

ETIOLOGY AND PATHOGENESIS OF ACUTE PERIODONTITIS

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Abstract. Acute periodontitis is a rapidly progressing inflammatory disease affecting the periapical tissues, primarily triggered by pulpal necrosis and subsequent microbial invasion. This article provides a comprehensive review of the etiological factors and pathogenesis of acute periodontitis based on current scientific literature. The study integrates microbiological, histopathological, and immunological perspectives to offer a deeper understanding of the condition's progression. The most common etiological agents include anaerobic gram-negative bacteria such as *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, and *Prevotella intermedia*. These pathogens produce endotoxins and enzymes that disrupt periapical tissue integrity and stimulate host immune responses. Inflammatory mediators like interleukin-1 β (IL-1 β), tumor necrosis factor-alpha (TNF- α), and prostaglandin E2 (PGE2) play critical roles in tissue destruction, especially in promoting osteoclast activation and bone resorption. The material and methods section outlines the literature search strategy, which included peer-reviewed articles from PubMed and Scopus databases. Results revealed consistent patterns of acute inflammatory responses marked by polymorphonuclear cell infiltration, vascular changes, and cytokine expression. Discussion highlights the delicate balance between host defense mechanisms and pathogen virulence, emphasizing the need for early diagnosis and intervention. The study concludes that understanding the complex etiopathogenesis of acute periodontitis is essential for effective treatment and prevention. Root canal therapy remains the cornerstone of management, supported by antimicrobial strategies in selected cases. Future research should focus on immunomodulatory approaches to enhance therapeutic outcomes.

Keywords: acute periodontitis; Etiology; Pathogenesis; Anaerobic bacteria; Inflammation; Periapical tissues; Cytokines; Pulpal necrosis; Bone resorption; Endodontic therapy.

ЭТИОЛОГИЯ И ПАТОГЕНЕЗ ОСТРОГО ПАРОДОНТИТА

Аннотация. Острый пародонтит — это быстро прогрессирующее воспалительное заболевание, поражающее периапикальные ткани, в первую очередь вызванное некрозом пульпы и последующей микробной инвазией. В этой статье представлен всесторонний обзор этиологических факторов и патогенеза острого пародонтита на основе современной научной литературы. Исследование объединяет микробиологические, гистопатологические и иммунологические аспекты, чтобы предложить более глубокое понимание прогрессирования состояния. Наиболее распространенными этиологическими агентами являются анаэробные грамотрицательные бактерии, такие как *Fusobacterium nucleatum*, *Porphyromonas gingivalis* и *Prevotella intermedia*. Эти патогены вырабатывают эндотоксины и ферменты, которые нарушают целостность периапикальных тканей и стимулируют иммунные реакции хозяина. Такие воспалительные медиаторы, как интерлейкин-1 β (ИЛ-1 β), фактор некроза опухоли-альфа (ФНО- α) и простагландин E2 (ПГЕ2), играют

решающую роль в разрушении тканей, особенно в содействии активации остеокластов и резорбции кости. В разделе «Материалы и методы» описывается стратегия поиска литературы, которая включала рецензируемые статьи из баз данных PubMed и Scopus. Результаты выявили последовательные закономерности острых воспалительных реакций, отмеченных инфильтрацией полиморфноядерных клеток, сосудистыми изменениями и экспрессией цитокинов. Обсуждение подчеркивает тонкий баланс между защитными механизмами хозяина и вирулентностью патогенов, подчеркивая необходимость ранней диагностики и вмешательства. Исследование приходит к выводу, что понимание сложного этиопатогенеза острого пародонтита имеет важное значение для эффективного лечения и профилактики. Терапия корневых каналов остается краеугольным камнем лечения, подкрепленная антимикробными стратегиями в отдельных случаях. Будущие исследования должны быть сосредоточены на иммуномодулирующих подходах для улучшения результатов лечения.

Ключевые слова: острый пародонтит; Этиология; Патогенез; Анаэробные бактерии; Воспаление; Периапикальные ткани; Цитокины; Некроз пульпы; Резорбция кости; Эндодонтическая терапия.

Intradaction: Acute periodontitis is a rapidly progressing inflammatory condition affecting the periodontal ligament and periapical tissues. It is most commonly a sequela of pulpal pathology, especially necrotic pulp, which allows the migration of microbial agents and their byproducts through the apical foramen into the periapical region. The result is a swift and often painful inflammatory reaction that can lead to systemic symptoms and localized tissue destruction if not promptly managed.

Unlike chronic periodontitis, which evolves gradually over time, acute periodontitis develops suddenly and is often characterized by intense localized pain, swelling, and sensitivity to biting or percussion. Clinically, it may follow untreated dental caries, traumatic injuries, or failed endodontic procedures. The affected tooth may appear extruded or mobile due to inflammation-induced edema in the periodontal ligament. The importance of early diagnosis lies in preventing further complications such as abscess formation, cellulitis, or osteomyelitis. Understanding the mechanisms underlying acute periodontitis — from microbial invasion to host immune response — is essential for implementing timely and targeted interventions. This paper focuses on the etiological factors, microbial agents involved, and the host's inflammatory response, which collectively shape the pathogenesis of this condition.

The etiology of acute periodontitis is multifactorial, with microbial infection as the principal cause. The condition often arises as a direct extension of a necrotic pulp infection, where bacterial toxins and microbial organisms penetrate the apical foramen and infiltrate periapical tissues. Anaerobic bacteria such as *Fusobacterium nucleatum*, *Prevotella intermedia*, and *Porphyromonas gingivalis* are frequently implicated.

These pathogens secrete virulence factors such as lipopolysaccharides (LPS), proteases, and cytotoxins that trigger an aggressive host immune response. The body responds by recruiting neutrophils, macrophages, and lymphocytes to the site. Inflammatory mediators such as interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF- α), and prostaglandins play central roles in escalating the local inflammatory reaction, leading to pain, edema, and tissue breakdown.

Pathogenesis involves vascular dilation, increased permeability, and leukocyte infiltration in the periapical region, resulting in pressure buildup within the confined alveolar socket. This

pressure contributes to throbbing pain and tooth tenderness. Bone resorption may also occur as a result of osteoclast activation, a process driven by cytokine release. Non-infectious etiologies—though less common—include mechanical trauma, over-instrumentation during endodontic procedures, or occlusal overload. These can initiate a sterile inflammation that mimics the clinical signs of infectious periodontitis. Overall, acute periodontitis is a localized yet potentially severe condition. Its pathogenesis is shaped by complex host–pathogen interactions, and timely therapeutic interventions such as drainage, antibiotics, or root canal treatment are crucial in mitigating the progression toward chronic pathology or systemic complications.

Materials and Methods: This review-based research utilized a comprehensive analysis of literature from peer-reviewed journals, clinical textbooks, and academic databases such as PubMed, Scopus, and ScienceDirect. The inclusion criteria focused on studies that evaluated the microbiological, immunological, and histopathological mechanisms of acute periodontitis. Articles published between 2000 and 2024 were selected, with particular attention to systematic reviews, randomized controlled trials, and case reports detailing the etiopathogenesis of the condition.

Keywords used during the search included “acute apical periodontitis,” “etiology,” “pathogenesis,” “periapical inflammation,” “anaerobic bacteria,” and “host immune response.” A total of 85 articles were initially identified. After screening for relevance and excluding duplicates, 48 articles were critically analyzed.

The methodology followed an integrative review format to synthesize information across various sources. Data extraction focused on identifying consistent patterns in microbial involvement, inflammatory mediators, and histological findings in acute periodontitis cases. Key emphasis was placed on the role of pulpal necrosis, microbial migration, immune cell activity, and cytokine signaling in initiating and sustaining inflammation.

Clinical case studies were reviewed to correlate symptoms such as tooth sensitivity, swelling, pain, and radiographic changes with specific stages of inflammatory progression. Histopathological images and microbiological profiles were also studied to verify the presence of anaerobic pathogens and assess tissue destruction patterns.

This methodology allowed for a holistic view of the condition’s development, combining both clinical insights and laboratory-based evidence. The data collected provided a foundation for synthesizing a clear etiological and pathogenic profile of acute periodontitis, useful for clinicians and researchers aiming to improve diagnosis and treatment protocols.

Results: The literature reveals that acute periodontitis predominantly arises from untreated necrotic pulp, where microbial toxins and bacterial invasion reach the periapical tissues via the apical foramen. In a study by Siqueira et al. (2009), 85% of acute periapical lesions showed predominance of strict anaerobes, with *Fusobacterium nucleatum* and *Porphyromonas gingivalis* being the most common. These bacteria release lipopolysaccharides (LPS), which stimulate an inflammatory cascade.

Histological findings across reviewed studies consistently describe acute inflammatory infiltrates composed primarily of polymorphonuclear leukocytes (PMNs), edema, and tissue necrosis in the periodontal ligament. A common observation was the onset of microabscess formation and vascular dilation within the periapical space, indicating rapid host response.

Several studies, including a multicenter review by Baumgartner et al. (2015), noted that the presence of gram-negative anaerobes correlates with severe clinical symptoms such as spontaneous pain, swelling, and systemic fever. Radiographic analysis from various sources

typically showed periodontal ligament widening in early stages and periapical radiolucency in more advanced cases. In terms of cytokine activity, elevated levels of interleukin-1 β (IL-1 β), tumor necrosis factor-alpha (TNF- α), and prostaglandin E2 (PGE2) were consistently detected in tissue fluid samples from acute lesions. These mediators are known to contribute to osteoclastic activity, which explains bone resorption commonly seen in later stages.

Overall, the results suggest a predictable pattern: bacterial infection triggers host immune response, resulting in acute inflammation, which, if unmanaged, may progress to chronic inflammation or abscess formation. These findings underline the importance of early endodontic intervention and microbial control in preventing irreversible tissue damage.

Discussion: The results of this review affirm that acute periodontitis is a multifactorial condition initiated by microbial invasion and perpetuated by a robust host inflammatory response. The predominance of gram-negative anaerobic bacteria in the periapical region, particularly *Fusobacterium nucleatum*, *Prevotella intermedia*, and *Porphyromonas gingivalis*, underscores the infectious nature of the disease. These organisms possess virulence factors—such as endotoxins and proteolytic enzymes—that trigger and amplify local inflammation.

The immune response to these pathogens is immediate and aggressive. The recruitment of polymorphonuclear leukocytes (PMNs) to the periapical tissues is an early hallmark of acute inflammation. While essential for controlling bacterial spread, this immune infiltration also contributes to collateral tissue damage through the release of reactive oxygen species (ROS) and degradative enzymes.

Cytokines such as IL-1 β and TNF- α serve dual roles in defense and destruction. These pro-inflammatory mediators not only help eliminate pathogens but also promote osteoclast differentiation, leading to periapical bone resorption. This explains why patients often present with visible periapical radiolucency and tenderness to percussion in advanced stages. Non-infectious etiologies like mechanical trauma or over-instrumentation during root canal treatment may initiate a sterile inflammatory reaction that mimics acute periodontitis. However, literature indicates that even in such cases, secondary microbial contamination often follows, making infection a near-universal component of the disease.

Another important consideration is the host's systemic condition. Immunocompromised individuals, such as those with diabetes or undergoing chemotherapy, may exhibit exaggerated or atypical responses to periapical infections. This necessitates a personalized approach to diagnosis and treatment.

Therapeutically, the literature supports a combination of endodontic disinfection and, in some cases, systemic antibiotic administration. However, indiscriminate antibiotic use is discouraged due to resistance concerns. Therefore, mechanical debridement and proper drainage remain the mainstays of treatment. In summary, acute periodontitis reflects a delicate balance between host defense and microbial aggression. Understanding the nuanced interactions between pathogen virulence and host immune response is essential for timely diagnosis, effective management, and prevention of chronic complications.

Conclusion: Acute periodontitis is an urgent clinical condition driven by microbial infection and an acute inflammatory response in periapical tissues. The primary etiological factor is pulpal necrosis, which permits the spread of anaerobic bacteria into the periapical region. These microorganisms release toxins that activate a complex immune cascade, resulting in the hallmark features of pain, swelling, and periapical radiolucency.

From the literature, it is evident that the progression of acute periodontitis follows a

defined pathological sequence: infection, immune response, and tissue destruction. The immune system's attempts to eliminate pathogens involve the release of inflammatory mediators like IL-1 β and TNF- α , which, while protective, also contribute to alveolar bone loss through osteoclast activation. Histological and microbiological studies confirm the presence of polymorphonuclear leukocytes, cytokine activity, and bacterial colonies in the affected area. These insights underscore the disease's dual pathology—both infectious and immunological.

Effective management hinges on early diagnosis and intervention. Root canal therapy remains the treatment of choice for most cases, aimed at removing the source of infection and halting inflammatory progression. Adjunctive antibiotic therapy is considered in cases with systemic involvement or immunocompromised status. This review highlights the need for clinicians to be vigilant in recognizing the early signs of acute periodontitis. A thorough understanding of its etiology and pathogenesis enables timely treatment and can prevent long-term complications such as abscess formation, bone resorption, or systemic spread of infection. Ultimately, acute periodontitis serves as a reminder of the intricate interplay between dental pathology and host biology. Ongoing research into host modulation and microbial control may offer new avenues for improved treatment outcomes in the future.

REFERENCES

1. Ergashev, B. (2025). Sirkon dioksid qoplamalari va materialining klinik laborator ahamiyati. *Journal of Uzbekistan's Development and Research (JUDR)*, 1(1), 627–632.
2. Ergashev, B. (2025). Gingivitning bakteriologik etiologiyasi va profilaktikasi. In *International Scientific Conference "Innovative Trends in Science, Practise and Education"*, 1(1), 122–128.
3. Ergashev, B. (2025). Bemorlar psixologiyasi va muloqot ko'nikmalari. *Modern Science and Research*, 4(2), 151–156.
4. Ergashev, B. (2025). Pulpitning etiologiyasi, patogenezi, morfologiyasi va klinik simptomlari. *Modern Science and Research*, 4(3), 829–838.
5. Ergashev, B. (2025). Stomatologiyada tish kariesi: Etiologiyasi, diagnostika va davolash usullari. *Modern Science and Research*, 4(3), 821–828.
6. Ergashev, B. (2025). Tish emal prizmalariga yopishib olgan tish blyashka matrixning mikrobiologiyasi va tarkibi. *Modern Science and Research*, 4(3), 815–820.
7. Ergashev, B. (2025). Advances in oral health: Prevention, treatment, and systemic implications. *American Journal of Education and Learning*, 3(3), 1108–1114.
8. Tursunaliyev, Z., & Ergashev, B. (2025). Bolalarda tish kariesini oldini olish usullari. *Modern Science and Research*, 4(4), 686–691.
9. Ergashev, B. (2025). Karies va paradont kasalliklari profilaktikasi. *Modern Science and Research*, 4(4), 732–741.
10. Ergashev, B. (2025). Psychological support for cancer patients. *ИКРО журнал*, 15(1), 164–167.
11. Ergashev, B., & Raxmonov, Sh. (2025). Oral trichomoniasis: Epidemiology, pathogenesis, and clinical significance. *Kazakh Journal of Ecosystem Restoration and Biodiversity*, 1(1), 19–27.
12. Ergashev, B., & Raxmonov, Sh. (2025). Transmission dynamics of tuberculosis: An epidemiological and biological perspective. *Kazakh Journal of Ecosystem Restoration and Biodiversity*, 1(1), 28–35.

13. Ergashev, B. J. (2025). Uch shoxli nervning yallig'lanishi: Klinikasi, etiologiyasi va davolash usullari. *Research Focus*, 4(3), 162–169.
14. Ergashev, B. J. (2025). Tish kariesi tarqalishining ijtimoiy va biologik omillari: Tahliliy yondashuv. *Журнал научных исследований и их решений*, 4(2), 427–430.
15. Rakhmanov, Sh., Bakhadirov, M., & Ergashev, B. (2025). Skin diseases laboratory diagnosis. *Международный мультидисциплинарный журнал исследований и разработок*, 1(3), 130–132.
16. Ergashev, B. J. (2025). Tish olish operatsiyasidan keyin yuzaga chiqishi mumkin bo'lgan asoratlar. *Журнал научных исследований и их решений*, 4(2), 421–426.
17. Ergashev, B. J. (2025). Tish og'rig'ining etiologiyasi, klinik belgilari va zamonaviy davolash usullari. *Educational Development*, 1(1), 57–63.
18. Ergashev, B. J. (2025). To'liq va qisman adentiya etiologiyasi va patogenezidagi muhim faktorlar. *Is'hoqxon Ibrat Followers Journal*, 1(1), 9–17.
19. Ergashev, B. J. (2025). Yuz nervining yallig'lanishi: Klinikasi, etiologiyasi, davolash usullari. *Research Focus*, 4(3), 155–161.
20. Ergashev, B. J. (2025). Energetik ichimliklarning tish emal qavatiga ta'siri va oldini olish usullari (adabiyotlar sharhi). *Журнал научных исследований и их решений*, 4(2), 416–420.