FRI0500

INCIDENCE OF CLINICAL FRACTURES IN ADULTS WITH KIDNEY TRANSPLANT

Pablo Rodríguez-Merlos¹, Yanielli Hernández², Gerardo Napky¹, Lina Leon², Victoria Navarro-Compán¹, Maria Lopez², Alejandro Balsa¹, Carlos Jimenez², Pilar Aguado¹. ¹La Paz University Hospital, Rheumatology, Madrid, Spain; ²La Paz University Hospital, Nephrology, Madrid, Spain

Background: Chronic kidney disease is associated with an increased risk of fractures. However, the existing evidence investigating the incidence of fractures after kidney transplant is scarce.

Objectives: To evaluate the incidence of clinical fractures in patients with kidney transplant and analyze the possible factors that may influence its occurrence.

Methods: A retrospective observational study of patients who underwent kidney transplant between 2005 and 2015 in a tertiary hospital was conducted. A minimum follow-up period of 6 months after transplant was required. Sociodemographic and anthropometric data, clinical risk factors for bone fractures, drugs use (steroids, immunosuppressive drugs, vitamin D analogues, calcium and vitamin D supplements and calcimimetics), as well as biochemical and densitometric data to assess bone metabolism were retrieved from medical records. The occurrence of new clinical fractures after the transplant and throughout the follow-up period was recorded. The statistical analysis included univariable analysis using chi-square and Fisher's exact tests for the qualitative variables and T-Student and Mann-Whitney U for the quantitative variables. Subsequently, logistic regression multivariable analysis was carried out to investigate which factors were associated with fractures occurrence.

Results: A total of 163 patients were included, 63 (38%) females, with a mean age of 51 ± 14.6 years. The etiology of kidney disease was polycystic kidney disease (24.2%), glomerulonephritis (23.6%), diabetes mellitus (5.5%), vasculopathy (5.5%), connective tissue disorders (2.4%) and miscellany (38.7%). The daily average dose of steroids one year after transplant was 5.8 ± 4.4 mg. Bone densitometry was performed before transplant in 27 patients (19.6%), ten of whom (37%) presented osteopenia and 7 (26%) osteoporosis. The ten-year probability of bone fracture risk (FRAX) before transplant was 2.6 ± 2.6 . Mean follow-up after transplant was 8.7 ± 3.5 years. During this period, 23 (13.9%) patients suffered a clinical fracture, with an average time of appearance after transplant of 5.5 ± 3.3 years. These were located in hip (6 - 26.1%), vertebrae (2 - 8.7%), extremities (9 - 39.1%) and hands and feet (6 - 26%). One year after transplant, 65.6% had vitamin D deficiency (<30 ng/dl serum calcidiol levels) and 12.2% had levels compatible with osteomalacic range (<10 ng/dl). The data of the descriptive study stratified by the fracture occurrence are shown in Table 1. Compared with the subgroup patients without (Fx -), patients with (Fx +) post-transplant fractures showed to be more frequently female (60.9% vs 34.5%, $p=0.01$), older (50.1 ± 14.6 vs 56.3 ± 14.1 $p=0.03$), had more pre-transplant major clinical FRAX (2.4 ± 1.9 vs 4 ± 4.7 , $p=0.03$), and higher levels of PTH (98.2 ± 75.7 vs 140.1 ± 86.9 $p=0.02$) and serum alkaline phosphatase [ALP] (90.2 ± 37 vs 117.8 ± 60.4 , $p=0.02$) after one year of transplant. Also, a trend to present a higher prevalence of pre-transplant clinical fractures was observed in the second group (5.6% vs 17.4%, $p=0.06$). No difference in the use of immunosuppressants were detected. The multivariable analysis showed a significant association between post-transplant fractures and female gender (OR of 3.8, $p=0.03$) and higher levels of ALP (OR of 1.01, $p=0.03$).

Conclusion: In patients with kidney transplant, an incidence of clinical fracture of 13.9% is observed after a mean follow-up of 8.7 year. The female population seems especially susceptible to present fractures. The persistence of a possible pre-transplant autonomous secondary hyperparathyroidism one year after kidney transplant seems to influence the risk of fracture.

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Table 1. Descriptive study and comparison between groups (Fx - and Fx +). The data are shown as N(%) and mean (±SD).

	Total	Fx -	Fx +	P
Total (n)	163	142	23	
Mean age (years)	51 ± 14.6	50.1 ± 14.6	56.3 ± 14.1	0.03
Gender (female)	63 (38%)	49 (34.5%)	14 (60.9%)	0.01
BMI	25.5 ± 4.1	25.5 ± 4	25.6 ± 4.7	0.8
Smoking habit	23 (13.9%)	20 (14.1%)	3 (13%)	0.8
Diabetes mellitus	19 (11.5%)	15 (10.6%)	4 (17.4%)	0.2
Presence of fracture before transplant	12 (7.3%)	8 (5.6%)	4 (17.4%)	0.06
Major clinical FRAX at transplant	2.6 ± 2.6	2.4 ± 1.9	4 ± 4.7	0.03
Major densitometric FRAX at transplant	5.6 ± 4.6	4.9 ± 3.7	7.6 ± 6.4	0.2
Average steroid dose 1y after transplantation (mg/d)	5.8 ± 4.4	5.9 ± 3.5	5.9 ± 2.6	0.8
Use of Calcitriol 1y after transplantation	53 (32.1%)	44 (31.3%)	9 (39.1%)	0.2
Use of Cinacalcet 1y after transplantation	19 (11.5%)	16 (11.3%)	3 (13%)	0.8
Use of Calcidiol 1y after transplantation	48 (29.1%)	42 (29.6%)	6 (26.1%)	0.4
Serum calcium 1y after transplantation	9.6 ± 2.1	9.7 ± 2.3	9.6 ± 0.4	0.1
Serum phosphate 1y after transplantation	3.2 ± 0.9	3.2 ± 0.9	3.1 ± 0.6	0.9
PTH 1y after transplantation (pg/ml)	104.3 ± 78.5	98.2 ± 75.7	140.1 ± 86.9	0.02
Serum calcidiol 1y after transplantation (ng/ml)	26.5 ± 22.8	27.6 ± 23.9	20.4 ± 13.5	0.15
Stratified serum calcidiol (>30-20-10-10<10)	31 (34.4%) 19 (21.1%) 29 (32.2%) 11 (12.2%)	28 (36.4%) 17 (22.1%) 24 (31.2%) 8 (10.4%)	3 (23.1%) 2 (15.4%) 5 (38.5%) 3 (23.1%)	0.3
Serum alkaline phosphatase (U/L)	94 ± 42	90.2 ± 37	117.8 ± 60.4	0.02
T-Score Spine Pretransplant	-1.3 ± 1.4	-1.4 ± 1.3	-1.3 ± 1.7	1
BMD Spine Pretransplant (g/cm ²)	0.793 ± 0.084	0.932 ± 0.163	0.904 ± 0.181	0.6
T-Score Neck before transplantation	-1.9 ± 1.1	-1.8 ± 1.2	-2.3 ± 1.1	0.4
BMD Neck before transplantation (g/cm ²)	0.697 ± 0.116	0.709 ± 0.121	0.659 ± 0.098	0.3
Stratified DXA (normal/osteopenia/osteoporosis)	10 (37%) 10 (37%) 7 (25.9%)	7 (35%) 8 (40%) 5 (25%)	3 (42.9%) 2 (28.6%) 2 (28.6%)	0.5

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EVALUATION OF BONE QUALITY BY TRABECULAR BONE SCORE (TBS) IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

Andrea Casabella¹, Sabrina Paolino¹, Elisa Alessandri¹, Vanessa Smith², Barbara Ruaro¹, Carmen Pizzorni¹, Alberto Sulli¹, Maurizio Cutolo¹. ¹Research Laboratory and Academic Division of Rheumatology, Department of Internal Medicine, University of Genova, IRCCS San Martino Polyclinic Hospital, Genova, Italy., Genova, Italy; ²Department of Rheumatology, Ghent University Hospital, Ghent, Belgium; Department of Internal Medicine, Ghent University, Ghent, Belgium; Unit for Molecular Immunology and Inflammation, VIB Inflammation Research Center (IRC), Ghent, Belgium., Ghent, Belgium

Background: Systemic lupus erythematosus (SLE) patients shown an increased risk of low bone mass as a result of multifactorial events: physical inactivity, persistent inflammation, low vitamin D levels (photosensitivity) and glucocorticoid treatment. Trabecular Bone Score (TBS), is an index extracted from the dual-energy X-ray absorptiometry (DXA) that provides an indirect measurement of bone axial microarchitecture and allows to get information about bone quality in several rheumatic diseases (1-4).

Objectives: The aims of this study were to examine the prevalence and risk factors for low bone mineral density (BMD) (osteoporosis or osteopenia) in female patients affected by SLE and to compare with matched healthy subjects (CNT).

Methods: 40 female patients (mean age 47 ± 14 years) affected by SLE and 40 age-matched CNT (mean age 49 ± 8 years) were enrolled. Bone Mineral Density (BMD, g/cm²) of the lumbar spine (L1-L4) was analyzed using a DXA scan (GE, Lunar Prodigy). Lumbar spine TBS was derived for each spine DXA examination using the TBS index (TBS iNsight Medimaps).

Results: The mean BMD $\pm$ SD was 0.85 ± 0.22 g/cm² at the lumbar spine and 0.64 ± 0.32 g/cm² at the hip in SLE patients. The prevalence of osteopenia was 39.0% and 16.4% of osteoporosis in SLE patients. Most of SLE patients (70%) presented a bone loss that was significantly lower when compared with control group ($p<0.001$). Likewise, lumbar spine TBS

score was found significantly lower in SLE patients compared with CNT (0.868 ± 0.227 vs 1.482 ± 0.113 , $p < 0.001$, respectively). An history of high-dose oral glucocorticoids (> 10 mg/day) was associated with the preservation of BMD at the lumbar spine but not in spinal trabecular bone as observed by TBS analysis.

Conclusion: SLE is associated with significant trabecular bone loss, which could not be caused by glucocorticoid therapy. This study confirms the role of TBS as new and safe diagnostic tool for the quantification of the bone quality in chronic and systemic inflammatory rheumatic diseases, such as SLE.

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FRI0502 CORRELATION OF THE DAILY DIETARY INTAKE OF CALCIUM WITH THE LEVEL OF BONE MINERAL DENSITY IN PATIENTS WITH HUMAN IMMUNODEFICIENCY VIRUS INFECTION

Walter Alberto Sifuentes-Giraldo¹, María Llop Vilaltella¹, Cristina Macía-Villa¹, Pilar Vizcarra², Marta Monsalvo², Mónica Vázquez Díaz¹, José Luis Casado¹.
¹Hospital Ramón Y Cajal, Rheumatology, Madrid, Spain; ²Hospital Ramón Y Cajal, Infectious Diseases, Madrid, Spain

Background: There are few studies in which the relationship between daily calcium intake and bone mineral density (BMD) in patients with human immunodeficiency virus (HIV) infection has been evaluated, as well as its correlation with other factors of risk for the development of fragility fractures in this population.

Objectives: To determine the correlation of daily calcium intake with the most predictive risk factors for fragility fracture in patients with HIV infection, as well as BMD values in a cohort of patients followed in a Tertiary Madrid hospital.

Methods: Cross-sectional evaluation of a prospective study carried out in a specialized unit in HIV/AIDS of a tertiary Madrid hospital. We included asymptomatic consecutive patients with HIV infection, older than 50 years, followed regularly between January 2014 and December 2016.

Results: A total of 128 patients were included (35 women, 27%), with a mean age of 57 years (range: 50-83) and body mass index of 23.8 kg/m² (range: 15.6 - 33.5). The mean time of HIV infection was 256 months (range: 202-306) and of antiretroviral therapy (ART) 219.7 months (range: 156-247). The average calcium intake obtained by dietary calculation (without supplementation) was 563.8 g/day (462 - 2772). Among the risk factors for fragility fracture (included in the FRAX): 44 (34%) reported smoking, 11 (9%) family history of fracture, 54 (42%) previous history of fracture, 8 (6%) significant alcohol consumption and 25 (21%) renal tubular dysfunction. According to the WHO classification based on BMD at the level of lumbar spine (LS) 50 (39%) were classified as osteopenia and 43 (34%) as osteoporosis, while at the femoral neck level said proportions were 83 (65%) and 9 (7%), respectively. A correlation was found between higher calcium intake with a longer time of ART ($\rho = 0.2$, $p = 0.02$), but not with the age or time of HIV infection. The calcium intake showed no correlation with serum calcium levels ($\rho = 0.05$, $p = 0.72$), serum phosphorus levels or bone biomarkers such as alkaline phosphatase, osteocalcin or P1NP, but an inverse relationship with the levels of β -crosslaps ($\rho = -0.21$; $p = 0.02$). Calcium intake was associated with greater exercise, assessed by the International Physical Activity Questionnaire (IPAQ) ($\rho = 0.23$; $p = 0.01$). The calcium intake was lower in patients with osteoporosis with respect to osteopenia or normal (hip 500 vs 580 vs 573, LS 507 vs 635 vs 585, respectively, $p = 0.04$ between osteoporosis/osteopenia). Although a lower calcium intake was found in patients with vertebral fractures, this was not significant (486 (236) vs 583 (317), $p = 0.09$).

Conclusion: Although recent meta-analyses show that calcium intake in non-HIV population is not related to the development of fragility fractures, in our cohort (with the limitations of a cross-sectional study) a probable association of daily calcium intake with a low BMD is evidenced. These findings should be confirmed in the longitudinal analysis of the data of the cohort.

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FRI0503 USING BONE MINERAL DENSITY VERSUS THE RATIO OF BODY MASS INDEX TO BONE MINERAL DENSITY TO PREDICT FRACTURE RISK IN HYPERTHYROIDISM

Khojasta Talash, Marwan Bukhari. *Royal Lancaster Infirmary, Lancaster, United Kingdom*

Background: Euthyroidism is important in the development of a normal skeletal system, with thyroid hormones acting as important regulators of bone homeostasis in adults¹. Hyperthyroidism, whether current or previous, increases the risk of developing osteoporotic fractures by stimulating osteoclastic bone resorption and hence bone remodelling, which overall results in decreased bone mineral density (BMD)². Generally, BMD is used as a predictor of fracture risk; however there has been recent research that suggests using the ratio of BMD to Body Mass Index (BMI) is a better marker of predicting fracture risk in obese patients than BMD alone³.

Objectives: Our research set out to find whether BMD alone or the ratio of BMI to BMD is a better predictor of fracture risk in patients with current or previous hyperthyroidism.

Methods: Data were used from a cohort of patients with current or previous hyperthyroidism, referred for DEXA scan to a District General Hospital between June 2004 and October 2010. The following were recorded: age, sex, whether a fracture was sustained, whether they had had steroid therapy at any point, BMI, BMD at L1-L4, BMD at femoral neck (left and right) and BMD at hip (left and right). Logistic regression models were fitted using fracture as the dependent variable. The independent variables for the first set of logistic regression models were BMD at each level and for the second set BMI:BMD ratio at the same levels. Data were adjusted for sex and age at scan. The fit of logistic models were compared using area under the ROC curves (AUC).

Results: 720 patients were used in the study, of whom 643 (89.3%) were female. Mean age was 63.6 years (SD 11.6) with age range of 28.4 to 89.6 years. 120 (16.7%) were recorded to have had steroid therapy at any point. Mean BMI was 25.9 kg/cm² (SD 4.77). 274 (38.1%) had sustained a fracture. Odds ratios and AUC values for each level were as shown in the table. The fit of the models using the ratio was generally superior to the fit of the models using BMD alone at the hips and femurs, as AUC values were generally greater for the ratio. At each individual level of the lumbar spine the BMD alone provided a better fit, however overall the ratio gave a slightly better fit.

Table 1. – Odds ratios (age- and sex-adjusted) and AUC values

Level	Odds Ratio and CI (BMD)	AUC (BMD)	Odds Ratio and CI (BMI:BMD)	AUC (BMI: BMD)
L1	0.128 (0.0510, 0.324)	0.6277	1.07 (1.04, 1.09)	0.6305
L2	0.161 (0.0697, 0.374)	0.6277	1.06 (1.03, 1.09)	0.6250
L3	0.128 (0.0510, 0.324)	0.6418	1.06 (1.04, 1.09)	0.6307
L4	0.188 (0.0879, 0.401)	0.6282	1.06 (1.03, 1.09)	0.6249
L1 to L4	0.120 (0.0496, 0.291)	0.6282	1.07 (1.04, 1.10)	0.6309
L FEMORAL NECK	0.0988 (0.0290, 0.336)	0.6219	1.05 (1.02, 1.07)	0.6285
R FEMORAL NECK	0.0450 (0.0877, 0.231)	0.6776	1.06 (1.03, 1.09)	0.6798
L TOTAL	0.0980 (0.0233, 0.412)	0.6812	1.07 (1.03, 1.10)	0.6936
R TOTAL	0.0623 (0.0136, 0.285)	0.6778	1.07 (1.03, 1.10)	0.6850

Conclusion: This study identifies that the BMI:BMD ratio does not provide better indication of fracture risk than BMD alone in our cohort of patients with current or previous hyperthyroidism. We have previously shown that the same is true for patients with rheumatoid arthritis. A limitation of this study is not stratifying by presence of other diseases or steroid use.