

*Research Article***Gallbladder Perforation with CT Scan****Emhmed Mohamed Saaid**

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Abstract

Perforation of the gallbladder is an uncommon complication of acute cholecystitis that is associated with relatively high mortality. Symptoms and clinical signs can be indistinguishable from those of uncomplicated acute cholecystitis, leading to delayed diagnosis. We reviewed the clinical and imaging findings in 9 patients with gallbladder perforation confirmed at surgery.

Key words: gallbladder, cholecystitis, high mortality.

Introduction

Although an uncommon complication, it is essential to consider the possibility of perforation of the gallbladder in cases of acute cholecystitis, as this condition is associated with a relatively high mortality rate. This paper highlights the imaging features on CT of 9 patients with gallbladder perforation who presented with an acute abdomen. As CT was the preliminary radiological investigation in most of these patients, it was not found practicable to perform an ultrasonographic examination in all, as several of the patients were taken up for exploratory laparotomy soon after the diagnosis was established on CT.

Methods and materials

CT of 9 patients with gallbladder perforation was performed on (multidetector CT; Philips). The protocol was as follows: 10 mm slice thickness and a collimation of 0 mm for the pre contrast scans, and a slice thickness of 0 mm and a collimation of 2.0 mm for the contrast scans; 120 kVp; 160 mAs; rotation time 0.5 s with feed/rotation, and 20 mm and 12.0 mm for the pre-contrast and contrast scans, respectively. 70 ml water-soluble iodinated contrast diluted to 1% was used as oral contrast.

The contrast scan was performed in the portal venous phase of enhancement approximately 60 s after the start of intravenous infusion of contrast. The scans were reviewed on the basis of the following criteria: the site and multiplicity of perforation, the presence of

gallstones and pericholecystic collections, and the presence of gas within the gallbladder/ abscesses. Extension of the inflammatory process into adjacent hepatic parenchyma and pathology in the pancreas and the lung bases were also noted. As CT was the preliminary radiological investigation in most of these patients, it was not practicable to perform ultrasonography as several of the patients were taken for exploratory laparotomy soon after the diagnosis was established on CT.

Results

9 patients were reviewed over a period of 9 months, with a range of 14 years to 47 years (mean age of 48 years). There were 6 females and 3 males. 8 patients (88.9%) had Type 2 perforation (subacute, localized) and only one had Type 1 (acute, generalized peritonitis) perforation.

Surgical confirmation of gallbladder perforation was obtained for all patients. In one patients (11.1%), the perforation was not detected on CT. four patients (44.4%) were either known to be diabetic or, on investigation, found to have elevated blood glucose. The most common presenting symptom was upper abdominal pain; only one patients were febrile at presentation. In the 8 patients (88.9%) in whom a perforation was identified at CT, 4 (44.4%) were solitary. Multiple perforations were identified in only two cases (22.2%). The fundus (44.4%) was found to be the most common site of perforation.

The mean identifiable diameter defect was 14 mm, with a range of 8 mm to 32 mm. Ultrasound was performed in 7 patients and gallbladder perforation was either misinterpreted or not identified in three. Four patients (47%) had biliary calculi, one of which were in the gallbladder

(Figures 1-5); one was in the common hepatic and two were in the cystic duct. A calculus in one patient was missed on CT because of its low attenuation value, which rendered it isodense with gallbladder contents. Pockets of air within

collections in the liver were seen in three out of five patients in whom perforation had led to the formation of an intrahepatic abscess (Figure 1). These were thought to have resulted from the presence of anaerobic organisms rather than from either enteric fistulation or incompetence of the sphincter of Oddi. The striking appearance of calculi within an intrahepatic collection was seen in only one case (Figure 1).

In two patients, perforation occurred in the context of emphysematous cholecystitis.

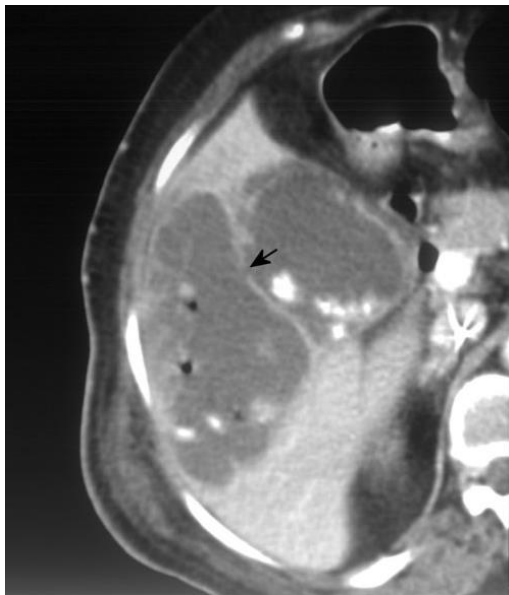


Figure 1. The gallbladder is grossly distended with poor

Figure 2. An axial image reveals a 9 mm wide defect (arrow) definition of its walls. A large encapsulated low-attenuation in the medial wall of a distended gallbladder collection within adjacent hepatic parenchyma shows internal loculation and a few air pockets. Multiple calculi are seen within both the gallbladder and the intrahepatic abscess. A rent in the lateral wall of the gallbladder (arrow) is seen

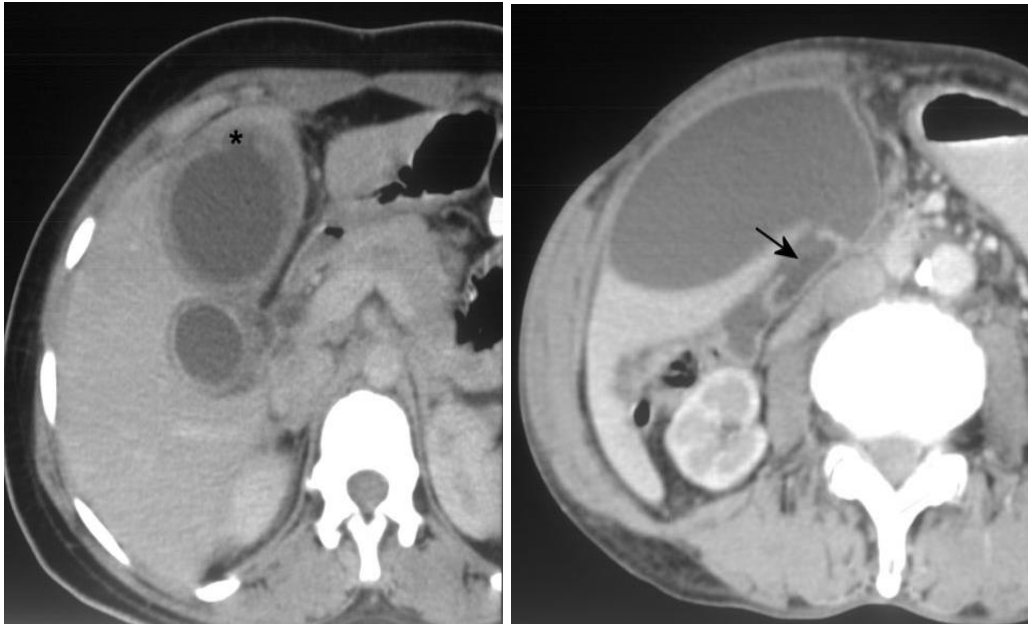


Figure 3. Emphyema of the gallbladder. Irregularity and wall

Figure 4. A contracted gallbladder communicates, with a thickening at the fundus (asterisk) suggests the probable site through a 11mm wide defect (arrow) along its of perforation. An anterior wall with encapsulated collection that tracts between the liver and parietal wall

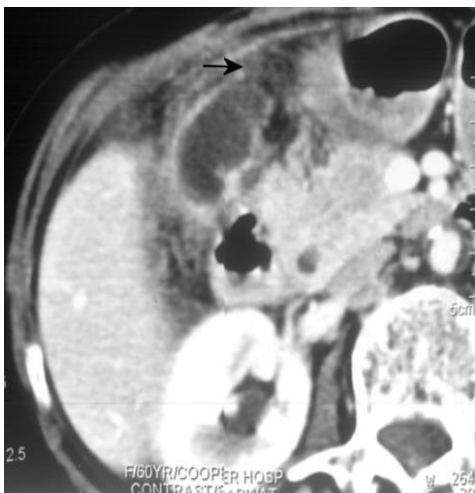


Figure 5. Pericholecystic stranding is seen near a large

Figure 6. A 25 777 mm calculus is seen in the defect (arrow) at the fundus of a distended gallbladder. Neck of an irregularly marginated gallbladder. A 10 mm rent (arrow) along the wall of gallbladder Results in extensive collection within RT lobe of liver

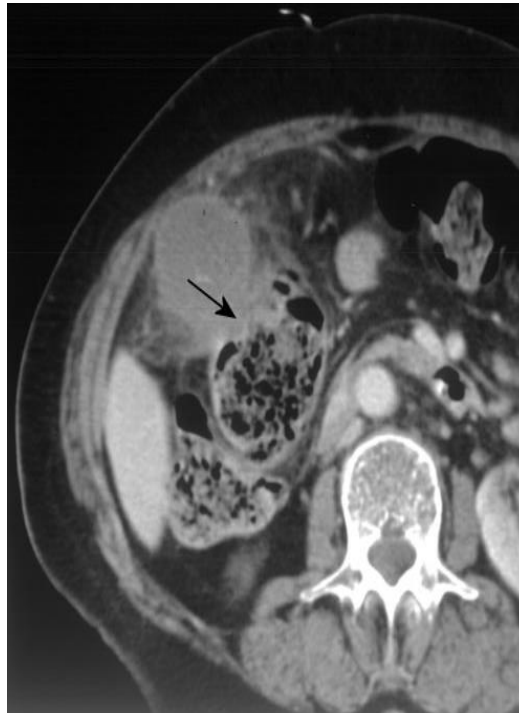


Figure 1. Pericholecystic inflammation confers a “starburst” appearance to the fundus of the gallbladder. The arrow highlights poor definition of the posterior margin of the wall of the gallbladder.

Discussion

Complications such as empyema, gangrenous cholecystitis and gallbladder perforation have been reported in 3–11% of all cases of acute cholecystitis^(1–3).

Perforation can occur as early as 3 days after the onset of acute cholecystitis, or after several weeks^(7,8). The sequence of events that leads to acute cholecystitis and subsequently to perforation is thought to result from occlusion of the cystic duct (most often by a calculus), resulting in retention of intraluminal secretions.

Distension of the organ with a consequent rise in intraluminal pressure impedes venous and lymphatic drainage, leading to vascular compromise and ultimately to necrosis and perforation of the wall of the gallbladder^(9,10). Because of its poor blood supply, the fundus of the gallbladder is the most common site of perforation⁽⁷⁾.

Despite a propensity for perforation in acalculous cholecystitis, most cases of perforation are associated with a calculus

because of the higher incidence of calculus cholecystitis⁽¹¹⁾.

Of our four patients with biliary calculi, stones in the hepatic duct, gallbladder (Figures 1–3) and the cystic duct were demonstrable in one, three and four patients, respectively. For perforation of a non-distended gallbladder (when infection is thought to be the cause), it has been suggested that enlarged Rokitansky–Aschoff sinuses have become infected, leading to necrosis and then rupture⁽⁹⁾.

Apart from cholelithiasis and infection, factors that predispose to perforation are malignancy, trauma and drugs (e.g. corticosteroids). Systemic diseases such as diabetes mellitus and atherosclerotic heart disease are also thought to be contributory⁽⁷⁾ and elderly patients are especially susceptible to gallbladder perforation⁽⁴⁾.

Glenn and Moore et al., in an early study reported an incidence of perforation of the gallbladder five times higher among those patients treated conservatively than among those

who underwent cholecystectomy⁽⁶⁾. The incidence of perforation is known to increase fourfold with a delay in surgery of more than 2 days from the onset of abdominal symptoms⁽⁴⁾. At surgery, the site of perforation is most commonly found to be sealed off by omentum and the transverse colon with adhesions to the liver capsule^(1,11), as seen in our study.

In the early decades of the last century, Niemeier categorized perforation into three types⁽¹²⁾: Type 1 (acute — 32% to 36%), which manifests with generalized peritonitis Type 2 (subacute — 41% to 51%), which denotes localization of fluid at the site of perforation with the formation of a pericholecystic abscess Type 3 (chronic — 1% to 14%), in which internal (bilio-biliary or bilio-enteric) or external fistulae occur⁽¹⁾.

In our study, a Type 1 perforation was detected in a single patient. This finding is in keeping with more recent reports in the medical literature that cite a higher incidence of subacute perforation⁽¹⁾.

The clinical presentation of gallbladder perforation may range from an acute generalized peritonitis to benign non-specific abdominal symptoms. Clinical differentiation between gallbladder perforation and uncomplicated cholecystitis can often be difficult because the bile leak from a ruptured gallbladder might be contained in the extra peritoneal gallbladder fossa, and hence might not produce symptoms of peritonitis immediately.⁽¹⁷⁾

Gore et al.,⁽¹⁴⁾ thought that perforation and abscess formation should be suspected clinically in those patients with acute cholecystitis who suddenly become toxic and whose clinical condition was found to deteriorate rapidly. A delay in diagnosing gallbladder perforation could be explained by the similarity of clinical manifestations to those of acute cholecystitis.

The site of maximal pain in regions other than those associated with gallbladder pathologies could lead to a mistaken diagnosis of acute appendicitis or of a perforated duodenal ulcer⁽¹¹⁾. If unattended, the mortality associated with this condition is as high as 20%⁽¹⁾ and is higher still in patients with immune deficiency

and in cases of malignancy⁽⁷⁾. The mortality rate is 10 times greater in acute than in subacute perforation⁽¹⁾.

As calculi are often missed on CT, sonography followed by CT is preferred in suspected biliary pathology; however, ancillary findings such as pneumobilia are more likely to be detected on CT. The crumpled wall of a decompressed gallbladder floating within fluid of the gallbladder fossa has a distinctive appearance and can be seen in some cases of Type 1 perforation.

Soiva et al., in their study of four cases of acute gallbladder perforation found that distension of the gallbladder and oedema of its walls may be the earliest signs of impending perforation⁽¹⁵⁾. They also observed that pericholecystic or free fluid in the peritoneal cavity was not found in uncomplicated acute calculus cholecystitis⁽¹⁵⁾. The presence of air within the wall of the gallbladder (emphysematous cholecystitis) prognosticates impending perforation⁵.

Following perforation, CT and ultrasonography both show complex fluid collections surrounding the gallbladder.

The wall of the gallbladder can appear focally disrupted and the gallbladder lumen may be seen within, or peripheral to, the pericholecystic abscess⁽¹⁴⁾.

Most studies of perforation of the gallbladder deduce that the wall of the gallbladder following perforation appears distended, thickened, oedematous and on occasion fails to be identified^(15,17). Our identification of the gallbladder on CT following perforation in each of the 9 cases reviewed is at variance with the common belief that the gallbladder might not be detected on CT in over a third of cases. Kimet al.,⁽¹¹⁾ in their comparative study of CT and ultrasonography on 7 patients with gallbladder perforation detected the site of perforation in 50% of patients on CT but in no patient on ultrasonography. However, they found both modalities equally effective in demonstrating pericholecystic fluid collections, gallbladder wall thickening and cholelithiasis. A study of 22 patients with gallbladder perforation by Sood et al.,⁽¹⁶⁾ in 2001 showed a marginally higher rate of detection of gallbladder wall defects on CT than with ultrasonography.

In 6 of our patients who underwent ultrasonography, perforation was either misinterpreted or not detected in three.

Ultrasonography in two cases missed small collections: an abscess in the liver on account of artefacts from an emphysematous gallbladder (Figure 9) and a perihepatic collection beneath the anterior chest wall remote from the gallbladder (Figure 6).

Ultrasonography is usually the initial mode of investigation in cases of suspected gallbladder perforation. As the sensitivity of CT in the detection of gallbladder perforation and biliary calculi was found to be 86% and 87%, respectively, we recommend that it be performed in all such cases.

References

1. Isch J, Finneran JC, Nahrwold DL. Perforation of the gallbladder. *Am J Gastroenterol* 1971;66:451-8.
2. Bennet GL, Balthazar EJ. Ultrasound and CT evaluation of emergent gallbladder pathology. *Radiol Clin North Am* 2003;41:1203-16.
3. Strohl EL, Diffenbaugh WG, Baker JH, Chemma MH. Collective reviews: gangrene and perforation of the gallbladder. *Int Abstr Surg* 1972;114:1-7.
4. Cope Z. A sign of gallbladder disease. *BMJ* 1970;3:147-8.
5. Glenn F, Moore SW. Gangrene and perforation of the wall of the gallbladder. *Arch Surg* 1942;44:677-86.
6. Madrazo BL, Francis I, Hricak H, Sandler MA, Hudak S, Gitschlag K. Sonographic findings in perforation of the gallbladder. *AJR Am J Roentgenol* 1982;139:491-6.
7. Meire H, Cosgrove D, Dewbury K, Wilde. Gallbladder pathology. In: Cosgrove D, Meire H, Dewbury K, editors. *Abdominal and general ultrasound*. Longman Group; 1993:190-8.
8. Martin JD Jr, Stone HH. Perforations of the gallbladder; areport of three cases. *Geriatrics* 1907;12:476-80.
9. Harland C, Mayberry JF, Toghil PJ. Type 1 free perforation of the gallbladder. *J R Soc Med* 1980;73:720-78.
10. Ong CL, Wong TH, Rauff A. Acute gallbladder perforation — a dilemma in early diagnosis. *Gut* 1991;32:907-8.
11. Kim PN, Lee KS, Kim IY, Bae WK, Lee BH. Gallbladder perforation: comparison of US findings with CT. *Abdom Imaging* 1994;19:239-42.
12. Smith R, Rosen JM, Alderson PO. Gallbladder perforation: diagnostic utility of cholescintigraphy in suggested subacute or chronic cases. *Radiology* 1986;168:73-6.
13. Sood B, Jain M, Khandelwal N, Singh P, Suri S. MRI of perforated gallbladder. *Australas Radiol* 2002;46:438-40.
14. Gore RM, Yaghami V, Newmark GM, Berlin JW, Miller FH. Imaging benign and malignant disease of the gallbladder. *Radiol Clin North Am* 2002;40:1307-23.
15. Soiva M, Pamilo M, Paivansaalo M, Taavitsainen M, Suramo I. Ultrasonography in acute gallbladder perforation. *Acta Radiol* 1988;29:41-4.
16. Johnson LB. The importance of early diagnosis of acute acalculus cholecystitis. *Surg Gynecol Obstet* 1987;164: 197-203.
17. Sood BP, Kalra N, Gupta S, Sidhu R, Gulati M, Khandelwal N, Suri S. Role of sonography in gall bladder perforation. *J Clin Ultrasound* 2002;30:270-4.