Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

Ameloblastic Carcinoma: A Case Report and Comprehensive Review of a Rare Odontogenic Malignancy

[1] Dr. Shivam Ghatge, [2] Prof. Dr. Pradeep D, [3] Dr. Aditya NK, [4] Dr. Murugesan Krishnan, [5] Dr. Hemavathy Murulidos

[1] Post Graduate Student, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[2] Professor & Research Head, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[3] Senior Lecturer, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[4] Professor, Head of Department, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

[5] Professor, Saveetha Dental College and Hospital, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai, Tamil Nadu, India

(Received: 25 August 2025 Revised: 27 September 2025 Accepted: 14 October 2025)

KEYWORDS

Ameloblastic carcinoma, Mandible, Surgical margins, Odontogenic carcinoma.

ABSTRACT:

Background: Ameloblastic carcinoma (AC) is a rare odontogenic malignancy that combines histological features of ameloblastoma with malignant cytology. Its rarity and infiltrative nature pose challenges for diagnosis, treatment, and prognosis.

Case Presentation: A 34-year-old male presented with swelling in the left mandible. Imaging revealed a 3.8 × 2.7 × 7 cm expansile lytic lesion. Histopathology confirmed ameloblastic carcinoma with nests and sheets of malignant odontogenic epithelial cells, peripheral palisading, and atypical mitoses. Final excision revealed a positive superior mucosal margin and critically close margins at medial (0.3 cm), posterior (0.5 cm), and lateral (0.5 cm) soft tissues. Adjuvant radiotherapy (66 Gy with boost) was administered. At 6 months, the patient remained disease-free.

Conclusion: Adequate surgical margins remain the cornerstone of AC management. When margins are positive or close, adjuvant radiotherapy is essential for locoregional control. Advances in molecular oncology (e.g., BRAF-targeted therapy) may further transform future management.

1. Introduction

Ameloblastic carcinoma (AC) is a primary odontogenic carcinoma, recognized by the World Health Organization (WHO) as an aggressive malignant tumor that exhibits histological features of ameloblastoma alongside concurrent cytological malignancy[1,3]. This is a crucial distinction from malignant ameloblastoma, which is defined as a tumor that metastasizes while retaining the benign histological features of a conventional ameloblastoma in both the primary and metastatic lesions[2,34]. This terminological clarity is essential for proper diagnosis and management, as the presence of malignant cytology in the primary lesion of an AC dictates a more aggressive initial approach.

2. Case Presentation

A 34-year-old male patient reported to our department with a progressively enlarging swelling in the left mandibular region since 1 year. OPG revealed a 4 × 7 cm radiolucent lesion involving the left body, angle, and ramus of the mandible, extending from the area distal to 36 to the posterior border of the mandible anterior-posteriorly and extending from the sigmoid notch to the lower border of mandible and involving the coronoid process. Contrast Enhanced Computer tomography of Head and neck region revealed an expansile lytic lesion noted epicentered in the body, angle, coronoid process, and ramus of the left hemimandible, measuring 3.8 x 2.7

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

x 7 cm in dimensions. The lesion appeared heterogeneously hypointense.

The patient then underwent incisional biopsy, which revealed nests and sheets of malignant odontogenic epithelial cells in a solid growth pattern with central necrosis[1,10]. The tumor cells exhibited features consistent with malignancy, including nuclear pleomorphism, hyperchromatism, an increased nucleus-to-cytoplasm ratio, and atypical mitotic figures[1,10]. Specific findings included peripheral palisading of cells with a loss of reverse polarity and vesicular nuclei, which are hallmarks of ameloblastoma-like differentiation[10]. Histopathologically, the lesion was suggestive of ameloblastic carcinoma.

Immunohistochemistry was performed to confirm the diagnosis and assess the aggressive nature of the tumor. The Ki-67 proliferation index was 22%, which is considered a high expression and correlates with the aggressive behavior of ameloblastic carcinoma[10].

The patient underwent a left hemimandibulectomy with modified radical neck dissection (MRND) under general anesthesia.

Surgical Approach and Mandibular Resection:

A combined submandibular and intraoral approach was utilized to access the mandible. Osteotomy lines were planned to ensure at least a 2 cm bony margin from radiologically evident tumor margins. The resection included the left mandibular body, angle, ascending ramus, coronoid process, and a portion of the condylar neck. Surrounding soft tissues including periosteum, overlying mucosa, and buccinator attachments were removed en bloc to minimize the risk of microscopic residual disease. Despite intraoperative frozen section analysis suggesting tumor clearance, the superior mucosal margin was later found to be involved microscopically, highlighting the limitations of intraoperative margin assessment in this region.

Neck Dissection:

A modified radical neck dissection (MRND, Type I–III with preservation of the spinal accessory nerve, internal jugular vein, and sternocleidomastoid muscle) was performed. This encompassed levels I–V lymph node groups. Although cervical lymph node metastasis is rare in ameloblastic carcinoma, advanced tumors (T3/T4) carry a risk of occult nodal spread. In this patient, no gross nodal involvement was evident intraoperatively, and histopathology confirmed all nodes negative for metastasis (0/24 nodes).

Reconstruction Strategy:

Primary vascularized bony reconstruction was deferred in anticipation of postoperative radiotherapy, as adjuvant treatment can compromise flap survival. The plan included secondary reconstruction with a free fibula flap after completion of radiotherapy and oncologic clearance.

Rationale for surgical extent: Hemimandibulectomy was chosen because of extensive intraosseous spread, which would render marginal resection oncologically inadequate. MRND was performed prophylactically in line with recommendations for T4 lesions, ensuring both therapeutic clearance of potential microscopic nodal disease and accurate pathologic staging.

Final histopathology showed nests and sheets of malignant odontogenic epithelial cells with peripheral palisading, vesicular nuclei, and moderate mitotic activity (1–2/HPF)[10]. Superior mucosal margin was positive, while medial mucosal margin (0.3 cm), posterior soft tissue (0.5 cm), and lateral soft tissue (0.5 cm) were critically close. Anterior bony and mucosal margins were clear by 2.0 cm. No perineural or lymphovascular invasion was seen. Pathologic staging: pT4aN0M0.

Given that there was inadequate marginal clearance on the medial, posterior, and lateral soft tissue margins, adjuvant radiotherapy was administered (66 Gy in 33 fractions with an additional 6 Gy boost to the superior margin)[44]. At 6-month follow-up, the patient remains disease-free clinically. The decision to administer adjuvant radiotherapy directly illustrates the multi-modal approach required for effective management of AC.

3. Discussion

Epidemiology and Natural History:

Ameloblastic carcinoma is an exceptionally rare malignancy, a characteristic that fundamentally shapes the clinical approach to the disease. Its rarity is best understood when contrasted with its benign counterpart, the ameloblastoma. The benign ameloblastoma is itself considered rare, with a incidence rate of 0.92 cases per

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

million person-years[4,29]. Ameloblastic carcinoma is significantly rarer, comprising only a small fraction of all odontogenic tumors, with estimates ranging from 1.5–2%[5,4]. In a study of 538 ameloblastoma patients, only 12 cases (2.23%) were identified as ameloblastic carcinoma[15]. The extreme rarity of AC is not a mere statistical observation; it is the primary reason why large-scale, prospective clinical trials are virtually impossible to conduct. This lack of robust, high-level evidence forces the medical community to rely on a scattered body of literature composed of single case reports, small institutional series, and retrospective reviews. This reliance on lower-level evidence is a critical consideration that must be acknowledged when discussing all aspects of AC management, from its diagnosis and treatment to its prognosis and long-term surveillance.

Ameloblastic carcinoma occurs over a wide age range, with a reported mean age of occurrence ranging from 39 to 56.3 years and a slight male predilection, with male-to-female ratios reported as high as 5:1 in some series[6,15]. The case of the 34-year-old male falls within this typical age demographic. Approximately 80-92% of cases are found in the mandible, particularly the posterior region, with the maxilla accounting for the remaining cases[2,15]. While the posterior mandible is the most common location, the anatomical site has been noted to be a critical prognostic factor. Lesions in the maxilla may have a poorer prognosis due to the spongy nature of the maxillary bone, which can facilitate tumor spread and infiltration of adjacent vital structures[24]. This suggests that anatomical location is not merely a descriptive feature but a key determinant of the required aggression of the surgical and adjuvant therapeutic approach.

Pathological and Molecular Diagnostics:

The definitive diagnosis of ameloblastic carcinoma is a histopathological one, based on the presence of cytological malignancy within a background of ameloblastoma-like differentiation[1,9,10]. Key microscopic features include nests and sheets of epithelial cells exhibiting nuclear pleomorphism, hyperchromatism, an increased nucleus-to-cytoplasm ratio, and atypical mitotic figures[1,8–10]. These features distinguish AC from benign ameloblastoma, which lacks these malignant cytological characteristics[5,10].

The diagnostic process for ameloblastic carcinoma has evolved beyond purely morphological assessment to include a detailed analysis of immunohistochemical (IHC) and molecular markers. This provides crucial information for both diagnosis and therapeutic planning.

- **Proliferation and Tumor Suppressor Markers:** Markers of cellular proliferation are essential for differentiating AC from its benign counterpart. The Ki-67 proliferation index is significantly higher in AC, with a mean labeling index of 21.6% in a study of 11 cases, ranging widely from 2% to 80%[11,10,25]. This high proliferative rate is a direct reflection of the aggressive biological behavior of the tumor[3]. Similarly, the expression of tumor suppressor proteins like p53 and p63 is consistently elevated in AC compared to ameloblastoma, reinforcing their role in the malignant transformation process and serving as useful diagnostic indicators[10,12,25].

- **Differential Markers:** Certain cytokeratins and other markers help to characterize the tumor's cellular origin and behavior. While Cytokeratin 19 (CK19) is a reliable marker for benign odontogenic tumors, its expression in AC is variable, a finding that suggests underlying genetic alterations in the malignant cells[12,25]. Emerging research has also identified other markers, such as SOX2 and GPC3, as potentially useful for accurately distinguishing between aggressive and malignant odontogenic tumors[12].

- **Molecular Markers and Targeted Therapy:** A critical advancement in the understanding of odontogenic tumors is the discovery of specific genetic mutations. The BRAF V600E mutation, a key driver in the mitogen-activated protein kinase (MAPK) signaling pathway, is highly prevalent in ameloblastomas and their malignant counterparts, occurring in up to 70% of cases[14,26,20]. The discovery of this actionable mutation has created a bridge between pathology and therapy, enabling a more precise, individualized treatment approach[16,17,18]. This fundamental shift from a purely histological diagnosis to one informed by molecular markers allows pathologists to not only confirm malignancy but also identify specific targets for therapeutic intervention, potentially transforming the management of unresectable or recurrent disease.

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

Table 1: Comparative Immunohistochemical and Histological Profile of Ameloblastoma vs. Ameloblastic Carcinoma

Feature	Ameloblastoma	Ameloblastic Carcinoma
Clinical Behavior	Slow-growing, painless swelling; locally aggressive[37]	Rapidly progressing painful swelling; extensive local destruction; higher metastatic potential[1,5,8]
Radiography	Radiolucent, uni- or multilocular; well-defined margins[1,5]	Radiolucent, uni- or multilocular; ill-defined margins; significant cortical destruction[1,2,5]
Cytological Atypia	Absent[5]	Present; nuclear pleomorphism, hyperchromatism, increased nucleus-to-cytoplasm ratio[10]
Mitotic Figures	Infrequent	Moderate to numerous; atypical mitotic figures[10]
IHC: Ki-67 LI	Low expression (<10%)[33]	High expression, mean 21.6% (range 2-80%); correlates with aggressive nature[3,10]
IHC: p53 & p63	Low or variable expression[10,12]	Higher expression; useful markers for malignancy[12,13]
IHC: CK19	Good marker for benign odontogenic tumors[12]	Variable expression, suggesting genetic alterations[12,13]
Molecular Markers	BRAF V600E mutation common (up to 70%); others include SMO gene mutations[14,24]	BRAF V600E mutation common; MAPK pathway activated[14,16]

Multimodality Treatment:

Surgical resection with wide margins remains the gold standard for achieving local control and providing the best chance of a cure for ameloblastic carcinoma[5,21,40]. The infiltrative nature of the tumor, which can extend beyond its visible or radiologically-defined borders, necessitates an aggressive surgical approach[1,7]. Recommended surgical margins for AC are consistently reported to be wider than those for benign ameloblastomas, with a minimum of 1-2 cm and some recommendations extending to 2-3 cm of bony margin[20,41,48].

The patient in the presented case, with its left hemimandibulectomy, aligns with these recommendations for radical resection. The patient also underwent a modified radical neck dissection, which is typically considered for cases with palpable lymph nodes or other high-risk features, despite the absence of perineural invasion in this specific case[8,42]. The histopathological finding of a positive superior mucosal margin and critically close soft tissue margins in the patient's final specimen highlights the immense challenge of achieving complete surgical clearance in the complex anatomy of the head and neck, a circumstance that necessitates the use of adjuvant therapy.

While benign ameloblastomas have traditionally been considered radioresistant, the role of adjuvant radiotherapy in the management of ameloblastic carcinoma has become increasingly important, particularly when surgical margins are compromised[5,22,23,44]. The patient in the case report, with positive and close margins, is a textbook example of the primary indication for this therapy. The administered dose of 66 Gy in 33 fractions, with an additional 6 Gy boost to the positive superior margin, is consistent with high-dose regimens (66-72 Gy) that have been shown to improve local control in patients with close or positive resection margins[23,44]. The success of this approach in the patient, who remained disease-free at 6-month follow-up, underscores an important therapeutic evolution. The role of radiation therapy for AC has shifted from a last-resort palliative measure for unresectable tumors to an essential and effective tool for achieving curative local control when anatomical constraints or tumor characteristics prevent adequate surgical clearance[22]. Modern, precise dose delivery techniques further enhance this role by improving efficacy while minimizing collateral tissue damage.

The landscape of systemic therapy for ameloblastic carcinoma is rapidly evolving. Historically, traditional

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

chemotherapy has had a very limited role in the management of AC, with poor outcomes reported for non-metastatic disease[2,25,26]. Its use has been largely confined to the palliative management of metastatic disease[2,21]. The discovery of the high prevalence of the BRAF V600E mutation in odontogenic tumors has paved the way for a more effective systemic approach[16,17,18]. Targeted inhibitors, such as dabrafenib (a BRAF inhibitor) and trametinib (a MEK inhibitor), have demonstrated significant efficacy in both ameloblastoma and ameloblastic carcinoma[16,18,30]. These drugs have shown remarkable tumor reduction and, in some cases, complete radiological remission, which can enable less morbid, "organ-sparing" surgeries[18,30]. This development could fundamentally change the treatment paradigm from a mutilating radical surgery to a more precise, function-preserving approach[34].

While specific data on the use of immunotherapy for AC is scarce, its potential role is significant[19]. Immune checkpoint inhibitors (e.g., pembrolizumab, nivolumab) have shown promising results in other head and neck malignancies, with neoadjuvant use leading to impressive tumor shrinkage and a robust immune response[38,45]. The logical next step in clinical research is to explore the use of these agents in ameloblastic carcinoma, given its classification as a head and neck malignancy and the potential for a less morbid, targeted treatment.

Table 2: Summary of Ameloblastic Carcinoma Treatment Modalities

Treatment Modality	Primary Indication	Rationale/Key Data	Limitations
Surgical Resection	Primary and recurrent lesions[21]	Gold standard for curative intent; wide margins (1-2 cm) are essential to prevent recurrence; high recurrence rates (up to 90%) for conservative approaches[32,49]	Anatomical constraints, potential for facial disfigurement and functional loss[24]
Adjuvant Radiotherapy	Inadequate surgical margins (positive or close)[5,22]	Improves local control; doses between 66-72 Gy have shown efficacy; shifts role from palliative to curative[23,44]	Risk of radiation-related morbidity; limited data from large trials due to rarity[22,23]
Targeted Therapy	BRAF V600E-mutated tumors; unresectable, recurrent, or metastatic disease[16,20]	BRAF/MEK inhibitors (dabrafenib, trametinib) can induce significant tumor regression, potentially enabling less morbid surgery[16,18,30]	Not all tumors harbor the mutation; long-term data on durability of response is limited[16,18]
Conventional Chemotherapy	Palliative care for metastatic disease[2,21]	Has a limited role with generally poor outcomes for non-metastatic disease[25,26]	No proven efficacy as a primary treatment[21,25,26]

Prognosis and Recurrence:

Multiple factors influence the prognosis of ameloblastic carcinoma, with margin status being the most critical. Data from a study by Naidu et al. (2018) reported a 5-year recurrence-free survival of 80% for R0 resections (negative margins), which dropped significantly to 55% for close margins and only 35% for margin-positive cases[34]. The presented case, with its positive and close margins, underscores the direct link between surgical clearance and patient prognosis, demonstrating the absolute necessity of aggressive adjuvant therapy to mitigate the risk of recurrence. Beyond margin status, certain histological features are also key prognostic indicators. Tumors exhibiting a plexiform pattern, hyperchromatism, increased mitotic index, and necrosis are associated with a higher rate of recurrence and death[10,13]. The presence of clear cells is a particularly ominous sign, as a majority of cases with this feature have a history of recurrence and mortality[10,13].

Ameloblastic carcinoma has a high propensity for local recurrence, even with proper surgical management. The recurrence rate is heavily dependent on the type of treatment rendered, with conservative approaches like enucleation alone associated with recurrence rates as high as 90%[32,49]. In contrast, wide surgical resection with 10-20 mm margins is associated with a much lower

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

recurrence rate (0-4.6%)[49]. Recurrence can be protracted, appearing anywhere from a few months to more than a decade after initial treatment[2,41]. The possibility of late recurrence necessitates a life-long surveillance protocol, which should include periodic physical and radiographic examinations to allow for early detection and intervention[28].

4. Conclusion

The management of ameloblastic carcinoma is a formidable challenge, primarily due to its extreme rarity and aggressive, infiltrative behavior. The reliance on a scattered body of evidence from case reports and small series, rather than large-scale, prospective clinical trials, is a pervasive reality that clinicians must navigate. The gold standard for treatment remains aggressive surgical resection with wide margins. The provided case report successfully demonstrates the critical role of adjuvant radiotherapy in salvaging a favorable outcome when a radical R0 resection is not achievable due to anatomical constraints.

The future of AC management is poised for a significant transformation driven by advancements in molecular oncology. The identification of key genetic drivers, such as the BRAF V600E mutation, has opened the door to targeted therapies that offer the potential to reduce tumor size, minimize surgical morbidity, and improve long-term outcomes. While conventional chemotherapy remains largely ineffective, the demonstrated efficacy of targeted agents offers a more precise and effective systemic treatment for unresectable, recurrent, or metastatic disease. The logical next step in clinical practice is to explore the role of emerging therapies, such as immunotherapy, within the context of controlled clinical trials, ideally through multi-center collaboration to overcome the challenge of this disease's rarity. The integration of precision medicine into the existing multi-modal treatment framework holds the promise of a conceptual revolution in how this rare and challenging malignancy is managed, moving from purely ablative surgery to a more tailored, function-preserving approach.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of Generative AI and AI-assisted technologies in the writing process':

During the preparation of this work the author(s) used CHATGPT AI in order to improve readability and language.. *After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication*

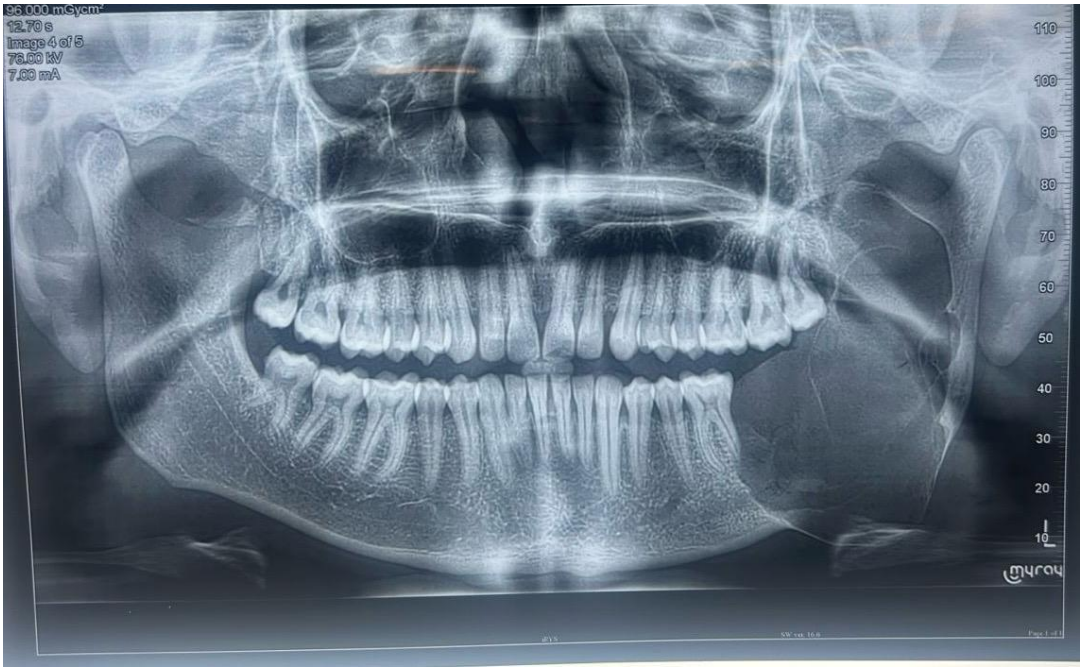


Fig. 3A: Preoperative orthopantomogram (OPG) showing an expansile radiolucent lesion involving the left mandibular body, angle, and ascending ramus with cortical thinning and destruction.

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

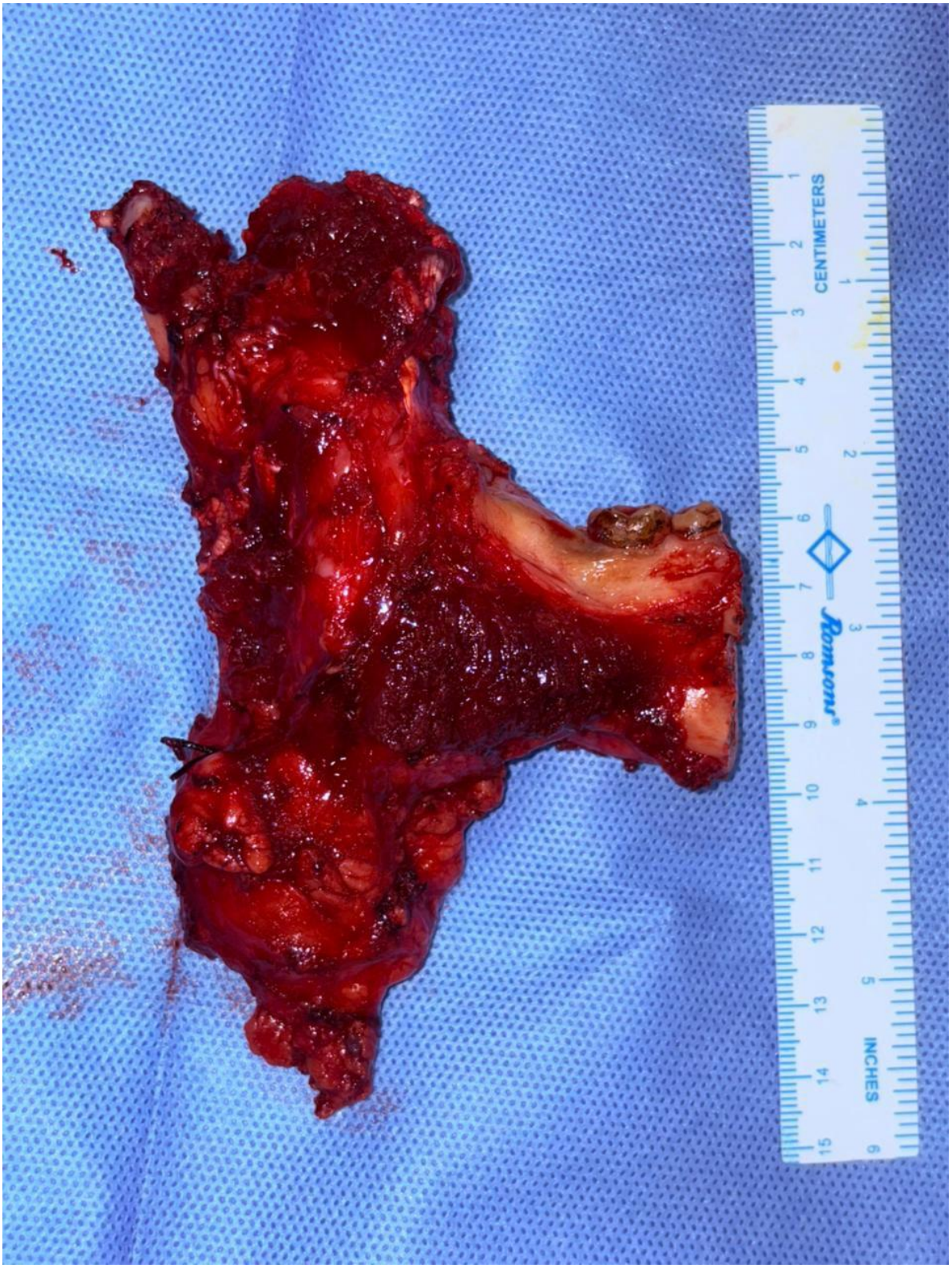
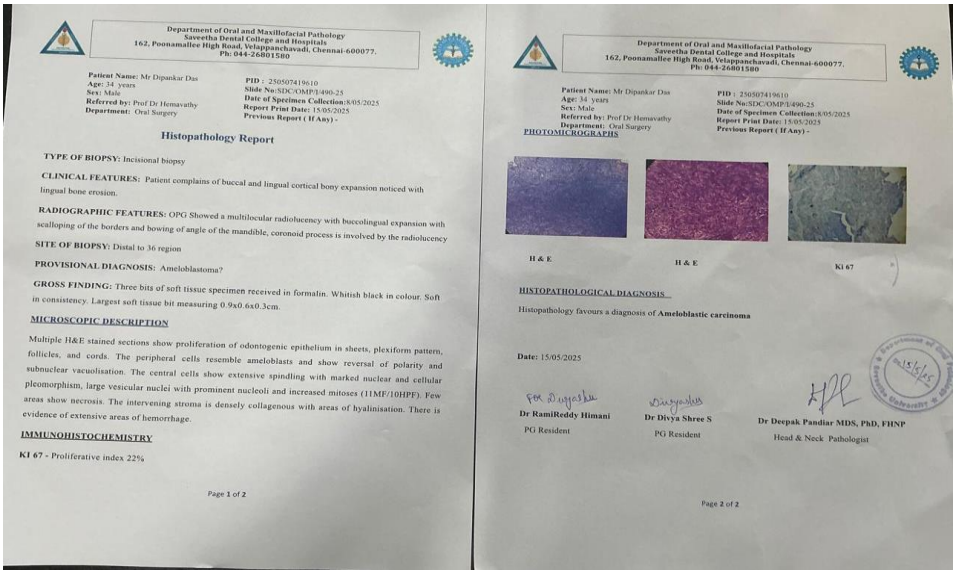


Fig. 3B: Gross excisional specimen (8 × 5 × 3 cm) from left hemimandibulectomy, demonstrating en bloc resection of the mandibular body, angle, and ramus with attached soft tissues. Surgical margins are inked for orientation.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727



Department of Oral and Maxillofacial Pathology
Saveetha Dental College and Hospitals
162, Poonamallee High Road, Velappanchavadi, Chennai-600077.
Ph: 044-26801580

Patient Name: Mr Dipankar Das
Age: 34 years
Sex: Male
Referred by: Prof Dr Hemavathy
Department: Oral Surgery

PID : 250507419610
Slide No:SDC/OMP/I/490-25
Date of Specimen Collection:8/05/2025
Report Print Date: 15/05/2025
Previous Report (If Any) -

Histopathology Report

TYPE OF BIOPSY: Incisional biopsy

CLINICAL FEATURES: Patient complains of buccal and lingual cortical bony expansion noticed with lingual bone erosion.

RADIOGRAPHIC FEATURES: OPG Showed a multilocular radiolucency with buccolingual expansion with scalloping of the borders and bowing of angle of the mandible, coronoid process is involved by the radiolucency

SITE OF BIOPSY: Distal to 36 region

PROVISIONAL DIAGNOSIS: Ameloblastoma?

GROSS FINDING: Three bits of soft tissue specimen received in formalin. Whitish black in colour. Soft in consistency. Largest soft tissue bit measuring 0.9x0.6x0.3cm.

MICROSCOPIC DESCRIPTION

Multiple H&E stained sections show proliferation of odontogenic epithelium in sheets, plexiform pattern, follicles, and cords. The peripheral cells resemble ameloblasts and show reversal of polarity and subnuclear vacuolisation. The central cells show extensive spindling with marked nuclear and cellular pleomorphism, large vesicular nuclei with prominent nucleoli and increased mitoses (11MF/10HPF). Few areas show necrosis. The intervening stroma is densely collagenous with areas of hyalinisation. There is evidence of extensive areas of hemorrhage.

IMMUNOHISTOCHEMISTRY

KI 67 - Proliferative index 22%

Page 1 of 2

Department of Oral and Maxillofacial Pathology
Saveetha Dental College and Hospitals
162, Poonamallee High Road, Velappanchavadi, Chennai-600077.
Ph: 044-26801580

Patient Name: Mr Dipankar Das
Age: 34 years
Sex: Male
Referred by: Prof Dr Hemavathy
Department: Oral Surgery

PID : 250507419610
Slide No:SDC/OMP/I/490-25
Date of Specimen Collection:8/05/2025
Report Print Date: 15/05/2025
Previous Report (If Any) -

PHOTOMICROGRAPHS

H & E H & E Ki 67

HISTOPATHOLOGICAL DIAGNOSIS

Histopathology favours a diagnosis of **Ameloblastic carcinoma**

Date: 15/05/2025

Dr RamiReddy Himani
PG Resident

Dr Divya Shree S
PG Resident

Dr Deepak Pandiar MDS, PhD, FHNP
Head & Neck Pathologist

Page 2 of 2

Fig. 3C: Low-power histopathology (H&E, ×100) revealing nests of odontogenic epithelial cells with peripheral palisading and ameloblastoma-like patterns, but with atypical features.

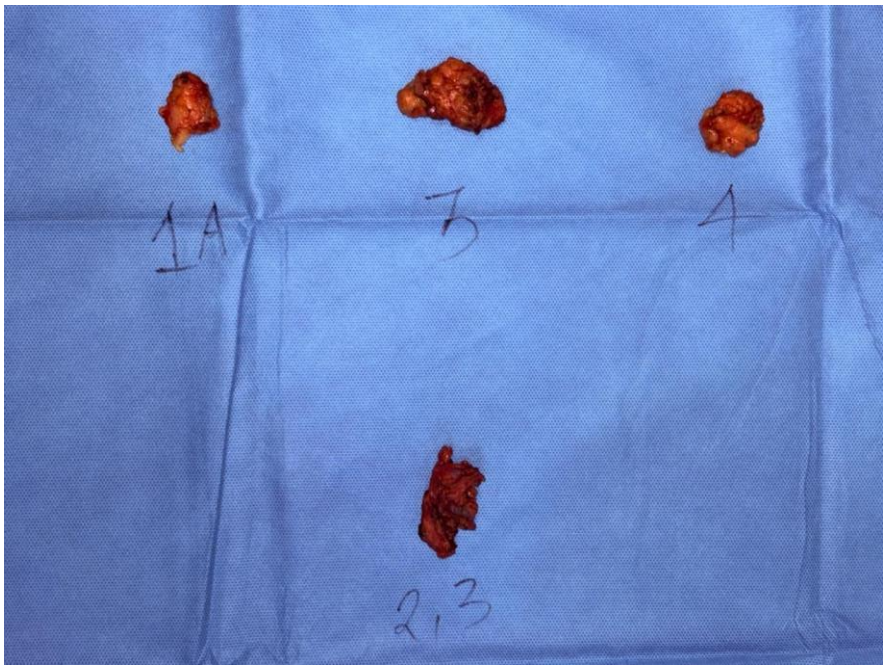


Fig. 3D: Resected cervical lymph node specimen following modified radical neck dissection, submitted for histopathological evaluation (0/24 nodes positive).

References:

1. ResearchGate. Differences between ameloblastic carcinoma and malignant ameloblastoma. Available at: https://www.researchgate.net/figure/Differences-between-ameloblastic-carcinoma-and-malignant-ameloblastoma_fig1_321535725.
2. Fortune Journals. Ameloblastic Carcinoma: Presentation of Two Case Reports. Available at: https://www.fortunejournals.com/articles/ameloblastic-carcinoma-presentation-of-two-case-reports.html.
3. Pratibha V, Manjunatha BS. Ameloblastic Carcinoma. **J Oral Maxillofac Pathol**. 2011;15(3):316. doi:10.4103/0973-029X.86701.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

4. Ajnr. Ameloblastic Carcinoma (AC) | American Journal of Neuroradiology. Available at: https://www.ajnr.org/ajnr-case-collections-diagnosis/ameloblastic-carcinoma-ac.
5. Avon SL, McComb J, Clokie C. Staging and treatment strategies for odontogenic malignancies. J Oral Pathol Med. 2013;42(7): 505-12. doi:10.1111/jop.12053.
6. Martínez-Martínez R, Mosqueda-Taylor A. Ameloblastic carcinoma: a clinico-pathological analysis of cases seen in a Nigerian Teaching Hospital and review of literature. J Oral Maxillofac Pathol. 2014;18(3):355. doi:10.4103/ 0973-029X.150937.
7. Slootweg PJ, Müller H. Malignant ameloblastoma or ameloblastic carcinoma. Oral Surg Oral Med Oral Pathol. 1984;57(2):168-76. doi:10.1016/0030-4220(84)90209-6.
8. Khan H, Qureshi SM, Khan AH, Khan MA. Ameloblastic carcinoma: A clinicopathologic analysis of 12 cases with a review of the literature. Oncol Lett. 2014;8(5):2137-41. doi:10.3892/ol. 2014.2230.
9. Yoon HJ, Hong SP, Lee JI, Hong SD. Ameloblastic carcinoma: an analysis of 6 cases with a review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;108 (6):904-13. doi:10.1016/j.tripleo.2009.07.039.
10. Rivas-Gutiérrez D, López-Sánchez RM, Sotomayor-Rivas M, Martínez-Martínez M. Ameloblastic carcinoma, primary type: case report, immunohistochemical analysis and review of the literature. Anticancer Res. 2012;32(4): 1515-21.
11. Boffano P, Zino F, Ghezzi F, et al. Ameloblastic carcinoma. J Oral Maxillofac Surg. 2014;72(2): 345-53. doi:10.1016/j.joms.2013.06.216.
12. MacDonald R, et al. The diagnostic usefulness of immunohistochemistry for odontogenic lesions. Diagn Pathol. 2014;9:112.
13. Rivas-Gutiérrez D, López-Sánchez RM, Sotomayor-Rivas M, et al. Ameloblastic carcinoma, primary type: case report, immunohistochemical analysis and review of the literature. Anticancer Res. 2012;32(4):1515-21.
14. Carlson ER, Gordon S, et al. Ameloblastoma: The gold standard of treatment. J Oral Maxillofac Surg. 2010;68(10):2375-81.
15. Yoon HJ, Hong SP, Lee JI, et al. Ameloblastic carcinoma: an analysis of 6 cases with a review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;108(6):904-13.
16. The Cleveland Clinic. Ameloblastoma. Available at: https://my.clevelandclinic.org/health/diseases/ 22143-ameloblastoma.
17. ResearchGate. Targeted therapies in ameloblastomas and amelobastic carcinoma-A systematic review. Available at: https://www. researchgate.net/publication/380289247_Targeted_therapies_in_ameloblastomas_and_amelobastic_carcinoma-A_systematic_review.
18. Ameloblastic carcinoma: a case report and evaluation. J Oral Maxillofac Surg. 2022;80(1): 65-71. doi:10.1016/j.joms.2021.09.006.
19. Wikipedia. Ameloblastic carcinoma. Available at: https://en.wikipedia.org/wiki/Ameloblastic_ carcinoma.
20. The Cleveland Clinic. Ameloblastoma. Available at: https://my.clevelandclinic.org/health/diseases /22143-ameloblastoma.
21. NORD. Ameloblastic Carcinoma. Available at: https://rarediseases.org/rare-diseases/ ameloblastic-carcinoma/.
22. Lee YS, et al. Treatment of ameloblastoma and ameloblastic carcinoma with radiotherapy. Oral Oncol. 2014;50(5):e6-e11. doi:10.1016/j.oral oncology.2014.03.003.
23. Gaultney JM, et al. The role of postoperative radiotherapy for ameloblastic carcinoma of the mandible: case report. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;114(5):e45-e49.
24. Ramadas K, et al. Ameloblastic carcinoma of maxilla. J Oral Maxillofac Surg. 2012;70(11): 2621-5.
25. Elzay RP. A review of 12 cases of ameloblastic carcinoma. J Oral Surg. 1982;40(3):139-43.
26. Brown J, et al. Targeted therapy for unresectable ameloblastoma. Lancet Oncol. 2017;18(3):e181-e190.
27. Pillai KR, Govindan A, Gopalan A. Surgical margins in mandibular malignancies: analysis and

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(5), 3037-3046 | ISSN:2251-6727

recommendations. Int J Oral Maxillofac Surg. 2014;43(9):1056-61.
28. American Cancer Society. Immunotherapy for Oral Cavity and Oropharyngeal Cancer. Available at: https://www.cancer.org/cancer/types/oral-cavity-and-oropharyngeal-cancer/treating/immunotherapy.html.
29. Hendra N. Global Incidence and Profile of Ameloblastoma. PhD Thesis. NVMKA. 2022.
30. The Ohio State University. Targeted Therapy for Ameloblastoma. Available at: https://wexnermedical.osu.edu/stories/ameloblastoma-targeted-therapy.
31. Canadian Cancer Society. Immunotherapy for oral cancer. Available at: https://cancer.ca/en/cancer-information/cancer-types/oral/treatment/immunotherapy.
32. Sampson J, et al. The Ameloblastoma: Primary, Curative Surgical Management. J Oral Maxillofac Surg. 2005;63(9):1268-76. doi:10.1016/j.joms.2005.04.013.
33. StatPearls. Ameloblastoma. Available at: https://www.ncbi.nlm.nih.gov/books/NBK545165/.
34. Naidu A, Gopalakrishnan R, Srinivasan B. Impact of close surgical margins in ameloblastic carcinoma. Oral Oncol. 2018;78:142-7.
35. The Cleveland Clinic. Ameloblastoma. Available at: https://my.clevelandclinic.org/health/diseases/22143-ameloblastoma.
36. Sood V, et al. Ameloblastic carcinoma: A rare case report and review of the literature. J Oral Maxillofac Pathol. 2014;18(3):477. doi:10.4103/0973-029X.150942.
37. Oral Surgery, Oral Medicine, Oral Pathology, and Oral Radiology. Available at: https://paperpile.com/s/oral-surgery-oral-medicine-oral-pathology-and-oral-radiology-citation-style/.
38. Medscape. Immunotherapy Tested in 'Notoriously Difficult' Oral Cancers. Available at: https://www.medscape.com/viewarticle/926008.
39. Cancer Research UK. Oral Cancer Clinical Trials. Available at: https://www.cancerresearchuk.org/about-cancer/find-a-clinical-trial/search/results?q=oral%20cancer.
40. Al-Aithan J, et al. Surgical Margins for Ameloblastoma: A Review. J Oral Maxillofac Surg. 2012;70(11):e272-e280. doi:10.1016/j.joms.2012.06.014.
41. Pillai K, et al. Ameloblastic carcinoma. Int J Oral Maxillofac Surg. 2014;43(9):1056-61.
42. The Cleveland Clinic. Ameloblastoma. Available at: https://my.clevelandclinic.org/health/diseases/22143-ameloblastoma.
43. National Cancer Institute. Clinical Trials for Head and Neck Cancer. Available at: https://www.cancer.gov/about-cancer/treatment/clinical-trials/search?CANCER_TYPE=head-and-neck-cancer.
44. ResearchGate. Radiotherapy in the Treatment of Ameloblastoma and Ameloblastic Carcinoma. Available at: https://www.researchgate.net/publication/267242349_Radiotherapy_in_the_Treatment_of_Ameloblastoma_and_Ameloblastic_Carcinoma.
45. The Ohio State University. Targeted Therapy for Ameloblastoma. Available at: https://wexnermedical.osu.edu/stories/ameloblastoma-targeted-therapy.
46. American Cancer Society. Immunotherapy for Oral Cavity and Oropharyngeal Cancer. Available at: https://www.cancer.org/cancer/types/oral-cavity-and-oropharyngeal-cancer/treating/immunotherapy.html.
47. Canadian Cancer Society. Immunotherapy for oral cancer. Available at: https://cancer.ca/en/cancer-information/cancer-types/oral/treatment/immunotherapy.
48. The Cleveland Clinic. Ameloblastoma. Available at: https://my.clevelandclinic.org/health/diseases/22143-ameloblastoma.
49. The Cleveland Clinic. Ameloblastoma. Available at: https://my.clevelandclinic.org/health/diseases/22143-ameloblastoma.