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CASE REPORT

A Large Mixed Radiolucent-Radiopaque Lesion In The Mandible- A Nobel Diagnostic Approach

RASTOGI S*, NIJHAWAN S, MODI M***, KUMAR A****, ASLAM N *****, LATHEEF F******

ABSRACT

Ameloblastoma is a true neoplasm of the enamel organ type tissue which does not undergo differentiation to the point of enamel formation. The term unicystic is derived from the macroscopic and microscopic appearance of the lesion. It is a well-defined, often large monocytic cavity with a lining focally, but rarely entirely composed of odontogenic (ameloblastomatous) epithelium. Predominant radiographical patterns for Unicystic Ameloblastoma are unilocular, scalloped, macromultilocular, pericoronal, interradicular, or periapical expansile radiolucencies. Some investigators believe that Unicystic Ameloblastoma arises from preexisting odontogenic cysts, in particular, from the dentigerous cyst, while others arise de novo. Immunohistochemical markers like lectins (Ulex europaeus agglutinin I and Bandeirea simplicifolia agglutinin I) and proliferating cells (proliferating cell nuclear antigen and Ki-67) may assist in their differential diagnosis. Hence, in our case report, we have tried to discuss in detail about the clinical, radiographical and histopathological features with differential diagnosis. The immunohistochemical importance has also been discussed.

Key Words: Unicystic ameloblastoma, radiolucent, radiopaque, PCNA, ki-67

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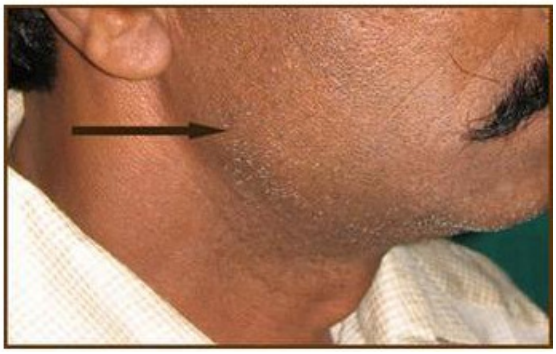
Introduction

Tumors and tumor-like growths arising from odontogenic tissues constitute a heterogenous group of particularly interesting lesions, as they display the various inductive interactions that normally occur among the embryological components of the developing tooth germ. In humans, tumors of odontogenic tissues are comparatively rare, comprising of about 1% of all jaw tumors. *Ameloblastomas* constitute almost half (48.9%) of the odontogenic tumors with female-to-male and maxilla-to-mandible ratios of 1:1.7 and 1:8, respectively. The mean (SD) age of the patients in this group was 15.1 (± 3.0) years (range, 4–19 years), with most patients (49%) in the age group of 3 years. Multicystic/solid and unicystic variants were diagnosed in 40 (89%) and 5 (11%) cases respectively. This case report described the systematic approach towards the diagnosis and treatment of this unique entity.

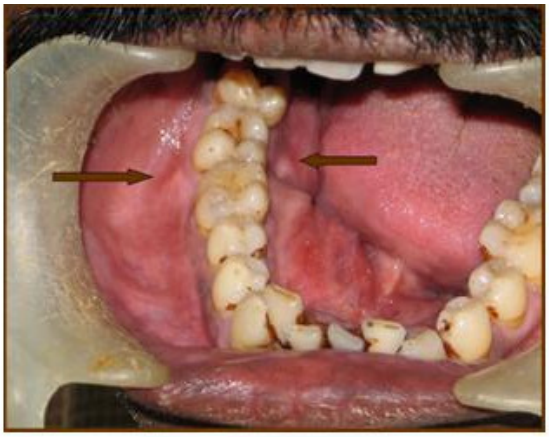
Case Report

A 46yr old male patient reported to our Outpatient department with a gradually increasing painless swelling on the right lower third of the face for the past one year. History revealed that the swelling was about 2x2cm when first noticed, which gradually increased to the present size of 6x7cm. There was no history of anesthesia or paraesthesia. On examination, the swelling was found to be confined to right lower jaw [Table/Fig 1]. Buccal and lingual cortical plate expansion was appreciated irt right canine to right third molar in the lower jaw , with palpable stony hard mass in the buccal and lingual vestibule

in the same region and was non-tender. Mucosa over the swelling was normal, with no secondary changes [Table/Fig 2].A provisional diagnosis of benign odontogenic neoplasm was made.



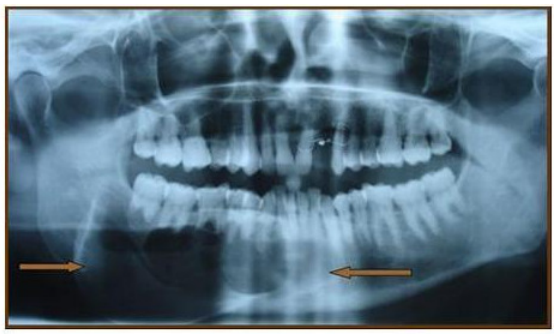
(Table/Fig 1) Extra-Oral Swelling Measuring About 6x7cm Present On The Right Lower Third Of Face.



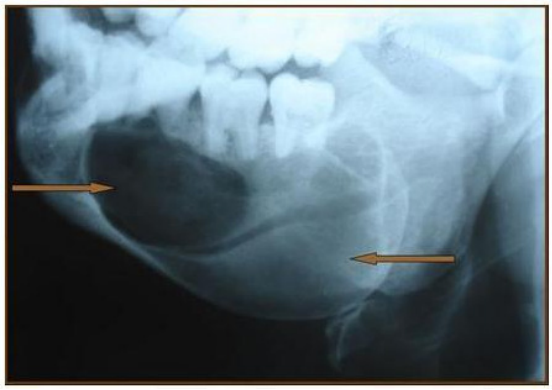
(Table/Fig 2) Obliteration Of Vestibular Space With Buccal And Lingual Cortical Plate Expansion

Investigations

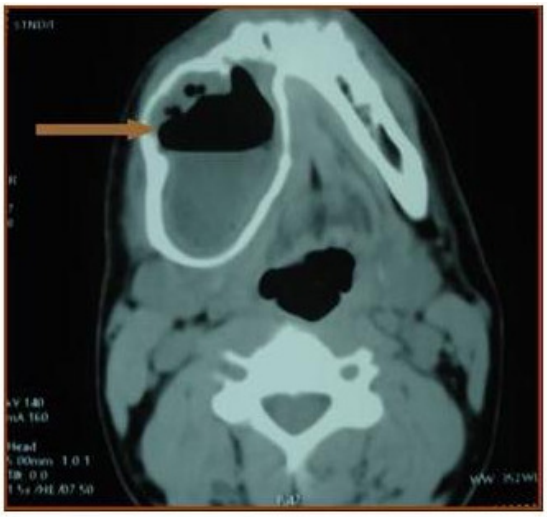
Vitality test of 31 to 34 and 41 to 48 showed normal response. On fine needle aspiration, a white cheesy material was aspirated, whose protein content was found to be 3.2gm/dl. The orthopantamograph revealed an expansile, mixed, radiolucent-radiopaque lesion in the body of the mandible, extending from 41 to 48 and which surprisingly had crossed the midline and extended upto 34 [Table/Fig 3] . The borders of the lesion were well-defined on all aspects, except distal to 48. The inferior alveolar canal was displaced inferiorly, with the resorption of apical third of roots irt lower central incisor to lower canine on the left side and from lower right side central incisor to lower right third molar on the intra-oral periapical radiograph. Fine radiopaque flecks along with loss of the trabeculae, was evident within an intense well-defined radiolucency irt lower right second premolar to lower right second molar [Table/Fig 4].Computed tomography on axial section at the level of the mandible revealed an expansile lesion within the mandible, with hypodense areas on the anterior half of the lesion [Table/Fig 5] . A 3-D reconstructed image of CT revealed multiple septae within the lesion [Table/Fig 6] .



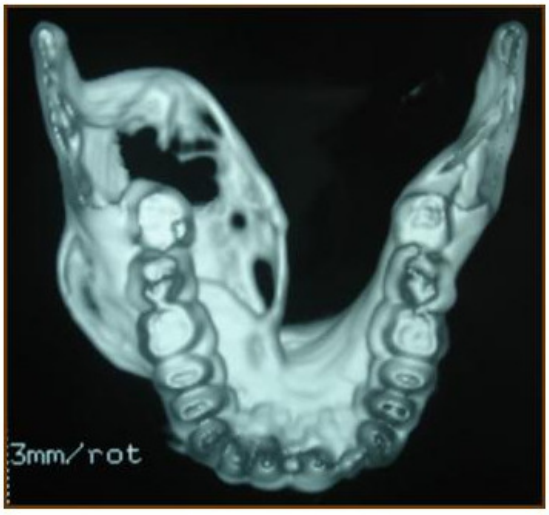
(Table/Fig 3) Orthopantamograph Revealing Mixed Radiolucent-Radioopaque Lesion Present In The Right Body Of Mandible Extending On To Opposite Side



(Table/Fig 4) Lateral Oblique View Of Right Mandible Showing Radioopaque Flecks Within Radiolucency, Downward Displacement Of Inferior Alveolar Canal And Resorption Of Roots Of Adjacent Teeth



(Table/Fig 5) Expansile Lesion Present In Body Of Mandible On Right Side, Crossing Midline And Extending Towards Opposite Side On Axial Section Of Computed Tomography.



(Table/Fig 6) 3-D Reconstructed Computed Tomography Revealed A Destructive Lesion With Multiple Septae And Perforation

Differential Diagnosis

The first in the list of differential diagnosis was Ameloblastoma, as it is the most common benign odontogenic tumor occurring in middle aged individuals and in the mandibular posterior region. It also presents with buccal and lingual cortical plate expansion and presents with multilocular radiolucency, but it rarely shows flecks of calcification. The next entity that was considered was Calcifying epithelial odontogenic tumor (CEOT), as this too has a predilection for the age range of 30-50 yrs and for the posterior areas of the mandible. CEOT presents as slow growing lesions and also reveals focal areas of calcification within the lesion, on radiographs. Odontogenic Keratocyst (OKC), commonly occurs in the mandibular posterior region and it has a tendency to grow in the anterior-posterior direction within the medullary cavity of the bone without causing obvious bone expansion. The margins tend to be densely sclerotic, with a scalloped outline and rarely cause root resorption of the adjacent teeth. The other lesions that can be considered for differential diagnosis are Central giant cell granuloma and Odontogenic myxoma [1],[2],[3],[4],[5].Central giant cell granuloma (CGCG) presents with multilocular radiolucency and often shows the resorption of the root surfaces of the adjacent teeth, as seen in our case [1],[2],[3]. The main characteristics which are useful for differentiating Ameloblastoma and CGCG are: Ameloblastomas tend to occur in an older age group and more often in the posterior mandible, and have coarse, curved, well-defined trabeculae. CGCG typically occurs anterior to the mandibular first molar and often crosses the midline [3]. CGCGs may have sclerotic borders. Internally, the lesion may be radiolucent or granular, or may contain thin wispy septa. CGCGs show uneven expansion or are undulating in nature, which may give the appearance of a double boundary [2].Odontogenic Myxoma in the early stage has an osteoporotic appearance, consisting of multilocular radiolucency with well-developed locules. In the second stage of break-out or in the destructive phase, it is characterized by loss of locules with significant expansion. These lesions may cross the midline and may cause root resorption and tooth displacement [1],[2],[3],[4],[5] [Table/Fig 7] .(Table 1-2)

(Table/Fig 7) Table 1 :Differentiating Clinical Features Between Ameloblastoma, CEOT, OKC, CGCG and Odontogenic Myxoma:

Features	Ameloblastoma	CEOT	OKC	CGCG	Odontogenic Myxoma
INCIDENCE	11% of Odontogenic tumors	0.4-3% of Odontogenic tumors	7-11% of Odontogenic Cysts	< 7% benign lesions of jaws	3-6% of Odontogenic tumors
AGE	≈33yrs 3rd - 4th decade	8-92yrs ≈36.9yrs	2nd-3rd decade	<20yrs	25-35yrs
SEX M: male F: female	M>F 1.1 : 1	M>F 6:5	M>F 1.7 : 1	F>M 2 : 1	M>F 3 : 2
SIGNS & SYMPTOMs	Painless swelling & Slow growth	Painless swelling & Slow growth	Painless swelling I/O drainage	Aggressive:pain, swelling, rapid growth Non-aggressive:	Asymptomatic
SITE	Mandible Posterior Ramus	Mandible: Maxilla 2:1, Premolar-molar region	Mandible Molar-Ramus	Mand>Max Ant to 1st molars May cross midline	Mand>Max Molar>PreM May cross midline

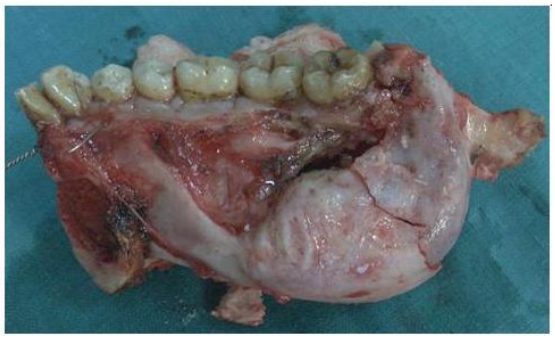
(Table/Fig 7) Table 2: Differentiating Radiographic Features Between

Ameloblastoma, OKC, CGCG, ABC and Odontogenic Myxoma:

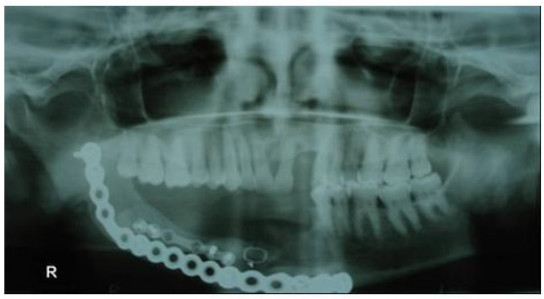
Features	Ameloblastoma	OKC	CGCG	ABC	Odontogenic Myxoma
Periphery and Shape	Smooth, Oval	Smooth, Round or Oval	Irregular/ Double boundary	Circular/ Irregular	Irregular
Borders / Margins	Well-defined & often curved	Well-corticated, unless sec infected, scalloped	Well-defined, smooth and scalloped	Well defined or ill-defined	Mand: well-corticated. Max: poorly defined
Cortical expansion	Both buccal and lingual	Minimal expansion, rare	Uneven expansion, both B & L, perforation	Balloon-like expansion of cortex	Both buccal and lingual
Internal structure	Multilocular/ Unilocular; Honeycoomb/Soap bubble	Radiolucent: common, Multilocular: curved internal septa	Multilocular, thin wispy septa	Multilocular, dense filamentous septa (wispy), converging towards centre	Multilocular, well-developed locules, Tennis racket, Step ladder
Association with impacted teeth	+ 38%	+	-	-	-
Root resorption	Extensive, knife edge pattern	Rare	Profound, irregular outline	Occasional	Rare, Cut-off/ knife-edge

Management

Surgical treatment of the right hemimandibulectomy, sparing the right condyle, was done [Table/Fig 8] (Figure7). The lesion was gently curetted out of marrow space by preserving the lingual periosteum. The lingual cortical plate was spared and one half of the genial musculature was preserved to prevent the back fall of the tongue. The surgically excised area was rehabilitated with an iliac crest graft. A bi-cortical iliac crest graft was harvested and minor adjustment in the graft was done to allow it to passively fit into the defect. An 8mm stainless steel screw was used to secure the graft on a pre-bent and adopted titanium plate (2.5mm). The patient is being followed-up for the past 1 ½yrs and the excised area shows eventful healing [Table/Fig 9]. (Figure8)



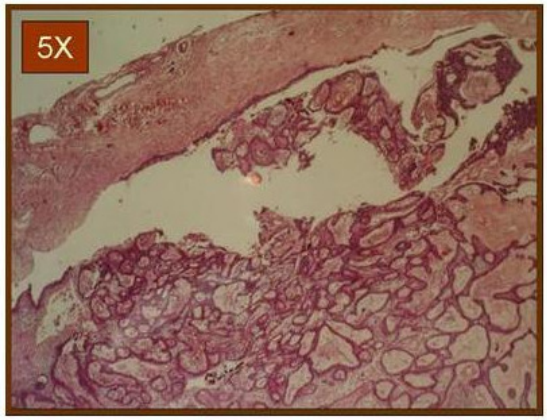
(Table/Fig 8) Fig 7: Picture Of The Excised Specimen



(Table/Fig 9) Fig 9: 5x Magnification Of The Histopathological Slide Stained With Hematoxylin And Eosin, Showed Parakeratinized Lining Epithelium Showing Keratin Flakes And Basal Cell Nuclei And Connective Tissue Stroma Containing Follicles Of Keratin And Odontogenic Epithelium

Histopathology

Microscopic examination of the specimen revealed the presence of a well encapsulated lesion, with parakeratinized lining epithelium, showing keratin flakes and basal cell nuclei. Anatomizing cords and strands of odontogenic epithelium, bound by columnar to cuboidal shaped ameloblast-like cells, surrounding more loosely arranged stellate reticulm- like cells, were noted. All these features were suggestive of Unicystic ameloblastoma with plexiform pattern (Table/Fig 10) (Figure 9) [6].

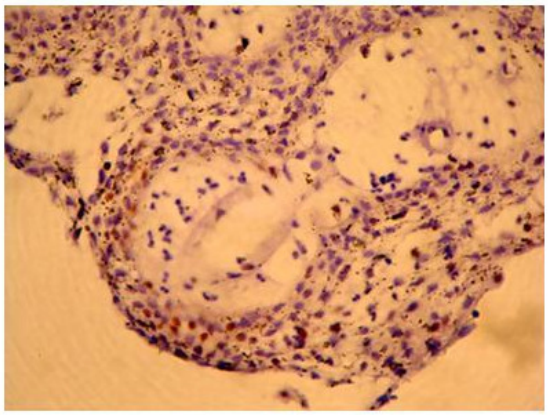


(Table/Fig 10) Fig 9: Radiograph Taken After 1½Yrs Of Follow-Up, Revealing Absence Of Signs Of Recurrence

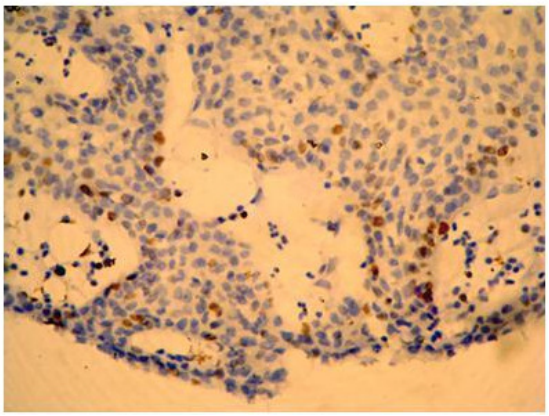
Immunohistochemistry

The Formalin tissue fixed block was treated with Antigen retrieval of Heat Induced Epitome Retreival. Primary antibodies of the Ki 67 Antigen (clone: BGX-Ki67) and PCNA (clone: PC-10) was used. The polymer-HRP

detection system and DAB chromogen was used. Positivity was considered when nuclei of the positive cells took up brown colour. Both PCNA and Ki 67 positivity was seen in few scattered tumour epithelial cells which were distributed focally, especially more in the periphery [Table/Fig 10] (Figure 10,11). Hence, even immunohistochemically, the lesion was distinguished from other odontogenic cysts.



(Table/Fig 10) Fig 10: 10x Magnification Of Immunohistochenical Stained Section Revealing Increased Positivity For Ki67



(Table/Fig 10) Fig 11: 10x Magnification Of Immunohistochenical Stained Section Revealing Increased Positivity For PCNA

Historical Review

Cusack JW (1827) first published a case, which was obviously an ameloblastoma. But, the detailed histopathological description was first made by Wedl (1853). He called the tumour,"Cystosarcoma or Cystosarcoma Adenoids", but suggested that it could have arisen from a tooth bud or from the dental lamina. Broca (1868) gave the first detailed description of solid/multicystic ameloblastoma, whereas the first histological drawing of ameloblastoma was made by Wagstaffe (1871). The detailed description of ameloblastoma was made by Falksson (1879). Malassez (1885) suggested the name "Epithelioma Adamantin". Derjinsky (1890) suggested the term "Adamantinoma" .

Ivey and Churchill (1930) used the name "Ameloblastoma". The first case of Peripheral Ameloblastoma, was made by Stanley and Krough (1959) [3],[7]. The concept of Unicystic Ameloblastoma (UA) was first introduced by Robinson and Martinez (1977), where they associated UA with dentigerous cysts, cytogenic ameloblastoma, extensive dentigerous cysts with intracystic ameloblastic papilloma, mural ameloblastoma, dentigerous cysts with ameloblastomatous proliferation and ameloblastoma developing in a radicular (or "globulomaxillary") cyst [8],[9].Gardner DG (1981) described a subtype of UA, plexiform UA, where the inner surface of the cyst may show one or several polypoid or papillomatous, pedunculated, exophytic masses, which in rare cases, fill the entire cyst lumen [10]. This subtype has also been called as intracystic, luminal or intraluminal ameloblastoma [9].

Discussion

Ameloblastoma is a true neoplasm of the enamel organ type tissue which does not undergo differentiation to the point of enamel formation. Robinson (1937) described it as unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent [3],[4]. WHO (1992) has described Ameloblastoma as a benign, locally aggressive, polymorphic neoplasm, which is presumably derived from the intraosseous remnants of the odontogenic epithelium.[5],[6] Various synonyms which are used for ameloblastoma are Adamantinoma, Adamantoblastoma, Epithelioma Adamantin, Multilocular Cyst, Adontomes embryolastiques and Epithelial odontoma [3],[7].

A recently published biological profile based on 3,677 ameloblastoma cases, has clearly demonstrated that it is no longer appropriate in any scientific study to use the diagnosis of ameloblastoma without specifying the type. Hence, based on clinical and radiographical

characteristics, histopathology, and behavioural and prognostic features, subtypes or variants of ameloblastomas can be presently distinguished as follows [7]:

1. The classic solid/ multicystic ameloblastoma (SMA)
2. The unicystic ameloblastoma (UA)
3. The peripheral ameloblastoma (PA)
4. The desmoplastic ameloblastoma (DA), including the so-called hybrid lesions

The term 'unicystic' is derived from the macro- and microscopic appearance of the lesion. It is a well-defined, often large monocytic cavity with a lining focally, but which is rarely entirely composed of odontogenic (ameloblastomatous) epithelium [10],[11]. Much confusion stems from the fact that a unicystic ameloblastoma may appear not only as a unilocular, but also as a multilocular bone defect [12]. UA can be divided into 2 categories [9]:

1. Histologically verified UAs which are associated with an unerupted tooth (dentigerous variant)
2. UAs lacking an association with an unerupted tooth (nondentigerous variant)

No data are available concerning the prevalence and incidence of UAs. The relative prevalence and incidence of UAs have been reported as between 5-22% of all types of ameloblastomas [13]. UAs are more commonly seen in younger patients, with 50% of cases being diagnosed during the second decade of life. The average age in one large series was found to be 23 years [14],[15],[16]. The gender distribution shows a slight male predilection with a male:female ratio of 1.6:1. However, when the tumor is not associated with an un-erupted tooth, the gender ratio is reversed to a male to female ratio of 1:1.8 [16].

Clinically, UA presents as a localized swelling, with occasional pain and signs of lip numbness. In cases of secondary infection, discharge or drainage can be noted [9],[15],[16]. The location of UA within the jawbone shows a marked predominance for the mandible, irrespective of the variant. The ratio of the maxilla: mandible is 1:7 for the dentigerous variant, versus 1:4.7 for the nondentigerous type [9],[15]. Radiographically, UAs have been divided into 2 main patterns: unilocular and multilocular. UAs have clear preponderance for the uniclocular pattern. This preponderance is predominantly marked for the dentigerous variant, where the unilocular to multilocular ratio is 4.3:1 and for the nondentigerous type, this ratio is 1.1:1[9],[12]. Eversole LR et al identified [6] predominant radiographical patterns for UA: unilocular, scalloped, macromultilocular, pericoronal, interradicular, or periapical expansile radiolucencies [12].

Some investigators believe that UA arises from preexisting odontogenic cysts, in particular a dentigerous cyst, while others maintain that it arises de novo. Robinson and Martinez (1997) argued that as the epithelium of odontogenic cysts and ameloblastomas have a common ancestry, a transition from a non-neoplastic to a neoplastic one could be possible, even though it occurs infrequently [8].Leider AS et al (1985) proposed three pathogenic mechanisms for the evolution of UA: [17].

The reduced enamel epithelium which is associated with a developing tooth undergoes ameloblastic transformation with subsequent cystic development

1. Ameloblastomas arise in dentigerous cysts or in other in which the neoplastic ameloblastic epithelium is preceded temporarily by a non-neoplastic stratified squamous epithelial lining.
2. A solid ameloblastoma undergoes cystic degeneration of the ameloblastic islands, with subsequent fusion of multiple microcysts and develops into unicystic lesions.

Li TJ et al (1995) made a comparison of proliferating cell nuclear antigen (PCNA) expression in the cystic tumour lining of UAs and found that all areas of the UA lining contained significantly more PCNA-positive cells than in the dentigerous cyst linings, even in areas where the epithelial morphology was similar to that of the dentigerous cyst lining. This finding was interpreted as favorable to the concept that UAs are de novo cystic neoplasms [18]. The 1992 edition of the WHO classification distinguishes between the three

histological subtypes of UA which correspond to the subgroups 1, 1.2 and 1.3. Subgroup 1 has an epithelial lining, of which some parts may show transformation to cuboidal or columnar basal cells with hyperchromatic nuclei, nuclear palisading with polarization, cytoplasmic vacuolization with intercellular spacing and subepithelial hyalinization. Subgroup 1.2 shows a combination of simple and intraluminal histological features. UA subgroup 1.2.3 shows the presence of intramural ameloblastoma tissue, as well as the subgroup 1.2. The last subgroup 1.3 exhibits a cyst with a luminal lining in combination with intramural nodules of solid / multicystic ameloblastoma [6],[9]. Hence, the case reported in this article corresponds to subgroup 1.3.

Several attempts have been made in the past to distinguish the lining of the UAs from that of odontogenic cysts. The immunohistochemical expression of blood cell carbohydrates and the epidermal growth factor receptor have shown no consistent difference between the odontogenic cysts and UA. However, immunohistochemical markers like lectins (Ulex europaeus agglutinin I and Bandeirea simplicifolia agglutinin I) and proliferating cells (proliferating cell nuclear antigen and Ki-67) may assist in their differential diagnosis.[18,19,20] Similarly, in our case, the immunohistochemical markers showed positive expression: PCNA and Ki-67 were noted more in the region of tumour islands and less in the cystic lining region. The cystic lining, particularly of OKC, typically reveals positivity for these proliferating markers. Hence, the lesion of our case was differentiated from the cysts of odontogenic origin.

Treatment planning depends on the histological type of UA. UA which is diagnosed as subgroups 1 and 1.2 may be treated conservatively (careful enucleation), whereas subgroups 1.2.3 mad 1.3 should be treated aggressively [16],[21]. The histological typing of the current case was 1.3 and hence, the lesion was treated aggressively with surgical resection. The recurrence rate for UAs after conservative surgical treatment (curettage or enucleation) are generally reported to be 10-20%, [16] and on average, less than 25% [22]. This is considerably less than 50-90% recurrence rates which are noted after the curettage of conventional solid or multicystic ameloblastomas [16].

Ameloblastoma is the most common odontogenic neoplasm. It presents with a numerous variety of clinical, radiographical and histopathological features. UA, a type of Ameloblatoma, too presents with a variety of clinical, radiological and histopathological features. Hence, it presents as a challenge both for it's diagnosis and treatment. There is always an on-going debate regarding the origin of UA. Immunohistochemical studies help us to know the nature of the lesion and also to differentiate the same from other cysts of odontogenic origin. Hence, it is essential that studies should be conducted on a large scale in order to know the origin and nature of the lesion.

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