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SYNERGISTIC APPROACHES TO JALODARA (ASCITES) IN ALCOHOL-ASSOCIATED LIVER DYSFUNCTION (AALD): INTEGRATING AYURVEDIC PROTOCOLS WITH MODERN THERAPEUTICS- A REVIEW

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ABSTRACT

Introduction:

Alcohol-Associated Liver Dysfunction (AALD) is a major global health issue, often progressing to conditions like cirrhosis and ascites (Jalodara), the latter characterized by excessive fluid accumulation in the abdomen. Current treatments face limitations such as high costs, recurrence, and long-term management challenges.

Methods:

This review explores modern medical and Ayurvedic management approaches for alcohol-induced cirrhosis with ascites. Modern methods, including diuretics, paracentesis, and liver transplantation, are compared with Ayurvedic protocols, such as Virechana (purgation) and herbal therapies like

Arogyavardhini Vati and Punarnava Mandoora. Dietary restrictions, particularly salt and water intake, are also evaluated.

Results:

Modern treatments offer symptom relief but are associated with risks of recurrence and financial burden. In contrast, Ayurvedic therapies emphasize liver protection, detoxification, and the prevention of fluid retention. Ayurvedic dietary guidelines (Pathya) include hepatoprotective foods like Mudga Amalaki Yusha, supporting liver function and minimizing ascites. Combined, these approaches target both symptom management and disease progression.

Discussion:

A synergistic treatment strategy incorporating Ayurvedic and modern therapies offers a comprehensive approach to AALD management. While modern medicine provides immediate relief, Ayurveda focuses on long-term liver health and holistic detoxification. Lifelong alcohol abstinence is crucial for treatment success, and further research is needed to validate the effectiveness of integrated approaches.

Conclusion:

Ayurvedic protocols complement modern treatments in managing AALD with ascites, offering a holistic and preventive approach. However, more clinical studies are necessary to establish their broader application and efficacy in clinical practice. Alcoholic Liver Disease (ALD) is a significant contributor to global liver-related morbidity, encompassing conditions like fatty liver, alcoholic hepatitis, and cirrhosis. ALD often progresses to cirrhosis and ascites, a condition marked by fluid accumulation in the abdomen, posing treatment challenges.

Keywords: Alcohol-Associated Liver Dysfunction (AALD), Cirrhosis and Ascites (Jalodara)

INTRODUCTION

As a part of life, alcohol consumption is often linked to festivities and pleasure for a large number of people. At present, alcohol ranks third globally in terms of risk factors that contribute to an increased burden of illness. Meanwhile, it has been demonstrated that alcohol is a strong liver toxin. A higher incidence of liver disorders is linked to its consumption. Alcohol consumption disorders account for over a million cases annually **[1]**, making them the most prevalent substance misuse diseases worldwide. In India, liver disease accounts for 3.5 million fatalities worldwide and 3% of all deaths, according to WHO statistics released in 2018 **[2]**. Male or female, liver cirrhosis mortality is directly related to the people who use it and is directly linked to alcohol volume and is correlated with alcohol intake. Alcoholic Liver Disease (ALD) is the aggregate term for a number of liver illnesses brought on by alcohol misuse **[3]**. ALD is a phrase that includes liver failure, cirrhosis, hepatocellular cancer, fatty liver, and alcoholic hepatitis **[4]**. Steatosis, often known as fatty liver, is the

most frequently formed early reaction in patients. One of its characteristics is the accumulation of fat molecules in the liver **[5]**. Alcoholic hepatitis is classified as an inflammatory liver damage that is typified by enlarged, dying hepatocytes and the formation of knotted clumps of insoluble proteins inside the hepatocytes called Mallory-Denk bodies.

Cirrhosis and alcoholic fibrosis: Fibrosis and its final stage, cirrhosis, are caused by aberrant extracellular matrix protein deposition, mostly by activated hepatic stellate cell. The development of cirrhosis proceeds from a period of compensation to a phase of decompensation, during which the organ is completely encased in scar tissue **[6]**. The latter is typified by the development of hepatic failure and/or portal hypertension. Ascites is thought to be a clinical outcome of portal hypertension, characterised by an abnormal build-up of serous fluid in the abdominal cavity's gaps between tissues and organs **[7]**.

Ayurveda provides a comprehensive explanation of hepatobiliary illnesses within the framework of Yakrddalyudara, or the functional dysfunction of the liver resulting in Udara vruddhi, or abdominal distension, also known as Udara roga.Jalodara is one of the eight varieties of Udara.

Acharya Charaka has declared it to be an irreversible illness, whereas Sushruta referred to all Udara rogas as "mahagada—a serious condition that is challenging to cure" **[8]**. Emaciation of the palm and sole **[9]**, oedema, and accumulation of free fluid in the belly are clinical signs of Jalodara.

Finally, all eight types of Udara—Yakruta and Pleehodara included—arrive in Jalodara. In the classics, many therapy strategies like as Shaman, Rakta mokshana, Virechana, and Shastra karma have been discussed for controlling alcohol-induced Jalodara. The treatment of this illness is complicated and difficult as the patient must make a lifelong commitment to not drink alcohol in order to begin the healing process. Contemporary management of Alcoholic Liver Cirrhosis induced Ascites aims towards removal of free fluid from the peritoneal cavity by tapping, administering Diuretics and managing symptomatically, which is at the risk of reoccurrence. Liver transplantation is also the choice of management at the end stage liver disease, but is expensive and risky for patient’s future life. Protection of liver from the toxic effect of Alcohol and arresting its pathology to slow down the progression by using effective Ayurveda treatment modalities like Nitya Virechana, Shaman in terms of Hepato protective, Hepatic stimulant drugs and Diet regimen is need of the hour.

Today's treatment for alcohol-induced livercirrhosis-induced ascites aims to remove free fluid from the peritoneal cavity by tapping, giving diuretics, and managing

symptomatically, which is at risk of recurrence. Liver transplantation is also an option for managing end-stage liver disease, but it is costly and dangerous for the patient's future. Effective Ayurvedic treatment modalities like Nitya Virechana, Shaman in terms of hepatoprotective, hepatic stimulant drugs, and diet regimen are essential. ALD requires a unique treatment strategy for its remission since it is a complicated disorder that involves all three doshas and changes over time. Many Ayurvedic drugs have been studied and shown effective separately. Under this perspective, the current study set out to assess the overall clinical effectiveness of the Ayurvedic therapy protocol in the management of Jalodara w.s.r. to Alcoholic Liver Disease.

Anulomaka, or haritaki, is a proven hepatoprotective utilised in Ayurvedic treatment protocols to eliminate the atimatra dosha from the body through the anal channel. Preliminary research suggests that Arogyavardhini Vati, Punarnava Mandoora, and Patola Katurihinyadi Kashaya are frequently used medications for liver problems.

Arogyavardhini Vati, Punarnava Mandoora, and Patola Katihinyadi Kashaya are commonly used drugs with preliminary evidence in liver disorders. Bhunimbadi Churna has properties of Deepana, Pachana, and Lekhana. Pathya-In Ascites, water and salt restriction is needed to control water retention in the body. Kharjura Draksha Mantha, Mudga Amalaki Yusha will aid in catalysing the medicinal properties, and milk will support hypoprotenemia, the main pathological cause of Ascites.

To evaluate the liver's surface and fluid content, ultrasound was used. Tests for liver function and other laboratory indicators were used to identify and categorise liver diseases, gauge the degree of liver damage, and monitor therapy response.

JLODARA(ACITIS) as UDARA-ROGA (GID)

Hepatobiliary diseases are extensively described in Ayurvedic texts. Yakrddalyudara is the term for the distension of the belly (Udara vruddhi) brought on by the functional dysfunction of the liver. One of the eight varieties of Udara is jalodara, which requires careful management.

The oldest known source of knowledge about illnesses and the therapeutic use of plants is said to be found in the Vedas. We may find references to Jalodara in the Atharva Veda, which is regarded as a legitimate source of Ayurveda. Jalodara was said to be caused by the god Varuna's curse **[10]**.

Charaka and Sushruta Samhita[11] provide a detailed account of the diseases Yakritodara and Jalodara. Acharya Sushruta referred to all Udararoga as "mahagada," which refers

to severe illnesses that are difficult to cure, and Acharya Charaka declared this to be an incurable condition.

There is a detailed account of Yakritodara and Jalodara in Astanga Hridaya **[12]** and Sangraha **[13]**.

The author Chakrapani offered commentary on the depiction of Yakritodara and Jalodara in Chakradatta **[14]**. Jalodara has been remarked upon by Dalhana and Indu, the commentators of Sushruta Samhita and Astanga Hridaya, respectively. In Madhava Nidana **[15]**, Madhavakara provides an explanation of the Yakritodara.

There are references to the therapy of Jalodara and the categorisation of Udara in Sharangadhara **[16]** and Bhavaprakash Samhita **[17]**. The categorisation and therapy of the ailment have been detailed by Bhavaprakash in a different chapter, Yakrit Rogadhikara. We find the mention of Yakritodara and Jalodara, which are originally from Sushruta, in Yogaratnakara **[18]**. Only the Chikitsa aspects of udara and Jalodara are discussed in Bhaishajya Ratnavali **[19]**.

Modern Ayurvedic doctors explain Yakrit-related illnesses in light of scientific advancements. The Greek word for ascites is "askos," which meaning sack.

YAKRIT (Liver)

Sushruta and Charaka acharyas have recognised the Yakrit as the moola sthana of Rakta vaha srotas **[20]** from ancient times. One of the koshtangas, it is located in the direction of abdomen's right side, down to the heart. It is the principal seat of the Ranjka pitta and is descended from Rakta in the foetal stage.

UDARA(Abdomen)

Definition: According to Acharya Charaka, Shotha is produced when Vata aggravates and takes sthanasamshraya between twak and mamsa; this is referred to as Udara when it happens in Kukshi.

General Udara Roga **[25]** Symptoms & Signs

General signs and symptoms of udara roga include gaseous distention of the belly, constipation, burning feeling, lethargy, debility, impaired digestive capacity, oedema, numbness of body parts, and buildup of flatus.

There are eight techniques to make bheda-udara rogas.

1. Vatodara, 2.Pittodara, 3.Kaphodara, 4.Sannipatodara, 5.Pleehodara, 6.Baddhodara, 7.Kshatodara, 8.Jalodara.

The upper abdomen is the site of Kshatodara manifestation. The origin of Baddhodara lies below the umbilicus. Pleehodara originates in the umbilical area, whereas udakodara can appear anywhere in the abdomen.

Jalakurmodara Symptoms

Patients at Jalodara are discovered to have significant abdominal distention, which has an elevated form like to a tortoise's back. In

this case, vata and kaphadoshas are predominant. This illness, called jalakurmodara, is extremely difficult to treat. Avastha **[22]**.

Ajatodaka laxana is characterised by the absence of shotha over the abdomen, which is aruna in colour, sounds, feels heavy, causes gudaguda, is covered in sirajala, and vayu is produced and eliminated extensively. Ajatodakavastha, on the other hand, is characterised by the absence of fluid accumulation, no mandagni, no fluid retention in the abdomen, no movement, or very little movement, mutralpata, and obstruction of faeces.

The abdominal cavity of Picchila Avastha has a buildup of sticky fluid.

The characteristics of Jatodaka laxana-Kukshi vruddhi include the absence of sirajala and the look of the belly like an air-filled leather bag, which denotes fluid buildup.

Dietary alcoholic syndrome **[23]** Alcohol-related liver disease (ALD) includes fatty liver, alcoholic hepatitis, and alcoholic cirrhosis, among other liver damage brought on by excessive alcohol intake. There will be fat infiltration in the liver cells of those with fatty liver disease. Hepatocytes become inflamed in alcoholic hepatitis. Cirrhosis is a late stage characterised by fibrosis, which ends in scarring and necrosis **[24]**.

CLINICAL FEATURES [25]: Compressed respiratory function, peripheral oedema, and increased abdominal circumference. In the event of massive ascites: dyspnoea, hepatic hypotrophy, malnourishment, wasting of muscles, excessive fatigue, and weakness.

DIAGNOSIS - Physical Examination: Bulging of Flanks, Fluid Wave or Presence of Shifting Dullness.

Ultrasound or CT scan,

Diagnostic Paracenthesis, Ascetic Fluid Culture.

Analysis of ascetic fluid is useful in the differential diagnosis of Ascites.

NIDANA

The main cause of alcoholic liver damage is alcohol.

Alcohol: The Arabic word "alkuhl," which means "essence," is the source of the English word "alcohol" **[26]** around Arabia, the distillation method was found around the ninth century AD. Section XVII of the Drugs and Cosmetics Rules of 1945 states that preparations containing alcohol produced on-site must have a maximum alcohol content of 16% and be packaged in no more than 120 millilitres **[27]**.

In everyday speech, the phrase "alcohol" refers to ethyl alcohol (C_2H_50H). It is a clear, colourless, flammable liquid with a burning flavour and a distinct spirituous smell.

Repaired spirit has 90% alcohol, absolute alcohol includes 99.95% alcohol, and industrial methylated spirit, often known as

denatured alcohol, is a combination of 95% alcohol and 5% wood naphtha.

Alcoholic drinks consist of a blend of water and alcohol, along with trace levels of congeners created concurrently with the fermentation process. Congeners are made up of organic acids, esters, and methyl alcohol in the case of brandy and wine. After the alcohol has completely been broken down, the stench might linger in the tissues for a few hours.

Since ancient times, alcohol has been utilised for medical purposes. An extensive explanation of Madya's characteristics, kinds, negative effects, etc., can be found in a different categorisation called Madya Vargain.

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When taken correctly, madya functions as "nectar," but when taken incorrectly, it functions as "poison." **[30]**

Madyas used medicinally fall into the following categories.

Prasanna, Kadambari, Jagala, Medaka, Surabija, Sidhu, Varuni, Asava, and Aristha are the Sura members.

Yakrit, Pitta, and Rakta get vitiated due to the madya's vidahi quality.

Navamadya: vidahi, abhishyandi.

Property of Abhishyandi: Kaphaprakopa & Agnimandya. In addition to these, madya has attributes like ashukari **[31]**, teekshna, sookshma, vyavayi, vikasi, and ushna, which would enable madya to enter the body's sukshmasrotases and boost the opposing characteristics of oja while reducing it **[32]**.

Jalodara can also result from eating a hot, spicy, and salty diet, vidahi anna, gara visha sevena, antrasphutan, mandagni, and papa karmi, as well as by not adhering to regimens after Vaman and Virechana karma.

Causes:

The amount and duration of alcohol use are the most significant risk factors for the development of ALD.

Quantity: In males, 40–80 g/d of ethanol develops fatty liver, whereas 160 g/d over a period of 10–20 years results in cirrhosis or hepatitis.

Four ounces of wine, one ounce of beer, or one ounce of 80 percent spirits all have

around 12 grammes of alcohol. Men who drink more than 14 drinks a week and women who drink more than 7 drinks a week are at a greater risk of getting alcoholic liver disease.

Gender: Women are more vulnerable than males.

Concurrent ALD infection with hepatitis C virus (HCV) is linked to severity at younger age.

Genetics: Alcoholic cirrhosis has been linked to patatin like phospholipase domain containing protein 3 (PNPLA3).

Risk factors include non-alcoholic fatty liver and obesity.

PATHOGENESIS OF ALD [26]

The primary metabolism of alcohol, or ethanol, occurs in the hepatocytes of the liver. The primary parenchymal cells in the liver are called hepatocytes. The main enzymes that oxidise ethanol are expressed at the greatest levels in cells. These elevated levels of catalase and alcohol dehydrogenase (ADH) ADH utilises nicotinamide adenine dinucleotide to catalyse the oxidation of ethanol. (NADH), acetaldehyde, and NAD+, which subsequently combine to create the very reactive and hazardous substance.

The oxidation of ethanol to acetaldehyde is catalysed by the hepatic enzyme CYP2E1. Along with aldehyde dehydrogenase's assistance Two enzymes, hepatocytes, do the quick oxidation within the mitochondria and reduces the harmful effects of acetaldehyde. The cellular redox potential results from this. These adjustments favour fatty acids. production and aid in the development of fatty liver.

The Gut–liver Axis- [26]

A Gram-negative bacterium's cell wall moves from the intestinal lumen into the portal circulation to reach the liver. Alcohol causes alterations in both the quality and quantitative makeup of the bacteria that live in the gut microbiota. Through their interaction with toll-like receptor 4, endotoxins in the liver activate hepatic stellate cells and Kupffer cells (TLR4). In addition to proinflammatory cytokines and chemokines, these cells also release reactive oxygen species (ROS), which when combined with alcohol cause damage to the hepatocytes.

The activation of different immune cells (such as neutrophils, T cells, and other Review of leukocytes) by alcohol and the effects of alcohol on fat (adipose) tissue, which cause damage to the liver, are additional variables that cause damage to the liver.

Ayurvedic perspective on madyapana that leads to Jalodara **[33]** Detailed explanation of the pathophysiological alterations brought on by Ayurvedic classics do not contain madya in yakruta sharira. Thus, this an effort is made to shed light on the pathophysiological alterations that are

occurring in Yakruta as a result of Madya Sevana with the assistance of modern idiom. Madya possesses attributes like ashukari, vikasi, and vyavayi. Because of this characteristic It circulates throughout the body after entering the sukshma srotas. reaches the liver by inhibiting the process of digestion. Since vidahi guna causes the first inflammatory reaction and vitiates the pitta. If the factor intake is sustained over time, necrosis will eventually occur. Sharirika and Kriyatmaka vikruti in Yakruta shall be observed at this point.

Yakruta is the ranjaka pitta's seat and the moolasthana of Rakta. Poshya rakta dhatu formation and ranjaka pitta formation are hampered when yakruta vikruti takes place. Therefore, symptoms of pandu roga and Kamala roga may manifest at this period. Additionally, the vitiation of kapha in amashaya and ranjaka pitta vitiation are added by Madya's Abhishyandi guna. The underlying disease causes Agnidushti, which in turn causes Agnimandya. As a result, the person has symptoms like appetite loss that causes ojakshaya, balakshaya, etc. Agnimandya ama uttpati will cause this to happen.

There will be vitiation of the prana, apana, Agni dushti, and the swedavaha and udakavaha srotas. Through the upasnehana process, doshas transit, or this udaka ansha will gather in the peritoneum, or audarya kala. If this avastha is not handled correctly, it will eventually reach Jalodara.

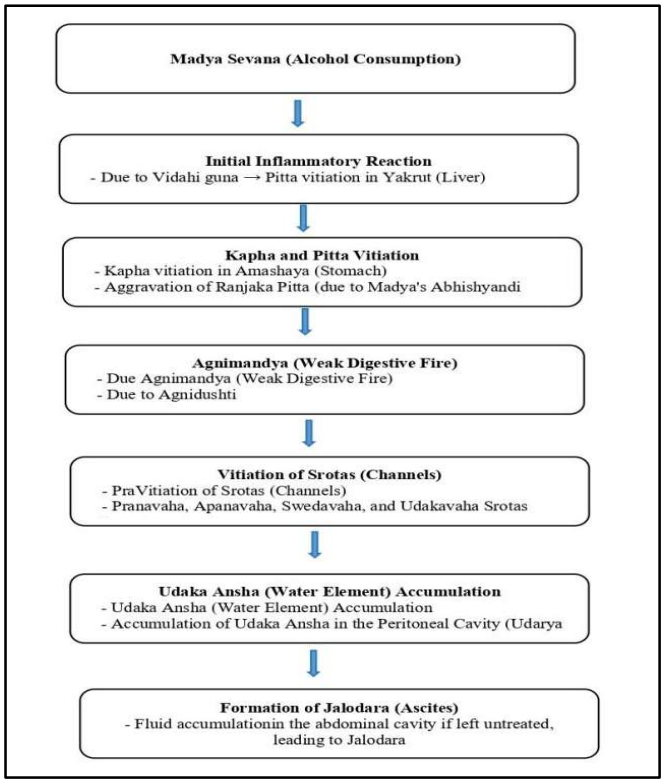

Figure 1: PATHOGENESIS OF JLODARA

SAMPRAPTI GHATAKAS [34]

 Dosha – Vata- Prana, Apana,Vyana,Samana Vata, Pitta – Ranjaka, Pachaka

Pitta Kapha –Avalambaka Kapha, Kledaka Kapha

 Dooshya – Rasa, Rakta, Mamsa, Meda

 Agni - Jatharagni,Ranjakagni,Pachakagni

 Ama – Ranjakagni, Pachakagni Mandya Janya Ama, Jatharagnimandya Janya Ama

 Srotas - Raktavaha Srotas, Swedavaha Srotas, Udakavaha Srotas

 Srotodusti - Sanga

 Udbhavasthana - Amashaya

 Sanchara Sthana - Rakta Vaha Srotas And Its Moola

 Adhishtana - Yakrit

 Vyakta Sthana – Udara

 Roga Marga - Bahya And Abhyantara

POORVA ROOPA

Prodromal symptoms, or poorva roopa, appear before to the disease's full presentation. The dosha that is triggered at the sthana samshraya stage will display the indications and symptoms of the impending udara illness, including 89Kshudhanasha, Ajeerna, Pada Shopha, Balakshaya, Shwasa, Udavarta, Adhmana, Atopa, and Rajijanma. Basti Ruja at Jathara.

ROOPA

The sickness known as dushya samurchana manifests after dosha. At this point, the Roopa symptoms start to show up. They appear at the fifth stage of the kriyakala, or vyaktavastha stage.

In the Kukshi, Padashopha, Mandagni, Karshya, Dourbalya, Vata-Purisha Sanga, Daha, and Trushna, the symptoms include Adhmana and Atopa.

CLINICAL FEATURES

Right upper quadrant discomfort, nausea, vomiting, diarrhoea, anorexia, malaise, and seldom jaundice are symptoms of alcoholic fatty liver disease.

Alcoholic Cirrhosis: Portal Hypertension, Ascites, Variceal Bleeding, Upper Gastrointestinal Haemorrhage, Fever, Spider Nevi, Jaundice, and Abdominal Pain.

UPADRAVAThe complexities of udara include Chardi, Atisara, Tamakashwasa, Trishna, Kasa, Hikka, Dourbalya, Parshwashoola, Aruchi, Svarabheda, and Mutrasanga. Ascites complications include hydrothorax and umbilical hernia.

ARISHTA LAXANA

These are the negative prognostic characteristics of udara that should be disregarded as they are regarded as asadhya. **[35]**

1. Koota Shoonaksha 8. Swayathu
2. Kutilopastham 9. Sarva Marmotha
3. Swasa 10. Upaklinnatanu 3.
4. Hikka 11. Moorcha 5. Aruchihi 11. Satrit
6. Atisara 13. Chardi
7. Tamamsagni Balashoni Priksheenam

PROGNOSIS [36]

Alcoholic hepatitis patients in critical condition had 30-day death rates greater than 50%. A poor prognosis is predicted by the presence of ascites, variceal haemorrhage, profound encephalopathy, or hepatorenal syndrome. Patients with cirrhosis issues who drink still have less than 50% probability of surviving for another five years.

SADHYA ASADHYATA

In comparison to Pleeha or Yakritodara, Doshika varieties have lower kricchra sadhya, whereas Yakritodara has lower kricchrasadhya in comparison to Sannipatodara. When an Udara achieves Jalodakavastha, they are all regarded the mostIf connected to upadravas, krishnasadhya may occasionally be treated with visha and shastra. With difficulty, the one that is new and occurs before jalodara in a robust individual can be cured.

TREATMENT FOR ALD

The cornerstone of therapy for alcoholic liver diseases is total abstinence from alcohol. focus on the psychological and nutritional state. The last resort for individuals with end-stage liver disease is liver transplantation.

The cornerstones of treatment include sodium restriction, bed rest, and diuretic usage.

Dietary sodium reduction for mild ascites. Moderate diuretic treatment involving 100-200 mg/d of spirolactone and 40-80 mg/d of furosemide, along with frequent large volume paracentesis. The best option is a liver transplant.

The most common medical treatment performed for diagnosis with appropriate informed permission is abdominal paracentesis. Most popular site: approx. 15 centimetres to the right lower abdominal quadrant's umbilicus.

CHIKITSA

Chikitsa is the procedure that brings the tridoshas, saptadhatus, and trimalas into balance. Nidana Parivarjana, first of all Chikitsa ought to be taken in. Nidana parivarjana refers to total abstention from alcohol.

As previously mentioned, dushti of ambu and swedavaha srotas are associated with the condition Jalodara. In Udarastha Twacha, the collecting of Apa Dhatu is extensive. Ascites, a complication of liver cirrhosis, can be regarded as jalodara. Thus, fluid will continuously accumulate in the abdominal cavity. It is important to routinely drain the accumulated fluid. Thus, it is best to embrace Virechana in the form of nitya Virechana. There are four kinds of virechana medicines.

1. Anulomana 2. Bhedana 3. Sramsrana 4. Rechana

When describing kala, Sushruta describes purishadhara kala as the inner layer that is present in unduka and aids in mutra and

purisha vibhajana **[37]**. As a result, the Virechana medications in the Pachyamanavastha reach this kala and prevent the partition of the drava and ghanamala. Additionally, because of their veerya, they drag all the fluid in other bodily regions and discharge it through the rectum. Many kinds of laxatives are referenced in contemporary literature. For example, osmotic agents. Osmotic agents pull water into the intestinal lumen by including compounds that are poorly absorbed **[38]**. Virechana medications function in this manner to lessen aap dhatu in the peritoneal cavity.

The classics also discuss Veshtana Chikitsa as a way to lessen the buildup of vata following chedana karma **[39]**.

Raktamokshan: Raktamokashan is recommended at Yakritodara at the Dakshina Bhu by Sira.

Vedhana Chikitsa in udara: this should be followed when there is an excessive buildup of fluid in the abdomen.

A few shamanic remedies:

Patoladi, Narayan, and Pipplyadi are examples of churnas. Churna Hapushadi Churna Nilinyadi

Ghrita Panchakola, Chitraka, and Rohitaka ghritas, Yavadi ghrita

Snuhi kshira ghruta, Kshara Nagaradi ghrita Pippalyadi and Vidangadi kshara

LepaThe classics also explain Chikitsa, which includes Arkapatrapattabandhan, Vastrapattabandhan, Devadaru, Palasha, Arka, Shigru, and Ashwagandha.

PATHYA APATHYA CHIKITSA

Apathya is not conducive to Pathya, which is compatible with the body.

Chikitsa is also known by the synonym pathya. A healthy, beneficial, or favourable regimen, or Pathya, is the only way to treat a disease; medication won't do much good on its own.

The Pathya aharas that are discussed in the context of the Udara basically have two functions.

i) To keep the patients' Agni stable.

ii) To keep the patient's nutritional condition stable.

Shamidhanya cereals, or dicotyledons, are high in protein. Classics also recommend Yusha of Shamidhanya. Proteins, lipids, calcium, riboflavin, vitamin A, and significant levels of other B-vitamins and minerals may all be found in cow's milk.

For this reason, a diet high in milk is thought to be balanced for people with Jalodara.

It is recommended in Yakritodara to cook takra in addition to vacha, shunti, shatahva, and saundhava. It is recommended in Jalodara to make takra in addition to vyosha.

Ksharas.

Asavarishtas are regarded as having a diuretic effect.

i) Ambupana

This is limited fluid consumption. Fluid restriction is required when hyponatraemia

is present. Water intoxication is caused by an increase in serum volume combined with a reduction in blood sodium level. Dilutional hyponatraemia, which is caused by a reduction in urine output, raises blood urea levels, causes sleepiness, and further lowers serum salt levels.

ii) Lavana Sevana

Hepatic cirrhosis with ascites frequently has low serum sodium levels.

When sodium ions are given to these individuals, their serum sodium levels don't rise but their fluid and salt retention does.

Vidahi ahara: Madya is contraindicated since she is vidahi, the cause of ALD.

Diabetics are used to increase the amount of fluid and sodium excreted in the urine, together with a diet high in proteins and low in sodium, as part of the therapy for ascites.

To replenish potassium salts that have been depleted, supplements may be required.

ANALYSATION

LFTs, or biochemical liver function tests, are widely used as screening tests for liver disease diagnosis. This is split up into:

1. Individuals who assess the liver's synthetic function (PT/INR, Sr. Albumin)
2. AST and ALT, which predominantly access hepatocyte damage
3. Those that are most affected by bile flow disorders, such as cholestasis—Bilirubin, AP, and GGT PT/INR Except for factor VIII, which is generated by vascular endothelial cells, all blood clotting factors are synthesised only by hepatocytes.

Serum prothrombin time measures factors VII, X, V, and II together.

Vitamin K is required for the biosynthesis of factors II, VII, IX, and X. The level of anticoagulation on warfarin medication is expressed using the international normalised ratio (INR).

Serum albumin: Hepatocytes are the only cells that can produce it. It has an extended half-life of 18–20 days, degrading by around 4% every day. Hypoalbuminemia is more prevalent in long-term Serum globulins are a class of proteins consisting of alpha and beta globulins that are predominantly generated by B cells and gamma globulins produced by B lymphocytes inside hepatocytes. In cases of chronic liver disease, they are elevated.

AST and ALT are serum enzymes. AST is present in the kidneys, brain, liver, cardiac muscle, and skeletal muscle. Leukocytes, erythrocytes, pancreas, and lungs in decreasing order of focus. Since ALT is predominantly present in the liver, it is a more focused a sign of damage to the liver. Amino transferases have a range that changes normally. broadly between 10–40 IU/L The diagnostic indicator for alcoholic liver cirrhosis is AST: AST<400IU/L, ALT>3:1, and the ALT is frequently normal because of alcohol caused pyridoxal phosphate deficiency.

The breakdown product of the porphyrin ring in proteins containing heme is bilirubin. There are two fractions of it in blood: conjugated and unconjugated.

Uncojugated bilirubin is bonded to albumin in the blood and is insoluble in water. The conjugated Bilirubin is water soluble and can therefore be excreted by the kidney. Normal values of total serum Bilirubin are reported to be between 1 to 1.5 mg/dL.

Alkaline Phosphatase- is present in canalicular membrane of the hepatocytes; GGT (Gamma Glutamyl Transpeptidase) is located in endoplasmic reticulum and in bile duct epithelial cells.

Other diagnostic test-Ammonia- During normal protein metabolism ammonia is produced in the body. The liver plays a role in the detoxification of ammonia converting it to urea, which is excreted by the kidneys.

Liver Biopsy: This can be done via laparoscopic, transjugular, or percutaneous methods. The percutaneous method is a safe procedure that can be easily carried out at the patient's bedside with the help of ultrasound guidance and local anaesthesia. Multiparameter blood tests are the most effective non-invasive test to detect hepatic fibrosis; ERCP, EUS, PTC, and Hepatic / Abdominal visceral angiography are among the invasive imaging studies that involve invasive imaging techniques. Computed tomography, radionuclide scanning, angiography, and radionuclide scanning are the methods used to study the liver disease.

In particular, ultrasound is helpful and is often thought of as a continuation of clinical evaluation. Sonographic characteristics associated with cirrhosis include regenerating nodules encircled by fibrous septae, a nodular surface, and coarse echogenic echo texture. A liver ratio of higher than 0.65 between the caudate and right lobes is highly specific for cirrhosis.

Imaging's function in ascites Radiologic tests are helpful in determining the aetiology of ascitis and in detecting minute quantities of ascetic fluid. As little as 100 millilitres of intraperitoneal fluid can be found using abdominal sonography. Even lower amounts of fluid are detected by CT.

DISCUSSION

Alcohol consumption, while socially prevalent, is a significant contributor to liver diseases globally. Alcoholic Liver Disease (ALD) includes conditions like fatty liver, hepatitis, and cirrhosis. Early-stage fatty liver results from fat accumulation in liver cells, while alcoholic hepatitis is marked by inflammation and hepatocyte damage. Continued alcohol use leads to fibrosis and cirrhosis, which ultimately cause liver failure and complications like portal hypertension and ascites—fluid accumulation in the abdominal cavity.

In Ayurveda, liver dysfunction due to alcohol is linked to Yakritodara, leading to

abdominal distension, with Jalodara being a specific form of Udara Roga resembling ascites. Ancient texts, such as those by Charaka and Sushruta, describe Jalodara as a severe and hard-to-treat condition. Classical Ayurvedic therapies include Shamana, Virechana, and Shastra Karma, aiming to detoxify the body and restore liver function. Herbal treatments like Arogyavardhini Vati, Punarnava Mandoora, and Patola Katurihinyadi Kashaya are used for their liver-protective and diuretic effects. Dietary restrictions on salt and water are also emphasized.

Modern treatments focus on fluid removal and diuretics, but recurrence is common. Liver transplantation remains the last resort, though it is costly and carries risks. Ayurveda's holistic approach, focusing on detoxification and hepatoprotective remedies, may complement modern treatments, emphasizing the need for lifestyle changes, including alcohol cessation, to manage ALD effectively. Pathya is a component of the Ayurvedic therapy regimen. Salt restriction is necessary to manage the body's water retention in cases of liver disease or ascites. The medicinal effects of Kharjura Draksha Mantha and Mudga Amalaki Yusha will be catalysed, and 500 millilitres of milk every day will assist to preserve hypoprotenemia, the primary pathological cause of ascites.

Salt limited diet: It was suggested to use less salt in the diet in order to create a negative sodium balance and prevent fluid retention. By reducing vascular volume, a salt-restricted diet decreases portal pressure and prevents further fluid buildup **[36]**.

Draksha Mantha and Kharjura: Kharjura is a vatapittashamaka, shita veerya, and madhura rasatmaka. A good source of vitamin B complex is kharjura.

It has potential antioxidant action. Thiamine is a crucial nutrient and in alcohol withdrawal its deficit develops hence Kharjura provided it.Draksha is shita viryatmaka, amla rasatmaka, and madhura. Ripe fruits have aphrodisiac, laxative, diuretic, antioxidant, and hepatoprotective properties **[41]**.,

The Mudga Amalaki Yusha – Mudga is Sanskrit for "which brings joy" or "delight." Qualities of mudga include shita veerya, katu vipaka, laghu, ruksha, madhura, and kashaya rasa. Mudga is an excellent source of proteins and carbs.It is also an excellent source of vitamins and minerals, such as thiamine and riboflavin. Organic acids, flavonoids, and dietary iron are all present in large amounts **[42]**.

As per Ayurvedic classics, amalaki possesses the qualities of rasayana, amla, madhura, kashaya, katu, tikta rasa, shita virya, and sara. It is a highly nutritious food that is rich in minerals, amino acids, and vitamins. It also acts as a diuretic, liver tonic, and reduces fluid volume in the extracellular

space. Water restriction up to 1lit/day: cirrhotic ascites is primarily caused by impaired renal sodium excretion, which results in positive sodium balance and water retention. Restricting water intake is advised to lessen the extracellular fluid volume. Milk: this food type contains essential amino acids, calcium, phosphorus, vitamin A, lactose, lactose, and essential fatty acids. It is both balya and rechaka.

CONCLUSION

Alcohol-induced liver disease represents a significant global health burden, particularly due to its progression from reversible fatty liver to irreversible cirrhosis and ascites. Modern medical approaches primarily focus on managing symptoms and prolonging life through interventions like diuretics, paracentesis, and liver transplantation. In contrast, Ayurveda offers a holistic approach to the management of Jalodara, involving a combination of detoxification therapies, herbal formulations, and lifestyle modifications aimed at reversing or slowing the disease progression.

Ayurvedic treatments such as Nitya Virechana and the use of hepatoprotective medications could offer complementary benefits, particularly in preventing the toxic effects of alcohol on the liver. However, patient compliance with a lifelong alcohol abstinence is crucial to achieving long-term remission. While more research is needed to validate Ayurvedic approaches within a modern clinical framework, integrative therapies offer promise for enhancing the quality of life in patients suffering from Alcoholic Liver Disease.

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