ALUMINIUM-INDUCED HIP FRACTURES: A HYPOTHESIS

BENGT MJÖBERG

The age-specific incidence of hip fractures has increased epidemically during the last four decades. The fragility of bone in the elderly has been associated with decreased mineral content, the cause of which is not known. There is an increased incidence of fragility fractures in patients with previous gastric surgery (Alffram 1964), in alcoholics (Nilsson and Westlin 1973) and in smokers (Rundgren and Mellström 1984), but no aetiological factor common to these three groups has been demonstrated. Fatigue fracture due to aluminium intoxication osteomalacia is well known in patients on chronic dialysis, and the aluminium content in trochanteric bone biopsies was therefore measured in patients with hip fractures.

Patients and Methods. Biopsies were taken from the trabecular bone of the greater trochanter at operation in 20 patients (seven men and 13 women) with hip fractures (14 cervical and six trochanteric). The aluminium and calcium content in the bone ash was measured by atomic absorption spectrophotometry (Frech et al. 1982).

Results. All specimens contained aluminium: the Al/Ca ratio varied from 0.9 to $12 \times 10^{-6}$. There was no correlation between the aluminium content and sex or type of fracture, but there was a tendency ($p < 0.05$) for a higher aluminium content in the younger patients.

Discussion. The increased acid pollution in industrialised countries since the 1950s has led to one of the greatest ecological changes in historical time; an expression of this is the extensive forest decline in Central Europe. In an acid environment, aluminium dissolves and becomes poisonous: pied flycatchers breeding close to acidified lakes, and thus feeding on insects containing aluminium, produce defective mineralisation of eggshells because of reduced calcium stores (Nyholm 1981).

The toxic effect of aluminium in man became obvious during the 1970s, when a number of patients on chronic dialysis were dialysed with tap water purified with aluminium sulphate (Parkinson et al. 1979) or consumed large quantities of aluminium hydroxide to control hyperphosphataemia (Skinner et al. 1983). Some patients developed fatigue fractures due to renal osteodystrophy (aluminium intoxication osteomalacia) as aluminium was incorporated in the mineralisation front and inhibited bone mineralisation.

It is possible that the increase in age-specific incidence of hip fractures, as well as other fragility fractures, may be caused by chronic low-grade aluminium intoxication — the greater the aluminium exposure, the greater the risk of an early fracture.

The increased incidence of fragility fractures in patients with previous gastric surgery, in alcoholics, and in smokers may thus be explained by an above-average consumption of antacids, whereas the epidemic of fragility fractures in industrialised countries may be a consequence of an increased aluminium exposure due to the acidification of our environment.

The chemical analyses were performed by Wolfgang Frech, PhD, Department of Analytical Chemistry, Umeå University, Sweden.

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REFERENCES


