Aluminium and Dialysis Associated Arthropathy

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Dear Sir,

We read with a great interest the letter of Canavese et al. [1] on aluminium and dialysis arthropathy. The authors have discussed some results published by our group in 1988 in a Lancet Letter [2] and we would like to reply.

The results of Canavese et al. confirmed that aluminium accumulates in synovial tissue of patients receiving long-term dialysis. However, in our study [2], we compared the concentrations of aluminium in synovial tissue and joint cartilage samples from 28 patients who had been receiving dialysis for an average of 123 months with those from controls of the same age who had not been given drugs containing aluminium, all of whom were operated on for carpal tunnel syndrome or arthropathy of the limbs. Aluminium concentrations were about 10-fold higher in the hemodialysed patients than in controls. In patients receiving dialysis the concentrations (expressed as mean $\pm$ SEM) were 17.4 ± 3.6 $\mu\text{g/g}$ wet weight in synovial tissue and 20.2 ± 6.6 $\mu\text{g/g}$ in cartilage. The respective values in control patients were 1.7 ± 0.4 and 2.4 ± 0.4 $\mu\text{g/g}$. The values of Canavese et al. in 17 hemodialysed patients (44 ± 24 $\mu\text{g/g}$) and in controls (13 ± 9 $\mu\text{g/g}$) are surprisingly higher than our values, particularly for the control patients, but we do not know which tissues (synovial tissues, ligament, etc.) were analysed.

In our study we reported that amyloid deposits were found in 23 of 28 patients and that the highest concentrations of aluminium were in patients with β_2 -microglobulin amyloidosis. Canavese et al. [1] found no difference in tissue aluminium concentrations in hemodialysed patients with β_2 -microglobulin amyloid deposits (45.2 ± 25 $\mu\text{g/g}$) and without (42.2 ± 25 $\mu\text{g/g}$). They conclude that their results did not confirm a direct role of local aluminium accumulation at the site of carpal tunnel tissue in the development of amyloid deposits in dialysed patients. However, the role of aluminium in the development of amyloid deposits cannot be demonstrated in a study of this type because the longer the duration of hemodialysis the more probable is the accumulation of aluminium and β_2 -microglobulin.

The physiopathology of dialysis associated arthropathy is certainly multifactorial (β_2 -amyloidosis, patient's age, dialysis membrane, duration of dialysis, hyperparathyroidism, microcrystals, iron, aluminium, etc.). Aluminium is toxic to various tissues, particularly nervous and bony tissues [3] but its role in the pathogenesis of dialysis associated arthropathy is unclear. Studies in animals have shown the inflammatory effect of aluminium. The accumulation of

aluminium might be simply a thesaurismosis favoured by aging, tissue destruction, or amyloid deposits, but it is possible that the presence of aluminium favours the accumulation of β_2 -microglobulin [4, 5] or at least that there is a synergy between aluminium and the β_2 -microglobulin fragments which cause amyloid deposits to form joint and bony abnormalities [5].

References

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