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ENUCLEATION WITH ADJUVENT THERAPY FOR TREATMENT OF AGGRESSIVE ODONTOGENIC KERATOCYST IN MANDIBLE: A CASE REPORT

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Abstract:
The maxillofacial region is frequently affected by the developmental odontogenic cyst known as an odontogenic keratocyst (OKC), which develops from the remnants of the dental lamina. Due to their ability to grow aggressively, potentially recur, and be linked to hereditary disorders, it is crucial to have an accurate diagnosis for OKC. The OKC stands out among jaw cysts and has a propensity to return associated with combative clinical behavior. This report discusses, a 27-year-old female, experiencing pain and swelling in the mandibular left anterior and posterior regions. After reviewing medical history and conducting medical, radiological, along with histopathological examinations, it was determined that she had an odontogenic keratocyst. In order to treat this condition, surgical enucleation, which is a conservative strategy known for its positive outcomes and BIPP packing have been utilized. The process of bone formation completes over time and the swelling shrinks in size.

Keywords: OKC, Enucleation, BIPP, KCOT, Jaw Cyst

INTRODUCTION

In 1956, Phillipsen categorized the odontogenic keratocysts.[1]The histological criteria for the diagnosis of OKC were first proposed by Pindborg and Hansen in 1962.[2] Among almost all oral cavity cysts, odontogenic cysts show a high level of aggressiveness. It is described as "a benign unicystic or polycystic, odontogenic origin, lined by para keratinized stratified squamous epithelium with the potential for invasive, infiltrative behaviors". OKC was categorized by the WHO as keratocystic odontogenic tumor (KCOT) in 2005 because it more closely resembles its neoplastic character and has PTCH1 gene mutation or inactivation[3], but this is not unique because this gene is also present in other developing cysts. Later In 2017, the WHO classification of Head and Neck tumors reclassified KCOT as a cyst and declined the prior term KCOT, as It is now thought of as a developmental cyst with more aggressive behavior[4] because the decrease in recurrence rate & features are unrelated to the tumor. With a slight male preference, OKC develops from the cell remains of the dental lamina and is typically diagnosed between the second and fourth decades of

life. Most of the patients are in the 20–29 and 40–59 age ranges.[5]The angle of the mandible and the posterior area of the maxilla often contain a combined 70% to 80% of the keratocysts.[1, 5] Growth occurs most frequently in the anteroposterior direction, and lesions can grow to vast sizes without noticeably altering the jaw skeleton. The higher activity of the cyst in the epithelial cells causes a higher hydrostatic pressure difference, which in turn stimulates the osteolytic activity of prostaglandins in the cells lining the cyst and the accumulation of hyperkeratotic scales in the cyst lumen, which tends to grow quickly.[6] This report presents a case concerning a woman who is 27 years old and has an OKC situated in her mandible.

CASE REPORT:

A 27-year-old female patient reported to the Department of Oral and Maxillofacial Surgery, Mahatma Gandhi Dental College and Hospital, with a complaints of swelling and pus discharge from the lower back tooth region for the past 6 months. There were no systemic diseases present, according to medical history. There is no history of any negative habits. An apparent facial asymmetry and swelling that measured approximately 4 cm x 5 cm and extended from the left body of the mandible to the right side of the parasymphysis region were visible on extraoral inspection. On palpation, the swelling was firm and sensitive. It extended inferiorly up to the lower border of the jaw and superiorly from below the lower lip.

On intraoral examination, there was vestibular obliteration and a widespread swelling running from the distal surface of tooth 36 to the distal surface of tooth 43, crossing the midline. [figure 1] On palpation, it was firm, and tender, and discharge was present. On aspiration, straw-colored fluid was present. A provisional diagnosis of OKC of the mandible with a differential diagnosis of unicystic ameloblastoma was made in light of the clinical finding mentioned above. An unilocular radiolucency with well defined borders, thinning cortical bone, and cortical expansion in the body region was visible on the panoramic radiograph (orthopantomogram) that extends from the mesial surface 37 to mesial surface of 45 teeth. Radiographic diagnosis of OKC was made along with a unicystic ameloblastoma and dentigerous cyst as a differential diagnosis.

Fig 1: Intraoral Swelling Extending From 36 To 43 Teeth Region

Fig 2: Panoramic Radiograph Showing Unilocular Radiolucency from 36 To 44

Fig 3: Intraoperative Photos of Lesion after Extraction and Marsupialization

Fig 4: OPG Showing Cavity Filled With BIPP Packing After Marsupialization

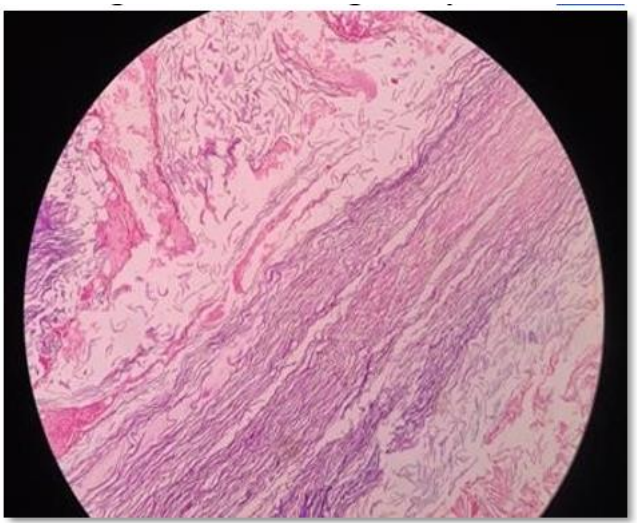

Fig 5:- Histopathological Slide

Revealing Cystic Lining with Hyperplastic Squamous Cell with Mild Hyperparakeratosis and Acanthosis suggestive of OKC

Operative procedure

After taking the informed consent of the patient, enucleation was done under local anesthesia. Under an aseptic condition, through intraoral incision, the fullthickness mucoperiosteal flap was raised. The lesion was enucleated, and the affected teeth were removed. It was necessary to remove the bone's thinned-out inner cortical lining. The cystic lesion was sent for histopathologic examination [Figure 5]. The corpal cavity was enucleated, and Bismuth Iodoform Paraffin Paste (BIPP) ribbon

placed in the cavity. Finally, the wound closure was done with a 3-0 mersilk suture. The final diagnosis, according to the histopathology report, is odontogenic keratocyst.

From one week after surgery until two years later, the patient underwent routine follow-up. Swelling was greatly reduced, and radiographically, bone growth could be seen eight months later. Two years after surgery, radiographic findings revealed healthy bone formation and a decrease of radiolucency in the cystic cavities.

Discussion:

The aggressive clinical and histologic lesion known as the OKC is distinct and common. Though some speculate that the basal cell component may be its likely origin, it typically originates in the dental lamina.[7] The mandible is involved in 70% or more of cases, particularly in the third molar, angle, and ramus regions. The maxillary third molar is the next most frequent location, followed by the mandibular premolar and maxillary canine region.[8] In contrast to the ortho-keratinized variation seen in OKC, the KCOT displays locally damaging and highly recurring behavior and is defined by para-keratinized epithelium. The basal layer budding into the connective tissue and numerous mitotic figures are revealed by KCOT. The Gorlin syndrome, also known as the bifid rib basal cell nevus syndrome, typically has several keratocysts.[9] Treatment options for OKC include a conservative treatment called enucleation and decompression, which eventually result in the cystic lesion completely disappearing.

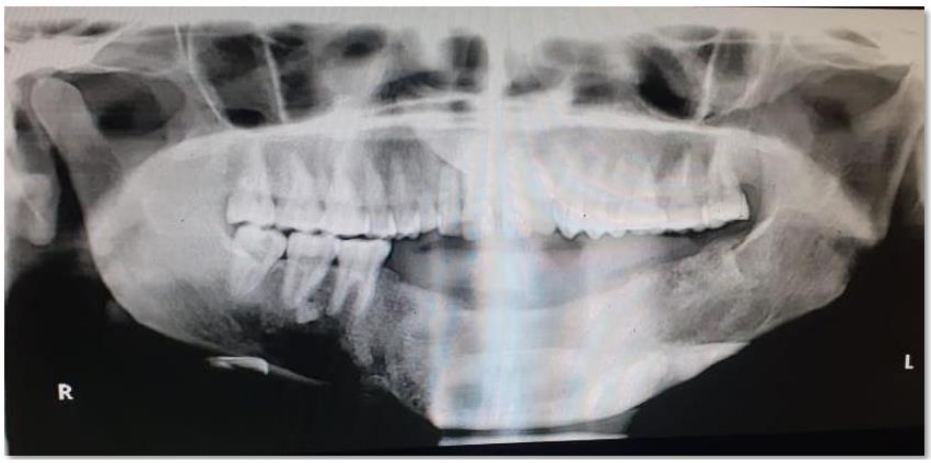

Fig 6: Follow up OPG after 2 years

They are recommended for limiting injury to other important tissues, preserving bone and teeth, and reducing the risk of pathologic fracture. These treatments work by exposing the cystic osmotic pressure to the surrounding oral cavity, which lowers it. It aids in the gradual shrinkage of the cyst as well as bone deposition near the lesion's edge. Segmental resection, en bloc surgery, and complex procedures are further therapy options.[10]

Rutherford Morison initially described bismuth iodoform paraffin paste (BIPP) in 1917. It is made up of 220g of paraffin base, 220g of bismuth subnitrate, and 440g of iodoform.[11]

The reported enucleation recurrence rate ranges from 17 to 56%. In order to remove any remaining cyst lining or islands within the cyst wall, many surgeons choose combining enucleation with adjuvant therapy. When both enucleation and adjunctive treatment are used together, a total reduction in the recurrence potential of 18% is observed.[12]

Conclusion

A precise diagnosis and a planned course of therapy are required for OKC due to its aggressive nature and high recurrence rates. It can be treated surgically or conservatively. Although the conservative approach necessitates long-term monitoring, it is advantageous and safe. However,

further study is still needed to determine the best therapy options for OKC given its genetic and molecular pathophysiology.

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