

Osteomalacia from Al and Mg antacids

Report of a case of bilateral hip fracture

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Presented is a case of bilateral spontaneous fracture of the femoral neck in a 40-year-old woman who suffered from severe phosphate depletion due to excessive intake of antacids containing magnesium and aluminum. Attention is drawn to this rare cause of osteomalacia, which can be easily treated.

Drugs containing magnesium and aluminum are used in the treatment of dyspepsia, and in addition to this, in dialysis to prevent ectopic calcification and osteitis fibrosa (Baker et al. 1974); uremia is often associated with hyperphosphatemia. Also patients with dialysis encephalopathy, which is caused by high concentrations of aluminum in the gray matter of the brain (Alfrey et al. 1976), will often suffer from osteomalacia, possibly because aluminum is deposited in the calcification front (Alfrey 1984).

Aluminum and magnesium can also cause osteomalacia by inducing phosphate depletion and a negative calcium balance, as in the case reported here.

Case report

After constant pain in the left hip with no preceding trauma, a 40-year-old woman presented with a subcapital fracture of the left femoral neck. The fracture seemed to be in the initial stages of healing. The serum phosphate level was low, but the reason for this could not be determined. The patient was known to have a long psychiatric history, including drug abuse, but denied being under any concurrent medical treatment. The patient also complained of muscle soreness and fatigue, and the diagnosis remained that of idiopathic osteomalacia. Mobilization with weight bearing was prescribed, but 6 months later, an identical fracture was found in the right femoral neck, and the fracture on the left side had failed to heal. Osteosynthesis with three Richards cannulated hip-pins was performed in both

hips. During hospitalization the patient complained of dyspepsia, and a duodenal ulcer was diagnosed, for which the patient claimed to have never received medical treatment.

However, it was discovered that she had bought large amounts of the magnesium- and aluminum-containing antacid Alminox[®] for her own use. For several months, an average of 120 tablets per day were purchased, corresponding to 12 g of magnesium oxide and 60 g of aluminum aminoacetate. These purchases were confirmed through the internal records of the pharmacy.

Laboratory examinations (normal values) were as follows: serum Ca 2.42 mmol/L (2.2–2.7 mmol/L); serum P 0.55 mmol/L (0.8–1.6 mmol/L); Urinary Ca 13.6 mmol/L (3.8–5.0 mmol/L); urinary P 0.02 mmol/L (25–65 mmol/L); and alkaline phosphatase (primary bone fraction) 1,007 U/L (80–275 U/L); finally, 25-calciferol, 1,25-cholecalciferol, and serum PTH were normal.

After the abuse had been stopped and normal serum phosphate restored, the fractures healed without complication.

Discussion

It is well known that osteomalacia may be caused by excessive intake of magnesium and aluminum (Dent and Winter 1974), but only 1 case of fracture caused by abuse of these drugs has been reported (Bloom and Flinchum 1960).

The biochemical background has been studied by Lotz et al. (1964) using human volunteers who gradually were phosphate-depleted. Magnesium and aluminum binds inorganic phosphate in the gastrointestinal tract, resulting in decreased serum and urinary phos-

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phate. The metal hydroxide, when binding phosphorus in the gut, releases calcium from a poorly soluble complex with phosphorus, thus leading to hypercalciuria. The phosphate complex is then excreted in the stool. It was shown that resorption of calcium and phosphorus from the bones took place during the depletion. Characteristically, with normal parathyroid function, serum calcium remains normal, but the calcium balance becomes negative.

In most cases, termination of the intake of the magnesium and aluminum, and increased ingestion of phosphate, either in milk or in the form of 15-30 mL phosphosoda three times daily, will restore normal serum levels (Janson et al. 1983).

Patients with fractures in the process of healing or osteoporotic patients should not be treated with these antacids without supplementary phosphate (Lotz et al. 1968).

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