Original Research Article

Evaluation of Thyroid Hormone Status in Acute ST Elevation Myocardial Infarction

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Abstract

Introduction:A change in thyroid function caused by non-thyroidal illnesses is found in acute myocardial infarction (AMI), as well as malnutrition, sepsis, and surgery. The goal of this study was to discover SES in AMI and its relationship to ventricular function and CKMB. **Aim:** To investigate whether low T3 syndrome in Acute ST elevation MI is associated with severity of myocardial injury. Severity of MI is evaluated by Isoenzyme CKMB and echocardiography.**Material and Methods:**Patients with acute ST elevation MI were studied over the course of a year in a cross-sectional, observational research. The researchers looked at 40 instances of AMI in a row. On day 1 (24-36 hours following the start of chest discomfort) and day 7, the thyroid profile and CKMB levels were measured. The left ventricular function was assessed by echocardiography.**Results:**There was no significant difference in serum T3, T4, TSH when comparing the mean values on day 1 and day 7. There was no link between the site of AMI and T3 levels, however there was a significant correlation between LVEF (p=0.05) and T3 levels. There was also a strong negative association between CKMB and T3 levels (p=0.0001).**Conclusion:**The LVEF and T3 levels had a significant relationship. There's also a strong negative relationship between CKMB and T3 levels. During short-term follow-up, those SES in AMI were linked to poor LV dysfunction.ma

Keywords: Thyroid hormone, Myocardial infarction**,** Left ventricular ejection fraction

Introduction

The thyroid hormone system is downregulated in severe disease from any source, including myocardial infarction and persistent heart failure, but not in otherwise healthy patients. The usual feedback control of thyroid homeostasis is disrupted in this situation, which is known as Euthyroid ill syndrome or low T3 syndrome [1].There is a decreeing T3levels in this circumstance, but no change in TSH levels. The conversion of the prohormone T4 to T3 is reduced due to decreased peripheral 5'deiodinase activity, and synthesis of reverse T3, an inactive metabolite, is increased, which is thought to be a typical feedback reaction to minimize metabolic demand. Thyroid hormone affects the cardiovascular system in a variety of ways . T3 raises heart rate, contractility, and cardiac output, which increases oxygen and nutrient consumption. It also lowers systemic vascular resistance and improves diastolic relaxation, resulting in a more efficient metabolic condition of the myocardium [2].SES has traditionally been viewed as an adaptive phenomenon in the setting of myocardial infarction to reduce the work load of the diseased heart by reducing energy consumption, but there is evidence that it has an effect on the prognosis, both long and short term, and that its presence predicts more frequent complications, poorer left ventricular function, and increased mortality. Changes in thyroid function are considered to be linked to a strategy for sustaining energy in the face of a disruption in systemic homeostasis induced by an acute ischemia event, or to inflammatory cytokines functioning as an inflammatory marker, or both. In acute myocardial infarction (AMI), the thyroid hormone system is rapidly deregulated [3]. This might be useful in the case of acute ischemia. In early samples, patients with angina exhibited greater amounts of interleukin-6 and c reactive protein, as well as a more depressed hormone system. Thyroid deficiency in individuals with angina may have existed prior to the onset of the infarction process. Low T3 levels are recognized to be important independent predictors of death in individuals admitted to the hospital for cardiac reasons [4]. The determination of reverse t3 levels might be a useful and easy tool for better identifying individuals with myocardial infarction who are at high risk of death. The significance of detecting "Euthyroid sick syndrome" in patients with coronary heart disease, which has been linked to a bad outcome in individuals with acute coronary syndrome.[4].As a result, the goal of this study is to see if low T3 levels are linked to the severity of Acute ST elevation MI, as measured by CKMB levels and left ventricular function.

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Material and Methods

It is a cross sectional observational study done over a period of 1 year January 2019 to September 2020 in Patients admitted to the ICCU, Gandhi hospital with acute ST elevation MI and an age of above 40 years, regardless of sex or clinical severity, were included in this study.Acute elevation of the ST Clinical history, ECG abnormalities, echo cardiography, and cardiac isoenzyme CKMB were used to identify MI. Thyroid function tests were performed twice, with the first sample taken 24-36 hours following the onset of chest discomfort and the second sample collected on day 7. Thyroid hormone levels were determined using a chemilumniscent assay, whereas CKMB levels were determined using an immunoinhibition technique.

Exclusion Criteria

Endocrine and thyroid abnormalities, and a history of overt thyroid disease Previous MI, cardiac illness, and the use of medicines such as amiodarone and phenytoin Anticoagulants, which are taken orally, 3 months prior to admission, intervention and surgical procedures were conducted in the case. Patients who have had radiography contrast media within the previous three months prior to admission, Renal failure, often known as chronic renal failure, is a condition Starvation, Sepsis.

Statically analysis – Continuous variables such as age, serum T3, T4, and TSH were expressed as mean ± SD. The relationship between serum T3 with LVEF and CKMB were calculated. Spearman correlation and the students' t test were used in the analysis. Significant was defined as a two-tailed 'p' value of less than 0.05.

Results

Over the course of a year, 40 patients with acute ST elevation MI were studied. There were 22.5 percent females (nine patients) and 77.5 percent males (31 patients) among these persons. The ratio of males to females is 3.4:1. Males were 56.51± 5.87 years old, while females were 61.13 ± 9.8 years old. There was no statistically significant difference between T3, T4, and TSH levels on days 1 and 7, with 'p' values of 0.2586, 0.8815, and 0.8741, respectively.

Table 1: Distribution of subjects according to serum T3, T4, TSH levels

Mean Thyroid Hormone Level	DAYIMEAN ±S.D	Day7Mean ±S.D	P VALUE
Serum T3 (ng/ml)	0.86±0.34	0.92±0.31	0.2586
Serum T4 level (mcg/dl)	7.01±2.07	6.96±2.05	0.8815
Serum TSH level (mcIU/ml)	2.12±1.55	2.08±1.46	0.8741

The most patients had inferior wall MI, which accounted for 40.20 percent of the total, or 16 patients, followed by anterior wall MI, which accounted for 33.30 percent of the total, or 14 patients. Low T3 was seen in 29.2 percent of patients with severe anterior wall MI, or four members, and in 15% of patients with inferior wall MI, or two members. Although there was no statistically significant connection between the reduction in T3 levels and the type of MI, i.e., 'p' value 0.11 for inferior wall MI and 0.403 for anterior wall MI, there was no statistically significant association between the reduction in T3 levels and the type of MI. Two individuals had low T3 in lateral wall MI (100%) and the 'p' value was 0.89. Anterior septal wall MI and anteriolateral l wall MI both had no patients with low T3.

Table 2:Correlation of serum T3 (day 1) with types of myocardial infarction

Location of MI	Serum T3 levels T3<0.8	T3>0.8	P value
Extensive anterior wall MIN=14	4(29 .20%)	10(70.80%)	P=0.110
Antero septal MIN=3	0	3(100%)	P=0.486
Antero lateral MIN=4	0	4(100%)	P=0.371
Inferior wall MIN=16	2(15%)	14(85%)	P=0.403
Lateral wall MIN=2	2(100%)	0	P=0.89

Patients were split into two groups based on their left ventricular ejection fraction (LVEF): those with an LVEF of less than 40% and those with an LVEF of more than 40%. On Day 1, LVEF and T3 levels are linked. Patients with an LVEF of less than 40% showed a higher rate of low T3. There were 14 members with LVEF less than 40%, and six of them had low T3. There were 26 members with LVEF greater than 40%, and one of them had low T3. Low T3 and LVEF had a significant relationship, with a 'p' value of 0.053.

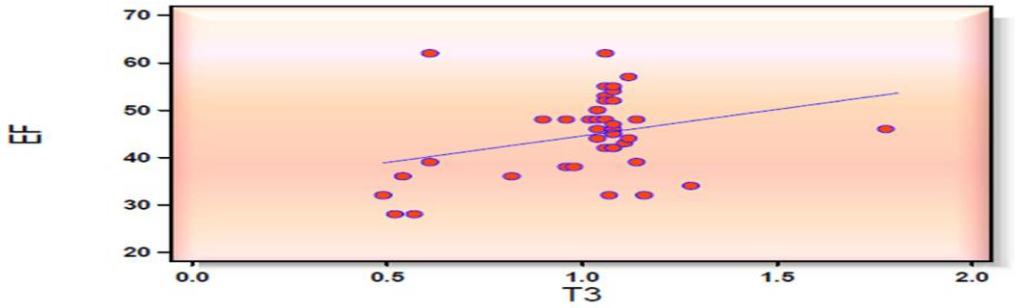

Fig 1: scatter diagram showing correlation between serum T3 and LVEF

On day 1 and day 7, CKMB was also tested and associated with T3 levels. On day 1, there was a statistically significant link between elevated CKMB and T3, with a 'p' value of 0.0001. T3 was shown to be lower the higher the CKMB.

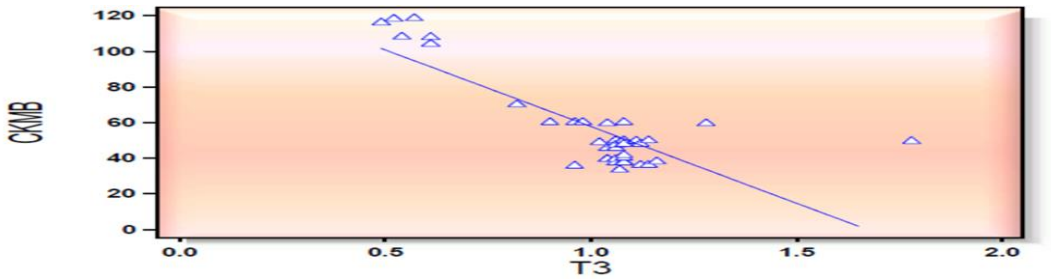

Fig 2: Correlation serum T3(Day1) with CKMB levels

Discussion

During the acute phase of myocardial infarction, the thyroid hormone was transiently down regulated in other wise Euthyroid people. The lowest blood T3 levels were detected 24 hours after the beginning of chest pain, at the same time as the highest serum CKMB levels, but total T4 and TSH serum concentrations remained within normal ranges and did not vary substantially in both groups. The degree of the decline was greater in the group of patients with LVEF less than 40%, who fared worse than those with LVEF greater than 40%. There was a strong negative connection between T3 and CKMB levels, showing that the severity of AMI and changes in T4 to T3 conversion had an adverse qualitative relationship.The likely reason for the temporary fall in serum T3 levels is multifactorial and can be ascribed to decreased hepatic conversion of T4 to T3 as a result of decreased 5' monodeiodinase activity, especially in severe heart failure. Reduced activity also decreases peripheral T4 to T3 conversion, directing it to the inactive reverse T3 pathway. Reduced serum protein binding, a larger distribution volume, and a shorter half-life [6].The hepatic mono Deiodinase activity appears to be inhibited by serum IL-6, which has been found to be elevated in the low T3 condition. Cardiac myocytes in the reperfused viable myocardium's border zone, monocytes, and macrophages all generate IL-6, which may play a role in the alterations in blood T3 levels following an acute MI. Low T3 syndrome is known to be caused by high catecholamine levels. The elevated amounts of catecholamines observed in individuals with AMI may be to blame for the alterations in thyroid hormone levels in the Euthyroidunwell syndrome.

Pavlou et al. [7]and Friberg et al. [5] have also reported down regulation in T3 levels in patients with acute Myocardial Infarction. It has been proposed that if the thyroid hormone system is downregulated when AMI occurs, this may be beneficial since the oxygen demand of the myocardium may be lowered when metabolic rate is reduced [6,5].. It's unclear if Euthyroid Sick Syndrome is beneficial after an AMI when damage has occurred and heart failure may be developing. Thyroid hormones affect ventricular function by increasing the activity and expression of sarcoplasmic calcium adenosine triphosphate.This ATPase is important for removing calcium from the cytosol during diastole, allowing actin–myosin cross bridging to be uncoupled. It is therefore crucial for heart diastolic function. It also modulates the amount of calcium in the sarcoplasmic reticulum accessible for systolic contraction, making it vital for heart systolic function. [6,7]. Intracellular calcium handling is impaired when the thyroid system is down regulated in AMI, which may lead to myocardial stunning and reperfusion damage owing to calcium overload. [8]. Furthermore, thyroid hormone system downregulation causes an increase in systemic vascular resistance and cardiac afterload. Cardiac output will be decreased if the heart is unable to cope with this. Thyroid hormone treatment at dosages adequate to restore serum T3 significantly improved cardiac function in animals, suggesting that T3 treatment in the setting of cardiac illness and altered thyroid metabolism might be helpful. The effects of T3 therapy on AMI patients are still being studied. The origin and consequences of Euthyroidsick syndrome in AMI can be better understood with prospective research on bigger patent materials. In addition to other factors, the biochemical marker may be useful in predicting the prognosis of our patients with coronary artery disease.The male to female ratio in this study was 3.4:1, indicating that men were more likely to be affected. Similarly, Leif Friberg et al. [5] found a male to female ratio of 2.4:1 in their study of patients with acute myocardial infarction, and a male to female ratio of 1.9:1 in another study by Friberg et al. [5]. Similar studies by Lymvaios et al. [9] and Pimentel et al. [10] observed male preponderance (male to female ratios of 1.6 : 1 and 1.5 : 1 respectively)Males are afflicted earlier and more frequently than females, and gender is a major non-modifiable risk factor for AMI. Males are impacted about twice as frequently as females in current and previous research. Higher HDL levels in premenopausal women provide some protection; however, following menopause, the level drops and the protective effect is lost.In the current study, the average age of onset of acute myocardial infarction was 57.98 ± 11.71 years. Male and female patients had a mean age of 56.51 ± 5.7 and 61.13 ± 9.8 years, respectively. The mean age of presentation is substantially lower than in western studies such as those conducted by Friberg et al. [5] (mean age for men and females 66 ± 13 years and 74 ± 9 years, respectively), and Lymvaioset al. [9] (mean age of presentation 62.3 ± 10.2 years). Heart disease was growing 5–10 years sooner among Asian Indians than in other groups throughout the world, according to Sharma et al. [11]. In Indians, the average age at which acute myocardial infarction first manifested itself was 53 years.On the first and seventh days of the trial, there were no significant changes in T3 T4 TSH levels. In their study, Friberg et al. [5] found that the thyroid hormone system was rapidly down regulated, with maximal changes occurring 24–36 hours after the onset of symptoms [mean total T3 levels decreased by 19 percent ($p=0.02$), inactive metabolite reverse T3 (rT3) levels increased by 22 percent ($p=0.01$), and TSH levels decreased by 51 percent ($p < 0.001$)].Similarly, Rajappa [12] examined thyroid profiles in acute myocardial infarction patients serially up to 7 days after start of symptoms and found a progressive rise in T3 with normalization on the 7th day. Pimental et al. [13] looked at mean T3, T4, and TSH on days 1, 4, and 7, and found that day 4 had the most hormonal alterations. On day 4, mean reverse T3 was at its greatest, although serum TSH, T4, and free T4 were normal. T3 levels were gradually normalised after 6 months in patients with a change in LVEF of less than 50%, but remained unaltered from baseline in subjects with a change in LVEF of more than 50%, according to Lymvaios et al. [9]

The degree of cardiac dysfunction determines the type of recovery in SES. Changes in reverse T3 are more noticeable, and further serial thyroid hormone estimation is more instructive. There was no estimate of reverse T3 or daily thyroid hormones in this research. As a result, there was likely no statistically significant difference between day-1 and day-7 results. In this investigation, there was no significant connection between T3 levels and the kind of AMI. We have not found any research that link SES to the kind of AMI, however there are studies that link SES to the degree and severity of AMI as measured by the CK-MB. More patients with suppressed T3 are likely to have substantial anterior wall MI, which implies more severe and extensive myocardial injury (due to a potential proximal left anterior descending artery lesion) and, as a result, leads in profound thyroid hormone system down-regulation. The present study's lower sample size, on the other hand, is unlikely to have produced a statistically significant association.

In this investigation, individuals with lower LVEF had a substantially higher rate of serumT3 lowering ($p < 0.05$). SerumT3 was substantially lower in patients with LVEF 40 percent compared to those with LVEF >40 percent ($p < 0.05$) when using a 40 percent cut-off. Lymvaios et al. [9] found that total T3 levels in plasma at 48 hours were substantially associated with LVEF at 48 hours ($r=0.50$, $p=0.0004$) in a comparable research. After a 6-month follow-up, consistently low T3 levels were linked to poor and non-significant LVEF recovery, corresponding with late functional recovery.Rajappa et al. [12] found that individuals with LVEF less than 50% had substantially lower T3 than those with LVEF more than 50% ($p < 0.001$). Patients with an atrioventricular plane displacement of less than 8 mm, equivalent to a left ventricular ejection fraction of around 35 percent, had a substantially ($p=0.03$)lower mean T3 concentration than other patients, according to Friberg et al. [5.] Patients with low fT3 exhibited a slightly lower left ventricular ejection fraction than those with normal fT3 ($p=0.025$), according to Iervasi et al. [14].

Pantose et al. [15] discovered a substantial connection ($r=0.56$, $p=0.0004$) between total T3 and EF percent. On day one, Adawiyah et al. [16] found a significant difference in killips categorization between the SES and non-SES groups ($p=0.030$). More patients hospitalized with Killips class III and IV (cardiogenic shock)

acquired SES in their research. Thyroid hormones are necessary for both systolic and diastolic cardiac function. Intracellular calcium handling is disrupted when the thyroidhormone system is down-regulated in AMI, which may lead to myocardial stunning and reperfusion damage owing to calcium overload. Furthermore, due to this down-regulation, there is an increase in systemic vascular resistance, which leads to an increase in cardiac workload.

Conclusion

In acute myocardial infarction, the thyroid hormone system is quickly downregulated. This might be useful in the case of acute ischemia. T3 levels are related to the severity of heart injury and may have predictive significance. As a result, T3 blood levels have a role in the development of the AMI severity index.The mean level of T3, T4, TSH on day 1 and the association between T3 and left ventricular dysfunction were not significantly different. A low T3 level was linked to a low LVEF and a high CKMB. As a result, during short-term follow-up, SES in AMI is linked with poor LV dysfunction.

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