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ODONTOGENIC KERATOCYST- A REVIEW ON VARIOUS TREATMENT MODALITIES

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INTRODUCTION

Odontogenic keratocyst/ OKC is a developmental cyst that was first described by Philipsen (1956). OKC is now referred to by the World Health Organization (WHO) as a keratocysticodontogenic tumour KCOT, and WHO defined it as ''a benign uni-or multi-cystic, intraosseous tumour of odontogenic origin, with a characteristic lining of parakeratinized stratified squamous epithelium and potential for aggressive, infiltrative behaviour **[1]**.

Previously classified under developmental odontogenic cyst of jaw by WHO in 1971 & 1992, OKC has been reclassified and renamed as keratocysticodontogenictumor (KCOT) in the WHO classifications of head and neck tumours in 2005 due to its aggressive behavior, high recurrence rates and specific histological characterstics.

However In 2017, the new WHO classification of Head and Neck pathology re-classified OKC into cyst category.

Epidemiologically OKC accounts for approx. 7.8 % of all cyst of the jaw and incidence vary from 4-16.5% . It occurs at all ages with peak incidence in 2nd and 4th decade of life. it predominantly occurs in white population with male :female ratio of 1.6:1. Location wise it is most commonly seen twice in mandible as compared to maxilla. In mandible it occurs usually in angle – ascending ramus region (69-83%). Mandibular cyst crosess the mid line and maxillary cyst may involve sinus and nasal floor, premaxilla and maxillary third molar region. OKC may arise from temporomandibular joint also.

OKC is one of the most aggressive odontogenic cysts. It can become quite large because of its ability for significant expansion, extension into adjacent tissues and rapid growth **[2]**. Different studies showed the incidence of KCOT to be 3–11% of the odontogenic cysts **[3, 4]**.

OKC arises from cell rests of the dental lamina **[5]**. KCOT typically shows a thin, friable wall, which is often difficult to enucleate from the bone in one piece, and have small satellite cysts within the fibrous wall. Therefore Odontogenic keratocysts often tend to recur after treatment **[6, 7]**.

OKC is mostly intraosseous lesion though peripheral counterpart also have been reported in buccal gingival in canine region of mandible. Peripheral OKCs have female predominance with male: female ratio of 1:2.2 **[8]**. Clinically presents as swelling with or without pain, discharge, displacement of teeth, occasionally paraesthesia of lower lip. The expansion of the cyst is very minimal in the initial stage and it is due to the classical characteristic of the cyst to grow in antero posterior direction in the medullary space of the bone. Expansion of buccal cortex in 30% of maxillary and 50% of mandibular regions **[9-11]**.

Generally, OKC's are solitary lesions unless they are associated with nevoid basal cell carcinoma syndrome **[4, 8]**. Syndromes associated with multiple OKC are Nevoid Basal cell carcinoma syndrome (NBCCS), Gorlingoltz syndrome, Marfans syndrome, Ehlers danlos syndrome, Noonans syndrome, Orofacial digital syndrome, Simpsongolabi-behmel syndrome **[12, 13]**.

Radiographically OKC presents as well defined unilocular or multilocular (25-40%) radiolucent lesion with smooth margin (corticated margin in secondarily infected cases), displacement of adjacent teeth without root resorption, lesion may contain impacted

tooth (25-40% cases), Expansion of cortical plates (buccal > lingual) with or without perforation. Cyst grows in medullary spaces of bone in antero-posterior direction, so bony expansion is minimal in initial stages **[8, 9, 14]**.

Aspiration and biopsy are helpful in diagnosing OKC. If the fluid aspirate is subjected to immediate histologic examination, keratin can often be seen under the microscope, andif the fluid is analyzed, the protein content (at <4.0 g/100 mL) is lower than that in serum (7.1 g/100 mL)

The histological features for the diagnosis of OKC includes thin Stratified squamous epithelium lining usually 8-10 uniform layers thick; lack of rete ridges/pegs; well defined basal cell layer having cuboidal or columnar cells arranged in palisaded fashion described as "picket fence or tombstone appearance"; thin, fibrous connective tissue wall; presence of satellite cysts, daughter cysts. Based on keratinisation, 2 variants are present namely orthokeratinized and parakeratinized. Parakeratinized is aggressive in nature and usually recurs. Hence aggressive treatment is required. Whereas orthokeratinized is less aggressive and conservative management is indicated.

DISCUSSION - TREATMENT MODALITIES

The treatment of the KCOT remains controversial. Treatments are generally classified as conservative or aggressive. Conservative treatment generally includes simple enucleation, with or without curettage, or marsupialization. Aggressive treatment generally includes peripheral ostectomy, chemical curettage with Carnoy's solution, cryotherapy, or electrocautery and resection **[2, 15-18]**.

The choice of treatment should be based on multiple factors; patient age, size and location of the cyst, soft tissue involvement, history of previous treatment and a histological variant of the lesion. The goal is to choose the treatment modality that carries the lowest risk of recurrence and the least morbidity **[19, 20]**.

Treatments are usually classified as conservative like Enucleation with or without curettage and marsupialization and aggressive like peripheral ostectomy and chemical curettage with Carnoy's solution, cryotherapy, or electrocautery and resection.

The various treatment options for OKC are

1. Enucleation: a) with primary closure
b) with curettage
c) with packing
d) with peripheral ostectomy
e) with chemical fixation
f) with cryosurgery
2. Marsupialization: a) only
b) followed by enucleation

3. Resection

Enucleation + primary Closure

Also known as Partsch II procedure. It is composed by the complete removal of KCOT from the bone cavity without any macroscopic remnants of the lesion **[18, 21, 22]**. The cyst along with its lining is completely removed in toto and primary closure is done. With enucleation, no bone, other than that required for surgical access is removed.

Enucleation + curettage

Enucleation and curettage is the traditional method for managing odontogenic cysts and is the gold standard by which all other methods are compared. The technique offers patients a minimally invasive procedure with little associated morbidity and few complications. Most odontogenic cysts, including keratocysts, can be removed effectively by simple enucleation of the cystic lining and meticulous curettage of the bony cavity. Wide exposure is necessary to allow complete access to the bony cavity.

Curettage refers to removal, by scraping, of the lesion along with an inexact or immeasurable thickness of surrounding bone. Typically curettage is accomplished by means of mechanical hand instruments.

Enucleation + primary packing

Once the cyst and the cystic lining is removed, the bony cavity is packed with Bismuth Iodoform Paraffin Paste (BIPP) or Whitehead varnish or chlorhexidine gauze dressing. The packing is left in situ within the cystic cavity and it should be periodically changed till the bony cavity gets obliterated.

Enucleation + peripheral ostectomy

Following enucleation, 1 to 2 mm of bone is removed beyond the visible margin of the lesion is adequate to improve the cure rate. Peripheral ostectomy refers to removal, by powered rotary instruments, of the lesion along with an inexact or immeasurable thickness of surrounding bone. Curettage differs from peripheral ostectomy only in the fact that curettage uses hand versus rotary-type instruments.

It is difficult to estimate how much bone to be removed with a drill. This process is made easier by the use of a vital staining technique. Methylene blue or crystal violet is painted on the bony walls of the enucleated cyst. It is allowed to penetrate into the bone. The cavity is then washed out and any bone retaining the stain is removed with a drill. This process usually removes around 2 mm of bone in the marrow and about 1 mm of cortical bone.

Enucleation + chemical cauterization

Carnoy'ssolution: After enucleation carnoy's solution is applied into the cavity. It is a cauterizing agent consists of 3ml chloroform, 6 ml of absolute alcohol, 1 ml of glacial acetic acid, and 1 gm of ferric chloride **[18, 23]**.

The most usual technique involves enucleation of the lesion followed by painting the sides of the cavity with Carnoy's solution, leaving it in place for 5 minutes, and then washing out the cavity. After washing out, the cavity has brown, denatured bone on its wall. Some practitioners leave this bone in place, whereas others remove it with a drill to get down to normal bone. This technique generally involves a removal of 1 to 2 mm of bone. Carnoy's solution is neurotoxic and chemically fixes the inferior alveolar or lingual nerves if it comes in contact with them for up to 2 minutes. The nerve should therefore be protected; bone wax can be used for protection of the inferior alveolar nerve **[24]**.

The other issue with Carnoy's solution, as originally formulated, is that it contains chloroform, now classified as a borderline carcinogen. Chloroform is currently not a component of this solution, due to the fact of being a carcinogenic agent **[25-28]**. Carnoy's solution must be applied to the bone defect for 3 min after the tumour's enucleation, preventing any axonal damages and optimising the elimination of any possible remaining tumour cells **[18, 25, 30]**. The main purpose of the application of this solution is to eliminate possible viable tumour epithelial cells remaining in the bone cavity **[23, 26, 29, 31, 32]**.

Enucleation + cryosurgery

Cryosurgery has been shown to be capable of producing cellular necrosis in bone while maintaining the inorganic osseous framework **[24, 33]**. Cell death with cryosurgery occurs by direct damage from intracellular and extracellular ice crystal formation plus osmotic and electrolyte disturbances **[34]**. Temperatures below – 20 degree Celsius are believed to cause cell death on a consistent basis **[35, 36]**.

Because of liquid nitrogen's unique ability to devitalize bone in situ while leaving the inorganic framework untouched, cryotherapy has been used for a number of locally aggressive jaw lesions, including Odontogenic keratocyst, ameloblastoma, and ossifying fibroma **[24, 37-40]**.

Cystic cavity is sprayed with liquid nitrogen twice for 1 minutes, with 5 min thaw between freezes. Bone grafting can be done simultaneously. Advantges of liquid nitrogen therapy are that Bony matrix is left in place to act as scaffold for new osteogenesis. Bone grafts can be placed immediately to promote healing and decreasing risk of pathological fracture. Act as haemostasis agent and reduce scarring. Despite the cellular necrosis that cryosurgery produces, advantages of the technique include a relative lack of bleeding and scarring. However, because of the difficulty in controlling the amount of liquid nitrogen

applied to the cavity, the resultant necrosis and swelling can be unpredictable **[37]**.

Marsupialisation only

Also known as "Decompression" & Partsch I. It the earliest treatment used & was first described by Partsch in 1892. In this process a window of 1 cm is made into the cyst and lining is sutured to oral mucosa process to convert the cyst into pouch so that cyst is decompressed and it exposes the cystic lining into oral environment. Cavity is then regularly packed open with iodoform gauge till endosseous healing. Decompression helps in relieving the intracystic pressure and allowing the new bone to fill the defect. It, consequently, saves the contiguous structures such tooth roots, the maxillary sinus or the inferior alveolar canal can be saved from surgical damage.

Marsupialisation followed by Enucleation

Another possible way of removing all pathological tissue without endangering vital structures or weakening the bone is a two stage procedure where marsupialisation is followed by removal of the rest of the lesion at a later stage after the thinned out bone has increased in dimension, thus avoiding injury to structures or the probability of pathological fracture. The resultant smaller cyst is therefore easier to completely excise subsequently

While this 2 stage procedure ensures complete removal without endangering the surrounding areas, the second stage allows for new bone formation thus ensuring a complete comprehensive removal of the lining, which was not possible in a single stage marsupialization procedure. In analysing 14 identifiable studies involving 938 patients, marsupialisation was found to have lesser recurrence than enucleation according to Wushou *et al* **[41]**.

Resection

The aggressive clinical behavior of some keratocysts is well recognized. Although rare, extension into vital anatomic regions occurs and can involve the skull base **[42, 43]**, infratemporal fossa **[44, 46]**, or orbit **[3, 45]**.

Composite, segmental, or marginal resection should be considered for some large recurrent keratocysts at any site in the maxillofacial skeleton and is relatively indicated for OKCs that involve the orbit, posterior maxilla, pterygopalatine fossa, skull base, or infratemporal fossa.

Large maxillary OKCs have the potential to become secondarily infected, and tend to be more locally destructive owing to secondary infection and the structural nature of the maxillary bone. Malignant transformation of the lining of OKCs is exceedingly rare. The only absolute indication is in cases of biopsy-proven carcinoma.

The concern with this method is the functional and esthetic morbidity associated with reconstructing continuity defects of the mandible and maxilla. Advances in mandibular reconstruction, microvascular surgery, and dental implants during the last two decades, however, have made this aggressive treatment modality less foreboding

Recurrences

Recurrence of the KCOT ranges from 2.5% **[47, 48]** to 62% **[48, 49]**. Different studies show different recurrence rates. The possible mechanisms of recurrence have been described by Voorsmit *et al*. **[47]** in 1981. These state that any lining epithelium left behind in the oral cavity may give rise to a new lesion formation. Daughter cysts, microcysts or epithelial islands can be found in the walls of the original cysts. New KCOTs may develop from epithelial offshoots of the basal layer of oral epithelium **[48]**.

Recurrences generally occur within 3 to 5 years after primary treatment. The patient should be under regular followup.Initially after 2 weeks of surgery followed by a month, 3 months, 6 months and thereafter consist of annual clinical examination and radiographic assessment (panoramic radiograph). The duration of recall is controversial, but lifetime follow-up is probably warranted. For cases that involve the orbit, skull base, or infratemporal fossa, a CT scan should be obtained annually for at least 5 years in conjunction with a thorough clinical examination. Cranial nerve dysfunction is an ominous sign that should raise suspicion of malignant disease.

CONCLUSION

Although the literature contains many reports regarding management of KCOT, debate still exists as to the most effective treatment for this lesion.

Initial evaluation must include a thorough history and physical examination, radiographic studies, and the development of a probable differential diagnosis. Depending on size, location, and behavior, the clinician should decide on an incisional versus excisional biopsy. Prior aspiration may be helpful. In patients with multiple OKCs, evaluation for the presence of basal cell nevus syndrome should be undertaken. Larger OKCs, with possible cortical perforation, deserve specialized radiographic assessment, such as CT in addition to plain films. Treatment of the OKC varies from enucleation and curettage to osseous resection. Various factors that should be considered in the selection of the appropriate treatment include size and extent, location, presence of perforation or soft tissue involvement, age of individual, and primary or recurrent nature of lesion. Long-term follow-up is suggested.

REFERENCES

[1] Barnes, L., Eveson, J.W., Reichart, P., Sidransky, D. (Eds.), 2005. Pathology and Genetics of Head and Neck Tumours. IARC Press, Lyon, WHO classification of tumours series.

[2] Morgan, T.A., Burton, C.C., Qian, F., 2005. A retrospective review of treatment of the Odontogenic keratocyst. J. Oral Maxillofac. Surg. 63, 635–639.

[3] Chuong, R., Donoff, R.B., Guralnick, W., 1982. The Odontogenic keratocyst. J. Oral Maxillofac. Surg. 40, 797–802.

[4] Payne, T.F., 1972. An analysis of the clinical and histologic parameters of odontogenic-keratocyst. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 33, 536–546.

[5] Neville, B.W., Damm, D.D., Allen, C.M., Bouquot, J.E., 1995. Oral and Maxillofacial Pathology. W.B. Saunders Co., pp. 496–512.

[6] Brannon, R.B., 1977. The Odontogenic keratocyst: a clinicopathologic study of 312 cases. Part II: Histologic features. Oral Surg. Oral Med. Oral Pathol. 43, 233–255.

[7] Gang, Tae-In, Huh, Kyung-Hoe, Yi, Won-Jin, Heo, Min-Suk, Lee, Sam-Sun, Kim, Jeong-Hwa, Moon, Je-Woon, Choi, Soon-Chul, 2006. Diagnostic ability of differential diagnosis in ameloblastoma and Odontogenic keratocyst by imaging modalities and observers. Korean J. Oral Maxillofac. Radiol. 36, 177–182.

[8] Chirapathomsakul, D., Sastravaha, P., 2006. A review of Odontogenic keratocysts and the behavior of recurrences. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 101, 5–9.

[9] Philipsen HP. Keratocystic-odontogenic tumor. Barnes L, Eveson JW, Reichart PA, Sidransky D, eds. 2005. World Health Organization Classification of Tumours: Pathology and Genetics Head and Neck Tumours. Lyon, France: IARC Press; 306-7.

[10] Hyun HK, Hong SD, Kim JW. 2009. Recurrent keratocystico-dontogenic tumor in the mandible: a case report and literature review. Oral Surg Oral Med Oral Pathol Oral RadiolEndod., Aug. 108(2): e7-10.

[11] Donoff RB, Harper E, Guralnick WC. 1972. Collagenolytic

activity in keratocysts. J Oral Surg., Dec. 30(12): 87984.

[12] Bakaeen G, Rajab LD, Sawair FA, Hamdan MA, Dallal ND. 2004. Nevoid basal cell carcinoma syndrome: a review of the literature and a report of a case. Int. J Paediatr Dent., Jul. 14(4):279-87.

[13] Gonzalez-Alva P, Tanaka A, Oku Y, *et al*. 2008. Keratocystico-dontogenic tumor: a retrospective study of 183 cases. J Oral Sci., Jun. 50(2):205-12.

[14] Sciubba JJ, Fantasia JE, Kahn LB; Odontogenic keratocyst. In:, eds. Atlas of Tumor Pathology: Tumors and Cysts of the Jaw. 3rd ed. Washington, DC: Armed Forces Institute of Pathology; 1999: 34-40.

[15] Meiselman, F., 1994. Surgical management of the odontogenic-keratocyst: conserva-tive appro-ach. J. Oral Maxillofac. Surg. 52, 960.

[16] Williams, T.P., Connor, F.A., 1994. Surgical management of the Odontogenic keratocyst: Aggress-ive approach. J. Oral Maxillofac. Surg. 52, 964.

[17] Bataineh, A.B., Al Qudah, M., 1998. Treatment of mandibular Odontogenic keratocysts. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 86, 42.

[18] Blanas, N., Freund, B., Schwartz, M., Furst, I.M., 2000. Systematic review of the treatment and prognosis of the odontogenic-keratocyst. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 90, 553.

[19] Rogerson, K.C., 1991. Gorlin's syndrome: an update on diagnosis and management. Oral Maxillofac. Clin. North Am. 3, 155.

[20] Williams, T.P., 1991. Surgical treatment of odontogenic-keratocysts. Oral Maxillofac. Clin. North Am. 3, 137.

[21] Kaczmarzyk T, Mojsa I, Stypulkowska J. A systematic review of the recurrence rate for keratocysticodontogenic tumour in relation to treatment modalities. Int J Oral Naxillofac Surg 2012; 41: 756–67.

[22] Johnson NR, Batstone MD, Savage NW. Management and recurrence of keratocystic-odontogenic tumor: a systematic review. Oral Surg Oral Med Oral Pathol Oral Radiol 2013; 116: 271–6

[23] Stoelinga PJW. Long-term follow-up on keratocysts treated according to a defined protocol.

Int J Oral Maxillofac Surg 2001; 30: 14–25.

[24] Bradley PF, Fisher AD. The cryosurgery of bone. An experimental and clinical assessment. Br J Oral Surg 1975; 13: 111–27.

[25] Ribeiro Junior O, Borba AM, Alves CA, de Gouveia MM, Coracin FL, Guimar~aes Junior J. Keratocysticodontogenic tumors and Carnoy's solution: results and complications 2012; 18: 548–57.

[26] Mendes RA, Carvalho JFC, van derWaal I. Characterization and management of the keratocystic-odontogenic tumor in relation to its pathological and biological features. Oral Oncol 2010; 46: 470–5.

[27] Morgan TA, Burton CC, Qian F. A retrospective review of treatment of the odontogenic-keratocyst. J Oral Maxillofac Surg 2005; 63: 635–9.

[28] Pitak-Arnnop P. Enucleation of keratocysticodontogenic tumours: study interpretation, technical refinement and future research. Clin Oral Investig 2010a; 14: 719–21.

[29] Gonzalez-Alva P, Tanaka A, Oku Y, Yoshizawa D, Itoh S, Sakashita H *et al*. Keratocysticodontogenic tumor: a retrospective study of 183 cases. J Oral Sci 2008; 50: 205–12.

[30] Frerich B, Cornelius CP, Wieth€olter H. Critical time of exposure of the rabbit inferior alveolar nerve to Carnoy's solution. 1994; 52: 599–606. J Oral Maxillofac Surg

[31] Stoelinga PJW. The treatment of Odontogenic keratocysts by excision of the overlying, attached mucosa, enucleation, and treatment of the bony effect with Carnoy solution. J Oral Maxillofac Surg 2005; 63: 1662–6.

[32] Gosau M, Draenert FG, M€uller S, Frerich B, B€urgers R, Reichert TE et al. Two modifications in the treatment of keratocystic-odontogenic tumors and the use of Carnoy's solution –a retrospective study lasting between 2 and 10 years. Clin Oral Invest 2010; 14: 27–34.

[33] Emmings FG, Neiders ME, Greene GW, *et al*: Freezing the mandible without excision. J Oral Surg 24:145, 1966.

[34] Whittaker DK: Mechanisms of tissue destruction following cryosurgery. Ann R Coll Surg Engl 66: 313, 1984.

[35] Smith JJ, Fraser J: An estimation of tissue damage and thermal history in the cryolesion. Cryobiology 11: 139, 1974.

[36] Rosen G, Vered IY: Cryosurgery for basal cell carcinoma of the head and neck. S Afr Med J 56: 26, 1979.

[37] Pogrel MA: The use of liquid nitrogen cryotherapy in the management of locally aggressive bone lesions. J Oral Maxillofac Surg 51: 269, 1993

[38] Webb J, Brockbank J: Treatment of the Odontogenic keratocyst by combined enucleation and cryosurgery. Int J Oral Surg 13:506, 1984

[39] Emmings FG, Gage AA, Koepf SW: Combined curettage and cryotherapy for recurrent ameloblastoma of the mandible. J Oral Surg 29: 41, 1971

[40] Sippel HW, Emmings FG: Cryotherapy in the treatment of recurrent ossifying fibroma. J Oral Surg 27: 32, 1969

[41] Wushou A, Zhao YJ, Shao ZM (2014) Marsupialization is the optimal treatment approach for keratocysticodontogenic tumour. J Cranio-Maxillofac Surg 42(7): 1540–1544

[42] Jackson IT, Potparic Z, Fasching M, *et al*. Penetration of the skull base by dissecting keratocyst. J Craniomaxillofac Surg 1993; 21: 319–25.

[43] Soost F, Stoll C, Gerhardt O, Neumann HJ. Keratocysts with an expansion to the skull base (German). Zentralbl Neurochir 1999; 60: 11–4.

[44] Worrall SF. Recurrent Odontogenic keratocyst within the temporalis muscle. Br J Oral Maxillofac Surg 1992; 30: 59–62.

[45] Hennis HL, Stewart WC, Neville B, O'Connor KF, Apple DJ. Carcinoma arising in an Odontogenic keratocyst with orbital invasion. Doc Ophthalmol 1991; 77:73–9.

[46] Lund VJ. Odontogenic keratocyst of maxilla: a case report. Br J Oral Maxillofac Surg 1985;23:210

[47] Voorsmit RA, Stoelinga PJ, Van Haelst UJ. The management of keratocyst. J Maxillofac Surg 1981, 9(4): 228–236

[48] Irvene GH, Bowerman JE (1985) Mandibular keratocyst: Surgical Management. Br J Oral Maxillofac Surg 1985, 23(3): 204–209

[49] Pindborg JJ, Hansen J. Studies on odontogenic cyst epithelium 2. Clinical and roentgenologic aspects of Odontogenic keratocyst. Acta. Pathol. MicrobiolScand., 1963, 58: 283–294.