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Acute Acalculous Cholecystitis and Small Bowel Obstruction: A Case Report and a Review of the Literature

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He presented with what was suggestive of a small bowel obstruction secondary to an incarcerated inguinal hernia. Subsequent investigation after operative management of the hernia denoted that it was all secondary to acalculous cholecystitis; we thus present our case and pitfalls in management.

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Acute Acalculous Cholecystitis and Small Bowel Obstruction: A Case Report and a Review of the Literature

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I. Case Report

73-year-old male presented with a four day history of abdominal pain, he had been doing heavy lifting for some time. Of note his significant medical history includes: peptic ulcer disease and a current smoker.

On review in the emergency department he was unwell with signs of sepsis. He was febrile, and guarding to examine in all quadrants particularly in the right side; Murphy's sign however was negative.

Initial investigations denoted a raised white cell count of 17 x 10^9 and CRP of 195mg/L. Abdominal Computed Tomography (CT) and erect chest x-ray revealed dilated loops of small bowel with multiple air/fluid levels (see image 1 below). Given the investigations denoting likely a small bowel obstruction secondary to an incarcerated hernia and the general decline in the patient operative management was sought.

Intraoperatively, it was revealed the femoral hernia only contained fat which was subsequently repaired. An indirect inguinal hernia was also identified, with the cord containing a lipoma. This was subsequently ligated and dissected.

Given non-progress to improvement post operatively the patient had further investigations, which incorporated an ultrasound initially and CT Cholangiogram which denoted acute acalculous cholecystitis (AAC) (see image 2). He also had a dilated biliary system however with normal liver function. As further sepsis continued and gram negative bacteraemia on blood cultures an emergency open cholecystectomy was conducted.

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Intraoperatively the cystic stump was controlled with closure with PDS suture and double clip with clip applicator, however the patient developed a post-operative biliary leak despite these measures.He was later transferred to a tertiary centre for endoscopic retrograde cholangiopancreatography (ERCP) and further stenting. The patient made a successful recovery.

II. Discussion and Literature Review

AAC is defined as an necroinflammatory disease of the gallbladder with a multifactorial pathogenesis.[1-3] We are not aware of any reported incidences of small bowel obstruction secondary to AAC.

AAC has been recognised for more than 150 years, despite this it remains an elusive diagnosis. Clinically, AAC is indistinguishable from acute calculous cholecysitis.[2, 4-6]This is likely because of the complex clinical setting in which this entity develops.[2] Acalculous cholecystitis is typically seen in patients who are hospitalized and critically ill, though it may also be seen in the outpatient setting.[3] Patients may also present with the complications of AAC including gall bladder necrosis and sepsis. It accounts for approximately 10% of all cases of acute cholecystitis and is associated with morbidity and mortality (10-90%).[2, 3]AAC occurs in about 0.2% to 0.4% of all critically ill patients usually about 60 years of age.[2, 4, 6, 7] There are multiple risk factors for developing AAC and these are listed in Table 1.[1-4]

Laboratory tests in patients with AAC are nonspecific[3]. The ultimate diagnosis of AAC usually rests on imaging.[2]

Ultrasonography (US) is usually the first test obtained in patients suspected of having acalculous cholecystitis.It has good sensitivity (100%) and specificity (90%) for diagnosing acalculous cholecystitis.[2, 3]Thickening of the gallbladder wall is the most reliable diagnostic feature.[3, 6]

CT is useful for the diagnosis of AAC.[2] The accuracy of CT scanning appears to be similar to that seen with ultrasonography.[3]

Cholescintigraphy can be useful in patients who are stable thatcan cope with the transport and in whom the diagnosis is unclear after US [3]. The sensitivity of

cholescintigraphy can be as low as 67% to 100%.[2, 3] The specificity ranges from 38% to 100%.[2]

However, cholescintigraphy takes hours to perform, so it is not recommended in critically ill patients in whom a delay in therapy can be potentially fatal.[2, 3]

Initial management of AAC includes empiric antibiotics targeting gram-negative and anaerobic pathogens after blood cultures have been taken.[3, 8]

Cholecystectomy is considered to be definitive therapy for AAC.[1-4, 9, 10] However, cholecystostomy can also be used as first line therapy, but there is debate whether this is appropriate.[2, 3, 9, 10]

Cholecystostomy may provide time to optimise the patient's condition for surgery.[2, 9, 11, 12] Failure to improve by cholecystostomy (defined by persistent fevers, signs of sepsis, or evidence of new multiorgan dysfunction) may be due to gangrenous cholecystitis, catheter dislodgement, bile leakage resulting in peritonitis, or an incorrect diagnosis of acalculous cholecystitis.[3] These patient's require will require cholecystectomy.[3]

However, if the gallbladder wall is ischaemic, necrotic, or perforated, cholecystostomy is not appropriate.[2, 3, 9]

When neither cholecystostomy nor cholecystectomy can be performed, direct endoscopic retrograde cholangiopancreatographic gall bladder drainage can be attempted to assist decompression. There have been isolated cases of success [3]. However, this generally is believed to be inferior therapy and seldom is performed.[2, 4, 13, 14]

Acalculous cholecystitis is difficult to diagnose, but an early correct assessment is essential to successful treatment as AAC.[2, 3]

Our case highlighted challenging goals, initially with small bowel obstruction presenting with likely secondary to an incarcerated hernia.

However further description and management noted this was all secondary to asmall bowel obstruction caused by possibly an inflamed gall bladder.In terms of clinical suspicion, possibly having done an ultrasound prior to laparotomy may have changed treatment methods, however this diagnostic dilemma exists. Our patient did not have a gallstone ileus, and given the multifactorial problems encountered it was a challenge.

Clinical suspicion of other pathologies should always be in a clinician's mind when assessing these unwell patients.

Table 1 : Risk factors for AAC. Adapted from Huffaman and Schenker [2].

Commonly associated risk factors for AAC
Trauma: leading to hospitalization; some factors particularly leading to the diagnosis of AAC in trauma are blood transfusions (>12 units), Injury Severity Score > 12, and tachycardia (> 120 bpm)
Recent surgery (unrelated to gall bladder, abdominal, or extra-abdominal, 13 to include cardiopulmonary disease)
Shock of any kind
Burn
Sepsis
Bacterial—Brucellosis, Q fever, leptospirosis, tuberculosis, scrub typhus, salmonellosis, cholera
Fungal—Candida (albicans, glabrata, torulopsis)
Parasitic—Cyclospora, microsporidia, Plasmodium falciparum and vivax, Schistosoma mansoni
Viral–Cytomegalovirus, Ebstein–Barr virus, 15a Dengue virus
Critical illness (any patient requiring ICU care)
TPN
Prolonged fasting
Rarely associated risk factors for AAC
Hypovolemia
Postendoscopic retrograde cholangiopancreatography
Increased length of hospital stay
Immunodeficiency: acquired immune deficiency syndrome, transplant
Chronic illness: diabetes, hypertension, atherosclerotic disease, obesity
Vasculitides: Churg–Strauss, giant cell arteritis, Henoch–Schönlein purpura, polyarteritis nodosa, lupus
Obstruction: ampullary stenosis, ascariasis, echinococcus, tumor (extrinsic or intrinsic)

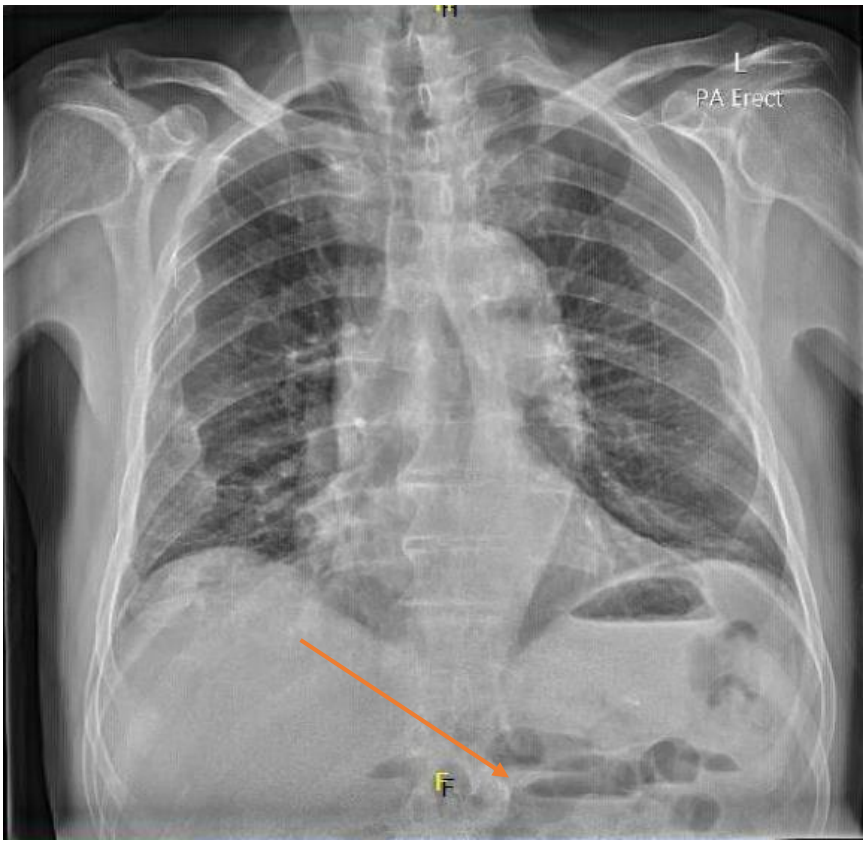

Image 1 : Chest x-ray displaying multiple air fluid levels

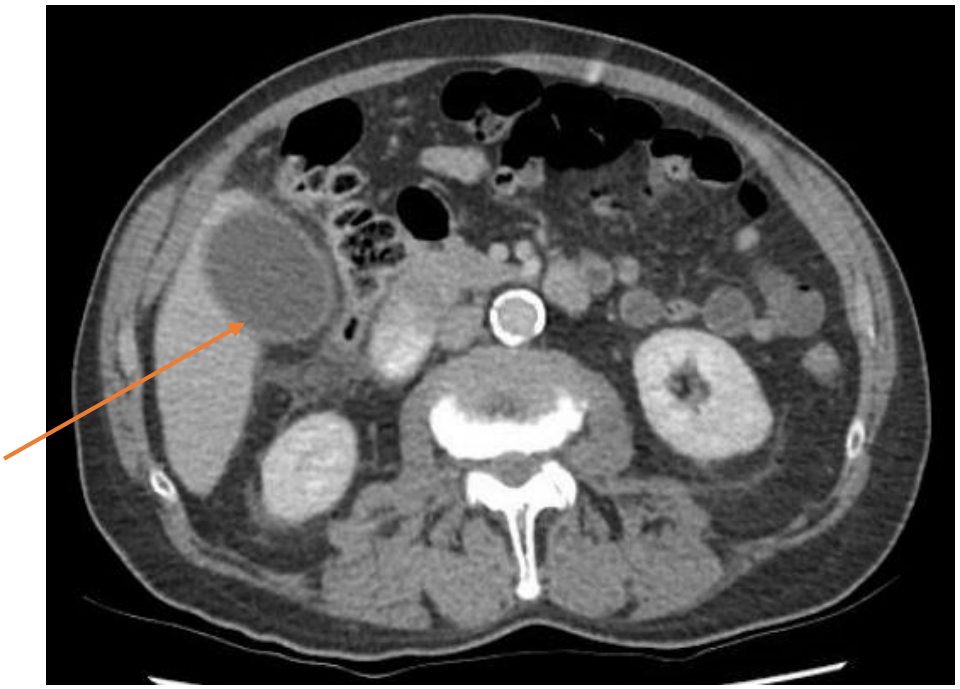

Image 2 : A distended gallbladder can be seen with thickened walls

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