



NON-SURGICAL AYURVEDIC MANAGEMENT OF CHOLELITHIASIS (*PITTASHMARI*): A CASE STUDY

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ABSTRACT

Cholecystectomy, the surgical removal of the gallbladder, remains the standard and most widely practiced treatment for cholelithiasis (gallstones). Nevertheless, increasing attention has been directed toward non-surgical alternatives, as conventional surgery entails complete organ removal. Timely management of gallstones is crucial not only for symptomatic relief but also for preventing serious complications such as acute cholecystitis. However, current medical therapies provide limited efficacy and accessibility in the conservative management of cholelithiasis.

Existing non-surgical modalities—including extracorporeal shock wave lithotripsy (ESWL), contact dissolution therapy using methyl tertiary-butyl ether (MTBE), and oral bile acid therapy with ursodiol or chenodiol—have demonstrated varying degrees of success but are constrained by safety concerns, recurrence rates, and procedural limitations. Although percutaneous cholecystostomy offers a minimally invasive alternative, its indications remain restricted. While cholecystectomy achieves a recurrence prevention rate of approximately 99%, 10–15% of patients develop post-cholecystectomy syndrome, underscoring the unmet need for safe, effective, and organ-preserving therapeutic strategies.

This article endeavors to advance the discourse on developing novel, evidence-based, non-surgical modalities for the management of cholelithiasis—aiming to provide a safer and more holistic alternative to conventional surgical intervention.

KEYWORDS: Cholelithiasis, Cholecystectomy *Pittashmari*, *Pittashaya*

INTRODUCTION

The presence of stones within the gallbladder is known as **cholelithiasis**. Gallstones form when the relative concentrations of various constituents in bile become imbalanced, leading to the precipitation

of solid particles. The composition of gallstones varies depending on **age, dietary habits, and ethnic background**.^[1]

Based on their chemical composition, gallstones are generally classified into **cholesterol, bilirubin (pigment), and mixed stones**.

Cholesterol Stones

Cholesterol stones range in color from light yellow to dark green, brown, or chalk white. They are typically oval, solitary, and measure about **2–3 cm in length**, often with a small dark central area. For a stone to be classified as a cholesterol stone, it must contain **at least 80% cholesterol by weight** (or **70%** according to the Japanese classification system).^[2]

Bilirubin (Pigment) Stones

Bilirubin or **black pigment stones** are usually small, dark, and multiple. They are primarily composed of **bilirubin polymer** (an insoluble pigment) and **calcium phosphate salts** present in bile. These stones generally contain **less than 20% cholesterol** (or **30%** per the Japanese classification).^[2]

Mixed Stones

Mixed or **brown pigment stones** contain **20–80% cholesterol** (or **30–70%** in the Japanese classification).^[2] Other common constituents include **calcium carbonate, palmitate phosphate, bilirubin, and bile pigments** such as **calcium bilirubinate, calcium palmitate, and calcium stearate**. Owing to their high calcium content, these stones are often radiopaque. Infections of the biliary tract may contribute to their formation, as bacterial **β-glucuronidase** enzymes hydrolyze bilirubin glucuronides, thereby increasing the amount of unconjugated bilirubin in bile.

Clinical Presentation

A characteristic symptom of gallstones is the “**Gallstone attack**”, characterized by intense pain in the **right upper quadrant** of the abdomen. This pain may radiate to the **back, right shoulder, or interscapular region** and is often accompanied by **nausea and vomiting**. Typically, the pain intensifies gradually over 30 minutes to several hours. Attacks are frequently precipitated by **fatty meals**, usually occur **at night**, and may resemble **renal colic** in their presentation.

Risk Factors

Several factors contribute to the development of gallstones, particularly **cholesterol stones**. These include **female sex, fertility, and a positive family history** of gallstone disease.^[3] **Obesity**^[4,5] and other components of **metabolic syndrome**^[6]—such as **Dyslipidemia** (especially **type IV hyperlipoproteinemia**^[7,8] with **hypertriglyceridemia** and **low HDL cholesterol**), **insulin resistance**, or **type 2 diabetes mellitus**^[9,10]—are also recognized risk factors. Gallstone formation is thus considered a possible complication of metabolic syndrome.^[11]

Oestrogen therapy increases the risk of gallstone formation, both in **women** (when used for contraception or hormone replacement therapy)^[12] and in **men with prostate cancer** receiving hormonal treatment.^[13,14]

Among **dietary factors**, a **short-term high-cholesterol**^[15] or **high-carbohydrate diet**^[16,17] has been associated with a higher incidence of gallstones, whereas the intake of **legumes**^[18], **unsaturated fats**^[19], **coffee**^[20], and **moderate alcohol consumption**^[21,22] appears to lower the risk.

Regular physical activity reduces the risk of **symptomatic gallstone disease** in both men and women, independent of weight loss.^[23,24] Conversely, **rapid or excessive weight loss**^[25,26] and **weight cycling**^[27,28] markedly increase the risk of gallstone formation; therefore, weight reduction should not exceed **1.5 kg per week**.^[29]

Certain **lipid-lowering agents**, particularly **fibrates**, can interfere with cholesterol and bile acid metabolism, leading to increased cholesterol secretion into bile and potential gallstone formation.^[30,31] However, compared to older agents such as **clofibrate**, the incidence of gallstones with **newer fibrates** is significantly lower.^[32]

Pathophysiology

Cholesterol gallstones develop when bile becomes supersaturated with cholesterol due to an imbalance between cholesterol concentration and bile salts. In addition to excessive biliary cholesterol, two major pathophysiological factors contribute to gallstone formation.

Gallbladder motility: Hypomotility or infrequent and incomplete gallbladder emptying leads to bile stasis and excessive concentration, favoring cholesterol crystal nucleation. Furthermore, the intricate anatomical configuration of the cystic duct can impose significant resistance to bile flow, exacerbating stasis and facilitating gallstone development.

Regulatory proteins in bile: Specific hepatic and biliary proteins regulate cholesterol crystallization. Certain proteins promote nucleation and aggregation of cholesterol monohydrate crystals, while others act as inhibitors; an imbalance between these can predispose to lithogenesis.

Estrogenic influence: Elevated Estrogen levels—such as those observed during pregnancy, hormone replacement therapy, or the use of combined estrogen-containing oral contraceptives—can increase hepatic cholesterol secretion and reduce gallbladder contractility. These combined effects enhance biliary cholesterol Supersaturation and stasis, thereby promoting cholesterol gallstone formation.

Medical Treatment

Cholesterol gallstones may be dissolved with oral Ursodeoxycholic acid, though treatment can last up to two years, and stones may recur if therapy is discontinued. Endoscopic retrograde sphincterotomy (ERS) following ERCP can relieve common bile duct obstruction caused by gallstones. Extracorporeal shock wave lithotripsy uses focused ultrasonic waves to fragment gallstones, which are then excreted in feces. This approach is suitable only for patients with a limited number of stones.

Surgical Treatment

Cholecystectomy effectively prevents gallstone recurrence, with a success rate of 99%, and is indicated for symptomatic patients. Most individuals tolerate gallbladder removal well, though 10–15% develop post-cholecystectomy syndrome, presenting with gastrointestinal discomfort, persistent right upper quadrant pain, or chronic diarrhoea (~10%).

Two surgical approaches are available:

- **Open cholecystectomy** involves an abdominal incision below the right ribs. Recovery typically requires 3–5 days of hospitalization, with normal diet resumed within a week and full activity after several weeks.
- **Laparoscopic cholecystectomy**, introduced in the 1980s, uses 3–4 small punctures for a camera and instruments. Patients are usually discharged the same day or after one night, with normal diet and light activity resumed within a week. Mild residual pain and reduced energy may persist for 1–2 months. When stones are accurately located preoperatively via cholangiogram, laparoscopic cholecystectomy is as effective as open surgery

Case Report

A 50-year-old male patient presented to the *Shalya Tantra OPD* of **Sri Babu Singh Daddu Ji Ayurvedic Medical College and Hospital** in May 2024, with a previously confirmed diagnosis of **Cholelithiasis**, exhibiting characteristic clinical features. According to the patient, he had been experiencing these symptoms for approximately one year.

The patient had previously consulted several reputed allopathic physicians and was advised to undergo surgical intervention due to a lack of symptomatic improvement. However, being apprehensive about surgery, he sought conservative *Ayurvedic* management at our institution.

Past History

- Burning sensation in the epigastric region
- Loss of appetite

Personal History

Parameter	Observation
Bowel	Once daily
Micturition	3–4 times per day
Appetite	Decreased
Sleep	Sound

General Examination

Vital Sign	Observation
Blood Pressure	120/70 mmHg
Pulse Rate	78 beats/min
Respiratory Rate	20 breaths/min
Temperature	98.2°F

Systemic Examination

- **Per Abdomen (P/A):** Soft and non-tender
- **Cardiovascular System (CVS):** S1 and S2 heart sounds normal
- **Central Nervous System (CNS):** Patient conscious and well-oriented
- **Respiratory System (RS):** Air entry bilaterally clear

Ayurvedic Management

According to *Ayurvedic* principles, the clinical features and pathogenesis of *Cholelithiasis* closely resemble *Pittashmari*, a type of *Ashmari* (urinary or biliary calculi) described in classical *Ayurvedic* texts. Considering the similarity in etiology and symptomatology, the treatment protocol for *Pittashmari* was adopted for this case.

The patient was administered the following *Ayurvedic* formulations in divided doses as summarized in **Table 1**.

Table 1. Ayurvedic Formulations Administered

Sr. No.	Name of Medicine	Dose and Frequency
1.	<i>Arogyavardhini Vati</i>	1 tablet, thrice daily (TDS)
2.	<i>Shankh Vati</i>	1 tablet, thrice daily (TDS)
3.	<i>Sutashekhar Rasa</i>	1 tablet, thrice daily (TDS)
4.	<i>Avipattikar Churna</i>	3 g per day
5.	<i>Gudhal Pushpa Churna</i>	3 g per day
6.	<i>Makaya Kshara</i>	250 mg per day
7.	<i>Ashmarihara Kwatha</i>	30 ml per day

Result

The patient reported symptomatic improvement within 15 days of initiating *Ayurvedic* therapy. After three months of continuous treatment, ultrasonographic evaluation was performed and compared with pre-treatment findings.

Ultrasonographic Findings

Parameter	Before Treatment (BT)	After Treatment (AT)
Gall bladder (GB) status	Partially distended; wall thickness 4–5 mm; lumen shows echogenic shadow suggestive of 7 mm stone	Normal in size; wall thickness 3 mm; lumen echo-free; no evidence of calculi

Observation

The post-treatment ultrasonography revealed complete resolution of the gallstone and normalization of gall bladder wall thickness, indicating effective management of *Pittashmari* through Ayurvedic therapy.

Discussion

The *Pittashaya Ashmari Nidanas* can be understood through the perspective of *Doshika Nidana* described in Ayurveda. The following *Doshika Nidanas* are relevant to *Pittashaya Ashmari*: *Kaphakara Nidana*, *Pittakara Nidana*, and *Pandu and Kamala Roga Nidana*.

According to Ayurvedic principles, the pathogenesis (*Samprapti*) of gallstones (*Pittashmari*) involves both *Kapha* and *Pitta* doshas. The consumption of *Kaphaprakopaka* and *Pittakaraka Nidanas* (causative factors) initially leads to *Kapha* accumulation, producing symptoms such as *Ālasya* (lethargy), *Gaurava* (heaviness), and *Mandāgni* (reduced digestive fire). The vitiated *Kapha* combines with *Pitta* in the *Pittashaya* (gallbladder), forming a thick, viscous substance analogous to biliary sludge, which obstructs the normal passage of *Vayu*. Consequently, the *Vata dosha* becomes aggravated due to its *Rūksha* (dry) and *Laghu* (light) properties, solidifying the viscous material into *Ashmari* (stone), thus resulting in *Pittashmari*, comparable to Cholelithiasis in modern medicine.

Pittashmari (gallstones) arises from the simultaneous vitiation of *Pitta* and the formation of *Ashmari*, as its nomenclature suggests. Therefore, management should focus on therapeutic agents possessing properties that counteract both these factors.

The ingredients of *Ashmarihara Kwatha*, *Arogyavardhini Vati*, and *Makaya Kshara* exhibit *Lekhana* (scraping), *Chedana* (cutting), *Bhedana* (breaking), *Mutrala* (diuretic), *Anulomana* (downward movement facilitator), *Deepana* (appetizer), *Pachana* (digestive), *Vedanasthapana* (analgesic), and *Tridosha-shamaka* (balancing all three doshas) properties. These actions assist in dissolving or reducing the size of *Ashmari* (stones).

Shankhavati helps reduce acidity and inflammation, alleviates gallbladder pain, aids in stone dissolution, and prevents infection. ***Sootasekhara Rasa*** stimulates hepatic and biliary function, enhancing digestion. ***Avipattikara Churna*** decreases acidity and inflammation, relieves gallbladder pain, purifies the liver and gallbladder, promotes digestion, and assists in dissolving calculi.

Conclusion

This case study demonstrates a positive outcome in the management of Cholelithiasis (*Pittashmari*) using *Ayurvedic* interventions. Ultrasonography of the abdomen revealed that the patient successfully eliminated a 7 mm gallstone within a three-month period through dedicated Ayurvedic treatment. Alongside this, the patient exhibited marked improvements in overall health, including enhanced digestion, energy levels, and well-being.

Ayurvedic management of gallbladder stones emphasizes a holistic approach, integrating dietary modifications, lifestyle adjustments, herbal formulations, and targeted therapies. This approach not only addresses the pathological formation of stones but also strengthens digestive and metabolic functions, promoting long-term health and prevention of recurrence.

The findings from this case highlight the potential of Ayurveda as an effective and safe alternative or adjunctive therapy for Cholelithiasis. By encouraging patients to embrace Ayurvedic principles, it is possible to achieve both clinical improvement and a better quality of life. Moreover, integrating traditional healing practices with modern understanding can contribute to public health promotion and support individuals in leading healthier, more balanced, and fulfilling lives.

REFERENCES

1. Channa NA, Khand FD, Khand TU, Leghari MH, Memon AN. Analysis of human gallstones by Fourier Transform Infrared (FTIR). *Pak J Med Sci.* 2007;23(4):546–50.
2. Kim IS, Myung SJ, Lee SS, Lee SK, Kim MH. Classification and nomenclature of gallstones revisited. *Yonsei Med J.* 2003;44(4):561–70.
3. Attili AF, Capocaccia R, Carulli N, et al. Factors associated with gallstone disease in the MICOL experience. *Hepatology.* 1997;26:809–18.
4. Stampfer MJ, Maclure KM, Colditz GA, Manson JE, Willett WC. Risk of symptomatic gallstones in women with severe obesity. *Am J Clin Nutr.* 1992;55:652–8.
5. Amaral JF, Thompson WR. Gallbladder disease in the morbidly obese. *Am J Surg.* 1985;149:551–7.
6. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet.* 2005;365:1415–28.
7. Ahlberg J, Angelin B, Einarsson K, Hellstrom K, Leijdt B. Prevalence of gallbladder disease in hyperlipoproteinemia. *Dig Dis Sci.* 1979;24:459–64.
8. Einarsson K, Hellstrom K, Kallner M. Gallbladder disease in hyperlipoproteinaemia. *Lancet.* 1975;1:484–7.
9. Einarsson K, Hellstrom K, Kallner M. Bile acid kinetics in relation to sex, serum lipids, body weights, and gallbladder disease in patients with various types of hyperlipoproteinemia. *J Clin Invest.* 1974;54:1301–11.
10. Nervi F, Miquel JF, Alvarez M, et al. Gallbladder disease is associated with insulin resistance in a high-risk Hispanic population. *J Hepatol.* 2006;45:299–305.
11. Scragg RK, Calvert GD, Oliver JR. Plasma lipids and insulin in gallstone disease: a case-control study. *Br Med J (Clin Res Ed).* 1984;289:521–5.
12. Grundy SM. Cholesterol gallstones: a fellow traveler with metabolic syndrome? *Am J Clin Nutr.* 2004;80:1–2.
13. Scragg RK, McMichael AJ, Seamark RF. Oral contraceptives, pregnancy, and endogenous oestrogen in gallstone disease—a case-control study. *Br Med J (Clin Res Ed).* 1984;288:1795–9.
14. Henriksson P, Einarsson K, Eriksson A, Kelter U, Angelin B. Estrogen-induced gallstone formation in males: relation to changes in serum and biliary lipids during hormonal treatment of prostatic carcinoma. *J Clin Invest.* 1989;84:811–6.
15. Angelin B, Olivecrona H, Reihner E, et al. Hepatic cholesterol metabolism in estrogen-treated men. *Gastroenterology.* 1992;103:1657–63.
16. Lee DW, Gilmore CJ, Bonorris G, et al. Effect of dietary cholesterol on biliary lipids in patients with gallstones and normal subjects. *Am J Clin Nutr.* 1985;42:414–20.
17. Scragg RK, McMichael AJ, Baghurst PA. Diet, alcohol, and relative weight in gallstone disease: a case-control study. *Br Med J (Clin Res Ed).* 1984;288:1113–9.
18. Tsai CJ, Leitzmann MF, Willett WC, Giovannucci EL. Glycemic load, glycemic index, and carbohydrate intake in relation to risk of cholecystectomy in women. *Gastroenterology.* 2005;129:105–12.
19. Nervi F, Covarrubias C, Bravo P, et al. Influence of legume intake on biliary lipids and cholesterol saturation in young Chilean men: identification of a dietary risk factor for cholesterol gallstone formation in a highly prevalent area. *Gastroenterology.* 1989;96:825–30.
20. Tsai CJ, Leitzmann MF, Willett WC, Giovannucci EL. The effect of long-term intake of cis unsaturated fats on the risk for gallstone disease in men: a prospective cohort study. *Ann Intern Med.* 2004;141:514–22.
21. Leitzmann MF, Willett WC, Rimm EB, et al. A prospective study of coffee consumption and the risk of symptomatic gallstone disease in men. *JAMA.* 1999;281:2106–12.
22. Leitzmann MF, Giovannucci EL, Stampfer MJ, et al. Prospective study of alcohol consumption patterns in relation to symptomatic gallstone disease in men. *Alcohol Clin Exp Res.* 1999;23:835–41.
23. Leitzmann MF, Giovannucci EL, Rimm EB, et al. The relation of physical activity to risk for symptomatic gallstone disease in men. *Ann Intern Med.* 1998;128:417–25.

24. Leitzmann MF, Rimm EB, Willett WC, et al. Recreational physical activity and the risk of cholecystectomy in women. *N Engl J Med*. 1999;341:777–84.
25. Liddle RA, Goldstein RB, Saxton J. Gallstone formation during weight-reduction dieting. *Arch Intern Med*. 1989;149:1750–3.
26. Gustafsson U, Benthin L, Granstrom L, Groen AK, Sahlin S, Einarsson C. Changes in gallbladder bile composition and crystal detection time in morbidly obese subjects after bariatric surgery. *Hepatology*. 2005;41:1322–8.
27. Syngal S, Coakley EH, Willett WC, Byers T, Williamson DF, Colditz GA. Long-term weight patterns and risk for cholecystectomy in women. *Ann Intern Med*. 1999;130:471–7.
28. Tsai CJ, Leitzmann MF, Willett WC, Giovannucci EL. Weight cycling and risk of gallstone disease in men. *Arch Intern Med*. 2006;166:2369–74.
29. Weinsier RL, Wilson LJ, Lee J. Medically safe rate of weight loss for the treatment of obesity: a guideline based on risk of gallstone formation. *Am J Med*. 1995;98:115–7.
30. Angelin B, Einarsson K, Leijdt B. Clofibrate treatment and bile cholesterol saturation: short-term and long-term effects and influence of combination with chenodeoxycholic acid. *Eur J Clin Invest*. 1981;11:185–9.
31. Angelin B, Einarsson K, Leijdt B. Biliary lipid composition during treatment with different hypolipidaemic drugs. *Eur J Clin Invest*. 1979;9:185–90.
32. Grundy SM, Vega GL. Fibric acids: effects on lipids and lipoprotein metabolism. *Am J Med*. 1987;83:9–20.