Aluminum and Dialysis Arthropathy

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Sir,

It is largely known that β_2 -microglobulin (β_2 -M) amyloidosis is associated with joint pain, bone cysts and carpal tunnel syndrome in long-term hemodialyzed patients, but the pathogenetic factors of such deposits still need to be clarified. In fact, though the main protein of dialysis amyloid is β_2 -M, serum β_2 -M levels are not predictive for arthropathy [1–3]. Thus, it is likely that still other factors (such as subclinical inflammatory processes of unknown causes) in addition to high concentrations of β_2 -M are required for the formation of amyloid deposits. Among these factors, a role for aluminum (Al) has been suggested [1, 4–6]. In 1988, in a Lancet letter, Netter et al. [4] reported Al accumulation both in synovial tissue and articular cartilage of dialyzed patients. Furthermore, they reported that amyloid deposits were found in 23 of the 28 patients, and the highest concentrations of Al were in patients with β_2 -microglobulin amyloidosis.

Yver et al. [5] showed an increased post-desferrioxamine serum Al increment in dialyzed patients with carpal tunnel syndrome and articular manifestations, suggesting that Al stores in synovial tissue may induce β_2 -M deposition and organization in fibrillar structure.

In order to evaluate further this hypothesis, we studied tissue removed during surgical carpal tunnel decompression by performing on the same sample both a spectro-photometric analysis of Al concentration and a histological examination. Amyloid deposits were diagnosed in the presence of the Congo-red-induced green birefringence and positive staining for β_2 -M by an immunoperoxidase method. The study was undertaken in 17 dialyzed patients (mean dialytic age 172 ± 39 months, range 72–240). Control samples were taken during surgery of 22 non-uremic patients with carpal tunnel syndrome.

Fig. 1. Al concentrations in tissue removed during surgical carpal tunnel decompression in control patients (A) and in dialyzed patients with (■) and without (#) β_2 -M amyloid deposits. The results confirm that Al concentration is significantly higher (44 ± 24 vs. 13 ± 9 $\mu\text{g/g}$, $p < 0.01$) in dialyzed patients in comparison to control patients (fig. 1). However, no differences are shown in Al concentration in dialyzed patients with ($n = 10$) and without ($n = 7$) β_2 -M amyloid deposits in the excised tissue (45.2 ± 25 vs. 42.2 ± 25 $\mu\text{g/g}$). No amyloid deposits were found in control samples.

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Both Al body burden and β_2 -M deposition may correlate with the duration of dialysis, and this fact must be taken in account when a relationship between the two factors is evaluated. However, our results do not seem to confirm a direct role of local Al accumulation at the site of carpal tunnel tissue in the development of amyloid deposits in dialyzed patients.

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