Silk sutures in the common bile duct

Silk ligatures occasionally act as the nidus for calculus formation in the biliary tract, the gall bladder, or the common bile duct. We report a patient in whom two silk sutures used to ligate the cystic duct during cholecystectomy subsequently caused obstructive symptoms. It is strongly recommended that only absorbable suture material be used to ligate the cystic duct.

Case report

A 69-year-old woman underwent cholecystectomy in December 1973 after an attack of cholecystitis. She had no history of jaundice. At operation a single large stone was palpable in the gall bladder; the cystic duct and common bile duct were not dilated. Because of the solitary stone and normal sized ducts, operative cholangiography was not performed. Postoperatively she had several episodes of biliary colic. The first attack occurred two weeks after discharge from hospital and this attack was accompanied by slight clinical jaundice. An intravenous cholangiogram done at that time showed no abnormality.

The patient was not seen again until August 1976, when she was referred back to the surgical outpatient department because of three severe attacks of colicky pain in the right upper abdomen within one week. She described the pain as exactly the same as that experienced before the cholecystectomy. She was readmitted for further investigation because it was felt in view of her history and the omission of operative cholangiography that there were retained calculi in the common bile duct. Nevertheless, a further intravenous cholangiogram with tomography showed that the common bile duct had a normal calibre with no stones present. After this investigation it was decided to keep the patient under outpatient review. The colicky pain continued and despite the absence of jaundice a transhepatic cholangiogram using a fine Okuda type needle was performed. On this occasion the common bile duct was slightly dilated and at the lower end of the common bile duct there were two filling defects, which were thought to be gall stones. A laparotomy and exploration of the common bile duct was performed on 9/277. The common bile duct was a little dilated but no calculi could be palpated. Irrigation of the duct using a fine catheter, however, flushed out two silk sutures (see figure). Probes could not be passed into the duodenum so the lacer was opened and the ampulla identified. A sphincterotomy was performed which allowed free passage of bougies; the common bile duct was closed with a T-tube in situ. One week after operation the T-tube cholangiogram showed free flow into the duodenum with no filling defect.

The notes of the first operation recorded that the cystic duct had been doubly ligated with silk sutures. It is reasonable to assume that these silk sutures found their way into the common bile duct soon after operation and were causing intermittent biliary colic. Four months after operation the patient was well.

Comments

Although the formation of stones around non-absorbable sutures has been known since Homan, we report a patient, surgeons continue to use silk for ligation of the cystic duct after cholecystectomy. This may be due to the misplaced distrust of surgeons in catgut or because some textbooks continue to advise silk for ligation of the cystic artery and cystic duct despite the many reports that silk may act as a nidus for stone formation. In a comprehensive review of stones resulting from suture material in the biliary tract Silvennoinen showed that stones were found to have formed only around non-absorbable suture materials. The first pain and transient jaundice in our patient were experienced three weeks after operation and possibly the sutures had entered the common bile duct by that time. Larmi and Silvennoinen showed how rapidly a silk thread in the wall of the gall bladder may reach the common bile duct and form a stone. This case report also illustrates the difficulty in diagnosis in patients who have pain after cholecystectomy. Only with the aid of a transhepatic cholangiogram were the filling defects seen.

It is generally recognised that non-absorbable materials should not be used in the vicinity of the urinary bladder, and we propose that the same principle should apply to the gall bladder.

We thank Mr D L Crosby for allowing us to present a patient under his care and also for his constructive criticism.

References


(Accepted 8 July 1977)

Department of Surgery, University Hospital of Wales, Cardiff
B1 REES, FRCS, senior surgical registrar
G JACOB, FRCS, senior surgical registrar

Chronic lymphatic leukaemia, chlorambucil, and sensorimotor peripheral neuropathy

We are unaware of chlorambucil ever having been implicated in causing a peripheral neuropathy and therefore report the following case.

Case report

A 49-year-old man presented in 1976 with a two-week history of bruising and purpura. He had had lymphocytoma cutis in 1964-7, which had been confirmed by biopsy. Blood count and film had been normal at that time. He had extensive purpura on his limbs and trunk. The liver was palpable 9 cm below the costal margin, and the spleen just palpable. There was no lymphadenopathy. There were no neurological abnormalities. Haemoglobin was 13.7 g/dl, white blood count (WBC) 179 × 10⁹/l, and platelets 13 × 10⁹/l. Red blood cells were normochromic and normocytic. Differential count showed 99% mature lymphocytes; many smear cells were seen. Bone marrow aspiration showed a heavy infiltration of mature lymphocytes. The picture was that of chronic lymphatic leukaemia.

He was treated with chlorambucil 10 mg/day and prednisone 20 mg/day. The chlorambucil was reduced to 2 mg over 10 weeks, the WBC having fallen to 18:2 × 10⁹/l. The dose of prednisone had been reduced to 5 mg/day. He then complained of a burning sensation in his fingertips and difficulty in typing his letters. His gait was unsteady, but he denied dizziness. The significant findings on examination were absent tendon reflexes unaccompanied by demonstrable sensory loss. The prednisone was tailed off and the chlorambucil continued. Two weeks later his symptoms had worsened dramatically. He was confined to a wheelchair because of repeated falls.

On admission to this hospital examination showed a glove and stocking superficial sensory loss; absent joint position sense in the fingers and toes; loss of vibration sense below the hips; and symmetrical weakness of distal muscle groups, which was more pronounced in the legs, with bilateral foot drop and loss of plantar flexion. The reflexes were absent, with flexor plantar responses.

Investigations—Haemoglobin concentration was 15·0 g/dl, WBC 13·0 × 10⁹/l, and platelets 43 × 10⁹/l. Differential count showed neutrophils 3·6 × 10⁹/l, lymphocytes 8·0 × 10⁹/l, monocytes 0·5 × 10⁹/l, and eosinophils 0·3 × 10⁹/l. Erythrocyte sedimentation rate; serum vitamin B₁₂, serum folate, red blood cell folate, plasma urea electrolyte, and creatinine concentrations; chest radiograph; liver function; protein electrophoretic strip and immuno-