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An unusual case with a patient with concurrent peptic ulcer perforation and gallbladder perforation

Zen Zuan Yong, Janaka Balasooriya

ABSTRACT

Concurrent gallbladder and peptic ulcer perforation are both life-threatening, rare conditions that present as an acute abdomen. Here we discuss a patient who presented with acute abdomen with computed tomography (CT) scan showing evidence of hollow viscous perforation and subsequent exploratory laparotomy revealing both gallbladder and gastric perforations. The discussion involves the rarity of such reported cases and mainstay treatment, which is surgical. These rare scenarios always warrant suspicion whether there is a possible connection, or cause to this. It is also extremely important to examine for other parts of the bowel and abdominal viscera for possible second pathology, even when an obvious perforation is found as it can be missed. Early surgical intervention is important and carries a much better prognosis in such patients presenting with this.

Keywords: Concurrent, Gallbladder perforation, Peptic ulcer perforation

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INTRODUCTION

Peptic ulcer disease occurs commonly and is found in 5–10% of the general population [1]. The pathophysiology of this condition commonly occurs due to damage unto the gastro-duodenal mucosa resulting in erosions and exposing gastro-duodenal secretions to underlying tissue. Rare complications that arise from this include perforations with an annual incidence of 0.004–0.014% and a mortality rate of 23.5% with a sample size-weighted average of 30 days [2].

Gallbladder perforation is a rare but life-threatening disease, which demands urgent surgical intervention and is often fatal when missed. Perforation may occur in calculous or acalculous cholecystitis, in 6–12% of cases and has a mortality rate of 12–42% [3, 4].

We present an unusual case of a patient presenting with both peptic ulcer and gallbladder perforations and could not find any similar cases reported in the literature.

CASE REPORT

A 73-year-old man was transferred from a regional hospital to Canberra Hospital for sepsis, suspected to be secondary to a right hip collection three weeks following a total hip replacement with orthopedic surgery.

On arrival he was found to be confused, and was complaining of abdominal pain, additionally, he had an episode of melena. On examination, he was febrile and tachycardic but not hypotensive, his abdomen was distended with generalized peritonism.

His medical history was significant for hypertension, diabetes mellitus, gastro-esophageal reflux disease, chronic obstructive pulmonary disease, and ischemic heart disease. A pacemaker has been inserted in five years ago for complete heart block. His surgical history includes a right-sided L5/S1 laminectomy with decompression, and resection of a neuroendocrine polyp in the rectum.

He had no documented history of known gallstones, nor did he have any specific risk factors for peptic ulcer disease, including excessive use of non-steroidal

anti-inflammatories (NSAIDs) or corticosteroids. Biochemical markers revealed a white cell count of 31×10^9/L and a C-reactive protein (CRP) of 316 mg/L, additionally he had an acute on chronic kidney injury. An abdominal X-ray showed some lucencies projecting over the liver suspicious for pneumoperitoneum. A subsequent CT abdomen and pelvis with intravenous (IV) contrast showed intraperitoneal free fluid and small volume pneumoperitoneum with an apparent focal discontinuity in the posterior wall of the first part of the duodenum (D1), suspicious for a perforated duodenal ulcer (Figure 1).

The decision was made by the surgical team for an urgent exploratory laparotomy. Intra-operatively, it was found that the patient had a 2.5 cm diameter pre-pyloric gastric perforation due to peptic ulcer disease, extending to the posterior wall of D1, as well as an acutely inflamed gallbladder with patchy necrosis, and a perforation adjacent the gastric perforation. There was gross contamination of the peritoneal cavity with both bile and gastric content (Figures 2–5).

The gallbladder perforation was temporarily sutured with polydioxanone suture (PDS) to minimize further bilious contamination and an open cholecystectomy was performed (Figure 3). Intraoperative cholangiogram was normal. The gastric perforation was primarily closed with PDS and an omental patch was secured over the primary repair. No biopsies were taken from the ulcer as it was benign in appearance. The peritoneal cavity was washed with copious normal saline until clear, and a 19 French Blake drain was placed at the site of repair. Interestingly, intra-operatively there was no evidence of gallstone disease.

Histopathology of the gallbladder shows no evidence of malignancy but an acute on chronic gangrenous cholecystitis with perforation. No stones were present in the gallbladder.

Postoperatively the patient was continued on a high dose proton pump inhibitor and empirical antibiotics. On day 5 post surgery, he had a CT chest/abdomen/pelvis due to persistent fevers which revealed a right iliac fossa (RIF) collection measuring up to 65×20 mm, large bilateral pleural effusions and lower lobe atelectasis which were managed conservatively. On day 9 post surgery, a repeat CT abdomen/pelvis was performed as inflammatory markers were rising with white blood cell (WBC) count 18×10^9/L (neutrophils of 16×10^9/L) and CRP 162 mg/L, which revealed a small bowel obstruction with a transition point identified in the left lower quadrant likely secondary to adhesions which was managed conservatively. The known collection in the RIF as well as the pleural effusions had both reduced in size but considering high inflammatory markers, an ultrasound-guided aspiration of the RIF collection was done which aspirated 5 mL of serious fluid. The patient continued to progress slowly, and on post-op day 22 was subsequently stepped down from acute care to a rehabilitation center.

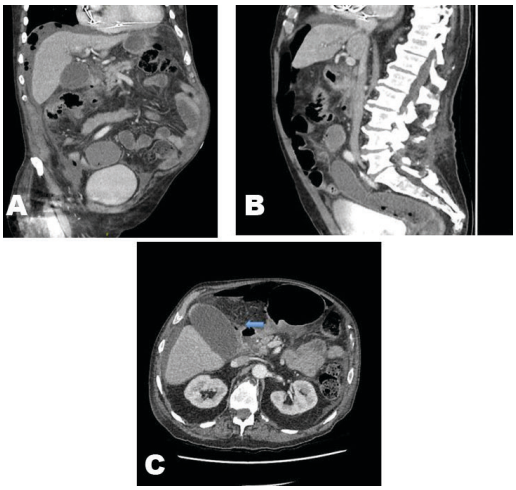

Figure 1: (A) Coronal view of CT intraperitoneal free fluid and pneumoperitoneum. (B) Sagittal view of focal discontinuity in the posterior wall of the pyloric region. (C) Arrow indicating possible site of perforation in the gallbladder wall.

Figure 2: Arrow indicating bile leak from the gallbladder.

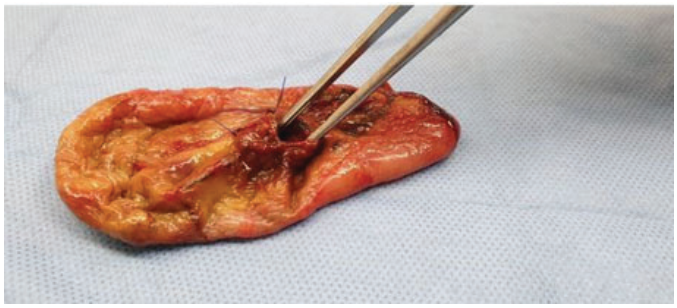

Figure 3: Perforation of around 10 mm in the gallbladder wall.

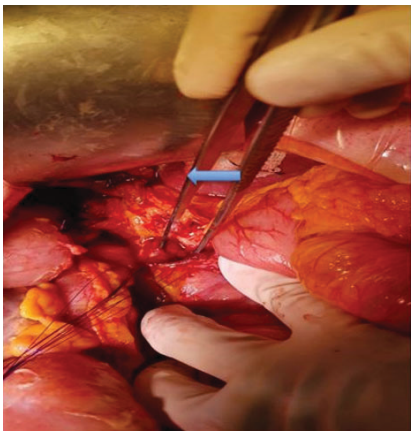

Figure 4: Illustrating perforation site at the pre-pylorus approximately 25 mm extending to the posterior wall with polydioxanone suture (PDS) applied with arrow pointing toward cystic stump.

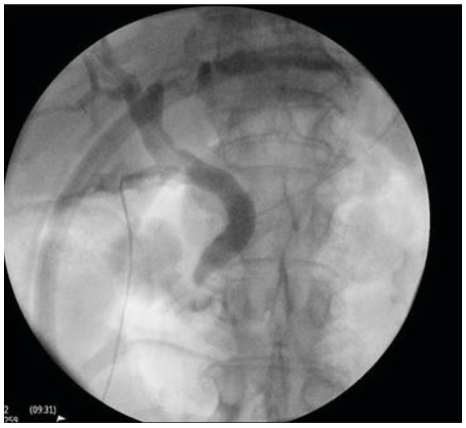

Figure 5: Intraoperative cholangiogram demonstrating the normal flow without filling defects.

DISCUSSION

Gastric perforations more likely in the adult population, and most commonly associated with peptic ulcer disease. Other causes include trauma, malignancy, and spontaneous perforation which is rare but can occur in newborns [5]. Perforations associated with peptic ulcer disease are most common in the duodenum (60%), followed by the gastric antrum (20%) and body (20%) [6].

Treatment for peptic ulcer perforations vary according to location, size, patient's hemodynamics and co-morbidities and, options include but not limited to primary repair with or without omental plug, distal gastrectomy and non-operative management in otherwise stable patients, or poor surgical candidates. In our patient, due to mobility of the wall, perforation could be repaired with primary closure with an omental plug.

Gallbladder perforations are usually a consequence of acute cholecystitis, occurring in 6–12% of such cases [7, 8]. Other causes of gallbladder perforations would be steroidal usage, malignancy, trauma, and vascular compromise [9]. The clinical presentation of gallbladder perforation can vary from mimicking a simple acute cholecystitis to that of diffuse peritonitis which more commonly affects the comorbid and higher aged group [10].

Gallbladder perforations can be classified into three types based on Niemeier's classification.

Type 1—which would be acute and is associated with generalized biliary peritonitis.

Type 2—subacute, consisting of fluid localized in the perforation site, localized peritonitis and pericholecystic abscess.

Type 3—chronic which includes the formation of internal and external fistulas.

Modified type 4—in which the gallbladder may have a perforation into the biliary tree, which would also include the formation of a cholecysto-biliary fistula [11].

Our patient had a type 1 perforation over peritoneal surface of gallbladder with leakage of bile directly into peritoneal cavity.

Abdominal pain, fever, and vomiting are common symptoms seen with this condition and CT is more sensitive for detecting gallbladder perforations than ultrasound, despite ultrasound being the preferred imaging modality for assessment of cholecystitis. The most common treatment options for gallbladder perforations would be cholecystectomy, abscess drainage, and peritoneal cavity washout.

Possibilities for concurrent gastric/duodenal and gallbladder perforation include direct pressure from the inflamed gall bladder on the stomach/duodenum or, peptic ulcer perforation with secondary acalculous cholecystitis and subsequent perforation. In our case, the perforation was thought not to be due to disruption of a cholecysto-duodenal fistula as the patient had no evidence of gallstone disease, and a fistula is unlikely in this context. Additionally, there were no inflammatory adhesions between the two organs.

There are no reported similar cases in the literature. One case report titled "Bowel hath no fury like a gallbladder inflamed" by Amad Khan has described a 73-year-old patient with necrotic cholecystitis and erosion into D1. However, in this case, there was no concurrent perforation of the gallbladder.

CONCLUSION

Although peptic ulcer perforation and gallbladder perforations are not infrequent presentations individually in acute surgical practice, it is extremely rare to have both pathologies concurrently. We present this unique case of a patient presenting with both pre-pyloric perforation and gallbladder perforation, where the surgical management led to a positive outcome. This case highlights the importance of exploring concurrent pathologies, even though the primary pathology is obvious.

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Author Contributions

Zen Zuan Yong – Conception of the work, Design of the work, Acquisition of data, Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Janaka Balasooriya – Analysis of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.