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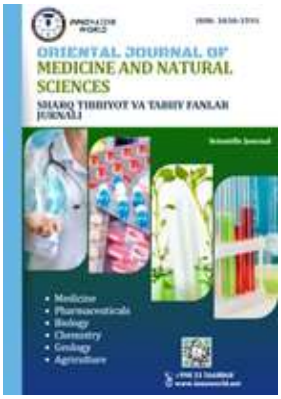

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CHRONIC GANGRENOUS PULPITIS: ETIOLOGY, CLINICOPATHOLOGICAL CHARACTERISTICS AND PATHOGENESIS

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Abstract: Chronic gangrenous pulpitis represents an advanced degenerative-inflammatory condition of the dental pulp characterized by extensive tissue necrosis and putrefactive changes. From a theoretical and scientific perspective, this pathology develops as a consequence of prolonged microbial invasion, compromised pulpal microcirculation, and progressive hypoxia within the confined pulp chamber. The present article provides a comprehensive theoretical analysis of chronic gangrenous pulpitis based exclusively on anatomical, histological, hydrodynamic, and statistical data derived from established scientific literature. Clinical case descriptions and patient-based observations are deliberately excluded to ensure a purely conceptual and evidence-based approach. Particular attention is given to the etiological factors that disrupt pulpal homeostasis, the molecular and cellular mechanisms underlying pulpal gangrene, and the anatomical peculiarities that predispose the pulp to irreversible degeneration. Furthermore, the article synthesizes findings from peer-reviewed articles, dissertations, and classical endodontic theories to elucidate the structural and functional alterations observed in chronic gangrenous pulpitis. By integrating hydrodynamic concepts of pulpal pressure, vascular collapse, and toxin diffusion, this review aims to clarify the pathogenesis of pulpal gangrene and its implications for dental hard tissues and periapical structures. The study contributes to a deeper theoretical understanding of chronic gangrenous pulpitis and provides a structured scientific basis for future experimental and translational research in endodontic pathology.

Keywords: chronic pulpitis, pulp gangrene, dental pulp, necrosis, microcirculation, hypoxia, inflammation, anaerobic bacteria, dentin permeability, toxins, histopathology, endodontics.

Intradaction: The dental pulp is a specialized connective tissue enclosed within the rigid walls of dentin and enamel, rendering it highly sensitive to environmental and pathological changes. Chronic gangrenous pulpitis represents one of the terminal stages of pulpal disease, arising from sustained inflammatory insults that exceed the adaptive capacity of pulpal tissues. Unlike acute inflammatory conditions, this form progresses gradually, often

without dramatic symptomatology, allowing extensive structural and functional deterioration over time.

From an anatomical standpoint, the pulp consists of a rich vascular network, nerve fibers, fibroblasts, odontoblasts, and immune cells. The limited collateral circulation within the pulp chamber creates a vulnerable microenvironment where even minor disturbances in blood flow can lead to hypoxia. The hydrodynamic theory of pulpal pathology emphasizes the role of altered intrapulpal pressure and impaired venous outflow in the development of ischemic damage. Over time, these changes result in irreversible degeneration and tissue death.

Etiologically, chronic gangrenous pulpitis is most frequently associated with untreated dental caries that permit bacterial toxins and metabolic byproducts to penetrate dentinal tubules. These substances initiate a prolonged inflammatory response that compromises cellular metabolism and enzymatic balance within the pulp. In contrast to acute pulpitis, where inflammatory exudate increases intrapulpal pressure and elicits pain, chronic gangrenous pulpitis often involves partial pulp necrosis that decompresses the chamber, masking overt symptoms.

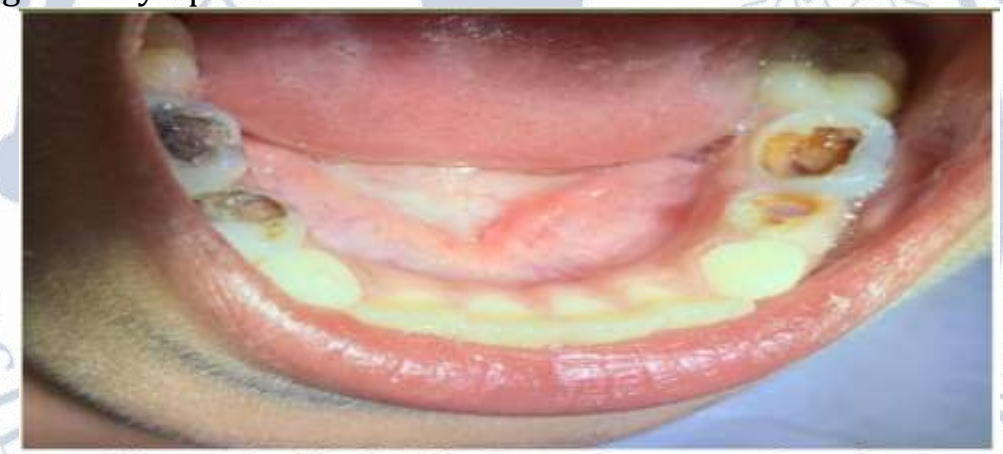

Figure 1: Clinical intraoral aspect, showing numerous extensive caries lesions
Source: Authors, 2024.

Microbiological studies have demonstrated that anaerobic bacteria dominate the necrotic pulp environment, producing proteolytic enzymes and volatile sulfur compounds responsible for tissue degradation. The persistence of these microorganisms further inhibits reparative processes and promotes gangrenous transformation. Statistically, epidemiological analyses indicate that molars and premolars are more commonly affected due to their complex anatomy and higher caries susceptibility.

This article aims to present a comprehensive theoretical overview of chronic gangrenous pulpitis, focusing exclusively on anatomical, hydrodynamic, histological, and statistical data derived from scientific literature. By excluding clinical case descriptions, the discussion remains centered on fundamental mechanisms governing disease progression. Such an approach is essential for refining conceptual frameworks in endodontic pathology and supporting evidence-based preventive strategies.

Materials and Methods: This theoretical review was developed through a structured search and analysis of scientific literature available in established academic databases. Electronic sources including PubMed, Scopus, Web of Science, Google Scholar, and ScienceDirect were utilized to identify peer-reviewed articles, systematic reviews, dissertations, and authoritative textbooks related to chronic pulpitis and pulpal necrosis.

Keywords and Boolean combinations such as "chronic gangrenous pulpitis," "pulp necrosis," "pulpal ischemia," "dental pulp pathology," and "hydrodynamic theory of pulp inflammation" were employed. Inclusion criteria encompassed publications focusing on anatomical structure, histopathological changes, microbial ecology, vascular dynamics, and epidemiological trends. Studies involving direct patient data, clinical trials, or case reports were deliberately excluded to maintain a purely theoretical scope.

Classical histological descriptions from foundational endodontic literature were integrated with contemporary molecular and microbiological findings. Dissertations and academic theses were included when they provided in-depth theoretical or experimental insights into pulp degeneration mechanisms. Data extraction emphasized structural alterations, biochemical pathways, and statistical prevalence patterns rather than therapeutic outcomes.

The collected materials were synthesized using a narrative analytical approach. Conceptual models of pulpal disease progression were compared across sources to identify common mechanistic themes. No quantitative meta-analysis was performed, as the objective was to construct a comprehensive theoretical framework rather than evaluate intervention efficacy.

Results: Analysis of the reviewed literature reveals that chronic gangrenous pulpitis is fundamentally a degenerative-inflammatory process driven by sustained ischemia and microbial activity. Anatomically, studies consistently report early disruption of the odontoblastic layer, followed by fibroblast degeneration and collapse of the extracellular matrix. Capillary stasis and thrombosis are frequently described as precursors to pulpal necrosis.

Histopathological findings indicate that necrosis initially develops in localized areas before progressing to involve the majority of the pulp chamber.

Coagulative necrosis predominates in the coronal pulp, while liquefactive necrosis is more common apically due to higher enzymatic activity. The accumulation of necrotic debris creates an anaerobic environment favorable to obligate anaerobes. Microbiological analyses demonstrate a predominance of gram-negative anaerobic species, including Porphyromonas and Prevotella, which produce endotoxins and proteases.

These compounds inhibit fibroblast proliferation and suppress angiogenesis, preventing reparative dentin formation. The hydrodynamic balance within the pulp is further compromised as venous outflow obstruction leads to chronic hypoxia. Statistical data from population-based studies suggest that

chronic gangrenous pulpitis accounts for a significant proportion of non-vital teeth extracted worldwide. Higher prevalence rates are observed in regions with limited preventive dental services and among older age groups. Posterior teeth exhibit greater susceptibility due to their complex root canal systems and increased occlusal stress.

Figure 2: Clinical aspect of the pulp polyp
Source: authors, 2024

Dissertation-based experimental models support the theory that prolonged low-grade inflammation allows partial tissue breakdown without triggering acute nociceptive responses.

This explains the chronicity and often asymptomatic nature of the disease. Collectively, the findings emphasize that chronic gangrenous pulpitis is not a sudden pathological event but the result of cumulative structural and metabolic failure.

Discussion: The synthesis of anatomical, histological, and hydrodynamic data underscores the multifactorial nature of chronic gangrenous pulpitis. The confined anatomy of the pulp chamber plays a central role in disease progression by limiting compensatory vascular responses. Unlike other connective tissues, the pulp lacks sufficient collateral circulation, making it exceptionally vulnerable to ischemic injury. The hydrodynamic theory provides a coherent explanation for the transition from reversible inflammation to irreversible necrosis.

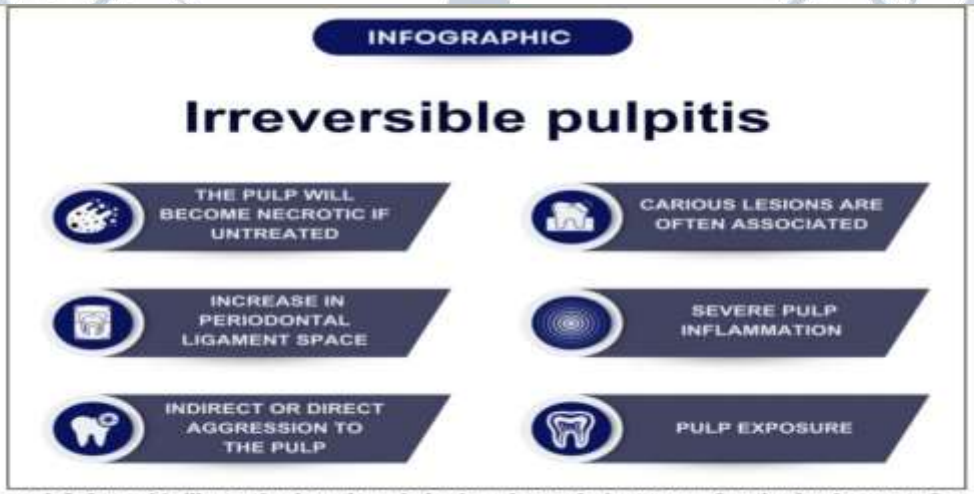

Figure 4: Infographic illustrating how the pulp begins a hyperplasic process after the development of a pulp pathological reaction (described by Alrifai *et al.*, 2022)
Source: Authors, 2024.

Chronic venous congestion reduces oxygen tension, impairing mitochondrial function and ATP production. Over time, cellular apoptosis gives way to necrosis, particularly in regions closest to the carious lesion.

Microbial persistence exacerbates this process by introducing endotoxins that disrupt immune regulation. The absence of acute inflammatory pressure allows necrotic tissue to remain within the pulp chamber, facilitating gangrenous decomposition rather than abscess formation. This distinguishes chronic gangrenous pulpitis from acute suppurative conditions.

Statistical correlations between socioeconomic factors and disease prevalence highlight the importance of preventive strategies. From a theoretical standpoint, reducing etiological exposure could interrupt the pathogenic cascade before irreversible changes occur. The reviewed literature collectively supports a model in which chronic gangrenous pulpitis represents the final stage of a continuum rather than an isolated pathology.

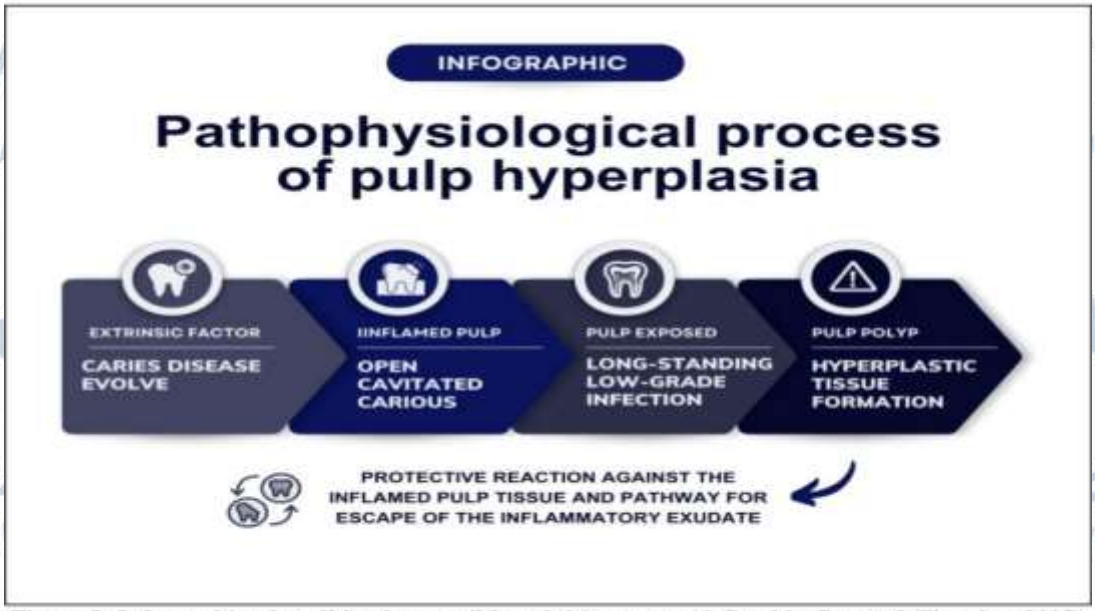

Figure 3: Infographics describing irreversible pulpitis aspects (defined by Lopes & Siqueira, 2015)
Source: authors, 2024

Conclusion: Chronic gangrenous pulpitis is a complex pathological entity rooted in anatomical confinement, vascular compromise, and sustained microbial activity. Theoretical analysis demonstrates that prolonged low-grade inflammation leads to irreversible pulpal degeneration without the overt manifestations typical of acute disease. Anatomical vulnerability, hydrodynamic imbalance, and microbial toxins collectively drive the progression toward gangrene. The absence of clinical symptomatology in many cases underscores the silent nature of this condition and explains its high prevalence in epidemiological studies. Understanding the etiological and pathogenetic mechanisms at a theoretical level provides valuable insight into pulpal disease dynamics and highlights the critical role of early preventive interventions. Future research should continue to refine molecular and hydrodynamic models of pulpal degeneration to enhance predictive frameworks in endodontic pathology. A purely scientific perspective, free

from clinical bias, remains essential for advancing foundational knowledge in dental pulp biology.

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