Sclerosing encapsulating peritonitis with perforation of the gastrointestinal tract: a serious complication of continuous peritoneal dialysis

To the Editor: Sclerosing encapsulating peritonitis (SEP) is one of the most serious complications of peritoneal dialysis. It is characterized by partial or intermittent bowel obstruction and survives subsequent high morbidity and mortality.[1] A 36-year-old female patient was admitted to the nephrology department with a 24-hour history of abdominal pain and intermittent vomiting. The patient had had chronic renal failure with right renal agenesis for 13 years. SEP was established 8 months previously, and peritoneal dialysis, performed for a long period, was stopped. The general surgery department was consulted. On physical examination, a tender lump was palpated in the umblical region of the abdomen with mild muscle guarding and rebound tenderness. Plain abdominal x-ray showed dilated loops of small bowel with air-fluid levels and free gas under the diaphragm. Abdominal ultrasonography revealed dilated bowel loops, especially in the center of the abdomen. There were localized fluid collections in whole recesses. At laparotomy, 400 mL of purulent fluid was aspirated. A fibrous capsule covering all abdominal viscera was revealed (**Figures 1 and 2**). The liver, stomach, appendix, small bowel loops, as well as the whole colon were also covered with this fibrous capsule, which appeared like a cocoon. Dissection of adhesions from other was impossible. Although we tried to find the perforation, we could not find it because of extensive adhesions and the fragility of the tissues. Histology of the peritoneal membrane showed thickened fibrocollagenous tissue with chronic non-specific inflammation. An enterocutaneous fistula developed on the fifth postoperative day. The patient died from sepsis 3 weeks after the operation.

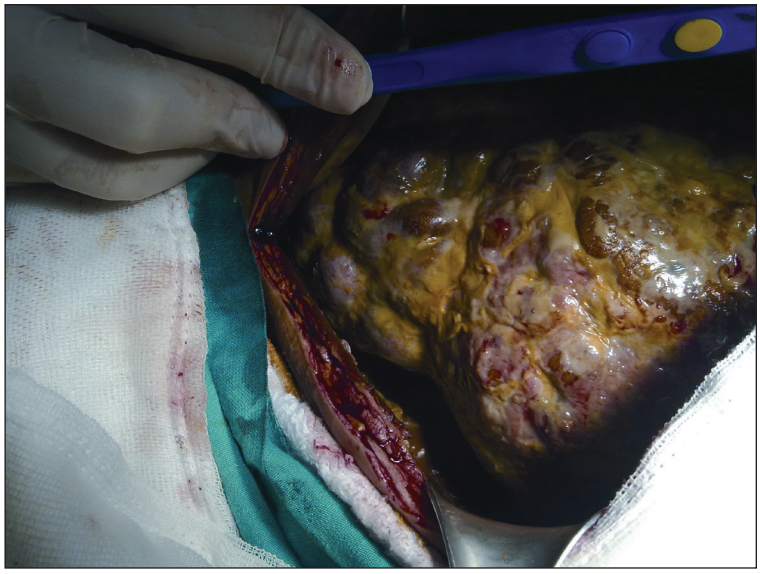

Figure 1. The fibrous capsule covering all abdominal viscera.

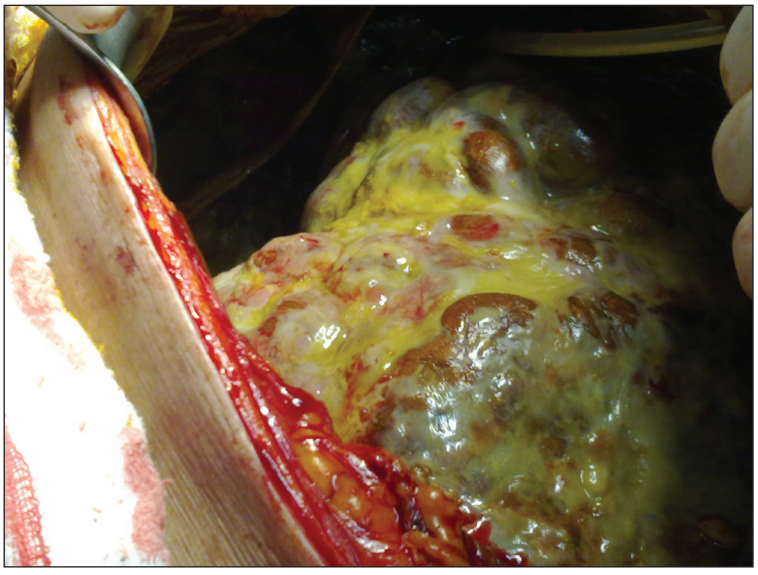

Figure 2. The fibrous capsule covering all abdominal viscera.

SEP is rare. The idiopathic form (also known as abdominal cocoon) was first described by Foo et al in 1978.[2,3] It is characterized by a thick grayish-white fibrotic membrane, partially or totally encasing the small bowel.[4] The fibrocollagenic cocoon sometimes extends to involve other organs such as the colon, liver and stomach. Clinically, it presents with recurrent episodes of acute, subacute or chronic small bowel obstruction, weight loss, nausea and anorexia, and sometimes with a palpable abdominal

mass, but some patients may be asymptomatic.[5] SEP can be classified as idiopathic or secondary. The secondary form of SEP has been reported to occur in association with peritoneal dialysis. Other rare causes include use of beta-blockers, abdominal tuberculosis, ventriculoperitoneal and peritoneovenous shunts, liver transplantation, systemic lupus erythematosus, cirrhosis of the liver, carcinoid syndrome, familial Mediterranean fever, asbestos exposure and recurrent peritonitis. The initial symptoms directly depend on disorders of the gastrointestinal tract. Despite the distinctive clinical findings strongly supporting a diagnosis of SEP, a radiologic examination is required to establish the clinical diagnosis of SEP. Laparotomy or laparoscopy is required to confirm the diagnosis. The key points in conservative treatment are early diagnosis, cessation of peritoneal dialysis with transfer to hemodialysis, sustained bowel rest with total parenteral nutrition, and corticosteroids. In the literature, there is no consensus for the treatment of SEP.[1,5]

It is very important to establish diagnosis before development of life-threatening complications of SEP such as intestinal obstruction and perforation because of high morbidity and mortality rates. Prevention and early diagnosis become even more important.

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REFERENCES

1. Nakamoto H. Encapsulating peritoneal sclerosis-A clinician's approach to diagnosis and medical treatment. Perit Dial Int 2005; 25:S30-38.
2. Foo KT, Ng KC, Rauff A, Foong WC, Sinniah R. Unusual small intestinal obstruction in adolescent girls: the abdominal cocoon. Br J Surg 1978; 65:427-30.
3. Kawaguchi Y, Kawanishi H, Mujais S, Topley N, Oreopoulos DG. Encapsulating peritoneal sclerosis: definition, etiology, diagnosis, and treatment. Perit Dial Int 2000; 20:S43-55.
4. Sahoo SP, Gangopadhyay AN, Gupta DK, Gopal SC, Sharma SP, Dash RN. Abdominal cocoon in children: a report of four cases. J Pediatr Surg 1996; 31:987-8.
5. Xu P, Chen LH, Li YM. Idiopathic sclerosing encapsulating peritonitis (or abdominal cocoon): a report of 5 cases. World J Gastroenterol 2007; 13:3649-51.

Capecitabine-induced terminal ileitis

To the Editor: Capecitabine (Xeloda, Roche) is an oral fluoropyrimidine prodrug which is a valuable substitute for parenteral 5-flourouracil (5-FU) for the treatment of colon cancer and metastatic breast cancer.[1] In comparison with intravenous 5-FU, capecitabine was associated with less frequent diarrhea (47.7% vs 58.2%), neutropenia and stomatitis.[2] These features have contributed to the increased use of capecitabine. There are two published case reports describing small bowel toxicity associated with capecitabine.[3,4] We report a patient with terminal ileitis secondary to the use of this drug. This is the first reported case which was reversible, isolated and proven by endoscopy and histology.

The patient was a 65-year-old Saudi male diagnosed with locally advanced and metastatic adenocarcinoma of the rectum. He received pelvic radiation therapy of 30 Gy given in 10 fractions over 2 weeks to control his pelvic disease. He was subsequently started on XELOX (capecitabine plus oxaliplatin) chemotherapy 4 weeks after finishing the radiation therapy. The dose of capecitabine was 1500 mg orally twice daily for 14 days every 21 days.[5] This was equivalent to almost 2/3 of the standard dose. Twelve days after the start of this chemotherapy, he was admitted with fever, abdominal pain, vomiting and diarrhea. Initially, he was treated with broad spectrum IV antibiotics and fluids. Capecitabine was discontinued. Stool for *Closteridium difficile*, parasites and culture were all negative. His course was prolonged and colonoscopy showed an isolated ulceration in the terminal ileum (**Figure 1**). The histopathology was consistent with inflammatory changes and eosinophilic infiltrate but no evidence of malignancy or granulomas (**Figure 2**). His condition improved with conservative treatment. The dihydropyrimidine dehydrogenase (DPD) mutation test was negative. A repeat colonoscopy 2 months later did not show any residual abnormality. He subsequently received a second cycle of XELOX chemotherapy 5 weeks after this first cycle, but the dose of capecitabine reduced to 1000 mg twice daily. He tolerated this well with no recurrence of gastrointestinal toxicity. He continued to respond and tolerated the treatment well.

Most patients tolerate capecitabine well. However, a number of patients develop severe and sometimes life-threatening toxicity after standard doses of capecitabine (which is 1250 mg/m^2 orally

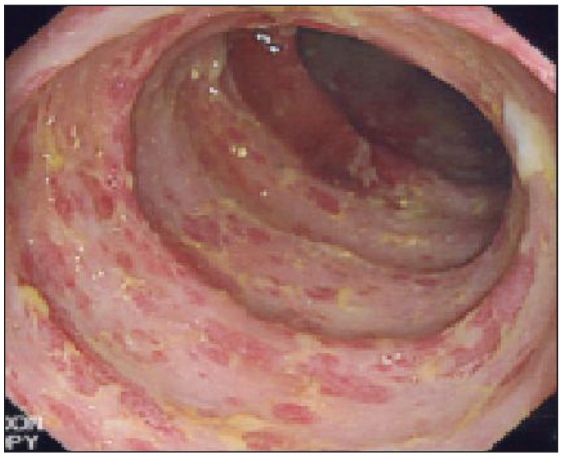

Figure 1. Colonoscopy showed isolated terminal ulceration.

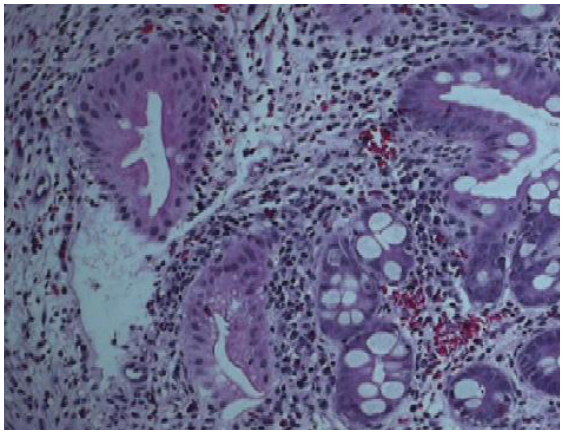

Figure 2. The histopathology was consistent with inflammatory changes and eosinophilic infilterate but no evidence of malignancy, granulomas or CMV (cytomegalovirus).

twice daily for 14 days every 21 days).[5] The major gastrointestinal manifestation of 5-FU cytotoxicity is diarrhea, which may be severe as well as dose-dependent and schedule dependant. However, in the patient described in this report, the colon appeared to be normal with toxicity limited to the the terminal ileum. This toxicity appeared to be dose dependant as there was resolution of the initial complaints and the patient was able to tolerate the treatment without suffering with an appropriate dose reduction. Radiation-induced ileitis was believed less likely to be the cause as the field of radiation exposure was away from the terminal ileum. In addition, there was a gap of 6 weeks between the end of radiation and the onset of the symptoms and a relatively low dose of radiation was utilized (30 Gy over 2 weeks). Finally the symptoms correlated with the dose of capecitabine.

There are very limited reports describing ileitis caused by capecitabine. We found two cases reported separately.[3,4] The first was of a patient receiving single agent capecitabine in the adjuvant setting. Similar to our patient, ileitis was confirmed by endoscopy and histologically. The second patient received XELOX and bevacizumab in the metastatic setting. Terminal ileitis was diagnosed clinically and radiologically without endoscopy examination. To our knowledge, our report describes for the first time a reversible capecitabine-induced terminal ileitis that was confirmed endoscopically and histologically. This complication was successfully prevented from recurrence by reduction of the capecitabine dose in the subsequent cycles. Physicians should be aware of this side effect, which could be life threatening but appears to be dose dependant.

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REFERENCES

1. Miwa M, Ura M, Nishada M, Sawada N, Ishikawa T, Mori K, Shimma N. Design of a novel oral fluoropyrimidine, capecitabine, which generates 5-fluorouracil selectively in tumors by enzymes concentrated in human liver and cancer tissue. Eur J cancer. 1998 Jul ;34(8): 1274-81.
2. Cassidy J, Twelves C, Van Cutsem E. First-line oral capecitabine therapy in metastatic colorectal cancer: A favorable safety profile compared with intravenous 5-fluorouracil/leucovorin. Ann Oncol 13:566-575, 2002.
3. Debora Barton ,Brazil, Marcelo Cruz, Brazil, and Vladimir Schraibman. Ulcerative Ileitis Secondary to Adjuvant Capecitabine for Colon Cancer: A Case Report. (UICC world Cancer Congress 2006) –abstract.
4. Bouma G, Imholz A. Ileitis following capecitabine use.NedTijdschr Source :DeventerZiekenhuis, afd. InterneGeneeskunde, Deventer, the Netherlands. Geneeskd. 2011;155:A3064.
5. Hyodo I, Shirao K, Doi T, Hatake K, Arai Y, Yamaguchi K,Tamura T. A phase II Study of the global dose and schedule of capecitabine in Japanese patients with metastatic colorectal cancer. Jpn J ClinOncol 2006; 36(7):410-7.