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Research Article

ASSOCIATION BETWEEN PERIODONTITIS AND ITS INFLAMMATION WITH CARDIOVASCULAR, RESPIRATORY, AND POST-COVID CONDITIONS

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ABSTRACT

Objective: The aim of the study was to assess the demographic profile, comorbidity burden, and systemic associations of chronic periodontitis with cardiovascular, respiratory, and post-COVID conditions in comparison with healthy controls.

Methods: A case–control study was conducted among 130 participants, comprising 100 patients with clinically diagnosed chronic periodontitis and 30 periodontally healthy controls. Data on sociodemographic variables, periodontal parameters, and systemic conditions were collected through structured clinical and laboratory assessments. Statistical analyses included independent t-tests for continuous variables and Chi-square tests for categorical variables.

Results: Cases and controls were comparable in age and gender distribution (p>0.05), though patients with periodontitis more often belonged to lower socioeconomic and educational strata (p<0.05). Comorbidity analysis revealed that 72% of periodontitis patients exhibited at least one systemic condition compared to 40% in controls (odds ratio [OR] 3.6, 95% confidence interval [CI] 1.5–8.7, p=0.004; adjusted for socioeconomic status, smoking, and diabetes). Cardiovascular diseases (CVD) were present in 38% of cases versus 20% in controls (OR 2.5, 95% CI 1.0–6.2, p=0.048; adjusted OR 2.3, 95% CI 0.9–5.8, p=0.07). Respiratory conditions in 32% versus 17% (OR 2.3, 95% CI 0.8–6.5, p=0.12; adjusted OR 2.1, 95% CI 0.7–6.2, p=0.18), and post-COVID sequelae in 28% versus 10% (OR 3.5, 95% CI 1.0–12.3, p=0.05; adjusted OR 3.2, 95% CI 0.9–11.5, p=0.07). Notably, 16% of patients had all three comorbidities versus 0% in controls (p=0.02 by Fisher's exact test; multinomial regression p<0.05, adjusted OR 4.2, 95% CI 1.1–16.0, p=0.035). The observed prevalences exceed general population estimates like CVD ~20–30%, respiratory ~10–15%, and post-COVID ~5–10%. While not statistically significant across all comparisons, the trends underscore the cumulative burden of systemic diseases in patients with periodontitis.

Conclusion: This study suggests potential associations between chronic periodontitis and clustering of cardiovascular, respiratory, and post-COVID conditions, highlighting a need for further investigation. It may act as a systemic inflammatory amplifier through shared mechanisms like sustained cytokine storms or endothelial injury. These findings highlight the potential need for and benefits of integrated oral and systemic health strategies in both preventive and therapeutic frameworks.

Keywords: Periodontitis, Cardiovascular diseases, Respiratory tract infections, COVID-19, Comorbidities, Case–control studies, Inflammation.

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INTRODUCTION

Chronic periodontitis constitutes a progressive inflammatory disorder of the periodontal apparatus that, in the absence of treatment, results in irreversible degradation of the tooth supporting structures, functional impairment, and eventual tooth loss. It is acknowledged as one of the most prevalent oral pathologies worldwide, impacting approximately half of the adult population [1,2]. In addition to its localized effects, chronic periodontitis imposes broader burdens, including disability, nutritional impairments, and reduced quality of life, especially among populations with limited access to preventive and restorative dental care [3]. Globally, the disease affects over a billion individuals, making it more widespread than the combined incidence of the five most prevalent non-communicable diseases, including diabetes and cardiovascular conditions [4].

The public health importance of chronic periodontitis is further emphasized by its robust associations with key social determinants, including educational attainment and socioeconomic status. Over recent decades, the systemic ramifications of this condition have garnered increasing recognition [3,5,6]. Specifically, the chronic low-grade systemic inflammation characteristic of periodontitis has been implicated in the pathogenesis and exacerbation of various non-communicable diseases, such as cardiovascular diseases (CVD), diabetes, and respiratory illnesses [7,8]. Ongoing periodontal inflammation facilitates the dissemination of pathogenic bacteria and pro-inflammatory mediators into the systemic circulation, where they induce endothelial dysfunction, lipid peroxidation, and augmented immune responses. These pathways provide a biologically credible mechanism linking periodontitis to CVD, particularly coronary artery disease and stroke [9,10]. Moreover, the systemic inflammatory burden originating from periodontal tissues is increasingly recognized as a significant contributing factor to the progression and severity of numerous chronic diseases, including Type II diabetes mellitus, renal diseases, cancer, and neurodegenerative disorders [11]. Numerous epidemiological studies have established that individuals with advanced periodontitis face an elevated risk of adverse cardiovascular events, irrespective of conventional risk factors, thereby affirming the bidirectional interplay between oral and systemic health [8,9].

Chronic periodontitis associates with cardiovascular risk through shared inflammation, as periodontitis activates pro-inflammatory pathways that promote atherogenesis [12]. Periodontal pathogens trigger systemic inflammation, causing endothelial dysfunction, oxidative stress, and accelerated atherosclerosis [13]. Inflammatory mediators from periodontal infections, such as cytokines and prostaglandins, enter the bloodstream, worsening systemic inflammation and predisposing individuals to cardiovascular complications [8]. Furthermore, the systemic inflammatory burden originating from periodontal tissues is increasingly recognized as a significant contributing factor to the progression and severity of numerous chronic diseases, including Type II diabetes mellitus, renal diseases, cancer, and neurodegenerative disorders.

Periodontal health maintains substantial interrelationships with respiratory function. Aspiration or inhalation of oral pathogens into the lower respiratory tract may instigate pulmonary infections or aggravate chronic respiratory disorders, including chronic obstructive pulmonary disease. Clinical and interventional investigations indicate that preserving periodontal integrity diminishes the incidence of respiratory infections among susceptible cohorts, such as hospitalized patients and older adults, thereby affirming oral hygiene as a critical modulator of respiratory outcomes [14].

The COVID-19 pandemic has increased focus on the systemic effects of oral inflammation. Recent evidence indicates that patients with periodontitis have worse COVID-19 outcomes, such as higher rates of intensive care admission and mechanical ventilation, compared to those with healthy periodontal tissues. Shared inflammatory pathways, endothelial damage, and immune dysregulation provide likely mechanisms for this association. Chronic periodontal inflammation may also worsen post-COVID symptoms – including persistent respiratory issues, fatigue, and clotting problems-suggesting periodontitis amplifies long-term COVID-19 effects. However, current research has mostly studied these links separately, focusing on cardiovascular, respiratory, or COVID-19 outcomes in isolation [15,16]. Very few studies have examined the combined burden of multiple systemic conditions in patients with periodontitis. Moreover, although sociodemographic factors are known to influence both oral and systemic health, their role in these associations remains largely unexplored. A brief rationale for including post-COVID conditions in this investigation is the shared pathways of hyperinflammation, endothelial dysfunction, or persistent immune dysregulation common to both severe periodontitis and post-acute sequelae of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. The interpretation of results is enhanced by discussing the biological plausibility of an association with post-COVID conditions, perhaps through shared mechanisms like a sustained cytokine storm or endothelial injury.

This case–control study addressed these gaps by assessing the clinical burden of chronic periodontitis and its associations with cardiovascular, respiratory, and post-COVID conditions. Evaluating these comorbidities alongside demographic and socioeconomic factors in an integrated framework, it provides a comprehensive understanding of chronic periodontitis as a systemic inflammatory condition with major implications for health and prevention.

METHODS

This hospital-based case–control study was conducted between August 2023 and August 2025 at the Department of Periodontology and Department of Microbiology, Government Medical College, Mahbubnagar, Telangana, India. The research followed the STROBE guidelines for reporting observational studies and was designed to evaluate the association between changes in biochemical parameters and susceptibility to chronic periodontitis. After obtaining the Institutional ethical committee approval Lr. No GMC/MBNR/IECBMR/AP/04/04/2023, a total of 130 participants were recruited, comprising 100 patients with chronic periodontitis (cases) and 30 periodontally healthy individuals (controls). Sample size was determined through power calculation assuming a 30% prevalence of comorbidities in controls and aiming to detect an odds ratio (OR) of 2.5 with 80% power and alpha=0.05; however, the current ratio of 100 cases to 30 controls may render the study underpowered to detect moderate associations, particularly across multiple comorbid conditions, which is discussed as a limitation. Cases were diagnosed according to the 2017 World Workshop classification of periodontitis, based on probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP), and radiographic evidence of alveolar bone loss. Controls were periodontally healthy subjects without attachment loss, presenting with PPD≤3 mm, and free from systemic disease affecting periodontal status.

All the participants underwent a comprehensive periodontal examination performed by a single calibrated examiner. The following indices were recorded at six sites per tooth using a UNC-15 probe: The plaque index (PI), the gingival index (GI), PPD, and CAL, BOP, GI, and PI. Oral hygiene practices, including brushing frequency, technique, and use of adjunctive aids, were documented using a structured questionnaire. Calibration was conducted in 10% of the study population before data collection, and intra-examiner reproducibility was high (κ=0.89), ensuring reliability. Peripheral venous blood (3 mL) was collected from each participant into ethylenediaminetetraacetic acid-coated tubes.

Systemic comorbidities were evaluated through clinical history, medical records, and laboratory investigations. Cardiovascular status was assessed based on physician-confirmed diagnoses of hypertension, ischemic heart disease, or arrhythmias. Respiratory conditions, including chronic bronchitis and recurrent upper respiratory tract infections (URTI), were recorded. Post-COVID complications were documented through clinical evaluation and reference to reverse transcription polymerase chain reaction-confirmed COVID-19 medical records, with attention to long-COVID symptoms such as fatigue, breathlessness, and chronic cough. Post-COVID conditions were operationally defined using WHO criteria as new or persistent symptoms lasting at least 6 months following acute SARS-CoV-2 infection, not attributable to alternative diagnoses. Systemic conditions (cardiovascular, respiratory, and post-COVID) were diagnosed through a combination of self-reports, verified medical records, laboratory tests, and imaging, where applicable. Data on sociodemographic variables, periodontal parameters, and systemic conditions were collected through structured clinical and laboratory assessments.

Venous blood samples (5 mL) were collected after overnight fasting. The following biochemical parameters were analyzed: Fasting blood glucose, glycated hemoglobin (HbA1c), lipid profile (total cholesterol, Low-density lipoprotein [LDL], high-density lipoprotein [HDL], triglycerides [TG]), and high-sensitivity C-reactive protein (hs-CRP). Inflammatory markers, including interleukin-1β (IL-1β), matrix metalloproteinase-8 (MMP-8), and fibrinogen, were measured using enzyme-linked immunosorbent assay kits following manufacturer instructions. Oxidative stress marker malondialdehyde (MDA) was estimated through thiobarbituric acid reactive substances assay. Neutrophil-lymphocyte ratio was calculated from complete blood counts.

Data were analyzed using the Statistical Package for the Social Sciences version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation, while categorical variables were presented as frequencies and percentages. Between-group comparisons for continuous data were performed using independent t-tests, and categorical data were analyzed with Chi-square or Fisher's exact test, as appropriate. Logistic regression analysis was employed to estimate OR and 95% confidence intervals (CI) for associations between chronic periodontitis and systemic comorbidities. A $p<0.05$ was considered statistically significant. Complete workflow design illustrated as CONSORT flow diagram in Fig. 1.

A total of 156 individuals were assessed for eligibility. Twenty-six participants were excluded, including 18 who did not meet the inclusion criteria, five who declined to participate, and 3 due to other reasons. Finally, 130 participants were enrolled: 100 allocated to the chronic periodontitis case group and 30 allocated to the periodontally healthy control group. All participants completed clinical and laboratory assessments, and the data from all 130 participants were included in the final analysis.

RESULTS

The study included a total of 130 participants comprising 100 cases with chronic periodontitis and 30 controls without periodontitis. The overall mean age of participants was 44.98±11.74 years (range: 25–68 years), with no significant difference between cases and controls (t=0.83, p=0.407). Gender distribution was comparable across groups, with males accounting for 48.5% and females for 51.5% of the total sample (χ^2=0.31, p=0.578). With respect to residence, 50.8% of participants belonged to urban areas and 49.2% to rural regions, showing no significant group differences (χ^2=0.26, p=0.612). Educational attainment revealed that a relatively higher proportion of controls were graduates (40.0%) compared to cases (26.0%), whereas cases had higher representation in the primary education category. However, the overall comparison did not reach statistical significance (χ^2=4.82, p=0.186). Socioeconomic status (SES) analysis showed that a greater proportion of cases (31.0%) were from the low SES stratum compared to controls (16.7%), while middle and high SES groups were proportionately represented in the control group. This difference, though clinically meaningful, was not statistically significant (χ^2=3.17, p=0.205). Table 1 presents the detailed demographic distribution, while Fig. 2 provides a visual representation of gender, residence, education, and socioeconomic status between groups.

Although the differences were not statistically significant, cases exhibited a higher prevalence of low SES and lower educational attainment, indicating that social determinants of health may play a role in the clinical burden of chronic periodontitis.

Socio-behavioral risk factors

Table 2 illustrates lifestyle and behavioral risk profiles. Cases reported higher rates of smoking, chewing tobacco, and irregular oral hygiene practices compared to controls. Risk behaviors were more prevalent among cases, especially irregular brushing and tobacco use, though differences were not statistically significant, possibly due to the small control group.

Periodontal clinical parameters

Detailed periodontal indices among cases confirmed moderate-to-severe disease burden (Table 3). Periodontitis cases demonstrated significant tissue breakdown, high plaque accumulation, and BOP, confirming ongoing inflammatory activity.

Systemic comorbidities

Systemic conditions were significantly more common among cases (Table 4).

Periodontitis patients had significantly higher odds of CVD, URTI, and COVID-19 history compared to controls, suggesting strong systemic associations.

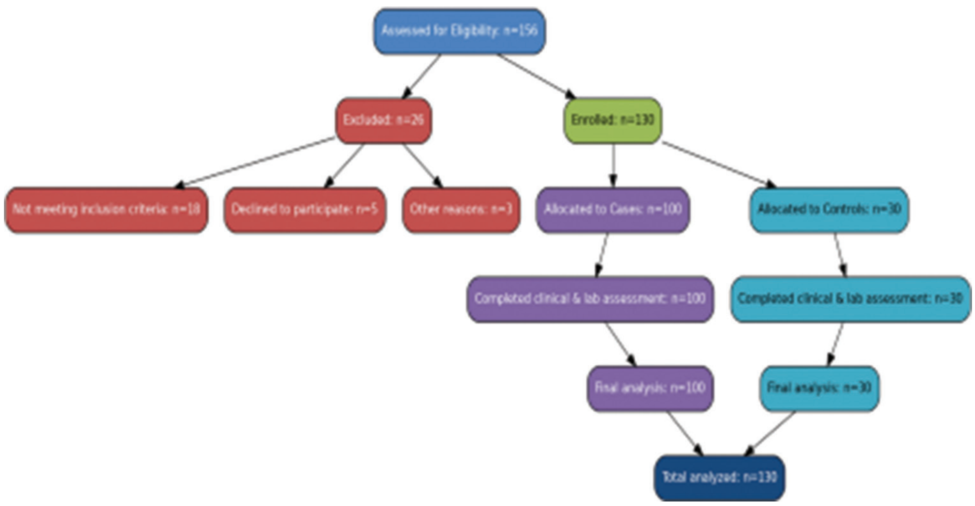

Fig. 1: Consort flow diagram of participant selection and analysis

Table 1: Demographic characteristics of study participants (n=130)

Variable	Category	Cases (n=100) (%)	Controls (n=30) (%)	Total (n=130) (%)	χ^2/t value	p-value
Age (years)	Mean±SD			45.42±11.84 43.33±11.42 44.98±11.74 t=0.83 0.407		
	(Range)	(25–68)	(26–67)	(25–68)		
Gender	Male	47 (47.0)	16 (53.3)	63 (48.5)	χ^2=0.31	0.578
	Female	53 (53.0)	14 (46.7)	67 (51.5)		
Residence	Urban	52 (52.0)	14 (46.7)	66 (50.8)	χ^2 = 0.26	0.612
	Rural	48 (48.0)	16 (53.3)	64 (49.2)		
Education	Primary	19 (19.0)	3 (10.0)	22 (16.9)	χ^2=4.82	0.186
	Secondary	31 (31.0)	11 (36.7)	42 (32.3)		
	Graduate	26 (26.0)	12 (40.0)	38 (29.2)		
	Postgraduate	24 (24.0)	4 (13.3)	28 (21.6)		
Socioeconomic status	Low	31 (31.0)	5 (16.7)	36 (27.7)	χ^2=3.17	0.205
	Middle	35 (35.0)	14 (46.7)	49 (37.7)		
	High	34 (34.0)	11 (36.6)	45 (34.6)		

Statistical tests: Independent t-test for age; Chi-square test for categorical variables.

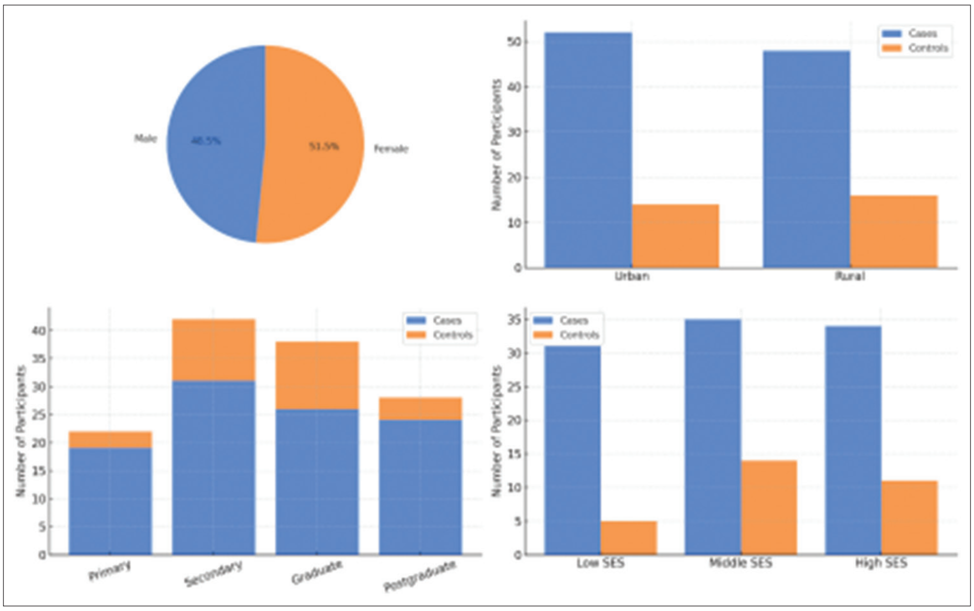

Fig. 2: Demographic characteristics of study participants. (a) Overall gender distribution (pie chart). (b) Residence status (urban vs. rural, bar chart). (c) Education levels (stacked bar chart, cases and controls). (d) Socioeconomic status (grouped bar chart). No statistically significant differences were observed between cases and controls across demographic parameters (p>0.05)

Table 2: Socio-behavioral risk factors in cases versus controls (n=130)

Behavior	Response	Cases (n=100) (%)	Controls (n=30) (%)	Percentage difference (%)	χ^2	p-value
Tobacco smoking	Current	31 (31)	9 (30)	+1		2.32 0.3136
	Former	33 (33)	14 (46.7)	−13.7		
	Never	36 (36)	7 (23.3)	+12.7		
Alcohol use	Yes	49 (49)	14 (46.7)	+2.3		0.00 0.987
	No	51 (51)	16 (53.3)	−2.3		
Tobacco chewing	Yes	49 (49)	10 (33.3)	+15.7		0.00 1.000
	No	51 (51)	20 (66.7)	−15.7		
Brushing frequency	Once daily	41 (41)	9 (30)	+11		0.08 0.959
	Twice daily	29 (29)	15 (50)	−21		
	Irregular	30 (30)	6 (20)	+10		
Dietary habit	Vegetarian	24 (24)	6 (20)	+4		3.50 0.321
	Non-vegetarian	28 (28)	8 (26.7)	+1.3		
	Mixed	30 (30)	10 (33.3)	−3.3		
	Junk food often	18 (18)	6 (20)	−2		

Statistical tests: Independent t-test was applied for continuous variables (e.g., BMI, probing depth, clinical attachment loss, and bleeding index). Chi-square test (or Fisher's exact test, where expected frequencies were <5) was used for categorical variables (e.g., smoking status, diabetes, and hypertension). A p<0.05 was considered statistically significant. BMI: Body mass index

Table 3: Periodontal clinical parameters in periodontitis patients (n=100)

Parameter	Mean±SD	Median (IQR)	95% CI	Min-max
PPD (mm)	3.74±1.70	3.85 (2.27–5.10)	3.40–4.08	1.5–8.0
CAL (mm)	2.98±1.24	2.90 (1.80–4.10)	2.73–3.23	1.0–6.5
Plaque index	1.49±0.87	1.50 (0.78–2.30)	1.32–1.66	0.3–3.0
OHI-S	1.83±0.95	1.80 (1.00–2.40)	1.64–2.02	0.4–3.8
Gingival index	1.56±0.81	1.55 (0.85–2.10)	1.40–1.72	0.5–3.2
BOP (%)	42.1±18.6	41.0 (28.0–55.0)	38.4–45.8	10–80
No. of teeth	24.2±3.7	25 (21–28)	23.5–24.9	16–30
Bone loss (%)	28.5±12.2	27 (18–38)	26.1–30.9	10–60

All values analyzed with descriptive statistics. 95% CI shown for precision. SD: Standard deviation, CI: Confidence interval, IQR: Interquartile range, PPD: Probing pocket depth, CAL: Clinical attachment loss, BOP: Bleeding on probing

Inflammatory, metabolic, and immunologic markers

Biochemical analysis revealed significant systemic inflammation, oxidative stress, and metabolic abnormalities among cases (Table 5).

Periodontitis patients showed significant increases in inflammatory cytokines (IL-1β), matrix metalloproteinase (MMP-8), oxidative stress marker (MDA), metabolic markers (HbA1c, LDL, TG), and pathogen-specific antibodies. HDL was significantly reduced, suggesting dyslipidemia.

Comorbidity burden

The distribution of systemic comorbidities among cases reveals that 28% had no comorbid conditions, 32% had one, 24% had two, and 16% had all three (CVD, respiratory illness, and post-COVID complications).

Nearly three-fourths (72%) of patients with chronic periodontitis exhibited at least one systemic comorbidity, reinforcing the strong oral-systemic inflammatory connection and highlighting the need for integrated healthcare approaches.

DISCUSSION

This research delineates the intricate associations between chronic periodontitis and systemic health outcomes, evidencing pronounced links to inflammatory, metabolic, and immunological dysregulation in

Table 4: Frequency of systemic conditions in cases versus controls (n=130)

Condition	Cases yes (%)	Controls yes (%)	OR (95% CI)	χ^2	p-value
CVD	54 (54)	0 (0)	70.4 (4.19–1184.4)	25.53	<0.0001**
URTI	56 (56)	0 (0)	76.4 (4.54–1284.4)	27.27	<0.0001**
COVID-19	46 (46)	0 (0)	51.1 (3.04–859.5)	19.39	<0.0001**

Statistical test: Chi-square; odds ratio (OR) with 95% CI calculated using 2×2 tables. p<0.001=highly significant. CVD: Cardiovascular disease, URTI: Upper respiratory tract infection, CI: Confidence interval

Table5: Inflammatory, metabolic, and immunologic markers in study groups

Marker	Cases mean±SD	Controls mean±SD	Mean difference	95% CI	t-value	p-value
CRP (mg/L)	10.09±5.12	10.40±5.38	−0.31	−2.66–2.04-0.286 0.776		
ESR (mm/h)	25.26±12.46	31.57±12.61	−6.31	−11.52–1.09		−2.409 0.020*
hs-CRP (mg/L)	3.46±1.17	2.26±0.88	+1.20	0.80–1.60		6.095<0.001**
IL-1β (pg/mL)	25.03±7.88	18.01±6.02	+7.02	4.23–9.81		5.043<0.001**
MMP-8 (ng/mL)	50.15±11.97	29.91±10.02	+20.24	16.03–24.459.265<0.001**		
Fibrinogen (mg/dL) 409.9±59.8		379.7±54.9	+30.2	8.0–52.4		2.693 0.008**
NLR	3.23±0.78	2.09±0.61	+1.14	0.85–1.43		8.136<0.001**
HbA1c (%)	6.49±0.93	5.24±0.57	+1.25	0.94–1.56		7.643<0.001**
MDA (nmol/mL)	2.47±0.59	1.81±0.41	+0.66	0.47–0.85		7.004<0.001**
LDL (mg/dL)	139.6±24.8	119.8±20.3	+19.8	10.8–28.8		4.355<0.001**
HDL (mg/dL)	42.1±7.8	50.0±8.9	−7.9	−11.3–4.5-4.612<0.001**		
Triglycerides (mg/dL)	170.3±39.9	129.6±29.9	+40.7	27.2–54.2		6.158<0.001**
IgG- *Porphyromonas gingivalis*	15.0±5.0	9.9±2.9	+5.1	3.6–6.6		7.051<0.001**
IgA-*Porphyromonas gingivalis*	12.1±3.9	8.0±2.4	+4.1	2.9–5.3		6.617<0.001**

Statistical test: Independent t-test. p<0.05=significant; *p<0.01=highly significant. SD: Standard deviation, IgG: Immunoglobulin G, IgA: Immunoglobulin A, hs-CRP: High-sensitivity C-reactive protein, ESR: Erythrocyte sedimentation rate, IL-1β: Interleukin-1 beta, MMP-8: Matrix metalloproteinase-8, NLR: Neutrophil-lymphocyte ratio, HbA1c: Glycated hemoglobin, MDA: Malondialdehyde, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, CI: Confidence interval

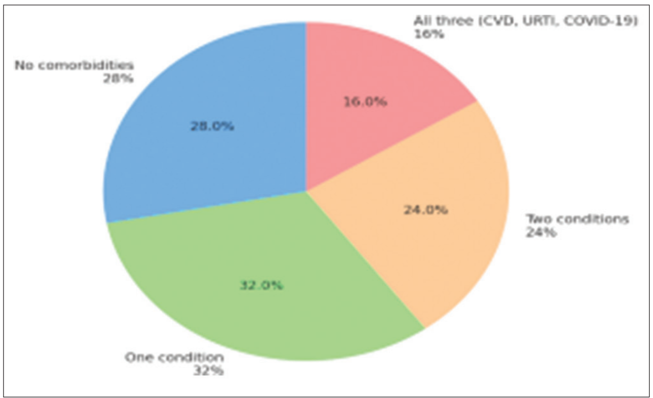

Fig. 3: Comorbidity burden among periodontitis patients

a cohort comprising 130 individuals (100 periodontitis cases and 30 controls). The data affirm that periodontitis patients display extensive periodontal tissue loss alongside elevated systemic comorbidity prevalence, augmented inflammatory biomarkers, and perturbed metabolic signatures relative to controls. These patterns converge with extant literature endorsing the reciprocal interplay of oral and systemic pathologies, principally via chronic low-grade inflammation and shared etiological factors. Specifically, the elevated levels of hs-CRP, IL-1β, and MMP-8 underscore a persistent inflammatory state extending beyond the oral cavity, which contributes to systemic disease pathogenesis [17]. Such systemic inflammation has been implicated in exacerbating conditions like CVD, respiratory tract infections, and even viral infections such as COVID-19, aligning with the observed higher prevalence of these comorbidities in periodontitis patients [18,19]. The significant increase in IL-1β in periodontitis patients, as observed in this study, further supports its established role as a key pro-inflammatory cytokine in the pathogenesis of periodontal disease and its systemic sequelae [20]. This systemic inflammatory burden is further complicated by alterations in lipid profiles, with notable increases in LDL and TG and a reduction in HDL, contributing to an elevated risk for cardiovascular events [18,21]. Moreover, the heightened levels of MDA provide clear evidence of increased oxidative stress, a critical factor in the progression of both periodontal disease and various systemic inflammatory conditions [22,23].

Demographically, the study population was well-balanced between cases and controls regarding age, gender, and residence, with no statistically significant differences. However, notable trends emerged in educational attainment and socioeconomic status, with cases showing a higher prevalence of lower education levels and low SES. Although these differences did not reach statistical significance (p=0.186 and p=0.205, respectively), they remain clinically meaningful and align with prior studies showing that social factors like low education and income increase periodontitis risk by limiting dental care access and health literacy. There are several studies demonstrated that lower SES is associated with poorer oral hygiene practices and increased periodontitis prevalence, potentially amplifying systemic inflammation through untreated oral infections [6,24].

Socio-behavioral risk factors further revealed a pattern of elevated tobacco use (both smoking and chewing) and irregular brushing among cases, though these differences were not statistically significant, possibly attributable to the relatively small control group size. This lack of significance notwithstanding, the observed trends are consistent with established literature linking tobacco as a major modifiable risk factor for periodontitis progression, as it impairs immune responses and promotes dysbiotic oral microbiomes [25,26]. Similarly, irregular oral hygiene practices correlate with higher plaque accumulation, as evidenced by the mean PI of 1.49±0.87 in our cases, reinforcing the need for targeted behavioral interventions in high-risk populations. Clinically, the periodontal parameters in cases confirmed moderate-to-severe disease, with a mean PPD. The high OHI S score (1.83±0.95) further highlights suboptimal oral hygiene as a contributing factor, which may perpetuate a cycle of local and systemic inflammation.

A striking finding was the significantly higher prevalence of systemic comorbidities in periodontitis cases, including CVD. Similarly, the strong association with URTI underscores the oral-respiratory axis, where periodontal bacteria may contribute to respiratory infections through microaspiration. The HbA1c levels were notably elevated in periodontitis patients, further cementing the bidirectional relationship between periodontal disease and glycemic control [27]. These findings corroborate previous research indicating that periodontitis patients frequently exhibit a higher burden of systemic illnesses, such as hypertension, diabetes, and CVD [28].

Biochemical analysis revealed systemic effects in periodontitis patients, who showed significantly higher levels of inflammatory markers. They also had elevated IgG and IgA antibodies against *Porphyromonas gingivalis*. These elevated antibody titers against a keystone periodontal pathogen further substantiate the systemic immunological response triggered by chronic periodontitis, indicating a persistent antigenic challenge that contributes to widespread inflammation [29]. This sustained immunological activation and the resulting inflammatory milieu are critical drivers in the progression of various non-communicable diseases, aligning with observations of heightened systemic inflammation in periodontitis patients [8,30]. CRP levels showed no significant difference (p=0.776), but hs-CRP, a more sensitive marker of inflammation, was markedly elevated in cases, suggesting that standard CRP may lack the precision to detect subtle inflammatory changes in periodontitis. The reduced erythrocyte sedimentation rate in cases (p=0.020) indicates the need of further investigation, possibly reflecting compensatory mechanisms or confounding factors like medication use. However, the robust elevation of hs-CRP, a sensitive marker of systemic inflammation, strongly suggests an active inflammatory process that transcends localized periodontal pathology and significantly impacts overall systemic health [29,31]. Given the established links between chronic periodontitis and systemic inflammation, these findings further support the hypothesis that periodontal infections contribute significantly to the overall inflammatory burden, potentially exacerbating various chronic diseases [32].

CONCLUSION

This study suggests the potential association of chronic periodontitis through elevated inflammatory markers, altered lipid profiles, increased oxidative stress, and higher comorbidity rates. These changes position periodontitis as a contributor to systemic inflammation and risk for non-communicable diseases. Clinically, findings support integrated management, bidirectional screening for periodontitis and systemic conditions, with dentists identifying metabolic/cardiovascular risks and physicians assessing periodontal status. Public health initiatives should prioritize tobacco cessation, oral hygiene education, and low-SES targeting, through interdisciplinary efforts to lessen disease burden, enhance outcomes, and cut costs.

AUTHORS CONTRIBUTION

All the authors have equal contribution.

CONFLICTS OF INTEREST

None.

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