CASE REPORT

ENHANCED HEALING WITH PLATELET-RICH FIBRIN AND BONE GRAFT FOLLOWING SURGICAL MANAGEMENT OF UNICYSTIC AMELOBLASTOMA: A CASE REPORT

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ABSTRACT

Unicystic Ameloblastoma (UA) is a rare odontogenic tumor displaying milder behavior and lower recurrence than solid variants. Although pediatric cases are uncommon (10–15%), UA is the predominant subtype in children.

We report a 12-year-old female presenting with a painless right mandibular swelling of six months' duration. Radiographs revealed a well-defined unilocular radiolucency with an "inverted-pear" appearance, tooth displacement, and cortical perforation. Treatment involved surgical enucleation, with healing augmented by platelet-rich fibrin (PRF) and hydroxyapatite bone graft. Histopathology confirmed the diagnosis of UA.

Follow-up at six and twelve months demonstrated satisfactory healing, excellent bone fill, and no recurrence. Because pediatric UA frequently mimics odontogenic cysts radiographically, CBCT and histopathology are essential for accurate diagnosis. This case supports conservative surgery with biologic grafting as an effective treatment for luminal variants in growing children.

KEY WORDS

Unicystic Ameloblastoma, pediatric ameloblastoma, mandible, PRF, CBCT, hydroxyapatite.

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INTRODUCTION

Ameloblastoma is a benign but locally aggressive odontogenic tumour arising from remnants of the dental lamina, enamel organ, or odontogenic epithelium. Ameloblastoma is characterised as a true neoplasm originating from enamel organ-type epithelium, exhibiting proliferative activity without progressing to full differentiation capable of enamel formation. It has been described very aptly by Robinson as being a tumour that is 'usually unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent'.[3] A systematic review and meta-analysis estimated the global incidence of ameloblastoma at 0.92 per million person-years, highlighting its rarity on a population level.[1] Despite low global incidence, ameloblastoma accounts for a significant proportion of odontogenic tumours in Asian and African populations.

In India, ameloblastoma represents around 4-5% of oral tumours and 13-15% of odontogenic tumours.[2] Regional analyses confirm a predilection for the posterior mandible, and a notable proportion of cases present in younger patients. Unicystic Ameloblastoma (UA) was first described by Robinson and Martinez (1977) as a cystic variant with ameloblastomatous epithelium lining the cavity.[3] It constitutes 10-15% of all ameloblastomas[4] and is especially prevalent in children and adolescents.

Recent demographic studies (China, 2024) show that Unicystic Ameloblastoma accounts for approximately 19.6% of ameloblastoma cases in large tertiary centres.[5] A 2024 Indian retrospective series (2006–2024) found Unicystic Ameloblastoma to present predominantly as a unilocular radiolucency in 70% of cases, frequently leading to initial misdiagnosis as an odontogenic cyst.[6]

Histopathologically, Unicystic Ameloblastoma is subclassified into:

- Luminal
- Intraluminal / Plexiform
- Mural

This classification guides management, as luminal and intraluminal variants respond well to conservative enucleation, whereas mural variants behave moreaggressively.[7]

The present case report describes a rare case of Unicystic Ameloblastomain a 12-year-old child, managed with enucleation and biologically assisted grafting, with excellent postoperative healing.

CASE REPORT

A 12-year-old female presented with a chief complaint of swelling in the lower right back tooth region of the jaw since last six months. The swelling had developed gradually, was painless, and caused mild facial asymmetry. There was no associated history of trauma, systemic illness, or similar familial lesions.

Extraoral examination revealed a mild, diffuse enlargement over the right lower facial region, with normal overlying skin and no palpable lymphadenopathy.

Intraoral examination revealed a soft, fluctuant, non-tender swelling covered by normal mucous membrane that was noted extending from the distal aspect of tooth 43 to the mesial aspect of tooth 45, accompanied by buccal cortical expansion, without any discharge [Figure 1]. 43 and 44 were tender on percussion.

Radiographic evaluation using an orthopantomogram demonstrated a well-defined unilocular radio lucency with an inverted pear-shaped configuration in relation to the 43, 44, 45 region, associated with displacement of the teeth but without evidence of root resorption [Figure2]. Buccal Cortical plate Perforation was found on CBCT examination.

Under Aseptic conditions and local anaesthesia,a Crevicular Incision was given with respect to distal aspect of 31 tothe Mesial aspect of 46. An envelope flap was raised. The cystic lining was seen and the lesion was removed in toto, minimising the chances of recurrence. The cavity was thoroughly curetted and irrigated with Betadine and Normal saline to eliminate residual epithelium [Figure 3]. 5ml of blood

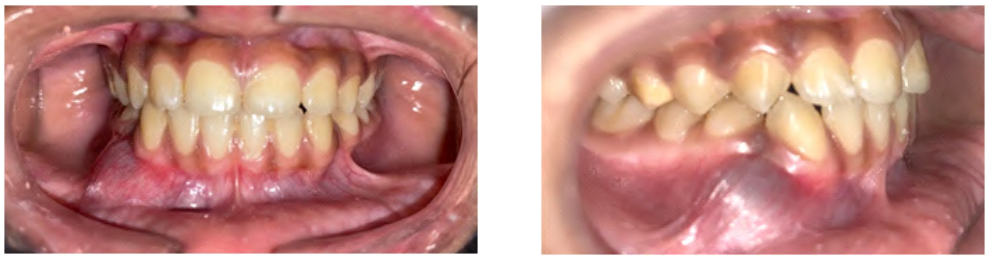

Figure 1: Intraoral pre-operative picture

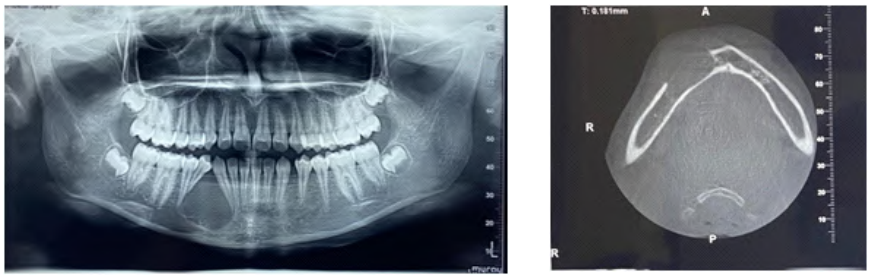

Figure 2: OPG demonstrating well well-defined unilocular radiolucent lesion in the right mandible and CBCT demonstrating buccal cortical plate perforation

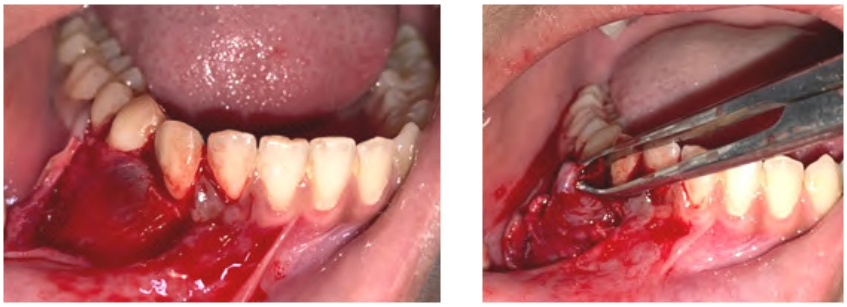

Figure 3. Intraoperative enucleation

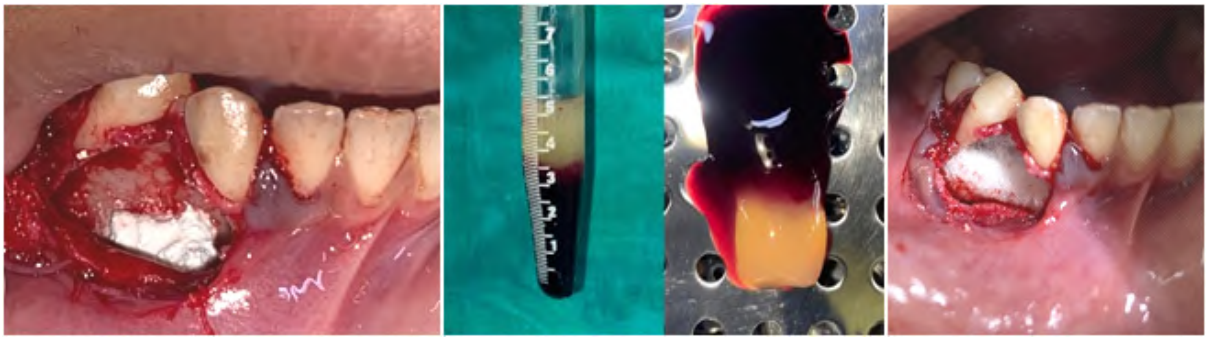

Figure 4. Placement of PRF, hydroxyapatite and Abgel

Figure 5:Intraoral picture after suture placement

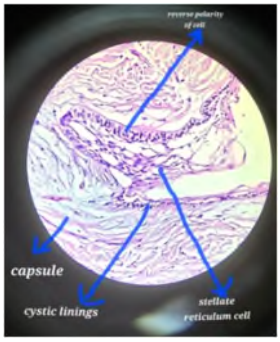

Figure 6: H&E stained Section Showing Ameloblastomatous changes

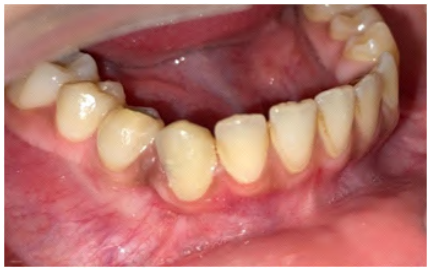

Figure 7: 1-month Post operative Intraoral picture

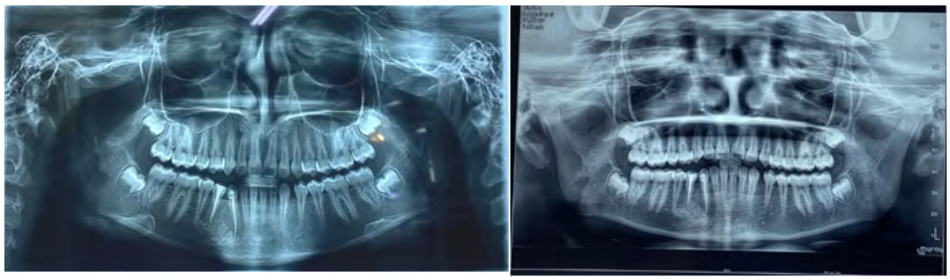

Figure 8: 6 months and 1 year follow up OPG showing bone formation

was drawn from the Median cubital vein of the left hand.

Platelet-rich fibrin (PRF) was prepared by centrifugation at 2300 rpm for eight minutes, was placed within the bony cavity to promote regenerative tissue response. Modified Hydroxyapatite granules mixed with autologous blood were subsequently packed into the defect to restore lost volume and support osteoconductive activity. An AbGel scaffold was then placed to facilitate hemostasis [Figure 4]. Bony Spicules were removed with a Bone Rongeur, and sharp bony margins were smoothened using a bone file. The surgical site was finally secured with primary closure using sutures [Figure 5].

On Histopathological examination, the H&E-stained sectionrevealed a cystic lesion lined by odontogenic epithelium exhibiting features characteristic of ameloblastoma. The lining epithelium was composed of a basal layer of tall columnar cells arranged in a palisaded pattern, with nuclei showing reverse polarisation away from the basement membrane. The supra-basal cells were loosely arranged and resembled stellate reticulum-like cells. The underlying cyst wall consisted of fibrous connective tissue without evidence of intramural tumour proliferation. Based on these findings, a diagnosis of Unicystic Ameloblastoma was established [Figure 6].

At the 1-month follow-up, the patient demonstrated good soft tissue healing with no evidence of infection or discharge [Figure 7]. At the 6-month follow-up, there was significant bone fill at the surgical site with no signs of recurrence, and the patient remained completely asymptomatic [Figure 8].

DISCUSSION

Unicystic Ameloblastoma is a distinct variant differing in clinical aggressiveness, radiographic

presentation, histopathology, and treatment needs compared to solid ameloblastomas. Its occurrence in children makes early and accurate diagnosis crucial to preserving jaw growth.

Global incidence studies indicate ameloblastoma is one of the rarest odontogenic tumors, making up less than 1 per million cases each year.[1] Indian meta-analyses indicate higher prevalence, possibly due to genetic and environmental factors.[2] In pediatric populations, Unicystic Ameloblastoma constitutes 10-15% of ameloblastomas,[4]with mandibular molar-ramus region most frequently affected.

Several theories have been proposed to explain the pathogenesis of Unicystic Ameloblastoma, including ameloblastic transformation of the reduced enamel epithelium, neoplastic change within a pre-existing odontogenic cyst, proliferation from remnants of the dental lamina, and mutation-driven epithelial proliferation, with BRAF V600E mutations being frequently reported in Unicystic Ameloblastoma.[8] Some authors suggest that the luminal and intraluminal variants may represent aborted forms of solid ameloblastoma, which may account for their relatively milder biological behavior.

Radiographically, Unicystic Ameloblastoma often mimics other odontogenic cysts such as dentigerous cysts, radicular cysts, and odontogenic keratocyst. Commonly observed features include a unilocular radiolucency, well-circumscribed corticated margins, tooth displacement, and occasional cortical perforation. In the present case, the characteristic inverted-pear-shaped radiolucency with buccal cortical plate perforation closely corresponded with patterns described in previous reports.[7]

According to Ackermann's classification, Unicystic Ameloblastoma is categorized into luminal (Group1), intraluminal or plexiform (Group2), and mural (Group3) variants. Luminal and luminal-intraluminal variants demonstrate relatively low recurrence rates of less than 10%, whereas mural variants have been associated with significantly higher recurrence rates, ranging from 30% to 35%.[4]

Management strategies are largely guided by the histopathological subtype. For luminal and intraluminal Unicystic Ameloblastoma, enucleation remains the standard treatment modality, with low recurrence rates when the cystic lining is removed intact. The high regenerative capacity of pediatric bone further supports conservative surgical management in children. Although adjunctive agents such as Carnoy's solution have been shown to reduce recurrence, their use in pediatric patients is approached cautiously due to the risk of damage to adjacent neurovascular structures.

For nearly two decades, platelet concentrates have been employed to enhance the repair and regeneration of oral tissues. The advent of platelet-rich fibrin (PRF) as an autologous regenerative biomaterial has marked a significant milestone. Within the fibrin scaffold, platelets serve a dual function by contributing to both hemostasis and the biological processes of wound healing. They act as reservoirs of essential growth factors, releasing a sustained concentration of bioactive molecules that facilitate cellular migration from adjacent tissues, stimulate cell proliferation and differentiation, and enhance morphogenesis. These mechanisms collectively promote effective regeneration of both bone and soft tissues.[9]

Nisha et al also used PRF to facilitate healing in management of peripheral ameloblastoma, although they concluded that the clinical results obtained cannot be generalized; clinical trials are required to establish the role of PRF in the surgical management of PA and other lesions.[10]

Grigolato et al concluded that Magnesium-enriched hydroxyapatite used as bone substitute in a mandibular defect due to ameloblastoma excision showed an effective bone regeneration at 25 months follow-up, demonstrating an excellent biocompatibility and a high osteo-integration property.[11]

The use of platelet-rich fibrin (PRF) and hydroxyapatite (HA) as adjuncts in the present case was based on their documented regenerative benefits. PRF promotes neo-angiogenesis, accelerates bone regeneration, and reduces postoperative infection, while hydroxyapatite provides osteo-conduction, maintains space, and prevents soft tissue collapse. Previous studies have demonstrated accelerated and more predictable healing when PRF is combined with HA grafts.

Overall, the present case shared several features commonly reported in Unicystic Ameloblastoma series, including an age comparable to the average pediatric population affected, a unilocular radiolucency as seen in approximately 70% of cases,[6] and minimal clinical symptoms despite extensive bone involvement. Complete enucleation supplemented with PRF and hydroxyapatite resulted in uneventful healing, consistent with outcomes reported in the literature for non-mural Unicystic Ameloblastoma.

CONCLUSION

This case demonstrates that Unicystic Ameloblastoma should be considered in the differential diagnosis of unilocular radiolucencies in children. Comprehensive imaging with CBCT and confirmatory histopathology are mandatory for diagnosis. Conservative enucleation with biologic augmentation is effective for luminal/intraluminal subtypes and offers excellent outcomes with preservation of mandibular growth.

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