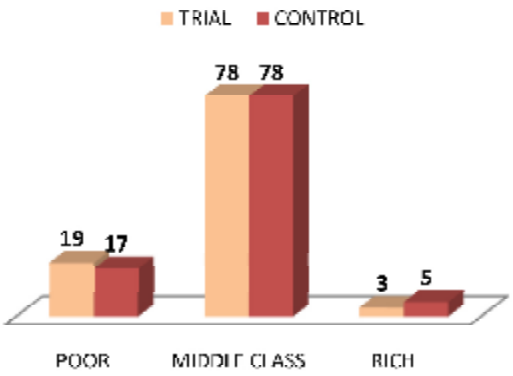
Among the 100 participants in the trial group, 22 were unmarried, 75 were married, 2 were divorcee and there was 1 widow. In the control group, 23 were not married, 72 were married and 5 were divorced.



Graph 4 - Marital status

Distribution according to Economic status

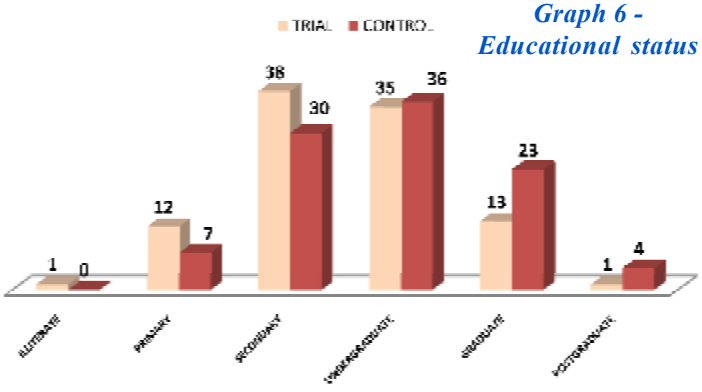
Among the 100 subjects in the trial group, 19 were of poor economic status, 78 were from the middle class, 3 were rich. In the control group, 17 were poor, 78 were from the middle class and 5 were rich.



Graph 5

Distribution according to Educational status

Among the 100 individuals in the trial group, 1 was illiterate, 12 were having primary education, 38 were with secondary

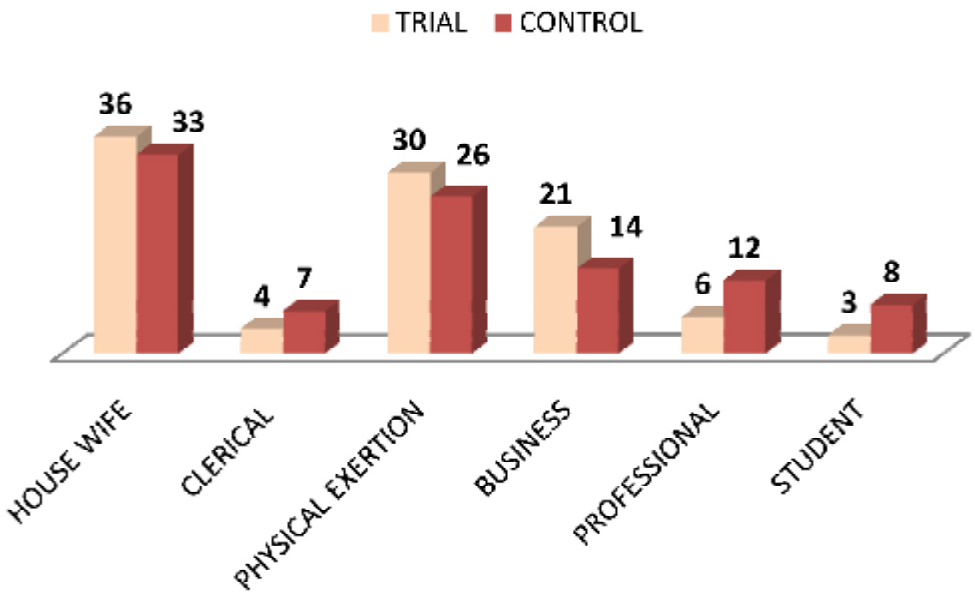


Graph 6 - Educational status

education, 35 were undergraduates, 13 were graduates and 1 was a post graduate. In the control group out of 100, there were no illiterates, 7 were with only primary education, 30 were of secondary level of education, 36 were undergraduates, 23 were graduates and 4 were post graduates.

Distribution according to Occupation

Among the 100 included in the trial group, as per the occupation, 36 were home managers, 4 of clerical job, 30 were doing a job with physical exertion, 21 were with business, 6 were professionals and 3 were students. Among the 100 in the control group, 33 were home managers, 7 were of clerical job, 26 were with a job having physical exertion, 14 were have business, 12 were professionals and 8 were students.



Graph 7 - Occupational status

Table 22 Vital parameters

Parameter	Mean ± SD	
	Trial group	Control group
Weight	58.07 ± 6.62	59.97 ± 12.38
Height	161.47 ± 6.8	161.39 ± 12.01
Pulse rate	75 ± 3.3	74 ± 2.85
Heart rate	74.8 ± 2.86	75.1 ± 2.36
Respiratory rate	15.07 ± 1.26	15.24 ± 1.42
Systolic BP	122.4 ± 10.46	121.5 ± 15.08
Diastolic BP	78.92 ± 3.46	79.7 ± 3.74
Body temperature	98.38 ± 0.21	98.1 ± 0.18

Distribution according to Weight

In the trial group, the mean weight of the subjects were 58.07 with a standard deviation of 6.62. In the control group, the mean weight was 59.97 with a standard deviation of 12.38.

Distribution according to Height

In the trial group, the mean height of the subjects were 161.47 with a standard deviation of 6.8. In the control group, the mean weight was 161.39 with a standard deviation of 12.01

Distribution according To Pulse rate

In the trial group the mean score of pulse rate was 75 with a standard deviation of 3.3, while in the control group, it was 74 with a S D of 2.85.

Distribution according to Heart rate

In the trial group, the mean score of heart rate was 74.8 with a Standard deviation of 2.86, while in the control group, it was 75.1 with a S D of 2.36.

Distribution according to Respiratory rate

In the trial group, the mean score of respiratory rate was 15.07 with a Standard deviation of 1.26, while in the control group, it was 15.24 with a Standard deviation of 1.42.

Distribution according to Blood pressure

In the trial group the mean score of systolic blood pressure was 122.4 with a Standard deviation of 10.46, while in the control group, it was 121.5 with a S D of 15.08. In the trial group, the mean score of diastolic blood pressure was 78.92 with a Standard deviation of 3.46, while in the control group, it was 79.7 with a Standard deviation of 3.74.

Distribution according to Body temperature

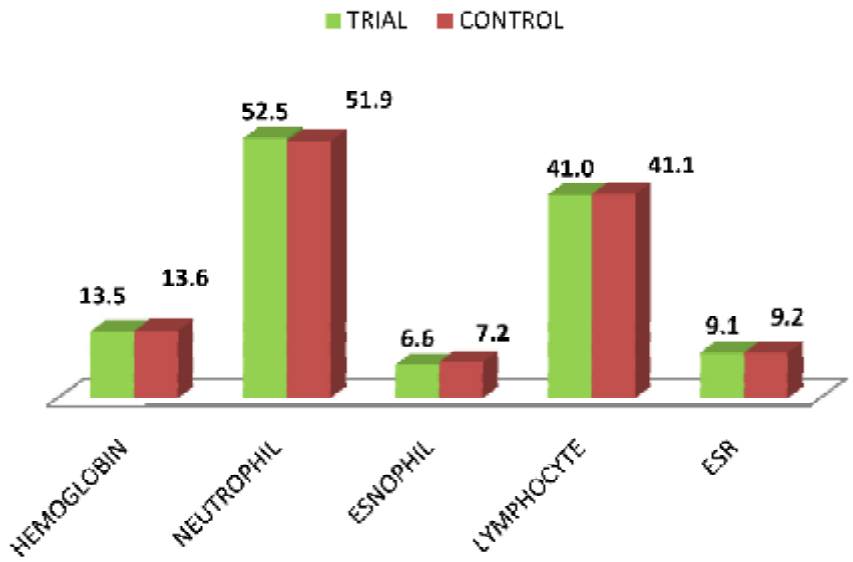
In the trial group the mean score of body temperature was 98.38 with a Standard deviation of 0.21, while in the control group, it was 98.1 with a Standard deviation of 0.18

Routine Blood examination

Table 23 Mean score of routine blood examination

Blood test	Mean ± SD	
	Trial	Control
Hemoglobin	13.5 ± 0.98	13.6 ± 0.9
Neutrophil	52.5 ± 4.11	51.9 ± 3.6
Esnophil	6.6 ± 1.93	7.2 ± 2.5
Lymphocyte	41.0 ± 4.09	41.1 ± 3.7
ESR	9.1 ± 4.29	9.2 ± 4.1
Leukocyte count	7718 ± 933.3	7889 ± 430.2
Random blood sugar	109.11 ± 14.76	112.78 ± 19.56
Total cholesterol	197.34 ± 12.23	201.79 ± 11.29

In the trial group, the mean score of hemoglobin was 13.5 with a standard deviation of 0.98 while in the control group it was 13.6, with a SD of 0.9. In the trial



Graph 8: Mean scores of routine blood examination

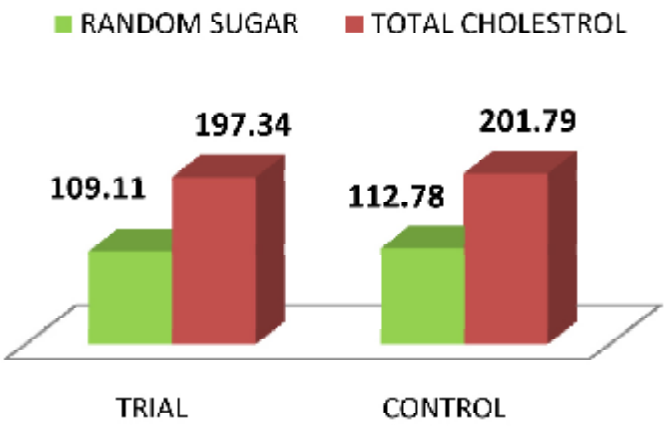
group, the mean score of neutrophil was 52.5 with a SD of 4.11 while in the control group it was 51.9 with a SD of 3.6. In the trial group, the mean score of esnophil was 6.6 with a Standard deviation of 1.93 while in the control group it was 7.2 with a SD of 2.5. In the trial group, the mean score of lymphocyte was 41 with a Standard deviation of 4.09 while in the control group, it was 41.1 with a SD of 3.7. In the trial group, the mean score of ESR was 9.1 with a Standard deviation of 4.29 while in the control group, it was 9.2 with a SD of 4.1.

Total leukocyte count

In the trial group, the mean score of total leukocyte count was 7718 with a Standard deviation of 933.3, while in the control group, it was 7889 with a Standard deviation of 430.2.

Random blood sugar and total cholesterol

In the trial group, the mean score of random blood sugar was 109.11 with a Standard deviation of 14.76, while in the control group, it was 112.78 with a SD of 19.56. In the trial group the mean score of total cholesterol was 197.34 with a Standard deviation of 12.23, while in the control group, it was 201.79 with a SD of 11.29.



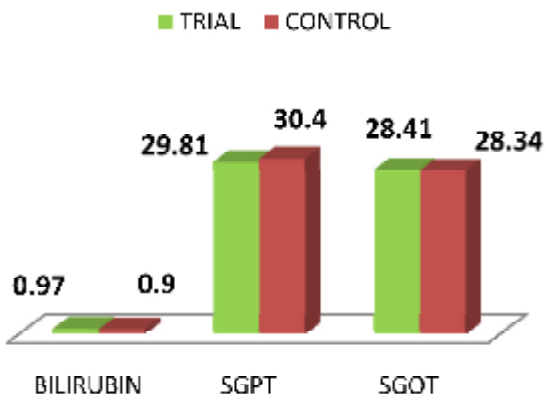
Graph 9:
RBS & Total Cholesterol

Liver Function Tests

Table 24 Mean values of LFT between groups

LFT	Mean ± SD	
	Trial	Control
Bilirubin	0.97 ± 0.17	0.9 ± 0.18
SGPT	29.81 ± 8.93	30.4 ± 6.37
SGOT	28.41 ± 7.51	28.34 ± 10.93

In the trial group the mean score of total bilirubin was 0.97 with a Standard deviation of 0.17, while in the control group, it was 0.9 with a SD of 0.18. In the trial group, the mean score of SGPT was 29.81 with a Standard deviation of 8.93, while in the control group, it was 30.4, with a SD of 6.37. In the trial group, the mean score of SGOT was 28.41 with a Standard deviation of 7.51, while in the control group, it was 28.34 with a SD of 10.93.



Graph 10:
Mean LFT between groups

ABDOMINAL EXAMINATION

Shape of the abdomen

In the trial group, 22 persons were having scaphoid abdomen, 24 were with flat abdomen and 54 were with distended abdomen. In the control group, 19

subjects were having scaphoid abdomen, 28 were with flat abdomen and 51 were having distended abdomen.

Table 25 Distribution according to abdominal shape

Shape of abdomen	Trial	Control
Scaphoid	22	19
Flat	24	28
Distended	54	51

In 10 % of the subjects in the trial group, scar was present on the abdominal wall, while in the control group it was seen in 21% of them. No one in both the groups presented with an abdominal wall with distended veins. Striae were present in 35% of the patients in the trial group and 36% in the control. Visible peristalsis was seen in 2% of the subjects in the trial group and 3% among the control group. Movement of abdominal wall on respiration was there, in all the individuals of both the groups.

Visible pulsation was observed in 8 subjects in the trial group and 2 subjects in the control. The feel of abdomen was soft and tender in 78 subjects of the trial and 72 of the control group. The feel was rigid and tender in 22 persons of the trial group and 28 of the control. Shifting dullness or fluid thrill was not elicited in any of the patients, from both the groups. Peritoneal rub or arterial bruit was also not able to observe, in any of them.

The percussion note was tympanitic in 7 subjects of the trial group and resonant in 93 of them. In the control group, the note was tympanitic in 3, resonant in 96 subjects and dull note in one of them. Peristaltic sounds were audible in all the subjects of both the groups.

Shape of the umbilicus

Table 26 Distribution as per the shape of the umbilicus

Shape of umbilicus	Trial	Control
Everted	3	4
Inverted	10	8
Normal	87	88

In the trial group, 3 subjects were having everted umbilicus, 10 were of inverted and 87 were with the normal umbilicus. In the control group, 4 subjects were having everted umbilicus, 8 were of inverted and 88 were having a normal umbilicus.

Distribution of tenderness in abdomen

Table 27 Distribution according to the elicited tenderness

Tender Area	Trial	Control
Rt hypochondrium	17	10
Epigastrium	98	97
Lt hypochondrium	12	10
Rt lumbar	15	13
Umbilical	80	78
Lt lumbar	3	3
Rt inguinal	1	0
Hypogastrium	2	0
Lt inguinal	2	0

On clinical examination of all the nine areas of the abdomen, tenderness was elicited in an assorted manner. In the trial group, 17% had tenderness on right hypochondrium, 98% perceived tenderness on epigastrium, 12% had tenderness on

the left hypochondrium, 15% on right lumbar, 80% had tenderness on umbilical region, 3% reported tenderness on the left lumbar region, 1% on the right inguinal region, 2% each on the hypogastrium and left inguinal region.

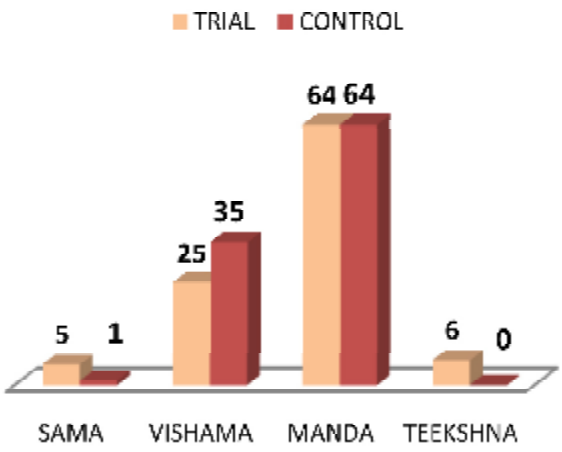
In the control group, 10% had tenderness on the right hypochondrium, 97% complained of tenderness on the epigastrium, 10% had the same on left hypochondrium, 13% felt tenderness on the right lumbar, 78% had tenderness on the umbilical region, 3% on left lumbar region, no one were having tenderness on the right inguinal region, the hypogastrium and the left inguinal region, on palpation.

FACTORS IN RELATION WITH FOOD AND DIGESTION

Table 28 Distribution as per the status of agni

Agni	Trial	Control
Sama	5	1
Vishama	25	35
Manda	64	64
Teekshna	6	0

Among the 100 subjects in the trial group, 5 were having samagni, 25 were of vishamagni, 64 were having mandagni and 6 with teekshna agni. Among the 100 subjects in the control group, only one was having samagni, 35 were of vishamagni, 64 were with mandagni and none was having teekshna agni.

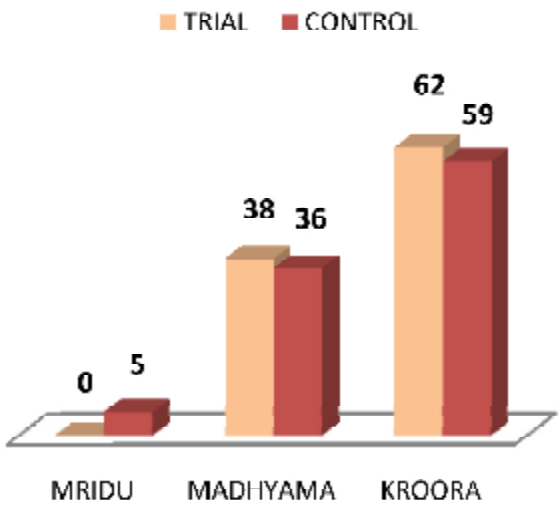


Graph 11: Status of agni

Table 29 Distribution according to the status of Koshta

Koshta	Trial	Control
Mridu	0	5
Madhyama	38	36
Kroora	62	59

Among the 100 participants in the trial group, none was having mridu koshta, 38 were with madhyama koshta, and 62 were of kroora koshta. In the 100 subjects of the control, 5 were having mridu koshta, 36 were of madhyama koshta and 59 with kroora koshta.



Graph 12: Status of Koshta

While considering the desa of the subjects, one was from jangala desa, one from anoopa desa and the rest 98 from sadharana desa. In the control group, 4 were from anoopa desa and the rest 96 were from the sadharana desa. Among the sara, in the trial group 96 were of madhyama sara and the rest 4 of avara in sara. In the control group, 98 were of madhyama sara and 2 were with avara in sara.

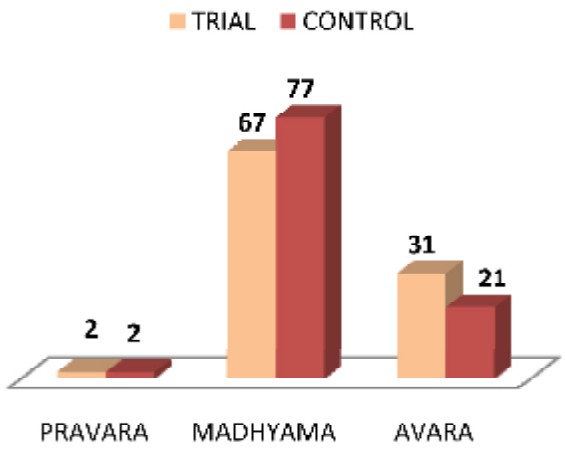
In the case of samhanana, in the trial group 98 were of madhayama and the rest 2 of avara in nature. In the control group, out of the 100 included, 95 were of madhyama and the rest 5, avara in samhanana. In pramana, among those in the trial group, 3 were of pravara, 94 madhyama and the rest 3 of avara in pramana. In the

control group, 2 were of pravara, 95 of madhyama and the rest with avara, in pramana. While considering the satmya, in the trial group 97 were of madhyama satmya and the rest 3 of avara in satmya. In the control group, 2 were of pravara in satmya, 97 of madhyama satmya and 1 with satmya of avara in nature.

Table 30 Distribution according to satwa

Satwa	Trial	Control
Pravara	2	2
Madhyama	67	77
Avara	31	21

In the trial group, 2 subjects were of pravara satva, 67 of madhyama in satva and 31 of avara satwa while in the control 2 were of pravara satva, 77 of madhyama satva and 21 persons with avara in satva.



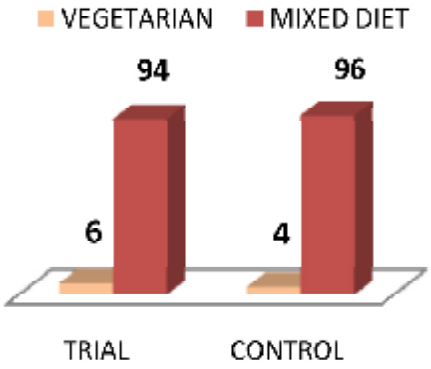
Graph 13: Distribution of Satwa

Regarding vyayama sakthi, in the trial group it was pravara in 3 persons, madhyama in 88 and avara in 9 of them. In the control group, the vyayama sakthi was pravara in 2 individuals, madhyama in 84 and avara in 14 of the subjects. While considering the vaya, all the subjects in both the trial and control groups were in the youvana category due to the peculiarity of the age limit, kept as the inclusion criteria.

Table 31 Distribution as per the type of the food

Type of food	Trial	Control
Vegetarian	6	4
Mixed diet	94	96

Among the 100 individuals in the trial group, 6 were vegetarians and the rest 94 were on mixed diet. Among the 100 in the control group, 4 were vegetarians and the rest ie. 96 were having a mixed diet.

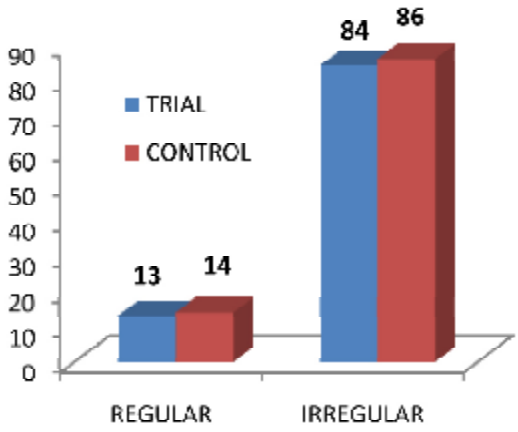


Graph 14
Type of the food

Table 32 Distribution according to the timing of the meals

Timing of meals	Trial	Control
Regular	13	14
Irregular	84	86

Among the 100 subjects included in the trial group, 84% were not able to follow a regular pattern in their diet, while in the control, 86% were not following a regular pattern in their diet.

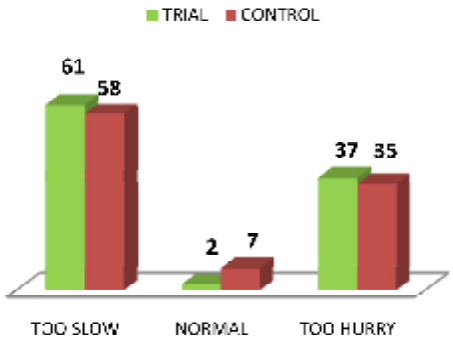


Graph 15:
Timing of the meals

Table 33 Distribution according to the pace of food intake

Food intake	Trial	Control
Too slow	61	58
Normal	2	7
Too hurry	37	35

Among the 100 subjects in the trial group, 61 were having their food at a slower pace, 2 were having food at a normal pace and the rest 37 were in a hurry, while having the food. Among the 100 in the control group, 58 were having their food at a slower pace, 7 were having food at a normal pace and the rest 35 were in a hurry, while having their food.

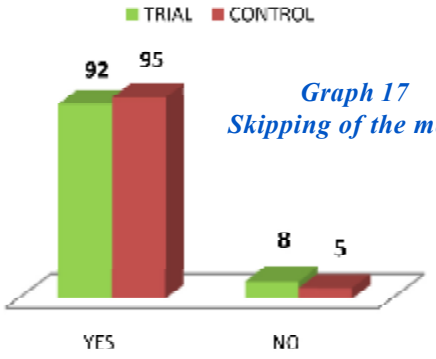


Graph 16: Food intake

Table 34 Distribution according to the skipping of meals

Skipping of meals	Yes	No
Trial	92	8
Control	95	5

In the trial group, 92% of the subjects were having the habit of skipping meals, while in the control group, 95% were skipping their meals in a regular manner.

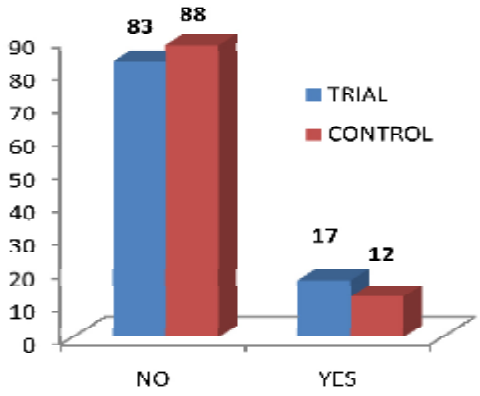


Graph 17
Skipping of the meals

Table 35 Distribution as per the intake of food after having appetite

Intake after appetite	No	Yes
Trial	83	17
Control	88	12

In the trial group, 83% of the subjects were consuming the food before feeling appetite, while in the control, 88% were having food, before they felt appetite.

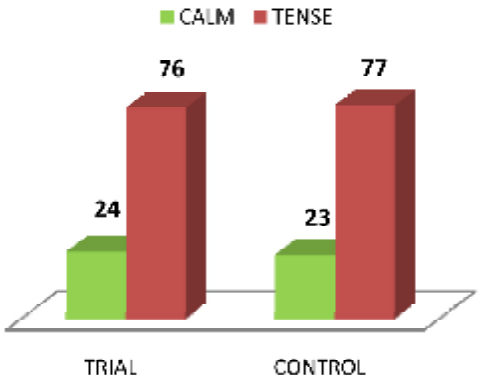


Graph 18: Intake of food after having appetite

Table 36 Distribution according to the atmosphere of food intake

Atmosphere food intake	Trial	Control
Calm	24	23
Tense	76	77

Among the 100 participants in the trial group, 24 were having food in a calm atmosphere while the rest 76 were taking it, in a tense atmosphere. Among the control, 23 were having food in a calm atmosphere while 77 were taking it, in an atmosphere which is tense.

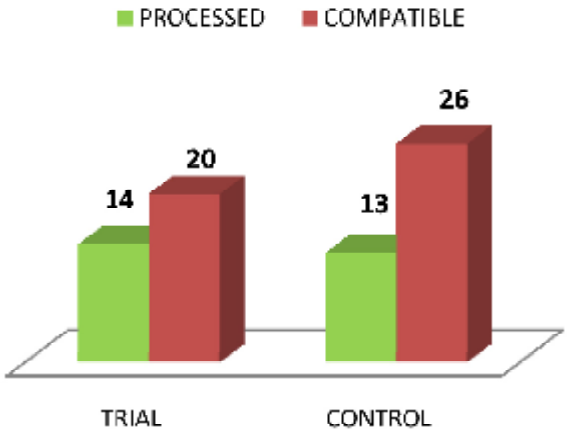


Graph 19: Atmosphere of food intake

Table 37 Distribution according to the intake of altered food

Food	Trial	Control
Processed	14	13
Compatible	20	26

14% of the individuals in the trial group were having processed food frequently while in the control, it was observed in 13% of them. 20% of the subjects in the trial group were having non compatible food while in the control group, it was observed in 26%.



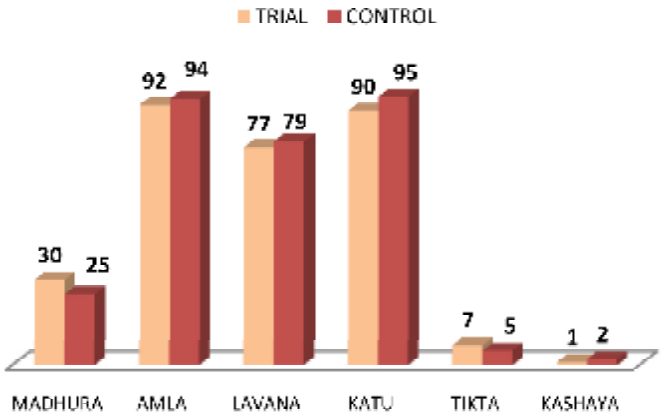
Graph 20: Intake of altered food

Table 38 Distribution according to the excess intake of rasas

Excessive use	Trial	Control
Madhura	30	25
Amla	92	94
Lavana	77	79
Katu	90	95
Tikta	7	5
Kashaya	1	2

Among the 100 subjects in the trial group as per the excessive use of rasas, 30 were using madhura rasa, 92 were using amla excessively, 77 were using lavana in excess, 90 were using katu rasa more, 7 were using tikta and 1 was using

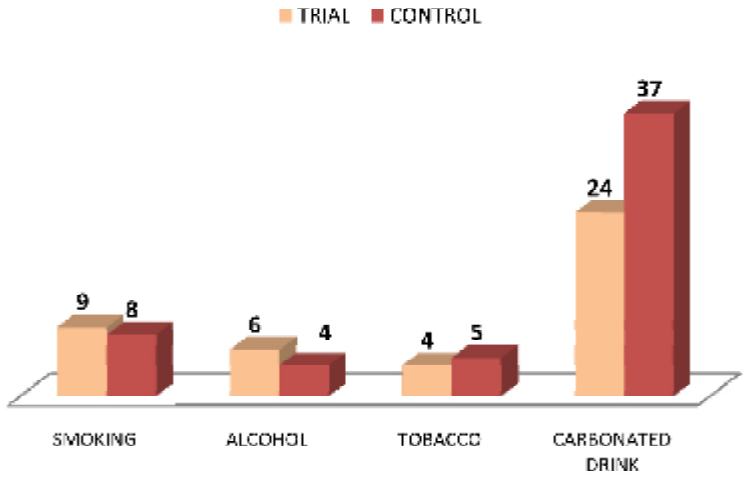
more of kashaya rasa. Among the 100 persons in the control group according to the excessive use of rasas, 25 were using more madhura, 94 were using amla excessively, 79 were having lavana in excess, 95 were using katu rasa more, 5 were using tikta and 2 were having more of kashaya rasa, in their dietary schedule.



Graph 21: Excess intake of rasas

Table 39 Distribution according to the various habits

Habit	Trial	Control
Smoking	9	8
Alcohol	6	4
Tobacco	4	5
Carbonated drink	24	37



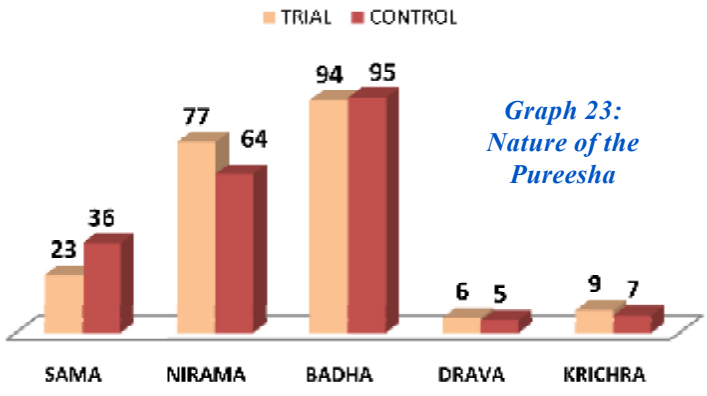
Graph 22: Distribution of habits

While assessing the existing contributory habits among the various subjects, in the trial group, 9 were smoking regularly, 6 were consuming alcohol, 4 were regularly using tobacco and 24 were having carbonated drink frequently. In the control group, 8 were with the habit of smoking, 4 were consuming alcohol, 5 were regularly using tobacco and 37 were having carbonated drinks frequently.

Table 40 Distribution according to the nature of the pureesha

Pureesha	Trial	Control
Sama	23	36
Nirama	77	64
Badha	94	95
Drava	6	5
Krichra	9	7

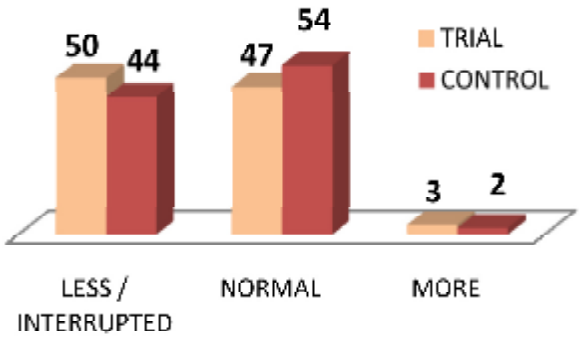
The patients were categorized according to the nature of pureesha and its pravritti. In the trial group, 23 were having sama mala, 77 were having mala which was nirama, 94 were having mala which was badha, 6 were having pureesha of drava in nature, 9 were having krichra mala pravrithi. In the control group, 36 were having sama mala, 64 were having mala which was nirama, 95 were having pureesha which was badha, 5 were having pureesha which was drava in nature, 7 were having krichra mala pravrithi.



Graph 23: Nature of the Pureesha

Table 41 Distribution according to the nature of the sleep

Nature of sleep	Trial	Control
Less / interrupted	50	44
Normal	47	54
More	3	2



Graph 24 : Nature of the sleep

In the trial group, out of 100 patients, 18 were having the habit of daytime sleep while in the control group there were 16, who were sleeping regularly, at day time. In the trial group, the mean night time sleep was 6.94 hours with a standard deviation of 1.00, while in the control, the mean night time sleep was 6.98 hours with a SD of 0.93. In the trial group, 50% of the subjects were having reduced or interrupted sleep while in control, it was 44% of whom, the sleep was interrupted.

Table 42 Distribution as per the family history of Amlapitta

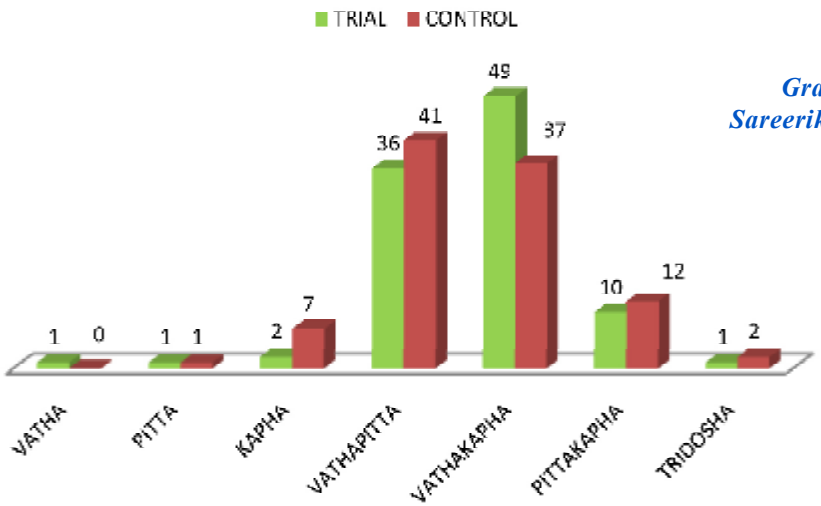
Family history	No. of patients	Percentage
Trial	31	31
Control	16	16

In the trial group out of 100 patients, 31 were having a positive family history of Amlapitta. In the control group, 16 patients were having a family history, suggestive of the disease.

Table 43 Distribution according to the sareerika prakrithi

Prakrithi	Trial	Control
Vatha	1	0
Pitta	1	1
Kapha	2	7
Vatha Pitta	36	41
Vatha Kapha	49	37
Pitta Kapha	10	12
Tridosha	1	2

On assessing the prakrithi of the included subjects, in the trial group there was one person with Vatha prakrithi, one with Pitta prakrithi, 2 with Kapha prakrithi, 36 with Vatha Pitta prakrithi, 49 with Vatha Kapha prakrithi, 10 with Pitta Kapha prakrithi and one with tridosha prakrithi. In the control group, there was no one with Vatha prakrithi, one with Pitta prakrithi, 7 with Kapha prakrithi, 41 with Vatha Pitta prakrithi, 37 with Vatha Kapha prakrithi, 12 with Pitta Kapha prakrithi and 2 with tridosha prakrithi among the included.

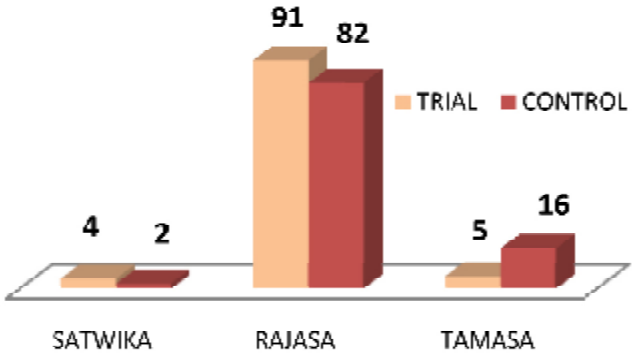


Graph 25 : Sareerika prakrithi

Table 44 Distribution according to the manasa prakrithi

Manasa prakrithi	Trial	Control
Satwika	4	2
Rajasa	91	82
Tamasa	5	16

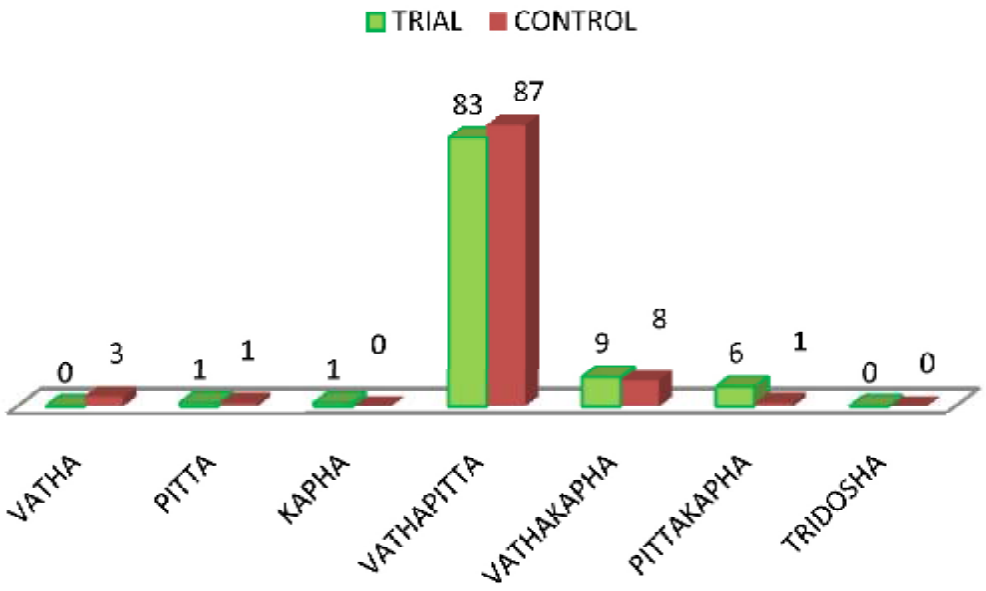
On assessing the manasa prakrithi, it was observed that, in the trial group 4 were having satvika prakrithi, 91 were of rajasa prakrithi and 5 with tamasa prakrithi. In the control group, 2 were having satvika prakrithi, 82 were possessing rajasa prakrithi and 16 were of tamasa prakrithi in nature.



Graph 26: Manasa prakrithi

Table 45 Distribution according to the Dosha status

Dosha	Trial	Control
Vatha	00	03
Pitta	01	01
Kapha	01	00
Vatha Pitta	83	87
Vatha Kapha	09	08
Pitta Kapha	06	01
Tridosha	00	00



Graph 27: Dosha status

On assessing the dosha among the included subjects, in the trial group there was one person with Vatha dosha, one with Pitta dosha, one with Kapha dosha, 83 with Vatha Pitta, 9 with Vatha Kapha dosha, 6 with Pitta Kapha and none with tridosha. In the control group, there were 3 persons with Vatha dosha, one with Pitta dosha, none with Kapha dosha, 87 with Vatha Pitta, 8 with Vatha Kapha dosha, one with Pitta Kapha and none with tridosha, among the included subjects.

Table 46 Distribution according to the involved dhatu

Dhatu	Trial	Control
Rasa	89	90
Rakta	1	2
Rasa and rakta	10	8

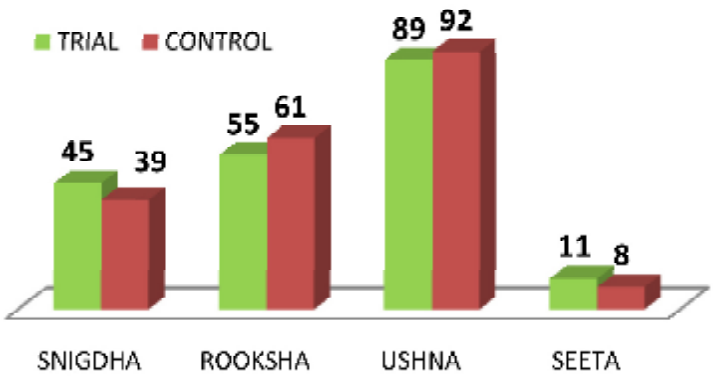
Considering the nature of the disease, among the seven dhatus from rasa to shukra, the dhatus ie. rasa, rakta and their combinations were recorded, in this

study. Among the 100 subjects in the trial group, in 89 of them rasa dhatu was affected. In one subject, rakta dhatu was involved and in 10 of them, both rasa and rakta were involved. In the control group, in 90 of the subjects rasa dhatu was involved, rakta was affected in 2 and in 8 among them, both rasa and rakta seems affected.

Table 47 Distribution according to the guna of food

Guna of food	Trial	Control
Snigdha	45	39
Rooksha	55	61
Ushna	89	92
Seeta	11	8

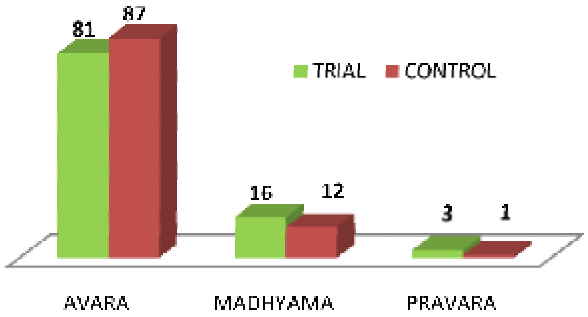
Among the 100 subjects in the trial group, 45 were using excess snigdha ahara and 55 were having rooksha ahara more. In the control group, 39 were using excess of snigdha ahara and 61 of ahara with excess in rooksha guna. In the trial group, 89 was using excess of ushna ahara and 11 were using more of seeta ahara. In the control group, 92 were using excess of ushna ahara and 8 were using seeta ahara more.



Graph 28: Guna of food

Table 48 Distribution according to the Abyavaharana sakthi

Abyavaharana	Trial	Control
Avara	81	87
Madhyama	16	12
Pravara	3	1

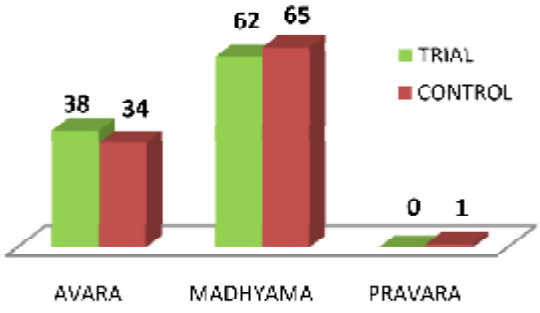


Graph 29: Abyavaharana sakthi

While considering the abyavaharana sakthi of the included, in the trial group among the 100, 81 were of avara in abyavaharana, 16 of madhyama and 3 of pravara in abyavaharana. In the control group, 87 were of avara in abyavaharana, 12 were of madhyama and one was of pravara in abyavaharana sakthi.

Table 49 Distribution according to the Jarana sakthi

Jarana	Trial	Control
Avara	38	34
Madhyama	62	65
Pravara	0	1



Graph 30: Jarana sakthi

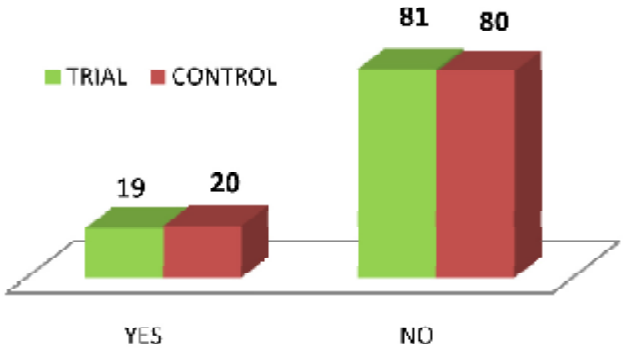
While considering the jarana sakthi of the included, in the trial group among the 100 subjects, 38 were of avara in jarana sakthi, 62 of madhyama and none with

pravara in jarana. Among the control, 34 were of avara in jarana, 65 were with madhyama and 1 of pravara, in jarana sakthi.

Table 50 Distribution according to the use of NSAID's

Frequent NSAID	Yes	No
Trial	19	81
Control	20	80

In the trial group, 19% of the subjects were using NSAID's regularly while in the control group 20% of the subjects were using NSAID's in a regular manner.



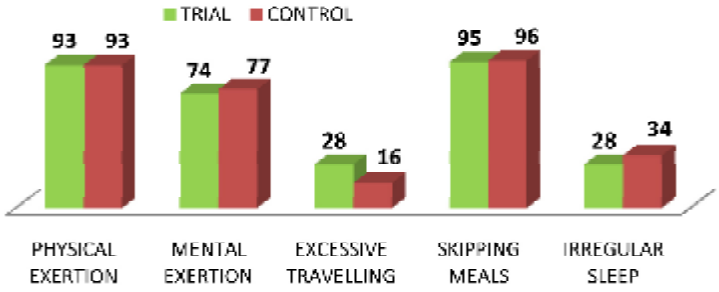
Graph 31: Use of NSAID's

Table 51 Distribution according to the factors affecting the digestion

Factors	Trial	Control
Physical exertion	93	93
Mental exertion	74	77
Excessive travelling	28	16
Skipping meals	95	96
Irregular sleep	28	34

While studying the various factors affecting the digestive process, in the trial group 93% were having physical exertion just after food intake, 74% were having

associative mental exertion, 28% were travelling excessively, 95% were skipping meals in an almost regular manner and 28% were having irregular sleep pattern. In the control group, 93% were having physical exertion just after food intake, 77% were having associated mental exertion, 16% were travelling excessively, 96% were skipping meals in a regular manner and 34% were having irregular sleep pattern.



Graph 32: Factors affecting the digestion

INDIVIDUAL SYMPTOMS OF FUNCTIONAL DYSPEPSIA

1. Abdominal Pain

The mean duration of abdominal pain in the trial group was 19.34 months with a Standard deviation of 17.02 while in the control, it was 16.76 with a SD of 14.19. Abdominal pain was reported in 99 % of the trial and 100% in the control group.

2. Fullness of Abdomen

The mean duration of fullness of abdomen in the trial group was 21.84 months with a Standard deviation of 17.41 while in the control group, it was 19.42 with a SD of 14.97. Abdominal pain was reported in 97 % in trial and 100% of those in the control.

3. Sucking Sensation

The mean duration of sucking sensation in the trial group was 15.17 months

with a standard deviation of 14.19 while in the control it was 12.13 with a SD of 10.15. Sucking sensation was reported in 35 % in the trial and 45% in the control.

Table 52 Distribution of individual symptoms of FD with duration

Symptom	Trial group		Control group	
Duration (months)	Mean ± SD	%	Mean ± SD	%
Abdominal pain	19.34 ± 17.02	99	16.76 ± 14.19	100
Fullness of abdomen	21.84 ± 17.41	97	19.42 ± 14.97	100
Sucking sensation	15.17 ± 14.19	35	12.13 ± 10.15	45
Belching	19.81 ± 11.69	91	17.62 ± 11.08	96
Acid regurgitation	11.94 ± 10.13	93	9.72 ± 7.29	88
Burning sensation	14.15 ± 11.66	99	10.96 ± 7.13	97
Nausea/ vomiting	9.62 ± 7.92	75	8 ± 5.49	71
Anorexia	19.23 ± 11.25	73	16.43 ± 10.04	81
Borborygymi	12.72 ± 5.51	33	15.04 ± 12.73	22
Increased flatus	18.02 ± 11.35	34	20.03 ± 13.5	31
Constipation	21.5 ± 11.8	93	22.3 ± 13.35	89
Post prandial fullness	20.92 ± 17.6	100	18.52 ± 12.25	100
Early satiation	19.48 ± 17.6	100	17.34 ± 12	100

4. Belching

The mean duration of belching in the trial group was 19.81 months with a standard deviation of 11.69 while in the control group, it was 17.62 with a SD of 11.08. Belching was reported in 91 % of those in the trial and 96 % of the subjects in the control.

5. Acid regurgitation

The mean duration of acid regurgitation in the trial group was 11.94 months with a standard deviation of 10.13 while in the control it was 9.72, with a SD of 7.29. Belching was reported in 93 % in the trial and 88 % of the subjects in the control.

6. Burning sensation in abdomen

The mean duration of burning sensation in the trial group was 14.15 months with a standard deviation of 11.66 while in the control group, it was 10.96 with a SD of 7.13. Burning sensation was reported in 99 % in the trial and 97 % of the subjects in the control group.

7. Nausea/ Vomiting

The mean duration of nausea/ vomiting in the trial group was 9.62 months with a standard deviation of 7.92, while in the control it was 8 with a SD of 5.49. Nausea/ vomiting were reported in 75 % of trial and 71 % of the subjects in control.

8. Anorexia

The mean duration of anorexia in the trial group was 19.23 months with a standard deviation of 11.25 while in the control, it was 16.43 with a SD of 10.04. Anorexia was reported in 73% of trial group and 81 % of the subjects in the control.

9. Borborygymi

The mean duration of borborygymi in the trial group was 12.72 months with a standard deviation of 5.51 while in the control, it was 15.04 with a SD of 12.73. Anorexia was reported in 33 % of trial and 22 % of the subjects in the control.

10. Increased flatus

The mean duration of increased flatus of abdomen in the trial group was

18.02 with a standard deviation of 11.35 while in the control, it was 20.03 with a SD of 13.5. Increased flatus was reported in 34 % in trial group and 31 % of the subjects in the control group.

11. Constipation

The mean duration of constipation in the trial group was 21.5 months with a standard deviation of 11.8, while those among the control it was 22.3, with SD of 13.35. Constipation was reported in 93 % in the trial and 89 % of the subjects among control.

12. Bothersome Post prandial fullness

The mean duration of bothersome postprandial fullness in the trial group was 20.92 months with a standard deviation of 17.6 while those in the control group it was 18.52, with a SD of 12.25. Constipation was reported in 100 % of both the groups.

13. Early Satiation

The mean duration of early satiation in the trial group was 19.48 months with a standard deviation of 17.6 while those in the control, it was 17.34 with a SD of 12. Early satiation was reported in 100 % in the trial group as well as control.

SYMPTOMS OF AMLAPITTA

1. Daha

The mean duration of daha in the trial group was 14.22 months with a standard deviation of 11.65 while in the control group, it was 10.97 with a SD of 7.27. The symptom of daha was reported in 100 % in those from the trial group and 99 % of the subjects among the control.

Table 53 Duration of symptoms of Amlapitta

Symptom Duration (months)	Trial group		Control group	
	Mean ± SD	%	Mean ± SD	%
Daha	14.22 ± 11.65	100	10.97 ± 7.27	99
Amlodgara	11.54 ± 10.2	94	9.61 ± 6.81	90
Chardi	9.72 ± 8.07	73	7.71 ± 5.22	69
Soola	19.03 ± 17.02	98	16.41 ± 13.01	99
Avipaka	17.95 ± 9.39	71	16.33 ± 9.85	72

2. Amlodgara

The mean duration of amlodgara in the trial group was 11.54 months with a standard deviation of 10.2 while in the control, it was 9.61 with a SD of 6.81. Amlodgara was reported in 94 % in the trial and 90 % of the subjects in the control group.

3. Chardi

The mean duration of chardi in the trial group was 9.72 months with a standard deviation of 8.07 while in the control group, it was 7.71 with a SD of 5.22. The symptom of chardi was reported in 73 % of the subjects from trial group and 69 % of the subjects in the control group.

4. Soola

The mean duration of soola in the trial group was 19.03 months with a standard deviation of 17.02 while among the control, it was 16.41 with a SD of 13.01. Soola was recorded in 98 % of those in the trial group and 99 % of the subjects in the control.

5. Avipaka

The mean duration of avipaka in the trial group was 17.95 with a standard deviation of 9.39 while in the control group, it was 16.33 with a SD of 9.85. Avipaka was reported in 71 % from the trial group and 72 % of the subjects in the control.

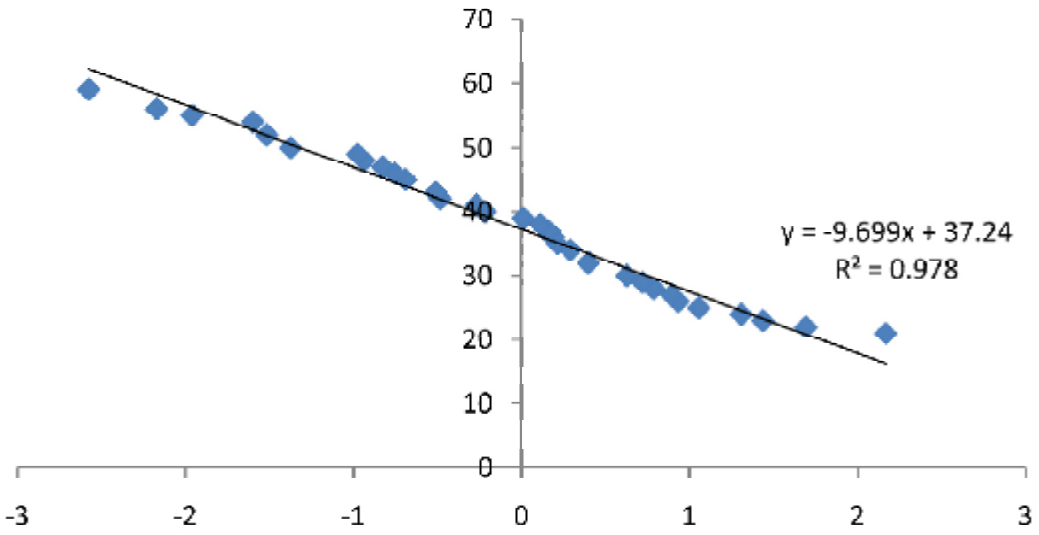
ASSESSMENT OF COMPARABILITY BETWEEN THE TWO GROUPS

1. Distribution of age between groups with Q- Q plot method

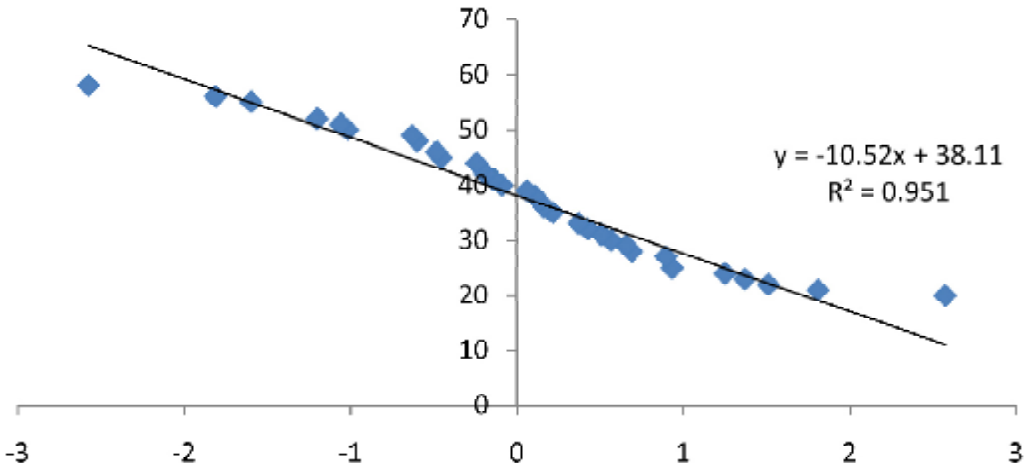
On comparing the graphs of the two groups, the distribution of age in both the groups follow normality, with very few outliers. The data thus indicates a good comparability.

2. Distribution of GSRS scores between groups with Q- Q plot method

Similarly, the baseline data was compared with the GSRS score distribution



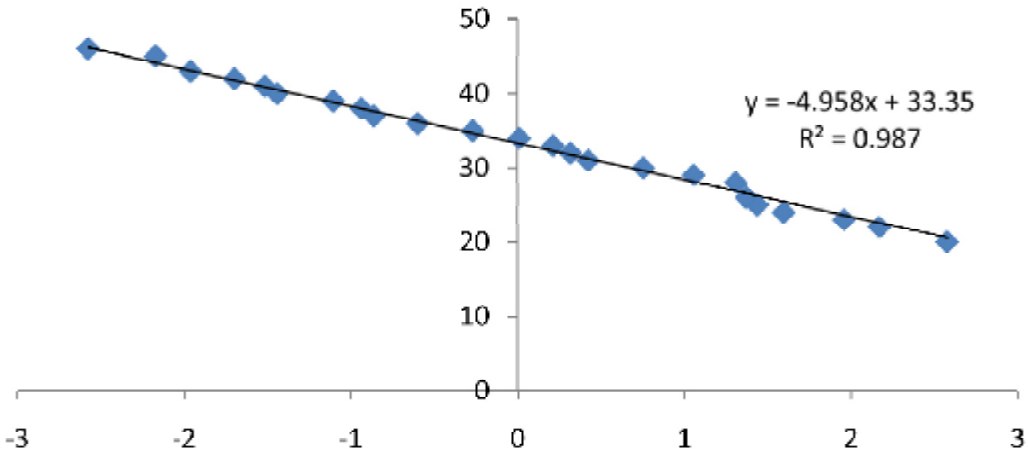
Graph 33: Q- Q plot for age in trial group



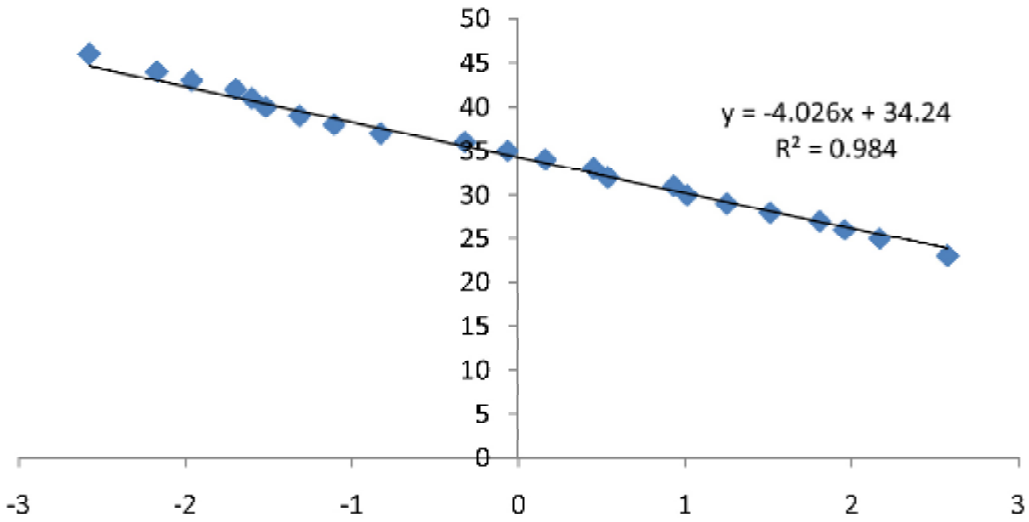
Graph 34: Q- Q plot for age in control group

in both the groups, to see the distribution. From the graphs on comparison, it was observed that the distribution of GSRS score in both the groups, follow normality, with very few outliers. So they are ideal and fit for comparison.

3. Levene's test for homogenity of variances



Graph 35: Q- Q plot for GSRS score in trial group



Graph 36: Q- Q plot for GSRS score in control group

Table 54 Levene's test – Age

Levene's Statistics	df1	df2	P Value
3.326	1	198	0.07

Testing for homogenity of variances for age using Levene's statistics, the test was found to be statistically insignificant (P>0.05). So the two groups are ideal for comparison.

Table 55 Levene's test – GSRS score

Levene's Statistics	df1	df2	P Value
3.13	1	198	0.08

Testing for homogenity of variances for GSRS using Levene's statistics, the test was found to be statistically insignificant (P>0.05). So the two groups are ideal for comparison.

4. Test for independence of variables

Table 56 Test for independence of variables – Trial group

Observed	Expected	χ^2	P value
30	36.6	0.9391	0.005847
29	22.4		
32	25.4		
9	15.6		

Table 57 Test for independence of variables – Control group

Observed	Expected	χ^2	P value
36	25.92	0.9943	0.00005
18	28.08		
12	22.08		
34	23.92		

The change in the GSRS scores in relation to the age of the subjects in the trial and control groups were computed and the strength of association was calculated, using the χ^2 test. The test was found to be statistically significant in both groups ($P< 0.01$ in Trial group and $P<0.001$ in Control group) showing the independence of variables and also the possibility for a comparison in between them.

Table 58 F test for independence of Variables – Both groups before treatment – Amlapitta rating scale

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	0.08	1	0.08			
Within Groups	1178.54	198	5.9522	0.01344	0.907823	3.888853
Total	**1178.62**	**199**				

ANOVA was performed to see whether the baseline score recorded using the Amlapitta rating scale in both the groups, were comparable or not. The test was not significant with the P value > 0.05, ie. the data is comparable at the baseline.

OBSERVATIONS ON THE EFFICACY OF THE THERAPY

1. Abdominal Pain

Table 59 Mean distribution of efficacy of therapy on Abdominal pain - both groups

	Trial	Control
BT	5.46 ± 1.08	5.74 ± 0.52
AT1	3.76 ± 0.87	4.20 ± 0.60
AT2	2.82 ± 0.83	3.29 ± 0.62
AF	1.95 ± 0.84	2.47 ± 0.64

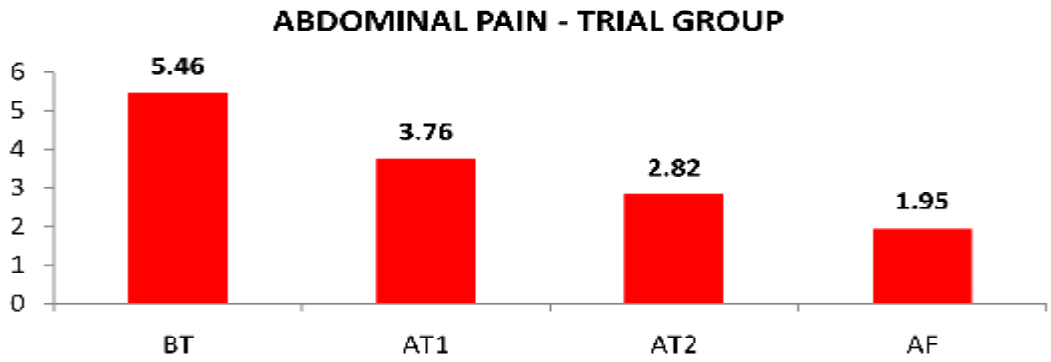
(BT – before treatment, AT1 – first assessment after 15 days, AT2 – second assessment after 30 days, AF – follow-up assessment after 45 days)

In the trial group, the symptom of abdominal pain of 100 patients showed a mean severity score of 5.46 ± 1.08 while in the control, it was 5.74 ± 0.52. After the treatment, during the first assessment (AT1), the mean score reduced to 3.76 ± 0.87 in the study group, while in the control, it reduced to 4.20 ± 0.60. In the second assessment (AT2), the mean score in the study group was 2.82 ± 0.83, while in control, it was 3.29 ± 0.62. During the follow-up (AF), the mean score in the study group was 1.95 ± 0.84, while in the control group, it was 2.47 ± 0.64.

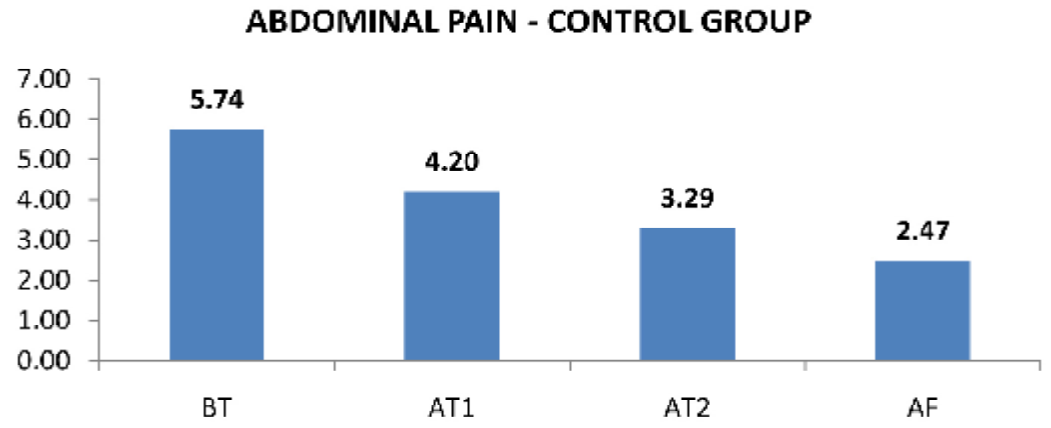
Table -60 F-test - trial group - Abdominal pain

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	677.408	3	225.803	268.853	3.96E-95	2.627441
Within Groups	332.59	396	0.8399			
Total	**1009.998**	**399**				

On comparing the mean differences after each assessment in trial group, using the Analysis of variance, the F value was found to be significant at 0.1% level ($P < 0.001$) indicating that there is difference in the mean scores during and after the treatment.



Graph 37: Mean distribution of efficacy of therapy on Abdominal pain – Trial group



Graph 38: Mean distribution of efficacy of therapy on Abdominal pain – Control group

Table -61 F-test - control group - Abdominal pain

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	588.3	3	196.1	540.26	1.5 E-139	2.6274
Within Groups	143.74	396	0.363			
Total	732.04	399				

On comparing the mean differences after each assessment in the control group, using the ANOVA, F value was found to be significant at 0.1% level (P< 0.001) indicating that there is difference in mean scores during and after the treatment.

Multiple Comparison Between Groups –Trial Group

Table -62 Tuckey Kramer test - trial group - Abdominal pain

Comparison		Mean difference	q	P Value
BT	AT1	1.7	33.1	<0.001
BT	AT2	2.64	51.402	<0.001
BT	AF	3.51	68.342	<0.001
AT1	AT2	0.94	18.302	<0.001
AT1	AF	1.81	35.242	<0.001
AT2	AF	0.87	16.939	<0.001

On comparing the various group mean scores of assessment among themselves using Tuckey Kramer test in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at P< 0.001 level. Similarly the comparisons AT1 Vs AT2 and AF also showed significance at 0.1% level. The comparison between AT2 and AF was also significant at P< 0.001 level.

Multiple comparison in between groups - Control group

Table -63 Tuckey Kramer test - control group - Abdominal pain

Comparison		Mean difference	q	P Value
BT	AT1	1.53	37.402	<0.001
BT	AT2	2.44	59.647	<0.001
BT	AF	3.27	79.937	<0.001
AT1	AT2	0.91	22.245	<0.001
AT1	AF	1.74	42.535	<0.001
AT2	AF	0.83	20.29	<0.001

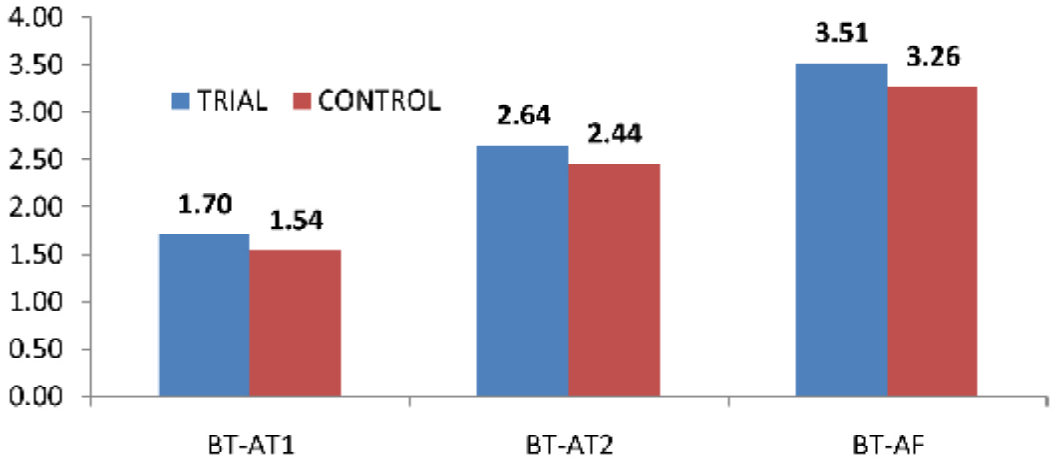
On comparing the various group mean scores of assessment among themselves, in the control group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. Similarly the comparisons AT1 Vs AT2 and AF also showed significance at 0.1% level. The comparison between AT2 and AF was also significant at 0.1% level.

Comparison between the two groups- unpaired t test

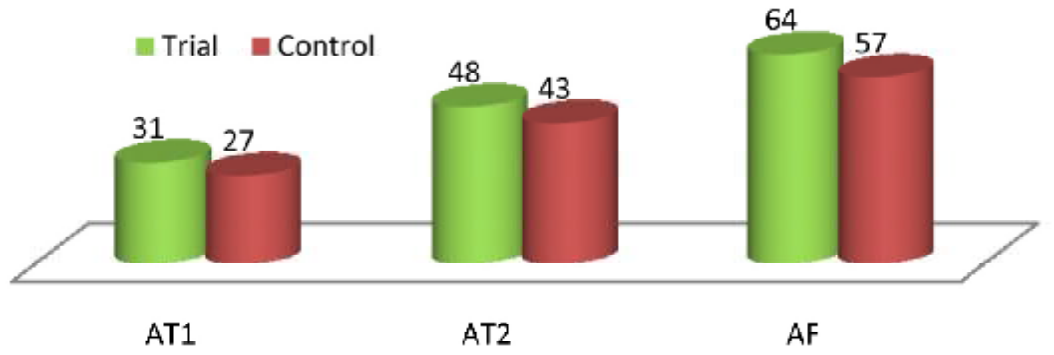
Table -64 Unpaired t test - between groups - Abdominal pain

Comparison	M D trial	M D control	t	P
BT - AT1	1.7 $\pm$ 0.56	1.53 $\pm$0.56	-4.173	< 0.001
BT - AT2	2.64 $\pm$ 0.85	2.44 $\pm$0.66	-2.799	< 0.01
BT - AF	3.51 $\pm$ 0.85	3.27$\pm$ 0.66	-2.01578	> 0.05

On comparing the efficacy of treatment between the trial and control groups with unpaired t test, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant (P< 0.001). The comparison of mean reduction before



Graph 39:
Mean distribution of the efficacy between groups- Abdominal pain



Graph 40: Distribution of the percentage of relief between groups- Abdominal pain

treatment (BT) and on second assessment (AT2) was significant at 0.1% level. On comparing the two groups between BT and on third assessment (AF), it was not significant (P> 0.05)

The percentage of relief for the symptom abdominal pain was 31% during the first assessment (AT1) in the trial and 27 % in the control . During the second assessment (AT2), the percentage of relief became 48% in the trial and 43% in the control. During the third assessment (AF), it was 64 % in trial and 57 % in control.

2. HEART BURN

Table 65 Mean distribution of efficacy on Heart burn - both groups

	Trial	Control
BT	4.98 ± 0.67	5.06 ± 0.75
AT1	3.54 ± 0.7	3.71 ± 0.76
AT2	2.41± 0.74	2.86 ± 0.82
AF	1.72 ± 0.87	2.58 ± 0.78

In the trial group, the symptom of heart burn among the 100 participants showed a mean severity score of 4.98 ± 0.67 while in the control, it was 5.06 ± 0.75. During the first assessment (AT1), the mean score was reduced to 3.54 ± 0.7 in the study group, while in the control, it was reduced to 3.71 ± 0.76. In the second assessment (AT2), the mean score in the study group was 2.41± 0.74, while in the control, it was 2.86 ± 0.82. During the follow-up (AF), the mean score in the study group was 1.72 ± 0.87, while in the control, it was 2.58 ± 0.78.

Table 66 F test - trial group- heart burn

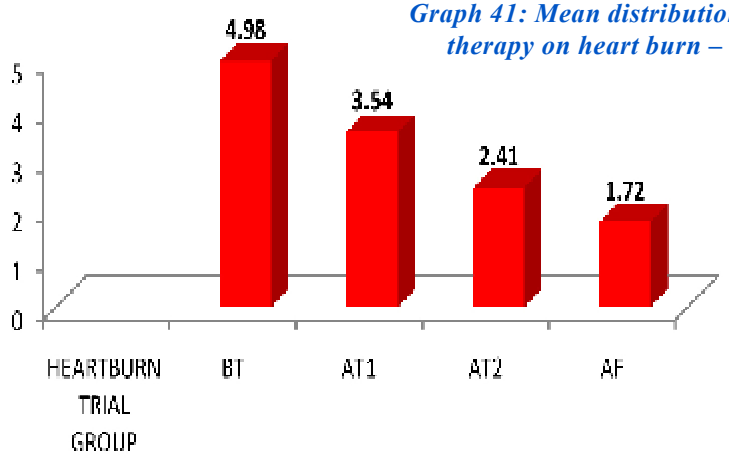
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	609.2875	3	203.096	363.672	2.3 E-113	2.627441
Within Groups	221.15	396	00.559			
Total	**830.438**	**399**				

On comparing the mean differences in the heart burn after each assessment in the trial group, using the ANOVA, the F value was found to be significant at 0.1% level ($P < 0.001$) indicating that there is a difference in the mean scores during and after the treatment.

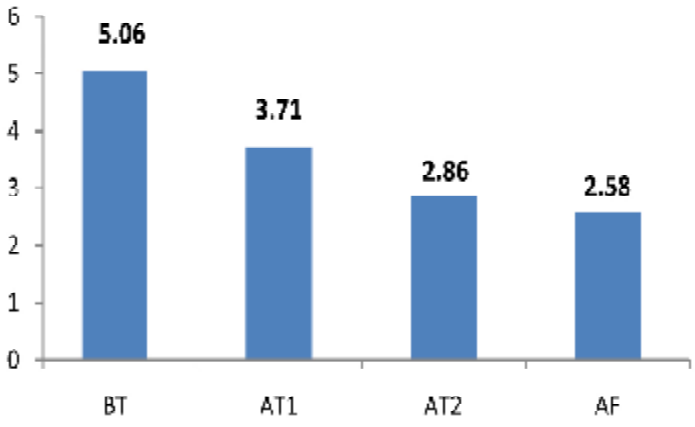
TABLE 67 F test - control group- heart burn

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	374.22	3	124.74	206.873	1.05 E-80	2.6275
Within Groups	238.78	396	0.603			
Total	**613**	**399**				

Graph 41: Mean distribution of efficacy of therapy on heart burn – Trial group



Graph 42: Mean distribution of efficacy of therapy on heart burn – Control group



On comparing the mean differences in heart burn after each assessment in the control group, using the ANOVA, the F value was found to be significant at 0.1% level ($P < 0.001$) indicating that, there is a difference in the mean scores during and after the treatment.

Multiple comparison in between groups – trial group

Table -68 Tuckey Kramer test - trial group – Heart burn

Comparison		Mean difference	q	P Value
BT	AT1	1.44	26.612	<0.001
BT	AT2	2.57	47.495	<0.001
BT	AF	3.26	60.246	<0.001
AT1	AT2	1.13	20.883	<0.001
AT1	AF	1.82	33.634	<0.001
AT2	AF	0.69	12.752	<0.001

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. Similarly, the comparisons AT1 Vs AT2 and AF also showed significance at P< 0.001 Level. The comparison between AT2 and AF was also significant at 0.1% level.

Comparison in between groups - control group

Table -69 Tuckey Kramer test - control group – Heart burn

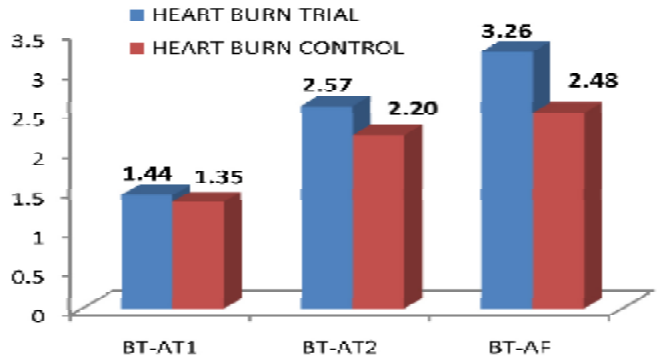
Comparison		Mean difference	q	P Value
BT	AT1	1.35	24.623	<0.001
BT	AT2	2.2	40.126	<0.001
BT	AF	2.49	45.415	<0.001
AT1	AT2	0.85	15.503	<0.001
AT1	AF	1.14	20.792	<0.001
AT2	AF	0.29	5.289	<0.01

On comparing the various group mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. Similarly the comparisons AT1 Vs AT2 and AF also showed significance at P< 0.001 level. The comparison between AT2 and AF was also significant, but at the 1% level.

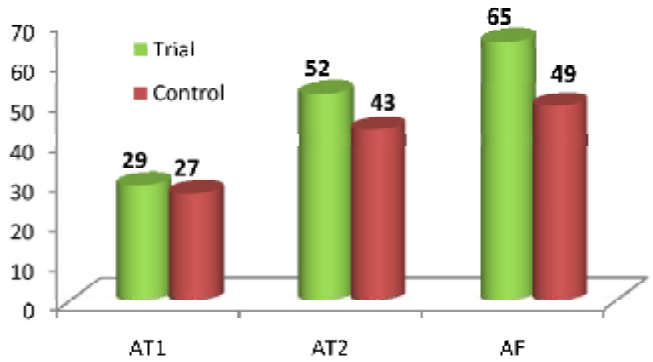
Comparison between the two groups- unpaired t test

Table -70 Unpaired t test - between groups – Heart burn

Comparison	M D Trial	M D Control	t	P
BT - AT1	1.44 ± 0.66	1.35 ± 0.64	-4.467	< 0.001
BT - AT2	2.57 ± 0.82	2.20 ± 0.79	-1.141	> 0.05
BT - AF	3.26 ± 0.96	2.49 ± 0.90	2.047	< 0.05



Graph 43: Mean distribution of the efficacy of therapy between groups- Heart burn



Graph 44: Percentage of relief between groups- Heart burn.

On comparing the efficacy of the treatment in heart burn between the trial and control groups using unpaired t test, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant ($P < 0.001$). The comparison between mean reduction before treatment (BT) and on second assessment (AT2) was not significant ($P > 0.05$). On comparing the two groups between BT and on third assessment (AF), it was significant at 5 % level ($P < 0.05$)

The percentage of relief for the symptom heart burn was 29% during the first assessment (AT1) in the trial group and 27 % in the control. During the second assessment (AT2), the percentage of relief became 52 % in the trial group and 43% in the control. During the third assessment (AF), it was 65 % in the trial and 49 % in the control.

4. ACID REGURGITATION

Table 71 Mean distribution of efficacy of therapy on Acid regurgitation - both groups

	Trial	Control
BT	4.23 ± 1.14	4 ± 1.50
AT1	3.04± 0.93	3.06 ± 1.24
AT2	2.25± 0.87	2.51 ± 1.08
AF	1.64 ± 0.82	2.22 ± 1.02

In the trial group, the symptom of acid regurgitation recorded a mean severity score of 4.23 ± 1.14 ,while in the control, it was 4 ± 1.50. After the treatment, during the first assessment (AT1), the mean score was reduced to 3.04 ± 0.93 in the study group, while in the control, it was reduced to 3.06 ± 1.24. In the second

assessment (AT2), the mean score in the study group was 2.25 $\pm$ 0.87, while in the control, it was 2.51 $\pm$ 1.08. During the follow-up (AF), the mean score in the study group was 1.64 $\pm$ 0.82, while in the control, it was 2.22 $\pm$ 1.02.

Table 72 F test -trial group – acid regurgitation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	375.02	3	125.0067	138.531	2.25 E-61	2.627441
Within Groups	357.34	396	0.9024			
Total	**732.36**	**399**				

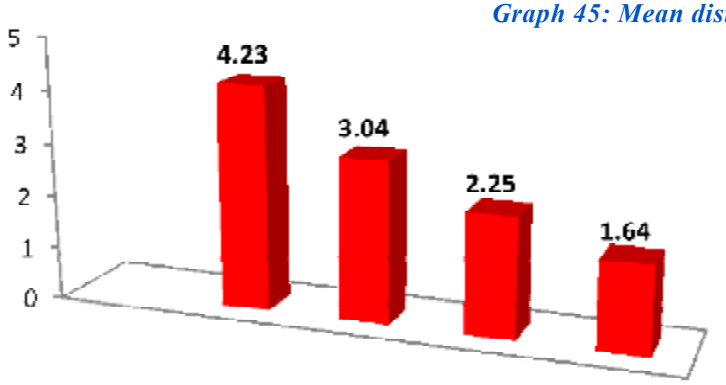
On comparing the mean differences in the symptom of acid regurgitation after each assessment in the trial group, using ANOVA, the F value was found to be significant at 0.1% level (P< 0.001) indicating that there is a difference in the mean scores during and after the treatment.

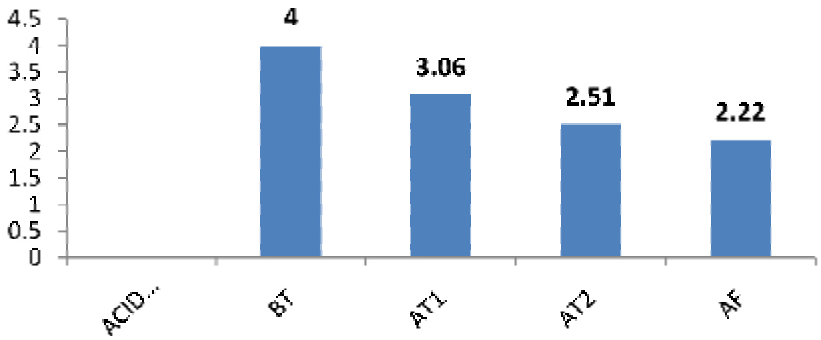
TABLE 73 F test – control group – acid regurgitation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	180.5675	3	60.1892	40.146	1.14 E-22	2.6274
Within Groups	593.71	396	1.4993			
Total	**774.278**	**399**				

On comparing the mean differences in acid regurgitation after each assessment in the control group, using the ANOVA, the F value was found to be significant at 0.1% level (P< 0.001) indicating that there is a difference in the mean scores during and after the treatment.

Graph 45: Mean distribution of efficacy on Acid regurgitation – Trial group





Graph 46: Mean distribution of efficacy on Acid regurgitation – Control group

Multiple Comparison in between groups - trial group

Table -74 Tuckey Kramer test - trial group – Acid regurgitation

Comparison		MD	q	P Value
BT	AT1	1.19	20.459	<0.001
BT	AT2	1.98	34.041	<0.001
BT	AF	2.59	44.529	<0.001
AT1	AT2	0.79	13.582	<0.001
AT1	AF	1.4	24.069	<0.001
AT2	AF	0.61	10.487	<0.001

On comparing the various group mean scores of assessment in acid regurgitation among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. Similarly the comparisons AT1 Vs AT2 and AF also showed significance at P< 0.001 Level. The comparison between AT2 and AF was also similarly significant.

Multiple Comparison in between groups – control group

Table -75 Tuckey Kramer test - control group – Acid regurgitation

Comparison		MD	q	P Value
BT	AT1	0.93	16.559	<0.001
BT	AT2	1.48	26.352	<0.001
BT	AF	1.76	31.337	<0.001
AT1	AT2	0.55	9.793	<0.001
AT1	AF	0.83	14.778	<0.001
AT2	AF	0.28	4.985	<0.01

On comparing the various groups mean scores of assessment in acid regurgitation among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. Similarly the comparisons AT1 Vs AT2 and AF also showed significance at P< 0.001 Level. The comparison between AT2 and AF was also significant, but at the 1% level.

Comparison between the two groups- unpaired t test

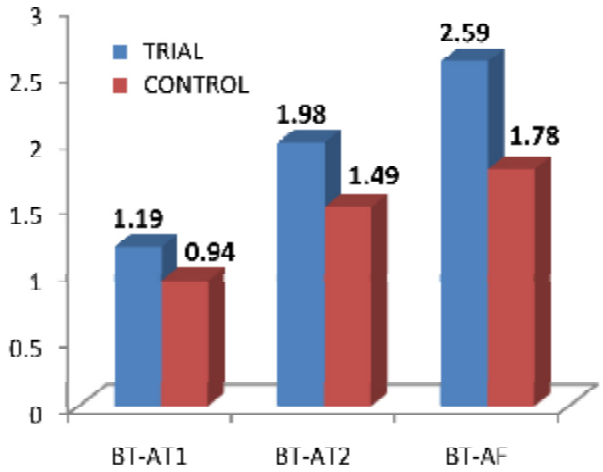
On comparing the efficacy of treatment in the symptom of acid regurgitation between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant at 0.1% level. The comparison of

mean reduction before treatment (BT) and on second assessment (AT2) was not significant (P> 0.05). On comparing the two groups between BT and follow up (AF), it was significant, but at the 5 % level (P< 0.05)

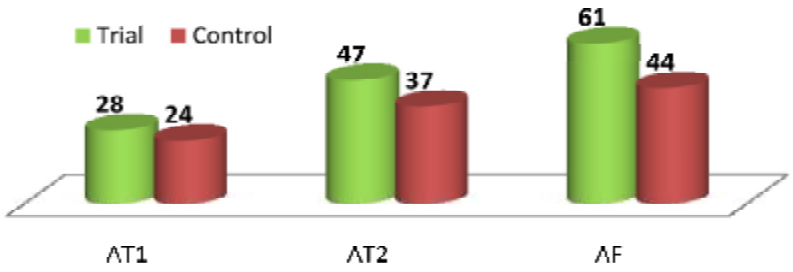
Table -76 Unpaired t test - between groups – Acid regurgitation

Comparison	M D Trial	M D Control	t	P
BT - AT1	1.19 ± 0.66	0.93 ± 0.64	-2.60	< 0.001
BT-AT2	1.98 ± 0.97	1.48 ± 0.94	0	> 0.05
BT - AF	2.59 ± 1.07	1.76 ± 1.02	2.23	< 0.05

The percentage of relief for acid regurgitation was observed as 28 % during the first assessment (AT1) in the trial group and 24 % in the control. During the second assessment (AT2), the percentage of relief became 47 % in trial and 37 % in the control group. During the follow up (AF), it was 61 % in trial and 44 % in the control group.



Graph 47: Mean distribution of the efficacy between groups- Acid regurgitation



Graph 48:
Distribution of the percentage of relief between groups- Acid regurgitation

5. SUCKING SENSATION

Table 77 Mean distribution of efficacy of therapy on sucking sensation - both groups

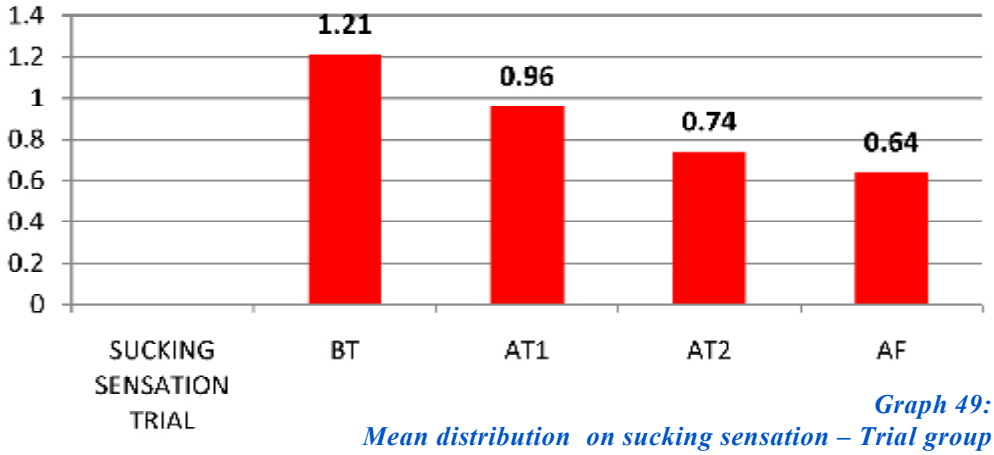
	Trial	Control
BT	1.21 ± 1.70	1.57 ± 1.83
AT1	0.96 ± 1.37	1.37 ± 1.65
AT2	0.74 ± 1.13	1.18 ± 1.43
AF	0.64 ± 0.97	1.1 ± 1.33

In the trial group, the symptom of sucking sensation showed a mean severity score of 1.21 ± 1.70 while in the control, it was 1.57 ± 1.83. After the treatment, during the first assessment (AT1), the mean score was reduced to 0.96 ± 1.37 in the study group, while in the control, to 1.37 ± 1.65. In the second assessment (AT2), the mean score in the study group was 0.74 ± 1.13, while in the control, it was 1.18 ± 1.43. During the follow-up (AF), the mean score in the study group was 0.64 ± 0.97, while in the control, it was 1.1 ± 1.33.

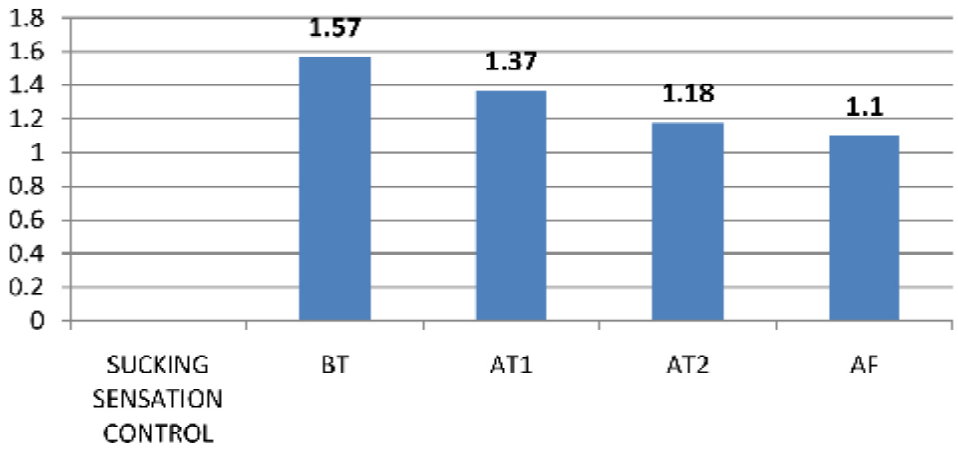
Table 78 F test - trial group – sucking sensation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	19.2275	3	6.40927	3.6746	0.0125	2.6274
Within Groups	690.71	396	1.7442			
Total	**709.938**	**399**				

On comparing the mean differences in the symptom of sucking sensation after each assessment in the trial group, using ANOVA, the F value was found to be significant at 5% level ($P < 0.05$) indicating that there is a difference in the mean scores during and after the treatment.



Graph 49: Mean distribution on sucking sensation – Trial group



Graph 50: Mean distribution on sucking sensation – Control group

Table 79 F test - control group – sucking sensation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	13.21	3	4.4033	1.7801	0.1504	2.6274
Within Groups	979.58	396	2.4737			
Total	**992.79**	**399**				

On comparing the mean differences in sucking sensation after each assessment in the control group, using the ANOVA, the F value was found to be not significant (P> 0.05) indicating that there is no difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

Table -80 Tuckey Kramer test - trial group – Sucking sensation

Comparison		MD	q value	P value
BT	AT1	0.25	5.756	< 0.001
BT	AT2	0.47	10.822	< 0.001
BT	AF	0.57	13.124	< 0.001
AT1	AT2	0.22	5.065	< 0.01
AT1	AF	0.32	7.368	< 0.001
AT2	AF	0.1	2.302	> 0.05

On comparing the various group mean scores of assessment in sucking sensation among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. The comparisons AT1 Vs AT2 was significant at 1% level (P<0.01). AT1 Vs AF showed significance at P< 0.001 level. The comparison between AT2 and AF was not significant.

Multiple Comparison in between groups – control group

On comparing the various group mean scores of assessment among themselves in the control, the comparison between BT Vs AT1 was significant at 1% level, BT Vs AT2 and AF showed high significance (P< 0.001). Similarly the comparisons AT1 Vs AT2 was significant at 1% level and AT1 Vs AF showed significance at 0.1% level. The comparison between AT2 and AF was not significant.

Table -81 Tuckey Kramer test - control group – sucking sensation

Comparison		MD	q value	P value
BT	AT1	0.2	4.96	<0.01
BT	AT2	0.39	9.672	<0.001
BT	AF	0.47	11.656	<0.001
AT1	AT2	0.19	4.712	<0.01
AT1	AF	0.27	6.696	<0.001
AT2	AF	0.08	1.984	>0.05

Comparison between the two groups- unpaired t test

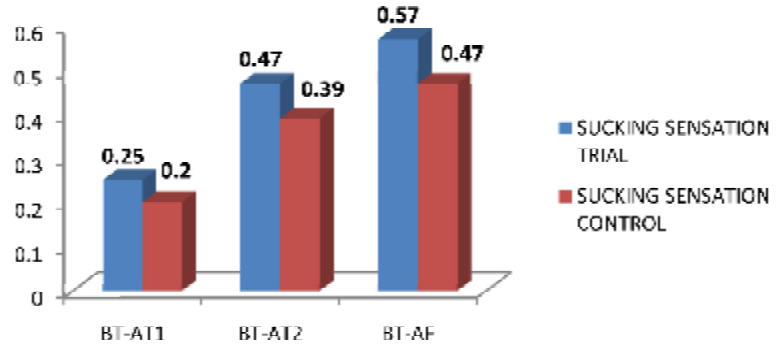
Table -82 Unpaired t test - between groups – Sucking sensation

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.25 $\pm$ 0.46	0.20 $\pm$ 0.45	-7.014	< 0.001
BT-AT2	0.47 $\pm$ 0.77	0.39 $\pm$ 0.67	-4.123	< 0.001
BT - AF	0.57 $\pm$ 0.87	0.47 $\pm$ 0.70	-3.582	< 0.001

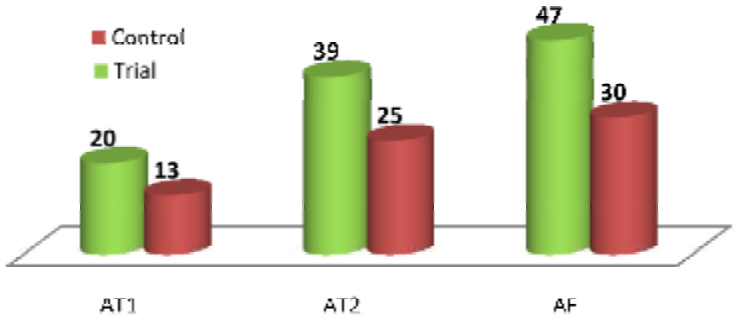
On comparing the efficacy of treatment in the symptom of sucking sensation between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant at 0.1% level. The comparison between

the mean reduction before treatment (BT) and on second assessment (AT2) was also highly significant (P<0.001). On comparing the two groups between BT and third assessment (AF), it was also equally significant.

The percentage of relief for the symptom sucking sensation was observed as 20% during the first assessment (AT1) in the trial group and 13% in the control. During the second assessment (AT2), the percentage of relief was 39% in the trial and 25% in the control. During the third assessment (AF), it was 47 % in the trial group and 30 % in control.



Graph 51: Mean distribution of the efficacy of therapy between groups- sucking sensation



Graph 52: Distribution of the percentage of relief between groups- sucking sensation

5. NAUSEA / VOMITING

In the trial group, the symptom of nausea/ vomiting of 100 subjects, showed a mean severity score of 3.31 $\pm$ 1.91 while in the control, it was 3.11 $\pm$ 2.07. With the medication, during the first assessment (AT1), the mean score was reduced to 2.33 $\pm$ 1.41 in the study group, while in the control, it was reduced to 2.33 $\pm$ 1.61. In the second assessment (AT2), the mean score in the study group was 1.75 $\pm$ 1.12, while in the control, it was 1.95 $\pm$ 1.37. During the follow-up (AF), the mean score in the study group was 1.32 $\pm$ 0.97, while in the control, it was 1.73 $\pm$ 1.24.

Table 83 Mean distribution of efficacy of therapy on Nausea/ vomiting - both groups

	Trial	Control
BT	3.31 $\pm$ 1.91	3.11 $\pm$ 2.07
AT1	2.33 $\pm$ 1.41	2.33 $\pm$ 1.61
AT2	1.75 $\pm$ 1.12	1.95 $\pm$ 1.37
AF	1.32 $\pm$ 0.97	1.73 $\pm$ 1.24

Table 84 F test - trial group – nausea/ vomiting

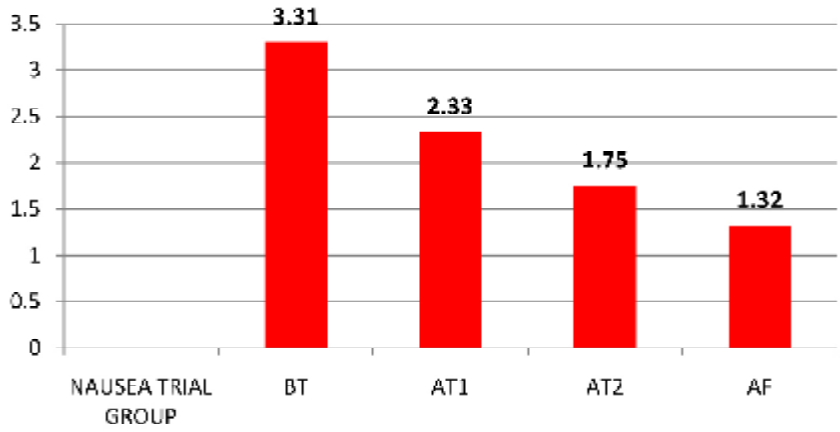
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	222.388	3	74.1292	37.8283	1.63 E-21	2.6274
Within Groups	776.01	396	1.9596			
Total	**998.398**	**399**				

On comparing the mean differences in nausea/vomiting after each assessment in the trial group, using the ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that there is difference in the mean scores during and after the treatment.

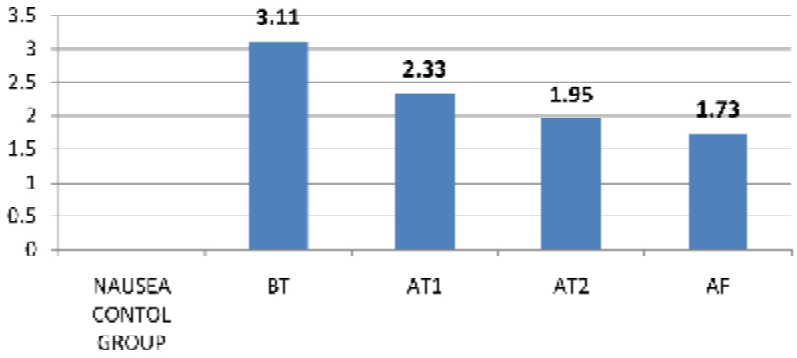
Table 85 F test – control group – nausea/ vomiting

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	110.28	3	36.76	14.323	7.06 E-09	2.6274
Within Groups	1016.36	396	2.567			
Total	**1126.64**	**399**				

On comparing the mean differences in nausea/ vomiting after each assessment in the control group, using the ANOVA, the F value was found to be highly significant (P<0.001) indicating that there is difference in the mean scores during and after the treatment.



Graph 53: Mean distribution on nausea/ vomiting – Trial group



Graph 54: Mean distribution on nausea/ vomiting – Control group

Multiple Comparison in between groups – trial group

Table -86 Tuckey Kramer test - trial group – Nausea/ vomiting

Comparison		MD	q value	P value
BT	AT1	0.98	14.936	<0.001
BT	AT2	1.56	23.776	<0.001
BT	AF	1.99	30.329	<0.001
AT1	AT2	0.58	8.84	<0.001
AT1	AF	1.01	15.393	<0.001
AT2	AF	0.43	6.554	<0.001

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. The comparisons AT1 Vs AT2 was significant at 1% level ($P<0.01$). AT1 Vs AF showed significance at $P< 0.001$ level. The comparison between AT2 and AF was also significant at the same level ($P< 0.001$)

Multiple Comparison in between groups – control group

On comparing the various group mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, BT Vs AT2 and AF, showed significance at 0.1% level. Similarly the comparison AT1 Vs AT2 and AF was significant at 0.1 % level ($P< 0.001$). The comparison between AT2 and AF was significant only at 5% level ($P< 0.05$).

Table -87 Tuckey Kramer test - Control group – Nausea/ vomiting

Comparison		MD	q value	P value
BT	AT1	0.78	13.812	<0.001
BT	AT2	1.16	20.541	<0.001
BT	AF	1.38	24.436	<0.001
AT1	AT2	0.38	6.729	<0.001
AT1	AF	0.6	10.625	<0.001
AT2	AF	0.22	3.896	<0.05

Comparison between the two groups- unpaired t test

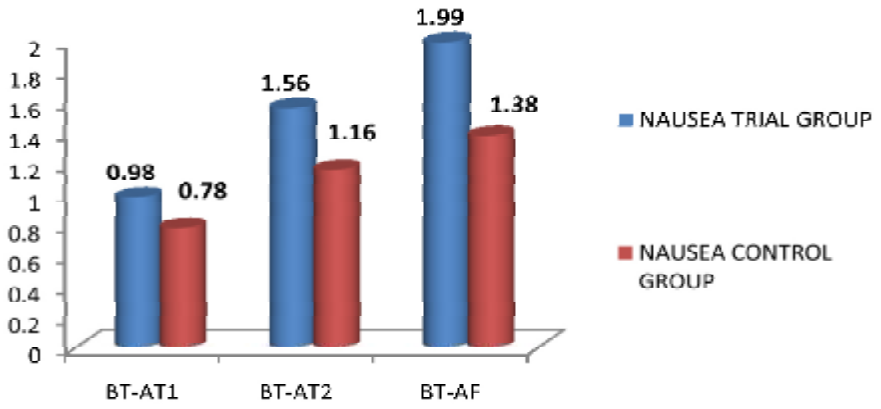
Table -88 Unpaired t test - between groups – Nausea/ Vomiting

Comparison	MD Trial	MD Control	t	P
BT - AT1	0.98 $\pm$ 0.78	0.78 $\pm$ 0.73	- 2.80	< 0.01
BT-AT2	1.56 $\pm$ 1.09	1.16 $\pm$ 0.97	- 0.68	> 0.05
BT - AF	1.99 $\pm$ 1.37	1.38 $\pm$ 1.12	0.62	> 0.05

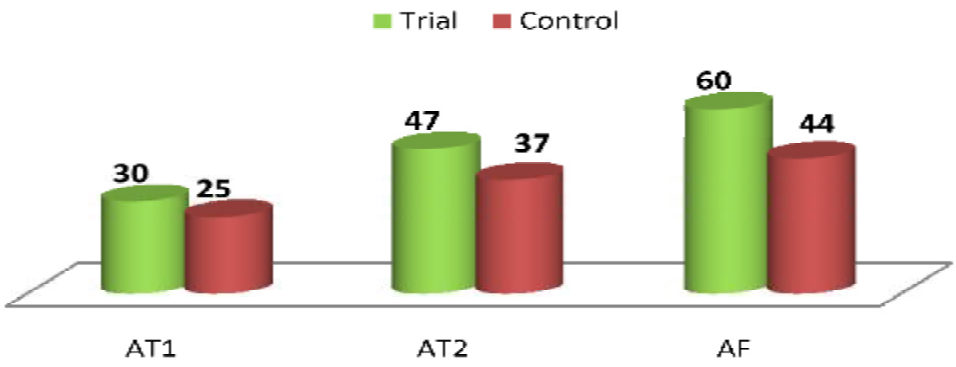
Unpaired t test was used for comparing the efficacy of intervention in the symptom of nausea/ vomiting between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 1% level. (P< 0.01). The comparison between, mean score before treatment (BT) and second assessment (AT2) was not significant (P> 0.05). On comparing the two groups between BT and follow up (AF), it was also not significant (P> 0.05)

The percentage of relief for the symptom of nausea/ vomiting was noted as 30% during the first assessment (AT1) in the trial and 25% in the control. During the second assessment (AT2), the percentage of relief was 47% in the trial group and

37% in the control. During the third assessment (AF), it was 60% in the trial group and 44% in the control.



Graph 55: Mean distribution of the efficacy of therapy between groups- Nausea/ Vomiting



Graph 56: Distribution of the percentage of relief between groups- Nausea/ Vomiting

6. BORBORYGYMI

In the trial group, the symptom of borborygymi showed a mean severity score of 1.61 ± 1.62 in the trial, while in the control group, it was 1.47 ± 1.67. After the medication, during the first assessment (AT1), the mean score was reduced to 1.2 ± 1.25 in the study group, while in the control, it was reduced to 1.17 ± 1.36. In the second assessment (AT2), the mean score in the study group was 0.92 ± 1.02, while in the control, it was 1.01 ± 1.17. During the follow-up (AF), the mean score in the study group was 0.76 ± 0.87, while in the control, it was 0.76 ± 0.87.

Table 89 Mean distribution of efficacy of therapy on Borborygymi - both groups

	Trial	Control
BT	1.61 $\pm$ 1.62	1.47 $\pm$ 1.67
AT1	1.2 $\pm$ 1.25	1.17 $\pm$ 1.36
AT2	0.92 $\pm$ 1.02	1.01 $\pm$ 1.17
AF	0.76 $\pm$ 0.87	0.76 $\pm$ 0.87

Table 90 F test - trial group – Borborygymi

Source of Variation	SS	df	MS	F	P value	F crit
Between Groups	41.6075	3	13.8697	9.2869	6.01 E-06	2.6274
Within Groups	591.39	396	1.49341			
Total	**632.998**	**399**				

On comparing the mean differences in the score of borborygymi, after each assessment in the trial group, using the ANOVA, the F value was found to be significant at 0.1 % level ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

Table 91 F test - control group – Borborygymi

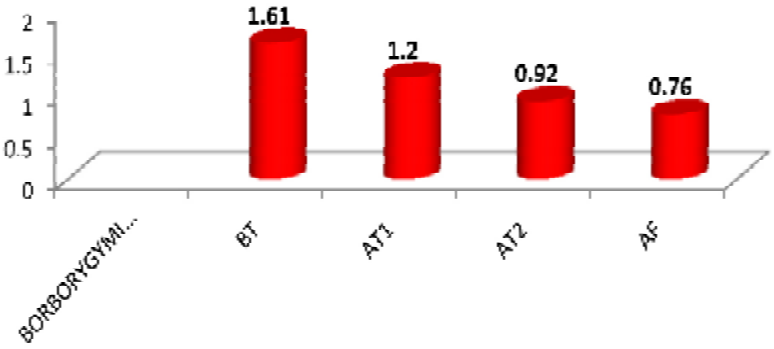
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	17.7275	3	5.909167	3.273822	0.021162	2.627441
Within Groups	714.77	396	1.804975			
Total	**732.4975**	**399**				

On comparing the mean differences in borborygymi, after each assessment

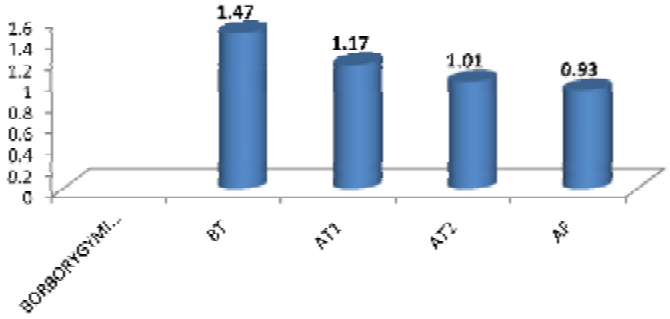
in the control group, using ANOVA, F value was found to be significant (P< 0.05) indicating that, there is difference in the mean scores, during and after the treatment.

Multiple Comparison in between groups – trial group

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. The comparisons AT1 VS AT2 and AF was also significant at 0.1% level. The comparison between AT2 and AF was not significant.



Graph 57:
Mean distribution of efficacy of therapy on Borborygymi – Trial group



Graph 58:
Mean distribution of efficacy of therapy on Borborygymi – Control group

Table -92 Tuckey Kramer test - trial group – borborygymi

Comparison		MD	q	P Value
BT	AT1	0.41	9.036	<0.001
BT	AT2	0.69	15.207	<0.001
BT	AF	0.85	18.734	<0.001
AT1	AT2	0.28	6.171	<0.001
AT1	AF	0.44	9.697	<0.001
AT2	AF	0.16	3.526	>0.05

Multiple Comparison in between groups – control group

Table -93 Tuckey Kramer test - Control group – borborygymi

Comparison	M D Trial	M D Control	t	P
BT	AT1	0.3	7.756	<0.001
BT	AT2	0.46	11.893	<0.001
BT	AF	0.54	13.962	<0.001
AT1	AT2	0.16	4.137	<0.05
AT1	AF	0.24	6.205	<0.001
AT2	AF	0.08	2.068	>0.05

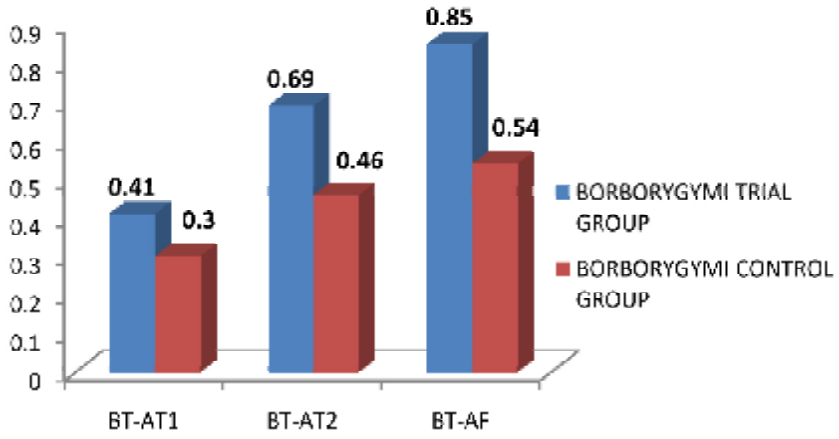
On comparing the various group mean scores of assessment among themselves in the control group in borborygymi, the comparison between BT Vs AT1, BT Vs AT2 and AF, was significant at 0.1% level (P< 0.001). The comparison AT1 Vs AT2 was significant at 5% level. (P< 0.05). The comparison AT1 Vs AF was significant at 0.1 % level (P< 0.001). The comparison between AT2 and AF was not significant.

Comparison between the two groups - unpaired t test

Table -94 Unpaired t test - between groups – Borborygymi

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.41 ± 0.49	0.30 ± 0.48	-5.65	< 0.001
BT-AT2	0.69 ± 0.81	0.46 ± 0.59	-2.75	< 0.01
BT - AF	0.85 ± 0.93	0.54 ± 0.77	-1.65	> 0.05

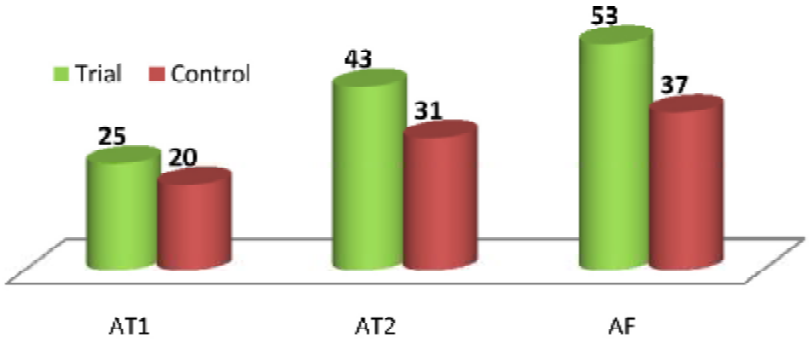
On comparing the efficacy of intervention in the score of borborygymi, between trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (P< 0.001). The comparison between mean reduction before treatment (BT) and on second assessment (AT2) was significant at 1% level (P< 0.01). On comparing two groups between BT and on third assessment (AF), it was not statistically significant (P>0.05).



Graph 59: Mean distribution of the efficacy of therapy between groups- Borborygymi

The percentage of relief in the symptom of borborygymi was observed as 25% during the first assessment (AT1) in the trial group and 20% in the control. During the second assessment (AT2), the percentage of relief was 43 % in the trial and 31%

in control group. During third assessment (AF), it was 53% in the trial and 37% in the control group.



Graph 60 : Distribution of the percentage of relief between groups- Borborygymi

7. ABDOMINAL DISTENSION

Table 95 Mean distribution of efficacy of therapy on Abdominal distension - both groups

	Trial	Control
BT	5.47 ± 0.90	5.52 ± 0.81
AT1	3.78 ± 0.85	4.01 ± 0.69
AT2	2.68 ± 0.86	3.11 ± 0.76
AF	2.01 ± 0.82	2.57 ± 0.71

In the trial group, the symptom of abdominal distension of 100 subjects showed a mean severity score of 5.47 ± 0.90 while in control, it was 5.52 ± 0.81.With the medication, during the first assessment (AT1), the mean score was reduced to 3.78 ± 0.85 in study group, while in the control, it was reduced to 4.01 ± 0.69. During second assessment (AT2), the mean score in study group was 2.68 ± 0.86, while in control, it was 3.11 ± 0.76. During the follow-up (AF), the mean score in the study group was 2.01 ± 0.82, while in control, it was 2.57 ± 0.71.

Table 96 F test - Trial group – Abdominal distension

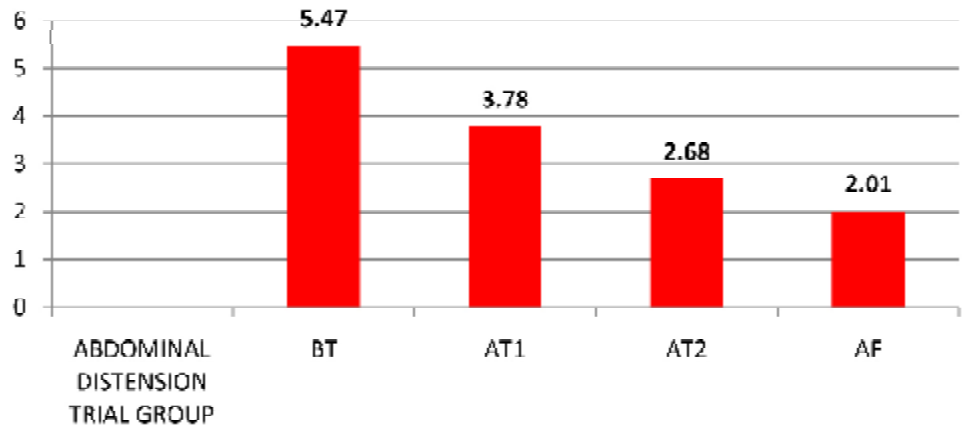
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	685.09	3	228.3633	308.831	2.7E-103	2.627441
Within Groups	292.82	396	0.73944			
Total	**977.91**	**399**				

On comparing the mean differences in the score of abdominal distension, after each assessment in the trial group, using the ANOVA, the F value was found to be significant at 0.1 % level ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

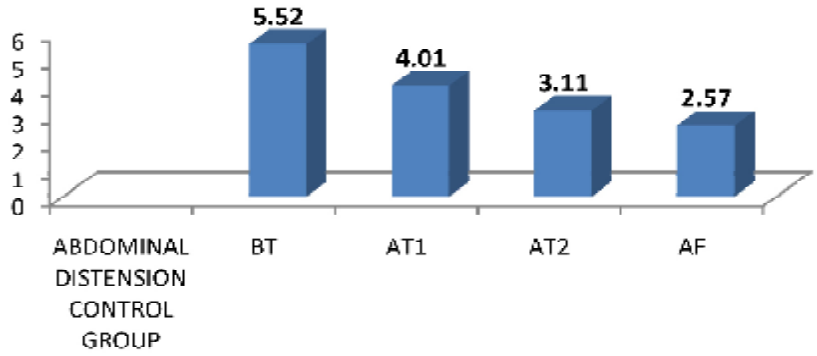
Table 97 F test - control group – Abdominal distension

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	499.1475	3	166.3825	299.1486	2.2E-101	2.627441
Within Groups	220.25	396	0.556187			
Total	719.3975	399				

On comparing the mean differences in abdominal distension, after each assessment in the control group, using the ANOVA, the F value was found to be significant at 0.1% level. ($P<0.001$) indicating that there is difference in the mean scores during and after the intervention.



Graph 61: Mean distribution on Abdominal distension – Trial group



Graph 62: Mean distribution on Abdominal distension – Control group

Multiple Comparison in between groups – trial group

Table -98 Tuckey Kramer test - trial group – Abdominal distension

Comparison		MD	q	P Value
BT	AT1	1.69	29.065	<0.001
BT	AT2	2.79	47.984	<0.001
BT	AF	3.46	59.507	<0.001
AT1	AT2	1.1	18.918	< 0.001
AT1	AF	1.77	30.441	<0.001
AT2	AF	0.67	11.523	<0.001

On comparing the various group mean scores of assessment among themselves in the trial group on abdominal distension, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level. The comparisons AT1 Vs AT2 and AF, was also significant at 0.1% level. The comparison between AT2 and AF was also significant. (P<0.001)

Multiple Comparison in between groups – control group

Table -99 Tuckey Kramer test - Control group – Abdominal distension

Comparison		MD	q	P Value
BT	AT1	1.51	24.469	<0.001
BT	AT2	2.41	39.053	<0.001
BT	AF	2.95	47.804	<0.001
AT1	AT2	0.9	14.584	<0.001
AT1	AF	1.44	23.335	<0.001
AT2	AF	0.54	8.751	<0.001

On comparing the various group mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF, was significant at 0.1% level (P< 0.001). The comparison AT1 VS AT2 and AF was also significant at the same level. (P< 0.001). The comparison between AT2 and AF was also similar in the significance.

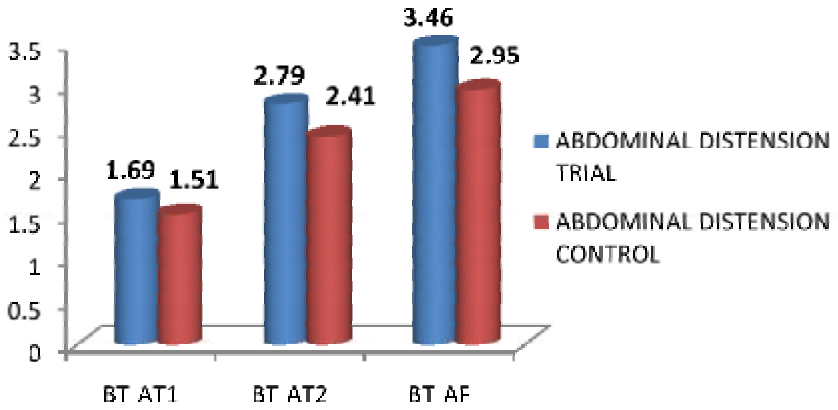
Comparison between groups- unpaired t test – Abdominal distension

On comparing the efficacy of treatment in the symptom of abdominal distension, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (P< 0.001). The

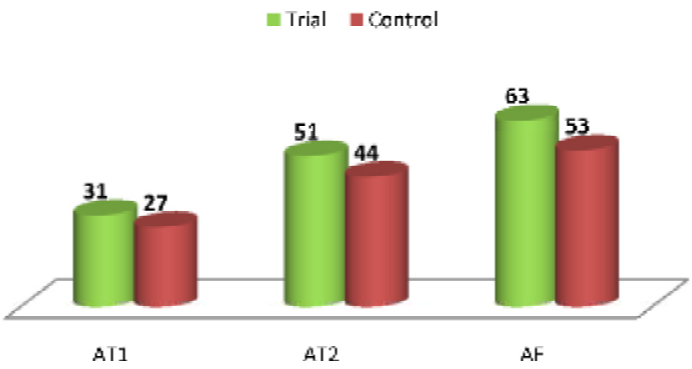
comparison of mean reduction before treatment (BT) and on second assessment (AT2) was not significant (P> 0.05). On comparing the two groups between BT and on third assessment (AF), it was also not significant (P> 0.05)

Table -100 Unpaired t test - between groups –Abdominal distension

Comparison	M D Trial	M D Control	t	P
BT - AT1	1.69 ± 0.79	1.51 ± 0.93	-2.631	< 0.01
BT-AT2	2.79 ± 0.94	2.41 ± 1.09	-0.834	> 0.05
BT - AF	3.46 ± 1.07	2.95 ± 1.05	0.067	> 0.05



Graph 63: Mean distribution of efficacy between groups - Abdominal distension



Graph 64: Percentage of relief between groups- Abdominal distension

The percentage of relief for the symptom of abdominal distension was 31% during the first assessment (AT1) in the trial group and 27% in the control. During the second assessment (AT2), the percentage of relief was 51% in the trial group and 44% in the control group. During the third assessment (AF), it was 63% in trial group and 53% in the control.

8. ERUCTATION

Table 101 Mean distribution of the efficacy of therapy on Eructation - both groups

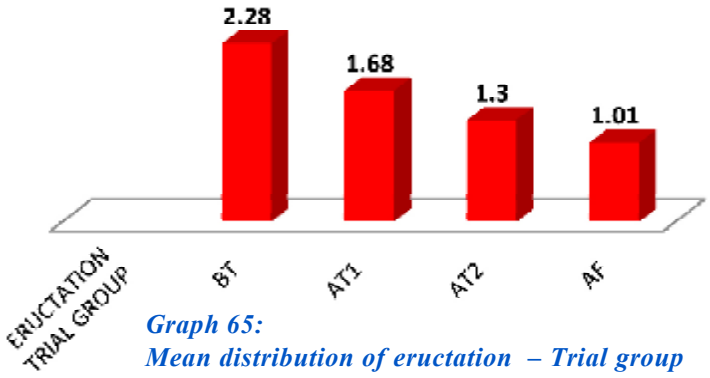
	Trial	Control
BT	2.28 $\pm$ 1.44	2.26 $\pm$ 1.46
AT1	1.68 $\pm$ 1.13	1.78 $\pm$ 1.25
AT2	1.3 $\pm$ 0.93	1.49 $\pm$ 1.06
AF	1.01 $\pm$ 0.77	0.93 $\pm$ 1.4

In the trial group, the symptom of eructation of 100 patients showed a mean severity score of 2.28 $\pm$ 1.44 while in the control group, it was 2.26 $\pm$ 1.46. After the intervention, during the first assessment (AT1), the mean score was reduced to 1.68 $\pm$ 1.13 in the study group, while in the control group it was reduced to 1.78 $\pm$ 1.25. During the second assessment (AT2), the mean score in the study group was 1.3 $\pm$ 0.93, while in the control group, it was 1.49 $\pm$ 1.06. During the follow-up (AF), the mean score in study group was 1.01 $\pm$ 0.77, while in control, it was 0.93 $\pm$ 1.4.

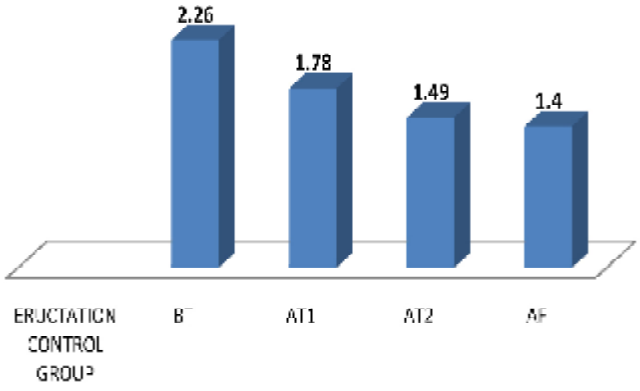
Table 102 F test - trial group – eructation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	90.2675	3	30.0897	25.0369	7.48E-15	2.627441
Within Groups	475.91	396	1.2018			
Total	566.1775	399				

On comparing the mean differences in the scores of eructation, after each assessment in the trial group, using the Analysis of variance, the F value was found to be significant at 0.1 % level ($P < 0.001$) indicating that, there is significant difference in the mean scores during and after the treatment.



Graph 65:
Mean distribution of eructation – Trial group



Graph 66:
Mean distribution of eructation – Control group

Table 103 F test - control group – eructation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	44.9875	3	14.99583	10.14426	1.88E-06	2.627441
Within Groups	585.39	396	1.478258			
Total	**630.3775**	**399**				

On comparing the mean differences in eructation, after each assessment in the control group, using the ANOVA, the F value was found to be significant at 0.1% level. (P<0.001) indicating that, there is difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

Table -104 Tuckey Kramer test - trial group – Eructation

Comparison		MD	q	P Value
BT	AT1	0.6	12.73	<0.001
BT	AT2	0.98	20.792	<0.001
BT	AF	1.27	26.944	<0.001
AT1	AT2	0.38	8.062	<0.001
AT1	AF	0.67	14.215	<0.001
AT2	AF	0.29	6.153	<0.001

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at P< 0.001 level. The comparisons AT1 Vs AT2 and AF was also significant at 0.1% level (P<0.001). The comparison between AT2 and AF was also similarly significant. (P<0.001)

Multiple Comparison in between groups – control group

On comparing the various group mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF, were significant at 0.1% level. The comparison AT1 Vs AT2 and AF was also significant at the same level. (P< 0.001). The comparison between AT2 and AF was not significant.

Table -105 Tuckey Kramer test - Control group – Eructation

Comparison		MD	q	P Value
BT	AT1	0.48	11.128	<0.001
BT	AT2	0.77	17.851	<0.001
BT	AF	0.86	19.937	<0.001
AT1	AT2	0.29	6.723	<0.001
AT1	AF	0.38	8.809	<0.001
AT2	AF	0.09	2.086	P>0.05

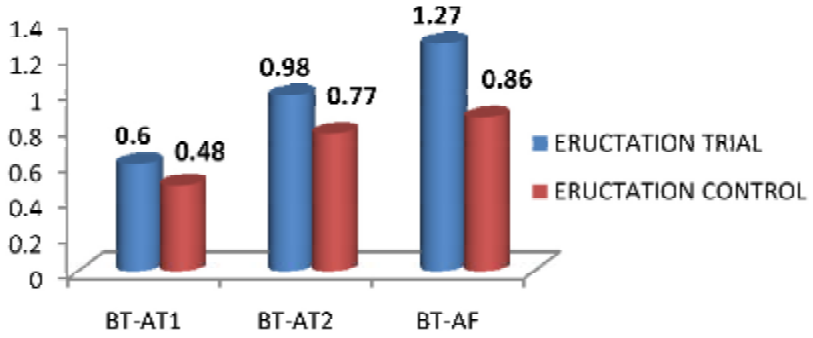
Comparison between the groups- unpaired t test – Eructation

Table -106 Unpaired t test - between groups –Eructation

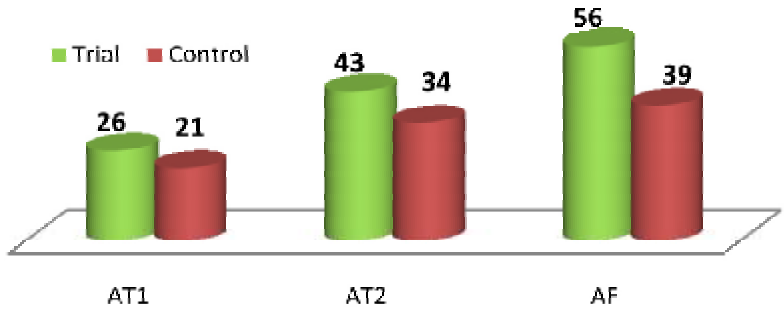
Comparison	M D Trial	M D Control	t	P
BT - AT1	0.6 $\pm$ 0.51	0.48 $\pm$ 0.54	-5.100	< 0.001
BT-AT2	0.98 $\pm$ 0.78	0.77 $\pm$ 0.71	-2.755	< 0.01
BT - AF	1.27 $\pm$ 0.93	0.86 $\pm$ 0.79	-0.736	> 0.05

On comparing the efficacy of treatment in the symptom of eructation, between the groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (< 0.001). The comparison of mean reduction before treatment (BT) and on second assessment (AT2) was significant at 1% level

(P< 0.01) . On comparing the two groups between BT and on third assessment (AF), it was not significant. (P> 0.05)



Graph 67: Mean distribution of the efficacy of therapy between groups- Eructation



Graph 68: Distribution of the percentage of relief between groups - Eructation

The percentage of relief for the symptom of eructation was recorded as 26% during the first assessment (AT1) in the trial and 21% in the control group. During the second assessment (AT2), the percentage of relief was 43% in the trial and 34% in the control. During the third assessment (AF), it was 56% in trial group and 39 % in the control.

9. INCREASED FLATUS

Table 107 Mean distribution of efficacy on increased flatus - both groups

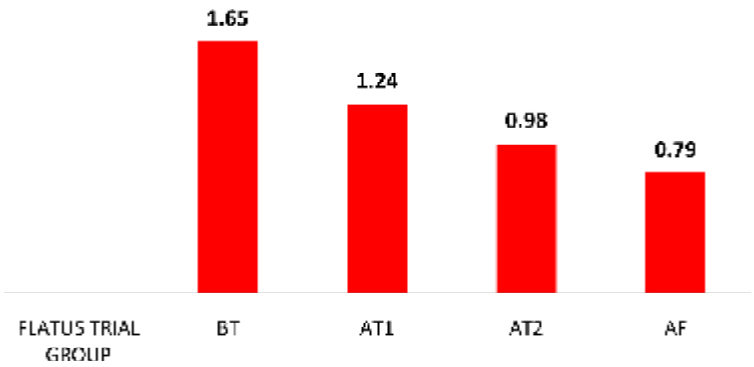
	Trial	Control
BT	1.65 ± 1.59	2.05 ± 1.59
AT1	1.24 ± 1.24	1.51 ± 1.22
AT2	0.98 ± 1.04	1.24 ± 1.05
AF	0.79 ± 0.88	1.05 ± 0.97

In the trial group, increased flatus of 100 subjects showed a mean severity score of 1.65 ± 1.59 while in the control, it was 2.05 ± 1.59. After intervention, during first assessment (AT1), the mean score reduced to 1.24 ± 1.24 in the study group, while in control, to 1.51 ± 1.22. In the second assessment (AT2), the mean score in study group was 0.98 ± 1.04, while in the control, it was 1.24 ± 1.05. During follow-up (AF), the mean score in study group was 0.79 ± 0.88, while in the control, it was 1.05 ± 0.97.

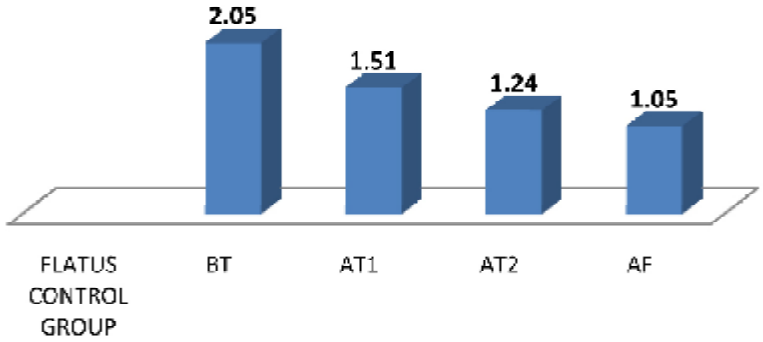
Table 108 F test - trial group – increased flatus

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	38.7275	3	12.9092	8.90055	1.01 E-05	2.627441
Within Groups	574.35	396	1.45038			
Total	**613.0775**	**399**				

On comparing the mean differences in the scores of increased flatus, after each assessment in the trial group, using the ANOVA, the F value was found to be significant at 0.1 % level ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.



Graph 69: Mean distribution of efficacy of therapy on increased flatus – Trial group



Graph 70: Mean distribution of efficacy of therapy on increased flatus – Control group

Table 109 F test – control group – Increased flatus

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	56.7075	3	18.9025	12.50211	7.94E-08	2.627441
Within Groups	598.73	396	1.51194			
Total	**655.4375**	**399**				

On comparing the mean differences in increased flatus, after each assessment in the control group, using the ANOVA, the F value was found to be significant at 0.1% level. ($P < 0.001$) indicating that, there is difference in the mean scores, during and after the intervention.

Multiple Comparison in between groups – trial group

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF showed high significance at 0.1% level in increased flatus. The comparisons AT1 Vs AT2 and AF was also significant at 0.1% level (P<0.001). The comparison between AT2 and AF was also significant, but at the 5% level.

Table -110 Tuckey Kramer test - trial group – Increased flatus

Comparison		MD	q	P Value
BT	AT1	0.41	9.089	<0.001
BT	AT2	0.67	14.853	<0.001
BT	AF	0.86	19.066	<0.001
AT1	AT2	0.26	5.764	<0.001
AT1	AF	0.45	9.976	<0.001
AT2	AF	0.19	4.212	<0.05

Multiple Comparison in between groups – control group

Table -111 Tuckey Kramer test - trial group – Increased flatus

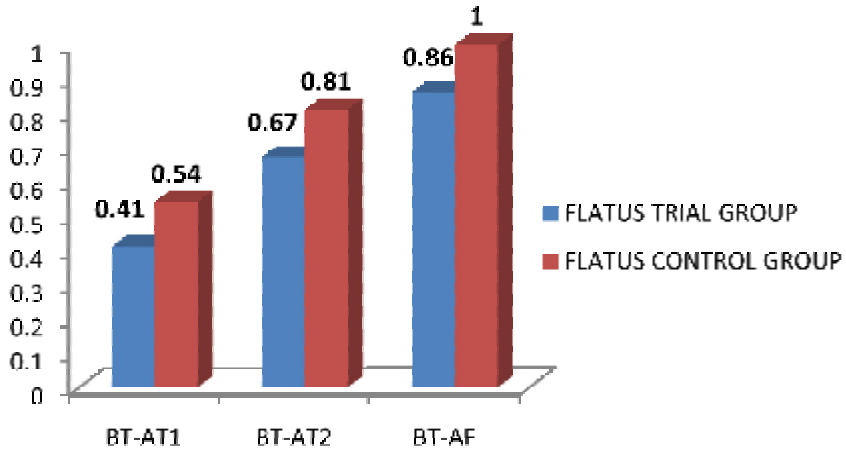
Comparison		MD	q	P Value
BT	AT1	0.54	11.08	<0.001
BT	AT2	0.81	16.62	<0.001
BT	AF	1	20.519	<0.001
AT1	AT2	0.27	5.54	<0.001
AT1	AF	0.46	9.439	<0.001
AT2	AF	0.19	3.899	<0.05

On comparing the various group mean scores of assessment in increased flatus among themselves, in the control group, the comparison between BT Vs AT1, AT2 and AF showed high significance at P< 0.001 level. The comparisons AT1 Vs AT2 and AF was also significant at 0.1% level (P<0.001). The comparison of AT2 and AF was also significant, but only at the 5% level. (P<0.05)

Comparison between groups- unpaired t test – increased flatus

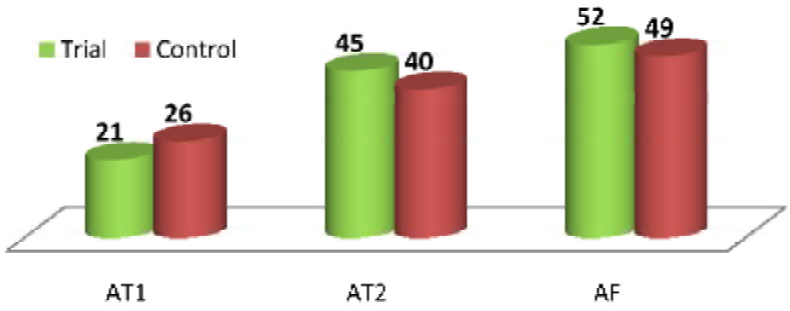
Table -112 Unpaired t test - between groups – Increased flatus

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.41 ± 0.51	0.54 ±0.58	-8.158	< 0.001
BT-AT2	0.67 ± 0.78	0.81 ± 0.77	-5.825	< 0.001
BT - AF	0.86 ± 0.92	1 ± 0.99	-4.719	< 0.001



Graph 71: Mean distribution of the efficacy of therapy between groups- Increased flatus

On comparing the efficacy of treatment in the symptom of increased flatus, between the trial and control, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level (P< 0.001). The comparison of mean reduction before treatment (BT) and on second assessment (AT2) was significant also



Graph 72: Distribution of the percentage of relief between groups- Increased flatus

at 0.1% level. (P< 0.001). On comparing the two groups between BT and on third assessment (AF), it was also highly significant. (P< 0.001)

The percentage of relief for the symptom of increased flatus was 21% during the first assessment (AT1) in the trial and to 26% in the control group. During the second assessment (AT2), the percentage of relief became 45% in the trial group and 40% in the control. During the third assessment (AF), it was 52% in the trial and 49% in the control.

6. DECREASED STOOL

Table 113 Mean distribution of efficacy of therapy on Decreased stool - both groups

	Trial	Control
BT	0.63 ± 1.29	0.55 ± 1.19
AT1	0.48 ± 1.00	0.49 ± 1.07
AT2	0.46 ± 0.96	0.43 ± 0.95
AF	0.41 ± 0.85	0.41 ± 0.90

In the trial group, the symptom of decreased stool of 100 patients showed a mean severity score of 0.63 ± 1.29 while in the control, it was 0.55 ± 1.19. After

the treatment, during first assessment (AT1), the mean score was reduced to 0.48 ± 1.00 in the study group, while in the control it was reduced to 0.49 ± 1.07. During the second assessment (AT2), the mean score in the study group was 0.46 ± 0.96, while in the control, it was 0.43 ± 0.95. At the follow-up (AF), the mean score in the study group was 0.41 ± 0.85, while in the control, it was 0.41 ± 0.90.

Table 114 F test - trial group – decreased stool

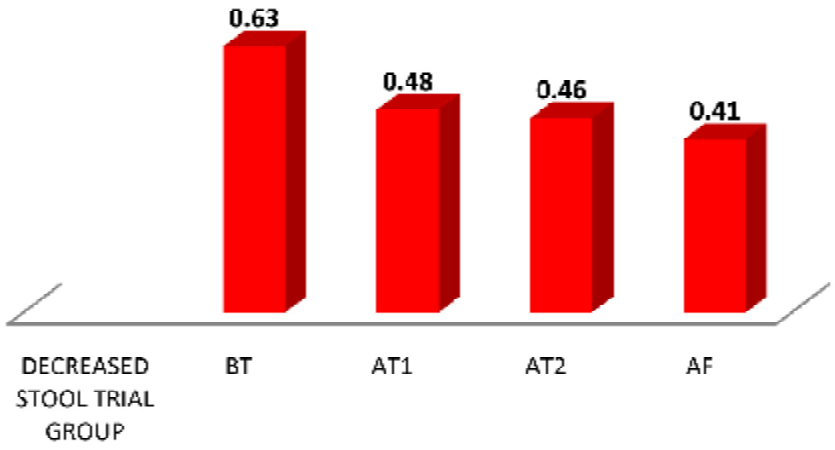
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	1.9475	3	0.6491667	0.6270153	0.597905	2.627441
Within Groups	409.99	396	1.0353283			
Total	**411.9375**	**399**				

On comparing the mean differences in the symptom of decreased stool, after each assessment in the trial group, using the Analysis of variance, the F value was found to be not significant (P> 0.05) indicating that, there is no significant difference in the mean scores during and after the treatment.

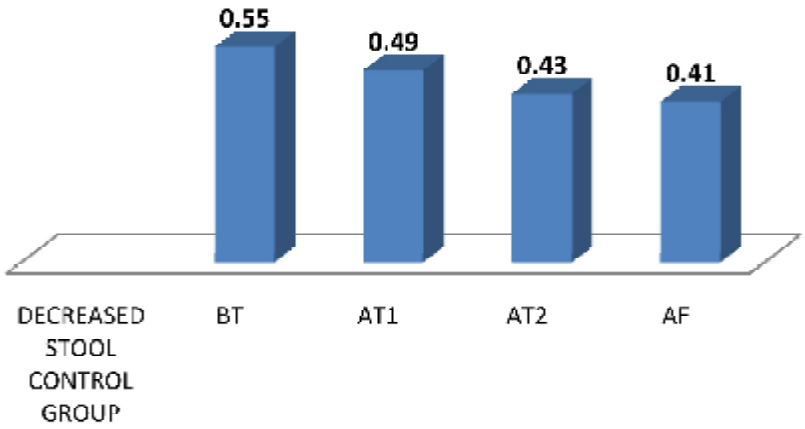
Table 115 F test - control group – decreased stool

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	1.2	3	0.4	0.374964	0.771111	2.627441
Within Groups	422.44	396	1.066768			
Total	**423.64**	**399**				

On comparing the mean differences in the symptom of decreased stool, after each assessment in the control group, using the ANOVA, the F value was found to be not significant (P> 0.05) indicating that there is no significant difference in the mean scores during and after the treatment.



Graph 73: Mean distribution of efficacy of therapy on Decreased stool – Trial group



Graph 74: Mean distribution of efficacy of therapy on Decreased stool – Control group

Multiple Comparison in between groups – trial group

Table -116 Tuckey Kramer test - trial group – Decreased stool

Comparison		MD	q	P Value
BT	AT1	0.15	5.736	<0.001
BT	AT2	0.17	6.501	<0.001
BT	AF	0.22	8.413	<0.001
AT1	AT2	0.02	0.7648	>0.05
AT1	AF	0.07	2.677	>0.05
AT2	AF	0.05	1.912	>0.05

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF was highly significant (P< 0.001). The comparisons AT1 Vs AT2 and AF were not significant (P> 0.05). The comparison between AT2 and AF was also not significant.

Multiple Comparison in between groups – control group

Table -117 Tuckey Kramer test - Control group – Decreased stool

Comparison		MD	q	P Value
BT	AT1	0.06	2.783	>0.05
BT	AT2	0.12	5.567	<0.001
BT	AF	0.14	6.495	<0.001
AT1	AT2	0.06	2.783	>0.05
AT1	AF	0.08	3.711	<0.05
AT2	AF	0.02	0.9278	>0.05

On comparing the various group mean scores of assessment among themselves in the control, the comparison between BT Vs AT2 and AF showed high significance at 0.1% level. But the comparison BT Vs AT1 was not significant. The comparisons AT1 Vs AT2 was not significant. But the comparison AT1 Vs AF was significant at 5% level. The comparison between AT2 and AF was also not significant.

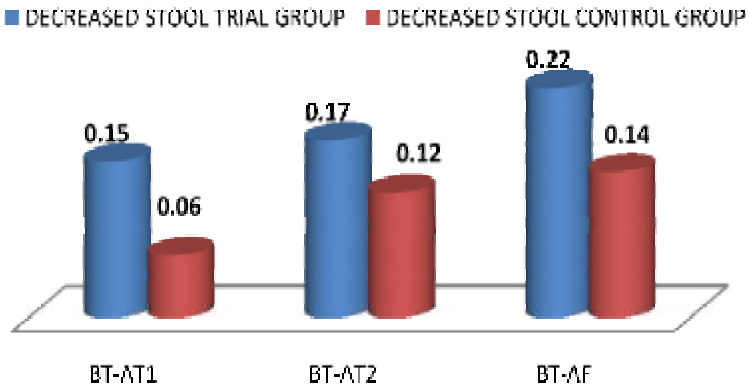
Comparison between two groups- unpaired t test – decreased stool

On comparing the efficacy of intervention in the symptom of decreased stool, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (< 0.001). The comparison of mean reduction before treatment (BT) and on second assessment (AT2) was

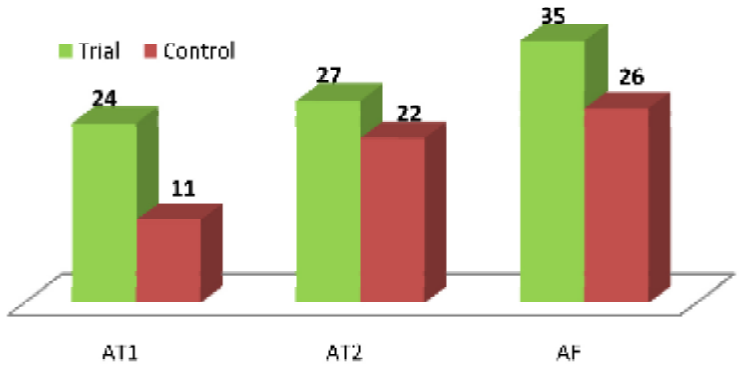
significant also at 0.1% level. (P< 0.001) On comparing the two groups between BT and on third assessment (AF), it was also highly significant. (P< 0.001)

Table -118 Unpaired t test - between groups – Decreased stool

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.15 ± 0.39	0.06 ± 0.43	-9.034	< 0.001
BT-AT2	0.17 ± 0.43	0.12 ± 0.38	-7.833	< 0.001
BT - AF	0.22 ± 0.5	0.14 ± 0.40	-6.510	< 0.001



Graph 75: Mean distribution of the efficacy of therapy between groups - Decreased stool



Graph 76: Distribution of the percentage of relief between groups - Decreased stool

The percentage of relief for the symptom of decreased stool was 24% during the first assessment (AT1) in the trial and 11% in the control group. During the second assessment (AT2), the percentage of relief was 27% in the trial group and 22% in the control. During the third assessment (AF), it was 35% in the trial group and 26% in the control.

7. INCREASED STOOL

None of the patients were recorded with the symptom of increased stool in both the groups and hence no statistical tests were performed, in this regard.

8. LOOSE STOOL

Table 119 Mean distribution of efficacy on loose stool - both groups

	Trial	Control
BT	0.3 $\pm$ 0.93	0.21 $\pm$ 0.84
AT1	0.22 $\pm$ 0.68	0.16 $\pm$ 0.65
AT2	0.18 $\pm$ 0.56	0.13 $\pm$ 0.53
AF	0.12 $\pm$ 0.38	0.1 $\pm$ 0.41

In the trial group, the symptom of loose stool among the 100 patients showed a mean severity score of 0.3 $\pm$ 0.93 while in the control group, it was 0.21 $\pm$ 0.84. After the medication, during first assessment (AT1), the mean score was reduced to 0.22 $\pm$ 0.68 in the study group, while in the control it was reduced to 0.49 $\pm$ 1.07. During the second assessment (AT2), the mean score in the study group was 0.18 $\pm$ 0.56, while in the control, it was 0.13 $\pm$ 0.53. During the follow-up (AF), the mean score in the study group was 0.12 $\pm$ 0.38, while in the control, it was 0.1 $\pm$ 0.41.

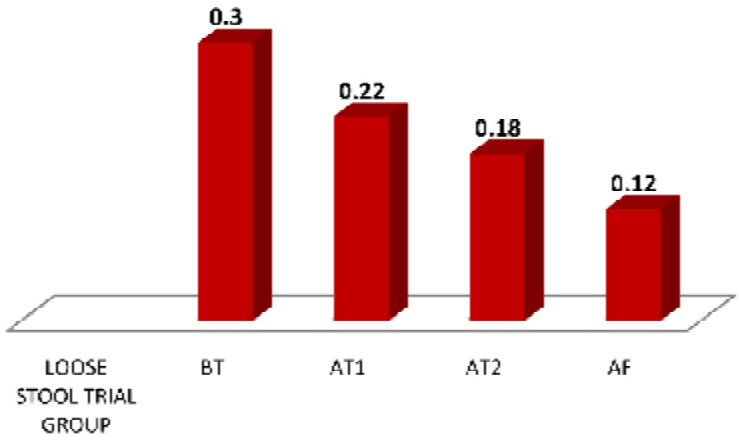
Table 120 F test - trial group – loose stool

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	0.0675	3	0.0225	1	0.392789	2.627441
Within Groups	8.91	396	0.0225			
Total	**8.9775**	**399**				

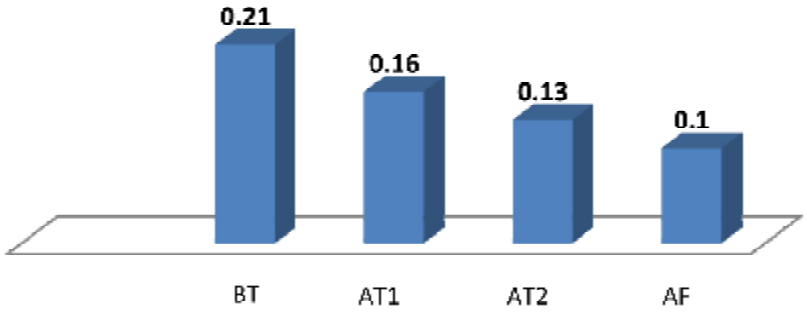
On comparing the mean differences in the symptom of loose stool, after each assessment in the trial group, using ANOVA, the F value was found to be not significant (P> 0.05) indicating that, there is no significant difference in the mean scores during and after the treatment.

Table 121 F test - control group – loose stool

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	0.0275	3	0.009167	0.060855	0.980324	2.627441
Within Groups	59.65	396	0.150631			
Total	59.6775	399				



Graph 77: Mean distribution of efficacy on loose stool – Trial group



Graph 78: Mean distribution of efficacy of therapy on loose stool – Control group

On comparing the mean differences in the symptom of loose stool, after each assessment in the control group, using the ANOVA, the F value was found to be not significant (P> 0.05) indicating that there is no significant difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

Table -122 Tuckey Kramer test - Trial group – Loose stool

Comparison		MD	q	P Value
BT	AT1	0.08	3.139	> 0.05
BT	AT2	0.12	4.709	< 0.01
BT	AF	0.18	7.063	< 0.001
AT1	AT2	0.04	1.57	> 0.05
AT1	AF	0.1	3.924	< 0.05
AT2	AF	0.06	2.354	> 0.05

On comparing the various group mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1 was not significant (P> 0.05). The comparison between BT and AT2 was significant at 1% level (P< 0.01). The comparison between BT and AF showed high significance(P< 0.001). The

comparisons AT1 Vs AT2 was not significant. The comparisons AT1 Vs AF were significant (P< 0.05). The comparison between AT2 and AF was not significant.

Multiple Comparison in between groups – control group

Table -123 Tuckey Kramer test - Control group – Loose stool

Comparison		MD	q	P Value
BT	AT1	0.05	2.359	>0.05
BT	AT2	0.08	3.775	<0.05
BT	AF	0.11	5.19	<0.01
AT1	AT2	0.03	1.416	>0.05
AT1	AF	0.06	2.831	>0.05
AT2	AF	0.03	1.416	>0.05

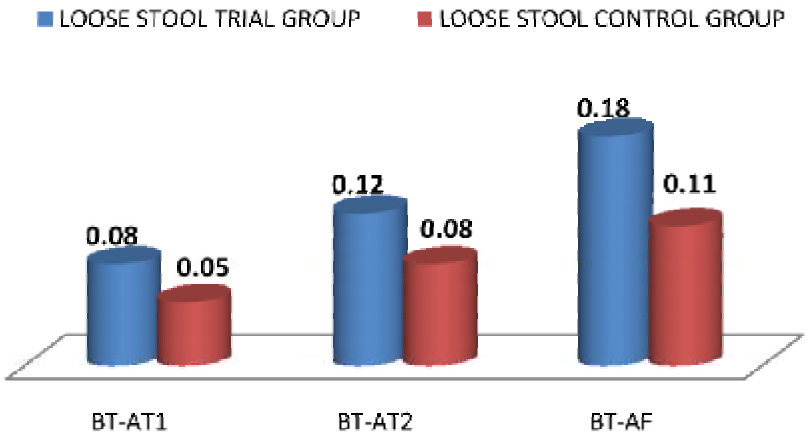
On comparing the various group mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1 was not significant (P> 0.05). The comparison between BT and AT2 was significant at 5 % level (P< 0.05). The comparison between BT and AF showed high significance at P< 0.01 level. The comparisons, AT1 VS AT2 and AF were not significant (P> 0.05). The comparison between AT2 and AF was also not significant (P> 0.05).

Comparison between the groups- unpaired t test – loose stool

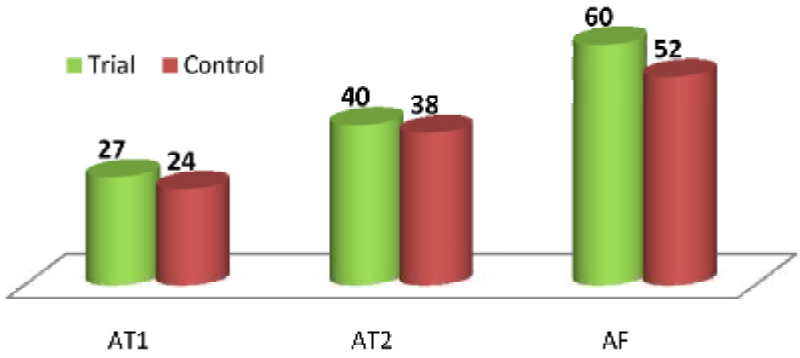
On comparing the efficacy of treatment in the symptom of loose stool, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (P< 0.001). The comparison between mean reduction before treatment (BT) and on second assessment (AT2) was significant also at 0.1% level. (P< 0.001). On comparing the two groups between BT and on third assessment (AF), it was also highly significant. (P< 0.001)

Table -124 Unpaired t test - between groups – Loose stool

Comparison	M D Trial	M D Control	t value	P value
BT - AT1	0.08 ± 0.31	0.05 ± 0.22	-12.449	< 0.001
BT-AT2	0.12 ± 0.41	0.08 ± 0.37	-8.367	< 0.001
BT - AF	0.18 ± 0.58	0.11 ± 0.47	-5.792	< 0.001



Graph 79: Mean distribution of the efficacy of therapy between groups- Loose stool



Graph 80: Distribution of the percentage of relief between groups- Loose stool

The percentage of relief for the symptom of loose stool was 27% during the first assessment (AT1) in the trial group and 24% in the control. During the second

assessment (AT2), the percentage of relief was 40% in the trial and 38% in the control group. During the final assessment (AF), it was 60% in the trial group and 52% in the control group.

9. HARD STOOL

Table 125 Mean distribution of efficacy on Hard stool - both groups

	Trial	Control
BT	2.63 ± 0.99	2.83 ± 0.73
AT1	2.26 ± 0.96	2.37 ± 0.77
AT2	1.88 ± 0.81	2.00 ± 0.71
AF	1.69 ± 0.88	1.65 ± 0.74

In the trial group, the symptom of hard stool among 100 subjects showed a mean severity score of 2.63 ± 0.99 while in the control, it was 2.83 ± 0.73. After the treatment, during the first assessment (AT1), the mean score was reduced to 2.26 ± 0.96 in the study group, while in the control, it was reduced to 2.37 ± 0.77. In the second assessment (AT2), the mean score in the study group was 1.88 ± 0.81, while in the control, it was 2 ± 0.71. During the follow-up (AF), the mean score in the study group was 1.69 ± 0.88, while in the control, it was 1.65 ± 0.74.

Table 126 F test - trial group – Hard stool

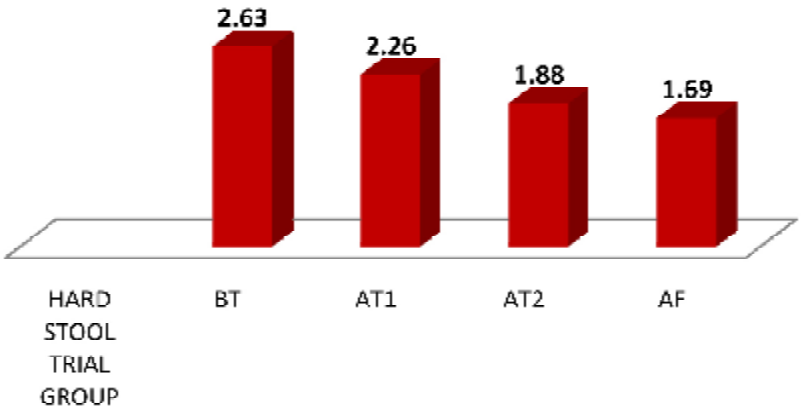
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	54.71233	3	18.2374	22.2674	2.429 E-13	2.6275
Within Groups	323.5132	395	0.8190			
Total	378.2256	398				

On comparing the mean differences in the symptom of hard stool, after each assessment in the trial group, using the ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that, there is significant difference in the mean scores during and after the treatment.

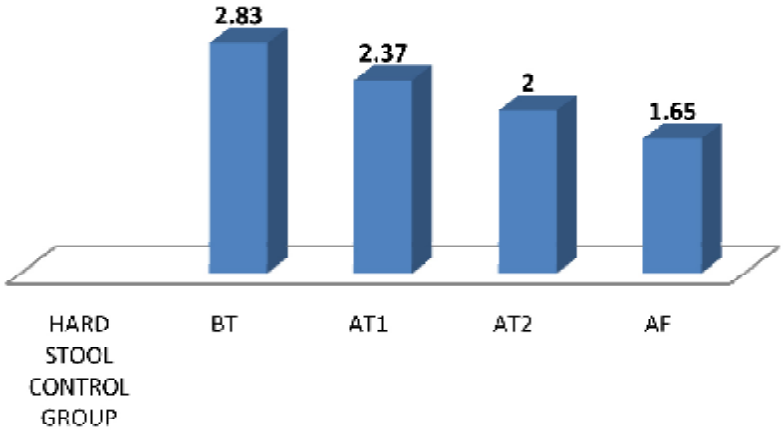
Table 127 F test - control group – Hard stool

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	76.7675	3	25.58917	46.8765	6.05 E-26	2.627441
Within Groups	216.17	396	0.54588			
Total	292.9375	399				

On comparing the mean differences in the symptom of hard stool, after each assessment in the control group, using the ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that, there is significant difference in the mean scores during and after the treatment.



Graph 81: Mean distribution of efficacy of therapy on hard stool – Trial group



Graph 82: Mean distribution of efficacy of therapy on hard stool – Control group

Multiple Comparison in between groups – trial group

Table -128 Tuckey Kramer test - trial group – Hard stool

Comparison		MD	q	P Value
BT	AT1	0.37	8.694	<0.001
BT	AT2	0.75	17.623	<0.001
BT	AF	0.94	22.088	<0.001
AT1	AT2	0.38	8.929	<0.001
AT1	AF	0.57	13.394	<0.001
AT2	AF	0.19	4.465	<0.01

On comparing the various groups mean scores of assessment among themselves in hard stool in the trial group, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1% level (P< 0.001). The comparison between AT1 Vs AT2 and AF was also significant at 0.1% level (P< 0.001). The comparison between AT2 and AF was also significant, but at 1% level (P<0.01).

Multiple Comparison in between groups – control group

Table -129 Tuckey Kramer test – control group – Hard stool

Comparison		MD	q	P value
BT	AT1	0.46	11.049	<0.001
BT	AT2	0.83	19.935	<0.001
BT	AF	1.18	28.342	<0.001
AT1	AT2	0.37	8.887	<0.001
AT1	AF	0.72	17.293	<0.001
AT2	AF	0.35	8.407	<0.001

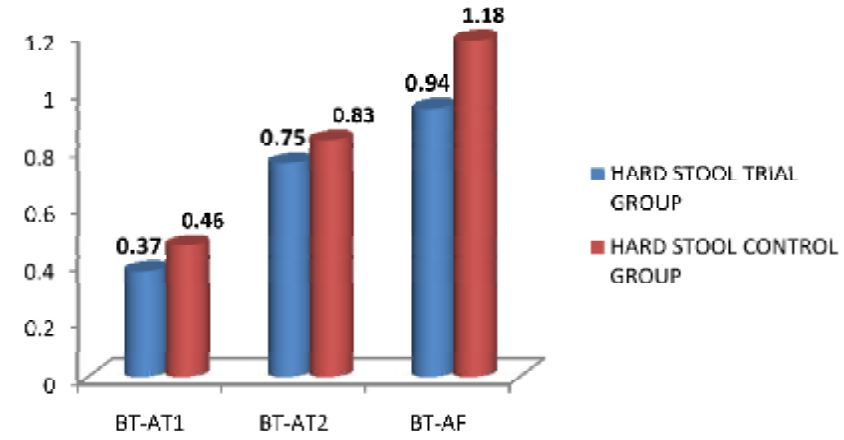
On comparing the various groups mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1% level (P< 0.001). The comparison between AT1 Vs AT2 and AF was also significant, at 0.1% level (P< 0.001). The comparison between AT2 and AF was also significant at the same level (P<0.001).

Comparison between two groups- unpaired t test – hard stool

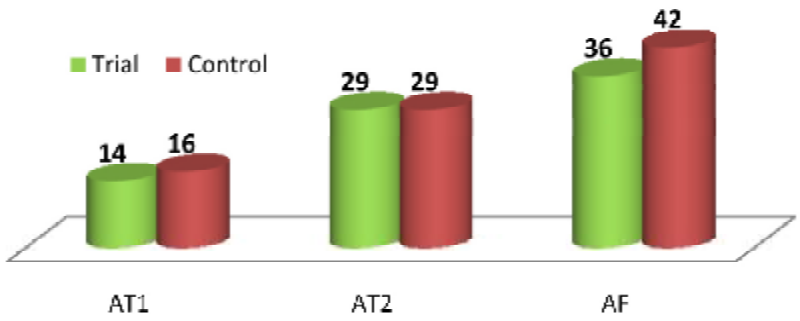
Table -130 Unpaired t test - between groups – Hard stool

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.37 $\pm$ 0.51	0.46 $\pm$ 0.50	-8.289	< 0.001
BT - AT2	0.75 $\pm$ 0.52	0.83 $\pm$ 0.55	-7.653	< 0.001
BT - AF	0.94 $\pm$ 0.75	1.18 $\pm$ 0.69	-7.276	< 0.001

On comparing the efficacy of treatment in the symptom of hard stool, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (P< 0.001). The comparison



Graph 83: Mean distribution of the efficacy of therapy between groups - Hard stool



Graph 84: Distribution of the percentage of relief between groups- Hard stool

between mean reduction before treatment (BT) and on second assessment (AT2) was significant also at 0.1% level. (P< 0.001). On comparing the two groups between BT and follow up (AF), it was also highly significant. (P< 0.001)

The percentage of relief for the symptom of hard stool was 14% during the first assessment (AT1) in the trial group and 16% in the control. During the second assessment (AT2), the percentage of relief was 29% in both the trial and control group. During the third assessment (AF), it was 36% in the trial group and became 42% in the control group.

10. URGENCY OF DEFECATION

Table 131 Mean distribution of efficacy of therapy on Urgency of defecation - both groups

	Trial	Control
BT	0.12 ± 0.61	0.15 ± 0.66
AT1	0.1 ± 0.50	0.11 ± 0.49
AT2	0.08 ± 0.42	0.11 ± 0.49
AF	0.07 ± 0.36	0.09 ± 0.43

In the trial group, the symptom of urgency of defecation showed a mean severity score of 0.12 ± 0.61 while in the control, it was 0.15 ± 0.66. After the intervention, during the first assessment (AT1), the mean score was reduced to 0.1 ± 0.50 in the study group, while in the control, it was reduced to 0.11 ± 0.49. On the second assessment (AT2), the mean score in the study group was 0.08 ± 0.42, while in the control group, it was 0.11 ± 0.49. At the follow-up (AF), the mean score in the study group was 0.07 ± 0.36, while in the control, it was 0.09 ± 0.43.

Table 132 F test - trial group – urgency of defecation

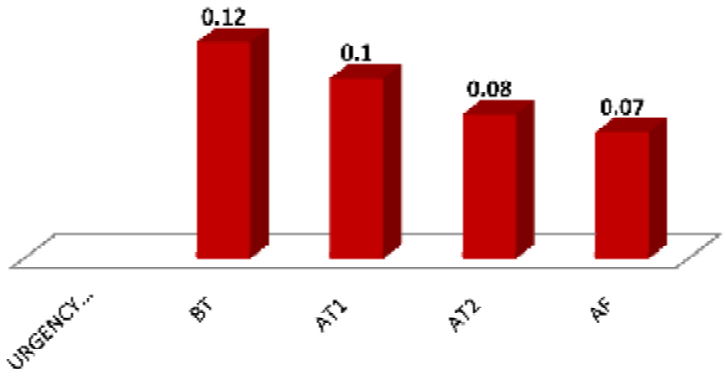
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	0.1475	3	0.0492	0.21295	0.88742	2.6274
Within Groups	91.43	396	0.23089			
Total	**91.5775**	**399**				

On comparing the mean differences in the symptom of urgency of defecation, after each assessment in the trial group using ANOVA, the F value was found to as not significant (P> 0.05) indicating that there is no significant difference in the mean scores during and after the treatment.

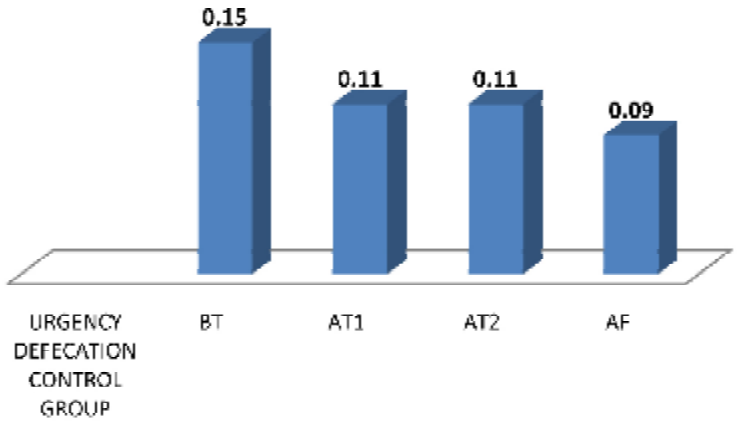
Table 133 F test – control group – urgency of defecation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	0.0475	3	0.01583	0.08606	0.967645	2.627441
Within Groups	72.85	396	0.18396			
Total	72.8975	399				

On comparing the mean differences in the symptom of urgency of defecation, after each assessment in the control group, using the ANOVA, the F value was found as not significant (P> 0.05) indicating that there is no significant difference in the mean scores during and after the treatment.



Graph 85: Mean distribution - Urgency of defecation – Trial group



Graph 86: Mean distribution - Urgency of defecation – Control group

Multiple Comparison in between groups – trial group

Table -134 Tuckey Kramer test - trial group – Urgency of defecation

Comparison		MD	q	P value
BT	AT1	1.44	26.612	<0.001
BT	AT1	0.02	1.456	>0.05
BT	AT2	0.04	2.912	>0.05
BT	AF	0.05	3.640	>0.05
AT1	AT2	0.02	1.456	>0.05
AT1	AF	0.03	2.184	>0.05
AT2	AF	0.01	0.728	>0.05

On comparing the various groups mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF was not significant. (P> 0.05) The comparison between AT1 Vs AT2 and AF was also not significant. The comparison of AT2 and AF was also not significant.

Multiple Comparison in between groups – control group

On comparing the various groups mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF was not significant. (P> 0.05) The comparison between AT1 Vs AT2 and AF was also not significant. The comparison between AT2 and AF was also not significant.

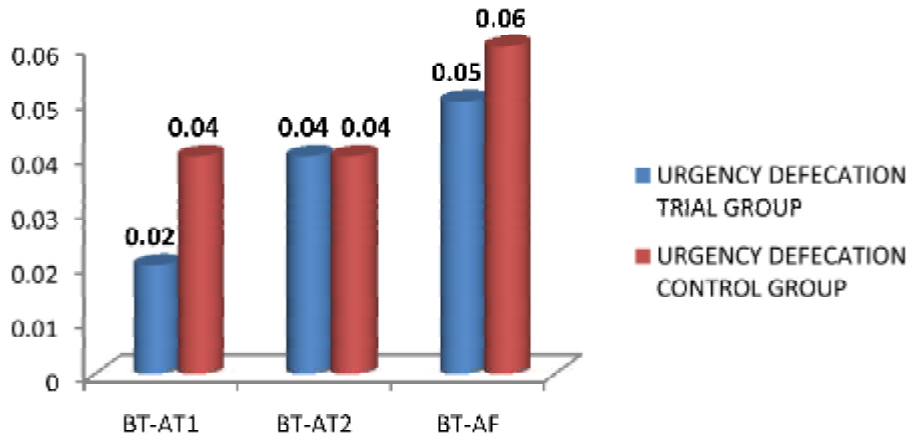
Table -135 Tuckey Kramer test – Control – Urgency of defecation

Comparison		MD	q	P Value
BT	AT1	0.04	2.744	>0.05
BT	AT2	0.04	2.744	>0.05
BT	AF	0.06	4.116	<0.05
AT1	AT2	0	0	>0.05
AT1	AF	0.02	1.372	>0.05
AT2	AF	0.02	1.372	>0.05

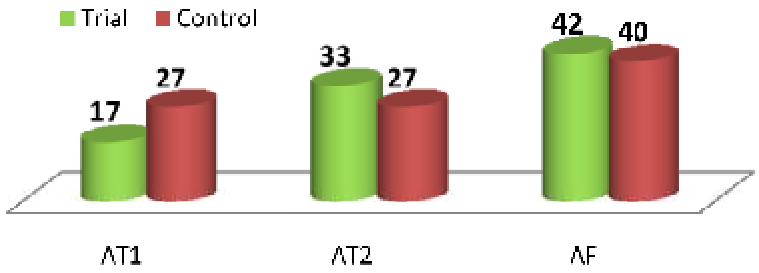
Comparison between groups- unpaired t test – urgency defecation

Table -136 Unpaired t test - between groups – Urgency of defecation

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.02 ± 0.14	0.04 ± 0.20	-21.48	< 0.001
BT - AT2	0.04 ± 0.24	0.04 ± 0.20	-15.99	< 0.001
BT - AF	0.05 ± 0.30	0.06 ± 0.31	-11.83	< 0.001



Graph 87 : Mean distribution of efficacy - Urgency of defecation



Graph 88 : Percentage of relief - Urgency of defecation

On comparing the efficacy in the symptom of urgency of defecation, between the trial and control groups, mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level. (P< 0.001). The comparison between mean score before treatment (BT) and on second assessment (AT2) was significant also at 0.1% level. On comparing the two groups between BT and on third assessment (AF), it was also highly significant. (P< 0.001).

The percentage of relief for the symptom of urgency of defecation was found as 17% during the first assessment (AT1) in the trial group and 27% in the control. During the second assessment (AT2), the percentage of relief was 33% in the trial group and 27% in the control. During the follow up (AF), it was 42% in the trial and 40% in the control.

11. FEELING OF INCOMPLETE EVACUATION

In the trial group, the symptom of feeling of incomplete evacuation showed a mean severity score of 0.48 ± 1.08 while in the control group, it was 0.49 ± 1.10. After the intervention, during the first assessment (AT1), the mean score was reduced to 0.35 ± 0.78 in the study group, while in the control, it was reduced to 0.34 ± 0.76. In the second assessment (AT2), the mean score in the study group was 0.27 ± 0.63, while in the control, it was 0.3 ± 0.69. During the follow-up (AF), the mean score in the study group was 0.22 ± 0.52, while in the control, it was 0.26 ± 0.61.

Table 137 Mean distribution of efficacy of therapy on Feeling of incomplete evacuation - both groups

	Trial	Control
BT	0.48 ± 1.08	0.49 ± 1.10
AT1	0.35 ± 0.78	0.34 ± 0.76
AT2	0.27 ± 0.63	0.3 ± 0.69
AF	0.22 ± 0.52	0.26 ± 0.61

Table 138 F test - trial group – Feeling of incomplete evacuation

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	2.9	3	0.966667	1.760324	0.154203	2.627441
Within Groups	217.46	396	0.54914			
Total	220.36	399				

On comparing the mean differences in the symptom of feeling of incomplete evacuation, after each assessment in the trial group, using ANOVA, the F value was found to be not significant (P> 0.05) indicating that there is no significant difference in the mean scores during and after the treatment.

Table 139 F test - control group – feeling of incomplete evacuation

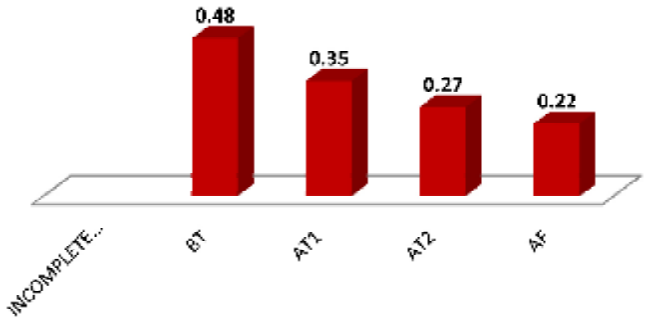
Source of Variation	SS	df	MS	F	P value	F crit
Between Groups	3.0275	3	1.009167	1.538992	0.203881	2.627441
Within Groups	259.67	396	0.655732			
Total	262.6975	399				

On comparing the mean differences in the symptom of feeling of incomplete evacuation, after each assessment in the control group, using ANOVA, the F value

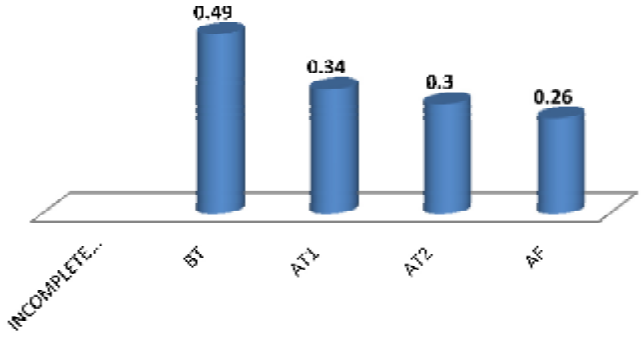
was found to be not significant (P> 0.05) indicating that, there is no significant difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

On comparing the various groups mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1 was significant at 5% level (P< 0.05). BT Vs AT2 and AF was highly significant. (P< 0.001) The comparison between AT1 Vs AT2 was not significant. AT1 Vs AF was significant at 5% level. (P< 0.05) The comparison between AT2 and AF was not significant.



Graph 89: Efficacy on Feeling of incomplete evacuation – Trial group



Graph 90: Efficacy on Feeling of incomplete evacuation – Control group

Table -140 Tuckey Kramer test - trial group – Feeling of incomplete evacuation

Comparison		MD	q	P Value
BT	AT1	0.13	4.424	< 0.05
BT	AT2	0.21	7.147	< 0.001
BT	AF	0.26	8.849	< 0.001
AT1	AT2	0.08	2.723	> 0.05
AT1	AF	0.13	4.424	< 0.05
AT2	AF	0.05	1.702	> 0.05

Multiple Comparison in between groups – control group

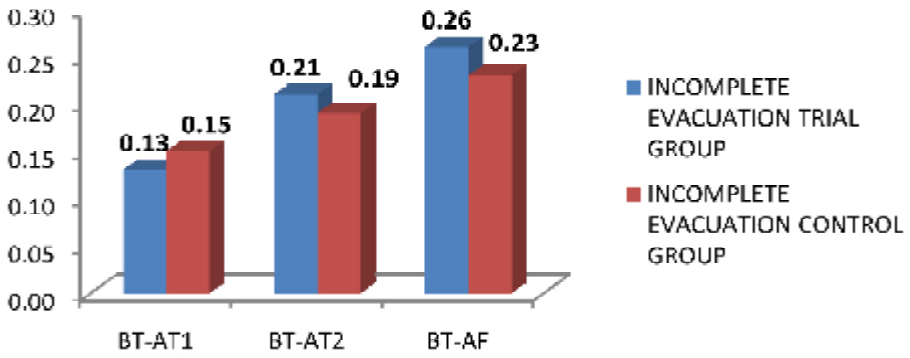
Table -141 Tuckey Kramer test - control group – Feeling of incomplete evacuation

Comparison		MD	q	P Value
BT	AT1	0.15	5.611	<0.001
BT	AT2	0.19	7.108	<0.001
BT	AF	0.23	8.604	<0.001
AT1	AT2	0.04	1.496	>0.05
AT1	AF	0.08	2.993	>0.05
AT2	AF	0.04	1.496	>0.05

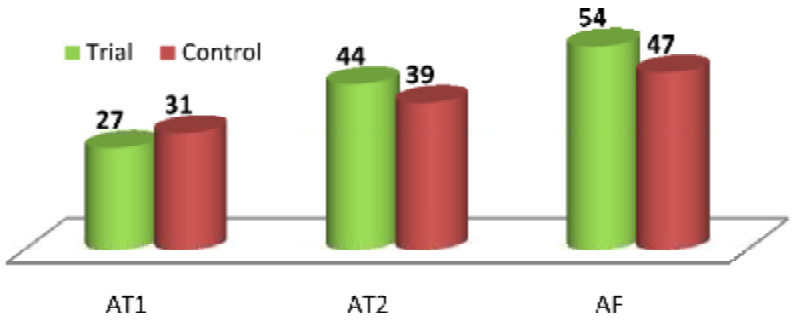
On comparing the various groups mean scores of assessment in the feeling of incomplete evacuation among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF was highly significant (P< 0.001). AT1 Vs AT2 and AF was not significant. The comparison between AT2 and AF was also not significant.

Comparison between groups- unpaired t test – feeling of incomplete evacuation

On comparing the efficacy of treatment in the symptom of feeling of incomplete evacuation, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 0.1% level (P< 0.001). The comparison between before treatment (BT) and on second assessment (AT2) was significant also at 0.1% level. (P< 0.001). On comparing the two groups between BT and on third assessment (AF), it was also highly significant. (P< 0.001)



Graph 91: Efficacy between groups - Feeling of incomplete evacuation



Graph 92: Percentage of relief between groups - Feeling of incomplete evacuation

Table -142 Unpaired t test - between groups – Feeling of incomplete evacuation

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.13 ± 0.34	0.15 ± 0.36	-10.54	< 0.001
BT-AT2	0.21 ± 0.52	0.19 ± 0.46	-6.89	< 0.001
BT - AF	0.26 ± 0.63	0.23 ± 0.57	-5.55	< 0.001

The percentage of relief for the symptom of feeling of incomplete evacuation was found as 27% during the first assessment (AT1) in the trial group and 31% in control. During the second assessment (AT2), the percentage of relief was 44% in trial and 39% in the control group. During the third assessment (AF), it was 54% in the trial group and 47% in the control.

12. TOTAL SCORE - GSRS

Table 143 Mean distribution of efficacy of therapy on Total score GSRS - both groups

	Trial	Control
BT	33.86 ± 5.0	34.75 ± 4.09
AT1	24.64 ± 4.24	26.37 ± 4.27
AT2	18.39 ± 4.24	21.6 ± 4.47
AF	14.11 ± 4.39	18.42 ± 3.39

In the trial group, the total score of GSRS among the 100 participants showed a mean severity score of 33.86 ± 5.0 while in the control group, it was 34.75 ± 4.09. During the first assessment (AT1), the mean score was reduced to 24.64 ± 4.24 in the study group, while in the control, it was reduced to 26.37 ± 4.27. In the

second assessment (AT2), the mean score in the study group was 18.39 ± 4.24, while in the control, it was 21.6 ± 4.47. During the follow-up (AF), the mean score in the study group was 14.11 ± 4.39, while in the control, it was 18.42 ± 3.39.

Table 144 F test - trial group – GSRS total score

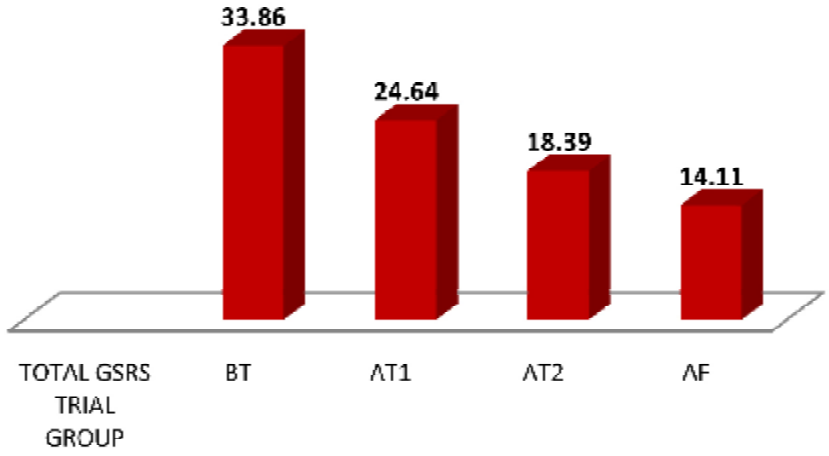
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	20240.67	3	6746.89	135.83	1.63 E-60	2.627441
Within Groups	19670.03	396	49.6718			
Total	39910.7	399				

On comparing the mean differences in the total score of GSRS, after each assessment in the trial group, using the ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

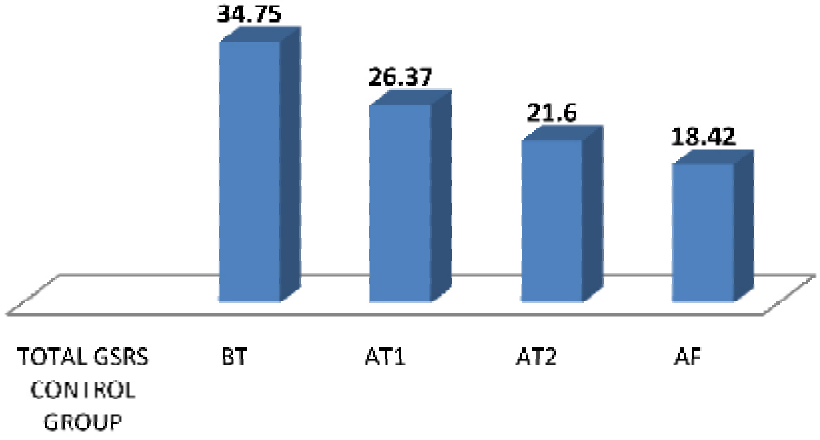
Table 145 F test - control group – GSRS total score

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	15147.09	3	5049.03	303.8432	2.6E-102	2.627441
Within Groups	6580.42	396	16.61722			
Total	21727.51	399				

On comparing the mean differences in the total score of GSRS, after each assessment in the control group, using the ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.



Graph 93: Mean distribution of efficacy of therapy on Total score GSRS – Trial group



Graph 94: Mean distribution of efficacy of therapy on Total score GSRS – Control group

Multiple Comparison in between groups – trial group

On comparing the various groups mean scores of assessment among themselves in the trial group, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level ($P < 0.001$) The comparison between AT1 Vs AT2 and AF was also significant at the same level ($P < 0.001$). The comparison between AT2 and AF was also similarly significant ($P < 0.001$).

Table -146 Tuckey Kramer test - trial group – Total score GSRS

Comparison		MD	q	P Value
BT	AT1	9.22	35.671	<0.001
BT	AT2	15.47	59.852	<0.001
BT	AF	19.75	76.411	<0.001
AT1	AT2	6.25	24.181	<0.001
AT1	AF	10.53	40.74	<0.001
AT2	AF	4.28	16.559	<0.001

Comparison in between groups – TUCKEY KRAMER – control group

Table -147 Tuckey Kramer test - Control group – Total score GSRS

Comparison		MD	q	P Value
BT	AT1	8.38	38.948	<0.001
BT	AT2	13.15	61.118	<0.001
BT	AF	16.33	75.897	<0.001
AT1	AT2	4.77	22.17	<0.001
AT1	AF	7.95	36.949	<0.001
AT2	AF	3.18	14.78	<0.001

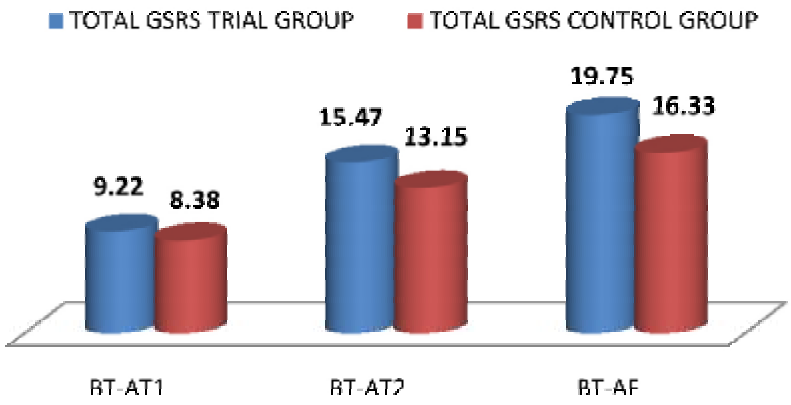
On comparing the various groups mean scores of assessment among themselves in the control group, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level ($P< 0.001$) The comparison between AT1 Vs AT2 and AF was also significant at the same level ($P< 0.001$). The comparison between AT2 and AF was also similarly significant ($P< 0.001$).

Comparison between groups - unpaired t test – GSRS total score

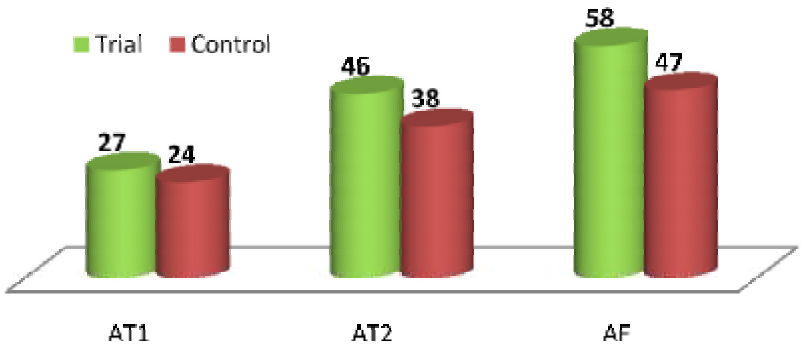
Table -148 Unpaired t test - between groups – Total score GSRS

Comparison	M D trial	M D control	t	P
BT - AT1	9.22 ± 2.84	8.38 ± 2.55	0.89	> 0.05
BT - AT2	15.47 ± 4.34	13.15 ± 3.67	3.20	< 0.01
BT - AF	19.75 ± 5.03	16.33 ± 3.30	4.85	< 0.001

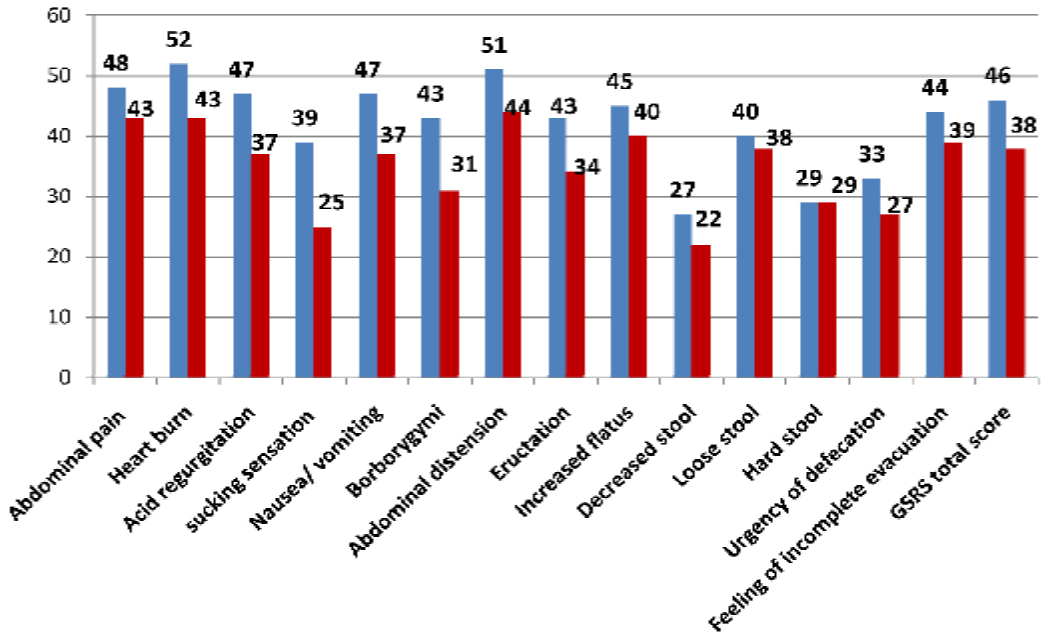
On comparing the efficacy of treatment on the total score of GSRS, between the trial and control groups with unpaired t test, the mean reduction before treatment (BT) and on first assessment (AT1) was not significant (P>0.05). The



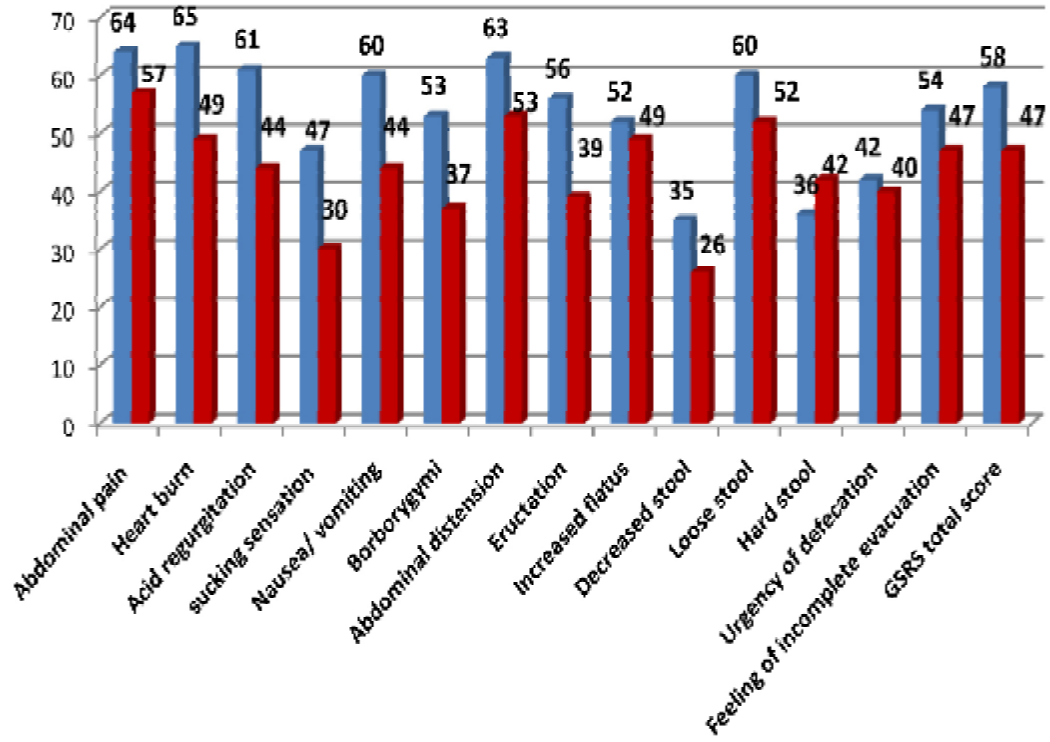
Graph 95: Efficacy of therapy between groups- Total score GSRS



Graph 96: Distribution of the percentage of relief between groups- Total score GSRS



Graph 97: Percentage of relief on individual symptoms GSRS - after trial



Graph 98: Percentage of relief on individual symptoms GSRS- after follow up

comparison between mean reduction before treatment (BT) and on second assessment (AT2) was significant at 0.1% level (P<0.001). On comparing the two groups between BT and third assessment (AF), it was also highly significant. (P< 0.001)

The response attained during the study on the individual symptoms of GSRS was assessed individually both after the treatment, and also after the followup. It was recorded that after the trial, maximum response was attained in the symptoms of abdominal pain, heart burn and abdominal distension.

Table 149 Level of significance on the efficacy on GSRS

Symptom	AT1	AT2	AF
Abdominal pain	< 0.001	< 0.1	> 0.05
Heart burn	< 0.001	> 0.05	< 0.05
Acid regurgitation	< 0.001	> 0.05	< 0.05
Sucking sensation	< 0.001	< 0.001	< 0.001
Nausea/ vomiting	< 0.05	> 0.05	> 0.05
Borborygymi	< 0.001	< 0.01	> 0.05
Abdominal distension	< 0.01	> 0.05	> 0.05
Eructation	< 0.001	< 0.01	> 0.05
Increased flatus	< 0.001	< 0.001	< 0.001
Decreased stool	< 0.001	< 0.001	< 0.001
Loose stool	< 0.001	< 0.001	< 0.001
Hard stool	< 0.001	< 0.001	< 0.001
Urgency of defecation	< 0.001	< 0.001	< 0.001
Feeling of incomplete evacuation	< 0.001	<0.001	< 0.001
Total score GSRS	> 0.05	< 0.01	< 0.001

After the follow-up, among the individual symptoms of GSRS, the response was maximum in the symptoms of abdominal pain, heart burn, nausea/ vomiting, abdominal distension and loose stool.

The percentage of relief for the total score of GSRS was found as 27% during the first assessment (AT1) in the trial group and 24% in the control. During the second assessment (AT2), the percentage of relief was 46% in the trial and 38% in the control group. During the third assessment (AF), it was 58% in the trial group and 47% in the control.

AMLAPITTA RATING SCALE

The selected symptoms of Amlapitta in the rating scale ie. daha, chardi, soola, avipaka, amlodgara and the total score of all these, were considered for assessment of efficacy of the therapy, in this study .

1. DAHA

Table 150 Mean distribution of efficacy on Daha - both groups

	Trial	Control
BT	3.7 $\pm$ 0.48	3.57 $\pm$ 0.62
AT1	2.72 $\pm$ 0.51	2.66 $\pm$ 0.54
AT2	1.9 $\pm$ 0.63	1.99 $\pm$ 0.63
AF	1.3 $\pm$ 0.67	1.71 $\pm$ 0.70

In the trial group, the total score of the symptom of daha among the 100 included, showed a mean severity score of 3.7 $\pm$ 0.48 while in the control, it was 3.57 $\pm$ 0.62. After the treatment, during the first assessment (AT1), the mean score was reduced to 2.72 $\pm$ 0.51 in the study group, while in the control, it was reduced to

2.66 ± 0.54. In the second assessment (AT2), the mean score in the study group was 1.9 ± 0.63, while in the control, it was 1.99 ± 0.63. During the follow-up (AF), the mean score in the study group was 1.3 ± 0.67, while in control, it was 1.71 ± 0.70.

Table 151 F test - trial group – Daha

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	325.23	3	108.41	322.397	6.7E-106	2.627441
Within Groups	133.16	396	0.3363			
Total	458.39	399				

On comparing the mean differences in the score of daha, after each assessment in the trial group, using the ANOVA, the F value was found to be highly significant (P< 0.001) indicating that, there is significant difference in the mean scores during and after the treatment.

Table 152 F test - control group – Daha

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	205.3475	3	68.44917	175.4085	2.44E-72	2.627441
Within Groups	154.53	396	0.390227			
Total	359.8775	399				

On comparing the mean differences in the score of daha, after each assessment in the control group, using ANOVA, the F value was found to be highly significant (P< 0.001) indicating that, there is significant difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

Table -153 Tuckey Kramer test - trial group – Daha

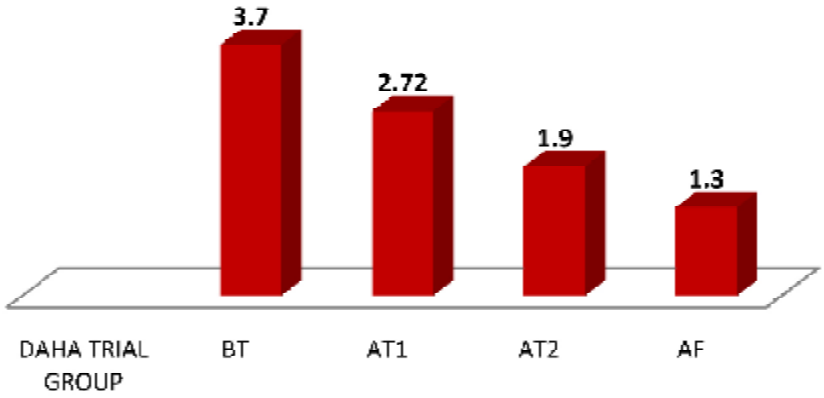
Comparison		MD	q	P Value
BT	AT1	0.98	24.565	<0.001
BT	AT2	1.8	45.119	<0.001
BT	AF	2.4	60.158	<0.001
AT1	AT2	0.82	20.554	<0.001
AT1	AF	1.42	35.594	<0.001
AT2	AF	0.6	15.04	<0.001

On comparing the various groups mean scores of assessment among themselves in the trial group for daha, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001) The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was also similarly significant (P< 0.001).

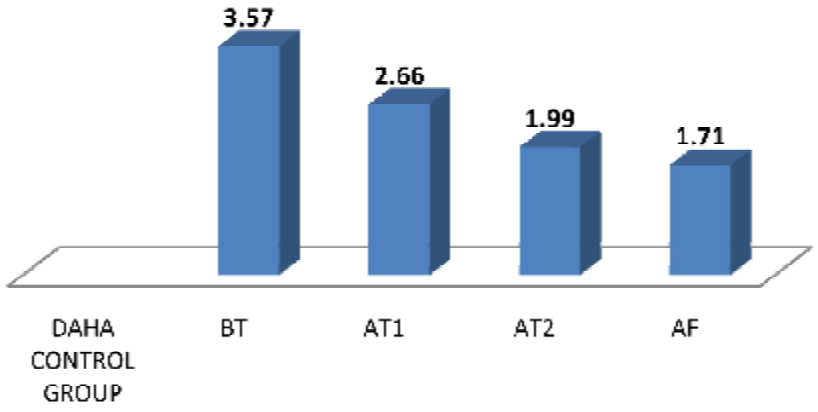
Multiple Comparison in between groups – control group

Table -154 Tuckey Kramer test - Control group – Daha

Comparison		MD	q	P Value
BT	AT1	0.91	21.361	<0.001
BT	AT2	1.58	37.088	<0.001
BT	AF	1.86	43.66	<0.001
AT1	AT2	0.67	15.727	<0.001
AT1	AF	0.95	22.3	<0.001
AT2	AF	0.28	6.573	<0.001



Graph 99: Mean distribution of efficacy of therapy on Daha – Trial group



Graph 100: Mean distribution of efficacy of therapy on Daha – Control group

On comparing the various groups mean scores of assessment among themselves in the control group for daha, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level ($P< 0.001$). The comparison between AT1 Vs AT2 and AF was also significant at the same level ($P< 0.001$). The comparison between AT2 and AF was also similarly significant ($P< 0.001$).

Comparison between two groups- unpaired t test – daha

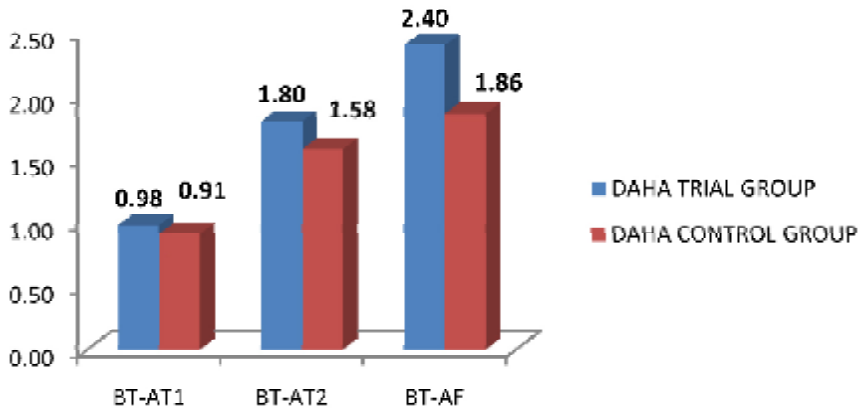
On comparing the efficacy of treatment in the score of daha, between the trial and control groups using unpaired t test, the mean reduction before treatment (BT) and on first assessment (AT1) was not significant ($P> 0.05$). The comparison

between mean reduction before treatment (BT) and on second assessment (AT2) was significant at 0.1% level. (P< 0.001). On comparing the groups between BT and on third assessment (AF), it was also not significant (P> 0.05).

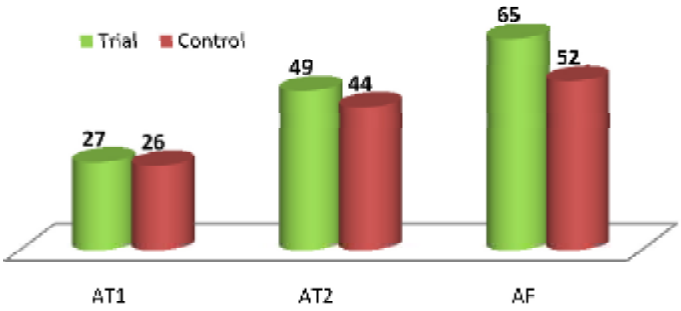
Table -155 Unpaired t test - between groups – Daha

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.98 ± 0.32	0.91 ± 0.35	1.47	> 0.05
BT-AT2	1.80 ± 0.55	1.58 ± 0.62	-3.369	< 0.001
BT - AF	2.40 ± 0.75	1.86 ± 0.78	0.369	>0.05

The percentage of relief for the score of daha was found as 27% during the first assessment (AT1) in the trial and 26% in the control group. During the second assessment (AT2), the percentage of relief was 49% in the trial group and 44% in the control. During the third assessment (AF), it was 65% in the trial and 52% in control group.



Graph 101 : Efficacy of therapy between groups- Daha



Graph 102: Percentage of relief between groups- Daha

2. CHARDI

Table 156 Mean distribution of efficacy on Chardi - both groups

	Trial	Control
BT	2.16 ± 1.36	2.09 ± 1.41
AT1	1.55 ± 1.04	1.58 ± 1.16
AT2	1.07 ± 0.84	1.35 ± 1.01
AF	0.89 ± 0.71	1.16 ± 0.87

In the trial group, the total score of the symptom of chardi among the 100 patients showed a mean severity score of 2.16 ± 1.36 while in the control, it was 2.09 ± 1.41. After the treatment, during the first assessment (AT1), the mean score was reduced to 1.55 ± 1.04 in the study group, while in the control, it was reduced to 1.58 ± 1.16. In the second assessment (AT2), the mean score in the study group was 1.07 ± 0.84, while in the control, it was 1.35 ± 1.01. During the follow-up (AF), the mean score in study group was 0.89 ± 0.71, while in the control, it was 1.16 ± 0.87.

Table 157 F test – trial group – Chardi

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	96.7875	3	32.2625	31.1237	4.38 E-18	2.6274
Within Groups	410.49	396	1.0366			
Total	**507.2775**	**399**				

On comparing the mean differences in the score of chardi, after each assessment in the trial group, using the ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that, there is significant difference in the mean scores during and after the treatment.

Table 158 F test – control group – Chardi

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	48.45	3	16.15	12.6707	6.34E-08	2.627441
Within Groups	504.74	396	1.2746			
Total	**553.19**	**399**				

On comparing the mean differences in the score of chardi, after each assessment in the control group, using the ANOVA, it was found to be highly significant ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

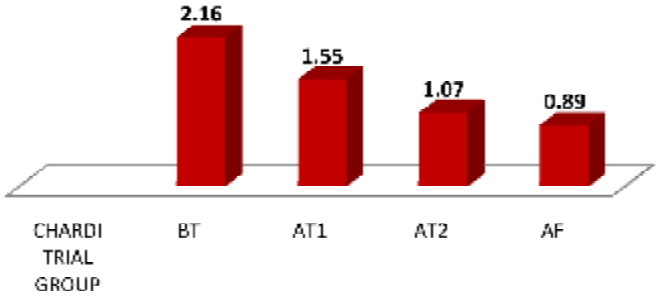
Multiple Comparison in between groups – trial group

On comparing the various groups mean scores of assessment among themselves in the trial group for chardi, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level ($P < 0.001$) The comparison between AT1 Vs AT2

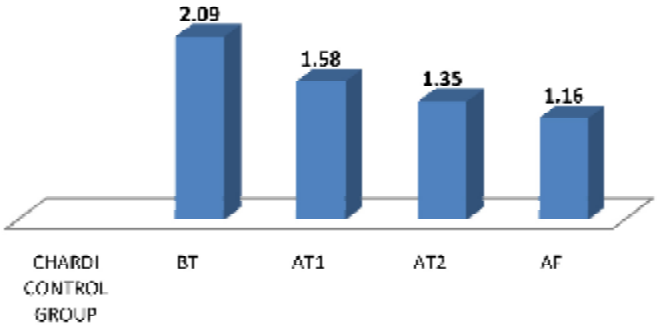
and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was not significant. (P> 0.05)

Table -159 Tuckey Kramer test - trial group – Chardi

Comparison		MD	q	P Value
BT	AT1	0.61	11.756	<0.001
BT	AT2	1.09	21.007	<0.001
BT	AF	1.27	24.476	<0.001
AT1	AT2	0.48	9.251	<0.001
AT1	AF	0.66	12.72	<0.001
AT2	AF	0.18	3.469	>0.05



Graph 103: Efficacy of therapy on Chardi – Trial group



Graph 104: Efficacy of therapy on Chardi – Control group

Multiple Comparison in between groups – control group

Table -160 Tuckey Kramer test - Control group – Chardi

Comparison		MD	q	P Value
BT	AT1	0.51	12.183	<0.001
BT	AT2	0.74	17.677	<0.001
BT	AF	0.93	22.215	<0.001
AT1	AT2	0.23	5.494	<0.001
AT1	AF	0.42	10.033	<0.001
AT2	AF	0.19	4.539	<0.01

On comparing the various groups mean scores of assessment among themselves in the control group in the symptom of chardi, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001) The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was also significant, but at 1% level. (P< 0.01)

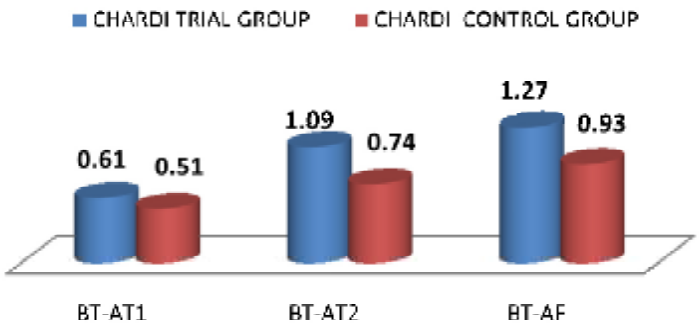
Comparison between two groups- unpaired t test – chardi

Table -161 Unpaired t test - between groups – Chardi

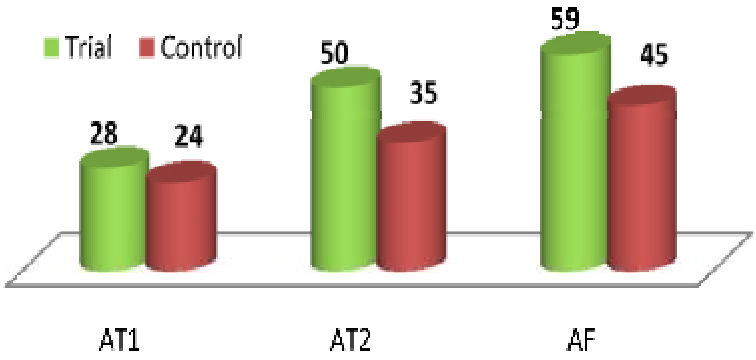
Comparison	M D Trial	M D Control	t	P
BT - AT1	0.61 $\pm$ 0.53	0.51 $\pm$ 0.54	- 5.281	< 0.001
BT-AT2	1.09 $\pm$ 0.88	0.74 $\pm$ 0.66	-1.365	>0.05
BT - AF	1.27 $\pm$ 1.01	0.93 $\pm$ 0.77	- 1.257	>0.05

On comparing the efficacy of treatment in the score of chardi, between the trial and control groups using unpaired t test, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant (P<0.001). The comparison

between score before treatment (BT) and on second assessment (AT2) was not significant. (P> 0.05) On comparing the two groups between BT and on third assessment (AF), it was also not significant. (P> 0.05).



Graph 105: Efficacy of therapy between groups- Chardi



Graph 106: Percentage of relief between groups- Chardi

The percentage of relief in chardi was found as 28% during the first assessment (AT1) in the trial group and 24% in the control. During the second assessment (AT2), the percentage of relief was 50% in the trial group and 35% in the control group. During the third assessment (AF), it was 59 % in trial group and 45 % in the control group.

3. SOOLA

Table 162 Mean distribution of efficacy on Soola - both groups

	Trial	Control
BT	3.46 ± 0.76	3.63 ± 0.51
AT1	2.54 ± 0.69	2.65 ± 0.67
AT2	1.92 ± 0.60	2.2 ± 0.64
AF	1.65 ± 0.64	1.74 ± 0.61

In the trial group, the total score of the symptom of soola among 100 subjects showed a mean severity score of 3.46 ± 0.76 while in the control group, it was 3.63 ± 0.51. After the treatment, during the first assessment (AT1), the mean score was reduced to 2.54 ± 0.69 in the study group, while in the control, it was reduced to 2.65 ± 0.67. In the second assessment (AT2), the mean score in the study group was 1.92 ± 0.60, while in the control group, it was 2.2 ± 0.64. During the follow-up (AF), the mean score in the study group was 1.65 ± 0.64, while in the control, it was 1.74 ± 0.61.

Table 163 F test – trial group – Soola

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	193.5875	3	64.5292	142.13	1.65 E-62	2.6274
Within Groups	179.79	396	0.45402			
Total	373.3775	399				

On comparing the mean differences in the score of soola, after each assessment in the trial group, using ANOVA, the F value was found to be highly significant ($P<0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

Table 164 F test – control group – Soola

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	195.49	3	65.16333	175.1845	2.81E-72	2.627441
Within Groups	147.3	396	0.37197			
Total	342.79	399				

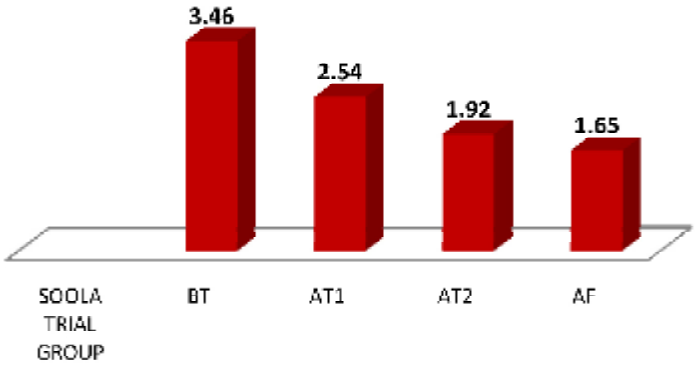
On comparing the mean differences in the score of soola, after each assessment in the control group, using the Analysis of variance, it was found to be highly significant ($P< 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

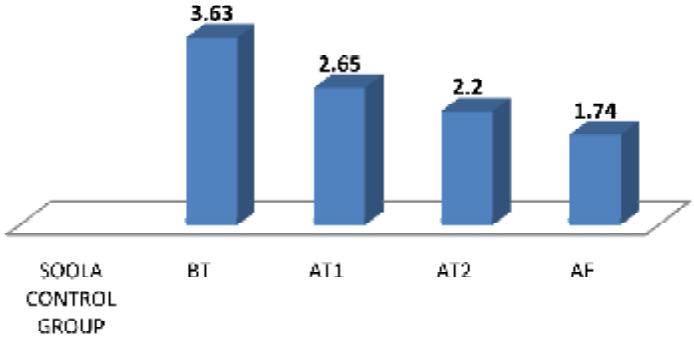
Table -165 Tuckey Kramer test - trial group – Soola

Comparison		MD	q	P value
BT	AT1	0.92	20.701	<0.001
BT	AT2	1.54	34.651	<0.001
BT	AF	1.81	40.726	<0.001
AT1	AT2	0.62	13.95	<0.001
AT1	AF	0.89	20.026	<0.001
AT2	AF	0.27	6.075	<0.001

On comparing the various groups mean scores of assessment among themselves, in the trial group for soola, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level ($P< 0.001$) The comparison between AT1 Vs AT2 and AF was also significant, at the same level ($P< 0.001$). The comparison between AT2 and AF was also significant at the same level ($P< 0.001$).



Graph 107: Efficacy of therapy on Soola – Trial group



Graph 108: Efficacy of therapy on Soola – Control group

Multiple Comparison in between groups – control group

Table -166 Tuckey Kramer test – Control group – Soola

Comparison		MD	q	P value
BT	AT1	0.98	21.622	<0.001
BT	AT2	1.43	31.551	<0.001
BT	AF	1.89	41.7	<0.001
AT1	AT2	0.45	9.929	<0.001
AT1	AF	0.91	20.078	<0.001
AT2	AF	0.46	10.149	<0.001

On comparing the various groups mean scores of assessment among themselves in the control group for soola, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001) The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was also significant at the same level (P< 0.001).

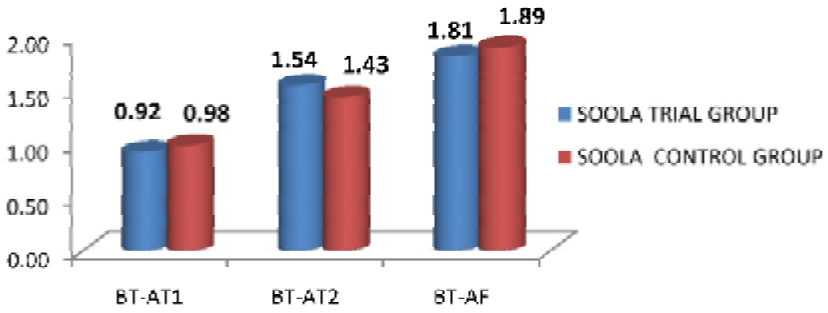
Comparison between two groups- unpaired t test – soola

On comparing the efficacy of treatment in the score of soola, between the trial and control groups using unpaired t test, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant (P< 0.001). The comparison between mean reduction before treatment (BT) and on second assessment (AT2) was also similarly significant (P< 0.001). On comparing the groups between BT and on third assessment (AF), it was also highly significant. (P< 0.001).

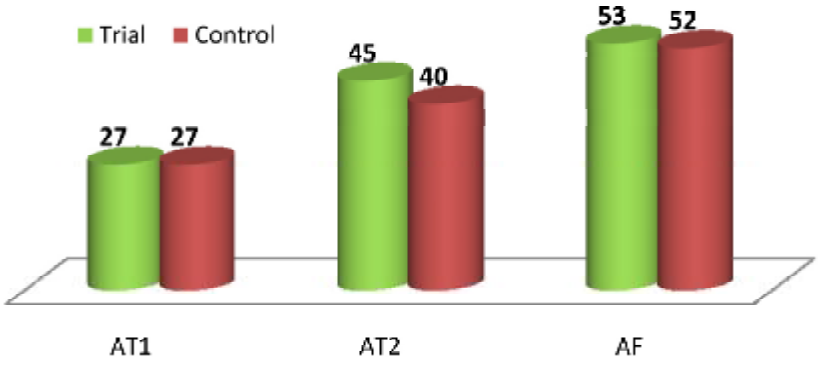
Table -167 Unpaired t test - between groups – Soola

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.92 ± 0.54	0.98 ± 0.59	- 6.896	< 0.001
BT-AT2	1.54 ± 0.72	1.43 ± 0.66	- 4.016	< 0.001
BT - AF	1.81 ± 0.76	1.89 ± 0.68	- 5.681	< 0.001

The percentage of relief for the score of soola was observed as 27% during the first assessment (AT1) in the trial and 27% in the control group. During the second assessment (AT2), the percentage of relief was 45% in the trial group and 40% in the control. During the third assessment (AF), it was 53% in the trial group and 52% in the control group.



Graph 109: Efficacy of therapy between groups- Soola



Graph 110: Percentage of relief between groups- Soola

4. AVIPAKA

Table 168 Mean distribution of efficacy on Avipaka - both groups

	Trial	Control
BT	1.91 ± 1.24	2.03 ± 1.30
AT1	1.55 ± 1.09	1.68 ± 1.14
AT2	1.10 ± 0.85	1.48 ± 1.01
AF	1.01 ± 0.76	1.24 ± 0.92

In the trial group, the total score of the symptom avipaka among the included, showed a mean severity score of 1.91 ± 1.24 while in the control, it was 2.03 ± 1.30. With the treatment, during the first assessment (AT1), the mean score

was reduced to 1.55 ± 1.09 in study group, while in control it was reduced to 1.68 ± 1.14. In the second assessment (AT2), the mean score in study group was 1.10 ± 0.85, while in the control group, it was 1.48 ± 1.01. During the follow-up (AF), the mean score in study group was 1.01 ± 0.76, while in the control, it was 1.24 ± 0.92.

Table 169 F test – trial group – Avipaka

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	52.4475	3	17.4825	17.44154	1.18 E-10	2.6274
Within Groups	396.93	396	1.00235			
Total	449.3775	399				

On comparing the mean differences in the score of avipaka, after each assessment in the trial group, using ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

Table 170 F test – control group – Avipaka

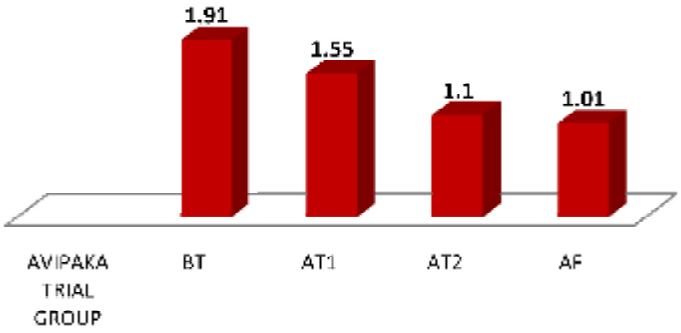
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	33.5075	3	11.16917	9.217059	6.6E-06	2.627441
Within Groups	479.87	396	1.211793			
Total	513.3775	399				

On comparing the mean differences in the score of avipaka, after each assessment in the control group with ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

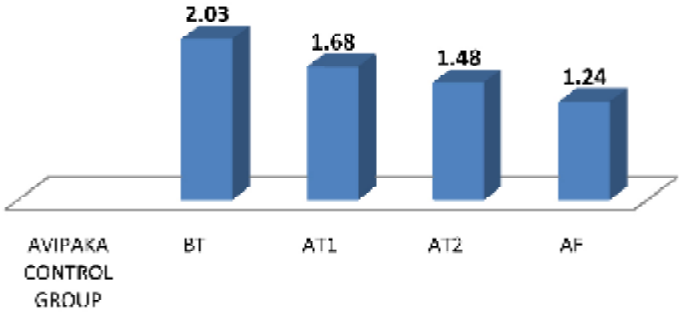
Multiple Comparison in between groups – trial group

Table -171 Tuckey Kramer test - trial group – Avipaka

Comparison		MD	q	P value
BT	AT1	0.36	8.16	<0.001
BT	AT2	0.81	18.361	<0.001
BT	AF	0.9	20.401	<0.001
AT1	AT2	0.45	10.2	<0.001
AT1	AF	0.54	12.24	<0.001
AT2	AF	0.09	2.04	>0.05



Graph 111: Efficacy of therapy on Avipaka – Trial group



Graph 112: Efficacy of therapy on Avipaka – Control group

On comparing the various groups mean scores of assessment among themselves, in the trial group for avipaka, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001). The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was not significant.

Multiple Comparison in between groups – control group

On comparing various groups mean scores of assessment among themselves in control for avipaka, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level. The comparison between AT1 Vs AT2 was significant at 1% level. The comparison between AT1 and AF was also significant at 0.1% level. The comparison between AT2 and AF was also significant at 0.1% level.

Table -172 Tuckey Kramer test – Control group – Avipaka

Comparison		MD	q	P value
BT	AT1	0.35	8.152	<0.001
BT	AT2	0.55	12.811	<0.001
BT	AF	0.79	18.401	<0.001
AT1	AT2	0.2	4.658	<0.01
AT1	AF	0.44	10.249	<0.001
AT2	AF	0.24	5.59	<0.001

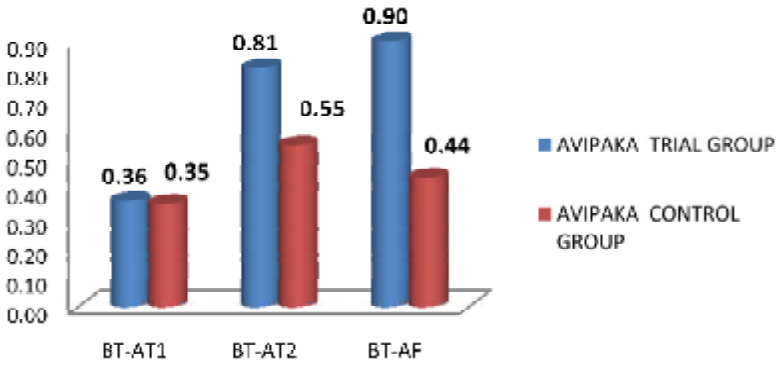
Comparison between two groups- unpaired t test – avipaka

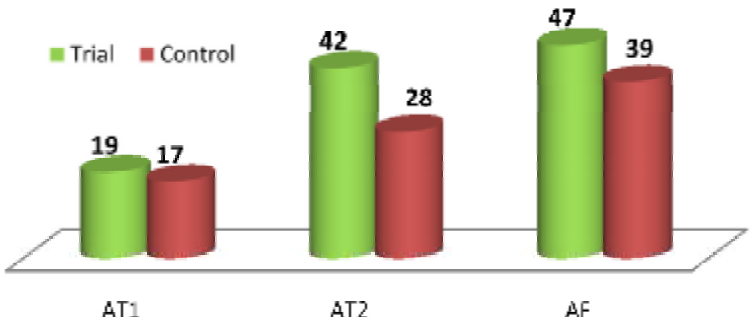
Table -173 Unpaired t test - between groups – Avipaka

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.36 ± 0.50	0.35 ± 0.35	- 7.052	< 0.001
BT - AT2	0.81 ± 0.75	0.55 ± 0.55	- 2.752	< 0.05
BT - AF	0.90 ± 0.77	0.44 ± 0.44	- 0.387	> 0.05

On comparing the efficacy of the intervention in the score of avipaka, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant (P< 0.001). The comparison between mean reduction before treatment (BT) and on second assessment (AT2) was also significant, but at 5% level (P< 0.05). On comparing the two groups between BT and on third assessment (AF), it was not significant. (P> 0.05)

Graph 113: Efficacy of therapy between groups- Avipaka





Graph 114: Percentage of relief between groups- Avipaka

The percentage of relief for the score of avipaka was found as 19% during first assessment (AT1) in the trial and 17% in control group. During the second assessment (AT2), the percentage of relief was 42% in trial group and 28% in control. During the third assessment (AF), it was 47% in the trial group and 39% in the control.

5. AMLODGARA

Table 174 Mean distribution of efficacy on Amlodgara - both groups

	Trial	Control
BT	3.1 ± 0.92	3.07 ± 1.13
AT1	2.18 ± 0.80	2.05 ± 0.87
AT2	1.55 ± 0.74	1.59 ± 0.75
AF	1.21 ± 0.67	1.32 ± 0.68

In the trial group, the total score of the symptom amlodgara showed a mean severity score of 3.1 ± 0.92, while in the control group it was 3.07 ± 1.13. After the treatment, during the first assessment (AT1), the mean score was reduced to 2.18 ± 0.80 in the study, while in the control group it was reduced to 2.05 ± 0.87. In the second assessment (AT2), the mean score in the study group was 1.55 ± 0.74, while in the control, it was 1.59 ± 0.75. During the follow-up (AF), the mean score in the study group was 1.21 ± 0.67, while in the control, it was 1.32 ± 0.68.

Table 175 F test – trial group – Amlodgara

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	206.86	3	68.95333	111.4056	2.59E-52	2.627441
Within Groups	245.1	396	0.618939			
Total	451.96	399				

On comparing the mean differences in the score of amlodgara, after each assessment in the trial group, using ANOVA, the F value was found to be highly significant ($P < 0.001$) indicating that, there is significant difference in the mean scores during and after the treatment.

Table 176 F test – control group –Aamlodgara

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	177.7675	3	59.25583	77.38963	2.05E-39	2.627441
Within Groups	303.21	396	0.765682			
Total	480.9775	399				

On comparing the mean differences in the score of amlodgara, after each assessment in the control group, using ANOVA, the F value was found to be highly significant (P< 0.001) indicating that, there is significant difference in the mean scores during and after the treatment.

Multiple Comparison in between groups – trial group

Table -177 Tuckey Kramer test - trial group – Amlodgara

Comparison		MD	q	P value
BT	AT1	0.92	19.422	<0.001
BT	AT2	1.55	32.722	<0.001
BT	AF	1.89	39.9	<0.001
AT1	AT2	0.63	13.3	<0.001
AT1	AF	0.97	20.478	<0.001
AT2	AF	0.34	7.178	<0.001

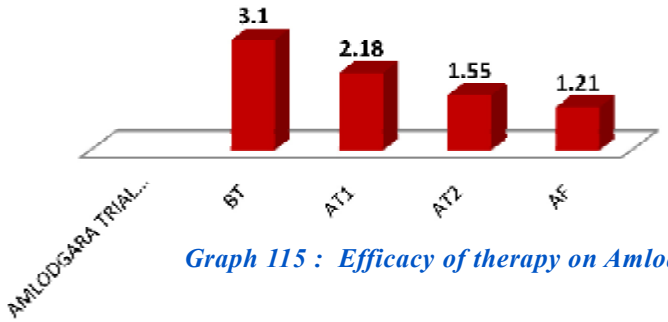
On comparing the various groups mean scores of assessment among themselves in the trial group for the symptom of amlodgara, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001) The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was also similarly significant.

Multiple Comparison in between groups – control group

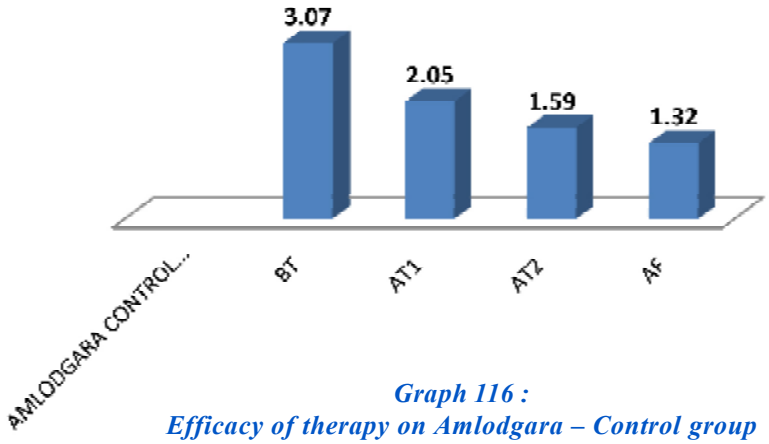
Table -178 Tuckey Kramer test - Control group – Amlodgara

Comparison		MD	q	P value
BT	AT1	1.02	20.233	<0.001
BT	AT2	1.48	29.357	<0.001
BT	AF	1.75	34.713	<0.001
AT1	AT2	0.46	9.125	<0.001
AT1	AF	0.73	14.48	<0.001
AT2	AF	0.27	5.356	<0.01

On comparing the various groups mean scores of assessment among themselves in the control group for amlodgara, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001). The comparison between AT1



Graph 115 : Efficacy of therapy on Amlodgara – Trial group



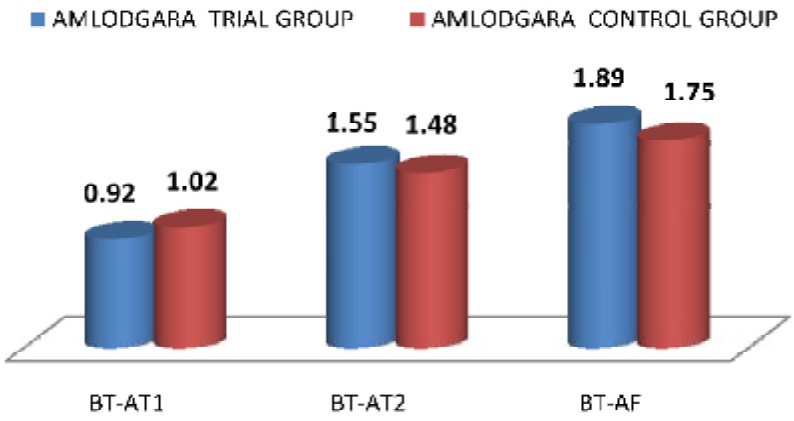
Graph 116 :
Efficacy of therapy on Amlodgara – Control group

Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was significant at 1% level (P< 0.01).

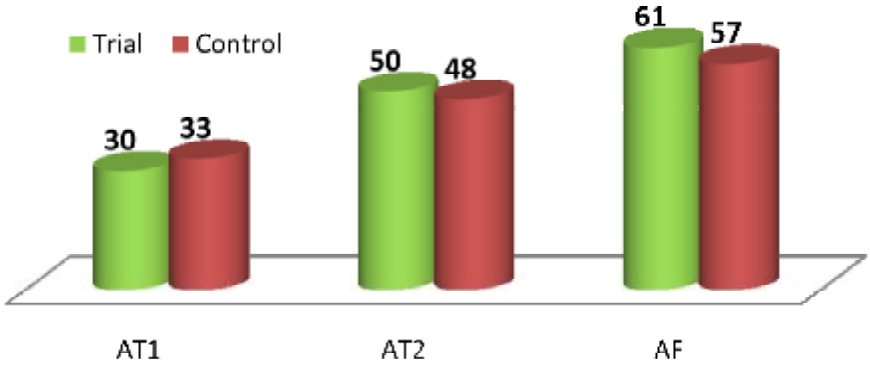
Comparison between groups- unpaired t test – Amlodgara

Table -179 Unpaired t test - between groups – Amlodgara

Comparison	M D Trial	M D Control	t	P
BT - AT1	0.92 ± 0.60	1.02 ± 0.70	- 6.54	< 0.001
BT - AT2	1.55 ± 0.80	1.48 ± 0.80	- 3.815	< 0.001
BT - AF	1.89 ± 0.84	1.75 ± 0.87	- 2.719	< 0.01



Graph 117: Mean distribution of the efficacy of therapy between groups - Amlodgara



Graph 118: Percentage of relief between groups- Amlodgara

On comparing the efficacy of treatment in the score of amlodgara, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was highly significant (P< 0.001). The comparison between mean reduction before treatment (BT) and on second assessment (AT2) was also significant the same level (P< 0.001). On comparing the groups between BT and on third assessment (AF), it was also significant, but at 1% level. (P< 0.01).

The percentage of relief for the score of amlodgara was found as 30% during the first assessment (AT1) in the trial and 33% in the control. During the second assessment (AT2), the percentage of relief was 50% in trial group and 48 % in the control. During third assessment (AF), it was 61% in trial and 57% in the control.

6. AMLAPITTA TOTAL SCORE

Table 180 Mean distribution of efficacy of therapy on Total Amlapitta score - both groups

	Trial	Control
BT	14.35 ± 2.42	14.39 ± 2.46
AT1	10.57 ± 2.10	10.52 ± 2.12
AT2	7.54 ± 1.82	8.48 ± 2.13
AF	6 ± 1.93	7.17 ± 1.76

In the trial group, the total score of Amlapitta among 100 subjects showed a mean severity score of 14.35 ± 2.42 while in the control group, it was 14.39 ± 2.46. After the treatment, during the first assessment (AT1), the mean score was reduced to 10.57 ± 2.10 in the study group, while in the control, it was reduced to 10.52 ± 2.12. In the second assessment (AT2), the mean score in the study group was 7.54 ± 1.82,

while in the control group, it was 8.48 $\pm$ 2.13. After the follow-up (AF), the mean score in the study group was 6 $\pm$ 1.93, while in the control, it was 7.17 $\pm$ 1.76.

Table 181 F test – trial group – Amlapitta total score

Source of Variation	SS	df	MS	F	P value	F crit
Between Groups	3884.21	3	1294.736	261.0436	1.94E-93	2.627441
Within Groups	1964.1	396	4.959849			
Total	**5848.31**	**399**				

On comparing the mean differences in the total score of Amlapitta, after each assessment in the trial group, using ANOVA, the F value was found to be highly significant ($P< 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

Table 182 F test – control group – Amlapitta total score

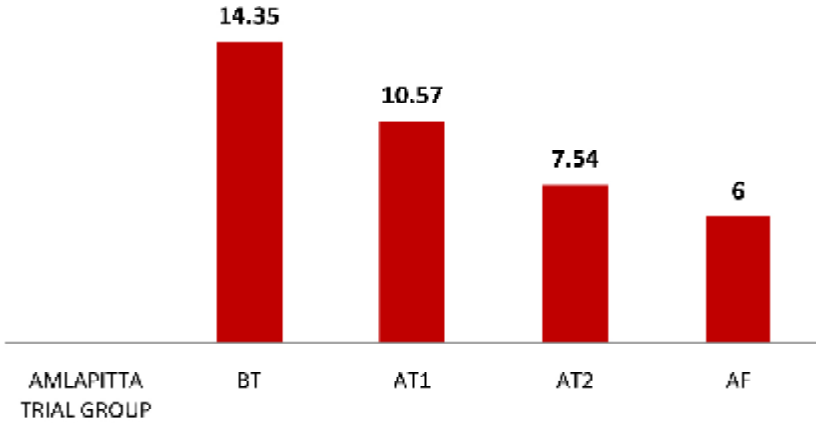
Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	2978.34	3	992.78	218.6764	1.21 E-83	2.627441
Within Groups	1797.82	396	4.5395			
Total	**4776.16**	**399**				

On comparing the mean differences in the total score of Amlapitta, after each assessment in the control group, using ANOVA, the F value was found to be highly significant ($P< 0.001$) indicating that there is significant difference in the mean scores during and after the treatment.

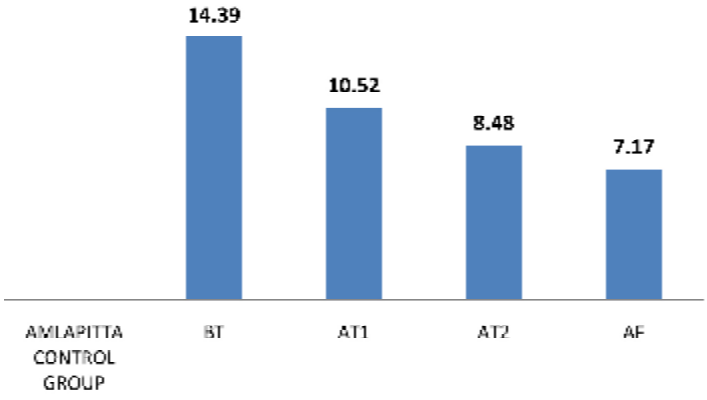
Multiple Comparison in between groups – trial group

Table -183 Tuckey Kramer test - trial group – Amlapitta total score

Comparison		MD	q	P Value
BT	AT1	3.78	30.56	<0.001
BT	AT2	6.81	55.057	<0.001
BT	AF	8.35	67.507	<0.001
AT1	AT2	3.03	24.497	<0.001
AT1	AF	4.57	36.947	<0.001
AT2	AF	1.54	12.45	<0.001



Graph 119: Efficacy of therapy on Total score Amlapitta – Trial group

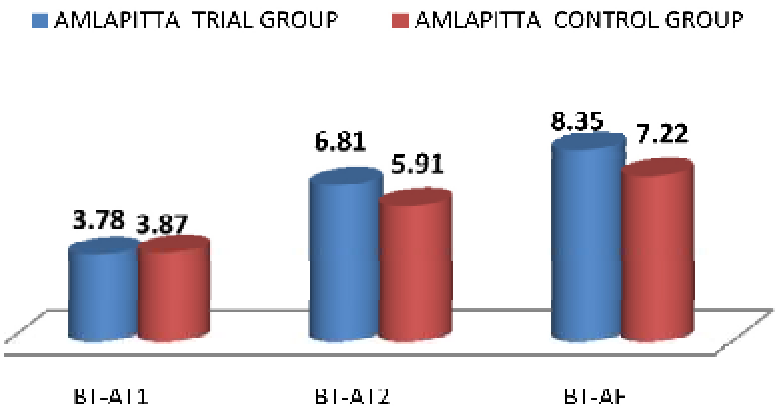


Graph 120: Efficacy of therapy on Total score Amlapitta – Control group

On comparing the various groups mean scores of assessment among themselves in the trial group for the total score of Amlapitta, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001) The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was also similarly significant.

Multiple Comparison in between groups – control group

On comparing the various groups mean scores of assessment among themselves in the control group for the total score of Amlapitta, the comparison between BT Vs AT1, AT2 and AF was significant at 0.1 % level (P< 0.001) The comparison between AT1 Vs AT2 and AF was also significant at the same level (P< 0.001). The comparison between AT2 and AF was also similarly significant.



Graph 121: Efficacy of therapy between groups- Amlapitta total score

Graph 122 : Percentage of relief between groups- Amlapitta total score

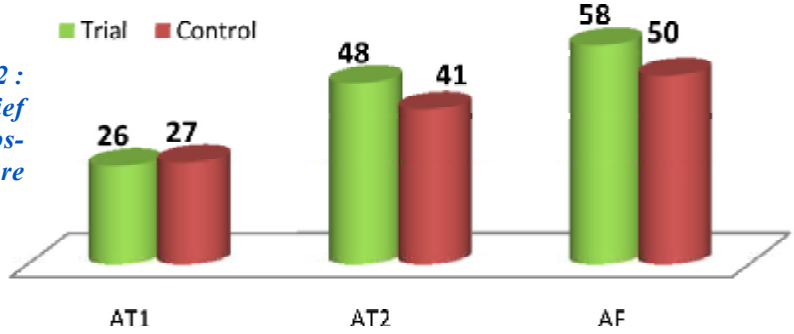


Table -184 Tuckey Kramer test - Control group – Amlapitta total score

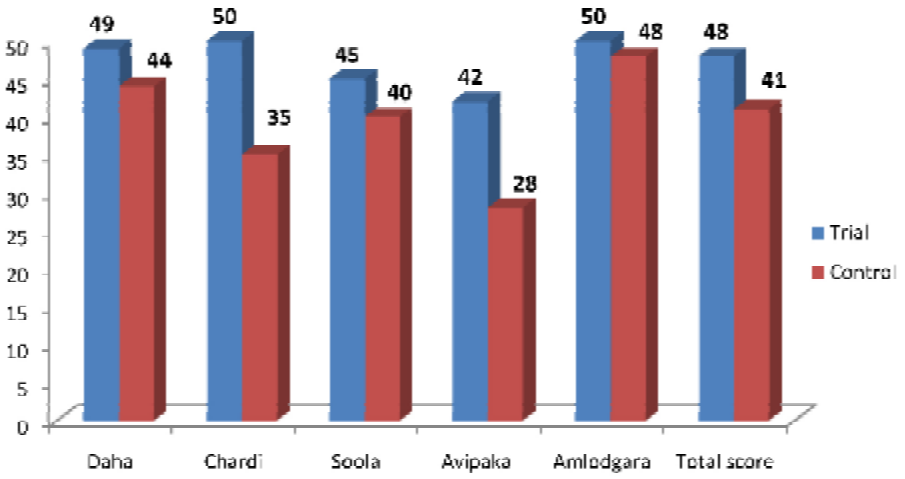
Comparison		MD	q	P Value
BT	AT1	3.87	29.733	<0.001
BT	AT2	5.91	45.406	<0.001
BT	AF	7.22	55.471	<0.001
AT1	AT2	2.04	15.673	<0.001
AT1	AF	3.35	25.738	<0.001
AT2	AF	1.31	10.065	<0.001

Comparison between groups- unpaired t test – Amlapitta total score

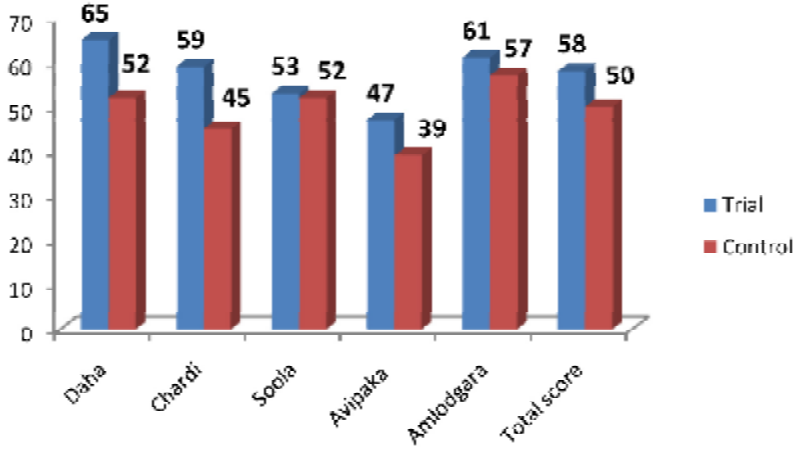
Table -185 Unpaired t test - between groups – Amlapitta total score

Comparison	M D Trial	M D Control	t	P
BT - AT1	3.78 $\pm$ 1.40	3.87 $\pm$ 1.80	- 2.58	< 0.01
BT-AT2	6.81 $\pm$ 2.15	5.91 $\pm$ 2.07	1.338	> 0.05
BT - AF	8.35 $\pm$ 2.41	7.22 $\pm$ 2.02	2.006	< 0.05

On comparing the efficacy of treatment in the total score of Amlapitta, between the trial and control groups, the mean reduction before treatment (BT) and on first assessment (AT1) was significant at 1% level (P< 0.01). The comparison between mean score before treatment (BT) and on second assessment (AT2) was not significant (P>0.05). On comparing the two groups between BT and on third assessment (AF), it was significant, but only at 5% level. (P< 0.05).



Graph 123: Percentage of relief on individual symptoms- After trial



Graph 124: Percentage of relief on individual symptoms- On follow-up

Efficacy of the therapy on individual score of Amlapitta

The level of significance of the therapy of the individual symptoms, in the Amlapitta rating scale, in both the groups were assessed, after the medication and also the follow up.

Table- 186 Level of significance of efficacy on Amlapitta rating scale

Symptom	Significance		
	AT1	AT2	AF
Daha	> 0.05	< 0.001	> 0.05
Chardi	< 0.001	> 0.05	> 0.05
Soola	< 0.001	< 0.001	< 0.001
Avipaka	< 0.001	< 0.05	> 0.05
Amlodgara	< 0.001	< 0.001	< 0.01
Total score	< 0.01	> 0.05	< 0.05

After the completion of the medication (AT2), the maximum response was observed in the symptoms of daha, chardi and amlodgara.

After the follow up period, it was observed that the major respondents were in daha and amlodgara, followed by chardi.

The percentage of relief in the total score of Amlapitta was found as 26% during the first assessment (AT1) in the trial group and 27% in the control. During the second assessment (AT2), the percentage of relief was 48% in the trial and 41% in the control. During the third assessment (AF), it was 58% in the trial group and 50% in the control group.

TESTING THE ASSOCIATION BETWEEN VARIOUS FACTORS

The association of various factors in the contribution of the disease Functional Dyspepsia as well as the Amlapitta was evaluated by using the Chi square test. For the same, the total score obtained in the GSRS rating scale was used as a standard, for studying the association. The median value of GSRS scale scores obtained in both the groups was calculated as 32. This was kept as a standard for testing the association as a cutoff value. The various contributory factors thought as relevant in the pathogenesis were tested for significance using the χ^2 test.

1. Use of excess snigdha ahara and GSRS score

Table 187 Distribution of subjects as per snigdha ahara and GSRS

Snigdha ahara	GSRS >32	GSRS<32	Total
Positive	58	26	84
Negative	74	42	116
Total	**132**	**68**	**200**

On testing the association between the excessive use of snigdha ahara in the diet, it was observed that the Odds ratio was 1.3 which was not statistically significant as the χ^2 value obtained was 0.5077 (P>0.05)

Table 188 χ^2 table on snigdha ahara and GSRS score

Observed	Expected	χ^2	P value
58	55.44	0.5077	0.4387
26	28.56		
74	76.56		
42	39.44		

2. Use of excess ushna ahara and GSRS score

Table 189 Distribution of the subjects as per ushna ahara and GSRS

Ushna ahara	GSRS >32	GSRS<32	TOTAL
Positive	**122**	**59**	181
Negative	**10**	**9**	19
Total	132	68	200

Table 190 χ^2 table on ushna ahara and GSRS score

Observed	Expected	χ^2	P value
122	119.46	0.6579	0.1959
59	61.54		
10	12.54		
9	6.46		

The Odds ratio calculated was 1.9 in this regard. On testing the association between the excessive use of ushna ahara in the diet, it was observed that the χ^2 value obtained was 0.6579 and it was not statistically significant (P> 0.05)

3. Excessive use of rasas and the GSRS score

a. Madhura Rasa

Table 191 Distribution of the subjects as per madhura rasa and GSRS

Madhura rasa	GSRS >32	GSRS<32	Total
Positive	**29**	**26**	55
Negative	**103**	**42**	145
Total	132	68	200

Table 192 χ^2 table on madhura rasa and GSRS score

Observed	Expected	χ^2	P value
29	36.3	0.9035	0.014
26	18.7		
103	95.7		
42	49.3		

On testing the association between the excessive use of madhura rasa in the diet, it was observed that the χ^2 value obtained was 0.9035 and it was statistically significant at 5% level ($P<0.05$). The calculated Odds ratio was 0.5 which was not supportive in this regard.

b. Amla Rasa

Table 193 Distribution of the subjects as per amla rasa and GSRS

Amla rasa	GSRS >32	GSRS<32	Total
Positive	**126**	**60**	186
Negative	**6**	**8**	14
Total	132	68	200

Table 194 χ^2 table on amla rasa and GSRS score

Observed	Expected	χ^2	P value
126	122.76	0.8096	0.0498
60	63.24		
6	9.24		
8	4.76		

Here the Odds ratio obtained was 2.8 which show much association between the use of amla rasa and GSRS. On testing the association for statistical significance, it was observed that the χ^2 value obtained was 0.8096 and it was significant at 5% level (P<0.05)

c. Lavana Rasa

Table 195 Distribution of the subjects as per lavana rasa and GSRS

Lavana	GSRS >32	GSRS<32	Total
Positive	**110**	**46**	156
Negative	**22**	**22**	44
Total	132	68	200

Table 196 χ^2 table on lavana rasa and GSRS score

Observed	Expected	χ^2	P value
110	102.96	0.9157	0.011
46	53.04		
22	29.04		
22	14.96		

The calculated Odds ratio was 2.4 in the case of use of excessive lavana rasa and GSRS score. On testing association between the two, it was observed that the χ^2 value obtained was 0.9157 and it was statistically significant at 5% level (P<0.05)

d. Katu Rasa

Table 197 Distribution of the subjects as per katu rasa and GSRS

Katu rasa	GSRS >32	GSRS<32	Total
Positive	**126**	**59**	185
Negative	**6**	**9**	15
Total	**132**	**68**	**200**

Table 198 χ^2 table on katu rasa and GSRS score

Observed	Expected	χ^2	P value
126	122.1	0.8692	0.027
59	62.9		
6	9.9		
9	5.1		

The Odds ratio was calculated as 3.2 in the matter of excess katu rasa and GSRS score. On testing the association of the same, it was observed that the χ^2 value obtained was 0.9157 and it was statistically significant at 5% level ($P<0.05$).

e. Tikta rasa

Table 199 Distribution of the subjects as per tikta rasa and GSRS

Tikta rasa	GSRS >32	GSRS<32	Total
Positive	**7**	**5**	12
Negative	**125**	**63**	188
Total	132	68	200

Table 200 χ^2 table on tikta rasa and GSRS score

Observed	Expected	χ^2	P value
7	7.92	0.4530	0.563
5	4.08		
125	124.08		
63	63.92		

On testing the association between the excessive use of tikta rasa in the diet, it was observed that the Odds ratio was 0.7 and also the χ^2 value obtained was 0.4530 and it was statistically not significant (P>0.05)

f. Kashaya Rasa

Table 201 Distribution of the subjects as per kashaya rasa and GSRS

Kashaya rasa	GSRS >32	GSRS<32	Total
Positive	**2**	**1**	3
Negative	**130**	**67**	197
Total	132	68	200

Table 202 χ^2 table on kashaya rasa and GSRS score

Observed	Expected	χ^2	P value
2	1.98	0.3220	0.98
1	1.02		
130	130.02		
67	66.98		

On testing the association between the use of excess kashaya rasa in the

diet, it was observed that the χ^2 value obtained was 0.3220 and it was statistically not significant (P> 0.05). The Odds ratio obtained was 1.03 supportive for the same.

4. Use of refrigerated food and GSRS score

On testing the association between the use of refrigerated food in excess and GSRS, it was observed that the χ^2 value obtained was 0.5680 and it was statistically not significant (P> 0.05). The Odds ratio calculated was 0.7 and also not supportive.

Table 203 Distribution of the subjects as per refrigerated food and GSRS

Refrigeration	GSRS >32	GSRS<32	Total
Positive	34	22	56
Negative	98	46	144
Total	132	68	200

Table 204 χ^2 table on refrigerated food and GSRS score

Observed	Expected	χ^2	P value
34	36.96	0.5680	0.325
22	19.04		
98	95.04		
46	48.96		

5. Skipping of meals frequently and GSRS score

On testing the association between the regular habit of skipping meals, it was observed that the χ^2 value obtained was 0.7310 and it was statistically not significant. The calculated Odds ratio was 0.4 which seems indicative of the same.

Table 205 Distribution of subjects as per skipping of meals and GSRS

Skipping meals	GSRS >32	GSRS<32	Total
Positive	**6**	**7**	13
Negative	**126**	**61**	187
Total	**132**	**68**	**200**

Table 206 χ^2 table on skipping of meals and GSRS score

Observed	Expected	χ^2	P value
6	8.58	0.7310	0.118
7	4.42		
126	123.42		
61	63.58		

6. Atmosphere of food intake and GSRS score

Table 207 Subjects as per atmosphere of food intake and GSRS

Food intake	GSRS >32	GSRS<32	Total
Positive	**26**	**21**	47
Negative	**106**	**47**	153
Total	132	68	200

Table 208 χ^2 table on atmosphere of food intake and GSRS score

Observed	Expected	χ^2	P value
26	31.02	0.7810	0.07
21	15.98		
106	100.98		
47	52.02		

On testing the association between the habit of having food in unsound atmosphere and GSRS score, it was observed that the χ^2 value obtained was 0.7810 and it was statistically not significant (P> 0.05). The Odds ratio was 0.54 suggestive of the same itself.

7. Intake after appetite and GSRS score

Table 209 Subjects according to intake after appetite and GSRS

Intake after appetite	GSRS >32	GSRS<32	Total
Positive	117	54	171
Negative	15	14	29
Total	**132**	**68**	**200**

Table 210 χ^2 table on intake after appetite and the GSRS score

Observed	Expected	χ^2	P value
117	112.86	0.7780	0.07
54	58.14		
15	19.14		
14	9.86		

Here the attained Odds ratio was 2.1 pointing to a positive association. On testing the association between the habit of intake after having appetite and GSRS, it was observed that the χ^2 value obtained was 0.7810 and it was not significant.

8. Irregular timing of meals and GSRS score

On testing the association between the timing of meals and GSRS score, it was observed that the χ^2 value obtained was 0.9790 and it was statistically highly significant (P< 0.001). The Odds ratio was not supportive, as it was only 0.25.

Table 211 Subjects according to irregular timing of meals and GSRS

Timing of meals	GSRS >32	GSRS<32	Total
Positive	10	17	27
Negative	122	51	173
Total	**132**	**68**	**200**

Table 212 χ^2 table on irregular timing of meals and the GSRS score

Observed	Expected	χ^2	P value
10	17.82	0.9790	0.0006
17	9.18		
122	114.18		
51	58.82		

9. Irregular sleep and GSRS score

The calculated Odds ratio was 1.73 indicative of a positive association. But on testing the statistical association between the two, it was observed that the χ^2 value obtained was 0.7505 and also it was not significant (P> 0.5).

Table 213 Subjects as per irregular sleep and GSRS

Irregular sleep	GSRS >32	GSRS<32	Total
Positive	**46**	**16**	62
Negative	**86**	**52**	138
Total	**132**	**68**	**200**

Table 214 χ^2 table on irregular sleep and the GSRS score

Observed	Expected	χ^2	P value
46	40.92	0.7505	0.1
16	21.08		
86	91.08		
52	46.92		

10. MENTAL EXERTION AND GSRS SCORE

Table 215 Distribution of the subjects as per mental exertion and GSRS

Mental exertion	GSRS >32	GSRS<32	Total
Positive	112	39	151
Negative	20	29	49
Total	132	68	200

Table 216 χ^2 table on mental exertion and the GSRS score

Observed	Expected	χ^2	P value
112	99.66	0.9960	0.00018
39	51.34		
20	32.34		
29	16.66		

The calculated Odds ratio was 4.2 which was too positive in the association. As expected, on testing the association between the mental exertion positive, and the GSRS score, it was observed that the χ^2 value obtained was 0.9960 and it was highly significant as well. ($P < 0.001$).

11. Physical exertion and GSRS score

Table 217 Distribution of subjects as per physical exertion and GSRS

Physical exertion	GSRS >32	GSRS<32	Total
Positive	**130**	**56**	186
Negative	**2**	**12**	14
Total	132	68	200

Table 218 χ^2 table on physical exertion and the GSRS score

Observed	Expected	χ^2	P value
130	122.76	0.9961	0.00022
56	63.24		
2	9.24		
12	4.76		

The attained Odds ratio in this regard was almost 14, which points to the high incidence of the exposed one. On testing the statistical association between the regular physical exertion and GSRS score, it was observed that the χ^2 value obtained was 0.9961 and it was highly significant as well. ($P< 0.001$)

12. Family history of Amlapitta and GSRS score

On testing the association between the positive family history and the severity of GSRS score, it was observed that the χ^2 value obtained was 0.6660 and it was not significant ($P> 0.05$). The calculated Odds ratio was also 0.6 which is also supportive of the weak contribution.

Table 219 Distribution of the subjects as per family history of Amlapitta and GSRS

Family history	GSRS >32	GSRS<32	Total
Positive	26	19	45
Negative	106	49	155
Total	132	68	200

Table 220 χ^2 table on family history of Amlapitta and the GSRS score

Observed	Expected	χ^2	P value
26	29.7	0.6660	0.185
19	15.3		
106	102.3		
49	52.7		

13. Excess intake of processed food and GSRS score

Table 221 Distribution of the subjects according to excess processed food and GSRS

Processed food	GSRS >32	GSRS<32	Total
Positive	**107**	**47**	154
Negative	**25**	**21**	46
Total	132	68	200

Table 222 χ^2 **table on excess processed food intake and the GSRS score**

Observed	Expected	χ^2	P value
107	101.64	0.8108	0.0497
47	52.36		
25	30.36		
21	15.64		

In the relation between the use of processed food in excess and the GSRS, it was observed that the Odds ratio was 1.9. On testing the statistical association between the two, it was observed that the χ^2 value obtained was 0.8108 and it was minimally significant ($P < 0.05$).

14. Stale food and GSRS score

Table 223 **Distribution of the subjects with stale food and GSRS**

Stale food	GSRS >32	GSRS<32	Total
Positive	**18**	**23**	41
Negative	**114**	**45**	159
Total	132	68	200

Table 224 χ^2 **table on excess stale food intake and the GSRS score**

Observed	Expected	χ^2	P value
18	27.06	0.9773	0.0008
23	13.94		
114	104.94		
45	54.06		

On testing the association between the regular stale food intake and the severity of the presentation as GSRS score, it was observed that the χ^2 value obtained was 0.9773 and it was significant at 0.1% level. (P< 0.001). The Odds ratio was not supportive for the same, as it was only 0.31 on computing.

15. Excessive travelling and GSRS score

Table 225 Distribution of the subjects according to excessive travelling and GSRS

Excess travel	GSRS >32	GSRS<32	Total
Positive	20	24	44
Negative	112	44	156
Total	132	68	200

Table 226 χ^2 table on excessive travelling and the GSRS score

Observed	Expected	χ^2	P value
20	29.04	0.9732	0.0011
24	14.96		
112	102.96		
44	53.04		

On testing the association between the habit of excessive travelling and the severity as GSRS score, it was observed that χ^2 value obtained was 0.9732 and it was significant at 1% level. (P< 0.01). The calculated Odds ratio in this regard was 0.33.

16. Excess use of carbonated drink and GSRS score

Table 227 Distribution as per excess use of carbonated drink and GSRS

Carbonated drink	GSRS >32	GSRS<32	Total
Positive	**37**	**24**	61
Negative	**95**	**44**	139
Total	132	68	200

Table 228 χ^2 table on excess use of carbonated drink and GSRS score

Observed	Expected	χ^2	P value
37	40.26	0.5898	0.2905
24	20.74		
95	91.74		
44	47.26		

On testing the association between the habit of excessive intake of carbonated drink and the severity of presentation as GSRS score, it was observed that the χ^2 value obtained was 0.2905 and it was not significant. (P>0.05). Similar was the case of the computed Odds ratio, which was 0.72.

17. Excessive NSAID use and GSRS score

The Odds ratio calculated was 2.3 in this regard, showing a positive contribution. On testing the statistical association between the same, it was observed that the χ^2 value obtained was 0.2905 and it was significant at 5% level. (P<0.05).

Table 229 Distribution of the subjects as per excess NSAID and GSRS

NSAID use	GSRS >32	GSRS<32	TOTAL
Positive	**31**	**8**	39
Negative	**101**	**60**	161
Total	132	68	200

Table 230 χ^2 table on excess use of NSAID and the GSRS score

Observed	Expected	χ^2	P value
31	25.74	0.8274	0.0475
8	13.26		
101	106.26		
60	54.74		

18. Quantity of food intake and GSRS score

The Odds ratio was calculated as 2.1 which was suggestive of the causation. On testing the statistical association between the quantity of food intake and the GSRS score, it was observed that the χ^2 value obtained was 0.7152 and it was not significant as well (P> 0.05).

Table 231 Distribution of the subjects as per quantity of food intake and GSRS

Quantity of food	GSRS >32	GSRS<32	Total
Positive	**123**	**59**	182
Negative	**9**	**9**	18
Total	**132**	**68**	**200**

Table 232 χ^2 table on quantity of food intake and the GSRS score

Observed	Expected	χ^2	P value
123	120.12	0.7152	0.133
59	61.88		
9	11.88		
9	6.12		

19. Mixed diet and GSRS score

Table 233 Distribution of the subjects with mixed diet intake and GSRS

Mixed diet	GSRS >32	GSRS<32	Total
Positive	**144**	**46**	190
Negative	**4**	**6**	10
Total	**148**	**52**	**200**

Table 234 χ^2 table on mixed diet intake and the GSRS score

Observed	Expected	χ^2	P value
144	140.6	0.9131	0.0119
46	49.4		
4	7.4		
6	2.6		

In those with the mixed diet, the calculated Odds ratio was 4.7 which was very much indicative of the same. On testing, the statistical association, between the mixed food intake i.e. both the vegetarian and non vegetarian food and the GSRS score, it was observed that the χ^2 value obtained was 0.9131 and it was significant at 5% level. ($P< 0.05$).

20. Excess non compatible food intake and GSRS score

Table 235 Distribution as per non compatible food intake and GSRS

Non compatible food	GSRS >32	GSRS<32	Total
Positive	**116**	**57**	173
Negative	**16**	**11**	27
Total	132	68	200

Table 236 χ^2 table on non compatible intake and the GSRS score

Observed	Expected	χ^2	P value
116	114.18	0.5136	0.4266
57	58.82		
16	17.82		
11	9.18		

The Odds ratio was obtained as 1.4 in those with the history of non compatible food. On testing the statistical association between the two, it was observed that the χ^2 value obtained was 0.5136 but it was not significant(P> 0.05).

21. Attained menopause and GSRS score

In the study, several subjects were there, who have attained menopause, before inclusion in the study, due to the peculiarity in the age of inclusion. So to perceive whether there is any aggravation of the GSRS scores after the same, a χ^2 test was performed.

Table 237 Distribution of the subjects after menopause and GSRS

Menopause	GSRS >32	GSRS<32	Total
Positive	**18**	**5**	23
Negative	**114**	**63**	177
Total	132	68	200

Table 238 χ^2 table on attained menopause and the GSRS score

Observed	Expected	χ^2	P value
18	15.18	0.6654	0.187
5	7.82		
114	116.82		
63	60.18		

The Odds ratio was computed as 2 regarding the same. On testing the association between the already attained menopause and the severity as the GSRS score, it was observed that the χ^2 value was 0.6654 and it was not statistically significant as well. (P>0.05).

22. Vishamagni and GSRS score

On testing the association between the persons who are presenting with vishamagni and the severity of presentation as GSRS score, it was observed that the χ^2 value obtained was 0.3614 and it was not significant (P> 0.05). The Odds ratio was supportive in this regard as it was only 1.1.

Table 239 Distribution of the subjects as per vishamagni and GSRS

Vishamagni	GSRS >32	GSRS<32	Total
Positive	**45**	**15**	60
Negative	**103**	**37**	140
Total	148	52	200

Table 240 χ^2 table on vishamagni and the GSRS score

Observed	Expected	χ^2	P value
45	44.4	0.3614	0.8328
15	15.6		
103	103.6		
37	36.4		

23. Mandagni and GSRS score

The computed Odds ratio was 1.3 regarding the mandagni. On testing the statistical association between the two, it was observed that the χ^2 value obtained was 0.5052 and it was not significant as well. (P>0.05).

Table 241 Distribution of the subjects according to mandagni and GSRS

Mandagni	GSRS >32	GSRS<32	Total
Positive	97	31	128
Negative	51	21	72
Total	148	52	200

Table 242 χ^2 table on mandagni and the GSRS score

Observed	Expected	χ^2	P value
97	94.72	0.5052	0.4438
31	33.28		
51	53.28		
21	18.72		

24. Kroora koshta and GSRS score

Table 243 Distribution of the subjects as per kroora koshta and GSRS

Kroora koshta	GSRS >32	GSRS<32	Total
Positive	93	28	121
Negative	55	24	79
Total	148	52	200

Table 244 χ^2 table on kroora koshta and the GSRS score

Observed	Expected	χ^2	P value
93	89.54	0.6143	0.253
28	31.46		
55	58.46		
24	20.54		

On testing the association between the persons presented with the condition of kroorakoshta and the severity as the GSRS score, it was observed that the χ^2 value obtained was 0.6143 and it was not significant.(P>0.05). The Odds ratio obtained was 1.5 of the same.

25. Vathapitta prakrithi and GSRS

Table 245 Distribution of subjects with Vatha Pitta prakrithi and GSRS

Vatha Pitta prakrithi	GSRS >32	GSRS<32	Total
Positive	**51**	**26**	77
Negative	**97**	**26**	123
Total	148	52	200

Table 246 χ^2 table on Vatha Pitta prakrithi and the GSRS score

Observed	Expected	χ^2	P value
51	56.98	0.8273	0.0475
26	20.02		
97	91.02		
26	31.98		

On testing the association between the persons who were of Vatha Pitta prakrithi and the severity of presentation as GSRS score, it was observed that the χ^2 value obtained was 0.8273 and it was significant at 5% level. ($P< 0.05$). But the Odds ratio was 0.6, which was not supportive of the attained significance.

26. Vatha Kapha prakrithi and GSRS score

On testing the association between the persons with Vatha Kapha prakrithi and the severity of presentation as GSRS score, it was observed that the χ^2 value obtained was 0.3409 and it was not significant ($P> 0.05$). Similarly the attained Odds ratio was only 1.04 which was not supportive of the contribution.

Table 247 Distribution of the subjects according to Vatha Kapha prakrithi and GSRS

Vatha Kapha prakrithi	GSRS >32	GSRS<32	Total
Positive	**64**	**22**	86
Negative	**84**	**30**	114
Total	**148**	**52**	**200**

Table 248 χ^2 table on Vatha Kapha prakrithi and the GSRS score

Observed	Expected	χ^2	P value
64	63.64	0.3409	0.9066
22	22.36		
84	84.36		
30	29.64		

27. Rajasa prakrithi and GSRS score

Here the computed Odds ratio was only 0.45. On testing association between the persons who were with rajasa prakrithi and severity of presentation as GSRS, it was observed that the χ^2 value was 0.6945 and it was not significant.

Table 249 Distribution of the subjects as per rajasa prakrithi and GSRS

Rajasa prakrithi	GSRS >32	GSRS<32	Total
Positive	**125**	**48**	173
Negative	**23**	**4**	27
Total	**148**	**52**	**200**

Table 250 χ^2 table on rajasa prakrithi and the GSRS score

Observed	Expected	χ^2	P value
125	128.02	0.6945	0.1542
48	44.98		
23	19.98		
4	7.02		

28. Home manager and GSRS score

Table 251 Distribution of subjects as per home manager and GSRS

Home manager	GSRS >32	GSRS<32	Total
Positive	**58**	**11**	69
Negative	**90**	**41**	131
Total	**148**	**52**	**200**

Table 252 χ^2 table on home manager and their GSRS score

Observed	Expected	χ^2	P value
58	51.06	0.8915	0.0185
11	17.94		
90	96.94		
41	34.06		

In the association between those who were home managers and the GSRS, the Odds ratio was calculated as 2.4. On testing the association between them, it was observed that the χ^2 value was 0.8915 and it was significant statistically. ($P < 0.05$).

29. Job with physical exertion and GSRS

Table 253 Distribution of the subjects as per job with physical exertion and GSRS

Physical exertion	GSRS >32	GSRS<32	Total
Positive	**40**	**16**	56
Negative	**108**	**36**	144
Total	**148**	**52**	**200**

Table 254 χ^2 table on job with physical exertion and GSRS score

Observed	Expected	χ^2	P value
40	41.44	0.4366	0.6051
16	14.56		
108	106.56		
36	37.44		

Here the computed Odds ratio was 0.83. On testing the statistical association between those doing job with physical exertion and the GSRS score, it was observed that the χ^2 value obtained was 0.4366 and it was not significant.

CORRELATION BETWEEN THE VARIOUS FACTORS AND GSRS

Table 255 Correlation between the contributory factors and GSRS

GSRS	1
Vishamagni	-0.00549
Mandagni	0.08429
VK prakrithi	0.06362
VP prakriti	-0.18782
Home manager	**0.16109**
Clerical job	-0.08333
Physical exertion	0.01443
Business	-0.13735
Professional	0.07845
Madhyama koshta	-0.08292
Kroora koshta	**0.11442**
Rajasa prakriti	-0.07293
VP dosha	0.09798
VK dosha	-0.03541
Female	**0.10563**
Male	-0.10563
Weight	0.07123
Physical exertion	**0.33152**
Mental exertion	**0.31038**
Age	0.11740
Skipping meals	0.05146
Irregular sleep	0.05939
Food timing	**0.26344**
Carbonated drink	-0.03710
Family history	-0.00190
Amla	**0.15561**
Lavana	**0.17817**
Katu	**0.21435**

Considering the various etiological and also other modifying factors contributing to the GSRS score, a multilinear correlation test was performed. The results showed a weak positive correlation in age, female, home managers, kroora koshta, physical exertion, mental exertion, food timing and the excess use of the rasas katu, amla and lavana in the diet with the attained score of GSRS. The level of association was computed and recorded below.

Table 256 Association of various factors and FD – level of significance

Area	Observation	Significance level
Rasas	Madhura, Amla, lavana, Katu	< 0.05
Prakrithi	Vatha Pitta prakrithi	< 0.05
Food	Irregular timings	< 0.05
Food	Processed food	< 0.05
Food	Previously prepared	< 0.001
Diet	Mixed	< 0.05
Life style	Physical exertion after food	< 0.001
Life style	Mental exertion	< 0.001
Life style	Excessive travelling	< 0.01
Drug	NSAID	< 0.05

REGRESSION

A multilinear regression analysis was performed on the above factors and the computed multiple regression coefficient, R was found to be 0.5, showing a positive contribution of the studied variables, in the magnitude of GSRS score. Further, about 25% of the GSRS was contributed by the studied variables as shown by the R square value.

Table 257 Multiple Regression coefficient on analysis

Multiple R	0.498399
R Square	0.248402

An ANOVA was performed with the changes in GSRS produced by the studied variables against those contributed by the other factors. The F statistic was found to be statistically significant at appropriate degrees of freedom at 0.1% level ($P < 0.001$).

Table 258 Anova table on the influence of the variables on GSRS

	df	SS	MS	F	Significance F
Regression	16	1036.431116	64.776945	3.7800757	4.82425E-06
Residual	183	3135.963884	17.136415		
Total	199	4172.395			

On analyzing the contribution of the individual factors on the GSRS score, physical exertion showed high significance at 0.1% level ($P<0.001$), while mental exertion showed significance at 1% level ($P< 0.01$) and food timing showed significance at 5% level ($P< 0.05$).

Table 259 Contributions of individual factors on the GSRS score

	Coefficients	SE	t Stat	P-value	Lower 95%	Upper 95%
Intercept	23.311	5.990	3.892	0.000	11.493	35.129
Age	0.007	0.033	0.202	0.840	-0.058	0.072
Sex	-0.816	0.835	-0.977	0.330	-2.464	0.832
Occupation	-0.146	0.265	-0.550	0.583	-0.670	0.378
Weight	0.000	0.032	0.009	0.993	-0.063	0.064

	Coefficients	SE	t Stat	P-value	Lower 95%	Upper 95%
Physical exertion	**4.764**	**1.291**	**3.689**	**0.000**	**2.216**	**7.311**
Mental exertion	**2.357**	**0.786**	**2.999**	**0.003**	**0.807**	**3.908**
Skipping meals	-0.381	1.476	-0.258	0.796	-3.294	2.532
Irregular sleep	0.302	0.676	0.447	0.655	-1.031	1.636
Agni	0.734	0.537	1.367	0.173	-0.325	1.794
Koshta	0.537	0.571	0.940	0.348	-0.590	1.665
Food timing	**2.259**	**0.915**	**2.468**	**0.015**	**0.453**	**4.065**
Carbonated drink	0.153	0.781	0.196	0.845	-1.388	1.694
Family history	0.426	0.728	0.585	0.559	-1.011	1.863
Sareerika Prakriti	0.619	0.362	1.709	0.089	-0.095	1.333
Manasa prakrithi	0.351	0.849	0.413	0.680	-1.324	2.025
Dosha	-1.218	0.884	-1.378	0.170	-2.962	0.526

REGRESSION OF RASAS

Table 260 Multiple Regression coefficient on analysis of rasas

Multiple R	0.317138
R Square	0.100577

Table 261 Anova table on the influence of rasas on GSRS

	df	SS	MS	F	Significance F
Regression	3	419.6459515	139.882	7.30580928	0.00011426
Residual	196	3752.749049	19.14668		
Total	199	4172.395			

A multilinear regression analysis was performed on the mostly administered rasas ie. amla, lavana and katu and the computed multiple regression coefficient R

was found to be 0.31, showing a positive contribution of the studied variables, in the magnitude of GSRS score. Further, about 10 % of the GSRS was contributed by the studied variables, as shown by the R square value.

An ANOVA was performed with the changes in GSRS produced by studied variables against those produced by other factors. The F statistic was found as statistically significant at appropriate degrees of freedom at 0.1 % level ($P< 0.001$).

Table 262 Contributions of individual rasas on the GSRS score

	Coefficients	SE	t Stat	P-value	Lower 95%	Upper 95%
Intercept	26.8651	1.6790	16.0002	0.0000	23.5538	30.1764
Amla	2.8669	1.2220	2.3462	0.0200	0.4570	5.2767
Lavana	2.0685	0.7511	2.7538	0.0064	0.5872	3.5499
Katu	3.4165	1.1786	2.8988	0.0042	1.0922	5.7408

On analyzing the contribution of individual factors on the GSRS score, amla rasa showed significance at 5% level ($P< 0.05$), while lavana rasa showed significance at 1% level ($P< 0.01$) and katu rasa also showed significance at 1% level ($P< 0.01$).

PERCENTAGE OF RELIEF FOLLOWING THE THERAPY ON GSRS

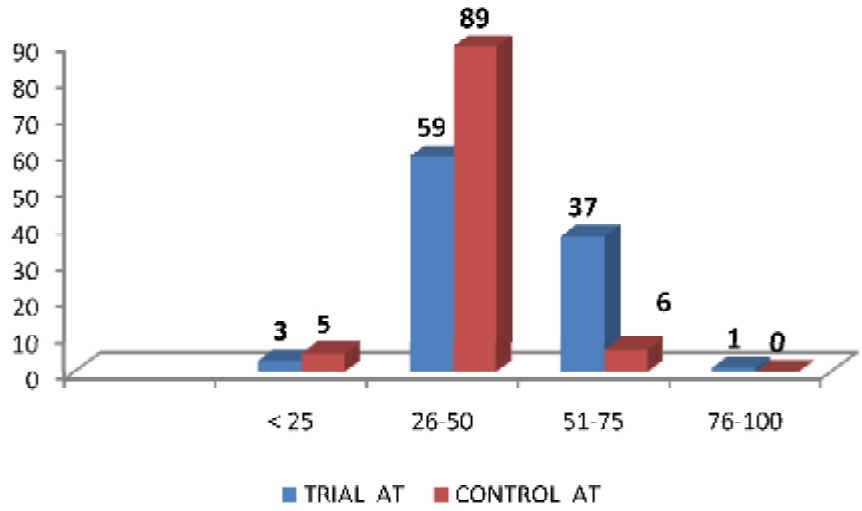
Table 263 Percentage of relief attained in both groups on GSRS

Percentage of relief	< 25	26-50	51-75	76-100
Trial after treatment	3	59	37	1
Trial after followup	2	9	88	1
Control after treatment	5	89	6	0
Control after followup	0	56	44	0

The 200 patients of both the groups were categorized according to the percentage of relief they attained, after the treatment period and also the follow-up on the observations recorded by GSRS scale. The grading done was

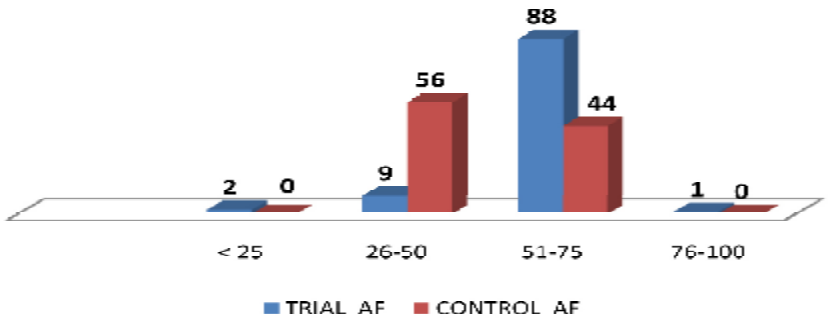
< 25%	-	unchanged
26% - 50%	-	slight improvement
51% - 75%	-	moderate improvement
76% - 100 %	-	marked improvement

In the trial group after the completion of medication, 3 remained in the group unchanged, 59 were in the group of slight improvement, 37 were in the group of moderate improvement and none in the group of marked improvement. In the trial group after the follow-up period after the medication, 2 were in the group unchanged, 9 in the group of slight improvement, 88 were in the group of moderate improvement and one subject, in the group of marked improvement.

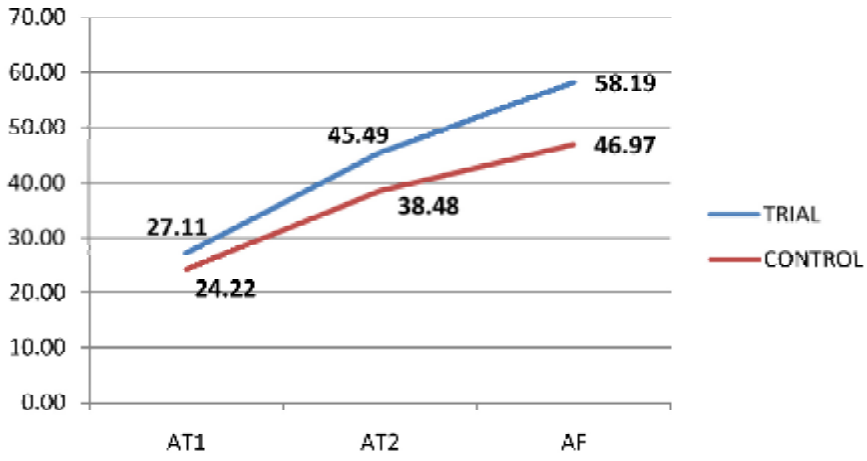


Graph 125: Percentage of relief after the intervention in both groups - GSRS

In the control group after the completion of medication, 5 remained in the group unchanged, 89 in the group of slight improvement, 6 were in the group of moderate improvement and 1 was there in the group of marked improvement. In the control group after the follow-up period after medication, none were in the group



Graph 126: Percentage of relief on the follow up in both groups -GSRS



Graph 127: Average Percentage of relief attained in both the groups on GSRS

unchanged, 56 were in the group of slight improvement, 44 in the group of moderate improvement and none was there in the group of marked improvement.

The average percentage of relief attained, in both the groups, on all the three assessments are given below.

PERCENTAGE OF RELIEF FOLLOWING THE THERAPY ON AMLAPITTA RATING SCALE

The 200 patients of both the groups were categorized according to the percentage of relief they attained after the treatment period and also the follow-up as per the observations recorded by the Amlapitta rating scale. They were graded as mentioned above.

Table 264 Percentage of relief attained in both groups on Amlapitta rating scale

Percentage of relief	< 25	26-50	51-75	76-100
Trial after treatment	4	37	59	0
Trial after follow up	3	9	87	1
Control after treatment	9	66	25	0
Control after follow up	2	35	63	0

In the trial group after the completion of medication, 4 were in the group unchanged, 37 were in the group of slight improvement, 59 were in the group of moderate improvement and none was in the group of marked improvement. In the trial group after the follow-up period of medication, 3 were in the group unchanged, 9 were in the group of slight improvement, 87 were in the group of moderate improvement and one was in the group of marked improvement.

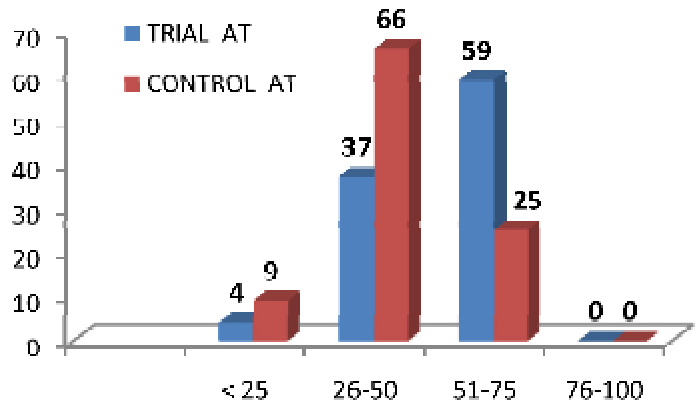
In the control group after the completion of medication, 9 were in the group unchanged, 66 were in the group of slight improvement, 25 were in the group of moderate improvement and none was in the group of marked improvement. In the control group after the follow-up period of medication, 2 were in the group unchanged, 35 were in the group of slight improvement, 63 were in the group of moderate improvement and none was in the group of marked improvement.

PERCENTAGE OF RELIEF FOLLOWING THE THERAPY ON AMLAPITTA RATING SCALE

The 200 patients of both the groups were categorized according to the percentage of relief they attained after the treatment period and also the follow-up as per the observations recorded by the Amlapitta rating scale. They were graded as mentioned above.

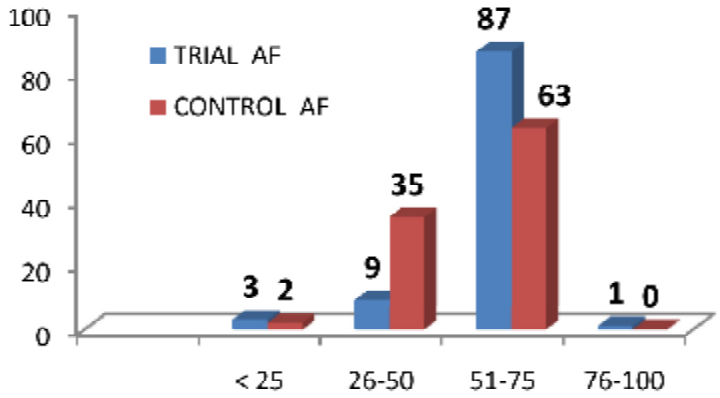
In the trial group after the completion of the medication, 4 were in the group unchanged, 37 were in the group of slight improvement, 59 were in the group of moderate improvement and none was there in the group of marked improvement. In the trial group after the follow-up period of medication, 3 were in the group unchanged, 9 were in the group of slight improvement, 87 were in the group of moderate improvement and one was there in the group of marked improvement.

In the control group after the completion of medication, 9 were in the group unchanged, 66 in the group of slight improvement, 25 were in the group of moderate

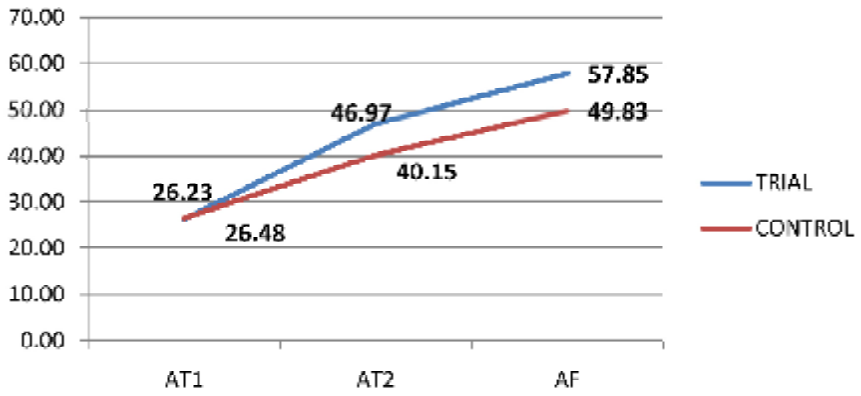


Graph 128: Percentage of relief after the intervention in both groups - Amlapitta

improvement and none was there in the group of marked improvement. In the control group after the follow-up period, 2 were in the group unchanged, 35 were there in the group of slight improvement, 63 in the group of moderate improvement and none was there in the group of marked improvement



Graph 129: Percentage of relief on the follow-up in both groups- Amlapitta



Graph 130: Average Percentage of relief attained in both groups on Amlapitta score

The diagram shows that the average percentage of relief attained by the study drug is better when compared with the control at the end of the intervention and also the follow-up period of the study. Both are having similar scores on the first assessment after 15 days of medication. The trial drug is maintaining the attained effect in a better manner when compared with the control group.

Chapter - 7

DISCUSSION, SUMMARY & CONCLUSION

DISCUSSION

The observations during the study were recorded and analyzed with the suitable statistical tests and the possible inference was made by comparing with the available and published studies and also the supportive conceptual facts.

Demographic Data

In the clinical study, there was a slight male predominance in both the groups. Overall, several studies conclude that in Functional Dyspepsia, slight male predominance is observed. The reason may be due to the relative variations in diet, travelling and also the excessive stress compared to females. Most population studies have been able to obtain relatively equal ratios of male: female ie. they have shown no differences in the prevalence of dyspepsia between genders, mostly where Ulcerative Dyspepsia is concerned[1]. Some other studies, in different populations, however, have also noted a consistent female preponderance with dyspepsia[2]. So noticeable variation cannot be observed in the case of gender in FD.

Half of the population was from the age group between 40 and 50 years. The remaining subjects were almost equally distributed in the other two groups which points to the increased occurrence of the disease, in the forties. All surveys that have been conducted have examined adults of 18 years or older. While most surveys have shown that dyspepsia does not emerge to be related to any particular age group, several studies have noted dissimilar trends. Peak prevalence of Ulcerative Dyspepsia have been noted between the ages 45-54 in a Canadian survey[3] whilst FD appeared to peak in Chinese subjects of the age group 41-50 years[4] and in Japanese adults of 50-59 years[5].

In contrast, a survey in urban part of Mumbai established that Ulcerative Dyspepsia was more prevalent in adults of age more than 40 years[6]. Despite these trends, age extremities have not been identified as a predictor of dyspepsia, either functional or structural types. Majority of the population based studies do not show any gender difference in dyspepsia prevalence and few studies concluded that the prevalence is more in the old age population. It can be assumed that the dietary alterations in a negative manner is contributing to the condition gradually. Also the changes happening in the tract, by several years of insult by the food habits is causative. These leads to dyspepsia at a later stage, with the chronicity in the contributory factors.

The gradual decrease in the level of agni by age along with the continuous indulging in the nidanas lead to the condition, at a later age group. Vaghbata mentions this while explaining the management of Grahani that, even though one is indulging to the nidana of grahani, the disease is manifesting, when the status of agni declines, after a certain period only.

The Muslim community, which was the majority, nearby the institution

and the district, were also reflected in the study, as they contributed up to 60%. It is also reported that the gastrointestinal problems are on the rise in this community, due to their excessive and regular intake of the spicy and non vegetarian diets. Likewise is the case of intake of processed and junk food as well. The role of the ethnicity is also being discussed worldwide. A reported study from Malaysia concludes with the increased prevalence of FD in the Chinese and the Indian population over there.[7]

The population was equally distributed among the married and unmarried groups. No studies pointing out to the relation between the marital status and dyspepsia could be traced out, at this stage.

Most of the people belongs to the middle class in economy. This seems as expected as the hospital is approached chiefly by the low and the middle class community, from the center part of Kerala. In majority of the population based studies, prevalence of dyspepsia have not found to be linked with the social class. However studies examining the details of socio economic status were able to elicit associations with dyspepsia. In a British study, it was found that people from lower socioeconomic classes, were more affected with Functional dyspepsia and other associated conditions[8].

Majority of the subjects were having an education of secondary level and above. This is also the reflection of the low and middle class community approaching this hospital. Also majority of the people from this state is having an education of at least the secondary level. A survey study from Canada revealed that chronic GI symptoms were more prevalent in adults with lower household income, who were unemployed and with lower educational levels which cannot be a generalisation.[9]

While observing the occupation,the majority were in two groups ie. those with job having physical exertion and also the home managers. The condition of

dyspepsia is more reported in those with physical exertion. Home managers were also affected as their food timings were too irregular and they were having a much more stress level, than expected. Business men were also affected as their food habits were irregular, they were involved in excessive travelling and also associated with stress as well. In a study conducted at Nigeria, a larger sized family together with occupational scatter, was reported to have strongly associated with UD[10]

Observations regarding Dasavidha pareeksha

Agni is one of the crucial factors to be studied in any disease peculiarly that affecting the GI tract. In this study, majority were having mandagni in both the groups. Vishamagni was also observed in more than a few. The relationship of agnimandya contributing to the manifestation of diseases such as Amlapitta is very much substantiated, by the number of patients reported with mandagni, in this peculiar study. Agnimandya leads to ama and the disturbed Pitta due to the above said nidana, incorporates with ama, leading to the Pitta becoming saama in nature. Hence the Kaphaja symptoms like hrillasa, gourava, avipaka etc. are manifested along with the Pitta symptoms such as hrit-kanta daha in Amlapitta. The initial management to be adopted is of Pitta Kapha samana, so as to subside the ama associated with Pitta. Later Pitta samana or Vatha Pitta samana protocol is advised as per the condition. Vishamagni is also contributing to the pathology by its alteration, but not as the usual level, as that of the mandagni.

Kroora koshta was observed in almost 60% of the subjects. It seems that kroora koshta is very much associated with the manifestation of symptoms of Amlapitta. The Vatha kopa in the GI tract in the case of kroora koshta is contributing to the symptoms of conditions like FD, resulting from the altered functioning of the other

doshas. Alteration in the SamanaVatha alters the status of both Kledaka Kapha and Pachaka Pitta, Vatha being the regulator. In this particular study, very few individuals were having mridu koshta, but the possible inverse relationship of the same, has to be studied in detail.

The nature of the pureesha is an indicator of the mechanism of digestion and its alteration is to be considered quite seriously, peculiarly in a GI tract disorder. That is the reason for pureesha pareeksha and its significance mentioned among the ashtavidha pareekshas. In this study, two parameters were assessed, whether the pureesha is sama or nirama and also whether the pravritti is badha or in the drava avastha, the details are mentioned in classics, while explaining the athisara.

In the study, majority were having malapravritti as nirama and a relation cannot be established between sama mala pravrithi and Amlapitta. 90% of them were having badha pureesha or were constipated. It seems that badha pureesha and dyspeptic symptoms are very much related, the associated Vatha kopa being the causative factor. The krichrapravritti of pureesha was also noticed in 10% of subjects.

The relationship between prakrithi and the increased occurrence of certain diseases according the prakrithi, has been studied. Almost 40% were having Vatha Pitta prakrithi. Also equal number was having Vatha Kapha prakrithi. All the other types of prakrithi, were fewer among the included subjects. An association of the same with the condition, is yet to be traced out.

When the manasa prakrithi was assessed, 85% were of the rajasa prakrithi. These point to the increased occurrence of dyspepsia, in those with the rajasa prakrithi. These people are more with the custom of intake of katu- amla- lavana- ushna ahara and also excess psychological features like krodha and matsarya, resulting in the

aggravation of Pitta and associated conditions. The other two manasa prakrithi's, the satwika and tamasa were not so dominant.

More than 60% of the subjects were of madhyama satwa. Actually it is found in studies that the Functional Dyspepsia and similar conditions are more found in the avara satwa persons. This study does not agree with the fact wholly but overall, one fourth of the patients were having avara satwa, as per the observation. This is expected to happen in functional conditions, with the psychological component as one of the main etiological factor.

The importance of satwa and its importance in the diagnosis as well as the management of diseases, have been explained by Charaka, in the Vimanasthana. Pravara satwa is also one of the contributors to the immune status of the individual. In a survey conducted in a selected population in Denmark, it was noted that UD was strongly associated with adults who had "experience of problems" and "psychological vulnerability[11].

More than 80% of the subjects were having Vatha Pitta as the dosha status of the condition. In about 10%, the dosha status was Vatha Kaphaja. The dosha status can be concluded as Vatha Pittaja in nature and in some of the subjects, Kapha also seems significant, may be due to the association of the ama and its similitude with the Kapha. The relationship between the corresponding prakrithi and dosha has to be studied further.

While assessing the affected dhatus, from the observed lakshanas clinically, rasa dhatu was involved in 90% of the subjects as the symptoms of agnimandya, praseka, gourava, glani etc. were reported. Both rasa and rakta dhatu were affected in about 10% of the subjects. We can infer that the rasa dhatu is the primarily affected

one, as it is having direct relation with the digestion and metabolism of food. The defective agni is not able to transform the ahara rasa to proper rasadhatu. There was also slight involvement of the rakta dhatu even though not considerably significant, due to the ahara and vihara, causing the aggravation of Pitta and also the mutual association of Pitta and raktha. The other dhatus are having a chance of vitiation as the chronicity occurs in due course, as all the dhatus entail the nutrition, from the properly digested and absorbed ahara.

Factors regarding food intake and digestion

Most of the subjects were non vegetarians ie. 90%, this was also due to the area wise distribution. This is an indication of the increased occurrence of dyspepsia among the non vegetarians, as per studies. 14% of the subjects in the study group and 13% in the control were having processed food, frequently. 20% among the study group and 26% in the control group were of the habit of consuming non compatible food, in a frequent manner. These also are an indicator of the contribution of such factors, in the manifestation of FD. In an urban survey in India, it was proved that no differences in dyspeptic symptoms occurred between vegetarians and meat-eaters whilst spicy, fried or food prepared outside the home contributed insignificantly, to worsening of symptoms[12].

Timing of meals is one of the most important aspects, regarding the digestion and absorption of the food. This is one of the areas which people are neglecting or not following now a days. In this study also, around 80% were having the habit of irregular timing of meals. Dyspepsia and irregular meal timings seem very much related, as per this study. Many were having the habit of skipping their meals, on a regular basis. Keeping a regular schedule is a necessity, for the timely digestion as well as absorption of the food.

The pace of intake of their food was slow in 2/3rd of the included and more than 1/3rd were having it, in a hurried manner. Alteration in the pace of food intake is really affecting the digestion as well as absorption of its contents, as per studies. The food habits have to be advised in such a manner so that, it may be chewed properly before swallowing, so as to support the digestion. Kharanada opines that one who is so paceful with food cannot evaluate the guna and dosha of it and those who are too slow cannot recognize the stage of tripthi ie. when to stop the food.

Almost 80% were having food, before they felt genuine appetite. This also seems affecting the mechanism of digestion. Around 10% were having processed food on a regular basis, which is also contributing to dyspepsia, as per studies. The overall rise in the intake of processed food has resulted, in the increased incidence of conditions like FD.

3/4th of the subjects were having their food in an atmosphere, which was not calm and was really tense as well. In the modern world, people are not allotting the time that is unique for the intake of food. They are having their food amidst of their stress situations with their profession and so. This is very much contributory to the conditions like Amlapitta. That is the reason behind the explanation of ahara vidhi and its details by Charaka so that, one must have food in a calm atmosphere with a relaxed mind, so as to enhance its digestion, as well as absorption. The altered psychological status, affects the intestinal movements and hence digestion.

In a retrospective study, it was observed that, alteration in the intake of meals such as changes in the usual timings, variation in the amount of food intaken and the regular practice of skipping meals, over a period of years is associated with the increased incidence of H Pylori infection and gastritis[13]. The exact pathogenesis is yet

to be explained, but the resultant alteration in the secretions of the tract and also the bicarbonate layer, creates a positive situation for the organism to invade. Also the action by the organism results in a complex interaction between genetic, socio economic, environmental and bacterial factors, resulting in multiple outcomes[14].

Quality of food

The excessive use of any of the six rasas or its combination was assessed to review the etiological component of FD. Among which the excessive use of mainly three rasas amla, lavana and katu were reported. About 90% were using amla rasa more, 75% were using much of lavana and around 90% were having katu rasa in excess. The excessive use of these rasas are very much contributing to the Pitta kopa and the manifestation of the disease. This also shows the importance of avoiding such rasas in the diet resulting in the Pitta kopa, leading to Amlapitta and similar conditions.

The guna or characteristics of the food is having direct association in the manifestation of any disorder. In this study whether the subjects were using any peculiar type of ahara in excess, was traced. The four main gunas, snigdha, rooksha, ushna and seetha were recorded. It was observed that 1/3rd were using snigdha guna in excess and ½ of them were using rooksha guna in excess. Seeta was used in excess in a few of the subjects overall. It seems that excessive use of both the snigdha as well as rooksha diet, leads to dyspeptic conditions, by affecting the act of digestion and altering the dosha status. As the agni is a sum of Samana Vatha, Pachaka Pitta and Kledaka Kapha, any alteration may cause distorted digestion. Similarly excessive use of ahara with the ushna guna contributes to Pitta kopa and hence dyspepsia. Ushna, rooksha and snigdha are the chief contributors here in this regard.

Factors of digestion

Abyavaharana is the capacity of food intake. It was affected considerably in most of the subjects. Abyavaharana sakthi was heena in almost 80% and it was pravara in minimum. From this it can be inferred that, abyavaharana sakthi is affected in almost all the subjects with dyspepsia. Aruchi is mentioned as one of the classical feature of Amlapitta as well.

Jarana sakthi is the ability to digest the intaken food in an appropriate manner. It was also affected as per the observation, but not to the extent of abyavaharana. In almost 60% of the subjects, jarana was madhyama in nature and it was heena in only 1/4th of them. The abyavaharana is affected much more than the jarana in this study, even though avipaka is also mentioned.

Factors affecting the proper digestion of the food were also assessed accordingly. Of which, majority of the subjects were having physical exertion. Mental exertion was observed in 3/4th of them. 90% of them were having the habit of skipping the meals on a regular basis. Irregular sleep pattern was there in 1/4th of them. Both physical and mental exertions and the habit of skipping of meals are contributing to dyspepsia causing impaired digestion, as per the study.

In this study, 1/3rd of the subjects of the study group were having the regular habit of carbonated drinks. Even though this is more seen in the society, the decrease in the number may be due to the inclusion of more the lower and the middle class. This is one of the habits contributing towards the manifestation of condition like dyspepsia, as per studies. This is to be modified or altered. In this study alcohol, tobacco and smoking habits were reported in less than 10% of the subjects. A survey from Canada showed that the heavy intake of cola or carbonated drink was associated

with markedly increased prevalence of dyspepsia.[15] Population studies in India and New Zealand have pointed to definite association between alcohol and UD[16].

Frequent use of NSAID'S are considered as a contributor to the manifestation of dyspepsia. In this peculiar study, 1/5th were using NSAID's in excess. This was less when compared in accordance with the available studies. In a published study NSAID's, Aspirin, hormone therapy, over the counter medications are significantly contributing to FD.[17]

In the study group, half of the patients were having reduced or interrupted sleep. The alteration in the sleep pattern is very much, affecting the abdominal physiology and contributing to conditions like dyspepsia, as per studies. Similarly, day time sleep is also having a role in the functioning of the abdomen. This may lead to Vatha Pitta aggravation and hence to the condition. But in this peculiar study, very few of the patients were only having the habit of day time sleep, which is not so significant here. Prajagara or insufficient sleep is being mentioned as one of the causes of indigestion by Acharya Charaka, even though the agni is of normal in status.

Family History

Family history is very much being blamed for the manifestation of the conditions such as Functional Dyspepsia and there are studies supportive for it. In this study, 1/5th of the subjects were with the family history of dyspepsic symptoms, overall. This is not in accordance with the available studies as in the case of positive family history. Even though published studies are not available as such in FD, the family history is very much significant, when all the types of dyspepsias were studied[18].

Abdominal Examination

Regarding the shape of the abdomen, half of them were having distended

abdomen. Even though it doesn't point to the abdominal dysfunction, it is a real indicator of the truncal obesity and abdominal fat deposition observed nowadays, in common. The lack of exercise or the zero exercise is the real contributor here. This is also affecting the normal peristaltic movements as well as the physiology of digestion. 1/3rd of the subjects were having the previous history of abdominal surgeries, prior to inclusion and were with the scar of previously performed surgeries. As far as tenderness is concerned, it was elicitable in the epigastrium in more than 95% of subjects and also in the umbilical region in more than 80% of them. In the other areas of the abdomen, the tenderness was distributed unevenly across the patients, on examination.

Symptoms of Functional Dyspepsia

The clinical presentation of those included in the study were observed, recorded and analyzed accordingly. The symptom of abdominal pain was noticed in almost all the patients from both the groups, with a mean duration of 18 months. Similar was the case of fullness of abdomen, with the mean duration of 20months. These two are the most prominent presenting symptoms of those with FD, as per the observations.

Belching was also seen in most of the included subjects. Acid regurgitation was noticed in almost 90% of the subjects from both the groups. The burning sensation was complained in almost 98% of the subjects overall, but the duration was much less than abdominal pain or fullness (mean duration of almost 12 months). The symptoms of abdominal pain and fullness were reported at an earlier stage than the burning sensation, in this study.

The symptom of sucking sensation was not so common and seen in 1/3rd only. The symptom of nausea or vomiting was noticed in almost 70% of the subjects with a mean duration less than 9 months. Anorexia was recorded in ¾th of the subjects

with a mean duration of almost 18 months. Borborygymi was observed in ¼th of the subjects with a mean duration of 12 months. The mean duration of increased flatus was around 18 months, but the symptom was there in only 1/4th of the subjects.

Constipation was one of the most dominant symptom observed in almost 90% of the subjects with a mean duration of 22 months. Bothersome post prandial fullness was observed in all the subjects from both the groups with a mean duration of 20 months. The symptom of early satiation with food intake was also observed in all the subjects, but with duration slightly less than 18 months. Anorexia and constipation were the most prolonged symptom observed followed by fullness abdomen and the abdominal pain.

Among the included subjects, it was tried to categorize them into the types of Functional Dyspepsia ie. Post prandial distress syndrome and the Epigastric pain syndrome, as per the Rome III criteria. Most were having an overlapping nature of the two subtypes. It seems that in the post prandial distress syndrome, the symptoms are much more Kaphaja in nature, also seen much more in the earlier stage of the disease and the management with pachana drugs that do not aggravate Pitta, is the prime option. In the Epigastric pain syndrome, it is much more Vathika in presentation and Vatha samana chikitsa, including gritha preparations are ideal in this regard.

Symptoms of Amlapitta

Daha was observed in 99% of the subjects with a mean duration of 12 months. Amlodgara was noticed in 92% overall, with a mean duration of 10 months. Chardi was only there in 70% of the subjects, with a mean duration less than 8 months. Soola was observed in almost 98% of the patients, with a mean duration of 18 months. Avipaka was there in 70% of them, with a mean duration of 16 months. Soola and

daha were the most dominating and prevalent symptoms with the soola being reported, at an earlier stage of presentation.

Efficacy of the therapy on the symptoms of Functional Dyspepsia

The observations attained regarding the various symptoms in both the groups on the GSRS score both individually and in total, were analyzed and the inferences being made, as per the appropriate tests.

In the symptom of abdominal pain, there was significant efficacy in both the groups. The comparison in between the groups was done and was also significant. The comparison between the two groups was highly significant on the first assessment, significant on second and not significant on the follow-up. The study drug seems to be of more swift in action compared with the control and as the intervention goes on, the efficacy seems similar in nature. There was also better percentage of relief in the study group. Mahatiktaka yoga by its action at the level of pachana, agnideepana and anulomana seems to have an efficacy on the symptom of abdominal pain, in the subjects. The drug is also able to subside the associated ama, which is contributing to soola.

In heart burn, the efficacy was there in both the groups after the intervention. There was slight less relief in the control, during the follow up period. This shows that the attained improvement in the heart burn was not maintained by the control group, during the follow-up period, as the study drug. On comparing the efficacy between the groups, it was highly significant on first assessment, not significant on second and it was minimal on follow-up. It can be inferred that the study drug works early and by the end of the medication, both are having similar efficacy. The percentage of relief was better in study group. The symptom daha is very much associated with

the Pitta dosha and many a drugs in the combination of Mahatiktaka seems Pittasamana, explaining the action of the drug in this context.

On comparison in acid regurgitation, it was inferred that both the groups were highly significant. The post hoc was significant at all levels with slightly less in control, during follow up.On comparing between groups, it was highly significant on first, not significant on second and minimal on final assessment. It can be inferred that the study drug performs more on the first and final assessments i.e. the initial stage and the follow-up with better relief percentage. The study drug is maintaining the attained efficacy in a better manner. The symptom of amlodgara has to be managed by the Pittahara as well as the Vathanulomana combination, which is accomplished by the study drug here.

On fixing the efficacy of the two groups on sucking sensation, it was found that the study group showed minimal significance than the control group, but was not effective statistically. Both the groups showed significance at all levels, except follow up. The benefit was not able to maintain by both the drugs. On comparison, the study drug showed highly significant difference than the control, in all assessments with a better percentage of relief. The study drug is definitely having superior efficacy than the control drug in this symptom. The sucking sensation is resulting from the agnimandya and the resultant vridhi of Kapha in the amasaya. The pachana and the anulomana effect of the drugs in the Mahatiktaka combination seems to overcome the condition.

In the symptom of nausea/ vomiting, there was highly significant efficacy in both the groups and also during the multi level comparison. The control drug is not performing upto the level as the study drug, on the follow up period. On comparison, the minimal significance on the first assessment was not able to maintain further even

though, better relief was reported in the trial. The symptom of nausea/ vomiting seems to result from amadosha and also the pratiloma gati of Vatha, which seems to have worked upon by the study drug, on its use. The pachana as well as anulomana drugs of the study drug are useful here.

There was high significance in borborygymi in the study group, while the control group showed only minimal significance, after the intervention. This point to the difference in the efficacy and both were not significant at the follow up. On comparison between groups, after the intervention, it was significant, but not on follow up. The study drug was not able to maintain the initial benefit in the later stages. The symptom of borborygymi seems due to the aggravation of the Vatha dosha and there is slight limitation on the action regarding the same as far as the Mahatiktaka tablet is concerned, due to the rookshata of the formulation.

Highly significant efficacy was observed in both the groups after the intervention, on abdominal distension.The study drug has a significant action better than control on the first 15 days of therapy and later, the two drugs are similar in efficacy. In the symptom of abdominal distension also, the Mahatiktaka tablet is not showing a promising effect due to its limitation on the action on the Vatha dosha due to the rooksha nature. The kashaya as a whole seems sthambana to an extent, like wise is the tablet. The attained effect may be due to resultant Vathanulomana, due to drug.

The pre and post therapy scores show high significance on eructation in both the groups pointing to their efficacy. The multiple comparisons were also significant in both, except the follow up period in control. It can be inferred that the control drug is not maintaining the efficacy as the study drug, during the follow-up period. The significance attained by the study drug after the intervention was not there, on follow

up. Here also the study drug was not having the expected efficacy on eructation as the attained vathanuloma was not upto the mark.

The intervention was highly significant in both the groups except during the follow-up period, in increased flatus. It shows that both the drugs are not maintaining the same results, in the follow-up period. On comparison between the two groups, it was found as significant on all the assessments. This shows that the study drug is having supremacy over the control, in increased flatus. Many of the drugs in the Mahatiktaka is renowned for pachana as well as anulomana activity, which is being presented here.

Both the groups were not statistically significant before and after the intervention and not effective, in managing the decreased stool. On comparing the efficacy between the two groups, it was found that it was highly significant on all the three assessments in favour of the study drug. The efficacy was not uniform throughout the therapy. Even though the efficacy was not statistically significant overall, the study drug works better than its control. The study drug is being used with success in many a conditions of the lower GI tract such as Irritable Bowel Syndrome, Inflammatory Bowel disease etc. in the clinical level. Here also, the pachana, deepana and anulomana factors are on the role.

The difference in the efficacy of the treatment was not statistically significant in both the groups, on loose stool. On the efficacy at various stages of assessment, the significance was between the first and last assessments only. The other levels were not significant. The improvement attained was not uniform. On comparing the significance between groups, it was highly significant on all the three assessments. Here also the study drug is having supremacy, even though there was not much difference on the pre

and post scores. Many of the drugs are having the function of pachana and grahi, which is very much relevant in this regard.

On assessing the efficacy on hard stool after the intervention, it was observed high significance in both the groups. On comparing the efficacy at the various stages, it was found as highly significant in both, but the study drug was not performing as the control on follow up. There was highly significant difference in the efficacy between the groups, at all levels of assessment. This shows that the study group is better on the symptom of hard stool but efficacy not maintained on follow up. Here also the anulomana property of drugs in the Mahatiktaka is performing the job.

There was no significant efficacy in both the groups with the intervention in the urgency of defecation. The control group performed a bit better when compared within the stages of assessment. On comparison between groups,the trial drug was highly significant in all the three levels of assessment. There was uneven distribution of efficacy between groups. The urgency of defecation is one of the symptoms mentioned in condition like Kaphaja athisara or Grahani. Here also a drug which is Kaphahara, pachana as well as grahi in action like Mahatiktaka, is having its own efficacy.

In the feeling of incomplete evacuation, both the groups were not significant. The multi level assessment was varied which shows that the initial response attained is not maintained at the other levels of the assessment, in both the groups. While comparing the efficacy between groups, it was found that the difference was highly significant between the groups, at all the assessments. The symptom resembling the feeling of incomplete evacuation has been explained by Acharya Vaghbata while explaining Kaphatisara lakshana and it is Vatha kaphaja in nature. The Kapha samana, pachana and the grahi nature of selected drugs of the Mahatiktaka is working here.

Efficacy on the GSRS total score

There was reduction in the total score of GSRS in both the groups, after the intervention. When it was assessed for efficacy before and after the treatment, it was observed that, both the groups were highly significant. When the efficacy was compared at all the stages of the assessment, high significance was observed at all levels, in both the groups, by post hoc test. The efficacy between the groups when compared showed varied results. It was not significant on 1st assessment, significant at 1% level on second and highly significant on the final assessment. There was better percentage of relief in the trial group.

The results indicate that, in the initial stages, the two drugs are having a similar efficacy. But as the intervention progresses, the study drug acts further and the results are maintained well, during the follow up period also, on comparison with control. This explains the improving significance throughout the intervention. Mahatiktakam kwatham tablet exhibits better results on its continuous administration. Even though it starts working at a slower pace, it makes up and performs better than the control, in due course of the therapy. The better percentage of relief is also an indicator of the same.

Efficacy of the therapy on symptoms of Amlapitta

The efficacy of both the groups on the selected symptoms of Amlapitta according to the Amlapitta rating scale was also evaluated, as per the recorded observations. The five selected as well as the common symptoms of Amlapitta, as well as the total score were recorded.

Pre and post intervention scores was highly significant in both the groups, in daha. It was found on multiple comparison that at all the levels, the efficacy is highly

significant in both the groups. On comparison between the groups, it was not significant on the first assessment, highly significant on second and not significant on the final one. The action of the drug varies throughout the course. The initial effectiveness was similar, but on finishing the therapy, the study drug has a better action but on the follow-up period, it was not maintained. There was positive difference in the case of study drug, in the percentage of relief. Mahatiktaka yoga is much Pitta samana in nature and is working on daha. But the improvement attained was not maintained during the follow up. This is an indication of the need for further continuation of the drug for attaining better results in daha.

The efficacy of the intervention was highly significant in both the groups in chardi. On the changes in each stage of assessment, the control was significant at all levels, but the study drug was not significant, during the follow up. On comparing between, it was only significant on first assessment only. The earlier supremacy of the study drug on the symptom of chardi was not maintained through the rest of the intervention, as there was no difference in efficacy. The maintanence during follow up was also poor for the study drug. It can be concluded that there is no difference in the efficacy on chardi between the groups after the intervention, as well as the follow-up. But there was significant difference between, after 15 days of the therapy. Mahatiktaka acts at a better pace on the symptom of chardi, but it was not maintained through out.

On computing the efficacy of the treatment in soola, it was observed that there was highly significant change, in both the groups. The post hoc test seems highly significant at all levels of assessment, in both the groups. On comparison between the groups, there was significant difference between the two groups at all the assessments. This shows the supremacy of the study drug in the condition of soola. There was no

significant difference between the two groups in the percentage of relief. Mahatiktakam kwatham tablet is more effective in soola, when compared with the control.

In avipaka also, it was found that the difference in efficacy was highly significant in both the groups, after the therapy. There was highly significant improvement at all the levels in control, but it was not significant on the follow up, in trial. The initial significance obtained on comparison was not maintained later and became insignificant. The difference of the percentage of relief was more during the 2nd assessment between the groups, indicating that the study drug was better after the intervention, but not maintained as such during the follow up.

Regarding the symptom amlodgara also, the efficacy in both the groups were highly significant. The comparison in between groups were also significant, but at a lower level on follow up. There was highly significant difference at all levels on comparison between. This indicates that the study drug is better than the control in relieving amlodgara and is working better on the follow up period.

Efficacy on the total score of Amlapitta

In the total score of Amlapitta, there was significant reduction in the total score in both the groups. On assessing the efficacy of the therapy, it was highly significant in both the groups. It seems that both the groups are effective in Amlapitta. On detailing the attained improvement in both the groups, it was found that in the study group as well as the control, it was highly significant at all the levels of the assessment showing the improvement at all the levels of the therapy. On comparing the groups for efficacy, it was significant at 1% level on the 1st assessment, not significant at 2nd assessment and minimally significant at the final assessment. This indicates that the efficacy varies between the two groups throughout, with a slight better response in the study group.

The initial response seems better in the study group but at the end of the intervention, both were similar in efficacy. But on the follow-up period, there was significance, as the study drug maintained the improvement in a better manner. On the percentage of relief, there was slight better improvement in the study group as well.

Level of significance on the GSRS score with the therapy

Highly significant difference was observed in the symptoms of sucking sensation, increased flatus, decreased stool, loose stool, hard stool, urgency of defecation and the feeling of incomplete evacuation. Of the above symptoms, except the sucking sensation, all are related with the lower GI tract, where the study drug shows superiority in action over the control. The drugs present in the Mahatiktaka yoga seems to be contributing to the efficacy. From the outcome, it seems that there is better efficacy for the study drug, in the symptoms of the GSRS score, in relation with the intestinal symptoms, when compared with the control drug.

The symptoms without significance after the completion of the intervention were heart burn, acid regurgitation, nausea/ vomiting and abdominal distension. The symptom heartburn and acid regurgitation showed minimal significance on the follow-up, which indicates that the study drug is maintaining the result, in a better manner, throughout the follow-up period, when compared with the control. There was no significant change in the symptom of the nausea and the abdominal distension on the follow-up assessment as well. But there was significant difference between the groups on the first assessment, in all the four symptoms mentioned above indicating to the better performance of the study drug in the earlier stages of the therapy.

On the symptom of abdominal pain, borborygymi and eructation, the high significance attained in the first assessment, was not maintained later. The significance

lessened at the end of the medication ie. at the second assessment and it was not significant after the follow up.

Nausea and abdominal distension were the symptoms without significance after the medication and also at the follow-up. There was significant difference in these two symptoms on the first assessment.

In the total score of the GSRS, it was observed that it was not significant on the first assessment. The significance was at 1% level on the 2nd assessment. During the 3rd assessment, it was observed that there was a highly significant difference between the groups. These point to the fact that, in the initial stages, both of the drugs have no noticeable difference, in the action. The control have better performance in the second half, but is not maintained as such in follow up. As the therapy goes on, the study drug performs better and is maintaining it well also in follow up period. This is the reason for the increase in significance throughout.

Level of significance of efficacy on the Amlapitta rating scale

The level of significance was high at all the assessments in the symptom of soola, as per the Amlapitta rating scale. It is the only symptom among these, which shows high significance at all the levels. Amlodgara was also significant on all the three assessments, but the significance level reduced on the follow-up (1% level) when compared with the other two assessments.

The symptoms of daha and avipaka were significant at the end of the medication, but not after the follow-up. The symptom daha had a highly significant difference at the end of the therapy, which was not maintained afterwards. The relief attained on chardi was neither significant after the treatment nor the follow-up period,

but there was significance in this symptom on the first assessment indicating the faster action of the study drug, on the Chardi.

On the total score of the Amlapitta rating scale, there was significance at 1% level on the first assessment, but the significance came to minimum after the follow-up period. There was no significant difference between the two groups after the completion of the intervention, on efficacy. The study drug is performing faster in the earlier phase and also maintaining the attained efficacy, through follow up. On the completion of the treatment, both were equal in efficacy.

There was slight difference between the results in the two scales due to the following reasons. There were only five symptoms and their total in the Amlapitta rating scale, instead of fifteen in the GSRS, along with the total score. The grading was from 0 to 4 in the Amlapitta rating scale, but it was from 1 to 7 in the GSRS.

Test for association between various factors on GSRS

The various factors that affect the condition of Amlapitta were assessed through the test of association with the chi square test. The observations recorded according to the GSRS score was used for testing the association. The median score of both the groups were computed as 32 and it was taken for comparison.

Among the six rasas, those having significant association with the GSRS score were computed. It was observed that, the madhura, amla, lavana and katu rasa in excess were having significant association with the GSRS score, in the subjects of this study. The high Odds ratio calculated of these rasas (>2), ie. katu, amla and lavana is an indicative that those who are indulging more on these rasas in diet, is further prone to FD. Tikta rasa and kashaya rasa seems to have no significant association in this regard.

The amla, lavana and katu rasas are having contribution to the manifestation of Amlapitta, as per several studies, as they result in the aggravation of Pitta. In this study, it was also observed that madhura rasa is also contributory. Eventhough the madhura rasa is not considered as a prime etiological factor, it leads to agnimandya and hence vitiation of Kapha or ama, which leads to dyspepsia like conditions of Kaphaja in nature. The symptoms like hrillasa, gourava, avipaka and klama of Amlapitta are very much related with Kapha. Madhura rasa also is to be restricted in the early stages of Amlapitta, where there is association of ama along with Pitta.

It was observed that there was no significant association between the excessive use of snigdha ahara as well as the use of ushna guna in ahara with the GSRS score.

The main alterations in the status of agni was assessed among the subjects in the study. How the alteration of the agni is affecting those with the dyspepsia was assessed, by the test for association. In those with mandagni and their GSRS score, the association with the two factors was not significant. In those with vishamagni, it was found that, there was no significant association as well. The computed Odds ratio was also supportive of the same.

Most of the included subjects were having the koshta as kroora. It seems very much along with those with the FD, in the clinical presentation. The association between the kroora koshta and the score was studied and found that there was no significant association between the two, in the study, even though Odds ratio was 1.5.

Among all the subjects assessed, the majority were of Vatha Pitta prakrithi and the Vatha Kapha prakrithi. In Vatha Pitta prakrithi, it was observed that, there was minimal significant association between it and the GSRS . It can be concluded that there is more chance of presenting FD in a Vatha Pitta prakrithi person. In the

Vatha Kapha prakrithi, it was noticed that there was no significant association with the GSRS score pattern. The Odds ratio was also supportive in this regard. In the manasa prakrithi, most were of rajasa prakrithi in nature, on assessment. So the association was also studied with the GSRS score. It was observed that, there was no significant association between rajasa prakrithi and the dyspeptic score, to formulate a conclusion.

It was assessed whether in those with the intake of refrigerated food frequently were having ascent in GSRS score by test of association, it was found as negative. Many of the subjects were skipping meals frequently, but on assessment, it was found that there was no significant association, with the GSRS score.

Many of the subjects were having their food in an unsound atmosphere. In the classics of Ayurveda, it is mentioned that the presence of mind is very much essential for the proper digestion of food. Along with that, the psychological factors are having utmost ability to affect the digestion, says Acharya Charaka. On studying, it was found that there was no significant association between the food taken in an unsound atmosphere and GSRS score, in this study population.

Food intake after feeling appetite is one of the qualities of ideal food as mentioned and not followed as such today, by the society. In this study also, the association between the food intake after having appetite and the GSRS score was observed and found to be not significant.

Irregular timing of meals is one of the commonest habits nowadays. It is one of the contributory causes for the dyspeptic symptoms. In this study the association between irregular meals and GSRS score was studied and found to be highly significant. This indicates the role of irregular timings in FD and its importance in management.

Many of the subjects were having processed food frequently and the

calculated Odds ratio was 1.9. It was found that the association was significant at 5% level, pointing to the role of the same, in the manifestation. Similarly due to the hectic schedule of their work, many were having the food, which was prepared so earlier. Those working abroad were having the habit of preparing food items for a week, making it warm and having it, when required due to their inconvenience. The association between the same and the GSRS score was also studied. It was found that there was highly significant association between stale food and the GSRS score. The ahara of paryooshita in nature is causing aggravation of all the three doshas, opines Acharya Vaghbata.

Many of the subjects included were having the habit of regular intake of carbonated drinks and the association was not significant between the same and GSRS score. It was also assessed that the quantity of food intake is having any sort of relation with the symptoms of dyspepsia. It was observed that there is no significant association between these two.

The dietary habits have been changed enormously within the last few years. People are using much non-vegetarian food than the previous period. In this study also many of the subjects were having mixed food. The computed Odds ratio in this regard was more than 4, indicating to the increased manifestation with the mixed diet. On testing the association, it was found that it was significant, pointing out to its magnitude. The compatibility of food is also having a key role in the dietary habits. On studying the association between the habit of regular intake of non compatible food and the GSRS score, it was not significant.

Physical exertion after food intake was noticed in several subjects in the study and hence its association was computed, with the GSRS score. There was also

a high Odds ratio as well. It was found that the association between the two is highly significant as expected. Similarly, mental exertion and its association were also worked out. The Odds ratio was about 4 and it was observed as highly significant and have to be considered, as a very prominent role. Now a days, all are having the habit of travelling excessively. This is definitely affecting the food habits and timings and hence also creating the digestive problems. In this study, such an association was being tried to trace out and concluded that, there was real significance.

Similar is the case of the habits of sleep as well. People are going to sleep very late and the early rising habits also have been reversed. The technological advancements and also the life style are contributory here. Likewise is the timing of food habits. It was studied whether there is any association between the alterations in the habits of sleeping and F D and observed that, it was not significant in this population.

Family history is one of the contributory factors in almost all the diseases including the Functional Dyspepsia. In this study, the association between those with a positive family history and the nature of the symptoms, were studied. It was found that there was no significant association between the two factors.

In those with the routine use of drugs like NSAID's, dyspepsia is being reported more, according to published studies. The Odds ratio was more than 2. It was observed that there was significant association between the frequent NSAID use and the GSRS. It is really pointing to the role of such drugs, in the manifestation of FD. Even the continuous use of kwatham for a certain period, with the preservative, is leading to the condition in many. Similarly the fast way of life has lead to the people using most of the food items with preservatives, as they are purchasing in a prepared manner, resulting in gastric disturbances.

Many female subjects included in the study were had attained menopause. For a matter of curiosity, it was observed whether there is any peculiarity in the dyspeptic scores in those, after menopause. Even though the calculated Odds ratio was 2, it was observed that there was no significant association in between.

Of all the categorized occupations, the most dominant were those performing a job with physical exertion, like casual laborers. Their level of GSRS was compared for the association. It was observed that there was no significant association between this type of job and the GSRS.

Similarly many of the subjects included were the home managers. They were also studied for the association as the Odds was more than 2. It was noticed that there was significant association between these group and the GSRS score. It was observed that the stress level in home managers are more than employed women. It can be concluded that the untimely food and the stressful life situations faced by them, is contributing to dyspepsia.

These factors are pointing to the contributing factors of the disease, and are very helpful for the aspect of the management. Among these the highly significant ones need special mentioning as, the physical exertion after intake of the food, mental exertion and the habit of intake the previously prepared food. Next in significance is the habit of excessive travelling which has influence on the dietary habits and the quality of food. All the other factors are also to be considered accordingly.

H Pylori

H pylorus is considered to have a key role in many of the reported dyspepsia. The role played by the organism in this condition is well studied worldwide[18]. It was studied, whether there is any association between the subjects positive with H

pylori and the severity of GSRS score, in this population. Here the calculated Odds ratio was 1.3 in this regard. On testing the statistical association between those with RUT positive for H pylori and the severity of FD as GSRS score, it was observed that the chi square value obtained was 0.456 and it was not significant. It was recorded that there was no significant association between the two factors in the case of FD, in this peculiar study.

Comparison of the efficacy of groups on H Pylori

In this study also, even though it was not listed among the objectives, those positive with H pylori on endoscopy, with the Rapid Urease Test (RUT), was also assessed for efficacy, after the intervention on IgG H pylori. Among the 100 subjects included in the trial group, 25 were positive for H pylori with RUT. 2 months after the intervention, on testing with the Immunocomb II Helicobacter pylori IgG kit, 19 were only positive and the rest 6 became negative[19]. In the control group, 18 were positive with RUT and after 2 months of the therapy, 16 were positive with IgG H pylori test, so the rest 2 became negative.

Even though it was seen in less number of patients in both groups, the results were assessed statistically using the unpaired t test with unequal sample. It was observed that, there was significant efficacy for the trial group against the control on the H pylori at 1% level. The action of the study drug in the condition of Functional Dyspepsia may be also due to the effectiveness of the drug against H pylori. Many drugs in the Mahatiktaka combination like yashti, darvi, vacha etc. are proven to have anti- H pylori action, as per reported trials[20]. Even though it cannot be generalized or concluded with this, we can plan for further studies with the same drug in an entirely positive population.

Correlation Analysis

In the correlation study conducted regarding the various factors of Functional Dyspepsia, it was observed that katu, amla and lavana rasa showed a positive correlation with the GSRS. These are the main rasas involved in the pathogenesis of the diseases, resembling Amlapitta.

A positive correlation was also noticed in the female gender, home makers, kroora koshta, job with physical exertion, job with mental exertion and irregular timing of food. These support the test for association, which had been explained earlier.

Female are more prone to stress and much anxious and are very much irregular in their food habits, which contributes to the observation. The irregularities in the bowel habits are always seem to aggravate the presentation. Similarly exertion at both the physical and the mental level is really contributive to diseases like FD. Similarly is the case of intake of meals not on proper time.

Regression Analysis

On the regression analysis done with the various factors contributing to the GSRS score, it was observed that the R square value was 0.25, which suggests that the GSRS score is contributed by 25% due to the studied variables. The succeeding ANOVA was highly significant, which points to the contribution of the variables in the manifestation.

On studying the individual variables, the physical exertion showed significance at 0.1% level, mental exertion showed a significance at 1% level and the irregular timing of meals showed significance at 5% level. These three can be considered as the most contributory among all the variables in this study, in the pathogenesis.

On studying the role of rasas, there was a positive regression coefficient pointing to the contribution of the amla, lavana and katu and it was highly significant on F test. While considering individually, lavana and katu was highly significant at 1% level, but amla at a minimal level.

Percentage of relief on GSRS

The percentage of relief observed after the intervention was analyzed between the groups. The whole subjects was graded into 4 groups, according to the relief observed. It was compared at two levels, after the therapy and after the follow-up period.

In the study group, after the therapy, 60% were slightly improved while more than 30% moderately improved. In the control group, almost 90% slightly improved and less than 6% moderately improved. After the follow up, in the study group 88% of the subjects were moderately improved while in control, 60% were having mild improvement and more than 40% moderately improved. This shows that the study drug is having efficacy which is not only maintaining, but also improving, throughout the follow-up period as well. The control drug responded well early but the rate of response was not up to the level of the study drug later. The control drug also maintained and improved the efficacy over the follow-up period but not to the extent of study drug. Every subject got a minimal response in the control group as there was no one unchanged one, unlike the study drug.

Percentage of relief on Amlapitta rating scale

In the study group, after the therapy, almost 60% were improved moderately while in control, 66% improved slightly only. On the follow up, 87% improved moderately in trial while upto 60% improved moderately in control. The study drug is having efficacy which is not only maintaining, but also improving throughout

the follow-up period. Initially the response was a bit more in the control group, but at the end of the intervention,the study drug shows better percentage after follow-up.

Mode of action of the drug

The characteristics of the Mahatiktaka yoga by its pharmacological properties individually were studied. The individual drugs of the Mahatiktaka yoga are having mainly tikta in rasa. The next rasas in dominance are kashaya, katu and madhura rasas. The combination is having the slight dominance of seeta veerya. The yoga is rooksha in guna as well as laghu, peculiarly when it is in the form of kwatha. The vipaka is mainly katu and to an extent, madhura.

The combination as a whole seems Pitta kaphahara in action as 56% of the drugs seems resemblance in action, accordingly. According to Kashyapa, the drugs most useful in the management of Amlapitta must be Kapha Pittahara in action. Also the apt as well as sodhana karma mentioned is also Vamana as the amasaya is the sthana of Kapha. This point to the significance of the Kaphahara drugs in the management. Eventhough the disease seems Paittika in nature as well as the presentation, the better management seems Kapha Pittahara, as the Pitta is mostly associated with ama ie. sama Pitta. For sama pitta, the treatment should be samana in nature, both to Kapha and Pitta and the Mahatiktaka yoga contributes in this regard. Amlapitta is one of the indications mentioned of the Mahatiktaka yoga, by Charaka.

A clinical study conducted by Snigdha etal. concluded that the Mahatiktaka Kwatha when administered in hyperglycemia associated stress, was effective in controlling the stress as per the Stress Assessment Questionnaire and also the blood sugar level. The anxiolytic activity of the drug was comparable with the benzodiazepine as per the study. This is very much contributory to the role of Mahatiktakam kwatham

in the management of FD, as the psychological factors are a great deal in contributory to the condition.

This is proved by the Mahatiktaka yoga by its efficacy, in this particular study. When administered as kwatha or kwatham tablet form, the combination as a whole works as Kaphapittahara as mentioned above. The drug when processed in the form of gritha ie. Mahatiktaka gritha becomes much more Vathahara in action as reported by the study on parinama soola, conducted by the CCRAS. It is good for chronic conditions and in cases with the considerable involvement of the Vatha. But the patient must be fit and ideal for snehana. If otherwise, we have to go for the kwatha or the tablet form.

While analyzing the individual symptoms on the basis of the three doshas, it was found that strict generalization is not possible. Among the Vathika symptoms, there was significant improvement in increased flatus, decreased stool, hard stool and the urgency of defecation. There was no significant difference in the efficacy in abdominal pain, abdominal distension and eructation. These symptoms are seemed to have much more response in the clinical level, the combination being administered in the gritha form and on attaining better anulomana.

There was significant response in both the symptoms, which are Paittika in nature, the heart burn as well as the loose stools. In the symptoms resulting from the Kapha dosha, it was observed that there was significant response in the sucking sensation and also the feeling of incomplete evacuation. But the symptom of nausea/ vomiting didn't respond significantly. The study drug seems to have better action as far as the lower GI symptoms are concerned, in comparison with the upper tract.

The mode of administration of the Mahatiktakam kwatham tablet seems

very comfortable for the subjects. The lack of preservatives in the combination is very much positive, as far as the dyspeptic features are concerned. On the subjects who are taking the prepared kwatha continuously, the preservatives are creating disturbances, peculiarly on the continuous use. Here they feel so much comfortable and an adequate dose can be administered as there is no matter of dilution. The palatability also is highly appreciable as compared to the kwatha. Many of the subjects were having regular travelling as a part of their profession and the medicine in the tablet form added to their comfort and also the regular dosage of the same as preferred by them. The shelf life of the kwatham tablet is more, when considered with the same kwatha. We can also propose studies with blinding method with the kwatham tablets.

Dropouts

There were 10 dropouts throughout the study, 5 each in the control group and the study group. Of these 5 subjects migrated to other countries as a part of their job, without completion of the intervention. Two people affected with episodes of fever during the treatment and stopped the drug in between. One got an attack of diarrhea and was discontinued. One discontinued person complained of the difficulty in swallowing of the study drug and stopped the same. One person had an attack of bronchial asthma in between, was admitted and discontinued the medicine. It seems that none had discontinued the medicine due to any relative adverse reactions of the drug. This indicates to the safe administration of Mahatiktakam kwatham tablet in the subjects.

SUMMARY

Functional Dyspepsia is one of the commonest clinical conditions in a gastroenterology OPD, affecting up to one fourth of the population in India. A variety of pathophysiologic mechanisms like dietary misconduct, motility disturbances, alteration in visceral sensitivity, psychological factors, H pylori etc. have been proposed to elucidate the symptoms of Functional Dyspepsia. Due to the varied pathogenic factors, in the management also several drugs are being tried out with varied efficacy.

In Ayurveda, the disease is being approached with the management protocol mentioned for the disease, Amlapitta. The disease had been dealt in detail in the treatises like Kasyapa samhitha and Madhava nidana. Here even though the core dosha seems Pitta, the protocol is to be adopted considering the associative dosha, which is Kapha in the initial stages and Vatha in the later stages of the disease. Hence a drug like Mahatiktaka which is a Pitta Kapha samana combination, is one of the options for the samana chikitsa, for the condition. The scope of sodhana chikitsa and satvavachaya chikitsa is also being discussed here in this regard.

Clinical study

Of the various drugs used successfully by the Ayurvedic fraternity, the Mahatiktaka yoga mentioned by Acharya Charaka was selected for the study due to two reasons, Amlapitta is one of the indications mentioned and also it seems effective, in the clinical scenario. It is also a drug with proven psychological efficiency and also is krimihara in action. Mahatiktaka was prepared in the form of kwatham tablet and used for the study.

The Allopathic counterpart, the PPI group of drug Omeprazole was used as control drug for the study. It was a Randomized Controlled Trial with the setting as the OPD and IPD of Vaidyaratnam P S Varier Ayurveda College, Kottakkal with the inclusion of 200 subjects, those fit with the proposed criteria and also furnishing written consent. The period of administration was 30 days continuously. The observations were analyzed according to GSRS score and the Amlapitta rating scale.

The overall efficacy of the therapy was significant at 1% level after the intervention and 0.1% level at the follow-up, on the GSRS score. As per the Amlapitta rating scale, there was no significant difference between the groups after the intervention, but on the follow-up, there was statistical significance at the 5% level.

The individual symptoms of abdominal pain, borborygymi, sucking sensation, increased flatus, decreased stool, loose stool, hard stool, urgency of defecation and the feeling of incomplete evacuation showed statistical significance between the groups, after the intervention. There was no significant difference in improvement between the groups in the heart burn, abdominal distension, nausea and acid regurgitation.

In the Amlapitta rating scale, there was significant difference between the

groups on daha, soola, avipaka and amlodgara while there was no significant difference in the symptom of chardi between the groups, after the intervention. After the follow-up period, the symptom of daha and amlodgara showed significant difference between the groups. The observation after the follow-up period shows that, the study drug is able to maintain the attained efficacy later.

Significant association was observed between the GSRS score and the contributory factors of Functional Dyspepsia like mental exertion, irregular food timings, excessive use of katu, amla, lavana rasas, processed food, physical exertion after food intake etc. Those having regular mixed dietary habits, excessive travelling, frequent use of NSAID's, Vatha Pitta prakrithi etc. were prone to the manifestation, as per the observations. In the selected subjects, the study drug showed statistically significant efficacy on H pylori, when compared with the control.

The drug administration can be considered as safe as there was no observable adverse effects recorded throughout the study.

CONCLUSION

The study entitled "A Randomized Controlled Clinical Trial to assess the role of Mahatiktakam Kwatham Tablet in Amlapitta w.s.r to Functional Dyspepsia" has two objectives.

The study drug Mahatiktaka gritha was studied to assess the role in Functional Dyspepsia. The study shows that there is significant efficacy of the drug on Functional Dyspepsia according to the GSRS score, at all the levels of the assessment. ($P<0.001$)

The efficacy of the study drug Mahatiktaka Kwatham tablet was compared with the control drug, Omeprazole in Functional Dyspepsia regarding its efficacy. It was observed that the overall efficacy of the therapy was significant at 1% level after the intervention and 0.1% level after the follow-up on the GSRS scale, when compared between the groups.

It was also observed that with the Amlapitta rating scale, there was no

significant difference between the groups after the intervention, but on the follow-up there was significance at 5% level.

The study drug was also having significant efficacy on H Pylori when compared with the control in selected subjects.

It can be concluded that the alternate hypothesis is accepted after the study. ie.

There is significant difference in the role of Mahatiktakam kwatham Tablet when compared with Omeprazole in Amlapitta w.s.r. to Functional dyspepsia.

Mahatiktaka kwatham tablet is effective in the management of Functional Dyspepsia and also safe and improves the quality of life of those affected with the disease.

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Chapter VII - Discussion, Summary and Conclusion

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APPENDICES

CASE RECORD FORM

TITLE **A Randomized Controlled clinical Trial to assess the role of Mahatiktakam kwatham Tablet in Amlapitta w.s.r to Functional Dyspepsia**

Scholar	:	**Vd Jithesh M**
Guide	:	Dr Renuka R Gayal
Institute	:	VPSV Ayurveda college , Kottakkal
University	:	Tilak Maharashtra Vidyapeeth, Pune

Name	:				
Case No	:				
OPD No	:			Date :	
Age	:		Sex	:	☐ M ☐ F
Group	:	A / B			
Religion	:	☐ Hindu	☐ Muslim	☐ Christian	☐ Others
Marital status	:	☐ Unmarried	☐ Married	☐ Divorced	☐ Widow
Address	:				
Telephone No.	:				
E mail ID	:				
Economic status	:	☐ Poor	☐ Middle class	☐ Rich	
Education	:	☐ Illiterate	☐ Primary	☐ Secondary	
		☐ Undergraduate	☐ Graduate	☐ Post graduate	
Occupation	:	☐ Home manager	☐ Job with physical exertion	☐ Professional	
		☐ Student	☐ Business	☐ Clerical	
		Height: ……. cms		Weight: ……... Kgs	
Addiction	:	☐ smoking	☐ alcohol	☐ others	
If yes, details	:				

Presenting complaints with duration

Symptom	**Duration (months)**	**Details**
Abdominal pain		
Fullness of abdomen		
Sucking sensation in epigastrium		
Belching		
Acid eructation		
Heart burn		
Nausea / vomiting		

Anorexia
Borborygymi
Increased flatus
Diarrhoea / constipation
Others

Past History

☐ DM ☐ HT ☐ Dyslipidaemia ☐ thyroid disorder ☐ others

Details :

Treatment History

Any continuous medication :
Regular use of NSAID's :
Recent Antibiotics intake if any :
Corticosteroids use :
Previous surgeries :
Occupational history :

☐ Sedentary ☐ physical exertion ☐ mental exertion
☐ Excessive travelling ☐ skipping meals ☐ irregular sleep pattern
Others :

Personal history :

Agni : ☐ sama ☐ vishama ☐ manda ☐ teekshna
Koshta : ☐ mridu ☐ madhyama ☐ kroora
Aahara : ☐ Veg ☐ Non- veg ☐ mixed
Rasa pradhanya
: ☐ Ma ☐ Aml ☐ La ☐ Kat
☐ Tikt ☐ Kash
Timings : ☐ regular ☐ irregular
Habits : a) smoking …… / day since……..
b) Alcohol ……. / day/ week since……..
c) Tobacco ……. / day since……..
d) Carbonated drinks ……/ day
Mootra : frequency ……/ day, ……. / night
Pureesha: ☐ saama ☐ niraama ☐ krichra
☐ badha ☐ drava
Pipaasa : ☐ less ☐ normal ☐ excessive
Sweda : ☐ less ☐ normal ☐ excessive
Nidra : …….. hr/ day, ……. hr/ night
: ☐ less ☐ interrupted ☐ normal ☐ more
☐ daytime sleeping

Menstrual history : ☐ Regular ☐ irregular cycle
Abnormalities if any –

Family history : ☐ yes ☐ no

Rogi pareeksha

Prakrithi : Sareerika V / P / K / VP / VK / PK / tridoshaja
Manasika – satwa / raja / tama

Vikrithi : Dosha – V / P / K / VP / VK / PK / tridoshaja

: Dooshya – Rasa/ Rakta/ Mamsa/ Medas/ Asthi/ Majja/ Sukra

Desa – jangala / anupa / sadharana

Saara : ☐ Pravara ☐ Madhyama ☐ Avara

Samhana : ☐ Pravara ☐ Madhyama ☐ Avara

Pramaana : ☐ Pravara ☐ Madhyama ☐ Avara
Ht ……. Cm, Wt…… Kg

Satmya : ☐ Pravara ☐ Madhyama ☐ Avara

Satwa : ☐ Pravara ☐ Madhyama ☐ Avara

Aaharasakti : Abyavaharana – ☐ Pravara ☐ Madhyama ☐ Avara

: Jarana – ☐ Pravara ☐ Madhyama ☐ Avara

Vyayamasakti : ☐ Uthama ☐ Madhyama ☐ Avara

Vaya : ☐ balya ☐ youvana ☐ vardhakya

GENERAL EXAMINATION

Pulse rate : ……./ min

Respiratory rate : ……../min

Heart rate : ……./ min

BP : ……./…….. mm Hg

Temperature : …….^{0}F

Systemic Examination

Cardiovascular :

Respiratory :

Nervous :

Urinary :

Locomotor :

Examination of G I System

Inspection

Shape of abdomen :

Abdominal wall : ☐ surgical scar ☐ distended veins ☐ striae
☐ peristalsis

Movement with respiration ☐ present ☐ absent

Umbilicus : ☐ everted ☐ inverted ☐ normal
☐ hernia

Pulsation : ☐ present ☐ absent

PALPATION

Feel : ☐ soft ☐ rigid ☐ tender

Tenderness : ☐ present ☐ absent

Areas of elicitable tenderness

☐Rt Hypochondrium ☐ Epigastrium ☐ Lt. Hypochondrium

☐Rt.Lumbar ☐ Umbilical ☐ Lt. Lumbar

☐Rt. Inguinal ☐ Hypogastrium ☐ Lt. inguinal

Organomegaly if any, details

Palpable mass :

PERCUSSION

Note : ☐ tympanitic ☐ resonant ☐ dull

Shifting dullness :

Fluid thrill :

AUSCULTATION

Peristaltic movements: ☐ present ☐ absent

Peritoneal rub : ☐ present ☐ absent

Arterial bruit : ☐ present ☐ absent

Investigations

Routine blood examination

Hb% TC DC -

ESR - mm/I Hr

Blood sugar Random :

Total cholesterol :

LFT :

Upper GI Endoscopy findings

Test for H pylori : ☐ Positive ☐ Negative ☐ Not done

BT AT

Details :

If positive, result of post test with date:

Aahara vidhi

TYPES OF FOOD HABITS

Freshly prepared food	☐ Yes	☐ No
Refrigerated and warmed	☐ Yes	☐ No
Quality of food, Snigdha/ rooksha, spicy etc	☐ Yes	☐ No
Quantity of intake	☐ Yes	☐ No
Intake after gaining appetite	☐ Yes	☐ No

No. of meals per day	☐ Yes	☐ No
Skipping of meals	☐ Yes	☐ No
Intake too slow/ too hurry	☐ Yes	☐ No
Intake of incompatible food	☐ Yes	☐ No
Taking processed /junk food	☐ Yes	☐ No
Intake in calm atmosphere	☐ Yes	☐ No

Diagnostic criteria for Amlapitta

Symptom	**Present**	**absent**	**Duration**
Daha			
Amlodgara			
Chardi			
Shoola			
Avipaka			

Atleast 3 symptoms from more than 3 months selected

Rome III criteria for functional dyspepsia

Symptom	**Present**	**absent**	**Duration**
Bothersome postprandial fullness			
Early satiation			
Epigastric pain			
Epigastric burning			

Criteria fulfilled for last three months selected

ASSESSMENT TOOLS

GSRS rating scale

Symptom	**Score (BT)**	**15 days**	**30 days**	**45 days**
Abdominal pain				
Heart burn				
Acid regurgitation				
Sucking sensation				
Nausea/vomiting				
Borborygymi				
Abdominal distension				
Eructation				
Increased flatus				
Decreased stool				

	BT	15 days	30 days	45 days
Increased stool				
Loose stool				
Hard stool				
Urgent need for defecation				
Feeling of incomplete evacuation				
Total Score				

The scoring will be done from 0 to 7 as shown below

1. No problem
2. Minimal problem (can be easily ignored without effort)
3. Mild problem (can be ignored with effort)
4. Moderate problem (cannot be ignored but does not influence my daily activities)
5. Moderately severe problem (cannot be ignored and occasionally limits my daily activities)
6. Severe problem (cannot be ignored and often limits my concentration on daily activities)
7. Very severe problem (cannot be ignored and markedly limits my daily activities and often requires rest)

A reduction of 50% or more in the total score will be considered as improved

Amlapitta symptoms score

Lakshana	**15 days**	**30 days**	**45 days**
Daha			
Chardi			
Soola			
Avipaka			
Amlodgara			
Total Score			

All the symptoms will be graded from 0 to 4

The difference in the scores will be considered for the improvement and graded as shown below.

- Marked improvement 76 % to 100 %
- Moderate improvement 51 % to 75 %
- Mild Improvement 25 % to 50 %
- Unchanged < 25 %

Signature of guide ***Signature of Scholar***

Gastrointestinal Symptom Rating Scale (GSRS)

Name :

A rating scale for the gastrointestinal symptoms in patients with irritable bowel syndrome and the peptic ulcer disease. Circle the number which best represents the current severity of the symptom.

1. Abdominal pains- Representing subjectively experienced bodily discomfort, aches and pains.

The type of pain may be classified according to the patient's description of the appearance and quality of the pain as epigastric, on the basis of typical location, association with acid-related symptoms, and relief of pain by food or antacids; as colicky when occurring in bouts, usually with a high intensity, and located in the lower abdomen; and as dull when continuous, often for several hours, with moderate intensity. Rate according to intensity, frequency, duration, request for relief, and impact on social performance.

0 No or transient pain
1 Occasional aches and pains interfering with some social activities
2 Prolonged and troublesome aches and pains causing requests for relief and interfering with many social activities
3 Severe or crippling pains with impact on all social activities

2. Heartburn. Representing retrosternal discomfort or burning sensations. Rate according to intensity, frequency, duration, and request for relief.

0 No or transient heartburn
1 Occasional discomfort of short duration
2 Frequent episodes of prolonged discomfort; requests for relief
3 Continuous discomfort with only transient relief by antacids

3. Acid regurgitation. Representing sudden regurgitation of acid gastric content. Rate according to intensity, frequency, and request for relief.

0 No or transient regurgitation
1 Occasional troublesome regurgitation
2 Regurgitation once or twice a day; requests for relief
3 Regurgitation several times a day; only transient and insignificant relief by antacids

4. Sucking sensations in the epigastrium. Representing a sucking sensation in the epigastriumwith relief by food or antacids. If food or antacids are not available, the sucking sensations progress to ache, and pains. Rate according to intensity, frequency, duration, and request for relief.

0 No or transient sucking sensation
1 Occasional discomfort of short duration; no requests for food or antacids between meals
2 Frequent episodes of prolonged discomfort, requests for food and antacids between meals
3 Continuous discomfort; frequent requests for food or antacids between meals

5. Nausea and vomiting. Representing nausea which may increase to vomiting. Rate according to intensity, frequency, and duration.
0 No nausea
1 Occasional episodes of short duration
2 Frequent and prolonged nausea; no vomiting
3 Continuous nausea; frequent vomiting

6. Borborygmus. Representing reports of abdominal rumbling. Rate according to intensity, frequency, duration, and impact on social performance
0 No or transient borborygmus
1 Occasional troublesome borborygmus of short duration
2 Frequent and prolonged episodes which can be mastered by moving without impairing social performance
3 Continuous borborygmus severely interfering with social performance

7. Abdominal distension. Representing bloating with abdominal gas. Rate according to intensity, frequency, duration, and impact on social performance.
0 No or transient distension
1 Occasional discomfort of short duration
2 Frequent and prolonged episodes which can be mastered by adjusting the clothing
3 Continuous discomfort seriously interfering with social performance

8. Eructation. Representing reports of belching. Rate according to intensity, frequency, and impact on social performance.
0 No or transient eructation
1 Occasional troublesome eructation
2 Frequent episodes interfering with some social activities
3 Frequent episodes seriously interfering with social performance

9. Increased flatus. Representing reports of excessive wind. Rate according to intensity, frequency, duration, and impact on social performance
0 No increased flatus
1 Occasional discomfort of short duration
2 Frequent and prolonged episodes interfering with some social activities
3 Frequent episodes seriously interfering with social performance

10. **Decreased passage of stools.** Representing reported reduced defecation. Rate according to frequency. Distinguish from consistency.
    0 Once a day
    1 Every third day
    2 Every fifth day
    3 Every seventh day or less frequently
11. **Increased passage of stools.** Representing reported increased defecation. Rate according to frequency. Distinguish from consistency.
    0 Once a day
    1 Three times a day
    2 Five times a day
    3 Seven times a day or more frequently
12. **Loose stools.** Representing reported loose stools. Rate according to consistency independent of frequency and feelings of incomplete evacuation.
    0 Normal consistency
    1 Somewhat loose
    2 Runny
    3 Watery
13. **Hard Stools.** Representing reported hard stools. Rate according to consistency independent of frequency and feelings of incomplete evacuation.
    0 Normal consistency
    1 Somewhat hard
    2 Hard
    3 Hard and fragmented, sometimes in combination with diarrhea
14. **Urgent need for defecation.** Representing reports of urgent need for defecation, feelings of incomplete control, and inability to control defecation. Rate according to intensity, frequency, and impact on social performance.
    0 Normal control
    1 Occasional feelings of urgent need for defecation
    2 Frequent feelings of urgent need for defecation with sudden need for a toilet interfering with social performance
    3 Inability to control defecation
15. **Feeling of incomplete evacuation.** Representing reports of defecation, with straining and a feeling of incomplete evacuation of stools. Rate according to intensity and frequency.
    0 Feeling of complete evacuation without straining
    1 Defecation somewhat difficult; occasional feelings of incomplete evacuation
    2 Defecation definitely difficult; often feelings of incomplete evacuation
    3 Defecation extremely difficult; regular feelings of incomplete evacuation

Modified with Seven point Likert scale.

0 None
1 Minimal
2 Mild
3 Moderate
4 Moderately severe
5 Severe
6 Very severe

Scoring for Amlapitha

The effect of the drug under trial was based mainly on the improvement in the cardinal signs and symptoms of the disease. To give some objectivity, the score was assigned to each of the major symptoms of the disease like Daha, Amolodgara, Shula, chardi and Avipaka. Similarly other symptoms were also given scores on the basis of this before and after the treatment score.

Scoring Pattern :-

DAHA :-

0- No Daha
1- Daha of mild degree in any area of kantha, udara, uras, kukshi
2- Daha of moderate degree relieves by antacid, milk, cool drink, ice-cream
3- Daha of severe degree and relived after digestion of food, or vomiting
4- Severe degree of Daha but relives by any measure mentioned above
5- Severe degree of Daha and does not get relief by any measure

CHARDI

0. No vomiting
1. Nausea and vomits occasionally
2. Frequency is not more than 2 to 3 per week
3. Frequency of vomiting is between 4 to 6 week
4. Frequency of vomiting is daily

SHULA

0. No pain
1. Slight pain which need not any medicine
2. Pain of some degree which subsides after taking cold, sweet, food, antacid, milk etc
3. Severe colicky unbearable pain but relieves after vomiting or relieves after digestion of food
4. Severe unbearable pain which does not subsides by either vomiting or antacids.

AVIPAKA

0. No avipaka
1. Avipaka occurs occasionally 2 - 3 times per week
2. Avipaka occurs daily but not severe
3. More than 2-3 ajirna ahara lakshanas like ashuddha udgara/anutsaha/glani present
4. Severe type of avipaka which does not subside without medicine

AMLODGARA

0. No Amlodgara
1. Sometimes during day
2. Amlodgara of moderate severity
3. Severe Amlodgara disturbing the patients
4. Small amount of fluid regurgitate to patients mouth

Effect on Agni and associated symptoms :-

(1) All the symptoms were given '2' scores to each if found present at all
(2) If any improvement in the symptom was noticed then it was given '1' score.
(3) If the symptom was found absent then it was given '0' score.
(4) This assessment was done before and after the treatment in both the groups.

Total assessment of the therapies was done on the basis of the relief in the main signs and symptoms of disease. Agnidusti and also the general signs and symptoms of disease. On the basis of these criteria total patients were divided in five categories as below.

< 25%	-	unchanged
26% - 50%	-	slight improvement
51% - 75%	-	moderate improvement
76% - 100 %	-	marked improvement

VAIDYARATNAM P.S. VARIER'S
ARYA VAIDYA SALA, KOTTAKKAL
Kottakkal - 676 503, Malappuram Dist., Kerala, India. Tel: 0483 2838000, 2742216-19 Fax: 91 483 - 2742572, 2742210
E-mail: info@aryavaidyasala.com, mail@aryavaidyasala.com
Website: www.aryavaidyasala.com

TO WHOMSOEVER IT MAY CONCERN

06.08.12

This is to state that the Mahatiktam kwatham tablet supplied to Dr. Jithesh M., Associate Professor, VPSV Ayurveda College, Kottakkal was prepared as a single batch and also the raw materials used were authenticated. The Mahatiktam kwatham tablet manufactured was standardised as per in-house specifications.

Dr. T.S. Madhavankutty
Chief (R&D)

Kottakkal
ayurveda

VAIDYARATNAM P.S. VARIER'S
ARYA VAIDYA SALA, KOTTAKKAL
Kottakkal - 676 503, Malappuram Dist., Kerala, India. Tel: 0483 2808000, 2742216-19 Fax: 91 483 - 2742672, 2742210
E-mail: info@aryavaidyasala.com, mail@aryavaidyasala.com
Website: www.aryavaidyasala.com

TO WHOMSOEVER IT MAY CONCERN

17.08.12

This is to state that the in-house quality specifications of Mahatiktam kwatham tablet include the following parameters that are followed in Arya Vaidya Sala, Kottakkal.

SN	PARAMETER	UNIT	STANDARD
01	Description	-	Brownish black tablet with bitter taste
02	Average weight	g	1.15 – 1.25
03	API content	g	0.85 – 0.95
04	Hardness	kg/cm^2	NLT 6
05	Thickness	mm	6.3 – 6.5
06	Disintegration Time	minutes	NMT 15
07	Weight variation	% w/w	± 10 %
08	Friability	% w/w	NMT 1
09	HPTLC fingerprint	-	Passes

Dr. T.S. Madhavankutty
Chief (R&D)

Kottakkal
ayurveda

Criteria for assessment of Koshta

1. Frequency per day

Less than one	–	1
Once/ twice	–	2
More than two	–	3

2. Consistency

Hard stool	–	1
Soft, well formed	–	2
Loose/ watery, not well formed	–	3

3. Urgency

No urgency at all	–	1
Moderate urgency – can be controlled	–	2
Marked urgency , cannot be controlled	–	3

4. After the intake of 200 ml milk, 100 gms grapes, 200 ml ikshurasa, 10 gms avipathy choorna

No change in bowel habit	–	1
Normal well formed stool	–	2
Watery/ not well formed stool	–	3

5. Whether changes in the food habits wiil affect your bowel hablts

Frequently hard	–	1
Occasionally	–	2
Freqently loose	–	3

Assessment

1 to 5	Kroora koshta
6 to 10	Madhyama koshta
11 to 15	Mridu koshta

Assessment of Prakrithi

Features	Vatha prakrithi	Pitta prakrithi	Kapha prakrithi
Appearance	Lean, tall or short	Medium	Short, obese
Skin	Thin, dry, rough	Flushes easily, moles	Thick, moist, smooth
Hair	Dry, rough, curly	Soft, premature graying, baldness	Thick, dark, oily, smooth
Appetite, food, drink	Variable, prefer warm	Strong, prefer cool	Low, prefer dry and warm
Bowel	Constipated, dry, scanty	Normal, loose, abundant	Regular, solid, soft
Sweat , odour	Scanty, no smell	Profuse, strong smell	Moderate , no smell
Activity, gait	Unstable, unsteady	Medium	Stable
Sleep	Disturbed	Moderate	Sound
Mood	Worried	Aggressive	Relaxed
Memory	Easy to learn and forget	Moderate	Gradual and Stable

സമ്മതപത്രം

ഞാൻ ..... പൂർണ്ണ സമ്മതത്തോടുകൂടി ഈ ഗവേഷണ പരിപാടിയിൽ പങ്കെടുക്കുന്നതാണ്. ഇതേപ്പറ്റിയുള്ള പൂർണ്ണ വിവരങ്ങൾ എന്റെ മാതൃഭാഷയിൽ എന്നെ ധരിപ്പിച്ചിട്ടുള്ളതാകുന്നു.

ഈ ഗവേഷണത്തിന്റെ വിഷയം അമ്ലപിത്ത (ഫങ്ഷണൽ ഡിസ്പെപ്സ്യ) രോഗത്തിൽ മഹാതിക്തകം ക്വാഥം ടാബ്ലറ്റിന്റെ പങ്ക് (എ റാൻഡമൈസ്ഡ് കൺട്രോൾഡ് ട്രയൽ ടു അസസ്സ് ദി റോൾ ഓഫ് മഹാതിക്തകം ക്വാഥം ടാബ്ലറ്റ് ഇൻ അമ്ലപിത്ത (ഫങ്ഷണൽ ഡിസ്പെപ്സ്യ) എന്നതാണ്.

കോട്ടക്കൽ ആയുർവേദ കോളേജിലെ കായചികിത്സാ വിഭാഗത്തിൽ വെച്ച് ഡോ: രേണുകാ. ആർ. ഗയാലിന്റെ (പൂനെ) മേൽനോട്ടത്തിലാണ് ഈ പഠനം നടക്കുന്നത്.

ഈ ഗവേഷണത്തിലെ മരുന്നുകൾ മൂലം യാതൊരുവിധത്തിലുള്ള പാർശ്വഫലങ്ങളും ഉണ്ടാകുന്നതല്ല, എന്ന് എന്റെ ചികിത്സകൻ എന്നെ ബോദ്ധ്യപ്പെടുത്തിയിട്ടുണ്ട്.

ഈ ഗവേഷണത്തിനായി എന്നിൽ നിന്നും ശേഖരിക്കുന്ന വിവരങ്ങൾ രഹസ്യമായി സൂക്ഷിക്കുന്നതാണ് എന്നും എന്റെ പേരോ, മേൽവിലാസമോ, പരസ്യപ്പെടുത്തുകയോ ദുരുപയോഗപ്പെടുത്തുകയോ ചെയ്യുന്നതല്ല എന്നും എന്റെ ചികിത്സകൻ എന്നെ ബോധ്യപ്പെടുത്തിയിട്ടുണ്ട്. ഈ ഗവേഷണത്തെക്കുറിച്ചുള്ള വിശദവിവരരേഖയും ഞാൻ കൈപ്പറ്റിയിട്ടുണ്ട്.

ഈ ഗവേഷണത്തിന്റെ ഏതു ഘട്ടത്തിലും ഇതിൽ നിന്നും പിന്മാറാനുള്ള സ്വാതന്ത്ര്യം എനിക്ക് ഉണ്ടായിരിക്കുന്നതാണ്.

ഈ സമ്മത പത്രത്തിലെ ഒപ്പ് ഈ ഗവേഷണ പരിപാടിയെക്കുറിച്ചുള്ള പൂർണ്ണ വിവരങ്ങൾ മനസ്സിലാക്കി എന്നതിനും, ഇതിന്റെ ഭാഗമായുള്ള ഔഷധസേവനത്തിനും, ചികിത്സയിൽ പങ്കെടുക്കുന്നതിനും സന്നദ്ധമാണ് എന്നതിന്റെയും സൂചനയാണ്.

രോഗിയുടെ ഒപ്പ് / വിരലടയാളം

ഗവേഷകന്റെ ഒപ്പ്

സ്ഥലം

തീയ്യതി

വിശദ വിവരരേഖ

ഒ. പി. നമ്പർ :

തിയ്യതി :

1) ഗവേഷണാധിഷ്ഠിത ചികിത്സാപദ്ധതിയുടെ പേര്	:	എ റാൻഡമൈസ്ഡ് കൺട്രോൾഡ് ട്രയൽ ടു അസസ്സ് ദി റോൾ ഓഫ് മഹാതിക്തകം കാഥം ടാബ്ലറ്റ് ഇൻ അമ്ലപിത്ത (ഫങ്ഷണൽ ഡിസ്പെപ്സ്യ)
2) ഗവേഷക വൈദ്യന്റെ പേര്	:	ജിതേഷ് എം.
3) ഗവേഷക വൈദ്യന്റെ ഒപ്പ്	:	
4) ചികിത്സക്കിടയിൽ സംഭവ്യമായ മറ്റ് പ്രശ്നങ്ങൾ	:	
5) ഗവേഷക ചികിത്സാ പദ്ധതിയുടെ സംരംഭക വിഭാഗത്തിന്റെ പേര്	:	കായചികിത്സാ വിഭാഗം
6) രോഗിക്ക് ചികിത്സാ സംബന്ധമായ എന്തെങ്കിലും ബുദ്ധിമുട്ടുണ്ടെങ്കിൽ ബന്ധപ്പെടേണ്ട ആൾ	:	ഡോ. ജിതേഷ് എം.
7) രോഗിക്ക് ചികിത്സാ സംബന്ധമായ എന്തെങ്കിലും ബുദ്ധിമുട്ടുണ്ടെങ്കിൽ ബന്ധപ്പെടേണ്ട സ്ഥാപനം	:	വി. പി. എസ്. വി. ആയുർവേദ കോളേജ്, കോട്ടക്കൽ
8) രോഗിക്ക് ചികിത്സാ സംബന്ധമായ എന്തെങ്കിലും ബുദ്ധിമുട്ടുണ്ടെങ്കിൽ ബന്ധപ്പെടേണ്ട നമ്പർ	:	9447582885
9) രോഗിക്ക് ചികിത്സാ സംബന്ധമായ എന്തെങ്കിലും ബുദ്ധിമുട്ടുണ്ടെങ്കിൽ ഡോക്ടർക്ക് പുറമെ ബന്ധപ്പെടാനുള്ള ഫോൺ നമ്പർ	:	9847150340
10) രോഗിക്ക് ചികിത്സാ സംബന്ധമായ എന്തെങ്കിലും പരാതിയുണ്ടെങ്കിൽ ബന്ധപ്പെടേണ്ട വ്യക്തി	:	ഡോ. സി.വി. ജയദേവൻ സൂപ്രണ്ട് വി.പി.എസ്.വി. ആയുർവേദ കോളേജ് കോട്ടക്കൽ

200 Random Numbers

Control group

104 124 066 092 120 183 086 084 145 106 117 085 181 093
011 003 133 089 168 131 096 149 189 123 121 072 073 178
010 059 167 042 163 055 171 125 115 162 151 038 175 017
039 024 069 135 139 021 027 063 154 081 194 054 180 004
078 109 190 172 174 187 129 102 130 047 150 094 156 170
148 045 074 013 197 153 032 195 159 012 052 186 185 136
083 188 122 177 173 118 134 034 155 025 014 101 080 103
088 002

Trial group

090 126 164 144 057 067 141 119 079 200 035 037 031 050
192 165 193 110 160 157 019 033 044 108 166 138 077 007
016 095 005 022 076 116 196 048 199 146 051 137 132 040
169 036 182 198 097 018 049 143 113 098 008 065 100 128
053 142 064 029 023 020 082 107 075 030 001 140 070 158
068 111 009 114 147 179 099 061 062 105 152 087 112 176
161 006 071 091 184 191 015 046 127 060 041 043 026 058
028 056

Specs: This table of 200 random numbers was produced according to the following specifications: Numbers were randomly selected from within the range of 1 to 200. Duplicate numbers were not allowed. This table was generated on 4/13/2012.