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Evaluation of Serum Malondialdehyde, Glutathione and Lipid Profile Levels in Iraqi Females with Type 2 Diabetes Mellitus

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Abstract

This study is carried out on patients with type 2 diabetes mellitus to assess the lipid profile, malondialdehyde and glutathione. Our study is concerned with 51 (Iraqi Arab females) patients of type 2 diabetes mellitus compared with 31 control subjects unified in age, sex and ethnic background. Lipid profile is measured by using commercially available kits, while the serum MDA and glutathione levels are measured by means of sandwich ELISA test using commercially available kits. Serum MDA is significantly higher ($P<0.001$) while glutathione is significantly lower ($P<0.001$) in type 2 diabetic patients when compared to the control. The normal levels of MDA (3.82 ± 0.77n mol/ml) and GSH (2.23 ± 0.54 µg/ml) recorded for the non-diabetic females are significantly ($p<0.001$) increased and depleted in the diabetic patients who record (6.78 ± 1.21 n mol/ml) and (1.29 ± 0.23 µg/ml) for MDA, GSH respectively. Parameters of lipid profile were significantly increase ($P<0.001$), while HDL cholesterol significantly decrease ($P<0.001$) in patients compared to the control group. The receiver operator curve (ROC) analysis of the forthcoming variations reveale the descending order of serum MDA (0.999), GSH (0.984), HDL (0.817), LDL (0.796), T.C (0.974), TG (0.727) & serum VLDL (0.722) showing a significant variation. In conclusion, the study shows low levels of GSH and high levels of MDA in diabetic patients indicating to an increased oxidative stress which is considered as the main cause to type 2 diabetes mellitus especially obese ones.

Key words: Malondialdehyde, Glutathione, Lipid Profile, Diabetes Mellitus

Introduction:

In recent years, a great number of studies have investigated the possible role of reactive oxygen species (ROS) in the etiology and pathogenesis of several

diseases [1-3]. The effects of lipid peroxidation in biological systems have been described in the development of type 2 diabetes mellitus (T2DM) [4].

In patients with T2DM lipolysis increased and glucose uptake decreased thus rising triglyceride (TG) formation by adipose tissue [5], additionally, treatment of T2DM with insulin induces accretion of lipids in the liver and skeletal muscle without upsetting whole-body insulin sensitivity subsequent to near-normoglycemia for 67 hour [6].

Endogenous ROS in high quantities devastate the innate antioxidant protection system leading to oxidative stress [7]. In T2DM, the glucolipotoxity is complicated with endothelial dysfunction and liable to oxidative stress, high blood glucose level is linked with free radical-mediated lipid peroxidation [8].

Enzymes of antioxidant are endogenous compounds to facilitate work in amalgamation to keep cells from ROS injure. High levels of lipids and protein have been detected in the sera of T2DM patients and their existence correlates with the growth of complications [9]. Many studies have showed increasing oxidative stress with decreased enzymes of antioxidant and vitamins in diabetic patients [10, 11].

Resistance of cells to damage caused by oxidative stress is determined by the capability of an array of antioxidant protection systems, from these reduced glutathione (GSH) which are the most ubiquitous and abundantly available inside human cells [12]. GSH is a small peptide synthesized from three amino acids (glutamate, cysteine, and glycine) in two steps. GSH contain a thiol group which plays an important role in every tissue of mammals in an opulent amount to defend an oxidative stress and considered as a forceful biomarker for the redox imbalance inside the cells [13]. Diabetes is connected with low levels of glutathione concentrations inside the cells [14], but the main cause of GSH deficiency presently is mysterious. lipids *in vivo* is mostly oxidized by ROS such as super oxide and peroxide radicals, which in turn are synthesized by lipoxygenases as a response to cell damage, malondialdehyde (MDA) is generated as a reasonably stable end product from the oxidative degradation of poly-unsaturated fatty acids (PUFA) [15].

The study is designed to find out the relation between lipid peroxidation lipoprotein levels to severity and complication of diabetes mellitus. Degree of lipid peroxidation ismeasured in terms of MDA along with antioxidants.

Materials and Methods:-

The present study is carried out in the National Diabetes Center for Treatment and Research at Al-Mustansiriya University between April 2012-September 2012. A total of 51 female patients of T2DM (aged 35-65 year.), previously diagnosed to contain T2DM based on the criteria of the specialist team on the diagnosis and categorization of T2DM. Thirty one identical in age and sex (females) healthy persons served as controls attended for regular health check up at the center. No one of the healthy control is taking any medication or nutritional complement; they are elected after complete physical assessment and laboratory tests. Sample collection: venous blood samples are collected in gel plain tubes, the samples are permitted to clot for two hour at 20-25 °C, then the samples are centrifuged for 15 minutes at 2000 rpm. Sera samples are stored immediately at -20 °C until using.

Plasma glucose is determined by a glucose oxidase method [16]. Total cholesterol and LDL and HDL cholesterol are measured by enzymatic analytical chemistry (CHOD-PAP

method, Roche Diagnostics, Milan, Italy) [17,18] as plasma triglycerides (GPO-PAP colorimetric enzyme test, Roche) [19]. Serum Malondialdehyde (MDA) is measured by enzyme linked immunosorbentassay ELISA (KA1206, Taipei City, Taiwan). Glutathione (GSH) level is performed by using a quantitative sandwich ELISA immunoassay (CSB-E09495h, Wuhan, Hubei, China).

Statistical Analysis :-

All the data have been presented as mean ± SD. One-way analysis of variance (ANOVA) is performed on each variable and the Bonferroni statistics is employed to compare the mean values from the different groups. Differences are considered significant at $P<0.05$. All statistical analyses are performed by using SPSS statistical software (version 19). Receiver operator curve (ROC) analysis is also employed**.**

Results:-

There is no significant statistical difference in mean age between cases and controls, Table 1, although the mean BMI is slightly higher among cases (32.7 ± 5.3 kg/m^2) compared to controls(30.8 ± 4.5 kg/m^2), but the difference is not statistically significant. The mean waist-hip ratio shows no statistical significant in case-control difference. The same table shows mean serum total cholesterol and triglycerides as significantly higher among cases with T2DM (204,6 ± 58.4 and 175.9 ± 85.8mg/dl) respectively compared to healthy controls (157.3 ± 25.3 and 109.4 ± 18.1 mg/dl) respectively. The effect of T2DM on these two serum lipid parameters is evaluated as a strong effect (Cohen's d > 0.8). The mean serum HDL is significantly low in cases with T2DM (41.8 ± 7.2mg/dl) compared to healthy controls (49.7 ± 4.9 mg/dl). The effect of T2DM on serum HDL is evaluated also as a strong effect (Cohen's d > 0.8).

The mean serum LDL and VLDL is significantly higher among cases with T2DM (126.7 ±52.9 and 35.1 ± 17.1 mg/dl respectively) compared to healthy controls (85.1 ± 22.7 and 22.1 ± 4.2 mg/dl respectively). The effect of T2DM on these two serum lipid parameters are evaluated as a strong effect (Cohen's d > 0.8).

Where as in type 2 diabetic patients serum MDA level is significantly higher and GSH level is found to be decreased, the values are statistically significant, MDA (3.82 ± 0.77n mol/ml) and GSH (2.23 ± 0.54 µg/ml) recorded for the non-diabetic females are significantly. (6.78 ± 1.21 n mol/ml) and (1.29 ± 0.23 µg/ml) ($P <0.001$).

Table 2 shows the receiver operator curve (ROC) analysis of the forthcoming variations reveals the descending order of FPG (0.999), MDA (0.999), GSH(0.984), HDL (0.817), LDL (0.796), total. C (0.794), TG (0.727) & VLDL (0.722) that show a significant variation. Table 3 shows the validity parameters for the selected indices when used as a test to predict the new cut-off values for the diagnosis of T2DM differentiating it from healthy controls.

Table (1): Showing the Status of Age, BMI, W-Hr, lipid profile, FPS , MDA and GSH in T2DM and Healthy controls

Parameters	Healthy controls (N=31)	Cases (T2DM) (N=51)	P (t-test)	Cohen's d (effect size)	SE controls	SE cases
Age (years)	(49 ± 8.9)	(51 ± 6.9)	($P = 0.26_{[NS]}$)		1.6	0.97
BMI (Kg/m^2)	(30.8 ± 4.5)	(32.7 ± 5.3)	($P = 0.11_{[NS]}$)	0.38	0.81	0.74
Waist -hip ratio	(0.91±0.04)	(0.91±0.05)	($P = 0.99_{[NS]}$)	0	0.007	0.007
Total cholesterol (mg/dl)	(157.3±25.3)	(204.6±58.4)	($P<0.001_{[HS]}$)	0.97	4.55	8.18
TG (mg/dl)	(109.4±18.1)	(175.9±85.8)	($P<0.001_{[HS]}$)	0.97	3.25	12.01
HDL cholesterol (mg/dl)	(49.7±4.9)	(41.8±7.2)	($P<0.001_{[HS]}$)	-1.23	0.87	1.01
LDL cholesterol (mg/dl)	(85.1±22.7)	(126.7±52.9)	($P<0.001_{[HS]}$)	0.95	4.08	7.41
VLDL cholesterol (mg/dl)	(22.1±4.2)	(35.1±17.1)	($P<0.001_{[HS]}$)	0.95	0.75	2.39
Fasting plasma glucose (mg/dl)	(96.3±8.6)	(196.3±56.1)	($P<0.001_{[HS]}$)	2.24	1.55	7.85
Serum MDA (nmol/ml)	(3.82±0.77)	(6.78±1.21)	($P<0.001_{[HS]}$)	2.77	0.141	0.17
Serum GSH (µg/ml)	(2.23±0.54)	(1.29±0.23)	($P<0.001_{[HS]}$)	-2.47	0.096	0.032

Data presented as mean + SD. **HS**-High is Significantly different from control group by one-way ANOVA, **NS**- Non Significantly different from control group, **BMI**-body mass index **FPG**-Fasting Plasma glucose, **TG**-Triglycerides, **HDL**- High density lipoprotein, **LDL**- Low density lipoprotein, **VLDL**- Very Low lipoprotein, **MDA**- Malondialdehyde, **GSH** – Glutathione

Table (2): ROC area for selected parameters when used as test to predict a diagnosis of DM differentiating it from healthy controls

Parameters		ROC area	P-values
Serum MDA	(n mol/ml)	0.999	<0.001
Fasting plasma glucose	(mg/dl)	0.999	<0.001
Serum GSH	(µg/ml)	0.984	<0.001
Serum HDL	(mg/dl)	0.817	<0.001
Serum LDL	(mg/dl)	0.796	<0.001
Serum total cholesterol	(mg/dl)	0.794	<0.001
Serum Triglycerides	(mg/dl)	0.727	<0.001
Serum VLDL	(mg/dl)	0.722	<0.001

Table (3): Validity parameters for selected indices when used as test to predict a diagnosis of DM differentiating it from healthy controls

				PPV at pretest probability =		NPV at pretest probability =10%
Positive if ≥ cut-off value	**Sensitivity**	**Specificity**	**Accuracy**	**50%**	**90%**	
Serum total cholesterol (mg/dl)						
102.0 (Highest sensitivity)	98.0	9.7	64.6	52.0	90.7	97.8
182.0 (Optimum cut-off)	64.7	93.5	75.6	90.9	98.9	96.0
195.5 (Highest specificity)	54.9	100.0	72.0	100.0	100.0	95.2
Serum Triglycerides (mg/dl)						
92.5 (Highest sensitivity)	72.5	32.3	57.3	51.7	90.6	91.4
142.0 (Highest specificity) (Optimum cut-off)	52.9	100.0	70.7	100.0	100.0	95.0
Serum HDL (mg/dl)						
39.5 (Highest specificity)	41.2	100.0	63.4	100.0	100.0	93.9
47.5 (Optimum cut-off)	78.4	67.7	74.4	70.9	95.6	96.6
56.5 (Highest sensitivity)	96.1	9.7	63.4	51.5	90.5	95.7
Serum LDL (mg/dl)						
32.5 (Highest sensitivity)	100.0	3.2	63.4	50.8	90.3	100.0
101.5 (Optimum cut-off)	68.6	87.1	75.6	84.2	98.0	96.2
122.5 (Highest specificity)	51.0	100.0	69.5	100.0	100.0	94.8
33.5 (Highest specificity)	43.1	100.0	64.6	100.0	100.0	94.1
Serum VLDL (mg/dl)						
19.5 (Highest sensitivity)	72.5	41.9	61.0	55.5	91.8	93.2
27.5 (Optimum cut-off)	54.9	87.1	67.1	81.0	97.5	94.6
33.5 (Highest specificity)	43.1	100.0	64.6	100.0	100.0	94.1
Serum MDA (n mol/ml)						
4.88 (Highest sensitivity) (Optimum cut-off)	100.0	96.7	98.7	96.8	99.6	100.0
5.10 (Highest specificity)	95.9	100.0	97.5	100.0	100.0	99.5
Serum GSH (µg/ml)						
1.45 (Highest specificity)	82.4	100.0	89.0	100.0	100.0	98.1
1.57 (Optimum cut-off)	96.1	96.7	96.3	96.6	99.6	99.6
1.93 (Highest sensitivity)	100.0	63.3	86.1	73.2	96.1	100.0
Fasting plasma glucose (mg/dl)						
109.5 (Highest sensitivity)	100.0	93.5	97.6	93.9	99.3	100.0
112.5 (Highest specificity) (Optimum cut-off)	98.0	100.0	98.8	100.0	100.0	99.8
5.10 (Highest specificity)	95.9	100.0	97.5	100.0	100.0	99.5

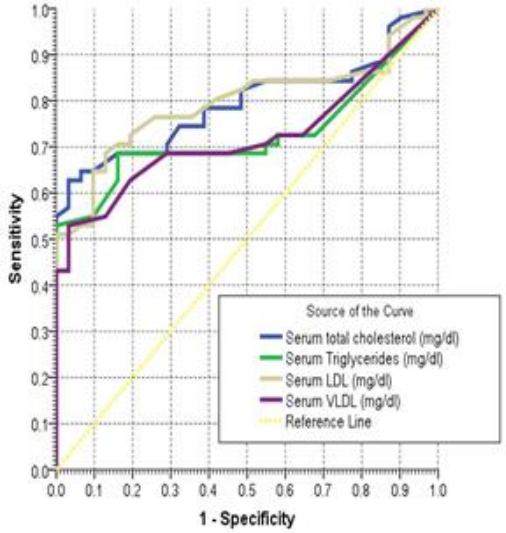

Fig. (1): ROC curve showing the trade-off between sensitivity (rate of true positive) and 1-specificity (rate of false positive) for selected parameters when used as test to predict a diagnosis of DM differentiating it from healthy controls. (lipid profile except (HDL-C)

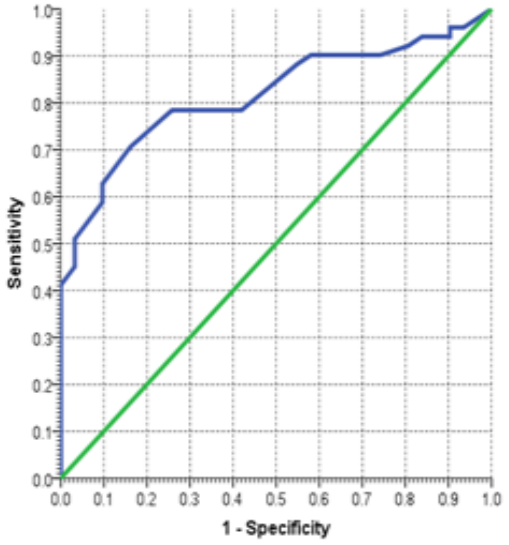

Fig. (2): ROC curve showing the trade-off between sensitivity (rate of true positive) and 1-specificity (rate of false positive) for (HDL-C) when used as test to predict a diagnosis of DM differentiating it from healthy controls

Discussion

Diabetes mellitus is a complex and multifactorial disease indulging severe insulin dysfunction in conjunction with gross abnormalities in glucose homeostasis, lipid and protein metabolism. The metabolic dysregulation associated with diabetes causes multiple organ systems that impose a heavy burden of morbidity and mortality from macrovascular and microvascular complications [20]**.** In diabetes, oxidative stress occur due to increased production of ROS such as superoxide radicals (O_2^-), hydroxide radicals ($OH^\bullet$), hydrogen peroxide (H_2O_2), which are found to be involved in the destruction of β cell of pancreas, thus decrease the insulin level and increase blood glucose levels[21]. Hyperglycaemia further generates more free radicals by non enzymatic glucose autoxidation and protein glycation [22]. Irregular metabolism of lipid is frequently presents in patients with T2DM [23]. Hypertriglyceridaemia usually accompanies decreased HDL-C, which is also a prominent feature of plasma lipid abnormalities seen in diabetic subjects [24]. The low level of HDL-C, which exerts anti-atherogenic and antioxidative effects when present in sufficient amounts, is a key feature of NIDDM (also known as type 2 diabetes mellitus). The reduced HDL-C levels are often accompanied by elevations in plasma TG levels[25] cholesterol ester transfer protein (CETP) is mediated by the procedure [26]. Insulin resistance (IR) underlies the changes to facilitate the occur hence in lipid parameters of T2DM, IR is coupled with elevated levels of TG and TC, and lesser concentrations of HDL-C [27]. The method dependable for hypertriglyceridaemia may be an amplified hepatic emission of VLDL and a deferred clearance of TG-rich lipoproteins, which might chiefly be due to augmented levels of substrates for TG manufacture, glucose, and free fatty acids. Glucose is a decreased activity of lipoprotein lipase (LPL), a key enzyme for lipoprotein-TG [28].

The oxidant/ antioxidant imbalance is characterized by an increase in lipid peroxidation, DNA oxidation damage products, and enzymes of liver disease. At the same time, adequate levels of the enzymatic and non enzymatic antioxidants, responsible for scavenging free radicals, are not efficient.

Oxidative stress also affects the levels of GSH, a non enzymatic antioxidant. GSH is involved in scavenging free radicals and plays an important role in maintaining the redox status of the cell. In oxidative stress condition GSH is oxidised to glutathione disulphide (GSSG) by the action of GPx, which is then again reduced to GSH by the enzyme glutathione reductase, with coupling reactions of NADPH to NADP [29]. MDA is a product formed as an end product of lipid peroxidation of cellular polyunsaturated fatty acids. Measurement of MDA helps to assess the degree of tissue damage [30]. Lipidperoxidation of cellular structures is thought to play an important role in development of late diabetic complications. Plasma MDA and erythrocyte membrane MDA are also found to be positively correlated with HbA1c which point toward connection of protein glycation in the formation of advanced glycation end products (AGEs) and generation of ROS [31]. ROS may directly oxidize erythrocyte glutathione and initiate membrane lipid peroxidation in type-2 diabetes mellitus.

Conclusion

There is a relationship connecting between peroxidation of lipid and glucose concentration, this relation plays a major role in increased lipid peroxidation in T2DM. In our study, MDA becomes greater than before and

GSH becomes lesser than before, which is the main cause for injury by T2DM.

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تقييم المستويات المصلية للمالونوثنائي الالديهايد و الكلوتاثيون وصورة الدهون الكاملة في النساء العراقيات المصابات بداء السكري من النوع الثاني

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الخلاصة

أجريت هذه الدراسة على مريضات بداء السكري النمط الثاني بهدف تقييم المستوى المصلي للمالونودايالديهايد (MAD) والكلوتاثايون (GSH) والمحتوى الدهني (total cholesterol, TG, HDL cholesterol, LDL cholesterol, VLDL cholesterol)، وكان التقييم بواسطة فحص الالايزا (ELISA) وعن طريق العدد المختبرية التجارية المتوفرة. أجريت الدراسة على 51 امرأة عراقية مصابة بداء السكري النمط الثاني ، ولغرض المقارنة اعتمدت 31 امرأة من الأصحاء (السيطرة) المتوافقين بالعمر والجنس والعرق(العراقيين العرب) مع المرضى.

اظهر المستوى المصلي للدم للمالونودايالديهايد زيادة معنوية وهي (6.78 ±1.21 n mol/ml) مقابل (3.82 ±0.77n mol/ml) في المرضى مقارنة بالسيطرة، بينما أنخفض معنوياً المستوى المصلي للكلوتاثايون (1.29 ±0.23 µg/ml) مقابل (2.23 ± 0.54 µg/ml) كما اظهر المستوى المصلي للدم للدهونات باستثناء HDL زيادة معنوية في المرضى بالمقارنة مع السيطرة.

للتمييز بين مرضى داء السكري النمط الثاني و مجموعة السيطرة استخدم تحليل Receiver Operator (ROC) Curve والذي أظهر الترتيب التنازلي بحسب الأهمية للمؤشرات التي أظهرت فروقاً معنوية وكان الترتيب لسكر بلازما الدم الصائم والمالونودايالديهايد (0.999) و GSH (0.984) و HDL (0.817) وLDL (0.796) وtotal cholesterol (0.794) و TG (0.727) و VLDL (0.722). نستنتج من ذلك ان انخفاض مستوى الكلوتاثايون وزيادة مستوى المالونودايالديهايد يتزامن مع زيادة خطورة داء السكري النمط الثاني خصوصا مع البدينين منهم.

الكلمات المفتاحية: المالونوثنائي الالديهايد، الكلوتاثايون، محتوى الدهون، البول السكري