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ORIGINAL ARTICLE

Chronic hemodialysis is associated with lower trabecular bone score, independent of bone mineral density: a case-control study

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Abstract

Summary We measured trabecular bone score (TBS) in 98 patients on permanent hemodialysis (HD) and 98 subjects with similar bone mineral density and normal kidney function. TBS was significantly lower in HD patients, indicating deteriorated bone microarchitecture, independent of bone mass. This might partially explain the increased fracture risk in HD.

Purpose In the general population, trabecular bone score (TBS) was shown to predict fracture independent of bone mineral density (BMD). In end-stage renal disease patients on hemodialysis (HD), the value of TBS is beyond that of BMD in currently unclear. Our aim was to assess lumbar spine (LS) TBS in HD patients compared with subjects with normal kidney function matched for age, sex, and LS BMD.

Methods We assessed TBS and LS and femoral neck (FN) BMD in 98 patient on permanent HD (42.8% males; mean age 57.5 ± 11.3 years; dialysis vintage 5.5 ± 3.8 years) and 98 control subjects (glomerular filtration rate > 60 mL/min) using DXA. We simultaneously controlled for sex, age (± 3 years), and LS BMD (± 0.03 g/cm^2).

Results HD patients had significantly lower LS TBS (0.07 [95% CI 0.03–0.1]; $p = 0.0004$), TBS *T*-score (0.83 SD [95% CI 0.42–1.24]; $p = 0.0001$)) and TBS *Z*-score (0.81 SD [95% CI 0.41–1.20]; $p = 0.0001$) than matched controls. TBS significantly correlated with LS BMD in both HD patients ($r = 0.382$; $p = 0.001$) and controls ($r = 0.36$; $p = 0.002$). The two regression lines had similar slopes (0.3 vs. 0.28; $p = 0.84$) with different intercepts (0.88 vs. 0.98). TBS adjustment significantly increased the 10-year fracture risk from 3.7 to 5.3 for major osteoporotic fracture and from 0.9 to 1.5 for hip fracture.

Conclusions HD patients have lower TBS than controls matched for LS BMD, indicating altered bone microarchitecture. Also, the magnitude of TBS reduction in HD patients is constant at any LS BMD. Adjustment for TBS partially corrects the absolute 10-year fracture risk.

Keywords Trabecular bone score · Bone mineral density · End-stage renal disease · Fracture risk

Introduction

Permanent hemodialysis (HD) is associated with an increased risk of hip fracture [1]. Although the risk of vertebral fracture (VF) was found both to be similar to that of the general population [2] or increased to the level of the osteoporotic patients [3], in HD patients, VF are associated with increased mortality [2]. In the general population, the most important risk factors for fracture are prior fractures [4] and low bone mineral density (BMD) [5]. BMD is readily measured using dual-energy x-ray absorptiometry (DXA). The predictive value of DXA measured BMD for vertebral and non-vertebral fractures has been proved in many prospective studies and is currently incorporated in risk assessment tools like FRAX [5]. Low DXA measured BMD is also associated with low trauma fractures in patients with HD [6]. However, the ability of BMD to predict fractures is lower in HD compared with the general population [6]. One of the many proposed explanations for this low

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predictive value is microstructural damage and trabecular loss of connectivity, a feature not captured by DXA.

Assessment of microarchitecture and trabeculae is challenging in clinical practice as methods are invasive and/or expensive. Trabecular bone score (TBS) is a surrogate marker of bone microarchitecture computed using the gray-scale variogram of DXA examinations [7]. Although there are some concerns regarding the physical parameters actually measured by TBS (trabecular thickness is of the same order of magnitude as the DXA scan resolution) [8], numerous cross-sectional, and prospective studies confirmed the value of TBS in predicting fracture in the general population [9]. Moreover, studies have shown that TBS adds value to DXA measured BMD and could be incorporated in the FRAX tool [10]. It works as a software add on the DXA machine and uses the same BMD scan.

All previous studies on TBS in end-stage renal disease (ESRD) patients have showed a significant correlation between lumbar spine (LS) TBS and BMD at both LS and total proximal hip (TPH) [11–14], similar to general population. Only one study compared TBS in ESRD patients with and without prevalent fractures and found a lower TBS in patients with non-vertebral fractures [14]. However, lower TBS was paralleled by lower BMD at the vertebral and non-vertebral sites. Interestingly, they did not find any correlation between TBS and vertebral fractures as could be expected [14]. The value of TBS beyond that of BMD in patients with ESRD on HD is presently unclear.

The aim of our study was to assess LS TBS in HD patients compared with subjects with normal kidney function matched for age, sex, and LS BMD.

Methods

Patients

We assessed in our department 98 patients with ESRD on permanent HD for at least 6 months. For each patient, we recorded age, sex, body mass index (BMI), years on dialysis, and medication. No patient had any partial or total parathyroidectomy. Seven (7.1%) patients had previous treatments with glucocorticoids (at least 3 months with a dose of at least 5 mg daily prednisolone equivalent). No patient had any previous or current anti-osteoporotic (bisphosphonates, denosumab, etc) treatment. The study was approved by the Institutional Ethic Committee. All patients signed an informed consent. Patients' characteristics can be found in Table 1.

Controls

The control group was set up using our Institutional Electronic Database. For each case, we matched a single control subject based on following simultaneous criteria: sex, age (± 3 years), and LS BMD (± 0.03 g/cm^2). The LS BMD least significant change is around 3% in our institution, translating into a ~ 0.03 g/cm^2 absolute difference. The 3 year loss of BMD at different skeletal sites is around 1% [15]. The mean ± SD BMD at the LS was 1.029 ± 0.179 g/cm2 in our HD group. A ± 3 years difference in age would roughly result in a ± 0.01 g/cm^2 change in BMD, below the BMD matching criterion. All controls had a glomerular filtration rate > 60 mL/min. The disorders potentially affecting musculo-skeletal system in the control group were the following (*n* [%]): primary hyperparathyroidism (14 [14.3%]), pituitary failure (8 [8.2%]), hypercortisolism (8 [8.2%]), acromegaly (3 [3.1%]), Turner syndrome (1 [1%]), Klinefelter syndrome (1 [1%]). Thirty-eight [38.7%] subjects were postmenopausal and 25 [25.5%]) had no significant comorbidities. Four (4.1%) control subjects had previous treatments with glucocorticoids (at least 3 months with a dose of at least 5 mg daily prednisolone equivalent) and seven (7.1%) had previous or current bisphosphonates treatment. Controls' characteristics can be found in Table 1.

Bone mineral density and trabecular bone score

BMD was measured at the LS, femoral neck (FN), and 1/3 radius in all patients and at the LS and FN in all controls using a General Electric Prodigy Lunar DXA (enCore Software 10,50,086). In all HD patients, the DXA scan was performed in the morning following the dialysis session. BMD was expressed as grams per square centimeter (g/cm^2) and T and Z scores were expressed in standard deviations (SD) and calculated using manufacturer's database.

TBS was calculated from the same LS DXA scan as BMD using TBS iNsight Software (version 2.2.0; Medimaps, Geneva, Switzerland).

Fracture risk was calculated using the FRAX tool for Romania in individuals over 40 years of age without a history of anti-osteoporotic treatments (93 HD patients and 86 controls). In patients (7 [7.2%]) and controls (4 [4.6%]) with previous glucocorticoid treatments FRAX was calculated with adjustment for glucocorticoid use. The fracture risk was afterwards adjusted for TBS.

Statistical analysis

All variables are expressed as mean ± standard deviation (SD) with the exception of fracture risk which is expressed as median (25, 75 percentile). Differences (95% confidence interval [95% CI]) between two means were calculated using Student's *t*-test. Correlation coefficients between two variables were calculated using Pearson's method. All statistic testing was carried out using MedCalc Software version 14.8.1 (Ostend, Belgium).

Table 1 Patients' and controls' matched characteristics

	Hemodialysis	Controls	Difference (95% CI)	*p* value
Males, *n* (%)	42 (42.8%)	42 (42.8%)		
Age (years)	57.5 ± 11.3	58.3 ± 11.5	0.8 (− 2.3 to 4.0)	0.6
Height (cm)	166.5 ± 9.2	163.9 ± 10.1	− 2.56 (− 5.3 to 0.1)	0.06
Weight (kg)	74.2 ± 14.2	74.7 ± 14.3	0.46 (− 3.5 to 4.4)	0.82
BMI (kg/m^2)	26.9 ± 4.9	27.6 ± 5.0	0.8 (− 0.6 to 2.2)	0.26
Dialysis vintage (years)	5.5 ± 3.8	NA		
LS				
BMD (g/cm^2)	1.029 ± 0.179	1.029 ± 0.179	0.0004 (− 0.05 to 0.05)	0.98
T-score (SD)	− 1.3 ± 1.4	− 1.3 ± 1.4	0.04 (− 0.3 to 0.4)	0.83
Z-score (SD)	− 0.7 ± 1.3	− 0.6 ± 1.3	0.1 (− 0.2 to 0.4)	0.58
TBS	1.193 ± 0.142	1.265 ± 0.135	0.072 (0.03–0.11)	< 0.001

Data are presented as mean ± SD, number (percentage) or as difference (95% CI) between controls and hemodialysis

Abbreviations: *BMD*, bone mineral density; *BMI*, body mass index; *CI*, confidence interval; *LS*, lumbar spine; *SD*, standard deviation; *TBS*, trabecular bone score

Results

Trabecular bone score

Hemodialysis patients had significantly lower LS TBS (0.07 [95% CI 0.03–0.1]; $p = 0.0004$) (Fig. 1a), TBS *T*-score (0.83 SD [95% CI 0.42–1.24]; $p = 0.0001$)) (Fig. 1b) and TBS *Z*-score (0.81 SD [95% CI 0.41–1.20]; $p = 0.0001$) (Fig. 1c) than age-, sex- and LS BMD-matched controls. As expected, TBS significantly correlated with LS BMD (Fig. 2a) and FN BMD (Fig. 2b) in both HD patients ($r = 0.382$; $p = 0.001$ and 0.40; $p = <0.0001$ respectively) and controls ($r = 0.36$; $p = 0.002$ and $r = 0.245$; $p = 0.01$ respectively). The two regression lines between LS BMD and TBS in HD patients and controls had similar slopes (0.3 vs. 0.28; $p = 0.84$) with different intercepts (0.88 vs. 0.98). Also, TBS correlated significantly with 1/3 radius BMD in HD patients ($r = 0.328$; $p = 0.001$). TBS *Z*-score did not correlate with dialysis vintage ($r = -0.08$; $p = 0.4$) or BMI ($r = 0.15$; $p = 0.15$). All results were not significantly altered after the removal of patients and controls with glucocorticoids or anti-osteoporotic treatments.

Femoral neck bone mineral density

Hemodialysis patients had significantly lower FN BMD (0.061 [95% CI 0.022–0.1]; $p = 0.002$) and FN BMD *T*-score (0.44 [95% CI 0.16–0.72]; $p = 0.001$) than age-, sex- and LS BMD-matched controls. Also, FN BMD correlated significantly with LS BMD in both HD patients ($r = 0.70$; $p < 0.0001$) and controls ($r = 0.67$; $p < 0.0001$).

Fracture risk

The absolute 10-year fracture risk was significantly higher in patients than in controls for both major osteoporotic fracture and hip fracture. Adjustment for TBS significantly increased the absolute 10-year fracture risk in HD patients, including those over 60 years of age (see Table 2). Out of the 93 HD patients, the absolute 10-years major osteoporotic fracture risk was increased in 83 (89.2%), decreased in 5 (5.3%) and was left unchanged in 5 (5.3%) patients after TBS adjustment. The absolute 10-years hip fracture risk was increased in 68 (73.1%), decreased in 13 (13.9%) and was left unchanged in 12 (12.9%) patients after TBS adjustment.

Discussion

Our study showed that HD patients have significantly lower LS TBS and FN BMD than sex-, age- and LS BMD-matched controls. To our knowledge, this is the first study that shows a lower TBS in HD patients, independent of BMD. Also, we show that the magnitude of TBS reduction in HD patients is constant at any LS BMD.

TBS was showed to be reduced in HD patients compared with controls [11] but this could be explained by the lower BMD associated to chronic HD. By controlling for BMD, we demonstrated a 0.07 reduction of TBS independent of BMD. As in other studies [11–14], we found a significant correlation between TBS and BMD. Our coefficient of correlation between TBS and LS BMD in HD patients ($r = 0.382$) was similar to previous studies: 0.5 in the study of Luckman [13] or 0.338 in the study of Aleksova [14]. We also found a significant correlation between TBS and FN BMD ($r = 0.40$),

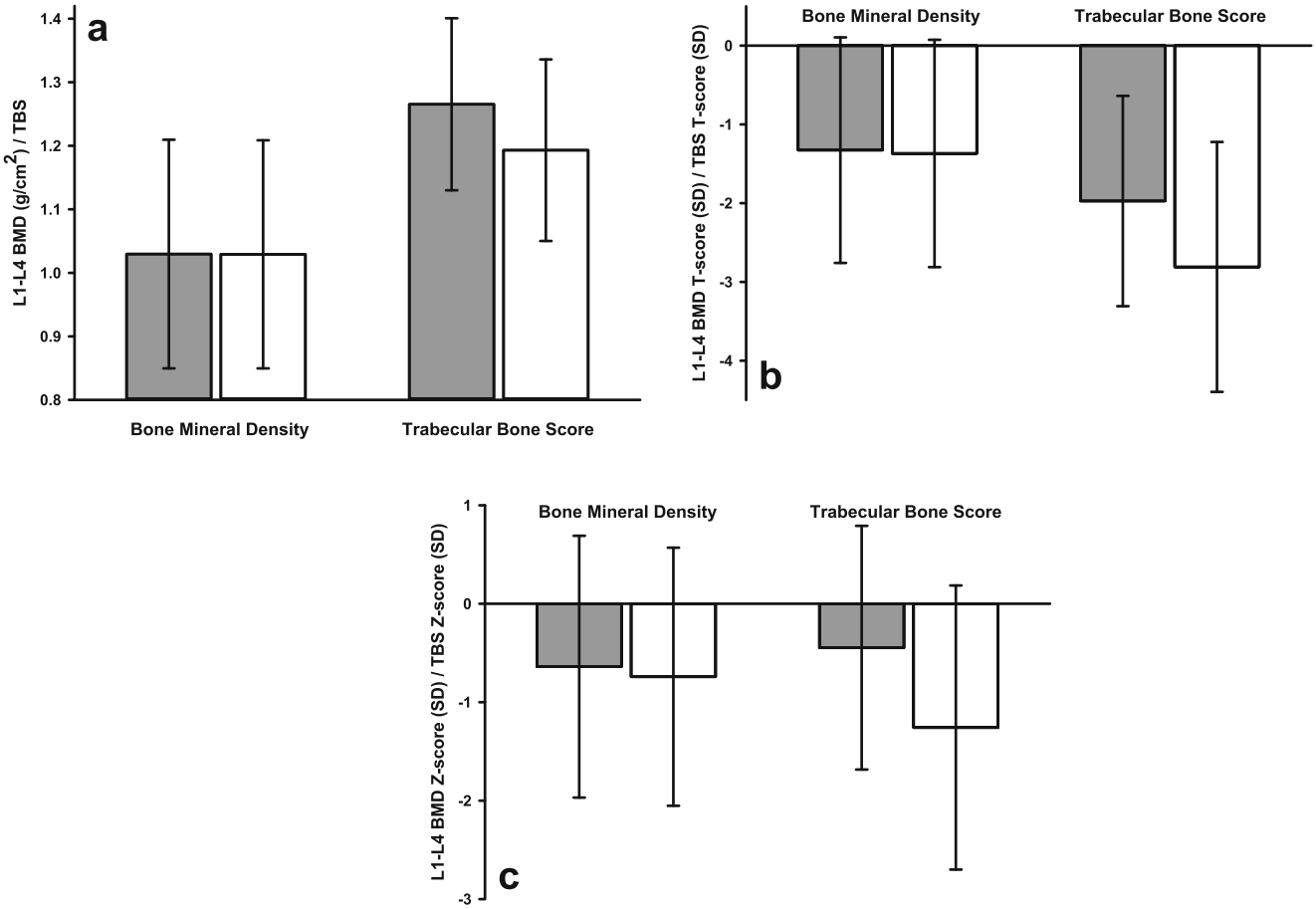

Fig. 1 Trabecular bone score (TBS) (**a**), TBS *T*-score (**b**), and TBS *Z*-score (**c**) in HD patients (white bars) compared with age-, sex- and BMD-matched controls (gray bars). *P* value > 0.5 for all BMD comparisons, *p* value < 0.001 for all TBS comparisons

similar to 0.412 in the study of Yavropoulou [11]. However, other studies found a correlation coefficient in the range of 0.028 [14] to 0.14 [13] without reaching statistical significance. It is noteworthy to highlight the fact that 50% of our control subjects had endocrine disorders associated with low BMD and TBS, so the difference in TBS between HD patients and general population could be even higher.

Another interesting finding was the lower BMD at the FN for HD patients compared with controls matched for LS BMD. Thus, the FN BMD could be more affected than LS by the disturbed endocrine milieu of ESRD [16, 17], explaining the increased fracture rate at the proximal femur.

Low TBS has been shown to predict fracture, both hip and vertebral, in the general population [9]. Thus, lower TBS of HD patients compared with BMD-matched controls, could partially explain the increased fracture rate in HD patients. LS TBS might be a measure of globally deteriorated bone architecture but at present it is difficult to extrapolate this for the femur as there is no femoral TBS. The additional fracture risk, if any, induced by lower TBS in ESRD population, above that posed by BMD, cannot be calculated at this moment.

One possible explanation of the lower TBS in HD patients, independent of BMD, is the deteriorated bone architecture characteristic of ESRD [18]. Although most of the studies involved peripheral skeleton through the use of high resolution peripheral quantitative computed tomography, it is expected that architectural changes involve the whole skeleton. Thus, TBS measured at the LS level would be just the clinical tool needed to assess non-invasively bone quality. We definitely cannot exclude the possibility of an artificially increased LS BMD in HD patients. Vascular calcifications, so prevalent in HD patients, and spine osteoarthritis could account for this increase at the LS level but not at FN or 1/3 radius [19, 20]. If the actual LS BMD is lower than measured than the control group is not correctly matched. In this case, the lower TBS in HD compared to control subjects would be just a measure of lower BMD and not of a deteriorated bone microarchitecture. However, the similar correlation of TBS with both LS and FN BMD in HD patients and controls suggest that the potential artificial increase in LS BMD is not clinically significant. Also, LS BMD correlated significantly with FN BMD, with a similar correlation coefficient in HD patients and controls.

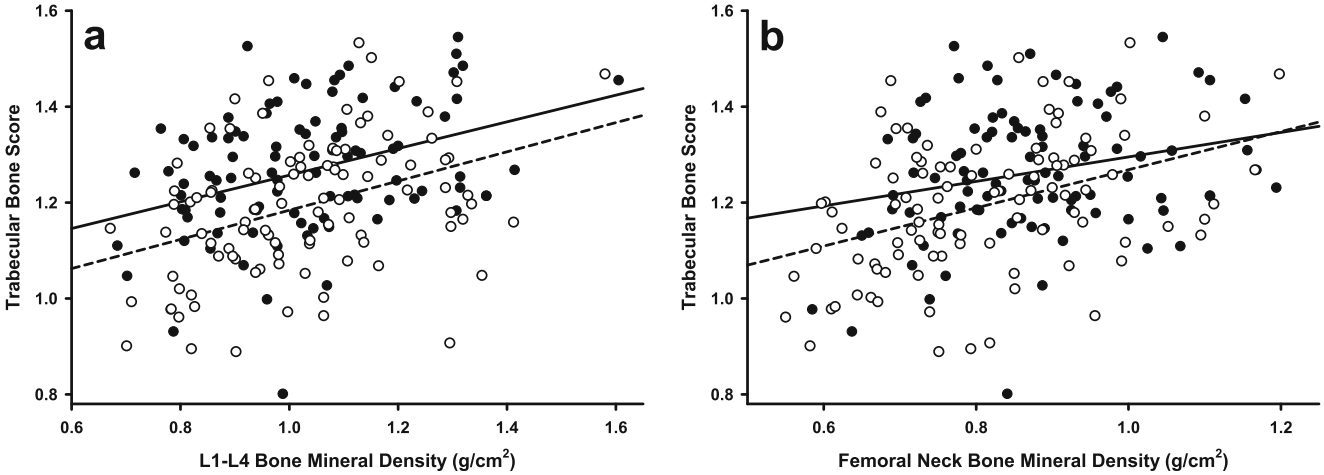

Fig. 2 Trabecular bone score (TBS) correlated significantly with LS BMD (**a**) and FN BMD (**b**) in both HD patients (white circles, dashed regression line) and controls (black circles, solid regression line)

Moreover, TBS is not affected by lumbar spine osteoarthritis [21]. BMD and TBS results could be impacted by the timing of DXA scan relative to dialysis session through the variable water content of soft tissue. However, a previous study in HD patients showed no significant differences in BMD before and after a dialysis session [22]. If a small effect is still present, excess extracellular water would rather underestimate BMD [23] in HD patients. To our knowledge no study addressed the potential effect of extracellular water on TBS.

The absolute 10-year major and hip osteoporotic fracture risk was significantly increased in HD patients compared with controls due to lower femoral neck BMD. However, this risk (0.9% for hip fracture) is much smaller than previously reported in incidence studies (> 10%) [24], a well-known underperformance of areal BMD in HD patients. Although the FRAX-based fracture risk was significantly increased after TBS adjustment to 5.3% for major osteoporotic fracture and to 1.5% for hip fracture it still underestimates the real fracture risk of HD patients [24].

The main limitation of our study is that we controlled for LS BMD but not for FN BMD. Controlling at the femoral level would eliminate the potential artificially increased BMD at the LS and also suggest any deteriorated bone architecture at the main fracture site in HD patients. However, controlling simultaneously for both LS and FN BMD is challenging as many ESRD patients are young and have very low BMD. Controlling only for LS BMD allowed us to enroll a significant number of patients and to measure bone architecture at the same site as BMD.

Another limitation of our study is that 35.7% of our control subjects had an endocrine disorder (primary hyperparathyroidism, Cushing syndrome, etc.) with important effects on bone mass and architecture. This is due to the large number of hemodialysis patients with young age and low BMD for whom healthy controls cannot be found. However, it is interestingly to note that HD patients have lower TBS even compared with patients with severe endocrine disorders. Also, some endocrine disorders from the control group may have different effects on trabecular or cortical bone. This may alter the correlation between FN BMD and LS TBS in control subjects. However, the lower LS TBS in HD than in control subjects remains significant as the controls were all matched for BMD at the same site (lumbar spine) were TBS is measured.

The strength of our study is the assessment of TBS in patients and controls with the same LS BMD. This allowed

Table 2 Absolute 10-year fracture risk before and after TBS adjustment

	Absolute 10-year major osteoporotic fracture risk		Absolute 10-year hip fracture risk	
	Unadjusted	Adjusted for TBS	Unadjusted	Adjusted for TBS
HD patients ($n = 93$)	3.7 (2.4, 6.1)*	5.3 (3.3, 8.2)#	0.9 (0.3, 2.1)*	1.5 (0.5, 3.2)#
HD patients ≥ 60 years of age ($n = 50$)	4.7 (3.5, 6.5)	6.5 (4.2, 9.1)#	1.7 (0.5, 2.6)	2.3 (0.8, 3.9)#
Controls ($n = 86$)	3.4 (2.1, 4.4)	4.1 (2.9, 5.1)	0.6 (0.2, 1.1)	0.7 (0.2, 1.3)

Data are presented as median (25, 75 percentile

*$p < 0.01$ vs. controls; #$p < 0.01$ vs. unadjusted HD patients

Abbreviations: *HD*, hemodialysis; *TBS*, trabecular bone score

us to describe an effect of chronic HD on bone quality, independent of bone quantity.

In conclusion, our study demonstrates that HD patients have an altered bone architecture, measured by LS TBS, independent of BMD. Adjustment for TBS partially corrects the absolute 10-year fracture risk.

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Compliance with ethical standards

The study was approved by the Institutional Ethic Committee. All patients signed an informed consent.

Conflicts of interest None.

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