The Correlation Between Blood Glucose Level and Lipid Profile in Hypothyroid Diabetic Patients in Baghdad

Nihad Hameed Dawood Dentistry College, Al-Mustansiriya University

Abstract:

The objective of this study was to investigate the effects of thyroid hormones disorders on serum lipid profile among the hypothyroidism diabetic patients. For this purpose blood samples were collected randomly from (40) subjects with hypothyroidism (TSH> 5 m IU/L, T4 < 50 n mol/L) (male and female) from the endocrine center of Al-Yarmook teaching hospital in the age range of (30- 65) years. Included (21) diabetic patients who had thyroid dysfunction (hypothyroidism) according to clinical examinations and laboratory results, (were chosen as case group) and the remaining (19) subjects were consider as control group.

The following variables were measured: Fasting serum glucose, thyroid hormones (T3, T4 and TSH) and lipid profiles (total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), Very Low density lipoprotein VLDL and triglycerides (TG)).

The statistical analysis of results of this study revealed a significant correlation between fasting serum glucose (FSG) with severity of thyroid hormones disorders levels (T3, T4 and TSH), beside significantly increased serum total cholesterol (include HDL, LDL and VLDL) and triglyceride (P. value < 0.05) with hyperglycemia.

We conclude that hyperglycemia is associated with significant changes in lipid profile and thyroid hormones level in hypothyroid-diabetic patient as compared with non diabetic control group with negative correlations.

الخلاصة:

ان الهدف من هذه الدراسة هو بيان تاثير اضطرابات هرمونات الغدة الدرقية على الدهون في الدم لدى المرضى المصابين بالسكري المصاحب لانخفاض وظيفة الغدة الدرقية.ولهذا الغرض تم اختيار مجموعتين الاولى شملت (21) من مرضى السكري الذين لديهم قصور في وظيفة الغدةالدرقية، وفقا للفحوصات السريرية والنتائج المختبرية، ومجموعة السيطرة شملت (19) شخص المتبقين وهم مصابين بانخفاض مستويات هورمونات الغدة الدرقية دون الاصابة بالسكري في مختلف المجاميع العمرية من كلا الجنسين.

. تم جمع عينات عشوائية من (40) شخصا (ذكورا وإناثا) من مركز الهرمونات في مستشفى اليرموك التعليمي في الفئة العمرية من (30 إلى 65) سنة من كلا المجمو عتين.

تم قياس المتغيرات التالية: نسبة السكر في مصل الدم، وكذلك هرمونات الغدة الدرقية وقياس انواع الدهون والتي شملت الكولستيرول الكلي والبروتين الدهني عالي الكثافة والمنخفض الكثافة والمنخفض الكثافة جدا والدهون الثلاثية إن التحليلات الإحصائية كشفت عن وجود إرتباط كبير بين ارتفاع نسبة السكر في مصل الدم مع شدة إضطرابات مستويات هرمونات الغدة الدرقية وترتبط ايضا مع زيادة كبيرة في الدهون شملت الكولسترول الكلي في الدم والدهون الثلاثية.

نستنتج أن ارتفاع السكر في الدم يرتبط ارتباطاً سلبيا مهما مع نسبة الدهون ومستوى الهرمونات في الغدة الدرقية للمرضى المصابين بداء السكري بالمقارنة مع مجموعة السيطرة.

Introduction:

Hypothyroidism is a clinical syndrome resulting from a decrease in the thyroid hormones T_4 and T_3 .

It is classified as primary, seconddary or tertiary according as to whether the cause of the disease is localized respectively in the thyroid, both subclinical and symptomatic hypothyroidisms are associated with various risk factors for atherosclerotic disease, diastolic hypertension, hypercholesterolemia, hypertriglyceridemia and endothelial dysfunction^[1]. Most of the thyroid hormone released is in the form of T4, as total serum T4 is 40-fold higher than serum T3^[2]. Free thyroxin (FT4), this is less active form of thyroid hormones converted to the more active thyroid hormone T3.Iodine is an essential nutrient for the normal growth, metabolism and regulation of thyroid hormones in humans and animals^[4].

Iodine deficiency could be associated with an increased risk of hypothyroidism due to lack of substrate for thyroid hormones synthesis as observed in area with severe iodine deficiency and it has a central role in thyroid epidemiology^[3].

Hypothyroidism can lead to the following problems (Heart and circulation problems include: Slow heart rate, high blood pressure (makes arteries less elastic).

Atherosclerosis, Diminished pumping ability (congestive heart failure), Gastrointestinal problems (constipation), Mental emotional problem (mental processes sluggish and personality placid), Neuromuscular problems (weakness, sluggish reflexes).

Also. Hypothyroidism alters lipoprotein metabolism in the liver and associated with increased cholesterol levels, specifically intermediate - density lipoprotein (IDL) and low density lipoprotein (LDL). Low levels of LDL receptors in the liver decreased hepatic lipase and lipoprotein lipase concentra $tion^{[2,3]}$.

Diabetes mellitus and thyroid diseases are two common endocrineiopathies seen in the adult population. Long term thyrotoxicosis, has been shown to cause beta cell dysfunction resulting in reduced pancreatic insulin content, poor insulin response to glucose and decreased rate of insulin secretion^[5].

In hypothyroidism, the synthesis and release of insulin is decreased ^[6].

The rate of hepatic glucose output is decreased probably due to reduced gluconeogenesis. Moreover; thyroid hormones have significant effects on the synthesis, mobilization and metabolism of lipids^[7]. Overt hypothyroidism is associated with significant increases in circulating concentrations of total and low density lipoprotein cholesterol (LDL-C)^[8]

Measurement of serum cholesterol levels is important as an indicator of liver function, intestinal absorption and billary function and in the diagnosis and classification of hyperlipoproteinemia elevated cholesterol levels may occur with hypothyroidism, diabetes and nephritic syndrome.

Elevated serum cholesterol levels correlate well with the incidence of coronary artery disease, stress, age, gender; hormonal balance and pregnancy affect normal cholesterol levels.

Depressed levels are associated with hyperthyroidism and sever liver disease [2, 4].

(as well as the HDL particles carry cholesterol from the cells back to the liver . HDL is known as good cholesterol because high levels are thought to lower the risk of heart disease ^[9].

The LDL particle is a lipoprotein that transports cholesterol to the cells often called bad cholesterol because of high levels of risk factor for coronary heart disease to be associated with obesity, diabetes and nephritis ^[10].

Triglycerides are the main lipids present in the human plasma, the others are cholesterol, phospholipids and non estrified fatty acids.

Triglycerides measurements are used in the diagnosis and treatment of patient with diabetes mellitus, liver obstruction, nephrosis and other disease associated with lipid metabolism^[9].

Hyperlipidaemia is a metabolic abnormality is frequently associated with diabetes mellitus. Its prevalence is variable, depending on the type and severity of diabetes, glycemic control, nutritional status, age and other factors.

The present study designed to study the correlation between blood sugar level and thyroid functionality disorders (hypothyroidism) by assessment the serum level of thyroid hormones (T3, T4 and thyroid stimulating hormone TSH) with level of different serum lipid components (total cholesterol. LDL. VLDL and Triglyceride) in diabetic patients compared with non diabetic hypothyroid patients.

Materials and Methods:

The study was performed during the period from February (2011) to December (2011) for this purpose (21) hypothyroid diabetic subjects were from people selected attended to specialized center for Endocrinology and Diabetes, their ages ranged between(30-65) years include(14) female (66.6%) and (7)male(33.4%).

These were compared with (19) control group (non diabetic hypothyroid patients), their ages ranged between (30-65) years include (12) female (63.2%) and (7) male (36.8%).

Careful history was obtained from patients including age, gender for each patient the following tests were carried out:

1- Thyroid function tests: Which include T3, T4, and TSH using radioimmunoassay method of

- (immunotech, company).
- 2- Blood sugar test:

(using enzymatic colorimetric method/ Spain).

3- Lipid profile (triglycerides, cholesterol and lipoproteins (HDL,LDL,VLDL):

CHOD-PAP-enzymatic By using colorimetric method and GPO-PAPenzymatic colorimetric method /from spectrum company (Egypt) and spin react company(Spain).

Laboratory Assessment:

samples Venous blood were collected after an overnight fasting, total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, triglyceride (TG) and fasting blood glucose (FBG) were tested for each patient was labeled as hypothyroid if the TSH was > 5 m IU/L, serum was separated and assays were performed within 24 hours was measured by immunoradiometric assay kits are intended for the quantitative determination of thyroid hormones in serum or plasma.

The principle of TSH assay is sandwich type assay, the kit has mouse monoclonal antibodies directed against two different epitopes of TSH and hence not competing is used.

The samples or standards are incubated in monoclonal antibody which is labeled with I¹²⁵, after incubation, the content of the tubes is aspirated and the tubes are rinsed so as to remove unbound I^{125} labeled antibody, the bound radioactivity is then determined in a gamma counter.

The concentration of TSH in the samples are obtained by interpolation from the standard curve also the level of T3 and T4 were measured by kits depending on radio immune assay method intended for the quantitative direct determination of thyroxin concentration in serum or plasma in which the radioimmunoassay of total T4 and T3 is a competitive immune analytical determination.

Unknown sample and standards are incubated together with I^{125} .

Thyroxin in monoclonal anti-T4 antibody -coated tubes, after incubation the contents of the tubes are aspirated and the bound activity is measured in a gamma counter, the concentration of T4 is reversely proportionate to the radioactivity measured ,the values of unknown samples are read off the calibration curve (the same procedure used to measure the T3 level).

The expected normal values of T4 between (60-160nmol/L) and the expected normal value to T3 between (1.2-2.8nmoI/L) (4, 19). The concentration of serum total cholesterol, HDL cholesterol and triglycerides were measured by a timed-end point method (Using Spectrum, Egyptian) and LDL cholesterol was calculated by Fried Wald's formula using SPINEACT auto analyzer, Spain).

kit

Statistical Analysis:

The following statistical data analysis approaches were used in order to analyze and assess the results of the study: **Descriptive data analysis**:

Mean, Standard Deviation, Range, Two Extreme values (min. and max.) readings. Person's correlation coefficient.

Inferential data analysis:

These were used to accept or reject the statistical hypotheses, which included the following: Analysis of Variance (ANOVA) for equality of means of several independent Groups with least significant difference (LSD).

Student t-test for testing of Person's correlation coefficient at levels of p values > 0.05 to be considered significant.

 Table-1: Summary of the studied parameters of thyroid function tests for the different studied groups with comparison significant.

Statistic	FBG(m mol/L)		T3nmoI/	L	T4nmoI/L		TSH m I U/L				
	Control	Нуро	Control	Нуро	Control	Нуро	Control	Нуро			
Mean	5.27	7.1	1.55	0.61	75.25	43.15	2.87	63.35			
S D	0.50	0.72	0.22	0.15	6.54	10.46	0.82	15.45			
Min	4.60	6.2	1.20	0.50	66.00	30.00	1.00	35.00			
Max	6.0	7.6	1.90	1.10	87.00	64.00	4.00	78.00			
Range	1.80	2.00	0.70	0.50	21.00	34.00	3.00	43.00			
C.S.	Cont. X	Hypo:	Cont. Y	К Нуро:	Cont. X	Hypo:	Cont. X	Hypo:			
P-value	P=0.377 NS		P=0.000 HS		P=0.000 HS		P=0.000 HS				

^(*) NS: Non sig. at P>0.05 ; S: Sig. at P<0.05 ; HS: Highly sig. at P<0.01; F.B.G : Fasting blood glucose ; T3:triiodothyronine ; T4:Thyroxine ;TSH: Thyroid stimulating hormone ; Control : non diabetic hypothyroidism ; Hypo : diabetic hypothyroidism . (By applying Least significant difference LSD after ANOVA).

Results:

The distribution of study and control groups according to the gender demonstrated that a large percentage of patients were women 14 (66.66%) in comparison with male 7 (33.34%).

In this study, the prevalence of hypothyroidism was more common among women than men.

Statistically, there is a highly significant difference was found between number of females to males in comparison with control group (p. $v_{alue}=0.000$).

As well as this study certain that most of hypothyroidism diabetic patients was in age above 40 years.

Thyroid hormones distribution of study population:

Results in table (1) showed the distribution of study and control groups according to (blood sugar level, serum level of T_3,T_4 and TSH), these results showed a significant differences between

majority of hypothyroid diabetic patients and control cases in which the results obtained from

table(1) showed that mean of blood glucose level of patients study group was (7.1 m mol/L), while the mean of FBG in control group was (5.27 m mol/L), the statistical analysis revealed no significant differences (p.value >0.05) was observed, in addition the results revealed low T_3 level(0.61 n mol /L)in study group and had high T3 (1.55nmoI/L) in control group with statistical significant highly differences p. value = 0.000), while the results of study group when compared to subjects showed statistically control significant decrease in the level of serum T4 (p. value = 0.000 HS), while the serum level of TSH was significantly increase in study group(63.35 m IU/L)(p. value = 0.000).

Table-2: Summary of the studied parameters of Lipid profile for the different groups with comparison significant. By applying least significant difference LSD after ANOVA.

Statistic	Chol (m mol/L)		TG (m mol/L)		LDL (m	mol/L)	HDL (m	mol/L)	VLDL(m mol/L)	
	Control	Нуро	Control	Нуро	Control	Нуро	Control	Нуро	Control	Нуро
Mean	4.81	7.16	1.26	2.60	3.26	5.39	0.97	1.38	0.36	0.50
Std.	0.69	0.58	0.39	0.64	0.87	0.64	0.10	0.33	0.08	0.15
Deviation										
Minimum	4.00	6.50	0.70	2.00	1.60	4.70	0.80	0.88	0.30	0.36
Maximum	6.10	8.30	2.00	4.00	4.70	6.65	1.10	1.90	0.55	0.85
Range	2.10	1.80	1.30	2.00	3.10		0.30	1.02	0.25	0.49
C.S.	Cont. X Hypo:		Cont. X Hypo:		Cont. X Hypo:		Cont. X Hypo:		Cont. X	Hypo:
P-value ^(*)	P=0.000 HS		P=0.000 HS		P=0.000 HS		P=0.000 H	IS	P=0.000 HS	
(*) ~										

^(*) NS: Non sig. at P>0.05; S: Sig. at P<0.05; HS: Highly sig. at P<0.01Chol. : Cholesterol; TG: Triglyceride; LDL: low density lipoprotein ; HDL : high density lipoprotein; VLDL :very low density lipoprotein; Control : non diabetic hypothyroidism ; Hypo : diabetic hypothyroidism . (By applying least significant difference LSD after ANOVA).

Lipid distribution of study population:

The mean levels of total serum cholesterol, triglycerides, LDL- chol., HDL- chol. and VLDL conducted in Table (2),in which the serum level found to be mean \pm SD as follow of serum level found to be (7.16 \pm 0.58, 2.60 \pm 0.64, 5.39 \pm 0.64, 1.38 \pm 0.33 and 0.50 \pm 0.15 mg/dl) respectively in study group.

In general these results revealed that all types of serum lipid profile were showed statistically significant increase in all patient group when compared to control subjects in which the levels of serum total cholesterol (P<0.001), serum triglycerides (P<0.001), serum LDL - cholesterol (P<.0001) as well as serum HDLcholesterol levels and VLDL did show statistically significant difference between the two groups (P<0.05), as shown in table-2.

Generally, the results of hypothyroidism diabetic's patients when compared to control non diabetic Subjects showed statistically significant increase in the levels of serum total lipids (P. < .001), so in this study the most characteristic lipid abnormality observed in diabetics hypothyroidism when compared to non diabetic persons.

ny pomyroluism maic parchis.											
Correlation	Parameters	Age	T3	T4	TSH	Chol	TG	LDL	HDL	VLDL	
		Groups									
Pearson's	F. blood	0.844	-	-0.194	-0.133	0.122	0.205	0.034	0.019	0.079	
Coefficients	glucose	(**)	0.209								
	Age Groups		0.061	0.097	-0.509	0.313	-	0.264	0.300	-0.269	
							0.127				
	Т3			0.925 (**)	-0.845 (**)	0.814 (**)	0.353	.870 ^(**)	-0.280	0.303	
	T4				-0.852 (**)	0.930 (**)	0.143).959 ^(**)	-0.258	0.080	
	TSH					-0.838	-	-0.850	0.014	0.062	
						(**)	0.052	(**)			
	Cholesterol						0.225	.980 ^(**)	-0.288	0.109	
	Triglyceride							0.204	-0.694 (*)	0.974 (**)	
	LDL								-0.268	0.119	
	HDL									-0.728 (**)	
Sig.	F. blood	0.001	0.281	0.296	0.357	0.368	0.285	0.462	0.479	0.415	
(1-tailed)	glucose										
	Age Groups		0.433	0.394	0.067	0.189	0.363	0.231	0.200	0.226	
	T3			0.000	0.001	0.002	0.159	0.001	0.217	0.197	
	T4				0.001	0.000	0.347	0.000	0.236	0.413	
	TSH					0.001	0.444	0.001	0.484	0.433	
	Cholesterol						0.266	0.000	0.210	0.382	
	Triglyceride							0.286	0.013	0.000	
	LDL								0.227	0.372	
	HDL									0.008	
(1)		7.1.1.1.									

Table-3: Pearson's correlation coefficients among different of the parameters for hypothyroidism male patients.

^(*)Sig. at P<0.05 ;^(**) HS: Highly sig. at P<0.01

The coefficients of correlation by using person's coefficients between fasting blood glucose in male diabetic hypothyroidism patients (study group) and the thyroid values and the lipid profile values are shown in Table-3.

The results recorded there was a significant negative correlation between glucose level(high) and serum level of T3 and T4 $(Low)^{(*)}$ Sig. at P<0.05; ^(**) HS: Highly sig. at P<0.01. These results confirmed by using significant one tailed correlation as shown in table-3.

A positive correlation was significant appeared between blood glucose (high) and lipid profile include (cholesterol, triglyceride, LDL, HDL and VLDL) in study subjects (^{**}) Sig. at P<0.05;^(**) HS: Highly sig. at P<0.01In both male and female.

In the current study, the FBG values were inversely related to the T3 and free T4 values. These results suggest that the effect of hypothyroidism in the lipid metabolism is more marked in patients with higher serum glucose levels.

Data illustrated in table (3 and4), demonstrated the distribution of study and control groups according to age group, the results confirmed present of highly significant differences with positive correlation, mean increase the severity of disease with old age.

nypomyroiuisin iemaie pauenis.											
Correlation	Parameters	Age Groups	T3	T4	TSH	Chol	TG	LDL	HDL	VLDL	
Pearson's	F.B.G	0.112	-0.290	0.152	-0.343	0.016	0.173	0.158	0.562 ^(*)	0.246	
Coefficients	Age Groups		-0.670 (*)	-0.660 (*)	0.659 (*)	0.579 (*)	0.489	0.504	-0.391	0.426	
	Т3			0.850 (**)	-0.650 ^(*)	-0.835 (**)	-0.805	0.776	0.824 (**)	•0.795 ^(**)	
	T4				0.943	-0.666 (*)	-0.539	-0.510	0.445	0.486	
	TSH					0.457	0.284	0.264 (**)	-0.163).208	
	Cholesterol						0.976	0.970	• 0.788 **)).945 ^(**)	
	Triglyceride							0.993	• 0.863 **)).988 ^(**)	
	LDL								• 0.844 **)).981 ^(**)	
	HDL									0.876 ^(**)	
Sig.	F.B.G	0.379	0.208	0.338	0.166	0.483	0.317	0.332	0.045).246	
(1-tailed)	Age Groups		0.017	0.019	0.019	0.040	0.076	0.069	0.132).110	
	Т3			0.001	0.021	0.001	0.002	0.004	0.002).003	
	T4				0.000	0.018	0.054	0.066	0.099	0.077	
	TSH					0.092	0.213	0.230	0.327).282	
	Cholesterol						0.000	0.000	0.003	0.000	
	Triglyceride							0.000	0.001	0.000	
	LDL							0.070	0.001	0.000	
(4)	HDL									0.000	

 Table-4: Pearson's correlation coefficients among studied parameters for hypothyroidism female patients.

^(*)Sig. at P<0.05 ;^(**) HS: Highly sig. at P<0.01

Discussion:

Hypothyroidism can be defined when thyroid gland, fails to produce enough of hormones called the thyroid hormones or when the body fails to use thyroid hormones efficiently. It is a common endocrine disorder, is more common in women, the elderly and those with autoimmune disorders^[13].

The correlation between age, gender and hypothyroidism:

The results of this study revealed that highly significant association among the increase of sugar serum level, disorders of thyroid hormones (T3, T4 and TSH) with increase the age.

So, the statistical analysis revealed a positive correlation with significant appearance (at p value < 0.05) between sugar level and aging while negative correlation of the level of thyroid hormones (T3, T4) in serum with age as shown in table (3,4).

These results can be discussed because the body's decrease in use of

T4correlates with age lead to decline in lean body mass, suggesting that tissue (i.e. muscle, skin, bone) decrease, which, may lead to reduced use and catabolism of thyroid hormones^[14].

T4 is the major hormone produced by the thyroid and undergoes monodeiodination in peripheral organs (mainly: liver, kidney and pituitary) to generate T3, the metabolically active thyroid hormone^[9].

The above results were in agreement with the results of Morganti et. $al^{[14]}$ and Niafar et.al.^[15].

Who reported that an increased prevalence of hypothyroidism is demonstrated in the older population.

While, the study of ^[16] showed that the higher prevalence of hypothyroidism among middle aged women and associated with an increase in total plasma cholesterol. In the population we studied, the results showed that thyroid dysfunction was more common among females (14) (66.66%) than males (7) (33.34%), this percentage is higher than that in a study performed in $Italy^{[18]}$. showed an increase in numbers of females over male due to disturbance in reproductive hormones, particularly estrogen and progesterone, causing fatigue or nervous tension or during the stage of pregnancy or the menstrual cycle, it had been found that estrogen hormone raises the concentration of the transport proteins for thyroxin (TBG) through slowing down the process of their removal from the blood and enhancing its production in the body, also the estrogen hormone stimulates the immune system, causing increased production of anti-thyroid antibodies^[19]. While another study found that the incidence of thyroid diseases in women after the cessation of menses was (2.4%) with clinical hypothyroidism and (23%) with subclinical hypothyroidism^[20].

The correlation among FBG, thyroid hormones and lipid profile:

The effects of the thyroid hormones on metabolism are many and varied. Thyroid hormones stimulate the basal rate of metabolism, oxygen (O₂) consumption, heat production and are necessary for normal nervous system development and growth. Thyroid hormones linear participate in the control of systemic blood pressure, stimulation of heart contractions, maintenance of body weight, stimulation of protein synthesis and breakdown, stimulation of carbohydrate metabolism with increases in gastrointestinal and cellular absorption of glucose, glycolysis gluconeogenesis and increase in and vitamin requirement^[21].

Results obtained from table (1) revealed the distribution of study and control groups according to TSH level, T3and T4 the table showed that patients group had high TSH level (mean= 63.3mIU/L), and (mean of TSH= 2.87mIU/Lgroup) had low TSH level in control And level of T3 and T4 (mean= 0.85 and 43.15nmoI/L) respectively, have low level when compared with control with statistically a highly significant differences in the thyroid hormones level in the study group (P. _{value}= 0.000) when compared with their TSH level in control group.

In the current study, the TSH values were inversely related to the free T4 values.

These results supporting the use of serum TSH level as the best test to detect abnormal thyroid function ^[8,9].

In addition, in this study the results confirmed that increase level of blood glucose significantly association with decrease serum level of T3 and T4 as shown in table 1, these results can be explained because most of hypothyroidism patients has been associated with disorders of glucose and insulin metabolism, involving defective insulin secretion in response to glucose, hyperinsulinemia, altered peripheral glucose disposal and insulin resistance.

According to in vivo data, hypothyroidism is associated with a decreased glucose-induced insulin secretion by the β cells due to changes in the physicochemical properties of the islet membranes and decreased amount of islets.

In addition, it is suggested that hypothyroidism is an insulin resistant state, interestingly; even subtle decreases in the levels of thyroid hormone within the normal range have been shown to correlate inversely with markers of insulin resistance.

In vivo data, emerging from studies in propylthiouracil-induced hypothyroid animals during euglycemic-hyperinsulinemic clamps, showed an association of hypothyroidism with an adipokinemediated insulin resistance ^[10,22]. Table (2and3) represented the distribution of study and control groups according to cholesterol, HDL,VLDL level, showed that most of study group has abnormal cholesterol level and the statistically analysis revealed there a high significant differences were found in cholesterol, VLDL, level in hypothyroid patients when compared with the control group (P. _{Value} Highly sig. at P<0.01).

The changes in the lipid profile are explained by the regulatory effect of thyroid. Dyslipidemia is commonly found in overt and subclinical hypothyroidism. In hypothyroid patients, despite the reduced activity of 3-hydroxyl-3-methyl glutaryl -Coenzyme A (HMG-Co A) reductase, there is often an increase in the serum total cholesterol concentration, mainly due to raised levels of serum LDL cholesterol and decreased activity of LDL - receptor's resulting in decreased receptor-mediated catabolism of LDL and IDL is the main the hypercholesterolemia cause of observed in hypothyroidism ^[9,23].

From this study it can be concluded that hypothyroidism is associated with lipid disorders that are characterized by sever or slightly elevated total cholesterol levels increased LDL - chol and lower HDL- chol.

In some studies reported that HDLcholesterol or LDL/HDL-cholesterol ratios have been shown to be inversely correlated with prevailing blood glucose levels ^[24].or with glycosylated hemoglobin levels, as an index of blood glucose control^[25].

This study has clearly shown that all lipid fractions are abnormally elevated in diabetics when compared with controls. There are studies which seem to suggest that the lipoprotein significantly altered by the degree of metabolic control ^[26]. However, this ^[27] has not been confirmed by others.

In general the alterations in thyroid status appear to be associated with changes in serum triglyceride concentrations.

In human hyperthyroidism, decreased, normal ^[28]. and increased serum concentrations of triglyceride have been observed. Concentrations of serum triglyceride, although usually elevated in human hypothyroidism have also been to be normal ^[29].

The results of this study agree with study of Schaeffer et. al.^[32].

inverse They observed that correlation between HDL-cholesterol with adiposity and TG levels. Some other studies, reported that HDL-cholesterol or LDL/HDL-cholesterol ratios have been shown significant increase with prevailing [24] levels or blood glucose with glycosylated hemoglobin levels, as an index of blood glucose control ^[21].

This study has clearly shown that all lipid fractions are abnormally elevated in diabetics when compared with controls. and suggest that the lipoprotein significantly altered by the degree of metabolic control, as well as realizing that most of the diabetics have a high probability of developing cardiovascular and cerebrovascular disease.

Several of past studies discuss the correlation between diabetic and lipid profile such as, Cohen et.al. (1979) showed significant increase in the level of serum LDL cholesterol cholesterol and in diabetics when compared with controls. In their study, serum HDL - cholesterol levels did not differ significantly in the two groups. Jain (1980) observed increase in the levels of serum total lipids, total cholesterol, serum triglycerides and serum phospholipids in diabetic subjects as compared to normal controls.

The study of Peret et. al. (1974) observed means serum triglyceride levels higher in diabetics in comparison to control subject. Bijlani et. al. (1984) and Casteli et. al. found HDL-cholesterol to be significantly lower in diabetics as compared to normal.

In general is well established, nearly all confirm the presence of an inverse relationship between thyroxin serum levels and cholesterol^[4].

Indeed, several studies indicate a modest relationship between triglyceride increase and hypothyroidism; although even this modest significance has resulted in continuing investigations other studies have demonstrated thyroxin's regulatory action on fatty acid mobilization, through interaction with the catecholaminergic system, mainly through modulation of the biolytic action of epinephrine^[38].

Our results showing an association between thyroid dysfunction and hyperlipidemia agree with the results of previous studies In addition, among subjects with hypercholesterolemia, Subclinical hypothyroidism was more common^[9].

These results suggest that the effect of hypothyroidism in the Lipid metabolism is more marked in patients with higher serum TSH levels even mild elevations of TSH are associated with changes in lipid profile significant enough to raise the cardiovascular risk.

References:

- Adrees, M.; Gibney, J.; El-Saeity, N. and Boran, G. Effects of 18 months of L-T4 replacement in women with subclinical hypothyroidism. Clinic. Endocrine. 2009. Vol. 71. Pp: 298-303.
- 2- Kuiper, G. G.; Kester, M. H.; Peeters, R. T. and Visser, T. J. Biochemical mechanisms of thyroid hormone de iodination . Thyroid; 2005. Vol. 15 (8). Pp: 787-98.
- 3- Kandhro, G. A.; Kazi, T. G.; Sirajuddin, A.; Kazi, N.; Afridi, H. I.; Arain, M. B.; Baig, J. A.; Shah, A. Q. and Syed, N. Evaluation of the iodine concentration in serum and urine of hypothyroid males using an inexpensive and rapid method. Pak. J. Anal. Environ. Chem. 2009. Vol. 10 (1&2). Pp: 67-75.
- 4- Ali, M.; Nouh, Ibrahim A. M.; Eshnaf, and Mohamed A. Basher, Prevalence of Thyroid Dysfunction and Its Effect on Serum Lipid Profiles in a Murzok, Libya Population. Thyroid Science. 2008. Vol. 3 (10). Pp: 1-6.
- 5- Bech, K.; Damsbo, P.; Eldrup, E. Beck-Nielsen, H. Roder, M. E. and Hurtling, S. G. β-Cell function and glucose and lipid oxidation in Graves'

disease. Clinical Endocrinology. 1996. Vol. 4 (1). Pp: 59-66.

- 6- Ahren, B.; Lundquist, I.; Hedner, P.; Valdemarsson, S. and Schersten, B. Glucose tolerance and insulin and C peptide responses after various insulin secretary stimuli in hyper- and hypothyroid subjects before and after treatment. Diabetes Res. 1985. Vol. 2 (2). Pp: 95-103.
- 7- Cappola, A. R. and Ladenson, P. W.; Hypothyroidism and Atherosclerosis. Journal of Clinical Endocrinology& Metabolism.2003. Vol. 88 (6). Pp: 2438 -2444.
- 8- Ardekani, M. A.; Rashidi, M. and Shojaoddiny, A. Effect of Thyroid Dysfunction on Metabolic Response in Type 2 Diabetic Patients. Iranian Journal of diabetes and Obesity. 2010. Vol. 2 (1). Pp: 20-26.
- 9- Rizos, C.; Elisaf, M. S. and Liberopoulos, E. N. Effects of Thyroid Dysfunction on Lipid Profile. The Open Cardiovascular Medicine Journal. 2011. Vol. 5. Pp: 76-84.
- 10- Abdul Hamid Zargar, A. H.; Wandroo, F. A. and Wadhwa, M. B. Serum Lipid Profile in Non-insulin-dependent Diabetes Mellitus Associated with Obesity. INT. J. DIAB. DEV. COUNTRIES. 1995. Vol. 15 (9). Pp: 9-13.
- 11- Stein, E. A. Lipid lipoproteins and apolipopoteins. In: NWTietz. ed. Philadelphia.W B aunders. 1987. Pp: 448.
- 12- Lania, A.; Persani, L. and Beck-Peccoz, P. Central hypothyroidism. Pituitary J. 2008. Vol. 11 Pp: 181–186.
- 13- Tchong, L.; Veloski, C. and Siraj, E.S. Hypothyroidism: Management Across the Continuum. JCOM. 2009. Vol. 16 (5). Pp: 231-235.
- 14- Morganti, S.; Ceda, G. P.; Saccani, M.; Milli, B.; Ugolotti, D.; Prampolinin, R.; Maggio, M. and Ceresini, G. Thyroid disease in elderly: sex-related differences in

clinical expression. J. Endocrinolo. Invest. 2005. Vol. 28 (11). Pp: 101-4.

- 15- Niafar, M.; Najafipour, F. and Bahrami, A. Subclinical thyroid disorders in postmenopausal women of Iran. J. Clini. & Diagno . Resea. 2009. Vol. 3 (6). Pp: 1853 - 1858.
- 16- Zand, J.; Spreen, A. N. and Lavalle, J. B. Smart medicine for heal their living. 1999. Hypothyroidism part 2, Janet Zand. Pp: 354.
- 17- Robert, C. G. and Ladenson, P. W. Hypothyroidism. Lancet. 2004. Vol. 363 (9411). Pp: 793-803.
- 18- Sherwood, L. Human physiology: from cells to system, 7th ed. 2008. Pp: 692.
- 19- Baha, M. Estrogen replacement therapy may exacerbate hypothyroiddism. Natio. Engla. J. Medic. 2001. Vol. 344. Pp: 1743-1749.
- 20- Schindler, A. E. Thyroid functions and post menopause. Gynecol. Endocrinol. 2003. Vol. 17.Pp: 79-85.
- 21- Stepnick, S. A.; Smith, G. J. and Groff. J. L. Microminerals, Advanced nutrition and human metabolism. 4thed. 2005. chapter 12. Pp: 471.
- 22- Peppa, M.; Koliaki, C.; Nikolopoulos, P. and Raptis, S. A. Skeletal muscle insulin resistance in endocrine disease. J. Biomedici. & Biotechnolo. 2010. Pp: 1-13.
- 23- Prakash, A. and Kumar Lai, A. Serum lipids in hypothyroidism: Our experience. India. J. Clinic. Biochem. 2006. Vol. 21 (2). Pp: 153-155.
- 24- Raiszadeh, F.; Solati, M.; Etemadi, A. and Azizi, F. Serum paraoxonase activity before and after treatment of thyrotoxicosis. Clin. Endocrinol. (Oxf). 2004. Vol. 60 (1). Pp: 75-80.
- 25- Muller, M. J.; Acheson, K. J.; Jequier, E. and Burger, A. G. Thyroid hormone action on lipid metabolism in humans: a role for endogenous insulin metabolism. 1990. Vol. 39 (5). Pp: 480-485.
- 26- Laakso, M.; Voutilainen, F. and Sarlund, H. Serum lipids and

lipoproteins in middle-aged noninsulin dependent diabetics. Atherosclerosis. 1985. Vol. 56. Pp:271.

- 27- Elkeles, R. S.; Wu, J. and Hambley, J. Hemoglobin A, blood glucose and high-density lipoprotein-cholesterol in insulin-requiring diabetics. Lancet. 1987. Vol. 2. Pp: 547.
- 28- Colwell, J. A. Vascular thrombosis in Type 2 diabetes mellitus. Diabetes. 1993. Vol. 42. Pp: 8-11.
- 29- Gimenez-Palop, O.; Gimenez-Perez, G. and Mauricio, D. Circulating ghrelin in thyroid dysfunction is related to insulin resistance and not to hunger, food intake or anthropometric changes. Eur. J. Endocrinol. 2005. Vol. 153 (1). Pp: 73 -79.
- 30- Jenkins, R. C.; Valcavi, R. and Zini. M. Association of elevated insulin-like growth factor binding protein-1 with insulin resistance in hyperthyroidism. Clin. Endocrinol. (Oxf). Vol. 52 (2). Pp: 187 -195.
- 31- Taskinen, M. R. Hyperlipidemia in diabetes. Clin. Endocrinol. Metab. 1990. Vol. 4. Pp: 743.
- 32- Schaeffer, E.; Levy, R. I.; Anderson, D.W. Plasma triglycerides in regulation of HDL-cholesterol levels. Lancet. 1978. Vol. 2. Pp: 391.
- 33- Cohen, A. M. and Fidel, J. Diabetes, blood lipids, lipoproteins, change of environment. Met. 1979. Vol. 28. Pp: 7.
- 34- Jain, A. P. and Gupta, D. P. Study of blood, lipids diabetes without any manifest vascular complications. JDAI. 1980. 20.
- 35- Paret, A. D.; Rowes, A. and Shahmanesh, M. Blood lipids in treated diabetics. Diabetologia. 1974. Vol. 10. Pp: 115.
- 36- Bijlani, P. K. and Shah Kokila Raheja,B.S. HDL cholesterol in diabetics.JAPI. 1984. Vol. 32 .Pp: 34-37.
- 37- Castelli, W. P.; Doyle, J. T. and Gordon, T. HDL cholesterol and other lipids in coronary heart disease: The

cooperative lipoprotein phenotyping study. Circulation. 1977. Vol. 55. Pp: 767.

- 38- Bratton, R. L. Bratton's family medicine board review. 3rd ed. P: 100.
- 39- Rao, V. S.; Kadarinarasimhiah, N. B.; John, S.; Hebbagodi, S.; Shanker, J. and Kakkar, V. V. Usefulness of C-Reactive Protein as a Marker for Prediction of Future Coronary Events in the Asian Indian Population: Indian Atherosclerosis Research Study. Internet. J. Vascu. Medic. 2010. Vol. 10. Pp: 1-8.
- 40- Ramalho, R.; Guimaräes, C.; Gil, C.; Neves, C.; Guimaräes, J. T. and Delgado, L. Morbid obesity and inflammation: A prospective study after adjustable gastric banding surgery. J. Obes. Surg. 2009. Vol. 19 (7). Pp: 915–920.

- 41- Dar, M. S.; Pandith, A. A.; Sameer, A. S.; Sultan, M.; Yousuf, A. and Mudassar, s. A potential marker for hypertension in Kashmiri population. Indi. J. Clini. Biochem. 2010. Vol. 25 (2). Pp: 208-212.
- 42- Wheeler, M. J. & Hutchinson, J. M. Methods for the investigation of thyroid function, Hormone assays in biological fluid, chapter 5, Vol. 324, PP: 90-91.
- 43- Hayes, A.W. Hormone assays and endocrine function, principle and methods of toxicology, 4th ed. 2001. chapter 30, P. 1398.
- 44- Sadock, B. J. & Safdock, V. A. Thyroid hormones, Kaplan and Sadock's concise textbook of clinical psychiatry, 3rd ed., P. 553.
- 45- Springhouse, N. Endocrine care, visual nursing: a guide to diseases, skills, and treatment. 2007. Pp: 348.