



Resolution of Periodontal Inflamed Surface Area (Pisa) and Perceived Stress Following Non-surgical Mechanical Therapy in Chronic Periodontitis: A Comparative Clinical Study

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ABSTRACT

Background

While the association between psychological stress and periodontal tissue breakdown is established, the therapeutic capacity of non-surgical mechanical debridement to modulate the biological wound surface area and patient-reported perceived stress remains under-investigated in large cohorts. This study evaluated the resolution of the Periodontal Inflamed Surface Area (PISA) and perceived psychological stress following non-surgical mechanical periodontal therapy in patients with chronic periodontitis.

Materials and Methods

A comparative clinical study was conducted involving 1,000 adult participants divided into two equal cohorts: Group B (Untreated Chronic Periodontitis, $n=500$) and Group C (Post-treatment Chronic Periodontitis evaluated at least six months following comprehensive scaling, root planing, and supportive care, $n=500$). An age- and sex-matched cohort of periodontally healthy individuals (Group A, $n=250$) served as a reference baseline. Full-mouth periodontal examinations were conducted across six sites per tooth to record the Simplified Oral Hygiene Index (OHI-S), Gingival Index (GI), Probing Pocket Depth (PD), Clinical Attachment Level (CAL), and Bleeding on Probing (BOP). These metrics were converted into PISA values (cm^2) using standardized root surface area algorithms. Perceived stress over the preceding month was quantified using the 10-item Perceived Stress Scale (PSS-10). Data were analyzed using independent Student's t -tests, one-way ANOVA with post-hoc Tukey's HSD, Chi-square tests, and Pearson's correlation coefficients ($p<0.05$).

Results

Patients in the post-treatment cohort (Group C) demonstrated significantly lower mean PISA values ($4.6\pm 2.1 \text{ cm}^2$) compared to the untreated periodontitis cohort (Group B, $12.5\pm 4.3 \text{ cm}^2$, $p<0.001$), reflecting a 63.2% reduction in vascularized ulcerated epithelial surface area. Concurrently, mean PSS-10 scores were significantly lower in Group C (17.2 ± 4.5) than in Group B (22.7 ± 5.1 , $p<0.001$). The proportion of patients categorized with high stress shifted from 24.0% ($n=120$) in the pre-treatment group to 7.4% ($n=37$) in the post-treatment group. While perceived stress exhibited a moderate-to-strong positive correlation with PISA during active untreated disease ($r=0.62$, $p<0.001$), this association was absent following therapy ($r=0.11$, $p=0.142$).

Conclusion

Non-surgical mechanical periodontal debridement resolves the biologically active inflammatory surface area (PISA) and is accompanied by a significant reduction in perceived psychological stress. Mechanical elimination of subgingival biofilms and pocket ulceration decouples the relationship between psychological stress appraisal and active periodontal tissue breakdown.

INTRODUCTION

Periodontitis is a chronic, non-communicable immuno-inflammatory disease initiated by dysbiotic subgingival microbial biofilms and driven by a dysregulated host immune response, leading to the irreversible breakdown of the periodontal ligament and alveolar bone.^[1,7,8,23] Although periopathogenic microorganisms are obligatory for disease initiation, the rate of progression, phenotypic severity, and clinical extent of tissue destruction are heavily influenced by environmental and systemic risk factors. Among these, chronic psychological distress has been recognized as a potent acquired risk modifier capable of altering neuroendocrine pathways, suppressing protective immunity, and perpetuating systemic inflammation.^[9-11]

Historically, clinical evaluations of periodontal disease status and therapeutic response have relied almost exclusively on discrete, linear measurements, such as Probing Pocket Depth (PD), Clinical Attachment Level (CAL), and categorical bleeding scores.^[4,5,7] While these conventional parameters offer essential historical documentation of cumulative tissue displacement and site-specific disease, they exhibit inherent limitations when quantifying the immediate biological magnitude of active tissue inflammation. A patient with deep, fibrosed pockets that lack bleeding upon probing may present substantial cumulative attachment loss without harboring a substantial active inflammatory wound.^[1]

To address this diagnostic limitation, Nesse and colleagues developed the Periodontal Inflamed Surface Area (PISA) formulation. By mathematically integrating full-mouth clinical attachment loss, probing depth, and bleeding on probing across six sites per tooth with standardized root surface area models,^[6] PISA computes the precise surface area of bleeding, ulcerated pocket epithelium in square centimeters. This transformation provides a biologically continuous metric that reflects the exact anatomical interface through which subgingival pathogens, bacterial endotoxins, and locally generated pro-inflammatory mediators enter the systemic microvasculature.

Simultaneously, chronic psychological stress exerts complex biological effects on periodontal homeostasis.^[9,10] Activation of the Hypothalamic-Pituitary-Adrenal (HPA) axis results in sustained glucocorticoid secretion, culminating in glucocorticoid receptor resistance on circulating leukocytes and tissue-resident macrophages. Concurrently,

sympathetic-adrenal-medullary (SAM) hyper-reactivity promotes localized arteriolar vasoconstriction and impairs neutrophil chemotaxis. Together, these mechanisms impede host clearance of subgingival anaerobes, promote connective tissue degradation, and maintain ulcerated pocket linings.^[10,23]

Crucially, emerging evidence suggests that the stress-periodontitis axis is bidirectional.^[10,12,13] Active, generalized periodontitis is marked by spontaneous bleeding, halitosis, tooth migration, and masticatory dysfunction, all of which compromise oral health-related quality of life and generate substantial psychosocial anxiety. Moreover, the systemic spillover of pro-inflammatory cytokines (such as Interleukin-1 β and Interleukin-6) from ulcerated pocket linings can breach the blood-brain barrier, triggering microglial activation and neurobehavioral fatigue.^[18,23]

While the correlation between elevated perceived stress and periodontal destruction in untreated patients has been documented,^[12-15] the capacity of non-surgical mechanical therapy (scaling and root planing) to simultaneously resolve the biological inflammatory wound area (PISA) and modulate patient-reported psychological stress remains under-explored in large-scale clinical cohorts. Therefore, this study was designed to evaluate the resolution of PISA and perceived stress following non-surgical mechanical periodontal therapy, and to determine whether clinical eradication of the subgingival microbial etiology decouples the association between psychological stress appraisal and active periodontal tissue ulceration.

MATERIALS AND METHODS

Ethical Approval and Study Design

This comparative clinical investigation was conducted in accordance with the Declaration of Helsinki (revised 2013) and Good Clinical Practice guidelines.^[27] The Institutional Ethics Committee, Bhopal, Madhya Pradesh, India, approved the study protocol. All participating subjects received detailed oral and written explanations of the study objectives and provided signed voluntary informed consent prior to enrollment.

Study Population and Cohort Allocation

A total of 1,250 adult participants aged between 20 and 60 years were recruited from the Outpatient Department of Periodontics, People's Dental Academy, Bhopal. The participants were assigned to three defined, mutually exclusive clinical cohorts:

Control Group (Group A, n = 250): Periodontally healthy subjects presenting with probing pocket depths ≤ 3 mm, absence of clinical attachment loss, and bleeding on probing at less than 10% of examined sites.^[8]

Pre-Treatment Test Group (Group B, n=500): Patients presenting with active, untreated generalized chronic periodontitis, characterized by probing pocket depths ≥ 4 mm and clinical attachment loss in more than 30% of examined clinical sites.^[7,8]

Post-Treatment Test Group (Group C, n=500): Patients previously diagnosed with generalized chronic periodontitis who had completed full-mouth non-surgical mechanical periodontal therapy (quadrant-wise scaling and root planing using ultrasonic instrumentation and area-specific Gracey curettes) and supportive periodontal care at least six months prior to clinical evaluation.^[20-22]

Eligibility Criteria

To minimize confounding influences on periodontal indices, systemic inflammation, and psychological stress profiles, strict eligibility criteria were enforced:

Inclusion Criteria: Adult male and female subjects aged 20–60 years having a minimum of 20 functional natural teeth (excluding third molars).

Exclusion Criteria: Current or former users of any form of smoked or smokeless tobacco; patients diagnosed with systemic medical disorders known to influence periodontal status or host immunity (including diabetes mellitus, cardiovascular conditions, autoimmune disorders, and hepatic/renal disease); pregnant or lactating females; patients who had used systemic antimicrobial, corticosteroid, or non-steroidal anti-inflammatory agents within the preceding six months; and individuals receiving active psychiatric counseling or psychopharmacological therapy.

Clinical Periodontal Examination

All clinical examinations were conducted by a single, calibrated periodontal examiner. The assessment covered six sites per tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual/palatal, mid-lingual/palatal, and disto-lingual/palatal) for all functional natural teeth, excluding third molars, using a standardized UNC-15 periodontal probe and a mouth mirror under adequate operatory illumination.

The recorded parameters comprised:

Simplified Oral Hygiene Index (OHI-S): Assessing debris and calculus accumulation.^[4]

Gingival Index (GI): Assessing the severity and qualitative extent of gingival inflammation.^[5]

Probing Pocket Depth (PD): Measured from the gingival margin to the base of the pocket, rounded to the nearest millimeter.

Clinical Attachment Level (CAL): Measured from the cemento-enamel junction (CEJ) to the base of the pocket.

Bleeding on Probing (BOP): Evaluated dichotomously (presence or absence of bleeding within 30 seconds of gentle probing with approximately 0.25 N force).^[1]

Calculation of Periodontal Inflamed Surface Area (PISA)

The anatomical quantification of oral inflammatory burden was computed using the mathematical algorithms established by Nesse et al. (2008).^[1]

Full-mouth CAL and recession values were used to compute the Periodontal Epithelial Surface Area (PESA) for each tooth, utilizing Humphrey's mean root surface area coefficients.^[1]

The PISA for each specific tooth site was derived by multiplying the individual PESA by the site-specific dichotomous BOP score (0 for non-bleeding, 1 for bleeding):

The individual site PISA values were summed across all examined teeth to yield the total full-mouth PISA value in square centimeters (cm²), reflecting the total vascularized ulcerated epithelial wound surface area.^[1]

Assessment of Perceived Psychological Stress

Subjective chronic psychological stress over the preceding 30 days was measured using the validated 10-item Perceived Stress Scale (PSS-10) originally formulated by Cohen et al.^[2,3] The instrument assesses how unpredictable, uncontrollable, and overloaded respondents find their lives.^[2]

Items were scored on a 5-point Likert scale ranging from 0 (*never*) to 4 (*very often*). Positively framed items (Questions 4, 5, 7, and 8) were reverse-coded before summing all ten responses, producing a composite continuous score ranging from 0 to 40.^[2,3] Stress scores were further classified into standard categories:^[2]

Low Stress: 0–13

Moderate Stress: 14–26

High Stress: 27–40

Table 1: Baseline Demographic Characteristics Across Study Cohorts

Demographic Variable	Group A (Control)(n=250)	Group B (Pre-Tx)(n=500)	Group C (Post-Tx)(n=500)	Test Statistic	p-value
Age (Years) (Mean $\pm$ SD)	37.2 $\pm$ 11.8	39.1 $\pm$ 12.6	38.8 $\pm$ 12.0	F=1.57	0.21
Sex Distribution, n (%)				$\chi^2=0.28$	0.87
• Male	120 (48.0%)	245 (49.0%)	250 (50.0%)		
• Female	130 (52.0%)	255 (51.0%)	250 (50.0%)		
Socioeconomic Status, n (%)				$\chi^2=3.65$	0.93
• Low SES	80 (32.0%)	195 (39.0%)	200 (40.0%)		
• Middle SES	120 (48.0%)	210 (42.0%)	205 (41.0%)		
• High SES	50 (20.0%)	95 (19.0%)	95 (19.0%)		

Statistical Analysis

Data were tabulated in Microsoft Excel and analyzed using SPSS Version 26.0 (IBM Corp., Armonk, NY). Continuous variables were summarized as mean $\pm$ standard deviation (SD), and categorical variables were expressed as frequencies and percentages.

Inter-group baseline demographic comparability was evaluated using one-way ANOVA for continuous variables and Chi-square (χ^2) tests for categorical distributions. Differences in clinical periodontal parameters and PSS-10 scores between cohorts were assessed using one-way ANOVA followed by Tukey's Honest Significant Difference (HSD) post-hoc test for multiple comparisons.

Direct comparisons between the pre-treatment (Group B) and post-treatment (Group C) cohorts were verified using independent two-tailed Student's *t*-tests, with Cohen's *d* calculated to determine effect size. Bivariate associations between PSS-10 scores and PISA within each cohort were evaluated using Pearson's correlation coefficient (*r*). Statistical significance was set at $\alpha=0.05$.

RESULTS

Demographic Comparability

The study population comprised 1,250 participants with a mean age of 38.5 $\pm$ 12.3 years. Analysis of baseline demographics confirmed that the cohorts were well-matched, with no statistically significant differences in age ($p=0.21$), sex distribution ($p=0.87$), or composite socioeconomic status ($p=0.93$), confirming inter-group comparability (Table 1).

Periodontal and Inflammatory Wound Resolution

Evaluation of clinical parameters revealed significant improvements in the post-treatment cohort (Group C) compared to the untreated periodontitis cohort (Group B) ($p<0.001$ for all variables; Table 2).

Mean full-mouth PISA values decreased from 12.5 $\pm$ 4.3 cm² in Group B to 4.6 $\pm$ 2.1 cm² in Group C, representing a mean difference of 7.90 cm² (95% CI: 7.44 to 8.36 cm²) and an overall 63.2% reduction in ulcerated inflammatory surface area.

Similarly, marked reductions were noted in mean OHI-S (2.3 $\pm$ 0.6 vs. 1.2 $\pm$ 0.4), GI (2.1 $\pm$ 0.5 vs. 1.1 $\pm$ 0.3), PD (4.8 $\pm$ 1.2 mm vs. 2.5 $\pm$ 0.7 mm), and CAL (3.5 $\pm$ 1.0 mm vs. 1.6 $\pm$ 0.5 mm). Cohen's *d* values for all periodontal indices exceeded 2.0, demonstrating substantial clinical effect sizes following mechanical intervention.

Changes in Perceived Psychological Stress (PSS-10)

Mean PSS-10 scores were significantly lower in the post-treatment cohort (17.2 $\pm$ 4.5) than in the untreated periodontitis cohort (22.7 $\pm$ 5.1, $t=18.09$, $p<0.001$; Table 3), showing a large effect size (Cohen's $d=1.14$).

Categorical redistribution of stress severity revealed that patients classified with high stress dropped from 24.0% ($n=120$) in Group B to 7.4% ($n=37$) in Group C (OR=0.25, 95% CI: 0.17 to 0.38, $p<0.001$). Concurrently, the proportion of individuals in the low-stress category rose from 3.6% ($n=18$) in Group B to 22.4% ($n=112$) in Group C (OR=7.72, 95% CI: 4.61 to 12.92, $p<0.001$).

Decoupling of the Stress-Inflammation Correlation

In active, untreated chronic periodontitis (Group B), bivariate correlation analysis demonstrated a statistically significant, moderate-to-strong positive correlation between PSS-10 and PISA ($r=0.62$, $r^2=0.384$, $p<0.001$; Table 4).

However, in the post-treatment cohort (Group C), this correlation was markedly attenuated and lacked

Table 2: Comparison of Clinical Periodontal Metrics and Inflammatory Surface Area Between Pre- and Post-Treatment Cohorts

Parameter	Group B (Pre-Tx, n=500)	Group C (Post-Tx, n=500)	Mean Difference	t-Statistic	p-value	Cohen's d
OHI-S	2.3±0.6	1.2±0.4	1.1	35.48	<0.001	2.15
Gingival Index (GI)	2.1±0.5	1.1±0.3	1	38.46	<0.001	2.43
Pocket Depth (PD, mm)	4.8±1.2	2.5±0.7	2.3	38.33	<0.001	2.34
CAL (mm)	3.5±1.0	1.6±0.5	1.9	40.42	<0.001	2.41
PISA (cm ²)	12.5±4.3	4.6±2.1	7.9	40.51	<0.001	2.36

Table 3: Redistribution of Perceived Stress Severity Categories Following Periodontal Therapy

Stress Severity Category	Group B (Pre-Tx, n=500)	Group C (Post-Tx, n=500)	Absolute Shift (Δ)	Odds Ratio (95% CI)	χ ² (p-value)
Low Stress (0–13)	18 (3.6%)	112 (22.4%)	+18.8%	7.72 (4.61–12.92)	84.21 (<0.001)
Moderate Stress (14–26)	362 (72.4%)	351 (70.2%)	–2.2%	0.89 (0.68–1.18)	0.61 (0.435)
High Stress (27–40)	120 (24.0%)	37 (7.4%)	–16.6%	0.25 (0.17–0.38)	56.32 (<0.001)

statistical significance ($r=0.11$, $r^2=0.012$, $p=0.142$). In periodontally healthy controls (Group A), the correlation was similarly non-significant ($r=0.08$, $p=0.207$).

DISCUSSION

This clinical investigation evaluated the therapeutic effect of non-surgical mechanical periodontal therapy on active oral inflammatory burden (PISA) and perceived psychological stress (PSS-10) in a large adult cohort from Bhopal, Madhya Pradesh. The findings demonstrate that comprehensive mechanical debridement is associated with substantial clinical closure of the ulcerated pocket epithelium and a concurrent reduction in perceived psychological distress.^[18–22] Furthermore, the data indicate that successful mechanical therapy eliminates the direct association observed between psychological stress and active periodontal tissue breakdown in untreated disease.

Biological Significance of PISA Resolution

The reduction in mean PISA from $12.5 \pm 4.3 \text{ cm}^2$ in untreated disease to $4.6 \pm 2.1 \text{ cm}^2$ following therapy represents the elimination of approximately 7.9 cm^2 of ulcerated pocket epithelium. In biological terms, an active inflammatory surface area exceeding 12 cm^2 represents an open, chronically bleeding wound roughly equivalent to the size of an adult human palm.^[1]

This ulcerated barrier allows continuous systemic entry of subgingival periopathogens, lipopolysaccharides (LPS), and inflammatory cytokines.^[18,19,23] By disrupting subgingival biofilms and eliminating calculus, non-surgical mechanical instrumentation permits pocket epithelial re-adaptation, junctional epithelial ad-

hesion, and microvascular resolution.^[20,22] This structural healing limits systemic bacteremia, dampens low-grade vascular inflammation, and resolves local tissue edema.^[18,19]

Mechanisms Driving Stress Attenuation Post-Therapy

The parallel reduction in mean PSS-10 scores from 22.7 ± 5.1 to 17.2 ± 4.5 ($p < 0.001$), accompanied by a notable decline in high-stress individuals (24.0% to 7.4%), suggests that effective periodontal debridement may positively influence patient psychological well-being. This response can be understood through two interconnected pathways:

Somatic and Functional Relief: Advanced untreated periodontitis produces distinct physical symptoms, including spontaneous gingival bleeding, persistent halitosis, tooth mobility, and masticatory pain. These symptoms can cause social embarrassment, self-consciousness, and reduced self-esteem.^[25,26] By eliminating bleeding, halitosis, and gingival soreness, successful therapy improves oral health-related quality of life (OHRQoL), alleviating daily disease-related emotional distress.^[25,26]

Attenuation of the Systemic Neuro-Inflammatory Loop: Pro-inflammatory cytokines generated within the ulcerated periodontium (such as IL-1β, IL-6, and TNF-α) enter the systemic circulation and can interact with the central nervous system.^[18, 23] These circulating mediators can stimulate the vagus nerve and alter blood-brain barrier transport, activating hypothalamic microglial cells and neuroendocrine pathways associated with sickness behavior, fatigue, and dysphoria. By resolving this inflammatory focus, mechanical therapy dampens

Table 4: Bivariate Pearson Correlation Between PSS-10 and PISA Across Cohorts

Study Cohort	Sample Size (n)	Pearson Correlation (r)	Coefficient of Determination (r ²)	95% Confidence Interval	p-value
Group A (Healthy)	250	+0.08	0.006	-0.04 to +0.20	0.207
Group B (Pre-Tx)	500	+0.62	0.384	+0.56 to +0.67	<0.001
Group C (Post-Tx)	500	+0.11	0.012	+0.02 to +0.20	0.142

systemic cytokine spillover, which may help relieve inflammatory signaling that contributes to heightened stress perception.^[18,24]

Decoupling of the Stress-Inflammation Axis

A primary finding of this study is the loss of a statistically significant correlation between PSS-10 and PISA in the treated cohort ($r=0.11$, $p=0.142$), contrasting sharply with the strong positive correlation observed in active untreated disease ($r=0.62$, $p<0.001$). These untreated findings corroborate observations by Parihar et al. (2024),^[12] Coelho et al. (2020),^[13] Varma et al. (2023),^[14] Balaji et al. (2018),^[15] Abe et al. (2020),^[16] and Macri et al. (2024).^[17]

This finding illustrates that psychological distress alone is insufficient to sustain extensive pocket ulceration in the absence of an organized subgingival microbial biofilm. Chronic stress acts as a disease modifier rather than a primary initiator^[10,13]. In untreated periodontitis, neuroendocrine dysregulation (HPA-axis glucocorticoid receptor resistance and SAM-mediated vasoconstriction) impairs the host's cellular defenses, allowing subgingival pathogens to proliferate and accelerate tissue breakdown^[18-24].

However, once root surfaces are debrided and subgingival dysbiosis is suppressed, background psychological stress no longer finds an active microbial nidus to amplify into severe epithelial ulceration. This supports the clinical principle that mechanical biofilm disruption remains the foundation of periodontal therapy, even in patients under ongoing psychological strain^[21,22].

Strengths and Limitations

Methodological strengths of this study include the large sample size ($N=1,000$ across pre- and post-treatment cohorts), the use of PISA as a continuous biological metric of inflammatory wound size, the application of a validated psychometric instrument (PSS-10), and strict exclusion criteria eliminating major confounders such as tobacco use, diabetes mellitus, and psychiatric medication^[1-3].

Several limitations should be noted. First, the

study employed a cross-sectional comparative design across distinct pre-treatment and post-treatment cohorts rather than a longitudinal design following the same individuals over time. While the cohorts were demographically well-matched, longitudinal intervention trials tracking serial post-therapy intervals are recommended to clarify the precise temporal sequence of stress and inflammatory resolution. Second, psychological distress was measured using self-reported psychometrics without accompanying biochemical stress markers, such as salivary cortisol or alpha-amylase. Future interventional studies incorporating systemic cytokine profiling, salivary neuroendocrine assays, and microbial sequencing will further elucidate the molecular pathways governing this response.

SUMMARY AND CONCLUSION

Summary

This comparative clinical investigation evaluated the therapeutic impact of non-surgical mechanical periodontal therapy on active oral inflammatory burden, quantified via the Periodontal Inflamed Surface Area (PISA), and perceived psychological stress, assessed via the 10-item Perceived Stress Scale (PSS-10), in adult patients with chronic periodontitis.

Evaluating 500 untreated periodontitis patients (Group B) alongside 500 patients who had received non-surgical mechanical debridement and supportive maintenance (Group C) demonstrated that mechanical instrumentation achieved a 63.2% reduction in vascularized ulcerated epithelial wound area, with mean PISA decreasing from $12.5 \pm 4.3 \text{ cm}^2$ to $4.6 \pm 2.1 \text{ cm}^2$ ($p<0.001$).

Concurrently, perceived stress scores declined significantly from 22.7 ± 5.1 to 17.2 ± 4.5 ($p<0.001$), with high-stress categorizations falling from 24.0% to 7.4%. Importantly, the moderate-to-strong positive correlation between stress and inflammatory surface area observed during active untreated disease ($r=0.62$, $p<0.001$) was absent following mechanical therapy ($r=0.11$, $p=0.142$), reflecting successful clinical healing and disease stabilization.

CONCLUSION

Non-surgical mechanical periodontal therapy significantly reduces the biologically active oral inflammatory surface area (PISA) and is accompanied by a significant reduction in perceived psychological stress.

Effective mechanical debridement decouples the correlation between psychological stress appraisal and active periodontal ulceration, confirming that microbial biofilm disruption remains central to disease management regardless of background stress.

Periodontal treatment delivers clinical benefits that extend beyond mechanical probing depth reductions, improving patient-reported stress profiles and contributing positively to overall well-being.

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