Sclerosing Peritonitis : Report of Three Cases

D. Dequanter, J. C. Lefebvre, L. De Pauw, J. Nortier, P. Kinnaert
Medicosurgical Department of Nephrology and Transplantation, Hôpital Erasme-ULB, Brussels, Belgium.

Key words. Sclerosing peritonitis ; peritoneal fibrosis ; peritoneovenous shunt ; peritoneal dialysis ; end-stage renal failure.

Abstract. Sclerosing peritonitis is a dramatic complication of continuous ambulatory peritoneal dialysis and chronic peritoneal inflammation. Both visceral and parietal surfaces of the peritoneal cavity are involved. A thickened peritoneum encloses the small intestine in a «cocoon» formation which often leads to intestinal occlusion. CT scan may help obtaining an early diagnosis but diagnosis is often established with some delay or even at the time of laparotomy. Our report describes three cases of this uncommon peritoneal fibrosis syndrome which caused intestinal obstruction requiring surgical intervention.

Introduction

Sclerosing peritonitis (SP) is an uncommon and potentially lethal complication of continuous ambulatory peritoneal dialysis (CAPD). Other aetiologies have been reported, including recurrent subclinical peritonitis, chronic oral intake of beta-adrenergic blocking agents and chronic peritoneal irritation due to dialysis solution (1). An inflammatory reaction transforms the peritoneal membrane into a thick fibrous tissue, enveloping the whole small bowel in a “cocoon” formation which may result in subocclusion or obstruction (2). Mortality rates up to 78% have been reported, despite removal of the peritoneal catheter, suppression of the suspected causal agent and/or surgical management (3).

We report three cases of sclerosing peritonitis (SP).

Case Reports

Case 1

In 1987, a 42-year old male patient developed an end-stage renal failure secondary to rheumatoid purpura. After 12 months of haemodialysis, he received a cadaveric renal allograft, which failed after 5 years due to chronic rejection. Haemodialysis had to be restarted. In 1993, he developed intractable ascites due to post-hepatitis B cirrhosis and a Le Veen peritoneovenous shunt was surgically inserted.

In January 1996, the patient presented with worsening abdominal discomfort, nausea, subpyrexia, ascites and biological inflammatory parameters. Similar prior episodes had been medically treated in another institution. Physical examination revealed generalized rigidity of the abdominal wall with right hypochondrial tenderness. Ultrasound examination demonstrated ascites. Obstruction of the Le Veen shunt was shown by isotopic investigation. All bacteriological tests were negative and the patient underwent the removal of the shunt through sub-costal laparotomy.

He received systemic antibiotic therapy without improvement of his clinical status. Gastrointestinal X-ray study revealed a delayed intestinal transit. Colonoscopy as well as Gallium isotopic scintigraphy were normal. Abdominal CT scan demonstrated severe peritoneal thickening, retraction of the mesentery and adherent small bowel loops leading to the diagnosis of SP. A median laparotomy was performed for adhesiolysis. The whole small intestine was enclosed in a thick and fibrous peritoneal capsule typical of SP.

After surgery, improvement of all symptoms was observed and the patient was given immunosuppressive treatment (prednisolone 20 mg and azathioprine 75 mg daily). He had no further symptoms related to his SP with a follow-up of 36 months.

Case 2

A 20-year old male patient had been treated by CAPD for 3 years, for end-stage renal failure due to obstructive uropathy. After a short interruption period following cadaveric renal transplantation which failed after 2 months, the patient had to restart CAPD.

From September 1994 to January 1995, the patient experienced 3 episodes of peritonitis due to staphylococcus aureus which were treated by vancomycin intra-peritoneally. In one episode, rifadine was orally added.

In May 1995, the patient was admitted for recurrent spastic abdominal pain episodes associated with vomiting and diarrhoea. A biological inflammatory syndrom
was present. Abdominal X-ray and CT scan confirmed the diagnosis of subocclusion. Gastric aspiration during several days solved the problem for the first and the second episodes. A small bowel study performed during the third one, detected an obstacle suspected to be adhesion or torsion of the jejunum.

A diagnostic laparoscopy showed a severe extensive SP involving the small bowel. Laparotomy was performed. Each encapsulated loop of small bowel was surgically lysed and the CAPD catheter was removed. The postoperative period was uneventful and the patient was discharged from the hospital with an immunosuppressive treatment (prednisolone - 20 mg/day, azathioprine - 50 mg/day). Up to now, the patient had no further problems of occlusion.

Case 3

A 45-year old male kidney transplant recipient presented at the emergency room with abdominal pain, vomiting, diarrhea and biological inflammatory parameters. The patient had already received two cadaveric renal allografts for end-stage renal disease. After the failure of his second graft, related to chronic rejection, the patient initiated CAPD. On July 1998, he received a third cadaveric renal allograft. The catheter of CAPD was removed after six weeks.

Abdominal X-ray and CT scan confirmed the diagnosis of subocclusion. Despite a medical supportive treatment, no improvement of his clinical status occurred. Laparotomy was performed, confirming the diagnosis of SP. The small bowel as well as the stomach and colon were involved.

Encapsulated intestinal loops were surgically lysed. During the procedure, a small bowel perforation occurred and was immediately repaired.

During the postoperative period, no clinical improvement was noticed. The patient remained septic and underwent a second laparotomy. A small bowel fistula was discovered and intestinal resection was performed.

Broad spectrum antibiotherapy (meropenem, ampicillin and amphotericine B), was initiated but was unsuccessful.

One month later, a second episode of peritonitis due to streptococcus E. Coli and candida albicans occurred. The patient was treated with intestinal resection. One month later, he presented a third episode resulting from an anastomotic leakage treated by ileostomy. The immunosuppressive therapy was withdrawn but the clinical condition remained unchanged. The patient died from septic shock.

Discussion

Sclerosing peritonitis is a rare but serious complication of peritoneal dialysis (4). The aetiology is usually unknown but is likely to be multifactorial. Early studies implicated chlorhexidine in alcohol sterilizing sprays, acetate dialysate, endotoxin from in-line bacterial filters (4). Heat sterilization of dialysate results in dextrose degradation products which are the major toxic factor in peritoneal dialysis solutions (5). Plasticisers and particulate matter in the dialysate as well as the hypertonicity and acidity of the dialysate have also been implicated (5). Other reports described other possible predisposing factors: recurrent peritonitis, intraperitoneal irritants such as antibiotics and b-blockers (7). In our 3 cases, sclerosing peritonitis occurred in association with peritoneal dialysis in two cases and with a Le Veen shunt in one case.

Clinically, SP is characterized by a continuous evolution from abdominal discomfort, nausea, vomiting and impaired ultrafiltration during CAPD to complete small bowel occlusion (1, 8). The most common complications are intestinal necrosis, enterocutaneous fistulas and malnutrition (4), responsible for a mortality rate due to sepsis and malnutrition between 20 and 78% (3, 9). The third patient (the one who died) had sclerosing peritonitis involving not only the small bowel but also the stomach and the colon. All intestinal loops were involved. In the two other cases, only the small bowel was involved by sclerosing peritonitis.

The diagnosis of sclerosing peritonitis is usually done at laparotomy. Specific features on abdominal CT scan and ultrasound of the peritoneum have been used to support the diagnosis (9). Ultrasonography shows a characteristic tri-layer structure of the thickened intestinal wall: an echogenic membrane, an echo-poor layer and the echogenic bowel contents (8). Abdominal CT scan discloses a characteristic aspect of sclerosing peritonitis. It detects the presence of a pathognomonic peritoneal thickening, enclosing the small bowel in a “cocoon” formation (8). This feature was present in our cases. Sometimes, an inflammation thickens and shortens the mesentery.

When surgery can be avoided, cessation of CAPD, early removal of catheter and initiation of total parenteral nutrition has been recommended as soon as diagnosis is suspected. (3, 9). Per-dialytic parenteral nutritional support is particularly indicated, as all these patients must be shifted to haemodialysis. In most reported cases, surgery had to be performed. It was associated with a high mortality rate, up to 78% (3, 9). During adhesiolysis, extreme care must be taken to avoid enterotomies or intestinal resection in this breakable bowel wall (1, 9). Indeed, there is a high incidence of fistula formation and anastomotic leakage when an intestinal suture or anastomosis are performed (10). In our third case, relapsing peritonitis occurred despite several attempts to close the intestine.
Immunosuppression has been recommended prior to surgery to reduce the adhesion of the sclerosed peritoneum to the bowel wall (3). Junor and McMillan (11) were the first to report better outcomes in patients with sclerosing encapsulating peritonitis receiving immunosuppressive therapy. In their study of 17 cases which were mainly secondary to chlorhexidine, four patients were transplanted using prednisolone 20 mg daily and azathioprine or cyclosporine. A further patient, transferred to haemodialysis, received prednisone 10 mg and azathioprine 50 mg daily. Of the five immunosuppressed patients, none died, while the twelve who did not receive immunosuppression died within one year with recurrent bowel obstruction. In our series, the three patients received immunosuppressive therapy; two are alive after 3 and 5 years of follow-up. The last patient, with a poor nutritional status, died from septic shock. Hawley C. et al. described a rapid and dramatic improvement in gastrointestinal function that occurred, after successful renal transplantation, in a woman with severe sclerosing peritonitis secondary to continuous ambulatory peritoneal dialysis. They postulate that the anti-inflammatory effect of the immunosuppressive agents was the most important factor leading to the patient’s recovery (6). A recent report by Bowers V. D. et al. (1) describes three cases of sclerosing peritonitis presenting after renal transplantation. These cases presented clinically as varying degrees of mechanical small bowel obstruction.

Their postoperative course from the gastrointestinal standpoint were unremarkable.

Bandhari S. et al. (3) reported a case in which, after 6 weeks of treatment with azathioprine and corticosteroids, there were a marked improvement in the degree of adhesions observed at a second laparotomy (3). Mori Y. et al. (7) published the case of a patient with peritoneal dialysis-related sclerosing peritonitis who responded to corticosteroid therapy alone.

In a review of their experience with sclerosing peritonitis, Selgas R. et al. (12) reported an apparent beneficial effect of renal transplantation and/or immunosuppression, including intraperitoneal corticosteroids.

Table 1 shows the different approaches with or without use of immunosuppression after surgery to treat sclerosing peritonitis.

When the diagnosis of small bowel obstruction is considered in a patient on CAPD or with another peritoneal catheter, sclerosing peritonitis should be considered as a possible aetiology and often represents a difficult surgical challenge. In addition, surgical exploration in this patient group, especially immunosuppressed individuals post-replacement transplant is a technically demanding labor-intensive procedure. Extreme care must be taken to avoid enterotomies, as any sutures lines in this abnormal bowel wall are difficult to work with and prone to fistula formation.

Although the mechanism of immunosuppression effect on sclerosing peritonitis is not well established, immunosuppressive therapy should be considered with the aim of achieving prolonged remission from this frequently fatal complication of peritoneal dialysis.

The beneficial effect of immunosuppression has not been clearly proven. However, our experience, confirmed by other reported cases, suggests that immunosuppression should be avoided in the cases of sclerosing peritonitis complicated by intestinal fistula.

References


D. Dequanter  
Paardestraat 48A  
B-1640 Sint Genesius Rode, Belgium