number of male and female participants for the study.

Among the screened population, 184 subjects were included in the study, who did not have diabetes mellitus, whose blood sugar was less than 140 mg/dl, who had more than 20 teeth, who were non-smokers, and who did not take medication for osteoporosis.

Four dentists measured probing attachment level at six sites per tooth (kappa values: 0.62–0.80). In addition, we utilized the data on bone mineral density (BMD) of the heel, which we measured using an Ultra-Sound Bone Densitometer (Fig. 3, LUNAR Corporation, U.S.A.). Ultrasound densitometry enables the measurement of the physical properties of bone, specifically BMD. The ultrasound measurement contains two criteria, the velocity (speed of sound, SOS) and frequency attenuation (broadband ultrasound attenuation, BUA) of a sound wave as it travels through a bone.

Stiffness is a clinical index combining SOS and BUA, which is calculated by the spread speed of supersonic waves. The formula is:

\[ \text{Stiffness} = \frac{\text{BUA} \times 0.67 + \text{SOS} \times 0.28}{0.28} \]

This charts the SOS and BUA into biological relevant ranges. Stiffness is significant in the monitor of the bone densitometer as the percentage for the value of a normal younger generation. Osteopenia is defined as a stiffness that is ≤ 85 for females and ≤ 69 for males.

Follow-up clinical surveys were done by measuring probing attachment level. Finally, 179 subjects who could participate in both the baseline and the follow-up examinations were included in the analysis.

We measured the number of progression sites that had a 5 mm of additional attachment loss during the 5 years. After dividing the subjects into an osteopenia group (OG) — where stiffness was ≤ 69 for females and ≤ 85 for males — and a no-osteopenia group (NOG), we evaluated the number of progressive sites that had a 5 mm of additional attachment loss during the three years by two-way analysis of variance (ANOVA).

The mean number of progressive sites for the OG and NOG, respectively, were 4.65 ± 5.51 and 5.26 ± 5.01 in females, 6.88 ± 9.41 and 5.41 ± 2.79 in males. The difference in the mean number of progressive sites between the OG and NOG was statistically significant by ANOVA after controlling the gender (p = 0.045).

In addition, after controlling serum albumin concentration, serum total cholesterol concentration, grip power/body weight, serum IgG concentration, gender, BMI and probing attachment level at baseline, multiple linear regression analysis was performed to assess the relationship between the stiffness at baseline and the number of sites with ≤ 5 mm additional attachment loss during the three years. The results of multiple linear regression analysis are presented in Figure 4. Stiffness and gender were associated with the number of progressive sites that had ≤ 5 mm additional attachment loss during the three years (stiffness: correlation coefficient = −0.199 (p = 0.001); gender: correlation coefficient = −4.412 (p = 0.020)).

The results showed that the subjects in the OG had a higher number of progressive sites for additional attachment loss than the subjects in the NOG. This three-year longitudinal study clearly demonstrated that BMD is a risk factor for periodontal disease progression in an elderly population.

Future Outlook

According to our findings about the link to BMD, some systemic factors that contribute to both loss of bone mass and periodontal disease progression have been identified.16 Maybe systemic factors of bone remodeling also modify local tissue response to periodontal disease. The BMD of the mandible is affected by the merit status of the skeleton and also by general diseases that cause generalized bone loss.

Recently, several biochemical markers of bone metabolism have been developed, including a urinary cross-linked N-telopeptides of type I collagen (NTx), a bone-specific alkaline phosphatase (BALP), and others. We would like to evaluate the relationship between BMD using these biomarkers and maxillary/mandibular general bone loss in our future studies.

The mouth and face are highly accessible parts of the body, sensitive to and able to reflect changes occurring internally. For the clinician, the mouth and face provide ready access to physical signs and symptoms of local and generalized disease. During routine oral examinations, periodontal disease, including maxillary/mandibular general bone loss, may be diagnostic of early osteoporotic changes in the skeleton.

References


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Fig. 1 Variables in the Niigata Study. The variables which we selected in the Niigata Study are shown.

Fig. 2 Outcomes of the Niigata Study. Significant relationships which were confirmed by the Niigata Study are shown.

Fig. 3 Ultrasound Bone Densitometer (Achilles Bone Densitometer®), LUNAR corporation, USA). An ultrasound signal is sent to os calcis. Stiffness is a clinical index combining speed of sound and broadband ultrasound attenuation, which is calculated by the spread speed of supersonic waves.

Fig. 4 Multiple linear regression and associated p-values. Stiffness and gender were associated with the number of progressive sites which had ≤ 5 mm additional attachment loss during the 3 years (stiffness: correlation coefficient = −0.199 (p = 0.001); gender: correlation coefficient = −4.412 (p = 0.020)).