



Diffuse peritoneal calcification

To the Editor,

A 45-year-old uremic female on continuous ambulatory peritoneal dialysis (CAPD) for the past 9 years and on current hemodialysis for 1 year presented with intermittent nausea, vomiting, diffuse abdominal fullness, and body weight loss of 9 kilograms in the past 6 months. She had anemia (hemoglobin 7.8 g/dL), increased C-reactive protein (2.8 mg/dL), and malnutrition. Immune study and tumor markers were un-revealing. The abdominal plain film showed linear calcification at the contour of the abdominal cavity (Figure 1). Computed tomography (CT) of the abdomen demonstrated peritoneal calcification, bowel wall thickening, and small intestinal obstruction with dilated loops (Figure 2, 3). Her clinical symptoms were refractory to anti-fibrotic agent (tamoxifen) and steroid for 6 months but resolved after exploratory laparotomy with enterolysis. Histologic examination of the peritoneal membrane showed typical peritoneal sclerosis with proliferation of fibro-connective tissue and inflammatory infiltrates.

Encapsulating peritoneal sclerosis (EPS), characterized by progressive peritoneal membrane inflammation and fibrosis with resultant intestinal encapsulation, is a catastrophic complication with exceedingly high morbidity and mortality in uremic patients on long-term CAPD. The diagnosis of EPS needs both clinical symptoms and typical radiological findings. Clinical symptoms include non-sterile bloody dialysate, progressive ultrafiltration failure, and gastrointestinal symptoms, such as vomiting, abdominal pain, intermittent bowel obstruction, weight loss, and malnutrition (1). Characteristic radiological features on abdominal CT consist of peritoneal membrane thickening and calcification, bowel thickening, bowel tethering, and bowel dilatation (2). Longer duration of exposure to PD fluid and repeated peritonitis are the most important risk factors.

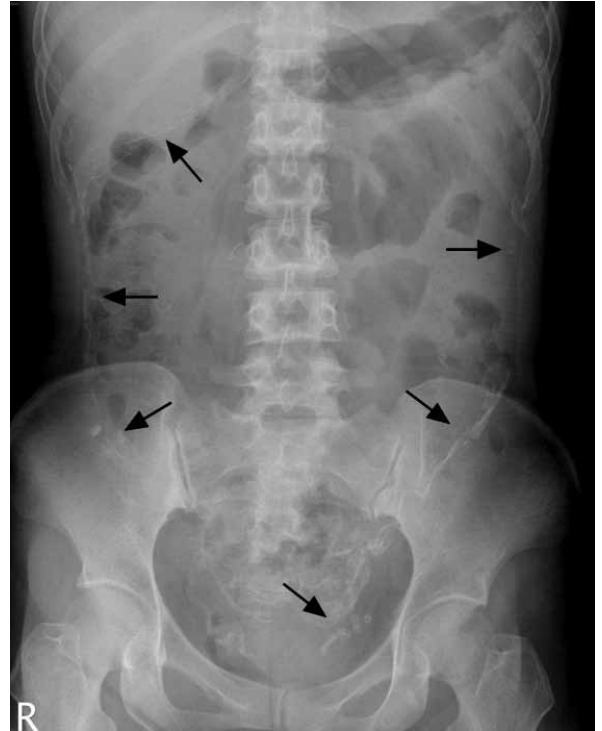


Figure 1. Extensive linear calcification of the peritoneum (black arrows).

The pathophysiology of EPS remains unclear and may be explained by a two-hit hypothesis. The first hit is peritoneal membrane damage of both function and structure by PD fluid, and the second hit is repeated peritonitis, leading to an uncontrolled fibrosing process with consequent encapsulation of the intestines. EPS can still evolve, even with withdrawal of PD, and is increasingly reported in patients on hemodialysis and receiving successful renal transplantation who have received previous PD (3). The medical management of EPS needs early cessation of PD to prevent further peritoneal damage and the use of corticosteroid and tamoxifen or in combination to reduce fibrosis. Surgical

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Figure 2. Diffuse peritoneal calcification with intestinal wall thickening and small intestinal obstruction with dilated loops (white arrow).

intervention, such as stripping the encapsulating membrane and enterolysis, is reserved for patients who have failed medical treatment or present with recurrent intestinal obstruction. Due to its vague and non-specific symptoms at an early stage, a higher index of suspicion of EPS is required for prompt recognition with appropriate management.

Ethics Committee Approval: Ethics committee approval was received for this case.

Informed Consent: Written informed consent was obtained from patient who participated in this case.



Figure 3. Dilated small intestine with air fluid level in the lumen and visceral calcification of bowel surface (arrow heads).

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