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ETIOLOGY, PATHOGENESIS AND TREATMENT OF PERICEMENTITIS

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ContentS

ANATOMICO-PHYSIOLOGICAL CHARACTERISTIC OF PERIAPICAL TISSUES. PECULIARITIES OF PATIENT EXAMINATION WITH PERIAPICAL PATHOLOGY. PERICEMENTITIS. ETIOLOGY AND PATHOGENESIS.

Dentogingival junction.

The complex of epithelial cell types and connective tissue forming the gingival attachment to the tooth and alveolar bone is called the dentinogingival junction. Coronally, the keratinized marginal gingival invaginates against the cervical enamel to form a partial or nonkeratinized epithelium-lined gingival sulcus with an average depth of 1.0 to 2.0 mm. A depth of more than 3.0 mm is generally considered pathologic and termed as a periodontal pocket.

From the base of the sulcus, which corresponds to the level of the CEJ in the young adult tooth, a layer of junctional epithelial cells forms an adhesive basement membrane seal against the cementum of the root. The thickness of the junctional epithelium narrows from 15 to 30 cells at the base of the sulcus to one to three cells apically. Over time, cumulative bacterial and mechanical irritation often result in a lower gingival level (longer clinical crown) and a corresponding increase in the width of the junctional epithelium. Like other oral epithelial cells, the junctional epithelial cells exhibit high mitotic activity, and the cells migrate coronally to the base of the sulcus to be desquamated. An extensive vascular plexus underlies the junctional epithelial cells, which are widely spaced to facilitate the passage of vascular and inflammatory cells into the gingival fluid of the sulcus. Bacterial colonization within the sulcus is discouraged by the combination of a rapidly disrupted cellular

base and the lavage and antibacterial action of the vascular transudate of gingival fluid.

The supra-alveolar connective tissue and lamina propria of the gingival are made up of dense interlaced bundles of collagen fibers supporting the gingival and affixing it to the periosteum and cementum of the hard tissues. The fibers are classified by attachment and function into the following groups: (1) dentogingival, attaching the gingival to the cementum; (2) alveologingival, affixing gingival to alveolar bone; (3) transseptal, connecting interproximal cemental surfaces; (4) dentoperiosteal, from alveolar crest to cementum, an extension of the periodontal ligament; and (5) circular, around the tooth.

APICAL PERIODONTITIS

Inflammation of the periodontium caused by irritants of endodontic origin may be termed apical periodontitis.

Pathways of infection

There are several routes through which microorganisms can reach the dental pulp. Openings in the dental hard tissue wall, resulting from caries, clinical procedures, or trauma-induced fractures and cracks are the most frequent portals of pulpal infection. However, microbes have also been isolated from teeth with necrotic pulps and apparently intact crowns. Endodontic infections of such teeth are preceded by pulp necrosis. It has been suggested that bacteria from the gingival sulci or periodontal pockets might reach the root canals of these teeth through severed blood vessels of the periodontium. However, it is very unlikely that microorganisms would survive the immunologic defenses between the marginal gingival and the apical foramen. The teeth may clinically appear intact but reveal microcracks in hard tissues. The latter

may provide portals of entry for bacteria. Pulpal infection can also occur through exposed dentinal tubules at the cervical root surface because of graps in the cemental coating.

It has been proposed that bacteria remaining in infected dentinal tubules can bee a potential reservoir for endodontic reinfection. Microbial infection has also been claimed to reach and seed in the necrotic pulp via the general blood circulation by the "anachoresis". However, bacteria could not be recovered from the root canals when the blood stream was experimentally infected unless the root canals were overinstrumented and, presumably, the apical periodontal blood vessels were injured during the period of bacteremia. Evidence that further discredits anachoresis as a potential source of necrotic pulpal infection comes from the study of Moller et al in which all experimentally devitalized pulps (n=26) in monkeys remained sterile for more than 6 months. Therefore exposure of the dental pulp to the oral cavity is the most important route of endodontic infection.

Microflora of infected and untreated necrotic pulp

The endodontic microbiologic nature of teeth with infected necrotic pulp and apical periodontitis has been extensively researched. However, the results of most of the earlier endodontic microbial culture studies have become irrelevant because of the difficulty of avoiding bacterial contamination from the oral surroundings and the absence of appropriate anaerobic methods for root canal sampling and cultivation of fastidious organisms.

In principle a sample of the vast oral microbiota can infect the tooth pulp when the integrity of dental hard tissues is lost. However, the remarkable feature of the endodontic flora is the small number of species that are consistently isolated from such root canals. Application of advanced anaerobic techniques helped to establish that the root canal flora of teeth

with clinically intact crowns but having necrotic pulp and diseased periapices is dominated by obligate anaerobes usually belonging to the genera *Fusobacterium, Porphyromonas, Prevotella, Eubacterium , Peptostreptococcus.* On the other hand, the microbial composition, even in the apical third of the root canal of periapically affected teeth with pulp canals exposed to the oral cavity by caries, is not only different but also less dominated by strict anaerobes. In addition, spirochetes have been found in necrotic root canals using microbiologic methods, dark-field microscopy, and transmission electron microscopy. Spirochetes are motile, invasive pathogens that are associated with certain marginal periodontitis and suggested causative agents of acute necrotizing ulcerative gingivitis. However, their role in apical periodontitis remains to be clarified.

Endodontic flora in previously root-filled teeth

The microbiologic nature of root-filled canals is far less understood than that of untreated, infected, necrotic dental pulps. This is probably a consequence of searching for non-microbial causes of purely technical nature for the failure of root canal treatments. The taxonomy of the endodontic flora of root canal-treated teeth depends on the quality of the treatment and obturation of the canals. As such, teeth with inadequate instrumentation, debridement, root canal medication, and poor obturation should be expected to harbor a flora that is similar to that found in untreated canals. On the other hand, only a very restricted number of species has been found in the root canals and periapices of teeth that have undergone proper, conventional endodontic treatment but that, on follow up, reveal persisting, asymptomatic periapical radiolucencies.

The bacteria found in these cases are predominantly gram-positive cocci, rods, and filaments. Using microbiologic techniques, species belonging to the genera *Actinomyces,*

Enterococcus, Propionibacterium are the most frequently isolated and characterized microorganisms from such root canals. The repeated recovery of *Enterococcus faecalis* deserves particular attention. Although *E.faecalis* is an insignificant organism in infected but untreated root canals, it is extremely resistant to most of the intracanal medicaments used, particularly to the calcium hydroxide-containing dressings. It can also survive in root canals as monoinfection, without any synergistic support from other bacteria. Thus *E.faecalis* is a recalcitrant candidate among the causative agents of failed endodontic treatments.

Earlier microbiologic studies and more recent correlative electron microscopic studies have shown the presence of yeastlike microorganisms in canals of root-filled teeth with unresolving apical periodontitis, so as to implicate fungi as potential therapy-resisting endodontic organisms. *Candida albicans* is the most frequently isolated fungus from filled teeth with apical periodontitis.

Pathogenicity of endodontic flora

Any microbe that infects the root canals has the potential to initiate a periapical inflammation. However, the virulence and pathogenicity of individual species vary considerably and can be affected in the presence of other microbes. Although the individual species in the endodontic flora are usually of low virulence, collectively they are pathogenic due to a combination of factors. These factors include (1) interactions with other microorganisms in root canal, so as to develop synergistically beneficial partners; (2) the release of endotoxins; (3) the synthesis of enzymes that damage host tissues; (4) the ability to interfere with and evade host defenses.

CLASSIFICATION OF THE PERICEMENTITIS. CLINIC, PATHOLOGICAL ANATOMY AND DIAGNOSIS OF ACUTE APICAL PERIODONTITIS

Apical periodontitis is inflammation of the periodontium caused by infection of the pulp canal system. It has been the subject of numerous terms and classifications. *Periapical lesions, apical granuloma and cysts, periapical osteitis,* and *periradicular lesions* are frequently used synonyms. Although *periradicular* includes inflammation of the furcal and lateral locations, it does not etymologically distinguish the pulpally derived periodontitis from marginally spreading lesions. The limitations of the various terms and the arguments for the preferential retention of *apical periodontitis* have been discussed recently.

Being an inflammatory disease, apical periodontitis can be classified on the basis of symptoms, cause, histopathology and so on. The World Health Organization (WHO) classified apical periodontitis under diseases of periapical tissues into several categories based on clinical signs.

WHO (1995)classification of diseases of periapical tissues

Code number	**Category**
K04.4	Acute apical periodontitis
K04.5	Chronic apical periodontitis (Apical granuloma)
K04.6	Periapical abscess with sinus (Dentoalveolar abscess with sinus, Periodontal abscess of pulpal origin)

K04.60	Periapical abscess with sinus to maxillary antrum
K04.61	Periapical abscess with sinus to nasal cavity
K04.62	Periapical abscess with sinus to oral cavity
K04.63	Periapical abscess with sinus to skin
K04.7	Periapical abscess without sinus (Dental abscess without sinus, Dentoalveolar abscess without sinus, Periodontal abscess of pulpal origin without sinus)
K04.8	Radicular cyst (Apical periodontal cyst, Periapical cyst)
K04.80	Apical and lateral cyst
K04.81	Residual cyst
K04.82	Inflammatory paradental cyst

This useful classification, however, does not take into account the structural aspects of the diseased tissues. As the structural framework forms the basis of understanding of the disease process, a histopathologic classification is used here. It is based on the distribution of inflammatory cells within the lesion, the presence or absence of epithelial cells, whether the lesion has been transformed into a cyst, and the relationship of the cyst-cavity to the root canal of the affected tooth.

Acute apical periodontitis is acute inflammation of the periodontium of endodontic origin that is characterized by the presence of a distinct focus of neutrophils within the lesion. It is said to be *primary* when the inflammation is of short duration and is initiated within a healthy periodontium in

response to irritants. It is called *secondary* when the acute response occurs in an already existing chronic apical periodontitis lesion. The latter form is also referred to as *periapical flare-up, exacerbation, or "phoenix abscess".* The lesions may be nonepithelialized or epithelialized.

Chronic apical periodontitis is a long –standing inflammation of the periodontium of endodontic origin that is characterized by the presence of a granulomatous tissue, predominantly infiltrated with lymphocytes, plasma cells, and macrophages. The lesions may be nonepithelialized or epithelialized.

Periapical true cyst is an apical inflammatory cyst with a distinct pathologic cavity that is completely enclosed in an epithelial lining so that no communication to the root canal exists.

Periapical pocket cyst is an apical inflammatory cyst containing a saclike, epithelium-lined cavity that is open to and continuous with the root canal.

Classification of the periapical inflammation (pericementitis)

1. Acute pericementitis
    - Serous
    - Purulent (suppurative)
2. Chronic pericementitis
    - Chronic fibrous pericementitis
    - Chronic granulating pericementitis
    - Chronic granulomatous pericementitis
3. Exacerbation of chronic pericementitis

Pathogenesis and histopathologic nature

The microbial and host factors outlined previously may allow clinicians to develop a cohesive view of the

pathogenesis of apical periodontitis and to describe the shifting histopathologic nature of various classes of lesions. The structural components of the lesions depend on the balance between the microbial factors and the host defenses. As the dynamic equilibrium at the periapex tilts toward or away from the host defenses (because of local or unrelated systemic factors), the histologic picture of the lesions can vary considerably. Therefore, the morphologic description of apical periodontitis based on a zonal pattern (originally described for inflammation induced in bone) does not seem to represent the componental variation existing in the majority of periapical lesions. In fact, great structural heterogeneity is the "norm" of Initially the tooth pulp is infected and becomes necrotic because of an autogenous oral microflora. The endodontic environment provides a selective habitat for the establishment of a mixed, predominantly anaerobic, microbial community in the apical part of the root canal. The products of such a polymicrobial flora residing in the apical root canal, collectively, have several biologic properties, such as antigenicity, mitogenic activity, vasoactivity, chemotaxis, enzymatic histolysis, and activation of the host defense. As the body defenses cannot exterminate the invaders (well entrenched in the necrotic root canal that is outside the body milieu), apical periodontitis do not heal by themselves. However, the inflammatory response at the tissues, which explains why infectious organisms are only seldom encountered in the body of periapical lesions. Further, many of the endodontic microbial species may not have tissue-invasive properties.

Acute apical periodontitis (primary)

This is usually caused by microorganisms residing in or invading from the apical root canal into the periapical tissue, but it may also be induced by accidental trauma, injury

from instrumentation, or irritation from chemicals and endodontic materials, each of which can provoke an intense host response of short duration. It is accompanied by clinical symptoms, such as pain, tooth elevation, and tenderness to pressure on the tooth.

Histopathologically the tissue changes are generally limited to the apical periodontal ligament and the neighboring spongiosa. They are characterized by hyperaemia, vascular congestion, oedema of the periodontal ligament, and extravasation of neutrophils. The latter are attracted to the area by chemotaxis, induced initially by tissue injury, bacterial products, and complement factor C_{5a}. As the integrity of the hard tissues (bone, cementum, dentin) has not yet been disturbed, the periapical changes are radiographically undetectable. If some noninfectious but irritating agents have induced inflammation, the lesion may subside and the structure of the apical periodontium will be restored by healing.

When the infection is involved, the neutrophils not only attack and kill the microorganisms but also release leukotriens and prostaglandins. The former (LTB_4) attracts more neutrophils and macrophages into the area, and the latter activate osteoclasts. In a few days the bone surrounding the periapex can be resorbed and a radiolucent area may be detectable at the periapex. Neutrophils die in great numbers at the inflammatory site and release enzymes from their “suicidal bags”, causing destruction of the extracellular matrices and cells.The self-induced destruction of the tissues in the “battle zone” is to prevent the spread of infection to other parts of the body and also to provide space for the deployment of reinforcements arriving in the form of more specialized defense cells as the battle prolongs to a protracted war.

During the later stages of the acute response, macrophages begin to appear the periapex. Activated

macrophages produce a variety of mediators, among which the proinflammatory (IL-1, IL-6, TNF-α) and chemotactic (IL-8) cytokines are of particular importance.These cytokines intensify the local vascular response, osteoclastic bone resorption, effector-mediated degradation of the extracellular matrices, and they can place the body on general alert by endocrine action to sharply raise the output of acute-phase proteins and other serum factors by hepatocytes. They also act in concert with IL-6 to up regulate the production of haematopoietic CSF, which rapidly mobilize the neutrophils and the promacrophages from bone marrow. The acute response can be intensified (particularly in later stages) by the formation of antigen and antibody complexes. The acute early lesion may take several possible courses, such as spontaneous healing, further intensification and spreading into the bone (alveolar abscess), "point" and open to the exterior (fistulation or sinus tract formation), or the lesion may become chronic.

Clinical features of acute apical periodontitis

Acute, localized, and permanent pain is typical for the acute process in the periapical tissues. At first the pain is not so acute but according to the changes in quality and quantity of the exudation, made intensive; throbbing pain occurs.
Acute pericementitis lasts for 2-3 days up to 2 weeks.
The process goes through two stages:

Stage1: intoxication of periapical tissues (the beginning of inflammation). Continuous pain and sensitiveness during mastication. Percussion of the tooth is painful. There are no changes in the gums near the affected tooth. Regional lymphatic glands can be slightly enlarged and somewhat painful.

Pathological anatomy: according to the influence of microbial, physical, and other factors cell metabolism in the periapical tissues is changed what leads to the accumulation of

altered lactic acid and acidosis, continued with oedema of connective tissue and increase of blood vessels permeability and polymorphonuclear migration.

Stage 2: is the stage of (pronounced) marked exudation and continuous pain with growing intensity. Mastication and even touching to the tooth are painful. Percussion of the affected tooth is sharply painful mostly in vertical but lately in all directions. Exudation in the apical tissues provokes the filling of tooth growing and its pathological mobility. Gums at the projection of periapical process are oedematous and inflamed. Sometimes the percussion becomes less painful but it leads to the oedema of the vestibule fold. Regional lymphatic glands are increased and painful. EPT>100

Differential diagnosis of acute apical periodontitis

1. Acute pulpitis.
2. Pulpitis, complicated with apical periodontitis:
    - spontaneous throbbing pain with irradiation,
    - pain increased after all types of irritants, during mastication and at the nighttime. Light periods is very short or absent at all.
    - After perforation of pulp cavity, pulp tissue is painful and bleeds during probing.
3. Acute periostitis:
    - Acute pain with irradiation
    - Oedema of facial soft tissues
    - Palpation is very painful
    - Percussion of caused tooth and neighbouring teeth are painful
    - Regional lymphatic glands are increased and painful

- Rtg – no changes in the bone tissue
- Asymmetry of the face

4. Acute periosteomyelitis:
    - Acute boring, darting pain in the jaw
    - Sickness, headache, ↑t°=38-40°C, fever, sleeplessness, tachycardia, tachypnea
    - Asymmetry of the face
    - Coated tongue, oedema and inflammation of the gums; fetor ex ore
    - Very painful percussion of all teeth on the half of jaw and their mobility

5. Maxillary sinusitis:
    - Headache, rhinorrhea, pain in maxillary sinus area, rough breathing; Rtg changes of maxillary sinus

Sign	*Acute serous apical periodontitis*	*Acute purulent apical periodontitis*	*Exacerbation of chronic apical periodontitis*
Anamnesis	Duration of the disease - 24 hours	Duration of the disease - few days	Continuous dull pain or acute self-arising pain in the past; duration – few days.
Kind of pain	Pain (or sensitivity) only during mastication.	Continuous dull (gnawing) and some times throbbing pain, that increased after touching to the tooth. Irradiation of the pain some times occurs. Filling of "tooth growing".	Continuous dull pain.
Mobility of caused tooth	_	+	+
Changes of gum's mucosa	_	Inflammation of the gum's mucosa; pain during palpation.	Oedema and inflammation of mucosa and, some times, skin. Pain during palpation. Presence of gingival fistula.
Common condition	Normal	Sickness, headache, ↑t°=38-40°C, leucocytosis.	Sickness, headache, ↑t°=38-40°C, leucocytosis.

Differential diagnosis of acute serous, purulent and exacerbation of chronic apical periodontitis

CLINIC, PATHOLOGICAL ANATOMY, DIAGNOSIS OF CHRONIC APICAL PERIODONTITIS AND CHRONIC APICAL PERIODONTITIS IN THE STAGE OF EXACERBATION

Generally, chronic apical periodontitis is an asymptomatic periapical lesion that is manifested radiographically. Bacteria and their endotoxins cascading out into the periapical region from a necrotic pulp cause an inflammatory reaction that produces extensive demineralization of cancellous and cortical bone. The resulting radiographically evident lesions may be large or small, diffuse or circumscribed. Occasionally there may be slight tenderness t percussion testing or palpation testing or both testing methods. Often the patient will say that, although nonpainful, the tooth feels “different” or “hollow” when percussed. A sinus tract (incorrectly referred to as a “fistula” or “gum boil”) represents frank suppuration and has been termed a chronic suppurative apical periodontitis or a chronic apical abscess. As pressure from pus is relieved by drainage through a sinus tract, the sinus tract may close temporarily. When the pressure from pus builds up again (along with slight tenderness to palpation), the sinus tract returns.

The general absence of symptoms, the presence of a periapiucal radiolucency, and the confirmation of pulp necrosis confirm the diagnosis of chronic apical periodontitis. A totally necrotic pulp provides a safe harbor for the primarily anaerobic microorganisms and their noxious allies: if there is no vascularity, there are no defense cells. For this reason only complete cleansing, shaping, and obturation of the root canal will eliminate the source of the periapical disease and create a microenvironment in which this periapical lesions can remineralize.

Pathogenesis of chronic apical periodontitis.

In the continual presence of irritants (e.g., bacteria or their products), the neutrophil-dominated early lesion gradually shifts to a macrophage, lymphocyte, and plasma cell-rich lesion that is encapsulated in a collagenous connective tissue. Such asymptomatic, radiolucent lesion can be visualized as a "lull phase", after the intense and "high casualty" battle in which neutrophils "fell" in great numbers but the foreing intrudes into the priapex were temporarily beaten and the enemy held back in the root canal. The macrophage-derived proinflammatory cytokines (i.e., IL-1,6, TNF-α) are powerful lymphocyte stimulators. Although the quantitative data on the various types of cells residing in chronic periapical lesions are probably far from representative, investigations based on monoclonal antibodies tend to suggest a predominant role for t-lymphocytes and macrophages.

Activated T-cells produce a variety of cytokines that down regulate the output of proinflammatory cytokines, leading to the suppression of osteoclastic activity and reduced bone resorption. On the other hand, the T cell-derived cytokines may concomitantly up-regulate the production of growth factors (i.e., TGF-β), with stimulatory and proliferative effects on fibroblasts and the microvasculature. T_{h1} and T_{h2} cell populations may participate in this process. The option to down regulate the destructive process explains the absence of (or slowed) bone resorption and rebuilding of the collagenous connective tissue during the chronic phase of disease. Consequently, the chronic lesions can remain "dormant" and symptomless for long periods of time without major changes in the radiographic status. However, at any time the delicate equilibrium prevailing at the periapex can be disturbed by one or more factors that may favor the microbial enemy stationed within the root canal. The microbes may advance into the periapex and the lesion spontaneously becomes acute, with

clinical manifestations (i. e., secondary acute apical periodontitis, periapical exacerbation, phoenix abscess). As a result, microorganisms can be found extraradicularly during these acute episodes, with possibly rapid enlargement of the radiolucent area. The presence of this characteristic radiographic feature is because of apical bone resorption occurring rapidly during the acute phases, with relative inactivity during the chronic periods. The progression of the disease, therefore, is not continuous, but happens in discrete leaps after periods of "stability".

Chronic apical periodontitis is commonly referred to as dental or periapical granuloma. Histopathologically it consists of a granulomatous tissue with infiltrate cells, fibroblasts, and a well-developed fibrous capsule. When the epithelial cells begin to proliferate, they may do so in all directions at random, forming an irregular epithelial mass in which vascular and infiltrated connective tissue becomes enclosed. In some lesions the epithelium may grow into the entrance of the root canal, forming a pluglike seal at the apical foramen. The epithelial cells generate an "epithelial attachment" to the root surface or canal wall, which in TEM reveals a basal lamina and hemidesmosomal structures. In random histologic sections the epithelium in the lesion characterically appears as arcades and rings. The extraepithelial tissue predominantly consists of small blood vessels, lymphocytes, plasma cells, and macrophages. Among the lymphocytes, T-cells are likely to be more numerous than B-cells and $CD4^+$ cells may outnumber $CD8^+$ cells in certain phases of the lesions. The connective tissue capsule of the lesion consists of dense collagenous fibers that are firmly attached to the root surface so that the lesion may be removed *in toto* with the extracted tooth.

Clinical changes are slightly expressed. The *chronic fibrous pericementitis* can be outcome of pulpitis treatment.

Complaints: generally, absent.
Objective changes: deep carious cavity, connected with pulp chamber.
Root canals are with or without filling material.
Exploration is not sensitive.
Percussion is not painful.
Thermal test is negative.
EPT ≥100
Pathological anatomy: decrease of cells and increase of the mass of the rough (connective) tissue. Small infiltrates and blood vessels sclerosis occurs. Thickening of the apical part of pericementum tissues taken place.
Radiographically: widening and deformation of the periodontal split.

Chronic granulating pricementitis characterized by asymptomatic course. Slight pain during the palpation of mucous opposite to the affected tooth is accompanied by the origin of the sinus near the projection of the root (it is the main sign of this type of pericementitis).
Objective changes: deep carious cavity, connected with pulp chamber.

Hyperemia of the gingival near the tooth can occur, more often during the exacerbation of chronic process.

Symptom of vasoparesis – after pressing on the gums by dull end of the instrument turn pale of mucosa occurs, that replace with stable hyperemia.

Presence of the *sinus tract* on the gums near the projection of the root of affected tooth is the main sign of this type of apical periodontitis.

Palpation of the gums is sensible as well as tooth percussion.

Regional lymphatic glands can be enlarged and painful.
Thermal test is negative.

EPT: >100

X-ray: focus of bone resorption (radiolucent area associated with the apex) with not clear border.

Pathological anatomy: formation of granulating tissue with many capillaries, fibroblasts, plasmatic cells, leucocytes, osteoclastic bone destruction and the tooth cement resorption. Toxic products of the local inflammation are absorbed into the blood system leading to the general intoxication.

Chronic granulomatous pericementitis.

In most cases clinically asymptomatic. Clinical signs are present only during the exacerbation of the disease. Diagnosis is put according to X-ray examination with the focus of bone resorption with a clear border and diameter up to 0.5 cm.

Pathological anatomy: this form of inflammation is no so active as chronic granulating pericementitis, because proliferation prevails over exudation. Pericemental tissues are partially replaced by granulating tissue. But in this case granulating tissue is limited by fibrous capsule. Majority of granulomas contain plasmatic cells, blood cells, and sometimes, epithelial cells, which can form the lining of granuloma. According to the presence of epithelial cells granulomas can be divided into simple (without epithelial tissue) and complicated, which are more likely to the periapical cysts (cyst have diameter more than 0.5 cm). Precisely granuloma can be differentiated from the cyst only according to histological investigations.

Periapical or radicular cyst

Periapical or radicular cysts are generally considered to be a direct sequel to chronic apical periodontitis, byt not every chronic lesion develops into a cyst. As has been stated before, there are two distinct categories of radicular cysts: (1) those

containing cavities completely enclosed in epithelial lining, and (2) those containing epithelium-lined cavities that are open to the root canals. The latter was originally described as "bay cysts" but has been newly designated as periapical pocket cysts. More than half of the cystic lesions are apical true cysts, and the reminders are the apical pocket cysts. In view of the structural difference between the two categories of cysts, the pathogenic pathways leading to the formation of them may differ in certain respects.

Periapical true cyst.

There have been several attempts to explain the pathogenesis of apical true cysts. The process of true cyst formation has been discussed as occurring in three stages. During the first phase the dormant cell rests of Malassez are believed to proliferate, probably under the influence of growth factors that are released by various cells residing in the lesion. During the second phase, an epithelium-lined cavity comes into existence.

There are two long-standing hypotheses regarding the formation of the cyst cavity:

1. The "nutritional deficiency theory" is based on the assumption that the central cells of the epithelial strands get removed from their source of nutrition and undergo necrosis and liquefactive degradation. The accumulating products, in turn, attract neutrophilic granulocytes into the necrotic area. Such microcavities containing degenerating epithelial cells, infiltrating leukocytes, and tissue exudates coalesce to form the cyst cavity lined by stratified squamous epithelium.
2. The "abscess theory" postulates that the proliferating epithelium surrounds an abscess formed by tissue necrosis and lysis because of the inherent nature of epithelial cells to cover exposed connective tissue

surfaces. During the third phase, the cyst grows, the exact mechanism of which has not yet been adequately clarified.

Histopathologically there are four major components in an apical true cyst: (1) cyst cavity, (2) epithelial cyst wall, (3) extraepithelial tissue, and (4) collagenous capsule.

The cavity, completely enclosed in epithelial lining, generally reveals necrotic tissue and, on occasion, cholesterol clefts and erythrocytes (the presence of the latter is probably due to hemorrhage). The thickness of the stratified squamous epithelium can vary from a few to several cell layers. The basal cell side of the epithelium is irregular, so as to form ridges. The tissue existing between the epithelial lining and the fibrous capsule usually consists of numerous blood vessels and infiltrating cells, predominantly T-lymphocytes, B-lymphocytes, plasma cells, and macrophages. Neutrophils, which are numerous in the epithelial lining, are rarely found in the extraepithelial area.

Periapical pocket cyst

A periapical pocket cyst is probably initiated by the accumulation of neutrophils around the apical foramen in response to the bacterial presence in the apical root canal. The microabscess so formed can become enclosed by the proliferating epithelium that, on coming in contact with the root tip, forms an epithelial collar with “epithelial attachment”. The latter seals off the infected root canal and microabscess from the periapical milieu. When the externalized neutrophils die and disintegrate, the space occupied by them becomes a microcystic sac. The presence of microbes in the apical root canal, their products, and the necrosed cells in the cyst lumen attract more neutrophilic granulocytes by a chemotactic gradient. However, the pouchlike lumen, biologically outside the periapical milieu, acts as a “death trap” to the

transmigrating neutrophils. As the necrotic cells accumulate, the saclike lumen enlarges to accommodate the debris and may form a voluminous diverticulum of the root canal space extending into the periapical area. Bone resorption and degradation of the matrices occurring in association with the enlargement of the pocket cyst may follow a similar molecular pathway, such as in the case of the periapical true cyst. From the pathogenic, structural, tissue dynamic, and host benefit standpoint, the pouchlike extension of the root canal space has much in common with a marginal periodontal pocket; hence the name periapical pocket cyst. Histologically the stratified squamous epithelial lining and the rest of the cyst wall are similar to those of a true cyst.

Differential diagnosis.

1. Chronic middle caries – short-lived pain after chemical and thermal irritants, percussion is not painful, EPT= 2 – 6 mcA, Rtg: radiolucency beneath the enamel surface.
2. Chronic deep caries - short-lived pain after chemical, thermal, and mechanical irritants which passing after their removing; percussion not painful, EPT= 2 – 6 – 10 mcA.
3. Chronic apical periodontitis (between each other)
4. Cysts (radicular and follicular)

PRINCIPLES AND SCHEME OF TREATMENT OF ACUTE APICAL PERIODONTITIS

Aim (purpose) of treatment:

1. Liquidation of periapical inflammation.
2. Prevention of complication origin.

3. Restoration of anatomical structure and function of the tooth.

Methods of apical periodontitis treatment:
1. Conservative
2. Surgical
3. Combinative (conservative-surgical)

Conservative method
Indications:
1. Acute and exacerbation of chronic apical periodontitis
2. Chronic apical periodontitis

Contraindications:
1. Increasing of inflammation in spite of periosteotomy and opening of the root apex.
2. Radicular cysts.

The main principle of treatment of acute apical periodontitis – to create a *good exudate evacuation*.
Ways of exudates evacuation:
1. Through the root canal
2. Through the dentogingival junction
3. Through the incision
4. Through the alveole

Acute toxic pericementitis (produced by the prolonged influence of As-paste or its overdose). The successful treatment needs quick amputation and extirpation of the pulp remnants. Root canals should be washed by antiseptics (1-2% chloramin, 3% hydrogen peroxide, furacilinum 1:5). After this procedure medications As-antidotes are left in the root canal (5% solution of unitiol, 1% solution of jodinolum). Electrophoresis with KJ can be recommended when inflammation is not acute. Temporary filling leaving one of the

abovementioned antidotes in the canals can close tooth. In next visit (after 1-2 days), in the case the signs of inflammation are absent root can be filled. In the case of acute inflammation (painful percussion, exudation from the canal) the tooth is left open till next visit.

Acute infectious pericementitis.

In the stage of acute inflammation and exudation the tooth is left open for 2-5 days (it is important the exudates to go out from the canal). Only in the first stage of acute inflammation, when there is little exudation tooth can be temporary closed, leaving in the canal antimicrobial and anti-inflammatory medications.

Trepanation (opening of the tooth) or removing the filling is painful and must be done under local anesthesia, and with the lowest pressure from the handpiece.

It is very important not only to widen the canal but also to open apical foramen. Only in this case the exudates will be let out.

When the signs of general intoxication are present (headache, rising of body temperature, weakness, changes in general blood formula), antibiotics, antimicrobial medications (active to anaerobic microorganisms) should be administrated.

When the periapical inflammation is complicated by periostitis, periosteotomy must be done (horizontal incision 2-3 cm long) to achieve exudates and let the pus go out.

In next visit, when the signs of acute inflammation decrease, the canal is mechanically treated and washed by antiseptic solutions (chloramin, furacilin, chlorhexydin, proteolytic enzymes or antibiotics) and closed by temporary filling. Root canal is usually filled in the third visit.

Technique stages:

I-st visit

1. Anesthesia
2. Operative field isolation
3. Carious cavity preparation
4. Evacuation of necrotic tissues from the root canal
5. Opening of the root canal apex
6. Cleaning of the root canal with strong antiseptics
7. Hermetization of the root canal with cotton with antiseptic, temporary filling (in the 1-st stage of periapical inflammation) *or* leaving the tooth open (in the 2-nd stage of periapical inflammation)

II-nd visit

1. Widening and cleaning of root canal
2. Hermetization of the root canal with cotton with antiseptic
3. Temporary filling

III-rd visit

1. Removing of the temporary filling
2. Cleaning of root canals with antiseptics
3. Drying of root canals
4. Filling of root canals
5. Tooth restoration

PRINCIPLES AND SCHEME OF TREATMENT OF CHRONIC APICAL PERIODONTITIS

Treatment of chronic periapical inflammation.

Chronic periapical inflammation belongs to the diseases, which produce the focus of chronic inflammation in the body. The treatment measures have to be chosen according to the size of the periapical bone resorption and patient's general condition (the presence of septic endocarditis, nephritis, rheumatism).

Treatment methods: conservative, surgical, surgical-conservative.

The aims of the doctor during the treatment are:

- Influence on the microorganisms of root canal (macrocanal).
- Influence on the microorganisms of microcanals
- Stopping the inflammation in the periapical tissues.
- Stimulation the regeneration in the periapical tissues.
- Desensibilization of the body.

To achieve goods results root canal must be widely opened at full length allows the influence on the periapical focus and excludes the canal as the seat (breeding ground, nidus) of bacterial toxins.

In the front teeth and often in premolars, where root canals are usually single and not obturated, treatment of chronic periapical inflammation will be less complicated.

Indications to conservative treatment are periapical changes up to 0.5 cm in diameter. The treatment is conducted in 2-3 visits.

I-st visit : necrectomy, formation of carious cavity, taking into consideration the necessity of good access to the canals with all endodontic instruments.

Portions under the protection of antiseptic solutions must evacuate necrotic remnants of the pulp very carefully. The dentist mustn't allow the debris from the root canal to be pushed beyond the apical foramen.

Tooth can be closed by temporary filling with strong antiseptic (pheresolum, phormakresol) and instrumentation of the canal will be done in next visit. In the I-st visit instrumentation can also be done but sometimes it can provoke exacerbation.

II-nd visit: (in 2-3 days) instrumentation of the canals, widening the canals. If the signs of exudates are finding on the paper point taken out from the canal, medication of the canal with enzymes, antiseptics has be continued.
When the canal is clean the dentist must check up the apical foramen (it must be open). Reamers or K-files can do the opening of apical foramen. Only in the case of opened apical foramen we can influence on the periapical focus of inflammation.

III visit: Filling of the root is usually done in the 2 or 3 visit. Before filling the root, biologically active pastes (with Ca (OH)2, lysozym, metacyl and others) can be put in periapical focus to stimulate bone regeneration.

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