Periodontal conditions of essential hypertension attendees to a general hospital in Hong Kong

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ABSTRACT

Background: Periodontal infection could induce systemic vascular/endothelial dysfunction signifying a potential link between hypertension and periodontitis.

Methods: Convenience sample of 204 adults attending a university teaching hospital without (C: control) or with essential hypertension [EH; n = 102, duration (mean ± SD) 11.4 ± 6.9 years] were surveyed in this cross-sectional study. Patients with concomitant systemic conditions were excluded. Dental history, oral hygiene habits, and blood pressure were recorded. Plaque score (Pl%), bleeding on probing (BOP%), probing pocket depth (PPD) and probing attachment level (PAL) were noted and periodontitis severity was determined according to AAP/CDC case definition guidelines.

Results: Both groups exhibited poor oral hygiene. EH group had higher mean full-mouth PAL/PPD (3.16/2.73 vs 2.51/2.40, $P < 0.001$). 51.0%/30.4% of the EH/C participants had severe periodontitis ($P < 0.001$). Regression analysis indicated systolic blood pressure, age, smoking and BOP% were associated with more severe periodontitis ($r^2 = 0.207$, $P < 0.05$) while BOP%, PAL and fewer missing teeth were associated with worse mean PPD ($r^2 = 0.612$, $P < 0.05$).

Conclusions: Within the limitations of this study, majority of the hospital attendees surveyed exhibited poor plaque control, while periodontitis severity was found to be associated with EH, and smoking.

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Keywords: age factors; essential hypertension; periodontal disease; risk assessment; smoking

Abbreviations and acronyms: AAP = American Academy of Periodontology; AHA = American Heart Association; BOP = bleeding on probing; BP = blood pressure; CDC = Centers

for Disease Control and Prevention; CEJ = cementoenamel junction; CPI = Community Periodontal Index; CPITN = Community Periodontal Index of Treatment Needs; CVD = cardiovascular disease; DBP = diastolic BP; DM = diabetes mellitus; EH = essential hypertension; FGM = free gingival margin; GR = gingival recession; KNHANES = Korean National Health and Nutrition Examination Surveys; PAL = probing attachment level; Pl = plaque; PPD = probing pocket depth; RR = relative risk; SBP = systolic BP; STROBES = Strengthening the Reporting of Observational Studies in Epidemiology

INTRODUCTION

Hypertension is defined as a persistently elevated blood pressure (BP) beyond an arbitrarily set normal level.[1] The primary cause of hypertension is physical inactivity.[2] After aerobic or progressive resistance exercise regimes, reduced systolic BP (SBP) and diastolic BP (DBP) in human adults could be observed[3] proposed to be mediated by renin-angiotensin system and sympathetic nervous system modulation.[4] Hypertension is considered one of the major modifiable risk factors for cardiovascular disease (CVD) such as coronary heart disease and congestive heart failure, peripheral (thrombotic, embolic, aneurismal, or haemorrhagic) vascular disease, stroke and renal failure,[5] with relative risk (RR) for myocardial infarction, stroke, and cardiovascular death at 1.78 - 2.19.[6,7] Together with borderline isolated systolic hypertension, hypertension is also associated with significantly increased risk for all-cause mortality: 41% for hypertension, 22% for isolated systolic hypertension.[6] Centers for Disease Control and Prevention (CDC) reported diseases of the heart, CVD, and essential hypertension (EH) as among the top ten leading causes of death in 2016 for Asian and non-Hispanic individuals in the United States of America.[8]

In Hong Kong, more than one-tenth of the adult population has hypertension.[9] The prevalence rate was said to be between 17 to 23% in 2000-2004.[10] Globally, a quarter of the adult population (over 1 billion) suffers from hypertension the same as for Australians.[11] Hypertension worldwide prevalence in adults has been predicted to increase to 29% by 2025.[12] In general, the prevalence rate increases with age.[13] Despite the large number of diagnosed cases of hypertension, only a proportion of these receive treatment; and only less than 10% of men and 7% of women achieved controlled blood pressure in the normal range (<120/80 mmHg) through anti-hypertensive treatment. The 36% men and 30% women who achieved systolic BP (SBP) of 120–139 mmHg and diastolic BP (DBP) of 80–89 mmHg through anti-hypertensive treatment, considered as controlled before 2017, are now considered as having

elevated BP or stage 1 hypertension.[10,14,15]

Periodontitis is a complex human disease having plaque as its causative agent, however periodontal infection is modifiable by a multitude of factors: smoking, age, gender, stress, diabetes mellitus (DM), while also being linked to hypertension, cardiovascular disease,[16] osteoporosis,[17] and dyslipidemia.[18] Systemic spillage of inflammatory mediators from chronic periodontitis is postulated to jeopardise BP control and hence impair cardiovascular health,[19] and periodontitis and hypertension share common risk factors: age, smoking, stress and low socio-economic status.[20,21]

Various studies over past few decades suggested that hypertension may be associated with periodontitis.[20,22-29] Patients with hypertension or higher SBP were found to have significantly higher Community Periodontal Index of Treatment Needs (CPITN) scores,[24] more gingival bleeding,[29] and fewer remaining teeth.[26] A 10% increase in bleeding on probing (BOP) was found to increase the average SBP by 0.5 mmHg thereby suggesting that BOP may associate with increasing odds of hypertension in US adult population.[29] Microbiologically, patients with high levels of putative periodontopathogens such as *Aggregatibacter Actinomycetemcomitans*, *Porphyromonas gingivalis, Tanerella forsythia* or *Treponema denticola*, were found to have increased SBP by 9 mmHg, and DBP by 5 mmHg with an odds ratio of 3.13 for hypertension.[28] A study on spontaneously hypertensive male rats showed an increased amount of attachment and bone loss compared to normotensive rats after ligature-induced periodontitis[30] indicating that such a negative association between systemic and oral health perhaps might be biologically plausible.

Pathophysiological evidence linking hypertension and periodontitis remained limited until recently when Czesnikiewicz-Guzik and colleagues[31] suggested interactions between periodontal infection and vascular/endothelial dysfunction, which perhaps is potentiated via nitric oxide, superoxide, reactive oxygen species and adverse effects on T-helper cells

function.[32] In light of the above we undertook a cross-sectional study aimed at characterising periodontal conditions of Hong Kong adult university hospital attendees who either had or who had not EH, conforming to the EH categorisations given in the 2017 revised guidelines for the prevention, detection, evaluation, and management of high BP in adults.[14] Factors associated with periodontal health were studied. We hypothesized that Chinese adults with EH would have poorer periodontal status.

METHODS

Study design

The study design was a convenient sample, cross-sectional study carried out according to the STROBES (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines.

Research ethics approval and consent

This study was approved by the Institutional Review Board of The University of Hong Kong (UW 09-203). All subjects were given verbal explanation regarding the investigation upon first contact. One week later, written informed consent was obtained from all those who agreed to participate. Personal identifiers were removed from all collected data.

Study participants

A total of 1,240 patient records were manually screened from November 2010 to July 2011 to identify patients with EH prior to commencement of each consultation session at the Hypertension Clinic of the university teaching hospital over a nine-month period. Patients were excluded if they had concomitant medical conditions or history of CVD - including stroke, coronary artery disease, valvular heart diseases, cardiomyopathy, heart failure and cardiac

arrhythmias - neoplasia, or DM. Patients who had taken antibiotics, anti-inflammatory or immunosuppressant agents within the previous three months or who had undergone periodontal treatment in the previous six months were also excluded from the study. A total of 476 patients satisfied the above criteria and were invited by co-author CWSC to take part and 102 consented to join (EH group). The recruitment of control participants soon followed targeting age-matched hospital attendees who were free from EH and any of the medical conditions, drug history or dental treatment history mentioned above. These control subjects were patients attending the Department of Orthopedics and Traumatology between the months of April-July 2011 and were invited to join by co-authors CHB and DS.

The sample size was determined according to an earlier study of this group or with reference to previously published survey similar to the present study.[24,33] Briefly, at least 100 participants each in case and control groups were needed.

BP Measurement

SBP, and DBP readings were measured twice with a 10-minute interval from the supported left arm of the rested and seated patient (first measurement after >10 min of rest) using Omron Automatic Blood Pressure Monitor (Model HEM-7200) (Omron Healthcare Co. Ltd., Kyoto, Japan) with an appropriately-sized arm cuff (22-32 cm or 32-42 cm sized cuff) prior to and after the research team-administered questionnaire had been completed. This was done in accordance with the guidelines set by the American Heart Association (AHA).[1] Mean SBP and DBP were the average of the two separate measurements recorded.[34]

Questionnaire

Prior to oral examination, dental history was recorded using a questionnaire tool, including history of dental visits, periodontal treatment, tooth loss, oral hygiene habits and smoking

history.

Periodontal examination

Periodontal examination was carried out by calibrated examiners JJNC and YZ at the Periodontology Clinic, Faculty of Dentistry, the University of Hong Kong. Examination was repeated in every tenth participant to assess inter-examiner reliability.

Clinical parameters were recorded at six sites per tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual) excluding third molars, using a periodontal probe (PCP-UNC 15, HuFriedy Manufacturing Co., Chicago, IL, USA). The presence or absence of supragingival plaque (Pl) was recorded. Probing pocket depth (PPD) was measured from the free gingival margin (FGM) to the base of the probing sulcus or pocket. Gingival recession (GR) was measured from the cementoenamel junction (CEJ) to the FGM and was recorded as an integer: positive value if FGM was apical to the CEJ, a negative value if it was coronal to it. Probing attachment level (PAL) was calculated by adding PPD and GR.[35] BOP was recorded as positive if bleeding occurred within 10-15 seconds after probing. The BOP% (percentage of sites with BOP) was calculated for each patient. Presence and absence of furcation involvement, mobility and the number of missing teeth (except third molars) were also recorded.

Data analysis

The data collected were entered into a computer using Microsoft Office Excel software. Each entry was cross-checked, then analysed using the statistical software package IBM SPSS Statistics v25 (IBM headquarters, New York United States). Participants were classified into five BP categories (Table S1) based on SBP and DBP recorded on the day of examination.[14] For BP control, EH participants were defined as having optimal control if SBP < 130 mmHg

and DBP < 80 mmHg.[14]

To assess gum health or severity of periodontal disease, the periodontitis case definition guideline set by the working group appointed by the American Academy of Periodontology (AAP) and the CDC for population-based studies was adopted.[36] Participants were classified into four categories (Table S2) based on PAL and PPD and the number of interproximal sites affected.[36]

The outcome variables (continuous variables) were tested for normality of distribution prior to further statistical analysis. Standard descriptive statistics (frequency, mean, standard deviation) were used to describe the patients' demographic characteristics, pattern of dental visits, oral hygiene practices, smoking history, BP readings, and oral health status. The mean values of the clinical parameters (PPD, PAL, BOP% and Pl%) were calculated for each participant. Chi-square test was used to compare the proportions of participants belonging to various independent variable subcategories between EH or control group. Univariate analysis using 2-sample t-test was used to compare means between two groups. Linear regression analyses were used to determine the factors that could explain the increased risk of having severe periodontal disease, PPD, PAL, higher SBP, higher DBP and missing teeth. The level of statistical significance was set at 0.05.

RESULTS

Participants' characteristics

A total of 102 EH (55 females, 52.4 ± 10.8 years-old) and 102 controls (56 females, 52.4 ± 10.2 years-old) participated in the study. Their smoking and tooth brushing habits are presented in Table 1. Less than 20% participants were current smokers. Majority of the participants reported twice-daily tooth brushing, but control participants self-reported better interdental

cleaning habits. Despite the majority of both groups reporting non-ideal dental attendance (>1 year), the reported dental attendance behaviour of the control group participants showed more of these to have greater time elapsed since the most recent dental visit (Table 1).

Medical history

According to their medical records, the EH participants were diagnosed to have suffered from the condition for 11.4 ± 6.9 years (range: 1 - 31 years). 78.3% had EH for >5 years and 79.2% had a family history of hypertension. More than two-thirds of the EH participants (68.9%) were taking a combination of drugs daily. The most common medications being taken by the EH group were calcium channel blockers (64.7%), beta-blockers (47.1%), angiotensin-converting enzyme inhibitors (42.2%), diuretics (18.6%), angiotensin II receptor antagonists (10.8%), statins (8.8%), and alpha-blockers (6.9%) making a total of 2.0%/28.4%/40.2%/27.5%/2.0% taking 0/1/2/3/4 kinds of medications. Despite this, only 4% or 9% of treated EH participants could achieve normal or elevated BP, respectively. Thirty-two percent of control participants had an elevated BP on the day of examination (Table 1).

Concerning the 66 EH participants who received calcium channel blocker, one was diagnosed with drug-induced gingival enlargement. The remaining EH patients and all control group participants had no clinical gingival enlargement detectable at examination.

Dental/periodontal conditions

The dental and periodontal status of the EH and control participants were as shown in Table 1. Results from duplicate examinations on 20 participants showed that inter-examiner reliability on periodontal status (PPD in mm or PAL in mm) were almost perfect or substantial (weighted Kappa = 0.82 or 0.79, respectively).

Percentage of BOP and missing teeth count were not significantly different between EH and control groups (Table 1). EH group, despite a lower mean full mouth plaque of 59%, showed higher full mouth mean PPD and PAL, with more subjects displaying moderate or severe periodontitis categories (Table 1).

Associations between study parameters

The potential associations between various parameters collected/recorded were investigated and are summarized in the Supplementary Information section.

Regression analyses

Linear regression analyses identified factors that might associate with AAP/CDC periodontal disease definition (Table 2).[36] More severe forms of periodontitis appeared to be associated with SBP, age, smoking, higher BOP%, and lower Pl% (Table 2). When hypertension case definition was used instead of S/DBP measurements in the regression analysis, more severe periodontitis category appeared to be associated with BP category, age and BOP% ($r^2 = 0.184$).

Full-mouth mean PPD appeared to be associated with BOP%, full-mouth mean PAL and fewer missing teeth (Table 3), similarly observed when hypertension case definition instead of S/DBP was used in the regression analysis. In addition to the three independent variables, Pl% appeared also associated with increased full-mouth PPD ($r^2 = 0.620$). Full-mouth mean PAL appeared to be associated with SBP, age, smoking, mean full-mouth PPD, missing teeth and lower Pl% (Table 4). With hypertension category replacing SBP/DBP as the independent variable, similar significant association, including the BP category, were observed in relation to full-mouth mean PAL ($r^2 = 0.720$).

Number of missing teeth was associated with age, Pl%, mean PAL, lower DBP and less mean PPD (Table 5). Hypertension category as an independent variable, even for less severe

BP categories was similarly significantly associated with missing teeth ($r^2 = 0.405$).

DISCUSSION

This study conveniently sampled participants who attended the Hypertension Clinic at a university teaching hospital. This Specialty Clinic attends to referral cases mostly from private medical practitioners or from other hospitals or clinics under the Hospital Authority or the Department of Health of the Hong Kong Special Administrative Region Government. Predominantly, the referred EH cases were patients with uncontrollable BP or with hypertension of unknown cause. The current report is unique in the sense that EH patients – non-secondary hypertension patients without any concurrent organ or organ-system problems – only were recruited. Despite the regular check-ups (2-3 times per year) by medical specialists, the majority (87.3%) of the EH participants followed in this study had suboptimal BP control (Table 1), which is in line with previous reports.[10,14]

The participants in this study had poor oral hygiene (mean Pl% ≥59%), with a considerable proportion having moderate or severe periodontitis (EH, 97%), a prevalence much higher than a cohort of Australian Cardiovascular Unit attendees (21%).[37] A report analysing results from the 2008-2010 Korean National Health and Nutrition Examination Surveys (KNHANES) showed that individuals reporting higher frequency of tooth brushing or using more secondary oral care products had lower SBP and hypertension prevalence even adjusting for periodontitis experience.[38] The Korean findings reflect direct or indirect positive impact of good oral health practices upon BP. In the current EH cohort, self-reported poor usage of floss/interdental brush was found to be associated with more severe AAP/CDC periodontitis case (Supplementary Information).

Using the AAP/CDC periodontitis case definition, the present study showed that half (51.0%) and almost a third (30.4%) of EH patients and controls, respectively, had severe periodontitis (Table 1), similar to attendees for episodic diseases with mild symptoms at another general hospital in Hong Kong.[33]

EH group despite having 'lower' Pl%, exhibited higher mean PPD, mean PAL, and periodontitis defined according to AAP/CDC (Table 1), which substantiates the current research hypothesis. The regression analysis indicated severe periodontitis to be significantly associated with smoking, age, BOP%, lower Pl% and SBP confirms the impact of EH on poor periodontal health (Table 2). The association of EH or SBP and periodontal disease experience is in line with the findings from recent meta-analyses[32,39] which included retrospective population data analysis,[29,40,41] and cross-sectional studies.[26,27] Other independent variables significantly associated with periodontitis in the current study: smoking, older age and higher full-mouth BOP%, are well established periodontal risks.[35,42]

High full-mouth mean PPD was observed associated with higher BOP%, higher mean full mouth PAL and fewer missing teeth (Table 3). The relationship between BOP%, increased PPD and PAL is well established.[43] A Swedish study surveyed 282 (141 with hypertension, defined as diastolic pressure >90 mmHg) out of 1,239 attendees to a dental clinic for annual check-up, and reported percentage PPD ≥ 5mm to be higher in individuals with hypertension after adjustment for sex, age, smoking and number of teeth.[44]

Full-mouth mean PAL was identified to be associated with SBP, age, smoking, lower Pl%, higher mean PPD and more missing teeth (Table 4). The relation between attachment loss, age and smoking is well established[45] while the present investigation highlights an association between SBP and attachment loss, being among the first reports regarding full-mouth mean PAL and its correlation with BP, oral hygiene habits and other oral findings.

More missing teeth among the current study participants was associated with age, Pl%,

mean PAL, lower mean PPD as well as lower DBP (Table 5). The relationship between number of standing/missing teeth and hypertension remains unclear. In a group of 98 postmenopausal Japanese women, DBP was associated with tooth loss.[46] A German population-based study (n = 4,185), showed an inverse association between number of teeth and SBP in men but not in women.[47] In 3,552 hypertension and/or myocardial infarction cases and 902 controls, the number of standing teeth among the Swedish surveyed was not associated with hypertension.[26] An urban population-based study at Florianopolis, Brazil, studied 1,720 20-59 years-old individuals, 56% females, for tooth loss, socioeconomic, habits and BP.[48] Increased SBP was associated with edentulism but not in the other categories of tooth loss. A retrospective population based analysis of the KNHANES data indicated missing teeth might be independently associated with hypertension.[49] A recent Shanghai, China population-based study (n = 3,677) reported ≥15 teeth lost was associated with severe hypertension.[50] Poor self-reported oral hygiene practise and age have been shown to be associated with tooth loss in treated periodontitis patients responsible for own supportive periodontal care arrangements[51] in Hong Kong, and individuals with high periodontal disease susceptibility/PAL had greater tooth loss.[52]

Several other studies proposed association between periodontitis, tooth loss and hypertension, or sequelae of hypertension, but many were sub-analyses of the data collected.[26,28,29,53] For example, the Harvard School Health Professionals Follow-up Study[54] indicated that loss of ≥18 teeth associated with increased risk (RR = 1.67) of coronary heart disease, primarily among those with history of periodontitis.[55]

Many of the reports available to date on hypertension and periodontitis used the Community Periodontal Index (CPI) or an earlier version,[21,24,27,40] number of periodontal pockets ≥ 5mm,[44] or periodontal severity index[26,53] as outcome measures for assessing periodontal disease. Others also used probing depths,[25,29] subgingival plaque samples,[28]

gingival bleeding,[29] radiographic bone loss and/or CAL and tooth loss.[26,46-48,53] Limited studies, such as the present one, are available examining the full-mouth periodontal condition of patients with EH. Unless inflation factors were adjusted, partial periodontal recording, e.g. the CPI approach, could potentially induce bias of the parameters measured.[56] Caution therefore, should be exercised in interpreting reports reporting partial periodontal data.

Optimal adult BP level was set by American College of Cardiology/AHA[14] (Table 1) based on the fact that increased CVD risk could be observed beyond such limits. The relative CVD risk may start to increase beyond BP as low as 115/75 mm Hg and this level of BP may begin to cause benign organ injury.[57,58] If BP could successfully be controlled e.g. via exercise, medication(s) or a combination, a 5 mmHg SBP reduction was estimated to lead to 14%, 9%, or 7% overall reduction in mortality rate due to stroke, coronary heart disease or all-cause.[59,60]

Czesnikiewicz-Guzik and coworkers[31] reported that intensive periodontal treatment - consisting of one stage full-mouth debridement plus 0.2% chlorhexidine gel application - for genetically susceptible hypertensive patients with moderate/severe periodontitis, improved S/DBP and endothelial function, highlighting the importance of appreciating a relationship between hypertension and periodontitis as shown in the present study, and raising the prospect that proper management of the periodontal disease could confer appropriate systemic health benefits. A 6-month clinical trial reported endothelial function improvement alongside better periodontal health after one stage full-mouth debridement plus local delivery of minocycline microspheres in severe periodontitis patients who had BP of approx.125/80 mmHg.[61]

Periodontal disease and EH are relatively silent entities with potentially profound harmful effects to humans especially if left untreated.[62] The detriment brought by the diseases, in isolation or combined, on an affected individual cannot be ignored. Hypertension is an important risk factor for CVD, one of the leading causes of mortality among adults. EH, although relatively rare among young adults before 20-25 years, may commence as a benign

condition, and could gradually cause target-organ damage over period of 10-20 years.[63] Insufficient attention to gum health and periodontal disease prevention and commencing smoking at or before 30-years of age might allow periodontitis initiation and progression.[64] Prevention of EH, a chronic systemic condition, and periodontitis could and should be administered via a collaborative common risk factor approach.[65]

This study indicates that the hospital EH attendees surveyed had more severe periodontal disease status than non-EH surgical clinic attendees, despite both groups having poor oral health and overt gum inflammation. The study was conducted applying recognized case definition for periodontitis and hypertension as recommended by Aguilera and coworkers.[32] A later community-based study in Hong Kong with participants of similar age range, found a high prevalence of hypertension at 48%.[66] Obviously oral health care providers should be aware of the prevalence of hypertension in populations they serve.

The convenient sampling method adopted in this study and the sample size suggest that the results observed may be not truly representative for Hong Kong or any other population, however, this study was conducted among one of the referring centres for hypertension in Hong Kong, and the research team was therefore able to select EH participants without confounding diseases and to match medical healthy hospital attendees as controls. Within the limitations of this study, smoking, higher BOP% and EH status were shown to be associated with periodontal disease among the participants.

CONCLUSION

The present cross-section study showed that those attending a university teaching hospital in Hong Kong exhibited in general poor plaque control, while those with EH had poorer periodontal conditions. Future studies involving large scale, prospective designs and

intervention studies are indicated to further investigate the relationships between oral health and the health of the circulatory system. Nonetheless, physicians in public health care settings and dentists should pay attention to preventing periodontitis, and recognize the importance of promoting, establishing and maintaining periodontal health in patients with EH.

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DISCLOSURE

All authors declare no conflict of interest associated with this publication.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of this article:

Supplementary information.

REFERENCES

1. Perloff D, Grim C, Flack J, Frohlich ED, Hill M, McDonald M, Morgenstern BZ. Human blood pressure determination by sphygmomanometry. Circulation 1993;88:2460-2470.
2. Booth FW, Roberts CK, Laye MJ. Lack of exercise is a major cause of chronic diseases. Compr Physiol 2012;2:1143-1211.
3. 2018 Physical Activity Guidelines Advisory Committee. 2018 Physical Activity Guidelines Advisory Committee Scientific Report. Washington, DC: U.S. Department of Health and Human Services, 2018. URL: 'https://health.gov/sites/default/files/2019-09/PAG_Advisory_Committee_Report.pdf'. Accessed April 2020.
4. Eicher JD, Maresh CM, Tsongalis GJ, Thompson PD, Pescatello LS. The additive blood pressure lowering effects of exercise intensity on post-exercise hypotension. Am Heart J 2010;160:513-520.
5. Stefanadi E, Tousoulis D, Androulakis ES, Papageorgiou N, Charakida M, Siasos G, Tsioufis C, Stefanadis C. Inflammatory markers in essential hypertension: potential clinical implications. Curr Vasc Pharmacol 2010;8:509-516.
6. O'Donnell CJ, Ridker PM, Glynn RJ, Berger K, Ajani U, Manson JE, Hennekens CH. Hypertension and borderline isolated systolic hypertension increase risks of cardiovascular disease and mortality in male physicians. Circulation 1997;95:1132-1137.
7. Gu Q, Burt VL, Paulose-Ram R, Yoon S, Gillum RF. High blood pressure and cardiovascular disease mortality risk among U.S. adults: The third national health and nutrition examination survey mortality follow-up study. Ann Epidemiol 2008;18:302-309; Erratum in: Ann Epidemiol 2008;18:515.
8. Heron M. Deaths: Leading causes for 2016. Natl Vital Stat Rep 2018;67(6):1-76.
9. Janus ED. Epidemiology of cardiovascular risk factors in Hong Kong. Clin Exp Pharmacol Physiol 1997;24:987-988.

10. Cheung BM, Wong YL, Lau CP. Queen Mary Utilization of Antihypertensive Drugs Study: use of antihypertensive drug classes in the hypertension clinic 1996-2004. Br J Clin Pharmacol 2005;60:90-97.

11. Peterson KL, Jacobs JP, Allender S, Alston LV, Nichols M. Characterising the extent of misreporting of high blood pressure, high cholesterol, and diabetes using the Australian Health Survey. BMC Public Health 2016;16:695.

12. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. Lancet 2005;365:217-223.

13. Singh M, Mensah GA, Bakris G. Pathogenesis and clinical physiology of hypertension. *Cardiol Clin* 2010;28:545-559.

14. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Himmelfarb CD, DePalma SM, Gidding S, Jamerson KA, Jones DW, MacLaughlin EJ, Muntner P, Ovbiagele B, Smith SC Jr, Spencer CC, Stafford RS, Taler SJ, Thomas RJ, Williams KA Sr, Williamson JD, Wright JT Jr. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. J Am Coll Cardiol 2018;71(19): e127-e248; Erratum in: J Am Coll Cardiol 2018;71(19):2275-2279.

15. Cheung BM, Lo JL, Fong DY, Chan MY, Wong SH, Wong VC, Lam KS, Lau CP, Karlberg JP. Randomised controlled trial of qigong in the treatment of mild essential hypertension. J Hum Hypertens 2005;19:697-704.

16. Seymour RA, Preshaw PM, Thomason JM, Ellis JS, Steele JG. Cardiovascular diseases and periodontology. J Clin Periodontol 2003;30:279-292.

17. Tezal M, Wactawski-Wende J, Grossi SG, Ho AW, Dunford R, Genco RJ.The relationship between bone mineral density and periodontitis in postmenopausal women. J Periodontol

2000;71:1492-1498.

18. Nibali L, D'Aiuto F, Griffiths G, Patel K, Suvan J, Tonetti MS. Severe periodontitis is associated with systemic inflammation and a dysmetabolic status: A case-control study. J Clin Periodontol 2007;34:931-937.

19. Sanz M, D'Aiuto F, Deanfield J, Fernandez-Avilés F. European workshop in periodontal health and cardiovascular disease—scientific evidence on the association between periodontal and cardiovascular diseases: a review of the literature. Eur Heart J Suppl 2010;12:B3-B12.

20. Hujoel PP, Drangsholt M, Spiekerman C, DeRouen TA. Periodontal disease and coronary heart disease risk. JAMA 2000;284:1406-1410.

21. Inoue K, Kobayashi Y, Hanamura H, Toyokawa S. Association of periodontitis with increased white blood cell count and blood pressure. Blood Press 2005;14:53-58.

22. Howell TH, Ridker PM, Ajani UA, Hennekens CH, Christen WG. Periodontal disease and risk of subsequent cardiovascular disease in U.S. male physicians. J Am Coll Cardiol 2001;37:445-450.

23. Amar S, Gokce N, Morgan S, Loukideli M, Van Dyke TE, Vita JA. (2003) Periodontal disease is associated with brachial artery endothelial dysfunction and systemic inflammation. Arterioscler Thromb Vasc Biol 2003;23:1245-1249.

24. Angeli F, Verdecchia P, Pellegrino C, Pellegrino RG, Pellegrino G, Prosciutti L, Giannoni C, Cianetti S, Bentivoglio, M. Association between periodontal disease and left ventricle mass in essential hypertension. Hypertension 2003;41:488-492.

25. D'Aiuto F, Parkar M, Nibali L, Suvan J, Lessem J, Tonetti MS. Periodontal infections cause changes in traditional and novel cardiovascular risk factors: results from a randomized controlled clinical trial. Am Heart J 2006;151:977-984.

26. Holmlund A, Holm G, Lind L. Severity of periodontal disease and number of remaining

teeth are related to the prevalence of myocardial infarction and hypertension in a study based on 4,254 subjects. J Periodontol 2006;77:1173-1178.

27. Franek E, Klamczynska E, Ganowicz E, Blach A, Budlewski T, Gorska R. (2009) Association of chronic periodontitis with left ventricular mass and central blood pressure in treated patients with essential hypertension. Am J Hypertens 2009;22:203-207.

28. Desvarieux M, Demmer RT, Jacobs DR Jr, Rundek T, Boden-Albala B, Sacco RL, Papapanou PN. (2010) Periodontal bacteria and hypertension: the oral infections and vascular disease epidemiology study (INVEST). J Hypertens 2010;28:1413-1421.

29. Tsakos G, Sabbah W, Hingorani AD, Netuveli G, Donos N, Watt RG, D'Aiuto F. (2010) Is periodontal inflammation associated with raised blood pressure? Evidence from a national US survey. *J Hypertens* 2010;28:2386-2393.

30. Leite CL, Redins CA, Vasquez EC, Meyrelles SS. Experimental-induced periodontitis is exacerbated in spontaneously hypertensive rats. Clin Exp Hypertens 2005;27:523-531.

31. Czesnikiewicz-Guzik M, Osmenda G, Siedlinski M, Nosalski R, Pelka P, Nowakowski D, Wilk G, Mikolajczyk TP, Schramm-Luc A, Furtak A, Matusik P, Koziol J, Drozdz M, Munoz-Aguilera E, Tomaszewski M, Evangelou E, Caulfield M, Grodzicki T, D'Aiuto F, Guzik TJ. Causal association between periodontitis and hypertension: evidence from Mendelian randomization and a randomized controlled trial of non-surgical periodontal therapy. Eur Heart J 2019;40:3459-3470.

32. Aguilera EM, Suvan J, Buti J, Czesnikiewicz-Guzik M, Barbosa Ribeiro A, Orlandi M, Guzik TJ, Hingorani AD, Nart J, D'Aiuto F. Periodontitis is associated with hypertension: a systematic review and meta-analysis. Cardiovasc Res 2020;116:28-39.

33. Leung WK, Siu SC, Chu FCS, Wong KW, Jin L, Sham ASK, Tsang CSP, Samaranayake LP. Oral health status of low-income, middle-aged to elderly Hong Kong Chinese with type 2 diabetes mellitus. Oral Health Prev Dent 2008;6:105-118.

34. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA 2003;289:2560-2572.

35. Ng SKS, Leung WK. A community study on the relationship between stress, coping, affective dispositions and periodontal attachment loss. Community Dent Oral Epidemiol 2006;34:252-266.

36. Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ. Update of the case definitions for population-based surveillance of periodontitis. J Periodontol 2012;83:1449-1454.

37. Cullinan MP, Palmer JE, Faddy MJ, Westerman B, Carle AD, West MJ, Seymour GJ. The influence of Triclosan on biomarkers of cardiovascular risk in patients in the cardiovascular and periodontal study (CAPS): A randomized controlled trial. J Periodontol 2015;86:847-855.

38. Choi HM, Han K, Park Y-G, Park J-B. Associations among oral hygiene behavior and hypertension prevalence and control: The 2008 to 2010 Korea National Health and Nutrition Examination Survey. J Periodontol 2015;86:866-873.

39. Martin-Cabezas R, Seelam N, Petit C, Agossa K, Gaertner S, Tenenbaum H, Davideau JL, Huck O. Association between periodontitis and arterial hypertension: A systematic review and meta-analysis. Am Heart J 2016;180:98-112.

40. Ahn Y-B, Shin M-S, Byun J-S, Kim H-D. The association of hypertension with periodontitis is highlighted in female adults: results from the fourth Korea national health and nutrition examination survey. J Clin Periodontol 2015;42:998–1005.

41. Pietropaoli D, Del Pinto R, Ferri C, Wright JT Jr, Giannoni M, Ortu E, Monaco A. Poor oral health and blood pressure control among US hypertensive adults: Results from the national health and nutrition examination survey 2009 to 2014. Hypertension 2018;72:1365-

1373.

42. Farina R, Scapoli C, Carrieri A, Guarnelli ME, Trombelli L, Prevalence of bleeding on probing: A cohort study in a specialist periodontal clinic. Quintessence Int 2011;42:57-68.

43. Lang NP, Joss A, Orsanic T, Gusberti FA, Siegrist BE. Bleeding on probing. A predictor for the progression of periodontal disease? J Clin Periodontol 1986;13:590-596.

44. Engström S, Gahnberg L, Högberg H, Svärdsudd K. Association between high blood pressure and deep periodontal pockets: A nested case-referent study. Ups J Med Sci 2007;112:95-103.

45. Martinez-Canut P, Lorca A, Magán R. Smoking and periodontal disease severity. J Clin Periodontol 1995;22:743-749.

46. Taguchi A, Sanada M, Suei Y, Ohtsuka M, Lee K, Tanimoto K, Tsuda M, Ohama K, Yoshizumi M, Higashi Y. Tooth loss is associated with an increased risk of hypertension in postmenopausal women. Hypertension 2004;43:1297-1300.

47. Völzke H, Schwahn C, Dorr M, Schwarz S, Robinson D, Doren M, Rettig R, Felix SB, John U, Kocher T. Gender differences in the relation between number of teeth and systolic blood pressure. J Hypertens 2006;241:257-1263.

48. Peres MA, Tsakos G, Barbato PR, Silva DA, Peres KG. Tooth loss is associated with increased blood pressure in adults - a multidisciplinary population-based study. J Clin Periodontol 2012;39:824-833.

49. Shin H-Y. Association between the number of teeth and hypertension in a study based on 13,561 participants. J Periodontol 2018;89:397-406.

50. Da D, Wanf F, Zhang H, Zeng X, Jiang Y, Zhao Q, Luo J, Ding D, Zhang Y, Wu B, Shanghai Aging Study. Association between tooth loss and hypertension among older Chinese adults: A community-based study. BMC Oral Health 2019;19:277.

51. Leung WK, Ng DKC, Jin L, Corbet EF. Tooth loss in treated periodontitis patients

responsible for their supportive care arrangements. J Clin Periodontol 2006; 33:265–275.

52. Rosling B, Serino G, Hellström MK, Socransky SS, Lindhe J. Longitudinal periodontal tissue alterations during supportive therapy. Findings from subjects with normal and high susceptibility to periodontal disease. J Clin Periodontol 2001;28:241-249.

53. Holmlund A, Holm G, Lind L. Number of teeth as a predictor of cardiovascular mortality in a cohort of 7,674 subjects followed for 12 years. J Periodontol 2010;81:870-876.

54. Joshipura KJ, Rimm EB, Douglass CW, Trichopoulos D, Ascherio A, Willett WC. Poor oral health and coronary heart disease. J Dent Res 1996;75:1631-1636.

55. Joshipura KJ, Hung HC, Rimm EB, Willett WC, Ascherio A. Periodontal disease, tooth loss, and incidence of ischemic stroke. Stroke 2003;34:47-52.

56. Susin C, Kingman A, Albandar JM. Effect of partial recording protocols on estimates of prevalence of periodontal disease. J Periodontol. 2005;76:262-267.

57. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R, et al. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet 2002;360:1903–1913.

58. Rapsomaniki E, Timmis A, George J, Pujades-Rodriguez M, Shah AD, Denaxas S, White IR, Caulfield MJ, Deanfield JE, Smeeth L, Williams B, Hingorani A, Hemingway H. Blood pressure and incidence of twelve cardiovascular diseases: lifetime risks, healthy life-years lost, and age-specific associations in 1.25 million people. Lancet 2014;383:1899–1911.

59. Stamler R. Implications of the INTERSALT study. Hypertension 1991;17(I suppl):I16-I20.

60. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, Roccella EJ, Stout R, Vallbona C, Winston MC, Karimbakas J. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. JAMA 2002;288:1882-1888.

61. Tonetti MS, D'Aiuto F, Nibali L, Donald A, Storry C, Parker M, Suvan J, Hingorani JD,

Vallance P, Deanfield J. Treatment of Periodontitis and endothelial function. N Engl J Med 2007;356:911-920. Erratum in: N Engl J Med 2018;378:2450.

62. Li X, Tse HF, Yiu KH, Jia N, Chen H, Li LS, Jin L. Increased levels of circulating endothelial progenitor cells in subjects with moderate to severe chronic periodontitis. J Clin Periodontol 2009;36:933-939.

63. Korner PI. Essential hypertension and its causes: neural and non-neural mechanisms. New York: Oxford University Press, 2007.

64. Ramseier CA, Anerud A, Dulac M, Lulic M, Culinan MP, Seymour GJ, Faddy MJ, Bürgin W, Schätzle M, Lang NP. Natural history of periodontitis: Disease progression and tooth loss over 40 years. J Clin Periodontol. 2017;44:1182–1191.

65. Sheiham A, Watt RG. The Common Risk Factor Approach: A rational basis for promoting oral health. Community Dent Oral Epidemiol 2000;28:399-406.

66. Leng XY, Chen XY, Chook P, Xiong L, Lin WH, Liu JY, Tomlinson B, Thomas GN, Lam TH, Lam KSL, Cheung BMY, Wong KS. Association between metabolic syndrome and carotid atherosclerosis: A community-based study in Hong Kong. Metab Syndr Relat Disord 2013;11:109-114.

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Table 1. Habits, dental attendance, blood pressure and periodontal status of the participants

		Hypertension				
		Case (n = 102)	Control (n = 102)	Test	Statistics	*P*-value
Smoking habit	Non-smoker	84 (82.4)	85 (83.3)	χ^2	0.034	0.853
	Smoker	18 (17.6)	17 (16.7)			
Tooth brushing habit	1×/day	17 (16.7)	18 (17.6)	χ^2	0.171	0.918
	2×/day	81 (79.4)	81 (79.4)			
	≥3×/day	4 (3.9)	3 (2.9)			
Use of floss/interdental brush	Daily	18 (17.6)	31 (30.4)	χ^2	9.374	0.009
	Occasionally	29 (28.4)	37 (36.3)			
	Never	55 (53.9)	34 (33.3)			
Last dental visit	≤1 year	45 (44.1)	53 (52.0)	χ^2	18.055	< 0.001
	>1 year ≤3 years	25 (24.5)	4 (3.9)			
	>3 years/never	32 (31.4)	45 (44.1)			
Systolic BP (mean ± SD)		142.08 ± 16.34	112.24 ± 7.96	t	-16.579	< 0.001
Diastolic BP (mean ± SD)		85.48 ± 8.96	68.47 ± 6.45	t	-15.560	< 0.001
		n (%)	n (%)			
BP category[†]	Normal[‡]	4 (3.9)	69 (67.6)	χ^2	160.59	< 0.001
	Elevated[‡]	9 (8.8)	33 (32.4)			
	Stage 1	26 (25.5)	0 (0.0)			
	Stage 2	61 (59.8)	0 (0.0)			
	Hypertensive crisis	2 (2.0)	0 (0.0)			
Pl%% (mean ± SD)		59.45 ± 21.46	77.42 ± 15.07	t	6.922	< 0.001

BOP% (mean ± SD)		58.56	±18.72	53.69	± 20.12	t	-1.787	0.075
Missing teeth (mean ± SD)		2.95	± 3.06	3.85	± 4.09	t	1.785	0.076
Mean PPD (mean ± SD)		2.73	± 0.73	2.40	± 0.52	t	-3.731	< 0.001
Mean PAL (mean ± SD)		3.16	± 0.94	2.51	± 1.01	t	-4.756	< 0.001
		n	(%)	n	(%)			
Periodontitis case definition[§]	No	3	(2.9)	23	(22.5)	χ^2	26.98	< 0.001
	Mild	0	(0.0)	6	(5.9)			
	Moderate	47	(46.1)	42	(41.2)			
	Severe	52	(51.0)	31	(30.4)			

BOP: bleeding on probing; BP: blood pressure in mm Hg; PAL: probing attachment level; PPD: probing pocket depth; SD: standard deviation
[†]BP (mmHg) categories: Normal: BP ≤119/≤ 79; Elevated: BP = 120-129/< 80; Stage 1: SBP = 130-139 or DBP = 80-89; Stage 2: SBP = 140-180 or DBP = 90-119; Hypertension crisis: SBP >180 and/or DBP >120 (Table S1).
[‡]BP (mmHg) control: Optimal: BP <130/< 80 for EH[14]
[§]No: No evidence of mild, moderate, or severe periodontitis; Mild periodontitis: ≥2 interproximal sites with AL ≥3 mm, and ≥2 interproximal sites with PD ≥4 mm (not on same tooth) or one site with PD ≥5 mm; Moderate periodontitis: ≥2 interproximal sites with AL ≥4 mm (not on same tooth), or ≥2 interproximal sites with PD ≥5 mm (not on same tooth); Severe periodontitis: ≥2 interproximal sites with AL ≥6 mm (not on same tooth) and ≥1 interproximal site with PD ≥5 mm (Table S2).[36]

Table 2. Regression analysis of variables associated with periodontitis case definition†

	Unadjusted				Adjusted			
Variables	Coefficient	*P*-value	95% CI		Coefficient	*P*-value	95% CI	
(Constant)	0.149	0.841	-1.312	1.609	-0.008	0.990	-1.222	1.206
Systolic BP	0.009	0.071	-0.001	0.019	0.008	0.028	0.001	0.014
Diastolic BP	-0.003	0.748	-0.020	0.014				
Age	0.011	0.089	-0.002	0.024	0.015	0.013	0.003	0.027
Sex	0.052	0.689	-0.205	0.309				
Smoking	0.364	0.041	0.014	0.715	0.378	0.026	0.046	0.710
User of floss/interdental brush	-0.054	0.522	-0.220	0.112				
Last dental visit	0.036	0.621	-0.107	0.178				
Pl%	-0.009	0.010	-0.016	-0.002	-0.009	0.011	-0.016	-0.002
BOP%	0.016	0.000	0.009	0.023	0.016	0.000	0.009	0.023
Missing teeth	0.018	0.346	-0.020	0.056				

r^2 (0.207) was computed using stepwise linear regression with variables that were significant ($P < 0.05$).

†Refer to Table S2 for details; no or mild periodontitis categories were collapsed into one group.

Table 3. Regression analysis of variables associated with full-mouth mean probing pocket depth

	Unadjusted				Adjusted			
Variables	Coefficient	*P*-value	95% CI		Coefficient	*P*-value	95% CI	
(Constant)	0.898	0.010	0.218	1.578	0.943	0.000	0.746	1.141
Systolic BP	-0.002	0.515	-0.006	0.003				
Diastolic BP	0.002	0.713	-0.007	0.010				
Age	-0.002	0.553	-0.008	0.004				
Sex	-0.033	0.590	-0.152	0.087				
Smoking	-0.108	0.194	-0.272	0.055				
User of floss/interdental brush	0.055	0.160	-0.022	0.132				
Last dental visit	-0.019	0.582	-0.085	0.048				
Pl%	0.003	0.060	0.000	0.007				
BOP%	0.007	0.000	0.003	0.011	0.009	0.000	0.006	0.012
Mean PAL	0.490	0.000	0.411	0.569	0.454	0.000	0.386	0.522
Missing teeth	-0.047	0.000	-0.067	-0.028	-0.047	0.000	-0.065	-0.029

r^2 (0.612) was computed using stepwise linear regression with variables that were significant ($P < 0.05$).

Table 4. Regression analysis of variables associated with full-mouth mean probing attachment level

	Unadjusted				Adjusted			
Variables	Coefficient	*P*-value	95% CI		Coefficient	*P*-value	95% CI	
(Constant)	-1.283	0.006	-2.201	-0.365	-1.164	0.004	-1.953	-0.375
Systolic BP	0.003	0.313	-0.003	0.010	0.007	0.001	0.003	0.012
Diastolic BP	0.009	0.114	-0.002	0.020				
Age	0.013	0.002	0.005	0.022	0.012	0.005	0.004	0.020
Sex	-0.025	0.758	-0.187	0.136				
Smoking	0.241	0.031	0.022	0.461	0.277	0.011	0.064	0.490
User of floss/interdental brush	-0.061	0.254	-0.165	0.044				
Last dental visit	0.039	0.396	-0.051	0.128				
Pl%	-0.008	0.001	-0.012	-0.003	-0.007	0.002	-0.011	-0.003
BOP%	0.005	0.062	0.000	0.010				
Mean PPD	0.896	0.000	0.752	1.041	0.971	0.000	0.846	1.097
Missing teeth	0.099	0.000	0.075	0.122	0.102	0.000	0.079	0.126

r^2 (0.716) was computed using stepwise linear regression with variables that were significant ($P < 0.05$).

Table 5. Regression analysis of variables associated with missing teeth

	Unadjusted				Adjusted			
Variables	Coefficient	*P*-value	95% CI		Coefficient	*P*-value	95% CI	
(Constant)	-0.281	0.909	-5.108	4.547	0.404	0.855	-3.943	4.752
Systolic BP	0.000	0.981	-0.032	0.033				
Diastolic BP	-0.051	0.073	-0.108	0.005	-0.051	0.010	-0.090	-0.013
Age	0.055	0.013	0.011	0.098	0.049	0.018	0.008	0.090
Sex	0.379	0.369	-0.452	1.209				
Smoking	0.282	0.627	-0.863	1.427				
User of floss/interdental brush	-0.174	0.527	-0.713	0.366				
Last dental visit	0.031	0.895	-0.431	0.493				
Pl%	0.026	0.029	0.003	0.049	0.032	0.002	0.012	0.052
BOP%	0.020	0.138	-0.007	0.047				
Mean PAL	2.626	0.000	1.994	3.258	2.780	0.000	2.189	3.371
Mean PPD	-2.300	0.000	-3.237	-1.363	-2.211	0.000	-3.097	-1.326

r^2 (0.409) was computed using stepwise linear regression with variables that were significant ($P < 0.05$).

Supplementary information

Periodontal conditions of essential hypertension attendees to a general hospital in Hong Kong

JJN Chiu, Y Zheng, SML Lai, WS Chan, SKW Yeung, HYC Bow, D Samartzis, EF Corbet, WK Leung.

METHODS

Blood pressure categories

BP was subcategorized as normal, elevated, stage 1, stage 2, or hypertension crisis as defined by Whelton et al. (2018) (Table S1).

Periodontitis case definition

For severity of periodontal disease, the periodontitis case definition guideline set by the working group appointed by the American Academy of Periodontology (AAP) and the CDC for population-based studies was adopted (Eke et al. 2012) (Table S2).

Data analysis

One-way ANOVA was used to compare means of categories classified under various subgroups. Phi and Cramer's v tests were conducted to measures the strength of relationship between two variables' categories (Akoglu, 2018).

RESULTS

Association between periodontal conditions, blood pressure or self-reported oral health behaviour

The association between oral hygiene habits, self-reported dental attendance, BP category, SBP or DBP and periodontal disease severity or PPD, PAL in an independent fashion were explored. In brief, S/DBP was significantly associated with BOP%, mean PPD, mean PAL and negatively associated with Pl% ($P < 0.001$, Pearson's Correlation). When BP categories were considered, BOP% was no longer significantly associated with the former ($P = 0.102$, one-way ANOVA).

Periodontitis case definition appeared very strongly associated with BP category (Phi and Creamer's V = 0.305), strongly associated with poor self-reported use of floss/interdental brush, and self-reported dental visit (Phi and Creamer's V ≥0.177).

Poor self-reported habit of flossing/interdental brushing appeared associated with more BOP% and higher mean PPD ($P < 0.04$, one-way ANOVA), while poor self-reported dental attendance was associated with Pl% ($P = 0.031$, one-way ANOVA).

Among the 102 EH participants, usage of calcium channel blockers (n=66), or not, did not associate with BOP% nor severity of periodontitis in terms of mean PPD, mean PAL or percentage sites PPD≥5mm (P≥0.135).

REFERENCES

Akoglu H. User's guide to correlation coefficients. Turk J Emerg Med. 2018;18:91–93.

Eke PI, Page RC, Wei L, Thornton-Evans G, Genco RJ. Update of the case definitions for population-based surveillance of periodontitis. J Periodontol 2012;83:1449-1454.

Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Himmelfarb CD, DePalma SM, Gidding S, Jamerson KA, Jones DW, MacLaughlin EJ, Muntner P, Ovbiagele B, Smith SC Jr, Spencer CC, Stafford RS, Taler SJ, Thomas RJ, Williams KA Sr, Williamson JD, Wright JT Jr. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. J Am Coll Cardiol 2018;71(19): e127-e248; Erratum in: J Am Coll Cardiol 2018;71(19):2275-2279.

Table S1. Categories of blood pressure in participants[†]

Blood pressure category	Systolic blood pressure (SBP)		Diastolic blood pressure (DBP)
Normal	< 120 mmHg	**and**	< 80 mmHg
Elevated	120 – 129 mmHg	**and**	< 80 mmHg
Hypertension stage 1	130 – 139 mmHg	**or**	80 – 89 mmHg
Hypertension stage 2	≥ 140 mmHg	**or**	≥ 90 mmHg
Hypertensive crisis	> 180 mmHg	**and/or**	> 120 mmHg

[†]Defined in accordance to Whelton et al. (2018). Individuals with SBP and DBP in two categories were designated to the higher BP category.

Table S2. Case definition of periodontitis[†]

Disease Category	Clinical Definition		
	PAL		PPD
No periodontitis	No evidence of mild, moderate, or severe periodontitis		
Mild periodontitis	≥ 2 interproximal sites with PAL ≥ 3 mm	**AND**	≥ 2 interproximal sites with PPD ≥ 4 mm (not on same tooth) or one site with PPD ≥ 5 mm
Moderate periodontitis	≥ 2 interproximal sites with PAL ≥ 4 mm (not on same tooth)	**OR**	≥ 2 interproximal sites with PPD ≥ 5 mm (not on same tooth)
Severe periodontitis	≥ 2 interproximal sites with PAL ≥ 6 mm (not on same tooth)	**AND**	≥ 1 interproximal site with PPD ≥ 5 mm

[†]Defined in accordance to Eke et al. (2012). Third molars were excluded.