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CASE STUDY

ENUCLEATION AND CHEMICAL CAUTERIZATION OF ODONTOGENIC KERATOCYST FOLLOWED BY PROSTHETIC REHABILITATION WITH DENTAL IMPLANTS; 2 YEAR FOLLOW UP

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ABSTRACT

First described by Philipsen in 1956, the odontogenic keratocyst is characterized by a large squamous keratinization of its border, an aggressive growth behaviour, a high recurrent rate and characteristic histological appearance. It is now designated by the World Health Organization as a keratocystic odontogenic tumour (KOT). Clinically, the KOT is manifested by an asymptomatic growth. Radiographically, it appears as a well-defined unilocular or multilocular osteolytic lesion. The diagnostic approach is based on a combined analysis of the medical history, the clinical appearance and the radiographic appearance. The diagnosis may be confirmed by the histopathological report. Finally, treatment consists of surgical excision and follow up is characterized by a high rate of recurrence.

Case Presentation: In this case report we present a 51-year-old male patient with a large odontogenic keratocyst and treatment with Enucleation and Chemical cauterization followed by dental implants with a 2 year follow-up. According to the patient's age and clinical findings, implants supported fixed prosthesis was the treatment of choice.

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INTRODUCTION

Odontogenic cysts and tumors constitute an important aspect of oral and maxillofacial pathology. Odontogenic cysts are encountered relatively commonly in dental practice. Odontogenic tumors, by contrast, are uncommon lesions. Even in the specialized oral and maxillofacial pathology laboratory, fewer than 1% of all specimens received are odontogenic tumors. With rare exceptions, epithelium-lined cysts in bone are seen only in the jaws. Other than a few cysts that may result from the inclusion of epithelium along embryonic lines of fusion, most jaw cysts are lined by epithelium that is derived from odontogenic epithelium. These are referred to as odontogenic cysts. Odontogenic cysts are subclassified as Developmental or Inflammatory in origin. The inciting factors that initiate the formation of Developmental cysts are unknown, but these lesions do not appear to be the result of an inflammatory reaction. Inflammatory cysts are the result of inflammation. The following table presents categories of odontogenic cysts modified from the 2005 World Health Organization (WHO) classification.

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Classification of Odontogenic Cysts

Developmental

- Dentigerous cyst
- Eruption cyst
- Odontogenic keratocyst*
- Orthokeratinized odontogenic cyst
- Gingival (alveolar) cyst of the newborn
- Gingival cyst of the adult
- Lateral periodontal cyst
- Calcifying odontogenic cyst†
- Glandular odontogenic cyst

Inflammatory

- Periapical (radicular) cyst
- Residual periapical (radicular) cyst
- Buccal bifurcation cyst

[*Although the odontogenic keratocyst is included with the odontogenic tumors in the 2005 World Health Organization (WHO) classification ("keratocystic odontogenic tumor"), the authors prefer to classify it as an odontogenic cyst]

Odontogenic Keratocyst A cyst derived from the remnants (rests) of the dental lamina, with a biologic behaviour similar to a benign neoplasm, with a distinctive lining of 6 to 10 cells in thickness, and that exhibits a basal cell layer of palisaded cells and a surface of corrugated parakeratin. This is the most interesting of jaw cysts. The term 'odontogenic keratocyst' was first used by Philipsen in 1956, while Pindborg and Hansen in 1963 described the essential features of this type of cyst. It is named keratocyst because the cyst epithelium produces so much keratin that it fills the cyst lumen. Furthermore, flattening of the basement membrane and palisading of the basal epithelial cells, reminiscent of odontogenic epithelium, are characteristics of odontogenic keratocyst. The odontogenic keratocyst is a distinctive form of developmental odontogenic cyst that deserves special consideration because of its specific histopathologic features and clinical behavior. There is general agreement that the odontogenic keratocyst arises from cell rests of the dental lamina. This cyst shows a different growth mechanism and biologic behavior from the more common dentigerous cyst and radicular cyst. Most authors believe that dentigerous and radicular cysts continue to enlarge as a result of increased osmotic pressure within the lumen of the cyst. This mechanism does not appear to hold true for odontogenic keratocysts, and their growth may be related to unknown factors inherent in the epithelium itself or enzymatic activity in the fibrous wall. Several investigators have suggested that odontogenic keratocysts be regarded as benign cystic neoplasms rather than cysts, and in the latest WHO classification of odontogenic tumors, these lesions have been given the name "keratocystic odontogenic tumor." The arguments to support this change in nomenclature largely rely on a few studies that have shown certain molecular genetic alterations that are also present in some neoplasms. Although there are wide variations in the reported frequency of odontogenic keratocysts compared with that of other types of odontogenic cysts, several studies that include large series of cysts indicate that odontogenic keratocysts make up 3% to 11% of all odontogenic cysts.

They are unique odontogenic lesions that have the potential to behave aggressively, that can recur, and can be associated with the nevoid basal cell carcinoma syndrome. Toller (1967) suggested that OKCs might be regarded as benign cystic neoplasms. Whether they are developmental or neoplastic continues to be debated. Studies indicate that a significant number of OKCs show clonal loss of heterozygosity of common tumor suppressor genes. The finding of clonal deletion mutations of genomic DNA in these cysts supports the hypothesis that they are neoplastic rather than developmental in origin. The odontogenic keratocyst is regarded as a distinctive entity because of its characteristic histology, proliferation kinetics, and behavior. Therefore, although keratinization may be present in many other types of cysts, the specific histologic pattern of the odontogenic keratocyst separates it from all others. Differences in cytokeratin, epithelial membrane antigen (EMA) and carcinoembryonic antigen (CEA) immunoreactivity between the parakeratinized OKC and the orthokeratinized variety have been demonstrated and the suggestion made that the latter having a considerably less aggressive behavior is different entity and should bear a different name orthokeratinized odontogenic cyst (Shear, 2002). There is general agreement that the origin of the odontogenic keratocyst comes from dental lamina remnants in the mandible and maxilla. However, the origin of this cyst from the extension of basal cells of the overlying oral epithelium has also been suggested.

Reclassification of the Odontogenic Keratocyst to Tumor: Keratocystic Odontogenic Tumor (KOT)

In 1967, Toller suggested that the OKC may best be regarded as a benign neoplasm rather than a conventional cyst based on its clinical behavior. The WHO has reclassified the lesion as a tumor based on several factors, that formed the basis of this decision.

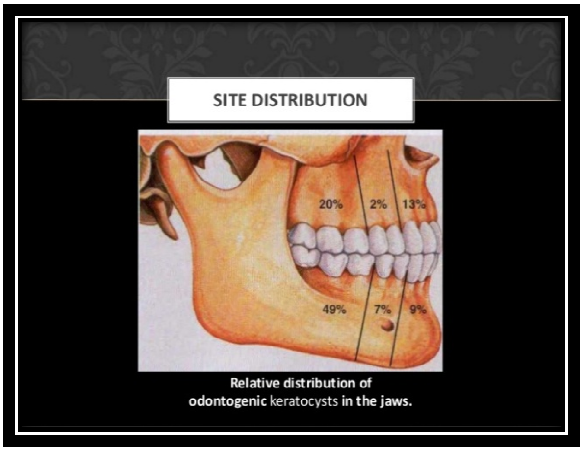
Behavior: The KOT is locally destructive and recurrence rate is very high.

Histopathologic features: The basal epithelial layer of KOT shows proliferation and budding into the underlying connective tissue in the form of daughter cysts and mitotic figures are frequently found in the suprabasal layers of the lesional epithelium. The odontogenic keratocyst typically shows a thin, friable wall, which is often difficult to enucleate from the bone in one piece. The cystic lumen may contain a clear liquid that is similar to a transudate of serum, or it may be filled with a cheesy material that, on microscopic examination, consists of keratinaceous debris. Microscopically, the thin fibrous wall is essentially devoid of any inflammatory infiltrate. The epithelial lining is composed of a uniform layer of stratified squamous epithelium, usually six to eight cells in thickness. Histologically, these cysts are formed with a stratified squamous epithelium that produces orthokeratin (10%), parakeratin (83%), or both types of keratin (7%). The epithelium and connective tissue interface is usually flat, and rete ridge formation is inconspicuous. Detachment of portions of the cyst-lining epithelium from the fibrous wall is commonly observed. The luminal surface shows flattened parakeratotic epithelial cells, which exhibit a wavy or corrugated appearance. The basal epithelial layer is composed of a palisaded layer of cuboidal or columnar epithelial cells, which are often hyperchromatic. Small satellite cysts, cords, or islands of odontogenic epithelium may be seen within the fibrous wall. These structures have been present in 7% to 26% of cases in various reported series. In rare instances, cartilage has been observed in the wall of an odontogenic keratocyst.
In the presence of inflammatory changes, the typical features of the odontogenic keratocyst may be altered. The parakeratinized luminal surface may disappear, and the epithelium may proliferate to form rete ridges with the loss of the characteristic palisaded basal layer and may ulcerate. When these changes involve most of the cyst lining, the diagnosis of odontogenic keratocyst cannot be confirmed unless other sections show the typical features described earlier. Some investigators recognize a microscopic orthokeratotic variant and include this lesion as a subtype of the odontogenic keratocyst. The variant of OKC that produces only orthokeratin acts somewhat differently than other OKCs. These almost always are found in a dentigerous association, usually around the mandibular third molar, and they are much less aggressive. They are not associated with basal cell nevus syndrome (orthokeratinized odontogenic cyst). However, these cysts do not demonstrate a hyperchromatic and palisaded basal cell layer, which is so characteristic of true odontogenic keratocysts.

Genetics: PTCH (patched), a tumor suppressor gene involved in both syndrome associated and sporadic KOTs, occurs on

chromosome 9q22.3 – q31. Normally, PTCH forms a receptor complex with the oncogene SMO (smoothened) for the SHH (sonic hedgehog) ligand. PTCH binding to SMO inhibits growth signal transduction. SHH binding to PTCH releases this inhibition. If normal functioning of PTCH is lost, the proliferation-stimulation effects of SMO are permitted to predominate. Evidence has shown that the pathogenesis of syndrome associated and sporadic KOTs involves a 'two hit mechanism', with allelic loss at 9q22. The 'two hit mechanism' refers to the process by which a tumor suppressor gene is inactivated. The first hit is a mutation in one allele, which, although it can be dominantly inherited, has no phenotypic effect. The second hit refers to loss of the other allele and is known as 'loss of heterozygosity' (LOH). In KOTs, this leads to the dysregulation of the oncoproteins cyclin D1 and p53. LOH in the 9q22.3–q31 region has been reported for many epithelial tumors, including basal cell carcinomas, squamous cell carcinomas, and transitional cell carcinomas; and LOH is by definition a feature of tumorigenic tissue.

Clinical and radiographic features: The largest and most detailed series of cases of odontogenic keratocyst has been published by Brannon, and his data are probably most representative of this lesion. The cyst may occur at any age, from the very young to the very elderly, although Brannon found it to be exceedingly rare under the age of 10 years. The peak incidence is in the second and third decades of life, with a gradual decline thereafter. In all series, there is a predilection for occurrence in males, ranging from 1.44:1 (Brannon), 1.46:1 (Browne) to 1.79:1 (Forssell). The mandible is invariably affected more frequently than the maxilla. In the mandible, the majority of the cysts occur in the ramus-third molar area, followed by the first and second molar area and then the anterior mandible. In the maxilla, the most common site is the third molar area followed by the cuspid region. Multiple odontogenic keratocysts occur with some frequency. Lesions found in children are often reflective of multiple odontogenic keratocysts as a component of the nevoid basal cell carcinoma syndrome. However, at other times, these multiple cysts are independent of the syndrome. There are no characteristic clinical manifestations of the keratocyst, although about 50% of the patients in Brannon's series were symptomatic prior to seeking treatment. Among the more common features are pain, soft-tissue swelling and expansion of bone, drainage and various neurologic manifestations such as paresthesia of the lip or teeth. The maxillary OKC tends to be secondarily infected with greater frequency than the mandibular ones, due to its vicinity to the maxillary sinus.



Radiographically most OKCs are unilocular, presenting a well-defined peripheral rim. Scalloping of the border is also a frequent finding and this represents variations in the growth pattern of the cyst. Multilocular radiolucent OKC is also observed, generally representing a central cavity having satellite cysts. When it is multilocular and especially if located in the third mandibular molar area, it may be confused radiographically with an ameloblastoma. Occasionally OKC may mimic a dentigerous cyst and contain the crown of a retained tooth within its lumen. The final diagnosis of any cystic cavity within the jaw bones will be achieved only after biopsy of the surgical specimen. Multilocularity (20%) is often present and tends to be seen more frequently in larger lesions. Most lesions, however, are unilocular, with as many as 40% noted adjacent to the crown of an unerupted tooth (dentigerous cyst position). Approximately 30% of maxillary and 50% of mandibular lesions produce buccal expansion. Mandibular lingual enlargement is occasionally seen. Proximity to the roots of adjacent normal teeth sometimes causes resorption of these roots, although displacement is more common. Sometimes these cysts displace the neurovascular bundle. Odontogenic keratocysts may be found in patients who range in age from infancy to old age, but about 60% of all cases are diagnosed in people between 10 and 40 years of age. There is a slight male predilection. The mandible is involved in 60% to 80% of cases, with a marked tendency to involve the posterior body and ascending ramus.

Small odontogenic keratocysts are usually asymptomatic and discovered only during the course of a radiographic examination. Larger odontogenic keratocysts may be associated with pain, swelling, or drainage. Some extremely large cysts, however, may cause no symptoms. Odontogenic keratocysts tend to grow in an antero-posterior direction within the medullary cavity of the bone without causing obvious bone expansion. This feature may be useful in differential clinical and radiographic diagnosis because dentigerous and radicular cysts of comparable size are usually associated with bony expansion. Multiple odontogenic keratocysts may be present, and such patients should be evaluated for other manifestations of the nevoid basal cell carcinoma (Gorlin) syndrome. Odontogenic keratocysts demonstrate a well defined radiolucent area with smooth and often corticated margins. Large lesions, particularly in the posterior body and ascending ramus of the mandible, may appear multilocular. An unerupted tooth is involved in the lesion in 25% to 40% of cases; in such instances, the radiographic features suggest the diagnosis of dentigerous cyst. In these cases, the cyst has presumably arisen from dental lamina rests near an unerupted tooth and has grown to envelop the unerupted tooth. Resorption of the roots of erupted teeth adjacent to odontogenic keratocysts is less common than that noted with dentigerous and radicular cysts.

The diagnosis of odontogenic keratocyst is based on the histopathologic features. The radiographic findings, although often highly suggestive, are not diagnostic. The radiographic findings in an odontogenic keratocyst may simulate those of a dentigerous cyst, a radicular cyst, a residual cyst, a lateral periodontal cyst, or the so-called globulomaxillary cyst (which is no longer considered to be a true entity). Odontogenic keratocysts of the anterior midline maxillary region can mimic nasopalatine duct cysts.

Case report

A 51 year old man came to our private practice with a complaint of painless swelling in the posterior region of left mandible which persisted for almost 2 years.

Case history

After the family and medical history were taken, a complete examination was performed, including clinical examination, Intraoral Radiographs and Panoramic Radiography. Systemic signs and symptoms, past medical history and hematologic tests were within normal limits. The radiographies from chest and skull were unremarkable and no cutaneous abnormality was revealed. No history of any trauma, difficulty in chewing food and no history of any deleterious habit like smoking, tobacco or betel nut chewing.

Intraoral findings - Clinical examinations revealed a soft expansion in the region between the lower left premolars and molars, from the vestibular side of the mandible with obliteration of left buccal vestibular region. The surface was smooth and the colour was consistent with the adjacent mucosa There were no symptoms of pain or paresthesia. On exertion of pressure, a white creamy exudate expressed out of the area between 18 and 19, but there was no tenderness or bleeding on further palpation.

Extraoral findings -Extraoral examination revealed that there was a diffuse swelling on left lower side of face measuring approximately 2 X 3.5 cm in size extended from angle of mandible posteriorly. Superiorly it extended from ala tragus line inferiorly upto lower border of mandible. The colour of swelling was same as that of surrounding skin. On palpation it was soft to firm in consistency, non tender, non-compressible, non-fluctuant and afebrile to touch. Extraorally, no presence of lymphadenophathy was noted**.** The skin color over the swelling was normal.

Radiographic and histopathological findings - In panoramic radiograph a large radiolucency with corticated border was revealed. Radiographic examination showed an extensive well circumscribed multilocular radiolucency involving the body of mandible extending from the mesial root of the first molar (19), further extending distally spanning the area of second molar (18), the angle of the mandible and ascending ramus. A lower occlusal radiograph confirmed the clinical finding of both lingual and buccal expansion of the cortical bone plates. The lower edge of the pathological formation was in the level of the inferior border of the mandible A biopsy was performed under local anaesthesia, and a histopathology report revealed the features of an odontogenic keratocyst.

Enucleation of the cystic lesions was performed under local anesthesia and tissue samples were obtained for histopathologic examination. The surgical specimens were sheet- like with cystic appearance. After processing, the tissue samples were sectioned and stained with hematoxylin and eosin (H&E).

The odontogenic keratocyst wall is thin with no evidence of superimposed inflammation. The lining epithelium is highly characteristic, and is composed of:

- A parakeratinized surface which is typically corrugated, rippled or wrinkled.
- A remarkable uniformity of thickness of the epithelium, ranging from 6 to 12 cells thick.
- A prominent palisaded, polarized basal layer of cells described as having a 'picket fence 'or ‘tombstone’ appearance.

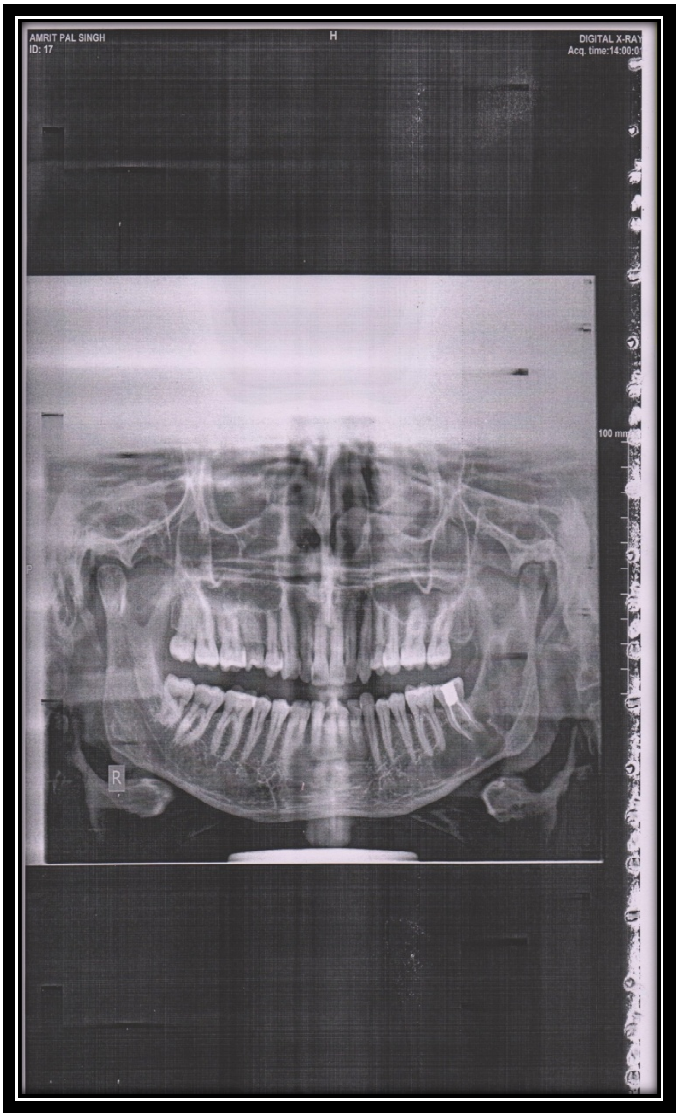


Figure 1. OPG showing Corticated Multilocular Radiolucency on Left Posterior side of the Mandible extending to Ascending Ramus of Mandible

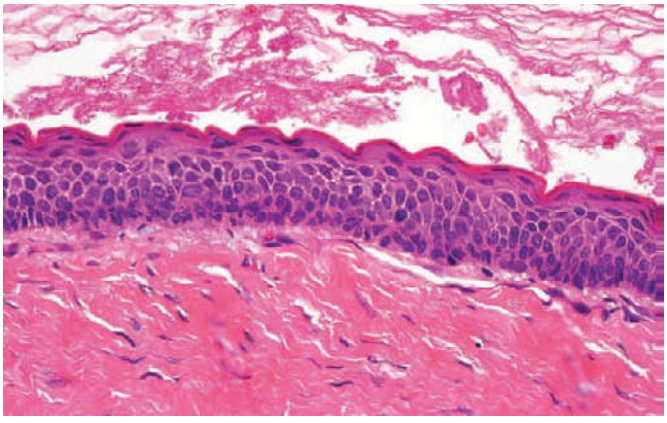


Figure 2. Lining is 6 to 12 cells thick with parakeratosis, surface corrugations, and palisading of basal cell nuclei

No rete ridges are present; therefore, the epithelium often sloughs from the connective tissue (94% of the time). The epithelium is thin, and mitotic activity is frequent; therefore, OKC grows in a neoplastic fashion and not in response to internal pressure. The connective tissue wall shows small islands of epithelium similar to the lining epithelium; some of these islands are small cysts. The apparent islands of

epithelium and small 'satellite' or 'daughter' cysts actually represent the ends of folds of the lining epithelium of the main cystic cavity which have been cut in cross-section; the linings of these cysts are very commonly folded. No evidence of Cellular Atypia and absence of any Dysplastic and neoplastic transformation of the lining epithelium in the odontogenic keratocyst.

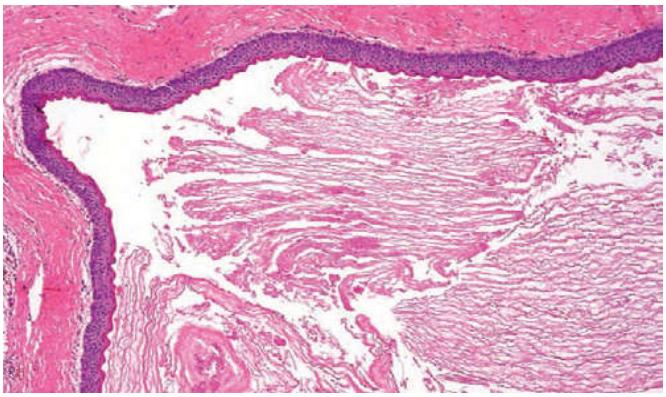


Figure 3. Cyst lined by uniformly thin epithelium with abundant keratin in lumen

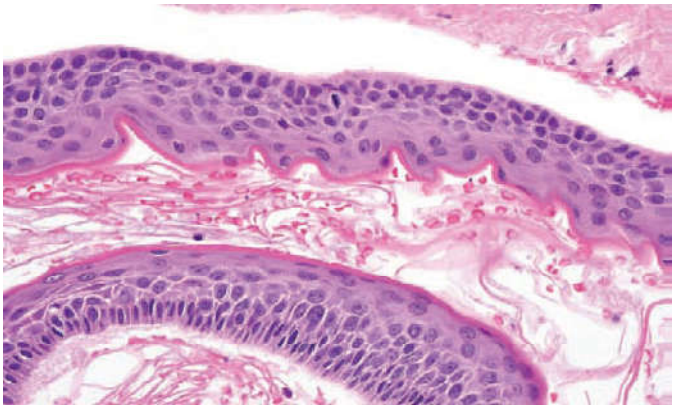


Figure 4. Typical palisading of basal cell nuclei, mild basal cell hyperplasia, and a mitotic figure

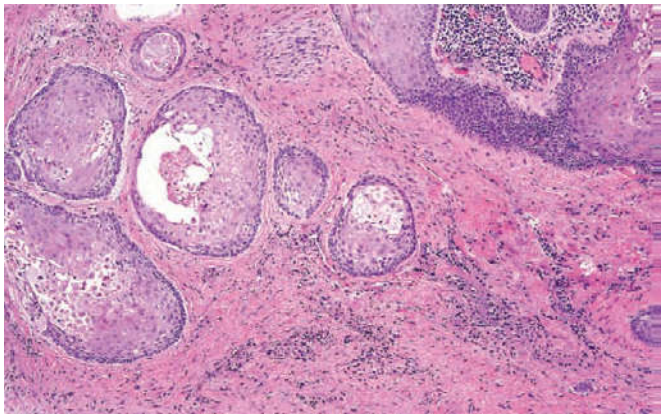


Figure 5. Satellite cysts in the wall

Cyst Contents (Aspirate)

The lumen of the keratocyst was filled with a straw colored, dirty white, viscoid suspension of keratin, thicker creamy material which has an appearance of pus, but without an offensive smell. The following are used to test the presence of keratin.

- Electrophoresis reveals low soluble protein content compared to patient's own serum, which is mostly albumin.
- Total protein is found to be below 4 g/100 ml(approx. 3.75 g/100 ml)
- Cholesterol, as well as hyaline bodies at the sites of inflammation, is present.

Treatment plan

The odontogenic keratocyst should be surgically excised. However, clinical experience has shown that complete eradication of the cyst may be difficult because the wall of the cyst is very thin and friable and may easily fragment. In addition, perforation of cortical bone, particularly in lesions involving the ramus, is common and this complicates total removal. Keratocysts enucleated in one piece recurred significantly less often than cysts enucleated in several pieces, and the recurrence rate in cases with a clinically observable infection, a fistula or with a perforated bony wall was higher with the majority occurring within five years of the surgical procedure. The size of the cyst did not seem to influence its prognosis after surgery, but those whose radiographic appearance was multilocular had a higher recurrence rate than those with a unilocular appearance. Furthermore, recurrence does not appear related to the presence of satellite cysts. On this basis, Browne concluded that recurrence of the keratocyst is due to the nature of the lesion itself, i.e. the presence of additional remnants of dental lamina from which a cyst may develop, and is not related to its method of treatment. Since recurrence may be long delayed in this lesion, follow-up of any case of odontogenic keratocyst with annual radiographs is essential for at least five years after surgery. After cystotomy and incisional biopsy, some surgeons have treated large odontogenic keratocysts by insertion of a polyethylene drainage tube to allow decompression and subsequent reduction in size of the cystic cavity. Such decompression treatment results in thickening of the cyst lining, allowing easier removal with an apparently lower recurrence rate.

Browne found no significant differences in recurrence rate following three basic methods of treating the lesions:

- Marsupialization
- Enucleation and primary closure
- Enucleation and packing open.

The patient desired a fixed prosthesis so the treatment plan was planned and followed accordingly. The treatment plan was divided into Surgical and Restorative Phases.

Surgical phase - Under all aseptic precautions in the operatory, Local anesthesia with adrenalin was given for tissue separation. Degloving incision was made 6-8 mm below the attached gingiva. Mucoperiosteal flap was raised and then small perforation was made for aspiration of cystic contents and extraction was done w.r.t. tooth 18 and 19. Then, the cystic linings was removed and chemical cauterization of the bony cavity with Carnoy's solution (100% ethanol, chloroform and glacial acetic acid in a 6:3:1 ratio with added ferric chloride) after cyst removal was done. Intraluminal injection of Carnoy's solution also has been used to free the cyst from the bony wall, thereby allowing easier removal and to destroy any residual cyst epithelium for a lower recurrence rate. The specimen was sent for histopathologic examination and a

diagnosis of OKC was made. A length of ribbon gauze was then soaked in Whitehead's varnish and packed into the bony cavity. The ribbon gauze was replaced at regular intervals until there was sufficient alveolar regeneration and soft tissue closure in the area of the lesion. Regular radiographic follow up demonstrated the almost complete regeneration of the bone in the surgical site 14 months after the initial surgery.

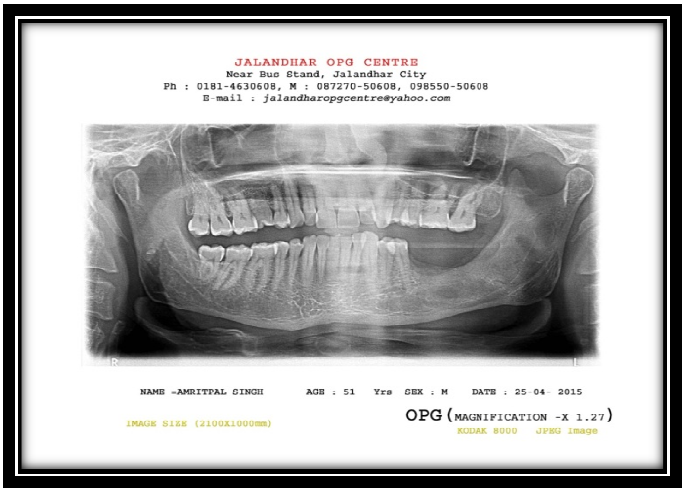


Figure 6. 7 Months post surgical OPG showing bone healing after Enucleation of OKC

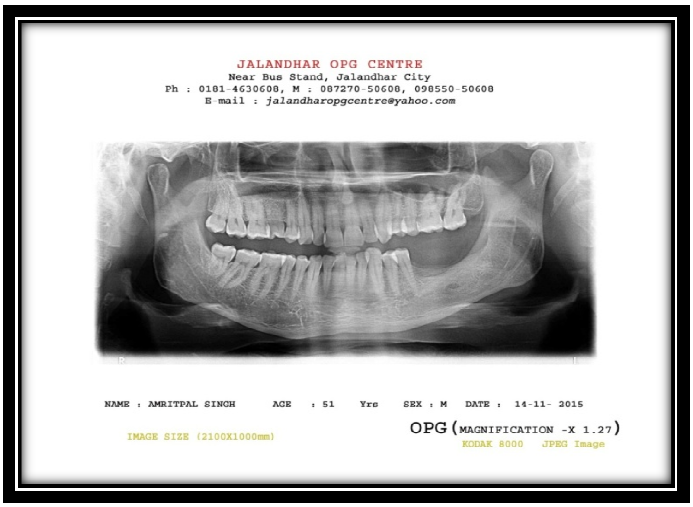


Figure 7. 14 Months post surgical OPG showing bone healing after Enucleation of OKC

Restorative phase - The options for replacement of the lost teeth included:

1. A removable partial denture
2. An implant-borne fixed bridge.

The patient did not want a removable prosthesis and, since the long span did not favour either conventional or adhesive bridgework, a decision was taken to provide an implant-borne bridge. Some 15 months after the original surgery to enucleate the cyst, two titanium implants were placed in the healed surgical site. Five months later the second-stage surgery was performed and subsequently two abutments were chosen. A metal ceramic bridge was made and fitted for a 2-month trial period to ensure comfort and cleansibility before final placement. On recall at 6 months, the patient reported satisfaction with both function and aesthetics and continued to demonstrate excellent oral hygiene. The radiographic appearance of the prosthesis at 6-month recall is shown in Figure 9.

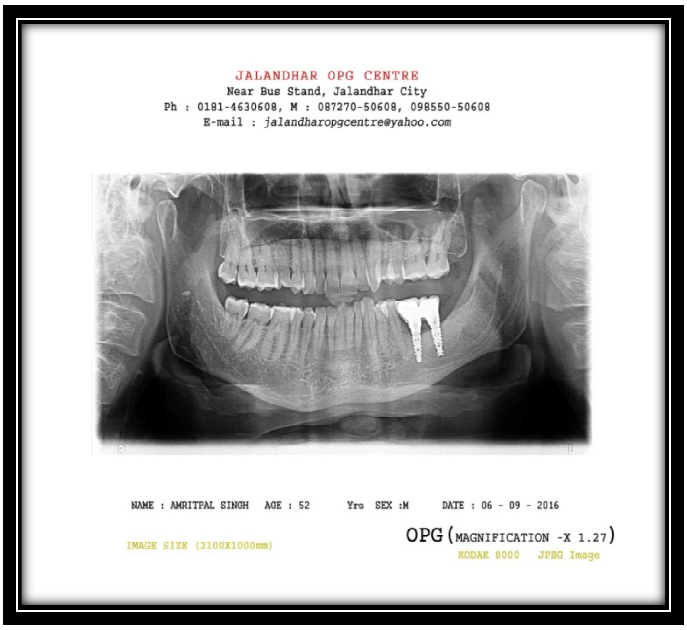


Figure 8. 23 Months post surgical OPG showing Final Restoration with Implants

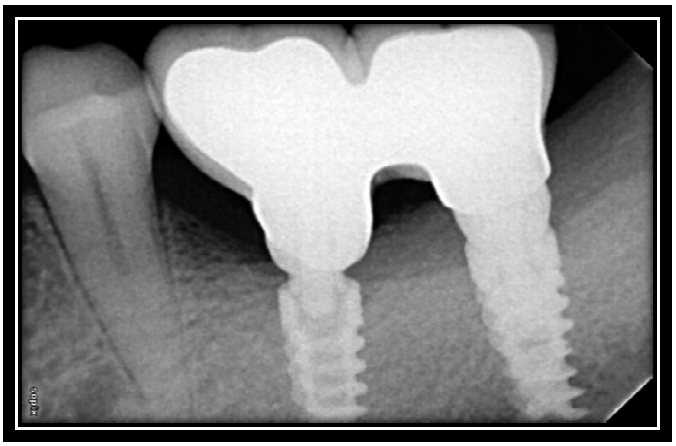


Figure 9. Radiograph showing appropriate bone level and no evidence of any complication and bone resorption after 6 months

DISCUSSION

Differential Diagnosis

- Dentigerous Cyst
- Ameloblastoma
- Primordial cyst
- Residual Cyst
- Radicular Cyst
- Traumatic Cyst
- Benign Odontogenic tumor
- Giant Cell Granuloma
- Odontogenic Myxoma

The treatment of the OKC remains controversial. Treatments are generally classified as conservative and aggressive. Conservative treatment generally includes simple enucleation, with or without curettage, using spoon curettes of

marsupialization. Aggressive treatment generally includes peripheral ostectomy, chemical curettage with carnoy's solution and resection. Some surgeons believe that the cyst can be properly treated with enucleation if the lesion is removed intact. However, complete removal of the OKC can be difficult because of the thin, friable epithelial lining, limited surgical access, skill and experience of the surgeon, cortical perforation, and the desire to preserve adjacent vital structures. The goals of treatment should involve eliminating the potential for recurrence while also minimizing the surgical morbidity. There is no consensus on adequacy of appropriate treatment of this lesion. Recurrence occurs due to the following reasons.

The first reason involves incomplete removal of the original cyst's lining. Secondly, it involves growth of a new OKC from small satellite cysts of odontogenic epithelial rests left behind by the surgical treatment. The third reason involves the development of an unrelated OKC in an adjacent region of the jaws, which is interpreted as a recurrence. Marx and stern believe that the two most common reasons for recurrence are incomplete cyst removal and new primary cyst formation. The majority of cases of recurrence occur within the first 5 years after treatment. Because of the problematic nature of these cysts, many attempts have been made to reduce the high recurrence rate by improved surgical techniques. Bramley recommends the use of radical surgery with resection and bone transplantation. Decompression or marsupialization seem to be more conservative options in the treatment of OKC. Marsupialization was first described by Partsch in 1882 for the treatment of cystic lesions. This technique is based on the externalization of the cyst through the creation of a surgical window in the buccal mucosa and in the cystic wall. Their borders are then sutured to create an open cavity that communicates with the oral cavity. This procedure relieves pressure from the cystic fluid, allowing reduction of the cystic space and facilitating bone apposition to the cystic walls. Currently, treatment involving careful and aggressive enucleation with close follow-up has been advocated for the OKC. John and James described the use of enucleation in conjunction with a chemical cauterizing agent and excision of overlying mucosa as a means of reducing recurrence.

Because the lining of the OKC is characteristically thin and friable, removal of the cysts in one piece may be difficult. Great care must therefore be taken to ensure complete removal of the cyst lining, without leaving behind remnants attached to the adjacent bone or soft tissue. The high recurrence rate associated with OKCs is a result of satellite cysts confined to the fibrous walls of the OKCs. It should be emphasized that if the fibrous capsule is completely removed, no satellite cysts will be retained to serve as a nidus for recurrence. In view of the possible recurrence of the cysts from basal cell proliferation and because of the fragility of the cyst wall and the presence of satellite cysts, the osseous walls of the defect are abraded with coarse surgical or acrylic burs to ensure that residual peripheral cystic tissue is removed. Enucleation is not always easy because the lining may be extremely thin and friable, and access in the depths of the mandible may be limited. Multilocular cysts with bony trabeculae present special problems, in as much as it is difficult to remove the lining in one piece. Enucleation with excision of the soft tissue overlying the OKCs has been proposed in an attempt to reduce the incidence of recurrence. A number of authors advocated the use of tanning with carnoy's solution (absolute alcohol, chloroform, glacial acetic acid, and ferric chloride) before enucleation of the cysts. This procedure is often followed by excision of the overlying mucosa in continuity with the lesion.

The bone showed good regenerative potential after that and was allowed to attain the size and texture close to normal where after it was successfully used anchor endosseous implants that enabled effective prosthetic rehabilitation. It is our opinion that this technique can be used in patients with large odontogenic keratocysts at either extremes of age where comorbidity of additional donor site, operative time, extraoral scar, and reliable bone available for implant placement are defining factors. Osseointegrated implants have become an extremely valuable treatment option in oral and maxillofacial prosthetic reconstruction. Advances in the understanding of the process of osseointegration and the development of placement techniques that ensure a high success rate have resulted in more applications for implants. They are particularly useful intra-orally in that they can replace the missing dentition without involving the adjacent teeth, as would be necessary in conventional fixed bridgework. The limitations of a removable prosthesis in an area of substantial tissue loss can be overcome by the provision of a fixed implant borne bridge. The aim of this case report is to demonstrate examples of the use of dental implants in situations were conventional treatment had little chance of success.

Conclusion

The recommended follow up for KCOTs is once in a year for at least 5 years. Radical operations, such as continuity resection may not be warranted always, as conservative management with Marsupialization, a little more aggressive with Enucleation with Chemical Cauterization seems to work and preserves function with least morbidity. Many authors are now advocating a more conservative approach in treating the single non syndromic odontogenic keratocyst. Recurring KCOTs will require a more radical surgery. Radical procedures such as resection should be reserved for keratocysts that involve vital structures, are recurrent or demonstrate malignant degeneration.

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