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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intussusception and typhoid fever made therapeutic decision-making difficult. Non-gangrenous intussusception is preferably managed by non-operative means such as hydrostatic or pneumatic reduction.⁵ However, the presence of fever, duration more than 24 hours, and features of peritonitis are relative contraindications for non-operative reduction (all being suggestive of bowel gangrene).⁵ In such cases even diagnostic barium enema studies are contraindicated for fear of colonic perforation. In the present case, the fever and abdominal signs of typhoid were mistaken for those of gangrenous intussusception and the opportunity for non-operative reduction was lost. The negative bacteriological results were probably due to previous antibiotic therapy, and we presume that the organism was resistant to ceftriaxone. The postoperative intestinal obstruction was due to the prolonged ileus of

typhoid fever. The absence of published evidence as to this possibility was the reason for the avoidable second laparotomy.

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