Medical Memoranda

Bleeding and Pancreatitis

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It is now recognized that significant bleeding is common in acute pancreatitis. Howard (1960) stated that since 1946 half the fatalities in his patients have been due to this cause. He also stated that the haemorrhage may be gastrointestinal or through the wound in cases which have been operated on. Kirby *et al.* (1955) noted that haemorrhage in acute pancreatitis was usually preceded by fever and most commonly occurred 14 to 21 days after the onset of the illness. They suggested that the sequence of events was pancreatitis and necrosis, leading to erosion of vessels. This seems a sufficient explanation for the bleeding within and around the pancreas at the onset and during exacerbations of pancreatitis, but it does not explain the gastrointestinal bleeding which occurs later in the disease and usually without accompanying evidence of continuing abnormal enzyme activity. In the case here reported post-mortem examination yielded a satisfactory explanation for prolonged gastrointestinal bleeding in a patient with pancreatitis.

CASE REPORT

A man aged 28, who had had a partial gastrectomy (Polya type) for duodenal ulcer in 1954, remained well until ten weeks before being referred to hospital in April 1963, when he complained of epigastric pain referred to the back, loss of appetite, and loss of weight. He was a pale, thin, otherwise healthy-looking subject and the whole upper abdomen was tender to palpation, but no abnormal mass was palpable. Barium-meal x-ray examination showed a small stomach remnant, normal in outline, and a normal stoma. Free hydrochloric acid was absent after histamine test meal and the stools did not contain occult blood. There was a mild hypochromic anaemia (Hb 9 g./100 ml.). A few days after admission pain became more severe and the serum amylase level was 800 units/100 ml. (Somogyi).

Eight months later he was readmitted because of increasing pain and worsening general condition. The haemoglobin level was now 6.8 g./100 ml. and the stools contained occult blood. Three days later fresh blood was vomited and there was frank melaena. The haematemesis was repeated on at least eight occasions in the ensuing three weeks, and on each occasion the vomiting was preceded by severe upper abdominal pain. Thirty-one pints (17.6 l.) of blood were transfused during this period. Four weeks after this second hospital admission signs of peritonitis developed and laparotomy was performed. The peritoneal cavity contained bile, which was escaping from a perforation in the duodenal stump. The perforation was closed and an interanastomosis between the afferent and efferent loops of the gastrojejunostomy was performed. Three months after the laparotomy the patient was discharged home apparently well.

Two months later (June 1964) he was readmitted with haematemesis and melaena, and in September and November similar incidents requiring further readmission to hospital occurred. On each occasion blood transfusion was necessary, 24 pints (13.6 l.) of blood being transfused. Gastroscopy (Mr. J. D. Rose) revealed intense mucosal congestion of the stomach remnant.

In January 1965 there was further massive bleeding, and as it seemed likely to originate in the stomach remnant this was removed. Examination during and after operation showed no gross abnormality either in the stomach remnant or at the stoma. Death took place two days later.

Post-mortem Findings.—Anastomosis between oesophagus and jejunum was intact. There were no oesophageal varices. Liver (1,900 g.): portal system dilated, no cirrhosis. Spleen (950 g.) was grossly enlarged; numerous adhesions to surrounding tissues. Pancreas was very hard; the splenic vein was thrombosed and entirely surrounded by tissue of hard consistency continuous with the pancreas. The cause of death was bleeding from the alimentary tract due to chronic congestive splenomegaly, arising from splenic-vein compression and thrombosis occurring as a result of pancreatitis.

COMMENT

The clinical improvement which occurred after the first laparotomy in this case suggested that his disability had been due to back pressure on the duodenal stump with ulceration and finally perforation of that stump, and that the improvement resulted from the decompression effected by the interanastomosis performed between the loops of the gastrojejunostomy. The events suggest that such back pressure did exist and there is clinical and experimental evidence that such back pressure may cause pancreatitis. McGowan and Wills (1964) found that a special relation existed between high serum amylase levels and partial gastrectomy, and that in these patients there was evidence of afferent-loop obstruction. They suggest the abnormal amylase levels are due to high pressure within the duodenum being conveyed along the pancreatic duct. McCutcheon (1962, 1964) stated that all the known facts about acute pancreatitis are consistent with the hypothesis that most cases are caused by reflux of duodenal contents along the pancreatic ducts. Hinton (1962) quotes experimental work by his associate Pfeffer, in which, using dogs and a closed duodenal loop, he found that when such a loop was left attached to the pancreas all the dogs developed acute pancreatitis, and this occurred even when the common bile duct had been divided. Thus it could not be due to bile regurgitation, and it was postulated that it must be due to increased pressure within the duodenal loop.

No other explanation for the pancreatitis in the case of this young patient is apparent and the post-mortem findings make it almost certain that the splenic-vein thrombosis and splenomegaly were secondary to these changes in the pancreas. The splenic vein receives blood from the short gastric veins and the left gastro-epiploic vein and it is reasonable to conclude that splenic-vein obstruction will lead to congestion in areas drained by these veins and that in some instances bleeding will occur. Gastrointestinal bleeding from this cause would be likely after some days or even weeks of illness and this accords well with the known facts when bleeding occurs in patients with pancreatitis.

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