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Chapter

TBS (Trabecular Bone Score) Used for Evaluation of Patients with Primary and Secondary Hyperparathyroidism

Lívia Marcela dos Santos and Bruno Marcos Mazoca Orozco

Abstract

The increase in parathyroid hormone (PTH) is related to a worse quality of bone mass. Bone densitometry, as an areal bone measurement, is not always able to assess bone microarchitecture. TBS (trabecular bone score) is a software that evaluates bone microarchitecture from the image of the lumbar spine obtained by bone densitometry. The articles have shown an excellent correlation of TBS with the risk of fragility fracture, especially in the individual who has primary hyperparathyroidism. We suggest that TBS may be an excellent method for assessing bone fragility in patients with hyperparathyroidism, especially if TBS is associated with bone densitometry.

Keywords: trabecular bone score, hyperparathyroidism

1. Introduction

TBS (trabecular bone score) is a parameter that indirectly measures bone microarchitecture, which is a limitation of DXA (dual-energy X-ray absorptiometry) [1, 2]. This assessment of bone microarchitecture is performed by the grayscale measurement of the lumbar spine image performed by DXA. TBS has the ability to assess the three-dimensional microarchitecture in an image obtained by two-dimensional DXA [3, 4]. The correlation that TBS presents with the parameters of computed microtomography proves its effectiveness. TBS shows a positive correlation with trabecular bone volume (BV) to tissue volume (TV) ratio (BV-to-TV), number of trabeculae, and their connectivity and stiffness; it has negative correlations with the space between trabecular and with the structure model index, measurement of rods and plates in trabecular bone [5, 6].

In a study involving 123 women and men, negative associations were found between TBS, structure model index (adjusted R2 = 69.1%), and space between trabeculae (adjusted R2 = 68.4%), positive associations were also observed between TBS and the number of trabeculae (adjusted R2 = 79.5%), and BV-to-TV (adjusted R2 = 0.830). The adjustments performed in this study were age, LS-BMD, sex, and anterior vertebral fracture (AVF) [7].

The classification of TBS is made: TBS greater than 1.310, reflects a normal bone microarchitecture, TBS between 1310 and 1230 reflects a moderately compromised bone microarchitecture, and TBS less than 1230 reflects a compromised bone microarchitecture, correlating with fragility fracture risk. In addition, it is important to remember that TBS is limited to women younger than 50 years and also with a body mass index (BMI) less than 15 and greater than 37 kg/m^2.

In 2015 the ISCD (International Society for Clinical Densitometry) officially positioned itself about TBS, with the following guidelines:

a. TBS is associated with the risk of vertebral, hip, and major osteoporotic fractures in postmenopausal women.

b. TBS can be associated with FRAX and with bone densitometry in the prediction of fractures in postmenopausal women and in elderly men [8].

2. Limitations of bone densitometry

The diagnosis of osteoporosis is currently based on bone mineral density (BMD) assessed by DXA. DXA evaluates the lumbar spine (L1-L4 vertebrae), femoral neck, total femur and radius sites. The data provided by the densitometer are in g/cm2, through these data we can compare the individual with the young adult population. The number of standard deviations coming from this evaluation generates the T-Score. The Z-Score evaluates the standard deviations comparing the individual to the population of the same age.

Through this analysis we classify the T-Score, based on the areal measurement of BMD:

- If the T-Score < −2.5 in any densitometric site is analyzed: We have a diagnosis of osteoporosis.

- A T-Score value <−2.5 is associated with a high risk of fragility fractures.

Osteopenia was classified as T-Score between 1.1 and 2.4, and the risk of fragility fracture is lower, but still exists. In addition, we have a higher absolute number of women with osteopenia compared to women with osteoporosis. Therefore, TBS is essential to distinguish women with osteopenia and increased fracture risk, because they have a degraded bone microarchitecture.

However, DXA offers some limitations, mainly because it makes a two-dimensional measurement, thus considers bone density in an areal manner, and does not have the ability to measure bone microarchitecture. Thus, when evaluating the lumbar spine image captured by DXA the TBS is able to add important information. Some studies have evaluated the ability of TBS to predict osteoporotic fractures and it has been shown to be an accurate predictor of fractures [9–12].

Thereby DXA may be an insufficient tool for determining bone strength and whether there is a satisfactory drug response for osteoporosis, mainly because it fails to measure bone microarchitecture [13–17].

TBS is a software that is easily applied in clinical practice, that is after the patient has bone densitometry the TBS can be analyzed, no additional examination is

required, it is also not an invasive examination or one that will expose the patient to further radiation [4].

3. The importance of TBS (trabecular bone score) in bone fracture

With the increase in life expectancy, calcium metabolism pathologies are progressively increasing, among them the most common, osteoporosis, but also primary hyperparathyroidism (PHPT) and secondary hyperparathyroidism (SHPT). Thereby, new technologies have been emerging to improve the assessment of fragility bone fracture risk.

There is no consensus on the best tool to predict fragility fractures, but without a doubt, TBS can contribute to the early diagnosis of a patient's fracture risk. TBS can be used in association with DXA, that is, we can evaluate the T-Score of bone densitometry and the absolute value of TBS. Another way to use TBS is in association with FRAX (Fracture Risk Assessment Tool).

FRAX is a tool that calculates the risk of major and minor fractures in 10 years. The use is simple, it is a digital questionnaire, which the doctor will fill in age, sex, weight, height, if the individual has had a previous fracture, if his parents had a hip fracture, if he is a smoker, if he takes glucocorticoids chronically, if he is diagnosed with rheumatoid arthritis, if he is diagnosed with secondary osteoporosis, and if he uses alcohol more than 3 times a week. Then the doctor adds the BMD value of the patient's femoral neck. It is considered that the patient should be treated if the FRAX shows more than a 3% chance of hip fracture or more than 20% of osteoporosis-related fractures in 10 years.

Since the FRAX is a tool that estimates the risk of fragility fractures, it can also be adjusted by the TBS showing greater sensitivity to diagnose fractures. A study conducted in the province of Manitoba involving over 3000 men, with a mean age of 67 years, found that the TBS-adjusted FRAX was able to predict major osteoporotic fractures and hip fractures [18]. The FRAX is not an algorithm that can perform follow-up as it has few modifiable characteristics. However, FRAX associated with TBS can be a follow-up tool, especially for individuals who have rheumatoid arthritis and fracture risk [19]. Thus the online FRAX has an adjustment with the TBS option [20].

We can consider that the association of data such as FRAX, DXA, and TBS increases the chance of predicting vertebral and hip fracture.

4. Parathyroid hormone (PTH) and bone metabolism

Excess parathyroid hormone (PTH), a characteristic present in all forms of hyperparathyroidism, is related to decreased bone mass and increased risk of fractures.

PTH, a protein hormone secreted by the parathyroid glands, has an action on bone tissue, on the kidneys, and indirectly on the intestine. Its action in bone tissue depends mainly on PTH receptors, which have been located in osteoblasts and pluripotent progenitor cells of mesenchymal lineage, but these receptors have not been identified in osteoclasts. We know that bone remodeling is a coupled process, that is, continuous stimulation of osteoblasts by excess PTH can generate increased bone resorption.

We know that this hormone stimulation on osteoblast can indirectly activate osteoclast through osteoprotegerin (OPG) has the ability to bind to the membrane receptor (RANK) on hematopoietic progenitor cells inducing differentiation into osteoclasts, and also stimulates the production of interleukin −6 which has a role in stimulating the production and activation of osteoclast formation [21].

Primary hyperparathyroidism (PHPT) is a frequent endocrine disease consisting of hypercalcemia and elevated or inappropriately normal PTH levels. With the ease of ordering laboratory tests, especially in the last three decades, the clinical presentation of PHPT has changed from symptomatic with frequent cystic fibrous osteitis to symptomatic and asymptomatic forms of PHPT.

The classic bone clinical presentation of this syndrome is subperiosteal bone resorption, brown tumors, generalized cystic fibrous osteitis, fragility fractures, and osteopenia or osteoporosis. The bone manifestation is related to the degree of hypercalcemia presented. So, the more severe hypercalcemia, the highest degradation of bone mass. Although the most important involvement is in cortical bone, which is located in the hip, the trabecular bone is also affected, seen densitometrically by the lumbar spine [22].

A double-blind study looked at 30 patients with PHPT confirmed by surgery and pathologically confirmed with MRI or CT scans. All had osteopenia, 60% bone resorption, 40% subperiosteal resorption, and more than 30% cortical bone resorption, and subchondral resorption. There were 19 (63.3%) cases with cystic fibrous osteitis/brown tumor. There were 5 (16.7%) patients who had a pathological fracture. The skeletal disease of HPTP should be differentiated from the osteolytic metastatic bone tumor, fibrous osteodysplasia, giant cell bone tumor, and aneurysmal bone cysts [23].

5. Why should we use the trabecular bone score (TBS) as an evaluative parameter in primary hyperparathyroidism (PHPT)?

In primary hyperparathyroidism we have a decrease in bone mass, increasing the risk of vertebral and nonvertebral fractures in these individuals, and the bone mass is classically analyzed by bone densitometry. The sites analyzed in densitometry are the lumbar spine (first to fourth lumbar vertebrae), the hip (involving the femoral neck and total femur), and the distal radius.

The definitive treatment for PHPT is surgery of the affected gland, but not all patients are chosen for surgery, there are well-defined criteria. One of these criteria involves the evaluation of bone mass, that is, having a diagnosis of osteoporosis by bone densitometry. But many individuals with PHPT who have a fragility fracture only have osteopenia. Therefore, other bone mass assessment techniques can be associated with densitometry to predict fragility fractures in this population, such as TBS. Furthermore, it was evidenced that densitometry might not predict vertebral fracture risk because probably the damage in trabecular bone was more in bone microarchitecture; TBS proposes to quantify bone microarchitecture indirectly [22, 24–26].

In individuals with HPTP, the TBS is able to assess bone microarchitecture and predict fracture risk [27]. One study evaluated the significant correlation between TSB and parameters measured on HRpQCT [28].

In a study conducted in Brazil in 2021, we have the analysis of 64 individuals with primary hyperparathyroidism before parathyroidectomy, an analysis of vertebral fragility fracture was performed and TBS was able to predict this type of fracture, but

DXA could not. Also, in that study, we have that most the vertebral fractures happened in patients with osteopenia [29]. Other articles also demonstrate the ability of TBS to help predict fragility fracture in individuals with PHPT [30].

In a study that evaluated over 150 patients with PTH, with only 10% being male, means age 59 years, mean BMI 26.2 ± 4.8 kg/m^2, 89% had a diagnosis of osteoporosis/osteopenia by LS-DXA. In the patients analyzed the fracture distribution was: 7.6% with vertebral fractures, and 13.2% with non-vertebral fractures. The mean TBS was in the partially degraded range (1.258 ± 0.115); 32% of patients had degraded microarchitecture (TBS ≤ 1.20), 51% had partially degraded microarchitecture (TBS > 1.20 and <1.35) and 17% had normal TBS. TBS correlated positively with BMD in the lumbar spine and femoral neck, and negatively with age and years since menopause. Patients with vertebral fractures had mean TBS values in the degraded range, significantly lower than those without vertebral fractures. Only 9.7% of patients with degraded or partially degraded TBS microarchitecture had normal lumbar spine T-Score scores, none with vertebral fractures [27].

Obesity has been shown to be a limiting factor in the analysis of skeletal microarchitecture when assessing TBS. The influence of adiposity in the abdominal region on skeletal microstructure in primary hyperparathyroidism has not yet been well evaluated by studies. An observational study evaluated the effect of obesity on TBS and bone mineral density (BMD) in individuals with PHPT at baseline and up to 2 years after parathyroidectomy. The study participants consisted of 30 men and women with this disease, undergoing parathyroid surgery. There were notable improvements in BMD of the lumbar spine and femoral neck in the obese and non-obese individuals, but no difference in TBS values in both groups at 24 months post-parathyroidectomy. Obese individuals had more degraded TBS values compared to non-obese individuals. In this study, obesity was associated with more degraded skeletal microarchitecture as measured by TBS in PHPT, despite similar values in bone density by DXA compared to non-obese subjects. TBS values did not improve post-parathyroidectomy in obese or non-obese subjects [31].

In a cohort of symptomatic patients with HPTP, including postmenopausal, premenopausal, and male patients, we showed that TBS was in the partially degraded range but was not independently associated with fractures.

6. TBS and secondary hyperparathyroidism

Although TBS is well documented to predict osteoporotic fractures, little is known about secondary hyperparathyroidism. Low vitamin D levels are known to increase bone turnover and weaken bone architecture. In addition, 25(OH) vitamin D has a preferential action on cortical bone compared to trabecular bone.

An article was published on its correlation with 25 hydroxyvitamin D in Lebanese men. In this study, which involved both women and men, a correlation was seen between 25OH vitamin D and TBS. Fifty-four men and 61 women between the ages of 18 and 35 years were evaluated. Participants with 25(OH)D insufficiency (between 21 and 29 ng/mL) were 55.7%, and those with 25(OH)D deficiency (≤ 20 ng/mL) were 11.4%. TBS correlated positively with 25(OH)D in men ($r = 0.393$; $p < 0.05$) and women ($r = 0.324$; $p < 0.05$). In both sexes, TBS was significantly higher in 25(OH) D-sufficient participants (≥30 ng/mL). In this study vitamin D positively affects bone health and suggests that maintaining an adequate level of vitamin D may be essential for optimal TBS values [32].

Many metabolic diseases are reversible after kidney transplantation, however, this is not the case with bone disease. The fracture rate can be as high as 44% following kidney transplantation, with most fractures being vertebral. These fractures can occur due to decreased bone mass and impaired bone microarchitecture. Previous studies have correlated TBS with trabecular and cortical bone volume, width measured by bone biopsy, and histomorphometry in patients with chronic renal failure disease [33].

In patients with chronic kidney disease, there was a correlation between LS BMD and TBS in prerenal transplantation. Patients with tertiary hyperparathyroidism before transplantation had a lower TBS even after kidney transplantation. Baseline parathyroid status continued to impact TBS BMD at 6 months post-transplant with patients with tertiary hyperparathyroidism at baseline having the lowest TBS and BMD at that time. Although successful kidney transplantation should in theory address the etiopathogenesis of bone disease arising from preceding renal failure, our finding, as well as that of others, suggests that in reality hyperparathyroidism, when very advanced, may result in consequences that persist up to the year following transplantation [34, 35].

The only study that proposed a value for TBS that supposedly discriminated between subjects with and without fracture was conducted in a predominantly Caucasian population in Canada. In that study, as mentioned earlier, almost 90% of TBS measurements were done in the first year after kidney transplantation and the mean TBS in patients who did not have a fracture was 1.37 0(0.125) vs. 1.30 (0.144) in those who did [36].

7. Conclusion

The TBS is a tool that can be easily applied in clinical practice and adds information regarding fragility fractures and osteoporotic fractures in patients with primary hyperparathyroidism. It is also worth mentioning that other pathologies such as secondary and tertiary hyperparathyroidism and other bone health conditions are candidates to have TBS as a bone study tool since they also predispose to fragility and osteoporotic fractures.

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