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AN AYURVEDIC APPROACH TO THE MANAGEMENT OF NON-CIRRHOTIC ALCOHOLIC STEATOHEPATITIS WITH HEPATOCELLULAR JAUNDICE: A CASE REPORT

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ABSTRACT

Alcoholic liver disease (ALD) is a progressive hepatic disorder caused by chronic alcohol consumption and ranges from steatosis to steatohepatitis and cirrhosis. In Ayurveda, alcohol-induced hepatocellular dysfunction can be correlated with *Kosthashrita Kamala* resulting from aggravated *Pitta* and *Rakta dushti*. Early-stage non-cirrhotic alcoholic steatohepatitis presents an opportunity for therapeutic reversal when managed appropriately. A 37-year-old male with a 12-year history of chronic alcohol intake, presented with yellowish discoloration of the sclera and urine, fatigue, anorexia, and nausea. The patient was managed with an Ayurvedic protocol of classical *Virechana Karma* followed by *Shamana Aushada Prayoga*. Post-treatment evaluation showed complete resolution of icterus and normalization of urine color with marked improvement in Liver function test. Ultrasonography demonstrated reduction in liver size from 18.4 cm to 16.0 cm with persistent mild fatty infiltration and no progression to cirrhosis. This case demonstrates Ayurvedic management, combined with appropriate dietary regulation, can produce significant clinical, biochemical, and radiological improvement in non-cirrhotic alcohol-induced liver disease. Early intervention at the steatohepatitis stage may prevent progression to irreversible hepatic damage. Further controlled studies are required to substantiate the

findings.

KEYWORDS: *Koshtashrita kamala,* alcoholic steatohepatitis, *Virechana, yakrit vikara.*

INTRODUCTION

The liver is an important organ in the human body that detoxifies various metabolites, synthesizes proteins, and produces biochemicals necessary for digestion. Liver diseases have become a major concern in clinical practice due to unhealthy lifestyles and chronic alcohol consumption. Alcoholic liver disease (ALD) is a spectrum of diseases caused by excessive alcohol intake. It may take the form of acute (alcoholic hepatitis) or chronic liver disease (steatosis, steatohepatitis, fibrosis and cirrhosis). The severity and prognosis of alcohol-induced liver disease depend on the amount, pattern and duration of alcohol consumption, as well as the diet, nutritional status and genetic predisposition of an individual.[1] While ALD is more commonly observed in developed Western countries, its prevalence is rising at an alarming rate in nations like India and Japan.[2] It is one of the leading causes of liver cirrhosis, accounting for approximately 20% to 50% of cases.[3]

Kamala is a disease according to Ayurveda, which encompasses the disorders of the Hepato-Biliary System in Modern Medicine. *Kamala* can manifest by excessive intake of *Pitta Vardhaka Ahara* and *Vihara*. Alcohol (*Madya*) possesses properties such as *ushna, tikshna, sukshma, ashu, vyavayi* and *vikasi* and this strongly aggravate *Pitta* leading to progressive vitiation of *Pitta* and *Rakta. Pitta Pradhana Tridoshas,* when elevated, end up in *Dhatu Shaithilya, Gourava, and Shithilendriyata* which further result in *Yakrit dushti* and manifestation of *haridra varna of netra, mutra* and *twak,* the cardinal features of *Kamala.* The treatment protocol mentioned for this disease in Ayurveda is mainly *virechana* along with various *tikta rasa pradana*, *pitta shamaka aushada prayoga*.[4,5,6]

The present case study describes the successful Ayurvedic management of a 37-year-old male patient diagnosed with *Kosthashrita Kamala* secondary to chronic alcohol consumption, corresponding to non-cirrhotic alcoholic steatohepatitis.

PATIENT INFORMATION

A 37-year-old male patient, residing in Bengaluru, presented with complaints of yellowish discoloration of eyes and urine (*haridra varna* of *netra* and *mutra*) associated with generalized body ache (*angasada*) since six months. The patient also reported fatigue

(*klama*), anorexia (*annadvesha*), nausea (*hrullasa*) and intermittent right upper abdominal pain (*udara shula*) for three months. The patient had a history of chronic alcohol consumption for 12 years, consuming approximately 650 mL of alcohol twice daily. He had attempted abstinence intermittently, during which he experienced tremors in both hands suggestive of alcohol withdrawal symptoms, leading to relapse of his habit. There was no history of diabetes mellitus, hypertension, thyroid disorders or abdominal distension suggestive of ascites.

FAMILY HISTORY: no relevant family history.

TREATMENT HISTORY

The patient had not received any treatment for liver disease prior to admission and was not on regular medication for any chronic illness.

PERSONAL HISTORY

- *Ahara*- Mixed diet
- *Nidra*- adequate (6-7 hours)
- *Jarana shakti- avara*
- *Koshta- Madyama koshta*

EXAMINATION

General Examination

The patient was of moderate build and well-nourished. Mild icterus was present. There was no pallor, cyanosis, clubbing, lymphadenopathy or pedal edema. Vital parameters were within normal limits, with a pulse rate of 75/min, respiratory rate of 18/min, temperature of 97 °F, and blood pressure of 130/80 mm Hg. Body mass index was 25.8 kg/m².

Systemic Examination

Gastrointestinal system

On inspection, the abdomen was soft with no distension and centrally placed umbilicus and no visible peristalsis or dilated veins. On palpation, mild tenderness was present in the right hypochondriac region without palpable organomegaly or mass. Percussion revealed dullness in the right hypochondrium. Bowel sounds were normal on auscultation.

Cardiovascular system

S1 and S2 heart sounds were normal with no added sounds.

Respiratory system

Bilateral normal vesicular breath sounds were heard without added sounds.

Central nervous system

The patient was conscious, alert and well oriented to time, place and person. Higher mental functions, cranial nerve examination and reflexes were within normal limits. No focal neurological deficit was noted.

Musculoskeletal system

Gait was normal with full range of movements in all limbs and no deformity or tenderness of the spine.

Ashtasthana Pareeksha

Nadi – Vata-pitta;

Mutra – Haridra varna, 3–4 times/day;

Mala – Loose, yellowish, 1–3 times/day;

Jihwa – Ishat lipta;

Shabda, Sparsha, Druk – Prakruta;

Akruti – Madhyama.

Dashavidha Pareeksha

Prakruti – Vata-pitta;

Vikruti – Pitta-pradhana tridosha;

Dushya – Rasa, Rakta;

Sara, Samhanana, Pramana, Sattva – Madhyama;

Ahara shakti – Avara;

Vyayama shakti – Madhyama;

Vaya – Madhyama.

INVESTIGATION

On 08/07/2025

Total bilirubin was 6.71 mg/dL with elevated direct bilirubin of 2.54 mg/dL and indirect bilirubin of 4.17 mg/dL. Urine examination showed dark yellow discoloration with the

presence of bile salts and bile pigments, supporting hepatic jaundice. Ultrasonography of the abdomen (done on 18/08/2025) demonstrated hepatomegaly without biliary obstruction, focal hepatic lesion or cirrhotic changes. The findings indicated diffuse hepatic involvement consistent with alcohol-related liver injury in a non-cirrhotic stage. These initial investigations confirmed the presence of hepatocellular jaundice secondary to alcohol-induced liver disease.

TREATMENT GIVEN

ON OPD VISIT: (15/07/2025) For a period of 15 days:

1. *Kumaryasava* 20ml BD (after food)
2. *Arogyavardini rasa 1-0-1* (after food)

Observation: Improved *agni*; *Klama* (fatigue) and *Hrullasa* (nausea) reduced comparatively.

ON IPD ADMISSION: (03/08/2025)

On admission, Classical *virechana* was planned.

(i) Shodananga Snehapana: (day1-3)

Snehapana with *kalyanaka ghrita* in *arohana matra- 30ml;60ml;100ml*

Advise: Ganji diet o*n Kshut pravrutti*

(ii) Vishrama kala (day 4-6)

Sarvanga Abhyanga with *Dhanwantara taila* followed by *Bashpa sweda*

Advise: diet: *Ganji,* Boiled Vegetables

(iii)*Virechana* (day 7)

Sarvanga Abhyanga with *Dhanwantara taila* followed by *Bashpa sweda*

Virechana with *Trivrut lehya* 70g with 100ml warm *ksheera*

(iv)*Samsarjana krama* (day 8-12)

Pathya was advised for 5 days with gradual dietary progression from *Manda* to *Peya* and *laghu Ahara* to restore *agni.*

OBSERVATION

- On the 3rd day of *snehapana, Samyak Snigdha lakshana–vatanulomana, Snigdha varchas, twak snigdhata (4 episodes of loose stools)* were observed.

- During *Vishrama kala,* the patient observed a significant reduction in *klama and angasada.*
- Total number of *virechana vega-12,* the patient went through the procedure well without complications.

DISCHARGE MEDICATION (09/08/2025)

(Post-*samsarjana krama,* for 10 days)

1. *Vasaguduchiyadi Kashaya 20ml-0-20ml* BD (before food)
2. *Arogyavardini rasa 1-0-1* (after food)

Observation: Significant reduction in *haridra netra* (icterus), *kampa*(tremors) and *klama* (generalised weakness).

***PATHYA* ADVISED**

Pathya

- ***Ahara***

Purana Shali (old rice), *Yava (*barley), *Goduma* (wheat), *Tikta-shaka*, bottle gourd, ridge gourd, ash gourd, *Dadima* (Pomegranate), *Amla, Takra(*buttermilk*), Sukhoshna jala (*luke warm water*).*

- ***Vihara***

Strict abstinence from alcohol, regular meals, adequate rest, Yoga, and pranayama.

Apathya

- ***Ahara***

Alcohol, spicy, sour, salty, fried, and oily foods, fermented and processed foods, red meat and heavy non-vegetarian diet, excess fat and junk foods, Curd (especially at night), Pickles, vinegar, bakery items, Refined sugar excess.

- ***Vihara***

Irregular sleep cycles, stress, suppression of natural urges, and over-exertion.

OUTCOME

1. SUBJECTIVE CRITERIA

Table 1: Outcome- Subjective criteria.

PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT
Icterus (sclera)	present	absent
Urine colour	Dark yellow	Pale yellow
Agni	*Manda*	Improved
Angasada	Present	absent

Figure 1: Before treatment.

Figure 2: After treatment.

2. OBJECTIVE CRITERIA

Table 2: Outcome- Laboratory findings.

LABORATORY PARAMETERS	BEFORE TREATMENT	AFTER TREATMENT	OUTCOME
Total Bilirubin	6.71 mg/dL	1.35 mg/dL	Marked reduction
Direct Bilirubin	2.54 mg/dL	0.79 mg/dL	Significant reduction
Indirect Bilirubin	4.17 mg/dL	0.56 mg/dL	Normalized
AST (SGOT)	56.6 U/L	28 U/L	Normalized
ALT (SGPT)	55.7 U/L	16.6 U/L	Normalized

Table 3: Outcome- Ultrasound imaging.

BEFORE TREATMENT (15/07/2025)	AFTER TREATMENT (18/08/2025)
Both lobes of liver are enlarged in size (measures 18.4cm). Shows diffuse increase in the echogenicity. Hepatomegaly with grade I fatty infiltrations.	Liver is enlarged in size (16.0 cm) and shows diffuse increase in parenchymal echogenicity. Hepatomegaly with mild fatty infiltration.

DISCUSSION

Alcoholic liver disease (ALD) represents a clinicopathological spectrum ranging from simple

steatosis to steatohepatitis, fibrosis, and cirrhosis, resulting from chronic and excessive alcohol consumption.[7] The present case was diagnosed as non-cirrhotic alcoholic steatohepatitis with hepatocellular jaundice, based on clinical presentation, biochemical parameters, and ultrasonographic findings. The absence of ascites, portal hypertension, cirrhotic morphology, or focal hepatic lesions indicated that the disease had not progressed to an advanced, irreversible stage.

The patient had a significant history of chronic alcohol intake for 12 years, consuming approximately 650 mL twice daily. Alcohol possesses *ushna, tikshna, sukshma, ashu, vyavayi* and *vikasi guna*, which contribute to rapid systemic dissemination and aggravation of *Pitta*.[8] Continuous intake leads to *Pitta prakopa* and *Rakta dushti*, ultimately affecting *Yakrit* (liver). This aligns with the Ayurvedic concept of *Kamala*, particularly *Kosthashrita Kamala*, where aggravated *Pitta* localizes in the gastrointestinal tract and hepatobiliary system, manifesting as *haridra varna* of *netra* and *mutra*. Clinically, the patient presented with classical features such as icterus, dark yellow urine, fatigue, anorexia, nausea, and right hypochondriac tenderness.

Considering the *Pitta-pradhana* pathology and *Yakrit dushti*, *Virechana* was selected as the principal *Shodhana* therapy. *Virechana* is indicated in *Kamala* for expulsion of vitiated Pitta from its principal seat.[9] Before *shodhana therapy*, appropriate *shamana aushadhi*, namely *Kumaryasava*[10] and *Arogyavardini Rasa*[11], were administered. These interventions, having hepatoprotective action, helped pacify the vitiated *Pitta Dosha*, while simultaneously enhancing *Agni* and *Bala*, thereby rendering the patient fit for the *Vyadhi-pratyanika shodhana therapy*, *Virechana*. *Shodhananga Snehapana* with *Kalyanaka Ghrita* which is indicated classically in *Kamala Roga*, ensured proper internal oleation and mobilization of *doshas*.[12] Following *Vishrama Kala* with *Abhyanga* and *Bashpa Sweda*, *Virechana* was administered using *Trivrut Lehya.*[13] The patient had 12 *vegas* without complications, indicating proper therapeutic purgation. *Samsarjana Krama* was implemented for five days to restore *Agni* gradually. Post-*Shodhana* management included *Vasaguduchiyadi Kashaya*[14] and *Arogyavardini Rasa* to maintain *Pitta shamana* and support hepatocellular recovery.

CONCLUSION

This case highlights that early-stage alcohol-induced liver disease corresponding to *Kosthashrita Kamala* can be effectively managed through appropriate *Shamana chikitsa* followed by classical *Shodhana* therapy and strict adherence to *pathya*. Early intervention at

the non-cirrhotic stage may prevent progression to irreversible liver damage. Through appropriate use of *shamana aushadi,* which helped pacify the vitiated *pitta dosha,* improve the *agni and bala,* rendering the patient fit for the *Shodana therapy*. Although limited to a single case, the findings indicate the potential role of classical Ayurvedic management in alcohol-induced hepatocellular disorders. Further well-designed clinical studies with larger sample sizes are warranted to validate these observations and establish evidence-based integrative protocols for alcoholic liver disease.

REFERENCES

1. Bruha R, Dvorak K, Petrtyl J. Alcoholic liver disease. World J Hepatol, Mar. 27, 2012; 4(3): 81-90. doi: 10.4254/wjh.v4.i3.81. PMID: 22489260; PMCID: PMC3321494.
2. Nesari M, Nangare N. Ayurvedic treatment of alcohol-induced liver disease: a case study. *South Eastern European Journal of Public Health (SEEJPH),* 2025; 26(S3).
3. Singh SP, Panigrahi S, Mishra D, Khatua CR. Alcohol-associated liver disease, not hepatitis B, is the major cause of cirrhosis in Asia. J Hepatol, 2019; 70: 1019-38.
4. Sharma P.V, Susruta Samhita Uttara Sthana. 44/6 with English Translation of text and Dalhana's commentary along with critical notes, Chaukamba VishvaBharathi oriental publishers and Distributors, Varanasi, 2004.
5. Astanghrdayam composed by Vagbhata, Nidana Stana13/19 with the commentaries Sarvangasundara of Arunadata and Ayurvedarasayana of Hemadri, Chaukhambha Orientalia, Varanasi.
6. Sharma R.K, Dash B., Charaka Samhita, Ciktsa Stana 16/36, Text With English Traslation 2 Critical Exposition Based On Cakrapani Datta's Ayureda Dipika, Chaukamba Vishvabhasati, Varanasi, 2007.
7. Hernandez-Tejero M, Clemente-Sanchez A, Bataller R. Spectrum, Screening, and Diagnosis of Alcohol-related Liver Disease. J Clin Exp Hepatol, Jan-Feb. 2023; 13(1): 75-87. doi: 10.1016/j.jceh.2022.10.002. Epub 2022 Oct 13. PMID: 36647416; PMCID: PMC9840079.
8. Agnivesha, Charak, Charak Samhita, Chikitsasthan 24/30 Hindi Commentary by Pandit Kashinath Shastri, Dr. Gorakhnath Chaturvedi, Chaukhambha Orientalia Publication Varanasi, 14th edition in, 1987; 671.
9. Agnivesha, Charak Samhita, Chikitsa Sthana 16/40, Chaukhamba Orientalia, Varanasi, Reprint edition, 2012; 441.

10. Krishna Sharma ed, Yogaratnâkara, Gulma chikitsa, Nirnaya Sagar Press, Bombay, 1929; 290.
11. Tripathi Indradev. Rasaratna Samuchchaya. 20/86-92, 3rd ed., Varanasi; Chaukhambha Sanskrit Bhawan, 2006; 252.
12. Agnivesha, Charak Samhita, Chikitsa Sthana 16/43, Chaukhamba Orientalia, Varanasi, Reprint edition., 2012; 441.
13. Agnivesha, Charaka Samhita, Dridhabala. In: Charaka Samhita, Kalpa Sthana, Shyama Trivrut kalpa Adhyaya, Chapter 7, Verse 6 Vaidya Yadavji Trikamji Acharya, editor. Varanasi: Chaukhamba Surbharti Prakashan, 2011; 32.
14. Translator Prof Sreekanthamurthy K.R. Astanga Hridaya. Varanasi: Chaukamba Krishnadas Acadamey, 2014; 449. (Krishnadas Ayurveda Series, 2.