

Thyroid Profile in Patients with Acute Coronary Syndrome

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Abstract

Original Research Article

Background: Thyroid hormone has the major role in the cardiovascular system function and cardiac a As well as to maintain the cardiovascular homeostasis. A slightly change in thyroid status actually affects cardiovascular mortality hemodynamic. The background of this study was to define the prevalence of thyroid dysfunction in acute coronary syndrome (ACS). **Objective:** To assess thyroid hormonal profile in patients presenting with acute coronary syndromes (ACS). **Methods:** A hospital based, retrospective, observational comparative study was carried out at the Dept. of Cardiology, Bangabandhu Sheikh Mujib Medical College and Hospital, Faridpur, Bangladesh from January to June 2020. Data of all patients with acute coronary syndrome presenting to hospital were collected in a pre-structured proforma and analyzed. **Results:** A total of 100 ACS patients between 21 years to 75 years with mean age of 59.23 ± 11.30 years were studied. One hundred and twenty seven (64.0%) were males. Among them 58 (58%) was ST segment elevated myocardial infarction (STEMI) patients while 42 (42%) were unstable angina/ non-ST elevated myocardial infarction (UA/ NSTEMI) patients. Total 23 (23.0%) patients had abnormal TFT of which 14(60.8%) had Euthyroid Sick Syndrome, 6(26.0%) had subclinical hypothyroidism, 3(13.0%) had subclinical hyperthyroidism and 1(4.2%) had low fT4 with normal fT3 and normal TSH. There was significant difference in TFT in patients with STEMI and UA/NSTEMI ($P=0.006$). There were higher rates of heart failure ($p= 0.001$ & 0.003 in STEMI & UA/NSTEMI respectively), longer length of hospital stay ($3+0.17$ days) and high mortality (more than 4 fold) in all types of ACS patients with abnormal TFT than ACS patients with normal TFT. **Conclusion:** There is higher prevalence of abnormal thyroid hormonal findings in ACS causing significant morbidity and mortality.

Keywords: Prevalence, Acute Coronary Syndrome, Prognosis, Thyroid Hormone Profile.

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INTRODUCTION

Thyroid hormone has a major role in the cardiovascular system function and cardiac hemodynamic [1], as well as to maintain the cardiovascular homeostasis [2]. In recent years, increased attention has been focused on various circulating biologically active substances, collectively known as plasma biomarkers, and their utility in coronary artery disease (CAD) and heart failure (HF) prognosis. 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]. Clinical sign characteristics of hyperthyroidism like tachycardia,

higher cardiac output, myocardial contractility, systolic blood pressure, and basal metabolism, as well as tremor suggest a hyperadrenergic state. This is all due to the sensitivity to catecholamine compounds [4, 5]. Nevertheless, the relation between anomalous thyroid function and cardiovascular effects remains indistinct [6]. Hypothyroidism instead, seems to evoke a hypoadrenergic state due to the presence of bradycardia, reduced basal metabolism and cardiac output, and the intracellular catecholamine production from circulation, which has been found to be lower during hypothyroidism [7, 8]. The subclinical hypothyroidism and hyperthyroidism have recently been documented as clinical entities with negative effects on the cardiovascular system [9]. Subclinical hypothyroidism

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is categorized by normal serum levels of FT4 and FT3, and slightly elevated serum thyrotropin (TSH) concentrations. This condition is associated with an initial reduced systolic function, diastolic hypertension, increased systemic vascular resistance, an atherogenic lipid profile, and inflammatory condition [10]. Subclinical hyperthyroidism is related to an increased risk of supraventricular arrhythmias, hypercoagulable state, and a mild decrease of coronary reserve. 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.

MATERIALS AND METHODS

A hospital based, retrospective, observational comparative study was carried out at the Dept. of Cardiology, Bangabandhu Sheikh Mujib Medical College and Hospital, Faridpur, Bangladesh from January to June 2020. Data of all patients with acute coronary syndrome presenting to hospital were collected in a pre-structured proforma and analyzed. 100 consecutive cases of acute coronary syndromes were taken for the study. Depending on the ECG findings and result of cardiac markers, the patients of ACS were categorized into following two groups as per American Heart Association (AHA) criteria. Group 1 considered cases showing ST depression / T wave inversion with normal or elevated cardiac markers. At the time of presentation, patients with UA and NSTEMI can be indistinguishable and therefore are considered together in these guidelines [11]. The diagnosis of NSTEMI is established if a patient with the clinical features of UA develops evidence of myocardial necrosis, as reflected in elevated cardiac biomarkers [12], and Group 2 included STEMI patients having symptoms of myocardial ischemia in association with electrocardiographic (ECG) ST elevation and release of biomarkers of myocardial necrosis.

Data were collected in a preformed proforma and analyzed in SPSS software version 16. The

significant difference between two groups was compared using ANOVA. Pearson’s correlation coefficient, Chi-square test, t-tests, etc. was used to find group association. Odds ratio was calculated for required appropriate values and p values were considered significant at a predetermined level of <0.05.

RESULTS

Out of One hundred (100) ACS patients between 21 years to 75 years with mean age of 59.32 ± 11.30 years were studied. 57 (57.0%) were more than 60 years, 35 (35.0%) were 40-60 years and 8 (8.0%) belonged to age group of 20-40 years. 64 (64.0%) were males. 58 (58%) were STEMI patients while 42 (42%) were UA/ NSTEMI patients. 38 (39%) patients were hypertensive and on medications while only 11 (11%) were diabetics. 67 (67%) patients were smokers. Thyroid hormone analysis was done in all the ACS patients and was found abnormal in 23 (23.0%) of the patients. Out of 64 males and 36 females with ACS, 13 (20.3%) patients had abnormal TFTs while 10 (27.7%) female patients with ACS had abnormal TFTs. Of the 23 patients with abnormal thyroid hormone profile, 14(60.8%) had Euthyroid Sick Syndrome, 6(26.0%) had subclinical hypothyroidism, 3(13.0%) had subclinical hyperthyroidism and 1(4.2%) had low ft4 with normal ft3 and normal TSH. Table 1 further divides the findings in subcategories of STEMI and UA/NSTEMI. Table 2 shows the comparison of abnormal TFT reports in patients with STEMI and UA/NSTEMI patients. It shows a statistical significant difference in TFT profile in these two groups. The above table (Table 3) shows that there was significant difference in patients with abnormal TFT reports presenting or having heart failure both in STEMI or UA/STEMI groups. Further analysis in mean average hospital stay and mortality with abnormal thyroid hormone profiles in these patients was analyzed. The mean hospital stay was significantly higher ($p < 0.001$) in both the groups (Table 4). Mortality was slightly higher in patients having abnormal TFT reports but not significant in both the groups ($p = 0.164$ and 0.1429 respectively) (Table 5).

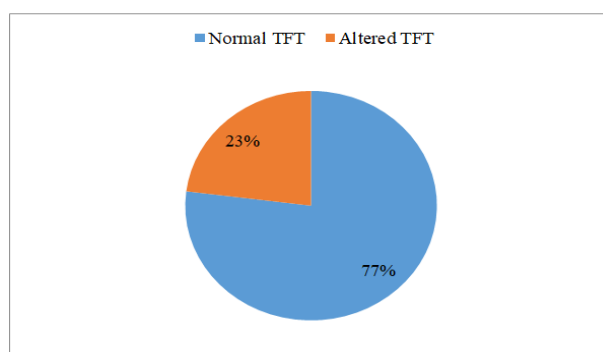


Figure 1: Thyroid hormone in pts with ACS

Table 1: Pattern of abnormal thyroid profile in patients with different categories (N=100)

Category	No of pts with Euthyroid sick syndrome	No of pts with subclinical hypothyroidism	No of pts with subclinical hyperthyroidism	No of pts with low fT4 but normal fT3 and TSH	Total pts with abnormal TFTs
STEMI	10	4	2	1	17
NSTEMI	4	2	1	0	6

Table 2: Comparison of abnormal TFT reports in patients with STEMI and UA/NSTEMI (N=100)

Category	Number of pts with normal TFT reports	Number of pts with abnormal reports	Odds ratio	p-value(Fisher exact probability test)
STEMI	41	17	2.59	0.006
UA/NSTEMI	36	6		

Table 3: Analysis of ACS patients with heart failure and abnormal thyroid (N=100)

Category	TFT reports	Number of patients with heart failure	Number of patients without heart failure	P-value(Fisher exact probability test)
STEMI	Normal	4	36	0.001
	Abnormal	6	12	
UA/NSTEMI	Normal	1	35	0.003
	Abnormal	2	4	

Table 4: Analysis of ACS patients of different thyroid status with average length of hospital stay (N=100)

Category	TFT reports	Mean average hospital stay (in days)	Mean difference (95% CI)	P-value
STEMI	Normal(n=41)	6+0.7	-3.0(-3.56 to 2.45)	<0.001
	Abnormal(n=17)	9+2.3		
UA/NSTEMI	Normal(n=36)	5+1.1	-2.0(-2.79to -1.22)	<0.001
	Abnormal(n=6)	7+2.01		

Table 5: Analysis of ACS patients of different thyroid status with average length of hospital day (N=100)

Category	TFT Report	Number of patients died	Number of patients survived	OR(95% CI)	P value*
STEMI	Normal	1	39	3.65(0.583 to 22.928)	0.164
	Abnormal	2	16		
UA/NSTEMI	Normal	0	36	NA	0.1429
	Abnormal	1	5		

DISCUSSION

Acute coronary syndrome is a condition that should be taken very serious because of its significant effect on thyroid gland homeostasis with repercussions in terms of morbidity and mortality. Patients with severe nonthyroidal illness often experience concomitant disorders in thyroid function. In severe illness of nonthyroidal origin including Existing proof has supported the assumption of a prognostic role for the low T3 syndrome in patients with ACS. Several clinical studies have been done to investigate the prognostic value of THs alterations in patients suffering from ACS. Alteration in the level of serum thyroid hormone profile has been described in various several non-thyroidal systemic illnesses including acute heart diseases in otherwise euthyroid patients which is called as “euthyroid sick syndrome” [4]. Present study assesses thyroid profile in ACS and compare between the ACS sub-groups. Our study showed abnormal thyroid hormonal findings in 23(23.0%) of patients with ACS. This result was comparable to various other

studies in other part of the world too. For example a study of 400 patients of ACS by Qari FA, thyroid dysfunction was reported in 23.0% of patients [13]. Similarly Khalil OA *et al.*, in their study of 196 patients of ACS, reported changes in thyroid hormone profile in 23% of their patients [14]. Mathur P *et al.*, in their study of 85 patients and Bayrak A *et al.*, in their study of 110 patients of ACS reported changes in thyroid hormone profile in 31.7% and 23.6% of patients respectively [15]. Thyroid dysfunction in acute coronary syndrome increases the relative risk of death by 5.49 fold than euthyroid patients [13, 14]. This study is done to see the status of thyroid profile in patients with ACS in our part of world. Our study showed a higher prevalence of abnormal thyroid hormone profile was seen in patients of STEMI group as compared to NSTEMI/UA group being 17 out of 23 (73.9%) in STEMI group and 6 out of 23 (26.1%) in NSTEMI/UA group. The difference is statistically significant (p=0.006). Similarly there was high prevalence of different thyroid patterns like euthyroid sick syndrome, subclinical hypothyroidism or hyperthyroidism and low fT4 but normal TSH and fT3

in STEMI group than UA/NSTEMI. These results are comparable to studies done before [13-15]. Takada K *et al.*, in year 1994 in their study of relationship of thyroid function and left ventricular function in Acute Myocardial Infarction in 52 patients admitted to Coronary Care Unit within 24 hours after the onset observed that non-survivors showed significantly lower levels of fT3 and fT4 48 hours after onset, and concluded that measurement of thyroid hormone in AMI is important in evaluating the severity of the condition and making a prognosis [16]. Our study also showed higher rates of heart failure ($p= 0.001$ & 0.003 in STEMI & UA/NSTEMI respectively), longer length of hospital stay and high mortality in all types of ACS patients with abnormal thyroid profiles. In STEMI patients undergoing PCI it was observed that low fT3 was an independent marker for MACE [17]. A small sample study has demonstrated an association of abnormal THs alterations with worse prognosis [8]. Short term and long-term mortality have been related with the low T3 syndrome in patients undergoing primary percutaneous coronary angioplasty for STEMI [18].

CONCLUSION

Alterations in thyroid function tests are common in patients with ACS, especially in STEMI patients. The low T3 syndrome represents a hormonal imbalance that may significantly influence pathophysiological mechanisms and cardiovascular hemodynamics. There is higher prevalence of abnormal thyroid hormonal findings in ACS causing significant morbidity and mortality. As a future direction, thyroid hormone profile done at the time of admission in patients with ACS may be used as a marker of prognosis along with other established scores or biochemical markers.

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