

**Kilmukhametova Yu.H.**

*assistant professor of Dental Therapy Department, Higher State Educational Establishment of Ukraine "Bukovinian State Medical University", Avanhardna str, 49/86, Chernivtsi, Ukraine, 58023, kilmukhametova.iuliia@bsmu.edu.ua*

**Batig V.M.**

*Doctoral degree in Medicine, head of the Dental Therapy Department, Higher State Educational Establishment of Ukraine "Bukovinian State Medical University", Chernivtsi, Ukraine*

**Basista A.S.**

*Assistant professor of Dental Therapy Department, Higher State Educational Establishment of Ukraine "Bukovinian State Medical University", Chernivtsi, Ukraine*

## **PERIODONTAL DISEASES ON THE BACKGROUND OF VARIOUS SOMATIC PATHOLOGIES (LITERATURE REVIEW)**

**Abstract** *This article presents the results of a review of the literature on periodontal diseases that arise against the background of concomitant somatic pathologies, their relationship and causes.*

**Key words:** *periodontal diseases, somatic diseases, cardiovascular pathology, pathology of the gastrointestinal tract, pathology of the endocrine system, kidney pathology, urolithiasis.*

**Introduction.** Periodontal diseases were and remain one of the most common and actual dental diseases. Thus, more than 60% of the population aged 40 suffer from this or that periodontal disease, in older people this figure increases to 100% [13]. Pathological changes in the periodontium often occur against the background of concomitant diseases, digestive disorders, metabolism, cardiovascular and endocrine diseases, hypersensitivity and infection of the body. In addition, one of the important causes of periodontal disease is pathogenic microflora [Bauermeister C., 2003].

Conditionally, all etiological factors can be divided into local and general. Among the local etiological factors that exert mechanical, chemical and biological (microbes, toxins) effects, dental deposits are of particular importance [7]. From non-mineralized dental deposits, dental plaque and soft dental plaque are of paramount importance for the onset of periodontal lesions. Among the common factors leading to the occurrence of periodontal pathology, the influence of general somatic diseases, which in 85.0% of cases are concomitant and activating the pathological process in periodontium, is unquestionable [4, 19].

Among the examined patients with rheumatism, chronic tonsillitis was observed in 52.6% of cases, complicated caries in 42.8% of patients. Quite often there are diseases of the

digestive tract, kidneys, eyes [21]. They also include eczema, neuritis, neuralgia, blood diseases. Among the general factors, great importance is attached to neurotrophic disorders, disturbances in the state of the periodontal vessels and the organism as a whole, metabolic disorders [15, 20].

With pulmonary tuberculosis, multiple tooth caries, specific stomatitis, periodontal lesions are observed. When examining 916 patients with tuberculosis, periodontal disease was detected in 80.7% (in healthy people this figure was 57%), and tuberculosis infection is more often (82%) accompanied by inflammatory changes in the gums [Gonta Z.M., 2009].

When examining children with primary forms of tuberculosis, it was determined that the incidence of gingivitis in children with a primary tuberculosis complex was 93.93%, taking into account symptomatic gingivitis in periodontitis; in children with tuberculous bronchoadenitis, the incidence of inflammatory gum disease was 83.70%; against the background of tuberculosis intoxication, these indicators were in 87.90% of children [Stadnik U., 2014]. When studying the features of the clinical course of periodontal diseases in children against the background of primary tuberculosis, a high prevalence of periodontal tissue pathology was found (68.8%), which depends on the child's age, clinical form and duration of tuberculosis. Catarrhal gingivitis

occurs in 59.3% of children, and periodontitis in 7.8-1.1%, with age, the prevalence of chronic forms increases [15]. The majority of children with tuberculosis have a poor state of oral hygiene (75.04%), with children not regularly cleaning their mouths or not brushing their teeth at all.

Periodontal disease in the pathology of the gastrointestinal tract occurs in 68-90% of the patients examined [4]. Analyzing the relationship between periodontal diseases and the gastrointestinal tract, most authors found that the pathology of the digestive system more often precedes the appearance of periodontal diseases [Emelyanova N.Yu., 2008].

Both in patients with generalized periodontitis and tonsillar pathology, and in patients without tonsilopathy, symptomatic catarrhal gingivitis was observed (depending on the stage of generalized periodontitis, acute or chronic). Patients with generalized periodontitis were more likely to have gingivitis of grade II severity, while in patients with generalized periodontitis and chronic tonsillitis, catarrhal gingivitis of grade III severity was more often detected [2].

In individuals with periodontal disease, hypothyroidism is associated with a decrease in overall immune resistance. According to the authors, this is due to the appearance of thyroid hormone deficiency in hypothyroidism, which leads to significant disturbances in metabolic processes in the body and a decrease in nonspecific resistance, which in turn contributes to the negative effect of oral microflora on periodontal tissue and leads to the development of inflammatory and inflammatory-dystrophic changes in it [Chornij AV, 2016].

Clinical dental examination of patients with gastroesophageal reflux disease showed that changes in periodontal tissues are noted in 84% of cases. At the same time, chronic catarrhal gingivitis was recorded in 67% of cases, and chronic generalized periodontitis of I-II degrees of severity was recorded in 23%. [6, 11].

The studies show that in patients with duodenal ulcer, the frequency of periodontal tissue lesions ranges from 84.6 to 97.3%. Periodontal disease occurs in 92% of patients with gastric ulcer, of which in 15.4% of cases it is catarrhal gingivitis, and in 76% generalized periodontitis [Matviychuk Kh.B., 2010]. Expressed

inflammatory phenomena in the tissues of periodont are also manifested with relapse of gastroduodenal ulcers [14].

It was found that in patients with chronic pancreatitis, the pathology of periodontal tissue is noted in 90% of cases [Kolesova N., 2008]. When examining the oral cavity of patients with intestinal dysbiosis, poor hygiene of the oral cavity and the presence of hard and soft dental deposits were observed in most cases, which is a prerequisite for the onset of inflammation in the periodontium [19].

In patients with liver and biliary tract diseases, 100% of periodontal lesions are noted [1]. Periodontal disease was found in 97.4% of patients with ulcerative colitis [8, 16].

In patients with chronic periodontitis, combined with chronic pathology of the digestive system, the clinical picture is characterized by complaints of soreness of the gums, their bleeding and bad breath. A local examination revealed a chronic inflammatory process of periodontal tissues, tooth mobility and, in some cases, purulence from periodontal pockets [2].

In patients with a combined course of chronic cholecystitis and pancreatitis, the prevalence of catarrhal gingivitis is 5.6%, and generalized periodontitis is 94.4%. The most common periodontitis is I and II severity, the development of which depends on the duration of somatic pathology. Generalized periodontitis in patients with a combined course of chronic cholecystitis and pancreatitis in 89.1% of cases is characterized by chronic course and is accompanied by a 2.4-fold deterioration in the state of oral hygiene [12].

Most often, patients with diabetes mellitus are found to have gingivitis and periodontitis [4, 17, 18]. According to A.A. Alekseeva, the diseases of periodontal tissues are found in 98% of patients with insulin-dependent diabetes mellitus. Among the adult population, the incidence of generalized periodontitis is 6 times higher than the incidence of gingivitis. Clinical examination of patients with diabetes mellitus revealed pathological changes of periodontal tissue with prevalence of gingivitis (46.6%) in 67% of cases. In 33% of patients with diabetes, the periodontium was intact [5, 9].

In acute nephritis with a short-term course, which in most cases ends in recovery, changes in periodontium are observed in the form of

gingivitis of mild and moderate severity, the serum calcium content is normal, and chronic nephritis shows significant changes in the periodontal tissues found in direct dependence on the duration and severity of the underlying disease [19].

Most patients with chronic renal failure complain of bad breath, a metallic taste; these symptoms are associated with an increase in the level of urea in the saliva, which, when ingested, splits with the release of ammonia. A number of authors [3] detected changes in taste sensitivity in patients with chronic renal failure. In these patients, periodontal lesions are associated with an elevated concentration of blood creatinine. In addition, an increase in the level of IgG in the blood, which is observed with generalized periodontitis in patients undergoing dialysis therapy, causes an increase in the synthesis of C-reactive protein (CRP). In patients with generalized periodontitis and CRF, disorders of calcium-phosphorus metabolism were detected, and as a result, bone density decrease, osteoporosis, affecting the mobility of teeth, recession of the gums and the presence of periodontal pockets.

Among patients with urolithiasis, the highest prevalence of periodontal disease was found, 94.59%, and in 79.85% of patients without concomitant pathology. Most commonly, people with urolithiasis are diagnosed with generalized periodontitis (62.14%) and chronic catarrhal gingivitis (16.60%). On the background of urolithiasis, periodontal lesions occur mainly in the form of gingivitis or marginal periodontitis. There is hyperemia and swelling of the gums, their bleeding. Attention is drawn to a significant amount of deposits on the surfaces of the teeth. This is due to the irrational hygiene of the oral cavity [17].

The more severe course of periodontitis in persons with chronic glomerulonephritis and chronic pyelonephritis was found, in comparison with healthy people, pronounced resorption of interalveolar septa is a manifestation of generalized nephrotic osteodystrophy. There is less pronounced hyperemia and bleeding gums, absence of purulence from the periodontal pockets, which is a consequence of the use of effective anti-inflammatory drugs in the

treatment of kidney diseases against the background of a decrease in overall resistance. At the same time, for this contingent of patients, progressive resorption of interalveolar septa, exposure of the necks of teeth, hyperesthesia of hard tissues, and the presence of wedge-shaped defects are characteristic [2]. A review of the literature data makes it clear that the pathology of the urinary system accompanied by metabolic, microcirculatory, immunity, etc. disturbances, lead to damages of periodontal tissues, because it is proved that the severity of periodontal diseases is directly related to the duration of the pathological process in the kidneys.

Periodontal disease is a polyetiological disease, the pathogenesis of which is associated with pathological processes in the body caused by disorders in the functioning of the most important body systems.

#### References:

1. Bandrivskiy YuL, Bandrivska NN, Avdieiev OV. *Vzaiemoz'iazok zakhvoriuvan parodontu iz somatychnoiu patolohiieiu [The connection between periodontal disease and major systemic diseases]. Galician Medical Journal. 2008;4:95-6 [in Ukrainian].*
2. Barylo OS, Skliaruk NV, Tsaryk N.P. *The features of condition of periodontium and oral hygiene in patients with chronic parodontitis on a background of chronic tonsillitis. Reports of Vinnytsia national medical university. 2014; 2(18):607-11.*
3. Al-Zahrani MS, Kayal RA, Bissada NF. *Periodontitis and cardiovascular disease: a review of shared risk factors and new findings supporting a causality hypothesis. QuintInt. 2006; 37(1):11-8.*
4. Vasilevskaya EM, Blashkova SL. *Pathogenetic aspects of the formation of periodontal disease in patients with coronary heart disease. Praktichna medicina. 2013; 7(73):154-6.*
5. Godovanets OI, Moroz AV. *Dental pathology in children with endocrine diseases. Clinical and experimental pathology. 2015;54(4):209-13.*
6. Goncharuk LV, Kosenko KN, Goncharuk SF. *Relationship of inflammatory periodontal diseases and somatic pathology. Sovremennaja stomatologija. 2011;1:37-40.*
7. Zabolotnyi TD, Borysenko AV, Pupin TI. *Zapalni zakhvoriuvannia parodontu: monohrafiia dlia studentiv vyshchyykh navchalnykh zakladiv,*

interniv, likariv-stomatolohiv, simeinyk hlikariv. Lviv: Gal Dent; 2013.

8. Kashivska RS, Rozhko MM. The changes in total protein level in blood serum and oral fluid of the patients during the treatment of generalized periodontitis combined with chronic liver diseases. *Ukrainian dental almanac*. 2015;5:14-7.

9. Kuzniak NB, Godovanets OI, Goncharenko VA. Stomatological morbidity in children with endocrine pathology. *Clinical and experimental pathology*. 2013; 2(44):100-2.

10. Kulyhina VM, Levytska OV. Rezultaty obstezhennia klinichnohos tanu ta hiiieny rotovoi porozhnyny khvorykh na dysbakterioz kyshkivnyka [Results of examination of the clinical condition and hygiene of the oral cavity of patients with intestinal dysbiosis]. *Proceedings of the III(X) annual meeting of Ukrainian dental association "Innovative technologies for dental practice"*; 2008 Oct 16-18; Poltava. Poltava: "Divosvit"; 2008. p.186-7 [inUkrainian].

11. Manashchuk NV, Chorniy NV, Shmanko VV. Interconnection between periodontium pathology and pathology of gastrointestinal tract. *Clinical Dentistry*. 2011;(1-2):23-7.

12. Moshel TM. Mikrobiolohichne obgruntuvannia zastosuvannia novoho sposobu likuvannia heneralizovanoho parodontytu u khvorykh z khronichnymy kholetsystpankreatytamy [The microbiological ground of application of new treatment method of marginal periodontitis in patients on a background of chronic cholecystopancreatitis]. *World of medicine and biology*. 2008;2(1):82-5 [inUkrainian].

13. Prodanchuk AI, Kiiun ID, Kroitor MO. Periodontopathy and somatic pathology. *Bukovinian medical herald*. 2012; Vol.16, 2(62):164-8.

14. Samoilenko AV. Modern aspects of etiology, pathogenesis and treatment of different clinical variants of generalized periodontitis. – Manuscript. Thesis for a doctor's degree by speciality 14.01.22 – stomatology. Institute of

Stomatology of AMS of Ukraine, Odesa, 2003.

15. Smoliar NI, Stadnyk UO. Osoblyvosti klinichnoho perebihu khvorob parodonta na foni pervynnoho tuberkulozu [Clinical manifestation of the periodontal diseases on a background of tuberculosis]. *Visnyk stomatologiy*. 2000;(2):36-38. [inUkrainian]

16. Tomashevskiyi Yal, Andrushko VT, Bumbar OI, Tomashevskya NYa, Bumbar ZO, Milkosh LYa. Study of bioethical aspects of genetic susceptibility of people to diabetes and research optimization for methods to prevent inefficiency disorders (based on the strategy of sustainable development). *Scientific bulletin of UNFU*. 2013;23(11):360-4.

17. Homenko LO, Gavrilenko TI, Ostapko OI, Moskovenko OD, Duda OV. Peculiarities of cytokine status in children with chronic catarrhal gingivitis accompanying with somatic pathology. *Bulletin of problems in biology and medicine*. 2013;4(1): 352-6.

18. Khomenko LO, Marushko YuV, Moskovenko OD, Duda OV. Relationship of inflammatory periodontal tissues and somatic diseases in children. *Literature review*. *Novyny stomatolohii*. 2015; 83(2):90-4.

19. Shylivskyi IV, Nemesh OM, Vaskiv NB. Osoblyvosti klinichnoho perebihu heneralizovanoho parodontytu u khvorykh na sechokam'ianu khvorobu [Clinical features of generalized eriodontitis in patients with urolithiasis]. *Praktichna medicina*. 2006;12(2):106-8 [inUkrainian].

20. Shylivskiyi IV, Nemesh OM, Honta ZM. Modern concepts of etiology and pathogenesis of inflammatory periodontal diseases, the irrelationship with pathology of urinary system (review of the references and author's own researches). *Buk Med Herald*. 2016; 20(1):224-7

21. Dahshan A, Patel H, Delaney J, Wuerth A, Thomas R, Tolia V. Gastroesophageal reflux disease and dental erosion in children. *J Pediatr*. 2002 Apr;140(4):474-8.