ijlpr 2023; doi 10.22376/ijlpr.2023.13.5.P90-P97

International Journal of Life science and Pharma Research

ISSN 2250-0480

Case Report

Odontogenic Keratocyst Mimicking Ameloblastoma

A Case of Odontogenic Keratocyst in the Posterior Ramus and Body of the Mandible Mimicking Ameloblastoma

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Abstract: The dental lamina gives rise to the odontogenic keratocyst (OKC), a common odontogenic cyst that affects the maxillofacial region. The OKC is distinct from other jaw cysts and is more likely to return when combined with aggressive clinical behavior. The recurrence rate in OKC is 25%–30%. In 2005, the World Health Organization (WHO) group categorized odontogenic keratocyst (OKC) as a tumor and proposed the abbreviation KCOT to differentiate the condition from the ortho keratinizing variant. The WHO reclassified KCOT as OKC in 2017 based on data demonstrating non-neoplastic clinical behavior. The 30-year-old male in this case study has OKC in the ramus and body of his jaw, near the right mandibular molar tooth. When the patient reported to us, he complained of pain and swelling, 48 was clinically absent, and there was an enlargement of the buccal and lingual cortical plates. Palpation revealed tenderness in the region distal to 48. CBCT revealed a sizable radiolucent lesion, making it difficult to determine whether it was an odontogenic keratocyst or ameloblastoma in the body and lower part of the ramus on the right side. Histopathology findings corroborated OKC's diagnosis. As a precaution, the affected teeth were extracted, marsupialization was done, and an ongoing follow-up was done for an additional 1.5 years. There hasn't been a recurrence of OKC, and it showed good healing. This case study aims to demonstrate the need for dentists to do in-depth investigations into each circumstance and offer patients better treatment options with ongoing patient monitoring and follow-up. Enucleation, marsupialization, and other surgical techniques are possible; however, in this case, our objective was to preserve the patient's mandible and facial features.

Keywords: keratocystic odontogenic tumor, odontogenic keratocyst, Keratocyst, Marsupialization, Odontogenic Ameloblastoma.

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Received On 28 January, 2023
Revised On 3 April, 2023
Accepted On 17 April, 2023
Published On 1 September, 2023

Funding This research did not receive any specific grant from any funding agencies in the public, commercial or not for profit sectors.

Citation Dr. Rakhi Chandak, Zareesh Akhtar and Dr. Manoj Chandak , A Case of Odontogenic Keratocyst in the Posterior Ramus and Body of the Mandible Mimicking Ameloblastoma.(2023).Int. J. Life Sci. Pharma Res.13(5), P90-P97
http://dx.doi.org/10.22376/ijlpr.2023.13.5.P90-P97

1. INTRODUCTION

The cystic lesions that most frequently affect the maxillofacial region are odontogenic cysts. They are generally divided into two groups: one for development, which includes keratocyst and dentigerous cysts, and the other for Inflammation, which includes radicular cysts.[1] The odontogenic keratocyst (OKC) was first described by Philipsen (1956), in 2005, it was designated by the World Health Organization as a keratocystic odontogenic tumor (KCOT). Then reclassified as 'odontogenic keratocyst (OKC)' in 2017.[2] It is "a benign unicystic or multicystic intraosseous tumors of odontogenic origin, with a unique lining of para keratinized stratified squamous epithelium and propensity for aggressive, infiltrative tendencies." .[3]The percentage of OKC versus other cysts of the jaws as given by different authors:[4]Hjorting-Hansen et al. (1969)[5] and Toller (1972)[6] 11%; Brannon (1976)[7] and Payne (1972)[8] 9%; and Pindborg and Hansen (1963)[9] 7%. The lesions can grow remarkably without appreciably deforming the jaw skeleton because growth occurs mostly in the anteroposterior dimension. The propensity for rapid growth is brought on by increased osteolytic activity of prostaglandin substances in the cell population of the cyst lining, higher activity of the epithelial cells lining the cyst, and higher accumulation of hyperkeratotic scales in the cyst lumen, resulting in a greater hydrostatic pressure difference.[10] The OKC has a unique and prevalent clinical and histologic lesion with aggressive nature. It often develops in the dental lamina, but some speculate that basal cell component may be its likely source.[11] The etiology of KCOT is related to the development of the dental lamina and, in particular, remnants of it after it has served its purpose. These dental lamina-derived epithelial islands are primarily seen in the periodontal ligament and gingiva. It clarifies the clinical entity of OKCs lateral follicular or periodontal appearance.[12] The characteristic features of OKC are the tendency to grow along the cancellous channels with very little cortical expansion. Numerous hypotheses regarding the growth of KCOT have been put forth. These include intraluminal hyperosmolality, active epithelial proliferation, the collagenolytic activity of the cyst wall, and synthesis of interleukin 1 and 6 by keratinocytes which will induce the secretion of keratocyte growth factor from interactive fibroblasts along with tumor necrosis factor leading to increased levels of prostaglandins and expression of the parathyroid related protein.[12] The mandible is involved in 70% or more cases, particularly in the third molar, angle, and ramus regions. Next, the most common site of occurrence is the maxillary third molar, followed by the mandibular premolar and maxillary canine region.[11] The unique clinical characteristics of OKC are the local destruction and tendency for diversity, especially when associated with syndromes like the naevoid basal cell carcinoma syndrome or Gorlin Gotz syndrome. The pathology's epithelial genesis is beyond question, but the causes are still a topic of speculation. The tumor predicates the mandibular 3rd molar region and usually manifests as multilocular radiolucency with well-defined scalloped margins.[12]

2. CASE REPORT

A 30-year-old male patient reported to our dental institute with a chief symptom of pain & swelling in the lower right back region of the jaw for 4-5 days. The patient was all right before 4 months; then, he experienced pain in the lower right mandibular area for which he had taken analgesics. Then 4-5 days back patient again experienced pain and swelling in the same area, for which the patient reported to the Department of Oral Medicine and Radiology. The pain was dull, aching in nature & non-radiating, which was not relieved after taking medication no history of trauma, no history of balm application, hot or cold fomentation.

2.1. Medical history

His dental background was unremarkable. Yet, according to medical documents, two days ago before the case report to the investigator, analgesics were repeatedly taken by the patient for chronic pain and swelling (directly purchased from the pharmacy).

2.2. Family History

The patient has no family history of diabetes, hypertension, asthma, or tuberculosis. When questioned about his past, he claimed that he had never smoked, chewed betel nuts or tobacco, drunk alcohol, or engaged in other harmful behaviors.

2.3. Observation

The patient's asymmetrical face with a 15 mm mouth opening on the right side was observed during the extraoral examination. The mouth opening showed a little deviation to the right. Palpatory results showed bilaterally synchronized motions without any clicking noise. Masticatory muscles appeared to be normal. There was no palpable submental or submandibular lymph node. (Fig-1).

Fig 1: (a, b, c) Extraoral view showing gross facial asymmetry on the right side of the face; (d) Mouth opening 15 mm approx.

A single diffuse swelling was seen on the right lower region of the jaw, extending Anteroposteriorly from 4 cm away from the corner of the mouth to the posterior border of the ramus of the mandible and superoinferiorly from 1cm below the sigmoid notch to the lower border of the mandible, of size:4*3cm approx. The skin over swelling was the same as adjacent. The surface was smooth. The swelling was nonreducible and non-compressible. The patient complained of paresthesia on the lower lip (right side). The swelling was afebrile to touch, hard, and tender on palpation. Intraoral examination revealed no visible pulsation, discharge, and sinus formation in the area of chief complaint. The buccal and lingual cortical plates of the right body and ramus (i.e.,47 & 48 areas) region of the jaw were expanded on palpation. No mobility of the tooth. Tenderness was present distal to 47 (fig-2)

Fig2: Intraoral view revealing clinically missing 48 and intraoral swelling distal to 47.

Clinically missing 48 were seen. Hence, a provisional diagnosis of the dentigerous cyst with 48 was made.

2.4. Special tests and investigation

OPG was done to examine the extent of the lesion (fig-3). The panoramic radiographic revealed a well-defined Unilocular radiolucency with scalloped and corticated margins in the mandibular posterior right body and ramus extending to 47,48 regions (fig-3).

Fig 3: OPG radiograph showing (encircled in red circle) a well-defined Unilocular radiolucency with scalloped and corticated margins seen in the mandibular posterior right body and ramus extending to 48,47 regions.

CBCT was done to examine the extent of the lesion three-dimensionally. (fig 4&5)

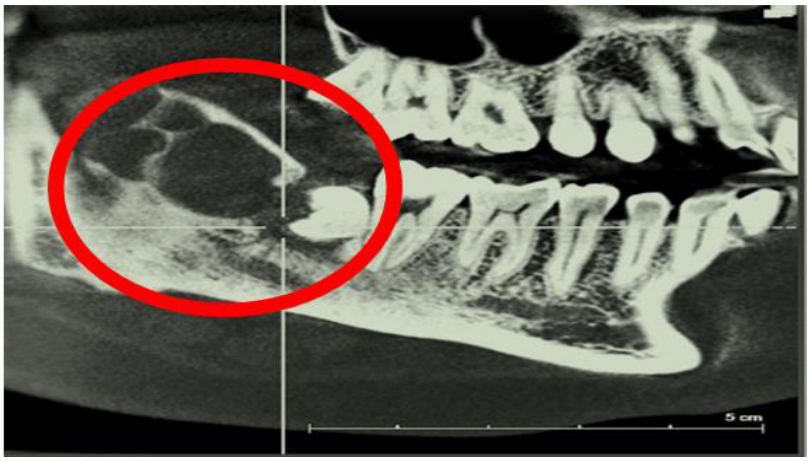

Fig4: CBCT revealed a well-defined multilocular radiolucency with scalloped and corticated margins in the mandibular posterior right body and ramus extending to 48 regions. (Encircled in red).

The CBCT revealed in the mandibular posterior right body and ramus a well-defined multilocular radiolucency with scalloped and corticated edges extending to 48 areas, with a size of approx. 8.4 × 23.9 mm. Internally, there was radiolucency and bone septa. The inferior alveolar canal displaced inferiorly.

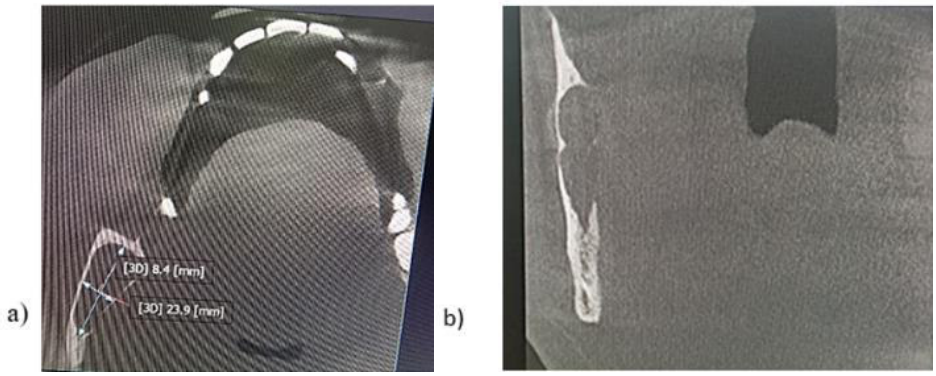

Fig 5: CBCT showing the extent of the lesion in a) axial, b) coronal view

(a) axial section showing the buccolingual expansion of the right-side body and ramus of the mandible. There is evidence of an impacted tooth within the osteolytic area. The cortical plates are intact. (b) coronal section revealing thinning of the right lingual cortical plate

A radiographic diagnosis of a Combined lesion of–Ameloblastoma in the ramus of the mandible on the right side & Odontogenic keratocyst in the body and lower part of the ramus was taken into consideration. Fine needle aspiration cytology yielded 0.5 ml thick yellowish-white cheesy material (fig 6). which, on cytological examination, revealed (PAP-stained smears) large RBCs,

lymphocytes, neutrophils, and abundant plasma cell infiltration along with areas of superficial epithelial cells adjacent to the RBC. The overall features were suggestive of Chronic Inflammation.

Fig 6: Fine needle aspiration revealed 0.5 ml thick yellowish-white cheesy material

2.5. Diagnosis

Henceforth, the final diagnosis of OKC was made with 48 tooth regions and the right ramus of the mandible.

2.6. Treatment plan

After taking the informed consent of the patient, marsupialization with peripheral osteotomy of the surgical bed was completed, followed by a single application of carnoy's solution, and extraction of 46,47,48, and 28 was done. Finally, the wound was closed with a 3-0 mursilk suture (Fig-7). The dressing was done with betadine ribbon gauze at intervals of 3 days postoperatively for 1 month. The cystic lining of the right retromolar area was removed and transported to the oral pathology department for histological investigation. The incisional biopsy revealed a 6-8 cell layer thick para keratinized stratified squamous epithelium (Fig-8). The epithelium lacked rete ridges and appeared detached from the underlying connective tissue in some areas. There were a lot of fibroblasts; collagen fibers grouped haphazardly, and a few blood capillaries here and there in the connective tissue. Chronic inflammatory cell infiltration was seen in the connective tissue area devoid of epithelium. A large region of extravasated blood element was visible in a few tissue fragments. The overall appearance suggested an inflammatory odontogenic keratocyst.

Fig 7- intraoral view after marsupialization, peripheral bone resection, and extraction of 46,47,48.

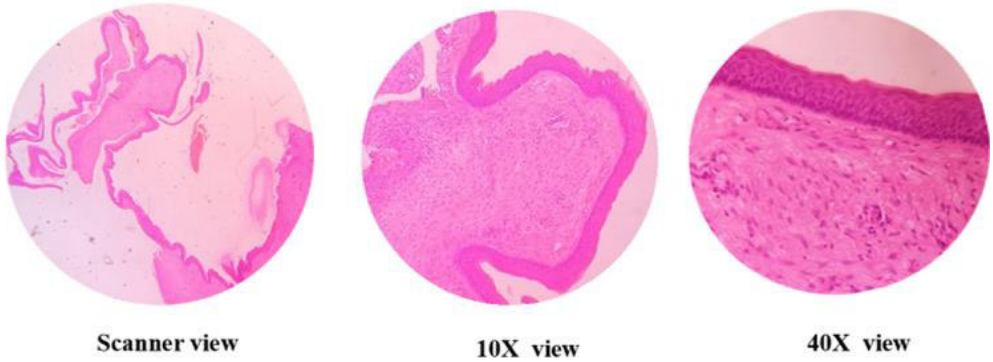

Fig 8: Photomicrograph showing para keratinized stratified squamous epithelium of 6-8 cell layer thick with epithelial lift from the connective tissue showing cleft formation (H&E, scanner, 10X, and 40 X magnification).

2.7. *Follow-up*

The patient is followed up every three months, and no recurrence is noted (fig-9).

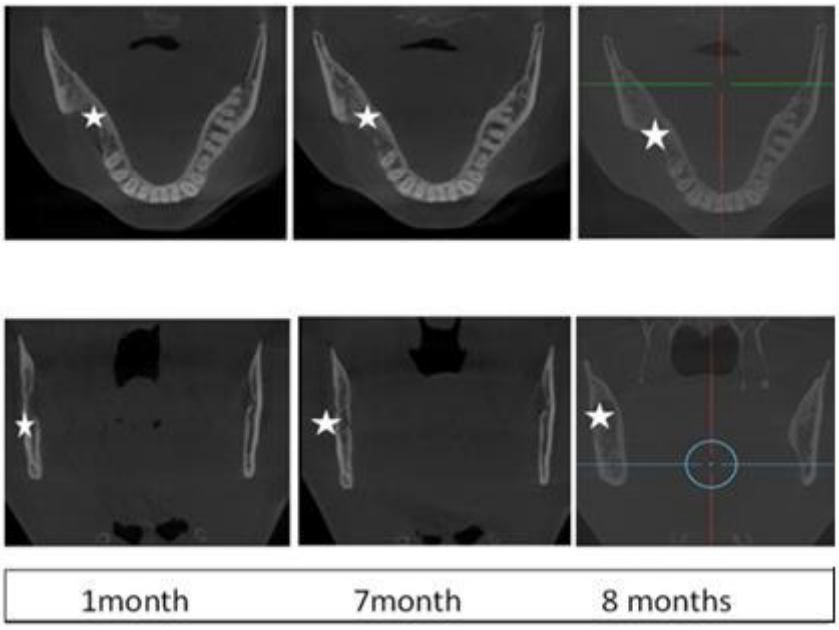

Fig 9-Postoperative radiograph after surgery at a 1,7, and 8-month follow-up showing complete resolution of the lesion as evidenced by replacement of radiolucency by radiopaque bone (represented by a white star) in the buccal and lingual bony perforation.

3. DISCUSSION

According to a study conducted by Borgehesi A et al. in 2018, OKC account for around 10% of all odontogenic cysts. The reported age distribution ranges from 8 to 82 years, with a high occurrence in the third decade of life. There is a slight male bias[13]. The same findings were observed in our case. A study by Hasen EH et al. in 1969 highlighted that keratocyst in the jaw appeared to be non-inflammatory. The epithelium is most likely derived from epithelial remnants from the dental lamina, and the researchers concluded that the dental lamina could yield keratinized epithelium.[5] Toller PA et al. in 1972 featured that the keratinizing epithelium surrounding the odontogenic keratocyst shows cell maturation rather than degeneration and can expand in size primarily through epithelial cell multiplication. The turnover of epithelial cells in odontogenic keratocyst is higher than in other cysts. [6] Yazdani J et al. in 2009 pointed out that OKC is unique among jaw cysts as it frequently recurs and is related to aggressive clinical behavior. OKC has a risk of relapse of 25-30%.[1] Brannon RB and Pinborg JJ suggested in their study that OKC has a peak incidence in the second and third decades of life. The mandible to the maxilla ratio was 2:1, and the ramus and third molar regions of the jaw were the most often used areas. The same was observed in this case; the lesion involved the mandible's body up to the mandible's angle and ramus. The dentigerous cyst was the most common diagnosis for keratocyst. These cysts may be very aggressive clinically. Their radiographic appearance was quite variable, although they resembled ameloblastomas; the same appearance was evident in our case, which caused a dilemma in arriving at a diagnosis. They have a high recurrence rate compared to other types of odontogenic cysts. [7,9] Haring JI conducted a study on OKC in 1988 and concluded that odontogenic keratocysts identified as multilocular lesions appeared to be larger, contain a more severe inflammation, and exhibit the presence of cholesterol granulomas more often than those identified as unilocular lesions. [10] Soni PK et al., in a case report in 2021, highlighted that radiographically, OKC presents predominantly as a unilocular radiolucency with well-developed sclerotic borders. They may also present as a multilocular radiolucency with a ratio of unilocular to multilocular varying from 3:112 to 1:1.3 .[11]Borghesi A et al. in their study done in 2018 stated that OKCs originate from the mandible, most common location is the posterior sextant, the angle, or the ramus. Large-size lesions are particularly common at the mandible's angle and ramus, which is evident in our case. Despite their aggressive behavior, OKCs usually cause minimal bone expansion because of their propensity to spread along the intramedullary space, "growing in the length of the bone." Large lesions, causing significant erosion of cortical plates and involvement of surrounding structures, may be seen in asymptomatic patients .[13]A study done by Pereira R.M.A et al. in 2018 concluded that large mandibular OKCs tend to grow predominantly along the length of the bone with minimal buccolingual expansion, especially within the body.[14] A study by MacDonald D in 2016 highlighted the unusual growth pattern on panoramic radiography, which may reveal a large radiolucent lesion with significant mesiodistal dimensions but no significant cortical extension. Large maxillary OKCs, on the other hand, show a substantial growth of the alveolar bone and frequently affect neighbouring structures. [15] In our case, the same thing was observed. Patients with OKC are usually asymptomatic in 80% of cases and continue to be that way for nearly 2 years. However, the most common symptoms are pain, edema, and secretions; in our case, patients report pain and swelling as their primary complaints. Investigations are done, which are essential for establishing a quick diagnosis. Typical radiographic features of OKC include radiolucent lesions with smooth and corticated margins, which can be unilocular (the most prevalent) or multilocular. With weird or non-existent tooth displacement, anteroposterior intraosseous growth can be seen. In this case, the patient had a unilocular lesion associated with tooth 48 and expansion of

the buccal and lingual cortical plates. The odontogenic keratocyst has varied and non-specific radiographic findings. In Browne's study (1970), 56 percent were unilocular, 20 percent had a single cavity with a scalloped margin, and 30 percent were multiloculated and invaded the entire jaw[16] same pattern was present in this case; the radiographically large multilocular radiolucency was seen involving the mandible and ramus area. The most appropriate treatment for OKC is still contentious and sparks much debate. Because of its histologic characteristics and noticeably less aggressive activity, some pathologists believe the ortho-keratotic subtype should be recognized as a distinct organism and dubbed an ortho-keratotic odontogenic cyst. This article's lesion is an ordinary parakeratotic OKC.[17] An OKC is typically described as having a thin, friable lining after surgery. The most common gross characteristic is fluid with caseous or cheeselike debris filling the cystic lumen. The fluid FNAC aspirated in this instance shared the same qualities as those mentioned in earlier research. When examining the gross surgical material before the histologic study, these distinctive characteristics typically enable the diagnosis of OKC. The report's gross findings were consistent with an OKC that was parakeratotic. [17]The radiological characteristics of OKCs include the following: 1) A corticated border that is frequently scalloped; 2) Development over the length of the mandibular bone, especially towards the lingual (medial) side; 3) Extrusion of erupted teeth, detachment or resorption of the roots of erupted teeth, and displacement of developing teeth; 4) Radiolucency of the lumen and, on rare occasions, a hazy or milky appearance of the lumen on the panoramic radiograph; 5) Sporadic bilateral or multiple cysts, which may be indicative of basal cell nevus syndrome.[10,18,19] Decompression and marsupialization, according to some writers, are better processes because they are more conservative and preserve important anatomical elements.[11] Others believe that therapy should be done in two stages: marsupialization first and then enucleation second. Some writers recommend just conservative enucleation with Carnoy's solution for OKC.[13] In 2012, Tabrizi R conducted a brief clinical investigation on 13 patients (8 males, 5 females) with biopsy-proven OKC, ranging in age from 16 to 31 years (mean, 22.4 y). Cyst diameters in the patients ranged from 25 to 90 mm when measured radiographically. Marsupialisation was used as a form of treatment. Post-treatment visits were made at intervals of six months, and the investigators followed up with the patients for at least 60 months. After finding that marsupialization is a successful and conservative treatment option for odontogenic keratocyst (OKC), they concluded that the cyst walls shrunk in three individuals and disappeared in ten patients. [20] In this case, marsupialization was intended, and the impacted tooth was extracted. The patient was followed up every three months, and no recurrence has been noted. This observation was the same as noted in previous studies. To contribute to the literature, we underline the need for long-term follow-up to identify any lesion-related recurrence when it does.

4. CONCLUSION

In this case report, a 30-year-old male patient with a cystic swelling clinically and radiographically mimicking ameloblastoma in the right-side ramus and body of the mandible. The histopathologic report supported the diagnosis of OKC. Although the OKC exhibited aggressive biological characteristics, in our case, the affected tooth was extracted, the cyst was marsupialisation and a long-term follow-up was conducted. It showed proper healing, and as of this writing, OKC has not returned. Enucleation, marsupialization, and other surgical techniques are possible; however, we aimed to preserve the patient's mandible and facial features in this case. This case study intends to demonstrate the need for dentists to thoroughly evaluate each scenario and provide better treatment options with long-term patient follow-up.

5. AUTHORS CONTRIBUTION STATEMENT

Rakhi Chandak, Zareesh Akhtar, and Manoj Chandak conceived the presented idea. Zareesh Akhtar performed the computations. Rakhi Chandak verified the diagnosis. All authors discussed the case and contributed to the final manuscript. Rakhi Chandak and Zareesh Akhtar wrote the manuscript with support from Rakhi Chandak & Manoj Chandak supervised the project. Zareesh Akhtar took the lead in writing the manuscript. All authors provided critical feedback and helped shape the manuscript Rakhi Chandak and Manoj Chandak conceived the study and oversaw the overall direction and planning.

6. CONFLICT OF INTEREST

Conflict of interest declared none.

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