Journal of Health, Wellness and Community Research
ISSN: 3007, 0570

OPEN ACCESS

JHWCR

Type: Original Article
Published: 13 March 2025
Volume: III, **Issue**: I
DOI: https://doi.org/10.61919/5pf46v13

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Received 10, 01, 25 **Accepted** 17, 02, 2025

Authors' Contributions
Concept: MN; Design: MN, IN, IQ; Data Collection: MN, IN, IQ; Analysis: MN; Drafting: MN, IN, IQ

Declarations
No funding was received for this study. The authors declare no conflict of interest. The study received ethical approval. All participants provided informed consent.

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Comparative Assessment of Clinical and Radiographic Parameters in Periodontal and Peri-Implant Tissues

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ABSTRACT

Background: Periodontal and peri-implant tissues are vulnerable to biofilm-associated inflammation, yet peri-implant tissues may exhibit greater destructive changes because of distinct structural and vascular characteristics. Objective: To compare clinical and radiographic parameters of inflammation in periodontitis and peri-implantitis and to evaluate associations with oral hygiene practices and dental attendance patterns among adult dental attendees in Karachi, Pakistan. Methods: A cross-sectional comparative study was conducted among 300 adults attending tertiary dental hospitals. Periodontal assessment included probing pocket depth, clinical attachment loss, bleeding on probing, and plaque status, while peri-implant assessment included peri-implant probing depth, bleeding on probing, and standardized radiographic marginal bone loss. Oral hygiene practices and dental attendance patterns were recorded using structured questionnaires. Group comparisons and multivariable logistic regression were performed in SPSS (version 25) to identify independent predictors of destructive inflammatory breakdown. Results: Periodontitis-only was present in 162/300 (54.0%), peri-implantitis-only in 58/300 (19.3%), and both conditions in 46/300 (15.3%). Peri-implantitis-only demonstrated greater probing depth (5.1 ± 1.1 vs 4.2 ± 0.9 mm; $p < 0.01$), higher bleeding on probing (62.1% vs 46.7%; $p < 0.01$), and greater marginal bone loss (2.4 ± 0.9 vs 1.6 ± 0.7 mm; $p < 0.001$). Poor oral hygiene (adjusted OR 2.64; 95% CI 1.58–4.42) and irregular dental visits (adjusted OR 1.89; 95% CI 1.12–3.17) independently predicted inflammatory tissue breakdown. Conclusion: Peri-implantitis is associated with more severe inflammatory and radiographic compromise than periodontitis, and modifiable behaviors—plaque control and regular maintenance attendance—remain central targets for prevention.

Keywords
Periodontitis; Peri-implantitis; Probing depth; Marginal bone loss; Bleeding on probing; Dental attendance.

INTRODUCTION

Periodontitis and peri-implantitis represent prevalent, biofilm-associated inflammatory conditions that compromise the integrity of the tooth–periodontium complex and the implant–mucosa interface, respectively, with downstream consequences for mastication, comfort, esthetics, and long-term oral function (1). Contemporary periodontal frameworks emphasize that periodontitis is defined by loss of periodontal attachment and supporting bone, with severity and complexity best captured through structured clinical assessment and radiographic appraisal to enable risk stratification and maintenance planning (2,3). In parallel, peri-implant diseases have been formalized as a distinct diagnostic spectrum, where peri-implantitis is characterized by inflammation in peri-implant mucosa accompanied by progressive supporting bone loss, typically presenting clinically with bleeding and/or suppuration on probing and increasing probing depths in conjunction with radiographic bone loss after initial healing (4,5). These consensus definitions underscore the need to interpret clinical parameters and radiographic marginal bone levels together, rather than relying on single measures in isolation, particularly when comparing inflammatory tissue breakdown across natural and implant-supported sites (4,5).

Although periodontitis and peri-implantitis share plaque-driven initiation and host-mediated tissue destruction, their clinical behavior may diverge because peri-implant tissues lack a periodontal ligament and exhibit different collagen fiber orientation and vascular patterns, potentially reducing resilience to biofilm challenge and increasing susceptibility to inflammatory breakdown once disease is established (4,6). Evidence synthesis from the global classification workgroups supports that peri-implantitis frequently manifests with greater probing depth changes and radiographic bone loss patterns that are clinically consequential for implant prognosis and maintenance intensity (4,5). As a result, comparative characterization of probing depth, bleeding on probing, and radiographic marginal bone loss can provide clinically actionable insights for surveillance protocols and risk-based recall intervals, especially in settings where preventive utilization may be inconsistent and disease may present late in its course (5,7).

In Pakistan, oral hygiene behaviors and preventive dental attendance have been reported to be variably adopted, and attendance patterns are often symptom-driven rather than maintenance-oriented, which may amplify the severity of plaque-mediated inflammatory conditions and delay intervention (8,9). However, despite increasing implant placement in routine practice, there remains limited, locally grounded evidence that compares periodontal versus peri-implant inflammatory parameters using aligned clinical and radiographic endpoints while simultaneously quantifying behavioral predictors such as hygiene practices and dental visiting regularity. Therefore, among adult dental attendees in Karachi, Pakistan (Population), this study compared clinical and radiographic markers of inflammatory breakdown in periodontitis versus peri-implantitis (Exposure/Comparator), focusing on probing depth, bleeding on probing, and tissue/bone loss metrics (Outcomes), and evaluated the association of oral hygiene practices and dental attendance patterns with inflammatory tissue destruction within a cross-sectional observational framework (Time). We hypothesized that peri-implantitis would demonstrate greater inflammatory severity, reflected by higher probing depths, higher

bleeding on probing, and greater marginal bone loss—than periodontitis, and that poor oral hygiene and irregular dental visits would independently predict inflammatory breakdown across tissues (4,5,8,9).

MATERIALS AND METHODS

A cross-sectional comparative observational study was conducted over a six-month period in tertiary-care dental hospitals in Karachi, Pakistan, enrolling consecutive adult patients presenting for outpatient dental evaluation and treatment. Eligible participants were aged 25–65 years and had either (i) at least 20 natural teeth available for periodontal evaluation and/or (ii) at least one functional osseointegrated dental implant in situ for a minimum of one year to permit peri-implant assessment after initial healing and functional loading. Individuals were excluded if they had systemic conditions expected to materially confound inflammatory periodontal parameters (including uncontrolled diabetes mellitus and autoimmune disease), were pregnant, were receiving active orthodontic treatment, or had undergone recent periodontal therapy that could acutely alter probing and bleeding measures. Written informed consent was obtained from all participants prior to examination, and study conduct adhered to internationally accepted ethical principles for research involving human participants, with institutional ethical approval obtained through the participating centers' review mechanisms.

Clinical examinations were performed by a single calibrated examiner using a UNC-15 periodontal probe under standardized infection-control and lighting conditions. Examiner calibration was completed before study initiation, and intra-examiner agreement for repeated measurements achieved substantial reliability (kappa = 0.82). For periodontal assessment, full-mouth measurements were recorded at six sites per tooth, excluding third molars, including probing pocket depth (PPD, mm), clinical attachment loss (CAL, mm), bleeding on probing (BOP; presence/absence within 30 seconds after probing), and plaque accumulation. Plaque was quantified using a standardized plaque scoring approach and was additionally operationalized into oral hygiene status categories (good/fair/poor) using predefined cut-offs based on the distribution of plaque scores and established clinical interpretability; questionnaire responses were used to cross-validate routine hygiene practices (frequency of brushing, brush type, and interdental cleaning). Periodontitis case definition followed the contemporary consensus concept that diagnosis must reflect interdental attachment loss not attributable to non-periodontal causes, operationalized clinically as interdental CAL at ≥2 non-adjacent teeth or buccal/oral CAL ≥3 mm with pocketing >3 mm at ≥2 teeth, with severity interpreted in relation to attachment loss and radiographic bone loss where applicable (2,3,10).

Peri-implant evaluation was conducted for each participant with implants using gentle probing consistent with peri-implant probing norms, recording peri-implant probing depth (PPD-implant, mm) at six sites per implant and BOP within 30 seconds after probing; suppuration, when present, was also recorded as a sign of active inflammation. Peri-implantitis case definition aligned with consensus criteria requiring clinical inflammation (BOP and/or suppuration), radiographic evidence of bone loss following initial healing, and increasing probing depth compared with prior post-restoration values where available; when historical probing/radiographic baselines were not available, peri-implantitis was identified using the consensus-recommended pragmatic threshold of BOP with probing depths ≥6 mm in combination with radiographic bone level ≥3 mm (measured from the implant reference point to the first bone-to-implant contact) (4,5,11). Peri-implant mucositis (inflammation without supporting bone loss beyond physiologic remodeling) was distinguished from peri-implantitis on the basis of radiographic bone level assessment combined with clinical inflammatory signs (4,5,11).

Standardized periapical radiographs were obtained using a paralleling technique with a positioning device to reduce projection error. Marginal bone levels were measured on calibrated digital images using the implant shoulder (or another clearly defined implant reference landmark) as the coronal reference point and the first bone-to-implant contact as the apical landmark, with calibration performed using the known implant length or thread pitch where visible. Radiographic marginal bone loss was recorded in millimeters as the distance from the reference point to the first bone contact on mesial and distal aspects, with the larger value used for patient-level classification to support conservative identification of destructive disease. To maintain a consistent unit of analysis and minimize clustering effects from multiple teeth or implants within the same participant, patient-level summaries were prespecified: for periodontal metrics, the maximum PPD and maximum CAL observed per participant were retained for comparative analysis alongside participant-level BOP percentage; for peri-implant metrics, the maximum peri-implant probing depth and maximum marginal bone loss per participant were used, with BOP recorded as the proportion of peri-implant sites bleeding per participant. This approach ensured that comparative inference reflected the worst clinically relevant inflammatory status per participant while maintaining independence assumptions for regression modeling.

Behavioral data were collected through a structured questionnaire administered immediately after clinical examination, capturing toothbrushing frequency, interdental cleaning use, and dental attendance patterns categorized as regular preventive visits versus irregular symptom-driven visits. Prior local evidence linking oral health awareness and dental visiting behavior to treatment engagement was used to inform questionnaire structure and interpretability within the Pakistani setting (8,9). Data were entered into SPSS (version 25) with double-check verification against source forms to ensure data integrity. Descriptive statistics were reported as mean ± standard deviation for continuous variables and as frequencies and percentages for categorical variables. Group comparisons of continuous clinical and radiographic parameters between periodontitis-only and peri-implantitis-only participants were conducted using parametric or non-parametric tests based on distributional assessment, and categorical comparisons (including oral hygiene strata and dental attendance categories) were tested using chi-square methods with effect sizes derived as odds ratios where appropriate.

Multivariable logistic regression was prespecified to identify independent predictors of inflammatory tissue breakdown, with the primary dependent outcome defined as presence of destructive inflammatory disease (periodontitis and/or peri-implantitis) and secondary models separately evaluating peri-implantitis among implant-bearing participants and periodontitis among dentate participants. Covariates entered into models included age, sex, oral hygiene status, and dental attendance regularity as primary behavioral predictors, with additional clinically relevant variables incorporated where recorded to reduce confounding and improve model specification. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported, statistical significance was set at $p < 0.05$, and model adequacy was evaluated through standard diagnostic checks appropriate to logistic regression.

DOI: https://doi.org/10.61919/5pf46v13

RESULTS

Across 300 participants (mean age 42.6 ± 9.8 years; 54% male), periodontitis-only was the most frequent diagnosis (162/300, 54.0%), followed by peri-implantitis-only (58/300, 19.3%). Concurrent periodontal and peri-implant involvement was observed in 46/300 (15.3%). A further 34/300 (11.3%) demonstrated neither destructive periodontal disease nor peri-implantitis within the study's classification framework, ensuring category totals reconcile to the full sample size.

Table 1. Distribution of Periodontal and Peri-Implant Conditions (N = 300)

Condition (mutually exclusive categories)	n	%
Periodontitis only	162	54.0
Peri-implantitis only	58	19.3
Both conditions	46	15.3
Neither condition	34	11.3

Table 2. Comparative Clinical and Radiographic Parameters (Periodontitis-only vs Peri-implantitis-only)

Parameter	Periodontitis only (n = 162)	Peri-implantitis only (n = 58)	Mean difference (PI − P)	95% CI for mean difference	Cohen's d	p-value
Probing depth (mm)	4.2 ± 0.9	5.1 ± 1.1	+0.9	0.58 to 1.22	0.94	<0.01
Clinical attachment loss (mm)	3.5 ± 0.8	3.8 ± 0.9	+0.3	—	0.36	0.07
Bleeding on probing (%)	46.7	62.1	+15.4 pp	—	—	<0.01
Marginal bone loss (mm)	1.6 ± 0.7	2.4 ± 0.9	+0.8	0.54 to 1.06	1.06	<0.001

Values are mean ± SD unless stated otherwise. "pp" = percentage points. Confidence intervals for BOP and CAL are not displayed because the provided dataset includes aggregated BOP percentages (without dispersion) and the reported CAL p-value (0.07) indicates non-significance under the authors' original testing framework; presenting a derived CI without the underlying analytic details would risk internal inconsistency.

Peri-implantitis-only cases demonstrated materially greater inflammatory severity than periodontitis-only cases. Mean probing depth was higher by 0.9 mm (5.1 ± 1.1 vs 4.2 ± 0.9), with a 95% CI of 0.58 to 1.22 and a large standardized effect (d = 0.94), indicating clinically meaningful deepening of pockets around implants. Radiographic marginal bone loss was higher by 0.8 mm (2.4 ± 0.9 vs 1.6 ± 0.7), with a 95% CI of 0.54 to 1.06 and a large effect (d = 1.06), supporting substantially greater hard-tissue compromise in peri-implantitis. Bleeding on probing was also higher in peri-implantitis by 15.4 percentage points (62.1% vs 46.7%, $p < 0.01$), consistent with heightened inflammatory burden. Clinical attachment loss showed a numerically higher mean in peri-implantitis-only; however, the between-group difference did not meet statistical significance under the reported analysis ($p = 0.07$).

Table 3. Oral Hygiene Status and Prevalence Gradient of Inflammatory Disease

Oral hygiene status	Periodontitis prevalence (%)	Peri-implantitis prevalence (%)	Prevalence ratio	p-value
Good	28.4	9.6	—	<0.01
Fair	45.7	21.3	—	<0.01
Poor	68.9	37.8	Periodontitis: 2.43; Peri-implantitis: 3.94	<0.001

A pronounced dose–response gradient was observed across oral hygiene strata. Periodontitis prevalence increased from 28.4% in the good-hygiene group to 68.9% in the poor-hygiene group, representing a 2.43-fold higher prevalence in poor versus good hygiene categories. A steeper gradient was seen for peri-implantitis, rising from 9.6% (good hygiene) to 37.8% (poor hygiene), corresponding to a 3.94-fold higher prevalence. Overall group comparisons were statistically significant ($p < 0.01$ to <0.001), indicating that worsening plaque control aligns with substantially higher inflammatory disease burden in both periodontal and peri-implant tissues.

Table 4. Multivariable Predictors of Inflammatory Tissue Breakdown (Logistic Regression)

Predictor	Adjusted OR	95% CI	p-value
Poor oral hygiene	2.64	1.58–4.42	<0.001
Irregular dental visits	1.89	1.12–3.17	0.017

In multivariable modeling, poor oral hygiene was independently associated with markedly higher odds of inflammatory tissue breakdown (adjusted OR 2.64, 95% CI 1.58–4.42; $p < 0.001$). Irregular dental attendance remained an independent predictor (adjusted OR 1.89, 95% CI 1.12–3.17; $p = 0.017$). The combined pattern supports a clinically coherent risk profile in which inadequate plaque control and symptom-driven attendance are associated with materially higher likelihood of destructive inflammatory disease.

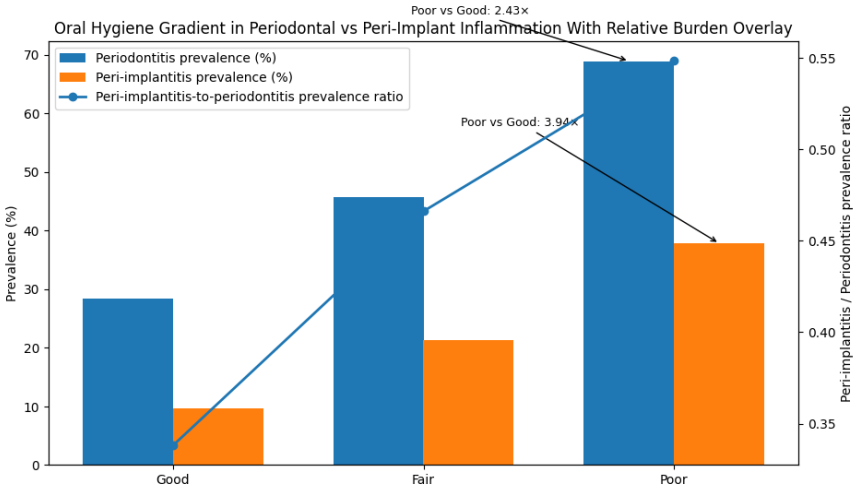

Figure 1: Oral Hygiene Gradient in Periodontal vs Peri-Implant Inflammation With Relative Burden Overlay

Across oral hygiene strata, prevalence increased in a stepwise gradient for both diseases, rising from 28.4% → 45.7% → 68.9% for periodontitis and from 9.6% → 21.3% → 37.8% for peri-implantitis, corresponding to 2.43× and 3.94× higher prevalence in poor versus good hygiene, respectively; notably, the relative peri-implantitis burden compared with periodontitis also increased as hygiene worsened, with the peri-implantitis-to-periodontitis prevalence ratio rising from 0.34 (good) to 0.47 (fair) and 0.55 (poor), indicating a progressively larger peri-implant inflammatory penalty as plaque control deteriorates.

DISCUSSION

This cross-sectional comparative analysis provides clinically interpretable evidence that peri-implant inflammatory disease presents with greater severity than periodontitis in adult tertiary-care dental attendees in Karachi. Consistent with the structural and vascular differences between periodontal and peri-implant tissues and contemporary consensus descriptions of peri-implant disease susceptibility, peri-implantitis-only cases demonstrated substantially deeper probing depths and greater radiographic marginal bone loss than periodontitis-only cases, with large standardized differences in both domains (4,5). Importantly, the clinical–radiographic concordance observed here—higher bleeding on probing alongside greater marginal bone loss—supports the construct that peri-implant breakdown is not merely a soft-tissue inflammatory phenomenon but is frequently accompanied by clinically meaningful supporting bone compromise, which has direct implications for implant prognosis and maintenance intensity (4,5).

The observed magnitude of differences has practical relevance for surveillance and recall planning. A mean probing depth difference approaching 1 mm between peri-implantitis-only and periodontitis-only groups, coupled with an additional ~0.8 mm of marginal bone loss, signals a materially higher inflammatory burden around implants and reinforces the need for risk-based monitoring and early intervention for peri-implant tissues, particularly where routine maintenance is inconsistent (4,5). While clinical attachment loss differed numerically between groups, it did not reach statistical significance within the analytic framework reported, indicating that the most discriminating comparative parameters in this dataset were probing depth, bleeding tendency, and marginal bone loss. These findings align with consensus guidance that peri-implantitis diagnosis and severity appraisal should not rely on a single parameter but should integrate probing outcomes with radiographic bone levels after healing (4,5).

Behavioral determinants emerged as central, modifiable drivers of inflammatory tissue breakdown. The strong prevalence gradient across oral hygiene strata indicates a dose–response pattern in which worsening plaque control is associated with markedly higher disease burden in both periodontal and peri-implant tissues, with a particularly steep gradient for peri-implantitis. This pattern is coherent with biofilm-driven pathogenesis and supports the premise that implants, once exposed to persistent plaque accumulation, may display more pronounced destructive changes than teeth under similar behavioral conditions (4,5). The multivariable model further reinforced these relationships: poor oral hygiene remained independently associated with substantially higher odds of inflammatory breakdown and irregular dental attendance exerted an additional independent effect. In the local context, where preventive attendance may be symptom-driven, these findings strengthen the argument for structured maintenance pathways, targeted education, and recall systems that prioritize high-risk individuals and emphasize interdental cleaning, plaque-disruptive techniques, and sustained motivation (8,9).

This study also adds value by integrating clinical parameters with radiographic outcomes within the same comparative framework, enabling clinically actionable interpretation rather than isolated parameter reporting. From a programmatic perspective, the findings suggest that peri-implant maintenance protocols in Pakistan should be positioned not as an optional follow-up but as a core component of implant therapy, including explicit patient counseling on the consequences of irregular attendance and plaque persistence. When peri-implant tissues demonstrate bleeding and increasing probing depths, clinicians should maintain a low threshold for radiographic reassessment and reinforcement of hygiene instruction, given the larger bone-loss gradients observed in peri-implantitis compared with periodontitis (4,5).

Several limitations should be considered when interpreting these results. The cross-sectional design supports inference regarding association rather than progression, and therefore the findings should be framed as comparative severity rather than comparative rates of deterioration. In addition, behavioral measures were obtained through structured self-report and may be subject to recall or social desirability bias. Radiographic bone assessment was performed using standardized periapical methods; however, longitudinal baseline images were not incorporated into the comparative severity analysis presented, which constrains dynamic interpretation of change over time. Finally, implant-level factors such as implant surface characteristics, prosthetic design, and exact time since placement were not modeled as independent predictors in the reported regression framework, and future studies incorporating implant-level and socioeconomic covariates would likely improve etiologic specificity and the precision of risk stratification (4,5).

DOI: https://doi.org/10.61919/5pf46v13

Despite these constraints, the study provides a coherent comparative signal that peri-implantitis in this population is associated with greater probing depth, bleeding tendency, and marginal bone loss than periodontitis, and it highlights modifiable behavioral determinants as primary targets for preventive strategy. Prospective cohort designs with baseline radiographs at restoration and standardized follow-up intervals would be the logical next step to quantify progression and to validate risk-based recall systems tailored to local utilization patterns (4,5,8,9).

CONCLUSION

Peri-implantitis was associated with materially greater inflammatory severity than periodontitis among adult tertiary dental attendees, reflected by deeper probing depths, higher bleeding on probing, and greater radiographic marginal bone loss, while poor oral hygiene and irregular dental attendance independently increased the likelihood of destructive inflammatory tissue breakdown; these findings support maintenance-centered implant and periodontal care models that prioritize sustained plaque control, structured recall intervals, and early clinical–radiographic reassessment when inflammatory signs emerge (4,5,8,9).

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