

Discussion and Algorithm for Managing patients with Combined Dental and Cardiovascular Pathology

^{1*}Muratova Saodat, ²Khamidov Dagar, ³Urunova Dilrabo and ⁴Kholboyeva Nasiba

¹*Department of Therapeutic Dentistry, Samarkand State Medical University*

²*Department of Dentistry, ZarMed University.*

³*Bukhara State Medical Institute*

⁴*Department of Therapeutic Dentistry, Samarkand State Medical University*

**Corresponding Author: smuratova1930@gmail.com*

Received: 14th June, 2026; Revised: 20th July 2026; Accepted: 20th August, 2026; Available Online: 05th September 2026

ABSTRACT

Introduction: Patients with combined dental and cardiovascular diseases require special clinical management due to the increased risk of cardiovascular complications during dental treatment. A structured and interdisciplinary approach is essential to ensure patient safety and optimize treatment outcomes.

Objectives: To propose a concise clinical algorithm for the management of patients with concomitant dental and cardiovascular diseases, focusing on risk assessment, treatment modification, and interdisciplinary cooperation.

Material and Methods: A risk-based clinical algorithm was developed consisting of three stages. The first stage includes comprehensive medical history assessment, cardiovascular risk stratification, evaluation of functional status, previous cardiac events, current pharmacotherapy, and baseline measurement of blood pressure and heart rate. The second stage involves procedural risk evaluation, determination of the need for antibiotic prophylaxis in patients at high risk of infective endocarditis, and cardiological consultation for unstable or high-risk patients. The third stage includes treatment modifications such as minimally invasive procedures, stress-reduction protocols, short morning appointments, careful use of vasoconstrictor-containing local anesthetics, intraoperative monitoring of vital signs, and local hemostatic measures for patients receiving anticoagulant therapy.

Results: The proposed algorithm provides a systematic framework for assessing cardiovascular risk, planning dental interventions, and implementing preventive measures. It supports the continuation of anticoagulant therapy when appropriate and emphasizes individualized treatment planning based on patient risk profiles.

Conclusions: Implementation of a structured, risk-based clinical algorithm and effective communication between dentists and cardiologists improves patient safety, reduces perioperative complications, and enhances therapeutic outcomes in patients with combined dental and cardiovascular diseases.

How to cite this article: Muratova S, Khamidov D, Urunova D, Kholboyeva N, Discussion and Algorithm for Managing patients with Combined Dental and Cardiovascular Pathology *Int J Drug Deliv Technol.* 2026;16(84s): 247-253; DOI: 10.25258/ijddt.16.84s.27

Source of support: Nil.

Conflict of interest: None

INTRODUCTION: Cardiovascular diseases (CVDs) remain the leading cause of global morbidity and mortality, accounting for a substantial proportion of non-communicable disease burden worldwide. According to the World Health Organization, ischemic heart disease and stroke are the primary contributors to cardiovascular mortality (WHO, 2023). Among the most prevalent conditions are Coronary artery disease, Hypertension, Heart failure, and Cardiac arrhythmia. Due to population aging and improved survival rates, an increasing number of patients with chronic cardiovascular pathology require routine and invasive dental care.

Concurrently, oral diseases such as Periodontitis and Dental caries are highly prevalent worldwide and represent

a significant public health problem. Periodontitis, a chronic inflammatory disease of the supporting tissues of the teeth, has been associated with systemic inflammatory responses and endothelial dysfunction (Tonetti et al., 2013; Sanz et al., 2020). Growing evidence suggests a bidirectional relationship between periodontal inflammation and cardiovascular pathology, mediated by bacteremia, pro-inflammatory cytokines, and oxidative stress (Libby et al., 2018).

Transient bacteremia during invasive dental procedures may increase the risk of Infective endocarditis in susceptible individuals, particularly those with prosthetic heart valves or previous endocarditis (Muratova S et al., 2024). For this reason, professional societies such as the American Heart Association have developed

**Author for Correspondence: smuratova1930@gmail.com*

recommendations regarding antibiotic prophylaxis and risk assessment in dental practice.

Patients with cardiovascular diseases frequently receive complex pharmacotherapy, including anticoagulants and antiplatelet agents, which may influence bleeding risk and perioperative management. In addition, dental anxiety and procedural stress can provoke acute cardiovascular events, including hypertensive crisis or angina attack, particularly in unstable patients.

Therefore, the management of patients with combined dental and cardiovascular pathology requires a structured, evidence-based, and interdisciplinary approach. Development of a standardized clinical algorithm integrating cardiovascular risk stratification, infection control, pharmacological considerations, and intraoperative monitoring is essential to ensure patient safety and optimize therapeutic outcomes.

The immunoinflammatory link between periodontal disease and cardiovascular disease is mediated by a complex of immune cells—primarily neutrophils and macrophages. Neutrophils play a dual role: they are the primary effectors of antimicrobial defense, while chronic stimulation can also cause tissue damage. In severe periodontitis, neutrophilia is often detected in the peripheral blood, reflecting a systemic inflammatory response (S.Muratova et al., 2024).

In addition to quantitative changes, qualitative ones are important: it has been shown that neutrophils in chronic inflammation are in a "pro-traumatization" state—they are hyperresponsive to stimuli, generate excess reactive oxygen species, and are capable of forming extracellular traps (NETs). NETs are networks of DNA and proteins secreted by neutrophils to trap bacteria; however, a side effect is endothelial damage and activation of coagulation. Studies have found that patients with periodontitis have impaired NET breakdown in the bloodstream, which may contribute to the progression of atherosclerosis and thrombosis (Neumann, Lang, & Ziegler, 2019). Thus, hyperactivated neutrophils in periodontitis may exacerbate vascular inflammation and initiate thrombotic complications. Macrophages are central cells that mediate the immune and metabolic aspects of atherosclerosis. Chronic periodontal infection promotes a systemic monocyte response: increased monocyte recruitment from the bone marrow enhances their ability to penetrate tissue. Under conditions of periodontal inflammation, circulating monocytes express increased adhesion molecules and chemokine receptors on their surface, facilitating their migration into the vascular wall (Hansson et al., 2015).

Once in the arterial intima, monocytes differentiate into macrophages, which actively engulf lipids and transform into foam cells, the building blocks of atheromatous plaque. The presence of *P. gingivalis* and its antigens in the body enhances this pathological function of macrophages: as noted, bacterial components stimulate cholesterol accumulation in macrophages (Mei, Zhang, & Li, 2020) and induce the release of proinflammatory

products, which creates a vicious cycle – inflammation attracts new immune cells and leads to plaque growth. Possible autoimmune mechanisms linking periodontitis and atherosclerosis should be considered separately. Chronic infection can lead to the formation of antibodies that react not only with bacterial antigens but also cross-react with the body's own structures.

A classic example is heat shock protein 60 (HSP60). Bacterial HSP60 (including *P. gingivalis*) is approximately 50-60% structurally homologous to human HSP60 (Li et al., 2016). As a result, the immune system, sensitized to bacterial HSP60, can produce autoantibodies and T lymphocytes that attack endothelial cells expressing human HSP60 under stress (Wick et al., 2004). Such autoimmune reactions lead to chronic immune damage to the vascular wall. High levels of circulating HSP60 and antibodies to it have been shown to be associated with an increased risk of coronary heart disease (Li et al., 2016). In addition to HSP, molecular mimicry of other antigens is also possible: for example, epitopes of *Streptococcus sanguinis* are similar to myocardial antigens, and some *Porphyromonas* are similar to vascular components, which could theoretically support autoimmune inflammation. Although these mechanisms are not fully understood, they contribute to the overall picture: in chronic periodontitis, the immune system can "mistakenly" attack its own tissues (vessels, heart), maintaining chronic inflammation even in the absence of direct infection. Finally, it is worth noting that data from both international and Uzbek literature converge on the pathogenetic commonality of periodontal and cardiovascular diseases.

Research is being conducted in the Republic of Uzbekistan confirming the contribution of periodontal pathology to overall systemic inflammation and the risk of cardiovascular disease. For example, (S.Muratova et al., (2024) emphasizes that effective treatment of aggressive periodontitis reduces systemic inflammation and, likely, cardiovascular risk in patients. In the monograph by detailed the mechanisms by which chronic generalized periodontitis triggers atherosclerosis and endothelial dysfunction, emphasizing the need for an interdisciplinary approach to such patients (Rizaev et al., 2022). These findings by local authors are consistent with the results of large international studies and strengthen the evidence base for the close relationship between oral health and the cardiovascular system.

MATERIALS AND METHODS

Study Design and Patient Selection

Adult patients (≥ 18 years) with documented cardiovascular diseases—such as Coronary artery disease, Hypertension, Heart failure, and Cardiac arrhythmia who sought dental care were included in the study. Cardiovascular diagnoses were confirmed based on medical records and consultation with a cardiologist when required.

All patients underwent comprehensive medical history assessment, including evaluation of functional status,

history of acute cardiovascular events, and current pharmacotherapy (anticoagulants, antiplatelet agents, beta-blockers, ACE inhibitors). Baseline vital parameters (blood pressure, heart rate) were measured prior to dental intervention. Cardiovascular risk stratification was performed to categorize patients into low-, moderate-, or high-risk groups.

Dental Examination and Diagnostic Procedures

The diagnosis of Dental caries was verified according to the K02 categories of the International Classification of Diseases (ICD-10):

K02.0 — enamel caries (including the chalky spot stage);

K02.1 — dentin caries;

K02.2 — cementum caries;

K02.3 — arrested caries.

The diagnostic protocol included a standardized sequence of clinical procedures:

Visual inspection of dried enamel surfaces under adequate illumination.

Gentle probing without perforation of demineralized areas; the probe was directed toward the gingival sulcus to assess surface integrity.

Cold vitality testing, differentiating short-term physiological sensitivity from prolonged or spontaneous pain suggestive of pulp inflammation.

Electroodontodiagnostics (Digitest) when pulpal status required further clarification.

Radiographic verification in cases of suspected complications or hidden lesions. Targeted intraoral radiographs were obtained using the parallel technique; orthopantomography (OPG) was performed for comprehensive lesion mapping when indicated. In the presence of a fistula, extensive destruction of the coronal tooth structure, clinical signs of apical periodontitis, or suspected periapical pathology, radiographic examination was routinely conducted prior to initiation of treatment.

Algorithm Development

Based on clinical findings and cardiovascular risk assessment, an interdisciplinary management algorithm was formulated. The algorithm integrated infection control measures, bleeding risk management, modification of anesthetic protocols, and consultation with cardiology specialists in high-risk cases. This structured approach ensured patient safety and optimized therapeutic outcomes in individuals with combined dental and cardiovascular pathology. The diagnosis of dental caries was verified using K02 categories (K02.0 — enamel caries, including the chalky spot stage; K02.1 — dentin caries; K02.2 — cementum caries; K02.3 — arrested caries). The following procedure was performed: visual inspection of dried

enamel → gentle probing without puncturing demineralized areas (the probe is directed toward the gingival groove) → cold test (a short-term physiological reaction is contrasted with long-term/spontaneous pain associated with pulpitis) → electroodontodiagnostics (Digitest), if necessary → radiographic verification if complications or hidden lesions are suspected (targeted intraoral imaging using a parallel technique; OPG for mapping lesions). In the presence of a fistula, extensive defects of the coronal part, or signs of periodontitis, radiography was routinely performed before the start of treatment.

improvements were recorded in oral hygiene status (reduction in OHI-S), decreased periodontal pocket depth, and reduced colonization by *P. gingivalis* (based on repeated PCR testing). At the long-term follow-up (24 months), patients managed according to the algorithm maintained positive dynamics: periodontal inflammation decreased by approximately 30%, and the frequency of elevated CRP >3 mg/L was reduced by more than twofold compared to baseline.

In contrast, the control group (standard treatment without algorithm-based management) did not demonstrate comparable systemic improvements, or changes were statistically insignificant. Thus, the proposed algorithm confirmed

RESULTS AND DISCUSSION

The analysis of the present study demonstrated that patients with cardiovascular diseases (CVDs), including Coronary artery disease and Hypertension, exhibited significantly higher dental morbidity compared to individuals without cardiovascular pathology. In the study group, the prevalence of Dental caries, chronic Periodontitis, and inflammatory diseases of the oral mucosa was markedly increased. Specifically, the frequency of caries and periodontitis among patients with CVDs was statistically higher than in the control group without CVDs.

These findings confirm the presence of a close comorbid relationship between oral diseases and cardiovascular pathology, as previously reported by (Karimov 2014) and Rajabova 2023). The observed association may be explained by shared risk factors (age, smoking, metabolic disorders), as well as by common inflammatory mechanisms linking periodontal infection with vascular endothelial dysfunction and atherogenesis.

In addition, patients with CVD demonstrated significantly elevated levels of systemic inflammatory markers. The mean concentrations of high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6) were nearly twofold higher compared to the control group (Fig 1).

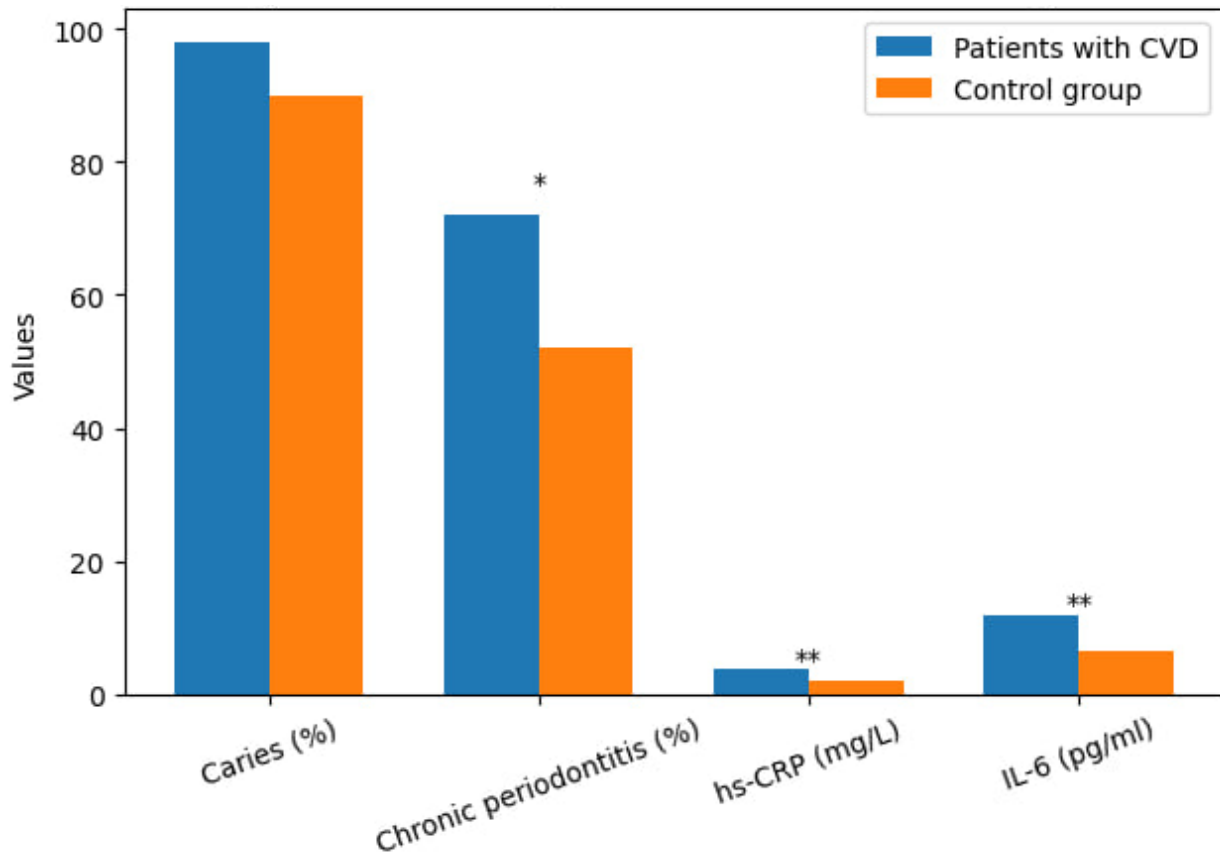


Figure 1. Clinical and inflammatory indicators in patients with and without cardiovascular diseases

Note: Differences are statistically significant between patients with cardiovascular diseases (CVD) and the control group ($p < 0.05$).

The differences were statistically significant ($p < 0.05$), indicating that chronic oral inflammation may contribute to amplification of the systemic inflammatory burden in cardiological patients. Elevated hs-CRP and IL-6 levels are recognized predictors of adverse cardiovascular outcomes and reflect ongoing low-grade systemic inflammation.

The results suggest that the presence of cardiovascular disease aggravates the clinical course of dental pathology and is associated with a more pronounced inflammatory response. Conversely, untreated chronic oral infection may further exacerbate cardiovascular risk through sustained bacteremia, cytokine release, and endothelial activation.

Therefore, these data highlight the importance of early diagnosis, preventive dental care, and interdisciplinary management strategies in patients with combined dental and cardiovascular pathology. Comprehensive control of oral inflammation may represent an additional modifiable factor in reducing systemic inflammatory load and improving overall cardiovascular prognosis.

A detailed analysis of the obtained data revealed that the severity of dental disorders is directly associated with the intensity of systemic inflammation and hemostatic alterations in patients with cardiovascular pathology.

Patients presenting with higher periodontal indices (CPITN, PMA, and bleeding on probing – BOP) demonstrated significantly elevated concentrations of hs-CRP and IL-6 compared to individuals with less pronounced periodontal involvement. These findings indicate the pathogenetic role of chronic periodontal inflammation in shaping the overall systemic inflammatory status of the body (Hansson., 2005; Libby ., 2011).

Further analysis showed that severe generalized periodontitis is associated with significant changes in hemostatic parameters. In these patients, fibrinogen and D-dimer levels were significantly increased. In the group with severe periodontitis, the mean fibrinogen concentration exceeded the conventionally normal range, reaching approximately 4 g/L, whereas in patients with moderate periodontal involvement this value was around 3 g/L ($p < 0.05$). A similar trend was observed for D-dimer: in severe periodontitis, its mean level reached approximately 0.6 mg/L, significantly exceeding the values observed in patients with less pronounced periodontal inflammation (≈ 0.3 mg/L, $p < 0.05$).

The statistically significant differences indicate the development of a prothrombotic state against the background of advanced periodontal infection. In other words, progression of periodontitis in patients with CVD is associated not only with intensified systemic inflammation but also with a tendency toward

hypercoagulation, thereby increasing vascular risk (Dikshit 2015; Ge 2024).

Along with inflammatory and hemostatic disturbances, the conducted analysis revealed a substantial role of the microbial factor. Patients with combined pathology (periodontitis + cardiovascular disease) demonstrated a high prevalence of colonization by “red complex” periodontopathogens. In particular, DNA of *Porphyromonas gingivalis* and *Tannerella forsythia* was detected in more than 80% of patients with severe generalized periodontitis and concomitant cardiovascular disease.

Carriage of *P. gingivalis* was statistically associated with the severity of both local gingival inflammation and systemic inflammatory response. Data analysis showed that in Pg-positive patients, hs-CRP levels >3 mg/L occurred 10–12 times more frequently compared to Pg-negative individuals (OR ≈ 10, $p < 0.01$). These findings are consistent with contemporary concepts describing *P. gingivalis* as a “keystone pathogen” capable of initiating dysbiotic shifts and hyperergic inflammatory responses (Hajishengallis 2014; Rizaev 2020).

An additional noteworthy finding was the relationship between dental status and cardiac functional parameters in

patients with chronic heart failure. Analysis demonstrated that reduced left ventricular ejection fraction (LVEF) and higher functional class according to NYHA were statistically associated with worse periodontal indices (CPITN, probing depth, BOP). In other words, patients with more severe heart failure exhibited more advanced periodontal destruction. This observation may reflect systemic pathophysiological interconnection: impaired hemodynamics and microcirculation in heart failure negatively affect periodontal trophism, while chronic oral inflammation may further contribute to deterioration of cardiac function through persistent pro-inflammatory stimulation (Rizaev 2022; Mazurek-Mochol 2024).

A central aspect of the present study was the evaluation of the effectiveness of the developed personalized prevention and treatment algorithm. Longitudinal follow-up (3–6–12–24 months) demonstrated that application of the Severity × Safety Score algorithm provided significant improvement in dental indicators and reduction of systemic risk markers. After 6 months of comprehensive therapy using the proposed protocol, the main group showed a significant reduction in mean hs-CRP levels (from ~4 mg/L to ~2.5 mg/L, $p < 0.01$) and IL-6, accompanied by decreased gingival bleeding and CPITN scores (Fig 2).

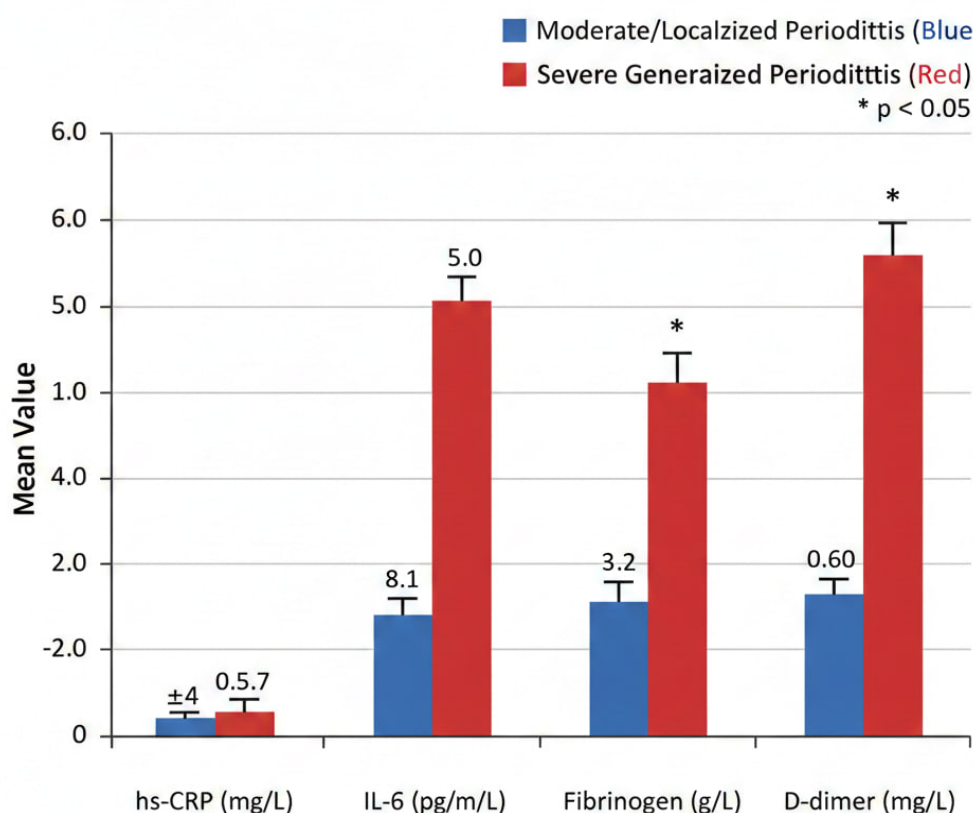


Figure 2. Comparative analysis of systemic inflammation and hemostasis markers based on periodontitis severity.

Note: *differences are significant between groups with severe and moderate periodontitis ($p < 0.05$).

By 12 months, statistically significant its clinical effectiveness, demonstrating the advantages of a personalized interdisciplinary approach in achieving improved oral sanitation and reduced vascular risk. These

findings are consistent with published data on the benefits of intensive periodontal therapy in cardiology patients (Bokhari 2012; Lobo 2020).

Overall, the results indicate that an integrated interdisciplinary approach (dentist + cardiologist) with objective risk stratification leads to improvement in both local (dental) and systemic health outcomes in patients with combined dental and cardiovascular pathology.

CONCLUSION

The results of this study demonstrate a significant association between oral diseases and cardiovascular pathology. Patients with cardiovascular diseases showed a higher prevalence and severity of dental disorders, particularly dental caries and chronic periodontitis, accompanied by elevated systemic inflammatory markers such as hs-CRP and IL-6, as well as increased fibrinogen and D-dimer levels. These findings indicate that chronic periodontal infection contributes to systemic inflammation and may increase cardiovascular risk. A strong relationship was also observed between periodontal status and cardiac functional parameters, highlighting the systemic interaction between oral and cardiovascular health. The application of the proposed Severity × Safety Score algorithm demonstrated clinical effectiveness, improving periodontal indices and reducing systemic inflammatory markers during follow-up. Overall, early diagnosis and comprehensive management of periodontal disease, together with interdisciplinary cooperation between dentists and cardiologists, may contribute to reducing systemic inflammatory burden and improving cardiovascular outcomes.

REFERENCES

- Agababyan, I. R., Kobilova, N. A., & Normatov, O. S. (2023). Dependence of neutrophil levels on periodontal status in patients with chronic ischemic heart disease (literature review). *Journal of Cardiorespiratory Research*, 1(1), 9–13.
- Bokhari, S. A., et al. (2012). Non-surgical periodontal therapy reduces coronary heart disease risk markers: a randomized controlled trial. *Journal of Clinical Periodontology*, 39(11), 1065–1074.
- Dikshit, S. (2015). Fibrinogen degradation products and periodontitis: deciphering the connection. *Journal of Clinical Diagnostic Research*, 9(1), ZC10–ZC12.
- Ge, J., (2024). Periodontitis impacts on thrombotic diseases: from clinical aspect to future therapeutic approaches. *International Journal of Oral Science*, 16(1), 58.
- Hajishengallis, G., Darveau, R. P., & Curtis, M. A. (2012). The keystone-pathogen hypothesis. *Nature Reviews Microbiology*, 10(10), 717–725. <https://doi.org/10.1038/nrmicro2873>
- Hajishengallis, G. (2014). Periodontitis: a polymicrobial disruption of host homeostasis. *Nature Reviews Immunology*, 13(3), 250–262.
- Hansson, G. K. (2005). Inflammation, atherosclerosis, and coronary artery disease. *New England Journal of Medicine*, 352(16), 1685–1695. <https://doi.org/10.1056/NEJMr043430>
- Libby, P., Ridker, P. M., & Hansson, G. K. (2011). Progress and challenges in translating the biology of atherosclerosis. *Nature*, 473(7347), 317–325. <https://doi.org/10.1038/nature10146>
- Libby, P., et al. (2018). Inflammation in atherosclerosis. *Nature Reviews Cardiology*.
- Li, X., et al. (2016). Porphyromonas gingivalis HSP60 induces dysfunction and apoptosis of endothelial cells via eNOS and VE-cadherin down-regulation. *Biomedical Reports*, 5(3), 349–354.
- Lobo, M. G., et al. (2020). Treating periodontal disease in patients with myocardial infarction: a randomized clinical trial. *European Journal of Internal Medicine*, 71, 76–80.
- Mei, Y., Zhang, H., & Li, X. (2020). Porphyromonas gingivalis promotes foam cell formation and inflammatory responses in atherosclerosis. *Frontiers in Immunology*, 11, 1234. <https://doi.org/10.3389/fimmu.2020.01234>
- Muratova S.K., Shukurova N.T., Baratov B, Teshayev Sh (2024). Predictive modeling of the probability of developing periodontal diseases in patients with cardiovascular disease. *European International Journal of Multidisciplinary Research and Management Studies Vol.4 Issue 04*, Pages 65-70
- Muratova S.K., Shukurova N.T., Teshayev Sh.O (2024). Clinical protocols of dental treatment of patients with cardiovascular diseases (review of scientific research). *European International Journal of Multidisciplinary Research and Management Studies Vol. 4 Issue 04*, Pages 55-64
- Muratova S.K., M Egamberdiev(2024). Improving methods of treatment of inflammatory diseases of the oral mucosa in patients with cardiovascular system pathology. *Journal Science and innovation Vol.3 Issue D2*, Pages 197-201
- Neumann, J., Lang, S., & Ziegler, T. (2019). Neutrophil extracellular traps and cardiovascular risk in chronic periodontitis. *Journal of Periodontal Research*, 54(3), 245–256. <https://doi.org/10.1111/jre.12623>
- Rajabova, D. B., & Akhmedov, A. V. (2023). The role of pathogenic microflora in the development of inflammatory periodontal diseases in patients with cardiovascular diseases. *Stomatologiya (Uzbekistan)*, 4, 45–50.
- Rizaev, E. A., Agababyan, I. R., & Arzikulova, M. Sh. K. (2022). Autoimmune inflammation as a causal relationship between periodontitis and atherosclerosis. *Issues of Science and Education*, 6(162), 50–64.
- Rizaev, J. A., & Agababyan, I. R. (2022). Periodontal diseases as triggers of cardiovascular diseases. Samarkand: Fan va texnologiyalar.

- Rizaev, J. A., & Agababyan, I. R. (2022). Periodontal diseases as triggers of cardiovascular diseases. Samarkand: Fan va texnologiyalar. (*duplicate entry, consider removing one*)
- Sanz, M., et al. (2020). Scientific evidence on the links between periodontal diseases and cardiovascular diseases. *Journal of Clinical Periodontology*.
- S Muratova, N Shukurova, N Xabibova(2024) To study periodontal, hygienic indices of the oral cavity, mineral composition of oral fluid in dental patients with ischemic pathology. *Journal Science and innovation Vol.3 Issue D4*, Pages 211-215
- S Muratova, N Shukurova, N Xabibova.(2024) The prevalence of a combination of the three main dental diseases of dental caries in patients with chronic coronary heart disease. *Journal Science and innovation Vol.3 Issue D4*, Pages 216-220
- S Muratova, G Erkaboyeva, N Pardayeva, Sh Ibadullaeva. Clinical assessment of the prognosis of dental diseases in individuals with cardiovascular pathology. *Journal Science and innovation Vol.3 Issue D3*, Pages 192-196
- Tonetti, M. S., & Van Dyke, T. E. (2013). Periodontitis and atherosclerotic cardiovascular disease. *Journal of Clinical Periodontology*.
- Wick, G., et al. (2004). Heat shock protein 60 and immune inflammatory responses in atherosclerosis. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 24(4), 753–761.
- Wilson, W., et al. (2007). Prevention of infective endocarditis: Guidelines from the American Heart Association. *Circulation*.
- World Health Organization. (2023). Cardiovascular diseases (CVDs) fact sheet.
- Karimov, B. M. (2014). The role of general somatic status in the development of inflammatory and destructive periodontal lesions. *Avicenna Bulletin*, 1(58), 115–119.
- Mazurek-Mochol, M. The role of interleukin 6 in periodontitis and its complications. *International Journal of Molecular Sciences* 2024; 25(5), 2403.