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REVIEW ARTICLE

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Glandular odontogenic cyst: insights into a rare and challenging lesion

Abstract:

The glandular odontogenic cyst (GOC) is a rare developmental odontogenic cyst characterized by an epithelial lining resembling salivary or glandular tissue. Its aggressive behavior and potential for recurrence pose challenges in both diagnosis and management. This review consolidates current knowledge on etiopathogenesis, clinical presentation, histopathological features, and treatment approaches. GOC primarily affects male adults, often presenting asymptomatically, and is most commonly found in the mandible. Radiographically, it appears as a unilocular or multilocular radiolucency, frequently associated with unerupted teeth. Histologically, GOC is distinguished by a multilocular cystic structure lined with epithelial cells, which may include "hobnail," clear, and mucous cells. While traditionally thought to lack MAML2 gene rearrangements, recent studies have identified MAML2 fusion transcripts in recurrent GOCs, suggesting a link to aggressive behavior. The differential diagnosis includes both benign and malignant conditions. Treatment options range from conservative management to radical resection, with recurrence being a notable concern. This review highlights the need for accurate diagnosis and long-term follow-up, as no histopathological features reliably predict recurrence. Further research is needed to optimize treatment strategies and deepen understanding of its biological behavior.

Keywords: Odontogenic cyst; Odontogenic lesion; Glandular odontogenic cyst; Diagnostic challenge.

INTRODUCTION

Glandular odontogenic cyst (GOC) is a developmental odontogenic cyst characterized by an epithelial lining that mimics salivary or glandular tissue. Initially described by Padayachee and van Wyk in 1987 as the "sialo-odontogenic cyst", GOC was subsequently renamed and formally recognized as a distinct entity by Gardner et al. in 1988[1,2]. Since its inclusion in the 1992 World Health Organization (WHO) classification, GOC has been acknowledged for its unique histopathological features and diagnostic complexity[3].

Statement of Clinical Significance
The glandular odontogenic cyst (GOC) is a rare developmental odontogenic cyst known for its aggressive potential and propensity for recurrence. This review underscores the diagnostic complexities and clinical implications of GOC, providing critical insights to enhance diagnostic precision, refine treatment strategies, and advance understanding of its molecular pathogenesis.

The last edition of WHO head and neck tumors classification further underscores the importance of identifying GOC as a discrete lesion within the spectrum of odontogenic cysts due to its clinical significance[4].

Despite its recognition, GOC remains exceedingly rare, with about 200 cases reported in the literature. Retrospective studies have reported a prevalence of GOC among odontogenic cysts ranging from 0.003 to 0.02%[5-7]. However, these numbers may be underestimated due to the challenges in publishing case reports and the diagnostic difficulties faced by pathologists, particularly in distinguishing GOC from other

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lesions, such as mucoepidermoid carcinoma (MEC). In fact, the rarity poses significant diagnostic challenges, necessitating comprehensive clinical, radiographic, and histopathological evaluations to distinguish GOC from other odontogenic and non-odontogenic lesions. Its potential for aggressive behavior and recurrence further highlights the need for accurate diagnosis and effective management strategies.

This study provides an in-depth exploration of GOC, emphasizing its diagnostic challenges, histopathological intricacies, and clinical implications. By consolidating current knowledge on its pathogenesis, diagnostic criteria and therapeutic approaches, this work aims to serve as a valuable resource for clinicians and researchers navigating the complexities of this rare odontogenic cyst.

LITERATURE REVIEW

Etiopathogenesis

GOC is classified as a developmental odontogenic cyst, with its etiology predominantly attributed to remnants of the dental lamina. These odontogenic epithelial cell rests are considered the primary source of its development. The expression of microscopic glandular features was attributed to the pluripotent potential of odontogenic epithelium. Despite these, the precise mechanisms underlying its pathogenesis remain unclear and are an area of active investigation.

A research using next-generation sequencing on six cases of GOC has failed to identify pathogenic mutations in a panel of 50 oncogenes and tumor suppressor genes, suggesting that its development may not follow the conventional genetic pathways associated with other cystic or neoplastic lesions[8]. This finding highlights a gap in the literature, underscoring the need for more extensive molecular analyses with larger cohorts to better elucidate the underlying genetic mechanisms of GOC.

Traditionally, GOCs are considered to lack *MAML2* gene rearrangements — a molecular hallmark commonly observed in MEC, aiding in their differentiation[9,10]. However, a case reported by Greer et al.[11] identified *MAML2* fusion transcripts in one recurrent GOC. The authors note that this finding does not provide definitive molecular evidence to support the theoretical progression of biologically aggressive recurrent GOCs to intraosseous MEC. Nevertheless, they emphasize that the identification of *MAML2* gene rearrangements in a biologically aggressive recurrent GOC is a significant finding that warrants further investigation in a larger series of similarly behaving GOCs[11].

Clinical features

GOC predominantly affects male adult patients, with the average age at presentation being approximately 50 years, and a peak incidence is observed between the 5th and 7th[12,13]. Clinically, most GOC cases are asymptomatic and are discovered incidentally during routine radiographic exams[4,13]. In some instances, GOC may present as a slow-growing expansion, often without significant symptoms. The mandible is the most affected site, particularly in the anterior region[14,15].

Radiographic presentation

The radiographic presentation of GOC is nonspecific, often mimicking other odontogenic and non-odontogenic conditions. The lesion is characterized by its appearance as an unilocular or multilocular radiolucency, typically surrounded by a radiopaque sclerotic margin, which may or may not be associated with root resorption. It is important to highlight that the 2022 WHO classification emphasizes that a radiolucent cystic lesion in the tooth-bearing areas of the jaw is an essential diagnostic criteria for GOC[4]. Other possible radiographic features include bone expansion, tooth displacement, cortical bone perforation, and root resorption[13]. Also, the multicenter study conducted by Heiliczer et al.[16] showed that almost a quarter of GOC cases were associated with unerupted teeth.

Histopathological characteristics

One of the most debated aspects of the GOC is the histopathological criteria necessary for its definitive diagnosis, as no established consensus exists in the literature. Numerous histological features have been identified[12]; however, it is important to emphasize that while the presence of a greater number of these characteristic features enhances diagnostic confidence, not all features are consistently observed in every case[4].

The histopathological characteristics of GOCs often include multilocular cystic structures lined by an epithelium of varying thickness. This lining ranges from a thin layer of flattened squamous or cuboidal cells to stratified squamous epithelium, frequently exhibiting localized epithelial thickenings or plaques, resembling those seen in lateral periodontal cysts. A consistent finding is the presence of cuboidal or low columnar cells with a "hobnail" appearance on the luminal surface[12]. Other distinguishing features may include intraepithelial microcysts, apocrine metaplasia, clear cells, papillary projections (tufting), cilia, and mucous cells. Figure 1 illustrated some of these characteristics.

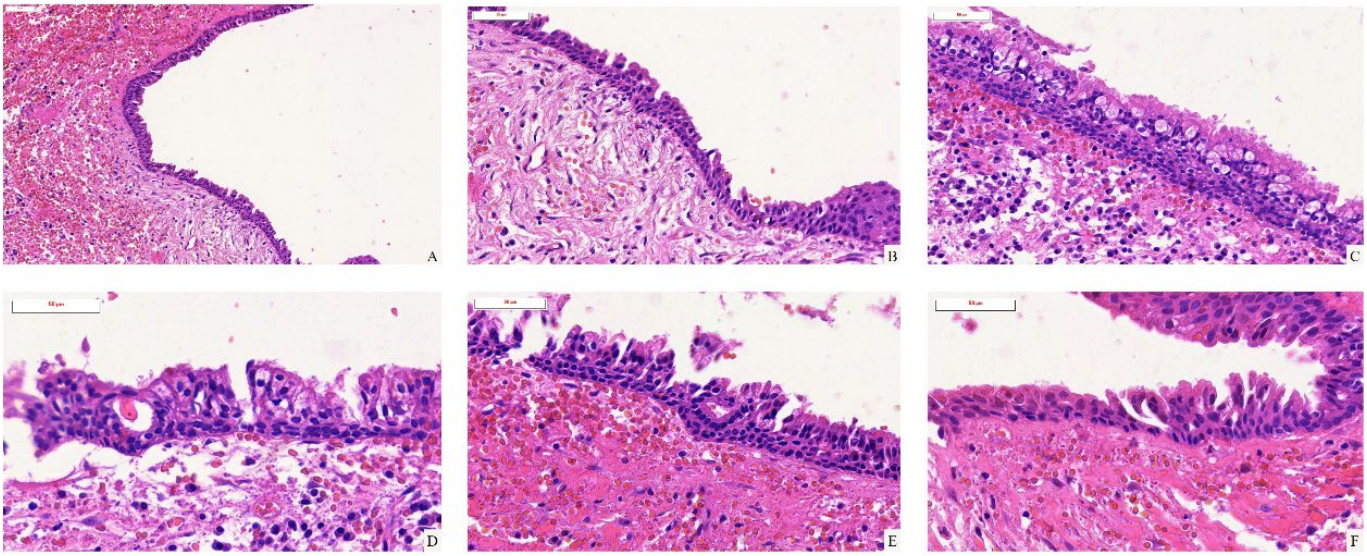

Figure 1. Representative photomicrographs of the glandular odontogenic cyst (hematoxylin and eosin stain). (A) Cystic cavity lined by nonkeratinized stratified squamous epithelium with focal ciliation (x20). (B) Variable thickness of the cyst lining (x40). (C) Cyst lining containing clear and mucous cells, with ciliation (x40). (D) Presence of small intraepithelial microcystic spaces (x40). (E) Duct-like structure within the cyst lining (x40). (F) Papillary projections ("tufting") of the epithelial lining (x40).

According to the 5th edition of the WHO Classification[4], the histopathological features of GOCs are as follows:

Cyst architecture: GOCs are often multilocular.

Epithelial lining: The lining epithelium varies in thickness, ranging from a thin layer of flattened squamous or cuboidal cells to stratified squamous epithelium. Whorled epithelial thickenings or plaques, like those observed in lateral periodontal cysts, are frequently present.

Luminal surface cells: Cuboidal or low columnar cells, often referred to as "hobnail" cells, are consistently found on the luminal surface in all cases.

Other characteristic features: intraepithelial microcysts; apocrine metaplasia; clear cells; papillary projections (tufting); cilia; mucous cells.

Biological behavior

The behavior of GOC has been investigated to better understand its potential for growth, recurrence, and tissue invasion. A series of 16 cases revealed elevated expression of proteins associated with cell invasiveness, indicating that invadopodia activity could be a mechanism through which GOCs achieve local invasion, offering a partial explanation for their distinctive biological behavior[17]. Felipe Junior et al.[18] found that GOCs exhibit high Cyclin D1 expression in both basal and suprabasal layers, suggesting a disruption in the G1-S phase of the cell cycle, which could promote epithelial proliferation. EGFR was expressed throughout the cyst lining, indicating that EGF might stimulate proliferation. Additionally, SOX2 was expressed in GOCs, though more locally compared to other cyst types. These findings point to molecular mechanisms that may contribute to the aggressive biological behavior of GOCs. Also, a recent multicenter study of 17 GOC samples found overexpression of hypoxia-related proteins, which may contribute to the explain the aggressiveness and distinctive biological behavior of GOCs[19]. Further research is needed to address this gap in the literature.

Differential diagnosis

The differential diagnosis includes various cystic lesions, such as dentigerous cysts with GOC-like features (e.g., mucous and ciliary prosoplasia), botryoid odontogenic cysts, and lateral periodontal cysts[20]. Table 1 provides a concise review to differentiate GOC from other lesions based on their unique demographic, clinical, radiographic, and histopathologic features.

The differentiation between GOC and central MEC, especially the low-grade and predominantly cystic variant, remains a diagnostic challenge. Microscopically, GOCs may display multi-compartmentalization or small epithelial islands within the cyst wall, which resemble MEC, suggesting a potential histopathological overlap. MEC typically contains smaller cystic spaces and displays a more prominent pattern of tumor islands

Table 1. Some differential diagnosis to glandular odontogenic cyst based on their demographic, clinical, radiographic, and histopathologic features.

Lesion	Demographic characteristics	Clinical and radiographic features	Histopathologic findings
Glandular odontogenic cyst	- Adults (40-60 years) - No gender predilection	- Anterior mandible - Slowly expanding painless swelling - Well-defined corticated unilocular or multilocular radiolucent lesion of tooth-bearing area of the jaw	- Lining epithelium with cuboidal or columnar cells, microcysts, duct-like structures, eosinophilic material, and mucous cells - Clear cell components and surface cilia may also be observed
Central mucoepidermoid carcinoma	- Middle-aged individuals - Slight female predilection	- Radiolucent lesion, often multilocular, may be associated with pain, swelling, or paresthesia	Presence of mucous, intermediate, and epidermoid cells in solid nests or cords. Infiltrative growth pattern and absence of a cystic lining. Mitoses may be evident
Lateral periodontal cyst	- Adults (40–70 years) - Slight male predilection	- Unilocular radiolucency adjacent to the roots of vital teeth, typically in the mandibular premolar region	Thin epithelial lining with focal plaques or thickenings. No mucous cells, duct-like structures, or microcysts
Botryoid odontogenic cyst	- Adults - No gender predilection	- Multilocular variant of lateral periodontal cyst, more aggressive behavior	Similar to lateral periodontal cyst but with multilocular growth. Epithelial plaques are more prominent, with occasional cystic expansion
Dentigerous cyst	- Young adults - No gender predilection	- Unilocular radiolucency around the crown of an unerupted tooth, typically well-corticated	- Non-keratinized epithelium, typically 2–4 cell layers thick, without mucous cells or microcysts. No duct-like structures or eosinophilic material
Odontogenic keratocyst	- All ages (strong peak in the second or third decade and a second smaller peak in the elderly) - Slightly male predilection	- Posterior mandible and ramus - Unilocular or multilocular radiolucency, well-circumscribed, may cause significant cortical expansion	- Parakeratinized epithelial lining - Palisaded hyperchromatic basal cells
Radicular cyst - Residual cyst	- Wide age range - Slightly male predilection	- Non-vital tooth for radicular cyst - Edentulous area for residual cyst - Radiolucency with well-defined borders	Lining of non-keratinized stratified squamous epithelium with a mixed inflammatory infiltrate. Lacks duct-like structures and mucous cells.
Nasopalatine duct cyst	- Adults (30-60 years) - Male predilection	- Radiolucency in the anterior maxilla near the incisive canal, heart-shaped radiographic appearance	Pseudostratified columnar epithelium with goblet cells and ciliated cells. Often surrounded by fibrous connective tissue containing neurovascular bundles

within a fibrous stroma[21]. Additionally, a variable population of intermediate cells can be observed, a feature absents in GOC[16]. However, a definitive distinction between low-grade CMEC and GOC has yet to be established. Some immunohistochemical findings provide valuable insights. In GOC, cytokeratin (CK) 19 is consistently positive, while CK18 is expressed in only 30% of cases. Conversely, central MEC is CK18 positive, with CK19 expressed in approximately 50% of cases[22]. Another marker described to discriminate between the two lesions was the Maspin for mucous cells, revealing negativity for GOC and positive for MEC[23].

Recent studies have highlighted the utility of molecular genetic testing, specifically for the *CRTC1/3::MAML2* gene fusion transcript, through methods like FISH or RT-PCR, as a reliable approach to distinguish both lesions, in which GOC typically not exhibiting the gene rearrangement, while MEC shows positivity in approximately 33% to 70% of cases[24,25].

Treatment and prognosis

The management of GOC remains a subject of discussion due to its aggressive nature and significant risk of recurrence. Several treatment options are available, ranging from conservative methods such as enucleation and curettage to more aggressive approaches like *en bloc* resection[26,27]. The choice of treatment depends on factors such as the size and location of the lesion. While enucleation and curettage are often considered first-line treatments, the recurrence rate following these methods has been reported to be as high as 30%, necessitating more aggressive approaches in certain cases[28]. *En bloc* resection may be considered for larger lesions or those with signs of recurrence, particularly in areas with limited surgical access. Campos et al. provided a significant reflection in their 2023 case report, highlighting that if a radical treatment approach had been taken, all teeth involved in the lesion would have been lost, leading to further impairments in speech, mastication, and facial aesthetics[27]. Additionally, the patient would have faced considerable morbidity, as resection of this magnitude would have required bone reconstruction through a free graft or microsurgical flap[27]. In this context, alternative and new modalities, such as targeted therapy, emerge as a promising area for further investigation, similar to ongoing research on other odontogenic lesions that

typically demand aggressive treatment, like ameloblastoma[29]. However, advancing these approaches first requires novel molecular studies to elucidate the underlying pathogenesis of GOC.

The literature emphasizes the importance of precise diagnosis and long-term follow-up in managing GOC to effectively monitor and address potential recurrences. The recurrence rates vary from 21% up to 55% cases[18]. In a recent systematic review conducted by Labrador et al.[30], 18 cases of recurrent GOC were evaluated. Their findings revealed that while no single histologic feature or combination of features could reliably predict recurrence, the type of treatment was the strongest correlation[30]. Notably, the recurrence rates were higher between 3- and 5-years post-treatment, suggesting that follow-up should continue for at least five years to ensure early detection and management of recurrences[30]. Although it is possible that intraosseous MEC may arise from GOC, the current evidence suggests that they are separate entities[14].

CONCLUSION

Odontogenic cysts are common lesions of the jaws, which all derive their lining from residues of epithelium involved in tooth development. They are classified into cysts of inflammatory or developmental origin[4]. However, some cysts, such as GOC, exhibit aggressive behavior and a tendency for recurrence, presenting a challenge in both diagnosis and management. The present review highlights the diagnostic complexities, biological behavior, and clinical implications of GOC, with the objective of consolidating current knowledge and guiding future research and clinical management strategies.

In summary, understanding the distinct features of GOC is essential for oral pathologists and surgeons to distinguish it from odontogenic neoplasms that may warrant more radical interventions. Furthermore, its unique histopathological characteristics provide a valuable opportunity to explore the molecular mechanisms involved in cystic development and mucous cell differentiation, contributing to a deeper understanding of odontogenic lesions. Given the rarity of GOC, more cases are needed to better understand its behavior and optimize treatment protocols.

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AUTHORS' CONTRIBUTIONS

LFS: conceptualization, investigation, methodology, writing – original draft. FMS: investigation, writing – review & editing. MNGU: writing – review & editing. AVBC: writing – review & editing. VPP: writing – review & editing. ES: writing – review & editing. RBM: supervision, writing – review & editing.

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