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Case Study

SUCCESSFUL AYURVEDA MANAGEMENT OF ALCOHOLIC LIVER DISEASE

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ABSTRACT

Alcoholic liver disease (ALD) encompasses a spectrum of liver conditions caused by chronic alcohol use, beginning with fatty liver, advancing to alcoholic hepatitis, and culminating in alcoholic cirrhosis- the irreversible and most severe stage of alcohol-induced liver damage. Diagnostic criteria for ALD are based on liver function tests and ultrasound (USG) of the abdomen to assess changes in liver function and structure. *Yakritddalyudara* and the signs and symptoms of ALD go parallel to each other. A 50-year-old male with a 22-year history of chronic alcohol consumption presents with yellowing of the sclera and urine, loss of appetite, abdominal distension, and bilateral lower limb and abdominal pain persisting for the past week. With the increased level of LFT and USG-abdomen report suggesting hepatomegaly with fatty changes, was examined and diagnosed as alcoholic liver disease and treated with *Shamana* and *Shodana Chikitsa*. The patient showed good improvement with normalization of appetite, bilateral lower back pain, abdominal pain, and yellowish discoloration of the sclera and urine. Lab investigations revealed a significant reduction in relief of symptoms, 83.5% improvement in total bilirubin, 48% improvement in AST, 29% improvement in ALT, and 8.4% improvement in ALP. In *Shodana karma, Nitya virechana* is adopted, and as a *Shamana* modality, *Nidana Parivarjana, Dosha Pratyanika*, and *Vyadhi Pratyanika Chikitsa* were adopted and showed a significant result in reducing signs and symptoms. It shows the effectiveness of the treatment method adopted.

INTRODUCTION

Alcohol abuse is a significant global health concern, with approximately 10–12% of Americans engaging in excessive alcohol consumption, making it the most commonly misused substance worldwide. Studies indicate that alcohol affects nearly 61% of the American population, while Europe reports the highest prevalence of Alcoholic Liver Disease (ALD). In contrast, India remains among the countries with the lowest alcohol consumption, with government statistics revealing that only 21% of adult men and approximately 2% of women consume alcohol.

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The liver possesses a remarkable regenerative capacity, but chronic alcohol consumption progressively impairs this ability. With each alcohol molecule metabolized, some liver cells perish. While the liver can regenerate new cells, prolonged alcohol abuse can significantly limit this process, leading to severe and often irreversible liver damage. ALD is the most common consequence of long-term alcohol-related liver injury, necessitating early identification, intervention, and adherence to evidence-based management strategies.[2]

The word *Yakrit*, based on the characteristic features it presents, can be correlated with the liver,[3] the largest of the abdominal viscera, which is an exocrine gland and an endocrine gland, and functions accordingly.[4] Based on the *Nidana, Samprapti*, and *Lakshana* of both diseases, alcoholic liver disease symptoms are consistent with *Yakritddalyudara*.[5]

Patient Information

A 50-year-old male presented to the *Kayachikitsa* outpatient department (OPD) of SDM Hospital, Udupi, with the following complaints. He was admitted on 08/09/2023 for further evaluation and management

Chief Complaints (Duration: 3 days)

- Yellowish discoloration of the sclera and urine
- Reduced appetite
- Abdominal distension
- Bilateral lower limb pain
- Abdominal pain

Associated Complaints

- Generalized weakness
- Sleep disturbances due to pain

Medical History

The patient has a normosthenic build and was well-nourished at the time of presentation. He is a known case of Type 2 Diabetes Mellitus and hypertension. His history of alcohol consumption is significant, as he had been consuming alcohol since the age of 22, with a recent abstinence period of six months.

Alcohol Consumption Pattern

- **Beer:** 5 pints/day (2841.31ml, 4–5% alcohol)
- **Brandy:** 1 quarter/day (180ml, 35–60% alcohol)

Clinical Course and Presenting Symptoms

One week before admission, the patient developed yellowish discoloration of the sclera and urine, along with abdominal distension and pain. The abdominal pain was stabbing in nature, continuous, and of moderate intensity (4/10 on the pain scale). He also experienced a significant reduction in appetite, which was exacerbated by abdominal distension.

Additionally, the patient complained of a dragging-type pain in both lower limbs for the past week. Six months earlier, he had an episode of abdominal pain and painful micturition, for which he sought medical attention. Due to the worsening of symptoms, he was admitted to our hospital for further evaluation and management.

Personal History

Table 1: Personal history

Bala	*Madyama*
Sleep	Disturbed
Addiction	• Alcohol (beer and brandy-60ml/ day) • Smoking (10 cigarettes/day) • Tobacco (3 times/day)
Bowel Habit	Regular & dark in color
Appetite	Reduced
Weight	68kg (08/09/23)

Clinical Findings- Physical Examination

Ashta Vidha Pareeksha

Table 2: *Ashta vidha pareeksha*

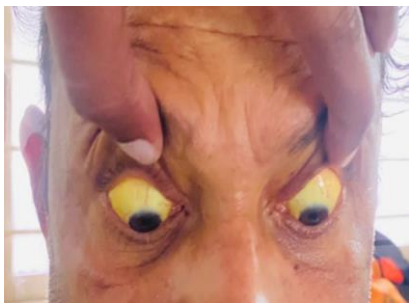
Nadi	*Pittaja nadi / 70 bpm*
Mutra	*Peeta mutra*
Mala	*Rakta varna*
Jiwha	*Lipta*
Shabda	*Prakrita*
Sparsa	*Anushna sheeta*
Druk	*Peeta varna*
Akruti	*Madhyama*

Clinical Findings

Table 3: *Dashavidha Pariksha*

Prakrti	*Pitta Vata*	
Vikrti	*Vata- Prana, Vyana, Apana, Samana*	
	Pitta- Pachaka, Ranjaka	
	Kapha- Kledaka, Avalambaka	
Sara	*Madhyama*	
Samhanana	*Madhyama*	
Pramana	*Madhyama*	
Satmya	*Madhyama*	
Satva	*Madhyama*	
Ahara Sakti	*Madhyama*	
Vyayama Sakti	*Avara*	
Vaya	*Madhyama*	

General Examination



- Pulse Rate: 70 bpm
- Respiratory Rate: 16 cycles/min
- Blood Pressure: 140/90mmHg
- Temperature: 98.2°F
- Built: Normal
- Nourishment: Fair
- Pallor: Absent
- Icterus: + in bulbar conjunctiva, palms, and soles
- Clubbing: Present (Grade 2)
- Edema: Pedal edema (pitting type)
- Lymphadenopathy: Absent

Systemic Examination

Cardiovascular System: S1 and S2 heard. No added sounds.

Respiratory System: Lungs are clear.

Central Nervous System: Conscious and oriented to time, place and person.

Memory: Intact

Romberg sign: Positive

Muscle power: 4/5 – All the limbs

Muscle tone: Normotonic

Gait- Ataxic gait

Involuntary movements- Action tremors

Reflexes: Normal

Gastrointestinal tract examination

Per abdominal examination

Inspection

- Abdomen: Globular
- Distension of Abdomen: Present
- Alcoholic facies: Absent

Palpation

- Local rise of temperature: Absent
- Guarding: Present in the right hypochondrium.
- Tenderness: Present in the Right hypochondrium.
- Palpable mass: Present in the left and right hypochondriac region in deep palpation.

Percussion

Percussion Note: Tympanic

Auscultation

Bowel Sounds: 3/minute

Investigations

Blood Investigation- Hb – 10.3 gm%

ESR- 150 mm U/hour (on 09/09/23)

Table 4: Liver Function Test (LFT) on 09/09/2023, with the following findings

Parameter	Result	Reference Range
Total Bilirubin	15.2 mg/dL	0.2 – 1.2 mg/dL
Direct Bilirubin	12.2 mg/dL	0.1 – 0.4 mg/dL
Indirect Bilirubin	3.0 mg/dL	0.2 – 0.8 mg/dL
Aspartate Aminotransferase (AST)	108.0 U/L	5 – 40 U/L
Alanine Aminotransferase (ALT)	62.0 U/L	7 – 56 U/L
Alkaline Phosphatase	261.0 U/L	44 – 147 U/L
Total Protein	6.0 g/dL	6.0 – 8.3 g/dL
Albumin	3.0 g/dL	3.5 – 5.2 g/dL
Globulin	3.0 g/dL	2.0 – 3.5 g/dL
A/G Ratio	1.0	1.2 – 2.2

USG Abdomen Findings on 09/09/23

Impression

- Mild hepatomegaly with grade I fatty infiltration and subtle surface irregularity along the left lobe – suggestive of parenchymal disease.
- Moderate splenomegaly
- Non-obstructive left renal calculi

Stool for Occult Blood- Positive (on 14/09/23)

Therapeutic Intervention

The patient was administered a structured treatment plan from 08/09/2023 to 18/09/2023, consisting of *Shodhana (Nityavirechana)* therapy and *Shamana Aushadhi* as per the *Chikitsa siddhanta* of Ayurveda.

***Shodana Chikitsa* (Detoxification Therapy)**

Nityavirechana with 10ml *Gandharvahastadi Eranda Taila*[5] daily from 08/09/2023 to 14/09/2023.

***Shamana Chikitsa* (Palliative Therapy)**

1. T. Nirocil 1-1-1 after food for 11 days
2. *Kalamegha syrup* 10ml TID after food for 11 days
3. *Punarnavadi kashaya*[6] 15ml TID after food for 11 days
4. T. *Gomutra haritaki*[7] 1-0-1 after food for 4 days

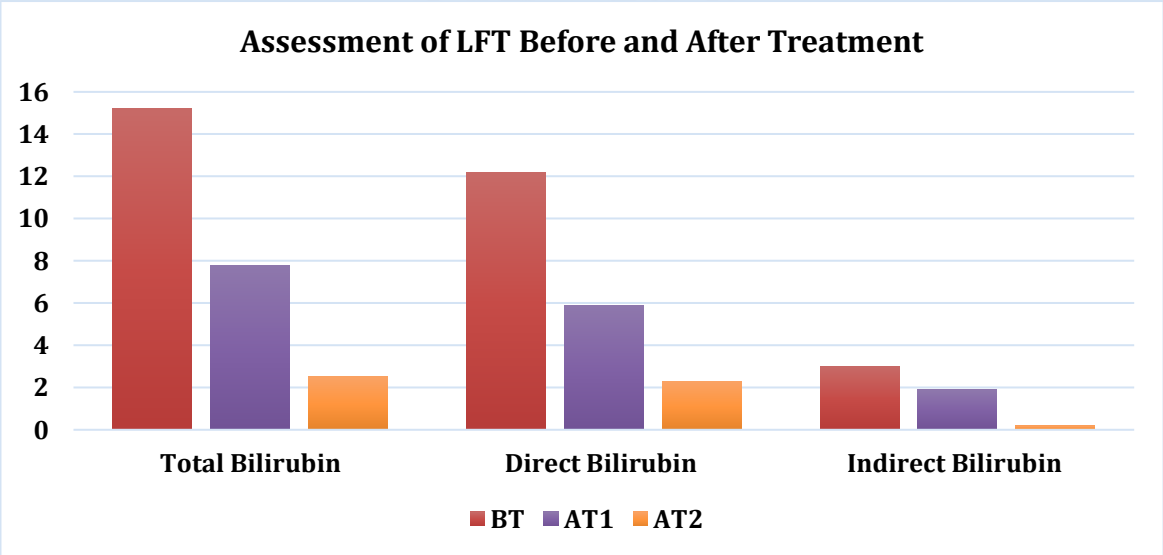
Therapeutic Timeline

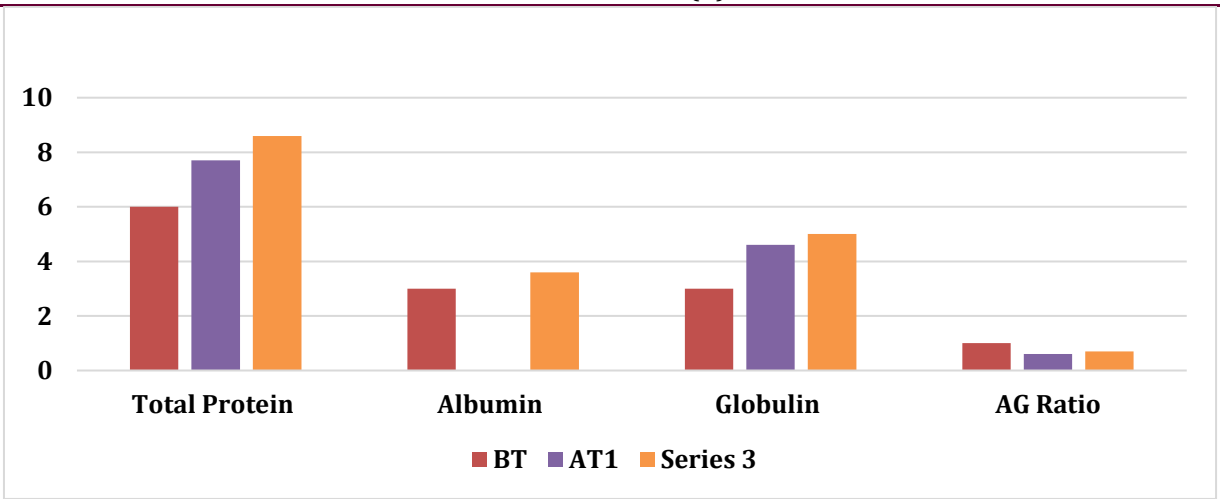
Table 5: Therapeutic Timeline

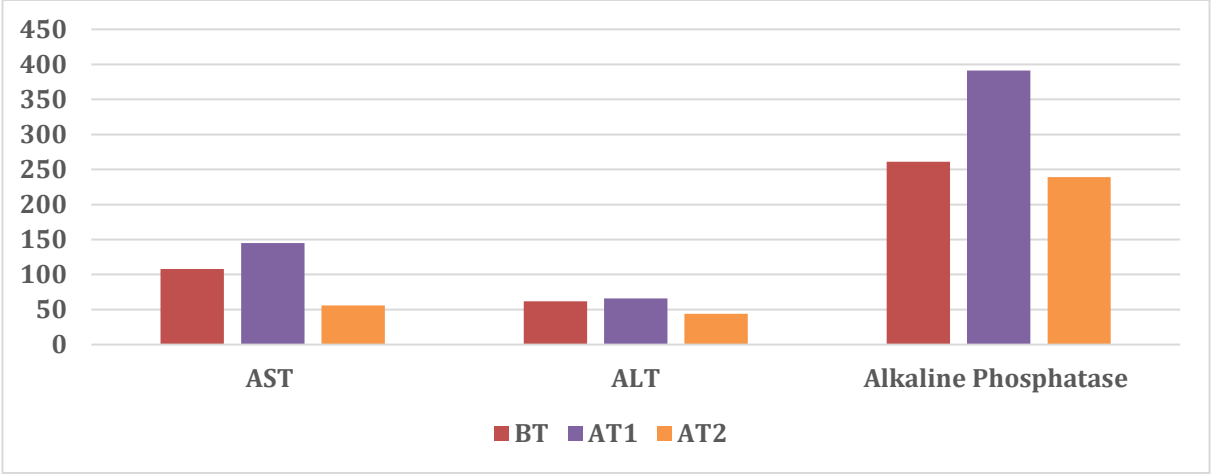
Date	Medications	Dosage & Frequency
09/09/23 - 14/09/23	1. T. Nirocil	1-1-1 (A/F)
	2. *Kalamegha* Syrup	10 ml TID (A/F)
	3. *Punarnavadi Kashaya*	15 ml TID (A/F)
15/09/23 - 18/09/23	1. T. Nirocil	1-1-1 (A/F)
	2. *Kalamegha* Syrup	10 ml TID (A/F)
	3. T. *Gomutra Haritaki*	1-0-1 (A/F)
	4. *Punarnavadi Kashaya*	15 ml TID (A/F)

Table 6: Assessment of LFT Before and After Treatment

LFT	09/09/23	12/09/23	18/09/23
Total Bilirubin	15.2 mg/dl	7.8mg/dl	2.5 mg/dl
Direct Bilirubin	12.2 mg/dl	5.9mg/dl	2.3 mg/dl
Indirect Bilirubin	3.0 mg/dl	1.9mg/dl	0.2 mg/dl
AST	108.0 U/L	145U/L	56.0 U/L
ALT	62.0 U/L	66.0 U/L	44.0 U/L
Alkaline Phosphatase	261.0 U/L	391U/L	239.0 U/L
Total Protein	6.0 g/dl	7.7g/dl	8.6 g/dl
Albumin	3.0 g/dl		3.6 g/dl
Globulin	3.0 g/dl	4.6g/dl	5.0 g/dl
AG Ratio	1.0	0.6	0.7







Follow-Up Medicines

Table 7: Follow-Up Medicines

S.No	Medicines	Dose
1	Tab Nirocil	1-1-1 A/F
2	*Kalamegha* Syrup	10ml TID A/F
3	*Punarnavadi Kashaya*	15ml TID B/F
4	Tab *Gomuthra Haritaki*	1-0-0 A/F
5	*Gandharvahastadi Eranda Taila*	10ml at 6 AM (Alternative Days)

DISCUSSION

Alcoholic liver disease (ALD) encompasses a wide range of conditions, including simple steatosis, cirrhosis, acute alcoholic hepatitis (AH) with or without cirrhosis, and hepatocellular carcinoma (HCC) as a potential complication of cirrhosis.

Mechanisms of alcoholic liver disease

Alcohol causes liver damage through several mechanisms. It is metabolized into acetaldehyde, which is toxic to liver cells (hepatocytes). Damaged hepatocytes release DAMPs, triggering immune cells and worsening liver injury. In the early stages, alcohol promotes fatty liver through its effects on lipogenesis and fatty acid oxidation. It also disrupts the gut microbiome and increases gut permeability, allowing bacterial products to reach the liver, further damaging it. Despite immune activation, the body's response becomes ineffective, leading to immune paralysis.[8]

Yakrutadalyodara, a form of *Udara Roga*, presents with symptoms such as weakness, loss of appetite, indigestion, retention of stool and urine, excessive thirst, malaise, vomiting, fainting, prostration, cough, difficulty breathing, mild fever, abdominal bloating, loss of digestive power, emaciation, a bad taste in the mouth, joint pain, and abdominal distension with colic pain.

Other notable signs include *Shyava Aruna Udara*, characterized by redness or discoloration of the abdomen, and the appearance of a network of veins with blue, green, or yellow coloration. These clinical features are commonly associated with liver enlargement *(Yakrdalyudara)* located on the right side of the abdomen. The etiology, signs, symptoms, and treatment of *Yakrutadalyodara* closely resemble those observed in *Plihodara* (splenic enlargement). Therefore, *Yakrutadalyodara* is often discussed alongside *Plihodara* due to their shared clinical characteristics

Alcohol consumption leads to vitiation of the three *Doshas- Vata, Pitta*, and *Kapha* and causes *Srotosanga* (blockage of circulatory channels), resulting in a condition classified as *Tridoshaja*, involving all three *Doshas* in its pathogenesis. Vitiated Kapha can be interpreted as a primary factor in the pathogenesis of hepatic steatosis, laying the groundwork for progressive liver damage and cirrhosis when left unaddressed. Clinical features such as ascites, oedema, and pruritis are indicative of *Kapha* involvement. *Vata Dosha* vitiation is associated with fibrotic changes, shrunken liver (atrophy of liver cells), genital atrophy, dyspnoea, and pain. On the other hand, *Pitta Dosha* vitiation is responsible for inflammatory changes, with recurrent hepatitis being another critical factor leading to liver cirrhosis. Symptoms like jaundice and hemorrhagic tendencies are hallmark indicators of active *Pitta* involvement**,** especially in the context of liver disorders such as cirrhosis. Although patients with Alcoholic Liver Disease (ALD) are primarily managed through strategies promoting abstinence from alcohol, alcohol itself acts as a toxin in higher doses. When combined with polyunsaturated fatty acids (PUFA), it induces oxidative stress and hepatotoxicity, further exacerbating liver damage.[9][9]

Alcoholic Liver Disease (ALD), particularly in the management of jaundice, hepatomegaly, and metabolic dysfunction through a structured approach combining *Shodhana Chikitsa* (detoxification therapy) and *Shamana Chikitsa* (palliative therapy).

Clinical Rationale & Pathophysiology

ALD, which results from chronic alcohol consumption, progressively impairs hepatic function and leads to hepatocellular damage, fibrosis, and cirrhosis. The patient's history of alcohol consumption for more than 25 years led to significant derangement of liver enzymes, hyperbilirubinemia, and hepatosplenomegaly. The treatment was tailored to restore liver function, promote hepatocyte regeneration, and remove metabolic toxins from the body.

From an Ayurveda perspective, the condition aligns with *Yakritdalyudara* (hepatic disease with hepatomegaly and ascitic tendencies), primarily involving *Pitta* and *Vata dosha* vitiation, along with derangement of *Ranjaka Pitta, Pachaka Pitta, Prana*, *Vyana, Apana*, and *Samana Vata*, and the accumulation of *Ama* (toxic metabolites).

Shodhana therapy *(Nityavirechana)* was initiated to remove *Pitta dosha* and cleanse hepatic channels *(Yakrit & Pleeha Srotas),* followed by *Shamana Chikitsa* to restore liver function and prevent fibrosis progression.

Therapeutic Response & Outcomes

The treatment approach led to marked improvements in both subjective and objective parameters, as evidenced by symptom resolution and normalization of LFT values. Key findings include: Bilirubin reduction: Total Bilirubin levels reduced from 15.2mg/dL to 2.5mg/dL, indicating significant resolution of cholestasis. Liver enzyme normalization: AST & ALT levels decreased from 108 U/L & 62 U/L to 56 U/L & 44 U/L, respectively, suggesting hepatocyte recovery. Nutritional improvement: Total protein levels increased from 6.0g/dL to 8.6g/dL, and albumin levels improved from 3.0g/dL to 3.6g/dL, reflecting better liver synthetic function. Symptomatic relief: Jaundice, abdominal pain, loss of appetite, and fatigue resolved within 10 days, demonstrating rapid response to therapy.

Pharmacological Analysis of Ayurvedic Medicines

The prescribed Ayurvedic formulations were carefully selected for their hepatoprotective, anti-inflammatory, and detoxifying properties. Below is a refined explanation of each formulation and its role in the treatment of liver conditions:

Gandharvahastadi Eranda Taila: Key ingredients such as *Gandharvahasta moola* (*Ricinus communis*), *Shunthi* (Zingiber officinale), and *Haritaki* (*Terminalia chebula*) exhibit properties like *Virechana* (purgative), *Ama-pachana* (detoxifying), *Vatanulomana* (regulating *Vata*), and *Yakrit-shodhana* (liver detoxification). This formulation induces *Nitya Virechana* to eliminate vitiated Pitta and alleviate liver congestion.

Nirocil Tablet: Which contains *Bhumyamalaki* (*Phyllanthus niruri*), is widely recognized in Ayurvedic and integrative medicine for its antiviral, hepatoprotective, and liver-rejuvenating properties. It acts as a *Pittahara* (*Pitta* pacifier), *Yakrit-uttejaka* (liver stimulant), and *Raktashodhaka* (blood purifier). It aids in hepatocyte regeneration, reduces bilirubin levels, and improves the liver enzyme profile. Studies by Raman Sharma et al. and Thyagrajan et al. highlight *Bhumyamalaki* as a promising treatment for Hepatitis

B due to its ability to reduce HBV DNA, oxidative stress, and hepatotoxicity. [10,11]

Kalamegha Syrup: This syrup combines *Kalamegha* (*Andrographis paniculata*), *Katuki* (*Picrorhiza kurroa*), *Punarnava* (*Boerhaavia diffusa*), and *Kakmachi* (*Solanum nigrum*). It functions as a *Yakrit-shodhaka* (liver detoxifier), *Pittashamaka* (reduces excess bile), and *Kledahara* (eliminates metabolic toxins). The formulation supports bilirubin metabolism, enhances liver function, and stimulates appetite.

***Punarnavadi Kashaya*:** A combination of herbs such as *Punarnava*, neem, *Patola, Nagara, Kiratatikta, Guduchi, Devadaru, Haridra,* and *Haritaki*, this formulation is acclaimed for its anti-inflammatory and diuretic properties. Its predominant *Tikta-Kashaya rasa* (bitter-astringent taste) pacifies *Pitta* and *Kapha* without aggravating *Vata*. Punarnava rejuvenates tissues, promotes cellular metabolism, breaks inflammatory pathways, and balances the *Tridoshas*.

Gomutra Haritaki is a classical Ayurvedic formulation with well-documented therapeutic actions, particularly beneficial in metabolic and hepatic disorders. It corrects *Agni*, regulates *Vata*, reduces hepatocellular inflammation, detoxifies liver and Supports lipid metabolism and combats liver issues. Its predominance of *Agni* and *Vayu Mahabhutas* supports liver detoxification and the removal of metabolic waste.

Comparison with Conventional Treatment

Conventional management of Alcoholic Liver Disease (ALD) typically involves supportive therapies such as ursodeoxycholic acid, antioxidants, vitamin supplements, and corticosteroids for severe cases. While these treatments are effective in managing symptoms, they do not directly address detoxification or metabolic correction. Ayurvedic formulations, through the incorporation of *Virechana* therapy and hepatoprotective herbs, not only enhance liver detoxification but also restore metabolic homeostasis. This comprehensive approach facilitates faster recovery while preventing long-term complications such as fibrosis.

CONCLUSION

This case demonstrates the potential of an integrated Ayurvedic approach, combining *Shodhana* and *Shamana* therapies, in effectively managing Alcoholic Liver Disease (ALD). The significant improvements observed in liver function parameters, symptomatic relief, and overall well-being highlight the therapeutic benefits of this approach. By addressing both detoxification and metabolic correction, Ayurvedic interventions offer a comprehensive strategy for managing ALD, potentially preventing long-term complications. Using this concept, treatment was carried out with the a forementioned medications, yielding remarkable symptomatic results in 11 days, including an 83.5% improvement in total bilirubin, 48% improvement in AST, 29% improvement in ALT, and 8.4% improvement in ALP While these results are promising, further well-designed clinical trials are warranted to validate these findings and establish the efficacy of Ayurvedic treatments in managing ALD and other complex liver disorders. Never forget that alcohol has an impact on our family, our finances, and our health.

Acknowledgement

We are thankful to the participant for providing informed consent to publish the results of the study.

Declaration of Patient Consent

Authors certify that they have obtained informed patient consent form, where the patient has given his consent for reporting the case along with the images and other clinical information in the journal. The patient/caregiver understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed

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