Clinical Brief

Massive Pancreatico-Pleural Effusion–An Often Unrecognised Entity

Deepa Gupta,[1] K.L. Chakraborty,[2] Sunil Gomber,[1] Anurag Krishna,[3] and G. Mehrotra,[4]

Deparments of [1]Paediatrics and [4]Radiology, University College of Medical Sciences and G.T.B. Hospital; [3]Paediatric Surgery,[3] Sir Ganga Ram Hospital, and [2]Radiological Imaging Department, Institute of Nuclear Medicine and Allied Sciences.

Abstract. Massive haemorrhagic pleural effusion secondary to pancreatitis in a five year old girl is described. The diagnosis was established on the basis of an exudative pleural effusion with an amylase level above 4,000 IU/l. Management of effusion was by intercostal tube drainage and antibiotics. **[Indian J Pediatr 2001; 68 (9) : 883-885]**

Key words *: Pancreas; Pleural effusion*

Pulmonary complications ranging from minimal atelectasis to respiratory failure are recognized but unusual complications of acute pancreatitis in children.[1] However, massive pleural effusion with the clinical evidence of acute pancreatitis is an uncommon and often unrecognosied entity. The present communication is regarding a case of haemorrhagic pleural effusion secondary to acute pancreatitis in a young child.

A five-year-old female child presented to the paediatric emergency with a 15 day history of respiratory distress, and mild fever. The respiratory distress had increased for 6 days prior to admission. There was no history of cyanosis, trauma to chest or abdomen, altered sensorium or chronic dirrhoea. History of contact with the tubercular patient was absent. Examination revealed a toxic, pale and malnourished child who was fully conscious and alert. The pulse rate was 138/min., respiratory rate 64/min. with marked intercostal and subcostal recessions and blood pressure was normal for the age (90/60 mm Hg). There was no evidence of lymphadenopathy. BCG scar was absent. Chest examination was suggestive of left (L) sided pleural effusion. Other systems were essentially normal.

Skiagram of the chest on admission showed complete opacification of left (L) half of chest with mediastinal shift to right and right sided minimal effusion. A diagnostic thoracocentesis was done on the left side of chest and 100 ml of uniformly stained fluid was aspirated.

On the basis of clinical examination and thoraco-centesis a possibility of haemorrhagic pleural effusion secondary to either tuberculosis, bleeding diathesis, chest trauma or intra thoracic neoplasm was kept.

Ultrasonography examination of chest and abdomen showed massive left pleural effusion, minimal right pleural effusion, with a thin walled cystic lesion in pancreas, 5 cm in diameter, containing 50-60cc. of thin fluid posterior to left lobe of liver. An inference of bilateral pleural effusion with pseudopancreatic cyst was made. Computerized tomography (CT) scan of the chest and abdomen revealed a left sided massive pleural effusion and a bulky, hypodense pancreas with ill defined fatty planes suggestive of pancreatitis (Fig. 1).

Pleural fluid, exudate, showed RBCs with no evidence of malignant cells. The biochemistry revealed a protein of 4gm/dl and sugar of 70mg/dL. No organisms were seen on gram staining and Ziehl Neelsen staining. Pleural fluid culture was sterile. Polymerase chain reaction (PCR) test of pleural fluid for tuberculosis was negative. The pleural fluid amylase level was 31, 160 IU/L with a concomitant serum amylase level of 714 IU/L. Peripheral blood smear examination showed mainly moderate normocytic normochromic RBCs, polymorphonuclear leucocytosis and normal platelets. Patient's bleeding time and clotting time were normal. Blood urea, serum electrolytes, blood

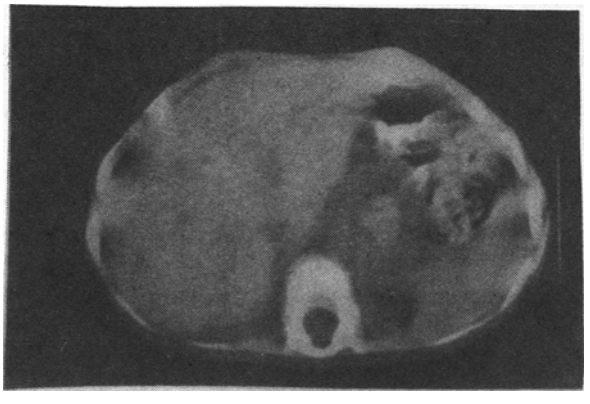

Fig. 1. CT abdomen shows hypodense, bulky pancreas with ill defined fatty planes suggestive of pancreatitis.

Reprint requests : Dr. Sunil Gomber, F-4 (A), Vijay Nagar, Delhi-110009. Fax : 2290495; E-mail : sunilgomber@hotmail.com

sugar, serum creatinine were within normal limits. Serum calcium was 7.6mg/dl. Blood culture was sterile after 48 hours of incubation. Bone marrow examination was essentially normal. Tuberculin test was negative.

Based on clinical, ultrasonography and computerized tomography scan findings along with increased pleural fluid and serum amylase level values, a diagnosis of haemorrhagic pleural effusion secondary to pancreatitis was established.

An intercostal tube was inserted on the left side of the chest and about 2 liters of haemorrhagic fluid was drained over 2 weeks and then the aspirate became purulent which grew klebsiella species on culture which was sensitive to amikacin and cefotaxime. Child was adequately treated with intravenous Amikacin and Cefotaxime for 6 weeks. The drainage tube was removed after 3 weeks after ensuring adequate lung expansion with no further re-accumulation. A subsequent USG done showed minimal left sided pleural effusion and a swollen pancreas containing a cystic lesion inside the body and tail of pancreas having around 10 ml of thin fluid. Repeat CT scan done after 4 weeks of the previous scan showed pseudocyst of the pancreas (Fig. 2.). The patient was finally discharged after 52 days of hospital stay.

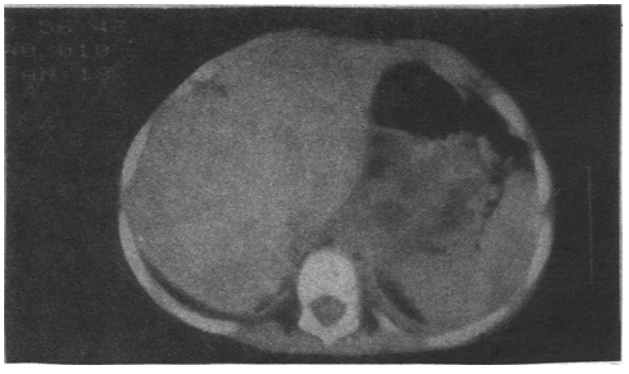

Fig. 2. CT abdomen after 4 weeks revealing a pseudocyst in the tail of the pancreas near splenic hilum

DISCUSSION

Pancreaticopleural fistula is a relatively rare complication of pancreatic disease, occurring in less than 1% of pseudocysts.[2] Cameron *et al*[3] suggested that the primary event is ductal disruption usually in the absence of acute pancreatic inflammation, in part explaining the lack of abdominal complaints in the majority of patients. Posterior disruption gives rise to peritoneal fistula that may track inferiorly into the pelvis[4] or superiorly through the diaphragmatic hiatus into the mediastinum, where it may form a pseudocyst,[5] or it may perforate into either or both pleural cavities or, rarely, into the pericardium.[6,7] Additionally pancreatic pseudocyst may enlarge; eroding directly to the diaphragm and giving rise to an effusion.[8,9] Few cases of pancreatic pseudocyst in the paediatric population have been reported in the literature.[10-13] The etiology of pseudocyst formation in children is quite different from that in adults. While toxins specially alcohol, are the cause of over 80% of pancreatic pseudocysts in adults, more than 60% of reported pseudocyst in children are the result of blunt abdominal trauma.[14] Other causes of pseudocyst formation in children have included pancreatitis (familial, infections and drug induced), intra-abdominal sepsis, and "idiopathic".[10-12] Pleural effusions associated with pancreatitis are usually haemorrhagic and have a high amylase content.[15]

An elevated serum amylase is often the first clue to the diagnosis of pancreaticopleural fistula. The elevation of serum amylase does not indicate active inflammatory disease but occurs as a result of passive absorption of amylase from the parietal/periotoneal surfaces.[16] Patients were considered to have pancreatico-pleural fistulae if (a) by radiologic, surgical or post-mortem examination, a pancreatic to pleural fistulous tract could be differentiated or (b) a large exudative pleural effusion was noted with an amylase level above 4000 I U/liter in the absence of neoplasm or oesophageal perforation.[17] Ultrasound may be useful in defining pancreatic abnormalities but computed tomography (CT) appears to be better in distinguishing pancreatic abnormalities like pseudocyst, mass lesion, calcification[18] and in defining extension of a pseudocyst into the mediastinum and pleura.

Initial management with conservative measures has considerable support in literature.[3] Thoracic drainage coupled with total parentral nutrition (TPN) and pharmacologic inhibition of pancreatic secretion (somatostatin) may result in the prompt resolution of the effusion with the closure of the fistula in upto 40% to 50% of cases.[19] Octreotide, a synthetic analog of somatostatin has a longer half life than somatostatin and can be administered subcutaneously. Most authors suggest continuing the above mentioned measures for 2-4 weeks.[20] Failure of medical therapy should be considered if there is failure of pleural effusion to clear, recurrence after the restarting of oral intake, or super infection.[17] For those patients who fail to benefit from medical therapy, surgery is indicated. Pre-operative assessment of pancreatic ductal anatomy with endoscopic retrograde cholangio-pancreaticography (ERCP) usually helps determine the precise site of fistula ligation. If pre-operative ERCP is unsuccessful pancreaticography may be performed at the time of surgery.[17] The specific operative approach depends upon the underlying anatomy of the pancreatic duct pathology. If the duct disruption is distal, distal pancreatic resection is often selected, and if the pancreatic duct leak or leaking pseudocyst is proximal, then Roux-en-Y loop drainage is utilized.[16] The outcome of these patients is usually good despite the advanced degree of underlying illness in many patients.

CONCLUSION

Chronic massive pancreatic pleural effusion (haemorrhagic or non-haemorrhagic) must be considered in the differential diagnosis of pleural effusion of unknown cause. Although infrequently reported, chronic massive pancreatic pleural effusion is probably more common than expected, and as awareness of the entity increases more may be identified.

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