Epithelial Dysplasia in KCOT: Ignored factor in the reasons of recurrence

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Odontogenic keratocyst (OKC) is an entity with special interest since years or more correctly decades; this is because of its notorious behavior in terms of clinical manifestation, aggressiveness & also its great potential for recurrence. Thus, it is redefined aptly in 2005 as Keratocystic odontogenic tumor (KCOT).[1]

A biopsy specimen of a case of 2 times recurrent KCOT came to the department of Oral & Maxillofacial Pathology department of the dental college, of a 32 year old female patient. Complete enucleation of the cyst was done along with segmental mandibulectomy was done as a treatment. After 6 months, the patient again came with swelling in the same left mandibular body region extending from first premolar to first molar area. On histopathology, the heamatoxylin & eosin stained section showed cystic lining lined in the part by a regular layer of parakeratinized stratified squamous epithelium supported by a thin fibrous capsule consistent with the appearance of a KCOT. Maximum areas along the lining showed increasing dysplastic change with abundance of hyperchromatic cells & increased basilar cell hyperplasia & Koilocytes [Fig1,2]. A tiny satellite cyst was also evident in the section. Patient's previous histopathology report also reported OKC with dysplasia.

Now, as a known fact, there are numerous causes of recurrence of KCOT. Some of the important ones are presence of satellite or daughter cysts, incomplete removal of cyst, fragile & thin lining of KCOT, development of new KCOTs from epithelial offshoots of basal cell layer of oral epithelium & syndrome associated cyst. Also, it is an established fact over years that KCOT show epithelial dysplasia & also they have potential for malignant transformation.[2,3,4] Evidence of epithelial dysplasia in KCOTs was documented back in 1987 when High et al.[2] studied the DNA content of KCOTs which underwent malignant transformation. They found that KCOT with epithelial dysplasia had a large additional peak to the right of the diploid G0/G1 peak & represented a DNA aneuploid G0/G1 component which has DNA index 2.0. This was similar to carcinoma arising from the cyst. Another marker which authenticates the epithelial dysplasia is the increased expression of p53 protien. The first study which found out increased p53 levels in some OKC's was that of Ogden et al. in 1992.[3] More recently, study done by Cox & Pittel et al.[4,5] showed that cysts showing dysplasia had increased p53 activity and had high degree of recurrence. As proven from the documented literature, odontogenic keratocyst showing features of epithelial dysplasia are having high chances of recurrence than those cysts having no features of epithelial dysplasia.[3,4] As also, greater risk of malignant transformation in the lining of cysts with dysplasia makes this feature worth consideration.[6] Hence, epithelial dysplasia should be considered as one of the causative factor alongwith the proven factors for recurrence of the cyst.

The present case sheds light on the recurrence potential of the cyst with dysplasia and also the aggressive therapy & long term follow up that should be employed to treat KCOTs with dysplasia. After enucleation of the KCOT with dysplasia safe margins should be employed. More studies are warranted in this direction as there is increasing documentation of KCOT having features of epithelial dysplasia.

Should KCOT with dysplasia be treated differently than the routine cases to avoid recurrence & what percentage of these undergoes malignant transformation is the question to be addressed.

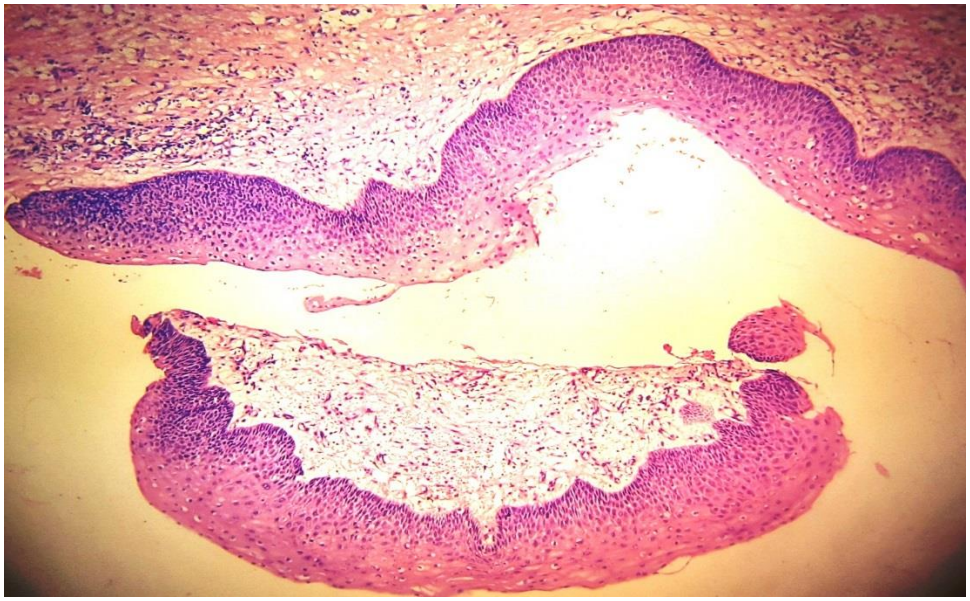

Fig. 1: legend- 10 X view showing cystic lining of OKC showing epithelial dysplasia

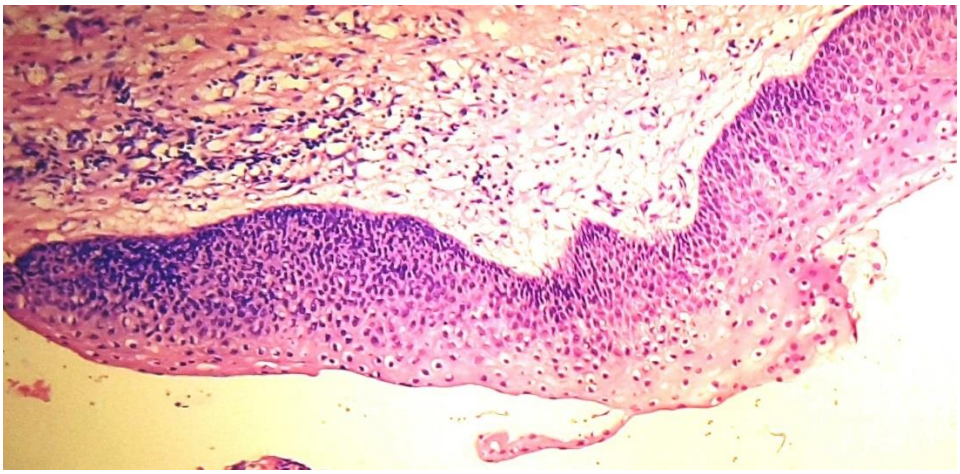

Fig. 2: legends- 40 X magnification showing dysplastic epithelial lining of OKC showing basilar cell hyperplasia, hyperchromatic cells, mitotic figures & koilocytes.

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