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## Chapter

# Salivary Duct Cyst

*Saurabh R. Nagar, Gabriela Fernandes, Shivani Bansal,  
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## Abstract

Salivary duct cysts (SDCs) are true cysts caused by obstruction of salivary ducts and are rare in minor salivary glands. Intraoral SDCs and mucoceles represent clinically salivary gland neoplasms, making diagnosis difficult and subject to errors in treatment. It is important for Oral and Maxillofacial Surgeons to include SDC in the differential diagnosis of swelling affecting buccal mucosa.

**Keywords:** salivary duct cyst, minor salivary glands, oral pathology

## 1. Introduction

The two major groups of oral cysts are divided based on odontogenesis: odontogenic cysts (OCs) and non-odontogenic cysts (non-OCs). The odontogenic cysts are distinguished by specific odontogenic markers. They have histological similarities with odontogenic structures and anatomical considerations, whereas the non-odontogenic group includes cysts that originate from specific areas or organs of the oral cavity such as naso-palatine duct/mid-palatine cysts, nasolabial cysts, and salivary cysts. There are some cysts that are included in this group, that are ubiquitous in the body such as aneurysmal bone cysts, lympho-epithelial cysts, and dermoid cysts [1].

Odontogenic cysts develop from the tooth-producing tissues and on the other hand, they originate from the remnants of dental lamina epithelium entrapped within the gingival named epithelial rests of “Serres,” or the epithelial remains of the “Malassez.” These cellular remnants have limited growth potential and they fall within the concept of the post-functional state of the dental lamina. The two types of dental cysts are generated by the two types of embryological residues. Periodontal cysts originate from the remnants of Serres and the orthokeratocysts, which are a more aggressive type of cyst with a neoplastic variant. The inflammatory radicular cyst originates from the residues of the Malassez. For this particular type of cyst, an infectious and/or inflammatory stimulus acting on a genetic predisposition has been proposed as the first pathogenic event causing the proliferation of cellular odontogenic remnants.

Cytokeratins are ideal markers for differential diagnosis of these cysts, being involved in physiological odontogenesis epithelium-specific markers of differentiation and have been proposed as ideal markers for differential diagnosis of these cysts, being involved in physiological odontogenesis, thoroughly cytokeratins 5 and 14 are

present in the basal cell layer of keratinized and non-keratinized epithelia along with a depletion in the layers above. Cytokeratins 1 and 10 are particular with respect to the spinous layer; cytokeratin 19 is specific with regard to the basal stratum layer of the non-keratinized epithelia; cytokeratins 13 as well as 4 are particular to the supra-basal cells of the tongue epithelium; there is the presence of K2p in the supra-basal epithelial cells of the hard palate and gingiva. In the course of odontogenesis, the cytokeratins form a unique expression; in the early bell stage, cytokeratin 14 is present in the “stellatum reticulum” along with cytokeratin 7; CK19 is expressed along with these cytokeratins in the cells of the enamel epithelium; although cytokeratin 14 is present

Cyst of oral bone tissue and periodontal	Soft tissue non-odontogenic cysts
Odontogenic cyst <ul style="list-style-type: none"> <li>• Inflammatory origin                             <ul style="list-style-type: none"> <li>Radicular necrotic cyst</li> <li>Collateral inflammatory cyst                                     <ol style="list-style-type: none"> <li>1. Paradental cyst</li> <li>2. Juvenile paradental cyst</li> </ol> </li> </ul> </li> <li>• Development origin                             <ul style="list-style-type: none"> <li>Dentigerous cyst                                     <ol style="list-style-type: none"> <li>1. Follicular cyst</li> <li>2. Germinal cyst</li> <li>3. Eruptive cyst</li> </ol> </li> </ul> </li> <li>• Parodontal cyst (Periodontal tissue)</li> <li>• Gingival cyst of infants                             <ol style="list-style-type: none"> <li>1. Newborn gingival cysts</li> <li>2. Dental lamina cysts</li> <li>3. Bohn’s nodules</li> </ol> </li> </ul> Non Odontogenic cysts <ul style="list-style-type: none"> <li>• Nasopalatine duct/midline palatine cyst</li> <li>• Mid-palatal raphe non-odontogenic cysts of infants (Epstein’s Pearls)</li> </ul>	Salivary cysts and salivary duct cysts Cysts of lymphatic tissue origin <ul style="list-style-type: none"> <li>• Cystic hygroma your name</li> <li>• Lymphoepithelial cysts</li> </ul>
Cysts of globulo-maxillary area	Dermoid and epidermoid congenital cysts
Cysts with malignant variant <ul style="list-style-type: none"> <li>• Orthokeratinized odontogenic cyst</li> <li>• Calcifying odontogenic cyst</li> </ul> Glandular cysts <ol style="list-style-type: none"> <li>1. Glandular odontogenic cyst</li> <li>2. Sialo-odontogenic cyst</li> </ol>	Nasolabial cysts
Cysts of maxillary sinus	Thyroglossal duct cyst
Pseudocysts of the bone in relation to the oral cavity <ul style="list-style-type: none"> <li>• Solitary bone pseudo cyst</li> <li>• Aneurysmal bone cyst</li> </ul>	

**Table 1.**  
Cysts of the oral cavity.

during the early bell stages which are ultimately substituted by cytokeratin 19 in differential ameloblasts; cytokeratins 7 and 13 are present in the “rests of Serres.” The structure of cytokeratins and their expression within the cells are based on the conditions of the environment and changes that occur in the functioning of the cell. Hence, an altered expression when detected becomes extremely helpful for the differential diagnosis of multiple diseases like cysts and tumors. Therefore, cytokeratins 5 and 6 are found in every single layer of the odontogenic cysts, cytokeratin 13 is present among the supra-basal cell layer of odontogenic cysts, while cytokeratin 20 is not present in any of the odontogenic cysts.

In this chapter, we suggest a simplified classification with regard to the cysts of the oral cavity. We plan to divide the various types of cysts into two main groups: (1) cyst of the osseous and periodontal tissue and (2) soft tissue origin non-odontogenic cyst. After which, these groups are further divided into subgroups depending on their relationship with the anatomical area, histological origin as well as clinical behavior (rate of recurrence, frequency, and malignant potential; **Table 1**). **Table 1** depicts the classification of the cysts of the oral cavity.

## 2. Cysts of the oral bone and periodontal tissue

### 2.1 Odontogenic cysts of inflammatory origin

Radicular necrotic cyst (RC) is the most common cyst of the oral cavity which is caused by the loss of pulp of the tooth (biological barrier) due to carious lesions or dental trauma. There is the presence of pulp necrosis and the cyst is derived from the cellular remnants of the “Malassez.” This can preside to form inflammatory radicular necrotic cyst which can either be periradicular or periapical. Initially, the granuloma forms, and after which it gives rise to a cyst whose epithelium demonstrates odontogenic CK19 in the superficial cell layers and co-expresses CK5 in the cyst lining. Residual radicular cysts are a unique variant of the radicular cyst which develops from apical granulomas or residual fragments of RC.

Collateral inflammatory cysts (Juvenile paradental cyst and Paradental cyst) have overlapping histological features with radicular cysts. Their etiology is also considered inflammatory or meta-traumatic. It is present on the lateral surface of a tooth which is vital. It occurs as a result of a chronic inflammatory process in the periodontal pocket. In a young patient, the juvenile paradental cyst is seen in the root area of the mandibular molars whereas it is present distally to a lower wisdom tooth in adults. These lesions are considered the same unit, regardless of the localization. The histological features of these cysts cannot be distinguished from those of the inflammatory radicular cysts but this appearance emphasizes the origin from the remains of the Mallasez.

It becomes important to differentiate these cysts from the other radiolucent jaw lesions, such as unicystic ameloblastoma, keratocystic odontogenic tumor, dentigerous cysts, and LPC. A combination of immunohistochemical markers such as CK10, CK13, CK17, PCNA, UEA, and perlecan can help in the differential diagnosis.

### 2.2 Odontogenic cysts of developmental origin

Dentigerous cysts surround the crown of a tooth that has not migrated into the oral cavity. They are named follicular, germinal, and eruptive cysts. There is an accumulation of pathological fluid in the layers of the reduced enamel epithelium or

between it and the crown of an unerupted tooth. CK5, CK6, and CK19 are present while CK7 is absent in this type of cyst.

Periodontal tissues (parodontal cysts and Botryoid cysts) form a single nosological group named “cyst of the periodontal tissue” because they are of dental origin and the periodontal tissue is contiguous to the teeth and bone. The cysts which affect the periodontal tissue are gingival cysts which are frequently present in adults and periodontal cysts (lateral parodontal cyst and its variant; botryoid cyst). The periodontal cysts are unicystic with a differential diagnosis of ameloblastoma. Two or three layers of flattened cells mimicking a squamous epithelium are present along with areas of nodular type thickening and clear cells rich in glycogen. The lateral parodontal cysts (LPC) have a multilocular variant defined as “Botryoid cyst.” They arise from the remnants of Serres incorporated into the periodontal tissue or from the reduced enamel epithelium of the follicle which expands to occupy a space in the periodontal ligament during the eruptive phase producing a parodontal cyst, while a gingival cyst may form due to a portion remaining in the gum after the eruption. CK13 and CK17 are expressed in the surface layers of the lateral parodontal cyst, whereas perlecan and UEA are present on the cell border of the whole layer. LPC is negative for CK10.

Botryoid odontogenic cyst is a rare pathological multilocular cyst that may or may not have close proximity to a root of a tooth. This is considered a variant of lateral parodontal cyst and it is derived from more groups of converging cellular debris of Serres. It has also been considered to represent a variant of glandular odontogenic cyst due to the presence of mucous cells and the columnar cells. Presence of CK18 and CK13 which is specific for rests of Serres, show the origin from the odontogenic tissues. CD56 and calretinin help in differentiating ameloblastoma from BOC as these markers are absent in the latter.

Gingival cysts of infants are also known as Newborn gingival cysts, Dental lamina cysts, or Bohn’s nodules. They are present in newborns and develop from the remains of the dental lamina. Presence of squamous epithelium lining with areas of parakeratosis and keratin-filled cavities.

### **2.3 Non odontogenic cysts**

Epstein’s Pearl are also known as Mid palatal raphe non-odontogenic cysts of infants. They have similar histological and clinical features to gingival cysts. They arise from non-odontogenic epithelial remnants after median palatal fusion.

Nasopalatine duct cysts are formed by the proliferation of epithelial remnants that are organized in clusters or cords and are present in the incisive canal of the maxilla. These remnants cause the formation of the cyst with the presence of a squamous ciliated epithelium layer.

### **2.4 Cysts of the globulomaxillary area**

These cysts display constant clinical and radiological features, however, they do not present the same histological features always. Various histological features such as stratified squamous (odontogenic), parakeratinic (orthokeratocystic), or cylindrical respiratory (non-odontogenic) epithelium have been described in these cysts. In this group of cysts, we can always include intraosseous cysts that develop between the roots of the lateral incisor and the canine teeth which causes divergence. Moreover, cysts originating from the respiratory epithelium remained trapped in the globule maxillary site and parodontal cyst. It is possible to find neoplastic cysts in this site.

## 2.5 Cysts with malignant variants with neoplastic characteristics

Orthokeratinized odontogenic cysts are characterized by a keratinized lining epithelium. The term keratocystic odontogenic tumor (KCOT) is preferred when the epithelium displays significant parakeratosis or orthokeratosis and presents more aggressively with a tendency to recur. Whereas, these two cysts show a different pattern of expression of CKs: OOC expresses CK1, CK2, CK10, and loricrin, while KCOT expresses CK4, CK10, CK13, CK16, CK17, and CK19, similar to the dental lamina. The differential immunohistochemical expression of CD-56, CD-105, and calretinin help in distinguishing these cysts from ameloblastoma from a clinical view point.

Calcifying odontogenic cyst (COC) have three entities, simple intraosseous COC, extra osseous peripheral COC, and the malignant form calcifying cystic odontogenic tumor. Radiologically, they show cystic imaging with small scattered areas of calcification which often resembles an odontoma. They have a peculiar histological pattern with their epithelial lining consisting of a basal layer of columnar cells and an overlying epithelium, which is thick and vacuolated. Furthermore, groups of eosinophilic cells with non-stainable cellular structures are visible in the epithelial lining, connective tissue capsule, or both. These cells are referred to as “ghost cells” and are considered dystrophic cells with aberrant keratinization or apoptotic cells with intracellular calcification. These particular cells are present in different pathological entities such as a craniopharyngioma, odontoma, pilomatrixoma, ameloblastic fibroma, and some visceral tumors. They lose the cytoskeletal components and become CK10/13 negative as they accumulate some substances during the differentiation process. While, CK14 is expressed in the basal layer, CK10/13 are present in the upper layer of the cyst. P63 expression is present in all layers of COC examined.

Glandular odontogenic cyst (GOC) contain acidophilic cuboidal or columnar cells arranged in glandular structures with papillary growth and projections into cyst-like spaces. This cyst can be differentiated by mucoepidermoid carcinoma based on the diverse immunohistochemical expression of mammary serine protease inhibitor (MASPIN) as well as Ki67 and P63 tumor markers.

Cysts of the maxillary sinus have three types of primary cysts, the first type is the true cysts which are due to an occlusion of the excretory ducts of the sinus mucous glands. The second type is mucoceles which are formed from the non-external drainage of normal mucous and the third type is secondary mucoceles. They are formed as a result of post-radical sinus surgery and probably due to residues of sinus mucosa forming a new mucocele in a closed compartment. Pseudocysts are also present, they are formed between the inner surface of the bone wall and the connective tissue layer while the sinus mucosa remains on the outside. These cysts may be formed due to allergies, inflammation of the maxillary sinus and mucosal odontogenic inflammation. Sometimes, the secondary odontogenic cysts develop in the bone base of maxilla and invade the maxillary sinus. These cysts are particularly “intrusive sinus oral cysts.”

## 3. Pseudocysts present in the oral cavity

Solitary bone pseudocysts (SBP) are devoid of any epithelium lining. They have a traumatic origin hence they are also known as bone pseudocysts or bone traumatic pseudocysts, whereas aneurysmal bone cysts (ABP) are blood-filled sinusoidal or cavernous spaces without cystic epithelium. The pathology of this cyst is similar to ABP. A trauma could lead to a bone hemorrhage and the clot may not be re-canalized

which eventually leaves the cavity devoid of content and may present as continuous micro-hemorrhages. This causes a local reaction of macrophages or vascular dilation. The ubiquitous protease USP-6 which is mapped on chromosoma 16q22 are used as a diagnostic tool for ABPs.

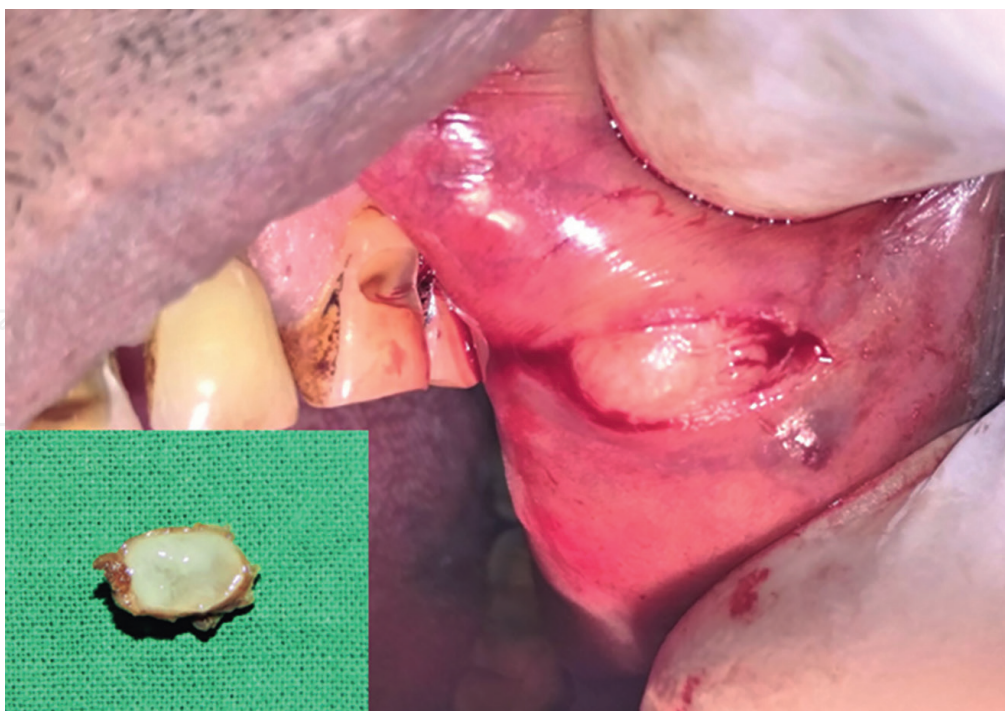
### **3.1 Soft tissue non-odontogenic cysts**

Nasolabial cysts are considered as one of the soft tissue non-odontogenic cysts. The concept that it is considered a fissural cyst is because it is related to the globule-maxillary cyst and its peripheral form is no longer valid. Histologically, it consists of a cyst that is lined by a bi-layered epithelium with a cuboidal basal layer and sometimes pseudo-stratified with goblet cells along with areas of squamous metaplasia. CK7 and CK19 are present in all layers whereas CK5 and CK6 are expressed only in the basal layer. The mucin in the goblet cells is positive for MUC-2 and MUC-5 AC. It is a developmental non-odontogenic cyst that originates from the lower portion of the naso-lacrimal duct.

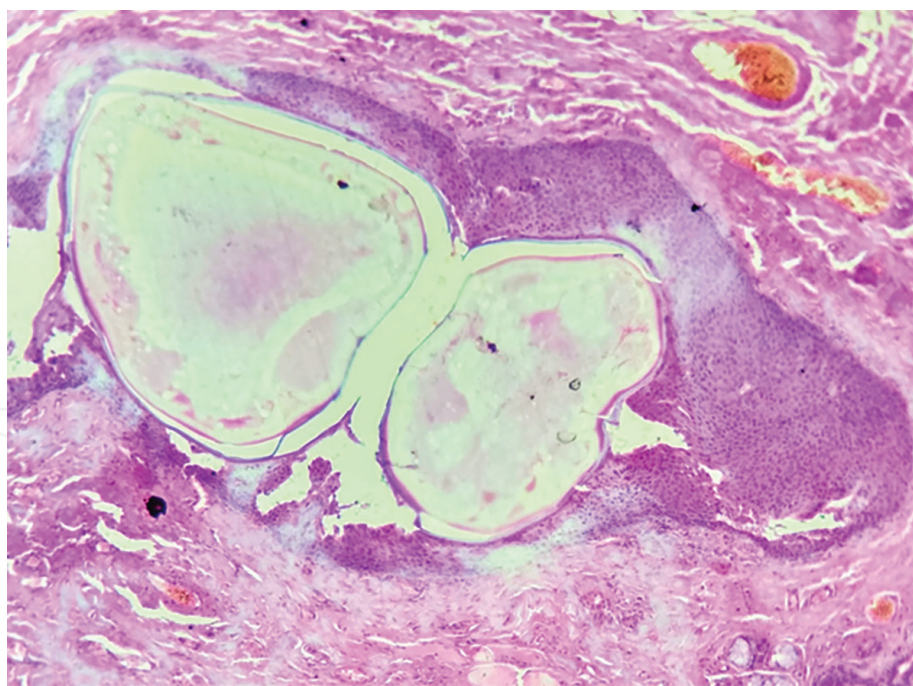
Dermoid and epidermoid congenital cysts are positive for CK10. They are derived from embryonic pluripotential cells trapped which was trapped during the early weeks of intrauterine life and subsequently develop into one or into all three layers—ectoderm, mesoderm, and endoderm. They are present in the floor of the mouth, tongue, parotid gland, and mandible.

Salivary cysts and pseudocysts are soft tissue cysts. While salivary retention cysts are considered as a pseudo-cyst. Histologically, oncocyte-like cells and pseudo-stratified columnar epithelium are present. The Ranula represents a mucocele on the floor of the mouth. It is formed due to salivary accumulation in the sublingual or the submaxillary gland which is followed by a rupture and extravasation of saliva in the surrounding connective. The presence of an epithelial coating is not there. Ranulas are located above the mylohyoid muscle which is known as the simple type or can grow downwards forming an hourglass shape which is known as the complex type.

Salivary duct cysts represent less than 10% of all salivary gland disorders, especially in the major salivary glands. Sialocyst is another name given to the salivary duct cyst (SDC). Salivary gland cysts can be either ephemeral or persistent. It is caused due to ductal obstruction that leads to cystic dilation of salivary ducts. Different terminologies have been given to these lesions; although, “salivary duct cyst (SDC)” is the most adequate by virtue of its origin being related to the epithelial lining of salivary gland ducts. SDCs typically occur in the major salivary glands with 80% occurring in the parotid and minimal cases are reported in the submandibular and sublingual glands. They present as dome-shaped, sessile, slow-growing, unilateral, asymptomatic, and compressible nodules in adult patients [2]. They are fluctuant to palpation but usually painless and if a sialolith is present then it may feel firmer. Secondary infection may present as mucus or pus from dilated ductal orifices when palpated. Blue tinge due to the Tyndall effect is present. They do not typically wax or wane over time, whereas its occurrence in minor salivary glands is rare and occurs more commonly in the sixth decade of life. Symptoms generally involve eating there was no pain or increase in the size of swelling. Intra-oral examination depicts a painless, mobile, non-compressible, soft, nodular, solitary swelling along with a soft brownish cystic sac filled with slimy gel-like material (**Figure 1**). Majority of the swellings range from 1 to 3 cm. Hematoxylin & Eosin (H&E) stained sections demonstrate a dilated salivary gland duct with intraluminal mucous plug (**Figures 2 and 3**) with dense chronic inflammatory infiltrate composed chiefly of lymphocytes around the dilated duct [3, 4]. Salivary



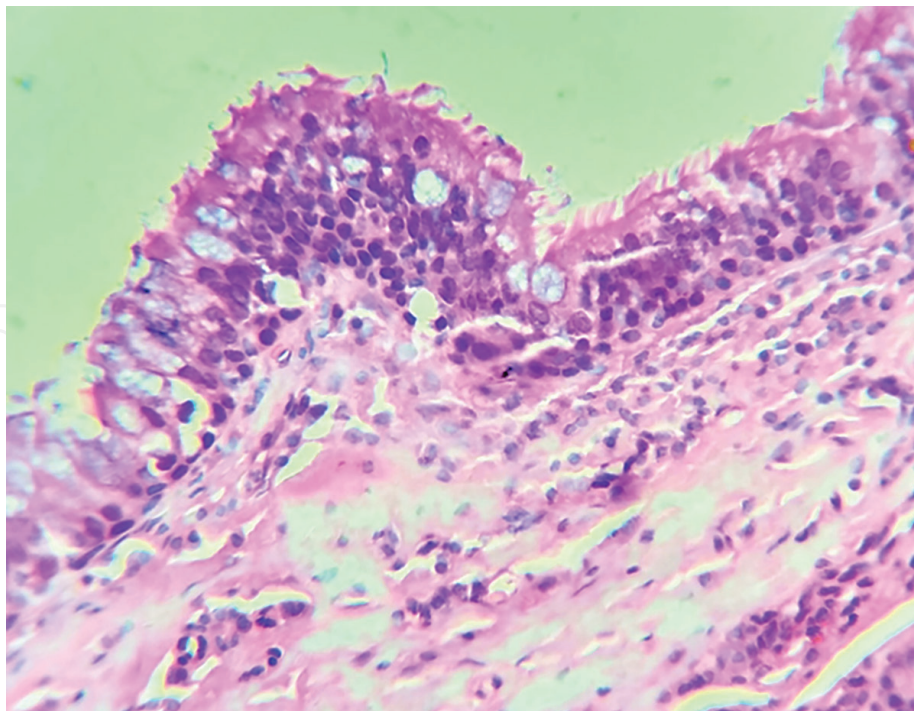
**Figure 1.** Clinical image shows surgical exposure of salivary duct cyst in the left buccal mucosa and gross specimen showing cystic sac filled with slimy gel-like material (inset).



**Figure 2.** Histopathological image showing cystically dilated salivary gland duct with intraluminal mucous plug and squamous metaplasia of the lining epithelium (H&E stain; ×100 magnification).

duct cysts may be congenital or acquired in origin. However, studies show that the vast majority of the cases are acquired and occur following obstruction in the duct. The exact factors that cause the obstruction are usually unknown, some suggestions include the involvement of mucus plugs, calculi, or postoperative or postinflammatory structures. Salivary secretion reduces with an increase in the age which may lead to





**Figure 3.** Histopathological image revealed cystic lining exhibiting ciliated, mucous, and oncocytoid metaplasia (H&E stain;  $\times 400$  magnification).

the formation of a mucous plug. This has detrimental effects as it blocks the salivary gland ducts that eventually result in dilation of the duct and the intraluminal pressure increases. Another study suggests that restriction of the duct is frequently reported to be associated with mouth wash which includes hydrogen peroxide, fragrant mouth washes, and toothpaste that control tartar accumulation. Luminal pressure marginally increases due to a continuous flow of saliva as a result of recreation. This invariably leads to ductal dilation.

Cysts in the salivary ducts often present themselves as an asymptomatic, unilateral swelling that usually occurs in sites that are less prone to trauma. They are evenly distributed between the contiguous area of the buccal mucosa, lower lip mucosa, mandibular vestibule, the floor of the mouth, hard and soft palate, as well as minor salivary glands with a size range of 0.8–10 cm in size [5]. They are rarely present in the major salivary glands and if involved, are usually found in the superficial lobe of the parotid with no involvement of the facial nerve. Salivary duct cysts may affect children to older adults, mostly those over 30–40 years of age. SDCs affect the male and female population equally.

Salivary gland cysts can often be an early manifestation of a salivary gland tumor, therefore diagnosis and early treatment interventions play an important role in the prognosis of this lesion. Presence of epithelial alterations, such as metaplasias and focal papillary proliferations observed, are comparable to similar changes seen in odontogenic cysts and maybe an early markers of tumor manifestation [6]. On rare occasions, these cysts can progress and develop into benign and malignant neoplasms such as adenocarcinoma and mucoepidermoid carcinoma from the lining of a salivary duct cyst.

Primary diagnostic interventions involve imaging and histopathology. Imaging helps with determining the extent of the involvement, the borders, and the core content. Internal blood flow is absent in color Doppler while CT reports the cyst as a well-circumscribed lesion with low-density areas. On MRI, these lesions appear as

high signal areas and no enhancement is observed upon administration of gadolinium while ultrasonography demonstrates a posterior acoustic enhancement lesion with imperceptible walls. Finally, sialography permits only indirect visualization of the cyst, evaluated by the displacement of the ducts around them [7].

Histologically, these lesions appear unilocular along with a ductal epithelium that may be cuboidal or columnar in conjunction with a completely, or partially lined squamous epithelium. Occasionally, oncocytic metaplasia is present. The lesions composed of oncocytic cells range from oncocytic metaplasia, and hyperplasia to benign and malignant neoplasms, including oncocytomas and oncocytic carcinomas [8].

SDCs may present clinical-pathological characteristics similar to those of salivary gland neoplasms, making diagnosis difficult and subject to errors in treatment. Pleomorphic adenoma, cystadenoma, and low-grade mucoepidermoid carcinoma may present in a similar manner to that of SDCs. Salivary duct cysts comprise 0.5–10% of all salivary gland cysts and ideally arise from the salivary duct as non-neoplastic lesions and a majority of these lesions are between 1 and 3 cm in dimension. Since these cysts demonstrate an epithelial cystic lining histopathologically, they are considered to be a “true cyst.” These cysts tend to develop post-obstruction because of the calculi, mucus plugs, trauma, or post-inflammatory scarring. In pediatric patients, the most common pathology lesions involving the parotid gland are benign neoplasms that are not limited to sialoceles, lymphoepithelial cyst, first branchial cleft cyst (BCC), and a vascular or lymphatic malformation. Salivary duct cysts can be easily histopathologically differentiated from branchial cleft cysts by the presence of their epithelial lining since the former have epithelium lining of duct cells similar to intercalated, striated, or excretory duct cells whereas the latter is lined with squamous or respiratory epithelium. SDCs are extremely rare and should not be overlooked as part of the differential for any cystic salivary gland lesion since they easily and very quickly progress into a larger lesion in a few months. Sometimes, they might resemble a lymphatic malformation such as chyle-filled cysts lined with endothelium that can be macrocystic or microcystic. And these lesions can appear identical on radiographs and both display high signal intensity on T2-weighted MRI. However, these can be distinguished via MRI and STIR images since SDCs tend to be well-circumscribed masses while lymphatic malformations can be infiltrative and permeate across fat planes [9].

One of the differential diagnoses for SDCs is papillary cystadenoma lymphomatosum (Warthin Tumor). In this, there is the presence of lymphoid stroma in the cyst wall and multiple papillary infoldings with a bilayer of columnar and oncocytic epithelial lining. The second differential diagnosis is gingival cyst of the adult. These transpire only on the gingiva which has no minor salivary glands. The third differential diagnosis is cystadenoma, low-grade mucoepidermoid carcinoma (LG-MEC). Cystadenoma presents often as a well-circumscribed or encapsulated neoplasm together with a collection of the proliferation of ducts. These cysts often occur in the parotid gland and their histological variants include papillary cystadenoma, papillary oncocytic cystadenoma, and papillary mucinous cystadenoma. SDCs may present clinical-pathological characteristics similar to those of mucocele. Clinically, both represents an asymptomatic nodule, but salivary duct cysts are rare in appearance comparing mucocele. In the latter, the presence of true salivary gland duct epithelial lining is absent and instead, this is mimicked by epithelioid macrophages at the periphery of the extravasated mucin. Mucocele is present in the lower lip mucosa and a younger age group contrary to SDCs.

The treatment option for salivary duct cysts includes cryosurgery, carbon dioxide laser surgery, and conservative surgical excision. Because of the fact that benign

tumors of the salivary glands are able to clinically mimic salivary duct cysts, excision is extremely necessary. Complete surgical excision along with the feeding minor salivary gland is curative. In this method, iatrogenic intraoperative damage may occur to the neighboring salivary gland parenchyma which would contribute to the development of postoperative mucocele. Then partial or total removal of the feeding major salivary glands may be needed. Chlorhexidine mouthwash and oral antibiotics are provided for secondarily infected SDCs. Sialagogues are also provided which may help in decreasing the risk of salivary stasis within dilated ducts by stimulating salivary flow.

In conclusion, intraoral salivary duct cysts are reactive ductal ectasia that develops secondary to intraluminal obstruction that may require clinical attention when it reaches a particular size. Although the SDCs is considered a rare condition in the oral cavity, it is important to include this lesion in the differential diagnosis of lesions that affect the buccal mucosa and may sometimes transform into a malignant lesion such as adenocarcinoma and mucoepidermoid carcinoma from the lining of a salivary duct cyst along with an association of a latent Epstein–Barr virus infection. With an increase in the aging population, SDC should be considered as one of the differential diagnoses in geriatric patients.

### **Conflict of interest**

The authors declare no conflict of interest.

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