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THE CONFIGURATION OF TISSUE ALLERGY TO **BONE ANTIGEN AND ITS ROLE IN THE DIAGNOSIS OF THE INITIAL DEGREE OF GENERALIZED** PERIODONTITIS WITH A DIFFERENT COURSE OF THE PATHOLOGICAL PROCESS IN THE PERIODONTAL COMPLEX

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ABSTRACT

ed: 20 May 2018 ed: 15 June 2018 ed: 12 July 2018	Abstract. Diseases of periodontal tissues occupy a leading place in the structure of dental diseases. Early diagnosis of the initial degree of generalized periodontitis (GP) is an effective way of secondary				
DRDS zed parodontitis; ensibilization; ntal complex; tigen.	prevention. The detection of tissue sensitization to bone antigen can be an adequate specific reaction for early diagnosis of GP. Aim: to study tissue sensitization to bone antigen as an indicative factor of changes in the alveolar process at the initial degree of GP. Materials and methods: the study is based on the observation of 132 patients with an initial degree of GP of chronic and exacerbated flow. The comparison group consisted of 71 patients, with generalized chronic catarrhal gingivitis (GCCG). Results: a marked degree of tissue sensitization to the bone antigen was observed in $61.0 \pm 5.72\%$ of patients (p<0.01) and in $80.0 \pm 5.13\%$ (p<0.01) patients with exacerbated flow of GP. Conclusions: The expressed degree of tissue sensitization to bone antigen in patients with GP of the initial degree can be considered as an indicative indicator in differential diagnosis of GP of the initial degree and catarrhal gingivitis.				

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Diseases of periodontal tissues, including generalized periodontitis (GP), consistently occupy one of the leading places in the structure of dental diseases [1]. Thus, according to recent epidemiological studies, the prevalence of GP is 80-100% with a persistent tendency to increase the

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frequency of GP in young and employable people with gender and population preferences [2, 3]. This circumstance causes serious concern of state, social, medical and scientific institutions [4]. The nonesthetic state of the soft tissues of the periodontal complex, the disruption of the functional capacity of the masticatory device due to early tooth loss, the duration of treatment usually with unstable results of treatment with short periods of remission, and significant material and time costs, have a profound effect on the psycho-emotional state of patients, distract them from active labor and personal life, constructing a deterioration in the patient's quality of life [5, 6].

Often, the low effectiveness of the results of GP treatment is explaining due to the lack of clear ideas about the cause-effect relationships in its pathogenesis. Despite the diversity of ideas about the etiology and pathogenesis of GP, most researchers agree that the qualitative and quantitative changes in the oral microbiome is the key paradigm of the antigenic microbial attack on periodontal tissue [7, 8]. Prolonged expansion under the influence of changes in the microbiome activates osteoclastic processes, disrupting the remodeling balance in favor of the prevalence of osteosorption over osteosynthesis [9]. The altered microbial landscape potentially provokes not only a high degree of microbial sensitization and endogenous intoxication of the body, but is also a key trigger for the change in the adequate, programmed response of the immune system to the antigenic load in the direction of the configuration of the new format of the immune response. This leads to the construction of an autoimmune component in the pathogenesis of GP [10]. Once started, the autoimmune reaction acquires the ability to self-support. This circumstance can influence not only the nature of the course and the result of treatment of GP, but also serve as a provocative factor for initiating a number of diseases of internal organs and systems, including such as rheumatism, rheumatoid arthritis, kidney disease, eye, etc. [11]. On the other hand, researchers reveal that malabsorption in diseases of the digestive tract, kidney disease, diabetes I and II type, often affiliated pathogenetically with GP, cause congruent interaction, determining the nature of the course of the disease, make it difficult to achieve a stable result of their treatment.

An analysis of information of scientific research suggests that the key factor which leads to the lack of a stable and predictable result of GP treatment is not so much due to the lack of clear ideas about the etiology and pathogenesis of GP but the main factor focuses on clinical and laboratory features of the course and treatment GP in patients with advanced disease, from initial to III degree (maximum) [12, 13]. At the same time, without detracting from the importance of such studies, it is important to note that the scientific works do not focus enough attention on the features of early diagnosis of GP. Simultaneously, we believe that only early diagnosis, as well as the choice of adequate treatment at the stage of premorbid state and initial degree of GP, can be an effective way of secondary prevention, which allows to prevent, delay or delay complex surgical, contradictory orthopedic interventions and, in ultimately, prevent or significantly slow down the process leading to early tooth loss.

It should be noted that clinical, radiologic, as well as laboratory diagnostics of advanced degrees of GP is not a problem. At the same time, the diagnosis of GP at the initial degree presents certain difficulties. Thus, the absence of clear markers which identify initial changes in the key moment of initiating the debut of the pathological process in GP, makes it difficult to diagnose and, as a result, to conduct opportune in full and adequate treatment. This circumstance often leads to the fact that the initial degree of GP is accepted and identified with different forms of gingivitis. As a result, the current treatment is directed to stopping, first of all, the inflammation process in the periodontal tissues in order to reduce the activity of osteoclasts without the inclusion of funds that normalize the metabolism of the bone tissue of the alveolar process.

Standard indicative criteria for the condition of the alveolar process, for example, the level of calcium, copper, strontium in blood plasma, bone-specific alkaline phosphatase, cholesterol, triglycerides of blood serum, oxyproline plasma, bone mineral density are quite burdensome for patients and are nonspecific indicators under impact of many components of the body, which makes it difficult to use, complicates the interpretation of the facts. In our opinion, the determination of tissue sensitization to bone antigen can be that adequate specific reaction that could help for early diagnosis of GP.

In this regard, **the purpose** of this research was to study tissue sensitization to bone antigen as an indicative factor of changes in the alveolar process at the initial degree of GP.

Proceeding from this, we set the following **tasks**:

1. To establish the level of tissue sensitization to bone antigen in patients with GP of the initial degree, chronic and exacerbated flow.

2. To determine the level of tissue sensitization to bone antigen in patients with generalized chronic catarrhal gingivitis in remission and exacerbation.

3. To conduct a comparative analysis of the revealed tissue sensitization indexes to the bone antigen in patients with GP of the initial degree and GCCG with a different pathological process.

Material and methods. Clinical, immunological and statistical methods were used to solve the tasks.

Under our supervision, there were 132 patients (67 women and 65 men) from 19 to 35 years old with an initial degree of GP (main group) with a chronic (72 people) and exacerbated (60 people) course.

The comparative group was consisted of 71 patients from 19 to 35 years old, equal in sex, with generalized chronic catarrhal gingivitis (GCCG) in the stage of remission (29 people) and exacerbation (42 people).

The control group consisted of 30 people similar to the age and sex without clinical signs of periodontal disease. Diseases of internal organs and systems, including the osteoarticular apparatus, in these examined people were excluded.

Interpretation of clinical indicators was assessed on the basis of subjective and objective symptoms using periodontal indices (PMA, Loe-Silness, GI, integral index of hygiene ART). Diagnosis of periodontal tissue disease was carried out in accordance with the classification of N.F. Danilevsky (1994).

Evaluation of tissue sensitization to bone antigen was determined in the inhibition of leukocyte migration (RIML). In RIML reaction, water-salt extract of bone tissue of group 0 (I) Rh (D) was used. The migration index was calculated by the formula:

$IM = \frac{migration area with antigen}{migration area without antigen},$

where IM, equal to 0.1-0.5, was corresponded to a high degree of sensitization. The reaction was taken 24 hours after blood collection.

The use of RIML was due to its high specificity and informativeness. It is included in the list of reactions recommended by WHO. Taking into account that the reaction is carried out outside the body (in vitro), conditions are created for multiple examination of the patient for diagnosis and at the stages of treatment.

Statistical processing of the results was carried out with parametric statistics methods using the standard program Statistica 6.0.

Results and its discussion. As a result of the research, we did not establish a reliable difference in the indicative periodontal parameters in the main and comparative groups with the chronic course of diseases - GP and generalized catarrhal gingivitis (table 1).

Thus, the GI, PMA, ARI indices corresponded to 1.432 ± 0.087 , 0.361 ± 0.018 , 0.467 ± 0.026 in the patients of the main group with GP of the initial degree, chronic course and 0.415 ± 0.091 , 0.348 ± 0.012 , 0.497 ± 0.025 in the comparative group, respectively GCCG (p ≤ 0.005).

It was found that the GP of the initial stage of the exacerbated flow characterized unsatisfactory indexes GI, PMA, ARI, which corresponded to 0.411 ± 0.086 , 0.348 ± 0.01 and 0.427 ± 0.027 , and in the GCCG group were 0.410 ± 0.08 , 0.341 ± 0.02 , respectively and 0.457 ± 0.021 ($p \le 0.005$). It is fair to note that, both in the GP groups and in the GCCG group, the periodontal parameters indicated a poor state of oral hygiene.

In the control group, these indices were significantly different and corresponded to 0.513 ± 0.071 , 0.416 ± 0.02 , 0.531 ± 0.029 (p ≤ 0.005).

Thus, it is possible to state an identical unsatisfactory state of oral hygiene in patients of both groups with HP and GCCG, more pronounced in the aggravated flow.

			Periodontal indices *		
Group	Diagnosis		GI, M±m	PMA, M±m	API, M±m
.ii	GP, initial degree, chronic course	2	1,432±0,087	0,361±0,018	0,467±0,026
Main	GP, initial degree, exacerbated current	0	0,411±0,086	0,348±0,013	0,427±0,027
ative	GHCG, remission	9	0,415±0,092	0,348±0,011	0,497±0,025
Comparative	GHCG, exacerbation	2	0,410±0,082	0,341±0,022	0,457±0,021
Control	clinically intact parodontium	0	0,513±0,071	0,416±0,021	0,531±0,029

Table 1. Indicative indices of periodontal tissue of patients with generalized periodontitis and generalized chronic catarrhal gingivitis

* - probability <0.05 in relation to the norm

The data on the degree of tissue sensitization to the bone antigen are presented in table 2.

Table 2. The frequency of tissue sensitization to bone antigen in generalized periodontitis and generalized chronic catarrhal gingivitis

		Tissue allergy		
Groups	Diagnosis	The number of examined	Bone antigen RIML *	
Main	GP, initial degree, chronic course	72	61,0±5,72	
Iviain	GP, initial degree, exacerbated current	60	80,0±5,13	
Comparative	GCCG, chronic course	29	0	
	GCCG, exacerbated current	42	0	
Control	Practically healthy	30	0	

* - % positive reactions

As follows from table 2, the expressed degree of tissue sensitization to the bone antigen in patients with GP of the initial degree with chronic course was observed in $61.0 \pm 5.72\%$ of patients (p <0.01) and only in $31.9 \pm 4.8\%$ of patients had a moderate degree of sensitization to the bone antigen.

In the acute course of GP, the initial degree, of tissue sensitization to the bone antigen was noted in $80,0 \pm 5,13\%$ (p <0,01) of the examined, which is significantly higher than in chronic course. In 20% of patients with GP of the initial degree of exacerbated current had a moderate degree of sensitization to the bone antigen was noted.

In no case in the patients of the comparative group, in both chronic and exacerbated catarrhal gingivitis, as well as in the control group, tissue sensitization to the bone antigen is not established.

Conclusions:

1. It is confirmed that the periodontal indices (PMA, bleeding index GI, integral index of hygiene ARI) can not be considered as indicative indicators reflecting the condition of the periodontal complex, but only reflect the condition of soft periodontal tissues.

2. The established expressed degree of tissue sensitization to the bone antigen in the prevailing majority of patients with GP of the initial degree, which is directly dependent on the nature of the

course, can be considered as an indicative pathognomonic index in differential diagnosis of GP of the initial degree and generalized chronic catarrhal gingivitis.

3. We suppose that the presence of similar and cross connective tissue antigens with a number of representatives of the oral microbiome can be a key trigger for a malfunction in the immune recognition of "own" "alien" with the further inclusion of a stable autoimmune reaction that determines the peculiarity of the course of the disease and the result of its treatment.

4. The revealed tissue sensitization to the bone antigen in patients with GP even at the initial degree requires mandatory inclusion in the general treatment regimen of osteotropic drugs, including preparations of vitamin D_3 , which provides differentiation of cells of the alveolar process, potentiation of carbohydrate, lipid metabolism.

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