

The Role of Digestive System Disorders in the Development of Inflammatory Periodontal Diseases

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Abstract The relevance of the study is determined by the widespread occurrence of inflammatory periodontal diseases, necessitating the identification of systemic factors contributing to their progression, among which special attention is given to gastrointestinal tract (GIT) disorders. The aim of the work is to investigate the impact of chronic gastroduodenal diseases, endocrine and immune dysfunctions on periodontal tissue health. The study involved patients with periodontal inflammation and concomitant GIT pathologies. A comprehensive dental examination was carried out, including laboratory tests, microbiological assessment for *Helicobacter pylori*, evaluation of microcirculation indicators, and analysis of immune system function. As a result, it was established that patients with gastroduodenal diseases, gastroesophageal reflux disease (GERD), carbohydrate metabolism disorders (type 2 diabetes), and *Helicobacter pylori* infection exhibit more pronounced periodontal inflammation characterized by edema, bleeding, impaired capillary blood flow, and a tendency towards generalized lesions. The findings emphasize the need to integrate GIT condition assessment into the diagnostic and therapeutic approach for periodontal patients, which will enhance the effectiveness of dental treatment and improve disease prognosis.

Keywords Periodontitis, Gingivitis, Gastrointestinal tract disorders, Gastroduodenal pathology, Gastroesophageal reflux disease, *Helicobacter pylori*, Microcirculation, Endocrine system, Immune regulation

1. Introduction

Modern research in the fields of medicine and dentistry emphasizes the intricate relationship between the functioning of the gastrointestinal (GI) tract and the health of periodontal tissues. Extensive studies reveal a high prevalence of periodontitis among patients with various gastrointestinal disorders. In the periodontal pockets of these patients, abnormally high levels of anaerobic microorganisms are often found, significantly exceeding normal levels.

Special attention in research is given to the impact of *Helicobacter pylori* infection on periodontal health. It has been established that patients infected with this bacterium have a much higher incidence of periodontal diseases, necessitating a comprehensive medical approach and collaboration between dentists and gastroenterologists.

Changes in the oral cavity, such as discomfort, itching, pain, and altered taste perception, can often be early indicators of GI tract problems. These symptoms may manifest earlier than classic gastric symptoms, making regular dental check-ups crucial for the early detection of GI diseases.

Disorders in the GI tract contribute to a general decrease in the body's resilience, affecting the immune and endocrine systems, microcirculation, mineral metabolism, and

connective tissue metabolism. This, in turn, increases the susceptibility of periodontal tissues to pathogenic microorganisms, exacerbating inflammatory processes including gingivitis and periodontitis.

2. Literature Review

In recent years, there have been significant advancements in the understanding of the mechanisms of development and the nature of periodontal diseases, as reflected in the scientific literature [1, pp. 72-75]. Contemporary research conducted by numerous scientists [2, pp. 321-444; 3, pp. 38-40; 4, pp. 96-99] demonstrates a substantial correlation between inflammatory processes in the periodontium and the functional state of internal organs. These findings underscore the importance of a comprehensive approach to treating periodontal diseases, considering their potential impact on overall human health.

Systemic mechanisms unify the pathogenesis of various diseases, defining their associative and interconnected nature with common risk factors. The significance of this aspect is particularly emphasized in cases of concomitant diseases of the internal organs and the oral cavity, as highlighted in scientific studies [5, pp. 72-80].

In digestive system diseases, conditions arise that promote inflammatory processes in the periodontium. This is due to the dysfunction of various regulatory systems in the

body [6, pp. 48-52]. Key aspects of such disorders include changes in the immune and endocrine systems, endotoxemia, microcirculation issues, as well as disruptions in neurohumoral regulation and psychosomatic influences, which are particularly characteristic of gastrointestinal tract pathologies [7, pp. 48-52]. Under these conditions, disturbances in connective tissue metabolism, mineral imbalance, and vitamin deficiencies may also occur, leading to a decrease in overall body resistance and increasing the risk of developing diseases such as gingivitis and periodontitis, especially under the influence of external factors, including bacterial colonization of dental plaque [8, pp. 55-57].

In the context of combining periodontal diseases with gastrointestinal tract (GI) pathologies in patients, special attention is given to gastroduodenal diseases [9, pp. 1548-1553]. These pathologies constitute a significant portion of gastroenterological diseases, ranging from 4% to 30% of the total [10, pp. 66-71]. This category includes conditions such as gastritis, gastric and duodenal ulcers, emphasizing the necessity for an integrative approach to diagnosing and treating patients with periodontal diseases [11, pp. 79-89].

The activation of free radical oxidation processes and the imbalance in the antioxidant defense system are crucial factors influencing the development of inflammatory processes in the periodontium. This phenomenon is particularly pronounced in elderly patients, who naturally exhibit a decrease in antioxidant levels, often requiring additional correction using natural antioxidants [12, pp. 20-22].

Numerous studies confirm the connection between the condition of periodontal tissues and the functionality of various systems and organs of the body. Patients with gastrointestinal tract diseases exhibit a high frequency of periodontitis of varying severity. Their periodontal pockets usually contain an increased number of anaerobic microorganisms, reaching 70-80%, which significantly exceeds the normal values of 20-30% [13, pp. 130-133]. In cases of GI tract dysfunction, periodontal involvement is registered in 86.7% of cases [14, pp. 79-81], while in patients with endocrine system disorders, including type II diabetes mellitus, the prevalence of periodontitis is 95%. There is also a noted connection between salivary gland hypofunction and other factors with the increased incidence of periodontal diseases [15, pp. 116-118; 16, pp. 12-43].

The study of the relationship between GI tract pathologies and periodontal health has revealed that patients infected with *Helicobacter pylori* have a higher incidence of periodontal diseases. This suggests the necessity for comprehensive medical monitoring of such patients by both dentists and gastroenterologists [17, pp. 4-7].

In GI tract pathologies, changes in the oral cavity often occur before the onset of painful gastric symptoms. Patients typically experience oral discomfort, often described as itching and pain that intensifies during eating. Taste perception disorders are also characteristic, manifesting as a sour taste. The presence of metallic dental prostheses can exacerbate these symptoms [18, pp. 888-943].

One of the noticeable characteristics of such gastrointestinal diseases is the formation of plaque on the tongue, which can vary in color from white to light yellow and brown. These changes can be caused by various factors, including diet, medication, smoking and other external influences. Despite the ease of removing such plaque, the tendency for it to quickly return remains. Plaque also forms on the teeth, contributing to the development or maintenance of inflammatory processes in the gums, such as gingivitis [19, pp. 14-17].

Dysfunctions in the gastrointestinal (GI) tract affect the overall resilience of the body, causing imbalances in several critical regulatory systems. This includes alterations in the immune and endocrine systems, microcirculation issues, mineral metabolism disturbances, connective tissue metabolism disruptions, and vitamin deficiencies. The combined effect of these dysfunctions increases the susceptibility of periodontal tissues to pathogenic oral microflora, which can exacerbate the development of inflammatory processes in the gums, including gingivitis and periodontitis [20, pp. 344-433].

There is a broad correlation between periodontal diseases and various somatic disorders [21, pp. 6-10; 22, pp. 385-395; 23, pp. 385-395]. Specifically, associations have been found between periodontitis and conditions such as diabetes mellitus, urolithiasis, subacute septic endocarditis, arterial hypertension, peptic ulcer disease of the stomach and duodenum, and liver pathologies. These connections highlight the importance of integrating periodontal disease treatment with a general medical approach to patient health [24, pp. 54-54].

Chronic gastritis, peptic ulcer diseases, chronic pancreatitis, hepatitis, and liver cirrhosis are often associated with oral cavity diseases such as gingivitis, periodontitis, and aphthous lesions of the mucosa. Studies emphasize the link between periodontal status and inflammatory bowel diseases, including Crohn's disease, where morphological changes in the periodontium are similar to those occurring in colorectal pathologies, including the presence of lymphoid granulomas [25, pp. 94-97]. Dysfunction in the digestive system also impacts the activity of salivary glands and the processes of demineralization and remineralization of dental enamel, contributing to the development of caries [26, pp. 220-227].

Peptic ulcer disease of the stomach and duodenum stands out in gastroenterology due to its high prevalence and tendency for complications [27, pp. 63-67]. Although the role of *Helicobacter pylori* (HP) in the development of this disease is confirmed, it is considered a systemic condition affecting not only the stomach but other parts of the digestive system as well [28, pp. 63-67].

Changes in periodontal tissues are common among patients with peptic ulcer disease of the stomach and duodenum. In GI tract pathologies, inflammatory and destructive processes in the periodontium usually manifest in a generalized and active form [29, pp. 191-194]. Studies indicate that digestive tract problems often precede the onset of periodontal diseases, and the extent of their impact on the periodontium directly correlates with the severity and duration of the digestive system disease [30, pp. 34-40].

It is generally accepted that the pathophysiological and pathomorphological changes occurring in the gastric mucosa and the periodontium have much in common. This understanding evolves in the context of the changing perspectives on the etiology and pathogenesis of GI tract ulcerative diseases [31, pp. 44-48; 32, pp. 282-284].

Peptic ulcers of the stomach and duodenum create specific conditions for the activation of inflammatory reactions in the periodontium, caused by disruptions in key body systems. These disruptions include imbalances in the immune and endocrine systems, problems with microcirculation and neurohumoral regulation, psychosomatic interactions, connective tissue metabolism disorders, mineral metabolism disturbances, and vitamin deficiencies [33, pp. 41-48]. All these factors collectively reduce the body's resistance and enhance the impact of external irritants, including microbial colonization of dental plaque, promoting the development of diseases such as gingivitis and periodontitis. Additionally, patients with peptic ulcers often experience oral hygiene problems, exacerbating the situation [34, pp. 282-284].

Research also points to the significant role of increased levels of calcium-regulating hormones, including parathyroid hormone and calcitonin, in the blood of patients with peptic ulcer disease. It is hypothesized that there is an initial increase in the production of gastrointestinal hormones (e.g., gastrin, cholecystokinin), which stimulate the thyroid gland to synthesize calcitonin. This process disrupts the balance between hypo- and hypercalcemic hormones and promotes bone tissue resorption in the periodontium, exacerbating inflammatory processes [35, pp. 130-133; 36, pp. 22-26; 37, pp. 682-692].

3. Discussion

Modern research in the pathogenesis of inflammatory periodontal diseases and peptic ulcer disease of the gastrointestinal tract pays particular attention to the role of disruptions in the body's defense system, especially the immune system. These dysfunctions significantly impact the body's ability to resist microbial attacks and repair damaged tissues, which is a key aspect in understanding the mechanisms of these diseases. There is a hypothesis that the presence of common antigens in the gastrointestinal mucosa can trigger autoimmune reactions that damage the gums. In cases of co-occurrence of peptic ulcer disease and periodontitis, disruptions in cellular immunity have been identified, including a reduction in active forms of neutrophilic granulocytes and a decrease in the level and functional activity of T-lymphocytes.

Clinical signs of autonomic nervous system dysfunction are common to both digestive system diseases and periodontal diseases, especially under conditions of acute and chronic stress. Stress plays a significant role in the pathogenesis of these diseases, affecting inflammatory processes through various mechanisms. In particular, during acute stress, there is an activation of lipid peroxidation processes in periodontal

tissues, an imbalance between proteases and their inhibitors, and impaired microcirculation.

A pivotal moment in medical science was the discovery by Australian scientists A. Marshall and B. Warren in 1983 of the role of *Helicobacter pylori* (HP) in the development of chronic gastritis, peptic ulcer disease, and stomach cancer. This discovery led to new methods for the treatment and prevention of these diseases. The study of HP has long focused on gastroenterology and microbiology, but as early as 1982, B. Marshall suggested that HP might localize outside the gastroduodenal zone. Modern research actively investigates the impact of HP on the development of various diseases, including ischemic heart disease, bronchial asthma, and periodontal diseases.

Helicobacter pylori (HP) is one of the most frequently encountered microorganisms in clinical practice. Epidemiological data indicate that more than 50% of the world's adult population is infected with this bacterium. The risk of HP infection increases with age and is influenced by socioeconomic status. The variability in the detection frequency of HP in the oral cavity, according to various studies, ranges from 0 to 100%, and its presence often correlates with poor oral hygiene. This bacterium can be found in dental plaque, saliva, gingival pockets, and on damaged areas of the oral mucosa. It is notable that HP is more frequently found on molars (in 82% of cases) compared to anterior teeth (59%), which may be related to its microaerophilic properties.

HP infection transmission typically occurs via fecal-oral or oral-oral routes. Iatrogenic transmission from one patient to another during medical procedures, such as gastroscopy, is also known, especially when proper instrument disinfection measures are not followed or the infectious nature of gastric diseases is underestimated.

In scientific circles, discussions continue about the potential reservoirs of HP. The oral cavity is of particular interest as a potential source of gastric mucosal reinfection and a route of transmission for the bacterium. There is a hypothesis that the oral cavity might be the primary site of HP colonization before its migration to the stomach. Some researchers suggest that HP can reside in periodontal pockets, creating favorable microaerophilic conditions for its existence. Meanwhile, other studies indicate that HP is present in the oral cavity only temporarily, for instance, due to the consumption of contaminated food or as a result of gastroesophageal reflux in patients with HP-associated gastroduodenal diseases.

The role of *Helicobacter pylori* (HP) in oral diseases continues to be an active area of research in scientific circles. The work of Chobanov R.E. and Mamedov R.M. revealed elevated HP levels in soft dental plaque and the contents of periodontal pockets. Special attention is given to the connection between HP and oral pathologies such as chronic generalized catarrhal gingivitis, periodontitis, glossitis, and cheilitis, which often exhibit severe courses in patients with HP-associated GI diseases.

The presence of HP in the oral cavity increases the risk

of gastric mucosal reinfection and peptic ulcer disease recurrences. Studies have shown that in patients with HP-associated chronic gastritis, successful eradication of the infection from the stomach is achieved in 92% of cases when HP is not detected in the oral cavity, with no recurrences observed over two years. However, when HP is present in the oral cavity in the same patient group, treatment effectiveness drops to 52%, and disease recurrences occur in 35% of patients within two years.

These data confirm the need to consider the presence of HP in the oral cavity when treating HP-associated diseases. Despite the effectiveness of modern methods for eliminating HP from the stomach, the problem of reinfection and recurrence of peptic ulcer disease remains relevant. According to studies by A.V. Tsymbalistov and N.S. Robakidze, even after successful treatment of gastric pathologies, HP persists in the gingival pockets and saliva of most patients. Therefore, a comprehensive approach to the treatment of HP-associated GI diseases should include the use of local antibacterial agents targeting HP and professional oral cleaning to effectively and completely eliminate the bacterium.

Gastroesophageal reflux disease (GERD) significantly impacts the relationship between the oral cavity and the esophagus, affecting periodontal health. GERD is characterized by the frequent movement of stomach contents into the esophagus, causing inflammatory changes in the lower esophagus and surrounding tissues. The widespread prevalence of heartburn, the primary symptom of GERD, affects 20% to 40% of the population in Europe and America, drawing significant attention to this condition.

GERD can progress, leading to serious complications such as peptic ulcers, esophageal strictures, bleeding, morphological changes in the epithelium, and damage to adjacent organs and systems. Modern studies emphasize that GERD initiates complex pathogenetic processes, worsening the course of concomitant diseases, extending beyond simple reflux esophagitis and heartburn.

Complications of GERD can affect various body systems, including bronchial asthma, recurrent pneumonias, pulmonary fibrosis, chronic cough, sinusitis, voice disorders, and even increase the risk of laryngeal cancer. Regular entry of stomach contents into the esophagus and oral cavity causes significant changes in the condition of the oral mucosa, the structure of tooth enamel, and periodontal health. Patients with GERD also experience changes in the composition and properties of saliva, affecting the function of the antireflux barrier and the overall defense mechanisms of the oral cavity and esophagus.

Damage to the soft tissues of the oral cavity, including the vermilion border of the lips, mucous membranes, tongue, and periodontium, as well as the hard tissues of the teeth, are characteristic signs of GERD. These changes, accompanied by variations in the composition of oral fluids, reflect the impact of the disease on the condition of the oral cavity. Dental enamel erosion is observed, correlating with the severity of GERD symptoms and the degree of esophageal damage.

4. Conclusions

It is important to emphasize that GERD affects not only the upper parts of the gastrointestinal tract but also the condition of the oral cavity, highlighting the need for a comprehensive treatment approach. This approach should include local treatment and the use of proton pump inhibitors. Social, economic, and medical aspects of chronic liver diseases are also important, given their widespread prevalence and prognostic difficulties.

Thus, the presence of gastrointestinal pathologies increases the risk of developing and worsening chronic inflammatory periodontal diseases. This underscores the importance of considering common pathogenetic mechanisms when developing treatment strategies for periodontitis in the context of gastrointestinal pathologies.

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