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Solid Ameloblastoma Involving Right Parasymphyseal Region: A Case Report

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Abstract
The Solid ameloblastoma is the most common histological variant of ameloblastoma, accounting for the majority of jaw lesions. It is a benign odontogenic epithelial tumor characterized by slow but persistent growth, local invasiveness, cortical bone expansion, and a high propensity for recurrence if inadequately treated. Although it most frequently occurs in the posterior mandible, its occurrence in the anterior mandibular region is relatively uncommon. This report describes a case of solid ameloblastoma in a 25-year-old female who presented with a painless swelling in the right anterior mandibular region. Radiographic examination revealed an expansile multilocular lesion associated with root divergence, root resorption, and cortical plate destruction. Histopathological examination confirmed solid ameloblastoma. The lesion was managed by surgical excision along with extraction of the involved teeth, followed by postoperative follow-up. No recurrence was observed during the minimum follow-up period of one year.

Keywords: *Solid Ameloblastoma, Conventional Ameloblastoma, Jaw Neoplasm, Odontogenic tumor, Anterior mandible*

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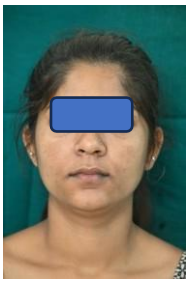
I. INTRODUCTION

Ameloblastoma is a benign epithelial odontogenic tumor of the jaws characterized by slow growth, local invasiveness, and a high tendency for recurrence. It is one of the most common odontogenic tumors and accounts for approximately 1% of oral tumors.[1,2] Conventional ameloblastoma, previously termed solid/multicystic ameloblastoma, is the most common type and occurs predominantly in the posterior mandible, although the maxilla may also be involved.[3] Clinically, it usually presents as a slow-growing, painless swelling, while radiographically it commonly appears as a well-defined unilocular or multilocular radiolucency, which may exhibit a characteristic "soap-bubble" or "honeycomb" appearance, often producing cortical expansion, tooth displacement, and root resorption.[2,3]

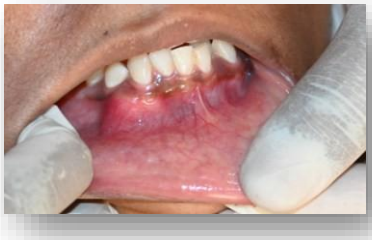
Histologically, conventional ameloblastoma is characterized mainly by follicular and plexiform patterns, with other variants including acanthomatous, granular cell, basal cell, and desmoplastic patterns. [4] The tumor is thought to arise from odontogenic epithelial remnants, including dental lamina and other remnants associated with tooth development. Molecular studies have demonstrated frequent alterations in the MAPK(Mitogen-Activated Protein Kinase) pathway, particularly the BRAF V600E(B-Raf Proto-Oncogene, Valine 600 → Glutamic Acid substitution) mutation, which contributes to tumor proliferation and progression.[5] Although benign in histology, conventional ameloblastoma can show extensive local bone destruction and has a significant risk of recurrence, particularly following inadequate conservative treatment.[2,5] Therefore, accurate diagnosis through clinical, radiographic, and histopathological evaluation is essential for appropriate management.

II. CASE REPORT

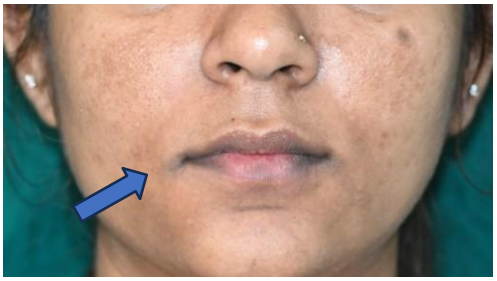
A 25-year-old female patient reported to the Department of Oral Medicine and Radiology with a chief complaint of painless swelling in the lower right front gum region for the past 3 months. The swelling was insidious in onset and gradually progressive, with no associated symptoms. No history of paraesthesia of lip. The patient's primary concern was the esthetics appearance of the swelling.

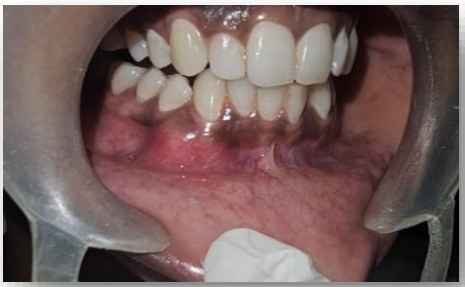


The patient gave a history of trauma five years back. The patient's past dental history revealed that she had visited a private dental practitioner two months ago, who referred her to the hospital for further evaluation and management.
The patient's past medical, surgical, family histories were non contributary. No adverse or parafunctional habits was noted.
On extra oral examination, mild facial asymmetry was observed involving the right lower third of the face. On palpation, the right submandibular lymph node was palpable, solitary, oval in shape, approximately 1 × 1 cm in size, firm in consistency, mobile, and tender.
On Intraoral Examination, there was an Obliteration of the right lower labial vestibule present in relation to teeth 43 and 44 region.



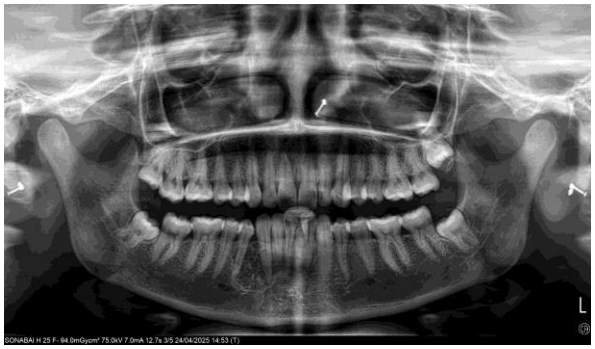
On hard tissue examination, Angle's Class II malocclusion associated with an anterior traumatic bite and crowding of the mandibular anterior teeth.
A solitary bony swelling was seen with respect to marginal, attached gingiva adjacent to teeth 43 and 44 causing vestibular obliteration of size 1×1cm, round in shape with mild labial and lingual cortical expansion. The overlying mucosa appeared smooth, shiny, no secondary changes noted & borders were well defined. No displacement of teeth irt 43 &44.





On palpation, all inspectory findings were confirmed on palpation. The swelling was non-tender, hard in consistency, non-compressible, immobile, and smooth in surface texture. No secondary changes or egg-shell crackling were noted. Teeth 43 and 44 were non-mobile and non-tender on percussion.
Based on the clinical findings, a provisional diagnosis of a benign expansile lesion of the right Para symphysial region in relation to teeth 43 and 44 was made.
The clinical differential diagnosis included odontogenic lesions, namely Odontogenic keratocyst and Ameloblastoma, and Non-odontogenic lesions, namely Central Ossifying Fibroma and Central Giant Cell Granuloma.
As a chair side investigation, vitality testing was done in relation to teeth 43 and 44 showed delayed response.
The patient was subjected to an orthopantomogram (OPG) as a screening radiograph where it revealed a full complement of teeth with impacted teeth 28, 38, and 48. A mixed radiolucent-radiopaque lesion was evident between teeth 43 and 44 with divergence of roots and internal curved septae. Inferior borders of the lesion demonstrated cortication.

OPG:



Intraoral Periapical Radiograph with respect to 43 and 44 showed, loss and indistinct of the periodontal ligament space in relation to teeth 43 and 44. A solitary unilateral mixed radiolucent–radiopaque lesion was observed between teeth 43 and 44, measuring approximately 2 × 3 cm. The lesion showed an ill-defined superior border with a relatively well-demarcated inferior border and demonstrated internal wispy trabecular architecture with indistinct intertrabecular spaces. Divergence of the roots of teeth 43 and 44 was also noted, along with a thick corticated peripheral border. Root resorption involving the apex of tooth 43 was present. In addition, the lesion exhibited multiple radiopaque flecks and thin, irregular curved septae, producing a multilocular "soap-bubble" appearance.

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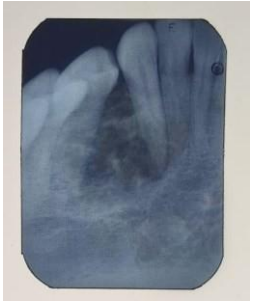
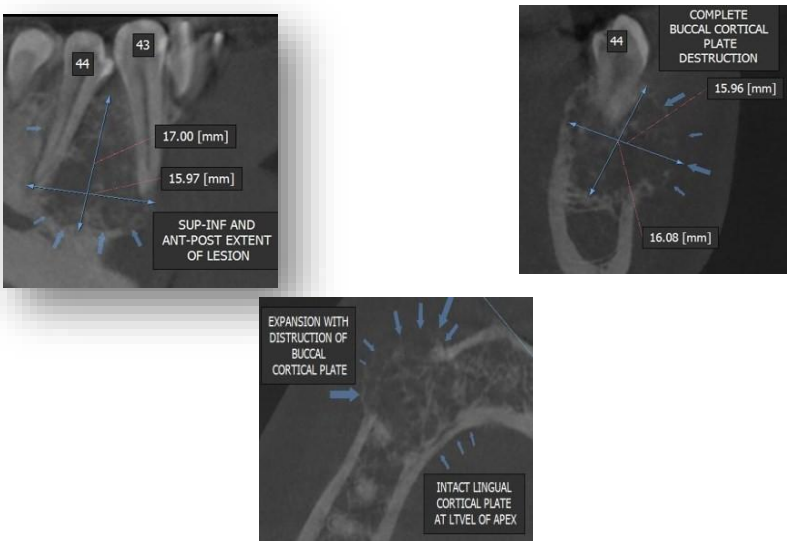


3D Imaging modality Cone Beam Computed Tomography (CBCT) taken wrt 43 and 44 with Field of view 8×8cm, Cone-beam computed tomography (CBCT) examination revealed a osteolytic lesion measuring approximately 15 × 17 mm in the periapical region between teeth 43 and 44 on coronal sections. Sagittal sections demonstrated complete destruction of the buccal cortical plate along with thinning of the lingual cortical plate. Axial sections further revealed expansion and destruction of the buccal cortical plate with associated thinning of the lingual cortical plate. In addition, destruction of the medullary bone was also noted.

CBCT:



Incisional biopsy was performed under local anaesthesia in relation to teeth 43 and 44.

Microscopic examination of haematoxylin and eosin-stained sections revealed numerous ameloblastomatous follicles within a mature fibrous connective tissue stroma. The follicles were lined by columnar basal cells exhibiting hyperchromatic nuclei with reversal of polarity. Central areas of the follicles showed stellate reticulum-like cells along with areas of squamous and basaloid differentiation.

Microscopic View: Microscopic examination of H &E-stained section.

40x Magnification

Photomicrograph show numerous ameloblastomatous follicles within fibrous connective tissue stroma

Based on the histopathological findings, a final diagnosis of solid or conventional ameloblastoma in relation to right Parasymphyseal region was established.

Surgical management involved subperiosteal excision of the lesion along with extraction of teeth 42, 43, 44, and 45. Following excision, Carnoy's solution was applied over the surgical site. Postoperatively, the patient was prescribed chlorhexidine mouthwash (10 mL) three times daily for 7 days, Antibiotics and analgesics were also prescribed. The patient was instructed to maintain a soft diet and was kept under periodic follow-up. The patient was followed up for 1 year, during which no evidence of recurrence or new lesion development was observed.

III. DISCUSSION

Ameloblastoma is usually unicentric, non-functional, intermittent in growth, anatomically benign, and clinically persistent.[6]Ameloblastoma is a common but enigmatic tumor of the jaw and constitutes about 10% of all tumors that arise in the mandible and maxilla. It is one of the most common benign odontogenic tumors.[2] It is locally invasive, slow-growing tumor of odontogenic epithelium, mainly arising from enamel tissue that has not undergone differentiation. It has high recurrence rate and a metastatic potential similar to malignant tumors.[7] It was first recognized by Cusack in 1827 and designated as an 'adamantinoma' in 1885 by the French physician Louis-Charles Malassez.[8] Term ameloblastoma was coined by Ivey and Churchill in 1930 a currently accepted term.[7]

Ameloblastoma shows a variable geographic prevalence with a global incidence of 0.92 cases per million person-years. Most epidemiological studies have revealed that ameloblastoma is either the most common or the second most common benign odontogenic tumor.[2] Higher incidence between the fourth and fifth decade of life, without predilection for any gender. Approximately 80% of all ameloblastomas are located in the posterior region of the mandible, followed by the anterior mandibular region, and posterior and anterior maxillary segments. Involvement of the sinus and nasal cavity regions is rare[8]. Ameloblastoma in children is considered rare.[9]

According to the WHO Classification of Head and Neck Tumours (5th edition, 2022), ameloblastoma is classified as a benign epithelial odontogenic tumor. The classification includes conventional ameloblastoma previously termed solid/multicystic ameloblastoma, unicystic ameloblastoma, extraosseous (peripheral) ameloblastoma, metastasizing ameloblastoma, and the newly recognized adenoid ameloblastoma. Desmoplastic ameloblastoma is now considered a histopathological subtype of conventional ameloblastoma rather than a separate entity.[8]

Conventional ameloblastoma develops from odontogenic epithelial remnants left behind after tooth development. Genetic mutations, especially BRAF V600E (60–80%), activate the MAPK pathway, causing abnormal cell growth and reduced cell death. Other mutations may involve KRAS(Kirsten Rat Sarcoma Virus *(KRAS proto-oncogene)*, NRAS (Neuroblastoma RAS Viral Oncogene Homolog), FGFR2(Fibroblast Growth Factor Receptor2), MAP2K1, SMO(Smoothened), and PTCH1.[5] The SHH(Sonic Hedgehog) pathway may also become abnormally activated, further increasing tumor growth and survival.[2] The surrounding tissue releases growth factors such as TGF-β(Transforming Growth Factor Beta), EGF(Epidermal Growth Factor), FGF(Fibroblast Growth Factor), and VEGF(Vascular Endothelial Growth Factor), which support tumor growth. Ameloblastoma cells also produce MMPs, which break down surrounding tissues and bone, while increased RANKL (Receptor Activator of Nuclear Factor Kappa-B Ligand) activates osteoclasts and causes bone resorption, leading to jaw expansion, cortical perforation, root resorption, and tooth displacement. Overall, genetic mutations and activation of these pathways cause increased tumor growth and tissue and bone destruction, resulting in a slow-growing but locally aggressive ameloblastoma with a tendency for recurrence.[9]

Clinically they are slow-growing, painless swelling of the mandible or maxilla is the most common presentation of ameloblastoma. Because of slow enlargement, early lesions are often asymptomatic and discovered incidentally on radiographs. Growth is expansile rather than infiltrative initially, leading to bone thinning and cortical expansion. Growth occurs in the buccolingual direction, resulting in significant expansion. The mean size of ameloblastomas at presentation is approximately 4 cm.[3] Pain is an uncommon symptom of conventional ameloblastoma and, when present, is usually associated with haemorrhage within or adjacent to the tumor. As the lesion enlarges, patients may develop facial deformity, malocclusion, mobility or displacement of teeth, and, in advanced cases, soft tissue invasion. Large lesions may also interfere with normal oral function, resulting in difficulty in chewing and speaking. Paraesthesia or numbness is uncommon but may occur due to compression or involvement of adjacent nerves. Maxillary ameloblastomas tend to spread more rapidly than mandibular lesions because of the thin cancellous bone of the maxilla and, if left untreated, may invade adjacent structures such as the maxillary sinus, nasal cavity, orbit, and skull base.[10]

An orthopantomogram (OPG) is usually the first imaging investigation and typically shows a well-defined unilocular or multilocular radiolucent lesion with the characteristic "soap-bubble" or "honeycomb" appearance. Common radiographic findings include cortical expansion, root resorption, tooth displacement, and association with an impacted tooth. Computed tomography (CT) is useful for assessing the size of the lesion, cortical bone destruction, cortical perforation, and extension into adjacent structures. In maxillary ameloblastomas, magnetic resonance imaging (MRI) provides better evaluation of soft tissue involvement and extension into the maxillary sinus, orbit, or skull base. Although the clinical and radiographic features are suggestive, histopathological examination of a biopsy specimen is essential for confirming the diagnosis of conventional ameloblastoma.[11]

The differential diagnoses included both odontogenic and non-odontogenic lesions. Odontogenic keratocyst (OKC) usually presents as a slow-growing, painless swelling and radiographically appears as a well-

defined unilocular or multilocular radiolucency, often with minimal cortical expansion. Ameloblastoma commonly presents as a slow-growing, painless swelling and typically appears as a well-defined unilocular or multilocular radiolucency, often showing cortical expansion, root resorption, and tooth displacement. Central ossifying fibroma generally presents as a slow-growing, painless, well-circumscribed swelling and may appear radiolucent in the early stage and become mixed or radiopaque as calcification increases, often causing cortical expansion and displacement of adjacent teeth. Central giant cell granuloma (CGCG) usually presents as a painless swelling and appears as a unilocular or multilocular radiolucency, which may cause cortical expansion, tooth displacement, and root resorption. Although these lesions may have overlapping clinical and radiographic features, histopathological examination is essential for definitive diagnosis.[12]

Conventional ameloblastoma previously known as solid/multicystic ameloblastoma most commonly exhibits two characteristic histological patterns: follicular and plexiform. In the follicular pattern, the odontogenic epithelium is arranged as discrete islands or follicles within a fibrous connective tissue stroma. In the plexiform pattern, the epithelial cells form interconnecting strands or networks separated by connective tissue. Both patterns may coexist in varying proportions within the same tumor. In addition to these major patterns, several histological variants have been described, including acanthomatous, granular cell, basal cell, desmoplastic, and keratinizing variants. These variants generally do not alter the biological behaviour or treatment of the tumor and represent histological variations of conventional ameloblastoma rather than separate entities.[13]

Surgical resection is the standard treatment for conventional ameloblastoma. Because of its locally infiltrative nature and high risk of recurrence, wide surgical excision with 1–1.5 cm of uninvolved bony margin is generally recommended. Conservative procedures, such as enucleation, curettage, marsupialization, cryotherapy, and the use of Carnoy's solution, preserve more tissue but are associated with a significantly higher risk of recurrence due to residual microscopic tumor cells. Tumor recurrence is mainly due to microscopic infiltration of tumor cells beyond the radiographic margins into the cancellous bone. Following tumor removal, bone reconstruction and dental rehabilitation are important to restore function, aesthetics, speech, and mastication. Long-term follow-up is essential because recurrence may occur even years after treatment.[2]

Recent advances in molecular biology have identified the BRAF V600E mutation as a potential therapeutic target, and targeted therapies have shown promising results in selected cases. However, further studies are required to establish the long-term efficacy and safety of these non-surgical treatment modalities.[14]

The prognosis of conventional ameloblastoma depends on its size, location, extent, histological type, surgical margins, and treatment. Although it is a benign tumor, it is locally aggressive and can cause severe bone destruction if untreated. Recurrence is higher after conservative treatment (40–90%), while radical surgery with adequate margins reduces recurrence to about 10–15%. Most recurrences occur within the first 5 years, but late recurrence can also occur. Therefore, long-term clinical and radiographic follow-up for at least 10 years is recommended. Maxillary ameloblastomas generally have a poorer prognosis because they can spread easily into nearby vital structures. [2]

IV. CONCLUSIONS

Ameloblastoma is the most common benign odontogenic tumor of the jaws, characterized by slow growth, local invasiveness, and a high risk of recurrence if inadequately treated. The present case of conventional (solid) ameloblastoma in the mandibular canine–premolar region demonstrated the characteristic clinical, radiographic, CBCT, and histopathological features that established the diagnosis.

Early recognition and accurate diagnosis using clinical examination, advanced imaging, and histopathological evaluation are essential for appropriate treatment planning. Radical surgical resection with adequate margins remains the treatment of choice to minimize recurrence, while recent advances in molecular genetics have introduced the potential role of targeted therapies in selected cases, offering promising future treatment options.

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