



Maxillofacial radiographic changes in renal osteodystrophy

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Abstract

The incidence of renal diseases continues to rise worldwide and as a consequence, increasing number of renal patients will probably require oral healthcare. Renal osteodystrophy refers to the wide spectrum of bone diseases in context to chronic renal diseases (CRD). Radiographic alterations affecting the jaws and facial skeleton are common and among the earliest signs of renal bone diseases. The aim of the present study is to review and highlight the various radiographic alterations affecting the maxillofacial region in renal osteodystrophy and enabling the general and specialty oral physicians to identify and diagnose them effectively.

Keywords: Renal osteodystrophy, Radiographic alterations, Oral health, Chronic renal diseases, Parathyroid hormone

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Introduction

Chronic renal diseases (CRD) are important health care problems throughout the world, with an increasing prevalence. Diseases of the kidney are a major cause of morbidity and mortality in both the developed and developing nations. CRD and their treatment can give rise to wide spectrum of systemic and oral manifestations. The management of such individuals requires that the clinicians must understand the multiple systems that can be affected (1).

Materials and Methods

This mini-review article discusses the pathophysiological mechanisms encountered the impact of chronic kidney disease on bone metabolism. For this review, we used a variety of sources by searching through Web of Science, PubMed, EMBASE, Scopus and directory of open access journals (DOAJ). The search was performed using combinations of the following key words and or their equivalents such as; renal osteodystrophy, radiographic alterations, oral health, CKD-mineral and bone disorder (CKD-MBD), chronic renal diseases and parathyroid hormone (PTH).

Renal osteodystrophy

Renal osteodystrophy (RO), also known as renal bone disease, refers to a spectrum of bone diseases, caused by a pathological alteration in metabolism of calcium, phosphate and bone in context to CRD and secondary hyperparathyroidism (2). Skeletal radiographic findings in RO include demineralization, brown tumors and osteosclerotic

changes in vertebrae. A spectrum of maxillofacial radiographic alterations can be observed in RO. Recognition of these manifestations in patients is important, since they may be the indicator of the presence or extent of the disease and therefore may be useful to the clinician in diagnosing the disorder, determining treatment requirements and assessing the prognosis of the disease (3).

The purpose of the present study is to review and highlight the various radiographic alterations affecting the maxillofacial region in RO. The study is projected to help general and specialty oral clinicians to recognize and diagnose typical maxillofacial radiographic alterations associated with RO. A proper radiographic examination of the oral cavity in patients with CRD is an invaluable aid to diagnose potentially life-threatening conditions at primary stages.

Discussion

Renal OD refers to the skeletal changes that result from CRD and are caused by disorders in calcium and phosphorus metabolism, abnormal vitamin D metabolism and increased parathyroid activity. Phosphate retention and decreased vitamin D conversion leads to hypocalcemia, which, in turn, stimulates the parathyroid chief cells to produce more PTH. PTH actions on bone normally increase the movement of calcium from bone into the extracellular fluid (ECF). They do this by stimulating the release of calcium across the bone membrane. PTH increases renal-tubular calcium reabsorption, mainly by an action on the distal convoluted tubule and connecting tubule. It reduces the proximal tubular reabsorption of phosphate,

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■ Implication for health policy/practice/research/medical education

The incidence of renal diseases continues to rise worldwide and as a consequence, increasing number of renal patients will probably require oral healthcare. Renal osteodystrophy refers to the wide spectrum of bone diseases in context to chronic renal diseases (CRD). Radiographic alterations affecting the jaws and facial skeleton are common and among the earliest signs of renal bone diseases.

thereby raising urinary phosphate excretion and lowering extracellular phosphate concentration. Long-term dialysis also inhibits healthy bone homeostasis, with aluminum deposition that interferes with bone mineralization. Subsequently, there are two major types of metabolic bone disease: high-turnover osteodystrophy (increased bone resorption and formation) and low-turnover or aplastic disease (adynamic bone and osteomalacia) (4).

Radiographic skeletal findings include generalized demineralization, sub-periosteal resorption (most frequently detected change and earliest detected change when affecting terminal phalanges), bone cysts, pathologic fractures, osteitis fibrosa cystica (brown tumors) and Rugger Jersey spine (osteosclerosis affecting the upper and lower margins of vertebral body) (3).

Radiographic alterations affecting the maxillofacial skeleton may be the earliest sign of CRD (2). The wide spectrum of maxillofacial osseous and dental radiographic alterations associated with RO is discussed in the following section.

Alterations in dental structures in renal osteodystrophy

While the skeleton may undergo decalcification, fully developed teeth are not directly affected; however, in the presence of significant skeletal decalcification, the teeth will appear more radiopaque (5). The following alterations can be observed in the dental structures in RO.

Obliteration and narrowing of pulp chambers

Narrowing or calcification of the pulp chamber of teeth of adults with chronic renal disease can occur (Figure 1). The exact cause of this dental change is not known. Renal allograft recipients have significantly more narrowing of the pulp chamber than those receiving hemodialysis (6). A strong correlation has been confirmed between the chronicity of the renal disease and the pulp narrowing in the premolar and the molar teeth of such patients. There is no consistent association between corticosteroid therapy and narrowing of the pulp chamber. It has been proposed that an early detection of the calcifications in these patients can provide life-saving information (7).

Teeth mobility and drifting

Deficient oral hygiene, uremic toxins and quantitative and qualitative changes of platelets predispose CRD patients to gingival changes ranging from severe gingivitis characterized by marked redness, swelling and tendency to bleed to periodontal pockets extending 3-6 mm apically to the

cemento-enamel junction (CEJ) increasing mobility and drifting of teeth with no apparent pathologic periodontal pocket formation may be seen in RO. Periapical radiolucencies and root resorption also may be associated with this gradual loosening of the dentition. The teeth may be painful to percussion and mastication (5).

Absence of lamina dura and widening of periodontal ligament space

Depending on the duration and severity of the disease, loss of the lamina dura may occur around one tooth or all the remaining teeth (Figure 2). The loss may be either complete or partial around a particular tooth (4). The result of lamina dura loss may give the root a tapered appearance because of decreased image contrast. A similar loss of lamina dura also may be seen in Paget disease, osteomalacia, fibrous dysplasia, sprue, Cushing and Addison diseases.

Delayed eruption of permanent teeth

Delayed eruption of permanent teeth has been reported in children with CRD (8).

Osseous manifestations of RO in maxillofacial region

CRD alter bone metabolism by multiple mechanisms. A wide range of maxillofacial bony anomalies can arise in CRD.



Figure 1. Narrowing and calcification of the pulp space in relation to 46

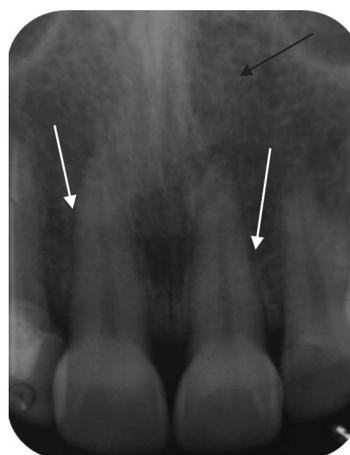


Figure 2. Absence of lamina dura and widening of periodontal ligament space in relation to 11 and 21 (white arrows). A change in the normal trabecular pattern is observed, resulting in a 'ground-glass appearance' (black arrow).

Demineralization of osseous structures and poor definition of anatomic landmarks

Demineralization and thinning of cortical boundaries often occur in the jaws in cortical boundaries such as the inferior border, mandibular canal and the cortical outlines of the maxillary sinuses (2,5). The density of the jaws is decreased, resulting in a radiolucent appearance (Figure 3). These changes appear most frequently in the mandibular molar region superior to the mandibular canal. The rarefaction in the mandible and maxilla is secondary to generalized osteoporosis.

Altered trabecular appearance

A change in the normal trabecular pattern may occur, resulting in a ‘ground-glass appearance’ of numerous, small, randomly oriented trabeculae. The finer trabeculae disappear later, leaving a coarser pattern (Figure 2). The mixed osteolytic and sclerotic changes of trabecula give the bone a ‘salt and pepper appearance’ (9).

Pathologic jaw fractures

Rarefaction in the mandible and maxilla is secondary to generalized osteoporosis (Figure 3). The compact bone of the jaws may become thinned and eventually disappear. This may be evident as loss of the lower border of the mandible. Decreasing thickness of cortical bone at the angle of the mandible correlates well with the degree of RO. Spontaneous and pathologic fractures may occur with the thinning of these areas of compact bone and may complicate dental extractions (5,6).

Abnormal healing of extraction sockets

Abnormal bone repair after extraction, termed “socket sclerosis” and radiographically characterized by a lack of lamina dura resorption and by the deposition of sclerotic bone in the confines of the lamina dura, has been reported in patients with RO (5,6) (Figure 4).

Metastatic calcifications

Metastatic calcification results when minerals precipitate into normal tissue as a result of higher than normal serum levels of calcium. Precipitation of calcium phosphate crystals occur into the soft tissues, such as the sclera, corner of the eye, subcutaneous tissue, skeletal and cardiac muscle and blood vessels (7). This also may occur in the oral and associated para-oral tissues. These calcifications are often visible radiographically (Figure 5).

Osteitis fibrosa cystica

The classic form presents with a combination of increased bone cell activity, peri-trabecular fibrosis, and cystic brown tumors. Radiographically, this appears as a constellation of cortical thinning of multiple bones, coarsened trabecular patterns and osteolytic lesions (Figure 6). The radiolucent lesions of hyperparathyroidism are called “brown tumors” because they contain areas of old hemorrhage and appear brown on clinical inspection. As these tumors increase in size, the resultant expansion may



Figure 3. Cropped panoramic image revealing demineralization of maxillo-facial osseous structures, resulting in decreased density of the jaws.



Figure 4. Abnormal healing of extraction sockets due to deposition of sclerotic bone in the confines of the lamina dura (black arrows).



Figure 5. Metastatic calcification seen mesial to 18 (black arrow).



Figure 6. Intra-oral periapical radiograph revealing of coarsened trabecular patterns (black arrows) and osteolytic lesions (white arrow), suggestive of osteitis fibrosa cystica.



Figure 7. Diffuse alterations of bony trabeculae with a ground glass pattern observed on conventional panoramic film with poor cortico-medullary distinction (black arrows).

involve the cortex. The brown tumor lesion contains an abundance of multinucleated giant cells, fibroblasts, and hemosiderin (4).

Bony changes resembling fibrous dysplasia

These alterations present with a classic ground glass pattern on both conventional films and computed tomography. Unlike true fibrous dysplasia, these findings can be diffuse and generalized, with poor corticomедullary distinction, an imaging finding not present in fibrous dysplasia (4) (Figure 7).

Uremic leontiasis ossea

It is characterized by significant hypertrophy of the jaws with serpiginous “tunneling” or channeling within the bone and poor visualization of the cortical bone. The cause of this unusual structure is not known; no specific patterns of microscopic changes explain the radiographic findings (4).

Thickening of diploic space of calvarium

In prominent hyperparathyroidism of RO, the entire calvarium has a granular appearance caused by the loss of central (diploic) trabeculae and thinning of the cortical tables. As a result, the diploic space of calvarium appears thickened radiographically (9) (Figure 8).

Conclusion

The accurate diagnosis of RO is essential to cope these potentially life-threatening conditions at a primary stage. An early detection of maxillofacial pathologies potentiates treatment to start sooner and may improve patient health and survival. A thorough knowledge of radiological alterations of maxillofacial region is important as these changes may serve as the earliest markers as well as disease severity indicators.

Authors' contribution

AK; Study design, preparation of manuscript and final revision. MK; Study design, manuscript edition and data interpretation.

Conflicts of interest

The authors declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

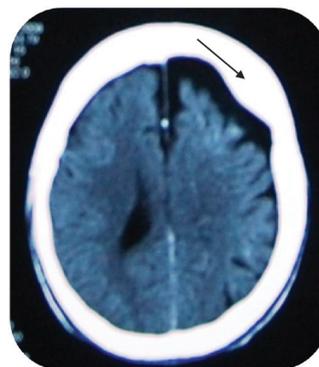


Figure 8. Thickened diploic space of calvarium (black arrow).

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