Focal Segmental Glomerular-Sclerosis-Like Lesions Associated with Eosinophilic Pneumonia and Trimethoprim and Penicillin Treatment

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Dear Sir,

Both drug-induced eosinophilic pneumonias and interstitial nephritides may occur after treatment with sulfonamides and penicillin [1-5]. Reports about the glomerular diseases due to these drugs are rare, especially focal glomerulosclerosis which is generally a primary disease [6].

Case Report

A 20-year-old female patient without significant past history was treated with Lida-prim (sulfonamide, a combination of sulfametrol and trimethoprim) because of fever (38 °C), cough and expectoration of white-colored sputum during 6 days. Because of persistent clinical signs, she was admitted to the Department of Pulmoallergology and treated with penicillin (a combination of crystalline and procaine penicillin G) 800,000 U/day during 4 weeks. The patient became afebrile on the 10th day, but hilar and interstitial pulmonary infiltrations were seen on chest radiograph (fig. 1). Laboratory investigations demonstrated severe anemia with hemoglobin 62 g/l and red blood cell count 2,100,000/mm3, with a leukocytosis of 12,000/mm3 and eosinophilia of 30%. Eosinophils were also noted in the sputum and bone marrow (14%). The patient started to be oliguric on the 26th day (400-600 ml) with occurrence of swelling and increase of serum urea to 21 mmol/l and creatinine 760 µmol/l. She continued the treatment at the Department of Nephrology, and polyuria started on the 3rd day (2,000-2,500 ml). Serum levels of urea (to 13 mmol/l) and creatinine (to 168 µmol/l) decreased spontaneously. Plasmaproteins, immunoglobulins and complement C3 and C4 were normal, proteinuria was slightly increased: 0.75-0.86 g/l. Antistreptolysin titer was 0, microbiological examinations of throat mucosa and sputum and testing of feces on helminthiasis gave also negative results. Control chest radiograph as normal.

Renal biopsy was performed on the 60th day of the start of the symptoms. 10 glomeruli were found on optical microscopy: 6 complete and 3 partially sclerosed (fig. 2). Only 1 was quite normal. Tubules presented slight epithelial degeneration; swelling of the interstitium with rare eosinophils was noted with rare eosinophils (fig. 3). Intra-renal blood
vessels were normal. Focal or segmental deposits of C3 (++), IgM (++), C1q (+) and IgG (+/-) were found on immunofluorescent microscopy.

The follow-up period was 4 years; the patient was afebrile, without respiratory tract problems and improvement of the blood cell count. Diuresis was 1,500-1,800 ml, serum urea 6.1-8.9 mmol/l, serum creatinine 89-93 µmol/l, creatinine clearance 0.9 ml/s. Proteinuria was still present with 0.65-1.5 g/l.

Pneumonia, blood eosinophilia, eosinophils in the sputum and the association with trimethoprim and penicillin treatment can include pulmonary changes of our patient in acute eosinophilic pneumonia [1, 2]. Glomerular sclerotic changes found on biopsy are more interesting and unusual: there were no signs of glomerular disease before pneumonia, and renal symptoms during follow-up did not correlate with the pathological findings. Absence of hypertension, nephrotic syndrome and progression of renal failure are unusual for focal sclerosing glomerulonephritis [6]. So we classified these renal lesions which were previously described to be associated with other conditions [7] as ‘focal segmental sclerotic-like’.

References

Grcevska/Polenakovic/Breskovska
Focal Segmental Glomerulosclerosis in Eosinophilic Pneumonia