Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology 2022;8(2):55–60

Content available at: https://www.ipinnovative.com/open-access-journals

Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology

Journal homepage: www.joooo.org

Review Article

OKC- an update on etiopathogenesis, clinical & radiological features

Shalini Sharma[1,*], Nagaraju Kamarthi[1], Sangeeta Malik [2], Sumit Goel[1], Swati Gupta[1], Abhinav Sharma[2]

[1]*Dept. of Oral Medicine and Radiology, Subharti Dental College and Hospital, Swami Vivekanand Subharti University,, Meerut, Uttar Pradesh, India*
[2]*Subharti Dental College & Hospital, Meerut, Uttar Pradesh, India*

ARTICLE INFO

Article history:
Received 02-06-2022
Accepted 22-06-2022
Available online 18-06-2022

Keywords:
Odontogenic keratocyst
Keratocyst odontogenic tumor
Update
Etiopathogenesis
Clinical features
Imaging
Treatment

ABSTRACT

Odontogenic keratocyst (OKCs), first described by Philipsen in 1956, is characterized by an aggressive behavior with a relatively high recurrence rate. Its complicated behavior creates confusion for both clinicians and pathologists. Patients with OKC are often asymptomatic but may present with pain, swelling, or discharge. The lesion may occur sporadically or associated with nevoid basal cell carcinoma (NBCCS). Proper imaging modality and histopathological investigations are required for the diagnosis and management of OKCs. The purpose of this article is to provide an overview of many features of OKC, with a focus on etiopathogenesis, clinical symptoms, imaging and histological aspects, and various treatment methods, as well as recurrence rate and prognosis.

1. Introduction

Cysts are abnormal, closed sac-like formations that contain a liquid, gaseous, or semisolid substance and are surrounded by tissue. Based on odontogenesis, oral cysts are separated into two categories: odontogenic cysts (OCs) and non-odontogenic cysts (NOCs) (non-OCs).[1] The most common lesions found in the jaws are odontogenic cysts. "Cavities filled with liquid, semiliquid, or gaseous fluid with odontogenic epithelial lining and connective tissue on the outside," according to the description.[2] They come from the epithelial component of the odontogenic apparatus, or fragments of it, which are implanted in the bone or the gingival tissues around it.[2] Oral pathological lesions are dominated by odontogenic cysts.[3]

OCs are best characterized as inflammatory or developmental based on their origin and etiology.[3] Inflammatory cysts form as a result of inflammation; however, the triggering elements that cause developmental cysts to form are unknown.[3]

Over 50 years ago, Philipsen (1956) coined the name "odontogenic keratocyst" (OKC) to characterize a category of odontogenic cysts with a distinctive histological appearance.[4] It makes up around 11% of jaw cysts.[5] (Figure 1)

In the World Health Organization (WHO) classifications of 1971 and 1992, the term "keratocyst" was used to designate any cyst with keratinization, and it was suggested as the official terminology for a specific form of an odontogenic cyst. OKC was classified into two histological types: parakeratinized and orthokeratinized.[6] Orthokeratinized variation was detected in 12% of instances while the parakeratinized form was seen in 90% of cases.[6]

* *Corresponding author.*
E-mail address: dr.shalinisharma23@gmail.com (S. Sharma).

https://doi.org/10.18231/j.joooo.2022.012

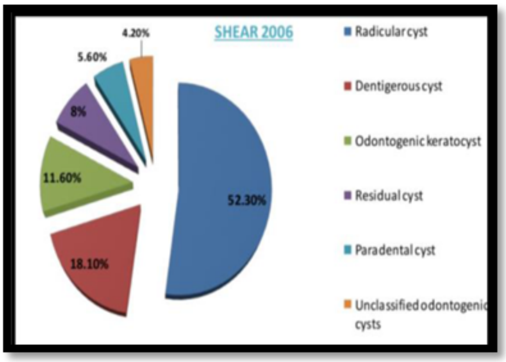

Fig. 1: Frequency ofodontogenic cyst in the oral region

1.1. *Classification of OKC*

The classification of odontogenic keratocysts (OKCs) is still a topic that many dental researchers discuss. While the first two WHO classifications of odontogenic lesions (Pindborg et al., 1971; Kramer et al., 1992) classified OKCs as developmental odontogenic cysts, the 2005 edition of the World Health Organization Classification of Head and Neck Tumours reclassified OKCs as neoplastic lesions under the name "keratocystic odontogenic tumors" (KCOTs).[7] Because KCOTs are locally aggressive jaw cystic lesions with theoretical growth potential and a proclivity for recurrence, it was classified as a benign neoplasm rather than a cyst by the WHO in 2005.[8,9] (Chart 1)

MALIGNANT TUMOURS	
	Odotnogenic epithelium with odontogenic ectomesenchyme, with or without hard tissue formation
Odontogenic carcinomas	
Metastasizing (malignant) ameloblastoma	Ameloblastomic fibroma
Ameloblastic carcinoma - primary tumour	Ameloblastic fibrodentinoma
Ameloblastic carcinoma - secondary tumour (dedifferentiated)	Ameloblastic fibro-odontoma
	Odontoma
Intraossoeus	- odontoma, complex type
Ameloblastic carcinoma - secondary type (dedifferentiated)	- odontoma, composite type
	Odontoameloblastoma
Peripheral	Calcifying cystic odontogenic tumour
Primary intraosseous squamous cell carcinoma - solid type	Dentinogenic ghost cell tumour
Primary intraosseous squamous cell carcinoma derived from keratocystic odontogenic tumour	
Primary intraosseous squamous cell carcinoma derived from odontogenic cysts	**Mesenchyme and/ odontogenic ectomesenchyme with or without odontogenic epithelium**
Clear cell odontogenic carcinoma	
Dhost cell odontogenic carcinoma	Odontogenic fibroma
	Odontogenic myxoma/ myxofibroma
Odontogenic sarcomas	Cementoblastoma
Ameloblastoma fibrosarcoma	
Ameloblastoma fibrodentino - fribro-odontosarcoma	**Bone-related lesions**
	Ossifying fibroma
BENIGN TUMOURS	Fibrous dysplasia
Odontogenic epithelium with mature fibrous stroma without odontogenic ectomesenchyme	osseous dysplasia
Ameloblastoma	Central giant cell granuloma
Ameloblastoma, unicystic type	Cherubism
Ameloblastoma, extraneous/ peripheral type	Aneurysmal bone cyst
Metastatisizing amelobalstoma	Simple bone cyst
Squammous odotnogenic tumour	
Calcifying epithelial odontogenic tumour	**Other tumours-**
Adenomatoid odontogenic tumour	Melanotic neuroectodermal tumour of infancy
Odontogenic keratocyst	

Chart 1: 2005 WHO classification of odontogenic tumors & cysts

OKC was recategorized by the World Health Organization in 2017 as an odontogenic cyst of developmental origin, with orthokeratinized cyst being classed separately. (Chart 2)

Table I: 2017 WHO classification of odontogenic tumors and cysts

Malignant Odontogenic Tumors
Ameloblastic carcinoma
Primary intraosseous carcinoma, NOS
Sclerosing odontogenic carcinoma
Clear cell odontogenic carcinoma
Ghost cell odontogenic carcinoma
Odontogenic carcinosarcoma
Odontogenic sarcomas
Benign Odontogenic Tumors
Epithelial Origin
Ameloblastoma, conventional
Ameloblastoma, unicystic type
Ameloblastoma, extraosseous/ peripheral type
Metastasizing (malignant) ameloblastoma
Squamous odontogenic tumor
Calcifying epithelial odontogenic tumor
Adenomatoid odontogenic tumor
Mixed (Epithelial-Mesenchymal) Origin
Ameloblastic fibroma
Primordial odontogenic tumor
Odontoma
Compound type
Complex type
Dentinogenic ghost cell tumor
Mesenchymal Origin
Odontogenic fibroma
Odontogenic myxoma/myxofibroma
Cementoblastoma
Cemento-ossifying fibroma
Odontogenic Cysts
Developmental Origin
Dentigerous cyst
Odontogenic keratocyst
Lateral periodontal and botryoid odontogenic cyst
Gingival cyst
Glandular odontogenic cyst
Calcifying odontogenic cyst
Orthokeratinized odontogenic cyst
Inflammatory Origin
Radicular cyst
Collateral inflammatory cyst

Chart 2: 2017- WHO classification of odontogenic tumors & cysts

2. Etiopathogenesis

The formation of the dental lamina, and particularly remnants of it after this organ has served its job, is likely to be linked to the etiology of OKC.[10] Due to the improbable probability of remains or offshoots of this dental lamina being situated in the mucosa posterior to the final molar, the common presence of OKC posterior to the 3rd molar region is difficult to describe if dental lamina is supposed to be the etiological derivation.[11] This may also arise from basal cells of overlying epithelium. The tendency for this illness to proliferate along the cancellous channels with relatively little cortical expansion is one of its distinguishing traits. To explain this, several theories of OKC expansion have been offered. Intraluminal hyperosmolality, active epithelial proliferation,[12] cyst wall collagenolytic activity,[13] and keratinocyte production of interleukin 1 and 6 are among them.

Autophagy, a lysosome-dependent catabolic process, plays a critical role in tumor growth regulation by degrading cellular proteins and organelles. Autophagy is a noteworthy finding in KCOTs, as it is induced during tumor formation and plays a crucial role in anti-apoptosis and tumor cell proliferation.[14]

2.1. *GENETICS Ptch gene*

Sonic Hedgehog (SHH), bone morphogenetic protein (BMP), Wnt, HGF, and FGF, as well as tumor suppressor genes functioning as cell growth regulators, influence the morphogenesis and cytodifferentiation of teeth. Tumor formation occurs when these genes are inactivated by mutations and/or loss of heterozygosity (LOH). 15 PTCH ("patched") is a tumor suppressor gene found on chromosome 9q22.3-q31.36–40 in both NBCCS and sporadic KCOTs. The binding of PTCH to SMO prevents the transmission of growth signals. This inhibition is released when SHH binds to PTCH. If PTCH's usual function is gone, SMO's proliferation-stimulating effects are permitted to take over. Atypical activation of the SHH signaling system during adulthood has been linked to the development of tumors.

Immunohistochemical examination of PTCH, SHH, and SMO expression patterns in sporadic KCOTs revealed that SMO expression is linked to KCOT recurrence.[15]

2.2. *Growth factors*

2.2.1. *Ki-67, PCNA, and p53*

Various studies of the proliferative activity of the lining epithelium of KCOTs have focused on the expression of p53, proliferating cell nuclear antigen (PCNA), and Ki67.[15]

2.2.2. *Clonality analysis*

A neoplasm's origin from a single clone of genetically identical cells is a distinguishing feature. Gomes et al. (2009) investigated the clonal genesis of 19 odontogenic tumors, including 6 OKC patients.[15] Twelve of the 16 instances with information indicated a monoclonal pattern. The remaining two OKCs were polyclonal, while four of the six were monoclonal. While the authors stressed that most odontogenic tumors, including OKCs, are clonal, they did attribute the four polyclonal occurrences to stromal or inflammatory cell contamination of samples. A substantial scattered inflammatory cell component was found in both polyclonal OKCs.

2.2.3. *Apoptotic mechanisms*

Recent research has found that bcl-2 positive cells are mostly found towards the base of the lining epithelium, supporting the idea that apoptosis does not occur in these cells. TUNEL-positive cells have only been seen at the surface layer of KCOTs, indicating that they have experienced severe apoptosis. Thus, bcl-2 prevents apoptosis in the basal and supra-basal layers to enhance cellular proliferation, whereas apoptosis maintains the thickness of the lining epithelium and allows the development of vast amounts of keratin in the surface layer of KCOTs. Given that in this type of lesion, there is a regulated equilibrium between cell proliferation, cell differentiation, and cell death, this could explain why KCOTs, despite having a neoplastic nature and a high proliferative capacity, do not form tumor masses.[15]

3. Incidence, Clinical Presentation, and Natural History

OKCs account for around 10% of all odontogenic cysts, with a wide age range (from 8 to 82 years) and a peak in the third decade of life.[16] Males have a minor majority in most series. OKCs are found in tooth-bearing areas. In the mandible, they occur twice as frequently as in the maxilla.[17] The posterior sextant, the angle, or the ramus are the most common sites where OKCs originate from the jaw.[9,18] The most typical locations of origin in the maxilla are the anterior sextant, primarily between the canine and lateral incisors, and the third molar region.[19,20] (Figure 2)

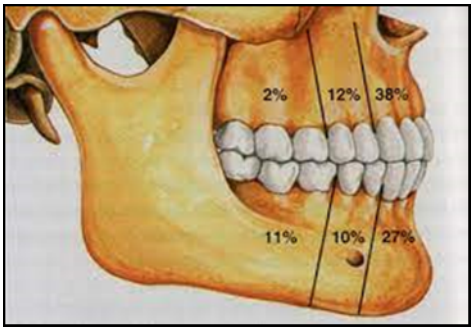

Fig. 2: Relative distribution of OKC in the jaws

In most situations, despite their aggressive activity, OKCs induce modest bone extension due to their propensity to expand through the intramedullary region, "increasing the length of the bone."[21] Asymptomatic patients may have large lesions that cause significant erosion of cortical plates and involvement of adjacent structures. OKCs rarely induce root resorption of adjacent teeth, unlike other odontogenic lesions with comparable aggressive tendencies, such as ameloblastomas.[22]

4. Imaging Techniques

Conventional radiography (primarily panoramic radiography), computed tomography (CT), and magnetic resonance imaging (MRI) are the most popular radiological imaging modalities employed in the study of OKCs.

4.1. *Panoramic radiography*

OPG is useful for determining the location, size, form, margins, and extension of odontogenic lesions such OKCs.

OKCs emerge on radiography as a well-defined unilocular or multilocular radiolucency with corticated edges. Around the roots of teeth, these cortices are frequently scalloped. Unilocular lesions are the most prevalent, but multilocular lesions are found in around 30% of patients, most typically in the mandible.[23]

Approximately 30% of OKCs are linked to at least one unerupted tooth, the third molars being the most prevalent.

This link is more common in younger patients.

However, because it provides a two-dimensional picture of maxillofacial structures with magnification, geometric distortion, and overlapping, this radiography technique has a restricted role. As a result, a three-dimensional imaging modality is frequently necessary for preoperative planning, especially in bigger lesions, to overcome these constraints.

4.2. Cone beam and multi-detector computed tomography

When examining an OKC, CBCT is thought to be more effective at demonstrating bony alterations in the cortical plates of the jaws (buccal, palatal, or lingual cortices), but MDCT is better at demonstrating internal density and soft tissue extension. The primary radiological aspects of an OKC, including size, shape (hydraulic or scalloping), edges (well-defined and corticated), internal appearance (unilocular or multilocular), and effects on nearby structures, may all be seen on a CT scan (tooth displacement, root resorption, maxillary sinus floor elevation, inferior displacement of mandibular canal).

CT also shows bony alterations (expansion in the buccolingual/palatal direction and erosion), internal density, and extension into soft tissue, which are all characteristics of OKCs. The OKCs in the mandible grow primarily mesiodistally throughout the length of the bone, resulting in limited enlargement of the buccal and lingual cortical plates.[24] (Figure 3) Furthermore, when OKCs arise from the alveolar bone next to the maxillary sinus, the sinus's floor is raised and its lumen is diminished.

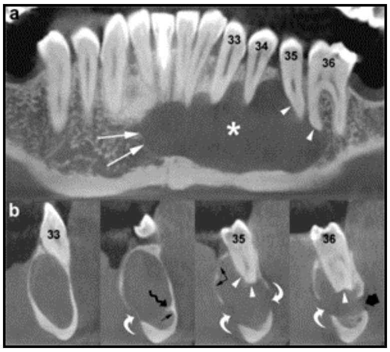

Fig. 3: CBCT image of OKC

4.3. Magnetic resonance imaging (MRI)

MRI is most commonly used as a supplement to CT (CBCT or MDCT), and it can be useful in some circumstances to provide a better view of interior characteristics and soft tissue involvement. The majority of OKCs had moderate or high signal intensity on T1-weighted sequences and heterogeneous signal intensity (from low to high) on T2-weighted sequences, according to several publications.[25]

5. Histopathology

The gross specimen analysis reveals a fluid-filled, thin-walled, friable cyst with derbies. The content's viscosity ranges from a straw-colored liquid to purulent, cheese-like materials. According to one study, a protein content of less than 4.0gm/100ml indicates an OKC diagnosis.

5.1. Epithelium

The histological characteristics are distinct and specific. The cysts are lined by a keratinized stratified squamous epithelium that is thin and varies in thickness from 5-to 8.

5.2. Basal cells

Basal cells are made up of a palisaded, polarized layer of cuboidal or columnar epithelial cells that are frequently hyperchromatic and have a 'picket fence' or 'tombstone' look.

5.3. Suprabasal layers

Suprabasal layer cells are polyhedral, with intracellular edema and intercellular bridges common. Mitotic figures are more frequently seen in the suprabasal layers than in the basal layers.

OKC lining is usually parakeratotic, but it can also be orthokeratotic, and both kinds can be found in different areas of the same cyst. Orthokeratinized OKCs make up 12 percent to 13 percent of keratinizing odontogenic cysts, and they're more common in the second to fifth decades of life, with a male predisposition, and they're more common in the mandible, with a tendency for the back of the jaw. The size can range from less than a centimeter to more than seven centimeters.[15]

5.4. Epithelial connective tissue

The epithelium-connective tissue capsule relationship is prone to failure, and separation occurs in numerous locations. Lysosomal enzyme activity may promote the restructuration of juxta epithelial collagen fibers. As a result of this infolding, the OKC grows.[15]

5.5. Connective tissue capsule

The mucopolysaccharides-rich fibrous capsule is thin and frequently loose or myxoid, with a small number of cells separated by stroma. The cystic epithelium does not differentiate independently of the stroma. As a result, keratocysts' biologic behavior may be influenced not only by the epithelium but also by the stroma.

Mast cells are found in vast numbers in the connective tissue walls, and they are more abundant just beneath the epithelium (subepithelial zone) than in the deeper sections (intermediate and deeper areas), according to Smith G et al.

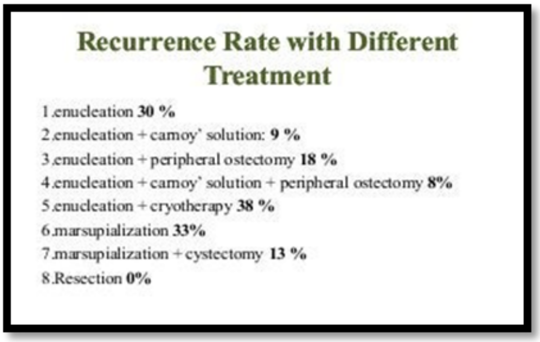

Fig. 4: Treatment modalities & recurrence rate of OKC

Mast cells play a significant role in cyst growth.[15]

5.6. *Role of inflammation*

Although OKC is classified as a developing lesion, the connective tissue wall is inflamed in the majority of instances. In cases when the cyst is near neighboring teeth, the presence of inflammation may be attributed to communications with the oral mucosa or by the periodontal ligament, which has been demonstrated to be active in recruiting inflammatory cells during a normal response.[15]

6. Differential Diagnosis

An OKC associated with an impacted tooth can mimic a dentigerous cyst. Similar to ameloblastoma, an OKC that are multilocular and located in the posterior sextant or ramus of the mandible can be mistaken for one. Finally, an OKC with a periapical location or involving an edentulous area may be mistaken for a radicular cyst or residual cyst. As a result, the most prevalent odontogenic lesions in the differential diagnosis of an OKC are dentigerous cyst, ameloblastoma, and radicular cyst, residual cyst and simple bone cyst.[26]

6.1. *Syndromic and non-syndromic multiple OKCs*

Multiple OKCs are thought to be one of the most important diagnostic criteria for NBCCS, and their existence may be the disease's initial indication. Multiple nevoid basal cell carcinoma, multiple OKCs, palmar or plantar pits, calcifications of the falx cerebri, and skeletal anomalies such as bifid, fused, or splayed ribs are all symptoms of NBCCS, also known as Gorlin–Goltz syndrome.[27]

7. Treatment of OKC

The therapy of these lesions can be divided into two types: conservative and aggressive. Enucleation with or without curettage, decompression, and marsupialization are all part of the conservative approach. Peripheral ostectomy (with rotating devices), cryotherapy (with liquid nitrogen), and Carnoy's solution application are all aggressive procedures. The goal of all of these procedures is to enucleate the cyst and reduce the risk of recurrence and surgical morbidity. Although, due to the small sample size, retrospective nature of the studies, limitations in the specifics presented of the therapy procedures, and heterogeneity of the control checkups, it is extremely difficult to evaluate the therapeutic results in diverse studies.[28] (Figure 4)

7.1. *Recurrence*

The recurrence rate of OKCs following surgery has been reported to be as high as 30%, with the majority of recurrences occurring after conservative treatments such as simple lesion enucleation. Patients with NBCCS and multilocular lesions had higher recurrence rates, according to reports.[29] Recurrences could be due to a variety of factors, including insufficient ablation of the epithelial cyst lining's highly active basal layer, the creation of small intramedullary satellite cysts left behind by conservative treatment, and the development of new lesions in the jaws' surrounding region. Some studies suggested that recurrence could be linked to the biological characteristics of the lesion and the presence of proliferative markers like Ki-67.[30]

8. Conclusion

Odontogenic keratocysts (OKCs) are odontogenic cysts that are benign but aggressive. They account for around 10% of all odontogenic cysts and are characterized by aggressive behavior. In determining the size of the lesions and their relationship to neighboring structures, a combination of clinical and radiological observations is helpful. With a better understanding of the histological nature, etiology, and recurrence affecting variables, OKC management should now focus on the following principles:

1. Accurate diagnosis
2. As far as feasible, conservative therapy
3. Adjuvant such as Carnoy's solution is used to keep the crucial exposure time near key structures constant.
4. The application of cryosurgery
5. At least 5 years of long-term follow-up
6. If necessary, repeat cryosurgery

9. Source of Funding

None.

10. Conflict of Interest

None.

References

1. Menditti D, Laino L, Domenico MD, Troiano G, Guglielmotti TM, Sava S, et al. Cysts and Pseudocysts of the Oral Cavity: Revision

of the Literature and a New Proposed Classification. *In Vivo*. 2018;32(5):999–1007.
2. Borrás-Ferreres J, Sánchez-Torres A, Gay-Escoda C. Malignant changes developing from odontogenic cysts: A systematic review. *J Clin Exp Dent*. 2016;8(5):622–8.
3. Sharifian M, Khalili M. Odontogenic cysts: a retrospective study of 1227 cases in an Iranian population from 1987 to 2007. *J Oral Sci*. 2011;53(3):361–7.
4. Li TJ. The odontogenic keratocyst: a cyst, or a cystic neoplasm. *J Dent Res*. 2011;90(2):133–42.
5. Madras J, Lapointe H. Keratocystic odontogenic tumor: reclassification of the odontogenic keratocyst from cyst to the tumor. *J Can Dent Assoc*. 2008;74(2):165.
6. Díaz-Belenguer A, Sánchez-Torres A, Gay-Escoda C. Role of Carnoy's solution in the treatment of keratocystic odontogenic tumor: a systematic review. *Med Oral Patol Oral Cir Bucal*. 2016;21(6):e689–95.
7. Guo YY, Zhang JY, Li XF, Luo HY, Chen F, Li TJ. PTCH1 gene mutations in Keratocystic odontogenic tumors: a study of 43 Chinese patients and a systematic review. *PLoS One*. 2013;8(10):e77305.
8. Antonoglou GN, Sándor GK, Koidou VP, Papageorgiou SN. Non-syndromic and syndromic keratocystic odontogenic tumors: systematic review and meta-analysis of recurrences. *J Craniomaxillofac Surg*. 2014;42:364–71.
9. Macdonald D. Lesions of the jaws presenting as radiolucencies on cone-beam CT. *Clin Radiol*. 2016;71(10):972–85.
10. Stoelinga PJW. 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(11):1662–6.
11. Stoelinga PJW, Peters JH. A note on the origin of keratocysts of the jaws. *Int J Oral Surg*. 1973;2:37.
12. Toller P. Origin and growth of cysts of the jaws. *Ann R Coll Surg Engl*. 1967;40(5):306–36.
13. Ahlfors E, Larsson A, Sjogren S. The odontogenic keratocyst: a benign cystic tumor? *J Oral Maxillofac Surg*. 1984;42(1):10–9.
14. Lia RF, Chena G, Zhaoa Y, Zhaoa YF, Liua B. Increased expression of autophagy-related proteins in keratocystic odontogenic tumors: its possible association with growth potential. *Br J Oral Maxillofac Surg*. 2014;52(6):551–6.
15. Thukral H, Singh PK, Jaiswal R, Kukreja R, Gahlot JK, Choudhary A, et al. Keratocystic Odontogenic Tumours: Etiology, Pathogenesis and Treatment Revisited. *World J Pharm Pharm Sci*. 2017;6(11):310–22.
16. Johnson NR, Gannon OM, Savage NW, Batstone MD. Frequency of odontogenic cysts and tumors: a systematic review. *J Investig Clin Dent*. 2014;5:9–14.
17. Harmon M, Arrigan M, Toner M, O'keeffe SA. A radiological approach to benign and malignant lesions of the mandible. *Clin Radiol*. 2015;70(5):335–50.
18. Kaneda T, Minami M, Kurabayashi T. Benign odontogenic tumors of the mandible and maxilla. || *Neuroimaging Clinics of North America*. 2003;13(3):495–507.
19. Johnson NR, Gannon OM, Savage NW, Batstone MD. Frequency of odontogenic cysts and tumors: a systematic review. *J Investig Clin Dent*. 2014;5:9–14.
20. Ali M, Baughman RA. Maxillary odontogenic keratocyst: a common and serious clinical misdiagnosis. *J Am Dent Assoc*. 2003;134(7):877–83.
21. Scarfe WC, Toghyani S, Azevedo B. Imaging of Benign Odontogenic Lesions. *Radiol Clin North Am*. 2018;56(1):45–62.
22. Avril L, Lombardi T, Ailianou A. Radiolucent lesions of the mandible: a pattern-based approach to diagnosis. *Insights Imaging*. 2014;5:85–101.
23. Sánchez-Burgos R, González-Martín-Moro J, Pérez-Fernández E, Burgueño-García M. Clinical, radiological and therapeutic features of keratocystic odontogenic tumours: a study over a decade. *J Clin Exp Dent*. 2014;6(3):259–64.
24. Mosier KM. Lesions of the jaw. *Semin Ultrasound CT MR* . 2015;36:444–50.
25. Minami M, Kaneda T, Ozawa K. Cystic lesions of the maxillomandibular region: MR imaging distinction of odontogenic keratocysts and ameloblastomas from other cysts. *AJR Am J Roentgenol*. 1996;166:943–9.
26. Koenig LJ, Tamimi DF, Petrikowski CG, Perschbacher SE. Diagnostic imaging: oral and maxillofacial. 2nd ed. Elsevier; 2017.
27. Barreto DC, Gomez RS, Bale AE, Boson WL, De Marco L. PTCH gene mutations in odontogenic keratocysts. *J Dent Res*. 2000;79:1418–22.
28. Aragaki T, Michi Y, Katsube K. Comprehensive keratin profiling reveals different histopathogenesis of keratocystic odontogenic tumor and orthokeratinized odontogenic cyst. *Hum Pathol*. 2010;41(12):1718–25.
29. Shear M. The aggressive nature of the odontogenic keratocyst: is it a benign cystic neoplasm? Part 1. Clinical and early References 97 experimental evidence of aggressive behavior. *Oral Oncol*. 2002;38(3):219–26.
30. Kuroyanagi N, Sakuma H, Miyabe S. Prognostic factors for keratocystic odontogenic tumor (odontogenic keratocyst): analysis of clinicopathologic and immunohistochemical findings in cysts treated by enucleation. *J Oral Pathol Med*. 2009;38(4):386–92.

Author biography

Shalini Sharma, Post Graduate Student

Nagaraju Kamarthi, Professor & Head

Sangeeta Malik , Professor

Sumit Goel, Professor

Swati Gupta, Associate Professor

Abhinav Sharma, Assistant Professor

Cite this article: Sharma S, Kamarthi N, SM, Goel S, Gupta S, Sharma A. OKC- an update on etiopathogenesis, clinical & radiological features. *J Oral Med, Oral Surg, Oral Pathol, Oral Radiol* 2022;8(2):55-60.