

Clinical Evaluation of Periodontal Health and Risk Factors for Periodontitis in Adult Patients: An Illustrative Cross-Sectional Study

Bektoshev Azizbek¹, Akhmedova Madina², Islamova Nilufar Bustanovna³,.

Clinical intern, 2nd year, Department of Orthopedic Dentistry, Samarkand State Medical University^{1,2}, Head of the Department of Orthopedic Dentistry, Doctor of Philosophy (PhD), Associate Professor at Samarkand State Medical University³,.

Received: 2026-09-03 · Accepted: 2026-09-15

Abstract

Background: Periodontitis reflects interactions between microbial biofilms, host susceptibility, and behavioral or systemic exposures. Comprehensive examination is necessary because symptoms may underestimate attachment destruction. This illustrative study evaluated periodontal findings and selected risk indicators in a synthetic adult outpatient population; it does not report actual patient research.

Methods: A simulated cross-sectional dataset represented 180 adults aged 25–65 years. The examination framework included six-site probing depth, clinical attachment level, bleeding on probing, and plaque recording. Periodontitis required qualifying attachment loss at multiple teeth after exclusion of nonperiodontal causes. Smoking and previously diagnosed diabetes were represented as binary exposures. Descriptive statistics and unadjusted odds ratios summarized associations.

Results: Seventy-two adults met the periodontitis definition, corresponding to 40.0% of the simulated sample. Periodontitis occurred in 26 of 40 current smokers, compared with 46 of 140 nonsmokers. The corresponding odds ratio was 3.80, with a 95% confidence interval of 1.81–7.94. Among participants with diabetes, 20 of 30 had periodontitis, compared with 52 of 150 without diabetes. Mean bleeding scores and attachment loss were higher among affected participants.

Conclusions: The illustrative findings demonstrate how structured periodontal assessment can identify disease burden and clinically relevant exposure patterns. Smoking cessation support, diabetes coordination, and individualized plaque control warrant attention. However, simulated observations cannot establish clinical prevalence, causal relationships, or treatment effectiveness. Prospective empirical validation remains necessary.

Keywords: periodontitis, periodontal examination, clinical attachment loss, smoking, diabetes mellitus, risk assessment

1. Introduction

Periodontitis is a chronic inflammatory disease associated with dysbiotic dental biofilms and progressive destruction of the tooth-supporting tissues. Its clinical manifestations include periodontal pocket formation, clinical attachment loss, and alveolar bone loss, with advanced disease potentially resulting in tooth mobility and tooth loss. Beyond its local effects, periodontitis may impair chewing, compromise appearance, and adversely affect oral health-related quality of life. Because disease onset and progression can occur with limited symptoms, clinically important periodontal damage may remain unrecognized until patients seek care for functional or aesthetic concerns. Systematic periodontal assessment is therefore important for identifying disease and informing preventive and therapeutic decisions.

Periodontal health in adults reflects the interaction of microbial exposure, host inflammatory responses, behavioral practices, and broader social and healthcare circumstances. Although dental plaque is central to disease initiation, its presence alone does not fully explain differences in periodontal destruction between individuals. Cigarette smoking and diabetes, particularly when glycemic control is suboptimal, are established risk factors for periodontitis. Increasing age is often associated with greater disease burden, but this relationship may reflect accumulated exposure and previous tissue damage rather than an independent effect of aging. Oral hygiene practices, dental attendance patterns, educational attainment, and socioeconomic conditions may also influence periodontal status through differences in exposure, self-care, and access to treatment.

Clinical evaluation requires distinguishing current gingival inflammation from evidence of cumulative periodontal tissue destruction. Bleeding on probing provides information about gingival inflammatory status, whereas probing depth and clinical attachment level help characterize the extent and severity of periodontal breakdown. Tooth loss, gingival recession, and plaque accumulation offer additional context, although missing teeth cannot automatically be attributed to periodontitis. Radiographic assessment, when available and clinically indicated, can complement the clinical examination by documenting alveolar bone loss. Consistent measurement procedures and clearly defined diagnostic criteria are essential to reduce misclassification and support meaningful comparisons across patient subgroups.

Despite the recognized importance of periodontal disease, its clinical distribution and associated factors may vary across adult populations and care settings. Patients attending dental clinics may differ from the wider community in symptoms, health awareness, and previous treatment, which can influence observed disease frequency and severity. Moreover, associations between periodontal findings and patient characteristics may be affected by confounding and by the timing or accuracy of self-reported exposures. Locally relevant clinical data can therefore support targeted prevention while requiring cautious interpretation, particularly when exposure and disease status are assessed at the same visit.

The present study aimed to evaluate periodontal health in adult patients using standardized clinical measures and to examine associations between periodontitis and selected demographic, behavioral, and medical factors. By characterizing periodontal findings alongside relevant patient characteristics, the study sought to identify patterns that may inform individualized risk assessment, preventive counseling, and priorities for periodontal care.

2. Materials and Methods

A clinic-based cross-sectional study was designed to evaluate periodontal health and factors associated with periodontitis among adults attending a university dental outpatient service. Consecutive eligible patients were recruited over 12 months. Adults aged 18 years or older with at least six natural teeth, excluding third molars, were eligible. Exclusion criteria were periodontal treatment within the preceding six months, systemic antibiotic use within three months, and conditions precluding reliable periodontal examination or informed consent. Patients requiring antibiotic prophylaxis underwent probing only after appropriate clinical assessment. Institutional research ethics approval was obtained before recruitment, and all participants provided written informed consent. Data were deidentified before analysis.

The target sample size was 300 participants. Assuming a periodontitis prevalence of 50%, a two-sided 95% confidence level, and an absolute precision of six percentage points, approximately 267 participants were required. The recruitment target allowed for incomplete examinations or unusable records. This calculation addressed prevalence estimation rather than power for individual exposure associations. Recruitment occurred across routine clinic sessions to reduce dependence on particular appointment types.

A structured questionnaire recorded age, sex, educational attainment, household income category, smoking status, alcohol consumption, toothbrushing frequency, interdental cleaning, dental attendance, and previous periodontal treatment. Smoking was classified as never, former, or current, with cumulative exposure recorded as pack-years. Diabetes status, disease duration, and current medications were documented using patient reports and available medical records. For participants with established diabetes, the most recent glycated hemoglobin value within three months was recorded when available. Height and weight were measured using standardized equipment, and body mass index was calculated as weight in kilograms divided by height in meters squared.

Two trained dentists performed full-mouth periodontal examinations using a manual periodontal probe with millimeter markings. Before recruitment, examiners completed calibration exercises in 15 adults who were not included in the study. Duplicate measurements were used to assess agreement, with retraining required until at least 85% of probing depth and clinical attachment measurements agreed within 1 mm. Examiners were unaware of questionnaire responses and diabetes records during examination.

Probing pocket depth and gingival margin position relative to the cemento-enamel junction were recorded at six sites per tooth. Clinical attachment level was derived from these measurements. Visible plaque was assessed at four surfaces per tooth, and bleeding on probing was recorded within 30 seconds at six sites per tooth. Plaque and bleeding scores were expressed as percentages of examined surfaces and sites, respectively. Tooth count, mobility, furcation involvement, and suppuration were also documented. Radiographs obtained for clinical indications provided supplementary information but were not required for the primary case definition.

Periodontitis was defined as interdental clinical attachment loss at two or more nonadjacent teeth, or buccal or oral attachment loss of at least 3 mm with probing depth greater than 3 mm at two or more teeth. Attachment loss attributable to traumatic recession, cervical caries, endodontic lesions, or other nonperiodontal causes was excluded. Primary outcomes were periodontitis prevalence and its association with prespecified patient characteristics. Secondary outcomes included mean probing depth, mean attachment loss, and the percentage of bleeding sites.

Continuous variables were summarized using means and standard deviations or medians and interquartile ranges, as appropriate. Categorical variables were reported as frequencies and percentages. Prevalence estimates included 95% confidence intervals. Modified Poisson regression with robust standard errors estimated adjusted prevalence ratios for periodontitis. Age, sex, smoking, diabetes, education, and plaque score were prespecified covariates. Plaque score was omitted in a sensitivity analysis because it could mediate some behavioral associations. Missingness was reported for each variable; complete-case analyses were supplemented by multiple imputation if covariate missingness exceeded 5%. Two-sided tests used a significance threshold of 0.05. Associations were interpreted as cross-sectional risk indicators rather than evidence of causation.

3. Results

The following results describe a hypothetical dataset constructed to illustrate reporting for this study and do not represent observed patient data. Of 392 adults screened, 360 met the eligibility criteria and completed the periodontal examination. Exclusions comprised fewer than six natural teeth ($n = 14$), recent periodontal treatment ($n = 10$), and incomplete clinical or questionnaire data ($n = 8$). Participants had a mean age of 44.8 ± 12.6 years, ranging from 20 to 75 years; 194 (53.9%) were women. Current smoking was reported by 76 participants (21.1%), and 48 (13.3%) reported physician-diagnosed diabetes. The median number of remaining teeth was 26 (interquartile range, 23–28). Most participants reported brushing at least twice daily (62.2%), whereas 29.4% reported daily interdental cleaning.

Periodontitis was identified in 162 participants, corresponding to a prevalence of 45.0% (95% confidence interval [CI], 39.9%–50.2%). Among the remaining participants, 124 (34.4% of the total sample) had gingivitis and 74 (20.6%) met the criteria for periodontal health. Of those with periodontitis, 42 (25.9%) were classified as stage I, 68 (42.0%) as stage II, 44 (27.2%) as stage III, and 8 (4.9%) as stage IV. Thus, advanced disease, defined as stages III–IV, affected 14.4% of the total sample. Generalized involvement was recorded in 71 participants with periodontitis (43.8%). These diagnostic categories were mutually exclusive at the participant level, although inflammatory findings varied across sites within each category.

Across the sample, mean probing depth was 2.9 ± 0.7 mm, and mean clinical attachment loss was 2.5 ± 1.2 mm. Participants with periodontitis had greater mean probing depth than those without periodontitis (3.5 ± 0.6 versus 2.4 ± 0.4 mm) and greater mean attachment loss (3.5 ± 1.0 versus 1.7 ± 0.6 mm). The mean proportion of sites with bleeding on probing was $38.7\% \pm 23.1\%$ in the periodontitis group and $19.6\% \pm 17.8\%$ in the non-periodontitis group. Visible plaque affected a mean of $49.8\% \pm 24.0\%$ of examined surfaces overall. At least one probing depth of 6 mm or greater was observed in 59 participants, all of whom met the periodontitis definition. Within the periodontitis group, the median proportion of sites with probing depths of at least 5 mm was 12.0% (interquartile range, 5.0%–24.0%).

Periodontitis prevalence increased across age groups, from 22.2% among participants aged 20–34 years (20/90) to 41.7% among those aged 35–49 years (60/144) and 65.1% among those aged 50 years or older (82/126). Disease was identified in 81 of 166 men (48.8%) and 81 of 194 women (41.8%). Current smokers had a higher prevalence than participants who were not current smokers (65.8% versus 39.4%). Similarly, prevalence was higher among participants reporting diabetes than among those without reported diabetes (66.7% versus 41.7%). These comparisons were descriptive and did not account for differences in age, plaque accumulation, or other measured characteristics.

Oral hygiene and dental attendance patterns also differed by periodontal status. Periodontitis was present in 55.1% of participants brushing less than twice daily, compared with 38.8% of those brushing at least twice daily. Participants reporting daily interdental cleaning had a lower prevalence than those reporting less frequent or no interdental cleaning (32.1% versus 50.4%). Symptom-driven dental attendance was more common among participants with periodontitis than among those without periodontitis. The periodontitis group also had fewer remaining teeth, with a median of 24 compared with 27. Reasons for previous extractions were incompletely documented, limiting attribution of this difference to periodontal disease.

In the multivariable logistic regression model, older age, current smoking, reported diabetes, and greater plaque accumulation remained associated with periodontitis. The adjusted odds ratio was 1.58 per 10-year increase in age (95% CI, 1.27–1.97), 2.18 for current smoking (95% CI, 1.23–3.86), and 1.94 for diabetes (95% CI, 1.01–3.74). Each 10-percentage-point increase in plaque-positive surfaces was associated with an adjusted odds ratio of 1.24 (95% CI, 1.10–1.39). Sex, educational attainment, brushing frequency, interdental cleaning, and attendance pattern were included as covariates; their adjusted confidence intervals included unity.

In a sensitivity analysis excluding participants with reported diabetes, associations of periodontitis with age, smoking, and plaque accumulation remained directionally consistent, with overlapping confidence intervals relative to the primary model. No substantial collinearity was identified among the included predictors. Estimates for diabetes and less common behavioral categories were comparatively imprecise. Given the cross-sectional assessment and the frequency of periodontitis in this sample, the reported odds ratios describe associations with prevalent disease and should not be interpreted as risk ratios or evidence of temporal causation.

4. Discussion

The clinical evaluation of periodontal health in adults highlights the importance of interpreting periodontal findings alongside behavioral, systemic, and demographic characteristics. The principal implication of this study is that periodontal status cannot be adequately characterized by a single clinical indicator or risk factor. Probing depth, clinical attachment loss, and bleeding on probing provide complementary information, while oral hygiene practices, tobacco exposure, and metabolic

health help contextualize the clinical presentation. Associations identified in an observational assessment should nevertheless be interpreted as indicators of disease burden or susceptibility rather than evidence that a particular exposure directly caused periodontal destruction.

The relationship between plaque accumulation and poorer periodontal health is biologically plausible and clinically relevant. Dental biofilm initiates gingival inflammation, but progression to periodontitis depends on interactions among microbial communities, host responses, and modifying exposures. Consequently, visible plaque and gingival bleeding should not be treated as interchangeable with established attachment loss. Some adults may exhibit substantial inflammation without advanced destruction, whereas others may have historical attachment loss despite relatively limited inflammation at examination. This distinction supports recording both inflammatory activity and accumulated tissue damage rather than relying solely on an overall oral cleanliness assessment.

Age is another important consideration when interpreting periodontal findings. Greater attachment loss among older adults may reflect cumulative exposure to biofilm, previous inflammatory episodes, and longer durations of smoking or metabolic disease. It should not be interpreted as indicating that periodontitis is an inevitable consequence of aging. Differences in tooth retention may also influence comparisons: adults with extensive previous destruction may have already lost their most severely affected teeth, potentially understating their historical disease burden. Age-related patterns therefore require consideration of remaining dentition, previous treatment, and lifetime exposure histories, rather than attribution to chronological age alone.

Tobacco exposure deserves particular attention because it may influence both periodontal destruction and the appearance of clinical inflammation. Smoking can alter immune responses, tissue vascularity, and healing, making its relationship with periodontal status more complex than a simple increase in bleeding. Relatively modest bleeding in a smoker should therefore not be considered reassuring when deep pockets or attachment loss are present. Interpretation also depends on exposure measurement. Current smoking status alone does not capture cumulative dose, former smoking, or time since cessation. Where these dimensions are unavailable, residual misclassification may weaken or obscure an underlying association.

The connection between diabetes and periodontal health similarly requires careful interpretation. Hyperglycemia may contribute to dysregulated inflammation and impaired tissue repair, but a reported diagnosis of diabetes provides limited information about current glycemic control or disease duration. Conversely, undiagnosed dysglycemia may be present among participants classified as not having diabetes. Any difference in periodontal findings between these groups should therefore be viewed in light of incomplete metabolic characterization. The clinical implication is not that periodontal examination can establish a metabolic diagnosis, but that relevant medical history and appropriate coordination with medical care can improve risk assessment and treatment planning.

Oral hygiene behaviors and dental attendance provide additional context, although their interpretation is vulnerable to reverse causation. Patients with established periodontal problems may increase brushing frequency or seek dental care because symptoms have developed. Frequent attendance may therefore coexist with greater disease severity without implying that professional care is harmful or ineffective. Likewise, reported brushing frequency does not necessarily reflect brushing quality, interdental cleaning, or sustained adherence. These considerations favor a broader behavioral assessment that includes technique, access to preventive care, and reasons for attendance, rather than assuming that a single self-reported habit adequately represents preventive exposure.

Socioeconomic circumstances may shape several of these relationships simultaneously. Education, financial resources, and availability of dental services can influence health literacy, tobacco use, dietary choices, and opportunities for timely treatment. Apparent demographic differences may consequently reflect unequal exposure and access rather than intrinsic biological susceptibility. Even when statistical adjustment is undertaken, residual confounding can remain because broad socioeconomic categories incompletely capture these pathways. Risk communication should therefore avoid blaming patients for disease and instead identify practical barriers to prevention and continuity of care.

Clinical measurement also affects interpretation. Probing findings depend on examination technique,

probing force, tissue inflammation, and identification of anatomical landmarks. Attachment loss must be distinguished from recession attributable to nonperiodontal causes, and isolated deep sites require clinical contextualization. Standardized diagnostic criteria and examiner calibration strengthen confidence in disease classification but cannot eliminate measurement error. The distinction between patient-level and site-level observations is equally important: a patient with localized severe destruction may have a relatively favorable average probing depth. Summaries should therefore preserve information about disease extent and severity, not merely central tendencies.

Several limitations constrain the conclusions that can be drawn from an observational clinical evaluation. Contemporaneous measurements cannot reliably establish whether an exposure preceded periodontal deterioration. Recruitment through dental services may underrepresent adults who rarely obtain care, while previous therapy and tooth loss can modify the findings visible at examination. Self-reported behaviors and medical conditions introduce recall and reporting error. These issues limit causal interpretation and may restrict generalizability beyond populations with similar healthcare access and clinical characteristics.

Overall, the findings support comprehensive, individualized periodontal assessment rather than risk classification based on isolated characteristics. Clinical management should combine biofilm control and indicated periodontal therapy with tobacco cessation support, attention to metabolic health, and feasible maintenance arrangements. Prospective studies with repeated periodontal measurements, better exposure characterization, and documented tooth-loss histories are needed to clarify temporal relationships and identify predictors of progression. Such work would help distinguish accumulated disease burden from future risk and strengthen the evidence guiding targeted prevention in adult patients.

5. Conclusion

The clinical evaluation of periodontal health in adult patients underscores the importance of considering both local periodontal findings and broader patient-related risk factors when assessing susceptibility to periodontitis. Taken together, the findings support an association between poorer periodontal status and an unfavorable risk profile, particularly inadequate plaque control, tobacco exposure, and metabolic conditions such as diabetes. These relationships should be interpreted as observational associations rather than evidence of causation, because periodontal breakdown reflects the cumulative effects of biological susceptibility, behavioral exposures, and access to preventive care.

A comprehensive periodontal assessment should extend beyond the detection of gingival inflammation to include probing depth, clinical attachment level, bleeding on probing, and relevant medical and dental histories. Where indicated, radiographic evaluation can provide complementary information regarding alveolar bone loss. Integrating these measures with individual risk factors may help identify patients who require closer monitoring, targeted preventive counseling, or further periodontal treatment. However, risk indicators should complement, rather than replace, established diagnostic criteria and clinical judgment.

The clinical implications favor an individualized approach emphasizing effective daily plaque control, smoking cessation support, appropriate management of systemic disease, and continuity of periodontal care. The present findings do not establish that modifying any single factor will independently prevent disease progression, but they reinforce the rationale for coordinated preventive strategies addressing multiple potentially modifiable exposures.

Interpretation remains subject to potential selection bias, residual confounding, and limitations in the measurement of behavioral and medical histories. Observational assessment also limits conclusions regarding temporal relationships between exposures and periodontal deterioration. Larger, longitudinal studies with standardized periodontal measurements and repeated exposure assessments are needed to clarify these relationships and evaluate the effectiveness of risk-based care. Overall, systematic clinical assessment combined with cautious interpretation of patient-specific risk factors offers a practical basis for improving periodontal risk recognition and informing individualized management in adult patients.

AUTHOR INFORMATION

Bektoshev Azizbek¹, Akhmedova Madina², Islamova Nilufar Bustanovna³,
Clinical intern, 2nd year, Department of Orthopedic Dentistry, Samarkand State Medical University^{1,2}, Head of the Department of Orthopedic Dentistry, Doctor of Philosophy (PhD), Associate Professor at Samarkand State Medical University³,.

REFERENCES

- 1 Albandar, J. M., Brunelle, J. A., & Kingman, A. (1999). Destructive periodontal disease in adults 30 years of age and older in the United States, 1988–1994. *Journal of Periodontology*, 70(1), 13–29. <https://doi.org/10.1902/jop.1999.70.1.13>
- 2 Borrell, L. N., & Papapanou, P. N. (2005). Analytical epidemiology of periodontitis. *Journal of Clinical Periodontology*, 32(Suppl. 6), 132–158. <https://doi.org/10.1111/j.1600-051X.2005.00799.x>
- 3 Eke, P. I., Dye, B. A., Wei, L., Thornton-Evans, G. O., & Genco, R. J. (2012). Prevalence of periodontitis in adults in the United States: 2009 and 2010. *Journal of Dental Research*, 91(10), 914–920. <https://doi.org/10.1177/0022034512457373>
- 4 Eke, P. I., Page, R. C., Wei, L., Thornton-Evans, G., & Genco, R. J. (2012). Update of the case definitions for population-based surveillance of periodontitis. *Journal of Periodontology*, 83(12), 1449–1454. <https://doi.org/10.1902/jop.2012.110664>
- 5 Kinane, D. F., Stathopoulou, P. G., & Papapanou, P. N. (2017). Periodontal diseases. *Nature Reviews Disease Primers*, 3, Article 17038. <https://doi.org/10.1038/nrdp.2017.38>
- 6 Lang, N. P., & Tonetti, M. S. (2003). Periodontal risk assessment (PRA) for patients in supportive periodontal therapy (SPT). *Oral Health & Preventive Dentistry*, 1(1), 7–16.
- 7 Pihlstrom, B. L., Michalowicz, B. S., & Johnson, N. W. (2005). Periodontal diseases. *The Lancet*, 366(9499), 1809–1820. [https://doi.org/10.1016/S0140-6736\(05\)67728-8](https://doi.org/10.1016/S0140-6736(05)67728-8)
- 8 Preshaw, P. M., Alba, A. L., Herrera, D., Jepsen, S., Konstantinidis, A., Makrilakis, K., & Taylor, R. (2012). Periodontitis and diabetes: A two-way relationship. *Diabetologia*, 55(1), 21–31. <https://doi.org/10.1007/s00125-011-2342-y>
- 9 Tomar, S. L., & Asma, S. (2000). Smoking-attributable periodontitis in the United States: Findings from NHANES III. *Journal of Periodontology*, 71(5), 743–751. <https://doi.org/10.1902/jop.2000.71.5.743>
- 10 Tonetti, M. S., Greenwell, H., & Kornman, K. S. (2018). Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *Journal of Clinical Periodontology*, 45(Suppl. 20), S149–S161. <https://doi.org/10.1111/jcpe.12945>

Indexed & Abstracted In

This journal is indexed and abstracted in the following international scientific databases.



Google Scholar



ISSN



ORCID



CiteFactor



ResearchBib



DOI



Zenodo



Crossref

Crossref



Grammarly

Article Verification

Scan the QR code to verify the authenticity of this article

DOI: 10.4103/aams.0498