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CASE STUDY

PERIPHERAL AMELOBLASTOMA WITH INTERDENTAL BONE LOSS: A RARE CASE REPORT RUNNING TITLE : PERIPHERAL AMELOBLASTOMA A RARE CASE REPORT

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ABSTRACT

The peripheral ameloblastoma (PA) is a rare, benign, extraosseous odontogenic soft tissue tumour that is confined to the gingiva or alveolar mucosa. The PA presents the same histological characteristics of intraosseous ameloblastoma, although it is less aggressive than this classical subtype. A 47-year-old non-smoker male patient reported to Department of Periodontics, SCB Dental College and Hospital, Cuttack with an asymptomatic lesion on the buccal gingiva of the canine-premolar-molar right mandibular region growing slowly for the last 1 year. Thorough clinical examination was carried out. Incisional biopsy, histopathological and radiographic investigation with CBCT was done for the lesion followed by excision of the lesion. In this report we present a clinical case of PA of the in the right mandibular gingiva, highlighting the importance of histological examination to the diagnosis.

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INTRODUCTION

Ameloblastoma is an odontogenic tumour of benign origin usually located in the jaw bone (Becelli *et al*., 2002). The sources of the tumour is thought to be from residual epithelium from tooth germ, epithelium of odontogenic cysts, stratified squamous epithelium, and epithelium of the enamel organ (Hollows *et al*., 2000). It represents approximately 1% of oral tumours; 80% of ameloblastomas occur in the mandible and the remaining 20% in the upper jaw (Becelli *et al*., 2002). The tumour is well defined radiographically with delineated cortical border. The internal structure of peripheral ameloblastoma can be completely radiolucent or have bony septae that provide multilocular or soap bubble patterns (Lagares *et al*., 2005). In contrast to intraosseous ameloblastoma, peripheral ameloblastoma (PA) has an extraosseous location. PA is a rare odontogenic tumour, representing about 1.3 to 10% of all ameloblastomas (Philipsen *et al*., 2001). The maximum incidence is observed between the fifth and sixth decades of life, with an average patient age at initial appearance of the lesion of 50 years. A slight male predominance has been reported. In most cases (65%), the lesion is located in the mandible, particularly on the lingual aspect of the premolar zone (representing 32.6% of all locations) (Philipsen *et al*., 2001).

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The PA are erythematous, ulcerative lesions, and papillary lesions, of the attached gingiva, exhibiting nodular masses with a smooth, granular, pebbly, warty or papillary surface (Gardner *et al*., 2005; Nonaka *et al*., 2013). PA are usually confined to the gingiva or the alveolar mucosa, however it may cause depression or pressure resorption of the underlying bone, a phenomenon known as "cupping" effect (Philipsen *et al*., 2001; Mintz *et al*., 1990). The purpose of this article is to report a new case of this rare anomaly occurring on the mandibular gingiva, associated with interdental bone loss.

Case History

A 47-year-old non-smoker male patient reported to Department of Periodontics Govt and Oral Implantology, SCB Govt Dental College and Hospital, Cuttack with an asymptomatic lesion on the buccal gingiva of the canine-premolar-molar right mandibular region (Fig 1). The lesion had been slowly growing for about 1 year with no change in its dimension for the past 8 months. The lesion was well demarcated measuring 1 x 1 x 0.5 cm, sessile and firm in consistency with surface ulceration on the margins of the lesion. Intraoral periapical radiographs showed interdental bone loss w.r.t 27, 28 region (Fig 2). CBCT report was in accordance with the IOPA with the 3D representation showing depression or loss of buccal cortical plate in the same region (Fig 3). Bone sounding revealed characteristic egg shell crackling of the lesion. An incisional biopsy was performed under local anaesthesia (Fig 4).

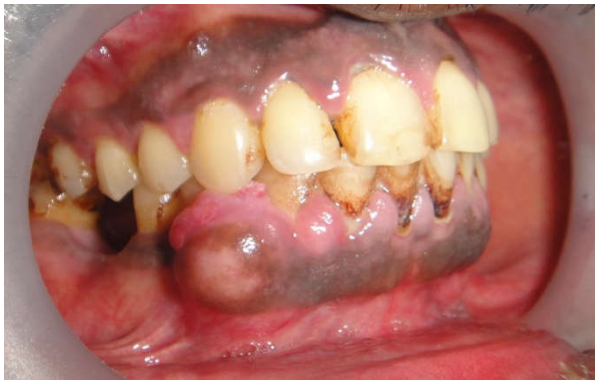

Figure 1. Initial presentation of sessile asymptomatic lesion on buccal keratinized gingiva of 27,28 region

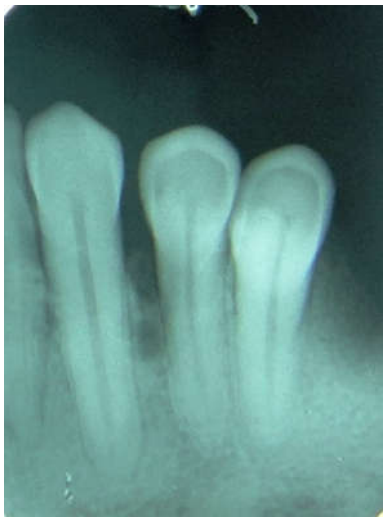

Figure 2. Periapical radiograph of 27,28 region showing interdental bone loss

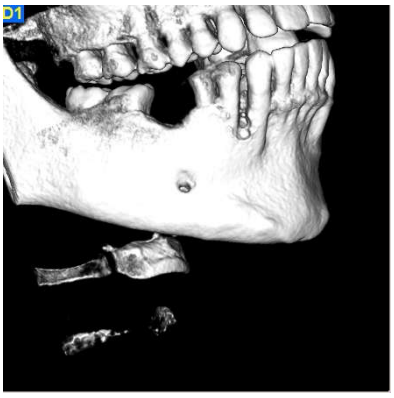

Figure 3. 3D CBCT representation of the lesion

The gross specimen revealed multiple bits of gyayish white tissue < 1cm. The microscopic examination showed the presence of an ameloblastoma composed of acanthotic stratified squamous epithelium over a fibrocollagenous stroma (Fig 5.). Many follicles were lined by peripheral radially arranged columnar cells and central stellate reticulum like cells with squamous metaplasia and cystic changes in the fibrous stroma (Fig 6). Overlying squamous epithelium is also seen in the micro section. The diagnosis of follicular peripheral ameloblastoma was confirmed. Excision of the lesion with osteoplasty in 27,28 region was performed after a period of 45 days from incision biopsy (Fig 7,8). No recurrences were observed in a 6 months follow-up period.

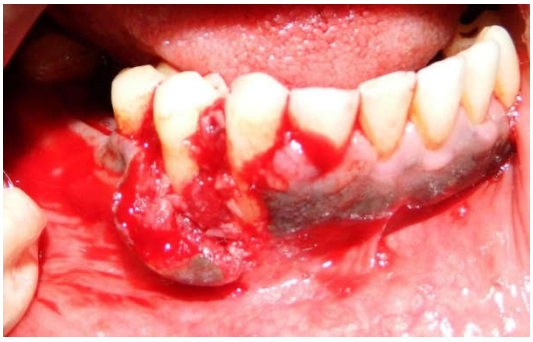

Figure 4. Incisional biopsy of the lesion

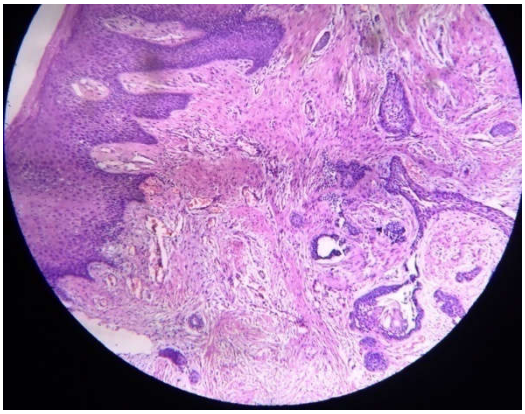

Figure 5. Microsection showing small islands of ameloblastoma in a mature fibrous stroma present below the oral epithelium (hematoxylin and eosin; x 4X)

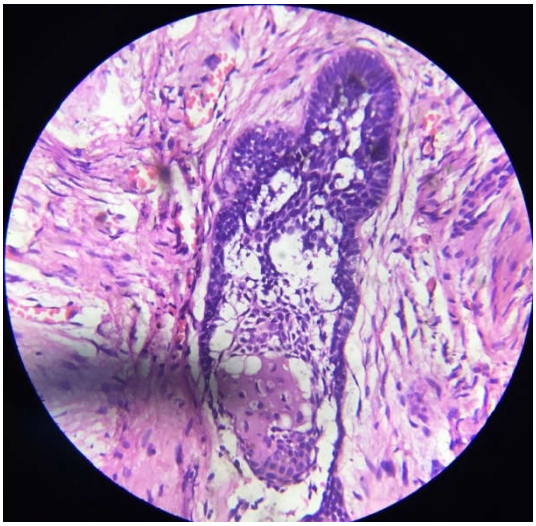

Figure 6. Microsection showingsolitary ameloblastoma follicle with peripheral tall columnar cells showing reverse polarity & centrally located stellate reticulum like cell (hematoxylin and eosin; x40X)

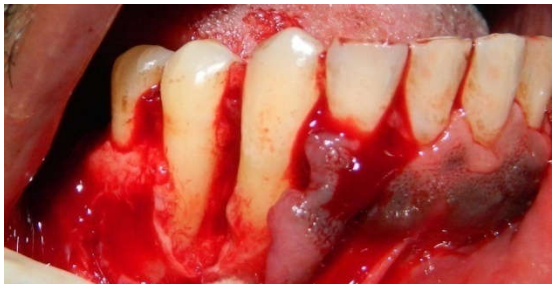

Figure 7. Excision of the lesion with osteoplasty was performed

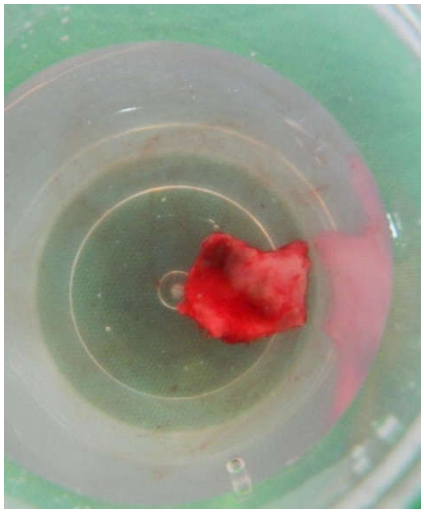

Figure 8. Excised lesion

DISCUSSION

The occurrence of ondontogenic tumours is one the rare features of the jaws. They comprise a group of uncommon lesions that share the common aspect of arising from the odontogenic tissues, either the epithelial component, the ectomesenchymal component, or both. Their clinical behaviour varies from harmless non-neoplastic hamartomas to highly aggressive tumours, such as central ameloblastoma. Peripheral ameloblastoma (PA) is a rare variant of benign odontogenic tumour that exhibits the histologic characteristics of its intraosseous counterpart but occurs in the soft tissues without bone involvement, except for erosion or depression of the bone crest (Philipsen *et al*., 2001; Nonaka *et al*., 2013). Epidemiological data shows the maximum incidence of PA in the sixth decade of life, 1.9:1 male/female ratio, an average age of presentation of 52.9 years in males and 50.6 years in females.[8] PA seems to be more male-predominant and to occur at a higher age than its intraosseous counterpart (Reichart**,** 2008; Pogrel, 2009). The most frequent onset site is the mandibular premolar region, followed by the anterior mandibular region and by the tuber maxillae. Approximately 7 cases of PA out of 10 occur in the lower jaw.[7] Although the occurrence of PA is a rarity comprising about 1% of ameloblastoma, they are the most common peripheral odontogenic tumours. One of the main problems regarding peripheral ameloblastoma is its possible origin. The two main theories are: 1) origin from the extraosseous epithelial remnants of the dental lamina and its organ derivatives within the underlying connective tissue and 2) origin from the basal cell layer of the oral mucosa, which is believed to have odontogenic potential (Mintz, 1990). The lack of bony infiltration of PA may be explained through the existence of a fibrous barrier surrounding the lesion generated by the gingiva and the periosteum. This aspect of the biological behaviour of PA makes this pathology greatly different from intraosseous ameloblastoma and has a great implications in deciding the treatment plan.

However in some cases a local depression or cortical bone erosion, described as cupping or saucerization, may occasionally be described (Philipsen *et al*., 2001; Nonaka *et al*., 2013; Buchner *et al*., 2006). In the present case the erosion of interdental alveolar bone is seen between 26 and 27 which may attributed to the pronounced cupping or saucerization and partly due to the inflammatory plaque component. The treatment of choice for PA is the surgical excision with proper disease-free margins. No extensive radical treatment is usually required (Pogrel, 2009). The differential diagnosis must be made with fibrous nodules, gingival tumours, peripheral odontogenic fibromas, peripheral ossifying fibromas, pyogenic granulomas, peripheral giant cell granuloma and also with very rare odontogenic gingival hamartoma and peripheral squamous odontogenic tumour. The current case had clinical appearance of reactive hyperplastic lesions, however the microscopic examination revealed the diagnosis of PA. Therefore, it is important to include peripheral ameloblastoma as a differential diagnosis of nodular lesions of gingiva and alveolar mucosa. Proper diagnosis and treatment of PA is detrimental for patient well-being.

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