Conflict of interest statement. None declared.

Renal Transplant Unit  
St George’s Hospital  
London, UK  
Email: jiri.fronek@Stgeorges.nhs.uk


doi:10.1093/ndt/gfl107

Advance Access publication 7 July 2006

Nephrotic syndrome as a manifestation of Toxocara canis infection

Sir,

Toxocariasis is the clinical term describing infection in the human host with either Toxocara canis or less commonly, T. cati, both of which are ascaride nematodes, normally parasitic for non-human host species. There are two main syndromes associated with toxocariasis, visceral larva migrans (VLM) and ocular larva migrans (OLM).

We describe nephrotic syndrome in a 41-year-old woman coincident with T. canis infection. The relationship between T. canis infection and glomerular disease is still unclear. There are very few reports of nephrotic syndrome associated with toxocariasis, and the present is, to our knowledge, the first reported in an adult.

A 41-year-old female presented to our department with fever, mild pain in the epigastrium and right upper quadrant, diarrhoea and marked eosinophilia. The patient had been working as a cleaner for a long time. Her medical history was clear and she was well until 3 weeks before admission, when she developed mild epigastric and right upper quadrant pain, accompanied by diarrhoea. The symptoms persisted and 4 days before admission she started having fever (38–38.7°C).

Upon admission, physical examination revealed mild pallor of the skin and conjunctivae, ankle oedema and mild tenderness in the right upper quadrant. There was no lymphadenopathy, or hepatosplenomegaly, and the body temperature was 37.8°C. Full blood count showed haemato- crit 29.4%, haemoglobin 9.3 g/dl, white blood cell count 3.33 x 10^6/l with neutrophils 25%, lymphocytes 6%, monocytes 2% and eosinophils 65% (absolute number 21.6 x 10^6/l), platelet count 445 x 10^6/l, ESR 102 mm/h. Urinalysis showed pH 7, specific gravity 1020, haemoglobin +++, protein +++++, RBC >100/HPF, granular and epithelial cell casts. The amount of protein in urine was 5.7 g/24h. Urine electrophoresis showed total non-selective proteinuria. Biochemistry tests showed normal liver and renal function (Creatinine: 0.6 mg/dl). Total serum proteins were 4.3 g/dl, albumin 1.5 g/dl, globulins 2.8 g/dl, calcium 7.4 mEq/l and cholesterol 234 mg/dl. Protein electrophoresis revealed a1 6.1%, a2 21.3%, b 10.9%, g 27.4%, albumin 34.3% and the measurement of serum immunoglobulins was IgG 864 mg%, IgA 209 mg%, IgM 746 mg%, IgE 837 mg%. Multiple stool, blood and urine cultures were negative. There was no...
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(+)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.

Conflict of interest statement. None declared.


doi:10.1093/ndt/gfl224

Advance Access publication 23 May 2006

Hyperphosphataemia and related mortality

Sir,

With great interest we read the editorial review of Jean et al. [1] on the relationship between hyperphosphataemia and mortality in end-stage renal disease patients. The authors summarize results from the large USRDS and DOPPS studies in which associations of hyperphosphataemia and increased mortality risks were found in haemodialysis (HD) patients. Jean et al. [1] evaluate the capacity of phosphate removal per session and per week by several dialysis techniques, including standard haemodialysis (SHD), haemodiafiltration (HDF), nocturnal daily haemodialysis (NDHD), short daily haemodialysis (SDH) and long haemodialysis (LHD). However, the authors did not mention peritoneal dialysis (PD) treatment in their comparison, which is in our opinion, an important dialysis modality as well.

Considering phosphate balance, there are some marked differences between HD and PD treatments. Due to obligatory protein losses via the peritoneal fluid, continuous ambulatory peritoneal dialysis (CAPD) patients are often prescribed a high-protein diet containing 1.2 to 1.3 g/kg/day which provides a phosphate intake of up to 1200 mg/day [2]. Effective phosphate removal with dialysis and gastrointestinal elimination of phosphate by oral phosphate binding agents is therefore necessary to achieve a neutral phosphate balance.

With PD treatment, on average, 315 mg/day (2200 mg/week) of phosphate is removed from the body, which is less than that with different types of HD treatment [2]. The removal of phosphate in PD patients depends on the