ORIGINAL ARTICLE

Prevalence of periodontitis and systemic inflammation assessed by C reactive protein in Sudanese hemodialysis patients with end stage renal disease

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ABSTRACT

Background: A potential bidirectional link between periodontitis and systemic diseases, including end-stage renal disease (ESRD), has long been speculated. Elevated systemic inflammation in ESRD patients may be exacerbated by chronic periodontal infection. This study aimed to assess the prevalence of periodontitis and its association with systemic inflammation, measured by serum C-reactive protein (CRP), in Sudanese patients receiving hemodialysis.

Methods: A multicenter, hospital-based, analytic cross-sectional study was conducted at six dialysis centers in Khartoum. One hundred ESRD patients aged 25–45 years, on maintenance hemodialysis, were recruited. Periodontal examination included plaque index (PI), bleeding on probing (BOP), probing pocket depth (PPD), and clinical attachment loss (CAL). Serum CRP levels were measured using immunofluorescent assay (cutoff >10 mg/L).

Results: The prevalence of periodontitis was 83%, with moderate cases being most common (47%). Mean CRP level in periodontitis patients was 7.89 ± 7.92 mg/L. No statistically significant association was found between periodontitis and elevated CRP (P = 0.99), nor between CRP and periodontal parameters (PI, BOP, PPD, CAL). However, CAL was significantly associated with gender (P = 0.036) and showed marginal association with dialysis duration (P = 0.242).

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Conclusion: Periodontitis is highly prevalent among ESRD patients on hemodialysis in Sudan, yet it was not found to be associated with elevated systemic inflammation as measured by CRP. Further studies with larger sample sizes and additional biomarkers are warranted to explore this relationship more deeply. (www.actabiomedica.it)

Key words: periodontitis, end-stage renal disease, C-reactive protein, hemodialysis, Sudan, systemic inflammation

Introduction

Chronic kidney disease (CKD) and periodontitis are progressive, inflammation-driven conditions that cumulatively impose a substantial public health burden worldwide (1). CKD affects nearly 10% of the global population, with end-stage renal disease (ESRD) representing its most advanced stage and associated with disproportionately high morbidity, mortality, and healthcare costs (2). According to the Global Burden of Disease (GBD) Study 2023, CKD ranks among the top 10 causes of death globally, with more than 4.6 million deaths annually attributable to impaired kidney function, reflecting a sustained rise over the past three decades (3). Mortality is disproportionately higher in low- and middle-income countries, where late detection, limited dialysis availability, and socioeconomic barriers exacerbate outcomes (4). In Sudan, CKD has emerged as a major noncommunicable disease challenge, with recent reports indicating a rising prevalence of risk factors such as hypertension, diabetes, and infectious diseases, and an increased dependence on hemodialysis as the primary modality for renal replacement therapy (5). Periodontitis, similarly, is a chronic, biofilm-mediated inflammatory disease leading to progressive destruction of periodontal tissues (6). It affects more than 1.1 billion people globally and has been consistently ranked by GBD as the sixth most prevalent human disease (7,8). Recent epidemiological data from Sub-Saharan Africa, including Sudan, demonstrate a high burden of periodontal disease driven by limited access to preventive dental services, low oral health awareness, socioeconomic constraints, and the presence of systemic comorbidities (9,10). Sudanese studies report a high prevalence of moderate to severe periodontitis among adults, yet national data remain fragmented, and vulnerable populations—such as individuals undergoing hemodialysis—are underrepresented in the literature (11,12). A growing body of evidence indicates a bidirectional relationship between CKD and periodontitis, mediated largely through systemic inflammation, immune dysregulation, endothelial dysfunction, and microbial translocation (13). Patients with ESRD exhibit impaired neutrophil function, altered cytokine profiles, and increased systemic oxidative stress, all of which heighten susceptibility to periodontal breakdown (13). In parallel, periodontal infection contributes to a sustained systemic inflammatory load, characterized by elevated circulating inflammatory mediators that may further exacerbate renal dysfunction. This interplay is clinically relevant because hemodialysis patients frequently present with worsened periodontal status, poor oral health-related quality of life, and increased risk of cardiovascular complications (14,15). C-reactive protein (CRP)—an acute-phase reactant strongly associated with cardiovascular and renal outcomes—serves as a key biomarker linking oral and systemic inflammation (16). Elevated CRP levels are well documented in ESRD patients due to chronic inflammation, recurrent infections, and dialysis-related factors (17). Periodontitis independently elevates CRP concentrations through sustained systemic dissemination of periodontal pathogens and their by-products (18). As such, assessing CRP in hemodialysis populations provides clinically meaningful insights into systemic inflammatory burden and may reflect modifiable oral health-related contributors. However, CRP-based characterization of inflammatory status among ESRD patients in Sudan is scarce, and its association with

periodontal disease severity has not been adequately explored (18). In this study, systemic inflammation was operationalized using CRP, which serves as a widely validated biomarker in ESRD research, although additional markers such as IL-6 and TNF-α were not measured. Although high-sensitivity CRP (hsCRP) provides finer discrimination at lower inflammatory ranges, resource constraints and laboratory availability in Sudanese dialysis centers necessitated the use of standard CRP assays, which remain widely used in ESRD clinical monitoring. Despite the compelling biological rationale and global data, there is a critical gap in Sudanese research regarding the coexistence of periodontitis and systemic inflammation among patients receiving hemodialysis (19). This gap is particularly concerning given the rising prevalence of ESRD in Sudan, resource limitations in renal care, and the absence of integrated oral–systemic health strategies within dialysis centers. Understanding the periodontal status and inflammatory burden of this high-risk population is essential for designing context-specific preventive protocols, improving multidisciplinary management, and reducing avoidable morbidity. Therefore, this study aimed to (i) determine the prevalence and severity of periodontitis among Sudanese patients with end-stage renal disease undergoing maintenance hemodialysis, and (ii) evaluate the association between periodontal disease status and systemic inflammation using serum C-reactive protein (CRP) as the primary biomarker. This approach was intended to clarify whether periodontal disease contributes measurably to systemic inflammatory burden in a population already characterized by chronic inflammation.

Patients and Methods

Study design

An analytic, observational, cross-sectional, hospital-based study was conducted to assess the periodontal status and systemic inflammatory burden among Sudanese patients with end-stage renal disease (ESRD) undergoing maintenance hemodialysis. The study specifically aimed to quantify the prevalence of periodontal disease and to examine its association with systemic inflammation, operationalized by serum CRP levels, in patients undergoing long-term hemodialysis.

Study area

The study was carried out in major renal hemodialysis centers across Khartoum, Sudan, including Alwaledain Hospital, Algameia Hospital, the Tropical Disease Hospital, the Military Hospital, Alnaow Hospital, and Albogaa Dialysis Center.

Study population

The study population consisted of adult patients diagnosed with ESRD and receiving hemodialysis as part of their renal replacement therapy. All participants were regular attendees of the selected dialysis centers.

Inclusion criteria

- Participants were eligible if they:
- Were diagnosed with ESRD and undergoing hemodialysis.
- Were between 25 and 45 years of age.
- Had been on hemodialysis for at least 3 months.
- Possessed at least 20 natural teeth.

Exclusion criteria

- Participants were excluded if they:
- Received periodontal treatment within the previous 3 months.
- Had systemic diseases other than ESRD. (Patients with uncontrolled diabetes or diabetes-associated acute inflammatory complications were excluded).
- Were pregnant or using contraceptive pills.
- Had taken antibiotics or anti-inflammatory medications within the past 3 months.
- Were edentulous.

Sample size

A minimum sample size of 92 was required to detect a medium effect size (Cohen's d = 0.5) with 80% power at α = 0.05 for CRP differences between

periodontal groups. We recruited 100 participants to compensate for potential variability.

Ethical approval and patient consent

This study was reviewed and approved by the Ethical Committee of the Sudan Medical Specialization Board (SMSB). The committee granted ethical clearance on 03 December 2019, which serves as the official approval reference (Approval Reference: SMSB/EC/03-12-2019). All study procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki, the Council for International Organizations of Medical Sciences (CIOMS) guidelines for research involving vulnerable patients, and national research ethics regulations.

Prior to enrolment, all participants received a complete explanation of the study objectives, clinical procedures, potential risks, and their rights as research subjects. Written informed consent was obtained from each participant before data collection. For patients undergoing renal dialysis, special ethical considerations were applied due to their classification as a medically vulnerable population. Participation was entirely voluntary, with explicit assurance that refusal or withdrawal from the study would not affect the quality of their medical or dialysis care. Clinical examinations and sample collection were performed in a manner that did not interfere with dialysis schedules or compromise patient safety. Confidentiality of all personal and clinical data was strictly maintained. Each participant was assigned a unique study code, and no identifying information was used in data analysis or reporting. The researchers adhered strictly to standards for the safe handling of biological samples, and all procedures minimized discomfort and prevented cross-infection, in accordance with recommended infection-control measures for immunocompromised patients.

Data collection procedures

Examiner training and calibration

All periodontal examinations were performed by a single calibrated examiner. Intra-examiner reliability was assessed prior to data collection using repeated measurements on 10 patients, yielding an intraclass correlation coefficient (ICC) of 0.80, indicating substantial agreement.

Clinical periodontal examination

A comprehensive periodontal examination was conducted for each participant, assessing:

- Plaque Index (PI): Recorded on four sites per tooth (mesiobuccal, distobuccal, mid-buccal, palatal/lingual) using the Silness and Löe (1964) criteria (20).
- Bleeding on Probing (BOP): Recorded dichotomously (+/–) at four sites per tooth.
- Probing Pocket Depth (PPD): Measured from the gingival margin to the base of the periodontal pocket using a Williams periodontal probe. A PPD ≥ 4 mm was indicative of periodontitis.
- Clinical Attachment Loss (CAL): Measured from the cementoenamel junction (CEJ) to the base of the pocket at four sites per tooth. CAL ≥ 2 mm was used to diagnose periodontitis.

Periodontal diagnosis followed the 2018 AAP/EFP Classification criteria, using CAL as the primary diagnostic threshold and PPD as the indicator of current inflammation (21).

Blood sample collection and CRP measurement

All samples were collected immediately before the dialysis session to minimize variability associated with dialysis-induced inflammatory shifts. Venous blood samples were collected using a sterile syringe into plain containers. Samples were kept in ice-cooled transport boxes and delivered to the laboratory within the same day. Serum was separated after centrifugation and analyzed within 24 hours.

CRP levels were quantified using an immunofluorescent assay (ichroma™) following manufacturer instructions. The detection buffer contained anti-human CRP fluorescence conjugate and anti-rabbit IgG fluorescence tracer. Two drops of the reaction mixture were

applied to the test device, incubated for 3 minutes, and subsequently analyzed to obtain CRP values (mg/L).

CRP INTERPRETATION

- Normal for ESRD patients: ≤ 10 mg/L
- Elevated: > 10 mg/L
- For healthy individuals, CRP < 2 mg/L was considered normal.
- Patients were examined during one of the three daily dialysis sessions (8–10 AM, 12–3 PM, 4–7 PM).

Statistical analysis

Data were analyzed using SPSS version 20 (SPSS Inc., Chicago, IL, USA). Descriptive statistics (means, standard deviations, frequencies, and percentages) were calculated for demographic, periodontal, and biochemical variables.

Analytical tests included:

- Independent t-test to compare CRP levels between periodontitis and gingivitis groups
- One-way ANOVA to assess differences in CRP across disease severity categories
- Chi-square test for associations between categorical variables
- Logistic regression for evaluating the effect of gender and dialysis duration on periodontal parameters
- Pearson correlation to assess relationships between periodontal parameters (PI, BOP, PPD, CAL) and CRP levels

A P-value < 0.05 was considered statistically significant.

Results

Demographic characteristics

A total of 100 ESRD patients undergoing hemodialysis were included in the study. The mean age was 34.46 ± 7.36 years, and 60% (n = 60) were male, while 40% (n = 40) were female. Only 7% of the sample had diabetes (all of whom had controlled disease without acute inflammatory complications, in accordance with the exclusion criteria), 18% were smokers, and 9% were snuff dippers (Table 1). All participants used a toothbrush for oral hygiene. The brushing frequency was once daily in 57%, twice daily in 35%, three times daily in 6%, and more than three times daily in 2%.

Clinical periodontal parameters

The mean (PI) was 1.85 ± 0.25**,** (BOP) was 1.16 ± 0.17**,** (PPD) was 1.35 ± 0.48 mm**,** and (CAL) was 1.39 ± 1.31 mm (Table 2)**.**

Based on periodontal diagnosis, 17% of the patients had gingivitis, whereas 83% were diagnosed with periodontitis (Figure 1). Among those with periodontitis:

- **18%** had mild periodontitis
- **47%** had moderate periodontitis (most common)
- **10%** had severe periodontitis

Periodontal clinical parameters by gender

Comparison of periodontal parameters between male and female participants showed no significant gender-related differences in PI, BOP, or PPD

Table 1. Demographic characteristics.

Variable	n (%) / Mean ± SD
Age (years)	34.46 ± 7.36
Male	60 (60%)
Female	40 (40%)
Smokers	18 (18%)
Snuff dippers	9 (9%)
Diabetics	7 (7%)

Table 2. Periodontal parameters.

Parameter	Mean ± SD
PI	1.85 ± 0.25
BOP	1.16 ± 0.17
PPD	1.35 ± 0.48
CAL	1.39 ± 1.31

(P = 0.406, 0.404, and 0.910, respectively). However, a significantly higher CAL was observed among males compared with females (1.629 ± 1.568 vs. 0.980 ± 0.437; P = 0.036). This indicates that male patients exhibited greater periodontal attachment loss despite similar plaque accumulation and gingival inflammation levels (Table 3).

Periodontal parameters in relation to dialysis duration

When periodontal parameters were stratified according to duration of dialysis, no significant differences were observed across the three groups in BOP, PI, or CAL (P = 0.480, 0.534, and 0.242, respectively). However, a significant association was detected for PPD (P = 0.022), with patients undergoing dialysis for 3–5 years demonstrating greater pocket depths compared to those with shorter or longer dialysis durations (Table 4).

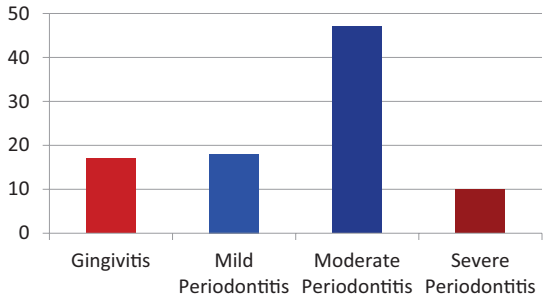

Figure 1. Distribution of periodontal diagnoses among the study population.

C-Reactive Protein (CRP) levels among study groups

CRP across periodontitis severity

The mean CRP levels for each periodontal category were:

- **Gingivitis:** 9.65 mg/L
- **Mild periodontitis:** 9.91 mg/L
- **Moderate periodontitis:** 10.79 mg/L
- **Severe periodontitis:** 12.97 mg/L

Although CRP increased progressively from gingivitis to severe periodontitis, one-way ANOVA showed no statistically significant difference (P = 0.765) (Table 5).

Table 3. Comparison of periodontal clinical parameters between male and female chronic renal failure patients.

Clinical Parameter	Male (Mean ± SD)	Female (Mean ± SD)	P-value
PI	1.871 ± 0.320	1.821 ± 0.249	0.406
BOP	1.178 ± 0.193	1.149 ± 0.139	0.404
PPD	1.328 ± 0.415	1.345 ± 0.613	0.910
CAL	**1.629 ± 1.568**	**0.980 ± 0.437**	**0.036***

Mean values and standard deviations (Mean ± SD) of plaque index (PI), bleeding on probing (POB), probing pocket depth (PPD), and clinical attachment loss (CAL) are presented for both genders. Asterisk (*) denotes statistically significant differences at P ≤ 0.05.

Table 4. Periodontal parameters among chronic renal failure patients according to duration of hemodialysis.

Clinical Parameter	3 months–2 years (Mean ± SD)	3–5 years (Mean ± SD)	>5 years (Mean ± SD)	P-value
BOP	1.445 ± 0.189	1.196 ± 0.145	1.166 ± 0.177	0.480
PI	1.825 ± 0.273	1.832 ± 0.307	1.898 ± 0.306	0.534
PPD	**0.093 ± 0.109**	**0.258 ± 0.322**	**0.199 ± 0.272**	**0.022***
CAL	0.389 ± 0.409	0.610 ± 1.094	0.328 ± 0.421	0.242

Values represent mean ± standard deviation (SD) of bleeding on probing (POB), plaque index (PI), probing pocket depth (PPD), and clinical attachment loss (CAL) across three dialysis-duration categories (3 months–2 years, 3–5 years, and >5 years). Asterisk (*) indicates statistically significant differences at P ≤ 0.05.

A one-way ANOVA showed no statistically significant difference in CRP levels among the four groups (P = 0.765). However, CRP increased numerically as severity increased.

CRP: GINGIVITIS VS. PERIODONTITIS

When grouped as gingivitis vs. any stage of periodontitis:

- **Periodontitis:** 7.89 ± 7.92 mg/L
- **Gingivitis:** 6.28 ± 7.31 mg/L

An independent t-test showed no significant difference between these two groups (P = 0.99) (Table 6).

Table 5. CRP across periodontitis severity.

Severity	Mean CRP (mg/L)
Gingivitis	9.65
Mild	9.91
Moderate	10.79
Severe	12.97

Table 6. CRP: Gingivitis vs Periodontitis.

Group	Mean CRP (mg/L)	P-value
Gingivitis	6.28 ± 7.31	
Periodontitis	7.89 ± 7.92	**0.99**

LOGISTIC REGRESSION FOR ELEVATED CRP (>10 MG/L)

Binary logistic regression was used to evaluate whether demographic or clinical variables predicted elevated CRP levels (>10 mg/L). The following variables showed **borderline associations:**

- **Smoking:** P = 0.054
- **Snuff dipping:** P = 0.081
- **Male gender:** P = 0.072
- **Dialysis duration:** P = 0.052
- Periodontitis severity, PI, BOP, PPD, and CAL were **not significantly associated** with CRP levels (Table 7).

CORRELATION BETWEEN PERIODONTAL PARAMETERS AND CRP

Pearson correlation analysis demonstrated **no statistically significant association** between serum CRP levels and any of the periodontal parameters assessed. There was no correlation between CRP and Plaque Index (PI) (P = 0.395), Bleeding on Probing (BOP) (P = 0.959), Probing Pocket Depth (PPD) (P = 0.972), or Clinical Attachment Loss (CAL) (P = 0.220).

Discussion

The present study investigated the prevalence of periodontitis and its relationship with systemic

Table 7. Logistic regression/ Predictors of elevated CRP (>10 mg/L).

Variable	P-value	Interpretation
Smoking	0.054	Borderline predictor of elevated CRP
Snuff dipping	0.081	Trend toward elevated CRP
Gender (Male)	0.072	Males tended toward higher CRP
Dialysis duration	0.052	Longer dialysis showed trend toward lower CRP
Periodontitis severity	0.765	Not a predictor of CRP
PI	0.395	Not associated
BOP	0.959	Not associated
PPD	0.972	Not associated
CAL	0.220	Not associated

A binary logistic regression model evaluated the effect of demographic and clinical variables on CRP.

inflammation, as assessed by serum C-reactive protein (CRP), among patients with end-stage renal disease (ESRD) undergoing hemodialysis in Khartoum, Sudan.

Prevalence and severity of periodontitis

In this study, 83% of hemodialysis patients had periodontitis, with moderate periodontitis (47%) representing the most common form. Although mean PPD values were low, CAL values indicated early periodontal breakdown typical of ESRD patients in younger age groups, consistent with uremia-related attachment loss without extensive pocketing. This is in strong agreement with previous studies demonstrating an elevated prevalence of periodontal diseases among patients on long-term dialysis (22,23). Altamimi et al. reported that hemodialysis patients exhibit greater susceptibility to moderate and severe forms of periodontal disease due to impaired host defenses, salivary dysfunction, and chronic uremic toxicity (24). Similar observations were made by Rani et al., who identified widespread periodontal destruction among dialysis patients, often exceeding that seen in healthy controls of comparable age (25). Notably, the patients in the present study were relatively young (25–45 years) yet already exhibited considerable periodontal breakdown. This observation is biologically plausible, as ESRD and its associated metabolic and immunological alterations, uremia-induced immune dysfunction, chronic oxidative stress, xerostomia, fluid shifts, and dietary restrictions, are known to accelerate periodontal disease progression (26). The presence of comorbid risk factors such as smoking and snuff use further increases susceptibility. Thus, the high prevalence of disease in this age group supports the notion that renal failure may contribute to earlier and more aggressive periodontal deterioration. The comparison of periodontal parameters between male and female patients revealed that plaque levels, gingival inflammation, and probing pocket depth did not differ significantly between genders. These findings indicate that basic oral hygiene status and superficial inflammatory changes were relatively similar in both groups. Previous studies in both general and medically compromised populations have similarly shown that gender alone does not strongly influence plaque accumulation or gingival indices when oral hygiene behaviors and clinical conditions are comparable (27). This suggests that behavioral factors such as oral hygiene practices, diet, or smoking rather than biological sex alone are usually responsible for variations in PI and BOP in most populations (28). However, a significant difference was evident in CAL, which was markedly higher among male patients. This pattern is consistent with existing periodontal epidemiology, where males often exhibit more severe periodontal tissue destruction than females, despite having similar plaque scores (29). Several biological explanations support this observation. Male patients may have a stronger pro-inflammatory response to chronic plaque exposure, higher baseline neutrophil activity, and generally weaker oral health–seeking behaviors compared to females. In addition, sex hormone–related modulation of the immune system can influence the progression of chronic inflammation. Estrogen has been shown to exert protective anti-inflammatory and antioxidant functions on gingival tissues, whereas androgens may predispose to more severe connective-tissue breakdown (30,31). In the context of renal failure patients, this difference becomes even more relevant. Chronic kidney disease (CKD) alters systemic immunity, oxidative stress levels, and circulating inflammatory mediators such as IL-6 and CRP (32). Male patients may be more susceptible to accelerated periodontal destruction under these dysregulated immune conditions. Therefore, the significantly greater CAL among males in this study likely reflects both the systemic vulnerability imposed by CKD and inherent gender-based susceptibility patterns documented in periodontal disease literature. A significant association was found between probing pocket depth (PPD) and dialysis duration, with patients undergoing dialysis for 3–5 years showing deeper pockets than those with shorter or longer exposure. This non-linear pattern has been described in prior nephrology-periodontology studies, which found that periodontal conditions may worsen during intermediate stages of dialysis due to cumulative inflammatory burden, fluctuations in serum calcium–phosphate balance, immune dysfunction, and worsening nutritional status during the mid-dialysis years (33). As dialysis duration increases beyond five years, some stabilization may occur because long-term

survivors typically represent a healthier subgroup with better immune adaptation, improved compliance with medical follow-up, and greater resilience to metabolic complications (33). Thus, the observed increase in PPD during the 3–5-year period may represent a time-window of heightened vulnerability where systemic inflammatory markers such as CRP and IL-6, in addition to oxidative stress, peak before stabilizing or being better managed with long-term dialysis adaptation (34). Interestingly, CAL did not differ significantly across dialysis durations, which may be explained by minimal gingival recession in this population. In many CKD patients, CAL and PPD are nearly equivalent, because gingival overgrowth, edema, and hyperplastic changes prevent recession from manifesting (35). Therefore, attachment loss measurements may appear stable even when pocket depths fluctuate temporarily.

CRP among gingivitis and different periodontitis groups

Although CRP values increased progressively from gingivitis to mild, moderate, and severe periodontitis, the differences were not statistically significant. Likewise, the comparison between gingivitis and periodontitis yielded no significant elevation in CRP among periodontitis patients. These findings contrast with several studies that reported significantly higher CRP levels in dialysis patients with periodontitis, suggesting that periodontal inflammation may contribute to systemic inflammatory burden in this population (36, 37). For instance, several authors have proposed periodontitis as an underrecognized inflammatory stimulus capable of elevating CRP and exacerbating cardiovascular risk in ESRD (38-40). However, our results are in line with other studies that found only weak or non-significant associations once confounding clinical factors were accounted for (40,41). The discrepancy between studies likely stems from several important considerations. First, ESRD itself is a state of persistent systemic inflammation, driven by factors such as dialysis membrane biocompatibility, recurrent infections, vascular access inflammation, malnutrition-inflammation complex, and cardiovascular comorbidities. This chronic inflammatory "background noise" may overshadow the comparatively smaller contribution of periodontal inflammation. Second, many studies that reported significant effects used high-sensitivity CRP (hsCRP) assays, which detect subtle changes in CRP in the range of 1–10 mg/L (41). In contrast, the current study used a standard CRP assay with a cutoff of >10 mg/L as elevated for ESRD patients. It is therefore possible that small but clinically relevant variations attributable to periodontal disease were not detected. Third, the relatively modest sample size particularly in the gingivitis and severe periodontitis groups, may have limited statistical power to detect differences. Moreover, the cross-sectional nature of the study captures CRP at a single time point, whereas CRP in ESRD patients fluctuates in response to intercurrent illness, dialysis efficiency, and other acute factors.

Predictors of elevated CRP

The logistic regression analyses revealed that smoking, snuff dipping, male gender, and dialysis duration showed borderline associations with elevated CRP, whereas periodontal severity and periodontal parameters did not. This pattern is consistent with existing research indicating that lifestyle and systemic factors play a dominant role in determining inflammatory burden in ESRD, far more than periodontal status alone (41,43). Smoking is a well-established mediator of systemic inflammation and cardiovascular risk, and its borderline association with elevated CRP in this cohort aligns with its known immunomodulatory effects (44). Similarly, dialysis duration has a complex and sometimes nonlinear relationship with systemic inflammation, with some studies reporting increased inflammation with longer dialysis and others showing stabilization with adequate dialysis management (41,45).

The absence of a significant predictive effect of periodontal disease supports the hypothesis that, although periodontal inflammation contributes to the overall inflammatory milieu, its individual impact is relatively small in the context of ESRD, where numerous stronger drivers of inflammation coexist.

Correlation between periodontal parameters and CRP

This study found no significant correlation between CRP and PI, BOP, PPD, or CAL. Existing

literature demonstrates mixed findings in this area. Some authors have reported strong associations between CRP and measures of periodontal inflammation such as the periodontal inflamed surface area (PISA) (46,47), while others reported weak or absent correlations, particularly in medically complex populations (48,49). The discrepancy is likely due to differences in measurement systems: traditional indices such as mean PPD or CAL provide only partial representations of the inflammatory burden, whereas quantitative metrics like PISA capture the total inflamed periodontal surface area, which correlates more strongly with systemic markers. Thus, the lack of correlation observed here may reflect limitations of the periodontal indices used or the masking effect of high systemic inflammation in ESRD. The fact that CRP is influenced by multiple non-oral factors in this population further reduces the likelihood of detecting direct linear correlations with periodontal measures (50). The findings of this study suggest that, while periodontal disease is highly prevalent and often severe among hemodialysis patients, its independent effect on systemic CRP is modest in the context of ESRD-associated inflammation. This does not diminish the importance of periodontal care in this vulnerable group, as improving periodontal health may still reduce local infection burden, enhance quality of life, and potentially contribute to long-term reductions in inflammation when combined with optimized dialysis management. However, the results indicate that CRP alone may not be sufficiently sensitive to detect periodontal contributions to inflammation in ESRD patients, especially when measured with standard assays. Despite the important insights provided, this study has several limitations that should be acknowledged. First, the cross-sectional design limits the ability to establish temporal or causal relationships between periodontal disease and systemic inflammation. A longitudinal follow-up or an interventional periodontal therapy trial would better clarify the directionality of this association. Second, although the sample size was adequate for preliminary analysis, the distribution of patients across periodontal severity categories—especially the gingivitis and severe periodontitis groups—was uneven, which may have reduced the power to detect statistically significant differences in CRP. Third, the use of a standard CRP assay rather than a high-sensitivity CRP (hsCRP) test may have masked subtle inflammatory changes attributable to periodontal disease, particularly within the low-grade inflammatory range common in chronic conditions. Fourth, periodontal assessment relied on traditional indices (PI, BOP, PPD, CAL), which do not quantify the total inflamed periodontal surface area; newer metrics such as PISA or PESA might have offered a more precise reflection of the periodontal inflammatory burden. Finally, ESRD patients often experience numerous non-oral inflammatory stimuli—including dialysis-related factors, nutritional status, oxidative stress, and comorbid infections—which may overshadow the contribution of periodontal inflammation to systemic CRP. A further limitation is the absence of a comparison group of ESRD patients without periodontitis, which could have allowed a clearer differentiation of periodontal contributions to systemic CRP levels independent of dialysis-related inflammation. These limitations underscore the need for larger studies using more sensitive inflammatory biomarkers and more comprehensive periodontal inflammation measures to further elucidate the relationship between periodontitis and systemic inflammation in ESRD populations.

Conclusion

This study demonstrates that periodontitis is highly prevalent among Sudanese patients undergoing hemodialysis, with moderate disease representing the most common presentation even in a relatively young ESRD population. Although serum CRP levels increased numerically with greater periodontal severity, no statistically significant association was observed between CRP and periodontal diagnosis, severity, or individual periodontal parameters. In contrast, lifestyle and systemic factors—such as smoking, snuff use, male gender, and dialysis duration—showed a stronger influence on systemic inflammation than periodontal status. These findings suggest that, within the complex inflammatory environment of ESRD, the contribution of periodontal disease to systemic CRP may be relatively modest and difficult to detect using routine inflammatory assays. Nevertheless, the high burden

of periodontitis observed reinforces the need for integrating periodontal screening and preventive dental care into the routine management of hemodialysis patients. Future research using larger samples, longitudinal designs, and more sensitive inflammatory markers (such as hsCRP) is warranted to clarify the potential systemic impact of periodontal treatment in this medically vulnerable population.

Ethic Approval: This study was reviewed and approved by the Ethical Committee of the Sudan Medical Specialization Board (SMSB). The committee granted ethical clearance on 03 December 2019, which serves as the official approval reference (**Approval Reference: SMSB/EC/03-12-2019**).

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g., consultancies, stock ownership, equity interests, patent/licensing, arrangement etc-) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: SHM and BGG contributed to the conception and design of the study. SHM and SMAA were responsible for data acquisition and clinical examination of the participants. NTH and MMR performed data analysis and interpretation. SHM, NTH, and BGG drafted the initial version of the manuscript. NTH and MMR critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

Declaration on the Use of AI: None

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