weak, slightly better on the left. Plantar flexion was good, but dorsiflexion was impaired, more in the ankles than the toes. Loss of sensation to pain and temperature persisted, a sensory level being established at D 8 on the left and D 10 on the right. There was normal appreciation of light touch, and joint position sensation was present in the toes. Vibration sense was absent in the lower limbs, though a few weeks later it had returned at all levels except the ankles. The ankle jerks were by then easily obtained, but the knee jerks remained diminished.

During the four months following the onset of the paraplegia he received anticoagulant and diuretic therapy, and his cardiac function continued to improve. There was a slight left ventricular heave and a slurred apical first sound; the blood pressure was 130/70 and the lungs were clear. Radiography showed that cardiac size had decreased since 23 March. He performed quite strenuous limb exercises without loss of breath or angina, but there was little further change in his neurological findings. He was discharged on 30 August 1968, walking quite well with leg callipers and the help of a Zimmer frame.

When seen again on 12 October, six months after the onset of paraplegia, there had been a slight generalized improvement in sensation to pain and temperature, though there was still a dorsal sensory level and an area of complete loss over the left shin. He was able to accomplish a slightly wider range of movements with his legs through skilful use of the available muscle power. It was doubtful whether the affected movements showed any true increase in power. Wasting of the quadriceps was still pronounced, and there had been no further change in the reflexes. Although all knee movements were weak, by hyperextending these joints he was able to stand unsupported and to walk while pushing his wheel-chair. Using callipers he could walk 100 yards (91 m.). There was no heart failure and no cardiac enlargement clinically. Heart sounds were normal, and blood pressure was 155/80. The E.C.G. was unchanged.

COMMENT

From the clinical nature of the paraplegia with sensory loss and the lack of permanent cerebral damage it would seem that a defective blood supply to the spinal cord (possibly related to atheroma of the aorta) was a factor in the selective infarction of the cord. The main lesion was in the lower cord, where there was evidence of anterior horn damage at L 3 and L 4, and sensory levels were established at D 8 on the left and D 10 on the right. The sensory defect was of the dissociated type, with marked loss of pain and temperature sensation and sparing of light touch and joint position modalities. Impairment of vibration sense was transient.

Henson and Parsons (1967) reported the case of a 59-year-old woman who developed an areflexic paraplegia and a low thoracic level of analgesia after a myocardial infarct which led to a 65-minute episode of ventricular fibrillation. They suggested that cord infarction was precipitated by this temporary circulatory impairment, but that this may have been preconditioned by vascular damage in an earlier thoracoplasty for tuberculosis. In their case, as in ours, it seems reasonable to postulate that in the presence of local vascular disease an episode of hypotension was capable of producing cord infarction.

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Rupture of Choledochus Cyst in Pregnancy

British Medical Journal, 1969, **3**, 573–574

Since Vater first described the condition in 1723 many cases of choledochus cyst have been recorded in the literature. The subject was fully reviewed by Shallow *et al.* (1946) and Attar and Obeid (1955). Rupture of the cyst is a rare complication, only six cases having been documented by 1956 (Tagart, 1956); one of these, according to Friend (1958), occurred two weeks after a normal delivery.

A search of the literature confirms this case to be the first recorded instance of rupture of a choledochus cyst during pregnancy.

CASE REPORT

The patient, an unmarried primigravida aged 17, was first seen at the booking clinic on 7 June 1968. She gave a 10-year history of recurrent attacks of abdominal pain for which no cause had been found. In 1961 she had been admitted to hospital with a short episode of jaundice. The serum bilirubin level was 1·1 mg./100 ml. A cholecystogram at that time showed poor concentration of the dye in the gall bladder, but no other abnormality. A diagnosis of infectious hepatitis was made. The jaundice faded and she was discharged after three weeks.

The pregnancy proceeded uneventfully until the twenty-eighth week, when she was admitted for investigation of severe epigastric pain radiating to the back. She had not vomited. There was no bowel or micturition upset and no vaginal bleeding.

On examination the patient was distressed and flushed, but did not appear ill; her temperature was 98·2° F. (36·8° C.), pulse 80 per minute, and blood pressure 110/70 mm. Hg. The abdomen was soft, and there was some tenderness in the epigastrium and in the right costal margin. Full blood counts, electrolytes, urea, and serum amylase were normal. The urine was not infected and did not contain porphyrins.

Twelve hours after admission the patient experienced sudden knife-like pains in the right iliac fossa; these had gradually spread to involve the whole abdomen. Her symptoms were unlike those of previous attacks and were accompanied by profuse vomiting. Her temperature was 100° F. (37·8° C.) and pulse 110. There was a strong fetor oris. The abdomen was rigid to palpation with maximal guarding in the right iliac fossa and pronounced rebound tenderness. A diagnosis of perforated acute appendicitis was made and laparotomy was performed on 20 October.

Operative Findings.—The peritoneal cavity contained a large quantity of heavily bile-stained fluid. Retroperitoneal oedema and bile staining were present in the region of the head of the pancreas, extending up into the lesser omentum and down along the right paracolic gutter. The gall bladder was collapsed, with no stones. The pancreas could not be identified with certainty on account of the oedema. The lesser sac was obliterated by adhesions and the aditus could not be seen. Though there was no evidence of fat necrosis a tentative diagnosis of acute oedematous pancreatitis was made. Cholecystostomy was performed and the abdomen was closed, leaving a drain to the region of the pancreas.

The patient was transferred to the intensive care unit and went into labour 24 hours later. Delivery was by low forceps under pudendal block. The infant, a girl of 3 lb. 9 oz. (1,600 g.), at first

responded well to resuscitative measures but died 24 hours after birth. At necropsy the cause of death was shown to be anoxia due to prematurity and hyaline membrane disease.

The patient made slow but satisfactory progress. A tube cholangiogram performed 11 days after operation outlined a choledochus cyst 10 cm. in diameter (see Fig. 1). It was assumed

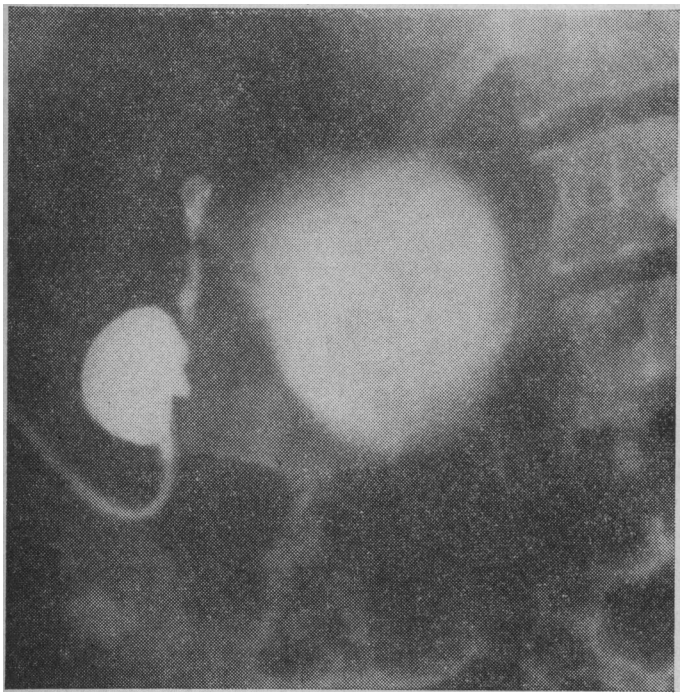

FIG. 1.—*X*-ray film showing choledochus cyst and collapsed gall bladder.

that the cyst had ruptured, giving rise to biliary peritonitis, but had re-formed in the postoperative period. It was decided to perform elective reconstruction of the biliary tree after three months, but after removal of the cholecystostomy tube the patient developed severe cholangitis necessitating early surgical intervention. This was done on 1 December.

Findings at Second Laparotomy.—A choledochus cyst containing small pigment stones involved the whole of the supraduodenal part of the common bile duct. There was an abscess cavity in the right paracolic gutter at the site of the original rupture. An operative cholangiogram showed gross distension and cysts of both hepatic ducts in the liver. The abscess was drained, the gall bladder removed, and the cyst anastomosed to the jejunum by Roux-en-Y (see Fig. 2).

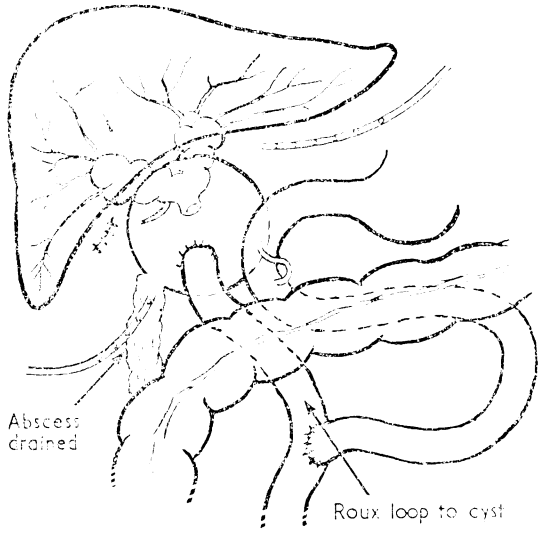

FIG. 2.—Operative procedure.

The patient made a good recovery and was discharged on 7 January 1969 free of symptoms. At a follow-up one month later she was extremely well, with no abnormal findings. When seen after three months she was in good health.

COMMENT

Choledochus cyst is a localized dilatation or enlargement of the biliary tree characteristically limited to the supraduodenal portion of the common bile duct. Sometimes there is stenosis or angulation of the ampulla of Vater, but usually no abnormality is found at the outlet of the common duct (Maingot, 1957).

Aetiologically the condition is generally regarded as a congenital anomaly of the duct system, a fact borne out by the high incidence in young patients. Sherlock (1965) quotes a figure of 80% of cases first discovered under the age of 10. The condition may present in the newborn, but rarely before one year. Aird (1949) maintains that pregnancy may precipitate and exaggerate the symptoms.

In this patient there was no evidence of the classical triad of obstructive jaundice, right upper abdominal pain, and a palpable tumour. The intermittent pain experienced since the age of 7 could be accounted for by bouts of cholangitis not producing sufficient obstruction to precipitate enlargement of the cyst.

Though the diagnosis was not made at the initial laparotomy, drainage of the biliary system was the correct primary treatment. It was not surprising that the degree of biliary peritonitis and surgical trauma precipitated labour. There was no indication for caesarean section at the time of laparotomy.

Definitive treatment of the condition formerly consisted of external drainage of the biliary tree with or without excision of the cyst. The high mortality from that procedure led to primary anastomosis of the cyst to the duodenum. Cholecystjejunostomy with a Roux loop has recently provided an excellent alternative.

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