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SUBCLINICAL HYPOTHYROIDISM AND CORONARY HEART DISEASE

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ABSTRACT Cardiovascular disease is the major cause of death all over the world. Cardiac patients have a high TSH, dyslipidemia and low vitD. While treating cardiac patients thyroid dysfunction is to be taken into consideration especially in old people .Total cholesterol, triglycerides, HDL-c, LDL-c and VLDL are evaluated compared to controls. Hence in population screening TSH, lipid profile and vit-D should be measured to assess cardiac risk.

KEYWORDS : Subclinical hypothyroidism, Cardiovasular disease.

INTRODUCTION

The relationship of thyroid hormonal abnormalities and cardiovascular disease goes well beyond the risk of atherosclerosis in association with hypothyroidism and the risk of atrial fibrillation in individuals with hyperthyroidism. [1]. The two organ systems are intimately linked by their embryological anlage, and the ubiquitous effects of thyroid hormone on the major components of the entire circulatory system: the heart, the blood vessels, and the blood[2]. Cardiac output is normally modulated by peripheral arteriolar vasoconstriction and dilatation, venous capacitance, and blood volume in response to tissue metabolic risks [3]. The major effects of thyroid hormones on the heart are mediated by Triiodothyronine (T3). T3 generally increases the fate and speed of systolic contraction and the speed of diastolic relaxation [4]. In addition, T3 decreases vascular resistance, including coronary vascular tone, and increases coronary arteriolar angiogenesis risk[4]. These multiple thyroid hormone effects are largely mediated by the action of nuclear based thyroid hormone receptors [5-7].

Hyperthyroidism is characterized biochemically by a low TSH level and elevated T4, T3, or both. Patients with hyperthyroidism can develop a life-threatening complication called thyroid crisis, requiring urgent therapy with beta-blockers, anti thyroid medication, and iodine. This complication can be precipitated by an acute illness such as a MI, infection, or other stress [8] in patients with underlying coronary disease or heart failure [9].

MATERIALS AND METHODS.

Blood was collected using disposable syringes and needles .Serum separated by centrifugation at 3500 rpm for 10 minutes using Kemi centrifuge.

Total cholesterol ,Triglycerides ,LDL-c and VLDL –c were measured using flex in Siemens Automated Chemistry Analyser supplied by Remedex. HDL-c measured using direct method .Total T_3,Total T_4 and TSH measured using Chemiluminescent Microparticle Immuno Assay (CMIA) using i1000SR instrument of Abbott USA.

RESULT

In the present study 50 cardiac patients had subclinical hypothyroidism with TSHas follows:-

Sl No	TSH(μIU/ml)	Patient No.
1.	6-10	14
2.	10-14	12
3.	15-20	10
4.	21-25	2
5.	26-40	7
6.	41-60	1
7.	61-80	1
8.	>100	3

All had cardiac risk and low vitD.

DISCUSSION

Cardiovascular diseases are the major cause of death worldwide and it has significant health related costs. A number of CVD risk factors can be modified thereby decreasing the CVD risk[10].

Subclinical hypothyroidism (SHT), a mild thyroid dysfunction, has been reported increasingly all over the world. The Wickham Survey and the Colorado study have shown prevalence of Subclinical hypothyroidism in 7.5% males and 3.1% females in general population [11]. SHT has clinical importance because of its high prevalence (4–20%), the risk of progression to overt hypothyroidism, and consequences associated with cardiac and lipid abnormalities [12]. A number of studies have reported that subclinical hypothyroidism is associated with an increased risk of coronary heart disease and there appears to be a significant increase in a cluster of metabolic CVD risk factors among people with SHT [13,14].

Similar to previous studies, the authors found preponderance of risk factors for CVD in SHT as compared to control. We observed significantly higher level of diastolic blood pressure, total cholesterol, TG, LDL- c, TC/HDL-c, LDL-c/HDL-c, and hs-CRP in cardiac patients as compared to control. High diastolic BP observed is consistent with that of study conducted in Israel, where diastolic blood pressure was 82 versus 75 mmHg in SH versus control women [15].

Study by Sharma et al. also demonstrated that patients with subclinical hypothyroidism had significantly higher levels of serum hs-CRP, Lp (α), total cholesterol, and LDL-C when compared to controls [16].

Patients with subclinical hypothyroidism had substantially increased risk of developing hypercholesterolemia and diastolic hypertension and having low HDL, undesirable LDL-C, and high hs-CRP. The alterations could lead to SHT. Patient has subclinical hypothyroid with TSH >10 mIU/L had increase risk of CVD. Diastolic BP, atherogenic lipid profile, and low grade inflammation might increase risk of developing CVD in SHT.

By understanding pertinent cardiovascular physiology and

pathophysiology, physicians will have a firmer basis for making the often-complicated recommendations for patient care even when evidence-based studies are not yet available. Routine TSH measurement merits implementation, especially in pregnant women, old aged women > 60 years and anyone whose risk of thyroid dysfunction is high.

Present studies have shown patient with SHT have high TSH and high risk for MI. This study also have shown cardiac patients have low vit D, atherosclerosis, Lipid profile low BP. Both put together, patient with SHT have severe cardiac risk factors and all of these effects have high risk for CVD. Hence whie managing SHT patient screening for TSH and thyroid supplementation could be worth wile.

CONCLUSIONS

Cardiac patients have a high risk of developing subclinical hypothyroidism. Cardiac patients have low levels of vit D and atherogenic lipid profile .Cardiac patients with subclinical hypothyroidism have added cardiac risk of MI. Hence cardiac patients with subclinical hypothyroidism have high TSH hyperlipidemia and low vitD all of which contribute to progressive development of cardiac dysfunction causes ischemia and myocardial infarction.

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