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Case report

Gastric outlet obstruction - looking for a syndrome: Bouveret or Mirizzi?

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ABSTRACT

Introduction and importance: Gastric outlet obstruction can result from any pathological process that causes intrinsic blockage or extrinsic pressure on the distal stomach and duodenum. Gallstone related gastric outlet obstruction is a well-known entity classically due to a cholecystoenteric fistula formation.

Case presentation: We present here a case of a 36-year-old man who presented with right upper quadrant abdominal pain associated with marked nausea and vomiting. Abdominal CT scan done in the emergency department revealed a large impacted infundibular gallstone with signs of acute cholecystitis, associated with prominent gastric distention. Gastric outlet obstruction was due to stenosis at the duodenal level from external compression by the large impacted stone with no evidence of fistula. Laparoscopic cholecystectomy was performed with total resolution of symptoms.

Clinical discussion: Gastric outlet obstruction can be secondary to many etiologies, and notably gallstone disease. Classically this is due to formation of a cholecystoenteric fistula and intrinsic obstruction by the migrated stone. Our case is unique in that a large impacted infundibular gallstone caused gastric outlet obstruction with absence of any fistula or gallstone migration.

Conclusion: Gastric outlet obstruction due to external compression by a non-migrated gallstone is a rare undescribed entity. Surgical treatment should not be delayed to prevent complications and fistula formation.

1. Introduction

Gastric outlet obstruction (GOO) is a clinical consequence of any pathological process causing intrinsic or extrinsic impediment of the distal stomach, pyloric channel or duodenum [1]. The most common presenting clinical manifestations include epigastric pain, nausea with or without vomiting, early satiety, abdominal distension or bloating and weight loss [2]. GOO due to an extrinsic mechanical obstruction secondary to acute calculous cholecystitis, in which the stone impacted in the gallbladder infundibulum herniates and compresses the duodenum, has never been reported. We herein report a rare case of GOO, secondary to an impacted infundibular gallstone, that resolved after laparoscopic cholecystectomy. The case report is presented in line with the SCARE criteria [3].

2. Case presentation

A 36-year-old gentleman, with no past medical or surgical history, presented to the emergency department for recurrent postprandial right upper quadrant (RUQ) pain of few weeks duration. However, this pain became associated with nausea, vomiting and decreased tolerance to oral food intake since few days, which compelled him to seek medical help.

On physical examination, the patient had a normal body mass index (BMI) and his vitals were within the normal range. He had non-icteric sclera. Abdominal examination revealed tenderness in the right hypochondrium and epigastric region along with abdominal distension. Abdominal radiography showed 1.8 cm calcification in RUQ with absence of small bowel distention. An abdominal computed tomography (CT) scan with intravenous (IV) contrast revealed the presence of a 1.8 cm gallstone in the gallbladder infundibulum exerting a mass effect on the proximal duodenal bulb. The gallbladder had a thickened wall with mild parietal contrast intake.

The stomach was fluid-filled and prominently dilated with a decompressed small bowel, thus raising the suspicion of gastric outlet obstruction (Fig. 1).

Laparoscopic cholecystectomy was performed under general anesthesia. During surgery, the infundibulum was dissected off the duodenal

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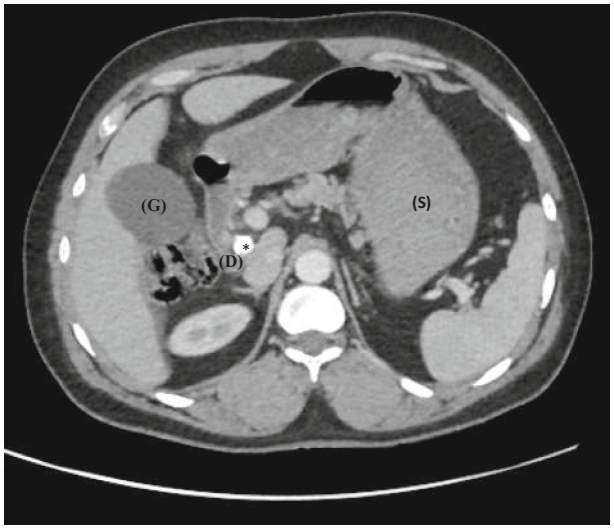

Fig. 1. Abdominal computed tomography (CT) scan reveals the presence of a 1.8 cm gallstone (*) in the gallbladder infundibulum with mild parietal contrast intake (G) compressing the duodenal bulb (D) associated with prominent gastric distention (S).

bulb. Pathology report revealed the presence of acute gangrenous cholecystitis and mural fibrosis, with no evidence of malignancy. By the second post-operative day, a CT scan was repeated and showed a decompressed stomach. This confirms the herniated impacted infundibular gallstone as an etiology for GOO. The patient had an uneventful recovery and was discharged.

3. Discussion

The present case highlights a very rare form of gastric outlet obstruction; GOO is the outcome of hindrance to the distal stomach, pyloric channel or duodenum due to any pathological setting [1]. Signs of GOO are often non-specific and may manifest similar to cholecystitis or with less specific presentation as bloating, early satiety, weight loss, among others [2]. The onset and severity of clinical features of GOO vary widely according to the etiology of obstruction, ranging from chronic mild to acute severe symptoms [4]. One cohort study showed that benign causes of GOO have bloating and satiety as the predominant clinical features [5]. On the other hand, the primary presenting symptoms are epigastric pain, vomiting and weight loss in patients with a malignant etiology [5]. Over the past decades, the prevalence of each etiology behind GOO have changed. Due to the development of H_2 blockers and proton pump inhibitors, peptic ulcer disease is no longer a common cause of GOO in the modern era [6]. As a consequence, malignancy now is behind more cases of GOO [5,7–9]. In rare occasions, a dislodged gallstone in the gastric or intestinal lumen via a cholecystoenteric fistula leads to gastric outlet obstruction, a condition known as Bouveret syndrome [10]. In this disorder, the gallstone reaches the intestinal lumen through an abnormal fistulous tract between the gallbladder neck and gastro-intestinal (GI) tract [10,11]. The fistula is most likely due to persistent pressure necrosis from gallstones impacted in the gallbladder neck or cystic duct compressing against the surrounding gallbladder wall, thus paving their way into the GI lumen [12,13]. With respect to intestinal occlusion secondary to a migrated gallstone via a cholecystoenteric fistula, another entity with a similar pathophysiology is Mirizzi type V syndrome [14]. In both syndromes, Bouveret and Mirizzi type V, intrinsic mechanical obstruction is the culprit of the disease, however; in the former, the obstruction occurs in the proximal part leading to GOO, whereas, in the latter, occlusion occurs due to an impacted gallstone in the distal portion of the GI tract, known as gallstone ileus [10,11,14,15].

Interestingly in our patient, the inflamed gallbladder was externally compressing the duodenum with the absence of any fistula or migrated gallstone, therefore ruling out the diagnosis of Bouveret syndrome. In our case, the subsequent inflamed and distended gallbladder along with its impacted infundibular gallstone herniated with time and became low-lying. This surrounding visceral displacement caused an external compression on the duodenal bulb, thus serving as the etiology behind GOO. Nevertheless, this pathophysiology resembles Mirizzi type 1, where the herniated infundibulum compresses the common bile duct (CBD) and causes jaundice in the absence of any fistula. The inter-occurrence of gallstone-related GOO, with absence of a fistula in the face of an impacted infundibular gallstone, made our case a chimeric combination of pre-Bouveret and Mirizzi type 1-like occlusion (Fig. 2).

4. Conclusion

Although gallstones have been implicated in rare cases of gastric outlet obstruction, it is mainly either due to fistula formation or gallstone migration. To the best of our knowledge, we presented the first case describing a large gallstone impacted in the infundibulum causing GOO due to extrinsic compression of the duodenal bulb, in the absence of any fistula. We believe our case implicate the necessity to consider gallstones as a possible etiology of GOO, even in the absence of migration or fistula. Physicians and surgeons should be aware of this uncommon occurrence to provide a timely effective management and prevent further complications.

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Availability of data and materials

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Ethical approval

N/a.

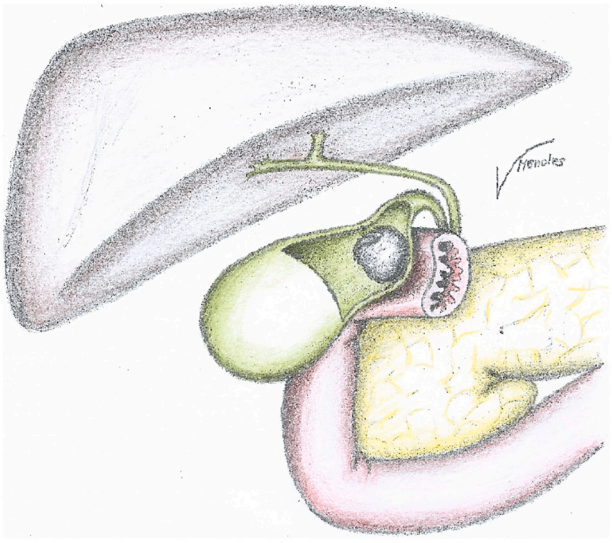

Fig. 2. The drawing demonstrates a large gallstone impacted in the infundibulum causing an extrinsic compression on the duodenum, in the absence of any fistula.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Registration of research studies

N/a.

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CRediT authorship contribution statement

Nour Ibrahim, Marwan Zein and Haidar A. Nasser collected the data of the patient and wrote the first draft of the manuscript. Nour Ibrahim performed the review of literature. Amal A. Nasser and Vanessa Mendes edited and reviewed the manuscript. Vanessa Mendes drew Fig. 2. All authors have read and approved the final manuscript.

Declaration of competing interest

The authors declare no conflict of interest.

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