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Study of Bone Mineral Density, Serum Sclerostin, and Parathyroid Hormone Levels in Children and Adolescents with Type 1 Diabetes Mellitus

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ABSTRACT

Background: Children and adolescents with type 1 diabetes (T1DM) are more probable to have compromised bone health, as changes in bone mineral density (BMD), serum sclerostin, and parathyroid hormone levels may be involved in skeletal issues. The purpose of this study was to evaluate bone mineral state in children and adolescents with type 1 diabetes.

Methods: The present prospective case-control trial was conducted on 75 children and adolescents with ages from 5 to 18 years old, both sexes, having T1DM (Patients group) and 25 healthy children (the control group). Patients were exposed to BMD, serum sclerostin and parathyroid hormone measurement.

Results: 25-hydroxyvitamin D (25 OH vit D), serum ionized calcium (Ca), parathormone hormone (PTH), phosphorus and alkaline phosphatase (ALP) were comparable between both groups. BMD was notably reduced in patients' group than control group ($P<0.05$). Sclerostin was significantly elevated in diabetic patients than control group ($P<0.001$). Level of Sclerostin had a positive correlation with diabetes duration, mean glycosylated hemoglobin per year in the diabetic patients ($P <0.001$). Sclerostin level stated a negative relation with total body and lumbar BMD Z score ($P<0.001$).

Conclusions: There was evidence of low BMD in diabetic patients valued by dual-energy X-ray absorptiometry scan, especially in long standing diabetes with poor glycemic control. Diabetic cases with better glycemic control showed better BMD and sclerostin levels.

Keywords: *Bone Mineral Density, Serum Sclerostin, Parathyroid Hormone, T1DM*

1. INTRODUCTION

Diabetes mellitus (DM) is a multifaceted metabolic condition marked by episodes of hyperglycemia and glucose intolerance It occurs either due to the destruction of beta cells in the pancreas, leading to insufficient insulin secretion (type 1), decreased sensitivity to insulin in target tissues despite normal insulin production (type 2), or a combination of both [1].

Type 1 DM is one of the two major forms of this disease which, in contrast to type 2 DM is characterized by a childhood onset. T1DM develops because of autoimmune damage of pancreatic beta cells, leading to gradual loss of insulin production. [2].

It is now evident that this disease also negatively impacts the skeleton. T1DM can alter bone metabolism, causing reduced in formation of bone and lower bone quality, which raises the risk of developing osteoporosis and experiencing fractures [3].

Bone growth and remodeling are substantial during childhood and teenage years, with 25% of greatest bone mass reached during teenage years. Therefore, it is likely that the effect of T1DM on skeletal health starts at the time of diagnosis in childhood and adolescence, resulting in skeletal alterations and insufficient bone mass development [4].

Since then, several studies have been performed to determine BMD in children and adolescents with T1D utilizing dual energy X-ray absorptiometry (DXA). Reduced BMD was observed in T1D cases during childhood and adolescence [5].

The mechanisms behind diabetic osteopenia and bone fragility in individuals T1D are not completely understood. The pathogenesis of these conditions is likely due to multiple contributing factors. Potential pathogenic mechanism is low bone turnover [6].

Many studies concluded that diabetes is related to low bone turnover that comes across as a common characteristic in diabetes. This reduced bone turnover in diabetes is assumed to be triggered by osteocyte dysfunction and elevated sclerostin levels leading to the formation of bone micro-cracks [7].

Sclerostin is a protein released by mature osteocytes and encoded by SOST gene, which suppresses formation of bone by blocking Wnt/β-catenin signaling [8].

This work aimed to estimate the bone mineral status in T1DM adolescents and children through assessment of BMD, BMD Z-score of total body and lumbar spine by DXA considering the chronological age (CA) and height age (HA) at the time of the measurement to identify any discrepancies between CA and HA that could impact BMD readings by DXA. Additionally, key bone metabolism markers were evaluated, including Ca serum levels, P, ALP, PTH, and sclerostin

2. PATIENTS AND METHODS:

The current prospective, case-control analysis was conducted on 75 adolescents and children aged from 5 to 18 years old, both genders, had T1DM, criteria are fasting blood glucose (FBG) levels of 126 mg/dl (7.0 mmol/l) or higher than or 2 hours postprandial blood glucose (PPBG) of 200mg/dl (11.1mmol/l) or higher, or random blood sugar (RBS) of 200mg/dl) (11.1 mmol/l) or higher, or glycosylated hemoglobin (HBA1c) of 6.5% or higher, with classic symptoms of DM (polydipsia, polyphagia, polyuria, weight loss, tiredness and lethargy) and 25 healthy children as a control group. This study was executed from January 2021 to January 2023, following approval from the Ethical Committee Tanta University Hospitals, Tanta, Egypt. An informed written consent was taken from relatives of cases.

We exclude cases with conditions which affect bone density (vitamin D deficiency, cushing syndrome, thyroid disorders, parathyroid disorders, systemic steroid use, malnutrition, type 2 DM, chronic systemic diseases such as chest, heart, or kidney diseases) from our study.

The participants were categorized into two groups: the patient group (n=75), consisting of individuals with T1DM, and the control group (n=25), which included healthy children matched for age and sex with the patients and had no symptoms of DM and were confirmed by fasting plasma glucose to be non-diabetic by American diabetes association criteria [9].

The whole patients underwent a comprehensive review of their medical history, clinical examination and laboratory tests, containing complete blood count (CBC), liver function tests (LFT), renal function tests (RFT), total cholesterol, low density lipoproteins (LDL), cholesterol, high density lipoproteins (HDL), triglyceride (TG), FBG level, 2 hours post prandial, HBA1c, last 4 HbA1c, mean HbA1c of last year, plasma protein , albumin levels, serum ALP, serum ionized Ca, serum phosphorus levels, serum PTH levels, 25 OH vit. D level and serum level of sclerostin and radiological investigations [DXA].

Serum sclerostin level

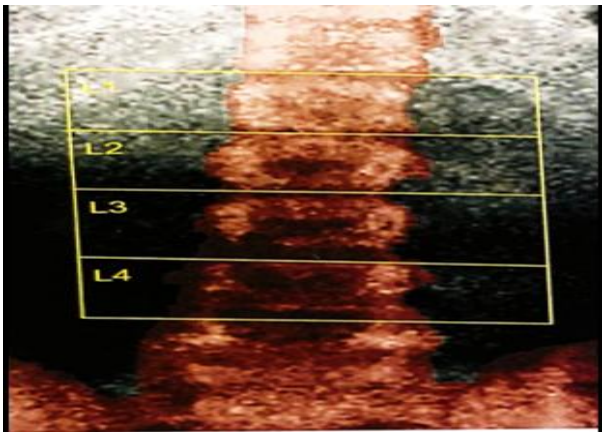
Enzyme immune-assay for the quantitative measurement of human serum sclerostin. Kits supplied by biomedical. Catalog number BI-20492.

Assay protocol: Begin by adding 150 µl of ASYBUF (red cap) to each well. Then, add 20 µl of STD/SAMPLE/CTRL (Standard/Sample/Control) in duplicate into respective well. Add 50 µl of AB (biotinylated anti-sclerostin antibody, green cap with green dye) to each well and gently swirl. Cover the plate securely and overnight incubate (18-24 hours) at room temperature (18-24°C) in the dark. Incubation above room temperature lowers the top OD. After incubation, aspirate the wells and wash them 5 times with 300 µl of diluted WASHBUF. After the final wash, firmly tap the plate on a paper towel to remove any leftover WASHBUF. In each well, add 200 µl of CONJ (Conjugate, Amber Cap) and cover tightly. Incubate for 1 hour at room temperature (18-24°C) in the dark. Aspirate and wash the wells 5 times with 300 µl of diluted WASHBUF. Next to the last wash, eliminate any excess WASHBUF by firmly pressing the plate on a paper towel. In each well, add 200 µl of SUB (Substrate, Blue Cap) and incubate for 30 minutes at room temperature (18-24°C) in the dark. Finally, add 50 µl of STOP (stop solution, white cap) to each well and measure the absorbance immediately at 450 nm.

Bone mineral density measurement

BMD values for the whole body and lumbar vertebrae (L1-L4) were assessed utilizing DXA, and both BMD and bone mineral content (BMC) were obtained. BMD values were then used to estimate BMD Z-scores based on CA and HA. Values calculated were in comparison with normal individuals of the same age and gender as normative data [10]. The BMD Z-score results were interpreted based on the 2013 Official Positions of the International Society for Clinical Densitometry (ISCD) for pediatrics and childhood as follows: When z-score of BMD is more than -1 SD, it is considered normal; a z-score between -2.0 and -1.9 SD is categorized as at risk for low BMD, and a z-score of -2.0 or lower is regarded as low BMD (increased fracture risk) [11]. **Figure 1**

(A)



(B)

(C)

Figure 1: (A) normal lumbar bone mineral density, (B) low normal lumbar bone mineral density and (C) low lumbar bone mineral density

The risk to participants and measures used to minimize these risks: to minimalize the risk of infection, the collection of samples was done under complete aseptic condition.

Statistical analysis

The statistical analysis was performed using SPSS v26 (IBM Inc., Chicago, IL, USA). The Shapiro-Wilks test and histograms were employed to determine the normality of the distribution of data. Quantitative parametric variables were presented as mean and standard deviation (SD), and the two groups were compared using the unpaired Student's T-test. Quantitative non-parametric data was reported as median and interquartile range (IQR) and examined using the Mann Whitney test. Qualitative variables were provided as frequency and percentage (%) and examined using the Chi-square test or Fisher's exact test, as appropriate. The Pearson moment relation equation was used to examine the relationship between several variables. A two-tailed P value of <0.05 was judged statistically significant.

3. RESULTS:

Demographic data, FH of DM and pubertal stage were comparable among both groups. The mean age of onset, DM duration and insulin dose respectively were 6.7±2.06, 3.5±1.27 and1.2±0.14. Thirty-nine patients (52.0%) of diabetic patients presented with the classic symptoms of diabetes, while 36 children (48.0%) presented with DKA. The mean of age of onset was 5.1±1.41in prepubertal group and was 8.1±1.47 in pubertal group. **Table 1**

Table 1: Demographic data, FH of DM, pubertal stage of the studied groups, age of onset, disease duration, mode of first clinical presentation, insulin regimen, puberty, age of onset in diabetic children

	Cases group	Control group	Test	P

		(n = 75)	(n = 25)		
Age (years)		10.5±2.78	11.1±3.05	t=0.892	0.374
Sex	**Male**	43(57.3%)	15(60.0%)	χ^2=0.055	0.815
	Female	32(42.7%)	10(40.0%)		
Weight (kg)		45.0(37.0–56.7)	42.3(36.15–55.1)	U=890.5	0.708
Height (meter)		1.52(1.43–1.59)	1.53(1.44–1.59)	t=0.266	0.791
BMI (%)		19.05(17.16–22.01)	19.33(16.89– 21.77)	U=897.5	0.750
Waist circumference (cm)		63.0±8.59	62.6±7.87	U=909.0	0.820
Waist hip ratio (%)		0.86±0.02	0.85±0.03	t=0.657	0.512
FH of DM	**No**	43(57.3%)	17(68.0%)	χ^2=0.889	0.346
	Yes	32(42.7%)	8(32.0%)		
Pubertal stage					
Prepubertal Tanner 1		33(44.0%)	11(44.0%)	--	0.891
Early Puberty Tanner 2		15(20.0%)	4(16.0%)		
Early puberty Tanner 3		13(17.3%)	5(20.0%)		
Late puberty Tanner 4		9(12.0%)	2(8.0%)		
Late puberty Tanner 5		5(6.7%)	3(12.0%)		
		Cases group (n = 75)			
Age of onset (years)		6.7±2.06		--	--
Duration of DM (years)		3.5±1.27		--	--
Mode of first clinical presentation	**Classical**	39(52.0%)		--	--
	DKA	36(48.0%)		--	--
Insulin dose (u/kg/d)		1.2±0.14		--	--
		Prepubertal (n = 33)	**Pubertal (n = 42)**		
Age of onset (years)		5.1±1.41	8.1±1.47	--	--

Data are exhibited as mean ± SD or frequency (%) or median (IQR). t: Independent t test, U: Mann Whitney U test, χ2: Chi square test. BMI: body mass index, FH: family history, DM: diabetes mellitus, DKA: diabetic ketoacidosis.

FBS, 2 hr. post prandial, last HbA1C, HbA1C mean last year and sclerostin were notably elevated in case group than control group (P<0.05). Hb, TLC, platelet, creatinine, ALT, AST, 25 OH vit D, S. ionized Ca, PTH, phosphorus, ALP, microalbuminuria, cholesterol, HDL, LDL and TG were comparable in the two groups. Sclerostin significantly increased in diabetic group with good and poor control than the controls (P <0.001). **Table 2**

Table 2: Comparison between studied groups regarding laboratory investigations

	Cases group (n = 75)	**Control group (n = 25)**	**Test**	**P**
FBS	130.5±15.53	86.0±19.76	U=389.5	**<0.001***

2 hr. post prandial	162.2±25.90	120.4±25.16	U=366.5	**<0.001***
Last HbA1C	9.1±1.22	5.0±0.23	t=27.165	**<0.001***
HbA1C Mean last year (%)	9.2±1.23	5.0±0.23	t=28.472	**<0.001***
Hb (gm/dl)	11.4±1.34	11.5±1.31	t=0.403	0.688
TLC (c/mm^3)	6960.67±1947.04	6522.0±1994.79	U=807.5	0.300
Platelet (c/mm^3)	257000.0(204000.0–302000.0)	259000.0(207000.0–336000.0)	U=903.5	0.787
Creatinine (mg/dl)	0.8±0.13	0.8±0.18	t=0.604	0.547
ALT (IU/L)	29.0(22.0–34.0)	30.0(21.0–36.5)	t=0.221	0.826
AST (IU/L)	31.0(27.0–38.0)	31.0(22.5–37.5)	U=833.5	0.407
25 OH vit D (ng/ml)	39.2±6.09	37.5±5.19	U=794.0	0.253
S. ionized Ca (mmol/l)	1.3±0.06	1.3±0.06	t=0.591	0.556
PTH (pg/ml)	32.4±6.61	29.8±7.32	U=716.0	0.077
Phosphorus (mg/dl)	4.6±0.34	4.6±0.38	t=0.065	0.948
ALP (U/L)	219.3±38.80	205.2±32.07	U=748.0	0.130
Microalbuminuria (mg/dl)	3.720±0.939	3.960±0.841	U=921.0	0.895
Cholesterol (mg/dl)	135.8±10.70	136.4±12.02	U=921.0	0.895
HDL	50.1±3.69	49.8±3.79	U=912.5	0.842
LDL	82.9±8.08	79.8±6.75	U=718.0	0.080
TG (mg/dl)	107.5±12.36	106.4±10.58	U=859.0	0.532
Sclerostin (pmol/L)	35.9±6.38	20.9±2.71	t=16.400	**<0.001***

Data is stated as mean ± SD or median (IQR). *Significant P value<0.05. t: Independent t test, U: Mann Whitney U test. FBS: fasting blood sugar, HbA1C: glycosylated hemoglobin, Hb: hemoglobin, ALT: alanine aminotransferase, AST: aspartate aminotransferase, 25 OH vit D: 25-hydroxyvitamin D, S: serum, Ca: calcium, PTH: parathormone hormone, ALP: alkaline phosphatase, HDL: high density lipoprotein, LDL: low density lipoprotein, TG: triglycerides.

BMD significantly reduced in diabetic group than healthy control (P<0.05). Classification of cases according to BMD revealed 5 cases with low normal lumbar spine BMD Z score, while 1 case shows low bone density with lumbar BMD Z score below -2. According to total body BMD Z score, only 2 cases show low normal BMD. **Table 3**

Table 3: Comparison between studied groups regarding BMD and its descriptive data

	Cases group (n = 75)	**Control group (n = 25)**	**Test**	**P**
Lumbar spine (L1- L4) (gm/cm^2)	0.7±0.14	0.8±0.17	t=2.716	**0.008***
Lumbar spine (L1- L4) (Z score)	-0.4(-0.6–0.2)	0.5(0.3–0.85)	t=7.625	**<0.001***
Total body (gm/cm^2)	0.7±0.13	0.8±0.15	t=2.466	**0.015***
Total body (Z score)	-0.3(-0.5–0.2)	0.6(0.4–0.9)	t=6.451	**<0.001***

Lumbar spine (L1-L4)	**Normal**	69(92.0%)	25(100.0%)	--	--
	Low normal	5(6.7%)	0(0.0%)		
	Low bone density	1(1.3%)	0(0.0%)		
Total body	**Normal**	73(97.3%)	25(100.0%)	--	--
	Low normal	2(2.7%)	0(0.0%)		

Data is stated as mean ± SD or median (IQR) or frequency (%). * Significant P value<0.05. t: Independent t test. BMD: bone mineral density.

Sclerostin, lumbar spine (L1- L4) and total body (Z score) were insignificantly different between males and females in the diabetic patients. **Table 4**

Table 4: Sclerostin, lumbar spine (L1- L4) and total body (Z score) of diabetic children

	Male (n = 43)	**Female (n = 32)**	**Test**	**P**
Sclerostin	35.7±6.31	36.2±6.58	t=0.259	0.796
Lumbar spine (L1- L4) (Z score)	-0.3(-0.7–0.3)	-0.5(-0.6–0.1)	t=0.601	0.549
Total Body (Z score)	-0.2(-0.5–0.2)	-0.4(-0.5–0.175)	t=0.865	0.390

Data is presented as mean ± SD or median (IQR). t: Independent t test.

Sclerostin significantly increased in diabetic cases with good and poor control than the controls (P<0.001). Sclerostin was elevated in diabetic cases with poor control compared to diabetic cases with better control. The lumbar spine and total body BMD Z score significantly reduced in diabetic cases both with good and poor control than the controls. Lumbar spine and total body BMD Z score were notably reduced in diabetic group with poor control than diabetic cases with good control (P<0.001). **Table 5**

Table 5: HbA1C, sclerostin and BMD of studied children

	Cases		**Controls (n = 25)**	**Test**	**P**
	HbA1C <7 Good control (n = 9)	**HbA1C >7 Poor control (n = 66)**			
Sclerostin	27.6±2.85	37.1±5.87	20.9±2.71	F=96.482	**<0.001***
Lumbar Spine (L1- L4) (Z score)	0.2(0.1–0.3)	-0.4(-0.7– -0.05)	0.5(0.3–0.85)	F=20.715	**<0.001***
Total Body (Z score)	0.2(0.05–0.3)	-0.3(-0.5–0.125)	0.6(0.4–0.9)	F=24.258	**<0.001***

Data is presented as mean ± SD or median (IQR). * Significant P value<0.05. F: ANOVA. HbA1C: glycosylated hemoglobin, BMD: bone mineral density.

Sclerostin levels were correlated positively with the diabetes duration and mean HbA1C per year in the diabetic group (P <0.001). Also, it stated a negative relation with total body and lumbar BMD Z score (P<0.001). There is no or weak correlation between sclerostin and age and PTH. Also, there was a substantial negative relation between the total body and lumbar BMD Z scores, the duration of diabetes, and the mean HbA1C per year in the diabetic patients (P< 0.001). **Table 6**

Table 6: Correlation between sclerostin, BMD and other variables in cases

	Sclerostin			
	r		**P**	
Age	0.146		0.168	
Duration of diabetes	0.794		**<0.001***	
Mean HbA1C per year	0.783		**<0.001***	
PTH	-0.157		0.179	
Lumbar spine (L1- L4) (Z score)	-0.543		**<0.001***	
Total body (Z score)	-0.565		**<0.001***	
	Lumbar spine (L1- L4) (Z score)		**Total body (Z score)**	
	r	**P**	**r**	**P**
Age	-0.217	0.062	-0.252	0.059
Duration of diabetes	-0.525	**<0.001***	-0.517	**<0.001***
Mean HbA1C per year	-0.552	**<0.001***	-0.532	**<0.001***
PTH	0.128	0.247	0.099	0.399
Sclerostin	-0.543	**<0.001***	-0.565	**<0.001***

r: correlation coefficient. * Significant P value<0.05. BMD: bone mineral density, HbA1C: glycosylated hemoglobin, PTH: parathormone hormone.

4. DISCUSSION

DM is a complex metabolic condition showing episodes of hyperglycemia and glucose intolerance. It develops due to lack of secretion of insulin due to loss of beta cell in the pancreas (type 1), loss of the insulin sensitivity in target organs despite normal insulin secretion (type 2), or both [1].

In this study, the last HbA1c notably increased in diabetic patients than control patients. Also, HbA1C mean last year significantly increased in the diabetic group than the controls.

There were comparable results as regards hematological values, renal functions, hepatic profile, microalbuminuria and lipid profile between the diabetic patients and the controls.

In the current study, there were comparable results regarding 25 hydroxy vitamin D level, serum ionized Ca, phosphorus, ALP and PTH between the diabetic group and the controls.

Our study shows that the serum level of sclerostin was notably elevated in the cases with diabetes than the control group. The mean value of sclerostin serum level was notably elevated in the diabetic patients than controls without significant differences between sex and with no correlation to age. Neumann et al. [12] illustrated that T1DM males and females with long standing T1DM had elevated sclerostin serum levels than controls.

Several analyses have assessed the sclerostin levels in adult cases with both T1D and T2D. Garcia-Martin et al. [13] noted that in T2DM males, not in females, serum sclerostin levels elevated compared to controls.

In adults, sclerostin levels also elevated in patients with prediabetes than healthy participants in the study done by Daniele et al. [14].

No differences had found in sex of T1D patients and healthy controls. However, sex variation in sclerostin was noted as well in T1D children and adults, but the outcomes were conflicting. In many studies, sclerostin levels elevated in males with T1D, in contrast Catalano et al. [15] found that females patients with T1DM exhibited higher sclerostin levels compared to males. Contrast to our outcome, Tsentidis et al. [16] demonstrated that children and adolescents with T1DM had comparable sclerostin levels to the controls, which showed a Gaussian distribution.

Sclerostin level was correlated positively with duration of diabetes in the diabetic group in our trial (P <0.001). Neumann et al. [12] stated that sclerostin serum levels were elevated in cases with long standing T1DM.

Our study shows that sclerostin level positively correlated with Mean HbA1C per year in the diabetic group (P <0.001). Additionally, sclerostin levels were observed to improve in patients with better glycemic control. Consistent with these findings, Gennari et al. [17] stated that there was a positive relation among HbA1C and sclerostin levels in type 2 diabetes, but not in type1.

The findings of adult T1DM analysis also vary. One trial revealed a positive relation between sclerostin and HbA1c in patients [12] and no association in two others [15].

Regarding this study, there was no relation between sclerostin level and bone metabolic markers including parathyroid hormone, serum ionized Ca, ALP and 25 OH vitamin D level.

Our study found no relation between sclerostin and PTH which aligns with the same finding reported by Kirmani et al. in healthy children [18].

This study showed that the BMD of the total body and lumbar spine was notably reduced in diabetic individual than the controls. Mean total body BMD was notably reduced in the diabetic group, and diabetic children also had a substantially decrease total BMD Z-score than the controls. Furthermore, the study found that the mean lumbar spine BMD was notably lower in the diabetic group, with diabetic children exhibiting a notably reduced lumbar BMD Z-score than the controls. They found no correlation between these differences and age or sex.

Our study described that 69 cases with normal lumbar BMD with Z score more than -1 while 5 patients with Z score between -1 to -2 with low normal BMD and only 1 case with reduced BMD with Z score below -2 who are prone to risk of fracture. The study also demonstrated that only 2 cases with low normal total BMD Z scores while the others are normal. Our finding was consistent with the finding at Tsentidis et al. [16] observed that BMD reduced in children and adolescents with T1DM compared to controls. As T1DM cases had reduced values of lumbar and total body BMD z-score than matched individuals. Loureiro et al. [19] found that BMD was substantially lower in diabetic children and adolescents in comparison with controls.

The Danish study found that the TBLH-BMD Z-score in schoolboys with T1D was comparable to that of healthy controls, while schoolgirls showed higher TBLH-BMD Z-scores, which corresponded with higher weight and BMI Z-scores. However, the difference in TBLH-BMD Z-scores between boys and girls was no longer present after adjusting for weight (and BMI), age, and HbA1c levels. Kumar et al. [20] demonstrated that children with T1D had BMD levels nearly identical to those of their healthy counterparts. Maddaloni et al. [21] performed a study on aging T1D individuals who had lived with the condition for 50 years or more. They revealed normal DXA Scan results showing a fracture risk in T1D patients comparable to that of their non-diabetic peers.

The present study showed negative correlation among total body and lumbar BMD z score and the duration of diabetes in the diabetic patients (P <0.001). Also, showing negative correlation between total body and lumbar BMD z score and the Mean HbA1C per year in the diabetic group (P <0.001). The same observations were observed by Loureiro et al. [19] illustrated a negative correlation between BMD and HbA1c in T1D group. Sayarifard et al. [22] reported that only higher HbA1c level significantly predicted reduction of BMD with no significance with diabetes duration. Leão at al. [2] reported that the length of the disease duration in T1DM patients plays a role in altering the total body BMD Z-score, with no correlation between HbA1c values with bone mass. Fuusager at al. [6] found a negative relation between the mean most recent HbA1c and BMD Z-score in children and adolescents with T1DM. Their study also revealed no correlation between BMD Z-score and age of diabetes onset or disease duration.

The current trial revealed a negative relation between sclerostin levels and total body and lumbar BMD z-scores in the diabetic group, which agreed with Ardawi et al. [23] who noted negative relation between sclerostin and BMD in older patients. Different from our results, Tsentidis et al. [16] revealed a positive relation between sclerostin and L1–L4 BMD and total body BMD z-scores in cases and control groups.

However, our trial had some limitations, as sample size was small. So, we recommend that assessment of sclerostin level in patients with T1DM especially with history of long duration and poor glycemic control. Dexa scan in individuals with T1DM especially with history of long duration and poor glycemic control for early detection of osteopenia and osteoporosis. Valuable glycemic control and better bone health for patients with type 1 DM. Larger population and upgrading workstation would be helpful to confirm the effect of sclerostin, which is an important marker of bone metabolism, on the diabetic cases' bone health and the values of DEXA scan as predictor of early diabetic bone complications.

5. CONCLUSIONS:

Adequate diabetic control is a very essential goal to avoid diabetic complications. There is evidence of increasing the level of serum sclerostin, which is an important bone metabolism marker, in type 1 DM cases especially in long standing diabetes with poor glycemic control. The elevated sclerostin levels can be a very important cause for bone health deterioration in

diabetic patients. Better sclerostin levels with better glycemic control. There is evidence of low BMD in diabetic patients assessed by DEXA scan, especially in long standing diabetes with poor glycemic control. Better BMD in diabetic cases with better glycemic control. There is no evidence of differences between the diabetic group and the controls regarding 25 OH vit. D level, serum ionized Ca, phosphorus, ALP and PTH.

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Conflict of Interest: Nil

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