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Case Report

Case report– Periapical cemento-osseous dysplasia–CBCT findings

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ABSTRACT

Periapical cemento-osseous dysplasia (PCOD) is an uncommon benign entity, usually asymptomatic, wherein the normal bone is replaced by fibrous tissue, with metaplasic bone and neo-formed cement. The present case report describes a case of a 17-year-old female who was diagnosed with PCOD on the basis of cone beam computed tomographic (CBCT) findings. CBCT enabled detailed visualization of the bone changes. This report described the special radiographic characteristics of PCOD, including discontinuity of the lingual cortex, minimal amount of root resorption irt 32. Flecks of radiopacities interspersed diffusely within the lesion on the CBCT sectional and three-dimensional images.

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1. Case Report

A 17-year-old female presented to department of oral medicine and radiology for evaluation of radiolucency in relation to 32 region (Figure 1) from the department of orthodontics. Her medical was non-contributory. Dental history revealed that she had undergone orthodontic consultation and Orthopantamograph was taken 10 months back which revealed no evidence of any radiolucency (Figure 2). Patient had no pain, swelling, or trauma in 32 region. Clinical examination of mandibular anterior region revealed no abnormality with healthy gingiva and with 2–3 mm pocket depths. There was no evidence of swelling, bony expansion or tenderness. 32 responded positively to cold, heat and electric pulp testing. On CBCT examination, a mixed radiopaque radiolucent lesion noted in the periapical region 31 and 32 noted (Figure 3). As per the axial view, radiolucency seem to be involving the adjacent regions (Figure 4). Based on the features, working diagnosis was intermediate stage of PCOD of mandibular left anterior teeth. As a part of treatment protocol, patient counseling was done and advised to have regular follow-up and radiologic evaluations at every 3 months.

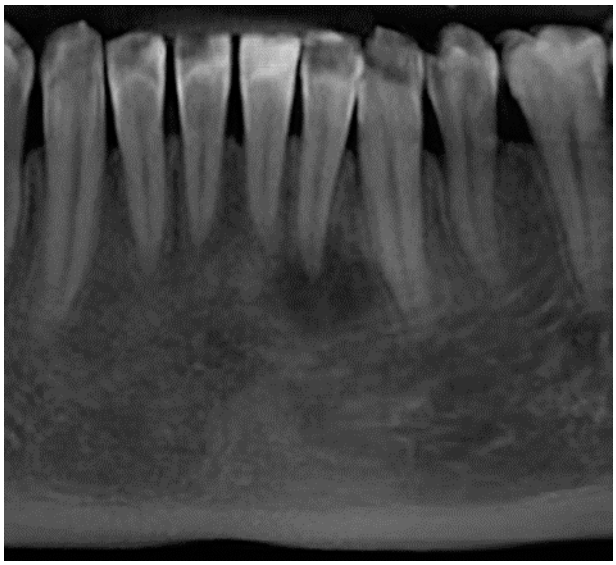

Figure 1: Cropped OPG showing radiolucency irt 32

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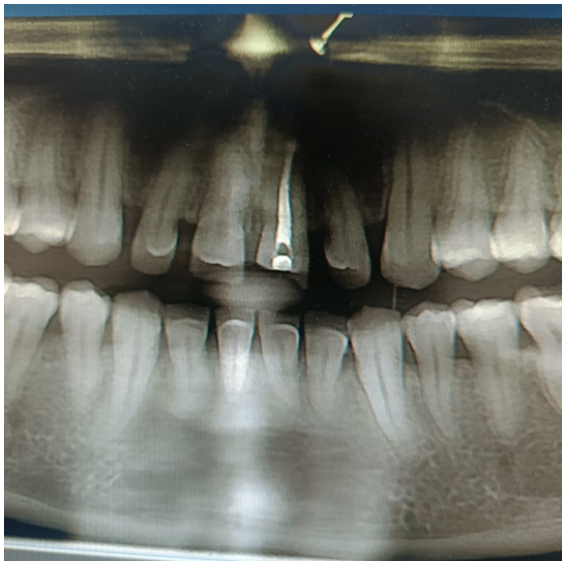

Figure 2: Cropped OPG shows no evidence of radiolucency irt 32

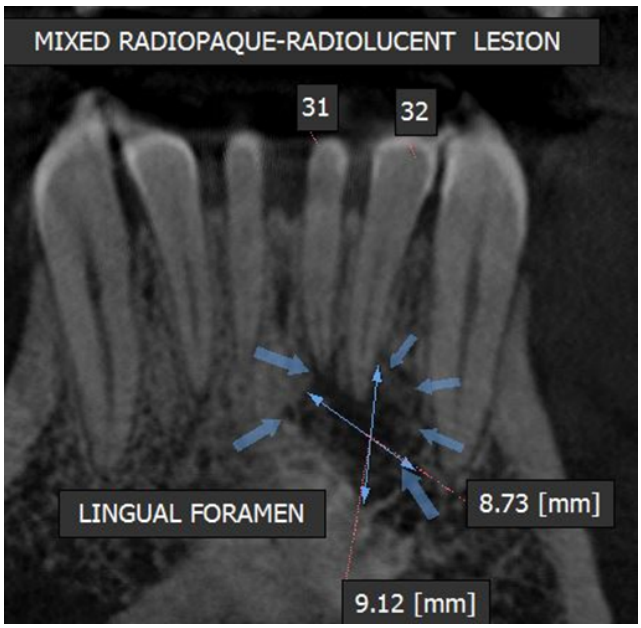

Figure 3: Coronal view

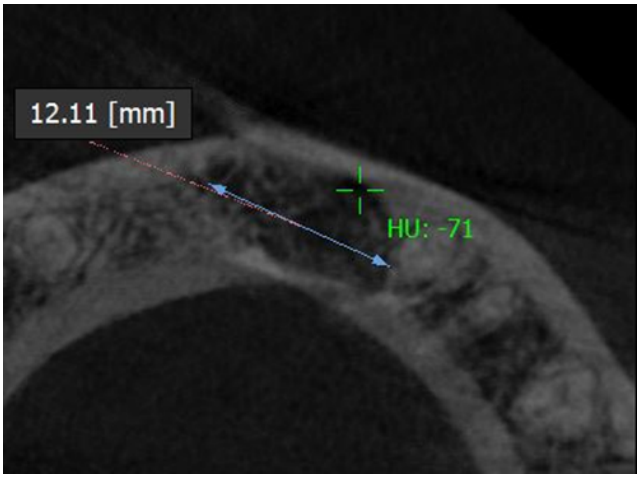

Figure 4: Axial view

2. Discussion

Periapical cemento-osseous dysplasia (PCOD) are categorised under Fibro-osseous lesions of the jaws (FOLs), a diverse group of processes that are characterized by fibrous tissue replacing normal bone and containing varying amounts of a mineralized substance.[1] As per the most commonly accepted classification by Waldron's, FOLs described as : (i) Fibrous dysplasia, (ii) Reactive (dysplastic) lesions and (iii) Fibro-osseous neoplasms. The World Health Organization (WHO) in 1992 quoted PCOD as periapical cemental dysplasia (PCD) (Kramer et al. 1992) and classified PCD as a type of cement-osseous dysplasia under non-neoplastic bone lesions in their histological typing of odontogenic tumours.[2]

Periapical cemento-osseous dysplasia arises from a group of disorders which are known to originate from undifferentiated cells of the periodontal ligament tissue.[3] In 1992, revised World Health Organization (WHO) guidelines, stated Cemento-Osseous Dysplasia as a form of neoplasm or other bone-related lesion; which is sub-divided into periapical cemental dysplasia (PCD), also known as periapical fibrous dysplasia, florid COD (also known as gigantiform cementoma or familial multiple cementoma) and other types.[3,4]

PCOD is a reasonably well-defined clinical-radiological entity, predominantly involving the apical areas of vital mandibular incisors. The term FCOD was recently recommended by Summerlin and Tomich has been previously described by different investigators under the designations of "osseous-dysplasia reaction of bone to injury" and "localized fibro-osseous cemental lesions" presumably reactive in nature.[5]

Hamilton BG Robinson (In 1956) coined similar lesions as periapical osseous dysplasia. Synonyms include periradicular fibrous dysplasia, periradicular cemental dysplasia, cementoma, periradicular osteofibrosis (Eleazer et al. 2012), periapical fibrous dysplasia (Kramer et al. 1992) and periapical osteofibrosis (Falace& Cunningham 1984).[4,6] It is found in review of the literature on COD Indicated that 59%, 37%, and 3% of blacks, Asians and Caucasians including Indian cases, respectively. Zegarelli et al reported the incidence of PCOD in the general population to be 2–3/1000 with middle age and female predominace.[7,8]

The etiopathogenesis of PCOD is still unknown. The commonly accepted hypothesis for the tissue of origin has been the periodontal ligament. Robinson (1956) stated that

occlusal forces induce tissue injury, initiating replacement of bony trabeculae with fibrous tissue leading to formation of cementum-like deposits and immature bone. [1]

Autosomal patterns of inheritance of familial PCOD cases have been reported by Young et al. (1989) and Thakkar et al. (1993). [9] The concomitant occurrence of osteomyelitis with cemento-osseous dysplasia (COD) has been reported in the literature. [10]

PCOD has been described as radiographically and histologically in three stages as per Sapp et al. 2002 [11]:

1. **Osteolytic stage :** Early stage is characterized by well-defined radiolucencies at the apex of one or more teeth. These radiolucencies mimic inflammatory periapical lesions of pulpal origin. Histologically, the tissue consists of cellular connective tissue replacing normal trabecular bone.
2. **Cementoblastic stage :** Is characterized by the presence of radiolucent areas containing nodular radiopaque deposits. Histologically, there is a mixture of spherical calcifications and irregularly shaped deposits of osteoid and mineralized bone.
3. **Mature stage:** Is characterized by well-defined, dense radiopacities surrounded by a radiolucent rim. The periodontal ligament can be seen separating the lesion from the root. [1,11]

Clinically the lesions may be single or multiple, always asymptomatic and common site of occurrence is the vital mandibular anteriors. Pulp vitality plays a vital role in arriving at a diagnosis of PCOD. Doppler flowmetry is recommended to evaluate blood flow in suspected teeth. CBCT provides valuable information for the endodontists about the relationship of the lesion to the involved root apices, expansion and/or thinning of the cortical plates and the status of the periodontal ligament and lamina dura. [1,7]

Radiolographic features depends on the phases of development. In the first or osteolytic phase, a circular radiolucent lesion is visible at the apex of the root; in the second or cement-blastic stage, the lesion shows a mixed appearance. The final or mature stage shows completely radiopacity. The radiolucencies surrounding the root apex are usually indistinguishable from inflammatory periapical lesions of pulpal origin. Histologically, the tissue consists primarily of cellular connective tissue replacing normal trabecular bone with calcified structures of insufficient size to be evident radiographically. Cementoblastic stage follows the initial lytic phase and is characterized by the presence of radiolucent areas containing nodular radiopaque deposits. Histologically, there is a mixture of spherical calcifications and irregularly shaped deposits of osteoid and mineralized bone. Late stage is characterized by well-defined, dense radiopacities usually surrounded by a radiolucent rim. The periodontal ligament can be seen separating from the roots. [1,12]

Final diagnosis is by patient's history, subjective symptoms, clinical findings and follow-up radiographic features. Though histologic and radiographic appearances are similar they require different treatment protocol. 'Keys to diagnosis' of PCOD includes: (i) Predilection for middle-aged black females; (ii) one or more (0.5 cm or less) circumscribed lesions in periapical areas of vital teeth; (iii) painless, non-expansile, usual location in anterior mandible; (iv) radiographic features can be radiolucent, mixed density, or opaque with radiolucent rim; and (v) cellular fibrous stroma with woven and/or oval calcifications. [1,13]

The differential diagnosis of PCOD depends on stages of the lesion. For early stage; apical periodontitis, periapical rarefying osteitis, for example as a periapical abscess, granuloma, or cyst are considered. For mature lesions– cemento-ossifying fibroma, Paget's disease of bone, chronic sclerosing osteomyelitis and cementoma. Cemento-ossifying fibromas (COF) has shown to occur in a younger population and lesions are larger in size with few patients exhibiting jaw expansion. The majority of cases do not show any relationship with teeth apices. Paget's disease of bone is more commonly seen in males, predominantly maxilla than mandible, with elevated serum alkaline phosphate and urinary hydroxyl-proline levels. Chronic sclerosing osteomyelitis being inflammatory disorder with cyclic episodes of pain and swelling and diffuse opacity with poorly defined borders. Cementoma appear as a well-defined radiopacity with a radiolucent rim associated with a tooth root showing signs of resorption. [3,12,14]

Though the lesion require no treatment, an accurate diagnosis is always essential. CBCT with the appropriate field of view and spatial resolution helps in assessing the location and extension of the lesion, internal mineralized structure, along with expansion and perforation of the cortical plates. [7]

3. Conclusion

This case highlights the significance of history, clinical examination, aided with CBCT for definitive diagnosis confirm the diagnosis. The combined analysis of these, with correct differential diagnosis, enables a diagnosis of periapical cemento-osseous dysplasia.

4. Source of Funding

None

5. Conflict of Interest

None

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