REVIEW PROF-598

OSTEOMALACIA;
VALUE OF PLAIN RADIOGRAPHY

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ABSTRACT

In this study, osteomalacia is discussed in detail. The plain film radiography is at its best when ever the lesions related to bones are reviewed. We will discuss the proper path of osteomalacia, with a brief emphasis on the physiology and pathology of the disorder so that the review is interesting for everybody. All the radiographic findings discussed in this pictorial are the original work of the authors and are done mostly in the Department of Radiology, K E Medical College/Mayo Hospital Lahore. Five cases are discussed, which were diagnosed as osteomalacia on the evidence available on plain radiography.

INTRODUCTION

While discussing osteomalacia[1,2,3] and its pathology, it is very difficult to leave out rickets, the real time brother of osteomalacia. Rickets[4] and osteomalacia are two diseases characterized by osteopenia[5].

Although these lesions are associated with a variety of entities, the underlying abnormality is due to deficiency of active form of vitamin D secondary to various causes[6,7,8] or to an alteration in calcium or phosphorus metabolism.

Bone formation is also regulated by parathyroid hormone, vitamin D and calcitonin[8,9]. An understanding of these substances is always helpful in appreciating the mechanism of bone formation[1,10].

PATHO-PHYSIOLOGY

Parathyroid hormone is a polypeptide secreted by the parathyroid gland, it is the most important regulator of extracellular calcium concentration . In the bones, it inhibits the osteoblasts and osteoclastic bone reabsorption is activated. The major function of parathyroid hormone in the kidney is tubular reabsorption of calcium and impedance of phosphate reabsorption. Another vital renal function of the parathyroid hormone is the activation of hydroxylase enzyme to form 1,25-dihydroxy vitamin D[11,12], the most effective form of the vitamin D. Recent studies have also shown that where vitamin D and parathyroid hormone act in consort, they increase calcium absorption from the gut.

Active vitamin D is the end result of a complex metabolic pathway that begins with ultraviolet radiation of 7-dihydrocholesterol in the skin. This substance is converted to cholecarciferol or vitamin D3 a few days

following the radiation. Vitamin D2 is a compound structurally similar to cholecarciferol that is artificially created by radiating the compound ergosterol found in yeast or fungi. When ingested as a food supplement it proceeds through the same metabolic pathways as endogenous vitamin D3. Two successive hydroxylations are necessary to form the most active hormone, 1,25 dihydroxy vitamin D. The initial hydroxylation occurs in the liver (25 OH vitamin D). The final hydroxylation occurs in the kidney. The enzyme needed for the later process is found in the kidney and requires the presence of parathyroid hormone.

The production of vitamin D is influenced by body needs and is regulated by serum calcium, phosphate and parathyroid hormone levels. Calcitonin, corticosteroids, growth hormone and thyroid hormones are probably of lesser importance. Lowered serum calcium levels results in the increased production of active vitamin D. The mechanism of action is elevation of parathyroid hormone circulatory levels, which prompt the kidneys to produce increased amounts of active vitamin D, biologic feedback control is effected by 1,25(OH2) vitamin D, which depresses parathyroid hormone by increasing serum calcium levels and inhibiting the parathyroid gland[13].

Calcitonin secreted from the C cell of the thyroid gland acts by inhibiting parathyroid bone reabsorption, thereby decreasing plasma calcium levels.

ETIOLOGY

From all the discussion above it must be now clear that what is the physiological pathway of occurrence of rickets and its adult form, the osteomalacia. this far in the osteomalacia, then the etiology might be good enough to be discussed over here. We refer to Table-I.

Table-I. Etiology of osteomalacia.

Vitamin D deficiency	25 (OH) Vitamin D Deficiency	1,25 $(OH)_2$ Vitamin D deficiency	Calcium/phosphorus imbalance
Gastrointestinal malabsorption	Liver disease	Renal osteodystrophy	Phosphorus loss
	Anticonvulsant therapy	-	Renal tubular dysfunction

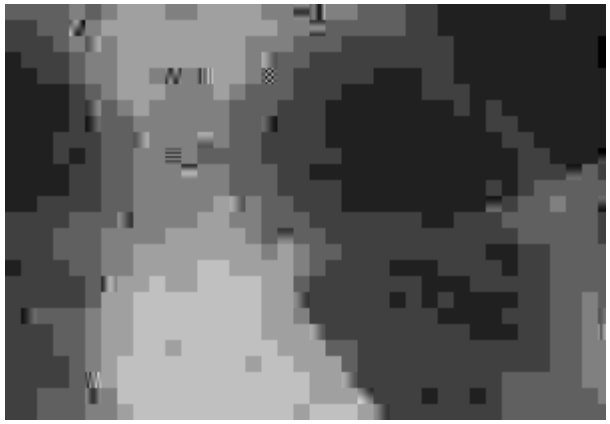

Figure-1. Looser zone, visible in the posterior end of 2nd rib.

The diagnostic features

Now we come to the real point that what are the specific features due to which we label the patient as having osteomalacia. In osteomalacia the bones loose their calcium, i.e. they become progressively demineralized. The cortex becomes thinned out. It must be differentiated from the other causes of osteopenia such as osteoporosis. The most specific feature of osteomalacia is the presence of looser transformation zones, also referred to as Milkman's pseudo fractures or Umbauzonen. These zones appear as radiolucent defects several millimeters thick that extend perpendicular to the cortex and are frequently symmetrical. The defects are more prominent in the outer areas of the cortex but can extend to the endosteal surface.

Figure-2. Pelvic x-ray of the same patient note the compressed pelvis and psedufractures.

Dialysis-induced osteomalacia associated with aluminum deposition is uncommon but nevertheless a significant problem[10].

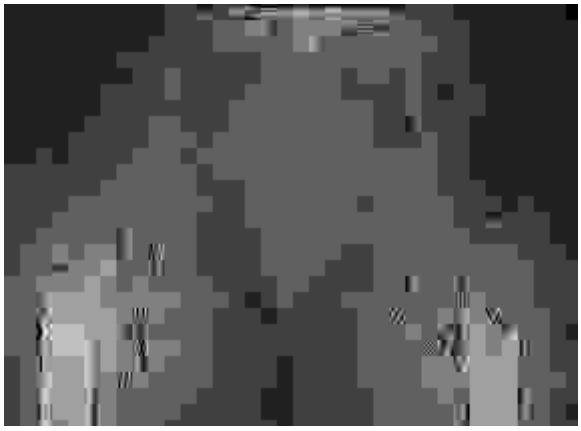

Figure-3. Pelvic x-ray of another patient, with similar findings.

The radiologic and histologic features are characteristic. The mechanism is still unclear. Aluminum may interfere with orderly deposition of bone by forming an insoluble complex with phosphates and inhibiting the precipitation of calcium apatite. Aluminum may be given to patients as oral aluminum hydroxide gels in order to bind phosphates in the gut or added to dialysate water in order to flocculate organic materials. In patients with renal disease, aluminum tends to accumulate in the body tissues.

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