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A longitudinal study of the relationship between periodontal disease and bone mineral density in community-dwelling older adults

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Abstract

Objective: Bone loss is a common feature of periodontitis and osteoporosis. Both diseases may share common etiologic agents which may either affect or modulate the process of both diseases. The purpose of this study was to evaluate the relationship between systemic bone mineral density (BMD) and periodontal disease among older people.

Materials and Methods: Among all 4542 inhabitants aged 70 years according to a registry of residents in Niigata city in Japan, 600 people were selected randomly. One hundred and eighty-four subjects who did not have diabetes mellitus, whose blood sugar was <140 mg/dl, who had more than 20 teeth, who were non-smokers, and who did not take medication for osteoporosis, were included in the study. Four dentists performed clinical evaluations on probing attachment level (PAL). We also utilized the data on BMD of the heel, which we measured using an ultrasound bone densitometer. Follow-up clinical surveys were done by measuring PAL after 3 years. Finally, 179 subjects who could participate in both the baseline and the follow-up examinations were included in the analysis. After dividing the subjects into an osteopenia group (OG) and non-osteopenia group (NOG), we evaluated the relationship between BMD and the number of progressive sites which had $\geqslant 3$ mm additional attachment loss during 3 years after controlling the known confounding factors.

Results: The mean number of progressive sites for the OG and the NOG, respectively, were 4.65 ± 5.51 and 3.26 ± 3.01 in females and 6.88 ± 9.41 and 3.41 ± 2.79 in males. Two-way analysis of variance was performed to discriminate among effects of gender, BMD, and gender–BMD interaction. A significant effect of BMD (OG or NOG, $p = 0.043$) with a significant interaction ($p = 0.038$) was observed. Furthermore, BMD was associated with the number of progressive sites which had $\geqslant 3$ mm additional attachment loss during the 3 years ($p = 0.001$) by multiple linear regression analysis.

Conclusions: This study suggested that there was a significant relationship between periodontal disease and general BMD.

Key words: bone loss; etiology; periodontal disease

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Periodontal destruction is frequently experienced by elderly people (Slade & Spencer 1995, Brown et al. 1996) and it contributes to as much as 40% of tooth extraction (Johnson 1993). Periodontal disease is characterized by absorption of alveolar bone as well as by loss of the soft-tissue attachment to tooth. On the other hand, osteoporosis is the most common metabolic bone disease among the elderly (65 years and older), and the incidence of osteoporotic fractures obviously increases with aging. Because bone loss is a common feature of periodontitis and osteoporosis, both diseases may share common etiologic agents which may either affect or modulate the process of both diseases. Given that the final expression of periodontitis is predicated by the complex interactions occurring within an intricate mosaic of host, microbial and environmental factors, it was felt that the contribution of bone mineral density (BMD) as a risk factor might be worthy of investigation (Offenbacher 1996). The clinical consequence of these findings suggest that physicians should be encouraged to send their osteoporotic patients to dentists for a periodontal examination and dentists should be encouraged to send their patients with severe periodontal disease for a medical examination for osteoporosis.

However, the relationship between osteoporosis and periodontal disease has been suggested in a limited number of studies. The results of some previous studies have indicated a relationship between periodontal disease and osteoporosis (Von Wowern et al. 1994, Mohammad et al. 1997, Tezal et al. 2000), while others have not shown any significant relationship (Elders et al. 1992, Klemetti et al. 1994, Lundstrom et al. 2001). All of these studies used the cross-sectional study design, and examined bone loss and periodontal condition in females. Even if the loss of BMD was more significant in females than in males, the role of factors involved in the regulation of BMD in males as well as in postmenopausal females needs to be evaluated further with reference to oral bone loss and periodontal disease. In addition, it is necessary to evaluate the relationship between BMD and progression of periodontitis in longitudinal studies.

Likewise, the results may easily be confounded by other factors such as intake of medications, smoking, race and age. Many of the studies conducted to date have been plagued by relatively small sample sizes and lack of adequate control of potential confounding variables. Larger studies are needed to better define the relationship between BMD and periodontal disease.

The purpose of this study was to evaluate the relationship between systemic BMD and periodontal disease, controlling the known confounding factors.

Materials and Methods

Subjects and clinical assessment

Initially, questionnaires were sent to all 4542 inhabitants aged 70 years according to a registry of residents in Niigata City in Japan, and they were informed of the purpose of this survey. The response rate was 81.4% ($N = 3695$). Among them, after dividing into male and female groups, 600 people were selected randomly in order to have approximately the same number of each gender for the study (screened population). The subjects for the study agreed to undergo medical and dental examinations, and signed informed consent forms regarding the protocol, which was reviewed and approved by the Ethics Committee of the Faculty of Dentistry, Niigata University. The examinations were performed at local community centers in Niigata City. Four dentists performed clinical evaluations on the following items: (1) number of teeth present, (2) probing attachment level (PAL). Mouth mirrors with a light, and pressure-sensitive plastic periodontal probes, set to give a constant probing force of 20 g and graduated at 1 mm intervals (VIVACARE TPS PROBE®, Schaan, Liechtenstein), were used. All functioning teeth, including third molars, were assessed, except for partially erupted teeth. PALs were measured at six sites per tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual) and rounded to the nearest whole millimeter. In cases where a restorative margin was apical to the cemento-enamel junction (CEJ), PAL was measured taking account of the anatomical features of the teeth and, if present, the CEJ of the adjacent tooth/teeth.

Seventeen volunteer patients were examined by each of the four examiners in the Faculty Hospital of Dentistry, Niigata University, and their results were compared. The percentage of agreement ranged from 70.0% to 100% for PAL. The κ ranged from 0.62 to 1.00 for PAL. The four examiners did not have any information on BMD of the subjects.

The subjects' height, weight and grip power were measured to the nearest 1 mm or 0.1 kg, respectively, to calculate the body mass index (kg/m^2, BMI) or grip power/body weight (kg/kg). We also utilized the data on BMD of the heel, which we measured using an ultrasound bone densitometer (Lunar Achilles™, GE Medical Systems, Madison, WI, USA). The ultrasound signal is sent to os calcis. Ultrasound densitometry enables the measurement of the physical properties of bone, specifically BMD. The ultrasound measurement contains two criteria, the velocity (speed of sound (s); SOS) and frequency attenuation (broadband ultrasound attenuation (dB/MHz); BUA) of sound wave as it travels through bone (Langton et al. 1984, Rossman et al. 1989). The stiffness is a clinical index combining SOS and BUA, which is calculated by the spread speed of supersonic waves. The formula is $(\mathrm{BUA} - 50) \times 0.67 + (\mathrm{SOS} - 1380) \times 0.28$. This charts the SOS and BUA into biologically relevant ranges. Stiffness is indicated in the monitor of the bone densitometer as the percentage for the value of the normal younger generation. Osteopenia was defined as a stiffness $\leqslant 85$ for 70-year-old males, and $\leqslant 69$ for females (Lunar Corporation 1991). Furthermore, a personal interview was performed to obtain the bulk of information regarding smoking habits, diabetes mellitus, and the intake of medications for osteoporosis. To monitor the general health condition, serum or plasma levels of disease markers were also investigated. These disease markers were immunoglobulins (serum IgG concentration), nutritional factors (serum albumin concentration and serum total cholesterol concentration), and blood sugar. Among the screened population, 184 subjects who did not have diabetes mellitus, whose blood sugar was < 140 mg/dl, who had more than 20 teeth, who were non-smokers, and who did not take medication for osteoporosis were included in the study.

Follow-up clinical surveys were done by measuring PAL after 3 years. As at the baseline examination, 97.3% of the subjects received the follow-up examination by the same four dentists.

Finally, 179 subjects who could participate in both the baseline and the follow-up examinations were included in the analysis.

Statistical analyses

Mean and standard deviation (SD) were used to characterize the continuous variables. Following Brown et al. (1994), a change in the attachment level of 3 mm or more was set as a conservative estimate of actual change taking place. Using the *t*-test, we compared stiffness, BMI, serum albumin concentration, serum total cholesterol concentration, grip power/body weight, serum IgG concentration, PAL at baseline and the number of sites with $\geqslant 3$ mm additional attachment loss during the 3 years between males and females.

Furthermore, we evaluated the relationship between stiffness at the baseline and the number of sites with $\geqslant 3$ mm additional attachment loss during the 3 years by two-way analysis of variance (ANOVA) for discriminating among the effects of gender, stiffness and gender–stiffness interaction. After controlling for serum albumin concentration, serum total cholesterol concentration, grip power/body weight, serum IgG concentration, gender, BMI and PAL at baseline, a multiple linear regression analysis was performed to assess the relationship between stiffness at the baseline and the number of sites with $\geqslant 3$ mm additional attachment loss during the 3 years. The level of significance was set at $p < 0.05$ for these tests.

Results

The mean number of teeth present was 25.37 ± 2.91. The average PAL was 2.61 ± 0.76. Table 1 shows the stiffness, BMI, serum albumin concentration, serum total cholesterol concentration, grip power/body weight, serum IgG concentration, PAL and the number of sites with $\geqslant 3$ mm additional attachment loss during the 3 years between males and females. The stiffness was 74.19 ± 10.65 for males and 59.42 ± 8.87 for females. A significantly greater loss of stiffness was found in females ($p < 0.001$). The serum total cholesterol concentration was significantly lower, and grip power/body weight and PAL were significantly higher in males.

After dividing the subjects into the osteopenia group (stiffness $\leqslant 69$ for females, $\leqslant 85$ for males, OG) and the non-osteopenia group (NOG), we evaluated the number of progressive sites which had $\geqslant 3$ mm additional attachment loss during the 3 years. The mean number of progressive sites for the OG and the NOG, respectively, were 4.65 ± 5.51 and 3.26 ± 3.01 in females, 6.88 ± 9.41 and 3.41 ± 2.79 in males (Fig. 1). Two-way ANOVA was performed to discriminate among effects of gender, stiffness and gender–stiffness interaction. As shown by the data in Table 2, significant effects of stiffness (OG/NOG, $p = 0.043$) with a significant interaction ($p = 0.038$) were observed. The number of progressive sites was significantly higher in the OG. Furthermore, we evaluated the mean number of teeth present at baseline and tooth loss during the 3 years. The mean number of teeth present at baseline for the OG and the NOG, respectively, were 24.91 ± 2.71 and 25.05 ± 3.10 in females, 25.80 ± 2.96 and 25.95 ± 3.15 in males. There was no significance between the OG and the NOG in females and in males. The mean number of teeth lost during the 3 years for the OG and the NOG, respectively, were 0.84 ± 2.32 and 0.74 ± 1.41 in females and 0.52 ± 1.17 and 0.73 ± 0.83 in males. There was no significance between the OG and the NOG in females and in males as well.

The results of multiple linear regression analysis are presented in Table 3. Stiffness and gender were associated with the number of progressive sites which had $\geqslant 3$ mm additional attachment loss during the 3 years (stiffness: correlation coefficient $= -0.199$ ($p = 0.001$), gender: correlation coefficient $= -4.412$ ($p = 0.020$)).

Table 1. Comparison of stiffness, body mass index (BMI), biochemical values, grip power/body weight, probing attachment level (PAL) and additional attachment loss between males and females

Variables	Subjects ($n = 179$)		*p*-value
	males	females	
stiffness (%, mean ± SD)*	74.19 ± 10.65	59.42 ± 8.87	<0.001
BMI (kg/m^2, mean ± SD)*	22.56 ± 2.59	22.69 ± 2.78	0.752
albumin (g/dl, mean ± SD)*	4.30 ± 0.28	4.33 ± 0.24	0.500
total cholesterol (mg/dl, mean ± SD)*	194.42 ± 26.90	213.37 ± 29.12	<0.001
grip power/body weight (kg/kg, mean ± SD)*	0.67 ± 0.10	0.48 ± 0.08	<0.001
IgG (mg/dl, mean ± SD)*	1515.61 ± 262.88	1566.19 ± 336.75	0.269
PAL (mean ± SD)*	2.77 ± 0.80	2.46 ± 0.68	0.005
number of sites with ⩾3 mm additional attachment loss (mean ± SD)†	5.99 ± 8.36	4.37 ± 5.11	0.116

IgG, immunoglobulin G.
*At baseline.
†During the 3 years.

Discussion

The results showed that the subjects in the OG had a higher number of progressive sites with $\geqslant 3$ mm additional attachment loss during the 3 years than the subjects in the NOG. This 3-year longitudinal study clearly demonstrated that BMD is a risk predictor for periodontal disease progression in an older population.

Some systemic factors which contribute to loss of bone mass and periodontal progression have been identified (Cummings et al. 1985, Genco & Löe 1993). There were some common factors such as smoking, nutritional deficiencies, age, intake of medications and immune dysfunction (Wactawski-Wende et al. 1996). Considering these facts, it is reasonable that this study showed a significant relationship between BMD and periodontal disease progression. Maybe, systemic factors of bone remodeling also modify local tissue response to periodontal disease.

The relationship between BMD and progression of periodontitis is difficult to establish because there were many potential confounding variables, including local factors. In our previous study of an older population, we found that the subjects who had more than 20 remaining teeth were less susceptible to periodontal disease (Hirotomi et al. 2002). The results of that study prompted us to evaluate the relationship between systemic BMD and periodontal progression after controlling for teeth present, in addition to other factors, such as gender, diabetes mellitus, smoking habits and intake of medications in this study. Likewise, we restricted the age of subjects to 70 years to eliminate the influence of age on periodontal disease progression.

Various researchers have proposed several plausible findings. Kribbs et al.

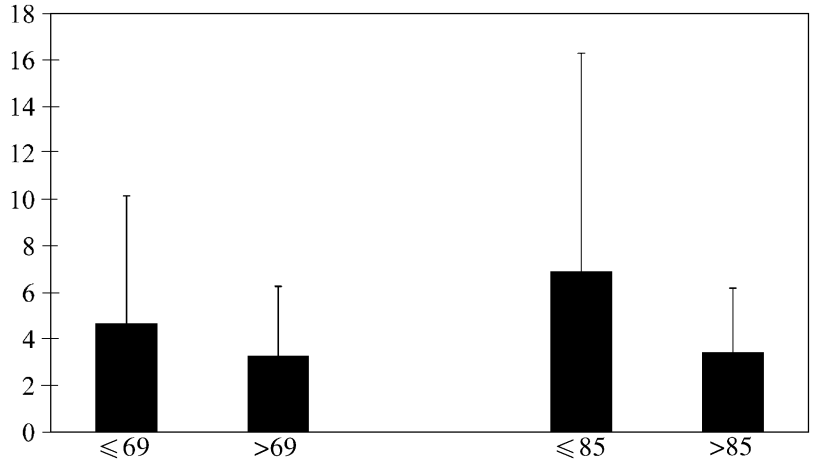

Fig. 1. Relationship between the number of progressive sites with ⩾3 mm additional attachment loss and stiffness by gender. The number of subjects: stiffness ⩽69 ($n = 74$) and >69 ($n = 19$) for females, ⩽85 ($n = 64$) and >85 ($n = 22$) for males. a, stiffness (%)

Table 2. The results of analysis of variance for the evaluation between additional attachment loss and bone mineral density and gender

Variables	Sum of squares	df	Mean square	*F*	*p*-value
stiffness (osteopenia/non-osteopenia)	191.67	1	191.67	4.140	0.043
gender (males/females)	136.30	1	136.30	2.940	0.088
stiffness × gender	309.34	2	154.67	3.340	0.038
residual	8148.89	176	46.30		
total	8458.22	178	47.52		

Table 3. Multiple linear regression and associated *p*-values

Independent variables*	Dependent variable				
	number of sites with ⩾3 mm additional attachment loss†				
	Coef.	Std. Err.	*p*-value	[95% CFI]	
stiffness (%)	−0.199	0.060	0.001	−0.317	−0.080
albumin (g/dl)	−4.286	2.200	0.053	−8.633	0.061
total cholesterol (mg/dl)	0.003	0.021	0.899	−0.039	0.044
grip power/body weight (kg/kg)	0.001	0.204	0.763	−0.341	0.464
IgG (mg/dl)	0.001	0.002	0.494	−0.002	0.005
gender (1: males, 2: females)	−4.412	1.881	0.020	−8.129	−0.695
BMI (kg/m^2)	0.195	0.231	0.401	−0.262	0.651
PAL‡	0.153	0.801	0.849	−1.431	1.736
_cons	35.687	12.544	0.005	10.896	60.479

$p = 0.033$, $R^2 = 0.106$. Coeff., coefficient; std. err., standard error; CFI, confidence interval; BMI, body mass index; IgG, immunoglobulin G.
*At baseline.
†During the 3 years.
‡Mean value of probing attachment level (PAL) at baseline.

(1990) observed a significant correlation between several skeletal bone mass measurements and the number of remaining teeth in 85 osteoporotic women between 50 and 80 years of age. Some other reports showed that mandibular bone mass was significantly correlated with skeletal bone mass as well (Klemetti et al. 1993, Von Wowern et al. 1994). Furthermore, the BMD of the mandible is affected by the mineral status of skeleton and also by general disease that causes generalized bone loss (Klemetti et al. 1993). On the contrary, Mohajery & Brooks (1992) found there was no correlation between skeletal and mandibular bone measurements. The results of these studies should be interpreted with caution since the number of subjects might be small, the age of subjects might have not been restricted, and the oral or skeletal bone loss might have been measured only in females.

In our study with adequate control of confounding variables, a weak relationship between BMD and periodontal disease progression existed although it was statistically significant. General BMD might not influence the alveolar bone loss directly in some cases. The skeleton is heterogenic, and bone density, bone turnover rate and bone remolding ability differ in some parts of the skeleton, suggesting that those regions, although related to each other, have some degree of independence. In addition, some bias such as local oral factors for alveolar bone loss might blur a clear relationship between systemic BMD and periodontal progression.

As our study was aimed at older subjects aged 70 years who had more than 20 teeth present, the subjects whom we examined might have been periodontitis-resistant. Therefore, it was difficult for PAL to contribute to inter-individual difference in resistance to periodontitis. This might be a reason for not having a significant relationship between periodontal disease condition such as PAL at baseline and additional attachment loss during the 3 years in this study. In addition, there was no significance in the number of teeth present at baseline, and tooth loss during the 3 years between the OG and the NOG in males and females. Therefore, the selection bias by the number of teeth present might be eliminated.

Likewise, ultrasonic bone density measurement was performed to evaluate BMD of the heel in this study. The ultrasound methods assess both bone volume and bone quality accurately and safely (Heaney et al. 1989). Some researchers have evaluated BMD by ultrasonic bone density measurement (Heaney et al. 1989, Resch et al. 1990). Ultrasound densitometry of the os calcis is highly reproducible and has a high correlation with BMD measured by dual-energy X-ray absorptiometry (DEXA) in different parts of the skeleton such as the spine or femur (Yamazaki et al. 1994).

In conclusion, this study suggested that there was a significant relationship between periodontal disease and general BMD in the present study.

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