ISSN - 2250-1991 | IF : 5.761 | IC Value : 79.96

Original Research Paper Dental Science

KERATOCYSTIC ODONTOGENIC TUMOUR- A CASE REPORT

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ABSTRACT

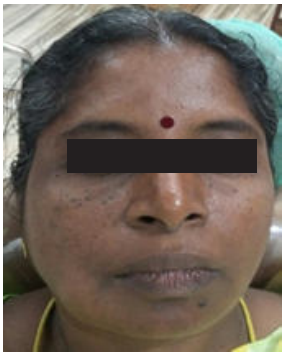
The purpose of this paper is to focus the hallmarks of the keratocystic odontogenic tumour (KCOT) which was formerly known as odontogenic keratocyst (OKC). In 1950 OKC was found first. KCOT characterized by aggressive in nature and high recurrence rate. Odotogenic keratocyst also accompanying the nevoid basal cell carcinoma syndrome. After radicular and follicular cysts, odontogenic keratocysts are the third most common cyst of the jaws.

KEYWORDS Keratocystic odontogenic tumour (KCOT), benign tumor, recurrence, treatment.

INTRODUCTION:

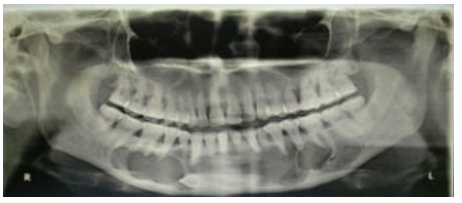
In 1876 OKC was first discovered[1]. In 1950 the word OKC was identified. Es.KCOT has peculiar clinical behaviour and histopathological features.[2] In 1956 again classified by Phillipsen. In 1962, further classified by Pindborg and Hansen. They also described the important features of cyst.[1]Odontogenic keratocyst producing so much of keratin so it is designate as keratocyst.Based on its clinical behaviour Toller distinguish as a benign neoplasm in 1967.[3] In 2005, the WHO Working Group recommended the term keratocystic odontogenic tumor (KCOT) and isolate the lesion from the orthokeratinizing variant .[4] OKC was clinically behave aggressive in nature and has high recurrence rate, and an association with nevoid basal cell carcinoma syndrome (NBSCC).[5]

CASE REPORT :



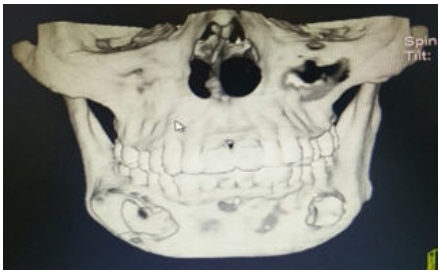
A 40 year old female patient reported to the department of oral surgery complaining of swelling in the lower anterior region for past 2 and half years. Patient's medical history was unremarkable. On extraoral examination mild swelling was present in the lower anterior region. Patient had numbness in the left lower lip for the past 20 days. Intraoral examination showed soft swelling present over the body of the mandible from 36 to 46 region with sharp bony margins. On palpation mild pain was present. On percussion there was no pain and no mobility was observed.

Orthopantamogram reveals,



Well defined radiolucency is seen with sclerotic border extending from 36 to 46. On right side end the lesion is multilocular on the left end the lesion appears unilocular. Measuring around 8x2 cm.

CT 3 D IMAGE:



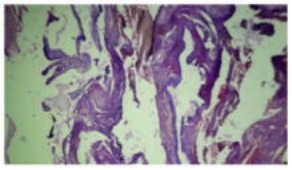
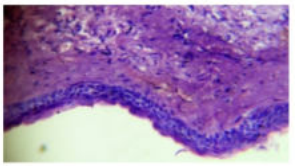
In this image OKC cyst involving bone is seen.

According to these clinical and radiological features, different diagnosis

- Dentigerous cyst
- Ameloblastoma,

Incisional biopsy of histopatholgy reveals that the given histopathologic section of soft tissue specimen shows cystic lumen, cystic epithelium and underlying connective tissue capsule. The epithelium is uniform in thickness showing 6-10 layers with basal cell palisading in some areas. Connective tissue shows

inflammatory cells, blood vessels and odontogenic epithelial islands.Diagnosis given as parakeratinized keratocystic odontogenic tumour.

DISCUSSION:

The odontogenic keratocyst is a histopathologically and behaviourally unique lesion. It is the most aggressive and recurrent of all the odontogenic cysts and shows characteristics as that of both a cyst and a benign tumor.[4] The OKC involves approximately 11% of cysts of the jaws & is most commonly situated in the mandibular ramus & angle.[6] As such, there were many implications that OKC be considered as a benign cystic neoplasm rather than cyst.[2] Shear published his enormous work on the aggressive nature of the odontogenic keratocyst and finally concluded it is a benign cystic neoplasm. Shear aggressively used the term "keratocystoma". Pathogenesis of KCOT was described by Regezi and other authors that the mechanisms that it has high proliferation rate, growth and expansion. Proliferation is due to expression of antiapoptotic proteins (bcl-2) and expression of matrix metalloproteinase (MMPs 2 and 9). PTCH gene mutation also play role in pathogenesis of this cyst.[1]

Recurrences:

The onset of recurrence of OKC has varied from 2.5% to 62%. The main consideration in the variation of the degree in these reports are mainly because some studies reported that Nevoid Basal cell carcinoma syndrome (NBCCS), duration of the follow-up period and based on treatment methods .[1]

Multiple OKCs often occur as a component of Noonan syndrome, NBCCS, orofacial digital syndrome, Ehler-Danlos syndrome, Simpson-Golabi-Behmel syndrome or other syndromes. NBCCS can also have accompanying skeletal features, such as frontal and parietal bossing and mandibular prognathism, and cutaneous abnormalities.[7]

If KCOT treated aggressively then the recurrence rate is comparitively low while more conservative methods prone to more recurrences. Histopathologic diagnosis of OKC, a defined follow-up period and method of treatment.[8]

Because of high recurrence rate rarely KCOT transform into squamous cell carcinoma. Still the etiology is questioned as developmental anomaly as contradictory to a neoplasm.[9]

Surgical treatment methods divided into conservative and aggressive by Morgan and colleagues. Conservative treatment is "cyst-oriented", where enucleation, with or without curettage, or marsupialization is done to preserve the anatomical structures because KCOTs commonly occurs in younger patients. It has been destined that a conservative approach is applicable not only for all age groups, but even in patients with NBCCS. Aggressive treatment applicable to "neoplastic nature" of the KCOT and includes peripheral ostectomy, chemical curettage with Carnoy's solution or en bloc resection. Aggressive modalities have been usually recommended for large KCOT, NBCCS and recurrent cases.[10]

Inflammed KCOT cases demonstrate the fibrous capsule in the wall of the connective tissue thickens. Ulceration of the epithelium also occur, which forms well-developed ridges, but keratinization freqently disappear. The fibrous capsule also shows dystrophic calcifications which is of unknown origin. The reclassification of the WHO in 2005 put us into doubt on the cystic nature of the parakeratinized type that was renamed keratocystic odontogenic tumour because many authors found that this form had higher mitotic activity and suppresses the action tumour suppressor genes.[11]

In cases with very large cysts the entire procedure can be divided and done. The decompression procedure can be performed under two steps. In the first step the cyst is opened and a drain is placed. After the cyst has shrunk enucleation can be done. After enucleation patient needs to use the drainage tube to irrigate betadine solution.[12]

OKC proliferative activity investigated by various authors and compared with other odontogenic cysts and tumours. Molecular studies revealed the neoplastic concept of OKC and loss of heterozygocity.[13]

Regardless of the source of epithelial cells, the etiology of KCOTs is correlated to genetic factors, in particular to mutation of tumor-suppressor PTCH gene, which is an essential part of sonic hedgehog (SHH) signaling pathway. Besides genetic factors, numerous studies described that dysregulation of cell cycle and proliferation would be an important for KCOT pathogenesis. It is conceived that KCOTs show increased cell proliferation rates and that such a phenomenon may be related to its aggressive growth. PCNA (Proliferating Cell Nuclear Antigen) is a protein which is demonstrated in the nucleus of replicating cells. It is considered to be a marker of cell replication. Ki-67 is an another marker of cell replication, was significantly more demontrated in KCOTs compared to other types of odontogenic cysts.[14]

CONCLUSION:

Due to their unique clinical and biological features, KCOTs still represent an important problem in oral and maxillofacial surgery and remain to be a subject of controversy among researchers and clinicians. The literature is limited to retrospective study designs and prospective studies need to be initiated. Due to their unique clinical and biological features, KCOTs still represent an important problem in oral and maxillofacial surgery and remain to be a subject of controversy among researchers and clinicians.

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