

Association Between Subclinical Hypothyroidism & Lipid Profile : A Pilot Study



Medical Science

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ABSTRACT

INTRODUCTION

Overt Hypothyroidism and lipid profile has shown to be positively correlated in many studies but this study is conducted to see whether subclinical cases of Hypothyroidism has any association with lipid profile or not and whether these subclinical cases have the same cardiovascular risk as seen in overt hypothyroid cases.

MATERIAL AND METHODS

The present is carried out in 30 Subclinical hypothyroid subjects and 30 normal healthy control subjects. Thyroid profile was assayed using ELISA and Lipid Profile by using Enzymatic assays.

RESULTS

Subclinical cases show positive correlation with lipid levels.

CONCLUSION

Subclinical hypothyroid patients shows the same positive association as seen in many studies of overt Hypothyroidism.

Introduction

The thyroid hormone plays a role in the regulation of the synthesis and in the metabolism and the mobilization of lipids. Dyslipidemia in subclinical hypothyroidism (SH) had always remain controversial. The present study is aimed to assess the association of SH with lipid abnormalities. Subclinical hypothyroidism (SH) is defined as the clinical status of elevated serum thyrotropin (TSH) levels, with normal levels of thyroxine (T4) and triiodothyronine (T3). It is a more common disorder than overt hypothyroidism with a prevalence of 1.4–7.8% in older populations and even greater percentage among women^{1,2}. Lipid abnormalities in patients with subclinical hypothyroidism are not consistent³. Many studies have shown significant increase in Total cholesterol, and LDLC and Triglyceride in patients with SH^{4,6}. By affecting the metabolism of the lipids, hypothyroidism stimulates the process of atherogenesis and thereby increasing the cardiovascular disease risk.

Patient & Methods:

This case control study was conducted in a tertiary care hospital of UP RIMS & R, Saifai, Etawah (U.P.) India. Thirty subclinical hypothyroid patients were taken as study group and thirty age & sex matched controls were taken. Inclusion criteria were TSH between 4.2-10 μ IU/mL, normal T4 and normal T3 levels. Healthy people with normal thyroid functions were recruited as controls. Exclusion criteria were smokers and alcoholics, diabetes mellitus, renal insufficiency, diagnosed cases of hypothyroidism or those already on treatment, polyglandular disorders, thyroid cancer. A detailed history was obtained from all patients and controls. After an informed consent, all patients and controls were subjected to complete physical examination and following investigations were done : Lipid profile like TC, TG, HDL, VLDL and LDL and thyroid function tests including serum TSH, T4 and T3. Samples for all these investigations were taken 12 hours after an overnight fasting. To estimate lipid parameters and thyroid hormones, serum was separated from the blood by centrifugation at 3000 rpm, and stored at -70°C until analysis. The estimation of the T3, T4 & TSH was done by using ELISA method. The serum lipid profile was estimated by the enzymatic CHOD-PAP method for Total Cholesterol, by the GPO method for Triglyceride and by the PVS/PEGME method for HDL cholesterol. These estimations were carried out by using ERBA-XL 300 (Transasia) Fully automated analyzer. LDL Cholesterol and VLDL Cholesterol were calculated by Friedwald's formula. All the

kits used were commercially prepared, Calbiotech kit was used for Thyroid profile estimation while System packs kits were used for Lipid profile estimation. Calibration was performed using Randox quality control sera. The quality control was established using Erbapath and Erbanorm.

Statistical Analysis:

All the continuous variables were expressed as mean \pm SD. All the results were discussed at 5% level of significance; P value < 0.05 was considered significant. Statistical Package for Social Sciences version 21.0 was used for statistical analysis.

Result:

Mean serum Total cholesterol, Triglyceride, LDL & VLDL were higher in Subclinical Hypothyroid patients as compared to Controls. Mean serum TC in Subclinical Hypothyroid patients was (199.3 \pm 43.9) mg/dL V/S (147.2 \pm 35) mg/dL in euthyroid controls (P value <0.05). Mean serum TG in Subclinical Hypothyroid subjects was (133.5 \pm 41.2) mg/dL V/S (101.4 \pm 31.9) mg/dL in Controls (P value <0.05). Mean serum LDL in Subclinical Hypothyroid patients was (114.7 \pm 46.4) mg/dL V/S (67.3 \pm 32.4) mg/dL in euthyroid controls (P value <0.05). Mean serum VLDL in Subclinical Hypothyroid was (26.7 \pm 8.2) mg/dL V/S (20.5 \pm 6.7) mg/dL in controls. There was no statistical significance measured in HDL levels on comparing the subclinical hypothyroid patients with the control group.

Table I (Comparison between lipid profile in Subclinical Hypothyroidism with controls)

Parameters	Subclinical cases	Controls	t value	p value
T3	1.5 \pm 0.6	1.4 \pm 0.6	0.530	NS
T4	8.7 \pm 1.9	8.8 \pm 1.3	-0.264	NS
TSH	7.4 \pm 1.4	1.5 \pm 1.1	15.962	<0.05
TC	199.3 \pm 43.9	147.2 \pm 35	4.729	<0.05
TG	133.5 \pm 41.2	101.4 \pm 31.9	3.139	<0.05
HDL	57.9 \pm 16.1	59.4 \pm 10.4	-0.404	NS
LDL	114.7 \pm 46.4	67.3 \pm 32.4	4.269	<0.05
VLDL	26.7 \pm 8.2	20.5 \pm 6.7	2.959	<0.05

Discussion:

Thyroid hormones affect the metabolism of lipids⁷. Several studies have assessed these effects in patients with hypo-

thyroidism. These studies have reported increased levels of total cholesterol & LDL-cholesterol^{8,9}. Our study showed significant dyslipidaemic changes in subclinical hypothyroid patients as compared to euthyroid controls. From our study we saw that the levels of Total cholesterol, Triglyceride, LDL & VLDL were all higher & statistically significant as compared to control groups, but we didn't find any correlation between levels of HDL in both cases & controls. The elevation in total cholesterol and LDL-C in hypothyroidism is accounted for by the effect of thyroid hormone on lipoprotein-lipase activity¹⁰ and the expression of the LDL receptor¹¹, and these changes probably play an important role in atherogenesis in untreated hypothyroidism. Triglycerides and LDL-C levels were significantly high in subclinical hypothyroid patients when compared with the controls in our study. In a substantial number of studies, total cholesterol and/or LDL cholesterol seem to be elevated in subclinical hypothyroidism compared with controls^{12,13}. In this respect, in our case control study, subjects with subclinical hypothyroidism had significantly higher levels of TC, TG, LDL & VLDL, thus displaying a atherogenic lipid profile when compared with healthy individuals. In another study of dyslipidemia in an Indian population of 100 patients with SH and 52 euthyroid controls above the age of 20 years, total cholesterol, triglyceride and LDL in the age group of 40-50 years were significantly elevated in SH¹⁴.

CONCLUSION:

analysis suggested that serum TC, LDL-C, TG and VLDL levels were increased in Subclinical Hypothyroidism however, no evidence suggested that subclinical hypothyroidism was associated with serum HDL-C levels. Higher serum TC, LDL-C and TG levels increases the risk of coronary heart disease, therefore cardiovascular status of Subclinical Hypothyroid patients should be monitored carefully. This study highlights the significant positive correlation between serum TSH and lipid profile. Therefore hypothyroidism could be one of the causes of secondary hyperlipidaemia and should be viewed as an independent risk factor for atherosclerosis along with obesity, hypertension, diabetes etc. An atherogenic lipid profile is the important forerunner of cardiovascular and cerebrovascular diseases, the major killers in today's modern world.

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