stable, except for a mild tachycardia. Because of the progression, daily haemodialysis was continued for a further 2 weeks, during which a detailed search for accompanying diseases was completed, with negative results [white blood cells = 6000/mm³, anti-neutrophil; antibody (–), anti-DNA (–), C3 = 1.531 g/l, C4 = 0.42 g/l, erythrocyte sedimentation rate = 98 mm, C-reactive protein = 12.8 g/l, fibrinogen = 5.27 g/l, etc.]. While the adequacy of dialysis was confirmed, the pericardial effusion did not change. Methylprednisolone at a dose of 40 mg/day was added, and a month later we noticed a moderate reduction of the effusion (anterior wall, 8.4 mm; posterior wall, 13.1 mm). The administration of methylprednisolone was tapered over 2 months to full cessation. However, 1 month after its discontinuation (6 months after the initiation of dialysis), a new increase in pericardial effusion was observed (anterior wall, 15 mm; posterior wall, 16 mm). The patient received a new course of steroid (1 mg/kg body weight), but a month later pericardial effusion had progressed further. Since the patient remained haemodynamically stable, the surgeon’s opinion was against intervention.

The type of surgical procedure utilized for cardiac tamponade is usually determined by local experience and by the condition of the patient. Acute cardiac tamponade with circulatory collapse should be treated with pericardiocentesis. On the other hand, pericardiocentesis is not recommended for effusions that do not produce circulatory compromise. The morbidity and the potential of mortality (due, for example, to right ventricular laceration) in this setting are not insignificant. In the literature, there are two justified indications for pericardiectomy or pericardiectomy: (i) any recurrence (and certainly more than one recurrence) if accompanied by cardiac tamponade; and (ii) if a recurrence is manifested principally by persistent pain, despite a trial of intensive medical treatment and evidence of serious steroid toxicity [2,3]. None of the above conditions were met in the present case. Our review of the literature led us to the administration of colchicine, 2 mg per day for 5 weeks, followed by 1 mg and then 0.5 mg per day, for a total of 18 months. Steroids were gradually tapered and finally stopped after 6 weeks.

Seven weeks after the initiation of colchicine, the pericardial effusion was diminished (anterior wall, 13.5 mm; posterior wall, 15 mm). Further reduction of the pericardial effusion was noticed 4 months after the initiation of colchicine (anterior wall, 7.6 mm; posterior wall, 9.7 mm); 6 months later, the remaining effusion did not exceed 5–6 mm. Thirty-six months later, the patient remains free of pericarditis.

In this rare case of resistant pericarditis, we used colchicine after the failure of intensive dialysis and steroids administered for an adequate time. In a review of the available data, Adler et al. reported numerous cases of non-uraemic recurrent pericarditis treated effectively with colchicine [4]. Colchicine is considered to exert its action through the inhibition of nucleated blood cell function and motility by blocking intracytoplasmic microtubule polymerization. This occurs independently of the underlying inflammatory process [5]. Some authors recommend its use even as an initial treatment of acute pericarditis. Satisfactory results are obtained with sufficient doses for an adequate time [6]. The administration of small doses of colchicine, as an alternative treatment of refractory pericarditis, is included in guidelines of the World Heart Federation [7], but its use in uraemic patients has not been reported in the literature. In the present case of intractable pericardial effusion, colchicine proved to be helpful, efficient and safe.

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Corticosteroid and tamoxifen therapy in sclerosing encapsulating peritonitis in a patient on continuous ambulatory peritoneal dialysis

Sir,
Sclerosing encapsulating peritonitis (SEP) is a clinical syndrome associated with ileus symptoms and irreversible sclerosis of the peritoneal membrane [1–4]. There is no evidence-based therapy for SEP [2]. Current suggestions include anti-inflammatory and immuno-suppressive drugs [2–7]. We report here a case of SEP, presenting with refractory peritonitis and severe abdominal symptoms.

Case. A 29-year-old woman developed end-stage renal disease secondary to reflux nephropathy in 1992. She was started on haemodialysis and was subsequently transferred to continuous ambulatory peritoneal dialysis. She was hospitalized with high fever, abdominal pain and turbid dialysate. Despite vancomycin and amikacine, she remained unwell. She was then switched back to haemodialysis. Abdominal tomography was diagnostic for SEP showing enlarged small bowel loops with an increase of peritoneal thickness. She underwent laparotomy and biopsies were taken from the peritoneum. Pathological examination confirmed the clinical diagnosis of SEP. The patient was given tamoxifen 10 mg/day and prednisolone 0.5 mg/kg/day. Her symptoms improved gradually over 2 months with an increase of serum albumin and body weight.

Chlorhexidine gluconate in alcohol, a cleanser for the peritoneal dialysis catheter, is responsible for the development of SEP [1,6,8]. Glucose-based dialysis solutions, recurrent peritonitis attacks, plasticizers and particles may also be aetiologic factors [6]. The diagnosis of SEP is generally made on a peritoneal biopsy. The mortality rate was up to
The initial step in therapy should be the cessation of peritoneal dialysis [2]. Therapy with corticosteroids is effective in patients with SEP, and steroid therapy should be considered as the first line therapy [4]. Tamoxifen, a non-steroidal anti-oestrogen drug, has been successfully used in the treatment of fibrosclerotic disorders [3,10]. Transforming growth factor β1 (TGF-β1) has a stimulatory effect on metalloproteinases-2 and -9 (MMP2 and MMP9) [11]. Since MMP9 degrades type IV and denaturated collagens, TGF-β1, production of which is stimulated by tamoxifen, might favour mesothelial healing by facilitating the removal of denaturated collagen. In conclusion, SEP is no more a fatal complication if peritoneal dialysis treatment is interrupted promptly, and if immunosuppressive agents are administered.

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