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RADIOLOGIC VIGNETTE

FLUOROSIS AND OSTEOMALACIA

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Clinical history

A 30-year-old taxicab driver presented with a 1-year history of low back pain radiating into the posterior aspect of both legs, stiffness during inactivity, and proximal upper and lower limb myalgia. Symptoms were poorly controlled with acetaminophen. The medical history was unremarkable. The patient was a vegetarian, smoked 5 cigarettes per day, and did not drink alcohol. He was born in the Punjab region of southern Asia and had lived there until moving to the United Kingdom at the age of 25 years. Physical examination findings included globally restricted movement of the lumbar spine, sacroiliac joint tenderness, grade 4/5 proximal muscle weakness, and inability to rise from a chair with folded arms. Neurologic findings were otherwise normal.

Serum biochemistry studies revealed the following: alkaline phosphatase 164 units/liter (normal <125), elevated bone isoenzyme levels, alanine transaminase 68 units/liter (normal <40), normal levels of bilirubin and gamma glutamyl transpeptidase, corrected calcium 2.42 mmoles/liter (normal 2.15–2.55), phosphate 0.65 mmoles/liter (normal 0.75–1.5), parathyroid hormone 6.5 pmoles/liter (normal 1.1–6.5), and 25-hydroxyvitamin D 14 nmoles/liter (normal 20–100).

Radiologic findings

Radiographs revealed no features of osteomalacia, but diffuse sclerosis and calcification of both entheseal and joint capsules were seen (Figures 1 and 2). These findings were characteristic of fluorosis. Serum and urine fluoride levels were thus measured; these levels were 146 μg/liter (normal 6–45) and 0.7 mg/liter (normal <1.6), respectively. Bone mineral density in the lumbar spine (L1–L4) was 2.284 gm/cm^2, with a T score of +9.20 and a Z score of +8.87.

The radiologic differential diagnosis of diffuse bone sclerosis included osteopetrosis, mastocytosis, myelofibrosis, prostatic metastases, renal osteodystrophy, hypoparathyroidism, and certain hemoglobinopathies, particularly sickle cell disease. The radiologic differential diagnosis of entheseal calcification included seronegative spondylarthropathy, diffuse idiopathic skeletal hyperostosis, calcium pyrophosphate deposition arthropathy, hypoparathyroidism, and X-linked hypophosphatemic osteomalacia.

Transiliac bone biopsy (Figures 3 and 4) revealed increased cancellous bone volume (36.5%; age-matched

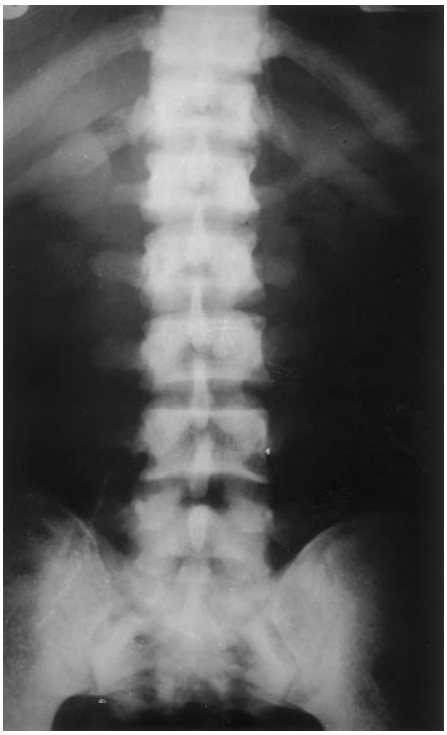

Figure 1. Radiograph of the lumbar spine and sacrum, showing diffuse bone sclerosis.

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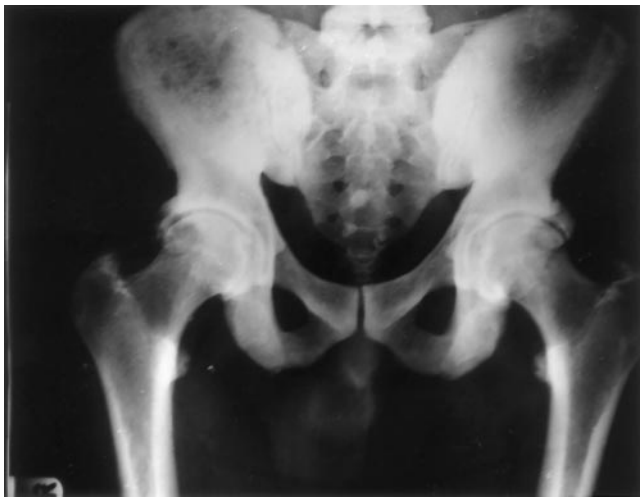

Figure 2. Radiograph of the pelvis and proximal femora, showing diffuse bone sclerosis and entheseal calcification at the greater and lesser femoral trochanters and the hip capsules.

normal <23%), areas of woven mineralized bone, increased bone surface covered by osteoid (40%; age-matched normal <15%), and increased osteoid thickness (average 6–12 lamellae; age-matched normal <3); most tetracycline- and oxytetracycline-stained surfaces showed single labels (results not shown).

Diagnosis: Osteomalacia with asymptomatic fluorosis

The clinical improvement suggests that the presenting symptoms were largely attributable to osteomalacia. However, any residual spinal rigidity is likely due to the effects of endemic fluorosis.

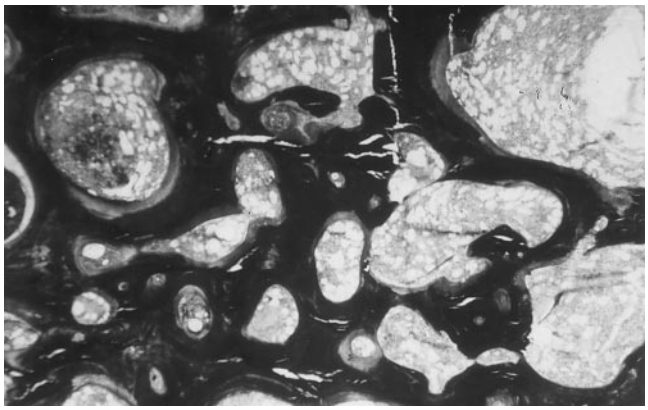

Figure 3. Toluidine blue–stained transiliac bone biopsy specimen, showing increased cancellous bone volume, increased bone surface covered by osteoid, and increased osteoid thickness.

Figure 4. Transiliac bone biopsy specimen under polarizing light, showing areas of woven mineralized bone.

Discussion

The serum biochemistry results in this patient, as well as the increased bone surface covered by osteoid, increased osteoid thickness, and single-labeling of most stained surfaces indicated a mineralization defect consistent with osteomalacia. The radiologic findings, bone mineral density measurements, increased cancellous bone volume, and areas of woven mineralized bone were consistent with previous exposure to fluoride. The normal urinary excretion of fluoride and the absence of woven osteoid suggests that the effects of fluoride were no longer active, despite the increased serum fluoride concentration.

Diet was the most likely source of fluoride in the patient, from endemic exposure in Punjab. Other recognized sources include industrial exposure during the manufacture of aluminum, steel, and glass, and exposure to the dust of fluoride-containing rock (1). The main effect of the fluoride ion is to stimulate new bone formation, which is mainly woven in character and imperfectly mineralized. The clinical effects are spinal rigidity and flexion deformities of joints, often due to entheseal, ligament, and tendon mineralization.

The patient was treated with oral vitamin D_2 (400 IU daily), resulting in a marked diminution of the back pain, a return of muscle strength, and normalization of serum alkaline phosphatase, phosphate, parathyroid hormone, and 25-hydroxyvitamin D levels over the subsequent 8 months.

REFERENCE

1. Smith R. Disorders of the skeleton. In: Weatherall DJ, Ledingham JGG, Warrell DA, editors. Oxford textbook of medicine. 3rd ed. Oxford: Oxford University Press; 1996. p. 3055–97.