IMAGING TEACHING CASE

Development of Encapsulating Peritoneal Sclerosis Following Bacterial Peritonitis in a Peritoneal Dialysis Patient

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INTRODUCTION

Encapsulating peritoneal sclerosis (EPS) is an uncommon and often fatal complication in patients on peritoneal dialysis (PD) therapy. EPS is a clinical syndrome characterized by symptoms of impaired intestinal motility, such as anorexia, nausea, vomiting, abdominal fullness, abdominal pain, absent bowel sounds, and constipation.[1] These presentations result from diffuse peritoneal thickening, sclerosis, calcifications, and encapsulation of the bowel loops.[1] Progression of EPS usually is considered to be insidious; however, we describe a patient who developed EPS immediately after an episode of bacterial peritonitis. The diagnosis of EPS requires a high index of clinical suspicion, especially in long-term PD patients with symptoms of ileus. Imaging studies can be very important in confirming the diagnosis.

CASE REPORT

Clinical History and Initial Laboratory Data

A 40-year-old woman with end-stage renal disease caused by chronic glomerulonephritis, who had received PD for 12 years, presented to the emergency department with fever, nausea, diarrhea, and diffuse abdominal pain after ingesting seafood in October 2008. She had controlled hypertension and had undergone parathyroidectomy for secondary hyperparathyroidism in January 2007.

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The patient's PD regimen included 5 exchanges of 2 L of PD solution daily. Before July 2007, all exchanges were performed using 2.5% dextrose solution (Dianeal; Baxter Healthcare CA, Singapore Branch, www.baxter.com), with occasional use of 4.25% dextrose solution. Since July 2007, one exchange of Dianeal has been replaced by 7.5% icodextrin (Extraneal; Baxter Healthcare CA, Singapore Branch). Her peritoneal equilibrium test result was low average in the first 4 years, became high average after 2001, and finally became high in the last 2 years. β-Blockers, including atenolol, metoprolol, and carvedilol, had been used for blood pressure control. There was no PD-related peritonitis during the 12 years before this episode.

On admission, physical examination showed an acutely ill–looking woman with a temperature of 38°C, distended and tympanic abdomen, hypoactive bowel sounds, and diffuse abdominal tenderness and rebound. The rest of the examination findings were normal. A peritoneal effluent study showed an increased white blood cell count of 450 cells/μL, with 45% neutrophils. Intraperitoneal cefazolin with ceftazidine was used empirically, then was changed to ceftriaxone when the PD effluent grew *Salmonella* group D1. Leukocytosis, nausea, and vomiting persisted after this treatment; therefore, the PD catheter was removed, and the patient was shifted to hemodialysis therapy on hospital day 13. Because of her continuing profound vomiting and a 6-kg weight loss, total parenteral nutrition was started, and a nasogastric tube was required to drain 2,000-3,000 mL of digestive fluid daily.

Imaging Studies

The kidney, ureter, and bladder radiograph obtained on admission showed extensive peritoneal curvilinear "eggshell"–like calcification, whereas calcification in the pelvis was more conglomerate. Bowel loops were dilated and separated (Fig 1).

Abdominal computed tomography on hospital day 5 showed dilated bowel loops with thickened walls, diffuse parietal and visceral peritoneal calcification, peritoneal thickening and enhancement, and encasement of the dilated bowel loops by the calcified peritoneum (Fig 2; Movies S1 and S2, provided as online supplementary material available with this article at www.ajkd.org).

Abdominal sonography then showed echogenic strands on visceral membrane, representing peritoneal or bowel wall calcification (Fig 3). Membrane formation anterior to the bowel wall results in a characteristic trilaminar appearance (Fig 3). The small-bowel series showed separated fixed bowel loops (Fig 4). The bowel wall and peritoneal thickening can be identified by the distance between the contrast

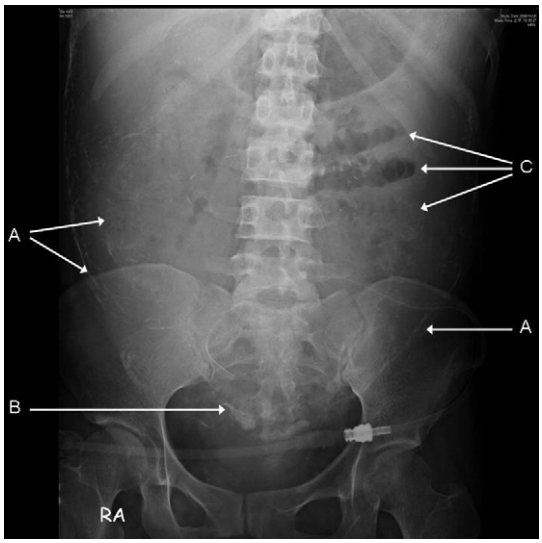

Figure 1. Kidney, ureter, and bladder film shows diffuse (A) curvilinear and (B) conglomerate peritoneal calcification. (C) At the left upper quadrant, separated and dilated bowel loops can be seen.

media and calcified peritoneum. Stagnation of the contrast medium was still noted after 30 hours.

Diagnosis

Acute EPS after bacterial peritonitis.

Clinical Follow-up

Tamoxifen, 20 mg, and methylprednisolone, 20 mg, were administered daily. Bowel motility improved after 7 days of treatment, and the patient could begin oral intake. The patient was discharged 2 weeks later with daily oral 20 mg of tamoxifen and 30 mg of methylprednisolone. She continued on hemodialysis therapy and had no symptoms of bowel obstruction after 6 months of follow-up.

DISCUSSION

EPS is an uncommon, but catastrophic, complication of long-term PD therapy in which the peritoneum becomes progressively thickened, causing encasement of the small intestine. Its high mortality rate is related to complications from bowel obstruction (such as malnutrition, sepsis, and bowel perforation) and complications after surgical enterolysis. The incidence of EPS reported in the literature ranges from 0.7%-7.3%.[1] Recent studies described the incidence to be 2.5% in Japan[2] and 3.3% in a single-center study in the United Kingdom.[3] The duration of PD treatment correlates with the risk and mortality of EPS.[1] However, EPS had been observed even after discontinuation of PD therapy or kidney transplant in PD patients.[1,4]

Clinical presentations of EPS are related to impaired small-intestinal motility and peritoneal inflammation and include abdominal pain, abdominal mass, nausea, vomiting, anorexia, malnutrition, weight loss, low-grade fever, hemorrhagic effluent, and recurring or nonresolving peritonitis. Another common clinical finding in

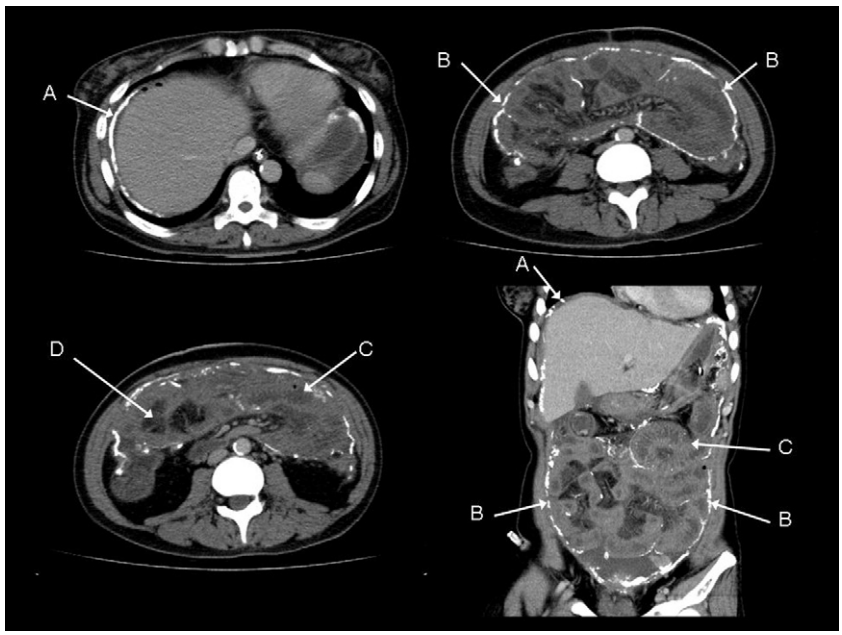

Figure 2. Abdominal computed tomography shows extensive (A) parietal and (B) visceral peritoneal calcification, with the small bowels encased by the calcified peritoneum (cocooning). Also evident were (C) the dilated bowel loops with thickened walls and (D) increased density of mesenteric fat.