Research Article

Ayurvedic Management Of Udar (Refractory Ascites) Secondary To Alcoholic Liver Cirrhosis With Umbilical Hernia: A Case Report

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Abstract

Background: Jalodara (ascites), a severe presentation of Udar Roga, corresponds closely to refractory ascites secondary to decompensated liver cirrhosis. Conventional medical management relies heavily on long-term diuretics and recurrent therapeutic paracentesis. This case report documents the complete resolution of paracentesis dependency and significant clinical improvement in a geriatric patient using conservative Ayurvedic management (Shamana Chikitsa) and strict dietary intervention (Pathya Ahara).
Case Presentation: A 66-year-old male with a history of chronic alcoholism presented with severe abdominal distension (abdominal girth: 110 cm), generalized weakness, mild pedal edema, constipation, and anorexia. He was a known case of alcoholic liver cirrhosis, Type 2 Diabetes Mellitus, Hypertension, Hypothyroidism and a secondary umbilical hernia. The patient required abdominal paracentesis every 10 days prior to presentation. Baseline laboratory evaluations revealed anemia (Hb: 7.9 g/dL) and renal impairment (Serum Creatinine: 1.93 mg/dL, eGFR: 42.24 mL/min/1.73 m²).
Interventions: Following one initial therapeutic paracentesis for emergency decompression, the patient was managed purely with oral conservative Ayurvedic formulations: Arogyavardhini Vati, Punarnava Mandoor, Chitrakadi Vati, Gokshuradi Guggulu, and a custom Churna Yoga (Ashwagandha, Punarnava, Musta, Kutaki, Haritaki, and Shankh Bhasma). The intervention was coupled with a salt-free diet consisting of cow's milk (Godugdha), pearl millet flatbread (Bajari Bhakri), and garlic (Lashuna) chutney.
Outcomes: Over a 5-month follow-up, the requirement for abdominal paracentesis was completely eliminated. Constipation and anorexia resolved within 15 to 30 days. Hemoglobin levels increased from 7.9 g/dL to 10.6 g/dL, Serum Creatinine reduced from 1.93mg/dL to 1.6 mg/dL, and pedal edema resolved fully without any adverse events.
Conclusion: Targeted Ayurvedic oral therapy (Shamana) combined with strict adherence to Pathya can break the disease pathogenesis (Samprapti Vighatana), manage fluid dynamics, and eliminate paracentesis dependency in high-risk, co-morbid cases of refractory ascites.

Keywords: Udar, Ascites, Alcoholic Liver Cirrhosis, Shamana Chikitsa, Case Report.

1. Introduction

Udar Roga encompasses a group of severe abdominal disorders described in classical Ayurvedic texts, among which Jalodara [6] represents the stage of fluid accumulation within the peritoneal cavity. In modern clinical practice, Jalodara correlates directly with ascites, most commonly secondary to decompensated liver cirrhosis resulting from chronic alcohol consumption (Madya Sevana).
Advanced stages often lead to refractory ascites, characterized by rapid fluid accumulation that responds poorly to standard diuretic therapy, necessitating frequent abdominal paracentesis. Recurrent paracentesis carries significant risks of infection, electrolyte imbalances, circulatory dysfunction, and progressive protein depletion.
While classical Ayurvedic management of Udar Roga often advocates aggressive purgation therapies (Nitya Virechana), such procedures carry high risks in elderly, debilitated patients with multiple co-morbidities like Diabetes Mellitus, Hypertension, and impaired renal function. This case report illustrates the successful management of refractory ascites using conservative oral palliative therapy (Shamana Chikitsa) and strict dietary protocols (Pathya), adhering strictly to the CARE (CAse REports) guidelines.

2. Patient Information

2.1 General Demographics

- **Age:** 66 years
- **Sex:** Male
- **Ethnicity:** Indian

• **Occupation:** Desk job (Employed)
• **Socioeconomic Status:** Middle class

2.2 Chief Complaints & Duration

The primary presenting symptoms and their clinical significance are outlined in Table 1 below:

Complaint	Onset / Duration	Clinical Significance	
Abdominal distension (Kukshi Vriddhi)	5 years	Primary feature of Jalodara (Fluid retention)	
Anorexia (Aruchi)	4 years	Severe Agnimandya and Ama accumulation	
General weakness (Daurbalya)	3 years	Dhatu Kshaya (Depletion of Rasa, Mamsa, and Ojas)	
Constipation (Malavashtambha)	Chronic / Ongoing	Apana Vayu dysfunction & Purishavaha Sroto-rodha	
Bilateral pedal edema (Pada Shopha)	2 years (Mild)	Systemic fluid retention (Kaphaja-Pittaja involvement)	
Reduced urine output (Alpa Mutrata)	2 years	Impairment of Udakavaha and Mutravaha Srotas	
Exertional dyspnea (Kshudraswasa)	Intermittent	Mechanical compression of diaphragm due to Udar Vriddhi	

2.3 History of Present Illness (HPI)

The patient was reportedly in stable health until five years ago when he gradually developed progressive abdominal distension secondary to alcoholic liver cirrhosis. The fluid accumulation required therapeutic paracentesis every two months initially.

Over the past year, sustained high intra-abdominal pressure caused an umbilical hernia protrusion. Due to decompensated liver disease, portal hypertension, and associated co-morbidities, surgical correction of the hernia was deemed high-risk and contraindicated. The patient's condition progressively deteriorated, increasing the paracentesis frequency to once every 10 days. Due to persistent exhaustion, severe loss of appetite, and high procedure dependency, he sought conservative Ayurvedic care.

2.4 Past Medical, Personal & Medication History

• **Systemic Illnesses:** Diagnosed with Alcoholic Liver Cirrhosis (complicated by portal hypertension and ascites). Known case of Type 2 Diabetes Mellitus, Essential Hypertension, and Hypothyroidism for 20 years.

• **Surgical / Interventional History:** Recurrent abdominal paracentesis for 5 years (frequency escalated to every 10 days). Secondary umbilical hernia protrusion.

• **Medication History:** Subcutaneous Insulin for diabetes, Antihypertensive therapy, and Levothyroxine for hypothyroidism. No long-term history of NSAID or steroid use.

• **Substance History:** Chronic alcohol intake since age 25 (primary etiological factor/Hetu for Yakriddalyudara / Jalodara).

• **Lifestyle (Vihara):** No daytime sleep (Divasvapna). Maintained a daily routine of walking approximately 3 km.

3. Clinical and Diagnostic Findings

3.1 Ayurvedic Diagnostic Assessment (Ashtavidha Pariksha)

The baseline Ayurvedic physical assessment is summarized in Table 2:

Examination (Pariksha)	Clinical Observation	Diagnostic Inference
1. Nadi (Pulse)	Pitta-pradhana Vataanubandhi	Primary Pitta aggravation associated with Vata morbidity
2. Mutra (Urine)	Decreased output (Alpa Mutrata)	Dysfunction of Udakavaha and Mutravaha Srotas

3. Mala (Stool)	Constipation (Malavashtambha)	Apana Vayu obstruction (Vatasaṅga) & Purishavaha Sroto-rodha
4. Jihva (Tongue)	Coated (Sama)	Presence of Ama (metabolic endotoxins) and Agnimandya
5. Shabda (Voice)	Normal (Prakrita)	Unimpaired Udana Vayu and vocal resonance
6. Sparsha (Skin)	Dry (Ruksha)	Rukshata due to Vata Vriddhi and Rasa Dhatu Kshaya
7. Drik (Eyes)	Conjunctival pallor, sunken eyes	Panduta (Rakta Dhatu Kshaya) and tissue exhaustion
8. Akruti (Build)	Medium build (Madhyama)	Moderate physical constitution, debilitated by chronic disease

3.2 Physical Examination & Vital Signs (Baseline)

- **Abdominal Girth:** 110 cm (measured at the level of the umbilicus)
- **Body Weight:** 64 kg
- **Abdominal Signs:** Marked abdominal distension with umbilical hernia protrusion. Positive shifting dullness on percussion, confirming ascites.
- **Pedal Edema:** 1+ bilateral non pitting pedal edema.
- **Vital Signs:** Blood Pressure: 140/90 mmHg; Pulse Rate: 62 beats/min; Respiratory Rate: 20 breaths/min.

3.3 Baseline Laboratory Parameters

- **Hemoglobin (Hb):** 7.9 g/dL
- **Serum Creatinine:** 1.94mg/dL
- **eGFR (MDRD):** 42.24 mL/min/1.73 m² (Stage 3b Chronic Kidney Disease/ Mutravaha Srotodusti)

4. Therapeutic Interventions

Management focused exclusively on oral palliative therapy (Shamana Chikitsa) and strict dietary management (Pathya Ahara). No Panchakarma or aggressive purgation (Virechana) was performed due to the patient's age, physical debility (Daurbalya), and multi-organ involvement.

4.1 Emergency Decompression & Abdominal Binding

To relieve severe abdominal tension and exertional dyspnea (Shwasa), one initial therapeutic paracentesis was conducted prior to initiating the Ayurvedic protocol. Immediately following fluid evacuation, strict abdominal binding (Pattabandha) was applied to help maintain intra-abdominal pressure, minimize the risk of rapid re-accumulation of fluid, and prevent Vata prakop associated with paracentesis.

4.2 Oral Pharmacotherapy (Shamana Aushadhi)

The oral Ayurvedic formulations initiated post-paracentesis and maintained continuously over the 5-month period are summarized in Table 3:

Medication / Formulation	Ingredients / Class	Dose & Frequency	Anupana / Timing
1. Custom Churna Yoga	Ashwagandha, Punarnava, Musta, Kutaki, Haritaki, Shankh Bhasma (Equal ratio)	2.5 g BD	After meals with warm cow's milk
2. Arogyavardhini Vati	Classical Mineral-Herbometallic formulation	250 mg Tab 1 BD	After meals with warm cow's milk
3. Chitrakadi Vati	Classical Carminative formulation	250 mg Tab 1 BD	After meals with warm cow's milk
4. Punarnava Mandoor	Classical Iron-Herbometallic formulation	250 mg Tab 1 BD	After meals with warm cow's milk

5. Gokshuradi Guggulu	Classical Diuretic / Renoprotective formulation	250 mg Tab 1 BD	After meals with warm cow's milk

4.3 Dietary Regimen (Pathya Ahara)

• **Primary Diet:** Pure cow's milk (Godugdha) combined with pearl millet flatbread (Bajari Bhakri) served with garlic (Lashuna) chutney prepared strictly without salt.

• **Dietary Rationale:** Salt restriction (Lavana Varjana) was maintained to prevent sodium-driven fluid retention. Pearl millet (Bajari) provided a low-glycemic, light (Laghu) carbohydrate source to maintain blood glucose stability and prevent hypoglycemia in this diabetic patient. Garlic provided Deepana Pachana and Vata-Anulomana properties while counteracting the cold quality (Sheeta Guna) of pearl millet.

5. Outcomes and Follow-up

The patient demonstrated 100% adherence to the prescribed medication and dietary instructions. Clinical outcomes across the 5-month follow-up period were as follows:

1. **Elimination of Paracentesis Dependency:** Following the initiation of treatment, fluid re-accumulation ceased. The patient required zero abdominal paracenteses over the entire 5-month intervention period.
2. **Gastrointestinal & Appetite Recovery:** Constipation (Malavashtambha) resolved completely within 15 days. Anorexia (Aruchi) and impaired digestion (Kshudhamandya) resolved within 30 days.
3. **Hematological & Renal Recovery:** Hemoglobin increased from 7.9 g/dL to 10.6 g/dL within 2 months, leading to full resolution of generalized weakness. Serum Creatinine reduced from 1.93 mg/dL to 1.6 mg/dL. Bilateral pedal edema resolved completely.

Table 4 outlines the chronological progression of key clinical and objective parameters:

Parameter	Baseline (Day 0)	Month 1	Month 2	Month 5
Paracentesis Frequency	Every 10 days	None required	None required	None required
Constipation (Mala)	Present (Malavashtambha)	Resolved	Resolved	Normal daily movement
Appetite (Agni)	Severe Aruchi	Fully restored	Normal	Normal
Hemoglobin (Hb)	7.9 g/dL	—	10.6 g/dL	Sustained (>10.5 g/dL)
Serum Creatinine	1.93 mg/dL	—	1.6 mg/dL	1.6 mg/dL
Pedal Edema	1+ Pitting	Reduced	Resolved	Resolved
Generalized Fatigue	Severe	Moderate improvement	Completely resolved	Completely resolved

Figures

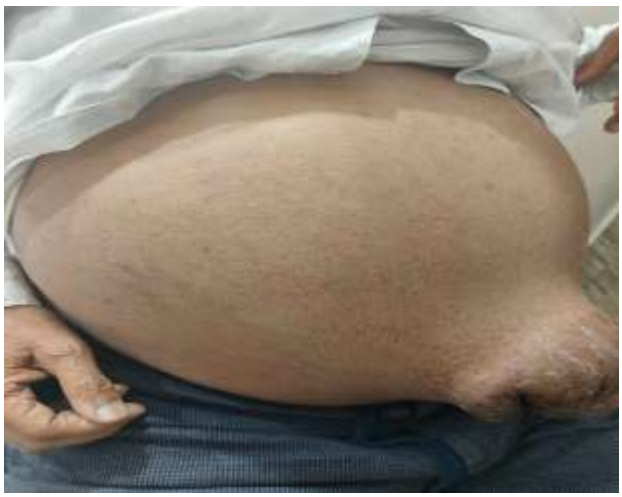

Fig 1(A)

Fig 1(B)

Fig 2

Photographic Evidence

Fig. 1. (A) Baseline presentation showing marked abdominal distention with fluid accumulation before initiation of treatment.

Fig.1.(B) First follow-up after one month of treatment, demonstrating a reduction in abdominal distention and fluid retention.
Fig. 2. Second follow-up demonstrating complete resolution of abdominal distention and absence of clinically evident fluid retention following treatment.

6. Discussion

6.1 Pathomechanism (Samprapti Vighatana)

Long-term alcohol abuse (Madya Sevana) aggravated Pitta and Vata Dosha, impairing Jatharagni and generating Ama. This led to structural and functional blockage (Srotorodha) in the Raktavaha, Udakavaha, and Purishavaha Srotas, manifesting as Yakriddalyudara and Jalodara. The fluid transudation into the abdomen was exacerbated by secondary depletion of Rasa-Rakta Dhatu and renal strain (Mutravaha Srotodusti).

Chronic Alcohol Consumption (Madya Sevana)
▼
Aggravation of Pitta & Vata + Severe Agnimandya
▼
Accumulation of Ama & Obstruction in Udakavaha & Raktavaha Srotas
▼
Jalodara (Refractory Ascites) + Pandu & Mutravaha Srotodusti
▼
Targeted Shamana Intervention (Pachana, Bhedana, & Srotoshodhana)
▼
Breakdown of Pathogenesis (Samprapti Vighatana) & Reversal of Fluid Accumulation

6.2 Pharmacodynamics of Formulations

• **Arogyavardhini Vati:** Arogyavardhini Vati is esteemed for its advantages, particularly for the liver. Arogyavardhini supports equilibrium, a healthy digestive system, and preserves the function of the liver. Katuki (Picrorhiza kurroa Royle ex Benth.), which functions as Pitta Virechana and acts on Yakrita, is its major component.[31,32]

Ascites can result from any pathology of the liver, heart, kidney, etc.; however, ascites from liver illness is challenging to cure, necessitating the necessity to address the pathology from its underlying source. These medications were given because the patient in the current instance also has hepatomegaly. It makes the liver work better.

• **Punarnava Mandoor:** Mandura is recommend for Pandu (anemia), Shotha (edema), and Shwasa (bronchial asthma), all of which resulted in a notable improvement for Pandu. Mandoor Bhasma combined with Punarnava stimulates hematopoiesis and restores liver function, raising Hemoglobin from 7.9 g/dL to 10.6 g/dL.[33,34]

• **Chitrakadi Vati:** Addresses Agnimandya and metabolizes Ama through its Deepana-Pachana action, resolving anorexia (Aruchi) and regulating bowel motility.

• **Gokshuradi Guggulu:** Targets the Mutravaha Srotas. Its Mutrala (diuretic) and Kledahara properties remove excess interstitial fluid, strengthen renal tissue, and improve Serum Creatinine levels from 1.93 mg/dL to 1.6 mg/dL.

• **Custom Churna Yoga:** Kutaki [21] provides Pachana and Bhedana (breakdown of fluid masses), directly arresting fluid accumulation. Punarnava and Musta execute targeted Kleda-pachana (digestion of excess fluid retention). Ashwagandha acts as a Balya and Rasayanaagent, countering Dhatu Kshaya and reversing severe fatigue (Daurbalya). Haritaki and Shankh Bhasma facilitate downward Vata movement (Anulomana) and relieve abdominal gas

6.3 Rationale of Dietary Regimen (Pathya)

Godugdha (Cow's Milk) is classical Pathya in Udar Roga that acts as a complete nutrient, mild diuretic, and gentle laxative while restricting free water intake. Bajari Bhakri & Garlic Chutney provided a sustained, low-glycemic carbohydrate source suited for diabetic management. Garlic (Lashuna) acts as an Agni-deepana agent, adds

necessary unctuousness (Snigdhata), and maintains Vata Anulomana. The absolute exclusion of salt (Lavana Varjana) eliminated the osmotic driver of fluid retention.

7. Conclusion

This case demonstrates that conservative Ayurvedic management using targeted oral formulations (Shamana Aushadhi) and strict diet restrictions (Pathya) can effectively manage refractory ascites (Jalodara) secondary to alcoholic liver cirrhosis. The treatment eliminated paracentesis dependency, improved hemoglobin and renal parameters, and restored quality of life in a geriatric patient with complex co-morbidities.

8. Patient Consent & Declarations

• **Informed Consent:** Written informed consent was obtained from the patient for the publication of this case report and associated clinical data.
• **Conflict of Interest:** The author declares no conflicts of interest.

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