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Systemic and lifestyle determinants of periodontitis: Evaluating the influence of smoking, obesity, erythrocyte sedimentation rate, and total leukocyte count

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Abstract

Introduction: Periodontitis is a prevalent multifactorial inflammatory disease caused by lifestyle factors such as smoking and obesity. The erythrocyte sedimentation rate (ESR) is a systemic inflammatory marker, but its relationship with periodontitis, smoking status, and obesity remains underexplored.

Objective: This study evaluates the interrelationships among smoking status, anthropometric indices (body mass index [BMI] and waist circumference [WC]), hematological indices (ESR, hemoglobin [Hb], and total leukocyte count [TLC]), and periodontal indices.

Methods: This cross-sectional study included 45 healthy adults, divided into three groups (n = 15 each): smokers with generalized moderate chronic periodontitis, non-smokers with chronic periodontitis, and periodontally healthy controls. Clinical assessments included anthropometric indices, inflammatory hematological indices, and periodontal indices. Statistical analysis was conducted using the Statistical Package for Social Sciences software, with significance set at $p < 0.05$.

Results: ESR levels were significantly higher in smokers with periodontitis (20 mm/hr) compared to non-smokers with periodontitis (14.33 mm/hr) and healthy controls (9.33 mm/hr), demonstrating a dose-dependent association with cigarette consumption. BMI was highest in smokers (29.47 kg/m^2), followed by non-smokers (25.35 kg/m^2) and controls (19.25 kg/m^2). Reduced Hb levels were observed in smokers, while TLC showed no significant variation among groups.

Conclusion: In the presence of both periodontitis and obesity, smoking appears to exert a synergistic effect, resulting in heightened systemic inflammation and worsened periodontal tissue damage, as evidenced by elevated ESR and diminished Hb levels, independent of changes in TLC. These findings underscore the importance

of incorporating lifestyle factors and systemic inflammatory markers into periodontal assessment to facilitate comprehensive patient management.

Keywords: Body mass index; Erythrocyte sedimentation rate; Hemoglobin; Obesity; Chronic periodontitis; Smoking; Systemic inflammatory biomarker

1. Introduction

Periodontitis is a chronic progressive inflammatory disease that affects more than 1.1 billion people globally, contributes to significant oral and systemic morbidity, and imposes a substantial socioeconomic burden.[1,2] Earlier, its pathogenesis was thought to be driven solely by microbial challenge, but recent evidence shows it also involves an interplay between dysbiotic biofilms and systemic or lifestyle modifiers.[3,4] Modifiers like alcohol, smoking, or tobacco consumption, and metabolic disturbances such as obesity and diabetes, are well-established determinants of disease susceptibility and severity. Approximately a 2.7-fold higher likelihood of developing periodontitis is exhibited among smokers. Smoking compromises immune function and wound healing and is associated with neutrophil dysfunction, oxidative stress, and dysbiosis that favor pathogens such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Fusobacterium* spp., further masking clinical signs of inflammation and complicating diagnosis.[3-7]

Obesity contributes to periodontal breakdown through metaflammation and adipokine production, including tumor necrosis factor alpha and interleukins (IL-6 and IL-1β), which amplify osteoclastogenic pathways and immune dysregulation. Anthropometric indices such as body mass index (BMI) and waist circumference (WC) correlate positively with clinical attachment loss (CAL), whereas newer measures such as weight-adjusted waist index (WWI) and waist-to-height ratio (WtHR) demonstrate superior predictive ability.[8]

Hematological inflammatory indices, such as the erythrocyte sedimentation rate (ESR), have gained importance, with elevated ESR reflecting increased acute-phase reactants and being frequently higher in individuals with periodontitis, particularly in smokers and those with metabolic comorbidities. Alterations in these indices, along with reduced hemoglobin (Hb) and erythrocyte count, further support the systemic nature of periodontal inflammation and its resemblance to anemia of chronic disease.[9,10]

Evidence regarding the potential synergistic effects of smoking, obesity, and systemic inflammation on periodontal health remains limited, especially in regional populations. The present study addressed this gap and evaluated the interrelationships among smoking status, anthropometric indices, and hematological inflammatory indices in individuals with and without chronic periodontitis. By comparing clinical, physiological, and systemic parameters across distinct risk groups, this study aimed to clarify how modifiable lifestyle factors collectively shape periodontal disease expression and inflammatory load.

2. Materials and methods

2.1. Study design and setting

This was a cross-sectional observational study conducted in the Department of Periodontology at The Oxford Dental College and Hospital, Bangalore, India, from November 2018 to July 2019, where participants were recruited from the outpatient department. The study complied with the ethical guidelines outlined in the Declaration of Helsinki, as modified in 2024, and all participants received a comprehensive explanation of the study objectives and procedures before providing written informed consent for enrolment. The Institutional Committee and Review Board of The Oxford Dental College, Bangalore, granted ethical approval (Ref No: 258/2017-2018).

2.2. Study population and sampling

A total of 45 healthy adults were enrolled in the study and were distributed equally among three groups of 15 each: Group 1: Current smokers with generalized chronic periodontitis; Group 2: Non-smokers with generalized chronic periodontitis; and Group 3 (control): Non-smokers with healthy periodontium. Patients attending the outpatient department during the study period were consecutively screened, and those who met the pre-defined eligibility criteria were recruited. Participants were randomly selected from the pool of eligible individuals to avoid selection bias.

2.3. Eligibility criteria

2.3.1. Inclusion criteria

Participants aged 18–60 years, of either sex, with a minimum of 20 permanent teeth and no history of systemic diseases

or conditions known to influence periodontal status or inflammatory markers, were included in the study. For Group 3 (control), additional requirements included the absence of sites with probing pocket depth (PPD) greater than 3 mm, no CAL, and no radiographic evidence of alveolar bone loss. For Groups 1 and 2, generalized chronic periodontitis, as defined according to the American Academy of Periodontology classification (1999)[11], should have PPD of 5–8 mm at multiple sites, CAL of 3–4 mm, and radiographic evidence of alveolar bone loss affecting more than 30% of assessed sites, as determined using orthopantomogram and intraoral periapical radiographs when required (Figure 1).

2.3.2. Exclusion criteria

Participants who had undergone any form of periodontal therapy (non-surgical or surgical) within the last six months, reported current or previous use of smokeless tobacco or alcohol abuse, or had taken systemic antibiotics, non-steroidal anti-inflammatory drugs, corticosteroids, or other immunosuppressive medications within the last three months, were excluded from the study. Individuals with known systemic diseases or conditions, such as diabetes mellitus, cardiovascular disease, autoimmune disorders, or hematologic disorders, as well as pregnant or lactating women, were also excluded. In addition, any history of smoking was not permitted in Groups 2 and 3.

2.4. Assessment of smoking status

Smoking status was determined using a structured interview, in which participants in Group 1 were classified as current smokers if they reported consuming at least one cigarette or beedi per day for a minimum duration of one year. For each smoker, the number of cigarettes or beedis smoked per day, the frequency of smoking episodes per day, and the total duration of the smoking habit in years were documented. Non-smokers in Groups 2 and 3 were defined as individuals with no lifetime history of using cigarettes, beedis, or any other tobacco products in any form.

2.5. Clinical examination and periodontal measurements

All participants underwent a detailed medical and dental history assessment, followed by a full-mouth periodontal examination performed by an expert under adequate artificial lighting, using a mouth mirror and a William's periodontal probe (Hu-Friedy, United States). Periodontal indices were recorded at six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, distolingual) for all teeth except third molars, and included the plaque index (PI)[12], gingival index (GI)[13], gingival bleeding index (BI)[14], PPD, and CAL. Periodontal status was further evaluated by measuring PPD from the gingival margin to the base of the pocket with a William's periodontal probe (Figure 2), and CAL from the cementoenamel junction to the base of the pocket, along with assessment of tooth mobility using a standard mobility index and furcation involvement using a Nabers probe, classified according to conventional furcation grading systems. To minimize inter-examiner variability, all measurements were obtained by a single trained examiner. In contrast, a second examiner re-evaluated a randomly selected subset of participants to confirm reliability, ensuring that inter-examiner differences remained negligible.

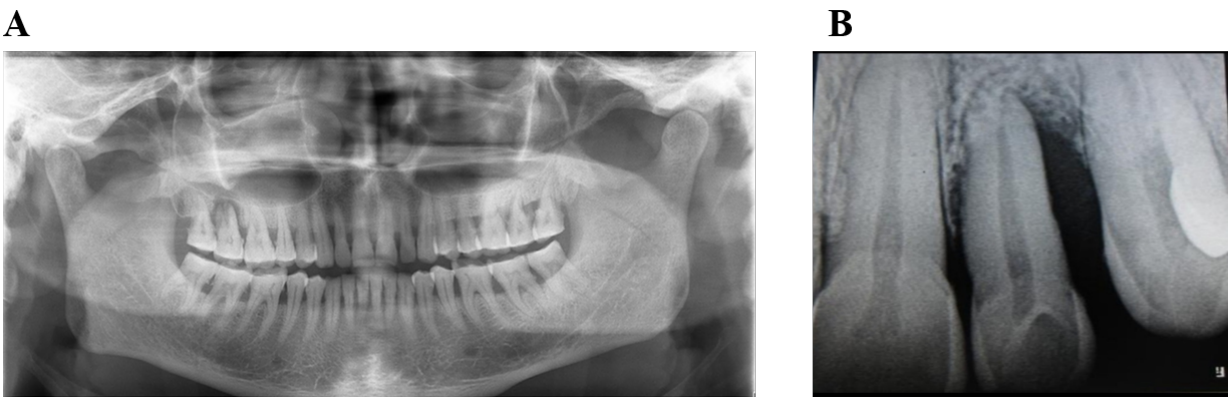

Figure 1. Radiograph showing alveolar bone loss in patient with chronic periodontitis. (A) Orthopantomogram and (B) intraoral periapical radiographs showing alveolar bone loss in a patient with chronic periodontitis.

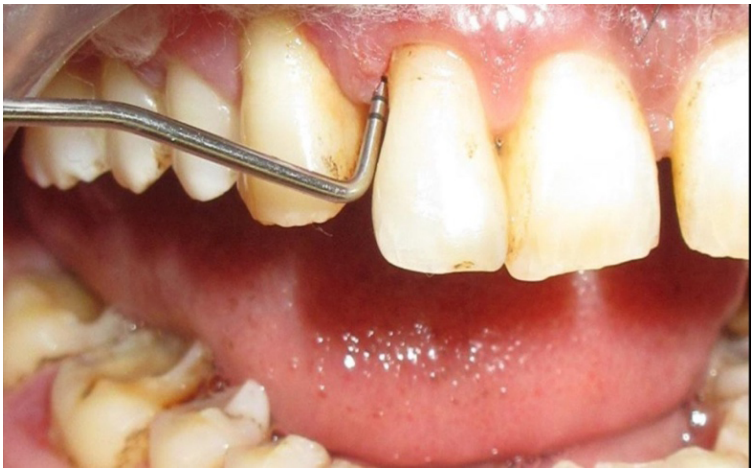

Figure 2. Probing pocket depth measurement using William's periodontal probe

2.5.1. Anthropometric indices

Physiological parameters were assessed for all participants by a trained examiner. Body weight (kg) was measured using a calibrated digital weighing scale with participants wearing light clothing and no footwear, and height (m) was recorded with a stadiometer while participants stood upright without shoes. BMI was then calculated as weight in kilograms divided by height in meters squared (kg/m^2), and WC (cm) was measured using a non-stretchable measuring tape placed at the midpoint between the lower margin of the last palpable rib and the upper border of the iliac crest at the end of a normal expiration.

2.5.2. Hematological inflammatory indices

A trained phlebotomist drew approximately 3–5 mL of venous blood from each participant under aseptic conditions, preferably in the morning after an overnight fast to minimize diurnal variation. The hematological inflammatory indices included ESR (mm/hr) measured by the Westergren method, and Hb (g/dL) and total leukocyte count (TLC) (cells/mm^3) were determined using an automated analyzer according to the manufacturer's guidelines. All laboratory investigations were carried out in the institutional clinical laboratory in accordance with established standard operating procedures and quality control protocols (Figure 3).

2.5.3. Outcome measures

The primary outcome measures were BMI (kg/m^2), ESR (mm/hr), and key periodontal indices, such as PPD (mm) and CAL (mm). Secondary outcomes included WC (cm), Hb (g/dL), TLC (cells/mm^3), and other periodontal indices such as PI, GI, BI, calculus index scores, along with smoking exposure in Group 1, quantified as the number of cigarettes or beedis smoked per day and the duration of smoking in years.

2.6. Statistical analysis

Data were entered into Microsoft Excel and analyzed using the IBM Statistical Package for the Social Sciences (version 22.0). Continuous variables were summarized as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Data normality was evaluated using the Shapiro–Wilk test, and intergroup comparisons of continuous variables (e.g., BMI, ESR, Hb, PPD, CAL) were conducted with one-way analysis of variance; where applicable, independent samples *t*-tests were employed for comparisons between two groups (such as PPD and CAL between Groups 1 and 2). Comparison of mean hematological inflammatory indices (ESR, Hb, and TLC) among the three study groups was performed using the Kruskal–Wallis test, followed by the Mann–Whitney *U* test for post hoc pairwise analysis. Correlations between clinical periodontal indices, smoking exposure, and hematological inflammatory indices were assessed using Spearman's rank correlation coefficient (Rho) due to the abnormal distribution of some variables, and the strength and direction of associations among ESR, Hb, PPD, CAL, and the number of cigarettes smoked per day were examined separately within each group. A two-tailed p-value < 0.05 was considered indicative of statistical significance.

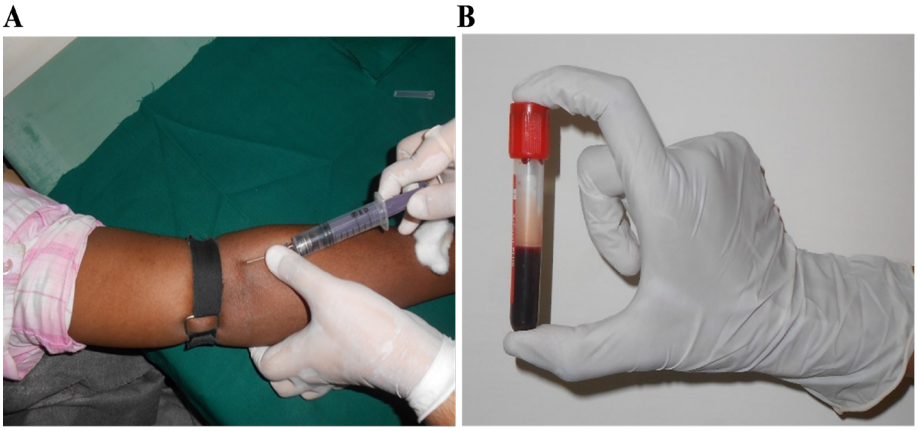

Figure 3. Blood collection procedure. (A) Venous blood collection from the antecubital fossa, and (B) venous blood sample collected in an ethylenediaminetetraacetic acid vacuum tube, showing separation of plasma and cellular components, held upright by a gloved hand under standard laboratory biosafety conditions.

3. Results

3.1. Demographic and anthropometric indices

The mean age ranged from 40.93 ± 7.30 years in Group 1 to 32.93 ± 9.58 years in Group 3, with no statistically significant difference in age across groups (p = 0.06). In contrast, gender distribution differed significantly among groups (p = 0.01), with Group 1 showing a markedly higher proportion of males (100%) than Group 2 (53.3%) and Group 3 (66.7%) (Table 1).

Marked differences were observed in the anthropometric indices across the three groups. Group 1 demonstrated the highest mean BMI (29.47 ± 5.32 kg/m^2) and WC (88.42 ± 7.30 cm), followed by Group 2 (25.35 ± 2.68 kg/m^2; 86.69 ± 8.46 cm) and Group 3 (19.25 ± 2.66 kg/m^2; 79.23 ± 6.70 cm), and intergroup differences in BMI and WC were statistically significant ($p < 0.001$). The analysis indicated that not all pairwise comparisons reached significance, as the difference between Groups 1 and 2 was not statistically significant ($p = 0.80$) (Table 2).

3.2. Hematological inflammatory indices

Comparison of mean hematological inflammatory indices (ESR, Hb, and TLC) among the three study groups was performed using the Kruskal–Wallis test, followed by the Mann–Whitney *U* test for post hoc pairwise analysis. Group 1 exhibited the highest ESR values (20.00 ± 9.66 mm/hr) compared with Group 2 (14.33 ± 3.48 mm/hr) and Group 3 (9.33 ± 3.70 mm/hr), reflecting a highly significant difference among the groups ($p < 0.001$). The test confirmed significant differences between Group 2 and Group 3 ($p = 0.001$) and between Group 1 and Group 3 ($p = 0.004$). Hb levels also differed significantly across groups ($p = 0.02$), with Group 1 showing the lowest mean value (13.89 ± 1.43 g/dL) and Group 3 the highest (15.23 ± 0.65 g/dL). In contrast, TLC did not vary significantly between the groups ($p = 0.79$). A statistically significant difference was observed for ESR and Hb ($p < 0.05$), whereas no significant difference was noted for TLC (Table 3).

3.3. Periodontal indices

Comparison of mean periodontal indices (PI, GI, PPD, and CAL) among the three study groups was performed using the one-way analysis of variance. PI scores were markedly higher in Group 1 (1.85 ± 0.45) and Group 2 (1.99 ± 0.52) compared with Group 3 (0.39 ± 0.23). Group 2 showed the highest GI values (2.02 ± 0.54), followed by Group 1 (1.01 ± 0.32) and Group 3 (0.42 ± 0.17). Statistically significant differences in PI and GI were observed among the three groups ($p < 0.001$). The analysis demonstrated that Group 3 had significantly lower PI and GI scores than Groups 1 and 2 ($p < 0.001$), while no significant difference in PI was found between Groups 1 and 2 ($p = 0.61$). These results indicate substantially better plaque control and reduced gingival inflammation in Group 3 compared with the other groups (Table 4).

Group 1 (2.70 ± 0.74 mm) demonstrated a significantly greater mean PPD than Group 2 (1.94 ± 0.38 mm; $p = 0.001$), as determined by an independent *t*-test. Likewise, CAL was markedly higher in Group 1 (0.83 ± 0.33 mm) compared with Group 2 (0.59 ± 0.17 mm; $p = 0.02$) (Table 5).

Table 1. Comparative distribution of age and gender across the three study groups

Variable	Group 1		Group 2		Group 3		*p*-value
Age (years), mean (SD)	40.93 (7.30)		38.00 (8.97)		32.93 (9.58)		0.06[a]
Range	28–55		28–58		25–54		
Gender, *n* (%)							
Males	15	100%	8	53.3%	10	66.7%	0.01*[b]
Females	0	0%	7	46.7%	5	33.3%	

Notes: *Statistically significant; [a]One way analysis of variance, [b]Chi-square test of independence.
Abbreviation: SD: Standard deviation.

Table 2. Comparison of mean body mass index and waist circumference across three groups

Parameters	Groups	*N*	Mean	Standard deviation	*p*-value[a]	Significant difference	*p*-value
Body mass index (kg/m^2)	Group 1	15	29.47	5.32	<0.001*	G1 vs. G2	0.01*
	Group 2	15	25.35	2.68		G1 vs. G3	<0.001*
	Group 3	15	19.25	2.66		G2 vs. G3	<0.001*
Waist circumference (cm)	Group 1	15	88.42	7.30	<0.004*	G1 vs. G2	0.80
	Group 2	15	86.69	8.46		G1 vs. G3	<0.005*
	Group 3	15	79.23	6.70		G2 vs. G3	<0.03*

Notes: * Statistically significant. [a]*p*-value derived by analysis of variance test.

Table 3. Comparison of mean hematological inflammatory indices among the three study groups

Parameters	Groups	*N*	Mean	Standard deviation	*p*-value[a]	Significant difference	*p*-value[b]
Erythrocyte sedimentation rate (mm/hr)	Group 1	15	20.00	9.66	<0.001*	G1 vs. G2	0.11
	Group 2	15	14.33	3.48		G1 vs. G3	0.004*
	Group 3	15	9.33	3.70		G2 vs. G3	0.001*
Hemoglobin (g/dL)	Group 1	15	13.89	1.43	0.02*	G1 vs. G2	0.95
	Group 2	15	14.03	1.35		G1 vs. G3	0.02*
	Group 3	15	15.23	0.65		G2 vs. G3	0.009*
Total leukocyte count ($cells/m^3$)	Group 1	15	7,706.67	1,081.97	0.79	G1 vs. G2	-
	Group 2	15	7,703.33	1,077.44		G1 vs. G3	-
	Group 3	15	7,926.67	901.16		G2 vs. G3	-

Notes: * Statistically significant. [a]*p*-value derived by Kruskal–Wallis test; [b]*p*-value derived by Mann–Whitney post hoc test.

Table 4. Comparison of mean plaque index and gingival index values among the three study groups

Parameters	Groups	*N*	Mean	Standard deviation	*p*-value[a]	Significant difference	*p*-value[b]
Plaque index	Group 1	15	1.85	0.45	<0.001*	G1 vs. G2	0.61
	Group 2	15	1.99	0.52		G1 vs. G3	<0.001*
	Group 3	15	0.39	0.23		G2 vs. G3	<0.001*
Gingival index	Group 1	15	1.01	0.32	<0.001*	G1 vs. G2	<0.001*
	Group 2	15	2.02	0.54		G1 vs. G3	<0.001*
	Group 3	15	0.42	0.17		G2 vs. G3	<0.001*

Notes: * Statistically significant. [a]*p*-value derived by Kruskal–Wallis test; [b]*p*-value derived by Mann–Whitney post hoc test.

Table 5. Comparison of probing pocket depth and clinical attachment loss scores between Group 1 and Group 2

Parameters	Groups	*N*	Mean	Standard deviation	Mean difference	*p*-value[a]
Probing pocket depth	Group 1	15	2.70	0.74	0.76	0.001*
	Group 2	15	1.94	0.38		
Clinical attachment loss	Group 1	15	0.83	0.33	0.24	0.02*
	Group 2	15	0.59	0.17		

Notes: *Statistically significant; [a]Independent sample *t*-test.

3.4. Correlation analysis

Probing pocket depth showed a substantial connection with daily cigarette consumption (Rho = 0.64; $p = 0.01$) and a somewhat favorable link with ESR (Rho = 0.53; $p = 0.04$) in Group 1. CAL showed a marked negative correlation with Hb (Rho = −0.74; $p = 0.002$) and a very strong positive correlation with ESR (Rho = 0.84; $p < 0.001$). Similar trends were observed in Group 2, although the correlation coefficients were smaller and less robust statistically. No significant correlations were observed between periodontal indices and hematological inflammatory indices in Group 3 (Table 6). In addition, the mean number of cigarettes smoked per day in Group 1 was 13.3 ± 3.6, with individual consumption ranging from 8 to 24 cigarettes.

4. Discussion

This cross-sectional observational study's main goal was to evaluate the relationships between four important variables across three predetermined risk groups: smoking, anthropometric indices (measured by BMI and WC), hematological inflammatory indices (ESR, Hb, and TLC), and periodontal indices (PI, GI, CAL, and PPD). It showed that smoking and obesity jointly enhance the periodontal and systemic inflammatory burden in chronic periodontitis. This is reflected as higher ESR, lower Hb levels, and more severe attachment loss in smokers with periodontitis compared with non-smokers with periodontitis and periodontally healthy controls. These findings support the concept that simple anthropometric and hematological indices can enhance periodontal risk stratification when interpreted alongside conventional clinical parameters.

The smoker–periodontitis group (Group 1) in this study exhibited the highest ESR (20.00 ± 9.66 mm/hr) and BMI (29.47 ± 5.32 kg/m^2), together with significantly greater PPD and CAL than non-smoking periodontitis subjects, indicating a synergistic inflammatory milieu driven by tobacco exposure, excess adiposity, and chronic periodontal infection. Elevated ESR in smokers with periodontitis, combined with the clear dose–response between cigarette consumption and ESR, is consistent

Table 6. Spearman's rank correlation analysis of periodontal indices with anthropometric indices, hematological indices, and smoking exposure in Groups 1 and 2

Groups	Variables	Value	PPD	CAL	PI	GI	BMI	WC	ESR	Hb	TLC	Cig/day
Group 1	PPD	Rho	1	0.64	0.02	0.18	0.05	0.08	0.53	−0.46	−0.19	0.64
		p	-	0.01*	0.93	0.52	0.87	0.77	0.04*	0.08	0.51	0.01*
	CAL	Rho	0.64	1	0.15	0.16	0.02	0.13	−0.74	−0.74	−0.25	0.65
		p	0.01*	-	0.61	0.56	0.93	0.66	<0.001*	0.002*	0.36	0.008*
Group 2	PPD	Rho	1	0.23	0.07	0.10	0.19	0.15	0.17	−0.46	0.35	-
		p	-	0.40	0.81	0.72	0.51	0.59	0.55	0.04*	0.21	-
	CAL	Rho	0.233	1	0.20	0.05	0.25	0.06	0.48	−0.31	−0.24	-
		p	0.404	-	0.49	0.85	0.37	0.83	0.07	0.26	0.39	-

Note: *Statistically significant.

Abbreviations: BMI: Body mass index; CAL: Clinical attachment loss; Cig/day: Cigarettes smoked per day; ESR: Erythrocyte sedimentation rate; GI: Gingival index; Hb: Hemoglobin; PI: Plaque index; PPD: Probing pocket depth; TLC: Total leukocyte count; WC: Waist circumference.

with previous reports that periodontitis and tobacco use upregulate acutephase reactants and amplify systemic inflammation.[15] The concomitant reduction in Hb levels in this group, in the absence of changes in TLC, suggests a pattern more consistent with anemia of chronic disease than with overt leukocytosis, in line with earlier work linking chronic periodontal inflammation to altered erythrocyte indices and reversible anemia following periodontal therapy.[16]

Additionally, compared to both vape users and non-smokers, Aldalaeen *et al.*[17] found that cigarette smokers had more severe periodontal degeneration, as seen by higher GI scores, lower PI scores, and larger CAL. The results of the study showed that tobacco use typically exacerbates periodontitis, as evidenced by elevated ESR values and increased acute-phase reactants such as fibrinogen, which is in accordance with the present study.

Yadav *et al.*[18] conducted a study and found that ESR was higher in periodontitis patients, which significantly reduced one month post scaling and root planing. They also measured PPD, PI, and GI before and 1 month after scaling and root planing to assess the association between periodontitis and ESR. Following scaling and root planing, all clinical indicators significantly improved, and the ESR decreased significantly (3.27 ± 1.24 mm/hr), highlighting the importance of periodontal health maintenance in reducing the systemic inflammatory burden. The results of our study are consistent with these studies.

Gitte *et al.*[19] conducted a study to compare hematological parameters, including ESR and TLC, between smokers and non-smokers in the Indian population. The mean ESR was higher in smokers (11.74 mm/hr) than in non-smokers (7.38 mm/hr), consistent with the present study. The TLC values were higher in smokers (8,050/mm^3) than in non-smokers (6,858/mm^3), whereas in our study, there was no statistically significant difference among the three groups.

Smoking exerts a "masking effect" on GI scores by attenuating overt clinical signs of inflammation—such as bleeding on probing and redness—despite ongoing periodontal tissue destruction. This effect is mainly attributed to nicotine-induced vasoconstriction, which reduces gingival blood flow and capillary density, as well as to impaired angiogenesis and decreased production of pro-inflammatory mediators.[20-22]

Nicotine stimulates the release of adrenaline and noradrenaline, which act on α1-adrenergic receptors to induce transient gingival vasoconstriction, while sustained exposure leads to reduced vascular density and diminished inflammatory cell infiltration. Heavy smoking amplifies these effects, producing dose-dependent reductions in GI and bleeding on probing compared with light smoking and non-smokers, which can complicate the accurate clinical assessment of gingival health. Smoking cessation reverses this phenomenon, with bleeding on probing increasing within weeks as vascular function normalizes.[23,24] This masking effect delays early identification of periodontitis in smokers, who tend to present with higher plaque indices and greater attachment loss despite deceptively low GI scores.[23,24]

Susanto *et al.*[25] investigated the connection between blood cell counts, systemic inflammatory markers, and periodontal inflamed surface area using correlation and multiple linear regression analyses. According to their findings, periodontal inflamed surface area and plaque

scores predicted platelet count ($p < 0.05$), and there were strong associations between periodontal inflamed surface area and ESR ($r = 0.32$; $p < 0.05$) and platelet count ($r = 0.527$; $p < 0.05$). These results suggest that periodontitis may be a risk factor for atherosclerosis in Indonesian individuals with type 2 diabetes mellitus and is linked to increased systemic inflammatory markers.

A clear dose-dependent association between cigarette consumption and ESR further supports the concept that smoking contributes to heightened systemic inflammation. Elevated BMI among smokers suggests a "triple burden" of inflammation arising from periodontal disease, tobacco exposure, and obesity-related chronic inflammation. Consequently, these findings underscore the necessity of comprehensive management strategies for periodontal patients who both smoke and are overweight. Obesity aggravates periodontal inflammation and worsens treatment outcomes through its chronic inflammatory state, disruption of immune regulation, and promotion of tissue breakdown. Addressing obesity can therefore improve the effectiveness of periodontal therapy. Healthcare professionals, including bariatric surgeons and dietitians, should counsel patients on the oral health risks associated with obesity, while non-dental clinicians should be able to recognize signs of periodontal disease to facilitate timely dental referrals.[26]

Patients undergoing bariatric surgery exhibit marked improvements in periodontal status compared with those who do not undergo such interventions, underscoring the value of individualized nutritional counseling, physical activity, and periodontal therapy in improving both oral and systemic health.[27,28] Nonetheless, evidence on surgical periodontal treatment outcomes in obese patients remains limited, and the risk of an exaggerated inflammatory response in this population may favor non-surgical approaches over surgical procedures to optimize healing.[28,29] In a cross-sectional study[30] of 718 schoolchildren aged 5–15 years, 15-year-olds showed a caries prevalence of 45.49%, and 52.8% had dental calculus, both higher than in younger children. Moreover, lower weight percentiles in 6- and 12-year-old students were associated with fewer healthy teeth. These observations point to a decline in caries but an increase in periodontitis, suggesting that body weight can influence oral health outcomes.

The present study also identified the lowest Hb levels in the smoker–periodontitis group, supporting the presence of chronic inflammation and a possible predisposition to anemia. Panneerselvam *et al.*[31] assessed systemic markers of anemia in patients with generalized aggressive periodontitis before and after phase I periodontal therapy. Fifteen patients were divided into Group A (pre-treatment) and Group B (post-treatment), and after three months, Group B showed significant improvements in hematological parameters, including higher Hb and red blood cell counts. These findings support an association between generalized aggressive periodontitis and reduced red blood cell indices, consistent with anemia of chronic disease, and indicate that non-surgical periodontal therapy can improve both periodontal status and anemia, highlighting the importance of managing chronic oral infections to alleviate anemic conditions.

By examining hematological parameters in 150 participants—50 healthy controls, 50 with chronic generalized gingivitis, and 50 with chronic generalized periodontitis—Anumolu *et al.*[16] further investigated the association between anemia and periodontitis. According to the study, patients with periodontitis had higher white blood cell counts and lower Hb and erythrocyte counts than healthy controls, but no discernible differences in other hematological indices.

While TLC values were comparable between groups, the lower Hb levels observed in smokers with periodontitis point to subtle hematological changes consistent with chronic inflammatory processes.

Jia *et al.*[32] examined the association between the Life's Crucial 9 lifestyle score and periodontitis severity among United States adults using data from 7,124 participants in the National Health and Nutrition Examination Survey 2009–2014. A 10-point increase in Life's Crucial 9 was linked to a lower risk of periodontitis (odds ratio = 0.858), reduced disease severity, and decreased CAL and PD. Tobacco exposure and glycemic control emerged as key determinants, with inflammatory markers accounting for approximately 37.7% of the relationship between Life's Crucial 9 and periodontitis, whereas oxidative stress showed no significant mediating effect. Life's Crucial 9 and Life's Essential 8 demonstrated similar predictive performance (area under the curve: 0.758 vs. 0.759). Overall, higher Life's Crucial 9 scores were associated with better periodontal status, suggesting that lifestyle modifications—particularly tobacco avoidance and improved glycemic control—may reduce periodontitis risk.

These findings support incorporating lifestyle evaluation and simple systemic markers, such as ESR, into routine periodontal assessment to enhance patient risk stratification. A clear dose–response pattern was also observed, with higher ESR values corresponding to greater daily cigarette consumption in this study. The smoker–periodontitis group also exhibited an elevated mean BMI (29.47 kg/m^2), indicating a triad of inflammatory burden from periodontal infection, smoking, and obesity. Interestingly, TLC remained unchanged, suggesting that

the systemic response preferentially involves acute-phase reactants such as fibrinogen rather than leukocytosis, thereby positioning ESR as a particularly sensitive inflammatory indicator in this context.

The principal limitation of this study is its cross-sectional design and a small sample of 45 adults (15 per group), which constrain statistical power and external validity. In the present study, Group 1 comprises all males, whereas Groups 2 and 3 comprise mixed genders. This gender confounding can independently influence exposure status and outcomes, including anthropometric indices, inflammatory hematological indices, and periodontal indices. The highest BMI (29.47 kg/m^2) found in the smoker–periodontitis group acts as a confounding factor, implying obesity as an inherently pro-inflammatory state which elevates the ESR and obscures the independent effects of smoking and periodontitis. This study also included unbalanced groups due to recruitment constraints, absence of a proper sample size calculation, and potential residual confounding despite stratification, matching, or multivariable modeling, as it undermined Strengthening the Reporting of Observational Studies in Epidemiology comparability assumptions, risk-biased effect estimates with increased type II error, and ultimately limited generalizability. Another limitation of this study is that participants were classified using the 1999 American Academy of Periodontology system rather than the current 2017 American Academy of Periodontology/ European Federation of Periodontology classification. Accordingly, direct translation of cases into contemporary stage and grade categories is approximate and may reduce comparability with newer studies. If re-screened under current guidelines, inclusion would be defined by periodontitis case definitions that use stage, extent, and grade, while exclusions would remain unchanged unless a study-specific criterion required reclassification of systemic disease-associated periodontitis or necrotizing disease.

Longitudinal cohort designs should be prioritized in the future to monitor ESR trajectories in at-risk populations and determine whether periodontitis and smoking can initiate or aggravate systemic complications. Additionally, reliance on ESR alone as a non-specific inflammatory marker further limits analytical precision. Hence, future research should incorporate mechanistic investigations using additional biomarkers, such as C-reactive protein, interleukin-6, and oxidative stress indicators, to elucidate the molecular pathways through which smoking and periodontal pathogens contribute to systemic inflammation.

5. Conclusion

Considering these limitations and future prospects, this study indicates that in chronic inflammatory conditions like periodontitis, smoking and obesity synergistically exacerbate periodontal breakdown and systemic inflammation, as evidenced by elevated ESR and BMI, underscoring the importance of incorporating lifestyle habits and basic inflammatory markers into periodontal evaluation. These observations from our study support the notion that these risk factors interact rather than act independently, as further corroborated by the dose-dependent increase in ESR with higher cigarette consumption. The findings demonstrate an association rather than a causal relationship between smoking, obesity, periodontal health, and systemic inflammation.

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Conflict of interest

The authors declare they have no competing interests.

Author contributions

Conceptualization: Rujuta Patil
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Ethics approval and consent to participate

This study was approved by the Ethics Committee of The Oxford Dental College and Hospital, Bangalore, India (Ref No: 258/2017-2018) and is in accordance with the Declaration of Helsinki. All participants provided written informed consent to participate in this study.

Consent for publication

Written consent was obtained from each of the subjects to publish their data and/or images.

Availability of data

All data analyzed have been presented in the paper.

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