DOI 10.29254/2077-4214-2023-4-171-15-19
UDC 616.314.17-008.1:616-002]616-036=111

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A MODERN VIEW ON THE PREVALENCE AND DEVELOPMENT OF PERIODONTAL TISSUE DISEASES AT GASTROESOPHAGEAL REFLUX DISEASE

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The problem of gastroesophageal reflux disease (GERD) is attracting more and more attention from researchers and doctors due to the high prevalence and diversity of its clinical manifestations. Gastroesophageal reflux disease is accompanied by dental manifestations. Most patients complain of changes in the oral cavity.

The aim of the study – to summarise the literature data on the prevalence and mechanisms of periodontal tissue diseases at gastroesophageal reflux disease according to the current scientific literature.

Recent publications show that the development of periodontitis is often caused by internal organs pathology. Other researchers suggest that generalised somatic diseases are comorbid in 85% of cases and, in turn, exacerbate the pathological process in the periodontium. The problem of comorbidity is to identify the primary disease. Scientists and clinicians are faced with the following variants of combined pathology interactions: periodontal manifestations of somatic diseases, combination of periodontal diseases and lesions of other systems, and clinical changes outside the periodontium caused by periodontal disease. The need for an in-depth study of GERD is associated with the fact that very often its dental manifestations can exist at patient without pronounced subjective symptoms. Patients may also have no complaints from the gastrointestinal tract. However, during dental examination, the dentist may assume presence of this disease based on the clinical manifestations of a set of changes in the oral cavity.

Thus, it can be concluded that periodontal tissue lesions associated with gastroesophageal reflux disease have a wide range of clinical manifestations and an upward trend.

Key words: *gastroesophageal reflux disease, inflammatory processes, periodontitis, combined pathology, prevention.*

Connection of the publication with planned research works.

This project is a fragment of the scientific topic of the Department of Orthopaedic Dentistry: "Ethiopathogenesis and treatment of major dental diseases using modern methods of rehabilitation of dental patients on the background of concomitant somatic pathology", state registration number 0122U000041.

Introduction.

High prevalence of periodontal disease, its tendency to progress with subsequent tooth loss, its onset at a young age, the reduced quality of the patients' life and significant financial costs make this medical and social problem relevant [1].

In Ukraine, periodontal tissue damage is diagnosed with varying frequency: at the age of 35-40 years, 50-80% of patients manifest symptoms of the disease, while after 40 years – 99.3%, that generally reflects the global tendency of pathology development. It has been proven that generalised periodontitis leads to tooth loss 5 times more often than in case of caries complications [2, 3]. It is important that early manifestations of the inflammatory process in periodontal tissues are already evident in adolescents and young adults with bleeding gums (32.1%), plaque (21.2%) and periodontal pockets (5.6%) [4, 5]. With age, inflammatory and destructive manifestations in the periodontium are diagnosed more often. Thus, at the age of 35-44 years old, the number of patients with periodontal pockets is 28.4%, at the age of 45-64 years old, there are 44.3% of such patients, and at the age of 65-74 years old – 73.7%. Chronic generalised periodontitis is more common and more severe at men [6, 7].

The problem of gastroesophageal reflux disease (GERD) is attracting more and more attention from researchers and doctors due to the high prevalence and diversity of its clinical manifestations. For example, GERD symptoms are diagnosed at 20-40% of the adult population aged 25 to 50 years old with equal frequency at men and women [3, 8, 9].

GERD is a chronic disease that occurs as a result of regularly repeated pathological effects of gastric contents on the esophageal mucosa as a result of gastroesophageal reflux [6, 7, 10].

One of the atypical (extra-esophageal) syndromes of gastroesophageal reflux disease is dental. According to various authors, the dental syndrome at GERD, which occurs as a result of inflammatory changes in the oral mucosa under the influence of refluxate, is characterised by the appearance of tooth enamel erosion, inflammation of periodontal tissues, and changes in the fungiform papillae of the tongue [8, 10, 11]. However, the prevalence, nature and severity of lesions of the oral cavity organs and tissues have not been studied. The mechanisms of their development remain unclear.

The aim of the study.

To summarise the literature data on the prevalence and mechanisms of periodontal tissue diseases at gastroesophageal reflux disease according to the current scientific literature.

Main part.

Gastroesophageal reflux disease is accompanied by dental manifestations. Most patients complain of

changes in the oral cavity. Characteristic features are a high prevalence of tooth damage by caries and non-carious lesions, inflammatory and inflammatory-dystrophic changes at periodontal tissues, subjective and objective changes at tongue and lips [9, 10].

Inflammatory processes in the oral cavity may be the first clinical signs of disorders at diseases of the digestive system. Presence of somatic diseases in the human body makes a significant difference in the etiopathogenesis of periodontal diseases. Combined pathology is characterised by a mutually burdened course of diseases due to the presence of a close functional relationship between the affected organs [6, 11].

It is a proven fact that the development of periodontitis is due to the combined effect of various exogenous and endogenous factors that prevail over the adaptive and protective properties of periodontal tissues. In turn, the barrier function of periodontium depends on anatomical and physiological features, in particular, the ability of gingival epithelium to keratinise, the direction of collagen fibre bundles, gingival turgor, plasma and mast cell activity, and the content of Ig A in gingival fluid. The provoking factors of this pathology are usually divided into local and general. Currently, a sufficient number of scientific papers have been published, in which the leading local factors invariably include malocclusion, dental plaque, the state of oral microflora, traumatic occlusion, dentures, orthodontic appliances, inferior fillings and unsanitised oral cavity. Independent predictors of periodontitis are heredity, dermatoglyphic parameters, and antigenic associations of the blood group system [11, 12].

A special role in the initiation and maintenance of the inflammatory process in the periodontium is played by microorganisms that produce enzymes, endotoxins, surface and capsular bacteria components [13].

A number of scientists in their works identify whole complexes of periodontogenic bacteria. For example, Yoshikawa H. and co-authors name bacteria-markers of periodontitis as Porphyromonas gingivalis (Pg), Actinobacillus actinomycetemcomitas (Aa), Bacteroides forthytus (Bf). A group of other researchers led by Socransky identified a bacterial risk factor for periodontal disease. It includes Porphyromonas gingivalis (Pg), Bacteroides forthytus (Bf) and Treponema dentolika (Td). Thus, there is a bacterial "risk factor" necessary for the periodontitis formation [13].

In modern understanding, it is the periodontogenic antigens of bacteria that regulate local tolerance and determine the unity of the "information transfer system" through a series of nuclear factors kappa B from the mucous membranes of the oral cavity and gastrointestinal tract. Cytokines and chemokines regulate the inflammatory process both locally and in distant areas of the body [14].

It has been proven that various endotoxins, including lipopolysaccharides and structural antigens of membranes of gram-negative bacteria, leukotoxins, lipoic acid are highly toxic products that activate the complement system, induce bone resorption and cause the Shwartzman phenomenon – local tissue necrosis. The end bacterial metabolism products, such as amines, indole, glycan and mercaptans, have an additional toxic effect. These substances inhibit the activity of fibroblasts, thereby weakening the repair of connective tissue and the bone's cellular process. Among the enzymes produced by microorganisms, collagenase, hyaluronidase, phosphatase, trypsin-like proteases, and aminopeptidase are capable of destroying periodontal tissue. The latter play the role of chemotactic factors for polymorphonuclear leukocytes, macrophages, and polynuclei, catalysing the acute stage of the inflammatory reaction. In addition to bone-resorbing effects, bacterial cell membrane components have immunosuppressive activity. The fact that bacteria evade the effects of nonspecific immune defence components and convert them into capsular components remains important [11, 12, 15].

Thus, the degree and nature of periodontal tissue damage is determined by the direct cytotoxic effect of microorganisms and evasion of immune reactions due to the complex action of all bacteria damaging factors.

Recent publications show that the development of periodontitis is often caused by internal organs pathology. Other researchers suggest that generalised somatic diseases are comorbid in 85% of cases and, in turn, exacerbate the pathological process in the periodontium. The problem of comorbidity is to identify the primary disease. Scientists and clinicians are faced with the following variants of combined pathology interactions: periodontal manifestations of somatic diseases, combination of periodontal diseases and lesions of other systems, and clinical changes outside the periodontium caused by periodontal disease [16, 17].

At the same time, a group of diseases with 100% pattern of periodontal tissue lesion is defined. These include hypertension, central nervous system disorders, atherosclerosis, metabolic disorders, vitamin deficiencies, osteoporosis and acid-dependent diseases (gastric and duodenal ulcers). Some studies point to violation of the regulatory influence of the autonomic nervous system, which leads to trophic disorders and decrease in the periodontal barrier function. The relationship between periodontitis and ulcerative colitis as a functionally unified gastrointestinal system has also been substantiated. As a rule, the severity and duration of diseases of internal organs correlates with the severity of chronic generalised periodontitis [15, 18].

Close relationship between diseases of the oral cavity and the gastrointestinal tract has been known for a long time. This is due to the anatomical proximity, similarity of blood supply, innervation and humoral regulation of the digestive tract. High dental morbidity is observed at presence of gastroduodenal zone diseases (chronic gastritis and duodenitis, duodenal ulcer). Thus, A.S. Artiushkevych provides data on the presence of changes in periodontal tissues at patients with various pathologies of the gastrointestinal tract in 76-91% of cases. He also diagnosed hyperesthesia of the hard tissues of teeth at patients with GERD and proposed methods of medical correction. In general, scientific literature contains few and fragmentary data on the patterns of periodontitis on GERD background [19].

A number of researchers suggest that chronic GERD, despite the sufficient monomorphism of clinical manifestations, is an etiologically and pathogenetically heterogeneous disease. Periodontal tissue lesions can develop under the combined influence of local and general factors on the background of changes in the body's reactivity. A number of pathogenetic factors that play a leading role in the development of periodontitis are similar

to those that activate the inflammatory process in GERD [18, 19, 20].

Motor-tonic disorders of oesophagus on the background of lower sphincter dysfunction cause a violation of the mineral composition and viscosity of saliva with the development of acidification (pH<5.0) and, according to many scientists, trigger a cascade of dental lesions in GERD. That is, constant excess of hydrochloric acid hydrogen ions "breaks through" the pre-epithelial level of protection, thereby disrupting the synthesis of epidermal factors, Ig A, lysozyme, lactoperoxidase, prostaglandin and complement, which makes it difficult to implement nonspecific mechanisms of mucous membranes. As a result, succession develops – the sequential replacement of saprophytic bacterial strains with opportunistic pathogens with disproportionate reproduction of the latter [21]. Whether pathological process develops in the periodontium or not, depends on a complex of factors such as local tissue reaction, antibacterial effect of saliva, immune mechanisms, periodontal tissue regeneration and general body reactivity. When the balance is tilted towards alteration, inflammation, chemotaxis, phagocytosis, activation of the complement system, production of interleukins and leukotrienes, and antibody production occur. It is important to note that normal activity of polymorphonuclear neutrophilic leukocytes and antibody production limits the inflammatory process without developing disease [22, 23, 24].

On the other hand, numerous studies have emphasised the importance of inflammatory damage at chronic diseases, in the context of their progression with the involvement of catabolic proinflammatory cytokines – IL-1, TNF-α – in the pathological process, which are associated with significant periodontal problems at patients with acid-dependent diseases. For example, TNF-α and IL-1 induce the synthesis of matrix metalloproteinases by fibroblasts, resulting in a decrease in production of collagen synthetase and proteoglycans, and trigger further cytokine response. At the same time, prostaglandin E2 activates osteoclasts and thus stimulates bone resorption [25, 26, 27].

Disruption of trophism in bone tissue and periodontal fibres caused by ischaemia, arterial and venous hyperaemia and lymphostasis, formation of an intraepithelial gap with gingival epithelial dystrophy at persistent inflammatory process supported by constant exposure to refluxate and bacteria are the main pathogenetic factors of chronic generalised periodontitis on the GERD background. As a result, there are dystrophic changes in periodontal tissues and necrosis of certain areas [28, 29, 30].

The need for an in-depth study of GERD is associated with the fact that very often its dental manifestations can exist at patient without pronounced subjective symptoms. Patients may also have no complaints from the gastrointestinal tract. However, during dental examination, the dentist may assume presence of this disease based on the clinical manifestations of a set of changes in the oral cavity.

Conclusions.

Thus, after analysing the literature, it can be concluded that periodontal tissue lesions associated with gastroesophageal reflux disease have a wide range of clinical manifestations and an upward trend. Therefore, an in-depth study of the cause-and-effect relationships and mechanisms of periodontal tissue diseases at gastroesophageal reflux disease is important for the development of preventive measures.

Prospects for further research.

Considering the relevance and prevalence of periodontal tissue lesions associated with gastroesophageal reflux disease among the population of Ukraine, it is necessary to continue studying this problem and identifying the leading causes that determine their development.

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СУЧАСНИЙ ПОГЛЯД НА ПОШИРЕНІСТЬ ТА РОЗВИТОК ЗАХВОРЮВАНЬ ТКАНИН ПАРОДОНТУ ПРИ ГАСТРОЕЗОФАГЕАЛЬНІЙ РЕФЛЮКСНІЙ ХВОРОБІ

Безушко А. В., Гасюк П. А., Воробець А. Б.

Резюме. *Вступ.* Значна поширеність захворювань пародонта, схильність їх до прогресування з подальшою втратою зубів, поява у молодому віці, зниження якості життя пацієнтів і значні фінансові затрати обумовлює актуальність даної медичної і соціальної проблеми.

Мета роботи – узагальнення літературних даних щодо поширеності та механізмів розвитку захворювань тканин пародонту при гастроезофагеальній рефлюксній хворобі згідно сучасної наукової літератури. Для аналізу поширеності та механізмів розвитку захворювань тканин пародонту при гастроезофагеальній рефлюксній хворобі використано бібліографічний, аналітичний методи дослідження та методику системного підходу.

Результати. З публікацій останніх років стає очевидним, що розвиток пародонтиту доволі часто зумовлений патологією внутрішніх органів. Висока стоматологічна захворюваність спостерігається при наявності захворювань гастродуоденальної зони (хронічному гастриті і дуоденіті, виразковій хворобі дванадцятипалої кишки). Запальні процеси в ротовій порожнині можуть являтися першими клінічними ознаками порушень при захворюваннях травної системи організму. Наявність соматичних захворювань в організмі людини вносить істотну відмінність в етіопатогенез захворювань пародонта. Для поєднаної патології характерним є взаємообтяжений перебіг захворювань за рахунок наявності тісного функціонального зв'язку між ураженими органами. Ряд дослідників висловлюють думку, що генералізований пародонтит, незважаючи на достатню мономорфність клінічних проявів, постає етіологічно і патогенетично гетерогенним захворюванням. Моторно-тонічні розлади стравохода на тлі дисфункції нижнього сфінктера зумовлюють порушення мінерального складу й в'язкості слини із розвитком ацидифікації (pH<5,0) та, на думку багатьох науковців, запускають каскад стоматологічних ушкоджень при ГЕРХ.

Висновки. Отже, провівши аналіз літературних джерел, можна зробити висновок, що ураження тканин пародонту, асоційовані з гастроезофагеальною рефлюксною хворобою, мають широкий спектр клінічних проявів та тенденцію до зростання. Тому для розробки профілактичних заходів важливим є поглиблене вивчення причинно-наслідкових зв'язків та механізмів розвитку захворювань тканин пародонту при гастроезофагеальній рефлюксній хворобі.

Ключові слова: гастроезофагеальна рефлюксна хвороба, запальні процеси, пародонтит, поєднана патологія, профілактика.

A MODERN VIEW ON THE PREVALENCE AND DEVELOPMENT OF PERIODONTAL TISSUE DISEASES AT GASTROESOPHAGEAL REFLUX DISEASE

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Abstract. *Inroduction.* High prevalence of periodontal diseases, their tendency to progress with subsequent tooth loss, occurrence at a young age, reduced quality of life and significant financial costs make this medical and social problem relevant.

The aim of the study – to summarise the literature data on the prevalence and mechanisms of periodontal tissue diseases at gastroesophageal reflux disease according to the current scientific literature. To analyse the prevalence

and development mechanisms of periodontal tissue diseases at gastroesophageal reflux disease, bibliographic, analytical research methods and the method of systematic approach were used.

Results. Recent publications show that the development of periodontitis is often caused by pathology of internal organs. High dental morbidity is observed at presence of the gastroduodenal zone diseases (chronic gastritis and duodenitis, duodenal ulcer). Inflammatory processes in the oral cavity may be the first clinical signs of disorders at diseases of the digestive system. The presence of somatic diseases in the human body makes a significant difference in the etiopathogenesis of periodontal diseases. Combined pathology is characterized by a mutually burdened course of diseases due to presence of close functional relationship between the affected organs. A number of researchers suggest that generalized periodontitis, despite sufficient monomorphism of clinical manifestations, is an etiologically and pathogenetically heterogeneous disease. Motor-tonic disorders of the esophagus against the background of lower sphincter dysfunction cause violation of the mineral composition and viscosity of saliva with the development of acidification (pH < 5.0) and, according to many scientists, trigger a cascade of dental damage at GERD.

Conclusions. Thus, after analyzing the literature, it can be concluded that periodontal tissue lesions associated with gastroesophageal reflux disease have a wide range of clinical manifestations and an upward trend. Therefore, for the development of preventive measures, it is important to study in-depth the cause-and-effect relationships and mechanisms of periodontal tissue diseases at gastroesophageal reflux disease.

Key words: gastroesophageal reflux disease, inflammatory processes, periodontitis, combined pathology, prevention.

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Conflict of interest:
The Authors declare no conflict of interest.

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Received 19.05.2023
Accepted 06.11.2023

DOI 10.29254/2077-4214-2023-4-171-19-24
UDC 616.311:612.017.01

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MODERN VIEW ON THE ISSUES OF IMMUNOLOGICAL TOLERANCE OF THE ORAL MUCOSA

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Modern molecular biology and genetics make it possible to consider the epithelium of the mucosal system through the prism of immunology and expand the understanding of the implementation mechanisms immune' response upon contact with the microflora of the oral cavity.

The purpose of the research is to analyze literature sources based on Scopus, Web of Science, MedLine, PubMed, NCBI databases, the study of which does not exceed 10 years, including literature reviews and the results of clinical studies.

Being part of the mucous membrane system, the buccal and gingival epithelium takes an active position in relationships with irritating factors from the external and internal environment. This allows it to be used to study the physiology and reactivity of mucous membranes, including as an indicator of local and general disorders of homeostasis. Oral mucosa is in a state of tolerance, which is only occasionally broken in the presence of certain "danger signals". Typically, regulatory T cells are considered a key player in this tolerance.

The oral mucosa is a site of intense immune activity where a wide variety of immune cells occur to provide the first line of defense against pathogenic organisms.

Key words: *immune response, mucosa, dendritic cells, effector sites.*