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Case Report

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PSEUDOCYST OF PANCREAS PRESENTING AS BILATERAL PLEURAL EFFUSION A RARE CASE ENTITY

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ABSTRACT

Pleural effusion as a consequence of acute pancreatitis is transient, usually left-sided; rarely right-sided and haemorrhagic causing difficulty in establishing the diagnosis, especially if the chest symptoms are disproportionately more than the abdominal symptoms. We present a case of an alcoholic male patient who presented with a history of pain in abdomen, no respiratory symptoms. His Ultrasonography of Abdomen was suggestive of multiple pseudocyst of pancreas with bilateral pleural effusion.

KEYWORDS: Pancreatitis, Pleural Effusion, Pseudocyst.

INTRODUCTION

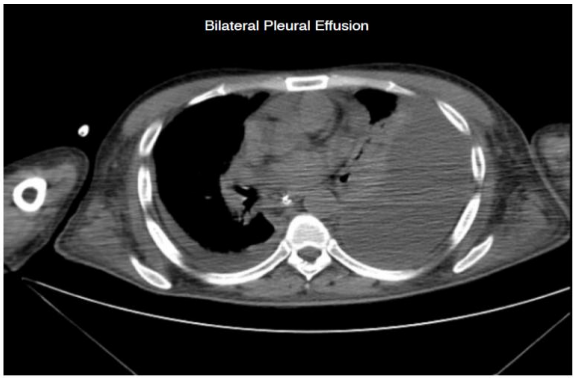
Pancreatitis generally causes transient left-sided pleural effusion (68%), 22% of the cases are bilateral and 10% are right-sided only. In only 33% of all patients with Pancreatitis, respiratory complications are clinically or radiographically detectable.[1] The underlying pancreatic disease especially if asymptomatic, the diagnosis can be missed. Pancreatic pseudocyst is a type of cystic tumor that most commonly occurs as a complication of acute or chronic pancreatitis, pancreatic injuries and pancreatectomy.[2] The clinicopathologic features of this cyst include a cystic wall without an epithelial cell lining and black cystic fluid with a high amylase concentration. Pancreatic pseudocyst can result in pleural effusion through different mechanisms, such as cystic fluid leakage leading to reactive effusion by stimulating the septum transversum and cystic fluid directly entering the pleural cavity to cause pleural effusion.[1] We report a rare case of a male alcoholic patient with pseudocyst of pancreas that developed bilateral pleural effusion with high amylase and lipase concentrations and growth of E. *coli*.

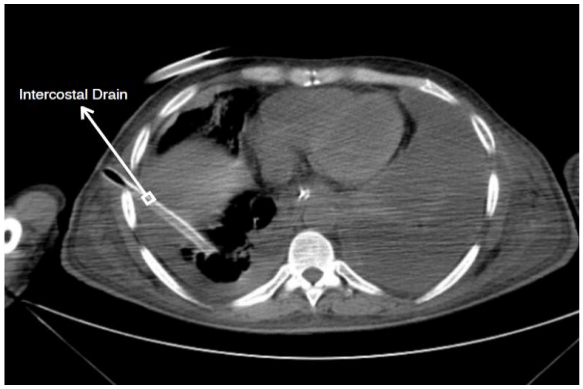
CASE REPORT

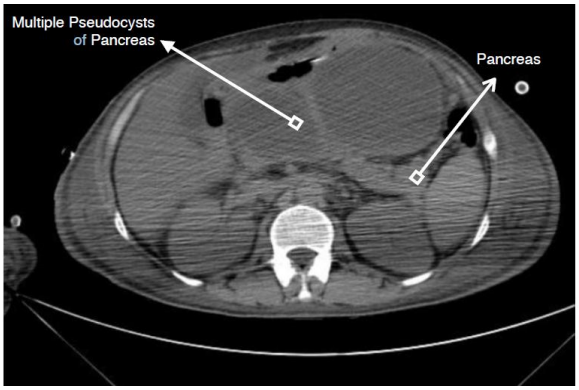
A 42 year-old male chronic alcoholic presented with history of abdominal pain radiating to the back along with nausea and vomiting since 5 days. He had no history of Diabetes Mellitus, Hypertension or Tuberculosis. There was no history of injury and any abdominal discomfort or pain. He was a non-smoker and chronic alcoholic probably consumed alcohol daily for the preceding eight years, but he stopped taking this since last 1month. On admission, the patient was febrile, pallor present, no lymphadenopathy, clubbing, cyanosis, icterus, pulse was 96/min, regular, and blood pressure was 110/60 mm Hg. Signs of chronic liver disease, such as palmar erythema, spider nevi, and parotid gland enlargement, were not detected. On examination, the abdomen was tender in the epigastric area. Liver span was normal. The examination of respiratory system revealed dull percussion note, absent vocal fremitus, and reduced breath sounds bilaterally. Examination other systems were normal.

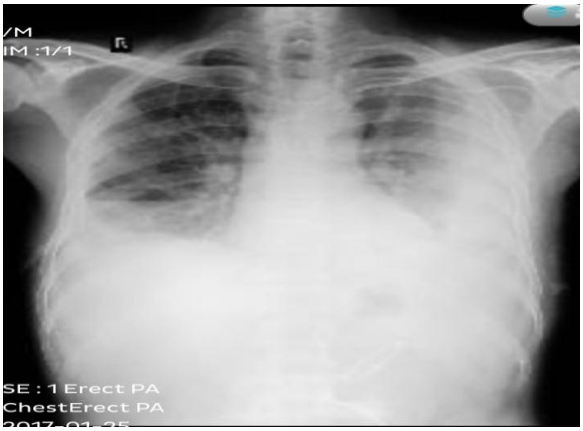
His blood parameters on admission showed haemoglobin of 9.8 g/dl, mean corpuscular volume 72 fl, total leukocyte count of 9600/cu mm and platelet count 2.27 lakhs/cu mm. His biochemical tests showed blood urea 32 mg/dl, serum creatinine 0.68 mg/dl, serum sodium 125 mmol/L, serum potassium 3.8 mmol/L; serum amylase 391 IU/L, serum lipase 231 IU/L, and serum lactate dehydrogenase (LDH) 580 IU/L. His liver function test revealed serum bilirubin 0.89 mg/dl, alanine transaminase 48 IU/L, aspartate transaminase 90 IU/L, alkaline phosphatase 172 IU/L, total proteins 5.9 g/dl, serum albumin 3.5 g/dl and INR 1.31. His urine routine examination was normal, and urine culture was sterile. Serum viral markers for HIV, HBV and HCV were non reactive. Ultrasound Abdomen was suggestive of Pseudocyst of Pancreas and Bilateral Pleural Effusion;

with borderline hepatomegaly and normal liver echo-texture. Chest X-Ray revealed massive right sided pleural effusion and moderate left sided pleural effusion. CT Abdomen revealed Pancreatitis with multiple pseudocysts; Moderate bilateral Pleural effusion with collapse of lower lobe of left lung. Pleural fluid cytology showed serous effusion with Pleural fluid amylase 2170 IU/L and lipase 290 IU/L; ADA was 24 IU/L. Pleural fluid culture and sensitivity revealed growth of E.c*oli* species sensitive to Ciprofloxacin, Tetracycline, Gentamycin, Cotrimoxazole and resistant to Amikacin and Ceftazidine. Pleural fluid cytology was negative for malignant cells. Treatment was done with drainage by an intercostal chest tube with fluid output monitoring, as well as conservative management of pancreatitis to decrease pancreatic exocrine secretion (nasogastric suction, total parental nutrition, somatostatin analogues), along with antibiotic therapy based on Pleural Fluid Culture and Sensitivity (Gentamycin in this case). The intercostal drain was removed once the fluid output was less than 50 ml. Patients was doing well on follow up.









DISCUSSION

Pleural effusion often occurs as a complication of pancreatic disorders such as acute pancreatitis, pancreatic abscess, pseudocyst, and chronic pancreatitis.[2] In older reports, only 3-7% of the patients of Acute Pancreatitis presented with or developed pleural effusion but it is nearing 50% in recent reports based on pleural fluid detection by CT.[3,4] they are usually left-sided effusion, reactive, often mild to moderate and characterised by normal amylase activity (below 100 U/L) and low protein concentration (<3 g/dl), often associated with acute pancreatitis and resolves during recovery.[5]

Presence of a Pancreatico-Pleural fistula (PPF) is also associated with pleural effusion in chronic alcoholics; this effusion is usually large, single-sided, recurrent, contains a high level of amylase above 1000 U/L and protein above 3 g/dl.[6,7] It rapidly accumulates and is refractory to drainage procedures. Bilateral pleural effusions account 15% of the cases while 20% were right-sided effusions.[8]

The pathogenic mechanism involved in the formation of the pleural effusion include direct contact of pancreatic enzymes with the diaphragm, haematogenous transfer of pancreatic enzymes into pleura, transfer of pancreatic secretions through trans-diaphragmatic lymphatics and formation of pancreatico-pleural fistula which results in direct communication of pancreatic pseudocyst with pleural cavity.[4,9] Rarely, there may spontaneous rupture of the pseudocyst into the pleural cavity causing massive pleural effusion.[10] PPFs have been noted in 2.3–4.5% of

patients presenting with pancreatic pseudocyst.[11] If the pancreatic duct disruption occurs posteriorly, an internal fistula may develop between the pancreatic duct and the pleural space, producing a pleural effusion (PPF) that is usually left-sided. If the pancreatic duct disruption occurs anteriorly, amylase- and lipase-rich peritoneal fluid accumulate (pancreatic ascites).[9] Sometimes pancreatic fluid tracks through the aortic or oesophageal hiatus into the mediastinum. Occasionally, secretions are contained within the mediastinum presenting as a mediastinal pseudocyst.[11] Once fluid enters the pleural space, the PPF is likely to result in a massive chronic pleural effusion especially in autoimmune condition.[12,13]

CONCLUSION

Pseudo pancreatic cyst should be taken into consideration when bilateral pleural effusion occurs, especially when it is recurrent. Treatment with drainage by a chest tube, with concomitant conservative treatment of the pancreatitis, is usually effective in massive pancreatic pleural effusions. Morbidity and mortality are reduced when a definite diagnosis is established and appropriate therapy rendered.

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