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OSTEOPOROSIS: INFLUENCE OF LIFESTYLE AND TYPE-2 DIABETES MELLITUS IN MIDDLE AGED MEN

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ABSTRACT

The study aims to evaluate the relationship between glycemic control in middle-aged passive smokers with type-2 diabetes mellitus, vascular damage and osteoporotic manifestations influenced by lifestyle. The relative impact of renotubular function, hormonal milieu, trace element and antioxidant status affecting lipoprotein metabolism and oxidizability would be determined relative to allied non-diabetic subjects. The results revealed potentiation of pathogenetic osteoporotic mechanisms by microvascular complications in diabetic passive smokers especially with sedentary lifestyle. It may be concluded that premature development of osteoporosis should be dealt with special concern to glycemic control and adjustment of hormonal milieu, trace element and antioxidant status in diabetic subject with orientation of lifestyle.

Keywords: Osteoporosis – lifestyle – smokers – type-2 diabetes mellitus – renotubular – hormonal milieu – trace element – antioxidant – lipoprotein .

INTRODUCTION

Several risk factors associated with nutritional aspects and sedentary lifestyle noted in Saudi Arabian[1] middle-aged diabetic men might be interlinked with oxidative stress[2]. Recently, growing understanding of the bone remodeling process suggests that factors involved with inflammation are linked to those critical for bone physiology and remodeling[3]. Moreover, it is known that smoking exerts a deleterious influence on the endothelium and that the mechanism is complex[4]. In reference, cigarette smoking is addictive and nicotine can exacerbate cardiac lesions[5]. A consequence of cigarette smoking exposure characterizes the negative effects of biochemical markers on vascular endothelium and antioxidant capacity in cases that have reduced physical access[6]. They may influence the continuous dynamic process of the bone remodeling cycle, which affects the coupled balance between resorption[7] and formation[8]. In this event, the outcome was noted to be implicated with compromised skeletal integrity[9] that may result from multifactorial events[10] imposed prior to the aging process[11]. In fact some epidemiological studies have referred to an estimated over 200 million cases worldwide suffering from osteoporosis[12]. The major complication of osteoporosis is an increase in fragility fractures leading to morbidity, mortality, and decreased quality of life[13]. In reference, certain health hazards of smoking may affect both active and passive smokers respective to the inhalation of emitted smoke containing cadmium, whereby its cumulative influence and deposition in bone tissues is documented to potentiate osteoporosis[14].

The present study aims to assess the amalgamated outcome of sedentary lifestyle, passive smoking and type-2 diabetes mellitus on multidisciplinary mechanisms influencing the pathogenesis of osteoporosis. Respectively, in such cases, the monitoring of selected indices would include glycemic status, vascular damage, antioxidant enzymes activity, and certain trace elements linked to oxidative stress. Also, lipoprotein metabolism and oxidizability would be evaluated to provide the coordination between variable strategies participating in early development of osteoporosis in middle-aged men.

PATIENTS AND METHODS

Thirty patients with type-2 diabetes mellitus "T2-DM" (involving 15 cases with osteoporosis "Group I" and 15 cases without "Group II") and 30 allied non-diabetic subjects (G III with and GIV without osteoporosis) were

selected from King Abdul Aziz University Hospital Outpatient Clinic.

Their age ranged from 36-48 years. They were subjected to full history taking and thorough clinical examination. They fulfilled the criteria of passive smoking respective to GI, GII, and GIII. Their exposure to environmental tobacco smoke (ETS) was evaluated by their answers to the following questionnaire[15]: "Does anybody, at present, smoke inside your home?" (Yes/No) and "Have you been regularly exposed to tobacco smoke in the last 12 months? ['Regularly' means on most days or nights]"(Yes/No). The duration of ETS exposure at follow-up was determined as reported hours/days at home, at workplace, restaurants, social gatherings, closed rooms, and/or at similar places. For analysis the sum of all responded hours was categorized in never ≤1 hr/d (not passive smokers), and >5 hrs/d (for passive smokers).

Cases with T2-DM had fulfilled the American Diabetes Association Criteria[16] that identified the presence of diabetes mellitus (DM) when the fasting or the random blood glucose levels were equal or more than 126 mg/dl and 200 mg/dl respectively (which is equal to 7 mmol/L and 11 mmol/L respectively). Definition of sedentary lifestyle includes factors such as low physical activity, ETS exposure, and high caloric dietary habits (rich in carbohydrates and lipid sores)[17]. All blood samples were collected after a fast of at least 14 hours.

Urine samples were collected on at least 3 different days and the average urinary thrombomodulin level[18], albumin excretion rate (UAE)[19] and creatinine clearance[20] were analyzed and recorded. In diabetic patients microalbuminuria was identified when UAE value was between 30-300 mg/24 hr. We excluded the subjects with UAE values ≥ 300 mg/24 hr or with overt proteinuria from this study. No subjects had tumours or other infections/disease. Radiological diagnosis of osteoporosis was done to identify decreased bone density[21], and was confirmed by bone densitometry[22,] which represents the most effective way of screening for osteoporosis[23]. Also, biochemical assessments of osteoporosis indices involved alkaline phosphatase (ALP)[24], osteocalcin (OC)[25], parathyroid hormone (PTH)[26] and urinary cyclic adenosine monophosphate (cAMP)[27].

Biochemical analysis included the following:

- Serum and urinary thrombomodulin determined by an enzyme immunoassay of Ishii *et al*.[18]
- Lipoprotein Lipase enzyme activity using a sandwich enzyme immunoassay method of Ikeda *et al*.[28]

Indices of Glycemic control involved:

- Fasting plasma glucose (by enzymatic method using the commercial kit provided by Sigma Chemical Company, Los Angles, California, USA.[29]
- Glycated hemoglobin (HbAlc) was assayed by ion exchange chromatography using the method of Maquart *et al*.[30]

Indices of Tubular Glomerular and Renal Function involved:

- Urinary thrombomodulin (UTM).[18]
- Urinary albumin excretion (UAE) was assayed by immunoturbidimetric method of Teppo[19].
- Serum creatinine measured colourimetrically using the method of Perrone *et al*.[31].
- Creatinine clearance was estimated by the method of Cockcroft and Gault [20.]

  Serum level of trace elements by atomic absorption spectroscopy included zinc[32], copper [32], cadmium[33] and selenium[34.]

Lipid profile: involved serum level of triglyceride[35], total cholesterol[36] and plasma lipoproteins[37] by enzymatic colourimetric method using the kits of Biomerieux France. Also lipid peroxidation product was estimated via thiobarbituric acid reactive substance TBARS[38]. Antioxidant enzymatic evaluation of superoxide dismutase (SOD) and glutathione peroxidase (GSHPx)[39] was performed.Assessment of serum dehydroepiandrosterone sulphate (DHEA-S)[40], insulin[41], insulin growth factor (IGF-1)[42] and insulin growth factor binding protein -3 (IGFBP-3)[43] and testosterone[44] were assessed by radioimmunoassay.

STATISTICS

Results are represented as means ±S.D. The statistical analysis of the data was carried

out using SPSS Package Version 11.5. Percentage of change between each group and the control group was made using Student "t"-test between the different groups.

ETHICAL CONSIDERATION

All patients involved in this research had been informed about the purpose of this study and had the right to discontinue participation without any effect on the medical care being given to them or any further follow up.

RESULTS AND DISCUSSION

In view of the present findings (Table 1), the magnitude of vascular damage in T2-DM passive smokers coordinated with increments of serum and urinary thrombomodulin (TM), HbAlc, and insulin (Table 3) verifying less efficient glycemic control. This was furthermore associated with sedentary lifestyle that predisposed to premature osteoporosis as shown in (Table 2). Hence, vascular damage developed owing to the dual influence of T2-DM and passive smoking, which subsequently potentiated oxidative stress (Table 5) that had a detrimental impact on bone health. This was reflected by the decrease in bone mass and loss of physical fitness that was observed in clinical findings herein GI> GII. This was in agreement with previous studies[45,46] showing the release of TM from endothelial cells into the plasma in response to vascular injury as occurring herein. It could be influenced by impaired renal clearance of TM [47,48], which may be attributable to the retention of the increased release of this small molecular weight degraded form of the TM. Thus, it may be derived from the release of TM by systemically damaged cells[48] especially from glomeruli affected by diabetic microangiopathy[49]. This was potentiated furthermore by passive smoking and sedentary lifestyle. Such an aspect was confirmed in T2-DM cases herein (Table 1) by the monitored increase in urinary albumin excretion (UAE) versus the decreased level of urinary TM (GI> GII> GIII> GIV) coinciding with tubulorenal disposition[50, 51].

Consistently, reduced bone density was noted in T2-DM cases whereby insulin – sensitizing nutrient chromium picolinate was indicated to reduce the rate of bone resorption[52] as well as to raise the level of DHEA-S which is known to play a role in preservation of bone density[41]. Such an interrelationship was verified herein (Table 3) by assessed decrements of both chromium and antioxidant DHEA-S, relative to the monitored alterations in osteoporotic indices (Table 2) and to trace element disbalance (Table 5). Apparently in cases with a sedentary lifestyle consuming a high caloric diet with poor antioxidant nutrients (due to low intake of vegetables and fruits), the relationship between osteoporosis and decreased antioxidant potency was illustrated more profoundly in T2-DM passive smokers. These cases exhibited higher increments of serum osteocalcin, urinary cAMP, and hyperparathyroidism (Table 2). This aligns with reported data[53] identifying a notable increase in (PTH) with lower levels of vitamin D in older subjects with hip fractures. Accordingly, the present data (Table 2) linked premature osteoporosis in T2-DM passive smokers to alterations in levels of osteocalcin, vitamin D and PTH (GI> GII> GIII), which was related elsewhere to bone loss[54,55]. Our data also confirms the influence of sedentary lifestyle in these cases on the activation of osteoclasts by PTH-mediated mechanisms. It implicates the role of PTH – induced rise in protein Kinase C activity whereby the physiological levels of insulin may reduce the ability of PTH to activate protein kinase C in osteoblasts[52]. This suggests that insulin may act as a physiological antagonist of bone resorption[56], thereby influencing osteoporotic mechanisms.

In harmony with reported data[57] on the mitogenic effect of insulin on endothelial smooth muscle cell proliferation[58], the endothelial barrier was noted to result in the proliferation of endothelial cells and generation of free radicals[59]. This was verified herein (Table 5) by the lower antioxidant enzymatic activity of superoxide dismutase (SOD) and glutathione peroxides (GSHPx) and lower values of DHEA-S (Table 2), as noted elsewhere.[2] Evidently, DHEA-S acts as both an antioxidant and androgenic precursor[60], which verifies the relationship of the assessed reduction in both the levels of DHEA-S and testosterone (Table 3) influenced by the mutual impact of T2-DM and sedentary lifestyle with passive smoking. In

parallel, the monitored decrease in testosterone (G1> GII > GIII vs. GIV) could also reflect the relationship between the magnitude of oxidative stress, impaired gonadal function[61] and glycemic control,[62] reflecting the interrelationship between osteoporosis risk factors[3,52] as noted elsewhere. Concordingly, the assessed increments of IGF-1 in such cases (Table 3) alongside the relative decrements of IGF- BP-3 (GI > GII > GIII > GIV) verifies a lower binding affinity to IGF-1 similar to previous reports[63]. In fact such an aspect would subsequently increase the bioavailability of IGF-1, substantiating the magnitude of oxidative stress as verified herein (Table 4) and previously illustrated in passive smoker subjects[64]. This was furthermore confirmed (Table 4) by assessed increments of the levels of thiobarbituric acid reactive substances (TBARS) and agrees with previous reports[65].

Evidently, the lower lipoprotein lipase activity in passive smoker T2-DM sedentary states monitored herewith (Table 4) aligns with dyslipidemia and glycemic status colinked with the magnitude of vascular damage. Mechanistically, it may be related to the attachment of lipoprotein lipase (LPL) to heparin sulfate proteoglycan (HSPG) moieties on the luminal surface of endothelial cells that readily hydrolyze triglyceride-rich lipoproteins[66]. In confirmation, increments of triglycerides were found in parallel to decrements of LPL herein (GI> GII> GIII vs. GIV). Thus, when generalized endothelial damage occurs by the collective impact of passive smoking, sedentary lifestyle and T2-DM, the extent of HSPG on the endothelial surface becomes decreased, coinciding with previous reports[5]. Subsequently, a lesser amount of the LPL moiety can attach to the luminal surface[67] emphasizing its interlinks with altered lipoprotein metabolism and appearing more profound in diabetic passive smoker sedentary states. (Table 4). Our data agrees with reports indicating a lower LPL mass in microalbumineric subjects that correlated inversely with HDL-C suggesting the critical role of LPL in dyslipidemia associated with diabetic complications[68]. Hence LPL, via its action on circulating fasting and postpranolial lipoproteins, is generally viewed as an antiatherogenic enzyme[69] and is linked with lipoprotein oxidizability[70]. Our data would align with the cumulative impact of DM-2 and smoking verified in other studies[71, 72].

Hence, T2-DM passive smoker cases had presented uncontrolled glycemic status (increments in HbAlc and insulin), with dyslipidemia and oxidative stress (higher levels of cholesterol, LDL-C, TG, VLD-C, and TBARS) versus lipoprotein oxidizability, hypoandrogenemia and lower antioxidant enzyme activity (lower levels of LPL, testosterone, HDL-C, DHEAS, SOD and GSHPx) as monitored herein (GI > GII > GIII > GIV). It reflects a higher value of atherogenic index influenced by passive smoking and the sedentary lifestyle of the diabetic cases under study. This interlinks the complex influence of hyperglycemia, oxidative stress and hypoandrogenemia to the development of dyslipidemia, lipoprotein oxidizability. In this context, these interactions emphasize the role of LPL in T2-DM patients who have microalbuminurea manifestations. This designates the significance of this interplay, which led many reports to identify the link between decreased LPL release and vascular damage [71,74,75,76].

The present data elaborates the coordination between the above-mentioned profile with disbalance in trace elements (Table 5) and osteoporotic indices (Table 2). Furthermore, our data illustrates how that could be influenced by life style, implicating the harmony between multidisciplinary mechanisms operating in response to unintentional cigarette smoke inhalation from the surrounding environment and the sedentary life of diabetics. It coincides with oxidative modification of LDL-c, which is a key event in the oxidation hypothesis of atherogenesis[75].

It has been suggested that a possible deficiency of certain trace elements such as chromium, zinc, selenium, and magnesium may be behind the decreased activity of antioxidant enzymes (such as SOD and GSHPx) [76,77,78] in diabetics.

Evidently, the decremental magnitude of change in serum zinc and selenium versus incremental values of serum copper and cadmium in diabetic sedentary states (Table 5)

reflects also the cumulative impact of passive smoking with T2-DM, aligning with previous data[79, 80]. It agrees with reports relating the appearance of bone lesions, osteoporosis and osteomalacia in cadmium exposed subjects[81] and formation of a high molecular weight biologically inactive complex of cadmium – selenium whereby consumption of selenium serves to protect against cadmium toxicity[14]. This was linked to observations that cadmium accumulates in bone and is associated with osteomalacia and osteoporosis[82,83]. Thereby in diabetic passive smokers, assessed increments of cadmium may replace zinc in metabolic enzymes influencing energy balance and intermediary metabolism in agreement with previous reports[84].

In conclusion, the present findings raise awareness of the influence of lifestyle (lack of exercise, passive smoking and nutritional aspects) and T2-DM on the premature development of osteoporosis in middle-aged Saudi men. Moreover, the diagnostic validity of monitoring glycemic control and gonadal hormonal levels alongside trace elements and antioxidant status proved to be a reliable follow up means for screening of osteoporotic manifestation in young patients with T2-DM. This may implement socioeconomic benefits in a trial to prevent the degenerative process, especially in subjects with a sedentary life style and vascular disposition that is reflected in a reduced physical self-concept that should be worthy of concern.

Table 1: Indices of Glycemic Control Vascular Damage and Glomerular and Renal Function.

	Diabetic Cases		Non-Diabetic Case	
	Sedentary G I	**Active G II**	**Sedentary G III**	**Active G IV (Control)**
Glycated Hemoglobin (HbAlc %)	11.17 ± 2.7	10.33 ± 2.5	6.85 ± 0.83	5.77 ± 0.9
% change	48.34	44.14	15.77	
t_1 (p)	0.88 (p>0.05)		3.42** (p<0.01)	
t_2 (p)	5.92** (p<0.01)	6.65** (p<0.01)		
Serum Thrombomodulin (U/ml)	26.80 ± 7.0	23.10 ± 5.8	18.70 ± 4.9	16.10 ± 4.1
% change	39.93	30.30	13.90	
t_1 (p)	1.58 (p>0.05)		1.58 (p>0.05)	
t_2 (p)	3.67** (p<0.01)	3.82** (p<0.01)		
Urinary Thrombomodulin (U/mg Creatinine)	51.4 ± 14.2	40.1 ± 12.6	30.1 ± 9.2	23.6 ± 8.1
% change	54.09	41.15	21.59	
t_1 (p)	2.31* (p<0.05)		2.05* (p<0.05)	
t_2 (p)	4.88** (p<0.01)	4.27** (p<0.01)		
Serum Creatinine (mg/dl)	1.31 ± 0.4	1.11 ± 0.3	0.82 ± 0.2	0.61 ± 0.15
% change	53.44	45.05	25.61	
t_1 (p)	1.55(p>0.05)		3.25** (p<0.01)	
t_2 (p)	4.24** (p<0.01)	5.77** (p<0.01)		
Urinary albumin excretion (mg/24h.)	70.8 ± 19.7	48.9 ± 17.3	34.6 ± 12.3	27.01 ± 7.3
% change	61.85	44.76	21.94	
t_1 (p)	3.24** (p<0.01)		2.06* (p<0.05)	
t_2 (p)	6.04** (p<0.01)	4.52** (p<0.01)		
Creatinine Clearance (ml/min)	81.4 ± 19.1	99.3 ± 21.6	111.7 ± 32.4	129.3 ± 30.4
% change	-58.85	-30.21	-15.76	
t_1 (p)	2.40* (p<0.05)		1.53 (p>0.05)	
t_2 (p)	3.12** (p<0.01)	3.12** (p<0.01)		

(Values expressed as Mean ± S.D)

t_1(p) t-test and significance of sedentary and active in both diabetic and non-diabetic groups

t_2(p) t-test and significance of sedentary and active in diabetic group with control group

* Statistically significant at p < 0.05

** Statistically significant at p < 0.01

Table 2: Indices Osteoporotic Manifestations.

	Diabetic Cases		Non-Diabetic Case	
	Sedentary G I	**Active G II**	**Sedentary G III**	**Active G IV (Control)**
Osteocalcin (µg/L)	6.1 ± 1.4	5.3 ± 1.1	4.5 ± 1.01	3.3 ± 0.9
% change	45.90	37.74	26.67	
t_1 (p)	1.74 (p>0.05)		3.44** (p<0.01)	
t_2 (p)	3.59** (p<0.01)	5.45** (p<0.01)		
Parathyroid hormone (pg/ml)	96.4 ± 19.1	84.0 ± 17.1	76.1 ± 14.5	63.4 ± 12.6
% change	34.23	24.52	16.69	
t_1 (p)	1.87(p>0.05)		2.56* (p<0.05)	
t_2 (p)	3.28** (p<0.01)	3.76** (p<0.01)		
Alkaline Phosphatase (U/L)	138.0 ± 27.0	122.0 ± 22.0	111.0 ± 24.0	87.0 ± 17.0
% change	36.96	28.69	21.62	
t_1 (p)	1.78(p>0.05)		3.16** (p<0.01)	
t_2 (p)	2.89** (p<0.01)	4.88** (p<0.01)		
Urinary Cyclic Adenosine monophosphate (cAMP/100ml GF)	6.4 ± 1.20	5.7 ± 1.10	4.9 ± 1.10	3.4 ± 1.01
% change	64.88	40.35	30.61	
t_1 (p)	1.67 (p>0.05)		3.89** (p<0.01)	
t_2 (p)	3.57** (p<0.01)	5.97** (p<0.01)		

(Values expressed as Mean ± S.D)

t1(p) t-test and significance of sedentary and active in both diabetic and non-diabetic groups

t2(p) t-test and significance of sedentary and active in diabetic group with control group

* Statistically significant at $p < 0.05$

** Statistically significant at $p < 0.01$

Table 3: Selected Hormonal Milieu in Group Under Study.

	Diabetic Cases		Non-Diabetic Case	
	Sedentary G I	**Active G II**	**Sedentary G III**	**Active G IV (Control)**
Insulin Growth Factor (ng/ml)	251.0 ± 39.0	220.0 ± 36.0	181.0 ± 24.0	154.0 ± 21.0
% change	38.65	30.00	14.92	
t_1 (p)	2.26* (p<0.05)		3.28** (p<0.01)	
t_2 (p)	5.92** (p<0.01)	6.13** (p<0.01)		
Insulin Growth Factor Binding Protein -3	2.52 ± 0.62	2.96 ± 0.71	3.64 ± 0.87	5.7 ± 1.3
% change	-126.19	-92.57	-56.59	
t_1 (p)	1.81(p>0.05)		5.10** (p<0.01)	
t_2 (p)	4.06** (p<0.01)	7.16** (p<0.01)		
Fasting plasma Insulin (IU/ml)	18.6 ± 5.3	16.2 ± 4.21	12.9 ± 3.7	8.6 ± 2.1
% change	53.76	46.91	33.33	
t_1 (p)	1.37(p>0.05)		3.91** (p<0.01)	
t_2 (p)	3.42** (p<0.01)	5.10** (p<0.01)		
DHEAS (ug/dl)	144.0 ± 49.0	186.0 ± 64.0	301.0 ± 117.0	378.0 ± 131.0
% change	-162.50	-103.23	-25.58	
t_1 (p)	2.02(p>0.05)		1.70(p>0.05)	
t_2 (p)	4.79** (p<0.01)	5.10** (p<0.01)		
Total Testosterone (nmol/l)	16.3 ± 4.2	18.6 ±3.9	20.4 ± 4.7	21.6 ± 5.2
% change	-32.52	-16.13	-5.88	
t_1 (p)	1.55(p>0.05)		0.66(p>0.05)	
t_2 (p)	2.52* (p<0.05)	1.79(p>0.05)		

(Values expressed as Mean ± S.D)

t1(p) t-test and significance of sedentary and active in both diabetic and non-diabetic groups

t2(p) t-test and significance of sedentary and active in diabetic group with control group

* Statistically significant at p < 0.05

** Statistically significant at p < 0.01

Table 4: Lipid Profile and Indices of Lipoprotein Metabolism and Oxidizability.

	Diabetic Cases		Non-Diabetic Case	
	Sedentary G I	**Active G II**	**Sedentary G III**	**Active G IV (Control)**
Lipoprotein Lipase (U mol/hr/ml)	6.19 ± 1.17	7.25 ± 2.5	8.85 ± 2.83	11.07 ± 2.9
% change	-78.84	-52.69	-25.08	
t_1 (p)	1.49(p>0.05)		2.12* (p<0.05)	
t_2 (p)	3.6** (p<0.01)	3.86** (p<0.01)		
Triglyceride (m mol/L)	2.86 ± 0.94	2.24 ± 0.92	1.66 ± 1.59	1.32 ± 0.51
% change	53.85	41.07	20.48	
t_1 (p)	1.83(p>0.05)		0.79(p>0.05)	
t_2 (p)	2.52* (p<0.05)	3.39** (p<0.01)		
LDL-C (m mol/L)	4.16 ± 1.33	3.87 ± 1.26	2.54 ± 0.9	2.18 ± 0.83
% change	47.60	43.67	14.17	
t_1 (p)	0.61(p>0.05)		1.14(p>0.05)	
t_2 (p)	3.91** (p<0.01)	4.34** (p<0.01)		
VLDL-C (m mol/L)	0.49 ± 0.19	0.43 ± 0.17	0.35 ± 0.09	0.28 ± 0.08
% change	42.86	34.88	20.00	
t_1 (p)	0.91(p>0.05)		2.25* (p<0.05)	
t_2 (p)	2.58* (p<0.05)	3.09** (p<0.01)		
HDL-C (m mol/L)	0.74 ± 0.21	0.79 ± 0.24	1.02 ± 0.9	1.14 ± 0.23
% change	-54.05	44.30	-11.76	
t_1 (p)	0.61(p>0.05)		0.50(p>0.05)	
t_2 (p)	1.17 (p>0.05)	4.08** (p<0.01)		
TBARS (m mol/L)	7.01 ± 1.67	6.3 ± 1.52	5.46 ± 0.97	4.89 ± 0.091
% change	30.24	22.38	10.44	
t_1 (p)	1.22(P>0.05)		2.27* (p<0.05)	
t_2 (p)	3.11** (p<0.01)	3.59** (p<0.01)		

(Values expressed as Mean ± S.D)

t_1(p) t-test and significance of sedentary and active in both diabetic and non-diabetic groups

t_2(p) t-test and significance of sedentary a nd active in diabetic group with control group

* Statistically significant at p < 0.05

** Statistically significant at p < 0.01

Table 5: Serum Level of Trace Elements Antioxidants.

	Diabetic Cases		Non-Diabetic Case	
	Sedentary G I	**Active G II**	**Sedentary G III**	**Active G IV (Control)**
Selenium (µg/dl)	83.0 ± 27.0	91.0 ± 30.0	177.0 ± 33.0	142.0 ± 39.0
% change	-71.08	-56.04	19.77	
t_1 (p)	0.77 (p>0.05)		2.65* (p<0.05)	
t_2 (p)	8.54** (p<0.01)	4.01** (p<0.01)		
Zine (µg/dl)	70.0 ± 18.0	84.0 ± 21.0	97.0 ± 27.0	118.0 ± 27.0
% change	-68.57	-40.48	-21.65	
t_1 (p)	1.96(p>0.05)		2.13* (p<0.05)	
t_2 (p)	3.22** (p<0.01)	3.85** (p<0.01)		
Copper (µg/dl)	150.7 ± 41.0	133.0 ± 36.0	121.0 ± 31.0	107.0 ± 26.0
% change	29.00	19.55	11.57	
t_1 (p)	1.26(p>0.05)		1.34(p>0.05)	
t_2 (p)	3.24** (p<0.01)	2.27* (p<0.05)		
Cadmium (µg/dl)	0.77 ± 027	0.68 ± 0.21	0.53 ± 0.17	0.4 ± 0.15
% change	48.05	41.18	24.53	
t_1 (p)	0.01(p>0.05)		2.22* (p<0.05)	
t_2 (p)	0.03(p>0.05)	4.20** (p<0.01)		
SOD (µ/mg Hb/min)	1.74 ± 0.61	191 ± 0.97	2.4 ± 0.84	2.97 ± 1.1
% change	-70.69	98.45	-23.75	
t_1 (p)	639.69** (p<0.01)		1.60(p>0.05)	
t_2 (p)	2.46* (p<0.05)	496.55* (p<0.01)		
GSH Px (µ/g Hb min)	22.7 ± 7.6	25.34 ± 8.4	37.1 ± 12.1	42.6 ± 12.7
% change	-87.67	-68.11	-14.82	
t_1 (p)	0.90(p>0.05)		1.21(p>0.05)	
t_2 (p)	3.90** (p<0.01)	4.39** (p<0.01)		

(Values expressed as Mean ± S.D)

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t2(p) t-test and significance of sedentary and active in diabetic group with control group

* Statistically significant at $p < 0.05$

** Statistically significant at $p < 0.01$

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