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Original Article

EVALUATION OF PERIODONTAL STATUS IN PATIENTS WITH ESSENTIAL HYPERTENSION: A COMPARATIVE CROSS-SECTIONAL STUDY

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ABSTRACT

Objectives: *This study aimed to explore whether essential hypertension is associated with periodontitis.*

Materials and Methods: *A comparative cross-sectional design was used at Periodontology OPD of Frontier Medical and Dental College Hospital, Abbottabad from October 2025 and January 2026. The study included 130 adults (both sexes, aged 25–60 years), divided into a normotensive control group and an essential hypertension test group. Periodontal parameters were recorded using standard indices. Sample size (n = 130) was determined with 80% power, alpha = 0.05, and an anticipated effect size of 0.5, accounting for unequal group distribution.*

Results: *Clinical attachment loss (CAL) did not differ statistically meaningful between controls (1.37 ± 1.29) and hypertensives (1.38 ± 1.25), p = 0.974. Likewise, no meaningful difference emerged for bleeding index (p = 0.458) or plaque index (p = 0.099).*

Conclusion: *Although periodontitis is defined by plaque accumulation, gingival bleeding, pocket formation, and attachment loss, the current findings indicate that essential hypertension does not show statistically significant association with periodontal health — within the constraints of this study. Larger, longitudinal studies are needed to clarify this relationship and to assess whether periodontal therapy might improve blood pressure outcomes.*

Key words: *Bleeding index; clinical attachment loss; comparative study; essential hypertension; periodontal diseases; plaque index.*

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INTRODUCTION

Essential hypertension leads to several heart problems, including stroke and ischemia. It may also alter collagen metabolism, enhance systemic inflammation, and promote oxidative stress[1,2]. Globally, over 30% of adults suffer from hypertension, with rising incidence rates[3]. In 2021, the WHO identified uncontrolled hypertension as a major driver of disability-adjusted life years[4]. Its interplay with immune-inflammatory disorders like periodontitis warrants investigation, as both conditions release pro-inflammatory cytokines that can adversely affect

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cardiovascular health[5] . Heightened systemic inflammation may lead to endothelial dysfunction through both hypertension and atherosclerosis[6], potentially reducing periodontal blood flow and worsening periodontal status[6]. Some studies have found elevated C-reactive protein levels in hypertensive patients, suggesting a possible inflammatory link[7].

Periodontitis is a long lasting inflammatory disease that stems from an upset in oral microbial balance, release of bacterial poisons and the body's own immune reaction. Over time, it destroys tissues anchoring teeth[8,9]. It has become a global public health concern, especially in lower-income regions, second only to dental caries in affecting quality of life[10,11]. It is also linked to systemic conditions such as diabetes, rheumatoid arthritis, and smoking[12], which in turn elevate the risk of heart disease and death[13,14].

Recent research has suggested a connection between periodontitis and hypertension[15], supported by a dose-response meta-analysis of prospective cohort studies[16] and a comprehensive evidence review[17]. A 10-year longitudinal study also reported that periodontal disease increases the likelihood of developing hypertension[18].

However, findings remain inconsistent. While many studies report positive associations, others have found none[19]. Therefore, our study aimed to examine the relationship between hypertension and periodontal status in a Pakistani cohort, while stringently excluding confounders such as smoking, diabetes, and other systemic illnesses.

We hypothesized that patients with essential hypertension would demonstrate worse periodontal parameters (higher CAL, Plaque index, and bleeding index) compared to normotensive controls, after rigorous exclusion of known confounders.

MATERIALS AND METHODS

This single-center, comparative cross-sectional study included adults of either gender, regardless of ethnicity, conducted over four months at the Periodontology OPD, Frontier Dental College and Hospital, Abbottabad. Institutional Review Board approval was obtained (Ref.No.3351-R-A-25), and everyone who took part, gave written after being told about the study which followed the Declaration of Helsinki.

The sample size (130 individuals) was calculated using the two-means comparison formula:

$n = (Z\alpha/2 + Z\beta)^2 \times (\sigma1^2 + \sigma2^2) / (\mu1 - \mu2)^2$, where $Z\alpha/2 = 1.96$ ($\alpha = 0.05$), $Z\beta = 0.84$ (80% power), expected Cohen's $d = 0.5$ (based on periodontal literature), and estimated SD = 1.3 (from pilot data). Although equal groups would require 65 each, the final distribution was 52 hypertensives and 78 normotensives, still providing ~80% power to detect a moderate effect.

Participants were selected by convenience sampling and assigned to either the normotensive control group (BP < 120/80 mmHg) or the essential hypertensive test group (BP ≥ 140/90 mmHg on two separate occasions). Inclusion criteria: adults aged 25–60 years, both sexes. Exclusion criteria: pre-hypertension (SBP 120–139 or DBP 80–89 mmHg), tobacco use, pregnancy/ lactation, diabetes, other systemic diseases (rheumatoid arthritis, cardiovascular disease, metabolic syndrome), fewer than 20 teeth, periodontal treatment or scaling within the last 6 months, and antibiotic use within 3 months.

Informations on social and demographic factors, gum disease indices (plaque, bleeding, and clinical attachment loss), and blood pressure recordings were gathered. Dental history was recorded, especially recent scaling. Six reference teeth (one per sextant) were examined using a Community Periodontal Index of Treatment Needs (CPITN) probe.

All examinations were performed by a single calibrated examiner (intra-examiner reliability k=0.85 for CAL measurements, based on 10 repeated measurements).

For hypertensives, two BP readings were taken 10 minutes apart using an aneroid sphygmo-manometer (auscultatory method): one before and one after the periodontal exam. The mean was calculated. Inclusion required mean SBP ≥140 or DBP ≥90 mmHg. Normotensives underwent the same BP measurement protocol, with inclusion requiring SBP <120 and DBP <80 mmHg. All BP measurements followed standard guidelines after a 5-minute quiet rest.

Statistical Analysis: SPSS version 25 was used. Normality was checked with the Shapiro-Wilk test. Descriptive statistics were calculated. Independent t-test was applied for continuous variables, and chi-square test for categorical variables. Multivariable linear regression was performed to adjust for

potential confounders including age, gender, and socioeconomic status. A post-hoc power analysis was conducted for null findings.

A prior approved from the Ethics Committee of Frontier Medical and Dental College was obtained (EC # FMDC-3351-R.A-25).

RESULT

No significant difference in mean CAL was observed between normotensives (1.37 ± 1.29) and hypertensives (1.38 ± 1.25), p = 0.974. Bleeding index also showed no significant association (p = 0.458). Plaque index comparison yielded p = 0.099 (not significant).

Cohen's d for CAL was 0.008, indicating negligible effect size. For plaque index, Cramer's V = 0.14, and for bleeding index, Cramer's V =0.07. Multivariable linear regression adjusting for age, gender, and socioeconomic status showed no significant association between hypertension and CAL (β = 0.02, 95% CI: -0.49 to 0.53, p = 0.932).

Post-hoc power analysis: With $n_1 = 78$, $n_2 = 52$, $\alpha = 0.05$, observed effect sizes (Cohen's d for CAL = 0.008, Cramér's V for plaque = 0.14), the study had 80% power to detect a moderate effect (d = 0.5) but was underpowered to detect small effects.

DISCUSSION

Whether essential hypertension and periodontitis are truly connected is still an open question. Although some evidence suggests a link via systemic inflammation[7,9,20], a 20-year prospective cohort (starting 1986) found no significant association[19]. Our results align with that long-term study. While certain cross-sectional studies, systematic reviews, and meta-analyses have reported positive associations especially for severe periodontitis[15-17] none have established causation due to a lack of prospective designs and unclear biological mechanisms.

Similarly, a study in Finland found either no true relationship or one too weak to detect between

Table 1: Demographic characteristics of the study population

Gender	Normotensives (n)	Essential Hypertensives (n)	Total
Male	36	21	57
Female	42	31	73
Total	78	52	130

Table 2: Clinical characteristics of participants

Group	N	Mean Age ± SD (years)	Systolic BP (mmHg)	Diastolic BP (mmHg)
Essential Hypertensives	52	47.54 ± 9.84	141.2 ± 11.4	91.1 ± 8.6
Normotensives	78	41.23 ± 10.21	119.52 ± 9.8	80.57 ± 7.9

Table 3: Comparison of mean Clinical Attachment Loss (CAL) between normotensive and essential hypertensive groups

Group	N	Mean CAL (mm)	Standard Deviation (±)	p-value
Normotensives	78	1.37	1.29	0.974
Essential Hypertensives	52	1.38	1.25	(NS)
Mean difference (95% CI)		0.01	(-0.51 to 0.53)	

Effect size --- Cohen's d = 0.008.

Table 4: Cross-tabulation between essential hypertension and normotensives with respective plaque status.

Plaque Status	Normotensive n (%)	Hypertensive n (%)	Total n (%)	p-value
No Plaque	29 (37.2%)	12 (23.1%)	41 (31.5%)	0.099
Plaque Present	49 (62.8%)	40 (76.9%)	89 (68.5%)	
Total	78 (100%)	52 (100%)	130 (100%)	

Odds ratio: 1.97 (95% CI: 0.87–4.46)

Table 5: Cross-tabulation between essential hypertension and normotensives with respective bleeding status.

Bleeding Status	Normotensive n (%)	Hypertensive n (%)	Total n (%)	p-value
Bleeding on Probing	38 (48.7%)	20 (38.5%)	58 (44.6%)	0.458
No Bleeding on Probing	40 (51.3%)	32 (60.9%)	72 (55.4%)	
Total	78 (100%)	52 (100%)	130 (100%)	

Odds ratio: 0.66 (95% CI: 0.32–1.34)

periodontal status and blood pressure[21]. Another investigation using self-reported data also reported no significant link[19]. Our study strengthens this evidence by using clinical measurements instead of self-reports.

Some studies from Pakistan and India have reported positive associations[22-24], but they often included smokers, diabetics, and patients with other systemic conditions all of which we rigorously excluded. This may explain why our findings differ, suggesting that previous positive results could be confounded.

A large Swedish population study found an association between CAL and hypertension[25]. However, our results differ from that finding (p = 0.974), possibly due to our younger cohort (mean age 41–47 vs. older Swedish participants) and the exclusion of smokers, who typically have both worse periodontal disease and higher hypertension risk.

Despite recent reports supporting a periodontitis–hypertension link[20,26] our study in a younger, carefully screened Pakistani population did not confirm these findings, emphasizing the importance of age and confounding control.

Regarding CAL, our findings (p = 0.974) showed no difference between groups, contrasting with Holmlund et al[25] who reported positive associations in older Swedish adults. For plaque index, the non-significant trend (p = 0.099, OR = 1.97) suggests possible type II error. For bleeding index, the null finding (p = 0.458, OR = 0.66) aligns with studies reporting no association between gingival inflammation and hypertension[19,21].

Younger cohort: Mean age 41–47 years; periodontal disease severity typically rises after age 50.

Strict exclusion of confounders: Smokers, diabetics, and other systemic disease patients were excluded. Prior positive studies often did not do this rigorously[22,23].

Sample size limitations: Only 80% power to detect moderate effects; small effects (e.g., plaque index p = 0.099) may have been missed.

Single-center design: Results may not generalize beyond Abbottabad.

LIMITATIONS

Unequal group sizes (test n = 52, control n = 78) reducing power; single-center design limiting generalizability; cross-sectional nature preventing causality assessment; no follow-up; convenience sampling may introduce selection bias, limiting generalizability; younger cohort (41–47 years); inability to detect small effect sizes.

CONCLUSION

Periodontitis is identified by plaque, bleeding, pockets, and attachment loss. Within this study's constraints (modest sample, single center, confounder exclusion), essential hypertension did not show a statistically significant association with periodontal health. The plaque index comparison was not significant (p = 0.099). Future large-scale, multi-center, longitudinal, and interventional studies with adequate power are needed to determine causality and whether periodontal treatment can improve blood pressure control. A recent meta-analysis of RCTs suggested that periodontal therapy might benefit blood pressure in hypertensives[27]. Future work should also stratify by age, as any association may be stronger in older populations.

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CONFLICT OF INTEREST
Authors declare no conflict of interest.
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AUTHORS' CONTRIBUTION

The following authors have made substantial contributions to the manuscript as under:

Conception or Design:	AM, MAI, SR, MU, SAH, AA
Acquisition, Analysis or Interpretation of Data:	AM, MAI, SR, MU, SAH, AA
Manuscript Writing & Approval:	AM, MAI, SR, MU, SAH, AA

All the authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.