ORIGINAL RESEARCH PAPER

Oral Pathology

# OSTEOSARCOMA OF THE MANDIBLE: A CASE REPORT OF CHONDROBLASTIC VARIANT WITH PATHOLOGICAL AND MOLECULAR INSIGHTS

KEY WORDS:

**Dr. Parul Dixit** Junior Resident, Government Dental College, Thiruvananthapuram

**Dr. R Rajeev** Associate Professor, Government Dental College, Thiruvananthapuram

**Dr. S.K Padmakumar** HOD & Professor, Government Dental College, Thiruvananthapuram

**Dr. R Rajeev** Associate Professor, Government Dental College, Thiruvananthapuram

**ABSTRACT**

Osteosarcoma is primarily a long-bone disease that rarely affects the facial bones. Chondroblastic osteosarcoma is a subvariant of osteosarcoma. Its defining characteristics include the presence of malignant spindle and polygonal cells, as well as a thick layer of chondroid matrix and interwoven neoplastic tissue deposition. Mandibular Chondroblastic osteosarcoma, in particular, is often overlooked and disregarded as a presumptive diagnosis at the time of initial presentation. Here, we present the case of a 38-year-old female patient with pain and mild swelling at the left side of mandible that was initially misdiagnosed as an osteomyelitis of the mandible. The subsequent histopathological examination confirmed the diagnosis of chondroblastic osteosarcoma of the mandible. This article illustrates a rare case of mandibular chondroblastic osteosarcoma, with a focus on the clinical and pathological features of the tumor that should be taken into account when making a differential diagnosis for oral bone lesions.

## INTRODUCTION

Osteosarcoma is a primary malignant bone tumour characterised by the production of osteoid or immature bone by malignant mesenchymal cells. Osteosarcoma of the jaw (OSJ) is a rare entity, accounting for approximately 6% to 10% of all osteosarcomas and about 1% of all head and neck malignancies[1]. The mandible and maxilla are the two craniofacial bones most commonly affected. OSJ differs biologically and clinically from osteosarcomas of the long bones; it generally affects an older age group, often between the third and fourth decades, whereas long-bone osteosarcomas typically occur in adolescents and young adults[2]. Furthermore, OSJ usually has a more favorable prognosis, fewer distant metastases, and better overall survival rates[3].

Clinically, patients with OSJ commonly present with localized swelling, which may be accompanied by pain, ulceration, paraesthesia, or tooth mobility. Radiographically, the lesions demonstrate diverse appearances ranging from radiolucent, mixed radiolucent-radiopaque to sclerotic patterns. Classic signs such as the 'sunburst' periosteal reaction may not always be evident in jaw lesions[4]. Histopathologically, osteosarcoma is subdivided into conventional types (osteoblastic, chondroblastic, and fibroblastic), as well as other variants including telangiectatic, small cell, and parosteal types[5]. Chondroblastic osteosarcoma (COS) is characterized by abundant hyaline cartilage alongside malignant osteoid and accounts for roughly 25% of osteosarcomas[6].

Given the overlapping clinical and radiological features with benign inflammatory or infectious jaw lesions, diagnosis of OSJ, particularly the chondroblastic variant, can be challenging and often delayed. Immunohistochemical and molecular studies have emerged as valuable tools to aid in accurate diagnosis and to differentiate COS from other cartilaginous tumors such as chondrosarcoma[7].

### Case Presentation

A 38-year-old female patient presented with a three-week history of numbness on the left side of the mandible extending to the midline. Her past medical history was notable for surgical extraction of the left mandibular third molar (tooth 38) approximately eighteen months prior. She denied any history of radiation exposure or long-term medication use, and there was no known family history of malignancy.

On clinical examination, paraesthesia was present over the left chin region, and tooth 37 showed tenderness on percussion. No swelling, ulceration, or discharge was observed in the region at the time of presentation. The overlying skin was normal, and vitality tests of anterior teeth were within normal limits. A distobuccal caries lesion was present in tooth 37 and was managed conservatively.

Panoramic radiography revealed a mixed radiolucent-radiopaque lesion in the region of the previously extracted 38, positioned superior to the inferior alveolar nerve (IAN) canal. A thin bony layer separated the lesion from the overlying soft tissues (Figures -1).

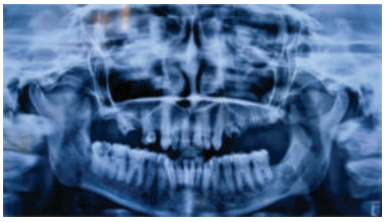

**Figure 1:** Panoramic radiograph showing a mixed radiolucent-radiopaque lesion in the left posterior mandible, superior to the inferior alveolar canal, suggestive of a malignant neoplasm such as chondroblastic osteosarcoma.

Subsequent cone beam computed tomography (CBCT) images showed a destructive lesion with ill-defined borders in the 38 region, with a well-defined radiopacity approximately 0.3 x 0.3 cm surrounded by radiolucency superior to the IAN canal, suggestive of sequestrum. The lesion extended anteriorly from the periapical region of tooth 37's distal root to the external oblique ridge posteriorly. Destruction of the residual alveolar ridge was noted superiorly, with involvement and enlargement of the IAN canal inferiorly, and root resorption of tooth 37's distal root was evident (Figures 2)

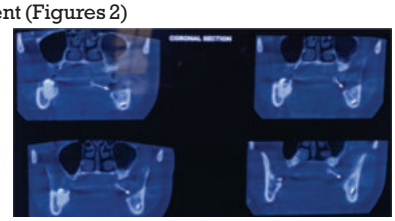

**Figure 2:** Coronal CBCT sections showing an ill-defined

osteolytic lesion with cortical destruction in the left posterior mandible.

The initial radiographic impression was osteomyelitis of the left posterior mandible. After 1.5 months, the patient developed pain in the affected region, prompting an incisional biopsy. Histopathological evaluation revealed moderately collagenous connective tissue stroma containing numerous malignant binucleated and multinucleated chondroblast cells exhibiting marked pleomorphism within chondroid matrix. Adjacent sections showed numerous malignant osteoblasts and tumor osteoid formation (Figures3a-3b). Based on these findings, a diagnosis of chondroblastic osteosarcoma was made.

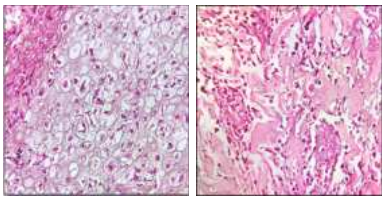

**Figure 3a & 3b:** (H&E, 400x) Figure 3a shows numerous pleomorphic chondroblasts with hyperchromatic nuclei and abundant chondroid matrix, while Figure 3b demonstrates malignant osteoblasts laying eosinophilic tumor osteoid, indicative of chondroblastic osteosarcoma.

## DISCUSSION

Osteosarcoma of the jaw is an uncommon tumor, constituting about 1% of all head and neck malignancies and approximately 6% to 10% of all osteosarcomas[5,6]. The chondroblastic subtype, which accounts for nearly 25% of osteosarcomas, is characterized by the coexistence of malignant cartilaginous and osteoid tissue[7]. The mandible is more frequently affected than the maxilla, and jaw osteosarcomas tend to affect a slightly older population than those arising in long bones[8].

Radiographically, OSJ lesions show variable presentations based on the balance of osteoid and cartilaginous matrix. Unlike long bone osteosarcomas that commonly demonstrate a sunburst periosteal reaction, jaw lesions often show mixed radiopaque-radiolucent appearances with poorly defined margins and can mimic infectious or inflammatory lesions such as osteomyelitis, as seen in this case[9].

Histopathological confirmation remains the gold standard for diagnosis. COS is distinguished from other variants by the presence of abundant malignant hyaline cartilage alongside tumor osteoid produced by malignant osteoblasts[6]. Distinguishing COS from chondrosarcoma is critical but challenging, as both tumors express cartilaginous features. Immunohistochemical markers are thus pivotal in differentiation. COS typically exhibits positivity for vimentin and S-100 protein, which mark mesenchymal and cartilaginous differentiation respectively, but these are not exclusive[7,10]. The nuclear transcription factor SATB2, associated with osteoblastic lineage, has been shown to be a sensitive and specific marker for osteosarcoma, including COS, aiding in differentiation from chondrosarcoma, which is generally SATB2-negative[10,11]. Additionally, galectin-1 overexpression in COS has been implicated in tumor progression and may serve as a prognostic marker[12].

At the molecular level, osteosarcomas demonstrate complex genetic alterations involving tumor suppressor genes and oncogenes. Alterations in TP53, RB1, and amplification of MDM2 are commonly reported[13,14]. These genetic events lead to dysregulation of cell cycle control and contribute to malignant transformation. While COS lacks the frequent IDH1/2 mutations seen in conventional chondrosarcoma, molecular testing for these mutations can provide useful diagnostic distinction when histopathology is equivocal[15].

Therapeutically, wide surgical excision remains the mainstay of treatment for OSJ, often supplemented by chemotherapy and/or radiotherapy depending on tumor grade and stage[8,16]. Jaw osteosarcomas tend to have a better prognosis than those of long bones, with fewer metastases and improved survival rates, but early and accurate diagnosis is essential to optimize outcomes[5].

This case highlights the importance of integrating clinical, radiological, histopathological, immunohistochemical, and molecular data for the diagnosis of chondroblastic osteosarcoma of the mandible. Misdiagnosis as osteomyelitis can delay appropriate treatment. Awareness of the pathological spectrum and the role of specific markers like SATB2 is vital for pathologists and clinicians managing such rare tumors.

## CONCLUSION

Chondroblastic osteosarcoma of the mandible is a rare malignant tumor with distinct pathological and clinical features that can mimic benign conditions, leading to diagnostic delays. A multidisciplinary approach involving detailed imaging, biopsy with thorough histopathologic examination, immunohistochemical profiling, and molecular analysis is crucial for accurate diagnosis and appropriate management. Awareness of key diagnostic markers and molecular alterations assists in distinguishing COS from other cartilaginous tumors, thereby guiding optimal treatment strategies and improving patient outcomes.

## REFERENCES


1. Krishnamurthy A, et al. Osteosarcoma of the jaw: a clinicopathologic review. J Oral Maxillofac Pathol. 2019;23(1):15-22.
2. Subramaniam SS, et al. Osteosarcoma of the jaws: a clinicopathologic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;107(5):711-716.
3. Boffano P, et al. Osteosarcoma of the jaws: a single institution experience with 35 cases. Head Neck Pathol. 2015;9(3):375-382.
4. Regezi JA, Sciubba JJ, Jordan RCK. Oral Pathology: Clinical Pathologic Correlations. 7th ed. Elsevier; 2016.
5. Mirra JM. Bone Tumors: Clinical, Radiologic, and Pathologic Correlations. 1st ed. Lea & Febiger; 1989.
6. Kim M, et al. Chondroblastic osteosarcoma of the jaw: a clinicopathologic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2012;114(3):386-393.
7. Xu G, et al. Immunohistochemical and molecular diagnosis of chondroblastic osteosarcoma: a review. Diagn Pathol. 2019;14(1):58.
8. Kaffe I, et al. Osteosarcoma of the jaws: a diagnostic challenge. Oral Oncol. 2017;72:77-85.
9. Philipsen HP, et al. Radiologic features of jaw osteosarcomas. Oral Surg Oral Med Oral Pathol. 1994;77(2):145-150.
10. Tsuji S, et al. SATB2 as a novel marker for osteoblastic differentiation in bone tumors. Mod Pathol. 2014;27(5):675-685.
11. Kurek KC, et al. SATB2 expression in chondroblastic osteosarcoma and chondrosarcoma: diagnostic implications. Hum Pathol. 2017;66:26-32.
12. Yu L, et al. Galectin-1 overexpression in chondroblastic osteosarcoma correlates with poor prognosis. Oncol Lett. 2018;15(4):5389-5396.
13. Perry JA, et al. Molecular insights into osteosarcoma pathogenesis. J Pathol. 2015;237(2):179-189.
14. Nakano K, et al. MDM2 amplification in osteosarcoma: molecular analysis and clinicopathological correlations. J Cancer Res Clin Oncol. 2016;142(9):1923-1932.
15. Amary MF, et al. IDH1 and IDH2 mutations are frequent events in central chondrosarcoma and chondromas but rare in osteosarcoma. J Pathol. 2011;224(3):334-343.
16. Bergh P, et al. Osteosarcoma of the jaw: treatment and outcome in 41 patients. Eur J Cancer. 1999;35(6):808-815.