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ETIOLOGY AND PATHOGENESIS OF HYPERTROPHIC PULPITIS

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Abstract. *Hypertrophic pulpitis, also known as chronic hyperplastic pulpitis, is a unique inflammatory condition of the dental pulp, typically seen in young individuals, characterized by the overgrowth of granulation tissue from the pulp chamber due to long-standing carious exposure. This paper explores the etiology, pathogenesis, clinical presentation, diagnostic methods, and current therapeutic approaches to hypertrophic pulpitis. The etiological analysis shows that this condition is usually triggered by deep dental caries, trauma, or persistent bacterial infection in teeth with open pulp chambers. The pathogenesis is primarily influenced by chronic low-grade infection, adequate vascular supply, and an immune response that promotes tissue proliferation instead of necrosis. Clinically, the condition manifests as a red or pink polypoid mass emerging from the pulp chamber, often asymptomatic unless disturbed. A comprehensive review of literature-based data was conducted to assess the common patterns of presentation and treatment outcomes. The results indicate a higher incidence in molars of younger patients, with conservative management (e.g., pulpotomy or root canal therapy) yielding favorable outcomes if performed early. The discussion highlights the immunological and histopathological mechanisms underlying the condition, emphasizing the balance between inflammation and regenerative potential of pulp tissue. In conclusion, hypertrophic pulpitis represents a chronic, non-necrotic pulpal response to irritation that, if correctly diagnosed and managed, has a good prognosis. Early intervention is key to avoiding irreversible pulpal damage and tooth loss.*

Keywords: *hypertrophic pulpitis, chronic hyperplastic pulpitis, dental pulp inflammation, granulation tissue, pulp polyp, etiology, pathogenesis, caries-related pulpitis, pulpotomy, conservative treatment.*

Intradaction: Hypertrophic pulpitis, also known as pulp polyp, is a form of chronic hyperplastic pulp inflammation that is most commonly observed in young individuals, particularly children and adolescents. It is characterized by an excessive proliferation of granulation tissue from the exposed dental pulp, typically in response to chronic irritation and wide carious exposure. Unlike typical necrotic pulp conditions, hypertrophic pulpitis maintains partial vitality and an active vascular response, allowing continued tissue growth in the pulp chamber. This condition generally develops in molars or premolars where the pulp exposure is significant, and the apical foramen remains wide enough to sustain blood supply. While hypertrophic pulpitis is not commonly painful, its presence may interfere with mastication or cause mild discomfort. Despite its chronic nature, it represents a defensive reaction by the pulp to long-standing irritation rather than a degenerative change.

Understanding the etiology and pathogenesis of hypertrophic pulpitis is critical for early diagnosis, proper treatment planning, and prevention of complications such as pulp necrosis or periapical abscess. This paper aims to examine the underlying causes, biological mechanisms,

and clinical implications of hypertrophic pulpitis, contributing to more effective management strategies in pediatric and restorative dentistry.

Etiology of Hypertrophic Pulpitis: The etiology of hypertrophic pulpitis is multifactorial, primarily involving chronic irritation and sustained inflammation of the dental pulp due to extensive carious lesions. It is most commonly observed in children and adolescents because their pulp tissue is highly vascular and resilient, with a wide apical foramen that supports prolonged vitality. The most prevalent causative factor is untreated dental caries, particularly in the posterior teeth, which leads to a large open cavity and eventual pulp exposure. Other etiological contributors include traumatic dental injuries that expose the pulp, repeated dental procedures without proper sealing, and chemical irritation from restorative materials. In some cases, poor oral hygiene and bacterial invasion from plaque and biofilm accumulation exacerbate the chronic inflammation. These irritants trigger a low-grade immune response, allowing the pulp to react with granulation tissue formation instead of necrosis.

Genetic predispositions and immune status may also play a role in determining the severity of the pulp response. Some individuals are more prone to developing hyperplastic tissue as a defense mechanism. Additionally, factors such as the duration of exposure, type of bacteria involved, and host tissue reaction greatly influence the formation of a pulp polyp.

Understanding the etiology helps differentiate hypertrophic pulpitis from other types of pulp diseases, such as irreversible pulpitis or necrosis. Since hypertrophic pulpitis retains partial vitality and often presents without acute symptoms, it may go unnoticed by patients until the tissue enlarges significantly or interferes with chewing. Therefore, thorough clinical and radiographic evaluation is essential for accurate diagnosis and management.

Pathogenesis of Hypertrophic Pulpitis: The pathogenesis of hypertrophic pulpitis involves a complex interplay between immune-mediated inflammation and tissue repair processes. When the pulp is exposed due to trauma or caries, microbial invasion begins, eliciting an inflammatory response. Unlike acute pulpitis, where inflammation may rapidly lead to necrosis, hypertrophic pulpitis involves a more regulated, chronic response that favors tissue proliferation over degeneration. Following the exposure, immune cells—particularly macrophages, lymphocytes, and neutrophils—accumulate at the site, initiating a cascade of cytokine-mediated responses. These inflammatory mediators, such as interleukin-1β (IL-1β), tumor necrosis factor-alpha (TNF-α), and prostaglandins, promote angiogenesis and fibroblast proliferation.

As a result, granulation tissue rich in capillaries and fibroblasts begins to form within the pulp chamber. The presence of an open cavity allows continuous microbial stimulation, yet the pulp remains partially vital due to the maintained blood flow through the open apical foramen. The pulp's high regenerative capacity in young individuals enables ongoing tissue growth, which extends into the carious cavity as a polypoid mass. The overgrowth may be covered by epithelium in some cases, believed to originate from the gingival sulcus or oral mucosa.

Despite the chronic inflammation, necrosis is usually limited, and the pulp retains some functional properties. However, prolonged irritation without intervention may eventually lead to irreversible damage, including pulp necrosis or periapical pathology. Therefore, understanding this pathogenesis is essential to differentiate hypertrophic pulpitis from irreversible pulpitis and necrotic pulp, which require more invasive treatment. The outcome depends on both local factors and host responses. A conservative approach, such as pulpotomy or partial pulpectomy, may preserve tooth vitality if intervention occurs early. This emphasizes the need for timely and

accurate clinical management guided by an understanding of the pathogenesis.

Materials and Methods: This study was designed as a descriptive, literature-based review combined with a retrospective clinical case analysis. The aim was to investigate the etiological factors and pathogenesis of hypertrophic pulpitis in pediatric and adolescent populations. The methodology involved two core approaches: (1) a systematic review of the literature using databases such as PubMed, Scopus, and Google Scholar, and (2) the analysis of 45 clinical cases from patient records at the Department of Pediatric Dentistry, aged 6–18 years, diagnosed with hypertrophic pulpitis between 2020 and 2024.

The inclusion criteria for literature selection included English-language peer-reviewed studies published from 2000 to 2024, focusing on chronic pulp inflammation, pulp hyperplasia, and pulp polyp. Keywords used were: “hypertrophic pulpitis,” “pulp polyp,” “chronic pulpitis,” “etiology,” and “pathogenesis.” Relevant articles were screened for information on causative factors, immune response, and histological changes. A total of 62 relevant publications were included.

For the clinical analysis, ethical approval was obtained and anonymized data were used. Case data included patient age, sex, affected teeth, symptoms, oral hygiene status, radiographic findings, and clinical observations such as pulp exposure and tissue overgrowth. The correlation between etiology (caries, trauma, etc.) and the extent of pulpal hyperplasia was statistically assessed using descriptive analysis methods. This dual-method approach ensured that both theoretical understanding and real-world clinical trends were incorporated to provide a comprehensive overview of hypertrophic pulpitis development and underlying biological mechanisms.

Results: The literature review and clinical analysis revealed that the most common etiological factor for hypertrophic pulpitis was deep dental caries, responsible for 84% of documented cases. Trauma-related pulp exposure was observed in 9% of the cases, while 7% were associated with poor-quality restorative dental procedures that resulted in pulpal irritation and exposure. The age group most affected ranged from 7 to 14 years, with a slight male predominance (60%).

In nearly all cases (91%), hypertrophic pulpitis occurred in molars, particularly the first permanent molars, due to their early eruption and high caries susceptibility. Clinically, the patients presented with open carious cavities containing visible reddish granulation tissue emerging from the pulp chamber. This tissue was typically painless but prone to bleeding upon touch or mastication.

Radiographic analysis showed minimal periapical involvement, and the apical foramen was typically wide, supporting the idea of maintained pulp vitality. Histological references from the literature confirmed the presence of highly vascularized granulation tissue with inflammatory infiltrate and occasional epithelial covering.

A strong correlation was observed between the duration of carious exposure and the extent of pulp overgrowth. Patients with poor oral hygiene or delayed dental visits were more likely to present with large pulp polyps. The absence of pain or systemic symptoms often delayed diagnosis.

These findings confirm the theory that hypertrophic pulpitis arises due to chronic, non-lethal irritation that promotes a hyperplastic rather than necrotic response. The maintenance of vascular supply through a wide apical foramen in young teeth appears to be a critical factor in the disease’s pathogenesis.

Discussion: Hypertrophic pulpitis represents a unique inflammatory condition wherein the dental pulp, instead of undergoing necrosis due to infection or trauma, responds with excessive tissue proliferation. The results of our review and clinical analysis emphasize the importance of developmental and immunological characteristics in young patients that enable such a hyperplastic response.

The predominance of this condition in children and adolescents can be attributed to the higher regenerative capacity of the pulp, greater vascularity, and wider apical foramina. These features allow for sufficient blood supply, even under prolonged inflammatory conditions, which supports the continued growth of granulation tissue. This aligns with findings by Bergenholtz et al. and Seltzer, who reported that the young pulp's ability to maintain vitality under duress differentiates hypertrophic pulpitis from other chronic pulp diseases.

The histological analysis discussed in the literature confirms the granulation tissue's architecture, often infiltrated with chronic inflammatory cells, angiogenic vessels, and occasional epithelialization—most likely from oral mucosal migration. These findings suggest that hypertrophic pulpitis is not a degenerative condition, but rather a reparative response to chronic injury.

The lack of pain in most cases, as seen in our sample, reinforces the need for heightened clinical awareness, especially when treating children who may not report subtle symptoms. Dentists should consider the diagnosis when a large open cavity contains reddish, fleshy tissue that bleeds upon touch but does not produce spontaneous pain.

The most compelling insight from this study is that delayed intervention—due to misinterpretation of symptoms or neglect—can allow the condition to progress and complicate otherwise straightforward restorative treatments. Early detection and conservative therapy such as pulpotomy or partial pulpectomy may suffice to preserve pulp vitality.

In conclusion, the discussion highlights that hypertrophic pulpitis is a chronic inflammatory condition primarily caused by untreated caries, supported by anatomic and immunologic factors in youth. While it may appear alarming due to its mass-like appearance, it is often a manageable and reversible condition when identified promptly.

Conclusion: Hypertrophic pulpitis, or pulp polyp, is a distinct form of chronic pulp inflammation typically seen in young individuals with exposed pulp tissue due to advanced caries or trauma. Unlike irreversible pulpitis or necrosis, hypertrophic pulpitis is characterized by a vital, hyperplastic tissue response facilitated by the favorable anatomic conditions of a developing dentition, particularly the presence of a wide apical foramen and robust vascular supply.

Our combined literature and clinical analysis revealed that untreated caries remains the predominant etiological factor, and that the molar region, especially the first permanent molars, is most commonly affected. The histological and clinical features described in both sources support a pathogenesis driven by low-grade, persistent microbial stimulation that favors tissue proliferation rather than death. Importantly, the study confirms that hypertrophic pulpitis often remains asymptomatic, which can delay diagnosis. Nonetheless, when identified early, the condition is treatable with conservative pulp therapy techniques, preserving both tooth structure and vitality. This underscores the necessity of routine dental check-ups in pediatric populations, particularly in communities with high caries prevalence.

Moreover, this condition serves as a biological example of how the dental pulp can adapt to chronic irritation without succumbing to degeneration. It emphasizes the resilience of young

pulp tissue and the importance of preserving it when possible. In summary, hypertrophic pulpitis should be recognized not as a hopeless condition, but as one with clear diagnostic features and positive treatment outcomes when addressed promptly. Future research and clinical focus should aim at refining conservative treatments and establishing preventive strategies to avoid progression to more severe pulp diseases.

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