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Enigmatic Ameloblastoma: A histologic perspective with various phenomenon in a single lesion: A rare case report

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Abstract
Ameloblastoma is an uncommon, benign, but locally aggressive odontogenic cancer that develops from the leftover Malassez epithelial residual, enamel organ, or dental lamina. With up to 10% of all cases, it is the most prevalent kind of odontogenic tumour of epithelial origin. Ameloblastoma is renowned for its capacity to cause widespread local destruction of bone and surrounding structures, even though it is considered benign. If treatment is not received, this might result in functional and cosmetic defects. After presenting to the department with the major complaint of swelling over the right posterior area of the jaw for the last week, the middle-aged patient was diagnosed with Follicular Ameloblastoma with Acanthomatous Change. This case is presented in the current case report. To determine the underlying pathogenetic process for such a rare incidence, the data must be published in the literature.

Keywords: Uncommon case report, odontogenic tumour, ameloblastoma

Introductions

Ameloblastoma is a rare, benign, but locally aggressive odontogenic cancer that arises from the remnants of the dental lamina, enamel organ, or Malassez epithelial residual [1]. It is the most common type of odontogenic tumour of epithelial origin, accounting for up to 10% of all cases [2]. Initially discovered by Broca in 1868, ameloblastoma primarily affects the mandibles. Compared to the maxilla, it is more prevalent in the mandible, particularly in the molar-ramus region [3]. The tumour is slow-growing and asymptomatic in its early stages, often presenting as a painless swelling [4]. Ameloblastoma is notorious for its ability to extensively destroy bone and surrounding structures locally, leading to both functional and Ameloblastoma is noted for its ability to locally destroy bone and surrounding tissue. Ameloblastoma is notorious for its ability to extensively destroy bone and surrounding structures locally, leading to both functional and Ameloblastoma is noted for its ability to locally destroy bone and surrounding tissues in huge numbers, even though it is considered benign. If treatment is received, this can lead to both functional and cosmetic defects [5]. Rarely, it may exhibit symptoms of malignant transformation or spread, generally to the lungs [6].

Based on their clinical, radiological, and histological characteristics, ameloblastomas are divided into a number of subgroups, such as [7, 8]:

1. The most prevalent and aggressive kind of ameloblastoma is conventional (solid/multicystic), which is prone to recurrence if left untreated.
2. **Unicystic ameloblastoma:** This condition manifests as a unilocular radiolucency and, with the right care, has a better prognosis.
3. **Peripheral ameloblastoma:** An uncommon kind that develops in soft tissues without affecting the bone.
4. **Malignant ameloblastoma and ameloblastic carcinoma:** Exceptionally uncommon forms that can spread and behave aggressively.

Ameloblastomas frequently show up as multilocular or "soap-bubble" radiolucencies on radiography, however unilocular patterns are also possible. The diagnosis is verified by histological analysis, which shows distinctive characteristics such a central region that resembles a loosely structured stellate reticulum and palisading basal cells that resemble

ameloblasts.

Clinicians must have a thorough understanding of ameloblastoma since early detection and the right treatment can reduce morbidity and enhance the quality of life for those who are affected. Therefore, this present case report presents one such case in which young patient reported to the department with the chief complaint of swelling on right side of lower jaw in the past 1 week, was diagnosed to be a case of Plexiform Ameloblastoma with Acanthomatous changes. In order to use the data to uncover the underlying pathogenetic process for such a rare incidence, it must be documented in the literature.

Patient Information

A young patient reported to the department with the chief complaint of swelling on right side of lower jaw in the past 1 week. Swelling was insidious in onset, associated with no history of pain or pus discharge in the same region. Patient also has no history of long-term systemic illness or sudden loss of weight, fever or night chills. Patient also has no case similar reported by anyone else in the family. Patient also has no history of tobacco or alcohol abuse. Patient appeared systemically healthy otherwise. On extraoral inspection, a bony hard swelling was observed in the mandible of size 3*3cm in size extending till angle of mandible. Palpation confirmed the inspectory findings. On intraoral examination, swelling was seen on either side of the alveolar cortical plates extending from 46 till 48 with missing teeth 46, 47, 48 in the same area. The swelling on palpation was bony hard, non-tender with no discharge upon pressing the area. The patient was then sent for Orthopantogram (OPG) (Fig.1.) and NCCT head and neck (Non-contrast Computed Tomography). (FIG.2.) OPG revealed multilocular radiolucent lesion extending from distal of 45 with impacted 46 and 48 extending till the lower border of mandible and posteriorly involving the ramus of mandible resulting in its complete erosion. NCCT revealed bilateral cortical expansion on right side of mandible with multiple buccal cortical perforation with displaced 46 and 48 extending till ramus of mandible. The patient underwent excisional biopsy and the lesion was sent for histopathological examination.

The excised tissue was processed and then stained with haematoxylin and eosin and then the slides were observed under microscope. These stained sections revealed small odontogenic islands arranged in the form of small and large follicles in mature connective tissue stroma. The follicles revealed tall columnar ameloblast-like cells with hyperchromatic round cell showing reverse polarity with subnuclear vacuolization and stellate-reticulum like cells in the centre showing squamous metaplasia with extensive cystic degeneration. (Fig.3.) Hyalinization is also observed around some follicles with compression of connective tissue around some follicles and extensive haemorrhage in these areas. A final diagnosis of Follicular Ameloblastoma with Acanthomatous changes was given. The patient was then kept for 3 months follow-up with complete resolution of the lesion and no recurrence noted till date. (Fig.2.)

Discussion

Adults in their fourth to fifth decades of life are typically affected by ameloblastoma, which grows slowly and shows no symptoms until it spreads to a larger lesion that puts pressure on nearby tissues [9]. Nevertheless, it is aggressive locally, has a high propensity to return, particularly if left untreated, and in rare cases, can spread. Therefore, the treatment should range from marsupialization to Enbloc resection followed by rebuilding of the resected area, depending on the extent and the histologic subtype identified [10].

The process of creating new blood vessels, or angiogenesis, is crucial to the formation of tumours because cancer cells divide more quickly and multiply faster than healthy cells, necessitating a larger blood supply to provide the proliferating cells with ongoing nutritional support [11]. The strenuous needs of proliferating cells lead to an imbalance in oxygen delivery and consumption, which causes hypoxia, a feature of human malignancies that results in treatment resistance. Hypoxia is regulated by hypoxia-inducible factor-1 (HIF-1), a basic helix-loop helix transcription factor composed of two subunits, HIF-1α and HIF-1β. The main gene linked to the onset of hypoxia is HIF-1β [12]. Hypoxia has also been linked to metastasis and the epithelial-mesenchymal transition in addition to treatment resistance [13]. Since HIF-1α expression has been observed to be directly correlated with prognosis rates and progression in follicular ameloblastoma, it has been investigated for prediction. There have been numerous discussions on the existence of Haemangiomatous Ameloblastoma, a distinct histologic subtype that was previously identified as the haemorrhage observed in the connective tissue and inside the follicles in this instance. According to Lucas, the vascular component of hemomatous ameloblastoma is only a secondary alteration. He claims that the stroma of these lesions experiences cystic degeneration, and that some blood vessels may expand and survive during this creation rather than shrinking, giving rise to the vascular component visible in the lesion [14].

According to Leider *et al*., cystic ameloblastoma might result from the cystic degeneration of a solid ameloblastoma; this could be connected to either the intrinsic production of proteinases (such as metalloproteinases or serine proteinases), which are enzymes that typically break down the central zone of the enamel organ after tooth development, or epithelial dys-adhesion brought on by defective desmosomes. Nevertheless, there is still a dearth of solid data supporting any of the suggested pathologies, despite the divergent views of numerous authors [15].

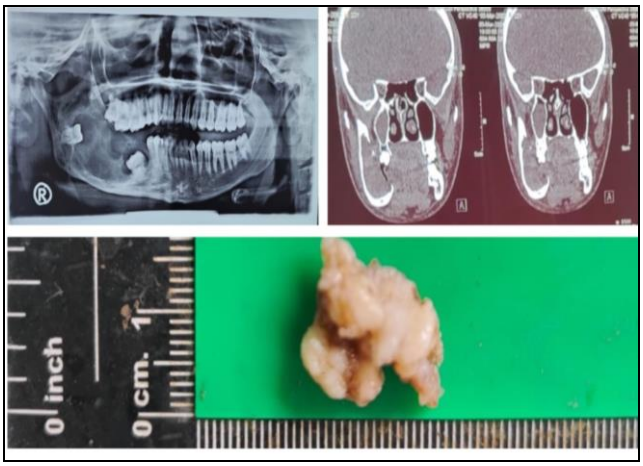

Fig 1: OPG, CBCT and Biopsy Specimen of the Patient

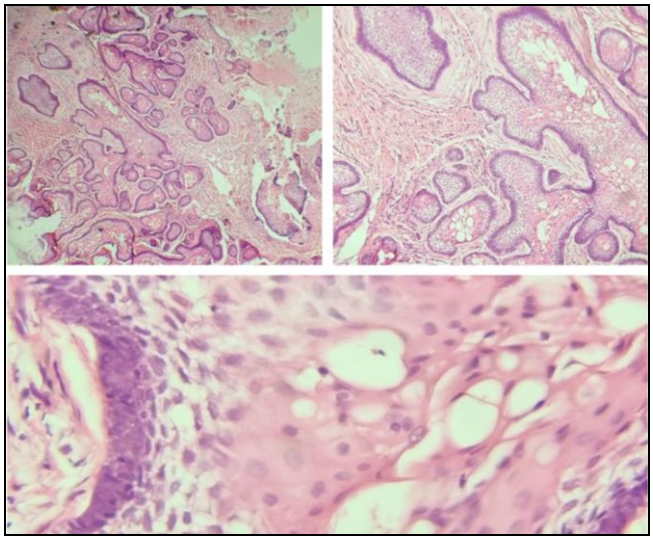

Fig 2: Histopathology of the Lesion at 4x, 10x, AND 40x

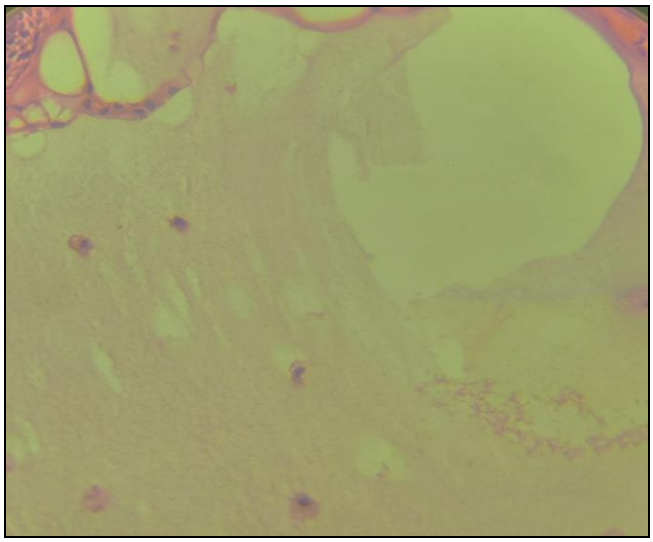

Fig 3: Complete Cystic Degeneration Seen within the Follicle

Conclusion

Despite being the most common tumour encountered and extensive literature existing on this category, still much work and extensive research is required to understand the pathogenesis underlying as to whether these histologic subtypes are separate lesions or they eventually convert hitherto. Also, the significance of multiple mechanisms in a single lesion must also be studied in order to know whether they are of any prognostic significance or are associated with a single genetic component lying underneath them that determines their behaviour and occurrence.

Legends

Declarations

Funding and/or Conflicts of interests/Competing interests: Not Applicable.

Data availability: All the data is available with the manuscript.

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