Study of Insulin Levels, HOME –IR, Body Mass Index, Iipids, and Oxidative Stress Factors in Iraqi Type 2 Diabetic Patients

Heba Mohammed Abed [1], Huda Farhan[2] and Ali Abd Al-Hussein[3]
(1) Medical Laboratory Sciences Technician, Diyala Health Department, Diyala, Iraq.
Correspondance author E-Mail : heba17700@gmail.com
(2, 3) College of Health and Medical Technology- Baghdad Middle Technical University, Baghdad, Iraq.
Corresponding author: E-mail(2) Hudafrhan@mut.edu.iq , E-mail(3): Shehdshosho@yahoo.com

Abstract

Increased oxidative stress is a well-established negative factor leading to insulin resistance, β-cell dysfunction, dyslipidemia, and finally leading to type 2 diabetes mellitus and its complications The study aimed to investigate some oxidative biomarkers, lipid profiles, and their association with hyperglycemia in patients with type 2 diabetes and pre-diabetics and compared the result with healthy control group. The present study conducted a case control study on 180 participants (87 females and 93 males) between the ages of 30 and 70 years, from the first of July 2023 until the end of September 2023. The participants were divided into three groups that included 60 patients with type 2 diabetes, 60 patients with pre-diabetes, and 60 healthy cases as a control group. The objective of this research was to measure the levels of, serum fasting blood sugar, glycated hemoglobin, oxidative biomarkers, insulin, HOMAIR, serum lipid profiles were evaluated, and estimate the impact of age and BMI on the results. The results showed that the level of lipid peroxidase (LPO), fasting blood sugar, glycated hemoglobin, insulin, HOMAIR and lipid profile in patients with type 2 diabetes and pre-diabetics groups are significantly increase than those in the control groups, while SOD (superoxide dismutase), GPX (glutathione peroxidase) and HDL, significantly lower in diabetic and pre-diabetics than those in the control groups. In conclusion, T2DM and pre-diabetics are conditions with increased oxidative stress, and the condition necessitates the consumption of antioxidants to combat oxidants, resulting in decreased total antioxidant status and increased lipid peroxidase.

Keywords: *Type 2 diabetes mellitus, Oxidative biomarkers, pre- diabetic, obesity and BMI.*

دراسة مستويات الأنسولين، وتقييم نموذج التوازن لمقاومة الأنسولين، ومؤشر كتلة الجسم، والدهون، وعوامل الإجهاد التأكسدي لدى مرضى السكري من النوع الثاني، العراقيين

هبة محمد عبد[1] ، أ.م.د. هدى فرحان احمد[2] وَ أ.د. علي عبد الحسين مهدي[3]

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الخلاصة

زيادة الإجهاد التأكسدي هو عامل سلبي يؤدي إلى مقاومة الأنسولين، وخلل في خلايا بيتا، وخلل شحوم الدم، ويؤدي في النهاية إلى داء السكري من النوع الثاني ومضاعفاته. هدفت الدراسة إلى دراسة بعض المؤشرات الحيوية المؤكسدة ومستويات الدهون وارتباطها بارتفاع السكر في الدم و مرض السكري من النوع الثاني ومجموعات ما قبل الاصابة بالسكري ومقانة النتائج مع مجموعة التحكم. أجرينا دراسة الحالات والشواهد على 180 مشاركًا (87 أنثى و93 ذكرًا) تتراوح أعمارهم بين 30 و70 عامًا، من أول يوليو 2023 حتى نهاية سبتمبر 2023. وتم تقسيم المشاركين إلى ثلاث مجموعات ضمت 60 مريضًا مصابًا بداء السكري من النوع الثاني، و60 مريضًا بمقدمات السكري، و60 حالة صحية كمجموعة ضابطة. كان الهدف من هذا البحث هو قياس مستويات السكر في الدم الصائم في الدم، والهيموجلوبين السكري، والمؤشرات الحيوية المؤكسدة، والأنسولين، وHOMAIR، وملامح الدهون في الدم، وتقدير تأثير العمر ومؤشر كتلة الجسم على النتائج. أظهرت النتائج أن مستوى بيروكسيداز الدهون (LPO)وسكر الدم الصائم والهيموجلوبين السكري والأنسولين و HOMAIR وملف الدهون في المرضى الذين يعانون من مرض السكري من النوع الثاني ومجموعات ما قبل الإصابة بالسكري يزيد بشكل ملحوظ عن أولئك في مجموعات التحكم، في حين أن SOD الفائق أكسيد ديسموتاز GPXالجلوتاثيون بيروكسيديز وHDL، كان أقل بشكل ملحوظ في مرضى السكري ومرضى ما قبل السكري من أولئك الموجودين في مجموعات التحكم. في الختام، T2DM ومقدمات السكري هي حالات تعاني من زيادة الإجهاد التأكسدي، وتستلزم الحالة استهلاك مضادات الأكسدة لمكافحة المؤكسدات، مما يؤدي إلى انخفاض الحالة الإجمالية لمضادات الأكسدة وزيادة بيروكسيداز الدهون.

الكلمات المفتاحية : *داء السكري من النوع الثاني، المؤشرات الحيوية المؤكسدة، مرحلة ماقبل السكري، السمنه ومؤشر كتلة الجسم.*

Introduction

Type 2 diabetes mellitus (T2DM) is a heterogenous, chronic metabolic disorder characterized by hyperglycemia, elevated insulin secretion, insulin resistance, β-cell dysfunction and its complications. T2DM prevalence is increasing worldwide, and this elevated is mainly due to the obesity epidemic [1]. Pre-diabetes is a state of raised glucose in plasma, in which has not yet reached the diabetics threshold and can predispose to the progress type 2 diabetes and cardiovascular diseases [2].

Insulin is polypeptide hormone, allows glucose to enter the liver, fat, and skeletal muscle cells from the blood and regulates carbohydrates, lipids, and protein metabolism. If there is insulin dysfunction resulting from impaired insulin secretion or function, hyperglycemia and result in diabetes complications and It poses a health threat to diabetics [3].

A body mass index (BMI) of 30 or more is considered obese. BMI is a height-based indicator of body weight [4]. Type 2 diabetes mellitus is more likely to occur in obese people [5]. In the obesity, that the cAMP-dependent enzyme hormone-sensitive lipase, is responsible for mobilizing free fatty acids (FFA) in circulation as a result of obesity-related adipose tissue growth. Furthermore,

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lipoprotein lipase releases free fatty acids (FFA) into the tissues through the lipolysis of TG-rich lipoproteins [6]. High levels of free fatty acids in the blood during obesity lead to insulin resistance, the primary characteristics of type 2 diabetes, which are made worse by the steadily increasing development of hyperglycemia [7].

T2DM is primarily characterized by hyperglycemia, which arises from insulin resistance and β-cell dysfunction. Insulin resistance (IR), the inability of tissues to fully respond to insulin, is the primary cause of hyperglycemia in type 2 diabetes (T2DM). The hormone is ineffective during the IR state, and eventually it causes hyperinsulinemia (an increase in the production of insulin) [8]. Prediabetes frequently has impaired beta-cell function and insulin resistance already [9].

Pancreatic β-cells, which are less abundant in antioxidant defense enzymes than other tissues, are especially susceptible to the deleterious effects of excessive ROS production. Hyperglycemia causes oxidative stress in these cells [10]. ROS have the ability to harm lipids, proteins, and DNA in pancreatic β-cells, which can result in β-cell dysfunction and death. Moreover, ROS have the ability to trigger stress-sensitive cellular pathways connected to reduced insulin secretion and insulin resistance [11].

Oxidative stress brought on by hyperglycemia has been connected to the emergence of insulin resistance, β-cell dysfunction, and the late complications of diabetes, via the following four primary molecular pathways: the hexosamine, protein kinase C-(PKC-) diacylglycerol (DAG), the polyol pathway, and the formation of advanced glycation end products (AGEs) [12].

The oxidative biochemistry of glucose, which is susceptible to autoxidation and the production of ROS, can be the source of oxidative stress, which can directly oxidize and damage proteins, lipids, DNA, and RNA. This can also activate a number of cellular stress-sensitive pathways in endothelial cells of both large and small vessels, causing cellular damage [13]. When there is an imbalance between ROS and antioxidants, oxidative stress occurs [11].

ROS are made up of both non-radical and free radical species. Molecules with one or more unpaired electrons are known as free radicals. Even though non-radical ROS molecules do not contain unpaired electrons, they can nonetheless be reactive and result in cellular damage [14]. The antioxidant system balances the production of ROS. The network of molecules and enzymes known as the antioxidant system shields cells from harm brought on by reactive oxygen species [15].

The most prevalent type of free radicals are superoxide radicals ($O_2^{\bullet-}$), which are quickly transformed into H_2O_2 by superoxide dismutase (SOD) and then removed by the enzymes catalase (CAT) and glutathione peroxidase (GPx).

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On the other hand, the Fenton reaction occurs when H2O2 produces •OH in the presence of metal cations Fe(II) [14]. One extremely reactive radical that can harm lipids, proteins, and DNA is the hydroxyl radical (•OH). Although cells have various defense mechanisms to preserve iron homeostasis and stop the production of •OH, they lack a specific defense mechanism against it [16].

One of the main factors thought to be responsible for the development of microvascular and macrovascular complications in type 2 diabetes is chronic hyperglycemia. It is well known that hyperglycemia responsible for damages proteins, lipids, and DNA, the degree of damage is correlated with the amount of reactive oxygen species (ROS) that hyperglycemia produces, which in turn causes oxidative stress [17].

Materials and Methods

Chemicals

An automated HumaReader HS (ELISA) is used to quantify insulin hormone, superoxide dismutase (SOD), glutathione peroxidase (GSH–Px) and lipid peroxidase (LPO), using sandwich enzyme – linked immunosorbent assay method. The Cobas c111 analyzer automatic biochemistry analyzer (Roche Diagnostics Ltd Company, Japan) was used to measure FBS, HbA1c and lipid profile. Insulin resistance was estimated by the homeostatic model assessment (HOMA IR) [HOMA IR = {Fasting glucose (mmol/l) x Fasting insulin (µIU/ml)/22.5}][18].

Study design

This case-control study was conducted during the period from the first of July 2023 until the end of September 2023, 180 Iraqi participants (87 females and 93 males) were chosen from those attending Baquba General Hospital, in Diyala-Iraq and Specialized Center for Endocrine Diseases and Diabetes, in Baghdad-Iraq. Blood was collected after 12 to 14h fasting (overnight). Written consent was obtained from each patient 180 subjects were examined in this study and included: (60) patients with type 2 diabetes mellitus (31 females, 29 males), (60) patients with pre-diabetic group (30males and 30 females) and 60 healthy persons for control group (26 females, 34 males), with ages (30-70 years). The diagnosis of T2DM was made using the WHO-recommended criteria. [19, 20].

Statistical analysis

SPSS version 26.0 (Chicago) was utilized for data analysis, and Microsoft Excel version 2016 was employed for statistical analyses. Student's t-test for difference between two independent means or Paired t-test for difference of paired observations, ROC, and post- hok tests was used to determine the significance between the difference of various means [quantitative data]. Simple frequency, percentage, mean, and standard deviation measurements will be shown for the data. Pearson's

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correlation analysis was used to determine the relationships between quantitative variables in this study. When the p-value was less than 0.05, the statistical significance was evaluated.

Results

Table 1, shows the patient's features. The mean (± SD) of age for diabetic group was (46.72 ± 11.76), the pre-diabetic group age mean (± SD) was (43.28 ± 10.82), and compared to the control (healthy group) age mean (± SD) was (39.69 ± 9.09) with P-value ≤0.05.

Table (1): The study markers were distributed according to the study groups.

Parameter	Studied group			P-value Between the groups
	Diabetic group G1 (n=60)	Pre-diabetic G2 (n=60)	Healthy G3 (n=60)	
Age (Years)	46.72±11.76[a]	43.28±10.82[a]	39.60±9.09	0.002
BMI (Kg/m^2)	29.25±3.93 [a]	29.16±4.07	22.51±2.190	≤0.001
FBS	193.16±38.00 [a]	118.91±5.56 [a,b]	104.65±11.61	≤0.001
HBA1C %	10.97±10.66 [a]	6.12±0.19 [b]	5.21±0.235	≤0.001
TG (mg/dl)	213.66±84.36 [a]	180.61±29.43 [a,b]	116.05±19.87	≤0.001
TC	219.63±60.77 [a]	222.38±45.43 [a]	161.90±13.36	≤0.001
HDL	45.91±13.38 a	55.40±13.79 a,b	77.13±15.31	≤0.001
LDL	115.46±35.26 [a]	97.46±26.80 [b]	86.63±11.21	≤0.001
Insuline hormone	9.44±4.71 [a]	5.27±2.85 [a,b]	1.84±0.29	≤0.001
HOMAIR	4.34±2.42 [a]	1.54±0.85 [a,b]	0.47±0.09	≤0.001
Glutathione peroxidase	5.88±1.32 [a]	10.99±5.61 [a,b]	19.84±5.65	≤0.001
Lipidperoxidase	6.04±1.56 [a]	3.21±1.28 [a,b]	1.61±0.54	≤0.001
Superoxide dismutase	1.24±0.51 [a]	2.87±0.83 [a,b]	5.20±1.14	≤0.001

a: statistically significant when compared to control. b: statistically significant when compared to diabetic group. SD: standard deviation. N: Number of patients.

The mean (± SD) of BMI was 29.25±3.93, 29.16±4.07, and 22.51±2.190 kg/m2 for diabetic G1, pre-diabetic G2, and healthy group G3, respectively, and the findings observed that there were significantly increase in the means of BMI (Kg/m2) in both DM groups (29.25±3.93) and Pre-diabetic groups (29.16±4.07) as compared to control (22.51±2.190) with P-value ≤0.05, as explained in Table 1.

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The findings observed that there were significantly increase in FBS (mg/dl) in both DM groups (M+SD 193.16±38.00) and Pre-diabetic groups (M+SD 118.91±5.56) as compared to control (M+SD 104.65±11.61) with P ≤0.001. In addition, there were highly significant differences between the levels of FBS (mg/dl) in T2DM groups and Pre-diabetic groups, also there were highly significant differences between the levels of FBS (mg/dl) in T2DM groups and healthy (control) groups, and highly significant differences between the levels of FBS (mg/dl) in pre-diabetic groups and healthy (control) groups, as explained in Table 2.

The results stated that there were highly significantly increase in the levels of HBA1C % in T2DM groups (M+SD 10.97±10.66) as compared to control (M+SD 5.21±0.235). In addition, there were highly significant differences between the levels of HBA1C% in T2DM groups (M+SD 10.97±10.66) as compared to Pre-diabetic group (M+SD 6.12±0.19) with P-value ≤0.001. While there were a non significant differences in the levels of HBA1C % in Pre-T2DM groups and control with P-value ≥0.05 as explained in Table 2.

Table (2): Comparative the mean levels of FBS (mg/dl) and HBA1C % between the studied groups. (n=180).

Parameter	Diabetic group G1	Pre-diabetic G2	Healthy G3	G1&G2		G1&G3		G2&G3	
	M+SD	**M+SD**	**M+SD**	**P-value**	**Sig.**	**P-value**	**Sig.**	**P-value**	**Sig.**
FBS (mg/dl)	193.16±38.00	118.91±5.56	104.65±11.61	0.0001	H.S	0.0001	H.S	0.001	H.S
HBA1C %	10.97±10.66	6.12±0.19	5.21±0.235	0.0001	H.S	0.001	H.S	0.419	N.S

In present study, highly significantly increase of TG (mg/dl) in both DM groups (213.66±84.36) and Pre-diabetic groups (180.61±29.43) as compared to control (116.05±19.87) with P-value ≤0.001.In addition there were highly significant differences between the levels of TG (mg/dl) in T2DM groups and Pre-diabetic groups P-value ≤0.001as explained in Table 3.

According to the findings, there was a significant increase of Cholseterol (mg/dl) in both DM groups (219.63±60.77) and Pre-diabetic groups (222.38±45.43) as compared to control (161.90±13.36) with P-value ≤0.01. In addition, there were no significant differences between the levels of TC (mg/dl) in T2DM groups and Pre-diabetic groups P-value ≥0.05 as explained in as explained in Table 3.

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The HDL distribution results revealed significant differences in the levels of HDL (mg/dl) in both DM groups (45.91±13.38) and Pre-diabetic groups (55.40±13.79) as compared to control (77.13±15.31) with P-value ≤0.001. In addition, there were highly significant differences between the levels of HDL (mg/dl) in T2DM groups and Pre-diabetic groups as explained in Table 3.

Also, there were significantly differences in the levels of in LDL T2DM groups (115.46±35.26) as compared to control (86.63±11.21) with P-value ≤0.001. Also, there were highly significant differences between the levels of LDL in DM groups (115.46±35.26) and Pre-DM group (97.46±26.80) with P-value ≤0.0001 as explained in as explained in Table 3.

Table (3): Comparative the mean levels of lipid profile parameters between the studied groups (n=180).

Parameter	Diabetic group G1	Pre-diabetic G2	Healthy G3	G1&G2		G1&G3		G2&G3	
	M+SD	**M+SD**	**M+SD**	**P-value**	**Sig.**	**P-value**	**Sig.**	**P-value**	**Sig.**
TG (mg/dl)	213.66±84.36	180.61±29.43	116.05±19.87	0.001	H.S	0.0001	H.S	0.0001	H.S
TC (mg/dl)	219.63±60.77	222.38±45.43	161.90±13.36	0.735	N.S	0.0001	H.S	0.0001	H.S
HDL (mg/dl)	45.91±13.38	55.40±13.79	77.13±15.31	0.0001	H.S	0.0001	H.S	0.0001	H.S
LDL (mg/dl)	115.46±35.26	97.46±26.80	86.63±11.21	0.0001	H.S	0.0001	H.S	0.026	H.S

The results showed, that there were significantly increase of Insulin hormones in both T2DM group (9.44±4.71) and Pre-diabetic groups (5.27±2.85) as compared to control (1.84±0.29) with P-value ≤0.001.In addition there were highly significant differences between the levels of Insulin hormones in T2DM groups and Pre-diabetic groups, and between T2DM and control group as explained in Table 4.

There was significantly differences in the levels of HOMAIR in both T2DM groups (4.34±2.42) and Pre-diabetic groups (1.54±0.85) as compared to control (0.47±0.09) with P-value ≤0.001. In addition there were significant differences between the levels of HOMAIR in T2DM groups and Pre-diabetic groups and between T2DM and control group as explained in Table 4.

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Table (4): Comparative the mean levels of insulin hormones and HOMAIR between the studied groups (n=180).

Parameter	Diabetic group G1	Pre- diabetic G2	Healthy G3	G1&G2		G1&G3		G2&G3	
	M+SD	**M+SD**	**M+SD**	**P-value**	**Sig.**	**P-value**	**Sig.**	**P-value**	**Sig.**
Insuline	9.44±4.71	5.27±2.85	1.84±0.29	0.0001	H.S	0.0001	H.S	0.0001	H.S
HOMAIR	4.34±2.42	1.54±0.85	0.47±0.09	0.0001	H.S	0.0001	H.S	0.0001	H.S

In this study, there were highly significant differences in the levels of Glutathione peroxidase in both T2DM groups (M+SD 5.88±1.32) and Pre-diabetic groups (M+SD 10.99±5.61) as compared to control (M+SD 19.84±5.65). In addition, there were highly significant differences between the levels of Glutathione peroxidase in both T2DM groups and Pre-diabetic groups, and also between T2DM and control group, as explained in Table 5.

Additionally, there was a highly significant increase of Lipidperoxidase in both T2DM groups (6.04±1.56) and Pre-diabetic groups (3.21±1.28) as compared to control (1.61±0.54). In addition, there was a highly significant increase in the levels of Lipidperoxidase in both T2DM groups and Pre-diabetic groups, as explained in Table 5.

Furthermore, as indicated in Table 5, there were significant increase in the levels of superoxide dismutase in both T2DM groups (1.24±0.51), Pre-diabetic groups (2.87±0.83) as compared to control (5.20±1.14) with P-value ≤0.001. In addition, there were highly significant increase between the levels of superoxide dismutase in T2DM groups and Pre-diabetic groups.

Table (5): Comparative the mean levels of oxidative enzymes between the studied groups (n=180).

Parameter	Diabetic group G1	Pre-diabetic G2	Healthy G3	G1&G2		G1&G3		G2&G3	
	M+SD	**M+SD**	**M+SD**	**P-value**	**Sig.**	**P-value**	**Sig.**	**P-value**	**Sig.**
Glutathione peroxidase	5.88±1.32	10.99±5.61	19.84±5.65	0.0001	H.S	0.0001	H.S	0.0001	H.S
Lipidperoxidase	6.04±1.56	3.21±1.28	1.61±0.54	0.0001	H.S	0.0001	H.S	0.0001	H.S
superoxide dismutase	1.24±0.51	2.87±0.83	5.20±1.14	0.0001	H.S	0.0001	H.S	0.0001	H.S

Discussion

The incidence of type 2 diabetes (T2DM), a common endocrine and metabolic disease with a complex pathogenesis, is rising [21]. The stage in between normal glucose regulation and diabetes is known as prediabetes. Increased risk of type 2 diabetes, cardiovascular events, and mortality are

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linked to prediabetes [22]. According to the American Diabetes Association, the status of diabetes and prediabetes was well-defined using HbA1c cutoff levels of less than 5.7% for non-diabetics, greater than 5.7% but less than 6.5% for prediabetes, and greater than or equal to 6.5% for diabetes mellitus [24]. In this study, the levels of serum antioxidant enzymes were evaluated in a group of individuals with type 2 diabetes, pre-diabetes, and healthy individuals (control group), and their associations with lipid profile, BMI, insulin hormon, HOMAIR, and HbA1c were discovered.

The findings were in line with Yadav et al. findings. who found that glucose intolerance prevalence [pre-diabetes and T2DM] rise in patients 46 years of age and older [24]. There are several contributing factors to take into account with relation to ageing and glucose intolerance. Ageing is a significant factor in the aging-related alterations in beta-cell function and insulin sensitivity [25]. The beta cells have ability to proliferate and their sensibility to apoptosis are decreased as individuals get older [26].

The current study's findings stated significantly increase in BMI in patients with T2DM and pre-diabetic than in control group. As seen in study by Klein et al. who found that T2DM and prediabetes are significantly increased by obesity, which is linked to increased intramuscular and intrahepatic triglyceride levels as well as increased distribution of fat in the abdomen and between the abdominal wall, since it consequently, it was resulting in both β-cell dysfunction and insulin resistance [27]. Obese people tend to have higher levels of oxidative stress biomarkers, which are correlated with triglyceride, LDL oxidation, BMI, and body fat percentage. Furthermore It has been demonstrated that obesity increases lipid peroxidation and lowers GPx and SOD action [28].

In the case groups were found to have highly-significant increase regarding FBS, HbA1c, but there was highly significant differences in HBA1c, amonge T2DM group and prediabetic group, and this agree with study by Yahyavi et al. [2]. It has long been known that HbA1c is a glycemic control indicator [29]. Diabetes and its associated macrovascular and microvascular complications are more common to occur in pre-diabetic. In clinical settings, hemoglobin (HbA1c) is the gold standard for glycemic control in diabetes [30]. Regardless of the HbA1c level, HbA1c variability is positively correlated with the risk of micro- and macrovascular complications in patients with type 2 diabetes [31]. In the present investigation and in agreement with SAAD et al. that both pre-diabetics and diabetics showed higher serum insulin and increased HOMAIR than non-diabetic controls [32]. An increase in FBS level was associated with high insulin resistance in pre-diabetics. If unchecked, this hyperglycemia would continue to rise, leading to type 2 diabetes. Insulin production and HOMAIR were consequently higher than in normal individuals [33]. T2DM is the most prevalent variation causing by combination of insulin resistance and relation insulin deficiency, because of pancreatic beta cell dysfunction [34].

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The results showed that high values of TC, TG, and LDL, along with a low level of HDL, observed in T2DM and pre-diabetic groups compared with control, and the results agree with study by Nayak et al. that revealed statistically significant reductions in HDL and increases in TG in pre-diabetic subjects [35], and agree with Kamble et al. that found that elevated oxidative stress was a result of dyslipidemia brought on by insulin resistance and inadequate glycemic control [36]. Insulin resistance leads to the buildup of TG, or free fatty acids, in the heart, pancreas, liver, and muscles. Insulin resistance also raises the activity of the liver lipase, which breaks down TG in LDL and HDL particles to produce denser and smaller LDL particles [37].

Numerous alterations, including increased triglyceride and decreased HDL levels, which are risk factors for macrovascular dysfunction, can be brought on by insulin resistance. Furthermore, prediabetic states already exhibit microangiopathic alterations brought on by elevated oxidative stress and inflammation [38].

Oxidative stress plays a crucial role in the pathophysiology of type 2 diabetes and the emergence of diabetic complications [39] [40].People with diabetes have higher glucose availability, which raises cellular activity and metabolism and increases ROS production [41].

The current study found that the T2DM group had lower GPx and SOD activity than the control group. These results concur with those of previous studies [42, 43]. Reduced activity of these enzymes could be attributed to the antioxidant defense system being depleted after an excess of free radicals is produced [44]. Increased glucose auto-oxidation, activation of the polyol pathway, and the production of advanced glycation end products (AGEs) are the main ways that diabetes can cause an excess of reactive oxygen species (ROS). When the antioxidant defense system in diabetics isn't working as it should, ROS build up and eventually cause increased oxidative stress and subsequent cellular damage [45].Superoxide dismutase (SOD) and glutathione peroxidase (GPx) are the two main endogenous cytoprotective antioxidant enzymes found in humans [46].

When assessing the antioxidant enzymes, the lipid peroxidase (LPO) was significantly higher in T2DM than in the pre-DM and control groups. As found in study by Bandeira et al. who came to the conclusion that diabetics had a significantly higher LPO than did pre-DM and control groups. Indicate that oxidative stress plays a role in the pathophysiology of diabetes and support the role that insulin resistance and hyperglycemia play in this process. Focus on showing how the elevated LPO in DM2 patients is closely linked to their elevated glycemic levels [47].

Conclusion

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Pre-diabetics and Type 2 Diabetes are conditions characterized by elevated oxidative stress. These conditions require consumption of antioxidants to counteract oxidants, which leads to a decrease in overall antioxidant status and an increase in lipid peroxidase.

Recommendations

Oxidative stress has a negative effect on diabetics, so it is very important to monitor the effectiveness of antioxidants and lipid peroxidase, because a decrease or increase in their effectiveness will increase diabetes complications.

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Reference

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