

# THE INFLUENCE OF PERIODONTAL THERAPY ON THE LEVEL OF SYSTEMIC INFLAMMATORY MARKERS IN CARDIAC PATIENTS

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## ABSTRACT

Given the proven role of low-grade chronic inflammation in the progression of atherosclerosis and cardiovascular disease, studying the effect of oral hygiene on markers of systemic inflammation appears clinically significant.

The study was conducted in the cardiology department of the Novosibirsk City Clinical Hospital No. 25 and included 110 patients with coronary heart disease and concomitant moderate to severe chronic generalized periodontitis. The study group (n=60) received standard cardiac therapy combined with comprehensive non-surgical periodontal care (professional hygiene, scaling and root smoothing, personal hygiene training, and antiseptic support). The control group (n=50) received only standard cardiac treatment and hygiene recommendations without active periodontal intervention.

Levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) were determined at baseline and after six months. In the study group, a statistically significant decrease in CRP by an average of 33%, IL-6 by 27%, and TNF- $\alpha$  by 25% compared to baseline was observed, while no significant changes were observed in the control group.

These results indicate that the treatment of chronic infection and inflammation in the oral cavity in cardiac patients is accompanied by a reduction in the systemic inflammatory response and can be considered as an additional approach to secondary prevention of cardiovascular complications.

**KEYWORDS:** chronic generalized periodontitis, systemic inflammation, C-reactive protein, interleukin-6, tumor necrosis factor- $\alpha$ , cardiac patients, periodontal therapy, cardiovascular risk.

## INTRODUCTION

Periodontitis is considered a chronic inflammatory disease of the periodontal tissues, characterized by the destruction of the tooth-supporting apparatus and the formation of pathological periodontal pockets. According to international epidemiological studies, severe forms of periodontitis affect up to 10% of the adult population and are associated with a significant reduction in quality of life and a high risk of tooth loss. Chronic periodontitis is currently considered not only a localized dental pathology but also a significant source of low-intensity systemic inflammation [2]. Periodontopathogenic microorganisms and inflammatory mediators enter the systemic circulation, maintaining subclinical inflammation, which is considered one of the key mechanisms of atherogenesis and the progression of cardiovascular disease [5].

Cardiovascular disease remains the leading cause of death worldwide. Numerous studies have demonstrated that chronic periodontitis is statistically associated with an increased risk of coronary heart disease, myocardial infarction, and cerebrovascular events [8]. It is believed that the common link in pathogenesis is an increase in the level of systemic inflammatory markers, as well as endothelial dysfunction and activation of the platelet component of hemostasis. Among the most studied markers of systemic inflammation are high-sensitivity C-reactive protein, interleukin-6, and tumor necrosis factor- $\alpha$ , which reflect the degree of chronic inflammatory stress and act as predictors of adverse cardiovascular outcomes [11].

In parallel, the concept of an "oral-systemic continuum" emerged, according to which the treatment of chronic foci of infection and inflammation in the oral cavity can be accompanied not only by a local effect but also by a reduction in systemic inflammation. Meta-analyses of randomized clinical trials have shown that non-surgical periodontal therapy leads to a statistically significant reduction in CRP and IL-6 levels, as well as a moderate decrease in systolic blood pressure. However, most of these studies were conducted in dental populations, while specially selected cohorts of cardiac patients with documented coronary artery disease have received limited study [6].

In real-world cardiology practice, chronic periodontitis often remains undiagnosed and undertreated [14]. Patients focus on their heart problems, while oral complaints are perceived as secondary. Meanwhile, evidence of the impact of periodontal treatment on systemic inflammation and cardiovascular risk necessitates a reconsideration of approaches to interdisciplinary collaboration between cardiologists and dentists. It is important to study the extent to which the inclusion of comprehensive periodontal therapy in the treatment of cardiac patients can modify the levels of key inflammatory markers and, therefore, potentially impact prognosis [10].

The aim of this study was to evaluate the impact of comprehensive non-surgical periodontal therapy on the levels of C-reactive protein, interleukin-6, and tumor necrosis factor- $\alpha$  in patients with coronary artery disease and chronic generalized periodontitis treated in the cardiology department of City Clinical Hospital No. 25, Novosibirsk.

## RESEARCH MATERIALS AND METHODS

The study was conducted as a comparative observational study at the Cardiology Department of City Clinical Hospital No. 25 in Novosibirsk from January to December 2025.

The sample included 110 patients over 40 years of age with diagnosed coronary artery disease (stable angina of functional class II-III or post-myocardial infarction) who had moderate to severe chronic generalized periodontitis diagnosed during a dental examination.

Inclusion criteria included the presence of at least 15 intact teeth, clinical signs of periodontitis with a periodontal pocket depth of at least 4 mm, radiographically confirmed alveolar bone loss, and a stable dose of cardiac therapy for at least four weeks prior to inclusion. Exclusion criteria included acute inflammatory diseases, active autoimmune and oncological processes, decompensated diabetes mellitus, stage IV-V chronic kidney disease, use of systemic anti-inflammatory or immunomodulatory drugs, and previous surgery less than three months prior to inclusion.

After signing informed consent, patients underwent a baseline cardiac and dental examination, with documentation of clinical and demographic characteristics, risk factors, hemodynamic parameters, laboratory parameters, and periodontal indices. High-sensitivity CRP levels were determined by immunoturbidimetry, and IL-6 and TNF- $\alpha$  concentrations were determined by enzyme-linked immunosorbent assay. Venous blood samples were collected in the morning after fasting. Laboratory tests were performed in the same certified hospital laboratory.

Patients were divided into two groups by simple random assignment. The study group included 60 patients who received comprehensive non-surgical periodontal therapy alongside standard cardiac therapy. This included motivation and training in individual oral hygiene with the selection of care products, professional oral hygiene with the removal of supra- and subgingival calculus, closed curettage of periodontal pockets (scaling and root smoothing), and a two-week course of antiseptic rinses with chlorhexidine-based solutions. If necessary, correction of traumatic occlusion and temporary splinting of loose teeth were performed.

The control group included 50 patients who continued to receive standard cardiac therapy according to current clinical guidelines. They received a one-time instruction in individual oral hygiene without active periodontal interventions during the entire observation period. Thus, the key factor distinguishing the groups was the presence or absence of comprehensive periodontal treatment. Levels of CRP, IL-6, and TNF- $\alpha$  were measured twice: at study entry and six months later. Key cardiac parameters, including the frequency of angina attacks, blood pressure, and lipid profile, were also assessed to ensure the comparability of initial cardiac therapy between the groups. Statistical data processing included checking the distribution of variables, using parametric and nonparametric tests depending on the data type, and calculating p-values for intergroup and intragroup comparisons. The significance level was set at 0.05.

## RESULTS AND DISCUSSION

The study included 110 patients, including 62 men and 48 women. The mean age in the overall sample was 61.3  $\pm$  8.4 years and did not differ significantly between groups. The duration of coronary artery disease exceeded five years in most subjects. The prevalence of traditional cardiovascular risk factors, including hypertension, dyslipidemia, smoking, and obesity, was comparable in both groups, minimizing the impact of these variables on the dynamics of inflammatory markers.

The clinical and demographic characteristics of the patients participating in the study are presented in Table 1.

**Table 1.** Clinical and demographic characteristics of patients

| Indicator   | Main group (n=60) | Control group (n=50) | p    |
|---|-------------------|----------------------|------|
| Age, years (M $\pm$ SD)                                 | 61,5 $\pm$ 8,2    | 61,0 $\pm$ 8,7       | 0,78 |
| Men, %  | 56,7              | 58,0                 | 0,88 |
| Duration of coronary heart disease, years (median, IQR) | 7,0 (5,0–10,0)    | 7,0 (4,0–10,0)       | 0,94 |
| Arterialhypertension, %                                 | 83,3              | 82,0                 | 0,86 |
| Activesmoking, %  | 31,7              | 34,0                 | 0,77 |
| Body mass index, kg/m <sup>2</sup> (M $\pm$ SD)         | 29,1 $\pm$ 3,8    | 28,7 $\pm$ 3,9       | 0,62 |
| Severeperiodontitis, %                                  | 48,3              | 46,0                 | 0,80 |

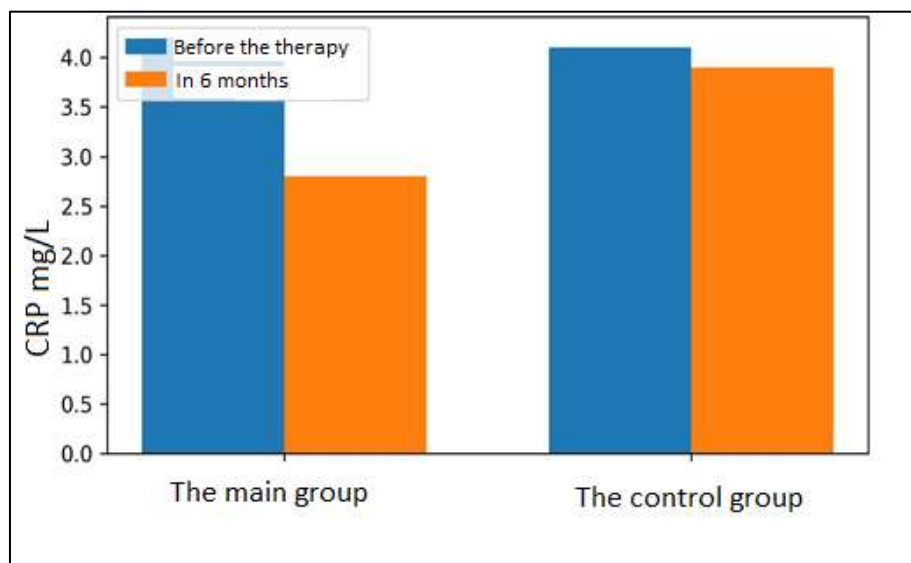
The baseline comparability of the groups in terms of key clinical and demographic indicators and periodontitis severity allows us to interpret the identified differences in the dynamics of systemic inflammatory markers primarily as reflecting the impact of periodontal therapy.

C-reactive protein is recognized as a key marker of systemic inflammation and a predictor of cardiovascular events. At baseline, the average CRP level in the study group was  $4.2 \pm 1.5$  mg/L, while in the control group it was  $4.1 \pm 1.4$  mg/L; the differences were not statistically significant. After six months, the study group showed a decrease in CRP to  $2.8 \pm 1.2$  mg/L, while in the control group it remained virtually unchanged at  $3.9 \pm 1.4$  mg/L.

**Table 2.** CRP Level Dynamics in Cardiology Patients with Periodontitis

| Group          | CRP at baseline, mg/L (M $\pm$ SD) | CRP after 6 months, mg/L (M $\pm$ SD) | Intragroup p | Intragroup p (after 6 months) |
|----------------|------------------------------------|---------------------------------------|--------------|-------------------------------|
| Main (n=60)    | 4,2 $\pm$ 1,5                      | 2,8 $\pm$ 1,2                         | < 0,001      | < 0,01                        |
| Control (n=50) | 4,1 $\pm$ 1,4                      | 3,9 $\pm$ 1,4                         | 0,18         | —                             |

The dynamics of CRP are presented in Figure 1, which clearly shows a significant decrease in the marker level in the main group in the absence of clinically significant changes in the control group.



**Figure 1.** CRP Dynamics

These data are consistent with the results of randomized trials, in which non-surgical periodontal therapy in patients with chronic periodontitis resulted in a reduction in CRP by approximately one-third of baseline values. In the context of the cardiac population, this reduction can be considered an additional reserve for modifying residual inflammatory risk, especially in patients who have achieved target lipid profile and blood pressure values but retain elevated CRP levels.

Interleukin-6 and tumor necrosis factor- $\alpha$  are proinflammatory cytokines that play a key role in the systemic inflammatory response. The pathogenetic link between periodontitis and cardiovascular disease is based on both the direct entry of periodontopathogenic bacteria into the bloodstream and a generalized cytokine response, which is reflected in the levels of these markers. At baseline, the average IL-6 level in the study group was  $4.8 \pm 1.6$  pg/ml, while in the control group it was  $4.7 \pm 1.5$  pg/ml. The average TNF- $\alpha$  level was  $7.5 \pm 2.1$  pg/ml and  $7.4 \pm 2.0$  pg/ml, respectively. Six months after periodontal therapy, IL-6 levels in the study group decreased to  $3.5 \pm 1.3$  pg/ml, and TNF- $\alpha$  levels decreased to  $5.6 \pm 1.8$  pg/ml. In the control group, the levels remained at baseline levels with minor, statistically insignificant fluctuations.

**Table 3.** IL-6 and TNF- $\alpha$  Level Dynamics

| Indicator             | Group Initial (M $\pm$ SD) | After 6 months (M $\pm$ SD) | Intragroup p  | p       |
|-----------------------|----------------------------|-----------------------------|---------------|---------|
| IL-6, pg/ml           | Main (n=60)                | 4,8 $\pm$ 1,6               | 3,5 $\pm$ 1,3 | < 0,001 |
| IL-6, pg/ml           | Control (n=50)             | 4,7 $\pm$ 1,5               | 4,6 $\pm$ 1,5 | 0,41    |
| TNF- $\alpha$ , pg/ml | Main (n=60)                | 7,5 $\pm$ 2,1               | 5,6 $\pm$ 1,8 | < 0,001 |
| TNF- $\alpha$ , pg/ml | Control (n=50)             | 7,4 $\pm$ 2,0               | 7,2 $\pm$ 2,0 | 0,29    |

The observed reduction in IL-6 and TNF- $\alpha$  levels in the study group supports the hypothesis that debridement of chronic inflammatory foci in the oral cavity leads to a reduction in the systemic cytokine response. These results are consistent with a meta-analysis showing a moderate but sustained decrease in IL-6 after non-surgical periodontal therapy. In cardiology practice, such dynamics may have clinical significance, given the role of IL-6 and TNF- $\alpha$  in the progression of atherosclerosis and vascular remodeling.

It is interesting to note that the reduction in cytokine levels in the study group was not accompanied by significant changes in traditional risk factors over the study period. This suggests that periodontal debridement was the primary modifying factor for systemic inflammation in this cohort. At the same time, the contribution of improved

adherence to cardiac therapy and non-pharmacological recommendations, which is often observed with more active interaction between medical staff and patients, cannot be completely ruled out. The overall reduction in CRP, IL-6, and TNF- $\alpha$  levels in the study group indicates a significant reduction in systemic inflammatory status after comprehensive periodontal therapy in addition to standard cardiac treatment. In the context of current understanding of residual inflammatory risk in patients with coronary heart disease, this effect can be considered an additional non-pharmacological tool for its correction.

International guidelines increasingly emphasize the importance of assessing chronic foci of infection and inflammation, including dental status, in patients at high cardiovascular risk [12]. The data obtained support the need to include periodontitis screening and subsequent targeted periodontal therapy in interdisciplinary care programs for cardiac patients. This is especially relevant for patients with persistently elevated CRP despite optimization of lipid-lowering and antiplatelet therapy [13].

It should be emphasized that a reduction in inflammatory markers alone does not directly demonstrate a reduction in the incidence of clinical cardiovascular events. However, given the accumulated data on the prognostic value of CRP and proinflammatory cytokines, it can be assumed that normalization of these parameters in response to oral hygiene can contribute to an improved prognosis in the long term. Large randomized trials with long-term follow-up and assessment of "hard" endpoints are needed to definitively confirm this hypothesis. However, it already seems rational to consider periodontal therapy as part of a comprehensive approach to reducing cardiovascular risk [4].

The organizational aspect of integrating dental care into the work of a cardiology hospital deserves special attention. As part of the study, a system was implemented for referring patients from the cardiology department for a consultation with a periodontist, followed by the necessary interventions. The positive experience of this interaction allows us to recommend similar models for implementation in other multidisciplinary hospitals, particularly in regions with a high prevalence of cardiovascular diseases and limited access to specialized dental care [6].

As part of the study, a structured periodontal care program was implemented in the main group, integrated into the therapeutic pathway of the cardiology department. The program included sequential stages from initial screening and motivation to maintenance therapy, with clear regulations on the timing of visits, the scope of interventions, and the distribution of responsibilities between specialists. This approach not only standardized the treatment of periodontitis in cardiology patients but also ensured the reproducibility of interventions in a multidisciplinary hospital setting.

The initial assessment of dental status was conducted within the first three days of hospitalization. A cardiologist reviewed the patient's medical history to determine whether they had any complaints of bleeding gums, tooth mobility, bad breath, or periodontal flare-ups. The patient was then referred to a hospital-based periodontist for a consultation. During the examination, hygiene and periodontal indices, periodontal pocket depth, recession, tooth mobility, and radiographic evidence of bone loss were recorded. Based on the examination results, an individualized periodontal treatment plan was developed, taking into account the patient's cardiac status and concomitant therapy [1].

The important component of the program was targeted motivation for maintaining personalized oral hygiene. Even during the initial visit, the patient was clearly informed of the relationship between chronic periodontal inflammation, increased systemic inflammation, and the potential for worsening coronary heart disease. A soft-bristled toothbrushing technique using a modified Bass method was demonstrated, emphasizing the need to use interdental brushes or dental floss depending on the width of the interdental spaces. Individually appropriate hygiene products were selected for the patient, taking into account the condition of the gums, the presence of orthodontic appliances, and manual abilities. A short, written care protocol, formulated in simple language, was also provided.

The main interventions included professional oral hygiene and non-surgical periodontal therapy. During the first week after hospitalization, in the absence of cardiovascular contraindications, supra- and subgingival calculus was removed using ultrasonic scaling and hand instruments. Treatment was performed quadrant by quadrant, allowing for the duration of the procedures to be tailored to the patient's tolerance and the extent of the lesion. After scaling, root smoothing and polishing of tooth surfaces were performed, which helped to reduce plaque retention and decrease the microbial load in the periodontal pockets.

After completing the main phase of therapy, antiseptic support was prescribed at the outpatient stage. The patient was recommended to rinse with a low-concentration chlorhexidine bigluconate antiseptic solution twice daily for ten to fourteen days, with additional emphasis on the need to continue mechanical plaque removal. In cases of severe inflammatory reactions and deep periodontal pockets, the periodontist could decide to locally apply antimicrobial agents in the form of gels or applications to the most affected areas.

The crucial part of the treatment package was a system of scheduled follow-up visits and maintenance therapy. The first follow-up examination, including reassessment of indexes, pocket depth, and gum condition, was conducted six to eight weeks after the main intervention. At this stage, additional treatment of specific areas was performed as needed, and personal hygiene recommendations were adjusted. Patients in the main group were then invited for maintenance visits every three to four months, during which they underwent professional cleaning, assessed adherence to home care, verified the presence of flare-ups, and repeated brief consultations on the relationship between periodontitis and cardiovascular risk.

Implementation of the program required formalization of patient routing and specialist interactions. The cardiologist initiated dental screening and, upon discharge, recorded the presence of chronic generalized periodontitis in the discharge summary and recommended continued follow-up with a periodontist. The

periodontist, in turn, informed the attending cardiologist of the examination results and the extent of the treatment performed, allowing for consideration of the dental status when assessing the overall inflammatory background. Cardiology nurses participated in the educational component, reminding patients of the need for regular dental cleaning and the use of prescribed care products, as well as monitoring compliance with recommendations during the early inpatient period. To ensure the reproducibility of the intervention package, a standard operating procedure for the care of cardiac patients with chronic periodontitis was developed and implemented. The document included selection criteria, an algorithm for scheduling periodontal consultations, a list of mandatory procedures, medical record-keeping requirements, and a follow-up monitoring plan. The existence of such a regulatory document allowed periodontal therapy to be expanded beyond one-time consultations and become a systemic component of comprehensive treatment.

The content and structure of the periodontal intervention package implemented in the study group are summarized in the table, facilitating the replication of this approach in other cardiac hospitals and can serve as a basis for developing local protocols for interdisciplinary collaboration.

**Table 4.** Specific activities of the periodontal care program for cardiac patients with chronic periodontitis

| <b>Program stage</b>                                       | <b>Implementation period</b>                | <b>Main content of the intervention</b>   | <b>Responsible specialist</b>          | <b>Expected effect</b>   |
|--|---|---|--|--|
| Primary periodontal screening                              | In the first 1–3 days of hospitalization    | Collection of anamnesis, examination of the oral cavity, assessment of indices, preliminary formulation of a diagnosis of chronic periodontitis | Dentist-periodontist, cardiologist     | Identification of patients with periodontitis, formation of a risk group for increased systemic inflammation |
| Motivation and hygiene training                            | At the initial dental visit                 | Explaining the link between periodontitis and cardiovascular risk, teaching toothbrushing techniques, and selecting personal hygiene products   | Dentist-periodontist                   | Increased adherence to oral care, reduced formation of soft plaque   |
| Professional hygiene and scaling                           | In the first 7-10 days with a stable status | Removal of supra- and subgingival deposits, ultrasonic and manual scaling by quadrants, root planing, polishing                                 | Dentist-periodontist, dental assistant | Reducing the microbial load, decreasing the depth of pockets, stopping severe inflammation                   |
| Local antiseptic and anti-inflammatory measures            | Within 10-14 days after scaling             | Prescribing antiseptic rinses, local application of gels and applications in areas of maximum inflammation                                      | Dentist-periodontist                   | Maintaining the anti-inflammatory effect, preventing exacerbations   |
| Follow-up visit with reassessment of periodontal condition | 6-8 weeks after the main treatment          | Re-evaluation of indices, pocket depth and tooth mobility, adjustment of the treatment plan and hygiene recommendations                         | Dentist-periodontist                   | Evaluation of therapy effectiveness, individualization of the support program                                |
| Supportive periodontal therapy                             | Every 3-4 months throughout the year        | Planned professional hygiene, adherence monitoring, local treatment of individual areas if necessary  | Dentist-periodontist                   | Maintenance of periodontitis remission, sustainable reduction of systemic inflammatory background            |
| Information exchange between specialists                   | At all stages of the route                  | Transfer of information on dental status and performed sanitation, reflection of data in medical reports and outpatient cards                   | Cardiologist, dentist-periodontist     | Ensuring continuity of treatment, taking into account the dental factor when assessing cardiovascular risk   |

Incorporating the described set of measures into the treatment process of a cardiology hospital allows periodontal therapy to be considered not as an isolated dental episode, but as an integrated part of secondary prevention in patients with coronary heart disease.

Standardization of stages, timeframes, and responsible individuals facilitates the implementation of such a program in practice and creates the preconditions for further expansion of interdisciplinary collaboration between cardiologists and dentists to reduce systemic inflammatory risk.

## CONCLUSION

Comprehensive non-surgical periodontal therapy in patients with coronary heart disease and chronic generalized periodontitis treated in a cardiology hospital is accompanied by a significant reduction in the levels of key systemic

inflammatory markers—C-reactive protein, interleukin-6, and tumor necrosis factor- $\alpha$ . In patients receiving only standard cardiac therapy without active periodontal debridement, no significant changes in these parameters were observed over a comparable observation period. The obtained results confirm the pathogenetic relationship between periodontal health and systemic inflammatory conditions in cardiac patients and demonstrate the feasibility of incorporating periodontal diagnostics and treatment into secondary prevention measures for cardiovascular complications. The implementation of interdisciplinary models of collaboration between cardiologists and dentists in city clinical hospitals, such as City Clinical Hospital No. 25 in Novosibirsk, can be considered a promising approach to improving the quality of medical care for patients at high cardiovascular risk.

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