Post-Legionellosis Proliferative Glomerulonephritis

Glomerulonefrite proliferativa associada à Legionellose

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To the Editor:

Today in developed countries, infection-related glomerulonephritides are incommon and often associated with debilitating diseases. Legionnaires' disease (LD) is one of the most common causes of severe community-acquired acute pneumonia in Europe and in up to 40% of cases of hospital-acquired pneumonia.[1] The acute kidney injury (AKI) in LD is a well described complication but the renal morphology has only been reported in a few cases. The Tubulointerstitial Nephritis (TIN) is the most diagnosed histological change;[2] instead, there are very few reports describing the glomerular injury.[3,4]

Our experience: a 41-year-old female, presented with mild dyspnoea, cough and acute renal failure (oliguria, creatinine: 5.5 mg/dL). Four weeks before, she suffered from flu-like syndrome; chest X-ray showed left lung infiltration and she was treated with ceftriaxone and claritromycine. At admission: blood pressure was 140/80 mmHg and cardiovascular, respiratory and abdomen examinations were unremarkable. Laboratory investigations: Hb 9.9 gr/dL, WBC 9960 × 10^9/L, PCR 15 mg/dL, proteinuria 3.8 g/24 h, ANA positive 1:80; HBV, HCV, Ab-DNA, ENA, c-ANCA and p-ANCA: negative. C3, C4, Immunoglobulin levels were normal. Chest X-ray and abdominal ultrasonography showed normal findings. A renal biopsy was performed: among 24 glomeruli, 7 showed extracapillary proliferation and epithelial crescents and 2 global scleroialinosis; the tubulointerstitial space displayed mild fibrosis with lymphocytic infiltration. Tubules and vessels were without relevant pathological findings, especially without signs of vasculitis. Immunofluorescent staining revealed linear IgG and C3 deposition along the glomerular capillary wall. Electron microscopy was not performed. These findings were consistent with an extracapillary-proliferative glomerulonephritis Figure 1). We performed pulse therapy with methylprednisolone (500 mg i.v. for 3 consecutive days) followed by oral prednisolone (37.5 mg/day) and cyclophosphamide 350 mg i.v.. After this treatment, we known the positive result of serum Legionella Pneumophila-1 antibody detection tests: the IgG-titre was 1: 128 and the IgM-titre was 1: 96. No sputum could be obtained for culture but the urine Ag-test for Legionella Pneumophila serogroup 1 was also positive. We diagnosed a crescentic glomerulonephritis post-LD. We did not continue steroid and immunosuppressant therapy. The renal function improved until the complete recovery after 3 months. We did not identify any source of her Legionella Pneumophila infection in the patient's apartment. LD is recognized as a multi-systemic illness. The TIN is a rare complication of LD and there are very few reports of glomerulonephritis in LD. Our experience highlights that in

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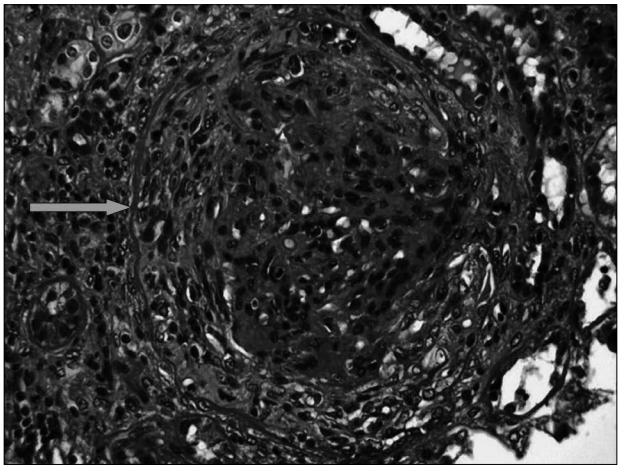
Figure 1. Fibrocellular crescent (green arrow) with compression of residual glomerular tuft (PAS, x 400).



patients with AKI and recent LD, a post-infection immunocomplex glomerulonephritis should be considered; we stress the importance of the renal biopsy in the differential diagnosis of AKI in LD.

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