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A study of the levels of oxidative stress status in sera among Iraqi patients with type 2 diabetes

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***Abstract*---**Background: Chronic hyperglycaemia and cluster of lipid abnormalities associated with Type 2 diabetes (T2D), is defined by decreases in high-density lipoprotein (HDL) and increases in triglyceride (TG) and small, dense low-density lipoprotein (LDL) concentrations. Type 2 diabetes mellitus (T2DM) leads to increased lipid peroxidation in the body, followed by the development of chronic complications due to oxidative stress. The aim of this study was carried out to compare total antioxidant (TAO) levels and oxidative stress in type 2 diabetes mellitus (T2DM) patients with that of healthy controls without diabetes. Methods: A total of 93 participants (68 T2DM divided into three groups: group I (19) patients with diabetic duration (<5 years), group II (19) patients with diabetic duration (≥5>10 years), group III (30) patients with diabetic duration (≥10 years) and 25 healthy people) gave their consent and participated in the study. Thus, to achieve that, total oxidant status (TOS), total antioxidant status (TAS) and oxidative stress index (OSI), as well as the levels of fasting plasma glucose (FPG), glycosylated haemoglobin (HbA1c) and lipid profile were measured in patients group and the healthy individuals. Results: Upon the comparison between patients group with different duration of diabetic and the control sample the results showed a significant rise in FPG, HbA1c, triglycerides, cholesterol, VLDL and a significant reduction in TAO and high-density lipoprotein cholesterol (HDL-C). Increased lipid peroxidation and reduced antioxidant levels were observed in T2DM patients. Conclusion: Early management through an antioxidant-rich diet and lifestyle changes in T2DM patients would help to avert the debilitating complications of diabetes.

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Keywords---oxidative stress, Iraqi patients, diabetes, antioxidant.

Introduction

Diabetes mellitus (DM) is a serious of metabolic disorders characterised by hyperglycaemia (abnormally elevated blood glucose levels)[1] which arise from the body's inability to use insulin to its full potential or to produce it [2]. DM is a lifetime progressive metabolic disease and according to International Diabetic Foundation (IDF) which is estimated that 537 million people have diabetes in 2021, and this number is projected to reach 643 million by 2030, and 783 million by 2045 [3]. DM is the reason for death universally and has become a difficult health problem for the twenty-first century [4]. Type 2 diabetes is the most common type of diabetes, it accounts for about 90% of diabetes [5] and is expected to affect almost 8% of the world Population by 2030 [6]. Type 2 diabetes is associated with numerous pathological complications, such as microvascular (neuropathy, nephropathy, retinopathy and myopathy) and macrovascular disease [7]. Myopathy (Diabetic skeletal muscle disease) is the less studied complication of poorly controlled diabetes [8,9]. Accounting risk factors for developing type 2 diabetes (T2DM) are: Older age, high plasma glucose, metabolic syndrome, high body mass index (BMI), and high glycated haemoglobin A1(HbA1c > 6%) [10,11,12]. Recently around 1.4 million of Iraqis have diabetes, reported T2DM prevalence in Iraq ranges from (8.5%) (IDF-age-adjusted) to (13.9%) [13]. Oxidative stress status is accountable for the development of chronic complications of DM and results from chronic hyperglycaemia, dyslipidaemia and elevated fatty acids in the circulation [14,15]. There are two reactive species: reactive oxygen species (ROS) and reactive nitrogen species (RNS) [16]. ROS such as (superoxide, hydroxyl radical, peroxyl radical and hydroperoxyl radical) and the non-radical ROS such as (hydrogen peroxide and hypochlorous acid), while RNS include nitric oxide, nitrogen dioxide, peroxynitrite and nitrous oxide [17,18]. Under normal physiological conditions most of the free radicals are produced in small amounts and scavenged by the endogenous antioxidant system. The antioxidant system includes molecules like (GSH, uric acid, albumin, bilirubin, lipoic acid, vitamin (E& C), transferrin, carotenoid, copper and zinc) and enzymes like (SOD, catalase, glutathione peroxidase) [19]. By several mechanisms antioxidants scavenge the free radicals. The enzymes degrade free radicals and vitamins C & E act as free radical scavengers. Vitamin E, a lipid soluble vitamin, interferes with chain reactions of lipid peroxidation. while vitamin C is a water-soluble molecule and usually scavenges hydroxyl radicals [9]. All the aerobic cells can produce the reactive oxygen and nitrogen species (RONS).[20] Therefore, it leads to OS because of overproductions and/or an impaired ability to neutralize them or repair the resulting damage to a wide range of macromolecules i.e., lipids, proteins, and nucleic acids [16]. Several other mechanisms are also implicated in the generation of oxidative stress (the imbalance occurs between production and removal of RONS) due to hyperglycaemia in diabetes. These mechanisms include glucose auto-oxidation, increased glucose flux through the polyol pathway, non-enzymatic and progressive glycation of proteins [21]. An increase in the polyol pathway depletes the NADPH, which is utilised by the enzyme aldose reductase that catalyses the conversion of glucose to sorbitol. Numerous studies have reported

increased oxidative stress in type 2 diabetes mellitus patients compared with their healthy counterparts [22,23,24].

Materials and Methods

Study Subjects

A total of 93 individuals participated in the current study divided into three groups: group I (19) patients with diabetic duration (<5 years), group II (19) patients with diabetic duration (≥5>10 years), group III (30) patients with diabetic duration (≥10 years) and 25 age and gender matched control group. Each group consists of 16 males and 14 females with ages ranging between 25to 75 years and a body mass index (BMI) ranging between 19 to 49 kg/m^2 . The exclusion criteria included: Patients who have had type 1 diabetic mellitus (T1DM) and other diabetic complication such as Patients on drugs which cause muscle injury or increase CK (e.g., Statins and Amiodarone), chronic renal failure, Cushing's disease, acromegaly, chronic pancreatitis, pancreatectomy, malignancies as well as history of smoking and alcohol drinking were excluded from the study. All samples were collected from patients attending AL-Yarmouk Teaching Hospital, and National Diabetes Center for Treatment and Research, Al Mustansiriya University, who were selected by the supervising specialist to be free of diabetic complications. The study protocol was approved by the Ethics-Committee of Department of Chemistry College of Science for Women University of Baghdad.

Seven to ten milliliters of venous blood samples were collected from patients and controls groups. The samples were taken at the time between 8.00 and 11.00 A.M after 12-15 hours of fasting. The blood was immediately divided into two portions, the first one (2.5ml) was transferred into Ethylene diamine tetra acetic acid EDTA tube, then the blood samples were stirred gently for few seconds to avoid blood clotting and the tubes were transported to the laboratory using cooling containers and stored at 4℃ (2days) for HbA1c. While the second one was used for serum collection by polyethylene tube the blood sample in a plain tube were allowed to clot for ten minutes at (37 ℃) in a water bath, then were centrifuged at (3000 x g) for 10 minutes. The collected serum was divided into several parts each part content (500 µl) and serum was stored frozen at (-20 ℃) until being used to estimate the different parameters included in the study. Hemolysed samples were discarded.

Experimental Fasting blood glucose (FBG) was determined by enzymatic colorimetric method using kit manufactured by Linear Chemicals based on Trinder reaction [25,26]. Hemoglobin $A1_c$ in blood was determined by immunoassay system an i-Chroma™ Hb A1c kit for fluorescence immunoassay technology. The Erel's methods were used to determine both TOS (µmol H_2O_2 Eq./L)20 and TAS (mmol uric acid Eq/l) [27] in serum samples (patients and control). The OSI which is an indicator of degree of OS was calculated according to the formula: OSI = TOS (µmol H_2O_2 Eq. / L) / TAS (µmol uric acid Eq. / L) [28]. Serum concentrations of total cholesterol, high-density lipoprotein cholesterol (HDL-cholesterol), and triglycerides were measured using routine enzymatic methods [29,30,31,32]. Low density lipoprotein cholesterol (LDL-cholesterol) levels were calculated using Friedewald's equation [33].

Statistical Analysis

Statistical Process for Social Sciences (SPSS) program version 24 was used for analysis the data of the present study. The comparisons between control and DM patients were performed by using independent sample t-test. Analysis of variances (ANOVA) test was used for the comparisons between control and the subgroups of patients. Post-Hoc least significant differences (LSD) test was used to obtain the significance between each two groups, this post-Hoc test was selected owing to the inequality of samples per groups. Chi-square test was used for the comparison of percentages. The association between variables in each subgroup of patients was examined by using Pearson's coefficient (r)

Results

Table 1 shows the anthropometric characteristics in the Patient and control group. The mean age was comparable across groups and showed a significant increase (p = 0.0001). The mean BMI for groups was high, with T2DM patients having higher BMIs compared with individuals in the healthy control group; the difference was statistically significant increase (p = 0.0001). Regarding the duration of diabetes, long duration of diabetes mellitus (DM) is associated with an increased risk of infection, table 1 also shows values of (mean ± SD) of diagnosis for duration of T2DM for all the studied groups. The results revealed a significant increase (p=0.0001) in group I (2.05±1.08), group II (6.53±1.68) and group III (14.60±4.28).

Table 1: Demographic characteristics of diabetic groups and control participants

Characteristics		Control	Patients I	Patients II	Patients III	*p*-value
Number		25	19	19	30	-
Age (year)	Mean±SD	45.96±10.71	49.47±9.63	52.26±8.37	57.40±8.33	0.0001*
	Min-Max	25-70	32-62	30-67	40-75	
Duration	Mean±SD	-	2.05±1.08	6.53±1.68	14.60±4.28	0.0001*
	Min-Max	-	1.0-4.0	5.0-9.0	10.0-30.0	
BMI	Mean±SD	22.71±2.10	30.19±7.17	26.39±3.41	28.26±3.94	0.0001*
	Min-Max	19.34-29.41	22.04-49.60	21.48-35.16	23.04±44.11	

Biochemical characteristics of participants are presented in table (2 and 3). FPG and HbA1c were significantly higher in T2DM patients compared with those of persons in the control group (p =0.0001). The FBG levels showed highly significant (p <0.05) when comparison between the results of (group I and group II) type 2 diabetic patients with (group III) type 2 diabetic patients. While, no significant results obtained when comparison between the results of group I with group II type 2 diabetic patients

Table 2: Serum glucose (Mean ± SD) in Patients and control

Groups	Mean±SD	Min-Max.	*p*-value		
			I	II	III
Control	100.52±16.82	78.0-137.0	0.001*	0.001*	0.0001*
Patients I	168.0±45.50	90.0-271.0	-	0.923	0.008*
Patients II	166.04±35.65	100.0-243.0	0.923	-	0.006*
Patients III	217.95±97.82	91.0-526.0	0.008*	0.006*	-

Table 3: HbA1c (%) in Patients and control

Groups	Mean±SD	Min-Max.	*p*-value		
			I	II	III
Control	3.90±0.57	3.20-5.0	0.0001*	0.0001*	0.0001*
Patients I	7.26±2.51	4.80-16.0	-	0.923	0.076
Patients II	7.31±1.38	5.50-10.40	0.923	-	0.094
Patients III	8.15±1.87	5.10-12.60	0.076	0.094	-

Table 3 shows (mean ± SD) of glycated haemoglobin for all the studied patient groups in addition to control group [(7.26±2.51), (7.31±1.38), (8.15±1.87) and (3.90±0.57)] respectively. The above results show that there is a significant increase (p =0.0001) in the level of HbA1c% when comparison between the results of type 2 diabetic patients group (I, II, III) with control group according to LSD test, while no significant in the level of HbA1c% when comparison between the results of type 2 diabetic patients group (I, II, III) themselves.

Table 4: Mean ± SD value of TG, TC, LDL-C, HDL-C and VLDL for the studied groups

Parameters	Groups	Mean ± SD	Min-Max.	*p*-value		
				I	II	III
TG	Control	90.03±26.66	38.81-160.63	0.019*	0.0001*	0.0001*
	Patients I	130.08±67.91	67.50-273.12	-	0.134	0.004*
	Patients II	157.19±42.08	92.0-213.54	0.134	-	0.195
	Patients III	178.37±69.55	75.67-332.85	0.004*	0.195	-
TC	Control	158.71±27.79	100.0-204.0	0.018*	0.001*	0.015*
	Patients I	192.93±64.37	96.0-335.86	-	0.371	0.825
	Patients II	206.54±45.23	129.11-304.56	0.371	-	0.227
	Patients III	189.90±46.81	110.0-296.88	0.825	0.227	-
HDL-C	Control	65.15±47.36	28.70-188.60	0.171	0.424	0.863
	Patients I	51.91±14.34	30.0-90.34	-	0.589	0.115
	Patients II	59.61±30.28	33.98-143.39	0.589	-	0.323
	Patients III	50.44±30.24	28.32-154.71	0.115	0.323	-
LDL-C	Control	84.91±20.66	47.88-123.53	0.330	0.349	0.047*
	Patients I	98.88±65.31	11.0-278.17	-	0.972	0.399
	Patients II	98.34±55.06	11.65-213.84	0.972	-	0.377
	Patients III	110.54±43.19	53.25-200.64	0.99	0.377	-
VLDL	Control	19.50±4.96	11.0-32.13	0.035*	0.0001*	0.0001*

	Patients I	26.95±13.80	14.0-54.62	-	0.015*	0.003*
	Patients II	36.20±10.24	18.0-66.57	0.015*	-	0.763
	Patients III	37.21±14.09	15.0-66.57	0.003*	0.763	-

According to LSD test the group I of patient showed significant difference ($p<0.05$) in triglyceride levels when compared with control group, while group II&III show significant increase ($p=0.0001$) when compared with control group, also no significant difference of mentioned parameter among patient's groups themselves ($p>0.05$) as shown in table 4. Also the results of TC and VLDL-C showed significant increase ($p<0.05$) between patients group and control, at the same showed significant increase ($p<0.05$) of VLDL when compared with control group, while patient group show no-significant differences ($p>0.05$) compared between them group, also no significant difference of mentioned parameters among patient groups themselves ($p>0.05$). The results of HDL-C in Table 4 also showed that there was significant difference ($p<0.05$) in HDL-C level between the studied groups. showed significant decrease ($p<0.05$) when compared with control group, while group show no-significant differences ($p>0.05$) compared with control group, also there is no-significant differences ($p>0.05$) between patient groups themselves. The results in Table 4 also showed significant difference ($p<0.05$) in LDL-C level between the T2DM group III and control groups, while no-significant differences ($p>0.05$) between T2DM (group II and group I) themselves, when compared to control.

Table 5: The (mean ± SD) of TOS and TAS in Patients and control

	Groups	Mean±SD	Min-Max.	*p*-value		
				I	II	III
TOS	Control	34.09±9.76	20.23-50.50	0.0001*	0.005*	0.0001*
	Patients I	53.12±16.72	29.56-89.70	-	0.207	0.756
	Patients II	47.55±13.74	30.90-84.56	0.207	-	0.389
	Patients III	51.75±17.80	29.0-82.89	0.756	0.389	-
TAS	Control	11.72±1.51	9.08-14.29	0.001*	0.0001*	0.0001*
	Patients I	9.92±2.01	6.89-13.78	-	0.482	0.014*
	Patients II	9.52±2.03	5.67-12.89	0.482	-	0.127
	Patients III	8.56±1.58	6.45-11.23	0.014*	0.127	-

Table 6: OSI in Patients and control

Groups	Mean±SD	Min-Max.	*p*-value		
			I	II	III
Control	0.30±0.09	0.18-0.51	0.0001*	0.0001*	0.0001*
Patients I	0.56±0.20	0.24-0.97	-	0.788	0.388
Patients II	0.54±0.25	0.29-1.08	0.788	-	0.322
Patients III	0.61±0.19	0.26-0.96	0.388	0.322	-

Oxidative stress is one of the parameters which was studied in serum of different types of diabetes mellitus by measuring individual oxidant and antioxidant parameters. Determination of TOS and TAS suggested to be better indicator of the presence of OSI, its use is likely to be more important than TAS and TOS values

[34]. Therefore, in the current study oxidative stress status were measured using TOS and TAS in serum samples of the Iraqi patients and the results are illustrated in table 5. Upon the comparison between patients group and the control sample, the results show that: significant increase (p = 0.0001) of TOS and OSI in sera samples in table 5 and 6, while there is a significant decrease (p < 0.001) of TAS in sera samples table 5. Comparison of the results of different patients group with diabetes mellitus showed an evident of no significant increase (p > 0.05) in TOS, TAS and OSI.

Table 7: Correlation between variables in Patients group I

Variable	TOS		TAS		OSI	
	R	*P*	r	*P*	R	*P*
TOS	-	-	0.375	0.113	0.857**	0.0001
TAS	0.375	0.113	-	-	-0.131	0.592
OSI	0.857**	0.0001	-0.131	0.592	-	-
Age	-0.428	0.068	-0.197	0.419	-0.283	0.240
BMI	-0.418	0.075	-0.130	0.596	-0.384	0.105
Duration	0.252	0.297	-0.050	0.839	0.305	0.204
Glucose	0.068	0.782	-0.101	0.680	0.135	0.583
HbA1c	0.054	0.826	0.176	0.471	-0.053	0.830
Tri	0.125	0.611	-0.046	0.851	0.107	0.664
Cholesterol	-0.030	0.902	0.365	0.124	-0.217	0.373
HDL	0.247	0.307	0.162	0.507	0.119	0.629
LDL	-0.191	0.435	0.304	0.206	-0.296	0.219
VLDL	0.088	0.721	-0.127	0.605	0.114	0.642

* Significant at P≤0.05, ** Significant at P≤0.01

Table 8: Correlation between variables in Patients group II

Variable	TOS		TAS		OSI	
	R	*P*	R	*P*	R	*P*
TOS	-	-	-0.428	0.067	0.870**	0.0001
TAS	-0.428	0.067	-	-	-0.799**	0.0001
OSI	0.870**	0.0001	-0.799**	0.0001	-	-
Age	-0.471*	0.042	0.222	0.361	-0.399	0.090
BMI	0.131	0.593	-0.106	0.666	0.076	0.758
Duration	-0.011	0.965	-0.051	0.835	0.023	0.927
Glucose	0.072	0.770	-0.266	0.271	0.225	0.354
HbA1c	0.103	0.676	0.003	0.990	0.020	0.936
Tri	0.357	0.134	0.131	0.593	0.147	0.549
Cholesterol	0.117	0..635	-0.535*	0.018	0.345	0147
HDL	0.308	0.200	-0.216	0.375	0.322	0.179
LDL	0.115	0.638	-0.436	0.062	0.271	0.263
VLDL	0.034	0.890	0.035	0.887	-0.006	0.982

* Significant at P≤0.05, ** Significant at P≤0.01

Table 9: Correlation between variables in Patients group III

Variable	TOS		TAS		OSI	
	R	*P*	R	*P*	R	*P*
TOS	-	-	-0.016	0.931	0.812**	0.0001
TAS	-0.016	0.931	-	-	-0.558**	0.001
OSI	0.812**	0.0001	-0.558**	0.001	-	-
Age	-0.022	0.907	-0.374*	0.042	0.165	0.384
BMI	-0.227	0.229	0.227	0.227	-0.233	0.215
Duration	-0.012	0.949	-0.255	0.174	0.126	0.506
Glucose	0.205	0.278	-0.212	0.262	0.272	0.147
HbA1c	0.270	0.149	0.154	0.416	0.082	0.667
Tri	0.048	0.802	-0.176	0.353	0.214	0.257
Cholesterol	0.248	0.186	0.114	0.550	0.222	0.239
HDL	0.183	0.332	0.391*	0.033	-0.032	0.868
LDL	-0.178	0.348	-0.207	0.272	-0.017	0.930
VLDL	0.110	0.564	-0.327	0.077	0.361*	0.050

* Significant at P≤0.05, ** Significant at P≤0.01

The correlation among all the studied groups and parameters in serum was done as illustrated in tables 7, 8 and 9. The results indicated a significant positive correlation between TOS and OSI in patients group I. In patients group II the results showed a significant negative correlation between TAS with (OSI, Cholesterol) as well as a significant negative correlation between TOS and age, while the results showed a significant positive correlation between TOS and OSI. Whereas in Patients group III a significant positive correlation between OSI and (TOS, VLDL) and between TAS and HDL as well as a significant negative correlation between TAS and (OSI, age).

Discussion

Diabetes mellitus (DM) is a disease that associated with abnormalities of carbohydrate metabolism, and can be identified by evaluation of blood glucose.[35] In the present study there was a marked rise in HbA1c and FPG levels in T2DM patients compared with the control population, indicating excessive glycosylation of haemoglobin and poor control of diabetes as reported by other studies [36]. Our results are in agreement with Dharambir et al [37] and Nife Oudah [38].

The association between the elevated level of HbA1c and the development of DM may be due to that is the previous exposure to high level of glucose. The high level leads to continuance of the harmful effects on the target cells long after reaching the glycemic control, or glucose normalization [39,40]. Glucose level showed significant increase in group of patient with duration of disease increase. A relation between FBS and duration of diabetes in this study is in agreement with the results obtained by a previous study which reported that the incidence of severe hyperglycemia increased with age and duration [41].
Multiple factors to be related with dyslipidemia among diabetic patients. Interestingly, age was significantly associated with dyslipidemia in diabetic

patients, which could be assigned to pressure of work and a lack of physical activity [42].

A decrease in HDL-C levels and an increase in the serum TG levels were observed in patients with diabetes, indicating the presence of dyslipidaemia. the uptake of free fatty acids (FFAs) by the skeletal muscle and adipose tissue is mediated by insulin, insulin resistance increase would result in increased FFAs delivered to the liver. This leads to the increased very low-density lipoprotein (VLDL) overproduction and concentrations, clinically manifesting as hypertriglyceridemia. An accumulation of the TG-rich lipoproteins in plasma can also result due to decreased lipoprotein lipase (LpL) activity [43]. Various studies were reported that hypertriglyceridemia and reduced plasma HDL-C is commonly present in T2DM [44,45,46].

Chronic hyperglycaemia, dyslipidaemia and elevated FFAs results in oxidative stress by stimulating ROS and RNS production, which attacks the lipids found in the plasma membranes, mitochondrial membranes and endoplasmic reticulum membranes and causes peroxidation [47]. Once lipid peroxidation is initiated, propagation chain reactions will occur until termination products are produced, such as lipid hydroperoxide, which further decompose to aldehydes such as MDA [48,49,50] in T2DM patients several factors, such as glycation, increased TG content and the reduced anti-oxidative properties of HDL are possible stimulators of LDL oxidation.[51] TG increased and decreased HDL-C can result in further oxidation of LDL in diabetes.27 Similar observations have been reported by other studies [52,53,54]. A significant decrease in TAO levels among T2DM patients was observed in this study and has also been reported by various other authors in their studies [23,54,55]. This reduction in TAO levels could be attributed to increased oxidative stress. This is evidenced by increased lipid peroxidation as well as by the excess utilisation of antioxidants against oxidative stress to minimise the damage.

The results of present study are in accordance with different studies carried only on serum such as a study by Sahaet al. [56] showed that a significantly higher plasma TOS level and significantly lower plasma TAS was observed in diabetic patients compared to controls. Rani and Mythili [57] showed that there was a significant decrease in the TAS among type 2 diabetes patients and significant increase in their malondialdehyde levels (which was used as an indicator of increased oxidative stress) in comparison to healthy controls. It also agrees with a study done by Joseph et al. [58] who reported that serum TAS was significantly lower in black South Africantype 2 diabetes mellitus patients compared to a matched control group. Ganjifrockwala et al. [59] also showed that there was asignificant increase in oxidative stress and significant decreasein total antioxidant levels in South African type 2 diabetesmellitus patients compared with controls. The serum resultswere also in accordance with the results obtained by Beyazyildizet al. [60] study on Turkish type 2 DM patients, which showed a significant increase in serum TOS levels but a significant decrease in serum TAS levels in type 2 patients compared to the control sample. Moreover, a significant decreased serum TAS levels was found on Turkish type 2 diabetic patients with PDR compared to control using the same colorimetric method as described by Erel[61,62]. Hyperglycaemia can induce abnormal metabolism which results in overproduction of free radicals and then leads to oxidative stress,[63] Oxidative

stress is an imbalance between free radicals formation and their scavenging (antioxidant system) that has been demonstrated as an increased production of pro-oxidative free radicals and/or diminished antioxidant defences in the body[64]. Blood contains many antioxidants that act to neutralize ROS, or prevent the release of ions responsible for initiating lipid peroxidation [65]. Merhan et al. [66] reported that in case of inflammation and hyperglycemia, antioxidant defense gets weakens and the increased reactive oxygen species leads to oxidative stress. The results of present study are confirmed by the calculation of OSI which is an indicator of the degree of oxidative

Conclusion

The differences in the oxidative stress status in serum samples of T2D Iraqi patients support the possibility of that oxidative stress plays a vital role in the development and pathogenesis of T2D. The results of this study suggest that hyperglycaemia and dyslipidaemia seen in the T2DM patients could lead to an increase in lipid peroxidation and oxidative stress and thus a decrease in TAO levels. This decrease in TAO levels could be due to its increased utilisation in order to scavenge the free radicals produced in high amounts due to increased oxidative stress. Early intervention and a diet rich in antioxidants can reduce the risk of developing complications and increase the longevity and quality of life of patients with diabetes

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