As outlined in a recent meta-analysis [6] and an editorial [7], attempts to reduce the total cost of anaemia therapy, thereby allowing more patients to benefit from the same healthcare system, are of utmost importance.

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**Peritonitis caused by *Agrobacterium tumefaciens* in a child on peritoneal dialysis**

Sir,

Although peritonitis in patients with terminal renal failure treated with peritoneal dialysis is most often caused by common organisms, many cases of peritonitis due to unusual pathogens have also been reported. Here we present a case of peritonitis caused by one such uncommon organism, *Agrobacterium tumefaciens*, in a girl with Jeune syndrome and terminal renal failure (the case was presented by us elsewhere) [1]. From the age of 4 years, the patient was treated by continuous cyclic peritoneal dialysis (CCPD). No case of peritonitis developed over the 3 years on CCPD.

At the age of 7 years, the patient was referred to the University Hospital because of the worsened state of her health. Physical signs on the last admission were slight face oedema, distended abdomen, no diuresis, no changes at the PD catheter exit site; blood pressure 130/110 mmHg. Peripheral blood counts were unremarkable. Blood chemistry was: blood urea nitrogen (BUN) 24.1 mmol/l, creatinine 1257 μmol/l, phosphate 2.08 mmol/l, compensated metabolic acidosis. The PD programme and antihypertensive treatment were intensified accordingly. Some 10 days later, abdominal pain and fever appeared, the dialysate became cloudy with 825 leukocytes/μl and Gram-negative rods were visible on a stained smear. Peripheral blood analysis showed: white blood cells $13.8 \times 10^9$/l with neutrophilia (56.4 %), erythrocyte sedimentation rate (ESR) 40 mm/h, C-reactive protein (CRP) 155 mg/l. The dialysate culture yielded growth of *A. tumefaciens* (identified by BBL Crystal™ Identification Systems, Enteric/Nonfermenter ID Kit, Becton, Dickinson) susceptible to amoxicillin clavulanate, ticarcillin clavulanate, ampicillin sulbactam and ciprofloxacin, and resistant to all aminoglycosides, cephalosporins and carbapenems tested. Treatment with ciprofloxacin intraperitoneally and ampicillin sulbactam intravenously resulted in a temporary decrease of dialysate leukocyte count; however, the dialysate did not become completely clear and a repeated dialysate culture showed the growth of the same organism. The PD was discontinued, a peritoneal catheter was removed and haemodialysis was introduced. Ciprofloxacin was given intravenously for 10 days. The patient’s state improved and she was discharged without any signs of bacterial infection.

A Medline search yielded only two publications on peritonitis caused by *Agrobacterium* sp. and only one of them in patients with end-stage renal disease maintained on chronic PD [2]. The two patients described initially responded to antibiotics, but later relapsed and required removal of the catheter.

*Agrobacteria* are Gram-negative rods widely distributed in the environment [3]. The names of the species *A. tumefaciens* and *A. radiobacter* are used interchangeably and are considered to be synonymous [3,4]. Infections caused by these organisms are often associated with the presence of plastic foreign bodies. The case observed by us confirmed that it was difficult to eliminate pathogens in the presence of indwelling devices, even with the use of the agents active *in vitro*. The antimicrobial susceptibility pattern of the strain isolated is of interest, because it was resistant to cephalosporins, aminoglycosides and carbapenems. A recent publication showed complete sensitivity to carbapenems, amikacin and ciprofloxacin [5].

Thus, peritonitis in patients on continuous PD can be caused by a wide range of opportunistic microorganisms, including *Agrobacterium* sp. The treatment should be based on the results of an individual susceptibility pattern; however, a cure can hardly be expected without removal of the peritoneal catheter.

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Chronic graft-versus-host disease (GVHD) is one of the most frequent complications after bone marrow transplantation. Nephrotoxicity related to the use of cyclosporine (CsA) is common, but nephrotic syndrome has been reported rarely. Membranous glomerulonephritis has been found in the majority of patients [1–6], and in one case minimal change nephrotic syndrome was reported [7]. All these cases had chronic GVHD.

We describe a patient with nephrotic syndrome developing after CsA withdrawal, who failed to improve with prednisone but achieved remission on CyA.

A 59-year-old male was diagnosed with severe aplastic anaemia. He received an allogenic transplant from his HLA-identical brother. Cyclophosphamide and total lymphoid irradiation were used as conditioning, and a short-term methotrexate plus CyA as GVHD prophylactic treatment was given for 1 year. CsA was tapered and withdrawn after 14 months. One month later, he presented with nephrotic syndrome with 24 h urine protein of 5 g, serum albumin of 2.2 g, normal renal function (serum creatinine 0.8 mg/dl and creatinine clearance 120 ml/min). Hepatic enzymes increased and skin lesions consistent with GVHD appeared. Search for antinuclear antibodies, serology for hepatitis B and C and VIH was negative. A percutaneous renal biopsy revealed 10 glomeruli. Seven were optically normal, one sclerosed and two had slight ischaemic changes and a small sclerosed lesion coincidental with an area of interstitial fibrosis and inflammation. The rest of the glomeruli were optically normal. Direct immunofluorescence staining was negative for IgG, IgA, IgM, C3, C4, C1q and fibrinogen antibody. Electronic microscopy showed small homogeneous subepithelial electron dense deposits without reaction of the basement membrane. These findings are characteristic of the diagnosis of early membranous nephropathy. The patient started with prednisone (60 mg), but 1 month later 24-h urine protein was 11 g and serum albumin 1.9 g/dl. He was then treated with CsA (4 mg/kg/day) and 20 mg prednisone. Four months later he had no clinical or laboratory features of GVHD or nephrotic syndrome. Prednisone was withdrawn and CsA tapered to 2 mg/kg/day during 12 months and to 1 mg/kg during 6 months. Currently he has a 24-h urine protein excretion of 600 mg, no clinical or laboratory features of GVHD and normal hepatic enzymes and renal function.

Our case underlines the importance of having optical and electronic microscopy to achieve a correct diagnosis and shows the occurrence of nephrotic syndrome immediately after CsA withdrawal reported with a good response to CsA re-introduction. Previous cases have been described in two patients who never received CsA [1], one during tapering [3] and four after discontinuation [2,6,7]. In one case, with improvement but not remission, a second biopsy [6] showed segmental sclerosis and again membranous nephropathy. Although autoantibodies were not present in most cases described in the literature, circulating or in situ immune complexes are responsible for membranous glomerulonephritis. However, cellular mechanisms may be implicated as sclerosis is also seen. Immunosuppression can easily control these manifestations. Our case report underlines the importance of CyA reintroduction in order to control GVHD-associated membranous nephropathy.

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