treatment for gastric SmCC remains controversial. The surgical approach follows the same criteria of adenocarcinoma as it is linked to the primary tumour location in the stomach.[2] Because of similarity in the biological and clinical characteristics of gastric and pulmonary SmCC, some authors affirmed that the validity of chemotherapy with a regimen specific for pulmonary SmCC may be suitable for the treatment of this tumour.[5] The combination chemotherapy usually consists of cisplatin and etoposide or irinotecan.[2] Multimodality approaches are promising but more studies are needed before any single combination can be recommended.[1]

TARUN RUSTAGI[1],
MURTUZA RAMPURWALA[1],
MRIDULA RAI[1],
SHARON FUENTES[2],
MICHAEL GOLIOTO[3]

Correspondence: Dr. Tarun Rustagi
Department of Internal Medicine[1]. University of Connecticut Health Center, Farmington and Department of Pathology[2], and Division of Gastroenterology[3], Hartford Hospital, Hartford, CT
Email: tarun.rustagi@yale.edu

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Chronic hepatic abscess due to gallbladder perforation: three cases and exact nomenclature

Introduction

Gallbladder perforation is a rare cause of secondary peritonitis in the Eastern hemisphere, with a higher mortality rate than that caused by perforation of other hollow viscera.[1,2] Owing to prompt management of symptomatic cholelithiasis, the incidence of gallbladder perforation has also declined worldwide in patients of acute cholecystitis (~1%).[3,4] Niemeier in 1934[5] classified gallbladder perforation into three types: type I—chronic perforation with the presence of a fistulous communication between the gallbladder and some other viscus; type II—subacute perforation where the perforated gallbladder is surrounded by an abscess walled off by adhesions from the general peritoneal cavity; and type III—acute perforation of the gallbladder into the free peritoneal cavity without protective adhesions. Though this classification has withstood the test of time, there is some debate regarding its modification, especially about inclusion of specific types such as cholecystohepatic or cholecystobiliary fistulae.[6–8]

Type II is the most common type (40%–63% cases) of gallbladder perforation. Most cases present with signs of localized peritonitis such as pain, fever, tenderness, and, leukocytosis.[9,10] Among type II, however, hepatic abscess with gallbladder perforation is very rare and has been reported in only a few cases.[8,11–13] We present three cases of gallbladder perforation with hepatic abscess formation. In all three patients, the presentation was insidious, with minimal symptoms. We wish to emphasize the unexpected location of the pathology as well as the uncommon presentation. The merits of modification of the Niemeier's classification are also discussed.

Case 1

An 18-year-old boy presented with mild upper right quadrant abdominal pain of 3 weeks' duration. There was no history of jaundice, acute pain, vomiting, fever, or other significant complaints. On examination, the boy was found to be afebrile and moderately-nourished with no abnormal signs. Ultrasound abdomen was suggestive of irregular thickening of the gallbladder with multiple calculi in the lumen, along with a hypoechoic septate subcapsular hepatic collection abutting the right anterior lower chest wall. Computed tomography (CT) scan revealed an irregularly thickened gallbladder wall with a defect in the fundal region, communicating with a localized subcapsular hepatic septate collection (**Figures 1a, 1b**). The gallbladder was also filled with sludge and calculi, and there

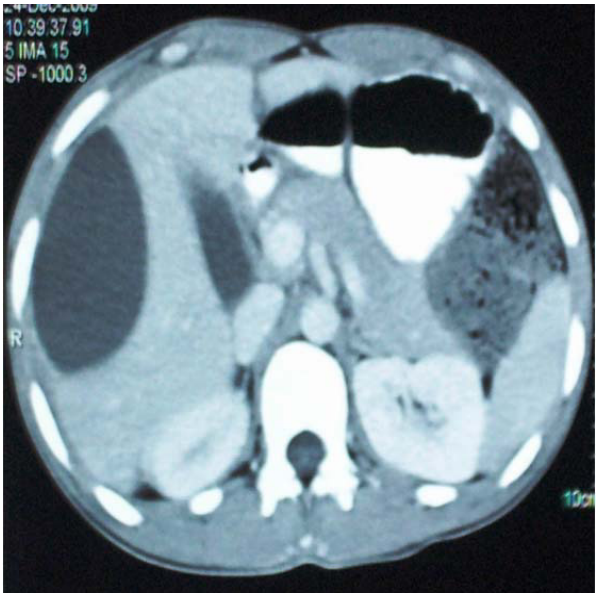

Figure 1a: CT image showing a defect in the gallbladder fundus and a localized subcapsular hepatic septate collection.

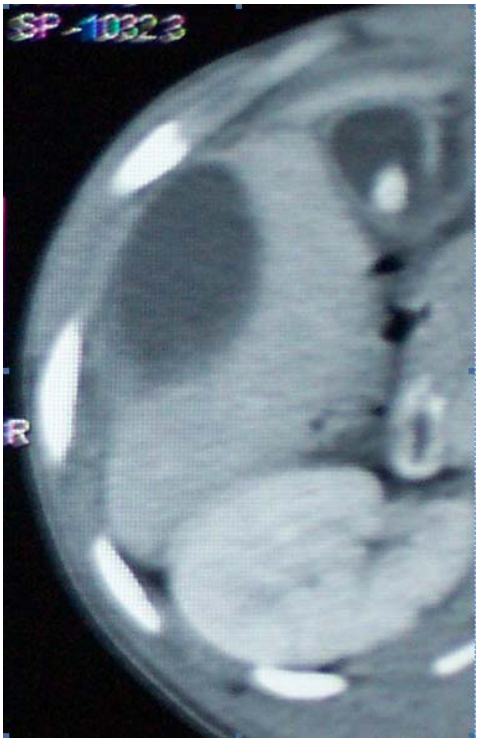

Figure 1b: Localized view of the same image showing communication between the fundus and the abscess cavity.

was significant pericholecystic fluid. The liver function tests and other blood tests were unremarkable. Laparoscopic cholecystectomy was planned. The findings were dense adhesions in the Calot's triangle, and a thickened, contracted intrahepatic gallbladder, necessitating conversion to an open procedure. On conversion, a walled-off collection of thick pus (~100 mL) communicating with the gallbladder fundus was drained. The gallbladder was dissected in retrograde fashion, and a subtotal cholecystectomy was performed. Postoperative recovery was uneventful, and histopathological examination revealed chronic calculous cholecystitis. The patient was well 4 months after the operation.

Case 2

A 40-year-old man reported to the surgery outpatient clinic with a history of mild pain in the right upper quadrant for the past 2 days. There was no history of fever, vomiting, jaundice, or other complaints in the recent or remote past. General and systemic examinations were unremarkable, except for mild tenderness in the right hypochondrium and lumbar region. Ultrasound abdomen revealed a large (~200 mL), hypoechoic collection abutting the inferior part of the right hepatic lobe. The gallbladder was collapsed; however, multiple calculi in the lumen and irregularity of the fundal wall could be identified. Ultrasound-guided aspiration of the fluid revealed bile with debris and pus, which was drained with a percutaneous catheter. There was no significant drainage in the next 48 hours. With a diagnosis of gallstones with gallbladder perforation, early laparoscopic cholecystectomy was attempted 2 days after the drainage. We found calculi in an intrahepatic gallbladder and a perforation at the fundus communicating with an inferior lobe liver abscess, walled-off by omentum. There was a minimal inflammatory peritoneal exudate. Cholecystectomy was completed without event. Histopathology revealed chronic calculous cholecystitis. The patient was well 6 months after the operation.

Case 3

A 42-year-old man presented with a 4-month history of colicky right upper quadrant pain. There were no other complaints. General examination was unremarkable. Ultrasound abdomen revealed gallbladder wall thickening with sludge in the lumen. There was a suspicion of discontinuity of the fundal wall. Magnetic resonance cholangiopancreatography (MRCP) showed a small perforation at the gallbladder fundus with communicating pericholecystic and intrahepatic fluid (**Figure 2**). No calculi were identified in the gallbladder. The biliary ductal system was normal. The patient underwent laparoscopic cholecystectomy, and a thickened gallbladder with a sealed fundal perforation and a small amount of intrahepatic pus was found. No symptoms were found after 3 months of follow-up.

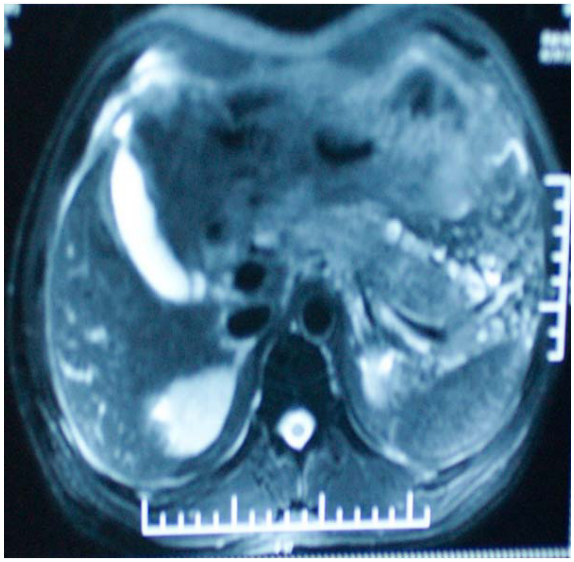

Figure 2: Magnetic resonance cholangiopancreatography (MRCP) image showing perforation at the gallbladder fundus communicating with a small intrahepatic collection.

Discussion

Perforation of the gallbladder is a rare cause of secondary peritonitis, with a mortality rate of up to 20%.[2] It may occur in both calculous and acalculous cholecystitis, with a decreasing incidence in the recent literature (<1%).[3,4] The factors responsible for increased risk of perforation have been shown to be cholelithiasis, infection, malignancy, trauma, steroid intake, diabetes, old age and male sex.[3,4,9,10] The clinical presentation may vary, as suggested by Niemeier (vide supra). The most common type is a subacute, localized abscess (type II), which is heralded by fever, pain, tenderness, and leukocytosis. These signs are indistinguishable from acute cholecystitis; hence, preoperative diagnosis is difficult. However, a "cold" presentation or a chronic abscess with minimal symptoms finds scant mention in the literature.[8–10] In all three of our patients, there were minimal clinical or biochemical indicators of the perforation. Kocher et al.[8] have suggested that the declining incidence of gallbladder perforation may be attributable to earlier and better management of symptomatic calculi. On the contrary, one may argue that, with better analgesics and antibiotics available, many perforations may be "silent" and may resolve on oral therapy.

Another distinguishing feature in our patients was an intrahepatic abscess communicating with a perforation at the fundus of an intrahepatic gallbladder. This was identified by imaging and at operation in all three cases. Intrahepatic perforation of the gallbladder with abscess formation has been reported in only a few adult cases, [8,11,12,14–15] and one paediatric case.[13] In most of these cases, the gallbladder was intrahepatic with the perforation present at the fundus. Laparoscopic cholecystectomy was either unsuccessful or not attempted in these patients. We were able to successfully complete laparoscopic cholecystectomy in two patients.

There is considerable debate in the literature regarding modification of the original Niemeier's classification. Anderson and Nazem[6] and Ibrarullah et al.[7] have favoured inclusion of cases of cholecystobiliary fistula under type IV gallbladder perforation, in view of the unique operative approach required in such cases. Similar to the most recent literature on the subject, Kochar et al. argued that, a "modified" Niemeier's classification may be used and all cases of fistula formation (cholecystobiliary, cholecystohepatic, cholecystocutaneous or cholecystoenteric) should be included under a single entity, i.e. type III. They are not in favour of a new type, in order to avoid discrepancies and maintain uniformity in reporting.[8] We agree with this suggestion and, accordingly, all our three cases can be classified under type III gallbladder perforation.

Diagnosis of intrahepatic abscess due to gallbladder perforation can be difficult, especially in the absence of symptoms and signs. Ultrasonography may be suggestive, but most findings are not specific for perforation.[16] These could be distension of the gallbladder and increased wall thickness/ oedema. Perforation can be demonstrated unmistakably only by a CT or magnetic resonance imaging (MRI [MRCP]) scan. These investigations are also valuable in locating other intra-abdominal collections.[11, 17] Since liver abscess is a common entity in India and other developing countries, these cases alert us to the possibility of gallbladder being the offending organ, necessitating cholecystectomy in addition to treatment of the abscess. However, CT scan is not cost-effective for all cases. Indications of CT scan in liver abscess are associated findings on ultrasound such as non-visualization of the gallbladder, impending gallbladder perforation or calculi within the abscess.[8, 11, 17]

Percutaneous drainage needs a mention here, to emphasize its utility in patients where immediate surgery is not feasible due to acute illness or logistical problems. In our second patient, early CT scan was not possible for reasons of logistics, prompting percutaneous drainage and early laparoscopic cholecystectomy.

NITIN AGARWAL,
PRADEEP SAINI,
ARUN GUPTA,
NAVNEET KAUR,

SURUCHI SHRESHTHA,
MOHAMMAD SHAZIB FARIDI

Correspondence: Dr. Nitin Agarwal,
Department of Surgery
University College of Medical Sciences and
Guru Tegh Bahadur Hospital,
Delhi 110096, India
Email: drnitinagarwal76@gmail.com

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Isolated periampullary tuberculosis: masquerading as periampullary carcinoma

Introduction

Periampullary tumours are mostly malignant; however, benign lesions usually have a similar presentation. Isolated tuberculosis of the periampullary area is extremely rare, with only one case reported in the literature[1] Tuberculosis of the pancreatoduodenal and periampullary area is also a rare entity with only a few cases reported so far.[2–4] Occurrence of tuberculosis in the periampullary area is rare due to its high gastric acid content, continuous motor activity and relative scarcity of lymphoid follicles.[5] All the cases reported were diagnosed after resection and the treatment was surgery followed by anti-tubercular therapy. We report the first case of isolated periampullary tuberculosis in a patient diagnosed preoperatively by endoscopic biopsy and managed conservatively with 6 months of anti-tubercular therapy.

Case report

A 56-year-old man presented with a history of jaundice for 4 weeks which was progressively deepening associated with pruritus and clay-coloured stools. He also had dull aching epigastric pain associated with loss of appetite and fever for the same duration. He had a past history of recurrent epigastric pain due to duodenal ulcer for which 15 years ago he underwent distal gastrectomy with Bilroth II anastomosis and truncal vagotomy after which he was asymptomatic. On examination, he was febrile (102 ^{O}F) and had deep icterus. He had tense cystic gallbladder palabable. Routine investigations showed mildly elevated total leucocyte count (13,000 cells/cmm) and elevated erythrocyte sedimentation rate (ESR), [65 mm at 1 hour]. Liver function tests showed: total bilirubin level 16 mg/dL with direct fraction of 12.5 mg/dL, aspartate transaminase 112 U/L, alanine transaminase 126 U/L and alkaline phosphatase 992 U/L. Chest X-ray was normal. Contrast-enhanced computed tomography (CECT) of the abdomen showed distented