Figure 1 Intraoperative sinogram

persisted and the patient was then referred to St Mark's Hospital, a colorectal tertiary centre. Here, when examined under anaesthesia, she was found to have two holes in the posterior vaginal wall. One of these was cannulated and an intraoperative sinogram was performed under fluoroscopic control. Contrast filled a cavity of about 5×5 cm. Occlusion of the inlet with a pledget provided enough pressure to show filling of an obstructed lymphatic vessel on the right side of the sacrum (Figure 1). The cannulated sinus was extended to facilitate drainage of chyle. The patient declined further interventions.

COMMENT

Chyloperitoneum is a rare complication of abdominal surgery that should be considered in cases of persistent abdominal distension. Sampling should yield sterile, lipaemic fluid. We have found no previous report of a lymphatic fistula draining externally via the rectum or vagina. To determine the site of the leak the mainstay of imaging is lymphangiography,¹ though its lack of total sensitivity is illustrated by the present case. Lymphangiography can be done as an intraoperative procedure during attempted surgical repair, and localization may be further improved by injection of patent blue violet.² CT in conjunction with lymphangiography can help to establish or exclude the presence of associated retroperitoneal masses or lymph node enlargement. It may also be of assistance in improving localization of abnormal retroperitoneal and pelvic lymphatic vessels. Lymphatic leaks have also been localized with the use of oral iodine-marked fatty acids (¹²³I-pentadecanoic acid).³

The management options for lymphatic collections and fistulae include a diet high in medium-chain triglycerides, a period of total parenteral nutrition, insertion of a peritoneovenous shunt, catheter drainage, sclerotherapy with tetracycline and surgical closure.⁴⁻⁷ If the present patient changes her mind about treatment the best option might be surgery to clear tissue away from the vaginal vault.

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Spastic paresis in Paget's disease

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Paget's disease should be considered in the differential diagnosis of spinal cord compression when MRI is suggestive of malignant disease.

CASE HISTORY

A woman of 60 had for eighteen months noticed altered sensation in her legs, beginning in the feet and progressing up both legs to involve the trunk to the level of her breasts. Her legs were also weak and stiff. She had become increasingly unsteady and had fallen several times; for the past year she had used a stick to walk, and for the last month a frame. Recent new features were urinary urgency and occasional incontinence. There was no history of acute

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Figure 1 **Sagittal MRI of spine.** (a) T1-weighted; (b) T2-weighted; (c) fat-saturation T1-weighted

Figure 2 **Sagittal CT of spine**

back pain but she reported a longstanding dull ache in the lumbar region. She had experienced no disturbance of vision or speech or swallowing.

On general examination the only noteworthy finding was a raised body mass index. The abnormal findings in the nervous system were bilateral weakness of hip flexion and knee flexion, MRC grade 4/5, with brisk lower limb reflexes, adductor jerks and extensor plantar responses consistent with a spastic paraparesis. She had bilateral sensory change to light touch and pinprick to the level of the 4th thoracic segment. Joint position sense and vibration were lost to her ankles. The combination of upper motor

neuron signs in the legs, normal findings in the arms and a sensory level suggested a lesion of the thoracic spinal cord. MRI (Figure 1) gave a low T1 and T2 weighted signal in the 1st, 3rd and 4th thoracic vertebrae with cord compression at the 3rd and 4th and an associated bright cord signal. The cord narrowing was principally due to posterior epidural tissue of mixed low and high T1-weighted and T2-weighted signals. Fat-saturated T1-weighted images indicated the bright portion of this tissue to be epidural fat.

Blood tests were normal apart from an alkaline phosphatase of 488 IU/L and an erythrocyte sedimentation rate of 12 mm/h. Protein electrophoresis was normal, as were the tumour markers carcino-embryonic antigen, Ca125, Ca199 and alpha fetoprotein. CT of the chest, abdomen and pelvis showed multiple areas of bony sclerosis and lucency, without obvious expansion, within many of the vertebral bodies, both iliac bones and the right 7th rib. There was no evidence of primary malignancy. CT of the thoracic spine (Figure 2) revealed expansion and mixed sclerosis and lysis of the bodies and posterior elements of the 1st, 3rd and 4th thoracic vertebrae. There was pronounced stenosis of the bony canal, mainly due to overgrowth of the posterior elements. The expansion of the bone seen on CT is characteristic of Paget's disease. The patient was treated with intravenous zoledronate and showed some improvement at 6-week follow-up.

COMMENT

Cord compression in Paget's disease of bone is due to both mechanical impingement and vascular distortion.¹ Histopathologically, an increase in multinucleated osteoclasts results in bone resorption with lytic lesions, and is followed

by osteoblastic activity and formation of abnormal new bone that is highly vascular, fragile and sclerotic. There is hyperaemia of the surrounding tissues. Osteoblasts are seen around thick and vascular trabeculae of new bone, and there may be wide seams of uncalcified osteoid. The end result is dense bony sclerosis, with the thickened, deformed bone characteristic of Paget's disease. In the patient reported here, epidural lipomatosis contributed to the cord narrowing. The signal characteristics of the abnormal bone on MRI may be indistinguishable from sclerotic bone metastases. However, expansion of the bone in association with sclerosis and lysis, well seen on plain radiographs or CT, is diagnostic of Paget's disease.

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Intussusception complicating typhoid fever

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Common surgical complications of typhoid fever are ileal perforation and gastrointestinal haemorrhage;^{1,2} intussusception has been little reported.

CASE HISTORY

A boy of 14 was admitted after 5 days of fever, 4 days of abdominal pain, and 3 days of bilious vomiting and passage of 'red currant jelly' stools. At another hospital he had received antibiotics without improvement. On examination the abdomen was distended, with bowel sounds absent and peritonism. An intraluminal mass was felt per rectum and the clinical diagnosis was intussusception. Abdominal radiographs showed dilated bowel loops and multiple fluid levels. Ultrasonographic demonstration of intussusception was precluded by the vast amount of bowel gas. At

laparotomy he was found to have an ileo-ileo-colic intussusception without pathological lead-point. Subserosal petechiae were seen all over the small bowel. The intussusception was easily reduced and the bowel was viable. Palpation of the terminal ileum revealed the Peyer's patches to be grossly enlarged. He was treated with ampicillin, gentamicin and metronidazole.

The abdominal tenderness and fever persisted post-operatively. On day 3, a Widal test was positive for both flagellar (H) and somatic (O) antigens of *Salmonella typhi* (1:320 dilutions); *S. paratyphi* agglutinins were negative. However, blood, stool and urine cultures did not grow typhoid bacilli. When the fever continued he was given intravenous ceftriaxone—to which *S. typhi* is usually sensitive in our region. During the subsequent 4 days, features of intestinal obstruction returned—abdominal distension, copious bilious nasogastric aspirate and multiple fluid levels on radiographs. The differential diagnosis was recurrent intussusception, perforation of the subserosal haemorrhagic spots or adhesive bowel obstruction.

A repeat laparotomy on day 7 showed 100 mL of exudative fluid in the peritoneal cavity (*Escherichia coli* grown on culture). The entire small bowel was inflamed and aperistaltic but no perforation, intussusception or adhesions were seen. The Peyer's patches were no longer enlarged. After peritoneal lavage, the abdomen was closed. The postoperative course was complicated by prolonged ileus necessitating total parenteral nutrition. On day 10 the Widal test was stronger, with H and O antigens of *S. typhi* present at 1:400; but *S. paratyphi* agglutinins remained negative. On day 15, because of persistent fever, the antibiotic was changed to intravenous ciprofloxacin, which was continued for 15 days. The patient lost his fever on day 16, bowel sounds returned on day 22 and he was discharged on day 29. Widal tests showed a gradual fall of agglutinin titres over the next 6 weeks; blood, stool and urine cultures remained sterile.

COMMENT

The association of typhoid fever and intussusception is intriguing. In mice³ the lipopolysaccharide found in salmonella cell walls has been reported to predispose to intussusception by altering gut motility. Matsushita *et al.*⁴ described intussusception in an adult with enterocolitis due to *S. typhimurium* but we have found no report of typhoid fever and intussusception in a child.

S. typhi, because of its affinity for lymphoid tissue, causes enlargement of Peyer's patches in the ileum. Still, enlargement of Peyer's patches is commonly blamed for idiopathic intussusception in infants,⁵ we hypothesize that such a mechanism is responsible for typhoid-induced intussusception. In our patient the coexistence of

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