SAS Journal of Medicine
Abbreviated Key Title: SAS J Med
ISSN 2454-5112
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology

Unicystic Ameloblastoma of the Maxilla Presenting as an Oroantral Communication: A Case Report

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DOI: https://doi.org/10.36347/sasjm.2026.v12i03.007 | **Received:** 01.02.2026 | **Accepted:** 12.03.2026 | **Published:** 14.03.2026

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Abstract **Case Report**

Background: Psoriasis is a chronic immune-mediated inflammatory disease that typically follows a controllable course under dermatological management. However, severe phenotypes may evolve into life-threatening systemic conditions requiring intensive care unit (ICU) admission. Despite this risk, psoriasis remains under-recognized as a cause of critical illness, and guidance on ICU management is limited. ***Objectives:*** To illustrate the spectrum of critical complications associated with severe psoriasis, identify clinical warning signs necessitating ICU admission, and highlight key principles of intensive care management through representative clinical cases. ***Methods:*** We report three severe and illustrative cases of psoriasis complicated by life-threatening systemic manifestations : methotrexate toxicity in end-stage renal disease, erythrodermic psoriasis, and generalized pustular psoriasis complicated by drug reaction with eosinophilia and systemic symptoms (DRESS). Clinical presentation, laboratory findings, therapeutic interventions, and outcomes are analyzed in the context of current literature. ***Results:*** All cases demonstrated rapid progression from cutaneous disease to systemic inflammatory failure. Two patients developed multiorgan dysfunction and died despite intensive care management. One patient with erythrodermic psoriasis recovered following aggressive ICU stabilization and subsequent biologic therapy. These cases underscore the role of systemic inflammation, immunosuppression, infection, and drug toxicity in driving critical illness among patients with psoriasis. ***Conclusions:*** Severe psoriasis should be regarded as a multisystem inflammatory disorder with the potential for critical deterioration. Early recognition, strict pharmacovigilance, prompt ICU referral, and close collaboration between dermatologists and intensivists are essential to improving outcomes. Integrated care pathways bridging dermatology and intensive care medicine are urgently needed for high-risk patients.

Keywords: Psoriasis; Intensive care; Erythrodermic psoriasis; Generalized pustular psoriasis; Methotrexate toxicity; DRESS syndrome; Critical illness; Multiorgan failure; Multidisciplinary management.

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INTRODUCTION

Ameloblastoma is a benign, locally aggressive odontogenic neoplasm arising from the epithelial remnants of the dental lamina. While it accounts for approximately 1% of all oral tumors, it is clinically significant due to its potential for extensive bone destruction and high recurrence rates [1,2]. The unicystic ameloblastoma (UA), first described as a distinct entity by Robinson and Martinez in 1977, represents a variant that shares clinical and radiographic similarities with odontogenic cysts but possesses a neoplastic epithelial lining [3,4]. UA accounts for approximately 5–15% of all ameloblastomas and is predominantly found in the mandible, with the posterior ramus being the most common site [5,6].

Maxillary ameloblastomas are comparatively rare, with a reported mandible-to-maxilla ratio ranging from 3:1 to as high as 13:1 [1,7]. Despite their lower incidence, maxillary lesions pose a significantly higher therapeutic challenge. The maxilla features thin cortical plates and cancellous bone, offering little resistance to tumor expansion. This anatomical vulnerability allows the tumor to silently invade the maxillary sinus, pterygopalatine fossa, or orbit before clinical symptoms, such as swelling or facial asymmetry, become apparent [4,8]. Furthermore, UAs frequently present as unilocular

radiolucencies associated with impacted teeth, leading to their frequent misdiagnosis as dentigerous cysts [7,9].

A missed diagnosis in the maxilla can lead to conservative treatments (like simple enucleation) that may be insufficient for aggressive subtypes, resulting in recurrence in anatomically complex areas [4]. We report a case of a maxillary unicystic ameloblastoma in a patient where the pathology was unmasked not by swelling, but by a persistent oroantral communication following tooth extraction. This case underscores the critical role of multi-modality imaging in differentiating benign cysts from neoplastic mimics in the maxilla.

CASE REPORT

A male patient in his 30s presented to the Department of Oral and Maxillofacial Surgery following a complicated dental extraction. The patient had undergone the extraction of the left maxillary third molar (tooth 28) at a private clinic due to local pain. Immediately following the removal of the tooth, the treating dentist noted a large, unexpected oroantral communication (OAC) and the presence of abnormal tissue within the socket, prompting an urgent referral.

On presentation, the patient reported mild pain but no purulent discharge or history of epistaxis. Intraoral examination revealed a significant expansile lesion involving the left side of the hard palate. A polypoid soft tissue mass, approximately 4 cm in diameter, was visible extending from the extraction site and the adjacent palatal mucosa. The overlying mucosa was non-ulcerated but distended.

A Computed Tomography (CT) scan of the maxilla was performed to assess the extent of the defect. The imaging revealed a massive, expansile, unilocular radiolucent lesion occupying the entire left maxillary sinus. The sinus walls exhibited a "blown-out" appearance, characteristic of a slow-growing but locally expansile pathology.

Crucially, the CT demonstrated extensive bony destruction, including:

- Complete resorption of the floor of the maxillary sinus.
- Breach of the lateral and posterior sinus walls.
- Posterior extension into the pterygoid space, with clear separation of the medial and lateral pterygoid plates.

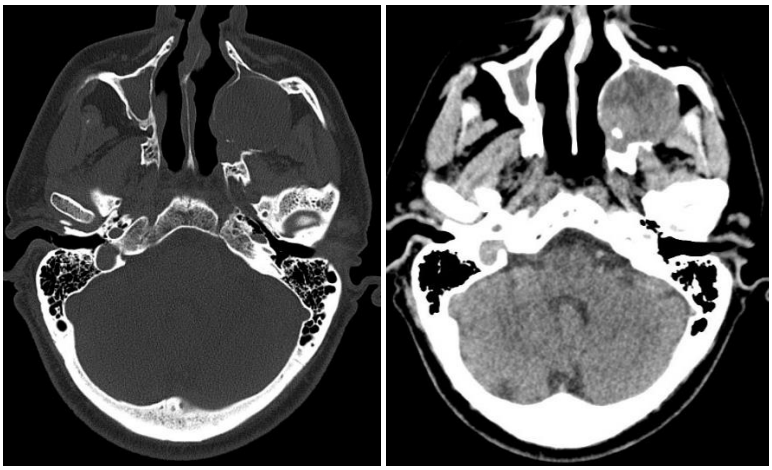

Figure 1: Axial CT in soft tissue and bone windows showing the maxillary sinus “blown out” appearance with ipsilateral extension to the pterygoid process

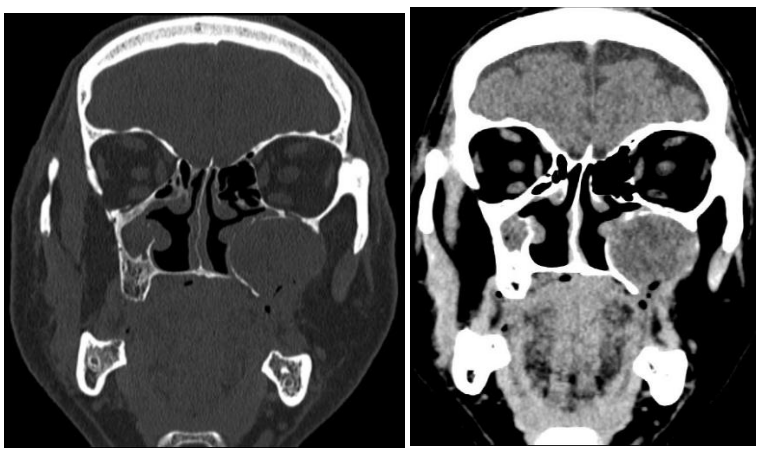

Figure 1 : Coronal CT demonstrates inferior extension to the oral cavity floor with an oroantral communication

An incisional biopsy was performed on the polypoid tissue accessible through the oroantral defect at the time of the initial extraction. Histopathological analysis confirmed the diagnosis of unicystic ameloblastoma. Given the extensive nature of the lesion and its invasion into the pterygopalatine/pterygoid region, the patient was scheduled for radical surgical resection with safety margins to ensure complete removal of the neoplasm.

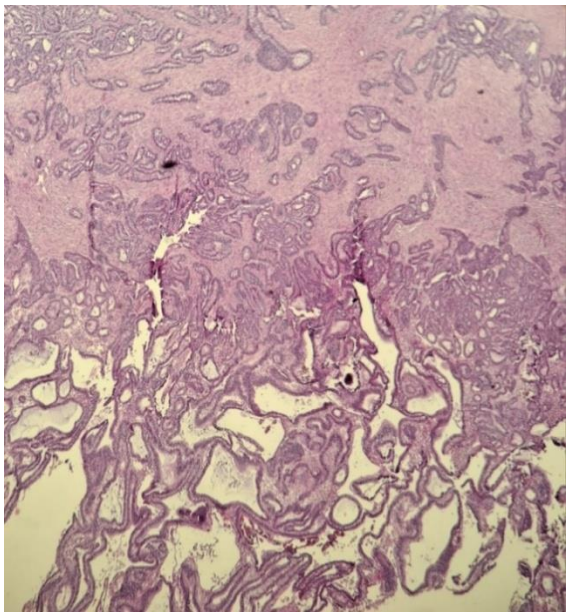

Figure 2 : Photomicrograph illustrating the histopathological features of ameloblastoma with solid, plexiform, and macrocystic growth patterns. Hematoxylin and eosin staining (H&E), original magnification ×10

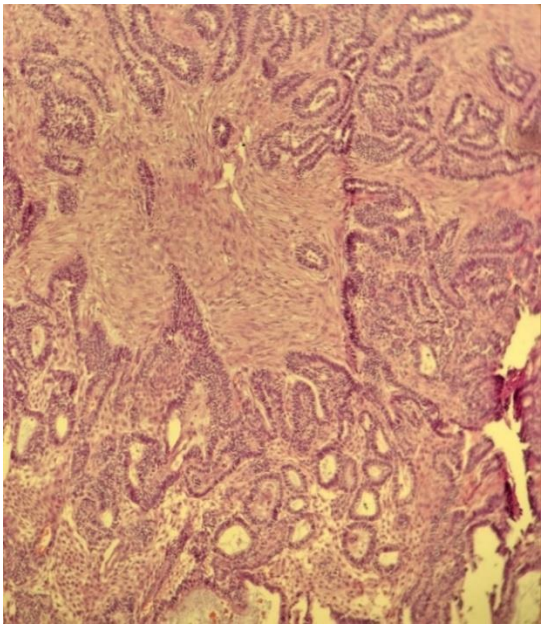

Figure 3 : Histopathological features of ameloblastoma: characterized by anastomosing cords and trabeculae of odontogenic epithelial cells dispersed within a loose connective stroma containing inflammatory cells. Haematoxylin and eosin staining (H&E), original magnification ×40

DISCUSSION

This case illustrates the deceptive and aggressive nature of maxillary unicystic ameloblastoma (UA), a pathology that can remain clinically silent until unmasked by a routine dental procedure. While ameloblastomas are predominantly found in the mandible (with a mandible-to-maxilla ratio of up to 13:1), the maxillary location poses a significantly higher risk to the patient due to anatomical factors [1,4]. As noted by Singh et al. and Agani et al., the maxilla is composed of thin cortical plates and cancellous bone, which offer minimal resistance to tumor expansion [1,4]. In our patient, this lack of resistance allowed the tumor to grow unobtrusively, filling the entire maxillary sinus

and destroying the sinus floor before any facial asymmetry or significant pain prompted a specific investigation. The extraction of the third molar (tooth 28) was the precipitating event that revealed the underlying pathology, presenting as a persistent oroantral communication (OAC) with polypoid tissue.

Radiographically, the distinction between a unicystic ameloblastoma and a benign odontogenic cyst (such as a dentigerous or radicular cyst) is notoriously difficult. Imase and Watanabe emphasize that UAs frequently present as well-defined, unilocular radiolucencies associated with impacted teeth, mimicking the appearance of a dentigerous cyst [7]. This mimicry represents a major diagnostic pitfall. In the present case, the lesion likely appeared as a pericoronal radiolucency associated with tooth 28 on initial dental radiographs. However, the subsequent CT findings—specifically the "blown-out" appearance of the sinus walls and the destruction of the lateral and posterior boundaries—were indicative of a more locally aggressive process than a simple cyst. This aligns with Viswanathan's observation that when typical cystic appearances occur in atypical clinical settings (or with aggressive bony expansion), the differential diagnosis must be broadened to include neoplasms [6].

A critical finding in this case was the posterior extension into the pterygoid space with separation of the pterygoid plates. This radiological sign is a marker of significant disease progression. Unlike mandibular lesions, which are often confined by thick cortical bone, maxillary ameloblastomas can spread rapidly into the retromaxillary and pterygopalatine regions [4]. Kalmegh et al. note that while UAs are generally considered less aggressive than solid multicystic ameloblastomas, the "mural" subtype (where tumor islands infiltrate the cyst wall) behaves aggressively and requires radical resection rather than simple enucleation to prevent recurrence [2]. The separation of the pterygoid processes in our patient necessitated a more extensive surgical approach to ensure clear margins in a region that is anatomically complex and difficult to access.

Finally, this case underscores the importance of histopathological evaluation for any tissue associated with non-healing extraction sites or unexpected oroantral communications. While the initial clinical presentation was a complication of extraction, the underlying cause was a neoplasm. As highlighted by Alarjani, the biological behavior of UAs can vary, and long-term follow-up is mandatory due to the risk of recurrence, which can occur years after the initial treatment [5].

CONCLUSION

Maxillary unicystic ameloblastoma represents a significant diagnostic challenge due to its rarity and its tendency to mimic benign odontogenic cysts. As demonstrated in this case, the thin cortical plates of the maxilla allow for rapid, silent expansion into the maxillary sinus and pterygoid space, often with minimal clinical symptoms until an event—such as a tooth extraction—unmasks the pathology. Clinicians must maintain a high index of suspicion when routine extractions result in unexpected oroantral communications or reveal polypoid soft tissue. In such scenarios, relying solely on periapical or panoramic radiography is insufficient. Advanced multi-modality imaging, specifically Computed Tomography (CT), is mandatory to delineate the true extent of bony destruction and soft tissue invasion. Early identification and aggressive surgical management are critical to preventing recurrence in the complex anatomy of the midface.

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