

Case Report

Acute acalculous cholecystitis as a rare cause of gastric outlet obstruction

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Received: 17 December 2022

Accepted: 08 January 2023

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ABSTRACT

Gastric outlet obstruction (GOO) is a condition that classically presents with upper abdominal pain and post-prandial vomiting due to mechanical obstruction secondary to luminal, intraluminal or extraluminal disease. Our case report, the first of its kind in the literature to our knowledge, describes GOO secondary to acalculous cholecystitis in a 58-year-old male who presented to the emergency department with abdominal pain and nausea. This unusual case highlights the need for clinicians to consider uncommon differentials in patients presenting with common symptoms such as abdominal pain and nausea.

Keywords: Gastric outlet obstruction, Acalculous cholecystitis, Cholecystitis

INTRODUCTION

Acalculous cholecystitis typically occurs in critically ill hospitalised patients or those with risk factors such as immunosuppression or major trauma.¹ Presenting features vary widely depending on the clinical context and may include right upper quadrant pain and a positive Murphy's sign, whereas in the critically ill patient acalculous cholecystitis may solely present with unexplained fever. GOO can similarly present with upper abdominal pain but typically without features of sepsis. Post-prandial vomiting is a classic feature and causes can be broadly divided into intraluminal, luminal and extraluminal, with malignancy underlying a large proportion of cases.² It is also worthwhile mentioning Bouveret syndrome, in which gallstones can enter the stomach or duodenum via an acquired fistula tract and result in GOO.³ The pathophysiology of this rare phenomenon clearly differs from that of our patient's, however, who developed GOO secondary to severe acalculous cholecystitis resulting in significant omental adhesions.

CASE REPORT

A 58-year-old male presented to the emergency department with a two-day history of epigastric pain, bloating and nausea. He had a significant cardiac history including a previous type A aortic dissection repair, ablation for supraventricular tachycardia and percutaneous coronary intervention for myocardial infarction. He had no other surgical history. On presentation he was haemodynamically stable and afebrile. Examination revealed epigastric and right upper quadrant tenderness without peritonism. Biochemical investigations demonstrated a white cell count of $17.7 \times 10^9/L$, a c reactive protein of 50mg/L and a mildly raised alkaline phosphatase of 123 U/L. Liver function was otherwise normal. Computerised tomography (CT) showed a thickened and distended gallbladder with adjacent fat stranding, suspected cholelithiasis and a significantly distended stomach (Figure 1).

Interestingly, abdominal ultrasound subsequently demonstrated acalculous cholecystitis. The patient was commenced on intravenous antibiotics and a nasogastric

tube was inserted, immediately draining 400 ml of gastric fluid. He then underwent a laparoscopic cholecystectomy, intra-operative cholangiogram and gastroscopy. Laparoscopy revealed a necrotic gallbladder and significant omental adhesions to the surface of the liver and encompassing the entire gallbladder, resulting in GOO. On-table gastroscopy demonstrated thick brown material within the stomach with no endoscopic cause for GOO seen. Recovery was complicated by a cerebral hypoperfusion event, with no lasting deficits. Histopathology demonstrated extensive gallbladder necrosis and no calculi. The patient had recovered well on review at his outpatient follow-up and denied any ongoing symptoms of GOO.

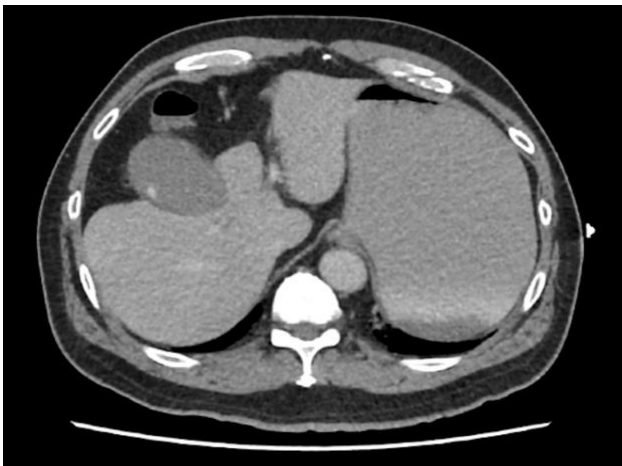


Figure 1: Axial section of abdominal CT demonstrating suspected cholelithiasis and dilated stomach.

DISCUSSION

To our knowledge, there is only one case report which presents a similar phenomenon in the surgical literature. Uko et al describe a massively dilated gallbladder extrinsically compressing the gastric pylorus and causing GOO in a 101-year-old woman.⁴ In our patient's case, however, it appears that GOO occurred secondary to extrinsic compression from omental adhesions caused by severe acalculous cholecystitis, which has not been described in the literature previously. To further add to the unusual nature of his presentation, our patient was reasonably well at the time of his presentation, unlike the vast majority of critically ill patients who present with acalculous cholecystitis. In addition, he lacked the usual risk factors for acalculous cholecystitis. In our patient's

case, a diagnosis of acute cholecystitis was made via CT and supplemented by ultrasonography, however it is worthwhile noting that ultrasonography is a more sensitive imaging modality and changes of cholecystitis may not be apparent on CT.⁵ Abdominal ultrasound should therefore be considered in patients with GOO where a cause is not demonstrable on CT or endoscopy.

CONCLUSION

Abdominal pain is a common presentation in the emergency department and is often accompanied by non-specific features such as nausea and raised inflammatory markers, making diagnosis challenging at times. This unusual presentation of a 58-year-old male with GOO secondary to acalculous cholecystitis serves as a reminder for clinicians to consider a broad range of differentials and select appropriate imaging modalities in the patient presenting with undifferentiated abdominal pain.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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Cite this article as: Aksakal G, Wong E. Acute acalculous cholecystitis as a rare cause of gastric outlet obstruction. *Int Surg J* 2023;10:283-4.