

APICAL PERIODONTITIS: ETIOLOGY,  
PATHOGENESIS, CLASSIFICATION

Minsk BSMU 2015

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ

БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ

1-я КАФЕДРА ТЕРАПЕВТИЧЕСКОЙ СТОМАТОЛОГИИ

# **АПИКАЛЬНЫЙ ПЕРИОДОНТИТ: ЭТИОЛОГИЯ, ПАТОГЕНЕЗ, КЛАССИФИКАЦИЯ**

## **APICAL PERIODONTITIS: ETIOLOGY, PATHOGENESIS, CLASSIFICATION**

Учебно-методическое пособие



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Рассматриваются вопросы этиологии, патогенеза и классификации апикального периодонтита. Материал базируется на имеющихся в отечественной и зарубежной литературе современных представлениях по данной проблеме.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке.

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## DEFINITION

The periodontium comprises the root cementum, the periodontal ligament and the alveolar bone. The apical periodontium is that part of the periodontium which surrounds the root apex.

The *cementum* and bone are composed of about 65 % of inorganic material (hydroxyapatite), 12 % of water. The organic matrix consists of 20 % of collagen and 3 % of noncollagenous proteins. Cementum covers the root surfaces of the teeth. It is avascular, without innervations and does not normally undergo remodeling. In apical periodontium, the cementum is cellular with cementocytes in lacunae and canaliculi.

*Alveolar bone* lines the sockets of the teeth. It is compact and may appear radiographically as a radiopaque narrow zone, the lamina dura. The alveolar bone is perforated by a large number of blood vessels; osteocytes are embedded in lacunae and canaliculi. Toward the periodontal ligament, the bone is covered by unmineralized osteoid and the bone-forming cells, osteoblasts.

*Periodontal ligament* presents unmineralized dense connective tissue that fills the space between the cement of the tooth root and alveolar bone. It contains many collagen fibers arranged at an angle between the root cement and alveolar bone. At the apex of the tooth, fibers diverge radially from the root surface and woven into the surrounding bone.

Periodontal ligament has a rich blood supply. The main source of blood supply is *alveolar artery*. Individual branches reaching apical periodontium rise coronally before penetrating into dental pulp, while gingival arterioles penetrate into the periodontal ligament extending in the apical direction. In addition, a large number of vessels in the periodontal ligament penetrate through the bone hole. Thus, in contrast to the dental pulp, periodontium has a rich collateral circulation. The capillary network is also well developed. It is more pronounced in the bone tissue, rather than at the root surface. Near the vessels are *lymph capillaries* that carry lymph to the regional lymph nodes. *Nerve trunks* in the periodontium are located along blood vessels. Among the nerve endings are found both myelinated and unmyelinated. Sensitive closure

system includes trigeminal nerve. Nerve endings are proprioceptors and pain receptors that respond to the slightest changes in tissue pressure.

The apical periodontium contains several *cell types*: cementoblasts, fibroblasts, mast cells, macrophages, osteoblasts, osteoclasts, clusters of epithelial cells.

Apical periodontitis is inflammation and destruction of periradicular tissues caused by etiological agents of endodontic origin. It is generally a sequel to endodontic infection (fig. 1).

Periapical periodontitis (also termed apical periodontitis, AP, or periradicular periodontitis) is an acute or chronic inflammatory lesion around the apex of a tooth root which is caused by bacterial invasion of the pulp of the tooth. The term is derived from *peri-* meaning «around», *apical* referring to the apex of the root (the tip of the root), and *-itis* meaning a disease characterized by inflammation. Periapical periodontitis can be considered a sequela in the natural history of dental caries (tooth decay), irreversible pulpitis and pulpal necrosis, since it is the likely outcome of untreated dental caries, although not always. Periapical periodontitis may develop into a periapical abscess, where a collection of pus forms at the end of the root, the consequence of spread of infection from the tooth pulp (odontogenic infection), or into a periapical cyst, where an epithelial-lined, fluid-filled structure forms (Wikipedia).

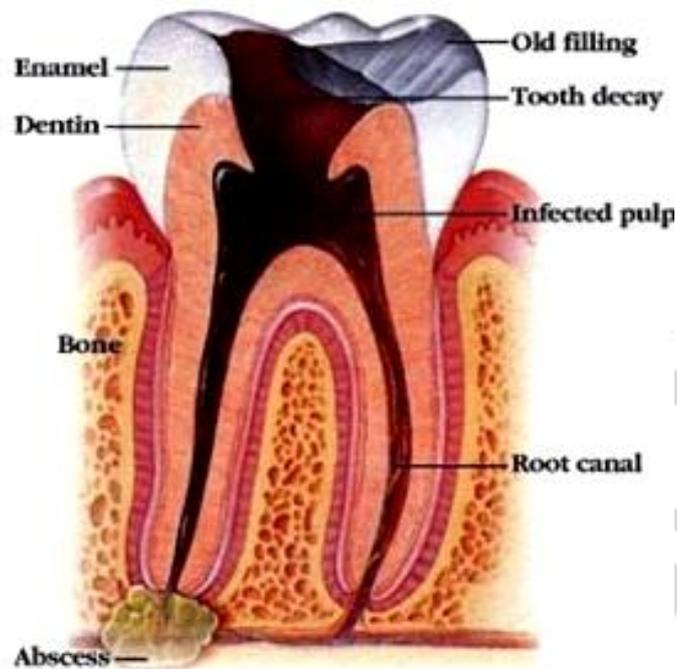


Fig. 1. Tooth with nonvital pulp with periapical abscess

## ETIOLOGY

Apical periodontitis can be caused by both exogenous and endogenous factors. *Exogenous* factors include microbes and their toxins and noxious metabolic byproducts, chemical agents, mechanical irritation, foreign bodies, and trauma. *Endogenous factors* include the host's metabolic products, such as urate and cholesterol crystals, as well as cytokines or other inflammatory mediators that activate osteoclasts.

*Trauma*, any direct blow to the tooth, can sometimes cause the pulp of the tooth to die and it may become infected by bacteria from the gum margins leading to apical periodontitis. A sudden bite on a hard object, undue pressure during [orthodontic treatment](#) or a filling, that is high, can sometimes cause acute periodontitis, though usually short-lived.

*Pulpitis, pulp necrosis, infection of the root canal, iatrogenic factors* can lead to the development of apical periodontitis.

*Pulpitis* can lead to changes in the periapical tissues. Inflammation in dental pulp causes dilation of blood vessels, increased vascular permeability with the release of fluid from the blood stream into the surrounding tissue. In

most cases, the lymph and blood vessels of healthy tissues provide a full evacuation of excess fluid. However, in case of damage to these compensatory mechanisms, the tissue pressure increases with the development of clinical signs of apical periodontitis.

Tooth cavity and root canals in the teeth with *necrotic pulp* are filled with dead cells, tissue fluid and tissue decay, the components of which have a cytotoxic effect. It was assumed that the presence of necrotic tissue inside the tooth cavity is one of the main etiological factors of apical periodontitis. However, today it is proved that the development of pronounced changes with periapical bone resorption and periapical granulomas formation occurs only in the case of penetration of the root canal of alien chemicals and proteins. This is due to the fact that the necrotic tissue decreases only a weak chemotactic activity in serum due to a weak immunogenic potential. Clinically benign course of the process is shown as lack of radiographic signs of inflammation in the absence of microbial contamination of necrotic tissue.

Development of periodontal bone resorption occurs only in the case of penetration of *microorganisms* into the necrotic pulpal tissue. Opening of the pulp chamber leads to *infection of the tooth pulp and root canals*. Anaerobic microflora predominates in root canals. If the entrance to the root canal is closed, the selective growth of anaerobic microflora begins. Anaerobic microorganisms comprise 50 % of microflora in 7 days. The term «root canal infection» refers to microbial contamination of the main canal, lateral canals, deltoid branches and dentinal tubules. More than 500 different microbial strains are present in the mouth. However, no more than 10 cultured strains are typically present in root canals.

The main species of microorganisms involved in the development of endodontic pathology (anaerobes, facultative forms and aerobes):

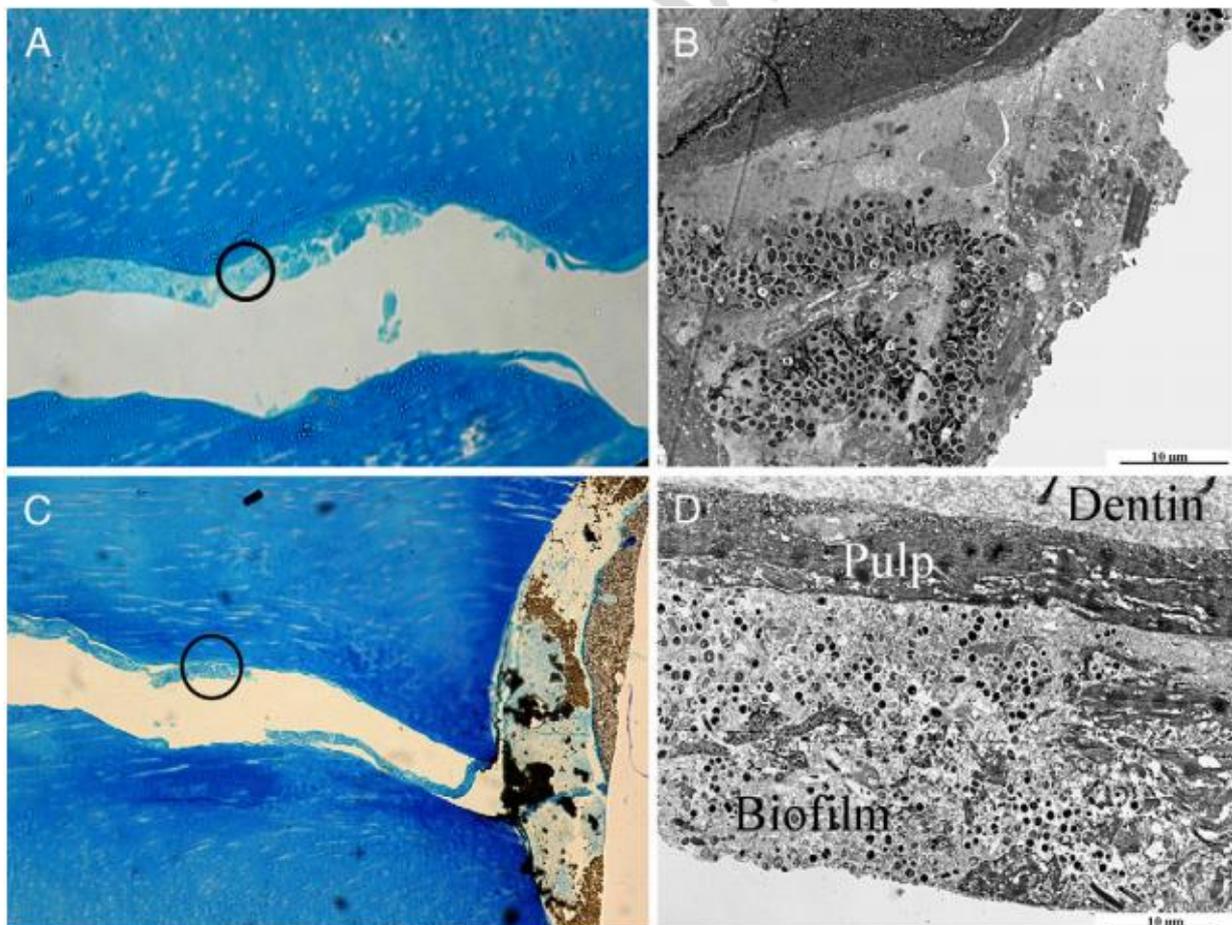
- Veillonella
- Bacteroides
- Campylobacter
- Fusobacterium
- Porphyromonas
- Prevotella
- Wolinella

- Selenomonas
- Mitsuokella
- Treponema
- Gemella
- Peptostreptococcus
- Eqqerthella
- Eubacterium
- Bifidobacterium
- Actinomyces
- Propionibacterium
- Neisseria
- Eikenetia
- Capnocytophaga
- Acunobacillus
- E. coli
- Enterobacter
- Klebsiella
- Serratia
- Proteus
- Pseudomonas
- Streptococcus
- Staphylococcus
- Enterococcus
- Actinomyces
- Lactobacillus
- Propionibacterium
- Bacillus
- Corynebacterium.

The so-called «red complex» (*B. forsythus*, *P. gingivalis* and *T. dentikola*) is found in 40 % of root canals. In the development of the disease, the «orange complex» plays a significant role. It includes *Campylobacter rectus*, *S. showae*, *Eubakterium nodatum*, *Fusobacterium nucleatum*, *P. intermedia*, *P. micros* and

*P. nigrescens*. The third group includes actinomycetes, whereas the fourth one consists of *Capnocytophaga*, *A. actinomycetemcomitans*, *Eikenella corrodens* and *Campylobacter concisus*. Streptococci form the fifth group, whereas the sixth group is formed by *Veyllonella parvula* and *Actinomyces odontolyticus*. Often, the microorganisms can be detected in root sealed canals with signs of apical periodontitis following a poor quality endodontic treatment (fig. 2).

Development of periapical inflammation can be due to the influence of *iatrogenic factors* such as *instrumental and pharmacological treatment* of canals, as well as the use of different *filling materials*. However, bacteria can be present in the canal and enter it during endodontic treatment. Thus, there is no doubt that many of the complications that were previously considered as a result of iatrogenic injury are actually the result of infection. After extirpation of the pulp, an open wound surface remains in the apical foramen. The inflammatory reaction is the first phase of defensive reaction. The complete regeneration is formed in 3–4 weeks. Intensity of tissue reactions that occur in response to the canal processing tool will depend on the degree of damage. Thus, the output of the tool beyond the apical foramen with rupture and injury to periapical tissues can lead to the development of acute apical periodontitis. Complete regeneration takes place in the absence of a bacterial factor. Penetration of infected endodontic instruments beyond the apical foramen leads to the spread of infection, bone resorption, proliferation of epithelial cells with cyst formation.



*Fig. 2. Ultrastructural Examination of Failed Molar Retreatment with Secondary Apical Periodontitis: An Examination of Endodontic Biofilms in an Endodontic Retreatment Failure (Adapt. from G. B. Carr et al., 2009)*

## PATHOGENESIS

Apical periodontitis may be acute (symptomatic) or chronic (asymptomatic). The pathogenesis of apical periodontitis involves an encounter at the periapex between the microbial and host factors (fig. 3).

The inflammation of periapical tissues is induced by microorganisms residing in the apical root canal, accidental trauma, injury from instrumentation, or irritation from chemicals and endodontic materials. Each of the factors can provoke an intense tissue response of short duration. The tissue response is limited to the apical periodontal ligament and the neighboring spongiosa. The result of typical neurovascular response of inflammation is hyperemia, vascular congestion, edema of the periodontal ligament, and extravasation of neutrophils. The latter are attracted to the area by chemotaxis, induced initially by tissue injury, bacterial products (LPS), and complement-factor C5a. The integrity of bone, cementum, and dentin has not yet been disturbed. If non-infectious irritants induced the inflammation, the lesion may resolve, and the structure of the apical periodontium may be restored.

«Apical periodontitis — a great battle»

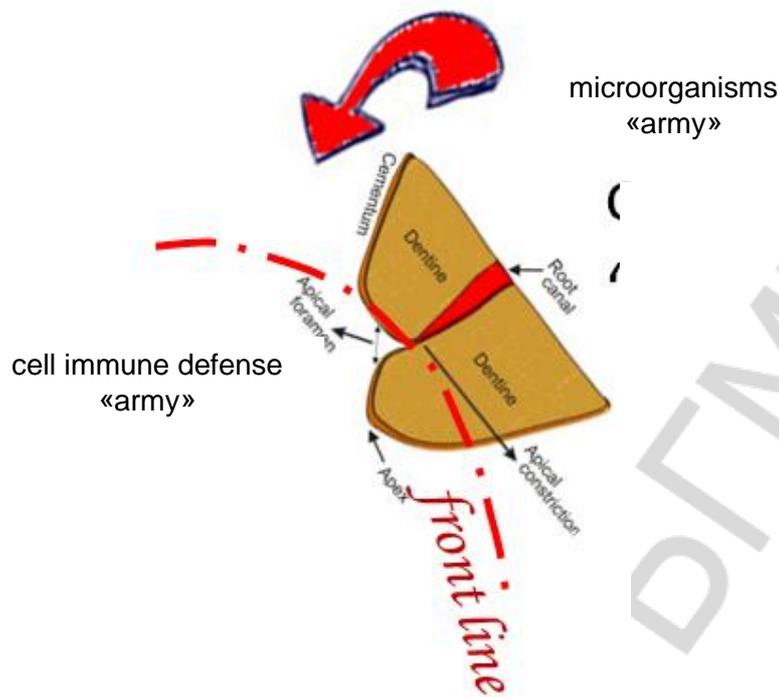
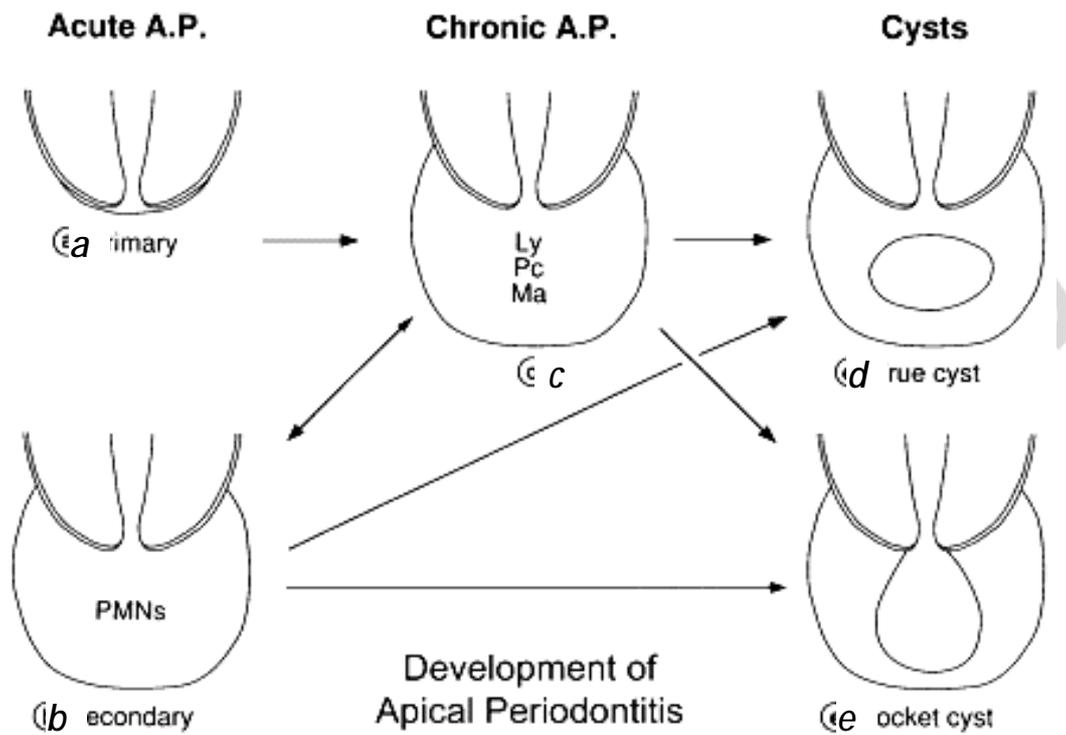


Fig. 3. Pathogenesis of apical periodontitis

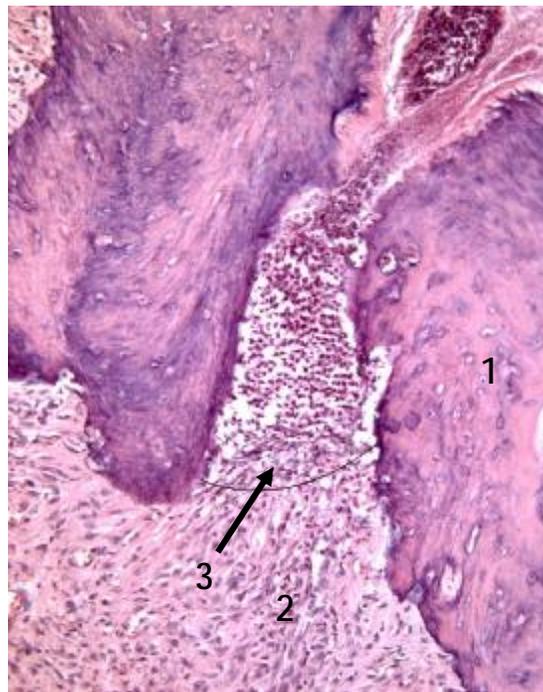
When infection is involved, the neutrophils fight the microorganisms and release leukotrienes and prostaglandins. Neutrophils and macrophages are attracted into the area, and osteoclasts become activated. In a few days' time, the bone surrounding the periapex can be resorbed. Neutrophils die at the inflammatory site and release enzymes from their cytoplasmic granules that cause destruction of the extracellular matrices and cells. The self-induced destruction of the tissues prevents the spread of infection to the other parts of the body and provides space for the infiltration of specialized defense cells. During the acute phase, macrophages also appear at the periapex. Activated macrophages produce a variety of mediators, among which the pro-inflammatory (IL-1, IL-6, and TNF- $\alpha$ ) and chemotactic (IL-8) cytokines are of particular importance. These cytokines intensify the local vascular response, osteoclastic bone resorption, and degradation of the extracellular matrices.

The possible outcomes of acute primary apical periodontitis are spontaneous healing, further intensification and spreading into the bone (alveolar abscess), open to the exterior (fistulation or sinus tract formation), or chronic apical periodontitis (fig. 4–10).

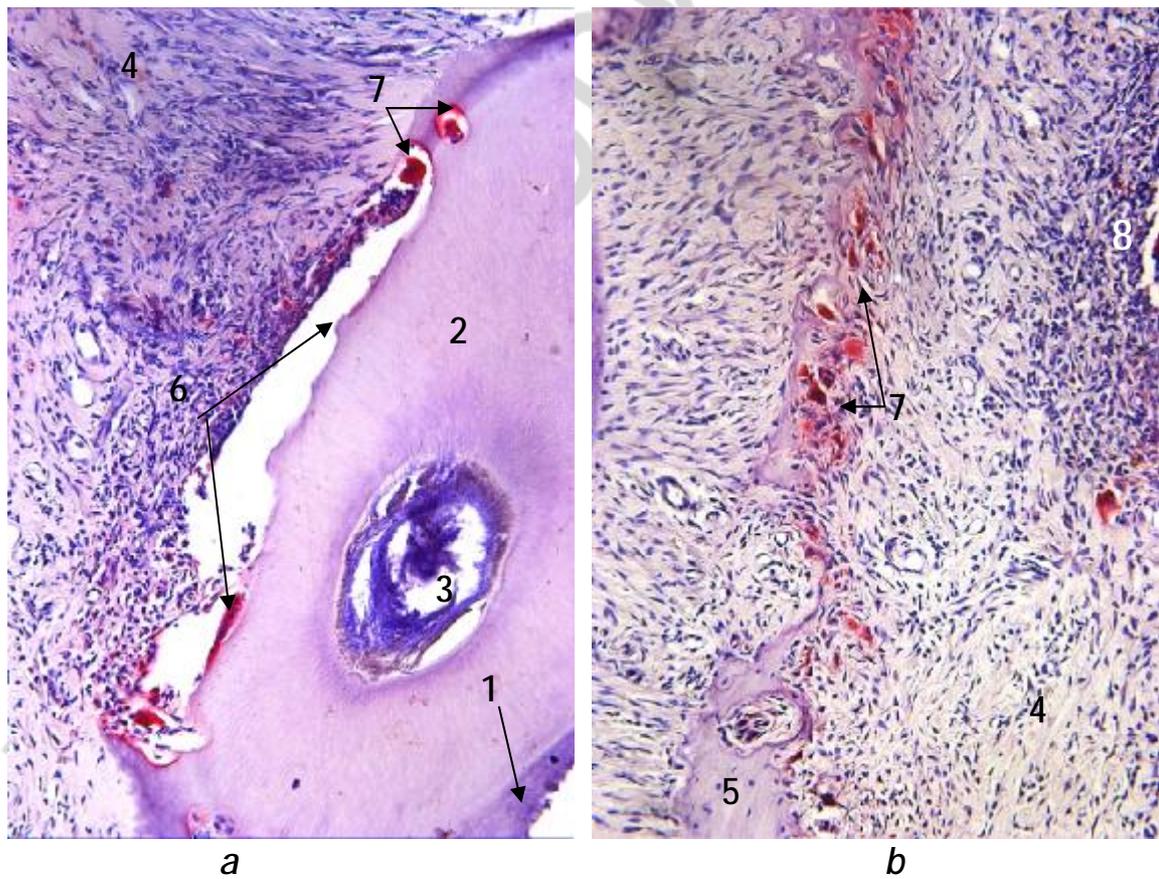


*Fig. 4.* Pathogenesis of acute (a, b), chronic (c), and cystic (d, e) apical periodontitis (AP) lesions. The acute lesion may be primary (a) or secondary (b), and is characterized by the presence of a focus of neutrophils (PMNs). The major components of chronic lesions (c) are lymphocytes (Ly), plasma cells (Pc), and macrophages (Ma). Periapical cysts can be differentiated into true cysts (d), with completely enclosed lumina, and pocket cysts (e), with cavities open to the root canal. Arrows indicate the direction in which the lesions can change.

(adapt. from Nair, 1998)



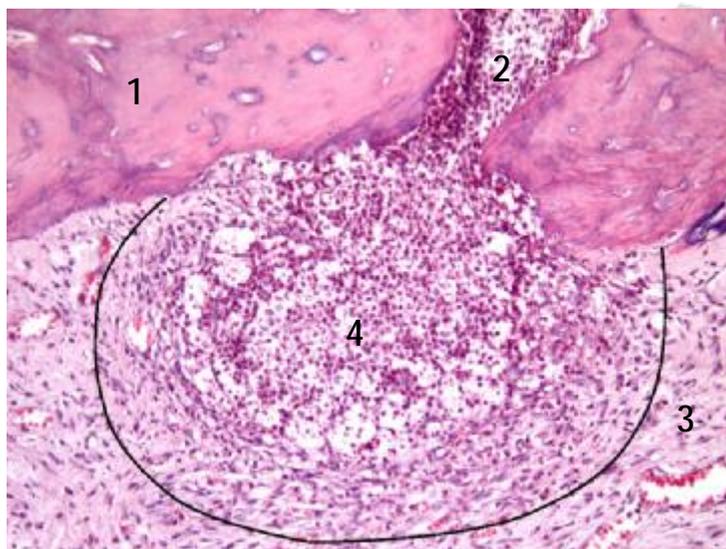
*Fig. 5.* The periapical abscess formation; the line marks the length of the inflammatory focus:  
 1 — root of the tooth; 2 — periodontal ligament; 3 — inflammatory focus. Magnifications  $\times 200$ .  
 Coloring: hematoxylin and eosin



*Fig. 6.* Resorption of the alveolar bone and hard tissues of the tooth root due to acute apical periodontitis:

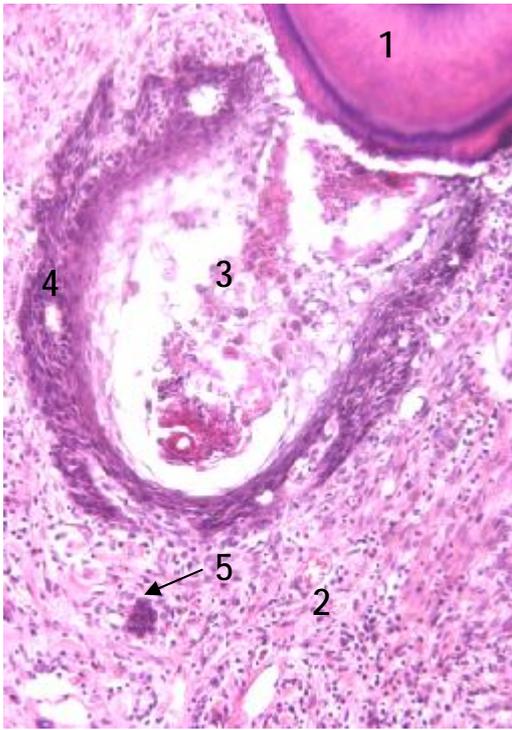
*a* — resorption of cement and dentin of the tooth root; *b* — resorption of the alveolar bone in the interdental septum; 1 — cement of the tooth root; 2 — dentin of the tooth root; 3 — root canal; 4 — periodontal ligament; 5 — alveolar bone; 6 — cement-dentin resorption; 7 — TRAP-positive cells (osteoclasts); 8 — periapical abscess. Magnifications  $\times 200$ . Coloring: hematoxylin

and TRAP-reaction



*Fig. 7.* Chronic apical periodontitis (apical granuloma); the line marks the length of the inflammatory focus:

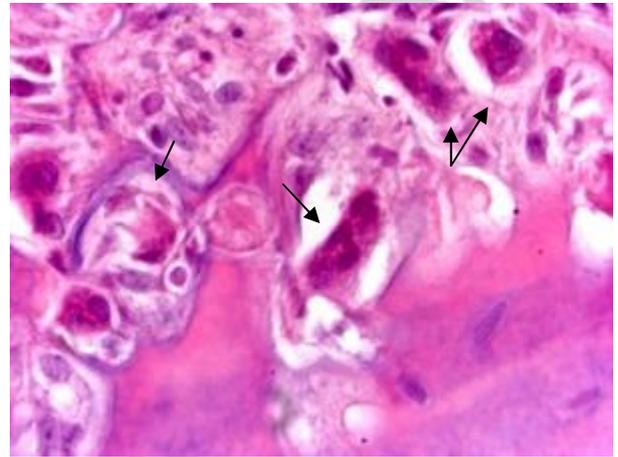
1 — cement of the tooth root; 2 — apical foramen; 3 — periodontal ligament; 4 — inflammatory focus. Magnifications  $\times 200$ . Coloring: hematoxylin and eosin



*Fig. 8. Radicular cyst:*

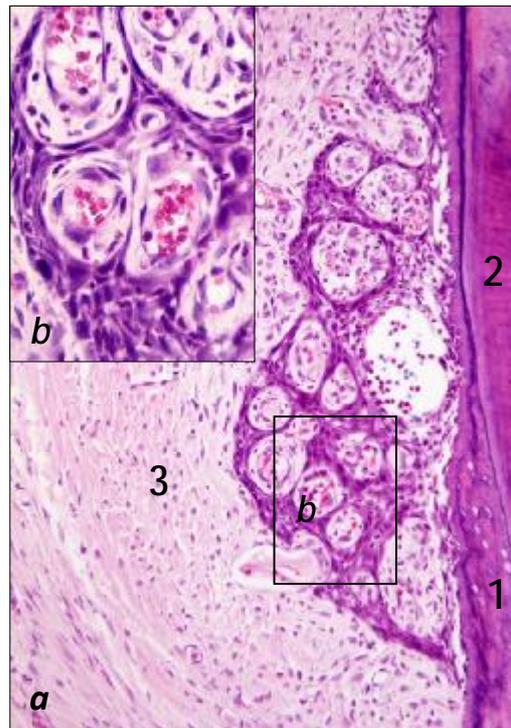
1 — root of the tooth; 2 — periodontal ligament; 3 — the contents of the cyst; 4 — cyst membrane; 5 — epithelial rests of Malassez. Magnifications  $\times 200$ . Coloring:

hematoxylinandeosin



*Fig. 9. Alveolar bone resorption, osteoclasts are indicated by arrows. Magnifications  $\times 1000$ .*

Coloring: hematoxylinandeosin



*Fig. 10. Complex granuloma:*

*a* — general view of the pathological formation, *b* — epithelial cells: 1 — cement of the tooth root; 2 — dentin of the tooth root; 3 — periodontal ligament. Magnifications *a* —  $\times 200$ ; *b* —  $\times 400$ . Coloring: hematoxylin and eosin

## CLASSIFICATION

The type of periapical periodontitis is usually classified according to whether it is an acute/symptomatic process or a chronic/asymptomatic process.

Acute periapical periodontitis, also termed acute apical periodontitis, acute periradicular periodontitis, or symptomatic periapical periodontitis.

Chronic periapical periodontitis, also termed chronic apical periodontitis, chronic periradicular periodontitis, or asymptomatic periapical periodontitis.

A periapical granuloma (also termed an apical granuloma or a radicular granuloma) is a mass of chronically inflamed [granulation tissue](#) that forms at the apex of the [root](#) of a [nonvital](#) (dead) tooth. However, a periapical granuloma does not contain [granulomatous inflammation](#), and, therefore, is

not a true [granuloma](#), but the term *periapical granuloma* is in common use (Wikipedia).

A relatively high incidence of periodontitis and serious difficulties of its diagnosis and treatment is especially necessary to develop a common classification. Even the classification of periodontitis proposed in the XIX century was based mainly on clinical signs. In 1891, O. K. Limberg systematized clinical signs of inflammation of the periodontium and proposed a classification.

In 1924, E. M. Gofung proposed a clinical and anatomical classification, which reflected localization and pathological changes in case of periodontal inflammation. The author has divided the processes that occur in the periodontium into acute and chronic.

#### I. Acute periodontitis.

1. Acute marginal periodontitis.
2. Acute apical periodontitis.
3. Acute diffuse periodontitis.

#### II. Chronic periodontitis.

1. Chronic fibrotic periodontitis.
2. Chronic granulomatous periodontitis.
3. Chronic granulating periodontitis.

However, the classification proposed in the early XX century incompletely revealed clinical features, which not always allowed to use it. I. G. Lukomski (1937) investigated the pathophysiological and pathomorphological changes in the periodontium in case of inflammation and proposed the classification that is now common in clinical practice. It allows a more detailed diagnosis of all forms of periodontitis and implement differential therapeutic measures. According to this classification, periodontitis is divided into 3 main groups:

#### I. Acute periodontitis (*periodontitis acuta*).

1. Acute serous periodontitis (*periodontitis acuta serosa*).
2. Acute suppurative periodontitis (*periodontitis acuta purulenta*).

#### II. Chronic periodontitis (*periodontitis chronica*).

1. Chronic fibrotic periodontitis (*periodontitis chronica fibrosa*).
2. Chronic granulomatous periodontitis (*periodontitis chronica granulo-matosa*).
3. Chronic granulating periodontitis (*periodontitis chronica granulans*).

### III. Exacerbations of chronic periodontitis.

#### Grossman's Classification

1. Acute periradicular diseases:
  - a. Acute apical periodontitis:
    - i. Vital;
    - ii. Nonvital.
  - b. Acute alveolar abscess.
  - c. Phoenix abscess.
2. Chronic periradicular diseases with areas of rarefaction:
  - a. Chronic apical periodontitis:
    - Chronic alveolar abscess;
    - Periapical granuloma;
    - Cystic apical periodontitis.
  - b. Persistent apical periodontitis.
3. Condensing osteitis.
4. External root resorption.
5. Diseases of the periradicular tissues of non-endodontic origin.

#### Ingle's Classification of Pulpoperiapical Pathosis

##### ***A. Painful Pulpoperiapical Pathosis***

1. Acute apical periodontitis
2. Advanced apical periodontitis
  - a. Acute apical abscess
  - b. Phoenix abscess
  - c. Suppurative apical periodontitis (chronic apical abscess)

##### ***B. Non-painful Pulpoperiapical Pathosis***

1. Condensing osteitis
2. Chronic apical periodontitis both incipient and advanced stages
3. Chronic apical periodontitis
  - a. Periapical granuloma
  - b. Apical Cyst
  - c. Suppurative apical periodontitis

International classification ICD-10

- K04 Diseases of pulp and periapical tissues
  - K04.4 Acute apical periodontitis of pulpal origin
    - Acute apical periodontitis NOS
  - K04.5 Chronic apical periodontitis
    - Apical granuloma
  - K04.6 Periapical abscess with sinus
    - Includes: dental abscess with sinus
    - dentoalveolar abscess with sinus
    - periodontal abscess of pulpal origin
    - K04.60 Sinus to maxillary antrum
    - K04.61 Sinus to nasal cavity
    - K04.62 Sinus to oral cavity
    - K04.63 Sinus to skin
    - K04.69 Periapical abscess with sinus unspecified
  - K04.7 Periapical abscess without sinus
    - Dental abscess without sinus
    - Dentoalveolar abscess without sinus
    - Periodontal abscess pulpal origin without sinus
    - Periapical abscess with no reference to sinus
  - K04.8 Radicular cyst

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CLASSIFICATION**

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