CASE REPORT

Odontogenic Keratocyst - A case Presentation

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ABSTRACT:

Keratocystic odontogenic tumor are considered to be benign, even though they are locally aggressive and show high recurrence rates after conservative management. The distinction between Keratocystic odontogenic tumor and other odontogenic cysts is necessary not only for the treatment purpose but also for its prognosis. Histopathological examination is the gold standard for the diagnosis. Also due to the advent of immunohistochemistry, it is shown that the proliferation index of odontogenic keratocyst is high. The treatment of these cysts need to be done with caution.

Key words: Odontogenic Keratocyst, Ki-67, Proliferation.

INTRODUCTION

The keratocystic odontogenic tumor (KCOT), formerly known as the odontogenic keratocyst (OKC), received its new designation in order to better convey its neoplastic nature.1 KCOT is noteworthy because of its ability to attain large size before any clinical signs and symptoms develop, its high recurrence rate and an association with nevoid basal cell carcinoma syndrome.2 Highest incidence of KCOT is seen in the third decade of life. Most often they are seen in mandible with a strong predilection for the molar ramus region.3 The keratocystic odontogenic tumor is believed to arise from dental lamina.1 It has been reported by many
studies that surgical enucleation with or without curettage is one of the accepted methods of treatment. Here we report a case of KCOT which was diagnosed by normal histological examination. We also carried out immunohistochemistry for Ki-67 antigen. The purpose of this presentation is to outline the aggressiveness and proliferation of Keratocystic odontogenic tumor.

**CASE REPORT**

A 36 year old male patient reported to our institute RKDF Dental College & Research Centre, Bhopal, with a complaint of swelling in the lower left back region of jaw since 3 months. Patient was apparently alright 3 months back. He noticed the swelling in the lower left back region of jaw few days back, for which he visited a local dentist who prescribed him some pain killers and referred to our institute. The swelling was smaller in size initially and came to present size gradually. The patient complained of slight discomfort. There was no history of any discharge from the swelling. His past medical history was not relevant. On Extraoral examination, no gross facial asymmetry was seen [Fig. 1]. The left and right submandibular lymph nodes were palpable, solitary, soft, freely mobile and slight tenderness was there on the left side. On Intraoral examination diffuse swelling was seen in relation to 34, 35, 36, 37 region obliterating the buccal vestibule extending antero-posteriorly from mesial surface of 33 to distal surface of 37 and superior-inferiorly from cervical region of 34 to vestibule. The size was approximately 2X4 cm, roughly oval shape. The overlying mucosa was similar to surrounding mucosa and no visible discharge was seen. On palpation all inspection findings were confirmed, the swelling being firm to hard in consistency, tender, fixed to underlying structure and there was no discharge on palpation. Grade I mobility was noted in 35, 36, 37 region. On radiographic examination, a well defined radiolucent shadow was seen in the left side of mandible, extending antero-posteriorly from distal surface of 33 to ramus region and superio-inferiorlly from interradicular region of 33, 34 to lower border of mandible [Fig. 2]. The borders were well corticated and scalloped. There was inferior displacement of mandibular canal and knife-edged root resorption was seen in relation to 34, 35, 36. An incisional biopsy was carried out for histopathology. The section was stained first with routine H & E stain. A provisional diagnosis of OKC was made. The patient was treated with surgical enucleation with curettage. Microscopic examination revealed that the epithelial lining was 6 to 8 layer thick and was parakeratinized. The basal cell layer had the typical palisaded pattern [Fig. 3]. The fibrous wall was myxoid. At places the epithelial lining was seen separating from the connective tissue [Fig. 4]. An immunohistochemical staining was done for Ki-67. High Ki-67 positivity was seen mostly in the suprabasal cell layer [Fig. 5].

**DISCUSSION**

First described by Philipsen in 1956, the Odontogenic keratocyst (OKC) is now designated by the World Health Organization as Keratocystic odontogenic tumor (KCOT) and is defined as “a benign uni- or multicystic, intraosseous tumour of odontogenic origin, with a characteristic lining of parakeratinized stratified squamous epithelium and potential for aggressive, infiltrative behavior” [4]. Recent molecular studies showing loss of heterozygosity of certain tumor suppressor genes in many odontogenic keratocysts have supported this opinion. The keratocyst is believed to originate from remnants of the dental lamina, following features such as a thin, bandlike lining of stratified squamous epithelium, a spinous cell layer 8 to 10 cells in thickness and a corrugated keratinized lining, a thin, inflammation-free connective tissue capsule, and a lumen-containing varying amounts of

Epithelial cells in KCOTs seem to have a different proliferative potential from those of other odontogenic lesions. Several studies have demonstrated the higher proliferation activity of the epithelial lining in KCOTs in relation to odontogenic cysts. Ki-67 antigen is the prototypic cell cycle related nuclear protein, expressed by proliferating cells in all phases of the active cell cycle (G1, S, G2 and M phase) and reaches a peak in the G2 and M phases. It rapidly degrades after mitosis with a half life of detectable antigen being an hour or less. It is absent in resting (G0) cells. Ki-67 antibodies are useful in establishing the cell growing fraction in neoplasms. Ki-67 positive cells were mostly detected in the suprabasal cell layer than the basal cell layer. This finding is in accordance to many studies. Kuroyanagi et al reported that high expression of Ki-67 in the basal layer of lining epithelium was seen in recurrent odontogenic keratocyst group. In contrast, non-recurrent odontogenic keratocyst group expressed Ki-67 mostly in suprabasal layers. These findings suggest that the epithelial lining may have an intrinsic growth potential. Enucleation is the preferred choice of treatment. Our patient was treated with enucleation with surgical curettage.

CONCLUSION

KCOTs are one of the aggressive lesions of the oral cavity. The chances of recurrence rate of KCOT is very high owing to presence to satellite cysts and thin and friable lining, along with the intrinsic potential of the lining. It has been proved time and again that the proliferative activity of KCOTs is very high. More work needs to be done to confirm if there is a role to be played by apoptosis. Also, due to its aggressive behavior the treatment requires proper skill to remove the complete lining of the cyst along with the fibrous wall.

REFERENCES

Figure 1: Extraoral examination showing no gross asymmetry.

Figure 2: Orthopantomogram showing well defined radiolucent shadow in the left side of mandible.

Figure 3: Lining epithelium showing parakeratinization and basal cell layer showing palisaded pattern.

Figure 4: Epithelial lining is seen from connective tissue.

Figure 5: Ki-67 positivity is seen mostly in the suprabasal cell layer.