Discoveries in Applied and Natural Science
Applied and Natural Science
Volume: 4 Issue: 1 Year: 2026
ISSN: 3030-301X | http://journals.proindex.uz

Bacteriological and Physiological Assessment of Porphyromonas gingivalis in Patients with Hypertensio

Mayson kooshi jasem
Babylon Technical Institute, Al-Furat Al-Awsat Technical University, 51015

Abstract: Hypertension is a multifactorial cardiovascular disorder that is gaining association with chronic inflammation and microbial influence, over and above the risk profile of hypertension. Among oral pathogens, Porphyromonas gingivalis has received increasing attention due to its role in periodontal dysbiosis as well as its potential systemic effects of chromosomes on endothelial function and inflammation as well as metabolic disturbance. The present study was prepared and performed a bacterium physiological evaluation of P. gingivalis in hypertensive patients and also its association with certain physiological parameters related to cardiovascular. This analytical case-control study consisted of 75 adults divided into two groups, 50 hypertensive patients and 25 participant groups (normotensive). Demographic and physiological variables were recorded, age, sex, body mass index, total cholesterol, and triglycerides. The blood pressure was measured using standard clinical procedures. Subgingival plaque samples were taken and analyzed for the presence of P. gingivalis by real-time polymerase chain reaction. Statistical analysis was done to assess the difference between the groups and to assess the correlation between bacteriological positivity and physiological parameters. The results revealed that hypertension was more common among the older subjects and was significantly related to high body mass index. Obesity was significantly more common in the hypertensive than in the normotensive controls. Although abnormally elevated cholesterol and triglycerides levels were more common in hypertensive patients, these differences were not statistically significant in the current sample. The bacteriological evaluation showed significantly higher frequency of positive presence of P. gingivalis in hypertensive patients than control. Within the hypertensive group, P. gingivalis positivity was more common in patients with obesity, total cholesterol and triglycerides. In addition, hypertensive patients positive for P. gingivalis showed greater mean systolic blood pressure, diastolic blood pressure, body mass index, total cholesterol and triglyceride values compared with Bacteriologically negative patients. the present findings were consistent with an association between the detection of P. gingivalis and an unfavorable physiologic profile in hypertensive patients. The co-existence of hypertension, obesity, dyslipidemia, and periodontal pathogens burden implies a biologically plausible interaction between the oral and systemic systems possible to pathogenesis to the expression of the cardiovascular risk. These findings give support for the need of larger studies integrating bacteriological, periodontal, inflammatory and vascular assessments to elucidate the clinical significance of this relationship.

Key points: Hypertension; Porphyromonas gingivalis; Periodontitis; Body mass index; Dyslipidemia; Cardiovascular risk; Real time polymerase chain reaction; Oral systemic association

Introduction

Hypertension is one of the most significant modifiable causes of cardiovascular morbidity and premature mortality worldwide and current guidelines continue to recognise blood pressure control as a main foundation of cardiovascular risk reduction [1]. Current pathophysiological models are no longer considering hypertension as a pure hemodynamic disorder, but rather as a chronic immuno-inflammatory disorder involving a dysregulation of cytokines, oxidative stress, endothelial dysfunction, vascular remodeling and target organ damage [2]. Periodontitis is a dysbiosis-driven, chronic inflammatory disease of the tooth supporting tissues, and recent growing interest has focused on its systemic nature since chronic, ongoing inflammation in the oral cavity has the

potential to reach beyond the periodontal microenvironment and affect disease pathways outside of the mouth [3]. Among the microorganisms implicated in periodontal dysbiosis, *Porphyromonas gingivalis* is regarded as a keystone pathogen because of its role in host immune system subversion, lack of correlation between inflammation and bacterial clearance and the shift of the oral ecosystem towards destructive chronic inflammation [4]. The pathogenic effects of *P. gingivalis* are mediated through a wide variety of virulence that include gingipains, fimbriae, atypical lipopolysaccharide, peptidyl arginine deiminase, hemagglutinins, and other factors that contribute to colonization, immune evasion, tissue invasion, and cellular injury in host cells [5]. An additional feature to reinforce the systemic relevance of this bacterium is the release of outer membrane vesicles that are efficient long-range virulence cargo carriers, which enable *P. gingivalis* to impact host tissues even when not associated with whole-cell dissemination [6]. From a cardiovascular standpoint there is a growing body of oral microbiome research in favor of the hypothesis that oral pathogens and their products may affect vascular disease by bacteremia, endotoxemia, inflammatory cytokine release, immunological activation, nitric oxide perturbation and metabolic signaling by microbes [7]. There has been further experimental evidence demonstrating that *P. gingivalis* outer membrane vesicles directly enhance endothelial permeability through the formation of stress fibers and vascular endothelial-cadherin degradation, which provides a plausible mechanistic basis for vascular injury and blood pressure dysregulation [8]. Epidemiological evidence has also reinforced the oral-systemic correlation and recent observational studies have continued to show significant associations between periodontitis and hypertension in adult populations although the size of association observed varies between cohorts and the study designs [9]. More importantly, a 5-year longitudinal study showed that increased probing depth, increased percentage of deep periodontal sites and poor oral hygiene were linked to increased systolic blood pressure and incident hypertension, with white blood cell count and C-reactive protein partially mediating the associations [10]. This biologic plausibility is borne out by evidence from intervention-based clinical trials by a recent meta-analysis demonstrating that non-surgical periodontal therapy can reduce systemic inflammatory markers and systolic blood pressure, leading to the speculation that at least some of the associated cardiovascular burden of periodontal inflammation may be modifiable [11]. At the pathogen-specific level, clinical evidence has demonstrated that *P. gingivalis* abundance is higher in subjects with established atherosclerotic cardiovascular disease compared with healthy controls, suggesting that this organism may actually be associated with high risk for the development of high-risk vascular phenotypes rather than the bacteria being a marker of local oral disease [12]. In cases of hypertensive patients with chronic periodontitis, salivary *P. gingivalis* levels were shown to diminish following periodontal debridement but in contrast blood pressure did not show significant improvement over short term follow up therefore pointing out the fact that while the bacteriological and physiological relationship is real it is mechanistically complex and is not yet completely unriddled [13]. The clinical significance of this relationship is strengthened by recent data derived from the cohort evidence suggesting an association between periodontitis and increased cardiovascular and all-cause mortality in persons with hypertension, thus extending beyond the discussion of association to prognosis [14]. Despite this progress, more recent reviews continue to highlight that the human evidence is heterogeneous and more studies are needed which directly incorporating the pathways of oral microbes with a well-defined, clearly phenotype cardiovascular outcomes in well-defined populations [15]. Accordingly, the present study was designed to carry on a bacteriological and physiological evaluation of *Porphyromonas gingivalis* in hypertensive patients aimed at clarifying the possible association with selected physiological alterations relevant to cardiovascular risk. Periodontitis is no longer considered a strictly localized disease of the mouth, as the latest evidence shows periodontal diseases are associated with systemic loads of inflammation through dysbiotic biofilms, chronic low-grade inflammation and spreading of periodontal pathogens and their products [16]. A recent 5-year longitudinal study found that increased periodontal probing depth, increased percentage of deep periodontal sites and poor oral hygiene were related to the development of increased systolic blood pressure and increased incidence of hypertension, with white blood cell count and C-reactive protein partly mediating these associations [17]. A recent systematic review and meta-analysis further indicated that periodontitis was associated with an increased cardiovascular disease risk in people who had already metabolic syndrome precursors like hypertension, obesity, dyslipidemia or dysglycemia [18]. In hypertensive populations specifically,

there has been a NHANES-based cohort evidence showing that periodontitis has been associated with increased all-cause mortality and cardiovascular mortality, which helps support the clinical relevance of investigating oral inflammatory burden in this patient group [19]. At a pathogen-specific level, profiling studies using PCR have reported an association between *P. gingivalis* and hypertension and dyslipidemia, suggesting that this organism may be of greater direct relevance to cardiometabolic risk than is generous periodontal status to these health processes [20]. The biologic plausibility of this association is supported by the latest microbiome research indicating that microbial communities in the oral cavity may affect the regulation of vascular health systemically (by immuno-inflammatory mechanisms) and through nitrate-nitrite-nitric oxide pathways of significance for cardiovascular homeostasis [21]. The interventional evidence in humans also remains incomplete, as salivary *P. gingivalis* levels were also shown to decrease following periodontal debridement in a group of hypertensive patients with chronic periodontitis, but the blood pressure was not significantly improved with short term follow up, suggesting that bacteriological change and physiological response may not move in parallel [22]. Recent reviews still conclude that the human data supporting an oral microbiota link to cardiovascular disease is mixed and that additional controlled population-based studies are necessary in order to link specific oral microbial pathways with well-defined cardiovascular phenotypes [23].

Aim of the Study

To bacteriologically identify the *Porphyromonas gingivalis* in hypertensive patients and to assess its correlation with selected physiological parameters linked to the cardiovascular risk, including profile of blood pressure, body mass index, and parameters related to the lipid profile.

Materials and Methods

Study design and setting

This analytical case-control study was carried out to know the bacteriological detection of *Porphyromonas gingivalis* in patients with hypertension and also to assess its relationship with some physiological parameters associated with cardiovascular risk. The study was conducted at clinical private, [Hilla, Iraq], in the period between [December, 2026] - [February, 2026]. [25].

Study population

A total of 75 adult subjects were recruited in the study and were divided into two groups. The case group had 50 patients with hypertension and the control group had 25 apparently healthy normotensive persons. Points of Recruitment Participants were recruited sequentially from outpatient clinics when clinical evaluation and eligibility criteria were met.

Inclusion criteria

The hypertensive group included adults 20 years and older who either had a history of medical diagnosis of hypertension, were on antihypertensive therapy or had office blood pressure values indicative of hypertension by contemporary recommendations of the European Society of Cardiology. The control group consisted of adults who did not have any known history of hypertension and had normal blood pressure at the time of enrollment in the study. Hypertension was categorized according to current ESC criteria, in which an office blood pressure of ≥140/90 mmHg was called hypertension [26].

Exclusion criteria

Participants were excluded if they received systemic antibiotics within the last 3 months, periodontal treatment within the last 6 months, had fewer than 6 natural teeth, had pregnancy, malignancy, chronic inflammatory or autoimmune disease, chronic kidney disease, acute systemic infection and any organ system disorder that could specifically change the oral microbial composition or the inflammatory state. These restrictions were imposed in order to minimize important sources of confounding in the periodontal and molecular bacteriological assessment.

Blood pressure measurement

Blood pressure was assessed using an automated sphygmomanometer with an appropriately sized blood pressure cuff after at least 5 minutes sitting rest. Three readings were taken at factors of 1-2 minutes apart and the mean of the last two measurements used for analysis. Measurements were done following modern blood pressure evaluation principles suggested within recent ESC guidance [27].

Collection of demographic and physiological data

A structured data sheet was used to capture age, sex, medical history, medication use and related clinical information. Body weight was measured accurately to the nearest 0.1 kg using a calibrated digital scale and height was measured accurately to the nearest 0.5 cm using a wall-mounted stadiometer. Body mass index was used as an indicator of obesity and was calculated as weight in kilograms divided by height in meters squared. After overnight fasting for 8 to 12 hours, the venous blood samples were collected from all of the participants under aseptic conditions. Serum was separated by centrifugation and used for the estimation of total cholesterol, triglycerides by standard enzymatic colorimetric procedures by using kits from different manufacturers by following the instructions provided by them.

Periodontal condition clinical examination

All the participants were subjected to periodontal examination by a trained examiner. Probing depth, clinical attachment loss and bleeding on probing were measured at six sites per tooth using a manual periodontal probe. Periodontal diagnosis and classification were determined using the 2018 AAP/EFP classification system, still the current reference system for the definition, staging, and grading of periodontitis cases in both the clinic and in research [28].

Fold of subgingival plaque samples

Subgingival plaque was chosen as the bacteriological specimen because it was a site-specific representation of periodontal pathogen burden and was commonly used in molecular studies targeting *P gingivalis*. Before the sampling supragingival plaque was removed gently and the sampling site was sheltered using cotton rolls and then air-dried. In the participants with periodontitis, the sample was taken from the deepest periodontal pocket, and in those without deep pockets, sampling was taken from the deepest gingival sulcus. Two sterile paper points were placed into the selected site for 30 seconds and then were placed into sterile microcentrifuge tubes with transport buffer. Samples were immediately stored at -80^{o}C until molecular processing. Real-time PCR studies and more recent evaluations of diagnostic methods continue to support subgingival plaque as a good specimen for the detection of *P. gingivalis* [29].

DNA extraction

Microbial DNA was extracted from the collected plaque samples by a silica membrane based commercial extraction kit such as QIAamp DNA Minikits (Qiagen, Hilden, Germany) according to the protocol of the manufacturer. The purity of DNA and concentration were assayed spectrophotometrically before amplification.

Detection of *Porphyromonas gingivalis* by real-time PCR

Detection of *P. gingivalis* was carried out by real-time polymerase chain reaction (Lamp, Megalopolis, Germany) of the bacterial *16S rRNA* gene. Real-time PCR was chosen as it was highly sensitive and specific in the detection of fastidious periodontal pathogens and was currently widely applied for the microbiological diagnosis in periodontitis [30]. A validation study found high specificity, and good agreement between results of a novel method of real-time PCR and a well-established commercial assay for periodontal pathogens, indicating the validity of this diagnostic method to detect *P. gingivalis* in subgingival plaque (Kuret *et al*., 2024). Amplification protocols were performed in the total reaction volume of 20 microliters that comprised extracted template DNA, SYBR Green master mix, forward and reverse primers, and nuclease-free water. The primer sequences for use with *P. gingivalis* were forward 5'-CGGGATTGAAATGTAGATGATGATGG-3' and reverse 5'-ACACCTTCCTCACGCCTTACG-3', resulting in a 200-bp-amplicon. Thermal cycling conditions were initial denaturation at 95^{o}C for 10 minutes, 40 rinse cycles of denaturing at 95^{o}C followed by annealing/extension at 60^{o}C. A melting curve analysis was done at the end of the runs to check for amplification specificity. A positive control with the known *P. gingivalis* DNA and a no template negative control were included in every run to prevent DNA contamination. Samples were termed as positive if amplification crossed a prudence-threshold and if it exhibited a particular melting profile as the one of the target organisms. Real-time PCR has also been shown to be more quantitative than the rapid chairside immunochromatographic detection method; however, the two have been shown to be in good agreement as recently evaluated [31][32][33][34].

Outcome variables

The main bacteriological outcome of the study was the presence or absence of *P. gingivalis* DNA in *P. gingivalis* subgingival plaque samples. The main physiological variables were systolic blood

pressure, diastolic blood pressure, body mass index, total cholesterol and triglycerides. Age and sex were selected as secondary variables.

Statistical analysis

Data were entered into the computer software Microsoft Excel and analyzed by the statistical software package Version 26.0 or its equivalent programme, with the use of the statistical package 'SPSS'. Continuous variables were represented as mean ± SD while categorical variables were represented as frequency and percentage. Normality of the continuous data was checked before inferential analysis. Independent-samples t-test was applied to compare values of normally distributed continuous variables from 2 groups; the Mann-Whitney U test was applied when a normality assumption could not be met. The Chi-square test or Fisher's exact test was used to compare categorical variables. Logistic regression analysis could further be used to evaluate if *P. gingivalis* positivity was independently related to hypertension after adjusting for age, sex, body mass index and lipid variables. A P value less than 0.05 was considered to be statistically significant.

Ethical considerations

The study protocol received the approval of the Institutional Research Ethics Committee. Written informed consent was obtained from all participants prior to being enrolled, clinically examined and having biological samples collected. All the procedures were performed in compliance with the ethical norms of the Declaration of Helsinki.

Results

A total of 75 subjects were recruited in the present study comprising 50 hypertensive patients and 25 normotensive controls. The distribution of ages among the hypertensives showed a steady increase in the older age groups. The highest proportion of hypertensive patients was seen in the age group of 60-69 years (32.0%) followed by 50-59 years old age group (28.0%), and low proportions were recorded in younger age groups. These results suggest that hypertension was more common in older people from the study population.

Table 1. Age-groups distribution among hypertensive patients

Age group (years)	No.	%
20–29	6	12.0
30–39	5	10.0
40–49	9	18.0
50–59	14	28.0
60–69	16	32.0
Total	**50**	**100.0**

Total cholesterol was found high in 37 of 50 hypertensive patients (74.0%), and normal cholesterol levels were found in 13 patients (26.0%). This result suggests hypercholesterolemia was high in hypertensive subjects of present study.

Table 2. Distribution of total cholesterol status of hypertensive patients

Total cholesterol	No.	%
Normal	13	26.0
Elevated	37	74.0
Total	**50**	**100.0**

Analysis of body mass index in hypertensive patients revealed the most common category of patients was obesity which numbered 30 patients (60.0%), followed by overweight patients in 15 patients (30.0%), while only 5 patients (10.0%) had normal body weight. These results support a high prevalence of hypertension in the group of subjects that had an excess of body weight and, notably, obesity.

Table 3. Distribution of body mass index categories among hypertensive patients

BMI category	No.	%
Normal weight	5	10.0
Overweight	15	30.0
Obesity	30	60.0
Total	**50**	**100.0**

High triglycerides were found in 35 hypertensive patients (70.0%) while normal levels were found in 15 patients (30.0%). This finding indicates that hypertriglyceridemia was also numerically frequent for hypertensive people.

Table 4. Distribution of triglyceride status among hypertensive patients

Triglycerides	No.	%
Normal	15	30.0
Elevated	35	70.0
Total	**50**	**100.0**

Comparison of total cholesterol status between hypertensive patient's vs normotensive controls revealed that higher cholesterol status was observed often among hypertensive patients (74.0%) as compared with the controls (56.0%). However, the difference was not statistically significant (P = 0.189), which shows that, although dyscholesterolemia was numerically more common among hypertensive individuals, the link between them was not statistically established in the current sample.

Table 5. Comparison of Total Cholesterol Status of Hypertensive Patients and Normotensive Controls

Total cholesterol	Hypertensive n (%)	Normotensive n (%)	P value
Normal	13 (26.0)	11 (44.0)	
Elevated	37 (74.0)	14 (56.0)	
Total	**50 (100.0)**	**25 (100.0)**	**0.189**

Similar pattern was seen for triglycerides. Measuring blood lipid levels for hypertension and wellness has shown that elevated triglycerides were found in 70.0% of the hypertensive patients and the normotensive controls received 64.0% probabilities of high triglycerides. The difference was not statistically significant (P = 0.793), suggesting that high triglycerides in triglycerides were common in both groups.

Table 6. Comparison of triglyceride status between hypertensive patients and controls (non-disposed patients)

Triglycerides	Hypertensive n (%)	Normotensive n (%)	P value
Normal	15 (30.0)	9 (36.0)	
Elevated	35 (70.0)	16 (64.0)	
Total	**50 (100.0)**	**25 (100.0)**	**0.793**

Body mass index was strongly and significantly linked to hypertension status. Obesity was observed in 60.0% of the hypertensive patients compared with only 12.0% in the normotensive controls, but, on the other hand, normal body weight was observed more often in the controls (64.0%) than in the hypertensive patients (10.0%). the difference between the two groups regarding the BMI categories was highly significant (P < 0.001) and thus suggests a strong relationship between excess body weight and hypertension.

Table 7. Comparison of categories of body mass index in hypertensive patients and normotensive controls

BMI category	Hypertensive n (%)	Normotensive n (%)	P value
Normal weight	5 (10.0)	16 (64.0)	
Overweight	15 (30.0)	6 (24.0)	
Obesity	30 (60.0)	3 (12.0)	
Total	**50 (100.0)**	**25 (100.0)**	**<0.001**

There was a statistically significant difference (P = 0.016) in age-group comparison between hypertensive patients and normotensive controls. The hypertensive group was concentrated mostly in 50-59-year and 60-69-year groups whereas, there was highest proportion in the 40-49 years age group in the control group. These findings support the contribution of increasing age on hypertension in the present study.

Table 8. Comparison of distribution of age group among hypertensive patients and normotensive controls

Age group (years)	Hypertensive n (%)	Normotensive n (%)	P value
20–29	6 (12.0)	3 (12.0)	
30–39	5 (10.0)	6 (24.0)	
40–49	9 (18.0)	11 (44.0)	
50–59	14 (28.0)	2 (8.0)	
60–69	16 (32.0)	3 (12.0)	
Total	**50 (100.0)**	**25 (100.0)**	**0.016**

Sex distribution showed male predominance in both the groups (84.0% of hypertensive patients and 88.0% of normotensive controls) and 16.0% and 12.0% of females, respectively. No statistically significant difference was noted between two groups (P = 0.908), showing that sex was not related to hypertension in the present sample.

Table 9. Comparison between hypertensive patients and normotensive controls for sex distribution

Sex	Hypertensive n (%)	Normotensive n (%)	P value
Male	42 (84.0)	22 (88.0)	
Female	8 (16.0)	3 (12.0)	
Total	**50 (100.0)**	**25 (100.0)**	**0.908**

Molecular analysis of *Porphyromonas gingivalis* detected significantly more positivity rate in hypertensive group compared to normotensives. Positive detection was found to be 31 out of 50 hypertensive patients (62.0%) as compared to 6 out of 25 controls (24.0%). The difference was statistically significant (P = 0.004) indicating that colonizing the mother's gums with *P gingivalis* may be more common in hypertensive individuals.

Table 10. Frequency of *Porphyromonas gingivalis* positive status in hypertensive patients and normotensive controls

P. gingivalis detection	Hypertensive n (%)	Normotensive n (%)	P value
Positive	31 (62.0)	6 (24.0)	
Negative	19 (38.0)	19 (76.0)	
Total	**50 (100.0)**	**25 (100.0)**	**0.004**

In the hypertensive group, *P. gingivalis* positivity was progressively higher in the different BMI groups. Only 20.0% of hypertensive patients with normal body weight were positive, compared with: 40.0% of overweight patients and 80.0% of obese patients. This association was statistically significant (P = 0.004), which means the positivity for *P. gingivalis* was more common in the hypertensive patients with higher body mass index.

Table 11. Association between the positivity of *P. gingivalis* and BMI categories of hypertensive patients

BMI category	Positive n (%)	Negative n (%)	P value
Normal weight	1 (20.0)	4 (80.0)	
Overweight	6 (40.0)	9 (60.0)	
Obesity	24 (80.0)	6 (20.0)	
Total	**31 (62.0)**	**19 (38.0)**	**0.004**

A significant relationship was found between the positivity of *P. gingivalis* and the total cholesterol status. Among the hypertensive patients with normal total cholesterol 30.8% of patients were positive for *P. gingivalis* while positivity among all hypertensive patients with elevated cholesterol was 73.0%. This difference was statistically significant (P = 0.018), indicating a possible close association between presence of *P. gingivalis* and dyscholesterolemia.

Table 12. Association between positivity for *P. gingivalis* and total cholesterol among hypertensive patients

Total cholesterol	Positive n (%)	Negative n (%)	P value
Normal	4 (30.8)	9 (69.2)	
Elevated	27 (73.0)	10 (27.0)	
Total	**31 (62.0)**	**19 (38.0)**	**0.018**

Frequently the positivity of *P. gingivalis* was also found to be significantly linked to triglyceride status. Among hypertensive patients but with normal triglycerides, positivity was seen in only

33.3% of them while the positivity raised to 74.3% in patients with elevated levels of triglycerides. This difference was statistically significant (P = 0.016) thus supporting a possible relationship that exists between the detection of *P. gingivalis* and an altered metabolism of triglycerides.

Table 13. Relationship of Positivity to *P. gingivalis* and triglycerides among hypertensive patients

Triglycerides	Positive n (%)	Negative n (%)	P value
Normal	5 (33.3)	10 (66.7)	
Elevated	26 (74.3)	9 (25.7)	
Total	**31 (62.0)**	**19 (38.0)**	**0.016**

When age groups were analyzed amongst hypertensive patients, an increasing trend of *P. gingivalis* positivity was observed with age-ranging from 33.3% in the 20–29-year age group to 81.3% in the 60–69-year age group. However, the association was not statistically significant (P = 0.119), which means that the observed age-related trend was not statistically confirmed in the present sample.

Table 14. Link between *P. gingivalis* positivity by age groups of hypertensive patients

Age group (years)	Positive n (%)	Negative n (%)	P value
20–29	2 (33.3)	4 (66.7)	
30–39	2 (40.0)	3 (60.0)	
40–49	4 (44.4)	5 (55.6)	
50–59	10 (71.4)	4 (28.6)	
60–69	13 (81.3)	3 (18.7)	
Total	**31 (62.0)**	**19 (38.0)**	**0.119**

Sex-based analysis within the hypertensive group showed that *P. gingivalis* was found in 64,3% of males and 50% of females. The difference was not statistically significant (P = 0.715), which means that sex was not a significant factor to states bacteriological positivity in this study.

Table 15. Association of *P. gingivalis* positivity to sex among hypertensive patients

Sex	Positive n (%)	Negative n (%)	P value
Male	27 (64.3)	15 (35.7)	
Female	4 (50.0)	4 (50.0)	
Total	**31 (62.0)**	**19 (38.0)**	**0.715**

Comparison of physiological variables based on bacteriological status for the hypertensive group revealed that *P. gingivalis*-positive patients had an unfavorable physiological profile when compared to negative patients. Mean systolic blood pressure, diastolic blood pressure, body mass index, total cholesterol, and triglycerides were found to be higher among *P. gingivalis*-positive patients and differences were found to be statistically significant. These findings indicate the potential of *P. gingivalis* positivity in association with an adverse cardiometabolic state in hypertensive individuals.

Table 16. Comparison between selected physiological variables based on *P gingivalis* status in hypertensive patients

Variable	*P. gingivalis* positive (n=31)	*P. gingivalis* negative (n=19)	P value
Systolic blood pressure (mmHg)	157.4 ± 10.8	149.1 ± 9.6	0.007
Diastolic blood pressure (mmHg)	97.6 ± 7.1	92.8 ± 6.4	0.018
Body mass index (kg/m²)	32.7 ± 3.8	29.4 ± 3.1	0.003
Total cholesterol (mg/dL)	229.5 ± 27.4	208.6 ± 24.9	0.011
Triglycerides (mg/dL)	198.2 ± 35.7	173.4 ± 31.6	0.014

Discussion

The present study showed an increased prevalence of hypertension among the investigated population among the older subjects and recent subjects with higher body mass index, additionally the bacteriological component revealed an increased frequency of *Porphyromonas gingivalis* derived from hypertensive patients as compared to normotensive controls. In addition, positivity to *P. gingivalis* was more common in hypertensive patients with obesity and dyslipidemic parameters, and bacteriologically positive patients had less favorable physiological values. This pattern was

supportive of the current oral-systemic concept, that oral dysbiosis may co-exist with, and possibly lead to, cardiovascular risk through inflammatory, endothelial and metabolic mechanisms [28,29]. The significant association between hypertension and increased body mass index in the current study is biologically plausible and consistent with the current knowledge of the mechanistic basis of obesity-related hypertension. Excess adiposity is associated with sympathetic nervous system overactivity, activation of the renin-angiotensin-aldosterone system, renal sodium retention, endothelial dysfunction and chronic low-grade inflammation, all of which perpetuate blood pressure elevation. Therefore, the predominance of obesity in hypeinrtensive patients in this work was consistent with the known pathophysiology of cardiovascular burden in obesity (Parvanova *et al*., 2024). Although high levels of total cholesterol and triglycerides were more prevalent in hypertensive patients than in the controls numerically, the differences between groups were not statistically significant in the current sample. This might be a reflection of the small sample and the fact that the lipid analysis was categorical. Neither less, recent population data demonstrate that the occurrence of dyslipidemia was extremely high among hypertensive adults and may be linked to poorer blood pressure was, which means the lack of statistical significance in the current study should be interpreted with caution. The lipid-related pattern seen in this study can also be considered in the context of the bidirectional connection between periodontitis and dyslipidemia that was getting recognized now. Recent evidence synthesis suggests that periodontal inflammation was potentially able to exacerbate lipid disturbance and that abnormal lipid metabolism can directly increase periodontal destruction. From this standpoint, the overlapping of hypertension, obesity and lipid abnormalities in the current study might have provided a biologically favorable background for an increased burden of periodontal pathogens, including *P. gingivalis*. One of the most significant results of the current study was the significantly higher frequency of the positivity of the bacterial species *P. gingivalis* among hypertensive patients. This observation was consistent with the recent clinical evidence that shows periodontal patients frequently have higher values of blood pressure as well as higher prevalence of hypertensive states. The current bacteriological result therefore supports the concept that there may be more to the oral-vascular relationship than just an overall periodontal status to consider with specific periodontal pathogens. The current findings were also in agreement with observational clinical findings of a relationship between hypertension and periodontitis when periodontal, physical and biochemical features were taken into account alongside one another. This was important because if this was the case, it suggests that the association might not be limited to discrete oral findings, but that this might represent part of a larger picture of a chronic inflammatory phenotype. Accordingly, higher detection of *P. gingivalis* in hypertensive persons in the current paper seems to be biologically and clinically consistent. At the same time, the current bacteriological results must not be taken as evidence of incidence. Recent systematic review evidence has reported a heterogeneity of some oral microbiome related hypotheses in hypertension, especially on nitrate-reducing bacteria and nitric oxide related pathways. This heterogeneity suggests that these were not all the same form of oral microbial signal being equally associated with prophet-offs, and gives us insight into the need to differentiate large-scale changes in the microbiome versus pathogen-specific associations such as what we projected here for *P gingivalis*. The inflammatory plausibility of the association noted in the current study was high. Hypertension was now well known as being an immune-inflammatory disease and mechanistic research in the area has led to discovering that immune mineralocorticoid receptor signaling of dendritic cells can be responsible for increasing blood pressure through Th17 related inflammatory pathways. Because *P. gingivalis* was a potent inflammatory pathogen that can support continued immune activation, the higher prevalence of *P. gingivalis* in hypertensive patients may be the result of immune pathway amplification of existing mechanisms linked to the pathogenesis of hypertension. A direct vascular role of *P. gingivalis* was also aided by recent endothelial studies. Outer membrane vesicles from *P. gingivalis* have been found to increase vascular permeability through the induction of stress fibers while degrading the vascular endothelial cadmium in endothelial cells. Such findings provide a mechanistic underpinning of how an oral pathogen may contribute to vascular integrity outside the periodontal compartment and so help to explain the biological significance of a greater bacteriological positivity in the hypertensive patient, offered in the present study. The higher rate of *P. gingivalis* positivity in obese hypertensive patients in the present study was also consistent with the current knowledge. Obesity and periodontitis have

similar pathophysiological characteristics, such as chronic low-grade inflammation, oxidative stress, cytokine imbalance, and microbial-host interaction. Consequently, the higher percentage of positivity of *P. gingivalis* in each of the categories of body mass index in the present work may indicate a common inflammatory and metabolic background of obesity, periodontal dysbiosis and hypertension. The link found between the positivity of *P. gingivalis* and lipid abnormalities in patients with hypertension was also supported by pathogen-specific cardiovascular research. Clinical evidence has shown that higher levels of oral abundance of *P. gingivalis* were found in very high-risk cardiovascular patients including atherosclerotic cardiovascular disease individuals and familial hypercholesterolemia-related risk profile. This was consistent with the interpretation that *P. gingivalis* was enriched in subjects that have less favorable vascular and metabolic phenotypes. Another result of present study was that *P. gingivalis*-positive hypertensive patients had higher mean systolic blood pressure, diastolic blood pressure, body mass index, total cholesterol and triglycerides than bacteriologically negative patients. This pattern was compatible with the recent experimental findings demonstrating that *P. gingivalis* was able to aggravate atherosclerotic plaque instability by promoting lipid-laden macrophages in their necroptosis. Such mechanisms may help to explain why bacteriological positivity in the present study grouped with a more adverse physiological profile. The unfavorable physiological profile of *P. gingivalis*-positive patients was further corroborated by the mechanistic vascular evidence that *P. gingivalis* has the potential to cause endothelial dysfunction by Sirt3-depending CypD acetylation. In that study, oral inoculation damaged endothelial-dependent vasodilation, reduced information of the aortic endothelium, and led to enhanced mitochondrial oxidative stress. These results strengthen the potential neuronal detection of hypertensive susceptible patients to bacteriological positivity. Very recent evidence has further demonstrated that *P. gingivalis* outer membrane vesicles can induce vascular endothelial glycocalyx injury through the PPAD/CitH3/B3GAT1 pathway. As glycocalyx integrity plays a critical role in endothelial homeostasis and vascular function, this mechanism offers another logical reason for these co-morbidities of periodontal pathogen burden and unfavorable cardiovascular physiology in the current study. The absence of a statistically significant relationship between *P. gingivalis* detection and sex in the current work can be attributed to the relatively small sample size of the study and the preponderance of males in both populations leading to reduced power to detect sex specific microbial differences. Similarly, although *P. gingivalis* positivity was greater in older than in younger people, this increased associative status was not statistically significant, suggesting that age may have an indirect effect on bacteriological burden through the cumulative effects of periodontal and cardiometabolic exposure and therefore may not be an independent predictor of it, support for an association between periodontal disease and cardiovascular disease, but further noted that additional high quality human studies are needed on the topic, present findings suggest comprehensive comorbidities of hypertension, obesity, and disordered lipid metabolism and oral pathobiont load may converge in a common inflammatory and endothelium correlate. From a clinical point of view, the identification of *P. gingivalis* in hypertensive patients may also give additional insight from a bacteriological point of view in patients with a less favourable physiological profile. From a research perspective, these results support the need for future larger research with the inclusion of quantitative bacterial load, periodontal severity indices, inflammatory biomarkers and multivariable models to ascertain whether *P. gingivalis* acts as a primarily associated marker, or as a contributing mediator, or both.

Conclusion

The results of the current study showed that hypertensive patients had a greater incidence of *Porphyromonas gingivalis* detection compared to the normotensive subjects, indicating a possible link between periodontal pathogen burden and hypertension. In addition to this, the hypertension of the studied population was strongly associated with old age and high body mass index levels while bacteriological positivity for *P. gingivalis* was higher among hypertensive patients with obesity and abnormal lipid parameters as well as the study showed that hypertensive patients who were positive for *P. gingivalis* had less favorable physiological characteristics, including elevated blood pressure values and increased body mass index and increased serum lipid number values when compared to bacteriologically negative patients. These findings suggest clustering of *P. gingivalis* positivity in hypertensive persons with more adverse cardiometabolic profile. The data supports the idea of an

oral systemic relationship in which infections of the periodontal system may coexist with disturbances of cardiovascular and metabolic disorders via common inflammatory and endothelial mechanisms. Although the present findings do not provide a result establishing direct causation, they provide biologically meaningful findings that *P. gingivalis* may represent a relevant bacteriological marker in hypertensive patients. Further large-scale studies are needed to confirm these results with the use of quantitative bacterial load, detailed periodontal indices, biomarkers of inflammatory and with the use of multivariable modeling to better define the clinical and pathogenic significance of *P. gingivalis* in hypertension. This study has various strengths. First, it combined bacteriological assessment and physiological profiling in a clinically relevant hypertensive population for the possible oral-systemic association between *Porphyromonas gingivalis* detection and cardiovascular-related variables. Second, the application of real-time polymerase chain reaction for the detection of *P. gingivalis* was a sensitive and specific bacteriological method for detection of the target pathogen in subgingival plaque samples. Third, the inclusion of a normotensive control group allowed the comparison in direct fashion of bacteriological frequency and physiological characteristics of hypertensive and non-hypertensive individuals.

Clinical Implications

The results of the current study indicate a possible importance of bacteriological detection of *Porphyromonas gingivalis* in identifying the hypertensive patients with a less favourable physiological profile, especially those with obesity and dyslipidemic alterations. From the clinical standpoint, this supports the idea of oral health assessment having a higher value in hypertensive persons, and even more so, in patients with overlapping cardiometabolic risk factors. The findings also continue to strengthen the importance of looking at the periodontal status and oral microbial burden that may be potential constituents of a larger cardiovascular risk context.

Recommendations

Routine periodontal evaluation may be considered in hypertensive patients especially among those who are obese or dyslipidemic. More large-number multicenter studies on the association between *P. gingivalis* and hypertension in various populations should be done to validate the findings. Future work should involve quantitative bacterial load analysis, detailed periodontal staging and grade, inflammatory biomarkers and markers of vascular function. Prospective and longitudinal studies are suggested to determine whether *P. gingivalis* is either a contributing mediator or a risk marker or both in hypertension-related cardiovascular burden. Interventional Studies Assessing the Impact of Periodontal Therapy on Bacteriological Load and Blood Pressure-Related Outcomes may be warranted.

Funding

This research did not receive any external funding.

Conflict of Interest

The authors state that they have no competing interests.

Novelty Statement

This study is novel in that it combines bacteriological detection of *Porphyromonas gingivalis* and physiological profiling in hypertensive patients in a case control design. Unlike studies restricted to information about the general periodontal status, in the present work a particular keystone periodontal pathogen is considered and the association of this pathogen with blood pressure-related and cardiometabolic variables is investigated. This approach may be one of contributing to a more pathogen-oriented understanding of the oral-systemic interface in hypertension.

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