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Pleural effusion due to concealed abdominal injury

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ABSTRACT

An eleven-year-old boy presented with massive left sided pleural effusion after non-response to intercostal chest tube drainage and empirical anti-tubercular treatment. The pleural fluid was hemorrhagic, did not grow any organism, nor had any malignant cell. A raised amylase level was indicative of pancreatic origin of the effusion. He had no abdominal sign. He had also developed disseminated intravascular coagulation. On revisiting history, the boy could recollect blunt abdominal injury prior to the onset of the illness. Endoscopic retrograde cholangiopancreatography revealed ductal leak at the neck of the pancreas that was endoscopically sealed using a stent. This case highlights the importance of a thorough search into the etiology of hemorrhagic pleural effusion. The symptoms and signs of pancreatic injury may be subtle, and the former should be suspected in case of blunt abdominal trauma.

Keywords: Blunt abdominal trauma, Disseminated intravascular coagulation, Pancreatic pleural effusion

Common causes of hemorrhagic pleural effusion are tuberculosis, trauma, malignancy and collagen vascular diseases [1]. Pancreatitis is an uncommon cause of pleural effusion. The leading causes of acute pancreatitis in children are biliary diseases, drugs, idiopathic, systemic diseases and trauma, followed by metabolic, hereditary and infectious diseases [2]. Abdominal trauma is one of the common causes (15 -40%) of pancreatitis in children, and blunt abdominal trauma being more common than penetrating injuries [3,4].

In a typical presentation of acute pancreatitis, upper abdominal pain may be mild or absent. Pancreatic injury may be missed or the diagnosis may be delayed because the initial symptoms and signs may be subtle, and this may add to the mortality and morbidity associated with the injury [3]. We report a case of pancreatitis due to blunt abdominal trauma which later presented as pleural effusion.

CASE REPORT

An 11-year-old Indian boy, presented with history of fever, cough, breathlessness, and weight loss over 2 months. He was treated with intercostal tube drainage and empirical anti-tubercular treatment at other centre; though, there was no tuberculosis contact. He was referred to us for further evaluation and treatment in view of non-response. On examination, he was undernourished and had tachypnea with respiratory distress. The air entry was reduced on the left side. A massive left sided pleural effusion was seen on radiography. On investigation, neutrophilic leucocytosis and raised erythrocyte sedimentation rate (67 mm at one hour) were notable.

The pleural fluid was hemorrhagic and had 120 cells/mm3 (neutrophils 20%, and lymphocytes 80%), plenty of red blood cells, elevated protein (5.9 gm/dl), and mildly elevated lactate dehydrogenase (450 IU/L) but

normal glucose. Microscopic examination of the pleural fluid did not reveal any microorganism or malignant cell. The polymerase chain reaction for tubercular bacilli and culture of the pleural fluid were also negative, so the Mantoux test. The pancreatic amylase level in pleural fluid was highly raised (1110 U/L). Serum amylase (906.3 U/L) and lipase (544.4U/L) levels were also elevated. Noting this, history was revisited. The patient could recollect trivial blunt abdominal injury in the school with fall over to a wooden desk, prior to the onset of illness. The associated abdominal pain had subsided in two days with oral medication.

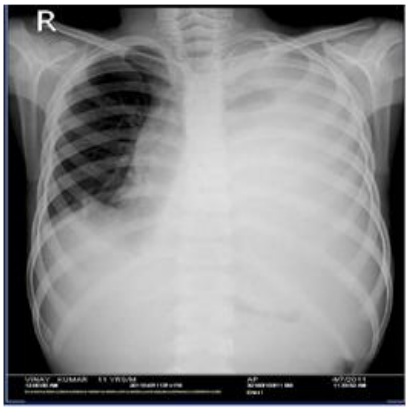

Figure 1: Chest X-ray showing massive left sided pleural effusion

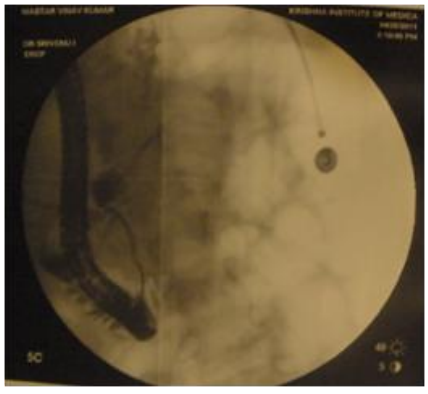

Figure 2: Ductal leak at the neck of the pancreas seen on Endoscopic Retrograde Cholangiopancreatography

On further workup, ultrasonography (USG) and computed tomography (CT) of abdomen revealed bulky pancreas with mediastinal collection, and echogenic thrombus in left internal jugular and subclavian veins. Fibrinogen disintegration product and D-dimer levels were elevated. Thrombophilia profile was normal. He was treated with inter-costal drainage, low molecular weight heparin and supportive care. Subsequently his drain output decreased and serum amylase level reduced to 220 U/L.

One week later, he developed vomiting, abdominal pain and tenderness. Serum amylase level was still elevated (226 U/L). Persistent symptoms and raised amylase prompted for Endoscopic Retrograde Cholangiopancreatography (ERCP). It revealed ductal leak at the neck of the pancreas (**Fig. 2**) for which endoscopic stenting was done. His condition improved over 2 weeks with normalization of serum amylase levels.

DISCUSSION

In children, 10 to 40% cases of acute pancreatitis are traumatic in origin and almost always have concurrent injury to other intra-abdominal organs. However, an isolated pancreatic injury is a relative enigma even in modern medical practice. The relatively protected retroperitoneal location of the pancreas protects it from most instances of blunt abdominal trauma. However, a sudden localized force to the upper abdomen can compress the organ against the vertebral column [4]. Blunt pancreatic injury is more common in children and young adults because they have a thinner or absent mantle of protective fat.

Acute pancreatitis in children can have variable presentation and may involve multiple organ systems. Respiratory complications such as pulmonary infiltrates or atelectasis (15%), pleural effusion (4%-17%), and pulmonary edema (8%-50%) are common [5]. Pleural effusions due to pancreatic diseases are often reactive with mildly elevated amylase levels. However, occasionally they can be exudative, bloody, and contain high amylase up to 30 times greater than the corresponding serum value [5]. Two common mechanisms of pancreatic pleural effusion are transdiaphragmatic lymphatic blockage or pancreatico-pleural fistula secondary to leak and disruption of the pancreatic duct or pseudocyst. Left-sided pleural effusion, once considered a marker of acute pancreatitis, is indeed a poor prognostic indicator and is associated with 20% to 30% mortality [5-8].

While the diagnosis of pleural effusion of pancreatic origin is based on a high amylase concentration, CT is necessary to define pancreatic abnormality and is capable of demonstrating pancreatic pseudocysts with direct

extension into the pleural cavity [5]. However, CT scan has low sensitivity to define ductal abnormality, wherein ERCP plays an important role in diagnosis as well as allows non-operative treatment [8-10]. Magnetic resonance cholangiopancreatography (MRCP) is alternative non-invasive diagnostic tool to assess ductal anatomy [4]. Blunt pancreatic injuries without ductal leak usually resolve with mere conservative management. On the other hand, damage to the ductal system, if inadequately treated or untreated, can result in prolonged morbidity [3,7].

Internal jugular vein thrombosis was considered secondary to subacute to chronic disseminated intravascular coagulation (DIC) due to ongoing pancreatic leak over a couple of months. Acute pancreatitis induces a strong inflammatory response, independent of the initiating factor for acinar cell damage. Inflammatory mediators in turn can influence hemostasis [3]. Coagulation abnormalities in acute pancreatitis can range from localized intravascular thrombosis to DIC. The latter when found upon hospital admission, is a poor prognostic indicator of acute pancreatitis [3].

CONCLUSION

Hemorrhagic pleural effusion in children demands a thorough investigation. Massive pleural effusion can mask the underlying pancreatitis. Increased pancreatic amylase in the pleural fluid can be an important clue. If conservative therapy fails, diagnostic and therapeutic ERCP should be considered in the management of pancreatic duct abnormality.

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