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IMPROVING THE SECONDARY PREVENTION OF PERIODONTITIS CAUSED BY PROSTHETICS IN MILITARY PERSONNEL

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ABSTRACT

This article cited as the results of the study the cytomorphological effectiveness, which reduced the cytomorphological indicators to the norm, which ensured the elimination of cytopathological signs, in addition to the antibacterial, detoxification, regenerator and anti-inflammatory effects under the influence of improved complex treatment in patients with post-prosthetic developmental acute periodontitis.

Risk factors, as defined by the World Health Organization (WHO), are "any property or feature of a person, or any effect on him that increases the likelihood of developing an illness or injury." From the point of view of developing measures for the primary and secondary prevention of certain diseases, including such socially significant ones as cardiovascular diseases and diabetes mellitus, which form over the years and are difficult to diagnose at their onset, it is the identification of risk factors for development and progression that is the most important task of modern medicine. Risk factors, being the etiological factor of the disease, are divided into modifiable (correctable), which require primary and secondary prevention, and unmodifiable (uncorrectable), which cannot be affected. Back in 2008, the American Academy of Periodontology formulated the assessment of the risk of periodontal tissue diseases as a process of qualitative or quantitative analysis of adverse factors contributing to the formation of this pathology [1].

M. Timmerman and G. van der Weijden note the biological relationship of risk factors with the onset of the disease, as well as the fact that if a patient has a risk factor, periodontal tissue disease may develop [2]. However, according to K. Gurevich and E. Fabrikant, periodontal diseases can develop even in the absence of risk factors [3]. A direct and significant relationship between risk factors and the development of periodontal pathology is also presented in other scientific studies [4-7]. The presented review article discusses the role and mechanisms of action of correctable risk factors for

the development and progression of periodontal tissue diseases (STDs). STDs with caries are the most serious threats to dental health and the main etiological factors of tooth loss. STD is traditionally defined as an inflammatory disease affecting both the soft and hard structures of the periodontium [8], which is also characterized by loss of the periodontal ligament and destruction of the surrounding alveolar bone [9].

STDs are inflammatory diseases of the oral cavity that can be limited only by the gum, as in gingivitis, or extend beyond the gingival tissue, leading to the loss of soft and hard structures, which affects the stability of teeth attachment to the alveolar bone, as it occurs in periodontitis [10].

Gingivitis is the most common form of periodontitis and is found all over the world. According to J. Albandar and T. Rams, its prevalence and severity are less pronounced in industrialized, developed countries compared to less developed regions [11]. J. Stamm notes that gingivitis is less common in children, more common in adolescents and young adults, and tends to level off in adulthood [12]. The presence of supragingival biofilm is closely related to the presence of gingivitis.

There are several modifiers of gingivitis severity: overgrowth-related medications such as cyclosporine, nifedipine, or dilantin; medical conditions such as diabetes and HIV infection; behavioral factors such as smoking, and conditions such as pregnancy or menopause. Until recently, the prevailing model of the pathogenesis of periodontal diseases indicated that gingivitis does not always develop into periodontitis [11]. However, according to M. Schatzle et al., there are indications that the prolonged presence of gingivitis is associated with increased attachment loss [4]. D Kinane and R. Attstom believe that gingivitis and periodontitis are actually links in the continuum of the same disease [13].

The early phase of the inflammatory process in periodontal tissues, conventionally defined as gingivitis, is characterized by moderate and self-terminating inflammation. If local inflammation progresses, the disease develops towards periodontitis, which back in 1999 was defined as microbially associated and host-mediated inflammation leading to loss of periodontal attachment [4]. At the subsequent 2017 World Seminar According to the classification of periodontal and peri-implantation diseases and conditions, it was stated that the diagnosis of periodontitis should be based on a clinical loss of attachment by peripheral detection using standardized periodontal probes of the erupted dentition with a reference to the cement-enamel joint[3]. M. Tonetti et al. It is emphasized that if periodontal diseases develop progressively and if left untreated, local complications form, such as the development of deep periodontal lesions, periodontal loss of bones and teeth, up to the diagnosis of chewing failure [4].

Recently, it has been hypothesized that some bacteria that cause periodontitis can actively enter the bloodstream from periodontal tissues and be located in many organs and tissues, thereby increasing the risk of developing pathologies characterized by an inflammatory and infectious component [8].

These usually include various types of malignancies (especially cancers of the digestive tract, pancreas, prostate, breast, uterus, lungs, esophagus and oropharynx, as well as lymphomas) [9], cardiovascular diseases [2], venous thromboembolism [1],

diabetes mellitus [2], rheumatic diseases [3], Alzheimer's disease, and dementia [], which together represent the most common pathologies worldwide [8].

The presented review article presents modern ideas about the correctable risk factors for periodontal tissue diseases, which is of interest not only from the point of view of a dentist, but also from a general medical point of view. The search for sources for the review article was carried out in the MEDLINE and PubMed databases using medical thematic headings, as well as the following terms in various combinations: "periodontal tissue diseases", "modifiable risk factors for periodontal tissue diseases", "periodontogenic microorganisms", "tobacco smoking", "diabetes mellitus", "cardiovascular diseases", "drug-induced periodontal tissue damage." The sources for the review article were supplemented by manual search in peer-reviewed journals and cross-references to the articles that were accessed.

Modifiable risk factors 1. Microorganisms that cause periodontal tissue diseases. Research data from G. Blandino et al. It is emphasized that mixed microbial pathogenic infections, in which certain groups of bacteria coexist, are etiological factors in the development of periodontal disease [9]. The bacterial puzzle in the oral cavity includes more than seven hundred different phylotypes, of which about four hundred species were found during the analysis of subgingival plaque [3].

According to A. Haffajee et al., hundreds of bacterial species are found in the subgingival microflora in periodontitis, and it is important that only a small number of them are associated with the progression of the pathological condition of periodontitis. The authors mainly isolated gram-negative anaerobic rods and spirochetes from the deepened periodontal pockets of the subgingival plaque [1]. Porphyromonas gingivalis [3] and Aggregatibacter actinomycetemcomitans [3] play an important role in the pathogenesis of adult periodontitis. While with the progression of periodontal tissue diseases, special importance is attached to Bacteroides forsythus, Prevotella intermedia [3], Peptostreptococcus micros and Fusobacterium nucleatum [6].

2. Smoking tobacco. Smoking is an important factor in the development of periodontal tissue diseases. Currently, a large amount of data has been accumulated on a higher level of periodontal diseases among smokers [7]. A. Zini et al. When conducting a cross-sectional study of two hundred and fifty-four adults aged 35-44 years in Jerusalem (limited to the Jewish population) using a stratified sample, as well as studying the community periodontal index, plaque index, and a self-administered questionnaire, it was found that a lower level of education was associated with severe chronic periodontitis ($p = 0.012$), as well as with smoking ($p = 0.030$) and higher plaque levels ($p < 0.001$).

Smoking was associated with a higher plaque level ($p < 0.001$), which, in turn, was associated with the presence of severe chronic periodontitis ($p = 0.020$), i.e. smoking had a significant destructive effect on periodontal tissues and increased the rate of progression of periodontal diseases [8]. Risk factors, including tobacco smoking, modify the host's response to exposure to bacteria in microbial plaque [9].

However, according to J. Bergstrom, people with tobacco smoking and periodontal tissue diseases showed fewer signs of periodontitis and bleeding gums compared with non-smoking patients [5]. This fact, according to J. Jensen et al., can be explained by the

fact that nicotine has a local vasoconstrictive effect, reducing blood flow and edema, as well as other clinical signs of inflammation [4]. In addition, Q. Wang et al. It has been found that the nicotine-acetylcholine receptor is of crucial importance in the formation of nicotine-related periodontitis [2].

3. Сахарный диабет. К важнейшим корригируемым факторам риска развития и прогрессирования ЗТП относятся нарушения углеводного обмена, в том числе и сахарный диабет. Пациенты с недиагностированным или плохо контролируемым сахарным диабетом 1-го или 2-го типа подвержены более высокому риску ЗТП.

The association between diabetes mellitus and increased susceptibility to oral infections, including periodontal diseases, has been demonstrated in numerous studies [4]. M. Seppalla et al. It is noted that periodontitis also progresses faster in poorly controlled diabetics [5], including in insulin-dependent diabetes mellitus [4], and H. Torstensson and A. Hugoson emphasize that the early age of onset of the disease is considered as a risk factor for more severe periodontal diseases [6]. And vice versa, according to J. Pucher and J. According to Srewart, most patients with well-controlled diabetes can maintain periodontal health and respond positively to periodontal therapy [7]. S. Grossi and R. Genco believe that despite the controversy on this issue in the scientific literature, it is an indisputable fact that the effect of glycemic control is closely related to the periodontal therapy regimen[8].

This fact is confirmed by numerous studies on the effect of periodontal treatment on glycemic control in diabetes mellitus [8]. According to C. Negrato et al., further prospective rigorous controlled studies with a large number of patients in ethnically diverse populations are needed to establish these relationships and that treatment of periodontal tissue diseases can positively affect glycemic control and possibly reduce the burden of complications associated with diabetes mellitus [4].

4. Cardiovascular diseases. The correctable risk factors for the development and progression of STD also include diseases of the cardiovascular system. Several biological mechanisms have been proposed to explain the relationship between periodontal diseases and cardiovascular diseases. It has been suggested that periodontal tissue diseases play an etiological or regulatory role in cardiovascular and cerebrovascular diseases, and lesions of the oral cavity are indicators of the progression of cardiovascular pathology, and the oral cavity can be a window determining the state of organs and body systems, as well as general health.

Periodontal diseases can predispose to vascular pathology under the influence of the subgingival microflora and the response of the macroorganism. It should be noted that vascular diseases and STD have many common risk factors, as well as similar mechanisms of pathogenesis [5].

Epidemiological and microbiological studies by J. Stein et al. confirmed the concept that the presence of STD can be considered a separate risk factor for cardiovascular and cerebrovascular pathology [1], as well as the development of premature birth in children with low birth weight [3]. According to T. Wu et al., STD is an independent risk factor for cerebrovascular diseases, especially ischemic stroke [4]. However, according to R. Garcia,

as well as M. Trevisan and J. Dorn, a link between periodontitis and cardiovascular pathology has not been found [5].

One of the mechanisms explaining the role of CTP in the development of cardiovascular diseases is the ability of the inflammatory phenomenon of periodontitis to influence the systemic distribution of locally produced mediators such as C-reactive proteins (CRP), interleukin-1b (1L-1b) and interleukin-6 (1L-6) and TNF-α [6]. Another indirect effect of periodontal infection, which may explain the link between STD and cardiovascular diseases, is that periodontal organisms contain proteins that cross-react with the heart. So, heat shock protein-60 (eng. HSP, heat shock protein), which is produced by Tannerella forsythia and Porphyromonas gingivalis, has approximately 60% homology with mammalian HSP.

It is known that antibodies to the heat shock protein are found in patients with periodontal disease. These antibodies to HSP of periodontal bacteria cross-react with HSP, which are found in damaged endothelium or atheromatous plaque. This can set in motion autoimmune phenomena and contribute to the formation of atheroma. There may also be common genetic mechanisms that provide a link between periodontal diseases and cardiovascular diseases [7].

References:

1. American Academy of Periodontology. American Academy of Periodontology statement on risk assessment. J Periodontol. 2008; 79(2): 202. doi: 10.1902/jop.2008.082001
2. Timmerman MF, van der Weijden GA. Risk factors for periodontitis. Int J Dent Hyg. 2006; 4(1): 2-7. doi: 10.1111/j.1601- 5037.2006.00168.x
3. Гуревич К.Г., Фабрикант Е.Г. Укрепление здоровья. М.: Профессионал; 2010.
4. Колесникова Л.Р. Артериальная гипертензия и стоматологическое здоровье у детей и подростков (обзор литературы). Acta biomedica scientifica. 2015; (3): 94-99.
5. Al-Taweel FB, Abdulkareem AA, Abdulbaqi HR. Association of modifiable and non-modifiable risk factors with periodontal Acta Biomedica Scientifica, 2022, Vol. 7, N 5-2 188 Dentistry Стоматология disease in Iraqi individuals: A retrospective study. J Stoma. 2019; 72(4): 222-227. doi: 10.5114/jos.2019.93298
6. Kim YT, Choi JK, Kim DH, Jeong SN, Lee JH. Association between health status and tooth loss in Korean adults: Longitudinal results from the National Health Insurance Service-Health Examinee Cohort 2002-2015. J Periodontal Implant Sci. 2019; 49(3): 158-170. doi: 10.5051/jpis.2019.49.3.158
7. Wellapuli N, Ekanayake L. Risk factors for chronic periodontitis in Sri Lankan adults: A population based case-control study. BMC Res Notes. 2017; 10(1): 460. doi: 10.1186/s13104-017-2778-3
8. Chapple ILC, Mealey BL, Van Dyke TE, Bartold PM, Dommisch H, Eickholz P, et al. Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol. 2018; 89(1): 74-84. doi: 10.1002/JPER.17-0719

9. Cui X, Monacelli E, Killeen AC, Samson K, Reinhardt RA. Impact of modifiable risk factors on bone loss during periodontal maintenance. Oper Dent. 2019; 44(3): 254-261. doi: 10.2341/18- 041-C
10. CDC. Periodontal disease. 2015. URL: https://www.cdc.gov/oralhealth/periodontal_disease [date of access: 26.01.2022].
11. Albandar JM, Rams TE. Global epidemiology of periodontal diseases: An overview. Periodontol 2000. 2002; 29: 7-10. doi: 10.1034/j.1600-0757.2002.290101.x
12. Stamm JW. Epidemiology of gingivitis. J Clin Periodontol. 1986; 13(5): 360-366. doi: 10.1111/j.1600-051x.1986.tb01473.x
13. Lindhe J, Okamoto H, Yoneyama T, Haffajee A, Socransky SS. Longitudinal changes in periodontal disease in untreated subjects. J Clin Periodontol. 1989; 16(10): 662-670. doi: 10.1111/j.1600-051x.1989.tb01037.x
14. Schätzle M, Löe H, Bürgin W, Anerud A, Boysen H, Lang NP. Clinical course of chronic periodontitis. I. Role of gingivitis. J Clin Periodontol. 2003; 30(10): 887-901. doi: 10.1034/j.1600- 051x.2003.00414.x