Case Report

Sclerosing encapsulating peritonitis associated with recurrent eosinophilic peritonitis

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Introduction

Sclerosing encapsulating peritonitis (SEP) is a rare but serious, sometimes fatal complication of continuous ambulatory peritoneal dialysis (CAPD) [1–6]. SEP is characterized by the marked sclerotic thickening of the peritoneal membrane and symptoms such as abdominal pain, nausea and vomiting, impaired intestinal motility including ileus.

On the other hand, eosinophilic peritonitis is observed in a notable proportion of patients treated with peritoneal dialysis and is generally thought to be a benign phenomenon which spontaneously heals without impairing peritoneal function in all patients [7–9]. We report a patient with eosinophilic peritonitis followed by SEP.

Case

A 52-year-old woman developed end-stage renal disease caused by chronic nephritis in May 1994, and was started on CAPD immediately after Tenckhoff peritoneal catheter insertion with four times exchanges of 1 1.5% peritoneal dialysate. Without any clinical symptoms, a cloudy effluent was noticed 8 days after commencing CAPD. Dialysate WBC count was 760/µl with 22% eosinophils. Serum IgE elevation and peripheral eosinophilia were not recognized. Cultures of effluent showed no growth of bacteria or fungus. No parasites nor their ova could be found in her stool samples. The diagnosis of eosinophilic peritonitis was made and this condition resolved after 1 week without any therapy. Since then, she had similar documented episodes of eosinophilic peritonitis at least three times. The duration of the cloudy effluent was less than 2 weeks. However, in April 1996, her eosinophilic peritonitis persisted for at least 1 month with impaired peritoneal function. Although the cloudy effluent disappeared, her peritoneal dysfunction did not recover. Peritoneal equilibration test (PET) [10] showed D/P 0.973, D/D₀ 0.171 which represents a high transporter status of peritoneum. In order to clarify the histological alterations of the peritoneum and decide about continuation of CAPD, a parietal peritoneum biopsy was done. Its histological findings showed the absence of mesothelial cells and an infiltrated surface with fibrin. The stoma consisted of proliferative fibroconnective tissue containing a predominantly mononuclear cellular infiltrate with occasional eosinophilic cells (Figure 1A). From these results, we concluded that continuing CAPD would be difficult and switched to haemodialysis in June 1996.

By August 1997, she complained of occasional nausea and abdominal distension. After that, her symptoms became worse with repeated vomiting. She was admitted in November. Ultrasound examination showed adhesion of bowels to the peritoneum and ascites. A barium meal showed gross dilatation of the stomach and duodenal loop from severe narrowing distal to the duodeno-jejunal flexure (Figure 2). A barium enema revealed no significant abnormality. In order to make a definite diagnosis, she underwent laparotomy. Most of her small intestines were encased in a white thick and fibrous peritoneal capsule (Figure 3). Along with nasogastric suction and total parenteral nutrition, she had steroid pulse therapy (methylprednisolone 1000 mg i.v.). After a course of treatment lasting 3 days, this was changed to 20 mg i.v. prednisolone. Three weeks after the laparotomy, a gastrografin meal indicated improving passage of contrast with no obstruction. After that, oral intake with fluid was started and she currently remains free of symptoms on a semi solid diet.

Discussion

SEP is the most serious complication of CAPD. In a report by the Japanese SCP study group [6], the
SEP associated with recurrent eosinophilic peritonitis

Fig. 1. Photomicrograph of a biopsy specimen of parietal peritoneum. (A) Mesothelial cells are absent. Attachment of fibrin, proliferation of fibroconnective tissue, and infiltrated cells at the perivascular area are seen. PAS stain × 200. (B) Infiltrated cells consist predominantly of mononuclear cells and eosinophilic cells (arrow). PAS stain × 400.

The mortality rate was up to 43.5%. The main causes are the problems related to bowel obstruction or complications from surgery. Concerning the diagnosis of SEP, the radiological features may be valuable [11,12], but the definite diagnosis is confirmed by laparotomy, which reveals the characteristic gross thickening of the peritoneum which encloses some or all of the small intestines in a cocoon of opaque tissue. In our patient, although SEP was strongly suspected, we considered that stenosis of the small intestines could have been solely induced by adhesion due to recurrent eosinophilic peritonitis. In order to make a definite diagnosis and relieve the mechanical bowel obstruction by surgical lysis of adhesion, laparotomy was done. As her condition was diagnosed definitely as SEP, a surgical procedure was avoided. Because it is well known that successful release is almost impossible and the mortality rate associated with surgical complications is very high [3,4,6], parenteral hyperalimentation and corticosteroid pulse therapy were selected [3,13].

Although the aetiology of SEP is unclear, some predisposing factors were implicated. These included peritonitis, acetate dialysates, chronic use of certain beta blockers, antiseptics used during bag exchanges [1–3,5,6]. When these factors are responsible, the most likely mechanism appears to be recurrent episodes of peritonitis. However, this condition is generally associated with a genuine bacterial or fungal infection. Her treatment was exclusively with lactate-buffer dialysate. She had no history of infection induced peritonitis and had never received any beta blockers.

As far as we know, there has been no report of SEP occurring after eosinophilic peritonitis. Eosinophilic peritonitis is usually defined as when there are more than 100 eosinophils present per millilitre of peritoneal effluent, of which eosinophils constitute more than 10% of its total WBC count [9]. The mechanism of eosinophilic peritonitis remains obscure, although a hypersensitizing reaction to some allergens related to CAPD system is thought to
Fig. 3. Macroscopic findings at laparotomy show the small bowel encased by a white thick peritoneum with adhesion (arrow).

offer an explanation. Plasticizers such as peritoneal catheters, bags, povidone-iodine and heparin have been incriminated [7–9]. However, these were unlikely to be allergens in the present case since the episode resolved despite their continued usage. As her serum IgE level was within the normal range and peripheral eosinophilia was not recognized, this eosinophilic reaction can be attributed to a process localized to the peritoneal surface. Eosinophilic peritonitis has been thought, in any case, to heal spontaneously without disturbance of peritoneal function or impairment of the structure of the peritoneum even though its cause is still unclear. However, our patient subsequently showed SEP after recurrent eosinophilic peritonitis. This fact suggested that even eosinophilic peritonitis may cause SEP if it recurs or persists, and early treatment, such as cessation of CAPD or corticosteroid therapy, should be considered in this condition.

References


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