Toxicariasis and Nephrotic Syndrome

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ABSTRACT. We describe a case of toxicariasis as a rare cause of nephrotic syndrome in an adult woman. This rare association was confirmed by elevated Toxocara-specific immunoglobulin M titers. Renal biopsy was not done because of prolonged activated partial thromboplastin time. Our patient was treated with prednisone and albendazole. These treatments resulted in partial remission of renal symptoms as well as the abatement of the Toxicariasis infection. The relationship between toxicariasis infection and glomerular disease is still unclear. In the literature, exceptional renal impairment secondary to toxicariasis have been described, especially in children. To the best of our knowledge, this is the second case of nephrotic syndrome associated with toxicariasis in adults.

Introduction

Toxicariasis is a helminthic zoonosis with a worldwide distribution, and until now, it continues to be an almost unknown disease among the general population and even among health professionals. Infection can present with a wide clinical spectrum varying from asymptomatic to severe organ injury. There are three major clinical syndromes associated with human toxicariasis; covert toxicariasis, ocular larva migrans, and visceral larva migrans. Visceral larva migrans is the most prevalent type of the disease and the classical presentation includes fever, hepatomegaly, and eosinophilia (>500/mm³). Renal involvement has been only rarely reported. In this case report, we describe a 69-year-old woman presenting with nephrotic syndrome with coincident toxicariasis infection.

Case Report

A 69-year-old woman was on follow-up in the cardiology department since 2012 for mitro-aortic valvulopathy with atrial fibrillation requiring anticoagulant therapy. She was hospitalized in the Cardiothoracic Surgery Department for surgical treatment of her valvulopathy. On admission, she was noted to have lower limb edema, and laboratory parameters showed features of nephrotic syndrome. She was, therefore, transferred to our department to evaluate the glomerulopathy. There was a history of pruritus lasting for one year.
Physical examination revealed mild pallor of the skin and conjunctivae, significant lower limb edema, and scratching lesions. The body temperature was 36.5°C. There was no lymphadenopathy or hepatosplenomegaly. The patient was eupnoeic and respiratory auscultation was unremarkable. Her blood pressure was 110/70 mm Hg, heart rate was 80 bpm, and cardiac examination revealed a holosystolic murmur heard at the apex, with radiation into the axilla with jugular veins distension but without the hepato-jugular reflux. Urinalysis showed pH 6, specific gravity 1025, hemoglobin 1+, protein 3+, and the absence of nitrites and leukocytes. Proteinuria was recorded as 5.91 g/24 h. Fractional excretion of sodium was <1%. Multiple stool, blood, and urine cultures were negative.

Complete blood count showed hemoglobin 9.1 g/dL, white blood cell count 8,300/mm³ with neutrophils 4600/mm³, lymphocytes 1800/mm³, monocytes 500/mm³, and eosinophils 1300/mm³, and platelet count 338,000/mm³. The erythrocyte sedimentation rate was 37 mm in 1 h and the C-reactive protein was negative. Liver functions were normal. Serum creatinine was 255 µmol/L, and urea was 21.2 mmol/L. Total serum proteins were 52 g/L, albumin 28 g/L, α1 globulins 2.3 g/L, α2 globulins 8.4 g/L, β globulins 6.4 g/L, gamma globulins 6.2 g/L, calcium 2.48 mmol/L, cholesterol 4.6 mmol/L, and triglycerides 2.2 mmol/L. The measurement of serum immunoglobulins was IgG 7.6 g/L, IgA 1.98 g/L, and IgM 0.48 g/L.

There was no serological evidence for a recent viral infection from HBV, HCV, CMV, or HIV. Serological tests were also negative for leishmaniosisis, schistosomiasis, hydatidosis but was strongly positive for toxocariasis. Enzyme-linked immunosorbent assay for toxocaral antibodies showed 1.9 U (>1.1) and the Western blot was also positive. Thyroid hormone levels were normal. Antinuclear, antineutrophil cytoplasmic antibodies, and cryoglobulins were all negative. Tumor markers (ACE, CA19-9, CA15-3, CA-125, and AFP) were also negative. Abdominal and pelvic ultrasounds and computed tomography scans of the brain were unremarkable. Ultrasound of the heart was normal. Endoscopy of the stomach and duodenum was unremarkable. A percutaneous biopsy of the left kidney was indicated but was not performed because of prolonged activated partial thromboplastin time despite discontinuation of anticoagulation.

On the basis of the strong serological positivity for toxocariasis and the marked eosinophilia, diagnosis of visceral larva migrans syndrome was made and the nephrotic syndrome was attributed to toxocariasis. The patient was treated with prednisone (1 mg/kg per os daily) and with albendazole (10 mg/kg per os twice a day for 7 days). The steroid treatment resulted in the complete disappearance of eosinophilia and negativity of the toxocara serology. After one month of prednisone therapy, total serum protein was 53 g/L, albumin was 29.5 g/L, and proteinuria had decreased to 1.69 g/24 h. However, the patient has not come for follow-up since April 2013.

**Discussion**

Toxocariasis is a multi-systemic disease of parasitic zoonosis that occurs, especially in young children. In adults, toxocariasis is unusual and infections appear to be mild or subclinical, but may provoke positive serological tests and sometimes, persistent eosinophilia. Many infections caused by *Toxocara* are asymptomatic, reaching 44.4% and systemic toxocariasis manifests around 15.5% of diagnosed cases. Because of the variability of signs and symptoms of the disease, in 1988, toxocariasis was divided into two main types: visceral larva migrans and ocular toxocariasis. Between 1992 and 1993, a third clinical form called covert toxocariasis was described in seropositive patients, gastrointestinal disturbances, weakness, and lethargy. Myocarditis, nephritis, and involvement of the central nervous system have also rarely been described. In a report on pediatric 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, nephrotic syndrome in a seven-year-old boy coincident with *Toxocara canis* infection was described and remission of renal symptoms was obtained after steroids treatment and abatement of the *Toxocara* infection. In this case, a biopsy was performed and it was consistent with minimal change disease and the nephrotic syndrome responded to corticosteroids. In an adult case report, corticosteroid-resistant nephrotic syndrome was described in a 41-year-old woman. Renal remission was obtained after the *ciclosporin* introduction. Our case is the second case reported in adults. In mice infected with *Toxocara*, the predominant renal lesion is mesangio-proliferative glomerulonephritis. Immunohistochemical studies established that renal alterations were associated with glomerular deposits of IgG, IgM, and third component of complement (C3).

**Conclusion**

The relationship between toxocariasis and glomerular disease is still unclear. Some authors suggested that immunomediated mechanism might possibly be involved in the genesis of kidney damage observed in this parasitic zoonosis. Toxocariasis should be considered as a possible cause, although rare of nephrotic syndrome, especially in the presence of a marked eosinophilia.

**Conflict of interest:** None declared.

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