Dear Sir,

The systemic vasculitides form a loosely associated group of diseases which have as their pathological basis a chronic necrotizing inflammation which affects vessels of varying size [1-3]. There are primary and secondary forms, primary which involve blood vessels exclusively, and secondary associated with diseases elsewhere [1-3].

Case Report

In February 1987, an 18-year-old man presenting with a 7-day history of fever (to 39 °C), headache, backache, abdominal pain, vomiting and oliguria (400-600 ml) was admitted. Blood pressure was normal (120/80 mm Hg) and fever with conjunctival injection was present only on the first day. Oliguria was followed by polyuria (1,700-3,200 ml/day) during the following 2 weeks. Spontaneous elimination of more than 10 Ascaris lumbricoides was noted in the fecal mass on the 3rd day. Renal biopsy was done on the 4th day. The patient was treated with pyrantel pamoate on the 5th and 19th day (6 × 125 mg). Bacteriological examinations of urine, feces and throat mucosa and blood cultures were negative. Antistreptolysin titer was 0, serological tests on viruses and brucellosis were also negative. Leukocytosis (13,000-17,000/ mm³) with eosinophilia (12-14%) was present. Increased serum urea, 35.7 mmol/l, and creatinine, 373 µmol/l, decreased to normal ranges. Proteinuria was between 0.8 and 2.7 g/l, plasma proteins, serum immunoglobulins and serum C3 were normal. Erythra with about 20 cells on a microscopic field was present. Mesangial proliferation, diffuse in 3 glomeruli and segmental in 7, was found, with 1 normal glomerulus. ‘Crescent’-like structure was seen in 1 glomerulus, formed by long fibroblast cells, some epithelial and inflam-
Fig. 1. ‘Crescent’-like formation formed by long fi-broblasts, epithelial and inflammatory cells. HE. 40 × 10 × 1.25.

Fig. 2. Intimal hyperplasia and mononuclear peri-vascular infiltration. HE. 16×10×1.25. Matatory cells. Swelling and lymphocyte aggregates were found in the interstitium with a moderate edema. Two smaller arteries were seen, one with a severe intimal proliferation and the other with fibrinoid necrosis and mononuclear cell infiltration of the wall. Immunofluorescent staining showed granular deposits of IgA(++), IgG(+), C3(+++) and fibrinogen(++) in glomeruli with mesangial diffuse or segmental distribution, with C3 deposits also in the arteriolar walls. Follow-up of the patient (4 years) presented complete recovery (fig. 1').

Fig. 3. Intramural fibrinoid necrosis. HE. 16 × 10 × 1.25. Fig. 4. Mesangial, granular deposits and afferent arteriolar deposits of C3. 10 × 10×1.25. Renal involvement, blood eosinophilia and affection of smaller arteries, arterioles and veins in our patient are characteristic for Zollinger HU, Mihatsch MJ: Histopathology of renal vasculitis. Kidney Int 1985;hypersensitivity angitis, but the outcome was quite different (1-5). We presented a case with secondary form of vasculitis and it is known that only cases with vasculitides associated caused by vascular diseases; in Renal Pathology in the lupus-like syndrome induced by hydralazine. Am J Nephrol 1987;7:71-73.


Grcevska/Polenakovic: Renal Vasculitis in Ascarisiasis