

Krasnoyarsk State Medical University Named After Professor V.F. Voino-Yasenetsky Department of Pathological Anatomy Named After Professor P.G.Podzolkov

Lecture 7. Contents:

Part 1.

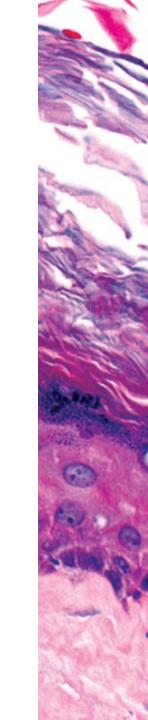
Periodontitis
Parodont
Gingivitis
Medicinal gingival hyperplasia
Gingval fibromatosis

Part 2.

Odontogenic cyst
Follicular cyst (dentigerous cyst)
Odontogenic keratocyst
Lateral periodontal cyst and botryoid odontogenic cyst
Gingival cysts
Glandular odontogenic cyst
Calcifying odontogenic cyst
Orthokeratinized odontogenic cyst

Lector: Vladimir A. Khorzhevskii

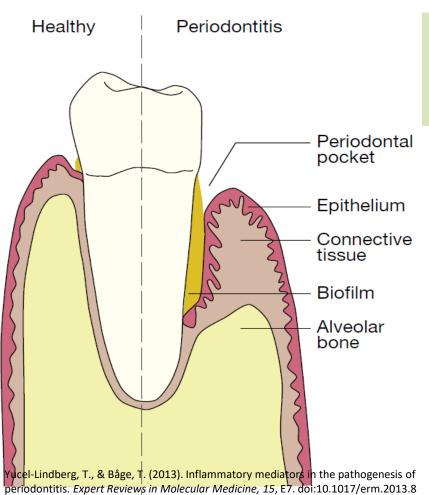
Candidate of Medical Sciences (PhD), Head of Pathology Department named after professor P.G. Podzolkov





Periodontitis

Periodontitis is an inflammatory process that affects the supporting structures of the teeth (periodontal ligaments) alveolar bone, and cementum.



Healthy periodontal tissue (left) and periodontitis (right). Periodontitis is characterised by degradation of the soft connective tissue and alveolar bone supporting the tooth, ultimately resulting in tooth loss.

Although 300 types of bacteria reside in the oral cavity, adult periodontitis is associated primarily with:

- 1. Aggregatibacter actinomycetemcomitans,
- 2. Porphyromonas gingivalis,
- 3. Prevotella intermedia.

(Actinobacillus)



Staging and Grading Periodontitis



The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions resulted in a new classification of periodontitis characterized by a multidimensional staging and grading system. The charts below provide an overview. Please visit **perio.org/2017wwdc** for the complete suite of reviews, case definition papers, and consensus reports.

· Localized (<30% of teeth involved);

Generalized; orMolar/incisor pattern

PERIODONTITIS: STAGING

Extent and

distribution

descriptor

Staging intends to classify the severity and extent of a patient's disease based on the measurable amount of destroyed and/or damaged tissue as a result of periodontitis and to assess the specific factors that may attribute to the complexity of long-term case management.

Initial stage should be determined using clinical attachment loss (CAL). If CAL is not available, radiographic bone loss (RBL) should be used. Tooth loss due to periodontitis may modify stage definition. One or more complexity factors may shift the stage to a higher level. See **perio.org/2017wwdc** for additional information.

	Periodontitis	Stage I	Stage II	Stage III	Stage IV	
Severity	Interdental CAL (at site of greatest loss)	1 – 2 mm	3 – 4 mm	≥5 mm	≥5 mm	
	RBL	Coronal third (<15%)	Coronal third (15% - 33%)	Extending to middle third of root and beyond	Extending to middle third of root and beyond	
	Tooth loss (due to periodontitis)	No tooth loss		≤4 teeth	≥5 teeth	
Complexity	Local	Max. probing depth ≤4 mm Mostly horizontal bone loss	 Max. probing depth ≤5 mm Mostly horizontal bone loss 	In addition to Stage II complexity: • Probing depths ≥6 mm • Vertical bone loss ≥3 mm • Furcation involvement Class II or III • Moderate ridge defects	In addition to Stage III complexity: Need for complex rehabilitation due to: Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defects Bite collapse, drifting, flaring < 20 remaining teeth (10 opposing pairs)	
	Add to stage as	For each stage, describe extent as:				





PERIODONTITIS: GRADING

Grading aims to indicate the rate of periodontitis progression, responsiveness to standard therapy, and potential impact on systemic health.

Clinicians should initially assume grade B disease and seek specific evidence to shift to grade A or C. See **perio.org/2017wwdc** for additional information.

	Progression		Grade A: Slow rate	Grade B: Moderate rate	Grade C: Rapid rate
Primary criteria	Direct evidence of progression	Radiographic bone loss or CAL	No loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
Whenever available, direct evidence should be used.	Indirect evidence of progression	% bone loss / age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectations given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease
Grade modifiers	Risk factors	Smoking	Non-smoker	<10 cigarettes/day	≥10 cigarettes/day
		Diabetes	Normoglycemic/no diagnosis of diabetes	HbA1c <7.0% in patients with diabetes	HbA1c ≥7.0% in patients with diabetes

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions was co-presented by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP).



CLASSIFICATION OF PERIODONTITIS according to I.G. Lukomsky

- 1. Acute periodontitis
- 2. Chronic periodontitis:
 - 2.1. Chronic fibrous periodontitis
 - 2.2. Chronic granulating periodontitis
 - 2.3. Chronic granulomatous periodontitis, or granuloma
- 3. Chronic periodontitis in the acute stage



Acute periodontitis

The phase of acute purulent inflammation is characterized by an increase in leukocyte infiltration, and upon microscopic examination, along with the usual signs of acute inflammation, abundant tissue infiltration with neutrophils can be observed; increasing infiltration of leukocytes leads to tissue melting and the formation of an abscess.



Complications:

collateral edema of the peri-maxillary tissues formation of an abscess formation of a fistula on the gums or skin of the face



Chronic fibrous periodontitis

In chronic fibrous periodontitis, there is a decrease in the number of cellular elements and an increase in coarse fibrous fibrous tissue.



The disordered arrangement of coarse-fibrous structures in the periodontium gives it the character of scar tissue and is accompanied by a diffuse thickening of the apical portion of the periodontium.



Chronic granulating periodontitis

is characterized by the formation of granulation tissue, which contains a large number of capillaries, fibroblasts, round plasma cells, lymphocytes and histiocytes.

Accompanied by destruction of peri-apical tissues with resorption of osteoclasts not only of the compact plate of the alveoli, but also of cement, and in some cases of dentin of the tooth root.

Granulations grow into the bone marrow spaces of the jaw, forming fistulas with purulent discharge, and sometimes subgingival or subcutaneous granuloma.



Intraoral Periapical Radiographs showing Periapical Granuloma

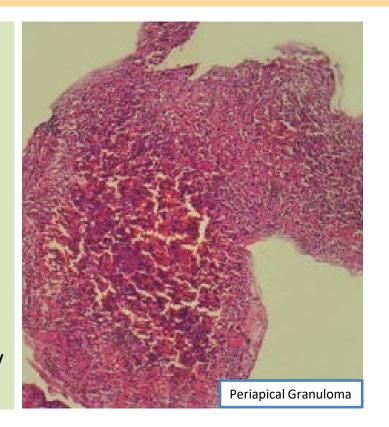


Chronic granulomatous periodontitis (granuloma)

- is a more stable and less active form than granulating, since inflammatory edema and inflammatory hyperemia in this form of inflammation are replaced by proliferative processes.

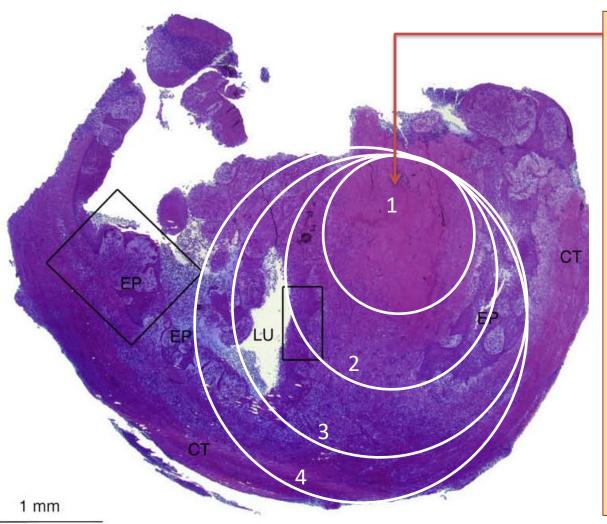
Histologic Features

- Fibrous tissue
- Periapical cyst
- Stratified squamous epithelial lining
- Inflammation is variable
- Chronic and acute: Lymphocytes, plasma cells,
- multinucleated giant cells, histocytes, eosinophils, and neutrophils
- Dystrophic calcifications
- Cholesterol clefts
- Foreign material if previously endodontically treated





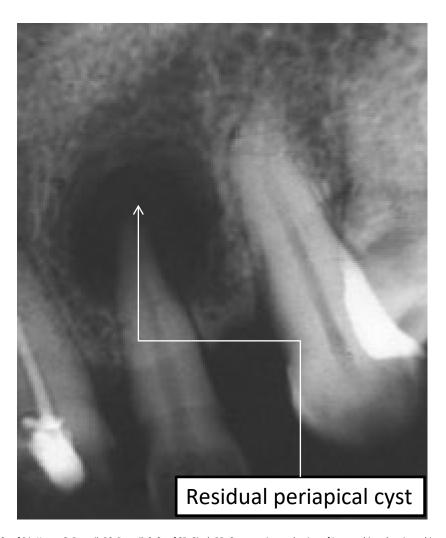
In a mature granuloma, there are several zones that represent a single type of body defense against infection from the root canal

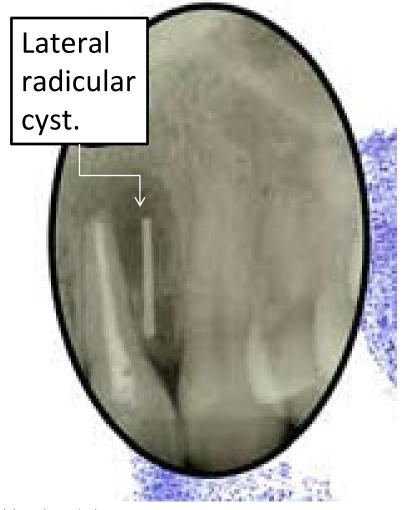


- 1. Zone of necrosis. This area contains necrotic tissue and bacteria.
- 2. Zone of contamination.
 This zone contains
 leukocytes, lymphocytes
 and osteoclasts.
- 3. Zone of irritation. The zone contains granulation tissue; there are no living microorganisms in this zone.
- 4. Zone of stimulation. The zone is characterized by the activity of osteo- and fibroblasts that create collagen fibers.



PERIAPICAL (RADICULAR) CYST





Saraf PA, Kamat S, Puranik RS, Puranik S, Saraf SP, Singh BP. Comparative evaluation of immunohistochemistry, histopathology and conventional radiography in differentiating periapical lesions. *J Conserv Dent*. 2014;17(2):164-168. doi:10.4103/0972-0707.128061



3 phases of the pathogenesis of the periapical cyst:

1

• The inflammatory response in the periapical region stimulates the proliferation of cells in the islets of Malasse.

7

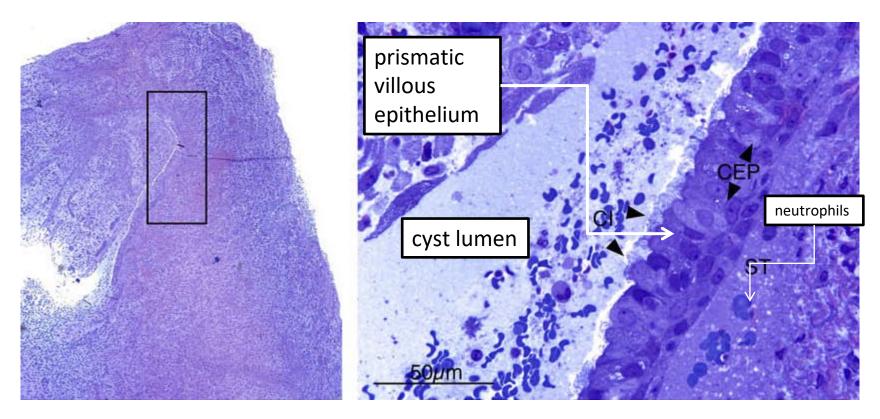
 Formation of the cavity begins with hydropic degeneration and necrosis of epithelial cells located in the center of the islets of Malasse due to malnutrition. Neutrophils enter the areas of necrosis, multiple small cavities are formed, which, merging, form one cavity filled with liquid.

2

• An increase in the concentration of various molecules in the formed cavity leads to an increase in the osmotic pressure in it. Under the influence of high pressure and the activity of macrophages and lymphocytes, bone resorption can be activated, which can contribute to a progressive increase in cavity formation



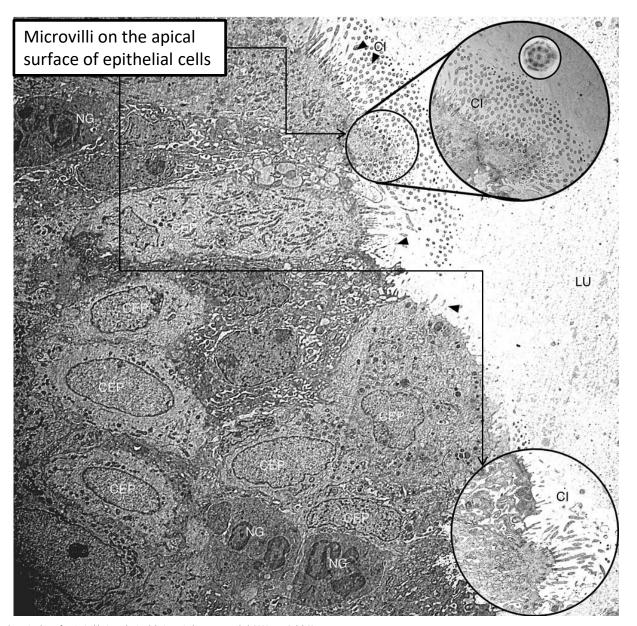
Morphologic structure of a periapical cyst (radicular cyst)



The epithelial lining formed by the prismatic villous epithelium is determined. In the environment of the epithelium, inflammatory infiltration is determined, which contains neutrophils.



electron microscopy



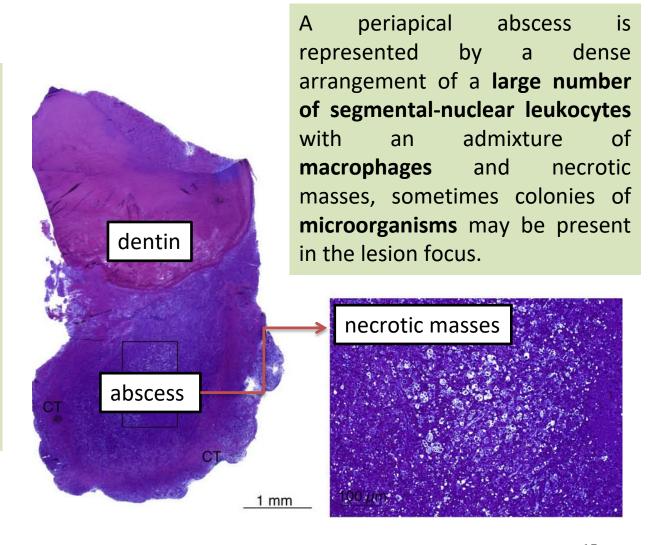


Periapical abscess

- is an acute focal purulent inflammation in the apex of a non-viable tooth

In most cases, the **source** of the infection is obvious, as a rule, it is **pulpitis**, however, cases of the development of a periapical abscess in the area of a healthy tooth have been described.

In addition to the above, a periapical abscess can occur as a result of trauma and therapeutic manipulations affecting the root canals.





Complications of a periapical abscess

- Sinusitis (most often maxillary).
- Osteitis, periostitis, osteomyelitis.
- Cellulite, phlegmon of the face and neck.
- Ludwig's angina.
- Thrombosis of the cavernous sinus.
- Sepsis.

PARODONT

 is a complex of tissues surrounding the tooth, which ensure the fixation of the tooth in the jaw and its functioning.



Gingivitis

inflammation limited to the soft tissue that surrounds the tooth.

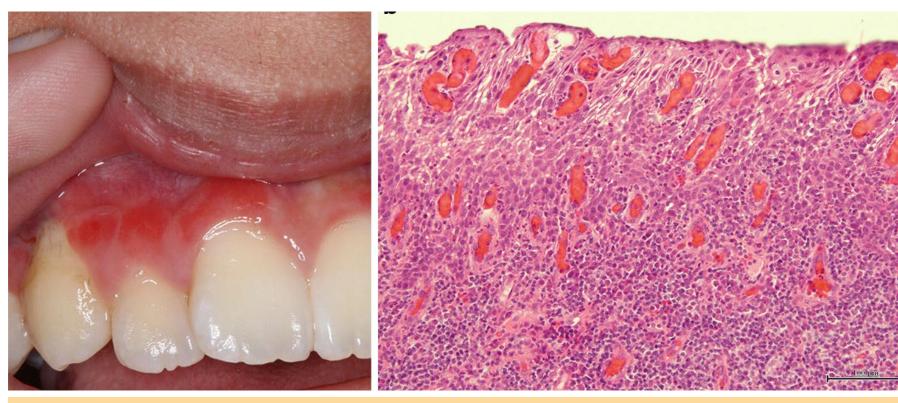
- Gingivitis associated with the formation of microbial plaque
- Allergic gingivitis
- Gingivitis due to drug exposure
- Gingivitis due to exposure to infectious agents
- Necrotizing ulcerative gingivitis
- Abscess of the gums
- Gingivitis, as a manifestation of dermatological, systemic and other diseases
- Gingivitis of pregnant women

MORPHOLOGY

Gingivitis in the initial stages is characterized by a **mild infiltration by polymorphonuclear leukocytes**, which are localized subepithelially. **As the progression** progresses, the **infiltration becomes more pronounced**, in the infiltrate, along with polymorphonuclear leukocytes, plasma cells and lymphocytes are determined. Areas of fibrosis, edema and hyperemia may be present in varying degrees of severity



Acute gingivitis



- -redness, moderate or severe swelling, and soreness of the gums.
- -Against this background, bleeding or even ulceration of the gums may appear.

MORPHOLOGY:

In the inflammatory infiltrate, along with plasma cells and lymphocytes, polymorphonuclear leukocytes are present.



Chronic gingivitis



Local factors play the greatest role in the development of chronic gingivitis - dental plaque, dental stones, etc.

Microscopically, there is a weak or moderate infiltration by plasma cells, lymphocytes and macrophages.

Chronic gingivitis is the condition that the oral health educator (OHE) will probably encounter most frequently. It is thought to affect 50–90% of the adult population, and can be present in children.



Necrotizing ulcerative gingivitis

• is an acute non-contagious infectious lesion of the gums, characterized by a sudden onset, the formation of ulcers in the area of the gingival papillae, bleeding and soreness in the lesion.



J Can Dent Assoc 2013;79:d44

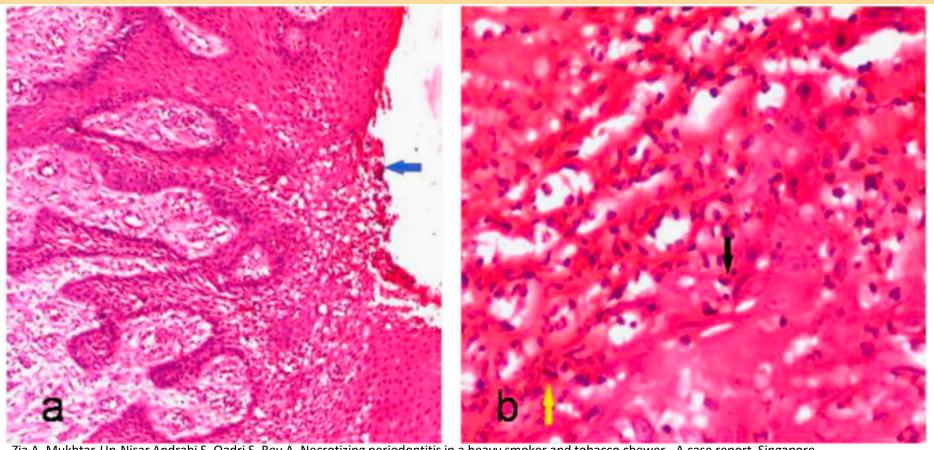
Characteristic microbiota:

- fusiform bacteria,
- Prevotella intermedia,
- Porphyromonas gingivalis,
- •Candida albicans (increased prevalence in HIV-positive patients)



Pathological features

are nonspecific, surface defects are determined on the gingival papillae, the bottom of which is covered with fibrinous-purulent exudate, the underlying lamina propria is hyperemic, infiltrated with lymphocytes, plasma cells and segmental-nuclear leukocytes.



Zia A, Mukhtar-Un-Nisar Andrabi S, Qadri S, Bey A. Necrotizing periodontitis in a heavy smoker and tobacco chewer - A case report. Singapore Dent J. 2015 Dec;36:35-8. doi: 10.1016/j.sdj.2015.07.001. PMID: 26684494.



Atypical gingivostomatitis (plasmacytic gingivitis)



Etiologically, the disease is usually allergic in nature.

Numerous cases of the development of this variant of gingivitis have been described as a response to the use of chewing gum, herbal toothpastes, etc.

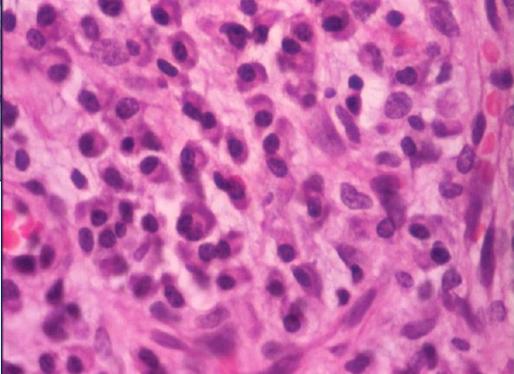
In some cases, the etiology of the disease cannot be established.

The onset of the disease is acute, characterized by soreness in the gums and hard palate. Increased pain is characteristic when using toothpaste, hot and spicy foods. Lesions of the mucous membrane of the hard palate are usually less pronounced.



Atypical gingivostomatitis (plasmacytic gingivitis)

A distinctive feature of the disease is a pasmocytic infiltrate.



Joshi C, Shukla P. Plasma cell gingivitis. *J Indian Soc Periodontol*. 2015;19(2):221-223. doi:10.4103/0972-124X.145830



Granulomatous gingivitis

It is characterized by the development of granulomatous inflammation in the gum region.





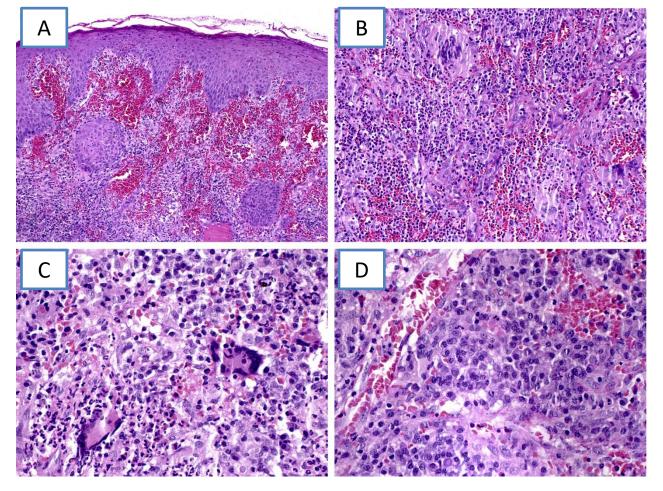
Fonseca, F.P., Benites, B.M., Ferrari, A.L.V., Sachetto, Z., de Campos, G.V., de Almeida, O.P., Fregnani, E.R. (2017) Gingival granulomatosis with polyangiitis (Wegener's granulomatosis) as a primary manifestation of the disease. *Australian Dental Journal* doi: 10.1111/adj.12441

- can be a manifestation of specific infectious granulomatous diseases (tuberculosis, syphilis, mycoses), systemic granulomatous diseases (Crohn's disease, sarcoidosis, Wegener's granulomatosis), develop when foreign bodies enter the mucosa, in particular during dental manipulations.

There is a variant of granulomatous gingivitis as an independent form, which can be established only after excluding all the above diseases - nonspecific granulomatous gingivitis.



Gingival granulomatosis with polyangiitis (Wegener's granulomatosis) as a primary manifestation of the disease



Fonseca, F.P., Benites, B.M., Ferrari, A.L.V., Sachetto, Z., de Campos, G.V., de Almeida, O.P., Fregnani, E.R. (2017) Gingival granulomatosis with polyangiitis (Wegener's granulomatosis) as a primary manifestation of the disease. *Australian Dental Journal* doi: 10.1111/adj.12441

The lesion demonstrated a mixed inflammatory infiltrate with lymphocytes, macrophages and multinucleated giant cells that were randomly distributed without well-organized granulomas (HE, ×200). (C) Higher magnification with multinucleated giant cells, plasma cells and a small neutrophilic abscess (HE, ×400). (D) Plasma cells predominated in some areas.



Desquamative gingivitis



Lesion of the gums, characterized by pronounced desquamation of the epithelium, as a result of which some areas of the gums acquire a bright red (polished) appearance.

The microscopic picture of desquamative gingivitis is determined by the disease, of which it is a manifestation.

Tofan EC, Părlătescu I, Ţovaru Ş, Nicolae C, Preda AS, Funieru C. Desquamative Gingivitis - A Clinicopathological Review. *Curr Health Sci J.* 2018;44(4):331-336. doi:10.12865/CHSJ.44.04.01

Most patients have diseases such as lichen planus and various forms of pemphigoid skin lesions. A relationship was found between the development of the presented pathology in individuals with an increased level of estrogen

Usually being a manifestation of a particular disease, cannot be considered an independent disease.



Medicinal gingival hyperplasia

It is an abnormal increase in gum tissue in response to the use of a particular drug.



Rev. Clin. Periodoncia Implantol. Rehabil. Oral vol.12 no.1 Santiago mar. 2019 http://dx.doi.org/10.4067/S0719-01072019000100050

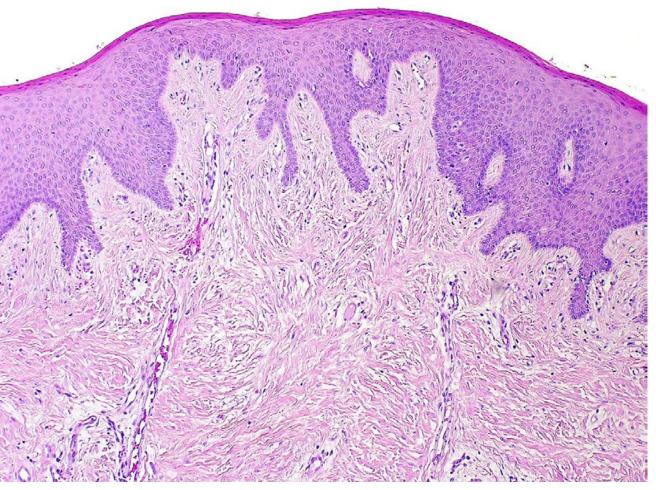
the enlargement of the gums is a consequence of the proliferation of the components of the extracellular matrix, and not the proliferation of cells.

Among the drugs that can cause gingival hyperplasia are anticonvulsants (carbamazepine, sodium valproate, phenytoin, etc.), calcium channel blockers (verapamil, nifedipine, diltiazem, etc.), oral cyclosporins, erythromycin, contraceptives.

There is a direct relationship between the severity of gingival hyperplasia and poor oral hygiene. 28



Histopathology



Kharazmi M, Carlsson AP, Hallberg P, Modig M, Björnstad L, Hirsch JM. Surgical approach to snus-induced injury of the oral mucosa. J Oral Sci. 2014 Mar;56(1):91-4. doi: 10.2334/josnusd.56.91. PMID: 24739713.

The histopathology of the lesions in all drug categories is similar and is characterized by excessive accumulation of extracellular matrix proteins such as collagen or amorphous ground substance. Varying degrees of inflammatory infiltrate exist, while an increase in the number of fibroblasts remains controversial.

The predominant type of infiltrating inflammatory cell is the plasma cell.



GINGVAL FIBROMATOSIS

Slowly progressive enlargement of the gums of a non-neoplastic nature due to the proliferation of the fibrous component of the connective tissue.



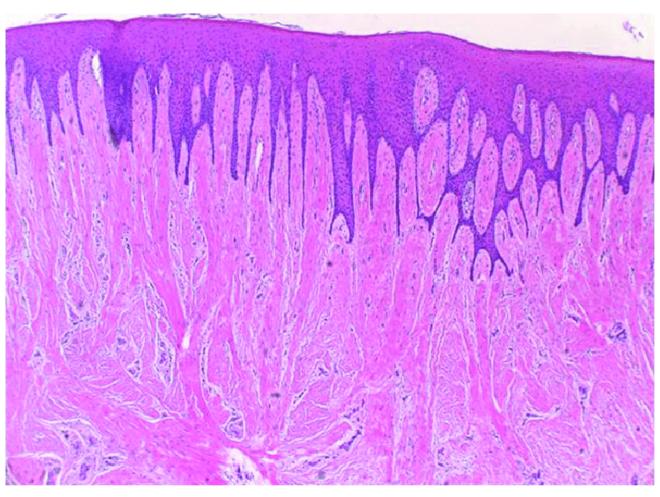
Hwang, Jihye & Kim, You-Lee & Kang, Seyoung & Kim, Sanguk & Kim, Seong Oh & Lee, Jae & Han, dong-hoo. (2016). Genetic analysis of hereditary gingival fibromatosis using whole exome sequencing and bioinformatics. Oral diseases. 23. 10.1111/odi.12583.

Gingival fibromatosis can be familial (hereditary) and idiopathic.
The defeat of the gums is characterized by diffuse or local enlargement of the gums, the consistency of which is dense, the mucous membrane is weakly mobile.

Sometimes the enlargement of the gums is pronounced to such an extent that they completely cover the crown of the tooth and prevent the lips from closing.

The gum in the affected area becomes dense, has a characteristic color, the surface can be smooth, granular, in some cases papillary outgrowths can form.

Histopathology



Hwang, Jihye & Kim, You-Lee & Kang, Seyoung & Kim, Sanguk & Kim, Seong Oh & Lee, Jae & Han, dong-hoo. (2016). Genetic analysis of hereditary gingival fibromatosis using whole exome sequencing and bioinformatics. Oral diseases. 23. 10.1111/odi.12583.

The affected gum is represented by dense connective tissue with low vascular and cellular components.

The superficial epithelium can form areas of deep immersion in the underlying fibrous base.
Signs of inflammation are either absent or mild.



Parodontitis

is an inflammation of the gum tissue, which is associated with the loss of the bone part of the periodontium and partial loss of the connective function of the periodontal ligament.

- characteristic feature of periodontitis is the formation of a periodontal pocket as a result of bone and periodontal destruction, as well as the migration of the cervical epithelium in the apical direction
- Microbial agents associated with chronic Parodontitis: Actinobacillus actinomycetemcomitans. Bacteroides, Porphyromonas gingivalis. and Prevotella intermedia.
- According to modern concepts, the pathogenesis of Parodontitis is based on two key conditions - polymicrobial synergy and dysbiosis. Factors secreted by cells of the inflammatory series (interleukins, TNF, prostaglandins) activate osteoclasts, which carry out bone resorption.
- Matrix metalloproteinase, activation, which stimulates the inflammatory response, is of great importance in the pathogenesis of periodontitis.



ODONTOGENIC CYST

A cyst is a closed cavity formation, as a rule, having its own wall; the lumen of the formation may contain liquid, gas, semi-solid substances, etc.

Jaw cysts are classified as odontogenic and non-odontogenic. The odontogenic epithelium takes part in the formation of an odontogenic cyst.

CLASSIFICATION OF JAW CYSTSODONTOGENIC CYST

- 1. Dysontogenetic cysts
- Follicular cyst
- Eruption cyst
- Gingival cyst of newborns
- Gingival cyst of adults
- Lateral periodontal cyst
- Calcified odontogenic cyst
- Glandular odontogenic cyst

2. Inflammatory cysts

- Periapical (radicular) cyst
- Residual periapical (radicular) cyst

NEODONTOGENIC CYST

- Simple cysts of bones
- Aneurysmal bone cysts



Follicular cyst (Dentigerous cyst)

Developmental odontogenic cyst that originates by separation of dental follicle from around the crown of an unerupted tooth.

Diagnosis requires correlation with radiographs or knowledge of radiographic findings.

Epidemiology

Second most common odontogenic cyst Most common developmental odontogenic cyst Multiple simultaneous dentigerous cysts uncommon

Represents 20% of epithelium lined jaw cysts Usually seen in teenagers / young adults, although can occur over a wide age range

Etiology

In normal tooth development, tooth enamel is produced by the enamel organ, an ectodermally derived specialized epithelium. After enamel formation is complete, the enamel organ epithelium atrophies. This reduced enamel epithelium eventually merges with the overlying mucosal epithelium to form the initial gingival crevicular epithelium of the newly erupted tooth. Dentigerous cysts form when fluid accumulates between the reduced enamel epithelium and the crown of the unerupted tooth.

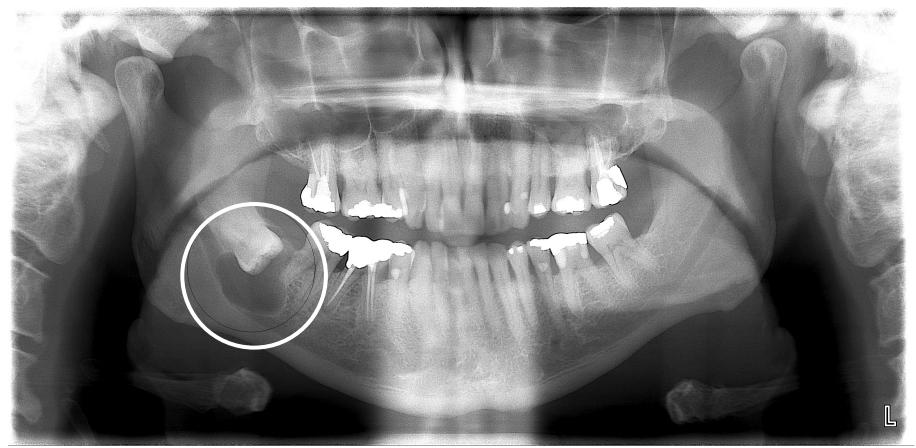


Sanjai K, Kumarswamy J, Kumar VK, Patil A. Florid cemento osseous dysplasia in association with dentigerous cyst. *J Oral Maxillofac Pathol*. 2010;14(2):63-68. doi:10.4103/0973-029X.72503



Radiology description

- Most commonly a well defined, unilocular radiolucency on X-ray
- Often has sclerotic rim
- Can cause resorption of adjacent teeth



DUNCROFT RADIOLOGY
Digital Diagnostic Imaging for Dentists
https://duncroftradiology.wordpress.com/



Histopathology





Odontogenic keratocyst (Keratocystic Odontogenic Tumor)

Definition

Odontogenic keratocyst (OKC) is an odontogenic cyst characterized by a thin, regular lining of parakeratinized stratified squamous epithelium with palisading hyperchromatic basal cells.



https://tumourclassification.iarc.who.int/chaptercontent/42/242

OKCs are most frequently (in 80% of cases) found in the mandible, with as many as half of all lesions located in the posterior body and ramus.

Cysts found in the posterior maxilla are more often associated with naevoid basal cell carcinoma syndrome.

Etiology

OKC is a developmental cyst that arises from remnants of the dental lamina. There is an association with mutation or inactivation of the PTCH1 gene, which activates the SHH signalling pathway and results in aberrant cell proliferation of the OKC epithelium.





Typical histology shows an uninflamed fibrous wall lined by a folded, thin, regular parakeratinized epithelium 5–8 cell layers thick, without rete ridges.

The parakeratin surface is typically corrugated, and the basal layer is well defined and often palisaded, with hyperchromatic nuclei and focal areas showing reversed nuclear polarity.





Focal areas may show reversal of nuclear polarity of the basal layer.





https://tumourclassification.iarc.who.int/chaptercontent/42/242

OKCs may show small satellite cysts or solid islands in the wall, or may have budding of the basal layer. These features are more commonly seen in cysts associated with naevoid basal cell carcinoma syndrome.



Lateral periodontal cyst and botryoid odontogenic cyst

Definition

Lateral periodontal cyst (LPC) is a developmental odontogenic cyst lined by non-keratinized epithelium, occurring on the lateral aspect or between the roots of erupted teeth. Botryoid odontogenic cyst (BOC) is the multicystic variant of LPC

Localization

LPCs / BOCs most frequently occur in the mandible, with < 20% arising in the maxilla. Almost all lesions have been reported to arise anterior to the molars, in particular in the premolar region

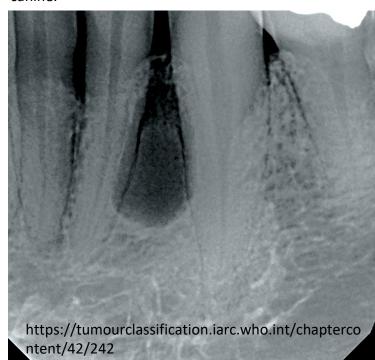
Epidemiology

LPCs / BOCs account for < 1% of odontogenic cysts

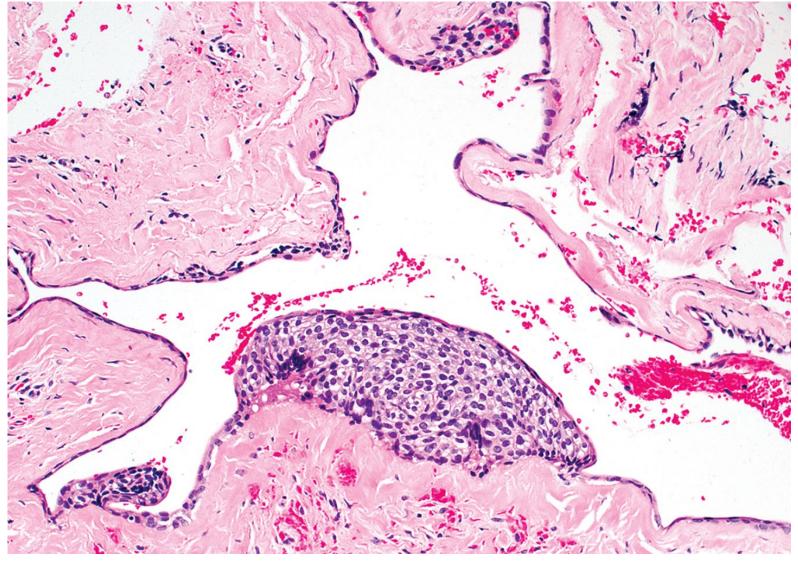
Etiology

LPC / BOC arises from odontogenic epithelial remnants, but the source is controversial. Origin from dental lamina, reduced enamel epithelium, or rests of Malassez has been proposed.

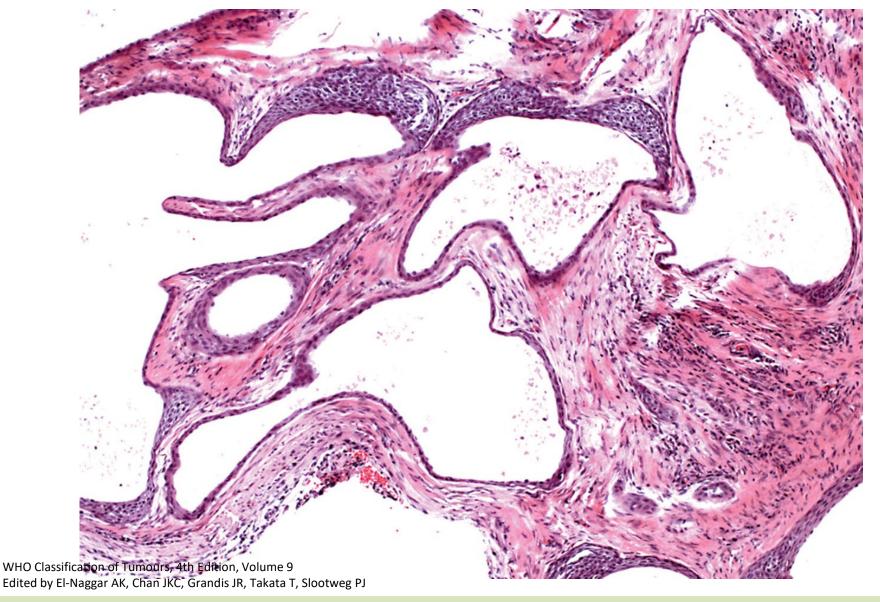
Radiograph shows a corticated radiolucency between the roots of the mandibular left second incisor and canine.







The lining is folded and shows a plaque-like thickening with whorling and clear cells.



The microscopic appearance of BOC is similar to that of LPC, except there are multiple cystic spaces.



Gingival cysts

Definition

Gingival cysts are odontogenic cysts found in the alveolar mucosa.

They can arise in adults and in infants.

Localization

In adults, most gingival cysts (as many as 75%) occur in the mandible in the premolar/canine region

Clinical features

Gingival cysts of the infant present as small (< 2 mm) white nodules on the alveolar mucosa, and are often multiple. Gingival cysts of the adult typically present as a painless, small, domeshaped elevation of the attached gingiva, resembling a blister.



WHO Classification of Tumours, 4th Edition, Volume 9
Edited by El-Naggar AK, Chan JKC, Grandis JR, Takata T, Slootweg PJ

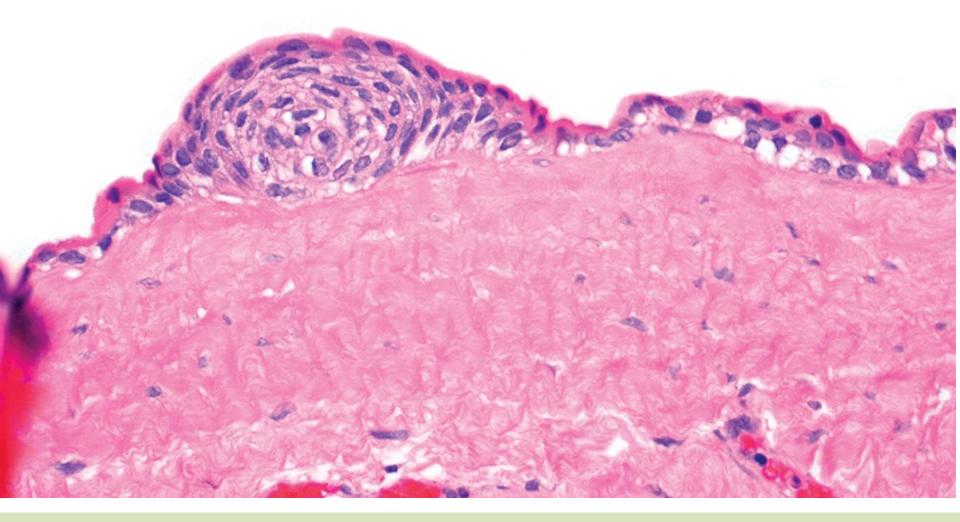
Epidemiology

Gingival cyst of the adult is rare, accounting for < 0.5% of odontogenic cysts

Etiology

The etiology is unknown. Gingival cysts are thought to be developmental cysts that arise from remnants of the dental lamina in the gingival or alveolar soft tissues (rests of Serres).





Plaque-like thickenings have a whorled appearance; note the basilar clear cells in the adjacent thin epithelial lining.



Glandular odontogenic cyst

Definition

Glandular odontogenic cyst (GOC) is a developmental cyst with epithelial features that simulate salivary gland or glandular differentiation.

Localization

GOC occurs exclusively in the jaws, with the mandible involved in about 75% of cases. Lesions in the maxilla tend to occur anteriorly.

Clinical features

The most common presentation is painless swelling. Radiographs reveal a well-defined unilocular or multilocular radiolucent lesion, which may have a scalloped border.

Radiography shows an extensive multilocular lesion crossing the midline and filling the body of the mandible.



WHO Classification of Tumours, 4th Edition, Volume 9
Edited by El-Naggar AK, Chan JKC, Grandis JR, Takata T, Slootweg PJ

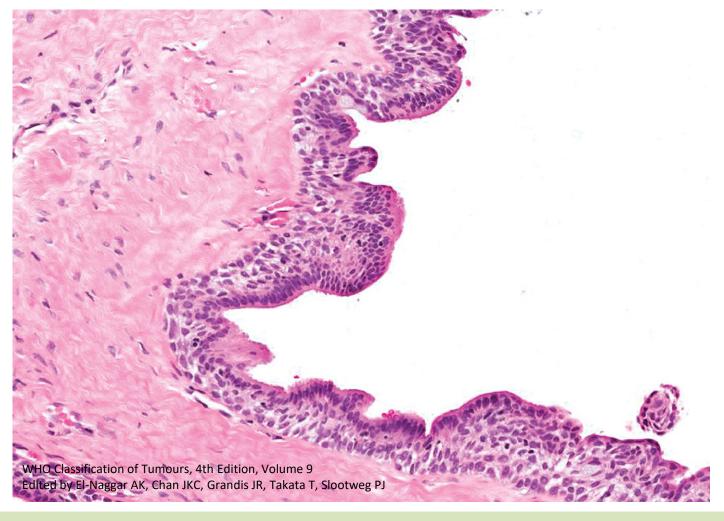
Epidemiology

GOC is rare, accounting for < 0.5% of all odontogenic cysts.

Etiology

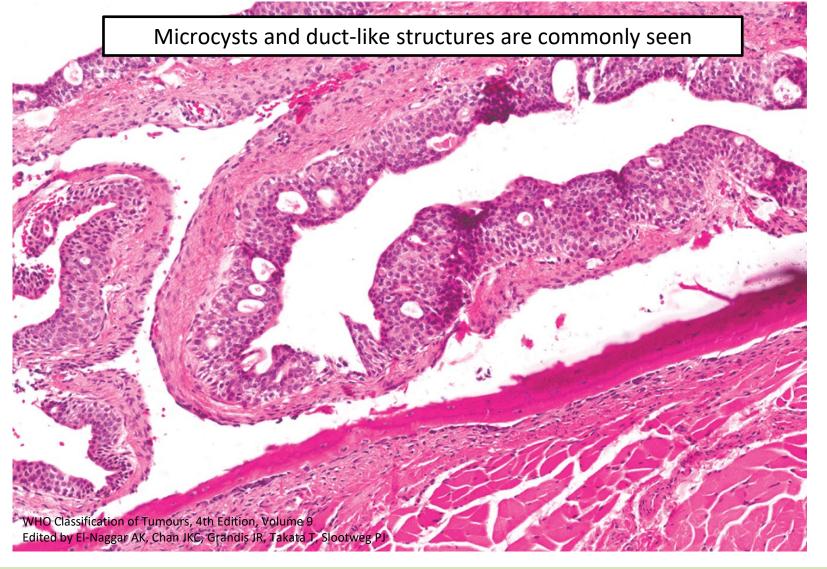
The etiology is unknown. GOC is thought to be a developmental cyst that arises from remnants of the dental lamina.





Diagnosis of GOC can be made when at least 7 of 10 specific criteria are present. Some criteria are present in all cases:

(1)variable thickness of the epithelium lining the cyst, from 2–3 cell layers of flattened squamous or cuboidal cells to thicker, stratified squamous epithelium, and (2) a luminal layer of cuboidal to low columnar cells, sometimes referred to as hobnail cells, present at least focally.



Other criteria are present in most cases: (3) intraepithelial microcysts, (4) apocrine metaplasia of the luminal cells, (5) clear cells in the basal and parabasal layers, (6) papillary projections (tufting) into the lumen, and (7) mucous cells.





An area showing ducts and prominent cilia.



Calcifying odontogenic cyst

Definition

Calcifying odontogenic cyst (COC) is a simple cyst lined by ameloblastoma-like epithelium, which contains focal accumulations of ghost cells.

Localization

COC can arise in either jaw, usually in the anterior regions. Lesions associated with odontomas have a predilection for the anterior maxilla.

Clinical features

The most common presentation is a painless swelling of the jaws. Radiographs reveal a well-defined radiolucent lesion, which is usually unilocular and may have a scalloped border. Tooth displacement and root resorption are common.

Epidemiology

COC is rare, accounting for < 1% of all odontogenic cysts. It occurs over a wide patient age range, with a mean patient age of about 30.



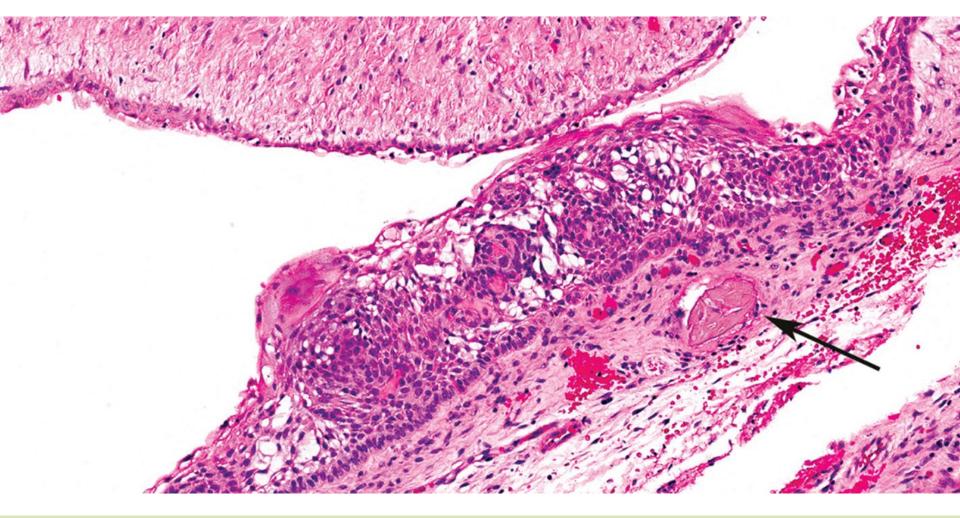
Many cases show foci of calcified tissue.

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PJ

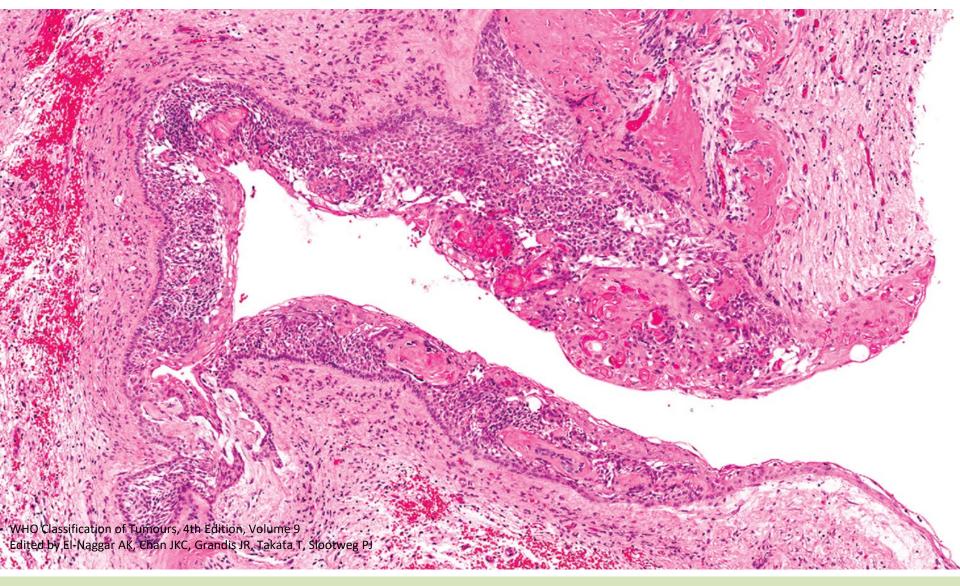
Etiology

On the basis of its behaviour and clinicopathological features, COC is now thought to be a developmental cyst that arises from the dental lamina



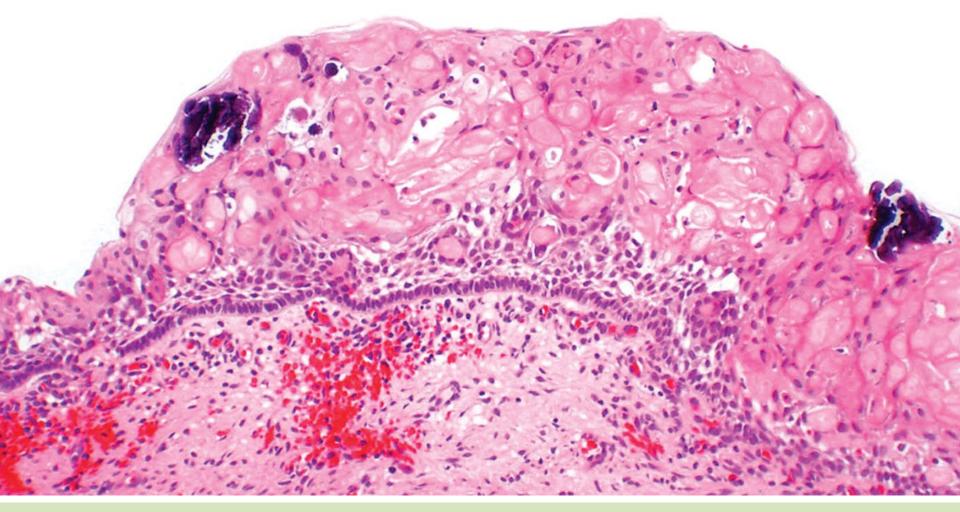


The lining is of variable thickness from only a few cell layers (top) to thick and ameloblastomalike (bottom). There is a palisading basal layer and focal accumulations of ghost cells can be seen in the wall (arrow); small squamous areas are also noted.



Focal accumulations of ghost cells can be seen throughout the epithelial lining and small sheets of dentinoid are visible in the wall (top right).





Ghost cells accumulate in the lining and form luminal nodules; note the areas of calcification.



Orthokeratinized odontogenic cyst

Definition

Orthokeratinized odontogenic cyst (OOC) is an odontogenic cyst that is entirely or predominantly lined by orthokeratinized stratified squamous epithelium.

Localization

OOCs are most frequently found in the mandible (accounting for 90% of cases), with about 75% of all lesions found in the posterior regions. Multiple and bilateral cases have been reported.

Clinical features

OOC usually presents as a painless swelling,. Radiology shows a well-demarcated unilocular radiolucent lesion, often with a corticated margin.

Epidemiology

OOCs account for about 10% of cases. They occur over a wide patient age range, with peak incidence in the third and fourth decades of life. Most studies show a male predilection



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Etiology

OOC is a developmental odontogenic cyst, but its pathogenesis is uncertain. An origin from remnants of the dental lamina is most likely

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A thin regular epithelial lining with a thick keratin layer which is lamellated and extends into the lumen



Thanks for attention!