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A Comparison Study of Some Biochemical Markers in Type 2 Diabetic Patients in Kerbala Between those Who are Non-Smokers and do Exercise and those who are Smokers and don't Exercise

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Abstract: Diabetes is a comprehensive term for a number of metabolic diseases that affect the body's ability to control blood glucose levels. Insulin is produced in the pancreas and helps to metabolize glucose for energy. Diabetes occurs when insulin production is weak. The current study was to examine the effect of smoking and exercise on biochemicals for type 2 diabetics Kerbala. Patients and Methods: Group A: Consists of 20 type 2 patients with diabetes, were smokers, and did not exercise and were treated by metformin. Group B consists of 20 patients of type 2 diabetes, non-smokers, exercise and metformin treatment. Control group: Normal healthy individuals for comparison, 20 healthy people participated in this study. Type 2 DM patients have controls characterized by age, fasting plasma glucose and fasting personal fasting Lipid profile: Comparison of the TG in different groups with control shows that all groups [control (TSC:4.08±0.615,TG: 1.05±0.314),group A(TSC: 5.01±1.479,TG: 3.51±1.961) and group B(TSC: 4.55±1.161,TG: 2.23±1.654)](unites in mmol/L) have a significant differences ,while for TSC there is a significant difference between all groups except with the group B. Fasting blood glucose: Comparison of the parameters in all groups[control(FBG:5.853±0.662) ,group A(FBG: 10.893±1.441) and group B(FBG: 9.042±1.071)](unites in mg/dL) shows that there is significant difference in the level of glucose. Exercise and exercise result in non smoking smoking results in controlling high blood sugar, dyslipidemia and effects of optimizing metformin in controlling blood sugar in type 2 diabetic patients.

Keywords: Smoking, Diabetes, Glycemic, Hyperglycemia and lipids.

INTRODUCTION

Diabetes is a group of metabolic disorders characterized by chronic excessive sugar caused by defects in the secretion of insulin, or the work of insulin or both. [1] It describes the metabolic disorder of multiple pathogens characterized by disturbances in metabolism of carbohydrates, fats and proteins. [2] In diabetes, carbohydrate, fat and protein disorders often play a role in diabetes complications. Hypercholesterolemia and hyperglycemia are often observed and are closely related to the degree of

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control of diabetes. It is reported that high-density lipoproteins in the serum are low in diabetic patients of both types.

Hyperglycemia may alter lipoproteins in a form that promotes atherosclerosis. LDL levels are often changed in diabetic patients. Fat peroxide products, which increase in patients with clinical and experimental diabetes, are important results of free stress derived from free oxygen. These products may be important in causing complications of blood vessels in diabetes. [3]

The liver plays a key role in regulating carbohydrate metabolism. It uses glucose as a fuel, has the ability to store glucose as glycogen and also synthesizes glucose from non-carbohydrate sources. This major function of the liver makes it susceptible to diseases in people with metabolic disorders, especially diabetes. [4]

Humans with DM may be at increased risk for AKF. AKF may occur in DM due to kidney, renal or post rennal causes such as diabetic ketoaccidosis, non-ketone hyperactivity, hypootension, sepsis, or renal factors.

These conditions carries a high rate of morbbidity and morttality, and management may be more complicated due to the presence of diiabetes and other pathogens. [5]

The aim is to compare the biochemical changes in non-smokers with diabetic patients taking metformin and non-diabetic patients who take metformin in the city of Kerbala and review information on DM 2 with a focus on its etiology and metabolic changes on parameters.

SUBJECT AND METHODES

Patients and Methods: This case control study was carried out at Alhussainy hospital in Kerbala city during the period of [July -September 2017]. The study carried out on [40] Iraqi male DM type 2 . These patients aged from 40 – 60 years**:**

Group [A]: consists of 20 Type 2 Diabetic patients, they were smokers, non-exercise and treated by metformin.

Group [B]: Consists of 20 patients of type 2 diabetes, were non-smoking, exercise and treatment by metformin. Diagnosis was performed by consultant physicians for patients who had T2DM depending on the patient's history, laboratory investigations, clinical examination, biomarkers, ECG and radiographic detection. From each subject, 3-5 ml of blood was obtained through venous thrombosis. Of fasting patients using a 5ml syringe, is used to estimate the glucose level, serum urea serum grease, creatinine and other aliquot were distributed in the test tube and left for an hour to coagulate at room temperatture.

Then, centrifuge at 3900 rpm for 10 minutes to collect the serum. All samples were obtained and the tubes were distinguished from each other by a specific bar code.

Statistical analysis: Statistical analyzes were performed using SPSS 19.0 for windows. Data was shown as mean ± SE using ANOVA mean and one value. The values of $P < 0.05$ were considered important.

RESULTS

1. Age and BMI:

All cases were male, aged between 40 and 60 years, and there were no statistically significant differences when comparing groups.

Table (3.1): age and BMI between groups (values are mean +S.D)

Parameters	Control group(n=20)	GroupA(n=20)	GroupB1(n=20)	P value
Age(years)	**52.44+0.53**	**54.83+0.79**	**50.02+0.2**	**NS**

2. Lipid profile:

Table 4 shows levels of lipids [TSC and TG] in the three groups of [normal] and non-diabetic / diabetic patients taking metformin [group A] and non-smokers / diabetic patients taking metformin [group 2].

Comparison of TG in different groups with control shows that all groups [control, A and B] have significant differences, whereas TSC there is a significant difference between all groups except with group B.

Table (3.2): comparison serum lipid profile in cigarette smokers/non-exercised/taking metformin group and control group.

Parameters	**[Control [n=20]**	**Cigarette smokers\non-exercised**	**P value**
		Group-A[n=20]	
TSC [mmol/L]	4.08±0.615	5.01±1.479	**[S]**
Triglycerides [mmol/L]	1.05±0.314	3.51±1.961	**[S]**

Data are expressed as Mean±SD. All values are in comparison with control group.

Table [3.2] shows lipid profile levels [TSC and TG] in control group and smokers / non-pathogenic group [A-group]. Comparing the parameters in Group A with the control shows that the TG and TSC levels were important.

Table (3.3): Comparison serum lipid profile in non-cigarette smokers/exercised/taking metformin group and control group.

Parameters	**[Control [n=20]**	**Non-Cigarette smokers\exercised**	**P value**
		Group-B[n=20]	
TSC [mmol/L]	4.08±0.615	4.55±1.161	**[NS]**
Triglycerides [mmol/L]	1.05±0.314	2.23±1.654	**[S]**

Data are expressed as Mean±SD. All values are in comparison with control group.

Table 3.3 shows the levels of the fat profile [TSC and TG] in the control group and group B. The comparison of the parameters in group B with control shows that there is no significant difference in the TSC level while there is a significant difference in the TG level.

Table (3.4): Comparison serum lipid profile in non-cigarette smokers/exercised/taking metformin group and cigarette smokers/non-exercised/taking metformin group.

Parameters	**cigarette smokers/non-exercised**	**Non-Cigarette smokers\exercised**	**P value**
	Group-A [n=20]	**Group-B [n=20]**	
TSC [mmol/L]	5.01±1.479	4.55±1.161	**[S]**
Triglycerides [mmol/L]	3.51±1.961	2.23±1.654	**[S]**

Data are expressed as Mean±SD. All values are in comparison with control group.

Table [3.4] shows the fat profile levels [TSC and TG] in Group A and Group B. A comparison of parameters in group A with group B shows that there is a significant difference in TSC and TG levels.

3. fasting blood sugar level:

Table [3.5] shows serum fasting glucose levels [FBS] in the three groups of [natural] control and smokers / non-diabetic patients taking metformin [group A] and non-smokers / diabetic patients taking metformin [group 2].Comparison of parameters in different control groups shows that all groups [control, A and B] have significant differences in glucose levels.

Table (3.5): comparison serum glucose in control group, group-A and group-B.

Parameters	**[Control [n=20]**	**Cigarette smokers\non-exercised**	**Non-Cigarette smokers\ exercised**	**P value**
		Group-A[n=20]	**Group-B[n=20]**	
FBS [mg/dl]	5.853±0.662	10.893±1.441	9.042±1.071	**[S]**

Values are expressed as mean±SD. All values are in comparison with control group.

Table [3.6] shows the levels of glucose in all groups [control, A and B]. Comparison of the parameters in all groups shows that there is significant difference in the level of glucose.

DISCUSSION

1. Lipid profile:

Results of patients in group A showed that this group had a significant difference in TSC levels compared to control. This increase in the level of this parameter associated with the sedentary lifestyle and cigarette smoking associated with high TSC. Cigarette smoking is associated with blood lipid disorder, a major risk factor for cardiovascular disease, and the mechanisms caused by smoking cause dyslipidemia to increase the release of catecholamine, Free fatty acids, which may increase the concentration of TSC. [6] The results showed that smoking was strongly and positively associated with total cholesterol in the blood and triglycerides. In other words. Total cholesterol levels and triglycerides are increased by smoking. [7] The levels close to TSC in both groups because of patients in group B were, first, no history of smoking habit, so there is no effect on the lipid profile, as mentioned above that smoking increases the level of fat. Second, the exercise has a role in maintenance level S. Ch closely to the normal range, and there is a study found that physical training significantly reduces TSC in the practice of type 2 diabetes. [8]. The mechaanisms behiind the effect of the exercize on the lipid profile are unknown, exercise appears to accelerate the ability of skeletal muscles to use lipids versus glycogen, thus reducing plasma fat levels. [9],[10]. Also the exercize itself may also elevate blood lipid consumption, hence lead to decrease lipid profile levels [11]. Mechanisms may involve the increased activity of lipo-protein lipase [LPL] - lipoprotein lipase responsible for TG hydrolysis in granules [12] . A study reported that heavy or prolonged exercise episodes could significantly increase plasma LPL activity, thus promoted LPL-mediated TG hydrolysis. [13]

In our study the mean value of TSC and TG in cigarette smokers is significantly higher [P<0.005] as compared to nonsmokers.

The comparison between group A and group B showed that there is significant difference between the two parameters [TSC and TG] in group B were significantly lower than in group A , due to the ability of exercise to reduce the levels of this parameters, and the absence of the effect of smoking as mentioned in a mechanisms illustrated above.

Patients in the two groups have taken metformin, which has a role in fat reduction [TSC and TG] and control the effect of lipid-disruptive disorder and has been approved in many studies. The treatment of metformin clearly indicates that after treatment with diabetic patients, he corrected dyslipidemia [13]. Some non-randomized studies have shown significant reductions in free fatty acids after treatment with metformin [14]. Newly diagnosed type 2 diabetics show essentially different lipid profile characteristics. Our study has agreed with previous studies; demonstrating that metformin as a single treatment reduced TSC and significantly reduced TG levels [15,16,17]. Based on this, we have concluded that the results in metformin diabetes therapy are used to improve lipid level in agreement with previous studies. Metformin can activate the upstream kinase called LKB1 primary. [18-20]

These are the basic steps in the formation of triglycerides also known as tglycerides [TG] which constitute the majority of VLDL produced by the liver. [21-23]. The second regulation of lipid generation is the phosphorylation of carboxyl acetyl and thus inhibit its activity. As a result, levels of chronic involvement in malonyl are reduced resulting in a decrease in the synthesis of fatty acids [need to produce TG] and promote oxidation of fatty acids. [24]

2. Fasting Blood Sugar:

In this segment, all groups were compared with fasting blood sugar [FBS]. From our results, there is a significant difference between all classes [A, B and control] in FBS value. The difference between control and group A, where the level of FBS is higher in group A compared with the control group is due to the habit of smoking and reduced physical activity and this leads to reduced ability of metformin to control blood sugar level. The effect of smoking showed increased insulin resistance [25]. Smoking is reduced by 10% to 40% in men who smoke compared with non-smokers [26, 27]. In subjects of type 2, insulin responses and oral glucose-C-peptide were significantly higher in nonsmokers and insulin resistance, and were positively associated with dose-dependent therapy [28]. Thus, insulin resistance caused by smoking in patients with type 2 DM.

CONCLUSION

From our findings, we concluded that patients with type 2 DM, which leads exercise, beneficial results in the control appear in high blood sugar [reduce insulin resistance, and dyslipidemia in the effects of improving metformin to control blood sugar In patients with type 2 diabetes compared with non-exercise type 2 diabetic patients. Also, our study clearly shows a strong relationship between high blood lipids and blood sugar with cigarette smoking, while physical activity is associated with increased insulin resistance. So quitting smoking is an important step in controlling DM and reducing its complications.

REFERENCES

[1] Riyadh Al-Zubaidi ,MohammedT.Abbas ,Salam K.Rubat Al-Sultany. Association Between Total antioxidant Capacity and Leptin Levels in Type-2 Diabetic Patients. Kerbala journal of pharmaceutical sciences. No. (12) 2017.

[2] World Health Organization. Definition, Diagnosis And Classification Of Diabetes Mellitus And Its Complications. Report Of Who. Department Of Non-Communicable Disease Surveillance. Geneva 1999.

[3] Academy Of Medicine. Clinical Practice Guidelines. Management Of Type 2 Diabetes Mellitus. Moh/P/Pak/87.04[Gu], 2004.

[4] Nhs. Diabetes Mellitus - Insulin Initiation - University Hospitals Of Leicester Nhs Trust Working In Partnership With Pcts Across Leicestershire And Rutland, May.

[5] Vakrani, G. P., Et Al. [2013]. "Acute Renal Failure In Diabetes Mellitus [Prospective Study]." J Nephrol Ther .

[6] Dipiro, Joseph T.; Talbert, Robert L.; Yee, Gary C.; Matzke, Gary R.; Wells, Barbara G.; Posey, L. Michael. Pharmacotherapy: A Pathophysiologic Approach. New York: Mcgraw-Hill; 2005.

[7] Parchwani Dn, Upadhyah Aa, Dussa Hc. Effect Of Active Smoking On Glucose Tolerance And Lipid Profile. 2013.

[8] Yadav B, Bade A, Singh J, Jha B. Comparative Study Of Lipid Profile In Smokers, Tobacco Chewers And Diabetic Patients. Journal Of Institute Of Medicine. 2007;27[3].

[9] Rönnemaa, T., K. Mattila, A. Lehtonen, And V. Kallio. A Controlled Randomized Study On The Effect Of Long-Term Physical Exercise On The Metabolic Control In Type 2 Diabetic Patients. Acta Med. Scand. 220:219–224, 1986.

[10] Diabetes Mellitus Prevention Program Research Group. Reduction In The Incidence Of Type 2 Diabetes Mellitus With Lifestyle Intervention Of Metformin. N Engl J Med 2002;346:393-403.

[11] Uk Prospective Diabetes Mellitus Study Group. Effect Of Intensive Blood Glucose Control With Metformin On Complications In Overweight Patients With Type 2 Diabetes Mellitus [Ukpds 34]. Lancet 1998;352:854-865.

[12] Gargiulo P, Caccese D, Pignatelli P, Brufani C, De Vitof,Marinor,Etal.Metformindecreasesplatelet Superoxide Anion Production In Diabetic Patients. Diabetes Mellitus Metab Res Rev 2002;18:156159.

[13] Beisswenger P, Ruggiero-Lopez, D. Metformin Inhibition Of Glycation Processes. Diabetes Mellitus Metab 2003;29:6s95-6s103.

[14] Earnest Cp, Artero Eg, Sui X, Et Al. Maximal Estimated Cardiorespiratoryfitness, Cardiometabolic Risk Factors, And Metabolic Syndrome In The Aerobicscenter Longitudinal Study. Mayo Clin Proc. 2013;88[3]:259–70

[15] Calabresi L, Franceschini G. Lecithin: Cholesterol Acyltransferase, High-Densitylipoproteins, And Atheroprotection In Humans. Trends Cardiovasc Med. 2010;20[2]:50–3

[16] Kobayashi J, Nohara A, Kawashiri M, Et Al. Serum Lipoprotein Lipase Mass:Clinical Significance Of Its Measurement. Clin Chim Acta. 2007;378:7–12.

[17] Gill H.C. The Cardiovascular Pathology Of Smoking. Am Heart J. 1988; 115[1 Pt 2]: 250– 7.

[18] Wulffele Mg, Kooy A, De Zeeuw D, Stehouwer Cd, Gansevoort Rt. The Effect Of Metformin On Blood Pressure, Plasma Cholesterol And Triglycerides In Type 2 Diabetes Mellitus: A Systematic Review. J Int Med 2004;256:1-14.

[19] Manzella D, Grella R, Esposito K, Giugliano D, Barbagallo M, Paliosso G. Blood Pressure And Cardiac Autonomic Nervous System In Obese Type 2 Diabetic Patients: Effect Of Metformin Adiabetes Mellitusinistration. Am J Hypert 2004;17:223-227.

[20] Salpeter Sr, Greyber E, Pasternak Ga And Salpeter Ee. Risk Of Fatal And Nonfatal Lactic Acidosis With Metformin Use In Type 2 Diabetes Mellitus: Systematic Review And Meta-Analysis. Arch Intern Med. 2003;163:2594.

[21] Kim Hj. Effects Of Rosiglitazone And Metformin On Inflammatory Markers And Adipokines: Decrease In Interleukin-18 Is An Independent Factor For The Improvement Of Homeostasis Model Assessment- Beta In Type 2 Diabetes Mellitus,Clin. Endocrinol. [Oxf.] 2007;66[2]:282-9.

[22] Riccio A, Del Prato S, De Kreutzenberg Sv And Tiengo A. Glucose And Lipid Metabolism In Non-Insulin Dependent Diabetes Mellitus: Effect Of Metformin. Diabetes Mellitus Metab. 1991;17:180–184.

[23] Shaw Rj, Lamia Ka, Vasquez D Et Al. The Kinase Lkb1 Mediates Glucose Homeostasis In Liver And Therapeutics Effects Of Metformin. Science 2005;310[5754]:1642-1646.

[24] Mohammed Talat Abbas, Riyadh Rashed AL-Tuma and Mohammed Jaffar Mohammed. THE ASSESSMENT OF OXIDATIVE STATE IN KERBALA PATIENTS WITH BENIGN PROSTATIC HYPERPLASIA BEFORE AND AFTER THE SURGERY. Biochem. Cell. Arch. Vol. 18, Supplement 1, pp. 000-000, 2018.

[25] Leiberman M, Marks Ad, Eds. Mark's Basic Medical Biochemistry A Clinical Approach. 3rd Ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2009:479-566.

[26] Clarke Pr, Hardie Dg. Regulation Of Hmg-Coa Reductase: Identification Of The Site Phosphorylated By The Amp-Activated Protein Kinase In Vitro And In Intact Rat Liver. Embo J 1990;9:2439-46.

[27] Attvall S, Fowelin J, Lager I, Von Schenck H, Smith U. Smoking Induces Insulin Resistance: A Potential Link With The Insulin Resistance Syndrome. J Intern Med. 1993;233:327–332.

[28] Facchini Fs, Hollenbeck Cb, Jeppesen J, Chen Yd, Reaven Gm. Insulin Resistance And Cigarette Smoking. Lancet. 1992;339:1128–1130.