



CBCT Features of Maxillary Ameloblastoma with Internal Calcifications Mimicking a Calcifying Epithelial Odontogenic Tumour: A Case Report

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ARTICLE INFO

Keywords:

*Ameloblastoma,
Calcifying epithelial
odontogenic neoplasm,
Cone-beam computed
tomography,
Differential diagnosis,
Intrinsic calcification,
Maxillary area*

Article History:

Received : 30/12/2025

Revision : 19/08/2026

Accepted : 25/08/2026

Published : 31/08/2026

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ISSN: 2775-0159



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ABSTRACT

Introduction: Internal calcifications are an unusual radiographic finding in ameloblastoma and may mimic those seen in calcifying epithelial odontogenic tumor (CEOT), posing a diagnostic problem.

Objective: We provide a case report of maxillary ameloblastoma with internal calcifications on cone beam computed tomography (CBCT) and discuss its radiographic differential diagnosis with CEOT.

Case: A 62-year-old male was sent to the Department of Dentomaxillofacial Radiology (Universitas Padjadjaran Dental Hospital) for CBCT examination of a non-painful tumor in the right posterior maxilla. Clinical examination showed facial asymmetry with an expansion of the right maxillary region. The CBCT scan showed a large, clearly defined, slightly corticated, mixed radiolucent-radiopaque lesion in the right anterior maxilla to posterior maxilla. The lesion showed a pattern of internal radiopaque foci and cortical expansion, several cortical perforations, and invasion into the right maxillary sinus and adjacent nasal canal.

Outcome: The main radiologic diagnosis made was ameloblastoma with the size of the lesion, well-defined borders, cortical expansion, cortical perforations and invasion into the neighbouring anatomical structures. In contrast, the presence of internal calcifications suggested the possibility of CEOT and many mixed radiolucent-radiopaque odontogenic lesions among the differential diagnoses.

Conclusion: Maxillary ameloblastoma can, at times, show internal calcifications, mimicking the look of CEOT radiographically. A detailed assessment of the location, border features, internal structure, cortical involvement and neighbouring anatomical features by CBCT is required to narrow the radiological differential diagnosis. Imaging itself is not adequate for a firm diagnosis, and the definitive requirement for verification is histological investigation.

Citation (Vancouver style):

Rahasdini P, Fauziyah E, Epsilawati L. CBCT features of maxillary ameloblastoma with internal calcifications mimicking a calcifying epithelial odontogenic tumour: A case report. *Indones J Dent.* 2026;6(2), 170-179.

INTRODUCTION

Ameloblastoma is a benign epithelial odontogenic tumour characterized by slow but locally invasive growth and a marked tendency for cortical expansion and bone destruction if left untreated. According to the 5th edition of the World Health Organisation (WHO) Classification of Head and Neck Tumours (2022), Ameloblastoma is classified as a benign epithelial odontogenic tumour. It includes conventional, unicystic, extraosseous/peripheral, and adenoid Ameloblastoma, the latter being a newly recognized entity.^{1,2} Ameloblastoma accounts for approximately 10–15% of all odontogenic tumours and most commonly affects the posterior mandible, whereas maxillary involvement is considerably less frequent. Despite its benign histopathological nature, maxillary ameloblastomas may exhibit more aggressive clinical behaviour because of the thin cortical bone and their proximity to adjacent anatomical structures, facilitating early extension into the maxillary sinus, nasal cavity, and surrounding soft tissues.¹⁻³

Radiographically, conventional Ameloblastoma typically presents as a well-defined unilocular or multilocular radiolucent lesion with a characteristic soap-bubble or honeycomb appearance. Additional imaging features include cortical expansion, cortical perforation, root resorption, and displacement of adjacent teeth. Although these findings are well established, internal calcifications are exceptionally uncommon in Ameloblastoma. Since calcification within Ameloblastoma was first described in 1958, only a limited number of cases with radiographically detectable internal calcifications have been reported. Consequently, the presence of internal calcifications may result in an atypical radiographic appearance and complicate the radiographic diagnosis.^{3,4}

Calcifying epithelial odontogenic tumour (CEOT), also known as Pindborg tumour, is a rare benign epithelial odontogenic neoplasm representing less than 1% of all odontogenic tumours.^{1,2} In contrast to Ameloblastoma, CEOT characteristically contains internal calcifications that appear radiographically as scattered radiopaque foci within a radiolucent lesion, producing the classic driven-snow appearance. As mineralization progresses, CEOT commonly presents as a mixed radiolucent-radiopaque lesion with variable internal calcification patterns, thereby creating considerable overlap in the radiographic appearance of mixed odontogenic lesions.^{2,5}

Because Ameloblastoma with internal calcifications is exceedingly rare, distinguishing it radiographically from CEOT and other mixed radiolucent-radiopaque odontogenic lesions may be challenging. Comprehensive evaluation using CBCT of lesion location, border characteristics, internal structure, calcification pattern, cortical expansion, cortical perforation,

and involvement of adjacent anatomical structures is essential for narrowing the radiographic differential diagnosis. Nevertheless, radiographic findings alone are insufficient to establish a definitive diagnosis, and histopathological examination remains the gold standard for diagnostic confirmation.³⁻⁵

This case report describes the CBCT features of a maxillary ameloblastoma with internal calcifications that radiographically mimicked CEOT. The report highlights the imaging findings that favoured Ameloblastoma as the primary radiographic diagnosis while discussing its radiographic differential diagnosis with CEOT and other mixed radiolucent-radiopaque odontogenic lesions. Because the patient was referred exclusively for CBCT evaluation, this report emphasizes the diagnostic value and limitations of radiographic assessment in the absence of histopathological confirmation.

CASE REPORT

A 62-year-old man was referred to the Department of Dentomaxillofacial Radiology, Universitas Padjadjaran Dental Hospital (RSGM Unpad), for cone-beam computed tomography (CBCT) evaluation of a painless swelling in the right posterior maxilla. Extraoral examination revealed facial asymmetry caused by diffuse swelling over the right maxillary region. The swelling was hard, non-fluctuant, and showed no clinical signs of inflammation (figure 1).



Figure 1. Intraoral and extraoral examinations revealed swelling extending from the anterior to the posterior region, involving teeth 11 to 16.

Intraoral examination demonstrated expansion of the right posterior maxillary alveolar mucosa extending from teeth 13 to 16. The swelling was soft in consistency and non-tender on palpation. The posterior maxillary teeth exhibited Grade II mobility (Figure 1).



Figure 2. CBCT Panorama View.

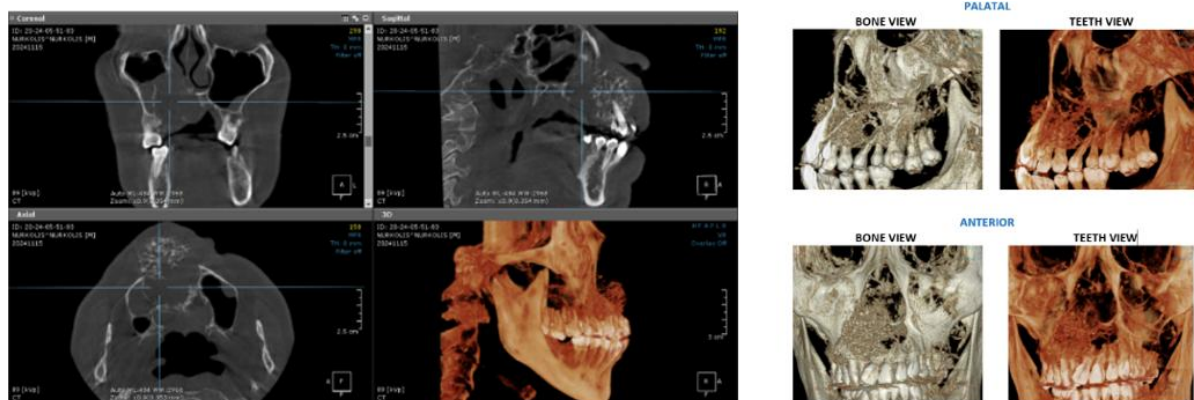


Figure 3. Shows an MPR case, where the image displays several CBCT slices, namely axial, sagittal, and coronal sections.

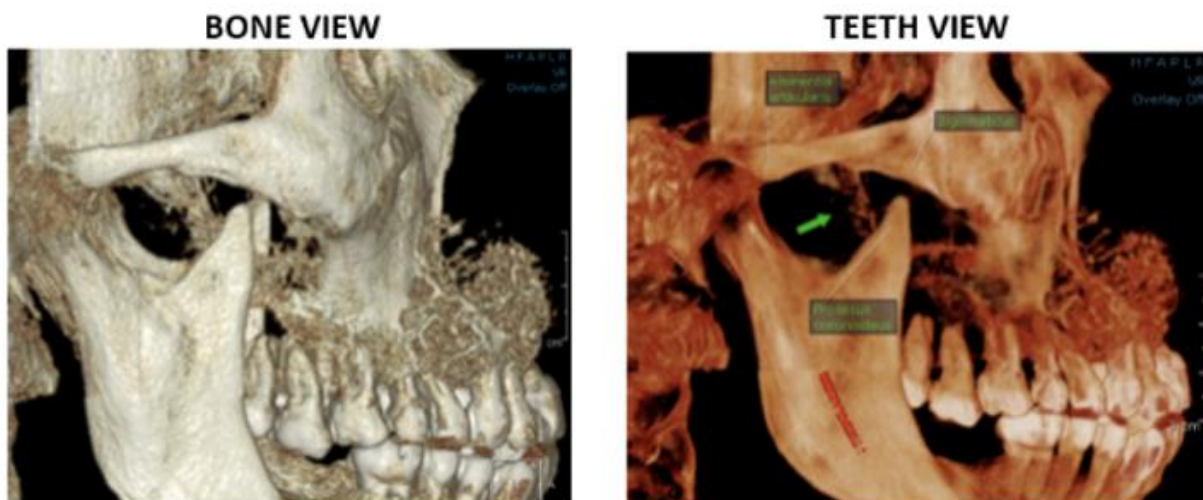


Figure 4. Multiplanar CBCT 3D reconstruction view of bone and teeth view.

CBCT panoramic reconstruction (Figure 2) showed a significant mixed radiolucent-radiopaque lesion spanning from the distal side of tooth 11 to tooth 18. The lesion consisted of several scattered internal radiopaque foci in a largely radiolucent background. Tooth 17 and 18 were displaced inferiorly relative to tooth 16. Congenital absence of teeth 47 and 48.

Multiplanar reconstruction (MPR) images (Figures 3 and 4) indicated better expansion of the lesion into the right maxillary sinus as well as into the adjacent nasal cavity with involvement of the right zygomatic bone and coronoid process. Coronal, sagittal, axial and 3D reconstructed images always revealed diverse internal architecture with many distributed internal radiopaque foci throughout the lesion.



Figure 5. coronal view (a), sagittal view (b), and axial view (c).

Cross-sectional CBCT pictures (Figure 5) showed significant buccal and palatal cortical enlargement with many cortical perforations, including buccal, palatal, labial, nasal, and maxillary sinus walls. Much of the right maxilla was involved in the lesion. It showed considerable involvement of neighboring anatomic structures including the right maxillary sinus, nasal cavity, zygomatic bone, and coronoid process, indicating a locally aggressive development tendency.

The major radiographic diagnosis was ameloblastoma based on the clinical presentation and the findings from the CBCT. These radiographical features, including extensive anteroposterior extension of the lesion, marked cortical expansion, multiple cortical perforations, displacement of adjacent teeth, and involvement of the maxillary sinus and surrounding anatomical structures are characteristic of conventional ameloblastoma, and this interpretation was supported. The presence of multiple scattered internal radiopaque foci raised the possibility of calcifying epithelial odontogenic tumor (CEOT). Still, the overall radiographic appearance was felt to be more consistent with ameloblastoma as CEOT typically presents as a relatively well-circumscribed mixed radio-lucent-radiopaque lesion with

characteristic driven-snow calcifications and generally demonstrates less extensive cortical destruction and spread to adjacent anatomical structures. So, CEOT was considered the closest radiological differential diagnosis.

Histopathological investigation was not accessible because the patient was submitted just for CBCT evaluation to characterize the lesion radiographically. Hence, the diagnosis given in this report is the first radiographic impression based on the imaging results and not a final diagnosis.

DISCUSSION

The present case is unique in that it showed many internal radiopaque foci within a massive maxillary odontogenic tumor that otherwise presented the characteristic radiographic characteristics of conventional ameloblastoma. Internal calcifications are very rare in ameloblastoma and can change its radiological appearance dramatically, making diagnosis difficult as they look very similar to the typical pattern of calcification seen in calcifying epithelial odontogenic tumor (CEOT). Although internal calcifications were present, the lesion exhibited a large anteroposterior enlargement with prominent cortical expansion, many cortical perforations, displacement of adjacent teeth and infiltration of maxillary sinus, nasal cavity, zygomatic bone and coronoid process. Collectively, these imaging features were thought to be more consistent with classical ameloblastoma than CEOT and highlight the need for thorough CBCT examination in refining the radiographic differential diagnosis prior to histological confirmation.⁶⁻⁸

Calcification in ameloblastoma is an extremely unusual entity. Most traditional ameloblastomas are completely radiolucent lesions radiographically, and a few cases have been documented to have radiographically identifiable interior calcifications. Kang et al. emphasized that the radiographically visible calcifications are rare and can be confused with mixed-density odontogenic lesions, especially CEOT, despite the presence of microscopic calcifications confirmed histopathologically. More recent results continue to demonstrate the rarity of this presentation, suggesting that ameloblastoma with radiographically identifiable interior calcifications is a rare imaging phenotype and not a recognized radiographic subtype. Therefore, the mere presence of internal calcifications should not be a criterion of exclusion for ameloblastoma in the differential diagnosis of mixed radiolucent-radiopaque odontogenic lesions.^{6,9}

The mechanism of calcification in ameloblastoma has not been fully explained. Various explanations have been suggested, including dystrophic calcification related to degenerative

changes within long standing tumors, squamous metaplasia with subsequent keratin production and inductive connections between odontogenic epithelial cells and the adjacent connective tissue. Histopathological investigations imply that these processes may favor focal mineralization inside the tumor leading to irregular calcified deposits without the organized look seen in CEOT. These mechanisms give a reasonable explanation for the heterogenous internal radiopaque foci seen in the present case.^{6,10,11}

The presence of internal calcifications suggested originally CEOT but the overall radiographic characteristics of the lesion favored ordinary ameloblastoma. This conclusion was supported by various imaging characteristics. First, the lesion had a wide anteroposterior extension covering a considerable part of the right maxilla with significant buccal and palatal cortical enlargement and many cortical perforations. Secondly, the extension into the right maxillary sinus and the surrounding nasal cavity was superior, and the zygomatic bone and coronoid process were involved, which indicated an aggressive growth pattern. The traditional maxillary ameloblastoma has aspects of imaging that are easily seen because the thin cortical bone allows early invasion of the neighboring anatomic structures. By contrast, CEOT may also cause cortical expansion and occasionally perforate the cortex, but more often presents as a relatively well-circumscribed mixed radiolucent-radiopaque lesion with characteristic punctate or granular calcifications that produce the classic driven-snow appearance. Therefore, the biological behavior and the general pattern of lesion extension shown on CBCT were in better agreement with ameloblastoma than CEOT, but the latter remained the closest differential diagnosis in radiography.¹²⁻¹⁵

Table 1. Differences between CEOT and ameloblastoma

Feature	Ameloblastoma	CEOT	Present case
Internal calcification	Rare	Common	Present
Pattern	Irregular	Driven-snow	Scattered
Cortical expansion	Frequent	Less frequent	Present
Sinus invasion	Possible	Rare	Present
Nasal cavity involvement	Possible	Rare	Present
Tooth displacement	Common	Common	Present
Root resorption	Common	Variable	Absent
Radiographic impression	Favored	Differential	Favored Ameloblastoma

As summarised in Table 1, conventional ameloblastoma and CEOT share several overlapping radiographic characteristics, including cortical expansion, tooth displacement, and a mixed radiolucent-radiopaque appearance when internal calcifications are present. Nevertheless, important differences facilitate the radiographic differential diagnosis. Internal

calcifications are a characteristic feature of CEOT and typically exhibit a punctate or granular driven-snow appearance. In contrast, calcifications in ameloblastoma are exceptionally uncommon and usually represent irregular dystrophic mineralisation without a specific radiographic pattern. Furthermore, extensive cortical perforation and invasion of adjacent anatomical structures, as observed in the present case, are more frequently associated with the locally aggressive behaviour of conventional ameloblastoma. Accordingly, evaluation of the lesion as a whole—including its location, border characteristics, internal architecture, pattern of calcification, cortical involvement, and relationship with adjacent anatomical structures—is more reliable than reliance on the presence of calcifications alone when establishing the radiographic differential diagnosis.^{7,12-15}

The principal limitation of this report is the absence of histopathological confirmation because the patient was referred exclusively for CBCT evaluation to characterise the lesion radiographically. Consequently, the diagnosis presented represents the primary radiographic impression rather than a definitive diagnosis. Nevertheless, this case highlights the important role of CBCT in assessing lesion extent, internal architecture, cortical integrity, and involvement of adjacent anatomical structures, all of which are essential for narrowing the radiographic differential diagnosis and supporting appropriate treatment planning. Although radiographic evaluation can substantially refine the diagnostic consideration, histopathological examination remains the gold standard for establishing the definitive diagnosis of odontogenic tumours.^{6,8,13}

Compared with previously reported cases, the present lesion occurred in the maxilla of a 62-year-old man and demonstrated extensive invasion of the maxillary sinus and nasal cavity with multiple cortical perforations. Unlike most previously reported cases, the internal calcifications were readily identifiable on CBCT, emphasising the value of three-dimensional imaging for detecting uncommon calcification patterns in ameloblastoma.

CONCLUSION

This example emphasizes the role of radiographic evaluation in the evaluation of odontogenic lesions and helping the process of differential diagnosis. However, radiographic evidence alone may not lead to a conclusive diagnosis, since several odontogenic tumors may have comparable imaging features. Histopathological investigation still plays an important role in validating the final diagnosis. An integrated strategy with clinical, radiographic and histological examinations is essential to reach an accurate diagnosis and arrange adequate treatment.

DECLARATION

Authors Contributions

All authors have read and approved the published version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of Interest

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.

Funding Statements

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Approval and Consent to Participate

Informed consent was obtained from all individual participants included in the study and Ethical approval was not required for this study because it involved no experimental procedures, medical interventions, or additional clinical procedures. The study was limited to the radiographic evaluation of existing clinical data obtained as part of routine patient care.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Declaration of Generative AI In Scientific Writing

The authors declare that no generative AI or AI-assisted technologies were used in the writing or preparation of this manuscript.

Acknowledgments

None.

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