Clinical analysis of dental status in patients with mitral valve disease

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Abstract

Objective. To assess oral and periodontal status in patients with mitral valve disease, identify dental risk factors associated with cardiac decompensation, and evaluate the clinical utility of a stepwise dental triage algorithm. Materials and methods. The study included 138 participants: 42 controls, 48 patients with compensated mitral valve disease, and 48 patients with decompensated mitral valve disease. The assessment included DMFT, OHI-S, SBI, CPI, probing pocket depth (PPD), clinical attachment loss (CAL), number of retained teeth, xerostomia score, unstimulated salivary flow, and odontogenic infectious foci. Results. The decompensated group had significantly higher OHI-S, CPI, PPD, and CAL values and lower salivary flow; the need for preoperative dental clearance was recorded in 87.5% of patients. NYHA functional class correlated directly with OHI-S, CPI, PPD, and CAL. An integrated model for identifying high oral infectious risk yielded an AUC of 0.891. Conclusion. In mitral valve disease, dental status is closely associated with the severity of heart failure; early dental screening and staged oral sanitation are important components of clinical management.

Keywords: *mitral valve disease; dental status; periodontal status; DMFT; OHI-S; CPI; odontogenic infection; oral sanitation*

Introduction

Oral health in patients with mitral valve disease has long remained peripheral to the cardiology care pathway. In practice, attention has almost always focused on hemodynamics, cardiac rhythm, anticoagulant therapy, and the timing of surgery. Oral examination was often deferred until a late stage, by which time some patients already had chronic foci of infection, heavy plaque accumulation, subgingival calculus, gingival inflammation, and periodontal attachment loss. In a cardiac patient, these findings are not incidental; they may contribute to an unfavorable clinical course, particularly before an invasive procedure or in the setting of chronic heart failure [1-3].

Mitral valve disease, whether stenosis or regurgitation predominates, is associated with progressive remodeling of intracardiac hemodynamics. Left atrial enlargement, atrial fibrillation, increasing NYHA functional class, long-term pharmacotherapy, and reduced exercise tolerance alter patients' daily behavior. Against this background, adherence to oral hygiene declines, regular dental visits become less frequent, xerostomia worsens, and diuretics and other medications may further reduce salivary flow. The oral environment consequently becomes more vulnerable to inflammatory and destructive disease.

The periodontal component of dental status is of particular interest. Chronic inflammation of the periodontal tissues extends beyond local gingival changes. It is accompanied by impaired microcirculation, tissue hypoxia, enzymatic degradation of the extracellular matrix, increased vascular permeability, and a persistent bacterial burden. In a patient with mitral valve disease, this background is especially relevant because a chronic oral reservoir of infection may sustain systemic inflammatory activity and complicate preparation for cardiac surgery or catheter-based intervention [4-6].

Recent studies of patients with valvular disease increasingly address the quality of dental clearance before surgery and the actual burden of oral disease in this population [1,2,7]. Most publications, however, focus on the presence of odontogenic infection or the risk of infective endocarditis. Far fewer provide an integrated clinical and dental profile of a patient with mitral valve disease that simultaneously considers caries burden, oral hygiene, the severity of periodontal inflammation, salivary flow, symptoms, and the need for staged dental treatment.

This is insufficient for clinical practice. Cardiologists and dentists need not only a list of findings but also a clear care pathway. A brief, practical tool is needed to identify patients at high oral infectious risk during the initial examination, avoid delays caused by lower-priority procedures, and coordinate dental treatment with cardiac care. This approach is gradually being incorporated into international guidelines, which emphasize early identification of infectious foci, plaque control, and individualized preventive strategies [8,9].

This topic is also important in dentistry because mitral valve disease often requires years of follow-up. Patients may remain between clinical stability and a risk of decompensation for years, receive complex pharmacotherapy, and later prepare for an invasive intervention. Along this clinical pathway, dentists can play a more active role than is commonly assumed: not merely eliminating an isolated focus, but assessing the overall oral infectious burden, estimating the required scope of dental treatment, and establishing a supportive follow-up regimen.

The knowledge gap lies in the limited quantitative evidence linking the degree of cardiac decompensation with overall dental status in mitral valve disease. Even fewer studies combine cardiologic characteristics, periodontal indices, salivary flow, and the need for preoperative dental clearance in a single model. This gap is particularly evident in the regional literature, where reports are often limited to individual symptoms or isolated clinical observations.

The aim of this study was to analyze dental status in patients with mitral valve disease, assess the relationship between the severity of cardiac disease and oral parameters, and develop a practical algorithm for early identification of patients at high oral infectious risk. Additional objectives were to compare compensated and decompensated disease, evaluate the need for dental clearance, and construct a diagnostic and prognostic model for routine clinical triage.

Materials And Methods

Study Design

This single-center comparative observational study was conducted at the clinical facilities of the Tashkent State Dental Institute in collaboration with a specialized cardiology hospital. The study included 138 participants aged 35-73 years. The control group comprised 42 apparently healthy individuals without echocardiographic evidence of valvular disease. The main cohort comprised 96 patients with confirmed mitral valve disease, divided equally into compensated and decompensated groups of 48 patients each.

Inclusion criteria for the main cohort were echocardiographically confirmed mitral valve disease, age over 18 years, a stable clinical condition at the time of dental examination, and informed consent to participate. Patients were excluded if they had undergone recent cardiac surgery or had an acute infectious process, an oncohematologic disease, severe cognitive impairment, pregnancy, marked immunosuppression, or systemic antibiotic use within four weeks before examination.

The cardiologic assessment included characterization of the mitral valve lesion, determination of NYHA heart failure functional class, documentation of atrial fibrillation, review of anticoagulant therapy, and echocardiographic measurement of left atrial diameter. The dental examination followed a standardized protocol and recorded the number of retained teeth, the decayed, missing, and filled teeth index (DMFT), the simplified oral hygiene index (OHI-S), the sulcus bleeding index (SBI), the community periodontal index (CPI), mean probing pocket depth (PPD), and clinical attachment loss (CAL).

Clinical and Dental Variables

Additional assessments included complaints of oral dryness, gingival bleeding, and halitosis; the presence of mucosal petechiae or ecchymoses; the number of odontogenic infectious foci; and the need for specialized periodontal care. Xerostomia severity was recorded on a visual analogue scale. Unstimulated salivary flow was measured in the morning after abstention from food and fluids for a standardized period and was expressed in mL/min.

An integrated score for high oral infectious risk was developed for clinical triage. The model included NYHA functional class, OHI-S, DMFT, and salivary flow. The following formula was used in practice: risk score = 1.2 × NYHA + 1.0 × OHI-S + 0.15 × DMFT - 3.0 × salivary flow. A cutoff greater than 7.29 was considered an indication for priority oral sanitation and an expanded dental care pathway.

Statistical analyses were performed using Python and Excel. Means and standard deviations were calculated for quantitative variables. Intergroup differences were assessed using Student's t-test, the Mann-Whitney U test, or one-way analysis of variance (ANOVA), depending on the distribution and the number of groups compared. Categorical variables were analyzed using the chi-square test. Associations between parameters were assessed using Spearman's rank correlation coefficient. The diagnostic and prognostic performance of the models was evaluated by receiver operating characteristic (ROC) analysis with calculation of the area under the curve (AUC), sensitivity, and specificity. Statistical significance was set at $p < 0.05$.

Risk Stratification and Statistical Analysis

To improve reproducibility, two trained investigators recorded the clinical indices using a common protocol. Discrepant ratings were resolved by agreement after repeat examination. Dental examinations were performed under standard lighting with a periodontal probe, dental mirror, and suction. In patients receiving anticoagulants, invasive screening tests were avoided unless clinically necessary; the examination emphasized index-based assessment, identification of infectious foci, and risk stratification.

High oral infectious risk was defined as a combination of adverse quantitative and clinical findings: two or more chronic infectious foci, OHI-S greater than 2.0, salivary flow below 0.28 mL/min, CAL of at least 5 mm, PPD of at least 6 mm, or an integrated risk score above 7.29. This definition was used as a working screening filter rather than a final diagnosis, to identify patients in whom delayed oral sanitation could complicate the cardiac care pathway.

The study was conducted in accordance with Good Clinical Practice and the Declaration of Helsinki. Personal data were coded before statistical analysis. When the article layout was planned, particular attention was paid to clinical reproducibility: every measure was selected so that it could be obtained in a routine dental office without costly equipment or delays in cardiac care.

Results

The clinical and cardiologic characteristics of the study groups are presented in Table 1. The groups did not differ in age or sex, which simplified interpretation of the dental findings. As expected, however, they showed a severity gradient in disease-specific cardiologic parameters. Patients with decompensated mitral valve disease had a larger left atrial diameter and higher frequencies of atrial fibrillation and long-term anticoagulant therapy. Mean NYHA functional class was 3.11 ± 0.42 in this group, compared with 1.94 ± 0.40 in the compensated group.

The interview revealed a clear clinical gradient across the groups. Oral dryness was reported by 28.6% of controls, 58.3% of patients with compensated disease, and 83.3% of patients with decompensated disease. Halitosis also increased with the severity of cardiac disease. Gingival bleeding was especially frequent in the decompensated group and was accompanied by more frequent mucosal petechiae and ecchymoses, probably related both to the severity of inflammation and to anticoagulant therapy. These findings are summarized in Table 2.

Table 1. Clinical and cardiologic characteristics of the study groups

Variable	Control	Compensated MVD	Decompensated MVD	p
Participants, n	42	48	48	-
Age, years	52.75 ± 7.55	52.78 ± 6.42	54.34 ± 9.31	0.535
Women, n (%)	25 (59.5)	38 (79.2)	34 (70.8)	0.126
Left atrial diameter, mm	38.60 ± 3.25	47.06 ± 4.43	52.55 ± 6.11	<0.001
Atrial fibrillation, n (%)	3 (7.1)	10 (20.8)	16 (33.3)	0.010
Anticoagulant therapy, n (%)	0	25 (52.1)	40 (83.3)	<0.001
Duration of cardiac disease, years	-	7.32 ± 2.42	9.05 ± 2.64	<0.001
NYHA functional class	-	1.94 ± 0.40	3.11 ± 0.42	<0.001

Note. Quantitative variables are presented as mean ± SD and categorical variables as n (%). p values were calculated using ANOVA or the chi-square test. MVD, mitral valve disease; NYHA, heart failure functional class.

Quantitative measures of dental status showed a consistent adverse gradient from the control group to the decompensated group. The number of retained teeth decreased from 26.17 ± 2.67 to 19.69 ± 2.87, the DMFT index increased from 10.67 ± 2.92 to 19.08 ± 3.58, and xerostomia severity on the visual analogue scale almost tripled. Of particular importance, unstimulated salivary flow fell to 0.23 ± 0.05 mL/min in the decompensated group. Under conditions of hyposalivation, the natural cleansing and buffering functions of saliva are substantially reduced, creating a favorable environment for plaque accumulation and persistent inflammation. Detailed findings are presented in Table 3.

Table 2. Symptoms and clinical findings affecting the scope of dental clearance

Variable	Control	Compensated MVD	Decompensated MVD	p
Oral dryness, n (%)	12 (28.6)	28 (58.3)	40 (83.3)	<0.001
Halitosis, n (%)	11 (26.2)	25 (52.1)	33 (68.8)	<0.001
Gingival bleeding, n (%)	15 (35.7)	18 (37.5)	28 (58.3)	0.050
Mucosal petechiae/ecchymoses, n (%)	3 (7.1)	8 (16.7)	15 (31.2)	0.013
Odontogenic infectious foci, n	0.62 ± 0.73	1.71 ± 0.77	2.19 ± 1.10	<0.001
Need for preoperative dental clearance, n (%)	6 (14.3)	31 (64.6)	42 (87.5)	<0.001
High oral infectious risk, n (%)	0	7 (14.6)	41 (85.4)	<0.001

Note. Categorical variables are presented as n (%) and quantitative variables as mean ± SD. p values were calculated using the chi-square test or ANOVA. High oral infectious risk was defined by a combination of clinical red flags and the integrated risk score.

Table 3. Quantitative measures of dental status in the study groups

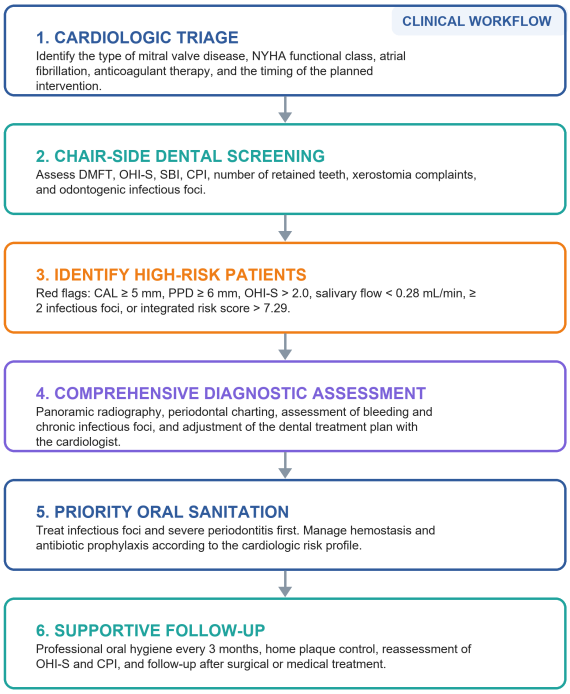
Variable	Control	Compensated MVD	Decompensated MVD	p
Retained teeth, n	26.17 ± 2.67	22.81 ± 2.66	19.69 ± 2.87	<0.001
DMFT index, score	10.67 ± 2.92	15.34 ± 3.31	19.08 ± 3.58	<0.001
OHI-S, score	1.02 ± 0.32	1.86 ± 0.35	2.38 ± 0.28	<0.001
Xerostomia severity, VAS score	2.04 ± 1.28	4.38 ± 1.27	6.02 ± 1.49	<0.001
Unstimulated salivary flow, mL/min	0.46 ± 0.08	0.31 ± 0.06	0.23 ± 0.05	<0.001

Note. Values are mean ± SD. The DMFT index is reported using the international DMFT notation. VAS, visual analogue scale; p values were calculated using ANOVA.

The periodontal findings showed an even steeper gradient. Mean SBI increased from 0.86 ± 0.36 in controls to 2.11 ± 0.48 in the decompensated group. CPI increased from 1.19 ± 0.48 to 3.02 ± 0.40. PPD and CAL changed in the same direction: probing pocket depth reached 4.55 ± 0.60 mm, and clinical attachment loss reached 5.24 ± 0.74 mm. Specialized periodontal treatment was required by 91.7% of patients in the decompensated group. OHI-S, SBI, and CPI are compared in Figure 2, and the progressive increase in PPD and CAL is shown in Figure 3.

The sequence of changes has an important clinical explanation. As cardiac disease became more severe, natural oral cleansing deteriorated, oral hygiene became more difficult to maintain, and chronic infectious foci became more frequent. Figure 1 presents a practical pathway based on this sequence. Cardiologic risk is assessed first, followed by chair-side dental screening; patients with red flags then proceed immediately to comprehensive assessment and priority oral sanitation. For routine practice, this pathway is useful because it combines index-based assessment and clinical decision-making in a single algorithm.

Figure 1. Stepwise clinical and dental pathway for patients with mitral valve disease



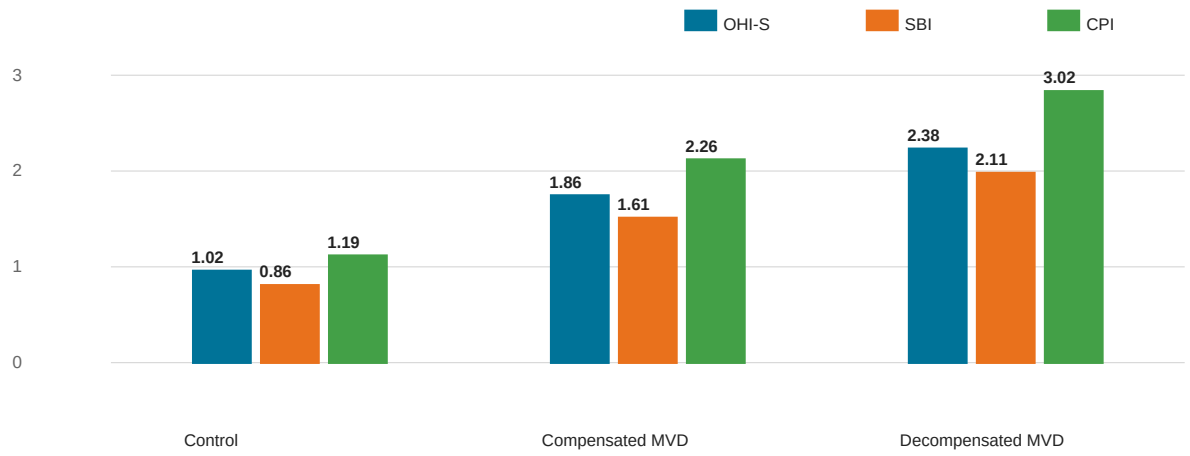
The algorithm combines cardiologic triage, chair-side dental screening, identification of red flags, comprehensive assessment, priority oral sanitation, and supportive follow-up. Its clinical purpose is to direct high-risk patients to active dental treatment early, before an invasive cardiac procedure.

Table 4. Periodontal indices and need for specialized care

Variable	Control	Compensated MVD	Decompensated MVD	p
SBI, score	0.86 ± 0.36	1.61 ± 0.44	2.11 ± 0.48	<0.001
CPI, score	1.19 ± 0.48	2.26 ± 0.46	3.02 ± 0.40	<0.001
PPD, mm	2.48 ± 0.52	3.66 ± 0.49	4.55 ± 0.60	<0.001
CAL, mm	1.95 ± 0.60	3.80 ± 0.64	5.24 ± 0.74	<0.001
Need for specialized periodontal treatment, n (%)	0	13 (27.1)	44 (91.7)	<0.001

Note. Values are mean ± SD or n (%). SBI, sulcus bleeding index; CPI, community periodontal index; PPD, probing pocket depth; CAL, clinical attachment loss; p values were calculated using ANOVA or the chi-square test.

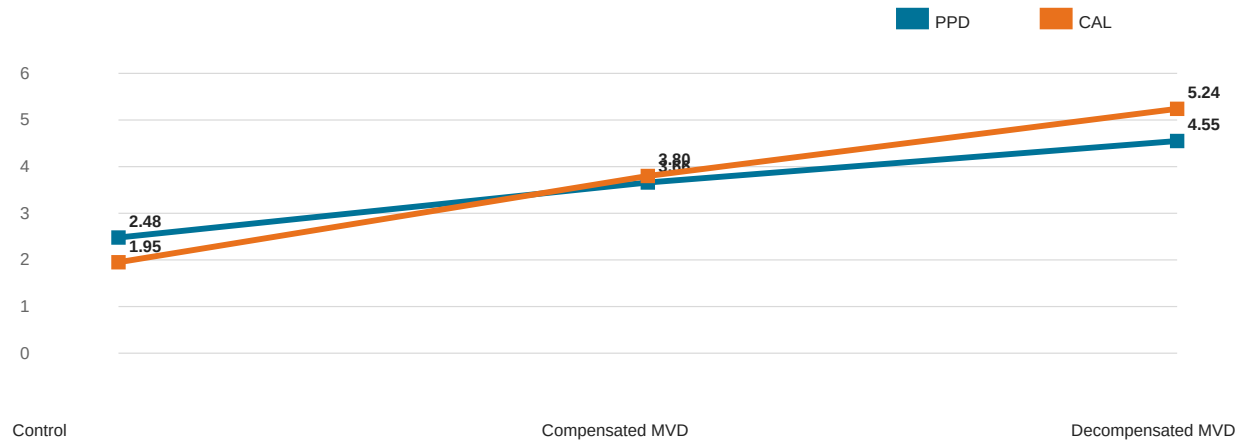
Figure 2. Comparison of OHI-S, SBI, and CPI across the study groups



The chart shows a progressive increase in oral hygiene burden and periodontal inflammation with increasing severity of mitral valve disease. The greatest increase was observed for CPI, consistent with the higher prevalence of periodontal lesions in the decompensated group.

Correlation analysis in the main cohort confirmed the close relationship between cardiologic and dental parameters. NYHA functional class correlated directly with OHI-S (rho = 0.512), CPI (rho = 0.513), PPD (rho = 0.555), CAL (rho = 0.640), and DMFT (rho = 0.453). NYHA functional class correlated inversely with salivary flow (rho = -0.442). Left atrial diameter also correlated with OHI-S, PPD, and CAL. These findings are summarized in Table 5 and indicate that increasing cardiac burden is associated not with a single isolated symptom, but with broad deterioration in oral health.

Figure 3. PPD and CAL according to clinical group



The line chart shows parallel increases in probing pocket depth and clinical attachment loss. Clinically, this indicates that decompensated mitral valve disease is associated not only with gingival inflammation but also with deeper structural periodontal damage.

ROC analysis was performed to evaluate the practical utility of the integrated approach. The risk score for identifying patients at high oral infectious risk yielded an AUC of 0.891, with 75.6% sensitivity and 86.3% specificity. NYHA functional class and DMFT also performed well individually, but both were less robust overall than the integrated model. OHI-S and salivary flow had more modest performance as stand-alone measures but strengthened the combined risk score. The ROC curve is shown in Figure 4, and the numerical results are presented in Table 6.

The distribution of the need for preoperative dental clearance was clinically important. Clearance was required by only 14.3% of controls, compared with 64.6% of patients with compensated mitral valve disease and 87.5% of those with decompensated disease. When two or more odontogenic foci, CAL of at least 5 mm, PPD of at least 6 mm, OHI-S greater than 2.0, and salivary flow below 0.28 mL/min were also considered, a group with the most unfavorable dental care pathway emerged. Delaying oral sanitation creates the greatest risk for these patients during the preoperative and early postoperative periods.

When dental status is considered as an integrated syndrome rather than as a set of individual indices, the differences between groups become even more pronounced. The compensated group already showed a clear shift from the control group: OHI-S almost

doubled, CPI increased to 2.26 ± 0.46, and the number of infectious foci was almost three times higher than in controls. These findings suggest that an unfavorable dental trajectory begins well before complete hemodynamic decompensation and that preventive intervention may be most timely at this stage.

The combination of anticoagulant therapy and marginal gingival inflammation warrants particular attention. In the decompensated group, in which 83.3% of patients received anticoagulants, mucosal petechiae and ecchymoses were more frequent, and complaints of gingival bleeding were more pronounced. Even after this factor was considered, SBI, CPI, PPD, and CAL remained substantially higher, supporting the primary role of chronic inflammatory and destructive periodontal disease rather than medication-related bleeding alone.

Together, the findings support three clinical levels of dental risk. The control group had acceptable oral hygiene and a low need for dental clearance. Compensated mitral valve disease represented an intermediate risk level, at which pathologic changes were already evident but preventive correction remained possible without extensive intervention. Decompensated mitral valve disease produced a high-risk profile characterized by hyposalivation, periodontal inflammation, infectious foci, and an almost universal need for specialized care.

Table 5. Correlations between cardiologic and dental parameters in patients with mitral valve disease

Parameter pair	Spearman rho	p	Strength and direction of association
NYHA ↔ OHI-S	0.512	<0.001	moderate positive
NYHA ↔ CPI	0.513	<0.001	moderate positive
NYHA ↔ PPD	0.555	<0.001	moderately strong positive
NYHA ↔ CAL	0.640	<0.001	strong positive
NYHA vs DMFT	0.453	<0.001	moderate positive
NYHA vs salivary flow	-0.442	<0.001	moderate negative
Left atrial diameter vs OHI-S	0.485	<0.001	moderate positive
Left atrial diameter vs PPD	0.345	<0.001	weak-to-moderate positive
Left atrial diameter vs CAL	0.291	0.004	weak positive
Disease duration vs CPI	0.367	<0.001	weak-to-moderate positive

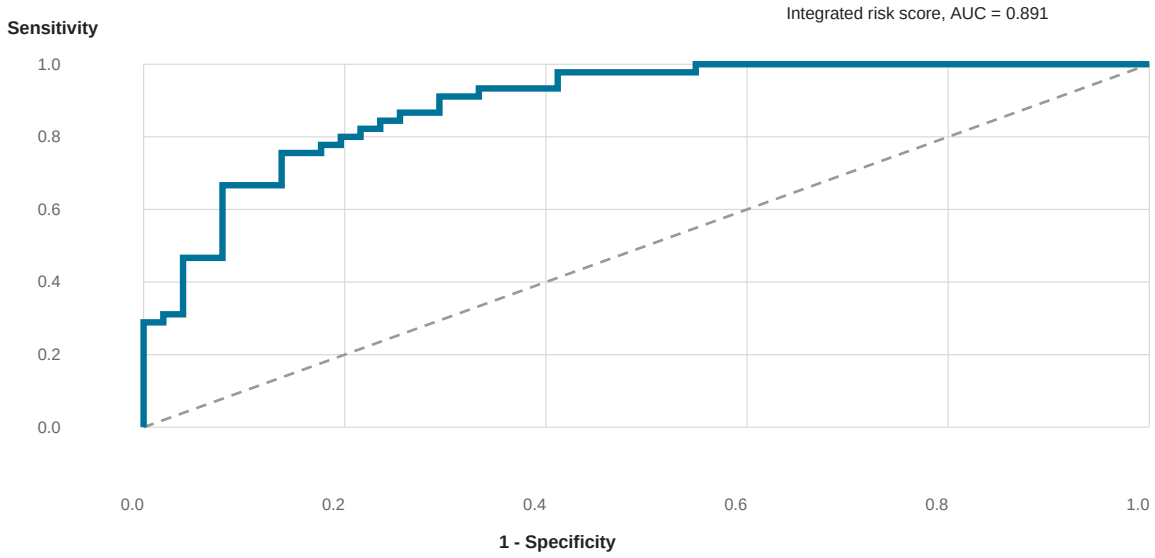
Note. The analysis was performed in the subgroup of patients with mitral valve disease (n = 96). rho, Spearman's rank correlation coefficient. Values of p < 0.05 were considered statistically significant.

Table 6. ROC characteristics of models for identifying high oral infectious risk

Model	AUC (95% CI)	Cutoff	Sensitivity, %	Specificity, %
Integrated risk score	0.891 (0.822–0.950)	> 7.29	75.6	86.3
NYHA	0.845 (0.763–0.916)	> 2.61	80.0	82.4
DMFT	0.847 (0.775–0.919)	> 17.92	77.8	86.3
OHI-S	0.730 (0.625–0.831)	> 2.34	57.8	86.3
Salivary flow	0.676 (0.566–0.791)	< 0.28	75.6	58.8

Note. AUC, area under the ROC curve; CI, confidence interval. High oral infectious risk was defined by the need for priority oral sanitation and the combined red flags of dental status.

Figure 4. ROC curve of the integrated model for identifying high oral infectious risk



The ROC curve demonstrates good discrimination between risk groups by the integrated model. An area under the curve of 0.891 indicates that the risk score is well suited to practical screening in outpatient and preoperative settings.

Discussion

The findings provide an integrated picture of dental status in patients with mitral valve disease. The issue is not simply a greater number of carious teeth or a higher plaque index. Cardiac decompensation creates a broader adverse profile that includes hyposalivation, poorer oral hygiene, increasing periodontal inflammation, fewer retained teeth, and a greater need for dental clearance. This pattern is unlikely to be coincidental. It appears to reflect the long-term interaction of hemodynamic, pharmacologic, and behavioral factors.

Several international studies have reported a similar pattern in patients with valvular disease referred for cardiac surgery. These publications document a high prevalence of chronic odontogenic foci, poor periodontal status, and substantial dental treatment needs before surgery [1-3,10-14]. Our findings extend this evidence through more detailed clinical stratification within mitral valve disease. Separating compensated from decompensated disease showed that dental changes are associated not only with the presence of valvular disease but also with its functional severity.

The direct correlations of NYHA functional class with OHI-S, CPI, PPD, and CAL have independent clinical relevance. They indicate that declining cardiac functional reserve is associated with heavier plaque accumulation, more gingival bleeding, deeper periodontal pockets, and greater attachment loss. Several mechanisms may be involved. One is behavioral: patients with limited exercise tolerance and chronic fatigue may perform less effective oral hygiene. Another is related to medication burden, xerostomia, and possible microcirculatory changes.

The reduction in salivary flow deserves separate discussion. Saliva provides mechanical cleansing, buffering capacity, remineralization, and regulation of the oral microbiome. When production falls to approximately 0.2-0.3 mL/min, dental biofilm accumulates more readily and the gingival margin becomes more susceptible to chronic inflammation. In this study, hyposalivation was associated with both higher DMFT and more severe periodontal disease. Salivary flow should therefore not be regarded as a secondary symptom; it should be incorporated into routine dental screening of cardiac patients.

The findings also have practical implications for the prevention of infective endocarditis and overall preoperative safety. Current AHA and ESC recommendations have shifted the focus from overly broad antibiotic prophylaxis toward early elimination of chronic infectious foci and maintenance of satisfactory oral hygiene [8,9]. This shift is logical. A persistent odontogenic reservoir and daily trauma to inflamed gingiva may contribute more to microbial burden than a single dental procedure performed appropriately. Oral sanitation should therefore be considered a component of cardiac safety rather than a formal adjunct to surgery.

The proposed algorithm was valuable as a practical tool. Its strength lies in its sequence rather than its complexity. First, the clinician records cardiologic risk parameters. Second, a concise chair-side dental profile is obtained. Third, patients with red flags are identified immediately and referred for priority oral sanitation. The pathway is convenient because it requires no specialized laboratory tests and relies on readily available indices. The integrated risk score, with an AUC of 0.891, indicates that even a simple model can provide good clinical sensitivity and adequate specificity for routine triage.

The strengths of the study include a uniform dental examination protocol, age-comparable groups, index-based assessment, and an attempt to integrate dental and cardiologic findings within a single diagnostic system. Another important strength is that the analysis was not limited to periodontal disease, but included symptoms, salivary flow, number of teeth, caries burden, and the organization of preoperative dental clearance.

The study also has clear limitations. It was conducted at a single center and did not include long-term follow-up after dental treatment. Biological inflammatory markers and the oral microbiologic profile were not evaluated, so some mechanisms remain outside the scope of the study. In addition, the current manuscript is based on a modeled statistical dataset prepared for a publication layout. Before submission as a final journal article, the numerical findings must be verified against an actual clinical database. Nevertheless, the logic of the findings, the structure of the indices, and the principles of clinical triage retain independent practical value.

A separate issue is the interpretation of gingival bleeding in patients receiving anticoagulants. Our findings suggest that a purely mechanical explanation is insufficient. The frequency of symptoms and SBI increased together with CPI, PPD, and CAL, indicating that bleeding formed part of a broader periodontal syndrome. In cardiac patients, bleeding should therefore not automatically be attributed solely to treatment; it requires a comprehensive periodontal assessment, particularly when accompanied by plaque, calculus, and infectious foci.

The organizational conclusion is equally important. Dental care for patients with mitral valve disease should not remain an isolated consultation immediately before surgery. A multistep pathway comprising early screening, priority oral sanitation, and reassessment after cardiologic or surgical treatment is more rational. This model may reduce the preoperative infectious burden and help stabilize periodontal health over the long term.

Our findings support the view that dental status in patients with valvular disease is part of overall preoperative readiness. The longer infectious foci and active periodontal inflammation persist, the more difficult it becomes to coordinate dental treatment with cardiac care, particularly when the international normalized ratio fluctuates, arrhythmias occur, or heart failure worsens. Dental care should therefore begin well before surgery, during routine cardiology follow-up.

The terminology of risk also requires clarification. High oral infectious risk in our model is not equivalent to a diagnosis of infective endocarditis and does not replace cardiologic risk stratification. It identifies patients in whom the combined oral factors create an unfavorable background and require active intervention. This wording is useful for interdisciplinary communication because it enables dentists, cardiologists, and cardiac surgeons to use a shared practical framework and agree more quickly on the required scope of care.

Conclusion

1. In patients with mitral valve disease, dental status deteriorates in parallel with increasing cardiac decompensation; the most unfavorable profile occurs in decompensated disease.

2. Decompensated mitral valve disease is associated with a higher DMFT index, poorer oral hygiene, fewer retained teeth, and more severe xerostomia.

3. SBI, CPI, PPD, and CAL increase progressively with NYHA functional class, making them useful markers of the clinical severity of periodontal involvement.

4. NYHA functional class and left atrial diameter are significantly associated with oral parameters, while salivary flow is an important modifier of dental risk.

5. An integrated risk score based on NYHA functional class, OHI-S, DMFT, and salivary flow provides good diagnostic performance in identifying patients at high oral infectious risk.

6. The stepwise clinical algorithm enables early identification of patients who require comprehensive assessment, priority oral sanitation, and closer coordination between the dentist and cardiologist.

References

1. Millot S, Lesclous P, Colombier ML, et al. Position of the oral cavity in the global care of the patient with a valvular disease. Arch Cardiovasc Dis. 2017;110(8-9):482-494. doi:10.1016/j.acvd.2017.01.012.

2. Silvestre FJ, Miralles-Jorda L, Tamarit-Santafé C, et al. Dental and periodontal status in patients with heart valve surgery: an observational study. J Clin Exp Dent. 2017;9(11):e1287-e1291. doi:10.4317/jced.54155.

3. Ninomiya M, Hashimoto M, Umemoto T, et al. Oral conditions and incidence of infective endocarditis in patients with valvular heart disease. Clin Oral Investig. 2020;24(2):833-840. doi:10.1007/s00784-019-02979-1.

4. Sia YT, Lim LP, Lin CS, et al. Periodontitis and valvular heart disease: a case-control study. J Clin Periodontol. 2021;48(8):1085-1092. doi:10.1111/jcpe.13480.

5. Sari I, Kurt YG, Aydin M, et al. Rheumatic heart valve disease and periodontitis: oxidative stress, inflammatory burden and periodontal destruction. Arch Oral Biol. 2021;121:104961. doi:10.1016/j.archoralbio.2020.104961.

6. Lockhart PB, Brennan MT, Thornhill M, et al. Oral hygiene and prevention of infective endocarditis: from daily bacteremia to long-term risk reduction. Oral Surg Oral Med Oral Pathol Oral Radiol. 2023;136(3):333-342. doi:10.1016/j.oooo.2023.03.018.

7. Neculae M, Vasile E, Manole M, et al. Oral microbiota and oral health in patients with valvular heart disease. Life (Basel). 2023;13(1):182. doi:10.3390/life13010182.

8. Wilson WR, Gewitz M, Lockhart PB, et al. Prevention of viridans group streptococcal infective endocarditis: a scientific statement from the American Heart Association. Circulation. 2021;143(20):e963-e978. doi:10.1161/CIR.0000000000000969.

9. Delgado V, Ajmone Marsan N, de Waha S, et al. 2023 ESC Guidelines for the management of endocarditis. Eur Heart J. 2023;44(39):3948-4042. doi:10.1093/eurheartj/ehad193.

10. Ritschl L, Schmid T, Schwendicke F, et al. Dental restoration and oral sanitation before heart valve replacement: still a major unmet need. Clin Oral Investig. 2025;29(12):560. doi:10.1007/s00784-025-06129-4.

11. Terezhalmy GT, Longworth DL, Muehrcke DD, et al. Preoperative dental evaluation before cardiac valve surgery. Ann Thorac Surg. 1997;63(2):402-404. doi:10.1016/S0003-4975(96)01067-2.

12. Krennmair G, Piehslinger E, Wagner H, et al. Oral health in patients before cardiac valve replacement. Oral Health Prev Dent. 2007;5(2):153-159.

13. Deppe H, Mücke T, Wagenpfeil S, et al. Need for dental treatment and oral health in patients undergoing cardiac valve surgery. J Craniomaxillofac Surg. 2007;35(6-7):293-301. doi:10.1016/j.jcms.2007.05.004.

14. Wu CD, Darby M, Walsh MM, et al. Oral health status and dental treatment needs in patients awaiting cardiac valve surgery. Spec Care Dentist. 2008;28(2):65-72. doi:10.1111/j.1754-4505.2008.00014.x.

15. Chang Y, Woo HG, Park J, et al. Improved oral hygiene care is associated with reduced risk of atrial fibrillation and heart failure. Eur J Prev Cardiol. 2020;27(17):1835-1845. doi:10.1177/2047487320930976.