Bone Lead and Renal Failure

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Dear Sir,

Winterberg et al. [1] recently reported on higher bone lead levels ($\mu\text{g/g}$ wet weight) in a small group of hemodi-alysis patients as compared to two small groups of patients with chronic renal failure or after renal transplantation. These results were reported as a reply to the observations of Martegani et al. [2], who found increased erythrocyte zinc protoporphyrin IX levels in hemodialy-sis patients as compared to chronic renal failure patients. These authors also found important amounts of chelat-able lead in the ultrafiltrate by treatment with EDTA during hemofiltration in patients normally under dialysis. They concluded that lead body burden depends on renal function and is increased in hemodialysis patients as compared to chronic renal failure or healthy subjects [1,2].

These findings are not in agreement with our findings published in 1988. Indeed we could not find any correlation between the degree of renal failure and bone lead by measuring bone lead/gram wet weight and lead/calcium ratio in transiliac bone biopsies of 35 patients with moderate degree of renal failure and 153 dialysis patients [3]. Levels in dialysis patients with well-documented analgesic nephropathy ($n = 10$) and no occupational lead exposure were in the same range as those from deceased subjects (cadavers) with past normal renal function and without clearly documented lead exposure (table 1).

Winterberg et al. [1] give no information on the occupational or environmental lead exposure in their hemodialysis group. Indeed, we found [3] high levels of bone lead

Table 1. Mean transiliac bone lead and bone lead/calcium ratio (mean $\pm$ SD)

not only in lead workers ($30.1 \pm 13.8 \mu\text{g/g}$ wet weight) but evenso in 8 dialysis patients (dialysis top 5%; $20.6 \pm 2.7 \mu\text{g/g}$ wet weight), 6 of them reporting occupational lead exposure or exposure to lead-contaminated water supply. The observed effect of slightly higher bone lead levels in the dialysis group, as reported by Winterberg et al. [1], could be the result of an increased past exposure to lead in these patients rather than the contribution of end-stage renal failure/dialysis to excessive lead body burden.

On the other hand, bone lead should be expressed as lead/calcium ratio, rather than $\mu\text{g/g}$ wet weight. This compensates not only for demineralization, known to occur during chronic renal failure and hemodialysis, but also for the non uniform distribution of lead throughout the skeleton [4]. Tibial bone, for instance, contains more lead than iliac bone [3].

High levels of chelatable lead in the ultrafiltrate of dialysis patients, as found by Martegani et al. [2], could either be the result of increased bone turnover (for instance hyperparathyroidism), rendering more lead available for exchange between the bone and plasma compartment during chelation therapy, and even more likely by altered pharmacokinetics of EDTA in patients with end-stage renal failure [5]. We should regret that based on very limited not well-controlled data the question of lead overload depends on renal function should erroneously be answered by yes. We have clearly shown in a substantial group of dialysis patients that renal failure per se does not play a role in the accumulation of lead.

References

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