(International Print/Online Journal)

SJIF IMPACT FACTOR: 5.565
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume3, Issue 6, Page No: 548-551
November-December 2020

Effect of L- thyroxine on levels of C- reactive protein in patients with hypothyroidism

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Type of Publication: Original Research Paper
Conflicts of Interest: Nil

ABSTRACT

Background: The present study was aimed to study levels of C- Reactive protein before and after treatment with L- thyroxin in hypothyroid cases.

Methods: 46 hypothyroid cases and 46 age and sex matched euthyroid control were included for studies Serum T3, T4, TSH were estimated to diagnose hypothyroidism. Levels of CRP was estimated before and 2 months after treatment with L- thyroxine.

Results: Significant decrease in the levels of T3 and T4 were found in hypothyroid cases as compared to controls. Serum TSH and CRP were significantly increased in hypothyroid cases. Two months after treatment with L- Thyroxine, significant decreases levels of CRP were observed.

Conclusion: Treatment with L- thyroxine promotes antiatherogenic activity. Prompt treatment of patients with hypothyroidism will reduce the risk of cardiovascular events in such patients.

Keywords: L-thyroxine , C-Reactive Protein , Hypothyroidism

INTRODUCTION

Thyroid hormones play an important role in metabolic homeostasis in adults. Coronary artery disease is twice common in patients with hypothyroidism an compared with age and sex matched controls [1,2]. Many studies have found that patients with subclinical and overt hypothyroidism have higher serum total cholesterol, LDL cholesterol and triglycerides[3,4]. C- reactive protein (CRP), an acute phase protein has evolved as a strong independent risk factor for cardiovascular disease in recent years. Patients with hypothyroidism have higher levels of CRP as compared to euthyroid persons. This dyslipidemia and inflammation may explain the association between hypothyroidism and atherosclerosis.

This study is to evaluate levels of CRP before and two months after L- thyroxine therapy in patients with hypothyroidism.

MATERIALS AND METHODS:

This study was conducted in the Department of Biochemistry and Medicine, Pt. B.D.S. PGIMS, Rohtak. Approval from institutional Ethical Committee was taken.

Age and sex matched 46 hypothyroid cases and 46 euthyroid controls were taken for this study. Fasting serum samples were subjected to estimation of T3 (by Radioimmunoassay) [5,6],T4 (by Radioimmunoassay) [7,8] and TSH (by immunoradiometric assay)[9,10].CRP was measured by turbidimetric method[11,12]. Pregnant women, patients on thyroxine, oral contraceptives, statins, history of

rheumatoid arthritis, coronary artery disease, any other acute or chronic inflammatory conditions, patients undergone thyroidectomy were excluded from this study.

RESULTS:

Table 1 shows a significantly ($p < 0.001$) decreased levels of T3 in the hypothyroid patients (53.8 ± 34.1) as compared to the healthy controls (129.3 ± 30.9). T4 levels were also significantly ($p < 0.001$) decreased in the hypothyroid patients (2.0 ± 1.5) as compared to the healthy controls (7.9 ± 1.9). There were also a significant ($p < 0.001$) increases levels of TSH in the hypothyroid patients (89.5 ± 24.5*) as compared to the healthy individuals (2.5 ± 1.0). The CRP levels were also significant ($p < 0.001$) increased in the hypothyroid patients (28.38 ± 11.9*) as compared to the healthy individuals (3.72 ± 1.04).

Table 1: Comparison of blood investigations in healthy controls and hypothyroid patients

Parameter	Control (Mean ± S.D)	Hypothyroid cases (Mean ± S.D)	P value
T3	12.9 ± 30.9	53.08 ± 34.1	< 0.001
T4	7.9 ± 1.9	2.0 ± 1.5	< 0.001
TSH	2.5 ± 1.0	89.5 ± 24.5	< 0.001
CRP	3.72 ± 1.04	28.38 ± 11.9	< 0.001

Table 2 shows a significantly ($p < 0.001$) increased CRP levels in the hypothyroid patients (28.38 ± 11.9* gm%) as compared to the healthy controls (3.72 ± 1.04 gm %) Two months after treatment the levels of CRP significantly ($p < 0.001$) decreased from 28.38 ± 11.9 to 4.57 ± 1.13 .

Table 2: Comparison of CRP levels in hypothyroid patients before and after treatment.

Parameter	Hypothyroid patients (Mean ± S.D)	Control (Mean ± S. D)
CRP before treatment	28.38 ± 11.9	3.72 ± 1.04
CRP after treatment	4.57 ± 1.13	3.72 ± 1.04
P value	< 0.001	

DISCUSSION:

We found significant increases in serum CRP levels in hypothyroid patients (28.38 ± 11.9) as compared to euthyroid control (3.72 ± 1.04). Similar findings were found by Crain et al and tuzu el at [13,14]. The increased levels of CRP in hypothyroid patients suggest an inflammatory condition that predisposes hypothyroid patients to premature atherosclerosis and future cardiovascular diseases.

Overt hypothyroidism increases low-density lipoprotein (LDL) cholesterol levels, induce diastolic hypertension, alters coagulability, and negatively affects vascular smooth muscle function. Treatment of overt hypothyroidism can moderate these negative effects, but the effect of treatment is less clear in patients with subclinical hypothyroidism. Reductions in total cholesterol levels are highest in patients who had the highestpretreatment thyroid- stimulating hormone (TSH) and lipid levels. Hypothyroidism treatment also can reduce hypertension and improve vascular smooth muscle function. Treatment of subclinical hypothyroidism may lower cholesterol levels in patients with hypercholesterolemia.

Cappola and Ladenson reviewed the most recent information abouthypothyroidism and cardiovascular risk factors.Patients with hypothyroidism appear to have elevated homocysteine and C- reactive protein (CRP) levels and altered flow- mediated, endothelium- dependent vasodilatation ; coagulation capacity also may be affected .Thyroxine (T_4) therapy can reduce homcysteine and CRP levels significantly in persons with overt hypothyroidism, but it may not be as useful in patients with subclinical disease. The authors conclude that overt hypothyroidism can cause atherosclerosis and other cardiac risk factors. Careful management of hypothyroidism can improve morbidity[15].

Our findings are contradictory to the study done by Lee WY, where serum CRP and Lp (a) levels, risk factors for atherosclerosis, were not found to be significantly affected by the degree of thyroid dysfunction. Increased risk of atherosclerosis in

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hypothyroidism does not appear to be associated with non- traditional cardiovascular risk factors, such as serum CRP , Lp (a) or apo A l levels[16].

Singh et al showed that both SCH and OH patients have elevated atherogenic and oxidative stress markers. Increased CRP and homocysteine might be a key molecule linking inflammation to oxidative stress in atherosclerosis.Thyroid hormones are the most important factors involved in the regulation of the basal metabolic condition. as well as in the oxidative metabolism[17].

Two months after treatment with L- thyroxine, the levels of CRP decreased significantly from 28.38 ± 11.9 to 4.57 ± 1.13 (table 2).Similar findings were found by Orhan Kursat Poyrozoglu et al [18]which showed that treatment with L- thyroxine can attenuate the cardiovascular risk in hypothyroid patients.

Haralampos Milinois et al found that L- thyroxine increased levels of HDL – C associated platelet activating factor- acetyl hydrolase (HDL- C/ PAF-AH) activity in patients with subclinical hypothyroidism. This explains the antiatherogenic effect of thyroid replacement therapy[19].

Raffale Marfella and colleagues studied the effect of L- thyroxine on innate immunity activity in plaques of patients with untreated and L- thyroxine treated subclinical hypothyroidism. They found that L-thyroxine inhibits innate immunity dependentrupture of plaque and thus promote plaque stabilization[20].

Apart from relationship between hypothyroidism and atherosclerosis, there exists multiple direct and indirect actions of thyroid hormone on cardiac and peripheral vascular function that can complicate management of hypothyroid patients with atherosclerotic coronary artery disease.

CONCLUSION:

Treatment with L- thyroxine promotes antiatherogenic activity. Prompt treatment of hypothyroid patients with L- thyroxine will reduce the risk of cardiovascular events in such patients.

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