REVIEW ARTICLE

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Radiographic dilemma of four systemic bone diseases: rickets, osteomalacia, renal osteodystrophy, and hypophosphatemic rickets (literature review)

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ABSTRACT

Objectives: Rickets, osteomalacia, renal osteodystrophy, and hypophosphatemic rickets are systemic diseases that have the same characteristics, namely the condition of bone softening or weak bone conditions. This condition is caused by low levels of calcium and phosphorus in the bones. As a result, the bones become of poor quality, which can be seen histologically in the form of a picture of bone osteoid that is less calcified. However, the etiology of the causes of abnormalities in these four diseases is different, and in particular, the picture in the radiograph certainly has its own character.

Review: The purpose of writing this article is to learn more about the special characteristics of these four systemic diseases. The writing method used is a narrative literature review. This article will discuss the four types of systemic diseases, starting from the definition, clinical appearance, appearance in radiographs, and the characteristics of each type. The results of the characterization that has been carried out indicate that, in general, radiography and other imaging methods are indeed difficult to distinguish because they have the same symptoms, but at the beginning of the case, the picture can be distinguished.

Conclusion: The conclusion that can be drawn from this article is that between rickets, osteomalacia, renal osteodystrophy, and hypophosphatemic rickets, there are specific characteristics that are the main key in recognizing each abnormality, especially radiographically, that can be recognized.

Keywords: *Osteomalacia, renal osteodystrophy, rickets, hypophosphatemic rickets*
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INTRODUCTION

Disorders of the jawbone caused by systemic diseases often serve as manifestations of underlying conditions that affect bone metabolism, soft tissue health, or inflammatory processes within the body. Systemic diseases can influence the normal shape and function of bones and the formation of teeth. Bone disorders induced by systemic diseases produce abnormalities through imbalances in serum concentrations of calcium and phosphate, and/or through abnormal function of osteoblasts, osteocytes, and osteoclasts.[1]

A disease caused by decreased serum calcium levels will alter the balance of calcium and phosphate levels, resulting in abnormal bone and tooth formation. Low serum calcium concentrations can also lead to the mobilization of calcium from bones, as well as a decrease in calcium levels within the bone.[1]

Mineral and bone metabolism disorders such as rickets, osteomalacia, renal osteodystrophy, and hypophosphatemic rickets are conditions often associated with underlying systemic disorders, such as vitamin D deficiency, calcium and phosphate imbalances, or chronic kidney disease. These conditions affect the bone mineralization process, including the jawbone, leading to characteristic changes in bone structure and impacting oral cavity function as well as overall dental health.[2]

Although the pathogenesis of each condition differs, the resulting radiographic features tend to exhibit similar patterns, such as decreased bone density, irregular trabeculae, thinning of the cortical bone, and the presence of radiolucent lesions in the jawbone area. This similarity presents diagnostic challenges for practitioners, particularly in distinguishing these conditions based solely on radiographic findings.[3]

Rickets and osteomalacia, for instance, are caused by vitamin D deficiency, which leads to impaired mineralization of the bone matrix, both before (rickets) and after (osteomalacia) the closure of the growth plates.[1] Meanwhile, renal osteodystrophy is a metabolic complication of chronic kidney disease, with secondary

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hyperparathyroidism triggering bone resorption. On the other hand, hypophosphatemic rickets, which is often genetic in nature, disrupts phosphate homeostasis, thereby worsening bone mineralization.[3]

In a clinical context, these radiographic similarities complicate the identification of the specific causes of bone disorders in the jaw, which can impact treatment decisions and prognosis for the patient. Therefore, it is important to review the existing literature to better understand this radiographic dilemma and seek improved diagnostic approaches to differentiate these conditions.

This literature review aims to explore the similarities and differences in radiographic features of rickets, osteomalacia, renal osteodystrophy, and hypophosphatemic rickets, as well as to identify additional parameters that may help improve diagnostic accuracy.

REVIEW

RICKETS AND OSTEOMALACIA

A synonym for rickets and osteomalacia is calcipenic rickets. Rickets and osteomalacia result from insufficient serum and extracellular levels of calcium and phosphate, minerals essential for the normal calcification of bones and teeth. This deficiency manifests as inadequate calcification of osteoid in bone formation and hypocalcification of dental enamel and dentin. Both disorders arise from defects in the normal metabolism of vitamin D, particularly $1,25(OH)_2D$, which is necessary for calcium absorption in the intestines.[1]

Rickets is a term typically used when the disease affects growing bones in infants and children, whereas osteomalacia refers to the condition when it affects the skeletal system in adults. Most patients with osteomalacia experience varying degrees of bone pain and muscle weakness.[1]

The clinical symptoms commonly observed in rickets and osteomalacia are typically characterized by widespread bone pain, particularly in the lower back, pelvis, and legs; proximal muscle weakness leading to a "waddling" gait; pseudofractures (small fracture lines resulting from weakened bones); and bone deformities (in severe cases or those associated with rickets).[4]

The earliest and most prominent feature of rickets and osteomalacia is the widening of the epiphyses of long bones. This represents a manifestation of a broad, unmineralized osteoid layer observed histologically. The resulting abnormal biomechanics lead to concavity and fissuring of the metaphyses of long bones. Weight-bearing bones, such as the femur and tibia, exhibit characteristic bowing. Greenstick fractures (incomplete fractures) occur in many patients with rickets.[1,4]

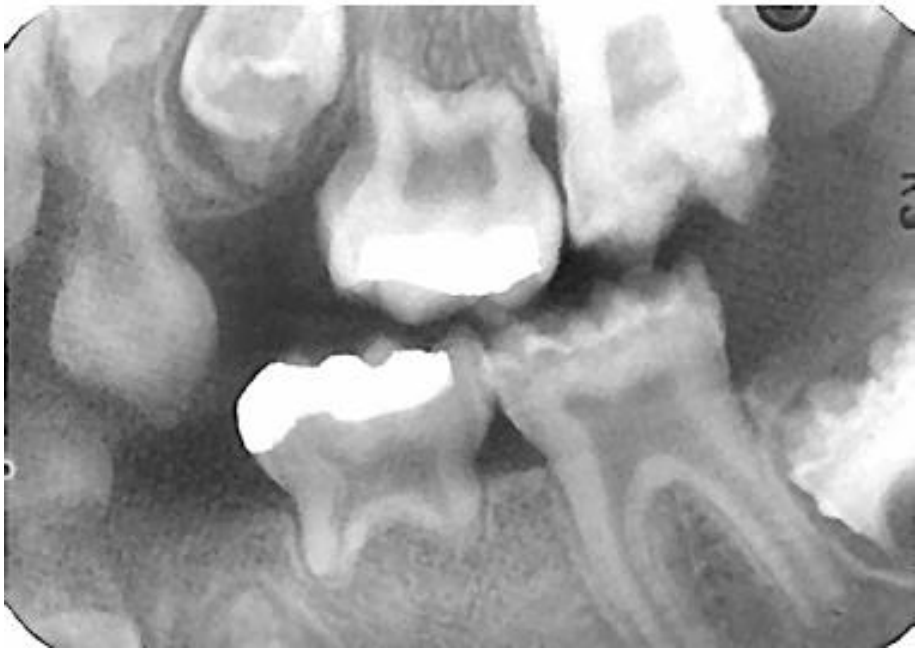

Figure 1. Rickets decreases mineralization (Source: Book of C. White S, J. Pharoah M. WHAITE PHAROAH - Oral Radiology - Principles and Interpretation 7 Edition)[1]

In osteomalacia, the cortical bone may appear thinned. Pseudofractures, which are band-like zones of poorly mineralized bone extending into the bone at right angles to its margin, may also be present. Pseudofractures most commonly occur in the ribs, pelvis, and weight-bearing bones, and are rarely observed in the mandible.[1]

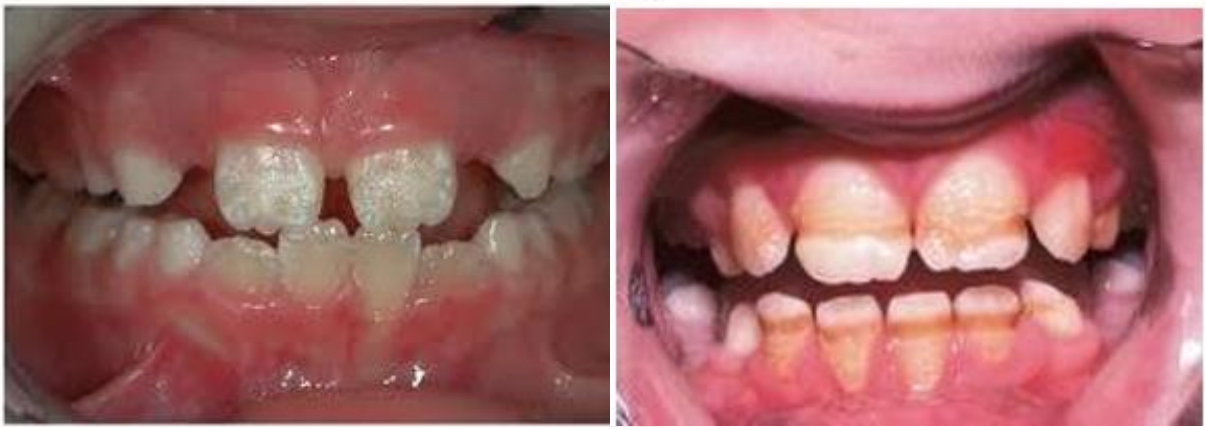

Figure 2. Enamel hypoplasia and tooth discoloration in rickets patients (Source: Journal of Dental Manifestation of Generalized Enamel Hypoplasia and the Calcium Role Due To Environmental Vitamin D Deficiency in Rickets and Osteomalacia Patients)[5]

In patients with rickets, changes in the jaw typically occur after alterations in the ribs and long bones. The cortical structure of the jaw, such as the inferior border of the mandible or the walls of the mandibular canal, may appear thinned. Within the cancellous portion of the jaw, the density, number, and thickness of trabeculae are reduced. This results in generalized radiolucency of the jaw; in severe cases, the jaw may appear so radiolucent that the teeth seem to lack bony support. Additionally, enamel hypoplasia is often observed, leading to tooth sensitivity, delayed tooth eruption, and tooth discoloration.[1,5]

Most cases of osteomalacia do not present manifestations in the jaw. However, when such manifestations appear in diagnostic imaging, they may present as generalized radiolucency and sparse trabeculation.[1]

RENAL OSTEODYSTROPHY

A synonym for renal osteodystrophy is renal rickets. Renal osteodystrophy (ROD) is a common complication of chronic kidney disease (CKD). ROD is a mineral metabolism disorder that occurs due to impaired kidney function. It is often associated with secondary hyperparathyroidism, calcium and phosphate imbalances, and vitamin D deficiency, all of which affect bone formation, including the jawbone.[2]

Chronic kidney failure leads to bone alterations by impairing the hydroxylation of 25(OH)D to $1,25(OH)_2D$, a process that takes place in the kidneys. The various biological functions of $1,25(OH)_2D$, particularly intestinal calcium absorption, are consequently hindered. This results in hypocalcemia and hyperphosphatemia. The imbalance in serum calcium and phosphate levels disrupts the normal calcification of teeth and bones. In addition to affecting bone and tooth formation, low serum calcium levels stimulate the parathyroid glands to secrete parathyroid hormone (PTH), leading to secondary hyperparathyroidism.[1]

Based on medical history, clinical examination, and radiographic findings, a diagnosis of renal osteodystrophy (ROD) with oral manifestations is established. Blood tests reveal low calcium levels, elevated parathyroid hormone (PTH), anemia, and increased alkaline phosphatase levels, consistent with secondary hyperparathyroidism and chronic kidney failure.[6,7]

Renal osteodystrophy can cause various oral manifestations, including Jawbone alterations, such as jaw enlargement due to increased osteoclastic activity. Dental abnormalities, including delayed eruption, enamel hypoplasia, tooth mobility, and malocclusion. Soft tissue changes, typically presenting as gingival inflammation, xerostomia, and a characteristic uremic odor. Secondary hyperparathyroidism in renal osteodystrophy leads to bone changes such as osteitis fibrosa and osteomalacia, which are commonly observed in patients with end-stage renal disease.[2,6,7]

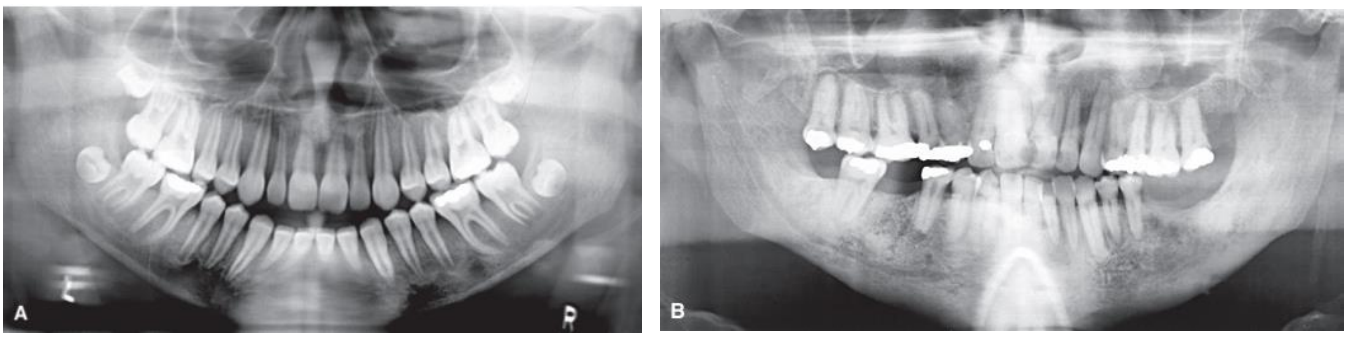

Figure 3. Two Cases of renal Osteodystrophy (Source: Book of C. White S, J. Pharoah M. WHAITE PHAROAH - Oral Radiology - Principles and Interpretation 7 Edition)[1]

The characteristic radiographic features of renal osteodystrophy typically include reduced bone density of the mandible and maxilla, often accompanied by thin cortical margins. In some cases, jawbone density may be greater than normal. Increased bone density is associated with a higher bone turnover rate, leading to an increased number and thickness of bone trabeculae. Altered trabecular patterns may also result in a granular bone appearance. Jaw enlargement has been reported in patients with kidney disease, with the increased size attributed to the expansion of the cancellous bone component. Teeth and Related Structures: Dental hypoplasia and hypocalcification may occur, sometimes resulting in the loss of visible enamel on diagnostic images. The lamina dura may be absent or poorly defined in cases of bone sclerosis.[1,8]

HYPOPHOSPHATEMIC RICKETS

Hypophosphatemic rickets is a hereditary disorder of renal phosphate regulation that leads to hypophosphatemia and affects tooth formation, including dentin hypomineralization and enlargement of the pulp chamber. This condition often results in bone deformities and dental abnormalities.[9]

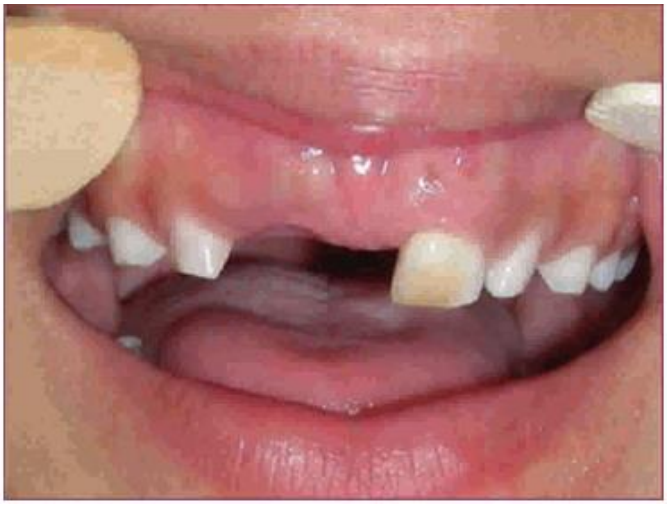

Figure 4. Clinical aspect of a patient with Hypophosphatemic Rickets (Source: Dental abnormalities and oral health in patients with Hypophosphatemic rickets)[9]

Hypophosphatemic rickets (HR) is also often referred to as a rare disorder characterized by inadequate bone mineralization due to abnormal renal phosphate wasting. This condition is caused by genetic mutations, most commonly X-linked dominant, involving the hormone FGF23 (Fibroblast Growth Factor-23), which regulates phosphate homeostasis. HR affects bone structures, including the cranial and facial bones.[2]

Another name for hypophosphatemic rickets is vitamin D-resistant rickets (phosphopenic type). Hypophosphatemic rickets represents a group of hereditary renal disorders that affect phosphate regulation, characterized by hypophosphatemia and normal or low levels of serum $1,25(OH)_2$ vitamin D and calcium. The failure to reabsorb phosphate in the distal renal tubules results in decreased serum phosphate levels (hypophosphatemia). Hypophosphatemia is associated with interglobular dentin and enlarged pulp chambers. Normal calcification of bone structures requires the correct amount and ratio of serum calcium and phosphate. Phosphate homeostasis is regulated by Fibroblast Growth Factor-23 (FGF23), parathyroid hormone (PTH), and vitamin D.[1,10]

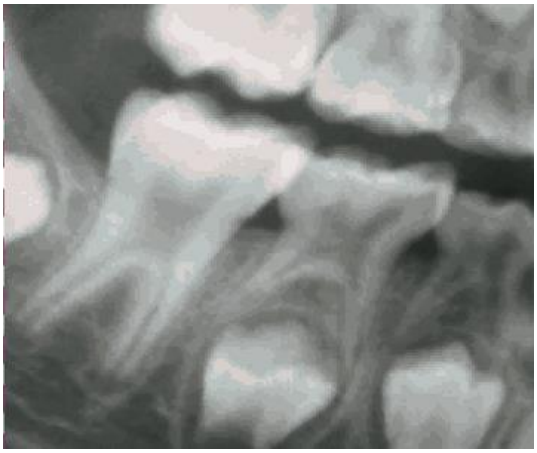

Figure 5. Radiological aspect of a patient with Hypophosphatemic Rickets (Source: Dental abnormalities and oral health in patients with Hypophosphatemic rickets)[9]

Dysfunction in any of these systems or renal tubulopathy can lead to hypophosphatemia. Hypophosphatemia may result in reduced bone density due to low calcium content, leading to abnormal trabecular formation, which can sometimes appear short and irregular, resulting in a granular bone pattern. Additionally, hypophosphatemia disrupts the normal calcification of dentin, leading to enlarged pulp chambers and root canals compared to normal structures.[11]

Hypophosphatemic rickets presents with variable clinical manifestations and often resembles other conditions, requiring a high index of clinical suspicion for accurate diagnosis. With a better understanding of its pathophysiology and the development of emerging therapies, the management of hypophosphatemic rickets has shifted toward a more evidence-based, targeted approach aimed at improving bone health and long-term patient outcomes.[11]

DISCUSSION

The earliest radiographic feature of rickets is characterized by the widening of the epiphyses of long bones. This finding represents a broad osteoid layer that remains uncalcified, as observed histologically. The resulting abnormal biomechanics lead to cupping and fraying at the metaphysis of long bones. Weight-bearing bones, such as the femur and tibia, often exhibit characteristic bowing. Additionally, greenstick fractures (incomplete fractures) are commonly observed in many patients with rickets.[1]

In osteomalacia, the cortical bone may appear thinner and is often accompanied by pseudofractures. These pseudofractures are band-like zones of poorly mineralized bone that extend into the bone at right angles to its edge. Pseudofractures most commonly occur in the ribs, pelvis, and weight-bearing bones, but are rarely observed in the mandible.[1,12]

In some cases of rickets, jaw changes typically occur after alterations in the ribs and long bones. The cortical structure of the jaw, including the inferior mandibular border and the mandibular canal wall, often shows thinning. Within the cancellous portion of the jaw, the density, number, and thickness of trabeculae are reduced, leading to generalized radiolucency of the jaw. In severe cases, the jaw may appear highly radiolucent, giving the impression that the teeth lack bony support.[1,12]

Most cases of osteomalacia do not exhibit manifestations in the jaw. However, when manifestations appear in radiographic images, they often show generalized radiolucency and thinned trabecular bone. In contrast, rickets in infants or children can lead to enamel hypoplasia in developing teeth.[12,13]

If the disease occurs before the age of 3, enamel hypoplasia is quite common. The early manifestations of rickets are visible in diagnostic radiographic images, involving both unerupted and erupted teeth. Radiographic images may also reveal delayed tooth eruption in early rickets. Additionally, the lamina dura and the cortical boundary of the tooth follicle may appear thin or lost.[1]

In some cases, osteomalacia does not affect the teeth because the teeth have fully developed before the onset of the disease. The lamina dura may be significantly thinned in individuals with long-standing or severe osteomalacia. The signs and symptoms of rickets may include delayed growth, delayed motor skills, pain in the spine, pelvis, and legs, and muscle weakness. Because rickets softens the growing tissue areas at the ends of a child's bones (growth plates), the disease may lead to skeletal deformities, such as bowed legs or knock knees, thickened wrists and ankles, and a protruding breastbone.[13,14,15]

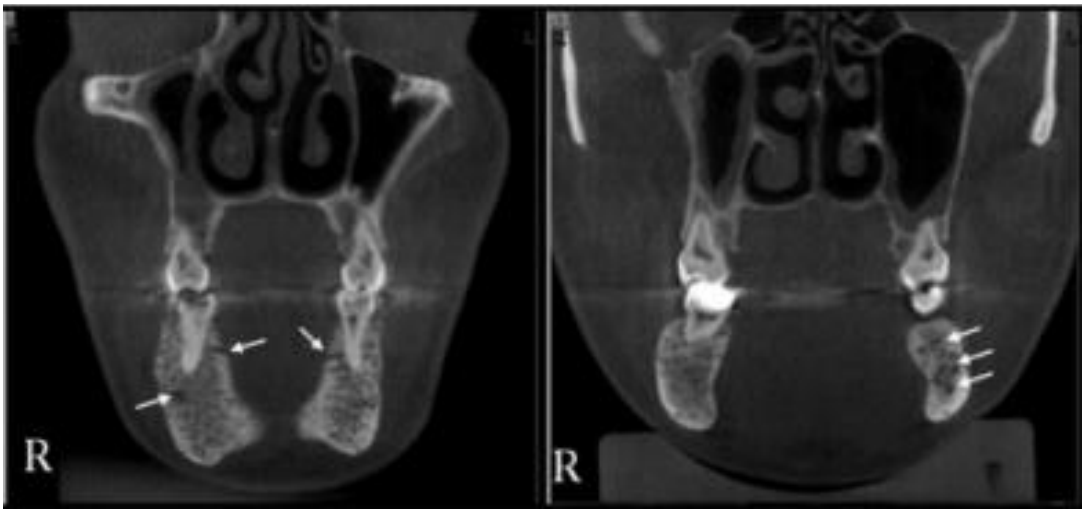

Figure 6. Osteomalacia; coronal cone beam CT image. Cortical and trabecular bone destruction (Looser's fracture) in the mandibular bone (white arrows) (Source: Journal of The importance of cone beam CT in the radiological detection)[15]

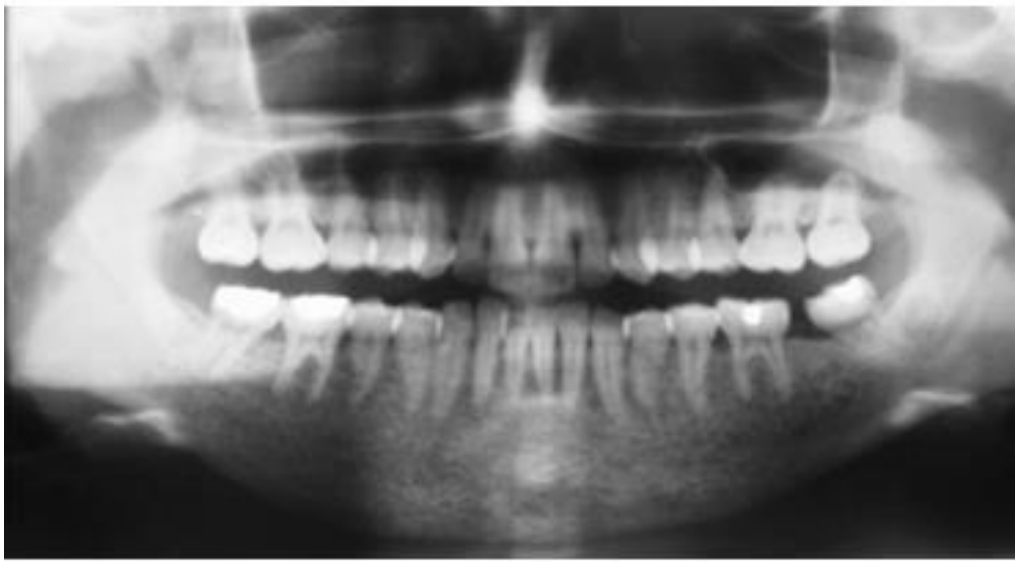

Figure 7. Conventional panoramic radiography of osteomalacia showed rarefaction and porosity in the jaws (Source: Journal of The importance of cone beam CT in the radiological detection)[15]

The radiographic features of renal osteodystrophy are generally highly variable. Some bone changes closely resemble those seen in rickets, while other changes are consistent with patients with hyperparathyroidism, typically showing widespread bone loss, thinning of cortical bone, and increased bone density, which is sometimes observed. Additionally, brown tumors with brown-colored lesions may also be present in affected patients.[1,15]

In renal osteodystrophy, the density of the mandible and maxilla is typically lower than normal, with thinner cortical bone regions. The jawbone is often larger than normal. The increased bone density is associated with an elevated bone turnover rate, resulting in an increased number and thickness of bone trabeculae. Altered trabecular patterns may also lead to a granular bone appearance. Additionally, the enlargement of the jaw is attributed to the expansion of the cancellous bone component.[8,16]

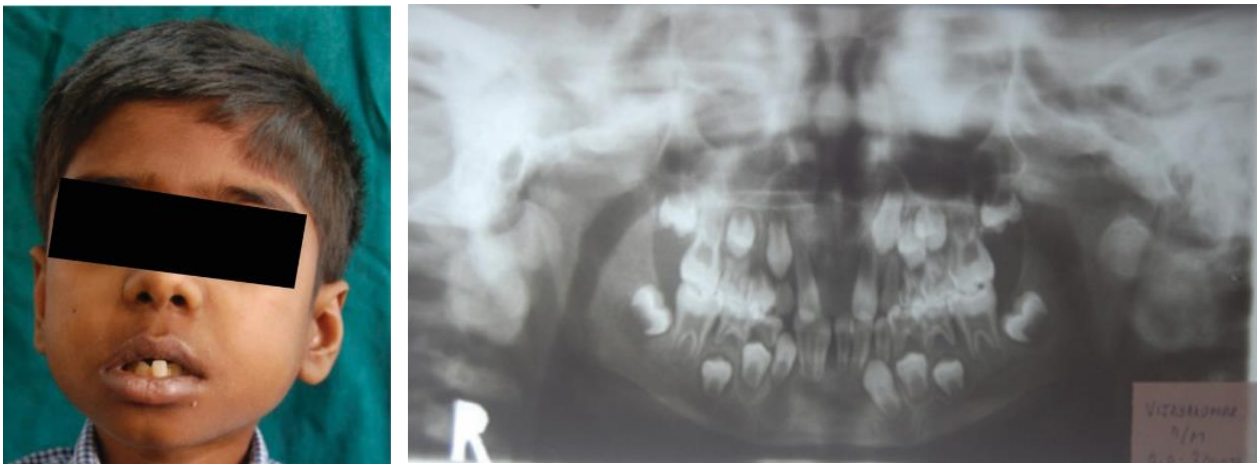

Figure 8. Clinical and radiographic findings in a patient with ROD (Source: Journal of Oral Manifestations in a Renal Osteodystrophy Patient)[16]

The characteristic and commonly observed radiographic manifestations in cases of renal osteodystrophy (ROD) include a "salt and pepper" pattern in the skull bones, loss of the lamina dura, and changes in the texture of bone trabeculae. Additionally, a ground-glass bone pattern, resembling that seen in fibrous dysplasia, may be present. In severe cases, significant jaw enlargement can occur, a condition known as uremic leontiasis ossea or "Sagliker syndrome".[8,15]

Hypophosphatemic rickets is a genetic disorder that causes bone metabolism abnormalities due to excessive phosphate excretion by the kidneys, leading to the body's inability to properly absorb phosphate, a mineral essential for bone and tooth formation. This condition impairs bone mineralization, resulting in growth disturbances, bone pain, deformities, and dental issues such as thin enamel and enlarged pulp chambers.[17,18]

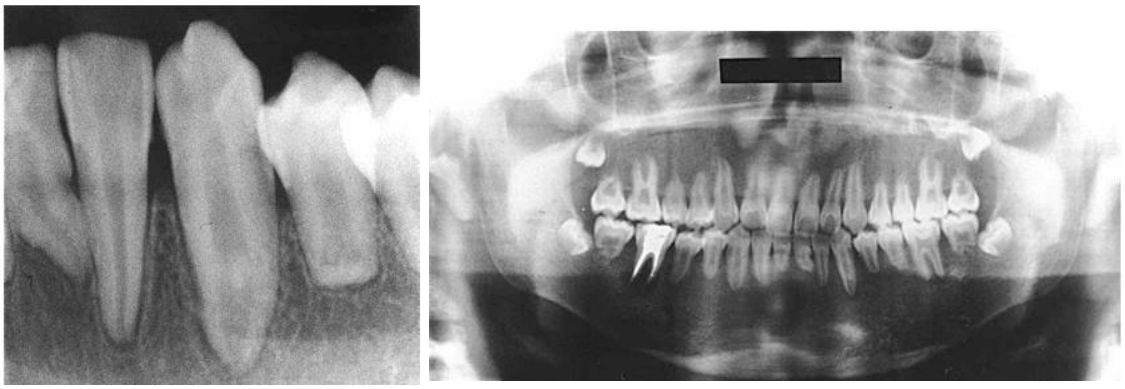

Figure 9. Clinical and radiographic findings in a patient with Hypophosphatemic rickets (Source: Journal of Familial hypophosphatemic vitamin D-resistant rickets: Dental findings and histologic study of teeth)[18]

Radiographic features in children with hypophosphatemic rickets differ from those of typical rickets. In adults, long bones may exhibit persistent deformities, fractures, or pseudofractures. In the jaw, radiographic findings commonly show areas of osteoporosis, and in extreme cases, the jaw may appear highly radiolucent. The cortical bone margins may be extremely thin or indistinct. Other manifestations include Sparse trabeculae with a granular bone pattern, resulting in reduced bone density in the jaw. Teeth may be underdeveloped, featuring a thin enamel cap and enlarged pulp chambers and root canals. Additionally, periapical and periodontal abscesses are frequently observed.[1,17,18]

Radiographic Features of Systemic Conditions Affecting Jaw Bone Density. Among all systemic conditions that result in reduced jaw bone density, radiographic images may reveal areas of both radiopaque and radiolucent bone density in the jaw. In advanced cases, several teeth may appear to lack proper bone support. Additionally, the cortical bone structure is typically observed to be thin and may sometimes be absent.[19,20]

Dental Changes Associated with Systemic Diseases Several systemic diseases occurring during tooth development can lead to dental alterations, characterized by thickening of the lamina dura, which merges with the periodontal membrane, forming part of the tooth structure. Changes in the teeth and related structures resulting from such systemic conditions may include Altered eruption timing (either accelerated or delayed), Enamel hypoplasia, and Loss of the lamina dura. Thus, early symptoms of systemic diseases may sometimes manifest in the teeth.[21,22,23]

CONCLUSION

The radiographic features of rickets, osteomalacia, renal osteodystrophy, and hypophosphatemic rickets are remarkably similar, as all four conditions represent manifestations of underlying mineral and bone metabolism disorders that affect the bone mineralization process. These conditions involve decreased bone mineralization due to imbalances in calcium, phosphate, or vitamin D levels. As a result, the bones appear less dense, exhibit radiolucent areas on radiographs, and display irregular trabecular patterns.

Since these three conditions affect bone mineralization in a similar manner, the radiographic features tend to exhibit comparable patterns, including mineral metabolism disorders that lead to decreased bone density, trabecular alterations, bone resorption, and disrupted tooth development patterns. Therefore, to differentiate these conditions, correlation with medical history, clinical examination, and laboratory tests is essential.

ACKNOWLEDGMENTS

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FOOTNOTES

All authors have no potential conflict of interest to declare for this article.

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