Nephritic Urinary Sediment: Not only in Proliferative Glomerulonephritis but also in Malignant Hypertension

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Dear Sir,

Nephritic urinary sediment is frequently found in severe proliferative glomerulonephritides, renal vasculitides or other conditions, such as hemolytic uremic syndrome, acute interstitial nephritis, Hantaan virus infection or chronic septicemia [1]. We report the case of a 53-year-old man admitted the Renal Unit of the Ospedale of Treviglio, Italy, on September 20, 1993, for rapidly progressive renal insufficiency and severe hypertension associated with nephritic urinary sediment not ascribable to the diseases mentioned above.

The patient had been in good health until 1991 when mild hypertension was detected. Three months before admission, the patient had had progressive weight loss (about 7 kg) and polyuria. On admission, the patient was asymptomatic. Physical examination was normal, blood pressure was 230/130 mm Hg, heart rate was 100 beats/min and temperature was 37 °C. Chest X-rays were normal. ECG showed signs of left ventricular hypertrophy. Fundoscopy showed hemorrhages with soft and hard exudates. Renal ultrasonography was normal. Serum creatinine was 3.1 mg/dl, serum urea 81 mg/dl, creatinine clearance 48 ml/min, plasma Na 143 mmol/l and plasma K 2.9 mmol/l. Hematocrit was 38.3%, erythrocytes were $4.3 \times 10^6/\text{mm}^3$, leukocytes were $6.8 \times 10^3/\text{mm}^3$ (with a normal eosinophil count), platelets were $3.4 \times 10^5/\text{mm}^3$. Anti-DNA and anti-glomerular basement membrane antibodies were absent, CRP and C3-C4 serum levels were normal, p- and c-antineutrophil cytoplasmic antibodies were negative. Plasma aldosterone concentration was high (orthostatic 3,243 pmol/l, clinostatic 989 pmol/l, normal values 97-832 and 20-416 pmol/l, respectively) and plasma renin activity was within the normal level (orthostatic and clinostatic values were 1.7 and 0.4 µg/l/h, normal values 1.5-5.7 and 0.2-2.8 µg/l/h. Urine output was 2,300 ml/

24 h. There was significant glomerular proteinuria (3 g/24 h). Microscopy revealed typical nephritic sediment characterized by dysmorphic hematuria (about 50 erythrocytes/HPF), leukocyturia (10 neutrophils/HPF) and severe cylindruria (more than one hyaline, granular and erythrocytic casts/LPF).

On September 29, 1993, a percutaneous renal biopsy was performed. By light microscopy, only two glomeruli were found, neither of which showed intra- or extracapillary proliferation. The only abnormality consisted of severe shrinking of the glomerular tufts, as seen in ischemia. There

was diffuse interstitial fibrosis without acute cellular infiltrates. Tubules were diffusely atrophic. Arteries and arterioles had narrowed lumens due to severe myointimal proliferation. The sections for immunofluorescent microscopy contained five glomeruli. No deposits of immunoglobulins, complement or fibrin (ogen) were observed. Ultrastructural examination of two glomeruli confirmed the presence of ischemic lesions only. Two arterioles had completely occluded lumens, caused by swollen endothelial cells and myointimal proliferation. These findings ruled out proliferative glomerulonephritides or renal vasculitides, and we made a diagnosis of proliferative endarteritis associated with malignant hypertension. Therefore, antihypertensive therapy was strengthened with oral nifedipine (60 mg/day) and clonidine (0.450 mg/day). In spite of normal blood pressure, creatinine slowly worsened, up to 4.1 mg/dl, but after 9 days of hospitalization progressively decreased. Urinary microscopy changes slowly cleared in parallel. The patient was discharged on October 1, 1993; serum creatinine was 3.1 mg/dl, urinary microscopy was normal and blood pressure was 120/60 mm Hg. Repeated microscopic evaluations of the urine during follow-up did not show any pathological features. At the last follow-up check, on March 24, 1994, serum creatinine was 2.5 mg/dl.

Comment. Our case shows that nephritic urinary sediment and rapidly progressive renal insufficiency can be observed in patients with renal changes due to malignant hypertension. This has been reported only once so far [2]. Since the clinical presentation may be similar to that caused by other renal diseases (i.e. proliferative glomerulonephritides, renal vasculitides, acute interstitial nephritis) which require immunosuppressive therapy, it is important to recognize this possibility, which can be diagnosed only by renal biopsy. The cause of the nephritic sediment is thought to be the presence of focal necrosis of the glomerular tuft, which has been observed in autopsy cases and, less frequently, in biopsy cases [3, 4]. We did not observe glomerular fibrinoid necrosis in the renal biopsy of our patient, but this may be due to the small number of glomeruli present in the sections. In conclusion, we think that malignant hypertension has also to be considered when a patient has rapidly progressive renal insufficiency with nephritic sediment.

References

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