Neglected Perforated Gall Bladder with Generalized Peritonitis in Old & Morbid Obese Patient

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ABSTRACT
The main cause of perforation of the gallbladder is cholecystitis with or without cholelithiasis. In old age, spontaneous perforation of gallbladder can be due to decrease in its blood supply, which can be due to atherosclerosis, focal vasospasm or localized vasculitis. Perforation of gallbladder is associated with high morbidity and mortality, if left untreated. Here we report a case of a 60-year-old male with perforation of gallbladder.

Keywords: Spontaneous perforation, chronic cholecystitis, gallbladder, diabetes mellitus, elderly, gangrenous cholecystitis, acute cholecystitis, cholelithiasis.

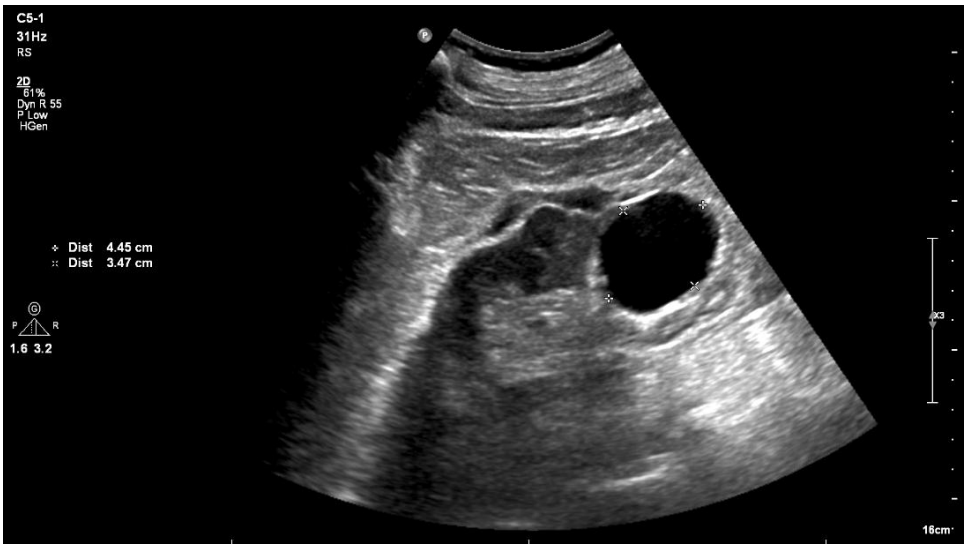
INTRODUCTION
The main cause of gallbladder perforation is cholecystitis with or without gallstones [1].The reported incidence of gallbladder perforation in acute cholecystitis is 2-18% [2]. However, cases reported as idiopathic or spontaneous gallbladder perforation are not only rare but also have features that are different from those occurring as a complication of cholecystitis. Their different features can be described as peritonitis caused by gallbladder perforation lacking the typical clinical presentation, radiological and histopathological characteristics of cholecystitis or gallbladder perforation [3].As a result, the diagnosis is often delayed or even missed.

CASE REPORT
A 61 year old male known case of DM, HTN, and old CVA came to ER complaining of generalized abdominal pain which started in the upper abdomen before 1 week, and aggravated in the last 3 days associated with vomiting. On examination patient was conscious, drowsy, vitals showed sinus tachycardia with relatively low blood pressure & fever, with marked dehydration, chest was clear, abdomen was distended with generalized tenderness & guarding more over right upper abdomen &B scanty urine output.

Laboratory studies:
TLC: 12.3, Hb: 14.2 g/dL, blood sugar: 29.3 mmol/L with positive ketone in urine & metabolic acidosis, BUN: 9 mmol/L, S. creatinine: 182.22 umol/L, serum Bilirubin: 56 umol/L (direct: 39.7), S. Na: 129 mmol/L, S. K: 4.3
D-DIMER: positive. ECG showed sinus tachycardia.

Imaging studies:
CXR:
Was done showed no air under diaphragm.

Ultrasound abdomen:
The gallbladder fossa showed a large hyperechoic structure with obvious acoustic shadowing & fluid collection at the area of gall bladder, but could not assess the wall of the gall bladder with perforated gall bladder & advised to do CT abdomen with contrast study. It showed also free fluid in the pelvis & perisplenic areas.



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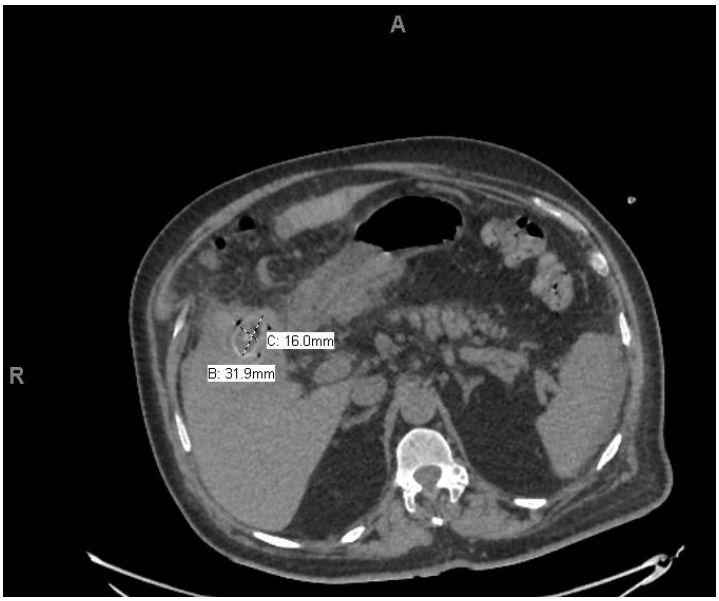
CT scan:
Evidence of multiple tiny free air locules (pneumoperitoneum) seen within right upper abdomen, within and around the gallbladder bed surrounding the known gallbladder stones (measuring about **3.2 x 1.6 cm**) with surrounding heterogeneous hyperdense structure and focal fluid within the gallbladder bed (in which inflamed GB perforation with adjacent small collection is suggested) associated with surrounding mesenteric fat stranding and multiple mesenteric small LNs more marked at the portahepatis and peripancreatic region along with free fluid seen around the liver and spleen .



TREATMENT

The patient immediately shifted to ICU for resuscitation & preparation for surgery. Then shifted to operating room and diagnostic laparoscopy was done. There was gangrenous and perforated gallbladder at the fundus with spread of the biliary contents on the abdominal cavity which cause pyogenic membranes over the bowel, & fluid collections in the subhepatic, perisplenic & pelvic areas.

Opening of the gallbladder from the site of perforation was done but could not reach the area of calots triangle due to the aggressive adhesions around the gall bladder, removal of the gall bladder stone was done and placement of drains in subhepatiec, left subphrenic & pelvic areas .Patient sent back to ICU as he was in septic shock and kept there for **5** days.

By day **5** postoperative, patient was improving & off inotropic support , tolerating orally & was shifted to the regular ward for about **7** days & the subhepatic drain was draining minimal serous fluid & no bile leak, his blood sugar became controlled, & renal function tests back to normal levels & ultrasound abdomen revealed no dilated biliary ducts nor intraperitoneal collections . Patient was discharged on **10TH** post-operative day in good condition.

OUTCOME AND FOLLOW UP

A regular follow up of the patient in surgery clinic for **3** months was done, & the patient was symptoms free & excellent outcome.

DISSCUSION

Gallbladder perforation is a rare but life-threatening event [4] In **1934**, **Niemeier *et al.*** [5] proposed a classification of gallbladder perforation type 1 - acute free perforation into peritoneal cavity; type **2** - subacute perforation with pericholecystic abscess; and type **3** - chronic perforation with cholecystoenteric fistula. A number of modifications have been proposed, but the basic classification still stands.

Roslyn *et al.* [6] reported in his study that type **1** and type **2** gallbladder perforations are mostly seen in young patients (< **50** years), and type **3** is seen in elderly with long history of stone disease. Our patient had a type **1** gallbladder perforation. **Estevao *et al.*** [3] insisted that almost all cases of gallbladder perforation were in fact secondary to a coexistent disease such as inflammation, trauma or obstruction. They also proposed a classification system, which

categorized the perforation of gallbladder into three groups:

1) spontaneous;

2) traumatic; and

3) iatrogenic. The spontaneous group is further sub-divided into an idiopathic and a secondary group, which includes acute inflammation, infection, lithiasis, congenital obstruction and anticoagulant therapy.

Although there is little consensus about the classification, the most possible mechanism of gallbladder perforation in acute cholecystitis is cystic duct obstruction (mostly by a stone at the neck) that causes retention of intraluminal secretions leading to rise in intraluminal pressure.

This raised intraluminal pressure compromises the venous and lymphatic drainage of gallbladder resulting in necrosis and finally gallbladder perforation [7] As gallbladder perforation occurs most commonly at fundus due to least blood supply (**60**% of the cases in the study of **Derici *et al.*** [4] this proves the importance of ischemic mechanism. Conditions such as cholelithiasis, infections, malignancy, diabetes, atherosclerosis, steroid therapy, etc. are all predisposing risk factors for gallbladder perforation [2].

Clinical diagnosis of spontaneous gallbladder perforation is very difficult and often delayed or missed because there are no classical symptoms and signs of gall bladder perforation. It is difficult to discriminate clinically between the patients with perforated gallbladder and those with uncomplicated acute cholecystitis. A sudden decrease in pain intensity caused by the relief of high intracholecystic pressure might herald the perforation according to **Chen *et al.*** [8] Perforation and abscess formation should be suspected in those patients with acute cholecystitis who suddenly become toxic and whose clinical condition deteriorates rapidly as suggested by ***Gore et al.*** [9] Ultrasonography is the initial radiological investigation done in most of the cases but finding are very non-specific for gallbladder perforation and mimic those seen in acute uncomplicated cholecystitis such as gallbladder distention (largest diameter > **3.5 - 4.0** cm), gallbladder wall thickening (> **3** mm), pericholecystic fluid collection, gallstones, coarse intracholecystic echogenic debris and bile duct dilatation.

Distention of the gallbladder and edema of its wall may be the earliest detectable signs of imminent perforation.

The hole sign [10] (a defect in the gallbladder wall) is the most specific finding. ***Solva et al.*** [11] showed in their study that distention of gallbladder and edema of its wall may be earliest sign of impending perforation on ultrasonography. Computed tomography (CT) scan is the most sensitive tool to diagnose gallbladder perforation [12] CT scan finding can be divided into primary gallbladder changes, pericholecystic changes and findings in extra-gallbladder organs. Primary gallbladder changes include wall thickening, wall enhancement, wall defect, intramural abscess, intramural gas, mural hemorrhage, presence of gallstones, bile duct stones or cystic duct stones, intraluminal membrane and intraluminal gas.

Pericholecystic changes include pericholecystic fat stranding, pericholecystic fluid collection, abscess or biloma formation and presence of extra-luminal stones.

Findings in organs other than the gallbladder consist of pericholecystic liver enhancement, liver abscess, portal vein thrombosis, lymphadenopathy, reactive mural thickening of adjacent hollow organs (hepatic flexure of colon and duodenum), intra-peritoneal free air, ascites and Mirizzi syndrome. The gallbladder perforation signs can be divided into direct and indirect signs: the demonstration of either calculi outside the gallbladder or a ruptured segment of the gallbladder wall is a direct indicator according to **Pedosa *et al*.** [13] indirect indicators include the presence of an abscess outside the gallbladder and the presence of gallstones together with thickening of gallbladder wall. Sensitivity of CT scan in the detection of gallbladder perforation and biliary calculi has been reported to be between 88% and **89**% [12]

Magnetic resonance imaging (MRI) may demonstrate the wall of the gallbladder and defects also. The biliary tree is better demonstrated by MRCP than other modalities in presence of gallbladder perforation in suspected cases of acute cholecystitis [14] **.**If USG and CT scans are not conclusive, the MRI is the modality of choice. However, the cost is the limiting factor.

In conclusion, to decrease the morbidity and mortality associated with gallbladder perforation, early diagnosis and surgical intervention are of prime importance.

Presence of risk factors certainly warrants an aggressive investigation to rule out this serious complication.

Gallbladder perforation should be considered in differential diagnosis in elderly patients presenting with peritonitis with an unknown etiology as was in this patient. Aggressive resuscitation & early surgical intervention enhance better outcome.

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