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Research Article

Hypertension, Periodontal Disease, and Potassium Intake in Nonsmoking, Nondrinker African Women on No Medication

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The purpose of this cross-sectional study was to investigate the association of periodontitis and/or tooth loss with hypertension by excluding the common confounders. Eighty-one Tanzanian women who were aged 46–58 years, nonsmokers, nonalcoholic drinkers, and on no medication underwent clinical examination. Multiple-regression analysis showed that the severity of periodontitis was significantly correlated with increased systolic blood pressure and diastolic blood pressure. Simple-regression analysis indicated that the severity of periodontitis was inversely correlated with 24-hour urinary excretion of potassium ($r = -0.579$, $P = 0.0004$) and also inversely with the frequency of intakes of green vegetables ($r = -0.232$, $P = 0.031$) and fruits ($r = -0.217$, $P = 0.0043$). Low-potassium intake in the diet mostly accompanied by low dietary fiber intake increases BP as well as periodontal inflammation. Potassium intake may be an important factor linking periodontitis and hypertension in middle-aged nonsmoking and nonalcoholic women on no medication, although chronic inflammation such as periodontitis may cause hypertension through a more direct mechanism.

1. Introduction

Periodontitis, which affects a large number of adults globally, is epidemiologically related to atherosclerotic vascular diseases and metabolic syndrome [1, 2]. Periodontitis as chronic inflammation destroys the supporting structure of the teeth and increases the level of C-reactive protein (CRP) [3–5]. Recent attention has focused on elevated serum CRP, a marker of systemic inflammation, as a strong and independent risk factor or predictor of hypertension (HT) [6]. The systemic response to periodontal infection is a possible pathway underlying the observed association between periodontitis and increased risk for HT.

Epidemiological systematic examinations about the noted relationship between periodontitis and vascular diseases should be conducted among healthy subjects who have never smoked [7] because smoking is regarded as a strong confounder and may spuriously inflate the association between periodontitis and vascular diseases. Alcohol consumption is also one of the risk factors that can lead to the development of not only HT [8] but also periodontitis [9]. In nonsmoking menopausal Japanese women, tooth loss but not periodontitis was proven to be significantly associated with an increased risk of HT [10]. Medical care is also a confounding variable that strongly affects epidemiological studies in developed countries such as Japan, where epidemiological analysis on the association between vascular diseases and oral health may be complicated by the common use of certain antihypertensive medications that affect not only blood pressure (BP) but also oral health status [11]. In order to avoid these confounders, we selected middle-aged African women on no medication, who were nonsmokers and did not consume

Table 1: Basic characteristics of participants.

Parameter	Women ($n = 81$)
Age (year)	52.09 ± 3.92
SBP (mmHg)	139.93 ± 26.60
DBP (mmHg)	80.33 ± 20.80
BMI	28.57 ± 6.69
Total-C	5.33 ± 1.23
TG	1.63 ± 0.93
HDL- C	1.34 ± 0.39
HbA1c	5.22 ± 1.43
Vegetables (days/week)	4.20 ± 2.32
Fruit (days/week)	3.74 ± 1.47
Periodontitis	2.37 ± 0.66
Tooth loss	3.70 ± 3.81

Values are presented as mean ± standard deviation (SD). BMI: Body Mass Index (body weight/height^2, kg/m^2), Total-C: serum total cholesterol (mmol/L), TG: triglycerides (mmol/L), HDL-C: high-density-lipoprotein cholesterol (mmol/L), and HbA1c: glycosylated hemoglobin (%). Vegetable and fruit intakes are expressed as the number of days per week when they were consumed. Periodontitis: the average CPTIN of each tooth. Tooth loss: number of teeth lost.

alcoholic drinks, and investigated the possible association of periodontitis and tooth loss with the risk factors for HT in the present study.

2. Methods

2.1. Study Design and Subjects. The present study was conducted in Tanzania according to the protocol for Cardiovascular Diseases and Alimentary Comparison (CARDIAC) Study coordinated by the World Health Organization (WHO) [12–14]. We previously reported associations between HT and its traditional risk factors such as body mass index (BMI), salt intake, and Na/K ratio in the CARDIAC study in Tanzania [14–18]. One hundred women aged 46–58, living in Temeke, Dar es Salaam, were randomly selected from an administrative list and invited to participate in the study by letter. Detailed oral examination was limited to women because of the higher prevalence of smokers and alcohol drinkers among men, who were therefore considered not suitable for analysis of the association of oral health with dietary factors [7]. Eighty-one women responded to the invitation for BP and anthropometrical measurements, 24-hour urine collection, blood sampling, dental examination, and lifestyle questionnaires. This study was approved by the Institutional Ethical Review Board of Muhimbili University College of Health Science. Written informed consent was obtained in accordance with the institutional rules.

2.2. Health Examination. In order to eliminate observer bias, BP was measured using a centrally calibrated automatic BP measurement system used for the CARDIAC Study (Khi machine) [12, 13]. BP was measured 3 times for each subject, and the average of the 3 measurements was used in this analysis. Blood was sampled after a 10–14 h fast. Urinary bags (U-container N, Ono Medical Company Osaka, Japan) were used for collecting 24-hour urine and the validity was confirmed by checking creatinine excretion [13]. Blood and urine samples for the CARDIAC Study were analyzed centrally at the former WHO Collaborating Center for Research on Primary Prevention of Cardiovascular Diseases, Graduate School of Human and Environmental Studies, Kyoto University, Kyoto, Japan. Details of other health examination methods were described in previous reports on the protocol and findings of the CARDIAC study in Tanzania [14].

2.3. Oral Examination. A single examiner (M.Y.) carried out, without knowing other health-related data, the oral examination including tooth count and the assessment of periodontitis, which was measured according to the WHO community periodontal index of treatment needs (CPITN). The specially designed WHO periodontal probe with a sensing force of not over 20 g was utilized. The ten teeth examined were 17, 16, 11, 26, 27, 47, 46, 31, 36, and 37; for each tooth, the highest index found was recorded according to the following scale: (0) periodontal health; (1) gingival bleeding; (2) calculus detected during probing; (3) pocket 4 to 5 mm deep; and (4) pocket 6 mm deep and over. Periodontal condition was reported as the average CPTIN condition of each tooth, the scoring for which is well established and regarded as the global standard measurement for epidemiological and screening studies on periodontitis [19–21].

2.4. Statistical Analysis. Data were analyzed using the program Statview 5 for microcomputers from the SAS Institute Inc. Simple correlation analysis using the Pearson method allowed the assessment of univariate relationships. The variables were regarded as normally distributed.

3. Results

In this study, we assessed the association of tooth loss and periodontitis with BP, and the traditional risk factors of HT in 81 women aged 46–58 years, whose basic characteristics are shown in Table 1. None of the subjects in this population was found to be using or have used drugs that affect lipid metabolism, BP, or blood sugar. Additionally, they had never smoked and never drunk alcohol because of their local Islamic religious discipline and culture.

In multiple-regression analysis, the severity of periodontitis was significantly correlated with systolic BP (SBP) ($r = 0.288$, $P = 0.018$) and diastolic BP (DBP) ($r = 0.293$, $P = 0.015$), and tooth loss (the number of teeth missing) was also significantly correlated with SBP ($r = 0.308$, $P = 0.010$) and DBP ($r = 0.417, P = 0.0005$) (Table 2). These results indicate that periodontitis and tooth loss are significantly and independently associated with increases in BP in nonsmoking middle-aged Tanzanian women. In addition, to assess the association between HT and periodontitis, participants were divided into 3 groups. The severely hypertensive (SBP > 180 or DBP > 110) group had a significant difference in the severity of periodontitis (CPITN, 2.82 ± 0.64; mean ± standard deviation (SD)) from the normal to borderline hypertensive BP (SBP < 160 or DBP < 100) group (2.29 ± 0.61, $P < 0.05$).

Table 2: Multiple-regression analysis of BP, periodontitis, tooth loss, and traditional risk factors possibly related to hypertension in middle-aged women.

Parameter	Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)	
	β	P value	β	P value
Age (year)	0.103	0.364	0.051	0.649
BMI	0.270	0.024†	0.299	0.012†
Total-C	0.074	0.602	0.125	0.374
TG	0.187	0.545	0.099	0.452
HDL-C	0.081	0.165	−0.056	0.668
HbA1c	0.259	0.190	0.139	0.194
Periodontitis	0.288	0.018†	0.293	0.015†
Tooth loss	0.308	0.010†	0.417	0.0005‡

Values are regression coefficients (β) and P values. †$P < 0.05$, ‡$P < 0.001$.
Multiple correlation coefficient $|R| = 0.609$ for systolic blood pressure, $|R| = 0.621$ for diastolic blood pressure.

Table 3: Simple correlation coefficients between periodontitis and tooth loss with traditional risk factors possibly related to hypertension in middle-aged women.

	Periodontitis			Tooth loss	
Versus	r	P value	Versus	r	P value
NaCl (g/day)†	−0.179	0.329	NaCl (g/day)	−0.112	0.547
KCl (g/day)†	−0.579	0.0004§	KCl (g/day)	−0.204	0.265
Na/K	0.160	0.385	Na/K	0.081	0.660
Mg (mg/day)	−0.336	0.060	Mg (mg/day)	−0.273	0.131
Total-C	0.002	0.986	Total-C	−0.102	0.400
TG	−0.007	0.951	TG	−0.083	0.495
HDL-C	0.059	0.627	HDL-C	−0.114	0.345
BMI	−0.160	0.184	BMI	−0.271	0.022‡
HbA1c	−0.071	0.558	HbA1c	−0.158	0.189

Values are correlation coefficients (r), ‡$P < 0.05$, §$P < 0.001$.
24-hour urine ($n = 32$), blood chemistry and BMI ($n = 81$), and †24-hour urinary sodium and potassium excretions were measured and the intakes were estimated as the amount of chloride salt.

Next, we tested the association of periodontitis and tooth loss with traditional risk factors for HT in women (Table 3). There was a strong inverse correlation of 24-hour urinary excretion of potassium (Y; amount estimated as KCL) with severity of periodontitis (X) ($Y = 3.144 - .433X$; $r = -0.579$, and $r^2 = 0.34$, $P < 0.0004$), but not with tooth loss. No other risk factors were correlated significantly with periodontitis. On the other hand, tooth loss was significantly correlated with BMI ($r = -0.271$, $P = 0.022$) but not with the 24-hour urinary excretion of potassium. Since 24-hour urinary excretion was closely related to dietary intake of potassium-rich vegetables and fruit [22], these results suggest that nutritional factors, particularly potassium intake, were associated with periodontitis but not with tooth loss.

Therefore, we further assessed the relationship between dietary factors and periodontitis. There were significant negative correlations of the intakes of green vegetables ($r = -0.232$, $P = 0.031$) and fruit ($r = -0.217$, $P = 0.043$) with the severity of periodontitis in women (Table 4). These results indicate that the intake of potassium-rich foods, such as green vegetables and fruit, is associated with periodontitis. The intake of skimmed milk, but not whole milk, was significantly inversely correlated with the severity of periodontitis. This might be related to the preference for skimmed milk among vegetarians.

4. Discussion

In this study, we demonstrated that periodontitis and tooth loss were significantly associated with an increased risk of HT in middle-aged African women on no medication without any direct influence of smoking and drinking alcohol. HT is associated with increased levels of the markers of inflammation, including CRP and proinflammatory cytokines [6]. Periodontitis as chronic bacterial infection by Gram-negative bacteria is also associated with increased levels of inflammation-related markers such as CRP in circulation [3–5]. Recent studies suggest that everyday events such as chewing and brushing of teeth contribute more significantly to the cumulative exposure of the vascular system to oral bacteria [23]. Periodontal pathogens were identified in carotid atheromatous plaques obtained by endarterectomy from patients [24]. In periodontitis, bacteremia and/or endotoxemia may trigger a systemic inflammatory response that, in turn, may cause endothelial dysfunction and thus increase BP and accelerate atherosclerosis.

Table 4: Simple correlation coefficients between periodontitis and dietary intakes expressed by the frequency or the amount.

	Periodontitis	
Versus	r	P value
Vegetables (days/week)	−0.232	0.031†
Fruit (days/week)	−0.217	0.043†
Skimmed milk (mL/day)	−0.304	0.004†
Whole milk (mL/day)	−0.097	0.375
Coconut milk (mL/day)	0.119	0.273
Meat (days/week)	0.058	0.598
Fish (days/week)	0.052	0.637

Values are correlation coefficients (r), †$P < 0.05$.

One possible pathway is that periodontal infection induces systemic inflammation contributing to the development of HT. Endothelial dysfunction through periodontal infection-inflammation pathway [3, 25] might be the link between periodontitis and HT. The emerging evidence on the positive association of other inflammatory disorders, such as systemic lupus erythematosus [26] and rheumatoid arthritis [27], with increased HT risk, supports this as a plausible mechanism. The second pathway is that periodontitis might be associated with HT-related nutrients and foods. In this study, the severity of periodontitis but not the number of teeth lost was significantly and strongly associated only with daily potassium intake among the traditional risk factors related to HT, including nutrients, cholesterols, obesity, and diabetes. Moreover, we found that periodontitis was associated with less intake of potassium-containing foods, fruit, and vegetables. These results suggest that high consumption of potassium, possibly reducing the risk of HT, might decrease periodontitis severity and partially explain the association between periodontitis and HT.

The mechanisms on the possible causative links of periodontitis with potassium intake could be speculated. Firstly, potassium intake checked by 24-hour urinary excretion is associated with the consumption of vegetables and fruit [22], which may beneficially influence oral health [28]. Secondly, the tissue destruction in periodontitis is considered to be caused mainly by an aberrant inflammatory response involving prolonged release of neutrophil enzymes and reactive oxygen species. Potassium is reported to inhibit reactive oxygen species formation by human white blood cells [29] as well as to protect against hypertensive vascular injury and cardiac dysfunction, though, reducing reactive oxygen species in salt-induced experimental hypertension [30]. Vegetables and fruit are primary dietary sources of antioxidants, such as vitamins C and E. Intake of potassium-rich vegetables and fruit might help to prevent periodontitis by reducing free radicals and oxidative stress in periodontal tissue.

Previously, we reported that the mean levels of 8-hydroxydeoxyguanosine (8-OHdG), a marker of oxidative DNA damage in 24-hour urine samples, were significantly higher in hypertensive subjects than in normotensive subjects in Tanzania [31]. Our results suggested that a beneficial effect of the intake of potassium-rich vegetables and fruit on HT and periodontitis might be related to their roles in inflammation or oxidative stress, and that 24-hour urinary excretion of potassium might be a good biomarker for the risk evaluation of periodontitis as well as HT. The possibility that people with severe periodontitis who lose more teeth tend to eat less vegetables to absorb potassium, and therefore have higher BP, may be excluded because 24-hour urinary potassium excretion was significantly inversely related only with periodontitis but not with tooth loss.

Several limitations in the present study should be considered. First, the cross-sectional design basically precludes any causal inferences about the role of potassium intake between periodontitis and HT. Second, our analysis was carried out only once. Nevertheless, this study has several strengths. First, we controlled other major traditional risk factors, including potential confounders such as age, gender, race, diabetes, hypercholesterolemia, BMI, smoking, and alcohol consumption. The age range was limited to 46–58 years old to avoid the aging influence on HT and periodontitis. Second, the subjects were particularly suitable for the investigation of dietary influence on BP and oral health because they were on no medication and neither consumed alcohol nor smoked. Both of these are well-known confounders causing periodontitis as well as risk factors of HT. Third, to avoid examiner bias, oral examination was performed by a single examiner who was unaware of other health examination data. In addition, to provide an adequate assessment of the severity of periodontitis, CPITN was used as recommended by the WHO. CPITN score has become widely accepted as the method of choice for epidemiological and screening studies for periodontitis [19–21]. Fourth, nutritional assessments were carried out using not only a food frequency questionnaire but also biomarkers from 24-hour urine samples from African Muslim women whose dietary customs did not vary on a daily basis.

5. Conclusion

The present study demonstrated the association among dietary potassium intake, periodontitis, and HT. Low-potassium intake in the diet mostly accompanied by low dietary fiber intake increases BP as well as periodontal inflammation. Moreover, periodontitis may raise BP through chronic inflammatory and oxidative stress mechanisms. Therefore, oral health is important in reducing the risk of HT, and traditional dietary customs of consuming more potassium from vegetables and fruit are expected to decrease the risk of HT and periodontitis, both presently deteriorating the quality of life in the elderly, although this expectation remains to be proven by long-term intervention trials.

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