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A Comprehensive Approach to *Pittasaya Asmari* w.s.r Cholelithiasis

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ABSTRACT

Cholelithiasis (Gall stone) refers to crystalline concretions of the bile components formed within the gall bladder or bile ducts. These are either cholesterol stones to account for 80% of the total and pigment stones comparing the remaining 20% Cholesterol gallstones usually contain less than 50% Cholesterol monohydrate with a mixture of bile pigments, proteins, fatty acids, and calcium salts.

Pigment stones are composed primarily of Calcium bilirubinate, and they contain less than 20% cholesterol. Cholelithiasis is often asymptomatic but can present with infrequent episodes of severe pain in epigastrium or in right upper quadrant with radiation to right scapula. Stones in common bile duct (Choledocholithiasis) also present with similar pain associated with Jaundice, apart from the systematic disturbance characterized by fever, vomiting, nausea, loss of appetite.

Ayurvedic classics do not consider Cholelithiasis as a separate disease, but the features in cholelithiasis can be compared with various diseases like *Pittaja udara shoola, yakrithadalyodara, shakhashrita kamala, Pittaja Gulma*. Cholelithiasis is primarily managed by surgery, but there is a demand to manage this disease with safe and effective *Ayurvedic* treatments. In this review article, various aspects of cholelithiasis are discussed through the format of *Ayurveda*.

Key Words *Cholelithiasis, Pittashaya shmari, Paneeya Kshara*

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INTRODUCTION

Gallstones are formed by the abnormal concretion of bile constituents within the gall bladder or the bile ducts. Gallstone disease occurs due to several reasons, which include the super-saturation of bile with Cholesterol, nucleation of cholesterol monohydrate with subsequent crystal retention resulting in stone growth and abnormal gallbladder motor function with delayed emptying or stasis of bile[1].

In *Ayurveda*, clinical manifestations explained in *Pittaja udarashoola, yakrithalyodara*, and *shakhashrita kamala* represent different hepato-biliary diseases including cholecystitis, but there lies hardly any direct references of a disease which can be correlated to cholelithiasis. While explaining the *samprapti* of *moothrashmari*, *Vagbhata* says that *ashmari* is formed in the *Vasti* similar to the formation of *rochana* in the *pittashaya* of a cow[2]. Hence, the *ashmari* (stones) formed in *Pittashaya* of humans resembling the -

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"*go*"- *rochana* can be considered as *pittashaya ashmari*.

Gall bladder is not described as a visceral organ in *Ayurvedic* texts. But there are various references which almost certainly indicate that gallbladder can be considered as *'kloma"*. *Acharya Dalhana*, in his commentary on *Sushruta Samhita* described *kloma* as *tila* like structure situated in the right side of the abdomen below the liver *(yakrut)*[3] very much similar to the gall bladder. It can be inferred from the facts, that the impression of *pittashaya* (gall bladder) is already there in *Ayurveda* and morphologically it is described in association with the liver. This may be the reason why *kloma* is not discussed in detail. Again, descriptions of *ashmari* or other diseases pertaining to *pittashaya* or *pittakosha* are not available in the classical *samhitas* of *Ayurveda*.

Cholelithiasis in *Ayurveda*

In *Ayurveda*, bile can be considered as *malarupi pitta*, having predominance of *pachaka* and *ranjaka pitta*. *Pachaka pitta* does digestion of the *Ahara* and due to *snehana* (oleating) and *Ranjana* (colouring) *gunas* of *ranjaka pitta*, *pureesha* gets the colour and lubrication.

Nidana* (Causative factors) for *pittashaya ashmari

The causative factors which increase *kapha* such as, *avyayama* (not having exercise), *divaswapna* (day sleep), *snigdha, madhura, picchila, guru ahara sevana* (intake of heavy,slimy,sweet,unctuous food substances) intake of *dadhi*{curd),*ghrita* (ghee), *mamsa* (meat), *tila* (sessame), *abishyandi ahara sevana* (intake of unctuous food), *adhyashana* (over eating), *samashana, pittakara nidana* such as *upavasa* (fasting), *katu, amla, lavana ahara sevana* (intake of spicy,sour,salty food).

The *Samprapti* (Pathogenesis) of *Pittashaya ashmari*

The components involved in the pathogenesis of *asmari* are

Srotas-Rasavaha .Raktavaha , Annavaha

Dosha-Kapha,Pitta

Dushya Rasa, meda

Agni-Jataragni

Sroto Dushti-Sanga

Udbhavastana- Amashaya

Sancharasthana-koshta

Adhishtana-Pittashya

Vyaktasthana-Pittashaya

There are no specific clinical manifestations for *pittashaya ashmari*. But the *lakshanas* explained in *haleemaka, pittaja udara shoola, pittodara* can be considered as complications occurred due to the presence of gall stone. Apart from that, the *lakshanas* explained for *vata* associated with *aama-* (*Vibandha*-constipation), *Agnisaada* (reduced appetite), *Aantrakoojana* (borborgysm), *Angavedana* (body ache), *Angashotha* (oedema), *Thoda* (pricking pain), *Angagraha* (body pain), *Gourava* (heaviness), *Arochaka* [4]

For *aama* associated with *kapha (Udgarabhaya, Pralepatwa, Picchilatwa, kshudhanasha)*[5], *Rasa pradoshaja vikara (ashrddha, Aruchi, Asya vairasya, Arasanjnata, trupti, Hrillasa, Gourava, tandra, Angamarda, Jwara, Pandutwa, Srotasam*

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rodha, Angasaada, Agninaasha)[6] also are very much similar with symptoms of *pittashaya asmari*. *Parshwashoola* explained by *Bhava prakasha* is similar to that of cholelithiasis[7].

Treatement Protocol in *Ayurveda*

Gallstone can be treated according to the line of treatment of *ashmari* explained by *Susrutha*[8] considering it as *pithashayagatha ashmari*, treatment procedures are:

- *Snehana*- for lubrication, smoothening and protecting the membranes
- *Kshara*- for dissolution of ashmari
- *kashaya*- for easy lavage of stones.

In this aspect, as far as cholelithiasis is concerned early *snehapaana* may hinder treatment progress hence *Aama chikitsa* or *chikitsa* for *Rasa pradushti* should be done, which include *deepana, pachana* with *trikatu* and *triphala, arogyavardhini vati, katuki churna* must be done. *Snehana* can be attained by using *Pippalyadi ghrutha, indukantha ghrutha, danti ghrutha ,shatpala ghrutha, varunadi ghrutha*.

For *kshara karma, Paneeya ksharas* like *Apamarga kshara, yava kshara, palasha kshara, surya kshara, kalyanaka kshara, shivakshara, pachana churna,* along with *narikela lavana* may be used. *Bhasmas* like, *Tamra bhasma, Annabhedi sindhura, kaaseesa bhasma* can be prescribed.

As *Kashaya, Pitta rechaka kashayas* like *Patola katurohinyadi, Chirivilvadi kashaya chitrakagranthyadi Kashaya* etc may be used as per the conditions. Once gall stone is cleared the integrity, metabolism in the gall bladder and liver is maintained by the usage of *Kashaya*.

Asavas- Ayaskriti, Kalameghasava,Bhringarajasava, Dantyarishta, Patolasava, Roheetakarishta, Nimbamrutasava may be prescribed. *Vati - Shivagutika* may be prescribed.

Shakashritha kamala[9]

In some cases when obstruction happens with manifestation of jaundice, white or clay coloured stools *(tila pishta nibha)* are formed. Then *Kaphahara chikitsa* till normal *purisha* colour attains has to be done. For this *kapha hara* and *Purcesha virajaneeya* drugs are to be used.

Cholelithiasis

Predisposing factors for Cholesterol and Pigment Gallstone formation can be summarised as follows:

Cholesterol Stones

1. Demographic: Prevalence is highest in North American Indians, Chilean Indians, and Chilean Hispanics, greater in Northern Europe and North America than in Asia, lowest in Japan.

2. Obesity: Normal bile acid pool and secretion but increased biliary secretion of cholesterol. 3. Weight loss: Mobilization of tissue cholesterol leads to increased biliary cholesterol secretion while enteric-hepatic circulation of bile acids is decreased.

4. Female sex hormones

a) Estrogens stimulate hepatic lipoprotein receptors, increase uptake of dietary cholesterol, and increase biliary cholesterol secretion.

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b) Natural estrogens, other estrogens, and oral contraceptives lead to decreased bile secretion.

5. Increasing age: Increased biliary secretion of cholesterol, decreased size of bile acid pool, decreased secretion of bile salts occurs as age increases.

6. Gallbladder hypomotility leading to stasis and formation of sludge.

a. Prolonged parenteral nutrition

b. Fasting c. Pregnancy.

7. Decreased bile acid secretion.

a. Primary biliary cirrhosis:

b. Genetic defect of the CYP7A1 gene.

8. Decreased phospholipid secretion:

a. Genetic defect of the MDR3 gene.

9. Miscellaneous.

a. High-calorie, high-fat diet.

b. Spinal cord injury

Pigment Stones

1) Demographic: Asia, rural setting

2) Chronic haemolysis.

3) Alcoholic cirrhosis.

4) Pernicious anaemia

5) Cystic fibrosis

6) Chronic biliary tract infection, parasite infections

7) Increasing age

Factors associated with gall stone formation

Impaired functioning of the gallbladder in emptying, absorption and excretion of the bile favours biliary concretions. Similarly, excess of cholesterol nucleating factors like mucus, glycoprotein or infections also increases the chances of developing stones. Any other reason that results in super saturation of bile is an accepted event in gall stone formation.

Types of gallstone (Incidence)[10]

a. Pure Stone (10%)

- Cholesterol stones
- Pigment Stones
- Calcium Carbonate stones.

b. Mixed Stone (90%)

Pathogenesis of Cholesterol Stones[11]

Cholesterol is soluble in bile by aggregation with water-soluble bile salts and water-insoluble lecithins, both of which act as detergents. When cholesterol concentrations exceed the solubilising capacity of bile (supersaturation), cholesterol can no longer remain dispersed and nucleates into solid cholesterol monohydrate crystals. Cholesterol gallstone formation involves four simultaneous defects.

- Bile must be supersaturated with cholesterol.
- Hypomotility of gall bladder to promote nucleation.
- Accelerated cholesterol nucleation in bile
- Mucus hyper-secretion in the gall bladder to traps the crystals, permitting their aggregation into stones.

Supersaturation of bile with cholesterol is the result of hepatocellular hypersecretion of cholesterol, mediated by the abnormal regulation of hepatic mechanisms for delivering cholesterol to bile. The abundant free cholesterol is toxic to the gallbladder, penetrating the wall and exceeding the ability of the mucosa to Gall bladder hypomotility ensues by then. It appears

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to result both from intrinsic neuromuscular dysmotility and from the diminished muscular responsiveness to cholecystokinin, the hormone secreted by the gut that promotes gallbladder contraction.

Pathogenesis of Pigment and Calcium Stones[12]

Pigment gallstones are complex mixtures of abnormal insoluble calcium salts of unconjugated bilirubin along with inorganic calcium salts. Unconjugated bilirubin is normally a minor component of bile but increases when infection of the biliary tract leads to release of microbial b-glucuronidases, which hydrolyse bilirubin glucuronides. Infection of the biliary tract occurs with Escherichia coli or Ascaris lumbricoides or by opisthorchis sinensis.

Clinical manifestation

Two thirds of gall stones are asymptomatic. Stones may cause acute or chronic cholecystitis, biliary colic, pancreatitis or obstructive jaundice. The most common symptom is intermittent pain below the right costal margin. Pain may radiate the back and to the scapula. Nausea, with or without vomiting and dyspepsia may occur. The patient may have episodes of acute abdominal pain called as biliary colic.

DISCUSSION

Cholelithiasis (gall stone) is a crystalline concretion formed within the gall bladder by accretion of bile components. Gallstone disease is identified as a multi-factorial disease. The risk factors include, obesity, rapid weight loss, high dietary glycaemic load, glucose intolerance, insulin resistance alcohol abuse, diabetes mellitus, hyper-triglyceridemia, certain drugs and pregnancy.

In similar fashion due to various *nidana* (causative factors) especially *kaphakara aharavihara*, there will be *pachakagnimandya* causing *aama* and also *kapha vriddhi* (Hyper secretion of cholesterol in the bile impairing bile acid and cholesterol ratio) and *karmahani* (Hypomotility of the gallbladder causing stasis and impaired function of cholecystokinin *(samana vata vikriti)* which in turn causes formation of *ashmari* in *pittashaya. Acharya Chakrapani* while commenting on *shakhashritha kamala*, says that *kamala* is produced due to obstruction of *shakhashritha pitta* entering to *koshtha* by *shleshma*, which can be correlated to the pathogenesis of gallstones obstructing biliary tract. Above pathogenesis can be appreciated in *Ashayapakarshaka gati* of *vayu* which brings *kapha* to normal *pittasthana* thus forming *avarodha* of *pitta* by *kapha*. *Ranjakagni mandya* and concurrent *Kaphaprakopa* renders *Kaphajakrimi* and formation of Pus which solidifies into *ashmari*. This can be correlated to formation of pigmented stones.

The most dreaded complication of asymptomatic gall stone is carcinoma of gall bladder. To prevent such complications, early management of gall stones is necessary. Cholesterol gall stones can be dissolved by oral Ursodeoxycholic acid, but it may be required that the patient takes this medication for 2 years and there is high

recurrence rate[13]. Gall stones can be broken up using a procedure called the Lithotripsy, however this form of treatment is suitable only when there is a small number of gall stones. Cholecystectomy, the surgical removal of gall bladder is the standard procedure to treat cholelithiasis. However, approximately 10-15% of population may develop a condition called Post cholecystectomy syndrome, which includes gastrointestinal distress and persistent pain in upper right abdomen, as well as 10% chance of developing chronic diarrhoea due to lack of gall bladder. So, there is a scope for *Ayurveda* in the arena of non surgical management of Cholelithiasis. *Ayurvedic* management includes *trividha chikitsa i) Nidana parivarjana ii) Apakarshana-snehana, ksharana, bhedana,* periodic *shodhana* and iii) *Prakruti vighata-kaphahara, Pitta hara*.

CONCLUSION

Cholelithiasis (gall stones) refers to crystalline concretions of bile components, cholesterol formed within the gall bladder or bile ducts. *Ayurvedic* classics do not project Cholelithiasis as a separate disease entity but the spectrum of features in cholelithiasis can be outlined among the various diseases mentioned like *Pittaja udara shoola, yakrithdalvodara, shakhashrita kamala, Pittaja Gulma.* In this aspect cholelithiasis even though exclusively managed with surgical assistance creates different problems post operatively in terms of metabolic disturbances. Hence *Ayurvedic* principles of management which mainly targets on the metabolic correction has to be applied. for better patient compliance.

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