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Case Report

Gallbladder volvulus mimicking a cardiac event – an unusual presentation

Abstract

Gallbladder volvulus (GV) is a rare surgical emergency in which the gallbladder twists on its pedicle and becomes gangrenous. It most commonly presents with symptoms similar to acute cholecystitis. Investigations are seldom diagnostic, with both pathology tests and imaging often unremarkable, and as such it is often diagnosed intraoperatively. Given the potential consequence of a ruptured gallbladder leading to biliary peritonitis, it is an important condition to consider and suspected cases should proceed to cholecystectomy as soon as possible. We report the case of an elderly female patient who initially presented as having an acute coronary syndrome then was found intraoperatively to have GV, and discuss clinical points of the disease.

Introduction

Gallbladder volvulus (GV), also known as gallbladder torsion, is a rare occurrence and difficult to diagnose in patients presenting with abdominal pain, even with the aid of imaging. First reported in 1898 by Wendel as a "floating gallbladder", approximately 500 cases have been described in the literature since [1-3]. Given the risk of ischaemia with subsequent gangrenous gallbladder progressing to perforation, it is important to be aware of this as a differential diagnosis and proceed to emergency cholecystectomy in suspected cases.

Case Report

An 84-year-old woman presented to our emergency department with sudden onset central chest and epigastric pain while at rest, associated with dyspnoea, dizziness, diaphoresis, nausea and vomiting. This was on a background of a previous cardiac event for which she had a permanent pacemaker, and was on dual antiplatelet agents. Given her significant cardiac history, she was admitted under the medical team and worked up for a potential NSTEMI. Despite empirical treatment with 300mg aspirin, GTN and multiple doses of analgesia, her pain worsened. Serial troponins returned as negative, however she had an elevated white cell count at 11.4 (x10 ^9/L), neutrophilia of 9.6 (x10^9/L), C-reactive protein of 88.9 mg/L and lactate was 2.7 mmol/L. Serum electrolytes and liver function tests were normal. She was noted to have a history of fundoplication for hiatus hernia and previous oesophageal dilatation for strictures, and although her abdomen examined as soft and non tender, a CT abdo pelvis was ordered to exclude incarcerated hiatus hernia, aortic dissection, ischaemic gut and mesenteric artery occlusion. The CT was reported as having haziness in the fat of the mid upper abdomen adjacent to the gallbladder with possible gallbladder infarction, but no gross gallbladder wall thickening, and there were no other abnormalities (Figure 1). Her pain then migrated to her abdomen the next day and she was referred to our surgical team, with findings of marked tenderness on palpation of the general right side of her abdomen including a positive Murphy's sign. Observations were stable and she was afebrile throughout. A second opinion on the CT scan was sought, and it was advised that the findings were consistent with acute cholecystitis with recommendation for an abdominal ultrasound. Intravenous ceftriaxone and metronidazole was commenced, and she was noted to be coagulopathic (INR 2.0, PTT 22.5 seconds, APTT 41.6 seconds, fibrinogen 4.7 g/L). Whilst awaiting surgery the patient underwent an abdominal ultrasound scan. This was reported as having probe tenderness to the gallbladder, a thickened gallbladder wall (4mm) but no increased vascularity or gallstones, and the common bile duct was 8mm (Figure 2). The patient proceeded to a laparoscopic converted to open cholecystectomy due to poor visualisation from significant adhesions. A right-sided upper transverse abdominal incision was performed and the gangrenous gallbladder was identified and easily externalised, and noted to be torted over 180 degrees clockwise at the region of the cystic duct and artery (Figures 3,4). Adjacent bowel and mesentery appeared normal. Post operatively she made an uncomplicated recovery and

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was discharged home in five days, with follow up two weeks later also unremarkable. Histopathology confirmed complete gangrenous necrosis of the gallbladder wall consistent with the effects of torsion, and no gallstones.

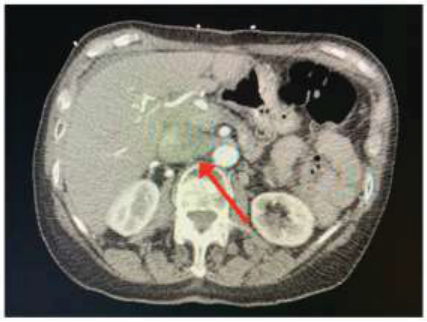

Figure 1: Axial image of CT abdomen pelvis with intravenous contrast showing non-specific mild haziness of fat adjacent to the gallbladder.

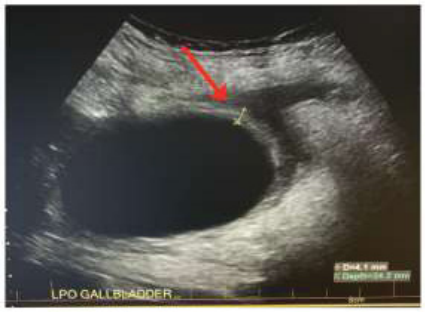

Figure 2: Ultrasound image showing diffuse thickening of the gallbladder wall to 4mm with pericholecystic fluid.

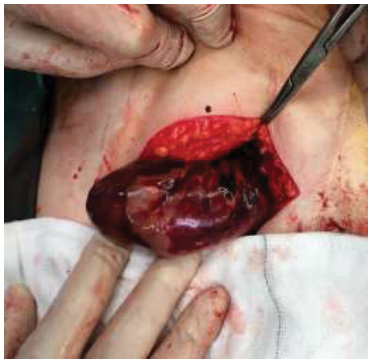

Figure 3: Externalised torted gallbladder with evident gangrenous gallbladder wall.

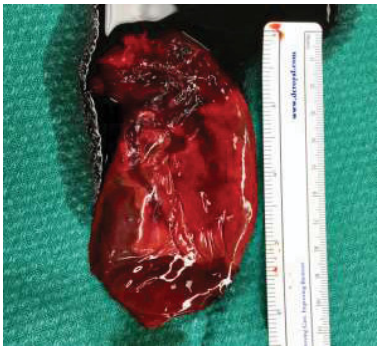

Figure 4: Gallbladder specimen measuring approximately 10cm in length, with evident areas of gangrenous necrosis.

Discussion

GV is the rotation of the gallbladder on its cystic artery and cystic duct mesenteric pedicle, causing both impaired arterial supply and biliary outflow, which can in turn lead to infarction and perforation. It occurs rarely, with an incidence of 1 in 365, 520 hospital admissions [4]. Torsion can be classed as complete (over 180 degrees) or incomplete (less than 180 degrees) [2].

The cause of GV remains unknown, however it is associated with several factors. The most common demographic in which GV occurs is in elderly females [5], such as in our case. Risk factors for this condition include age over 70 years old, female to male sex ratio of 3:1, weight loss, liver atrophy, spinal deformities and anatomical variations such as a lengthy mesentery [2,4,5]. Additionally, peristalsis of adjacent viscera such as the stomach, duodenum and transverse colon is thought to contribute to rotational movement of the gallbladder, with a theory that the first two lead to clockwise rotation and that anticlockwise rotation results from the latter [2,5]. Interestingly, cholelithiasis is not significantly associated with this phenomenon, with gallstones present in only 25-50% of patients [3,4].

Although it can occur in all age groups, with some paediatric cases reported [4,6], advanced age is an important characteristic as it relates to visceroptosis, a concept in which mesentery atrophies, elongates and becomes more flexible over time, which can cause the gallbladder to become less firmly attached to its liver fossa, or "float", and predispose it to torsion [2,4].

Patients most commonly present with symptoms mirroring acute cholecystitis [6,7]. On clinical examination there may be a positive Murphy's sign or a palpable mass in the right upper quadrant [2,6,7]. Other differential diagnoses include biliary colic, ascending cholangitis, pyelonephritis and right-sided colon malignancy. In our search of the literature, no cases presented initially as a suspected acute coronary syndrome – whether this can be attributed to clinician bias, subjectivity on reporting of symptoms by the patient or radiation of pain to the chest and epigastrium or the combination of these potential factors is uncertain.

There is no clear consensus on which investigations should be performed to effectively diagnose GV. Pathology tests such as full blood count, liver function tests, C-reactive protein and lactate may be unremarkable [2-4]. Additionally, imaging with CT, ultrasound or MRI is rarely diagnostic, though some signs have been described to aid identification of GV. Ultrasonography may show a diffusely thickened and hypoechoic gallbladder wall indicating inflammation and/or gangrene, a gallbladder not adherent to its bed, and multiple linear echoes converging to form a cone tip at the gallbladder neck [8]. CT may show a distended gallbladder outside of its fossa with wall thickening and oedema, with the cystic artery classically located to its right, together torsion of its cystic pedicle causing a "whirl sign" [6,9]. MRI is the most sensitive, with T2 weighted images useful in showing gallbladder wall necrosis, but this is rarely used in the emergency setting [3,9]. As such, GV is often misdiagnosed and usually found intraoperatively, with only 9.8% of all cases diagnosed prior [10].

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GV is treated with urgent cholecystectomy, either laparoscopically or via laparotomy. Prognosis is good if the patient proceeds promptly to an operation, with mortality reduced to less than 5% [4]. Of note, given the risk of biliary peritonitis secondary to gallbladder perforation, measures should be taken to expedite surgery in a safe manner in those who are at high anaesthetic and surgical risk (i.e. those on anticoagulation and/or antiplatelet agents).

Conclusion

GV should be considered in patients presenting with acute abdominal pain with other potentially masking significant medical history, especially in the elderly population. Blood tests and imaging are not guaranteed to be diagnostic as there are no pathognomonic features, and interpretation of imaging may differ between clinicians, demonstrating the difficulty in diagnosing this condition pre-operatively. Surgery should not be delayed due to anticoagulation or coagulopathy as there is risk of perforation and subsequent biliary peritonitis.

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