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## Adenoid Ameloblastoma of the Mandible: A Rare Histopathological Case Report

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### Abstract

Ameloblastoma is the second most common odontogenic tumour, traditionally classified into six histopathological variants. The recent 2022 WHO classification broadened the spectrum of odontogenic lesions by introducing a novel entity, highlighting the evolving nature of jaw pathology. We report a case of this newly recognized entity in a 46-year-old male patient with mild pain in the mandibular anterior teeth region during chewing. Radiographic evaluation revealed a multilocular radiolucent lesion with root resorption. Histopathological examination demonstrated odontogenic epithelial islands with duct-like structures, microcyst formation, squamous metaplasia, and juxta-epithelial hyalinization. IHC confirmed BRAF V600E negativity, supporting the diagnosis of adenoid ameloblastoma. This case emphasizes that deceptively minor symptoms may conceal significant pathology, highlighting the importance of timely recognition.

**Keywords:** *Ameloblastoma, Odontogenic Tumors, Jaw Neoplasms, Neoplasms classification, BRAF Mutation.*

### I. INTRODUCTION

Odontogenic tumours encompass a wide spectrum of lesions derived from the tooth-forming apparatus, ranging from innocuous hamartomas to locally aggressive neoplasms. Among them, Ameloblastoma, the second most common odontogenic tumour, has traditionally been classified into six histopathological variants: Follicular, Plexiform, Acanthomatous, Granular cell, Desmoplastic and Basal cell types [1]. Despite its benign nature, it is clinically significant due to its infiltrative growth, recurrence potential, and ability to mimic other jaw pathologies. The 2022 World Health Organization (WHO) classification of head and neck tumours introduced Adenoid Ameloblastoma (AA) as a distinct entity, reflecting the evolving understanding of jaw pathology [2]. We present a case of this newly recognized variant with an unusual presentation confirmed by histopathology and immune histochemistry. This case highlights the need for timely recognition, as deceptively minor symptoms may conceal significant pathology.

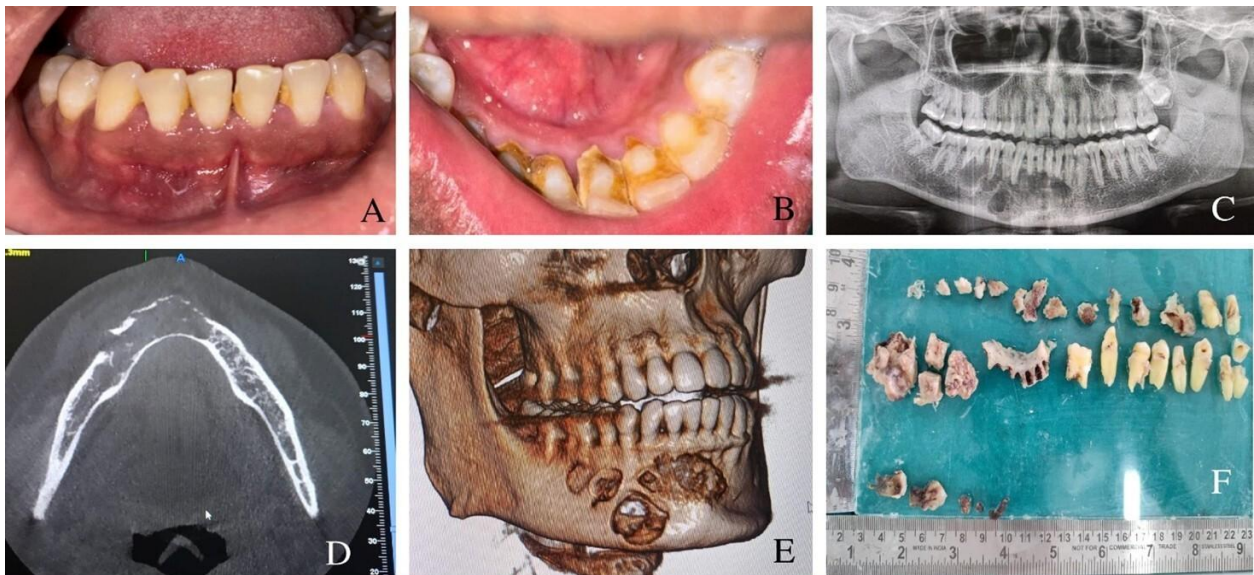
### II. CASE REPORT:

A 46-year-old male presented to the outpatient department with mild pain in the mandibular

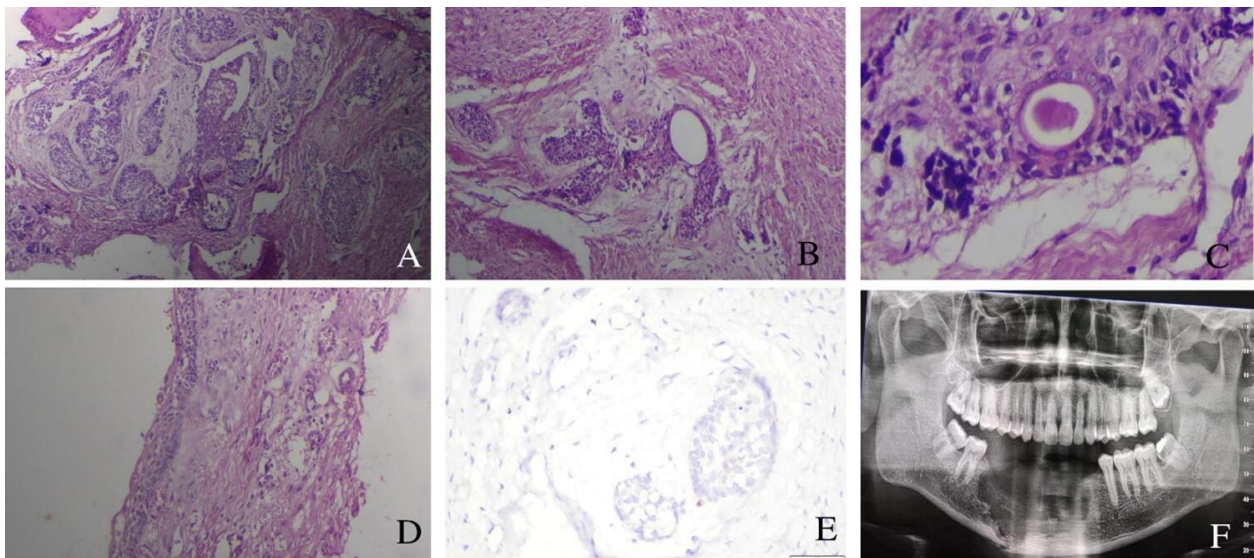
anterior region during chewing for one month. The pain initially presented as sensitivity and gradually progressed into a dull, persistent ache, without swelling or pus discharge. Extra oral examination revealed no facial asymmetry. Intraorally calculus was evident (figure 1A, 1B) and teeth 41, 42 and 43 were tender on percussion, leading to a provisional clinical diagnosis of Apical Periodontitis. Orthopantomogram (OPG) revealed a multilocular radiolucent lesion extending mesiodistally from tooth 33 to 46 and superoinferiorly from the interradicular region to 0.2mm short of inferior border of the mandible with root resorption in tooth 31, 32, 41, 42 and 46 (figure 1C). Cone Beam Computed Tomography (CBCT) demonstrated a well-defined hypodense multilocular lesion with corticated margins, cortical thinning and expansion (figure 1D). Three-dimensional reconstruction delineated the lesion's full extent and relationship to adjacent structures (figure 1E). Based on these findings, an odontogenic keratocyst (OKC) was considered the most likely provisional diagnosis, with Ameloblastoma, Central giant cell granuloma, Central mucoepidermoid carcinoma, Primary Intraosseous squamous cell carcinoma and metastatic tumours as radiographic differentials.

Under general anaesthesia, teeth from 33 to 46 were extracted following the placement of a

and processed for histopathological examination (figure 1F). Histopathological examination revealed



**Fig 1:** A, B - Clinical intraoral view shows calculus deposition and gingival inflammation. C - OPG revealed a multilocular radiolucent lesion from tooth 33 to 46 with root resorption in 31, 32, 41, 42 & 46. D - CBCT demonstrated a well-defined hypodense lesion with cortical thinning and expansion. E - Three-dimensional reconstruction delineated the lesion's full extent and relationship to adjacent structures. F- Cystic and solid specimen, extracted teeth and alveolar socket received in lab which were subjected to processing.



**Fig 2:** A - Histopathologically multiple odontogenic epithelial islands were evident within dense stroma. B - Islands showing duct like structures with lumen and squamous metaplasia. C - Duct like structures showing eosinophilic secretory material. D - Cystic degeneration of the solid tumour with subepithelial hyalinization suggestive of inductive effect. E - IHC showing negativity for BRAF V 600E. F - 3 month follow up OPG showing fracture of right parasymphysis.

crevicular incision. On surgical exposure, a cystic lesion was noted in the incisor region, while a solid tumour was observed in the molar, premolar regions. Based on these findings, the surgeons suspected a hybrid lesion. However, subsequent evaluation confirmed that the cystic component represented degeneration within a solid tumour rather than a true hybrid pathology. Wide surgical excision was done and Carnoy's solution was applied to minimize recurrence. In the laboratory, multiple specimens – including extracted teeth, associated soft tissue and alveolar socket tissue – were received

multiple odontogenic epithelial islands within a dense fibrous stroma, peripherally lined by columnar ameloblast like cells and cuboidal cells surrounding central loosely arranged stellate reticulum like cells. Duct-like structures, microcyst formation in a cribriform pattern, squamous metaplasia and juxta epithelial hyalinization suggestive of an inductive effect were also noted (figure 2A, 2B, 2C & 2D). Immunohistochemistry confirmed BRAF V 600 E negativity, supporting the diagnosis of adenoid ameloblastoma (figure 2E).

At the three-month follow-up, the patient developed a Para symphyseal fracture requiring internal fixation (figure 2F), which occurred subsequent to surgery due to marked thinning of the bone by the lesion.

### III. DISCUSSION:

Adenoid Ameloblastoma (AA) has recently been incorporated into the 2022 WHO classification of odontogenic tumours and cysts of the jaws as a benign epithelial odontogenic tumour with aggressive behaviour[3]. Adenoid features in ameloblastoma were first identified by Waldron in

1959, and the term "adenoid ameloblastoma" (AA) was later introduced by Brannon in 1994.[4]. It predominantly affects adults (76.5%) with a male predilection (63.3%). The mean age at presentation is 40.8 years, with reported cases ranging from 15 to 70 years and only one case has been documented in a patient younger than 18 years. Anatomically, AA is slightly more common in the mandible (53.3%) than in the maxilla (46.7%) [5].

Histologically, AA demonstrates epithelial components resembling conventional ameloblastoma, accompanied by duct-like structures, epithelial whorls, and a cribriform architectural pattern. Additional findings may include dentinoid deposition, clusters of clear cells, and ghost-cell keratinization. While the ameloblastomatous component reflects conventional ameloblastoma, the adenoid structures resemble those of Adenomatoid Odontogenic Tumour (AOT) [6]. Differential diagnoses include aggressive AOT, clear cell odontogenic carcinoma, Ameloblastic carcinoma, and dentinogenic ghost cell tumour [7].

The molecular basis of AA remains incompletely understood. Unlike conventional ameloblastoma and AOT, AA does not exhibit the KRAS or BRAF mutations seen in these two entities, respectively. Recent report by Bastos et al. identified CTNNB1 hotspot mutations in Adenoid ameloblastoma accompanied by nuclear accumulation of beta-catenin. This finding is similar to that seen in dentinogenic ghost cell tumours (DGCT), with which Adenoid ameloblastoma shares histological, behavioural, and radiological features [5].

The present case highlights several important considerations. First, the clinical presentation was subtle with localised pain during mastication and no overt swelling or facial asymmetry. Such subtle symptoms can easily be mistaken for common inflammatory conditions, as reflected in the initial provisional diagnosis of Apical Periodontitis. Further, OPG revealed a multilocular radiolucent

lesion with root resorption, a feature more suggestive of aggressive pathology, thereby prompting further investigation. Second, the histopathological findings were crucial in establishing the diagnosis. The presence of ameloblast-like cells and stellate reticulum confirmed the odontogenic origin, while duct-like structures with eosinophilic material and induction effect distinguished this lesion from conventional Ameloblastoma. The absence of BRAF V600E mutation supported the diagnosis of Adenoid ameloblastoma, distinguishing it from other variants that commonly harbour this mutation.

Before recognition by WHO, only about 50 cases had been documented. Sharma et al. (2023) reported two cases with extensive histological overlap, emphasizing the diagnostic challenges and the need for IHC confirmation [8]. Sachdev et al (2022) provided a systematic review of Adenoid ameloblastoma with dentinoid, emphasizing its rarity and diagnostic challenges [9]. More recently, Keshwar et al. (2024) presented histopathological insights into a rare case, reinforcing the need for careful differentiation from other odontogenic tumours [10].

From a management perspective, wide surgical excision was performed in the present case, consistent with the principles of Ameloblastoma treatment. Given the aggressive nature of AA and its infiltrative growth pattern, conservative approaches are inadequate. Although recurrence potential remains uncertain due to rarity of reported cases, resection with adequate margins and long-term follow-up is considered the most appropriate treatment strategy. Comparative analysis with AOT and DGCT highlights the importance of distinguishing AA accurately, as treatment strategies differ significantly.

### IV. CONCLUSION

In conclusion, this case reinforces the broader clinical lesson that odontogenic tumours may present with minimal symptoms yet harbour significant pathology. Early recognition, thorough radiographic assessment, and histopathological confirmation are indispensable for accurate diagnosis and timely management. As more cases are reported, a clearer understanding of the biological behaviour, recurrence potential, and optimal treatment strategies of AA will emerge, guiding evidence-based clinical practice. Incorporation of molecular profiling and IHC markers will further refine diagnostic accuracy and help distinguish AA from histologically overlapping odontogenic tumours.

**Ethics:** "Written informed consent was obtained from the patient for publication of this case report and accompanying images."

**Funding/Conflict of Interest:** "The authors declare no conflict of interest."

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