RESEARCH PAPER

Ayurvedic management of HBV-Associated Chronic Liver Parenchymal Disease with Ascites: A Case Report

Dr. Malhar Nesari[1*], Dr. Ninad Nangare[2]

[1*]Medhamrut Ayurved Chikitsalay, Pune (411038), Maharashtra, India

[2]Bharati Vidyapeeth (Deemed to be University) College of Ayurved, Pune (411046), India

Corresponding author: Dr. Malhar Nesari, Medhamrut Ayurved Chikitsalay, Pune (411038), Maharashtra, India

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ABSTRACT

Background: Chronic Liver Parenchymal Disease (CLPD) is a progressive hepatic condition damaging the functional parenchyma, often due to viral hepatitis (HBV), metabolic disorders, or toxins, leading to fibrosis, cirrhosis, ascites, and complications like portal hypertension.

CASE PRESENTATION: A 61-year-old female patient presented in May 2025 with body weight 59 kg, moderate ascites, pain in the right hypochondriac and epigastric regions, anorexia, and generalized weakness for two months. History included diabetes and HBsAg-positive chronic HBV. Clinical findings: palpable splenomegaly, ascites; USG abdomen showed coarse liver echotexture, borderline splenomegaly, moderate ascites; initial LFTs: bilirubin 1.31 mg/dL, SGOT (AST) 57 U/L, SGPT (ALT) 39 U/L. Diagnosed as CLPD.

Intervention: A four-month herbo-mineral Ayurvedic regimen: May-June - Trailokya Chintamani Rasa, Tamra Bhasma, Pippali, Haritaki with detox diet; July (during HBV flare with bilirubin spike to 9.12 mg/dL) - Guduchi, Sootshekhar Rasa, Triphala, Nimb, Sariva, Manjishtha, Bawachi; August - Phalatrikadi Guggulu, Gandharva Haritaki, Guduchi, Loha Bhasma. Monthly LFT/USG abdomen monitoring.

Outcome: Complete resolution of ascites, pain, anorexia; 3 kg weight loss. LFT normalization by August 30: bilirubin 0.81 mg/dL, SGOT (AST) 26.3 U/L, SGPT (ALT) 34 U/L. Follow-up USG abdomen showed mildly coarse liver echotexture with reduced ascites.

Conclusion: This case illustrates Ayurveda's potential to reverse CLPD through Yakrit-shodhana, Pitta-shamaka, and Rasayana therapies, even in HBV context with acute flares. Demonstrates safety and efficacy as integrative approach; warrants larger clinical trials.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and clinical details.

Conflict of Interest: The authors declare no conflict of interest.

Keywords- Ayurveda, Ascites, Chronic Liver Disease, Hepatitis B, Yakrut Vikara

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Introduction

Liver Parenchymal Disease (LPD) indicates a range of liver conditions that can affect its functional tissue, known as Parenchyma. LPD is also known as chronic liver disease. This liver parenchymal disease can lead to diffuse (widespread) changes, causing mild to severe infections, autoimmune diseases, genetic disorders, and more. [1]

The spectrum of etiologies is broad for chronic liver disease, which includes toxins, alcohol abuse for a prolonged period, infection, autoimmune diseases, genetic and metabolic disorders. Cirrhosis is a final stage of chronic liver disease that results in disruption of liver architecture, the formation of widespread nodules, vascular reorganization, neo-angiogenesis, and deposition of an extracellular matrix. The underlying mechanism of fibrosis and cirrhosis at a cellular level is the recruitment of stellate cells and fibroblasts, resulting in fibrosis, while parenchymal regeneration relies on hepatic stem cells. [2]

Chronic infection with Hepatitis B Virus (HBV) and Hepatitis C Virus (HCV) remains one of the leading causes of CLPD worldwide. Persistent viral replication induces continuous inflammation, hepatocyte necrosis, and fibrogenesis, which over time leads to cirrhosis and hepatocellular carcinoma. [3]

Metabolic Dysfunction-associated Fatty Liver Disease has become the fastest growing cause of chronic liver disease globally. It is strongly linked with obesity, diabetes mellitus, insulin resistance, and dyslipidemia [4,5]

From an Ayurvedic perspective, such chronic liver involvement is understood under the broad framework of Yakrit Vikara, where vitiation of Pitta at the hepatic site, impairment of Agni (digestive and tissue-transforming fire), and obstruction of Rasa–Rakta carrying channels (Srotorodha) culminate in conditions described as Udara Roga (including Jalodara), Kumbhakamala, and

Pleehodara. Classical texts emphasize that when the pathological process is addressed at the level of Agni and Srotas, there remains potential for functional restoration, particularly through Yakrut-shodhana (liver cleansing), Pitta-shamana (pacification of deranged bile), and Rasayana (tissue regeneration) measures. While contemporary biomedicine focuses primarily on etiologic control such as antivirals for HBV or lifestyle and metabolic correction in MAFLD—these do not always achieve structural reversal of fibrosis. [6]

Case Report

Patient information: A 61-year-old female patient (weight:59 kg) came with complaints of moderate abdominal distension due to ascites, pain in the right hypochondriac and epigastric regions, loss of appetite, and generalized weakness persisting for approximately two months. Associated complaints included fatigue and yellowish discoloration of urine. At the time of admission in May 2025 at Medhamrut Ayurvedic Clinic, the patient was conscious and cooperative with normal vital functions (BP 130/70 mmHg, pulse 80 bpm). The patient had a known history of diabetes mellitus and tested positive for HBsAg (chronic HBV infection), with no history of hypertension or any surgical intervention. She had no relevant family history.

Clinical Findings

General examination showed the patient was moderately nourished with average build (BMI 24.3 kg/m²), Vitals were stable, with blood pressure 130/70 mmHg, pulse 80 bpm. On systemic examination, cardiovascular system (CVS), respiratory system, and central nervous system (CNS) were within normal limits.

Per-abdomen examination

Inspection: Abdomen showed moderate uniform distension with fullness in all quadrants and prominent flanks suggestive of intra-abdominal free fluid (ascites). Umbilicus was centrally placed but slightly everted. No visible dilated veins, scars, or skin discoloration noted. Respiratory movements of abdomen were reduced but present.

Palpation: Abdomen was soft with mild distension and shifting dullness confirmed. Liver was not palpable; spleen was just palpable below costal margin (borderline splenomegaly). Fluid thrill was distinctly positive confirming moderate ascites. No guarding, rigidity, tenderness, or palpable masses detected. Temperature over abdomen was normal.

Investigations and Diagnostic Assessment

1. Ultrasonography Abdomen (March 24, 2025) Abdominal sonography revealed coarse echotexture of liver suggestive of chronic parenchymal disease, thickened gallbladder wall, borderline splenomegaly (spleen just palpable below costal margin), and moderate ascites in all quadrants confirming peritoneal fluid accumulation. No evidence of portal vein thrombosis, hepatic focal lesions, or gross cirrhosis was noted.
2. Liver Function Tests (April 15, 2025)
Total bilirubin: 1.31 mg/dL (Direct: 0.74 mg/dL) - Mild elevation
SGOT (AST): 57 U/L ↑ (Normal: <40 U/L)
SGPT (ALT): 39 U/L ↑ (Normal: <40 U/L)
The Pattern was consistent with hepatocellular injury with mild cholestatic component.
3. Serological Markers
HBsAg: Reactive (chronic HBV infection confirmed)
Diabetes mellitus (managed on oral hypoglycemic).

Diagnostic Assessment

Tridosha Pariksha: Predominant Pitta prakopa at Yakrit sthana with Kapha avarana manifesting as Agni mandya, Rasa dhatu sanga, and Udakavaha srotorodha
Nadi Pariksha: Pitta anubandhi kapha nadi
Jihva Pariksha: Coated tongue with yellowish tint
Dhatu Pariksha: Rasa dhatu vriddhi with Rakta-Mamsa dhatu kshaya
Srotas Pariksha: Udakavaha, Rasavaha srotas atipravritti with Annavaha srotas sanga
Final Ayurvedic Diagnosis: Yakrit Vikara with Jalodara and Pleehodara.

Table 1. Investigations and Outcomes

Date	**Total Bilirubin (mg/dL)**	**Direct Bilirubin (mg/dL)**	**SGOT** (AST) **(U/L)**	**SGPT** (ALT) **(U/L)**	**Ultrasound (USG** abdomen**) Findings**
March 24, 2025	—	—	—	—	Coarse liver, thickened GB, borderline splenomegaly, moderate ascites.
April 15, 2025	1.31	0.74	57	39	—
June 7, 2025	1.36	0.68	52	58	—
July 8, 2025	9.12	7.3	51.1	60.3	—
August 1, 2025	1.39	0.37	47.2	39	Mildly coarse liver, mild splenomegaly, right renal calculus.
August 30, 2025	0.81	0.18	26.3	34	Reduction in Ascites (Clinical/USG abdomen)

Therapeutic Intervention and Timeline

Initial diagnostic imaging via ultrasonography on March 24, 2025, revealed a liver with coarse echotexture, thickened gallbladder, borderline splenomegaly, and moderate ascites. Subsequent blood work on April 15, 2025 showed elevated bilirubin (1.31 mg/dL) and liver enzymes (SGOT (AST) 57 U/L, SGPT (ALT) 39 U/L), leading to diagnosis of Yakrut Vikara (liver disorder) [7,8]. Initial treatment from May 2025 comprised oral Trailokya Chintamani [9,10], Tamra Bhasma [11,12] Pippali [13,14], and Haritaki [15] with strict liver detoxification diet and monthly hepatic monitoring.

By June 7, 2025, continued medications yielded 3.0 kg weight reduction indicating metabolic improvement and fluid reduction. Treatment adjustment on July 8, 2025 addressed hyperbilirubinemia spike (9.12 mg/dL) using Guduchi Tablet [16], Sootshekhar Rasa, Triphala powder, Nimba powder, Sariva, Manjishtha and Bakuchi, successfully stabilizing condition as HSV symptoms resolved.

August 2025 protocol shifted to Phalatrikadi Guggul, Gandharva Haritaki powder, and Haritaki powder. Ultrasound on August 1, 2025 confirmed progress with mildly coarse liver echotexture, mild splenomegaly, and reduced ascites. Protocol refinement by end of August included Guduchi powder and Loha Bhasma. Final pathology reports on August 30, 2025 showed normalized LFTs (bilirubin 0.81 mg/dL, SGOT(AST) 26.3 U/L, SGPT 34 U/L), normal creatinine, and significant ascites resolution with Phalatrikadi Guggul and Guduchi continuation.

Table 2. Therapeutic Intervention Timeline, Dosage, and Clinical Outcomes

Date/Phase	Investigations	Intervention	Dosage	Frequency	Clinical Outcome
Mar 24, 2025	USG abdomen: Coarse liver, moderate ascites	Diagnosis: Yakrut Vikara	-	-	-
Apr 15, 2025	LFT:Bilirubin1.31mg/dL,SGOT (AST) 57 U/L, SGPT 39 U/L	Treatment initiation	-	-	-
May-Jun 2025	Monthly monitoring	Trailokya Chintamani	125mg	OD	3.0 kg weight loss (Original weight – 59 kg)
		Tamra Bhasma	15mg	BD	Metabolic improvement
		Pippali	250mg	BD	Fluid reduction
		Haritaki	1000mg	BD	LFT: Bilirubin 1.36 mg/dL
Jun 7, 2025	LFT: SGOT(AST) 52 U/L, SGPT (ALT) 58 U/L	Continued initial regimen	-	-	Clinical improvement
Jul 8, 2025	LFT: Bilirubin 9.12mg/dL (HSV flare)	Guduchi Tablet	250mg	BD	HSV symptoms resolved
		Sootshekhar Rasa	500mg	BD	Hepatic stabilization
		Triphala powder	1000mg	BD	-
		Nimba powder	500mg	BD	-
		Sariva	1000mg	BD	-
		Manjishtha	1000mg	BD	-
		Bakuchi	500mg	BD	-
Aug 1, 2025	USG abdomen: Mildly coarse liver, reduced ascites	Phalatrikadi Guggul	1000mg	BD	USG improvement

		Gandharva Haritaki	1500mg	HS	Reduced splenomegaly
		Haritaki powder	500mg	BD	-
End Aug	-	Guduchi powder	500mg	BD	-
		Loha Bhasma	125mg	BD	-
Aug 30, 2025	LFT: Bilirubin 0.81mg/dL, SGOT(AST) 26.3 U/L, SGPT (ALT) 34 U/L	Discharge: Phalatrikadi Guggul + Guduchi	Continuatio	Monthly monitorin g	Complete resolution

Follow-up and Outcome

June 7, 2025 (1-month follow-up): Patient continued initial herbo-mineral regimen (Trailokya Chintamani, Tamra Bhasma, Pippali, Haritaki) with strict detoxification diet. Demonstrated 3.0 kg body weight reduction indicating improved metabolism and reduced fluid retention. LFTs showed Total bilirubin 1.36 mg/dL, SGOT (AST) 52 U/L, SGPT(ALT) 58 U/L with clinical ascites improvement.

July 8, 2025 (2-month follow-up, HSV flare): Acute hyperbilirubinemia spike to Total bilirubin 9.12 mg/dL (Direct 7.3 mg/dL) during Herpes simplex virus infection. Treatment adjusted to Guduchi Tablet, Sootshekhar Rasa, Triphala powder, Nimb, Sariva, Manjishtha, and Bakuchi. HSV symptoms completely resolved with stabilization of hepatic functions.

August 1, 2025 (3-month USG abdomen assessment): Ultrasonography revealed progressive improvement - mildly coarse liver echotexture (vs baseline coarse), mild splenomegaly (vs borderline), significant ascites reduction, and incidental non-obstructive right renal calculus. Treatment continued with Phalatrikadi Guggul, Gandharva Haritaki, Haritaki.

Discussion

The clinical management of Chronic Liver Parenchymal Disease (CLPD) aims to halt fibrosis progression and control complications like ascites and portal hypertension. In this case, the patient's condition diagnosed as Yakrut Vikara in Ayurveda demonstrated remarkable symptomatic and biochemical reversal through a four-month targeted herbo-mineral regimen.

The treatment strategy centered on liver detoxification (Yakrut-shodhana) and cellular rejuvenation (Rasayana). Haritaki and Guduchi provided hepato protection and regeneration by safeguarding parenchymal cells and preventing fibrous changes critical for reversing CLPD's architectural disruption. Guduchi specifically demonstrated its potency during the hyperbilirubinemia flare (9.12 mg/dL), rapidly restoring bilirubin to 0.81 mg/dL.

Tamra Bhasma and Sootshekhar Rasa regulated Yakrut Pitta (bile secretion) while enhancing digestive fire (Agni), improving nutrient assimilation and metabolic function evident in the patient's 3.0 kg weight stabilization and abdominal girth reduction. Trailokya Chintamani and Phalatrikadi Guggul acted as potent anti-inflammatory (Shothahara) and hepatostimulant agents, addressing portal congestion responsible for moderate ascites resolution.A distinctive feature was the patient's resilience during acute Herpes simplex infection in July 2025. Guduchi, Sariva, and Manjishtha supported liver function through this hepatic stressor, preventing decompensation and achieving rapid bilirubin normalization by August. Complete SGOT(AST) /SGPT (ALT) normalization (57→26.3 U/L, 39→34 U/L) indicates effective hepatocyte repair and functional parenchyma restoration.

Trailokya Chintamani serves as Rasayana for ascites, splenomegaly, and Yakrit-Pleehodara. Tamra Bhasma stimulates Yakrut Pitta circulation, corrects Agnimandya, and shows hepatoprotection in toxicity models, indicated for hepatomegaly and ascites. Haritaki provides hepatoprotective regeneration beyond laxative effects. Sootshekhar Rasa pacifies Pitta pathology, normalizing bile secretion and Agni. Phalatrikadi Guggul acts as Yakrut-uttejak (hepatostimulant), Shothahara, and Rasayana promoting hepatocellular regeneration. Guduchi prevents fibrosis and normalizes deranged liver functions.While modern HBsAg management focuses on viral suppression, this Ayurvedic protocol demonstrates adjunctive potential for structural recovery. Yakrut-shodhana and Rasayana therapies warrant validation through larger prospective trials as integrative CLPD management.

Chronic liver disease associated with hepatitis B virus infection remains a major cause of hepatic morbidity worldwide. Persistent viral infection leads to chronic hepatocellular injury, inflammation, fibrosis, and complications such as ascites and splenomegaly. In the present case, the patient presented with moderate ascites, hepatocellular enzyme elevation, and ultrasonographic findings suggestive of chronic liver parenchymal disease.

In Ayurveda, such clinical manifestations may be correlated with Yakrit vikara and Udara roga, particularly conditions resembling Jalodara, where pathological accumulation of fluid occurs due to impairment of digestive and metabolic processes. The underlying pathology may be explained through Pitta dushti at Yakrit sthana along with disturbance of Rasa and Rakta dhatu metabolism, leading to fluid accumulation and abdominal distension.

The Ayurvedic therapeutic approach aims at correcting Agni dysfunction, removing accumulated Dosha, and restoring normal liver function. In the present case, the

selected herbo-mineral formulations were intended to improve hepatic metabolism, reduce inflammatory processes, and support fluid regulation.

Gradual clinical improvement was observed during the course of therapy, with reduction in abdominal distension, improvement in appetite, and reduction in ascitic fluid as evidenced by follow-up ultrasonography. Laboratory parameters also showed stabilization of liver enzymes and bilirubin levels. These findings suggest that Ayurvedic interventions may contribute to symptomatic improvement and functional support of hepatic physiology.

Similar observations have been reported in previous studies evaluating Ayurvedic formulations in liver disorders, where hepatoprotective and antioxidant properties of herbal ingredients contributed to improvement in liver function parameters and overall clinical condition.

However, the present report represents a single clinical observation, and further controlled studies with larger sample sizes are required to establish efficacy and mechanisms more conclusively.

The therapeutic effects observed in the present case may be attributed to the pharmacological actions of the herbal components present in the prescribed Ayurvedic formulations. Many Ayurvedic medicinal plants used in liver disorders possess hepatoprotective, antioxidant, anti-inflammatory, and immunomodulatory properties. These properties help in protecting hepatocytes from oxidative stress, reducing inflammatory injury, and improving metabolic functions of the liver.

Experimental studies have demonstrated that several herbal constituents commonly used in Ayurvedic hepatoprotective formulations can modulate hepatic enzyme activity, enhance bile secretion, and support detoxification pathways. Antioxidant compounds present in these herbs may reduce lipid peroxidation and oxidative damage in hepatic tissue, thereby contributing to stabilization of liver function.

In addition, certain herbal components exhibit diuretic and mild laxative actions, which may help in reducing fluid retention and abdominal distension associated with ascites. Through these combined mechanisms, Ayurvedic therapy may contribute to symptomatic relief and functional improvement in patients with chronic liver disease.

Limitations

The present report represents a single case observation; therefore, the findings cannot be generalized to a larger population. Advanced fibrosis assessment tools such as elastography or liver biopsy were not performed, which limits the ability to objectively evaluate structural reversal of liver damage. Additionally, long-term follow-up beyond the reported duration would be necessary to assess sustained therapeutic benefits. Further controlled clinical studies are required to validate the effectiveness and mechanisms of Ayurvedic interventions in chronic liver disease associated with hepatitis B infection.

Conclusion

The present case demonstrates that Ayurvedic therapeutic intervention was associated with clinical improvement in a patient with HBV-associated chronic liver disease presenting with ascites. Symptomatic relief, improvement in appetite and weakness, and reduction in ascitic fluid were observed during the course of treatment. While the findings suggest a potential supportive role of Ayurvedic therapy in the management of chronic liver disease, further systematic clinical studies are required to establish its efficacy and safety.

Patient Perspective

The patient reported severe discomfort due to moderate abdominal distension (ascites), right hypochondriac/epigastric pain, loss of appetite, generalized weakness, fatigue, and restricted mobility before treatment. During the treatment, she experienced gradual reduction in abdominal girth, pain relief, improved appetite, and increased energy levels. By the end of four-month treatment, she returned to normal daily activities with complete symptom resolution and improved quality of life. The patient acknowledged the efficacy of the Ayurvedic herbo-mineral regimen, compassionate care, and remarkable improvement observed throughout the treatment course at Medhamrut Ayurvedic Clinic.

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