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CASE REPORT

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Periapical cemento-osseous dysplasia masquerading as asymptomatic chronic apical periodontitis in a Chinese woman: A case report

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Key Clinical Message

Cemento-osseous dysplasia (COD) belongs to a group of fibrous osseous disorders that can masquerade as periapical inflammatory conditions in the jawbones. We present a rare case of COD occurring in a patient who also had periapical periodontitis concurrently. When faced with a patient exhibiting no symptoms, diagnosis may be challenging.

Abstract

COD is a fibrous osseous disorder similar to periapical chronic inflammatory conditions and other cysts in the jaw bones on the radiograph. There is a rare case of COD occurring in a 49-year-old Chinese woman who also had chronic periapical periodontitis concurrently. The lesions were incidentally discovered in the mandibular anterior tooth region during the patient's imaging examination. The patient exhibited no symptoms, and diagnosing the conditions may be challenging due to the confusingly similar radiological features that present as a radiolucent lesion in the periapical region. The final diagnosis was made through pulp vitality tests, cone beam computed tomography (CBCT), and clinical examinations. The COD was recommended for annual follow-up. Root canal therapy has been performed for chronic apical periodontitis. According to the diagnostic process of COD, a thorough history check, multiple clinical examinations, and imaging studies should be emphasized to prevent misdiagnosis and avoid unnecessary or inappropriate therapies. The patient was followed up for 6 years. The recovery of chronic apical periodontitis in tooth #31 and the transformation of osteolytic and osteogenesis of COD had been observed in CBCT.

KEYWORDS

CBCT, cemento-osseous dysplasia, chronic apical periodontitis, periapical cemento-osseous dysplasia, pulp vitality tests, WHO classification

1 | INTRODUCTION

COD is a non-neoplastic, reactive, dysplastic, osseous fibrous lesion that occurs in the tooth-bearing areas of the jaws.[1,2] COD mostly remains asymptomatic. Rarely, the lesion can cause bleeding, pain, and drainage due to secondary infection.[3] Based on their radiological and clinical features, there are three subtypes: focal, florid, and periapical.[4,5] Focal cemento-osseous dysplasia (FCOD) is a solitary condition typically affecting the posterior mandible. However, florid cemento-osseous dysplasia (FLCOD) mainly occurs in two or more quadrants of jaw bones, often in a bilaterally symmetric fashion. Periapical cemento-osseous dysplasia (PCOD) is a common type of COD. It primarily affects the anterior mandible and involves single or multiple vital teeth. Radiologically, COD shows three representative features according to its process.[6] The early stage is a completely radiolucent lesion presenting as circular or oval areas of bone resorption. During the middle stage, a mixed radiolucent-radiopaque internal structure can be observed within the lesion. In the mature stage, the lesion becomes radiopaque. The initial stages of COD are similar to periapical radiolucencies.[7] Therefore, it is necessary to conduct a differential diagnosis based on clinical manifestations, advanced radiological examinations, and pulp vitality tests. Here, this case reported a PCOD of the mandibular anterior teeth in a 49-year-old Chinese woman. The patient's informed consent was obtained in this case.

2 | CASE HISTORY/ EXAMINATION

This 49-year-old Chinese female patient visited the Department of Stomatology at Shandong Provincial Hospital on May 18, 2018, for the extraction of impacted teeth. The panoramic tomography (Figure 1A) revealed a radiolucent lesion with a well-defined border located at the apex of tooth #32. The patient was then referred to the endodontic department for evaluation and treatment of the lesion.

After a detailed history, the lesion was asymptomatic. During the extraoral examination, no abnormalities were detected. During the intraoral examination, no abnormal oral mucosa or soft and hard tissue expansion was observed upon inspection. Caries and periodontal disease were also absent in the patient. Two small enamel defects caused by long-term external force in teeth #21 and #31 were found, which are known as the melon seed teeth (Figure 1A,B). Further percussion examination revealed mild discomfort in the left mandibular anterior teeth.

3 | METHODS

Puzzlingly, the pulp vitality tests revealed that all mandibular anterior teeth were vital, except for tooth #31. Because tooth #31 had no vitality and the presence of apical radiolucencies on tooth #32 in the panoramic tomography, intraoral periapical radiographs and CBCT scans were conducted to assess the apical lesions and internal structures more clearly. The intraoral periapical radiograph (Figure 2A) revealed a radiolucent lesion corresponding to the root of tooth #31, with a widened periodontal ligament space. The lamina dura surrounding the apical area of tooth #31 was lost. Further, CBCT could provide us with precise diagnostic information. The sagittal-sectional CBCT images (Figure 2B,C) showed that the continuity of the labial cortical plates was observed at teeth #32 and #33. However, erosion of the labial cortex of a lesion was found at tooth #31 (Figure 2D). On the coronal-sectional CBCT (Figure 2E,F), the lesions associated with the teeth #32, #33, and #41 were mixed radiolucent-radiopaque, whereas the lesion at the apex of the tooth #31 was radiolucent. Based on the patient's clinical and radiographic findings, the diagnoses of chronic apical periodontitis of tooth #31 caused by occlusal trauma and PCOD were made.

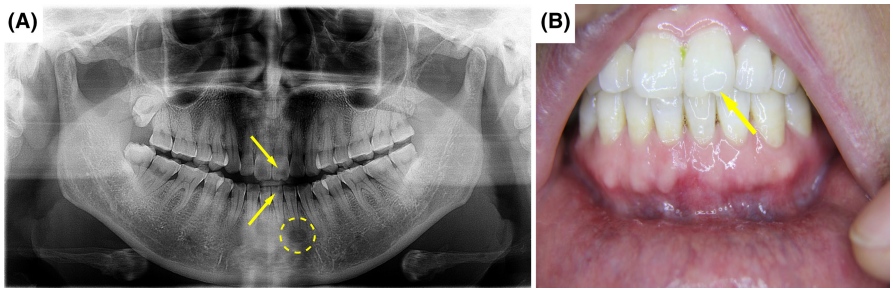

FIGURE 1 Panoramic tomography and preoperative photograph of the case on May 18, 2018. (A) Panoramic tomography shows a periapical radiolucent lesion linked to tooth #32 and the melon seed teeth of teeth #21 and #31 (the circle and arrows). (B) Preoperative photograph revealing the melon seed tooth of tooth #21 (an arrow).

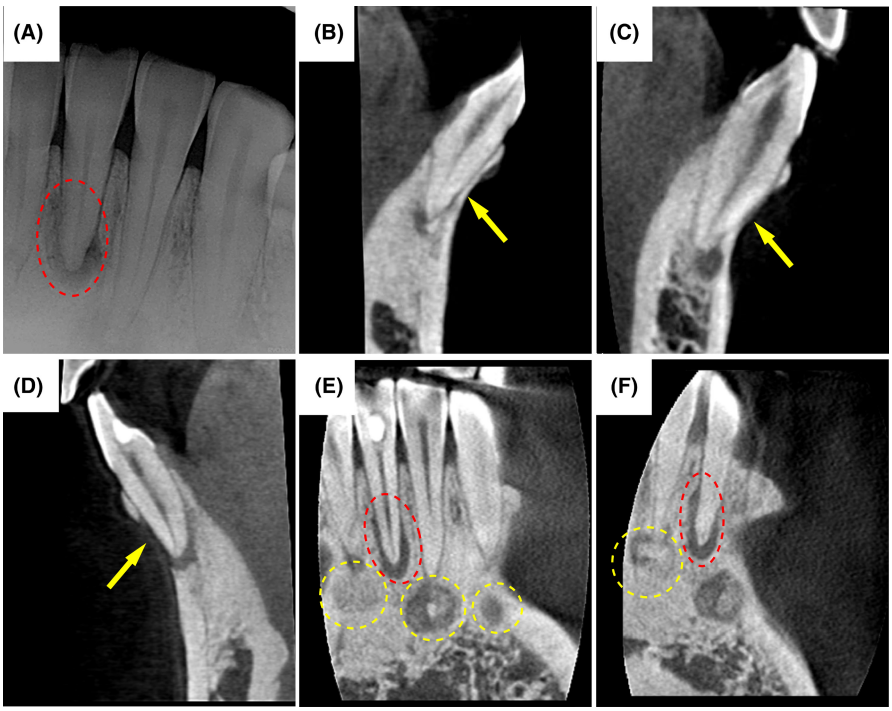

FIGURE 2 Intraoral periapical radiograph and cone beam computed tomographic images of the case on May 18, 2018. (A) An intraoral periapical radiograph reveals a radiolucent lesion surrounding the apical root of tooth #31 (a red circle). (B–D) CBCT sagittal-sectional images illustrating the relationship between the labial bone plate and the lesion around teeth #32, #33, and #31 (arrows). (E, F) CBCT coronal-sectional showing mixed radiolucent and radiopaque masses surrounded by a radiolucent halo in each tooth: #41, #32, and #33 (yellow circles), and a radiolucent lesion surrounding the apical root of tooth #31 (red circles).

3.1 | Recommendations

Root canal therapy was recommended on tooth #31, and periodic radiographic follow-up was recommended for PCOD.

4 | RESULTS

After a 6-year follow-up, there was no discomfort in the patient. No significant abnormality was found through oral examination. No abnormal oral mucosa or soft and hard tissue expansion was discovered (Figure 3A). CBCT showed no significant changes in the labial bone plates of teeth #32 and #33, but the labial bone plate of tooth #31 had become almost continuous and intact compared to the preoperative images (Figure 3B–D). In terms of bone remodeling, the previous translucent lesion of tooth #31 had disappeared, and the radiolucent lesion around the apical root of tooth #31 had been restored by radiopaque masses gradually. However, preexisting mixed radiolucent-radiopaque lesions surrounded by teeth #41, #32, and #33 had some osteolytic or osteogenic changes, which were not obvious. In short, the images illustrated the recovery of the lesion in tooth #31 after root canal treatment, with the intact labial plate and bone healing in the apical alveolar bone around tooth #31. PCOD had undergone some bone remodeling during the 6 years. We recommend continued follow-up for the patient.

5 | DISCUSSION

COD, a benign fibro-osseous lesion of the jaws, can resemble a radiolucent periapical inflammatory lesion when it develops during the osteolytic stage of its maturation.[7] It is reported that black middle-aged females have a predisposition to the condition.[8] The nomenclature and classification of COD have been controversial in almost every edition of the World Health Organization (WHO) classification of Head and Neck Tumors.[9] In 1971's WHO classification, periapical cemental dysplasia (periapical fibrous dysplasia) was categorized as a cementomatous lesion which also included cementifying fibroma, benign cementoblastoma (true cementoma), and gigantiform cementoma (familial multiple cementomas).[10] These lesions were previously thought to be associated with cementum and considered separate diseases. However, cemento-osseous dysplasias were classified into non-neoplastic bone lesions according to the 1992 WHO classification.[11] The condition was divided into periapical cemental dysplasia (periapical fibrous dysplasia), florid cemento-osseous dysplasia (gigantiform cementoma, familial multiple cementomas), and other cemento-osseous dysplasias based on age, gender, histopathologic, radiographic, and clinical features.[11,12] By analyzing the clinical, imaging, and pathological characteristics of 221 cemento-osseous dysplasia lesions, Summerlin and Tomich proposed a specific classification: focal COD, with the similar and differentiating features of periapical cemental dysplasia and florid

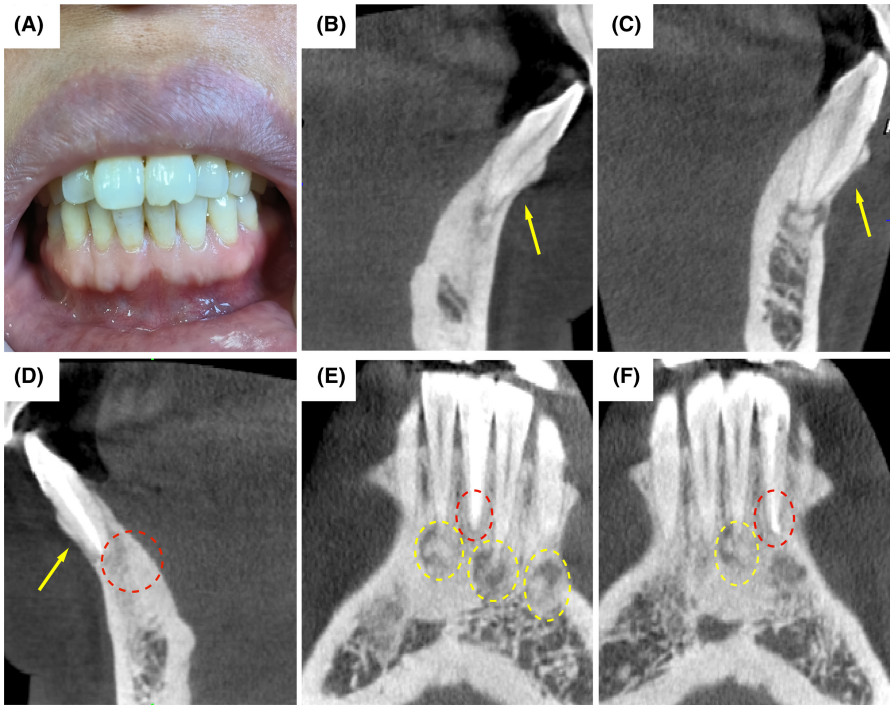

FIGURE 3 Extraoral photograph and cone beam computed tomographic images of the case on May 4, 2024. (A) Extraoral photograph revealing no abnormal soft or hard tissues in the mandibular anterior teeth region. (B, C) CBCT sagittal-sectional images illustrating no significant changes in the labial bone plates of teeth #32 and #33(arrows). (D) CBCT sagittal-sectional images showing the intact labial plate and bone remodeling in the apical alveolar bone around tooth #31 (a yellow arrow and red circle). (E, F) CBCT coronal-sectional showing some transformations of osteolytic and osteogenesis around teeth #41, #32, and #33 (yellow circles), and a bone restoration area surrounding the apical root of tooth #31 (red circles).

cemento-osseous dysplasia.[13] The term osseous dysplasia was substituted for cemento-osseous dysplasia, released in 2005 by the WHO.[14] Although the lesion was defined as a histopathological origin of cementoid elements, osseous dysplasia was established due to the challenge of distinguishing cementum from bone.[15] Osseous dysplasia consists of four lesions types: periapical, focal, florid osseous dysplasia, and familial gigantiform cementoma.[14] The 2017 edition of the WHO classification reverted to cemento-osseous dysplasia, belonging to fibro-osseous and chondro-osseous lesions, to confirm that they were odontogenic diseases originating in the periodontal ligament.[15] Familial gigantiform cementoma was no longer classified as COD but as an entity characterized by multiple, multi-quadrant conditions. In some cases, there was an autosomal dominant inheritance pattern of familial gigantiform cementoma.[15] In the 2022 WHO classification, COD was categorized as a benign fibro-osseous lesion in the jaw bones, which could be divided into three distinct subtypes based on the anatomic site and degree of progression of the lesion: periapical, focal, and florid. In addition, familial florid cemento-osseous dysplasia was added to COD's subtypes considering the ANO5 gene mutation in one family.[16] As can be seen above, the evolution of the name and classification of cemento-osseous dysplasia is extremely complicated with each edition of the WHO Classification of Head and Neck Tumors update.

COD has no specific etiological factor but is heritable, which means this disease may occur in families in practice.[17] Thus, when COD is present in a patient, the doctors must help the patient's family members understand the implications of the disease.[18] Although there is no consensus on the pathogenesis of COD, several hypotheses have been proposed. COD is considered a type of defective bone remodeling, which is initiated by local injury or an underlying hormonal imbalance.[19] Another hypothesis suggests that COD is a response to local factors.[20] In recent years, some molecular mechanisms of COD have been published. The RAS-MAPK signaling pathway has been proven to be activated in patients with COD.[2] Research shows that the signaling pathway also promotes the progression of breast cancer, which tends to occur in middle-aged women.[21] Some studies indicate that estrogen is directly correlated with breast cancer.[22] Despite these similarities in pathogenesis and factors, there is currently no conclusive explanation for the relationship between the two diseases. In the present case, the 49-year-old woman is around menopause. There is already evidence suggesting that genetic regulation can alter estrogen levels in postmenopausal women.[23] Further study

may be needed to evaluate the relationship between estrogen and COD in female patients. Milan M. Patel et al.[24] have shown that GNAS mutations are absent in COD lesions, which differs significantly from fibrous dysplasia in terms of molecular mechanism.

No reliable clinical examination methods have been utilized so far. Imaging manifestation is the primary method for diagnosing COD. In the initial stage, radiolucency is observed around the periapical areas, which often leads to confusion between COD and periapical periodontitis.[25] Some researchers[26] suggest that it is valuable to perform advanced imaging, such as computerized tomography (CT) or magnetic resonance imaging (MRI), to provide additional information. In their retrospective analysis, half of the cases with well-defined borders, featuring a sclerotic or corticated periphery and a radiolucent internal rim, account for most CODs. As shown in our patient's panoramic tomography (Figure 1A), a translucent lesion is evident at the apex of tooth #32. Meanwhile, an intraoral periapical radiograph (Figure 2A) shows that the apical region of tooth #31 is surrounded by a limited radiolucent lesion with uneven density. The border of this area is well-defined and rough. The widened periodontal membrane and the lost lamina dura are visible on tooth #31. CBCT (Figure 2E,F) shows that the lesions connected to teeth #32, #33, and #41 are mixed radiolucent and radiopaque masses. The lamina duras of the above teeth are intact and continuous, which is a crucial differentiation between PCOD and chronic periapical inflammatory diseases in terms of imaging performance. COD typically progresses gradually and matures over time, leading to an increasing number of radiopaque areas in the initially empty cavity. Finally, it has developed into a nongrowing radiopaque area with a lobular, ginger root-like appearance.[18] As shown in the patient, bone remodeling has been observed (Figure 3E,F). Unlike COD, chronic periapical inflammation can lead to alveolar bone loss, resulting in increased translucency linked to the tooth root.[27] In addition, researchers have found that female patients suffering from COD have an unusual microstructure of the mandible, with thinner cortical bone.[28] Root absorption is a characteristic imaging finding of PCOD, with a frequency of up to 57.1%.[29] It is well established that histopathology is another reliable method that can be used to diagnose COD. It is observed that normal bone formation is gradually replaced by extensive fibrous tissue and cement-like mineralization in the lesion tissue. Given that the pathogenic sites of COD always exist in the hypovascular mandible, and considering that pathological examination is an invasive procedure, asymptomatic patients may choose to decline pathological surgery to prevent osteonecrosis or fractures.[30] Diagnosis is made based on clinical features and radiographic examination.[7,18]

For the majority of patients with COD who do not exhibit any obvious clinical symptoms, conservative treatment and regular follow-up can be recommended. The effect of COD on the patients is limited, but the complications arising from COD may cause more serious health hazards. Thus, if the patients complain of unbearable symptoms such as excruciating pain, recurrence, or facial deformities, doctors should provide them with symptomatic treatments as soon as possible. Pascal Grün et al.[31] reported that a Caucasian woman with COD underwent minimally invasive surgery due to the recurrence of the lesion 2 years ago. Upon reexamination, bone remodeling was observed in the lesion. Siew-Tin Ong et al.[32] chose to pare the bone buccally to address the complaint of persistent mandibular swelling in a Chinese man with COD. Concerning PCOD, tooth extraction should not be performed on patients. After tooth extraction in patients with PCOD, nonhealing sockets, pain, and swelling may occur, necessitating additional treatment.[33] In our case, since the patient did not exhibit any symptoms and the teeth involved in PCOD are all vital, instead of other treatments, follow-up is required.

Recently, a few clinicians have found that patients also have simple bone cysts in addition to COD. Lee et al.[34] suggest that when the COD develops during the maturation stage, the lesion can transform into a simple bone cyst due to a lack of vascular circulation and blockage of venous return. J.W. Chadwick[35] proposed the hypothesis that the COD-associated simple bone cyst that occurs in middle-aged women with osteoporosis is caused by insufficient osteoblast activity to balance bone remodeling. In other words, there are few osteoprogenitor cells in the jaw bones to provide materials for bone mineralization, resulting in the formation of an empty cavity.

PCOD is a type of COD that mainly occurs in the anterior region of the mandible and is not related to teeth.[8] Compared to florid COD, another type of COD with more diverse clinical manifestations and symptoms such as buccal expansion, pain, and numbness, patients with PCOD are often less likely to notice the presence of this disease and may discover it incidentally.[33,36] Therefore, finding and diagnosing PCOD early and accurately remains a challenge for dentists.

In this case, it is important to emphasize some key differential diagnostic points regarding PCOD, chronic apical periodontitis, condensing osteitis, and developmental odontogenic tumors and cysts in the jaw bone.

Chronic apical periodontitis[37] is a common infectious disease that is often secondary to pulpitis. The disease is rarely caused by trauma. In this case, it is the habit of chewing hard food for a long time that leads to persistent chronic trauma. There is no statistical significance in the gender of the patients. Chronic apical periodontitis

typically indicates a history of toothache. Performances of images can reflect the connection between inflammation and the periodontal ligament. Eskalldoo et al.[38] advise using pulp vitality tests to distinguish odontogenic inflammatory lesions from other conditions such as COD or tumors.

Condensing osteitis is considered to be a chronic inflammatory disease to stimulate excessive bone growth.[39] CBCT shows that the lesion characterized by an ill-defined zone is related to adjacent periodontal and periapical diseases. The more severe the inflammation of teeth, the wider and denser the sclerotic area. However, the teeth associated with COD are vital, and the radiolucent rim surrounding the radiopaque masses is a typical feature in radiographs.[40]

Central ossifying fibroma should also be differentiated from COD. It is often found in middle-aged women.[41] It is a rare benign fibro-osseous lesion that usually occurs in the posterior mandibular teeth. It will invade the lower margin of the mandible and cause to bend downward when it is enlarged gradually, which is considered a typical radiograph feature.[42] A self-limited growth course is mostly accepted by professors when COD occurs in a patient.[2] It is unusual to find a cortical bone bending in the mandible in the COD process.

Odontoma is regarded as a kind of odontogenic hamartomatous tumours, which is mostly diagnosed in the first two decades of life in patients.[43] It is clinically characterized by jaw expansion, pain, and facial asymmetry.[44] Research has shown that it often causes abnormal dentition manifestations such as retention of primary teeth and loss of permanent teeth.[45] The classification is divided into two types based on histopathological characteristics: the complex type and the compound type. Radiographically, the lesion is characterized by a thin radiolucent area surrounding the central radiopaque masses with a clearly outlined boundary. The compound type presents the central masses as plenty of tooth-like structures, while the complex type appears as disordered calcified tissue.[46] Compared to the rapid growth course and representative imaging findings, the characteristics of COD are completely different.

The odontogenic keratocyst is a kind of developmental epithelial cysts. It tends to be observed in people aged between 20 and 40 years, occurring in the mandibular ramus. The characteristic radiograph is a multilocular radiolucent zone with a scalloped-shaped border and tooth root displacement. It often grows rapidly in an anteroposterior direction, which can lead to bone expansion, tooth displacement, or infection.[47] The calcifying odontogenic cyst is likely to develop in the anterior maxilla with swelling symptom. Although irregular radiopaque structures appear in the radiographs, the lesion does not go through three stages of COD typical evolution and sometimes may contain teeth.[48]

In the present case, considering that biopsy may lead to infection and the patient was symptomless, we recommended a conservative method to make the diagnosis by detailed consultation, clinical examination, imaging examination, and comprehensive differential diagnosis. MacDonald-Jankowski considered asymptomatic COD as a disease that does not require routine biopsies, but rather long-term follow-up to perfect a diagnosis.[49] He also created a flowchart for differential diagnosing maxillofacial fibro-osseous lesions from radiographic features.[50] Taha Emre Köse et al.[51] believed that biopsy is not recommended if clinical and imaging evidence is sufficient to diagnose COD. If the patient has symptoms such as paresthesia and infection, the doctor may perform a biopsy while removing the lesion to rule out malignant diseases.[31]

6 | CONCLUSION

In conclusion, PCOD is a rare fibro-osseous condition that affects the maxilla and mandible. During its initial maturation stage, it presents as radiolucent masses in the periapical region, often resembling endodontic periapical lesions. PCOD is typically asymptomatic and requires regular follow-up. Conducting a thorough clinical examination and comprehensive imaging studies is necessary to make an accurate diagnosis and develop an appropriate treatment plan.

AUTHOR CONTRIBUTIONS

Yunjing Ma: Investigation; validation; visualization; writing – original draft; writing – review and editing. **Dong Fang:** Data curation; formal analysis; resources; writing – original draft; writing – review and editing. **Mei Ji:** Conceptualization; funding acquisition; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author, Mei Ji, upon

reasonable request. The data are not publicly available due to privacy or ethical restrictions.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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REFERENCES

1. Choudhary SH, Supe NB, Singh AK, Thakare A. Florid cemento-osseous dysplasia in a young Indian female: a rare case report with review of literature. *J Oral Maxillofac Pathol*. 2019;23(3):438-442. doi:10.4103/jomfp.JOMFP_289_18
2. Haefliger S, Turek D, Andrei V, et al. Cemento-osseous dysplasia is caused by RAS-MAPK activation. *Pathology*. 2023;55(3):324-328. doi:10.1016/j.pathol.2022.10.006
3. Mohan RP, Verma S, Singh U, Agarwal N. Florid osseous dysplasia. *BMJ Case Rep*. 2013;2013:bcr2013010431. doi:10.1136/bcr-2013-010431
4. Wright JM, Vered M. Update from the 4th edition of the World Health Organization classification of head and neck Tumours: odontogenic and maxillofacial bone tumors. *Head Neck Pathol*. 2017;11(1):68-77. doi:10.1007/s12105-017-0794-1
5. Kim JH, Song BC, Kim SH, Park YS. Clinical, radiographic, and histological findings of florid cemento-osseous dysplasia: a case report. *Imaging Sci Dent*. 2011;41(3):139-142. doi:10.5624/isd.2011.41.3.139
6. Resnick CM, Novelline RA. Cemento-osseous dysplasia, a radiological mimic of periapical dental abscess. *Emerg Radiol*. 2008;15(6):367-374. doi:10.1007/s10140-008-0758-6
7. Daviet-Noual V, Ejeil AL, Gossiome C, Moreau N, Salmon B. Differentiating early stage florid osseous dysplasia from periapical endodontic lesions: a radiological-based diagnostic algorithm. *BMC Oral Health*. 2017;17(1):161. doi:10.1186/s12903-017-0455-5
8. Komabayashi T, Zhu Q. Cemento-osseous dysplasia in an elderly Asian male: a case report. *J Oral Sci*. 2011;53(1):117-120. doi:10.2334/josnusd.53.117
9. Beylouni I, Farge P, Mazoyer JF, Coudert JL. Florid cemento-osseous dysplasia: report of a case documented with computed tomography and 3D imaging. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998;85(6):707-711. doi:10.1016/s1079-2104(98)90039-7
10. Kramer IR, Pindborg JJ, Shear M. The WHO histological typing of odontogenic tumours. A commentary on the second edition. *Cancer*. 1992;70(12):2988-2994. doi:10.1002/1097-0142(19921215)70:12<2988::aid-cncr2820701242>3.0.co;2-v
11. Huh JK, Shin SJ. Misdiagnosis of florid cemento-osseous dysplasia leading to unnecessary root canal treatment: a case report. *Restor Dent Endod*. 2013;38(3):160-166. doi:10.5395/rde.2013.38.3.160
12. Gonçalves M, Píspico R, Alves Fde A, Lugão CE, Gonçalves A. Clinical, radiographic, biochemical and histological findings of florid cemento-osseous dysplasia and report of a case. *Braz Dent J*. 2005;16(3):247-250. doi:10.1590/s0103-64402005000300014
13. Summerlin DJ, Tomich CE. Focal cemento-osseous dysplasia: a clinicopathologic study of 221 cases. *Oral Surg Oral Med Oral Pathol*. 1994;78(5):611-620. doi:10.1016/0030-4220(94)90174-0
14. Zhang J, Yu Y, Tang W, Pan J, Jing W. Cemento-osseous dysplasia: a detailed comparison of the 2005 and 2017 WHO classifications and case analysis. *Cureus*. 2023;15(11):e49041. doi:10.7759/cureus.49041
15. Speight PM, Takata T. New tumour entities in the 4th edition of the World Health Organization classification of head and neck tumours: odontogenic and maxillofacial bone tumours. *Virchows Arch*. 2018;472(3):331-339. doi:10.1007/s00428-017-2182-3
16. Vered M, Wright JM. Update from the 5th edition of the World Health Organization classification of head and neck tumors: odontogenic and maxillofacial bone tumours. *Head Neck Pathol*. 2022;16(1):63-75. doi:10.1007/s12105-021-01404-7
17. Nel C, Yakoob Z, Schouwstra CM, van Heerden WF. Familial florid cemento-osseous dysplasia: a report of three cases and review of the literature. *Dentomaxillofac Radiol*. 2021;50(1):20190486. doi:10.1259/dmfr.20190486
18. Brody A, Zalatnai A, Csomo K, Belik A, Dobo-Nagy C. Difficulties in the diagnosis of periapical translucencies and in the classification of cemento-osseous dysplasia. *BMC Oral Health*. 2019;19(1):139. doi:10.1186/s12903-019-0843-0
19. Das BK, Das SN, Gupta A, Nayak S. Florid cemento-osseous dysplasia. *J Oral Maxillofac Pathol*. 2013;17(1):150. doi:10.4103/0973-029x.110735
20. Galgano C, Samson J, Kuffer R, Lombardi T. Focal cemento-osseous dysplasia involving a mandibular lateral incisor. *Int Endod J*. 2003;36(12):907-911. doi:10.1111/j.1365-2591.2003.00736.x
21. Tang YH, Rockstroh A, Sokolowski KA, et al. Neuropilin-1 is over-expressed in claudin-low breast cancer and promotes tumor progression through acquisition of stem cell characteristics and RAS/MAPK pathway activation. *Breast Cancer Res*. 2022;24(1):8. doi:10.1186/s13058-022-01501-7
22. Szmyd M, Zanib A, Behlow V, et al. Modulation of estrogen receptor alpha (ERalpha) and tumor suppressor gene BRCA1 in breast cancer cells by Bazedoxifene acetate (BZA). *Cancers (Basel)*. 2024;16(4):699. doi:10.3390/cancers16040699
23. Yu C, Bakshi A, Bell RJ, et al. Genome-wide association study identifies genetic regulation of oestrone concentrations and association with endometrial cancer risk in postmenopausal women. *EBioMedicine*. 2024;101:104997. doi:10.1016/j.ebiom.2024.104997
24. Patel MM, Wilkey JF, Abdelsayed R, D'Silva NJ, Malchoff C, Mallya SM. Analysis of GNAS mutations in cemento-ossifying fibromas and cemento-osseous dysplasias of the jaws. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2010;109(5):739-743. doi:10.1016/j.tripleo.2009.12.016
25. Shibata N, Inamoto K, Naitoh M, Ariji E. Clinical assessment of cemento-osseous dysplasia based on three-dimensional diagnostic imaging: a case report. *Aust Endod J*. 2021;47(1):105-112. doi:10.1111/aej.12488
26. Alsufyani NA, Lam EW. Osseous (cemento-osseous) dysplasia of the jaws: clinical and radiographic analysis. *J Can Dent Assoc*. 2011;77:b70.
27. Luo X, Wan Q, Cheng L, Xu R. Mechanisms of bone remodeling and therapeutic strategies in chronic apical periodontitis. *Front Cell Infect Microbiol*. 2022;12:908859. doi:10.3389/fcimb.2022.908859

28. Kato CN, Barra SG, Pereira MJ, et al. Mandibular radiomorphometric parameters of women with cemento-osseous dysplasia. *Dentomaxillofac Radiol*. 2020;49(4):20190359. doi:10.1259/dmfr.20190359
29. Gumru B, Akkitap MP, Deveci S, Idman E. A retrospective cone beam computed tomography analysis of cemento-osseous dysplasia. *J Dent Sci*. 2021;16(4):1154-1161. doi:10.1016/j.jds.2021.03.009
30. Fenerty S, Shaw W, Verma R, et al. Florid cemento-osseous dysplasia: review of an uncommon fibro-osseous lesion of the jaw with important clinical implications. *Skeletal Radiol*. 2017;46(5):581-590. doi:10.1007/s00256-017-2590-0
31. Grün P, Schneider B, Bandura P, Pfaffeneder-Mantai F, Bytyqi D, Turhani D. Bone remodelling after minimally invasive surgical management of a recurrent florid cemento-osseous dysplasia in a Caucasian woman - 18 years follow-up of a unique case - a case report. *Int J Surg Case Rep*. 2023;105:108074. doi:10.1016/j.ijscr.2023.108074
32. Ong ST, Siar CH. Florid cemento-osseous dysplasia in a young Chinese man. Case report. *Aust Dent J*. 1997;42(6):404-408. doi:10.1111/j.1834-7819.1997.tb06086.x
33. Roghi M, Scapparone C, Crippa R, Silvestrini-Biavati A, Angiero F. Periapical cemento-osseous dysplasia: clinicopathological features. *Anticancer Res*. 2014;34(5):2533-2536.
34. Lee YP, Huang BW, Chiang CP, Hwang MJ. Florid cemento-osseous dysplasia with concomitant occurrence of two simple bone cysts in the mandible - case report. *J Dent Sci*. 2020;15(3):383-385. doi:10.1016/j.jds.2020.05.009
35. Chadwick JW, Alsufyani NA, Lam EW. Clinical and radiographic features of solitary and cemento-osseous dysplasia-associated simple bone cysts. *Dentomaxillofac Radiol*. 2011;40(4):230-235. doi:10.1259/dmfr/16355120
36. Demyati AK. Florid Cemento-osseous dysplasia associated with secondary infection - a case report. *Ann Maxillofac Surg*. 2023;13(2):232-235. doi:10.4103/ams.ams_49_23
37. Cope AL, Francis N, Wood F, Chestnutt IG. Systemic antibiotics for symptomatic apical periodontitis and acute apical abscess in adults. *Cochrane Database Syst Rev*. 2018;9(9):Cd010136. doi:10.1002/14651858.CD010136.pub3
38. Eskandarloo A, Yousefi F. CBCT findings of periapical cemento-osseous dysplasia: a case report. *Imaging Sci Dent*. 2013;43(3):215-218. doi:10.5624/isd.2013.43.3.215
39. Abrahams JJ, Berger SB. Inflammatory disease of the jaw: appearance on reformatted CT scans. *AJR Am J Roentgenol*. 1998;170(4):1085-1091. doi:10.2214/ajr.170.4.9530065
40. Urs AB, Augustine J, Gupta S. Cemento-osseous dysplasia: clinicopathological spectrum of 10 cases analyzed in a tertiary dental institute. *J Oral Maxillofac Pathol*. 2020;24(3):576. doi:10.4103/jomfp.JOMFP_139_20
41. Carey K, Koenigsberg R, Kuklani R, Deitrick P. Expansive central ossifying fibroma of the right mandible. *Cureus*. 2024;16(1):e52863. doi:10.7759/cureus.52863
42. Hosseini FA, Moslemi E. Central ossifying fibroma, periapical cemento-osseous dysplasia and complex odon-toma occurring in the same jaw. *Clin Pract*. 2011;1(2):e36. doi:10.4081/cp.2011.e36
43. Goswami M, Pattu M, Johar S, Narula V. Unveiling the enigma of multiple odontomas in pediatric dentistry: a rare clinical presentation. *Cureus*. 2024;16(6):e62654. doi:10.7759/cureus.62654
44. Pethagounder Thangavelu R, Rajaram Mohan K, Mathew FS. Mystery behind a broken, discolored right maxillary central incisor: a case report. *Cureus*. 2022;14(3):e22828. doi:10.7759/cureus.22828
45. Iatrou I, Vardas E, Theologie-Lygidakis N, Leventis M. A retrospective analysis of the characteristics, treatment and follow-up of 26 odontomas in Greek children. *J Oral Sci*. 2010;52(3):439-447. doi:10.2334/josnusd.52.439
46. Nguyen DK, Van Huynh D. Clinical and radiological characteristics of odontomas: a retrospective study of 90 cases. *Imaging Sci Dent*. 2023;53(2):117-126. doi:10.5624/isd.20220184
47. Batra M, Mohod S, Sawarbandhe PA, Dadgal KV. Multilocular radiolucent pathology in the body and ramus of the mandible: a case report. *Cureus*. 2024;16(7):e63722. doi:10.7759/cureus.63722
48. Rajendra Santosh AB. Odontogenic cysts. *Dent Clin N Am*. 2020;64(1):105-119. doi:10.1016/j.cden.2019.08.002
49. Macdonald-Jankowski DS. Focal cemento-osseous dysplasia: a systematic review. *Dentomaxillofac Radiol*. 2008;37(6):350-360. doi:10.1259/dmfr/31641295
50. MacDonald-Jankowski DS. Fibro-osseous lesions of the face and jaws. *Clin Radiol*. 2004;59(1):11-25. doi:10.1016/j.crad.2003.07.003
51. Köse TE, Köse OD, Karabas HC, Erdem TL, Ozcan I. Findings of florid cemento-osseous dysplasia: a report of three cases. *J Oral Maxillofac Res*. 2013;4(4):e4. doi:10.5037/jomr.2013.4404

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