Biochemical studies on the interelationship between insulin, leptin and thyroid function in diabetic patients

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ABSTRACT

The present study was conducted to assess the interrelationship and the influence of the coexistence of diabetes, leptin, thyroid and biochemical variables related to carbohydrate and lipid metabolism in patients suffered from either Type 1 diabetes (T1DM; IDDM) or Type 2 diabetes (T2DM; NIDDM). The study was performed on 100 individuals collected from the outpatient and inpatient clinics of diabetes**.** They divided into 5 groups as follow; volunteer apparently healthy, IDDM with complications, IDDM without complications, NIDDM with complications and NIDDM without complications. The obtained results revealed an increase in the level of glycosylated hemoglobin (HbA_{1c}) in individuals with higher levels of blood glucose, hypercholesterolemia and hypertriglyceridemia in diabetic patients with more increase in patients suffered from IDDM with complications. Patients suffered from NIDDM with complications show higher level of leptin than other groups. There is an increase in serum TSH level in Patients suffered from IDDM with complications than other groups with reduction in T3 and T4 levels. On the other hand, there is a decrease in serum TSH level in Patients suffered from NIDDM with complications than other groups with elevation in T3 and T4 levels. In conclusion, the present study indicates a significant negative correlation between serum TSH and leptin. A positive correlation is found between serum T4 and leptin in NIDDM with complications. A negative correlation is showed between serum T3, T4 and leptin in IDDM with complications. Additionally, there is a significant positive correlation between serum leptin and cholesterol, also between serum leptin and triglycerides in IDDM with complications.

Key words: Diabetes, obesity, leptin, thyroid function.

INTRODUCTION

Diabetes mellitus is a syndrome initially characterized by loss of glucose homeostasis, resulting from defects in insulin secretion, insulin action or both

Leading to impaired metabolism of glucose and other energy-yielding

hyperglycemia ***(Kahn et al, 2006 and Kalra, 2009).***

Obesity is one of the most important healthY risks of our time through increased risk of diabetes, dyslipidemia, kidney disease, cardiovascular disease, thyroid dysfunction and cancer ***(Biondi, 2010).***

Leptin is a 15-kDa hormone secreted mainly by adipocytes, although leptin expression in placenta, fetal tissue, stomach and other tissues was initially described as a protein important in food intake and body weight regulation. It transport from plasma crossing the blood– brain barrier through a saturable transport system and acting on receptors in the lateral and medial regions of the hypothalamus to suppress food intake and stimulate energy expenditure to regulate appetite and energy balance ***(Friedman and Halaas,1998; Konukoglu et al, 2006).*** Insulin is adipogenic, promotes fat deposition in the body, while leptin expression increases after peak insulin secretion during the feeding cycle ***(Kalra, 2008).***

Diabetes mellitus type 1 may be associated with thyroid dysfunction either hpothyroidism (Hashimoto's disease) or hyperthyroidism (Graves' disease) that are autoimmune disorders, with development influenced by genetic and environmental factors ***(Hawa et al, 2006 and Gonzalez et al, 2009)***.

It has been known that, HbA_{1c} can be used as a potential biomarker for fuels such as lipids and proteins. The disease is progressive and associated with a high risk of vascular diseases. There is macrovascular complications as coronary artery disease, cerebro vascular disease and microvascular complications, that found to cause nephropathy, retinopathy and neuropathy ***(White et al, 2003; D'Elia et al, 2011 and Papa et al, 2013).***

Diabetics suffer from either type 1 or type 2 diabetes. Type 1 diabetes (T1 D.M; Insulin Dependant Diabetes Mellitus; IDDM) is a debilitating autoimmune disease caused by T-cell-mediated gradual destruction of B –cells of pancrease, leading to either insufficient or complete lack of insulin production. T1DM is increasing in incidence worldwide, particularly in young children whom have gained more weight ***(Devendra and Eisenbarth, 2003; Gilliam et al, 2006).***

Type 2 diabetes (T2DM; Non Insulin Dependant Diabetes Mellitus NIDDM) is a progressive chronic disease that can manifest at any age due to largely persistent metabolic imbalance engendered by myriads of internal and external environmental factors, including diet and lifestyle changes ***(Lua et al, 2012).*** Increases in episodic basal and post-prandial insulin secretion initiated by these environmental shifts gradually and expedites B-cell dysfunction and loss that eventuates into unremitting

NIDDM with complications; Group V consisted of 20 patients suffered from NIDDM without complications. Two blood samples were collected from each person; the first was taken on sodium florid for determination of glycosylated hemoglobin (HbA_{1c}) that was estimated by using a fast ion exchange resin separation method ***(Niederau and Reinauer, 1981)*** by using commerical kit of Stanbio Laboratory, Inc, 1261 N Main St, Boerne, TX. The second sample was taken into a clean tube, centrifuged at 3000 r.p.m for 15 minutes and serum separated for determination of 2h post prandial blood glucose according to ***(Trinder, 1969)*** and total Cholesterol (T.C) according to ***(Richmond, 1973 and Allain et al, 1974)*** by using a commercial kit of Stanbio Laboratory. Serum triglycerides (T.G) was estimated according to ***(Fossati and Prencipe, 1982)*** by using commerical kit of Axiom diagnostic. HDL– C was estimated according to ***(Burstein et al, 1970 and Lopez-Virella et al, 1977)*** by using commerical kit of Bio Systems.

LDL- C was calculated by formula of ***(Friedewald et al, 1972).***

Serum LDL–C (mg /dl) = TC – HDL.C – $\frac{T.G}{5}$

TSH, T3, T4 and Leptin were measured in serum using a commercially available ELIZA kit

predicting dyslipidemia in type 2 diabetic patients in addition to glycemic control ***(Varashree and Bhat, 2011).*** Indeed, hyperlipidemia is the commonest complication of diabetes mellitus and it predisposes them to premature atherosclerosis and macrovascular complications. Common lipid abnormalities in diabetes are raised triglycerides, LDL-C, cholesterol and decreased HDL-C. Therefore good glycemic control can prevent development and progression of lipid-abnormalities among patients with diabetes mellitus ***(Uttra et al, 2011)***.

MATERIAL AND METHODS

The study was performed on 100 individuals collected from the outpatient and inpatient clinic of diabetes at Al Azhar University Hospitals (Al Hussein and Sayed Galal). Also outpatient clinic at El Sahel Teaching Hospital and El Manshawy Hospital were represented in our study. Those individuals are divided into 5 groups as follow; Group I consisted of 20 volunteer apparently healthy individual with age ranging from 15-39 years were chosen as control group; Group II: consisted of 25 patients suffered from IDDM with cardiovascular disease (CVD) as a major complication of diabetes; Group III consisted of 15 patients suffered from IDDM without complications; Group IV consisted of 20 patients suffered from

paired T-test and correlation. Data are given as mean± standard error (S.E) ***(Snedecor and Cochran, 1982).***

(Bio Check, Inc., 323 Vintage Park, Dr.).

Statistical analysis: Statistical analysis were carried out with SPSS using the one way ANOVA,

Results

Table (1): *Determination of fasting blood glucose, HbA_{1c}, leptin level in studied groups.*

Groups	Glucose (mg /dl)	HbA_{1c} (%)	Leptin (µg/dl)
Group I	95.50 ±3.04	6.84±0.10	5.82±0.32
Group II	295.05±11.41	11.80±0.28	104.86±2.65
Group III	189.85±5.72	7.71±0.19	83.20±3.72
Group IV	216.82±6.72	9.63±0.19	136.91±3.57
Group V	177.25±5.22	8.49±0.08	16.92± 0.84

Group I= Control group; Group II= Diabetic patients (IDDM) with complications; Group III = Diabetic patients (IDDM) without complications; Group IV= Diabetic patients (NIDDM) with complications & Group V= Diabetic patients (NIDDM) without complications.
Values represent mean ±SE.

Table (2): *Determination of TSH, T3 and T4 level in studied groups.*

Groups	TSH (µIU/ml)	T4 (µg/ dl)	T3 (ng/dl)
Group I	1.06±0.13	6.87±0.30	126.22 ±4.92
Group II	3.20±0.15	5.63±0.23	107.90±4.20
Group III	3.03±0.18	10.31±0.31	160.55±3.75
Group IV	0.27±0.04	12.24±0.34	191.69±2.01
Group V	1.81±0.18	7.49±0.21	153.30±3.74

Table (3): *Determination of T.C, T.G, HDL- C& LDL- C level in studied groups.*

Groups	T. C (mg/dl)	T.G (mg/ dl)	HDL- C (mg/dl)	LDL- C (mg/dl)
Group I	162.21±5.61	98.64±4.06	37.92± 0.80	92.89 ±4.50
Group II	310.70±8.15	234.25±4.92	57.65±1.41	236.2 ±8.78
Group III	203.20±2.50	126.55±5.46	54.60±1.21	143.29 ±3.35
Group IV	288.43±6.85	255.69±4.38	65.47±1.20	206.82 ±6.67
Group V	213.90±3.04	159.05±9.54	49.60±1.77	144.09 ±3.35

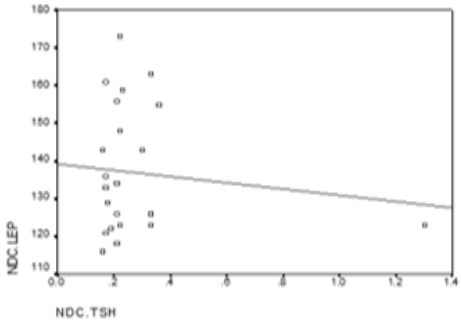

Figure A

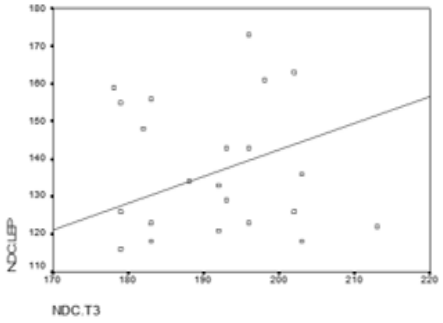

Figure B

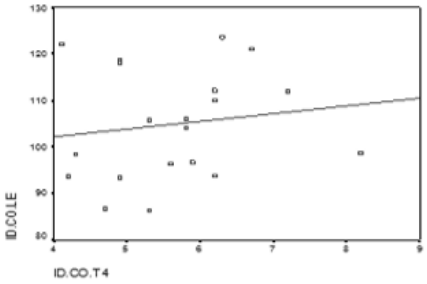

Figure c

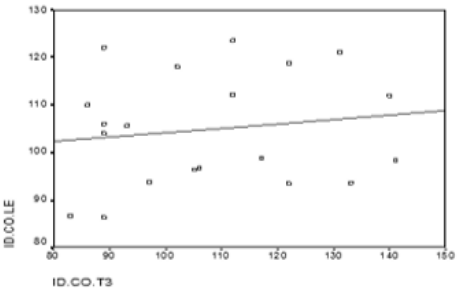

Figure D

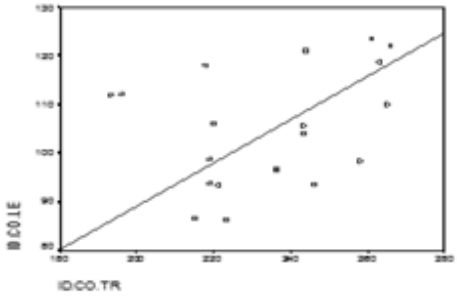

Figure E

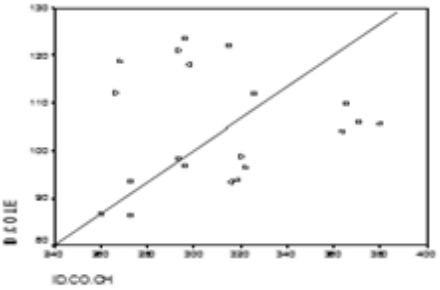

Figure F

Further investigation proposed that, the increase in serum leptin was exponentially with increased fat mass in obese subjects whom are at increased risk of type 2 diabetes mellitus. Since, the B-cell may be adversely affected by chronic increased leptin level which exerts a dynamic regulatory restraint on insulin efflux from B-cells eventually leading to diabetes ***(Soodini and Hamdy, 2004; Otukonyong et al, 2005; Hamed et al, 2011).***

The conclusion of, ***Niswender and Schwartz (2003)and Gilliam et al (2006)*** hypothesized that leptin plays a role in the development of autoimmunity by facilitating the generation of autoantibodies targeting pancreatic beta cells promoting a T1DM immune response.

Our work recorded an increase in the level of glycosylated haemoglobin in individuals with higher levels of blood glucose, this result came in accordance with the result of ***(Varashree and Bhat, 2011)*** **who** reported that, human erythrocytes are freely permeable to glucose and within each erythrocyte glucose can bind non enzymatically to hemoglobin and glycated hemoglobin is formed that is dependent on the ambient glucose concentration. The glycation process is slow and continuous that occurs over days to 3-4 months. In a normal person about 3-6% of HbA is glycated; in a diabetic patient the percentage of HbA may double or

DISCUSSION

The obtained results revealed an increase in the level of glycosylated haemoglobin in individuals with higher levels of blood glucose, there are a high significant increases in serum fasting blood glucose, HbA_{1c} and leptin levels in both diabetic patients (IDDM, NIDDM) either with or without complications than control group. Patients suffering from IDDM with complications show higher level of fasting blood glucose and HbA_{1c} than other groups, while patients suffered from NIDDM with complications show higher leptin level than other groups **(table 1).**

Several assumptions have been suggested to explain the hyperglycemia in diabetes. This is plausible that, hyperglycemia in IDDM may be attributed to either insufficient or complete lack of insulin production according to the degree of B- cell destruction ***(Devendra and Eisenbarth, 2003; Gilliam et al, 2006)***. Concerning hyperglycemia in NIDDM that more prevalent among obese subject may be due to insulin receptor insensitivity, insulin resistance and diminished downstream insulin receptor signaling in target cells. The relentless compensatory insulin hypersecretion to normalize blood glucose levels under these conditions expedites B-cell dysfunction that eventuates into unremitting hyperglycemia ***(Kahn et al, 2006 and Kalra, 2009).***

(Devendra and Eisenbarth, 2003; Marzullo et al, 2010) address the intriguing hypothesis of a link between obesity and hypothyroidism where high leptin level in obese persons increasing the susceptibility to thyroid autoimmunity, which in turn entails a high risk of developing hypothyroidism (Hashimoto's disease) where a lymphocyte infiltrate destroys the thyroid gland.

On the other hand, there is a reduction in serum TSH level in patients suffered from NIDDM with complications than other groups with elevation in T3 and T4 levels **(table 2).** As well as, there is a significant negative correlation between serum TSH and leptin in NIDDM with complications **(figure A).** A positive correlation is found between serum T4 and leptin in NIDDM with complications **(figure B**). These results is agree with the result of ***De- Pergola et al (2007)*** who reported that, there was a positive correlation has been reported between the T3 to T4 ratio and both waist circumference, body mass index (BMI) in obese patients. This finding suggests a high conversion of T4 to T3 in patients with central fat obesity due to increased deiodinase activity by leptin as a compensatory mechanism for fat accumulation to improve energy expenditure. This is not surprising becauseT3 regulates energy metabolism, thermogenesis and plays a critical role in glucose, triple depending on the degree of hyperglycemia). The evidences of ***(Tsukahara et al (2003) and Abd-El Dayem et al (2012)*** suggested that, a small proportion of glycated products are then irreversibly transformed, over several weeks to months, into advanced glycosylation end products (AGEs) that accumulate in a variety of collagenous structures, such as vascular wall collagen and basement membranes result in endothelial dysfunction and vascular complications. There is increasing evidence that AGEs play a pivotal role in atherosclerosis and renal failure in diabetes.

These data exhibit an increase in serum TSH level in Patients suffered from IDDM with complications than other groups with reduction in T3 and T4 levels **(table 2).** Thus, the present study is consistent with many others which reported that T4 exerted negative feedback on TSH ***(Simşek et al, 1997; Imaizumi et al, 2011)***.

It was obvious in the present study that, there is a negative correlation is showed between serum T4 and leptin in IDDM with complications **(figure C),** also between serum T3 and leptin in IDDM with complications **(figure D).** These result is agree with the results of ***(Johnson, 2006 and Okten et al, 2006)*** that confirm that patients with type 1 DM have higher prevalence of positive thyroid autoantibodies than healthy controls. Moreover, The issues of

atherosclerosis, and coronary artery disease. The most important products of adipose tissue collectively referred to as adipocytokines, include adiponectin, leptin, tumor necrosis factor–alpha (TNF-), interleukin-6 (IL-6), resistin, plasminogen-activating inhibitor–I (PAI-1) and angiotensinogen.

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lipid metabolism, food intake, and the oxidation of fatty acids.

Our study reveals high prevalence of hypercholesterolemia and hypertriglyceridemia which are well known risk factors for cardiovascular disease in diabetes. Since, patients suffered from IDDM with complication show higher level of total cholesterol, LDL- C, and triglycerides than other groups **(table 3)**. These results agree with the result of ***(Vinodmahato et al (2011)*** who revealed that, insulin affects the liver apolipoprotein production. It regulates the enzymatic activity of lipoprotein lipase and cholesterol ester transport protein. All these factors are likely cause of dyslipidemia in diabetes mellitus. Moreover, insulin deficiency reduces the activity of hepatic lipase resulting in hypertriglyceridemia.

Also, our study reveals a significant positive correlation between serum leptin and triglycerides **(figure E**), as well as between serum leptin and cholesterol **(figure F)** in IDDM with complications. These results are confirmed with the results of ***(Soodini and Hamdy (2004); Vinodmahato et al (2011); Uttra et al (2011)*** whom demonstrated that, an increased amount of adipose tissue or its disproportionate distribution between central and peripheral body regions is related to the development of insulin resistance, type 2 diabetes mellitus, hypercholesterolemia, hypertriglyceridemia dyslipidemia,

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الملخص العربي

دراسات كيميائية حيوية على العلاقة بين الأنسولين، هرمون الليبتين ووظيفة الغدة الدرقية في مرضى البول السكري

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أجريت هذه الدراسة لتقييم العلاقة وتأثير التعايش بين مرض البول السكري، الليبتين والغدة الدرقية والمتغيرات البيوكيميائية الخاصة بالتمثيل الغذائي للكربوهيدرات و للدهون في المرضى الذين يعانون سواء من النوع الاول أو النوع الثاني لمرض البول السكري. وقد أجريت الدراسة على ١٠٠ شخص تم تجميعهم من العيادات الخارجية والعيادات الداخلية لمرض البول السكري و قسموا إلى ٥ مجموعات على النحو التالي؛ اصحاء متطوعين، مرضى النوع الاول مع وجود مضاعفات، مرضى النوع الاول بدون مضاعفات، مرضى النوع الثاني مع وجود مضاعفات، مرضى النوع الثاني بدون مضاعفات. وقد أظهرت النتائج زيادة في مستوى الهيموجلوبين السكري في الدم في الأشخاص الذين يعانون من مستويات أعلى للسكر في الدم، ارتفاع الكوليسترول و الجليسريدات الثلاثية بالمصل في مرضى البول السكري مع زيادة أكثر في المرضى من النوع الاول مع وجود مضاعفات. وجد أعلى مستوى لهرمون الليبتين بالمصل **في** المرضى من النوع الثاني مع وجود مضاعفات مقارنة بالمجموعات الأخرى. وهناك زيادة في مستوى الهرمون المنشط للغدة الدرقية مع انخفاض في مستوي هرمونات الغدة الدرقية T3, T4 بالمصل في المرضى من النوع الاول مع وجود مضاعفات مقارنة بالمجموعات الأخرى. على الناحية الأخرى وجد انخفاض في مستوى الهرمون المنشط للغدة الدرقية مع ارتفاع في مستوي هرمونات الغدة الدرقية T3, T4 في المرضى من النوع الثاني مع وجود مضاعفات مقارنة بالمجموعات الأخرى. في الختام، تشير الدراسة الحالية الى وجود علاقة سلبية ذات دلالة إحصائية بين الهرمون المنشط للغدة الدرقية واللبتين و وجدت علاقة إيجابية بين هرمونات الغدة الدرقية واللبتين **في** المرضى من النوع الثاني مع وجود مضاعفات. وأظهرت وجود علاقة سلبية بين هرمونات الغدة الدرقية واللبتين في المرضى من النوع الاول مع وجود مضاعفات. بالإضافة إلى ذلك،وجد ان هناك علاقة إيجابية بين اللبتين والكوليسترول، وأيضا بين اللبتين و الجليسريدات الثلاثية في المرضى من النوع الاول مع وجود مضاعفات.