SYSTEMATIC / NARRATIVE REVIEW ARTICLE

CT and MRI findings of acute calculous cholecystitis and its complications in Singapore: A pictorial review

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ABSTRACT

Introduction: Acute cholecystitis (AC) is a common problem encountered in surgical practice. This occurs due to obstruction of the cystic duct by calculi resulting in inflammation of the gallbladder. Increasingly, contrast enhanced computed tomography (CECT) and Magnetic Resonance Imaging (MRI) scans are being used for assessment. While the imaging features of AC are well recognized and extensively described in the literature, radiological features of the rarer complications related to AC such as pseudoaneurysm formation and gallbladder volvulus are less well known. We aim to describe these rarer findings in our pictorial review, to better educate the clinician and radiologist, such that timely diagnoses can be reached, and relevant management can be affected.

Methods: A collection of cases showing the common acute gallbladder pathologies and complications such as acute cholecystitis, gangrenous cholecystitis, emphysematous cholecystitis, haemorrhagic cholecystitis, Mirizzi's syndrome, gallbladder perforation and abscess formation, were collected between July 2016 and March 2018 at two different medical institutions in Singapore. In addition, rarer cases of gallbladder volvulus and vascular complications such as cystic artery pseudoaneurysms and vessel erosions, were also followed up.

Results: The CT and MRI imaging features of these conditions were discussed, with key diagnostic imaging features emphasized.

Conclusion: Acute gallbladder pathologies are commonly encountered in day-to-day radiology practice. Knowledge of the rarer gallbladder pathologies and their key imaging features will help the radiologist, in particular, the on call radiologist in training, improve diagnostic accuracy and allow for timely management.

KEYWORDS:
Acute calculous cholecystitis, computed tomography, magnetic resonance imaging, complications

INTRODUCTION

Acute cholecystitis (AC) is a common problem encountered in surgical practice and it occurs due to obstruction of the cystic duct by calculi resulting in inflammation of the gallbladder. Gallstones affect 10-15% of the adult population in developed countries, with 1-4% of such patients developing symptoms each year.[1] In the United States, AC occurs in up to 1% of patients with known gallstones per annum.[2]

Radiological examinations are a cornerstone in the evaluation of patients who present with AC. Traditionally, ultrasound is the modality of choice in the investigation of patients who are suspected to have an acute gallbladder pathology. However, the images obtained with ultrasound is operator and patient dependent. Furthermore, ultrasound may not detect complications such as perforation or gangrene.

Contrast-enhanced computed tomography (CECT) scans of the abdomen and pelvis are increasingly being performed in patients with suspected acute cholecystitis, due to the short scan time and increasing availability in most departments. CECT is also able to detect complications of AC such as perforation or gangrene. Magnetic Resonance Imaging (MRI) is not commonly used as a primary imaging modality to image patients with acute gallbladder pathologies. However, magnetic resonance cholangiopancreaticogaphy (MRCP) is useful in the detection of choledocholiathiasis and variant biliary anatomy which would be helpful in preoperative planning. Complications such as gangrene and abscess formation may also be more discernible on MRI.

The imaging features of AC are well recognized and extensively described in literature. However, the radiological features of the complications related to acute cholecystitis such as pseudoaneurysm formation are less well described on CECT and MRI. These complications may be difficult to diagnose prospectively if the radiologist is unaware of their appearances. Accurate diagnosis is imperative for timely and appropriate management.

A. Acute calculous cholecystitis

An impacted gallstone in the gallbladder neck or cystic duct accounts for approximately 90-95% of cases of acute cholecystitis. The proposed pathophysiology of cholecystitis is as follows: the impacted stone leads to biliary stasis followed by inflammation of the gallbladder secondary to bacterial overgrowth. Distension of the gallbladder also causes reduced perfusion from the cystic artery and resultant ischaemia. The gallstones may also contribute to the inflammatory response by stimulating prostaglandins.[3]

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Patients characteristically present with a history of severe, sudden onset of constant or colicky pain in the right hypochondrium with nausea with vomiting. Clinical examination may reveal a positive Murphy's sign and peritonitis. Patients are usually more than 25 years old with a female preponderance (1:3 ratio).[4]

On CECT imaging, radiodense cholecystolithiasis is found 15-20% of the time,[3] associated with mural hyperenhancement, pericholecystic fluid and inflammatory fat stranding. The gallbladder is often distended and demonstrates mural thickening of more than 3mm.[5] Occasionally, enhancement of the adjacent liver parenchyma due to reactive hyperaemia may be present.

On MR imaging, gallstones were recognized as dependent signal voids on T2w imaging. Cholesterol stones may appear as T1 hyperintense foci. Similar to CECT, the gallbladder is often distended. The walls of the gallbladder were thickened and hyperintense on T2. In such instances, MRCP may be useful to show retained calculi within the common bile duct which may predispose the patient to cholangitis and biliary obstruction.

Cholecystectomy is the definitive management, with percutaneous cholecystostomy as an alternative should the patient be a poor surgical candidate. A small percentage of patients may develop potentially lifethreatening complications, which will be further elaborated later in this paper. With prompt surgical intervention, the prognosis for uncomplicated cases of acute calculous cholecystitis is excellent, with a low mortality rate of 3-4%.[6] Majority of the patients with AC do not experience complications. However, a small percentage of patients who may develop potentially life threatening complications such as gallbladder empyema, gangrenous cholecystitis and gallbladder perforation. Recognition of these entities is important as prompt surgical intervention would be required.

B. Complications of acute calculous cholecystitis

1. Gallbladder Empyema

Gallbladder empyema is a severe form of AC resulting from stagnant bile in the gallbladder superinfected with microorganisms that lead to pus formation in an acutely inflamed gallbladder. It is estimated to occur in 5 % to 15% of cases diagnosed to have acute cholecystitis.[7] Pus in the gallbladder increases the intraluminal pressure and may lead to wall necrosis and perforation if there is no prompt intervention. The CECT findings of gallbladder empyema are indistinguishable from that of acute cholecystitis.

On MR imaging, pus or purulent bile demonstrates restricted diffusion due to the large quantities of inflammatory cells within the purulent fluid. The diagnosis of empyema can thus be suggested by presence of fluid demonstrating diffusion restriction, when accompanied by an inflamed wall of a distended gallbladder.

Identification of these findings by the radiologist would enable prompt treatment, as this is a surgical emergency with a high propensity for septic shock.[8-10]

2. Gangrenous cholecystitis

Gangrenous cholecystitis is a relatively common complication of AC, affecting approximately 39% of patients with acute calculous cholecystitis.[11] Elderly patients, and patients with a history of diabetes mellitus and white blood cell count > 15,000 cells/mL are at an increased risk of having gangrenous changes at presentation. It occurs due to ischaemia and necrosis of the gallbladder wall. This can be further complicated by perforation, sepsis, abscess formation. Thus, it carries a high rate of mortality and morbidity.[11] CT is known to have high specificity but low sensitivity for acute gangrenous cholecystitis.

The commonest CECT finding of gangrenous cholecystitis would be lack of gallbladder wall enhancement. The gallbladder wall is often thin and irregular in appearance.

On MR imaging, there is usually asymmetric irregular wall thickening with inhomogeneous or absence of wall enhancement on post-contrast sequences. Mural ulcers may be present appearing as concave hyperintense areas on T2 fat saturated sequences. The presence of intramural membranes is the most specific sign of gangrenous cholecystitis and this is demonstrated very well on MRI, where the intraluminal membranes appear as linear T2 hypointense filling defects within the lumen of the gallbladder.

Patients are often treated with an emergent cholecystectomy under the cover of broad spectrum intravenous antibiotics. Percutaneous cholecystostomy may be performed in patients who are not eligible for emergent cholecystectomy to relieve the infection and act as a bridge to definitive surgery.

3. Emphysematous cholecystitis

Emphysematous cholecystitis is a potentially life threatening anaerobic infection of the gallbladder. It is defined as presence of intramural or intraluminal gas in the setting of AC without demonstrable communication to the gastrointestinal tract.

It is more commonly seen in men and typically occurs in diabetic patients.[12] Clostridium welchii is the most frequently cultured organism.[13-14] Vascular compromise of the cystic artery is thought to be one of the contributing factors, leading to gallbladder ischaemia. This can result in increased incidence of complications such as gallbladder wall gangrene and perforation, causing mortality rates to rise as high as 15-25%.[12-15] There are also cases of emphysematous cholecystitis in cancer patients on sunitinib, a tyrosine kinase inhibitor.

Gas foci can be seen within the gallbladder wall or lumen or pericholecystic tissues in emphysematous cholecystitis on CECT imaging.

On MR imaging, intramural gas is seen as signal voids along the non dependent aspect of the gallbladder. This is in contrast with gallstones which will be seen at the dependent portions. This feature helps distinguish intramural gas from an intramural stone. Other MR imaging findings of emphysematous cholecystitis resemble those of gangrenous cholecystitis, including irregular or asymmetric thickening of the wall with heterogeneous high signal intensity on fat suppressed T2 weighted images and fat suppressed T1

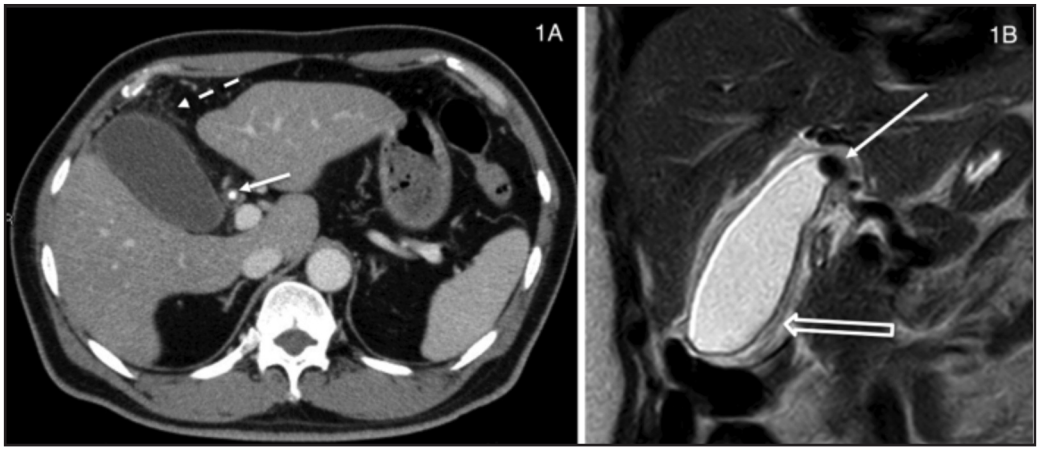

Fig. 1: (A) is a CT scan of of a middle aged gentleman who presented with 2 days history of right sided abdominal pain and fever. Axial CECT showed a distended gallbladder secondary to an impacted gallstone at the cystic duct (arrow), associated with pericholecystic fat stranding (dashed arrow) and mild gallbladder wall thickening. (B) is an MRI of a middle aged Chinese lady who presented with right upper quadrant pain. Coronal T2 HASTE sequence showed a calculus (arrow) in the neck of the gallbladder associated with mural thickening (open arrow), typical for acute calculous cholecystitis.

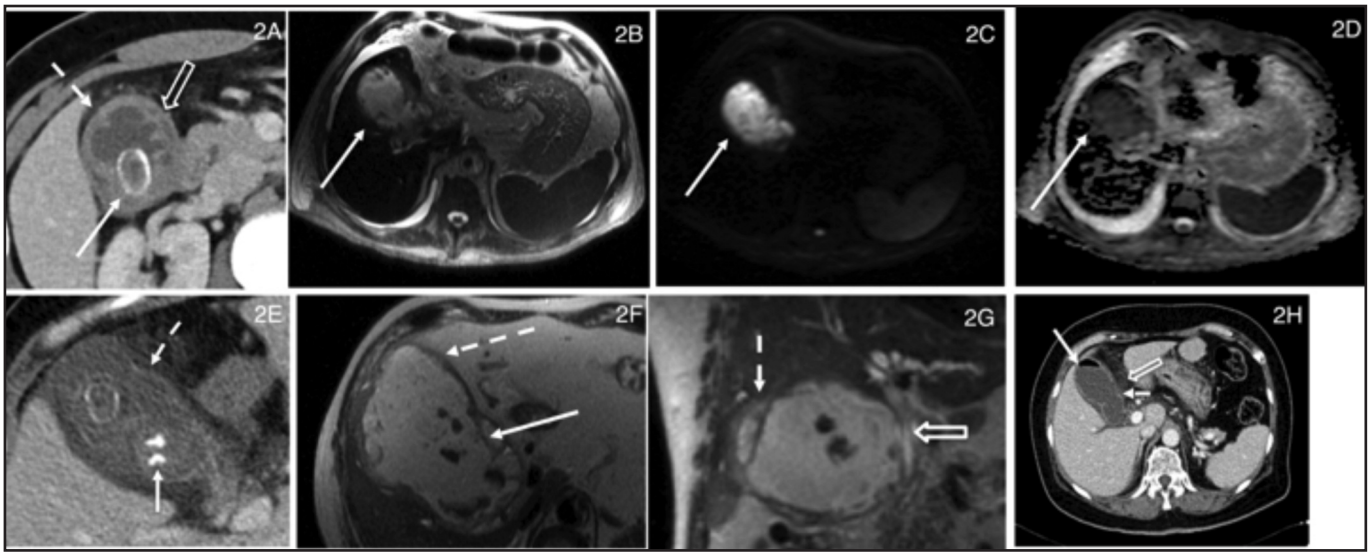

Fig. 2: (A) is a CT scan of a middle aged gentleman who presented with right hypochondrial pain. Axial CECT showed a calculus (arrow) lodged in the neck of the gallbladder. The gallbladder wall was thickened (dashed arrow) and also showed irregular mural outpouchings (open arrow) which suggests mural ulceration. During surgery, the lumen of the gallbladder was filled with pus. On CECT, AC is indistinguishable from gallbladder empyema. (B-D) are MRI images demonstrating the presence of T2w hypointense material layering within the dependent aspect of the gallbladder, representing calculi. In the non dependent aspect of the gallbladder, there is T2w hyperintense material (arrow, Fig 2B) demonstrating restricted diffusion (arrow, Fig 2C, 2D). During surgery, the lumen of the gallbladder was filled with pus, corresponding to the T2w hyperintense material showing restricted diffusion. On CECT, acute cholecystitis is indistinguishable from gallbladder empyema. (E) is a CT scan image of a middle aged lady presented with abdominal pain and septic shock. Axial CECT showed that the gallbladder contained multiple calculi (arrow). The wall of the gallbladder was irregular, thin and poorly enhancing (dashed arrow), in keeping with gangrenous change. Intraoperatively, the gallbladder showed patchy areas of gangrene. (F-G) are images of a middle aged gentleman was in a subacute inpatient ward for rehabilitation after a motor vehicle accident. He developed fever and abdominal pain. Axial T2 HASTE sequence showed the gallbladder to be markedly distended. Multiple linear striations are seen along the wall of the gallbladder which are suggestive of intraluminal membranes (arrow). Note that the gallbladder wall is also thinned (dashed arrow). The contour of the gallbladder is distorted with an outpouching seen posteriorly, worrisome for focal perforation (open arrow). (H) is a CT scan image of a middle aged gentleman who presented with acute onset of epigastric pain radiating to the back. Axial CECT showed intraluminal gas with gas fluid level (arrow). The gallbladder wall is thickened with areas of ulceration (dashed arrow) with surrounding fat stranding (open arrow). No abnormal communication with the gastrointestinal tract is seen on the rest of the images. There was no history of prior biliary surgery or instrumentation. Findings are in keeping with emphysematous cholecystitis, which was confirmed intra-operatively.

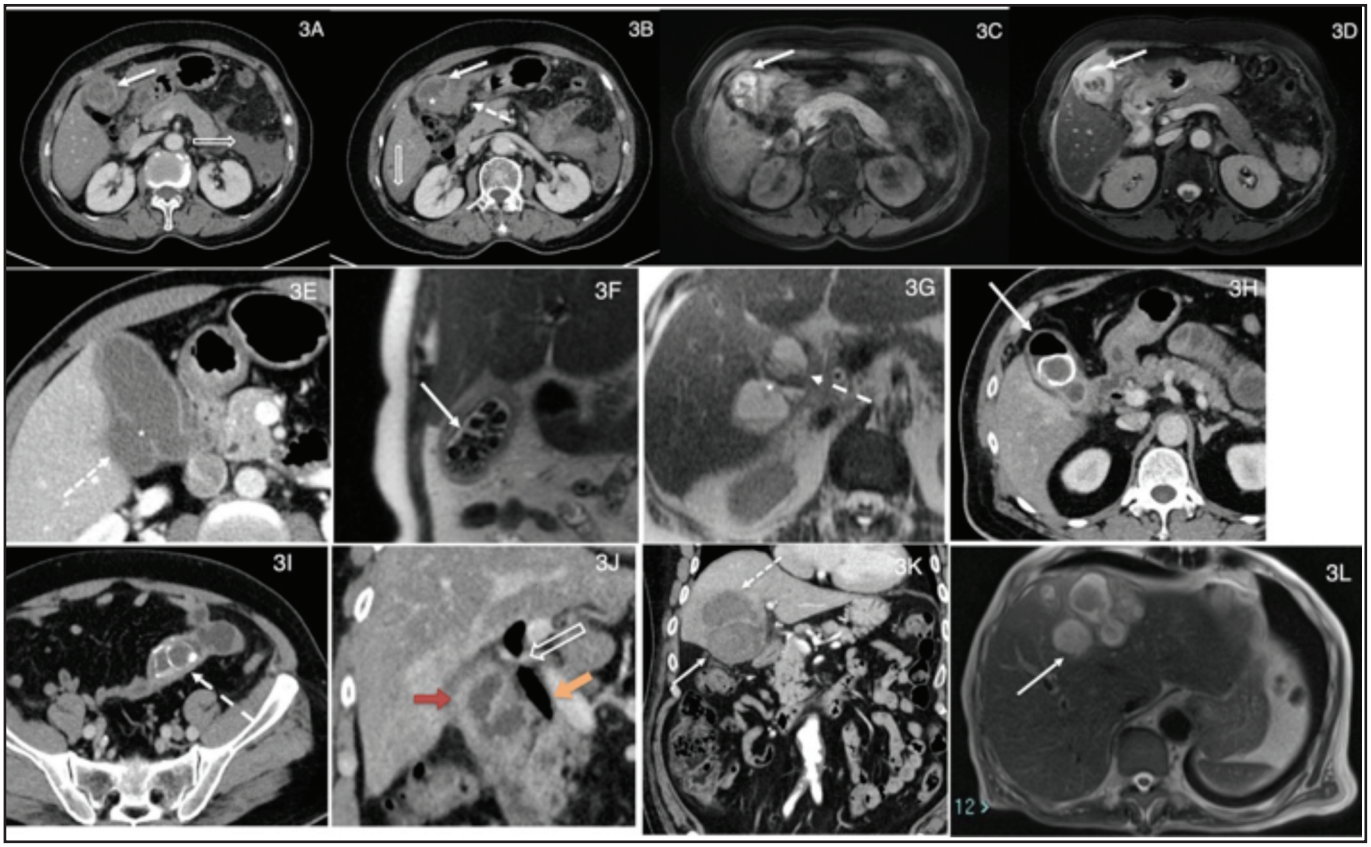

Fig. 3: (A-D) are abdominal CT scan sections of an elderly lady who was on antiplatelets for ischemic heart disease and presented with acute right abdominal pain and vomiting. (A-B) are axial CECT scans that showed heterogenously dense content within the gallbladder with thickened gallbladder wall (arrow), worrisome for intraluminal haemorrhage. Within the medial aspect of the wall, there was focal mural defect (asterisk) with an adjacent haematoma (dashed arrow). A small amount of hyperdense ascites was noted, which appeared as suspicious for haemoperitoneum (open arrow). (C-D) show the subsequent T1w MRI and T2w MRI images, respectively that demonstrated heterogeneous T1w hyperintense and T2w hypointense material within the gallbladder (arrow). Haemorrhagic cholecystitis with focal perforation and haemoperitoeum was confirmed intra-operatively. (E) is that of a patient who presented with right upper quadrant pain for 5 days. Axial CECT showed a focal outpouching at the lateral wall of the gallbladder, in keeping with a focal region of perforation (dashed arrow). A definite mural defect (asterisk) can also be seen. (F-G) are MRI images of an elderly lady who presented with fever and right upper quadrant pain. F is a coronal T2 HASTE image that showed multiple signal voids within the gallbladder in keeping with calculi. (G) is an axial T2 HASTE image that shows a focal mural defect (asterisk) and pericholecystic collection (dashed arrow). Findings are in keeping with perforation. (H-J) are CT scan images of a patient who presented with symptoms of small bowel obstruction. H is an axial CECT scan that showed the presence of gas and calculi within the gallbladder (arrow). (I) is an axial CT scan that shows a calculus that was detected in the ileum (dashed arrow) which was causing intestinal obstruction. (J) indicates that a closer evaluation of the gallbladder on coronal CECT showed a small cholecystoduodenal fistula (open arrow) between the gallbladder (red arrow) and the air filled duodenum (yellow arrow). These findings in (H-J) are in keeping with gallbladder perforation complicated by gallstone ileus. (K) shows coronal CECT showed a distended and inflamed gallbladder with heterogeneously dense intraluminal content and indistinct wall (arrow). An adjacent multi loculated intrahepatic abscess (dashed arrow) is noted with hyperdense material within it, likely secondary to debris or blood product. Pericholecystic inflammatory stranding is also detected. These findings suggest acute cholecystitis with hepatic abscess formation. (L) is that of subsequent MRI of the same patient with perforated cholecystitis showed a large, multi loculated T2 weighted hyperintense fluid collection (arrow), compatible with a hepatic abscess secondary to perforated acute cholecystitis.

weighted images, indicative of intramural hemorrhagic necrosis and formation of micro abscesses.[16]

Similar to gangrenous cholecystitis, cholecystectomy is the definitive treatment for emphysematous cholecystitis. Emphysematous cholecystitis generally carries with it a poorer prognosis, and the mortality rate in emphysematous cholecystitis as high as 25%, compared with 4% in acute cholecystitis.[6]

4. Hemorrhagic cholecystitis

Hemorrhagic cholecystitis refers to haemorrhage into the gallbladder lumen as a result of mucosal ulceration and necrosis. A few isolated cases of haemorrhagic cholecystitis occurring in patients with haemophilia or on anticoagulation have been described.[17-19] However, bleeding due to cholecystitis is extremely rare and is postulated that the inflammation associated with cholecystitis may cause vascular thrombosis.[20-22]

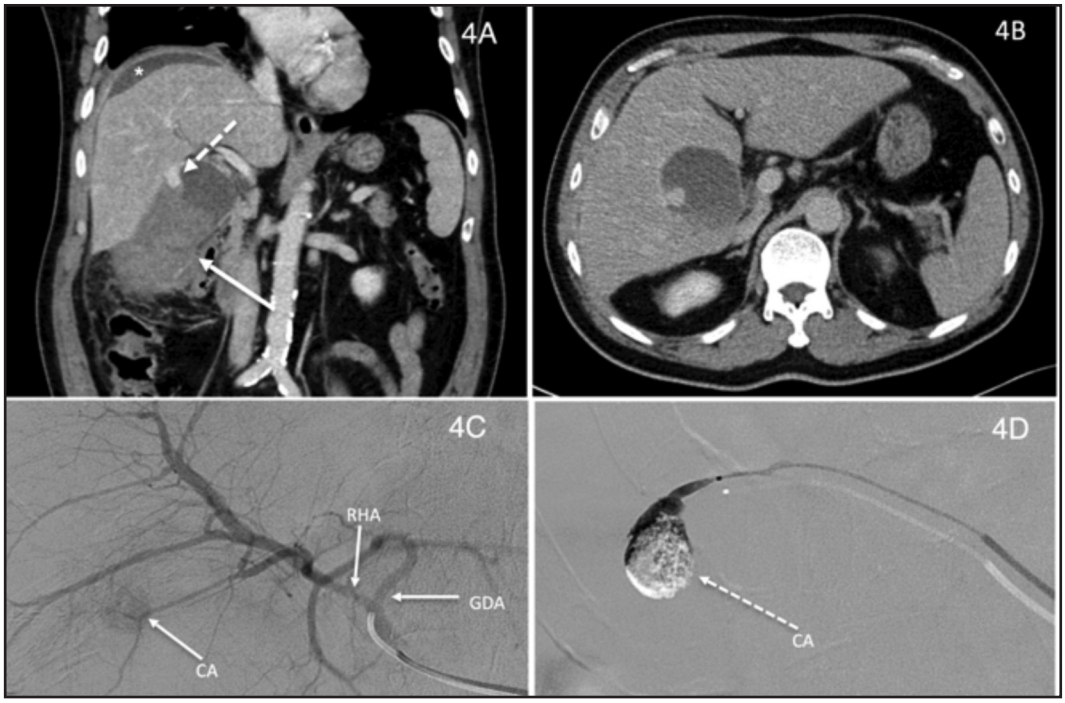

Fig. 4: (A) 60 year old male patient presented with three days duration of right hypochondrial pain. (A-B) are coronal and axial CECT, respectively that showed a distended gallbladder with layering hyperdensity (arrow) due to the presence of blood / sludge. There was a focal nodular hyperdensity at the lateral gallbladder wall in a non dependent aspect which was similar in configuration on the venous and delayed phases, suspicious for a pseudoaneurysm (dashed arrow). There was also a right subdiaphragmatic collection of low attenuation, possibly representing ascites (asterisk). The patient subsequently underwent angioembolisation. (C) is a selective angiogram of the hepatic artery that showed contrast blush from the cystic artery. (D) shows the super selective angiogram of the cystic artery that revealed a widenecked pseudoaneurysm (dashed arrow), which was embolised using glue. *RHA = right hepatic artery, GDA = gastroduodenal artery, CA = cystic artery

The presenting features of hemorrhagic cholecystitis are similar to AC, with right hypochondrial pain being a dominant feature. Where blood is passed through the biliary tree into the gut, the patient also may have additional symptoms of haematamesis or melena. Furthermore, blood clots may form, giving rise to mechanical obstruction of the bile ducts, resulting in painful jaundice, and mimicking the passage of gallstones. This oozing of blood via the ampulla of Vater, known as hemobilia, can be visualised on endoscopy.[23] On CECT imaging, debris and blood products are seen as high density material layering within the gallbladder lumen. The presence of gallstones may be obscured by the intra luminal blood. Free fluid seen elsewhere in the abdomen and pelvis may be simple or high in density, depending on whether hemoperitoneum is present due to perforation. As in majority of patients with AC, there is often free pericholecystic fluid and gallbladder wall thickening.

On MR imaging, blood in the wall and lumen of the gallbladder is T1w hyperintense due to the presence of methemoglobin. A fluidfluid level may be seen in the lumen of the gallbladder and extrahepatic bile ducts. Haemoperitoneum or free fluid can be seen in the peritoneal cavity if the gallbladder is perforated.

Angioembolisation of the bleeding vessel may be undertaken to stop the bleed and to stabilize the patient.[23] Subsequently, an elective cholecystectomy can be performed when the patient had recovered from the acute episode.

5. Perforated cholecystitis

Gallbladder perforation is a potential complication of AC, with a reported mortality rate ranging from 12-42%.[24] Increased intraluminal pressure within the gallbladder lumen results in ischaemia of the gallbladder wall, rendering it susceptible to perforation. Perforation occurs in 2-11% of acute cholecystitis patients.[11]

The fundus is the commonest site of perforation, given that it is the most distal part of the gallbladder with the least blood supply.[25] Fundal perforations are less likely to be covered by the omentum.[25] As a result, bile drains into the peritoneal space, resulting in free biliary peritonitis and stones within the peritoneal cavity. Non fundal perforations are easily sealed by the omentum or the surrounding intestines. Hence, the inflammatory process is confined to the right upper quadrant with formation of a pericholecystic abscess.[25]

CECT may show visible defect in the wall of the gallbladder. More commonly, a focal outpouching is seen which suggests the presence of a defect. Patients often have bile leakage as a result of perforation and a pericholecystitic fluid collection with adjacent inflammatory fat stranding may result.

On MR imaging, there was disruption of the gallbladder wall, which shows up as a mural defect. Often, a pericholecystic abscess is seen adjacent to the site of perforation. When a pericholecystic abscess is present, the gallbladder lumen communicates with the abscess through the disrupted gallbladder wall.

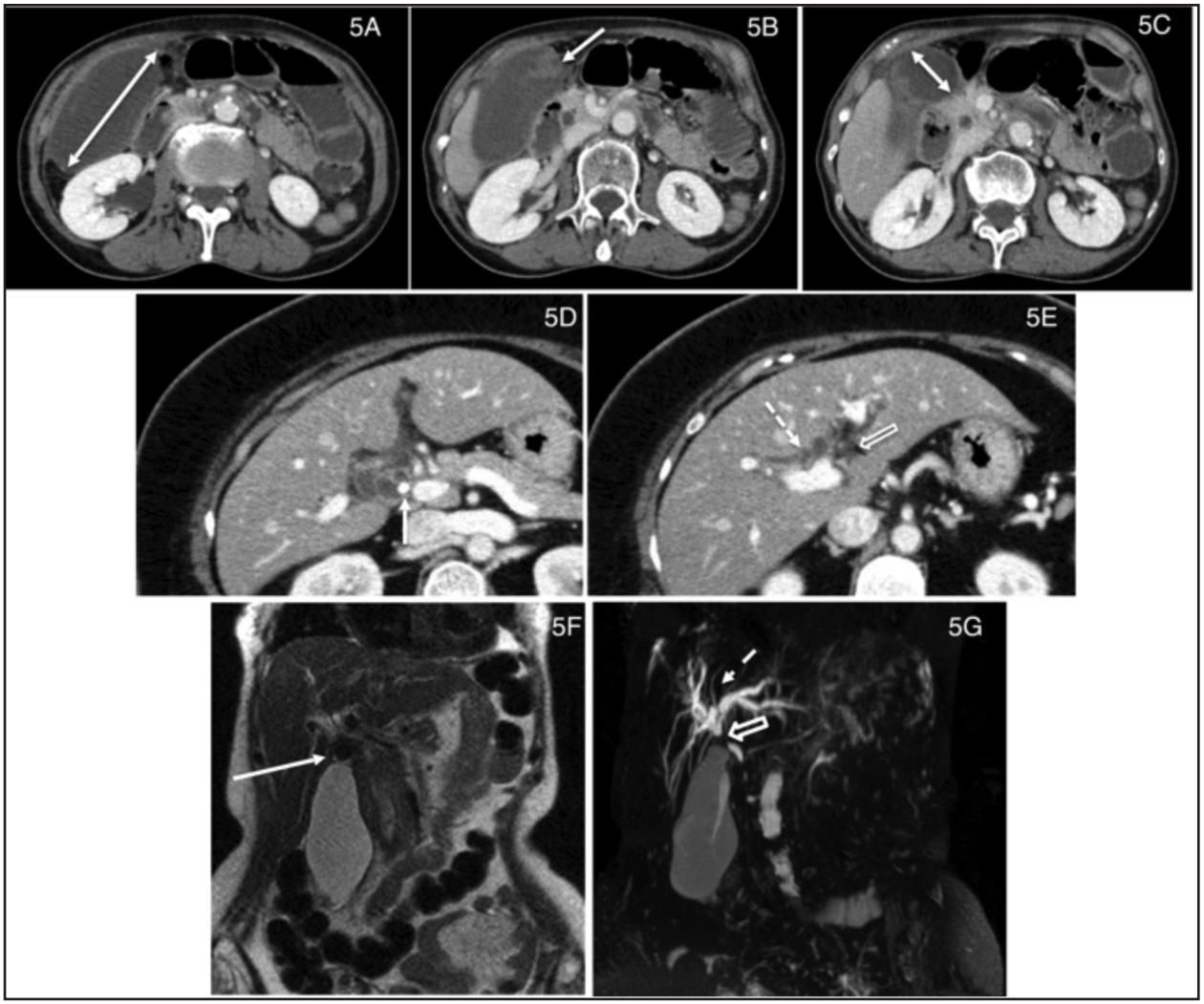

Fig. 5: (A-C) are axial CECT images of an elderly female who presented with right upper quadrant pain and vomiting. The gallbladder was markedly distended with a change in axis at the mid body (double headed arrow in Fig 5A and 5C). Enhancing soft tissue is noted at the point of axis change (Fig 5B, arrow). There was gallbladder torsion noted intra-operatively. (D-E) are those of an elderly lady presented with epigastric pain and vomiting. Axial CECT (Fig. 5D) showed an impacted gallstone within the cystic duct (arrow) compressing on the common hepatic duct (open arrow). (E) shows that the common hepatic duct (open arrow) and intrahepatic biliary tree (dashed arrow) were dilated (Fig. 5E). Findings were consistent with Mirizzi's syndrome.
Figure 5(F-G) are those of a patient who presented with right upper quadrant pain and jaundice. Coronal T2 HASTE image showed a calculus is lodged in Hartmann's pouch of the gallbladder (arrow in Fig. 5F). As a consequence of the mass effect, this had caused compression on the common hepatic duct (open arrow), with resultant dilation of the intra hepatic biliary ducts (dashed arrow) as demonstrated on the 3D MRCP images (Fig. 5G), accounting for the patient's jaundice.

The inflamed gallbladder can also perforate into the adjacent liver parenchyma, resulting in the formation of an intrahepatic abscess.

The treatment for perforated cholecystitis is that of an emergent cholecystectomy with abdominal lavage to treat peritonitis.[25,26] Drainage of the abscess if present, should also be performed in the same setting.

6. Cystic artery pseudoaneurysms

Formation of a cystic artery pseudoaneurysm is a very rare complication of AC. Typically patients present with a combination of upper abdominal pain, hematemesis and jaundice, known as Quinke's triad.[27,28] Approximately 50 cases have been reported worldwide.[29] More than half these cases were associated with cholecystitis or cholelithiasis, and more than 75% of these patients presented with haemobilia resulting from rupture of the aneurysm.

In AC inflammatory changes surrounding the gallbladder can result in fibrosis encasing the cystic artery and weakening its wall, giving rise to aneurysm formation.[29] These aneurysms are likely to rupture when the tamponade effect of the surrounding inflammatory fluid tension is lost. Hence, drainage of the pericholecystic fluid via cholecystostomy is usually delayed until embolisation of the pseudoaneurysm is completed.[30,31]

On CECT imaging, the pseudoaneurysm appears as a well circumscribed hyperattenuating lesion on the plain phase.

This lesion subsequently opacifies in the arterial phase and remains persistently hyperattenuating on delayed images. High density material in the lumen of the gallbladder and biliary ducts (haemobilia) may be seen with pericholecystic inflammatory changes.

As cystic artery pseudoaneurysm is a rare entity, there is currently no consensus for its clinical management, although it may range from the minimally invasive selective angioembolisation for unstable patients, to definitive cholecystectomy.

C. Others

1. Gallbladder volvulus

Gallbladder volvulus or torsion is an uncommon cause of acute abdomen. It occurs along the axis of the cystic duct and artery, compromising vascular supply.[32] It occurs when there is enough mobility of the gallbladder to allow it to rotate around a fixed pedicle by at least 180 degrees. While it has been postulated that precipitating factors for gallbladder volvulus or torsion include intense peristalsis of the neighboring organs, kyphoscoliosis of the spine and tortuous atherosclerotic cystic artery, no real cause has been identified to date.[33,34]

The entity is commonly misdiagnosed as cholecystitis before surgery, although some imaging findings are present which may assist the radiologist in reaching the right diagnosis.

CECT often demonstrates a large, distended gallbladder with loss of normal enhancement and abrupt tapering of the cystic duct. The 'beak and twirl' sign immediately distal to the point of torsion as well as a change in the anatomical position of the gallbladder from vertical to horizontal can be demonstrated.[35,36] The gallbladder may also be seen outside of its anatomical fossa with surrounding inflammatory changes.

On MR imaging, there is high signal intensity in the wall on T1w imaging as a result of coagulation necrosis with intramural haemorrhage. This diagnosis can be confirmed with contrast enhanced images. The treatment for gallbladder volvulus is emergent cholecystectomy to prevent ischaemia, infarction and overwhelming sepsis.[32]

2. Mirizzi syndrome

Mirizzi syndrome is defined as common hepatic duct (CHD) obstruction caused by an extrinsic compression from an impacted stone in the Hartmann's pouch of the gallbladder or in the cystic duct. Obstruction may be due to direct mass effect or stricture formation in the CHD due to repeated inflammation. Predisposing factors include long cystic duct running parallel to the CHD or low insertion of cystic duct into common bile duct.

Patients typically present with jaundice, fever, and right upper quadrant pain. It has been postulated that patients with Mirizzi syndrome are predisposed to gallbladder malignancy due to recurrent inflammation and biliary stasis. Cholecystocholedochal fistula may develop due to chronic inflammation/pressure necrosis, with gallstones eroding from cystic duct into bile duct.

CECT and MR findings for Mirizzi's syndrome are similar, with the presence of a large, impacted gallstone in the gallbladder neck. Dilated intra and extrahepatic biliary ducts are seen, with the former more commonly noted. The gallbladder wall may be diffusely thickened and enhance with contrast.

The treatment for Mirizzi syndrome include endoscopic removal of the obstructing calculus and subsequent definitive cholecystectomy.

CONCLUSION

Acute gallbladder pathologies are commonly encountered in surgical practice. As CECT and MRI are increasingly performed and image quality on CECT improves, acute gallbladder pathologies are increasingly and more confidently diagnosed. There is a paucity of published literature on the complications of AC, which includes erosion of the adjacent vessels, cystic artery pseudoaneurysm formation and gallbladder volvulus. Knowledge of the rarer gallbladder pathologies and their key imaging features will help the radiologist and residents in training improve diagnostic accuracy. This would assist the surgeon in either planning the surgery or in guiding further management.

CONFLICT OF INTEREST

None to declare.

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