

ETIOPATHOGENETIC ASPECTS OF SYNTROPY AND MUTUAL BURDEN IN THE PATHOLOGY OF THE GASTROINTESTINAL TRACT AND THE DISEASES OF THE ORAL CAVITY

T.I. Pupin, Z.M. Honta, O.M. Nemesh, I.V. Shylyivskiy, Kh.B. Burda, O.O. Maksym

Danylo Halytsky Lviv National Medical University, Lviv, Ukraine

Key words: diseases of the oral cavity, generalized periodontitis, mucous membrane of the oral cavity, diseases of the gastrointestinal tract, etiology, clinical course, pathogenesis, syntropy.

Bukovinian Medical Herald.
2024. V. 28, № 2 (110). P. 126-132.

DOI: 10.24061/2413-0737.28.2.110.2024.19

E-mail:
zoryanagonta@gmail.com

Abstract. The purpose of the work is to study the main etiopathogenetic mechanisms of development and peculiarities of the clinical course in the comorbidity of diseases of the oral cavity and pathology of the gastrointestinal tract by analyzing the results of scientific research published in modern scientific publications.

Material and methods. Search, systematization, elaboration and analysis of scientific works of domestic and foreign authors, which present the modern results of research on etiopathogenesis and peculiarities of the clinical course in comorbidity of diseases of the oral cavity and gastrointestinal tract, with the involvement of search engines Google Scholar, PubMed and other electronic resources. The bibliosematic and analytical methods are used in the research.

Results. The problem of syntropies, the basis of which is the association of etiopathogenetic and pathogenetic factors of certain diseases, is an actual problem in modern medicine. The analysis of data from modern scientific literary sources proves that the development and progression of periodontal and oral mucosa diseases depends not only on the influence of local factors but also is a reaction to the influence of pathogenic factors that arise as a result of diseases of the gastrointestinal tract. Steady persistence of microflora in certain areas of the digestive tract can complicate the course of dental and gastrointestinal diseases. A gradual change in the properties of representatives of the conditionally pathogenic group of microorganisms, their association with specific diseases of the oral cavity and gastrointestinal tract indicates their role in the pathogenesis of many diseases. Mechanisms of free radical damage to cellular structures and changes in humoral and cellular immunity are important common links in the pathogenesis of periodontal and gastrointestinal tract diseases. The development of an imbalance of the endocrine system and psycho-emotional stress has a significant impact on the process of mutual burden of diseases of the gastrointestinal tract and oral cavity.

Conclusion. Today considerable factual material has been accumulated regarding the understanding of common mechanisms of etiopathogenesis and syntropy of diseases of the oral cavity and pathology of the gastrointestinal tract. This knowledge makes it possible to develop effective comprehensive measures for the diagnosis, prevention and treatment of both diseases of the oral cavity and diseases of the gastrointestinal tract, taking into account the mutual burden of these pathologies.

ЕТІОПАТОГЕНЕТИЧНІ АСПЕКТИ СИНТРОПІЇ ТА ВЗАЄМОБТЯЖЕННЯ ПРИ ПАТОЛОГІЇ ШЛУНКОВО-КИШКОВОГО ТРАКТУ І ЗАХВОРЮВАННЯХ РОТОВОЇ ПОРОЖНИНИ

T.I. Пупін, З.М. Гонта, О.М. Немеш, І.В. Шилівський, Х.Б. Бурда, О.О. Максим

Ключові слова: захворювання порожнини рота, генералізований пародонтит, слизова оболонка порожнини рота, хвороби шлунково-кишкового тракту, етіологія, клінічний перебіг, патогенез, синтропія.

Буковинський медичний

Резюме. Мета роботи – вивчення основних етіопатогенетичних механізмів розвитку та особливостей клінічного перебігу при коморбідності захворювань порожнини рота і патології шлунково-кишкового тракту шляхом аналізу результатів наукових досліджень, висвітлених у сучасних наукових публікаціях.

Матеріал і методи. Пошук, систематизація, опрацювання та аналіз наукових праць вітчизняних і зарубіжних авторів, в яких представлені сучасні результати досліджень етіопатогенезу та особливостей клінічного перебігу при коморбідності захворювань порожнини рота і шлунково-кишкового тракту, із залученням пошукових систем Google Scholar, PubMed та інших електронних ресурсів. У дослідженні застосовано бібліосемантичний та

вісник. 2024. Т. 28, № 2
(110). С. 126-132.

аналітичний методи.

Результати. Проблема синтропії, в основі якої лежить асоціація етіологічних і патогенетичних факторів певних захворювань, є актуальною проблемою сучасної медицини. Аналіз даних сучасних наукових джерел літератури доводить, що розвиток і прогресування стоматологічних захворювань залежить не тільки від впливу місцевих чинників, а є реакцією на дію патогенних факторів, які виникають внаслідок захворювань шлунково-кишкового тракту.

У хворих з патологією органів системи травлення відбувається суттєве послаблення захисних функцій імунної системи, дисбіотичні порушення як у шлунково-кишковому тракті, так і в ротовій порожнині. Вони сприяють поглибленню та генералізації патологічного процесу в тканинах порожнини рота, обтяженню, прогресуванню та ускладненню стоматологічних захворювань. Стійка персистенція мікрофлори в окремих ділянках травного тракту ускладнює перебіг стоматологічних і шлунково-кишкових захворювань. Важливою спільною ланкою патогенезу захворювань пародонта та шлунково-кишкового тракту є механізми вільнорадикального ураження клітинних структур та зміни гуморального та клітинного імунітету. Розвиток дисбалансу ендокринної системи та психоемоційне напруження має значний вплив на процес взаємного обтяження хворобами шлунково-кишкового тракту та ротової порожнини.

Висновок. На сьогодні накопичено значний фактичний матеріал щодо розуміння загальних механізмів етіопатогенезу та синтропії захворювань порожнини рота та патологій шлунково-кишкового тракту. Ці знання дають змогу розробити ефективні комплексні заходи діагностики, профілактики та лікування як захворювань порожнини рота, так і захворювань шлунково-кишкового тракту з урахуванням взаємного обтяження цих патологій.

Introduction. Numerous scientific studies during many years have convincingly shown that any systemic disorders in the human body are reflected in the oral cavity in some way or another [1,2,3,4]. At the same time, the severity of damage to the organs of the oral cavity is directly dependent on the duration and severity of the course of somatic diseases, by means of a negative influence on the human body's defense system, strengthen the course of dental diseases. The analysis of data from modern scientific literary sources proves that the development and progression of periodontal and oral mucosal diseases depends not only on the influence of local factors, but is also a reaction to the influence of pathogenic factors that arise as a result of systemic diseases of the human body [3,5,6]. In addition, there are research results on the influence of the condition of the oral cavity on the occurrence and course of general somatic diseases [7,8,9].

The modern studies of many scientists indicate a close relationship between a high level of dental morbidity, including periodontal tissue diseases, and lesions of the gastrointestinal tract (GIT). The state of the teeth, periodontal tissues, and oral mucosa, as the beginning of the digestive tract, is closely related to the pathology of the esophagus, stomach, intestines, and digestive glands. Changes in the oral cavity are often the primary manifestations of gastrointestinal tract pathology [10,11,12,13]. The problem of syntropies, the basis of which is the association of etiological and pathogenetic factors of certain diseases, is an actual problem in the modern clinic of internal diseases. In chronic gastritis, peptic ulcer disease of the stomach and duodenum, chronic colitis and enterocolitis, a variety of changes in the mucous

membrane of the oral cavity (ММОС) are registered, the degree of expression of which depends on the form, severity and duration of the course of the main disease, which is explained by a close anatomical and functional connection [14,15,16,17].

Aim. The study the main etiopathogenetic mechanisms of development and peculiarities of the clinical course in the comorbidity of diseases of the oral cavity and pathology of the gastrointestinal tract by analyzing the results of scientific research published in modern scientific publications.

The analysis of data from modern scientific literary sources proves that the development and progression of diseases of the oral cavity depends not only on the influence of local factors, but is also a reaction to the influence of etiopathogenetic factors that arise as a result of diseases of the gastrointestinal tract [16,17,18,19]. At the same time, according to a number of authors, pathological changes in the oral cavity become a source of chronic infection and lead to gastrointestinal tract dysfunction. Inflammatory-dystrophic processes in the maxillofacial system lead not only to the loss of teeth and a decrease in chewing function, but also to a violation of the secretory-motor function of the organs of the digestive system [1,10,16]. In addition, a decrease in the rate of saliva secretion in inflammatory diseases of the gastrointestinal tract has been diagnosed, and a direct relationship between the degree of severity of generalized periodontitis (GP) and the level of compensation for gastrointestinal diseases has been defined [15,17,20].

With increased acidity of gastric juice, increased salivation, hypertrophy of the tongue papillae, pallor and

Оригінальні дослідження

swelling of the oral mucosa, catarrhal gingivitis are often observed, while with decreased acidity of the stomach, a coated tongue, papilla smoothness, hyposalivation, dry lips, angular cheilitis, bad breath, bitterness, heartburn, rash on the red border of the lips and on the oral mucosa are revealed. It is known that the aftertaste in the mouth is associated with a violation of the functions of the closing (valvular) structures of the upper part of the digestive tract: with insufficiency of the pharyngeal and cardiac valves, sour aftertaste is noted, and with insufficiency of the pharyngeal, cardiac, and pyloric valves – bitter one, the so-called "duodenogastric reflux" [6,10,15].

In chronic gastroduodenitis (CGD), recurrent forms of stomatitis, herpetic lesions of the lips, various forms of cheilitis and gingivitis, edema, petechiae, and increased mucosal pattern are detected [13]. Studies show that a decompensated form of caries is found in children with CGD, its prevalence and intensity are high, and the state of MMOC is unsatisfactory. A direct relationship between the pH of the oral fluid and the acid-forming function of the stomach has been established, therefore the hard tissues of the teeth in such patients are more affected by the carious process as a result of unsatisfactory hygiene and a more aggressive oral cavity environment [20].

Exfoliative cheilitis, xerostomia, dental hyperesthesia and inflammatory periodontal disease are more common in patients with gastroesophageal reflux disease (GERD) than in the general population. A pathognomonic symptom of gastroesophageal reflux disease (GERD) is erosion of tooth enamel, they are diagnosed in 20-48% of patients with pathological gastroesophageal reflux [9,21]. The severity of the damage to the hard tissues of the teeth correlates with the severity of GERD symptoms, the severity of esophagitis and is associated with a displacement in the pH of the oral fluid to the acidic side [22,23].

Many clinical observations have proven the mutually aggravating effect of gastric ulcer disease of the stomach and duodenum with GP. The inflammatory periodontal diseases in combination with gastric ulcer disease (GUD) were found in 83.3% of observed patients, duodenal ulcer disease (DUD) in 93.3% of observed ones. The frequency of periodontal tissue lesions ranges from 84.6% to 100% in patients with gastric ulcer disease of the stomach and duodenum. Damage to the mucous membrane (cheilitis and glossitis), periodontal diseases were diagnosed in the majority of patients with GUD and DUD [24].

Intestinal microbiocenosis is a highly organized system that affects the homeokinesis ("health - disease") of a person in various living conditions with the help of qualitative and quantitative shifts. The initial formation of this system begins with the microflora of the oral cavity. It is known that when the balance of the microbiocenosis of the oral cavity is disturbed, the number of pathogenic microflora (periodontopathogenic, odontogenic, tonsilogenic) increases, conditions are created for constant pathogenic contamination, as well as sensitization of the digestive tract and the body as a whole [9,25]. Enterobacteria, peptostreptococci, staphylococci, and streptococci were found from the alveolar groove in patients with diseases of the digestive tract. Convincing

data were obtained that the composition of the microflora in the stomach and small intestine is mostly similar to the bacterial spectrum of the oral cavity. According to the authors, the clinical course of periodontitis is directly dependent on the degree of dysbiosis of the oral cavity [26,27,28].

Against the background of existing diseases of the gastrointestinal tract in adults and children, the microbial landscape of the oral cavity, as well as the species and quantitative composition of its microbiocenosis, undergo radical changes. Certain microorganisms are associated with some diseases of the gastrointestinal tract, and the primacy of the lesions is unclear, whether the diseases of the gastrointestinal tract induce periodontal disease, or vice versa - periodontal diseases induce lesions of the tract. There is an assumption that the complex folded relief of the mucous membrane of the stomach and duodenum is a place of accumulation of random microflora. It is possible that the microorganisms that colonize the oral cavity and cause periodontal disease, getting into the stomach, persist in its pre-epithelial mucous layer and, accordingly, deepen the gastritis clinic [9].

The correlation between the level of population of the oral cavity by periodontopathogenic *Porphyromonas gingivalis*, *Tannerella forsythensis*, *Treponema denticola*, *Actinobacillus actinomycetemcomitans*) and cariogenic *Streptococcus mutans* and *S. Sobrinus* with precancerous diseases of the upper gastrointestinal tract (chronic atrophic gastritis, metaplasia and dysplasia of the mucosa) [13]. *Campylobacter concisus* (associated with periodontitis and gingivitis) was able to cause acute and chronic inflammation of certain parts of the gastrointestinal tract, and is often identified in Crohn's disease and ulcerative colitis [10,11].

Today, the main etiological factor of GUD and DUD is considered to be *Helicobacter pylori* - a gram-negative bacterium, the characteristic features of which are a multi-layered membrane, one unipolar flagellum, and pronounced urease activity. In case of infection, *Helicobacter pylori* colonizes the epithelium of the stomach, mainly its antral part, less often they are found in the body and bottom of the stomach. The place of greatest concentration of *Helicobacter pylori* is in the depth of the pits of the glands. The presence of bacteria in the duodenum was confirmed only in areas of gastric metaplasia. Due to high urease activity, the bacillus survives in an acidic environment [29,30].

Helicobacter pylori enters the digestive tract through the oral cavity with contaminated food. It has been proven that dental plaque contains *Helicobacter pylori*, and a large number of authors consider it to be an etiological factor in the occurrence of gingivitis and periodontitis [31]. The presence of *Helicobacter pylori* in dental plaque and the mucous membrane of the gums is a powerful source of infection of the mucous membrane of the stomach and duodenum. Along with the stomach, the oral cavity is a reservoir of *Helicobacter pylori*, and therefore a source of colonization of lower parts of the digestive tract [32,33].

The correlation of the prevalence and severity of oral cavity diseases with the stage, duration and severity of

gastrointestinal diseases indicates not only topographical relationships, but also a close reflex and humoral connection between the organs of the oral cavity and the gastrointestinal tract. It has been proven that MMOC receptors are a source of reflexes that influence the secretory and motor activity of the gastrointestinal tract. In turn, the oral cavity is the effector field of the reflexes from the internal organs. As a result, all diseases of the digestive organs to one degree or another are manifested on the MMOC [34,35].

The general key links in the pathogenesis of periodontal and gastrointestinal tract diseases are: an inflammatory reaction associated with the mechanisms of free radical damage to cellular structures, the development of an imbalance of the immune and endocrine systems, disturbances in microcirculation, neurohumoral regulation, psychosomatic relationships, changes in the metabolism of connective tissue, mineral metabolism, and deficiency vitamins, which leads to functional and organic disorders in the MMOC, the development of inflammatory and dystrophic changes in the periodontal tissues, disorders of the masticatory apparatus [34,36,37,38].

Among the factors that protect the periodontium and the mucous membrane of the gastroduodenal region, the state of the pro-oxidant-antioxidant system is of great importance. In particular, the activation of free-radical oxidation processes during periodontal tissue inflammation is associated with the pathogenic influence of associations of dental plaque microorganisms as an exogenous inducer of the radical oxidation chain and the development of mixed-type hypoxia, aggravating the course of inflammation. In conditions of hypoxia, all types of metabolic processes are disturbed, products of lipid peroxidation (LPO) accumulate, which enter into reactions that lead to the development of pathological conditions [37].

The mechanisms of local and general humoral immunity play an important role in the protection of the epithelium and tissue homeostasis in the combination of pathology of the gastrointestinal tract and the maxillofacial system [34]. The researchers established that in addition to humoral changes, shifts in cellular immunity are observed in the combined pathology of periodontal disease and peptic ulcer disease, namely: a decrease in the content of T-lymphocytes (suppressors and helpers), an increase in the content of B-lymphocytes and a decrease in the activity of lymphocyte blast transformation reactions. Diseases of the gastrointestinal tract and biliary tract lead to a deficiency in the absorption of important substrates, a violation of the barrier functions of the digestive system and are accompanied by significant immunological changes, which can be considered as a secondary acquired immunodeficiency and become one of the causes of the development of pathological changes in the periodontium. In addition, studies have shown that in patients with chronic gastroduodenitis, gastric and duodenal ulcers, the indicators of non-specific resistance of MMOC are reduced, in patients with duodenal ulcers, the activity of lysozyme and the adsorption activity of epitheliocytes is significantly lower than in healthy individuals [38,39].

A number of authors believe that one of the reasons for the rapid progression of inflammatory periodontal diseases is an increase of calcium-regulating hormones (parathyroid and calcitonin) in the blood of patients with peptic ulcer disease. Scientists consider an increased level of gastrointestinal hormones (gastrin, cholecystokinin, and others) in peptic ulcer disease to be the starting mechanism of this process. With the transition from the rarely recurring course of gastric and duodenal ulcers to frequent relapses, the frequency of gingivitis and periodontitis increases by almost 50%. These hormones, affecting directly or indirectly the C-cells of the thyroid gland, increase the production of calcitonin, which leads to a disturbance in the balance between hormones of hypo- and hypercalcemic action and an increase in resorptive processes in the periodontium [40,41,42].

The researchers have proven the possibility of the occurrence of periodontal pathology and ulcer disease under the influence of a shift in the autonomic nervous system, which is realized through the activation of the tone and its parasympathetic department. It was found that the reaction of periodontal tissues to acute stress in the experiment is characterized by the activation of lipid peroxidation, violation of the proteinase inhibitory potential, an increase in the content of sialic acids, a violation of hemocirculation, a decrease in bone density and the development of structural changes in them. With chronic stress, the development of destructive changes in periodontal tissues is observed, which is reflected in increased bone tissue resorption, disruption of the structure of cells and intercellular substance, and hemocirculation. The significant role of psycho-emotional stress in the pathogenesis of peptic ulcer disease is convincingly confirmed by clinical observations. 61% of patients associate relapses of DUD with the presence of psychotrauma. Acute and chronic stress, as a powerful etiological factor of DUD, also is an integral part of periodontal tissue inflammation [43,44].

Conclusions

1. Summing up the results of an analytical review of the literature on lesions of the organs and tissues of the oral cavity in patients with concomitant pathology of the gastrointestinal tract, it has been proven the significant increase in dental morbidity in this contingent of patients.

2. The occurrence of pathological changes in the hard tissues of the teeth, periodontal tissues and mucous membrane of the oral cavity occurs when the general condition of the body changes due to the actions of several endogenous factors. Established etiopathogenetic mechanisms of the damage to the digestive system's organs (teeth, periodontium, mucous membrane of the oral cavity, saliva glands, stomach, pancreas, liver, intestines), which have the genetic affinity and common development. Their close relationship and joint formation of the pathogenetic chain of the specified violations are proven.

3. There is a significant weakening of the protective functions of the immune system: dysbiotic disturbances are both in the gastrointestinal tract and in the oral cavity in patients with the pathology of the digestive system's organs. They contribute to the deepening and

Оригінальні дослідження

generalization of the pathological process in the tissues of the oral cavity, aggravation, progression and complications of the dental diseases.

4. Steady preservation of microflora in certain areas of the digestive tract complicates the course of the dental and gastrointestinal diseases. The gradual change in the properties of representatives of the conditionally pathogenic group of microorganisms, their association with specific diseases of the oral cavity and gastrointestinal tract indicates their role in the pathogenesis of many diseases.

5. An important common link in the pathogenesis of periodontal and gastrointestinal tract diseases are the

mechanisms of free radical damage to cellular structures and changes in humoral and cellular immunity. The development of an imbalance of the endocrine system and psycho-emotional stress has a significant impact on the process of mutual burden of the gastrointestinal tract's and oral cavity's diseases.

So, the approach to the examination and treatment of dental patients with combined diseases of the digestive tract should be complex, with the involvement of gastroenterologists. Understanding the etiopathogenetic mechanisms will contribute to the development of effective measures for the diagnosis, prevention and treatment of both oral cavity and gastrointestinal tract diseases, taking into account the mutual burden of these pathologies.

References

- Hajishengallis G. Interconnection of periodontal disease and comorbidities: evidence, mechanisms, and implications. *Periodontol* 2000. 2022;89(1):9-18. DOI: 10.1111/prd.12430.
- Bawaskar HS, Bawaskar PH. Oral diseases: a global public health challenge. *Lancet*. 2020;395(10219):185-86. DOI: 10.1016/S0140-6736(19)33016-8.
- Bui FQ, Almeida-da-Silva CLC, Huynh B, Trinh A, Liu J, Woodward J, et al. Association between periodontal pathogens and systemic disease. *Biomed J*. 2019;42(1):27-35. DOI: 10.1016/j.bj.2018.12.001.
- Bhuyan R, Bhuyan SK, Mohanty JN, Das S. Periodontitis and Its Inflammatory Changes Linked to Various Systemic Diseases: A Review of Its Underlying Mechanisms. *Biomedicine*. 2022;10(10):2659. DOI: 10.3390/biomedicine10102659.
- Gheorghe D, Camen A, Popescu D, Sincar C, Pitru A, Ionele CM, et al. Periodontitis, Metabolic and Gastrointestinal Tract Diseases: Current Perspectives on Possible Pathogenic Connections. *J Pers Med*. 2022;12(3):341. DOI: 10.3390/jpm12030341.
- Nemesh OM, Shylyvskiy IV, Honta ZM, Moroz KA, Myhal OO. Certain aspects of the systemic etiopathogenesis of dystrophic-inflammatory periodontal diseases (a literature review). *Zaporozhye Medical Journal*. 2024;26(2):154-8. <https://doi.org/10.14739/2310-1210.2024.2.291888>.
- Bogatu SI, Yaremenko II, Liubchenko EA, Levitsky AP. Stan tkanyn porozhnyny rota u khvorykh na hastroduodenit [The state of mouth tissues in patients with gastroduodenitis]. *Visnyk stomatolohii*. 2018;1:28-31. (in Ukrainian).
- Wang Zh, Gong J, Ding Ch. Genetic evidence for the oral-gut axis between periodontitis and inflammatory bowel disease. *J Dent Sci*. 2023;18(4):1904-5. DOI: 10.1016/j.jds.2023.07.017.
- Kozak M, Pawlik A. The Role of the Oral Microbiome in the Development of Diseases. *Int J Mol Sci*. 2023;24(6): 5231. DOI: 10.3390/ijms24065231.
- Bertl K, Burisch J, Pandis N, Bruckmann K, Klinge B, Stavropoulos A. Periodontitis prevalence in patients with ulcerative colitis and Crohn's disease - PPCC: A case-control study. *J Clin Periodontol*. 2022;49(12):1262-74. DOI: 10.1111/jcpe.13615.
- Rosier BT, Marsh PD, Mira A. Resilience of the oral microbiota in health: Mechanisms that prevent dysbiosis. *J Dent Res*. 2018;97(4):371-80. DOI: 10.1177/0022034517742139.
- Lam Gr, Albarrak H, McColl CJ, Pizarro A, Sanaka H, Gomez-Nguyen A, et al. The Oral-Gut Axis: Periodontal Diseases and Gastrointestinal Disorders. *Inflamm Bowel Dis*. 2023;29(7):1153-64. DOI: 10.1093/ibd/izac241.
- Lisetska IS, Rozhko MM. Klinichniy stan ta osoblyvosti mikrobiotsenozu tkanyn parodontu u pidlitkiv z kataral'nym hinhivitom ta khronichnym hastroduodenitom [Clinical status and peculiarities of microbiocenosis of periodontal tissues in adolescents with catarrhal gingivitis and chronic gastroduodenitis]. *Modern pediatrics*. 2018;5:20-5. http://nbuv.gov.ua/UJRN/Sped_2018_5_6. (in Ukrainian).
- Koka VM, Starchenko II, Mustafina HM, Royko NV. Suchasni pohliady na funktsional'nu morfolohiiu slyzovoi obolonky iazyka ta ii zminy v umovakh somatychnykh zakhvoriuvan' ta vplyvu indyvidual'nykh ekzohennykh [Modern views on the functional morphology of the mucous membrane of the tongue and its changes in the conditions of somatic diseases and the influence of individual exogenous]. *Visnyk problem biologii i medycyny*. 2019;3:27-30. DOI: 10.29254/2077-4214-2019-3-152-27-30. (in Ukrainian).
- Poberezhna HM, Kulyhina VM, Hadzhula NH, Povsheniuk AV, Horai MA, Kurdysh LF. Stomatolohichni zakhvoriuvannia u khvorykh z patolohiieiu shlunkovo-kyshkovoho traktu [Dental diseases in patients with pathology of the gastrointestinal tract]. *Visnyk Vinnytskoho natsionalnoho medychnoho universytetu*. 2023;27(2):323-30. DOI: 10.31393/reports-vnmedical-2023-27(2)-25. (in Ukrainian).
- Newman KL, Kamada N. Pathogenic associations between oral and gastrointestinal diseases. *Trends Mol Med*. 2022;28(12):1030-39. DOI: 10.1016/j.molmed.2022.05.006.
- Zahrani MS, Alhassani AA, Zawawi KhH. Clinical manifestations of gastrointestinal diseases in the oral cavity. *The Saudi Dental Journal*. 2021;33(3):835-41. DOI: 10.1016/j.sdentj.2021.09.017.
- Dvornyk VM, Roshchuk OI, Belikov OB, Havaleshko VP. Features of the clinical course of periodontal diseases in patients with gastric and duodenal ulcer with fixed dentures. *World of Medicine and Biology*. 2023;19(83):48. DOI: 10.26724/2079-8334-2023-1-83-48-52.
- Byun SH, Min C, Hong SJ, Choi HG, Koh DH. Analysis of the Relation between Periodontitis and Chronic Gastritis/Peptic Ulcer: A Cross-Sectional Study Using KoGES HEXA Data. *Int J Environ Res Public Health*. 2020;17(12):4387. DOI: 10.3390/ijerph17124387.
- Lisetskaya IS, Rozhko MM, Kutsyk RV. Dynamics of clinical parameters and changes of microbiocenosis of periodontal tissues after complex treatment in adolescents with generalized catarrhal gingivitis and chronic gastroduodenitis. *International Journal of*

Medicine and Medical Research. 2018;4:59-66. DOI: 10.11603/ijmmr.2413-6077.2018.2.9650.

21. Polishchuk TV. Proiavy zakhvoriuvan' shlunkovo-kyshkovoho traktu v rotovii porozhnyni ditei [Manifestations of diseases of the gastrointestinal tract in the oral cavity of children]. *Visnyk problem biologii i medycyny*. 2019;2:55-9. DOI: 10.29254/2077-4214-2019-2-1-150-55-59. (in Ukrainian).

22. Piyush LG, Jigna SS, Shilpa JP, Jayasankar PP. Oral Manifestations in Patients with Gastro-Esophageal Reflux Disease: A Hospital-Based Case-Control Study. *Journal of Indian Academy of Oral Medicine & Radiology*. 2023;35(1):56-60. DOI: 10.4103/jiaomr.jiaomr_116_21.

23. Faraoni JJ, Barone de Andrade J, Machado de Matos LL, Palma-Dibb R. Effect of Duodenogastric Reflux on Dental Enamel. *Oral Health Prev Dent*. 2020;18(1):701-6. DOI: 10.3290/j.ohpd.a45073.

24. Roschuk OI, Havaleshko VP, Khukhlina OS. Osoblyvosti syndromu obtyazhennia vnaslidok komorbidnosti vyrazkovoï khvoroby shlunka, dvanadtsyatypaloi kyshky ta parodontu v osib iz neznimnyimi zubnymi protezamy [Peculiarities of the burdening syndrome due to the comorbidity of peptic ulcer of the stomach, duodenum and periodontal disease in people with fixed dentures]. *Naukovyi visnyk Uzhhorodskoho universytetu*. 2022;1:88-92. <https://dspace.uzhnu.edu.ua/jspui/handle/lib/50236>. (in Ukrainian).

25. Bogatu SI. Poyednana patolohiia: zakhvoriuvannia parodontu ta hastroduodenal'noyi kyshky (ohliad literatury) [Combined pathology: periodontal and gastroduodenal diseases (literature review)]. *Innovatsii v stomatolohii*. 2017;3-4:40-6. (in Ukrainian).

26. Bogatu SI. Patohenychna rol' bakterii helicobacter pylori u rozvytku zapal'nykh zakhvoriuvan' porozhnyni rota [The pathogenetic role of the bacteria helicobacter pylori in the development of inflammatory diseases of the oral cavity]. *Innovatsiï v stomatolohiï*. 2022;1:2-11. DOI: 10.35220/2523-420X/2022.1.1. (in Ukrainian).

27. Wei X, Zhao HQ, Ma Ch, Zhang AB, Feng H, Zhang D, et al. The association between chronic periodontitis and oral Helicobacter pylori: A meta-analysis. *PLoS One*. 2019;14(12):0225247. DOI: 10.1371/journal.pone.0225247.

28. Zhang L, Chen X, Ren B, Zhou X, Cheng L. Helicobacter pylori in the Oral Cavity: Current Evidence and Potential Survival Strategies. *Int J Mol Sci*. 2022;23(21):13646. DOI: 10.3390/ijms232113646.

29. Coulthwaite L, Verran J. Potential pathogenic aspects of denture plaque. *Br J Biomed Sci*. 2007;64(4):180-89. DOI: 10.1080/09674845.2007.11732784.

30. Ryabokon EH, Oleinichuk VV. Prooksydantno-antyoksydantnyi status u khvorykh na parodontyt na tli hastroduodenal'noi patolohii, asotsiovanoi z H. pylori infektsiieiu [Prooxidant-antioxidant status in patients with periodontitis against the background of gastroduodenal pathology associated with H. pylori infection]. *Ukrayinskyi stomatolohschnyi almanach*. 2011;1:167-70. (in Ukrainian).

31. Ansari SA, Iqbal MU, Khan TA, Kazmi SU. Association of oral Helicobacter pylori with gastric complications. *Life Sci*. 2018;205:125-30. DOI: 10.1016/j.lfs.2018.05.026.

32. Liu Y, Li R, Xue X, Xu T, Luo Y, Dong Q, et al. Periodontal disease and Helicobacter pylori infection in oral cavity: A meta-analysis of 2727 participants mainly based on Asian studies. *Clin Oral Investig*. 2020;24(7):2175-88. DOI: 10.1007/s00784-020-03330-4.

33. Hu Z, Zhang Y, Li Z, Yu Y, Kang W, Han Y, et al. Effect of Helicobacter pylori infection on chronic periodontitis by the change of microecology and inflammation. *Oncotarget*. 2016;7(41):66700-712. DOI: 10.18632/oncotarget.11449.

34. Nemesh OM, Honta ZM, Slaba OM, Shylyvskyy IV. Pathogenetic mechanisms of comorbidity of systemic diseases and periodontal pathology. *Wiad Lek*. 2021;74(5):1262-67. <https://doi.org/10.36740/WLek202105140>.

35. Kuzenko EV, Romaniuk AM. Zapal'ni zakhvoriuvannia parodontu: patohenez i morfohenez [Inflammatory periodontal diseases: pathogenesis and morphogenesis]. *Sumy*; 2016. 137 p. (in Ukrainian).

36. Kononova OV. Vzayemozv'iazok rivnia psykholohichnoho stresu ta urazhennia parodontu [Interrelation between the level of psychological stress and periodontal lesion]. *Suchasna stomatolohiia*. 2018;5:35-9. <https://doi.org/10.33295/1992-576X-2018-5-32-36>. (in Ukrainian).

37. Sczepanik FSC, Grossi ML, Casati M, Goldberg M, Glogauer M, Fine N, et al. Periodontitis is an inflammatory disease of oxidative stress: We should treat it that way. *Periodontol 2000*. 2020;84(1):45-68. DOI: 10.1111/prd.12342.

38. Shevchuk MM, Shkrebnyuk RYu, Dyryk VT. Vyznachennia kontsentratsii azotystykh metabolituv u plazmi krovi ta rotovii ridyni u khvorykh iz zakhvoriuvanniamy tkanyh parodonta na tli zahal'nosomatychnykh zakhvoriuvan' [Determination of the concentration of nitrogen metabolites in blood plasma and oral fluid in patients with periodontal tissue diseases against the background of general somatic diseases]. *Ukrainian Journal of Medicine, Biology and Sports*. 2020;5(3):328-36. DOI: 10.26693/jmbs05.03.328. (in Ukrainian).

39. Shevchuk MM, Dyryk VT, Shkrebnyuk RYu. Vyvchennia markeriv imunozapal'noi vidpovidi ta endotelial'noi funktsii tkanyh parodonta pislia likuvannia u patsientiv z CPITN>2 [Study of markers of immune-inflammatory response and endothelial function of periodontal tissues after treatment in patients with CPITN>2]. *Bulletin of problems of biology and medicine*. 2020;3:366-69. DOI: 10.29254/2077-4214-2020-3-157-366-369. (in Ukrainian).

40. Bedeniuk OS, Korda MM. Rol' oksydatyvnoho ta nitrooksydantnoho stresu v patohenezi heneralizovanoho parodontytu na tli khronichnoho hastrytu [The role of oxidative and nitrooxidative stress in the pathogenesis of generalized periodontitis against the background of chronic gastritis]. *Medychna ta klinichna khimiya*. 2016;4:11-15. DOI: 10.11603/mcch.2410-681X.2016.v0.i4.7248. (in Ukrainian).

41. Pupin TI, Honta ZM, Shylyvskyy IV, Nemesh OM, Burda KhB. The role of adaptive-stress response in the pathogenesis of periodontal diseases. *Wiad Lek*. 2022;75(4):1022-25. DOI: 10.36740/WLek20220420119.

42. Zolotukhina OL, Romanova YG. Patohenychni aspekty rozvytku zakhvoryuvan' tkanyh parodonta na tli patolohiï shlunka [Pathogenetic aspects of the development of diseases of periodontal tissue against a background of stomach pathology]. *Visnyk problem biologii ta medytsyny*. 2018;2:23-5. DOI: 10.29254/2077-4214-2018-2-144-23-25. (in Ukrainian).

43. Zabolotnyy TD, Matviychuk KhB, Shamlyan OV. Zahostrennia heneralizovanoho parodontytu iak stresova reaktsiia u khvorykh na vyrazkovu khvorobu dvanadtsyatypaloi kyshky, uskladnenu krovotecheiu [Exacerbation of generalized periodontitis as stress reaction in patients with duodenal peptic ulcer disease complicated by bleeding]. *Visnyk problem biologii ta medytsyny*. 2014;2:35-9. (in Ukrainian).

Оригінальні дослідження

44. Kharchenko AV, Yelinska AM, Shepitko VI, Stetsuk EV. Khronichnyi periodontyt u khvorykh na khronichnu vyrazkovu khvorobu dvanadtsyatypaloi kyshky [Chronic periodontitis in patients with chronic duodenal ulcer]. Svit medytsyny ta biolohiyi. 2022;1:232-36. DOI: 10.26724/2079-8334-2022-1-79-232-236. (in Ukrainian).

Information about authors

Pupin T.I. - Candidate of Medical Sciences, Associate Professor of the Department of Therapeutic Stomatology, Periodontology and Stomatology, Faculty of Postgraduate Education, Danylo Halytsky Lviv National Medical University, Ukraine.

Honta Z.M. - Candidate of Medical Sciences, Associate Professor of the Department of Therapeutic Stomatology, Periodontology and Stomatology, Faculty of Postgraduate Education, Danylo Halytsky Lviv National Medical University, Ukraine.

Nemesh O.M. - Candidate of Medical Sciences, Associate Professor of the Department of Therapeutic Stomatology, Periodontology and Stomatology, Faculty of Postgraduate Education, Danylo Halytsky Lviv National Medical University, Ukraine.

Shylyvskiy I.V. - Candidate of Medical Sciences, Associate Professor of the Department of Therapeutic Stomatology, Periodontology and Stomatology, Faculty of Postgraduate Education, Danylo Halytsky Lviv National Medical University, Ukraine.

Burda Kh.B. - Candidate of Medical Sciences, Associate Professor of the Department of Therapeutic Dentistry, Periodontology and Stomatology, Faculty of Postgraduate Education, Danylo Halytsky Lviv National Medical University, Ukraine.

Maksym O.O. - dentist of the Stomatological Center of Danylo Halytsky Lviv National Medical University, Ukraine.

Відомості про авторів

Пупін Т.І. - канд. мед. наук, доцент кафедри терапевтичної стоматології, пародонтології та стоматології ФПДО, Львівський національний медичний університет імені Данила Галицького, м. Львів, Україна.

Гонта З.М. - канд. мед. наук, доцент кафедри терапевтичної стоматології, пародонтології та стоматології ФПДО, Львівський національний медичний університет імені Данила Галицького, м. Львів, Україна.

Немеш О.М. - канд. мед. наук, доцент каф. терапевтичної стоматології, пародонтології та стоматології ФПДО, Львівський національний медичний університет імені Данила Галицького, м. Львів, Україна.

Шилівський І.В. - канд. мед. наук, доцент кафедри терапевтичної стоматології, пародонтології та стоматології ФПДО, Львівський національний медичний університет імені Данила Галицького, м. Львів, Україна.

Бурда Х.Б. - канд. мед. наук, доцент кафедри терапевтичної стоматології, пародонтології та стоматології ФПДО, Львівський національний медичний університет імені Данила Галицького, м. Львів, Україна.

Максим О.О. - лікар-стоматолог Стоматологічного центру Львівського національного медичного університету імені Данила Галицького, м. Львів, Україна.

Надійшла до редакції 29.03.24

Рецензент – проф. Годованець О.І.

© T.I. Pupin, Z.M. Honta, O.M. Nemesh, I.V. Shylyvskiy, Kh.B. Burda, O.O. Maksym, 2024