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Case Report

Gastric outlet obstruction in the setting of acute cholecystitis ☆

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ABSTRACT

Acute cholecystitis is a rare cause of gastric outlet obstruction (GOO) and may manifest with nonspecific symptoms which may preclude or delay diagnosis. During the latter part of the 20th century, benign disease was responsible for most cases of GOO in adults with peptic ulcer disease accounting for up to 90% of these cases. Although precise estimates on GOO incidence and prevalence are lacking, as many as 2000 surgical operations were reportedly performed annually for GOO in the United States in the 1990s. We present clinical and imaging findings of a case of a 101-year-old woman with GOO resulting from extrinsic compression from a massively dilated gallbladder related to acute cholecystitis. In addition, we review other rare biliary causes of GOO.

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Introduction

Gastric outlet obstruction (GOO) may result from any pathologic process that results in either intrinsic or extrinsic obstruction of the pyloric channel or duodenum. Intrinsic processes such as peptic ulcer disease, caustic ingestion or Crohn's disease may cause inflammation, edema, strictures and chronic scarring of the pyloric channel or duodenal bulb [1]. Extrinsic inflammatory processes in the region of the pylorus such as acute pancreatitis or pancreatic fluid collections

may cause significant surrounding edema and stranding that may result in compression and displacement of adjacent structures [1]. Malignant obstruction may also occur via local invasion or mass effect from extensive metastatic lymphadenopathy.

The prevalence of GOO is believed to have waned in recent years due to improved treatment of peptic ulcer disease, its leading etiology [1,2]. With this decline, the etiology of GOO is changing with current clinical data estimating that approximately 50%-80% of all GOO cases in the modern era are attributable to malignancy [3,4]. The most common

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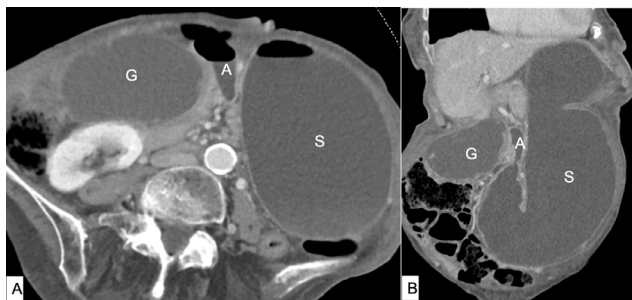


Fig. 1 – Axial (A) and Coronal (B) contrast-enhanced CT images showing marked, fluid-filled dilatation of the stomach (S) with compression of the gastric pylorus/first portion of the duodenum (A) by the dilated gallbladder (G) with irregular gallbladder wall thickening.

malignancy to cause GOO is primary gastric adenocarcinoma [5]. Other cancers that have been reported to be associated with GOO include pancreatic and gallbladder malignancy [5]. Rarely, a benign mass such as a duodenal lipoma can predispose to gastric outlet obstruction [6]. Here, we report a rare case of GOO involving acute cholecystitis and complete resolution of symptoms after cholecystectomy tube placement.

Case Report

A 101-year-old woman with a past medical history of heart failure, hypertension, chronic pulmonary emboli, atrial fibrillation, and scoliosis with accentuated thoracic kyphosis presented to the emergency department with 3–4 days of epigastric pain, persistent nausea, abdominal distension, and inability to tolerate anything per mouth. Per report, she was alert and oriented, and in no acute distress on presentation. Initial physical was notable for a mildly distended abdomen with tenderness to palpation in the epigastric region. Lab values were notable for mild leukocytosis (12.3 K/uL), hyponatremia (126 mEq/L), hypokalemia (3.2 mEq/L) and hypochloremia (3.5 mEq/L). Computed tomography (CT) (Fig. 1) revealed a markedly dilated, fluid-filled stomach with decompressed small bowel, suspicious for gastric outlet obstruction. The gallbladder was dilated with an irregularly thickened wall and mass effect on the adjacent gastric pylorus and proximal duodenal bulb.

Given her CT findings, the patient was made NPO and surgical consultation was obtained. Multiple attempts by the emergency department and surgery staff to place a nasogastric tube for emergent gastric decompression were unsuccessful. This was presumed to be secondary to an extremely tortuous distal esophagus as well as severe thoracolumbar scoliosis. Interventional radiology emergently decompressed the gallbladder with a percutaneous cholecystostomy tube resulting in resolution of the patient's nausea and abdominal distension (Fig. 2 A and B). A CT performed the following day demonstrated a decompressed stomach (Fig. 3 A and B), confirming the distended gallbladder as the etiology of gastric outlet obstruction. Following gallbladder decompression,

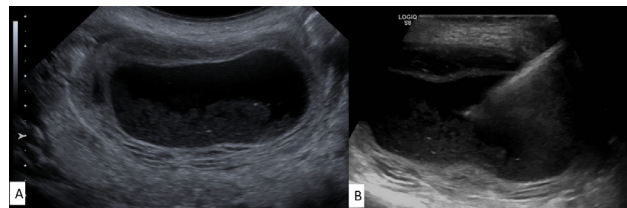


Fig. 2 – Ultrasound images (A, B) demonstrating intraluminal sludge and marked gallbladder wall thickening, compatible with acute cholecystitis, for which percutaneous cholecystostomy tube was placed (B).

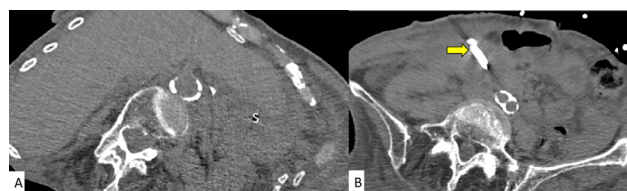


Fig. 3 – Axial noncontrast CT images (A, B) confirm decompressed stomach (S) following cholecystostomy tube (arrow) placement.

the patient's diet was gradually resumed. Follow-up labs also demonstrated interval resolution of leukocytosis and a return to baseline of abnormal electrolytes. Her clinical status improved during the remainder of her hospital course until her discharge.

Discussion

Gastric outlet obstruction in the setting of acute cholecystitis is a rare occurrence with very few cases documented in medical literature. Acute cholecystitis refers to inflammation of the gallbladder which predominantly occurs due to gallstone disease. Gallstones may lead to obstruction of the cystic duct by stones trapped in the spiral valve of Heister [7] and gallbladder distension. Subsequent gallbladder distension, inflammation and edema may result in significant external pressure and displacement of other surrounding visceral structures, which may cause GOO, as seen in our patient.

Clinical manifestations of GOO are variable and often nonspecific. Our patient's chief symptoms of epigastric pain, abdominal distension and nausea have been shown to be commonly associated with GOO, but similar symptoms are frequently seen in acute cholecystitis. In one series of 49 patients, many of whom had malignancy as the source of GOO, the most common clinical features were epigastric pain (94%), vomiting (92%), and weight loss (63%) [8]. In another cohort of 30 patients with benign causes of GOO, the most common presenting features were early satiety (53%) and bloating (50%) [8].

There are multiple potential manifestations of GOO on imaging. Conventional abdominal radiographic findings of gaseous gastric distention, gastric air-fluid levels and paucity of small bowel gas are suggestive but nonspecific. CT may

show similar findings but will often suggest a specific cause of GOO, like it did in this case. CT is also more sensitive for detecting potential complications of GOO including free air, gastric pneumatosis, or free fluid in the abdomen. Fluoroscopic guided upper gastrointestinal studies with barium or water-soluble contrast may also be utilized to differentiate partial from complete gastric outlet obstruction and may give clues about the underlying etiology. Further evaluation with upper endoscopy may be performed if imaging findings are inconclusive.

Another rare biliary association to consider in the setting of gastric outlet syndrome is Bouveret's syndrome, a variant and proximal form of gallstone ileus [9]. Bouveret's syndrome is characterized by migration of a gallstone into the intestinal lumen via a bilio-enteric fistula [9]. It is thought to be most likely due to long-standing pressure necrosis from stones rather than acute cholecystitis. Multiple bilio-enteric fistulous connections have been reported in the literature including cholecystoduodenal, cholecystocolic, cholecystogastric, and choledochoduodenal subtypes [9]. The most common of these is the cholecystoduodenal subtype which reportedly occurs approximately 60% of the time [9] and may result in gastric outlet obstruction. Unlike Bouveret's syndrome, typical gallstone ileus usually occurs more distally and may result in mechanical small bowel obstruction secondary to impacted stones. Although it is one of the rarest forms of mechanical bowel obstructions, reports indicate that it is a cause of nonstrangulating mechanical small bowel obstruction in 1% to 4% of all patients and as high as 25% in elderly populations [10]. When it occurs, obstruction most commonly occurs at the level of the terminal ileum (60%-70%) [9].

Conclusion

While gallstones have been implicated in rare cases of gastric outlet obstruction as noted above, to our knowledge, there is no published report of gallbladder dilatation from acute cholecystitis causing gastric outlet obstruction. It is likely that it was a combination of gallbladder distension and inflammation as well as the patient's scoliosis that contributed to these findings. We believe our case gives credence to the idea that

several disease processes may be implicated in GOO as well as exemplifying how imaging findings were critical in the emergent setting to help identify the underlying cause, which led to prompt treatment. Clinicians should be aware of this uncommon phenomenon to ensure effective and timely treatment in this patient population and prevent adverse outcomes.

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