

## Case Report

# Different outcomes of spontaneous gall bladder perforation owing to timing of surgical intervention: report of 2 cases

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## ABSTRACT

Gall bladder perforation (GBP) is a rare life-threatening complication with a high mortality due to atypical clinical presentation and delay in diagnosis due to atypical clinical presentation. Case report 1, 70-year-old female presented with complaints of pain abdomen, vomiting and blackish pigmentation over abdomen since a week. She was in shock on presentation, with necrotic patch over abdomen and abscess collection at umbilicus. Ultrasound revealed GBP at fundus with thick collection in gastrohepatic recess with overlying abdominal wall cellulitis. CECT was not possible and the patient could not be taken for surgery due to unstable vitals and expired the next day. Case report 2, a 68-year-old male presented with complaints of pain abdomen over right side with vomiting. Abdomen was tender diffusely with guarding in right hypochondrium. Ultrasound showed distended gall bladder (GB) with multiple calculi. Magnetic resonance cholangiopancreatography (MRCP) showed a GBP at fundus with pericholecystic collection extending into hepatogastric recess. He underwent total cholecystectomy and post-operative period was uneventful. Acute cholecystitis has a perforation rate of 2-11% due to cystic duct obstruction, ischemia and necrosis. Our first patient had type-III GBP and was in shock and did not survive due to bad condition on arrival whereas the second patient had type-I GBP and underwent cholecystectomy without any complications. Type-I and type-II GBP as proposed by Niemeier have better outcomes compared to type-III. Rapid diagnosis and surgical intervention are very much necessary for reducing mortality as they rarely present with typical signs and symptoms of perforation.

**Keywords:** Gall bladder, Perforation, Cholecystitis, Niemeier classification

## INTRODUCTION

Gall bladder perforation (GBP) is a rare but life-threatening complication of Acute cholecystitis with surgeons in dilemma due to atypical presentations.<sup>1</sup> It has a reported mortality rate of 12-42% requiring immediate surgical intervention.<sup>1</sup> This high rate may be due to delay in the diagnosis as they lack typical clinical presentation and radiological characteristics. CECT Abdomen and Ultrasound abdomen may help in the preoperative diagnosis but most perforations of gall bladder (GB) are diagnosed during surgery resulting in significant

morbidity and mortality with significant impact on outcomes.<sup>2</sup>

The mortality among the patients of GBP secondary to acalculus cholecystitis is more compared to calculus cholecystitis owing to severe infection and intense inflammation with non-obstructive cholecystitis. This along with pre-existing immune compromised state leads to thrombosis of blood vessels and trans-luminal necrosis and perforation.<sup>1,2</sup>

Clinically the patients may not give symptoms of biliary colic rather symptoms mimic those of hollow viscus

perforation which when ruled out due to absence of gas under the diaphragm in plain erect abdominal radiogram makes the definitive diagnosis difficult leading to other investigations like ultrasound abdomen and CECT abdomen causing delayed diagnosis and poor outcomes.<sup>2</sup> This study reports 2 interesting cases of GB perforation with interesting outcomes.

### CASE REPORT 1

A 70-year-old female, non-hypertensive, non-diabetic presented to the emergency with complaints of pain abdomen for 1 week and complaining of swelling and pigmentation over the abdomen for 5 days. She also had history of vomiting and loose stools for 1 week. Pain was of throbbing type over the upper abdomen more towards to the right and radiating to the entire abdomen. History of fever present since a week. No history of previous complaints in past, no history of previous surgeries or tobacco chewing or drug abuse or alcoholism.



**Figure 1: Abdominal wall cellulitis with necrosis.**



**Figure 2: Abscess collecting at the umbilicus imminent for rupture.**

On examination her general condition was poor, she was tachycardic, hypotensive (70/50mmHg), febrile (102°F) and drowsy. Abdominal examination revealed anterior abdominal wall cellulitis with hyperpigmentation and abscess formation (Figure 1). Local rise of temperature

was present at the umbilicus (Figure 2). There was diffuse tenderness and guarding all over the abdomen. Her blood work revealed that she was severely anemic (Hb-4g%, reticulocyte 4.5%), markedly elevated leucocyte count (41000/cumm). Her creatinine was 2.6mg/dl and urea-68.7mg/dl. Her albumin was 2.2g/dl and rest of LFT was normal. LDH was raised to 286U/L. Stool was negative for occult blood.

Ultrasound abdomen revealed thickened GB wall with thinned out wall and suspicious defect of 3mm at the fundal region s/o GBP with thick collection of size 7.5x3.2x6.3 in gastrohepatic recess s/o abscess communicating with the subcutaneous tissue with overlying abdominal wall cellulitis s/o type III GBP (Figure 3). CECT could not be done due to the poor general condition and unstable vitals of the patient (Figure 4).

Her BP was falling below 60/40mmHg and she was put on inotropes and higher antibiotics with adequate fluids and analgesics. Her urine output was low and she developed MODS and septic shock. Her vitals deteriorated overnight inspite of inotropic support. She could not be taken for surgery due to unstable vitals and expired the next morning.



**Figure 3: Gall bladder perforation with pericholecystic collection.**



**Figure 4: Collection communicating with subcutaneous tissue forming a fistula.**

## CASE REPORT 2

A 68-year-old male diabetic and non-hypertensive patient presented to the emergency with acute severe pain over the upper abdomen since previous night along with nausea and vomiting. Pain was of continuous in nature more towards the right side of the abdomen and radiating to the rest of the abdomen and to the back. He had no history of fever or abdominal distension. He gave history of similar complaints in the past 6 months back for which he took conservative treatment and was relieved of the pain. He had no history of drug abuse, alcoholism or tobacco chewing or any previous abdominal surgery.

On examination he was normotensive, afebrile, tachycardic, tachypnic and deeply jaundiced. On per abdomen examination there was no distension or any pigmentation. Tenderness was present over the epigastrium, right hypochondrium and right lumbar region with overlying guarding and good bowel sounds. He had elevated Leukocyte count (27200/cumm), markedly raised serum bilirubin (Total-8.9mg/dl, Direct-6.9mg/dl) and elevated ALP, SGOT and SGPT levels. His RFT was within normal limits with high Random Blood sugar levels.

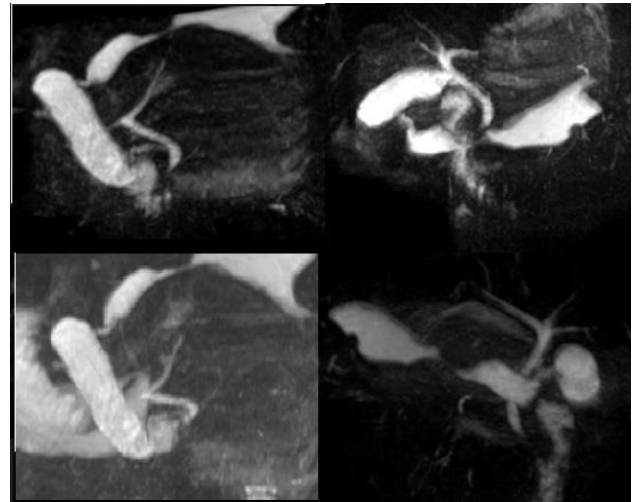
Ultrasound Abdomen showed well distended GB with multiple calculi, largest 8x6.5mm with thickened wall measuring 4mm (Figure 5). Patient was administered third generation cephalosporins and adequate analgesics and fluids keeping the patient nil by mouth. By the evening of admission, the pain suddenly reduced and patient was comfortable with mild pain abdomen over right side. Suspecting a CBD stone, MRCP was done which revealed a defect of 2.6mm in fundus of GB with pericholecystic collection extending into hepatogastric region s/o type I GBP (Figure 6).



**Figure 5: Distended gall bladder with multiple calculi.**

Patient was administered parenteral antibiotics (meropenem) and was taken for exploratory laparotomy which revealed a perforation over the fundus of GB with bilious peritoneal collection and spilled gall stones. Total

cholecystectomy was done and abdomen closed after giving peritoneal lavage and placing a drain in subhepatic region. Post-operative period was uneventful. Parenteral antibiotics given for 7 days and converted into oral antibiotics for 7 more days. Postoperative LFT was normal within 3 days with low bilirubin and liver enzymes. Oral feed was started on 3rd postoperative day and drain was removed on 7th postoperative day and was discharged. Follow up was uneventful with no complications.



**Figure 6: MRCP showing gall bladder perforation with free peritoneal spill.**

## DISCUSSION

Gall bladder perforation was first reported in by Duncan in 1844.<sup>3</sup> Asymptomatic cholelithiasis affects up to 10% of the adult population in wealthy nations among whom 2% develop Acute cholecystitis out of which 2-11% have GBP.<sup>4</sup> As the gall bladder has a dual blood supply, though acute cholecystitis is common, avascular necrosis and gangrene with perforation is relatively uncommon.<sup>5</sup>

Acute uncomplicated cholecystitis is more common in females whereas spontaneous GB perforation is more common in males.<sup>6,16</sup> Commonest age being 48-60 years.<sup>2</sup> GBP results most commonly due to persistent cystic duct occlusion by an impacted calculus causing a rise in intracholecystic pressure, epithelial injury, release of phospholipases, degradation of cell membranes and intense inflammatory reaction.<sup>5</sup> There are many predisposing factors for GBP like infections, malignancy, trauma, drugs, old age, corticosteroid therapy and systemic diseases like atherosclerosis, diabetes mellitus and immunocompromised state. The most probable mechanisms for GBP are- 1) cystic duct obstruction due to stone causing GB distension and systemic dehydration leading to bile concentration 2) impairment of blood supply due to GB distension and underlying systemic illness like atherosclerosis or localised vasospasm 3) ischaemia and necrosis leading to perforation of the GB



wall.<sup>7</sup> GBP can occur as early as 24 hours or after a few days to weeks after the onset of acute cholecystitis.<sup>17</sup>

Estevao-Costa proposed a classification for causes of GBP<sup>8</sup> as follows in (Table 1).

Causes of GB perforation	
Spontaneous	a. Idiopathic
	b. Secondary
	i. Lithiasis
	ii. Inflammation/Infection (predisposing factors- atherosclerosis, diabetes, malignancy, immune-suppression iii. Other (Congenital obstruction, <i>Salmonella typhi</i> , anticoagulants)
Traumatic	a. Penetrating b. Blunt
Iatrogenic	-

GBP most commonly occurs at the fundus of the GB attributing the vascular route as the most important cause as fundus is the most distal part with regards to blood supply.<sup>9</sup> Next is the body and the least is the infundibulum and neck. A sudden decrease in pain intensity may occur due to perforation which causes relief of high intracholecystic pressure.<sup>5</sup>

Ultrasound features include wall thickening (>3mm), distension, presence of gallstones, coarse intracholecystic echogenic debris, bile duct dilation and defect in the wall appearing as 'Hole sign'. Distension of GB with oedema of its wall may be the earliest sign of imminent perforation.<sup>10</sup> 'Hole sign' is the most specific finding in abdominal CT. MRCP is superior due to its ability in detecting CBD stones, biliary dilatation and in presenting the relation of the pericholecystic collection between the abdominal wall and the gall bladder.<sup>10</sup> Elevated liver enzymes, especially alkaline phosphatase levels along with rise of bilirubin levels are commonly observed.<sup>5</sup>

Niemeier in proposed a classification of GBP which was later modified by Fletcher and Ravdin in and Roslyn 1951 and Busuttil 1979 in as follows - type I, Acute free perforation into the peritoneal cavity; type II, subacute perforation with pericholecystic abscess formation and type III, chronic perforation with a cholecysto-cutaneous or cholecysto-enteric fistula formation. Anderson and Nazem in 1987 reported a new type of fistula and labelled it as type IV, chronic perforation with cholecysto-biliary fistula formation.<sup>11-14</sup>

As seen in case 1 the patient came with GBP with Cholecysto-cutaneous fistula with abdominal wall cellulitis i.e. type III GBP. The patient developed MODS and rapidly deteriorated within a few hours and was fatal inspite of all the resuscitative measures to save her. Management with faster diagnosis and early surgical

intervention is necessary to reduce the mortality in such patients. As the general condition of the patient was rapidly deteriorating, the surgery was called off to stabilize her.

In the Case 2 the patient presented with acute severe pain abdomen with features of peritonitis mimicking the features of intestinal obstruction or biliary obstruction due to high serum bilirubin levels along with sudden relieving of pain abdomen mimicking the feature of dislodgement of obstructed GB but which was actually due to the type I GBP and fall in intracholecystic pressure. All these caused delay in diagnosis and the surgery as the typical features of GBP were lacking in this patient.

As far as the surgical modalities are concerned Type I GBP can be managed by cholecystectomy, peritoneal lavage and by keeping an abdominal drain. Type II GBP can be managed by placing an ultrasound guided percutaneous drainage tube followed by cholecystectomy once infection is relieved. For type III GBP additional surgical procedures such as repair of fistula are required along with cholecystectomy.<sup>15,10</sup> In type IV GBP cholecystectomy with choledocholithotomy is effective. Reconstruction of biliary tree can be done including duct enterostomy or simple closure over a T tube.<sup>14</sup>

## CONCLUSION

Rapid meticulous diagnosis and early surgical intervention is very much required to reduce the mortality and morbidity in the patients of GBP as these patients do not usually present with typical signs and symptoms of perforation which can lead to formation of internal and external fistulae and worsen the situation and increase the mortality rates. The earlier the surgical intervention and lower the type of perforation i.e. type I or type II, the better the post-operative outcomes and lesser the mortality compared to type III. Modalities like enhanced CT, ERCP, MRCP consume a lot of time leading to delayed diagnosis. So, it depends on the sole decision of the surgeon to take ahead the management protocol as most of the GBP are diagnosed intraoperatively.

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