Scope International Journal of Science, Humanities, Management and Technology. ISSN : 2455-068X

Vol.3 Issue 4 (2017) 189 - 193. Submitted 25/11/2017. Published 30/12/2017

A STUDY TO ASSESS THE RELATION BETWEEN SEVERITY OF HYPOTHYROIDISM

S.Kalaivani, Assistant Professor, Department of Biochemistry, Shri Sakthi Kailassh Women's College, Salem.

Abstract:

Hypothyroidism is the most common pathological hormone deficiency (Roberts et al, 2004). The variety of end-organ effects and wide range of disease severity—from entirely asymptomatic individuals to patients in coma with multisystem failure—can make hypothyroidism an elusive clinical entity (Roberts et al, 2004). Hypothyroidism was first described in the 19 th century. Impaired memory, slowed mental processing, depression, nerve entrapment syndromes, ataxia, muscle weakness and muscle cramps are the most common neurological symptoms which may be caused by hypothyroidism (Roberts et al, 2004). Disorders of the thyroid gland are among the most common endocrine maladies. Hypothyroidism is the most prevalent form of thyroid disease and symptoms may include memory and learning impairment, depression, psychotic behaviour, retarded locomotor ability, somnolence, progressive intellectual deterioration.

Keywords: Behaviour, 19th century, Hypothyrodism.

1. INTRODUCTION

Disruption of thyroid hormone production during fetal and early neonatal development leads to a suite of permanent deficits in intelligence and sensorimotor function in humans. Clinical hypothyroidism indicates a pervasive deficit in thyroid hormone actions, including modulation of calorigenesis and oxygen consumption in most tissues and additional organ- specific effects. The action of thyroid hormones (THs) in the brain is strictly regulated, since these hormones play a crucial role in the development and physiological functioning of the central nervous system (CNS). Transient reductions in thyroid hormone during critical periods of brain development can have devastating and irreversible effects on neurological function (Gilbert et al, 2004). The hippocampus is a brain region sensitive to thyroid hormones and is a necessary substrate for some forms of learning and memory. Subregions within the hippocampus display distinct ontogenetic profiles and have shown differential vulnerability to some indices of thyrotoxic insult. Synaptic function can be readily assessed in the hippocampus, yet little information exists on the consequences of early thyroid hormone insufficiency on the neurophysiological integrity of this structure. Thyroid hormone is essential for proper development of the mammalian CNS. Previous studies have documented a decrease in the ability of neonatal hypothyroid animals to learn and to habituate to maze tests and an increase in spontaneous activity (Darbra et al, 2003). On the other hand some observers stated that children with hypothyroidism had no apparent specific impediments to learning unrelated to intelligence Cognitive neurological symptoms are common in myxoedema, in particular a general slowing of cognitive functions with memory impairment and apathy. This may progress insidiously to a global cognitive impairment of dementia. Appropriate treatment with replacement therapy will sometimes improve mental changes,though they are frequently only partially reversible. Prompt treatment of maternal hypothyroidism, identified by increased TSH, is being advocated to mitigate a negative effect on the woman and her child.

However, even a moderate transient period of maternal hypothyroxinemia at the beginning of rat neurogenesis disrupts neuronal migration into cortical layers. These findings reinforce the epidemiological evidence that early maternal hypothyroxinemia—when neuronal migratory waves are starting—is potentially damaging for the child. Detection of an inappropiate first trimester FT4 surge that may not result in increased TSH, may be crucial for the prevention of learning disabilities in a significant number of unborn children.

2. RELATED WORK

The relationship between the thyroid axis and psychiatric symptoms and disease is well established. In particular, clinical hypothyroidism leads to depressive symptoms which resolve with replacement therapy. The relationship between alterations of thyroid function and primary major depression is more complex. While various abnormalities of the thyroid axis have been identified, none is specific for depression and there is no clear evidence that thyroid hypofunction is a significant etiological factor in major depression. The literature on thyroid hormone treatment of depression, particularly treatment-resistant major depression, is highly promising and warrants further investigation. Greater understanding about thyroid–brain relationships may yield important information about the etiology of major mood disorders. Nevertheless, these abnormalities usually returned to normal after thyroxine replacement if the duration of hypothyroidism was less than 5 months. Regarding PNCS, all groups of thyroidectomized rats showed normal conduction before and after thyroxine therapy.

The study was conducted in the department of Biochemistry, tertiary care teaching hospital in North Karnataka, India. Ethical clearance was obtained from the Institutional ethics committee. Informed consent obtained from all the participants. Study was conducted over a period of one year from March 2014 to February 2015. Among the patients who underwent thyroid profile evaluation referred from different departments, 120 participants were selected of age group between 20 to 60 years. They were divided into four groups depending on TSH levels to assess the severity of hypothyroidism. Each group composed of 30 subjects. Group-I: Patients with euthyroid state i.e TSH levels between 0.4-4.5 □IU/mL taken as controls, Group-II: TSH levels of 5-20 □IU/mL, Group-III: TSH levels of 21-50 □IU/mL and Group-IV: TSH levels of >50 □IU/mL. Patients with renal disease, hepatic disease, diabetes mellitus, myocardial infarction, patients on statins and women on oral contraceptive pills were excluded from the study.

This study indicated that, in ratsthe peripheral nervous system seemed to be more resistant to hypothyroidism than the central nervous system, or (2) the pathogenesis of central and peripheral nerve dysfunction in hypothyroid rats might occur through different mechanisms After thyroxine replacement, the central conduction dysfunction usually returned to normal if the hypothyroid state was not more than 5 months in duration. However, when hypothyroid state persisted over 7 months or more, there would be an incomplete recovery for central conduction disorder. This study brought out the concept of ‘therapeutic window’ in reversing the central nervous dysfunction caused by hypothyroidism in adult rats. Processes under the control of TH (thyroid hormones) range from learning and anxiety-like behaviour to sensory function. At the cellular level,TH controls events as diverse as axonal outgrowth, hippocampal synaptic activity and the patterning of opsin photopigments necessary for colour vision. Overall, TH coordinates this variety of events in both central and sensory systems to promote the function of the nervous system as a complete entity.

3. METHODOLOGY

Muscle involvement in a variety of forms is a common complication of adult-onset hypothyroidism. Hypothyroid myopathy spans a clinical spectrum that includes a number of different manifestations. The reported prevalence of hypothyroid myopathy symptoms and signs is variable. In a prospective cohort study, 79% of adult patients with hypothyroidism had muscle complaints (myalgias, cramps or weakness) . Asymptomatic CK elevation has been reported to occur in 37% to 60% of hypothyroid patients .

Parameters	Group-I	Group-II	Group-III	Group-IV	ANOVA F	p-value
T.C mg/dL	162.49±31.17	213.56±45.9	233.97±41.68	297.06±38.11	59.91	0.0001
LDL-C mg/dL	100.9±12.43	141.23±28.36	158.16±28.55	202.5±52.57	43.79	0.0001
TGS mg/dL	121.43±10.93	168.83±35.31	183.9±29.07	261.33±45.99	93.59	0.0001
HDL-C mg/dL	37.31±4.47	38.07±5.34	38.53±5.06	42.3±7.7	4.04	0.009
VLDL mg/dL	24.28±2.18	33.76±7.06	36.78±5.81	52.26±9.1	93.59	0.001
AI	0.52±0.34	0.64±0.11	0.68±0.01	0.81±0.25	9.01	0.0001
TC/HDL CRI-1	4.41±0.97	5.59±1.26	6.01±0.85	7.31±1.76	27.0	0.0001
LDL/HDL CRI-2	2.74±0.43	3.71±0.83	4.11±0.93	4.9±1.36	26.9	0.0001

Table.1.Profile

Other significant manifestations associated with hypothyroidism include: myalgias, severe muscle weakness, polymyositis-like syndrome, rhabdomyolysis, and acute compartment syndrome (ACS). Rhabdomyolysis after withdrawal of thyroid hormone in a patient with papillary thyroid cancer has been described previously. risk of coronary heart disease and other forms of atherosclerotic vascular disease increases with rising plasma cholesterol concentration. Early diagnosis and proper management can significantly reduce the mortality and morbidity. The increase in lipid levels can be reversed by thyroid hormone supplementation14. Development of atherosclerosis in cholesterol fed animals is enhanced by the presence of hypothyroidism and reduced when thyroid hormones is administered15. Thus the present study was conducted with the objective of assessing the severity of dyslipidemia and to evaluate the role and significance of lipid ratios like Atherogenic index, Castelli's risk index-1 and Castelli's risk index-2 in early identification of individuals at risk for coronary artery disease (CAD) in hypothyroid patients beyond the routinely done lipid profile. Calculating certain lipid ratios using these parameters help in early identification of individuals at risk for CAD.

4. ANALYSIS

Thyroid hormones have significant role to play in lipid metabolism. The current study showed significant increase in TC, TG, and LDL-C in hypothyroid patients compared to controls which is in accordance with other studies16,17. Our study also showed effect of hypothyroidism on lipid parameters is more marked in patients with higher serum TSH levels. As TSH levels goes on increasing dyslipidemia also increased. A study conducted in Andhra Pradesh on female patients, suggests that effect of hypothyroidism on the serum concentration of lipids is more marked in patients with higher serum TSH levels. Hence lipid abnormalities exhibit great individual variability and there might be a potential link between hypothyroidism and atherosclerosis. Decreased thyroid secretion greatly increases the plasma

cholesterol concentration because of decreased rate of conversion of cholesterol to bile acids and consequent diminished loss in the feces due to decreased number of LDL receptors on liver cells19. According to FA Khan significant increase in levels of TC in hypothyroid patients compared to controls20. Hypercholesterolemia is due to decreased activity of Study done by Jiskra et al, in hypothyroidism the number of LDL receptors in the liver decreases and causes delayed clearance of LDL as a result there is an increase in overall cholesterol and LDL-C22. Our study results are in consistent with Ravi Shekhar and et al, who reports that total cholesterol and LDL levels were elevated in hypothyroidism and their levels decreases with treatment23. Hypothyroidism is associated with increased triglyceride levels. This is due to decreased activity of lipoprotein lipase (LPL), which results in decreased clearance of triglyceride rich lipoproteins24. Our study also revealed that there is significant increase in triglycerides in hypothyroid patients (all groups) compared to controls.

increase in serum HDL levels in hypothyroid patients of all the groups compared to control group. But study done by Lakshmi LJ and et al showed that there is significant decrease in HDL levels in hypothyroid patients compared to controls which is contradictory to our results27. The lipid ratio predicts cardiovascular disease risk better than isolated lipoprotein subfractions. Our study shows there is significant increase ($p < 0.05$) in TC/HDL and LDL/HDL ratios in cases compared to controls which is in accordance with study done by Khan FA. The LDL-C/HDL-C is a better predictor for risk of heart disease than LDL-C alone. The several studies have found that the LDL-C/HDL-C ratio is an excellent monitor for effectiveness of lipid lowering therapies. If the ratio of TC/HDL is more than 3.5 risks is more. Similarly LDL/HDL ratio more than 2.5 is also detrimental28. Our study shows there is statistically significant (< 0.001) positive correlation between TSH with serum levels of TC, LDL-C, TGs and HDL-C. Study done by few authors also found that there was significant positive correlation between serum TSH values and lipid parameters.

CONCLUSION

Our study results reveal that there is dyslipidemia in hypothyroid patients and also suggests that the effect of hypothyroidism on lipid parameters is more marked in patients with higher serum TSH levels. Hence patients presenting with dyslipidemia are recommended to be investigated for hypothyroidism. Study also reveals the importance of calculating lipid ratios from individual lipid profile parameters without any economic burden to the patients. Lipid ratios are better indicators of dyslipidemia and cardiovascular risk in hypothyroid patients by these simple calculations.

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