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Original Article

Does Oxidative Status Affect Serum Sclerostin Levels in Patients with Type 2 Diabetes Mellitus?

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Abstract

Introduction: Sclerostin is a glycoprotein known as a negative regulator of bone formation, predominantly expressed by mature osteocytes. There is no causative evidence information on the role of sclerostin in the pathogenesis of type 2 diabetes mellitus (T2DM) in humans.

Aim: This study aimed to investigate the relationship between serum sclerostin levels and oxidative status and biochemical parameters in T2DM patients and healthy people.

Materials and methods: This cross-sectional study, conducted in a clinical trial center, included 45 subjects with T2DM and 45 subjects as controls.

Results: Serum sclerostin, total oxidative status (TOS), albumin, and ferritin levels were significantly higher in T2DM patients than in the control group ($p<0.05$). Total antioxidant status (TAS) was significantly higher in the control group ($p<0.05$). There was a weak positive correlation between sclerostin and TOS ($r=0.23$, $p=0.03$) and a weak negative correlation between sclerostin and TAS ($r=-0.28$, $p=0.03$).

Conclusions: We have demonstrated that serum sclerostin levels increase in patients with T2DM and that the increased sclerostin levels are associated with oxidative stress.

Keywords

type 2 diabetes mellitus, sclerostin, TAS, TOS

INTRODUCTION

T2DM is a global health problem characterized by irregularity of carbohydrate, lipid, and protein metabolism resulting from impaired insulin secretion, insulin resistance, or a combination of both.[1] Serum sclerostin levels are elevated in patients with T2DM. This increase is thought to be associated with disease duration and blood glucose bad control (HbA1c), and this molecule is at least a potential mediator in the development of diabetes-related bone

disease.[2] Sclerostin is the key molecular coordinator of both bone formation and bone resorption. Irregularity of sclerostin expression forms the basis of pathophysiology in skeletal disorders characterized by loss of bone mass and the damaging effects of some cancers on bone tissue.[3] Immunohistochemical approaches state that sclerostin is expressed in osteocytes in human and rodent bones.[4] Vitamin D is very important for bone metabolism. Vitamin D deficiency may cause abnormalities in calcium and phosphorus levels.[5] Oxidant-antioxidant balance is important for the homeostasis of an organism. Oxidative stress occurs when this balance is disrupted. Oxidative stress involves macromolecular oxidative damage, induces tissue protein denaturation, DNA damage, and lipid peroxidation, and interferes with the normal metabolic activity of the body, leading to the emergence and/or development of diseases.[6] Information on the role of sclerostin in the pathogenesis of T2DM in humans remains insufficient, and the relationship between sclerostin and oxidative stress remains unclear.

AIM

This study aims to compare the relationship between serum sclerostin level and oxidative status, HbA1c, folate, B12, ferritin, vitamin D, Ca, P, Mg in T2DM patients and healthy people.

MATERIALS AND METHODS

Study group

The study included 45 T2DM patients (case group) admitted to Dicle University Hospitals Endocrine Clinic and outpatient clinic and 45 age- and sex-matched healthy individuals (control group) older than 18 years of age. The diagnosis of T2DM (determined by the international American Diabetes Association) was made according to the study group diagnostic criteria. Blood samples were taken from individuals diagnosed with T2DM and from the healthy control group after obtaining a signed voluntary consent form. All controls included in the study had normal glucose homeostasis as evaluated with their fasting glucose levels and glycated hemoglobin (HbA1c) measurement. Blood samples were taken from the antecubital vein and serum was obtained by centrifugation at 4000 rpm for 5 minutes after an average of 15 minutes. The obtained serums were stored in Eppendorf tubes at −80°C until performing the tests.

Compliance with the ethical standards

All human studies were approved by the appropriate ethics committee and were therefore performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. All persons gave their informed consent prior to their inclusion in the study. The study was conducted according to the Helsinki Declaration rules and was approved by the Institutional Ethics Committee of Dicle University Faculty of Medicine (No. 2020/297).

Exclusion criteria

Patients with familial hypercholesterolemia, connective tissue diseases and vasculitis, history of cerebrovascular disease (stroke, transient ischemic attack), diabetes, or peripheral vascular disease due to any other cause were excluded from the study. In addition, patients with a known history of coronary heart disease or acute decompensated heart failure, patients with end-stage renal disease, presence of severe uncontrolled hypertension were excluded from this study.

Laboratory analyses

Serums stored on a working day were gradually dissolved (at +4°C) to be studied in both groups and vitamin D, Ca, Mg, B12, folate, ferritin, phosphorus, albumin levels were studied by electrochemiluminescence method (Cobas e 601-Roche Diagnostics, USA) in the central laboratory of Diyarbakir Gazi Yaşargil Training and Research Hospital. Hemoglobin A1C level (Bray, Ireland/Kansas City, MO, USA) was studied by boronate affinity high-pressure liquid chromatography (HPLC) method. All tests were carried out according to the manufacturer's instructions. Sclerostin (Thermo Fisher-Multiskan) and TAS and TOS levels of both groups were studied by the microELISA method.

TAS and TOS measurement

Serum TAS and TOS measurements were determined using the new automatic measurements method developed by Erel.[7,8] TAS for Assay Range Samples containing 0.1–3.5 mmol Trolox Equiv./L. TOS for Assay Range Samples containing 0.2–80 μmol H_2O_2 Equiv./L.

Statistical analysis

All data editing and statistical analyses were performed using SPPS 21. Graphpad prism 9 program was used for graph drawing. Results were provided as mean ± standard deviation (SD) and min-max. Kolmogorov-Smirnov and Shapiro-Wilk tests were performed. Mann-Whitney U test was used to compare the groups. The Spearman non-parametric correlation was calculated. *P* value <0.05 was considered statistically significant.

RESULTS

The mean age, BMI, and biochemical results of the 90 people included in our study are given in **Table 1**. There was no significance when Ca (9.49±0.59 mg/dl; min: 8.6,

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max: 11.2), P (3.64±1.02 mg/dl; min: 2.9, max: 5.03), Mg (2.04±0.31 mg/dl; min: 1.56, max: 3.15), B12 (624±290 pg/ml; min: 202, max: 1184), folate (9.99±5.5 ng/ml; min: 5.43, max: 23.5), and vitamin D (24.1±11.8 µg/L; min: 7.2, max: 77.69) levels of the T2DM group and Ca (9.25±0.35 mg/dl; min: 1.5, max: 10.6), P (3.30±0.67 mg/dl; min: 2.6, max: 4.5), Mg (2.11±0.15 mg/dl; min: 1.9, max: 2.5), B12 (683.9±155.3 pg/ml; min: 321, max: 900), folate (11.6±3.03 ng/ml; min: 7, max: 17), and vitamin D (25.2 ±6.5 µg/L; min: 11.2, max: 41.94) levels of the control group were compared. The TAS (0.88±0.29 mmol Trolox equivalent/l min: 0.16, max: 1.39) and albumin (3.75±0.5 g/dl; min: 3.80, max: 5) levels of the T2DM group were significantly lower than the TAS (1.88±0.24 mmol Trolox equivalent/l min: 1.29, max: 2.40) and albumin (4.6±0.61 g/dl; min: 3.6, max: 5.2) levels of the control group **(Figs 1, 2)**.

The HbA1c (10.58±3.22%; min: 6.83, max: 14) and TOS (402.8±150.7 µmol; H_2O_2 equivalent/l min: 70.93, max: 633.9) levels of the T2DM group were significantly higher than the HbA1c (3.92±0.63%; min: 2.21, max: 5.23) and TOS (41.2±25.03 µmol; H_2O_2 equivalent/l min: 16.1, max: 143.2) levels of the control group ($p<0.05$). Sclerostin level (168.4±74.9; pg/ml min: 31.59 max: 283) was found to be significantly higher in the T2DM group than in the control group (121.09±71.3; min: 20.32, max: 256) ($p<0.05$) **(Table 1, Fig. 3).**

Finally, there was a significant positive correlation between HbA1c values of the groups and TOS and sclerostin levels ($r=0.77$, $p=<0.01$; 0.23, $p=0.026$). There was a significant negative correlation between the HbA1c and TAS levels of the groups ($r=-0.77$, $p=<0.01$). There was a significant negative correlation between Mg values and TOS values of the groups ($r=-0.23$, $p=0.03$). There was a positive correlation between TAS values whereas there was a negative correlation between folate values and TOS values of the groups ($r=-0.30$, $p=0.004$; $r=0.30$, $p=0.005$) **(Table 2)**.

Table 1. Comparison between T2DM and non-diabetes controls

	T2DM (n=45) Mean ± SD	**Non-diabetic controls (n=45) Mean ± SD**	***P* value**
Age (years)	64.0±10.8	32.1±8.79	NS: 0.39
BMI (kg/m^2)	27.0±3.0	25.8±4.1	NS: 0.62
HbA1c (%)	10.58±3.22	3.92±0.63	<0.01
Calcium (mg/dl)	9.49±0.59	9.25±0.35	NS: 0.254
P (mg/dl)	3.64±1.02	3.30±0.67	NS: 0.467
Mg (mg/dl)	2.04±0.31	2.11±0.15	NS: 0.08
Albumin (g/dl)	3.75±0.5	4.6±0.61	<0.01
B12 (pg/ml)	624±290	683.9±155.3	NS: 0.225
Folate (ng/ml)	9.99±5.5	11.6±3.03	NS: 0.060
Ferritin (ng/ml)	178.4±189	115.50±89.45	0.03
Vit D (µg/L)	24.1±11.8	25.2 ±6.5	NS: 0.621
TAS (mmol Trolox equivalent/l)	0.88±**0.29**	1.88±**0.24**	<0.01
TOS (µmol H_2O_2 equivalent/l)	402.8±150.7	41.2±25.03	<0.01
Sclerostin (pg/ml)	169.4±74.9	120.09±71.3	0.048

Table 2. Correlation analysis of serum TAS, TOS, sclerostin, Vit D, HbA1c, albumin, Mg, and folate

	TAS		**TOS**		**Sclerostin**		**Vitamin D**	
	r	***p***	**r**	***p***	**r**	***p***	**r**	***p***
HbA1c	−0.77**	<0.01	0.77**	<0.01	0.23*	0.026	−0.14	0.18
Albumin	0.47**	<0.01	−0.58*	0.00	0.44**	<0.00	0.42**	<0.01
Mg	0.020	0.85	−0.23*	0.03	0.06	0.57	−0.2	0.06
Folate	0.30**	0.005	−0.30**	0.004	−0.074	0.488	0.056	0.6
TAS	1,000	NS	−0.72**	0.000	−0.28	0.03	0.28	0.03
TOS	−0.72**	0.000	1,000	NS	0.23	0.031	−0.17	0.11

TAS: total antioxidant status; TOS: total oxidant status; Spearman correlation analysis was performed to determine the relationship between parameters. $p<0.05$ results were considered statistically significant. **Correlation is significant at the 0.01 level (2-tailed); r: correlation coefficient

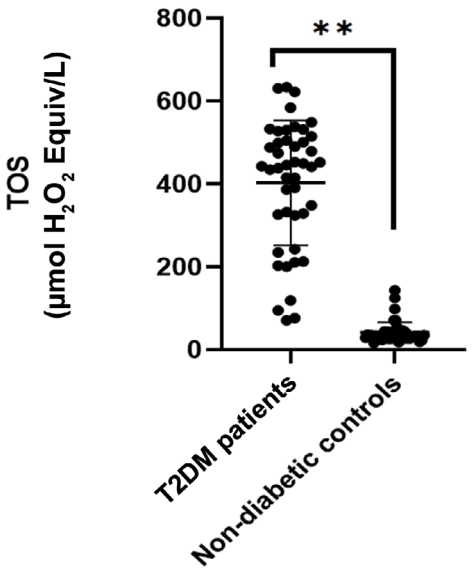

Figure 1. TOS levels in T2DM patients and non-diabetic controls. TDM2: type 2 diabetes; Control group: non-diabetic controls; ** p<0.01 versus control. Values are mean ± SD, Mann-Whitney U test, SD: Standard deviation, min and max.

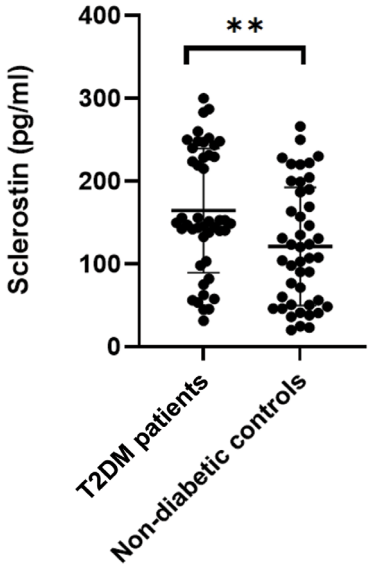

Figure 3. Sclerostin levels in T2DM patients and non-diabetic controls. TDM2: type 2 diabetes; Control group: non-diabetic controls; ** p<0.01 versus control. Values are mean ± SD, Mann-Whitney U test, SD: Standard deviation, min and max.

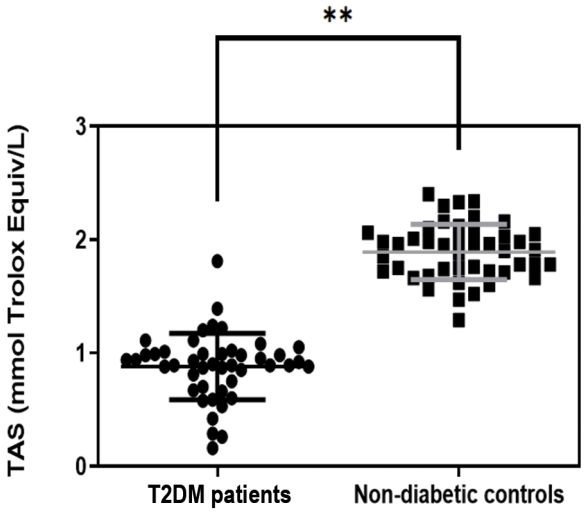

Figure 2. TAS levels in T2DM patients and non-diabetic controls. T2DM: type 2 diabetes; control group: non-diabetic controls; ** p<0.01 versus control. Values are mean ± SD, Mann-Whitney U test; SD: Standard deviation, min and max

DISCUSSION

This is the first study to reveal the relationship between serum sclerostin levels and oxidative stress and other biochemical outcomes in T2DM patients and healthy people. Our study showed that high sclerostin levels were associated with oxidative stress in patients with T2DM. T2DM is a metabolic disorder characterized by hyperglycemia with pathophysiological factors resulting essentially from the combination of insulin resistance and insufficient insulin secretion.[9] Sclerostin responds to the mechanical stress applied to the skeleton and plays an important role in regulating the reshaping of the bone. Sclerostin, which is largely expressed by the SOST gene in osteocytes from bone cells, is a small protein. In addition, sclerostin has been reported to act at a certain distance to regulate adipocytes, energy homeostasis, and mineral metabolism in the kidney.[10,11] In addition, immunohistochemical studies in human and rodent bones have shown that sclerostin is not expressed in osteoblasts or lining cells.[12] Lack of sclerostin expression or secretion in humans causes hereditary, high bone mass formation characterized by exaggerated bone formation such as sclerosteosis, Van Buchem disease, and craniodiaphyseal dysplasia.[13] Sclerostin quickly became a promising molecular target for the treatment of osteoporosis and other skeletal diseases. Useful skeletal results were observed in animal studies and clinical trials using neutralizing antibodies to sclerostin.[3] Compounds that inhibit sclerostin have been shown to stimulate bone formation, reduce osteoporosis, and induce robust increase in bone mineral density.[10] Sclerostin levels have been positively associated with abdominal fat, dyslipidemia, especially HDL and LDL cholesterol levels, and HbA1c. This suggests that sclerostin plays a role in the pathogenesis of the metabolic disease.[14,15] Serum sclerostin levels in our study were found to be higher in the T2DM group than those in the control group **(Fig. 1)**. Significantly higher serum sclerostin levels were detected in male and female patients with T2DM compared to non-diabetic controls in previous studies.[2,16,17] Circulating sclerostin levels have been shown to increase and sclerostin levels have been reported to be positively associated with fasting glucose production

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and insulin resistance measurements even in the case of prediabetes when insulin resistance and insulin secretion first change.[18] Higher sclerostin levels are produced by osteoclast cultures formed from the old bone marrow compared to young mice, suggesting that sclerostin produced from the osteoclast may contribute to a decrease in bone formation caused by aging.[19] There are contradictions in the reports revealing the relationship between serum sclerostin and bone mass even though most studies agree that circulating sclerostin increases with age.[20-22] It is associated with metabolic syndrome and diabetes in humans, similar to mice overexpressing sclerostin. The relationship between sclerostin and HbA1c and diabetes duration has been previously demonstrated and can be explained by strengthening the process of overproduction of reactive oxygen species (ROS) and the formation of advanced glycation end products (AGES) as a result of varying glucose metabolism.[23] High levels of glucose in T2DM lead to the accumulation of advanced glycosylation end products (AGEs) in the organic bone matrix by a process known as non-enzymatic glycation. HbA1c is a common example of early-stage glycation.[24] High glucose concentrations cause oxidative stress. Permanent hyperglycemia and increased oxidative stress play an important role in the development of secondary diabetic complications.[25] Oxidative stress is the imbalance between antioxidants and oxidants. Excessive cellular levels of ROS damage proteins, nucleic acids, lipids, membranes, and organelles. This damage has been associated with various diseases such as diabetes.[26,27] The main sources of oxidative stress are mitochondria in diabetes mellitus. Production of high glucose mitochondrial oxygen radicals is thought to be an important factor underlying diabetes complications.[28,29] In addition, another study reported that glucose and its metabolites react with hydrogen peroxide in the presence of iron and copper ions, forming hydroxyl radicals during auto-oxidation in diabetes and thus supporting ROS formation and the development of diabetic complications.[27] TOS is usually used to detect the general oxidation status of the body whereas the TAS is used to detect the general antioxidant status of the body.[7,8] The T2DM group was found to have low TAS compared to the control group whereas TOS was found to be high in our study **(Figs 2, 3).** Our results are consistent with some previous studies. Picu et al. investigated the values of the parameters characterizing the oxidant/antioxidant balance of T2DM patients and reported that TOS levels were higher in patients with T2DM compared to healthy individuals.[30] Vitamin D is a steroid hormone that plays a role in the regulation of musculoskeletal function.[31] Vitamin D is a key molecule in calcium and phosphate homeostasis.[32] Vitamin D deficiency affects osteocytes and changes serum sclerostin levels. An inverse relationship was reported between serum 25-OHD and sclerostin levels in healthy postmenopausal women in one study.[22] Cidem et al. investigated the effects of vitamin D3 treatment on serum sclerostin levels in young adult women with significant vitamin D deficiency. Twenty-six subjects were treated orally with calcium (1,200 mg/day for 2 months) and vitamin D3 (300,000 IU/week for 1 month). They found that sclerostin levels decreased significantly due to the increase in vitamin D when they examined post-treatment sclerostin levels.[33] However, sclerostin levels were higher in the T2DM group even though there was no significant difference between vitamin D, Ca, P values of the T2DM group and vitamin D, Ca, P values of the control group. In addition, we found that high sclerostin levels were not associated with vitamin D, Ca, P levels in T2DM patients. Furthermore, the high TOS value in patients with T2DM suggested that this might be associated with high sclerostin levels. Several studies on ROS, oxidative stress, and bone have hypothesized that aging-induced oxidative stress antagonizes the Wnt signaling pathway leading to reduced bone formation.[34]

CONCLUSIONS

This study showed that serum sclerostin levels were elevated in patients with T2DM and that high sclerostin levels were also associated with oxidative stress. We determined a positive relationship between high oxidative stress and high sclerostin levels in patients diagnosed with T2DM. It was determined that high sclerostin levels may be a stress factor in the pathophysiological process of T2DM.

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Влияет ли окислительный статус на уровни склеростина в сыворотке у пациентов с сахарным диабетом 2 типа?

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Резюме

Введение: Склеростин представляет собой гликопротеин, известный как негативный регулятор формирования кости, преимущественно экспрессируемый зрелыми остеоцитами. Нет причинно-доказательной информации о роли склеростина в патогенезе сахарного диабета 2 типа (СД 2) у людей.

Цель: Исследование было направлено на изучение взаимосвязи между уровнем склеростина в сыворотке крови, оксидативным статусом и биохимическими показателями у больных СД 2 и здоровых людей.

Материалы и методы: Это поперечное исследование, проведённое в центре клинических исследований, включало 45 лиц с СД 2 и 45 лиц в качестве контрольной группы.

Результаты: Уровень склеростина в сыворотке, общий оксидативный статус (ООС), альбумин и ферритин были значительно выше у больных СД 2, чем в контрольной группе (p<0.05). Общий антиоксидантный статус (ОАС) был достоверно выше в контрольной группе (p<0.05). Между склеростином и ООС наблюдалась слабая положительная корреляция (r=0.23, p=0.03) и слабая отрицательная корреляция между склеростином и ОАС (r=-0.28, p=0.03).

Заключение: Мы продемонстрировали, что уровни склеростина в сыворотке увеличиваются у пациентов с СД 2 и что повышенные уровни склеростина связаны с окислительным стрессом.

Ключевые слова

сахарный диабет 2 типа, склеростин, ОАС, ООС