

Original research

Assessing the Role of Serum Creatinine as a Systemic Marker in Chronic Periodontitis Before and After Nonsurgical Periodontal Therapy

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ABSTRACT

Background: Periodontitis is a chronic inflammatory condition that may influence systemic biomarkers, including renal-function markers such as serum creatinine (SCR). Previous studies have reported conflicting associations. This study aimed to investigate the effect of nonsurgical periodontal therapy (NSPT) on serum creatinine levels in systemically healthy individuals with moderate to severe chronic periodontitis.

Materials & Methods: Thirty systemically healthy participants (aged 30–50 years) were recruited and divided into two groups: Group 1 (n=15) with moderate to severe chronic periodontitis and Group 2 (n=15) periodontally healthy controls. Clinical parameters (Plaque Index [PI], Gingival Index [GI], Probing Pocket Depth [PPD], Clinical Attachment Level [CAL]) and serum creatinine levels were recorded at baseline for both groups. Group 1 received NSPT (scaling and root planing), followed by reassessment at 90 days. Data were analysed using paired and independent t-tests ($p < 0.05$).

Results: At baseline, the periodontitis group showed significantly higher serum creatinine (0.6920 ± 0.07163 mg/dL) compared to controls (0.6427 ± 0.03674 mg/dL; $p = 0.025$). PPD and CAL were also significantly higher in Group 1 ($p < 0.001$). After 90 days of NSPT, periodontal parameters improved significantly ($p < 0.05$), and serum creatinine decreased significantly (from 0.6920 ± 0.07163 to 0.6620 ± 0.04346 mg/dL; paired t-test, $p = 0.004$).

Conclusion: Serum creatinine is elevated in systemically healthy individuals with moderate to severe periodontitis and is associated with periodontal severity. Short-term NSPT significantly reduces serum creatinine levels alongside periodontal improvements.

Introduction

Periodontitis is a chronic inflammatory disease resulting in inflammation of the tissues supporting the teeth, progressive loss of clinical attachment, and alveolar bone destruction if untreated¹. It is highly prevalent globally and disrupts systemic homeostasis through persistent low-grade inflammation^{2,3}. Strong bidirectional associations have been established between periodontitis and systemic conditions such as cardiovascular disease, type 2 diabetes mellitus, rheumatoid arthritis, and chronic kidney disease (CKD)^{4–9}.

Serum creatinine (SCR), a waste product of muscle metabolism, is filtered almost exclusively by the

glomeruli and serves as a widely used, cost-effective, and accessible biomarker for estimating glomerular filtration rate (GFR) and assessing renal function^{10,11}. Elevated SCR levels indicate reduced GFR, commonly seen in renal dysfunction or CKD. Emerging evidence suggests that periodontitis-induced systemic inflammation may contribute to subclinical renal strain, even in systemically healthy individuals, making SCR a potential systemic marker of periodontal burden^{12–14}.

Nonsurgical periodontal therapy (NSPT), including scaling and root planing (SRP), remains the cornerstone of periodontal management, effectively reducing local

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inflammation and bacterial load¹⁵. Beyond oral benefits, NSPT has demonstrated systemic effects, such as improved endothelial function, reduced blood pressure, and decreased inflammatory markers (e.g., hs-CRP, IL-6)¹⁶⁻¹⁸. However, its impact on renal biomarkers like SCR in non-CKD populations remains inconsistent, with some studies showing reductions and others no change¹⁹⁻²¹.

The association between periodontitis and SCR, and the influence of NSPT on SCR, are not fully elucidated. Therefore, the aim of this study was to investigate the effect of NSPT on serum creatinine levels in systemically healthy individuals with chronic periodontitis.

Material and methods

This prospective interventional study was conducted at the Department of Periodontology and Oral Implantology, People's Dental Academy, Bhopal.

Thirty systemically healthy, non-smoking participants aged 30 – 50 years with at least 20 natural teeth were recruited from the outpatient department. Inclusion criteria for Group 1 included moderate to severe chronic periodontitis (PPD \geq 5 mm and CAL \geq 3 mm in \geq 2 sites). Exclusion criteria included renal dysfunction (SCR >1.2 mg/dL), periodontal therapy in the past 6 months, smoking, alcohol abuse, pregnancy/lactation, uncontrolled hypertension/diabetes, bleeding disorders, anticoagulant use, antibiotic/NSAID/steroid use in past 3 months, and poor oral hygiene.

Participants were examined by a single calibrated examiner. Clinical parameters (PI [Silness & Løe, 1964], GI [Løe & Silness, 1963], PPD, CAL) were recorded at six sites per tooth using a UNC-15 probe at baseline for both groups and at 90 days for Group 1 only.

Venous blood (5 mL) was collected from the median cubital vein by a trained phlebotomist, processed in the institutional laboratory, and serum creatinine was measured using the kinetic Jaffe method.

Group 1 received full-mouth NSPT (supra- and subgingival SRP with ultrasonic scalers and hand instruments) in staged sessions, followed by oral hygiene instructions (modified Bass technique). Supportive therapy was provided as needed.

Data were analysed using SPSS version 26.0. Independent t-tests compared intergroup differences at baseline; paired t-tests assessed intragroup changes post-NSPT. Significance level was $p < 0.05$.

Results:

At baseline, Group 1 (periodontitis) had significantly higher PPD (4.513 ± 0.2066 mm vs. 1.540 ± 0.4437 mm), CAL (4.607 ± 0.2017 mm vs. 1.540 ± 0.4437 mm), and serum creatinine (0.6920 ± 0.07163 mg/dL vs. 0.6427 ± 0.03674 mg/dL; $p=0.025$) compared to Group 2.

Post-NSPT (90 days), Group 1 showed significant improvements in all periodontal parameters ($p < 0.05$) and a significant reduction in serum creatinine (to 0.6620 ± 0.04346 mg/dL; paired t-test, $p=0.004$).

Discussion

The bidirectional relationship between periodontitis and systemic diseases is well-established, with chronic periodontal inflammation contributing to low-grade systemic effects through mechanisms such as bacterial translocation (e.g., *Porphyromonas gingivalis* and its virulence factors entering the bloodstream), cytokine release (IL-6, TNF- α , CRP), and oxidative stress^{22, 23}. These processes can induce endothelial dysfunction and vascular inflammation, potentially extending to renal tissues and subtly impairing glomerular filtration even in

TABLE 1: COMPARATIVE EVALUATION OF CLINICAL PARAMETERS PRE AND POST NSPT.

<i>Parameters</i>	<i>Group 1</i>		<i>Mean Difference</i>	<i>Statistic</i>	<i>P- value</i>
	<i>Baseline</i>	<i>Postoperative</i>			
<i>SCR VALUE</i>	0.6920+- 0.07163	0.6620+- 0.04346	0.03000	t= 3.416	0.004
<i>GI</i>	2.520+-0.2336	1.6533+- 0.24746	0.86667	t= 9.595	0.000
<i>PI</i>	2.673+- 0.1486	1.7000+-0.34226	0.97333	t= 9.163	0.000
<i>PPD</i>	4.513+- 0.2066	4.2267+-0.19074	0.28667	t= 5.204	0.000
<i>CAL</i>	4.607+-0.2017	4.2800+-0.23361	0.32667	t= 4.666	0.000

TABLE 2: COMPARATIVE EVALUATION OF CLINICAL PARAMETERS BETWEEN THE TWO GROUPS.

<i>Parameters</i>	<i>Group 1</i>	<i>Group 2</i>	<i>Mean Difference</i>	<i>Statistic</i>	<i>P- value</i>
<i>SCR VALUE</i>	0.6920+- 0.07163	0.6427+- 0.03674	0.04933	t= 2.373	0.025
<i>GI</i>	2.520+-0.2336	0.380+-0.1699	2.1400	t= 28.695	0.000
<i>PI</i>	2.673+- 0.1486	0.667+-0.4593	2.0067	t= 16.099	0.000
<i>PPD</i>	4.513+- 0.2066	1.540+-0.4437	2.9733	t= 23.530	0.000
<i>CAL</i>	4.607+-0.2017	1.540+-0.4437	3.0667	t= 24.370	0.000

systemically healthy individuals^{12,14}. Serum creatinine (SCR), as a simple, widely available marker of glomerular filtration rate (GFR), has been investigated as a potential systemic indicator of this periodontal-renal interplay^{10,11}.

In the present study, baseline serum creatinine was significantly higher in the moderate-to-severe chronic periodontitis group (0.6920 ± 0.07163 mg/dL) compared to periodontally healthy controls (0.6427 ± 0.03674 mg/dL; $p=0.025$), alongside markedly worse periodontal parameters (PPD and CAL; $p < 0.001$). This finding aligns with several observational studies reporting elevated SCR in periodontitis patients, suggesting subclinical renal strain from persistent inflammation^{24,25}. For instance, Kshirsagar et al. (2005) and Hattatoglu-Sönmez et al. (2011) documented higher SCR in periodontitis cohorts, attributing it to systemic inflammatory burden^{27,28}. Recent meta-analyses (2018–2023) have reinforced a positive association between periodontitis and chronic kidney disease (CKD) markers, with odds ratios ranging from 1.73 to 4.2 for CKD prevalence in periodontitis patients, even after confounder adjustment^{8,9,16,27}.

However, conflicting evidence exists. Shimazaki et al. (2013) reported an inverse association in Japanese middle-aged males, where higher SCR correlated with lower PPD/CAL, possibly due to population-specific factors (e.g., male-only cohort, ethnicity, or compensatory hyperfiltration in early disease)³⁰. Brotto et al. (2019) found no association using multiple renal markers in systemically healthy individuals, suggesting SCR may lack sensitivity for detecting subtle periodontal effects in non-CKD populations²⁵. These discrepancies likely stem from differences in periodontitis staging (e.g., 1999 vs. 2017 classifications), sample

demographics, and confounders like BMI/muscle mass (controlled here via exclusion criteria).

The key novel finding of this study was the significant reduction in serum creatinine following nonsurgical periodontal therapy (NSPT) in the periodontitis group (from 0.6920 ± 0.07163 to 0.6620 ± 0.04346 mg/dL; paired t-test, $p=0.004$), accompanied by robust improvements in all clinical parameters ($p < 0.05$). This suggests that reducing local periodontal inflammation and bacterial load via scaling and root planing may confer short-term systemic benefits on renal biomarkers, even in otherwise healthy individuals. NSPT is the gold standard for controlling periodontal infection, with well-documented reductions in gingival inflammation, probing depths, and systemic inflammatory markers (hs-CRP, IL-6)¹⁵⁻¹⁸.

This result contrasts with some prior interventional studies in systemically healthy populations. For example, a 2023 interventional study ($n=68$) reported higher baseline SCR in stage II/III periodontitis but no significant change post-NSPT ($p=0.12$) despite periodontal improvements³¹. Similarly, Graziani et al. (2010) observed no SCR alteration at 180 days follow-up in healthy subjects²⁸. Meta-analyses on NSPT in CKD patients have shown variable renal effects—some report modest eGFR improvements or reduced urinary albumin, while others find no change in SCR, possibly due to longer disease duration or irreversible damage^{19,20,26}. The significant SCR reduction here may reflect earlier intervention in non-CKD individuals, where inflammatory burden is reversible, or the 90-day timeframe capturing acute post-treatment effects before potential rebound.

Mechanistically, NSPT likely mitigates systemic inflammation, reducing cytokine-driven endothelial stress in renal vasculature and improving GFR

transiently^{22,23}. However, SCR's limitations (influence of muscle mass, diet, hydration) and the study's short follow-up may explain why effects are modest (mean decrease ~0.03 mg/dL). No direct correlation analysis was performed between SCR changes and periodontal improvements, but the parallel reductions suggest a plausible link.

Strengths of this study include strict inclusion/exclusion criteria (excluding smokers, uncontrolled systemic conditions), single calibrated examiner, and standardized NSPT protocol. Limitations include small sample size (n=30), relatively short 90-day follow-up (longer periods might reveal sustained or rebound effects), lack of additional renal markers (e.g., cystatin C, eGFR, urinary albumin), and no blinding of the examiner (though blood analysis was lab-based). Future research should employ larger cohorts, longer follow-ups (>6–12 months), multifaceted renal panels, and inflammatory mediators to clarify causality and dose-response relationships.

In conclusion, this study provides evidence that moderate-to-severe periodontitis in systemically healthy adults is associated with elevated serum creatinine, and short-term NSPT can significantly lower SCR alongside periodontal healing. These findings support the concept of periodontitis as a modifiable contributor to subtle systemic renal effects and highlight the potential broader health benefits of periodontal care.

Conclusion

In systemically healthy individuals, moderate to severe periodontitis is associated with elevated serum creatinine. Short-term NSPT significantly reduces SCR levels while improving periodontal health, suggesting potential systemic renal benefits.

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