Bone mineral density in type 2 diabetes mellitus determined by measurement of body mass index/handgrip strength ratio in a cross sectional study

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ABSTRACT

Background: Diabetes mellitus (DM) causes substantial morbidity and death in most organs. Osteoporosis is a metabolic bone disease. Type 2 DM is associated to osteoporosis and muscular weakness (T2DM). However, research on type 2 diabetes and its impact on BMD and handgrip strength is sparse (HGS).

Methods: The research involved 130 people aged 25-60. This is the Quetelet index. HGS was measured using a handgrip dynamometer. HGS max kg and ET in seconds were recorded. A bone sonometer measured BMD at the tibia's distal end. The T-score and Z-score were used to examine the results.

Results: In diabetics, BMI correlated with BMD and HGS max (P = 0.032). BMD correlated weakly with HGS max and ET. Non-diabetic men had greater HGS, whereas non-diabetic females had higher HGS and ET (P <0.002). T2DM and non-diabetics had similar BMD. Conclusion: In our study, we noticed superior muscular strength among non-diabetics and no significant difference in BMD between diabetics and non-diabetics, but incidence of osteoporosis was larger among diabetics albeit statistically not significant. Keywords: Type 2 Diabetes Mellitus; Bone Mineral Density; Body Mass Index; Handgrip Strength; Endurance time

INTRODUCTION

Diabetes mellitus (DM) is a serious health issue in Pakistan as well. DM affects most organ systems, causing significant morbidity and death. Osteoporosis is a frequent metabolic bone disease among the elderly. Previous research have consistently shown that Type 1 diabetes reduces bone mineral density (BMD), but Type 2 diabetes (T2DM) has variable outcomes, being lowered, improved, or similar values to healthy controls. Inconsistent outcomes may be attributable to research design, BMD measuring techniques, location, patient selection, and complication occurrence or absence.

T2DM has been connected to musculoskeletal syndrome muscle weakness. Diabetics have significantly reduced grip strength. Handgrip strength predicts future fractures independently of BMD. It is an objective exam for assessing fall and fracture risk. T2DM is characterised by obesity and hyperinsulinemia. However, research on T2DM and its impact on BMD and HGS is sparse. Our study's main goal is to better understand the link between T2 DM, BMI, BMD, and HGS.Even with normal or increasing BMD, T2DM patients are more prone to fragility fractures. Limited evidence links DM to faster bone loss. Age is also a risk factor for bone loss and osteoporosis, and DM is known to disrupt bone mineral metabolism. Our country underestimates metabolic bone disease owing to ignorance. Early detection of bone loss in diabetes individuals may help avoid future fracture risk.T2DM has been connected to musculoskeletal syndrome muscle weakness. Diabetics have significantly reduced grip strength. Handgrip strength predicts future fractures independently of BMD. It is an objective exam for assessing fall and fracture risk.T2DM is characterised by obesity and hyperinsulinemia. Earlier research linked BMI to BMD.

However, research on T2DM and its impact on BMD and HGS is sparse. Our study's main goal is to better understand the link between T2 DM, BMI, BMD, and HGS.

MATERIALS AND METHODS

Our study comprised 130 people aged 30-70, both males and females. This is a cross-sectional research done with informed permission from BMD camp and diabetes clinic participants. Our trial lasted 6 months. The research covered all known T2DM clinic participants. The research excluded hypertensives, drinkers, smokers, bedridden/fracture patients, and those using medicines that alter bone mineral metabolism. The height of the subjects was measured against a wall, barefoot, with a tape measure. Weighed in Kg wearing light indoor attire. BMI (kg/m2) = wt (kg)/Ht (m2). The subjects were then divided into three groups based on their BMI: normal weight (19-25 kg/m2), overweight (25-30 kg/m2), and obese (31 kg/m2).

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HGS was measured with a handgrip dynamometer. HGS is the maximum force of all finger flexion. Participants were sitting with their elbows by their sides, flexed at right angles, and their wrists neutral, with support below the dynamometer. In this position, the participant must fully compress the HGS dynamometer. The amount of static force that the hand can compress/squeeze around a dynamometer measures HGS. Take the mean of three grip strength tests. Endurance time (ET) is defined as the time taken to reach 70% of T max in seconds

The Sunlight MiniOmniTM bone sonometer-Adults Configuration measures BMD in the tibia distal end over the shin. The T-score and Zscore were used to examine the results. The T-score is the most directly relevant patient risk metric. The T-score compares the participants' scores to a speed of sound (SOS) value. The T-score indicates how far the present patient's SOS result deviates from the mean. T-scores >1.0 are typical. T-score 0.9-2.6 is osteopenic, while Tscore 2.6 is osteoporosis. Abnormal BMD included osteopenia and osteoporosis. As a screening technique, bone ultrasonography has good sensitivity and specificity in predicting low bone mass.

RESULTS

Table 1: Statistical descriptions of the participants and

Variables	Diabetics	Controls	
	Mean±SD	Mean±SD	
Age	56.801±8.548	49.386±12.649	
Height	1.324±0.143	1.6034±0.280	
Weight	70.554±10.342	70.586±11.856	
BMI	27.675±5.869	28.865±5.145	
HGS max	23.875±10.337	28.453±10.784	
ET in seconds	16.763±5.879	18.298±7.456	
BMD	-1.478 ± 1.0349	-1.398±1.0289	

Endurance time

BMD and BMI had a week negative association (r = 0.032) that was not statistically significant (P= 0.705), whereas BMD and HGS max in sec had a week positive connection (r=0.213) that was statistically significant (P=0.017). BMD and ET had a slight positive connection (r=0.142), which was not statistically significant (P=0.118). BMI and HGS max in sec had a positive connection (r= 0.182), which was statistically significant (P= 0.032). BMI and ET had a weak positive connection (r=0.051), although it was not statistically significant (=0.538). HGS max and ET had a positive connection (r = 0.299) that was statistically significant (P 0.001). The difference in BMI, HGS max, ET, and BMD between diabetics and non-diabetics was not statistically significant. However, non-diabetics exhibited considerably greater HGS

and ET than diabetics in men (0.05). HGS and ET were statistically significant among female diabetics and non-diabetics, with P <0.001 and 0.04 respectively. The distribution of participants with varied BMD grading among diabetics and non-diabetics was not statistically significant among men (P = 0.856). The distribution of participants with varied BMD grading among diabetics and non-diabetics was not statistically significant among females (P = 0.762).

Males (BMD)	Diabetic (%)	Non- Diabetic%	P -value
Normal (01)	47	46	0.882
OPE (-12.5)	45	44	
OPO (>-2.5)	8	9	
Females (BMD)	Diabetic	Non diabetic	P -value
Normal (01)	39	38	0.705
OPE (-12.5)	42	45	
OPO (>-2.5)	19	17	

DISCUSSION

In our analysis, there was a small negative association between BMI and BMD in T2DM, but this was not statistically significant. When BMI and HGS max were compared, there was a small negative correlation. Men with non-diabetes had much more HGS, but non-diabetic females had higher HGS and ET, the latter being extremely significant (P <0.002). We discovered no significant change in BMD between two groups. Although not statistically significant, osteopenia was more prevalent in non-diabetic men and women, but osteoporosis was more common in diabetes men and women of BMD, which evaluates mineral density in bones. Low BMI is a risk factor for low BMD. When adjusted for age, diabetes, hypertension, and exercise, BMI explained the BMD association better. According to research's moderate to morbid obesity may not be a protective factor for osteopenia. Our study's participants had a high BMI (25.4341 + 3.62399), which would explain the minor unfavorable correlation. In our analysis, BMD associated with HGS max and ET in type II DM. They observed that HGS deteriorated quicker in T2DM female patients than in non-DM healthy controls beyond the age of 40, suggesting that decreasing muscular strength may play a crucial role in osteoporosis in DM patients. Ansari observed that diabetes-related connective tissue illnesses, neuropathy, or vasculopathy may have a synergistic impact on the increased incidence of musculoskeletal difficulties, as indicated by lower HGS max and ET in T2DM. Our findings reveal a minor negative association between BMI and HGS max. Ilich et al. reported that osteosarcopenic obese women with rising adiposity had the lowest HGS scores. Men with non-diabetes showed much more HGS, but non-diabetic females had higher HGS and ET, the latter being extremely significant (P <0.001). Our findings mirrored those of Ahmed et al. who discovered T2DM is connected with lower muscle power and thus HGS max. Diabetes produces lower muscle strength relative to age-matched healthy adults due to insulin tissue resistance and hyperglycemia. Insulin resistance inhibits postprandial muscle protein synthesis, lowering muscle mass in T2DM patients. It is also hypothesised that a decline in mitochondrial number in muscle cells and an increase in circulating cytokines impair glycogen synthesis. Motor neuron involvement in T2DM may induce muscular weakness. Our analysis identified no significant variations in BMD between diabetes patients and age- and sex-matched healthy controls. Our finding showed no significant variation in BMD between two groups. Notably, non-insulindependent DM exhibited normal bone mineral metabolism. However, we discovered that non-diabetic males and females had more osteopenia, whereas diabetes males and females had higher osteoporosis, but not statistically significant. Concluded that T2DM women had greater BMD and decreased risk

Our study indicated a minor negative association between BMI and BMD among diabetic adults. Leslie et al. observed comparable results. In their study, Fawzy et al. and Mishra et al discovered a strong association between T2DM and increased BMI, presumably related to

hyperinsulinemia. In T2DM, there is frequently too much insulin. Insulin is known to have an anabolic action on bone due to its structural similarities to IGF1. It also interacts with IGF1 receptors on osteoblasts, which is critical for new bone development. Nikolay's study suggests that T2DM patients are more prone to fractures. A larger BMD does not protect against fracture risk in T2DM patients due to changing bone structure, leading in lower bone strength and increased fracture risk. Diabetes may impact bone in numerous ways. A series of nonenzymatic interactions between glucose and proteins may culminate in the development of advanced glycation end products (AGEs) in bone collagen. AGEs weaken the elasticity and increase the permeability of blood vessels, contributing to diabetic vascular complications. Bone fragility is known to be worsened by AGEs in collagen. T2DM patients may have lower, similar, or higher BMD at different ages and anatomical locations, hence each patient should be examined independently. They urge that all T2DM patients be tested for osteoporosis risk so that suitable preventative and lifestyle strategies including exercise, calcium, and Vitamin D supplements may be prescribed.

Our study's strength is that T2DM may not change BMD, but it may reduce muscular strength, as evidenced by lower HGS in T2DM compared to controls. Although not statistically significant, osteopenia was more prevalent in non-diabetic men and women, but osteoporosis was more common in diabetes men and women. Our study's limitations include that we did not group diabetic patients by disease duration to determine BMD, and we only analyzed BMD at one site. We haven't examined how lifestyle impacts bone and muscle metabolism. Further studies splitting patients into diabetes duration groups, assessing BMD at multiple places, and considering lifestyle may reveal a definitive relationship between T2DM and BMD.

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