ORIGINAL RESEARCH PAPER

Ayurveda

MANAGEMENT OF ALCOHOLIC LIVER DISEASE THROUGH AYURVEDA - A CASE STUDY

KEY WORDS: Ascites, Alcoholic liver disease, Jaloder, Ayurvedic management

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ABSTRACT

In Alcoholic Liver Disease (ALD) there is destruction and regeneration of liver parenchyma leading to fibrosis and cirrhosis.Use of alcohol in excess quantity, duration and patterns of drinking is the commonest cause of ALD. A 52 years old male visited OPD complaining of anorexia, distended abdomen, yellowish discoloration of eyes and yellow urine, dysponea, bilateral pedal edemasince 3-4 months, but since 1 month there was increasein all above symptoms. Serum Bilirubin, SGOT were elevated. USG showed ascites with chronic parenchymal disease (cirrhosis). Based on the history and clinical examination, the condition was diagnosed as jalodara due to Yakritodar. The treatment administered in this condition was Agni dipana, Nitya virechana and also Shamana medications. During this treatment, improvement was seen in appetite. Bilateral pedal edema, abdominal girth was reduced. Total bilirubin, SGOT was reduced. These observations show that Ayurveda treatment is beneficial in cases where liver functions are altered and leads in complications like ascites.

INTRODUCTION:

Chronic and excessive ingestion ofalcohol is common cause of alcoholic liver disease all over the world including India.[1] Alcoholic Liver Disease (ALD) involves a process of progressive destruction and regeneration of liver parenchyma leading to fibrosis and cirrhosis. Alcohol consumption exceeding 60-80 gm/day in men and 20 gm/day in women increases the risk of liver injury.[2] About 90% of patients found to have Ascites due to Liver Cirrhosis, abdominal neoplasm, CHF or abdominal TB and out of which 75% are due to Alcoholic Cirrhosis only[3]. The pathogenesis of alcoholic injury is unclear but liver injury associated with acute and chronic alcoholism. There are three stages in alcoholic liver disease i.e. alcoholic steatosis (fatty liver), alcoholic hepatitis and alcoholic cirrhosis.[4]

Alcohol acts as a direct hepatotoxin. Consumption of alcohol initiates inflammatory cascade by its metabolism to acetaldehyde, resulting in a variety of metabolic responses. Steatosis from lipogenesis, fatty acid synthesis, and depression of fatty acid oxidation appears secondary to effects on sterol regulatory transcription factor and peroxisome proliferator-activated receptor α (PPAR-α). Chronic alcohol ingestion causes hepatocyte injury and impaired regeneration leads to fibrogenesis[5].

Patients with fatty liver will present with right upper quadrant discomfort, nausea and rarely jaundice and patients with Alcoholic hepatitis usually present with fever and signs of hepatocellular failure such as jaundice, ascites, encephalopathy[6] and patients with cirrhotic changes presents with jaundice, ascites and peripheral edema.[7] Jaundice is usually due to failure of hepatocytes to excrete bilirubin resulting into conjugated hyperbilirubinemia. The normal parenchyma of liver is replaced by scar tissue in cirrhosis, therebyincreasing resistance to blood flow causing higher pressure in portal venous system finally resulting in portal hypertension. Ascites is accumulation of fluid in the peritoneal cavity, which is sequel of portal hypertension.[8] The management of ascites in modern science is by appropriate use of diuretics, paracentesis and other medications depending upon the cause and symptoms manifested.

Ayurveda correlates Alcoholic liver disease with Jalodara characterized by accumulation of fluid in Udara which is a complication of Yakritodara. Excessive consumption of ushna (hot), kshara (alkaline), lavana (salty), vidahi, amla (sour), ruksha (dry), virruddhaahara etc. in persons with mandagni(decreased digestive functions) aggrevates Pitta, Kapha and Vata that obstructs Udakavaha and Swedavahasrotas and then accumulation of fluid into the Udara by Upasnehanyaya (osmosis and altered capillary pressure) and then result in formation of different Udararogas[9,10].Nitya virechana, agnideepana and surgical interventions are mentioned procedures in the management of Jalodara[11,12].

CASE REPORT:

A 52 year's old male patient, hypertensive since 2 yr under tab. Amlokind AT, non diabeticapproached the OPD with chief complaints of kshudhaalpata (anorexia), udaravridhi (distended abdomen), ubhayapada shotha (bilateral pedal edema), peetanetrata (icterus), peetamutrata (yellowish urine), dysponea since 3-4 months but there was increase in symptoms since 1 month. He was a known alcoholic since 25-30 years.

Examination: General examination revealedpeetanetrata, ubhayapada shotha (bilateral pedal edema). On examination weight was 80kg, pulse rate was 84/minute and blood pressure was 110/70 mm of Hg. On inspection; udarautsedha with transverse umbilicus was observed. On palpation, udara was mridu (soft and non tender). On percussion, udakapoornadrutisankshobha (fluid thrill), shifting dullness and horse shoe dullness were observed. USG abdomen revealed cirrhotic liver, mild spleenomegaly and severetappableasites, secondary to portal hypertension. Liver function tests presented with elevated bilirubin, SGOT. The satva and samhanana of the patient were madhyama. Prakriti was vatapradhanakapha.

Diagnosis: Based on the clinical presentation and examination with radiological and laboratory tests; the patient was diagnosed with chronic liver disease with severe ascites. This manifestation is compared with jalodara due to yakritodara.

Treatment adopted: Shamana medications include Hinguvachadichurna 500mg, Avipattikarchurna 1gm, Palashkshar 30mg withanupanaadrakaswarasa and madhu,Sharapunkhamula 2gm withanupanatakra TDS. After 12 days of above treatmentSharapunkhamulachurna dose was increased by 2gm i.e. 4gm of sharapunkhamulachurna was given to patient. Similarly after 20 days, hinguvachadichurna, avipattikarachurna and palashakshar

dose was changed to twice a day. After 19 day,1gm Pippalimulachurna was givensamanakali i.e. with meal. After 30 days of treatment, Vardhman pippali rasayana with milk TDS given for 42 days.Nitya virechana with 20ml Dantyarishta in afternoon and night after meal.Tying Erandatailabharjita leaves of arka(Calatropisprocera) on udara andapplication of punarnava and hinguvachadilepa on ubhyapada. On the 3rd day of admission shastrakarma(Paracentesis) done.

DISCUSSION:

Liver is palpable in early stages of liver cirrhosis, but as the condition progresses, it starts to shrink and cannot be palpable in advanced stages. In current case vitiation of pitta and vatadosha, rasa and raktadushya, as a consequence of excess alcohol consumption, results in accumulation of toxins in liver. The deranged raktadhatu gets accumulated in liver and spleen that are the raktavahastotomula leading to their vridhi (hepato-spleenomegaly).[13] When the vitiated rakta in liver and spleen blocked by kaphadosha, hyperactivity of these organs causing in altered blood profile leading to the manifestation of pandu (anemia), kamala (jaundice) etc.[14]

The treatment principle of udararoga isdipana chikitsa and nitya virechana, balaprapti, yakritottejaka chikitsa. As the patient is of Madhyama bala, vatakaphaprakruti, Madhyama satva;on 3rd day of admission, shastrakarma (paracentesis) was done and then cotton cloth was applied around the abdomen tightly to prevent recurrence of asitic fluid. Removal of asitic fluid reduced tension over diaphragm and helped to get relief in dysponea. Deepanapachana of dosha is done with help of hinguvachadichurna, avipattikarachurana andpalashakshara. Hinguvachadichurna, Palashkshar is kaphavatashamaka; its deepana, pachanagunas increases agni i.e. improves digestive system and vatanuloman, lekhan and bhedana properties reduces the obstruction of srotasa.Avipattikarachurna helps to improve overall digestion and improve loss of appetite; it also acts as amruduvirechaka, pitta rechaka. Anupana of all three drugs is adrakaswarasa and madhu to increase its efficacy. Nitya virechana was planned to eliminate the sanchitadosha with the help of dantyarishta, which is anulomaka, yakrutottejaka and also used in udara. Sharapunkhamula (Tephrosiapurpura) has hepato-protective properties that prevents fibrous changes and promotes regeneration of parenchymal tissue. It has a prabhav like plihaghna (reduces spleenomegaly) and also it is yakrutottejaka. Pippali mula due to its katu rasa katuvipakaushnavirya it is kaphavatahara and also it has plihaghna, deepanapachan properties. Vardhaman pippali rasayana, pippali has properties liketikshna, ushna, dipaniya, kaphahar. It is akaturasatmakatikshna-ushnaviryadravya, therefore it helps to improve appetite,it has hepato-protective activity and rejuvenation.Arkapatrabandhanareduces vataprakopa due to its mruduswedana. It reduced the srotorodh in udara.

Role of diet is as important as medicine in jalodara. Godugdha is the only complete food which is full of nutrients and easily digestible. Takrapana, Chapati made of jwara is supplementary food with godugdha.

With the intervention of all these shodhana, shaman and surgical procedures (paracentesis); liver function tests showed marked improvement, appetite was improved, abdominal girth reduced significantly. Pedal edema was also reduced significantly.

Table no 1: Observations of abdominal measurements

Date	Circumference at umbilicus	1" above umbilicus	1" below umbilicus
28/6/18	108 cm	110 cm	106 cm
4/7/18	108 cm	110 cm	105 cm
9/7/18	102 cm	100 cm	98 cm
12/7/18	98 cm	96 cm	94 cm
16/7/18	92 cm	90 cm	90 cm

Table no 2: Liver Function Tests, Haemoglobin

Parameters	27/6/18	10/7/18
Total bilirubin (mg/dl)	5	2.4
Direct bilirubin (mg/dl)	2.5	1.1
Indirect bilirubin (mg/dl)	2.5	1.3
SGPT (IU/L)	38	21.7
SGOT (IU/L)	109	80.2
Serum Albumin (g/dl)	2.9	2.1
Sr. Total protein (g/dl)	9	6.7
Alkaline phosphatase (IU/L)	81	59
Haemoglobin	10.6	10.4

Table no. 3: USG (Abdomen and Pelvis)

Date	USG (A+P)
28/6/18	Cirrhotic liver, mild spleenomegaly and severe tappableasities are secondary to portal hypertension.
26/7/18	Hepatic appearance suggests alcoholic liver disease. Marginal spleenomegalyseen.Noasites seen
14/8/18	Ascites has completely regressed. Spleen is normal in size. Liver also appears normal in size.

On discharge, the condition of the patient was good. Appetite and general health were improved. Direct, indirect bilirubin and SGOT levels were reduced considerably. Icterus was reduced, pedal edema was reduced.

CONCLUSION:

Results observed in this case shows the importance of ayurveda treatment modalities in the management of manifestations, like Alcoholic liver disease.

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