Letter to the Editor

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# Vascular Injury in Acute Renal Failure due to Leptospirosis Is Not Associated with Antineutrophil Cytopiasmic Antibody


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Dear Sir,

Antineutrophil cytopiasmic antibodies (ANCA) are markers of various vasculitic syndromes. The clinical spectrum of ANCA-associated diseases ranges from renal limited forms to systemic vasculitis affecting multiple organs like Wegener's granulomatosis and microscopic polyarteritis [1]. Some authors [2] have suggested that ANCA might play a pathogenic role in these diseases, but seasonal variation in the onset and the flu-like prodrome suggest that infectious agents could also be important in the etiopathogenic mechanisms. ANCA was first described in arbovirus-associated vasculitis [3], but, as far as we know, no other research was done on ANCA related to infectious vasculitis.

Leptospirosis, when occurring as Weil's syndrome, is a systemic vasculitis characterized by the injury of the endothelium of capillaries, as well as arteritis involving medium and large arteries [4]. The mechanisms of vascular damage are not completely known.

The most widely accepted theory is a toxin-induced lesion [4, 5], but an immuno-logic mechanism is also proposed [6].

If an immunologic mechanism is involved in leptospirosis vasculitis, it could be due to ANCA association.

Thus, we studied 10 patients with leptospirosis (Weil's syndrome) characterized by acute renal failure [plasma creatinine > 180 µmol/l (2.0 mg/dl)], jaundice [total serum bilirubin > 17 µmol/l (1.0 mg/dl)] and fhrom-bocytopenia (platelet count < 150 · 10V1). The diagnosis was confirmed by epidemiologic data and a positive agglutination test to Lep-tospira interrogans (titer > 1:1,600).

Patients were 9 men and 1 woman, aged 15-38 years (31 ± 7 years). Eight to twenty days (12 ± 5 days) after leptospirosis symptoms (fever and myalgia) had begun, a blood sample was collected for ANCA determination. ANCA was determined by indirect im-munofluorescence microscopy using alcohol-fixed neutrophils as substrate [1]. Other laboratory data obtained on the same day showed: total serum bilirubin = 376 ± 239 µmol/l (22 + 14 mg/dl), plasma crea-tinine = 513 ± 203 µmol/l (5.8 ± 2.3 mg/dl) and platelet count = 76.8 ± 44.1 · 109/1. Although all patients had severe disease none showed positive ANCA.

ANCA-associated vasculitis usually begins after a flu-like prodrome and has a seasonal incidence [2]. These factors suggest an infectious agent involved in its pathogenesis. In leptospirosis vasculitis mechanisms other than ANCA must be involved. Maybe viral, but not spirochetal, agents could promote ANCA-induced neutrophil activation with resultant vascular injury [2],

In leptospirosis the present results suggest that vascular injury might be due to direct toxin effect or another immunological factor but not ANCA.

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