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RESEARCH ARTICLE

THE IMPACT OF CHRONIC PERIODONTITIS ON THE RISK OF ATHEROSCLEROSIS AND CORONARY HEART DISEASE

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Abstract: The article discusses the problem of the influence of chronic periodontitis on the risk of developing atherosclerosis and coronary heart disease. Taking into account the current understanding of atherosclerosis as a chronic inflammatory process and the data on the systemic nature of inflammation in periodontal diseases, the possible pathophysiological mechanisms of the relationship between the two pathologies are analyzed. A clinical and laboratory study of 50 patients examined the association between the presence of chronic periodontitis, the level of markers of systemic inflammation, subclinical signs of atherosclerosis, and the frequency of clinically manifest coronary artery disease. It has been shown that individuals with chronic periodontitis have higher levels of high-sensitivity C-reactive protein, greater intima-media thickness of the carotid arteries, a more unfavorable lipid profile, and a higher incidence of clinically diagnosed coronary artery disease compared to patients without signs of periodontal inflammation. Possible ways of chronic infection and persistent inflammation in periodontal tissues influence on endothelial dysfunction, systemic inflammation and progression of atherosclerotic lesions of arteries are discussed, as well as limitations of interpretation of the obtained data in terms of cause-and-effect relationships. Practical conclusions about the importance of dental status for assessment of cardiovascular risk are formulated and promising directions of further research are outlined.

Keywords: Chronic periodontitis, atherosclerosis, coronary heart disease, cardiovascular risk, systemic inflammation, endothelial dysfunction

INTRODUCTION

Chronic periodontitis is one of the most common inflammatory diseases in humans and is considered one of the leading causes of tooth loss in adults and the elderly. According to epidemiological studies, various forms of periodontitis are present in a significant portion of the adult population, and severe forms of periodontal disease are among the most common non-communicable diseases in the world. At the same time, atherosclerotic cardiovascular diseases, including coronary heart disease, remain the leading cause of mortality and disability, which has led to a growing interest in studying non-traditional risk factors, including inflammatory oral diseases [9].

In recent decades, a significant amount of data has been accumulated indicating a statistically significant association between periodontal diseases and atherosclerotic cardiovascular diseases [10]. Several large-scale observational studies have shown that the presence of periodontitis is associated with an increased risk of atherosclerotic cardiovascular disease and acute coronary events, even after accounting for traditional risk factors such as age, smoking, hypertension, dyslipidemia, and diabetes mellitus [11]. It is emphasized that the evidence of a causal relationship is still insufficient, but the combination of clinical and experimental data makes the hypothesis of the influence of periodontitis on atherogenesis pathophysiologically justified [5].

Potential mechanisms of the relationship between periodontitis and atherosclerosis are considered in the context of the concept of chronic low-intensity inflammation. It has been shown that inflammation in periodontal tissues is accompanied by transient bacteremia, the penetration of microbial antigens and inflammatory mediators into the systemic circulation, and an increase in the levels of pro-inflammatory cytokines and C-reactive protein. The detection of DNA of typical periodontopathogens in atherosclerotic plaques, as well as data on the effect of treatment for severe periodontitis on markers of vascular inflammation and intima-media thickness, suggests that chronic periodontal infection may contribute to the development of atherosclerotic lesions [1].

In addition, it is emphasized that periodontitis and atherosclerosis share common risk factors, including smoking, diabetes mellitus, obesity, and low socioeconomic status. This complicates the interpretation of the associations observed and requires careful consideration of confounding factors in data

analysis. However, given the high prevalence of periodontal diseases, even a moderate increase in cardiovascular risk associated with chronic periodontitis can have significant implications for public health [3].

The aim of this work is to assess the impact of chronic periodontitis on the indicators of subclinical atherosclerosis and the risk of developing coronary heart disease based on the results of a clinical and laboratory study, as well as to discuss the possible pathophysiological mechanisms of the identified associations.

The study was conducted at two state medical institutions in the Kabardino-Balkarian Republic, located in Nalchik: The Republican Clinical Hospital of the Ministry of Health of the Kabardino-Balkarian Republic and the Republican Dental Center named after T.Kh. Tkhazaplishiev of the Ministry of Health of the Kabardino-Balkarian Republic.

MATERIAL AND METHODS

The study was performed as a one-time observational study with the inclusion of 50 patients who applied for routine dental care. The sample included men and women aged 35 to 65 years, who did not have a history of acute inflammatory diseases in the last four weeks and severe systemic diseases that could significantly affect the level of systemic inflammation, except for common cardiovascular risk factors.

The dental examination was carried out with an assessment of the oral hygiene index, the depth of periodontal pockets, the level of attachment of the gums, the presence of bleeding on probing and radiological signs of alveolar bone resorption. The diagnosis of chronic periodontitis was established in the presence of characteristic clinical and radiological signs of inflammatory-destructive periodontal lesions with a duration of symptoms of more than three months. Patients with localized forms of acute periodontitis, as well as those who had received specialized periodontal treatment less than six months before the examination, were excluded from the study.

Depending on the presence of chronic periodontitis, the patients were divided into two groups that were comparable in terms of age and gender. The first group consisted of 25 patients with clinically and radiographically confirmed chronic generalized periodontitis of mild to moderate severity. The second group included 25 patients without signs of inflammatory periodontal diseases, who had either clinically healthy periodontium or mild gingivitis without bone tissue destruction.

The cardiological examination included measurement of blood pressure, registration of an electrocardiogram in 12 leads, and collection of anamnestic data on the presence of diagnosed coronary artery disease, angina pectoris, myocardial infarction, and coronary revascularization. If relevant documentation was available, the results of previous stress tests and coronary angiography were also taken into account. The 10-year cardiovascular risk stratification was performed using a scale similar to the commonly accepted risk models based on age, gender, blood pressure, smoking, and lipid profile.

The laboratory examination included determination of the levels of total cholesterol, low-density and high-density lipoproteins, triglycerides, fasting glucose, as well as high-sensitivity C-reactive protein (hs-CRP) as a marker of systemic inflammation. To assess subclinical atherosclerosis, an ultrasound examination of the carotid arteries was performed with measurement of the thickness of the intima–media complex of the common carotid artery by the standard technique and detection of the presence of local atherosclerotic plaques. The average value of the intima-media complex thickness was calculated as the arithmetic mean of three consecutive measurements on each side.

The statistical analysis included a descriptive characterization of the sample, presenting quantitative indicators as mean values and standard deviations, and categorical indicators as proportions.

The Student's t-test was used to compare quantitative parameters between the two groups, provided that the distribution was normal; the chi-square test was used to compare proportions. The study also evaluated the association between the presence of chronic periodontitis and increased intima-media thickness, as well as the presence of clinically manifest coronary artery disease, using simple logistic regression.

RESULTS AND DISCUSSIONS:

The Republican Clinical Hospital of the Ministry of Health of the Kabardino-Balkarian Republic (Nalchik) is the largest multidisciplinary medical and diagnostic center in the republic, providing primary, specialized, and high-tech medical care to adults, including patients with cardiovascular diseases. The hospital has therapeutic and cardiology departments, a department of vascular pathology, and a consultative and diagnostic service that provide a wide range of examinations necessary for the verification of atherosclerotic vascular lesions and coronary heart disease. The institution has a well-developed laboratory and instrumental base, which allows for a standardized assessment of cardiovascular risk factors, lipid metabolism indicators, and concomitant pathology.

The choice of the Republican Clinical Hospital as a clinical base is due to the fact that this state institution concentrates a significant flow of patients with atherosclerosis and coronary heart disease from various regions of the

Kabardino-Balkarian Republic, which allows for the formation of a representative sample for analyzing the association of chronic periodontitis with cardiovascular risk. The presence of specialized cardiology departments and a regional vascular center provide for comprehensive cardiological examinations, and the state status and subordination to the Ministry of Health of the KBR contribute to the implementation of unified diagnostic and treatment standards.

The dental link of the study was carried out by the GBUZ "Republican Dental Center named after T.Kh. Tkhazaplizhev" of the Ministry of Health of the Kabardino-Balkarian Republic (Nalchik), a specialized state institution that provides outpatient dental care to the adult and child population of the republic. The center includes a therapeutic, surgical, and children's department, as well as other structural units, which allows for comprehensive diagnosis and treatment of dental and periodontal diseases, including chronic periodontitis.
The clinical and demographic characteristics of the patients included in the study are presented in Table 1.

Table 1 – Clinical and demographic characteristics of the examined patients (n = 50)

Indicator	The general group (n=50)	Chronic periodontitis (n=25)	Without chronic periodontitis (n=25)	p
Age, years (M ± SD)	51,2 ± 7,8	52,0 ± 7,5	50,4 ± 8,0	0,48
Men, %	54	56	52	0,78
Body mass index, kg/m² (M ± SD)	28,6 ± 3,9	29,1 ± 4,1	28,1 ± 3,7	0,42
Active smoking, %	38	44	32	0,36
Arterial hypertension, %	46	52	40	0,41
Type 2 diabetes, %	16	20	12	0,45
Average estimated 10-year risk, % (M ± SD)	11,4 ± 4,7	12,6 ± 4,9	10,2 ± 4,3	0,11

The obtained data demonstrate the comparability of the groups in terms of the main traditional cardiovascular risk factors, including age, gender, body mass index, frequency of smoking, arterial hypertension and diabetes mellitus. The differences in the calculated 10-year cardiovascular risk were tended towards higher values in the group of patients with chronic periodontitis, however, within the small sample did not reach statistical significance.

Biochemical and instrumental indicators of lipid metabolism, the level of systemic inflammation, and the severity of subclinical atherosclerosis are presented in Table 2.

Table 2 – Indicators of lipid profile, systemic inflammation, and subclinical atherosclerosis in patients with and without chronic periodontitis

Indicator	Chronic periodontitis (n=25)	Without chronic periodontitis (n=25)	p
Total cholesterol, mmol/L (M ± SD)	5,7 ± 0,8	5,3 ± 0,7	0,08
Low-density lipoproteins, mmol/L (M ± SD)	3,6 ± 0,7	3,1 ± 0,6	0,02
High-density lipoproteins, mmol/L (M ± SD)	1,08 ± 0,21	1,18 ± 0,23	0,09
Triglycerides, mmol/L (M ± SD)	1,9 ± 0,6	1,6 ± 0,5	0,07
hs-CRP, mg/L (median [25-; 75-percentile])	3,4 [2,1; 4,8]	1,9 [1,1; 2,7]	0,01
Average IMT, mm (M ± SD)	0,89 ± 0,12	0,79 ± 0,11	0,004
Presence of atherosclerotic plaques, %	40	20	0,12
Clinically manifest IHD, %	32	12	0,08

The results demonstrate a number of patterns that are consistent with the literature data on an unfavorable cardiovascular profile in patients with chronic periodontitis. In the group of patients with periodontitis, higher values of the concentration of low-density lipoproteins and high-sensitivity C-reactive protein were noted, as well as a statistically significant increase in the average thickness of the intima–media complex compared to patients without inflammatory periodontal diseases. The differences in the frequency of detection of atherosclerotic plaques and clinically manifest coronary artery disease were only trends; more pronounced differences might have been detected in a larger sample.

The data obtained confirm the hypothesis that chronic periodontitis is associated with increased activity of systemic inflammation and more pronounced signs of subclinical atherosclerosis. The revealed increase in the thickness of the intima–media complex and levels of hs-CRP in patients with periodontitis is consistent with the results of other studies demonstrating the relationship of inflammation in the periodontium with indicators of vascular inflammation and structural changes in the arterial wall.

A simple logistic regression analysis showed that the presence of chronic periodontitis was associated with an increased likelihood of having an increased intima-media thickness (defined as a value above the age-specific median level in the sample). The estimated relative risk was approximately 2.1, with a wide confidence interval due to the small sample size, but it reflected the direction of the association. When age and smoking were included in the model, the estimated effect was slightly reduced, indicating the importance of controlling confounding factors, but it still showed a tendency for chronic periodontitis to have an adverse effect on vascular parameters.

From the point of view of pathophysiology, the results obtained can be interpreted within the framework of the concept of chronic systemic inflammation initiated by a local infectious-inflammatory process in the periodontium. The presence of a chronic focus of infection contributes to repeated episodes of bacteremia, activation of the innate immune response, increased levels of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor alpha, as well as the synthesis of C-reactive protein in the liver [2].

These inflammatory mediators affect the vascular endothelium, increase the expression of adhesion molecules, promote the influx of monocytes into the arterial intima and their differentiation into macrophages, which accelerates the formation of atherosclerotic plaques and increases their vulnerability.

The conceptual diagram of the possible influence of chronic periodontitis on atherogenesis is presented in Figure 1.

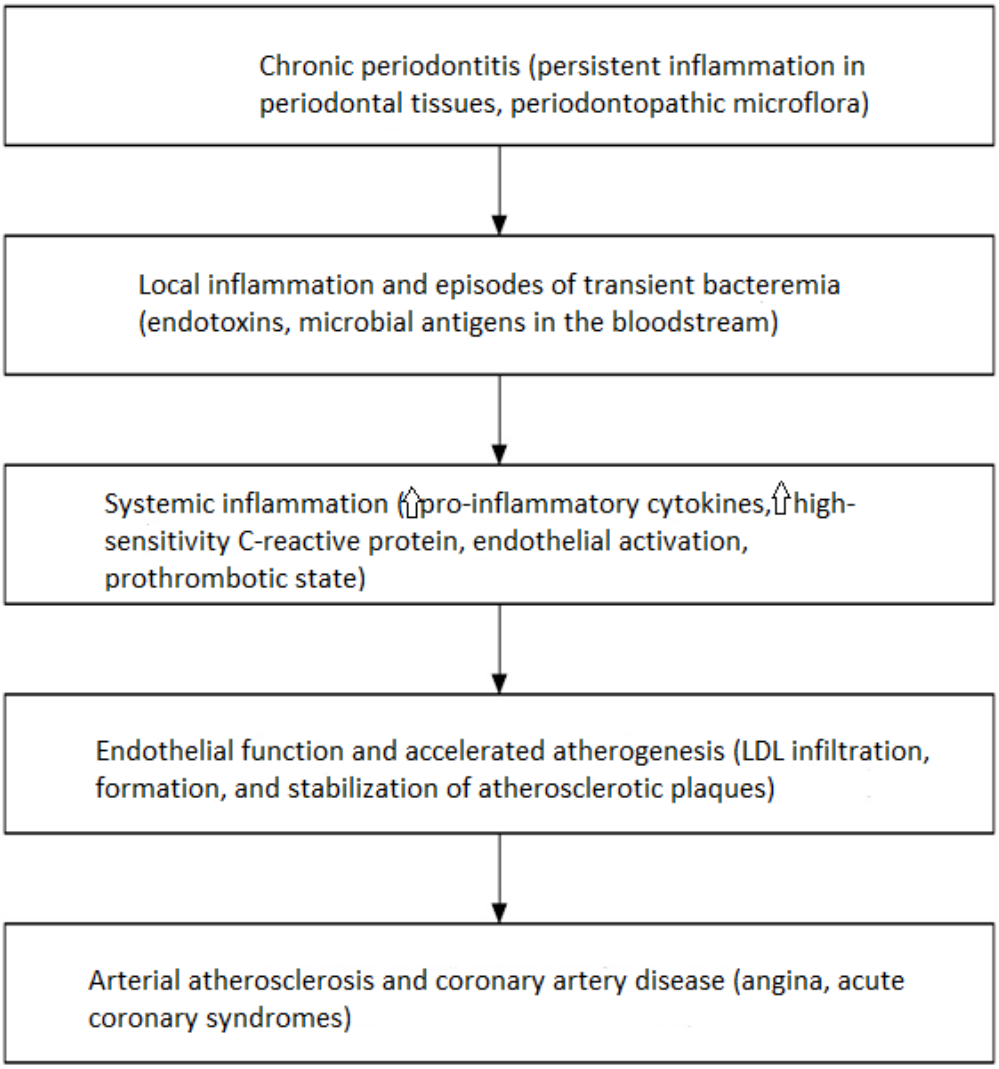

Figure 1 – Scheme of the proposed pathophysiological mechanisms of the effect of chronic periodontitis on atherosclerosis and coronary heart disease

In this scheme, the initial link is chronic inflammation in periodontal tissues, supported by the persistence of periodontopathic microflora and an imbalance in the local immune response. This local process is accompanied by the entry of bacterial endotoxins and fragments of cell walls into the systemic circulation, as well as repeated episodes of

transient bacteremia. At the next level, systemic inflammation develops, manifested by increased levels of pro-inflammatory cytokines and C-reactive protein, endothelial cell activation, and a shift in the hemostatic balance towards a pro-thrombotic state. This leads to endothelial dysfunction and increased infiltration of the arterial wall with low-density lipoproteins, which, along with changes in the lipid profile, contributes to the accelerated formation and destabilization of atherosclerotic plaques. The final link in the chain is the clinical manifestations of atherosclerotic coronary artery disease in the form of coronary artery disease, including stable angina and acute coronary syndromes [7].

When interpreting the results, it is necessary to take into account a number of study limitations. First of all, the design of a one-time observational study does not allow for unambiguous conclusions about the causal nature of the identified associations. There is no opportunity to trace the dynamics of vascular changes against the background of treatment of chronic periodontitis, which is especially important in the context of the hypothesis discussed in the literature about the modifiable nature of cardiovascular risk associated with the condition of periodontium. In addition, the small sample size limits the statistical power of the study, making it difficult to detect subtle differences and reliably assess the impact of potential confounding factors [4].

Nevertheless, even within the framework of the conducted observation, the identified differences in the levels of hs-CRP, low-density lipoproteins, and intima-media thickness appear to be clinically significant. They confirm the appropriateness of including data on dental status, particularly the presence of chronic periodontitis, in the expanded assessment of overall cardiovascular risk in middle-aged and elderly patients, especially in the presence of additional risk factors [6].

From a practical point of view, the results support the idea of interdisciplinary collaboration between dentists, general practitioners, and cardiologists in the management of patients with high cardiovascular risk. The presence of severe periodontal inflammation in such patients should be considered as a potential marker of an unfavorable cardiovascular profile and a reason for a more thorough assessment of risk and optimization of modifiable factors, including blood pressure control, lipid metabolism, glycemic control, and smoking cessation. At the same time, the treatment of chronic periodontitis in itself cannot be considered as a substitute for standard cardiovascular prevention, but should complement existing approaches as a component of a strategy for managing systemic inflammation.

CONCLUSION

The study demonstrated a statistically significant association between chronic periodontitis and indicators of systemic inflammation and subclinical atherosclerosis in middle-aged patients. Compared to patients without inflammatory periodontal diseases, individuals with chronic periodontitis had higher levels of high-sensitivity C-reactive protein, a more atherogenic lipid profile, and significantly thicker intima-media complex of the carotid arteries. There was a tendency towards a higher frequency of clinically manifest coronary artery disease in the group of patients with chronic periodontitis.

The obtained data are consistent with the modern concept of the involvement of chronic inflammation in the pathogenesis of atherosclerosis and confirm the pathophysiological plausibility of the hypothesis about the influence of chronic periodontitis on cardiovascular risk. At the same time, it is emphasized that the established associations cannot be considered as proof of a direct cause-and-effect relationship, since the results were obtained within the framework of a one-time observational study and in a limited sample.

The practical significance of the work lies in substantiating the need to take into account the dental

status, primarily the presence of chronic periodontitis, in a multifactorial assessment of cardiovascular risk. Therapeutic and preventive measures aimed at oral cavity sanitation and control of chronic inflammation in periodontium can be considered as a potentially important component of an integrated strategy for the prevention of atherosclerotic cardiovascular diseases, especially in patients with a high initial risk.

The prospects for further research include conducting large-scale prospective studies to assess the impact of chronic periodontitis treatment on the dynamics of subclinical atherosclerosis, the frequency of coronary events, and overall cardiovascular mortality, as well as elucidating the molecular mechanisms underlying the relationship between local periodontal inflammation and systemic vascular inflammation. An additional area of development could be the creation of integrated prevention programs that combine dental and cardiological approaches to risk management, with an assessment of their clinical and economic effectiveness.

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