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Case Report

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CASE STUDY OF EMPHYSEMATOUS CHOLECYSTITIS

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ABSTRACT

Emphysema of the gallbladder is a rare complication of acute cholecystitis. It presents as an acute fulminating cholecystitis with toxaemia setting in rapidly. It is characterized by the presence of gas in the gallbladder wall and pericholecystic tissue, early diagnosis is essential as its squeal are gangrene and perforation It is common amongst diabetics and elderly males and has a high mortality of 15-25% [1] Early cholecystectomy is indicated. We present one such case of emphysematous cholecystitis managed successfully by early cholecystectomy without ERCP.

KEYWORDS: cholecystitis, toxaemia, pericholecystic.

CASE REPORT

A 51-year-old man, known diabetic with IHD having asymptomatic cholelithiasis since 5 yrs. Patient was already on oral anti-coagulant therapy for IHD and was hosptalised elsewhere for 5 days with a diagnosis of acute cholecystitis and transferred when he started exhibiting signs of septicemia.

On admission clinically he had all signs of septicemia. Diabetic status HbA1C-9.10 His LFT's showed S.Bilirubin4.42, D/I 3.40/0.92, SGPT-78, SGOT-93, AlkPo4-93, Alb/Gl-3.43/2.53 the white blood count was 9600/cmm, Platelets-141000, RFT showed Bl.Urea-65, S.Creat-1.76. X-Ray showed bilateral pleural effusion. USG report was emphysematous cholecystitis with possibility of perforation, and formation of sub-diaphragmatic abscess.

CT imaging Figure 1 & 2

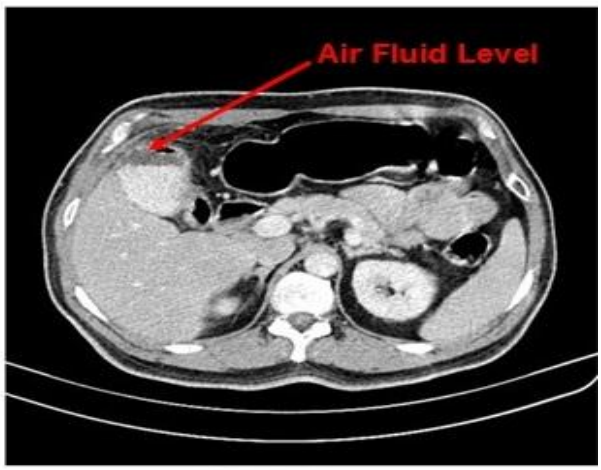


Figure 1

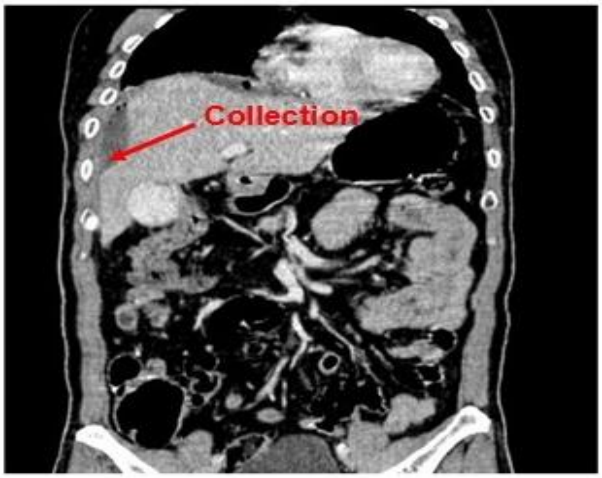


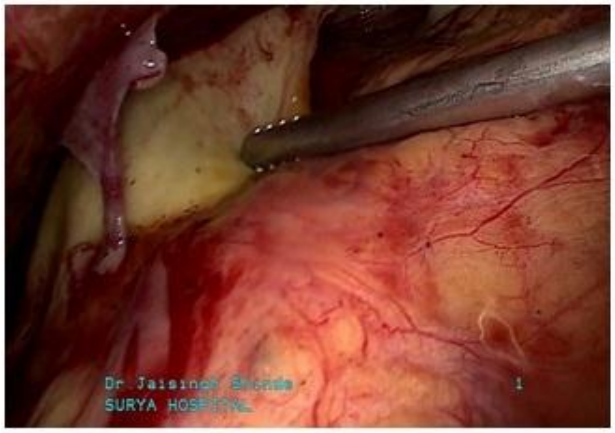
Figure 2

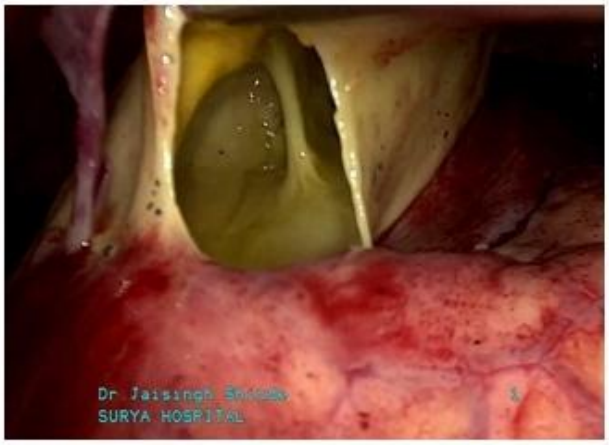
Sagittal and coronal reformatted image of Multislice CT showing hyperdense material within the gall bladder lumen suggestive of multiple tiny calculi / hyperdense sludge. Note anterior focal thinning of walls of GB with air pockets within raising suspicion for emphysematous cholecystitis. Focal subdiaphragmatic collection is seen, again containing air pockets. The CBD diameter is around 4 mm. intra luminal pathology cannot be well visualized.

Since patient was jaundiced, in acute renal failure and on Tab. Clopitabs , he was conserved with Inj Piperalcillin, witholding Tab.Clopitab and adequate IV fluids He settlied down haemodynamically in 2 days and was taken for laparascopic cholecystectomy, after 5days. ERCP was contemplated but not done after reviewing the subsequent laboratory profile and discussion with a

Gastroenterologist. Patient was explained of an ERCP if the necessity arose post-operatively.

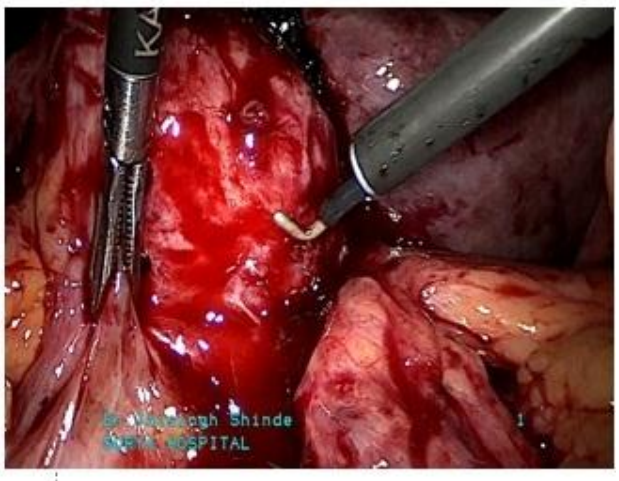
Operative Images

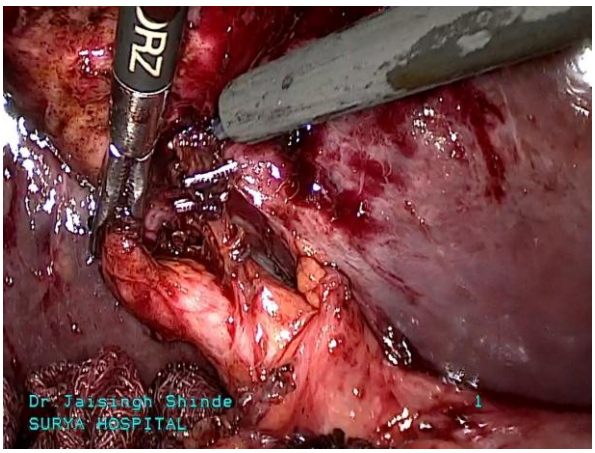




Opearative findings were- there was intense fibrinous adhesions between the anterior abdominal wall in the region of the right hypochondrium. There was a huge multiloculated sub-diaphragmatic collection .Careful dissection of the lump in which the colon and duodenum needed to be separated, the gallbladder wall was reached with difficulty, perforation was noticed in the fundic area.

Operative Images





After careful blunt dissection with a suction canula and gauze the cystic duct and cystic artery were dissected and the cystohepatic triangle exposed. The cystic duct was milked, partially opened and its proximal portion was irrigated with a suction irrigation canula to ensure it being stone free .Total cholecystectomy was performed the gallbladder wall near the fundus was perforated

The culture of the bile showed no growth. HP report showed emphysematous cholecystitis with multiple small stones. The cystic artery showed evidence of thrombosis. The postoperative course was uneventful. His lab reports were practically normal and ERCP was not recquired. The patient was discharged from hospital on the fifth postoperative day. Follow-up 6mths is uneventful and the jaundice is totally subsided and the RFT's are normal.

DISCUSSION

Emphysematous cholecystitis also known as gas phlegm in the gallbladder was first reported by Stolz in 1901. It is characterized by gas within the lumen with or without concomitant pericholecystic infiltration of gas. The most common cause of acute emphysematous cholecystitis is obstruction to the neck of the gallbladder. The pathology can either be a stone (95%), kink, enlarged lymph node, tumour or anomalous vessel which obstructs the cystic vessels. The cystic artery being an end artery, the sole arterial supply of the gallbladder [2] occlusion or stenosis

of the artery results in compromised viability of the gallbladder Ischemia facilitates the proliferation of anaerobic gas-forming organisms and bacterial translocation occurs in the devitalized tissue. This theory is substantiated by the fact that emphysematous cholecystitis are seen after cystic artery embolisation or torsion of the gallbladder (Coldwell DM, Hottenstein DW, Ricci JA, Wengert PA) Emphysematous cholecystitis as a complication of hepatic arterial embolization[3] Impaction of stones in the cystic duct leads to localized edema of the wall, which contributes to the vascular compromise of the gallbladder. Failure to find micro-organisms in 50% of cases in the non-emphysematous cases operated early suggest that the initial infection may not be of bacterial origin. The chemical infection gets augmented by bacterial invasion condition is more common when comorbidities like diabetes, elderly and hypertension are present.

Acute non-calculous cholecystitis is encountered in infective disorders like typhoid,and pneumonia , where infection is via blood or lymphatics. Incidentally emphysematous cholecystitis is 3 times more when patient has a pre-existing calculi in the gallbladder. Emphysematous cholecystitis has also been reported as an adverse event caused by drugs like sunitinib,which are administered for the treatment of gastrointestinal stromal tumor (GIST), probably due to the thromboembolic side effect of this class of drugs (vascular endothelial growth factor [VEGF] receptor inhibitors).[4] The pathogenesis of acute cholangitis which accompanies emphysematous cholecystitis is biliary infection. Biliary obstruction raises the intraductal pressure in the bile duct to levels high enough to cause cholangiovenous or cholangiolymphatic reflux.Prior to the 1970s the mortality rate of patients with acute cholangitis was reported to be over 50%, [5] but advances in intensive care, new antibiotics, and biliary drainage dramatically reduced the mortality rate to less than 7% by the 1980s.[4 & 8] even now the severe form of acute cholangitis remains a fatal disease unless appropriate timely management is instituted. Predictive values for CBD stones are that the cholestatic liver biochemical tests generally progressively increase with the duration and severity of biliary obstruction which were absent in our case. Transabdominal USG has a relatively poor sensitivity (22%-55%) for detecting CBD stones, the predictive value for choledocholithiasis, with multiple small (5 mm) stones posing a 4-fold higher risk of migration into the duct as opposed to larger and/or solitary stones.[6] The prevalence of choledocholithiasis in patients with symptomatic cholelithiasis is 5-10%. USG has a 95% to 96% predictive value.12, 28 to R/O choledocholithiasis, CT is similar to ultrasound, with a specificity of 97% [7] The diagnostic accuracy of MRCP range between 90– 96%.If proper antibiotics are administered immediately, the infection clears up in 75% of patients. The organism and appropriate antibiotics depend upon the fact whether the infection was hospital or community acquired. Our patient was never recently hospitalized, so the infection was community acquired and response to antibiotics was better. If cholangitis does not improve, the infection may spread and become life-threatening. Either surgery or ERCP is required to open and drain the ducts. In our case the patient responded well to conservative therapy ERCP was contemplated in the waiting period but was not done by weighing the risk of introducing infection in a responding patient with no strong evidence of choledocholithiasis. Patient was taken for laparascopic cholecystectomy after explaining that he may have to undergo ERCP if warranted. On the operation table the cystic duct was irrigated to clear the small stones. Cystic duct was milked. Per-operative cholangiography was not performed. Patient showed a good post-operative recovery and is asymptomatic now for 6 months.

CONCLUSION

Acute emphysematous cholecystitis is a fast spreading entity common in diabetics. It has a fulminant course and recquires prompt diagnosis and early surgery. When all facilities are available, one has to judiciously utilize them for optimum results, as they could prove counter-productive. In our case we could manage the case without ERCP and patient responded well.

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