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Ayurveda Management of Ascites secondary to End Stage Alcoholic Fatty Liver Disease : A Single Case Study

Darshana Vyas [1*], Rachana Gandhi [2] Raj Jha[3]

[1] Associate Professor and HoD, [2] Lecturer, Kayachikitsa Department, [3] Intern Scholar, Government Ayurved Mahavidyalaya, Panigate, Vadodara, Gujarat, India

ABSTRACT:

Alcohol is considered one of the commonest causes of cirrhosis of the liver in men, especially in countries like India. *Jalodara* (ascites) is the pathologic accumulation of fluid within the peritoneal cavity. It is the most common complication of cirrhosis and occurs in about 50% of patient with decompensated cirrhosis in 10 years. The development of ascites denotes the transition from compensated to decompensated cirrhosis. Mortality increases from complications such as spontaneous bacterial peritonitis and hepatorenal syndrome. Mortality ranges from 15% in a year to 44% in 5 years. It can be also called *Shotha* (anasarca). Here Ayurveda plays a revolutionary role in decreasing symptoms and reducing the chances of recurrent ascites. It involves *Nidana Parivarjana* (stopping the causative factors), dietary changes (primarily on Milk diet) and medicines. A 39 years old male comes to our OPD with complaints of severe abdominal distension, pedal edema, anorexia, polyarthralgia and scrotal swelling since 6 months. He was admitted and put on only a Milk diet with *Shothahara Chikitsa* along with *Nitya Virechana* and *Asthapana Basti* for 45 days. Every day during hospitalization patient's weight, abdominal girth, input-output along with other vitals such as blood pressure, pulse rate and respiratory rate was monitored closely. After 1 week of medications, the patient's condition improved. All parameters including abdominal girth, body weight, pedal edema, and liver enzymes were reduced satisfactorily. This provides evidence for effectiveness of Ayurveda in management of ascites in end stage liver disease.

KEYWORD: *Asthapana Basti, Cirrhosis, Jalodara, Nitya Virechana, Shotha.*

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***Corresponding Author:**
Vd. Darshana Vyas
Associate Professor & HoD,
Kayachikitsa Department, Government Ayurved Mahavidyalaya, Panigate, Vadodara, Gujarat, India
Email: drdhvyas@gmail.com

INTRODUCTION:

Ascites is the commonest manifestation of end stage liver disease. It is characterized by increased abdominal girth, everted umbilicus, visible spider veins. Increased abdominal girth is due to accumulation of fluid (>25ml)

in peritoneal cavity. The most common etiology of cirrhosis in adults in India is alcohol (43.2%), followed by NAFLD/cryptogenic (14.4%), HBV (11.5%) and HCV (6.2%). Mortality increases from complications such as spontaneous bacterial peritonitis and hepatorenal syndrome. Mortality ranges from 15% in a year to 44% in 5 years. [1]The prevalence of etiological causes of liver disease was similar in the different zones of the country. The proportion of alcohol-related cirrhosis has increased significantly over the last three decades, while the proportion of viral-related cirrhosis has shown a declining trend.[2] According to *Ayurveda*, it falls under the category of *"Udararoga*". Main causative Factor is *"Manda Agni*". It leads to improper formation of *Rasa dhatu*, here it is neither utilized by body or not discarded by body so it accumulates in the peritoneal cavity i.e *Udara*. Hence it is named as *"Jalodara*".[3] In this entity there is vitiation of *Tridosha*. Vitiated *Vata* and *Kapha* leads to the formation of fluid and its accumulation in the abdomen simultaneously it will cause dryness of skin and lips. Vitiated *Pitta* is responsible for anorexia and icterus.

Here Ayurvedic Management includes [4] : *Nidana Parivarjan* (avoiding causative factors), *Nitya Virechana* (regular purgation), A*gni Deepana* (Appetizing agents) reducing the edema and ascites with *Shothahara Dravyas* , *Pathya Aaahar-Vihar* (particularly on only milk Diet). This line of treatment yields best results in ascites and reduces its recurrence by correcting hepatic pathology. Conventional line of treatment includes diuretic, oral amino acids, digestives enzymes that provide symptomatic relief to the patient, from here Ayurveda plays its role. Drugs such as *Haritaki, Katuki* reduces vitiated *doshas* through *Adho Marga* that results into *Kostha Shuddhi* and accumulation of ascitic fluid. Other drug such as *Punarnava* act as potent diuretic as well as hepatoprotective agent. Unlike diuretics such as frusemide and spironolactone it doesn't cause any electrolyte derangement.

Predictive models for the prognosis of cirrhosis estimate the ten-year survival in patients with compensated cirrhosis at 47%, but this drops to 16% once a decompensating event occurs. The Child-Turcotte-Pugh (CTP) scoring or classification uses serum albumin, bilirubin, PT, ascites, and hepatic encephalopathy to classify patients with cirrhosis into classes A, B, and C. One- and two-year survival rates for these classes are 100% and 85% (A), 80% and 60% (B), and 45% and 35% (C). The model for end-stage liver disease (MELD) score is another model used to predict the short-term mortality of patients with cirrhosis. It uses serum bilirubin, creatinine, and INR to predict mortality within the next three months. [5]

CASE REPORT:

A 39 yrs old male came with complaints of severe abdominal distension, pedal edema, anorexia, polyarthralgia and scrotal swelling for 4 months.

History of present Illness:

The patient was healthy before 1.5 yrs. He was addicted to alcohol and consumed it on a regular basis i.e 100 ml per day for last 10 yrs. He is a driver by profession and belongs to lower middle class. Initially he develops anorexia before 1 year and jaundice was diagnosed. He took some medications that included PPIs (pantoprazole 40mg), oral amino acids such as ornithine, aspartate and digestive enzymes such as pancreatin from the nearby physician and his symptoms were cured. Afterwards he continued alcohol consumption. Again after 3-4 months he

develops anorexia, post meal vomiting, mild abdominal distension, polyarthralgia. He consulted a nearby physician and get investigation done. It showed liver parenchymal disease with cirrhotic changes. His blood investigations showed elevated liver enzymes, leukocytosis, thrombocytopenia and borderline anemia. Gradually his condition worsens and there was distension of abdomen with gross pedal and scrotal edema. He also had 2 episodes of general clonic tonic seizure in Dec'23. He was admitted and treated for the same. He also undergone paracentesis and 3.5 liters of fluid was aspirated. After not getting satisfactory results, patient comes to *Kayachikitsa* OPD of Government Ayurveda hospital, Vadodara. After consultation he was admitted to *Kayachikitsa* IPD for further treatment and management. Past history shows No history of any Systemic illness**.** No any surgical History**.** No any significant history of same illness. Physical Examination shows Blood pressure-110/70 mm of Hg, Pulse-110/min, Body Temperature-99.0°F, Icterus Present ++, Pedal Edema ++++, Pallour +, Respiratory Rate- 24/min. Systemic Examination (Per abdomen) was performed. In which inspection reveals Grossly distended abdomen, Visible veins, Shiny skin,[6] Palpation reveal Liver and spleen not palpable due to severe ascites, Percussion shows Fluid Thrill and shifting Dullness Present.

Pathya-Apathya

Restricted diet was given to the patient. Whenever patient feels Hunger and thirst milk (cow milk) is provided.[7] Around 1400 to 1800 ml of milk was consumed per day.Milk diet was continued for 3 months of hospitalization and post hospitalisation for 3 months.

Udara Bandhan was Done for straight 14 days. Elastocrepe bandaging was done with mild pressure around the abdomen.[8] Same procedure was done in Lower Limbs for 2 weeks.

1. Grades of ascites

Grade 1	Mild ascites detectable only by ultrasound examination 100 ml and CT.
Grade 2	Moderate ascites manifested by moderate symmetrical distension of the abdomen, detectable with flank bulging and shifting dullness.
Grade 3	Large or gross ascites with marked abdominal distension, directly visible, confirmed with the fluid thrill test.[9]

2. Grades of Pedal edema

Grade +1	up to 2mm of depression, rebounding immediately.
Grade +2	3–4mm of depression, rebounding in 15 seconds or less.
Grade +3	5–6mm of depression, rebounding in 60 seconds. Grade +4: 8mm of depression, rebounding in 2–3 minutes.[10]

3. Grading of anorexia is done on the basis of amount of milk consumed by the patient in 24 hours.
4. Grading of polyarthralgia is based on intensity of pain described by patient.

Table-1: showing list of medicines given during treatment:

Medicine	Dosage	Anupana	Time	Duration
Haritaki churna (Terminalia chebula)	10gms	*Guda(jaggery)*	In morning Empty stomach	45 days
Punarnava Mandur	500mg	Milk	12 hrly after meal*	60days
Aarogyavardhini Vati	500mg	Milk	12 hrly after meal*	30 days
Trikatu Churna	1gms	Milk	12 hrly Before meal*	30 days
Katuki Churna (Picrorhiza kurroa)	3gms	Milk	In morning Empty stomach	30 days
Punarnava Guggulu	500mg	Milk	Thrice a day Before meal	60 days
Punarnava churna(Boerhavia diffusa)+ Kalmegha churna(Andrographis paniculata)+ Guduchi Churna(Tinospora cordifolia)	2 gms each	Milk	12 hrly Before meal*	60 days

*signifies only milk as a diet.

Table-2: Comparisons of Parameters:

Parameters	Before Treatment	After Treatment
USG ABDOMEN	It shows Chronic Liver Parenchymal Disease-Cirrhosis. Moderate to severe ascites.(9th dec 2023)	Mild ascites along with cirrhotic changes.(April 2024)
2D Echocardiography	Tachycardia noted Normal chamber Dimension No RWMA at rest Grade 1 LVDD Normal LV Systolic function, LVEF 60% (11th Dec 2023)	
Haemoglobin	9.5 gm/dl	7.2 gm/dl
Total leukocyte count	4220 per cmm	10000 per cmm
Platelet count	61000 cmm	150000 per cmm
S Bilirubin (total)	17.5 mg/dl	1.9 mg/dl
S. Creatinine	0.6 mg/dl	0.9 mg/dl
SGPT	46	51
SGOT	160	49
HIV	**Negative**	
HbsAg	**Negative**	

Table -3: Trend of abdominal girth during treatment:

Date	Abdominal Girth (in centimeters)		
	Standing	Sitting	Lying
22/02/2024	100.5	102.5	97.5
27/02/2024	97.5	100.5	98
02/03/2024	95.5	98.5	95
07/03/2024	97	99	98
12/03/2024	92	99	92
18/03/2024	88	95	89
23/03/2024	86.5	91.5	86
28/03/2024	84	90	83.5
02/04/2024	83	85.5	81
08/04/2024	83	87	82

Table-4: Grading of symptoms along with the resolution after treatment:

Date	Abdominal distension	Pedal Oedema	Anorexia	Polyarthralgia
22/02/24	+++	++++	+++	++++
27/02/24	+++	+++	++	++
03/03/24	+++	+++	++	+
10/03/24	+++	++	+	+
20/03/24	++	++	-	-
30/03/24	++	++	-	-
08/04/24	++	+	-	-

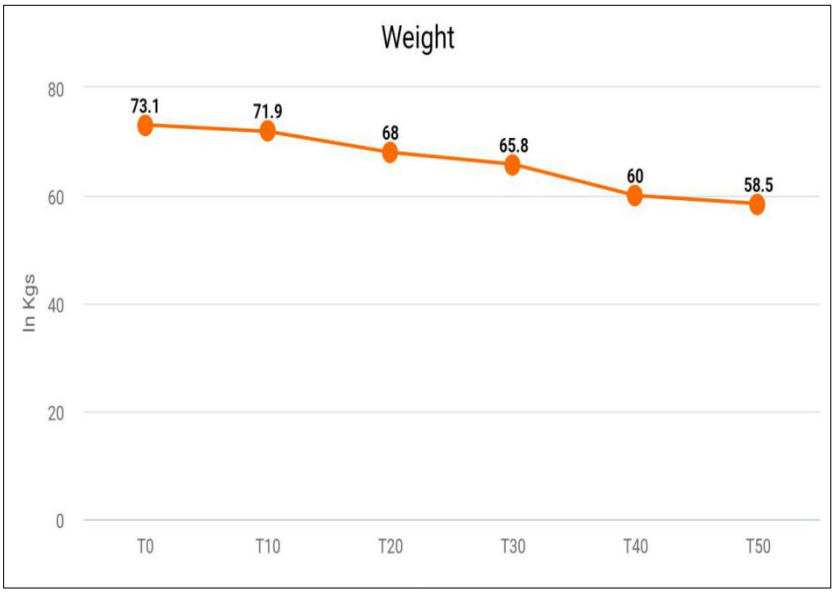

Figure 1 shows trend of weight during treatment phase

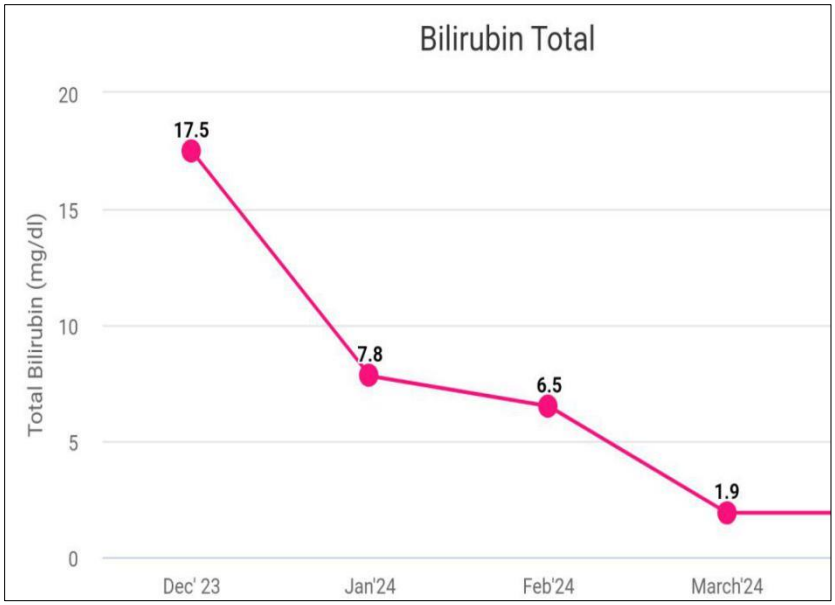

Figure 2 shows trend of S. Bilirubin(total) during treatment phase

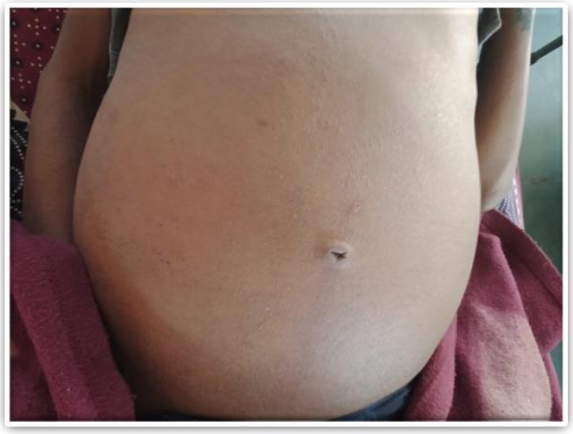

Figure-3.1: Before treatment

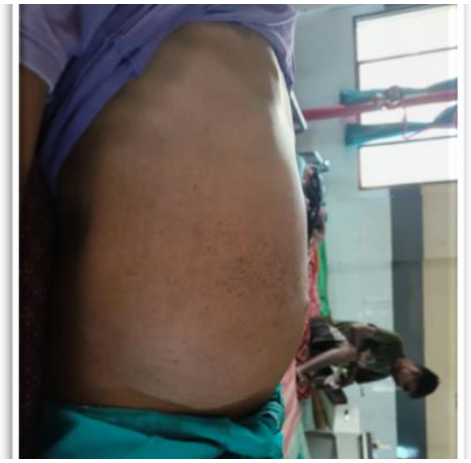

Figure-3.2: After treatment

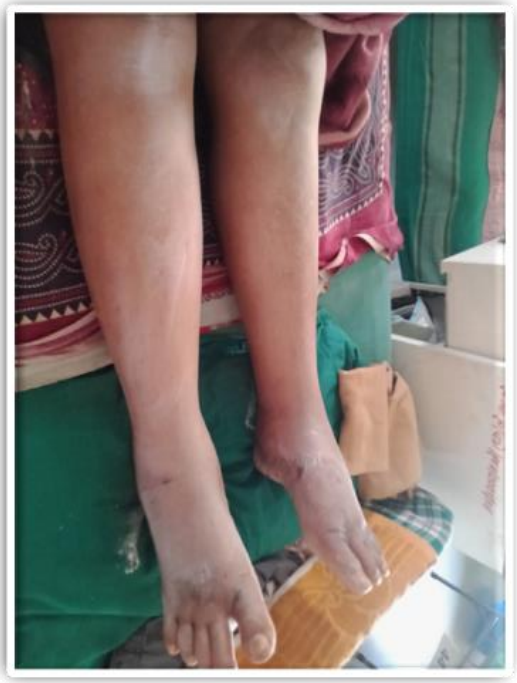

Figure-4.1: Before treatment

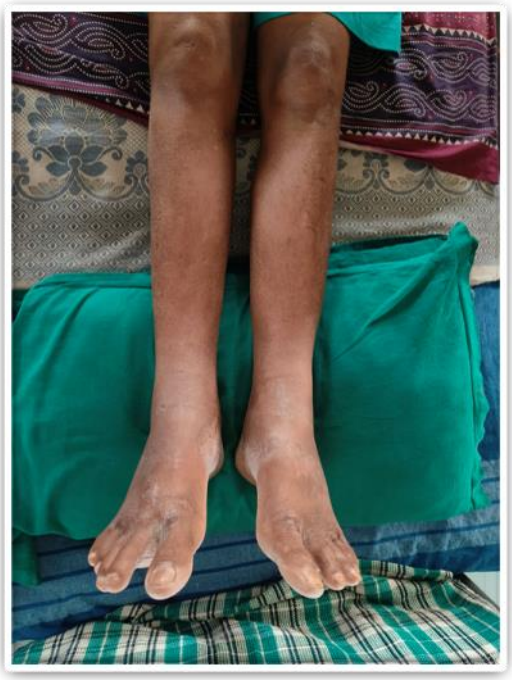

Figure-4.2: After treatment

DISCUSSION:

This case is the classical example of the potency that is carried by *Ayurveda*. Acharya Charaka explains *Jalodara* in *Chikitsa sthana* with its line of treatment [11]. Main causative factor is this case is regular alcohol consumption that lead to *Tridosha* vitiation and diminished the digestive fire i.e *Manda Agni* [12]. Continuing the causative factors in primary stage leaded to increased severity of symptoms. First step in treatment is "*Nidana Parivanjana*". Here patient was advised to stop alcohol and follow *Pathya Aahar Vihar*. *All Udararoga* is considered as *Kruchha Sadhya* from its manifestation of symptoms. The main causative factor is *Manda Agni* in present due excessive alcohol consumption. The main aim of treatment is to reduce peripheral edema and improvement of appetite of patient by reducing altered liver enzymes.

Line of treatment includes use of *Kaphahara Dravya , Shothahara* to clear accumulated *Udaka*(ascitic fluid) along with *Deepana Dravya* to provoke the *Agni* and *Virechak Dravya* to clear out vitiated *pitta*. *Trikatu Churna- Sunthi*(*Zingibar officinale*)+*Maricha*(*Piper nigrum*)+*Pippali*(*Piper longum*) was administered 1gms 12 hrly. This yield excellent result in boosting appetite due to *ushna, tikshna guna*[13]. For *nitya Virechana Haritaki* (*Terminalia Chebula*) 10 gms with *Guda*(jaggery) was given empty stomach as mentioned by acharya Charaka in *Udara Chikitsa*[14]. To give synergetic effect to *Haritaki* the strong purgative *Katuki Churna (Picorrhiza kurroa*) was also given. This combination resulted into passing stool around 8-12 times per day, that clears vitiated dosha and helps in *Stroto Shuddhi*. For pedal edema, one of the *Dravya* of *Shothahara Gana* mentioned by *Charaka Acharya* was used in management i.e *Punarnava* (*Boerhavia diffusa*). For aiding elastocrepe bandaging was done of both lower limb to provide mechanical pressure from outer source.it was kept was kept for 14 hrs per day. Combination of *Punarnava*, *Kalamegha* and *Guduchi* was given due rejuvenating properties as well as hepato protective agent[15][16][17] .

Arogyavardini Vati being proven as excellent *Rakta Sodhak* , it reduces deposition of fat droplets over liver thus prevents further cirrhosis of liver[18]. It yields best result in early stage of steatosis. But, in this case it is administered as preventing agent. A course of *Niruha Basti* was given for alleviating *Vata* resulting into relief in poly arthralgia and vomiting [19]. So combination of *Deepana-Pachana-Nitya Virechana-Rakta Shodhan*, symptoms are subsided without causing any major complication throughout treatment phase.

CONCLUSION:

From above points, we can conclude that ayurveda can play role to get significant results in ascites. Patient got 80-90% relief in his symptoms. One of the reasons of good result is excellent patient compliance. Following the basic line of treatment described by acharya *Charaka* leads to extra ordinary results including the symptoms and biochemistry. One of the best parts is unlike often recurrence nature of ascites, in this case recurrence or worsening of present condition was not observed. From this point we can conclude that strict diet along with *Nitya Virechana- Deepana* one can manage ascites secondary to end stage liver disease. This also increases the wellbeing and life expectancy of patient.

Limitation of study:

As this is a single case study, it is difficult to generalize the study's validity and impossible to establish a cause - effect relationship. As a result, we recommend conducting large - scale clinical trials

Declaration of patient consent:

The authors certify that they have obtained all appropriate patient consent for using clinical information reporting in the journal. The patient understand that his name and initial will not be published and due effort will be made to conceal patient's identity.

Conflict of interest: Author declares that there is no conflict of interest.

Guarantor*:* Corresponding author is guarantor of this article and its contents.

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