THE BONE SCAN IN PATIENTS WITH ALUMINIUM-ASSOCIATED BONE DISEASE

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Summary

The bone scans of 32 patients on regular dialysis who received desferrioxamine therapy for fracturing osteomalacia secondary to aluminium intoxication are reviewed. All scans show the same pattern, with lack of tracer deposition in bone and deposition in soft tissues. Therapy with desferrioxamine controlled the aluminium intoxication in all cases, and in 21 patients the bone scan reverted to normal or showed a pattern typical of hyperparathyroidism.

Introduction

The value of radioisotope bone scans for the diagnosis of renal osteodystrophy was first described in 1976 by Olgaard [1]. The few reports which have appeared since that time [2] maintain the quantitative and not the qualitative interpretation of this diagnostic technique. Almost simultaneously, Drüeke [3] and ourselves [4] have supported the usefulness of this technique for differentiating between fracturing osteomalacia secondary to aluminium intoxication and osteitis fibrosa due to the secondary hyperparathyroidism of regular dialysis patients.

In secondary hyperparathyroidism there is excessive deposition of the tracer in bone, both in long and short bones. There is no deposition outside bony structures (Figure 1A).

In osteomalacia, the scan pattern has completely opposite characteristics, with almost no deposition of the tracer in the bones, which are difficult to see and show an ‘erased’ appearance, and heavy deposits in non-bony tissues both in the thorax and in the abdomen, so that the full silhouette of the body with the legs and calves becomes visible (Figure 1C).

Between these two extreme patterns, A and C, there is an intermediate one in which excessive deposits of the tracer in the facial bones and the cranial vault coexist with deposits in the thorax and abdomen. This pattern is found in patients who have high serum values of both aluminium and PTH.
Pattern N in Figure 1 is a normal bone scan in a haemodialysis patient. The present study evaluates the clinical, analytical and X-ray data of patients with a type C bone scan, corresponding to aluminium intoxication, before and after therapy with desferrioxamine.

Patients

The study group comprised 60 patients under regular dialysis treatment at two different dialysis Units. The first Unit used a softener from 1968 to 1980, at which time a mixed bed ionic resin water processing system was installed; the second Unit used a softener from 1972 to March 1982, then installing a reverse osmosis system.

Thirty-nine patients had a type C bone scan, but only 32 received desferrioxamine therapy, as only these patients had clinical osteomalacia; 16 of them were being treated at Unit 1, and 16 at Unit 2. the remaining, 28 patients, nine from Unit 1 and 19 from Unit 2, formed the control group.

All patients treated with desferrioxamine presented bone pain, more or less marked proximal myopathy and type C bone scan. Eleven of them had costal fractures, three had femoral neck fractures, and two dialysis dementia.

Methods

The serum aluminium was assayed in a flameless atomic absorption spectrophotometer with a graphite furnace; the PTH was determined by a C-terminal-
fragment-sensitive radioimmunoassay. The bone scans were carried out with a
gamma camera after intravenous injection of 10–15mCi of $^{99m}$Tc labelled
diphosphonate (MDP). The statistical analysis was performed by non-parametric
methods, using the Mann-Whitney ‘U’ test and the Wilcoxon ‘T’ test; the chi-
square method was also used for some data.

Desferrioxamine was given at a dosage of 1g, dissolved in 100ml normal
saline and infused via the arterial line over the first two hours of each haemo-
dialysis session, three times weekly, for six consecutive months; this treatment
was started in January 1983.

Aluminium and PTH values and bone scans were recorded immediately before
starting desferrioxamine treatment, and again one month after this therapy was
stopped.

Results

Prior to the institution of desferrioxamine therapy, the comparison of the data
of the 39 patients with type C bone scan and the 21 patients in the control
group yielded the following differences: the patients with type C bone scans had
higher serum aluminium values, $88.63 \pm 74.74$ versus $39.58 \pm 27.85\mu g/L
(p<0.001); they had lower PTH values, $4.72 \pm 3.00$ versus $15.09 \pm 10.54\text{ng/ml}
(p<0.001), and they had been undergoing dialysis treatment for longer, $49.33 \pm
36.07$ versus $30.24 \pm 35.18$ months (p<0.05).

Under desferrioxamine therapy the clinical symptoms of osteomalacia and
dialysis dementia disappeared, all fractures healed, and the EEG studies showed
considerable improvement in both patients.

Figure 2 shows the changes in serum aluminium both in the group treated
with desferrioxamine and in the one without. In the treatment group the initial

![Figure 2](image-url)
values are very high, with a mean value of 98.04 \( \mu g/L \) and a large standard deviation, as there are individual values of up to 398 \( \mu g/L \). After treatment the mean value was 34.60 \( \mu g/L \) with a small standard deviation, and of course with a statistically highly significant difference. The control group had practically unchanged values, 46.01 \( \mu g/L \) at the beginning of the study and 47.80 \( \mu g/L \) at the end.

All patients in the treatment group had a type C bone scan, whereas only seven patients in the control group had this pattern. After treatment, only 11 patients still have the type C pattern (\( p < 0.01 \)), which persisted in five patients of the control group (\( p \) NS) (Figure 3).

Figure 3

Figure 4 shows the changes in serum PTH in the treatment and control groups before and after treatment. The initial values show a large difference between the groups; the patients who were treated with desferrioxamine were near normal, 4.30 ± 2.79 versus 12.97 ± 17.30 ng/ml (\( p < 0.001 \)). Six months
later, the control group showed no significant changes, while those of the treatment group have risen to 6.20 ± 4.47 ng/ml (p<0.05).

This change in PTH explains why, in some cases, the bone scan did not revert towards normal but to type A, characteristic of hyperparathyroidism (Figure 5).

Discussion

The bone scan with $^{99m}$Tc MDP reflects the calcium turnover in bone [5]; it is therefore logical that in situations of secondary hyperparathyroidism there is excessive uptake of the tracer by the bones [1] which results in the type A pattern. In osteomalacia the situation is fully reversed, the deposition of calcium salts in the bone is slowed and therefore the binding of MDP will be less and slower. The skeleton of the patients is barely visible or not at all, giving rise to the type C pattern. In this pattern, the deposition of the tracer delineating the soft tissues, or as intense deposits in the thorax and abdomen, may be due to two causes: either it shows simply the presence of the tracer in the circulating blood, or it really reflects an excessive deposition of calcium in the viscera and soft tissues.

Akrill, in 1980, first reported the treatment of aluminium intoxication with
desferrioxamine [6]; other authors have later confirmed it [7] and also point out the appearance of increased plasma PTH as the aluminium is eliminated from the body. In our patients, therapy with desferrioxamine improves the clinical, biochemical and X-ray findings of aluminium intoxication, but at the same time induces higher serum PTH. Coincidentally, the bone scans change from the type C pattern (osteomalacia) either to type N (normal) or to type A (hyperparathyroidism). In our opinion, all these data prove that the type C pattern is caused by aluminium intoxication, although it could be a reflection of the low calcium turnover in bone and might therefore exist in any other form of osteomalacia in patients under regular dialysis treatment.

References

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