Perforation of Common Bile-duct with Pancreatitis after Childbirth

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Pancreatitis during or shortly after pregnancy is well recognized but uncommon, though Langmade and Edmondson (1951) believed that it would be diagnosed more often if borne in mind when nausea, vomiting, and epigastric pain occurred in pregnancy or the puerperium. Spontaneous rupture of the common bile-duct in the absence of surgery, trauma, or obstructing stone is exceedingly rare (Hart, 1951). The case reported below of spontaneous rupture of the common bile-duct during an attack of post-partum pancreatitis seemed worth recording as the clinical features make some contribution to an understanding of possible aetiological factors in both conditions.

CASE REPORT

A married woman aged 26 years was admitted on 9 August 1963 at 2.30 a.m. complaining of abdominal pain and vomiting. Nine weeks previously she had delivered normally a full-term healthy baby. The pregnancy had been normal except that in the last six weeks she had three attacks of colicky xiphisternal pains radiating to the shoulder blades. Two weeks after delivery the pains recurred, coming about every other day two hours after meals, and two weeks later they were radiating also down towards the umbilicus. The pains started at 9 p.m. on the night of admission, were occurring every 10 minutes, and lasting three minutes, when suddenly at 1.0 a.m. she experienced very severe generalized abdominal pain referred to both shoulder tips, and vomited six times. There was no history of any other significant gastro-intestinal symptoms, there were no abnormal dietetic factors, and her only serious past illness was infective hepatitis at 10 years.

On examination she looked ill, but was not shocked or anaemic. Her temperature was 99.2° F. (37.3° C.), pulse 92, blood-pressure 170/100. The abdomen was rigid and tender all over but not distended, bowel sounds were diminished, she was tender on rectal examination, other systems were normal, and radiographs of the abdomen showed no abnormality. The clinical picture suggested peritonitis due to perforation of a viscus. At laparotomy the peritoneal cavity contained several ounces of free bile and acute oedematous pancreatitis was present. The gall-bladder appeared healthy but tensely distended, the common bile-duct was tense but of normal calibre and no stones were felt. On compressing the gall-bladder bile leaked from two tiny cracks in a small patch of fat necrosis on the right side of the common duct. The biliary tract was decompressed by cholecystostomy, which produced some biliary "sand" with otherwise normal-looking bile, and Morrison's pouch was drained.

The results of investigations subsequently carried out were Hb 94%, W.B.C. 8,000/c.mm., E.S.R. 45 mm./hr. (Westergren), serum amylase 1,400 Somogyi units. The calcium, phosphate, cholesterol, bilirubin, and liver-function tests were normal except for an alkaline phosphatase of 26 K.-A. units, and there was no glycosuria. Post-operatively she was treated with gastric aspiration, intravenous fluids, pethidine, and tetracycline. No bile drained from Morrison's pouch, a cholangiogram taken on the tenth day through the cholecystostomy tube was normal but showed pancreatic-duct reflux, and she was well on discharge home.

In January 1964 she was readmitted for cholecystectomy and sphincterotomy in view of the evidence of sphincter dysfunction. She reported no pains since the previous admission, there were no abnormal physical signs, her blood-pressure was 110/70 mm. Hg, the serum amylase, liver-function tests, fasting blood sugar and lipids were normal, and the W.R. was negative. At operation the

pancreas was moderately nodular, the common bile-duct and sphincter of Oddi were normal, and the gall-bladder appeared normal, though histology later showed chronic inflammation. The post-operative course was satisfactory and she was well at follow-up.

COMMENT

Hart (1951) found in the literature 10 cases of spontaneous rupture of the common bile-duct, the cause of which the various writers attributed to one or more of the following factors: increased pressure due to spasm of the sphincter of Oddi; necrosis secondary to calculus; infection; diverticulum; necrosis secondary to thrombosis of mural vessels; and activation of reflux pancreatic juice through a common opening of the pancreatic and bile-ducts resulting in tissue digestion. Ellis and Cronin (1960) thought that mural infection was a likely explanation, possibly infection of an intramural gland. Tiny perforations leaking only under pressure suggested that in this case the cause was increased pressure due to obstruction of the sphincter, with the duct wall weakened either by mural infection or by tissue digestion. The latter hypothesis cannot be discounted, in view of the fat necrosis on the duct. The obstruction may have been due to spasm or oedema perhaps aggravated by infection or calculi in the ampulla of Vater.

Langmade and Edmondson (1951) tabulated clinical features in 53 collected cases of pancreatitis associated with pregnancy and found first symptoms or an exacerbation occurring in the puerperium in 73%. In his series of 90 cases of pancreatitis Joske (1955a) had 11 females whose symptoms had started within a few months of childbirth. Joske believed that pancreatitis associated with pregnancy was a form of relapsing pancreatitis of obscure metabolic origin (1955b) and discussed various metabolic changes in normal pregnancy, including hyperlipaemia in relation to aetiology. Doubilet (1957) maintained that a common channel of the bile and pancreatic ducts was present in all cases of relapsing pancreatitis, the key to aetiology being spasm of the sphincter of Oddi. Smith (1962) suggested that contamination of the pancreatic duct with infected bile might sometimes be an aetiological factor.

No evidence of primary metabolic disorder was found in this case, and the biliary "sand" was probably due to stasis and infection. There was increased pressure in the bile-ducts at operation, histological evidence of cholecystitis, and a common channel. The tense distension of the gall-bladder in spite of leakage suggested that obstruction of the sphincter preceded the symptoms of pancreatitis. The obstruction almost certainly played a major part not only in the perforation of the common bile-duct but probably in the pancreatitis as well, though perhaps the pancreas had been made susceptible by metabolic factors.

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