

Does Periodontal Treatment Help in Arterial Hypertension Control? A Systematic Review of Literature

Neus Lanau¹ Javier Mareque-Bueno¹ Michel Zabalza²

¹Department of Oral Medicine and Public Health, Faculty of Dentistry, Universitat Internacional de Catalunya, Barcelona, Spain

²Department of Oral Medicine and Public Health, Faculty of Dentistry and Faculty of Medicine, Universitat Internacional de Catalunya, Barcelona, Spain

Address for correspondence Neus Lanau Solé, DDS, Department of Oral Medicine and Public Health, Faculty of Dentistry, Universitat Internacional de Catalunya, C/Josep Trueta 08195, Sant Cugat del Vallès, Barcelona, Spain (e-mail: neuslanau@uic.es).

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Abstract

Arterial hypertension and periodontal diseases are two of the pathologies with more prevalence worldwide. In the last few years, several scientific evidences have demonstrated the relationship between both diseases. Besides the etiopathogenic and causal relationship, some recent publications have pointed out that the therapeutic approach of periodontitis could have positive effects on the control of arterial hypertension. The aim of this systematic review is to determine whether there is a decrease in or better control of blood pressure after performing nonsurgical periodontal treatment in patients with periodontitis.

A thorough search in PubMed, Scopus, and ISI Web of Science databases with the keywords “‘periodontal disease’ OR ‘periodontitis’ OR ‘periodontal’ AND ‘blood pressure’ OR ‘hypertension’ OR ‘arterial hypertension’” was conducted. The quality of the reported information was assessed following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement for systematic reviews.

Eight articles were considered for this systematic review. Five of the studies showed statistically significant reduction in systolic blood pressure (SBP) values.

Despite the limitations of the review, nonsurgical treatment of periodontal disease seems to reduce SBP values. Further research with larger and longer-term clinical trials are needed to demonstrate this potential positive effect.

Keywords

- ▶ periodontitis
- ▶ hypertension
- ▶ blood pressure
- ▶ periodontal disease
- ▶ periodontal therapy

Introduction

Arterial hypertension is one of the most important and better studied cardiovascular risk factor and is associated with an increased risk of cardiovascular morbidity and mortality.¹

Arterial hypertension affects 30 to 45% of the adult population and is the leading cause of premature death in the world.² Its high prevalence is a global public health problem; it is estimated that by 2025 a third of the world's population could be hypertensive.²

In this scenario, the identification of hypertensive subjects and the control of their blood pressure (BP) measurements, to avoid cardiovascular events, is an unquestionable health goal.

The etiopathogenic mechanisms that trigger the onset of arterial hypertension are varied, multifactorial, and not fully known; they include hemodynamic mechanical changes and stiffness in the arterial wall, autonomic and neurohormonal dysregulations, endothelial dysfunction, oxidative stress, and local and systemic inflammation.³

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Periodontal diseases, gingivitis and periodontitis, are very prevalent multifactorial inflammatory pathologies caused by bacteria that affect periodontal tissues. The prevalence of periodontitis is estimated to be between 20 and 50% of the worldwide population.⁴ Among its etiopathogenic mechanisms there is a chronic inflammation response catalyzed by different mediators.^{5,6}

The etiopathogenic parallels and the high prevalence of both diseases in the same patients have raised the search for the relationship between both pathologies and the exclusion of confounding factors such as age, gender, smoking, educational level, socioeconomic status, obesity, and diabetes. In recent years, there have been numerous scientific evidences that support this relationship more solidly.^{5,7-9} Its relationship has also been taken into account in different scientific societies and has been mentioned in dental and medical guides that address the management of high BP.^{5,10}

Beyond the etiopathogenic and causal relationship, of great importance is the publication of some papers in which it is exposed that therapeutic approach of periodontal disease could have effects on the control of BP measurements.¹⁰ Although the evidence is very limited, it is a very important concept since many of the hypertensive patients are not treated or do not reach recommended BP goals, or are poorly controlled despite various medical and pharmacological treatments. Arterial hypertension is a modifiable risk factor, and any progress or improvement in its control affects individual cardiovascular risk.

These findings highlight the relevant role that dentists and dental treatments can play in the management of arterial hypertension either in the field of primary prevention through the identification of untreated patients or as secondary prevention, implementing oral treatments for the management and control of high BP.

The aim of this review is to determine if there is a decrease in or better control of BP after performing nonsurgical periodontal treatment in patients with periodontitis.

Materials and Methods

Primary Outcome

The primary outcome of this systematic review is to determine changes in BP measurements following periodontal therapy.

Inclusion/Exclusion Criteria

The eligibility criteria were defined according to PECO strategy. This acronym represents the patient (P), exposition (E), comparison (C), and outcome (O) characteristics of the eligible question.

The inclusion criteria considered original intervention articles, published in peer-reviewed journals, including all kinds of longitudinal studies (randomized clinical trials, cohort studies and case-control studies) that included adult patients with periodontal pathology (P), which evaluated exposed (E) and nonexposed patients to periodontal therapy (C), and assessed the association between changes in BP and periodontal therapy (O). The search strategy was limited

to articles published in English and studies conducted in humans. The exclusion criteria were case reports, reviews, descriptive studies, opinion articles, technical articles, and case series. Individuals under 16 years of age and pregnant women were also excluded.

Search Strategy and Study Selection

A comprehensive search of the PubMed, Scopus, and ISI Web of Science databases from its inception through November 2019 was conducted to identify studies that evaluated the relationship between arterial hypertension and periodontitis, and more specifically studies that evaluated the changes in BP after conducting nonsurgical periodontal treatment.

We queried MeSH terms and the article text for the following search terms: ('periodontal disease') OR ('periodontitis') OR ('periodontal') AND ('blood pressure') OR ('hypertension') OR ('arterial hypertension').

The articles resulting from this search were screened manually, first based on the title, then the abstract, and finally the complete manuscript, to determine their appropriateness for inclusion in the literature review. References cited in the included articles were also reviewed to identify additional published articles not identified by the database search.

Data Extraction

Selected publications were independently reviewed by two investigators (MZ and NL). The extracted data included information about the study design characteristics, group and patients' characteristics (periodontal status and BP assessment), the exact nonsurgical procedure, and the reported results. Disagreements between the authors were resolved through consensus. Quality assessment of all included articles was performed independently by a reviewer as part of the data extraction process.

The quality of the reported information included in each article was assessed following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement¹¹ for the improvement of the publication of systematic reviews (**–Supplementary Appendix A** [online only]).

Definitions of Periodontitis and Hypertension Diagnosis of Arterial Hypertension

Hypertension was defined as systolic blood pressure (SBP) ≥ 140 mm Hg and/or diastolic blood pressure (DBP) ≥ 90 mm Hg or the use of antihypertensive medications.²

Diagnosis of Periodontitis

Following the guidelines of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions,¹²⁻¹⁴ in the context of clinical care, a patient is a "periodontitis case" if:

- Interdental CAL (clinical attachment loss) is detectable at ≥ 2 nonadjacent teeth.
- Buccal or oral CAL ≥ 3 mm with pocketing ≥ 3 mm is detectable at ≥ 2 teeth but the observed CAL cannot be ascribed to nonperiodontitis-related causes.

Results

Studies Included

The study selection process for inclusion in the review is summarized in ►Fig. 1 (diagram flow). The database search strategy identified 395 potentially eligible references. After screening titles and abstracts, 52 full-text articles were reviewed in their entirety. Forty-four articles were excluded because no periodontal intervention was performed or were focused only on the metabolic syndrome. Three additional articles were included after review of references. Eventually eight studies were included in the literature research (►Table 1).

Description of the Study Characteristics

The eight studies included in this review had variable design and quality, and no meta-analysis was performed because of the heterogeneity of the identified studies. The population of the studies varied from 40 to 125 patients depending on the article. Moreover, the types of studies were different: five randomized clinical trials,¹⁵⁻¹⁹ one interventional prospective cohort study,¹⁰ one clinical intervention trial,²⁰ and one pilot intervention study.²¹ There were also different types of periodontal treatment compared. While some authors compared basic periodontal therapy (dental hygiene) and intensive periodontal treatment (scaling and root planning),¹⁹ some others compared intensive periodontal treatment with or without administration of antibiotics, both local antimicrobials¹⁵ and systemic.^{16,20} There was another group of studies that had an intervention group where periodontal intensive treatment was performed and a control group that received no treatment until the end of the study.^{17,18} Finally, there were two studies that only had one group of patients that received nonsurgical periodontal treatment, dental hygiene, or scaling and root planning according to need.^{10,21}

Discussion

The relationship between periodontal disease and arterial hypertension seems quite evident and there is significant

scientific evidence that points to this direction. However, the association between basic or intensive periodontal therapy and the improvement in BP levels is not entirely clear.

First, the studies that address this issue have different and varied results. The consistency and magnitude of the association is not clear in all of them because different results and conclusions arise; from the significant decrease in BP measurements after an intensive periodontal treatment to the invariability in BP levels. Moreover, the groups are not homogenous, the measurements of BP are different among studies, and the follow-up is diverse.

Three of the included studies compared two groups of patients depending on whether periodontal treatment was performed with or without antibiotic therapy,¹⁵⁻¹⁷ both systemic and locally delivered in periodontal pockets. The first one, by D'Aiuto et al,¹⁵ is a randomized clinical trial in which 40 patients with periodontal chronic disease, defined as 50% of dentition with periodontal probing pocket depths ≥ 4 mm and with radiographic documentation of alveolar bone loss, were distributed in two groups. The first one received standard periodontal therapy (scaling and root planning session) and the second one received the same therapy to which small doses of local antibiotic therapy (minocycline microspheres) were added. The follow-up was performed 1, 2, and 6 months after the intervention. A decrease in SBP was detected at 2 months in the antibiotic therapy group with a mean difference of 7 ± 3 mm Hg (95% confidence interval [CI]: 1–12; $p = 0.0211$). Moreover, this decrease was more important in smokers. However, the reduction was not maintained at 6 months nor did occur in DBP.

Bizzarro et al¹⁶ also performed a randomized controlled clinical trial comparing two groups of patients ($n = 110$). One group received basic periodontal therapy (scaling and root planning) and the second one received the same therapy with administration of systemic antibiotics (amoxicillin 375 mg and metronidazole 250 mg both thrice daily for 7 days). Periodontitis was defined as CAL of at least ≥ 3 mm in ≥ 2 nonadjacent teeth and patients were included if they had $\geq 30\%$ alveolar bone loss at ≥ 2 teeth per quadrant and presence of ≥ 2 teeth per quadrant with periodontal pockets ≥ 5 mm with at least ≥ 3 mm of CAL and at least 50% of all sites in the mouth with bleeding on probing. The follow-up was performed 3, 6, and 12 months from baseline. A decrease in SBP was observed in both groups 12 months after treatment ($p < 0.05$) but with no statistically significant difference between them (reduction of 2.7 mm Hg in control group and 5.4 mm Hg in the antibiotic group). No changes in DBP were observed.

In 2017, Jockel-Schneider et al²⁰ performed a clinical intervention trial with 55 patients also comparing periodontal treatment (scaling and root planning) with or without administration of systemic antibiotic (amoxicillin 500 mg and metronidazole 400 mg thrice daily for 7 days). The periodontal parameters stated to be elected were: appearance of periodontal pockets ≥ 6 mm at a minimum of four teeth. Follow-up was performed at 12 months. No statistically significant changes in SBP or DBP were observed in any group of patients.

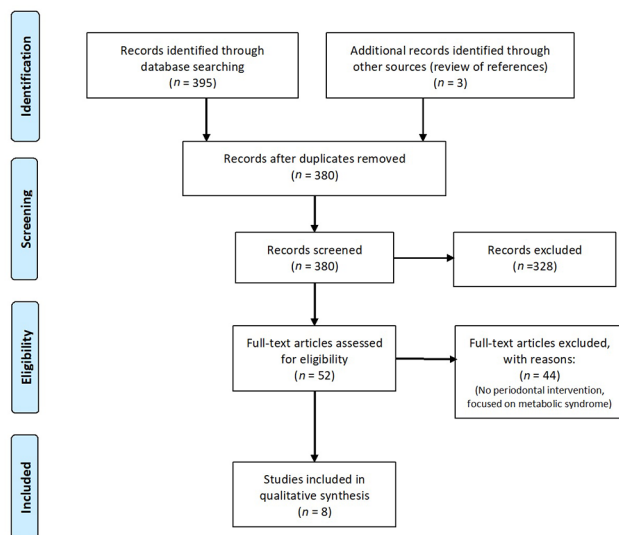


Fig. 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) diagram flow of the selection process.

Table 1 Results of literature research

Author, y, country	Type of study	n	Periodontal intervention	Follow-up	Reduction in BP	Results
D'Aiuto, 2006, UK ¹⁵	Randomized single-blind interventional trial	40	Group A: scaling and root planning; Group B: scaling and root planning + local antimicrobial	1, 2, and 6 mo	Reduction in SBP in Group B (7 ± 3 mm Hg) at 2 mo ($p = 0.0211$); not stable at 6 mo	Intensive periodontal treatment reduces systolic BP
Taylor et al, 2010, Australia ¹⁷	Randomized controlled prospective trial	125	Group A: intervention group (periodontal treatment); Group B: no treatment until end of study (3 mo)	3 mo	No changes in BP	BP levels did not change significantly in any of participants during study
Vidal et al, 2013, Brazil ¹⁰	Interventional prospective cohort study	26	Only one group, no control group; nonsurgical treatment (dental hygiene or scaling and root planning according to need)	3 and 6 mo	Reduction in SBP (12.5 mm Hg) and DBP (10.0 mm Hg) after 6 mo	Periodontal therapy significantly reduced levels of BP in refractory hypertensive patients
Hada et al, 2015, India ¹⁸	Randomized trial	55	Control group: no periodontal treatment; Experimental group: scaling and root planning	1, 3, and 6 mo	Reduction in SBP (7.1 mm Hg) in treatment group in 6 mo with no change in lifestyle ($p < 0.05$)	Scaling and root planning is effective in reducing significantly systolic BP
Houcken et al, 2016, Holland ²¹	Pilot intervention study	45	Only one group; nonsurgical periodontal treatment	3 and 6 mo	Decrease in SBP (2.9 mm Hg) after 6 mo ($p = 0.04$)	Peripheral systolic blood pressure significantly reduced after treatment
Bizarro et al, 2017, Holland ¹⁶	Randomized controlled clinical trial	110	Group A: basic periodontal therapy; Group B: basic periodontal therapy + antibiotics	3, 6, and 12 mo	Decrease in SBP of 2.7 mm Hg in Group A and 5.4 mm Hg in Group B at 12 mo ($p < 0.05$)	Systolic BP decreased in both groups with no statistically significant difference between them; no changes in DBP
Jockel-Schneider et al, 2018, Germany ²⁰	Clinical intervention trial	55	Group A: scaling and root planning + antibiotic; Group B: scaling and root planning	12 mo	No changes statistically significant from baseline to 12 mo	Peripheral blood systolic pressure was unchanged
Zhou et al, 2017, China ¹⁹	Randomized clinical trial	107	Group A: dental hygiene; Group B: scaling and root planning	1, 3, and 6 mo	Reduction in SBP (12.57 mmHg) and DBP (9.65 mm Hg) in Group B after 6 mo ($p < 0.05$)	Systolic BP and diastolic BP outcomes markedly reduced after treatment

Abbreviations: BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure.

There is another group of studies from the included ones that compare the BP levels between groups of patients with no periodontal treatment (or only a dental hygiene) and periodontal treatment (scaling and root planning).¹⁸⁻²⁰ The first one by Taylor et al¹⁷ is a randomized controlled prospective trial with 125 patients comparing no periodontal treatment with scaling and root planning. Periodontitis parameters for the inclusion were six or more sites with ≥ 5 mm periodontal probing depth and ≥ 2 mm attachment loss at teeth other than third molars. The duration of the study was 3 months and no changes in BP were observed. The second study by Hada et al¹⁸ was also a randomized clinical trial with 55 patients. The control group did not receive periodontal therapy and

the experimental group had two sessions of scaling and root planning. Periodontal parameters for the inclusion were: patients with at least 14 teeth (excluding third molars) and advanced chronic gingivitis and mild-to-moderate chronic periodontitis (at least four teeth with CAL ≤ 4 mm at a minimum of one site). Follow-up was performed 1, 3, and 6 months after the intervention. At 6 months, a statistically significant reduction of 7.1 mm Hg in SBP in the intervention group was observed ($p < 0.05$). The third study by Zhou et al¹⁹ is a randomized clinical trial with 107 patients that compared BP levels in two groups of patients: the first one received a dental hygiene and the second one received periodontal treatment (scaling and root planning). Patients

with moderate to severe periodontitis were included considered as the presence of at least two sites between adjacent teeth with ≥ 4 mm attachment level and at least two such sites with ≥ 5 mm pockets. Follow-up was performed 1, 3, and 6 months after the intervention. At 6 months, BP levels were markedly reduced in the treatment group. They observed an absolute difference of 12.57 mm Hg in SBP and 9.65 mm Hg in DBP (95% CI: 10.45–14.69 and 7.06–12.24; $p < 0.05$).

Finally, there are two studies of the included ones that only have one group, the treatment group with no control group.^{10,21} The first one, by Vidal et al.,¹⁰ is an interventional prospective cohort study of 26 patients that had periodontal treatment, a dental hygiene or scaling and root planning according to its need. The periodontal parameters in this study were the diagnosis of generalized advanced chronic periodontitis. Follow-up was done 3 and 6 months after the intervention. After 6 months, a significant reduction in SBP and DBP was observed, 12.5 mm Hg and 10.0 mm Hg, respectively. The last study included in the review was done by Houcken et al.,²¹ a pilot intervention study of 45 patients with only one treatment group (scaling and root planning was performed). The follow-up was done 6 months after the intervention, where a significant reduction in SBP was observed (from 119.8 ± 14.6 mm Hg to 116.9 ± 15.1 mm Hg; $p = 0.04$).

With all the studies reviewed we can conclude that periodontal treatment could have positive effects on BP measurements, specifically in SBP. Five of the studies showed statistically significant reduction in SBP values.^{15,17–19,21} However, while in some articles¹⁹ questions about changes in lifestyle or habits were asked in the follow-up visits, in other ones,¹⁶ confounders such as smoking habits, diet, or physical activity were not taken into account; or even smoking patients were not included in the research.¹⁸ Moreover, the periodontal parameters of inclusion and exclusion criteria were different in the studies, either because of the different definitions of periodontal diseases considered, or because in some studies the most severe forms of periodontitis were not included.¹⁸ Furthermore, different evaluation methods of hypertension were applied. From our point of view all these different criteria may have affected the results due to a potential bias. The lack of publications and clinical studies that address this association, their heterogeneity, the role of inflammation in this process as bias, the variability that can occur in BP measurements, and the different guidelines in the treatment of periodontal disease must be taken into account when analyzing these two diseases.

Our study has several limitations, which are also inherent to many systematic reviews. The retrospective nature of our review, incorporating data from published studies and not on individual patients, limits the availability of information on some issues, such as different clinical end points, comorbidities, or concomitant therapies. No meta-analysis was performed, which was probably because of the heterogeneity of the identified studies.

Finally, the results observed forces us to reflect and ask ourselves new questions and challenges, as whether

periodontal disease is a marker or mediator or some patients may have a genetic or metabolic susceptibility²² that might also play a part, as well as whether it affects only the SBP by some unknown mechanism or the periodontal treatment really has an initial effect on the BP but not maintained over time.

Conclusions

Hypertension is an important modifiable cardiovascular risk factor and therefore all measures aimed at identifying and controlling its development and progression are a global public health priority.

Although there are few publications that address this topic, nonsurgical treatment of periodontal disease could have a positive effect on the reduction of SBP values. To our knowledge this is the first systematic review that points in this direction.

Further research with larger and longer-term clinical trials are needed to demonstrate this association. Periodontitis could influence the prevalence of hypertension and periodontal interventions could play an important role in the prevention of cardiovascular diseases.

Authors' Contributions

All persons who meet authorship criteria are listed as authors, and all authors certify that they have participated in all the parts of the realization of the manuscript.

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Conflict of Interest

None declared.

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