Volume 3, Issue 6, June 2025 ISSN (E): 2938-3765

DIFFERENTIATING DENTAL DISEASES WITH SIMILAR SYMPTOMS: A FOCUS ON PERIODONTITIS

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Abstract
This article addresses the challenge of differentiating dental diseases with similar clinical signs, using periodontitis as an example. Symptomatic similarities between periodontitis, pulpitis, endodontic-periodontal diseases, and gingival hypoplasia often complicate diagnosis. The article analyzes the possibilities of accurate and precise diagnosis using modern diagnostic methods such as clinical examination, X-rays, CBCT, thermotesting, and others[12]. The importance of differential diagnosis for distinguishing diseases and ensuring effective treatment is emphasized.

Keywords: Periodontitis, Pulpitis, Differential Diagnosis, Gingival Hypoplasia, Endodontic-Periodontal Diseases, Squamous Cell Carcinomas, Periapical Abscesses.

Introduction

Approximately 700 species of microorganisms colonize the human oral cavity. The bacteria residing in the oral cavity are primarily commensal, along with a sparse population of pathogenic bacteria. Periodontitis is one of the most common diseases affecting the teeth, leading to the disruption of the supporting structures and surrounding dental tissues. The term "periodontitis" is composed of two parts: "periodont-" meaning "the structure surrounding the teeth" and "itis" meaning "inflammation." Periodontitis initially arises from the gingival tissues, and if left untreated, the inflammation penetrates deeper tissues, altering bone homeostasis and resulting in tooth loss[1].

Periodontal disease has a multifactorial origin. The primary causative agent identified in periodontitis is the bacterial biofilm that grows on tooth surfaces. In addition to local factors, the development of the disease is influenced by plaque and calculus, genetics, environmental factors, the patient's systemic health, lifestyle habits, and various social determinants[2]. The harmful effects of periodontopathogens are not limited to the periodontium but also negatively impact the

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systemic health of patients. Periodontitis is a disease that develops as a result of the localization of the inflammatory process in the periodontal tissues[4].

Based on localization, periodontitis is classified into the following types:

Apical (apical) periodontitis — when inflammation is located at the tip of the tooth root;

Marginal periodontitis — when inflammation affects the tissues along the root;

Diffuse periodontitis — occurs when the entire attachment apparatus is affected.of dentin. It is associated with severe pain and a risk of pulp damage.

Etiology

The cause of periodontal inflammation is often infection. The infection can enter the periodontal spaces through the canal of the tooth root, where the pulp becomes inflamed and its root part is affected by the infection. The microbes and toxins from the inflamed pulp can also infiltrate the periodontal tissues, causing inflammation. Infectious periodontitis is a complication of untreated or improperly treated pulpitis[3]. The infection can spread to the periodontium from the pathological gingival pockets when periodontitis is present, as well as through the hematogenous route during severe infectious diseases (such as angina, scarlet fever, and influenza)[5].

Periodontitis can also develop as a result of a sudden mechanical force: a sudden blow, injury, or fall. In such cases, the anterior teeth are often affected. Severe damage can lead to the destruction of the periodontium, resulting in the infection of the dental pulp, which can subsequently spread to the apical periodontium[6]. Periodontitis can also develop from weak but persistent trauma. This can be caused by excessive fillings, crowns, or bridges. Such traumatic effects accumulate at the apex of the tooth root. Eliminating the cause of this chronic trauma helps in the healing of the periodontal area, preserving the vitality of the pulp in such teeth[7].

Periodontitis can also develop under the influence of chemicals used during dental manipulations. This often occurs with arsenic preparations (in excessive doses) and strong antiseptics (such as formaldehyde, phenol, etc.) when the filling material impacts the periapical tissues[8]. Allergic periodontitis can also develop, where patients have a high sensitivity to medications used in root canal treatment and filling. In addition to apical periodontitis, marginal periodontitis can also develop, which occurs when food debris (from needles, toothbrushes, etc.) is removed, damaging the surrounding connective tissue and leading to marginal (border) periodontitis. Marginal periodontitis can also develop when the dentist places fillings that extend beyond the edges or artificial crowns that penetrate deeply into the tooth neck. The cause of marginal periodontitis may be chemically active preparations[9]. For example, if an arsenic preparation is not adequately sealed, it can lead to necrosis of the dental papilla and the septum between the teeth.

Diagnosis

In radiographs, pathological changes in the bone tissue where the focus of inflammation is located may not be detected; however, sometimes the periodontal space may be widened due to swelling in that area. Results from electric pulp testing (EPT) indicate that the pulp has been destroyed. Peripheral blood analysis typically does not show significant changes; in some cases, a slight increase in the number of leukocytes (up to 10,000 per microliter) and an increase in the erythrocyte sedimentation rate (ESR) may be noted[11].

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Differential Diagnosis of Periodontitis:

Disease Name	Main Distinguishing Features from Periodontitis
Chronic Gangrenous Pulpitis	Strong pain is felt during probing through the tooth cavity and with heat application.
Acute Suppurative Periostitis (Jaw)	Pain is intense on palpation, with the presence of a periapical abscess.
Radicular Cyst	A clearly defined circular radiolucency is identified in the bone tissue on radiographs.
Moderate Caries	Probing reveals decay at the enamel-dentin junction, with no changes in the periapical tissues.

Pulpitis is inflammation of the pulp (nerve tissue), the inner tissue of the tooth, caused by microorganisms, injury, or chemical effects.

Etiology (Causes):

- Deepening caries reaching the pulp chamber.
- Mechanical or thermal injury to the tooth.
- Incorrect dental treatment (poor filling, antiseptics).
- Infection ascending through periodontal diseases (rarely).

Pathogenesis:

Bacteria or toxins enter the pulp and trigger an inflammatory reaction. Because the pulp chamber is enclosed, pressure increases, causing severe pain. Without treatment, this process ends with necrosis (tissue death).

Classification:

1. **Acute pulpitis:**
   - Serous (initial stage)
   - Acute purulent (progressive)

2. **Chronic pulpitis:**
   - Chronic fibrous (fibrinous)
   - Chronic purulent
   - Chronic gangrenous (putrefactive)

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Differences Between Pulpitis and Periodontitis:

Distinguishing Features	Pulpitis	Periodontitis
Pain	Intensifies at night	Constant, worsens when pressure is applied to the tooth
Percussion	Usually painless	Very painful
Electric Pulp Test (EPT)	No response or low response	Pulp is necrotic (dead)
Radiograph	Normal or shows caries	Widened periodontal space

1. Periapical Abscess:
Periapical abscesses are localized infections around the tooth root, often resulting from pulp diseases. Clinical and radiographic signs may resemble periodontitis but have distinct differences.

2. Endodontic-Periodontal Diseases:
In these cases, inflammation of both the pulp and periodontium occurs simultaneously. Clinical signs such as pain and swelling may be associated with both sources.

3. Leukemia and Drug Effects:
In leukemia, painless gingival hypertrophy can resemble periodontitis. Additionally, drugs such as calcium channel blockers, immunosuppressants, and anticonvulsants can cause gingival hypertrophy.

4. Squamous Cell Carcinoma:
Squamous cell carcinoma is a severe cellular tumor whose symptoms can be confused with periodontitis. In such cases, the teeth do not respond to any mechanical or pharmacological treatment.

CLASSIFICATION
According to the International Classification of Diseases (ICD-10):

- **K04.4** Acute apical periodontitis (originating from pulp).
- **K04.5** Chronic apical periodontitis (apical granuloma).
- **K04.6** Purulent apical abscess (with fistula): dentoalveolar and dentogenic.
  - **K04.60** Fistula connected to the maxillary sinus.
  - **K04.61** Fistula connected to the nasal cavity.
  - **K04.62** Fistula connected to the oral cavity.
  - **K04.63** Fistula connected to the skin.
  - **K04.69** Unspecified fistulous periapical abscess.

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- **K04.7** Periapical abscess without fistula (dentoalveolar abscess, dentogenic abscess).
- **K04.8** Root cyst (apical, periodontal, and periapical):
  - **K04.80** Apical and lateral cyst.
  - **K04.81** Residual cyst.
  - **K04.82** Inflammatory paradental cyst.
  - **K04.89** Unspecified root cyst.
- **K04.9** Other diseases of pulp and periapical tissues.

Treatment

If preserving the affected tooth is deemed appropriate (i.e., if the crown portion is significantly intact, the root canal is accessible, and complete endodontic opening is possible), the following procedures are performed:

1. Opening the Focus of Inflammation: Create conditions for the continuous drainage of exudate.
2. Opening the Root Canal: If the canals are blocked, short, or curved, or obstructed by foreign bodies, and complete endodontic access is not possible, tooth extraction is recommended.
3. Anesthesia: The procedure is conducted under infiltration anesthesia using amide-type anesthetics such as articaine (Ultrakain D-S, Ultrakain D-S forte). These preparations have a short latency period (approximately 3 minutes), are well-accepted by tissues, and are 6 times more potent than novocaine and 3 times more potent than lidocaine and trimethocaine. Additionally, their anesthetic effect lasts longer and has lower toxicity.

In cases where tooth extraction is necessary (especially against a background of inflammation), curettage of the socket is not recommended, as it can disrupt the demarcation zone and lead to the spread of infection into the bone tissue. Instead, the following procedures are recommended:

- Rinsing the Tooth Socket: Use warm antiseptic solutions.
- Novocaine Blockades: Apply 2-3 times with 5-7 ml of 0.5% novocaine solution.
- Warm Rinsing Baths: Use antiseptics or herbal infusions in the mouth (however, hypertonic solutions like salt or soda are not recommended, as they can act as strong chemical irritants and exacerbate inflammation).
- Physiotherapy: Utilize UHF (ultrahigh frequency therapy), helium-neon laser irradiation (HNL), and aeroionotherapy.

Complex Treatment Includes:

- **Analgesics:** Analgin*, Pentalgin*, Nurofen*, Solpadein*.
- **Non-Steroidal Anti-Inflammatory Drugs (NSAIDs):** Aspirin*, Ketonal*, Diclofenac*.
- **Antihistamines:** Dimedrol*, Suprastin*, Tavegil*, Pipolfen*.
- **Circulation Enhancers:** Dimefosfon*, Dibazol*.
- **Vitamins** and **Immunostimulants**.

Acute periodontitis typically presents with a normergic inflammatory reaction, so there is usually no need to prescribe antibiotics and sulfanilamides. However, in patients with weakened immunity, a sluggish inflammatory response, or when the disease is complex with general intoxication of the body, antibacterial therapy may be employed to prevent the spread of inflammation to other tissues[10].

In such cases, the use of immunostimulators and immunomodulators is advisable. The final outcome of the disease is generally positive. Properly conducted treatment often concludes with

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recovery. However, if endodontic or general treatment is improperly performed, this process may transition into a chronic stage[13].

Global Statistics

In 2021, the number of individuals affected by severe periodontitis was approximately **1.06695 billion** (95% confidence interval: **896.55–1.23484 billion**), representing an age-standardized prevalence rate of **12.5%**. [PubMed, BioMed Central]

The global prevalence rate of periodontitis in 2021 was **17,011.6 cases per 100,000** population. [BioMed Central, PMC, iv.iiarjournals.org, The Washington Post, PubMed]

Forecast for 2050: The number of individuals with severe periodontitis is expected to exceed **1.5 billion**.

European Statistics

In the European region, the age-standardized prevalence rate of severe periodontitis was highest in 2021.

In Spain, the prevalence of severe periodontitis was **4.3%** in 2010, while in Italy, it was **13.1%**. [efp.org]

Asian Statistics

In South Asia, the prevalence rate of severe periodontitis was **17.57%** (14.73–20.14%) in 2021. [PubMed, Online Library]

In China, the number of individuals affected by severe periodontitis in 2021 was **220,238,287 cases**. [ScienceDirect, The Washington Post, PubMed]

Central Asian Statistics

Sierra Leone, Gambia, and Cape Verde reported the highest prevalence rates of periodontitis in 2021.

Statistics for Uzbekistan

The estimated prevalence rate of severe periodontitis among the population aged 15 and older was **6.4%** in 2021. [PubMed, efp.org]

In the Fergana region, the prevalence rate of periodontitis was **14.2%** in 2021.

Conclusion

The clinical manifestations of dental diseases can often be similar in many cases. Distinguishing between the signs of periodontitis, pulpitis, endodontic-periodontal diseases, and other oral pathologies is a crucial and complex issue. The analyses presented in this article indicate that modern diagnostic tools, particularly methods such as EOD (Electric Pulp Testing), thermal tests, and radiography, when used alongside clinical details, enable clear differentiation between diseases [14].

Differentiating periodontitis from other diseases is not only vital for determining the treatment strategy but also plays a crucial role in preserving the tooth, alleviating the patient's painful

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condition, and preventing complications. Additionally, the article emphasizes the etiology and pathogenesis of these diseases, providing an in-depth discussion of the factors that contribute to their development [15].

Therefore, every dentist must possess knowledge of modern differential diagnostics, approach each clinical case individually, and identify diseases based not only on symptoms but also on a comprehensive analysis. This approach allows for not only accurate diagnosis but also effective and complete treatment.

References

1. Aas JA, Paster BJ, Stokes LN, Olsen I, Dewhirst FE. Identification of the normal bacterial flora of the oral cavity. J Clin Microbiol. November 2005; 43(11): 5721-32. [PMC free article] [PubMed] [Data list]
2. Hajishengallis G, Darveau RP, Curtis MA. The keystone-pathogen hypothesis. Nat Rev Microbiol. October 2012; 10(10): 717-25. [PMC free article] [PubMed] [Reference list]
3. Petersen PE, Baehni C. Periodontal health and the global community. Periodontol 2000. October 2012; 60(1): 7-14. [PubMed] [Data list]
4. L. Yu. Yusupova, I. Kh. Ataullaeva. Dental Diseases: A textbook for medical colleges. Tashkent - "ILM ZIYO" - 2015, pp. 51-55, 56-63.
5. Pediatric Therapeutic Dentistry: National Guidelines / Ed. B. K. Leontiev, JI. II. Kiselnikov. - 2nd ed. revised. GEOTAR-Media. 2021. - and additional. - Moscow, 952 p. (Series "National Guidelines") ISBN 978-5-9704-8173-0, Chapters 27-29 on periodontitis. L.P. Kiselnikov, N.Yu. Basilieva, V.G. Alpatov.
6. Chronic Stomatology: Textbook / V.V. Afanasyev [et al.]; Ed. B. Afanasyev. 3rd ed., revised. M.: GEOTAR-Media, 2021. 400 p.: Ill. ISBN 978-5-9704-6080-1, Chapter 7 on Inflammatory Diseases of the Jaw-Face Area (T.T. Faizov, V.V. Afanasyev).
7. Surgical Dentistry: A textbook for the accreditation of specialists / Ed. E.A. Bazikyan. - Moscow: GEOTAR-Media, 2023. - 832 p.: Ill. - DOI: 10.33029/9704-7471-6HSJ-2023-1-832. ISBN 978-5-9704-7471-6, Chapter 6 on Odontogenic Inflammatory Diseases 6.1 on Periodontitis.
8. M.I. Azimov. JARROHLIK STOMATOLOGIYA PROPEDEVTIKASI textbook. Tashkent – 2009, p. 81.
9. National Library of Medicine, National Center for Biotechnology Information. Neha Mehrotra1; Saurabh Singh2. 1’2 Integral Institute of Medical Sciences. Article on Periodontitis.
10. Peres MA, et al. "Burden of severe periodontitis and edentulism in 2021, with projections up to 2050: The Global Burden of Disease 2021 study." Journal of Periodontal Research. 2024. [PubMed, BioMed Central]
11. Wang Y, et al. "Global burden of periodontal diseases among the working-age population: A systematic analysis." BMC Public Health. 2025. [BioMed Central]
12. "Oral Health Country Profile: Uzbekistan." World Health Organization. 2022. [cdn.who.int]
13. "Prevalence of Periodontitis Among The Population: Findings From a Study in Fergana, Uzbekistan." World of Science Journal. 2025. [journals.innoscie.com]

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

14. "Economist report shows prevalence of periodontitis unchanged over 20 years." European Federation of Periodontology. 2021.
15. "Global burden and trends of oral disorders among adolescent and young adult populations: A systematic analysis." The Lancet. 2024. [PMC, Nature]

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