serological evidence for a recent viral infection from HBV, HCV, EBV, CMV, or HIV. Serological tests were also negative for Brucella, Salmonella, Rickettsia, Trichinellosis, Filariasis, Echinococcosis and Cysticercosis, but were strongly positive for toxocariasis. Enzyme-linked immunosorbent assay for toxocarial antibodies showed 2.7 U (>1.1). Antinuclear, antineutrophil cytoplasmic antibodies, cryoglobulins and immune complexes were all negative. Computed tomography scans of the lungs, brain and abdomen were unremarkable. A bone marrow aspirate and biopsy revealed increased cell infiltration of the bone marrow with the predominance of eosinophils and a normal caryotype. Endoscopy of the stomach and duodenum was unremarkable and biopsy showed eosinophilic infiltration. Ultrasound of the heart and ophthalmoscopy were normal. A percutaneous biopsy of the left kidney was performed. Histopathological examination revealed 29 glomeruli with diffuse thickening of the glomerular capillary wall with spikes and podocyte hypertrophy, patchy tubular cell swelling and patchy tubular atrophy with loss of the brush border and absence of interstitial inflammatory cell infiltrate. Immunofluorescence showed fine granular IgG(+) and C3d(+) and IgM(+) deposits in the capillary wall and also IgM(+) deposits in the mesangium, while IgA, Clq and C4 were negative. This was consistent with membranous glomerulonephritis stage 0–1. The mesangial deposits suggested a secondary cause. On the basis of the strong serological positivity for toxocariasis and the marked eosinophilia, diagnosis of VLM syndrome was made. The nephrotic syndrome was attributed to toxocariasis. The patient was treated with prednisone (1 mg/kg p.o. daily) for the marked eosinophilia and with albendazole (10 mg/kg p.o. twice a day for 7 days). The steroid treatment resulted in the complete disappearance of eosinophilia and fever within 48 h. The patient was discharged from hospital in good clinical condition. After 1 month of prednisone therapy, total serum protein was 5.4 g/dl, albumin 2.8 g/dl and proteinuria had decreased to 1.5 g/24 h. Two months later, while still under prednisone therapy (0.6 mg/kg), proteinuria increased to the level of 2 g/24 h. At this point, ciclosporin was initiated at a dose of 3 mg/kg and prednisone was slowly tapered over the next 2 months, then stopped. Two months after ciclosporin treatment, proteinuria was 300 mg/24 h and 5 months after ciclosporin introduction, nephrotic syndrome is still in remission.

Toxocara infection can cause three distinct clinical syndromes in humans: VLM, OLM and covert toxocariasis. Myocarditis, nephritis and involvement of CNS have been described. In a report on paediatric patients from Egypt, toxocara infection was found in 10.7% of patients presenting with renal disease. Two of these patients had nephrotic syndrome; however, a kidney biopsy was not performed. In another case report from the Liverpool School of Tropical Medicine, nephrotic syndrome in a 7-year-old boy coincident with *T. canis* infection was described. In this case, a biopsy was performed and it was consistent with minimal change disease and the nephrotic syndrome responded to corticosteroids. In mice infected with toxocara, the predominant renal lesion is mesangio proliferative glomerulonephritis. Toxocariasis may manifest as nephrotic syndrome and should be considered in a patient, who presents with marked eosinophilia and nephrotic syndrome, as a possible, although rare, cause.

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dialysate volume and besides the glucose concentration, a higher dwell volume also increases phosphate removal [3]. However, when the residual renal function diminishes, a neutral phosphate balance can only be obtained by using extremely high dialysate volumes, which is hardly feasible in clinical practice.

Although it has been shown that a somewhat better control of serum phosphate is possible in patients on CAPD compared with those on HD [2,3], phosphate control is a major problem in PD patients as well. As part of the Netherlands Cooperative Study on the Adequacy of Dialysis (NECOSAD), a large prospective multicentre study, we included a total of 1629 incident dialysis patients of whom 36% was treated with PD [4]. The majority of the 586 PD patients were treated with CAPD (87%) and had a significantly lower mean plasma phosphate concentration than HD patients 3 months after the start of dialysis (1.73 vs 1.87 mmol/l, \(P<0.0001\)). A large proportion of both the HD and PD patients (53 and 41%, respectively) had plasma phosphate concentrations exceeding the target range advised in the K/DOQI guidelines for bone metabolism and disease [4,5]. More importantly, the all-cause mortality risk was significantly increased by 40% in HD and 60% in PD patients with plasma phosphate concentrations above the K/DOQI target [4].

We conclude that phosphate retention is, as in the HD patients, a major problem in PD patients as well and that the control of serum phosphate is a highly relevant issue in both the patient groups.

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