PICTORIAL MEDICINE

Abdominal cocoon: uncommon cause of intestinal obstruction in peritoneal dialysis patient

A 47-year-old woman presented with abdominal pain and no bowel opening for 2 days in March 2011. Having been on continuous ambulatory peritoneal dialysis (CAPD) for 10 years, she had been switched to haemodialysis for 4 years because of recurrent peritonitis. Abdominal X-ray showed small bowel obstruction and curvilinear peritoneal calcification (Fig 1). Computed tomography (CT) of the abdomen showed thickened calcified peritoneum encapsulating loops of small bowel. These features were compatible with encapsulating peritoneal sclerosis. Absence of mural enhancement within the small bowel suggested ischaemia (Fig 2). Exploratory laparotomy revealed multiple adhesions encapsulating loops of small bowel, with resultant closed loop obstruction. Segments of small bowel were gangrenous and perforated, and there was faecal contamination of the retroperitoneum. Peritoneal biopsy showed fibrous tissue with hyalinisation, necrosis, and dystrophic calcification (Fig 3). Staining for acid-fast bacilli intra-operatively was negative. Adhesiolysis and resection of non-viable small bowel was performed, but 1 day later the patient succumbed.

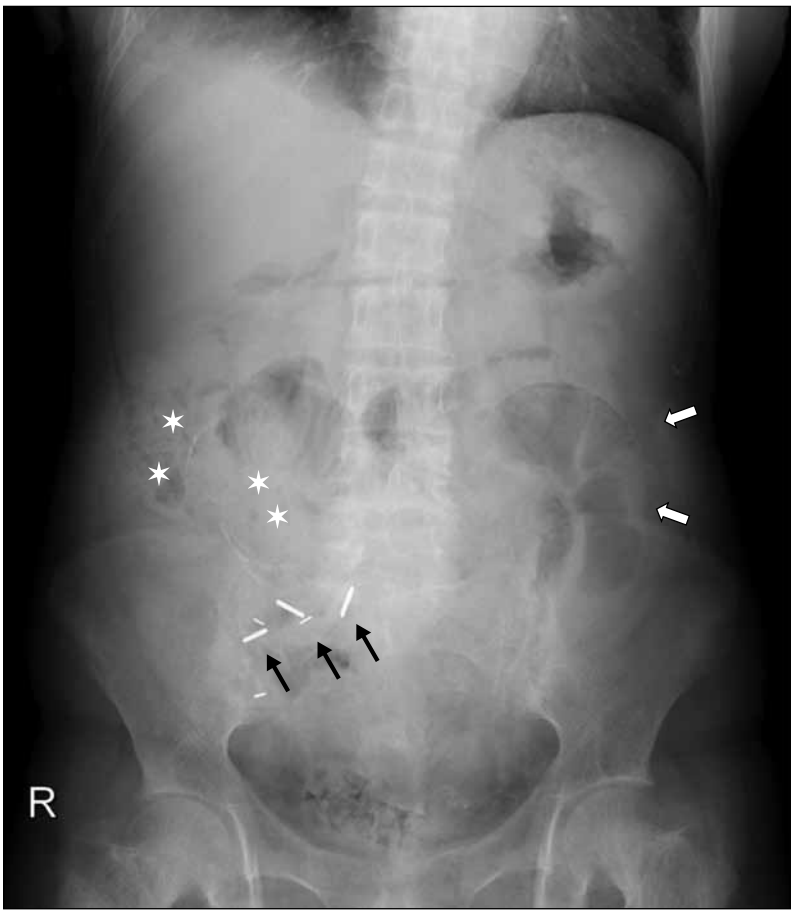

FIG 1. An abdominal X-ray showing dilated small bowel and peritoneal calcification
White stars indicate peritoneal calcifications; white arrows indicate dilated small bowel loops; black arrows indicate metal clips placed during a previous renal transplant

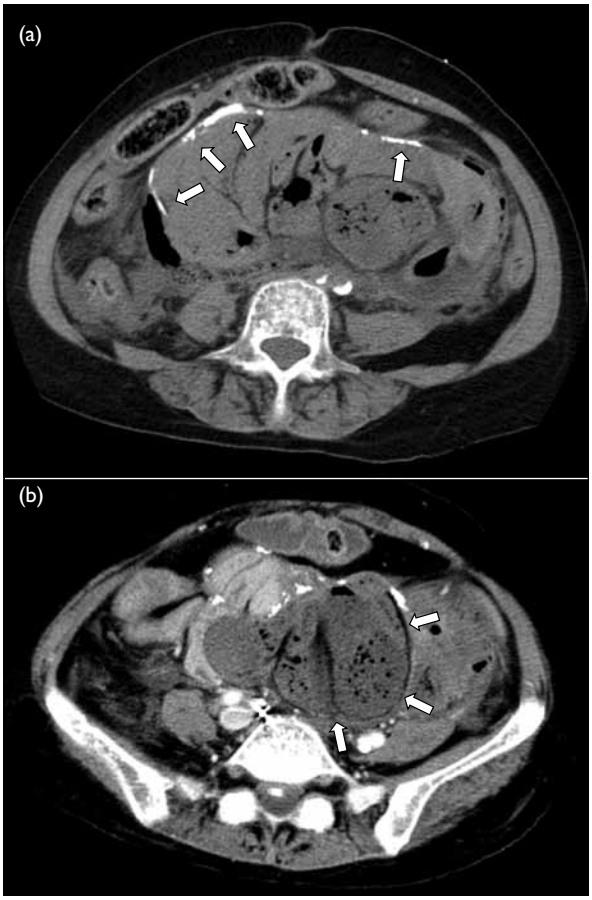

FIG 2. Computed tomography scans of the abdomen in (a) pre-intravenous and (b) post-intravenous contrast phases
(a) White arrows indicate peritoneal thickening with calcification encapsulating the dilated small bowels loops. (b) White arrows indicate absence of mural enhancement of the cocooned small bowels

Encapsulating peritoneal sclerosis is a rare, devastating complication of peritoneal dialysis, believed to result from chronic intra-abdominal inflammation but may be multifactorial. It affects less than 2% of such patients; its frequency increases with increasing duration of CAPD.[1,2] Presenting clinical symptoms are often insidious and non-specific,

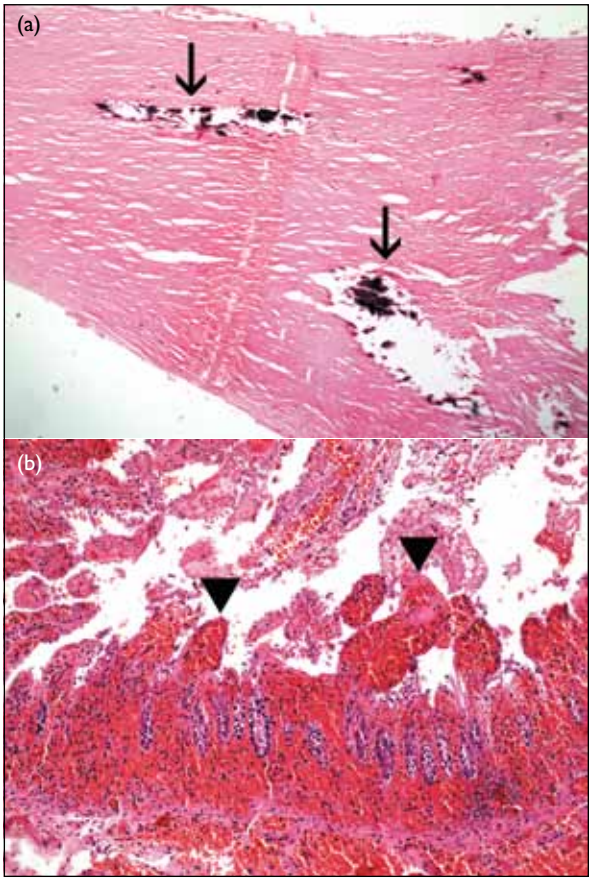

FIG 3. (a) The peritoneal lining is markedly thickened with fibrosis with foci of dystrophic calcification (arrows) [H&E]. (b) The small intestine shows ischaemic necrosis and sloughing of necrotic villi (arrowhead) [H&E]

and the diagnosis is usually made radiologically or surgically. Abdominal radiography (usually the first imaging investigation) may yield peritoneal calcifications. Diffuse peritoneal calcification is evident in advanced disease; occasionally encapsulating peritoneal sclerosis may ensue (even in the absence of peritoneal calcification). Contrast-enhanced CT allows better evaluation of peritoneal thickening, peritoneal calcification, and encapsulation of involved small bowel loops.[3] Important differential diagnoses include tuberculosis peritonitis (especially in this locality) and mucinous tumour metastasis. Curative surgical excision of thickened peritoneum and adhesiolysis may be curative; immunosuppression and other conservative measures (cessation of peritoneal dialysis and bowel rest) are less effective. In these patients, early radiological imaging facilitates timely diagnosis and intervention to deal with encapsulating peritoneal sclerosis.[4]

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