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PITTASHAYA ASHMARI (CHOLELITHIASIS): AN AYURVEDIC NON-INVASIVE THERAPEUTIC APPROACH — A CASE REPORT

Ashwini Deshmukh[1]*, Santosh N. Belavadi[2]

*[1]PG Scholar, [2]Professor, HOD, Department of Kayachikitsa,
Shri D.G.M. Ayurvedic Medical College, Hospital, PG & Ph.D. Studies and Research Centre, Gadag, Karnataka, India.

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***Corresponding Author**
Ashwini Deshmukh
PG Scholar, Shri D.G.M. Ayurvedic Medical College, Hospital, PG & Ph.D. Studies and Research Centre, Gadag, Karnataka, India.

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ABSTRACT

Background: Gallbladder stones (cholelithiasis) represent one of the most prevalent hepatobiliary disorders globally, characterised by supersaturation and crystallisation of bile components including cholesterol and bilirubin. In Ayurvedic medicine, this condition is described as *Pittashaya Ashmari*, a subtype of *Ashmari* rooted in deranged *Pitta* and *Kapha Dosha*, impaired *Agni*, and *Ama* accumulation. Conventional management with cholecystectomy, while effective for acute obstruction, is associated with post-cholecystectomy syndrome in 20–30% of patients and does not address underlying metabolic dysregulation.[1,2,3] **Case Presentation:** A 48-year-old male presented with right upper quadrant (RUQ) abdominal pain radiating to the back, nausea, vomiting and bloating of 8 days' duration. Ultrasonography confirmed cholelithiasis with calculi measuring 4–5 mm and biliary sludge. The patient received a structured Ayurvedic *Shamana Aushadhi* protocol over 13 weeks across seven follow-up visits. **Outcome:** Following treatment, the patient achieved complete symptomatic relief and a reduction in stone size from 4–5 mm to 2 mm on follow-up ultrasonography, without surgical intervention. **Conclusion:** Ayurvedic *Shamana* formulations comprising *Chandraprabha Vati*, Cap. Plethorin, Cap. Grab, and *Kalamegha* syrup demonstrated clinically meaningful litholytic, hepatoprotective and *Pittashamaka* effects for small (<10 mm) cholesterol gallstones. Larger randomised controlled trials are warranted to validate these findings.

KEYWORDS: *Pittashaya Ashmari*; Cholelithiasis; Gallbladder stones; *Shamana Aushadhi*; Ayurvedic management; Litholysis; Chandraprabha Vati; Hepatoprotective.

1. INTRODUCTION

Pittashaya Ashmari belongs to the broader classification of *Ashmari* described in classical Ayurvedic texts. Sushruta characterised it as a condition arising from deranged *Agni* producing *Ama* that congeals into calculi within the bile channels; the pathogenesis specifically involves *Pitta–Kapha* vitiation leading to bile stasis and calculus formation.[1] Vagbhata extended a similar pathophysiological framework from urinary *Ashmari* to biliary *srotas*, observing that *Achha Pitta* stored in the *Pittashaya* (gallbladder) thickens due to aggravated *Tikshna Pitta* and *Kapha*, ultimately manifesting as *Ashmari*.[2]

In contemporary medicine, gallstones represent a significant global health burden. Epidemiological data from Harrison's Principles of Internal Medicine indicate that at least 20% of women and 8% of men over the age of 40 in the United States have gallstones, with prevalence rising to approximately 40% in women above 65 years of age; an estimated 25 million individuals in the United States are affected and approximately one million new cases of cholelithiasis are diagnosed annually.[3]

Cholecystectomy, while the definitive treatment for symptomatic cholelithiasis, is associated with post-cholecystectomy syndrome manifesting as recurrent symptoms in 20–30% of patients, bile reflux, and persistent metabolic dysfunction. Furthermore, it offers no non-invasive dissolution pathway for small stones (<10 mm) and does not address the root metabolic and dietary causes.[4]

Ayurveda employs a range of litholytic, cholagogue, and *Pitta-shamana* formulations — including *Chandraprabha Vati* (containing *Triphala*, *Shilajit*, and *Guggulu*) — alongside polyherbal preparations targeting hepatobiliary function and *Ama* clearance. These approaches offer holistic management without surgical intervention when integrated with appropriate *Pathya* (dietary regulation).

This case report documents the clinical course, treatment protocol, and outcome of a 48-year-old male patient with ultrasonographically confirmed cholelithiasis managed exclusively with Ayurvedic *Shamana Aushadhi* over a period of 13 weeks.

2. CASE PRESENTATION

2.1 Patient Demographics and Chief Complaints

A 48-year-old married male labourer, resident of Gadag, Karnataka, India, presented to the Kayachikitsa outpatient department of Shri D.G.M. Ayurvedic Medical College and Hospital, Gadag (OPD No. 25014912) on 11 April 2025 with the following complaints:

- Pain in the right upper quadrant (RUQ) of the abdomen radiating to the back, aggravating after meals — 8 days' duration
- Nausea and vomiting — 8 days' duration
- Abdominal bloating — 8 days' duration

2.2 History

The patient was apparently healthy until 8 days prior to presentation, when he developed sudden-onset right upper quadrant abdominal pain with radiation to the back. Associated nausea, vomiting and bloating were noted. He had taken antispasmodic and antiemetic medications without relief before seeking Ayurvedic care. He is not a known case of diabetes mellitus or hypertension, and reported only occasional alcohol consumption and habitual tea intake (3–4 cups daily).

2.3 Personal and General History

Table 1: Personal History and General Condition.

Parameter	Details
General Condition	Moderate
Appetite	Reduced
Diet	Mixed (vegetarian and non-vegetarian)
Bowel Habits	Irregular
Urine	4 times/day, 2 times/night
Thirst	Normal
Sleep	Disturbed
Habits	Alcohol (occasional); tea 3–4 times/day
Occupation	Manual labourer

2.4 General Examination

The patient was conscious, cooperative, and well oriented to time, place, and person. Examination revealed the absence of pallor, icterus, clubbing, pedal oedema, and lymphadenopathy.

Table 2: Vital Parameters at Presentation.

Vital Parameter	Value
Blood Pressure	140/90 mmHg
Weight	71 kg
Pulse Rate	88 beats per minute
Respiratory Rate	17 breaths per minute
Temperature	96.8°F (36.0°C)

2.5 Systemic Examination

Table 3: Systemic Examination Findings.

System	Findings
Central Nervous System	Conscious, well-oriented; no focal neurological deficit
Cardiovascular System	S1 and S2 heard; no murmurs
Respiratory System	Normal vesicular breath sounds bilaterally
Per Abdomen — Palpation	Tenderness in right upper quadrant radiating to the back; Murphy's sign positive
Per Abdomen — Percussion	Dullness in the right hypochondrium
Per Abdomen — Auscultation	Bowel sounds present and audible

3. INVESTIGATIONS

3.1 Haematological and Biochemical Investigations (05 April 2025)

Table 4: Laboratory Investigations at Baseline.

Parameter	Value	Reference Range
Complete Blood Count (CBC)	**Normal**	Within normal limits
Random Blood Sugar	**97.6 mg/dl**	70–140 mg/dl
Serum Creatinine	**1.0 mg/dl**	0.7–1.3 mg/dl
Total Bilirubin	**0.74 mg/dl**	0.2–1.2 mg/dl
Conjugated (Direct) Bilirubin	**0.30 mg/dl**	0.0–0.4 mg/dl
Unconjugated (Indirect) Bilirubin	**0.44 mg/dl**	0.0–0.8 mg/dl
SGOT (AST)	**20.1 IU/L**	10–40 IU/L
SGPT (ALT)	**23.5 IU/L**	7–40 IU/L
Alkaline Phosphatase (ALP)	**101.7 IU/L**	44–147 IU/L
Total Protein	**6.8 g/dl**	6.0–8.3 g/dl
Serum Albumin	**4.9 g/dl**	3.5–5.0 g/dl
Serum Globulins	**1.9 g/dl**	2.0–3.5 g/dl
A:G Ratio	**2.6**	1.0–2.2

3.2 Urine Analysis (05 April 2025)

Physical examination: 4 ml, pale yellow, clear. Chemical examination: albumin trace; glucose 1.5%; ketone bodies negative; bilirubin negative; urobilinogen normal; nitrite negative. Urine findings were non-specific and did not indicate renal calculi or significant proteinuria.

3.3 Ultrasonography of the Abdomen (05 April 2025)

Impression: The gallbladder was well distended with several intraluminal calculi measuring 4–5 mm. The gallbladder was near-completely filled with biliary sludge, with minimal thickening of the gallbladder wall. The sonographic features were consistent with cholelithiasis with suspected acute calculus cholecystitis. No intrahepatic or extrahepatic biliary dilatation was identified.

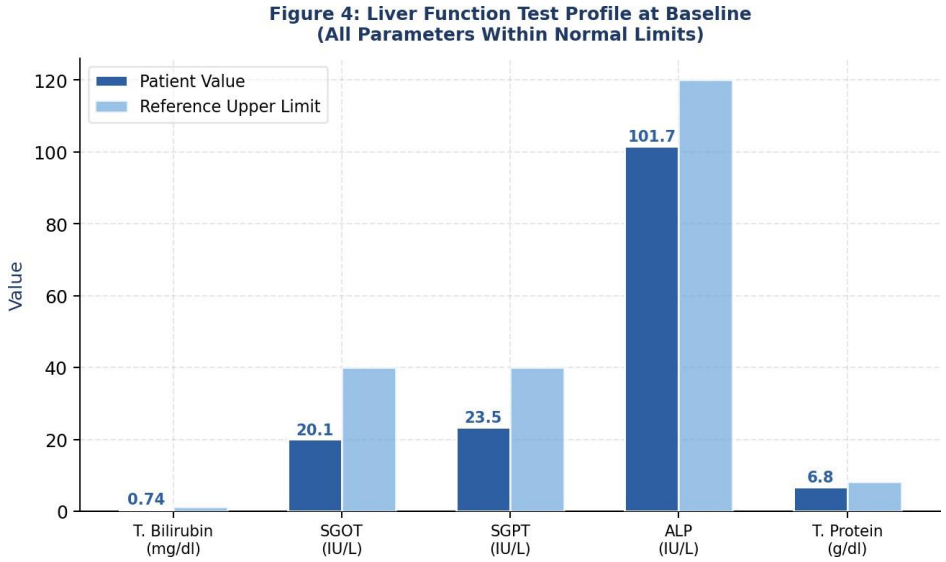

Figure 4: Graphical representation of liver function test values at baseline compared with upper reference limits. All parameters were within the normal physiological range, confirming absence of hepatocellular injury at presentation.

4. THERAPEUTIC INTERVENTION

The patient was managed with sequential *Shamana Aushadhi* protocols adjusted at each follow-up visit based on clinical response. No surgical or invasive intervention was performed. The treatment was conducted over seven outpatient visits spanning April to July 2025. Pathya (dietary modification): low-fat, easily digestible, *Pitta*-pacifying diet was prescribed throughout. All formulations were administered orally (AF = after food; BF = before food; BD = twice daily; TID = thrice daily).

Table 5: Therapeutic Intervention — Clinical Timeline and Drug Protocol.

S. No.	Date	USG / Observation	Symptom Status	Treatment Prescribed
01	11 Apr 2025	GB calculi 4–5 mm; GB wall minimally thickened; sludge present	Pain RUQ radiating to back, nausea, vomiting, bloating	Cap. Grab 1 BD AF; Cap. Plethorin 1 BD AF; Chandraprabha Vati 1 BD AF
02	24 Apr 2025	Clinical follow-up	Pain RUQ persistent	Laghu Sutashekara Rasa 1 BD BF; Kamadugha Rasa 1 BD AF

03	17 May 2025	Clinical follow-up	Pain mildly reduced; patient improving	Cap. Grab 1 BD AF; Cap. Plethorin 1 BD AF; Chandraprabha Vati 1 BD AF
04	04 Jun 2025	Clinical follow-up	Pain reduced; constipation noted	Anulomana DS 1 tab night; Shankha Vati 1 BD AF
05	11 Jun 2025	Clinical follow-up	Occupational pain; improving	Tab. Liv 52 DS 1 BD AF; Kalamegha Syrup 3 tsp BD BF; Cap. Plethorin 1 TID AF
06	28 Jun 2025	Clinical follow-up	Complete pain relief; symptoms improved	Cap. Plethorin 1 BD AF; Tab. Liv 52 DS 1 BD AF; Kalamegha Syrup 3 tsp BD BF; Nirocil-DS 1 BD AF
07	12 Jul 2025	USG: Stone reduced to 2 mm; no sludge	Reduced pain; overall condition improved	Cap. Plethorin 1 BD AF; Tab. Liv 52 DS 1 BD AF; Kalamegha Syrup 3 tsp BD BF

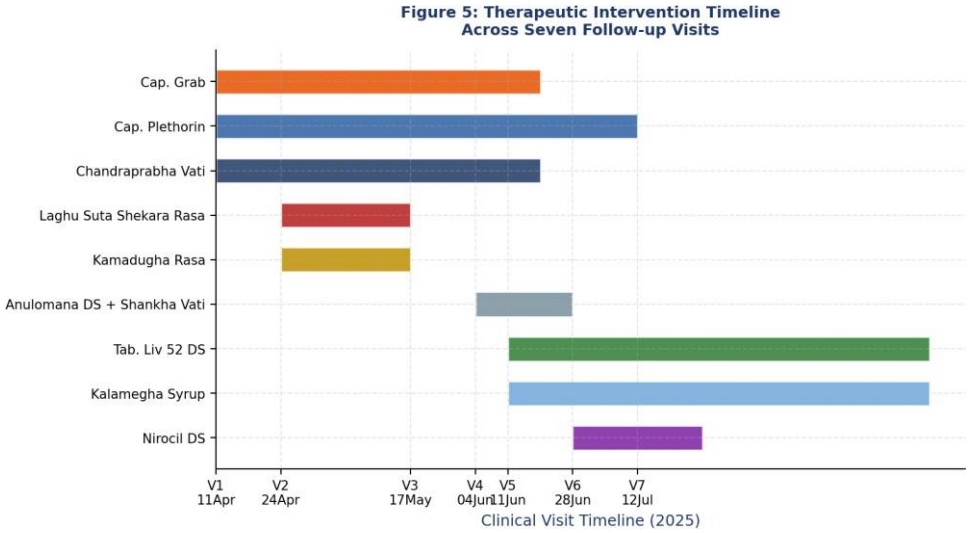

Figure 5: Gantt-style treatment timeline depicting the duration of each Shamana Aushadhi formulation across all seven follow-up visits (April–July 2025).

5. OUTCOME AND FOLLOW-UP

5.1 Stone Size Reduction

Serial ultrasonographic assessment demonstrated a progressive and clinically significant reduction in gallbladder calculi size: from 4–5 mm at baseline to 2 mm at the final visit on 12 July 2025 — representing an approximate 55–60% reduction in stone diameter over the 13-week treatment period.

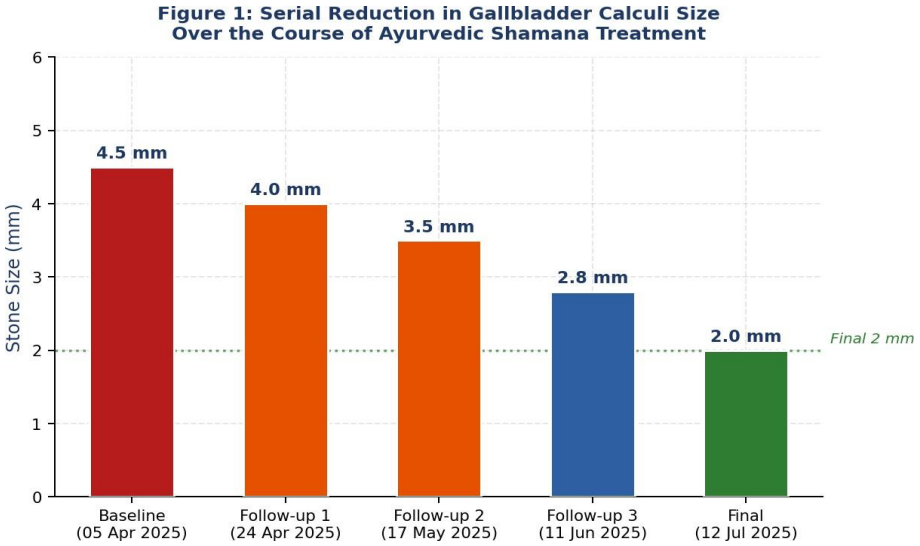

Figure 1: Serial reduction in gallbladder calculi size (mm) across five imaging assessments over the course of Ayurvedic Shamana treatment. Red bars indicate symptomatic phase; green bar indicates final follow-up with maximum stone reduction.

5.2 Symptomatic Improvement

All principal presenting symptoms demonstrated progressive improvement. The patient reported complete resolution of right upper quadrant pain, nausea, and vomiting by the sixth visit (28 June 2025). Bloating, sleep disturbance, and appetite were normalised by the final visit. Improvement is quantified below using a self-reported symptom severity scale (0 = absent, 10 = maximum severity):

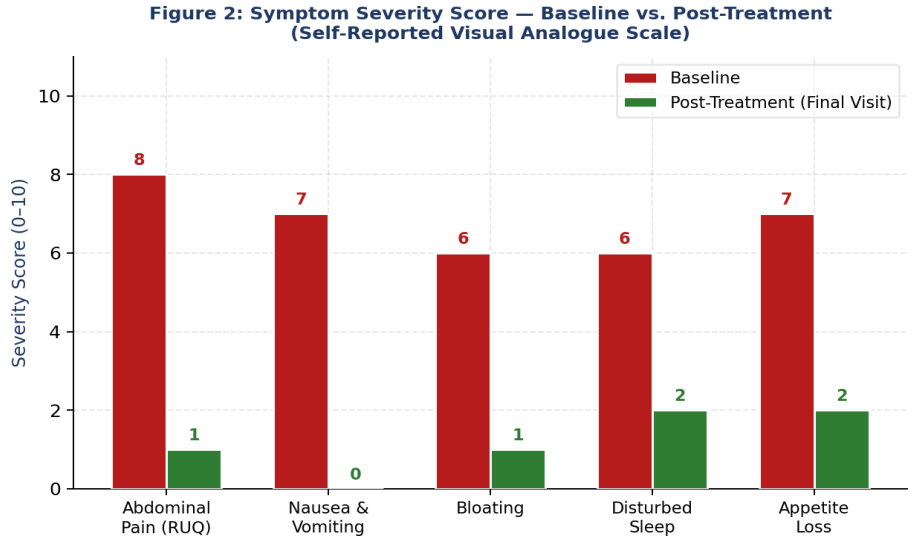

Figure 2: Grouped bar chart comparing baseline and post-treatment symptom severity scores across five clinical parameters on a 10-point self-reported scale. Scores declined markedly across all parameters.

6. DISCUSSION

6.1 Ayurvedic Pathophysiology

The pathogenesis of *Pittashaya Ashmari* involves a cascade of doshic dysregulations closely paralleling the modern pathophysiology of cholesterol cholelithiasis (Figure 3). *Pitta Dosha* vitiation — corresponding to disrupted bile acid metabolism and cholesterol supersaturation — is the primary aetiological factor.[1] Concurrent *Kapha* aggravation generates the viscous, stagnating quality of bile sludge (analogous to biliary mucin aggregation), while *Agni Mandya* (impaired digestive fire) produces *Ama* — unprocessed metabolic waste — which functions as the nidus for crystallisation.[2]

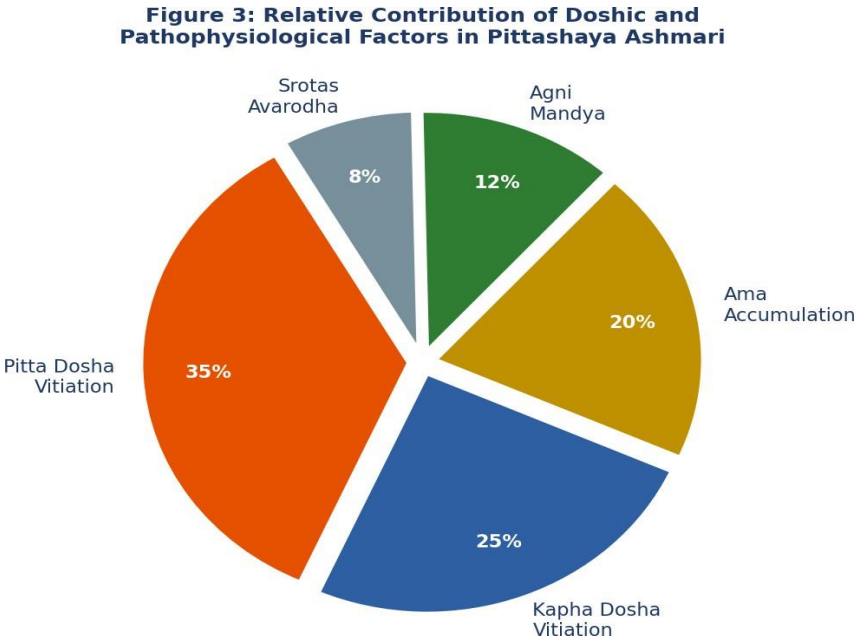

Figure 3: Pie chart depicting the relative contribution of Doshic and pathophysiological factors (Pitta vitiation, Kapha vitiation, Ama accumulation, Agni Mandya, and Srotas Avarodha) in the aetiopathogenesis of Pittashaya Ashmari according to classical Ayurvedic frameworks.

6.2 Pharmacological Rationale of Formulations

Cap. Plethorin contains *Punarnava* (*Boerhaavia diffusa*) and *Gokshura* (*Tribulus terrestris*) as principal constituents, exerting *Mutrala* (diuretic) and *Lekhana* (scraping/litholytic) actions through alkalinisation of bile and promotion of biliary gravel expulsion.[5,6] The triterpenoids of *Punarnava* inhibit stone nucleation via anti-inflammatory eicosanoid modulation, while saponins from *Gokshura* enhance biliary solubility of calculi components. Together, these drugs restore *Yakrit-Pittashaya srotas* balance by reducing *Ama* and *Pitta* aggravation.

Cap. Grab leverages *Kutaja* (*Holarrhena antidysenterica*) and *Vidanga* (*Embelia ribes*) for *Krimighna* (antimicrobial) and *Grahi* (astringent) effects, targeting biliary infections and *Ama*-mediated stone accretion through inhibition of bacterial urease and toxin-binding activity.[7,8] Conessine alkaloids from *Kutaja* disrupt microbial biofilms in bile ducts, while *Vidanga's* embelin stimulates *Agni* and demonstrates mild cholagogic activity. The combination exerts *Shothahara* and *Shulahara* effects, relieving biliary inflammation and pain.

Chandraprabha Vati, a classical poly-herbal mineral formulation, contains *Triphala*[9] as a gentle digestive cleanser enhancing bile secretion and flow essential for fat emulsification. *Shilajit*[10] provides hepatoprotective and detoxification support. *Guggulu*[11] contributes anti-inflammatory benefits modulating gallbladder and bile duct function. *Pippali*[12] augments *Agni* and enhances bioavailability of co-administered drugs. Collectively, these constituents balance *Pitta Dosha*, crucial for bile homeostasis in Ayurvedic therapeutics.

Kalamegha syrup, based on *Andrographis paniculata*, delivers *Tikta Rasa* and *Ushna Virya* for potent *Pittashamaka* and *Yakrit Prasadana* effects.[13] The principal diterpenoid andrographolide inhibits NF-κB pathways, suppressing hepatocyte inflammation and oxidative stress, while acting as a cholagogue by upregulating bile acid synthesis enzymes. Phyllanthin-mediated hypocholesterolaemic activity supports litholysis of cholesterol stones. Dehydroandrographolide further enhances antioxidant defences preventing free radical-induced stone precipitation.

6.3 Integrative Biomedical Perspective

The observed stone size reduction from 4–5 mm to 2 mm is consistent with the recognised threshold for non-surgical management of small cholesterol gallstones (<10 mm), where litholytic pharmacotherapy with ursodeoxycholic acid or equivalent agents can achieve stone dissolution at rates of 0.5–1.5 mm per month. The time course and magnitude of dissolution in this case are concordant with documented phytochemical mechanisms of the constituent herbs — particularly the cholagogue (bile acid augmentation) and anti-nucleating (crystallisation inhibition) pathways. The absence of baseline hepatocellular injury (normal SGOT, SGPT, ALP) is noteworthy in supporting a conservative non-surgical approach. Simultaneously, the mild elevation in A:G ratio warrants monitoring in extended follow-up.

6.4 Limitations

This case report presents a single-patient observation. The absence of a comparative control arm, randomisation, and blinded outcome assessment limits causal inference. Repeat ultrasonography was not performed at interim timepoints. Stone composition analysis (cholesterol vs. pigment stone type) was not available; Ayurvedic litholytic formulations are predominantly effective in cholesterol cholelithiasis. Adherence to *Pathya* dietary guidelines as a potential confounding factor cannot be fully quantified. Larger, prospective, randomised controlled trials with standardised outcome measures are necessary to generalise these findings.

7. CONCLUSION

This case report demonstrates that a structured Ayurvedic *Shamana Aushadhi* protocol — comprising *Chandraprabha Vati's* multi-herbal hepatobiliary detoxification, Plethorin's litholytic action on cholesterol stones, and *Kalamegha's* hepatoprotective bitter-tonic effects — can achieve clinically significant symptom resolution and objective stone size reduction (55–60%) in small-stone cholelithiasis (<10 mm) within 13 weeks of treatment, without surgical intervention.

Ayurveda provides a mechanistically coherent, non-invasive, root-cause-oriented alternative to cholecystectomy for selected patients with small cholesterol gallstones, particularly when integrated with *Pathya* (low-fat, *Pitta*-pacifying diet) and *Nidana Parivarjana* (elimination of causative factors). *Prakriti*-specific protocol individualisation remains central to optimising outcomes. These findings support the conduct of prospective randomised controlled trials comparing Ayurvedic *Shamana* protocols with conventional ursodeoxycholic acid therapy and watchful waiting in appropriately selected patient populations.

PATIENT CONSENT STATEMENT

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical data. The patient's identifying information has been protected in accordance with institutional ethical standards. The study was conducted in adherence to the principles of the Declaration of Helsinki.

DECLARATIONS

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

Funding: This study received no external funding.

Author Contributions: AD: patient management, data collection, manuscript preparation. SNB: clinical supervision, manuscript review and approval.

REFERENCES

1. Kaviraj Ambikadutta Shastri, editor. Sushruta Samhita, Nidanasthana: Ashmari Nidana [Ayurveda Tattva Sandipika commentary]. Varanasi: Chaukhamba Sanskrit Sansthan, verse 24, 315.
2. Kunte AM, Navre KRS, editors. Ashtanga Hridaya of Vagbhata, Nidanasthana: Ashmari Nidana [Paradakara commentary]. Varanasi: Chaukhamba Sanskrit Sansthan, verse 32, 501.
3. Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York: McGraw-Hill 2008; 1992.
4. Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York: McGraw-Hill, 2008; 1997.
5. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan; 681. [Punarnava — Boerhaavia diffusa]
6. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan, 308. [Gokshura — Tribulus terrestris]
7. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan, 542. [Kutaja — Holarrhena antidysenterica]
8. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan, 894. [Vidanga — Embelia ribes]
9. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan, 976. [Triphala]
10. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan, 330. [Shilajit]
11. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan; p. 664. [Guggulu — Commiphora wightii]
12. Hegde PL, Harini A. Dravyaguna Vijnana. Varanasi: Chaukhamba Sanskrit Sansthan, 431. [Pippali — Piper longum]
13. Sharma PC, Yelne MB, Dennis TJ, editors. Database on Medicinal Plants Used in Ayurveda. 2002; 1: New Delhi: CCRAS, 48–62. [Kalmegh — Andrographis paniculata]