adenitis of lacrimal glands with local conjunctival injection. Sclerae white. No signs of iritis." Rapid recovery ensued, and by March 28 all symptoms and signs had disappeared.

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An Unusual Case of Acute Pancreatitis

The following case of a woman aged 39 who died of acute pancreatitis on the 37th day of her disease presented two unusual features: (1) the appearance, in the early stage of the disease, of a syndrome which was indistinguishable from diabetes mellitus; and (2) death from intra-abdominal haemorrhage.

CASE REPORT

The patient was admitted to hospital on January 9, 1950, with the following history. For two years she had been subject to repeated attacks of severe epigastric pain with some flatulence, the symptoms being unrelated to food and not relieved by taking more food or alkaline powders. On January 5 she had an extremely severe attack of this indigestion. There were no further definite symptoms, although she felt unwell, until three days later, when, after a normal bowel motion, she developed severe upper abdominal pain and vomited. The pain and vomiting continued until her admission. She admitted to some backache when questioned, but could not localize it to either the lumbar or the interscapular region. When the urine was found to be loaded with sugar and acetone she was questioned about a history of loss of weight, of polyuria, or of polydipsia. She denied ever having had these symptoms.

On examination she was pale, anxious, and moderately shocked: blood pressure 130/105, temperature 98° F. (36.7° C.), pulse 120, respirations 25. Her tongue was dry and furred. The normal respiratory movements of the abdomen were diminished; there was tenderness and guarding over the upper abdomen and slight tenderness over the lower abdomen; she felt a sharp pain on the release of pressure over the whole abdomen; there was no diminution of liver dullness; the abdomen was silent on auscultation. Rectal examination disclosed acute tenderness in the pouch of Douglas. Examination of the respiratory and cardiovascular systems revealed no abnormality. Urine: brick-red with Benedict's solution; acetone +++ (Rothera's test); chlorides, 3 g. per litre (Marriott's test); diastatic index (result not received until after laparotomy), 250 units. Blood chemistry: sugar, 258 mg. per 100 ml.; urea, 93 mg. per 100 ml.; chlorides, 468 mg. per 100 ml.; protein, 5 g. per 100 ml. Radiography showed no evidence of gas under the diaphragm.

Because the clinical features were so much more like a perforated peptic ulcer than an acute pancreatitis and the diastatic index was not available, it was decided to do a laparotomy. An intravenous infusion of normal saline was started, and 10 units of soluble insulin was given together with 30 g. of glucose intravenously. On opening the peritoneal cavity sero-sanguineous fluid escaped. Numerous areas of fat necrosis were seen on the peritoneum and the great omentum. The gall-bladder contained at least one large calculus. A diagnosis of acute pancreatitis having been made, a drain was placed in the lesser sac and the incision closed.

The patient made satisfactory progress for the first 10 days. On the morning after operation the blood-sugar level had fallen to 153 mg. per 100 ml. A further dose of 20 units of soluble insulin covered by 50 g. of glucose intravenously was given. The urine was tested with Benedict's solution every four hours, and, apart from one orange result on the third post-operative day (this was associated with a blood-sugar level of 267 mg. per 100 ml.), the test

persistently gave a green or blue result. A glucose-tolerance test was done on the 13th day after operation. The result was as follows:

Time	Blood-sugar Level	Urine
Fasting	90 mg. per 100 ml.	No sugar
1 hour	175 " " " "	" " " "
1 1/2 hours	205 " " " "	Sugar "+++"
2 " "	174 " " " "	" "+++"
2 " "	202 " " " "	" "+++"

The whole-blood chlorides remained within normal limits.

During the first week there was a purulent bronchitis, but up to the 10th post-operative day the patient's general condition gradually improved. On this day a thick yellow discharge from the drain began, and she started to go downhill. She lost her appetite and began to waste rapidly. The nature of the discharge soon altered, portions of necrotic pancreas being seen. The discharge began to digest the abdominal wall. Continuous suction was applied to the drain, and this proved very effective in protecting the abdominal wall. A severe anaemia was associated with the rapid wasting. On the 14th post-operative day the haemoglobin was 10.56 g. per 100 ml. (72%). There was no response to iron, liver, or transfusion, and her condition deteriorated rapidly. Thirty-seven days after the onset of the acute illness dark blood began to pour from the drainage tube. A blood transfusion was begun, and she received 3 litres of stored blood. She died six hours after the haemorrhage began.

Post-mortem examination showed thick blood-stained fluid and clots in the peritoneal cavity, a plastic peritonitis involving particularly the small intestine, fat necrosis of the greater omentum, and disintegration of the left half of the pancreas. Histological examination of the surviving portion of the pancreas showed the normal architecture to be quite well preserved; groups of acini showed disorganization and autolysis, but there was no haemorrhagic infiltration. The pancreatitis in this situation appeared to be resolving. The gall-bladder contained a large stone and several smaller calculi; its wall was moderately thickened.

COMMENT

The interest in this case arises not from the presenting symptoms and signs but from the complications.

Although it is not uncommon for there to be a slight glycosuria, so far as can be ascertained it is uncommon for a severe glycosuria and hyperglycaemia to be found. There can be no doubt that before the onset of the acute pancreatitis this patient had no symptoms to suggest that she suffered from severe diabetes mellitus and it seems unlikely that she had even mild diabetes. Yet 36 hours after the onset of the acute illness her urine was loaded with sugar and acetone and her blood-sugar level was considerably raised. This diabetic state slowly subsided and her blood-sugar level and urine were normal by the 12th post-operative day. Next day the glucose-tolerance test was abnormal. The pathogenesis of this phenomenon was probably a complete temporary inhibition of the islet tissue due to the action of the escaping enzymes. The islet tissues then gradually recovered, though incompletely. It is well known that mild diabetes mellitus may follow recovery from acute pancreatitis. It is interesting to note that although this was a severe case of the disease the histological examination of the pancreas showed many areas of apparently normal pancreatic tissue.

I do not know of any other case of acute pancreatitis in which haemorrhage from a splenic vessel was the cause of death, but it cannot be considered a surprising complication. It must be presumed, in spite of the histological findings, that the disease was still active, that the enzyme was still being liberated, and that this digested the wall of the vessel.

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