Evaluation of biochemical markers and bone mineral density in patients with chronic kidney disease stage 5D at the start of hemodialysis treatment

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Background. Patients with chronic kidney disease (CKD) have significant disorders of bone and mineral metabolism. In addition, they can also develop other bone disorders including osteoporosis. This study evaluated the bone mineral density (BMD) of patients at the start of hemodialysis treatment as well as the relationship between BMD and possible risk factors or biochemical markers.

Methods. The study was performed in 82 patients (28 females, 54 males). BMD was measured by dual-energy X-ray absorptiometry (DXA) at the lumbar spine and the proximal femur.

Results. We found a high prevalence of 25-hydroxyvitamin D deficiency (96%; mean levels 30.0 ± 17.7 nmol/L) and a reduction of BMD in comparison with gender- and age-matched normal population values at the total hip (*Z*-score = -0.31 ± 1.11) and the femoral neck (*Z*-score = -0.48 ± 1.16), but not at the lumbar spine (*Z*-score = 0.68 ± 1.81). The prevalence of *T*-scores ≤ -2.5 SD in the group of patients over 50 years was 52.0% in females and 33.3% in males. BMD positively correlated: with male gender and calcium levels at all measured sites, with age at the lumbar spine and with weight or BMI at the proximal femur.

Conclusion. CKD patients at the start of hemodialysis treatment had a high prevalence of low *T*-score values, corresponding to values for osteoporosis in the general population. BMD at the proximal femur was below the expected average for age and gender, but at the lumbar spine, BMD in hemodialysis patients was above average in persons without known CKD.

Key words: bone mineral density, DXA, chronic kidney disease, hemodialysis, osteoporosis

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INTRODUCTION

Skeletal changes are initiated at the early stage of chronic renal failure, as estimated from reduced bone mineral density (BMD) (ref.[1]). Men and women with impaired kidney function are at increased risk of bone loss, even with minimal reduction in kidney function[2]. It is reasonable to assume that many patients with chronic kidney disease (CKD) have other disorders of bone that contribute to the final picture of renal osteodystrophy. Osteoporosis is the most prevalent bone disorder in the general population, but relatively little attention has been paid to its possible contribution to the bone alterations observed in patients with CKD, particularly in the increasing population of middle and older age groups that account for more than half of the patients on dialysis[3]. There is no reason why osteoporosis cannot accompany the derangements in bone metabolism that characterize chronic kidney disease. In fact, osteoporosis could and should be included in the broad characterization of chronic kidney disease-mineral and bone disorder (CKD-MBD), as recently proposed by the "Kidney Disease: Improving Global Outcomes" (KDIGO) working group. The pathophysiology leading to osteoporosis or CKD-MBD shares many common, yet distinctly different pathways. Both pathways may lead to the impairment of bone strength and the occurrence of low-trauma fractures[4].

Osteoporosis is generally defined as a skeletal disorder characterized by compromised bone strength predisposing to an increased risk of fracture[5]. In the general population, osteoporosis can be clinically diagnosed by the presence of a low trauma (fragility) fracture or by measuring BMD (ref.[6]). Osteoporosis may be diagnosed if the *T*-score of the lumbar spine, total hip, or femoral neck is -2.5 SD or less[7].The challenge in clinical practice is how to discriminate between osteoporosis and CKD-MBD. The diagnostic tools used to identify osteoporosis in the general population are not suitable for detecting the complex bone and metabolic changes that occur with chronic kidney disease. The interpretation of BMD in CKD patients is still a subject of controversy[5].

Almost all studies of BMD in patients with CKD stage 5D have investigated patients on long term hemodialysis treatment. The present study was designed to determine BMD in patients at the beginning of hemodialysis treatment and to assess the relationship between BMD and age, gender, diabetes mellitus, body mass index, residual renal function and biochemical markers (serum levels of 25-hydroxyvitamin D, parathyroid hormone, estradiol, calcium, phosphate, calcium-phosphate product and blood pH).

MATERIALS AND METHODS

Study subjects

Eighty-two CKD stage 5D patients from our dialysis unit were investigated. All patients had been undergoing hemodialysis treatment for less than 3 months, were clinically stable and their estimated glomerular filtration rate (eGFR) before the first dialysis treatment was < 0.25 mL/s/1.73 m^2. Exclusion criteria included the following: previous kidney transplantation, corticosteroid treatment and hormone replacement therapy. All patients were Caucasian. None of the patients had a history of osteoporotic fracture. The study was approved by the local ethics committee and all patients gave written informed consent.

The etiology of renal failure was diabetic nephropathy in 22 patients (27%), renal vascular disease in 20 (24%), glomerulonephritis in 14 (17%), tubulointerstitial nephritis in 11 (13%), adult cystic kidney disease in 5 (6%), other renal disease in 7 (9%), and unknown in 3 (4%). Diabetic nephropathy was the cause of renal failure in 63% of diabetic patients in this study, whereas among the other diabetics, the cause was different, mostly renal vascular disease (17%). All diabetic patients suffered from type 2 diabetes.

Biochemical measurements

Blood samples were collected after an overnight fast, before hemodialysis treatment, into plastic collection tubes containing lithium heparinate as an anticoagulant. Plasma was separated by centrifugation at 2000 × *g* for 15 min at 4 °C. Concentrations of the following substances were assayed: calcium, creatinine and phosphate, analyzed photometrically (AU 5420, Beckman Coulter, Inc., Brea, CA), and total 25-hydroxyvitamin D (DiaSorin, Inc., Stillwater, MN) and estradiol by RIA (Orion Diagnostica, Espoo, Finland). The concentration of intact parathyroid hormone (iPTH: parathyroid hormone 1-84 with 44.8% cross reactivity of fragment 7-84) was measured by Immulite 2000 (Siemens Medical Solutions Diagnostics, New York, NY) and blood pH by Radiometer ABL 835 FLEX (Radiometer Medical ApS, Brønshøj, Denmark). The analysis of calcium, creatinine, and phosphate showed inter-assay coefficients of variation between 2 and 5%. The analysis of 25-hydroxyvitamin D, estradiol, and parathyroid hormone showed inter-assay coefficients of variation of 8.3%, 12.3% and 8.7%, respectively. All biochemical markers were measured repeatedly once a month within a 3-month period before measurement of BMD. The mean value of 3 assays was taken for further assessment. Estimated glomerular filtration rate was calculated using the abbreviated MDRD (Modification of Diet in Renal Disease) formula.

Bone mineral density measurements

Determination of BMD was performed in each patient within three months of the start of hemodialysis treatment. Measurements were performed on a QRD 2000 bone densitometer (Hologic Inc.), using dual-energy X-ray absorptiometry (DXA). The results are expressed as *T*-scores and *Z*-scores and in absolute values (g/cm^2). *T*-scores and *Z*-scores reflect the number of standard deviations by which a patient value differs from the sex-matched young adult reference mean or from the sex- and age-matched mean, respectively. These scores are also expressed as a percentage. The reference database was created by the manufacturer, based on 1000 measurements of the lumbar spine (650 females, 350 males) and over 1400 measurements of the hip (750 females, 730 males) in healthy volunteers. BMD was assessed in two areas of the central skeleton, at the lumbar spine (L1 through L4) and at the site of the left proximal femur (at the femoral neck and the total hip).

Statistical analysis

Group characteristics are shown as mean ± SD. Potential differences between the groups were assessed by the Wilcoxon two-sample test. Differences between the groups in the prevalence of osteoporosis and osteopenia were tested by the Chi-Square test. Stepwise multiple regression analysis was applied to investigate relationships between BMD and risk factors or biochemical markers. Statistical analysis was performed using NCSS (Hintze J. (2001) NCSS and PASS, Number Cruncher Statistical System, Kaysville, UT, WWW.NCSS.COM).

RESULTS

Demographic and biochemical data

Patient data and statistical comparisons between the groups of females and males, non-diabetics and diabetics are presented in Table 1. Nine patients (11%) were under the age of 50 years, 41 patients (50%) between 50 and 70 years and 32 patients (39%) over the age of 70 years. Patients were predominantly male (66%). Diabetics accounted for 43% of the patients and were older and had a higher body mass index (BMI). Males had significantly higher values of eGFR, 25-hydroxyvitamin D (25(OH) D) and estradiol than females (89% of the female patients were post-menopausal). Higher eGFR values but lower 25(OH)D and iPTH levels were measured in the group of diabetic patients than in non-diabetics and these differences were statistically significant. Severe deficiency of 25-hydroxyvitamin D (below 12.5 nmol/L) was found in 17% of hemodialysis patients, mild deficiency (from 12.5 to 40 nmol/L) in 60%, and insufficiency (from 40

Table 1. Demographic and biochemical data.

Variable	All subjects (*N* = 82)	Females (*N* = 28)	Males (*N* = 54)	*P**	Non-diabetics (*N* = 47)	Diabetics (*N* = 35)	*P***
Age (years)	65.2 ± 14.0	66.9 ± 15.5	64.4 ± 13.2	NS	61.9 ± 14.5	69.7 ± 12.0	< 0.01
Weight (kg)	75.2 ± 14.7	68.1 ± 13.3	78.8 ± 14.2	< 0.01	73.4 ± 13.9	77.6 ± 15.6	NS
Height (cm)	169.7 ± 9.5	160.6 ± 5.9	174.5 ± 7.3	< 0.0001	171.2 ± 10.2	167.8 ± 8.2	NS
BMI (kg/m^2)	26.1 ± 4.5	26.4 ± 4.9	25.9 ± 4.3	NS	24.9 ± 3.5	27.6 ± 5.2	< 0.05
eGFR (mL/s/1.73m^2)	0.16 ± 0.05	0.14 ± 0.05	0.17 ± 0.05	< 0.01	0.14 ± 0.05	0.18 ± 0.06	< 0.05
25(OH)D (nmol/L)	30.0 ± 17.7	23.7 ± 10.2	33.2 ± 20.0	< 0.05	35.7 ± 20.2	22.7 ± 10.0	< 0.01
iPTH (pmol/L)	34.9 ± 31.9	36.9 ± 45.9	33.9 ± 21.8	NS	42.1 ± 39.3	25.3 ± 12.5	< 0.01
Estradiol (nmol/L)	0.062 ± 0.052	0.052 ± 0.077	0.067 ± 0.032	< 0.0001	0.065 ± 0.066	0.058 ± 0.027	NS
Calcium (mmol/L)	2.22 ± 0.17	2.26 ± 0.16	2.20 ± 0.17	NS	2.21 ± 0.16	2.23 ± 0.17	NS
Phosphate (mmol/L)	1.88 ± 0.45	1.87 ± 0.45	1.88 ± 0.44	NS	1.95 ± 0.51	1.79 ± 0.33	NS
Ca x P (mmol^2/L^2)	4.18 ± 1.03	4.22 ± 1.01	4.15 ± 1.05	NS	4.31 ± 1.16	3.99 ± 0.80	NS
blood pH	7.35 ± 0.04	7.34 ± 0.04	7.36 ± 0.04	NS	7.35 ± 0.04	7.37 ± 0.04	NS

Notes: BMI, body mass index; eGFR, estimated glomerular filtration rate; 25(OH)D, 25-hydroxyvitamin D; iPTH, intact parathyroid hormone; Ca x P, calcium phosphate product. Data are presented as means ± SD; *P**, comparison between females and males; *P*** comparison between non-diabetics and diabetics; NS, not significant.

Table 2. Bone densitometric data for all subjects and for subgroups of females, males, non-diabetics and diabetics.

	All subjects (*N* = 82)	Females (*N* = 28)	Males (*N* = 54)	*P**	Non-diabetics (*N* = 47)	Diabetics (*N* = 35)	*P***
LUMBAR SPINE							
g/cm^2	1.080 ± 0.231	1.000 ± 0.137	1.120 ± 0.258	< 0.01	1.068 ± 0.270	1.096 ± 0.170	NS
T-score (SD)	-0.31 ± 1.69	-0.79 ± 1.29	-0.06 ± 1.82	NS	-0.49 ± 1.81	-0.07 ± 1.51	NS
T-score (%)	96.8 ± 17.0	91.7 ± 13.5	99.3 ± 18.2	NS	95.0 ± 18.2	99.2 ± 15.2	NS
Z-score (SD)	0.68 ± 1.81	0.85 ± 1.76	0.59 ± 1.84	NS	0.43 ± 1.97	1.01 ± 1.54	NS
Z-score (%)	108.5 ± 21.1	112.8 ± 23.5	106.5 ± 19.7	NS	105.8 ± 22.9	112.2 ± 18.2	NS
NECK							
g/cm^2	0.742 ± 0.146	0.669 ± 0.098	0.779 ± 0.152	< 0.001	0.740 ± 0.135	0.745 ± 0.161	NS
T-score (SD)	-2.13 ± 1.12	-2.39 ± 0.96	-2.00 ± 1.19	NS	-2.20 ± 1.03	-2.04 ± 1.25	NS
T-score (%)	75.2 ± 13.3	71.5 ± 11.5	77.0 ± 13.8	NS	74.3 ± 12.1	76.3 ± 14.8	NS
Z-score (SD)	-0.48 ± 1.16	-0.40 ± 1.27	-0.52 ± 1.12	NS	-0.67 ± 1.06	-0.24 ± 1.26	NS
Z-score (%)	92.1 ± 19.3	90.5 ± 26.3	92.9 ± 15.1	NS	88.6 ± 20.1	96.8 ± 17.4	NS
TOTAL HIP							
g/cm^2	0.872 ± 0.158	0.762 ± 0.106	0.927 ± 0.152	< 0.0001	0.857 ± 0.143	0.891 ± 0.177	NS
T-score (SD)	-1.37 ± 1.07	-1.83 ± 0.89	-1.15 ± 1.09	< 0.05	-1.50 ± 0.95	-1.20 ± 1.21	NS
T-score (%)	82.1 ± 16.3	74.5 ± 18.5	85.8 ± 13.8	< 0.01	79.6 ± 16.9	85.3 ± 15.1	NS
Z-score (SD)	-0.31 ± 1.11	-0.34 ± 1.18	-0.29 ± 1.08	NS	-0.50 ± 0.96	-0.05 ± 1.25	NS
Z-score (%)	94.6 ± 19.1	92.0 ± 25.9	95.9 ± 14.8	NS	90.9 ± 19.4	99.6 ± 17.6	< 0.05

Notes: Data are presented as means ± SD; P*, comparison between females and males; P**, comparison between non-diabetics and diabetics; NS, not significant.

to 75 nmol/L) in 19%. Normal levels of 25(OH)D above 75 nmol/L were found in only 4% of patients (3 males, non-diabetics). Low levels of intact parathyroid hormone (below 16.5 pmol/L) were measured in 22% of patients, the mean (from 16.5 to 33.0 pmol/L) in 41%, and high levels (above 33.0 pmol/L) in 37% of the patient population. Classification of degrees of 25(OH)D deficiency and iPTH levels was based on K/DOQI guidelines[8].

Bone mineral density

Bone densitometric data are shown in Table 2. The lowest values of BMD expressed in g/cm^2 among all patient groups were found at the femoral neck, while the highest were found at the lumbar spine, where BMD values expressed as *Z*-scores were above the age- and sex-matched mean. Female patients had significantly lower BMD (in g/cm^2) at all measured sites as compared with males and furthermore, BMD values expressed as *T*-scores

Table 3. Prevalence of *T*-score values corresponding to osteoporosis and osteopenia for all subjects and for subgroups of females, males, non-diabetics and diabetics.

	All subjects (*N* = 82)	Females (*N* = 28)	Males (*N* = 54)	*P**	Non-diabetics (*N* = 47)	Diabetics (*N* = 35)	*P***
LUMBAR SPINE				NS			NS
T-score ≤ -2.5 SD	6.2%	7.4%	5.6%		8.7%	2.9%	
T-score -1.0 to -2.49 SD	33.3%	44.4%	27.8%		37.0%	28.6%	
Normal BMD	60.5%	48.1%	66.7%		54.3%	68.6%	
NECK				NS			NS
T-score ≤ -2.5 SD	35.8%	48.1%	29.6%		39.1%	31.4%	
T-score -1.0 to -2.49 SD	53.1%	40.7%	59.3%		50.0%	57.1%	
Normal BMD	11.1%	11.1%	11.1%		10.9%	11.4%	
TOTAL HIP				< 0,05			NS
T-score ≤ -2.5 SD	17.3%	33.3%	9.3%		15.2%	20.0%	
T-score -1.0 to -2.49 SD	45.7%	44.4%	46.3%		54.3%	34.3%	
Normal BMD	37.0%	22.2%	44.4%		30.4%	45.7%	

Notes: In the general population *T*-score ≤ -2.5 SD = Osteoporosis, *T*-score -1.0 to -2.49 SD = Osteopenia; *P**, comparison between females and males; *P***, comparison between non-diabetics and diabetics; NS, not significant.

Table 4. Prevalence of *T*-score ≤ -2.5 SD in the area of the proximal femur and/or at the lumbar spine in the age group over 50 years.

BOTH GENDER			FEMALES			MALES		
All > 50 y (*N* = 73)	> 50 - 70 y (*N* = 41)	> 70 y (*N* = 32)	All > 50 y (*N* = 25)	> 50 - 70 y (*N* = 12)	> 70 y (*N* = 13)	All > 50 y (*N* = 48)	> 50 - 70 y (*N* = 29)	> 70 y (*N* = 19)
39.7%	41.5% *	37.5% **	52.0%	50.0% *	53.8% **	33.3%	37.9% *	26.3% **

Notes: y, years; there was no statistically significant difference between groups * and **.

were significantly lower at the total hip. There were no statistically significant differences in BMD values between non-diabetic and diabetic patients, except for BMD expressed as *Z*-score % at the total hip.

The prevalence of *T*-scores in the ranges corresponding to values for osteoporosis and osteopenia in the general population are shown in Table 3. *T*-scores ≤ -2.5 SD were found most frequently at the femoral neck and this applies to all patient groups. Females had low values of *T*-scores significantly more frequently than males at the total hip. The differences in *T*-scores ≤ -2.5 SD between non-diabetic and diabetic patients were not significant.

Table 4 shows *T*-scores ≤ -2.5 SD in the area of the proximal femur and/or at the lumbar spine in the group of patients aged over 50 years. *T*-scores ≤ -2.5 SD were found most often in female patients over the age of 70 years, but the differences between the age groups from 50 to 70 years and over 70 years were not statistically significant. There were 9 patients (6 males and 3 females) in the age group up to 50 years (not listed in Table 4) and only one female had a *T*-score < -2.5 SD.

Significant factors influencing BMD findings selected by stepwise multiple linear regression are presented in Table 5. The selection was carried out among the factors listed in Table 1, including gender and diabetes mellitus.

DISCUSSION

The study found a high prevalence of 25-hydroxyvitamin D deficiency in our group of CKD stage 5D patients at the start of hemodialysis treatment. This condition is known to be common in hemodialysis patients[9,10]. We found significantly lower levels of 25(OH)D in females and diabetics than in males and non-diabetics, which is in agreement with findings of other studies[9-11]. A high prevalence of vitamin D deficiency suggests the possibility of a significant number of patients with osteomalacia in our study.

Measured values of iPTH were lower in diabetic than in non-diabetic subjects. It is known that diabetic hemodialysis patients are often characterized by a relative hypoparathyroidism and reduced bone remodeling[12,13].

Table 5. Independent variables reaching significance from stepwise linear multiple regression analyses using lumbar spine, femoral neck and total hip BMD as dependent variables (all subjects).

	LUMBAR SPINE (R^2 = 0.16)			NECK (R^2 = 0.32)			TOTAL HIP (R^2 = 0.44)		
	Regression Coefficient	Standardized Coefficient	*P*	Regression Coefficient	Standardized Coefficient	*P*	Regression Coefficient	Standardized Coefficient	*P*
Calcium	0.3213	0.23	< 0.05	0.1897	0.22	< 0.05	0.3023	0.32	< 0.001
Male gender	0.1473	0.30	< 0.01	0.0839	0.27	< 0.01	0.1867	0.56	< 0.0001
Age	0.0044	0.27	< 0.05	-	-		-	-	
Weight	-	-		0.0036	0.36	< 0.001	-	-	
BMI	-	-		-	-		0.0084	0.24	< 0.01

Poor glycaemic control in diabetic hemodialysis patients is associated with lower PTH values compared to diabetic hemodialysis patients with good control[14].

We identified significant differences in values of residual renal function between female and male patients, as well as between non-diabetic and diabetic patients. Jamal et al. did not find that bone loss increased with deteriorating kidney function[2]. Linear regression analysis in our study also showed no association between eGFR and BMD. Therefore, we believe that bone mineral density findings cannot be explained by the relatively small differences in the measured eGFR.

This study found a reduction in BMD, especially at the femoral neck and to a lesser extent, also at the total hip. In contrast, the mean value of *T*-scores at the lumbar spine was within the normal range and the mean value of *Z*-scores was actually above the average value expected for age- and sex-matched controls. Meta-analysis of densitometric studies published in KDIGO guidelines showed, that in areas with a greater proportion of cortical bone (proximal femur, forearm, total body), *Z*-scores for patients with CKD stage 5D were approximately -0.5 to -1 standard deviation; but at the lumbar spine, *Z*-scores were closer to the average in persons without known CKD (ref.[15]). These differences in BMD findings could arise for several reasons. The effects of increased parathyroid hormone may be different on cortical and trabecular bone and be dependent on the severity of hyperparathyroidism[16]. Since the proximal femur contains more cortical bone than the vertebral body, hyperparathyroidism may have a different effect on DXA findings at the lumbar spine and in the area of the proximal femur. In addition, artefacts can cause inaccuracies in DXA measurements in the lumbar spine. Any calcium in the path of the X-ray beam will contribute to the BMD measurement (degenerative disc disease, osteophytes, osteoarthritis with hyperostosis, aortic calcifications, etc.) and cause false elevation[3,16-18].

Lower BMD and higher prevalence of *T*-score values corresponding to osteoporosis in females in this study are consistent with data in the general population[19,20]. Female sex negatively correlated with BMD at all measured skeletal sites. Several studies of dialysis patients have reported the same findings[21-24]. On the other hand, some other studies have found no correlation between gender and BMD (ref.[25-27]).

In our study, no statistically significant differences were found either in BMD values (expressed as g/cm^2 and *T*-score) or in the prevalence of *T*-scores ≤ -2.5 SD between non-diabetic and diabetic patients. Only at the total hip, *Z*-score (expressed as %) was significantly higher in diabetics. The results of studies dealing with bone mineral density in patients with diabetes in the general population are inconsistent. However, a recent meta-analysis of 15 studies involving 3,437 diabetics and 19,139 non-diabetics, showed that BMD in type 2 diabetics was significantly higher in the area of the proximal femur and at the spine[28]. Studies in dialysis patients reported different conclusions: lower BMD in diabetics[21,29-31], no difference between non-diabetics and diabetics[32], or no difference at the spine but higher BMD at the forearm[33].

In the present study, a high prevalence of low *T*-score values (corresponding to values for osteoporosis) was found in the patients aged over 50 years. Studies in the general population of this age reported a lower prevalence of osteoporosis. The estimated prevalence of osteoporosis in Germany in 2003 was 39% in females and 9.7% in males[34], while data from Sweden showed the prevalence to be 21.2% and 6.3%, respectively[35]. According to a Canadian survey from 2009, 19% of females and 3% of males aged 50 or older reported having been diagnosed with osteoporosis[19]. The prevalence of low BMD is increased among patients with end-stage renal disease. Patients with later stages of CKD share similar risk factors with the general population for osteoporosis and are also affected by bone disease associated with CKD (ref.[3,26,36,37]). It should be noted that the bone disease associated with CKD is complex and multifactorial and BMD measurements alone may not be adequate to characterize the bone disorder[3].

Calcium levels positively correlated with BMD at all measured sites. Several studies in CKD patients have not previously reported this association[25,38-40]. It should be noted that calcium levels in our study were measured only during the three months before the determination of BMD and therefore do not reflect the long-term impact of calcium levels on BMD.

Positive association between age and BMD at the

lumbar spine can be explained by age-related changes. Artefacts in DXA measurements at this site are most common in the elderly population[3,37].

The results of our study demonstrate a positive correlation between BMD at the regions of the proximal femur and weight or body mass index. Low body weight is known to be a risk factor for low BMD in the general population and several studies in CKD patients observed the same relationship[40-43].

In conclusion, this study confirmed a high prevalence of 25-hydroxyvitamin D deficiency in hemodialysis patients, especially in women and diabetics. A significant reduction of BMD was found in the area of the proximal femur, but not at the lumbar spine. This difference may result from the different effects of parathyroid hormone on cortical and trabecular bone as well as the frequent false elevation of BMD in the lumbar spine in the elderly hemodialysis population. Compared to data obtained from the general population, the present study showed a higher prevalence of low *T*-score values (corresponding to values for osteoporosis) in patients over 50 years. Male gender and calcium levels were positively correlated with BMD at all measured sites. Age was positively correlated with BMD only at the lumbar spine, whereas weight and BMI correlated only in the area of the proximal femur.

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