EVALUATION OF BHEDANA KARMA OF YG3 IN YAKRUT VIKARA

A thesis submitted to

TILAK MAHARASHTRA VIDYAPEETH, PUNE

For the Degree of

DOCTOR OF PHILOSOPHY (Ph. D.)

Subject	**:**	**Kayachikitsa**
Under Faculty of	**:**	**Ayurved**
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Under the Guidance Of

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Name of the Department	**:**	**Ayurved**
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CERTIFICATE

This is to certify that the thesis entitled, **"Evaluation of Bhedana Karma of YG3 in YakrutVikar"** which is being submitted herewith for the award of the Degree of Vidyavachaspati (Ph.D.) in Ayurveda of TilakMaharashtra Vidyapeeth, Pune is the result of original research work completed by **Vd. Mrs.BhagyashreeSukumarSardeshmukh**under my supervision and guidance. To the best of my knowledge and belief the work incorporated in this thesis has not formed the basis for the award of any degree or similar title of this or any other University or examining body.

Place - Pune
Date – 15/12/2016

Prof. Dr.Vineeta V. Deshmukh
M.D. Ph.D. (Ayurved)

DECLARATION

I hereby declare that the thesisentitled “Evaluation of BhedanaKarma of YG3 in YakrutVikar” completed and written by me has not previously formed the basis for the award of any degree or other similar title or any other university or examining body.

Place - Pune

Date –15/12/2016

Vd.Mrs. Bhagyashree Sukumar Sardeshmukh

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INDEX

INTRODUCTION

Liver is one of the vital organs in the human body. It performs many significant functions likemetabolism, protein synthesis and detoxification of chemicals. If these functions are hampered it can lead to several conditions from hyperbilirubinemia to even hepatocellular carcinoma.

Chronic liver disorders are the major health problems around the world. Liver disease is the 5th most common cause of death.[1] The most important cause of liver disorders & especially liver cirrhosis is chronic alcohol consumption.[2]

Liver cirrhosis was estimated to be responsible for over one million deaths in 2010, which is approximately 2% of all deaths worldwide. Other causes for liver disorders include non-alcoholic fatty disorders, infections like hepatitis A, B etc. or overuse of medicines.[3]

Infections of liver also have a great impact on liver conditions. An estimated 240 million people worldwide are chronically infected with hepatitis B.

Together, hepatitis B and C are the most common causes of liver cirrhosis and hepatocellular cancer. All these disorders may land into fatal complication like portal hypertension, G.I. bleeding to ascitis or even Hepatocellular carcinoma.

The prevalence of Cancer in India is 2.5 million with over 8, 00,000 new cases & 5, 50,000 deaths occurring each year.[4] Most of these cases report for diagnostic & treatment services in the advanced stages which leads to poor survival & high mortality rate. This becomes an emotional & economical burden for the families of Cancer patients.

1. https://www.cdc.gov
2. http://www.healthcommunities.com/liver-disease/liver-disease-overview.shtml
3. Sherlock's Diseases Of The Liver And Biliary System, Dr. Dooley, Dr. Lok, Dr. Burroughs, Dr. Heathcote, 12th Edi, Wiley – Blackwell Publications, Pg. No. 219
4. http://www.indiancancersociety.org/what-do-we-do/pdf/poona-rep610.pdf

Various treatment modalities are employed to fight against Cancer including Surgery, Chemotherapy, Radiotherapy, Hormone therapy etc. These have been only limited success in the cure and till date cancer is incurable.

One of the Cancer types which has poor prognosis and survival is – Hepatocellular Carcinoma. It is the sixth most common cancer and a second most common cause of cancer death worldwide. There were 745,000 deaths due to liver cancer in 2012.[5]

HCC is one of the most serious results of cirrhosis and is responsible for 70-90% of cases of primary liver cancer. Prognosis in such cases is very poor as world wide data shows that no single drug or combination has given systematically reproducible leads to greater than 25% response rates or has any effect on survival. One of the important treatment modality for various liver disorders id Liver transplant. Liver is the 2nd most commonly transplanted major organ after kidney[6]**.** It is estimated that approximately 25,000 liver transplants were conducted globally in 2013 but the survival after transplantation is very low.[7]

Ayurved has explained Liver as Yakrut. Ayurved considers Yakrut as the main reservoir for blood (Moolasthana of Raktavahasrotasa).[8] It plays a vital role in the process of production of Raktadhatu and it's circulation. It is also a Pitta sthana. When doshas get vitiated in Yakrut, it results in several Yakrutvikara. Thus the main treatment of Pitta – Raktavikaralways belongs to the treatment of liver.

Treatment of Yakryt Vikara explained in Ayurved includes Shodhana, Shaman and Pathyakara Aahara and Vihara. Amongst Shodhana chikitsa,Virechana and Raktamokshana are the treatments of choice of Yakrut Vikaras. They detoxify Pitta dosha and Rakta Dhatu. 4 types of Virechana are explained in Sharangadhar Samhita i.e.Anulomana, Stramsana, Bhedana and Rechana.[9] Amongst them Bhedana is a type

5. Cancer Principal and Practice of oncology, Vincent T. Devita, Jr.,Samuel Hellman , Steven A. Rosenberg, 7th edition, Pg. No. 1130
6. http://www.emedicinehealth.com/health-topics/article_em.htm
7. http://www.healthline.com/health-slideshow/liver-transplant
8. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Vimansthan, 5/8, Pg. No. 250
9. Sharangdhar Samhita (2000), Pt. Parashuram Shastri, Vidyasagar, Chaukhambha Orientalia, 4th Edi., 1st Part, 4/3-7, Pg.No. 35-36

of virechana which not only softens the stool and evacuates it from bowel, but it also eliminates accumulated and vitiated doshas, dhatus and malas from specific organs.Based on our previous experience of treating patients of Yakrut vikara, we have selected 3 medicines in this study for management of Yakrutvikara. Patients are divided in 2 groups to assess efficacy of bhedana action of these medicines along with their organ specific action. Among them Yakrut pleehari Rasa explained in Bhaishajya Ratnavali, Kutki churna and Kumari KalpaVati are administered to Group A patients of Yakrut vikara and first 2 medicines to Group B patients.

Thus the effects of Bhedana Karma of these Ayurvedic formulations will be studied in both the Groups.

AIM AND OBJECTIVES

Aim :

To evaluate the Bhedana Karma of YG3 in YakrutVikara.

(YG3 : Combination of Yakrut Pleehari LohaVati, Kutaki Churna & Kumari KalpaVati)

Objectives :

1) To standardize Yakrutplihari Loha Vati, Kutaki Churna and Kumari Kalpa Vati.
2) To assess the role of YG3 on symptoms of Yakrut Vikara
3) To evaluate the effect of YG3 on biochemical parameters such as Hemogram and liver function tests

LITRETURE REVIEW

A) LITERATURE REVIEW OF YAKRUT FROM AYURVEDIC PERSPECTIVE

A-I) Nirukti and synonym of Yakrut

Nirukti:

The word Yakrut is derived from the root kr with the prefix ya and suffix tuk. i.e. Yakrut is the organ that controls all the functions.

यक्नं मतस्नाभ्यां यक्नः प्नाशिभ्यो विवृहामिते (ऋग्वेद १०।१६३।३)[१०]

यं संयमं करोति कृ क्विप् तुक् च ।
यक्नं हृदयसमीपे वर्तमानः कालमांसविशेषो यकृत् तस्मात् । (शब्दकल्पद्रुम । खण्ड ४) [११]

यकृतक्लोमानां च पर्वताम् । बृहदारण्यक उपनिषद्[१२]

यं संयमं करोति कृ क्विप् तुक् च । कुक्षौ दक्षिणभागस्थे मांसपिण्डम् । तव्दर्धके रोगे च ।[१३]
(वाचस्पत्यम् । खण्ड ४)

Yakruta is known by Takima and Yakna in vedas.

Synonyms[14] :

कुक्षेः दक्षिणभागस्य मांसखण्डम् ।
तत्पर्यायः । कालखण्डम् । इति अमरः ।२।६।६६।
कालखल्लम् । कालेयम् । कालकम् । इति हेमचंद्रः ।
करण्डा / महास्नायु । इति रभसः ।

10. Rugved,Pt. satavyakar, Paradi sansthan, Gujrath, Pra. Samskaran – 1962
11. Shabdakalpadrum, Syar Raja- Radhakantadeo- Bahadurena Virachitaha, Nag Publishers, 1st Edi
12. Bruhadaaranyak Upanidshada, Geeta Press, Gorakhpur, Pu. Sanskaran 1962
13. Vachaspatyam, 6th Part, Shri Taranath Tarka vachaspati Bhattachharyena, Chaukhambha PublicationsPg.No. 2046
14. Shabdakalpadrum, Nag Publishers, 1st Edi

Kalakhandagam

Kalakhallam

Kaleya

Kalakam

Karanda

Mahasnayu

Mamsapinda

Jyotisthana – related to Agni

Raktadhara

A – II) YAKRUT SHARIR (Anotomy and Physiology of Yakrut from Ayurvedic perspective)

Yakrut Sthana :

Yakrut is a matruja avayava in koshthanga.[15]Yakrut has relation with kloma because both are situated in the right side of the heart.[16]Yakrut is also in relation with the part of small and large intestines where maladharakala is situated. [17]

पंचदश कोष्ठांगानि तद्यथ यकृच्च । (सु.शा. ९/१२)
हृदयस्य अधोदक्षिणतः यकृत । (सु.शा. ४/३०)
दक्षिणतो यकृत क्लोम च ।

यकृत्समन्तात् कोष्ठ च तथाडन्त्राणि समाश्रिता ।
उण्डुकस्थं विभजते मलं मलधरा कला (सु.शा. ४/१६)

Acharya Sushruta considered Yakrut and pleeha as originated from the blood. [18]

गर्भस्य यकृतप्लीहानौ शोणितजौ । (सु.शा. ४/२४)

15. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 9/12
16. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 4/30, Pg. No. 117
17. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 4/16, Pg. No. 110
18. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 4/24, Pg. No. 116

Yakrut as Aashaya:

Aashaya is Adhishthana. Susruta has mentioned seven and eight Aashaya in male and female respectively. These are Vatashaya, Pittashaya, Kaphashaya, Raktashaya, Amashaya, Pakvashaya, Mutrashaya and in female Garbhashaya. [19]

आशयास्तु वाताशयः पित्ताशयः श्लेष्माशयो रक्ताशयः आमाशयः
पक्वाशयो मूत्राशयः स्त्रीणां गर्भाशयोऽष्टम इति ।। (सु.शा. ५/७)

Pittashay Yukta Yakrut and Agnyashayais considered as Pittashaya while Yakruta with Pleeha is considered as Rasktashaya.

Yakrut and Kala:

Kala is dhatvashayantaramaryada i.e. which separates dhatu from the aashayas. Acharya Sushruta had mentioned seven types of kala out of which raktadharakala isspecifically found in sira, Yakrut and pleeha.[20]

व्दितीया रक्तधरा नाम मांसस्याभ्यंन्तरतः तस्या शाणितं विशेषश्च सिरासु यकृतप्लीह्नोश्च भवति ।। (सु.शा. ४/९)

Acharya Sharangadhara in addition to Raktadhara kala has described Yakrut and pleehadharakala.

Yakrut and Peshi (Muscles):

Out of the sixty-six muscles found in the abdomen, six are situated in relation to the Yakrut, pleeha and unduka.[21]

..... षट् यकृतप्लीहोण्डुकेषु ।। (सु.शा. ५/४७)

19. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 5/7, Pg. No. 150
20. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 4/9, Pg. No. 109
21. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 5/47, Pg. No. 170

Yakrut and Sira :

Acharya Susruta had described forty main sira out of which ten are raktavaha, which are bound to Yakrut and pleeha. These ten siras further get divided into 175-raktavahasira that supply rakta all over the body.[22]

..... रक्तवाहिन्यश्च यकृतप्लीन्होः एवमेतानि सप्त सिराशतानि । (सु.शा. ७/५)

When normal rakta flows through these sira throughout the body it nourishes all dhatus,gives complexion to body, touch sensation, and all such other functions. [23]

धातूनां पूरणं वर्णं स्पर्शज्ञानमसंशयम् ।
स्वाः सिराः संचरद्रक्तं कुर्याच्चान्याम् गुणानपि ।। (सु.शा. ७/१३)

But if the vitiated rakta flows through these sira, it causes various blood borne diseases.[24]

यदा तु कुपितं रक्तं सेवतं स्ववहाः सिराः ।
तदाडस्य विविधा रोगा जायन्ते रक्तसंभवाः ।। (सु.शा. ७/१४)

Yakrut and Agni :

आयुरारोग्यवीर्योजोभूतधात्वग्निपुष्टये ।
स्थिता पक्वाशयद्वारि भुक्तमार्गार्गलेव सा ।। (सु.शा. ३/५१)

Luster of the skin, its complexion, energetic condition and working capacity of the body, its general health, nutrition and growth, etc. and the life forces (prana) depend on agni. The normal functioning as well as the vitiation of dosha depends upon normal agni and hence the maintenance of normal agni is absolutely essential for health.[25]

22. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 7/5, Pg. No. 207
23. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 7/13-14, Pg. No. 208
24. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 7/13-14, Pg. No. 208
25. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 3/51, Pg. No. 394

Types of Agni

Thirteen types of agni are described in ayurvedic texts. These are:

- Kayagni or jatharagni
- Dhatvagni: 7 (rasadhatvagni, raktadhatvagni, mamsa- dhatvagni, medodhatvagni, asthidhatvagni, majjadhatvagni and sukradhatvagni
- Bhutagni: 5 (bhaumyagni, apyagni, taijasagni, vayavyagni and nabhasagni.)

Jatharagni:

This is central to all Agnisof the body. Food is first digested and absorbed by the gastrointestinal tract. This is jatharagnipaka. It is then transported to liver for bhutagnipaka and finally in the dhatus, by the dhatvagnipaka.

At every place, the pachanakriya (digestion) results in two products, the nourishing (Prasada Bhaga) and the waste (Kitta Bhaga).

These three are the normal metabolic level of activity and there can be three corresponding levels of defective metabolism traceable in turn to defective enzyme complex at the respective places or feeble digestive power.

Raktavahasrotas moolasthana:

The group of organs which produces and maintains raktadhatu (Raktotpatti karya) is called as raktavahasrotas.

- According to Acharya Charaka,Yakrut and pleeha are the moolasthana (Origin) of raktavahasrotas. [26]

शोणितवहाना स्त्रोतसां यकृन्मूलं प्लीहा च । (च.वि. ५/८)

26. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Vimansthan, 5/8, Pg. No. 250

- According to Acharya Sushruta, Yakrut, pleeha and raktavaha dhamanis are the moolasthana (Origin) of raktavahasrotas. [27]

रक्तवहे व्दे, तयोर्मूत्यं यकृप्लीहानौ रक्तवाहिन्यश्च धमन्यः।।

सु.शा. ९/१६

Raktavahasrotas

As Yakrut is stated to be the moola of raktavahasrotas, the function of Yakrut is seen through this srotas only. In the process of dhatuparinama, raktadhatu is stated to be developed from rasadhatu.According to Sushruta the ranjakapitta is located in Yakrut and pleeha.[28]

यत्तु यकृतलीह्नोः पित्तं तस्मिन् रन्जकोऽग्निरितति संज्ञा । (सु.सू. २१/१०)

There are three pittas that take part in this process - rasagni, raktagni and ranjakapitta. Acharya Vagbhata considers amashaya as the seat of ranjakapitta.[29]

आमाशयाश्रयं पित्तं रन्जकं रसरजनात् । (अ.हृ.सू. १२/१३)

Raktavaha strotas dushti

Raktavahasrotas is vitiated by following causes

विदाहीन्यन्नपानानि स्निग्धोष्णानि द्रवाणि च ।

रक्तवाहीनि दुष्यन्ति भजता। चातपानलौ । (च.वि. ५/१४)

1. Consumption of food having properties of vidahi, snigdha, ushna and drava.
2. Excessive exposure to the sun or fire [30]

27. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 9/16, Pg. No. 241
28. Sushrut Samhita (2005), Dr.Ambita Datta Shastri, Chaukhamba Sanskrit Samsthan, New Delhi, 1st Part, Sutrasthan, 21/10, Pg. No. 86
29. Asthanghruday (2000), Pt. BhisagacharyaParadkarVaidya, Krishnadas Academy, Varanasi, Sutrasthan, 12/13, Pg. No. 194.
30. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Vimansthan, 5/14, Pg. No. 251

Raktavaha strotas vidhha lakshane :

तत्र विध्दस्य श्यावाङ्गता ज्वरो दाहः पाण्डुता शोणितागमनं रक्तनेत्रता च

(सु.शा. ९/१६)

When the raktavaha strotasas are injured they cause[31] –

1. Shyavangata (Change in colour of body)
2. Jwara (Fever)
3. Daha (Burning sensation)
4. Panduta (Anaemia)
5. Shonitaagamanam (Discharge of blood)
6. Raktanetrata (Redness of eyes)

Rakta Dhatu Dushti : [32]

प्रदुष्टबहुतीक्ष्णोष्णौर्मद्यैरन्यैश्च तद्विधैः ।

तथाडतिलवणक्षारैरम्लैः कटुभिरेव च ।।

कुलत्यमाषनिष्पावतिलतैलनिषेवणैः ।

पिण्डालुमूलकादीनां हरितानां च सर्वशः ।।

जलजानूपबैलानां प्रसहानां च सेवनात् ।

दध्यम्लमस्तुसुक्तानां सुरासौषीरकस्य च ।।

विरुध्दानाभुपक्लिन्नपूतीनां भक्षणेन च ।

भुक्त्वा दिवा प्रस्वपतां द्रवास्निग्धगुरुणि च ।।

अत्यादानं तथा क्रोधं भजतां चातपानलौ ।

छर्दिवेगप्रतीघातात् काले चानवसेचनात् ।।

श्रमाभिघातसंतापैरजीर्णाध्यशनैस्तथा ।

शरत्कालस्वभावाच्च शोणितं संप्रदुण्यति ।। (च.सू. २४-५-१०)

1) Excess consumption of alcohol, salt, sour, pungent food
   Kulathha, masha, nishpav, tila taila, mulaka, sour curd

31. Sushrut Samhita, Dr.Ghanekar B., Meharchand Publications, New Delhi, 13th Ed. Sharirsthan, 9/16, Pg. No. 241
32. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Sutrasthan, 24/5-10, Pg. No. 124

2) Virudhhahar

3) Atyashan, Adhyashan, Ajeernashan

4) Diwaswap

5) Krodha

6) Aatapasevan and Agnisevana

7) Chardivega pratighata

8) Shrama

9) Abhighata

10) Sharad rutu

Since rakta is a pittasthana, vitiation of pitta may lead to the vitiation of rakta.

Sign and symptoms: Following diseases are caused by vitiation of rakta.[33]

..... वक्ष्यन्ते रक्तदोषजाः ।

कुष्ठवीसर्पपिडका रक्तपित्तमसृग्दरः ।।

गुदमेढ्रास्यपाकश्च प्लीहा गुल्मोडथ विद्रधिः ।

नीलिका कामला प्यग्डः पिप्लवस्तिलकालकाः ।।

दद्रुश्चर्मदलं श्चित्रं पामा कोठास्त्रमण्डलम् ।

रक्तप्रदोषाज्जायन्ते ..... ।। (च.सू. २८/१२)

Kushtha	Visarpa
Raktapitta	Asrgadara
Asyapaka	Pleeha
Kamala	Nilika
Tilakalaka	Dadru
Pama	Asramandala
Kotha	Pidika
Gudapaka	Medhrapaka
Gulma	Vidradhi
Vyanga	Piplava
Charmadala	Shvitra

33. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Sutrasthan, 28/12, Pg. No. 179

Acharya Sushruta added Mashaka, Nyachha, Indralupta, Vatashonita, Arsha and arbuda to this catagory.

Raktakshaya:

Following sign and symptoms can be seen in a patient with decrease of raktadhatu[34]:

शोणितक्षये त्वक्पारुष्यमम्लशीतप्रार्थना सिराशैथिल्यं च । (सु.सू. १५/१३)

1. Roughness, cracks, dryness and loss of luster of skin
2. Desire for amla (Sour) and sheeta (Cold in potency) substances
3. Decay and degeneration of sira

Raktavruddhi:

Following signs and symptoms can be seen in increase of raktadhatu[35]:

रक्तं रक्ताङ्गाक्षितां सिरापूर्णत्वं च । (सु.सू. १५/१८)

1. Raktaanga-akshita (redness of body and eyes)
2. Sirapurnatva (fullness of sira)

RaktavahasrotasViddha Lakshane (Injury to Raktavahasrotas):

An injury to raktavahasrotas produces the symptoms like shyavangata (paleness of body) , jwara (fever), daha(burning sensation) panduta (anaemia), shonitagamana (bleeding) and raktanetrata (redness of eyes)[36]

तत्र विध्दस्य श्यावाग्डता ज्वरो दाहः पाण्डुता शोणितागमनं रक्तनेत्रता च । (सु.सू. ९/१६)

34. Sushrut Samhita (2005), Dr.Ambita Datta Shastri, Chaukhamba Sanskrit Samsthan, New Delhi, 1st Part, Sutrasthan, 15/13 Pg. No. 58
35. Sushrut Samhita (2005), Dr.Ambita Datta Shastri, Chaukhamba Sanskrit Samsthan, New Delhi, 1st Part, Sutrasthan, 15/18 Pg. No. 60
36. Sushrut Samhita (2005), Dr.Ambita Datta Shastri, Chaukhamba Sanskrit Samsthan, New Delhi, 1st Part, Sutrasthan, 9/16, Pg. No. 241

B) YAKRUT VIKARA FROM AYURVEDIC PERSPECTIVE

B-I) KAMALA –

Nirukti :

'kamaan lati iti kamala' i.e. unwillingness or giving upthe urges.

Acharyas explained kamala under panduroga.

Synonyms:

In classics the term kamala has been explained by different names:

- In Atharva veda, kamala was known as haridraka, harima, harita. Vilohitatva.
- Dalhnana, while commenting, Acharya Sushruta's view, stated that kamala, panaki, kumbhakamala, lagharka, alasa, alaskhya, etc., are different stages of panduroga and all these terms are considered as the synonyms of kamala.
- Astangahrudaya has noted lodhara as the synonym of kamala.
- Chakrapani has used the term 'alpapitta kamala' as the synonym of shakhasrita kamala and 'bahupitta kamala' as the synonym of koshthashakhashrita kamala.

Samprapti:

Samprapti is defined as the mechanism of doshas gets vitiated by their nidan & their spread in the body to produce a disease. All the changes occurring in the body from the cause to the manifestation of a disease come under samprapti.

a) Koshthashrit kamala

When patient, suffering from panduroga, takes pittavardhaka substances in excess, too much vitiation of pitta produces kamala roga by causing rakta & mansadhatudushti.[37]

पाण्डुरोगी तु योऽत्यर्थं पित्तलानि निषेवते ।

तस्य पित्तमसृग्मांसं दग्ध्वा रोगाय कल्पते ।। (च.चि. १६/३४)

37. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Chikitsasthan, 16/34, Pg. No. 528

Mainly ushna & tikshna (Pittavardhak guna) produces tikshagni & state of also cause rakta dhatu dushti. Likewise excessive exercise, sex & contact with sunlight also cause vitiation of rakta dhatu. Of these, pitta again causes rakta dhatu dushti & excessive pitta dosha vruddhi.

The excessive pitta vruddhi due to all the above mechanisms produces abnormality in the raktavahastrotas & raktavahastrotasmoola, yakrut in which dosha-dushya sammurchana occurs. Then atipravrutti of pitta occurs through pittavaha strotas into koshtha, resulting in the dark yellow colouration of the purisha.

b) Shakhashrit kamala

तिलपिष्टनिभं यस्तु वर्चः सृजति कामली ।
श्लेष्मणा रुध्दमार्ग तत् पित्तं कफहरैर्जयेत् ।। (च.चि. १७/१२४)

Dietary items having madhura in taste & ruksha (dry), sheeta (cold in potency), guru (heavy to digest),excessive exercise & vegavidharan are the nidan of shakhashrita kamala as described by Acharya Charaka & Vagbhata. These nidan vitiate kapha & vata, which leads to the production of ama in the amashaya by causing manda & vishamagni respectively.[38]

This ama is having guru, sheeta, manda & ruksha guna. Dosha vitiating guna reaches the rasadhatu though rasayani&takes part in the formation of Rasadhatu& excessive kaphavruddhi.

On reaching raktadhatu, it also causes pittavruddhi in Raktadhatu to some extent. These vruddha dosha produce abnormality in the raktavahastrotas & raktavahastrotasmoola, yakrut& take part in the dosha-dushyasammurchhana there.

38. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Chikitsasthan, 17/124, Pg. No. 532

Due to sankochain the normal pittavahastrotas caused by rukshaguna of vitiated vata &manda-guru guna of vitiated kapha results in strotas - sanga in the pittavahastrotas. Therefore pitta cannot reach into the koshtha through its normal pathway & the purisha is not coloured there due to absence of pitta; so patient excretes tilapishtanibhashweta varchas, which is the cardinal sign & differentiating symptom of shakhashrita kamala.

Pitta begins to accumulate in yakrut due to obstruction in pittavaha strotas. From where it sends to the eye, skin, nails & buccal cavity & results in the yellow colouration of the sites just like haridra.

It is clear from the above description that in shakhashrita kamala, one is localized decrease of pitta in the koshtha & other is localized increase of pitta in the shakhas, which is not vruddhi in reality but is only 'vimargagamana' of pitta due to strotosanga.

Roopa (Manifestation of disease)

According to pathogenesis, various types of kamala are described which are mentioned below –

- Koshthashrita kamala,
- Shakhashrita kamala,
- Kumbha kamala,
- Halimaka
- Lagharaka
- Panaki.

Different symptoms of these types are described as follows:

a) Koshthashrita kamala lakshana:

- ❖ Haridra mutrata (urine), netrata (eyes), twak (skin), nakha (nails), mukha (face), shakrut (faeces)
- ❖ Bhekabha (frog skin like appearance)
- ❖ Daha (burning sensation)
- ❖ Avipaka (indigestion)

- ❖ Daurbalya (weakness)
- ❖ Hatendriya (Emaciated)
- ❖ Aruchi (Tastelessness)
- ❖ Sada (malaise)

b) Shakhashrita kamala lakshna:

- ❖ Haridra mutrata (yellow urine), netrata (eyes), tvak (skin)
- ❖ Mala – tila-pishtanibha (clay coloured) or shweta varna (white stool)
- ❖ Atopa, vishtambha (flatulence)
- ❖ Hrudaya gaurava (heaviness in chest)
- ❖ Daurbalya (weakness)
- ❖ Alpagni (decreased digestive power)
- ❖ Parshwa arati (flank pain)
- ❖ Hidhma (hiccough)
- ❖ Shwasa (respiratory trouble)
- ❖ Aruchi (tastelessness)
- ❖ Jvara (fever)

c) **Kumbhakamala:** Kumbhakamala occurs when the svatantra kamala in pitta predominant patient is left untreated. The prognosis is poor and kumbhakamala is comparable to hepatic failure in modern parlance. Lakshana of kumbhakamala are as follows:

- ❖ Kalantara (produced after long standing kamala)
- ❖ Krushna mutra (blackish urine)
- ❖ Krushna shakrut (blackish stool)
- ❖ Bhrsam shonata (profuse oedema)
- ❖ Sarakta mukha (reddish face), sarakta akshi (reddish eyes)
- ❖ Sarakta chhardi (blood vomiting)
- ❖ Sarakta shakrut (blood in stool)
- ❖ Sarakta mutra (haematuria)
- ❖ Daha (burning sensation)
- ❖ Aruchi (tastelessness)
- ❖ Trut (thirst)
- ❖ Tandra (drowsiness)

- ❖ Moha (fainting)
- ❖ Nashtagni (loss of appetite)
- ❖ Nashta sandnya (unconsciousness)

d) **Halimaka:** Halimaka is also known as lodhara or alasa. It occurs due to vata pitta vitiation. It is having following symptoms:

- ❖ Harit varna (Greenish appearance)
- ❖ Shyava varna (Blackish appearance)
- ❖ Pita varna (Yellowish appearance)
- ❖ Bhrama (dizziness)
- ❖ Trushna (thirst)
- ❖ Strishu aharsha (loss of libido)
- ❖ Mrudu jwara (mild fever)
- ❖ Tandra (drowsiness)
- ❖ Balabhransha (weakness)
- ❖ Anala bhransha (decreased appetite)
- ❖ Utsaha kshaya (depression)
- ❖ Angamarda (body ache)
- ❖ Aruchi (tastelessness)

e) **Lagharaka:** It is mentioned by Acharya Sushruta having following symptoms:

- ❖ Jwara (fever)
- ❖ Angamarda (body pain)
- ❖ Sada (tiredness)
- ❖ Bhrama (dizziness)
- ❖ Tandra (drowsiness)
- ❖ Kshaya (emaciation)

f) **Panaki** : It is Characterized by following symptoms.

- ❖ Santapa (fever)
- ❖ Pandu (pallor)
- ❖ Bhinna varchas (loose stools)
- ❖ Bahirantascha Pitata (external and internal yellow discoloration)
- ❖ Netra raga (sub conjuctival haemorrhage)

Treatment of Kamala

1) Koshthashrita kamala Chikitsa[39]

..... कामली तु विरेचनैः । (च.चि. १६/४०)

a) Nidanaparivarjan (To avoid causative factors)
b) Shodhana (Detoxification of the body)
c) Shamana (Pacification of Doshas)

a) **Nidanaparivarjan:**

This is the first line of management of koshthashrita kamala. There should be strict avoidance of pitta vruddhikara diet & regimen.

b) Shodhana:

❖ Purvakarma:

- Snehapana: Doshotkleshana is performed with snehapana. Aggregated vata dosha comes to its normal activity with the use of ghruta. Ghruta is yogavahi so that it carries the properties of other drugs in medicated ghruta. The guna of pitta are ushna, tikshna, drava, amla, sara & katu & therefore it can be alleviated by the use of snigdha, sheeta, mrudu, guru, madhura dravya such as ghruta. In kamala before shodhana, snehana ghrutas are mentioned - Mahatikta ghruta, Panchagavya ghruta, Kalyanaka ghruta, Dadima ghruta
- Swedana is contraindicated as kamala is a pitta dominant disease & dosha are already in liquid form.

❖ **Pradhankarma (Virechana)**:

- Virechana is the best shodhana for elimination of pitta & is subsequently kaphahara also. In koshthashrita kamala mruduvirechana also mentioned but if the rogi is krura koshtha and rogibala is enough then tikshna virechana can be given. Mrudu virechana are adviced in kamala with Tikta Dravya. Eg. Following drugs are described by Aharya Charakafor the virechana in kamala; Aaragwadh, Danti, Trivrut etc.

39. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Chikitsasthan, 16/40, Pg. No. 528

c) **Shamana:** Shamana is pacification of the remaining dosha after shodhana & also when the patient is stable & having alpadosha.

2) Shakhashrita kamala Chikitsa[40]

कटुतीक्ष्णोष्णलवर्णभृशाम्लैश्चाप्युपक्रमः।

आपित्तरागाच्छकृतो वायाश्चाप्रशमादभवेत् ।।

स्वस्थानमागते पित्ते पुरीषे पित्तरञ्जिते ।

निवृत्तोपद्रवस्य स्यात् पूर्वः कामलिको विधिः ।। (च.चि. १६/१३१)

In Shakhashrita/Ruddhapatha kamala, passage of pitta is obstructed by kapha. Removal of this obstruction by drugs that are kaphaghna is the first step in the treatment. These drugs are generally having the ushna, tikshna & lekhana properties & amla, katu rasa.

In Shakhashrita kamala, the malarupa pitta is situated in shakhas. So shodhana like virechana cannot expel the vitiated dosa unless they are brought back to the koshtha. Special measures like vruddhi, abhishyanda, paka, strotomukha shodhan &vayu nigraha will bring back dosha to koshtha.

Once the strotorodha is removed & mala becomes peeta varni , then treatment line of koshthashrita kamala can be followed.

B-II) YAKRUT ARBUDA :

There is no separate description of tumors of the liver either malignant or benign, in any of the Ayurvedic classics.

In samhitas there is description of conditions like arbuda, granthi, vidradhi, gulma, etc. But it is possible to understand the malignant or benign conditions of the liver with the help of the pathogenesis described.

40. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Chikitsasthan, 16/131, Pg. No. 532

Arbuda has been described by Susruta as a rounded slow growing, fixed, slightly painfull, large, deep seated, non-suppurating, fleshy mass any where in the body.

It occurs due to derangement of mamsadhatu by vitiated dosas. Vagbhata has not given separate definition for arbuda but mentioned that the large granthi can be called as arbuda.

Etiopathogenesis:

Due to mithyahara - vihara, all the three dosha get vitiated. They influence different dhatu i.e. mamasa, meda, rakta, etc. at different sites and gives rise to localized swelling due to kha-vaigunya. Due to predominance of medodhatu and kaphdosha, the arbuda usually does not undergo suppuration.

The etiopathogenesis of mamsarbuda and medorbuda has been mentioned separately. When vitiated dosha affect the rakta resulting into constriction of sira, it may precipitate suppuration or may give rise to rapidly growing projecting fleshy growth called raktarbuda. Whereas regarding mamsarbuda, Susruta has mentioned trauma as a causative factor thay may lead to the origin of mamsarbuda. He also added that habit of excessive consumption of meat can cause Mansarbud.

When this pathogenesis occurs at the site of liver it is yakrutarbuda. Hence we can divide it in 3 types as vatarbuda, pittarbuda, kapharbuda, raktarbuda in liver.

Management

We can treat Yakrutarbuda on the basis of general treatment principles of gulma, arbuda and kamala.

B-III) YAKRUT VIDRADHI

Definition –

Vidradhi is defined as aggravated dosa located in bone, after vitiating rasa, rakta, mamsa and medas gradually produce sever inflammation swelling, deep rooted, painful, round or flat. The vidradhi occurs in the kostha then it is called as Abbhyantra vidradhi.[41]

महामूलं रुजावन्तं वृत्तं चाप्यथवायतम् ।

तमाहुर्विद्रधिं धीरा विज्ञेयः स च षड्विधः ।। (च.नि. ९/५)

Antar Vidradhi:

गुर्वसात्म्यविरुध्दान्नशुष्कसंसृष्टभोजनात् ।

अतिव्यवायव्यायामवेगाधातविदाहिभिः ।।

पृथक् सम्भूय वा दोषाः कुपिता गुल्मरुपिणम् ।

वल्मीकवत्समुन्नद्धमन्तः कुर्वन्ति विद्रधिम् ।। (च.नि. ९-१६-१७)

By taking heavy, unhabitual, wrong combinations of food, dry food.Excessive sex, physical exercise, suppression of natural urges, all the three dosa get aggravated separately or collectively cause abhyatara vidradhi.[42] It is shaped like gulma. Liver is one of the ten places which are described in texts where these aggravated dosha can get lodged to produce the internal abscess.[43]

गुदे बस्तिमुखे नाभ्यां कुक्षौ वड्क्षणयोस्तया ।

वृक्कयोर्यकृति प्लीहिन हृदये क्लोग्नि वा तथा ।। (सु.नि. ९/१८)

41. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Nidansthan, 9/5, Pg. No. 263
42. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Nidansthan, 9/16-17, Pg. No. 264
43. Sushrut Samhita (2005), Dr.Ambita Datta Shastri, Chaukhamba Sanskrit Samsthan, New Delhi, 1st Part, Nidansthan, 9/18, Pg. No. 264

Clinical features

The clinical features of abhyantara vidradhi are said to be same as that of bahya vidradhi.

1. Vataj

   Too rough, highly painful and varied in ways of origin and suppuration discharge if present in thin.

2. Pittaja

   Like the ripen udumbara fruit or blackish, fever, burning sensation, arise and suppurate rapidly discharge if present is yellow in colour.

3. Kaphaja

   Like earthen saucer, pale, cold, stiff with mild pain, itching and arising and suppurating slowly; discharge if present is white in color.

4. Sannipatik

   Pain, discharge of various colors, raised rim, severe, large and suppurates irregularly.

5. Vidradhi due to external trauma or due to rakta vitiation has symptoms similar pittaja type.

Specifically all the abscesses occurring in liver area have a pathognomonic feature of dyspnoea and thirst.

Treatment

Treatment of the abscess depnds on whether it is Ripe (Pakva) or unripe (Apakva).

1. If it is **Apakva (Unripe)**, treatment line will be similar to Shopha (Swelligng). It includes following measures,

- Apatarpana
- Abhyanga
- Upanaha
- Snehana
- Alepa
- Swedana
- Pacana
- Vamana

- Pariseka
- Vimlapana
- Visravana
- Virechana

The above measures can be used as per thr condition of the patient and drugs can also be used according to the dosa involved.

- Madhusigru added with drugs according to dosa and used in foods, driks and paste cures unripe abscess or it may be used with water, sour, gruel and urine.
- Silajatu may be taken with decoction of above groups of drugs according to dosa.
- Superior type of guggulu, sunthi and devadaru can be used.
- Unctuous poitice and carminative drugs can be used.
- Siravedha in Right arm (cubital vei) of Lt side between axial and breast.

2. If it is **Pakva**(ripen), it should be incised and treated like wound.

Pre operative measures – following drugs can be given I a patient with abhyantara yakrt vidradhi

- Kashaya – decoction of varunadi gana drugs added with Ushakadi gana drugs.
- Ghrut – ghee cooked with above drugs and also with purgative drugs alleviated abscess eary in the morning.
- Niruha and anuvasana –Enema should be prepared with above mentoned three groups – varunadi, usakadi and virecana gana mixed with sneha.

Surgical – if the abscess is riped then excision (bhedhan) should be done.

Post operative measures.

- After excision patient should take varunadi drugs or madhu shigru along with maireya, sour gruel, sura and asava.
- Patient should eat rice cooked with decoction of shigru root, mixed with yellow mustard along with soups of yava, kola and kulttha.
- One should use tilvakaghrta or trivrtadi grut daily in the morning.

B-IV) YAKRUTODARA

Udara roga is named on the part of the body Udar itself. Udar vyadhi is one of the ashtaumahagada. Hence is difficult to treat than other systemic diseases.Acharya Chakrapani has mentioned that Udara disease is an display of Shotha, hence, it is seen that After the Udarchikitsa adhyaya falls after Shotha chikitsa in Charak Samhita.
This disease occursmainly due to Mandagni.

Udar roga is a term used to denote enlargement of abdomen. There are 8 types of Udara described in texts:-

1. Vatodara
2. Pittodara
3. Kaphodara
4. Sannipatodara
5. Plihodara
6. Bhaddodara
7. Kshatodara
8. Udakodara.

Out of these 8 types, Plihodara is also known as Yakrutdalyoladara when along with spleen hepatic pathology is seen as the primary cause (Hepatospleenomegaly).
The causative factors of Plihodar/ Yakrutodar :

1) Travelling immediately after having excess food
2) Excessive indulgence in sex
3) Excessive use of Panchakarma procedures
4) Excessive exercise
5) Vitiation of Rakta Dosha

Etiology –

Due to the above mentioned causes displacement of Plisha/Yakrut occures leading to vitiation of Agni and it's Adhishthana causing Plihodar/ Yakrutodar.

Symptoms –

1) Agnimandya (Loss of appetite), Aruchi (loss of taste), Avipak (Indigestion), (Aadhman)flatulence

2) Trushna (Excessive thirst)
3) Shwas (Dyspnea)
4) Jwara (Fever)
5) Panduta (Anaemia)
6) Murchha (Vertigo), Bhrama (Dizziness)
7) Chardi (Vommitting)
8) Daha (Burning Sensation)
9) Vata – Mutra –Purisha sanga (Difficulty in defecation or urination)
10) Tammapravesh(Syncope)

Yakrutodara is more common than Plihodara and mostly cause due to excess alcohol consumption.

B-V) YAKRUT DALYODARA

This type of Udara vyadhi involves pathology of liver & spleen. Symptoms of Yakrutdalyodara are as follows:-

- Daurbalya (Weakness)
- Arochaka (Loss of Appetite)
- Avipak (Indigestion)
- Pipasa (Thirst)
- Angamarda (Bodyache)
- Kasa (Cough)
- Mrudu jwara (Low grade fever)
- Karshya (Emaciation)
- Anaha (Distension of bowel)
- Asyavairasya (Tastelessness in the mouth)
- Tamahpravesha (darkness in front of eyes)
- Parvabheda (pain in joints)
- Shwasa (Breathlessness)

SIGNS :-

- Charak mentions appearing of nila (blue), harita (greenish), haridra (deep yellow) coloured lines on the abdomen as the sign of this disease.
- Colour of abdomen may changes to arunavarna
- Pallor

TYPES :-

There are three types of Yakrutdalyodara mentioned as follows :-

- Vataja Yakrutdalyodara – Udavarta, Ruja (dull pain) and anaha
- Pittaj Yakrutdalyodara – Jvara, moha, thirst and burning sensation.
- Kaphaja Yakrutdalyodara – Gaurav, aruchi, hardness of liver.

CHIKITSA :-

- Raktamokshana – Siravedha
- Swedan – Fomentation should be done with cow's urine in this disease.
- After removal of Doshas by Raktamokshana, Shaman chikitsa should be started.
- Takra preparation – Takra is advised with madhu, taila, vaca, sunthi, satahva, kushta and saindhav.

B-VI) YAKRUTVRUDDHI

Yakrutvriddhi or enlargement of liver.

❖ Symptoms :

- Pain in hypochondrium, right shoulder & right leg, heaviness in right hand, discolouration of stool,cough, fever, bitter taste in mouth, restlessness, haematuria, weakness, constipation & yellow colouration of eyes.
- Patient feels pain of varying nature like toda, bheda& feels better on lying on left side. Insomnia, burning sensation & thirst.
- Edema may develop as the consequence & it may lead to abscess formation, which is thought to be of poor prognosis with high mortality.

It seems that the enlargement of spleen or liver which do not cause the enlargement of the abdomen may be known as plihavriddhi & yakrutvriddhi.

If the enlargement of spleen or liver causes enlargement of abdomen, then those clinical conditions are known as plihodara & yakrutdalyodara

Nidan

Excessive intake of alcohol, suppression of natural urges, taking excessive ushna, tikshna, guru food, sleeping in day time, night awakening, excessive indulgence in sex, trauma, excessive physical work are considered to be the cause of the yakrutroga.

Rupa

Symptoms of Yakrutvriddhi :

Along with the enlargement of yakrut (liver), the patient with yakrutvriddhi has the symptoms of kapha& pitta dosha. In addition to these, the patient may have :

Mandajwara (mild fever)

Mandagni (diminished digestive power)

Kshinabala (weakness)

Atipandu (extreme anemia)

Chikitsa :

Acharyas have mentioned that etiology, sign, symptoms & treatment of yakrutvriddhi or yakrutdalyodara are same as that have plihavriddhi or plihodara respectively.

C) BHEDANA CHIKITSA IN YAKRUT VIKARA

Virechana Chikitsa in Yakrut Vikara :

Importance: Virechanam Pittaharanam Shreshtham.

Virechana is the main procedure followed in Pitta Vikara.

The important and unavoidable factor in Samprapti of Yakrut vikara is Rakta Dushti as Yakrut is the moolasthana of Raktavaha srotasa. Similarly Sushrut has mentioned that Yakrut and Pleeha are the organs which are originated from Rakta dhatu in embryonic stage**.**

Thus treatment of Yakrut vikara is based on the treatment of vitiated Rakta dhatu as quoted in Vidhishonitiya Adhyaya of Charaka Samhita.[44]

कुर्याच्छोणितरोगेषु रक्तपित्तहरीं क्रियाम्।

विरेकमुपवासं च स्त्रावणं शोणितस्य च।।

The treatment is mentioned as –

1. Raktapittahari chikitsa (Treatment principle) (Treatment pacifying Rakta dhatu and Pitta dosha)
2. Virechana (Purgation)
3. Upawasa (Fasting)
4. Raktamokshana (Bloodletting)

Virechana being a treatment of choice of Raktapradoshaja vikara like Yakrut vikara, we have selected it for our study.

Depending upon mode of action of Virechana, Acharya Charaka and Acharya Sharangdhra described threefold and fourfold varieties of Virechana.

Types of Virechana ;

- Aacharya Charak has described 3 main types of virechana – Sukhavirechan, Mruduvirechan, Tikshnavirechan.

44. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Sutrasthan, 24/18, Pg. No. 125

- Aacharya Sharangadhar has explained 4 main types of Virechana –

1) Anulomana
2) Stramsan
3) Bhedan
4) Rechan

1) Anulomana

The procedure in which medicinedigests &pacifies doshas and malas, seperates them from all attachments and evacuates from the body is known as Aulomana Eg. Haritaki[45]

कृत्वा पाकं मलानां यद्विभत्वा बन्धमधो नयेत् ।
तच्चानुलोमनं ज्ञेयं यथा प्रोक्ता हरीतकी ।। (शा.स. प्रथमखंड) ४/३ / ३५

2) Stramsan

The procedure in which medicine brings out sticky, digested or undigested doshas & malas from koshtha is known as Stamsana[46].

Eg.Aragwadha

पक्तव्यं यदपक्त्वैव श्लिष्टं कोष्ठे मलादिकम् ।
नयत्यधः स्त्रंसनं तद्यथा स्यात्कृतमालकः ।। (शा.स. प्रथमखंड) ४/४ / ३५

3) Bhedana

The procedure in which medicine helps to disintegrates doshas and mala and remove them out of the body is known as Bhedana[47].

Eg. Katuki

मलादिकमबध्दं च बध्दं वा पिण्डितं मलैः
भित्वाधः पातयति तद्भेदनं कटुकी यथा ।। (शा.स. प्रथमखंड) ४/५ / ३५

4) Rechana

The procedure in which medicine converts vitiated doshas and malas in liquid form and brings out of the body is known as Rechana[48].

Eg. Trivrutta

विपक्व यदपक्वं वा मलादि द्रवतां नयेत् ।

रेचयत्यपि तज्ज्ञेयं रेचनं त्रिवृता यथा ।। (शा.स. प्रथमखंड) ४/६ / ३६

Bhedana Karma is the choice of treatment in Yakrut Vikara.It is explained in detail below –

Bhedan Definition

According to Sharagdhar those drugs that disintegrates doshas and mala and remove them out of the body are called Bhedan Dravya.

All fast acting purgatives fall under the bhedan drugs like Kutki

Acharya Sushrut specifically defines Shyamadigana as Bhedan drugs.

Shyamadi Gana (Sushrut Samhita Sutrasthan 38/14)[49]

श्यामामहाशामात्रिवृत्दन्तीशंङ्खिनीतिल्वक-

कम्पिल्लककरम्यकक्रमुकपुत्रश्रेणीगवाक्षीराजवृक्षकरञ्जद्वय

गुडूचीसप्तलाच्छगलान्त्रीसुधाःसुवर्णक्षीरी चेति।।

Nishottar, Mahashyama, Danti, Shankhini, Tilvak, Kabila, Ramyak, Supari, Putrashreni, Gavakshi, Amaltas, Putikaranj, Guduchi, Satla, Vruddhdaruk, Sehund, Suvarnakshiri are the herbs included in Shyamadigana which cures Gulma and Visha (Poison) and also helps in treatment of flatulence & Ascites.

Bhedaniya Gana (Reference : Charak Sutrasthan 4/4)

Suvaha, Arka, Eranda, Agnimukha, Chitra, Chitrak, Chirbilwa, Sankhini, Shakuladini, Swarnakshirini, these ten are mass – breaking drugs[50].

45.- 48. Sharangdhar Samhita (2000), Pt. Parashuram Shastri, Vidyasagar, Chaukhambha Orientalia, 4thEdi., 1st Part, 4/3-7, Pg.No. 35-36

49. Sushrut Samhita (2005), Dr.Ambita Datta Shastri, Chaukhamba Sanskrit Samsthan, New Delhi, 1st Part, Sutrasthan, shyamadi Gana, 38/14, Pg. No. 143

50. Charak Samhita, Chakrapani Datta, Munshiram Manoharlal publication, 4th Edi., Sutrasthan, 4/4

D) DRUG REVIEW

D-1) YAKRUTPLIHARI LOHA VATI

Yakrutplihari Loha Vati (Ref. - BhaishajyaRatnavali 41/162 - 166)[51]

यकृत्प्लीहोदरारिलौहम् -

स्वर्ण रौप्यं तथाताम्रं वङ्गश्चाभ्रं समाक्षिकम् ।

सर्वार्ध्दं जारितंलौहं कल्पयेत्कुशलो भिषक् ।।

श्रृङ्गबेररसेनापि शेफालीदलजैरसैः ।

स्वरसैर्बिल्वपत्राणां क्वाथैश्च कटुतिक्तजैः ।।

रसेन बहुमञ्जर्या भावयेच्च त्रिधात्रिधा ।

वल्लमात्रं प्रदातव्यं पर्पटक्वाथसंयुतम् ।।

प्लीहानं यकृतंश्वासंकासञ्चविषमज्वरम् ।

गुल्मशोथोदरानाहमग्रमांसमरोचकम् ।।

कामलांपाण्डुरोगञ्चिरकालानुबन्धिनम् ।

सर्वान् रोगान्निहन्त्याशु वातपित्तकफोद्भवान् ।। भै.र. ४१/१६२-१६५

Total 5 varieties of Yakrutplihari Loha Vati are described in the text (bhaishajya Ratnavali).

Out of them below mentioned variety is chosen as it contains specially Suvarnabhasma, Raupyabhasma & Suvarnamakshikbhasma which are having Rasayana and Vishaghna action.

51. Bhaishajya Ratnavali, Kaviraj Ambika data Shastri, Chaukhambha Sanskrit Sansthan, Varanasi, 13th Edi, 41/162 – 166, Pg.No. 551

Table No. 1 : Contents Of Yakrutplihariloha vati

	Contents	**Dosage**
1.	SuvarnaBhasma	1 Tola (10 gm.)
2.	RaupyaBhasma	1 Tola (10 gm.)
3.	TamraBhasma	1 Tola (10 gm.)
4.	VangaBhasma	1 Tola (10 gm.)
5.	SuvarnaMakshikBhasma	1 Tola (10 gm.)
6.	AbhrakBhasma	1 Tola (10 gm.)
7.	JaritaLoha	3 Tola (30 gm.)

The mixture of these 7 bhasma is triturated with decoction or fresh juice of following herbs.

Decoction of each herb should be triturated for 3 times.

BhavanaDravya (Herbs used for trituration)

1) Ardraka Swarasa - Fresh juice of Ginger- (Zingiber *officinale*)
2) Shephali - dala – Swarasa
   i) Fresh juice of Nirgundi leaves (Vitex negundo)
   ii) Fresh juice of Parijataka leaves (Nyctanthus *arbortristis*)
3) Bilva - patra – Swarasa -Fresh juice of leaves of Bilva (Aegle *marmalos*)
4) Kirtatikta Kwatha - Decoction of Kiratatikta
5) Tulasi Kwatha - Decoction of Tulasi (Oscimum *sanctum*)

Shephali is the synonym of Nirgundi and Parijataka

Out of them – Nirgundi is described as Pleeha – gami & Parijataka as Yakrut – gami.

As both of these dravya are acting on moolasthana of Raktavahasrotasa, trituration of both of them for 3-3 times was given.

Importance of Trituration (Bhavana)

1. It increase potency of the medicine
2. It directs medicine and helps its rapid uptake towards affected organ.

Dosage :

1 Valli(3 - 3 Ratti)

Duration :

3 months to 9 months.

It is used in following diseases-

- Pleehodara (Spleenormegaly)
- Yakruttodara (Hepatomegaly)
- Shwasa (Breathlessness)
- Kasa (Cough)
- Vishamajwara (Fever)
- Gulma (Tumour)
- Shotha (Swelling)
- Udara (Ascitis)
- Aanaha (Flatulence)
- Agramansa
- Arochaka (Loss of taste)
- Kamala (Jaundice)
- Pandu – chirakalanubandhi (Anemia)

Description & Mechanism of action of Yakrutplihari Loha Vati

Table No. 2 : Description of bhasmas used in Yakrutplihari Loha Vati

Bhasma	**Rasa**	**Veerya**	**Vipaka**	**Guna**	**Karma**
Suvarna	Madhura	Sheeta	Madhura	Snigdha	Pandu, Kasa, Shwasakrucchata, Jwara, Bharakshaya, Atisara
Roupya	Kashaya Amla	Sheeta	Madhura	Snighdha Laghu	Jwara
Tamra	Tikta Kashaya Amla	Ushna	Madhura		Jwara, Kasa, Agnimandya, Udarshula, Jatodara, Yakrutshula

Vanga	Tikta Kashaya Lavana	Ushna	Katu	Ruksha Guru	Kasa, Shwasakrucchhata, Chhardi
SuvarnaMakshik	Madhur Titka Kashaya Amla	Sheeta	Madhura	Laghu	Pandu, Agnimandya
Abhrak	Madhura	Sheeta	Madhura	Snighdha	Pandu, Jwara, Shwasa, Kasa, Udara, Agnimandya, Jatharashula
JaritaLoha	Tikta Kashaya	Sheeta	Madhura	Ruksha Guru	Pandu, Pratishyaya, Kasa, Shwasa, Jwara, Udarashula, Bharakshaya, Chhardi, Atisara, Yakrut Shula

Table No. 3 : The mode of action of bhasmas in Yakrutplihari Loha Vati

Suvarna Abhraka	**Raupya Loha Tamra**	**Suvarna – makshik**	**Tamra**	**Tamra Loha Raupya**	**Raupya**	**Suvarna Raupya Vanga Abhrak Suvarna – makshik**	**Suvarna Tamra Loha Vanga Abhrak Suvarna – makshik**
Increasing rasa dhatvangi	Increasing rakta dhatvagni	Rakta vardhan and giving strength to Raktadharakala	Yakrut gami	Pleehagami	Siragami	Rasayana	Vishaghna

Table No. 4 : Bhavanadravya of Yakrutplihari Loha Vati

Dravya	Rasa	Veerya	Vipaka	Guna	Karma
Aardraka	Katu	Ushna	Madhura	Tikshna, Ruksha,	Chhardi, Kasa, Shwas, Shula, Vibandha, Aanaha, Udara
Nirgundi	Tikta Kashaya Katu	Ushna	Katu	Laghu	Kasa, Shula, Shotha, Aadhmana
Parijatak	Katu Tikta	Ushna	Katu	Laghu	Jwara, Kasa
Bilva	Kashaya, Tikta, Katu	Ushna	Katu	Laghu, Tikshna, Snigdha	Grahi
Kiratatikta	Tikta	Sheeta	Katu	Ruksha Laghu	Jwara, Kasa, Shwas, Shotha
Tulasi	Katu Tikta	Ushna	Katu	Ushna Tikshna	Kasa, Shwasa

Table No. 5 : Role of herbs of Bhavanadravya

Jatharagni deepan	Raktadoshanashak	Yakrut - gami	Pleeha - gami	Bhedana
Ardrak	Kiratatikta	Parijatak	Nirgundi	Aardraka
Bilwa	Tulsi			

A) Mode of action of Yakrutplihari Loha Vati as a whole :

Mostly all the bhasma in this kalpa are sheeta but bhavana dravya are ushana. Due to ushna guna of bhavana dravya, rapid and easy uptake of the medicine towards yakrut & pleeha is possible. Mostly all the bhasma are acting as Prasadana & Rasayana on these organs.

1. Give strength to Raktadharakala
2. Increase Rasa – Raktadhatvagni and improve formation of Rasa - Rakta.
3. Give strength to the organs - Yakrut and Pleeha due to Rasayana karma.
4. Give strength to Sira for improving circulation of Rasa – Raktadhatu.

D-II) KUMARI KALPA

Kumari Kalpa Vati - (Aloe plus tab) - 250mg

(Propritery medicine of Atharva ayurved Pharmacy)

Table No. 6 : Contents of Kumari kalpa vati (Aloe plus tab)

Ingredients	Quantity
Kumari Swaras	250mg
Haritaki	25 mg
Bibhitak	25 mg
Bhumyamalaki	25 mg
Sunth	25 mg
Marich	25 mg
Pippali	25 mg
Vidanga	25 mg
Musta	25 mg
Sharapunkha	25 mg

D-II) Kutaki (Katuka)

Table No. 7 : Description of Kutaki (Katuka)

Dravya	Rasa	Veerya	Vipaka	Prabhav	Guna	Karma
Katuka	Tikta	Sheeta	Katu	--	Ruksha, laghu	Bhedana, deepan, astra – daha – vishamajwaranashan etc

Mode of action :

Kutaki with its Bhedana action eliminates vitiated dosha from yakrut.

D- III) Kumari Kalpa Vati (Aloe Plus tab) :

Table No. 8 : Description of Kumari kalpa (Aloe Plus tab)

Dravya	Rasa	Veerya	Vipaka	Prabhav	Guna	Karma
Kumari Swaras	Tikta	Sheeta	Katu	Bhedan	Snigdha, Pichhila	Tridoshaghna, Rasayan, Balya, Vrushya, Gulmahara, Yakrut – Pliha vrudhhi hara, etc.
Haritaki	Kashaya, katu, tikta, madhur, amla	Ushna	Madhur	Tridoshahara	Laghu, ruksha	Rasayan, Takrut gada hara, kamala – pliha vikarahara, etc.
Bibhitak	Kashaya	Ushna	Madhur	--	Ruksha Laghu	Bhedana, kruminasana, pittaroganahanam etc
Bhumyamalaki	Tikta Kashay Madhur	Sheeta	Madhur	--	Laghu, Ruksha	Pandu – pitta – astra – kushta – vishapaha etc
Sunth	Katu	Ushna	Madhur	Grahi	Snigdha Laghu	Shotha – Udara – pandu – hrudrog nashak etc
Marich	Katu	Ushna	Katu	--	Laghu, tiksha	Chhedan, shoshan, deepan, shwas – kasa-kushta hara etc.
Pippali	Katu	Anushnasheeta	Madhur	--	Laghu, snighdha, tikshna	Pleehagulmaghna, vrushya, rasayana, shwas – kasa – jwarahara etc

Dravya	Rasa	Veerya	Vipaka	Prabhav	Guna	Karma
Vidanga	Katu	Ushna	Katu	--	Laghu, tiksha	Shoola – aadhman – krumi – kushtha nashak etc
Musta	Katu, tikta, kashaya	Sheeta	Katu	--	Laghu, Ruksha	Pitta – astra – trushna – jwaranashak, Atisar – krumighna etc.
Sharapunkha	Tikta, kashay	Ushna	Katu	Bhedan	Laghu	Yakrut – pliha – gulma – krumi nashak

Mode of action of Kumari Kalpa as a whole –

1) Kumari kalpa dravyas act mainly as Yakrut pliha vrudhhinashak by reducing the inflammation of liver.
2) Also the combination enhances the functions of Yakrut by pitta – rakta dosha shaman and producing Shudhha Rakta.

E) LITRETURE REVIEW

LIVER ANATOMY& PHYSIOLOGY – FROM MODERN MEDICAL SCIENCE[52]

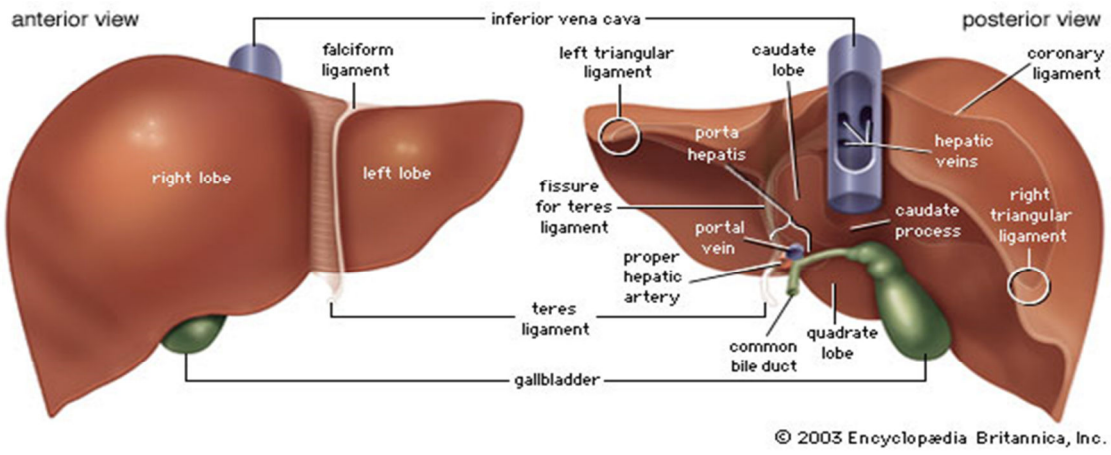
The liver is the largest and heaviest gland in the body. The liver is also called the "hepar". The adjective "hepatic" from this word hepar is applied to many structures related with this organ.

In the living subject the liver is reddish brown in colour, soft in consistency and very friable. It weighs about 1500 g in males and about 1300 g in females.

Position

The liver is situated in the right upper quadrant of the abdominal cavity. It occupies the whole of the right hypochondrium, the greater part of the epigastrium and extends into the left hypochondrium reaching up to the left lateral line. From the above it is sheltered by the ribs and costal cartilages, except in the upper part of the epigastrium where it is in contact with the anterior abdominal wall.

Structure



The liver is wedge shaped. It resembles a four-sided pyramid lay on one side. It has five surfaces. There are: i. Anterior, ii. Posterior, iii. Superior, iv. Inferior and v. Right.

The inferior surface is demarcated anteriorly by a sharp inferior border.

The sharp anterior part is marked by the notch for the ligamentumteres&cystic notch for the fundus of the gall bladder.

Liver lobes

The liver is divided into two lobes - right and left lobe by the attachment of the falciform ligament anteriorly and superiorly, by the fissure for the ligamentumteres inferiorly, and by the fissure for the ligamentumvenosum posteriorly.

a) The Right Lobe

It is much larger i.e. six times larger than the left lobe.Lesser segments of right lobe are the caudate and quadrate lobe. The caudate lobe is situated on the posterior surface. It is connected to the right lobe of the liver by the caudate process. Below and to the left it present a small rounded elevation called the papillary process. The quadrate lobe is situated on the inferior surface and is rectangular in shape.

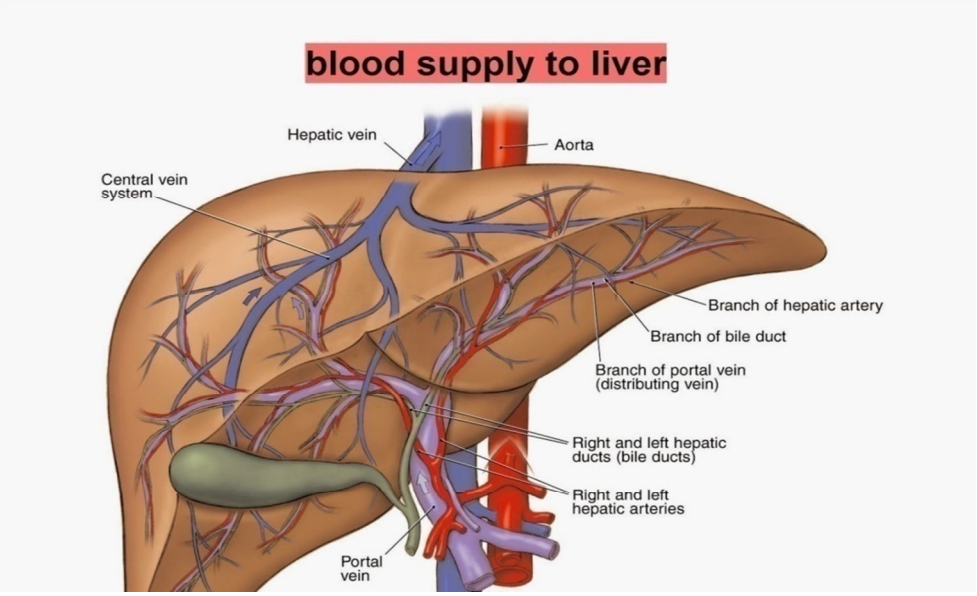
It is bounded anteriorly by the inferior border, posteriorly by the portahepatis, on the right by the fossa for the gall bladder & on the left by the fissure for the ligamentumteres.

The portahepatis is a deep, transverse fissure about 5cm long, situated on the inferior surface of the right lobe of the liver. It lies between the caudate lobe above and the quadrate lobe below and in front. The portal vein, the hepatic artery and the hepatic plexus of nerves enter the liver through the portahepatis, while the right and left hepatic ducts and a few lymphatics leave it.

b) The Left Lobe

The left lobe of the liver is much smaller than the right lobe and forms only one sixth of the liver. Near the fissure for the ligamentumvenosum, its inferior surface presents a rounded elevation, called the omental tuberosity (tuber omentale).

Blood Supply



The liver has a duel blood supply. The portal vein supllies venous blood from intestine and spleen while hepatic artery supplies arterial blood.These vessels enter the liver through a fissure called portahepatis. Within the liver, they are dividing to form segmental vessels, which further divide to form interlobular vessels. Further ramifications of the interlobular branches open into the hepatic sinusoids. Hence the hepatic arterial blood mixes with the portal venous blood in the sinusoids.

Venous drainage

Hepatic sinusoids drain into interlobular veins, which join to form sub lobular veins. These in turn unite to form the hepatic veins, which drain directly into the inferior vena cava.The hepatic veins are arranged in two groups- upper & lower.

The upper group consists of three large veins - right,left&middle.The lower group consists of a variable number of small veins from the right lobe & the caudate lobe.

Lymphatic drainage

The superficial lymphatics of the liver run on the surface of the organ beneath the peritoneum, and terminate in caval, hepatic, paracardial and coeliac lymph nodes. Some vessels from the coronary ligament may directly join the thoracic duct. The

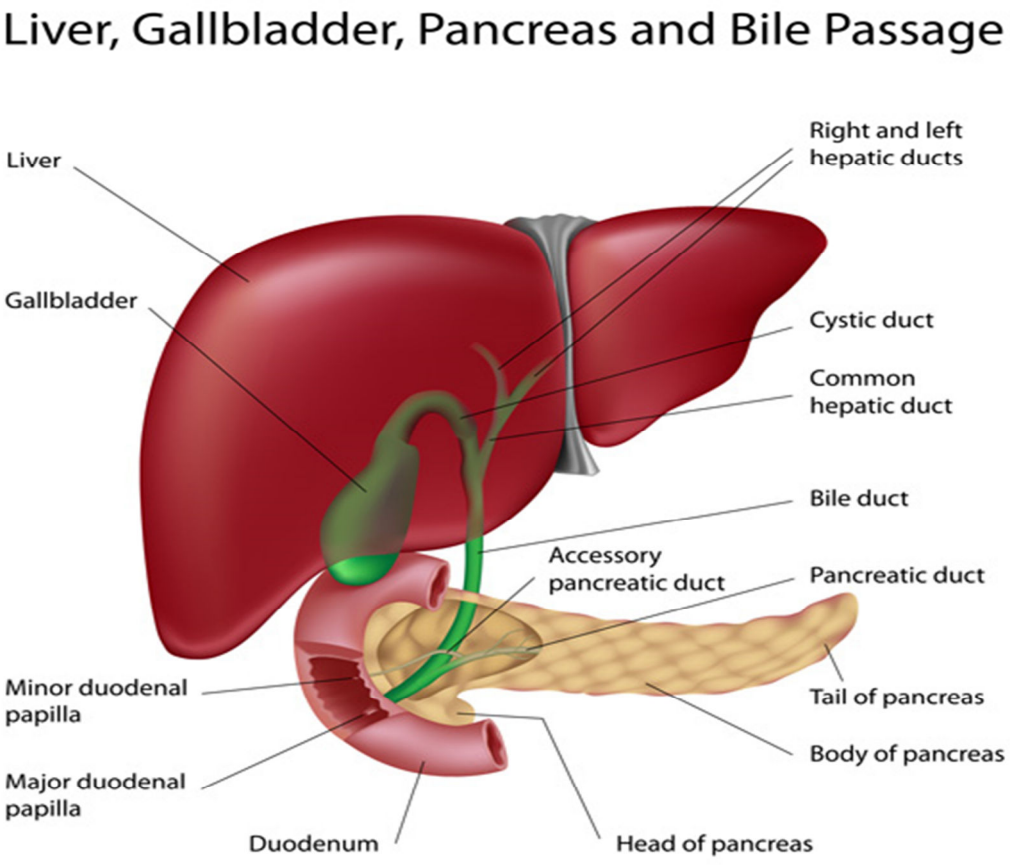
deep lymphatics end partly in the nodes around the end of the inferior vena cava and partly in the hepatic nodes.

Nerve supply

The liver receives its nerve supply from the hepatic plexus. It contains sympathetic and parasympathetic fibers. Nerves also reach the liver through its various peritoneal ligaments.

Extra Hepatic Biliary Apparatus

The biliary apparatus collects bile from the liver, stores it in the gall bladder and transmits it to the second part of the duodenum.



The apparatus consists of -

i) The right and left hepatic ducts

ii) The common hepatic duct

iii) The gall bladder

iv) The cystic duct

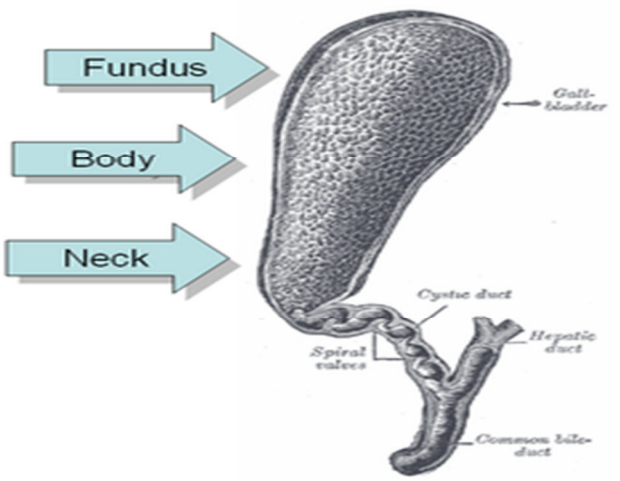
v) The bile duct

i) **Hepatic ducts:** The right and left hepatic ducts emerge at the portahepatis from the right and left lobes of the liver.

ii) **Common hepatic duct:** It is formed by the union of right and left hepatic ducts near the right end of the portahepatis. It runs downwards and is joined by the cystic duct to the form the bile duct.

Accessory hepatic ducts are present in about 15% of subjects. They usually issue from the right lobe of the liver, and terminate either in the gall bladder or in the common hepatic duct anywhere in its course, or even in the upper part of the bile ducts.

iii) **Gall bladder:**



This is a pear-shaped reservoir of bile situated in a fossa on the inferior surface of the right lobe of the liver. The gall bladder is 7 to 10 cm long, 3 cm broad at its widest part, and about to 30 to 50 ml in capacity.

The gall bladder is divided into i. the fundus; ii. body and iii. neck.The fundus projects beyond the inferior border of the liver, in the angle between the lateral border of the right rectus abdominis and the 9^{th} costal cartilage. The body lies in the fossa for the gall bladder on the liver. The upper narrow end of the body is continuous with the neck at the right end of the portahepatis. The neck is the narrow upper end of the gall bladder. It is situated near the right end of the portahepatis. It becomes continuous with the cystic duct. Its junction with the cystic duct is marked by a constriction. The mucous membrane of the neck is folded spirally to prevent any obstruction to the inflow or outflow of bile.

The postero-medial wall of the neck is dilated outwards to form a pouch (Hartmann's pouch). Some regard this pouch as a normal feature of the gall bladder, but others consider it to be pathological. Gallstones may lodge in this pouch.

Liver function

Liver is one of the vital organs of the body. It performs many metabolic and haemostatic functions, which are summarized below:

1. Metabolic function: Liver is the organ where maximum metabolic (actions are carried out. It plays an important role in energy metabolism of carbohydrates, proteins, lipids and vitamins; and many hormones are carried out in liver,
   - **i.** Carbohydrate metabolism: When blood glucose is high, as occurs just after eating a meal the liver converts glucose to glycogen and glycerides for storage. The liver is especially important in maintaining a normal blood glucose level. When blood glucose is low, the liver can break down glycogen to glucose and release glucose into the blood stream. The livercan also convert certain amino acids and lactic acid to glucose and it converts other sugars such as fructose and galactose into glucose.
   - **ii.** Lipid metabolism: Hepatocyte stores some triglycerides, break down fatty acids to generate ATP, synthesize lipoproteins, which transport fatty acids, triglycerides and cholesterol to and from body cells. Synthesizes cholesterol and use cholesterol to make bile salts.
   - iii. Protein metabolism: Hepatocyte deaminate (remove amino group from amino acids) so that amino acids can be used for ATP production or converted to carbohydrates or fats. The resulting toxic ammonia is then converted into the less toxic urea which is excreted through urine. Hepatocytes also synthesize most plasma proteins such as alpha and beta globulins, albumin, prothrombin and fibrinogen.
2. **Storage function:** In addition to glycogen, the liver is prime storage site for certain vitamins (A, $B_{12,}$ D, E, and K) and minerals (iron and copper), amino acids that are released from the liver when needed elsewhere in the body.
3. **Secretion of Bile:** Liver secretes bile, which contains bile salts, bile pigments, cholesterol, fatty acids and lecithin. The function of bile is mainly due to bile salts. The bile salts are required for digestion and absorption of fats in the intestine. Bile helps to carry away waste products and breakdown fats, which are

excreted through faeces or urine.

4. **Synthetic function:** Liver produces glucose by gluconeogenesis. It synthesizes all the plasma proteins and other protein such as clotting factors and complement factors, steroids and hormone binding proteins, somatomedins and heparin.
5. **Excretory function:** Liver excretes cholesterol, bile pigments, heavy metals (Pb, As, Bi, etc.) toxins, bacteria like typhoid and viruses (Hay fever).
6. **Heat production:** Due to metabolic function maximum heat is produced in the liver.
7. **Haemopoietic function:** In fetus (hepatic stage), the blood cells are produced in liver. It stores Vitamin B12, necessary for erythropoiesis and iron, necessary for synthesis of haemoglobin in red blood cells. Liver produces thrombopoietin that promotes production of thrombocytes.
8. **Haemolyticfunction:**Reticuloendothelial cells (Kuffer's cells) of the liver destroy the senile red blood cells after the life span of 120 days.
9. **Inactivation of Hormones & Drugs:** Liver catabolizes hormones such as growth hormone, parathromone, cortisol, insulin, glycogen and estrogen. It also inactivates the drug particularly the fat-soluble drugs. The fat-soluble drugs are converted into water-soluble substances, which are excreted through bile or urine.
10. **Defensive function:** The reticuloendothelial cells (Kuffer's cells) of the liver play important role in the defense of the body. The foreign bodies like bateria or antigens are swallowed and digested by reticuloendothelial cells of liver by means of phagocytosis. The reticuloendothelial cells are also involved in production of some substances like interleukins and tumor necrosis factor, which activate the immune system of the body.
11. **Detoxification function:** Liver is involved in the removal of toxic property of harmful agents. The detoxification in liver occurs in two ways:
    i. By total destruction of the substances by means of metabolic degradation
    ii. By converting toxic substances into nontoxic materials by means of conjugation with glucuronic acid or sulfates.

Normal bilirubin metabolism

Normal metabolism of bilirubin can be conveniently describedunder four main headings viz., source, transport, hepatic phase and intestinal phase.

1. **Source of bilirubin:** About 80-85% of the bilirubin is derived from the catabolism of haemoglobin present in senescent red blood cells. The destruction of the erythrocytes at the end of their normal life span of 120 days takes place in the reticuloendothelial system in the bone marrow, spleen and liver. The remaining 15-20% of the bilirubin comes partly from non-haemoglobinhaem-containing pigments such as myoglobin, catalase and cytochromes, and partly from ineffective erythropoiesis.
2. **Transport of bilirubin:** Bilirubin on release from macro-phages circulates as unconjugated bilirubin in plasma tightly bound to albumin. Certain drugs such as sulfonamides and salicylates compete with bilirubin for albumin binding and displace bilirubin from albumin, thus facilitating bilirubin to enter into the brain in neonates and increase the risk of kemicterus. Bilirubin is found in body fluids in proportion to their albumih content such as in CSF, joint effusions, cysts, etc.
3. **Hepatic phase:**On coming in contact with the hepatocyte surface, unconjugated bilirubin is metabolized which involves three steps: hepatic uptake, conjugation and secretion in bile.
    i. Hepatic uptake: Albumin-bound unconjugated bilirubin upon entry into the hepatocyte is dissociated into bilirubin and albumin. The bilirubin gets bound to cytoplasmic protein glutathione-S-transferase (GST).
    ii. Conjugation: Unconjugated bilirubin is not water-soluble but is alcohol-soluble and is converted into water-soluble compound by conjugation. Conjugation occurs in endoplasmic reticulum and involves conversion of unconjugated bilirubin by the action of microsomal enzyme, bilirubin-UDP-glucuronosyltransferase, to mono-and diglucuronides.

    Conjugated bilirubin is bound to albumin in two forms: reversible and irreversible. Reversible binding is similar to that of unconjugated bilirubin. However, when present in serum for a long time (e.g. in cholestasis, long-standing biliary obstruction, chronic active hepatitis), conjugated bilirubin is bound to albumin irreversibly and is termed delta bilirubin or biliprotein. This irreversible conjugated delta bilirubin is not excreted by the kidney

and remains detectable in serum for sufficient time after recovery from the diseases.

iii. Secretion into bile: Conjugated (water soluble) bilirubin is rapidly transported directly into bile.

4. **Intestinal phase:** Appearance of conjugated bilirubin in the intestine is followed by either direct excretion in the stool as stercobilinogen, which imparts the normal yellow colour to the stool or may be metabolized to urobilinogen by the action of intestinal bacteria.

Conjugated bilirubin is normally not reabsorbable. Whereas it is metabolic product, urobilinogen is reabsorbed from the small intestine and reaches enterohepatic circulation. Some of the absorbed urobilinogen is resecreted by the liver into the bile while the rest is excreted in the urine as urobilinogen.

52. Human Anatomy, Vol. 2, Dr.B.D.Chaurasia,4th Edi., CBS Publishers, New Delhi, Pg.No.- 288 to 293.
Davidson's Principle And Practice Of Medicine, Dr.Nicki R. Colledge, Dr.Brian R. Walker, Dr.Stuart H. Ralston, 21st Edi, Elsevier's Publications,Pg.No. - 919

E) LIVER DISEASES FROM MODERN MEDICAL SCIENCE

E- I) Liver Cirrhosis[53]

Cirrhosis is diffuse process with fibrosis and nodule formation. It is a slowly progressing disease in which healthy liver tissue is replaced with scare tissue, hence preventing the liver from working properly.

The scar tissue blocks the flow of the blood through hormones, drugs and naturally produced toxins. Also the production of proteins and other substances made by the liver is hampered.

According to NIH it is the 12th leading cause of death.

Causes of liver cirhhosis:

- Alcohol
- NASH (Non-Alcoholic Steato Hepatitis)
- Chronic viral infection of liver (Hepatitis B, C & D)
- Biliary obstruction
- Haemochromatosis
- Wilson's Diseases
- Alpha-1-antitrypsin deficiency
- Cystic fibrosis
- Glycogen storage disease
- Galectosemia
- Venous outflow obstruction
- Autoimmune hepatitis

Pathogenesis

Cirrhosis in general is initiated by hepatocellular necrosis. Destruction of hepatocytes causes collapse of normal lobular hepatic parenchyma which is followed by fibrosis around necrotic liver cells and there is formation of compensatory regenerative nodules.

Decompensation:

Desompensation is the cirrhosis which is complicated by one or more of the followingdiseases : Jaundice, Ascitis, Hepatic encephalopathy, bleeding varices.
Usually ascitis is the first sign.

Survival for these patients is 1 ½ year, they can be considered for liver transplantation.

Symptoms:

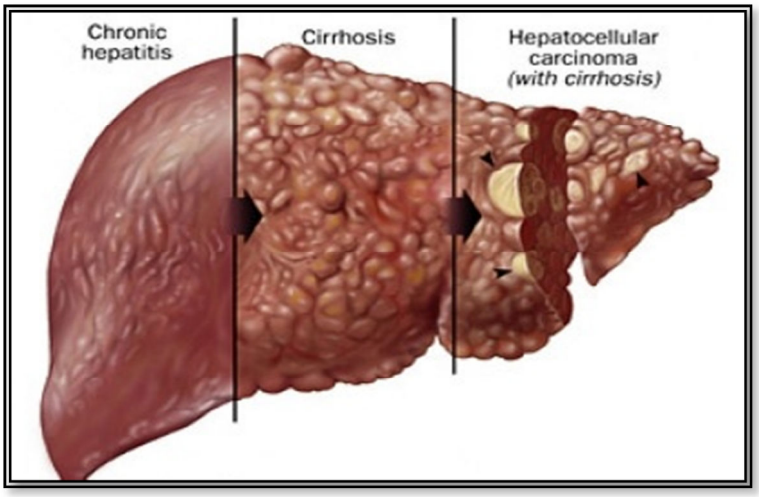
Weakness, muscle wasting, wt. loss, clubbing of nails, fever, skin pigmentation,Perpura over arms, shoulders, vascular spiders, palmer erythema, white nails and gonadal atrophy.

Compensated:

In this condition decompensation may be precipited by bacterial infection, surgery, trauma or medication.
Survival for these patients is 10 years

Symptoms:

Hepatomegaly, splenomegaly, portal hypertension.



Clinical features:

1. Loss of appetite
2. Fatigue
3. Weight loss / sudden weight gain
4. Bruises
5. Icterus
6. Itching
7. Oebema
8. Malena
9. Fever

Diagnosis:

1. USG Abdomen
2. CT Scan Abdomen(Multiphase CT)
3. MRI Scan Abdomen
4. Fibroscan – Transient Elastography Non Invasive method of evaluating.
5. Liver Biopsy or HPR

Treatment of cirrhosis

1) General

- Complete rest
- Maintainance of adequate and balanced diet.
- Withdrawal of alcohol
- Avoidance of obesity
- Early detection of HCC
- Use of diuretics
- Maintenance of renal functions
- Prevention of varicealhaemorrhage.
- Artificial liver support.
- Hepatocytes transplant &orthorptic liver transplant

2) Specific

- Viral hepatitis (B,C &D) –Antiviral treatment
- NASH – Weight Loss
- Metabolic – Haemochromatosis – Venesection
- Wilson's Disease – Copper Chelator
- X-Antitrypsin det. – Transplant
- Type IV Glucogenesis – Transplant
- Galectosemia – Widraw milk and milk products
- Autoimmune – Immunospuppression.

53. Sherlock's Diseases Of The Liver And Biliary System, Dr.Dooley, Dr.Lok, Dr.Burroughs, Dr.Heathcote, 12th Edi, Wiley – Blackwell Publications, Pg.No. 103 - 117

E – II) VIRAL HEPATITIS [54]

E – II a) HEPATITIS A

Hepatitis A is a liver disease caused by the hepatitis A virus which is a RNA virus.

Transmission

- Food or water contaminated with the faeces of an infected person.
- Inadequate sanitation and poor personal hygiene.Most infection occurs in childhood.

  Hepatitis A infection does not cause chronic liver disease and is rarely fatal. But it can cause debilitating symptoms and acute liver failure which is often fatal.

Symptoms

- The incubation period of hepatitis A is usually 14–28 days.
- Fever, malaise, loss of appetite, diarrhoea, nausea, abdominal discomfort, jaundice

Diagnosis

- Specific diagnosis is made by the detection of HAV-specific Immunoglobulin G (IgM) antibodies in the blood.
- Additional tests include reverse transcriptase polymerase chain reaction (RT-PCR) to detect the hepatitis A virus RNA, and may require specialised laboratory facilities.

Treatment

- There is no specific treatment for hepatitis A. Recovery from symptoms following infection may be slow and may take several weeks or months.
- Most important is the avoidance of unnecessary medications.
- Acetaminophen / Paracetamol and medication against vomiting should not be given.

Prevention

- Good sanitation practice
- Immunization

E – II b) Hepatitis B

Hepatitis B is a life-threatening chronic liver infection caused by the hepatitis B virus which leads to Cirrhosis and further complications.

Transmission of hepatitis B:

The hepatitis B virus can survive outside the body for at least 7 days. During this time, the virus can still cause infection if it enters the body of a person who is not protected by the vaccine.

Incubation period - 75 days

The virus may be detected within 30 to 60 days after infection and can persist and develop into chronic hepatitis B.

Mode of Transmission –

- Perinatal transmission
- Intra – venous route due to uasage of infected needles & syringes
- Infected sexual partners
- Health workers
- Haemphiliacs and their carers due to exposure to blood products
- Haemodialysis
- Heredetory

Symptoms

- Mostly asymptomatic during the acute infection phase.
- Acute illness
- Jaundice
- Fatigue
- Nausea & vomiting
- Abdominal pain.
- Hepatomegaly, splenomegaly and adenopathy.
- Hepatitis B usually causes chronic liver infection that can later develop into cirrhosis of the liver or liver cancer.

Children less than 6 years of age who become infected with the hepatitis B virus are the most likely to develop chronic infections.

In adults hepatitis B is a life-threatening liver infection. It is a major global health problem. It can cause chronic infection and cause high risk of death from cirrhosis and liver cancer.

A vaccine against hepatitis B has been available since 1982. The vaccine is 95% effective in preventing infection and the development of chronic disease and liver cancer only due to hepatitis B.

Diagnosis

- Laboratory diagnosis of hepatitis B infection focuses on the detection of the hepatitis B surface antigen HBsAg.
- WHO recommends that all blood donations be tested for hepatitis B to ensure blood safety and avoid accidental transmission to people who receive blood products.
- Acute HBV infection is characterized by the presence of HBsAg and immunoglobulin M (IgM) antibody to the core antigen, HBcAg.
  During the initial phase of infection, patients are also seropositive for hepatitis B e antigen (HBeAg). HBeAg is usually a marker of high levels of replication of the virus. The presence of HBeAg indicates that the blood and body fluids of the infected individual are highly contagious.
- Chronic infection is characterized by the persistence of HBsAg for at least 6 months. This increases the risk for developing chronic liver disease and liver cancer.

Treatment

- No specific treatment for acute signs & symptoms of Hepatits B.
- Nutitional balance, fluid replacement in case of acute dehydration.
- Chronic HBV may respond to PG interferon alfa 2a or other antivirals eg, lamivudine, entecavir, adefovirdipivoxil. This treatment can slow the progression of cirrhosis, reduce incidence of liver cancer and improve long term survival.
- Treatment is often needs to be followed throughout the life as these antiviral drugs only helps to suppress the replication of the virus and doesn't cure Hepatitis B infection.
- Interferon injections though only can be considered in certain high income settings, as this may shorten treatment duration. But this treatment requires careful monitoring as it has significant adverse effects.
- In patients who develop Hepatocellular carcinoma due to background of Cirrhosis of liver, progresses rapidly, treatment options are limited and the outcome is in general poor. But the prognosis still remains low.

Prevention

WHO Recommendations for Hepatitis B Vaccination

- All infants should receive Hepatitis B vaccine under Immunization schedule.
- People who frequently require blood or blood products, dialysis patients, recipients of solid organ transplantations;
- IV drug abusers.
- Care givers who come in contact with carrier of patients with chronic HBV infection.
- People with multiple sexual partners;
- Health-care workers

E – II c) HEPATITIS C

Hepatitis C virus (HCV) is a RNA flavivirus which causes both acute and chronic infection. Acute HCV infection is usually asymptomatic, and rarely associated with life-threatening disease. Unlike Hepatitis B, about 15–45% of infected persons spontaneously clear the virus within 6 months of infection without any treatment but the remaining 55–85% of persons will develop chronic HCV infection. Of those with chronic HCV infection, the risk of cirrhosis of the liver is between 15–30% within 20 years.

Transmission

The hepatitis C virus is a bloodborne virus. It is most commonly transmitted through:

- IV Drug abusers
- Reuse or inadequate sterilization of medical equipment.
- Blood transfusion of unscreened blood
- Perinatal transmission
- Hepatitis C is not spread through breast milk, food, water or contact

Symptoms

- The incubation period for hepatitis C is 2 weeks to 6 months.
- Mostly asymptomatic.
- Fever
- Fatigue
- Low appetite
- Nausea and vomiting
- Abdominal pain

- Jaundice
- Joint pain

HCV infection Laboratory Tests :-

- LFT
- Anti HCV antibodies.
- Recombinant Immunoblot assay
- HCV – PCR
- Liver Biopsy if HCV PCR Positive to assess liver damage and need for treatment.
- Screening for anti-HCV antibodies with a serological test identifies people who have been infected with the virus.

Treatment

- Hepatitis C seldom requires treatment as immune response in some people helps to clear the infection.
- Interferon and Ribavarin are the durg of choice. But this treatment is frequently accompanied with Significant side effects and sometimes life-threatening adverse reactions.

E – II d) HEPATITIS D

This is an Incomplete RNA Virus, exists only with HBV and hence, spreads only with HBV. Clubbing of two viruses together hence, increases the risk of developing hepatits and cirrhosis. This is tested by Anti HDV antibody. Treatment option in HDV includes Interferon – alpha which has limited success in this disease.

E – II e) HEPATITIS E

Hepatitis E is caused by Hepatitis E Virus which is again is a RNA Virus. This virus almost exhibits same features as HAV. Treatment is symptomatic and disease is self limiting.

Hepatitis A, Hepatitis D, Hepatits E are usually self limiting with prodromal symptoms in acute phase of infection and usually doesn't cause Cirrhosis of Liver.

54. Sherlock's Diseases Of The Liver And Biliary System, Dr.Dooley, Dr.Lok, Dr.Burroughs, Dr.Heathcote, 12th Edi, Wiley – Blackwell Publications, Pg. No. 367 - 424

F) NEOPLASMS OF LIVER[55]

Benign Neoplasm of Liver:

Haemangioma: It is common among all benign tumors of the liver.

More commonly seen in females than males (5:1). It is usually solitary, symptomless, only found in necropsy or incidentally at operation.

Complication: Major complication is rupture with intraperitonealhaemorrhage which is often seen in children & pregnant women. The condition is fatal.

Management: Radiation therapy is the first line of treatment.

Malignant tumors of Liver:

Mainly two types – Hepatocarcinoma &Cholangiocarcinoma

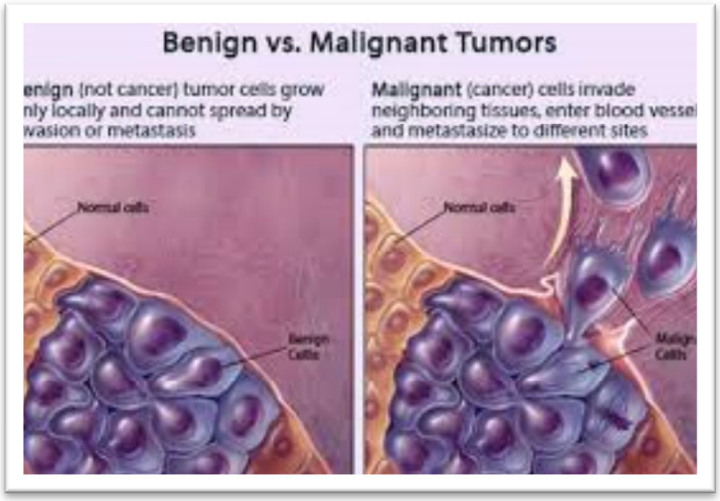
Hepatocarcinoma

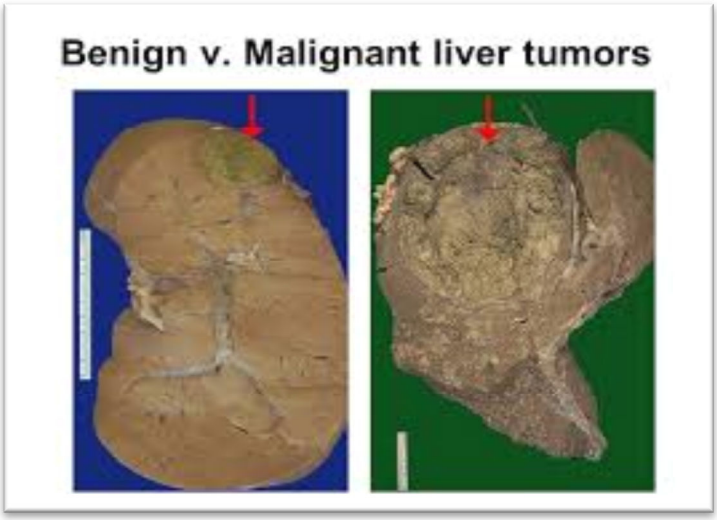
Causative factors:

- Hepatitis A
- Alcoholic cirrhosis
- Haemochromatosis
- Parasitic infestation
- Low protein intake
- Alpha-antitrypsin deficiency
- Blood group B

Table No.9 : Classification of primary Hepatic tumor

	Benign	**Malignant**
A	Hepatocellular tumors Hepatocellular adenoma	Hepatocellular carcinoma Hepatoblastoma
B	Billiary tumors – Bile duct adenoma (cholangioma)	Cholangiorcarcinoma, combined Hepatocellular &Cholangiorcarcinoma, Cystadenocarcinoma
C	Mesodermal tumors - Haemangioma	Angiosarcoma, Embryonal Sarcoma





Alpha toxins (Potent Carcinogen): These are found in wheat, soyabins, corns, oats

Clinical features

- Weakness, malaise, upper abdominal pain & weight loss
- Abdominal pain
- Jaundice
- Hepatomegaly
- Ascites

55. Cancer Principal and Practice of oncology, Vincent T. Devita, Jr.,Samuel Hellman , Steven A. Rosenberg, 7th edition Manual of clinical oncology, Dr.denis a. Casciato, 5th edi, JP Indian edition, Pg.No. 219 - 225

Stages of liver cancer [56]

The stage of cancer is a description of how much is the cancer spread in the body using TNM system.

The staging system is based on the result of physical examination, imaging tests (USG, CT / MRI etc.) as well as the results of surgery if it has been done.

TNM –

T: It describe the number and size of the primary tumor(s) measured in cm and whether the cancer has grown into nearby blood vessels or organs.

N: Describes the external of spread to nearby regional lymph nodes.

M: Indicates whether the cancer has metastasized (Spread) to distant parts of the body. (The most common sites of liver cancer spread are lungs and bones)

T Groups:

Tx: Primary tumour cannot be assessed.

T0: No evidence of primary tumour.

T1: A single tumour or (any size) that has grown into blood vessels.

T2: Either a single tumour (any size) that has not grown into blood vessels or more than one tumour where no tumour is larger than 5 cm (about 2 inches) across.

T3a: More than 1 tumour with at least 1 tumour larger than 5 cm across.

T3b: At least one tumour (any size) that has grown into a major branch of a large vein of the liver (Portal / hepatic vein)

T4: The tumour (any size) has grown into a nearby organ (other than the gall bladder) or the tumour is growing into the thin layer of tissue covering the liver.

N Groups:

Nx: Regional (nearby) lymph nodes cannot be assessed.

N0: The cancer has not spread to the regional lymph nodes.

N1: The cancer has spread to the regional lymph nodes.

M Groups:

M0: The cancer has not spread to the distant lymph nodes / other organs.

M1 : The cancer has spread to the distant lymph nodes or other organs liver cancer most often spreads to the lining of the belly (peritorium) the lungs and to bones.

Stage grouping :

Stage – I: T1 N0 M0

There is a single tumour that has not grown into any blood vessels. The cancer has not spread to nearby lymph nodes / distant sites.

Stage II: T2 N0 M0

Either there is a single tumour (any size) that has grown into blood vessels / there are several tumours and all are 5 cm (2 inches) or less across. Cancer has not spread to nearby lymph nodes or distant sites.

Stage III A: T3a N0 M0

More than 1 tumour and at least one is larger than 5 cm. Cancer has not spread to nearby lymph nodes / distant sites.

Stage III B: T3b N0 M0

At least one tumour is growing into a branch of major vein of the liver (portal / hepatic) cancer has not spread.

Stage III C: T4 N0 M0

Tumour is growing to nearby organ other than the gall bladder / a tumour has grown into outer covering of the liver cancer has not spread.

Stage IV A: Any T N1 M0

Tumour of any size / no. and may have grown into blood vessels / other organs cancer has spread to nearby lymph nodes but not spread into distant organs.

Stage IV B: Any T Any N M1

Cancer has spread to other parts of the body.

Other staging systems:

- Barcelona Clinical Liver Cancer System (BCLC)
- Cancer of the liver Italian programm system (CLIP)
- Okuda system

56. http://www.cancer.org/acs/groups/cid/documents/webcontent/003114-pdf.pdf

Investigations for HCC:

1. Blood Examination –

   Erythrocytosis, BSP & Alkaline phosphatase level in absence of bone disease & obstructive jaundice is considered presumptive evidence of Hepatocellular carcinoma. Alpha-fetoprotein (AFP) in the serum by immune-electrophoresis & immunoassay technique.
2. Ultrasonography, Radionucliotide scans (liver scans), CT scan
3. Selective hepatic arterography

TREATMENT [57]

Options:

- Surgery
- Ablation
- Embolization
- Targeted Therapy
- Chemotherapy

- **Surgical Removal : (**Complete / partial)

  It is a removal of tumour and surrounding tissue.

- For early stage of liver cancer
- Small tumours < 5cm
- Hepatectomy: Part of the liver is removed and the remaining section of liver takes care of the functions of entire liver and many regrow to its normal size within few weeks. But may not be possible if patient has advanced cirrhosis.
- Liver transplantation - When cancer has not spread out of the liver and suitable donor is found and very specific criterias are met.

1) Thermal ablation : RFA

- Radiofrequency ablation uses heat to destroy cancer cells.
- It may be given through skin, through laparoscopy or during a surgery when the patient is sedated.

2) Percutaneous ethanol injection :

Alcohol is injected directly into the liver tumour to destroy it. S/E includes fever and pain after the procedure. But it is very simple, safe and particularly effective for a tumour smaller than 3 mm. But it is used less often and has been largely replaced by RFA.

3) Chemoembolization :

- This is a type of chemotherapy treatment in which drugs are injected directly into the hepatic artery and then the flow of blood through the artery is blocked for a short time so the chemotherapy stays in the tumour longer.
- Blocking the blood supply to the tumour helps to kill cancer cells.

4) Radiation therapy

- External beam radiation therapy is radiation given from a machine outside the body. It is not often used for HCC.
- Internal Radiation therapy –It is used as placing radioactive beads into the artery that supplies the tumour with blood in a manner similar to chemoembolization.

5) Targeted Therapy :

- It is a treatment that targets the cancer's specific genes, proteins or the tissue environment that contributes to cancer growth and survival.
- This blocks the growth and spread of cancer cells while limiting damage to cancer cells.
- For HCC anti-angiogenesis drugs are the most common targeted therapy.
- It stopps the angiogenesis(the process of forming new blood vessels) hence it causes starvation of the tumour.
- Sorafenib for advanced HCC given orally.

6) Chemotherapy :

a. Systemic chemotherapy treatment: It is delivered through the blood stream to reach cancer cells throughout the body. It is given through IV tubes placed into a vein using a needle.

b. Regional chemotherapy Treatment: A small pump is surgically placed in the body to deliver chemotherapy directly to the blood vessels that feed the tumour. But chemotherapy is not as much of use for HCC.

   S/E of chemotherapy depend on the individual and the dose used but they can include nausea and vomiting, hair loss, loss of appetite or bruising, tingling of hands and feet, headache.

7) Cryosurgery :

- It uses extreme cool ice crystals to freeze and kill cancer cells.

8) Hepatic Arterial Infusion :

- It uses an anticancer drug injected into a Cather that has been placed in the major artery supplying blood to the liver.
- It is a type of chemo treatment and it doesn't have many side effects.

9) Immunotherapy or Biologic Therapy

- It is designed to boost the body's natural defences to fight with the cancer.
- Side effects are similar to flue, fever, chills, muscle pain, headache.

Metastatic Tumors

Liver is the second most common organ after lymph nodes for metastatic carcinomas. Bronchogenic carcinoma is the most common primary carcinoma causing liver metastasis. While others are colorectal, pancreas, breast & stomach tumors.

Metastasis in Liver reaches by four routes

- Portal vein
- Lymphatic spread
- Hepatic artery
- Direct infiltration

The growth rate of the Metastatic Tumor is more rapid than the original lesion. Metastasis in

Clinical features

- Weight loss
- Anorexia
- Hepatomegaly with firm nodularity & irregular margin
- Ascites
- Jaundice

Special Investigations

a) Liver Function tests
b) Carcinoembryonic antigen (CEA)
c) Serum AFP is not raised in liver metastasis unlike hepatocellular carcinoma.
d) Radionucleotide scan; CT scan & angiography

Treatments:

Treatment of liver metastasis is only considered when

- There is no systematic metastasis
- Primary tumours has been treated properly
- Patients condition is well to tolerate major operative procedures

57. Cancer Principal and Practice of oncology, Vincent T. Devita, Jr.,Samuel Hellman , Steven A. Rosenberg, 7th edition Sherlock's Diseases Of The Liver And Biliary System, Dr.Dooley, Dr.Lok, Dr.Burroughs, Dr.Heathcote, 12th Edi, Wiley – Blackwell Publications

G) HEPATOMEGALY

Abnormal enlargement of liver.

Common Causes of Hepatomegaly:

- Alcoholic liver disease (alcoholic fatty liver disease, alcoholic hepatitis and cirrhosis)
- Nonalcoholic fatty liver disease(a lifestyle-related metabolic disease)
- Viral hepatitis (hepatitis A, B, C, D or E)
- Malignancy: Metastatic or primary (Hepatocellular carcinoma).
- Haemolytic anaemias

Other Causes of Hepatomegaly:

- **Haematological Cancers :** Leukaemia, Lymphoma,Myeloproliferative disorders ,Sickle cell disease
- **Genetic diseases :**Hemochromatosis, Wilson's disease, Glycogen storage diseases
- **Infections :** Liver abscess, Other parasitic infections, recurrent fever
- **Drug-induced**: Due to medications such as acetaminophen and amoxicillin-clavulanate
- **Toxic hepatitis**: From exposure to poisons eg.Industrial chemicals
- **Systemic diseases:** Amyloidosis, Autoimmune hepatitis, Primary biliary cirrhosis, Primary sclerosing cholangitis.

PREVIOUS WORK DONE

Ph.D. Thesis Work –

1975 – Banaras Hindu University, Varanasi – Effect of indigenous drugs in the management of certain liver diseases.

1986 - Banaras Hindu Univesity ,Varanasi – A critical study of Ayurvedic clinical hepatomegaly & treatment of certain liver disorders with Kalamegha.

2006 – Delhi University – Effect of Phalatrikadi Kwath in the management of Koshtashakhashrita Kamala w.s.r. to Hepatocellular Jaundice.

Post Graduate Thesis Work –

2005 – Gujarat Ayurved University,Jamnagar – Yakrut Dosha

2005 – Gujarat Ayurved University,Jamnagar –Kamala with practical role of Trikatu.

MATERIAL AND METHODOLOGY

❖ **Inclusion Criteria –**

- Patients with confirm diagnosis of following liver disorders :

a) Hepatocellular Carcinoma (Stage I to IV)
b) Liver Cirrhosis
c) Liver metastasis
d) HBsAg+ ve
e) Hepatitis B & C
f) Hepatomegaly

- Patients which are not responding to Surgical treatment, Chemotherapy, Radiation, RFA (Radiofrequency Ablation), Chemoembolization, Liver transplant.
- Patients of age group 20 – 70 yrs.
- Patients of either sex.

❖ **Exclusion Criteria –**

- Patients having following disorders :

a) Ascitis
b) Portal vein obstruction
c) Spleenomegaly
d) Cholengiocarcinoma

- Patients undergoing Chemotherapy, Radiation therapy, RFA therapy and Chemoembolization.

❖ **Clinical trial –**

Clinical Trial was carried out as follows -

- **Group A : (Experimental group)** –

Patients receiving combination of Yakrut Plihari Loha Vati, Kutakichurna & Kumari KalpaVati i.e. YG3. 22 patients were enrolled in this group.

- **Group B : (Control group) –**

  Patients receiving Yakrutplihari Loha Vati & Kutakichurna.It is named as YG2 combination. 26 patients were enrolled in this group.

❖ **Dose Design –**

- **Table no. 10 - Dose Design For Group A :**

Medicine	**Reference**	**Standardization**	**Dose**	**Anupan**	**Kala**
Yakrut Pleehari Loha & Kutaki Churna	Bhaishajya Ratnavali (41/162 – 166)	Manufactured as per GMP Norms and Standardized as Per IHS	125mg+ 500mg	Ghruta 2.5 gms	Rasayana Kala (Morning) & Antarabhakta Kala (Evening).
Kumari Kalpa Vati	(Proprietary medicine of Atharva Nature Health Care Pvt Lmtd)	Manufactured as per GMP Norms and Standardized as Per IHS	250 mg (2 tab)	Warm water	Vyanodan Kala (After Lunch & Dinner)

- **Table no. 11 - Dose Design For Group B :**

Medicine	**Standardization**	**Dose**	**Anupan**	**Kala**
Yakrut Pleehari Loha & Kutaki Churna	Manufactured as per GMP Norms and Standardized as Per IHS	125mg + 500mg	Ghruta 2.5 gms	Rasayana Kala (Morning) & Antarabhakta Kala (Evening).

***GMP – Good Manufacturing Practices**

***IHS – In House Standardization**

❖ **Duration of treatment –**

- Patients received the treatment up to 6 months.

❖ **Methodology–**

1. Patients of Liver disorders mentioned in the inclusion criteria were selected for the study.
2. Literature study was done on Yakrutavayava, Yakrutvikruti, Yakrut Chikitsa, Bhedana Karma etc.
3. Modern science was studied in detail for Liver organ, Liver disorders, necessary investigations, Treatment modalities etc.
4. Standardized Ayurvedic medicines were used for the study i.e.
   YG3 (Combination of Yakrut Plihari Loha + Kutaki Churna + Kumara Kalpa Vati) and YG2 (Yakrut PlihariLoha + Kutaki Churna).
5. The study was an open labelled controlled clinical trial.
6. Patients were divided in 2 groups as mentioned above.
7. Specially designed informed written consent was taken.
8. Detail case was taken on specially designed Case Record Form.
9. Assessment with Standard criteriaand required investigations was done in patients of Yakrut Vikar before starting the treatment, middle of the treatment and at the end of the treatment.
10. Statistical analysis was done using ManWhitney Z test for symptoms, unpaired t Test for percentage of weight loss.

❖ **Assessment Criteria with Gradation -**

- **Clinical Parameters**

- According to Ayurved

- According to Modern medical science

- **Pathological Parameters**
- **Radiological Parameters**

A) Table no. 12 - Clinical Symptoms

Grading	**0**	**1**	**2**	**3**
Udarshool / Yakrut-pradeshishul	No Symptoms	mild pain not interfering with function	moderate pain: pain or analgesics interfering with function, but not interfering with activities of daily living	severe pain: pain or analgesics severely interfering with activities of daily living
Yakrut-plihavrudhi	No Symptoms	>or = 2 Fingers	2 fingers – 4 fingers	Whole abdomen occupied by the enlargement of liver
Agnimandya	No Symptoms	loss of appetite	oral intake significantly decreased	requiring IV fluids
Aruchi	No Symptoms	Without any alteration in intake	Significant alterations in intake	Severe tastelessness with nausia
Hrullas	No Symptoms	able to eat	oral intake significantly decreased	no significant intake, requiring IV fluids
Sharira Shaithilya	No Symptoms	Mild	Moderate	Severe
Avipaka	No Symptoms	Mild indigestion	Moderate indigestion with other GI symptoms	Significant indigestion needs medical care
Daha	No Symptoms	Mild	Moderate	Severe
Hatendriya	No Symptoms	Ultered normal activites	Activities getting restricted	Significant ulteration in activities
Bhekavarnata	No Symptoms	Mild	Moderate	Severe

Grading	**0**	**1**	**2**	**3**
Shithila Mala Pravrutti	No Symptoms	increase of < 4 stools/day over pre-treatment	increase of 4-6 stools/day, or nocturnal stools	increase of ³7 stools/day or incontinence; or need for parenteral support for dehydration
Netra Pitata	No Symptoms	Sclera appearing slightly yellow	Sclera appearing moderately yellow	Sclera appearing signinificantly yellow
Twak Pitata	No Symptoms	Yellowish discoloration of Skin	Skin colour becomes moderately yellow	Skin colour significantly yellow

A) Table no. 13 - Clinical conditions -

Grading	**0**	**1**	**2**	**3**
Itching	No Symptom	occasional itching which is bearable	Frequency & intensity of itching increased	Itching all over the body with increased frequency
Jaundice	No Symptom	Mild symptoms of Jaundice with bilirubin level more than 1.2 gm/dl	Symptoms of jaundice increased moderately with raised LFTs	Medical attention required with significantly high symptoms
Haematemesis	No Symptom	Occurred only once	More than 2 episodes of haematemesis	Life threatening conditions
Weight loss	No Symptom	Not more than 1 to 2 kgs in short period of time	More than 2 kgs with other GI symptoms in short period of time	Significantly low wt. in very short period of time

Grading	**0**	**1**	**2**	**3**
Fullness	No Symptom	Symptoms but not interfering with GI tract	Symptoms interfering with GI tract	--
Abdominal swelling	No Symptom	Mild	Moderate	Severe
Vomiting	No Symptom	Occasional episodes not much interfering with intake	More than 2 episodes of per day with sign of dehydration	Severe dehydration
Malaise	No Symptom	Mild	Moderate	Severe
Bone pain	No Symptom	Bearable without medication	Needs frequent medication for pain relief	Regularly on high dose of medication and still uncomfortable
Tremors	No Symptom	Mild	Moderate	Severe
Disorientation	No Symptom	Present	--	--
Asthenia / Weakness	No Symptom	increased fatigue over baseline, but not altering normal activities	moderate decrease in performance status by 1 ECOG level or 20% Karnofsky	severe (e.g., decrease in performance status by 2 ECOG levels or 40% Karnofsky

Table no. 14 - Karnofsky Score

	Karnofsky Score
100	Normal, no complaints, no evidence of diseases.
90	Able to carry on normal activity, minor signs or symptoms of diseases.
80	Normal activity with effort, some signs & symptoms of diseases.
70	Cares for self, unable to carry on normal activity or do active work.
60	Requires occasional assistance but is able to care for most of his or her needs.
50	Requires considerable assistance & frequent medical care.
40	Disabled, required special care & assistance.
30	Severely disabled, hospitalization indicated death not imminent.
20	Very seek, hospitalization indicated, death not imminent.
10	Moribund fatal processes progressing rapidly.

Table no. 15 – Pathological Parameters

Investigations	**Normal**	**Low**	**Medium**	**High**
HB	12-14 gm/dl	< 8 gm/dl	8-12 mg/dl	>14 gm/dl
WBC	4000-10000/cmm	< 2000 / cmm	2000-4000/cmm	>10000 /cmm
Platelet Count	1.5-4.5 lacs/cmm	< 50000 / cmm	0.5-1.5 lacs/cmm	> 4.5 lacs/cmm
Sr. Bilirubin	0-1.23 g /dl	--	--	>1.23 mg/dl
SGPT	0 – 30 IU/L	30 – 50 IU/L	50 – 90 IU/L	> 90 IU/L
SGOT	0 – 40 IU/L	40 – 60 IU/L	60 – 100 IU/L	> 100 IU/L
Serum Alk. Phosphatase	44 – 147 U/L	147 – 200 U/L	200 – 300 U/L	> 300 U/L
Sr. Protein	6.4-8.3 g/dl	< 6.4 g/dl	> 8.3 g/dl	--
Sr. Albumin	3.5-5 g/dl	< 3.5 g/dl	> 5 g/dl	--
Sr. Globulin	2.6-4.6 g/dl	< 2.6 g/dl	> 4.6 g/dl	--
Type of Hepatitis	--	--	--	--

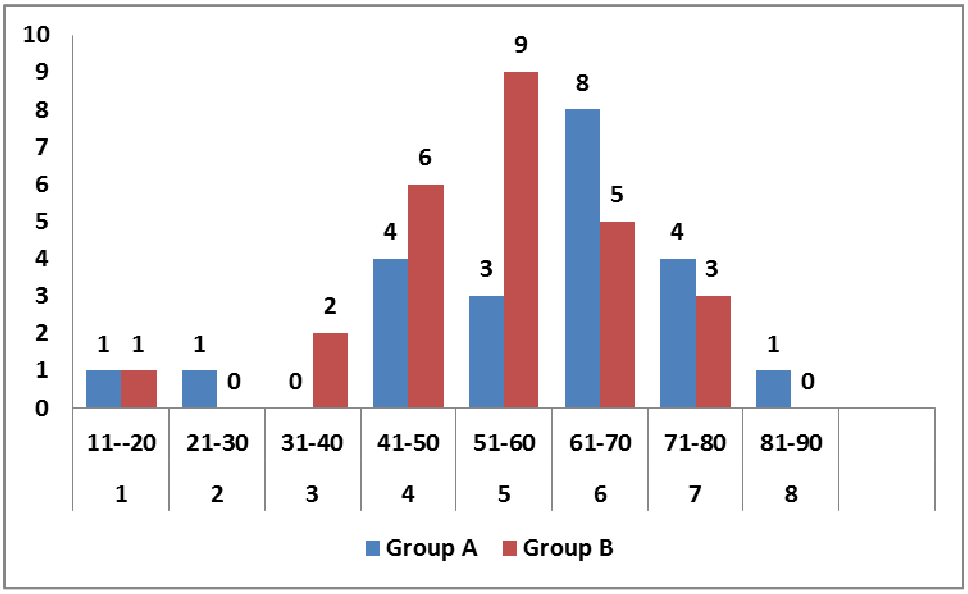
OBSERVATIONS AND RESULTS

I) OBSERVATIONS OF DEMOGRAPHIC DATA

Table 16 – Showing Age wise distribution of patients of Yakrut vikara treated with Ayurvedic medicines

Sr. No	Age	Group A	Group B
1	11-20	1	1
2	21-30	1	0
3	31-40	0	2
4	41-50	4	6
5	51-60	3	9
6	61-70	8	5
7	71-80	4	3
8	81-90	1	0
	Total	**22**	**26**

Graph 1 – Graphical representation of age wise distribution of patients of Yakrut vikara treated with Ayurvedic medicines

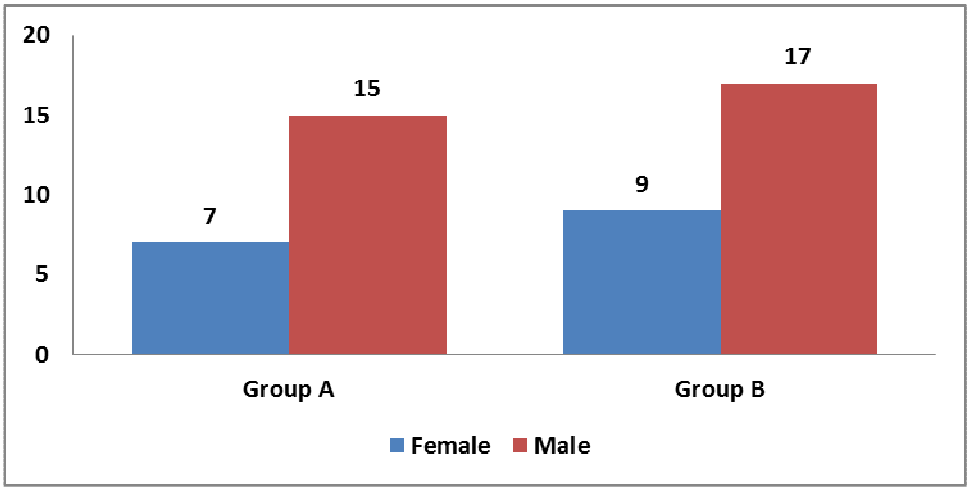


Proportion of patients of Yakrut vikara in both groups is higher in age 41 yrs to 80 yrs.

Table 17 – Showing Sex wise distribution of patients of Yakrut vikara treated with Ayurvedic medicines

Sr. No	Sex	Group A	Group B
1	Female	7	9
2	Male	15	17
	Total	22	26

Graph 2 – Graphical representation of Sex wise distribution of patients of Yakrut vikara treated with Ayurvedic medicines

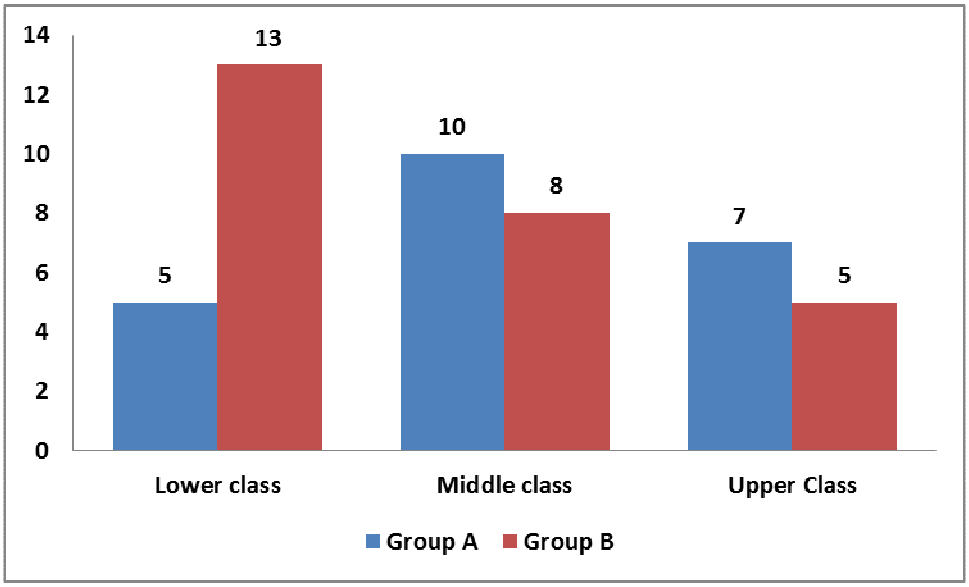


In both the groups of patients of Yakrut vikara, number of male patients were more than that of female patients. This may be due to the fact that the alcohol addiction is more common in males, which is the main cause of Yakrut vikara.

Table 18 – Showing distribution of Socio – economical status of patients of Yakrut vikara treated with Ayurvedic medicines

Sr. No	Socio-economical status	Group A	Group B
1	Lower class	5	13
2	Middle class	10	8
3	Upper Class	7	5
	Total	22	26

Graph 3 – Graphical representation of distribution of socio–economical status of patients of Yakrut vikara treated with Ayurvedic medicines

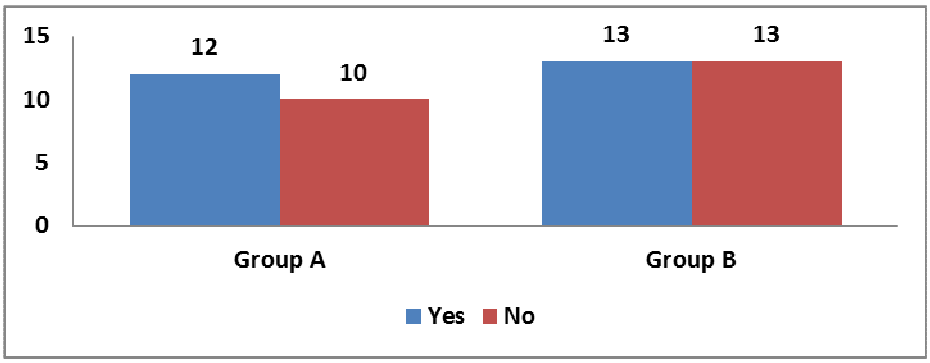


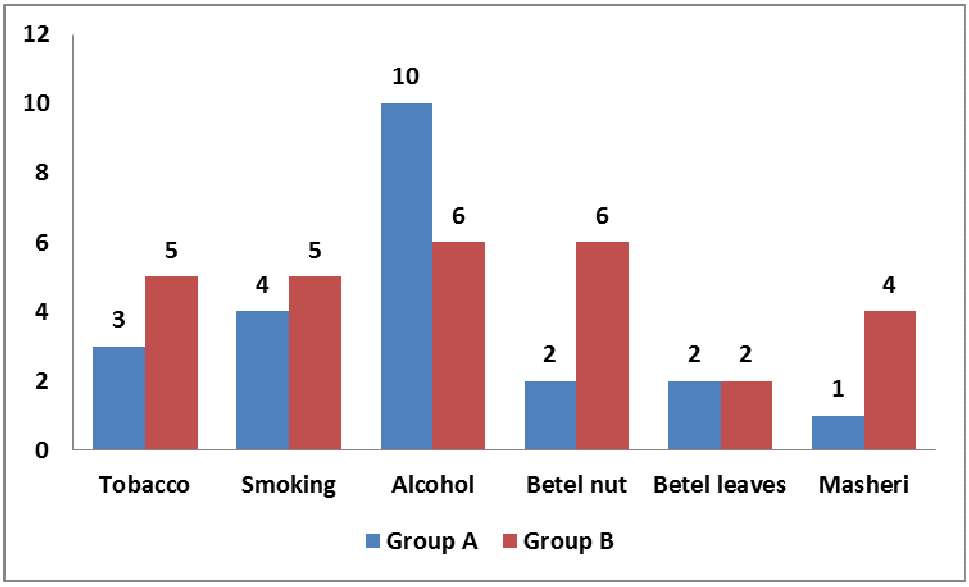
In study group (group A) socio-economically middle class patients were more (10), while lower class patients were more in group B (13).

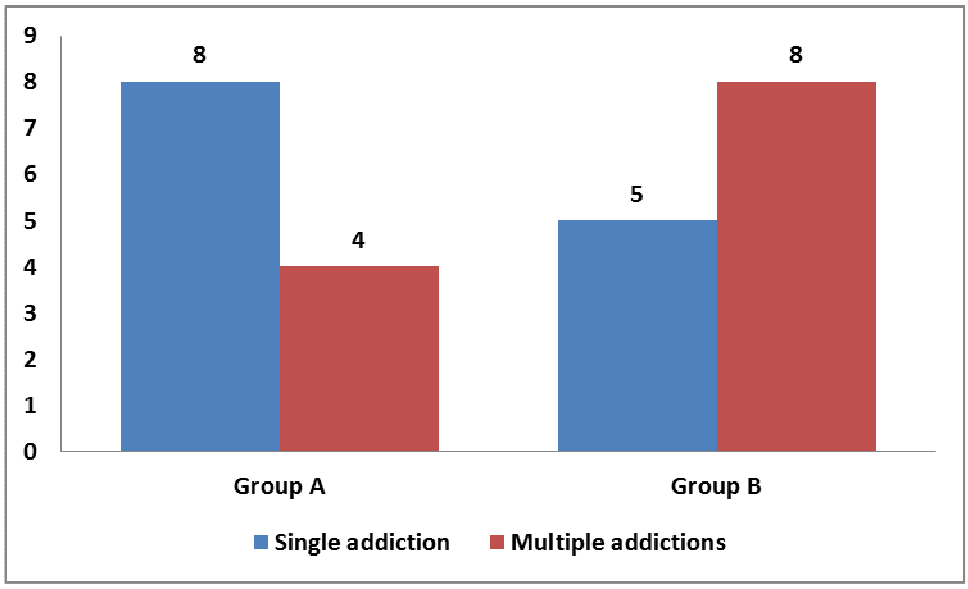
Table 19– Showing number of patients of Yakrut vikara treated with Ayurvedic medicines, having history of addiction as a risk factor

Addiction	Group A	Group B
Yes	12	13
No	10	13
Total	22	26
Types of addiction		
Addiction	**Group A**	**Group B**
Tobacco	3	5
Smoking	4	5
Alcohol	10	6
Betel nut	2	6
Betel leaves	2	2
Masheri	1	4
Number of addictions		
	Group A	**Group B**
Single addiction	8	5
Multiple addictions	4	8

Graph 4 – Graphical representation of number of patients of Yakrut vikara treated with Ayurvedic medicines, having history of addiction as a risk factor







In our study 12 patients in group A and 13 patients in group B had history of addiction. Alcohol consumption and multiple addictions are very common risk factors of Yakrut vikara.

Table 20– Showing number of patients of Yakrut vikara treated with Ayurvedic medicines, having dietary risk factors

Dietary items as risk factors	Group A	Group B
Green chilli	12	8
Curd	10	6
Pickle	9	7
Non vegetarian food	7	6
Tomato	5	5
Red chili powder	5	4
Raw onion	4	4
Garlic	4	4
Extra salt	4	3
Deep fried food	4	5
Spicy food	4	4
Banana	2	2
Kakavi	2	2
Lemon	2	2
Tamarind	2	2
Cold drinks	2	2
Virudhhanna	2	5
Fish	2	2
Bakery products	2	2
Excess sweets	2	2
Salted butter	2	1
Sprouts	2	1
Outside food	0	2
Stale food	1	2

Graph 5 – Graphical representation of number of patients of Yakrut vikara treated with Ayurvedic medicines, having dietary risk factors

In our study green chilli, curd, pickle, non-vegetarian food, tomato and red chili powder are observed as main risk factors of Yakrut vikara.

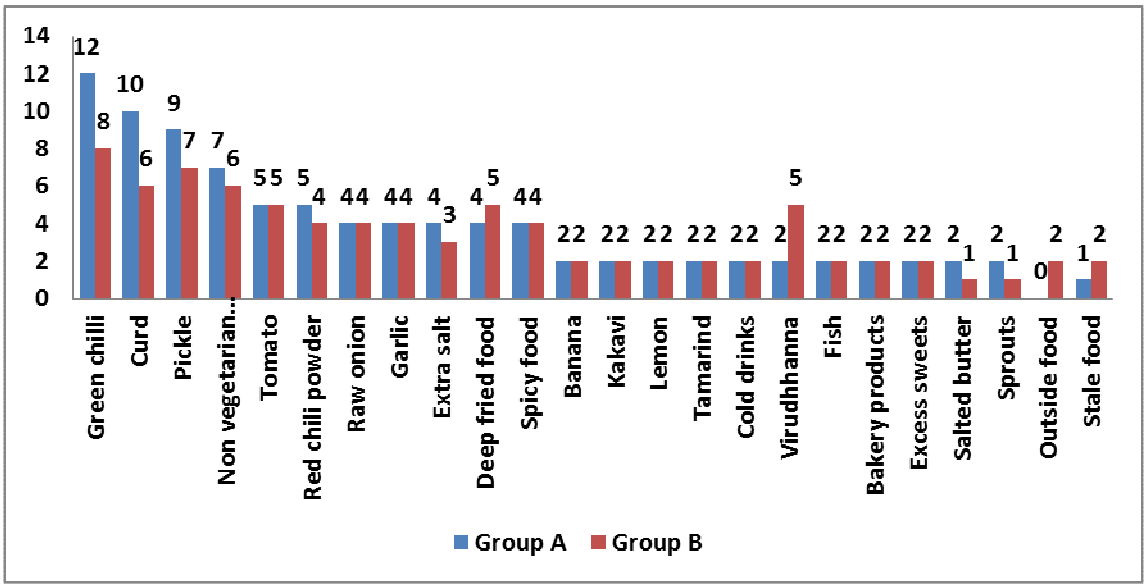
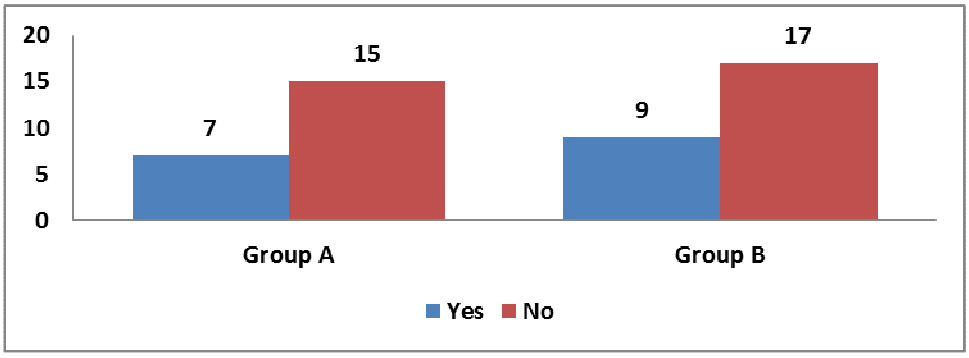


Table 21 - Showing number of patients of Yakrutvikara treated with Ayurvedic medicines, having hereditary diseases as risk factor

Sr. No	Hereditary diseases as risk factor	Group A	Group B
1	Yes	7	9
2	No	15	17
	Total	22	26

Graph 6 – Graphical representation of number of patients of Yakrut vikara treated with Ayurvedic medicines, having hereditary diseases as risk factor



Heredity is an important risk factor of Yakrut vikara. Yakrut being Matruja avayava, heredity of diseases of matruja avayava is commonly seen in Yakrut vikara.

Heredity of CA stomach, CA bresat, Liver Cirrhosis, Hepatocellular carcinoma, CA throat, CA oesophagus was found in patients included in the study.

Table 22– Showing number of patients and type of diseases as a hereditary risk factor in patients of Yakrut vikara treated with Ayurvedic medicines

Sr. No.	Hereditary disease	Group A	Group B
1	Cirrhosis	1	0
2	Hepatocellular carcinoma	1	1
3	CA Stomach	2	0
4	CA Oesophagus	0	1
5	Leukemia	0	1
6	CA unknown origin	1	0
7	CA Breast	2	5
8	CA Ovary	0	1
9	CA Throat	1	2
10	CA Maxilla	0	1

Graph 7 – Graphical representation of number of patients and type of diseases as a hereditary risk factor in patients of Yakrut vikara treated with Ayurvedic medicines

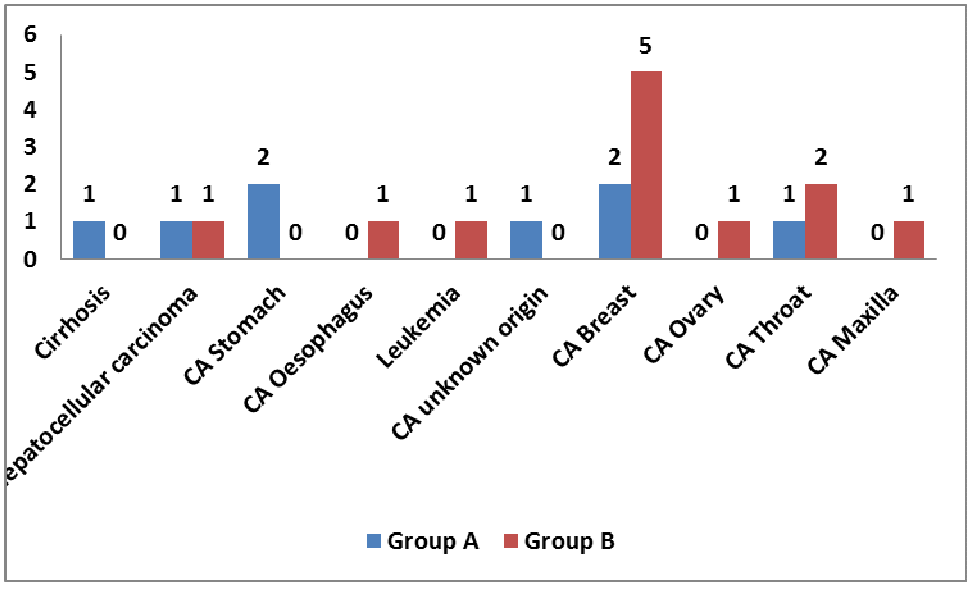
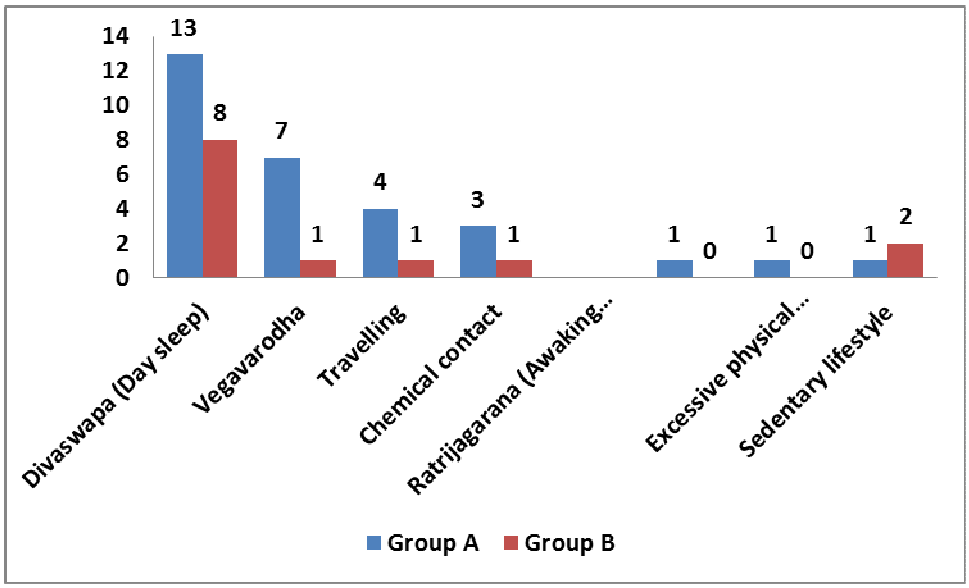


Table 23 – Showing number of patients of Yakrut vikara treated with Ayurvedic medicines, having abnormal lifestyle (Vihara) as risk factor

Sr. No	Abnormal lifestyle as a risk factor	Group A	Group B
1.	Diwaswapa (Sleeping in daytime)	13	8
2.	Vegavarodha (Holding natural urges)	7	1
3.	Chankramana (Travelling)	4	1
4.	Chemical contact	3	1
5.	Ratrijagarana (Awaking till late night)		
6.	Long term contact with hot atmosphere	1	0
7.	Excessive physical exertion	1	0
8.	Sedentary lifestyle	1	2

Graph 8 – Graphical representation of number of patients of Yakrut vikara treated with Ayurvedic medicines, having abnormal lifestyle (Vihara) as risk factor



Abnormal vihara is responsible for Agnidushti and Raktadushti. Diwaswapa, vegavarodha, chankramana, long lasting contact with chemicals and sedentary lifestyle are observed as major risk factors of Yakrut vikara in our study.

Table 24 – Showing number of patients of Yakrut vikara treated with Ayurvedic medicines, having mental stress as a risk factor in patients of Yakrut vikara

Sr. No	Mental stress	Group A	Group B
1	Yes	11	19
2	No	11	7
	Total	22	26

Graph 9 – Graphical representation of number of patients of Yakrut vikara treated with Ayurvedic medicines, having mental stress as a risk factor in patients of Yakrutvikara

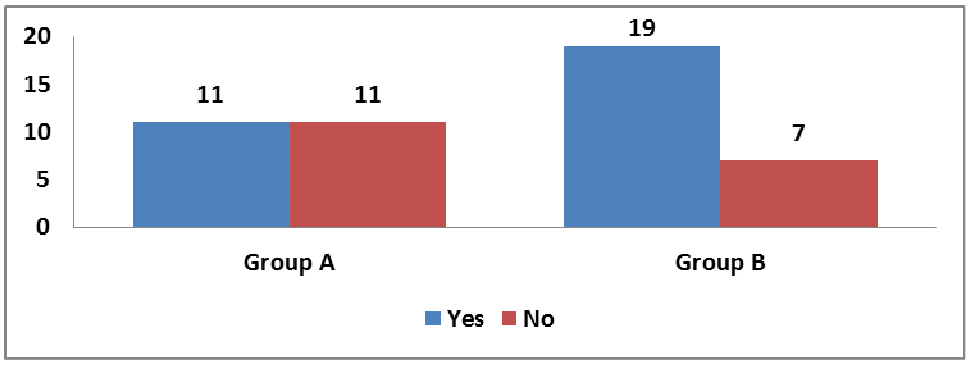
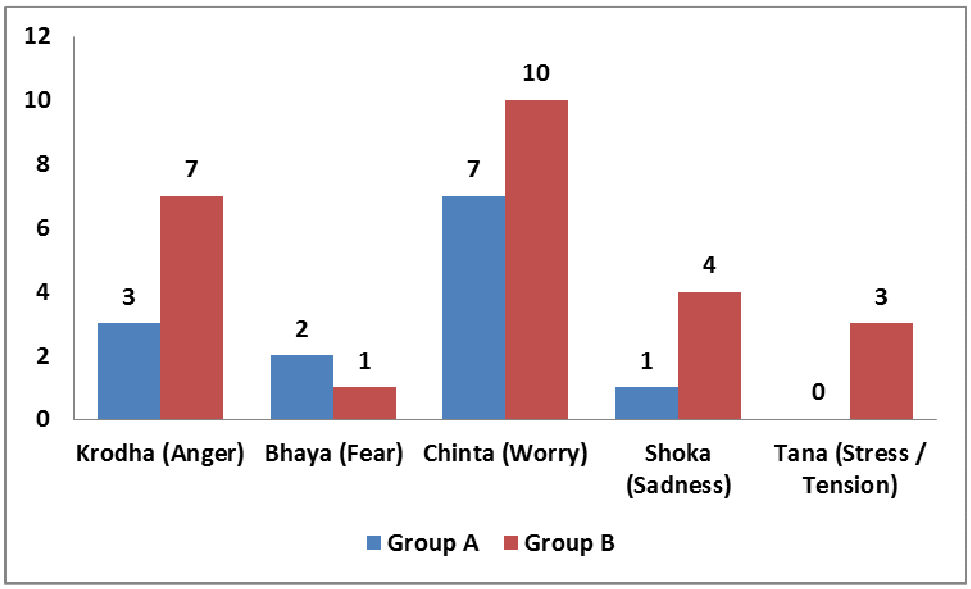


Table 25 – Showing number of patients of Yakrutvikara treated with Ayurvedic medicines and their type of mental stress as a risk factor

Sr. No	Type of Mental stress	Group A	Group B
1	Krodha (Anger)	3	7
2	Bhaya (Fear)	2	1
3	Chinta (Worry)	7	10
4	Shoka (Sadness)	1	4
5	Tana (Stress / Tension)	0	3

Graph 10 – Graphical representation of number of patients of Yakrut vikara treated with Ayurvedic medicines and their type of mental stress as a risk factor

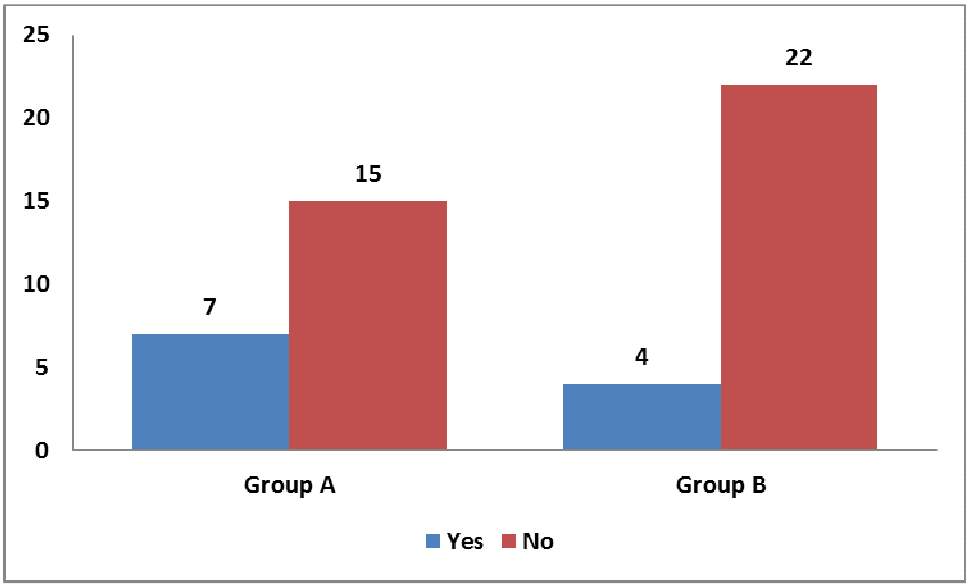


Mental stress is a cause of Rasa and Rakta dhatu dushti, leading to Yakrut vikara. In our study, 50% patients in both the groups had mental stress. Among manasa bhava, chinta (worry) is found to be evident risk factor of Yakrut vikara.

Table 26 – Showing number of patients of Yakrutvikara treated with Ayurvedic medicines having Aghata (External trauma) as a risk factor

Sr. No	History of external trauma	Group A	Group B
1	Yes	7	4
2	No	15	22
	Total	22	26

Graph 11 – Graphical representation number of patients of Yakrut vikara treated with Ayurvedic medicines having Aghata (External trauma) as a risk factor



II) OBSERVATIONS ON SYMPTOMS OF YAKRUT VIKARA

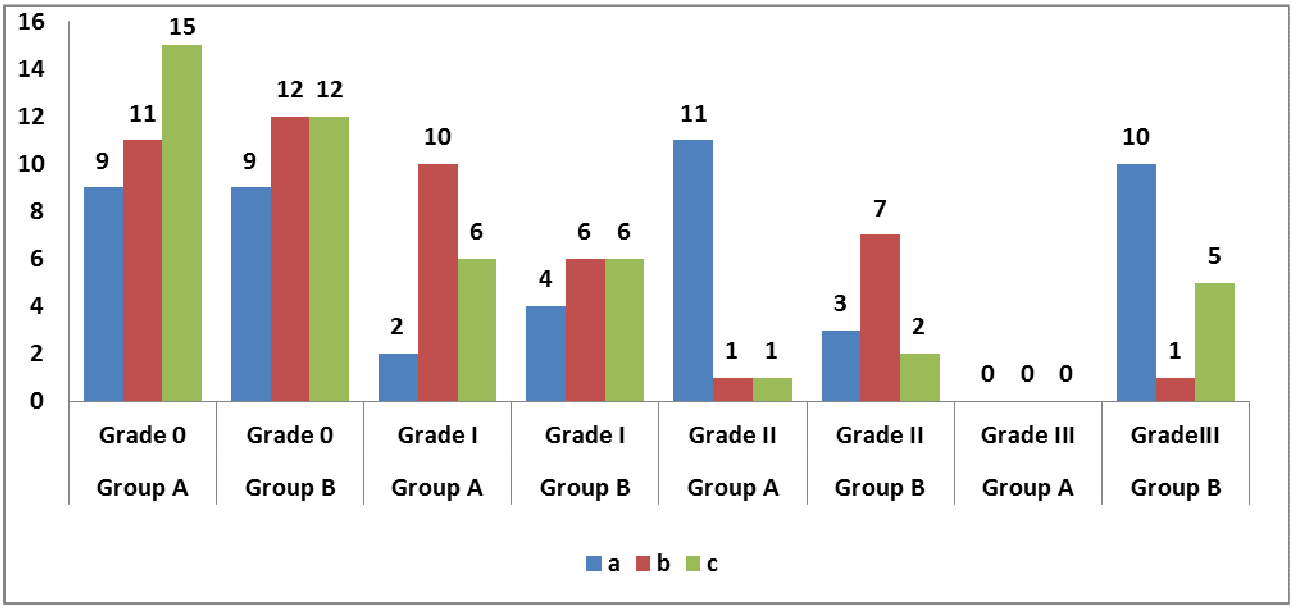
- Symptoms of Yakrut vikara are graded from 0 to III.
- Grade 0 indicates absence of symptom, while grade III represents severe symptom.
- Each symptom is assessed before starting Ayurvedic treatment (time point a), in the middle of the treatment (time point b) and at the end of treatment (time point c). Statistical analysis of symptoms is done by 2 ways –

1. Inter group analysis – Analysis of symptom within 1 group at time points b with a and c with a.
2. Intra group assessment – Analysis of symptom within group A and group B at certain time points i.e. at time points b and c.
   This type of assessment is essential to assess role of 2 treatment modalities individually and comparatively and thus finally to draw a conclusion about their efficacy as bhedana karma.

Table 27 – Showing gradation of Udarshool / Yakrut pradeshi shool

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade I**		**Grade II**		**Grade III**	
A	9	9	2	4	11	3	0	10
B	11	12	10	6	1	7	0	1
C	15	12	6	6	1	2	0	5

Graph 12 – Showing gradation of Udarshool / Yakrut pradeshi shool



9 patients from group A and B did not suffer from Udara shula / Yakrut pradeshi shula at time point a. At time point c, 15 patients from group A and 12 patients from group B did not have Udara shula / Yakrut shula. This indicates effectiveness of YG3 in reducing Udara shula / Yakrut pradeshi shula. 2 patients in group A and 4 patients in group B had grade I Udara shula / Yakrut pradeshi shula at time – point a, while 6 patients in each group had grade I Udara shula / Yakrut pradeshi shula at time point c indicating better improvement in Udara shula with YG3 medicine. Grade II Udara shula / Yakrut pradeshi shula was observed in 11 patients in group A and 3 patients in group B at time-point a. Number of patients having grade II symptom reduced to 1 and 2 at time point c. This observation also supported the fact that YG3 is highly effective to minimize Udara shula / Yakrut pradeshi shula by its bhedana karma. Grade III Udara shula was not present in a single patient of group A at all 3 time-points (a,b,c), thus it was not possible to compare the efficacy of YG3 and YG2 in 2 groups.

Very significant statistical result is obtained in Udarshool / Yakrut pradeshi shul in Group A patients, when the grading is compared between time points c and a ($p=0.004$) and significant statistical result is obtained when compared between time points b and a ($p=0.03$). This implies efficacy of YG3 in reducing Udarashool by

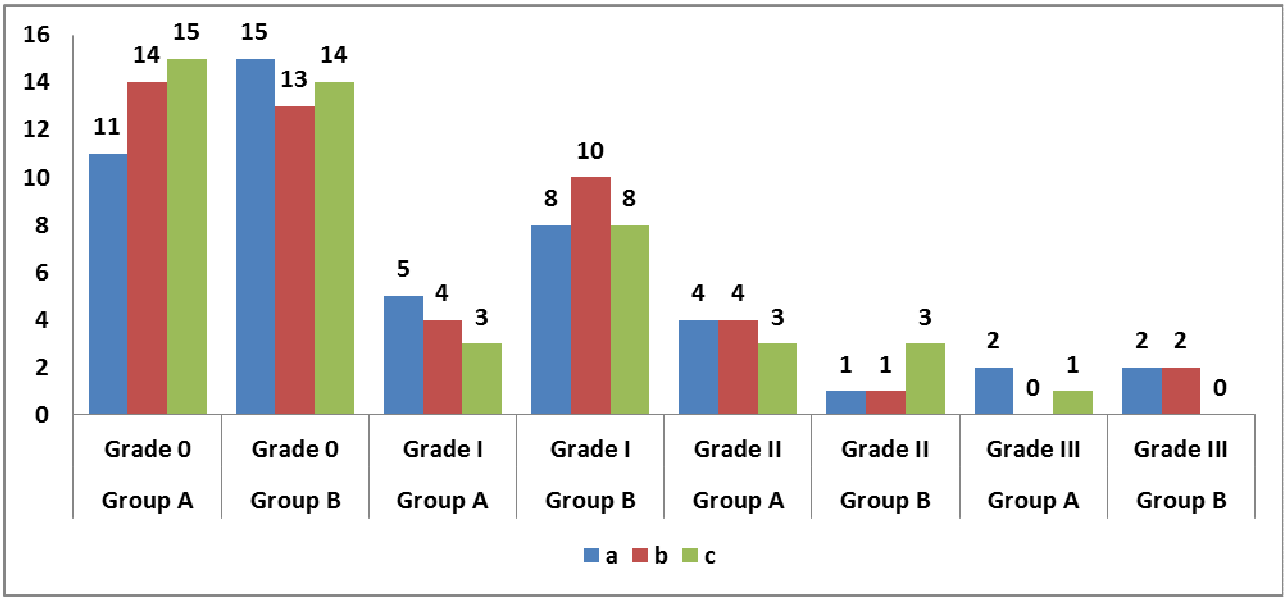
eliminating accumulated and vitiated doshas and malas. As the results are significant at both time points (b and c), it suggests that YG3 has its bhedana action not limited for short period ie upto time point b, but persists for longer duration (till time point c).

Reduction in Udarshool / Yakrut Pradesh shool in group B patients is statistically significant when compared between time points b and a (p=0.04), but it is not significant when compared between time – points c and a. This indicates short term efficacy of YG2 on Udarshool / Yakrut Pradesh shool.

Table 28 – Showing gradation of Yakrutplihavrudhi

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade I**		**Grade II**		**Grade III**	
A	11	15	5	8	4	1	2	2
B	14	13	4	10	4	1	0	2
C	15	14	3	8	3	3	1	0

Graph 13 – Showing gradation of Yakrutpliha vrudhi



Yakrutpleehavruddhi is the major symptom of Yakrut vikara. In our study it is observed that Yakrutpleehavruddhi was not present in 15 patients of group A at time – point c, which was present in 11 patients at time-point a. At the same time in group B, 14 patients did not have Yakrutpleehavruddhi at time – point c, which was present in 15 patients at time-point a.

Grade I Yakrutpleehavruddhi was present in 5 patients of group A, which remained in 3 patients at time-point c. Equal number of patients showed grade I Yakrutpleehavruddhi in group B at time points a and c.

In group A and B, grade II Yakrutpleehavruddhi was seen in 4 and 3 patients and 1 and 3 patients at time points a and c respectively.

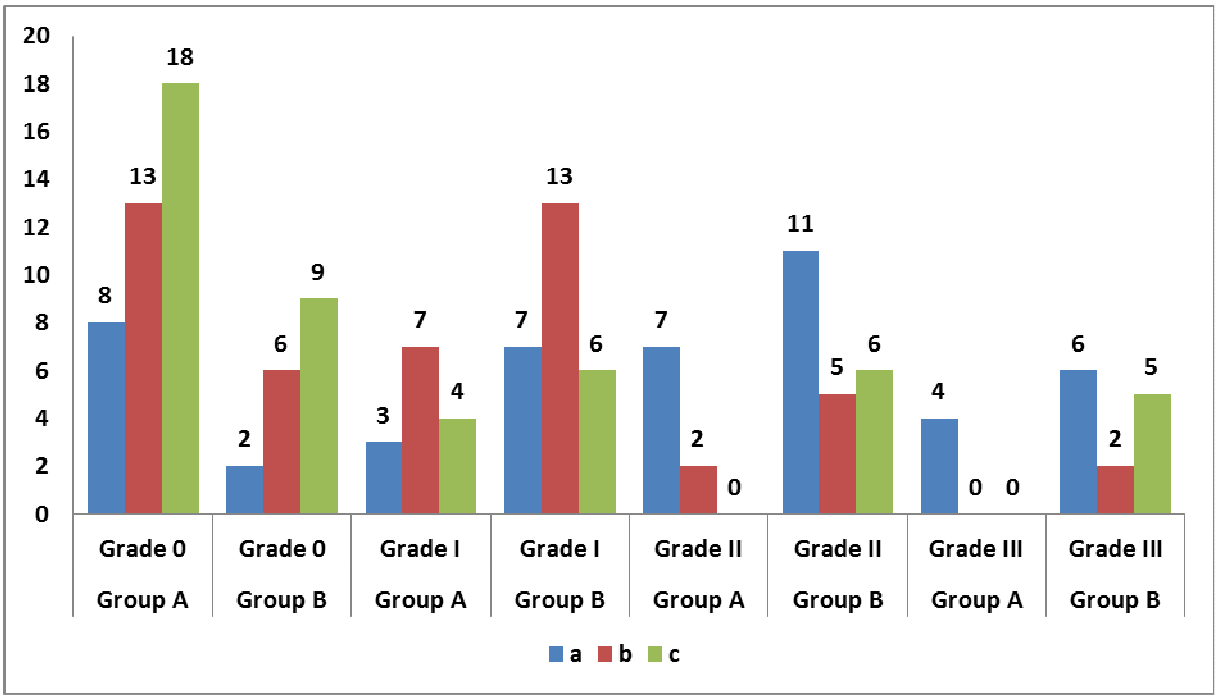
Grade III Yakrutpleehavruddhi, which is a huge enlargement of liver and spleen, was initially present in 2 patients of group A and persisted in 1 patient at time point c. In group B patients it showed declining trend from 2 to 0 at time-points a and c.

Statistical analysis at time points b and a is very significant ($p=0.0067$) and significant ($p=0.01$) at time points c and a for group A.

Table 29 – Showing gradation of Agnimandya

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	8	2	3	7	7	11	4	6
B	13	6	7	13	2	5	0	2
C	18	9	4	6	0	6	0	5

Graph 14 – Showing gradation of Agnimandya



8 patients of group A and 2 patients of group B had normal agni at time point a. At time point c, number of patients increased to 18 and 9 in group A and B respectively, who had normal agni.

Grade I Agnimandya was seen in 3 patients of group A at time-point a and 4 patients at time point c. In group B it was observed in 7 patients at time-point a and 6 patients at time point c.

Grade II agnimandya was present in 7 patients of group A at time point a, while none of the patient was suffering from agnimandya at the end of treatment with YG3. In group B, 11 and 6 patients had grade II agnimandya before and at the end of treatment.

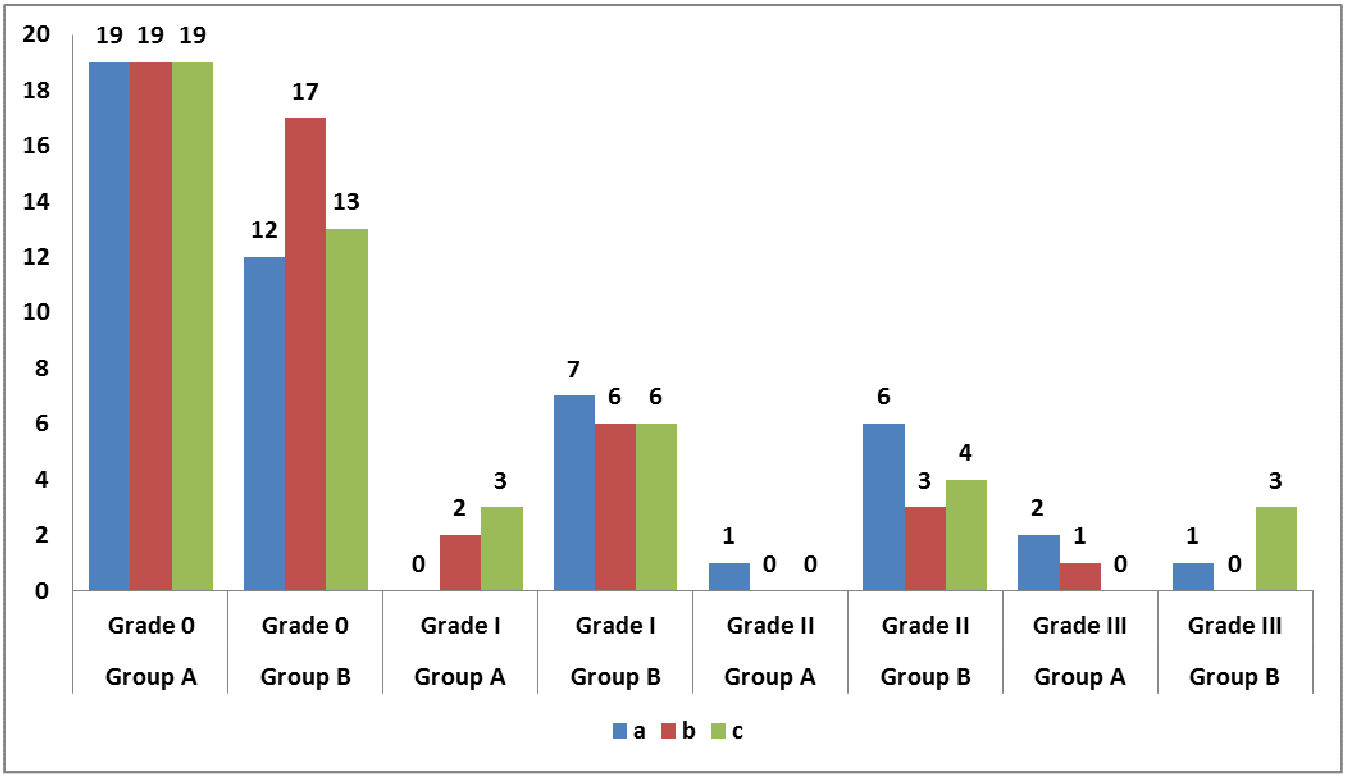
4 patients in group A had grade III agnimandya in the beginning of treatment, while none of the patient had grade III agnimandya at the end of the treatment in this group. On the other hand, 6 and 5 patients of group B had grade III agnimandya in the beginning and at the end of treatment respectively.

There is an extremely significant improvement in agnimandya in group A patients at time point c ($p<0.0001$) and very significant improvement at time point b ($p=0.006$).

Table 30 – Showing gradation of Aruchi

Time points	Number of patients							
	Study group	Control	Study group	Control	Study group	Control	Study group	Control
	Grade 0		Grade i		Grade ii		Grade iii	
A	19	12	0	7	1	6	2	1
B	19	17	2	6	0	3	1	0
C	19	13	3	6	0	4	0	3

Graph 15 – Showing gradation of Aruchi



19 patients in group A did not suffer from Aruchi before starting treatment. Numbers of patients remain same at the end of treatment in this group. 12 patients in group B did not suffer at time point a, while 13 patients in this group did not have Aruchi at time point c.

None of the patient had grade I Aruchi in group A at time-point a, while 3 patients suffered from it at time point c. In group B, 7 patients had grade I Aruchi at time point a, while 6 patients had grade I Aruchi at time point c.

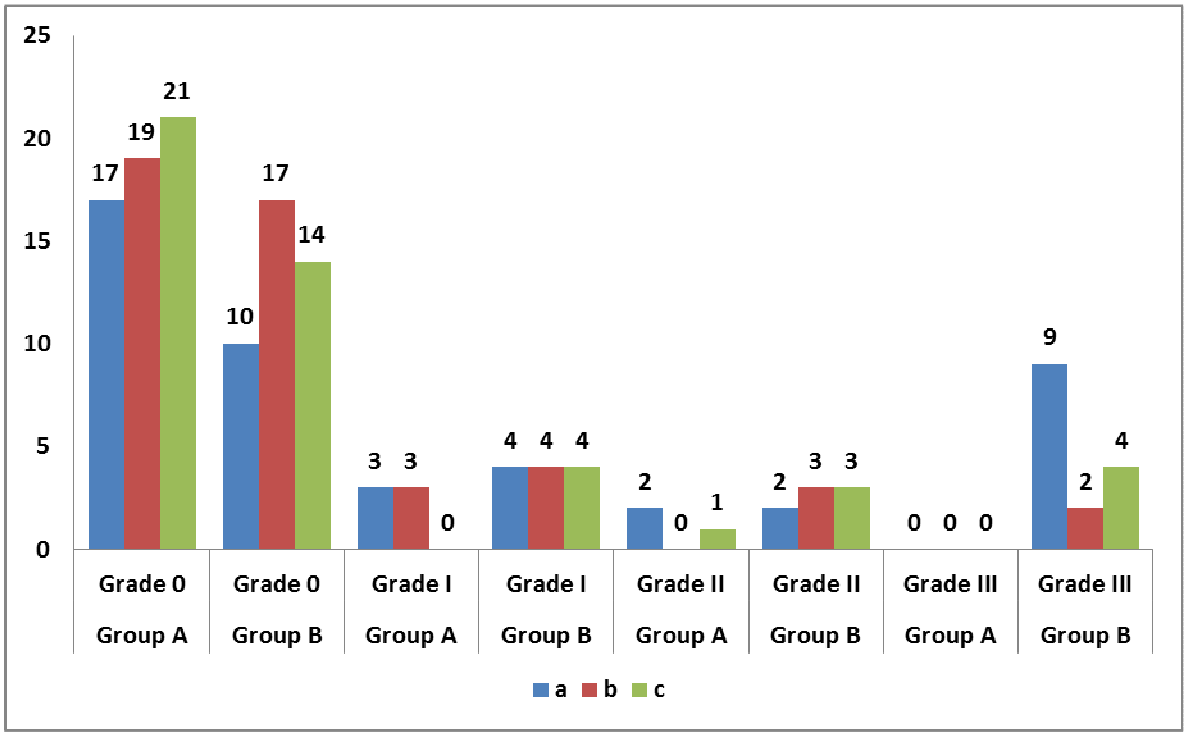
Grade II Aruchi was seen in 1 patient in group A at time-point a and none of the patient had grade II Aruchi at the end of the treatment. 6 patients were suffering from grade II Aruchi at time –point a in group B, while at time point c only 4 patients were suffering from it in this group.

Grade III Aruchi was seen in 2 patients of group A in the beginning of treatment, while none of the patient suffered from it at the end of treatment. 1 patient at time point a and 3 patients at time point c had grade III Aruchi in group B.
Role of YG3 is not found statistically significant in Aruchi.

Table 31 – Showing gradation of Hrullas

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	17	10	3	4	2	2	0	9
B	19	17	3	4	0	3	0	2
C	21	14	0	4	1	3	0	4

Graph 16 – Showing gradation of Hrullas



17 patients in group A did not suffer from Hrullas before starting treatment. Numbers of patients increased to 21 at the end of treatment in this group. 10 patients in group B did not suffer at time point a, while 14 patients in this group did not have Hrullas at time point c.

3 patients had grade I Hrullas in group A at time-point a, while none of the patients suffered from it at time point c. In group B, 4 patients had grade I Hrullas at time point a and c.

Grade II Hrullas was seen in 2 patients in group A at time-point a and 1 patient had grade II Hrullas at the end of the treatment. 2 patients were suffering from grade II Hrullas at time –point a in group B, while at time point c, 3 patients were suffering from it in this group.

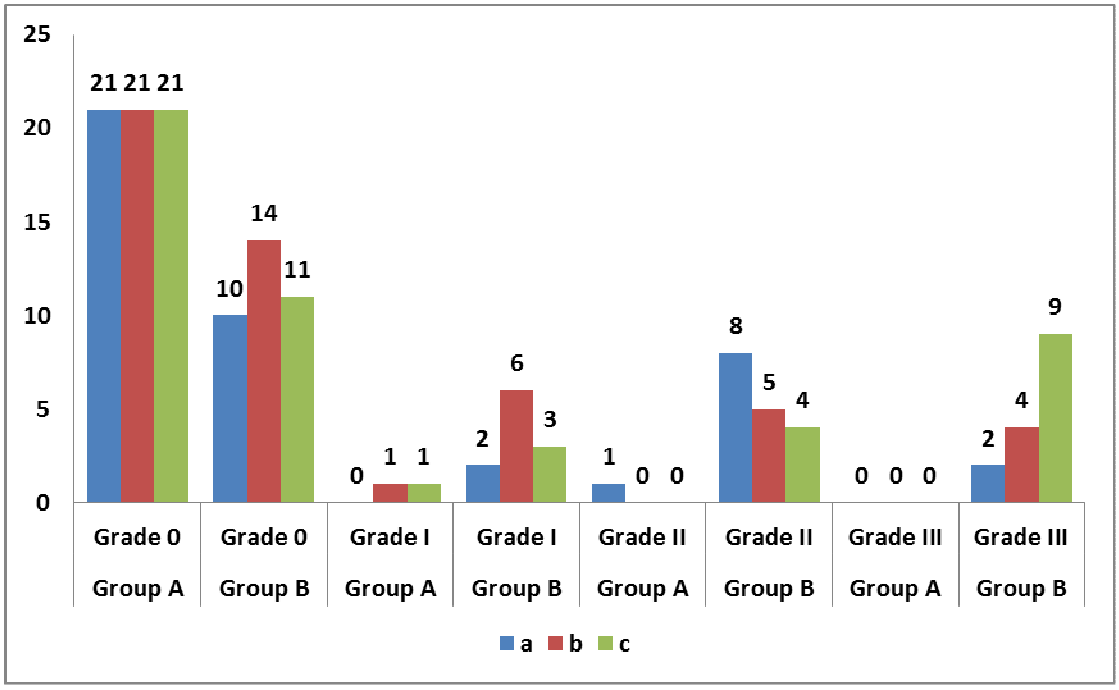
Grade III Hrullas was not seen in any patient of group A in the beginning of treatment and at the end of treatment. 9 patient at time point a and 4 patients at time point c had grade III Hrullas in group B.

YG2 is found to be effective in relieving Hrullas statistically at time point b.

Table 32 – Showing gradation of Sharira Shaithilya

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	21	10	0	2	1	8	0	2
B	21	14	1	6	0	5	0	4
C	21	11	1	3	0	4	0	9

Graph 17 – Showing gradation of Sharira Shaithilya



21 patients in group A did not suffer from Sharira Shaithilya before and after starting treatment. 10 patients in group B did not suffer from it at time point a, while 11 patients in this group did not have Sharira Shaithilya at time point c.

None of the patient had grade I Sharira Shaithilya in group A at time-point a, while 1 patient suffered from it at time point c. In group B, 2 patients had grade I Sharira Shaithilya at time point a, while 3 patients had grade I Sharira Shaithilya at time point c.

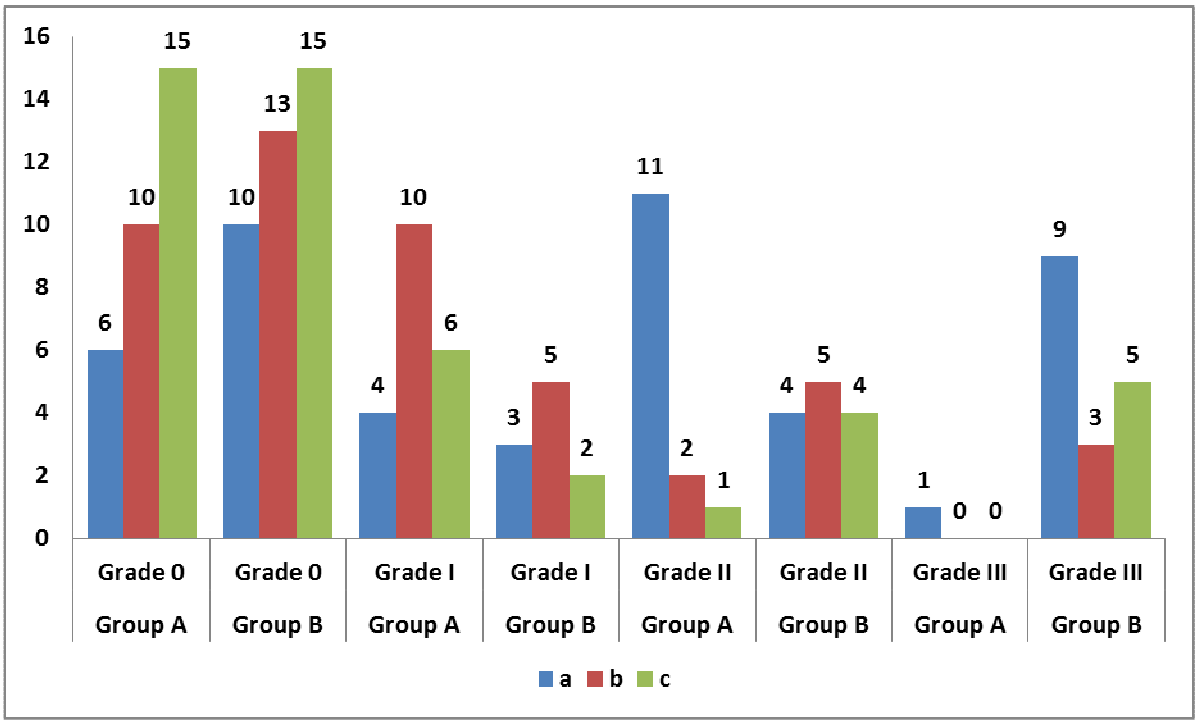
Grade II Sharira Shaithilya was seen in 1 patient in group A at time-point a and none of the patient had grade II Sharira Shaithilya at the end of the treatment. 8 patients were suffering from grade II Sharira Shaithilya at time –point a in group B, while at time point c only 4 patients were suffering from it in this group.

Grade III Sharira Shaithilya was seen any patient at time points a, b and c in group A. 2 patients at time point a and 9 patients at time point c had grade III Sharira Shaithilya in group B.

Table 33– Showing gradation of Avipaka

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	6	10	4	3	11	4	1	9
B	10	13	10	5	2	5	0	3
C	15	15	6	2	1	4	0	5

Graph 18 – Showing gradation of Avipaka



6 patients in group A did not suffer from Avipaka before starting treatment and 15 patients in this group did not have Avipaka at the end of treatment. 10 patients in group B did not suffer at time point a, while 15 patients in this group did not have Avipaka at time point c.

4 patients had grade I Avipaka in group A at time-point a, while 6 patients suffered from it at time point c. In group B, 3 patients had grade I Avipaka at time point a, while 2 patients had grade I Avipaka at time point c.

Grade II Avipaka was seen in 11 patients in group A at time-point a and only 1 patient had grade II Avipaka at the end of the treatment. 4 patients were suffering from grade II Avipaka at time –point a and c in group B.

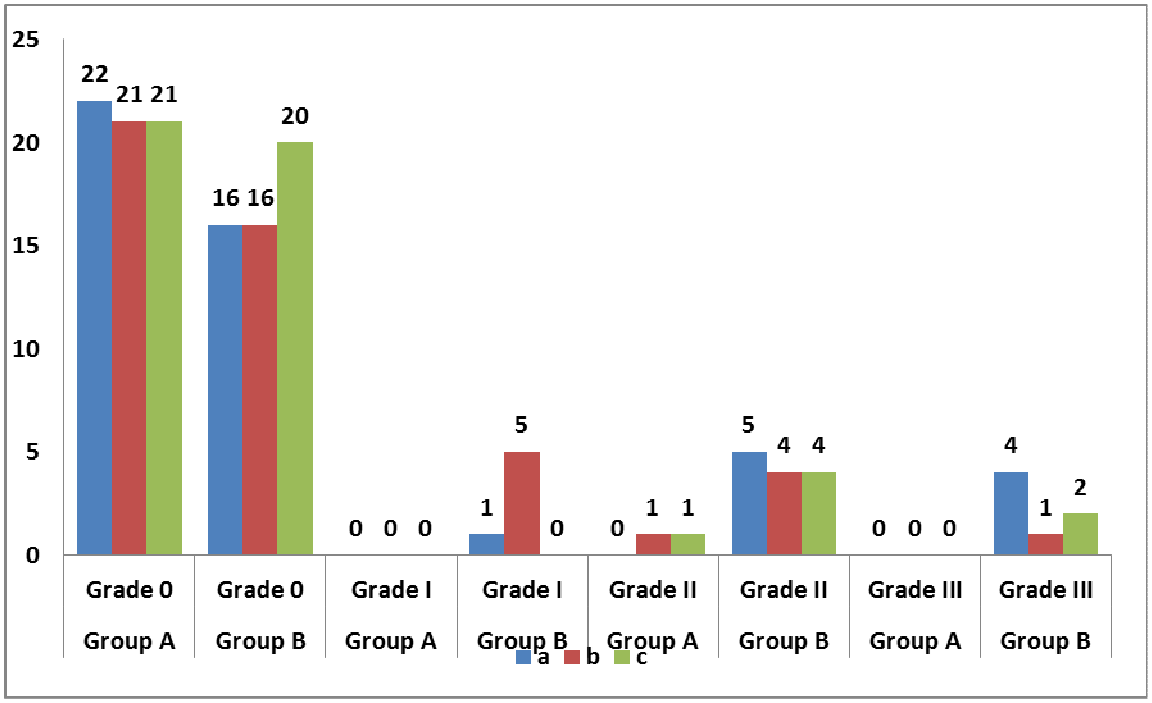
Grade III Avipaka was seen in 1 patient of group A in the beginning of treatment, while none of the patient suffered from it at the end of treatment. 9 patients at time point a and 5 patients at time point c had grade III Avipaka in group B.

Role of YG3 is found to be statistically significant in Avipaka at time points b and c (p=0.008 at b and p=0.0002 at c)

Table 34 – Showing gradation of Daha

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	22	16	0	1	0	5	0	4
B	21	16	0	5	1	4	0	1
C	21	20	0	0	1	4	0	2

Graph 19 – Showing gradation of Daha



22 patients in group A did not suffer from Daha before starting treatment and 21 patients in this group did not have Daha at the end of treatment. 16 patients in group B did not suffer at time point a, while 20 patients in this group did not have Daha at time point c.

None of the patients had grade I Daha in group A at time-points a, b and c. In group B, 1 patient had grade I Daha at time point a, while none of the patient had grade I Daha at time point c.

Grade II Daha was not seen in any patient in group A at time-point a and only 1 patient had grade II Daha at the end of the treatment.5 patients were suffering from grade II Daha at time –point a and 4 patients at time point c in group B.

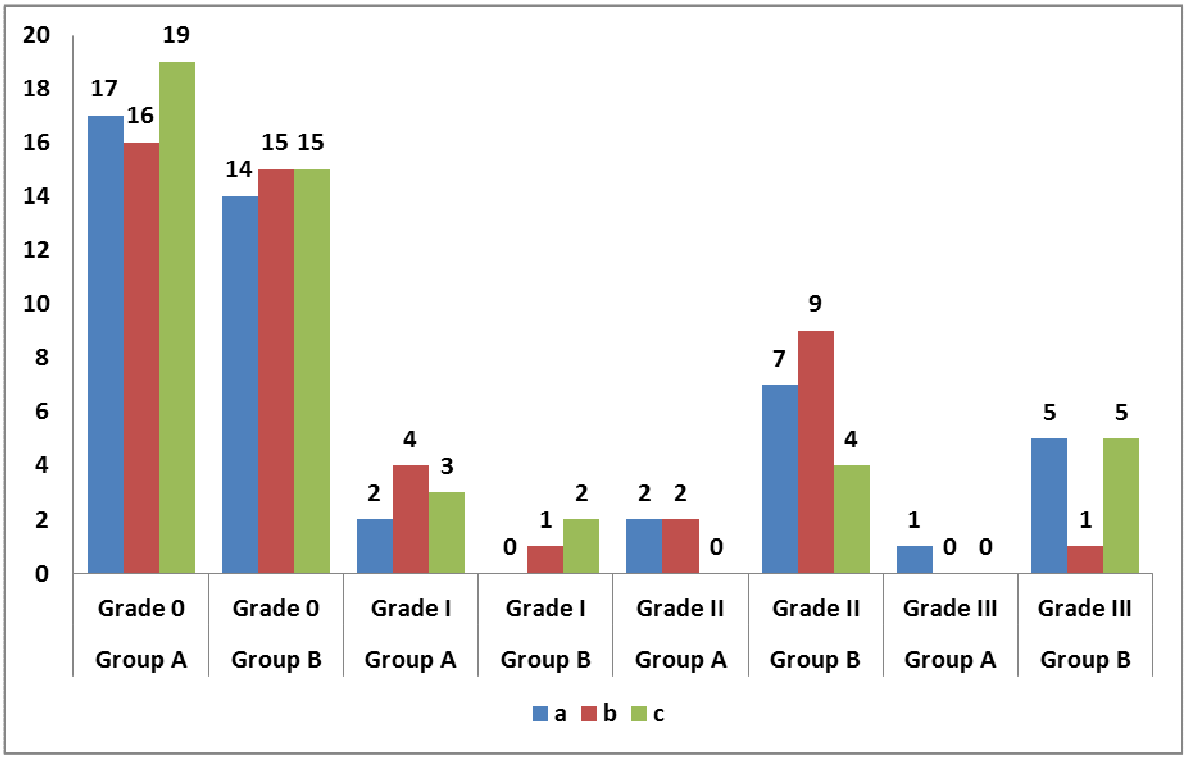
Grade III Daha was not seen in any patient of group A in the beginning and at the end of treatment. 4 patients at time point a and 2 patients at time point c had grade III Daha in group B.

Role of YG3 is found to be statistically significant in Daha at time point c (p=0.04)

Table 35 – Showing gradation of Hatendriya

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	17	14	2	0	2	7	1	5
B	16	15	4	1	2	9	0	1
C	19	15	3	2	0	4	0	5

Graph 20 – Showing gradation of Hatendriya



17 patients in group A did not suffer from Hatendriya before starting treatment and 19 patients in this group did not have Hatendriya at the end of treatment. 14 patients in group B did not suffer at time point a, while 15 patients in this group did not have Hatendriya at time point c.

2 patients had grade I Hatendriya in group A at time-point a, while 3 patients had Hatendriya at time point b. In group B, none of the patient had grade I Hatendriya at time point a, while 2 patients had grade I Hatendriya at time point c.

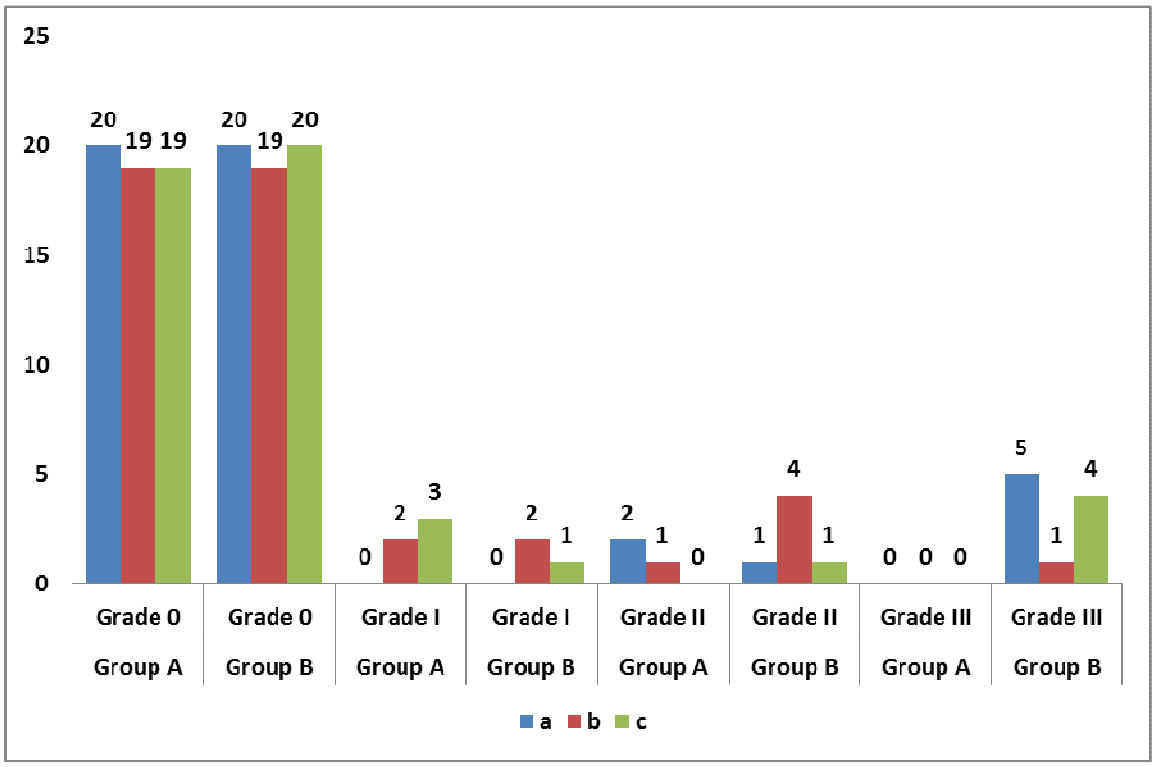
Grade II Hatendriyatva was seen in 2 patients in group A at time-point a and none of the patient had grade II Hatendriya at the end of the treatment. 7 patients were suffering from grade II Hatendriya at time –point a and 4 patients at time point c in group B.

Grade III Hatendriyatva was seen in 1 patient of group A in the beginning of the treatment and none of the patient had Hatendriyatva at the end of treatment. 5 patients each at time points a and c had grade III Hatendriya in group B.

Table 36 – Showing gradation of Bhekavarnata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	20	20	0	0	2	1	0	5
B	19	19	2	2	1	4	0	1
C	19	20	3	1	0	1	0	4

Graph 21 – Showing gradation of Bhekavarnata



20 patients in group A did not suffer from Bhekavarnata before starting treatment and 19 patients in this group did not have Bhekavarnata at the end of treatment. 20 patients each in group B did not suffer at time point a and c.

None of the patient had grade I Bhekavarnata in group A at time-point a, while 3 patients had Bhekavarnata at time point b. In group B, none of the patient had grade I Bhekavarnata at time point a, while 1 patient had grade I Bhekavarnata at time point c.

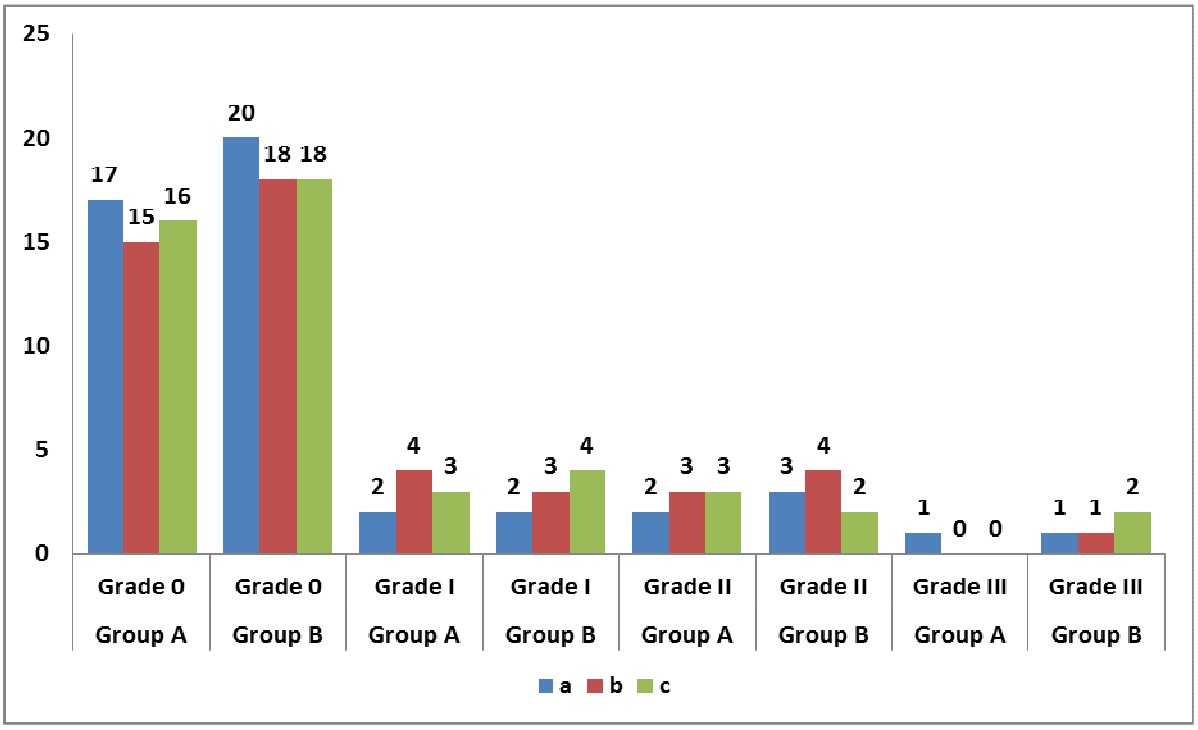
Grade II Bhekavarnatatva was seen in 2 patients in group A at time-point a and none of the patient had grade II Bhekavarnata at the end of the treatment. 1 patient each was suffering from grade II Bhekavarnata at time –points a and c in group B.

Grade III Bhekavarnatatva was not seen in any patient of group A in the beginning and end of the treatment. 5 patients had grade III Bhekavarnata at time points a and 4 patients had this symptom at time point c in group B.

Table 37 – Showing gradation of Shithila Mala Pravrutti

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	17	20	2	2	2	3	1	1
B	15	18	4	3	3	4	0	1
C	16	18	3	4	3	2	0	2

Graph 22 – Showing gradation of Shithila Mala Pravrutti



17 patients in group A did not suffer from Shithila Mala Pravrutti before starting treatment and 16 patients in this group did not have Shithila Mala Pravrutti at the end of treatment. 20 and 18 patients at time points a and c respectively, in group B were not suffering from Shithila Mala Pravrutti.

2 patients had grade I Shithila Mala Pravrutti in group A at time-point a, while 3 patients had Shithila Mala Pravrutti at time point b. In group B, 2 patients had grade I Shithila Mala Pravrutti at time point a, while 4 patients had grade I Shithila Mala Pravrutti at time point c.

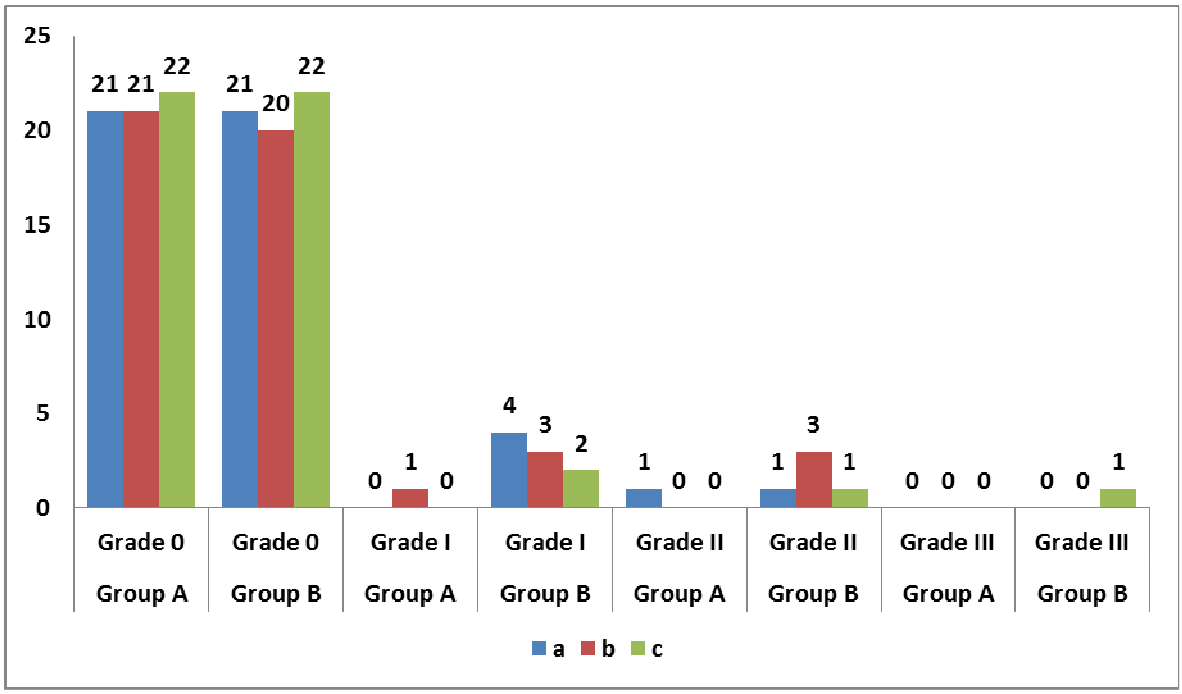
Grade II Shithila Mala Pravruttitva was seen in 2 patients in group A at time-point a and 3 patients had grade II Shithila Mala Pravrutti at the end of the treatment. 3 patients were suffering from grade II Shithila Mala Pravrutti at time –points a and 2 patients at time point c in group B.

Grade III Shithila Mala Pravruttitva was seen in 1 patient of group A in the beginning and in none of the patient at the end of the treatment. 1 patient had grade III Shithila Mala Pravrutti at time point a and 2 patients had this symptom at time point c in group B.

Table 38 – Showing gradation of Netra Pitata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	21	21	0	4	1	1	0	0
B	21	20	1	3	0	3	0	0
C	22	22	0	2	0	1	0	1

Graph 23 – Showing gradation of Netra Pitata



21 patients in group A did not have Netra Pitata before starting treatment and 22 patients in this group did not have Netra Pitata at the end of treatment. 21 and 22 patients at time points a and c respectively, in group B did not show Netra Pitata.

None of the patient had grade I Netra Pitata in group A at time-points a and c. In group B, 4 patients had grade I Netra Pitata at time point a, while 2 patients had grade I Netra Pitata at time point c.

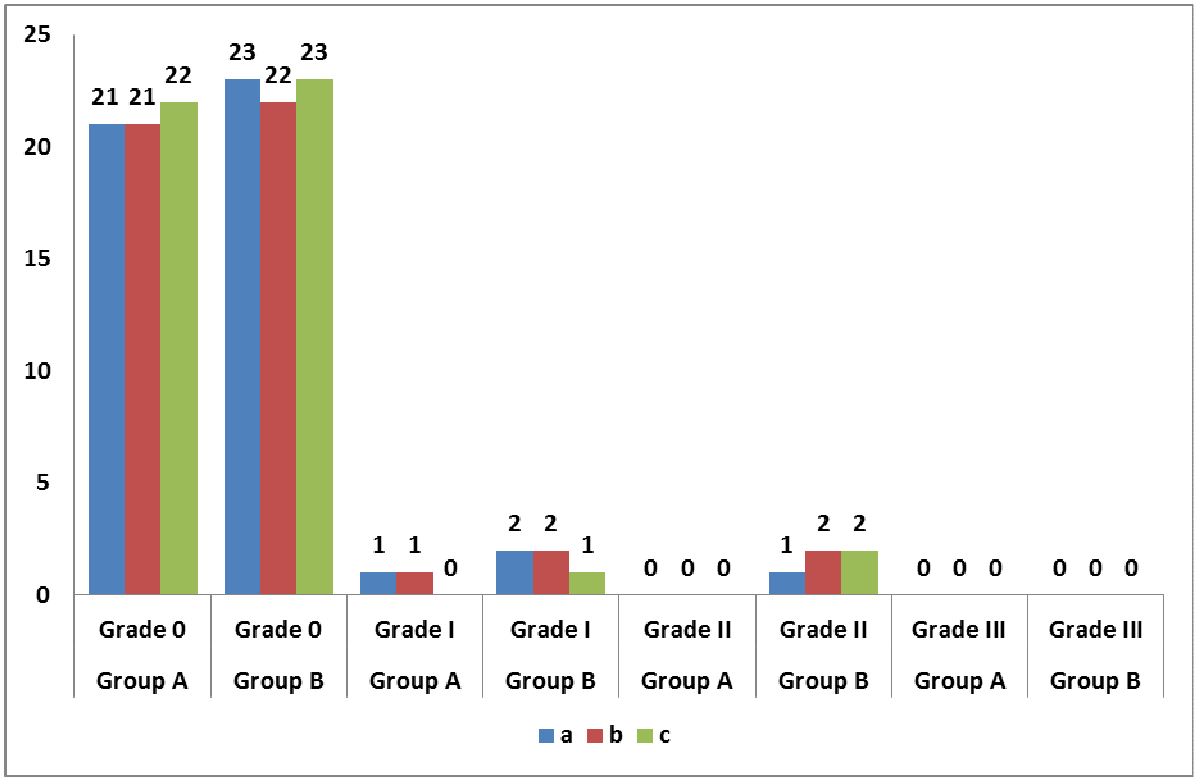
Grade II Netra Pitatatva was seen in 2 patients in group A at time-point a and none of the patient had grade II Netra Pitata at the end of the treatment. 1 patient each was suffering from grade II Netra Pitata at time –points a and c in group B.

Grade III Netra Pitatatva was not seen in a single patient of group A in the beginning and in none of the patient at the end of the treatment. No patient was presented with Netra Pitata at time point a and 1 patient had grade III Netra Pitata at time point c in group B.

Table 39 – Showing gradation of Twak Pitata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	21	23	1	2	0	1	0	0
B	21	22	1	2	0	2	0	0
C	22	23	0	1	0	2	0	0

Graph 24 – Showing gradation of Twak Pitata



21 patients in group A did not have Twak Pitata before starting treatment and 22 patients in this group did not have Twak Pitata at the end of treatment. 23 patients each at time points a and c respectively, in group B did not show Twak Pitata.

1 patient had grade I Twak Pitata in group A at time-points a and none of the patient had this symptom at time point c. In group B, 2 patients had grade I Twak Pitata at time point a, while 1 patient had grade I Twak Pitata at time point c.

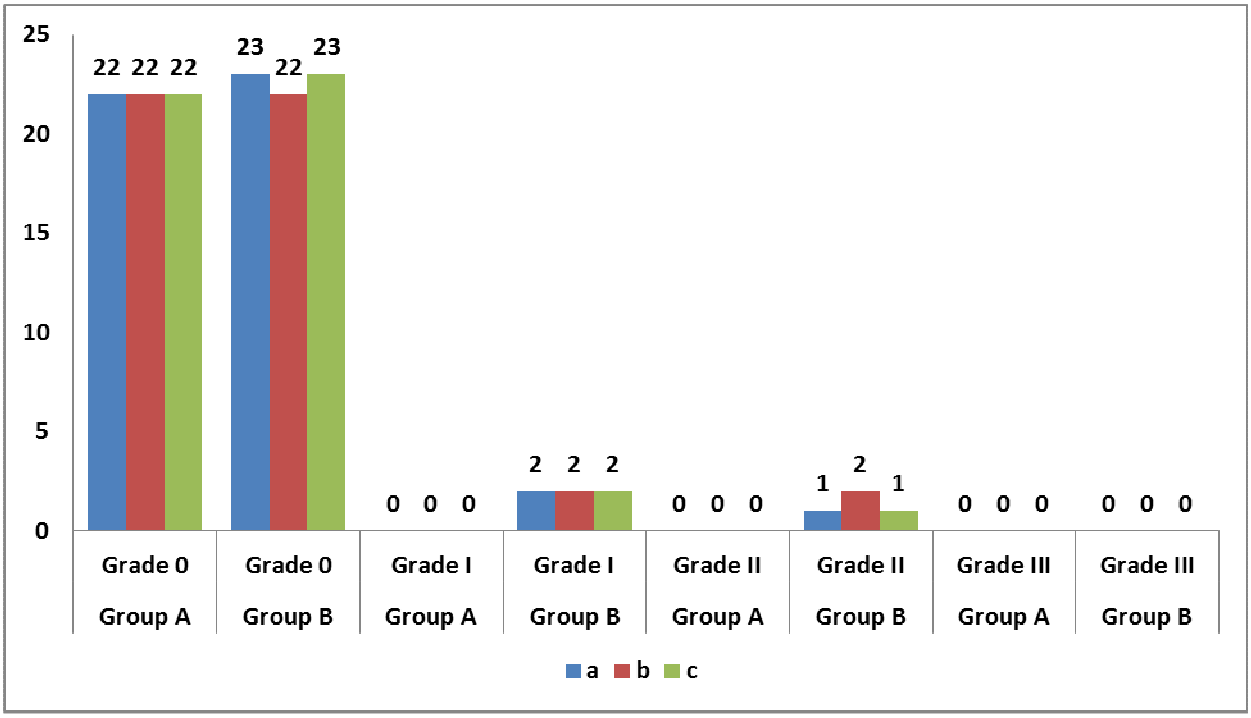
Grade II Twak Pitatatva was not seen in a single patient in group A at time-points a and c. 1 patient at time point a and 2 patients at time point c were.

Grade III Twak Pitatatva was not seen in a single patient of group A and group B in the beginning and at the end of the treatment.

Table 40 – Showing gradation of Nakha pitata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	22	23	0	2	0	1	0	0
B	22	22	0	2	0	2	0	0
C	22	23	0	2	0	1	0	0

Graph 25 – Showing gradation of Nakha pitata



22 patients of group A and 23 patients of group B did not have Nakha Pitata before starting and at the end of treatment.

None of the patient at time point a and c had grade I Nakha Pitata in group A. In group B, 2 patients each had grade I Nakha Pitata at time points a and c.

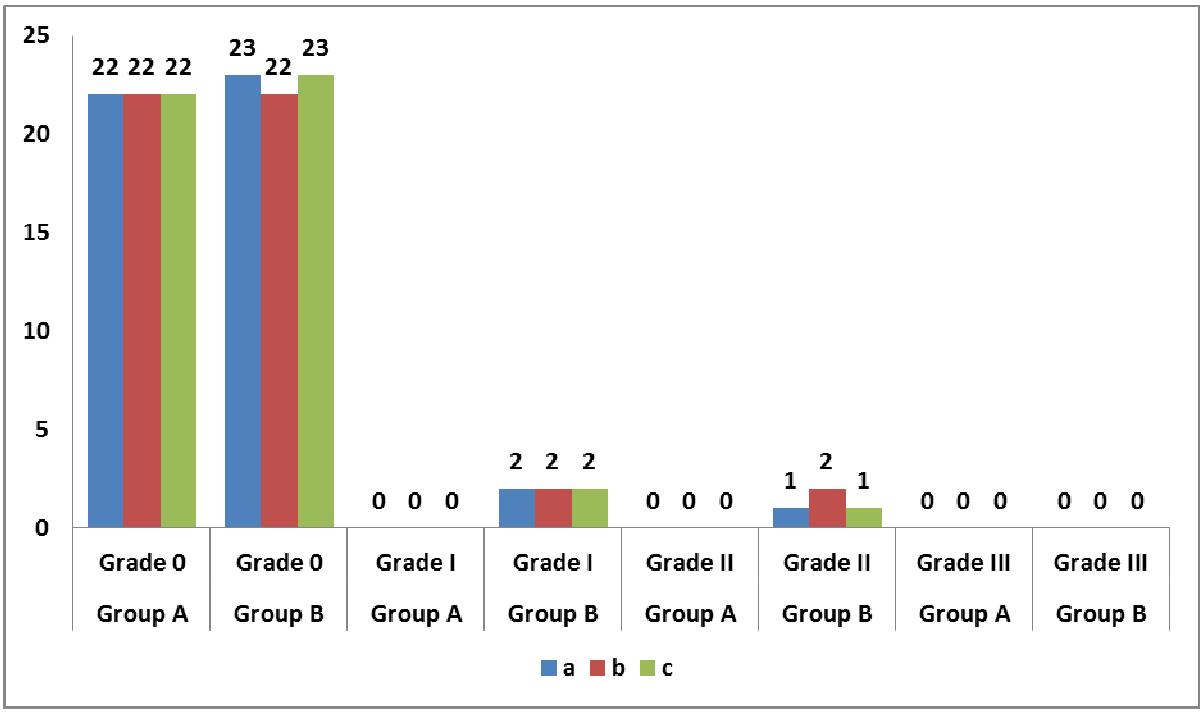
Grade II Nakha Pitata was not seen in a single patient in group A at time-points a and c. 1 patient each at time point a and c had Nakha Pitata in group B.

Grade III Nakha Pitata was not seen in a single patient of group A and group B in the beginning and at the end of the treatment.

Table 41 – showing gradation of Mukh Pitata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	22	23	0	2	0	1	0	0
B	22	22	0	2	0	2	0	0
C	22	23	0	2	0	1	0	0

Graph 26 – Showing gradation of Mukh Pitata



22 patients each of group A and 23 patients each of group B did not have Mukha Pitata before starting and at the end of treatment.

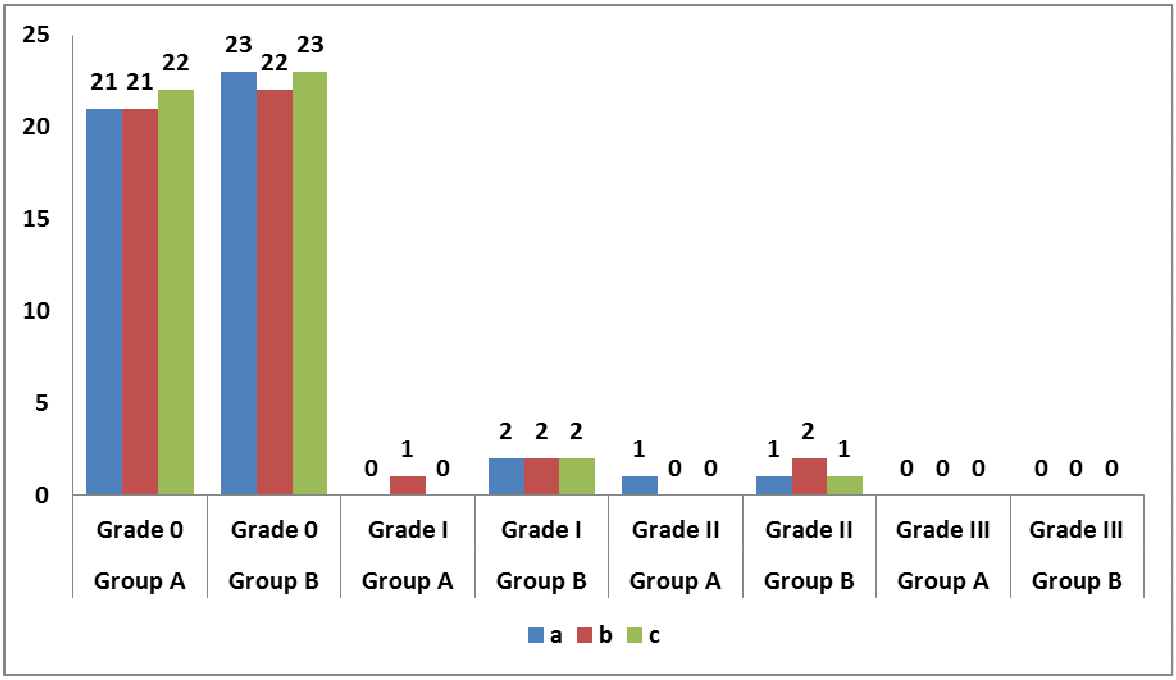
None of the patient at time point a and c had grade I, II and III Mukha Pitata in group A. None of the patient had grade III Mukha Pitata at time point a and c in group B.

In group B, 2 and 1 patients each had grade I and II Mukha Pitata respectively at time points a and c.

Table 42 – Showing gradation of Malapitata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	21	23	0	2	1	1	0	0
B	21	22	1	2	0	2	0	0
C	22	23	0	2	0	1	0	0

Graph 27 – Showing gradation of Malapitata



21 patients in group A did not have Mala Pitata before starting treatment and 22 patients in this group did not have Mala Pitata at the end of treatment. 23 patients each at time points a and c respectively, in group B did not show Mala Pitata.

None of the patient had grade I Mala Pitata in group A at time-points a and c. 2 patients each had this symptom at time points a and c in group B.

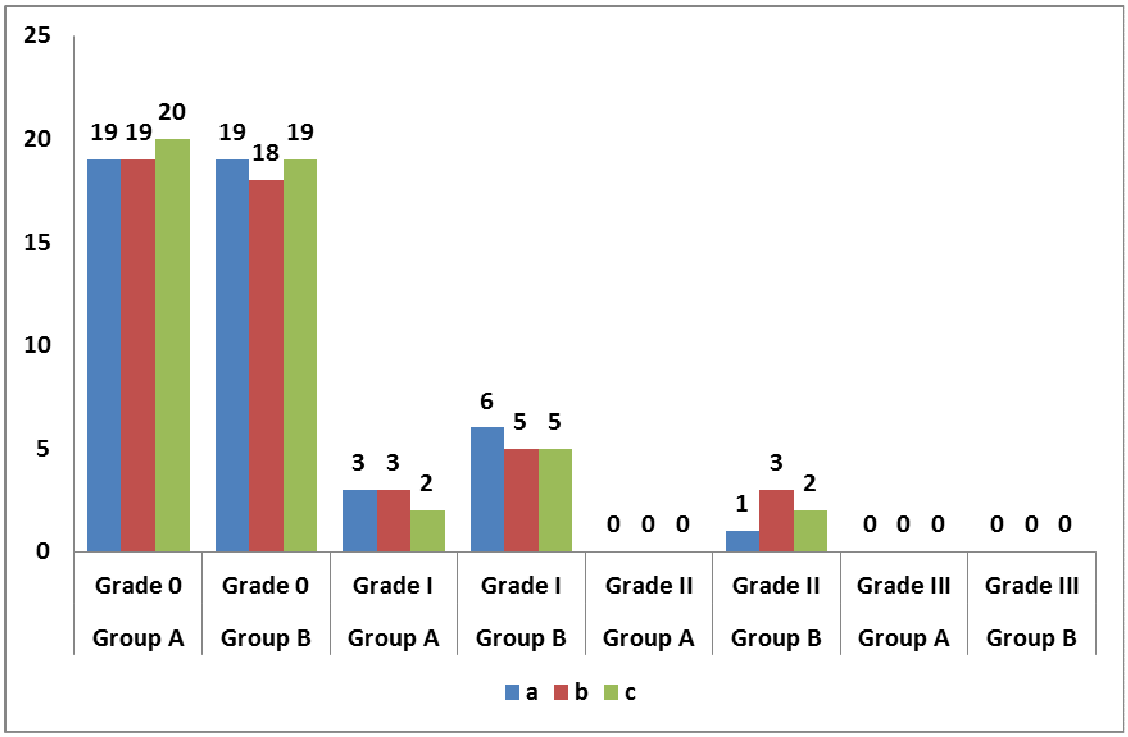
Grade II Mala Pitatatva was seen in a single patient in group A at time-points a and in none of the patient at time point c. 1 patient each at time point a and c had Mala Pitata in group B.

Grade III Mala Pitatatva was not seen in a single patient of group A and group B in the beginning and at the end of the treatment.

Table 43 – Showing gradation of Mutra Pitata

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	19	19	3	6	0	1	0	0
B	19	18	3	5	0	3	0	0
C	20	19	2	5	0	2	0	0

Graph 28 – Showing gradation of Mutra Pitata



19 patients in group A did not have Mutra Pitata before starting treatment and 20 patients in this group did not have Mutra Pitata at the end of treatment. 19 patients each at time points a and c respectively, in group B did not show Mutra Pitata.

3 patient at time point a and 2 patients at time point c had grade I Mutra Pitata in group A. 6 patients at time point a and 5 patients at time point c had grade I Mutra Pitata in group B.

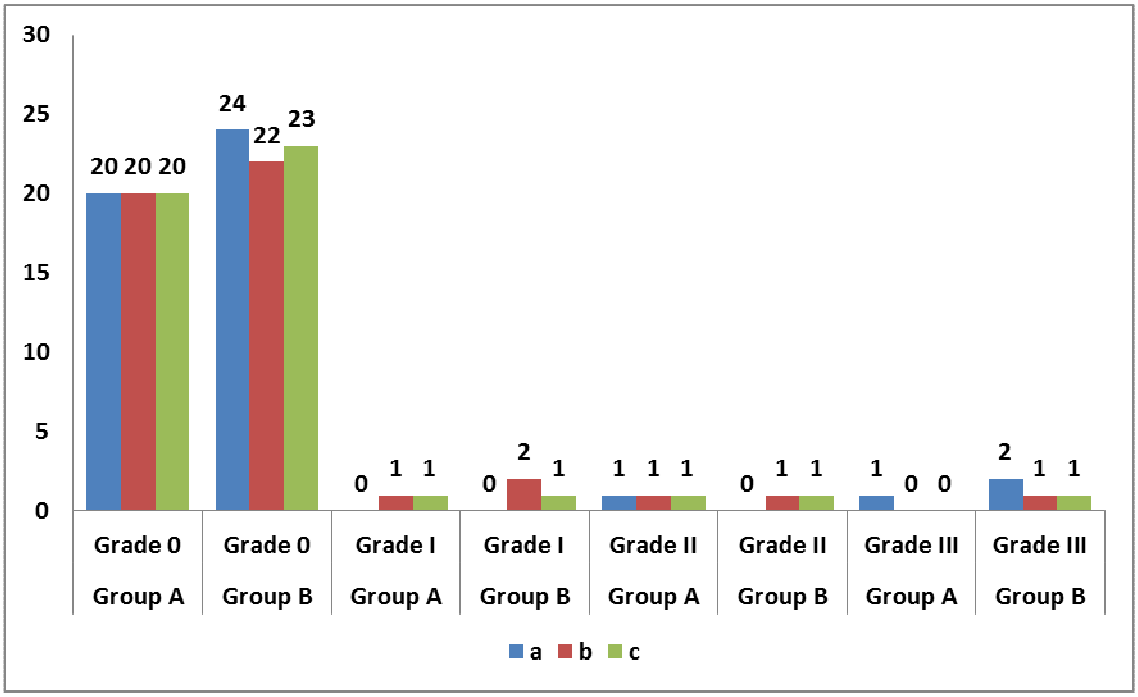
Grade II Mutra Pitatatva was not seen in a single patient in group A at time-points a and c. 1 patient of group B at time point a and 2 patients at time point c had grade II Mutra Pitata.

Grade III Mutra Pitatatva was not seen in a single patient of group A and group B in the beginning and at the end of the treatment.

Table 44 – showing gradation of Jaundice

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	20	24	0	0	1	0	1	2
B	20	22	1	2	1	1	0	1
C	20	23	1	1	1	1	0	1

Graph 29 – Showing gradation of Jaundice



20 patients each in group A did not have Jaundice before starting treatment and at the end of treatment. 24 patients at time point a and 23 at time point c in group B did not show Jaundice.

None of the patient at time point a and 1 patient at time point c had grade I Jaundice in group A. None of the patient at time point a and 1 patient at time point c had grade I Jaundice in group B.

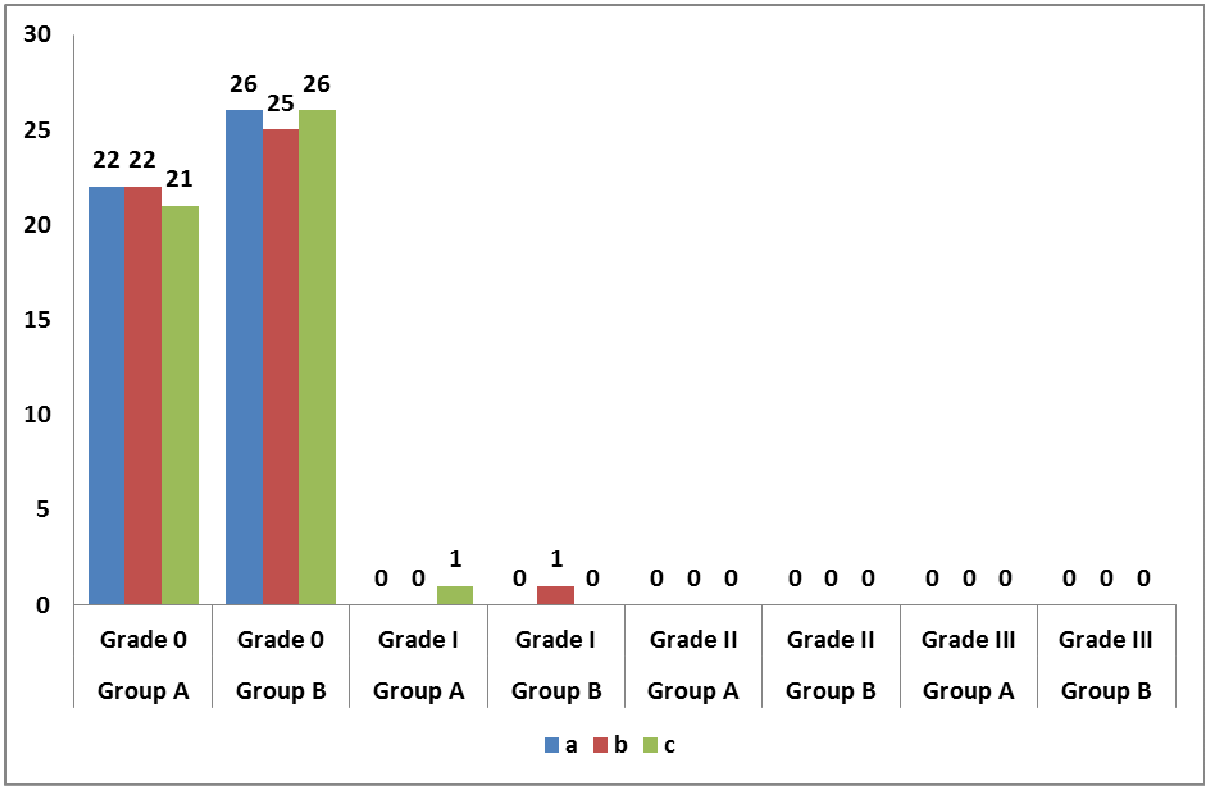
Grade II Jaundice was seen in a single patient in group A at time-points a and c. None of the patient of group B at time point a and 1 patient at time point c had grade II Jaundice.

Grade III Jaundice was seen in a single patient of group A at time point a and in none of the patient at time point c. In group B 2 patients had Jaundice in the beginning and 1 patient at the end of the treatment.

Table 45 – Showing gradation of Heamatemasis

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	22	26	0	0	0	0	0	0
B	22	25	0	1	0	0	0	0
C	21	26	1	0	0	0	0	0

Graph 30 – Showing gradation of Heamatemasis



22 and 21 patients in group A did not have Haematemasis before starting treatment and at the end of treatment respectively. 26 patients each at time points a and c in group B did not show Haematemasis.

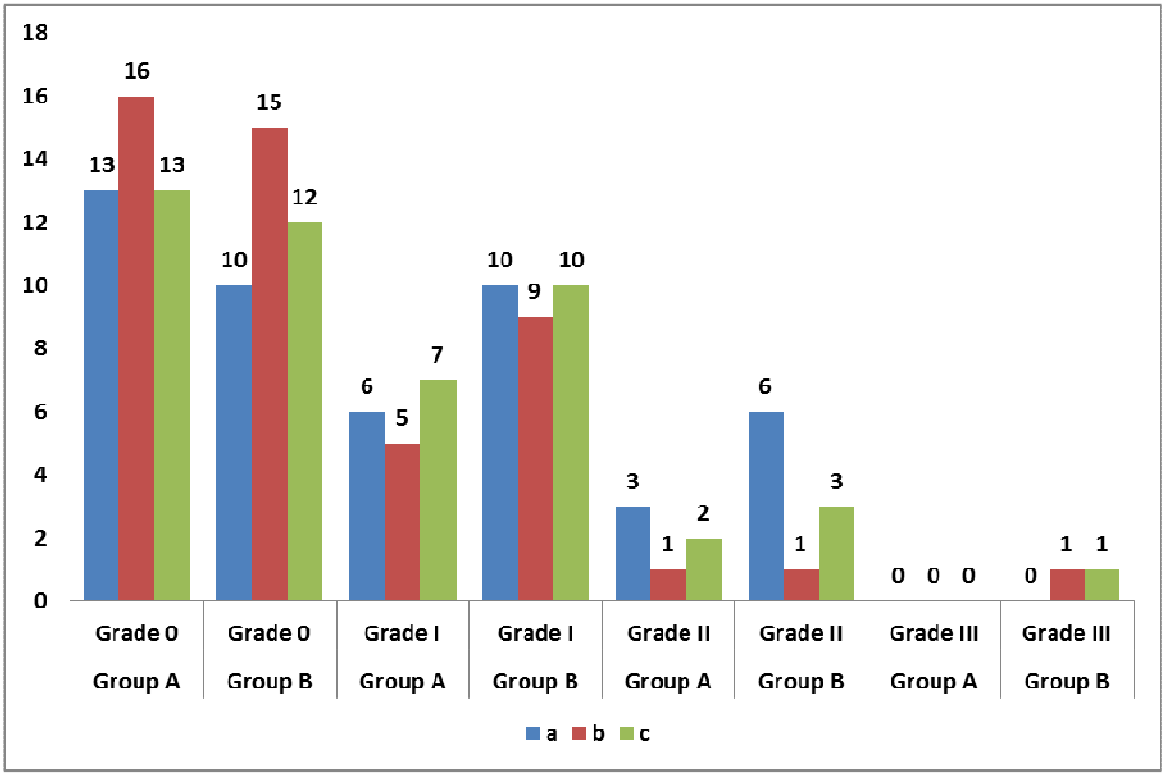
None of the patient at time point a and 1 patient at time point c had grade I Haematemasis in group A. None of the patient at time point a and c had grade I Haematemasis in group B.

Grade II and III Haematemasis was not seen in any patient in group A and B at time-points a and c.

Table 46 – Showing gradation of Weight loss

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	13	10	6	10	3	6	0	0
B	16	15	5	9	1	1	0	1
C	13	12	7	10	2	3	0	1

Graph 31 – Showing gradation of Weight loss



13 patients each in group A did not have Weight loss before starting treatment and at the end of treatment respectively. 10 patients at time point a and 12 at time point c in group B did not show Weight loss.

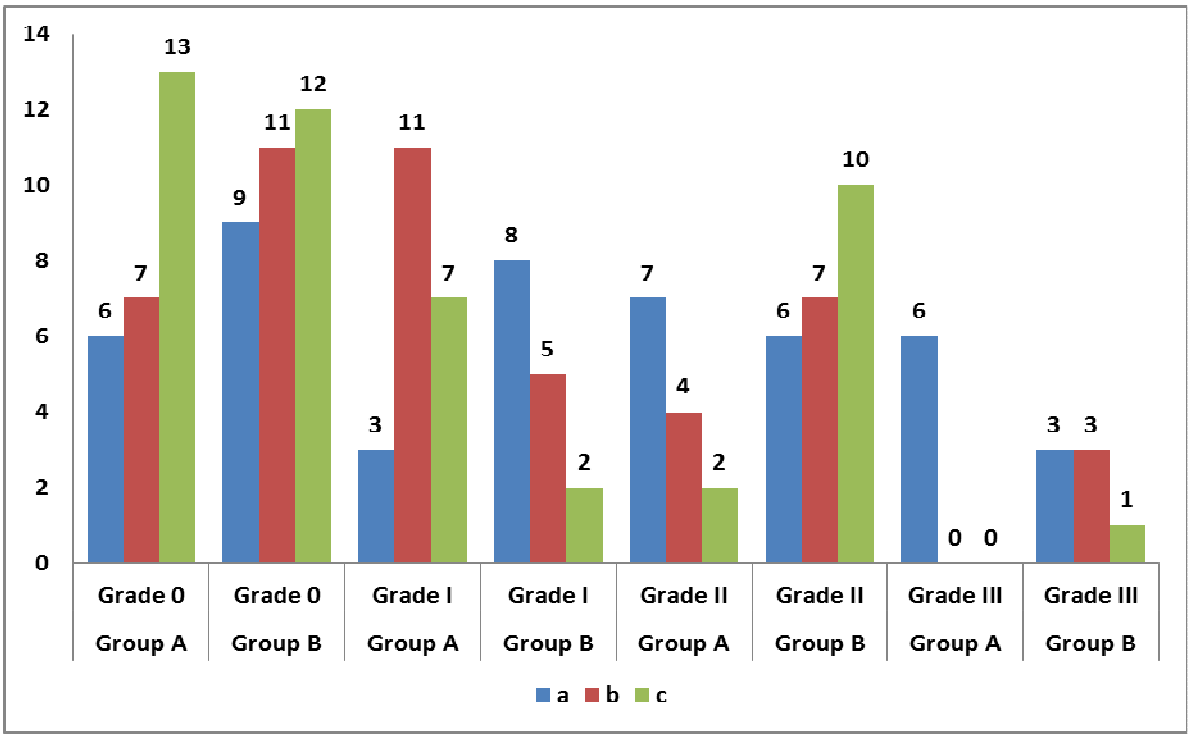
6 patients at time point a and 7 patients at time point c had grade I Weight loss in group A. 10 patients each at time point a and c had grade I Weight loss in group B. Grade II Weight loss was seen in 3 patients in group A at time-point a and in 2 patients at time point c. 6 patients of group B at time point a and 3 patients at time point c had grade II Weight loss.

Grade III Weight loss was not seen in a single patient of group A at time points a and c. In group B none of the patient had Weight loss in the beginning and 1 patient at the end of the treatment.

Table 47 – Showing gradation of Full ness

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	6	9	3	8	7	6	6	3
B	7	11	11	5	4	7	0	3
C	13	12	7	2	2	10	0	1

Graph 32 – Showing gradation of Full ness



6 patients in group A did not have Fullness before starting treatment and 13 patients at the end of treatment. 9 patients at time point a and 12 at time point c in group B did not show Fullness.

3 patients at time point a and 7 patients at time point c had grade I Fullness in group A. 8 patients at time point a and 2 patients at time point c had grade I Fullness in group B.

Grade II Fullness was seen in 7 patients in group A at time-point a and in 2 patients at time point c. 6 patients of group B at time point a and 10 patients at time point c had grade II Fullness.

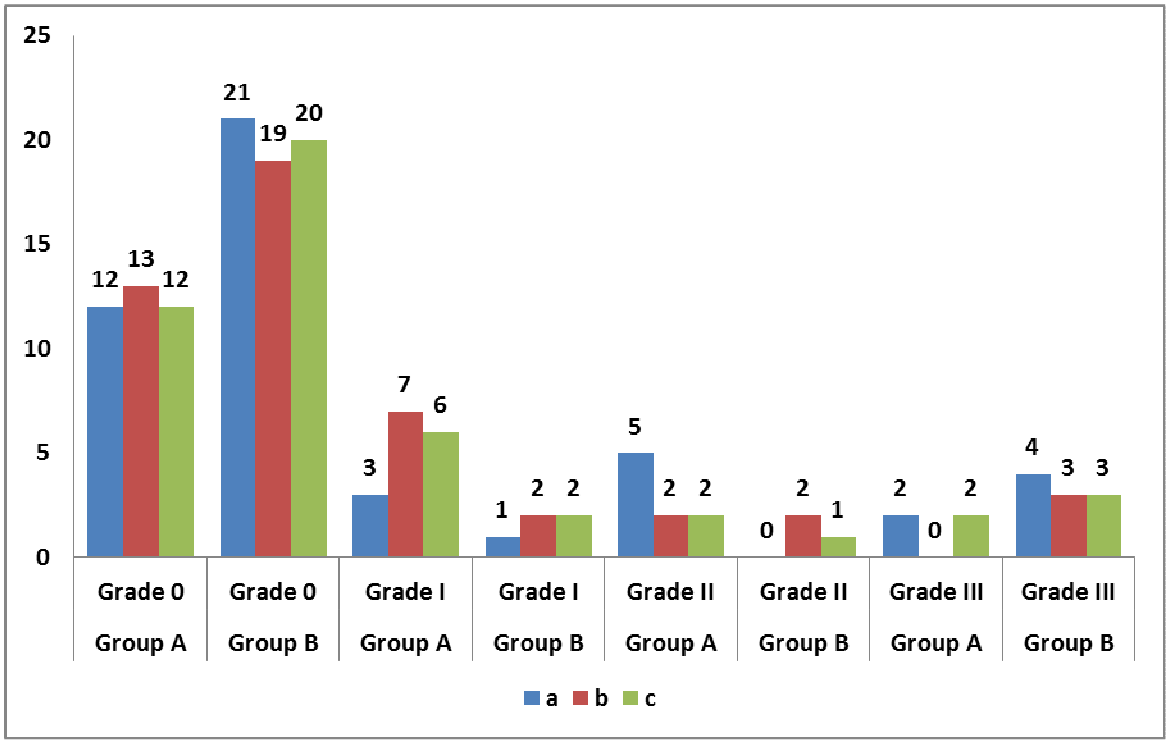
Grade III Fullness was seen in 6 patients of group A at time points a and in none of the patient at time point c. In group B 3 patients had Fullness in the beginning and 1 patient at the end of the treatment.

Statistically extremely significant results are obtained in group A patients for the symptom fullness when studied at time point c (p=0.0005) and significant results at time point b (p=0.017). Very significant results are obtained when analysis was carried out in group A patients at time points b with (p=0.003) and c with a (p=0.0011).

Table 48 – Showing gradation of Abdominal Swelling

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	12	21	3	1	5	0	2	4
B	13	19	7	2	2	2	0	3
C	12	20	6	2	2	1	2	3

Graph 33 – Showing gradation of Abdominal Swelling



12 patients each in group A did not have Abdominal swelling before and after starting treatment. 21 patients at time point a and 20 at time point c in group B did not show Abdominal swelling.

3 patients at time point a and 6 patients at time point c had grade I Abdominal swelling in group A. 1 patients at time point a and 2 patients at time point c had grade I Abdominal swelling in group B.

Grade II Abdominal swelling was seen in 5 patients in group A at time-point a and in 2 patients at time point c. None of the patient of group B at time point a and 1 patient at time point c had grade II Abdominal swelling.

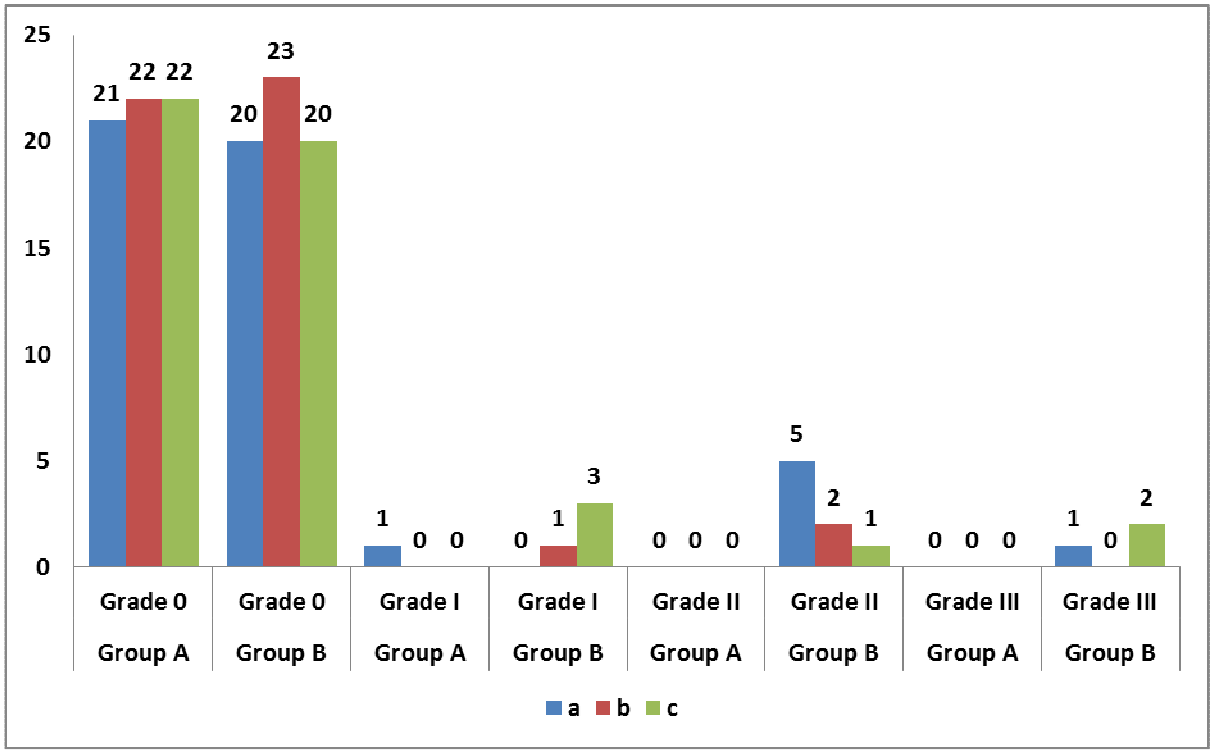
Grade III Abdominal swelling was seen in 2 patients each of group A at time points a and c. In group B 4 patients had Abdominal swelling in the beginning and 3 patients at the end of the treatment.

Very significant improvement in abdominal swelling (p=0.003) is found in group A patients at time point b.

Table 49 – Showing gradation of vomiting

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	21	20	1	0	0	5	0	1
B	22	23	0	1	0	2	0	0
C	22	20	0	3	0	1	0	2

Graph 34 – Showing gradation of vomiting



21 patients at time point a and 22 patients at time point c in group A did not have vomiting. 20 patients each at time point a and c in group B did not suffer vomiting.

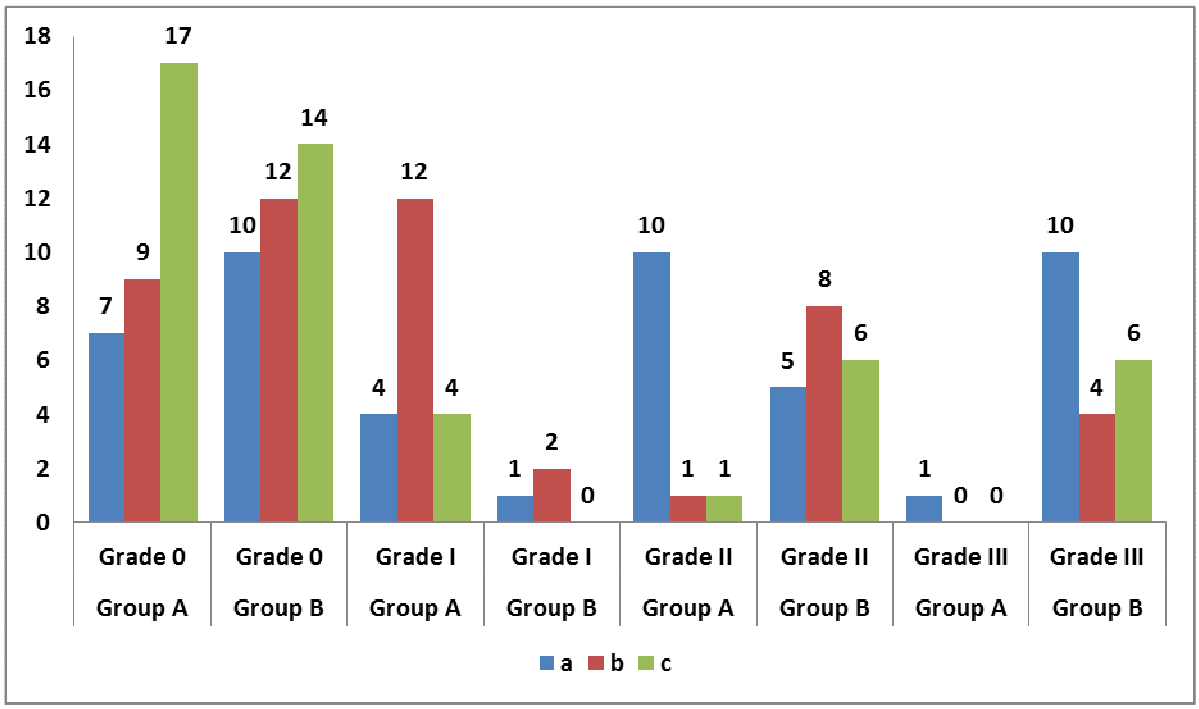
1 patient at time point a and none of the patient at time point c had grade I vomiting in group A. None of the patient at time point a and 3 patients at time point c had grade I vomiting in group B.

Grade II vomiting was not seen in a single patient in group A at time-points a and c. 5 patients of group B at time point a and 1 patient at time point c had grade II Vomiting. Grade III vomiting was seen in none of the patient of group A at time points a and c. In group B 1 patient had vomiting in the beginning and 2 patients at the end of the treatment.

Table 50 – Showing gradation of Malaise

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	7	10	4	1	10	5	1	10
B	9	12	12	2	1	8	0	4
C	17	14	4	0	1	6	0	6

Graph 35 – Showing gradation of Malaise



7 patients at time point a and 17 patients at time point c in group A did not have malaise. 10 patients at time point a and 14 patients at time point c in group B did not suffer malaise.

4 patients each at time points a and c had grade I malaise in group A. 1 patient at time point a and none of the patients at time point c had grade I malaise in group B.

Grade II malaise was seen in 10 patients in group A at time-point a and 1 patient at time point c. 5 patients of group B at time point a and 6 patients at time point c had grade II Malaise.

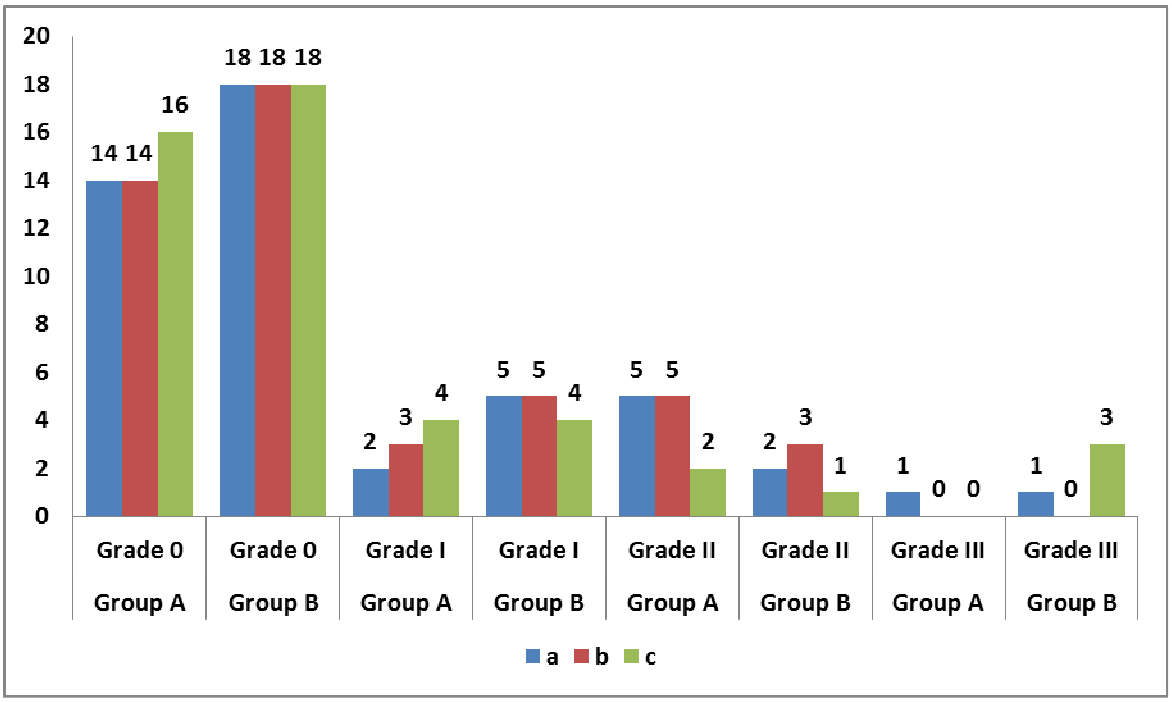
Grade III malaise was seen in 1 patient of group A at time points a and in none of the patient at time point c. In group B 10 patients had malaise in the beginning and 6 patients at the end of the treatment.

Malaise is found to be statistically extremely significant (p=0.0002) at time point c and significant at time point b (p=0.01) in group A.

Table 51 – Showing gradation of Bone pain

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	14	18	2	5	5	2	1	1
B	14	18	3	5	5	3	0	0
C	16	18	4	4	2	1	0	3

Graph 36 – Showing gradation of Bone pain



14 patients at time point a and 16 patients at time point c in group A did not have bone pain. 18 patients each at time point a and c in group B did not suffer bone pain. 2 patients each at time point a and 4 patients at time point c had grade I bone pain in group A. 5 patients at time point a and 4 patients at time point c had grade I bone pain in group B.

Grade II bone pain was seen in 5 patients in group A at time-point a and 2 patients at time point c. 2 patients of group B at time point a and 1 patient at time point c had grade II Bone pain.

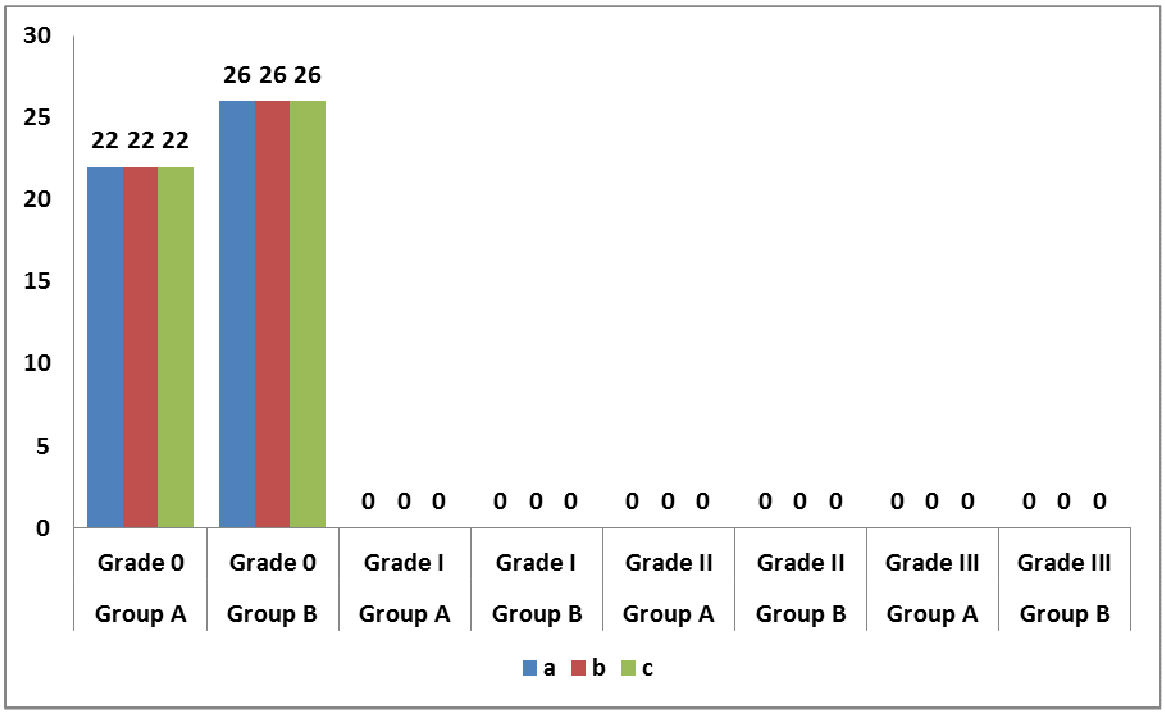
Grade III bone pain was seen in 1 patient of group A at time points a and in none of the patient at time point c. In group B 1 patient had bone pain in the beginning and 3 patients at the end of the treatment.

Efficacy of YG3 on bone pain is found to be statistically significant (p=0.02) at time point c in group A.

Table 52 – Showing gradation of Tremors

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	22	26	0	0	0	0	0	0
B	22	26	0	0	0	0	0	0
C	22	26	0	0	0	0	0	0

Graph 37 – Showing gradation of Tremors



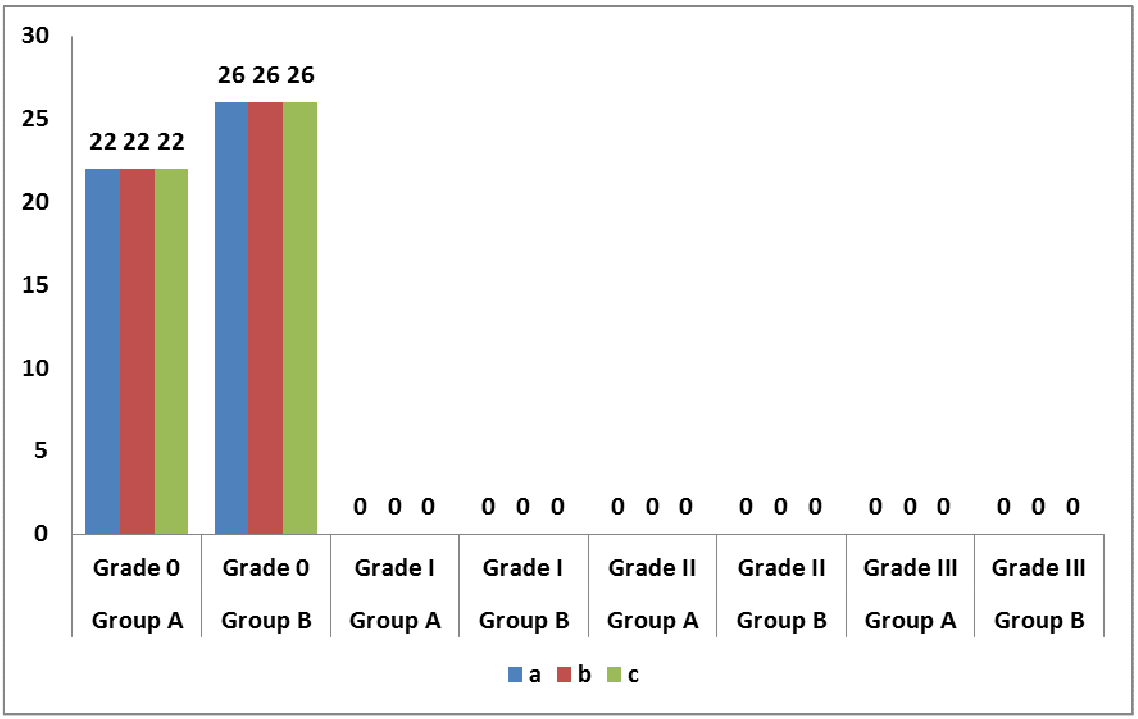
22 patients each at time points a and c in group A did not have tremors. 26 patients each at time points a and c in group B did not have tremors.

None of the patient had grade I, II and III tremors at time points a and c in group A and B.

Table 53 – Showing gradation of Disorientation

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	22	26	0	0	0	0	0	0
B	22	26	0	0	0	0	0	0
C	22	26	0	0	0	0	0	0

Graph 38 – Showing gradation of Disorientation



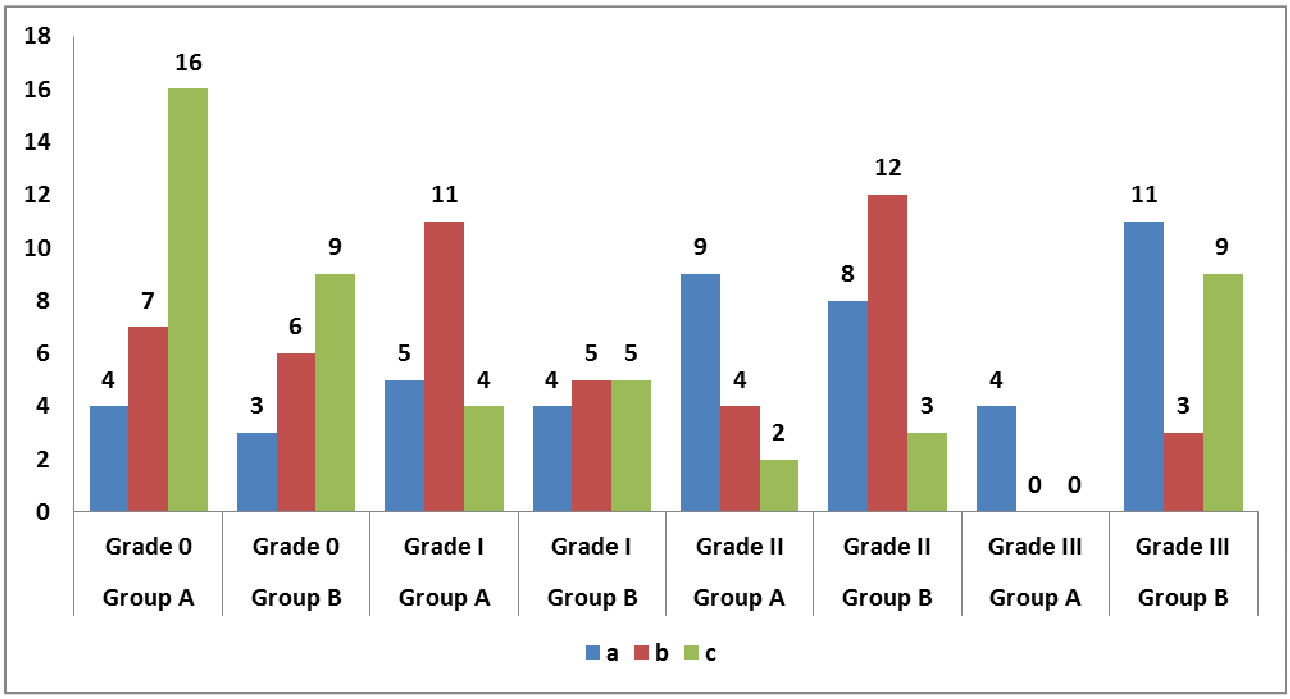
22 patients each at time points a and c in group A did not have disorientation. 26 patients each at time points a and c in group B did not have tremors.

None of the patient had grade I, II and III disorientation at time points a and c in group A and B.

Table 54 – Showing gradation of Weakness

	Number of patients							
Time points	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**	**Study group**	**Control**
	Grade 0		**Grade i**		**Grade ii**		**Grade iii**	
A	4	3	5	4	9	8	4	11
B	7	6	11	5	4	12	0	3
C	16	9	4	5	2	3	0	9

Graph 39 – Showing gradation of Weakness



4 patients at time point a and 16 patients at time point c in group A did not have weakness. 3 patients at time point a and 9 patients at time point c in group B did not suffer from weakness.

5 patients each at time point a and 4 patients at time point c had grade I weakness in group A. 4 patients at time point a and 5 patients at time point c had grade I weakness in group B.

Grade II weakness was seen in 9 patients in group A at time-point a and 2 patients at time point c. 8 patients of group B at time point a and 3 patients at time point c had grade II Weakness.

Grade III weakness was seen in 4 patients of group A at time points a and none of the patient at time point c had weakness. In group B 11 patients had weakness in the beginning and 9 patients at the end of the treatment.

Efficacy of YG3 on weakness is found to be statistically extremely significant ($p<0.0001$) at time point c, very significant ($p=0.008$) at time point b in group A. Effectiveness of YG2 is also statistically significant ($p=0.045$) on weakness in group B.

Statistical Analysis of symptoms of Yakrut Vikara

Table 55 - Group A-Internal Group assessment of Symptoms (Time point c to a)

	Mean of time-point a	**Mean of time – point c**	**SD FOR time point a**	**SD FOR time-point c**	**p**	**SIGNIFICANCE**
Udarshool / Yakrut pradeshi shul	1.09	0.36	0.97	0.58	0.004	Very. Significant
Yakrutplihavrudhi	0.86	0.54	1.03	0.91	0.28	
Agnimandya	1.31	0.18	1.17	0.39	<0.0001	Extremely .Significant
Aruchi	0.36	0.13	0.95	0.35	0.30	
Hrullas	0.31	0.09	0.64	0.42	0.17	
Sharira Shaithilya	0.09	0.04	0.42	0.21	0.65	
Avipaka	1.3	0.3	0.94	0.58	0.0002	Extremely. Significant
Daha	-	-	-	-	-	-
Hatendriya	0.40	0.13	0.85	0.35	0.17	
Bhekavarnata	0.18	0.13	0.58	0.35	0.75	
Shithila Mala Pravrutti	0.40	0.40	0.85	0.730	0.99	
Netra Pitata	-	-	-	-	-	-
Twak Pitata	-	-	-	-	-	-
Nakha Pitata	-	-	-	-	-	-
Mukha Pitata	-	-	-	-	-	-
Mala Pitata	-	-	-	-	-	-
Mutra Pitata	0.13	0.09	0.35	0.29	0.64	
Jaundice	0.22	0.13	0.75	0.46	0.63	

Jaundice	0.22	0.09	0.61	0.29	0.35	
Haematemesis	-	-	-	-	-	
Weight loss	0.54	0.50	0.73	0.67	0.83	
Fullness	1.59	0.50	1.18	0.67	0.0005	Extremely. Significant
Abdominal swelling	0.86	0.72	1.08	0.98	0.66	
Vomiting	-	-	-	-	-	-
Malaise	1.22	0.27	0.97	0.55	0.0002	Extremely. Significant
Bone pain	0.68	0.36	0.99	0.65	0.21	
Tremours	-	-	-	-	-	-
Disorientation	-	-	-	-	-	-
Asthenia / Weakness	1.59	0.36	1.008	0.65	<0.0001	Extremely. Significant

Graph 40 - Group A-Internal Group assessment of Symptoms (Time point c to a)

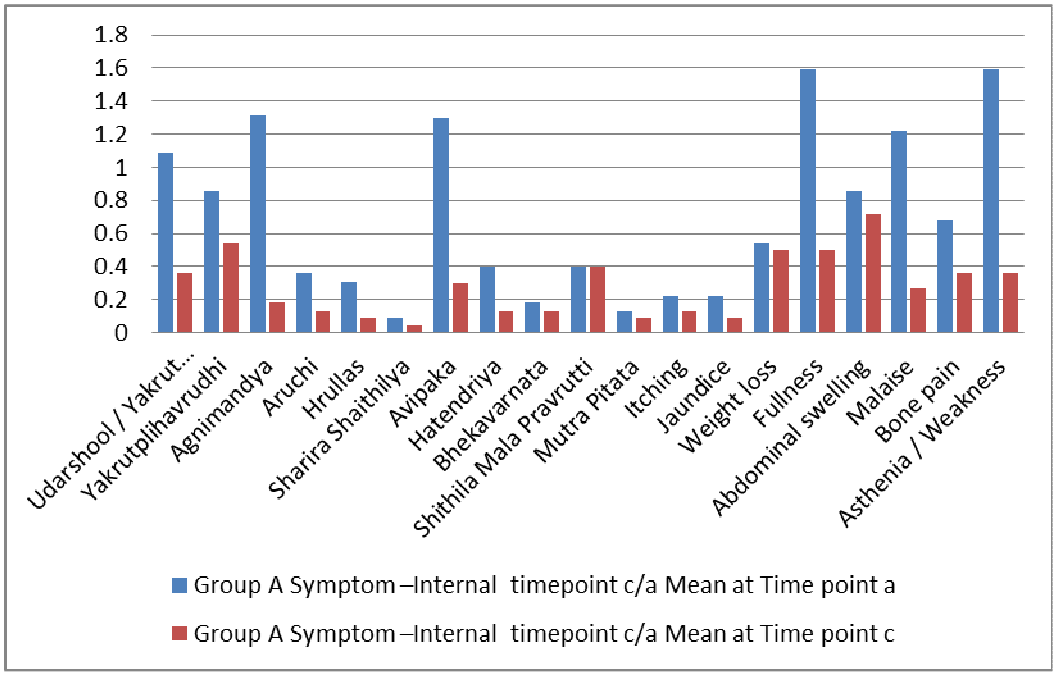


Table 56 - Group A-Internal Group assessment of Symptoms–(Time point b to a)

	Mean of time point a	Mean of time point b	SD FOR time point a	SD FOR time point b	P	SIGNIFICANCE
Udarshool / Yakrut pradeshi shul	1.091	0.5455	0.97	0.59	0.03	Significant
Yakrutplihavrudhi	0.86	0.54	1.03	0.80	0.26	
Agnimandya	1.318	0.500	1.17	0.67	0.006	Very. Significant
Aruchi	0.36	0.22	0.95	0.68	0.58	
Hrullas	0.31	0.13	0.64	0.35	0.25	
Sharira Shaithilya	0.09	0.04	0.42	0.21	0.65	
Avipaka	1.318	0.63	0.941	0.65	0.008	Very. Significant.
Hatendriya	0.40	0.36	0.85	0.65	0.84	
Bhekavarnata	0.18	0.18	0.58	0.58	<0.9	
Shithila Mala Pravrutti	0.40	0.45	0.85	0.73	0.85	
Netra Pitata	0.09	0.04	0.42	0.21	0.65	
Twak Pitata	0.04	0.04	0.21	0.21	>0.9	
Mala Pitata	0.09	0.04	0.42	0.21	0.65	
Mutra Pitata	0.13	0.13	0.35	0.35	>0.9	
Jaundice	0.22	0.13	0.75	0.46	0.63	
Jaundice	0.22	0.13	0.61	0.46	0.58	
Weight loss	0.54	0.31	0.73	0.56	0.25	
Fullness	1.5	0.8	1.18	0.71	0.017	Significant
Abdominal swelling	0.86	0.50	1.08	0.67	0.18	
Malaise	1.22	0.63	0.97	0.58	0.01	Significant
Bone pain	0.68	0.59	0.99	0.85	0.74	
Asthenia / Weakness	1.6	0.85	1.008	0.71	0.008	Very. Significant

Graph 41 -Group A-Internal Group assessment of Symptoms–
(Time point b to a)

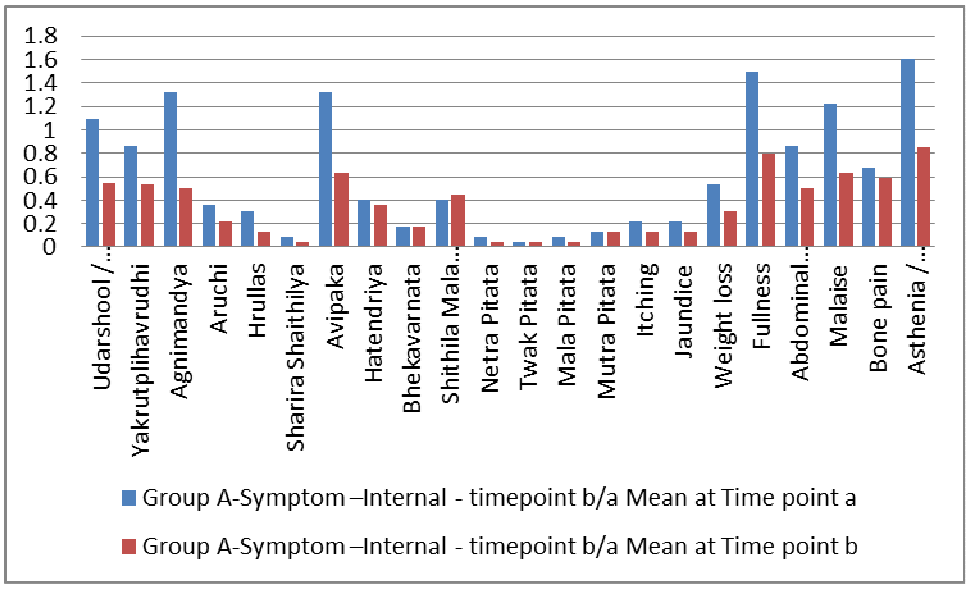


Table 57 - Group B-Internal Group assessment of Symptoms–(Time point b to a)

	Mean of time point a	**Mean of time point b**	**SD FOR time point a**	**SD FOR time point b**	**P**	**SIGNIFICANCE**
Udarshool / Yakrut pradeshi shul	1.53	0.88	1.33	0.95	0.04	Significant
Yakrutplihavrudhi	0.61	0.69	0.89	0.88	0.75	
Agnimandya	1.80	1.11	0.89	0.86	0.006	
Aruchi	0.84	0.46	0.92	0.70	0.098	
Hrullas	1.40	0.61	1.35	0.98	0.02	Significant
Sharira Shaithilya	1.42	0.92	1.3	1.09	0.13	
Avipaka	1.4	0.92	1.33	1.09	0.11	
Daha	0.88	0.61	1.21	0.89	0.36	
Hatendriya	1.115	0.84	1.27	1.04	0.40	
Bhekavarnata	0.65	0.50	1.23	0.90	0.61	
Shithila Mala Pravrutti	0.42	0.53	0.85	0.90	0.63	
Netra Pitata	0.23	0.34	0.51	0.68	0.49	

Twak Pitata	0.15	0.23	0.46	0.58	0.60	
Nakha Pitata	0.15	0.23	0.46	0.58	0.60	
Mukha Pitata	0.15	0.23	0.46	0.58	0.60	
Mala Pitata	0.15	0.23	0.46	0.58	0.60	
Mutra Pitata	0.30	0.42	0.54	0.70	0.51	
Jaundice	0.23	0.26	0.81	0.72	0.85	
Jaundice	0.26	0.19	0.82	0.49	0.68	
Haematemesis	-	-	-	-	-	-
Weight loss	0.84	0.53	0.78	0.76	0.15	
Fullness	1.11	1.07	1.03	1.09	0.89	
Abdominal swelling	0.50	0.57	1.1	1.06	0.79	
Vomiting	0.50	0.19	0.94	0.56	0.16	
Malaise	1.57	1.15	1.36	1.19	0.23	
Bone pain	0.46	0.42	0.81	0.70	0.85	
Tremours	-	-	-	-	-	-
Disorientation	-	-	-	-	-	-
Asthenia / Weakness	2.03	1.46	1.03	0.98	0.045	Significant

Graph 42- Group B-Internal Group assessment of Symptoms–(Time point b to a)

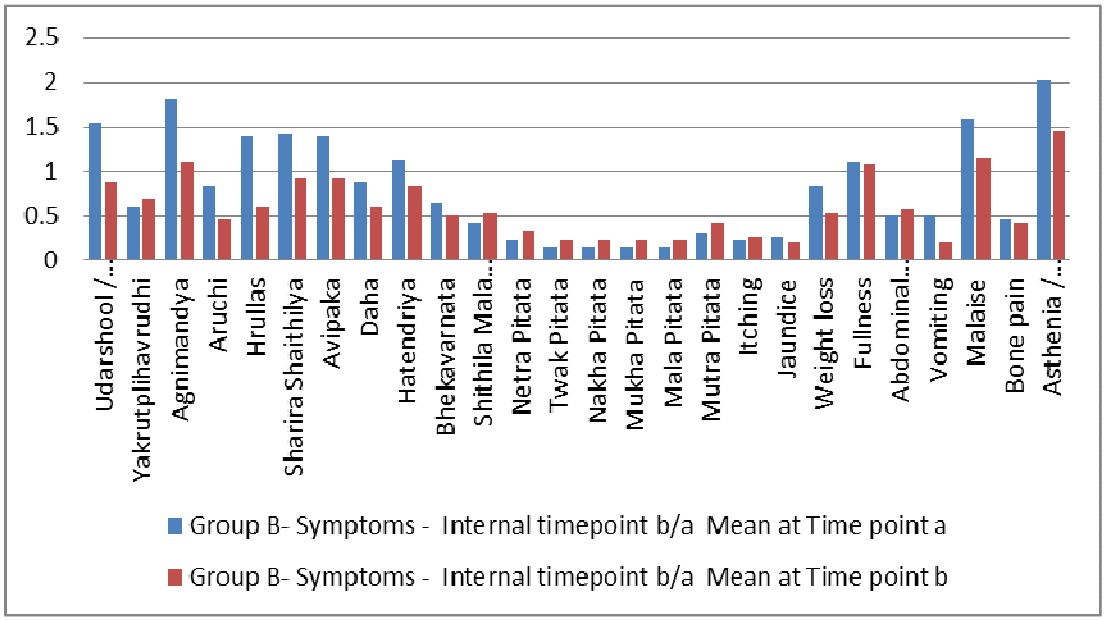


Table – 58 - Group B -Internal Group assessment of Symptoms–(Time point c to a)

	Mean of time point a	Mean of time point c	SD FOR time point a	SD FOR time point c	T	p	SIGNIFICANCE
Udarshool / Yakrut pradeshi shul	1.53	1.03	1.33	1.18	1.43	0.15	
Yakrutplihavrudhi	0.61	0.69	0.89	0.97	0.29	0.76	
Agnimandya	1.8	1.26	0.9	1.1	1.8	0.06	
Aruchi	0.84	0.88	0.92	1.07	0.13	0.89	
Hrullas	1.40	0.84	1.35	1.15	1.57	0.12	
Sharira Shaithilya	1.42	1.07	1.30	1.12	1.02	0.31	
Avipaka	1.46	0.96	1.33	1.24	1.39	1.16	
Daha	0.88	0.53	1.21	1.02	1.11	0.27	
Hatendriya	1.11	0.96	1.27	1.24	0.43	0.66	
Bhekavarnata	0.65	0.57	1.23	1.13	0.23	0.81	
Shithila Mala Pravrutti	0.42	0.53	0.85	0.94	0.46	0.64	
Netra Pitata	0.23	0.26	0.51	0.72	0.22	0.82	
Twak Pitata	0.15	0.19	0.45	0.56	0.26	0.79	
Nakha Pitata	0.15	0.15	0.46	0.46	0.00	>0.99	
Mukha Pitata	0.15	0.15	0.46	0.46	0.00	>0.99	
Mala Pitata	0.15	0.15	0.46	0.46	0.00	>0.99	
Mutra Pitata	0.30	0.34	0.54	0.62	0.23	0.81	
Jaundice	0.23	0.23	0.81	0.71	0	>0.99	
Jaundice	0.26	0.23	0.82	0.58	0.19	0.84	
Haematemesis	-	-	-	-	-	-	-
Weight loss	0.84	0.73	0.78	0.82	0.51	0.60	
Fullness	1.11	1.11	1.03	1.17	0	>0.99	
Abdominal swelling	0.5	0.5	1.1	1.0	0	>0.99	
Vomiting	0.5	0.42	0.94	0.90	0.29	0.76	
Malaise	1.57	1.15	1.36	1.31	1.13	0.26	
Bone pain	0.46	0.57	0.81	1.02	0.44	0.65	
Tremours	-	-	-	-	-	-	
Disorientation	-	-	-	-	-	-	-
Asthenia / Weakness	2.03	1.46	1.03	1.30	1.76	0.08	

Graph 43 -Group B -Internal Group assessment of Symptoms–(Time point c to a)

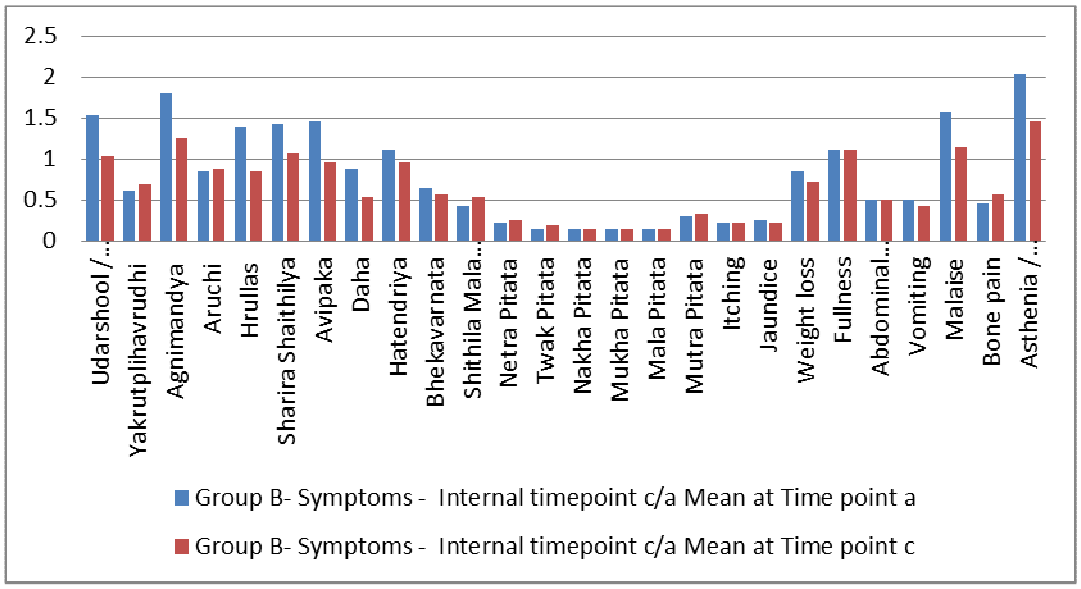


Table – 59 - Intra Group assessment of Symptoms -Group A (b-a) VS Group B (b-a)

	Mean of group A (b-a)	**Mean of group B (b-a)**	**SD FOR A**	**SD FOR B**	**T**	**P**	**SIGNIFICANCE**
Udarshool / Yakrut pradeshi shul	-0.54	-0.65	0.59	0.84	0.50	0.61	
Yakrutplihavrudhi	-0.31	0.07	0.56	0.39	2.83	0.0067	Very Significant
Agnimandya	-0.81	-0.69	0.79	0.61	0.61	0.54	
Aruchi	-0.13	-0.38	0.46	0.69	1.42	0.16	
Hrullas	-0.18	-0.7	0.39	1.11	2.19	0.0333	significant
Sharira Shaithilya	-0.04	-0.5	0.21	0.86	2.4	0.01	significant
Avipaka	-0.68	-0.53	0.47	0.76	0.76	0.44	
Daha	0.09	-0.2	0.42	0.72	2.04	0.04	significant
Hatendriya	-0.45	-0.26	0.37	0.66	1.39	1.16	
Bhekavarnata	0.00	-0.15	0.30	0.54	1.17	0.24	
Shithila Mala Pravrutti	0.045	0.11	0.57	0.58	0.41	0.68	
Netra Pitata	-0.04	0.11	0.21	0.43	1.5	0.11	
Twak Pitata	-	-	-				
Nakha Pitata	-	-	-				
Mukha Pitata	-	-	-				
Mala Pitata	-0.04	0.07	0.21	0.27	1.7	0.09	Not quite

							significant
Mutra Pitata	-	-	-				
Jaundice	-0.09	0.03	0.29	0.34	1.38	0.17	
Jaundice	-.009	-0.07	0.29	0.68	0.08	0.92	
Haematemesis	-	-	-				
Weight loss	-.022	-.03	0.68	0.83	0.35	0.72	
Fullness	-0.72	-0.03	0.63	0.87	3.08	0.003	Very Significant
Abdominal swelling	-.036	0.07	0.49	0.48	3.11	0.003	Very Significant
Vomiting	-0.04	-0.3	0.21	0.67	1.73	0.08	Not quite significant
Malaise	-0.59	-0.42	0.50	0.75	0.88	0.38	
Bone pain	-0.09	-0.03	0.42	0.34	0.47	0.63	
Tremours	-	-	-				
Disorientation	-	-	-				
Asthenia / Weakness	-0.72	-0.57	0.63	0.80	0.70	0.48	

Graph 44 -Intra Group assessment of Symptoms -Group A (b-a) VS Group B (b-a)

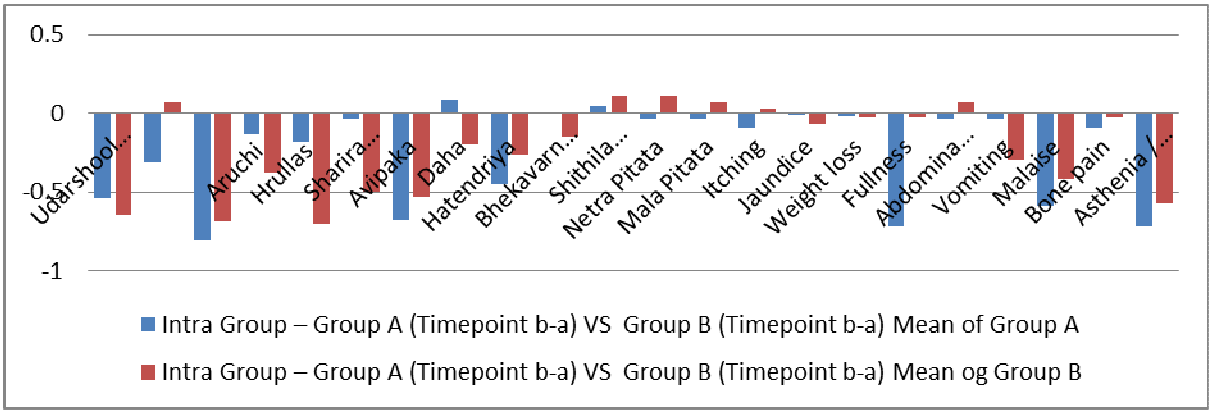
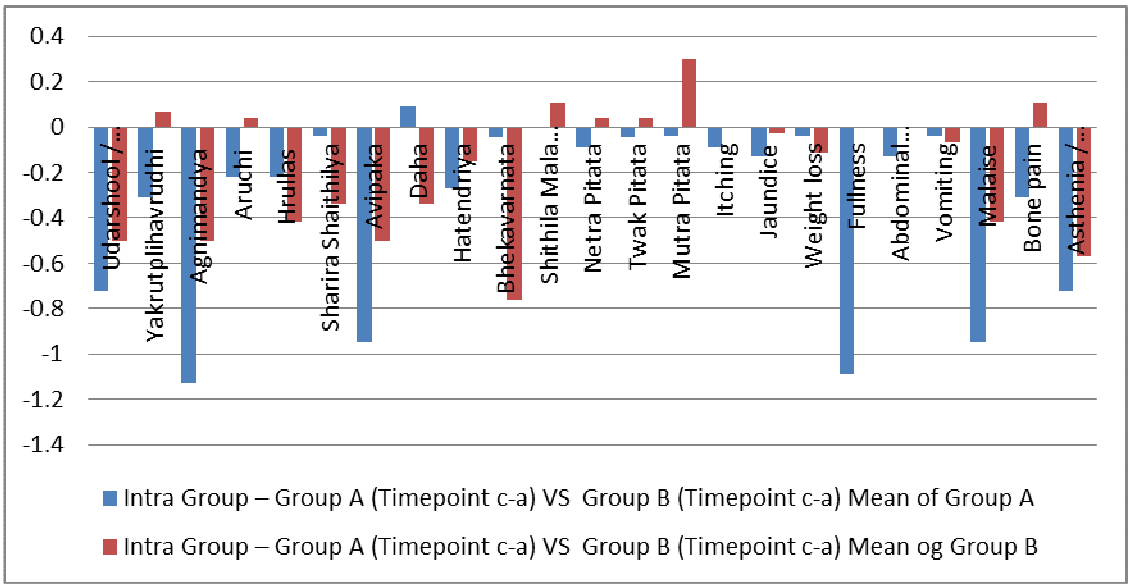


Table – 60 - Intra Group assessment of Symptoms -Group A (c-a) VS Group B (c-a)

	Mean of group A (c-a)	**Mean of group B (c-a0**	**SD FOR A**	**SD FOR B**	**T**	**P**	**SIGNIFICANCE**
Udarshool / Yakrut pradeshi shul	-0.72	-0.50	0.76	1.27	0.73	0.46	
Yakrutplihavrudhi	-0.31	0.07	0.71	0.39	2.4	0.01	Significant
Agnimandya	-1.13	-0.5	1.12	1.14	1.82	0.07	
Aruchi	-0.22	0.038	0.61	1.18	0.95	0.34	
Hrullas	-0.22	-0.42	0.68	1.39	0.60	0.55	
Sharira Shaithilya	-0.04	-0.34	0.21	1.19	1.16	0.25	
Avipaka	-0.95	-0.50	0.89	0.98	1.65	0.10	
Daha	0.09	-0.34	0.24	0.93	2.01	0.04	Significant
Hatendriya	-0.27	-0.15	0.88	0.83	0.47	0.63	
Bhekavarnata	-0.045	-0.76	0.37	0.48	0.24	0.80	
Shithila Mala Pravrutti	0.00	0.11	0.87	0.51	0.56	0.57	
Netra Pitata	-0.09	0.038	0.42	0.59	0.84	0.40	
Twak Pitata	-0.045	0.038	0.21	0.59	0.62	0.53	
Nakha Pitata	-	-	-				
Mukha Pitata	-	-	-				
Mala Pitata	-	-	-				
Mutra Pitata	-0.04	0.3	0.37	052	0.62	0.53	
Jaundice	-0.09	0.00	0.29	0.56	0.67	0.5	
Jaundice	-0.13	-0.03	0.35	0.77	0.54	0.58	
Haematemesis	-	-	-				
Weight loss	-0.04	-0.11	0.84	1.03	0.25	0.80	
Fullness	-1.091	0.00	1.019	1.131	3.482	0.0011	Very Significant
Abdominal swelling	-0.13	0.00	0.88	0.28	0.74	0.46	
Vomiting	-0.04	-0.07	0.21	0.93	0.15	0.87	
Malaise	-0.95	-0.42	0.89	1.02	1.89	0.06	
Bone pain	-0.31	0.11	0.77	0.51	2.303	0.02	Significant
Tremours	-	-	-				
Disorientation	-	-	-				
Asthenia / Weakness	-0.72	-0.57	0.63	1.36	0.47	0.63	

Graph 45 -Intra Group assessment of Symptoms -Group A (c-a) VS Group B (c-a)



III) OBSERVATIONS ON BIOCHEMICAL INVESTIGATIONS

- Biochemical tests like Hemogram and Liver Function test are done before starting Ayurvedic treatment (time point a), in the middle of the treatment (time point b) and at the end of treatment (time point c). Statistical analysis of results of these tests (Hemoglobin, White Blood Cells, Platelets, S. Bilirubin, SGOT, SGPT, Serum Alkaline Phosphatase, Serum Protein, Serum Albumin and Serum Globulin) is done by 2 ways –

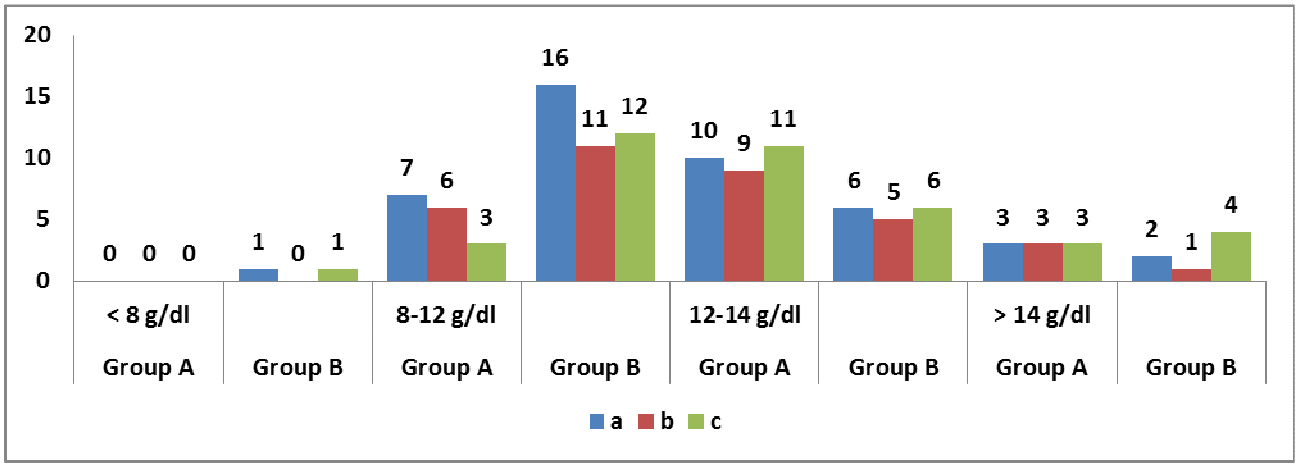
3. Inter group analysis – Analysis of results of tests within 1 group at time points b with a and c with a.
4. Intra group assessment – Analysis of results of tests within group A and group B at certain time points i.e. at time points b and c.

   This type of assessment is essential to assess role of 2 treatment modalities individually and comparatively and thus finally to draw a conclusion about their efficacy on biochemical investigations.

Table 61 – showing effect of Ayurvedic medicines on Hemoglobin

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	< 8 gm/dl Severe anemia		**8-12 mg/dl Anemic**		**12-14 gm/dl WNL**		**>14 gm/dl Above WNL**	
A	0	1	7	16	10	6	3	2
B	0	0	6	11	9	5	3	1
C	0	1	3	12	11	6	3	4

Graph 46 -Effect of Ayurvedic medicines on Hemoglobin



Patients of experimental and control group were divided in four sub groups according to their Hb levels i.e.

Sub group 1 - Hb - > 8 gm/dl

Sub group 2 - 8 – 12 gm/dl

Sub group 3 - 12 – 14 gm/dl (Normal range)

Sub group 4 - > 14 gm/dl (Normal range)

- 7 Patients from group A and 17 patients from Group B had low Hb i.e. (< 12 gm/dl)

  At Timepoint A i.e. before starting Ayurvedic treatment. Among them 1 patient from group B had Hb level < 8 gm/dl at time point A which remains same at Timepoint C.

  In sub group of patients having Hb 8 – 12 gm/dl, there was a drop out of number of patients (7 at Time- point A, 6 at Time - point B and 3 at Time - point C) in group A, while the drop out of number of patients in group B is not so significant (16 at Time - point A, 11 at Time - point B and 12 at Time - point C).

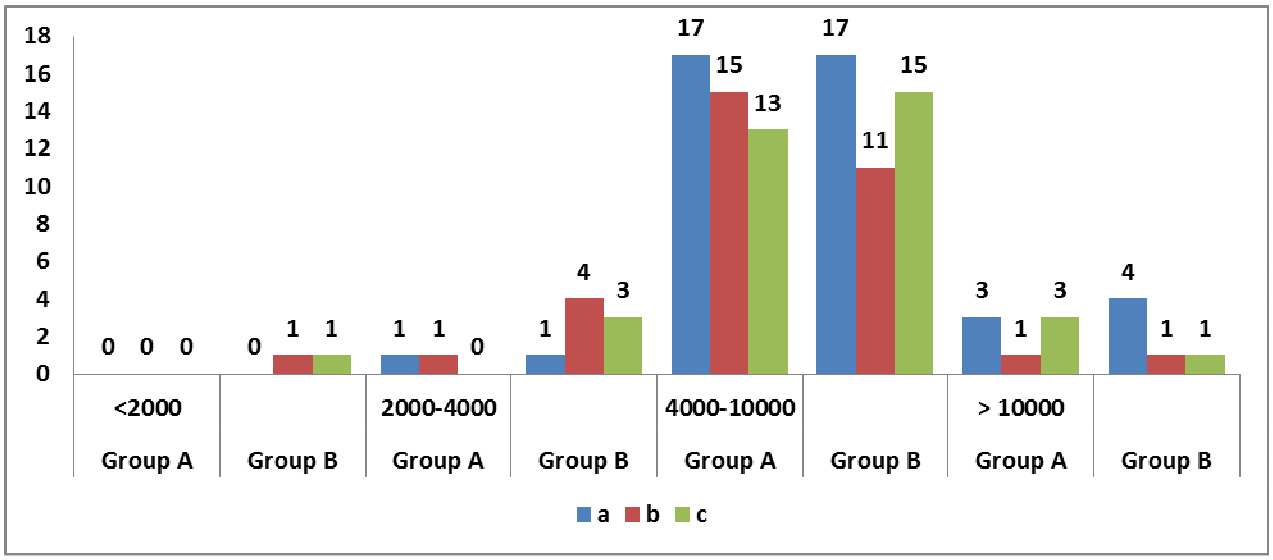
- Hb level more than 12 gm/dl is considered as normal Hb level. The patients in this group are again sub divided in 2 subgroups i.e. having Hb levels 12 – 14 gm/dl and > 14 gm/dl. Rising trend of number of patients is observed in sub group of patients having 12 – 14 Hb in group A (10 at Time - point A, 9 at Time - point B and 11 at Time - point C), while the number of patients

remained same in group B at Time –point A and C with a drop of 1 patient at Time – point B.

Table 62 – showing effect of Ayurvedic medicines on WBC

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	< 2000 / cmm Severe Leukopenia		**2000-4000/cmm Leucopenic**		**4000-10000/cmm WNL**		**>10000 /cmm Leucocytosis**	
A	0	0	1	1	17	17	3	4
B	0	1	1	4	15	11	1	1
C	0	1	0	3	13	15	3	1

Graph 47 - Graphical representation Effect of Ayurvedic medicines on WBC



Patients in both the groups are divided in 4 subgroups as per WBC count -

Sub group 1 - < 2000 /cmm

Sub group 2 – 2000 - 4000 /cmm

Sub group 3 - 4000 - 10000 /cmm (Normal range)

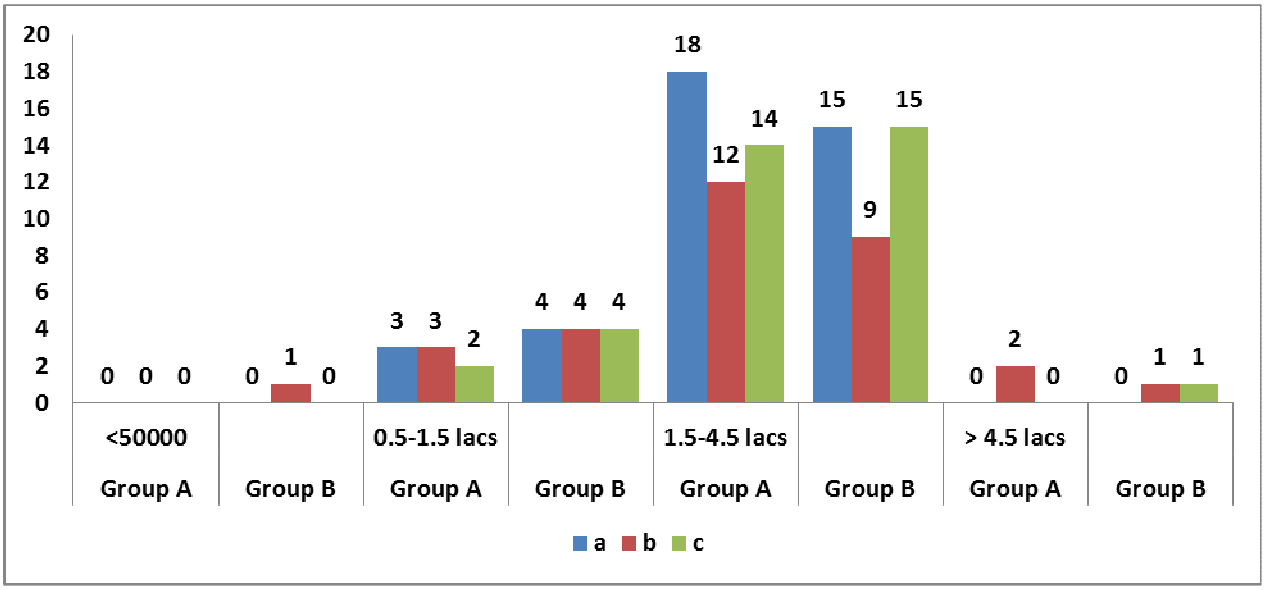
Sub group 4 - > 10000 /cmm

17 patients in each group (Group A & B) fall under the normal range of WBC count (4000 – 10000/cmm). Thus it is not worthwhile to analyse the data of efficacy of YG3 on inadequate number of patients having leucopenia and leucocytosis.

Table 63 – Showing effect of Ayurvedic medicines on Platelets

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	< 50000 / cmm Severe Thrombocytopenia		**0.5-1.5 lacs/cmm Thrombocytopenia**		**1.5-4.5 lacs/cmm WNL**		**> 4.5 lacs/cmm Throbocytosis**	
A	0	0	3	4	18	15	0	0
B	0	1	3	4	12	9	2	1
C	0	0	2	4	14	15	0	1

Graph 48 – Showing effect of Ayurvedic medicines on Platelets



Platelet Count

Patients in both the groups are divided in 4 subgroups as per Platelet count -

Sub group 1 - < 50000 /cmm

Sub group 2 – 50000 - 100000 /cmm

Sub group 3 - 100000 - 450000 /cmm (Normal range)

Sub group 4 - > 450000 /cmm

18 patients from group A and 15 patients from group B fall under the normal range of platelet count (100000 - 450000 /cmm). Thus it is not worthy to analyse the data of efficacy of YG3 on inadequate number of patients having low or high platelet counts.

Table 64 – Showing effect of Ayurvedic medicines on Bilirubin (Total)

	Number of patients					
Time points	**Group A**	**Group B**	**Group A**	**Group B**		
	0-1.23 g /dl WNL		**>1.23 mg/dl Jaundice**			
A	12	17	5	1		
B	11	7	3	6		
C	11	13	3	5		

Graph 49 – Showing effect of Ayurvedic medicines on Bilirubin (Total)

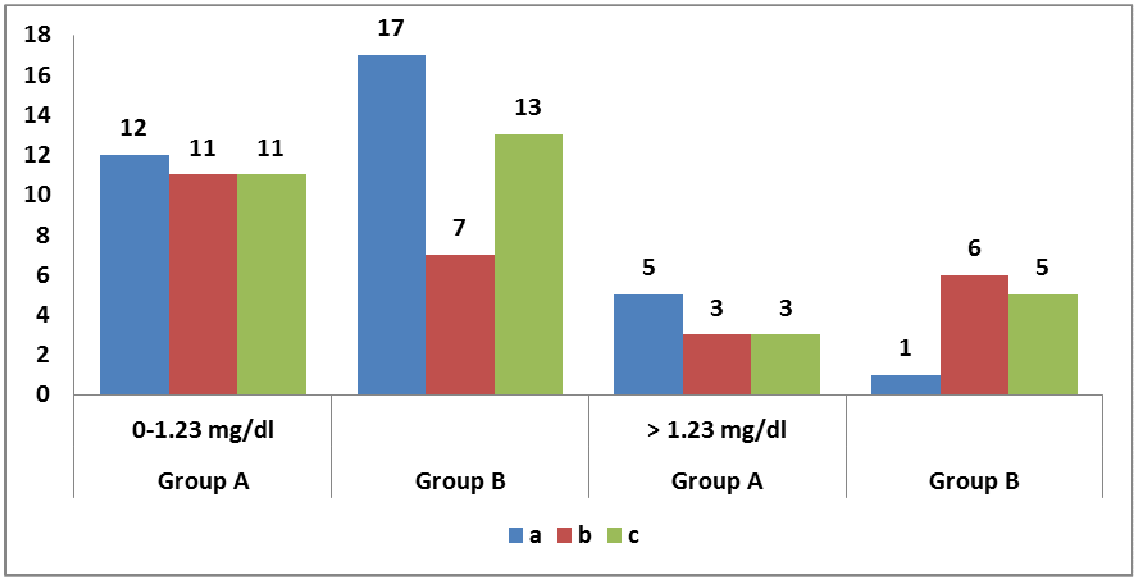
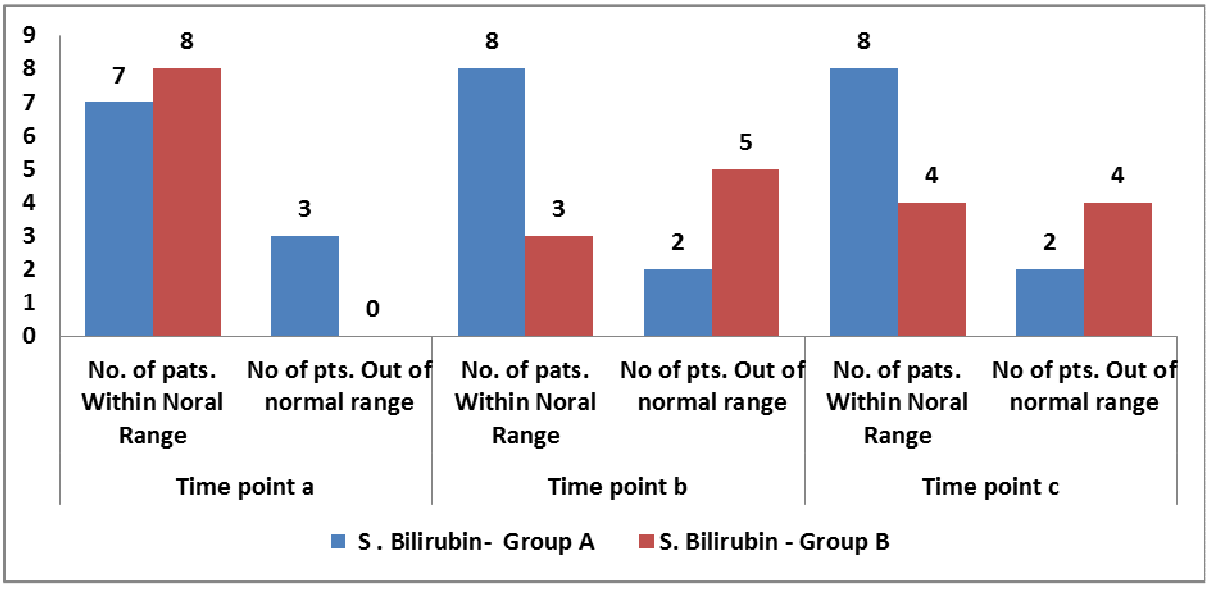


Table 65 - showing pattern of S. Bilirubin levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)

	Time point a	Time point b	Time point c			
	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range
S . Bilirubin- Group A	7	3	8	2	8	2
S. Bilirubin - Group B	8	0	3	5	4	4

Graph 50 - Showing pattern of S. Bilirubin levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)



Bilirubin

Patients in both the groups are divided in 2 subgroups as per Bilirubin count -

Sub group 1 – 0- 1.23 mg/dl (Normal)

Sub group 2 – mg/dl (Abnormal)

- 12 Patients in group A and 17 patients in group B were having normal levels of total Bilirubin. It was maintained within normal limits in 11 patients in group A at both time – points b and c, while total bilirubin remained in normal range in 13 patients of group B at time – point c. This suggests better efficacy of YG3 in the improvement of liver functions.
- In second sub group where levels of bilirubin were already elevated indicating liver dysfunction, 5 patients laid in group A and 1 patient laid in group B at time – point a.

  In spite of known liver disorders in patients of both groups, declining rate of number of patients was observed in group A (5 → 3 → 3) at time –points b and c.

  On the other hand increasing trend of number of patients with elevated values of total bilirubin was observed in group B at time –points b & c (1 → 6 → 5).

Table 66 – Showing effect of Ayurvedic medicines on SGOT

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	0-40 IU/L		**40-60 IU/L**		**60-100 IU/L**		**>100 IU/L**	
A	7	12	2	3	2	2	4	0
B	6	10	4	2	3	1	1	0
C	5	14	4	1	2	3	2	0

Graph 51 - Showing effect of Ayurvedic medicines on SGOT

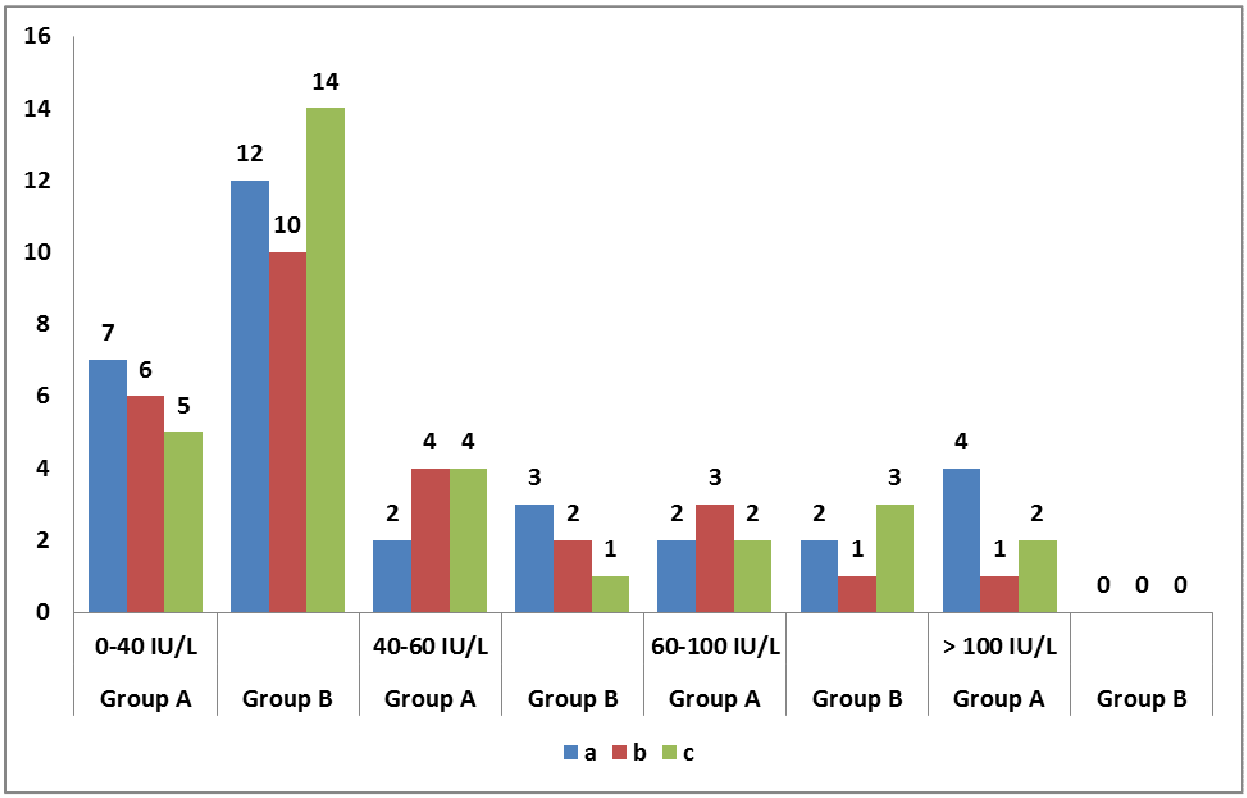


Table 67 - showing pattern of S.G.O.T. levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)

	Time point a		Time point b		Time point c	
	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range
S.G.O.T - Group A	5	4	2	7	3	6
S.G.O.T - Group B	3	3	5	1	5	1

Graph 52 - Showing pattern of S.G.O.T. levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)

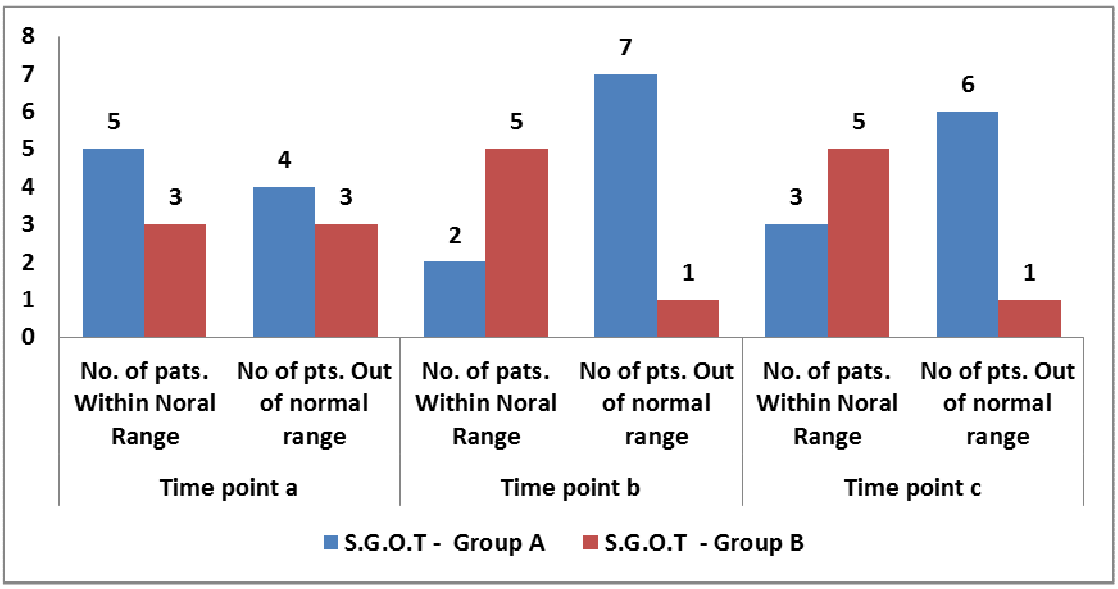


Table 68 – showing effect of Ayurvedic medicines on SGPT

Time points	Number of patients							
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
	0-30 IU/L		30-50 U/L		50-90 IU/L		>90 IU/L	
A	5	9	6	5	1	3	4	1
B	5	5	5	4	3	3	1	2
C	6	11	5	4	2	2	1	1

Graph 53 – showing effect of Ayurvedic medicines on SGPT

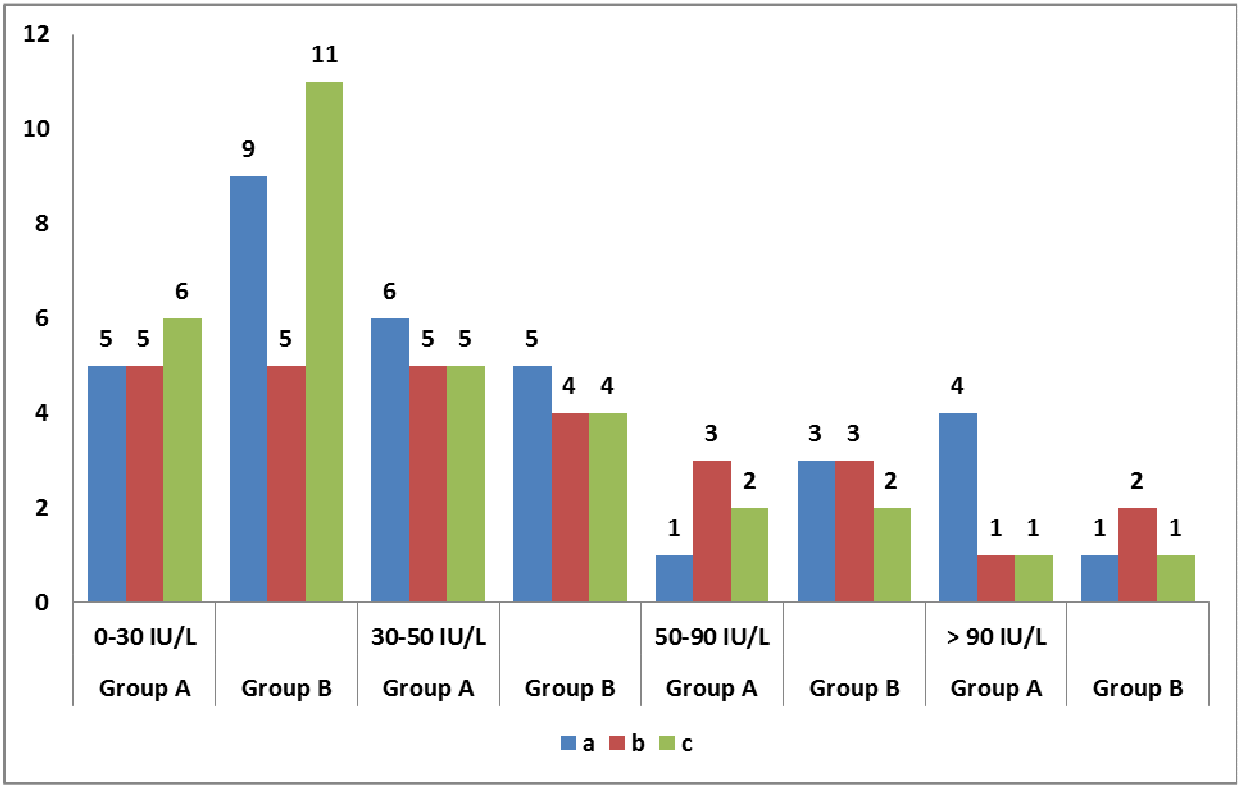
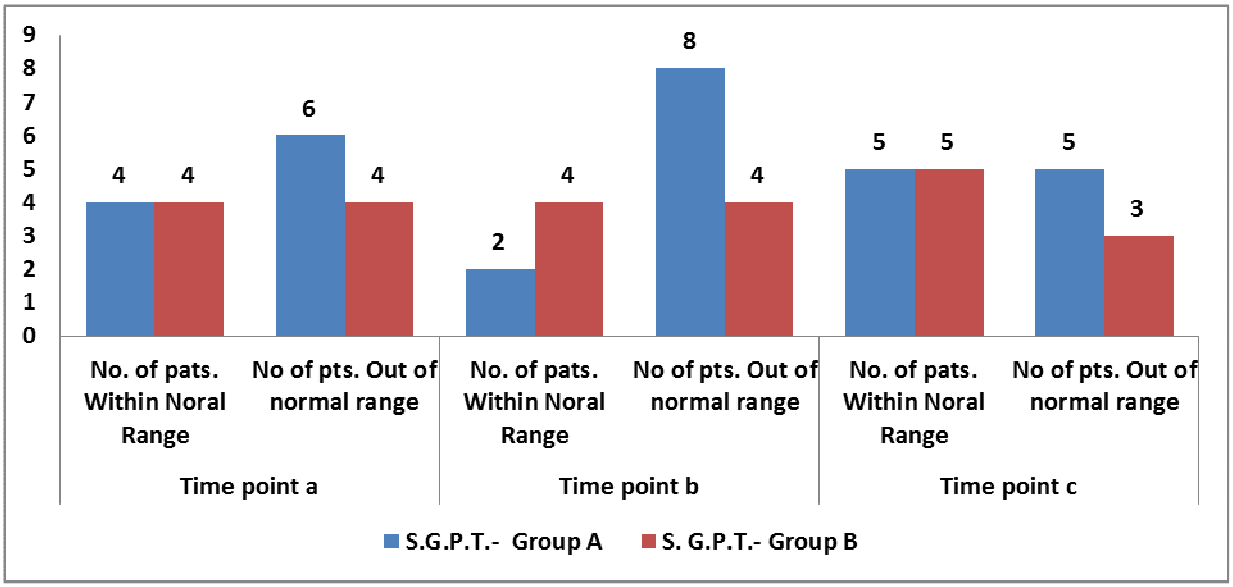


Table 69 - showing pattern of S.G.P.T. levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)

	Time point a		Time point b		Time point c	
	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range
S.G.P.T.- Group A	4	6	2	8	5	5
S. G.P.T.- Group B	4	4	4	4	5	3

Graph 54 -showing pattern of S.G.P.T. levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)



SGOT and SGPT

Patients in both the groups are divided in 4 subgroups as per SGOT count -
Sub group 1 – 0 – 40 IU/L (Normal range)
Sub group 2 – 40 – 60 IU/L
Sub group 3 – 60 – 100 IU/L
Sub group 4 - > 100 IU/L

Patients in both the groups are divided in 4 subgroups as per SGPT count -
Sub group 1 – 0 – 30 IU/L (Normal range)
Sub group 2 – 30 – 50 IU/L
Sub group 3 – 50 – 90 IU/L
Sub group 4 - > 90 IU/L

8 patients of group A and 5 patients of group B showed elevated levels of SGOT while 11 patients of group A and 9 patients of group B showed elevated levels of SGPT.

In our study elevated levels of SGOT & SGPT are sub divided in 3 subgroups.
SGOT - In the middle subgroup number of patients remained constant in group A and increased in group B.
SGPT - In the last subgroup number of patients reduced in group A and remained constant in group B.

Table 70 – showing effect of Ayurvedic medicines on Alkaline Phosphatase

Time points	Number of patients							
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
	44-147 U/L		147-200 U/L		200-300 U/L		>300 U/L	
A	11	7	1	4	1	4	1	0
B	10	4	2	1	2	3	0	2
C	6	9	1	4	1	1	1	2

Graph 55 – showing effect of Ayurvedic medicines on Alkaline Phosphatase

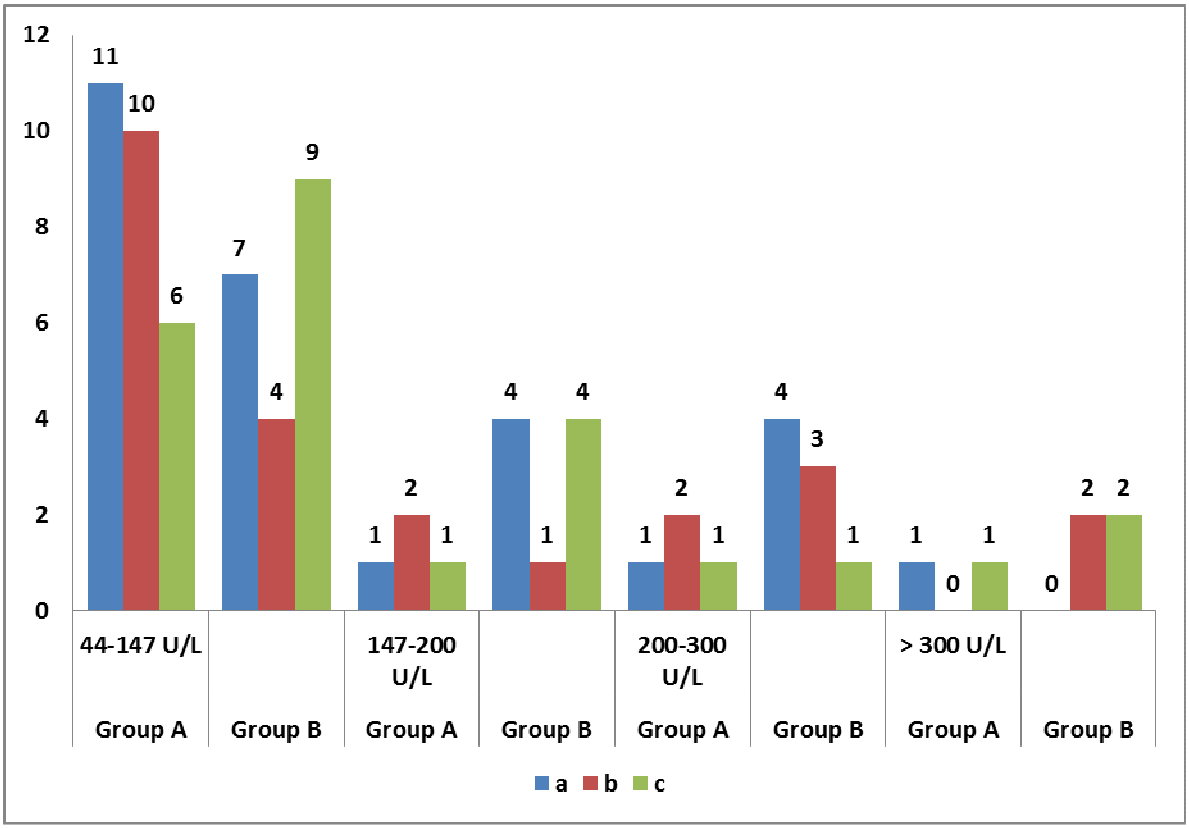
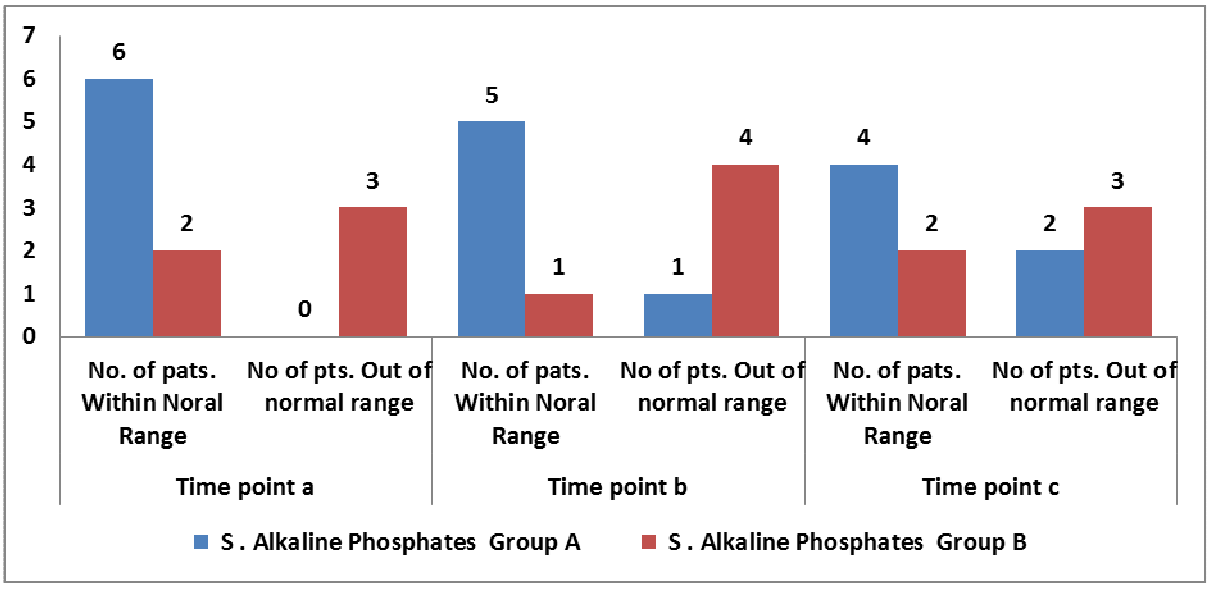


Table 71 - showing pattern of S. Alkaline phosphates levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)

	Time point a		Time point b		Time point c	
	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range
S . Alkaline Phosphates Group A	6	0	5	1	4	2
S . Alkaline Phosphates Group B	2	3	1	4	2	3

Graph 56 - showing pattern of S. Alkaline phosphates levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)



Alkaline Phosphatase

Patients in both the groups are divided in 4 subgroups as per SGPT count -

Sub group 1 – 44 – 147 U/L (Normal range)

Sub group 2 – 147 – 200 U/L

Sub group 3 – 200 – 300 U/L

Sub group 4 - > 300 U/L

Elevated levels of alkaline phosphatase are divided in subgroups 2, 3 and 4 as mentioned above.

Number of patients remained same at time – points a and c in subgroups 2 and 4 in both study and control groups.

We also observed declining trend of number of patients at time – points a and c in subgroup 2 in group B.

Table 72 – showing effect of Ayurvedic medicines on Serum Proteins

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**		
	< 6.4 g/dl		**6.4-8.3 g/dl**		**>8.3 g/dl**			
A	2	2	11	10	1	0		
B	1	4	10	4	0	0		
C	0	4	6	10	0	0		

Graph 57 - showing effect of Ayurvedic medicines on Serum Proteins

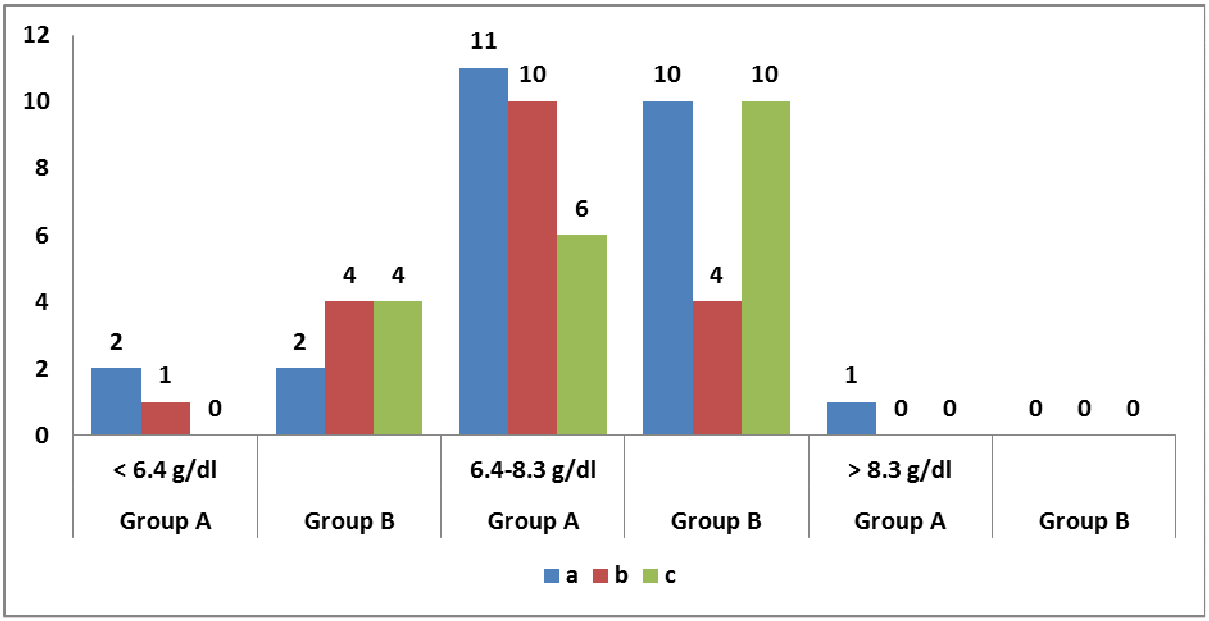
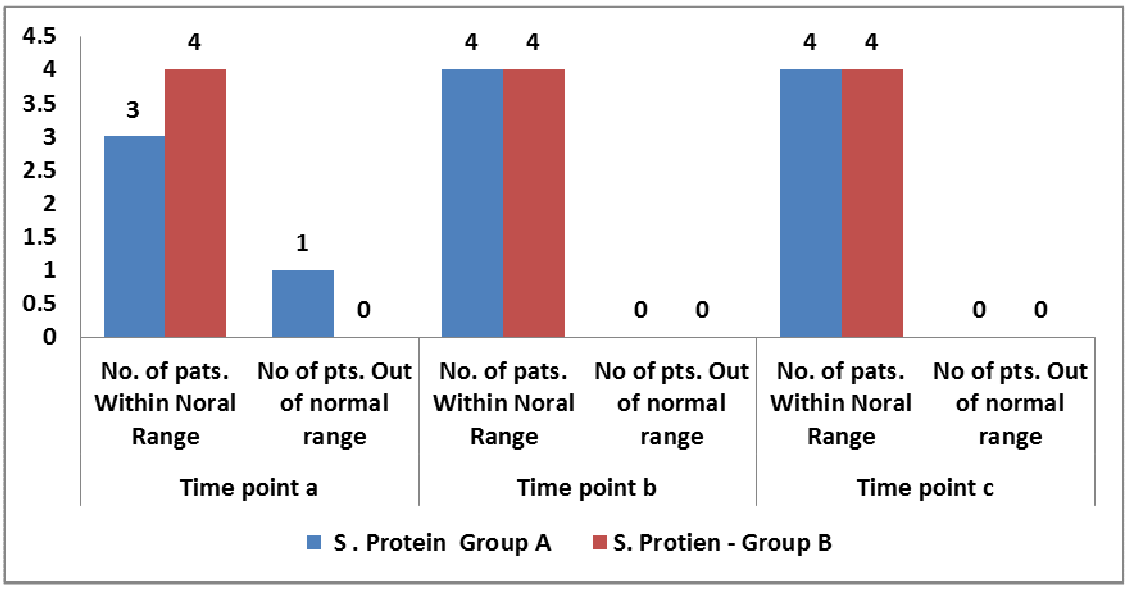


Table 73 - showing pattern of s. Protein levels of patients in both groups (Group a and Group B) at 3 time points (a,b,c)

	Time point a		Time point b		Time point c	
	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range
S . Protein Group A	3	1	4	0	4	0
S. Protien - Group B	4	0	4	0	4	0

Graph 58 - showing pattern of s. Protein levels of patients in both groups (Group a and Group B) at 3 time points (a,b,c)



Proteins

Patients in both the groups are divided in 2 subgroups as per Protein count -

Sub group 1 – (Normal)

Sub group 2 – > (Abnormal)

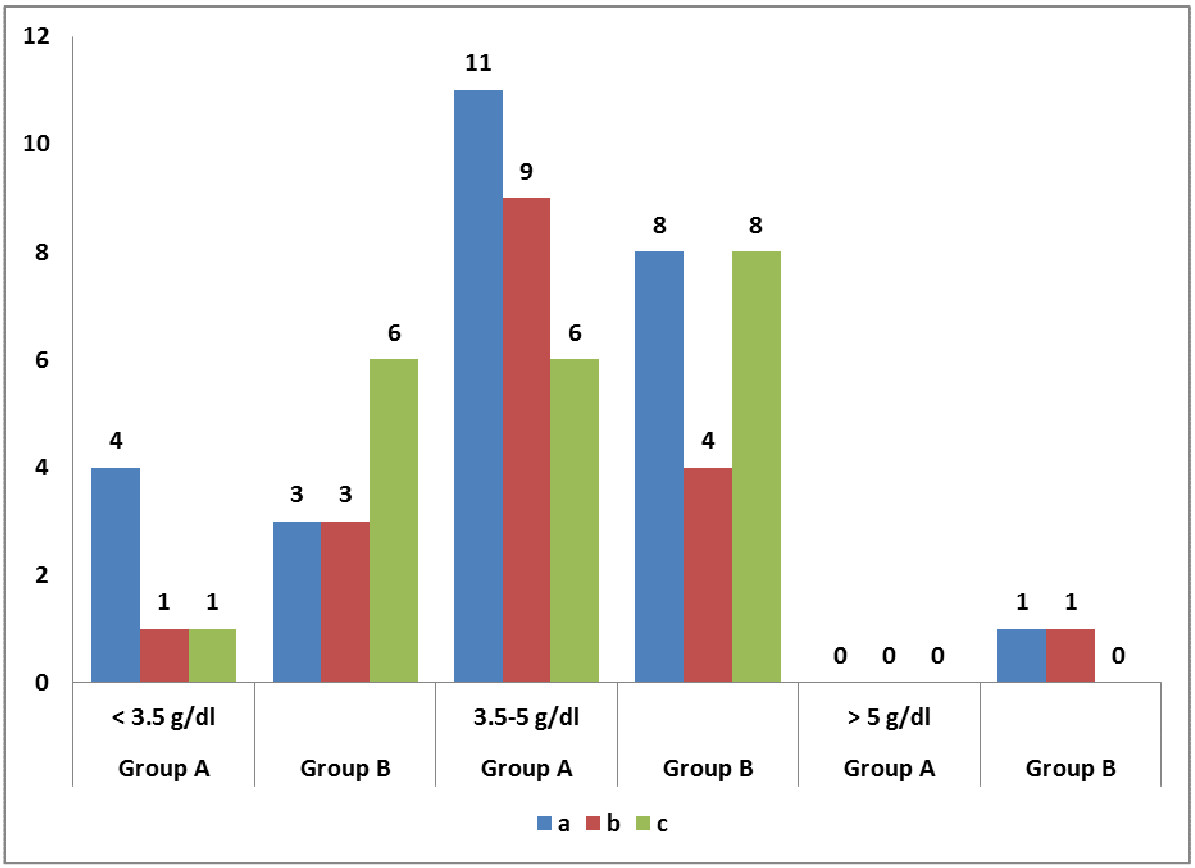
In our study most of the patients of Group A and B laid in normal range of serum protein.

Patients with hypoproteinemia were 2 in each group at time – point a. There was improvement in the level of protein in group A patients and no patient was having hypoproteinemia at the end of the treatment. While 4 patients of group B were found to have hypoproteinemia at the end of the treatment.

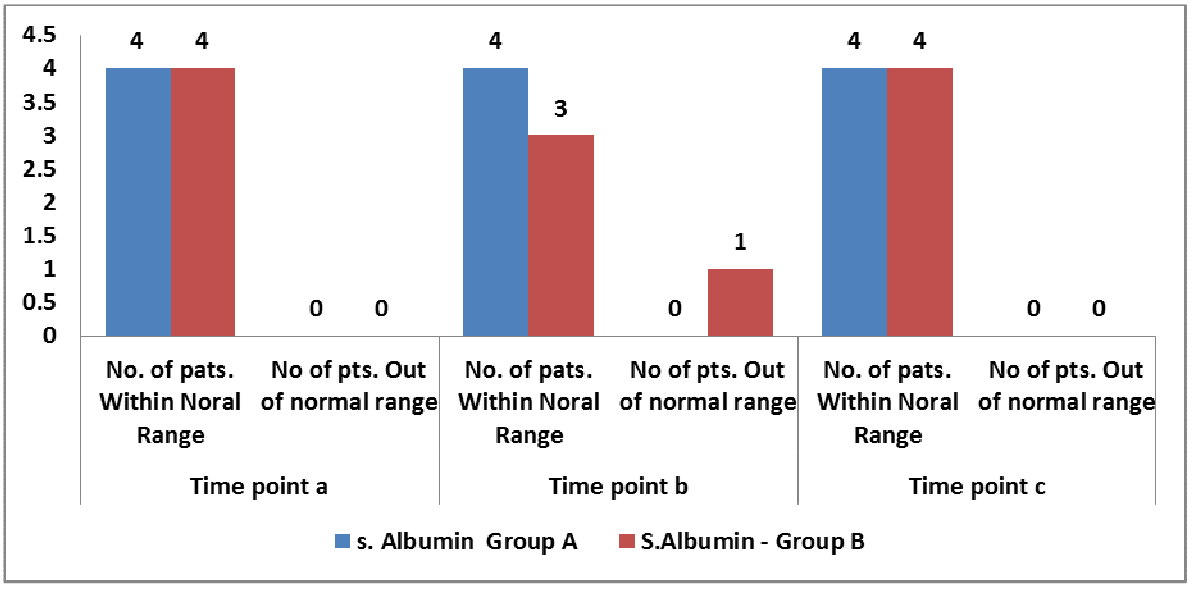
Table 74 – showing effect of Ayurvedic medicines on Serum Albumin-

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**		
	< 3.5 g/dl		**3.5 – 5 g/dl**		**>5 g/dl**			
A	4	3	11	8	0	1		
B	1	3	9	4	0	1		
C	1	6	6	8	0	0		

Graph 59 - showing effect of Ayurvedic medicines on Serum Albumin



	Time point a		Time point b		Time point c	
	No. of pats. Within Noral Range	**No of pts. Out of normal range**	**No. of pats. Within Noral Range**	**No of pts. Out of normal range**	**No. of pats. Within Noral Range**	**No of pts. Out of normal range**
s. Albumin Group A	**4**	**0**	**4**	**0**	**4**	**0**
S.Albumin - Group B	**4**	**0**	**3**	**1**	**4**	**0**



Albumin :

Hypoalbuniemia was seen in 4 patients of Group A and 3 patients of Group B before starting Ayurvedic medicines. At time point c i.e at the end of treatment only 1 patient had hypoalbuniemia in group A, against 6 patients were presented with hypoalbuniemia in group B at the end of treatment.

Table 76 – showing effect of Ayurvedic medicines on Serum Globulin-

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**		
	< 2.6 g/dl		**2.6-4.6 g/dl**		**>4.6 g/dl**			
A	2	2	13	10	0	0		
B	1	3	8	5	0	0		
C	0	2	7	11	0	0		

Graph 61 – showing effect of Ayurvedic medicines on Serum Globulin

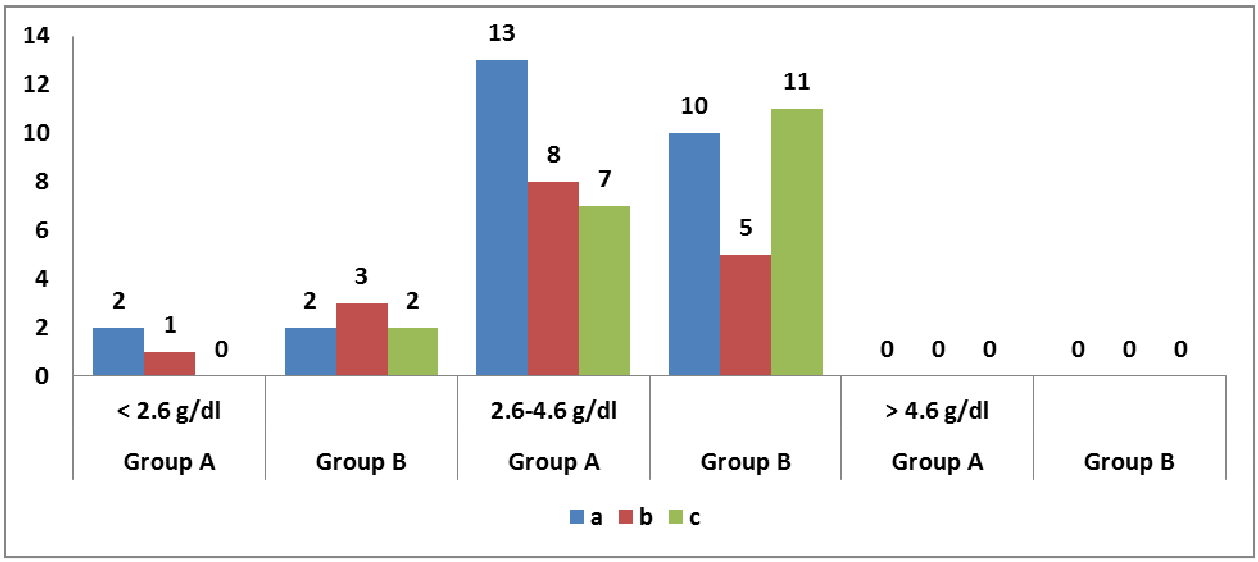
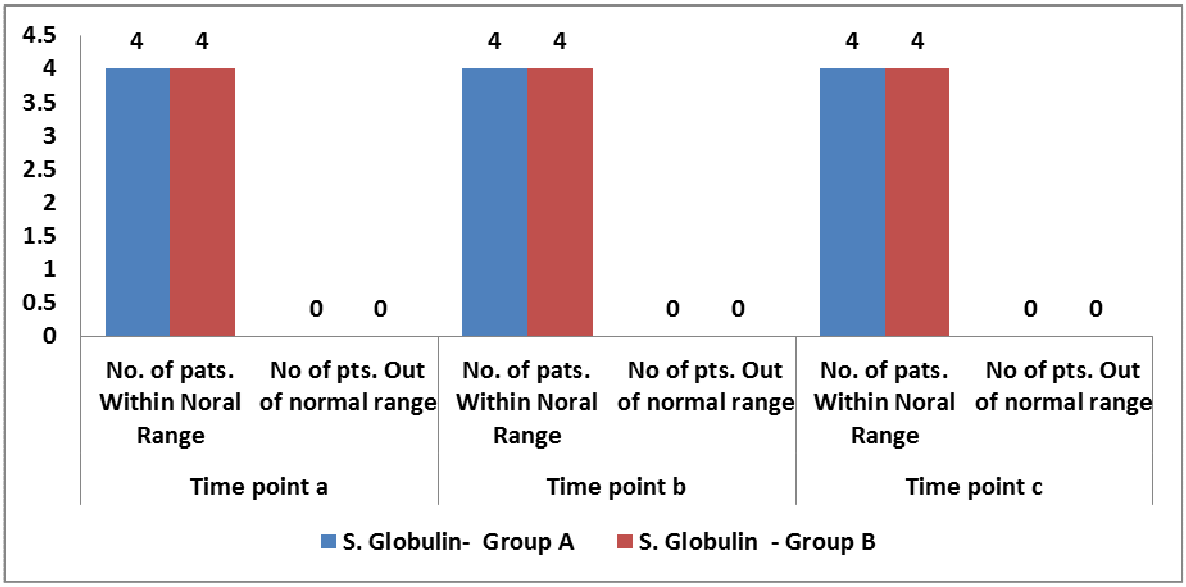


Table 77 - showing pattern of S. Globulin levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)

	Time point a		Time point b		Time point c	
	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range	No. of pats. Within Noral Range	No of pts. Out of normal range
S. Globulin- Group A	4	0	4	0	4	0
S. Globulin - Group B	4	0	4	0	4	0

Graph 62 - showing pattern of S. Globulin levels of patients in both groups (Group a and Group B)at 3 time points (a,b,c)



Globulin

Hypoglobuliniemia was seen in 2 patients of both the Groups (Group A and Group B) before starting Ayurvedic medicines. At time point c i.e at the end of treatment no patient

had hypoglobulinemia in group A, while patients remained constant i.e. 2 in group B at the end of treatment

Statistics of pathological investigations -

Table 78 - Intra Group (c-time point - a time point)

	Mean of Group A (c-a)	Mean of Group B (c-a)	SD For Group A	SD for Group B	P	Significance
HB	0.04	0.65	1.05	1.74	0.16	Not Significant
WBC	276	-642	2857	2562	0.24	Not Significant
PLATELET	22215	29773	89286	140225	0.82	Not Significant
S Bilirubin (Total)	-0.06	0.52	0.41	1.27	0.065	Not quite significant
SGOT	4.09	2.87	83	23.4	0.94	Not Significant
SGPT	-51	1.55	140	34	0.07	Not quite significant
S.Alkaline Phosphatase	1.6	29	164	117	0.54	Not Significant
S. Protein	-0.45	1.66	1.9	10.4	0.49	Not Significant
S. Albumin	-0.21	-0.77	0.33	0.74	1.3	Not Significant
S. Globulin	0.27	0.12	0.27	0.15	0.94	Not Significant

Table 79 - Intra Group (b time point - a time point)

	Mean of Group A (b-a0	**Mean of Group B (b-a)**	**SD For Group A**	**SD for Group B**	**P**	**Significance**
HB	0.05	0.85	1.07	1.54	0.046	Significant
WBC	-135	-978	1831	2484	0.19	Not Significant
PLATELET	23773	-157	93889	98791	0.39	Not Significant
S Bilirubin (Total)	0.09	0.69	0.93	1.31	0.1	Not Significant
SGOT	-1.8	1.6	84	19.4	0.83	Not Significant
SGPT	-47	6.9	134	30	0.05	Not quite significant
S.Alkaline Phosphatase	-8.6	34.8	131	118	0.29	Not Significant
S. Protein	0.36	2.38	0.56	10.8	0.52	Not Significant
S. Albumin	0.20	-0.27	0.15	0.58	1.7	Not Significant
S. Globulin	0.46	-0.30	0.29	0.47	2.8	Significant

Table 80 - Inter Group – Group A- (b-time point - a time point)

	Mean of time point a	Mean of time point b	SD For time point a	SD for time point b	P	Significance
HB	12.6	12.6	1.55	1.22	0.8926	Not Significant
WBC	8473	8323	5102	5604	0.9302	Not Significant
PLATELET	230050	256200	70916	147695	0.4797	Not Significant
S Bilirubin (Total)	0.9156	1.022	0.42	0.94	0.6841	Not Significant
SGOT	75	73	59	77	0.9454	Not Significant
SGPT	95	50	136	40	0.2588	Not Significant
S.Alkaline Phosphatase	146	137	123	73	0.8233	Not Significant
S. Protein	6.8	7.2	0.63	0.65	0.1863	Not Significant
S. Albumin	3.8	3.2	0.4	1.8	0.58	Not Significant
S. Globulin	3.3	3.0	0.5	1.7	0.32	Not Significant

Table 81 - Inter Group –Group A (c time point - a time point)

	Mean of time point a	Mean of time point c	SD For time point a	SD for time point c	P	Significance
HB	12.6	12.6	1.5	1.5	0.92	Not Significant
WBC	8473	8777	5102	5786	0.86	Not Significant
PLATELET	230050	230262	70916	82467	0.99	Not Significant
S Bilirubin (Total)	0.91	0.83	0.42	0.46	0.62	Not Significant
SGOT	75	79	59	80	0.88	Not Significant
SGPT	95	42	136	41	0.17	Not Significant
S.Alkaline Phosphatase	146	147	123	100	0.96	Not Significant
S. Protein	6.8	6.9	0.63	0.40	0.58	Not Significant
S. Albumin	3.8	2.8	0.46	1.6	1.1	Not Significant
S. Globulin	3.3	2.8	0.5	1.6	0.5	Not Significant

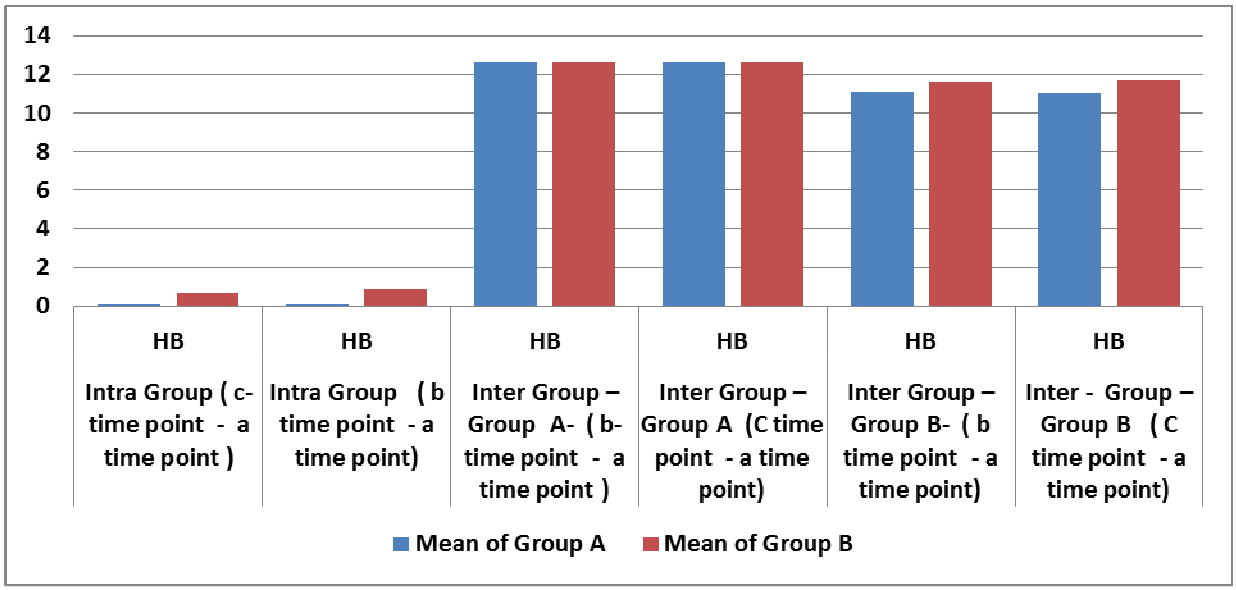
Table 82 - Inter Group – Group B- (b time point - a time point)

	Mean of time point a	**Mean of time point b**	**SD For time point a**	**SD for time point b**	**P**	**Significance**
HB	11.07	11.64	2.16	1.37	0.34	Not Significant
WBC	7052	5765	2864	2754	0.16	Not Significant
PLATELET	232784	220120	106832	131819	0.75	Not Significant
S Bilirubin (Total)	1.006	2.17	1.28	2.17	0.06	Not Significant
SGOT	35.7	32.6	18	17	0.63	Not Significant
SGPT	38.2	48.6	30	31	0.34	Not Significant
S.Alkaline Phosphatase	162	224	64	208	0.28	Not Significant
S. Protein	7	6.5	0.9	0.9	0.32	Not Significant
S. Albumin	4.3	3.2	0.44	1.9	1.1	Not Significant
S. Globulin	3	2	0.4	1.4	1.2	Not Significant

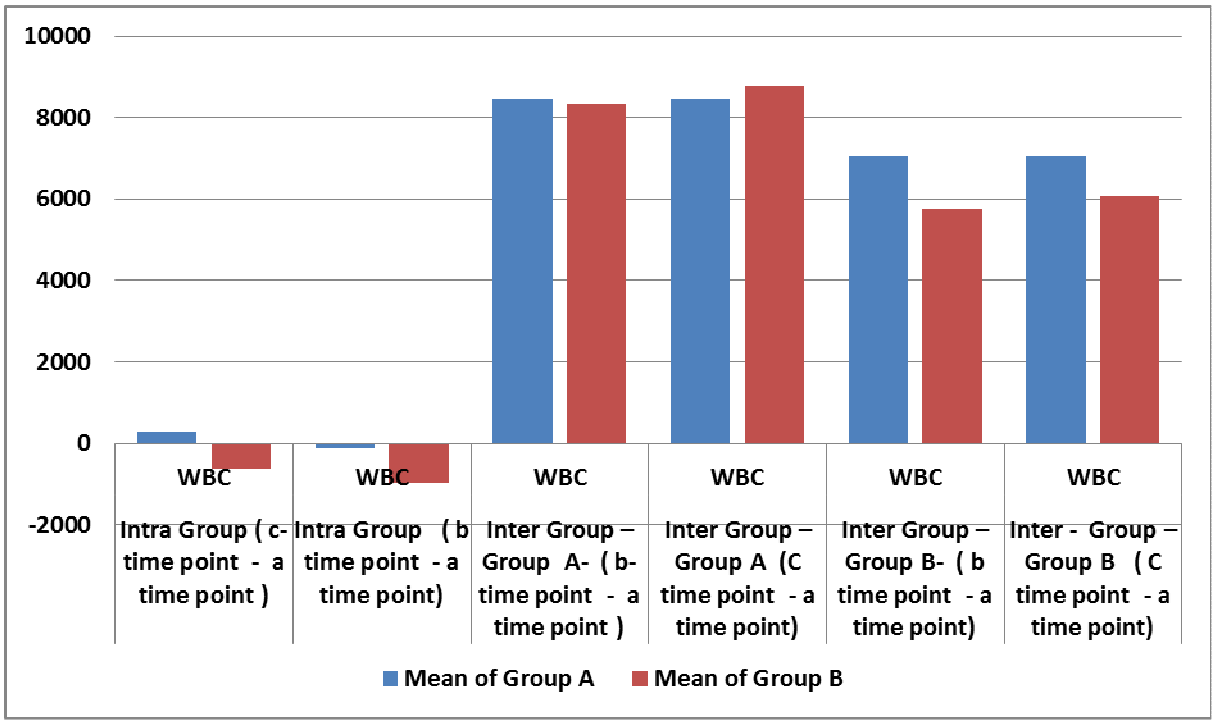
Table 83 - Inter - Group – Group B (c time point - a time point)

	Mean of time point a	**Mean of time point c**	**SD For time point a**	**SD for time point c**	**P**	**Significance**
HB	11	11.7	2.1	2.3	0.33	Not Significant
WBC	7052	6057	2864	2339	0.22	Not Significant
PLATELET	232784	274700	106832	160407	0.34	Not Significant
S Bilirubin (Total)	1.00	1.36	1.28	1.52	0.44	Not Significant
SGOT	35.7	32.5	18	22	0.63	Not Significant
SGPT	38.2	32	30	24	0.5	Not Significant
S.Alkaline Phosphatase	162	202	64	250	0.55	Not Significant
S. Protein	6.9	10.6	0.9	14.2	0.37	Not Significant
S. Albumin	4.3	2.8	0.44	1.7	1.6	Not Significant
S. Globulin	3	2.5	0.4	1.4	0.66	Not Significant

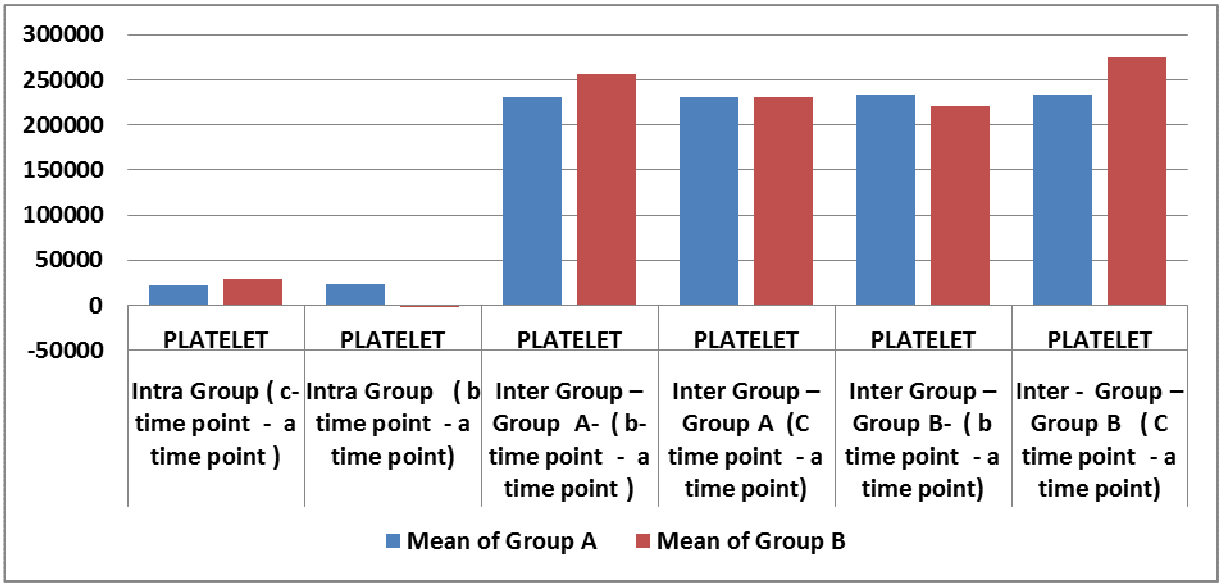
Graph 63 - showing pattern of HB levels of patients in both groups at various time points



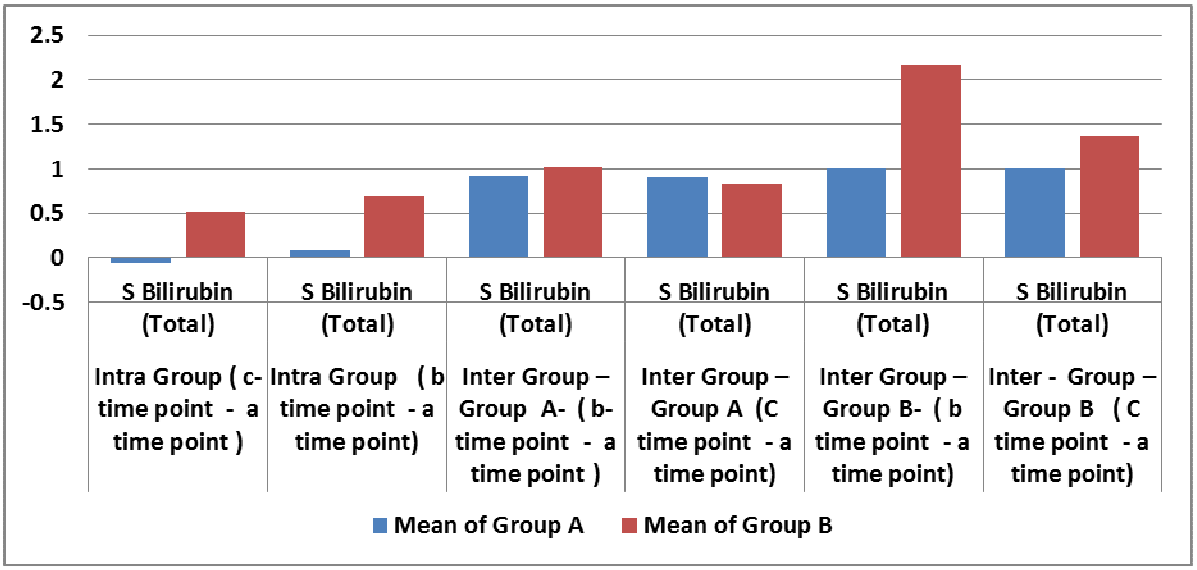
Graph 64- showing pattern of WBC levels of patients in both groups at various time points



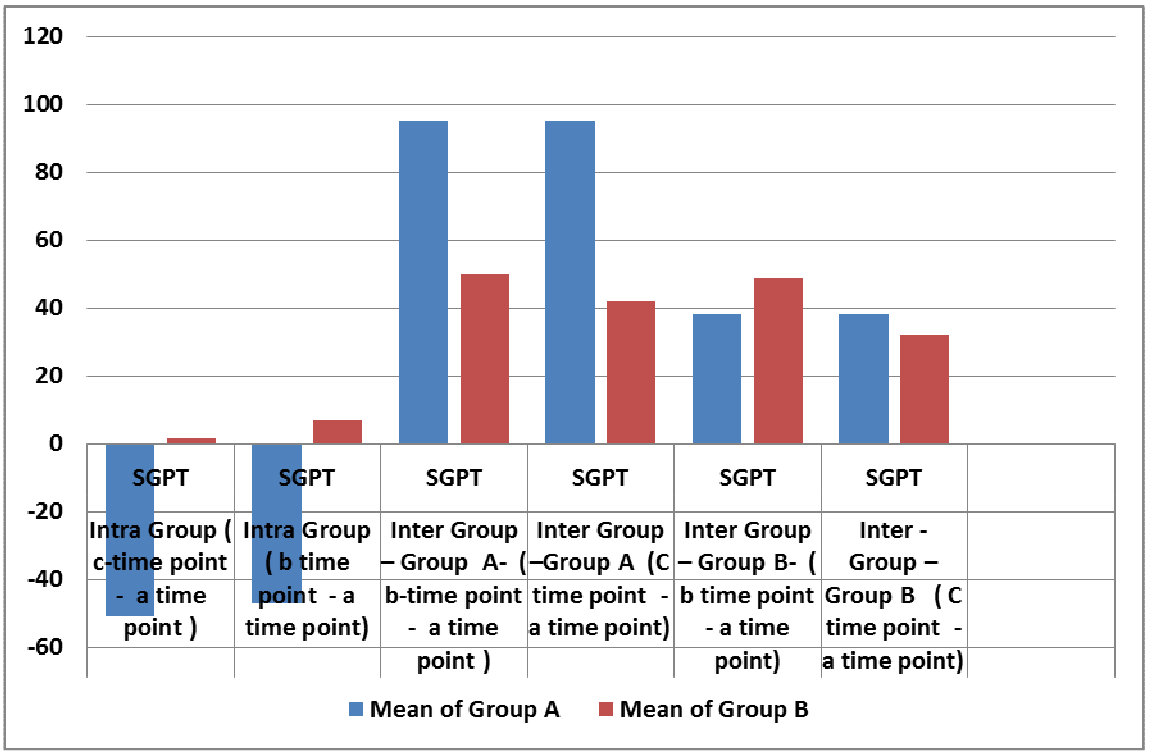
Graph 65 - showing pattern of platelet levels of patients in both groups at various time points



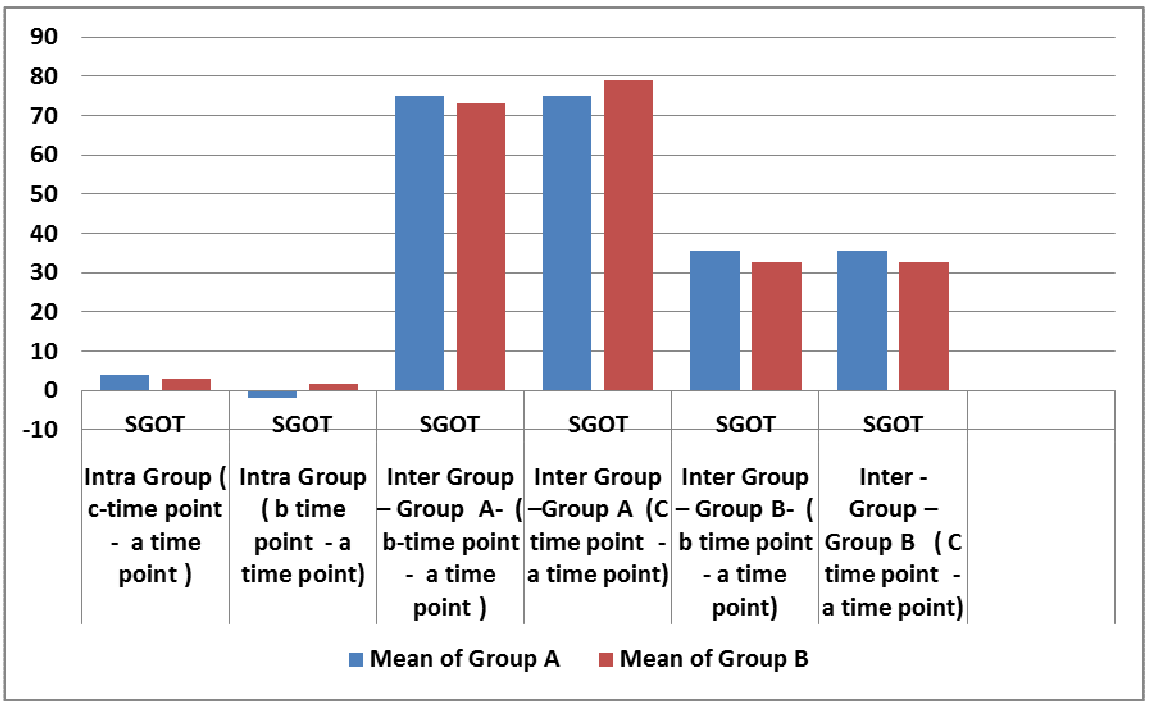
Graph 66 - showing pattern of S. Bilirubin levels of patients in both groups at various time points



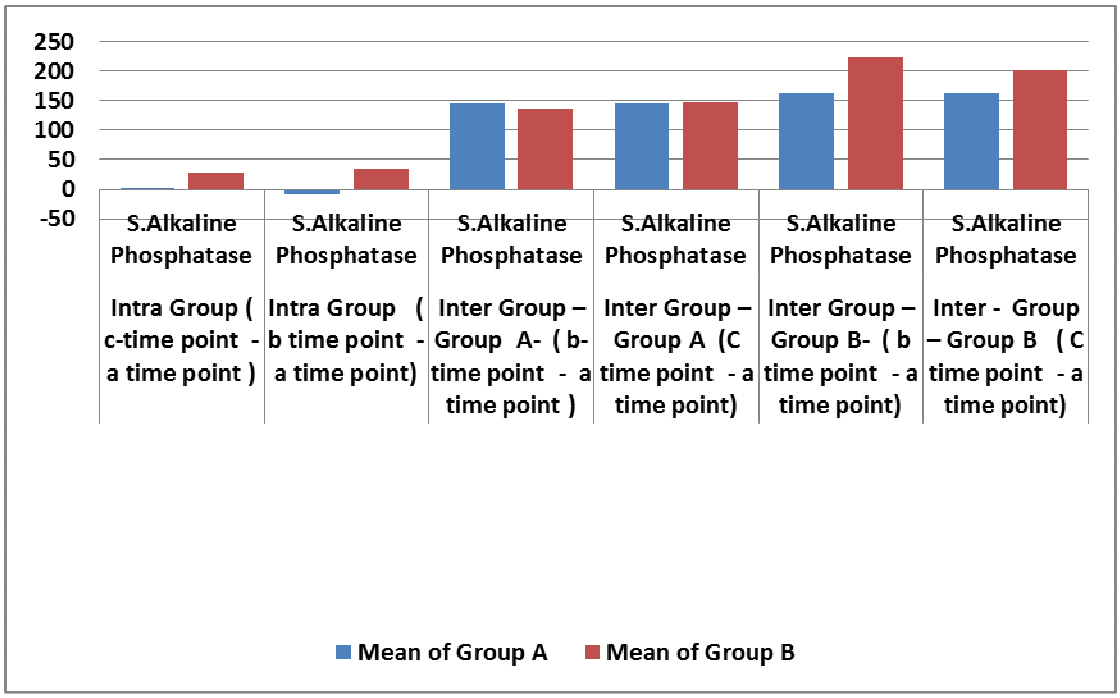
Graph 67 - showing pattern of S.G.P.T. levels of patients in both groups at various time points



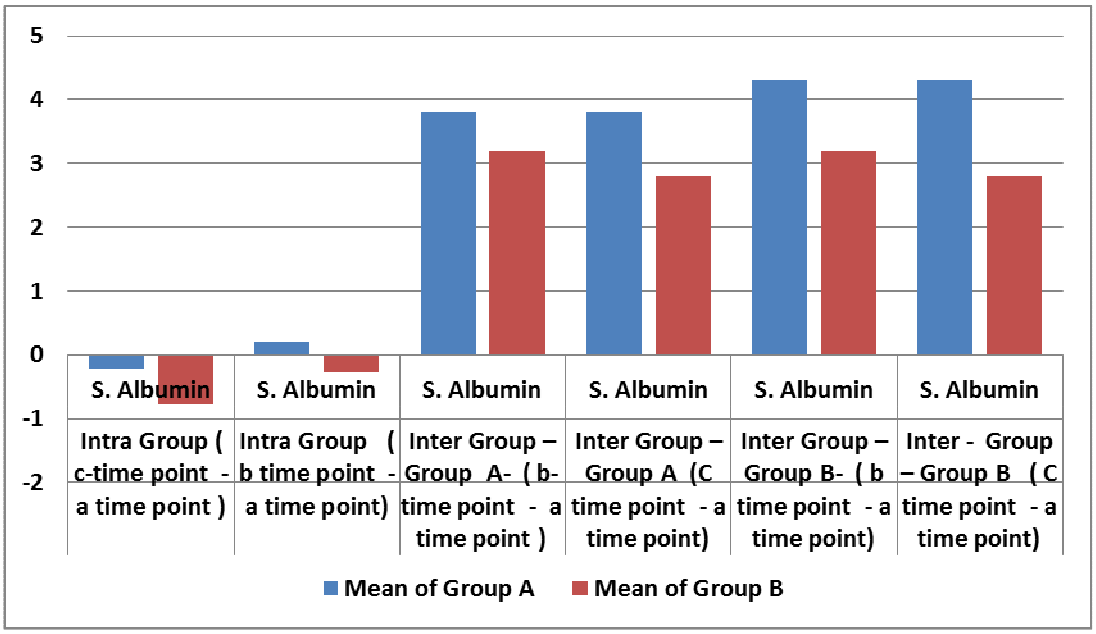
Graph 68 - showing pattern of S.G.O.T levels of patients in both groups at various time points



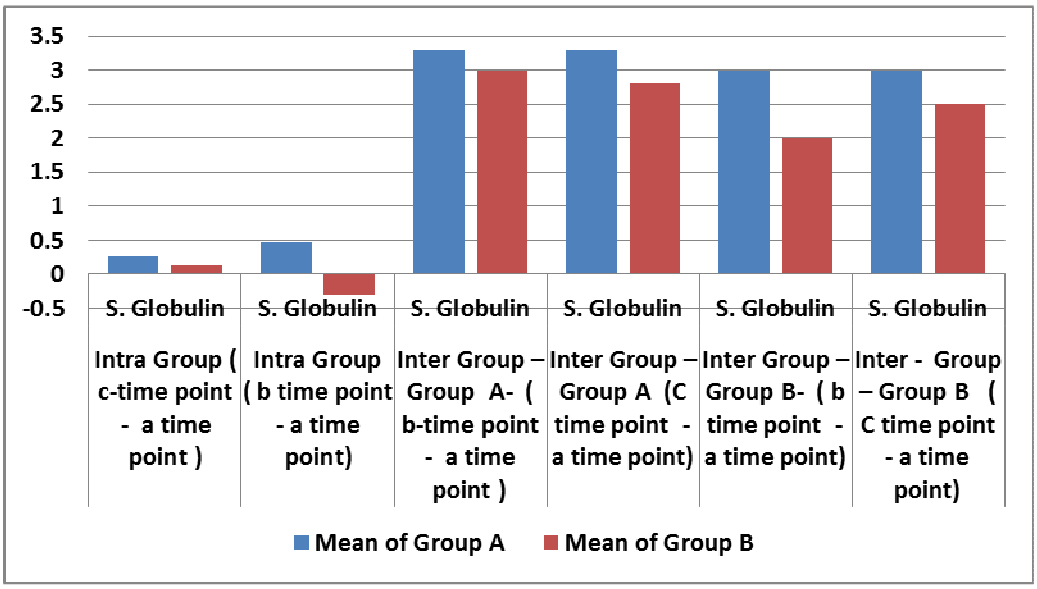
Graph 69 - showing pattern of S.Alkaline phosphates levels of patients in both groups at various time points



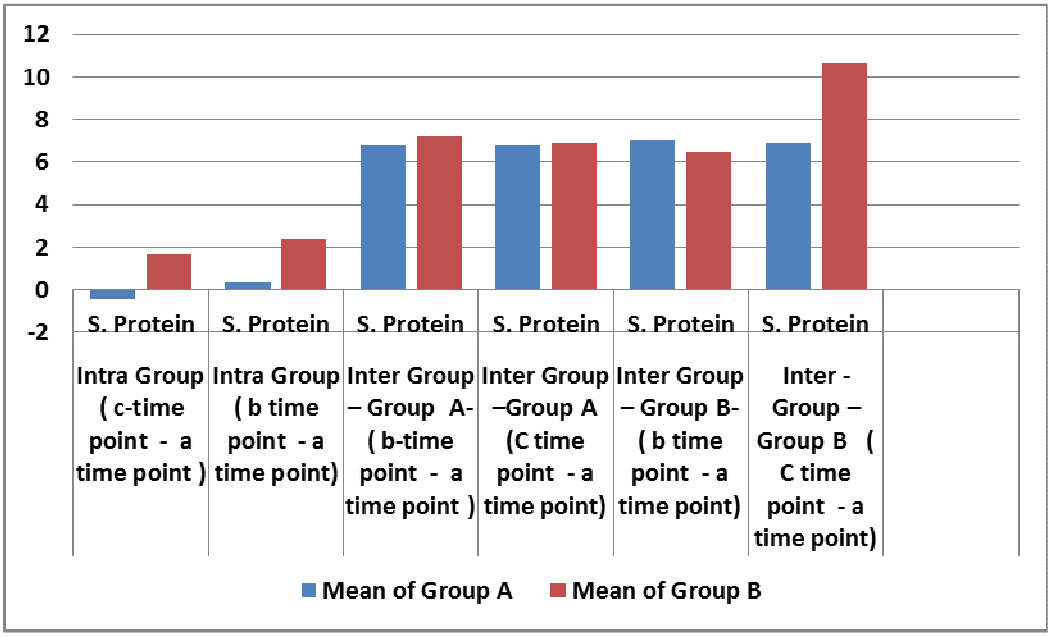
Graph 70 -showing pattern of S. Albumin levels of patients in both groups at various time points



Graph 71 - showing pattern of S Globulin levels of patients in both groups at various time points



Graph 72- showing pattern of S protein levels of patients in both groups at various time points



IV) OBSERVATIONS ON CLINICAL PARAMETERS (ASHTAVIDHA PARIKSHA, WEIGHT AND KARNOFSKY SCORE)

Table 84 – showing effect of Ayurvedic medicines on Nadi

Number of patients						
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	Uttam (1)		**Madhyam (2)**		**Hin (3)**	
Before	**3**	**6**	**18**	**9**	**1**	**11**
After	**7**	**6**	**13**	**11**	**2**	**9**

Graph 73 – showing effect of Ayurvedic medicines on Nadi

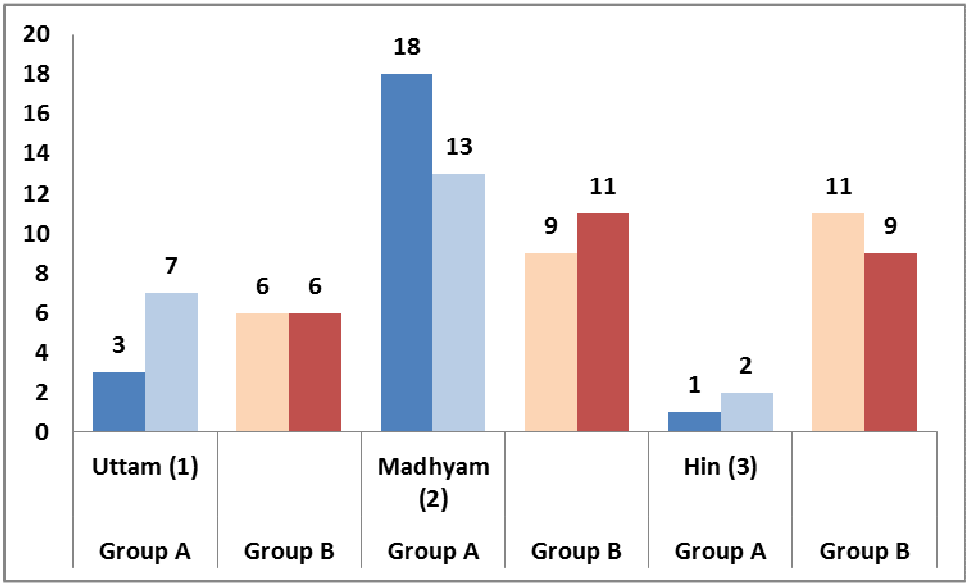


Table 85 - Effect of effect of Ayurvedic medicines on Mala

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	Bandhun (1)		**Grathit (2)**		**Bhasarat (3)**		**Dravmal(4)**	
Before	**10**	**15**	**9**	**8**	**2**	**2**	**1**	**1**
After	**12**	**13**	**6**	**6**	**4**	**4**	**0**	**3**

Graph 74 - Effect of effect of Ayurvedic medicines on Mala

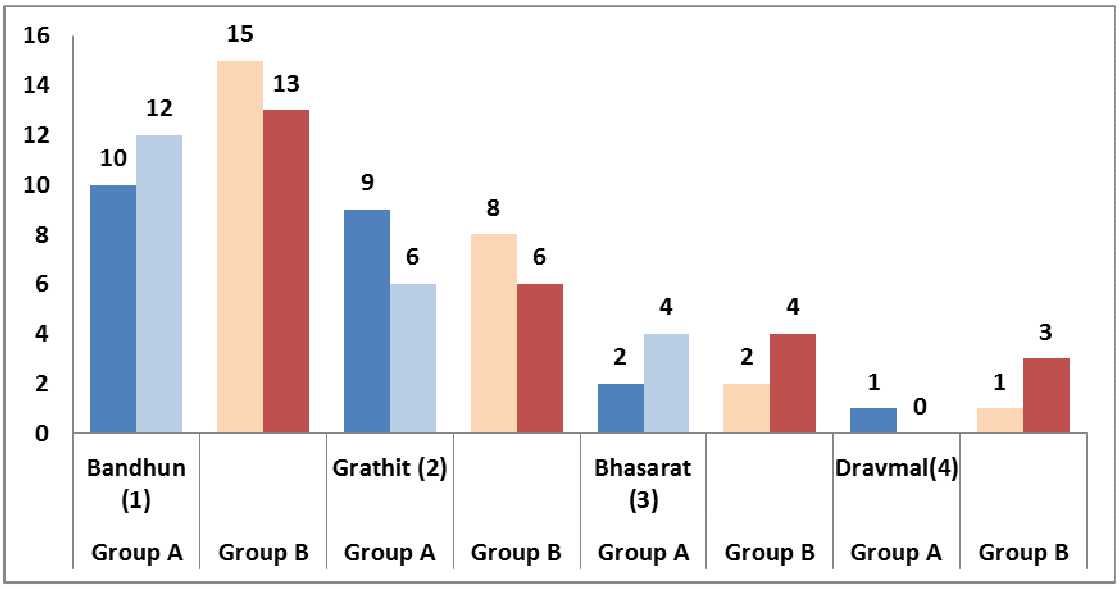


Table 86 - Effect of Ayurvedic medicines on Mutra

	Number of patients			
Time points	**Group A**	**Group B**	**Group A**	**Group B**
	Prakrut (1)		**Vikrut (2)**	
Before	**16**	**16**	**6**	**10**
After	**19**	**18**	**3**	**8**

Graph 75 - Effect of Ayurvedic medicines on Mutra

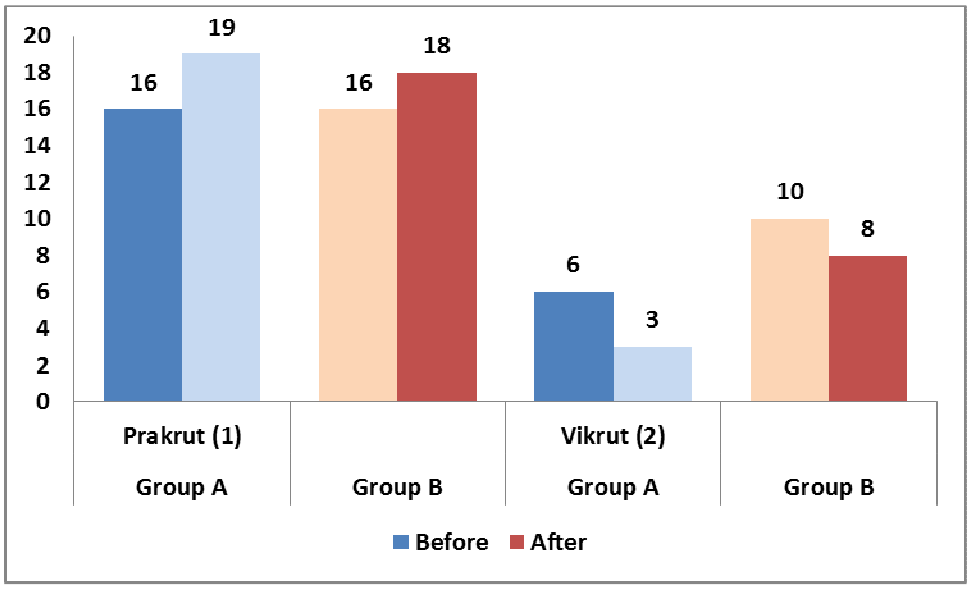


Table 87 – Effect of Ayurvedic medicines on Jivha

	Number of patients			
Time points	**Group A**	**Group B**	**Group A**	**Group B**
	Niram (1)		**Sam (2)**	
Before	**6**	**9**	**16**	**17**
After	**15**	**8**	**7**	**18**

Graph 76 – Effect of Ayurvedic medicines on Jivha

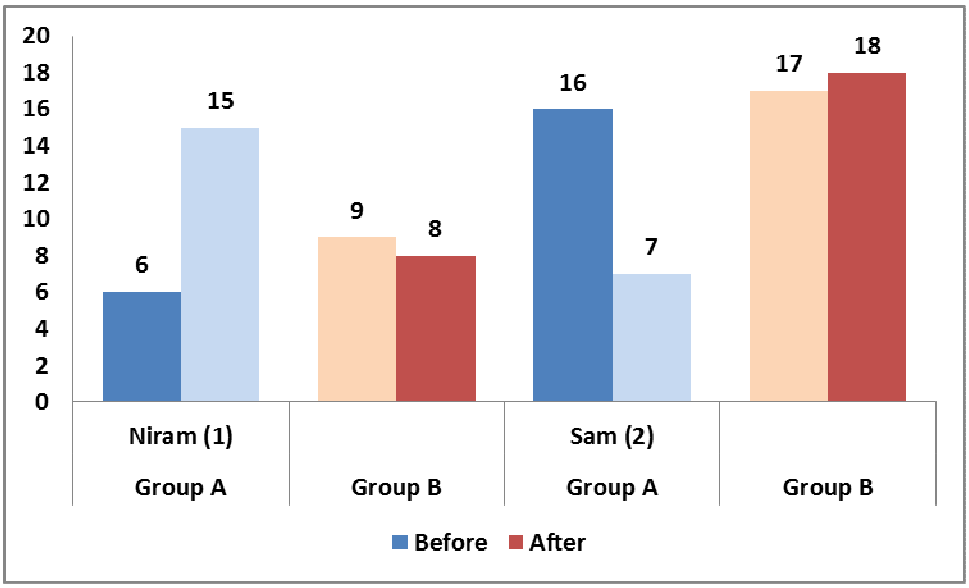


Table 88 – Effect of Ayurvedic medicines on Shabda

	Number of patients			
Time points	**Group A**	**Group B**	**Group A**	**Group B**
	Prakrut (1)		**Vikrut (2)**	
Before	**20**	**21**	**2**	**5**
After	**22**	**21**	**0**	**5**

Graph 77 – Effect of Ayurvedic medicines on Shabda

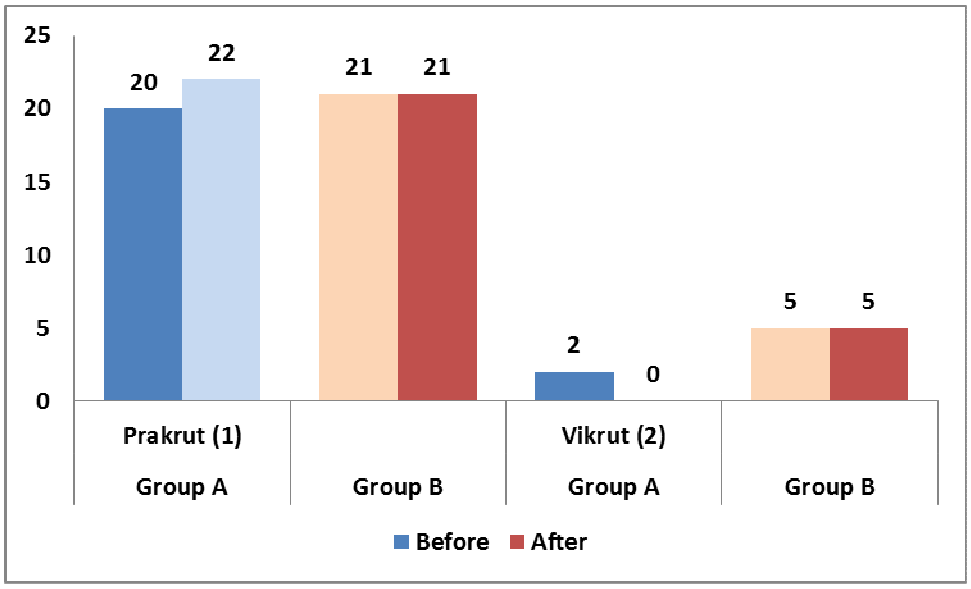


Table 89 – Effect of Ayurvedic medicines on Sparsha

Number of patients						
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	Prakrut (1)		**Ushana (2)**		**Sheet (3)**	
Before	**19**	**19**	**3**	**7**	**0**	**0**
After	**21**	**19**	**1**	**7**	**0**	**0**

Graph 78 – Effect of Ayurvedic medicines on Sparsha

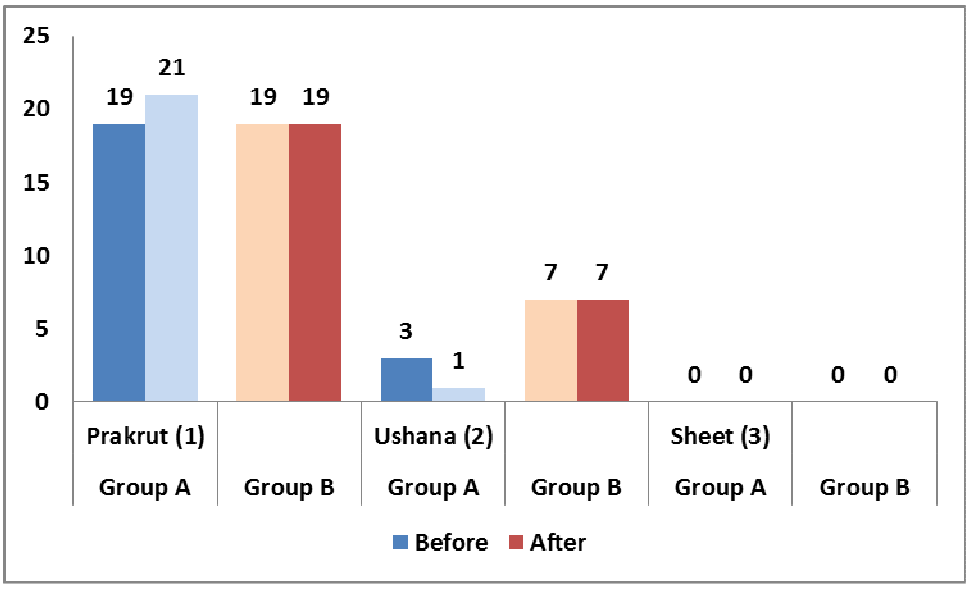


Table 90 - Effect of Ayurvedic medicines on Druk

	Number of patients			
Time points	**Group A**	**Group B**	**Group A**	**Group B**
	Prakrut (1)		**Vikrut (2)**	
Before	**22**	**20**	**0**	**6**
After	**22**	**19**	**0**	**7**

Graph 79 - Effect of Ayurvedic medicines on Druk

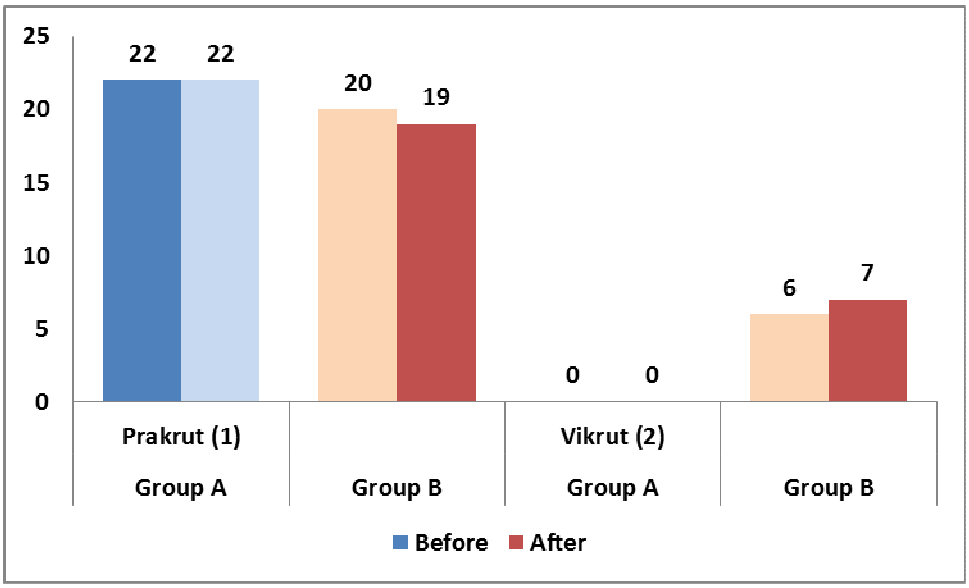


Table 91 – Effect of Ayurvedic medicines on Akruti

Number of patients						
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	Sthul (1)		**Madhyam (2)**		**Krusha (3)**	
Before	**5**	**3**	**17**	**16**	**0**	**7**
After	**5**	**4**	**17**	**15**	**0**	**7**

Graph 80 – Effect of Ayurvedic medicines on Akruti

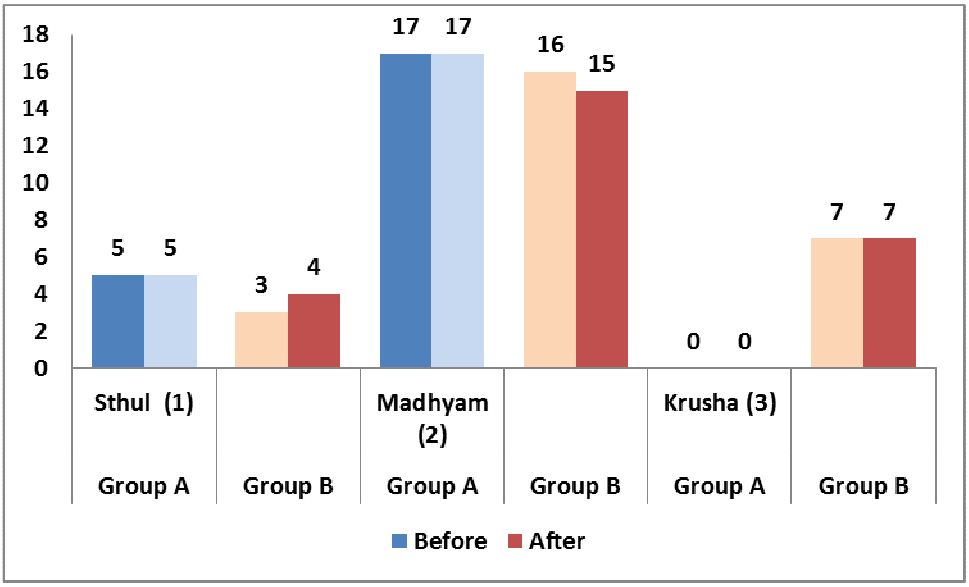


Table 92 – Effect of Ayurvedic medicines on weight

	Number of patients							
Time points	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**	**Group A**	**Group B**
	30-50 kg		**50-60 kg**		**60-70 kg**		**> 70 kg**	
Before	**2**	**5**	**7**	**7**	**8**	**9**	**5**	**5**
After	**4**	**6**	**4**	**8**	**6**	**8**	**8**	**4**

Graph 81 – Effect of Ayurvedic medicines on weight

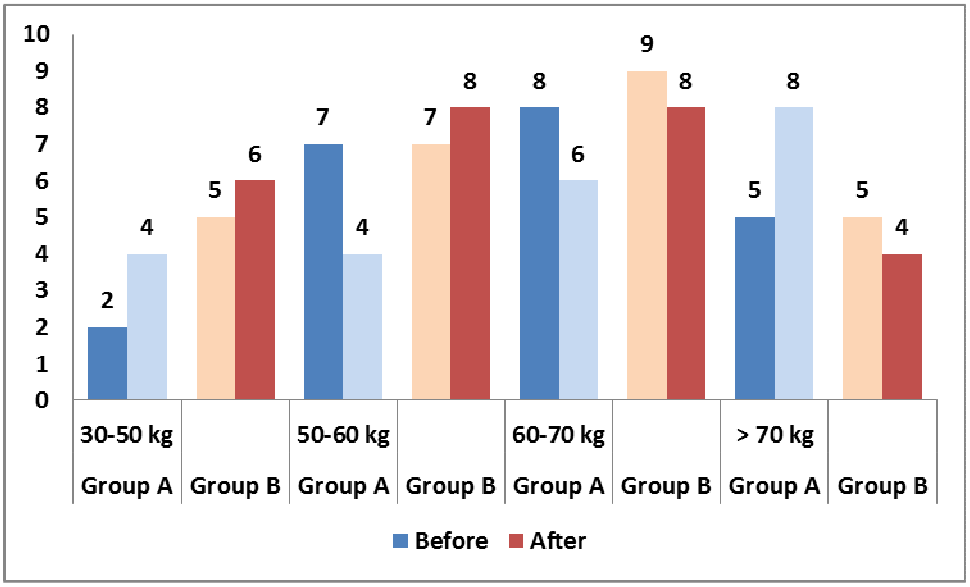
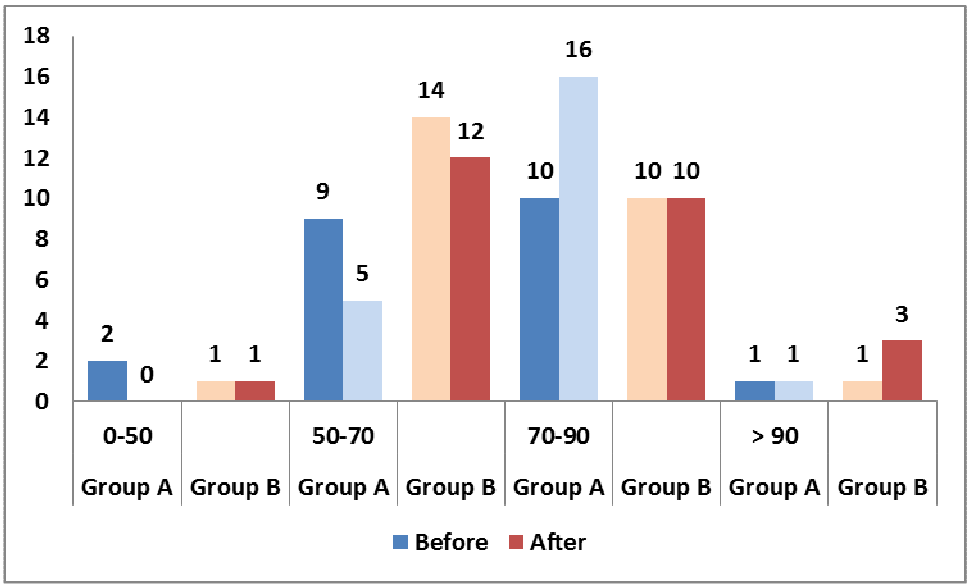


Table 93 – Effect of Ayurvedic medicines on Karnofsky score

Time points	Number of patients							
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
	0-50		50-70		70-90		> 90	
Before	2	1	9	14	10	10	1	1
After	0	1	5	12	16	10	1	3

Graph 82 – Effect of Ayurvedic medicines on Karnofsky score

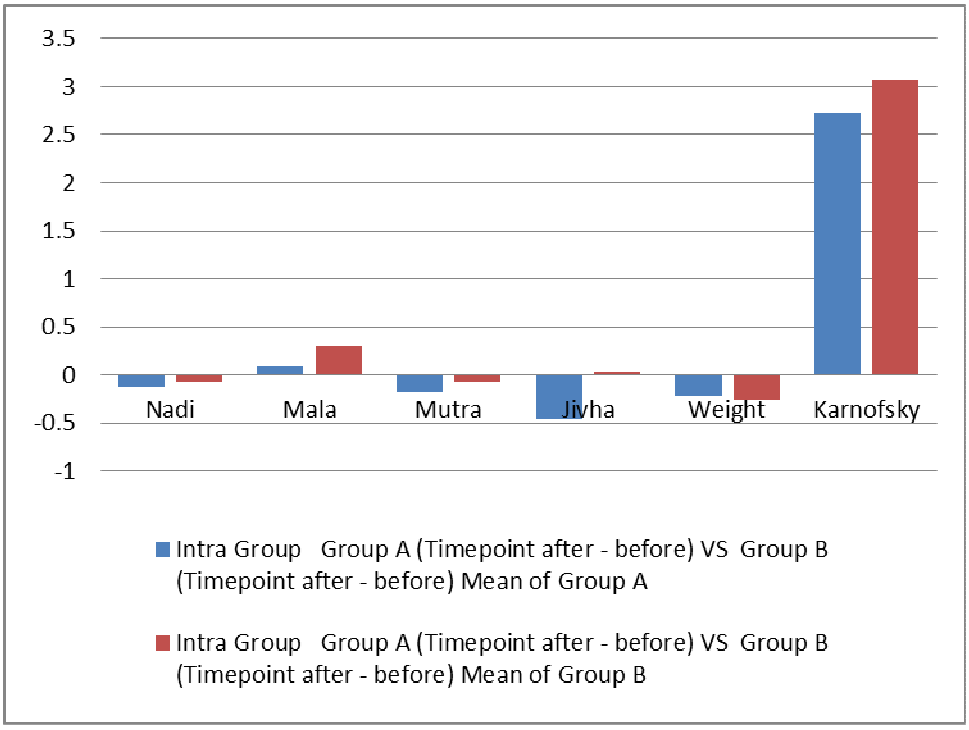


Statistical Analysis – Ashtavidh pariksha and clinical examination

Table 94- Intra Group assessment (Time point After – Time point Before)

	Mean of Group A	**Mean of Group B**	**SD For Group A**	**SD for Group B**	**P**	**Significance**
Nadi	-0.13	-0.07	0.63	0.48	0.71	Not significant
Mala	0.09	0.3	0.86	0.78	0.1	
Mutra	-0.18	-0.07	0.5	0.27	0.36	
Jivha	-0.45	0.038	0.5	0.59	0.0039	Very significant
Weight	-0.22	-0.26	3.3	4.3	0.97	Not significant
Karnofsky	2.72	3.07	19.5	10.4	0.93	Not significant

Graph 83 - Intra Group assessment [Time point After (c) – Time point Before (a)]



Statistical Analysis of - Intergroup Assessment of Karnofsky score

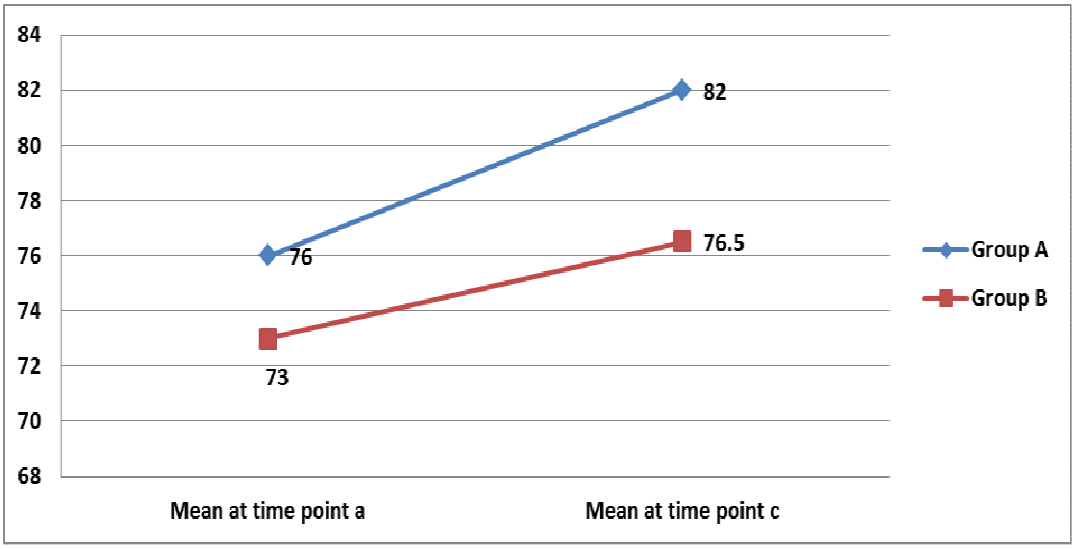
Table 95 - Inter Group assessment of Group A [Time point After (c) – Time point Before (a)]

Group A					
Mean at time point a	**Mean at time point c**	**SD at time point a**	**SD at time point b**	**p Value**	**Significance**
76	82	9.5	10.4	0.0403	Significant

Table 96 - Inter Group assessment of Group B [Time point After treatment (c) – Time point Before treatment (a)]

Group B					
Mean at time point a	**Mean at time point c**	**SD at time point a**	**SD at time point b**	**p Value**	**Significance**
73	76.5	10.9	15.2	0.4064	Not Significant

Graph 84 – Inter group assessment of group A and group B at time points a and c



DISCUSSION

A) Discussion on demographic data :

- Patients of Yakrutvikara like Hepatocellular carcinoma, cirrhosis, hepatomegaly, hepatitis B and C and liver metastasis are included in the study.
- Proportion of patients of Yakrutvikara in both groups is higher in age group between 41 yrs to 80 yrs.
- In both the groups number of male patients were more than that of female patients. This may be due to the fact that the addiction is more common in males, which is the main cause of Yakrutvikara.
- In study group (Group A) socio-economically middle class patients were more (10), while lower class patients were more in Group B (13). This observation is due to random selection of patients.
- In our study 12 patients in Group A and 13 patients in Group B had history of addiction. Alcohol consumption and multiple addictions are very common risk factors of Yakrutvikara.
- Addiction in the form of alcohol, tobacco, smoking, betal nut etc is a prevailing cause of Yakrutvikara. These dravyas are Ushna, Tikshna, Vyavayi and due to these propertiesthey vitiate Pitta dosha and Raktadhatu. Yakrut being abode of Raktadhatu, addiction is an evident cause of Yakrutvikara.
- Green chilli, curd, pickle, non-vegetarian food, tomato and red chili powder are observed as major risk factors in Yakrutvikara.Agnidushti plays an important role in Samprapti of Yakrut Vikara. Thus abnormal dietary items, when consumed in excess and frequently, causes Agnimandya, Raktadushti and leads to Yakrutvikara.
- Heredity is an important risk factor of Yakrut vikara. Yakrut being Matruja avayava, heredity of diseases of matruja avayava is commonly seen in Yakrut vikara.
- In our study heredity of CA stomach, CA breast, cirrhosis, Hepatocellular carcinoma, CA throat, CA oesophagus was observed patients of Yakrut vikara.
- Abnormal vihara is responsible forAgnidushti and Raktadushti. Diwaswapa, Vegavarodha, Ati - chankramana, long lasting contact with chemicals and

sedentary lifestyle are observed as major risk factors of Yakrutvikara in our study.

- Mental stress is a cause of Rasa and Raktadhatudushti, leading to Yakrutvikara. In our study, 50% patients in both the group had excessive mental stress. Among manasabhava, chinta (worry) is found to be evident risk factor of Yakrutvikara.

B) DISCUSSIONS ON SYMPTOMSOF YAKRUT VIKARA:

Signs and symptoms of Yakrutvikara from Ayurvedic and Allopathic perspective like Udarshool / Yakrutpradeshishul, Yakrutplihavrudhi, fullness, abdominal swelling, Agnimandya, Aruchi, Hrullas, Avipaka, vomiting, malaise, Shithila Mala pravrutti, Netrapitata, Twakpitata, itching, jaundice, Sharira Shaithilya, weakness, Daha, Hatendriya, Bhekavarnata, weight loss, bone pain, tremors, disorientation were assessed in patients at 3 time-points, a=before starting Ayurvedic treatment, b=mid of Ayurvedic treatment and c=at the end of Ayurvedic treatment. Each symptom is graded as per gradings mentioned in materials and methods at all these 3 time-points.

Man Whitney Z test for symptoms, unpaired t Test for percentage of weight loss and Chi square test for comparison of values in both the groups are used for statistical analysis.

- **Udarashoola / Yakrutpradeshishoola**

Udarashoola / Yakrutpradeshishoolaisare the common symptoms of Yakrutvikara. It is mainly caused due to accumulation of dushtadoshas in Yakrut and udara.

YG2 contains combination of Yakrut Pleehari Loha and Kutki, which has a bhedana action on mala especially in bowel, whereas YG3 contains combination of Yakrut Pleehari Loha, Kutki and Kumari Kalpa, which possesses bhedana action on doshas and malas accumulated in Yakrut as well.

This is due to Yakrutgamitva and Bhedana property of Kumari, which is the main ingredient in Kumari Kalpa Vati.

- **Yakrut Pleeha Vruddhi**

  Yakrutpleehavruddhi is developed in many patients of Yakrutvikara due to vitiation of Raktadhatu. Yakrut and pleeha being mulasthana of Raktavahasrotasa, vitiated Raktadhau gets accumulated in Yakrut and Pleeha leading to Yakrutpleehavruddhi. Yakrutpleehari Loha and Kutki are mainly improving function of Raktadhatu, Yakrut and Pleeha.

  Majority of the contents of KumariKalpa (Kumari, Haritaki, Pippali and Sharapunkha) are directly mentioned as Yakrutpleehavruddhinashakie reducing hepatomegaly and splenomegaly.

  Due to bhedana action of Kumari and Kutki, vitiated and accumulated Doshas and Malas in Yakrut and Pleeha are eliminated through stool. This process helps to detoxify liver and spleen; improve functions of Pitta dosha and Raktadhatu and thus to reduce the enlargement of liver and spleen.

- **Agnimandya**

  Yakrut being a site of Agni, Agnimandya is an important factor in Samprapti of Yakrutvikara.

  YG3 contains additional medicine Kumari Kapla, which contains Agnideepanadravya like Shunthi, Maricha, Pippali and Vidanga along with Bhedanadravya like Kumari and Anulomanadravya like Haritaki. Thus significant statistical improvement in Agnimandya is observed in Group A at both time points –c v/s a ($p<0.0001$) and b v/s a ($p=0.006$).

- **Avipaka**

  Mandagni being an important pathological factor in Samprapti of Yakrutvikara, Avipaka is a commonly observed in these patients.

  Thus KumariKalpa containing Pachakadravya like Musta, Shunthi, Maricha, Pippali and Vidanga is effective in relieving Avipaka in patients of Yakrutvikara.

Due to this specific action of Kumari Kalpa in addition to Bhedana action, effect of YG3 is found to be statistically significant in Avipaka at both the time points b and c (p=0.008 at b and p=0.0002 at c).

- **Daha**

  Daha is associated with Pitta vruddhi and Raktadushti, which could be managed with Kumari Kalpa which contains sheeta and Pittashamakdravya like Kumari and Bhumyamalaki.

  Thus YG3, a combination of Yakrutpleehari Loha, Kutki and Kumari Kalpa is found to be effective in management of Daha (p=0.04 at time point c)

- **Fullness**

  Fullness in abdomen is the commonly seen symptom in patients of Yakrutvikara due to accumulation of vitiated doshas in Pakwashaya and Yakrut.

  Bhedanadravya like Kutkiis beneficial to relieve this symptom due to evacuation of mala through anus. Kumari Kalpa, which contains a substantial amount of Kumari, possesses Yakrutgamitva and Bhedana action.

  Thus fullness in abdomen is significantly relieved in study group patients who are treated with YG3. Statistically efficacy of YG3 is proved with very significant p values at time points b (0.003) and c (0.0011).

- **Abdominal swelling**

  Abdominal swelling in Yakrutvikara is mainly due to Yakrut Vruddhi. Very significant improvement in abdominal swelling (p=0.003) is found in group A patients at time point b. This is due to bhedana and Yakrutgami action of YG3.

- **Bone pain**

  Efficacy of YG3 on bone pain is found to be statistically significant (p=0.02) at time point c in group A.

Bone pain in Yakrutvikara is seen due to uttarottardhatukshaya (i.e. Mansa kshaya, Asthikshaya due to Margavarodha of Raktadhatu).

YG3 due to its Bhedana, Yakrutgami and Rasayana action, helps to eliminate DoshaSanghata and Margavarodh. It is thus pacifies prakupitaVata and beneficial in management of bone pain.

- **Weakness**

  Preenana (nourishment) and jeevana (vitality) are the karma of Rasa and Raktadhatu respectively. Sharirabala (strength of a person) mainly depends on normal state of Rasa and Raktadhatu.

  In Yakrutvikara, weakness is seen in many patients due to dushti of Rasa and Raktadhatu jeopardizing their normal functions. Suvarna and Raupyabhasmain YPL and Kumari in KumariKalpa are Rasayana and thus give strength to all dhatus, especially Raktadhatu.

  Thus YG2 and YG3, both are observed to be useful in reducing weakness.

Discussion on symptoms which did not significantly respond to YG2 and YG3

Group A-Internal Group assessment of Symptoms (Time point c to a)

Aruchi, Hrullas, Sharira Shaithilya, Hatendriya, Bhekavarnata, Shithila Mala Pravrutti, Mala Pitata, Mutra Pitata, Weight loss were the symptoms which were not found to be statistically significant when assessed at time points c. The patients in group A were receiving combination of YG3. The above symptoms doesn't show any significant change at the end of treatment. It explains that YG3 does not have a significant effect on them.

Group B -Internal Group assessment of Symptoms–(Time point c to a)

Aruchi, Hrullas, Sharira Shaithilya, Hatendriya, Bhekavarnata, Shithila Mala Pravrutti, Daha, Mala Pitata, Mutra Pitata, Netra Pitata, Twak Pitata, Nakha Pitata, Mukha Pitata, Vomitting, Weight loss were the symptoms which were not found to be

significant when assessed at time points c in group B. Patients in group B were receiving combination of YG2.

Intra Group assessment of Symptoms -Group A (c-a) to Group B (c-a)

Intra group assessment of symptoms in both the groups A and B did not show a significant results in symptoms namely Aruchi, Hrullas, Sharira Shaithilya, Hatendriya, Bhekavarnata and Shithila Mala Pravrutti, indicating very minimal efficacy of YG2 and YG3 on these symptoms

Haematemesis, Tremours and Disorientation were not present in any patient from either group A or group B.

C) DISCUSSION ON LABORATORY TESTS CARRIED OUT IN YAKRUT VIKARA:

Biochemical parameters which are likely to become abnormal in Yakrutvikara, based on the pathogenesis of Yakrutvikara were selected as assessment criteria in the study. Thus Hemogram (Hemoglobin, White Blood Cell count, Platelets), Liver Function test (Serum bilirubin, SGOT, SGPT, Serum Alkaline phosphatase, Serum Protein Serum Albumin, Seum Globuline) were carried out in patients of both groups before, mid and at the end of Ayurvedic treatment.

- **Discussion on the levels of Hemoglobin**

  Drop out in number of patients in sub group 2 (having low Hb) at Time – points b and c as compared to Time – point a indicates the efficacy of YG3 combination in increasing Hb level as compared to YG 2 combination in patients of Yakrut Vikar.

  At the same time rising trend of patients in sub group 3 (having adequate Hb) in study group also indicates the effectiveness of YG3 combination in increasing Hb level as compared to YG 2 combination in patients of Yakrut Vikar.

  YG3 and YG2 both Ayurvedic medicines contain Kutaki as a common Bhedan Dravya while YG3 contains Kumari as an additional Bhedan Dravya which enhances the effect of Bhedana Karma of YG3.

Bhedana Dravyas, as per it's definition and commentary of Adhamalla on Sharangdhara Samhita, have a peculiar property of eliminating the accumulated, blended and vitiated Doshas and Malas through evacuation of bowel.

Action of Bhedana Dravya is also extended on various Koshthanga where doshas and Malas are accumulated, blended and vitiated.

While describing Bhedana Karma, Sharangdhar has quoted Kutaki as an example. It means Kutaki shows wide range of Bhedana Karma in the body. But when the action of Bhedana on specific Koshthanga is expected, organ - specific Bhedana Dravya should be chosen to gain the additional Bhedana effect. Yakrutgamitva & Bhedana Karma of Kumari serves both the purposes in Yakrut Vikar where vitiated Doshas are accumulated.

A combination of YPL, Kutaki and Kumari eliminates accumulated and vitiated Doshas and Malas in Yakrut and thus detoxifies it.

Yakrut is mentioned as the moolasthana of Raktavaha Strotas. Once it is detoxified, a process of formation of Shudhha Rakta (blood having good oxygen binding capacity) is achieved. This explains the observation of increase in Hb in experimental group as compared to control group.

- **Discussion on Serum bilirubin levels**

Elevated bilirubin is observed in two conditions –

1) Excessive break down of RBCs (Haemolysis) leading to excessive formation of haem which afterwards converted into bilirubin.
2) Accumulation of excessive bilirubin in the liver due to less excretion through urine & stools.

Elimination of excessive bilirubin in the liver is achieved with Bhedana Karma of Kutaki whereas Kumari serves both the purposes i.e. it helps in restricting the excessive haemolysis due to its properties of Rakta and Pitta doshanashaktva

and Yakrutplihagamitva and elimination of excessive bilirubin through faeces with its Bhedan Karma.

Our observations regarding comparison of total bilirubin levels in both the groups also support the efficacy of YG3. We observed that less number of patients remained in the normal range of total bilirubin in Group B as compared to Group A at time – point c [Group A 12(a)→ 11(c) and Group B 17(a) → 13(c)].

We also observed the declining trend of number of patients at time –points b & c in Group A having elevated bilirubin levels at time –point a. On the contrary rising trend of number of patients was observed at time –points b & c in Group B having elevated bilirubin at time –point a.

- **Discussion on the levels ofSGOT and SGPT:**

  Serum glutamic oxaloacetic transaminase (SGOT) and Serum glutamic-pyruvic transaminase (SGPT) are the liver enzymes. Among them SGOT is an exclusive liver enzyme. While SGPT is also present in liver and other parts of the body like heart, kidney and skeletal muscles.

  Thus elevated levels of SGOT and SGPT are mainly indicative of liver disorders.

  In our study elevated levels of SGOT & SGPT are sub divided in 3 subgroups depending upon level of elevation as mentioned in observations and results.
  Effectiveness of YG3 on SGOT and SGPT was observed in sub group 3 and 4 respectively indicating usefulness of YG3 when levels of SGOT and SGPT are very high.

  YG3 due to being a unique combination of Yakrutpleehari Loha, Kutki and Kumari Kalpa, seems to be improving functions of liver, probably by increasing liver enzymes.

- **Discussion on the levels ofAlkaline phosphatase:**

  Serum alkaline phosphatase is produced by liver, bone, and placenta. It is excreted in the bile.

  An elevated alkaline phosphatase levels, in the absence of bone diseases and pregnancy generally reflects hepatobiliary diseases v.i.z. biliary tract obstruction, hepatitis, cirrhosis and metastatic liver disease.

  In our study 3 patients of Group A and 8 patients of Group B had elevated levels of alkaline phosphatase at time – point a, but during the course of treatment we have not found significant difference in patients having elevated levels of alkaline phosphatase in both the groups.

- **Discussion on the levels ofProteins:**

  In our study most of the patients of Group A and B laid in normal range of serum protein. Those who were presented with hypoproteinemia were responded well with YG3 as compared to YG2.

  This can be explained with the additional organ specific Bhedana effect of Kumari in YG3.

- **Discussion on the levels ofAlbumin :**

  Albumin is prepared mainly in the liver. It is responsible for tissue growth and healing. Hypoalbuminemia is caused mainly due to acute and chronic inflammation. It is commonly seen in hepatic cirhhosis, nephrotic syndrome, heart failure & malnutrition.In our study it is observed that number of patients of hypoalbuniemia have been reduced in Group A at time – point c as compared to time – point a. On the contrary number of patients of hypoalbuniemia have been increased in Group B at time – point c as compared to time – point a. This indicates efficacy of YG3 on treating acute and chronic inflammation responsible for improvement in liver function in the form of tissue growth and healing.

Though the condition hyperalbuminemia is also associated with liver disorders i.e. hepatitis, cirrhosis, hepatocellular necrosis, it is not possible to comment on role of YG3 in this condition due to absence of patients of hyperalbuminemiain group A at all three time – points and to compare them with Group B patients.

- **Discussion on the levels ofGlobulin:**

  Hypoglobulinemia is associated with weak immune system and malabsorption of lipids and fat soluble deposits which is a common condition in of Yakrutvikar.

  In our study a drop in number of patients with hypoglobulinemia was observed in Group A patients while studying at time – points a, b & c successively. Number of patients with hypoglobulinemia remained same in group B at time – points a & c.

  As YG3 contains KumariKalpa (consisting Kumari in half quantity), it is beneficial to improve immune system due to Rasayan action of Kumari. Kutaki along with Kumari in YG3 also acts as Bhedana leading to elimination and detoxification of accumulated and vitiated Doshas in Yakrut. Once the organ is detoxified, a process of absorption of lipids and fat soluble vitamins is accelerated. This explains the improvement in globulin levels in our study group patients.

D) Discussion on Ashtavidha Pariksha :

Ashtavidhaparikshas are the tools of clinical examination of a patient from Ayurvedic perspectives. They comprise examination of Nadi, Mutra, Mala, Jivha, Shabda, Sparsha, Druk and Akruti. In our study, Ashtavidhapariksha of each patient in Group A and B was carried out at every follow – up. Analysis of Ashtavidhapariksha is done before and at the end of the study.

- **Nadi Parikshan** :

  It was examined on the basis of it's bala as – Uttamabala Nadi, Madhyamabala Nadi and Hinabala Nadi. Statistically no significant difference in Nadibalawas observed in both groups.

- **Mutra Parikshan** :

  It was examined as prakrut and vikrut. Bahumutrata, alpamutrata, mutrakrushrata, mutraghat, mutrapitata, mutraaaraktata were considered as Vikrutmutra.

6 patients in Group A and 10 patients in Group B had vikrutmutrata at time – point a (beginning of treatment). Number of patients reduced to 3 and 8 in Group A and B respectively at time – point c (at the end of treatment).

In the category vikrutmutra majority of patients were having Mutrapitata. Mutrapitata is one of the important symptoms of Yakrut Vikara.

Reduction in number of patients of vikrutmutrapravrutti at time – point c indicates efficacy of YG2 and YG3 in Yakrut Vikara.

- **Mala Parikshana** :

  It was divided in four types according to the consistency as well formed, hard, semi – solid and loose stools.

  Effect of PurishaBhedana was observed similar in both the groups as Kutki is a common content of medicines in both groups, which has Mala bhedana action

- **Jivha Parikshan**:

  In Ayurvedic method of examination, Jivhaparikshan is categorised as Saam (Coated) and Niraam (Clean).

  SaamJivha is seen in various disorders of digestive and excretory system whereas Niraamjivha is indicative of proper functioning of Jatharagni and digestion.

  Agnidushti is the main cause of all sorts of Yakrutvikar. Thus jivhaparikshan is essential for the assessment of treatment response in Yakrutvikar.

  In our study statistically very significant difference was found in nature of Jivha of Group A patients (P = 0.0039)

E) DISCUSSION ON CLINICAL EXAMINATION :

- **Body weight :**

  Excessive weight gain and weight loss is was observed in Yakrutvikar due to yakrutplihavrudhhi and dhatukshaya respectively.

  No significant difference in weight was observed in both the groups.

- **Karnofsky Score :**

  Karnofsky score indicates general well-being of patients.

There is not much statistical difference in Karnofsky scores of Group A and B at the end of treatment (intra group assessment), who received a common immunomodulatory medicines in YPL.

However, a significant improvement in Karbofsky score (p=0.403) was observed in group A patients, when comparison was done at the end (time point c) and in the beginning of the treatment (time point a). This significant difference is not seen in group B patients.

Improvement in general well-being is mainly achieved by Rasayana medicines.
In our study medicines, 4 dravyas lie in the category of Rasayana i. e. Suvarna and RaupyaBhasma in YPL (one of the common medicines in YG2 and YG3) and Kumari and Pippali in Kumari Kalpa (One of the medicines of YG3).

Among them Suvarna and Raupya Bhasma are Kamya Rasayan that means they act as Sarvadhatu Rasayan boosting total immunity of the body whereas Kumari and Pippali in Kumarikalpa act as Naimittik Rasayan that means they improve functions of specific organ i.e. Yakrut.

Significant improvement in Karnofsky score during inter group assessment in group A (time point c v/s a) is attributed to additive effect of Naimittik Rasayana in YG3 along with Kamya Rasayana.

F) DISCUSSION ON BHEDANA KARMA OF YG3 AND YG2 IN YAKRUT VIKARA

Rechana – The medicine which liquefies Pakwa (digested) of Apakwa (Undigested) doshas and malas and evacuated through bowel, it is termed as Rechana. E.g. Trivrut

Anulomana – The medicine which makes Paka of doshas and malasie.pacify them, breaks their blending thoroughly and then eliminate them from bowel is known as Anulomana . E.g. Haritaki

Sransana – The medicine which evacuates doshas and malas through bowel, which are Paktavya (i.e. undigested and in the process of digestion) and thus are existing in the Kostha, are known as Sransana. E.g. Aaragwadha

Bhedana –The medicine which breaks badhha /abadhha /pindita/ grathita /ruksha/ shushka and dushitadoshas and malas and eliminates through bowel, is called as Dhedana. E.g. Kutaki

These are the definitions of 4 varieties of Virechanadravya as mentioned in SharagdharaSamhita (with Deepika and Gudharthadeepika commentary).

Yakrutvikara are mainly outcome of vitiation of Pitta Dosha and Raktadhatu which are presented in various forms like baddha, abadhha, grathita. Dushtadoshas and malas are accumulated in liver causing various types of liver disorders. Thus all of them should be eliminated through bowel, which can be better achieved by bhedana type of Virechanadravya.

In our study, study and control both groups were treated with a common bhedanadravya – Kutki. Study group patients were additionally treated with Kumari Kalpa containing Kumari (Aloe vera) as a main content and possessing Bhedana karma. A peculiarity of Kumari is that, it is Yakrutgami and performs bhedana of accumulated and vitiated doshas and malas from liver as well as from bowel. Thus YG3 seems to be possessing effective bhedana karma and is more beneficial in Yakrutvikara as compared to that of YG2.

CONCLUSION

- YG3 (A combination of Yakrut pleehari Loha, Kutki churna and Kumari KalpaVati) is found to be effective in relieving symptoms namely Udarashool/Yakrutpradeshi Shoola, Agnimandya, Avipak, Fullness of abdomen, Malaise and Weakness in the mid of the treatment in Group A patients. This improvement was also supported by statistical analysis.
- Improvement in symptoms of Udarashoola, Agnimandya, Avipak, Fullness of abdomen, Malaise and Weakness were also observed at the end of the treatment.
- Patients in Group B treated with YG2 (A combination of Yakrut pleehari Loha and Kutki churna) were symptomatically relieved in Udarashool/Yakrutpradeshi Shoola, Hrullas and Weakness.
- Patients in Group A showed statistically very significant results in Yakruplihavrudhhi, Hrullas, Sharirshaithilya, Daha, Fullness and Abdominal swelling at mid of the treatment.
- At the end of the treatment patients in Group A had statistically significant results in symptoms–Yakruplihavrudhhi, Daha and Fullness.
- Statistical analysis of Laboratory investigations when conducted between Group A and Group B, significant improvement in Hemoglobin in the mid of the treatment was observed.
- Improvement in quality of life and feeling of well-being assessed by Karnofsky score were observed in group A patients at the end of treatment.
- Bhedana action of YG3 in Yakrut Vikara is found to be evident as compared to YG2.

SCOPE OF FURTHER STUDY

- Bhedana action of YG3 and YG2 has to be assessed on individual Yakrut Vikara i.e. separately on Liver cirrhosis, HCC, Liver metastasis, Hepatitis etc.
- More precise radiological and pathological assessment tools like Liver scan, AFP can be used to assess efficacy of Ayurvedic medicines.

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Tilak Maharashtra Vidyapeeth, Pune

The Late Vaidya P.G.Nanal Department of Ayurveda

CASE RECORD FORM

Title – Evaluation of Bhedana Karma of YG3 in Yakrut Vikar

Name of Scholar – Vd. Bhagyashree S.Sardeshmukh

Name of Guide – Dr. Vineeta V. Deshmukh

Date:

Name of patient: **OPD No. :**

Address:

Contact No. :

Sex: **Occupation:**

Qualification:

Type of Work: **Work Duration:**

Vartaman Vyadhivrutta:

Purvavyadhi

Kulaja itihas - Swakula /Pitrukula /matrukula

Vyasan

Alcohol /Betel nut / Betel leaf/Tobacco/ Cigarette / Gutakha / Others

Praman :

Purva Chikitsa / Purva shastrakarma

Indriyaparikshan

Dnyanendriya -

Karmendriya –

- **Strotas Parikshana-**

1. Pranavaha strotas -

2. Udakvaha strotas -

3. Annavaha strotas -

4. Rasavaha strotas -

5. Raktavaha strotas -

6. Mansavaha strotas -

7. Medovaha strotas -

8. Asthivaha strotas -

9. Majjavaha strotas -

10. Sukravaha strotas -

11. Aartavaha strotas -

12. Purisavaha strotas -

13. Mutravaha strotas -

14. Swadevaha strotas -

- **Nidanpanchak**

1. Hetu

   Aaharaja -

   Viharaja

   Manasik

2. Purvarupa

3. Rupa

4. Upashaya / Anupshaya

5. Samprapti

Chikitsa - Group –A/ Group B

Ashtavidha Pariksha -

Nadi – **Shabda -**

Mala- **Sparsha –**

Mutra – **Druk -**

Jivha – **Aakruti -**

Clinical Examination -

Pulse - **B.P. –** **Wt. -**

R.S. - **CVS -**

P/A - **CNS –**

Karnofsky - **ECOG -**

Assessment Of Bhedana Karma -

Sr. No.	Symptoms (Present / Absent)	Months						
		Baseline	1	2	3	4	5	6
1	Netra Pitata							
2	Twak Pitata							
3	Nakha Pitata							
4	Mukha Pitata							
5	Mala Pitata							
6	Shithila mala Pravrutti							
7	Mutra Pitata							
8	Bhekavarnata							
9	Hatendriya							
10	Daha							
11	Avipaka							
12	Sharir Shaithilya							
13	Aruchi							
14	Yakrut Vrudhhi							
15	Yakrutpradeshi Shoola							

According to Modern Science :

Symptom	Months													
	Baseline		1		2		3		4		5		6	
	Severity	Duration	Severity	Duration	Severity	Duration	Severity	Duration	Severity	Duration	Severity	Duration	Severity	Duration
Abdominal pain														
Weight loss														
Weakness														
Malaise														
Fullness														
Anorexia														
Abdominal swelling														
Hepatomegaly														
Tenderness in the liver														
Jaundice														
Vomiting														
Hematemesis														
Bone pain														

Respiratory symptoms														
Asthenia														
Itching														
Tremors														
Disorientation														

Signature of Guide **Signature of Student**

PATIENT CONSENT FORM

Title of the Study: 'Evaluation of Bhedana Karma of YG3 in Yakrut Vikar'

Name of the Participant:

Documentation of the informed consent:

I, ……. have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.
4. My rights and responsibilities have been explained to me by the researcher.
5. I have been explained the researcher of all the treatments I am taking or have taken in the past ……. months including any desi (alternative) treatments.
6. I hereby give permission to the investigator to release the information obtained from me as result of participation in this study ethics committee. I understand that they may inspect my original records.
7. My identity will be kept confidential if my data is published or presented.
8. I have had my questions answered to my satisfaction.
9. I have decided to be in the research study.

I am aware, that if I have any questions during this study, I should contact at one of the addresses listed above. By signing this consent form, I attest that the information given in this document is true.

Name and signature / thumb impression of the participant (or legal representative if participant incompetent) :

Name :

Address :

Contact No. :

Date : Sign of the Participant :

रुग्ण संमतीपत्रक

अभ्यासक्रमाचे शीर्षक : 'Evaluation of Bhedana Karma of YG3 in Yakrut Vikar'

रुग्णाचे नाव :

रुग्ण संमतीपत्रक नियम :

मी रुग्ण पत्रकातील सर्व माहिती वाचून किंवा मला वाचून दाखविले आहे.

मी विचारल्या जाणाऱ्या प्रश्नांची उत्तरे देण्यास संमती दर्शवित आहे.

१. मी रुग्ण पत्रकात नमूद केलेली सर्व माहिती वाचली आहे.

२. मला रुग्ण संमती पत्रक समजवून सांगण्यात आले आहे.

३. मला माझे हक्क अभ्यासकाने माहिती करुन दिले आहे.

४. मला अभ्यासाबद्दल माहिती देण्यात आली आहे.

५. मी सध्या घेत असलेल्या सर्व औषधोपचाराची माहिती अभ्यासकाला देत आहे.

६. मी ओळख (Identity) माहिती (Publish) करताना गोपनीय ठेवण्यात यावी.

७. मी माझी उत्तरे समाधानकारकरीत्या देत आहे.

८. मी स्वतः अभ्यासाचा एक भाग होण्यास तयारी दर्शवित आहे.

प्रौढ व्यक्तीसाठी :

रुग्णाचे नाव :

नाव :

सही / अंगठा :

दिनांक :

BHARATIYA SANSKRITI DARSHAN TRUST

Regd. under B.P. Trust Act of 1950 No. 29, Bombay R. No. E 626 (Pune) 27-3-1979

HEAD OFFICE :
1170/31, Revenue Colony,
Opp. Hotel Span Executive,
Shivajinagar, Pune - 411005. MS India.
Website : www.ayurved-for-cancer.org

CENTRE :
'Vishwa Shanti Dham', Keshnand Road,
Wagholi, Tal. Haveli, Pune-412207, MS India.
Ph. : +91-20-67346000 / +91 9545508890
E-mail : ictrcpune@gmail.com

CERTIFICATE OF ANALYSIS

(Finished Product)

Name of Product : Atharva Aloe Plus Tab
Batch no. : 01/13 **Batch Quantity** : 2000 Tabs
In- Date : 01-09-2013 **Best before Dt** : 08-2018
Sampled Qty : 120 Tabs **Sampling date** : 02/09/13
Analysis date : 02/09/13 **Reporting date** : 03/09/13

S.N.	Test	Result	Specification
1.	Description	Light Greenish brown colored, circular, compressed, biconcex, uncoated tablet with characteristic odour.	Light Greenish brown colored, circular, compressed, biconcex, uncoated tablet with characteristic odour.
2.	Average Weight	302mg	285mg – 315mg
3.	Diameter	8 mm	8 – 9 mm
4.	Thickness	4 mm	3-4 mm
5.	Hardness	2 kg/sq.cm	2-4 kg/sq.cm
6.	Disintegration Time	12.30 min	NMT 30 min
7.	Friability	0.49 % w/w	NMT 1 % w/w

Remark: The above sample complies/~~Not complies~~ as per IHS

Analyzed & Approved by:

SRF
Integrated Cancer Treatment
& Research Centre

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Wagholi, Tal. Haveli, Pune-412207, MS India.
Ph. : +91-20-67346000 / +91 9545508890
E-mail : ictrcpune@gmail.com

CERTIFICATE OF ANALYSIS

(Finished Product)

Name of Product	: YPL Kutki Tab		
Batch no.	: 01/13	**Batch Quantity**	: 6089Tabs
Mfg. Date	: 09-05-2013	**Best before Dt**	: 04-2018
Sampled Qty	: 120 Tabs	**Sampling date**	: 09/05/13
Analysis date	: 10/05/13	**Reporting date**	: 11/05/13

S.N.	Test	Result	Specification
1.	Description	Dark brown colored, circular, compressed, biconcex, uncoated tablet with characteristic odour.	Dark brown colored, circular, compressed, biconcex, uncoated tablet with characteristic odour.
2.	Average Weight	233mg	210mg – 240mg
3.	Diameter	8 mm	8 – 9 mm
4.	Thickness	4 mm	3-4 mm
5.	Hardness	2 kg/sq.cm	2-4 kg/sq.cm
6.	Disintegration Time	11.15 min	NMT 30 min
7.	Friability	0.68 % w/w	NMT 1 % w/w

Remark: The above sample complies/~~Not complies~~ as per IHS

Analyzed & Approved by:

Bhavan

SRF
Integrated Cancer Treatment
& Research Centre