Review Article

Odontogenic Keratocyst: Developing a Protocol for Surgical Intervention

Rajesh Ashok Kshirsagar, Rajat Chandrashekhar Bhende, Pratik Hemantkumar Raut, Vrushika Mahajan, Vishal Jugalkishor Tapadiya*, Vikram Singh*

Department of Oral and Maxillofacial Surgery, Bharati Vidyapeeth Dental College and Hospital, Pune, Maharashtra, India

*These authors contributed equally to this work.

Abstract

The aim of this study was to report the outcome of a conservative treatment protocol – "enucleation and packing open" for odontogenic keratocyst (OKC). Ten patients with OKC were treated at our institute by peripheral ostectomy, enucleation followed by open packing. This conservative treatment protocol was selected because of relatively young age of the patients and relatively large size of the lesions. All the cases were monitored at regular predetermined intervals using clinical evaluation and panoramic radiographs. There was no evidence of recurrence during follow-up. The conservative treatment protocol for OKC, based on enucleation followed by open packing would be a possible choice in view of the simplicity of surgical procedure and low morbidity. This treatment modality has a low recurrence rate and may be particularly useful in young patients and patients with advanced systemic disease not amenable to major surgical intervention.

Keywords: Conservative treatment protocol, enucleation, odontogenic keratocyst

Introduction

The odontogenic keratocyst (OKC), earlier referred to as the keratocystic odontogenic tumor, is a distinctive form of the developmental odontogenic cyst. It deserves special consideration because of its peculiar histopathological features and clinical behavior. Treatment options range from a conservative approach to extensive surgeries and include marsupialization, enucleation, *en bloc*, and segmental resection.

The OKC remains a mystery. In comparison to other cysts of the jaws, OKC is unique because of its distinguishing clinical features, including potentially aggressive behavior, high recurrence rate, and an association with the nevoid basal cell carcinoma syndrome (NBCCS).[1] The etiology of OKC is probably closely related to the development of the dental lamina and its remnants after this organ has served its purpose. Various terminologies used by authors[2,3] for OKC are mentioned in Table 1.

Many attempts have been made to reduce the high recurrence rate of OKCs by modifications in the operative technique. Advocates of conservative treatment suggest that marsupialization yields results comparable to those obtained with more extensive surgery such as enucleation, *en bloc*, and segmental resection.[4] The reported frequency of recurrence in various studies ranges from 5% to 62%.[3]

In this study, we present ten ($n = 10$) cases of OKC treated by peripheral ostectomy, enucleation followed by packing open through an intraoral approach. Young patients and patients with advanced systemic disease not amenable to major surgical intervention are also included.

Materials and Methods

Case 1

A 25-year-old female was referred to the Department of Oral and Maxillofacial Surgery, BVDU, Dental College and Hospital, Pune, India, with a chief complaint of swelling and pain located at the posterior body and ascending ramus of the right hemimandible. In the clinical examination, facial asymmetry was absent. Intraorally, mild swelling was present

Address for correspondence: Dr. Rajat Chandrashekhar Bhende, Department of Oral and Maxillofacial Surgery, Bharati Vidyapeeth Dental College and Hospital, Pune, Maharashtra, India.
E-mail: docrcb@rediffmail.com

Access this article online

Quick Response Code:

Website: www.amsjournal.com

DOI: 10.4103/ams.ams_137_18

How to cite this article: Kshirsagar RA, Bhende RC, Raut PH, Mahajan V, Tapadiya VJ, Singh V. Odontogenic keratocyst: Developing a protocol for surgical intervention. Ann Maxillofac Surg 2019;9:152-7.

Table 1: Nomenclature of odontogenic keratocyst

Terminology	Author	Years
Dermoid cyst	Mikulicz	1876
Cholesteatoma	Hauer	1926
	Kostecka	1929
OKC	Philipsen	1956
Primordial cyst	Shear	1960
	Shear and Altini	1976
	Pindborg *et al*.	1971
Keratocystoma	Shear	2003
Keratinizing cystic odontogenic tumor	Reichart and Philipsen	2004
OKC	Philipsen	2005
OKC	WHO	2017

OKC: Odontogenic keratocyst

in the right mandibular third molar region. The overlying mucosa appeared normal on color and texture. There was no involvement of lymph nodes. The patient was evaluated radiographically by panoramic radiography. The imaging revealed a unilocular radiolucency extending from the mid-ramus to the right second molar [Figure 1].

Fine-needle aspiration yielded thick white "cheesy" material. Under local anesthesia, an incision extending from the mandibular ramus distal to the right third molar was carried anteriorly around the crevices of the anterior mandibular teeth till right first molar and a full thickness mucoperiosteal flap was raised. Extraction of the right third molar was done. The lateral cortical plate adjacent to the cyst was removed. The cystic cavity was curetted from the right mid-ramal area to right second molar. The resulting cavity was packed with povidone iodine gauze. The packing was replaced, during the recall visits, three times in a week for 4 months following the initial surgery. The patient was reviewed radiographically every 6 months during the follow-up period. At the end of the follow-up period, no evidence of recurrence was noticed [Figures 2 and 3].

Case 2

A 74-year-old female patient came with the complaint of a swelling extending from the right mandibular canine to the left mandibular molar region. The swelling was tender. It was first noticed by the patient approximately 7 months ago. Detailed case history revealed underlying advanced renal and cardiac disease not permitting extensive surgery under General anesthesia. Clinical examination revealed an oval and fluctuant swelling extending from the right canine to the left first molar region. It was covered by normal oral mucosa. Radiographic evaluation was performed using a panoramic view. It revealed an expansile lesion extending from the right premolar tooth to the left third molar region [Figure 4].

The cortex was expanded, but it was intact and thin. Fine-needle aspiration yielded yellowish "cheesy" material. Considering the size of lesion, it was decided to manage it conservatively. Under local anesthesia, all involved teeth were extracted. Labial ostectomy was done, and cyst was enucleated. The cystic cavity was curetted. The labial cortex was removed. Postoperatively, the cystic cavity was packed with povidone iodine gauze. It was replaced in recall visits three times in a week for 4 months following the surgery. The histopathological examination confirmed the diagnosis of OKC. The patient was called for clinical and radiological evaluation regularly after treatment. There was no evidence of residual disease on radiographic examination at follow-up [Figures 5 and 6].

Case 3

A 19-year-old male was referred to the Department of Oral and Maxillofacial Surgery, BVDU, Dental College and Hospital, Pune, India, with a chief complaint of swelling and pain from the right side of mandible. His medical history was unremarkable. According to the history obtained from the patient, pain and swelling were first noticed 9 months ago. The orthopantomograph revealed a lesion involving the entire right ramus of mandible extending to the second premolar on the right along with impacted third molar was present [Figure 7].

Extraoral examination revealed that there were a swelling and expansion at right mandibular angle region. A yellow serous liquid with cheesy content was obtained from a fine-needle aspiration material. Buccal cortical plate removed. Enucleation followed by open packing treatment was performed. After the operation, the resulting bony cavity was packed with povidone iodine gauze. It was changed in recall visits three times in a week for the 4 months. There was no sign of recurrence at a postoperative period [Figures 8 and 9].

Discussion

Epidemiology

OKCs of the jaw have been the most debated pathologic cystic lesions in the maxillofacial region.[5] There has been a great attention in the OKC since it became apparent that it may grow to a large size before it manifests clinically and that, unlike other jaw cysts, it has a particular tendency to recur following surgical treatment.[2] OKC may be found in population, which range in age from infancy to old age.[3] There is a slight male predilection.[3,6] Literature review suggests, mandible is involved in 60%–80% of cases, with a noticeable tendency to involve the posterior body and ascending ramus of mandible.[3,6]

Clinical features

Patients with OKCs may complain of pain, swelling or discharge. Rarely, they experience paraesthesia of the lower lip. Some are oblivious about the lesions until they attain large size or develop pathological fractures. Some OKCs may be identified unexpectedly during radiographic examination. In many cases, patients are astonishingly free of symptoms until the cysts reach a large size.[2]

Similar to other intraosseous jaw lesions, the cyst may lead to displacement of the teeth. The occurrence of large OKC involving the maxillary sinus and causing destruction of the

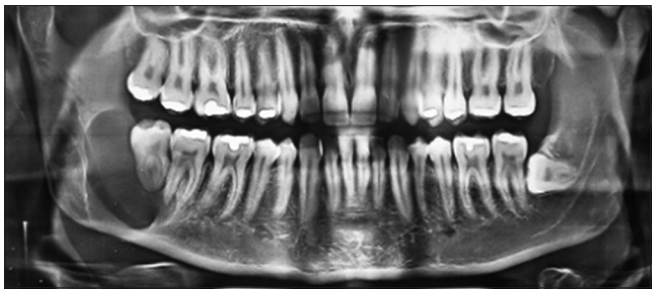

Figure 1: Unilocular radiolucency extending from the mid-ramus to the right second molar

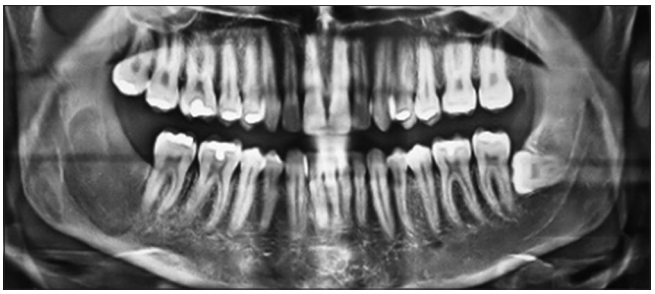

Figure 2: After 6 months

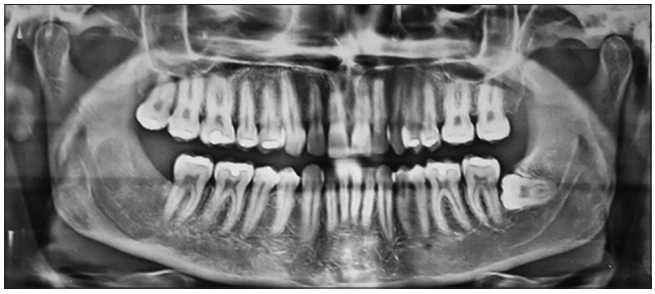

Figure 3: After 1 year

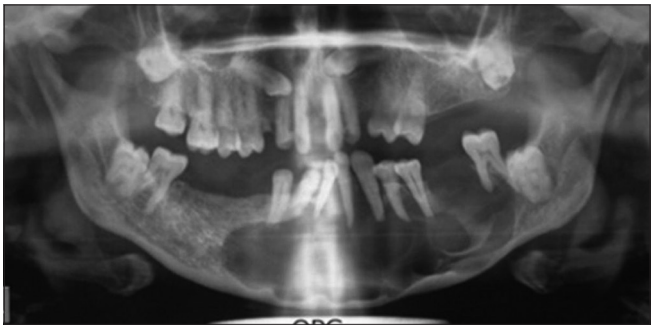

Figure 4: Expansile lesion extending from the right premolar tooth to the left third molar region

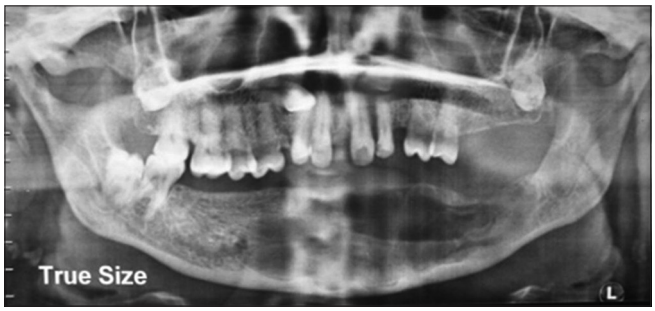

Figure 5: There was no evidence of residual disease after 6 months

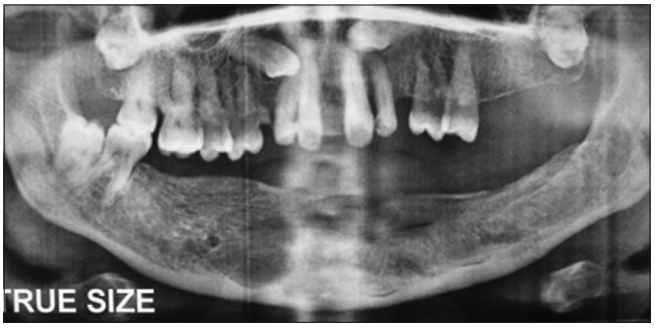

Figure 6: No evidence of recurrence of disease after 1 year

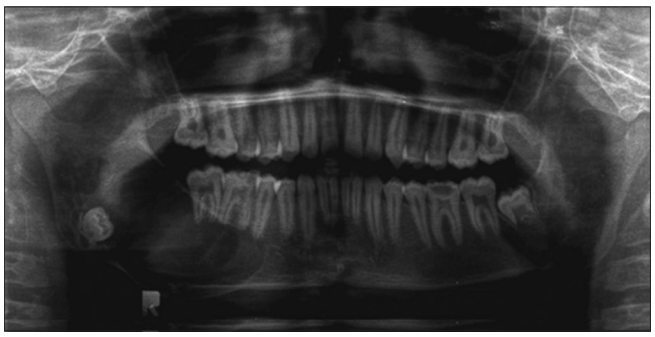

Figure 7: A lesion involving the entire right ramus of mandible extending to the second premolar on the right along with impacted third molar was present

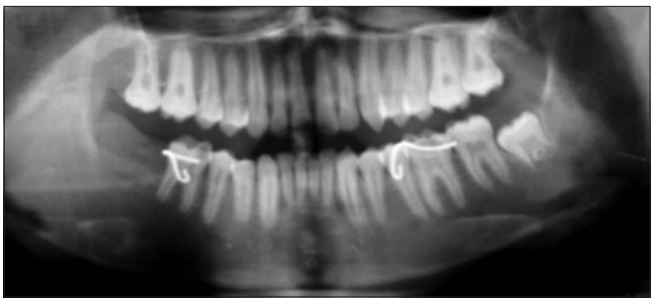

Figure 8: No sign of recurrence at a postoperative period after 6 months

floor of the orbit, proptosis of the eyeballs has been described by Lund.[7] Penetration of cortical bone and involved surrounding soft tissues due to the aggressive behavior of OKC is documented by Emerson *et al*.[8] Jackson *et al*. reported that OKC extended from the maxilla and eventually involved the base of the skull, "behaving rather like a low-grade squamous cell carcinoma."[9]

Nevoid basal cell carcinoma syndrome

Multiple OKCs occur in 75% of the patients involved with the NBCCS. There are some differences between the cysts in patients with NBCCS and those with isolated keratocyst. In most of the cases, initially, only a solitary cyst is present, but additional cysts may develop over periods ranging from 1 to 20 years. In NBCCS, cysts are frequently associated with the

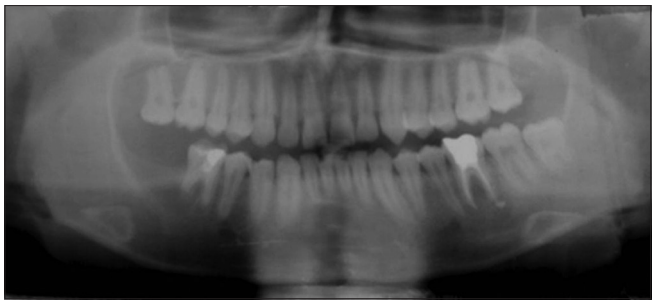

Figure 9: Satisfactory healing of lesion after 1 year

crowns of unerupted teeth; on radiographs, they may resemble dentigerous cysts.[3]

Genetic alterations

The neoplastic concept of OKC is aided by molecular studies that verified loss of heterozygosity.[10-12] These studies instituted evidence of allelomorphic loss mainly in the p16, p53, PTCH, MCC, TSLC1, LTAS2, and FHIT genes.[13]

P53, a tumor-suppressor gene is frequently mutated in various malignant neoplasms.[14] Positive p53 labeling in epithelial cells of the OKC has been shown by a number of studies.[15-18] Although it was not followed by p53 gene alteration using stranded conformation polymorphism (polymerase chain reaction–single-strand conformation polymorphism).[17] It suggests that p53 mutation is not a significant event to OKC pathogenesis.[13]

Mutations of PTCH1in OKCs associated with NBCCS were first described by Lench and colleagues.[19] Pan and Li demonstrated that Ki-67 labeling index in the epithelium of OKCs with PTCH1 mutation was significantly higher than in cases with no PTCH1 mutation. An additional study showed that Gli1, a downstream signaling molecule of the SHH/PTCH pathway, is overexpressed in OKC.[20]

Epigenetic alterations are considered important events in the tumorogenesis of benign and malignant tumors of the head and neck.[21] The Drosophila patched gene (PTCH1) methylation has been suggested as an alternative to mutational causes of the PTCH pathway deregulation in tumors associated with NBCCS syndrome.[22,23] OKC has presented methylation of the P21 gene.[24]

It is suggested that, residues of OKC lining which are left behind at operation, the apparently high metabolic activity of the epithelium, as demonstrated by the high activity of oxidative enzymes representing glycolytic, citric acid, and pentose phosphate shunt mechanisms, were likely to be factors of importance to consider among reasons for the high incidence of OKC recurrences.[25]

Within 2 days of explanting the OKCs, there was the growth of epithelial cells and fibroblast-like cells, which showed moderate-to-high activity of NADH-diaphorase and acid phosphatase.[26]

Biological markers

Early studies showed that fluids from keratinizing cysts have lower soluble protein levels than nonkeratinizing cysts and it was suggested that a protein level of <4.0 g/100 mL would indicate a diagnosis of OKC.

In the fluid of OKCs, an antigen was localized to the epithelial cell which was not present in the fluids neither of other cyst types nor in plasma or saliva. They called it keratocyst antigen (KCA).[27,28] A later study suggested that the keratin might become soluble in the cyst fluid by proteolysis. This relationship of keratin and KCA would enable the use of commercially available antikeratin antibodies in the preoperative diagnosis of the OKC.[29]

A major antigen, lactoferrin was identified in the aspirated fluid of all OKCs which was apparently of epithelial origin, but not a keratin. It is a secretory substance present in the azurophilic granules of polymorphonuclear leukocytes and body secretions but not in serum.[30] OKC fluids contained significantly higher concentrations of lactoferrin than fluids from the other cysts, but with a wide range of values for each group and this could therefore not be regarded as an absolute marker for OKCs.[31]

Radiographic assessment

The manifestation of large or multilocular lesions on the panoramic radiograph requires a further radiographic evaluation. The hard and soft-tissue involvement should be assessed by computed tomography scans. The radiographic picture of OKC is unilocular or multilocular radiolucency with scalloped and well-defined margins.[32] It is not easy to differentiate radiographically between OKCs and other cysts of the jaws.[33] The differential diagnosis of OKC may be benign odontogenic or nonodontogenic neoplasms lacking calcification, other odontogenic cysts, and intraosseous vascular lesions.[32-35] Unerupted tooth is associated with the lesion (25%–40%). Resorption of the roots of erupted adjacent teeth to OKC is less common than dentigerous and radicular cysts.[3]

Diagnosis

The diagnosis of OKC is primarily based on the histopathological features. It typically shows a thin, friable wall, which is often difficult to enucleate from the bone in one piece. The cystic lumen may consist of a clear liquid, similar to a transudate of serum; or a cheesy material. The thin fibrous wall is essentially lacking of inflammatory infiltrate. Perhaps small satellite cysts, cords, or islands of odontogenic epithelium are seen within the fibrous wall.[3]

The radiographic findings in OKC may replicate a dentigerous cyst, a radicular cyst, a residual cyst, a lateral periodontal cyst. OKC of the anterior midline maxillary region can impersonate nasopalatine duct cysts. Peripheral OKC within the gingival soft tissues has been reported rarely.[3]

Treatment modalities

The treatment should aim at the elimination of possible vital cells left behind in the defect from the original lining or derived from microcysts in the wall. The choice of treatment approach should be based on the size of the cyst, recurrence status, and radiographic evidence of cortical perforation.[4] Stoelinga

Table 2: Treatment protocol for odontogenic keratocyst

Radiographic feature	Age	Recurrence			
		First surgical intervention		Second surgical intervention	
		Involving inferior border of mandible	Cortical/soft-tissue perforation	Involving inferior border of mandible	Cortical/soft-tissue perforation
Unilocular	Young	Marsupialization	Enucleation with Carnoy's solution	Enucleation and packing open	*En bloc* resection with reconstruction
	Adult and geriatric patients	Marsupialization	Enucleation with Carnoy's solution/Cryosurgery	Enucleation and packing open	*En bloc* resection
Multilocular	Young	Marsupialization	Enucleation with Carnoy's solution	Enucleation and packing open	*En bloc* resection with reconstruction
	Old	Marsupialization	Enucleation with Carnoy's solution/cryosurgery	Enucleation and packing open	En bloc resection

advocated the use of cauterizing agent such as Carnoy's solution. In case of multilocular cyst, one should eliminate the bony septae as to assure proper treatment of the resulted cavity with Carnoy's solution. If the cyst has penetrated through the lingual or buccal cortex, electrocauterization should be carried out to avoid a recurrence in the soft tissues. Elimination of the epithelial islands and microcysts located in the overlying, attached mucosa should be assured by excising the part of the mucosa. Preferably, the cyst with the attached overlying mucosa should be removed in 1 piece.[36]

Yildirim *et al*. advocated a conservative treatment protocol for OKC. He suggested enucleation followed by open packing. The resulting cavity was irrigated with mixture of normal saline and chlorhexidine gluconate. The resulting cystic cavity was packed with iodoform gauze impregnated with bacitracin ointment to minimize the risk of recurrence in each recall visits. The benefit of this protocol lies in the minimal surgical morbidity, decreased incidence of damage to associated structures such as the inferior alveolar nerve and developing teeth.[37]

Recurrence

OKC has a particular tendency to recur after surgical treatment. The first to point out this peculiarly aggressive behavior were Pindborg and Hansen (1963). Recurrence is encountered more often in mandibular OKC, particularly those in the posterior body and ascending ramus. Multiple recurrences are not unusual. Although many OKCs recur within 5 years of the original surgery, a significant number of recurrences may not be manifested until 10 or more years after the original surgical procedure. Long-term clinical and radiographic follow-up, therefore, is necessary.[3] Yagyuu *et al*. found in their study that the mean length of recurrent OKC lesions (62.8 ± 6.5 mm) was greater than that of nonrecurrent lesions (43.0 ± 4.0 mm) ($P = 0.0363$).[38]

It is suggested that there may be an inherent tendency to develop such cysts; any remnants of dental lamina may form the target for new OKC formation such as patients with the NBCCS.[39] OKCs may arise from proliferations of the basal cells of the oral mucosa, predominantly in the third molar region and ascending ramus of the mandible.[2]

Follow-up

The literature suggests that most recurrences will present the first 5 years after primary treatment. The recommended follow-up for OKCs is once a year the first 5 years postoperatively. As the recurrences or newly developed OKCs may also present late clinically, a follow-up every 2 years thereafter seems a reasonable policy. Pogrel recommended follow-up, primarily with panorama type radiographs, every 6 months for 2 years, every year for 5 years, and every 2 years for 10 years in asymptomatic patients.[40]

In our opinion, "enucleation and packing open" is an excellent treatment option for OKC fulfilling the above requirement. Major surgical intervention such as resection and reconstruction should be limited to cases with recurrence [Table 2]. Therefore, the patients should have radiographic and clinical examinations at regular intervals.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Jordan RC. Histology and ultrastructural features of the odontogenic keratocyst. Oral Maxillofac Surg Clin North Am 2003;15:325-33.
2. Shear M, Speight PM. Odontogenic Keratocyst. In: Cysts of the Oral and Maxillofacial Regions 4th ed. Pune, Maharashtra: Bharati Vidyapeeth Dental College; 2007. p. 6-58.
3. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial Pathology. 3rd ed. Pune: Saunders Elsevier; 2018. p. 683-7.
4. Forssell K, Forssell H, Kahnberg KE. Recurrence of keratocysts. A long-term follow-up study. Int J Oral Maxillofac Surg 1988;17:25-8.
5. Zhou H, Hou R, Ma Q, Wu K, Ding Y, Qin R, *et al.* Secondary healing after removal of large keratocystic odontogenic tumor in the mandible:

Enucleation followed by open packing of iodoform gauze. J Oral Maxillofac Surg 2012;70:1523-30.
6. el-Hajj G, Anneroth G. Odontogenic keratocysts – A retrospective clinical and histologic study. Int J Oral Maxillofac Surg 1996;25:124-9.
7. Lund JV. Odontogenic keratocyst of the maxilla: A case report. Br J Oral and Maxillofac Surg 1985:23:210-215.
8. Emerson TG, Whitlock RI, Jones JH. Involvement of soft tissue by odontogenic keratocysts (primordial cysts). Br J Oral Surg 1972;9:181-5.
9. Jackson IT, Potparic Z, Fasching M, Schievink WI, Tidstrom K, Hussain K. Penetration of the skull base by dissecting keratocyst. J Craniomaxillofac Surg 1993;21:319-25.
10. Agaram NP, Collins BM, Barnes L, Lomago D, Aldeeb D, Swalsky P, *et al.* Molecular analysis to demonstrate that odontogenic keratocysts are neoplastic. Arch Pathol Lab Med 2004;128:313-7.
11. Henley J, Summerlin DJ, Tomich C, Zhang S, Cheng L. Molecular evidence supporting the neoplastic nature of odontogenic keratocyst: A laser capture microdissection study of 15 cases. Histopathology 2005;47:582-6.
12. Malcić A, Jukić S, Anić I, Pavelić B, Kapitanović S, Kruslin B, *et al.* Alterations of FHIT and P53 genes in keratocystic odontogenic tumor, dentigerous and radicular cyst. J Oral Pathol Med 2008;37:294-301.
13. Gomes CC, Diniz MG, Gomez RS. Review of the molecular pathogenesis of the odontogenic keratocyst. Oral Oncol 2009;45:1011-4.
14. Partridge M, Costea DE, Huang X. The changing face of p53 in head and neck cancer. Int J Oral Maxillofac Surg 2007;36:1123-38.
15. Lo Muzio L, Staibano S, Pannone G, Bucci P, Nocini PF, Bucci E. Expression of cell cycle and apoptosis-related proteins in sporadic odontogenic keratocysts and odontogenic keratocysts associated with the nevoid basal cell carcinoma syndrome. J Dent Res 1999;78:1345-53.
16. Lombardi T, Odell EW, Morgan PR. P53 immunohistochemistry of odontogenic keratocysts in relation to recurrence, basal-cell budding and basal-cell naevus syndrome. Arch Oral Biol 1995;40:1081-4.
17. Li TJ, Browne RM, Prime SS, Paterson IC, Matthews JB. P53 expression in odontogenic keratocyst epithelium. J Oral Pathol Med 1996;25:249-55.
18. Clark P, Marker P, Bastian HL, Krogdahl A. Expression of p53, ki-67, and EGFR in odontogenic keratocysts before and after decompression. J Oral Pathol Med 2006;35:568-72.
19. Lench NJ, Telford EA, High AS, Markham AF, Wicking C, Wainwright BJ. Characterisation of human patched germ line mutations in naevoid basal cell carcinoma syndrome. Hum Genet 1997;100:497-502.
20. Sun LS, Li XF, Li TJ. PTCH1 and SMO gene alterations in keratocystic odontogenic tumors. J Dent Res 2008;87:575-9.
21. Weber A, Wittekind C, Tannapfel A. Genetic and epigenetic alterations of 9p21 gene products in benign and malignant tumors of the head and neck. Pathol Res Pract 2003;199:391-7.
22. Cretnik M, Musani V, Oreskovic S, Leovic D, Levanat S. The patched gene is epigenetically regulated in ovarian dermoids and fibromas, but not in basocellular carcinomas. Int J Mol Med 2007;19:875-83.
23. Pritchard JI, Olson JM. Methylation of PTCH1, the patched-1 gene, in a panel of primary medulloblastomas. Cancer Genet Cytogenet 2008;180:47-50.
24. Moreira PR, Guimarães MM, Guimarães AL, Diniz MG, Gomes CC, Brito JA, *et al.* Methylation of P16, P21, P27, RB1 and P53 genes in odontogenic keratocysts. J Oral Pathol Med 2009;38:99-103.
25. Magnusson BC. Odontogenic keratocysts: A clinical and histological study with special reference to enzyme histochemistry. J Oral Pathol 1978;7:8-18.
26. Stenman G, Magnusson B, Lennartsson B, Juberg-Ode M. *In vitro* growth characteristics of human odontogenic keratocysts and dentigerous cysts. J Oral Pathol 1986;15:143-5.
27. Yli-Paavalniemi P, Tuompo H, Calonius PE. Proteins and inflammation in jaw cysts. Proc Finn Dent Soc 1976;72:191-5.
28. Kuusela P, Hormia M, Tuompo H, Ylipaavalniemi P. Demonstration and partial characterization of a novel soluble antigen present in keratocysts. Oncodev Biol Med 1982;3:283-90.
29. Kuusela P, Ylipaavalniemi P, Thesleff I. The relationship between the keratocyst antigen (KCA) and keratin. J Oral Pathol 1986;15:287-91.
30. Douglas CW, Craig GT. Evidence for the presence of lactoferrin in odontogenic keratocyst fluids. J Clin Pathol 1987;40:914-21.
31. Douglas CW, Craig GT. Quantitation of lactoferrin in odontogenic cyst fluids. J Clin Pathol 1989;42:180-3.
32. Giuliani M, Grossi GB, Lajolo C, Bisceglia M, Herb KE. Conservative management of a large odontogenic keratocyst: Report of a case and review of the literature. J Oral Maxillofac Surg 2006;64:308-16.
33. Myoung H, Hong SP, Hong SD, Lee JI, Lim CY, Choung PH. Odontogenic keratocyst: Review of 256 cases for recurrence and clinicopathologic parameters. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;91:328-33.
34. de Avila ED, de Molon RS, Massucato EM, Hochuli-Vieira E. Relationship between the prevalence of the dentigerous cyst and the odontogenic keratocyst tumor and the current etiologic hypothesis. J Craniofac Surg 2009;20:2036-40.
35. Meara JG, Shah S, Li KK, Cunningham MJ. The odontogenic keratocyst: A 20-year clinicopathologic review. Laryngoscope 1998;108:280-3.
36. Stoelinga PJ. The treatment of odontogenic keratocysts by excision of the overlying, attached mucosa, enucleation, and treatment of the bony defect with carnoy solution. J Oral Maxillofac Surg 2005;63:1662-6.
37. Yildirim G, Ataoglu H, Kalayci A, Ozkan BT, Kucuk K, Esen A. Conservative treatment protocol for keratocystic odontogenic tumour: A follow-up study of 3 cases. J Oral Maxillofac Res 2010;1:e7.
38. Yagyuu T, Kirita T, Sasahira T, Moriwaka Y, Yamamoto K, Kuniyasu H. Recurrence of keratocystic odontogenic tumor: Clinicopathological features and immunohistochemical study of the hedgehog signaling pathway. Pathobiology 2008;75:171-6.
39. Soskolne WA, Shear M. Observations on the pathogenesis of primordial cysts. Br Dent J 1967;123:321-6.
40. Pogrel MA. The keratocystic odontogenic tumor. Oral Maxillofac Surg Clin North Am 2013;25:21-30, v.