PAINLESS ACUTE PANCREATITIS

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Introduction
PAIN is the most prominent feature of acute pancreatitis and its severity is sometimes considered a rough index of the degree of pancreatic damage (Blumenthal and Probststein, 1959). It has also been said to be rarely, if ever, absent (Richman, 1956). The following is a report on a patient who had several fairly typical episodes of acute pancreatitis, followed by a final fatal attack, with complete absence of pain.

Case Report
A soldier aged 39 was admitted to hospital in May 1959. An exacerbation of bronchitis had been followed by severe colicky epigastric pain, which spread through to the back, accompanied by repeated vomiting. The serum amylase was suggestive of acute pancreatitis at 291 Somogyi units; he was treated accordingly and his condition improved. Five days later there was a recurrence of pain, constant in nature this time, and the amylase rose to 1,600 units. An intravenous cholecystogram after recovery showed good concentration and normal function of the gall-bladder.

Four times in the next year the patient suffered similar but less severe episodes, all apparently following acute respiratory infections with a productive cough; but only once did the patient report sick. In July 1960 he was sent to hospital again, after a further attack of bronchitis had been followed by four days of very severe vomiting; the patient was emphatic that he had had no pain at all on this occasion. However, he was obviously dehydrated and profoundly shocked with a barely palpable pulse and a blood pressure of 80/65. The abdomen was distended, with some epigastric tenderness but no rigidity; bowel sounds were present. Investigations revealed a serum chloride of only 52 m. equiv. litre and a serum amylase strikingly elevated at 6,000 Somogyi units per 100 ml. Treatment included intravenous fluids, tetracycline, atropine and gastric aspiration.

The patient had passed a little urine shortly before coming to hospital, but twelve hours after admission only 20 ml. were obtained by catheterization. The restoration of fluid and electrolyte balance resulted in a free flow of urine and a marked improvement in his general condition. After four days, when the amount of gastric aspirate was getting much less, he became confused and disoriented with the development of a coarse tremor. The abdomen became more distended and, at this stage, laparotomy was undertaken; the diagnosis was confirmed and cholecystostomy performed to drain the biliary system. However, the patient became increasingly
jaundiced and lost consciousness the day after operation; death occurred after several generalized convulsions.

**Post-mortem Findings**

The greater omentum was stiffened and adherent to the loops of intestine it covered. The mesentry of the small intestine showed oedema and fat necrosis and contained thrombosed radicles of the portal vein. The pancreas was grossly oedematous and disorganized by fibrosis and necrosis: its coverings were hemorrhagic. The pancreatic duct and common bile duct had a common pathway of 5 mm. into the duodenum. The portal vein contained adherent ante-mortem thrombus. The gall-bladder was normal.

Histologically the features were extensive fibrosis and inflammatory infiltration of the pancreas, an early perilobular fibrosis of the liver accompanied by small and large fatty droplet change, acute cholangitis and an inflammatory reaction around the portal vein with early organization of its contained thrombus.

**Discussion**

In the last fatal episode the diagnosis of acute pancreatitis was made, despite the lack of pain, on the previous history and the serum amylase level: it was confirmed at laparotomy and autopsy. The serum amylase level was particularly high, but there was significant oliguria on admission, and since amylase is excreted in the urine, this would itself raise the serum level to some extent. A figure of 1,388 units (measured by Van Loon’s method with normal range 60-160 units) has been reported with acute renal insufficiency alone (Meroney *et al*., 1956).

Evans *et al.* (1958), in a retrospective study, found pathological evidence of acute interstitial pancreatitis in ten cases with no record of pain in the final illness, but absence of hemorrhage and necrosis was one of the criteria for inclusion in their series. We have found no previous record of painless acute hemorrhagic pancreatitis. For it to be recurrent, however, is by no means unusual; Thal *et al.* (1957) found old scarring at autopsy in 36 out of 42 cases.

Local venous thrombosis is often associated with acute pancreatitis (Enquist and Gliedman, 1958), but involvement of the portal vein itself does not seem to be common. It was thought in this case to have been a major factor leading to the death of our patient.

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**REFERENCES**


