MASSIVE PLEURAL EFFUSION IN PANCREATITIS: 2 CASE REPORTS

J Abisheganaden, K N Sin Fai Lam, L S Chew

ABSTRACT

We report 2 patients with left-sided pleural effusion occurring in the setting of acute pancreatitis. Both patients had a strong history of alcohol consumption. In each case the pleural fluid amylase was markedly elevated, higher than that in the serum. The effusions resolved with closed chest tube drainage and the pancreatitis subsided with conservative therapy. In patients presenting with significant pleural effusions and acute upper abdominal symptoms, a thoracentesis with determination of the amylase titre may provide a quick means of diagnosing acute pancreatitis.

Keywords: *pancreatitis, pleural effusion, amylase*

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INTRODUCTION

Signs and symptoms of disorder in the lungs may accompany acute and chronic pancreatitis. Such involvement is a valuable lead in the diagnosis of pancreatic inflammation and has been well described in the literature. The pleuropulmonary complications of acute pancreatitis include pleural effusions, basal atelectasis, parenchymal infiltration, diaphragmatic elevation, bronchopleural fistula, pleural reaction, aspiration abscess, pancreatic pseudocyst and the adult respiratory distress syndrome[1]. Up to 15% of patients with acute pancreatitis[2] and an occasional patient with pancreatic pseudocyst or an acute exacerbation of chronic relapsing pancreatitis develop pleural effusions. We discuss 2 such cases of pleural effusion complicating acute pancreatitis and review the subject.

CASE REPORTS

Case 1

Mr A, aged 38 years, was admitted for complaints of left upper abdominal pain for one week associated with progressive dyspnoea. There was no history of trauma, nor symptoms to suggest congestive cardiac failure. He was a chronic consumer of alcohol, drinking on average one bottle of Chinese wine per day for the last 20 years.

He was afebrile but dyspnoeic at rest. Blood pressure was 110/70 mmHg on admission. A massive left pleural effusion was clinically evident (see CXR - Fig 1) and the abdomen was tender with guarding over the left hypochondrium. The full blood count showed leucocytosis (wbc 14000) and haemoglobin of 13.0g/dl. 800 ml of serosanguinous pleural fluid was aspirated on the day of admission for relief of breathlessness. Closed chest drainage via chest tube insertion was subsequently performed and this drained a total of 3100 ml of pleural fluid. Acute pancreatitis was supported biochemically by the elevated serial serum amylase and urinary diastase levels as shown in Table I.

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Fig 1 – Massive left pleural effusion on chest radiography (Case 1)

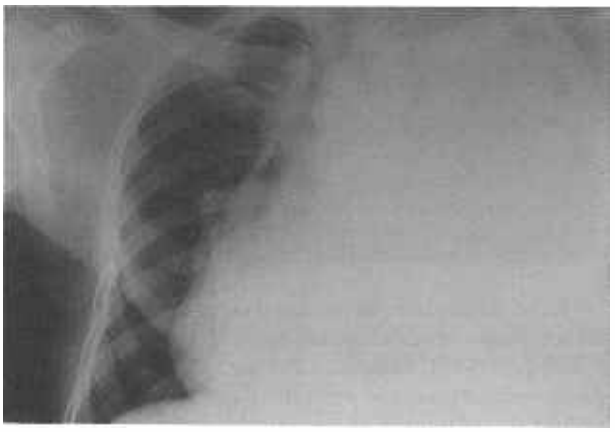


Table I – Serial serum, urinary and pleural fluid amylase levels.

Day	Serum amylase (NR 58-382 U/L)	Urinary diastase (NR 169-2200 U/L)	Pleural fluid amylase
1	1350	11650	45000
2	1410	4700	22500
3	1095	5550	16600
4	1324	6456	–
6	496	598	–

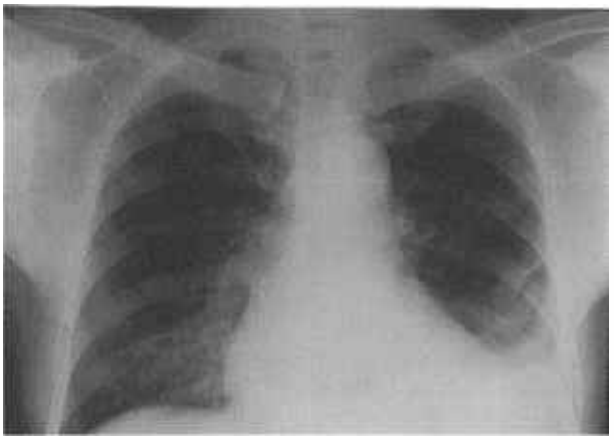
The pleural fluid was exudative, with specific gravity of 1.018 and packed with red blood cells. It was sterile on culture. Neither malignant cells nor acid fast bacilli were found. The pleural fluid amylase levels were markedly elevated. The serum calcium, inorganic phosphate, urea, electrolytes and creatinine levels were normal. Liver function test revealed a mild hypoproteinemic state with total protein 61 g/l (62-82) and albumin 28 g/l (37-51). Ultrasound of the pancreas was inconclusive as it was difficult to image the pancreas. There were no cystic collections.

He was treated conservatively with intravenous fluids, broad spectrum antibiotics and bowel rest. The effusion resolved with closed chest-tube drainage. However, he was discharged at his own request 10 days after admission. He was asymptomatic at that time.

Case 2

The second case is a 42-year-old Chinese male who first presented in 1988 to the medical unit for pleuritic chest pain of 3 weeks' duration. He had no other respiratory or systemic symptoms. He had a strong drinking history, averaging 2 bottles of beer or stout a day for 10 years. He also had a 10 pack-year smoking history. On examination, he had signs of a left pleural effusion and this was confirmed on radiology (Fig 2). Further investigation revealed that this was exudative (specific gravity 1.029, total protein 4.4 g/dl) and sterile. The effusion resolved with needle aspiration. He was treated empirically with oral ampicillin for a presumptive left lower lobe pneumonia.

Fig 2 - Left-sided pleural effusion on chest radiography (Case 2)



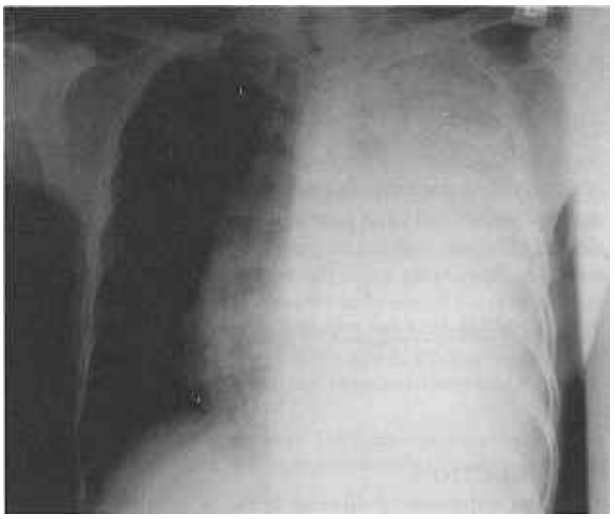
Eight months later, he presented to the surgical department for acute onset upper abdominal pain. The serum amylase was elevated at 920 U/L and urinary diastase 8900 U/L on admission. An explorative laparotomy revealed a grossly swollen pancreas with an area of necrosis at the posterior aspect of the head. He recovered with bowel rest and rehydration.

He subsequently developed 2 further episodes of acute pancreatitis which resolved with conservative treatment. No pancreatic calcifications were seen on plain abdominal films. Ultrasonography revealed a normal hepatobiliary system. The head of the pancreas was grossly normal. The body and tail were obscured by overlying bowel shadows. Three years after his first presentation, he was again admitted for acute on chronic pancreatitis with central abdominal pain and a serum amylase level of 580 U/L. On this admission, he was found to have a massive left pleural effusion (Fig 3). Further investigation revealed that this was exudative (specific gravity 1.032, total protein 5.1 g/dl) and with an elevated pleural fluid amylase level of 2560 U/L. The pleural fluid was sterile (no organisms seen, no bacteria or acid fast bacilli cultured) and cytology was negative for malignancy. A CT scan of the chest revealed left pleural effusion with partial consolidation of the left lung. There were no mediastinal masses (Fig 4). He recovered with closed chest tube drainage and intravenous anti-microbial therapy(gentamicin and crystalline penicillin).

DISCUSSION

Acute pancreatitis as a cause of pleural effusion is well documented. However, attempts to estimate the frequency of such effusions have yielded widely differing results. Hammarsten found it in 15% of his series of patients with acute pancreatitis[2]. Lipp and Aaron found pleural effusion in 40% of their cases, usually on the left side[3]. Coffey reported 6 instances of pleural effusion in 135 patients with pancreatitis[4]. In all 6, the effusion

Fig 3 – Massive left-sided pleural effusion on chest radiography (Case 2)



was on the left. Of 31 cases of pancreatitis with pleural effusion in which information was given, 21 effusions were on the left only, 3 on the right, 7 were bilateral[2]. In those series which relied upon radiological examination, the recorded frequency of pleuropulmonary abnormality in patients with pancreatitis varied between 14% and 53%. Considering pleural effusion separately, the figures varied between 3% and 17%.

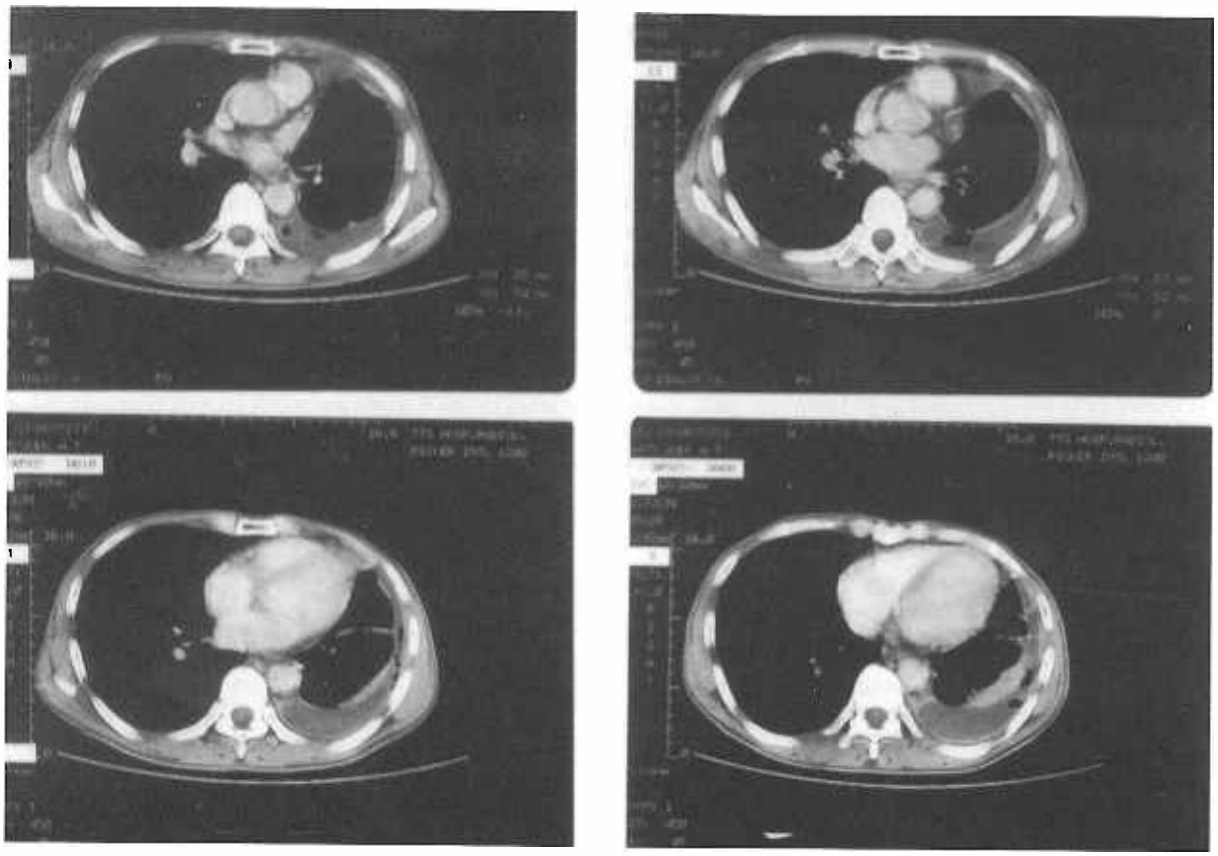
Our first patient presented with acute pancreatitis and a massive left pleural effusion. The pleural fluid amylase was markedly elevated compared with the serum value, which was also significantly elevated. Although elevated serum concentrations of amylase can be seen in a variety of conditions, the concentration of amylase in the pleural fluid is elevated only in pancreatitis, ruptured oesophagus, and occasionally in malignant pleural effusions due to bronchogenic and pancreatic carcinoma. In a ruptured oesophagus the effusion is almost always on the left side, the fluid tends to be purulent, and the elevated concentration of amylase is of salivary origin, due to leakage of swallowed saliva into the pleural space[5]. In bronchogenic carcinoma the elevation is also secondary to the salivary type of amylase.

The second patient was presumed to have a parapneumonic effusion when he was first seen in 1988. Although the diagnosis of pancreatitis was only established in 1989 when he underwent a laparotomy, its causative role in the inflammatory changes of the pleura was not realised earlier as he did not manifest any symptoms or signs of pancreatic disease at that first presentation. It was only three years later in 1991 when he developed a massive pleural effusion secondary to another attack of pancreatitis that the pleural fluid amylase level was confirmed to be elevated.

Several authors have investigated pleural fluid amylase in patients with effusions unrelated to pancreatitis. Wemer, Hammarsten et al, and Warter et al, examining 7, 12 and 21 pleural effusions respectively, found the amylase levels in all cases to be within normal limits. Modai, Hazard, and Domart similarly found a slightly elevated amylase level in only one effusion, secondary to carcinoma, out of 29 examined[6].

There is no clear-cut explanation for the formation of pleural effusions in pancreatitis, including the fact that they occur more often on the left side than on the right. Several theories have been postulated, viz: i) direct contact of pancreatic enzymes with the diaphragm, ii) haematogeneous carriage of pancreatic enzymes to pleura, iii) direct movement of fluid from abdomen to thorax, iv) diaphragmatic perforation by a pancreatic

Fig 4 – Computerised tomography showing left pleural effusion with partial consolidation of the left lung (Case 2)



pseudocyst, and v) transfer of fluid into pleural cavity by trans-diaphragmatic lymphatics[6].

The movement of enzymes into the pleural cavity by a lymphatic route would appear to be of considerable importance. There are rich lymphatic connections between the abdomen and thorax through the diaphragm. Such connections serve as auxiliary channels and are used during times of obstruction of lymph drainage, as would occur in pancreatitis with its inflammatory reaction[7]. Anatomically, the tail of the pancreas comes in direct contact with the diaphragm. With the acute inflammation of pancreatitis, direct contact with the pancreatic secretions may give the needed chemical irritation to cause the pleural effusion, and with retrograde lymph drainage to maintain the elevated pleural amylase. Such effusions are occasionally massive and the fluid is often haemorrhagic. The elevated amylase titre in the pleural fluid appears to persist for a longer time than the elevated serum levels during the period of recovery[8]. The striking predilection of enzyme-rich effusions for the left pleural cavity may be largely explicable on simple anatomical grounds, since the pancreas is more intimately related to the left hemidiaphragm than to the right. In the few instances of right-sided pleural effusion, dissecting pancreatic pseudocyst or aberrant location of pancreatic tissue to the right of the midline are thought to be possible explanations.

Although pleuropulmonary changes have been found in approximately 30% of cases of acute pancreatitis, appreciable amounts of pleural effusion have been observed in 15%, according to Hammarsten et al[2]. There is little doubt, however, that small amounts occur more frequently. Other thoracic signs described have included a sluggish, elevated or immobile diaphragm, interlobar adhesions, basilar atelectasis and pneumonic infiltrates[9-11]. Such radiological features may occur in various forms of inflammation of the upper abdomen, especially when ileus is present. Therefore, these changes are not specific for pancreatitis. The parenchymal lung infiltration is not easily explained, but in some cases may be due to a complicating bacterial pneumonitis.

It is evident that, regardless of the pathogenesis of the effusion seen in pancreatitis, an appreciable number of patients do have abnormal chest X-ray findings. This indicates that roentgenographic examination of the chest will be of aid in the diagnosis. Furthermore, when fluid is present, a thoracentesis with examination of the fluid for its amylase content should be taken and compared to that of a simultaneous serum specimen as it may help confirm the diagnosis of pancreatitis[8,11].

CONCLUSION

Pleural effusion is a frequent complication of acute pancreatitis. These 2 cases illustrate that pleural effusion accompanying an obscure acute disease of the upper abdomen may provide an easy avenue to the diagnosis. In both cases the effusion occurred on the left side and both had elevated amylase values. These observations agree with those of previously reported cases. Only rarely has pleural effusion occurred on the right side. In the presence of pleural effusion, a thoracentesis with determination of the amylase titre in the fluid may provide a quick and easy means of diagnosing acute pancreatitis.

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