An unusual cause of bowel obstruction

A Huang, D L McWhinnie, G P Sadler

A 73-year-old woman presented with a 24-hour history of colicky abdominal pain, abdominal distension and vomiting. Medical history revealed that her bowels were opening normally and there was no history of recent change in bowel habit or weight loss. She was hypertensive and had not undergone previous abdominal surgery. The patient had previously been investigated for rectal haemorrhage when a barium enema was performed (figure 1). Examination of her abdomen revealed distension with no scars or external herniae. On auscultation tinkling bowel sounds were heard. No peritonism was present and digital rectal examination was normal. Plain abdominal radiograph was obtained (figure 2). The patient underwent an exploratory laparotomy, following which she made uneventful recovery. A post-operative small bowel enema confirmed the diagnosis (figure 3).

Figure 1 Barium enema

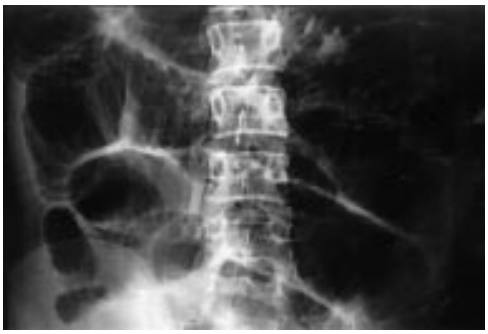

Figure 2 Plain abdominal X-ray

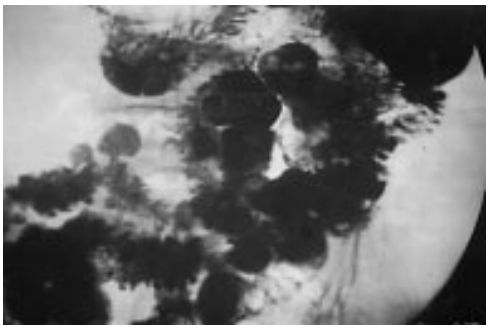

Figure 3 Small bowel enema

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Questions

1 What is the diagnosis?
2 What are the differential causes of this condition?
3 What are the surgical options at laparotomy?

Answers

QUESTION 1
Mechanical small bowel obstruction.

QUESTION 2
Small bowel obstruction may be divided into luminal, intrinsic or extrinsic (box 1). Adhesions, herniae and intra-abdominal neoplasms account for 95% of cases with all other conditions being relatively rare. At laparotomy the patient was found to have an enterolith impacted in the ileum with proximal small bowel distension and distal collapse. Multiple jejunal diverticulae were noted, commencing at the ligament of Treitz and extending distally for 45 cm. The enterolith had migrated from a diverticulum into the small bowel lumen and impacted distally causing a bolus obstruction. Extensive diverticular disease of the large bowel was also noted and the rest of the laparotomy was normal.

Causes of small bowel obstruction

Luminal
- foreign bodies
- faeco- and enteroliths
- gallstones
- bezoars
- parasites
- polypoidal tumours

Intrinsic
- atresia
- tumours
- strictures (including tuberculosis, Crohn's)

Extrinsic
- adhesions
- herniae
- volvulus
- intussusception
- bands
- inflammatory masses
- neoplastic masses

Box 1

QUESTION 3
Small bowel obstruction due to an enterolith may be relieved either by enterotomy or by simply crushing the enterolith digitally and milking the fragments into the caecum (as was done in this case). In rare cases of small bowel diverticular disease, where intussusception, haemorrhage or perforation are present, the diseased segment should be resected and primary anastomosis performed. It may, however, be necessary to exteriorise the bowel if gross contamination or infection is present.

Obstruction caused by band adhesion or volvulus can frequently be relieved by the division of the band without resection. However, where the bowel is ischaemic or gangrenous, resection with primary anastomosis should be performed.

Learning points

- causes of small bowel obstruction may be categorised into luminal, intrinsic and extrinsic
- most jejunal diverticula are asymptomatic (60%). When present, symptoms include abdominal discomfort, flatulence, borborygmi, malabsorption, pseudo-obstruction, stasis or 'blind loop' syndrome
- surgical intervention may be necessary for intestinal obstruction, perforation, haemorrhage and neoplastic growth
- obstruction may arise from enterolith formation, volvulus, intussusception, and adhesion bands

Box 2

Discussion

Jejunal diverticula are usually acquired, multiple, and located on the mesenteric border of the small bowel where the vessels penetrate the muscle. Incidence at autopsy is 0.7%,[1] but this is probably an underestimate as the radiological detection rate is up to 2.3%.[2] Aetiology is unclear but formation may be from disordered small bowel function and structure, leading to abnormal intestinal motility.[3] Synchronous colonic diverticulosis is present in 30–61% of patients.[4]

Asymptomatic jejunal diverticula discovered incidentally should be left alone. About 40% of patients with small bowel diverticula are symptomatic.[5] These include abdominal discomfort, flatulence, borborygmi, malabsorption, pseudo-obstruction, stasis and 'blind loop' syndrome. In 10% of patients surgical intervention is necessary. Reasons include intestinal obstruction, haemorrhage and perforation. Neoplastic growth may also occur and include fibroma, lipoma, carcinoma and sarcoma formation.[2]

Intestinal obstruction may arise from enterolith formation, intussusception or volvulus.[1 6 7] In the latter situation the diverticulum acts as a pivot, especially where previous diverticulitis results in adhesive band formation. Such adhesions may also cause obstruction by direct kinking of the bowel or by trapping another loop of bowel underneath.

Final diagnosis

Mechanical small bowel obstruction secondary to an enterolith arising from jejunal diverticulum.

Keywords: jejunal diverticulum; enterolith; bowel obstruction

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Adverse drug reaction

Disseminated intravascular coagulation and vasculitis during propylthiouracil therapy

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The use of antithyroid drugs in the early 1940s revolutionised the management of hyperthyroidism. Since their introduction, a variety of adverse reactions, including haematological, dermatological, and rheumatological effects, have been associated with these drugs. The incidence of these side-effects is similar to many other commonly used drugs, ie, 1–5%.[1] The most common side-effects include skin rash, fever, arthralgias/arthritis and neutropenia while lupus-like reaction, vasculitis, hepatitis, agranulocytosis and thrombocytopenia are uncommon.[2] Disseminated intravascular coagulation (DIC) is a rare adverse effect of propylthiouracil therapy.[3] Herein we report a case of DIC and vasculitis following a short course of propylthiouracil therapy.

Case report

A 42-year-old African-American woman with Grave's disease, diagnosed 20 years earlier, had received propylthiouracil (100 mg tid) for the past 2 weeks because of recent exacerbation of symptoms. She was admitted to the hospital because of sudden onset of palpable purpuric rash, which started on the face and later spread to her trunk and extremities.

Laboratory test results on admission disclosed the following data: haemoglobin 12.2 g/dl, haematocrit 37.8, white blood cells 15.5×10^9/l, platelets 49×10^9/l, erythrocyte sedimentation rate 23 mm/h, free thyroxine 3.7 ng/dl (normal 0.71–1.85); and thyroid-stimulating hormone <0.03 mIU/ml. Tests for erythrocytes were normal. Tests for coagulation studies revealed prothrombin time 14.3 s (10.6–13.4); activated partial prothrombin time 28.0 s (18–38); D-dimer test positive; fibrinogen degradation products positive; fibrinogen level 344 mg/dl (152–392). Serum complement studies showed C_3 133 mg/dl (88–200), C_4 12 mg/dl (16–47) and CH_{50} 126 U/ml (100–300 CH_{50}). Serum haptoglobin level was normal.

Urinalysis showed trace protein and moderate blood in the urine. Antinuclear, anti-DNA single stranded, anti-DNA double stranded, antihistone, antithyroglobulin and antineutrophil cytoplasmic antibody (ANCA) titres were normal. Blood cultures were negative and chest X-ray was normal. Bone marrow aspiration showed myelosuppression with no evidence of leukaemia. Skin biopsy showed acute vasculitis involving small and medium-sized vessels with fibrin thrombi. No immunohistochemical testing was done. The skin of the left cheek revealed focal superficial epidermal and dermal haemorrhagic necrosis with marked acute inflammation and pustule formation.

The patient was admitted to the hospital and treated with intravenous methylprednisolone 125 mg every 8 hours. Propylthiouracil was discontinued. She responded to intravenous methylprednisolone and the purpuric rash gradually disappeared. Subsequently steroids were tapered over next 2 weeks. The haematological abnormalities returned to normal.

Discussion

The most frequent adverse effects related to propylthiouracil and methimazole, the two most commonly used thionamides, are haematological. Transient leucopenia, perhaps the most common side-effect, has been reported in 12% of adults and up to 25% of children,[4] while cutaneous adverse reactions occur in 3–5% of adults and up to 18% of children.[4 5] Generalised maculopapular and papular purpuric eruptions are perhaps the most common thionamide-induced cutaneous reactions, but rarely bullous haemorrhagic, generalised vesicular and necrotic ulcerative forms have been described.[5] Propylthiouracil induces a clinically distinctive cutaneous eruption consisting of symmetrical, tender, palpable purpuric lesions, often in a livedoid pattern and curiously involves the ear lobes and malar areas.[5–9] Cutaneous vasculitis is usually seen early in the course of propylthiouracil therapy, but has also been observed after long-term treatment. Its exact incidence is not known. Vasculitic involvement of skin is far more common than other organs. Cases of nephritis, myositis, and cavitary pulmonary infiltrates have been reported.[6 7]

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