ration were not significantly different between the diabetic

Results

cision criterias for the survey were: Diabetes diagnosed at ≥30 years of age and patients who had type 2 diabetes for at least 5 years. The two groups were similar for physical activity and no history of smoking and previous history of hormone therapy. Patients with clinically relevant scoliosis or ectopic calcification were excluded.

Subjects and Methods

We studied 161 post-menopausal diabetic women with type 2 diabetes and a control group. We examined bone mineral density using the DXA technique at the lumbar and femoral regions and in a subgroup of patients, we also measured the levels of markers of bone remodelling. We found significantly higher levels of bone mineral density at the lumbar and femoral levels in the diabetic subjects compared with the control group. Moreover, we found higher level of urinary calcium in the controls. On the basis of these results, we suggest that osteoporosis cannot be considered a complication of type 2 diabetes.

Key words: Type 2 diabetes, Denosimetry, Osteoporosis

Introduction

Several diseases have been described as a cause of osteoporosis, such as diabetes mellitus but its results are uncertain in type 2 diabetes. Osteopenia is more severe when diabetes begins in the pubertal age and the reduction in the bone mineral density is more significant in the first 5 years after the onset of disease. It is reported that demineralisation is also related with the level of HbA1c (1,2). Some authors have reported low bone mineral density (BMD) in patients with type 2 diabetes but other studies found normal or higher levels than normal (1).

Giacca et al. found no differences in radial BMD between control subjects and patients with type 2 diabetes (4). Buysschaert et al. reported low BMD values in male patients with type 2 diabetes but normal values in female patients with type 2 diabetes (2). Kraukauer et al. found lower BMD values in type 2 diabetic patients than non diabetic patients (6).

The aim of this study is to examine the osteoporosis in type 2 diabetes and to ascertain whether it is a condition predisposing to reduced BMD.

Subjects and Methods

We studied 161 post-menopausal diabetic women with mean body mass index (BMI) of 30.17 ± 4.9 SD. The inclusion criterias for the survey were: Diabetes diagnosed at >30 years of age and patients who had type 2 diabetes for at least 5 years. We excluded subjects affected by diseases that can influence bone metabolism, history of any systemic diseases, hip and vertebral fracture and subjects treated with insulin and any drug that can interfere with bone turnover.

As a control group, we selected 90 healthy, non-diabetic women of similar age, menopausal age and BMI to diabetic patients. They had no history of any systemic disease, hip and vertebral fracture and drug administration that can interfere with bone metabolism.

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In diabetic patients, no subjects had high creatine levels.
patients and controls (Tab. 1). We did not find significant differences between diabetic and control subjects in the levels of serum calcium, phosphorus, ALP, and osteocalcin (Tab. 2).

Significant difference was found between two groups in the level of urinary calcium which was higher in the controls than diabetics *(p=0.005)*.

Bone mineral density, measured at the lumbar (L2-L4) and femoral (neck and total hip) regions were higher in type 2 diabetic patients than controls (Tab. 3). In diabetic patients, there was no correlation between age and BMD of the lumbar, femoral neck and total hip. The duration of diabetes did not correlate with BMD. However, BMI correlated with BMD of the femoral neck and total hip (*r* = 0.236, *p* = 0.330) but not with lumbar spine (L2-L4).

### Discussion

DEXA technique is preferable to the methods used for the evaluation of bone mineral density for its high precision and accuracy, low radiation dose and rapidity of execution (1). Our data agree with these of other authors who used the same technique.

We conclude that BMD is higher in patients with non insulin treated type 2 diabetes when compared with healthy subjects at the same age and sex. In addition, we did not observe evidence of greater bone resorption in the patients affected by type 2 diabetes than in normal subjects. Glucometabolic control may also protect type 2 diabetic patients and decreases bone turnover as in our diabetic patients (5).

At lumbar and femoral levels in the diabetic group, protection of cortical and trabecular bone which is inconsistent with the result of Isaia et al. (5). They explained the difference of BMD at lumbar and femoral levels by protection of cortical bone (5). They also found lower levels of PTH and urinary crosslinks in diabetics then in the control group (5,12).

Van Dalen PL et al. found higher than normal BMD in type 2 diabetic subjects, but Tuominen et al. found similar BMD values in type 2 diabetic patients and controls (14). Tuominen et al. also found lower BMD in type 2 diabetic men then women (13). Sex difference may be explained by obesity (13).

Our study does not confirm the results of previous studies that reported similar BMD values in type 2 diabetic and control subjects. Anabolic effect of hyperinsulinemia in patients with type 2 diabetes may result in increased bone mineral density by favoring osteoblastic activity. Obesity is also associated with increased BMD as a result of hyperinsulimia (8).

Pekern et al. reported that patients with non insulin dependent diabetes mellitus and especially overweight women have a normal or increased BMD (9). Rivshay et al. suggested that BMI is more important determinant of BMD than hyperinsulimia (10). However, Sosa et al. found that diabetic patients were obese with a higher body mass index than controls and there was no evidence that non insulin dependent diabetes mellitus causes any change in bone mineral density (11). In the study, we found statistically significant correlation between BMI and BMD of the femoral neck and total hip in diabetic patients.

Kwon et al. suggested that age, duration of diabetes and menopause duration in diabetics appear to be the risk factors for decreased BMD at the lumbar vertebra (7). We did not find any correlation between these factors and BMD in our diabetic patients. From a clinical point of view, we conclude that, bone mineral density is higher in patients with type 2 diabetes with non insulin treated diabetes. Osteoporosis cannot be considered as a complication of diabetes, but further studies are needed to explain the mechanism pathophysiologically.

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**Tab. 1: Characteristics of examined subjects (mean ± SD).**

| Age (years) | Menopause (years) | BMI (kg/m²) | Menarche age (years) |
|-------------|-------------------|-------------|---------------------|
| Diabetic subjects (n=161) | 61.06 ± 7.4 | 14.6 ± 8 | 30.17 ± 4.9 | 13.11 ± 0.8 |
| Control subjects (n=90) | 60.3 ± 7.4 | 13.4 ± 8 | 28.73 ± 4.4 | 13.28 ± 1.1 |

P-value 0.456 0.278 0.22 0.300

**Tab. 2: Bone mineral density (BMD), T score, Z score at lumbar and femoral level in diabetic patients and in the control group (mean ± SD).**

| L2-L4 | Control subjects | Diabetic subjects | P-value |
|-------|-----------------|-----------------|---------|
| T-Score | -2.05 ± 1.1 | -2.9 ± 0.7 | 0.01 |
| Z-Score | -0.42 ± 1.2 | -1.3 ± 1.4 | 0.01 |
| Femur (neck) | 0.355 ± 0.1 | 0.708 ± 0.1 | 0.01 |
| T-Score | -1.6 ± 1.0 | -2.1 ± 0.8 | 0.01 |
| Z-Score | 0.2 ± 0.9 | 0.3 ± 0.9 | 0.01 |
| Total hip | 0.802 ± 0.01 | 0.856 ± 0.4 | 0.00 |
| T-Score | -0.2 ± 0.8 | -1.5 ± 0.6 | 0.01 |
| Z-Score | -0.1 ± 0.9 | 0.6 ± 0.8 | 0.01 |

**Tab. 3: Levels of markers in diabetics and controls ± mean (SD).**

| Calcium (mg/dl) | 8.2 ± 0.12 | 8.5 ± 0.16 | 0.46 |
| Phosphorus (mg/dl) | 4.1 ± 0.14 | 4.1 ± 0.16 | 0.25 |
| ALP (U/l) | 195 ± 20 | 190 ± 15 | 0.23 |
| Osteocalcin (ng/ml) | 28.82 ± 1.4 (n=60) | 26.72 ± 3.2 (n=58) | 0.32 |
| Vitamin calcium | 123 ± 38 | 147 ± 67 | 0.005 |

**Discussion**

DEXA technique is preferable to the methods used for the evaluation of bone mineral density for its high precision and accuracy, low radiation dose and rapidity of execution (1). Our data agree with these of other authors who used the same technique.

We conclude that BMD is higher in patients with non insulin treated type 2 diabetes when compared with healthy subjects at the same age and sex. In addition, we did not observe evidence of greater bone resorption in the patients affected by type 2 diabetes than in normal subjects. Glucose control may also protect type 2 diabetic patients and decreases bone turnover as in our diabetic patients (3).

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From a clinical point of view, we conclude that, bone mineral density is higher in patients with type 2 diabetes with non insulin treated when compared with normal subjects. Osteoporosis can not be considered as a complication of diabetes, but further studies are needed to explain the mechanism pathophysiologyically.