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GASTROESOPHAGEAL REFLUX DISEASE AND DENTAL DISORDERS

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Abstract

Generalized periodontitis is the most common inflammatory periodontal disease. Currently, it can be concluded that the local mechanisms of periodontitis development have been largely deciphered. At the same time, periodontitis is considered not only as a localized periodontal inflammation caused by the microflora of dental plaque, but also as a whole-body response to bacterial infection. On the other hand, it is clear that systemic processes play a significant role in the pathogenesis of generalized periodontitis, leading to profound changes in the body's internal environment and, consequently, to structural damage to periodontal tissues.

Currently, a number of general somatic factors have been identified that accompany and influence the development of inflammatory periodontal diseases: atherosclerosis, gastrointestinal diseases, endocrine pathology, gastrointestinal tract diseases, and biliary tract pathology. These perspectives are united by the idea that periodontal tissues respond stereotypically with structural shifts under the influence of a wide variety of changes in the body. The anatomical and

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physiological proximity of periodontal and gastrointestinal tract tissues, as well as their shared innervation and humoral regulation, create the preconditions for periodontal involvement in the pathological process associated with gastrointestinal diseases. Research into the pathogenetic relationships and interactions between diseases of the internal organs and the periodontium is a pressing issue for both dentistry and gastroenterology. The role of the diffuse neuroendocrine system in the pathogenesis of inflammatory periodontal and gastrointestinal diseases remains poorly understood. Cells of the diffuse neuroendocrine system are located throughout the gastrointestinal tract; they regulate digestion and participate in trophic processes, regeneration, and proliferation. In recent years, melatonin, serotonin, and histamine—potent regulators of biological and pathological processes—have attracted particular attention from researchers. Mast cells in the oral cavity, which synthesize biologically active substances such as histamine, heparin, leukotrienes, melatonin, and serotonin—are closely linked to the functions of the diffuse neuroendocrine system. The involvement of mast cells in the regulation of inflammation and tissue regeneration has led to increased research interest in their role in the development and progression of inflammatory periodontal diseases. The functional activity of mast cells cannot but change under conditions of pathology of the esophagus, stomach and biliary tract.

The general pathomorphological picture that determines the course and prognosis of a chronic disease, including periodontitis, is the severity of disturbances in the cellular renewal of epithelial cells of the digestive tract, which can be used both for the purposes of preclinical diagnosis of the disease and for determining its course and prognosis.

Gastroesophageal reflux disease (GERD) is a chronic condition characterized by the retrograde reflux of gastric contents into the esophagus and oral cavity. Exposure to hydrochloric acid and pepsin damages not only the esophageal mucosa but also the oral tissues, leading to the development of various dental

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disorders. The most common dental manifestations of GERD include enamel erosion, tooth hypersensitivity, caries, xerostomia, and inflammatory diseases of the mucosa and periodontium. These changes can serve as early clinical signs of the disease and are often detected by a dentist before the patient consults a gastroenterologist. Pathogenetic mechanisms include decreased oral pH, enamel demineralization, impaired salivary protective properties, and altered microbiomes. The duration and frequency of reflux episodes play a significant role, directly affecting the severity of dental disorders. Studying the relationship between GERD and dental changes is of great practical importance, as it enables timely diagnosis, comprehensive treatment, and the development of effective preventive measures. A multidisciplinary approach involving a gastroenterologist and a dentist is key to successful patient management.

Keywords: Gastroesophageal reflux disease, GERD, dental disorders, enamel erosion, caries, dental hypersensitivity, xerostomia, acid reflux, oral cavity, periodontium.

The aim of the study was to examine the clinical aspects of the relationship between gastroesophageal reflux disease and dental disorders, as well as to determine the specifics of their diagnosis and prevention.

Research objectives: To study the etiology and pathogenesis of gastroesophageal reflux disease. To determine the impact of GERD on oral tissues. To identify the main dental manifestations in patients with GERD. To analyze diagnostic methods for dental disorders. To evaluate the relationship between the severity of GERD and the severity of dental changes. To develop recommendations for prevention and comprehensive treatment.

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Research methods. To study the relationship between gastroesophageal reflux disease (GERD) and dental disorders, a comprehensive clinical and instrumental study was conducted. The study included patients with a confirmed diagnosis of GERD of varying severity. The diagnosis was established based on clinical symptoms (heartburn, acid regurgitation, discomfort behind the breastbone), as well as instrumental methods - fibrogastroduodenoscopy (FGDS) and 24-hour pH-metry. The dental examination included: clinical examination of the oral cavity; assessment of the condition of hard dental tissues; detection of enamel erosion and carious lesions; determination of the degree of dental hyperesthesia; assessment of the condition of the oral mucosa; examination of periodontal tissues; determination of the level of oral hygiene.

Additionally, saliva was analyzed (to determine pH, buffering capacity, and salivary flow rate). Patient questionnaires were also administered to identify complaints and risk factors. The data obtained were statistically analyzed using variation analysis methods to determine the reliability of the results.

Study results

The study revealed a significant correlation between gastroesophageal reflux disease and dental disorders. Most patients with GERD showed changes in the hard tissues of the teeth. The most common manifestation was enamel erosion, primarily on the palatal and vestibular surfaces of the teeth. The severity of erosions increased with the long-term progression of the disease. Dental hyperesthesia was observed in a significant proportion of patients and manifested itself as increased sensitivity to temperature, chemical, and mechanical irritants. An increased prevalence of caries was also noted, which is associated with a disruption of the protective properties of saliva and a decrease in its buffering capacity. Saliva analysis revealed a decrease in pH, which contributes to enamel demineralization. Soft tissue symptoms included signs of mucosal inflammation,

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a burning sensation, and dry mouth (xerostomia). A number of patients were diagnosed with periodontal disease of varying severity.

For the first time in dentistry, the role of the diffuse neuroendocrine system of the oral cavity, esophagus, and stomach in the development and progression of isolated and combined pathologies, as well as its influence on key cellular proliferation processes, has been established. Preclinical and instrumental signs of inflammatory periodontal diseases have been identified based on the study of apoptosis indicators and the diffuse neuroendocrine system.

For the first time, the main indicators of cell proliferation were studied and the universal role of proliferating cell nuclear antigen as the main diagnostic and prognostic criterion for assessing the course of the pathological process in the oral cavity and digestive organs was demonstrated.

For the first time in dentistry and gastroenterology, the influence of Helicobacter expansion on the course of the inflammatory-dystrophic process, indicators of cellular renewal and the diffuse neuroendocrine system in patients with inflammatory periodontal diseases has been determined.

The treatment tactics for patients with chronic generalized periodontitis in combination with diseases of the esophagus, stomach and gallbladder have been determined depending on the state of the diffuse neuroendocrine system and cellular renewal of epithelial cells of the gingival mucosa.

For the first time, based on a study of the indicators of the diffuse neuroendocrine system and cellular renewal of gingival epithelial cells, criteria for the onset of remission, as well as factors determining the recurrent course of chronic generalized periodontitis, have been established.

Based on the conducted research, new diagnostic criteria for the onset, course, and outcome of inflammatory periodontal diseases associated with gastrointestinal pathology were developed. It was established that when assessing the course and treatment outcomes of chronic generalized periodontitis, it is necessary to consider the dynamics of changes in the quantitative density of mast

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cells in the gingival mucosa and antrum, which produce serotonin, melatonin, and histamine. The role of Helicobacter pylori expansion in the onset and course of generalized periodontitis, as well as its impact on the synthesis of essential biologically active substances and regenerative factors, was determined. Uncontrolled apoptosis was found to be the primary factor in the progression of the pathological process both in the oral cavity and in the lower gastrointestinal tract, the significance of which is determined by the degree of bacterial expansion and the patient's local hormonal status. It has been proven that the state of the oral serotonin-melatonin system determines the course and prognosis of isolated and combined periodontal and gastrointestinal pathologies, and its parameters can be used for diagnostic purposes and to determine treatment strategies. Values for proliferating cell nuclear antigen and apoptosis index have been determined, which can form the basis for expert systems for assessing the effectiveness of treatment for chronic generalized periodontitis associated with gastrointestinal pathology.

Dynamic characteristics of the indicators of hormonal regulation of reparative processes, the indicators of which are the proliferating cell nuclear antigen and the serotonin-melatonin system, can be used in dentistry and the clinic of internal diseases to assess the prognosis of the course of diseases and early preclinical and instrumental manifestations of relapse.

Chronic inflammatory periodontal diseases are associated with altered rates of oral epithelial cell turnover and the structural and functional organization of mast cells, which secrete serotonin, melatonin, and histamine. Chronic inflammatory periodontal diseases associated with gastroesophageal reflux disease, chronic gastritis, and chronic cholecystitis are associated with more pronounced changes in epithelial cell turnover and the diffuse neuroendocrine system of the oral cavity and upper gastrointestinal tract. Impaired epithelial cell turnover in the oral, esophageal, and gastric mucosa is associated with the persistence of Helicobacter pylori and the influence of the main neurohormones of mast cells, which produce

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serotonin, melatonin, and histamine. The uniformity and unidirectionality of changes in diffuse neuroendocrine system parameters and cellular renewal of epithelial cells in the oral, esophageal, and gastric mucosa indicate common mechanisms for the development of inflammatory and degenerative changes in the upper gastrointestinal tract. Severe chronic generalized periodontitis is characterized by suppression of cellular proliferating nuclear antigen and uncontrolled apoptosis, rendering any form of conservative treatment ineffective. Severe periodontitis is associated with pronounced atrophy of the esophageal and gastric mucosa, suggesting the existence of universal mechanisms regulating apoptosis in the oral cavity and upper gastrointestinal tract. Combined pathology of the periodontium, esophagus, stomach, and biliary system requires long-term comprehensive treatment, with remission occurring later. To diagnose, assess the severity of the course and predict inflammatory periodontal diseases, in addition to traditional clinical and instrumental methods, it is necessary to study proliferative factors and the main indicators of the diffuse endocrine system of the oral cavity.

It was found that the severity of dental problems directly correlates with the frequency and duration of reflux episodes. Patients with more severe GERD had more pronounced oral lesions. Thus, the study results confirm the close clinical relationship between GERD and dental problems and emphasize the need for a comprehensive approach to diagnosis and treatment.

Chronic generalized periodontitis is characterized by changes in the structural organization of the diffuse neuroendocrine system of the oral mucosa, which are detectable in 100% of cases. This is manifested by an increase in the overall mast cell population of the gingival mucosa in mild to moderate periodontitis and a decrease in their number in severe stages of the disease. Therefore, chronic generalized periodontitis can be classified as a disease caused not only by a bacterial infection but also by neurohormonal abnormalities.

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Conclusions. The nature of changes in diffuse neuroendocrine system parameters and cellular renewal of epithelial cells of the gingival mucosa depend on the severity of periodontitis. Chronic generalized periodontitis of mild to moderate severity is characterized by hyperplasia of mast cells producing melatonin, serotonin, and histamine, and an increased apoptosis index against a background of decreased cellular proliferation of gingival epithelial cells. Severe periodontitis is characterized by a complete depletion of the main elements of the local neurohormonal system, which is accompanied by uncontrolled apoptosis and a sharp suppression of proliferating cellular nuclear antigen. The identified changes in the quantitative characteristics of mast cells of the gingival mucosa correlated with their levels in the antrum of the stomach; however, changes in the gingival mucosa were more pronounced than in the gastric mucosa. This fact indicates a single unidirectional response of the mast cell population and a consistent involvement in the inflammatory-dystrophic process of the digestive tract, starting from its proximal sections.

A high degree of correlation between the apoptosis index of the gingival mucosa and the antral part of the stomach in patients with chronic generalized periodontitis with combined pathology of the digestive organs indicates universal mechanisms for regulating apoptosis in the upper gastrointestinal tract.

Helicobacter pylori infection in chronic generalized periodontitis associated with upper gastrointestinal tract pathology is detected in 100% of cases in periodontal pockets, 84% in the lower third of the esophagus, and 82.5% in the antrum of the stomach. The severity of inflammatory and degenerative changes in the periodontium correlates with the degree of bacterial expansion in the esophagus and stomach.

The presence of unified mechanisms of neuroendocrine regulation and cellular renewal in epithelial cells of the oral cavity, esophagus, and stomach necessitates comprehensive treatment for patients with combined periodontal and gastrointestinal pathology. Low proliferating cell nuclear antigen (PcM <50%)

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and high apoptosis index (Iapor1 >150%), coupled with total hypoplasia of gingival mast cells secreting serotonin, melatonin, and histamine, are absolute indications for surgical treatment of chronic generalized periodontitis.

Achieving remission in chronic generalized periodontitis of varying severity with associated gastrointestinal pathology depends not only on the stage of the disease but also on the presence of atrophic and erosive lesions of the upper gastrointestinal tract. Combination therapy has the best results in patients with superficial gastritis, catarrhal esophagitis, and acalculous cholecystitis.

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