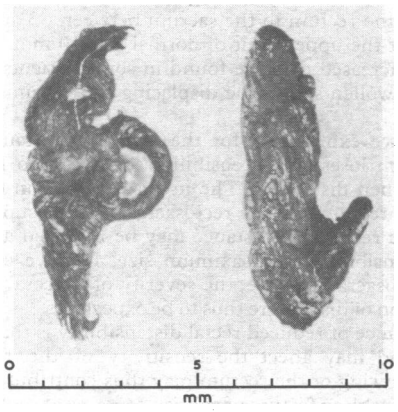
Silk sutures in the common bile duct

Silk ligatures occasionally act as the nidus for calculus formation in the biliary tract, the gall bladder, or the common bile duct.[1] 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 9/2/77. 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 latter 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 two silk sutures removed from common bile duct.

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[2] described such 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[3] 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[4] 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[5] 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.

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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) 179×10^9/l, and platelets 13×10^9/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 $18{\cdot}2 \times 10^9$/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 tying his laces. 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 $13{\cdot}0 \times 10^9$/l, and platelets 43×10^9/l. Differential count showed neutrophils $3{\cdot}6 \times 10^9$/l, lymphocytes $8{\cdot}6 \times 10^9$/l, monoctyes $0{\cdot}5 \times 10^9$/l, and eosinophils $0{\cdot}3 \times 10^9$/l. Erythrocyte sedimentation rate; serum vitamin B_{12}, serum folate, red blood cell folate, plasma urea electrolyte, and creatinine concentrations; chest radiographs; liver function; protein electrophoretic strip and immuno-

globin estimations; and glucose tolerantce were all normal. There were no urinary porphyrins or porphobilinogen. Rectal biopsy showed no amyloid. Viral serology and antinuclear factor, Rose-Waaler, and VDRL slide tests gave negative results. The cerebrospinal fluid was sterile, with no leucocytes and a protein concentration of 0·3 g/l.

The findings were consistent with a peripheral sensorimotor neuropathy; the origin was obscure.

Five days after admission chlorambucil was stopped. One week later there was noticeable improvement, the patient being mobile with two crutches. After three weeks he was discharged. Two months later he needed only a walking-stick; all reflexes were present, except the ankle jerks, and there was substantial recovery of power in dorsiflexion and plantar flexion. Nerve conduction studies showed normal proximal nerve conduction in the median nerve (41 m/s). There was an increased terminal latency of 10 ms with temporal dispersion. Both the antidromic median and ulnar sensory volleys showed increased latency and diminished amplitude of response. The clinical diagnosis was thus confirmed.

Studies after 10 weeks showed a median nerve conduction velocity of 41 m/s, with a terminal latency of 6 ms—a significant improvement.

Comment

Peripheral sensorimotor neuropathy has rarely been recorded in chronic lymphatic leukaemia.[1 2] A detailed study of the neurological manifestations of the reticuloses did not include a single case.[3] The central nervous system manifestations of chlorambucil toxicity are likewise extremely uncommon and present as lethargy, drowsiness, and subsequent convulsions.[4 5] Peripheral neuropathy has not been noted. The cause of this patient's neuropathy remains obscure, but chlorambucil cannot be excluded.

We thank Professor F G J Hayhoe for his kind permission to report this case.

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Rectosacral distance and rectal size in ulcerative colitis

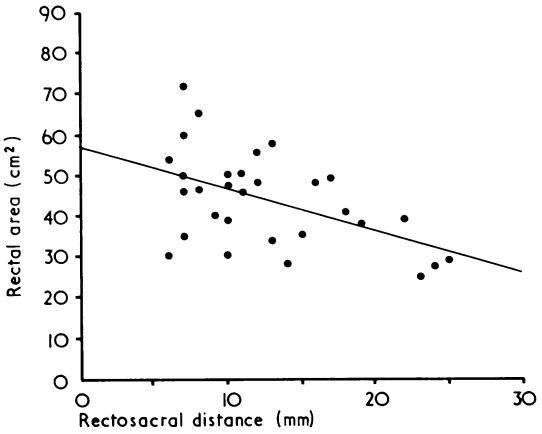
The space between the rectum and sacrum may be increased in colitis.[1 2] We have examined the relation between rectosacral distance and rectal size in colitis and tried to correlate the results with the age at onset, length of history, and extent of the disease.

Methods and results

Measurements were made on true lateral films of the pelvis taken when the rectum was fully distended with air during double-contrast barium-enema examinations. The distance between the dorsal line of the barium and the ventral surface of the sacrum was measured at the midpoint of the fourth sacral vertebra. Rectal size was measured with a 10-mm grid as the area below a perpendicular line drawn to a tangent at the level of S2. No correction was made for magnification.

Measurements in 31 patients with ulcerative colitis (15 women and 16 men, each with a mean age of 44 years) were compared with those in 20 controls (10 women and 10 men; mean ages 51 and 46 years respectively) who underwent barium-enema examination for gastrointestinal symptoms but had a normal rectum on sigmoidoscopy. The significance of difference between the two groups was assessed with Student's t test, and correlations were calculated by means of linear regression analysis.

The mean rectosacral distance was 12·5 ± SE 1 mm in the patients with colitis compared with 5·4 ± 0·5 mm in the controls ($P < 0·001$). Conversely, the rectal area in the patients with colitis was 43·6 ± 2 cm², compared with 59·6 ± 2 cm² in the controls ($P < 0·001$). The figure shows the inverse correlation ($r = 0·53$; $P < 0·01$) between the rectosacral distance and rectal size in the patients with colitis. There was a positive correlation between the duration of symptoms and the rectosacral distance ($r = 0·49$; $P < 0·01$).



Regression line showing inverse correlation between rectal area and rectosacral distance in 31 patients with colitis.

The earlier the age at onset of the colitis, the smaller the rectum tended to be ($r = 0·36$; $P < 0·01$). No significant correlation was found between rectal size and the length of history or between the extent of the colitis and rectal size or rectosacral distance. In the controls there was no correlation between age and rectal size or rectosacral distance.

Comment

Rudhe[1] found that the upper limit of the normal rectosacral distance in children was 7 mm at S4. Edling and Eklof[2] measured the shortest distance from the rectum to the sacrum between S3 and S5 in adults and found that the upper limit of normal was 10 mm. These authors ascribed the increased distance found in some patients with colitis to periproctitis, swollen soft tissue displacing the rectum forwards from the bone.

An alternative explanation for the increased distance in colitis is that the rectum loses its distensibility so that it no longer fills the sacral curve when distended. The inverse relation in colitis between the size of the rectum and the rectosacral distance supports this concept. Thus the rectosacral distance may be regarded as an indication of rectal distensibility and maximum size. Positive correlations between the rectosacral distance, the severity of mucosal inflammation,[1] and the duration of disease are thus to be expected.

The importance of reduced rectal distensibility is that it tends to be irreversible and may affect the sensitivity of the rectum to distension.[3] Narrowing of the rectum may thus contribute to urgency of defecation, a disabling feature of colitis.

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