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ORIGINAL ARTICLE

Association between trabecular bone score and type 2 diabetes: a quantitative update of evidence

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Abstract

Summary Patients with type 2 diabetes have an increased risk of fracture despite having a higher areal bone mineral density. This meta-analysis showed that compared with controls, diabetic patients had a lower trabecular bone score (TBS) than non-diabetic individuals, suggesting that TBS can be a useful measurement for the assessment of fracture risk in diabetic patients.

Introduction The association between type 2 diabetes and trabecular bone score (TBS) has not been clear. The present study sought to answer the specific question of whether patients with type 2 diabetes have a lower TBS than those without diabetes.

Methods Using electronic and manual search, we identified 12 studies that had examined the association between type 2 diabetes and TBS between 2013 and 2019. These studies involved 35,546 women and 4962 men aged 30 years and older. We extracted the mean and standard deviation of TBS for patients with and without diabetes. The synthesis of effect sizes was done by the random effects meta-analysis model.

Results Patients with diabetes had significantly lower TBS than those without diabetes, with standardized mean difference being − 0.31 (95% CI, − 0.45 to − 0.16). The difference was greater in women (− 0.50; 95% CI, − 0.69 to − 0.32) than in men (− 0.04; 95% CI, − 0.17 to 0.10). Compared with normal individuals, those with prediabetes had significantly lower TBS ($d = -0.13$; 95% CI, − 0.23 to − 0.04; $P = 0.005$). There was heterogeneity between the studies, with the index of inconsistency (I^2) ranging from 92% (in women) to 69.5% (in men).

Conclusion Patients with type 2 diabetes have a lower TBS than non-diabetic individuals, suggesting that TBS can be a useful measurement for the assessment of fracture risk in diabetic patients.

Keywords Bone mineral density · Meta-analysis · Trabecular bone score · Type 2 diabetes

Introduction

There is a paradox in the relationship between diabetes, bone mineral density, and fracture. In the general population, individuals with a higher bone mineral density (BMD) have a lower risk of fragility fracture, such that each standard deviation increase in BMD is associated with approximately 50% reduction in fracture risk [1]. Patients with type 2 diabetes are known to have a higher BMD, but they have a higher risk of fragility fracture. The association between diabetes and fracture is particularly apparent for hip [2] and vertebral fracture [3]. This paradoxical relationship suggests that the etiology of fracture in diabetic patients is different from that of the general population.

One of the etiologies of fracture is trabecular bone score (TBS), which can be considered an index of bone fragility. The measurement of TBS reflects the variance in trabecular bone component of the trabecular-rich lumbar spine, or a

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surrogate of bone microarchitecture [4]. Cross-section studies also found a correlation between TBS and HRpQCT [5] and MRI measures [6]. The dispersion of mineral content can be seen as a complementary measure of bone strength (in addition to bone mineral density). TBS can be derived from a lumbar spine BMD scan by determining the slope of the log-log transformation of the 2-D variogram, relating gray-level variance in dual-energy X-ray absorptiometry (DXA) images [7]. Several studies have shown that lower values of TBS were associated with an increased risk of fragility fracture [8], and more importantly, the association was independent of bone mineral density (BMD) and age [9].

Diabetes may be associated with altered bone microarchitecture. Under that hypothesis, it can be predicted that patients with type 2 diabetes have a lower TBS measurement. Indeed, several observational studies have reported that compared with those without diabetes, TBS was lower in diabetic patients [10–13]. However, a number of studies found no statistically significant association between TBS and diabetes [14, 15]. The difference in findings between studies is expected because of—among others—the differences in population characteristics, sampling strategies, and data analytic techniques. The discrepancy in findings could also be attributable to the duration of diabetes, antidiabetic medication use, and HbA1c levels. In such a scenario, meta-analysis is an attractive approach to resolve conflicting evidence. The present study was undertaken to test the hypothesis that type 2 diabetes is associated with lower TBS by synthesizing all peer-reviewed published evidence regarding the difference in TBS between diabetes and non-diabetes individuals.

Materials and methods

Search strategy and study inclusion

An electronic search of the literature was carried out using PubMed, Ovid, and ISI Web of Knowledge resources (all-year time span) to identify studies relating trabecular bone score and diabetes. The initial keywords used for the search included "trabecular bone score*" OR "TBS*" OR "osteoporosis*" OR "bone health*" concatenated with "type 2 diabetes" OR "diabetes" OR "HbA1c." In addition, we manually searched review articles and checked reference lists of original articles to identify studies that might have been missed from the electronic search. The inclusion criteria were (a) original studies published in English language journals, reporting data on trabecular bone score and diabetes; (b) observational studies; (c) using iNsight Software on lumbar spine dual-energy X-ray absorptiometry scan technology; and (d) human studies on individuals aged 20+ years. We excluded review papers, case-control and interventional studies, animal studies, and studies on children or adolescents.

Two reviewers (LHP and TN) independently identified eligible articles according to the above criteria. Discrepancies in opinion as to whether studies should be included in the analysis were resolved by discussion.

Data extraction and synthesis

Data extraction was also done independently by two reviewers. For each study, we extracted data relating to study characteristics and outcomes. Specifically, the following data were extracted: authors, journal, year of publication, study design, ethnicity, age group, gender, number of participants, and trabecular bone score (TBS) measurement. If more than one paper with the same data were identified, only the one that contained the definitive data was included. Some studies reported mean and interquartile range, and we estimated the standard deviation using the method described by Wan et al. [16]. When studies reported both unadjusted and adjusted means (i.e., least squares means), we used the adjusted means for the meta-analysis.

For each study, we computed the difference in TBS between normal and diabetes or prediabetes groups. Let m_0 and m_1 denote the mean TBS for diabetic and non-diabetic individuals, respectively; then, the standardized mean difference or effect size is defined as $d = (m_0 - m_1)/s$, where s is the common standard deviation. Thus, $d = 0$ indicates that there is no difference in TBS between the 2 groups, whereas $d < 0$ indicates that TBS in diabetic patients is lower than in individuals without diabetes.

The synthesis of d across studies was done by the random effects models [17, 18]. The National Research Council 1992 [19] considers random effects models to be more appropriate in fitting real-world data which come from populations with varying average effect sizes and they have a strong assumption about representativeness. Briefly, we calculated effect size (ES) and its standard deviation (SD) for individual studies. It is assumed that each ES is normally distributed with a "true" but unknown mean θ_i and a within-study variance σ^2. The collection of θ_i across studies is assumed to follow a normal distribution with unknown mean θ and between-study variance τ^2. The classical random effects method recognizes the possibility of heterogeneity of between-study variation (i.e., τ^2 could be different from 0) but with a fixed value. All parameters of the random effects model were estimated by the inverse variance weighting method as implemented by the "metafor" [20] within the R language [21].

The heterogeneity of correlations across studies was assessed by Cochran's Q statistic [22] and the coefficient of inconsistency (I^2). The latter is an estimate of the proportion of total variation in study estimates that is due to heterogeneity [23]. Subgroup analyses by age, gender, and ethnicity were also carried out as specified in the analysis protocol.

Table 1 Characteristics of individual studies relating trabecular bone score to diabetes

First author, year	Study design	Sex	Normal		Prediabetes		Diabetes		Adjustment for covariates	TBS software version
			n	Mean (SD)	*n*	Mean (SD)	*n*	Mean (SD)		
Leslie (2013)	CS	Women	27,051	1.245 (0.125)			2356	1.194 (0.112)	Age, BMI, diseases	GE Prodigy 1.8
Dhaliwal (2014)	CS	Women	43	1.298 (0.132)			57	1.228 (0.140)	No adjustment	GE iDXA 1.0
Kim (2015)	CS	Men	894	1.314 (0.09)			325	1.294 (0.09)	Age, BMI	GE Prodigy 2.0
		Women	1144	1.350 (0.068)			370	1.343 (0.077)	No adjustment	
Zhukouskaya (2016)	CS	Women	65	1.159 (0.150)			20	1.057 (0.180)	No adjustment	Hologic Discovery?
Bonaccorsi (2017)	CS	Women	88	1.215 (0.110)			80	1.173 (0.100)	No adjustment	Hologic Discovery 2.1
Xue (2017)	CS	Men	34	1.300 (0.130)			17	1.280 (0.120)	No adjustment	GE Prodigy 2.1
		Women	257	1.280 (0.110)			74	1.200 (0.110)	No adjustment	
Caffarelli (2017)	CS	Women	265	1.314 (0.146)			131	1.254 (0.156)	No adjustment	GE Prodigy 2.1
Wagner (2017)		Women	188	1.340 (0.107)	22	1.330 (0.109)	22	1.240 (0.087)	No adjustment	Not reported
Iki (2017)	CH	Men	1370	1.193 (0.082)			313	1.193 (0.089)	Age, BMI	Hologic QDR4500 2.1
Holloway (2018)	CS	Men	318	1.293 (0.109)	172	1.280 (0.110)	65	1.259 (0.107)	Age, height, weight	GE Prodigy 2.1
		Women	381	1.274 (0.134)	85	1.216 (0.136)	48	1.186 (0.141)	Age, height, weight	
Rianon (2018)	CS	Men	42	1.270 (0.110)			14	1.330 (0.110)	No adjustment	Hologic Discovery 2.1
		Women	59	1.240 (0.110)			38	1.170 (0.090)	No adjustment	
Ho-Pham (2019)	CS	Men	887	1.395 (0.079)	406	1.391 (0.079)	105	1.399 (0.079)	Age, BMI	Hologic Horizon 2.1
		Women	1635	1.351 (0.091)	833	1.331 (0.090)	234	1.332 (0.089)	Age, BMI	

CS, cross-sectional study; *CH*, cross-sectional study

Publication bias was examined by a funnel plot [24]. Furthermore, the radial plot (the Galbraith plot) and the standardized residual plot were used to assess asymmetry and publication bias [25].

Results

Characteristics of studies

An initial search yielded 208 articles written in English with contents relating to the TBS and diabetes. However, after excluding articles that did not meet the inclusion criteria, we identified 12 studies that had been published in peer-reviewed journals between 2013 and 2019 [10–15, 26–29], a thesis [30], including our data [31] (Table 1). The 12 studies involved 40,508 individuals (35,546 women and 4962 men) with age ranging from 30 to 92 years. Three studies were conducted on Asian populations [14, 15, 31] and 9 studies were on Caucasians [10–13, 26–30]. Six studies were conducted on women [10–12, 26, 29, 30], 1 study on men [14], and 5 studies included both men and women [13, 15, 27, 28, 31].

Most of the studies were designed as cross-sectional investigations, comparing TBS between diabetes and non-diabetes individuals. There were 3 studies [13, 30, 31] that provided data pertaining to 3 groups of individuals, namely normal, prediabetes, and diabetes. The median sample size of all studies was 389 but with a wide range (from 85 to 29,407 individuals).

TBS and diabetes

Overall, patients with diabetes had a lower TBS than those without diabetes, and the standardized mean difference ($d = -0.31$; 95% confidence interval [CI], -0.45 to -0.16) was statistically significant ($P < 0.0001$) on random effects analysis (Fig. 1). However, there was substantial heterogeneity among studies ($I^2 = 91\%$; $P < 0.001$) (Table 2).

In an attempt to explain the heterogeneity, we conducted analyses on subgroups defined by sex and diabetic groups. In women, the overall difference in TBS between diabetes and non-diabetes was -0.50 (95% CI, -0.69 to -0.32) ($P < 0.0001$). However, in men, the difference in TBS between diabetes and non-diabetes was not statistically significant ($d = -0.04$; 95% CI, -0.17 to 0.10; $P = 0.59$). There was heterogeneity between the studies, with the index of inconsistency (I^2) ranging from 55% (in men) to 82% (in women). Compared with normal individuals, those with prediabetes had significantly lower TBS ($d = -0.13$; 95% CI, -0.23 to -0.04; $P = 0.005$), and this difference was independent of sex.

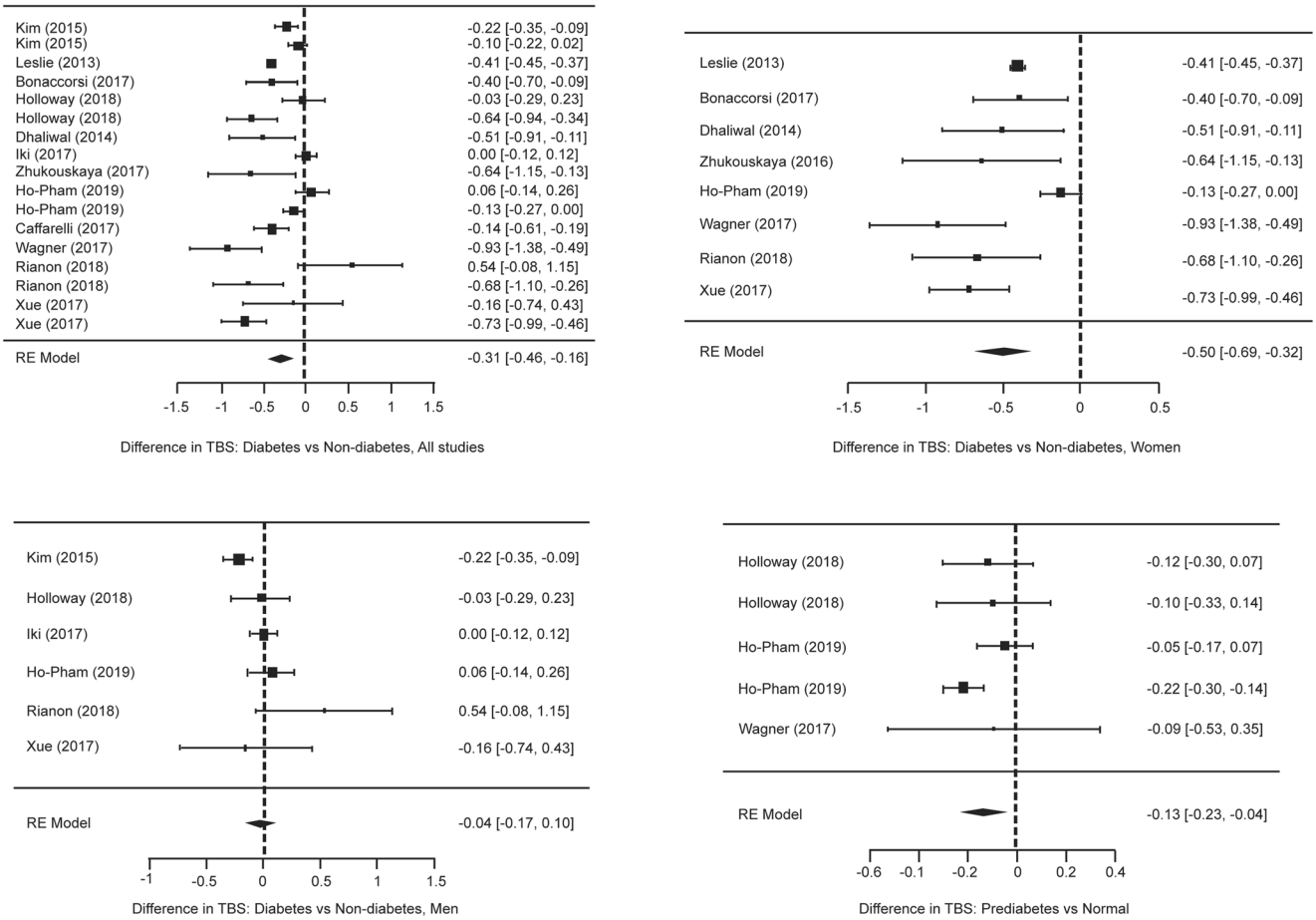

Fig. 1 Forest plot of the difference in TBS between diabetes and non-diabetes (normal and prediabetes) individuals for all studies (top left panel), women (top right panel), men (bottom left panel), and prediabetes vs normal (bottom right panel)

The evaluation of study-level correlation between mean difference and standard error (Fig. 2) showed no evidence of publication bias although it is a one small study ($n = 12$).

Discussion

Bone disease is recognized as a serious complication of diabetes, but the mechanism of this relationship has not been clear. It seems that the measurement of areal bone mineral density is inadequate for the assessment of fracture risk in patients with type 2 diabetes. It has been suggested that TBS can help predict the risk of fracture in type 2 diabetes. If this proposition is true, then diabetic patients should have a lower TBS than those without diabetes. Our result of meta-analysis is consistent with this proposition: diabetes was associated with lower TBS. There was evidence that individuals with "prediabetes" status also had a lower TBS than those with

Table 2 Summary of difference in trabecular bone score between diabetes and non-diabetes individuals

Analysis	k	Standardized mean difference (95% CI)	Index of heterogeneity (I^2)
Diabetes vs non-diabetes individuals			
All studies	12	− 0.31 (− 0.45 to − 0.16)	0.91**
Women	8	− 0.50 (− 0.69 to − 0.32)	0.82**
Men	6	− 0.04 (− 0.17 to 0.10)	0.55**
Prediabetes vs normal individuals			
All studies	3	− 0.13 (− 0.23 to − 0.04)	0.49**

k = number of studies

**Denotes statistically significant at $P < 0.01$

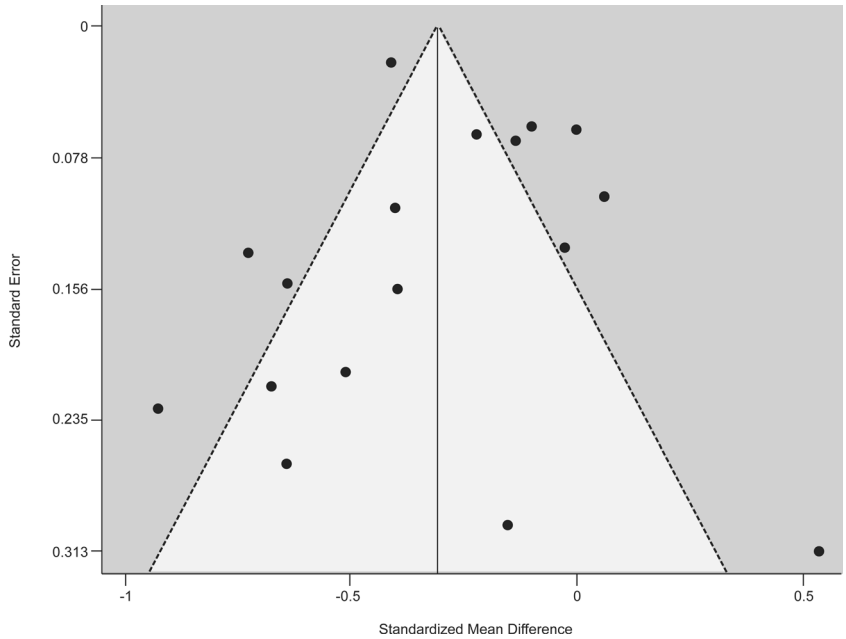

Fig. 2 Funnel plot of standardized mean difference versus standard error

normal glucose levels. These results deserve further elaboration.

The effect size we observed in this analysis was modest. On average, the difference in TBS between diabetic patients and non-diabetic individuals was − 0.04, or equivalent to − 0.31 standard deviation. Each standard deviation decrease/lower in TBS is associated with a 1.4-fold increase in fracture risk [32]. Thus, ~ 0.30 standard deviation decrease in TBS is expected to increase the risk of fracture by ~ 11%. In an empirical study, each standard deviation lower in TBS was associated with a 1.27-fold (95% confidence interval, 1.10 to 1.46) increase in the risk of fragility fracture in women with type 2 diabetes [10]. In a recent meta-analysis [33], it was found that diabetes was associated with a 32% increase in the risk of total fracture. Thus, the difference in TBS between diabetes and non-diabetes cannot completely explain the increased risk of fracture among diabetic patients. Moreover, we and others [34–36] have shown that diabetic patients have a lower cortical volumetric BMD (and hence a lower bone strength [37]) which might explain why they have a greater risk of fracture at cortical-rich bone sites. Taken together, the higher fracture risk in diabetic patients is due to multifactorial factors, including deterioration of trabecular bone and reduced bone strength.

The mechanism of the association between TBS and type 2 diabetes is not clear. However, it can be hypothesized that the association may be mediated by advanced glycation end (AGE) product. Advancing age is associated with the accumulation of AGE in the bone matrix, leading to increased bone fragility [38]. Patients with type 2 diabetes have a higher accumulation of pentosidine, a product of AGE [39]. Moreover, in a recent study on patients with type 2 diabetes, a correlation between TBS and pentosidine was observed [40]. Thus, AGE may be a mediator in the relationship between TBS and type 2 diabetes, but this hypothesis needs to be tested in a well-characterized prospective study.

The association between diabetes and TBS was observed in both women and men. However, the effect size was greater in women (0.5 standard deviation) than in men (0.04 standard deviation). A recent large-scale study in men ≥ 65 years found no significant association between type 2 diabetes and vertebral fracture [41]. Taken together, the evidence so far appears to suggest that the relationship between fracture and diabetes was more pronounced in women than in men. Why there was a difference in the TBS-diabetes relationship between men and women? We postulate that the difference could be due to the effect of degenerative changes on the DXA measurement of bone density in the lumbar spine [42]. Degenerative changes in the lumbar spine obscure the age-related change in lumbar spine BMD in men. Because TBS is derived from DXA spinal scan, its measured value is also likely to be affected by degenerative changes, and this could explain why the association between TBS and diabetes in men is not apparent.

The effect size observed in this study should be interpreted in relation to statistical adjustment. TBS is known to be statistically related to body mass index and age. However, most studies did not adjust the difference between diabetes and non-diabetes for these covariates. In some studies, the observed difference in TBS between diabetes and non-diabetes was statistically insignificant after adjusting for age and body mass index. In those studies that did adjust for age and/or body mass index, we used the adjusted mean rather than the unadjusted mean.

As with any meta-analysis, the exclusion of pertinent unpublished studies represents a threat to the validity of the result. However, in this analysis, we found no evidence of systematic publication bias by all methods (funnel, radial, and standardized residual histogram). Nevertheless, there was significant heterogeneity among the studies included in the analysis, and we dealt with this problem by a random effects analysis and subgroup analysis [19]. The heterogeneity could be due to the discrepancy of sample sizes and measurement of variables which were not the case in the random effects analysis of ref. [43]. Another threat of validity is that the association between TBS and glucose levels might not be linear. Different versions of TBS software could also introduce bias into the study, but this is unlikely as we used the standardized difference as a measure of effect size which presumably controlled for difference in measurements. We could not assess whether the association between type 2 diabetes and TBS is due to the physiology and duration of the disease or antidiabetes medications. Finally, all studies included in the analysis were cross-sectional, and no causal inference could be drawn between TBS and diabetes.

In conclusion, we have shown that patients with type 2 diabetes have a lower trabecular bone score than those without diabetes or normal glucose levels. However, the magnitude of association was more pronounced in women than in men. These findings suggest that trabecular bone score may be a useful measurement for the assessment of fracture risk in patients with type 2 diabetes.

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Compliance with ethical standards

Conflicts of interest None.

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