ODONTOGENIC KERATOCYST

Abstract

Odontogenic Keratocysts (OKC) are benign, intra-osseous tumors of odontogenic origin lined with parakeratinized stratified squamous epithelium. OKC's etiopathogenesis is unknown, but numerous hypotheses exist. According to various theories, OKCs originate from the residual dental lamina, basal cells, or even tissue responses to inflammation or trauma. There is also the theory that suggests OKC to be of neoplastic origin. Intraosseous OKCs affect the mandible more than the maxilla. OKCs occur in all ages, with a slight male predilection. OKCs recur between 2.5% and 62.5% after surgical excision, with higher rates in syndromic patients. Due to their growth-inhibiting and proliferative nature, OKCs are aggressive. Antigen-antibody epithelial cell markers can diagnose OKCs. Compared to OKCs without PTCH1 mutations, the expression of Ki67 was found to be significantly elevated in OKCs with PTCH1 mutations. The presence of elevated P53 levels in Odontogenic Keratocyst (OKC) has been suggested as a potential mechanism underlying cell cycle alterations. Odontogenic Keratocyst treatment is controversial and multifaceted. Enucleation, marsupialization, and decompression are conservative treatments, while molecular treatment inhibition of Hedgehog signaling. Some of the treatment modalities include enucleation followed by the use of Carnoy's solution, peripheral ostectomy, cryotherapy, and resection. OOC and OKC are aggressive but rarely malignant.

Keywords: Ki67 Nevoid Basal Cell Carcinoma Syndrome Odontogenic Keratocyst PTCH1 Mutations Recurrence

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I. INTRODUCTION

One of the common abnormalities affecting the developing tooth apparatus is an odontogenic lesion, which can be either a cyst or a tumor. Cysts are pathological cavities that contain fluid or semifluid material and are lined by epithelial tissue [1]. The Odontogenic Keratocyst (OKC) is a jaw cyst with a tendency for local invasion and recurrence. It was initially identified and described in 1876 but was labeled by Philipsen in 1956 as the odontogenic keratocyst. Pindborg and Hansen proposed histological criteria for diagnosing OKC in 1962. Odontogenic Keratocyst (OKC) is the third most common odontogenic cyst and accounts for approximately 10% of all oral cysts. There are two subtypes of OKC: solitary cysts (sporadic) and multiple cysts as part of the Gorlin-Goltz or Nevoid Basal Cell Carcinoma Syndrome (NBCCS), an uncommon autosomal dominant disease. Approximately 85% of syndromic and 30% of sporadic cases have mutations of the PTCH1(patched homolog 1) gene, indicating possible shared pathogenesis among OKC subtypes.[1]

OKC is defined as “a benign uni- or multi-cystic, intra-osseous tumor of odontogenic origin with a characteristic lining of para-keratinized stratified squamous epithelium and potential for aggressive, infiltrative behavior”. [2] The OKC is believed to originate from the epithelial remains of the tooth germ, the extension of basal cells layer of the overlying/surface epithelium, or hamartomatous proliferation of odontogenic epithelium.

It may be associated with carious teeth, impacted teeth, or tooth-bearing areas showing multiple lesions, destruction, and recurrence, and may progress to malignancy. In 2005, the pathology was reclassified as a Keratocystic Odontogenic Tumor (KCOT) as a result of investigations revealing genetic molecular alterations that are also present in neoplasms. Yet, in 2017, the World Health Organization (WHO) renamed it as an Odontogenic Keratocyst (OKC) because numerous papers demonstrated that the PTCH gene mutation could be present in non-neoplastic lesions, including dentigerous cysts, and many researchers suggested that resolution of the cyst after marsupialization was not compatible with a neoplastic process. The OKC remains part of the cyst classification in the most recent 2022 edition of the WHO classification of head and neck tumors [2].

II. ETIOPATHOGENESIS

A complete understanding of the etiopathogenesis of Odontogenic Keratocyst remains elusive. Nevertheless, numerous theories have been postulated to elucidate the genesis of this phenomenon. OKCs originate from mandibular and maxillary dental lamina remnants. Also suggested as a possible cause of this cyst is the expansion of basal cells in the oral epithelium. A rapid rate of cell division and overexpression of the Bcl-2 protein may be responsible for the cyst. PTCH gene mutations on chromosome 9q22.3-q31 cause OKCs. Nevoid Basal Cell Carcinoma Syndrome (NBCCS) and sporadic Odontogenic Keratocysts display a genetic mechanism involving two or more chromosome loci on 9q22.3. This mechanism causes the overexpression of a number of proteins, including cyclin D1 and p53.

The Developmental theory is a framework that seeks to explain and understand the processes and patterns of human development. Based on this framework, it is believed that Odontogenic Keratocysts originate from residual components of the dental lamina or its basal cells [3]. The aforementioned remnants may persist during tooth development and endure

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cystic transformations, leading to the formation of Odontogenic Keratocysts (OKCs). The OKC developmental theory has been attributed to mutations in particular genes, such as PTCH1 (patched homolog 1). [1] Stoelinga et al. (2004) conducted a study in which they found a correlation between Odontogenic Keratocysts and the development of teeth [4].

According to the Odontogenic Theory, OKCs originate in the residual odontogenic epithelium. Therefore, the epithelial lining of Odontogenic Keratocysts exhibits similarities to the enamel organ, dental lamina, and reduced enamel epithelium. It is widely believed that these residual components endure cystic changes, resulting in the formation of odontogenic keratocysts. This lends credence to the theory that OKCs originate from odontogenic tissues [3,5]. In a study conducted by Gadbail et al. (2010), the immunohistochemical profile of Odontogenic Keratocysts was evaluated. The study demonstrated the presence of positive staining for cytokeratins 13 and 19, which are commonly associated with odontogenic epithelium [6].

The Neoplastic Theory proposes that Odontogenic Keratocysts should be classified as genuine neoplastic lesions as opposed to cysts caused by developmental processes. The findings suggest that the epithelial lining of OKCs shows characteristics consistent with malignant proliferation. Several studies have documented genetic alterations, namely mutations in the PTCH1 and TP53 genes, that are frequently associated with the development of tumors [7]. The association between odontogenic keratocysts and neoplastic syndromes, such as Nevoid Basal Cell Carcinoma Syndrome, supports the neoplastic hypothesis. Previous research uncovered the presence of TP53 gene mutations in a subset of odontogenic keratocysts, indicating a potential malignant trait. As an example, research conducted by Li et al. (2011) revealed the presence of TP53 mutations in 42 percent of the examined cases.

According to the Reactive Theory, OKCs result from the localized tissue response to inflammation or trauma. Persistent inflammation or traumatic injury to the mandible may initiate the development of Odontogenic Keratocysts, as proposed. However, there is a limited understanding of the precise mechanisms underlying this theory [3]. As has been observed, trauma or chronic inflammation can initiate the development of odontogenic keratocysts (OKCs). According to a study by Li et al. (2008), patients with Odontogenic Keratocysts (OKCs) frequently manifested a history of trauma or inflammation in the affected region. Several studies have documented elevated concentrations of pro-inflammatory cytokines, namely interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha), in the cystic fluid of OKCs, lending support to the reactive theory [3].

III. CLASSIFICATION

The classification (2022) of odontogenic tumors, similar to its preceding versions (2017), is based upon their biological characteristics, specifically their potential for being malignant or benign. The latest classification (TABLE 1) provides a more precise definition of the various subtypes of odontogenic cysts and tumors.

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Table 1: WHO Classification of Odontogenic Lesions (2022)

Odontogenic Tumours
Benign epithelial odontogenic tumors 1. Adenomatoid odontogenic tumor 2. Squamous odontogenic tumor 3. Calcifying epithelial odontogenic tumor 4. Ameloblastoma, unicystic 5. Ameloblastoma, extraosseous/peripheral 6. Ameloblastoma, conventional 7. Adenoid ameloblastoma 8. Metastasizing ameloblastoma
Benign mixed epithelial & mesenchymal odontogenic tumors 1. Odontoma 2. Primordial odontogenic tumour 3. Ameloblastic fibroma 4. Dentinogenic ghost cell tumor
Benign mesenchymal odontogenic tumors 1. Odontogenic fibroma 2. Cementoblastoma 3. Cemento-ossifying fibroma 4. Odontogenic myxoma
Malignant odontogenic tumours 1. Sclerosing odontogenic carcinoma 2. Ameloblastic carcinoma 3. Clear cell odontogenic carcinoma 4. Ghost cell odontogenic carcinoma 5. Primary intraosseous carcinoma, NOS 6. Odontogenic carcinosarcoma 7. Odontogenic sarcomas
CYSTS OF THE JAWS
1. Radicular cyst 2. Inflammatory collateral cysts 3. Surgical ciliated cyst 4. Nasopalatine duct cyst 5. Gingival cysts 6. Dentigerous cyst 7. Orthokeratinised odontogenic cyst 8. Lateral periodontal cyst and botryoid odontogenic cyst 9. Calcifying odontogenic cyst 10. Glandular odontogenic cyst 11. Odontogenic keratocyst

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IV. CLINICAL FEATURES

The clinical features of odontogenic keratocyst can vary, but some common characteristics include:

1. **Location:** OKCs are intraosseous lesions that more frequently affect the mandible than the maxilla. The ratio of mandible to maxilla for OKCs is approximately 2:1, with a small number of peripheral cases. They are frequently accompanied by impacted teeth, occurring in approximately 25 to 40 percent of cases. There is a tendency for impacted teeth to be located in the mandible's posterior body and ascending ramus. OKCs in the maxilla are commonly located between the canine and lateral incisors or near the third molar.

2. **Age and Gender:** OKCs can occur at any age, with peaks in the second and third decades, as well as the fifth and sixth. Males are affected 1.6 times more frequently than females, indicating a slight male predilection.

3. **Size:** Odontogenic keratocysts exhibit a range of sizes, that spans from a few millimeters to several centimeters in diameter. Larger cysts may cause more pronounced symptoms and jaw expansion.

4. **Pain:** Odontogenic keratocysts are typically painless unless they become infected or exert pressure on surrounding structures. Pain may be experienced if the cyst becomes secondarily infected or if it causes nerve compression.

5. **Swelling:** As the cyst grows, it can cause swelling and expansion of the affected jaw. The swelling is usually painless, but it may cause facial asymmetry in more advanced cases.

6. **Tooth Displacement and Mobility:** Due to the pressure exerted by the growing cyst, adjacent teeth may be displaced. Displacement of teeth can lead to occlusal changes and may result in malocclusion. In some cases, teeth may become mobile due to the resorption of their roots [2,5,7,8].

7. **Expansion and Cortical Thinning:** As odontogenic keratocysts grow, they cause progressive expansion of the jawbone. This expansion occurs by resorption of the surrounding bone, leading to thinning of the cortical bone plates. The extent of expansion and cortical thinning can be assessed through clinical and radiographic examination [3].

8. **Pathological Fracture:** In rare cases of extensive expansion and weakening of the jawbone, an odontogenic keratocyst can lead to a pathological fracture, especially after minor trauma [5].

9. **Sinus Involvement:** If an odontogenic keratocyst affects the maxilla and extends into the maxillary sinus, it can cause sinus-related symptoms such as nasal obstruction, chronic sinusitis, or discharge [5].

10. **Recurrence:** OKCs have a recurrence rate ranging from 2.5% to 62.5 % even after surgical excision, with the recurrence rate being higher in patients with NBCCS [8].

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Here are some of the reasons for the high recurrence rate of OKC:

1. **Epithelial Lining:** The thin, fragile, fibrous capsule that surrounds the cyst and the tendency of the epithelium to detach from the fibrous wall makes cyst removal challenging [7].
2. Multiple daughter cysts/ satellite cysts [6].
3. **Cortical Bone Perforation:** OKCs can enter and destroy cortical bone, including adjacent tissues. OKC remains in bone or soft tissues following surgery enhances recurrence risk. The cystic epithelium can infiltrate cancellous bone, forming microcysts or islands of epithelial cells that can rapidly expand and generate new cysts [9-11].
4. High mitotic index of cyst epithelium [6,9-11].
5. **Keratinization and Cystic Fluid:** Keratinized material in the OKC helps it resist therapy. Keratin causes inflammation and epithelial cell proliferation, causing recurrence. Growth factors and cytokines in OKC cystic fluid encourage cell proliferation and cyst regrowth [6,9-11].
6. Supraepithelial and subepithelial separation of the epithelial lining [9].

V. NEVOID BASAL CELL CARCINOMA SYNDROME (NBCCS)

Nevoid Basal Cell Carcinoma Syndrome [NBCCS], also known as Gorlin Syndrome or Basal Cell Nevus Syndrome, is a rare genetic condition that affects the skin, nervous system, eyes, endocrine system, and bones. It is also known as Hereditary Cutaneo-Mandibular Polyonocosis, Multiple Nevoid Basal Cell Epithelioma-Jaw Cysts, or Bifid Rib Syndrome.

The clinical features of the syndrome include,

1. **Cutaneous Anomalies:** Multiple basal cell carcinoma, epidermal cysts of the skin, palmar pitting, palmar and plantar dyskeratosis, and dermal calcinosis.

2. **Dental and Osseous Anomalies:** Multiple odontogenic keratocysts (OKC), mild mandibular prognathism, frontal and temporoparietal bossing, shortened fourth metacarpals, kyphoscoliosis, bifurcated ribs, spina bifida occulta.

3. **Ophthalmic Anomalies:** Mild ocular hypertelorism, wide nasal bridge, dystopia canthorum, congenital blindness, and internal strabismus.

4. **Neurological Anomalies:** Mental retardation, dural calcification, bridging of Sella, agenesis of the corpus callosum, congenital hydrocephalus, and the occurrence of medulloblastoma.

5. **Sexual Anomalies:** Hypogonadism, calcified ovarian fibroma [7,12].

Clinical and genetic testing diagnoses Nevoid Basal Cell Carcinoma Syndrome. The autosomal dominant Gorlin-Goltz syndrome has multiple diagnostic criteria, but only two major and one minor or one major and three minor criteria are needed to diagnose it (TABLE 2). The odontogenic keratocyst is typically present in this syndrome.

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Table 2: Diagnostic Criteria for NBCCS [12]

MAJOR CRITERIA	MINOR CRITERIA
Multiple basal cell carcinoma or one under 20 years old.	Congenital malformations like cleft lip or palate, frontal bossing, hypertelorism
Multiple OKCs of the jaws	Macrocephaly
Palmar/plantar pits	Ovarian fibroma
Bifid, fused ribs	Medulloblastoma
Calcified falx cerebri	Sprengel deformity, prominent pectus deformity, and marked syndactyly of the digits.
Nevoid basal cell carcinoma in a first-degree relative	Sella turcica bridging, vertebral anomalies, modeling malformations of the hands and feet, or flame-shaped hands and feet.

The treatment of Nevoid Basal Cell Carcinoma Syndrome typically involves the participation of dermatologists, geneticists, oral surgeons, and other specialists, who employ a multidisciplinary approach. The treatment may involve surgical removal of skin tumors, monitoring and treatment of mandible cysts, routine skin examinations, and management of other related conditions. As the syndrome can be inherited autosomal dominantly, genetic counseling is also an essential component [12].

VI. RADIOLOGICAL FEATURES

Odontogenic keratocysts (OKCs) exhibit distinct features including well-defined unilocular or multilocular radiolucent regions, accompanied by a well-defined radiopaque peripheral rim and scalloped borders. The ratio of unilocular to multilocular radiolucency associated with OKC in the maxilla was 6:1, whereas it was 1.9:1 in the mandible [3]. Unilocular lesions are predominant whereas the multilocular variant is observed in approximately 30% of cases and is most frequently associated with larger lesions. About 40% of the unilocular variant is noted adjacent to the crown of an unerupted tooth [8].

There are four distinct radiologic manifestations of odontogenic keratocysts: replacement, envelopment, extraneous, and collateral. Replacement odontogenic keratocysts (OKCs) are the dental structures that develop at the site of missing teeth. The envelopment type describes odontogenic keratocysts (OKCs) that encase an adjacent unerupted tooth. Extraneous odontogenic keratocysts (OKCs) are those that are located in the ascending ramus, away from the teeth. In contrast, OKCs that occur close to the root surfaces are classified as collateral. It has been observed that OKCs may cause tooth displacement and, in rare cases, root resorption [8].

VII. HISTOPATHOLOGICAL FEATURES

The cysts are histologically characterized by the presence of a stratified squamous epithelium that synthesizes orthokeratin (10%), parakeratin (83%), or a combination of both types (7%).

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1. **Epithelium:** The thickness of the odontogenic keratocyst wall is typically minimal unless there has been co-existing inflammation. The lining epithelium exhibits distinctive features, primarily consisting of a stratified squamous epithelial surface that is parakeratinized. This surface is commonly observed to be corrugated, rippled, or wrinkled in its appearance. The epithelium exhibits a notable consistency in its thickness, typically spanning a range of 6 to 10 cell layers. The presence of a well-defined palisaded and polarized basal layer of cells is frequently characterized by a visual resemblance to a "picket fence" or "tombstone" configuration. The basal cell layer typically exhibits the presence of mitotic figures (FIGURE 1B) [7].

2. **The Epithelium-Connective Tissue Interface:** The interface between the epithelium and connective tissue is characterized by a lack of rete pegs and the potential for basal cell layers to bud, leading to the formation of satellite cysts. Due to the limited adhesion between the epithelium and the connective tissue capsule which may be related to the compromised anchoring fibrils owing to the presence of active collagenolytic enzymes within the odontogenic keratocyst wall, the interface demonstrates multiple instances of separation. (FIGURE 1A) [3].

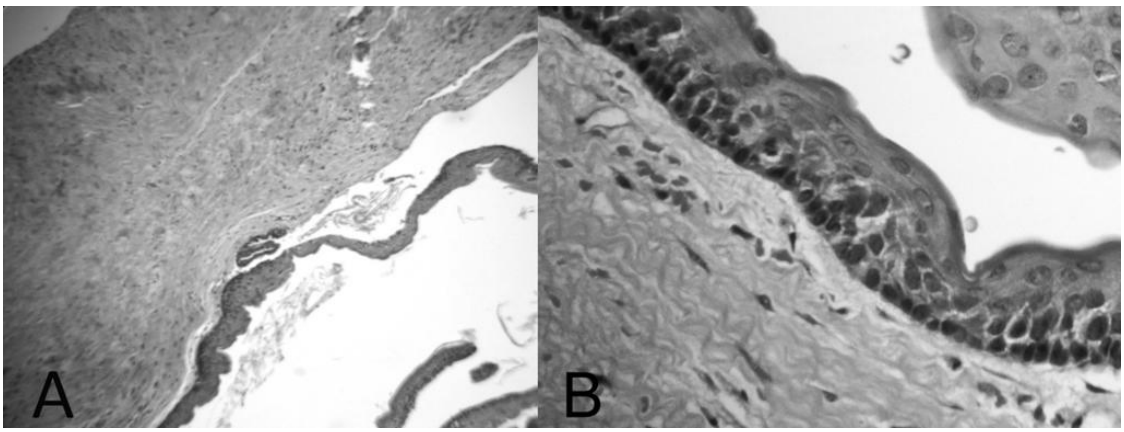

Figure 1: A) Microphotograph (10X) with the arrow pointing to the separation of epithelium from connective tissue.
1. B) Microphotograph (40X) of OKC lesion showing 6-8 layered corrugated epithelium with parakeratin and palisading nuclei with a “tombstone” appearance, of the lining.

3. **Connective Tissue Capsule:** The odontogenic keratocyst (OKC) has a thin and fragile fibrous capsule that is notable for its lack of cells, which are sparsely dispersed within a stroma that is abundant in mucopolysaccharides. In approximately 7–21% of cases, especially in those with Nevoid Basal Cell Carcinoma Syndrome (NBCCS), epithelial islands or daughter cysts have been observed within the cyst wall [3].

4. **Cystic Lumen:** The lumens may contain thin straw-colored fluid or a thicker creamy substance. Sometimes the lumen has lots of keratin, while at other times it has little. Cholesterol, as well as hyaline bodies at the sites of inflammation, may also be present. Toller's electrophoretic test of the cyst fluid showed a very low soluble protein level compared to the patient's serum [3].

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5. **Other Findings:** Odontogenic keratocysts (OKCs) exhibit several additional microscopic features, namely the occurrence of Rushton bodies (7%-32%), dystrophic calcification (10%-21%), Koilocytosis (17.1%), as well as the development of cartilage and dentinoid (17.1%). There is a slightly higher prevalence of dystrophic calcifications in primary odontogenic keratocysts (OKCs) that do not exhibit recurrence within five years, as compared to those that do recur [3].

VIII. GENETIC ASPECT OF OKC

The Hedgehog Signaling Pathway (HhS Pathway) is crucial in various biological processes, including embryonic development, cell proliferation, and cell fate determination. It consists of two receptors, PTCH1 and smoothened receptor (SMO), which collaborate in their functioning. The Sonic Hedgehog (SHh) signaling molecule is responsible for activating PTCH1, which suppresses SMO's signaling activity, obstructing excessive cell proliferation. In the presence of SHh or mutational inactivation, PTCH1's inhibitory function on SMO is lost, leading to the activation of GLI1, an oncogene associated with glioma. PTCH1 mutations increase the likelihood of cancer development.

Ki67 expression is significantly elevated in odontogenic keratocysts (OKCs) harboring PTCH1 mutations compared to those without mutations. The expression of P53 is more pronounced in odontogenic keratocysts compared to dentigerous cysts and dental follicles. The upregulation of P53 in odontogenic keratocyst specimens may provide a potential explanation for alterations in the cell cycle.

COX-2, a protooncogene, is involved in various biological processes within the epithelial lining of OKCs. Overexpression of protooncogene Bcl-2 in OKCs results in heightened cellular proliferation. Transforming growth factor-alpha (TGF-alpha) is an oncogene associated with epidermal growth factor (EGF) and has been observed in the basal and parabasal cell layers of OKCs and can act as a growth factor for OKCs. YAP/TAZ, an oncogene, is involved in organ size and tissue regeneration following injury. has the dysregulated activity of the YAP/TAZ gene may be associated with the proliferative activity of OKCs [3].

IX. IMMUNOHISTOCHEMISTRY

Odontogenic keratocysts (OKCs) exhibit aggressive behavior due to their increased proliferative potential and growth-inhibiting nature. 91% of odontogenic keratocysts (OKCs) were immunopositive for cyclin D1, epidermal growth factor receptor (EGFR), and carcinoembryonic antigens (CEA). Positive expression of the p53 protein has been observed in odontogenic keratocysts (OKCs), which increases their proliferative capacity and promotes a more aggressive phenotype. In odontogenic keratocysts (OKCs), the number of Langerhans cells (LCs) increases in the presence of inflammation, whereas elevated levels of CD34 correlate with recurrence risk. Typically, the Ki-67 protein is used as a proliferation marker to demonstrate the accelerated rate of cell division in odontogenic keratocysts (OKCs) compared to other cystic lesions [3,13-14].

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The diagnosis of odontogenic keratocysts (OKCs) can be facilitated through the utilization of epithelial cell markers that rely on antigen-antibody reactions. Several examples of epithelial cell markers, along with their respective layers, include (TABLE 3): - [7]

Table 3: Epithelial Cell Markers

1. P53 expressed in basal and suprabasal layers
2. p63 expression in basal and suprabasal layers
3. Ki-67 positive cells in the suprabasal layer of OKC.
4. CK16 is expressed strongly in suprabasal layers.
5. CK17 has strong reactivity in all layers.
6. CK19 is expressed in suprabasal cells and some basal cells
7. IPO-38 in superficial layers.
8. Bcl-1 expressed in basal layers

X. DIFFERENTIAL DIAGNOSIS

Dentigerous cysts, ameloblastoma, odontogenic myxoma, adenomatoid tumor, and ameloblastic fibroma are potential differential diagnoses to be considered in the case of odontogenic keratocyst (OKC). In addition, it is worth considering radiolucent, non-odontogenic tumors such as central giant cell granuloma, traumatic bone cyst, and aneurysmal bone cyst [7].

XI. TREATMENT AND PROGNOSIS

The management of odontogenic keratocyst (OKC) is a multifaceted and controversial matter, lacking a universally applicable treatment approach. The selection of treatment is contingent upon the patient's rate of recurrence and the minimal level of morbidity. A range of therapeutic modalities exist encompassing molecular, conservative, and aggressive approaches. Several factors, including age, lesion size, recurrence status, and radiographic evidence of cortical perforation, are to be considered while selecting an appropriate treatment.

The molecular treatment approach encompasses the administration of an orally administered pharmaceutical agent that effectively hinders the Hedgehog (Hh) signaling pathway, exemplified by the utilization of GDC-0449. Oral inhibitors targeting the Hedgehog (Hh) signaling pathway, such as Vismodegib, have undergone rigorous testing and have received approval for their application in clinical settings. According to this treatment plan, multiple large odontogenic keratocysts (OKCs) have completely regressed after two years of therapy.

The conservative treatment involves enucleation, marsupialization, and decompression techniques. Enucleation followed by open packing can be considered a judicious therapeutic approach owing to its straightforwardness and minimal likelihood of recurrence. Marsupialization can cause increased chances of recurrence; however, it is recommended that marsupialization be subsequently followed by enucleation. The

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phenomenon of decompression is commonly linked to a reduced likelihood of recurrence; however, it necessitates the utilization of two surgical approaches and an extended duration of treatment.

Aggressive therapeutic interventions involve the utilization of various techniques, including the application of Carnoy's solution after enucleation, peripheral ostectomy, cryotherapy, and resection. Carnoy's solution has been widely recognized as an effective approach in the treatment of odontogenic keratocysts (OKC) through enucleation. However, it is important to note that this particular procedure is accompanied by a significant incidence of morbidity. The utilization of ultrasonic debridement is a novel treatment approach that involves removing remnants of the cystic cavity's epithelial lining. This technique aims to protect the integrity of the surrounding bone and adjacent tissues [3].

Malignancy is rare in OOC and OKC. Cysts with ortho-keratinized surfaces have an increased risk of cancer, however, the evidence is ambiguous. OKCs are more aggressive than other odontogenic cysts. A review found SCC from OCC or other odontogenic cysts. 60% of primary intraosseous SCCs from odontogenic cysts were inflammatory, such as residual and radicular cysts, and 14% were OKC or OOC [15].

XII. CONCLUSION

The research on odontogenic keratocyst (OKC) has enhanced our understanding of its nature and available treatments. Through collaborative efforts and advancements in diagnosis, treatment, and genetic insights, clinicians are better equipped to address the challenges posed by OKC. However, long-term monitoring and continued research are necessary to improve patient outcomes and develop targeted therapies. The progress made so far provides a strong foundation for further advancements in the management of OKC and associated syndromes.

REFERENCES

[1] Florindo JB, Bruno OM, Landini G. Morphological classification of odontogenic keratocysts using Bouligand–Minkowski fractal descriptors. Computers in biology and medicine. (Comput. Biol. Med.) 2017 Feb 1; 81:1-0.
[2] Favia G, Spirito F, Lo Muzio E, Capodiferro S, Tempesta A, Limongelli L, Lo Muzio L, Maiorano E. Histopathological Comparative Analysis between Syndromic and Non-Syndromic Odontogenic Keratocysts: A Retrospective Study. Oral. 2022 Jul 22;2(3):198-204
[3] Elshafei MM, Afifi NS, Ghazy SE, Gad HA, Rasmy MM. Odontogenic Keratocyst: A Review of Histogenesis, Classification, Clinical Presentation, Genetic Aspect, Radiographic Picture, Histopathology, and Treatment. Egyptian Journal of Histology (Egypt. J. Histol.). 2022 Jul 1;45(2):325-37.
[4] Stoelinga PJ. The odontogenic keratocyst revisited. International Journal of Oral and Maxillofacial Surgery (J Oral Maxillofac Surg). 2022 Nov 1;51(11):1420-3.
[5] Chen P, Liu B, Wei B, Yu S. The clinicopathological features and treatments of odontogenic keratocysts. American Journal of Cancer Research (Am J. Cancer Res.) 2022;12(7):3479.
[6] Gadbail AR, Chaudhary M, Patil S, Gawande M. Actual Proliferating Index and p53 protein expression as prognostic marker in odontogenic cysts. Oral Diseases (J.Oral Dis.). 2009 Oct;15(7):490-8.
[7] Dwivedi D. Odontogenic Keratocyst: A Systemic Review. European Journal of Pharmaceutical and Medical Research (Eur J Pharm Med Res.) 2021;8(4):654-0.
[8] Sarfi D, Bouya M, Yahya IB. Conservative management of a large odontogenic keratocyst: A case report. Advances in Oral and Maxillofacial Surgery (J. Adv Oral. Maxillofac Surg.). 2022 Jan 1; 5:100238.
[9] Nayak MT, Singh A, Singhvi A, Sharma R. Odontogenic keratocyst: What is in the name? Journal of natural science, biology, and medicine. 2013 Jul;4(2):282.

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[10] Li TJ. The odontogenic keratocyst: a cyst, or a cystic neoplasm? Journal of dental research. (J.Dent. Res.) 2011 Feb;90(2):133-42.
[11] Bera RN, Tandon S, Tiwari P, Mishra M. Recurrence and Prognosticators of Recurrence in Odontogenic Keratocyst of the Jaws. Journal of Maxillofacial and Oral Surgery (J Maxillofac Oral Surg.). 2023 Jan 5:1-2.
[12] Jawa DS, Sircar K, Somani R, Grover N, Jaidka S, Singh S. Gorlin-Goltz syndrome. Journal of oral and maxillofacial pathology (J. Oral Maxillofac. Pathol.): JOMFP. 2009 Jul;13(2):89.
[13] Kadashetti V, Patil N, Datkhile K, Kanetakar S, Shivakumar KM. Analysis of expression of p53, p63 and proliferating cell nuclear antigen proteins in odontogenic keratocyst: An immunohistochemical study. Journal of Oral and Maxillofacial Pathology (J. Oral Maxillofac. Pathol.): JOMFP. 2020 May;24(2):273.
[14] Chandrashekar C, Patel P, Thennavan A, Radhakrishnan R. Odontogenic keratocyst: Analysis of recurrence by AgNOR, p53 and MDM2 profiling. Journal of Oral and Maxillofacial Pathology (J. Oral Maxillofac. Pathol.): JOMFP. 2020 Jan;24(1):184.
[15] Mahdavi N, Zavarei M, Derakhshan S, Nasab MH. Orthokeratinized odontogenic cyst: Report of eight cases and review of literature regarding its malignant transformation. Journal of Oral and Maxillofacial Pathology (J. Oral Maxillofac. Pathol.): JOMFP. 2021 Mar;25(Suppl 1): S11.

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