ENDOSCOPIC ASSESSMENT OF GI TRACT W.R.T. GRAHANI SHARIR IN CLINICALLY DIAGNOSED PATIENTS OF PITTAJ GRAHANI ROGA.

A Thesis

SUBMITTED TO THE

TILAK MAHARASHTRA VIDYAPEETH PUNE

FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

In

Rachana Sharir (Ayurveda)

Under the Board of Ayurveda Studies

BY

Vd. Yogesh Vitthal Gaikar

(P. R. No. 05613007883)

Under the Guidance of

Prof. Dr. Chandrashekhar D. Vaikos

Department of Ayurveda

December, 2019

Annexure IV

CERTIFICATE OF THE SUPERVISOR

It is certified that work entitled "**Endoscopic Assessment of GI tract w.r.t. Grahani Sharir in Clinically diagnosed patients of Pittaj Grahani Roga.**"

is an original research work done by **Dr. Yogesh Vitthal Gaikar**

Under my supervision for the degree of Doctor of Philosophy in **Rachana Sharir (Ayurveda)**

to be awarded by Tilak Maharashtra Vidyapeeth, Pune. To best of my knowledge this thesis

- embodies the work of candidate himself/herself
- has duly been completed
- fulfils the requirement of the ordinance related to Ph. D. degree of the TMV
- up to the standard in respect of both content and language for being referred to the examiner.

Signature of the Supervisor

Annexure III

Tilak Maharashtra Vidyapeeth, Pune

Undertaking

I Dr. YOGESH VITTHAL GAIKAR is the Ph. D Scholar of the Tilak Maharashtra Vidyapeeth in Rachana Sharir subject. Thesis entitled ENDOSCOPIC ASSESSMENT OF GI TRACT W.R.T. GRAHANI SHARIR IN CLINICALLY DIAGNOSED PATIENTS OF PITTAJ GRAHANI ROGA under the supervision of Dr Chandrashekhar Vaikos , I Solemnly affirm that the thesis submitted by me is my own work. I have not copied it from any source. have gone through extensive review of literature of the related published / unpublished research works and the use of such references made has been acknowledged in my thesis. The title and the content of research is original. I understand that, in case of any complaint especially plagiarism, regarding my Ph.D. research from any party, I have to go through the enquiry procedure as decided by the Vidyapeeth at any point of time. I understand that, if my Ph.D. thesis (or part of it) is found duplicate at any point of time, my research degree will be withdrawn and in such circumstances, I will be solely responsible and liable for any consequences arises thereby. I will not hold the TMV, Pune responsible and liable in any case.

I have signed the above undertaking after reading carefully and knowing all the aspects therein.

Signature : 

Address : F/103, 1st Floor, Saipooja Bldg., Station Road, Behind Bus Depot,
Vikhroli (East), Mumbai - 400083.

Ph.No. : 9987519516 e-mail : yogeshgaikar83@gmail.com

Date : Place : Mumbai

ACKNOWLEDGMENT

गुरुर्ब्रह्मा गुरुर्विष्णु गुरुर्देवो महेश्वरः ।

गुरुःसाक्षात परर ब्रह्म तस्मै श्री गुरवे नमः ।।

This work is an outcome of constant encouragement, the valuable guidance and supervision from my guide Prof. Dr. Chandrashekhar Vaikos MD, Ph.D., Dept of Rachana Sharir, Govt Ayurved College Nanded He is a constant source of inspiration for me throughout my Post Graduation. He emphasized the minute details pertaining to perfection regarding the presentation of data. This is a great opportunity for me to express my profound gratitude to my respected guide.

A research work is always a combined effort, hard work and endurance of a team. A single person can not achieve it. Right from the selection of the topic to the typing, printing and binding of dissertation, numerous people have helped me and supported me. This dissertation is dedicated to all those people who were always with me. I am thankful for their help, guidance and wishes.

I am Thankful To Department of Ayurved, Tilak Maharashtra Vidyapeeth, Pune for giving me an opportunity to study in the Ph.D. Dept. of Ayurveda.

I am very thankful to Prof. Dr. Abhijeet Joshi, Dean, Faculty of Ayurveda, Tilak Maharashtra Vidyapeeth, Pune who provided all the essential facilities for the completion of this thesis.

I express my very sincere gratitude to Dr. Sachin Sarportdar, Dr. S.M. Sathye, Dr. Prashant Tople, Dr. Sanjay Satpute, Dr. Arun Dudhamal, Dr. Manoja Joshi for their invaluable guidance

and support to me for the completion of my research work.

I am very much thankful to my colleagues and friends Vd. Yogesh Yelmar, Vd. Rohan Patil, Vd. Vinayak Chatane for their co-operation in compilation of this dissertation. I am also thankful to my all friends who have helped me directly or indirectly in my research work.

I am very much thankful to Sion Ayurved Mahavidyalaya & Seth R.V. Ayurvedic Hospital Sion Mumbai India and Lokmanya Tilak Municipal Medical College & General Hospital Sion, Mumbai

I am very much thankful to Joy Hospital and Dr. Roy Patankar, Dr. Meghraj Ingale, Dr. Milind Patil for their timely co-operation and guidance during the study.

I am very much thankful to Mr. Arjun Angne and Mrs.Sushama Pathak for their timely support in printing of my thesis.

Last but not the least, My salutation to my beloved parents Mr. Vitthal Rambhau Gaikar and Mrs. Bijala Vitthal Gaikar who made every possible step of my life. Without their blessings, I could not complete this work. I am also thankful my dear brother Nilesh Gaikar and life-partner Dr. Meenakshi who supported me a lot in my life.

Finally, I thank Godess Saraswati who strengthened me and covered with her blessings throughout.

VD. YOGESH V. GAIKAR

INDEX

LIST OF TABLES & GRAPHS

ABBREVIATIONS

1	च. सं.	-	Charak Samhita
2	सु. सं.	-	Sushrut Samhita
3	अ. सं.	-	Ashthang Sangrah
3	अ. हृ	-	Ashthang Hrudaya
4	मा. नि.	-	Madhav Nidan
5	भा. प्र.	-	Bhava Prakash
6	यो. र.	-	Yoga Ratnakar
7	शा. सं.	-	Sharangdhar Samhita
8	हा. स.	-	Harit Samhita
9	वं. सं.	-	Vangsen Samhita
10	ग. नि.	-	Gada Nigraha
11	च. द.	-	Chakra Datta
12	भे. सं.	-	Bhela Samhita
13	भै. र.	-	Bhaishajya Ratnavali
14	र. र.	-	Rasaratnasamuchyaya
15	सू.	-	Sutrasthan

16	नि.	-	Nidansthan
17	वि.	-	Vimansthan
18	चि.	-	Chikitsasthan
19	सि.	-	Siddhisthan
20	शा.	-	Sharirasthan
21	खि.	-	Khilasthan

INTRODUCTION

Knowledge of Sharir is conducive to the well being of the individual. Our Ancient Acharyas have extolted the knowledge of sharir & has given much importance for same. Though the acharyas through their wisdom have tried to eloborate certain concepts very wisely & precisely, still their literature need an approach based on practically rather than logically oriented work, which can be accepted easily by the modern medical science.

"शरीर विचयः शरीरोपकारार्थमिष्यते ।

ज्ञात्वा हि शरीरतत्वं शरीरोपकारकरेषु भार्विषु ज्ञानमुत्पद्यते ।

तस्माच्छरीर विचयं प्रशंसन्ति कुशल ।।ह्न

- च. शा. ६ / ३

Today it is resposibility of ayurvedic scholars to analyse various principle & unexpalined facts with the help of different pramanas (Agam, Anuman etc.) Through their guidelines, we should utilize various means to achieve our goal to interpret different concepts of Ayurveda with the help of various other sciences.

' रोगाः सर्वेऽपि मन्देग्नौ '।

अ.ह्न. नि. १२/१

In Ashtang Hruday Nidansthan it is mentioned that the root cause of every disease is 'Agnimandya' and grahani being the adhistan of agni it is necessary to study the grahani organ in detail.

Grahani has been described as a kosthang/organ, a part of Annavaha or mahasrotas/digestive system & as a "disease" in different ayurvedic classical text. Grahani is a unique term used as the name of Kosthang/organ and disease. Hence while reading it may

create some sort of ambiguity in readers mind. Vagbhatta has given a list of some obstinate (Mahagada) diseases and grahani roga has been included in that list. This shows the seriousness of the disease and its prognostic aspect is also not favourable. Such diseases which are obstinate, difficult to treat and are associated with numerous complications require a careful attention of the learned physicians. So hereby, an effort is being made to identify and associate the structural changes seen in grahani, i.e. small intestine viz. duodenum, jejunum and ileum in pittaj grahani roga mentioned in Madhav nidan with the help of modern diagnostic aids, so that it would support the diagnostic methods in ayurvedic text to diagnose pittaj grahani roga early and treat it effectivly.

In a souvenir publish by Mumbai rajkiya anusandhan samiti in the year 1956 after a sharir saudnya parishad, it is metioned on parishisht 7 and pg no. 22 that amashaya can be co-related to stomach and pakwashaya with colon. Considering this fact the below mentioned quotes suggest that grahani is the avayava in between stomach and colon which is also termed as laghvantra.

षष्ठी पित्तधरा नाम या कला परिकीर्तिता

पक्वामाशय मध्यस्था ग्रहणी सा प्रकीर्तिता ।।

सु. उ. ४० / १६९

" तदधिष्ठानमन्नस्य ग्रहणाद् ग्रहणीमता।

सैव धन्वन्तरिमते कला पित्तधराव्ह्या।। "

अ.ह्र.शा. ३/५०

There are different views and approach about the location of grahani among ancient ayurvedic experts which should be confirmed with the help of modern investigative methods.

Keeping in mind the above view this research project was planned and carried out.

Aims and Objectives

AIM

- To determine and correlate the structural changes in grahani avayav with reference to pittaj grahani vyadhi

OBJECTS

- To study and assess the subjectwise relevance of grahani sharir
  a. Anatomically
  b. To determine anatomical location and extent of grahani

REVIEW OF AYURVEDIC LITERATURE

PREVIOUS WORK DONE

A) National Institute of Ayurved

Sharma K.C. -

Mahasrotasa ke

Pariprekshya main Grahani Ka Vaigyanika Adhyana

1994 - Jaipur

B) State Ayurvedic College

Devendra Singh -

An anatomical determination of Grahani w.s.r. to

grahani roga

1994 - Lucknow

C) Govt. Ayurved Mahavidyalaya

Ambilduke Premlal -

Ayurvedokta Grahani ya Avayavache

Shareera Rachanashastra Drishtya Vishesha Adhyayana

2000 - Nagpur

After the review of previous work done in 1994 at Lucknow & Jaipur and recently in 2000 at Nagpur, the literary compilation done about grahani is insufficient in applied perspective. In the previous work the focus was mainly on the location of grahani and its ayurvedic concepts.

LITERARY REVIEW

The exact origin of ayurveda is lost in the midst of antiquity and is difficult to pinpoint, which have been place around 6,000 B.C. The references of grahani cannot be seen in old traditional text like veda, purana & darshans but we can find certain word in rugveda and atharveda such as 'apva' which is indicative of certain obdominal diseases

' अमिषां चित्तं प्रतिलोभयंति ग्रहणांगानि अप्वे परेहि ।
अभिप्रेहि निदहतस्यु शोकेरन्धेन मित्रास्तमसा सचंताम् ।।'

- ऋग्वेद ११/१०३/१२

In rugveda 'apva' word is used to indicate a disease of abdomen

' हरिणामते अंगेभ्योऽषायन्तरोदरम् ।
यक्ष्मोधामन्तरात्मनो बहिर्निमिन्त्रिसा महे ।।'

- अथर्ववेद ९/८/९/२८०

'मृत सर्प समा नाडी ग्रहणी रोग मादिशेत ।'

- रावणपुराण

Therefore it is clear that in the ancient literature we can find very few references about grahani but which are important according to historical view

CHARAK SAMHITA

In the 13th chapter of charak samhita sutrasthan we can find the reference of grahani organ as the site of agni

" विरेचयन्ति नैतानि क्रूरकोष्ठं कदाचन ।
भवति क्रूरकोष्ठस्य ग्रहण्यत्युल्बणानिला ।।"

- च.सू. १३/६८

" उदीर्णपित्ता ऽ ल्पकफा ग्रहणी मन्दमारुत ।

मृदुकोष्ठस्य तस्मात् स सुविरेच्यो नरः स्मृतः ।।"

- च.सू. १३/६९

उदीर्णपित्ता ग्रहणी यस्य चाग्निबलं महत ।

भस्मीभवति तस्याशु स्नेहः पीतो ऽ अग्नितेजसा ।

- च.सू. १३/७०

In the 14th chapter of chikitsasthan grahanidosha has been described in view of nidanpanchak and chikitsa in detail after the arsharoga.

" त्रयो विकाराः प्रायेण ये परस्परहेतवः ।

अर्शांसि चातिसारश्च ग्रहणीदोष एव च ।।"

- च. चि. १४/२४४

In the 3rd chapter of siddhisthan grahani has been described during the description of vamparshvasthiti in the bastividhi

"वामाश्रयो ऽग्निर्ग्रहणीगुदं च तत् पार्श्व-

संस्थस्य सुखोपलब्धिः ।

लीयन्त एवं वलयश्च तस्मात् सव्यं

शयानोऽर्हति बस्तिदानम्।।"

- च.सि. ३/२४

SUSHRUT SAMHITA

"पक्वामाशय मध्यस्थं पित्तं चतुर्विधमन्न पानं पचति, विवेचयति च दोषमुत्र पुरीषाणि तत्रस्थमेव चात्मशक्त्या शेषाणां पित्तस्थानांना शरीरस्य चाग्निकर्माणा ऽनुग्रहं करोति "

- सु.सू. २१/१०

1.Sushrut has described grahani with respect to location of pitta which resides in between amashaya & pakavashaya

2. In Shushrut sharir sthan grahani has been described with respect to the pittadhara kala.

3. In Sushrut samhita uttar tantra 40th chapter, purvarupa of grahani along with nidan panchak is described in detail.

षष्ठी पित्तधरा नाम या कला परिकीर्तिता

पक्वामाशय मध्यस्था ग्रहणी सा प्रकीर्तिता ।।

सु. उ. ४० / १६९

ASHTANGA SANGRAHA & ASHATANGAHRIDAYA

1. In the 5 th chapter of sharirsthan of ashtangasangraha, grahani is described with respect to the digestion process carried out by the agni situated in the pittadharakala

"षष्ठी पित्तधरा नाम पक्वामाशयमध्यस्थ सा

हि अन्तराग्नेधिष्ठानतया आमाशयात पक्वाशयोन्मुखं

अन्न बलेन विधार्य शोषयति, पचति, पक्वं च विमुंचति ।

दोषधिष्ठितासु दौर्बल्यादाममेव ।

ततोऽसौ अन्नस्य ग्रहणात पुनर्ग्रहणीसंज्ञा,

बलं च तस्या पित्तमेव अग्नि अभिधानं ।।

अ.सं.शा. ५/४०.

2. In the 3rd chapter of the ashatanghrudya sharirsthan accepting the view of dhanvantari grahani has been described as follows :-

" तदधिष्ठानमन्नस्य ग्रहणाद् ग्रहणीमता।

सैव धन्वन्तरिमते कला पित्तधराव्ह्या।। "

अ.हृ.शा. ३/५०

भुक्तममाशये रुदध्वा सा विपाच्य नयति अधः ।

बलवत्यबला त्वन्नमामेव विमुञ्चति।।"

अ.हृ.शा. - ३/५२.

LAGHUTRAYI :

1. In the 4th chapter of madhavnidan grahani has been described with respect to it's nidan.

2. In the 4th chapter of bhavprakash madhyakhanda grahani has been described as 'pachakashaya".

3. In the 7th chapter of sharangdharsamitha purvardha grahani has been described in between pakvashaya & amashaya.

"पष्ठी पित्तधरा नाम या कला परिकीर्तिता।

पक्वामाशय मध्यस्था ग्रहणी इति अभिधियते।"

- शा. सं. अ. ७/६

OTHER AYURVEDIC CLASSICS

Harit samhita

"यदल्पाल्पम क्रमेशते निषेवितम मलं भगाधारगतं च नित्यंम् ।

हत्वाऽन्तरग्निम् कुरुते नरस्य विकारम् आहू ग्रहणी इति संज्ञाम् ।"

- हा.सं. ३/ ३

Bhela Samhita :

1. In the 6th chapter of sutrasthan 'Madyaprashansa' grahani has been described

2. In the 23rd chapter of the sutrasthan grahani roga refrences are found

3. In vimansthan it is described as

"ग्रहणी दूषणी योक्तं मन्दाग्ने : तु चिकित्सितम् ।"

भे.सं.वि. ३/१०

4. In the 7th chapter of chikitsasthan it is described with respect to the following quotes.

अतिशीतादी बस्तीप्रयोग दोषा : ।

भे.सं.चि. ३/१०

Vangasen Samhita :

He has described grahani similar to charak samhita

Yogratnakar :

Grahani has been described in the poorvardha of Yogratnakar.

Rasaratnasammuchaya :

Grahani has been described in the 16th chapter of Rasaratnasammuchaya

(16/ 203)

Bhaishajyaratnavali :

Grahani has been described in the 8th chapter of Bhaishajyaratnavali.

Gadnigraha :

Vaidya Sodhal has described grahani in the 3rd chapter of Gadnigraha.

Chakradatta :

Chakrapani Datta has accepted the views of charkacharya and repeated the same.

RECENT AYURVEDIC TEXT

Siddhant nidanam

Acharya Gananathsen has described grahani in the 5th chapter with respect to modern anatomy.

Saundnyapanchak Vimarsh :

In this grantha Acharya Gananathsen has described the relation of pittadhara kala & grahani in a beautiful way.

Pratyakshashariram :

Acharaya gananathsen has described it as the initial part of shudrantra in the 3rd chapter of ashayakhand.

Parishadya shabdharth shariram :

Pandit Damodar Sharma has campared grahani with the small intestine of modern anatomy.

Ayurvediya sharir

Dr. Dhirendranath Banerjee has described it as the mucous membrane of small intestine and further described its anatomy and physiology

In short, seeing the historical review we can say that grahani is mostly described through sharir kriyatmak and vikruti vidnyanatmak view. So we have to first take a review of physiological and pathological aspect and then approach towards the rachanatmak (Anatomical) view.

AYURVEDIC REVIEW

NIRUKTI OF GRAHANI SHABDA (ETYMOLOGY)

Nirukti means interpretation of a specific word

Nirukti of grahani means how the word grahani is derived or originated.

In amarkosh the nirukti of grahani is mentioned as follows :

१. ग्रहः–ग्राहः इति ग्रहणस्य, ग्रहे ग्राहो।

२. निर्बन्धपरागार्कादयो ग्रहाः ।

निर्बन्ध, उपराग आग्रहविशेष अर्कादिग्रह ।

३. उपरागो ग्रहा-चन्द्रार्क ग्रहण, सूर्यादि ग्रहो राहुग्रस्ते त्विन्दो

च पूष्णिच उपरागः ग्रहः इति।

The above quotes is said with respect to sun and other planet but in daily routine ग्रहण word is used for accepting, holding, maintaining etc.

The term grahani is derived from the 'ग्रह' आदाने dhatu

Further the pratyaya ड़िप. is attached to it.

i.e. 'ग्रह' + 'ड़िप'.

According to rules of grammer grahani word has been derived by adding 'ड़िप'. pratyaya to the word 'ग्रह' which means to hold. The addition of 'ड़िप' pratyaya leads to the composite word becoming feminine. This word 'ग्रहणी' needs to be understood as गृ ग्रहणात इति ग्रहणी or 'गृण्हाति अन्नरसो'.

PARYAYA (SYNONYMS)

As Grahani is mainly described with its physiological and pathological view the following text shows the usage of the word Grahani in context as a organ which makes its anatomical study more simple

१. अग्निऽधिष्ठानम् - Used as the adhisthan of Agni

२. आशयेअंत्रानाम् - as it has ashay appearance and it accepts the food

३. अग्निस्थान - In reference to the location

४. तेजपथः - Being the path of Agni

५. पक्वामाशयमध्यम - As it is situated between stomach & intestine

६. आमपक्वाशयान्तरम् - In reference to the site of Pitta

७. अग्न्याशय - 'नाभेरुर्ध्वमग्न्याशयः स एवं ग्रहणीस्थानम कथितम् ।' (आढम्मल्ल टिका - शार्गधर)

८. पित्तधराकला - In laghutrayi & bhruhatrayi it is mentioned as inner linning of intestine

९. पच्यमानाशय - Shreekanthadatta, the student of madhukoshakar vijayrakshit has described as 'अग्नेःपच्यमानाशय'

१०. पूरितात - Rajnighantukar has described as small intestine

११. पक्तिस्थान - 'पक्तिस्थानात् ग्रहण्याः ।' - हेमाद्री

१२. ग्रहणीनाडी - Due to the pipe like appearance madhukoshakar has described intestine as ग्रहणीनाडी

१३. क्षुद्रांत्रावयव - Ashtanga sangraha has described it with respect to the loops of small intestine in inguinal hernia

१४. दहनाशय - As digestion process takes in this place

१५. पाचकाशय - Bhavaprakash has described it on basis of Agni & Pitta

References -

१. "अग्न्याधिष्ठानम् अन्नस्य ग्रहणाद् ग्रहणी मता।
नाभेरुपरि सा ह्याग्निबलोपस्तम्भबृंहिता।।"

– च. चि. १५/५६

२. "स्थानानि आमअग्निपक्वानाम् मुत्रस्य रुधिरस्य च ।
ह्रदुण्डुकः फुप्फुसश्च कोष्ठ इत्याभिधियते ।।"

- सु. चि. २/१२

३. "स्वल्पम् यदा दोषविवध्दम आमं लीनं न तेजःपथमावृणोति ।
भवति अजीर्ण तदा बुभूक्षा या मन्दबुाध्दिं विषवत निहन्ति ।"

- सु. सू. २१/६

४. "दोषस्थानानि अतः अर्ध्व वक्ष्याम् ।
पक्वामाशयमध्यं पित्तस्य आमाशय श्लेष्मणः ।।"

- सु. सू. ४/६

५. "आमपक्वाशयान्तरे च त्रिधा जायन्ते ।"

- सु. शा. (धमनीव्याकरण)

६. "उर्ध्वम् अग्न्याशयो नाभीर्वामभागे व्यवस्थितयः ।"

- शारंगधर संहिता अ. ७

७. "तद्ऽधिष्ठानम् अन्नस्य ग्रहणात् ग्रहणी मता ।
सैव धन्वन्तरिमते कला पित्तधराह्वया ।"

- अ. ह्र. शा. ३/५०

UTPATTI OF GRAHANI (ORIGIN)

The utpatti (origin) of intestine is described by shushrut as follows :

'असृजः श्लेष्मणश्चापि यः प्रसादः परो मतः ।

तं पच्यमानं पित्तेन वायुश्चापि अनुधावति ।

ततोऽस्यान्त्राणि जायन्ते गुदं बस्तिश्च देहिन ।।'

- सु. शा. ४/२६-२७

The intestine is formed from the cavity created by the vayu after the digestion of the essence of rakta and kapha by the pitta. Here the word antra applies to both small intestine (Grahani) and Large intestine.

GRAHANI - MATRUJ BHAVA

Garbha is a constitution of the following factors called as Shadbhavas i.e Matruja, Pitruja, Rasaja, Atmaja, Satvaja, Satmyaja.

जसे - "तत्रगर्भस्य पितृज मातृज रसज आत्मज सात्म्यजानि
शरीरलक्षणानि व्याख्यासामः।
गर्भस्य केशश्मश्रु लोमास्थि नख दंत सिरास्नायू धमनी
रेतः प्रभृतीनि स्थिराणि पितृजानि।
मांस शोणित मेदोमज्जाह्रन्नाभियकृत्प्लीहान्त्रगुदप्रभृतीनि
मृदुनि मातृजानि।"

- सु. शा. ३ / ३३

Grahani (Antra) is classified in matruja bhava, as it is mrudu (Soft) organ.

References :-

तद्यथा - त्वक् च लोहितं च मांस च मेदश्च नाभिश्च हृदयं च क्लोम च यकृच्च प्लीहा च वृक्कौ च बस्तिश्च पुरीषाधानं चामाशयश्च पक्वाशयश्चोत्तरगुंद चाधरगुदं च क्षुद्रान्त्रं च स्थूलान्त्रं च वपाच वपावहनं चेति (मातृजानि) ।।

च. शा. ३/६

तेषु मातृजानि मृदूनि त्वग्रक्तमांस मेदोमज्जानाभि हृदयामाशयगर्भाशय कृत्प्लीहाक्लोमान्त्रगुदादीनि ।।

अं. सं. शा. ५/७

लोहितं च मांसं च नाभिश्च हृदयं च क्लोम च
यकृच्च प्लीहाच वृक्कौ च बस्तिश्च पुरीषाधारणं
चामाशयश्चोत्तरगुदयश्च क्षुद्रान्त्रं च
स्थूलान्त्रं च वपाच वपावहनं चेति मातृजानि ।।

का. शा.

Hence all the acharyas have classified grahani (Antra) in the matruj bhava. This Suggests that grahani is a very soft and delicate structure Here the word antra applies to both small intestine (Grahani) and Large intestine.

GRAHANI - RACHANA SHARIR

STHANA OF GRAHANI (Site)

The meaning of the word sthana is location. Here it means the location or the site at which Grahani is located.

The sthana or site of any organ is of prime importance therotically as well as clinically.

The word sthana is derived as follows -

स्थान - स्था + ल्युट्

ज्ञापके निग्रहस्थानम्

The sthana of grahani as described in the Ayurvedic texts is as follows :-

It is located above the umbilical region and is supported and nourished by the strength of Agni.

स्थान -

" अग्निऽधिष्ठानं अन्नस्य ग्रहणाद् ग्रहणीमता ।

नाभेरुपरि सा ह्याग्निबलोपस्तंभ बृहिता ।"

- च. चि. १५/५६

Sushruta has described the sixth kala which has been described as the pittadhara kala (pitta containing sheath) situated between pakvasaya and amashaya as grahani.

षष्ठी पित्तधरा नाम या कला परिकीर्तिता

पक्वामाशय मध्यस्था ग्रहणी सा प्रकीर्तिता ।।

सु. उ. ४० / १६९

Asthangsangrahakar has described pittadharakala as grahani in view of it's function of with holding the food.

"षष्ठी पित्तधरा नाम पक्वामाशयमध्यस्थ सा

हि अन्तराग्नेधिष्ठानतया आमाशयात पक्वाशयोन्मुखं

अन्न बलेन विधार्य शोषयति, पचति, पक्वं च विमुंचति ।

दोषधिष्ठितासु दौर्बल्यादाममेव ।

ततोऽसौ अन्नस्य ग्रहणात पुनर्ग्रहणीसंज्ञा,

बलं च तस्या पित्तमेव अग्नि अभिधानं ।।

- अ. सं. शा. ५/४०

Vaghabhatt has described it at the pakvashayadwar that is at the upper part of pakvashaya.

'स्थिता पक्वाशयद्वारि भुक्तमार्गार्गलेव सा ।'

- अ. हृ. शा. ३/५१

It is situated at the entrance of the pakvashaya and acting as a bolt to the door of pathway of food.

'वामाश्रयो ऽग्निर्ग्रहणी गुदं च तत् पार्श्वसंस्थस्य ।'

- च. सि. ३/२४

Charaka in siddhisthan has described its site in the left of the abdomen and lateral to the rectum, which can be taken as ileum.

In Ayurveda the term Koshtha and Ashaya have been used in reference to the cavity and vital organs both present in Madhya sharir or Antaradhi.

" स्थानानि आमाग्नि पक्वानां मूत्रस्य रुधिरस्य च

हृदुण्डूकः फुप्फुसश्च कोष्ठ इति ऽभिधियते ।"

- सु. चि. २/१२

Charaka has described it in the Koshtha as follows:-

Charaka has enumerated only 15 koshthangas that includes Antra (Small Intestine & Large Intestine)

पञ्चदश कोष्ठाङ्गानि तद्यथा् - नाभिश्च हृदयं च क्लोम च यकृच्च प्लीहा

च वृक्कौ च बस्तिः च पुरीषाधारश्च आमाशयश्च पक्वाशयश्च उत्तरगुदं च अधरगुदं च

क्षुद्रान्त्रं च स्थूलान्त्रं च वपावहनं चेति ।। च. शा. ७/१०

Bhel who has been considered as the best commentator on Ayurvedic anatomy has given a list of 15 koshthangas, which include Nabhi, Hridaya, Kloma, Yakrit, Pleeha, two Vrikka, Basti, Purishadhara, Amashaya, Uttarguda, Adharguda, Kshudrantra, Sthoolantra, and Vapavahan.

पञ्चदश कोष्ठाङ्गानि तद्यथा् - नाभिश्च हृदयं च क्लोम च यकृच्च प्लीहा च

वृक्कौच बस्तिःच पुरीषाधारश्च आमाशयश्चोत्तरगुंद च अधरगुंद च क्षुद्रान्त्रं च

स्थूलान्त्रंच वपावहनं चेति ।। भे. शा. ७/४

Hridaya, Kloma, Phuphhus, Pleeha, Unduka, 2Vrikka, Nabhi, Dimba granthi, Antra and Basti are the koshthangas.

कोष्ठाङ्गानि स्थितान्येषु हृदयं क्लोम फुप्फुसम् यकृत्प्लीहोन्दुकं वृक्कौ

नाभिडिम्बान्त्रबस्तयः।। अ. हृ. शा. ३/१२

The great Ayurvedic anatomists - Bhel, Kashyapa and Vagbhata all have used the terms Uroguha and Udarguha for Koshtha.

Hence Antra [small intestine (Grahani) and large intestine] is a subpart of Koshtha i.e. Koshthanga located in the Udaraguha.

GRAHANI - PRAMANA (DIMENSIONS)

Charaka has suggested the methods of physical examination by which one can judge the proportionate relations, the norms of size, height, length and breadth of different body organs and parts. This method is known as Anjali and Anguli Pramana method. The dimensional extent of grahani (Antra) has been described three and half vyam long in males & three vyams in females

"सार्धत्रिव्यामान्यन्त्राणि पुंसां, स्त्रीनामर्धव्यामहीनानि ।"

- सु. शा. ५/९

"तानि सार्ध त्रिव्यामानि पुंसाम । त्रिव्यामानि स्त्रीणाम्"

- अ. सं. ५/२८

१ व्याम - ४ हाथ

३ १/२ व्याम - १४ हाथ - २१ ते २२ फुट

The above described dimensions regarding antra are applied to both shudrantra (Small intestine) and stulantra (Large intestine)

AGNI AND GRAHANI

In ayurveda prime importance is given to agni

The word agni is formed from **'अग'** (गतौ) Dhatu

'अंगति उर्ध्व गच्छति, अग्रयत्वं प्रापयति ।'

Which means, Agni is the entity which always goes in the upward direction so it is always in the leading position.

In body different metabolism are carried out with the help of Agni. The main function of Agni is to transform the heterogenous elements into homologus ones for the synthesis of tissue elements like rasa, rakta etc.Therefore the main strength utilised in formation of different dhatus in the body is agni. Therefore its normal state is very essential for the promotion and preservation of positive health. Therefore it is included in the Twelve pranas.

AGNI MAHATMYA :-

" आयुवर्णो बल स्वास्थ्मुत्साहोपचयौ प्रभा ।

ओजस्तेजोऽग्नय: प्राणाश्चोक्ता देहाग्निहेतुका : ।

शान्तेऽग्नौ म्रियतेयुक्ते चिरंजीवत्यनामय:।

रोगी स्यात् विकृते, मूलमग्नि तस्मात् निरुच्यते ।"

- च. चि. १५/३-४

The physiological activity of Dehagni is acknowledge by the quality of life, power or strength of body and mind, health, enthusiasm, growth, glow of skin, immunity, body heat, digestive power and vitality.

Therefore Jatharagni is considered to be the most important sustaining factor of living beings.

Acharya Bhel has described about agni as follows:-

" अग्निवायु मनुष्याणां प्राणा : तत्र प्रतिष्ठिता :

बलं आरोग्यमायुश्च तस्मात्प्राणायुषी विद्यातऽग्निमुले शरिरीणाम् ।

सोऽग्नि समुचितं सुखं दूखं तदाश्रयम् ।

म्रियते हयुपशान्तेऽग्नौ युक्ते चोष्याणि जीवति ।

चतुर्विधं : पचत्याग्निः समं तीक्ष्णं तथा मृदु ।

त्वेषमं चेतितेषा तु यस्यमोऽग्निस्य शस्यते ।"

- भेल ४

AGNI PRAKARA -

" इति भौतिकधात्वन्न पत्तृणां कर्म भाषितम् ।"

- च. चि. १५/३८

The physiological activities can be understood only by the detail study of thirteen types of 'Agni' i.e. Jatharagni, Panchbhautikagni and Saptadhatwagni.

" भौतिका:पुञ्च , धात्वग्न्यः सप्त :, अन्नपक्ता एकः अत्रं च

यात्यग्न्यन्तराणि उपधातु मलादि गताणि तान्यपि अवरुध्दानि

भूताग्निषु एव अप्रधान्यद्न्यामर्किंचित्कराणि नोक्ताणि ।"

- चक्रपाणि

STHANA

" अग्निऽधिष्ठानं अन्नस्य ग्रहणात ग्रहणी मता ।

नाभेरुपरि सा अग्निबलोपस्तम्भ बृंहिता ।"

- च. चि. १५/५६

The sthana of agni described in the Ayurvedic texts is as follows :-

It is located above the umbilical region in grahani.

" स्वस्थानस्थस्य कायाग्नेरंशा धातुषु संश्रिता : ।

तेषां सादाति दीप्तिभ्यां धातुवृध्दी क्षयोद्‌भवा : ।"

- अ. हृ. सू. ११/३४

Kayagni present in its own place, has portion of itself, present in the dhatu, also the decrease & increase give rise in the increase & decrease of dhatu respectively. The preceeding dhatu which either increase or decrease give rise to the succeeding dhatu of the same condition. On this Arun Datta has commented as follows.

" स्वस्थानं कायाग्ने : पक्वामाशयो मध्यम् .....

..... कायाग्ने जाठराणलस्य, अशा भागो सादेत, मांद्येण ।"

- अरुणदत्त

As kaya means whole body, kayagni means portion of Jatharagni.

" स्वस्थानस्थस्य ग्रहणीस्थस्य । कायाग्ने अन्नपक्तु ।"

- हेमाद्री

" जाठरः प्राणिणामग्नि : काय इत्यभिधीयते ।

यस्तं चिकित्समत्सीदन्तं स एवं कायचिकित्सकः ।।"

- भोज

<u>THE FUNCTION OF AGNI IN GRAHANI</u>

" अन्नादानकर्मा तु प्राणः कोष्ठं प्रकर्षति ।

तद् द्रवैर्भिन्नसङ्घातं स्नेहेन मृदुतां गतम् ।

समाने नावधूतोऽग्निरुदर्यः पवनेन तु ।

काले भुक्तं समं सम्यक् पचति आयु : विवृध्दये ।

एवं रस मलायान्नमाशयस्थमधः स्थित : ।

पचति अग्निर्यथा स्थाल्यामोदनायाम्बुतण्डुलम् ।।"

- च. चि. १५/६-८

As the fire placed below helps in the cooking of food namely rice and water kept in a vessel placed thereon, so does the prana, draws the food in to kostha which is softened by the kledak kapha and thereafter the agni stimulated by the samanavayu helps in digestion of food of appropriate quality in required quantity and at the right time for the production of rasa and mala.

" अन्नस्य पक्ता सर्वेषां पक्तृणाम्धिपो मताः ।

तन्मूलास्ते हि तद्वृदृध्दिक्षयवृध्दिक्षयात्मकाः ।।"

- च. चि. १५ / ३९

" जाठराग्निना पूर्व कृते संघातभेदे पश्चात भूताग्नयः

स्वं स्वं द्रव्य पचति इति ।"

- चक्रपाणि

Immediately after the digestion of complex food , the simplified food is futher catalyzed into guna of panchabhautik dravya to increase their own property as parthiva, apya, agneya, vayaviya and nabhas in the whole body (Deha) with the help of their own ushma or agni.

" तच्चादृष्टहेतुकेन विशेषेण पक्वामाशयमध्यस्यं पित्तं चतुर्विधं

अन्नपानं पचति विवेचयति च दोषरसमूत्रपूरीषाणि, तत्रस्थमेव ।"

चात्मशक्त्या शेषाणां पित्तस्थानां शरीरस्य चाऽग्निकर्मणाऽनुग्रहं

करोति, तस्मिन् पित्ते पाचकोऽग्निरिति संज्ञा। "

- सु. सू. २१/१०

The pitta situated between pakvashaya and amashaya by invisible mechanism digest four types of food and drinks, seperates dosha, rasa, urine and faeces and as it is seated there, it supports by its innate power to the remaining seats of pitta and also the body with functions of Agni. Therefore the name 'Digestive fire' is given to this agni.

" अदुष्टहेतुकेन प्राक्तनकर्महेतुनत्यर्थ :

पक्वामशयमध्यस्थमिति नाभिस्थम : ।

चतुर्विधमन्नपानं पचतिति अशीतं खादितं लीढं पीतं च पचतीत्यर्थ ।

विवेचयति च पृथक्करोति दोषरसमूत्रपुरीषाणि ।"

- डल्हण

ग्रहणी आश्रित अग्नि दुष्टी चे स्वरुप

"ग्रहणी आश्रितो अग्निदोषः एवं चाश्रयाश्रयीणोरभेदोपचार्

ग्रहणी शब्देन ग्रहण्याश्रितो अग्निदोषोऽपि गृहयते । "

- चक्रपाणि

गृहण्यां बलं अग्नेर्हि स चापी ग्रहणी श्रीताः ।

तस्मात संधूषिते वन्हौ ग्रहणी संप्रदुष्यती ।।

- सु. उ. ४०/१६९

When Agni is in its normal state it is the strength of Grahani. But due to its ashrayashrayi relation when agni is impaired it is responsible for the causation of grahani dosha

" यश्चाग्नि पुर्वमुद्दिष्टो रोगानीके चतुर्विध :

तं चापि ग्रहणीदोषं समवर्जं प्रचक्ष्महे ।"

- च. चि. १५/७१

Charkacharya has described four types of Agni viz tikshna, manda, vishama & sama. The first three types of disorders of agni constitute grahani dosha. This shows the inseperable relation of agni and grahani.

"सिराविभागे ये चोक्ता विषमाद्या स्त्रयोऽग्नयः।

तेऽपिस्युर्ग्रहणीः दोषाः समस्तु स्वास्थकारणम् ।।''

- अ. सं. १५

The ashtangsangrahakar has described grahani roga and included the vishmadi agnis described in the sira vibhag in grahani dosha.

"दूर्बलो विदहति अन्नं तद् यात्यूर्ध्वमधोऽपि वा

अधस्तु पक्वामांम वा प्रवृत्तं ग्रहणीगदः ।"

- च. चि. १५ / ५१

When agni is durbala vidaha of food occurs which moves upwards and downwards in a gastrointestinal tract. The digested and undigested food moves downwards this condition is called Grahani Gada. So it is advised to protect the agni of the body.

Form the above anatomical and pathological references it is clear that grahani is the site of agni.

PACHAKPITTA AND GRAHANI

Pitta word is derived from 'Tap-santape' dhatu which means to digest, to break, to seperate and these all are the functions of pitta.

"विसर्गोदानविक्षेपैः सोमसूर्यानिलयथा।

धारयन्ति जगद्देहं कफपित्तनिलास्तथा ।।"

- सु. सू. २१/८

As said by shushrutacharya the vital humours Vayu, Pitta and Kapha maintain the integrity of the animated organism by creating, assimilating and diffusing strength in the same way as the moon, the sun and the wind maintain the integrity of the terrestrial globe.

" न खलु पित्त व्यातिरेकादन्योऽग्निरुपलभ्यते ।

आग्नेयतत्वात् पित्ते दहनपचनादिष्वभिप्रवर्तमाने

अग्निवदुपचारः क्रियतेऽन्तरग्निरिति।"

- सु.सू २१/९

" अग्निरेव शरीरे पित्तान्तर्गतः कुपिता कुपितः

शुभाशुभानि कार्याणि करोति ।"

- च. सू. १२/११

According to above quotes agni cannot sustain whithout pitta. Residing in the pitta agni carries out the digestion process. We cannot imagine an independent agni.

PACHAK PITTA VIVECHAN

Sthan

"दोषस्थानि अत उर्ध्व वक्ष्याम् ।

पक्वामाशयमध्यम् पित्तस्य, आमाशयश्लेष्मण : ।

- सु.सू २१/६

तर पक्वामाशयमध्यगम् .....

..... बलदानेन पाचक नाम ततस्मृतम् ।

- अ. हृ. सू. १२/१०

According the above reference Pitta is situated in between pakvashaya and amashaya which is the location of grahani. On this acharya Hemadri has comentated as follows :-

"पाचकस्य स्थानकर्माण्याह तत्रेति पाचकस्य पक्वामाशयमध्यं

ग्रहणाख्य स्थानम् अन्नस्य पचन पक्वा सारकिट्टाविभागो ।"

- हेमाद्रीः

Functions :-

" तत्र पक्वामाशयमध्यगम् ।

पञ्चभूतात्मकत्वेपि यत्तैजस गुणोदयात्

त्यक्तद्रवत्वं पाकादिकर्मणानलशब्दितम्

पचत्यन्नं विभजते सारकिट्टौ पृथक् तथा

तत्रस्थमेव पित्तानां शेषाणाम अपिअनुग्रहणम्

करोति बलदानेन पाचकं नाम तत् स्मृतम् ।"

- अ. हृ. सू. १२/१०

It is located in between pakwashaya and amashaya. Though it is composed of panchamahabhut because of increase of Tejas mahabhut in it, it is devoid of liquidity (i.e. though it is liquid it does not posses snigdha, sheeta and such other properties of mahabhut). It is called by the term 'Anala' because of its functions of paka i.e. digestion and tranformation of food material.

It cooks the food divides it into essence and waste seperately. Being localised

there, it bestows grace to other pitta present there by giving them strength (Power of functioning) this is called ''Pachak pitta''

" तत्र यदामाशयपक्वाशयमध्यस्थं पंचमहाभूतात्मकत्वेऽपि

तेजोत्कर्षात क्षपितसागुणं ततश्च त्यक्त द्रवस्वभाव सहकारि

कारणै वायुर्क्लेदनदिभिरक्त ग्रहणदहन पचनादि क्रियाल्वधाग्निशब्दं

पित्तमन्नं पचति सारकिट्टौ विभजति शेषाणि च पित्तस्थानानि

तत्रस्थमेवानुग्रहति तत् पाचकमित्युच्यते।।"

- अ. सं. सू. २० / १३

Therefore residing in its own place pachak pitta digests the food, seperates the essence and waste, acting on the different dhatus in the body thereby bestowing its grace.

"तदन्न पचति तत्सारत्वं किट्टत्वं चान्नस्य सम्पादयति शेषाणि

पित्तस्थाननिधात्वादिन्यनु ग्रण्हाणि स्वस्थानस्थितमेव

तेषुपकरोतिऽत्यर्थ : ।" - इन्दु.

Therefore after analysing the above references we can realise that agni located in grahani is called by different names such as pachakpitta, jatharagni, antaragni, kayagni etc.

PITTADHARAKALA AND GRAHANI

To study the anatomy of grahani, it is necessary to study the pittadharakala situated in the grahani. In ayurvedic text pittadharakala is described with its pathological view. The deformity in the pittadharakala situated in grahani is responsible for the grahani roga.

" कलाः खल्वऽपि सप्तसंभवन्ति धात्वाशयान्तरमर्यादाः ।"

- सु.शा. अ. ४/५

Seven kalas (membranes) appear as structure interveining between dhatus (Rasa, rakta etc.) and there seats (Viscera).

"यस्तु धात्वाशयान्तुरेषु क्लेदोऽवतिष्ठते स
यथास्वं उष्माभिः विपक्व
काष्ठ इव सारः धातुसारः धातुरसविशेषो
वा अल्पत्वात् कला संज्ञाः ।"

- अ. सं. शा. ५

As on the cutting wood, its pith is observed, likewise dhatu is found dissecting the musculature

" यथा ही सारः काष्ठेषु छिद्यमानेषु दृश्यते।
तथा हि धातुर्मांसेषु छिद्यमानेषु दृश्यते ।।
स्नायुभिश्च प्रतिच्छन्नान् सन्ततांश्च जरायुणा ।
श्लेष्मणा वेष्टितांश्चपि कलाभागांस्तु तान् विदुः ।।"

- सु. शा. ४/६-७

The kalas are those which are covered by ligaments spread as membranous structure like amniotic membrane and sneared with shleshma (mucus).

षष्ठी पित्तधरा नाम या कला परिकीर्तिता
पक्वामाशय मध्यस्था ग्रहणी सा प्रकीर्तिता ।।

सु. उ. ४० / १६९

"षष्ठी पित्तधरा, या चतुर्विधमन्नपानमामाशयात्

प्रच्युतं पक्वाशयोपस्थितं धारयति ।"

- सु. शा. ४/१८

Sixth kala known as pittadhara kala is situated between amashaya and pakvashaya and is the seat of internal fire because of this, holding strongly the movement of four types of food material passing from amashaya into the pakvashaya to digest them with the heat of pitta.

"षष्ठी पित्तधरा नाम पक्वामाशयमध्यस्थ सा

हि अन्तराग्नेधिष्ठानतया आमाशयात पक्वाशयोन्मुखं

अन्न बलेन विधार्य शोषयति, पचति, पक्वं च विमुंचति ।

दोषधिष्ठितासु दौर्बल्यादाममेव ।

ततोऽसौ अन्नस्य ग्रहणात पुनर्ग्रहणीसंज्ञा,

बलं च तस्या पित्तमेव अग्नि अभिधानं ।।

- अ. सं. शा. ५/४०

The sixth kala is named as pittadhara and is located in between pakvashaya and amashaya. Being the abode internal fire (digestive activity) it withholds by force the movement of food material passing from the amashaya in to the pakvashaya, digest the food by the heat of pitta, absorbs it and allows the digested food to move further, under the influence of decreased activity of the doshas present there in it allows even undigested food to move further. Hence this is also known as Grahani in view of its function of withholding the food. Its strength is from pitta itself, known as Agni. These activated by this fire like activity maintains the welfare of the body.

"तद्ऽधिष्ठानमन्नस्य ग्रहणाद् ग्रहणी मता ।

सैव धन्वन्तरीमते कला पित्तधराह्वया ।।

आयुरारोग्यवीर्योजोभूतधात्वाग्निपुष्टये ।

स्थिता पक्वाशयद्वारी भूक्तमार्गार्गलेव सा ।।"

- अ. हृ. शा. ३/ ५०-५१

It's seat is grahani so called because it withholds the food, for a certain time inside the small intestine to fecilitate digestion. In the opinion of dhanvantari it is the kala known as pittadhara situated at the entrance of the pakvashaya and acting as a bolt to the door of pathway channel of food, it is responsible for the duration of life health, valour, ojas, strength of the bhutagni and dhatvagani.

" तस्मांत् साऽसौ पित्तधराकला क्षुद्रांत्राणाम् अन्तरावरणी ।"

- ग. से. (संज्ञापञ्चक विमर्श)

Pittadharakala stands for the digestive & absorptive processes when the food travels down from the stomach to the colon. The inner linning of small intestine situated between stomach and large intestine can be considered as grahani because kala is the inner linning membrane inside the container of the dhatu and ashaya. Here dhatu means the wall of small intestine and ashaya means the lumen of small intestine, therefore the inner linning of small intestine can be considered as grahani.

From the above discussion it is clear that pittadharakala is suitated in shudrantra i.e. small intestine. Its function is carried out in the parts of small intestine i.e. duodenum, jejunum & ileum. Most of the functions is carried out in duodenum & jejunum and gradually decreases in ileum.

DOSHASTHANA, AVASTHAPAKA AND GRAHANI

DOSHASTHANA

Pittadosha - Pachakapitta

पितं पञ्चात्मंक तत्र पक्वमाशयमध्यगम् ।

- अ. ह्र. सू. १२/१०

पक्वामाशयमध्यम् पित्तस्य ।

- सु.सू. २१/६

According to the above quotes grahani is site of pitta dosha. According to the description the pitta dosha is known as Pachakpitta.

VATADOSHA - PRANVAYU AND SAMANVAYU

प्राणापान समानैस्तु सर्वतः पवनैस्त्रिभिः ।

धमायते पाल्यते चापि स्वां स्वां गतिमनस्थितैः ।।

- सु. सू. ३५/२८

"समानोऽग्निसमीपस्थः कोष्ठे चरति सर्वतः ।

अन्नं गृण्हाति, पचती, विवेचयति मुञ्चति ।।

- अ. ह्र. सू. अ. १२/८

Samanvayu located near the Agni (fire) moves in the koshta, witholds the food brought by the pranavayu from the amashaya in the ailmentary tract for some time, digests, seperates the essence and wastes and eliminates the wastes

"समानेनावधूतोऽग्निः उदर्यः पवनेन तु ।"

- च. चि. १५/७

स्वेददोषाम्बुवाहिनी स्त्रोतांसि समतिष्ठीतः ।

अंतराग्नेश्च पाश्वर्तः समानोऽग्निबलप्रदा ।।

- च.चि. २८ / ८

KAPHADOSHA - KLEDAK KAPHA

"यस्त्वामाशयसंस्थितः क्लेदकः सोडन्नसंङ्घात् क्लेदनात ।"

- अ. हृ. सू. १२/१६

The prime location of kledak kapha is amashaya where the food is moistened and softened. According to panchamahabhut siddhant due to some part of kledak kapha the food in the grahani gets the properties of snehatva and mrudutva. So some part of kledak kapha resides in grahani.

AVASTHAPAKA

"अवस्थिक पाकः अवस्थापाकः ।"

"आदौ षड्समप्यन्नं मधुरीभूतमरियेत् ।

फेनीभूतं कफं, यातं विदाहात् अम्लतां तताः ।

पित्तमायाशयात्कुर्याच्यवमाने, च्युतं पुनः ।

अग्निना शोषितं पक्व पिडितं कटुमारुतम् ।।"

- अ. हृ. शा. ३। ५७-५८

"अन्नस्य भुक्तमात्रस्य षड्रसस्य प्रपाकतः ।

मधुराद्यात् प्राक् कफो भावात् फेनभूत उदीर्यते ।।

परं तु पच्यमानस्य विदग्धस्याम्लभावतः ।

आशयाच्चवमानस्य पित्तमच्छउदीर्यते ।

पक्वाशयं तु प्राप्तस्य शोष्यमाणस्य वन्हिना ।

परिपिण्डितपक्वस्य वायुः स्यात् कटुभावतः ।।"

- च. चि. १५। ६-१०

The food we eat undergoes three avsthapaka in the order as follows. :

1. Madhur Avasthapaka
2. Amla Avasthapaka
3. Katu Avasthapaka

The juices formed in stages of digestion in stomach and small intestine (grahani) as a result of the action of agni on food is called as avasthapaka.

After the conversion of solid food particles into semisolid in the madhur avasthapak in the stomach the simplified food particles becomes ready for further digestion by the action of pitta, this semi digested food goes into small intestine from the stomach for it's complete digestion (Amlaavasthapak) where the pitta comes in the small Intestine (grahani). In this avasthapaka due to the influence of amlaguna vidaha of food occurs. If the food is pitta vardhak then it adds to the guna of the pitta emerging in the small intestine making it significant. The food is held in the small intestine (grahani) till the food is completely digested.

According to prakruti every man has different type of agni according to which the food is digested and held in the small intestine for a definite time. Digestion also changes with the quality and quantity of food. Due to faulty food habits and lifestyle, doshas become vikrut in turn agni becomes weak because of which small intestine (grahani) some time pushes undigested, foul smelling, watery and sometimes hard food particles before the time which causes periodic motions, which are some time watery and some time hard (Muhurbaddham, Muhurdravam)

This is called as grahani dosha.

ANNAPARINAMAN IN GRAHANI

"प्राणाः प्राणाभृताम् अन्नं लोकोऽभिधावति ।''

- च. सू.२७ / ३४९

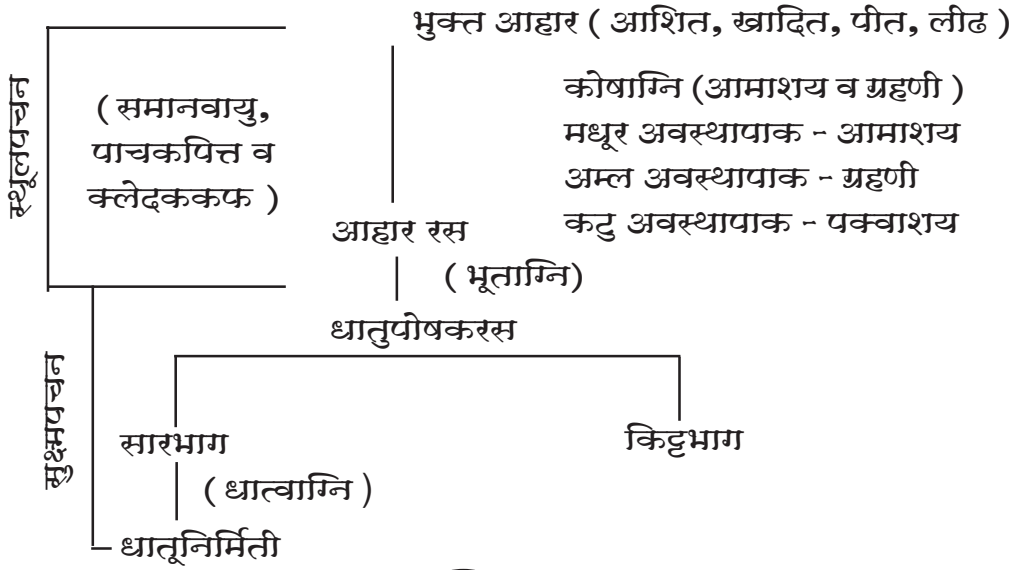
Acharyas while explaining the importance of food has quoted food as prana. The four types of food we eat saves the prana. That is the reason every body shows the need for food.

पंञ्चभूतात्मके देहे आहारः पाञ्चभौतिकः ।

विपक्वः पञ्चधा सम्यक् गूणान् स्वानभिवर्धयेत् ।।

- सु. सू. ४६/५२४

As per the above quotes ayurved explains the presence of panchmahabhuta in the food we it. Also the simplest component (Aharasa) formed after the action of kosthagni on food is again acted upon by the bhutagnis to produced dhatu posaka rasa which is again divided into sara and kittabhag. This sara is again acted upon by their respective dhatwagni to produce the respective dhatu. In this way the process of digestion (pachan can be divided into sthula pachan and sukshma pachan



In the modern text it has been explained that the food contains proteins, fats, corbohydrates & various vitamins and minerals. This ingredients are digested by the different types of secretion and juices secreted by the different organs of the body. For e.g. The pancreatic juice secreted by the pancreas, bile juice from the liver and succus entericus from the intestine acts upon the food to digest the various ingredients in food to convert in to simple components which nourishes the body. The above juices can be co-related with the jatharagni, bhutagni & dhatwagni of ayurved.

EMBRYOLOGICAL DEVELOPMENT OF GRAHANI

The epithelial linning of the various parts of the gastrointestinal tract is of endodermal origin. Endoderm, which is at first in the form of a flat sheet, is converted in to a tube by formation of head, tail & lateral folds of the embryonic disc. This tube is the gut. The primitive gut is in free communication whith the rest of the yolk sac. The part of the gut cranial to this communication is the foregut, the part caudal to the communication is the hindgut, while the intervening part is the midgut.

Duodenum

The superior (or first) part and the upper half of the descending (or second) part of the duodenum are derived from the foregut. The rest of the duodenum develops from the most proximal part of the midgut (Fig. A). The part of the gut that gives rise to the duodenum forms a loop attached to the posterior abdominal wall by a mesentery (mesoduodenum) (Figs B, C). Latter, this loop falls to the right. The mesoduodenum then fuses with the peritoneum of the posterior abdominal wall, with the result that most of the duodenum persists in relation to a small part of the duodenum adjacent to the pylorus. This is the part seen in radiographs as the duodenal cap.

In keeping with its development, the proximal part of the duodenum is supplied by branches of the coeliac artery and the distal part by branches of the superior mesenteric

Jejunum and ileum

The jejunum and most of the ileum are derived from the prearterial segment of the midgut loop. The terminal portion of the ileum is derived from the postarterial segment proximal to the caecal bud (Fig. D).

MODERN REVIEW

ABDOMEN & ITS NINE REGIONS

For purpose of description of the position of the viscera, the abdomen is divided into the nine regions of pair of vertical & a pair of horizontal planes which can be indicated on the surface by lines.

The vertical line one on either side of the midline connecting the midclavicular & midinguinal points are called the right & left vertical planes. Similarly two tranverse lines mark the transpyloric & the intertubercular planes. The transpyloric planes lies midway between the suprasternal notch & the upper margin of the pubic symphysis. An approximate guide to this plane is a horizontal line drawn midway between the xyphoid process & the umbilicus. This planes corresponds to the level of the lower margin of the body of the first lumbar vertebra. In this planes lie the pylorous of the stomach, the fundus of the gall bladder, the neck of the pancreas, the hilum of the left kidney, the point of origin of the superior kidney, the point of origin of the superior mesenteric artery from the abdominal aorta & the joint of commencement of the portal vein.

The transtubercular planes passes through the tubercle of the iliac crests on the either side of the body and through the upper border of LV5 behind.

The nine subdivisions of the abdomen demarcated by the intersection of these two horizontal and the two lateral planes are the epigastric, umbilical & hypogastric in the middle, flanked on either side by the hypochondric, lumbar & iliac regions.

However, some authorities divides the abdomen into the above mentioned regions by replacing the transpyloric plane by the subcostal plane which runs across the lower part of the tenth costal cartilage in front and the upper border of the body of LV3 behind.

Although, most of the viscera are liable to change either position in the living according to the posture adopted and tonicity of muscle.

GENERAL CONSIDERATION OF DIGESTIVE TRACT.

It is a long muscular tube consisting of the following parts from above downwards.

The mouth, tongue, pharynx, oesophagus, stomach & digestive canal. The ducts of the salivary gland, opens into the mouth. The proximal end of the stomach (its junction with oesophagus) is guarded by the cardiac sphincter. The distal end of the stomach is guarded by the pyloric sphincter.

The small intestine begins after the pyloric sphincter & consists successively of the following subdivisions duodenum, jejunum & ileum. The duodenum receives food from stomach, where the duodenum joins the jejunum, connective tissue & muscle fibres thickens the mesentry. The bile duct & pancreatic duct jointly opens in it through ampula of vater. The small intestine is very long & is roughly about 7.6 meters. The great length of the small intestine provides enough time & surface area, so that digestion & absorption of foodstuff may be complete.

The small intestine opens into the next part the large intestine. The opening between them is guarded by iliocaecal sphincter. In the large intestine water is absorbed & the faeces are formed. The large intestine opens into the last part, rectum & anal canal & it opens outside through the anal orifice. Peritoneum is a serous membrane & lines the interior of the abdominal cavity. The parietal outer layer is in contact with the body wall & the visceral layer envelops the abdominal organs.

The mesentry is the continuation of the peritoneum & extends to the small & large intestines from the dorsal body wall. The lesser omentum hangs from the greater curvature of the stomach over the intestine to the colon as an apron. In this fold fat may accumulate.

SURFACE ANATOMY OF SMALL INTESTINE

The duodenum commences 1.5 cm to the right of midline in the transpyloric planes & extend upwards and laterally for about 5cm and then descends vertically in the umbilical region for about 7cm till it reaches a level about 1cm above the umbilicus. It then runs transversely across the abdomen for 10cm and then ascends about 2.5 cm to the left of the midline to end at the duodenojejunal flexure which is at the level of LV2 i.e 2.5 cm below the transpyloric plane.

The Jejunum which forms the upper two fifth of small intestine occupies the umbilical region chiefly but may extends into adjacent lumbar region as well.

The iIeum forms the lower three fifth of small intestine & occupies the hypogastric & pelvic regions.

ANATOMICAL CONSIDERATION OF SMALL INTESTINE

The small Intestine, a coiled tube, extends from the pylorus to ilocaecal sac 6 to 7 meters long, gradually diminishing in diameter towards its termination.

The small intestine occupies the central & lower parts of abdominal cavity, usually within the colonic loop it is related in front to the greater omentum & abdominal wall, a portion may reach the pelvis in front of the medium. It consists of a short curved sessile section, the DUODENUM and a long, greatly coiled, part attached to the posterior abdominal wall by the mesentary, the proximal two fifth being the JEJUNUM, the distal three fifth, the ILEUM.

DUODENUM :-

The term duodenum is derived from the greek word 'Dudekadaktulos' meaning twelve fingers, The width of twelve fingers is about 25cm. This is the first part of small intestine, it is also the shortest widest, thickest and the most fixed part of small intestine. It extends from pylorus to duodenojejunal flexure, a length is about 25cm. It forms "c" shaped curved, the concavity of which is occupied by the head of pancreas. Its course is described successively as superior, descending, horizontal and ascending parts respectively.

The first superior part, is 5cm long and begins at the level of LV1 on the right side on the midline.

RELATION

Anteriorly are the quadrate lobe of liver and gall bladder. Posteriorly are lesser sac, the gastrodeunal artery and common bile duct with portal vein behind these & the inferior venacava still further back behind the peritoneum superiorly is the epiploic foramen, then just below and behind the foramen the right gastropancreatic fold. Inferiorly is the head of

pancreas and the orcades of the superior pancretioduodenal vessels.

The second (Descending) part is a vertical and descends from neck of gall bladder to the right of the bodies of LV2 & LV3 across the hilum of the right kidney and the commencement of the ureter. It is about 8cm long and is itself crossed by the ascending colon, the hepatic flexure laterally together with the past of right lobe the liver and head of pancreas medially. About half way down its postmedial surface, combined common bile & pancreatic ducts pierce the duodenal wall.

The horizontal part - is also about 8cm - 10cm long runs, generally at or below the subcostal plane.

Relations : Anteriorly are the roots of the mesentry, coils of gut and the trunk of the superoir mesentric artery.

Posteriarly are right ureter, psoas muscle, inferior venacava and aorta from the right to left.

Superiorly are the head and uncinate process of the pancreas.

Inferiorly are the coils of jejunum

The fourth ascending part is about 2.5 cm long and runs upwards and slightly to left side of the midline to bend downwards as the duodenojejunal flexure just below the roots of the transverse mesocolon on at the level of LV2.

Relations - Anteriorly are coil of jejunum, transverse colon, lesser sac & stomach. Posteriorly and to the right is the left margin of the abdominal aorta and upper part of the root of the mesentery.

Posterio-laterally is the left kidney and left ureter.

Superiorly is the body of pancreas.

Suspensary muscle of Duodenum

This is a fibromuscular band which suspends and supports the duodenojejunal flexure.

It arises from the right crus of diaphragm close to right side of the oesophagus, passes downwards behind to pancreas and is attached to posterior surface of duodenojejunal flexure & third & fourth part of duodenum.

It is made up of a striped muscle fibres in upper part, Elastic fibres in middle part & plain muscle fibres in lower part. Normally, its contraction increases the angle of doudenojejunal flexure. It is a important landmark in the radiological diagnosis of incomplete rotation and malrotation of small intestine.

Arterial Supply

Duodenum is supplied by superior pancreaticoduodenal artery & below it by the inferior pancreticoduodenal artery. First part of duodenum receives additional supply from right gastric artery, supraduodenal artery, Retroduodenal branches of gastroduodenoartery, some branches of right gastro epiploic artery.

Venous Drain:-

It drains into splenic, superior mesentric & portal veins.

Lymphatic Drain:-

Most of lymph vessel from the duodenum drain into in the pancreaticoduodenal nodes presents at the junction of pancreas & duodenum, then lymph passes partially to hepatic nodes to coeliac node & partially to mesentric nodes. Vessels from first part drain into pyloric node & through then to the hepatic nodes.

Nerve Supply

Sympathetic nerve of the spinal segment T9 & T10 & parasymphathetic nerve from the vagus, pass through the coeliac plexus & reaches the duodenum along the artery.

Applied Anatomy of Duodenum

1. The first part of the duodenum is not adequately supplied by blood from the superior pancreaticoduodenal artery especially in its proximal part. Nature attempts at remedying this inadequacy by providing a small branch from the hepatic or gastro duodenal artery which vascularizes the proximal portion. It's terminal twigs are end arteries. Thrombosis or obstruction leads to neurosis & peptic ulceration of this part of the duodenum.

2. The acidic chyme is squirted through the pylorous to inpinge on the anterolateral wall of the first part of the duodenum. The above factors may explain the reason why duodenal ulcer. are common in this part of the duodenum. Sometimes acid secreting (Oxyntic) cells are also formed in this region.

3. The anterolateral surface of the first part of the duodenum abuts on to the supramesocolic compartment of peritoneum & hence, it is here that infection occurs following perforation.

4. Perforation of ulcers on the posteromeidial surface of the proximal part of the duodenum will cause infection of the lesser sac.If the perforation is more distal, then infection may also pass up along the inferior vena cava & pus collects in the right extraperitoneal subphrenic space.

5. The surgical treatment of choice in cases of chronic duodenal ulcer is to divide the vagal nerve tissues.

(Truncal vagatory) remove the pyloric artery & perform anastomosis between the body of the stomach & the distal duodenum (gastroduodenostomy). The combined procedure abolishes the cephalic phase of acid secretion. The anastomosis also provide rapid emptying of in stomach contains into distal duodenum. The last is a vital step since vagotomy causes the pyloric sphincter to remain in the contracted state.

6. The duodenojejuneal flexure is a surgical land mark.

7. In constant peritoneal recesses called duodenal fossae lie in relation to the fourth part of the duodenum, these pockets are important only because loops of bands may be trunked into them causing strangulation hernia.

8. Since the duodenum is fixed to posterior wall of abdomen. It is ruptured following a very heavy blow in the upper abdomen. The close relationship of the aorta to it is worth remembering in operative procedures.

9. The common congenital anomalies are diverticula & duplicator & severe ones are atresias & stenosis.

THE JEJUNUM & ILEUM

The rest of small intestine extends from the duodenojejunal flexure to ileoceacal valve, ending at the junction of caecum & ascending colon. It is completely covered by this peritoneum excepts along its mesentric border.

THE JEJUNUM

It has a diameter of 4cm and internal diameter of 2.5 cm. It is thicker walled, redder and more vascular. Its circular mucosal folds are large & frequent & its villi larger, Aggregated lymph follicle are almost absent from the proximal jejunum, circular fold can be felt through its wall since they are absent from the distal ileum. Palpation allows a crude

distinction between upper & lower intestinal levels. The Jejunum lies largely in the upper left infracolic compartment and umbilical region but may extends into surrounding areas. The first coil occupies which recess between the left part of the transverse mesocolon & the left kidney.

THE ILEUM

It has a diameter of 3.5 cm, its wall is thinner than that of the jejunum. A few & small circular folds occur proximally but these are small & disappear in its distal parts. Aggregated lymphatic follicles are larger & more numerous than that of jejunum.

The ileum is mainly in the hypogastric & pelvic region. Its terminal part usually lies in the pelvis from which it ascends over the right psoas major & right iliac vessels to end in the right illac fosa, opening into the medial side of the junction between the caecum and colon.

Fan like mesentric attachment of the jejunum & ileum to the posterior abdominal wall allows free movement, each with adopting to changes in front & posterior.

BLOOD SUPPLY

The Jejunum & ileum are supplied by branches for the superior mesentric artery & are chained by corresponding veins.

LYMPHATIC DRAINAGE

Lymph from lacteals drains into plexuses in the wall of gut. From there it, passes into lymphatic vessels in the mesentery & ultimately drains into nodes present in front of the aorta at the origin of the superior mesenteric artery.

NERVE SUPPLY

They are supplied by the symphathetic & parasymphathetic nerves from segments T6 to T10 of the spinal chord.

APPLIED ANATOMY OF JEJUNUM & ILEUM

1. It may cause intestinal obstruction.

2. Acute inflammation of the diverticulum (meckel's) may produce symptoms that resemble to those of appendicitis.

3. It may be involved in other diseases similar to those of the intestine like

   a. Commonest site for intussusception at iliocaecal junction

   b. Worm infestation

   c. Paralytic ileus

   d. Loop of small bowel is favoured site for anastomosis

4. Jejunal feeding can be given in situations were the stomach and duodenum are either suitable or unavailable for receiving oral nutrition which can be performed by surgical jejunostomy or insertion of feeding tube.

HISTOLOGICAL STRUCTURE OF THE SMALL INTESTINE

The intestinal wall has four layers

1. Serous
2. Muscular
3. Submucos
4. Mucous

The serosa is visceral peritoneum with a subserous statum of loose connective tissue. The musculari's externa is thicker in the proximal intestine, consisting of a thin external circular layer of non striated myocytes. The submucosa is loose connective tissue carrying blood vessels, lymphatic & nerves. The mucosa is thick & very vascular in the proximal small intestine but the inner & less vascular in the distal. It forms circular folds & the whole surface is covered by filiform or linguifom intestinal villi.

Circular folds - Plicae circularis or Valves of kerlong.

(For Absorptive purposes)

They are large, crescentric folds of mucosa which project into the intestinal lumen transverse to the long axis. Unlike gastric folds they are not obliterated by distension. Most extends round about one half or two thirds of the luminal circumference. Some are complete circles, some bifurcate & join adjacent folds. Some are spinal but extend little more than once resend the lumen through occasionally two or three times. Larger folds are about 1m deep but most are smaller. Larger & smaller folds are often alternate.

The muscularis mucosa of small intestine contains external longitudinal & internal cicular layer of non striated myocytes. It extends into the circular folds. Internal to it is connective tissue fibres, lymphocytes, eosinophilic leucocytes, macrophages, most cells

capillaries, lymphatic vessels & non myelinated nerve fibres plasma cells are numerous, lymphatic follicle extends from muscularis mucosa into the submucosa. Between the intestinal villi, crypts of Lieberkuhn and Brunner's gland are present. Their ducts extending through the muscular's mucosae to expand into the submucosa. Their secretions contains mucosubstances, bicarbonate, iron & a small amount of enzyme helping to activate trypsinogen from the pancreas.

INTESTINAL VILLI

They are the highly vascular processes just visible to the naked eye & project from the entire intestinal mucosa. Large & numerous in the duodenum & Jejunum, small & fewer in the ileum.

STRUCTURE

It has a core of reticular tissue containing a lymph vessel or lacteals blood vessels, nerves & non striated myocytes covered by columnar epithelium on a basement membrane.

THE LACTEALS

Usually single but occasionally double, starts in a closed dilated extremity near the villus summit & discred to empty into a lymphatic plexus in the lamina propria.

MYOCYTES

Derived from the musculari's mucosa cluster around the lacteals from the base to the summit of the villus.

Contraction of these myocytes milks the lacteals.

BLOOD VESSELS

Form a capillary plexus in the lamina propia enclosed in reticular tissue capillaries are lined by few straited endothelium, probably to ensure the rapid uptake of nutrients diffusing from epithelium.

THE EPITHELIUM

1) Absorptive columnar cells

2) Interspersed goblet cells.

INTESTINAL GLANDS

They are numerous throughout the intestinal mucosa. They are tubular, perpendicular pits, opening of small circular apertures & visible with a simple lens as minute dots between the villi. Their thin walls consist of a columnar epithelium or basement membrane, associated with which is a rich capillary plexus. The epithelium consist of

a) Un differentiated stem cells.

b) Zymogenic (paneth) &

c) Argentaffein cells.

Duodenal glands of Brunner, limited to the duodenum are sited in the submucous connective tissue i.e. their ducts traverse the muscularis mucosa. Largest & most numerous near the pylorus & forming as almost complete duodenum, they gradually diminish & disappear at the duodennojejunum junction.

Solitary lymphatic follicle scattered throughout the intestinal mucosa, are most numerous in the distal ileum. Each follicle surrounded by the opening of intestinal glands, consist of dense reticlar tissue packed with lymphocytes & a dense capillary network.

Aggregated Lymphoid Follicles

They are circular or oval each containing 10 = 260 follicles. Aggregate follicles, largest & most numerous in the ileum, are small, circular & few in the distal jejunum & only occasionally in the duodenum.

BLOOD SUPPLY

Jejunal & ileal arteries, stem from the superior mesenteric branches of which, reaching the mesenteric border, extends between the serosal & muscular layers. From these, numerous stem traverse the muscle, supplying it & forming an intricate submucosal plexus from which minute vessels pass to glands villi.

LYMPHATIC DRAINAGE

The lymph vessels are arranged at two levels mucosal & muscular. Lymph vessels from mucosa & submucosa are joined by vessels from lymph spaces at the bases of solitary follicle & drain to larger vessels at the mesenteric aspects of the gut.

NERVE SUPPLY

Vagus & thoracic splanchenic nerve through the coeliac ganglia & superior mesenteric plexuses.

Fibres pass to the myenteric plexus or Aurebach's plexus of nerves & ganglions between the circular & longitudinal layers of the muscularis externa, which they supply. From this a secondary submucosal plexus or Meissner's plexus is derived, formed by branches perforating the circular muscular layer.

THE MYENTERIC PLEXUS CONYROLS MAINLY THE GASTRONTESTINAL MOVEMENTS AND THE SUBMUCOSAL PLEXUS CONTROLS MAINLY GASTROINTESTINAL SECRETION AND LOCAL

BLOOD FLOW.

The myenteric plexus consists mostly of linear chains of many interconnecting neurons that extend the entire length of the gastrointestinal tract. Because the myenteric plexus is a linear plexus & because it lies between the longitudinal & circular masses of intestinal smooth muscles; it is concerned mainly with controlling motor activity along the length of the gut.

When stimulated its principal effects are:

1) Increased tubic contraction, or tone of the gut wall.

2) Increased intensity of rhythmical contractions.

3) Slightly increased rate of the rhythm of contractions

4) Increased velocity of conduction of excitatory waves along the gut wall, causing more rapid movement of the peristaltic waves.

अन्नं गृण्हाति पचति विवेचयति मुञ्चति ।वा. सू. १२/८

The submucosal plexus, in contrast to the myenteric plexus, is mainly concerned with controlling function within the inner wall of each minute segment of the intestine.

For instance, many sensory signals originate from the gastrointestinal epithelium & are then integrated in the submucosal plexus to help control local intestinal secretion, local absorption, & local contraction of submucosal muscle that causes various degrees of folding of the stomach mucosa.

Functional types of movements in the gastrointestinal tract.

Two types of movements occur in the gastrointestinal tract.

1) Propulsive movements: These cause food to move forward along the tract at an appropriate rate for digestion and absorption. (Peristalsis)

2) Mixing movements : Which keep intestinal contents thoroughly mixed all the times.

MOVEMENTS OF SMALL INTESTILE

Mixing contractions - (segmentation contractions)

The maximum frequency of the segmentation contractions in small intestine is determined by the frequency of show waves in the intestinal wall, which is the basic electrical rhythm. Because this frequency is about 12 per minute in the duodenum, the maximum frequency is also about 12 per minute. This occurs only under extreme conditions of stimulation.

In the terminal ileum, the maximum contractions are usually 8 per minute. The segmentation contractions become exceedingly weak when the excitatory activity of the enteric nervous system is blocked by atropine. therefore even though it is the slow wave in the smooth muscle itself that control the segmentation contractions, these contractions are not effective without background excitation by the enteric nervous system, especially by the myenteric plexus.

Propulsive Movement- (peristalsis):

Peristaltic activity of the small intestine is greatly increased after meal. This is caused due to a so-called gastroenteric reflex initiated by the distension of stomach & conducted principally through the myenteric plexus from the stomach down along the wall of the small intestine

.**GASTROINTESTINAL REFLEXES :** The anatomical arrangement of the enteric nervous system and its connections with the sympathetic and parasympathetic systems supports three types of gastrointestinal reflexes.

1) Reflexes that occur entirely in the enteric nervous system : They include reflexes that control gastrointestinal secretions, peristalsis, mixing contractions, local inhibitory effects and so forth.

2) Reflexes from gut to prevertebral sympathetic ganglion and back to gastrointestinal tract:

These reflexes transmit signals for long distances in the gastrointestinal tract, such as signals from the stomach to cause evacuation of the colon (gastrocolic reflex), signals from colon & small intestine to inhibit stomach motility (enterogastric reflex) & reflex from the colon to inhibit emptying of ileal contents into the colon (colonoileal reflex).

3) Reflexes from gut to the spinal cord or brainstem & then back to the gastrointestinal tract : They include specially - a) reflexes from stomach and duodenum to the brainstem & back to stomach - by way of the vagus nerve to control gastric motor and secretory activities.

The enterogastric reflexes are especially sensitive to the presence of irritants & acids in the duodenal chyme . (समानो अग्निसमीपस्थ :), often becoming strongly activated in as little as 30 sec. For instance, whenever the pH of the chyme in the duodenum falls below about 3.5 to 4, the reflexes frequently block the further release of acidic stomach contents into the duodenum until the duodenal chyme can be neutralized by the pancreatic and other secretions. The more important control of stomach emptying resides in feedback signals from the duodenum, including both, the enterogastric nervous system feedback reflexes & hormonal feedback.

MOVEMENTS CAUSED BY THE MUSCULARIS MUCOSA AND MUSCLE FIBERS OF THE VILLI :

The muscularis mucosae can cause short or long folds to appear in the intestinal mucosa; it can also cause the folds to move progressively to newer areas of the mucosa.

In addition, individual fibers from this muscle extend into the intestinal villi & cause them to contract intermittently. The mucosal folds increase the surface area exposed to the chyme, thereby increasing the rate of absorption. The contractions of the villi shortening,

elongating & shortening again- "milk" the villi, so that lymph flows freely from central lacteals into the lymphatic system. Both of these types of contraction also agitate the fluids surrounding the villi, so that progressively newer areas of fluid become exposed to absorption.

These mucosal and villous contractions are initiated by local nervous reflexes in the sub-mucosal plexus that occur in response to chyme in the small intestine.

(अन्नं गृण्हाति पचति विवेचयति मुञ्चति ।)

PHYSIOLOGICAL CONCEPTS

Physiology of small intestine is described best by short description of its functions of Reception, Secretion, Digestion, Absorption & Peristalsis as a Stimulating function.

1. Reception :

It (duodenum) receives the propelled from stomach, Bile and pancreatic juice through common hepatopancreatic duct which open in duodenum.

2. Secretion :

It secret the juices i.e. intestinal juice from the intestinal glands, Bile & Pancreatic juice through common hepatopancreatic duct which helps commonly for digestion.

FEW CONCEPTS ABOUT DIGESTIVE JUICES :-

1. Only juice does not contain all the enzymes necessary for digestion of all the different type of foodstuff.

Eg.. - Saliva contains only carbohydrate splitting enzymes where as gastric juice contains both fats & proteins splitting enzymes but not acting on carbohydrate.

2. The particular digestive juice cannot digest the particular type of food up to completion. It will digest up to some certain stage, the product will be handed over to the next digestive juice.

Eg. - Gastric juice digest the protein up to the stage of peptone, pancreatic juice carries the digestion of peptone further up to the lower peptide & then completed up to the amino-acids by succus intericus.

In Intestine Digestion takes place by the action all three juices.

1. Succus entericus :-

Composition :- Water 98.5 %

Solids 1.5 %

0.8 % salts of sodium, potassium, calcium & magnesium with salt of chloride, bicarbonate concentration is higher that is blood or Intestinal fluid in the form of inorganic salts.

Organic salts is 0.7 % in the forms of activator

Enteropeptidase which activate trypsinogen into trypisyn. It also contains following enzymes.

1. Proteolytic (crepsyn)

a. A mixture of enzymes containing dipeptidase & Amino peptidase.

b. Several enzymes acting on the different fraction of nucleic acid, such as nuclease, nucleopeptidase, nucleosidase

c. Arginase acts as arginine producing urea & arinithin

2. Carbohydrate Spliting

a. Amylase :- formed in traces, acts as starch & dextrin

b. Sucrose :- Digest sugarcane

c. Maltase :- acts as maltose

d. Iso Maltase

e. Lactase :- break down lactose

3. Fat sptitling :- lipase

4. Other enzymes :-

Alkaline phosphatase, Cholesterol entinase, Lecithinase.

Bile :-

Bile is both product of secretions as well as excretion of liver.

Formation of Bile by the liver is an active process but entry of the Bile into the Duodenum is intermittent & takes place only after meal.

Composition :-

Mainly Water & Solids in 89 & 11 percentage.

1. In organic salts
2. Bile salts
3. Bile pigments
4. Cholesterol, Lecithin & trees of fatty acids, soap bile is essential for life Although it does not contain enzymes yet it acts as a very important juice.

Bile serve the following functions :

1. Digestion : Complete digestion of fats and to some extent of proteins or carbohydrate due to the presence of bile salts.

a. By reducing surface tension

b. By Activating actin

c. Solvent action.

2. Due to presence of bile salts following subs are absorbed.

a. Fats by hydrotropic action

b. Iron & calcium

c. Vit k.

3. Excretion :- certain subs are excreated through bile for instance

a. Metal like Cu, Zn, Hg, etc.

b. Toxins & bacteria.

c. Bile pigments

d. Cholesterol a lecthin

4. Laxative action - Bile salts stimulate peristalsis when directly introduced into the canal it stimulate peristalsis of these parts.

Pancreatic Juice :- Secretion

Two phases - **1) Nervous 2) Chemical**

1) Nervous Phase :- Starts 1-2 minutes after taking foods the reflex is purely unconditioned. Unlike gastric juice there is no conditioned reflux here thus stimulus for this secretions arises both in mouth (during chewing) as well as from the stomach after the food is swallowed.

2) Chemical phase :- Starts when stomach empties into duodenum this is due to hormone like subjects called secretin and pancreozymin. Secretin thus enter the blood stream, goes to pancreas and stimulate the secretion. The secretin fraction stimulate the secretion of water, alkali and other salts of pancreas increases the enzyme content. On the whole juice stimulated by secretin is rich in alkali but poor in enzyme. Whereas secretions produced by stimulation of the vagus is poor in alkali but rich in enzyme. Pancreatic secretions varies with the type of food. Meat stimulates a secretion type of juice, fat stimulate the 'vagus' type, whereas bread elicits a mixed type of secretion.

Digestion :- From the above description, digestion of the food stuff in small Intestine take place in 3 ways.

1) The soluble enzymes-entropeptidase + Amylase act on trypsinogen & starch respectively.

Enteropeptidase -------- trypsinoges

Amylase ---------- Starch

2) Shed cell (Erephase) -------- Polypeptides, dissachharides & Fats (succus Entericus)

3) Insoluble Enzymes ----- Corresponding substrates during the course of absorption.

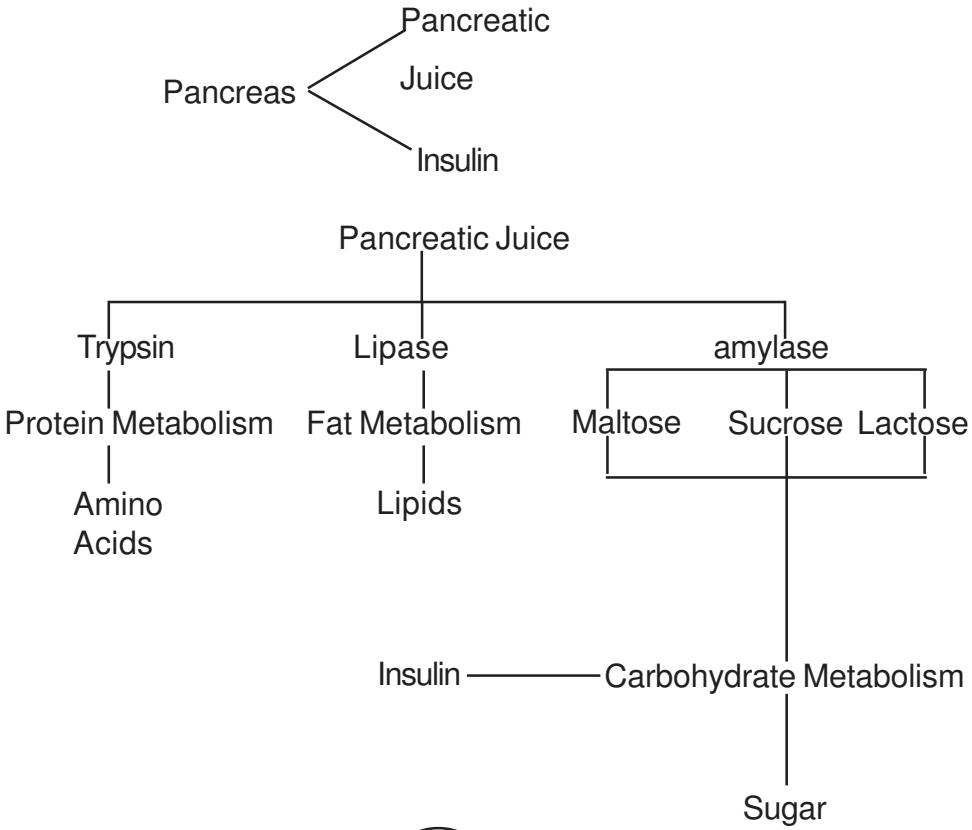
(2) Bile salt helps for digestion by

1) Reducing surface tension. So that fats are converted into an emulsion. The fine globules of fats, due the innumeral numbers, render a large surface area for the enzyme (Lipase) to act. Due to this, process of digestion is quickened.

2) Activating action :- The bile salts, by virtue of the cholic acid radicle, acts as a specific activator for different lipases.

3) Solvent action :- Bile acts as a good solvent due to this property it serves as a good medium for the intereating fat & fat spliting enzymes.

Digestion Due to Pancreas



ABSORPTION :-

Small Intestine is the chief place for absorption. Its upper part is more concerned with secretions, whreas the lower part for absorption. The end product of the digestion as well as the salts, vitamins and water are all absorbed through the small intestine. The enormous length of the small intestine affords a large surface area for maximum possible digestion and absorption. The villi of small intestine are the absorbing units. They are more numerous in the lower parts than the upper. These are about 50,00,000 villi in the human intestine with a total absorbing area of nearly 10 sq. meters. The end product of protein and carbohydrate digestion are absorbed by the vascular capillaries in the villi and are then drained into the portal system. But the fats mainly pass through the lacteals of the villi and are then carried by the lymphatic. The nature of the factors which determine different route of absorption for the different food stuff is not known.

Stimulatory Function :- Peristalsis

Peristalsis is described to be a composite wave, consisting a wave of relaxation followed by wave of constriction. It is a translatory movement and travels down the gut in an aboral direction i.e. away from the mouth type

a. First type : is a slow gentle wave moving at the rate of 1 - 2 cm second which dies out easily after travelling a short distance.

b. Second type : is a very swift wave travelling through entire length of small gut at the rate of 2 -35 cm/s (average 10 cm) known as such peristalsis.

c. Third type : i.e. Antiperistalsis

In every respect it is same with peristalsis excepting that its direction is opposite.It

moves in oral direction it is present in 2nd & 3rd part of duodenum in man only. weak antiperistalsis also occurs in the terminal part of the ileum, thus preventing the rapid passage of the ileal contents into the caecum.In the duodenum it helps through admixture as well as causes duodenal regurgitation into the stomach. These antiperistalsis movement occurs due to presence of sensitive receptor area in region which respond to the qualities of chyme and concerned with dealing the passage of chyme in to lower portion of the gut facilitating more scope for digestion & absorption.

CLINICAL CONSIDERATION OF GRAHANI ROGA

GRANTHOKTA LITERATURE

"अर्शोऽतिसार ग्रहणी विकाराः

प्रायेण चान्योन्य निदान भूताः

सन्नेऽनले सान्ति न सन्ति दीप्ते

रक्षेद तस्तेषु विशेषतो ऽग्निम ।।

- अ. हृ. चि. ८/१६४

"दुर्बलो विदहित अन्नं तद्यात उर्ध्वमऽधोऽपि वा ।

अधुस्तु पक्वमामं वा प्रवृत्त ग्रहणीगदः।

उच्यते सर्वमेवान्नं प्रारो हयस्य विदहयते।।"

- च. चि. १५ / ५१-५२

"सा दुष्टा बहुशो भुक्तमाममेव विमुञ्चति ।

पक्वं वा सरुजं पूति मुहुर्बध्दं मुहुर्द्रवम्।।"

- सु. उ. ४० / ७१

The disease in which agnimandya occurs due to faulty food habits and life styles and due to ashrayaashrayi relation of the grahani organ and agni it causes the vidaha of food because of which the patients void stool which is partly pakva and partly apakva, this condition is called as grahanigada. In this condition the entire food material usually remain in vidagdha state (i.e. part of it gets digested, the other part remaining undigested).

Charaka other than the above explanation has included all the three types agni dosha other than samagni in grahani roga as quoted below.

"यश्चाग्निः पूर्वमुदिष्टो रोगानीके चतुर्विधः ।

तं चाऽपि ग्रहणीदोषं समवर्ण प्रचक्ष्महे ।।''

- च. चि. १५/७१

As jatharagni is influenced by doshas mandagni, tikshnagni & vishamagni are included in grahani roga. In roganik chapter charaka has described the symptoms of four types of agni and its relation with doshas as mandagni from kapha, tikshnagni from pitta, vishamagni from vayu & samagni from dosha. Vaghabhatta accepting the views of charakacharya has described the above as follows :

"सिराविभागे ये चोक्ता विषमाद्या स्त्रयोऽग्नयः।

तेऽपिस्युर्ग्रहणीः दोषाः समस्तु स्वास्थकारणम् ।।''

- अ. सं. १५

Grahani roga - Samanya lakshanas

The following symptoms are found in grahani roga.

अतिसृष्ट अतिद्रवम् मलप्रवृत्ती

अविपाक, प्रसेक, छर्दी, अरुची, आस्यवैरस्य,

सर्वरसप्रियता, आमगंधिउद्गार, ज्वर,

In chronic stage शोथ, बलक्षय, तमकश्वास, दाह, मूर्च्छा

the symptoms are seen

"अतिसृष्टं विरुध्दं वा द्रवं तदुपदिश्यते।

तृष्णारोचकवैरस्य प्रसेकतमकान्वितः ।

शूनपादकरः सास्थिपर्वरुक छर्दन ज्वरः ।

लोहामर्गन्धिस्तिक्ताम्ल उद्गारश्चास्य जायते ।।''

- च. चि. १५ / ४

In association with trushna (Morbid thirst), vairasya (Distaste in Mouth) Praseka (excessive salivation) the afflicted person voids stool in large quantity either in solid or liquid form. He also suffers from oedema in legs and hands, pain in bones, phalanges, vomiting, fever and eructation having metabolic smell, smell of ama (undigested food) and bitter as well as sour tastes.

अथ जाते भवेज्जन्तुः शुनपादकरः कृश : ।

पर्वरुग्लौल्यतृट्छर्दिज्वरारोचकदाहवान

उग्दिरेचछुक्ततिक्तामललोहधूमामगन्धिकम् ।

प्रसेकमुखवैरस्यतमकारुचिपीडितः ।

- सु. उ ४० / १६५

When the disease manifests the patients gets oedema in feet and hands and suffers from emaciation, pain in joints, glutony, thirst, vomiting, fever, anorexia and burning sensation. He vomits the material having vinegar like bitter and sour taste with metallic smoky and fishy odour, excessive salivation, abnormal taste in mouth and dyspnoea.

"सामान्य लक्षणं कार्श्य धूमकस्तमको ज्वरः ।

मूर्च्छा शिरोरुकः विष्टम्भः श्वयथु कर पादयोः ।।"

- अ. ह्र. नि. ८/२१

It's general symtoms are emaciation, feeling of hot fumes coming out from mouth, dyspnoea, fever, fainting, headache, stasis of undigested food in the stomach and swelling of hands and feet.

VISHESH LAKSHANAS

पैत्तिकग्रहण्या

कट्वजीर्णविदाह्यम्लक्षाराद्यैः पित्तमुल्बणम् ।

आप्लवयद्धन्त्यनलं जलं तप्तमिवानलम् ।।११।।

सोऽजीर्णं नीलपीताभं पीताभः सार्यते द्रवम् ।

पूत्यम्लोद्गरहृत्कण्ठदाहरुचितृडदितः ।।१२।। (च.चि.अ.१५)

Pitta gets aggravated by the intake of food ingredients which are pungent, heavy, vidhahi, sour, alkaline etc. This aggravated pitta suppresses agni. The patient voids loose stool containing undigested material which is either bluishyellow or yellow in colour he also suffers from eructation having foul smell, sour taste burning sensation in cardiac region and throat, anorexia as well as morbid thirst.

SAMPRAPTI

Nidan Sevan

Katu, Amla, Guru Vidahi, Kshar, Atyadhik Sevan

Faulty Food After Diarrhoea

Psychological

Agnimandya

Grahanidushti (Aashryashri Bhav)

Dosha Sanchay (Kledak Kapha, Samanvayu, Pachakpitta) — Sanchay

Consistant Nidan Sevan

Increased Agnimandya

Laghu Anna api na pachati

Adhik Dosh Sanchay in Grahani — Prakop

Saman Vayu Prerak — Prasar

Indigested Food in Grahani (Khavaigunya) — Sthansaunshraya

Due to prakop of Apanvayu after dosha sanchay
Symptom of Atisrushti, Atidrav
or constipation if dosh sanchay doesn't occur — Vyakta

Pitabh, Neel Pitabh, Drav Mala Pravrutti, Pooti Amlodgar, Hrut Daha, Kantha Daha, Aruchi Trushna — Bheda

MODERN TEXT LITERATURE

Tropical sprue or sprue syndrome and coeliac diseases described in modern text can be correlated with ayurvedic grahani roga. The symptoms of shataja & kshayaja grahani described by gananathasen is similar to that of intestinal tuberculosis

Tropical Sprue :

Psiliosis or Sprue is a tropical disease of unkown etiology associated with dearrangement of gastrontestinal tract characterised by a inability to absorb efficiently fat, glucose & calcium.

The site of this complication is small intestine because the digestion of fats by the Bile Juice & Pancreatic Juice occurs in the small intestine. This complication can be compared with the 'Agnidushti' mentioned in the samanya samprapti of grahani roga.

Sprue :- Characteristic features.

1. Apyrexial Morning Diarrhoea with Bulky, acidic, pale, frothy & fatty stool.
2. Inflammatory lesion in mouth.
3. Anaemia
4 Emaciation & wasting
5. Intestine flatulance
6. Peripheral Neuritis
7. Oedema of Feet in advanced cases.

Coeliac disease features :-

1. It is the sensitivity of Intestinal mucosa to gluten protein of wheat.
2. Diarrhoea,
3. Weight loss,
4. Anaemia,
5. Peripheral neuropathy,
6. Oedma.
7. Angular stomatitis,
8. Skin pigmentation
9. Dermatitis

In both above, the common histopathological finding is subtotal villous atrophy & Partial Villous atrophy respectively tropical sprue & coelic disease.

Some structural features, functional & infectional diseases of small intestine like DUODENITIS, CROHN'S disease etc. are common to that of grahani roga e.g. Muhurbhadham muhurdravam stoolhabit & constipation symptom.

PATHOPHYSIOLOGY OF STRUCTURAL CHANGES IN SMALL INTESTINE

Infection with Helicobacter pylori

1. (H. pylori) is the *most common cause* of ulcer. As a consequence, administration of antibiotics has been shown to be the most efficacious treatment in most ulcer patients not receiving nonsteroidal anti-inflammatory drugs. H. pylori probably survives the acidic environment of the mucus layer because it possesses a special urease. The bacterium uses this to produce CO2 and NH3, and $HCO3^-$ and $NH4^+$, respectively, and can thus itself buffer H^+ ions in the surroundings. H. pylori is transmitted from person to person, causing inflammation of the gastric mucosa (gastritis, especially in the antrum. A gastric or duodenal ulcer is ten times more likely to develop in such cases than if a person does not suffer from gastritis of this kind. The primary cause of such an ulcer is a disorder in the epithelium's *barrier function*, brought about by the infection. It is likely that, together with this ulcer formation due to the infection, there is also an increased chemical attack, as by *oxygen radicals* that are formed by the bacteria themselves, as well as by the leukocytes and macrophages taking part in the immune response, or by *pepsins*, because H. pylori stimulates pepsinogen secretion.

2. The fact that infection of the gastric antrum. Also frequently leads to duodenal ulcer is probably related to *gastrin secretion being increased* by the infection. As a result, acid and pepsinogen liberation is raised and the duodenal epithelium is exposed to an increased chemical attack. This causes *metaplasia* of the epithelium, which in turn favors the embedding of H. pylori, leading to *duodenitis* and increased metaplasia, etc.

3. A further common cause of ulcer is the intake of NSAIDs, for example, indomethacin, diclofenac, aspirin (especially in high doses). Their anti-inflammatory and analgesic action is based mainly on their inhibitory effect on cyclo-oxygenase, thus *blocking prostaglandin synthesis* (from arachidonic acid). An undesirable effect of NSAIDs is that they systemically block prostaglandin synthesis also in gastric and duodenal epithelia. This decreases $HCO3^-$ secretion, on the one hand (weakened mucosal protection and stops inhibition of acid secretion, on the other. In addition, these drugs damage the mucosa locally by nonionic diffusion into the mucosal cells (pH of gastric juice << pKa' of the NSAIDs). During intake of NSAIDs an acute ulcer may thus develop after days or weeks, the inhibitory action of these drugs on platelet aggregation raising the danger of bleeding from the ulcer.

4. Acute ulcers also occur if there is very severe stress on the organism (stress ulcer), as after major surgery, extensive burns, and multi-organ failure ("shock"). The main cause here is probably impaired blood flow through the mucosa correlated with high plasma concentrations of cortisol.

5. Often psychogenic factors favor ulcer development. Strong emotional stress without an outward "safety valve" (high cortisol levels) and/or disturbed ability to cope with "normal" stress, for example, in one's job, are the usual causes. Psychogenically raised secretion of gastric acid and pepsinogen, as well as stress-related bad habits (heavy smoking, antiheadache tablets [NSAIDs], high-proof alcohol) often play a part.

6. Smoking is a risk factor for ulcer development. A whole series of moderately effective single factors seem to add up here . Alcohol in large quantities or in high concentration damages the mucosa, while moderate drinking of wine and beer increases

gastric secretion through their nonalcoholic components. Rare causes of ulcer are tumors that autonomically secrete gastrin (*gastrinoma*, Zollinger– Ellison syndrome), systemic *mastocytosis*, or *basophilia* with a high plasma histamine concentration.

7. Apart from antibiotics and (rarely necessary) surgical intervention, the treatment of ulcer consists of lowering acid and pepsinogen secretion by blocking H2 and M1 receptors and/or of H^+/K^+ ATPase. Treatment with antacids acts partly by buffering the pH in the lumen, but also has further, as yet not fully understood, effects on the mucosa.

8. In malabsorption of carbohydrates, the reduced Na^+ absorption in the upper small intestine (diminished Na^+ symport with glucose and galactose) leads to reduced water absorption. The osmotic activity of the nonabsorbed carbohydrates additionally results inwater secretion. However, bacteria in the large intestine can metabolize up to 80 g/d (divided over four meals) of nonabsorbed carbohydrates into organic acids useful for providing energy that together with water are absorbed in the colon. It is only the large amounts of marked gas produced (flatulence) that provide evidence of carbohydrate malabsorption. However, if > 80 g/d (i.e., > 1D 4 of normal carbohydrate supply) is not absorbed or the intestinal bacteria are decimated by antibiotics, diarrhea occurs.

9. Secretory diarrhea (in the narrow sense) occurs when Cl^- secretion of the small intestinal mucosa is activated. Within the mucosal cells Cl^- is secondarily actively enriched by a basolateral $Na^+K^+2\ Cl^-$ symport carrier and is secreted via luminal Cl^- channels. These open more frequently when the intracellular concentration of CAMP rises. CAMP is formed in greater amounts in the presence of, for example, certain laxatives and bacterial toxins (Clostridium difficile, Vibrio cholerae). Cholera toxin causes massive diarrhea (up to 1000

mL/h) that can rapidly become life-threatening because of the loss of water, K^+, and $HCO3^-$ (hypovolemic shock, hypokalemia, nonrespiratory acidosis).

10. Overproduction of VIP (vasoactive intestinal peptide) by pancreatic islet cell tumors also causes high cAMP levels in intestinal mucosa cells leading to copious, life threatening diarrhea: pancreatic “cholera” or watery diarrhea syndrome. There are several reasons why diarrhea occurs after resection of the ileum and of part of the colon. Bile salts, normally absorbed in the ileum, cause accelerated passage through the colon (reducedwater absorption). In addition, the nonabsorbed bile salts are dehydroxylated by the bacteria in the colon. The resulting bile salt metabolites stimulate the secretion of NaCl and H_2O in the colon. Finally, there is also a lack of active absorption of Na+ in the resected intestinal segments.

1. Maldigestion and Malabsorption A defect in the processing and enzymatic splitting within the gastrointestinal tract is called *maldigestion*; a disorder of absorption is called *malabsorption*. As both of them are closely intertwined, they are grouped together here as malabsorption (in the wider sense).

2. Malabsorption may affect the three energy carriers of food, i.e., fats, proteins, and carbohydrates, as well as vitamins, iron, calcium, magnesium, and trace elements, for example, zinc.

3. Malabsorption of the enterohepatically circulating bile salts is also clinically significant. The respective site of absorption of these substances is determined by:

1) the number and duration of preceding steps of processing and splitting; and

2) the provision in the intestinal segments of specific mechanisms of absorption.

4. Thus, monosaccharides such as glucose and galactose can be absorbed at the beginning of the duodenum; disaccharides must first be split by the enzymes of the brush border; polysaccharides (just like proteins and fats) must first come into contact with pancreatic juice, with the result that they may not be absorbed until they reach the jejunum Rapid emptying of the stomach can mean that the place of absorption is moved distally, i.e. intestinal segments which lie further downstream can take over absorption that, in the long term, can lead to a change in the mucosa. The ileum, for example, may take on jejunum-like properties. This is not possible with substances for which only the terminal ileum possesses specific absorption mechanisms (cobalamine, bile salts).

5. Normal digestion and absorption consists of the following serial steps :

1. Mechanical processing of food (chewing, distal gastric peristalsis)

2. *Luminal digestion* (gastric, intestinal, and pancreatic juices; bile)

3. *Mucosal digestion* by enzymes of the brush border

4. *Absorption* by the mucosal epithelium

5. *Processing* in the mucosal cell

6. *Transportation* into blood and lymph

through which the absorbed substances reach the liver and the systemic circulation, respectively.

6. The causes of malabsorption can affect all these steps : After gastric resection and / or vagotomy, the stimulation of enteral hormone secretion (CCK, e.g.) is reduced and the synchronization of chyme apportioning with pancreatic secretion, gallbladder emptying, and choleresis is disturbed. Furthermore, passage through the small intestine is accelerated and the pH in the duodenal lumen is too acidic, so that the digestive process may be greatly disturbed (enzyme inactivation, bile salt precipitation). A gastrinoma (*Zollinger–Ellison syndrome*) can cause malabsorption for the same reason.

7. Pancreatic diseases, for example, chronic pancreatitis, carcinoma of the pancreas, cystic fibrosis , or resection of the pancreas may lead to malabsorption due to a *lack of important enzymes* (lipase, colipase, trypsin, chymotrypsin, amylase, etc.) as well as of $HCO3^-$ which is necessary for buffering acidic chyme.

8. Atrophic gastritis with achlorhydria will firstly diminish gastric digestion and secondly favor *colonization of the small intestine with bacteria*. This may also be caused by stasis in the small intestine due to diverticulosis or a small-intestine shunt (blind loop syndrome). The bacteria deconjugate bile salts and split the binding between cobalamine and intrinsic factor. The resulting cobalamine malabsorption leads to cobalamine deficiency, as does a reduced intake (strictly vegetarian diet; it is true also for breastfed infants of such

mothers, because their milk also lacks cobalamine), intrinsic factor deficiency (achlorhydria; see also, lack of enzymatic liberation of cobalamine from its binding with other proteins (high gastric pH, trypsin deficiency), or resection of the terminal ileum, the site of absorption of the cobalamine intrinsic factor complex.

9. Lack of brush-border disaccharidase causes malabsorption of the corresponding disaccharide. A lack of *lactase*, which splits lactose into glucose and galactose, is common. Lactase deficiency, which goes hand in hand with intolerance to milk and lactose containing foods, is rarely congenital, but often develops after weaning. There are marked ethnic differences.

10. Defects of specific mucosal carriers cause specific malabsorption. In Hartnup disease, for example, there is a specific carrier defect for certain neutral amino acids; in *cystinuria* for cationic (basic) amino acids and cystine. (The uptake of the affected amino acids as dipeptides is undisturbed, because the mucosa has its own carrier for dipeptides).

11. Global defects of mucosal digestion and absorption occur in *diffuse mucosal diseases*, such as celiac disease, tropical sprue, Crohn's disease, Whipple's disease, AIDS, infections (e.g., with Salmonella), *radiation enteritis*, and after *resection* of large portions of the small intestine.

12. In addition to alcohol (pancreatic insufficiency, chronic liver disease), a number of drugs cause malabsorption: *colchicine* (inhibits division of crypt cells and disaccharidases), *neomycin* and similar antibiotics (inhibit division of crypt cells and disaccharidases; precipitate bile salts and micellar fatty acids), *methotrexate* (inhibits folate absorption), *cholestyramine* (binds bile salts), certain *laxatives*, *biguanides*, etc.

13. Especially in fat absorption, processing within the mucosal cells (formation of

chylomicrons) is an important partial step whose disturbance in *abetalipoproteinemia* results in fat malabsorption. Another cause is *lymphatic blockage* (lymphangiectasia, lymphoma, etc.).

14. Finally, malabsorption naturally occurs if blood flow through the intestine is disturbed (ischemia, e.g., in vasculitis).

15. The consequences of malabsorption are dependent on the kind of malabsorbed substance: Malabsorption of proteins can lead to *muscular atrophy* and *weight loss*, while any resulting hypoproteinemia will result in *edema.* Malabsorption of carbohydrates in the small intestine means that some of them are metabolized to *short-chain fatty acids* and to gases (CO2, H2) resulting in distension and flatulence. If more than 80 g/d of carbohydrates fail to be absorbed, osmosis- induced watery *diarrhea* occurs.

16. Malabsorption of fats is characterized by fatty stools (*steotorrhea*) and leads to *weight loss* from a lack of these high-calorie components of food. Malabsorption of the fat-soluble vitamins A, D, E, and K occurs especially if fat malabsorption is caused by a *lack of bile salts* or by other reasons of *abnormal formation of micelles*.

17. This is because these vitamins can only reach the absorbing mucosa in an uninterrupted lipophilic milieu for which micelles are essential.

18. If vitamin K deficiency occurs, the glutamyl residues of prothrombin and other blood clotting factors cannot be carboxylated in the liver, and thus *bleeding* may occur. Vitamin D deficiency causes *rickets* in children and *osteomalacia* in adults. In vitamin A deficiency *hyperkeratosis* and *night blindness* develops.

MATERIAL AND METHODS

MATERIAL

1. Compilation of all references about the term grahani from classical ayurvedic text (Samhitas) and contemporary other literature was done.

2. Information from modern text about structures co-relating the different concept of grahani mentioned in ayurveda was collected.

3. Attempts were made to visualize and see the structures co-relating grahani by cadaveric dissection method.

4. A consecutive 109 patients who where between the age group 16 to 60 irrespective of sex, who fulfilled the following criteria was enrolled in our study.

5. **Inclusion Criteria :** The patients were selected after preliminary diagnosis of Grahani Roga with the help of questionnaire prepared based on samanya lakshanas of Grahani Roga as per Madhav Nidan and again patients representing classical symptoms of Pittaj Grahani Roga were selected.

6. **Exclusion Criteria :-** Patients with acute conditions like Haematemesis, Anorectal Bleeding and major Systemic and Chronic illness such as Koch's, CA, HIV, HBsAg+ve patients were excluded from the study.

METHODS

Informed consent :

The patients under going this study were informed about the nature of study and written consent of each patient was taken.

1. Detailed case history was noted down with his present illness and was analysed

2. Patients Abhyavaran shakti, Jaran Shakti , Agni parikshan and koshta parikshan was done with the help of prashna pariksha.

3. All selected patients were asked about their stool frequency, volume, consistence, presence of blood/mucus, borborygmi, abdominal pain, upper GI symptom and degree of weight loss.

4. All patients underwent blood investigations like complete haemogram with blood indices and erythrocyte sedimentation rate.

5. We performed Esophagogastroduodenoscopy and collected four biopsy samples from second part of duodenum(D2) and one from the duodenal bulb from all the patients. The status of duodenal folds were recorded(normal, attenuation, scalloping, of mucosal folds). Colonoscopy with ileal intubation was performed in selected patients. Histological grading of D2 biopsies were given according to modified Marsh criteria.

6. Since the cardinal symptoms of grahani roga is MUHURBADHA MUHURDRAVAM mala parikshan was done by following methods. :

A) Bhautik parikshan :

a) Jalanimajjan : To find whether the stool gets submerged or floats on the water. While doing this test the stool sample was put in the testube containing distill water and observed whether it gets submerged of floats on the water.

b) Sahanan : To know the consistency of the stools.

Gross examination of stool with the naked eye was done to know consistency of stool (Formed, soft, liquid, semi solid, semi liquid, watery).

B) Three consecutive samples were sent to detect opportunistic parasites. Laboratory examination was done in which macroscopic and microscopic examination of stools was done.

Laboratory diagnosis of parasitic infection was based on the gross examination of the specimen and viewing it under microscope.

Some of the identifying characteristic based on gross examination was.

1. Large round worm - Ascaris lumbricoides -

   Pinkish in colour 0.3 - 0.5cm thick and 15cm long

2. Pin worm - (enterobius vermicularis) - White in colour and 1cm long

3. Adult Helminth Like Trichuris Trichiura, Hook worm (Ancylostoma Nectator) and dwarf tapeworms (hymenolepis nana) was found in stool but usually seen only after medication.

4. Presence of Fresh Blood and Mucus was seen in liquid stool which is suggestive of amoebic dysentry. It was confirmed under microscope.

Microscopic Examination

Microscopic examination of stool was necessary to identify helminth eggs and larvae as well as protozoan cysts and trophozoites. This was done by direct wet mount of fresh stool, preserved specimen or faecal concentrates.

6. With the help of trividha pariksha detailed examination of Annavaha and Purishvaha strotas was done.

TRIVIDHA PARIKSHAN

Samanya Parikshan - Nadi, Mala, Mutra, Jivha etc. was done.

Indriya Parikshan

Auscultation	Shravanendriya Parikshan	- Antrakujan
Inspection	Chakshurindriya Parikshan	- Age of patients
		Colour of abdomen normal / abnormal
		The aligment of the internal organs of abdomen.
		The upachaya / apachaya of muscles of abdomen.
Smelling	Ghranendriya Parishan	- Foul smell
Palpation	Sparshnendriya Parishan	- The feel of the skin of abdomen Cold - Hot, Soft - Hard, Rough - Smooth, Cyst - Abnormal Growth
Percussion	Akotan- Tenderness rigidity guarding sign	

Darshan Parikshan (Inspection)

1. The colour of the skin of abdomen

Whitish / Yellowish

When there is worm infestation in the patient we find discolouration of skin under face rather on abdomen.

2. The colour of tongue

Sama-shwetavarniya - constipation.

3. Swelling on cheeks and eyesocket Most found in kaphaj grahani

4. Malavarna

Greenish , Neelpeet - Pittaj grahani

Phenyukta - Vataj grahani

Shweta, Sandra - Kaphaj grahani

5. The colour of vomitus

Coffeeground, Phenyukta - Chhardi seen in Vataj grahani

Greenish yellowish -Chhardi seen in Pittaj grahani.

Whitish ghana - Chhardi seen in Kaphaj grahani

6. Increase in the shape of abdomen

Tensness on the abdomen - Gaseous abdomen,

abdomen filled with fluid

7. Weight loss and Muscle wasting

Seen in vataj and sannipataj grahani

Sparshan Parikshan (Palpation)

1) Abdomen -

Skin - Decreased tone of the skin - Severe Loose motion

PAIN IN ABDOMEN

(Tenderness)

Pain in Epigastric Region	-	Amlapitta, Agnimandya, Chhardi, Indigestion
Pain in Hypogastric Region	-	Constipation, Krumi.
Pain in Umbilical Region	-	Grahani, Krumi, Pravahika.
Pain in Left Hypochonric Region	-	Agnimandya
Pain in Left Lumbar Region	-	Atisar Pravahika

AKOTAN

(Percussion)

Baddha Dhvani	-	Indigestion
Guda-gudahat	-	vistabdha ajirna (Indigestion)
Antrakoojan	-	Amlapitta

LAKSHANAS

The lakshanas found in different types of grahani roga was studied in the following ways :

Daurbalya :

१) बलहीनता.

(चक्र टीका/ च. सू. १६/७)

२) शरीर बलहानि, मांसापचय :

(चक्र / च. नि. १/३३)

३) दुर्बलस्यभाव :

षञ् । वाचस्पतिमिश्र.

Bala is explained by the ability to do the exercise (vyayam shakti) or the ability to bear the stress from Apatapraman. As per the comentators wasting of muscles (maunsapachaya) is considered as daurbalya in vatapradhan grahani and the ability to bear the the stress is considered in kaphapradhan grahani. (आकृशस्यार्पि दौबल्यम) Which is done through the prashana pariksha.

Here not only maunsapachaya but also apachaya of dhatus like shukra & ojha is considered as duarbalya.

Gaurav :

कायस्य गौरवम् :

Heaviness in the body, feeling of wet blanket, heaviness in particular organ. Amautpatti because of agnimandhya in the rasadhatu causes heaviness in the body.

Parshva Uru Vankshan Greevaa Ruka :

रुक् पीडा, साच पार्श्वदिभिः संबध्यते ।

मधुकोष आ. दर्पण.

Pain in ribs, thighs, hip joint, neck.

Karshya :

१) कृशता, उपशोषित अल्परस धातुजन्य मांसहीनं शरीरं मांसक्षयो वा ।

सु. नि. ३/१७

२) रसदोषज विकारेषूएक :

सु. सू. २४/९

३) रसनिमित्तमेव स्थौल्यं कार्श्यंच।

च. सू. २१-१४-१४

Karshya means lean. The person who has less muscles on the body due to decrease in rasa dhatu & maunsadhatu is called as Karshya.

Adhamana

१) आध्मानं जीर्णं जीर्यतिच 'अन्ने' इतिशेषः । मधुकोष.

२) आध्मानं जीर्णं जीर्यंतिच सतिभक्ती 'अन्ने' इतिशेषः । आ. दर्पण.

During or after digestion the abdominal discomfort caused due to accumulation of flatus (vayu), is said to be adhamana. The dryness in small intestine is due to accumulation of flatus. It is increased two hours after having food and in evening period between four to six. It is reduced by udarsavahan and abhyantar snehapan.

MALAPRAVRUTTI

Amayukta :

Undigested food components are observed in stools with foul smell sometimes hard substance which in normal condition gets digested but here it comes through the stools as it is.

Dravayukta :

In vataj grahani 'वृध्दीः सर्व रसानां' this symptom should be related to the above. The type of food which the person eats has an effect on the stools. For e.g. after drinking lots of sugarcane juice it is noticed that the person has dravayukta malapravrutti. Therefore the above symptom should be related with the type of food taken.

Phenyukta :

Frothy loosemotion this symptom should be related with the type of food in take e.g. palak, spicy food, dalada increases the above symptom.

Bhinnam Shelshma Santushta :

भिन्नाम श्लेष्मसंतुष्ट :- भिन्नं च तद् आमश्लेमाभ्यां संसष्ट चेतिसमासार्थ ।। मधुकोष

Motions with scattered mucoid stools in which undigested food components with mucus are observed.

Hrullas :

ह्रल्लास : ह्रदयस्य उत्क्लेशनम् । (सु.उ. ५६/२१/२२)

१) अन्तरवस्थितदोषाणां - मितस्ततः संचलनं न पुनस्तत्प्रवृत्ति :

(सु.उ. ३९/१०१)

mouth brush symptoms is seen along with varied gustatory sensation.

Chhardi :

छर्दी : १) वान्ति वमनम् । च. चि. २०.५

आमाशयात मुखमार्गेण दोषाणां बहिर्गमनम् ।

Emesis of the doshas.

Madhur Udgar

मधुर उद्गार : मधुरः मधुरत्वेनोपलक्षित उद्गारः ।

मधुकोश आतंक दर्पण

belching with sweet taste.

Pooti Amlodgar

पूति अम्ल उदगार :- सधूमोदगार :।

आतंक दर्पण

पुति दुर्गंध

(च. वि. ८-९७)

belching with sour taste.

Vairasya :-

वैरस्य :- विरुद्धरसास्यता । - Disturbed (Abnormal) taste.

Kantha shosh daha :-

कंठशोष दाह - burning sensation in throat

Trushna :-

तृष्णा - thirst

bowel sounds :-

Burning sensations at anal, cardiac, abdominal & forehead region :-

- गुदह्रदय उदर मस्तक दाह

Tastelessness :-

अरोचक

DISSECTION

METHOD OF DISSECTION

A) Midline incision was taken from xiphoid process to pubic symphysis

B) Skin was dissected laterally to expose the superficial fatty layer of superficial fascia.

C) Superficial fatty layer along with deep membranous layer was dissected and reflected laterally to expose the muscles of anterior abdominal wall.

D) The external oblique, internal oblique and transversus abdominis were reflected from the lateral aspect to the medial side.

E) Anterior rectus sheath, rectus abdominis muscle and posterior rectus sheath were released from its attachment on the sternum and reflected down wards to expose the peritoneum.

F) The peritoneal cavity was opened by making a vertical and transverse incision.

G) In the right upper flap the falciform ligament was identified extending on to the Liver.

H) The cord like ligamentum teres (obliterated umbilical vein) was felt in its free edge.

I) The falciform ligament was traced to the Liver, where it divides the Liver, into right and left lobes.

J) The hand was inserted over the right lobe of Liver, between the Liver and the diaphragm and the superior coronary ligament was felt.

K) Similarly the inferior layer of coronary ligament was felt by inserting the hand under the right lobe of Liver, and pushing it upwards between posterior surface of the Liver and the diaphragm.

L) The left lobe of the liver was gently lifted up to expose the stomach and lesser omentum, which contains Common bile duct, Hepatic artery, and Portal vein in its free border.

M) These structures were exposed by peeling of the peritoneum with the forceps.

N) On cutting these stuctures the inferior vena cava was seen forming the posterior boundary of epiploic foreman.

O) The inferior vena cava was cut close to the Liver.

P) The falciform ligament, coronary ligament and left traingular ligment which connect to the Liver with diaphragm were cut.

Q) The Liver was gently pushed downwards and another cut was made on the inferior vena cava just below the diaphragm and the Liver was removed.

R) To study the small intestine an attempt was made to see its internal stuctural.

S) In which the pyloric sphincter & transverse colon was raised to see the initial part of DUODENUM and its relation with the Pancreas & Gall Bladder.

T) The mesentry of the small intestine was exposed by turning the transverse colon and its mesentry upwards.

U) Small intestine was turned to the left and was cut through the right layer of peritoneum of the mesentry along the line of its attachment to the posterior abdominal wall and stripped from the mesentry.

V) Fat was removed from the mesentry to expose the superior mesentric vessels in its root and their branches in the mesentry.

W) Numerous lymph nodes were noted along the superior mesentric plexus surrounding the vessels.

X) To visualise the internal structure of duodenum a four inch longitudinal incision was taken and following structures were seen.

(1) Common bile duct which was checked by probing.

(2) Finger shaped projection called villi were seen coming from mucosal membrane.

Y) A pair of ligatures were tied round the jejunum close to duodenojejunal flexure and another pair around the ileum close to the caecum and small intestine was cut between each pair of ligature and removed by dividing the mesentry close to the intestine

Z) The piece of intestine was washed with water and opened logitudinally to see the following stuctures :

(a) The larger diameter and thickness of the jejunum was noted.

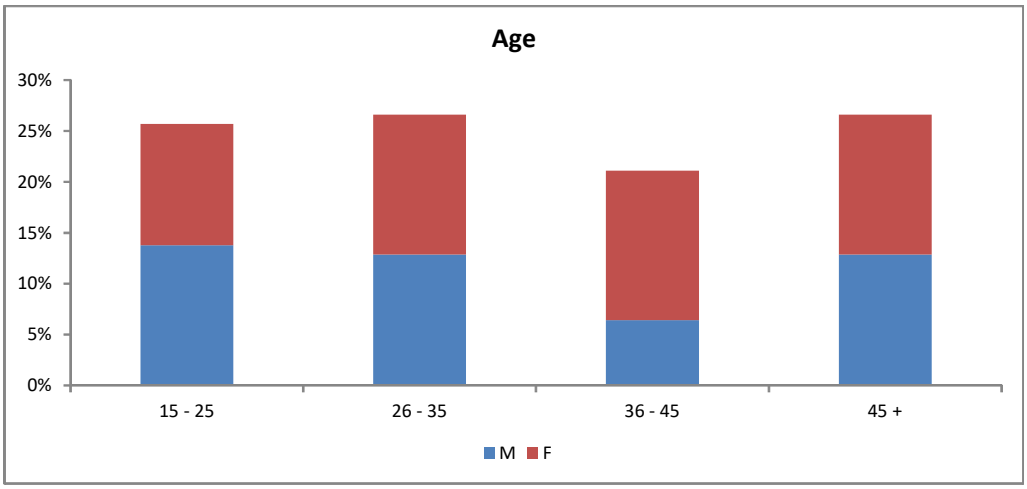
(b) Transverse circular folds of whole thickness of the mucous membrane were seen which became progressively smaller and less numerous from the proximal jejunum to the

terminal ileum where they were missing.

(c) The internal surface was covered with finger shaped projection called villi visualised with the help of hand lens which were large and numerous in the jejunum than in ileum but aggregate lymph follicles were seen in ileum.

AGE AND SEX WISE DISTRIBUTION OF PATIENTS

Age	M	F	Grand Total
15-25	13.76%	11.93%	25.69%
26-35	12.84%	13.76%	26.61%
36-45	6.42%	14.68%	21.10%
45+	12.84%	13.76%	26.61%
Grand Total	**45.87%**	**54.13%**	**100.00%**



1. By going through the table it is seen that 25.69 % patients were from the age group 15 to 25 out of which 13.76% were male & 11.93% were female

2. 26.61% patients were from age group 26 to 35 out of which 12.84% were male & 13.76% were female.

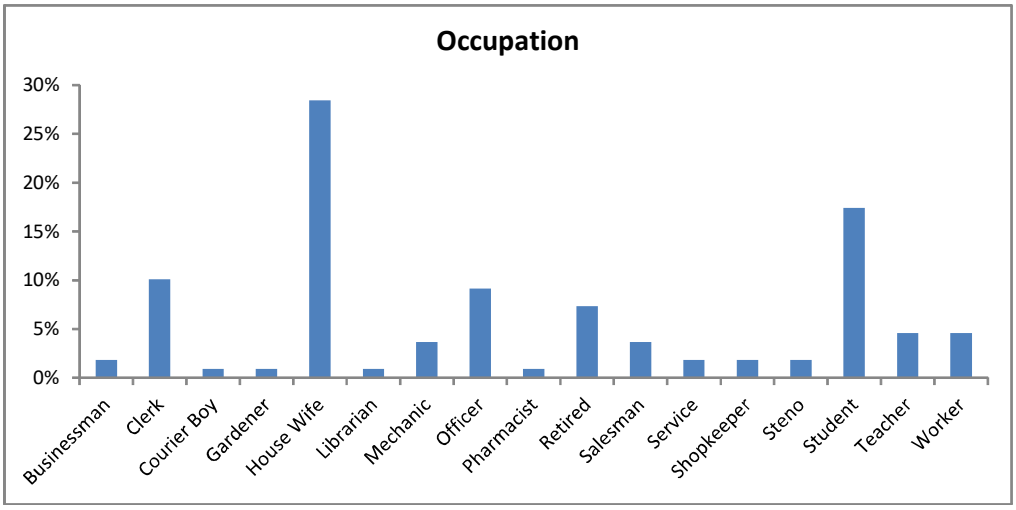
3. 21.10 % patients were from age group 36 to 45 out of which 6.42% were male & 14.68% were female.

4. 26.61% patients were from age group 45+ out of which 12.84% were male & 13.76% were female.

It is seen that maximum i.e. 52.30% were in the age group of 15 to 35 belonging to young and middle age people which indicate that persons during this age normali ignore the dietic codes.

OCCUPATION WISE DISTRIBUTION OF PATIENTS

Occupation	Percent	Count
Businessman	1.83%	2
Clerk	10.09%	11
Courier Boy	0.92%	1
Gardener	0.92%	1
House Wife	28.44%	31
Librarian	0.92%	1
Mechanic	3.67%	4
Officer	9.17%	10
Pharmacist	0.92%	1
Retired	7.34%	8
Salesman	3.67%	4
Service	1.83%	2
Shopkeeper	1.83%	2
Steno	1.83%	2
Student	17.43%	19
Teacher	4.59%	5
Worker	4.59%	5
Grand Total		109

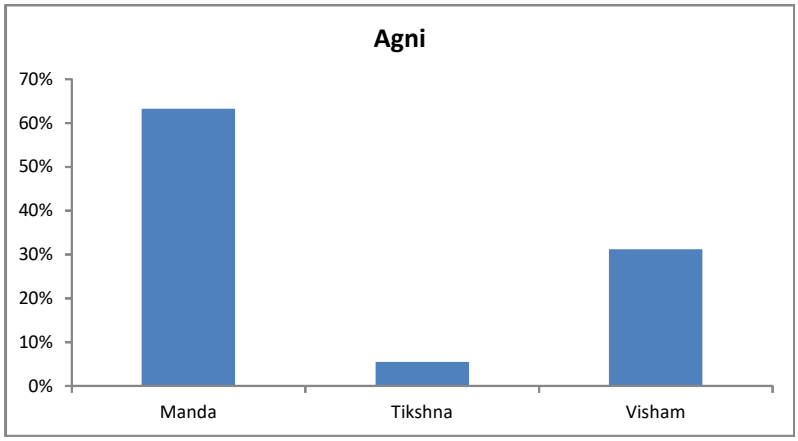


1. Occupation wise distribution suggest that Housewives 28.44% are more prone to grahani roga as they have faulty food habits and do not follow any dietic codes followed by Students, Clerks, Officers and 17.43%, 10.09% and 9.17% respectively, who do not it their food at regular time due to their busy schedule.

2. Teachers and Workers 5.00% each were reported with grahani roga were as 2.00% of Steno, Shopkeepers and Salesman were reported with grahani roga.

AGNI WISE DISTRIBUTION OF PATIENTS

Agni	Percentage	Number
Manda	63.30%	69
Tikshna	5.50%	6
Visham	31.19%	34
Grand Total		**109**



ANALYSIS OF AGNI :-

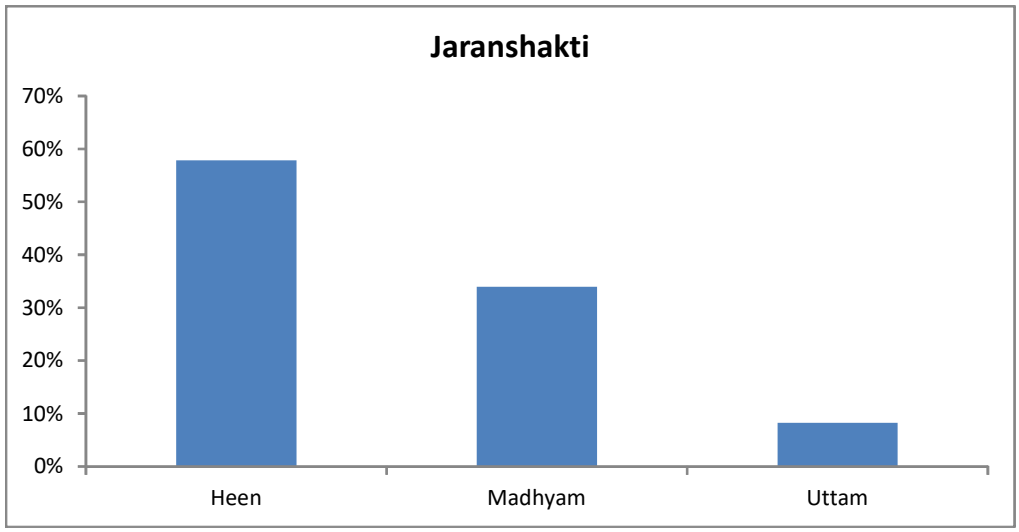
1. 63.30% patients of pittaj grahani were having mandagni

2. Vishamagani was seen in 31.19 % of patients of pittaj grahani.

3. 5.50% patients of pittaj grahani were having tikshnagni.

JARANSHAKTI WISE DISTRIBUTION OF PATIENTS

Jaranshakti	Percentage	Number
Heen	57.80%	63
Madhyam	33.94%	37
Uttam	8.26%	9
Grand Total		**109**



ANALYSIS OF JARANSHAKTI GRAPH :-

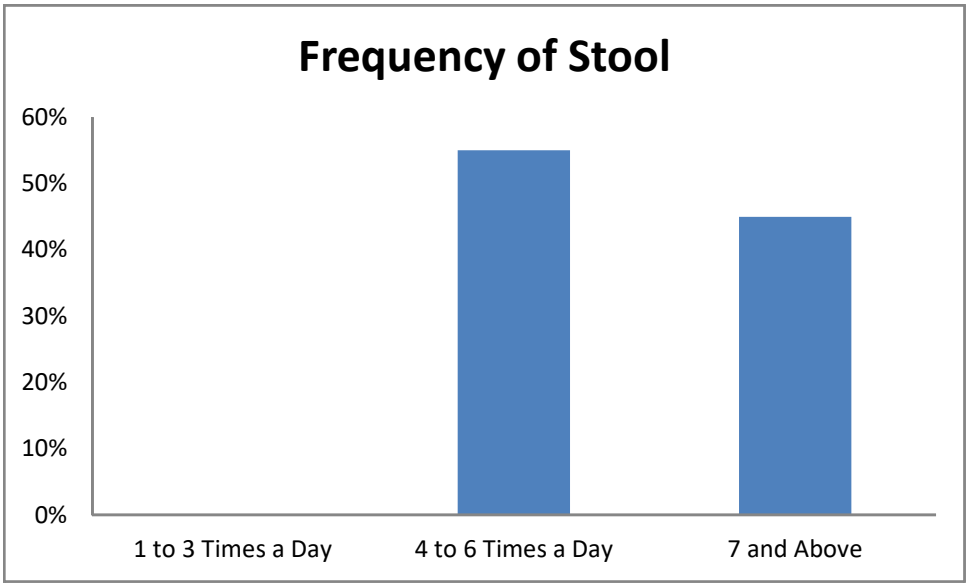
1. 57.80% patients of pittaj grahani were having Heen Jaranshakti.

2. Madhyam Jaranshakti was found in 33.94% of pittaj grahani patients.

3. 8.26% patients of pittaj grahani were having Uttam Jaranashakti.

DISTRIBUTION OF FREQUENCY OF STOOL

Frequency of Stool	Percentage
1 to 3 Times a Day	0%
4 to 6 Times a Day	55.03%
7 and Above	44.97%



1. Each group consisting of 3 frequencies.

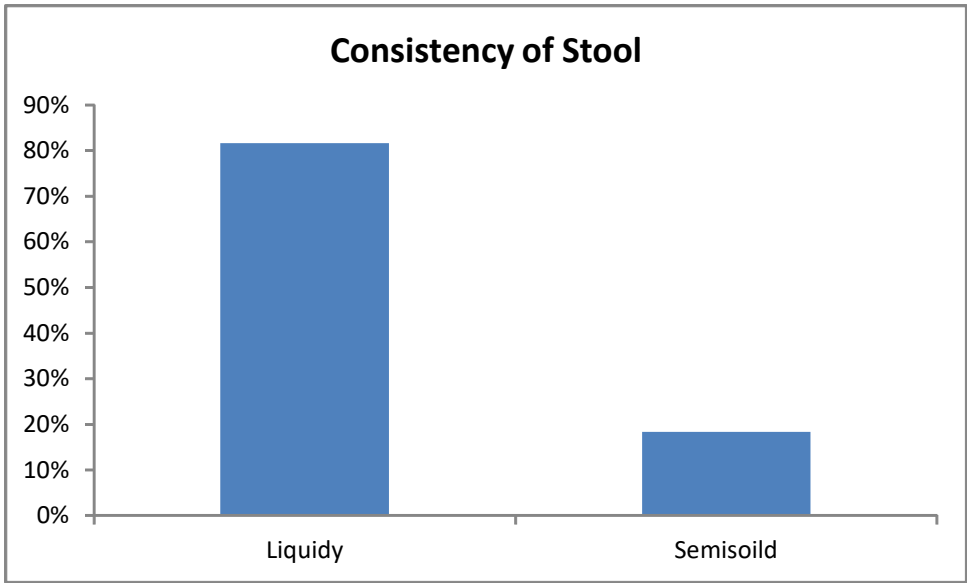
2. By going through the table it seen that 0.0% of the patients were having frequency of stools between 1 to 3, & 55.03% had the stools frequencied between 4 to 6, While 7& above stool frequencies were recorded in 44.97% of the patients.

3. Thus the patients of grahaniroga practically shows increased frequency averaging to 4 to 6.

✦ This is just showing the tendency of the disease.

DISTRIBUTION OF CONSISTENCY OF STOOL

Consistency of Stool	Percentage	Count
Liquidy	81.65%	89
Semisoild	18.35%	20
Grand Total		**109**



Appearance of stool was classified in to 2 categories semisolid & Liquidy.

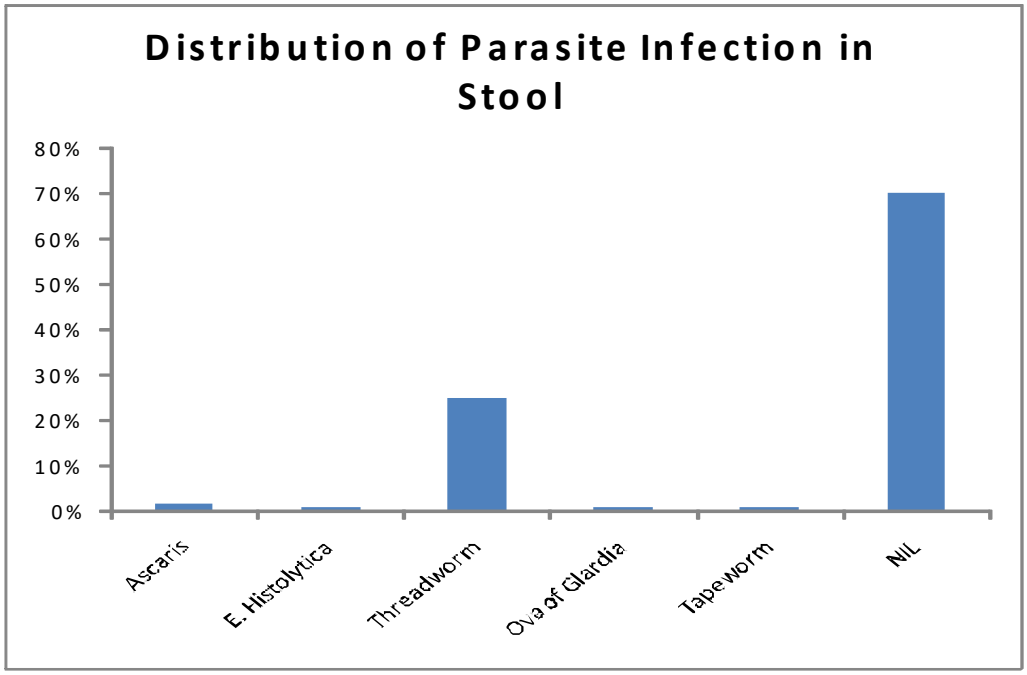
1. Liquidy stool were seen in 81.65% in pittaj grahani patients.

2. Semisolid stool were seen 18.35% in pittaj grahani patients.

- Above findings shows that the patients of grahani roga mostly Liquidy has appearance of stool which can be co-related with the cardinal symptom of grahaniroga which is muhurbadha muhurdrava malapravrutti.

DISTRIBUTION OF PARASITE INFECTION IN STOOL

Parasite Infection in stool	Percentage
Ascaris	2%
E.Histolytica	1%
Threadworm	25%
Ova of Glardi	1%
Tapeworm	1%
Nil	70%



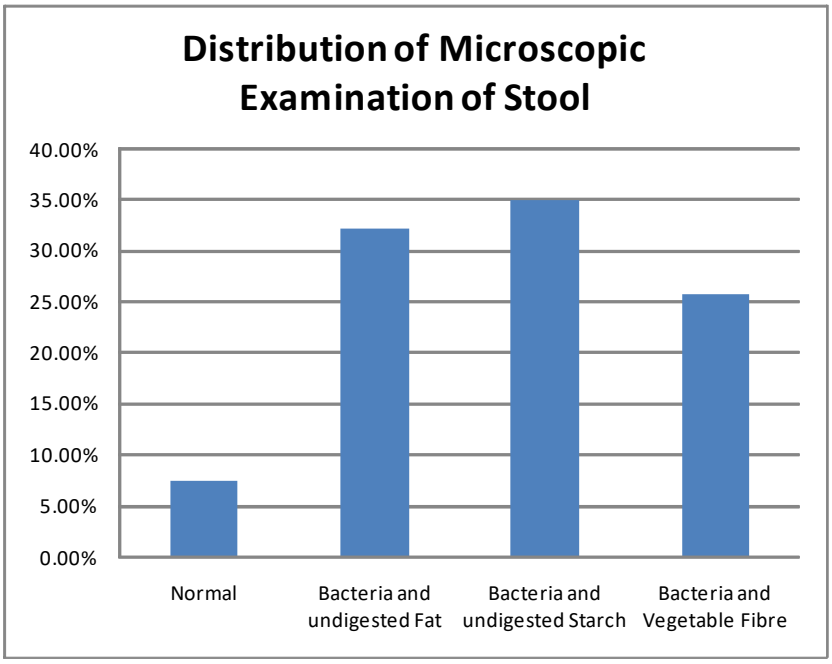
1. On the microscopic examination of stool it was observed that 25% patients of pittaj grahani patients had helminthes in their stool i.e. Thread worm 2%, and ascaris (2%), tape worm 1% and protozoas like Ova of Giardia (1%) & E-Histolytica (1%).

2. 70% patients of pittaj grahani were found with no worm infestation.

This suggest that worm infestation is one of the cause of grahani roga.

DISTRIBUTION OF MICROSCOPIC EXAMINATION OF STOOL

Microscopic Examination of Stool	Percentage	Count
Normal	7.33%	8
Bacteria and undigested Fat	32.11%	35
Bacteria and undigested Starch	32.11%	38
Bacteria and Vegetable Fibre	25.69%	28
Grand Total		109



1. In the microscopic examination of stools four specialties were observed in the stools.

2. By going through the table it is seen that 32.11% of the patients were having Bacteria and undigested fat .

3. It is seen that 34.87% of the patients were having Bacteria and undigested starch.

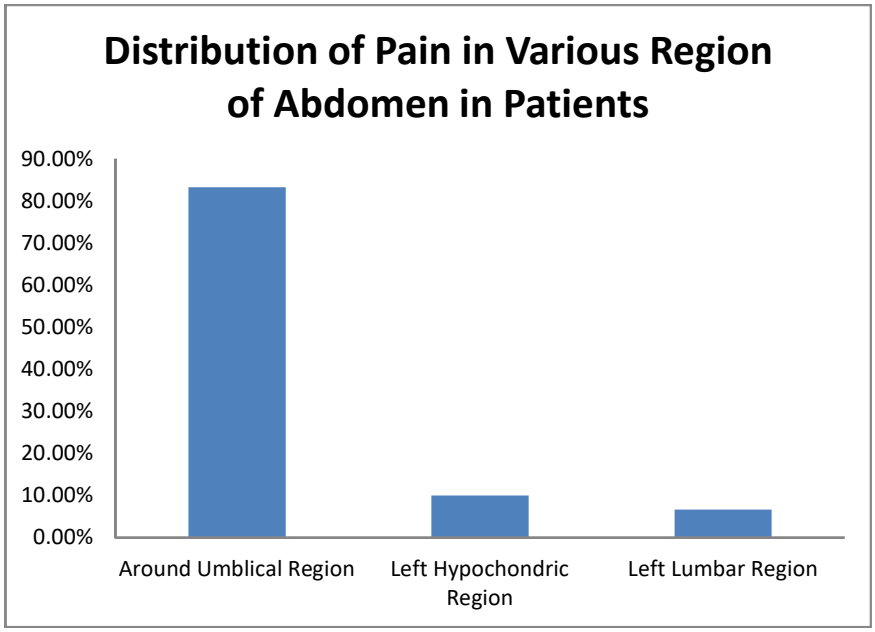
4. It is seen that 25.69% of the patients were having Bacteria and vegetable fibre.

5. All most every patient had bacteria in their stool.

6. Presence of these undigested substances in the stools, is suggestive of the weakness of the digestive process i.e. (Jaranshakti).

DISTRIBUTION OF PAIN IN VARIOUS REGION OF ABDOMEN IN PATIENTS

Region of Pain in Abdomen	Percentage
Around Umblical Region	83.33%
Left Hypochondric Region	10%
Left Lumber Region	6.67%



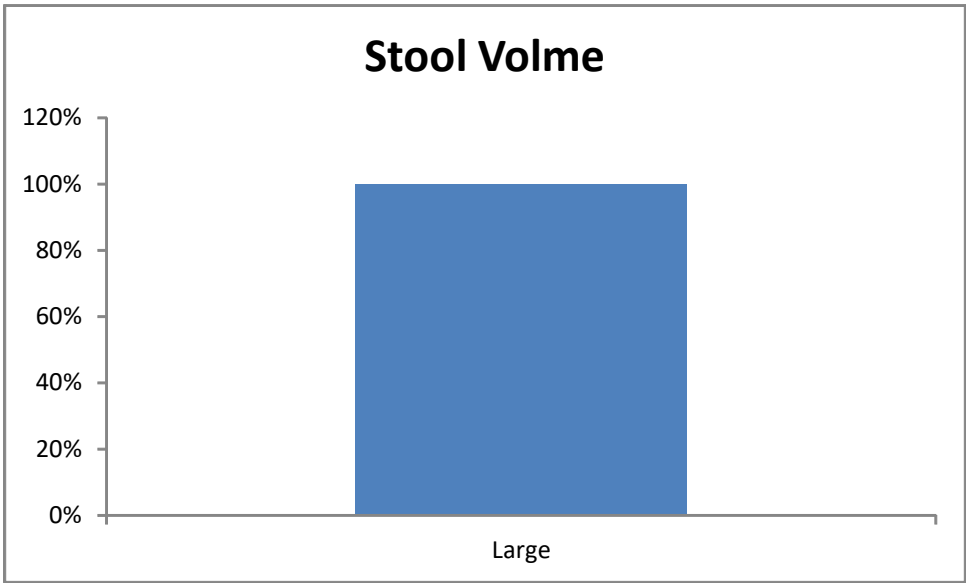
1. By going through the table it is seen that 83.33% of the patients were having pain around umbilical region .

2. 10% patients of pittaj grahani had pain in left hypochondric region.

3. 6.67% patients of pittaj grahani had pain in Left Lumbar region.

DISTRIBUTION OF STOOL VOLUME

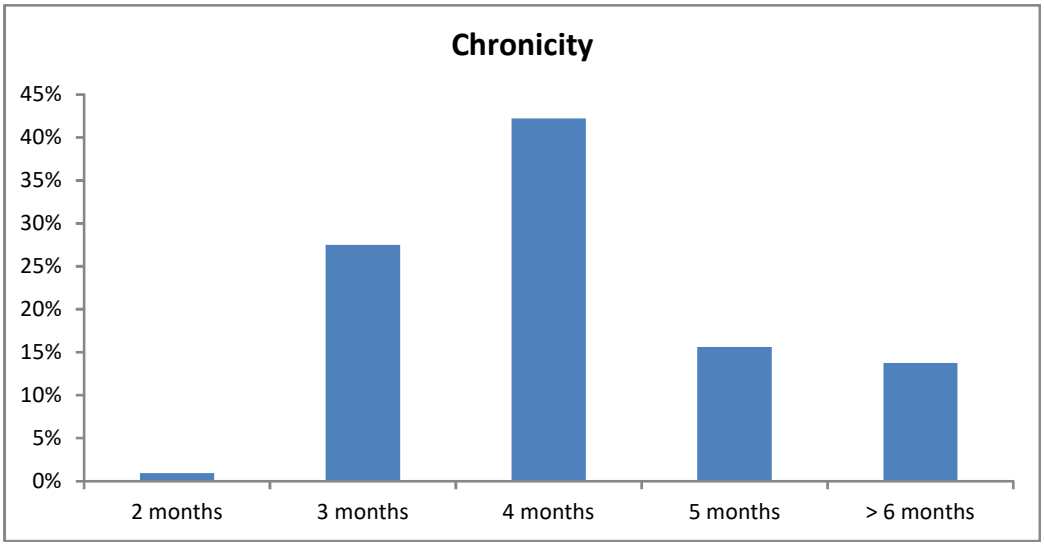
Stool Volume	Percentage	Count of Sr. No.
Large	100%	109
Grand Total		**109**



All the patients of pittaj grahani had large volume of stool.

CHRONICITY WISE DISTRIBUTION OF PATIENTS

Chronicity	Percentage	Count
2 months	0.92%	1
3 months	27.52%	30
4 months	42.20%	46
5 months	15.60%	17
> 6 months	13.76%	15
Grand Total		**109**



1. By going through the table it is seen that 42.20% of the patients were having disease from 4 month.

2. 27.52% patients of pittaj grahani had disease from 3 months.

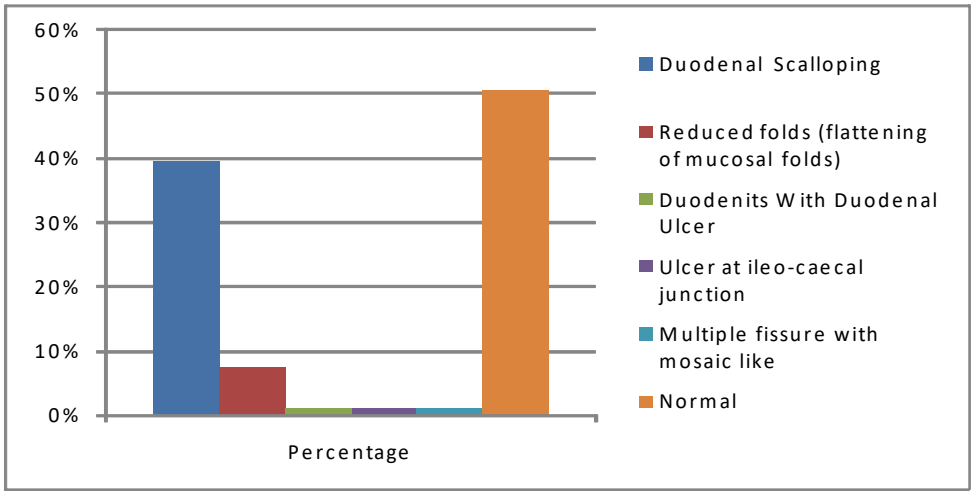
3. 15.60% patients of pittaj grahani had disease from 5 months.

4. And 13.76% patients of pittaj grahani had suffered from disease from more than 6 months and 0.92% of patients suffered from 2 months.

While considering the chronicity of PittajGrahni Roga it is absorved that 80% patients were having symptoms of Grahani Roga from 3 months to 7 months median duration of symptoms was 4 months.

DISTRIBUTION OF STRUCTURAL CHANGES IN GRAHANI (SMALL INTESTINE) OF SELECTED PATIENTS

Structural Charges in Grahani Roga	Percentage	Count
Duodenal Scalloping	39%	43
Reduced folds (Flattening of mucosal folds)	7.34%	8
Duodenits with Duodenal Ulcer	0.92%	1
Ulcer at ileo-caecal junctuion	0.92%	1
Multiple fissure with mosaic like	0.92%	1
Normal	50.45%	55



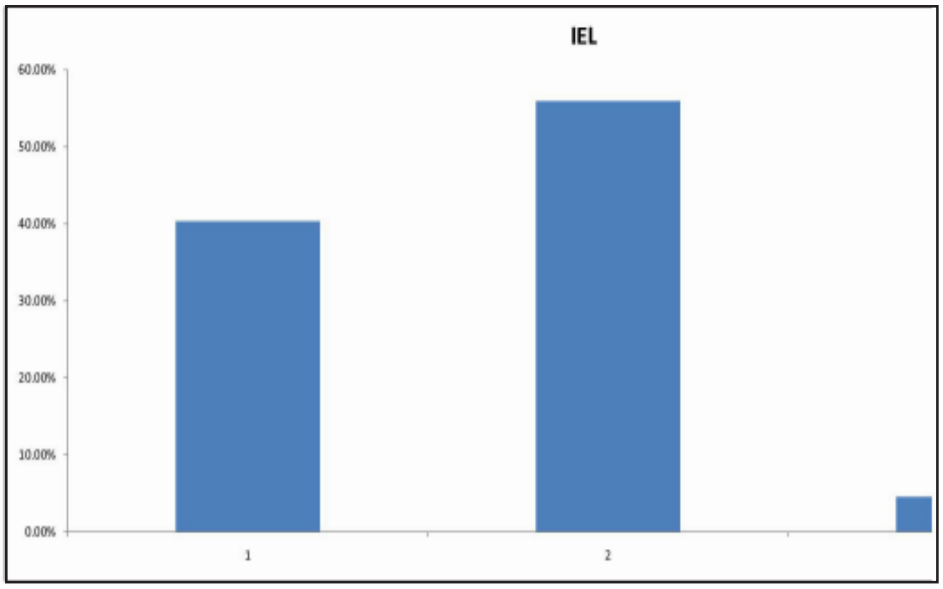
By going through the table in the pittaj grahani the following structural changes where seen.

1. Duodenal Scalloping 39.45%
2. Reduced folds (flattening of mucosal folds) of mucosal folds 7.34%
3. Duodenits With Duodenal Ulcer 0.92%
4. Ulcer at ileo-caecal junction 0.92%
4. Multiple fissure with mosaic like appearance with mucosal nodularity 0.92 % and 50.45% of patients showed Normal scopies.

DISTRIBUTION OF PATHOLOGICAL CHANGES IN GRAHANI (SMALL INTESTINE) OF SELECTED PATIENTS

INTRAEPITHELIAL LYMPHOCYTOSIS WISE DISTRIBUTION

IEL	Percentage	Count
WNL	40.36%	45
upto 30	55.96%	59
30-50	4.58%	5



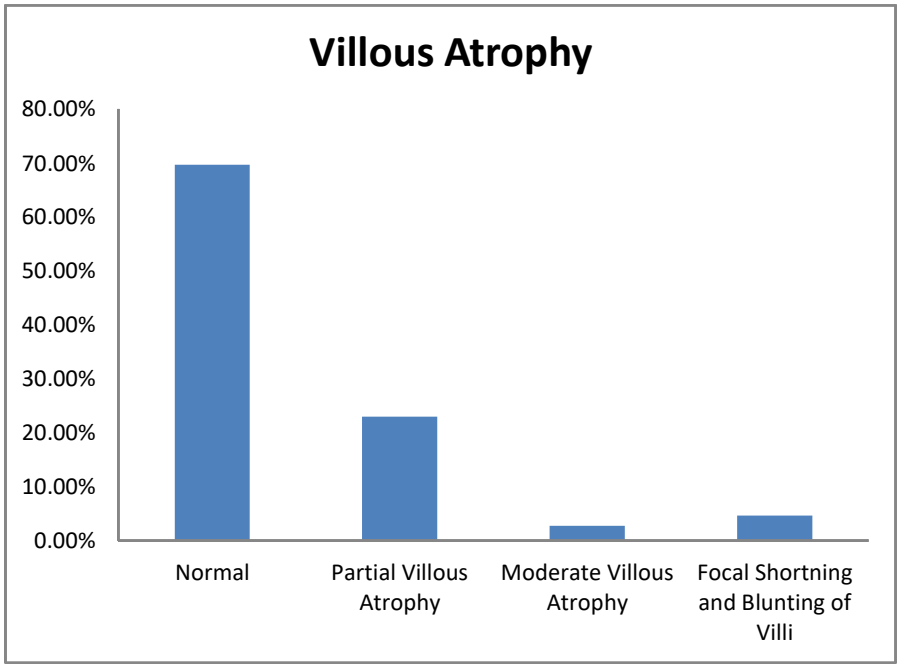
By going through the table in the pittaj grahani the following distribution of IEL (pathological changes) where seen.

1. 55.96% showed borderline (values between 26 and 29 per 100 epithelial cells) Intraepithelial Lymphocytosis
2. 4.58% showed significant Intraepithelial Lymphocytosis (>30 per 100 epithelial cells)
3. 40.36% showed Lymphocytes epithelial within normal limits.

DISTRIBUTION OF PATHOLOGICAL CHANGES IN GRAHANI (SMALL INTESTINE) OF SELECTED PATIENTS

VILLOUS ATROPHY WISE DISTRIBUTION

Villous Atrophy	Percentage	Count
Normal	69.72%	76
Partial Villous Atrophy	22.93%	25
Moderate Villous Atrophy	2.75%	3
Focal Shortning and Blunting of Villi	4.58%	5



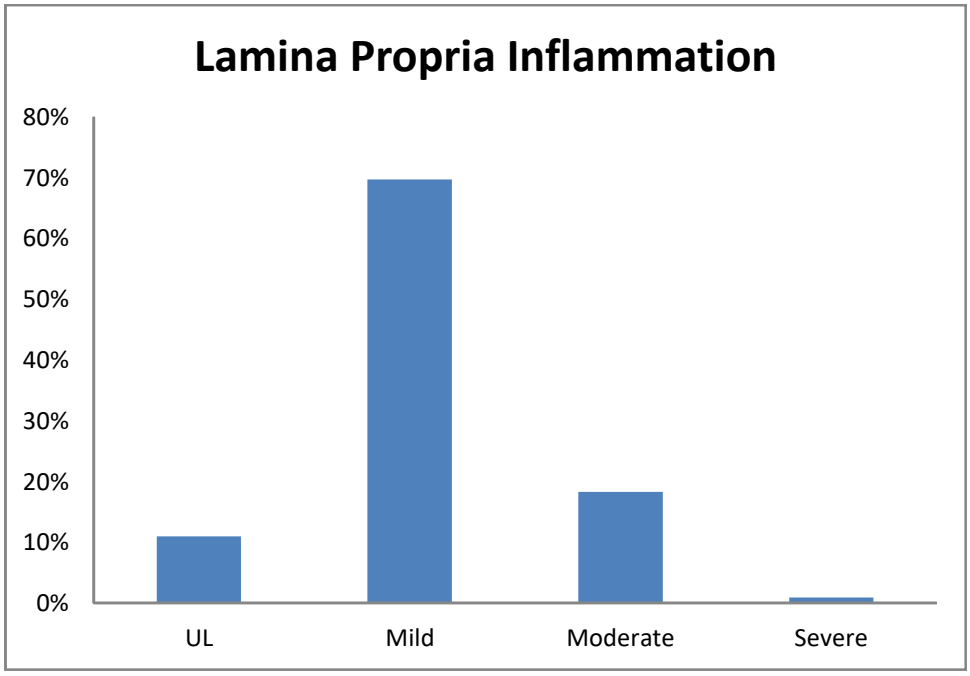
By going through the table in the pittaj grahani the following distribution of Villous Atrophy (pathological changes) where seen.

1. 22.93% showed Partial Villous Atrophy.
2. 2.75% showed Moderate Villous Atrophy.
3. 4.58% showed Focal Shorting and Blunting of Villi.

DISTRIBUTION OF PATHOLOGICAL CHANGES IN GRAHANI (SMALL INTESTINE) OF SELECTED PATIENTS

LAMINA PROPRIA INFLAMMATION WISE DISTRIBUTION

Lamina Propria Inflammation	Percentage	Count
UL	11%	12
Mild	69.2	76
Moderate	18.34%	20
Severe	0.91%	1



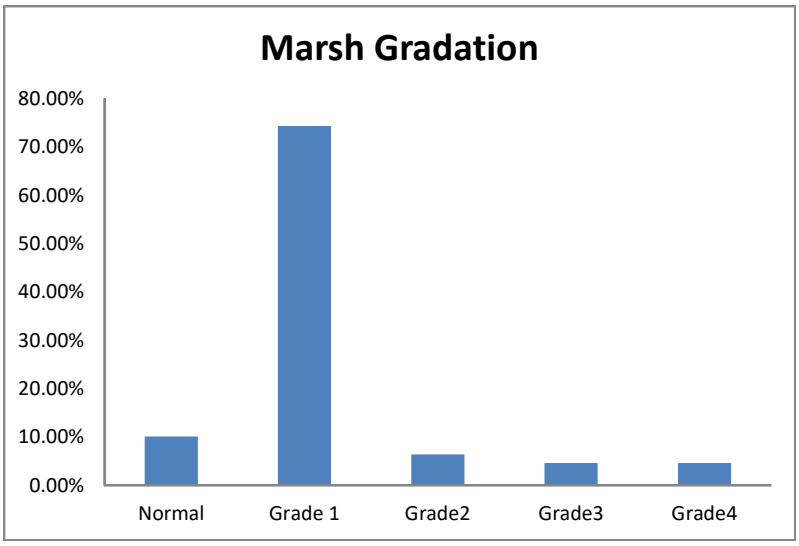
By going through the table in the pittaj grahani the following distribution of Lamina Propria Inflammation (pathological changes) where seen.

1. 69.72% showed mild Lamina Propria Inflammation
2. 18.34% showed moderate Lamina Propria Inflammation
3. 0.91% showed severe Lamina Propria Inflammation
4. 11% showed had normal Lamina Propria.

DISTRIBUTION OF PATHOLOGICAL CHANGES IN GRAHANI (SMALL INTESTINE) OF SELECTED PATIENTS

MARSH GRADATION WISE DISTRIBUTION

Marsh Gradation	Percentage	Count
Normal	10.09%	11
Grade 1	74.31%	81
Grade 2	6.42%	7
Grade 3	4.58%	5
Grade 4	4.58%	5
Grand Total		109



By going through the table in the Pittaj Grahani the following distribution of Marsh Gradation where seen.

1. 74.31% showed grade 1
2. 6.42% showed grade 2
3. 4.58% showed grade 3 and grade 4 each.
4. 10.09% showed normal

DISCUSSION

" तथाविध हि केवल शरीर ज्ञाने शरीराभि निवृत्ती ज्ञाने

प्रकृतिविकारज्ञाने च निःसंशयाभवन्ति ।"

- च. शा. ६/३

In the Ayurvedic text 'sharira' word includes not only rachana sharir but also utpattisharir, prakrutisharir & vikrutisharir so knowledge of sharira is very important to every physician.

व्याध्युपसृष्टानां व्याधिपरिमोक्षः स्वस्थस्य रक्षणं च ।''

"इह खलु आयुर्वेद प्रयोजनं ।।

सु. सू. १ / १३

To fulfill the above moto of ayurveda and for treating any disease or any individual, knowledge of the organ which is to be treated is very essential. In Ayurvedic text Grahani is mostly described as a disease for e.g. grahanigad, grahani rog, grahanidosha, grahaniruk etc. Also it is described as a organ as below :

षष्ठी पित्तधरा नाम या कला परिकीर्तिताः

पक्वामाशय मध्यस्था ग्रहणी सा प्रकीर्तिता ।।

सु. उ. ४० / १६९

Therefore the word grahani described in the ayurvedic text creates a lot of confusion among the scholars & followers of ayurved. Taking into consideration the above facts it is necessary to clear the concept of the term grahani accordingly.

Due to today's fast life & modern lifestyle there is change in food and living habits of the people resulting in to various diseases especially functional digestive problems.

In ayurveda there is a popular quotation

'' रोगाः सर्वेऽपि मन्देग्नौ ''।

(अ.हृ.नि. १२/१)

According to this quotation it is clear that the root cause of every disease is 'Agnimandya' and grahani being the adhisthan of agni it is necessary to study the grahani organ in detail.

There are different views about grahani in modern science, some expert believe duodenum and few say mucosal membrane of small intestine as grahani . So to fix the demarcating line of this organ, a comparative study of ayurvedic and modern text is needed. Therefore grahani should be studied according to the references in the ayurvedic text, and with respect to its anatomy, physiology, pathology & with the help of cadaveric dissection.

Vagbhatta has given a list of some obstinate (Mahagada) diseases and grahani roga has been included in that list. This shows the seriousness of the disease and its prognostic aspect is also not favourable. Such diseases which are obstinate, difficult to treat and are associated with numerous complications require a careful attention of the learned physicians. So hereby, an effort is being made to identify and associate the structural changes seen in grahani, i.e. small intestine viz. duodenum, jejunum and ileum in Pittaj Grahani Roga mentioned in Madhav Nidan with the help of modern diagnostic aids, so that it would support the diagnostic methods in ayurvedic text to diagnose Pittaj Grahani Roga early and treat it effectivly.

GRANATHOKTA RACHNATMAK EXPLANATION

In charaksamhita chikitsasthan, Charaka has described the position of grahani above the umbilicus (nabhiupari). Shushruta in uttartantra has mentioned its sthan (Site) in

between pakwashaya and amashaya. So we can considered the sthan of grahani in between amashaya and pakwashaya. If we considered amashaya as stomach and pakwashaya as large intestine, the structure in between is nothing but shudrantra i.e. small intestine wich is divided in to three parts viz. duodenum, jejunum & ileum.

Asthangsangrahakara has described the pittadharakala as grahani in view of it's function of withholding the food. The inner linning of small intestine situated between stomach and large intestine can be considered as grahani because kala is the inner linning membrane inside the container of the dhatu and ashaya. Here dhatu means the wall of small intestine and ashaya means the lumen of small intestine, therefore the inner linning of small intestine can be considered as grahani.

Ashtanghrudayakar has described its site at pakwashayadvar, which is suggestive of the opening of ileum to the caecum. As far as location is concerned charaka in siddhisthan has described its site in the left of the abdomen and lateral to rectum, which can be taken ileum.

In the nutshell from all the references mentioned above shudrantra can be considered as the sthan of grahani extending from duodenum to ileum.

As per Granthokta Sharir Kriyatmak Explanation :-

In the process of digestion the role of agni, pranvayu, samanvayu & apan vayu is important. These components are controlling the digestion and absorption of the digested food material, assimilation of the same and excretion of undigested waste products (mala) in the form of stool and urine. As per modern physiological concept the intestinal movements are of two types 1) mixing contractions and 2) propulsive contractions. The vagus nerve controls the Gastrointestinal tract especially its various secretions and peristaltic movments.

The controlling neuro transmiter is acetyl - choline. This acetyl - choline binds with cholinergic receptors and show its effects. All this mechanism is tuned very finely. For proper breakdown and mixing of food material with the juice (pachakpitta) it is necessary to hold the food (Grunhati) upto appropriate time in the small intestine (grahani). As per ayurvedic principles this mechanism of controling the digestion and absorption can be co-related with the function of grahani i.e. grunhati, pachati which are controlled by agni samanvayu and pranvayu. Majority of the food is digested in small intestine with the help of intestinal juice and after digestion most of the absorption of the digested food material occurs in small intestine. After this absorption the other important function is excretion which include excretion of waste product (mala) and urine (mutra) which is controlled by autonomus nervous system. Ayurveda has explained this excretion as the function of apanvayu. This process is called Digestion (Pachan). As per ayurveda these are four important functions of grahani which are performed in and by grahani i.e. small intestine.

Any stuctural changes in small intestine in it's various layers like mucosa, submucosa, muscularis mucosa and serosa along with defect in mesentric or myentric plexus (Enteric nervous system) may produce abonormality and eventually get resulted in to pathology like altered stool habits and its consistency (muhurbadham muhurdravam). This also includes formation of ulcerations, flattening of mucosal folds with congestion in it which leads to udardaha, Chhardi, Avipak etc.

As per Granthokta Vikruti Vidynanatmak Explanation :-

The faulty food habits and tensions causes agnimandya in the patients. As the disease progresses involvement of various agni takes place. At the begininig symptoms of jatharagni vitiations can be observed in the form of anorexia and loss of appetite. In the

second phase when the disease becomes chronic, the symptoms of dhatwagni vitiations can be seen in the form of structural changes and finally finest level of agni is also affected in which pathogenesis of the digestive functions on various level are disturbed. These patients show overall symptoms of malnourishment and malabsorption. Due to malnourishment patients also show psychological symptoms like anxiety and irritability. These patients shows the altered pattern of stool habits and changed consistency of stool which is the cardinal symptom of the Grahani i.e. (Muhurbhadham muhurdravam).

DISCUSSION OF CLINICAL STUDY

In the present study, 109 patients of Pittaj Grahani were selected as per samanya lakshanas in madhav Nidan and were selected as per the vishesh lakshanas of Pittaj Grahani in Madhav Nidan. They were subjected to modern diagnostic investigations like OGD scopy colonoscopy to see the structural changes in Pittaj Grahani Roga and four biopsy samples from second part of duodenum(D2) and one from the duodenal bulb from all the patients for histopathological study. Histological grading of D2 biopsies were given according to modified Marsh criteria.

In the study it was observed that 54.13% males and 45.87% females were having Pittaj Grahani. More male patients were found suffering from Pittaj Grahani Roga. With this study any firm conclusion about the prevalence of disease gender wise cannot be concluded. (M:F , 1.2 : 1).

While following the dietary habits, 70% of patients were found having faulty food habits, therefore in the long term they were prone to have Pittaj Grahani Roga. Almost 52.30% were from the age group 15-35 years belonging to young and middle age because persons from this age group normally ignore the ideal dietary habits.

While considering the occupation, house wives 28.44%, students 17.43%, clerk 10.09% , officer 9.17% were more prone to disease who were irregular about their food timings due to their busy schedule and faulty food habits.

While considering the chronicity of Pittaj Grahni Roga it is observed that 80% patients were suffering from Grahani Roga from 3 to 7 months and remaining 20% were having symptoms for less than 3 months. Median duration of symptoms was 4 months.

During the study it is observed that the symptom pain in abdomen, was dominantly present in 66% patient, pain in umbilical region was seen in 83.33% patients and pain in left hypochondric region was found in 10% patients and 6.67% patients had pain in left lumbar region. This shows that PittajgrahaniRoga patients usually have pain around umbilical region.

Analysis of agni showed that 63.30% of patients of Pittaj Grahani were having Mandagni followed by Vishamagni in 31.19% patients and 5.50% patients having Tikshnagni. Agni has important role in Grahani. In mandagni, patients cannot digest food properly. Vishamagni has unpredictable behavior, sometimes it can digest food and sometimes cannot digest food properly. In the study it was seen that when patients suffered from indigestion they had increased lakshanas like pooti Amlodgar 81.65%,trushna 65.84%, mukhpaka (stomatitis)62.39%, Antrakujan (borborygmi) 84.40% which are the striking feature of Pittaj Grahani Roga. A part from this, symptoms like hrullas (nausea), chardi (vomiting) 17.43%, Adhman (bloating) 5.50% which were less dominant are striking feature of vattaj and kaphaj grahani respectively. The importance of state of agni is clearly reflected in the study along with its significance with respect to Pittaj Grahani Roga.

On the objective analysis of jaranshakti on the above mentioned jirna ahar lakshana and fact mentioned in Ayurveda that it takes one yam that is 3 to 3 ½ hrs for complete digestion of food. It was observed that patients57.80% had Heen Jaranshakti followed by33.94% Madhyan Jaranshakti and 8.26% Uttam Jaranshakti.

''Muhurbaddham Muhurdravam'' being the cardinal symptom of Grahani Roga detail mala parikshan as per ayurvedic test and modern laboratory stool examination was done. In bhautik parikshan when all the stool sample was put in distill water, all submerged in it which is a sign of ''sama mala''. Also the stool was found to be foul smelling in 74.31% of patients

which is also a sign of ''sama mala''. This coincides with the quote "sa dushto bahusho bhuktam amameva vimunchati."

In the objective study of frequency of stool in Pittaj Grahani patients it was observed that the average stool frequency in day times was 6 (ranging from 5-8) and night time was 1 (1-8). It was observed that no patients were having frequency of stools between 1-3 and 55.03% were having frequency between 4-6 while 7 and above stool frequency were recorded in 44.97% of patients. Thus the patients of Pittaj Grahani Roga practically shows increased frequency averaging 6 showing the tendency of the disease. Also it shows increased frequency of diarrhea in day time rather than nocturnal showing that it is organic disease not a functional disorder.

On the microscopic examination of stool. It was observed that 25% patients of Pittaj Grahani had helminthes like threadworm, 2% had ascaris, 1% had tapeworm, And 1% had protozoas like E. histolytica and ova of giardia in their stool each. This shows that helminthe threadworm is one of the cause of pittaj grahani.

The stool sample of all the patients was physically examined for its consistency and was grouped under 2category semisolid and liquidy.The observations showed that 81.65% of the patients had liquidy and 18.35% had semisolid consistency of stool, which can be co related with cardinal symptoms of GrahaniRog i.e. ''Muhurbaddham Muhurdravam".

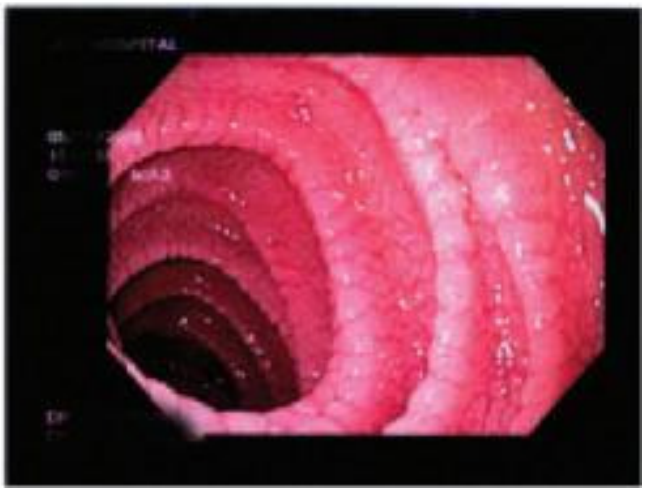
During the study it as observed that all the patients had large voluminous stool (>1 litre/ day) showing the tendency of disease.

On microscopic examination of stool 4 specific observation were noted i.e. bacteria, bacteria and undigested food particle/vegetable fibre, bacteria and undigested fats/oil, bacteria and undigested starch. About 25.69% had undigested food particle and bacteria suggestive of

increased GI motility which can happen in any case of diarrhea. The observation also showed that 32.11% had undigested fat and bacteria suggestive of malabsorption leading to weight loss. Signs like knuckle hyperpigmentation was seen in 77.60% patients and stomatitis in 62.39% of patients suggestive of nutritional deficiencies. Microscopically structural changes were observed in particular parts of small intestine where these nutrients are particularly absorbed, because of weak digestive power (Heen Jaranshakti). The observation also showed that 34.87% patients had undigested starch in stool suggestive of malabsorption of starch resulting in gases formation viz CO_2, H_2 leading to symptoms like bloating 5.50%, distention and flatulence

Mean Hb percentage 8.83 $\pm$1.07 and platelet count 1.54 (1.37-1.80) lakh/mm3 were on lower side while mean corpuscular volume (MCV) found to be high 96.65$\pm$7.44 which indicates B12 deficiency, weak digestive power (mandagni) and malabsorption.

Explanation of the structural changes seen in grahaniroga.



1. Scalloping of duodenal folds- scallops are large shellfish with two flat fan shaped shells. Scallops are a series of small curves or v shaped neckline that form a ornamental border or edging on a particular thing

2. Flattening of the mucosal surface with reduction in the number of duodenal folds- are highly suggestive of villous atrophy.

3. Multiple fissures with mosaic like appearance where the fissure circumscribe areas of mucosal nodularity is a manner similar to the grouting around mosaic tile.

4. Ulcer :- They are usually round sharply punched out defects in the mucosa that penetrate at least in to the submucosa, usually into the muscularis & sometimes more deeply.

5. Punched out ulcer :- The middle portion of bowel seen here has a thickened wall and mucosa has lost the regular folds & contain deep fissure. The serosal surface demonstrates reddish indurated adipose tissue that creeps over the surface. The areas of inflammations tend to be discontinuous through the bowel " skip lesion".

Generally the condition of the mucosa of the small intestine is observed by the endoscopist to find out the abnormality in the small intestine with the help of endoscope. In all type of diseases related to small intestine, the mucosa is affected so the mucosa of the small intestine can be co related with the concept of Kala sharir (Pitta Dhara Kala) mentioned in ayurvedic classical books which is also named as Grahani in Ayurvedic text.

Ayurvedic concept of sharir has its broad view which includes Rachana Sharir (Anatomical Structure) and Kriya Sharir (Physiological Process). However the disturbances in anatomical structures and physiological process eventually leads to pathological changes

In the Ayurvedic aspect, the Grahani is a disease in which the capacity to hold the food (grahan), digestion of food (pachan) proper assimilation and absorption of digested food (Vivechan) and excretion (Munchan) of the waste product (mala) is effected due to the disturbance in agni and samanvayu. This disturbance can be assessed with the help of clinical and investigational methods.

Pitta (agni) is a biochemical entity which is responsible for the digestion of food. In pittaj grahani roga symptoms like Trushna (Thirst), pootiamlodgar (putrification) are mentioned predominantly. In present study, in the patients of Pittaj Grahani scalloping, reduced number of folds, duodenitis with duodenal ulcer and multiple fissure with mucosal nodularity in small intestine were observed in 39.45%, 7.34%, 0.92% and 0.92% patients respectively which shows there are significant structural changes in the mucosal lining of the duodenum hence ayurvedic pathological concept of symptomatology of Pittaj Grahani was confirmed.

The above investigational finding obtain from the observation in the present study shows that the organs affected in grahani roga are duodenum and ileum which are parts of shudrantra i.e. the small intestine.

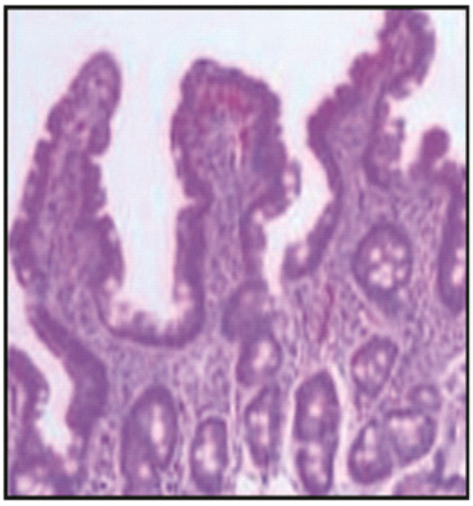
Explanation of the histopathological changes seen in pittaj grahani roga

1. Intraepithelial lymphocytosis -A certain number of CD3+ intraepithelial T lymphocytes (up to 30 per 100 epithelial cells) are normally present in the villi. Immuno histochemical analysis helps to provide a more accurate count. Indeed, when immuno histochemistry is used for CD3, the upper limit is 25 IELs per 100 epithelial cells and the 95% confidence limit is 29 CD3+ IELs per 100 epithelialcells.

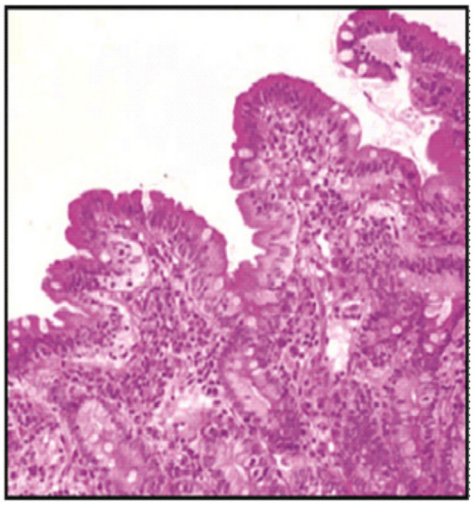
Consequently, it has been suggested that values between 26 and 29 CD3+ IELs should be reported as borderline intraepithelial lymphocytosis and that >30 CD3+IELs should be diagnosed as intraepithelial lymphocytosis.

2. Laminapropria Inflammation-.

The lamina propria is a layer of reticular connective tissue that provides the structural support for the mucosa, but it also contains many cellular elements important for absorption and immunity. The lamina propria is rich in arterioles, venules, lacteals, nerve fibrils, fibroblasts, lymphocytes, macrophages, neutrophils, eosinophils and mast cells. Inflammation marked by capillary dilatation, leukocytic infiltration often loss of function is called as laminapropria inflammation.



Marsh Grade I villous atrophy.



Partial villous atrophy.

3. Villous atrophy - The mean height of a duodenal villi is 700um. Shortening of this length is called as villous atrophy. Normal Villous : Crypt ratio ranges roughly from 3:1 to 5:1 Villous atrophy is defined as a flattening of surface secondary to the shortening and blunting of the intestinal villi. The degree of shortening can be variable.

Total villous atrophy occurs when the V:C ratio varies from 0:1 to 1:1 and partial villous atrophy when it varies from 1:1 to 4:1. The normal villous to crypt ratio in jejunal mucosa is 4:1 or 5:1. Villous atrophy is a non-specific reaction of the intestinal mucosa to a variety of injuries. The pathogenesis is either associated with a hyper-regenerative increase in crypt cell mitosis leading to crypt elongation (eg, in patients with Gluten Sensitive Enteropathy or it results from hyporegeneration—that is, reduced mitotic rate with shortened crypts (as seen in starvation or total parenteral nutrition). In tropical sprue, an as-yet unidentified infective or toxic agent leads to damage to intestinal crypt stem cell thereby causing reduced absorptive cell lineage and resultant villous atrophy. Also contributory to the pathogenesis is the ileal brake mechanism resulting in SIBO.

Histologically the surface epithelial cells are quite atypical cuboidal or even squamoid with loss of microvilli often vacoulated and sometimes infiltrated by inflammatory changes. There is loss of crypts, loss of brush border and increased mitotic activity. The loss of villi is striking. Pittajgrahani patients showed varying degrees of villous shortening and crypt elongation. In addition to blunting, the villi sometimes showed fusion. In the partial villous atrophy seen in 22.93% patient of pittaj grahani this villous : crypt ratio is reduced to 2.5:1 or 2:1 and 4.58% patients of pittaj grhani showed focal shortening and blunting of villi. Also only 2.75% patients of pittaj grahani showed moderate villous atrophy. Complete villous atrophy was not observed even in single pittaj grahani patient !

Intraepithelial lymphocytosis was seen in 58.72% of pittaj grahani patients. In which 55.96% had borderline IEL while only 4.55% showed significant IEL. It can be concluded that IEL is moderately increased (borderline).

Almost 88.99% pittaj grahani patients showed lamina propria inflammation. Out of majority i.e. 69.72% patients showed mild lamina propria inflammation and 18.34% showed moderate while only 0.91% showed severe lamina propria inflammation. It means that the structural change (inflammation of lamina prorpia) is mild in majority patients of pittaj grahani.

According to Marsh gradation of small intestine it was observed that 74.31% of pittajgrahani patients showed grade I while 6.42% showed grade II and 4.58% each showed grade III & IV. It means that in majority patients of pittaj grahani increase in Intraepithelial lymphocytosis was observed.

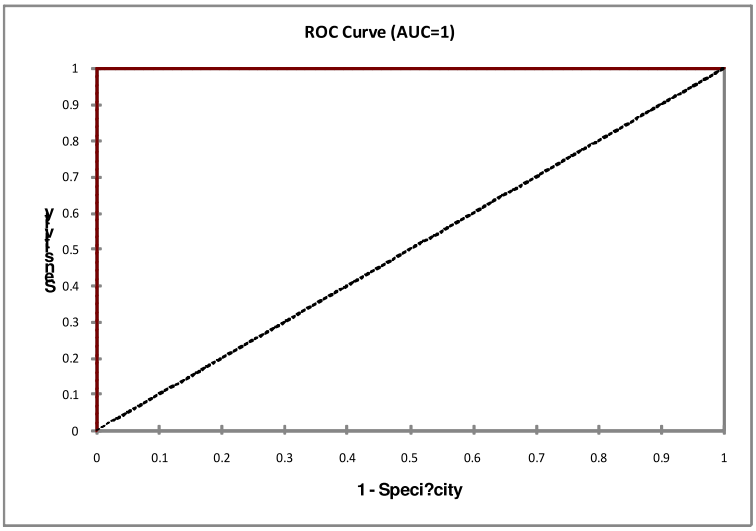
10.09% patients had normal histopathological findings.

After the analysis of the statistical data, The co-relation between various structural changes and various symptoms of pittaj grahani and pathological findings was determined using Logistics Regression.

The ROC 1 is a good indicator of models goodness of fit. The variable on demographic like age etc. and variables on pittaj grahani symptoms seems to be statistically significant to determine the impact on scalloping . The conclusion is that persons with symptoms of pittaj grahani seems to have scalloping of duodenum.

Classification table for the training sample (Variable scalloping):				
from\to	0	1	Total	% correct
0	66	0	66	10.00%
1	1	42	43	97.67%
Total	**67**	**42**	**109**	**99.08%**

ROC Curve (Variable scalloping):

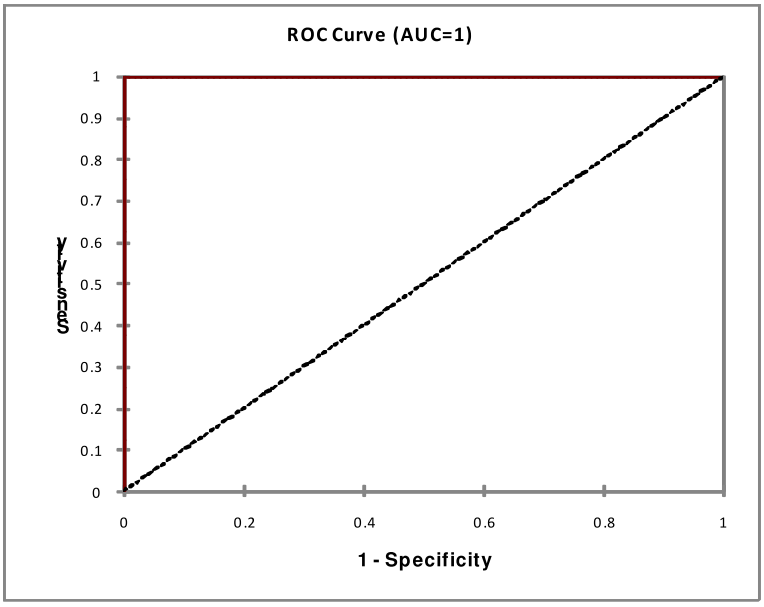


Similarly the ROC 1 is a good indicator of models goodness of fit. The variable on demographic like age etc. and variables on pittaj grahani symptoms on seems to be statistically significant to determine the impact on "reduced number of folds". The conclusion is that persons with symptoms of pittaj grahani seems to have "reduced number of folds" of duodenum.

Classification table for the training sample (Variable reduced no of folds):

from\to	0	1	Total	% correct
0	100	0	100	100.00%
1	0	8	8	100.00%
Total	**100**	**8**	**108**	**100.00%**

ROC Curve (Variable reduced no of folds):



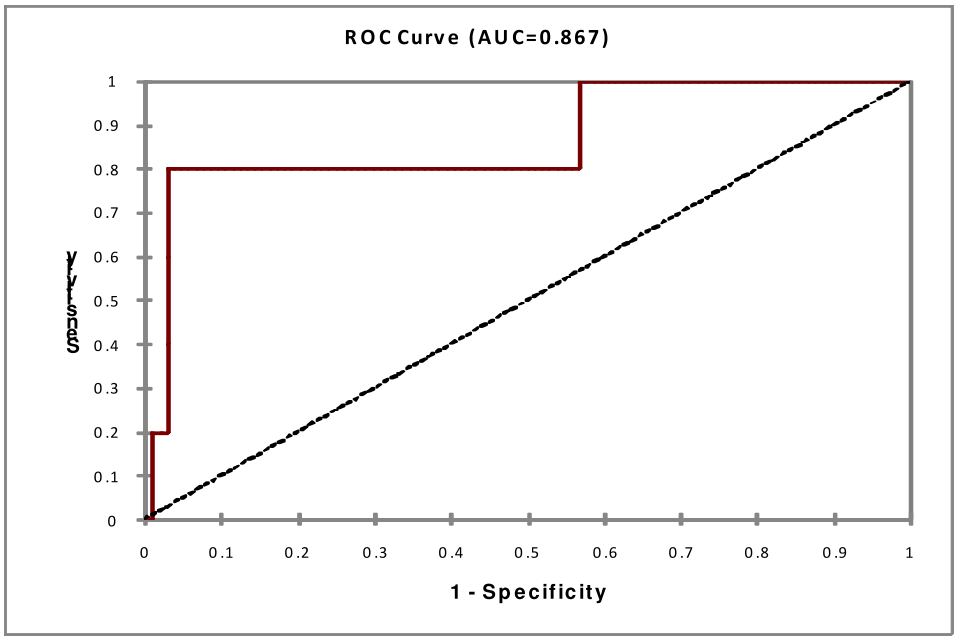
Area under the curve :1

The relationship between histopathological variables and variables related to symptoms of pittaj grahani and pathological finding (stool examination including blood examination) was determined using Logistics Regression. The ROC of 0.867 is a good indicator of models goodness of fit. The variables on demographics like age etc, variables on pittaj grahani symptoms, seems to be statistically significant to determine the impact on IEL. The conclusion is that persons with symptoms of pittaj grahani and seems to have increased IEL.

Classification table for the training sample
(Variable IEL_derived):

from\to	0	1	Total	% correct
0	101	3	104	97.12%
1	4	1	5	20.00%
Total	**105**	**4**	**109**	**93.58%**

ROC Curve (Variable IEL_derived):



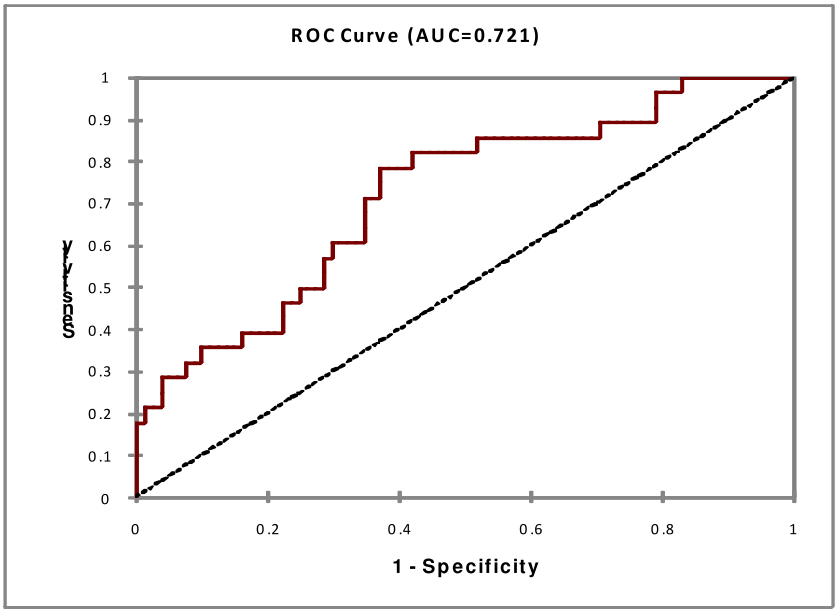
Area under the curve : 0.867

The ROC of 0.721 is a good indicator of models goodness of fit. The variables on demographics like age etc and variables on pittaj grahani symptoms, seems to be statistically significant to determine the impact on Villous atrophy. The conclusion is that persons with symptons of pittaj grahani seem to have Villous atrophy.

Classification table for the training sample (Variable VILLOUS ATROPHY):

from\to	0	1	Total	% correct
0	81	0	81	100.00%
1	26	2	28	7.14%
Total	**107**	**2**	**109**	**76.15%**

ROC Curve (Variable VILLOUS ATROPHY):



Area under the curve: 0.721

Based on the above observation it can be conclude that Symptoms of pittaj grahani and pathological findings of the patients have direct co relation with various histopathological and stuctural changes in grahani.

SUMMARY

Todays modern life-style and strenuous quick life is leading to faulty dietary and living habits of people which has made them prone to gastrointestinal disturbances like grahani roga, so it is the need of time to upgrade the knowledge about grahani organ and disease. Also for exact diagnosis and effective treatment of different type of Grahani Roga it is necessary to study the structural changes in grahani avayav in Grahani Roga. So the study entitled " Endoscopic Assessment of GI Tract W.R.T. Grahani Sharir in Clinically diagnosed patients of Pittaj Grahani Roga" has been undertaken.

Ayurvedic Literature reveals that the word grahani is used in both reference as an organ and mostly as a disease.

Grahani is the location (Sthan) of agni which is also called by various name such as Kayagni, Pachakpitta etc.

Grahani performs various functions like grunhati, pachan (digestion), vivechan (Assimilation) and absorption of food while travelling from stomach to ileum that is Laghuantra which is named as Small Instestine in modern text. So anatomically site (Sthan) of grahani described in ayurvedic text can be co related with the four mucosal layers of small intestine.

The structural changes seen in pittaj grahani roga are similar to the changes seen in different diseases of small intestine mentioned in modern text.

The structural changes found in pittaj grahani roga through endoscopy are as follows -

1. Duodenal Scalloping
2. Flattening of mucosal folds
3. Duodenitis with duodenal ulcer
4. Ulcer at ileo-caecal junction
5. Multipale fissure with mosaic like appearance with Mucosal nodularity.

The Histopathological changes found in pittaj grahani roga are as follows -

1. Varying degrees of villous shortening, blunting and atrophy
2. IEL moderately increased (Borderline)
3. Significant lamina propria inflammation

Pittaj Grahani patients showed grade I mucosal changes as per Marsh Gradation significantly. Co-relation of the degree of the variation of Villous Atrophy, IELand Lamina propria inflammation with the symptoms of Pittaj Grahani Roga in Pittaj Grahani Roga patients can be obtained by taking a bigger sample size for study, which is a matter of further research.

CONCLUSION

After completion of this present project it can be concluded that as far as grahani sharir is concerned mostly duodenum can be considered as the site of grahani.

To be very precise, with the help of endoscopic assessment the following structure (microscopic) of small intestine (grahani) i.e. mucosal and sub mucosal layers of small intestine can be considered as the site (sthan) of grahani.

In most of the Pittaj Grahani patients (75percent) Grade I mucosal changes as per Marsh Gradation was observed.

All these above findings and statistical test applied confirms that structural involvement and changes in Grahani avayav are directly related with the symptoms of Pittaj Grahani vyadhi.

BIBLIOGRAPHY

AYURVEDIC LITERATURE

1. Rigveda
Edited by Shri Manmokshamul Mularbhatt. Published by Krishnadas Academy, Varanasi.

2. Atharva Veda
Edited by Shri Shankar Pandurang Pandit. Published by Shri Krishnadas Academy, Varanasi..

3. Sushruta Samhita
By Kaviraja Ambikadatta Shastri (Edition 2002)
Published by Chaukhambha Samskrita Samsthana

4. The Sushrut Samhita :
By Kaviraj Kunjalal Bhishagratna, Published by Choukhamba Sanskrit series office, Varanasi-1(3RD Edition 1981)

5. Sushrut Samhita :
By Prof K.R. Shrikantha Murthy, Published by Choukhamba orientalia(Vol.1st, Edition 2nd 2004)

6. Charaka Samhita :
Edited with Charaka Chandrika by Dr. Bramhananda Tripathi,
Published by Chaukhamba Surabharati Prakashan, Varanasi (Edition 2005)

7. Charak Samhita Vol.1st & 2nd :
By Prof. Priyavat Sharma, Published by Choukhamba orientalia(Vol.1-1st Edition 1981), (Vol.2-1st edition 1983)

8. Ashtanga Hrudaya:
By Bramhanand – Tripathi (Edition – 2000)
Published by Chaukhambha Samskrita Samsthan

9. Ashtanga Hrudaya:
Commentary by Arundatta (Sarvangsundari) & Hemadri (Ayurveda Rasayana), Published by Choukhamba Orientalia, Varanasi, (Edition1882)

10. Ashtanga Sangraha :
By Vd. Anant Damodar Athavale (Edition 1st Sept. 1980)
Published by Godavari Publishers and Book Promoters, Nagpur

11. Bhavprakash Samhita :
Acharya Ramanath Dwivedi, Choukhamba Publication, (1st Edition)

12. Sharangdhara Samhita :
By Acharya Radhakrishna Parasara (Edition 1984)

13. Ayurvediya Shabdakosha :
By Vaidya Lakshman Shastri Joshi,
Vaidya Venimadhav Shastri Joshi, Vaidya Narayan Hari Joshi, Maharashtra Rajya Sahitya & Sanskruti Mandal, Mumbai.

14. Shabdakalpadrum :
By Raja Radhakant Dev.,Published by Naga Prakashana, Delhi

15. Parishadya Shabdarth Sharir :
By Ayurvedacharya P. Damodar Sharma Gaud.

16. A Sanskrit English Dictionary :
By Sir Monier Monier Williams, Published by Motilal Banarasidass publisher pvt. Ltd.(1st Edition 1990)

17. Vachaspatyam part-3 :
By Shri Taranath Bhattacharya

18. Sushrut Sharirsthana:
Commented by Dr. B.G.Ghanekar,Published by Meherchand Lacchmandas Publication (Reprint 1992)

19. Madhava Nidan
Madhukosha with Vidyotini, Hindi commentary by Sudarshana Shastri

20. Raja Nighantu
Commentary by Indradev Tripathi
Chaukhamba Krushnadas Academy, Varanasi.

21. Yogaratnakar
Commentary by Vidyotini
Laxmipati shastri edition 1961

22. Harit Samhita
By Pandit Ramalamba Shastri edition 1985

23. Bhel Samhita
Vd. V. S. Venkatsubramanyam Shastri
Vd. C. Raj Rajeshwar Sharma

24. Vangasen Samhita
By Dr. Rajiv Kumar Roy

25. Amarkosha
By Shree Pandit Hargovinda Shastri

26. Pratyaksha Shariram
By Kaviraj Shree Gananath Sen

27. Doshadhatumala Vidnyan
By Vaidyaratna Phadke

MODERN LITERATURE

1. Gray's Anatomy
By Henry Gray edited by Peter Williams & others, Churchil Livingston Publications (38th Edition, 1995)

2. Gray's Anatomy For Students
(1st Edition, 2005), Elsivier Churchil Livingston Publications.

3. Principal of Anatomy and Physiology, Tortora & Derrickson
(11th Edition, 2006), John Wiley & Sons, Inc. U.S.A.

4. Textbook of Pathology : By Harsh Mohan, Published by Jaypee brothers medical publishers pvt ltd(4th Edition 2000)

5. Textbook of Medical Physiology : By Guyton

6. Sleisenger and Fordtran's,Mark Feldman, Lawrence S. Friedman,
Lawrence J. Brandt. Gastrointestinal and Liver Disease 10th Edition Volume 1, © 2016 by Saunders imprint by Elsevier

7. Human Embryology : By Indebir Singh & J. P. Pala Eighth Edition Reprint 2007

8. Principles of Internal Medicine - Harrisons.

9. Textbook of Pathology - Robbins

10. Textbook of Histology - Krishna Garg.

11. Dr. Baghel's Text Book
(For selecting topic and for review of previous work done)

Patrikas & Journals

1 **Ayurvidya - 5**
Grahani Roga Parisawhad
Published by Rastriya Shikshan Mandal Pune.

2 **Madhu Jivan**
Grahani Roga Visheshank
Part 3 August 1985
Published by Madhavi Prakashan Mahim Mumbai - 16.

3 Marsh MN, Crowe PT. Morphology of the mucosal lesion in gluten sensitivity. Baillieres Clin Gastroenterol 1995;9;273-293

4. S. Sera, P.A. Jani an approach to dueodenal biopsies; 2006;59, 1133 -1150

5 Mathan M. Mathan VI, Baker SJ. An electron-microsopic study of jejunal muscosal morphology in control subjects and in patients with tropical sprue in southern India. Gastroenterology 1975;68;17-32.

6 Ghoshal UC, Kumar S. Misra A. Choudhuri G. Pathogenesis of tropical sprue: a pilot study of antroduodenal manometry, duodenocaecal transit time & fat-induced ileal brake, Indian J. Med Res 2013;137;63-72

7. Bhagwan Thakur, Peeyush Mishra, Nutan Desai, Sanjeev Thakur, Jacob Alexander, Prabha Sawant : Profile of Chronic Small bowel diarrhea in adults in western india : A hospital based study. Tropical Gastroenterology 2006; 27

8. Uday C. Ghoshal, Mansi Mehrotra, Sunil Kumar, Ujjala Ghoshal, Narendra Krishnani, Asha Misra, Rakesh Aggarwal & Gourdas Choudhuri: Spectrum of malabsorption syndrome among adults & factors differentiating celiac disease & tropical malabsorption .Indian J Med Res 136, September 2012,

9. Yadav P, Das P, Mirdha BR, Gupta SD, Bhatnagar S, Pandey RM, Makharia GK. Current spectrum of malabsorption syndrome in adults in India. Indian J Gastroenterol. 2011 Feb;30(1)

10. Dutta AK, Balekuduru A, Chacko A. Spectrum of malabsorption in India—tropical sprue is still the leader. J Assoc Physicians India. 2011 Jul;59

11. Murray JA, Rubio-Tapia A. Diarrhoea due to small bowel diseases. Best Pract Res Clin Gastroenterol. 2012 Oct;26(5)

12. P D Thomas, A Forbes, J Green, P Howdle, R Long, R Playford, M Sheridan, R Stevens, R Valori, J Walters, G M Addison, P Hill, G Brydon : Guidelines for the investigation of chronic diarrhoea, 2nd edition *Gut* 2003;52

13. Margaret-Mary G Wilson, David R Thomas, Laurence Z Rubenstein, John T Chibnall, Stephanie Anderson, AmyBaxi, Marilyn R Diebold and John E Morley; Appetiteassessment : simple appetite questionnaire predicts weight loss in community - dwelling adults and nursing home residents. Am J Clin Nutr 2005;82:1074 to 1081.

14. Nicholas J. Talley, M.B. PhD., Sidney F. Phillips, MD, Christine M. Wiltgen, B.S., Alan R. Zinsmeister, Ph.D., L. Joseph Melton III, M.D. ; Assessment of Functional Gastrointestinal Disease: The Bowel Disease Questionnaire ; Mayo Clin Proc, November 1990, Vol 65 : 1456 to 1479

Web Sites:

· www.google.com
· ww.bmj.com
· www.wikipedia.org

QUESTIONNAIRE

1. How is your appetite ?

2. Do you have nausea ? If yes, Is it often or rarely ? When, Empty stomach / after food / only after spicy food / after over eating or any other ?

3. Do you have vomiting? If yes, Is it often or rarely ? When, Empty stomach / after food / only after spicy food / after over eating or any other ?

4. Do you have any abdominal discomfort –like excessive gas especially with frequent belching, abdominal bloating or distention or flatus ?

5. Do you have any abdominal pain ? Where does the pain start ? Does it radiate or travel anywhere ? What is the pain like ? Is it aching, burning ,gnawing or what?

6.How severe is the pain ? Is it bearable ? Does it interferes with your usual activities ? Does it make you lie-down ?

7.How are your bowel movements ? Have you noticed any change in your bowel habits ? Do you have any difficulties ? What is the frequency of your bowel movements ?

8. What is the color & bulk (quantity) of your stools ? Are the stools greasy or oily ? Frothy ?foul smelling? Accompanied by mucs, pus or blood ? Since how long you are suffering from this stool habits ?

9. Does the pain start suddenly or gradually ? When it begins ? How long it last ? What is the pattern over a 24 hr period ? Are there any symptoms associated with pain such as fever or chills ?

10.Do you burp ?If yes, What type of taste does it reveal to your mouth? Sweet,Bitter or any other ?

CASE REPORT FORM (CRF) FOR THE DISSERTATION

Endoscopic Assessment of GI tract w.r.t. Grahani Sharir in Clinically diagnosed patient of pittaj GrahaniRoga.

Ph.D. (Scholar)
Vd. Yogesh Vitthal Gaikar

Guide
Prof. Dr. Chandrashekhar D. Vaikos

GENERAL INFORMATION

IPD. No.	:		OPD No.
Name	:		Age :
Address	:		Religion:
Occupation	:		Education
Desh	:		sex:
	Janma desh	:	Anoop / Jangal
	Vyadhi Utpatti Desh	:	Anoop / Jangal
Kala	:		
	Janma Kala	:	Adan / Visarga
	Vyadhi Utpatti Kala	:	
	Rutu	:	

VEDANA VISHESH

VARTAMAN VYADHI VRUTTA

POORVA VYADHI VRUTTA

TRIVIDH PARIKSHA

PERSONAL HISTORY

1.	Ahar	:	Vegetarian / Non- Vegetarian / Mix
2.	Vihar	:	
3.	Vyasana	:	Tea / Coffee / Alcohol / Tobacco / Mishri / Smoking
4.	Nidra	:	Anidra / Atinidra / Alpanidra / Samyaknidra / Divaswap
5.	Agni	:	Visham / Tiksan /Manda / Sama
6.	Koshth	:	Mrudu / Madhyama / Krura
7.	Weight	:	Kg.
8.	Temperature	:	F
9.	Pulse	:	Minute
10.	Blood Pressure	:	mm of Hg
11.	Abhyavaran Shakti	:	
12.	Shudha	:	
13.	Jaran Shakti	:	

ASHTA VIDHA PARIKSHANA

1.	Nadi	:
2.	Mutra	:
3.	Mala	:
4.	Jivha	:
5.	Shabda	:
6.	Sparsha	:
7.	Druk	:
8.	Aakruti	:

SROTAS PARIKSHANA

1. UDAKVAHA :

   Jivha Talu, Kantha, Oshtha, Kloma. Shushkata / Pipasa

   Jivha : Kantha :

   Talu : Kloma :

   Trushna :

2. ANNAVAHA :

   Anannabhilasha / Avipaka / Arochaka / Chhardi / Aadhman

   Oshtha : Vamaparshwa :

   Udar Parikshana :- Darshana :- Aakotana :-

   Sparshana :- Shravan :-

3. RASAVAHA :-

   Ashraddha / Angamarda / Aruchi / Jwara / Aasyavairasya /
   Pandu / Klaibya / Hrullas / Krushata / Gaurav /
   Akalvalaypalita / Tandra

   Hruday :- Nadi :-

4. PURISHAVAHA :-

   Malapravrutti :- Sakashta /Sasneha/ Sashula / Sashabda / Alpa / Bahu /
   Drava / Grathit

   Aantra :- Guda:-

   Malapravrutti :-

   Frequency :-

MALAPARIKSHAN (Bhautik Parikshan)

Jala Parikshan

Bhrus Duragandha Yukta

Sahanan

MALAPARIKSHAN (Microscopic Examination)

Ova

Cyst

Other

MALAPARIKSHAN (Mordern Aspect)

Frequency of Stools	1-3	4-6	7-9
Appearance	Watery	Semisolid	Semi Liquid
Colour	Whitish Yellow	Greyish	Blackish
Reaction	Acidic	Alkaline	Neutral
Odour			
Examination			

VISHESH PARIKSHAN

Sr No	Sign / Symptoms	Vataja	Pittaja	Kaphaja	Sannipataj
1.	Malapravrutti (Stool habits)	Aamyukta , Phenyukta, Dravayukta	Dahayukta , Neelpeeta , Drava	Bhinna Shelshma yukta	Taral Aamyukta Snigdha
2.	Apachana / Chhardi (Indigestion/ Vomiting)	Chhardi	Pooti Amlodgar	Madhura Udgar , Arochaka	Food Intolerance of all taste
3.	Other GIT. Symptoms	Udarvriddhi , Adhamana, Vairasya , Kanthashosha	Kanthashosha Kanthadaha Trushna	Hrullasa	Aantrakoojan
4.	Deha Aakruti (Physical Appearance)	Karshya, Daurbalya		Akrushyasapi Daurbalya	Daurbalya
5.	Sthanik peeda (localised Pain)	Parshva , Uru, Vankshan, Greevaa, Shoola, Parikartika	Guda , Hrudaya Udar, Mastaka Daha	Guda , Hrudaya, Udar, Mastaka , Parsva Gaurav	Katishoola

1. Vataja :-
2. Pittaja :-
3. Kaphaja :-
4. Sannipataj :-

MODERN SCOPY FINDINGS

A) OGDscopy

Histopathology

Patient Information Sheet

Project Title:

Endoscopic assessment of GI tract w.r.t Grahani Sharir in clinically diagnosed patients of Pittaj Grahani Roga

Introduction:

The small intestine is both a secretory and absorptive organ. Disorders of the small intestine can be primarily malabsorptive or secretory in nature but most disorders on the small intestine mucosa result in both excess secretion and failure of absorption.

Diarrhoea of small bowel origin is more often noninflammatory and high output. Improvement of diarrhoea after fasting suggests an osmotic component of the symptoms. Postprandial diarrhoea, bloating, malodorous flatus, and pale stools which leave an oil slick or are difficult to flush suggest malabsorption.

Weight loss despite a normal appetite might suggest malabsorption though loss of appetite due to pain on eating can also be a cause. Pain is a common symptom of small bowel diseases including coeliac disease. The pain may be focal or diffuse; it is often associated with meals, bloating and/or distension. Generalized malaise or fatigue are also common though nonspecific.

Aims and Objectives:

1. To determine and correlate the structural changes in grahani avayav with reference to pittaj grahani vyadhi.

2. To study and asses the subjectwise relevance of grahani sharir Anatomically and to determine anatomical location and extent of grahani avayav.

Assessment method:

Patients either referred or presented by themselves will be included. Patients with diarrhea of more than 1 month which fulfills the features of small bowel type of diarrhea/ Pittaj Grahani Roga. All patients will undergo evaluation in form of Stool routine, micro and special stains. Patients will also undergo OGD scopy with D2 biopsy and ileocolonoscopy with ileal biopsy. USG abdomen and if needed.

Benefits of taking part in the study:

By being a part of the study, the unaware patients can be brought to light about the nature of their diarrhoea. They can be educated about early consultation before symptoms of micro or macronutrient deficiency manifest. Also patient can be educated about other risk factors for chronic diarrhoea such as poor hygiene or contact with a case of tuberculosis. Patients can be educated regarding need to modifiy the diet for control of diarrhea such as in celiac disease or lactose intolerance.

Rights of the patient:

If the patient wishes not to be a part of the study, he may do so. The patient has the right to stop answering, allowing tests as per his or her wish at any moment during the assessment period.

Dr. Chandrashekhar Vaikos : 9422871600
Dr Meghraj Ingle: 9320979659
Dr Yogesh Gaikar: 9987519516

रोगी सूचना पत्रक

परियोजना शीर्षक :-

पित्तज ग्रहणी यह वैद्यकीय निदान किए हुए रुग्णों मे ग्रहणी शारिर के संदर्भ मे पाचन संस्थान का दुर्बीण द्वारा मुल्यांकन.

परिचय :-

छोटी आंत एक स्त्रावी और अवशोषण अंग दोनों है. छोटी आंत के विकार प्रकृति में मुख्य रुप से अवशोषणीय या स्त्रावी हो सकते है, लेकीन अधिक स्त्राव और अवशोषण की असफलता यह दोनों छोटी आंत म्युकोसा के बहुतांश विकारों मे पाया जाता है.

छोटी आंत मूल के दस्त अधिक बार बिन–सुजन-संबंधी और उच्च उत्पादक है. उपवास के बाद दस्त के लक्षणों मे सुधार से एक आसमाटिक घटक पता चलता है. भोजन के बाद दस्त, सूजन, बदबुदार,पीला मल, अधोवायु और एक तेल चालक छोड, या फ्लश करने के लिए मुश्किल होता है. यह अवशोषण को दर्शाता है.

एक सामान्य भूख के बावजूद वजन में कमी, अवशोषण की असलफता दर्शाता है. हालांकी खाने के बाद दर्द होना यह भी एक कारण हो सकता है. दर्द सीलिएक रोग सहित छोटी आंत की बीमारीयों के एक आम लक्षण है. दर्द फोकल या फैला हुआ हो सकता है. यह अक्सर भोजन, सुजन या बढाव के साथ जुडा हुआ होता है. सामान्यकृत अस्वस्थता या थकान भी आम लक्षण है.

लक्ष्य और उद्देश्य :

१. पित्तज ग्रहणी व्याधी मे ग्रहणी अवयव में होने वाले रचनात्मक बदलों का निश्चितीकरण करके उनका संदर्भ स्थापित करना.

२. ग्रहणी शारिर का अध्ययन करके ग्रहणी अवयव का रचनात्मक स्थान और प्रमाण निश्चित करना.

आकलन विधी

१. मरीजों का स्वयं द्वारा भेजा या प्रस्तुत या तो शामिल किया जाएगा. दस्त और किसी भी दो सूक्ष्म या पुष्टिकर कमियों के सबुत की छोटी आंत प्रकार की सुविधाओं को पुरा जो अधिक से अधिक १ महिने के दस्त के साथ मरीजों को शामिल कर रहे हैं. सभी रोगियों को कुछ रक्त जांच सीबीसी और स्टुल दिनचर्या, सुक्ष्म और विशेष दाग के रुप में मुल्यांकन से गुजरना होगा. मरिजो को एन्डोस्कोपी से गुजरना होगा, मरीजों को डी २ बायोप्सी के साथ स्कोपी और इलेअल बायोप्सी के साथ कोलोनोस्कोपी और यदि आवश्यक हो तो पेट की यूएसजी की जाएगी.

अध्ययन में भाग लेने के लाभ :

अध्ययन का एक हिस्सा होने के नाते, अनजान रोगियों को उनके दस्त की प्रकृति के बारे में प्रकाश में लाया जा सकता है. वे सूक्ष्म या पोषक तत्व कमी प्रकट के लक्षणों से पहले प्रारंभिक परामर्श के बारे में शिक्षित किया जा सकता है. इसके अलावा रोगी को स्वच्छता या तपोदिक के एक मामले के साथ संपर्क के रुप मे क्रोनिक दस्त के लिए अन्य जोखिम वाले कारकों के बारे में शिक्षित किया जा सकता है. मरीजों को ऐसी सिलीएक रोग या लैक्टोज असहिष्णुता के रुप में दस्त के नियंत्रण के लिए आहार मे बदलाव करने की जरुरत के बारे में शिक्षित किया जा सकता है.

रोगी का अधिकार :

यदी रोगी को अध्ययन का एक हिस्सा नही बनना चाहता है, तो वह ऐसा कर सकते है. रोगी मूल्यांकन अवधि के दौरान किसी भी क्षण में उसके या उसकी इच्छा के अनुसार परिक्षण, जवाब देने की इच्छा या जवाब देने की अनुमति रोकने को अधिकार है.

डॉ. चंद्रशेखर वायकोस : ९४२२८७१६००
डॉ. मेघराज इंगळे : ९३२०९७९६५९
डॉ. योगेश गायकर : ९९८७५१९५१६

रुग्ण माहिती पत्रक

प्रकल्प शीर्षक :

पित्तज ग्रहणी हे वैद्यकीय निदान झालेल्या रुग्णांमध्ये ग्रहणी शरीराच्या संदर्भात पाचन संस्थेचे दुर्बिणीद्वारे मुल्यांकन.

परिचय

लहान आतडे एक स्त्रावी आणि शोषणक्षम शरीराचा अवयव आहे. लहान आतड्यांच्या संबंधित विकार प्राथमिक कुअवशोषणीय किंवा विमोचन मुळे असु शकतो. जादा विमोचन आणि शोषण या दोन्ही प्रकारांमध्ये लहान आतड्याच्या श्लेष्मल त्वचेवर परिणाम होऊन विकार होतो.

लहान आतड्याच्या मुळातुन होणारा अतिसार अनेकदा दाहक नसलेला आणि उच्च उत्पादन असतो. उपवासा नंतर अतिसाराच्या लक्षणांची सुधारणा होणे, आसरण संबंधी घटक सूचित करते. जेवणानंतरचा अतिसार, गोळा येणे, दुर्गंधी येत असलेला पोटातील वायु आणि एक तेल पूर्णपणे सोडुन किंवा फ्लश करणे कठीण असते. हे लहान आतड्यांची पदार्थ शोषणाची असमर्थता सुचवते.

सामान्य भुक असुनही वजन कमी होणे हे देखील पदार्थ शोषणाची लहान आतड्याची असमर्थता सुचित करते व खाल्यावर वेदना होणे हे देखील भुक न लागण्याचे एक कारण असु शकते. वेदना हे लहान आतड्याच्या रोगाचे एक सामान्य लक्षण आहे. वेदना फोकल किंवा विखुरलेला असू शकते. ती वेदना अनेकदा जेवणाशी / पोटफुगीशी संबंधीत असते. सामान्य धुसफुस किंवा थकवा हे देखील त्याचे सामान्य लक्षण आहे.

धोरण व उद्दिष्टे

१. पित्तज ग्रहणी व्याधी संदर्भात ग्रहणी अवयवातील रचनात्मक बदलांचे निश्चितीकरण करुन संदर्भ स्थापित करणे.

२. ग्रहणी शारीरचा अभ्यास करुन ग्रहणी अवयवांचे रचनात्मक स्थान आणि प्रमाण निश्चित करण्यासाठी.

मुल्यांकन

रुग्णास स्वत: किंवा कोणी सादर केल्यास समाविष्ट केले जाईल. १ महिन्याच्या अतिसार असलेले व पित्तजग्रहणीचे लक्षण असलेल्या रुग्णांना समाविष्ट केले जाईल. सर्व रुग्णांची रक्त तपासणी, सीबीसी आणि स्टूल नियमानुसार, सुक्ष्म व विशेष डाग स्वरुपात मूल्यमापन केले जाईल. तसेच ओजीडी स्कोपी व डी- २ बायोप्सी आणि इलेअल बायोप्सी सह कोलानोस्कोपी केली जाईल. आणि आवश्यक असल्यास पोटाची सोनोग्राफी केली जाईल

अभ्यासात भाग घेण्याचे फायदे :

अभ्यास एक भाग घेतल्याने नकळत रुग्णांना त्यांच्या अतिसाराच्या स्वरुपाबद्दल अधिक माहिती दिली जाऊ शकते. सुक्ष्म किंवा पुष्टीकर घटकांच्या कमतरतेच्या लक्षणांची माहिती देऊन त्यावर योग्य उपायाची माहिती दिले जाऊ शकते. तसेच रुग्णाला अशा स्वच्छता किंवा क्षयरोग संपर्कात होणाऱ्या तीव्र अतिसार च इतर जोखीम घटक याविषयीचे माहिती दिली जाऊ शकते. रुग्णांना सीलिक या उदर रोगाचा किंवा दुग्धशर्करा सहन न होऊन होणाऱ्या अतिसाराच्या नियंत्रणासाठी आहार बदलण्याची गरज असल्यास त्याबद्दलची माहिती दिली जाऊ शकते.

रुग्णाच्या अधिकार :

रुग्णाला अभ्यासात सहभागी होण्याची इच्छा नसेल तो नकार देऊ शकतो. रुग्णाला मूल्याकंन कलावधीमध्ये कोणत्याही क्षणी त्याच्या किंवा तिच्या इच्छेनुसार चाचण्या, उत्तर देणे, थांबविण्याचा अधिकार आहे.

डॉ. चंद्रशेखर वायकोस : ९४२२८७१६००
डॉ. मेघराज इंगळे : ९३२०९७९६५९
डॉ. योगेश गायकर : ९९८७५१९५१६

INFORMED CONSENT FORM

Endoscopic assessment of GI tract w.r.t Grahani Sharir in clinically diagnosed patients of Pittaj Grahani Roga

I have been explained about the study in detail by the doctors. I understand that participation in study is voluntary and I am free to withdraw any point of time without giving any reasons. My refusal for consent to participate in the study will not affect my medical care in any way.

I have read and understood the information provided in the patient information sheet. I have been explained the nature and purpose of study in detail.

I hereby agree to take part in the above mentioned study voluntarily, without force.

If you wish to ask any queries you can contact any of the investigators given below.

Investigator's sign:

Name:

Date:

Patient's sign:

Name:

Date:

Witness sign

Name:

Date:

Contact numbers

Dr. Chandrashekhar Vaikos : 9422871600
Dr Meghraj Ingle: 9320979659
Dr Yogesh Gaikar: 9987519516

माहितीपूर्ण संमती पत्र

पित्तज ग्रहणी हे वैद्यकीय निदान झालेल्या रुग्णांमध्ये ग्रहणी शारीराच्या संदर्भात पाचन संस्थेचे दुर्बिणीद्वारे मुल्यांकन

माझ्या डॉक्टरांनी विस्तृतरीत्या अभ्यासाबद्दल माहिती देऊन स्पष्ट केले आहे. मी अभ्यासात स्व-ईच्छेने सहभागी होत आहे. आणि मी कोणत्याही क्षणी या अभ्यासातुन माघार घेऊ शकतो / शकते, कोणतेही कारण न देता. अभ्यासात संमतीसाठी माझा / माझे नकार देणे कोणत्याही प्रकारे वैद्यकीय सुविधा प्रभावित करणारी नाही.

मी रुग्ण माहिती पत्रक वाचले आहे आणि समजुन घेतले आहे. मला तपशील अभ्यासाचे स्वरुप आणि हेतु स्पष्ट केले आहे व ते मला समजले आहे.

मी येथे कोणत्याही सक्ती शिवाय व स्व-इच्छ वर उल्लेख केलेल्या अभ्यासात भाग घेत आहे.

आपण कोणतेही शंका अथवा प्रश्न विचारु इच्छित असल्यास खालील दिलेल्या तपासणीस संपर्क साधु शकता.

तपासणीसाची सही
नांव :
दिनांक

रुग्णाची सही
नांव
दिनांक

साक्षीदाराची सही
नांव :
दिनांक :

संपर्क क्रमांक
डॉ. चंद्रशेखर वायकोस : ९४२२८७१६००
डॉ. मेघराज इंगळे : ९३२०९७९६५९
डॉ. योगेश गायकर : ९९८७५१९५१६

सुचित सहमति फॉर्म

पित्तज ग्रहणी यह वैद्यकीय निदान किए हुए रुग्णों मे ग्रहणी शारिर के संदर्भ मे पाचन संस्थान का दुर्बीण द्वारा मुल्यांकन.

मेरे डॉक्टरोंने विस्तारसे अध्ययन के बारे में बताया है । मै अध्ययन में भागीदारी स्वैच्छिक है की समझते है और मैं किसीभी कारण देनेके बिना किसीभी समय वापस लेने के लिए स्वतंत्र हुँ. अध्ययन में भाग लेने के लिए सहमति के लिए मेरे मना करने के लिए किसीभी तरह से अपनी चिकित्सीय देखभाल को प्रभावित नहीं करेगा ।

मैने पढा है और रोगी सुचना पत्रक में दी गई सूचना को समझ लिया है. मै विस्तार से अध्ययन की प्रकृति और उद्देश्यकी व्याख्या की गई है ।

मैं इसके द्वारा बलके बिना, स्वच्छासे ऊपर उल्लेख अध्ययन में भाग लेने के लिए सहमत है ।
आप किसी भी प्रश्न पूछना चाहते है, तो आप नीचे दिए गये जांचकर्ताओं के किसी भी संपर्क कर सकते है ।

अन्वेषक के हस्ताक्षर :
नाम :
दिनांक

रुग्ण के हस्ताक्षर :
नाम
दिनांक

गवाह के हस्ताक्षर
नाम :
दिनांक :

संपर्क नंबर
डॉ. चंद्रशेखर वायकोस : ९४२२८७१६००
डॉ. मेघराज इंगळे : ९३२०९७९६५९
डॉ. योगेश गायकर : ९९८७५१९५१६

SR N	IPD NO	DATE	age	sex	occupation	desh	agni	jaranshakti	SYMPTOMS-DIARRHOEA					
									FREQUENCY OF STOOLS		Duration	VOLUME	consistency of	undigested food
									DAY TIME	NOCTURNAL				
1	2015020208	25-02-15	33	F	house wife	annop	manda	heen	5 to 6 times a day		6 months	large	liquidy	_
2	2015030120	18-03-15	52	M	Teacher	anoop	manda	heen	4to 5 times a day		3 montha	large	semisolid	_
3	2015040147	17-04-15	38	F	Teacher	anoop	manda	heen	4to 5 times a day		4 months	large	liquidy	f+
4	2015050151	22-05-15	24	F	steno	anoop	visham	madhyam	5 to 6 times a day		4 months	large	liquidy	_
6	180625	14-09-15	46	F	house wife	anoop	manda	madhyam	5 to 6 times a day		4 months	large	liquidy	_
7	107395	14-09-15	32	M	salesman	anoop	manda	heen	4to 5 times a day		4 months	large	liquidy	f+
8	162590	14-09-15	40	F	house wife	anoop	visham	madhyam	5TO 6times a day		4 months	large	liquidy	f+
9	115956	16-09-15	30	F	house wife	anoop	visham	madhyam	10to 12 times a day		3 montha	large	semisolid	_
10	154727	16-09-15	30	F	house wife	anoop	visham	uttam	5TO 6times a day		2 months	large	semisolid	_
11	57139	24-09-15	24	M	student	anoop	manda	heen	4to 5 times a day		3 montha	large	semisolid	_
12	40311	30-09-15	38	M	clerk	anoop	manda	madhyam	8 to 10 times a day		4 months	large	liquidy	_
13	175353	03-10-15	28	M	worker	anoop	visham	heen	3 to 4 times a day		5 months	large	liquidy	_
14	196854	12-10-15	37	F	officer	anoop	visham	heen	7 to 8times aday		4 months	large	semisolid	f+
15	58968	12-10-15	40	M	mechanic	anoop	manda	heen	8 to 10 times a day		3 months	large	liquidy	_
16	73917	30-11-15	19	M	student	anoop	manda	heen	6 TO 7 TIMES A DAY		5 months	large	liquidy	_
17	74546	01-12-15	20	M	student	anoop	visham	madhyam	8 to 9 times a day		4 months	large	liquidy	_
18	77299	16-12-15	25	F	clerk	anoop	manda	heen	3 to 4 times a day		5 months	large	liquidy	_
19	244659	22-12-15	59	M	retired	anoop	tikshna	uttam	8 to 9 times a day		3 months	large	semisolid	_
20	250492	22-12-15	58	F	house wife	anoop	manda	heen	5 to 6 times a day		4 months	large	liquidy	_
21	224587	22-12-15	27	F	clerk	anoop	visham	madhyam	3 to 4 times a day		5 months	large	liquidy	f+
22	239731	24-12-15	50	M	worker	anoop	visham	madhyam	4 to 5 times a day		3 months	large	liquidy	f+
23	271440	24-12-15	57	M	retired	anoop	manda	heen	8 to 9 times a day		4 months	large	semisolid	_
24	79178	28-12-15	35	F	house wife	anoop	manda	heen	4 to 5 times a day		3 months	large	semisolid	_
25	16370	28-12-15	40	F	librarian	anoop	visham	madhyam	6 TO 7 TIMES A DAY		4months	large	liquidy	f+
26	251497	30-12-15	19	F	student	anoop	manda	madhyam	4 to 5 times a day		4 months	large	liquidy	_
27	78668	30-12-15	19	M	student	anoop	manda	heen	4 to 5 times a day		3 months	large	semisolid	_
28	226570	30-12-15	20	F	student	anoop	manda	madhyam	7 to 8 times aday		4 months	large	liquidy	_
29	280318	30-12-15	24	M	salesman	anoop	tikshna	uttam	7 to 8 times aday		6 months	large	liquidy	_
30	78884	04-01-16	18	F	student	anoop	manda	heen	4 to 5 times a day		4 months	large	semisolid	f+
31	254999	04-01-16	32	F	house wife	anoop	manda	heen	5 to 6 times a day		3 months	large	liquidy	f+
32	80749	04-01-16	30	M	officer	anoop	manda	heen	4 to 5 times a day		4 months	large	liquidy	_
33	1115	09-01-16	59	F	house wife	anoop	manda	heen	4 to 5 times a day		4 months	large	liquidy	_
34	233596	11-01-16	35	M	mechanic	anoop	manda	heen	7 to 8 times aday		6 months	large	liquidy	f+
35	1641	14-01-16	30	M	shopkeeper	anoop	manda	heen	4 to 5 times a day		9 months	large	liquidy	_
36	1321	14-01-16	58	F	house wife	anoop	visham	madhyam	3 to 4 times a day		4 months	large	liquidy	_

37	8131	14-01-16	50	M	businessman	anoop	manda	madhyam	8 to 10 times a day		4 months	large	liquidy	f+
38	2346	20-01-16	59	M	retired	anoop	manda	heen	7 to 8 times aday		3 months	large	semisolid	f+
39	2797	20-01-16	25	F	steno	anoop	manda	madhyam	4 to 5 times a day		4 months	large	liquidy	_
40	3188	20-01-16	43	M	gardener	anoop	manda	heen	5TO 6times a day		4months	large	liquidy	_
41	2190		38	F	clerk	anoop	visham	madhyam	4to 5 times a day		6months	large	liquidy	_
42	4461	25-01-16	32	F	house wife	anoop	visham	madhyam	7 to 8 times a day		6 months	large	liquidy	_
43	3037	25-01-16	47	F	house wife	anoop	visham	uttam	6 to 8 times day		3 months	large	liquidy	_
44	7883	25-01-16	32	F	clerk	anoop	manda	heen	3 to 4 times a day		4 months	large	semisolid	f+
45	4868	01-02-16	49	M	mechanic	anoop	manda	madhyam	7 to 8 times a day		6 months	large	liquidy	_
46	209025	01-02-16	40	F	service	anoop	visham	heen	7 to 8 times a day		3 months	large	liquidy	_
47	6053	05-02-16	35	M	shopkeeper	anoop	visham	heen	8 to 10 times a day		7 months	large	liquidy	_
48	21335	15-02-16	33	F	house wife	anoop	manda	heen	5 to 6 times aday		6 months	large	liquidy	_
49	4616	16-02-16	20	F	student	anoop	manda	heen	6 to7 times day		3 months	large	liquidy	_
50	22986	16-02-16	20	F	student	anoop	visham	heen	3 to 4 times a day		4 months	large	semisolid	_
51	13299	10-03-16	20	M	student	anoop	manda	heen	10 TO 12 TIMES A DAY		3 months	large	liquidy	f+
52	6801	16-03-16	26	F	clerk	anoop	visham	madhyam	7 to 8 times day		3 months	large	liquidy	f+
53	14589	16-03-16	21	M	student	anoop	manda	heen	5 to 6 times aday		3 months	large	liquidy	_
54	15707	21-03-16	32	M	worker	anoop	tikshna	uttam	10 TO 15 TIMES A DAY		3 months	large	liquidy	_
55	6103	23-03-16	35	F	house wife	anoop	manda	heen	6 to7 times day		4 months	large	liquidy	f+
56	14880	31-03-16	23	M	salesman	anoop	visham	madhyam	4 to 5 times a day		4 months	large	liquidy	_
57	12261	06-04-16	45	F	house wife	anoop	visham	madhyam	5 to 6 times aday		4 months	large	liquidy	_
58	39157	06-04-16	45	F	Teacher	anoop	manda	heen	3 to 4 times a day		4 months	large	liquidy	_
59	18460	06-04-16	50	F	house wife	anoop	manda	heen	5 to 6 times aday		5 months	large	liquidy	_
60	71357	11-04-16	44	F	house wife	anoop	visham	madhyam	5 to 6 times aday		3 months	large	liquidy	_
61	48595	16-04-16	37	M	businessman	anoop	manda	madhyam	4 to 5 times aday		5 months	large	liquidy	f+
62	2016010166	16-04-16	57	M	retired	anoop	manda	heen	7 to 8 times a day		5 months	large	liquidy	f+
63	15276	27-04-16	35	F	clerk	anoop	manda	madhyam	4 to 5 times day		4 months	large	liquidy	_
64	22699	09-05-16	55	F	house wife	anoop	tikshna	uttam	5 to 6 times a day		4 months	large	semisolid	_
65	87407	18-05-16	24	M	officer	anoop	manda	heen	5 to7 times a day		3 months	large	liquidy	f+
66	49752	20-05-16	45	F	service	anoop	manda	heen	5 to 6 times a day		3 months	large	liquidy	_
67	19804	30-05-16	53	F	house wife	anoop	manda	heen	6 to 8 times day		4 months	large	liquidy	_
68	93853	01-06-16	26	M	salesman	anoop	manda	heen	4 to5 times day		4 months	large	liquidy	f+
69	73868	09-06-16	45	M	worker	anoop	manda	heen	6 to 8 times day		7 months	large	liquidy	f+
70	95485	15-06-16	30	F	officer	anoop	manda	heen	4 to 5 times day		3 months	large	liquidy	_
71	107819	15-06-16	21	M	student	anoop	manda	heen	6 TO 7 TIMES A DAY		3 months	large	liquidy	_
72		17-06-16	19	F	student	anoop	manda	heen	3 to 4 times a day		4 months	large	liquidy	_
73	20795	22-06-16	57	M	retired	anoop	manda	heen	6 TO 7 TIMES A DAY		3 months	large	semisolid	_
74	22103	22-06-16	25	M	mechanic	anoop	visham	madhyam	7 to 8 times a day		6 months	large	liquidy	f+
75	227801	23-06-16	35	F	house wife	anoop	manda	heen	6 to7 times a day		3 months	large	liquidy	_
76		24-06-16	56	F	house wife	anoop	manda	madhyam	8 to 10 times day		9 months	large	liquidy	_

77		27-06-16	34	M	officer	anoop	visham	madhyam	8 to 10 times day		4 months	large	liquidy	_
78		01-07-16	18	F	student	anoop	visham	madhyam	3 to 4 times day		3 months	large	liquidy	_
79	33971	29-06-16	58	M	retired	anoop	visham	uttam	7 to 8 times a day		4 months	large	liquidy	_
80	34987	29-06-16	25	M	courier boy	anoop	visham	madhyam	4 to 5 times aday		5 months	large	semisolid	_
81	106766	13-06-16	25	F	clerk	anoop	manda	heen	4 to 5 times a day		5 months	large	liquidy	f+
82	38421	15-07-16	40	F	house wife	anoop	manda	madhyam	5 to 8 times a day		4 months	large	liquidy	f+
83	108197	20-07-16	45	F	house wife	anoop	visham	heen	5 to 8 times a day		4 months	large	liquidy	_
84	124409	20-07-16	18	M	student	anoop	visham	heen	7 to 8 times a day		3 months	large	liquidy	_
85	122921	22-07-16	57	F	house wife	anoop	manda	heen	7 to 8 times a day		3 months	large	liquidy	f+
86	76581	22-07-16	19	M	student	anoop	manda	heen	4 to 5 times a day		4 months	large	liquidy	_
87	38765	22-07-16	45	F	house wife	anoop	visham	madhyam	5 to8 times day		4 months	large	liquidy	_
88	28108	02-08-16	55	F	house wife	anoop	manda	heen	6 to 8 times day		5 months	large	liquidy	_
89	136029	05-08-16	27	M	clerk	anoop	tikshna	uttam	4 to 6 times day		5 months	large	liquidy	_
90	43925	05-08-16	32	M	clerk	anoop	manda	heen	5 to 8 times a day		6 months	large	liquidy	_
91	44386	10-08-16	37	F	Teacher	anoop	visham	madhyam	3 to 4 times day		7 months	large	liquidy	f+
92	1386678	10-08-16	54	F	house wife	anoop	visham	madhyam	6 to 8 times day		3 months	large	liquidy	f+
93	1082	18-08-16	56	F	house wife	anoop	manda	heen	4 to 6 times day		3 months	large	liquidy	_
94	46121	26-08-16	19	M	student	anoop	manda	heen	4 to 6 times day		4 months	large	semisoild	_
95	160325	14-09-16	50	M	officer	anoop	visham	madhyam	7 to10 times day		5 months	large	liquidy	f+
96	121261	14-09-16	22	F	student	anoop	manda	madhyam	4 to6 times day		4 months	large	liquidy	_
97	53258	14-09-16	53	F	house wife	anoop	manda	heen	6 to8 times day		5 months	large	liquidy	_
98	52788	14-09-16	31	F	clerk	anoop	visham	madhyam	4 to 6 times a day		5 months	large	liquidy	f+
99	144362	14-09-16	39	F	Teacher	anoop	manda	heen	3 to 6 times day		4 months	large	liquidy	f+
100	51261	14-09-16	40	M	officer	anoop	manda	madhyam	4 to 6 times a day		3 months	large	semisolid	_
101	150751	14-09-16	30	M	officer	anoop	manda	heen	3 to 6 times day		4 months	large	liquidy	_
102	53979	20-09-16	54	M	retired	anoop	tikshna	uttam	4 to 5 times day		4 months	large	semisolid	_
103	135734	20-09-16	27	M	worker	anoop	manda	heen	7 to 10 times day		5 months	large	liquidy	_
104	32186	20-09-16	35	M	pharmacist	anoop	manda	heen	5TO 6times a day		4 months	large	liquidy	f+
105	34976	20-09-16	55	M	retired	anoop	manda	heen	7 to 10 times day		4 months	large	liquidy	_
106	55275	29-09-16	43	F	house wife	anoop	manda	heen	5 to 6 times a day		5 months	large	liquidy	_
107	157086	01-10-16	47	M	officer	anoop	manda	heen	4 to 5 times aday		4 months	large	semisolid	
108	201608008	09-08-16	40	M	OFFICER	anoop	visham	madhyam	5TO 6times a day		5 months	large	liquidy	f+
109	201630109	11-03-16	51	F	HOuse wife	anoop	manda	heen	7 to 10 times day		6 months	large	liquidy	f+
110		17-06-16	19	F	STUDENT	anoop	manda	heen	6 to 8 times day		4 months	large	liquidy	_

							ABD PAIN	VOMITING	BURPING	ANOREXIA	WEIGHT LOSS	BLOATING
blood	mucus	OIL	FLATUS	URGENCY	Borborygmi	SMELL						
_	_	_	(+)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	_	(-)	(-)	(+)	(+)	n	n	n	y	y	y
_	_	_	(-)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	o+	(+)	(+)	(+)	(-)	Y	n	y	y	y	y
_	_	_	(+)	(+)	(+)	(+)	Y	n	n	y	y	n
_	m+	_	(-)	(-)	(+)	(+)	n	n	y	y	y	n
_	_	_	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	o+	(+)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	_	(-)	(-)	(+)	(-)	n	n	n	y	y	n
_	m+	o+	(+)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	o+	(-)	(+)	(-)	(+)	Y	n	y	y	y	n
_	mucus ++	o+	(+)	(-)	(+)	(-)	Y	n	y	n	y	n
_	_	_	(+)	(-)	(+)	(-)	Y	y	y	y	y	n
_	mucus +	_	(-)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	_	(-)	(+)	(+)	(-)	Y	y	y	y	y	y
_	mucus +	_	(-)	(-)	(-)	(+)	Y	n	y	y	y	n
_	m+	o+	(+)	(-)	(+)	(-)	n	n	y	y	y	n
_	_	_	(+)	(-)	(+)	(-)	n	n	y	n	y	n
_	_	O+	(+)	(+)	(-)	(+)	Y	n	n	y	y	n
_	_	_	(-)	(+)	(-)	(-)	Y	n	y	y	y	n
_	m+	_	(+)	(-)	(+)	(+)	n	n	y	y	y	n
_	m+	o+	(-)	(-)	(-)	(+)	n	n	y	y	y	n
_	_	_	(+)	(+)	(-)	(-)	Y	n	y	y	y	n
_	m+	_	(-)	(+)	(+)	(-)	n	y	y	y	y	n
_	_	o+	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	o+	(-)	(+)	(-)	(+)	n	n	y	y	y	n
_	_	_	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	o+	(+)	(-)	(+)	(-)	Y	n	n	y	y	n
_	m+	_	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	_	(+)	(-)	(+)	(-)	n	y	y	y	y	n
_	_	_	(-)	(+)	(+)	(+)	Y	y	y	y	y	n
_	m+	_	(-)	(+)	(+)	(-)	Y	y	y	y	y	n
_	_	_	(+)	(-)	(+)	(-)	n	n	y	y	y	n
_	_	o+	(+)	(+)	(+)	(-)	n	y	y	y	y	n
_	m+	_	(-)	(-)	(+)	(+)	Y	n	y	y	y	n

_	m+	_	(-)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	_	(+)	(+)	(+)	(+)	n	y	y	y	y	n
_	m+	_	(-)	(+)	(+)	(+)	Y	n	y	y	y	n
_	_	o+	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
_	_	_	(+)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	o+	(+)	(+)	(+)	(+)	Y	n	y	y	y	n
_	m+	o+	(-)	(-)	(+)	(+)	Y	n	y	y	y	n
_	_	_	(-)	(-)	(+)	(+)	Y	n	y	y	y	n
_	M+	_	(+)	(+)	(+)	(+)	n	n	y	y	y	n
_	_	_	(+)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	_	(-)	(-)	(+)	(-)	Y	n	n	y	y	y
_	_	o+	(+)	(+)	(+)	(-)	Y	y	y	y	y	n
_	_	_	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
_	_	_	(+)	(-)	(+)	(-)	n	n	y	y	y	n
_	m++	_	(+)	(+)	(-)	(-)	n	n	n	y	y	n
_	m+	_	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	o+	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
_	m+	_	(+)	(+)	(-)	(+)	n	n	y	y	y	n
_	_	_	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	o+	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
_	_	o+	(-)	(+)	(+)	(-)	Y	y	n	y	y	n
_	_	_	(-)	(-)	(+)	(-)	n	n	y	y	y	n
_	m+	o+	(-)	(-)	(+)	(-)	n	n	y	y	y	n
_	m+	_	(-)	(-)	(+)	(-)	Y	n	n	y	y	n
_	_	_	(-)	(-)	(+)	(-)	Y	n	y	y	y	y
_	_	_	(+)	(+)	(+)	(+)	Y	y	y	y	y	n
_	_	_	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
_	m+	_	(-)	(+)	(+)	(-)	n	n	y	y	y	n
_	_	_	(-)	(+)	(+)	(-)	Y	n	n	y	y	n
_	_	o+	(-)	(+)	(+)	(-)	n	n	y	y	y	n
	_	_	(-)	(-)	(-)	(-)	Y	n	y	y	y	n
	_	_	(-)	(-)	(-)	(+)	Y	n	y	y	y	n
	_	_	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	m+	o+	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
	m+	_	(-)	(+)	(+)	(-)	n	n	n	y	y	n
	m+	o+	(-)	(-)	(+)	(-)	Y	y	y	y	y	n
	_	o+	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	_	_	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	_	_	(-)	(-)	(+)	(-)	n	n	y	y	y	n
	_	_	(-)	(+)	(+)	(-)	n	y	n	y	y	n

	m+	_	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
	m+	o+	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
	_	_	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	m+	o+	(-)	(-)	(+)	(-)	n	n	y	y	y	n
	_	_	(-)	(-)	(+)	(+)	Y	y	n	y	y	y
	m+	_	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	_	o+	(-)	(-)	(+)	(-)	Y	n	y	n	y	n
	_	_	(-)	(+)	(+)	(-)	Y	n	n	y	y	n
	_	_	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
	m+	o+	(-)	(-)	(-)	(+)	n	n	y	y	y	n
	_	o+	(-)	(-)		(-)	Y	y	y	y	y	n
	_	_	(-)	(+)	(+)	(+)	Y	n	y	y	y	n
	_	o+	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
	m+	_	(-)	(-)	(+)	(-)	n	n	y	y	y	n
	m+	_	(-)	(-)	(+)	(+)	n	n	y	y	y	n
	m+	_	(+)	(-)	(+)	(-)	Y	y	n	n	y	n
	_	_	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	_	_	(-)	(-)	(+)	(-)	n	n	y	y	y	n
	m+	_	(+)	(-)	(+)	(+)	Y	n	y	y	y	n
	m+	o+	(-)	(+)	(+)	(-)	Y	n	y	y	y	n
	m+	_	(+)	(-)	(-)	(+)	Y	n	y	y	y	n
	_	_	(+)	(-)	(+)	(-)	Y	y	n	y	y	n
	_	_	(-)	(+)	(-)	(-)	n	n	y	n	y	n
	m+	_	(+)	(-)	(-)	(-)	n	n	y	y	y	n
	m+	_	(-)	(-)	(-)	(-)	Y	n	n	y	y	n
	_	_	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
	m++	_	(-)	(-)	(+)	(-)	n	y	y	y	y	n
	m++	_	(+)	(-)	(+)	(-)	Y	n	y	y	y	n
	_	_	(-)	(-)	(+)	(+)	Y	n	n	n	y	n
	m++	o+	(+)	(+)	(+)	(-)	n	n	y	y	y	n
	_	o+	(-)	(-)	(+)	(-)	Y	n	y	y	y	n
	m+	o+	(+)	(-)	(+)	(+)	n	n	n	y	y	n
	_	o+	(+)	(-)	(+)	(-)	y	y	y	n	y	n
	m+	_	(-)	(-)	(+)	(-)	n	n	n	y	y	n

STOMATITIS	THIRST	SKIN RASH	JOINT PAIN	PERSONAL HISTORY			GENERAL EXA	PULSE	BP	TONGU	CONJU	SCLERA	PALLOR	ICTERU
				DIET	ALCOHOL	SMOKING								
y	y	n	n	mixed	n	n		72	114/84	N	N	N	+	-
y	y	n	n	mixed	n	n		80	110/80	N	N	N	-	-
y	y	n	n	mixed	n	n		76	104/76	N	N	N	+	-
y	n	n	n	mixed	n	n		80	100/74	N	N	N	-	-
n	y	n	n	mixed	n	n		70	108/72	y	N	N	+	-
y	n	n	n	mixed	y	n		68	112/70	N	N	yellow	-	+
n	y	n	n	mixed	n	n		72	116/70	N	y	N	+	-
y	y	n	n	mixed	n	n		78	120/72	N	N	N	-	-
n	n	n	n	mixed	n	n		74	118/74	N	N	N	-	-
n	y	n	n	mixed	n	n		68	116/70	N	y	N	+	-
n	y	n	n	mixed	n	y		78	110/70	N	N	N	-	-
n	n	n	n	mixed	y	y		64	100/68	N	N	N	-	-
n	y	n	n	mixed	n	n		68	102/66	N	N	N	-	-
y	y	n	y	mixed	y	n		70	108/68	N	y	N	+	-
y	y	n	n	mixed	n	n		80	104/70	N	N	N	-	-
y	y	n	n	mixed	n	n		72	100/68	N	y	N	+	-
y	n	n	n	mixed	n	n		70	102/66	y	y	N	+	-
y	y	n	n	mixed	y	y		68	114/70	N	N	yellow	-	+
y	y	n	y	mixed	n	n		74	112/68	N	y	N	+	-
n	y	n	n	mixed	n	n		76	112/70	N	N	N	+	-
n	y	n	n	mixed	y	n		80	108/68	y	y	N	+	-
n	n	n	y	mixed	n	y		72	104/64	N	N	N	+	-
y	y	n	n	mixed	n	n		66	106/68	N	N	N	-	-
y	y	n	n	mixed	n	n		68	108/64	N	N	N	+	-
n	y	n	n	mixed	n	n		64	110/62	y	N	N	+	-
y	n	n	n	mixed	n	n		70	112/64	N	N	N	+	-
y	y	n	n	mixed	n	n		72	114/70	N	y	N	+	-
n	y	n	n	mixed	y	y		66	102/68	N	N	N	+	-
n	n	n	n	mixed	n	n		70	104/68	N	y	N	+	
y	y	n	n	mixed	n	n		68	108/66	N	N	N	+	-
n	n	n	n	mixed	n	n		66	106/64	y	N	N	+	-
y	y	n	y	mixed	n	n		72	104/66	y	N	N	+	-
y	y	n	n	mixed	y	n		78	114/62	N	y	N	+	-
n	y	n	n	mixed	n	y		68	116/68	y	N	N	+	-
y	n	n	n	mixed	n	n		72	112/68	N	N	N	+	-

n	y	n	n	mixed	y	y		66	114/66	N	y	N	+	-
n	n	n	n	mixed	n	n		72	100/64	N	N	N	-	-
n	y	n	n	mixed	n	n		68	110/64	N	N	N	-	-
n	n	n	n	mixed	n	n		70	112/68	N	y	N	+	-
n	y	n	y	mixed	n	n		72	116/66	N	N	N	+	-
n	n	n	n	mixed	n	n		68	118/70	y	N	N	+	-
y	y	n	n	mixed	n	n		70	114/58	N	y	N	+	-
n	n	n	n	mixed	n	n		72	112/60	N	N	N	-	-
n	y	n	n	mixed	y	n		68	118/68	N	N	N	-	-
n	y	n	n	mixed	n	n		76	116/66	N	N	N	-	-
n	n	n	n	mixed	y	y		68	118/68	y	N	N	+	-
y	y	n	n	mixed	n	n		72	114/64	N	N	N	-	-
n	n	n	n	mixed	n	n		74	112/62	N	y	N	+	-
n	y	n	n	mixed	n	n		76	114/60	N	N	N	-	-
y	y	n	n	mixed	n	n		64	116/62	N	N	N	-	-
n	y	y	n	mixed	n	n		66	114/64	N	y	N	+	-
n	n	n	n	mixed	n	n		68	116/66	N	N	N	+	-
n	y	n	n	mixed	y	y		70	112/64	y	N	N	+	-
y	y	n	n	mixed	n	n		72	114/66	N	y	N	+	-
n	n	n	n	mixed	n	y		74	112/62	N	N	N	-	-
n	y	n	n	mixed	n	n		76	114/64	N	N	N	+	-
n	y	n	n	mixed	n	n		68	112/66	N	N	N	-	-
y	y	n	n	mixed	n	n		70	112/64	N	N	N	+	-
n	n	n	n	mixed	n	n		76	114/64	N	y	N	+	-
n	y	n	n	mixed	n	n		72	116/66	N	y	N	+	-
y	y	n	n	mixed	n	n		68	104/68	N	N	N	-	-
n	n	n	n	mixed	n	n		70	106/66	N	N	N	-	-
n	y	n	n	mixed	n	n		68	108/64	N	N	N	-	-
n	y	n	y	mixed	n	n		70	104/66	N	N	N	-	-
y	n	n	n	mixed	n	n		72	104/68	N	N	N	-	-
n	y	n	n	mixed	n	n		74	104/66	N	y	N	+	-
n	y	n	n	mixed	y	n		68	106/64	N	N	N	+	-
y	n	n	n	mixed	n	y		70	106/66	N	y	N	+	-
n	y	n	n	mixed	n	n		74	102/64	y	N	N	+	-
n	y	n	n	mixed	n	n		68	108/62	N	N	N	+	-
y	n	n	n	mixed	n	n		70	104/66	N	y	N	+	-
n	y	n	n	mixed	n	n		74	106/64	N	N	N	+	-
n	y	n	n	mixed	y	n		66	104/66	N	N	N	-	-
n	n	n	n	mixed	n	n		64	102/64	N	N	N	-	-
n	y	n	n	mixed	n	n		68	100/66	N	y	N	+	-

y	n	n	n	mixed	y	y		70	102/68	N	y	N	+	-
n	n	n	n	mixed	n	n		72	102/64	N	N	N	-	-
n	y	n	n	mixed	y	n		74	104/66	N	N	N	+	-
n	y	n	n	mixed	n	n		70	104/66	N	N	N	-	-
n	n	n	y	mixed	n	n		76	102/62	N	N	N	-	-
y	n	n	n	mixed	n	n		80	112/68	y	N	N	+	-
n	y	n	n	mixed	n	n		70	112/64	N	y	N	+	-
n	y	n	n	mixed	n	n		72	108/66	N	N	N	-	-
n	n	n	n	mixed	n	n		74	106/66	N	N	N	+	-
n	y	n	n	mixed	n	n		76	102/68	N	N	yellow	-	+
n	y	n	n	mixed	n	n		68	102/64	N	N	N	-	-
y	n	n	n	mixed	n	n		66	104/66	N	N	N	-	-
n	y	n	n	mixed	n	y		70	102/64	N	y	N	+	-
n	y	n	n	mixed	n	y		68	102/62	y	N	N	+	-
n	n	n	n	mixed	n	n		70	104/64	N	y	N	+	-
n	y	n	n	mixed	n	n		72	106/66	N	N	N	-	-
n	y	n	n	mixed	n	n		68	104/64	N	N	N	-	-
y	n	n	y	mixed	n	n		70	110/64	N	N	N	-	-
n	y	n	n	mixed	y	n		72	112/66	N	N	N	-	-
n	y	n	n	mixed	n	n		74	114/68	N	N	N	-	-
n	n	n	n	mixed	n	n		74	112/68	N	N	N	-	-
n	y	n	n	mixed	n	n		76	102/70	y	N	N	+	-
y	y	n	n	mixed	n	n		68	108/66	N	y	N	+	-
n	n	n	n	mixed	y	n		70	112/68	N	N	yellow	-	+
n	y	n	n	mixed	n	y		72	110/64	N	N	N	-	-
n	y	n	n	mixed	y	y		74	108/64	N	N	N	+	-
n	n	n	n	mixed	y	n		76	106/62	N	N	N	-	-
n	y	n	n	mixed	n	n		78	108/64	N	N	N	-	-
y	y	n	n	mixed	y	n		80	104/68	N	N	N	-	-
n	n	n	n	mixed	n	n		82	104/64	N	N	N	-	-
y	n	n	n	mixed	n	n		84	110/68	N	N	N	-	-
n	y	n	n	mixed	n	n		80	106/70	N	y	N	+	-
n	n	n	y	mixed	y	y		76	104/74	N	N	N	-	-
y	y	n	n	mixed	n	n		68	106/76	N	y	N	+	-

OEDEMA	STOMA	SKIN(kr	SYSTEMIC EXAMINATION										INVESTIGATIONS		cbc	
			GI										STOOL ROUTI	stool microscopic	Hb	Tlc
			ORAL	INSPEC	PALPATION				AUSCULTATION							
						TENDERNE	SPLEEN	LIVER		RS	CVS	CNS				
y	y	y	N	N	N	-	NP	N	N	N	N	N	N	N	9	4300
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	6-8PUS CELLS	10	6000
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	N	8.6	5600
y	y	y	ULCERA	N	N	-	NP	2cmHM	N	N	N	N	N	OCCULT BLD POS	9	6700
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.2	5600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	giardia lamblia+	8.7	6320
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.9	6700
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	12	9200
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	2to4 /hpfpuscells	14	9400
n	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.6	5400
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	14	7700
y	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	10.6	4600
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	10	8100
y	y	y	N	N	N	-	NP	N	N	N	N	N	N	4to5/hpf pus cells	9.4	7000
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.6	8400
y	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.8	7000
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	9	6500
y	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.3	4600
n	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.4	5600
y	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	7.8	6100
n	n	y	N	N	N	-	NP	2cmHM	N	N	N	N	N	fat globules	8.2	5400
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.6	5100
n	n	n	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.2	4800
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	8.8	4900
n	n	n	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.2	5200
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.9	6000
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	4 TO 6PUS CELLS	7.6	5700
n	n	n	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.2	3900
n	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	8.6	4200
y	y	n	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.9	4100
y	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8	6400
y	n	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.2	6700
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.9	7000
y	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.3	4800
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.6	3900

n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	8.7	5600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	9.1	6700
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.6	6600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	7.6	4800
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.3	5600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.4	5400
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	8.6	5600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	9.6	4600
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	9.3	5300
y	y	n	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.2	4500
n	y	n	N	N	N	-	NP	2cmHM	N	N	N	N	N	fat globules	8.6	4700
y	y	n	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	9.2	7000
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	8.6	6200
y	y	n	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	9.1	4890
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.2	4700
y	y	n	ULCERA	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	8.7	5200
y	y	n	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	7.5	5900
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.4	4900
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.3	6000
n	y	n	N	N	N	-	NP	2cmHM	N	N	N	N	N	fat globules	9.6	5500
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.2	4600
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	9.3	4500
y	n	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	7.2	4600
n	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.8	4000
n	n	y	N	N	N	-	NP	3cmHM	N	N	N	N	N	fat globules	8.2	4100
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	vegetable fibre	9.3	5600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	ova of giardia and fa	9.6	5800
n	y	n	ULCERA	N	N	-	NP	N	N	N	N	N	N	vegetable fibre and	9.7	4600
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	9.4	3900
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.6	4500
y	n	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.2	4800
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	7.4	6100
y	y	n	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.6	5600
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	8.2	4500
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	vegetable fibre and	8.6	4700
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.4	4900
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	7.9	3900
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	9.2	4500
y	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	9.3	5700
y	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.8	5200

n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	7.6	4900
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	10	4700
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	7.8	4800
n	n	y	N	N	N	-	NP	2cmHM	N	N	N	N	N	6 TO 8 PUS CELLS	9.8	4500
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	9.2	5400
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.5	5300
y	y	yn	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	8.3	5700
n	y	n.	ULCERA	N	N	-	NP	N	N	N	N	N	N	vegetable fibre	9.2	6800
y	y	y	N	N	N	-	NP	N	N	N	N	N	N	vegetable fibre and	8.2	4700
n	y	y	N	N	N	-	NP	2cmHM	N	N	N	N	N	1to 2/hpfpuscells	9.2	4600
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	9.8	5600
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	8.9	4800
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	7.9	4900
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	8.6	5100
n	y	n	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	8.2	4800
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	9.3	4900
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	vegetable fibre	9.5	5100
y	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	9.6	5200
n	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	fat globules	8.9	5400
y	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	vegetable fibre and	9.2	4700
n	n	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	9.4	4800
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.7	5100
n	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	vegetable fibre	8.8	5300
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	8.9	5400
y	n	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	9.3	5500
n	y	y	N	N	N	-	NP	2cmHM	N	N	N	N	N	fat globules	8.7	5600
y	n	n	N	N	N	-	NP	2cmHM	N	N	N	N	N	vegetable fibre	8.9	4700
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	1to 2/hpfpuscells	9.4	4800
y	y	y	N	N	N	-	NP	N	N	N	N	N	N	3TO4 PUSCELLS	9.2	4900
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	6 TO 8 PUS CELLS	9.1	5000
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	9.1	5100
n	y	y	N	N	N	-	NP	N	N	N	N	N	N	fat globules	8.7	4600
y	n	n	ULCERA	N	N	-	NP	3cmHM	N	N	N	N	N	fat globules	8.9	5100
n	y	y	ULCERA	N	N	-	NP	N	N	N	N	N	N	Vegetable fibre	9	4800

				upper GI Scopy									
Dc	Platelet co	MCV	peripheral smear	oesophagus	stomach	duodenum							
							high top folds		reduced nc	reduced height		scalloping	
	1.50L	92	MACROCYTIC								normal		
	1.60L	88	MICROCYTIC								normal	+	
	1.37L	96	MACROCYTIC								normal		
	1.09L	97	MACROCYTIC		Liinear erythrema in antrum and distal body						normal		
	1.72L	101	MACROCYTIC								+	+	
	1,56L	105	MACROCYTIC		multiple pre pyloric erosions						normal		
	1.9L	78	MICROCYTIC								normal		
	2.11L	93	MACROCYTIC		prolapsing gastropathy						normal		
	2.49 L	65	MICROCYTIC								normal		
	1.67L	94	MACROCYTIC								1 +	+	
	1.56L	86	MICROCYTIC								normal		
	1.35L	97	MACROCYTIC								normal		
	2.70L	98	NORMAL								normal	+	
	2.12 L	92	MACROCYTIC	oesaphageal candidiasis							1+	+	
	3.30L	86	MICROCYTIC		prolapsing gastropathy						normal		
	2.2L	89	MICROCYTIC								normal		
	1.76L	104	MACROCYTIC								1 +		
	1.56L	97	MACROCYTIC								normal		
	1.30L	99	MACROCYTIC		FUNDUS ATROPHY						normal		
	1.23L	107	MACROCYTIC									+	
	1.34L	92	MICROCYTIC								1+	+	
	1.40L	97	NORMAL								normal	+	
	1.42L	88	MICROCYTIC					REDUCED FOLDS			1+	+	
	1.40L	91	MICROCYTIC								normal		
	1.36L	105	MACROCYTIC									++	
	1.38L	108	MACROCYTIC		Gastritis						1+	+	ERYTHREMA
	1.23L	93	MACROCYTIC		GASTRITIS						NORMAL		
	1.43L	89	MICROCYTIC		ATROHIC GASTRIC MUCOSA						1+		
	1.36L	79	MICROCYTIC								normal	+	
	1.22L	88	MICROCYTIC								normal	+	
	1.34L	97	MACROCYTIC					REDUCED FOLDS			1+	+	
	1.44L	96	MACROCYTIC									+	
	1.20L	92	MICROCYTIC		PAN GASTRITIS								EROSIONS
	1.23L	94	MACROCYTIC									+	
	1.40L	99	MACROCYTIC								normal		

	1.32L	92	MICROCYTIC								normal		
	1.45L	106	MACROCYTIC									+	
	1.32L	94	MICROCYTIC								1+	+	
	1.33L	105	NORMAL					LOSS OF FOLDS					
	1.16L	96	MACROCYTIC								NORMAL	+	
	1,20L	97	MACROCYTIC								NORMAL	+	
	1.31L	93	MICROCYTIC								NORMAL		
	1.32L	108	MACROCYTIC		Prolapsing Gastro Pathy						MILD		
	1.18L	111	MACROCYTIC		GASTRITIS						NORMAL		
	1.22L	109	MACROCYTIC								NORMAL		
	1.45L	104	MACROCYTIC								MILD	+	
	1.36L	96	MACROCYTIC								MILD+	+	
	1.40L	89	MICROCYTIC								MILD	+	
	1.56L	90	MICROCYTIC								MILD	+	
	1.90L	93	MICROCYTIC		gastritis						NORMAL		
	1.86L	95	MACROCYTIC					reduced folds				+	
	1.56L	106	MACROCYTIC								reduced height		
	1.54L	107	MACROCYTIC		gastritis						NORMAL		
	1.60L	96	MACROCYTIC								NORMAL	+	
	1.96L	86	MACROCYTIC		prolapsing gastropathy						NORMAL		
	1.34L	79	MICROCYTIC		GASTRITIS						NORMAL		
	1.89L	93	MICROCYTIC								NORMAL	+	
	1.22L	96	MACROCYTIC								NORMAL		
	1.32L	90	MICROCYTIC		PAN GASRTITIS						NORMAL		
	1.54L	96	MACROCYTIC								NORMAL	+	
	1.65L	103	MACROCYTIC								NORMAL		
	1.72L	104	MACROCYTIC							+			
	1.88L	98	MACROCYTIC								NORMAL	+	
	1.74L	89	MACROCYTIC								NORMAL	+	
	1.80L	96	MACROCYTIC		PAN GASTRITIS						NORMAL		
	1.34L	94	MICROCYTIC		GASTRITIS						NORMAL		
	1.36L	97	MACROCYTIC		GASTRITIS						NORMAL		
	1.42L	98	MACROCYTIC		GASTRITIS						NORMAL		
	1.53L	95	MACROCYTIC								NORMAL		
	1.55L	101	MACROCYTIC								NORMAL		
	1.51L	102	MACROCYTIC					REDUCED FOLDS					
	1.40L	112	MACROCYTIC								NORMAL		
	1.80L	96	MACROCYTIC		GASTRITIS						NORMAL		
	1.85L	92	MACROCYTIC		GASTRITIS						NORMAL		
	1.66L	98	MACROCYTIC		GASTRITIS								

	1.56L	110	MACROCYTIC								NORMAL		
	2.10L	106	MACROCYTIC					reduced folds			NORMAL		
	1.44L	99	NORMAL								NORMAL	+	
	1.82L	96	MACROCYTIC							+	NORMAL		
	1.76L	100	MACROCYTIC								NORMAL	+	
	1.45L	105	MACROCYTIC								NORMAL		
	1.54L	97	MACROCYTIC		GASTRITIS						NORMAL		
	1.86L	106	MACROCYTIC								NORMAL	+	
	1.82L	107	MACROCYTIC					reduced folds			NORMAL		
	1.90L	108	MACROCYTIC		PAN GASTRITIS						NORMAL		
	2.15L	104	MACROCYTIC								NORMAL	+	
	1.78L	104	MACROCYTIC								NORMAL		
	1.45L	93	MICROCYTIC								NORMAL		
	1.56L	96	MACROCYTIC								NORMAL		
	1.60L	89	MICROCYTIC		gastritis						normal		
	1.89L	94	MICROCYTIC								NORMAL	+	
	1.92L	94	MACROCYTIC							+	NORMAL		
	1.83L	97	MACROCYTIC								NORMAL		
	1.72L	98	NORMAL								NORMAL	+	
	196L	95	MACROCYTIC		GASTRITIS								
	1.89L	101	MACROCYTIC		GASTRITIS							SCALLOPING+	
	1.45L	103	MACROCYTIC	PROLAPSING GASTROPATHY								SCALLOPING+	
	1.48L	104	MACROCYTIC	LES	GASTRITIS								
	1.44L	99	MACROCYTIC	LES	GASTRITIS LINEAR EROSIONS								
	1.96L	94	MACROCYTIC	LES	PAN GASTRITIS								
	1.54L	96	MACROCYTIC								normal	+	
	1.64L	97	MACROCYTIC		GASTRITIS								
	1.55L	98	MACROCYTIC	LAX LES									
	1.97L	87	MICROCYTIC					reducedfolds			normal	+	
	1.76L	95	MACROCYTIC								normal		
	1.80L	96	NORMAL		gastritis						normal	+	
	1.46L	97	MACROCYTIC								FLATTENIN	+	
	1.48L	101	MACROCYTIC	LAX LES							FLATTENIN	+	
	1.78L	95	MACROCYTIC					REDUCED FOLDS					

	histopathology												
	slide numb	villi	v:c	lamina propria		intra /sub	diagnosis						
ULCER IF FOUND													
	1631	partial mo	partial moder	MODERATE		5 TO 10	Duodenitis with partial moderaate villous atrophy						
	2254	HELMINTH	PARTIAL mod	MODERATE		2 TO 5	Duodenitis with partial moderaate villous atrophy and helminthiasis						
	3197	Partial mo	Partial moder	MODERATE		2 to 5	Duodenitis with partial moderate villous atrophy with focal ulceration						
	4312	NORMAL	normal	MILD		2TO 5	Mild duodenitis grade1						
	7510	NORMAL	normal	MILD		2TO5	Mild duodenitis grade1						
	7567	NORMAL	normal	MILD		2TO 5	Mild duodenitis grade1						
	7580	NORMAL	normal	MILD		2TO 5	Mild duodenitis grade1						
	7651	NORMAL	NORMAL	MILD		2TO5	MILD chronic DUODENITIS grade1						
	7677	NORMAL	NORMAL	MILD		2TO5	Mild duodenitis grade1						
		PARTIAL VI	PARTIAL VILL	MILD		5TO 10	GRADE 4 PARTIAL VILLOUS ATROPHY DUODENITIS						
	8102	PARTIAL VI	PARTIAL VILL	MILD		5TO 10	GRADE 4 PARTIAL VILLOUS ATROPHY DUODENITIS						
		NORMAL	normal	MILD		2TO5	Mild duodenitis grade1						
	8101	FOCAL SH	FOCAL SHORT	MODERATE		5 to10	moderate chronic duodenitis grade1						
		PARTIAL VI	PARTIAL VILL	MILD		5TO 10	GRADE 4 PARTIAL VILLOUS ATROPHY DUODENITIS						
		NORMAL	2.5:1	MODERATE		2TO5	moderate chronic nonspecific duodenitis grade1						
		NORMAL	2.5:1	MILD		2TO5	moderate chronic nonspecific duodenitis grade1						
	9970	NORMAL	3:01	MODERATE		5 to 10	moderate chronic duodenitis grade1						
	10144	NORMAL	3:01	MODERATE		2 TO 5	non specific chronic duodenitis grade1						
	10142	NORMAL	INADEQUATE	MODERATE		2TO 5	non specific chronic duodenitis grade1						
	10145	NORMAL	INADEQUATE	MODERATE		2TO 5	non specific chronic duodenitis grade1						
	10172	NORMAL	2.5:1	MODERATE		2 TO 5	moderate chronic nonspecific duodenitis grade 1						
	10175	NORMAL	4:01	MILD		2 TO 5	moderate chronic nonspecific duodenitis grade 1						
	10264	NORMAL	3:01	MILD		2 TO 5	mild chronic nonspecific duodenitis grade1						
	10291	NORMAL	NORMAL	MILD		2TO5	mild chronic nonspecific duodenitis grade1						
	10320	NORMAL	3:01	MILD		2 TO 5	mild chronic nonspecific duodenitis grade1						
	10321	NORMAL	1:02	MILD		5 TO10	mild chronic nonspecific duodenitis grade1						
	10327	NORMAL	4:01	MILD		5 TO10	mild chronic duodenitis grade1						
	10328	NORMAL	INADEQUATE	MILD		2TO5	mild chronic nonspecific duodenitis grade1						
	90	NORMAL	NORMAL	MILD		WNL	mild chronic nonspecific duodenitis grade1						
	88	NORMAL	NORMAL	MILD		WNL	mild chronic nonspecific duodenitis grade1						
	7383	PARTIAL VI	PARTIAL VILL	MODERATE		5TO 10	GRADE 4 PARTIAL VILLOUS ATROPHY DUODENITIS						
	243	NORMAL	NORMAL	MILD		2TO5	chronic duodenitis grade1						
		NORMAL	NORMAL	MIL;D		2TO5	chronic duodenitis grade1						
	396	ATROPHIC	4:01	MODERATE		35 TO 40							
	404	NORMAL	4:01	MILD		WNL	chronic duodenitis grade1						

	498	NORMAL	NORMAL	MILD		WNL	chronic duodenitis grade1				
	558	NORMAL	INADEQUATE	MILD		WNL	chronic duodenitis grade1				
	567	NORMAL	3:01	MILD		WNL	superficial duodenitis grade1				
	589	FLATTENIN	4:01	MODERATE		50TO 60					
	440	NORMAL	MORMAL	MILD		WNL	mild chronic duodenitis grade1				
	710	NORMAL	3:01	MILD		WNL	mild chronic duodenitis grade1				
	711	NORMAL	3:01	MILD		WNL	mild chronic duodenitis grade1				
	714	SHORETEN	1:05 SHORETE	MILD		5 TO 10	moderate ns duodenitis				
	967	NORMAL	2.5:01	MODERATE		5 TO 10	mild duodenitis grade1				
	970	NORMAL	3:01	MODERATE		5 TO 10	mild chronic duodenitis grade2				
	1039	SUPERFICIA	2.5:01	MILD		5 TO 10	mild chronic duodenitis grade1				
	1338	NORMAL	3:01	MILD		WNL	chronic duodenitis grade1				
	1329	NORMAL	4:01	MILD		WNL	chronic duodenitis grade1				
	1332	SUPERFICIA	2.5:01	MILD		5 TO 10	chronic duodenitis grade1				
	1912	NORMAL	3.5:01	MILD		15 TO 20	chronic duodenitis grade2				
	2121	FLATTENIN	2.5:1	MODERATE		50 TO 60	chronic duodenitis grade4				
	2122	NORMAL	2.5:1	MILD		WNL	mild duodenitis grade1				
	2215	tall	TALL	MILD		10 TO 15					
	2295	focal blunt	3:01	MILD		5TO 10	mild duodenitis grade1				
	2490	NORMAL	3:01	mild		WNL	mild duodentis grade1				
	2660	SHORETENING OF VILLI		SEVERE		WNL	chronic duodenitis grade1				
	2652	NORMAL	2.5:1	MILD		WNL	mild duodeniutis grade1				
	2651	NORMAL	3:01	MILD		WNL	chronic duodenitis grade2				
	2750	NORMAL	2.5:1	MILD		WNL	chronic duodenitis grade1				
	2885	FOCAL ATROPHY		MILD		5 TO 10	chronic duodenitis grade1				
	635	ATROPHY	ATROPHY	MODERATE		5 TO 10					
	3163	FOCAL SHORTENING OF V		MILD		5 TO 10	chronic duodenitis grade1				
	3385	NORMAL	NORMAL	MILD		WNL	mild non specific duodenitis				
	3618	NORMAL	NORMAL	MODERATE		5 TO 10	moderate duodenitis grade1				
	3805	NORMAL	UNDER LIMIT	MILD		WNL	mild chronic nonspecific duodenitis				
	3917	NORMAL	NORMAL	MILD		WNL	moderate duodenitis grade1				
	3978	NOALRM	NORMAL	MILD		WNL	moderate duodenitis grade1				
	4189	SHORTENING OF VILLI		MILD		8 TO 10	mild chronic nonspecific duodenitis				
	4382	NORMAL	NORMAL	MILD		WNL	chronic nonspecific duodenitis				
	4386	NORMAL	NORMAL	MILD		5 TO 10	mild duodenitis grade1				
	5655	NORMAL	NORMAL	UL		WNL	mild duodenitis grade1				
	4502	NORMAL	NORMAL	MILD		8 TO 10	chronic duodentis grade1				
	4503	NORMAL	NORMAL	MILD		5 TO 10	chronic duodentis grade1				
	4564	NORMAL	NORMAL	MILD		8 TO10	mild chronic duodentis grade1				
	5959	MODERATE DISTORTION		MODERATE		5 TO 10	DUODENITIS WITH MARKED VILLOUS ATROPHY				

	6012	NORMAL	NORMAL	UR		WNL	chronic duodenitis grade1					
	6185	NORMAL	NORMAL	UR		WNL	chronic duodenitis grade1					
	4712	FOCAL SHO	ATROPHY OF	MODERATE		WNL	chronic nonspecific duodenitis					
	4716	NORMAL	NORMAL	MILD		WNL	chronic nonspecific duodenitis					
	5005	NORMAL	NORMAL	MILD		5TO 10	mild chronic nonspecific duodenitis					
	5061	NORMAL	NORMAL	MILD		WNL	mild duodenitis grade1					
	5184	NORMAL	NORMAL	MILD		WNL	normal					
	5186	NORMAL	NORMAL	MILD		WNL	mild duodenitis grade1					
	5354	NORMAL	NORMAL	UR		WNL	normal					
	5353	NORMAL	NORMAL	MILD		WNL	mild duodenitis grade1					
	5349	NORMAL	NORMAL	MILD		WNL	normal					
	5494	NORMAL	NORMAL	MILD		WNL	MILD DUODENITIS grade1					
	5591	NORMAL	NORMAL	MILD		WNL	normal					
	5593	NORMAL	NORMAL	MILD		WNL	mild duodenitis grade1					
	5697	NORMAL	2:01	MILD		2TO5	mild chronic duodentis grade1					
	5695	NORMAL	2:01	MILD		2TO 5	mild chronic duodentis grade1					
	5923	NORMAL	NORMAL	MILD		2TO5	mild chronic duodentis grade1					
	6178	NORMAL	NORMAL	MILD		2TO5	mild chronic duodentis grade1					
	6596	NORMAL	NORMAL	UR		WNL	normal					
	6597	NORMAL	NORMAL	UR		WNL	normal					
	6611	NORMAL	NORMAL	mild		2 TO 5	mild duodenitis grade1					
	6621	NORMAL	NORMAL	MILD		2TO 5	mild duodenitis grade1					
NODULARITY	6602	NORMAL	NORMAL	UR		WNL	normal					
	6603	NORMAL	NORMAL	UR		WNL	normal					
	6608	NORMAL	NORMAL	UR		WNL	normal					
	6717	NORMAL	NORMAL	MILD		2 TO 5	MILD DUODENITIS grade1					
	6721	NORMAL	NORMAL	MILD		2 TO 5	MILD DUODENITIS grade1					
	6730	NORMAL	NORMAL	MILD		2TO5	mild duodenitis grade1					
	6724	NORMAL	NORMAL	UR		WNL	normal					
		NORMAL	NORMAL	MILD		WNL	mild duodenitis grade1					
		NORMAL	NORMAL	MILD		2TO 5	mild duodenitis grade1					
	7786	MODERATI	NORMAL	UR		WNL	MODERATE DUODENAL VILLOUS ATROPHY					
	2392	PARTIAL M	NORMAL	MILD		2TO5	DUODENITIS WITH PARTIAL MILD VILLOUS ATROPHY					
	5655	NORMAL	NORMAL	UR		WNL	normal					