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Clinical Study of Aetiology of Cirrhosis of Liver with special reference to Ayurvedic Dietary and Lifestyle factors and Systemic Involvement

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Abstract

Background – Recent observations suggest that the true rate of chronic infection after clinically apparent acute hepatitis B is as low as 1 per cent in normal individuals. Also, only 10 to 15 per cent of alcoholics develop Cirrhosis of the liver, suggesting that other factors might be influencing the impact of the above factors on the liver. In view of the above facts and the obscure concepts of *Prakriti*, *Agni*, *Dincharya*, *Ritucharya*, *Ahar vidhi-visheshyatan*, and healthy lifestyle in modern medicine, it is essential to study cirrhosis of the liver with respect to the factors mentioned above and to examine their role in its development.

Aims and Objectives - The aims and objectives of the present work is to study the aetiopathogenesis of *Kumbhakamala* (Cirrhosis of liver) in terms of Ayurvedic dietary, lifestyle factors and systemic involvement.

Material and Method – 40 Patients having clinical features of cirrhosis of the liver were selected without any prejudice from the O.P.D. & I.P.D. sections of Pakwasa Samanvaya Rugnalaya and All India Ayurveda Research Institute, Hanuman Nagar, Nagpur.

Observations – Proper observations are recorded in tabular format, along with their analyses.

Discussion –*Pittavardhak* dietary and lifestyle regimen in conjunction with faulty dietary habits like *samshan*, *adhyshan*, *vishmashan*, *virruddhashan* is commonly observed in patients of cirrhosis of the liver. Pitta dominance is observed in the genetic body constitution of cirrhotic patients.

Conclusion - After assessing the clinical features, aetiological factors, and pathology explained in both Ayurvedic and modern science, we can fairly correlate Kumbhakamala with cirrhosis of the liver. Ayurvedic pitta-enhancing dietary and lifestyle factors may contribute to the severity of pathology.

Keywords: Cirrhosis of the liver, *Pittvardhak ahar, pitta prakriti*

Introduction :

Ayurveda, the science of life, studies life with respect to health and disease, as well as the various factors that promote and deteriorate health. *Ahara* (diet) and Vihara (lifestyle) are the two most important factors. A healthy diet and lifestyle promote health, and vice versa. Along with *Brahamacharya* (celibacy and good conduct) and *Nidra* (sleep), Ahar forms the three sub-pillars (*Trayopstambha*) of life. In Ayurveda, the unwholesome conjugation of sense organs and objects (*Asatmya Indriyartha Samyog*), purposeful intellectual errors (*Pradnyapradh*) and the consequences of time (*Parinam*) with respect to Ahar and Vihar have been told as the three basic causes responsible for the development of the disease. Ayurveda is of the view that "microbes, no matter how virulent they are, will not create any disease in the body, unless the equilibrium stage of Doshas gets disturbed". This equilibrium is governed by many intrinsic and extrinsic factors, such as *Ahara, Vihara, Agni, and Prakriti*, related to *vyadhikshamatva* (immunity), etc. According to Acharya Vagbhata, all diseases are due to *Mand-Agni* (decreased appetite), which in turn depends on a number of factors, such as *Ahara, Vihara, Prakriti, Vikriti*, Season, Time, etc. This *Mand-Agni* is unable to digest the diet as a result of which the accumulation (*Sanchaya*) of toxic material (*Ama*) takes place, which is the first stage of pathogenesis of any disease. This stage leads to further stages of pathogenesis and causes the disease at the site of affection. There are a number of diseases that are caused by the vitiation of specific doshas (basic humours), which in turn are aggravated by specific dosha-aggravating diet and lifestyle. Kamala is one of them. In Ayurveda, Kamala is defined as a disease characterised by deep yellow discolouration of the eyes, nails, skin, and face, along with fatigue, malaise, indigestion, anorexia, etc.

In modern science, a similar condition is called jaundice, which can be caused by various factors. Hepatitis Viruses like A, B, C, D, E, G and alcoholic hepatotoxicity are the commonest causes responsible for it.[1]

The human knowledge of Kamala is as old as the Vedas. The first reference to Kamala, along with its treatment, is observed in the Atharvaveda, where it is called 'Hariman'. (Hymen 1-22/1, 2 & 4). The period of the Atharvaveda is approximately 2500-3000 BC. For a long time, the disease kamala (jaundice) has been affecting the masses and is one of the major health problems throughout the world, responsible for considerable morbidity and mortality from its acute or chronic sequelae.

According to one study, there are more than 200 million (20 crore) HBsAg carriers in the world. In India, approximately 4.5 crore people are infected with HBV, and 1 crore with HCV, and with a lack of satisfactory treatment and awareness, it remains the 5th single most important factor responsible for mortality in the age group between 14 and 45 years, averaging nearly 2.5 lakh deaths per year. [2]

Kumbhakamala (Cirrhosis of liver) is a chronic stage of the disease Kamala which can be fairly correlated with modern syndrome of cirrhosis of liver in which pathologically there is necrosis of hepatocytes, followed by fibrosis and nodule formation and the liver architecture is diffusely

abnormal interfering with the liver blood flow, producing the features of portal hypertension like hematemesis, malena, encephalopathy, ascites with or without edema feet, etc.[3] which are also seen in *kumbhakamala.*

Recent observations suggest that the true rate of chronic infection after clinically apparent acute hepatitis B is as low as 1 per cent in normal individuals. Also, only 10 to 15 per cent of alcoholics develop Cirrhosis of the liver, suggesting that other factors might be influencing the impact of the above factors on the liver. [4,5] In view of the above facts and the obscure concepts of *Prakriti, Agni, Dincharya, Ritucharya, Ahar vidhi-visheshyatan,* and healthy lifestyle in modern medicine, it is essential to study cirrhosis of the liver with respect to the factors mentioned above and to examine their role in its development. The present literary and clinical study will also help in understanding *Kumbhakamala* (cirrhosis of the liver) and various other types of Kamala in the light of modern science.

Review Of Literature:

Charaka in Chikitsasthan 16/34-37 described the pathogenesis of Koshthashakhashrit Kamala, Kumbhakamala. Haleemaka in 16/132-134 and Shakhashrit Kamala in 16/124-126.[8] According to him, if a patient of Pandu consumes Pitta aggravating factors, then that vitiated Pitta burns (*dagdhwa*) the blood and flesh, resulting in Bahu-Pitta Koshthashakhashrit Kamala, which, after chronicity, leads to Kumbhakamala. *Kaphasamurchhit Vayu,* vitiated due to excess intake of dry, cold, sweet, heavy diet, suppression of natural urges, intense exercise, etc., forces out Pitta from its obstructed site (*Pittasya Patham Kaphen Rudhham*) and throws it into circulation, causing Shakhashrit Kamala. In a patient of Pandu, specific vitiation of Vata & Pitta results in a greenish, yellowish, greenish-blackish complexion; that stage is called Haleemaka.

Vagbhata followed Charaka's view, but he held that Koshthashakhashrit Kamala can occur without the preexistence of anemia in a person of *Pitta Prakriti*. (A.S. Ni. 13/17-21, A.S. Chi. 18/40-43) [10] [11]

Sushruta in Uttaratana 44/11-14 mentioned that if a person, after Pandu roga or any other disease, consumes alcohol, sour/rotten food, or harmful/toxic/antagonistic food in excess, then vitiated Pitta causes Kamala. If this patient of Kamala develops generalised edema and arthralgia, then that stage is called as Kumbhasahv (Kumbhakamala - Dalhan) [8] If this patient of Kumbhasahav develops fever, fatigue, listlessness, giddiness, drowsiness, greenish, greenish-blackish or bluish tint due to specific vitiation of Vata and Pitta, then that stage is called as Haleemaka. After analysing the views of Charaka, Sushruta, Vagbhata, commentators like Chakrapani, Dalhan, the pathogenesis of Koshthashakhashrit Kamala, Shakhashrit Kamala, Kumbhakamala, and Haleemaka can be given as:

In the first type of pathogenesis, the said patient of Pandu is the patient of Pittaj Pandu (Hemolytic anemia) where there is already jaundice is present but to a lesser extent (indicated by the word *'peeta chakshu'*) as we usually notice in patients of hemolytic anemia (low serum bilirubin) But as these patients consumes Pitta aggravating aetiology like alcohol, smoking, hot, chilly, pungent food etc.

Pitta aggravates further, and weak, friable RBCs begin to break down, resulting in a more pronounced jaundice [6, 7], indicated by the word *Haridra* (deep yellow like turmeric), reflected in yellow eyes, yellowish urine, etc. Here, the prime pathology is of *Asruk dagdhwa* (Hemolysis). Prime *strotodushti* is *Atipravritti* (excessive production). This is Koshthashakhashrit Kamala Panduroga-purvika. After cessation of the acute episode, when the chronic sequelae of this pathology produce extensive edema and bony pain, that stage is called Kumbhakamala. And if repeated episodes produce greenish, yellowish or greenish-blackish hue, then that stage is called Haleemaka.[9]

In the second type of pathogenesis, if a patient with any Pitta-dominated disease or a person of Pitta constitution consumes Pitta-aggravating factors like alcohol, excess sexual intercourse, hot, chilly, pungent diet, etc., then there occurs intense vitiation of Pitta resulting in acute hepatic necrosis. Here, the primary pathology is of the *Mansa dagdhwa* type (Hepatolysis), as there is an *ashrayashrayi* relationship between the liver and Pitta. The term *'Mansa dagdhwa'* here refers to hepatic necrosis (hepatolysis). The predominant *strotodushti* here is of *Atipravritti* and sang type. (Excessive production and defective excretion) This type of hepatocellular jaundice in Ayurveda is called Koshthashakhashrit Kamala Pandurogavina.[12] If this condition is left untreated, then it becomes chronic, and although there is no apparent jaundice, the internal pathology involving the liver continues, leading to hepatic fibrosis, indicated by the word *'Kharibhuta'*, giving rise to a condition called Kumbhakamala. The meaning of *Kharibhuta* given by Chakrapani is hardness (*kathortamup-gata*) and intense dryness (*Ati rukshitaha*). In Ayurveda, there are three types of *paka* described viz. *mridu, madhyam* and *khara*. Of these, *mridu paka* is one which formed by mild heating, *madhyam* by moderate heating and *khara* by excessive heating. The relevance of *paka* theory here is very important because the term used in the pathology of K.S.Kamala is dagdhwa (meaning 'to Heat/burn'). Notably, one of Pitta's functions is heating.From the above, one can understand the nature of the pathology. The only difference between ancient and modern science is the way it is described and the terminology used. They have used the terminology prevailing at the time, or sometimes examples, to describe various aspects. As of today, we know that liver fibrosis occurs in cirrhosis, which in Ayurveda is known as Kumbha-kamala. Extensive edema (*Mahan Shofa*), including ascites at this stage, suggests hypoalbuminaemia reflecting severe liver pathology. Hematemesis (*Sraktachhardi*), Malena (*Krishna Shakrita), and encephalopathy indicate* portal hypertension. In this condition, an additional superinfection or acute hepatic necrosis may produce features such as low-grade fever, anorexia, malaise, and icterus. Or long-standing jaundice (on oxidation of bilirubin to the greenish-yellowish or greenish-blackish biliverdin) produces a greenish-yellowish or greenish-blackish hue; that stage is called Haleemaka.[13] Whenever in patients of Kamala, Kumbhakamala or Haleemaka, intense vitiation of *Apan Vayu* results in diarrhoea and other G.I. disturbances, then that stage is called Apanaki.

Material And Methods:

Place of the study

Patients having clinical features of cirrhosis of the liver were selected without any prejudice from O.P.D. & I.P.D. sections of Pakwasa Samanvaya Rugnalaya and All India Ayurveda Research Institute, Hanuman Nagar, Nagpur.

Sample Size

40 diagnosed patients of cirrhosis of the liver is taken for the present study

Selection Of the Patient:

A detailed case history and clinical-pathological findings were recorded on the clinical proforma specifically designed for the present research study.

Diagnostic criteria

Diagnostic criteria were established to cover both modern and Ayurvedic aspects of the disease. The patients having a history of prolonged or excessive alcohol intake or patients chronically infected with the hepatitis B or C virus, having the following clinical features and supported by radiological findings as well as deranged liver function tests, were termed patients of cirrhosis of the liver.

Clinical Features Of Cirrhosis of the Liver:

Symptoms:

Specific

- Abdominal distention due to ascites
- Ankle swelling due to fluid retention
- Haematemesis and melena due to gastrointestinal haemorrhage.
- Pruritus
- Gynecomastia in males
- Loss of libido in males
- Amenorrhoea in females
- Confusion and drowsiness

Non-Specific

- Anorexia
- Fatigue
- Emaciation/weight loss
- Weakness
- Listlessness
- Indigestion
- Constipation
- Malaise

Signs :

General	compensated	Decompen-sated
Fever	Hepatomegaly (initially)	Disorientation
Jaundice	Spleenomegaly	Drowsiness
Loss of hairs	Gynaecomastia	Coma
	Scratch marks over	Flapping tremors
	Spider naevi	Fetor hepaticus
	Perpura	Ascites
	Testicular atrophy	Dilated veins over the abdomen
	Palmer erythema	Edema feet
	Dupuytren's contractures	

History And Clinical Examination:

After confirming the diagnosis, the detailed history with respect to age, sex, habitat, socio-economic status, mode of onset of the disease, family history, drug history, previous or associated disease history with duration, dietary habits, addiction with duration, and state of mind were recorded on the specially prepared clinical proforma.

Examination Of Patients By Ayurvedic Method:

All patients were thoroughly examined for vitiated *Doshas, Dooshyas, Strotas, and Nidanpanchak. Dash-Vidh and Ashta-Vidh pariksha* were also

conducted as described in ancient Ayurvedic classics.

Study Of Nidan:

Each case was carefully interviewed to assess the presence of various risk factors in patients, as explained by modern as well as Ayurvedic science. *Sannikrishta Nidan* (acute) is a recent provoking factor leading to the acute onset of the disease, while *Viprakrishta Nidan* is an old (chronic) risk factor.

General Examinations:

General clinical examinations were done for height, weight, blood pressure, pulse rate, temperature, pallor etc.

Systemic Examinations:

Under it following systems were examined and special stress was given on gastrointestinal and nervous system.

Respiratory system (RS):- It was examined for breathing pattern, air entry, presence of any other abnormality.

Cardiovascular system (CVS) :- Under it the heart was examined for rate, rhythm, heart sounds and any other abnormality.

Gastro-Intestinal system (GIS) :- It was examined for the presence of hepatomegaly, spleenomegaly, and ascites. The abdominal girth on, above and below the umbilicus was noted in patients with ascites.

Central Nervous System (CNS):- It was examined for the presence of features of encephalopathy, such as disorientation, confusion, dysgraphia, drowsiness, coma, etc.

Laboratory Investigations

Hematological: - The routine hematological examinations like Haemoglobin percentage (Hb%), Total Leukocyte Count (TLC), Differential Leukocyte Count (DLC), Erythrocyte Sedimentation Rate (ESR), Peripheral Smear (PS) to see the degree of anemia, its type and also for evidence of infective pathology.

Biochemical:

Liver Function Tests:

1. Serum Bilirubin (Conjugated & Unconjugated): To assess the severity of jaundice and to know its type.

2 Serum AST & ALT: To assess the severity of hepatocellular damage.

3. Serum Alkaline Phosphatase: To assess the severity of cholestasis wherever thought necessary.

4. Serum Proteins (Albumin & Globulin): To assess the severity of functional pathology of the liver.

5 Serum Prothrombin Time: To assess the severity of dysfunction of the liver and the coagulopathy.

6. Serum Viral Marker: - To know the type of hepatitis virus.

Serum Electrolytes:

1. Serum Sodium: To assess the severity of hyponatremia.

2. Serum Potassium: To assess hypo or hyperkalemia.

Kidney Function Tests:

To assess the presence of hepato-renal failure and the severity of functional pathology of the kidneys.

1. Serum Creatinine

2. Blood urea

Peritoneal Fluid Examination:

To assess the fluid for transudation or exudation, and also for evidence of bacterial peritonitis and other pathologies.

Miscellaneous:

Blood sugar and other appropriate investigations as required and based on the presence of associated disease.

Radiological:

Ultrasound: To assess the type of liver pathology, as well as for evidence of portal hypertension and the presence of ascites.

Endoscopy:

For evidence of varices.

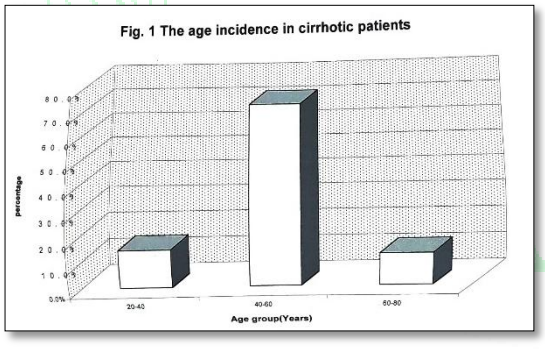
Result & Observations:

The results and observations of the present study are given as follows.

1. Age incidence in cirrhotic patients:

Table No. 1 (n = 40)

Sr	Age group (years)	No. of patients	%
1	20-40	6	15%
2	40-60	29	72.5%
3	60-80	5	12.5%
	Total	40	100%

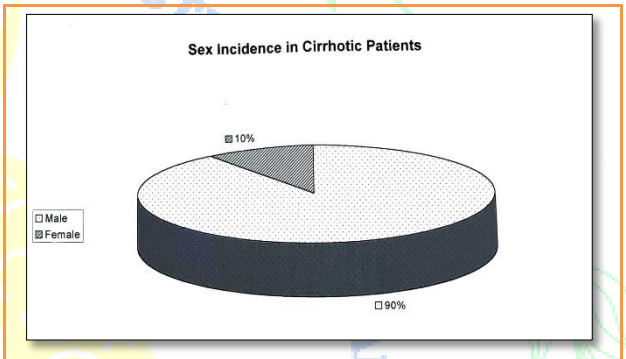


Age incidence in cirrhotic patients

2. Sex incidence in cirrhotic patients:

Table No. 2 (n=40)

Sr	Sex	No. of patients	Percentage
1	Male	36	90 %
2	Female	4	10 %
	Total	40	100%



Sex incidence in cirrhotic patients

Habitat (*Desha*) in cirrhotic patients:

Table No. 3 (n = 40)

Sr	Area	No. of patients	%
1	Rural	13	32.5 %
2	Urban	27	67.5 %
	Total	40	100%

Out of 40 patients registered in the present study, the majority of patients (72.5%) were in the age range of 40 to 60 years, 90% of the patients were male, and the majority of them (67.5%) were from an urban area.

Incidence Of Socio-Economic Status:

The patients with an annual income of less than Rs. 35,000/- were placed in the lower-income group. The annual Income between Rs. 35,000 and 65,000/- were placed in the middle-class income group. And patients having an annual income of

more than Rs. 65,000/- were categorised under the higher-class income group.

4. Table No. 4 (n=40)

Socio-Economic Status

Sr	Sex	No. of patients	%
1	Lower	15	37.5%
2	Middle	12	30 %
3	Higher	13	32.5%
	Total	40	100%

Although the percentage of cirrhosis in the lower income group is slightly on the higher side, no significant variation is seen in the occurrence of cirrhosis as far as socio-economic status is concerned.

5. Incidence Of Dietary Habits (*Ahar*)

Table No. 5 (n = 40)

Sr	Dietary habit	No. of patients	%
1	Vegetarian	5	12.5%
2	Non-Vegetarian	0	0 %
3	Mixed	35	87.5%
	Total	40	100%

In the present study, out of 40 patients suffering from cirrhosis, 35 (87.5%) were having a mixed diet

6. Incidence Of Addiction:

Table No. 6 (n=40)

Sr	Addiction	No. of patients	%
1	Only alcohol	6	15%
2	Alcohol with smoking	21	52.5%
3	Alcohol with tobacco	4	10%
4	Only smoking	1	2.5%
5	No addiction	7	17.5%
6	Other	1	2.5%
	Total	40	100%

Alcohol addiction is found in 77.5% patients. Out of which 52.5% were also addicted to smoking and 10% with tobacco chewing. No addiction was found in 17.5% patients. The other addiction 2.5% found was of Fortwin. The duration of alcoholism was in the range of 10 to 25 years. The mean duration of alcoholism was 17.5 years.

7. Incidence of State of Mind (*Mansic hetu*)

Table No. 7 (n = 40)

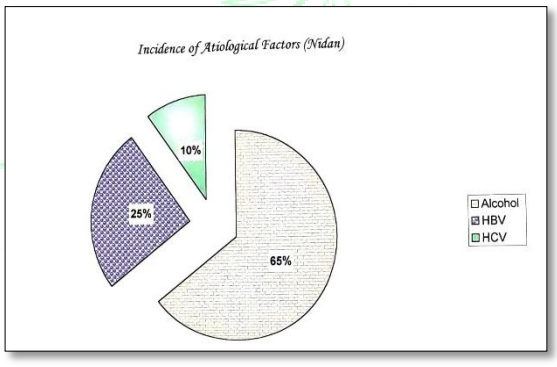
Sr	Mental status	No. of patients	%
1	Anxious	8	20 %
2	Tense	11	27.5%
3	Irritable	9	22.5%
4	Relaxed	12	30%
	Total	40	100%

A stressful state of mind was observed in 70% patients with cirrhosis of the liver.

Incidence Of Aetiological Factors (*Nidan*):

Table No. 8 (n = 40)

Sr	Aetiological factors	No. of patients	%
1	**Alcohol**	26	65%
2	**HBV**	10	25%
3	**HCV**	4	10%
	Total	40	100%



Incidence Of Aetiological Factors (*Nidan*):

In 65% of the patients, alcohol was directly responsible for the development of cirrhosis, while in 25% patient main aetiological factor was hepatitis B virus, and in the remaining 10%, it was hepatitis C virus.

9. *Ahar Hetu* (Dietary factors):

The criteria for specific dosha aggravation were as follows:

Vatvardhak ahara :

Katu, Tikta, Kashay rasatmak and Laghu, Ruksha, Vishtambhi gunatmaka etc.

Pittavardhak ahara:

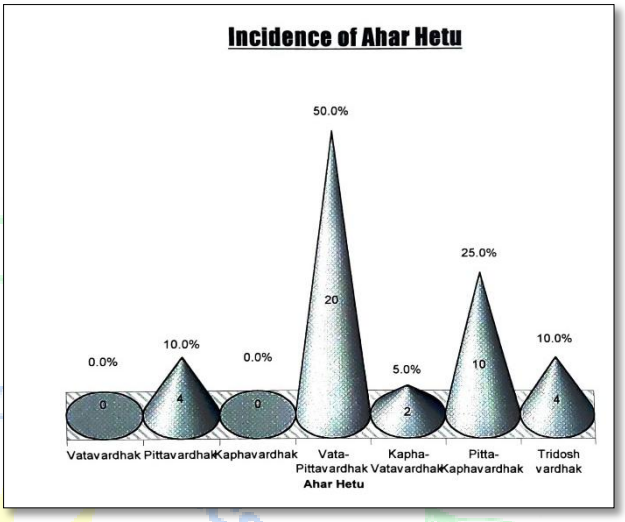
Katu, Lavan rasatmak and Ushna, Tikshna, Vdahi, gunatmak etc.

Kaphavardhak ahara:

Madhur, Amla rasatmak and Shit, Guru, Vishyandi gunatmaka etc.

Table No. 9 showing *Ahar hetu* (Dietary factors) in cirrhotic patients. (n = 40)

Sr	*Ahara*	No. of patients	%
1	*Vatavardhak*	0	0%
2	*Pittavardhak*	4	10%
3	*Kaphavardhak*		
4	*Vata-Pittavardhak*	20	50%
5	*Kapha-Vatavardhak*	2	5%
6	*Pitta-Kaphavardhak*	10	25%
7	*Tridosh Prakopak*	4	10%
	Total	40	100%



Incidence Of Ahar Hetu

The diet of 50% patients with cirrhosis of the liver was *Vat-Pittavardhak*. In 25% of patients, it was *Pitta-Kaphavardhak*, and only 10% followed a plain *Pittavardhak* diet.

10. *Ahar Paddhati* (Dietary Habit) :

The dietary habit was divided into four parts as follows:

1. ***Samshan***:- Eating a healthy and an unhealthy diet together.

2 ***Vishmashan*** - Irregularity of diet with respect to time and quantity.

3. ***Adhyshan***:- Taking a diet before the digestion of the earlier one.

4. ***Viruddhashan*** :- Consuming an antagonistic diet.

Table. 10 (n=40) Showing *Ahar paddati* of the patients

Sr	*Ahar Paddhati*	No. of patients	%
1	*Samshan*	0	0%
2	*Vishmashan*	0	0%
3	*Adhyashan*	0	0%
4	*Viruddhashan*	4	10%
5	Combination of 1 & 2	5	20%
6	Combination of 1 & 3	6	15%
7	Combination of 1, 2 & 3	14	35%
8	All The Above	8	20%
9	None of The Above	4	10%
	Total	**40**	**100%**

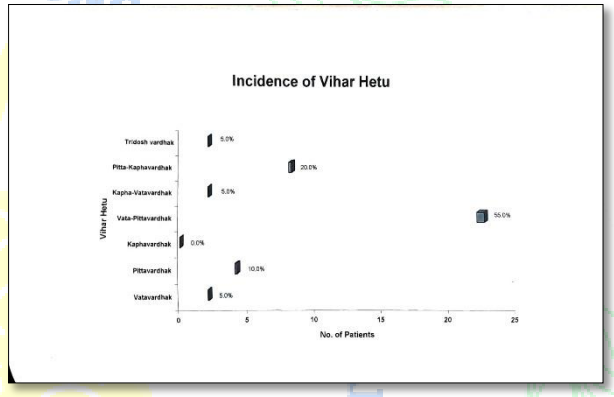
The combination of *samshan, vishmashan and adhyshan* was observed in 35% patients of cirrhosis of the liver. The combined *Ahar Paddhati of Samshan and Vishamshan* was observed in 20% patients. While in 20% of patients, it was a combination of *samshan, vishmashan, adhyashan,* and viruddhashan.

11. *Vihar Hetu* (Lifestyle factors):

Lifestyle factors like *Vegvidharan* (forceful suppression of natural urges), *Ativyayam* (intense exercise), and *Marutsevan* (e.g., excessive driving) were the *Vatavardhak vihar hetu. Ativyavay, Atapsevan* (e.g. working in hot conditions), etc., were the *Pittavardhak hetu and Diwaswap, Avyayam,* etc., were the observed *Kaphaprakopak vihar hetu.*

Table No. 11 (n = 40) *Vihar Hetu*

Sr	*Vihar*	No. of patients	%
1	*Vatavardhak*	2	5 %
2	*Pittavardhak*	4	10 %
3	*Kaphavardhak*	0	0%
4	*Vata-Pittavardhak*	22	55%
5	*Kapha-Vatavardhak*	5	20%
6	*Pitta-Kaphavardhak*	--	---
7	*Tridoshaj*	14	35%
	Total	**40**	**100%**



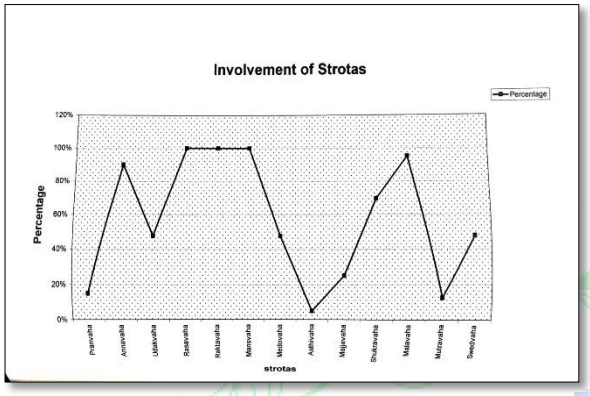
Incidence of Vihar Hetu

The lifestyle of 55% patients of cirrhosis was *Vata-Pittavardhak*. In 20% patients, it was observed as *Pitta-Kaphavardhak*. In 10% it was *Pittavardhak*. The rest of the lifestyle accounted for only 5% each.

12. Incidence Of *Strotas* Involved:

Table No. 12 (n = 40)

Sr	*Strotas*	No. of patients	%
1	*Pranwaha*	6	15%
2	*Annawaha*	36	90%
3	*Udakwaha*	19	47.5%
4	*Rasavaha*	40	100%
5	*Raktawaha*	40	100%
6	*Manswaha*	40	100%
7	*Medowaha*	19	47.5%
8	*Asthivaha*	2	5%
9	*Majjavaha*	10	25%
10	*Shukravaha*	25	69.4%
11	*Malavaha*	38	95%
12	*Mutravaha*	5	12.5%
13	*Swedvaha*	19	47.5%
	Total	**40**	**100%**



Incidence Of *Strotas* Involved

The *Rasavaha*, *Raktavaha*, and *Mansvaha strotas* were involved in all patients with cirrhosis of the liver. *Malavaha* in 95% of patients, *Annavaha* in 90% of patients, and the *Swedvaha* and *Medovaha strotas* were involved in 47.5% of patients. While *Majjavaha* was involved in 25% of patients, *Mutravaha* in 12.5%, and *Pranvaha strotas* in 15%. The *Shukravaha strotas* were involved in 69.4% of the 36 male patients.

13. *Prakriti Parikshan* (constitution):

The criteria of specific Prakriti were based on the features of a particular Prakriti observed in the patient. The separate chart showing features of different *Prakrities* is given in the 'appendix'

Table No. 13 (n = 40)

Sr	*Prakriti*	No. of patients	%
1	*Vataj*	1	2.5%
2	*Pittaj*	2	5%
3	*Kaphaj*	--	--
4	*Vata-Pittaj*	11	27.5%
5	*Kapha-Vataj*	14	35%
6	*Pitta-Kaphaj*	12	30%
7	*Tridoshaj*	--	---
	Total	40	100%

Pitta-Kaphaj Prakriti was observed in 35% patients. *Pitta-Vataj* was observed in 27.5% of patients, and *Kapha-Vataj* in 30%. Single *Doshaj Prakriti* was observed in only three patients. Of which, 5% were *Pittaj Prakriti* and only 2.5% were *Vataj Prakriti*.

14. Associated Complications

Table No. 14 (n = 40)

Complications	No. of patients	%
Encephalopathy	10	25%
Bacterial peritonitis	5	12.5%
Renal Failure	3	7.5%
Pleural Effusion	4	10%
Hematemesis	11	27.5%

Encephalopathy was observed in 25% of patients, Bacterial peritonitis in 12.5%, Hematemesis in 27.5%, and renal failure in 7.5%. Pleural effusion in 10% cases.

15. Incidence Of Associated Disease:

Table No. 15 (n = 40)

Disease	No. of patients	%
Encephalopathy	2	5%
Diabetes	4	10%
Myxoedema	1	2.5%
Malaria	3	7.5%
Active Hepatitis	6	15%

Diabetes was associated with 10% cases, Pulmonary Tuberculosis was associated with 5% cases, Myxoedema and Thyrotoxicosis with 2.5% cases. Active hepatitis was seen in 15% cases. Falciparum Malaria was present in 7.5% cases.

16. General Clinical Findings

Table No. 16 [n=40]

weight of the patients (Kg):

Weight	No. of patients	%
35-55	20	50%
55-75	17	42.5%
>75	3	7.5%
Total	40	100%

The weight of 50% patients with cirrhosis of the liver was less than 50Kg. The mean weight of 42.5% of patients was 60.3 kg. The mean weight of 92.5% patients was less than 55 kg, indicating weight loss in patients with cirrhosis of the liver.

Table No. 17 [n = 40]

pulse rate of the patients:

Pulse	No. of patients	%
60-80	16	40%
80-100	15	37.5%
>100	9	22.5%
Total	40	1005

The mean pulse rate was 76/min in 40% of patients, 86/min in 37.5%, and 100/min or more in only 22.5%.

Table No. 18 [n=40]

temperatures of the patients (°F)

Temperature (°F)	No. of patients	%
99-100	11	27.5%
100-102	3	7.5%
>102	2	5%
Total	40	100%

In all 40% patients of cirrhosis of the liver were febrile, but usually there is a low-grade temperature, as evident from the above table

Table No. 19(a) [n = 40]

systolic B.P. of the patients(mmHg)

Systolic B.P. (mmHg)	No. of patients	%
90-110	11	27.5%
110-130	21	52.5%
>130	8	20%
Total	40	100%

The systolic blood pressure of 52.5% patients was in the range between 110-130 mmHg and the blood pressure of 27.5% patients was in the range between 90-110 mmHg.

Pathological Investigations:

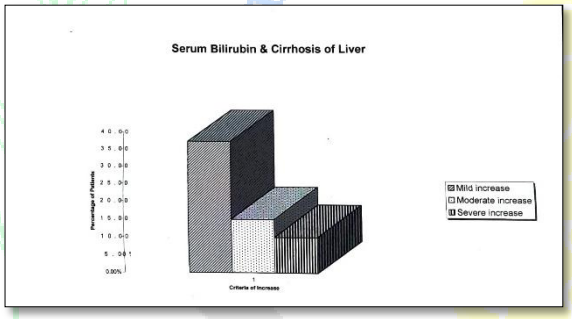
20. Serum Bilirubin and Cirrhosis:

Table No. 20 (n = 40)

Total Bilirubin (mg/dl)	No. of patients	%	Mean Bilirubin
Mild (1.5-5)	15	37.5%	2.6
Moderate (5-10)	6	15%	6.2
Severe (>10)	4	10%	24.3

Conjugated Bilirubin (mg/dl)	No. of patients	%	Mean Bilirubin
Mild (0.5-2.5)	26	65%	1.2
Moderate (2.5-5)	6	15%	3.6
Severe (>5)	4	10%	17.4

The mild increase in total serum bilirubin was observed in 37.5% patients, moderate increase in 15% and severe increase in 10% cases, and their mean value is shown in the table. The rise in total serum bilirubin may not be obvious all the time, as the sum of conjugated and unconjugated bilirubin may reach the normal level, but the above table shows the relative increase of conjugated bilirubin, though total serum bilirubin may remain normal. The above findings also indicate a relative rise in conjugated bilirubin over unconjugated bilirubin in cirrhosis of the liver.



Serum Bilirubin & Cirrhosis of Liver

21. Serum AST & ALT and Cirrhosis of the Liver:

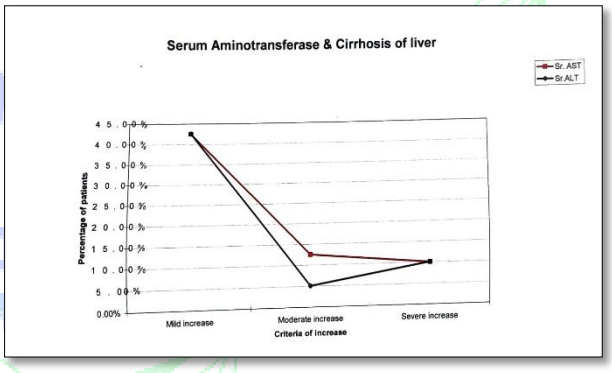
Table No. 21 (n=40)

Serum Aminotransferase IU/L

AST (IU/L)	No. of patients	%	Mean AST
Mild (40-80)	17	42.5%	61.7
Moderate (80-120)	5	12.5%	113.2
Severe (>120)	4	10%	130.2

ALT (IU/L)	No. of patients	%	Mean ALT
Mild (40-80)	17	42.5%	54.8
Moderate (80-120)	2	5%	92.5
Severe (>120)	4	10%	174.3

The mild increase in serum AST and ALT was observed in 42.5% patients. A moderate increase in serum AST was observed in 12.5% patients. Serum ALT was moderately elevated in 5% of patients. A severe increase in serum AST and ALT was observed in 10% patients. The mean increase in each category is shown in the table. In contrast to hepatitis, where a higher increase (> 2 to 300 IU/L) in serum aminotransferase levels occurs, only a mid to moderate (< 200 IU/L) increase in aminotransferase levels occurs in cirrhosis of the liver.



Serum Aminotransferase & Cirrhosis of Liver

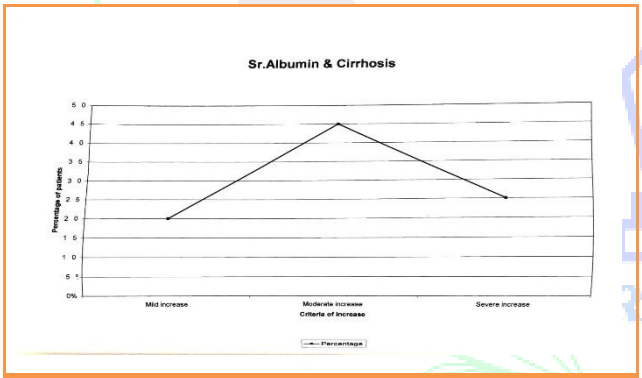
22. Serum Proteins And Cirrhosis Of Liver:

Table No. 22(a) (n = 40]

Serum Proteins (total)

Total Proteins (g/dl)	No. of Patients	%	Mean Proteins
Mild (6-5.5)	12	30%	5.8
Moderate (5.5-4.5)	14	35%	5.2
Severe (<4.5)	2	5%	3.0

Albumin (g/dl)	No. of Patients	%	Mean ALB
Mild (3.5-3)	8	20%	3.2
Moderate (3-2.5)	18	45%	2.7
Severe (<2.5)	10	25%	1.9



Serum Albumin & Cirrhosis of Liver

A mild decrease in total serum proteins was observed in 30% of patients, and a moderate decrease in 35%. A severe decrease was noted in 5% of patients. A mild decrease in serum albumin was observed in 20% of patients, and a moderate decrease in 45%. A severe decrease was noted in 25% of patients. The mean decrease is shown in the table. As many as 90% patients with cirrhosis of the liver show a decrease in serum albumin level, indicating a relative decrease in hepatic function.

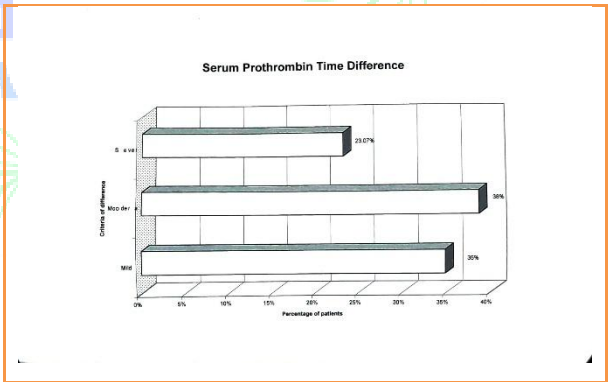
23. Serum Prothrombin Time And Cirrhosis Of Liver:

The serum prothrombin time was measured in 26 patients with liver cirrhosis. Findings are summarised in the following tables:

Table No. 23(a) (n = 26)

Pro-thrombin (S)

PT (S)	No. of Patients	%	Mean PT
Mild (75-95)	8	30.7%	84.7
Moderate (55-75)	14	53.8%	65.3
Severe (< 55)	4	15.3%	51.6



Serum Prothrombin Time Difference

A mild increase in the serum prothrombin time was observed in 34.61% of patients. Moderate increase in 38.46% patients. A severe increase was observed in 23.07% of patients. The mean prothrombin time difference for each category is shown in the table. The overall increase in serum prothrombin time was observed in 25 of 26 patients, reflecting hepatic dysfunction in cirrhosis.

Discussion:

The majority of patients (72.5%) were aged 40 to 60 years. The mean duration of alcoholism in patients of alcoholic cirrhosis was observed as 17.5 years. This long period required to develop cirrhosis of the liver after alcoholism explains the chronic nature of the disease and the reason behind the majority of patients falling in the above age group.

The high prevalence of cirrhosis in males compared to females (10%) was observed, which can be understood (90%) by the fact that the habit of alcoholism is much more common in males than in females in Indian society.

Our study shows that the urban population was more affected (67.5%) than the rural population (32.5%). No particular reason was obvious for this variation. However, several factors, such as the relatively high risk of HBV or HCV infection in the urban population, and Faulty dietary habits and lifestyle, may be responsible for the high urban susceptibility.

The socio-economic status does not seem to influence the occurrence of cirrhosis as it was almost equally present in different socio-economic groups

The habit of a mixed diet was seen in 87.5% patients of patients with cirrhosis of the liver. Although at present no particular line of demarcation could be sketched to show why cirrhosis is common in people having mixed dietary habits, there may be some influencing idiopathic dietary factors that need to be explored through extensive research in this regard

A stressful state of mind was observed in 70% of patients, suggesting that it may also contribute to disease susceptibility through mechanisms such as decreased immune response and altered physiology. Alcohol addiction was seen in 77.5% patients of patients with cirrhosis of the liver. Out of this alcohol was directly responsible in 65% cases for developing cirrhosis. This definite contribution of alcohol in the development of cirrhosis of the liver is well documented by many other researchers.

Amongst aetiological factors, alcohol was directly responsible for the development of cirrhosis in 65% cases. While HBV was responsible in 25% cases and HCV in 10% cases.

Our study shows that female patients are more likely to develop cirrhosis from chronic HCV infection, as all the 10% cases of HCV-induced cirrhosis were females. The association of alcohol and HCV has been suggested by many researchers, but in our study, we did not notice such an association. However, the association of alcohol was found in 40% cases of HBV-induced cirrhosis of the liver. The interactions between alcohol and hepatotrophic viruses may cause a greater extent of hepatic injury, and it may also affect the course of the disease.

The Vata-Pittavardhak diet was found in 50% of patients. While *Pitta-Kaphavardhaka* was observed

in 25% of patients, and plain *Pittavardhak* in 10% of patients. Although mixed *doshvardhak* dietary training was observed, the relative preponderance of *Pittavardhak* diet in patients of cirrhosis of the liver could well be assessed

The more or less combination of *Samshan, Vishmashan, Adhyashan, Viruddhashan, aharpaddhati was observed in as many* as 90% patients. The high prevalence of these faulty dietary habits among cirrhotic patients should prompt researchers to analyse their role in detail.

The *Vat-Pittavardhak* lifestyle was observed in 55% patients. While *Pitta-kaphavardhak* was observed in 20% of patients, plain *Pittavardhak was observed* in only 10%. Although the adoption of a mixed lifestyle was observed, the Pitta aggravating lifestyle was predominantly seen in cirrhotic patients.

The involvement of *the Rasavaha, Raktavaha, and Mansvaha strotas was the most consistent finding regarding strotasic involvement*. The other significantly involved strotas were Annavaha (90%), *Malavaha (95%), Shukravaha (69.4%), Swedvaha (47.5%), Udakvaha (47.5%), Majjavaha (25%), Mutravaha (12.5%).* Our study shows that *the Rasavaha, Raktavaha, and Mansvaha strotas are essentially involved in liver cirrhosis*. The involvement of other strotas indicates the extent and severity of the disease.

The *Pitta-Vatik* prakriti was seen in 27.5% patients. While. Pitta-*Shleshmik prakriti* was observed in 35% of patients, and plain *Paitik prakriti* in 5%. It shows that Pitta predominates in the genetically determined body constitution of the majority of patients with cirrhosis of the liver. Hence, persons having Pitta dominant prakriti should exercise caution while consuming alcohol and other Pitta-aggravating diet and lifestyle.

Our study recorded the presence of the following clinical features with their frequency in patients with cirrhosis of the liver:

Samhanan, Praman, Aharshakti and Vyayamshakti in patients with cirrhosis of the liver. The *Pitta-Vatik prakriti* was seen in 27.5% patients. While *Pitta-Shleshmik prakriti* was observed in 35% of patients, plain *Paitik prakriti* was observed in 5%. It shows that Pitta predominates in the genetically determined body constitution of the majority of patients with cirrhosis of the liver. Hence, persons having Pitta dominant *prakriti* should exercise caution while consuming alcohol and other *Pitta-aggravating* diet and lifestyle. Our study recorded the presence of the following clinical features with their frequency in patients with cirrhosis of the liver:

From the above findings, it can be said that patients with established features of portal hypertension and a history of chronic alcohol consumption or chronic infection with HBV or HCV can be labelled as patients with cirrhosis of the liver. Amongst associated complications (*Upadrava*) of cirrhosis, encephalopathy was observed in 25% patients, hematemesis in 27.5% patients, bacterial peritonitis in 12.5% patients, pleural effusion in 10% patients and renal failure in 10% patients

The association of cirrhosis with other diseases (*Vyadhisankar*) was also observed in our study, including pulmonary tuberculosis in 5% of patients, diabetes in 10% of patients, myxoedema and thyrotoxicosis in 2.5% of cases each, and

Falciparum malaria in 7.5% of patients. These *vyadhisankara* affect the prognosis of the disease. From a pathological investigation point of view, hyperbilirubinemia was observed in as many as 62.5% patients. Of these, 60% of patients were in the mild range. Conjugated bilirubin was predominantly increased. Hence, it can be said that in cirrhosis, a usually mild increase in serum bilirubin occurs, resulting in conjugated hyperbilirubinemia.

Conclusions:

- *Pittavardhak* dietary and lifestyle regimen in conjunction with faulty dietary habits like *samshan, adhyshan, vishmashan, virruddhashan* is commonly observed in patients of cirrhosis of the liver.
- The dominance of Pitta is observed in the genetic body constitution of the cirrhotic patients.
- Cirrhosis of the liver is common in males as compared to females.
- Alcoholic cirrhosis is more prevalent than postviral cirrhosis.
- Females are more prone to developing postviral cirrhosis.
- The pathological findings observed in the present study support the established view.

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