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Correlation of Serum Lipid Profile and Hypothyroidism in Patients Attending Gujarat Adani Institute of Medical Science, Bhuj, Gujarat.

KEYWORDS: cholesterol, hypothyroidism, lipid profile, thyroid

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ABSTRACT In order to determine whether the screening of lipid profile is justified in patients with hypothyroidism we estimated serum lipids in cases having different levels of serum TSH. 59 patients of hypothyroidism in the age group of 18 to 62 yrs were studied for thyroid profile over a period of one year. On the basis of serum TSH level the cases were divided into three groups: In the first group TSH concentration was 8.5±2.99 µIU/ml, 95% confidence interval (CI), whereas serum total cholesterol and LDL-cholesterol levels were 192±37.32 and 129±29.17 mg/dl respectively. The statistical analysis of these two groups showed a significant correlation between raised TSH levels and serum total cholesterol and LDL-cholesterol ($P < 0.05$ & $P<0.01$) respectively. We conclude that hypothyrodism is associated with changes in lipid profile.

INTRODUCTION:
Hypothyroidism is defined as a deficiency of thyroid activity, which results from reduced secretion of both T3 and T4 irrespective of the cause (1). Hypothyroidism causes a derangement of many parameters of our body which contributes to the development of atherosclerotic cardiovascular disease. These include alterations in lipid profile, hemodynamic changes, endothelial dysfunction, coagulation disturbances, metabolic and hormonal changes, and changes in homocysteine and C-reactive protein levels.

Thyroid hormones have significant effects on synthesis, mobilization and metabolism of lipids. Overt hypothyroidism is associated with significant increase in circulating concentrations of total LDL-Cholesterol leading to coronary artery disease.

Hypercholesterolemia is favored due to the hormone deficit and to the decreased activity of lipoprotein lipase (2, 3)

There is a known pathogenic relationship of patients with hypothyroidism developing atherosclerotic cardiovascular disease. This risk of atherosclerotic cardiovascular disease is attributable to dyslipidemia noticed in hypothyroid states (i.e., increased circulating levels of LDL-Cholesterol and lipoprotein. The suggested mechanism for elevated levels of LDL-Cholesterol is due to decrease in its catabolism caused by hypothyroidism (4)

HDL cholesterol level was found reduced in some other studies on hypothyroid patients. Decreased thyroid secretion greatly increases the plasma concentration of triglycerides. Nikkilia& Kekki11 have stated that hypertriglyceridemia in hypothyroidism is due to decreased activity of lipoprotein lipase (LPL), which results in decreased clearance of triglyceride-rich lipoproteins.(5)

The present study aims to assess the association of hypothyroidism with lipid abnormalities.

MATERIALS AND METHODS:
This study was conducted at Gujarat Adani Institute of Medical Science, Bhuj, Gujarat. 59 patients clinically diagnosed as hypothyroidism, in the age group of 18 to 62 yrs. were studied, over a period of one year. Patients with chronic renal failure,diabetes mellitus, liver diseases, chronic diseases, pregnancy and age less than 20 and more than 60 years were excluded.Patients with TSH level above 6 µIU/ml (since the upper limit of normal range given by kit manufacture is 6µIU/ml) were considered to be having hypothyroidism. As per serum TSH level the cases were divided into three groups:

Group I with levels of 6-20 µIU/ml,
Group II with levels of 21-40 µIU/ml
Group III with levels above 40 µIU/ml

Patients were selected in such a way that the groups were comparable in size and age distribution. Apart from these criteria, the selection was random. 12 hours of overnight fastingblood sample was collected from antecubital vein. Serum was separated and assays were performed within 24 hrs. Serum T3 & T4 were measured by micro plate competitive enzyme Immunoassay & TSH measured by micro plate immunoenzymeteric assay (Monobind, Costa Mesa, USA). (6, 7)

The concentration of serum total cholesterol, HDL cholesterol and triglycerides were measured by a timed-end point method (using Synchron CX5 auto analyzer) and LDL cholesterol was calculated by Friedwald'sformula. (8)

Data collected was subjected to standard statistical analysis, such as confidence interval (CI), correlation (r) and t –test.

Table 1:Levels of T3, T4, TSH and Lipid profile in different groups

PARAMETERS	NORMAL RANGE	GROUP 1 (n=15)	GROUP 2 (n=15)	GROUP 3 (n=15)
T3 (ng/ml) 95% Confidence Interval	0.8 –1.9	0.7±0.50 (0.12)	0.7±0.35 (0.19)	0.6±0.52 (0.26)
T4 (ng/ml) 95% Confidence Interval	5.0-13.0	5.3±1.78 (0.67)	4.9±2.16 (1.19)	2.0±1.84 (0.93)
TSH (ng/ml) 95% Confidence Interval	0.3-6.0	8.5±2.99 (1.07)	34±3.85 (1.94)	67±10.82 (5.47)

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Total cholesterol (mg/dl) 95% Confidence Interval	130-220	192±37.32 (13.31)	201±35.40 (18.42)	301±70.4 (35.65)
Triglycerides (mg/dl) 95% Confidence Interval	35 –160	146±67.93 (24.30)	145±68.52 (35.18)	223±63.91 (32.34)
HDL (mg/dl) 95% Confidence Interval	35-55	39±7.48 (2.67)	37±7.20 (3.69)	42±10.64 (5.38)
LDL Cholesterol (mg/dl) 95% Confidence Interval	up to 150	129±29.17	134±36.33 (18.89)	214±61.64 (31.19)

RESULT and DISCUSSION:

In group-I subject the mean value of T3, T4 and TSH was found to be 0.7±0.50 ng/ml, 5.3 ±1.78 µg/dl and 8.5 ± 2.99 µIU/ml respectively. The mean levels of total serum cholesterol, HDL-chol, LDL-chol andtriglycerides were found to be 192±37.32, 39±7.48, 129 ± 29.17 and 146 ± 67.93 mg/dl respectively.

In group-II patients the mean T3, T4 and TSH levels were 0.7±0.35 ng/ml, 4.9±2.16 µg/dl and 34±3.85 µIU/ml respectively. The mean value of total cholesterol, HDL-Chol,

LDL-Chol and triglycerides were found to be 201±35.40, 37±7.20, and 134±36.33 mg/dl respectively. The mean values of T3, T4 and TSH (in group III) were found to be 0.6±0.52 ng/ ml, 2.0 ± 1.84 µg/dl and 67.57±10.82 µIU/ml respectively. The mean levels of total serum cholesterol, HDL-chol, LDL-chol and Triglycerides were found to be 301±70.46, 42±10.64, 214±61.44 and 223±63.91 mg/dl respectively.

In hypothyroid patients, despite the reduced activity of HMG CoA reductase, there is often an increase in the serum total cholesterol concentration, mainly due to raised levels of serum LDL cholesterol and intermediate density lipoprotein (IDL) cholesterol(9). Decreased thyroid secretion greatly increases the plasma concentration of cholesterol because of decreased rate of cholesterol secretion in the bile and consequent diminished loss in the feces due to decreased number of low density lipoprotein receptors on liver cells (10). Decreased activity of LDL receptors resulting in decreased receptor-mediated catabolism of LDL and IDL is the main cause of the hypercholesterolemia observed in Hypothyroidism (11).

Hypothyroid patients may also exhibit elevated levels of HDL-C mainly due to increased concentration of HDL_2 particles. Indeed, due to a reduction of HL activity a decrease in HDL_2 catabolism is observed. Moreover, decreased activity of the CETP results in reduced transfer of cholesteryl esters from HDL to VLDL, thus increasing HDL-C levels (12). In some studies we find confronting results regarding high density lipoprotein cholesterol levels in hypothyroidism. In thyroidectomizedrats there was 25.9% decrease in HDL-C level, suggesting a defect in HDL metabolism10. HDL cholesterol level was found reduced in some other studies on hypothyroid patients.(5)

Decreased thyroid secretion greatly increases the plasmaconcentration of triglycerides6. Nikkilia& Kekki11 have stated that hypertriglyceridemia in hypothyroidism is due to decreased activity of lipoprotein lipase (LPL), which results in decreased clearance of triglyceride-rich lipoproteins.(5)

CONCLUSION

Hypothyroidism is one of the most common causes of secondary dyslipidemia. Therefore, before starting hypolipidemic therapy, the evaluation of thyroid function is needed.

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