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Correlation between the Level of Serum Free Triiodothyronine and the Degree of Myocardial Injury in Patients with Acute ST Segment Elevation Myocardial Infarction

Ahmed Kamal[1*], Ayman A Elmagd[2], Ahmed Y Hegab[3], Abdel-aziz Rezk[4] and Mohamed S El-Shorbgy[5]

[1]*Cardiology Resident, Al-Azhar University, Egypt*

[2]*Professor of Cardiology, Faculty of Medicine, Al-Azhar University, Egypt*

[3]*Consultant of Cardiology, National Heart Institute*

[4]*Assistant Professor of Cardiology, Faculty of Medicine, Al-Azhar University, Egypt*

[5]*Assistant Professor of Clinical Pathology, Faculty of Medicine, Al-Azhar University, Egypt*

[*]**Corresponding author:** Ahmed Kamal, Cardiology Resident, Al-Azhar University, Egypt, Tel: +201006900743; E-mail: dr_a.kamal87@yahoo.com

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Abstract

Background: Previous studies have suggested that hypothyroidism correlated with coronary artery diseases (CAD) mortality in long-term cohort studies, but whether the thyroid function status is associated with myocardial injury in acute ST-elevation myocardial infarction (STEMI) has not been investigated sufficiently.

Aim: We aimed to investigate whether the lower free triiodothyronine level in acute STEMI is associated with more severe myocardial injury.

Methods: This was a prospective observation cohort study that was conducted from August 2016 to April 2017 and included 50 patients presented to the coronary care Unit in Al Mataryia teaching hospital with acute STEMI and treated with thrombolysis. All patients were subjected to complete clinical assessment, serial ECGs, Echo, full labs, thyroid hormones, cardiac biomarkers, C-reactive protein (CRP).

Results: There were 8 patients (16%) who had hypothyroidism including low-T3-syndrome (4 patients, 8%), subclinical hypothyroidism (3 patients, 6%) and clinical hypothyroidism (1 patient, 2%). After adjusting for conventional risk factors (age, gender, smoking, diabetes mellitus, dyslipidemia, hypertension), free triiodothyronine (FT3) was significantly and negatively correlated with CKMB (r=-0.294, P=0.038), cTnI (r=-0.368, P=0.009), and positively correlated with EF (r=0.385, P=0.006), indicating that the lower thyroid hormone level correlates with the more severe cardiac injury in STEMI patients. FT3 also had a negative correlation with CRP (r=-0.404, P=0.004), which might indicate that hypothyroidism may activate the inflammation response.

Conclusion: The lower FT3 level correlates with higher level of cardiac biomarkers and lower left ventricular ejection fraction (LVEF), Low T3 syndrome may be a predictor for myocardial injury in STEMI. These results may warrant further study to investigate whether reversing the hypothyroidism could benefit the STEMI patients.

Keywords: Myocardial injury; Thyroid hormone; Free triiodothyronine; Acute ST segment elevation myocardial infarction

Introduction

Thyroid hormones have a major role in the cardiovascular system function and cardiac hemodynamics [1], as well as to maintain the cardiovascular homeostasis [2]. A slightly change in thyroid status affects ventricular function, serum cholesterol levels, and heart rate and rhythm, and increases risk of coronary artery disease and cardiovascular mortality [3]. Nevertheless, the relation between anomalous thyroid function and cardiovascular effects remains indistinct [4].

The active hormone T3 increases heart rate, contractility, cardiac output, and thus elevates consumption of oxygen and nutrients [2]. However, it can also decrease systemic vascular resistance and improve the ability of diastolic relaxation, leading to a more efficient metabolic status of myocardium [5].

The "low T3 syndrome" is a profile of low serum triiodothyronine (T3), normal thyroxin (T4), and normal TSH that can be seen in acute or chronic illnesses. This syndrome leads to the similar changes in cardiac function (decreased maximal rate of contraction and relaxation) and gene expression (alteration in myosin heavy chain isoform expression) as does primary hypothyroidism. In patients with cardiac disease, this syndrome is a major cause of death [6].

The thyroid gland produces predominantly inactive T4. This means that we must convert T4 to T3, called thyroid conversion, in order to have normal thyroid responses. This conversion takes place primarily in the liver and gut mucosa, more than 80% of the biologically active hormone triiodothyronine (T3) derives from peripheral conversion of prohormone thyroxine (T4) secreted by the thyroid gland [7].

Patients and Methods

This prospective observational study involved 50 patients from the attendants of the emergency department, Al Matarya teaching hospital (MTH) who admitted with the diagnosis of ST elevation myocardial infarction (STEMI) and received streptokinase 1.5 MU as per the institutional protocol, during the period from August 2016 to April 2017.

Patients were observed in the intensive coronary care unit for at least five days. All patients presented, within 6 h from symptom onset with ECG show ST-segment elevation myocardial infarction.

ST elevation at the J point, in at least 2 contiguous leads, ≥ 2 mm (0.2 mV) in men ≥ 40 years, 2.5 mm (0.25 mV) in men <40 years or ≥ 1.5 mm (0.15 mV) in women in leads V2–V3 and/or ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads [8].

Exclusion criteria were patients using corticosteroids, amiodarone and other drugs that are known to affect thyroid functions or who had received any iodinated contrast agent within the previous two weeks. Also patients with established diseases that are known to affect thyroid function tests, such as neoplasia, chronic renal failure, chronic obstructive pulmonary disease requiring antibiotic therapy, liver cirrhosis, active infection, and diabetic ketoacidosis will be excluded from the study.

The study protocol was approved by Al-Azhar University, Faculty of Medicine. A chart review was performed, and data were collected including patient demographics, medical history, examination, laboratory tests, ECG, echocardiography.

History was taken including age, gender, smoking was recognized as a lifetime history of >100 cigarettes in their entire life and had continued smoking in the last 6 months was considered a positive smoking history, while ex-smokers were defined having history of smoking at least 100 cigarettes in their entire life and had completely stopped smoking for at least 6 months [9], Current diabetes mellitus was recognized as having history of DM on admission with the use of oral anti-hyperglycemic full agents or any extended release insulin and confirmed by laboratory HbA1c on admission if more than 6.5% [10], dyslipidemia was defined by total cholesterol ≥ 220 mg/dl, triglyceride ≥ 150 mg/dl, high-density lipoprotein (HDL cholesterol) ≤ 40 mg/dl or current use of anti-hyperlipdemic drug [11], hypertension was defined as systolic/diastolic blood pressure ≥ 140/90 mmHg or patients having history of hypertension and current use of any antihypertensive medications [12], Family history of premature coronary artery disease was defined as fatal or non-fatal events in first degree relatives men <55 or women <60 years [11].

Full clinical examination was done including vital signs, cardiac examination to assess the Killip classification for each patient. Hypotension was defined as systolic blood pressure ≤ 90 mmHg requiring inotropic support with medications or intra-aortic ballon pump (IABP). Heart failure included advanced congestive heart failure (New York Heart Association functional class III/IV) or acute heart failure (Killip class II–IV).

Twelve leads ECG were obtained to confirm the diagnosis. M-mode, two-dimensional echocardiography and doppler examination was performed for all patients in the left decubitus position during normal respiration using a GE Vivid 5 Ultrasound Machine (Echo Pac; GE Vingmed, Horten, Norway) according to the recommendations of American Society of Echocardiography to detect left ventricle size, left atrium size, left ventricular ejection fraction, any wall motion abnormalities or ischemic complications and to detect any morbidities during the in hospital follow up.

Laboratory tests were done including routine labs that include liver and kidney functions. Serum cardiac markers that include Serial measurement of cardiac troponin I (CTnI), myocardial band of creatine kinase (CK-MB) and creatine kinase (CK). Measured hormones and their respective reference values are: CK (up to 195 U/L), CK-MB (up to 25 U/L), Troponin I (0-0.1 ng/ml). Lipid profile within 24 h of admission include total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), low- density lipoprotein–Cholesterol (LDL-C). Thyroid profile, after obtaining informed consent, blood samples (5 ml venous blood) was collected then sera was prepared for laboratory testing and kept at -20°C till time of analysis.

Free T3, Free T4, TSH will be measured using electrochem-iluminescent method (cobas-e411-Roche). Measured hormones and their respective reference values are: free T3 (1.3-5 pg/ml), free T4 (0.8-2 ng/dl), TSH (0.4-4 mIU/l) (Table 1).

Variables	Mean ± SD	Range
Age (years)	53.4 ± 5.9	42.0–68.0
Systolic blood pressure (mmHg)	124.9 ± 17.2	91.0–167.0
Diastolic blood pressure (mmHg)	81.6 ± 12.2	57.0–104.0
Heart rate (beat/minute)	78.3 ± 10.8	60.0–111.0
Ejection fraction%	49.5 ± 9.8	22.0–68.0
LVESD (cm)	4.0 ± 0.7	2.7–6.0
LVEDD (cm)	5.3 ± 0.6	3.8–7.0
Left atrial diameter (cm)	3.9 ± 0.5	2.3–4.7
Total cholesterol (mg/dL)	214.5 ± 58.5	157.6–377.0
Triglycerides (mg/dL)	164.4 ± 67.0	118.0–350.0
LDL (mg/dL)	138.8 ± 53.6	78.0–286.0
HDL (mg/dL)	42.8 ± 8.8	21.0–56.0
Creatine Kinase (IU/L)	1605.7 ± 432.1	844.0–2854.0
Creatine Kinase-Myocardial Band (IU/L)	217.2 ± 56.3	102.0–378.0
Troponin-I (ng/mL)	35.5 ± 12.7	11.0–60.0
C-Reactive Protein (mg/dL)	42.5 ± 6.7	29.0–63.0
Free Tri-iodothyronine	2.7 ± 1.2	0.8–6.2
Free Tetra-iodothyronine (ng/dL)	1.4 ± 0.3	0.3–2.0
Thyroid Stimulating	2.3 ± 1.2	0.4–5.3

Hormone (mIU/L)		

Table 1: Mean and range of the study variables.

Low T3 syndrome defined as a profile of low serum triiodothyronine (T3) below the normal reference value, normal thyroxin (T4) and normal TSH that can be seen in acute or chronic illnesses [6].

Subclinical hypothyroidism defined as elevated TSH above the normal reference value with normal free T3 and normal free T4 [13] (Table 2).

Overt hypothyroidism defined as elevated TSH above the normal reference value with decreased free T3 and T4 [13].

Variables		No.	%
Sex	Male	28	56
	Female	22	44
Smoking		19	38
Hypertension		26	52
Diabetes mellitus		6	12
Dyslipidemeia		17	34
	Chest pain	43	86
Presentations	Epigastric pain	4	8
	Dyspnea	3	6
STEMI	Anterior	29	58
	Inferior	21	42
	Euthyroid	42	84
	Low-T3 syndrome	4	8
Thyroid function	Subclinical	3	6
	hypothyroidism		
	Clinical	1	2
	hypothyroidism		
	Post infarction	6	12
	angina		
	Congestive Heart	8	16
	Failure		
Complications	Arrhythmia	3	6
	Shock	1	2
	Death	1	2

Table 2: Baseline characteristics of the study population.

During their hospital stay, patients with ST elevation myocardial infarction were observed for evidence of both in hospital morbidity and mortality. Morbidity among patients with ST elevation myocardial infarction was defined as post MI angina or significant arrhythmias or post MI heart failure or cardiogenic shock or mechanical complication during the hospital stay. Mortality was defined as in hospital death in patients admitted with ST elevation myocardial infarction.

Data were analyzed using Statistical program for social science (SPSS) version 20.0. Quantitative data were expressed as mean ± standard deviation (SD). Qualitative data were expressed as frequency and percentage. Independent samples t-test of significance was used when comparing between two means. Chi-square (X2) test of significance was used in order to compare proportions between two qualitative parameters.

Results

This was a prospective cross sectional observational study that involved 50 patients who presented to the emergency department of the Al Matareya Teaching Hospital (MTH) with acute ST-elevation myocardial infarction and treated with thrombolytics, within the period between August 2016 to April 2017.

The Mean age was 53.4 ± 5.9 years (ranged from 42-68 years). Males represented 56% (28 patients) of the study population while females represented 44% (22 patients). Fifty two percent (26 patients) were hypertensive while twelve percent (6 patients) were diabetic and thirty eight percent (19 patients) were smokers. Thirty four percent of study group (17 patients) had history of dyslipidemia.

From 50 patients presented with STEMI, 58% (29 patients) of them presented with anterior MI and 42% (21 patients) with inferior MI 86% (43 patients) of patients presented with chest pain, 8% (4 patients) with epigastric pain, and 6% (3 patients) with dyspnea.

As regard clinical examination. The mean heart rate (HR) was 78.3 ± 10.8 Bpm, the mean of systolic blood pressure (SBP) was 124.9 ± 17.2 mmHg and the mean of diastolic blood pressure (DBP) was 81.6 ± 12.2 mmHg.

In our study group, the mean Ejection fraction (EF) was 49.5 ± 9.8% ranged from 22.0-68.0%, while the mean of left ventricular end systolic diameter (LVESD) was 4.0 ± 0.7 cm ranged from 2.7-6.0 cm, the mean of left ventricular end diastolic diameter (LVEDD) was 5.3 ± 0.6 cm ranged from 3.8-7.0 cm and the mean of left atrial diameter (LAD) was 3.9 ± 0.5 cm ranged from 2.3-4.7 cm (Figures 1 and 2).

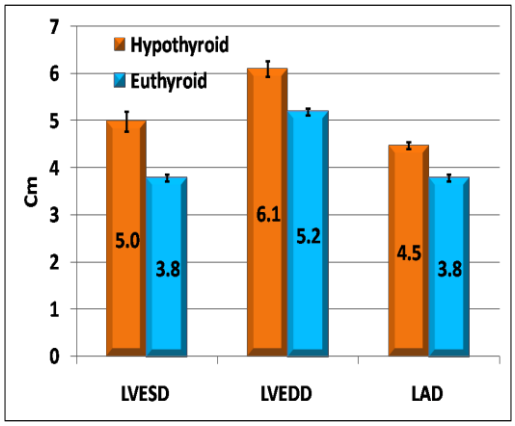

Figure 1: Comparison between hypothyroid and euthyroid patients regarding cardiac dimensions.

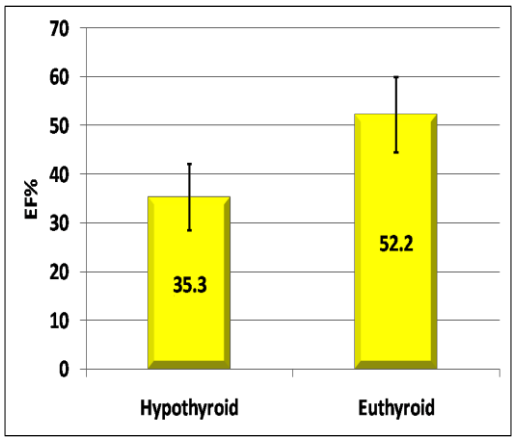

Figure 2: Comparison between hypothyroid and euthyroid patients regarding EF.

Among study group, the mean of total cholesterol was 214.5 ± 58.5 mg/dl while the mean of triglycerides was 164.4 ± 67.0 mg/dl, the mean of low density lipoproteins (LDL) was 138.8 ± 53.6 mg/dl and the mean of high density lipoproteins (HDL) was 42.8 ± 8.8 mg/dl.

The mean of total creatine kinase (CK) was 1605.7 ± 432.1 IU/L ranged from 844.0-2854.0 IU/L, while the mean of creatine kinase-myocardial band (CK-MB) was 217.2 ± 56.3 IU/L ranged from 102.0-378.0 IU/L, the mean of troponin-I was 35.5 ± 12.7 ng/ml ranged from 11.0-60.0 ng/ml and the mean of CRP was 42.5 ± 6.7 mg/dl ranged from 29.0-63.0 mg/dl.

In our study group, the mean of free tri-iodothyronine (T3) was 2.7 ± 1.2 pg/ml ranged from 0.8-6.2 pg/ml, the mean of free tetra-iodothyronine (T4) was 1.4 ± 0.3 ng/ml ranged from 0.3-2.0 ng/ml and the mean of thyroid stimulating hormone (TSH) was 2.3 ± 1.2 mIU/L ranged from 0.4-5.3 mIU/L. 84% (42 patients) of the study group were euthyroid, 8% (4 patients) had low Free T3 level, 6% (3 patients) had subclinical hypothyroidism and 2% (1 patient) had clinical hypothyroidism (Figure 3).

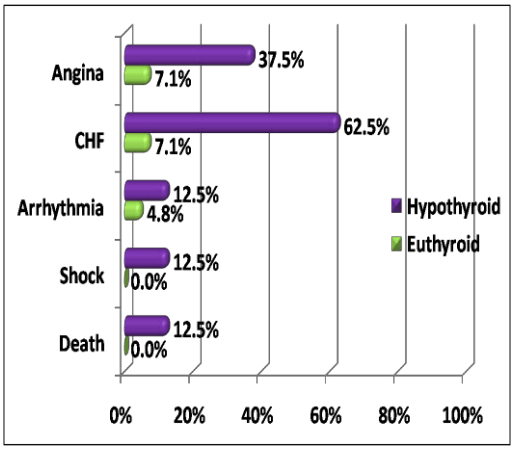

Figure 3: Comparison between hypothyroid and euthyroid regarding complications

Among our group, 12% (6 patients) had post infarction angina, 16% (8 patients) had congestive heart failure, 6% (3 patients) had arrhythmia, 2% (1 patient) had cardiogenic shock and 2% (1 patient) died (Figure 4).

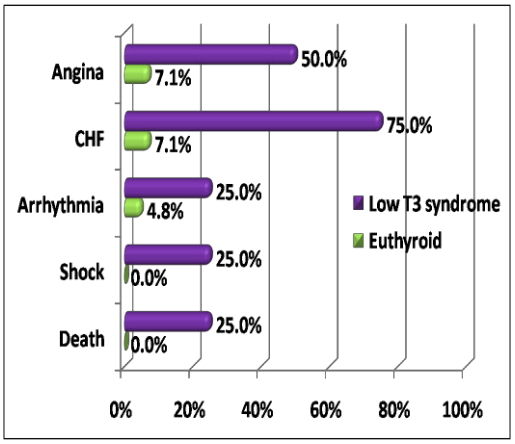

Figure 4: Comparison between low T3 syndrome and euthyroid patients regarding complications

We divided our patients according to thyroid status into two groups; hypothyroid group and euthyroid group. There was statistically significant difference between the two groups according to ejection fraction (p value<0.001), LVESD (p value<0.001), LVEDD (p value<0.001) and LAD (p value<0.001).

There was statistically significant difference between the two groups according to CK (p value<0.001), CK-MB (p value<0.001), troponin (p value<0.001) and CRP (p value<0.001) (Figures 5 and 6).

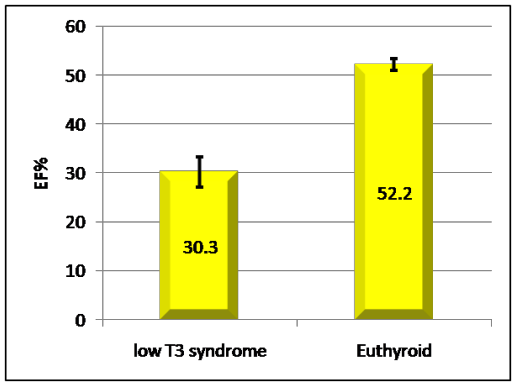

Figure 5: Comparison between low T3 syndrome and euthyroid patients regarding EF

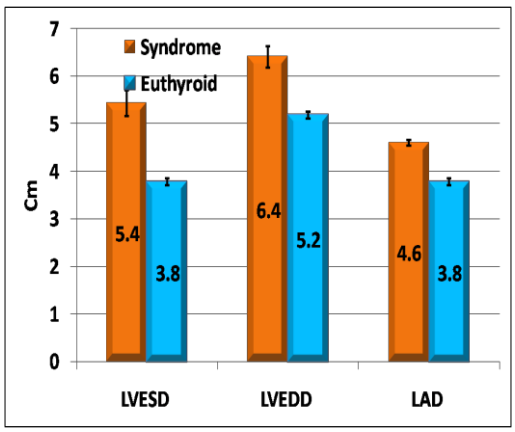

Figure 6: Comparison between hypothyroid and euthyroid regarding cardiac dimensions

Complications were more frequent among hypothyroid cases than euthyroid cases. There was statistically significant difference between the two groups only with post infarction angina (p value=0.044) and congestive heart failure (p value<0.001).

There was no statistically significant difference between the two groups regarding demographic characteristics and co-morbidities, types of STEMI, presentation and clinical condition and lipid profile.

Also, we compared between patients with low T3 syndrome and euthyroid patients regarding study variables and we found that there was statistically significant difference between the two groups according to EF (p value<0.001*), LVESD (p value<0.001*), LVEDD (p value<0.001*) and LAD (p value<0.001*).

There was statistically significant difference between the two groups according to CK (p value<0.001*), CK-MB (p value<0.001*), troponin-I (p value<0.001*) and CRP (p value<0.001*).

Complications were more frequent among low T3 syndrome cases than euthyroid cases with statistically significant difference only with congestive heart failure (p value=0.005).

There was no statistically significant difference between the two groups regarding demographic characteristics and co-morbidities, presentation, types of STEMI and clinical condition and lipid profile.

There was statistically significant positive correlation between FT3 and EF (p value=0.006) and negative correlations between FT3 and LVESD (p value=0.007), LVEDD (p value=0.009), LAD (p value=0.046), CK (p value=0.020), CK-MB (p value=0.038), troponin (p value=0.009) and CRP (p value=0.004). There were significant positive correlations between TSH and LVESD (p value=0.025), LVEDD (p value=0.039) and CRP (p value=0.015), negative correlation between TSH and EF (p value=0.031).

Discussion

The present study aimed to evaluate the impact of low circulating free T3 on the degree of myocardial injury in STEMI patients.

Of 50 patients, males represented 56% of study group. 12% of study group had history of diabetes, 34% of patients were dyslipidemic while 52% had history of hypertension and 38% were smokers. The mean heart rate (HR) was 78.3 ± 10.8 bpm, the mean of systolic blood pressure (SBP) was 124.9 ± 17.2 mmHg and the mean of diastolic blood pressure (DBP) was 81.6 ± 12.2 mmHg.

In our study, we found that, out of 50 patients, 8 patients (16%) were hypothyroid; 4 of them (8%) had low T3 syndrome, 3 patients (6%) were subclinical hypothyroid and 1 patient (2%) had clinical hypothyroidism. Also we found that the mean of total creatine kinase (CK) was 1605.7 IU/L, the mean of creatine kinase-myocardial band (CK-MB) was 217.2 IU/L, the mean of troponin-I was 35.5 ng/ml and the mean of CRP was 42.5 mg/dl. 70% of study populations had EF <55% by conventional transthoracic echocardiography with mean EF of 49.5% (Table 3).

Variables		Hypothyroid (N=8)	Euthyroid (N=42)	P-value
Age (years)		50.9 ± 6.7	53.9 ± 5.7	^0.187
Sex	Male	4 (50.0%)	24 (57.1%)	&0.718
	Female	4 (50.0%)	18 (42.9%)	
Smoking		5 (62.5%)	14 (33.3%)	&0.232
Hypertension		4 (50.0%)	22 (52.4%)	&1.000
Diabetes mellitus		1 (12.5%)	5 (11.9%)	&1.000
Dyslipidemia		4 (50.0%)	13 (31.0%)	&0.419
Presentations	Chest pain	8 (100.0%)	35 (83.3%)	&1.000
	Epigastric pain	0 (0.0%)	4 (9.5%)	
	Dyspnea	0 (0.0%)	3 (7.1%)	
STEMI	Anterior	7 (87.5%)	22 (52.4%)	&0.117
	Inferior	1 (12.5%)	20 (47.6%)	
Systolic Blood Pressure (mmHg)		119.4 ± 7.3	126.0 ± 18.4	^0.327
Diastolic Blood Pressure (mmHg)		77.6 ± 5.8	82.4 ± 12.9	^0.313

Heart Rate (beat/minute)	79.5 ± 4.3	78.1 ± 11.6	^0.743
Ejection Fraction %	35.3 ± 6.8	52.2 ± 7.7	<0.001*
LVESD (cm)	5.0 ± 0.6	3.8 ± 0.5	<0.001*
LVEDD (cm)	6.1 ± 0.5	5.2 ± 0.5	<0.001*
Left Atrial Diameter (cm)	4.5 ± 0.2	3.8 ± 0.5	<0.001*
Total cholesterol (mg/dL)	209.5 ± 64.1	215.5 ± 58.2	0.795
Triglycerides (mg/dL)	160.6 ± 73.0	165.1 ± 66.7	0.865
LDL (mg/dL)	132.1 ± 58.3	140.1 ± 53.3	0.704
HDL (mg/dL)	45.3 ± 9.2	42.4 ± 8.8	0.404
Creatine Kinase (IU/L)	2182.4 ± 456.2	1495.9 ± 332.2	<0.001*
Creatine Kinase-Myocardial Band (IU/L)	280.5 ± 26.8	205.1 ± 52.3	<0.001*
Troponin-I (ng/mL)	52.6 ± 6.3	32.2 ± 10.9	<0.001*
C-Reactive Protein (mg/dL)	53.3 ± 5.9	40.5 ± 4.6	<0.001*
Post infarction angina	3 (37.5%)	3 (7.1%)	0.044*
Congestive Heart Failure	5 (62.5%)	3 (7.1%)	<0.001*
Arrhythmia	1 (12.5%)	2 (4.8%)	0.414
Shock	1 (12.5%)	0 (0.0%)	0.16
Death	1 (12.5%)	0 (0.0%)	0.16

Table 3: Comparison between hypothyroidism and euthyroid patients regarding study variables

Among our group, 12% (6 patients) had post infarction angina, 16% (8 patients) had congestive heart failure, 6% (3 patients) had arrhythmia, 2% (1 patient) had cardiogenic shock and 2% (1 patient) died (Table 4).

		Low T3 syndrome	Euthyroid	
Variables		(N=4)	(N=42)	P-value
Age (years)		48.5 ± 4.8	53.9 ± 5.7	0.073
	Male	2 (50.0%)	24 (57.1%)	
Sex	Female	2 (50.0%)	18 (42.9%)	1
Smoking		3 (75.0%)	14 (33.3%)	0.135
Hypertension		3 (75.0%)	22 (52.4%)	0.614
Diabetes mellitus		1 (25.0%)	5 (11.9%)	0.44
Dyslipidemeia		1 (25.0%)	13 (31.0%)	1
	Chest pain	4 (100.0%)	35 (83.3%)	
Presentations	Epigastric pain	0 (0.0%)	4 (9.5%)	&1.000
	Dyspnea	0 (0.0%)	3 (7.1%)	
	Anterior	3 (75.0%)	22 (52.4%)	
STEMI	Inferior	1 (25.0%)	20 (47.6%)	&0.614
Systolic Blood Pressure (mmHg)		118.5 ± 4.0	126.0 ± 18.4	^0.427
Diastolic Blood Pressure (mmHg)		77.8 ± 5.3	82.4 ± 12.9	^0.483
Heart Rate (beat/minute)		81.0 ± 3.9	78.1 ± 11.6	^0.628
Ejection Fraction %		30.3 ± 6.2	52.2 ± 7.7	<0.001*
LVESD (cm)		5.4 ± 0.5	3.8 ± 0.5	<0.001*
LVEDD (cm)		6.4 ± 0.5	5.2 ± 0.5	<0.001*
Left atrial diameter (cm)		4.6 ± 0.1	3.8 ± 0.5	<0.001*
Total cholesterol (mg/dL)		229.1 ± 91.0	215.5 ± 58.2	0.672
Triglycerides (mg/dL)		184.0 ± 104.2	165.1 ± 66.7	0.607
LDL (mg/dL)		148.5 ± 82.3	140.1 ± 53.3	0.775
HDL (mg/dL)		43.8 ± 12.2	42.4 ± 8.8	0.774
Creatine Kinase (IU/L)		2536.5 ± 382.5	1495.9 ± 332.2	<0.001*
Creatine Kinase-Myocardial Band (IU/L)		301.0 ± 21.2	205.1 ± 52.3	<0.001*
Troponin-I (ng/mL)		57.0 ± 4.1	32.2 ± 10.9	<0.001*
C-Reactive Protein (mg/dL)		58.0 ± 4.2	40.5 ± 4.6	<0.001*
Post infarction angina		2 (50.0%)	3 (7.1%)	0.053
Congestive Heart Failure		3 (75.0%)	3 (7.1%)	0.005*
Arrhythmia		1 (25.0%)	2 (4.8%)	0.224
Shock		1 (25.0%)	0 (0.0%)	0.087
Death		1 (25.0%)	0 (0.0%)	0.087

Table 4: Comparison between low T3 syndrome and euthyroid patients regarding study variables

Our study demonstrated that low T3 is an important marker of the severity of the illness and predicted mortality in CCU patients and that was concordant with Kumar KV, et al. [14] prospective observational study that was conducted on 100 consecutive patients (52 M; 48 F).

Our recorded data suggested increased serious events were associated with low T3 syndrome and that was concordant with Coceani M, et al. [15] and Ozcan KS, et al. [16].

In our study, we found that-out of 50 patients-8 patients (16%) were hypothyroid; 4 of them (8%) had low T3 syndrome, 3 patients (6%) were subclinical hypothyroid and 1 patient (2%) had clinical hypothyroidism. In contrast, WANG Wen-yao et al. [17] was conducted on 582 patients over two years, found that there were 76 patients (13.06%) who had hypothyroidism including low T3 syndrome (34 patients, 5.84%), subclinical hypothyroidism (28 patients, 4.81%) and clinical hypothyroidism (14 patients, 2.41%). Rodrigo Caetano Pimentel et al. [18] was conducted on 70 patients presented with acute coronary syndrome, 13 patients (18.6%) had low T3 syndrome. Faiza Abdulaziz Qari et al. [19] was conducted on 400

patients presented with acute coronary syndrome. Out of these 400 patients overall hypothyroidism prevalence was 7.8%, while subclinical hypothyroidism in our study was 2.7%. Overt hyperthyroidism and subclinical hyperthyroidism was reported 2.0% and 0.5%, respectively. euthyroid sick syndrome was noticed in 41 patients (10.2%).

In our study, we found that STEMI with or without thyroid disease did NOT correlate with demographic parameters such as age, sex, hypertension, smoking, dyslipidemia or diabetes mellitus at presentation that was concordant with WANG Wen-yao et al. [17]. In contrast, Iervasi G, et al. [20], a study conducted on 573 cardiac patients, stated that there was statistically significant relation with age (p value 0.003), diabetes mellitus (p value 0.046) and dyslipidemia (p value 0.0057). In our study hypothyroidism was slightly more common among females than males while in Faiza Abdulaziz Qari et al. [19], hypothyroidism were four times more common among females (Table 5).

	FT3		FT4		TSH	
Variables	R	p	R	P	r	p
Age (years)	0.12	0.408	0.051	0.725	-0.02	0.89
SBP (mmHg)	0.1	0.492	-0.129	0.373	-0.129	0.372
DBP (mmHg)	0.04	0.785	-0.142	0.326	-0.119	0.411
HR (beat/min)	0.225	0.117	0.181	0.208	-0.093	0.523
EF %	0.385	0.006*	-0.042	0.771	-0.305	0.031*
LVESD (cm)	-0.375	0.007*	0.068	0.641	0.318	0.025*
LVEDD (cm)	-0.366	0.009*	0.008	0.955	0.293	0.039*
LAD (cm)	-0.284	0.046*	0.006	0.968	0.247	0.083
Cholesterol (mg/dl)	0.182	0.205	0.047	0.747	-0.071	0.624
Triglycerides (mg/dl)	0.187	0.194	0.061	0.672	-0.048	0.74
LDL (mg/dl)	0.181	0.21	0.038	0.796	-0.08	0.58
HDL (mg/dl)	-0.171	0.234	-0.008	0.956	0.089	0.54
CK (IU/L)	-0.328	0.020*	-0.064	0.66	0.259	0.07
CK-MB (IU/L)	-0.294	0.038*	-0.028	0.848	0.201	0.161
Troponin-I (ng/mL)	-0.368	0.009*	-0.017	0.905	0.25	0.08
CRP (mg/dL)	-0.404	0.004*	0.047	0.748	0.342	0.015*

Table 5: Correlation between thyroid profile and other study variables

Among our group, 12% (6 patients) had post infarction angina, 16% (8 patients) had congestive heart failure, 6% (3 patients) had arrhythmia, 2% (1 patient) had cardiogenic shock and 2% (1 patient) died (Figures 7 and 8).

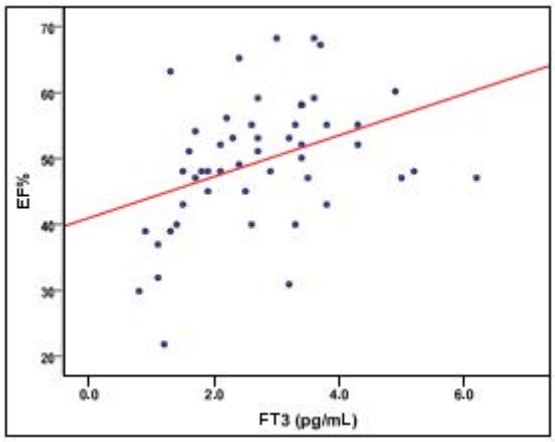

Figure 7: Correlation between FT3 and EF

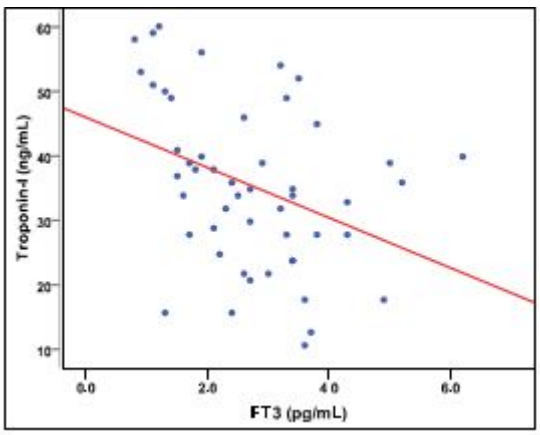

Figure 8: Correlation between FT3 and troponin-I

Our study demonstrated that low T3 is an important marker of the severity of the illness and predicted mortality in CCU patients and that was concordant with Kumar KV, et al. [14] prospective observational study that was conducted on 100 consecutive patients (52 M; 48 F). Our recorded data suggested increased serious events were associated with low T3 syndrome and that was concordant with Coceani M, et al. [15] and Ozcan KS, et al. [16].

In our study, we found that-out of 50 patients-8 patients (16%) were hypothyroid; 4 of them (8%) had low T3 syndrome, 3 patients (6%) were subclinical hypothyroid and 1 patient (2%) had clinical hypothyroidism. In contrast, WANG Wen-yao et al. [17] was conducted on 582 patients over two years, found that there were 76 patients (13.06%) who had hypothyroidism including low T3 syndrome (34 patients, 5.84%), subclinical hypothyroidism (28 patients, 4.81%) and clinical hypothyroidism (14 patients, 2.41%). Rodrigo Caetano Pimentel et al. [18] was conducted on 70 patients presented with acute coronary syndrome, 13 patients (18.6%) had low T3 syndrome. Faiza Abdulaziz Qari et al. [19] was conducted on 400 patients presented with acute coronay syndrome. Out of these 400 patients overall hypothyroidism prevalence was 7.8%, while subclinical

hypothyroidism in our study was 2.7%. Overt hyperthyroidism and subclinical hyperthyroidism was reported 2.0% and 0.5%, respectively. euthyroid sick syndrome was noticed in 41 patients (10.2%).

In our study, we found that STEMI with or without thyroid disease did NOT correlate with demographic parameters such as age, sex, hypertension, smoking, dyslipidemia or diabetes mellitus at presentation that was concordant with WANG Wen-yao et al. [17]. In contrast, Iervasi G, et al. [20], a study conducted on 573 cardiac patients, stated that there was statistically significant relation with age (p value 0.003), diabetes mellitus (p value 0.046) and dyslipidemia (p value 0.0057). In our study hypothyroidism was slightly more common among females than males while in Faiza Abdulaziz Qari et al. [19], hypothyroidism were four times more common among females.

In our study, we assessed myocardial injury by the level of cardiac biomarkers and echocardiography (EF, LVESD, LVEDD, and LAD), this was concordant with WANG Wen-yao et al. [17] that also used the same parameters to assess myocardial injury. In contrast, Ceremuzynski L, et al. [21] also suggested that FT3 was correlated with cardiac injury assessed by echocardiography but authors took the synergic area (AA) and wall motion score index (WMSI) as the parameters for cardiac injury.

In our study, we used cardiac biomarkers (troponin, cpk and ckmb) as a marker for severity of myocardial injury and that was concordant with Setiadi BM, et al. [22] and Kavsak PA, et al. [23].

In our study, there was significant increase in cardiac biomarkers (CKMB, CPK, Troponin) in patients with low T3 syndrome and that was concordant with WANG Wen-yao et al. [17].

As an inflammatory marker, increased level of CRP is frequent in the acute phase of MI. Free T3 showed a negative correlation with CRP with P value 0.004, which indicated that hypothyroidism might activate the inflammation response and it might partly be the reason why the worse the hypothyroidism is, the worse the myocardial injury is and that was concordant with WANG Wen-yao et al. [17]. Also in Stumpf C, et al. [24], a study conducted on 81 STEMI patients, found that CRP increase significantly in acute setting of STEMI and it was negatively correlated with EF.

In our study, we found that lower level of T3 correlated with more decrease in EF (p value=0.006) and more increase in cardiac dimensions detected by echocardiography; LVESD (p value=0.007), LVEDD (p value=0.009) and LAD (p value 0.046), that was concordant with Iervasi G [20], which stated that the mean EF in euthyroid patients was 52.0 ± 14 and mean EF in low T3 syndrome group was 46.2 ± 14.1.

Conclusion

Low T3 syndrome is considered a predictor for myocardial injury in STEMI patients, high incidence of complications and poor prognosis. Lower Free T3 level in patients presenting with acute STEMI, correlates with higher level of cardiac markers, CRP and lower left ventricular ejection fraction.

Recommendations

Measuring free T3 in other ACS patients such as unstable angina and Non-STEMI and then comparing their values with our study values to reach maximum benefit.

Compare between STEMI patients who receive thrombolytic therapy and those who had primary PCI regarding free T3 level and its implications on myocardial injury.

Investigate whether reversing the hypothyroidism could benefit the STEMI patients or not. Also increase sample size and add more variables.

Limitations

First, the study included only STEMI patients and excluded Non-ST segment Elevation Acute Coronary Syndrome (NSTE-ACS). Second, relatively small sample size of this study. Third, the results were obtained from only one center (Matareya Teaching Hospital). Fourth, different echocardiographers with variable skills.

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