Mesangio-Capillary Glomerulonephritis and the Nephrotic Syndrome in the Course of Disseminated Tuberculosis

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Dear Sir,

Murray et al. [1] reported in the July issue of this Journal a case of rapidly progressive glomerulonephritis in a patient with pulmonary tuberculosis treated with rifampicin. In the light of a recent personal case we wish to present an alternative physiopathology of glomerular disease associated with tuberculosis.

Case Report

A 46-year-old man was hospitalized for pitting edema and hypertension (230/120 mm Hg). His temperature was normal. Physical examination disclosed liver and spleen enlargement. His past history was unremarkable. Mantoux test was strongly positive. Laboratory investigations disclosed nephrotic syndrome: urinary protein output 8 g/24 h, serum albumin 2.4 g/dl and serum globulins 1.9 g/dl. He had microscopic hematuria (24,000 red cells/minute) and mild renal insufficiency (serum creatinine 120 μ mol/l, creatinine clearance 55 ml/min). The following tests were within normal limits: serum complement fractions, Waaler-Rose, circulating immune complexes (polyethylene-glycol) and antinuclear antibodies. Lung radiographs revealed a cavity in the upper right lobe, and acid-fast bacilli were present in the sputum. Liver biopsy disclosed epithelioid granulomas. Liver specimen culture was negative. Percutaneous renal biopsy showed mesangiocapillary glomerulonephritis, and immunofluorescence was positive with anti-IgG, IgM, C1q, C4 and C3 antisera. Hypertension was controlled with β -blockers and dihydralazine, nephrotic syndrome was symptomatically treated with indomethacin (3 mg/kg for 2 months, tapered to a stop by 6 months), and tuberculosis was treated with INH, rifampicin and myambutol. The lung lesion healed within 3 months. Proteinuria gradually subsided and disappeared after one year. After a 4-year follow-up, there was no proteinuria, no blood cells in the urine and renal function was unchanged. Persisting mild hypertension was controlled with β -blockers. On light microscopy, a control renal biopsy yielding 25 glomeruli showed 8 sclerotic glomeruli,

disappearance of proliferation, persisting modification of basement membranes by focal thickening, double contours and a few spikes on the outer aspect, and some capsular adhesions. Immunofluorescence revealed mostly fixation of anti-IgM antiserum along some basement membranes but no significant staining by anti-IgG, anti-IgA, anti-C3, anti-C1q and antifibrinogen antisera.

Discussion

In 1983, Shribman et al. [2] reported the case of a man with miliary tuberculosis and focal proliferative glomerulonephritis. They assumed that the renal lesions were the consequence of immune-complex deposition. The filiation between tuberculosis and the renal disease seemed to be substantiated by clearing of the glomerular lesions after 6 months of antituberculous treatment. Our case is similar. It is noteworthy that our patient received rifampicin and that glomerulonephritis subsided with antituberculous treatment.

Mesangiocapillary glomerulonephritis can be a complication of subacute and chronic bacteremia and of visceral abscesses [3]. In such a case, circulating immune complexes are not always detected [4], and hypocomplementemia is not constant [5]. In this respect, it is not illogical to postulate that disseminated tuberculous infection might initiate a similar process and lead to occasional cases of mesangiocapillary glomerulonephritis. Nevertheless, a coincidence between a common disease, such as tuberculosis, and a frequent type of glomerulonephritis, such as mesangiocapillary glomerulonephritis, is the first explanation which should be considered. Conversely, the discovery of glomerulonephritis at the beginning of treatment of tuberculosis should not systematically be ascribed to drugs.

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