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HAEMORRHAGIC PLEURAL EFFUSION IN ACUTE PANCREATITIS

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The literature contains very few reports of haemorrhage remote from the pancreas in acute pancreatitis. This is probably not a true reflection of its incidence and 2 cases are recorded here drawing attention to this feature of the disease.

CASE REPORTS

Case 1

B.K., a Native female aged 53, was admitted to hospital on 20 September 1955 in a moribund state. The story was difficult to elicit but apparently she had complained of intermittent attacks of abdominal pain during the past 4 months. The pain was in the epigastrium and radiated to the hypochondrium on the left and to the back between the shoulder blades. During the 2 days before admission to hospital the abdominal pain was severe and persistent and she would not take any food.

On admission the patient looked very ill. She was apathetic and disorientated; marked general weakness and hypotonia. Evidence of severe peripheral circulatory collapse—cold, clammy extremities, profuse sweating, only the femoral pulses palpable, pulse 120 beats per minute and the blood pressure unrecordable. She was cyanosed. No clinical jaundice. No evidence of congestive cardiac failure. Generalized abdominal tenderness was observed, more marked in the epigastrium. There was slight guarding of the left upper abdominal wall and the physical signs of a left basal pleural effusion were elicited; a chest X-ray confirmed this finding and on aspiration of the left chest heavily blood-stained fluid was withdrawn. The patient died before further investigation or treatment could be instituted. The provisional clinical diagnosis was pulmonary infarction.

Autopsy was performed 24 hours after death. The findings were: Acute haemorrhagic pancreatitis with complete necrosis of the pancreas and extensive haemorrhage into it. Necrosis of the peripancreatic retroperitoneal fat and of the fat of the mesentery of the small intestine and transverse mesocolon. Serosanguinous ascites was present. The muscle of the diaphragm was normal but there was a fibrinous exudate on the peritoneal surface of the left vault. The left pleural cavity contained a heavily blood-stained effusion measuring approximately 3 pints. There was no pulmonary infarction or thrombosis of the pulmonary vessels, and the left lung, which was compressed against the hilum, was otherwise normal. There was a slight excess of lightly blood-stained fluid in the pericardial sac. A chronic cholecystitis was observed. There were multiple faceted mixed gall-stones in the gall-bladder and several similar stones in the common bile-duct which was dilated to a diameter of 1 cm. The splenic vein contained a recent ante-mortem thrombus except at its two extremities and there was diffuse fatty change in the liver but no evidence of cirrhosis. Histologically the pancreatic and fat necrosis were confirmed. There was slight periductal and periacinar fibrosis.

Case 2

V.B., European male aged 46, was admitted to hospital on 16 March 1956. He had been a heavy drinker for the last 10 years. He had suffered from attacks of upper abdominal pain, which were always precipitated by alcoholic bouts, the attack of pain lasting 1 day. Following such an episode at the end of February he experienced a pleuritic pain in the left lower chest anteriorly and this was associated with pyrexia. The chest pain lasted for 5 days and recurred early in March, when it was associated with a non-productive cough. He had been tired and listless during that time.

On admission to hospital an ill-defined cystic mass was palpable in the left upper quadrant of the abdomen. This mass moved moderately well on respiration. There was stony dulness at the left base and in the left axilla. In this area there was diminished air-entry and a friction rub was audible. The blood pressure was 150/110 mm. Hg, but there was no other evidence of cardiovascular disease. Approximately 1 pint of dark, uniformly blood-stained fluid was aspirated from the left chest. This fluid was centrifuged in a Wintrobe tube and the red deposit constituted 35% of the total. Unfortunately there is no record of the character of the supernatant fluid. The protein content of the aspirate was 4·5 g.%, and no tubercle bacilli were seen in smears of it. There was a mixed growth of salivary organisms and non-pathogenic yeasts from a fresh sputum specimen and no tubercle bacilli were seen in a 24-hour specimen. Serum albumen was 4·0 g.%, serum globulin 2·5 g.%. Serum amylase 40+ units and 20 units per c.c. on different occasions (normal 10 units per c.c.). 24-hour urinary diastase 128,000 units, 167,000 units and 292,000 units on different occasions (normal 30,000 units). The glucose tolerance test showed a diabetic curve: Fasting blood-sugar 137 mg.%; ½ hour after ingestion of 50 g. of glucose, 275 mg.%; 1 hour 207 mg.%; 2 hours 163 mg.%. Blood urea 17 mg.%, and serum calcium 8·9. mg.%. Electrocardiograph was within normal limits. The sedimentation rate was 90 mm. in the first hour (Westergren). Blood count: Haemoglobin 10·9 g.%, packed-red-cell volume 37%, white blood-cells 5,600 per c.mm., peripheral blood-smear normal. The urine contained a trace of glucose. Straight X-ray of the abdomen showed diffuse pancreatic calcification and

barium meal revealed displacement of the stomach with distortion of its contour by a mass in the lesser sac.

COMMENT

Haemorrhage into the pancreas and structures in close proximity to it is an essential feature of the haemorrhagic variety of the disease. It is thought to be due to the local effects of the proteolytic digestion of blood vessels. Haemorrhage into areas remote from the pancreas may be caused by one of two different mechanisms—the tracking of the proteolytic enzymes to beyond the peritoneal cavity, or a general bleeding tendency. A few cases of such remote haemorrhage have been described in the literature. Beck[1] recorded one case with a right-sided haemothorax and scattered haemorrhages in the lung together with a small haemopericardium. Smith,[2] Werner[3] and Anglem and Lee,[4] each recorded an example of haemothorax in acute pancreatitis. In 2 of these the side was recorded, the one the right and the other the left. Petechiae on the buttocks have been noted.[5] Renal haemorrhage and haemorrhage into the skin of the abdominal wall is also scantily recorded.[6,7]

While the authenticity of the first case is clear, the diagnosis of haemorrhagic pleural effusion in the second is a clinical one and possibly open to other interpretation. The fluid withdrawn might have come from the cyst or mass below the diaphragm, and the radiological appearances might have been due to an elevated left dome. While this possibility cannot be completely excluded we feel that the patient most likely had a left-sided haemothorax. This effusion may have been formed during an acute flare-up of chronic pancreatitis.

The blood in the pleural sacs of these two patients could have been the result of either of the two postulated mechanisms. If one invokes the local effects of enzymes then they would have to gain access through the natural openings of the diaphragm. In the absence of any other effects of these enzymes in the pleural cavity of the first patient it seems likely that the bleeding may have resulted from a general bleeding tendency. Innerfield *et al.*[8,9] showed in rabbits and in dogs that the intravenous administration of trypsin prolonged the bleeding and coagulation times, caused a fall in prothrombin, Ac-globulin, antifibrinolysen, fibrinogen, and antithrombin. The fall in the plasma level of the last is transient and there is a secondary sustained rise of antithrombin. They also showed that there was a parallel between the plasma antithrombin and trypsin levels. The initial drop in antithrombin may increase the tendency to thrombosis temporarily, followed later by a bleeding state. It seems likely that the general bleeding state is induced by the liberation of activated trypsin into the systemic circulation.

Case 1 demonstrates that acute pancreatitis must be considered in shocked states associated with haemorrhagic pleural effusions. Pulmonary infarction is a much commoner cause of such a clinical picture, and abdominal pain occurring in the latter may further complicate the issue.

SUMMARY

1. Two cases with haemorrhage into the left pleural sac in acute pancreatitis are recorded.
2. Pancreatitis should be considered in the differential diagnosis of haemothorax.
3. Possible mechanisms are discussed.

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