

# **Abdominal Cocoon: A Case of Sclerosing Encapsulating Peritonitis**

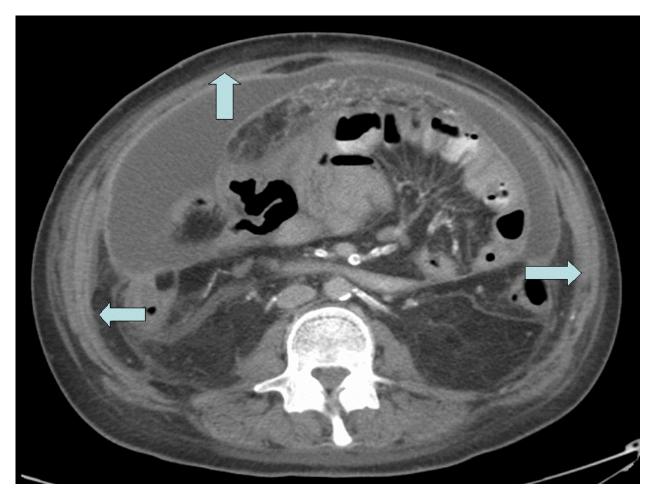


FIGURE. CT image of the abdomen shows dilated small bowel loops surrounded by thickened fibrous peritoneum.

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## INTRODUCTION

Sclerosing peritonitis encapsulatum (SPE) is also known as encapsulating peritoneal sclerosis or abdominal cocoon. SPE is a rare, but lethal, complication of continuous ambulatory peritoneal dialysis (CAPD) and represents an advanced stage of peritoneal sclerosis.

# **CASE**

A 43-year-old male with Type 1 diabetes and end-stage renal disease (ESRD) was admitted with persistent vomiting and diffuse abdominal pain for 1 week. Prior to this admission, he had been hospitalized three times in a span of 2 months due to recurrent peritonitis. He had been on CAPD for 8 years with a recent change to hemodialysis (HD) 2 weeks prior. Examination revealed soft abdomen with diminished bowel sounds, diffuse tenderness, and no rigidity or guarding. Labs revealed a white cell count of 13,000/µl, hemoglobin 10.7 g/dl, blood urea nitrogen 40 mg/dl, creatinine 5.3 mg/dl, AST 61 IU/l, ALT 44 IU/l, alkaline phosphatase of 1336 IU/l, and normal total bilirubin. The computerized tomography (CT) scan of the abdomen showed presence of ascites, peritoneal thickening, cholelithiasis, and possible cholecystitis. Ascitic fluid analysis and cultures were negative for infection. The patient underwent open cholecystectomy, but there was no improvement in the severity of abdominal pain. A repeat CT scan 1 week later showed reaccumulation of ascites, omental thickening, and peritoneal enhancement findings consistent with SPE. The patient was started on total parenteral nutrition (TPN), but failed to improve and died in the hospital 2 weeks later.

# **DISCUSSION**

Etiology of SPE is unknown, but the pathogenesis due to persistent exposure of mesothelial cells to transforming growth factor  $\beta$  and proliferation of peritoneal fibroblasts is widely accepted. Predisposing factors include recurrent peritonitis and chronic peritoneal irritation due to dialysate. The annual incidence of SPE in CAPD patients is 0.37 per 1000 patient years, with a male predominance (5:2), and the median duration on CAPD is 62 months. Presentation is generally nonspecific with abdominal pain, distension, nausea, and vomiting. Patients often experience a reduced peritoneal clearance or impaired peritoneal ultrafiltration. Classic CT scan findings include thickened adhered bowel loops, loculated ascites, and peritoneal enhancement. The definitive diagnosis is by laparotomy and biopsy. Initial management involves the removal of dialysis catheter, discontinuation of CAPD, initiation of TPN, and bowel rest. The use of steroids, immunosuppressives, and tamoxifen has been reported to be beneficial, but needs to be validated. Surgery is high risk for fistula formation and anastomotic leakage. Despite various therapeutic modalities, the mortality rate is 56–93%.

### CONCLUSION

Patients who are and were on CAPD presenting with symptoms and signs of bowel obstruction and otherwise unexplained abdominal pain should be evaluated for SPE.

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