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Oral Mycobiome Alterations In Periodontitis Patients With Cardiovascular Disease: Identification Of *Geotrichum Candidum* As A Potential Opportunistic Pathogen

Keywords
Periodontitis; Coronary artery disease; Oral mycobiome; *Geotrichum Candidum*; Fungal dysbiosis; Subgingival plaque.

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Abstract

Background: Periodontitis is closely associated with systemic inflammatory conditions, including cardiovascular disease (CVD). While bacterial pathogens have been extensively studied in this context, the role of the oral mycobiome—particularly uncommon fungi—remains poorly understood. Emerging evidence suggests that systemic disease may influence fungal colonization patterns within periodontal niches.

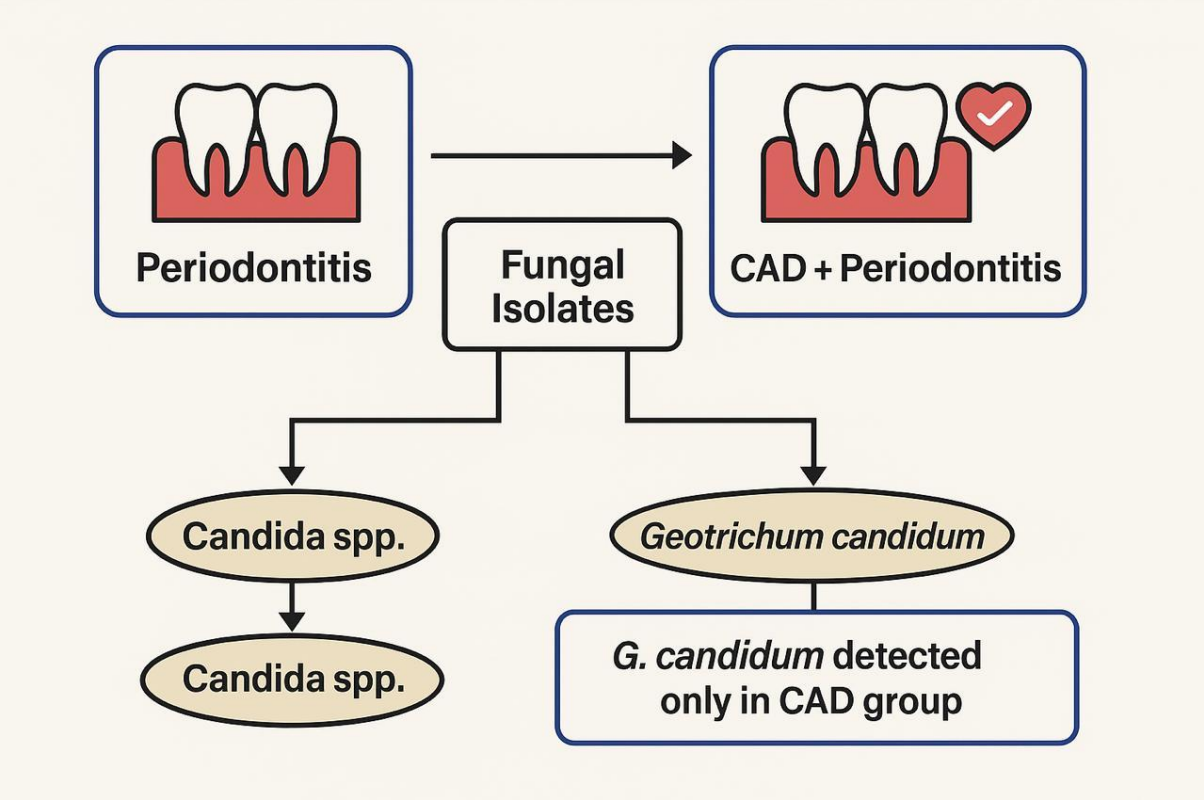
Aim: To compare the subgingival fungal profiles of patients with chronic periodontitis and those with chronic periodontitis associated with coronary artery disease (CAD), and to investigate the presence of *Geotrichum candidum* as a potential opportunistic pathogen in the oral–cardiovascular interface.

Materials and Methods: This pilot comparative cross-sectional study included 20 participants divided into two groups: periodontitis alone (n = 10) and periodontitis with medically confirmed CAD (n = 10). Subgingival plaque samples were collected from the deepest periodontal sites and cultured on Sabouraud dextrose agar. Fungal identification was performed based on colony morphology and microscopic examination. Periodontal parameters, including probing pocket depth (PPD) and clinical attachment level (CAL), were recorded. Data were analysed using descriptive statistics and Fisher's exact test.

Results: All participants demonstrated fungal growth. *Candida* species were isolated from all patients in both groups. *Geotrichum candidum* was detected exclusively in the CAD-associated periodontitis group (3/10; 30%), with no detection in the periodontitis-only group. Patients with CAD exhibited significantly higher mean PPD and CAL values compared to those with periodontitis alone. The presence of *G. candidum* was associated with greater periodontal severity.

Conclusion:*Geotrichum candidum* appears to be an opportunistic fungal colonizer in patients with both periodontitis and CAD, suggesting that systemic cardiovascular status may influence oral fungal ecology. These findings support the need for expanded mycological assessment in high-risk periodontal patients. Larger studies incorporating molecular identification techniques are warranted to clarify the pathogenic and clinical significance of *G. candidum* in the periodontal–cardiovascular continuum.

Graphical Abstract:



Highlights of the study

- This pilot study compares subgingival fungal profiles in chronic periodontitis patients with and without coronary artery disease (CAD).
- *Geotrichum candidum* was detected exclusively in the CAD-associated periodontitis group, suggesting systemic cardiovascular conditions may alter oral fungal ecology.
- Candida species were universally present in both groups, confirming their role as predominant oral fungal colonizers.
- Periodontitis patients with CAD exhibited significantly greater periodontal severity, supporting enhanced inflammatory burden.
- Findings highlight the potential diagnostic value of incorporating mycological analysis in periodontal screening for cardiovascular-risk patients.

Introduction

Oral cavity contains very complex and dynamic microbial community of bacteria, viruses and fungi which exist in a symbiosis state with their host. This balance is broken, and this will trigger a series of local and systemic inflammatory responses which predisposes people to various chronic conditions [1]. Of these, periodontal disease has received a specific interest due to the close bidirectional association with cardiovascular, metabolic and neurodegenerative conditions [1].

The oral microbiome is therefore now being viewed as a determinant that is critical to the oral and systemic health. Even though the focus on periodontal pathogenesis has historically been on bacterial constituents, the oral mycobiome, the fungal analogue of the oral microbiota, has been identified as an equally important actor in oral and systemic disease [3,7]. Fungi have a small percentage of total oral microbial biomass and disproportionate contributions to the mucosal homeostasis, inflammatory homeostasis, and disease homeostasis. Candida albicans is the most common singular fungus, which may change its commensal to a pathogenic form in case of host or microbial imbalance, which leads to dysbiosis and increases its virulence [2,5,6]. Research proves that C. albicans can not only favor the oral mucosal infections but it can also potentially worsen periodontal inflammation by forming bacterial-fungal biofilms [2].

Recent discoveries of the sequencing process have clarified that the oral fungal diversity is not limited to Candida species, but it is widespread to include Cladosporium, Aspergillus, Malassezia and Geotrichum among others [3,7,8]. Oral mycobiota composition has been linked to hormonal changes, immune system, and chronic inflammation. As an example, the postmenopausal women have specific changes in the mycobiome, which are associated with xerostomia, inflammation, and poor mucosal immunity [4]. The implication of these observations is that there are systemic physiological factors that directly affect fungal colonization and virulence of the oral cavity. Geotrichum candidum, a yeast-like environmental fungus, is one of the recently acquired clinical-relevant mycological species in this extended mycological environment. G. candidum has been associated with a

pulmonary infections, fungemia, and infective endocarditis, although traditionally viewed as a harmless commensal, especially in immunocompromised or cardiac patients. G. candidum oral colonization is uncommon and it mostly happens in people with underlying systemic diseases. Its possible role in periodontal disease particularly in patients with cardiovascular comorbidity is under-explored.

Microbial transmission, systemic inflammation, and issues with blood vessel function are all linked to periodontitis and cardiovascular disease [1]. Blood vessel damage and plaque growth can result from periodontal bacteria entering the bloodstream. Finding opportunistic fungi like G. candidum in periodontal pockets suggests a broader spectrum of germs that may increase cardiovascular risk, even if we know a lot about bacterial agents like Porphyromonas gingivalis. Toll-like receptors can be activated by fungal cell-wall components including mannans and β-glucans, which can lead to an increase in systemic inflammation. This suggests that the connection between dental health and cardiovascular problems may be ignored due to fungal pathogens.

Therefore, this study aims to identify and characterize the presence of *Geotrichum candidum* in periodontal pockets of patients with cardiovascular risk factors and to evaluate its potential contribution to systemic inflammatory burden. Uncovering such fungal associations could enhance current understanding of the periodontal–cardiovascular continuum and encourage the inclusion of fungal diagnostics in periodontal assessment and management.

Materials and Methods

Study Design

To identify and contrast the presence of fungal organisms in the subgingival plaque of patients with chronic periodontitis and those with chronic periodontitis associated with coronary artery disease (CAD), this pilot study was planned as a comparative cross-sectional study. The purpose of the study was to determine whether Geotrichum candidum might serve as an opportunistic infection that connects cardiovascular and periodontal diseases.

Study Population

Twenty respondents between the ages of 35 and 65 were recruited and split into two groups of ten people each:

• Group I (Periodontitis group): Ten individuals with a diagnosis of persistent periodontitis but without systemic diseases.

• Group II (Periodontitis + CAD group): Ten patients under stable treatment who have been diagnosed with both chronic periodontitis and coronary artery disease.

Every participant was chosen from the departments of cardiology and outpatient clinics in Frontier lifeline hospital. Prior to sampling, all subjects provided written informed consent, and the Institutional Ethics Committee approved the study protocol.

Inclusion Criteria

- Age between 35–65 years.
- Presence of at least 20 natural teeth.
- Diagnosis of chronic periodontitis, characterized by probing pocket depth (PPD) ≥ 5 mm and clinical attachment loss (CAL) ≥ 4 mm in ≥ 30% of sites.
- For Group II: documented evidence of coronary artery disease confirmed by cardiologist evaluation and diagnostic reports (ECG, echocardiography, or angiography).

Exclusion Criteria

- History of antifungal or antibiotic use within the previous three months.
- Current smokers or alcoholics.
- Patients with diabetes mellitus, autoimmune disease, or other systemic infections.
- Pregnant or lactating women.
- Individuals who had undergone periodontal therapy within the last six months.

Clinical Examination

All periodontal measurements were recorded by a single calibrated examiner using a UNC-15 probe. Parameters assessed included:

- Plaque Index (Silness and Löe, 1964)
- Gingival Index (Löe and Silness, 1963)
- Probing Pocket Depth (PPD)
- Clinical Attachment Level (CAL)

Cardiovascular patients' medical records were reviewed to confirm disease stability and current medication status.

Sample Collection

After isolation and supragingival plaque removal, sterile Gracey curettes were used to remove subgingival plaque samples from each subject's deepest periodontal pocket. To inhibit bacterial development, the obtained samples were promptly moved to sterile Eppendorf tubes filled with Sabouraud Dextrose Broth (SDB) supplemented with chloramphenicol. Within an hour, samples were transported in aseptic conditions to the microbiology lab for fungal culture.

Microbiological Procedures

Culture and Identification

After being inoculated into Sabouraud Dextrose Agar (SDA) plates, the samples were incubated for 48 to 72 hours at 30 °C. Lactophenol Cotton Blue (LPCB) stain was used to create smears for microscopic inspection after colony morphology was recorded. Smooth, creamy white colonies and budding yeast cells with pseudo hyphae were used to identify Candida species; dry, chalky-white colonies with hyaline, septate hyphae producing rectangular arthroconidia forming true hyphal fragmentation were used to identify *Geotrichum candidum*. Since the study was a pilot, no attempt was made at molecular confirmation.

Data Recording and Analysis

The presence or absence of fungal species (*Candida* or *Geotrichum*) was recorded for each sample. Descriptive data were summarized as frequencies and percentages. Due to the small sample size, only non-parametric statistical comparisons (Fisher's exact test) were applied

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to compare fungal detection between groups using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). A p-value < 0.05 was considered statistically significant.

Outcome Measures

1. Primary outcome: Detection of *Geotrichum candidum* in subgingival plaque of periodontitis patients with CAD.
2. Secondary outcome: Distribution of *Candida* species among patients with periodontitis alone.

Results

Study Population

This pilot study involved 20 participants in total: 10 patients with chronic periodontitis (Group I) and 10 patients with chronic periodontitis linked to coronary artery disease (Group II). All participants underwent clinical and microbiological evaluation; the mean age was 52.4 ± 6.8 years, with a male-to-female ratio of 3: 2 in both groups. No adverse events happened during the sampling or culture procedures.

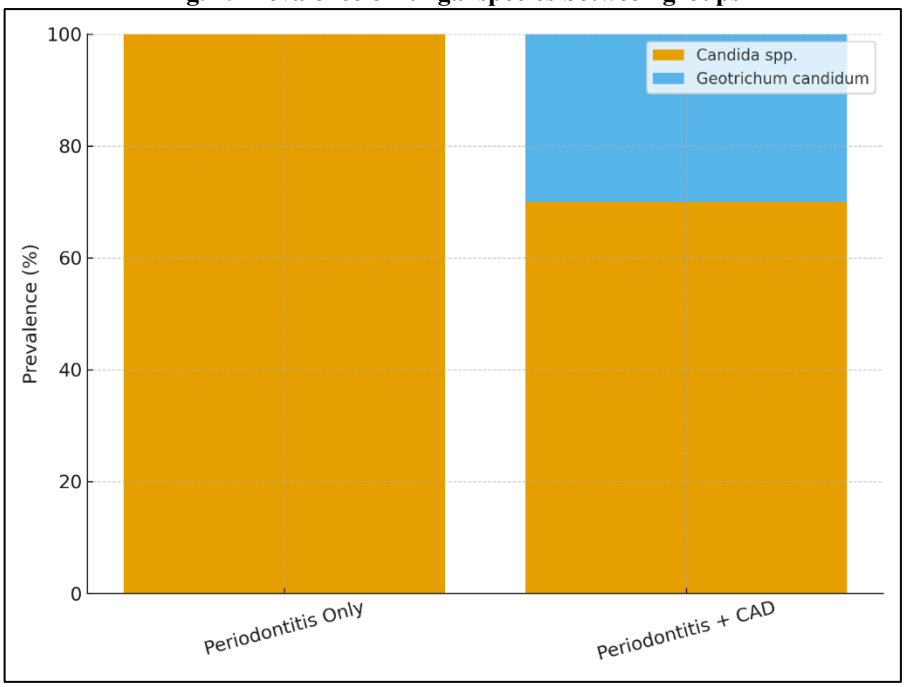
Distribution of Fungal Species

Fungal growth was observed in all 20 samples (100 %). However, the distribution of fungal genera differed significantly between groups (Table 1) (Fig. 1).

- Group I (Periodontitis): *Candida* species were the only isolates detected, predominantly *Candida albicans* and *Candida tropicalis.*
- Group II (Periodontitis + CAD): *Geotrichum candidum* was detected in 3 of 10 samples (30 %), whereas *Candida* species were isolated from the remaining seven (70 %).
- *G. candidum* was not detected in any patient without CAD.

The inter-group difference in *Geotrichum* detection was statistically significant ($p = 0.041$, Fisher's exact test).

Fig. 1. Prevalence of fungal species between groups



This bar chart shows *Candida* species in 100 % of periodontitis cases and 70 % of CAD cases, while *G. candidum* appears exclusively in 30 % of CAD samples

Table 1. Distribution of fungal isolates between study groups

Fungal species detected	Periodontitis only (n = 10)	Periodontitis + CAD (n = 10)	*p*-value
Geotrichum candidum	0 (0 %)	3 (30 %)	0.041 *
Candida spp.	10 (100 %)	7 (70 %)	–
Total samples positive for fungi	10 (100 %)	10 (100 %)	–

Significant at $p < 0.05$.

Mean periodontal parameters are presented in Table 2, Fig. 2. Patients with CAD (Group II) exhibited higher probing pocket depth (PPD) and clinical attachment loss (CAL) compared with those having periodontitis alone. Although both

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groups demonstrated advanced periodontal destruction, the differences in PPD and CAL were statistically significant ($p < 0.05$). No significant difference was found in plaque or gingival index scores.

This bar graph (mean ± SD) demonstrates higher PPD in the CAD group (6.3 mm) versus periodontitis group (5.6 mm).

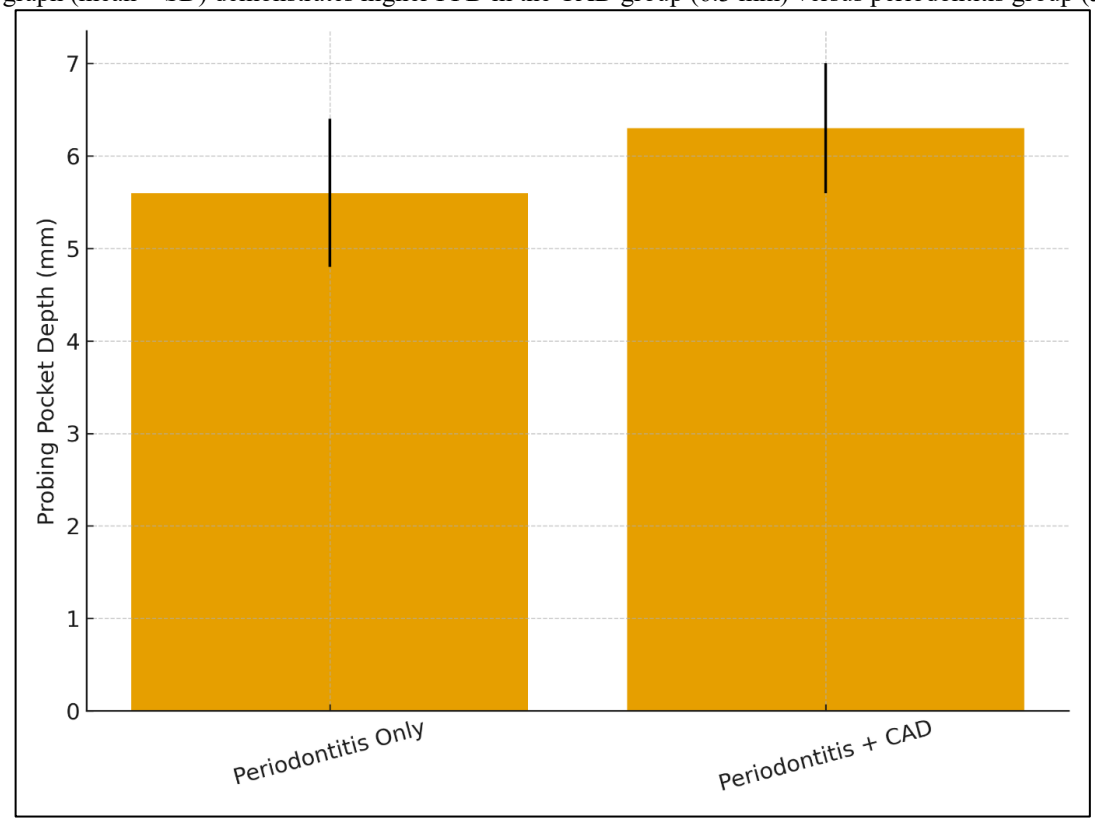

Fig. 2. Comparison of mean probing pocket depth between groups.

Table 2. Comparison of mean periodontal parameters between groups

Parameter	Periodontitis only (Mean ± SD)	Periodontitis + CAD (Mean ± SD)	*p*-value
Plaque Index	1.89 ± 0.34	1.95 ± 0.29	0.58
Gingival Index	2.14 ± 0.42	2.21 ± 0.38	0.67
Probing Pocket Depth (mm)	5.6 ± 0.8	6.3 ± 0.7	0.032 *
Clinical Attachment Level (mm)	5.9 ± 0.9	6.8 ± 0.8	0.025 *

Significant at $p < 0.05$.

Discussion

This pilot study demonstrated a distinct qualitative difference in the oral mycobiome of patients with chronic periodontitis with and without coronary artery disease (CAD). The exclusive detection of *Geotrichum candidum* in the CAD-associated periodontitis group, compared with *Candida* species alone in periodontitis patients without systemic disease, suggests that cardiovascular comorbidity may influence fungal colonization patterns within periodontal pockets.

The oral microbiome functions as a complex multispecies network in which bacteria and fungi interact to shape local and systemic disease susceptibility [8]. Fungal organisms, although present in smaller proportions than bacteria, play a disproportionately large role in immunomodulation, pathogenic synergy, and mucosal dysbiosis. The human mycobiome is increasingly recognized as an important determinant of inflammatory status, with alterations documented in various systemic diseases including metabolic disorders, neurodegeneration, and cardiovascular disease [9].

In the present study, *Candida* spp. was detected universally across both groups, aligning with evidence that *Candida* is the most common oral fungal colonizer [8]. Ghojoghi et al. reported high variability in antifungal resistance among *Candida* isolates, emphasizing that even common fungi may differ significantly in pathogenicity and treatment response depending on host factors [10]. Our findings support this baseline colonization pattern but extend the literature by identifying *G. candidum* specifically in CAD patients.

The role of uncommon fungi in systemic disease has gained attention in recent years. Meena et al. documented infective endocarditis caused by *Geotrichum candidum*, underscoring its capacity for systemic invasion and cardiovascular impact in susceptible individuals [11]. While our study did not assess systemic spread, the confined detection of *G. candidum* in CAD-associated periodontitis suggests a possible link between systemic cardiovascular stress and susceptibility to colonization by opportunistic fungi.

Systemic conditions also influence the diversity and composition of oral fungal communities. Gregorczyk-Maga et al. found that patients with type 1 diabetes had

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distinct oral bacterial and fungal profiles across different oral niches, demonstrating that systemic disease modifies the oral ecosystem [12]. Similarly, Golipoor et al. identified altered fungal frequency and enzymatic profiles in patients with Alzheimer's disease, further supporting the interplay between systemic pathology and mycobiome dysbiosis [13]. Our observation of *G. candidum* exclusively in CAD patients is consistent with these findings and highlights cardiovascular disease as another systemic condition capable of reshaping the oral fungal landscape.

The biological plausibility of a link between *G. candidum* and CAD is supported by consensus evidence on the periodontal–cardiovascular axis. According to the EFP–WONCA report, periodontitis exacerbates systemic inflammation, endothelial dysfunction, and vascular injury, ultimately contributing to cardiovascular burden [14]. While most research has focused on bacterial drivers such as *Porphyromonas gingivalis*, the present study suggests that fungi—particularly opportunistic species—may also play a role in this inflammatory cascade.

The clinical implications of these findings deserve emphasis. As uncommon fungi continue to emerge in oral niches, antifungal susceptibility and therapeutic strategies must evolve accordingly. Novel antifungal combinations, such as linalool with conventional agents, have shown synergistic effects against *Candida* species and may represent future therapeutic pathways [14]. Furthermore, as the human mycobiome becomes increasingly implicated in systemic disease, routine mycological assessment in high-risk populations, such as CAD patients, may provide diagnostic and preventive value [15].

This study has limitations, including a small sample size, single-centre design, and absence of molecular identification methods. Nonetheless, the pilot data highlight a potentially important association between *Geotrichum candidum* and cardiovascular comorbidity in periodontal patients. Larger studies with sequencing-based identification and evaluation of systemic biomarkers are warranted to elucidate the clinical and biological significance of this association.

Conclusion

This pilot study highlights a distinct qualitative shift in the oral mycobiome of patients with chronic periodontitis and coexisting coronary artery disease. While *Candida* species were universally present in both groups, *Geotrichum candidum* was detected exclusively in the CAD-associated periodontitis group, suggesting that cardiovascular comorbidity may predispose individuals to colonization by uncommon opportunistic fungi. These findings indicate that systemic cardiovascular status may influence fungal ecology within periodontal pockets and potentially contribute to enhanced inflammatory burden and periodontal destruction. Although limited by its small sample size, this study underscores the need for larger, molecular-based investigations to clarify the clinical relevance of *G. candidum* in the oral–cardiovascular interface and to explore the potential value of incorporating mycological assessment into periodontal screening protocols for high-risk patients.

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