INTRODUCTION:
Ameloblastoma is a benign, locally aggressive neoplasm of odontogenic epithelial origin. It is the second most common odontogenic neoplasm next to odontoma. Its incidence rate is around 0.5 per million population per year, with highest reported cases in Asia and African continents. Ameloblastoma can occur as four variants - unicystic, solid/multicystic, desmoplastic and peripheral (extraosseous) variants, of which unicystic accounts for about 13% of all cases. Granular cell change in ameloblastoma though is a recognized phenomenon; its occurrence however is rare in unicystic entity. Here is a unique case of unicystic ameloblastoma exhibiting granular cell differentiation and diverse histopathological presentation, which has perforated the bone and presented as soft tissue growth (peripheral variant).

KEYWORDS:
Peripheral ameloblastoma, Granular variant, Follicular variant, Acanthomatous, Plexiform variant.

CASE REPORT:
A 27 year old male reported with a chief complaint of swelling in left lower back tooth region since 5 months. Patient was apparently asymptomatic 5 months back, later noticed a small growth in lower left molar region associated with mild pain. He gave a history of gradual increase of growth from the past 3 months.

On examination no significant facial asymmetry was noticed. Intra-orally, a diffuse growth in the alveolar mucosa distal to 37 was noticed, measuring 2 X 1 cm extending anterioposteriorly from distal surface of 37 to retromolar region and superioinferiorly from occlusal level of 37 extending into vestibule. Obliteration of buccal vestibule was noticed. Color of the growth was slightly erythematous, with indentations of left maxillary posterior teeth (Figure - 1). On palpation, the growth was tender, firm and non-pedunculated. Based on history and clinical findings, a provisional diagnosis of fibro-epithelial hyperplasia was considered and panoramic imaging was advised.

Orthopantomogram (OPG) revealed, a unilocular radiolucency with diffuse borders over left ramus of the mandible involving the crown and a portion of root of impacted 38 (Figure - 2). Considering these findings Computed Tomography (CT) was advised to know the extent of lesion. It showed bony destruction on the left posterior mandibular region extending from distal aspect of impacted 38, involving the ascending ramus of the mandible up to coronoid process (Figure - 3).
Figure – 3: 3D image shows evidence of bony destruction involving the ascending ramus & coronoid process of the mandible.

Based on the radiographic findings, a differential diagnosis of dentigerous cyst in relation to 38 was made. Prior to incisional biopsy, fine needle aspiration was performed and it was found to be negative. Incisional biopsy was planned under local anaesthesia at the lesional site and two specimens were collected, one from the alveolar mucosal growth and the other from bone for histopathological analysis.

On microscopic examination, the H&E stained section from the soft tissue specimen revealed a parakeratinized stratified squamous epithelium of even thickness overlying a fibrovascular connective tissue stroma showing odontogenic tumor epithelial cells arranged in the form of follicles which were lined peripherally by tall columnar ameloblast like cells with reversal of polarity, and superficial stellate reticulum like tissue. The stellate reticulum in few follicles exhibited cystic degeneration and acanthomatous changes in the form of squamous metaplasia. The intervening connective tissue stroma showed dilated vascular channels engorged with RBCs. A diagnosis of "peripheral ameloblastoma" was made (Figure - 4).

On microscopic examination, the stained section from the intra bony lesion showed a well defined cystic lumen lined by ameloblastomatus epithelial lining with basal tall columnar ameloblast like cells & superficial stellate reticulum like tissue. The cystic lining showed mural proliferations into the connective tissue capsule with subepithelial hyalinization (Figure - 5). The connective tissue capsule also showed numerous interconnecting, anastomosing cords & strands of odontogenic tumor epithelial cells with intervening connective tissue showing multiple dilated blood vessels typical of plexiform ameloblastoma (Figure - 6). Deeper connective tissue capsule revealed solid ameloblastomatus follicles, while few exhibiting acanthomatous change with squamous metaplasia and others show granular differentiation of the central stellate reticulum like tissue (Figures - 7, 8, 9). A diagnosis of "mural unicystic ameloblastoma - granular cell variant" was made.

On microscopic examination, the H&E stained sections from deeper connective tissue stroma exhibiting odontogenic tumour epithelial islands showing (a) cystic degeneration & (b) squamous metaplasia. (Peripheral Ameloblastoma).

DISCUSSION:
Ameloblastoma is a benign, slow-growing, locally invasive tumor of odontogenic origin involving the mandible (80 %) and maxilla, the
Surgical sites be examined, along with the radiographs thoroughly for Gardener points out that the characteristic slow growth of ameloblastomas is significant in that it may take years before recurrence becomes evident. Therefore, it is imperative that the surgical sites be examined, along with the radiographs thoroughly for at least 10 years and preferably longer. However, similar to the other types of solid or multicystic ameloblastomas, the prognosis is more dependent on the method of surgical treatment, i.e. granular cell ameloblastomas treated by enucleation or curettage exhibit a high recurrence rate, due to the fact that the border of the tumor within cancellous bone lies beyond the apparent macroscopic surface and the radiographic boundaries of the lesion. Therefore, radical surgical methods are recommended. Noteworthy is that, granular cell ameloblastoma has a tendency to metastasize. Patients should be kept under periodic observation because of reports of recurrences occurred even up to 8 years after initial treatment.

Conclusion:

Like most of the peripheral ameloblastomas which presented as soft tissue growths were falsely interpreted as extracapsular lesions, our reported case showed a similar presentation of a peripheral ameloblastoma, but which was a component of an intra-osses ameloblastoma which has perforated the alveolar bone giving rise to a soft tissue growth. Hence, thorough radiological and histopathological investigations are indicated before committing a diagnosis of peripheral ameloblastoma. From a surgical point of view, the present lesion should be treated as a solid ameloblastoma, though it is unicystic, because histologically it is differentiated from other aggressive ameloblastomatous lesions by a more aggressive granular cell differentiation. Therefore, an early diagnosis and prompt surgical treatment in granular cell ameloblastoma is of prime importance.

References: