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Case Report

Acute acalculous cholecystitis after blunt abdominal trauma

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ABSTRACT

Severe trauma is a recognised risk factor for developing acute acalculous cholecystitis (AAC), which is associated with a high mortality rate. The sonographic features of AAC have been well described in the literature and debate continues over its definitive management. We present the case of a 52-year old man who presented with right upper quadrant pain three days after sustaining blunt abdominal trauma. Leucocytosis with neutrophilia and an elevated CRP level were noted on haematological investigation. The liver function tests and amylase were within normal limits. Radiological investigations using ultrasound scan and computed tomography demonstrated gallbladder features suggestive of AAC. The patient was managed with intravenous antibiotics and simple analgesia and did not require any surgical or radiological intervention. Follow-up ultrasound scan, performed three months after the initial presentation, demonstrated a tortuous acalculous gallbladder. Despite a delayed presentation and a low Injury Severity Score, a high index of clinical suspicion for AAC should be maintained given the history of significant trauma.

Keywords: Abdominal trauma, Acalculous cholecystitis, Ultrasound

INTRODUCTION

Acalculous cholecystitis is clinically indistinguishable from calculous cholecystitis in its presentation. Acute acalculous cholecystitis (AAC) is associated with a significant mortality rate and therefore early diagnosis and intervention are vital to management.[1-3] We present the case of a man who was admitted with AAC three days after a closed, blunt traumatic upper abdominal injury. We discuss the multifactorial pathogenesis, the diagnostic radiological features and the benefits of operative versus non-operative management of AAC. The patient made an uneventful recovery with non-operative management.

CASE REPORT

A 52-year old caucasian painter-decorator presented to the emergency department with an acute four hour history of sudden-onset right upper quadrant abdominal pain, nausea, but no vomiting. The colicky pain's onset was at rest and it radiated under the right subcostal region to the inferior angle of the right scapula. Of note, the patient was a pedestrian involved in a road traffic accident three days prior to onset of abdominal pain. While crossing the road, he was hit by a motorcyclist riding at about 20 mph. The motorcycle's handle bars impacted into either side of the epigastric region. The patient mobilised at the scene of the accident and decided not to seek medical attention as he felt generally well.

The patient was a hypertensive, treated with Ramipril. He smoked 10 cigarettes a day for 30 years and drank alcohol on a social basis. On physical examination, he was apyrexial, but tachycardic with no signs of anaemia or jaundice. There was no respiratory and cardiovascular compromise. Inspection of the abdomen revealed a horizontal, well demarcated, rectangular bruise bilaterally overlying the sixth to eight ribs anteriorly. No other abdominal skin bruising was noted. The abdomen was generally soft and tenderness, but no peritonism, was elicited in the right upper quadrant. Murphy's sign was positive.

Full blood count revealed a white cell count of 12.9×109/L with neutrophilia (11.3×109/L) and platelets of 195×109/L. The haemoglobin, urea and electrolytes were within normal limits. Bilirubin was mildly elevated at 30umol/L, but ALP, ALT and amylase were within normal limits. The CRP was markedly raised at 118 mg/L. Dipstick urinalysis was negative for blood.

The patient was commenced on intravenous Co-amoxiclav and Metronidazole based on the clinical suspicion of acute cholecystitis. Chest and abdominal radiographs were unremarkable. Following an abdominal ultrasound (US) scan, the radiologist concluded that there was sonographic evidence of cholecystitis but no calculi evident, therefore suggesting acalculous cholecystitis.

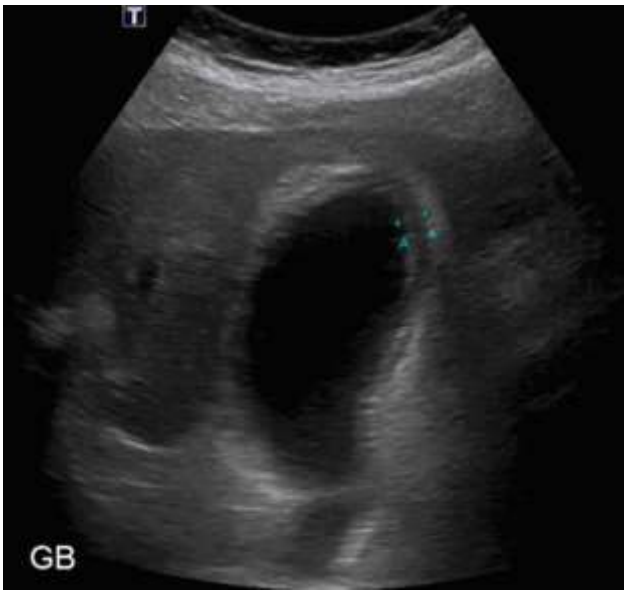

Figure 1: Abdominal Ultrasound scan.

As shown in Figure 1 US scan demonstrating a thick-walled gallbladder with a maximum measured wall thickness of 5 mm, and gallbladder wall oedema. No definite calculus was seen within the gallbladder. The common bile duct was dilated up to 7 mm but no intrahepatic duct dilatation was seen. Normal blood flow was demonstrated in the hepatic and portal veins.

The next morning, the patient complained of markedly worse epigastric pain not relieved by simple analgesia. There were no associated symptoms. The patient remained apyrexial but tachycardic. The abdomen was still soft but marked tenderness was now elicited in the epigastrium. A gastrograffin meal, small bowel study and a computed tomography (CT) scan of the abdomen and pelvis were performed. These investigations excluded duodenal and other occult intra-abdominal visceral injury. On CT scan a thickened gallbladder wall with pericholecystic fluid, but no cholelithiasis was observed as shown in Figure 2. Additionally, the common bile duct was dilated at 9mm with no intrahepatic duct dilation

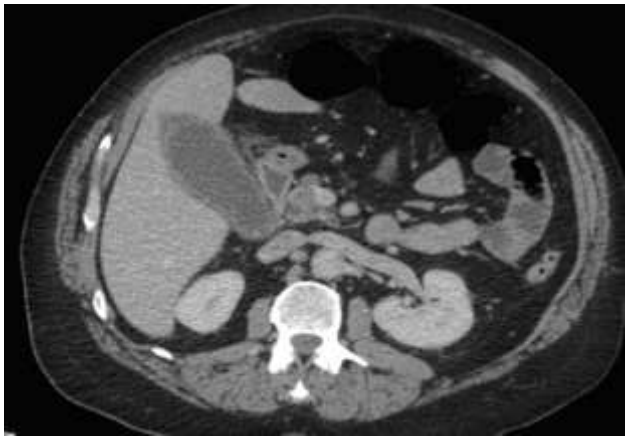

Figure 2: CT scan of the abdomen.

The patient's symptoms improved 24 hours into the admission. The pain was controlled with a combination of simple and low-dose opiate analgesia. As a result of the clinical improvement the patient self-discharged, thereby not completing his course of oral antibiotics.

A follow-up abdominal US scan showed a very tortuous gallbladder with a variable wall thickness up to 3 mm. A non-shadowing soft tissue area adherent to the posterior mid-gallbladder wall was noted likely to be adherent organised haematoma. The patient declined elective laparoscopic cholecystectomy.

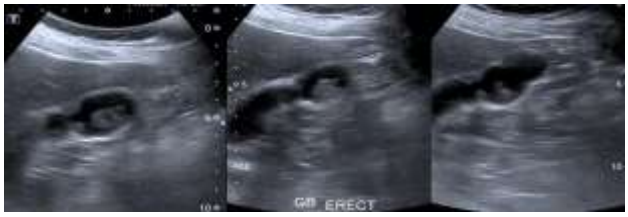

Figure 3: Follow-up abdominal US.

Figure 3 reveals an ultrasound scan demonstrating a tortuous gallbladder with a 17 mm × 15 mm × 12 mm non-shadowing soft tissue area adherent to the posterior mid-gallbladder wall that was not present in the previous imaging. The shadowing demonstrated no vascular flow or focal tenderness.

DISCUSSION

Acalculous cholecystitis accounts for 10% of all cases of acute cholecystitis and is characterized by an acute inflammation of the gallbladder in the absence of cholelithiasis.[3] AAC has a multifactorial pathogenesis comprising trauma, recent cardiothoracic surgery or intra-abdominal surgery not involving the gall bladder, shock, burns, sepsis, critical illness requiring intensive care, total parenteral nutrition, opioid analgesia, prolonged assisted ventilation and positive pressure ventilation, multiple blood transfusions, chronic illness, diabetes, hypertension, atherosclerosis.[4-10]

It is clinically indistinguishable from acute calculous cholecystitis and the findings of right upper quadrant pain, fever, leukocytosis and abnormal liver function tests are non-specific clinical findings of AAC. Critically ill patients are more susceptible to developing AAC, but trauma is a well-known risk factor.[4]

In their prospective study, Pelinka et al concluded that the major risk factors of developing trauma-related acalculous cholecystitis were patients with an Injury Severity Score > 12, tachycardia > 120 beats per minute and severe shock needing transfusion of > 12 units of packed red cells.[4] Furthermore, the study identified a longer duration of ventilatory support, high levels of positive end expiratory pressure (PEEP), opioid analgesia and total parenteral nutrition were minor risk factors for developing acalculous cholecystitis after trauma. Risk factors for the patient in our case study include trauma, although tachycardia is the only identifiable major risk factors as suggested from Pelinka et al study, and hypertension.

Gangrene, perforation of the gallbladder, empyema, peritonitis and death are the complications of the AAC. Mortality, based on data from the literature, is quoted as 30% (range 10%-90%).[3] While ischaemic-reperfusion injury is a central pathogenic feature, findings from a number of histopathological studies suggest a complex sequence of pathophysiological events like increased leukocyte migration leading to increased interstitial oedema associated with local microvascular occlusion therefore promoting deeper bile infiltration in the gallbladder wall.[11-15]

US and CT remain the main radiological investigations of choice as both are equally accurate in the diagnosis of AAC.[16-18] Radiologic criteria have also been developed for hepatobiliary iminodiacetic acid (HIDA) scintigraphy.[16] US of the gallbladder in critically ill patients and/or those with severe trauma is the most accurate radiological investigation for diagnosis of AAC. Serial US scanning has a higher sensitivity and specificity than daily blood investigations.[4,19] The most studied diagnostic criteria for US scan are given as below.[2]

Diagnostic triad of acute acalculous cholecystitis

- Gallbladder wall thickness > 3.5 mm
- Sludge
- Hydrops

Minor criteria

- Pericholecystic fluid
- Intramural gas
- Sloughed mucosa

Deitch's study reported the sensitivity and specificity of a 3.0 mm gallbladder wall thickness to be 90% and 100% respectively, but a gallbladder wall thickness of 3.5mm demonstrated a sensitivity of 98.5% and specificity of 80%.[20] Thickening of the gallbladder wall to > 3.5 mm is the most reliable criterion for the diagnosis of acute cholecystitis whereas 3.0 mm is suggestive but not conclusive evidence of acute cholecystitis.[21,12] Our patient's US and CT findings together demonstrated the diagnostic triad of AAC.

For many years, open or laparoscopic cholecystectomy had been the definitive therapy for AAC. Laparoscopic surgery has been favoured due to its diagnostic and therapeutic potential, with a diagnostic accuracy of 90-100%.[22,23] Furthermore, laparoscopic cholecystectomy is less invasive and has a similar morbidity and mortality compared with open cholecystectomy.[3] More recent studies argue that percutaneous cholecystostomy is now the definitive management of AAC as it is associated with a lower morbidity than emergent cholecystectomy and can be used an effective bridge to elective cholecystectomy.[24]

Our patient's case demonstrates that despite a delayed presentation and a low injury severity score, a high index of clinical suspicion for AAC should be maintained given the history of significant trauma.[25] Furthermore, this case demonstrates the classic radiological (US and CT) features of AAC and the successful non-operative management of the condition.

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