Peripheral Ameloblastoma of Mandible: A Rare Case Report

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Abstract

Peripheral ameloblastoma (PA) is a rare, benign, extrasosseous neoplasm of the odontogenic epithelium. It is an exophytic growth which is localized to the soft tissues overlying the tooth-bearing areas of maxilla and mandible. In most cases, there is no radiological evidence of bone involvement, except for cupping or saucerization of the underlying bone. Histologically, the PA consists of proliferating odontogenic epithelium that exhibits the same histomorphological cell types and patterns as seen in the intraosseous ameloblastoma. The PA is a slow growing and non-invasive, and recurrence is uncommon following excision, in contrast to central ameloblastoma which is locally invasive and can destroy large segments of the jaw. We report here a case of the plexiform type of PA.

Keywords: Extraosseous, Exophytic, Peripheral, Plexiform pattern

INTRODUCTION

Peripheral ameloblastoma (PA) also known as extrasosseous ameloblastoma is a rare odontogenic soft tissue tumor which is reported to account for approximately 1-2% of all the ameloblastomas.¹ It has many histological characteristics same as that of solid/multicystic ameloblastoma (SMA), but it occurs in the soft tissues overlying the alveolar process of both the jaws without invading the underlying bone.²

It was reported for the first time by Kuru in 1911. However, what he described was not a PA, rather an intraosseous SMA that penetrated through the alveolar bone and fused with the oral epithelium eventually presenting as a peripheral lesion clinically. Gullifer and Chin reported many cases which were able to fulfill all the requirements of true PAs. Stanley and Krogh in the year 1959 were the first to completely report the case of a PA as documented by Philipsen and Reichart.³

PA is a slow, benign, asymptomatic lesion with the relatively smooth surface. The color of the lesion varies between normal or pink and red or dark red. There is no radiologic evidence of bone involvement except for superficial erosion or saucerization of the underlying bone. Histologically, the PA resembles the intraosseous form, consisting of a proliferation of ameloblastic epithelium set in a dense collagenous stroma.⁴

Most oral surgeons and pathologists consider it as a non-invasive lesion that cannot penetrate into the underlying bone. Few cases of PA with jawbone resorption have also been reported. Akioedo (1998) has reported a PA with unusual invasive features and invasion of maxillary posterior posterior region as documented by Tabatabaei and Akhavan.⁵

The diagnostic criteria of PA include origin from the overlying epithelium, presence of odontogenic epithelium islands in the lesion, and lack of a potential to bone infiltration. The recommended treatment is wide excision down through periosteum. Recurrence has been noted infrequently.⁶

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CASE REPORT

A 70-year-old male patient reported to the Department of Oral Medicine and Radiology, I.T.S Dental College, Greater Noida with a chief complaint of swelling in left lower front tooth region since 3 months. The patient was apparently asymptomatic 3 months back when he first noticed the swelling, which was intermittently increasing in size and has reached to the present size. The patient had no relevant past medical and dental history. On examination, a pedunculated swelling was present in left lower front tooth region extending from distal aspect of left lateral incisor to mesial aspect of left first premolar which was covered by normal mucosa approximately 2.5 cm × 2.5 cm in size, soft in consistency. All the involved teeth were tested vital. The radiographic examination did not demonstrate any bony destruction. An excisional biopsy was performed under local anesthesia, and the microscopic evaluation revealed proliferation of epithelium in the form of plexiform strands from the surface epithelium (Figures 1-3). Budding of surface epithelium was noted in few areas (Figure 4). The strands had ameloblast like cells at the periphery with a polarized nucleus and stellate reticulum like cells in the center of these ameloblast like cells (Figures 5 and 6). Mature fibrous connective tissue stroma with few blood vessels was also noted. A diagnosis of plexiform PA was made.

DISCUSSION

The PA - also known as the extraosseous ameloblastoma has several of the same histologic characteristics of an intraosseous, infiltrating ameloblastoma. It is a painless, sessile, firm, and exophytic growth, the surface of which is usually smooth and in some cases has been described as “granular” or “pebbly.” In other cases, the surface exhibits a “papillary” or “warty” appearance. The color of the lesion varies between normal or pink and red or dark red.3 The average size of the lesion ranges from 1 to 2 cm in diameter.1 Our case presents clinically as a pedunculated swelling which was covered by normal mucosa, approximately 2.5 cm × 2.5 cm in size, soft in consistency.
Potential sources of PA include odontogenic remnants of the vestibular lamina, pluripotent cells in the basal cell layer of the mucosal epithelium and pluripotent cells from the minor salivary gland.\(^7\) The most common site is soft tissue overlying tooth-bearing areas of maxilla and mandible. Mandible is most commonly involved than the maxilla. Premolar region is the most commonly involved site in mandible followed by the anterior mandibular region. In maxilla most commonly involved site was the soft palatal tissue of maxillary tuberosity.\(^8\) Our case also occurred in the most common site, i.e., the mandible. Five extra gingival lesions have been reported under the term PA.\(^3\)

The age range of patients with PAs range between 16 and 92 years, with an overall mean age of 50.2 years.\(^1\) It has a slight male predilection with a male to female ratio of 1.9:1.\(^8\) The age of the patient in the present case also coincides with that reported in the literature, i.e., 70 years.

There is no radiologic evidence of bone involvement. Radiographically erosion of the bone or a superficial bony depression, cupping or saucerization may be noticed caused by pressure resorption rather than resorption caused by the neoplastic invasion.\(^3,9\) Our case goes in accordance to the cases reported where there was no bony erosion or involvement.

On histopathological examination, the tumor consists of proliferating odontogenic epithelium that exhibits the same histomorphologic cell types and patterns as seen in the SMA. The stroma is that of mature, fibrous connective tissue. Most of the epithelial islands exhibit palisading of columnar basal cells, but a stellate reticulum is seldom conspicuous. Some of the squamous cells in the acanthomatous nests may show “ghosting” features. Presence of clear cells has also been seen.\(^8,11\)

These clear cells are cytomorphologically and histochemically identical to those reported to occur in the dental lamina and in several other lesions of odontogenic origin such as the lateral periodontal cyst, the gingival cyst of adults, the calcifying ghost cell odontogenic tumor, the calcifying epithelial odontogenic tumor, and the clear cell odontogenic carcinoma.\(^3\) Histopathological evaluation of our case confirmed the plexiform type of PA.

PA should be differentiated from peripheral reactive lesions such as peripheral odontogenic fibroma, peripheral giant cell granuloma, pyogenic granuloma, fibrous epulis, papilloma, fibroma, odontogenic gingival epithelial hamartoma, and basal cell carcinoma.\(^8\)

As noted by Gardner,\(^12\) the term PA is potentially dangerous in that this diagnosis may lead to unnecessarily aggressive treatment.\(^12\) The current treatment of choice is conservative supraperiosteal surgical excision with adequate disease-free margins.\(^8\)

**CONCLUSION**

PA is an uncommon odontogenic neoplasm. Recurrences are rare, even in conservative surgeries with the resection of a small amount of normal tissue. Despite the non-aggressive course and low recurrence rate of the PA, long-term follow-up is absolutely necessary.

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