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Original Research Article

Free Triiodothyronine as a Novel Acute Phase Reactant in Acute Myocardial Infarction

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ABSTRACT

The burden of both cardiovascular mortality and morbidity has escalated in recent time. Atherosclerosis initiates the events of cardiovascular diseases. Inflammation initiates atherosclerosis whereas myocardial infarction is the end point of the atherosclerosis trajectory. Free triiodothyronine, the unbound portion, is used traditionally as thyroid function test analyte. Literature reviews suggests an immense role of free triiodothyronine as an inflammatory marker in pathogenesis of atherosclerosis and chronic kidney disease. The purpose of this observation study was to determine the serum concentration of free T3 in patients suffering from myocardial infarction and evaluate its association with C-reactive protein (CRP) and compare with healthy subjects. The study participants were evaluated with ECG and Echocardiography for cardiac changes. The data was calculated and compared using IBM SPSS software. The serum concentration of free T3 was significantly lower in cases (p=0.02). In this instance, it was observed that free T3 may be associated with inflammation of cardiovascular disease.

Keywords: Atherosclerosis, triiodothyronine, C-reactive protein

INTRODUCTION

Acute myocardial infarction (AMI), the leader of cardiovascular mortality, results from cessation of myocardial blood supply due to blockage of coronary arteries. The blockage in Coronary circulation mainly arises from the rupture of an atherosclerotic plaque. [1] Myocardial infarction is the end of the trajectory of atherosclerosis whereas free radical mediated endothelial injury and dyslipidemia initiates the event of atherosclerosis. Recent observations suggest that the atherosclerotic process is characterized by a low-grade inflammation altering the endothelium of the coronary arteries and is associated with an increase level in markers of inflammation such as acute phase proteins and cytokines. Inflammation plays an important role in determining atherosclerotic plaque stability. C-reactive protein (CRP) is a sensitive but nonspecific marker for inflammation. [2] With the advent of laboratory technology high-sensitivity CRP assays are used as a means of screening the patients at risks for cardiovascular disease. [3] The thyroid gland and cardiovascular system are intimately related. Studies have shown, that thyroid hormone has a fundamental role in cardiovascular homeostasis, influencing cardiac contractility, heart rate, diastolic function and systemic vascular resistance. [4] Abnormal metabolism of thyroid may lead to various heart diseases, like accelerated coronary atherosclerosis. [5] Coceani et al.

have demonstrated that free triiodothyronine (T3) levels are inversely correlated with the presence of coronary artery disease (CAD), whereas Low triiodothyronine (T3) syndrome causes an adverse prognosis for CAD. [6] In this instance, a study has been done to detect whether free triiodothyronine has a role in the inflammatory process of acute myocardial infarction.

MATERIALS AND METHODS

The present work is a hospital based cross sectional study and based on the measurement of free T3 and CRP in serum of AMI patients and healthy controls. For this purpose, blood samples were drawn from 37 AMI patients diagnosed by history, physical examinations & routine laboratory investigation (ECG and Troponin T).Similarly, 37 age and gender matched healthy subjects were also enrolled in the study. The study was conducted after receiving approval from the Institutional Ethical Committee. The samples were collected after getting proper consent from both patients and control. Patients free from any thyroid disease, AMI and infection were selected as control. Patients with previously history of thyroid disease, history of intake of iodine containing drugs, radioactive iodine were excluded. The estimation of free T3 and CRP was done in the Department of Biochemistry of Nil Ratan Sircar Medical College and Hospital, Kolkata within the study period of one month. Serum free T3 and CRP was estimated using ELISA and immunoturbidimetry respectively. The methods were checked for in-house performance parameters like precision (within and between run). The precision was found to be 6.9% (desirable imprecision <4.7%,) and 9% (desirable imprecision <21.1%). [7]

Statistical methods:

The age, values of free T_3 and CRP the cases suffering from acute myocardial infarction and healthy controls were extrapolated in Microsoft excel. The data was checked for normal distribution using Kolmogorov Smirnov test. The values were normally distributed as p >0.05. The mean values were assessed and compared using Student's t-test. The p values less than 0.05 were considered statistically significant. The statistical analysis was done using IBM SPSS 16 software.

RESULTS

The mean age of the diseased patients and controls was 56.2±2.06years. The mean values of serum free T3 was higher in healthy controls in comparison to diseased patients as evident from table 1. The independent sample t –test between the free T3 concentrations showed that the serum concentration of Free t3 was significantly lower in the diseased patients. The p-value is .02. The result is significant at p < .05. On the other hand, the mean concentration of CRP was 2.81 mg/dl in diseased patients which was significantly (P=0.04) higher than the healthy controls (1.14 mg/dl). The mean concentration of LDL and HDL in diseased patient was 124 mg/dl and 32 mg/dl respectively.

Table: 1 Showing the mean values of age, free T3 and CRP and expressed Mean±SEM

SL.NO	GROUP	Mean fT3 (pg/ml)	Mean CRP(mg/dl)	Student's t-test
1.	Diseased cases	1.89 +0.13	2.81+0.40	P=0.02
2.	Healthy Control	2.6 +0.27	1.14+0.27	P=0.04

* P < 0.05 considered significant.

DISCUSSION

The mean age of the diseased patient is 56.2±2.06years and this is quite similar to the epidemiological data suggesting the mean age of cardio-vascular mortality. After statistical analysis it is evident, that the mean concentration of Free T3 is significantly lower whereas CRP is significantly higher in diseased patients. Thus, Free T3 may be a negative acute phase reactant. Moreover, the findings of our studies are in accordance with study of Harun et al. [8] suggesting that the levels of FT3 were reduced in patients with CSFP.

Moreover, literature review suggested the hypothyroidism may stimulate the atherosclerotic process and related coronary flow pattern. Previously both subclinical and overt hypothyroidism states have been found to be associated with coronary artery disease. The study findings are also in accordance with the findings of Ascheim and Hryniewicz in 132 cardiac outpatients. [9] Various mechanisms have been postulated for cardiovascular risks in hypothyroidism like impaired cardiac contractility [10] and diastolic function, increased systemic vascular resistance, [11] hypercholesterolemia, C-reactive protein, [12] homocysteine and decreased endothelial-derived relaxation factor. The pathophysiology for low circulating T3 and free T3 is the reduced enzyme activity of5'monodeiodinase responsible for peripheral conversation of T4 to T3. However, the correlation coefficient between CRP and free T3 is -0.3. Thus there is a negative association between CRP and free T3 though not so strong. This may be due to lower sample size. Moreover, hsCRP is a better marker for cardiac risk assessment. So, it would have been better if the work would have been done with hsCRP

CONCLUSION

Despite the limitations imposed by small sample size of samples, low free T3 is evident inflammatory marker in AMI. However, the exact mechanism of free T3 in inflammatory process of atherosclerosis and AMI is yet to be established and warrants study with larger of population. Moreover, analysis of other parameters like hsCRP, coronary flow by angiography may strengthen the study results and help us to use low free T3 as a predictive marker.

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