



Original Article

Non Surgical Management of Cholelithiasis (Pittashmari): A Case Study

Sharma R. K.^{1*}, Shah H.S.², Gohel J.K.³¹Professor Department of Shalyatantra, Government Akhandanand Ayurveda College, Ahmedabad, Gujarat-India²Associate Professor, Department of Shalyatantra, Government Akhandanand Ayurveda College, Ahmedabad, Gujarat-India³PG Research Scholars, Department of Shalyatantra, Government Akhandanand Ayurveda College, Ahmedabad, Gujarat-India

ABSTRACT

As a matter of fact, surgery (cholecystectomy or gall bladder removal) is the most common form of treatment for cholelithiasis (gallstones). However, the fact that surgically removing gallstones requires the removal of an entire organ has led to a growing interest in non-surgical treatments for gallstones. Besides alleviating symptoms, treatment for gallstones is necessary to avoid a progression that can result in severe conditions such as acute cholelithiasis. But so far as the medical management of cholelithiasis is concerned, it is not up to the mark in modern healing system. Although ursodiol or chenodiol in the form of oral bile acid pills, extracorporeal shock-wave lithotripsy (ESWL), contact dissolution therapy [injecting a solvent known as Methyl tertiary-butyl ether (MTBE) into the gallbladder to dissolve the gallstones], Percutaneous Cholecystostomy are good non-surgical measures, their roles are either limited or these are not free from adverse effects. Obviously, there is an urgent need of help from Alternative therapy to counter these difficulties. This article is a step in the direction of making an availability of a safe and effective non-surgical management of cholelithiasis to the ailing mankind.

Key words: Cholelithiasis, Cholecystectomy, Pittashmari, Herbal medicine

ARTICLE INFO: Received: 16 Oct. 2018; Review Completed: 15 Jan. 2019; Accepted: 09 Feb. 2019; Available online: 15 Feb. 2019



Cite this article as:

Sharma R. K.*, Dr. Shah H.S., Dr. Gohel J.K., Non Surgical Management of Cholelithiasis (Pittashmari): A Case Study, Asian Journal of Pharmaceutical Research and Development. 2019; 7(1): 34-37

DOI: <http://dx.doi.org/10.22270/ajprd.v7i1.443>

*Address for Correspondence

Dr. R. K. Sharma, Professor, Department of Shalyatantra, Government Akhandanand Ayurveda College, Ahmedabad, Gujarat-India

INTRODUCTION

Presence of stones in the gallbladder is referred to as cholelithiasis. The stones are formed when the concentrations of various constituents in the gall bladder are not in the desired proportions. The composition of gallstones is affected by age, diet and ethnicity.¹ On the basis of their composition, gallstones can be divided into the following types:

Cholesterol stones

Cholesterol stones vary from light yellow to dark green or brown or chalk white and are oval, usually solitary, between 2 and 3 cm long, each often having a tiny, dark, central spot. To be classified as such, they must be at least 80% cholesterol by weight (or 70%, according to the Japanese- classification system).

Bilirubin stones

Bilirubin ("Pigment", "Black Pigment") stones are small, dark (often appearing black), and usually numerous. They are composed primarily of bilirubin (insoluble bilirubin pigment polymer) and calcium (calcium phosphate) salts

that are found in bile. They contain less than 20% of cholesterol (or 30%, according to the Japanese-classification system).²

Mixed stones

Mixed ("Brown pigment") stones typically contain 20–80% cholesterol (or 30–70%, according to the Japanese-classification system). Other common constituents are calcium carbonate, palmitate phosphate, bilirubin and other bile pigments (calcium bilirubin ate, calcium palmitate and calcium stearate). Because of their calcium content, they are often radiographically visible. They typically arise secondary to infection of the biliary tract which results in the release of β -glucuronides (by injured hepatocytes and bacteria) which hydrolyzes bilirubin glucuronides and increases the amount of unconjugated bilirubin in bile.

A characteristic symptom of gallstones is a "gallstone attack", in which a person may experience intense pain in the upper-right side of the abdomen, often accompanied by nausea and vomiting, that steadily increases for approximately 30 minutes to several hours. A patient may

also experience referred pain between the shoulder blades or below the right shoulder. These symptoms may resemble those of a "kidney stone attack". Often, attacks occur after a particularly fatty meal and almost always happen at night, and after drink.

Risk factors Female gender, fecundity, and a family history for gallstone disease are strongly associated with the formation of cholesterol gallstones⁵.

Obesity^{4,5} as well as other factors contributing to the metabolic syndrome⁶ such as dyslipidemia (in particular hypolipoproteinemia type IV^{7,8} with hypertriglyceridemia and low HDL cholesterol), hyperinsulinemia-insulin resistance^{9,10} or overt type 2 diabetes are risk factors for the development of gallstones, itself supposed to be a complication of the metabolic syndrome¹¹. Oestrogen- treatment enhances the risk, both in women when used for contraception or hormone-replacement¹² and in men with prostatic cancer¹³⁻¹⁴.

Among specific dietary factors, short-time high cholesterol¹⁵ as well as high-carbohydrate diets were associated with increased risk for gallstones¹⁶⁻¹⁷, and in highly prevalent areas, the intake of legume¹⁸, while unsaturated fats¹⁹, coffee²⁰, and moderate consumption of alcohol²¹⁻²² seem to reduce the risk. Also physical activity was found to decrease the risk for symptomatic gallstone disease, both for men and women²³⁻²⁴, and independent of weight reduction. On the other hand, rapid active weight loss²⁵⁻²⁶ and weight cycling^{27,28} strongly increase the risk for the development of gallstones. Thus, weight reductions should not exceed 1.5 kg per week²⁹. Fibrates, used for the treatment of dyslipidemia, interfere with cholesterol and bile acid synthesis and increase cholesterol secretion into bile³⁰⁻³¹. However, in contrast to the prototype Clofibrate, during treatment with newer fibrates only a relatively small percentage do actually develop gallstones³².

Pathophysiology

Cholesterol gallstones develop when bile contains too much cholesterol and not enough bile salts. Besides a high concentration of cholesterol, two other factors are important in causing gallstones. The first is how often and how well the gallbladder contracts; incomplete and infrequent emptying of the gallbladder may cause the bile to become over concentrated and contribute to gallstone formation. This can be caused by high resistance to the flow of bile out of the gallbladder due to the complicated internal geometry of the cystic duct.³³ The second factor is the presence of proteins in the liver and bile that either promote or inhibit cholesterol crystallization into gallstones. In addition, increased levels of the hormone estrogen, as a result of pregnancy or hormone therapy, or the use of combined (estrogen-containing) forms of hormonal contraception, may increase cholesterol levels in bile and also decrease gallbladder movement, resulting in gallstone formation.

Medical Treatment

Cholesterol gallstones can sometimes be dissolved by oral ursodeoxycholic acid, but it may be necessary for the patient to take this medication for up to two years.³⁴

Gallstones may recur, however, once the drug is stopped. Obstruction of the common bile duct with

Gallstones can sometimes be relieved by endoscopic retrograde sphincterotomy (ERS) following endoscopic retrograde cholangiopancreatography (ERCP). Gallstones can be broken up using a procedure called extracorporeal shock wave lithotripsy (often simply called "lithotripsy")³⁴, which is a method of concentrating ultrasonic shock waves onto the stones to break them into tiny pieces. They are then passed safely in the feces. However, this form of treatment is suitable only when there is a small number of gallstones.

Surgical Treatment

Cholecystectomy (gallbladder removal) has a 99% chance of eliminating the recurrence of cholelithiasis. Surgery is only indicated in symptomatic patients. The lack of a gallbladder may have no negative consequences in many people. However, there is a portion of the population — between 10 and 15% — who develop a condition called post cholecystectomy syndrome³⁵ which may cause gastrointestinal distress and persistent pain in the upper-right abdomen, as well as a 10% risk of developing chronic diarrhea.³⁶

There are two surgical options for cholecystectomy:

- Open cholecystectomy is performed via an abdominal incision (laparotomy) below the lower right ribs. Recovery typically requires 3–5 days of hospitalization, with a return to normal diet a week after release and to normal activity several weeks after release.³⁷
- Laparoscopic cholecystectomy, introduced in the 1980s,³⁸ is performed via three to four small puncture holes for a camera and instruments. Post-operative care typically includes a same-day release or a one night hospital stay, followed by a few days of home rest and pain medication.³⁹ Laparoscopic cholecystectomy patients can, in general, resume normal diet and light activity a week after release, with some decreased energy level and minor residual pain continuing for a month or two. Studies have shown that this procedure is as effective as the more invasive open cholecystectomy, provided the stones are accurately located by cholangiogram prior to the procedure so that they can all be removed.

CASE REPORT

A 36 year old female patient reported at OPD of Akhandanand Ayurveda College Ahmedabad in feb.2017 as a diagnosed case of Cholelithiasis with its full-fledged signs and symptoms. As per the patient he had developed these symptoms in past 6 months. In an attempt to get rid of these problems he consulted many renowned Allopathic doctors, but owing to no improvement in the condition the patient was advised to undergo surgery. Very much reluctant to surgery, the patient visited our hospital for a conservative treatment.

AYURVEDIC MANAGEMENT

As per the etiology and clinical presentations, Cholelithiasis is akin to *Pittashmari* described in Ayurveda. Therefore taking *Pittashmari* line of treatment

into account, the patient was switched on to following Ayurvedic medicines in this way- Mixed powder was given in three divided doses.

Table: 1 Ayurvedic medicines in three divided doses

S.No.	Ayurvedic medicines	Dose
1	Gokshurchurna(<i>Tribulus terrestris</i>)	3 gm/day
	Punarnavachurna(<i>Boerhavia diffusa lin</i>)	3 gm/day
	Pasanbheda churna(<i>saxifrage ligulata</i>)	2 gm/day
	Hazrulahuda bhasma	250 mg/day
	Kokilakshkshar	250 mg/ day
2	Tab Arogyavardhini vati	1 TDS
3	Tab Chandraprabha vati	1 TDS

DISCUSSION

According to Ayurveda, all the three Doshas viz. Vata, Pitta and Kapha play a role in formation of gallstones. Excessive increase of Pitta (caused by hot, spicy food, alcohol etc.) creates the basis for stone formation. Kapha increased by fatty, heavy foods mixes with Pitta and produces a highly sticky mixture. Vata dries this mixture and moulds it into shape of a stone. Ayurvedic treatment eliminates the need for surgery by assisting the body to expel the stones naturally.

Cholelithiasis has been compared with Pittashmari. As the name suggests, Pittashmari borrows both Pittavardhaka and ashmari producing etiological factors in its causation. Therefore management of Pittashmari. /Cholelithiasis should incorporate the medicines having properties to nullify both the factors.

Ingredients of Gokshurchurna Punarnavachurna Pasanbhedachurna-Hazrulahudabhasm Kokilakshkshar- Arogyavardhinivati have Lekhana, Chhedana, Bhedana, Mootrala Bastishodhana, Anulomana, Deepana, Paachana, Vedanaasthaapana and

Kaphashaamaka properties, so it is also helpful to dissolve/ reduce the size of Ashmari (stone). Chandraprabha vati have predominantly Pittashmaka property and thus these are responsible for inhibition of further stone formation.

CONCLUSION

In this case study, the patient has shown encouraging results during the management of Cholelithiasis (*Pittashmari*). As per the USG-abdomen, the patient has got rid of 6 to mm of gall stone within only 3 months of short duration by adopting Ayurvedic treatment. In addition, the general condition of the patient has also improved positively.

Therefore, on the basis of observations and results of this case study, it can be inferred that Ayurveda has the potential to treat cholelithiasis effectively and hence the sufferers must be advised to get benefitted from the Ayurvedic healing sciences and give active participation in national prosperity by leading enthusiastic and happy lives.

REFERENCES

- Channa, Naseem A.; Khand, Fateh D.; Khand, Tayab U.; Leghari, Mhammad H.; Memon, Allah N. "Analysis of human gallstones by Fourier Transform Infrared (FTIR)". Pakistan Journal of Medical Sciences, 2007; 23(4): 546–50.
- Kim IS, Myung SJ, Lee SS, Lee SK, Kim MH. "Classification and nomenclature of gallstones revisited". Yonsei Medical Journal, 2003; 44(4): 561–70.
- Attili AF, Capocaccia R, Carulli N et al. Factors associated with gallstone disease in the MICOL experience. Multi center Italian Study on Epidemiology of Cholelithiasis. Hepatology 1997; 26: 809–18.
- 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
- Amaral JF, Thompson WR. Gallbladder disease in the morbidly obese. Am J Surg 1985; 149: 551–7.
- Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005; 365: 1415–28.
- Ahlberg J, Angelin B, Einarsson K, Hellstrom K, Leijd B. Prevalence of gallbladder disease in hyperlipoproteinemia. Dig Dis Sci 1979; 24: 459–64
- Einarsson K, Hellstrom K, Kallner M. Gallbladder disease in hyperlipoproteinaemia. Lancet 1975; 1: 484–7.
- 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 hyper lipoproteinemia. J Clin Invest 1974; 54: 1301–11.
- Nervi F, Miquel JF, Alvarez M et al. Gallbladder disease is associated with insulin resistance in a high risk Hispanic population. JHepatol 2006; 45: 299–305.
- Scragg RK, Calvert GD, Oliver JR. Plasma lipids and insulin in gall stone disease: a case-control study. Br Med J (Clin Res Ed) 1984; 289: 521–5.
- Grundy SM. Cholesterol gallstones: a fellow traveler with metabolic syndrome? Am J Clin Nutr 2004; 80: 1–2
- Scragg RK, McMichael AJ, Seamark RF. Oral contraceptives, pregnancy, and endogenous estrogen in gall stone 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 gall stone 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.
33. Experimental investigation of the flow of bile in patient specific cystic duct models M Al-Atabi, SB Chin... - *Journal of biomechanical engineering* 2010.
34. National Health Service. "Gallstones—Treatment". NHS Choices: Health A-Z - Conditions and treatments. London: National Health Service, 2010.
35. Jensen. "Postcholecystectomy syndrome". Omaha, Nebraska: Medscape (WebMD), 2010.
36. Marks, Janet; Shuster, Sam; Watson, A. J. "Small-bowel changes in dermatitis herpetiformis". *The Lancet*, 1966; 288(7476): 1280–2.
37. Database of Systematic Reviews (4): CD006231. National Institute of Diabetes and Digestive and Kidney Diseases 2007. "Gallstones". Bethesda, Maryland: National Digestive Diseases Information Clearinghouse, National Institutes of Health, United States Department of Health and Human Services. Retrieved 2011.
38. Keus, Frederik; de Jong, Jeroen; Gooszen, H G; Laarhoven, C JHM; Keus, Frederik. "Laparoscopic versus open cholecystectomy for patients with symptomatic cholelithiasis". *Cochrane* 2006