Frequency of Renal Amyloidosis Secondary to Tuberculosis

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Dear Sir,

Although tuberculosis occurs infrequently it has attracted increasing attention in recent years. In part this reflects an increased awareness of its renal complications such as amyloid deposition in the kidneys. Amyloid is often first deposited in the mesangium of the glomeruli and later extends along the basement membrane. Initially present as small focal nodules, these deposits eventually become larger, obliterate the capillary lumen and replace the entire tuft. Ultimately the glomeruli become atrophic and structureless and replaced by amyloid surrounded by concentric layers of collagen.

Proteinuria was reported to be present in about 80% of patients and was not necessarily correlated with the extent of glomerular involvement [1]. Some investigators have noted that the severity of proteinuria correlates better with the presence of spicules and the destruction of podocytes. The greatest loss of protein probably occurs in areas where the basement membrane is disrupted by amyloid and denuded of its epithelial covering. 60% of the patients were reported to develop the nephrotic syndrome, while renal insufficiency was found in more than half of the patients at the time of diagnosis [2].

We studied the occurrence rate of tuberculosis causing renal amyloidosis. 237 patients with renal amyloidosis diagnosed by biopsies were investigated. Only 81 (35.6%) had a previous history of tuberculosis and the remaining 156 patients had amyloidosis due to other causes. Among these patients with renal amyloidosis secondary to tuberculosis, 55 (67.9%) were males and 26 (32%) were females. The mean age was 45.22 ± 3.5 years (range 21-58 years). The duration of

the disease was found to vary from 1 to 6 years with an average of 2 ± 1 years. 79% of patients showed clinical manifestation of the nephrotic syndrome; only 23.4% had developed renal failure. Hepatomegaly was present in 47 (58%) and splenomegaly in 18 (22%) patients. 12 patients (14.8%) showed gastric atony, 17 patients (20.9%) emphysema and 11 patients bronchiectasis (13.5%). Pleural effusion was present in 9 cases (11.1%); 23 (28.3%) patients were hypotensive and 5 cases (6.1%) had peripheric neuropathy and 10 (12.3%) a fever. Laboratory findings revealed the ESR to be high in 27 (33.3%), leukocytosis in 16 (19.7%), leukopenia in 5 (6.1%), lymphocytosis in 3 (3.7%) and eosinophilia in 22 (27.1%) patients. In 58 (71.6%) patients the hematocrit was below 30%, and in 4 cases (4.9%) pancytopenia and in 12 (14.8%) trombocytopenia were present. In 61 patients (75.3%) α_1 -, α_2 - and γ -

globulins were found to be increased and albumins to be decreased. Total proteins were below normal in 59 cases (72.8%). Total lipids were increased in 21 (25.9%) cases, total cholesterol in 18 (22.2%) patients. In 9 cases (23.4%) urea clearances were 50% below normal. In 79% (64 patients) proteinuria was more than 3 g/day, in 14.8% (12 cases) 1-1.5 g/day and in 6.1% (5 cases) it was slight. Hematuria was seen in 69 cases (85.1%) and in 14 patients (17.2%) there was pyuria.

In the present study the occurrence rate of renal amyloidosis secondary to tuberculosis is shown to be about 35 %; it has previously been reported to be 47% by Boussema et al. [3], 50% by Urban et al. [4] and 3.6% by Bely and Apothy [5] and 12% by Toydshima et al. [6]. The nephrotic syndrome was found to be the most common clinical manifestation in 79% of patients whereas in the literature it was reported to be 50% by Zollinger and Mihatsch [7].

In conclusion, it can be said that Myco-bacterium tuberculosis is responsible for the development of renal amyloidosis in a significant number of cases; therefore in cases of secondary amyloidosis, tuberculosis should always be kept in mind as one of the most common causative underlying diseases. It should always be the first disease to be investigated; in patients with the nephrotic syndrome and a previous history of tuberculosis, secondary amyloidosis should always be considered as the most common renal lesion.

References

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