A CASE STUDY ON AYURVEDIC MANAGEMENT OF MIRIZZI SYNDROME.

Rahul Ravi P¹, Mythrey. R.C ²

¹- Final year PG Scholar, Department of PG Studies in Kayachikitsa, Government Ayurveda Medical College, Mysuru, Karnataka, India.
²- Professor and HOD, Department of PG Studies in Kayachikitsa, Government Ayurveda Medical College, Mysuru, Karnataka, India

ABSTRACT

Mirizzi syndrome is an unusual presentation of gallstones occurring when a calculus gets impacted in either Hartmann’s pouch of the gallbladder or the cystic duct and causes obstruction of the common hepatic duct by extrinsic compression.¹

The serious surgical complications such as bile duct injury encountered during laparoscopic cholecystectomy makes Mirizzi syndrome a potentially dangerous manifestation of impacted gallstones.²

The pathophysiology of Mirizzi syndrome involves extrinsic compression of the bile duct by pressure applied upon it indirectly by an impacted stone in the infundibulum or neck of the gallbladder. In turn, the resulting chronic inflammation and ulceration form varying degrees of cholecysto-biliary fistula. Furthermore, cholecysto-enteric fistula may also occur.³

Only 0.1% of patients with gallstones will develop this condition and women of reproductive age or on birth control estrogen medication have a 2-fold increase in gallstone formation compared to males.³

In Ayurveda, this symptomatology can be understood as an advanced stage of Kamala which is complicated by Pittashmari⁴. Here is a single case study of a 52-year-old female presented with right hypochondrial pain, jaundice and vomiting. The condition was symptomatically well managed by Shamana aushadhi mentioned in the context of Kamala, Medo roga and Ashmari.

Keywords: Mirizzi, gallstones, Kamala, Ashmari

INTRODUCTION

Mirizzi syndrome is a rare condition caused by the obstruction of the common bile duct or common hepatic duct by external compression from multiple impacted gallstones or a single large impacted gallstone in Hartman's pouch. Presenting symptoms are similar to cholecystitis but may be confused with other obstructing conditions such as common bile duct stones and ascending cholangitis due to the presence of jaundice.

Mirizzi syndrome is relatively uncommon and only 0.1% of patients with gallstones end up developing this condition. The prevalence increases as the person ages. Obesity increases the likelihood of gallstones, especially in women, due to increase in the biliary secretion of cholesterol. On the other hand, patients with drastic weight loss or fasting have a higher chance of gallstones secondary to biliary stasis. Furthermore, there
is also a hormonal association with gallstones. Estrogen has been shown to result in an increase in bile cholesterol and a decrease in gallbladder contractility.

The clinical presentation of Mirizzi syndrome ranges from asymptomatic to non-specific, with obstructive jaundice (27.8–100%) being the most common, elevated liver enzymes (AST/ALT), right upper quadrant abdominal pain (16.7–100%), and constitutional symptoms such as fever, nausea, vomiting, diarrhoea, and constipation. Although rare, Mirizzi syndrome may also present with gallstone ileus.

The pathophysiological process leading to the subtypes or stages of Mirizzi syndrome, has been explained as an inflammatory phenomenon secondary to a pressure ulcer caused by an impacted gallstone at the gallbladder infundibulum. The impacted gallstone together with the inflammatory response, causes first external obstruction of the bile duct, and eventually erodes into the bile duct evolving into a cholecysto-choledochal or cholecysto-hepatic fistula with different degrees of communication between the gallbladder and bile duct. The importance and implications of this condition are related to their associated, and potentially serious, surgical complications such as bile duct injury (0%-22%) especially with laparoscopic cholecystectomy.5

There was a 52-year-old female presenting with right hypochondrial pain, jaundice and vomiting. The case was diagnosed as Mirizzi syndrome on the basis of ultrasonography. Usually, such conditions are referred for surgical management which was here refused by the patient. The condition was very well managed by Ayurvedic Shamana aushadhi mentioned under the treatment principles of Kamala, Medo roga and Ashmari.

METHODOLOGY

CASE REPORT

Personal history

Female patient aged 52 presented with right upper quadrant abdominal pain and constitutional symptoms such as fever, nausea and vomiting. Elevated liver enzymes (AST/ALT) and CT findings were suggestive of obstructive jaundice. The complaints were persisting from 1 month at the time of first consultation(10/5/22).

Observations

Built- Obese
Condition- Apathic

GENERAL EXAMINATION:

<table>
<thead>
<tr>
<th>AYURVEDIC EXAMINATION</th>
<th>GENERAL EXAMINATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prakruti-kapha pitta</td>
<td>Palour- absent</td>
</tr>
<tr>
<td>Nadi- Manda gati, kaphaja</td>
<td>Icterus- Present</td>
</tr>
<tr>
<td>Mala- Badha mala ,Prakruta varna</td>
<td>Clubbing- Present</td>
</tr>
<tr>
<td>Mutra- Peeta varna</td>
<td>Lymphedenoathy- absent</td>
</tr>
<tr>
<td>Jihwa- Lipta</td>
<td></td>
</tr>
<tr>
<td>Shabdhya- prakrutha</td>
<td></td>
</tr>
<tr>
<td>Sparsha- prakrutha</td>
<td></td>
</tr>
<tr>
<td>Druk- Peeta varna</td>
<td></td>
</tr>
<tr>
<td>Akruthi- Sthula</td>
<td></td>
</tr>
<tr>
<td>Sara, Satwa, samhanana- Madhyama</td>
<td></td>
</tr>
<tr>
<td>Aharashakthi- Avara</td>
<td></td>
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<tr>
<td>Vyayamashakthi- Avara</td>
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</tbody>
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Symptoms

Fever, vomiting, right hypochondrial pain, nausea and tiredness.
CT abdomen and pelvis reports as on 14/1/22
Ct suggestive of focal cholecystitis with sludge
Dilated intra and extra hepatic biliary radicals with choledocholithiasis and sludge
Mild hepatomegaly with bilaterally dilated hepatic ducts

USG abdomen and pelvis reports as on 12/5/22
Dilated CBD and IHBR with solitary cystic duct calculus-suggestive of Mirizzi syndrome
Mild Hepatomegaly with Fatty liver Grade 1

USG abdomen and pelvis reports as on 12/7/22
Mild IHBR dilatation in both lobes of liver.
Multiple calculi noted in Gall bladder largest 12 mm
CBD -10mm, few small calculi noted in mid CBD

Lab investigations reports as on 12/5/22
Complete blood count and differential count were within normal limits from the first consultation itself.

Comparative reports of LFT findings are as given below

<table>
<thead>
<tr>
<th>Liver function test</th>
<th>Before intervention (12/5/22)</th>
<th>After intervention (12/7/22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct bilirubin</td>
<td>8.59 mg/dl</td>
<td>0.26 mg/dl</td>
</tr>
<tr>
<td>Indirect bilirubin</td>
<td>0.11 mg/dl</td>
<td>0.11 mg/dl</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>8.70 mg/dl</td>
<td>0.37 mg/dl</td>
</tr>
<tr>
<td>SGOT</td>
<td>54 U/L</td>
<td>19 U/L</td>
</tr>
<tr>
<td>SGPT</td>
<td>49 U/L</td>
<td>38/U/L</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>368 IU/L</td>
<td>325 IU/L</td>
</tr>
<tr>
<td>S. Albumin</td>
<td>3.2g/dl</td>
<td>3.9 g/dl</td>
</tr>
<tr>
<td>S. Globulin</td>
<td>3.6g/dl</td>
<td>3.8 g/dl</td>
</tr>
<tr>
<td>Urine Bile salt/pigments</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>ESR</td>
<td>75 mm/hr</td>
<td>52 mm/hr</td>
</tr>
</tbody>
</table>

Treatment
Since the patient refused to undergo surgical intervention for Mirizzi syndrome, she was prescribed with following Shamana aushadhi.

1. Vasaguluchyadi Kashaya $^6$ - 30 ml BID Before food
2. Arogya vardhini vati $^1$ - 1 TID After food
3. Laghu sutasekhara rasa $^2$ - 1 BID Before food
4. Gokshuradi guggulu - 1 BID After food
5. Cap.Pithorin - 1 BID After food

Along with the medications, strict Pathya was also instructed. It included avoiding the usage of oily, fried food items and all types of dairy and non-vegetarian products. Fat free diet with plenty of water intake was advised.

OBSERVATIONS AND RESULTS
Symptomatic relief was noticed on post intervention consultation. There was no pain abdomen, further episodes of vomiting and fever were also not noted. Jaundice signs like icterus and yellowish discolouration of urine also reduced. There was noticeable increase in appetite and general condition of the patient. Post-treatment LFT reports were suggesting dramatic improvement in functional aspects of liver with bilirubin and liver enzymes coming back to normal range except alkaline phosphatase level. However, post study USG scan does not show any significant improvement in the status of hepato-biliary system except for fatty liver
and hepatomegaly changes. On follow up assessment after 30 days, condition of the patient was maintained as the same with no further episodes of fever, vomiting and abdominal pain.

**DISCUSSION**

Intervention selected for the current case included drugs which are either *Yakrut poshaka*, *Pitta shamana* or *Ashmari nashana* in action. *Vasa guluchyadi yoga* is known for its action in *Kamala* by virtue of its *Pitta rechaka* effect. Ingredients such as *Vasa, Guduchi, Triphala* have *Pitta shleshmahara* property and drugs such as *Boonimba* and *Nimba* works at the level of *Yakrut* and *Pittashaya*.

*Arogya vardhini* is a renowned hepato-protective drug that also gives *Rechaka* effect because of ingredients like *Katuki* and *Chitraka*. Hence it would be helpful to overcome the *avarana* effect of *Kapha*. *Laghu sutasekharana* is one of the best *Pitta shamana dravya* available which works without hampering *Jatara agni*. *Gokshuradi guggulu* and Cap.*Pithorin* were included with the view to disintegrate the *Pitta ashmari*. Along with the intervention, lifestyle modification including the *Pathya* would have reduced the work load on liver and gallbladder, thereby improving the condition of the patient.

**CONCLUSION**

Conditions like Mirizzi Syndrome which are best treated with surgical interventions cannot be cured completely through *Shamana aushadhi*. But for patients who are not willing to undergo surgery or contra indicated for the same can be very well managed symptomatically through ayurvedic interventions. *Yakrit uttechaka*, *Pitta rechaka* and *Ashmari bedhana* actions of the selected interventions proved to be effective in the current clinical condition.

**REFERENCE**


