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Management of Odontogenic Keratocyst Crossing Mandibular Midline by Conservative Method

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Abstract

Parakeratinized odontogenic keratocyst are antagonistic variant of odontogenic keratocyst with increased recurrence rate, they are benign and habitually destructive and tend to recur after conservative surgical treatment. They should therefore be differentiated from other cysts of the jaw. Odontogenic Keratocysts possess outpouchings and microscopic daughter cysts from which recurde sence may arise, it warrants exceptional attention because of its remarkable histopathologic features and biologic behavior, 80 to 90% of cases exhibit parakeratinization in the cystic epithelium, multiple okc s are noted in nevoid basal cell carcinoma syndrome, an autosomal dominant multisystematic disease, this work aimed to present a case of very extensive odontogenic keratocyst in a 45yrs old male.

Keywords: Parakeratinized OKC, Enucleation, keratin cells, Daughter cysts, Recurrence.

Introduction

The odontogenic keratocyst was first described in 1876, named by Phillipsen in 1956 and has been integrated with a tendency to recur[1] In 2005 WHO classification, Odontogenic keratocyst was comprised under tumors as a Keratocystic odontogenic tumor[2]. In the latest 2017 WHO classification, again they encompassed it under cysts as OKC as the current evidence is lacking to justify the continuation of keratocystic odontogenic tumor as tumor[3]. A male prevalence has been investigated in KCOT.[4] KCOT has been noted in a wide age range, with a peak incidence found in the second and third decades. The mandible is more frequently involved than the maxilla: About 65-83% of KCOTs transpire in the mandible. Both in maxilla and mandible, it has a tendency to occur in the posterior part of the jaw. [4],

Materials And Methods

Case Report

A male patient reported to our unit with chief complaint of swelling in his left lower one third of his face since 3months,which was originally small in size and gradually progressed to a present size, no associated pain ,no history of trauma ,no systemic illness, On extra oral examination the swelling is non tender,hard in consistency, left submandibular lymph nodes palpable which is non tender and mobile, On intra oral examination the swelling is palpable on left buccal vestibule, non tender,hard in consistency approximately measuring of about 2x3cm in size extending from 44 to 48 region, Buccal and lingual cortical expansion noted with respective to the swelling. The teeth in the mandibular arch were non vital, Panoramic radiograph (OPG) revealed a well-defined multilocular radiolucency involving the left half of the mandible upto 48 region which is horizontally impacted , Within the radiolucency, manifestation of internal septa and impacted 38 were present.

Treatment Done

Aspiration biopsy of the lesion obtained white cheesy material which on cytological examination revealed keratin .Incisional biopsy was carried out and histopathological report suggested of odontogenic keratocyst, root canal treatment was carried out from 42 upto 37 and patient was taken under general anesthesia for the Enucleation, mechanical and chemical curettage. crevicular incision given from 45 region upto 38 with distal release, full thickness mucoperiosteal flap reflection carried out, horizontally impacted 38 was sectioned and removed using elevator ,white cheesy material was removed and cystic lining as well, apicectomy was done from 42 to 37,modified carnoy s solution(composed of 6ml of absolute alcohol,1ml of glacial acetic acid,1gm of ferric chloride) was placed in the cystic cavity for 3 minutes using ribbon guaze and retrograde filling using glass ionomer cement was carried out, excised specimen was submitted for histopathological examination which revealed fragments of fibrous cyst wall lined by 6-7 layers of uniform stratified squamous epithelium, stroma infiltrated by chronic inflammatory cells suggestive of okc, Once the histopathologic examination revealed the lesion to be an odontogenic keratocyst, the patient was consulted for the evaluating for the Nevoid Basal Cell Carcinoma syndrome. Investigation done for PTCH1 gene mutations and the patient was not found to have a genetic mutation.

Discussion

OKC are comparatively familiar developmental odontogenic cysts and incidence reported from 10–12% of all jaw cysts. Toller (1967) suggested that the OKC to be regarded as a benign cystic neoplasm, compared than an odontogenic cyst.[6] In 1992, OKC was the preferred terminology for cysts with keratinized lining reported.[7] In 2005, WHO reclassified intraosseous parakeratinized variant as a tumor.[4] The OKCs transpire either from

epithelial remnants of tooth germ in the mandible and maxilla or the basal cell layer of the overlying surface epithelium [8] The accurate diagnosis and treatment of odontogenic keratocysts is crucial for three reasons: a) Keratocysts are considered more aggressive than other odontogenic cysts [9], b) Its recurrence rate is higher than odontogenic cysts.[9] c) The probability of associated with neoplastic basal cell carcinoma syndrome makes the patient with multiple cysts a candidate for the syndrome[10,] On aspiration OKCs restrain a dirty white, viscoid material of keratin with a stint of pus, but without an odour ,The treatment of the OKC remains debatable, In general ,treatment modalities are classified as conservative and aggressive. Conservative treatment generally incorporates simple enucleation, with or without curettage, using spoon curettes of marsupialization. Aggressive treatment generally comprises peripheral osteotomy, chemical curettage with carnoy's solution and resection, Recurrence occurs due to the following reasons. The first reason is because of the incomplete removal of the cyst's lining (thin, friable epithelial lining). Secondly, it includes growth of a new OKC from small satellite cysts of odontogenic epithelial rests left behind, The third reason involves the development of an unrelated OKC in an adjacent region of the jaws, which is interpreted as a recurrence. Marx and stern trusted that incomplete cyst removal and new primary cyst formation are the most common reasons for recurrence.[11] Pindborg and Hansen (1963) proposed the histological criteria, which were confirmed by Browne in 1970 and 1971 [5] ·Histopathologically an OKC has a fibrous wall lined by epithelium with a thin layer of stratified squamous epithelium ,This epithelium has a basal cell layer of six to eight cells thick and a lining of flattened keratotic epithelial cells. The formed keratin lines the luminal surface of the epithelial cells in a slightly wavy of corrugated pattern [12] OKCs have been classified into parakeratotic and orthokeratotic subtypes , These types depends on histological characteristics of the lining and the type of keratin produced. Compared with the parakeratotic subtype, the orthokeratotic subtype give rise to keratin closely resembling the normal keratin produced by the skin. The keratin does not contain nuclei. The parakeratotic subtype has a more disordered production of keratin. The keratin contains nuclei and is referred to a para keratin. The parakeratotic type is the most frequent (80%) and has a aggressive nature of clinical presentation than the orthokeratinized variant [13]. Immunohistochemical studies have suggested higher levels of interleukin-1α in OKC compared with dentigerous cyst , Interleukin-1α plays a crucial role in expansion of OKCs by increasing the secretion of keratocyte growth factors from interactive fibroblasts.[14]

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Legends Figure

Fig 1: Preoperative Orthopantomogram

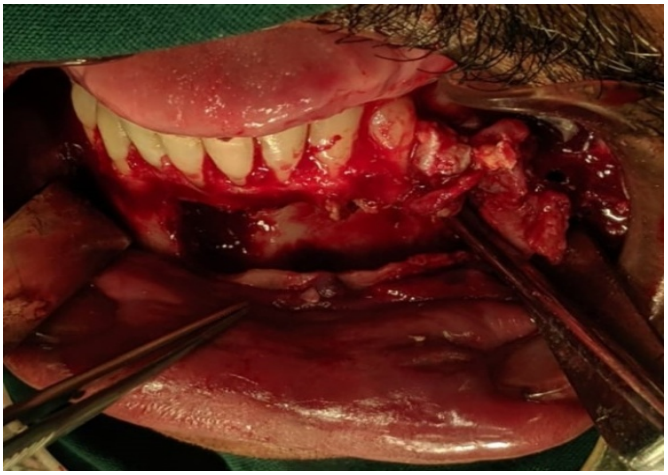

Fig 2: Cyst Enucleation

Fig 3: Sectioned Impacted Tooth

Fig 4: Excised Specimen

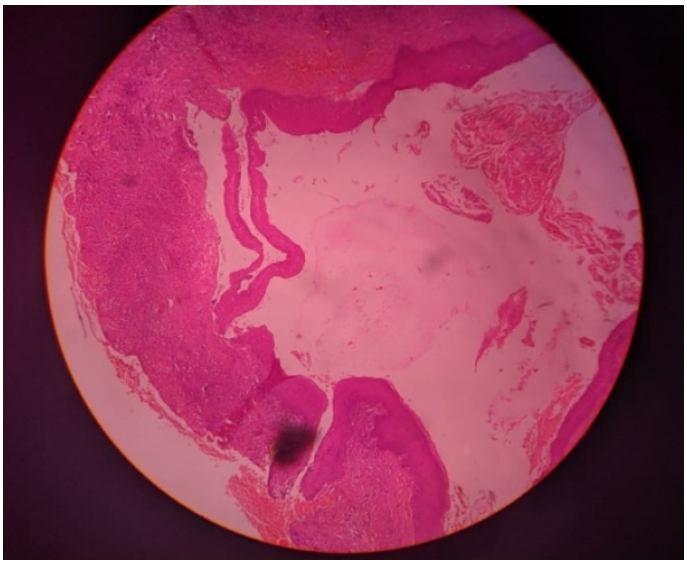

Fig 5: Histopathological Picture

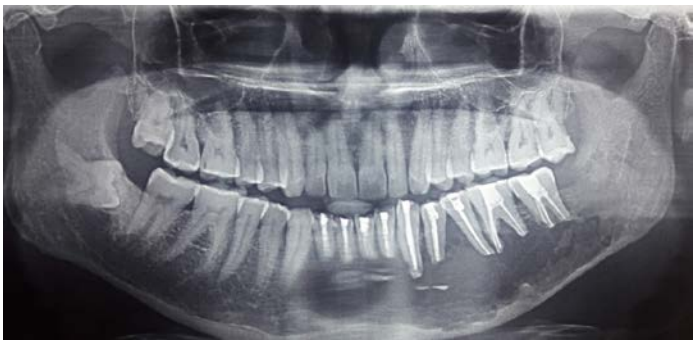

Fig 6: Postoperative Orthopantomogram