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PERIODONTAL THERAPY IN THE COMPLEX TREATMENT OF PATIENTS WITH CORONARY HEART DISEASE: A SYSTEMATIC REVIEW

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ABSTRACT

Modern dentistry includes many unresolved scientific and practical issues: despite significant medical achievements, the main dental diseases (carious lesions, periodontal pathology, lesions of the oral mucosa) remain relevant. Their medical and social indicators are extremely high, and the improvement of diagnosis, treatment and prevention remains in high demand.

The aim of the work is to systematize interventional data on periodontal therapy in patients with coronary heart disease and to determine which endpoints provide the greatest practical benefits.

The qualitative synthesis included 7 studies with a total of 725 participants. In all studies after treatment, there was a distinct improvement in local periodontal parameters.

Changes in the lipid profile and data on severe cardiovascular outcomes remained limited. Periodontal therapy in patients with coronary heart disease looks like a clinically justified supplement, primarily as a way to reduce the inflammatory load, but it is too early to interpret it as an independent means of secondary cardiological prevention.

Keywords: periodontitis, periodontal therapy, coronary artery disease, coronary heart disease, C-reactive protein, endothelial function, systematic review.

INTRODUCTION

Periodontitis is a chronic inflammatory disease of periodontal tissues that can lead to bone destruction and tooth loss. There is a large amount of evidence indicating a relationship between oral diseases and systemic diseases, especially cardiovascular pathologies such as coronary heart disease (CHD). Coronary artery disease is one of the most common and serious heart diseases, which is characterized by impaired blood supply to the heart muscle due to atherosclerosis of the coronary arteries. [1]

Cardiovascular diseases continue to make the main contribution to global mortality, and atherosclerotic forms of myocardial damage make up a particularly severe part of this group. According to the World Health Organization, 19.8 million people died from cardiovascular causes in the past 2025 [1]. Periodontitis, at first glance a local dental pathology, is no less common.

Numerous studies confirm that the presence of chronic inflammatory processes such as periodontitis can worsen the course of coronary heart disease,[2] increasing the risk of complications such as myocardial infarction. [3] Systemic inflammation caused by periodontitis pathogens leads to an increase in the level of inflammatory markers, which can negatively affect the state of the cardiovascular system. In patients with coronary heart disease, special attention is paid to controlling the level of inflammation and the general condition of the body, which makes the treatment of periodontitis in such patients especially important. [4]

Severe forms of the disease affect more than 1 billion people [2]. When one patient has a combination of coronary heart disease, smoking, diabetes mellitus, overweight and chronic inflammation in the oral cavity, the clinical picture becomes not only more complicated, but also noticeably less predictable.

It is noteworthy that the relationship between periodontitis and coronary heart disease is no longer being discussed as an exotic hypothesis, but as a direction with sufficient biological plausibility. Through an inflamed periodontal

wound, bacteria and their components can enter the systemic bloodstream, support the synthesis of pro-inflammatory mediators, shift the coagulation balance, and enhance endothelial dysfunction [4, 5]. Against this background, CRP, IL-6, TNF-α, adhesion molecules, and vasoreactivity indices are considered as intermediate markers through which dental intervention can theoretically affect cardiological risk [4, 7].
Nevertheless, the clinical literature does not look quite even. Some studies report a decrease in CRP, fibrinogen, leukocytes, and part of lipid fractions after scaling and routine planning, while others record only a local dental effect, and vascular parameters either change slightly or do not change at all [6-8].
From a practical point of view, this creates a simple but unpleasant question. What exactly does a cardiological patient get after periodontal treatment: only a reduction in bleeding gums, or also a measurable systemic gain, even if moderate? As the analysis of the work shows, the answer depends on which outcome to take as a basis, how pronounced the initial inflammation is, at what stage of the course of coronary heart disease the intervention is performed and how short the subsequent control was [1]. The purpose of this review was to systematize these data with a focus on clinically applicable results.

MATERIALS AND METHODS OF RESEARCH

The inclusion criteria were as follows: adult patients; confirmed coronary heart disease, coronary artery disease, or recent myocardial infarction; presence of chronic periodontitis/periodontitis; interventional study design; mandatory assessment of local periodontal indices and at least one systemic indicator - CRP, IL-6, TNF-alpha, fibrinogen, leukocytes, lipid profile, blood pressure, endothelial function or clinical cardiovascular events. Work without intervention, research protocols, reviews, materials without numerical results, and publications where the population with coronary artery disease did not stand out from the mixed sample were excluded.
Due to the pronounced heterogeneity of interventions and time points, no meta-analysis was performed; qualitative synthesis was used with elements of frequency accounting of favorable effects by groups of endpoints. For practical interpretation, local periodontal results, systemic inflammation, vascular surrogates, and lipid-hemodynamic parameters were separately distinguished.
The study focused on the type of group distribution, the completeness of follow-up, the objectivity of laboratory and instrumental endpoints, as well as the risk of confusion due to smoking, diabetes, drug therapy, and varying severity of the initial inflammation. This technique does not replace the formal scale of the risk of systematic error, but it is convenient for an educational article: you can immediately see where the effect is based on an array of data, and where it is more likely to be a preliminary signal.

RESULTS AND DISCUSSION

The qualitative synthesis included 7 interventional studies, with a total of 725 patients. Five studies dealt with stable coronary heart disease or stable coronary artery disease, one with patients with recent STEMI, and another with patients with atherosclerotic cardiovascular pathology and concomitant periodontitis. The follow-up period ranged from 6 weeks to 6 months. In most publications, non-surgical therapy was used: occupational hygiene, oral hygiene instruction, subgingival removal of dental deposits, and scaling and routine planning.
Treatment of periodontitis in patients with coronary heart disease requires an integrated approach that takes into account both local and systemic inflammatory factors. Patients with coronary heart disease are characterized by a more severe course of periodontitis and lower effectiveness of standard treatment methods, which is associated with the presence of chronic systemic inflammation. To improve the results of periodontitis treatment in such patients, close cooperation between dentists and cardiologists is necessary to optimize both local and general therapy aimed at controlling inflammatory processes in the body.
In all included studies, periodontal therapy produced a pronounced local effect. Bleeding during probing, pocket depth, and attachment rates decreased either significantly or very noticeably in absolute terms [8]. This is the most reproducible result of the review. It is repeated both in studies with stable coronary artery disease and in studies in patients after an acute coronary event.
In patients after STEMI, after 6 months in the treatment group, the proportion of teeth with a pocket depth of ≥ 4 mm decreased from 75.0% to 11.0%, the proportion of areas with a loss of attachment of ≥ 4 mm - from 90.0% to 50.0%, bleeding during probing - from 79.0% to 17.0%, visible plaque - from 77.0% to 12.0%. In shorter studies, the same vector was obtained without such detailed percentages, which is probably due not to the lack of effect, but to the format of the presentation of the results.
From the perspective of daily clinical work, this is not a small thing. If the inflammatory load in the oral cavity really decreases so consistently, this is where the basic effect that the doctor can almost certainly expect is located. All subsequent system changes are already layered on this level, and do not arise instead of it.

Table 1 - Key quantitative changes after periodontal therapy

Local effect	Systemic/vascular effect	Practical reading
BOP/PD/CAL improved	CRP >3 mg/l: -38,0% vs +4,0%; ARR 12,5%; fibrinogen ↓; WBC↓	The strongest anti-inflammatory signal
Local indexes have improved	TNF-α↓; IL-6↓; CRP↓	Confirms cytokine response

A pronounced local response	IL-6: 2,8±4,4 → 1,7±2,5; hs-CRP: 2,4±2,2 → 1,4±1,2	Quick effect in 6 weeks
Periodontal status has improved	САД -7,1 мм рт. ст.; VLDL -5,16 мг/дл; hs-CRP без значимого снижения	Spot cardiometabolic effect
Periodontal parameters have improved	ΔFMD -0,3% vs -0,1%; there is no clear gain; vascular markers are more stable	Neutral FMD with stable coronary heart disease
Periodontal indicators are better in the test group	With CRP ≥3 mg/l: 11.3±12.8 → 5.7±4.1; IL-6 and IL-8 are lower than in the control	The payoff is stronger with a high CRP
PD ≥4 mm: 75.0% → 11.0%; BOP: 79.0% → 17.0%	FMD: 9.0±4.4 → 12.1±5.6; intergroup Δ 3.4%; markers of inflammation without differences	The best vascular signal after STEMI

The table contains indicators that provide the doctor with applied information: where the effect was distinct, where it was moderate, and where it remained neutral.

Systemic inflammatory markers reacted less uniformly, but they were much more interesting from a cardiological point of view. In patients with angiographically confirmed coronary artery disease after non-surgical therapy, the number of people with CRP >3 mg/l in the intervention group decreased by 38.0%, while in the control group it increased by 4.0%; the absolute risk reduction was 12.5% [8]. At the same time, fibrinogen and white blood cell count decreased. As the analysis of the works shows, it is this publication that still remains the main argument in favor of the systemic anti-inflammatory effect of periodontal therapy in coronary patients.

TNF-α, IL-6, and CRP decreased in patients with stable coronary heart disease after 3 months of follow-up [9]. In a controlled study by G. Mariotti et al. 6 weeks after full-volume periodontal therapy, IL-6 decreased from 2.8±4.4 to 1.7±2.5 ng/L, and hs-CRP decreased from 2.4±2.2 to 1.4±1.2 mg/l [10]. Here the sample is small and the distribution by subgroups is not ideal, but the direction of the changes coincides with stronger work.

In the overall assessment, the differences between the groups in terms of biomarkers were not so sharp, however, in the subgroup of patients with baseline CRP >3 mg/l in the test group, the indicator decreased from 11.3±12.8 to 5.7±4.1 mg/l. In addition, after 3 months, the levels of IL-6 and IL-8 in the treatment group were lower than in the control group. Comparing these data with the Bokhari results suggests that patients with an initially more pronounced inflammatory background have the greatest chance of seeing a systemic benefit.

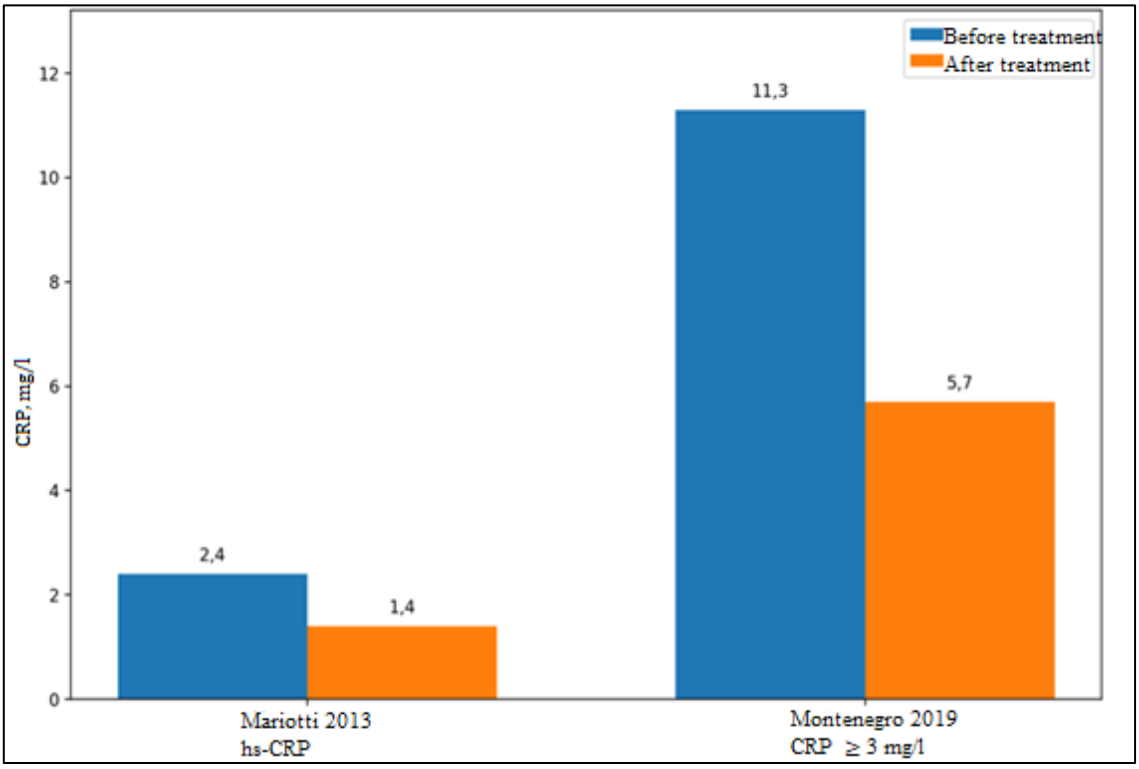

Figure 1 - Examples of CRP reduction after periodontal therapy

Endothelial function turned out to be the outcome where the differences between the studies are particularly pronounced. In patients with stable coronary heart disease, after 3 months, periodontal therapy did not provide an advantage in blood flow-dependent brachial artery dilation (FMD): the change in FMD in the test group was -0.3%, in the control -0.1%, the intergroup difference was statistically insignificant [9]. At the same time, the periodontal condition improved in the intervention group, and the markers of vascular inflammation behaved slightly more calmly than in the control group.

A completely different pattern was obtained in patients after recent STEMI. In the treatment group, FMD increased from 9.0±4.4% to 12.1±5.6%, while in the control group it remained almost unchanged: 12.2±7.2% versus 11.9±4.0%. The intergroup difference in the change was 3.4% and reached statistical significance. Here we can say that the vascular response to periodontal therapy may depend not only on the procedure itself, but also on the clinical period of coronary heart disease. After an acute coronary event, endothelial reactivity appears to be more mobile, although this assumption needs to be confirmed in large samples.

The lipid profile and hemodynamics changed moderately; after SRP, a decrease in systolic blood pressure by 7.1 mmHg and a decrease in VLDL by 5.16 mg/dl were recorded, while there was no statistical significance for other clinical and biochemical parameters [11]. This leads to a simple practical idea: you should not expect a rapid correction of cholesterol in a patient with coronary heart disease from periodontal treatment.

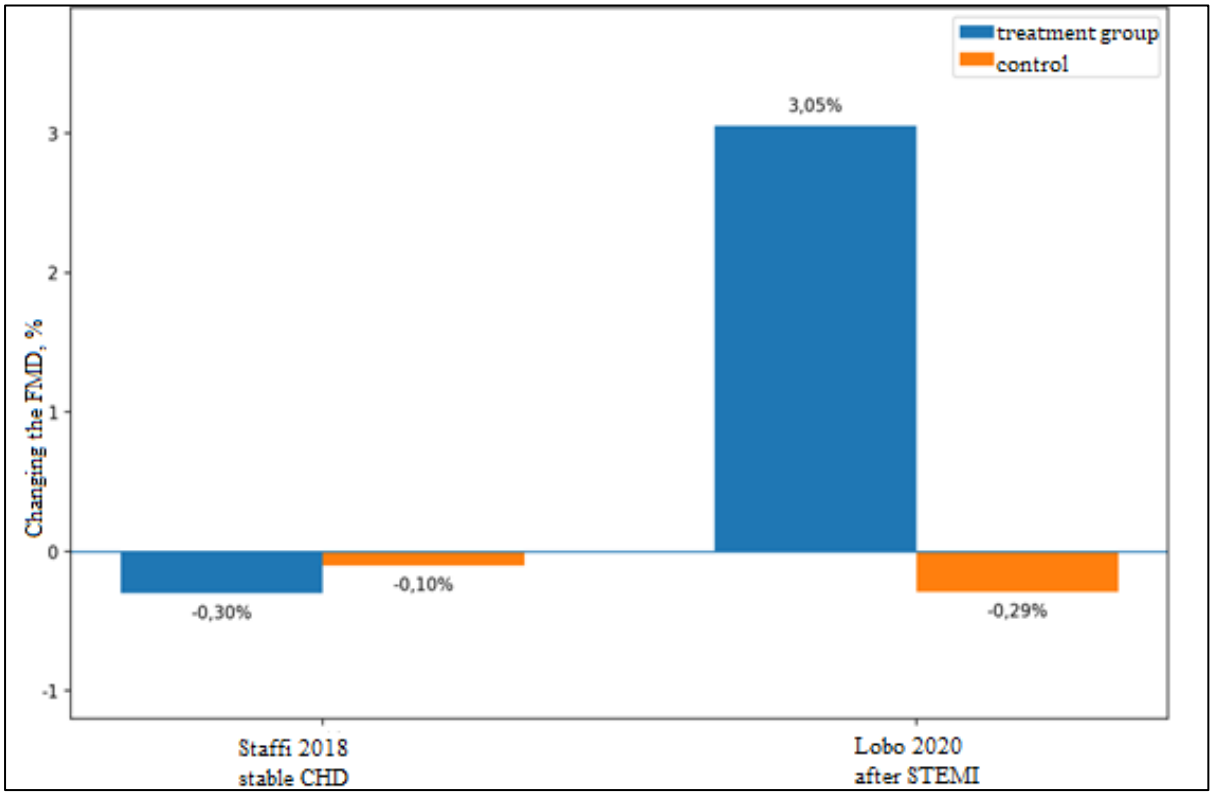

Figure 2 - FMD Change

With stable coronary heart disease, no statistically significant increase in FMD was recorded, whereas after recent STEMI, a pronounced positive shift was observed [11].

If you collect research data, you get a completely working scale of expectations. The local indexes of the oral cavity are in the first place: they change the fastest and most predictably. The next tier is CRP and a part of cytokines, where a positive shift occurs frequently, but not in every observation scenario. FMD, blood pressure, and lipid fractions are even lower. Here, the effect either depends on the clinical situation, or remains selective.

For a doctor, this is more useful than an abstract phrase about the connection between the oral cavity and the heart. A patient with active periodontitis and coronary artery disease is highly likely to receive a reduction in bleeding, pocket depth, and bacterial load. A patient with an initially elevated CRP appears to have a greater chance of showing laboratory improvement. But it is still impossible to promise him a reduction in the frequency of recurrent heart attacks or a reliable drop in cholesterol based on available publications. This line of expectations looks honest and clinically convenient.

Table 2 - Practical matrix of the expected effect in a patient with coronary artery disease

Domain	Favorable signal	Numerical guideline	Practical interpretation	Stability
Local indexes	7 out of 7	Reduction of bleeding, PD and plaque in all jobs	This effect can be expected in almost every patient	High
CRP and cytokines	4 out of 6	CRP >3 mg/L: -38.0%; hs-CRP 2.4 → 1.4; CRP 11.3 → 5.7 in the subgroup	They are most likely to improve in patients with underlying inflammation.	Moderate
of FMD	1 out of 2	Saffi: neutral; Lobo: intergroup Δ FMD 3.4%	The effect is possible, but depends on the clinical period of coronary heart disease	Limited

Blood pressure and lipids	1 out of 3	Hada: SAD -7.1 mmHg; VLDL -5.16 mg/dl	It is not suitable as an independent treatment goal	Low
Tough events	0 out of 7	No power tests for heart attack/mortality were found	A reduction in events cannot be promised	Very low

The matrix helps to translate disparate publications into clinical language. The local dental effect and the reduction of the inflammatory load are at different levels of evidence. Local indices are almost always responsible, CRP and cytokines are often responsible, and FMD, blood pressure, lipids, and severe cardiovascular outcomes still give less stable results.

In order not to focus only on individual studies, the results of the primary synthesis were compared with the reviews of recent years. A systematic review by E. Roca-Millan et al., focused specifically on cardiovascular patients, showed that CRP decreased in 77.8% of the included studies, TNF-α - in 66.7%, IL-6 - in all studies where it was evaluated [6]. CAD-specific meta-analysis by C. Liu et al. In 619 patients, he confirmed a significant gain in CRP, but found no convincing differences in IL-6, FMD, and lipids [10]. A broader meta-analysis by R. Meng et al. In 2024, he again showed a decrease in CRP and IL-6, and also a moderate effect on systolic pressure, in the absence of a stable effect on FMD and lipid fractions [11].

The picture, in fact, is quite harmonious. The local dental effect is reliable. Systemic inflammation is often reduced, but not always. Vascular surrogates and lipid profile respond selectively. Hard endpoints such as repeated heart attack, stroke, and cardiovascular death are almost unexplored in designs capable of giving a firm conclusion. An updated statement from the American Heart Association dated 2026 confirms this: the link between periodontitis and atherosclerotic cardiovascular diseases is more convincingly supported than before, but the translation of this link

For real practice, this means the following. Periodontal therapy in patients with coronary heart disease can be considered as an addition to standard cardiological treatment if the goal is to rehabilitate the chronic focus of inflammation, reduce bleeding, reduce bacterial load and, in some patients, reduce systemic inflammatory markers. This cannot be substituted for lipid correction, antiplatelet therapy, blood pressure control and diabetes mellitus. But it is already difficult to consider periodontal treatment as a secondary cosmetic measure.

There is a separate issue of security. The included clinical trials did not report an excess of acute cardiovascular complications after non-surgical periodontal therapy [12]. The EFP/WHF consensus and clinical guidelines for dental interventions indicate that in most patients there is no need to cancel antiplatelet agents or anticoagulants in advance before typical dental procedures; the problem is usually solved by local hemostasis and competent planning. However, the early post-infarction period and recent stenting require particularly careful coordination with a cardiologist.

CONCLUSION

An analysis of studies with a total of 725 patients showed that non-surgical periodontal therapy in patients with coronary heart disease consistently improves local dental parameters - bleeding, pocket depth and the condition of periodontal attachment.

The most convincing systemic effect is associated with a decrease in CRP and some pro-inflammatory cytokines. The gain is more pronounced in patients with initially higher inflammatory activity.

Endothelial function, lipid profile, and blood pressure change less predictably. For FMD, the effect turned out to be different in stable coronary heart disease and after a recent myocardial infarction, and there is still insufficient evidence of a decrease in the frequency of recurrent cardiovascular events.

For the joint management of cardiological and dental patients, the practical conclusion is as follows: active periodontitis in a patient with coronary heart disease is better considered as a treatable inflammatory load, and periodontal therapy is a reasonable addition to standard secondary prevention, but not its replacement.

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