Total and Regional Bone Mineral Content in Patients with Non-Insulin Dependent Diabetes Mellitus

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Summary Total body bone mineral content and bone mineral content in various body sites were measured by dual-photon absorptiometry in 103 patients with non-insulin-dependent diabetes mellitus (NIDDM) and the findings were compared with those for 214 non-diabetic control subjects matched for age and body weight. Neither total body bone mineral content (TBBM) nor the bone mineral density of the third lumbar vertebra (L3 BMD) in the diabetic subjects differed from the values in control subjects of either sex, but the values were significantly decreased in patients diseased for at least five years when compared with control subjects. Regional bone mineral measurement showed prominent bone loss in the truncal site, but no reduction in bone mass was found in the head, pelvis, arms, or legs in either male or female patients. These results suggest that reduced TBBM and L3 BMD are associated with duration of the disease and that a site-specific bone defect is present in NIDDM.

Key Words bone mineral content, bone mineral density, dual-photon absorptiometry, non-insulin-dependent diabetes mellitus

Albright and Reifenstein first reported the coexistence of osteopenia and diabetes mellitus in 1948 (1). Although bone mass has fairly consistently been demonstrated to be decreased in patients with insulin-dependent diabetes mellitus (IDDM)(2-6), the relationship between bone mass and non-insulin-dependent diabetes mellitus (NIDDM) is still controversial. Heath and coworkers failed to find an increased incidence of fracture in about 1000 diabetic subjects as compared with a non-diabetic population (7). However, bone mass has been variously reported to be normal (8), increased (9), or decreased (2,10) in patients with NIDDM.

In order to address this issue further, we measured total body bone mineral content (BMC) and bone mineral content in various body sites in NIDDM patients, using dual-photon absorptiometry (DPA).
Table 1. Clinical characteristics of the study group.

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th></th>
<th>Female</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Control</td>
<td>NIDDM</td>
<td>Control</td>
<td>NIDDM</td>
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<tr>
<td>Number</td>
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<td>57</td>
<td>138</td>
<td>46</td>
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<tr>
<td>Age (yr)</td>
<td>54.8±10.3</td>
<td>55.0±9.5</td>
<td>58.0±10.9</td>
<td>59.5±9.9</td>
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<tr>
<td>Height (cm)</td>
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<td>163.6±6.1</td>
<td>151.2±6.0</td>
<td>151.2±5.0</td>
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<tr>
<td>Weight (kg)</td>
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<td>60.5±11.7</td>
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<td>22.5±3.8</td>
<td>23.8±4.9</td>
<td>24.7±5.4</td>
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<td>Duration of DM (yr)</td>
<td>8.5±7.2</td>
<td></td>
<td>7.2±6.8</td>
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</tbody>
</table>

All data are expressed as mean±S.D.

SUBJECTS AND METHODS

Table 1 shows some of the clinical characteristics of the subjects. The healthy control group consisted of 76 men with a mean age of 54.8 years and 138 women with a mean age of 58.0 years. The diabetic group consisted of 57 men and 46 women with NIDDM, with mean ages of 55.0 and 59.5 years, respectively. Height, body weight, and body mass index (BMI) were determined on the scanning day. The mean duration of the disease was 8.5±7.2 (SD) years in men and 7.2±6.8 years in women. None of the subjects had abnormal renal or liver function as demonstrable by routine tests. All subjects were free of any drugs known to influence bone and calcium metabolism. The controls had no known medical problems. All diabetic patients were classified as NIDDM according to National Diabetes Data Group (1979) criteria (11). The patients were matched for age, height, body weight, and BMI with the control subjects (Table 1).

A dichromatic bone densitometer (model 2600, Norland Corp., WI, U.S.A.) with a 153-gadolinium photon source was used to measure BMC. Total body bone mineral content (TBBM), lean body mass (LBM), and regional bone mineral density (BMD) were obtained from the total body scans. The regions scanned were the head, trunk, pelvis, legs, and arms. The value for arm BMD was expressed as the mean of the sum of right and left arm BMD values. The BMD of the third lumbar vertebra (L3 BMD) was evaluated from lumbar spine scans. BMD (g/cm²) was calculated as BMC divided by bone area. The coefficient of variation in the measurement of BMC and BMD utilizing our instrument was less than 2% (12).

ANALYTICAL PROCEDURE

Statistical analysis was performed using the unpaired Student’s t-test. Data are shown as means ± SD. Differences were considered significant when p values were less than 0.05.

RESULTS

Figure 1 shows the mean TBBM/LBM (mean ± 2SD) as a function of age in the
healthy controls. TBBM/LBM was employed for more accurate evaluation of TBBM, since the former parameter is not influenced by height and body weight (13). The mean TBBM/LBM showed no age-related changes in men, but it decreased in women over forty years of age. These changes were essentially similar to those in L3 BMD reported by us previously (12). As shown in Fig.2, the mean TBBM/LBM values in male and female diabetic patients were 5.07 ± 0.56% and 5.11 ± 1.08%, respectively, which did not differ from the respective levels in healthy controls (4.98 ± 0.52% in men, 5.16 ± 0.83% in women). However, in patients with a NIDDM duration of five years or more,
Fig. 3. Comparison of L3 BMD in control subjects, all diabetic subjects studied, and diabetic subjects diseased for 5 years or more.

TBBM/LBM showed a small but significant decrease from the control value for each sex (4.54 ± 0.55% in men, p<0.03; 4.70 ± 1.07% in women, p<0.05). The results were similar to those obtained for L3 BMD (Fig.3). Figures 4 and 5 show the results of regional BMD in controls and diabetics. A significant reduction in BMD was detected in the trunk only, and not in the bone of other parts of the body, including the head, pelvis, arms, and legs, in both male and female diabetics.

DISCUSSION

Levin et al. reported that single photon absorptiometry (SPA) showed a bone loss greater than 10% in 60% of NIDDM patients examined (2). Ishida et al., who utilized microdensimetry (MD), also reported that bone mass was significantly decreased in about 40% of diabetic patients examined (10). On the other hand, Johnson et al. found that the bone mass determined by SPA was greater in postmenopausal diabetic women than in control postmenopausal subjects (9). It should be pointed out, however, that SPA and MD were carried out at the forearm or metacarpus, where cortical bone was dominant. Since cortical bone mass is known to be a less accurate index of the osteoporotic state than trabecular bone mass (14), changes in cortical bone mass may not always parallel those in the mass of trabecular bone such as the lumbar spine and might not necessarily reflect what is occurring in the entire body bone (15).

The present study showed that the BMD of L3, which is rich in trabecular bone, in
NIDDM patients was similar to that in healthy subjects. This finding was consistent with Weinstock's observation (8) that the BMD of the lumbar spine in diabetic women did not
differ from that in non-diabetic women. This current study also shows that there was no significant difference in TBBM between the two groups. This may explain, in part, the epidemiologic finding that fracture did not occur more often in a diabetic than in a non-diabetic population (6).

Findings on the effects of disease duration on the magnitude of reduction in bone mass in diabetics have so far been ambiguous. In IDDM, several investigators (3, 4, 6) postulated that bone loss occurred before or only a few years following the clinical onset of diabetes, and that no further loss occurred thereafter. On the other hand, most studies of NIDDM have shown that the duration of diabetes was not related to the degree of bone loss in cortical (2, 10) or trabecular bone (8). We found, however, that patients with NIDDM of five years duration or more had significantly lower TBBM/LBM and L3 BMD than the control subjects. The reason for this discrepancy is not clear, but it may be due to differences in investigatory methodology and patient characteristics. The reduced TBBM/LBM and L3 BMD in the diabetics was not related to an age-dependent decline, since there was no significant difference in age between the entire diabetic group (55.0 ± 9.5 years in men, 59.5 ± 9.9 in women) and the sub-group with a duration ≥ five years (57.0 ± 9.8 in men, 60.1 ± 9.7 in women). Our finding of a reduction in bone mass five years after the clinical onset of diabetes suggested that the bone loss was due to a complication of the disease rather than to any inherent abnormality of bone formation in the patients. Earlier studies have shown that the severity or poor control of diabetes is a possible etiologic factor leading to osteopenia (3, 4).

Although there was no significant difference in BMD values in the head, pelvis, arms, or legs between the two groups we studied, a prominent deficit of BMD was detected in the truncal region in men as well as in women. To our knowledge, this site-specific bone defect has not been reported to date. The mechanism by which bone mass is selectively decreased in the trunk still remains to be clarified, but it seems reasonable to assume that bone loss commences in the trunk in NIDDM. If this is so, it may be of importance to measure truncal BMD for the early detection or prevention of osteopenia in this disease.

REFERENCES


