

ETIOLOGICAL FACTORS OF CATARRHAL GINGIVITIS

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Abstract: *Catarrhal gingivitis is one of the most frequently encountered inflammatory conditions of the periodontal tissues in dental practice. It predominantly affects the marginal gingiva and interdental papillae and is characterized by hyperemia, swelling, bleeding upon mechanical stimulation, and subjective discomfort. Although the condition is generally considered reversible at the early stage, its progression without timely intervention can lead to chronic inflammation and subsequent periodontal breakdown. The aim of this paper is to systematize the etiological factors associated with catarrhal gingivitis and to emphasize the importance of comprehensive prevention and management.*

Keywords: *catarrhal gingivitis, dental plaque, local irritants, hormonal imbalance, vitamin deficiency, periodontal inflammation, oral health.*

Introduction: Gingivitis represents an initial stage in the pathogenesis of periodontal diseases and serves as a critical point for early intervention. Among its various clinical forms, catarrhal gingivitis is the most common, particularly in children, adolescents, and adults with inadequate oral hygiene. It is essential to recognize that catarrhal gingivitis does not occur in isolation but arises from a complex interplay of local and systemic factors. Identifying and addressing the underlying causes is the cornerstone of effective treatment and long-term prevention.

Local Etiological Factors

The primary cause of catarrhal gingivitis is microbial plaque accumulation on the tooth surfaces, especially in the cervical and interdental regions. Dental biofilm harbors a diverse population of microorganisms, including *Streptococcus mutans*, *Actinomyces* spp., and anaerobic bacteria, which initiate and perpetuate the inflammatory process. Toxins and metabolic by-products released by these microorganisms provoke an immune-inflammatory response, resulting in vascular dilation, infiltration of leukocytes, and degradation of connective tissue components.

Additionally, supragingival and subgingival calculus, which form due to mineralization of plaque, act as persistent irritants and exacerbate the inflammatory process. Faulty dental restorations, poorly contoured fillings, and overhanging margins further contribute to plaque retention and mechanical trauma of the

gingival tissues. Malocclusion and crowding of teeth may hinder adequate oral hygiene and create niches for plaque accumulation.

In the context of pediatric and adolescent patients, improper brushing techniques and neglect of oral hygiene are among the most prominent predisposing factors. The use of orthodontic appliances may also increase the risk of catarrhal gingivitis due to the difficulty in maintaining optimal cleanliness.

Systemic Etiological Factors

In addition to local irritants, several systemic conditions can predispose individuals to gingival inflammation. Among them, hormonal changes during puberty, menstruation, pregnancy, and menopause play a significant role by altering the vascular permeability and immune response of the gingival tissues. These hormonal fluctuations can enhance the host's sensitivity to plaque and exacerbate the clinical manifestations of gingivitis.

Nutritional deficiencies, particularly of vitamin C (ascorbic acid), which is essential for collagen synthesis and capillary integrity, are known to impair tissue resistance and healing. Likewise, deficiencies in B-group vitamins, especially B2, B6, and B12, are associated with mucosal atrophy and desquamative gingivitis.

Chronic gastrointestinal diseases, such as gastritis, duodenitis, and intestinal dysbiosis, may also affect oral health by impairing nutrient absorption and contributing to systemic inflammation. Respiratory infections and chronic tonsillitis have been reported to influence gingival conditions through hematogenous spread of pathogens or persistent immune activation.

Furthermore, general immune suppression—whether due to underlying systemic disease, medication (e.g., corticosteroids or cytostatics), or stress—reduces the host's ability to control local infection, thereby contributing to the persistence and exacerbation of gingival inflammation.

Clinical Manifestations and Complications

Clinically, catarrhal gingivitis presents with redness, swelling, and bleeding of the gingiva upon probing or brushing. Halitosis, discomfort while eating, and increased sensitivity may also be reported. If untreated, the condition may evolve into chronic gingivitis with fibrotic changes or progress to periodontitis, characterized by attachment loss and bone resorption.

In children, prolonged gingival inflammation may interfere with the normal eruption of permanent teeth and increase the risk of malocclusion. In adults, the chronic form of gingivitis poses a significant risk for progression to destructive periodontal disease, particularly in the presence of systemic comorbidities.

Conclusion: In summary, catarrhal gingivitis is a multifactorial disease with both local and systemic etiological components. While dental plaque remains the leading cause, the role of hormonal changes, nutritional deficiencies, systemic

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illnesses, and immune status cannot be overlooked. A comprehensive approach to diagnosis and treatment must include the identification and elimination of all contributing factors. Patient education, motivation, and regular professional care are critical to the effective management and prevention of catarrhal gingivitis. Continued research into host–microbe interactions and individualized prevention strategies will further enhance outcomes in clinical practice.

