

REVIEWS

ODONTOGENIC KERATOCYST: A COMPREHENSIVE REVIEW

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ABSTRACT

INTRODUCTION: An odontogenic keratocyst (OKC) is a developmental cyst of the jaws characterized by aggressive growth and a high recurrence rate, leading to ongoing challenges in its classification and management.

AIM: This article aims to provide a comprehensive overview of the OKC, examining its etiology, clinical and radiographic features, histopathology, and current treatment strategies.

MATERIALS AND METHODS: This review synthesizes information from relevant literature, including historical accounts of OKC classification, clinical case reports, and studies detailing its pathogenesis, histopathology, and treatment modalities.

RESULTS: OKC presents with variable clinical features, often in the mandible, and radiographically appears as a well-defined radiolucent lesion. Histologically, it is characterized by a thin, parakeratinized epithelium. Treatment typically involves surgical removal, but recurrence is a concern.

CONCLUSION: An OKC remains a clinically significant lesion requiring careful diagnostic evaluation and tailored management to minimize recurrence and associated morbidity.

Keywords: odontogenic keratocyst, cysts, jaws, maxilla, mandible

INTRODUCTION

Odontogenic cysts of the jaws have a long history, with early documentation dating back to the 17th century. The odontogenic keratocyst (OKC), a particularly noteworthy developmental cyst, was first described in the late 19th century. Since then, its classification and understanding have evolved significantly, marked by changes in terminology and ongoing

debate about its true nature. This article aims to provide a comprehensive overview of the OKC, encompassing its historical context, clinical features, pathogenesis, and current diagnostic and treatment approaches.

AIM

The primary objective of this study is to analyze the clinical presentation, radiological features, and histopathological characteristics of OKCs. Additionally, the study aims to evaluate the efficacy of various treatment modalities in managing OKCs and preventing recurrence. By assessing patient outcomes and identifying key prognostic factors, this research seeks to contribute to the optimization of diagnostic and therapeutic approaches, ultimately improving patient care and treatment success rates.

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MATERIALS AND METHODS

This review article is based on information from 35 full-text articles published in prestigious medical journals between 1967 and 2024.

RESULTS AND DISCUSSION

Classification

Cystic swellings of the jaws were ostensibly first documented by Scultetus in 1654, though it was not until 1728 that Fauchard postulated a potential association between these lesions and the dentition (1).

The terminology and classification of odontogenic cysts have undergone numerous revisions over the years due to their ambiguous nature. The earliest recorded designation in the literature was *dental cyst*, a term introduced by John Hunter in 1774. Among other names that were utilized in the following two centuries were *dermoid cyst* and *primordial cyst* (2).

The OKC stands as an enigmatic and paradoxical developmental cyst, its existence first chronicled by Mikulicz in 1876 as an integral component of a hereditary maxillofacial disorder. Nevertheless, it was not until 1926 that it was initially ascribed the nomenclature *cholesteatoma*, a term connoting a cystic or *open* conglomeration of keratinous squames ensconced within a living epithelial matrix (3).

In 1956, Philipsen assigned the lesion the name by which it is presently recognized—odontogenic keratocyst. The quintessential clinical and histopathological attributes of the cyst were meticulously elucidated by Pindborg and Hansen in 1963. Thereafter, in 1967, Toller postulated that the odontogenic OKC ought to be reclassified as a benign cystic neoplasm, owing to its markedly aggressive biological disposition (4). In the 2005 WHO classification of head and neck tumors, the OKC was subsumed under the category of odontogenic neoplasms, a decision predicated on its aggressive nature and the presence of PTCH gene mutations (5). However, numerous scholars contended that the evidence supporting its classification as a neoplasm was inadequate, culminating in its reclassification under odontogenic cysts in 2017. Their argument rested on the observation that OKC undergoes complete regression following decompression, a phenomenon inconsistent with true neoplasms (6). Nevertheless, the designation *keratocystic*

odontogenic tumor (KCOT) has persisted as a synonymous term for OKC (6).

Epidemiology and Clinical Features

The OKC is renowned for its insidious propensity for aggressive expansion within its anatomical vicinity and its exceptionally high recurrence rate. This recurrent nature is underpinned by a constellation of contributory factors, including suboptimal therapeutic interventions, incomplete extirpation of the lesion, an elevated mitotic index within the cyst's epithelial lining, considerable cystic dimensions, and its precise anatomical positioning—an aspect that becomes particularly formidable when surgical access is impeded (7).

Despite their aggressive nature, OKCs typically incite minimal osseous expansion, as their proliferation remains largely confined to the intramedullary compartment, allowing them to propagate insidiously within the bone.

Lesions of considerable magnitude, characterized by pronounced cortical plate erosion and involvement of adjacent anatomical structures, may paradoxically remain asymptomatic, thereby eluding early clinical detection and culminating in a protracted diagnosis.

Odontogenic keratocysts predominantly manifest in adults between the ages of 30 and 50, constituting approximately 14.3% of all odontogenic tumors (8). In their early stages, OKCs are largely asymptomatic, as even sizable lesions fail to provoke significant jaw expansion, rendering them imperceptible upon facial examination (9). However, in certain cases, patients may present with symptoms such as swelling, pain, sensory abnormalities, purulent discharge, or dental mobility, with swelling being the most frequently observed clinical feature (10).

As an OKC continues its unchecked proliferation, progressive osseous expansion ensues, eventually culminating in facial deformity. The thinning of the cortical bone layer results in a palpatory sensation akin to a ping-pong ball and may elicit a brittle, parchment-like sound upon examination. In advanced cases, pathological fractures may occur, or the delicate bone plate may be entirely resorbed, producing a detectable fluctuation upon palpation (11).

Furthermore, the OKC may extend into the nasal cavity and maxillary sinus, and in severe instances, encroachment upon the orbital structures can precipitate visual disturbances, including diplopia. When situated in proximity to dentition, OKC can exert compressive forces on adjacent teeth, leading to displacement, loosening, or tilting, frequently culminating in tooth loss. Of particular clinical significance is the cautious approach required when extracting mobile teeth in OKC patients. If the mobility stems from cystic involvement, extraction may inadvertently rupture the lesion, revealing sebaceous-like material within. Additionally, secondary infections of the tumor can provoke systemic manifestations, including swelling, pain, fever, and generalized malaise, further complicating the clinical course (12).

Like other odontogenic lesions OKCs arise exclusively within tooth-bearing regions of the jaws. They exhibit a marked predilection for the mandible, where they occur at twice the frequency observed in the maxilla (13). When originating in the mandible, they predominantly localize to the posterior sextant, particularly the angle and ramus (14). In contrast, maxillary OKCs most frequently emerge in the anterior sextant—typically between the canine and lateral incisor—as well as in the region of the third molar (15). Notably, larger lesions are especially prevalent at the mandibular angle and ramus (16).

From a positional standpoint, OKCs may be found in a periapical, pericoronal, or lateral root orientation. However, in approximately 30% of cases, they exhibit no discernible relationship with any dental structures, further underscoring their enigmatic and unpredictable nature (17).

Radiographically, OKCs present as well-demarcated radiolucent lesions, either unilocular or multilocular, encased within corticated margins. The unilocular variant predominates, whereas the multilocular form is observed in approximately 30% of cases, with a particular predilection for the mandible (18).

On panoramic radiographs, mandibular unilocular OKCs may exhibit sparse and incomplete septations within the lesion, a phenomenon more frequently encountered in larger OKCs than in their smaller counterparts. This radiographic appearance, coupled with the lesion's aggressive behavior,

underscores the importance of careful differential diagnosis.

Their propensity for invasive growth necessitates precise diagnostic evaluation, with radiological imaging—primarily computed tomography (CT) and, in select cases, magnetic resonance imaging (MRI)—playing a pivotal role in their identification and management.

Although imaging alone may not always yield a definitive diagnosis, a comprehensive understanding of both the typical and atypical radiological characteristics of OKCs is indispensable for accurate assessment and therapeutic planning (19).

Pathogenesis and Molecular Aspects

The OKC can manifest sporadically or as a part of autosomal dominant condition known as nevoid basal-cell carcinoma syndrome (NBCCS) (Gorlin syndrome/Gorlin–Goltz syndrome) (20).

The neoplastic paradigm of OKCs is substantiated by an array of molecular investigations that have elucidated a pronounced loss of heterozygosity. The scholars conducting these studies identified compelling evidence of allelic depletion, predominantly affecting pivotal tumor-suppressor genes, including p16, p53, PTCH, MCC, TSLC1, LTAS2, and FHIT (21,22). Given that these genetic loci are intrinsically linked to a spectrum of human neoplasms, their inactivation provides a robust molecular framework underpinning the markedly aggressive biological behavior exhibited by OKCs. Furthermore, empirical observations have delineated a heightened prevalence of allelic loss in daughter cysts, reinforcing their role in the lesion's recurrent and infiltrative nature (22).

The etiology of odontogenic OKCs remains contentious, though they predominantly arise intraosseously from odontogenic epithelium, particularly remnants of the dental lamina. While OKCs in dentate regions likely originate from these remnants, the absence of such structures in the mucosa of the third molar region suggests an alternative mechanism. The presence of epithelial islands in the overlying mucosa implies a possible contribution from basal cell extensions of the oral epithelium (23).

Peripheral OKCs are rare, occurring primarily in the gingiva and buccal mucosa. Their presence in the gingiva reinforces the hypothesis of deriva-

tion from residual dental lamina or proliferative basal cells of the oral epithelium (24,25).

Histopathology and Microscopic Features

Histopathologically, an OKC is distinguished by a cystic lumen lined with a uniform, 6–8-layered corrugated parakeratinized stratified squamous epithelium. The epithelial lining exhibits a basal layer composed of tall columnar cells arranged in a palisading pattern, with reverse nuclear polarity (26). The vertical arrangement of the nuclei of the basal cell is imparting the basal layer a tombstone phenotype.

The basal cell layer typically exhibits some mitotic figures, with mitotic activity notably elevated in cysts associated with NBCCS. The suprabasal cells are polyhedral in shape, displaying intercellular edema and well-defined intercellular bridges. These cells exhibit a higher mitotic rate than the basal layer and lack the characteristic gradual flattening of epithelial cells. Instead, there is an abrupt transition between the suprabasal cells and the keratin layer. The surface keratinization is parakeratotic, presenting a corrugated and rippled appearance with retained nuclear remnants (23).

The epithelium-connective tissue interface is characteristically flat, lacking rete pegs, and exhibits a propensity for basal cell budding and the formation of satellite cysts. Additionally, multiple areas of clefting are observed at the interface, attributed to the weak attachment between the epithelium and the connective tissue capsule (27). This phenomenon is explained by proteolytic enzymes acting on anchoring fibrils (28).

Alramadhan et al. meticulously analyzed a cohort of 554 OKC cases, among which 33 were unequivocally identified as syndromic. Their findings elucidated a repertoire of distinct histopathological hallmarks that set the syndromic cases apart with remarkable specificity. Syndromic OKCs exhibited distinct microscopic features compared to their sporadic counterparts. One of the most prominent characteristics is full-thickness hyperchromasia, creating a carcinoma in situ-like appearance with uniform nuclear changes throughout the epithelium. Additionally, numerous rete ridge formations were present, a feature uncommon in sporadic OKCs. The parakeratin layer was scant, with minimal corrugation, further distinguishing syndromic cases. The cystic lin-

ing varied in thickness, reflecting an irregular epithelial architecture. A proliferative lining with intramural papillary projections suggested heightened epithelial activity, while cellular vacuolization extends from the basal cell layer to the luminal surface, contributing to the unique histological profile of syndromic OKCs (29).

A frequently observed histological alteration following marsupialization is the transformation of the parakeratinized epithelium of OKCs into oral-lining epithelium or orthokeratinized epithelium. A study by Awni and Conn reported that in a series of 17 cases, seven (41%) exhibited a loss of parakeratinization, with two cases undergoing a complete transition to a non-keratinized lining after decompression. However, these findings were not statistically significant and showed no correlation with the duration of treatment (30).

Immunohistochemistry

Routine diagnostic procedures seldom utilize ancillary testing to differentiate OKC from its mimics. However, immunohistochemical profiling reflects OKC's aggressive nature and distinguishes it from less aggressive lesions. A recent meta-analysis by Almeida et al. finds significant differences in the IHC profiles of the dentigerous cyst and the OKC. Elevated Ki-67, p53, Bcl-2, and PCNA in OKC suggest a more aggressive behavior. Furthermore, genetic markers, such as PTCH1 mutations, call for personalized treatment plans. These findings improve diagnostic precision and support targeted therapies, which could significantly impact patient management in oral and maxillofacial surgery (31).

Differential Diagnosis

An OKC should be considered in the differential diagnosis of nearly all cystic odontogenic lesions, making the recognition of its key histologic features essential. However, several factors may obscure its diagnosis. Inflammatory changes or secondary infection can significantly alter its classic morphology, causing the epithelial lining to thicken, develop rete ridges, and lose its characteristic basal palisading and luminal parakeratinization, resembling dentigerous cysts or unicystic ameloblastomas. Additionally, the orthokeratinized odontogenic cyst (OOC) presents a diagnostic challenge, as it produces orthokeratin and features a prominent granular layer. Un-

like OKC, OOC is less aggressive, with a recurrence rate of approximately 2%. While foci of orthokeratin may be seen in OKC, the presence of definitive OKC features warrants its classification as such. Given the clinical significance of distinguishing OKC from other odontogenic cysts, thorough histologic examination of all submitted tissue is imperative to ensure accurate diagnosis (32).

Other lesions frequently considered in the differential diagnosis include the dentigerous cyst and the radicular cyst. However, these conditions lack the distinct corrugated parakeratin seen in OKC, as well as the epithelial clefting associated with the underlying stromal capsule. The dentigerous cyst is characterized by a relatively thinner epithelial lining, typically consisting of only two to four layers. On the other hand, the radicular cyst, a classic example of an inflammatory odontogenic cyst, commonly demonstrates prominent inflammation within the fibrous wall, along with pronounced pseudoepitheliomatous hyperplasia (8).

Treatment Modalities and Recurrence

The standard treatment for OKCs involves enucleation, excision of the overlying mucosa, and curettage of the underlying bone to minimize recurrence (27). In cases of secondary infection in developmental odontogenic cysts, preoperative antibiotic therapy is generally recommended (33). Enucleation can be followed by the application of Carnoy's solution, a commonly employed adjunct alongside cryotherapy and peripheral ostectomy to further reduce recurrence risk. A follow-up period of at least 10 years is advised to monitor potential relapse (34).

To lessen the chance of recurrence after enucleation and peripheral ostectomy, various supplementary treatments can be used on the remaining tissue. These treatments work by chemically treating the peripheral lining. Carnoy's solution, traditionally used, resulted in roughly an 11% recurrence rate when applied for three minutes to minimize side effects. However, the US Food and Drug Administration (FDA) banned its use in 2013 due to chloroform's carcinogenic properties. Consequently, a modified Carnoy's solution, without chloroform, was developed. More recently, 5-fluorouracil has been introduced for topical use, utilizing its antimetabolic effects to induce cell death (35).

Recurrence is primarily attributed to several factors, including the persistence of satellite cysts post-surgery, the friability of the epithelial lining due to the absence of rete ridges and metalloproteinase-mediated collagen degradation, the cyst's extension into cancellous bone complicating surgical margin determination, and a high mitotic index within the cystic epithelium (27).

CONCLUSION

The OKC remains a significant entity in oral and maxillofacial pathology due to its unique characteristics and clinical behavior. Accurate diagnosis, based on a combination of clinical, radiographic, and histopathologic findings, is essential to differentiate OKC from other odontogenic lesions. Effective treatment, tailored to the individual case, is crucial to minimize recurrence and manage associated morbidity. Continued research into the pathogenesis and molecular mechanisms of OKC is warranted to further refine diagnostic and therapeutic approaches.

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