Evaluation of Thyroid Profile as a Predictor of Poor Prognosis in Acute Coronary Syndrome Patients – A Study of 120 Cases

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ABSTRACT

Background: Thyroid hormones have significant cardiovascular effects, and alterations in thyroid function are frequently observed in critically ill patients, including those with acute coronary syndrome (ACS). Low triiodothyronine (T3) syndrome and subclinical thyroid dysfunction have been associated with adverse cardiovascular outcomes. This study aimed to evaluate the role of thyroid profile as a predictor of poor prognosis in patients presenting with ACS. **Objectives:** To assess the prevalence of thyroid function abnormalities in ACS patients and determine their association with short-term in-hospital outcomes. **Methods & Materials:** This prospective observational study included 120 consecutive patients diagnosed with ACS. Thyroid profile (TSH, free T3, free T4) was measured within 24 hours of admission. Patients were followed during hospital stay for major adverse cardiac events (MACE) including mortality, cardiogenic shock, heart failure, arrhythmias, and recurrent ischemia. Statistical analysis was performed to assess associations between thyroid abnormalities and outcomes. **Results:** Thyroid dysfunction was detected in 46.7% of patients, with low T3 syndrome being the most common abnormality (31.7%). Patients with abnormal thyroid profiles had significantly higher rates of in-hospital MACE ($p < 0.05$), including mortality and heart failure. Low T3 levels were independently associated with poor prognosis. **Conclusion:** Abnormal thyroid profile, particularly low T3 syndrome, is common in ACS patients and is associated with worse in-hospital outcomes. Thyroid hormone assessment may serve as a useful prognostic marker in ACS.

Keywords: *Acute Coronary Syndrome, Thyroid Profile, Low T3 Syndrome, Prognosis, Cardiovascular Outcomes.*

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Introduction

Acute coronary syndrome (ACS), which includes ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina, remains a major cause of morbidity and mortality worldwide. Despite substantial advances in reperfusion strategies, pharmacotherapy, and secondary prevention, early risk stratification continues to be a cornerstone in the management of ACS. Accurate identification of high-risk patients allows timely intervention, optimal utilization of resources, and improved clinical outcomes. Conventional prognostic indicators include age, hemodynamic status, left ventricular function, cardiac biomarkers, and electrocardiographic changes; however, these parameters may not fully reflect the complex systemic response associated with acute myocardial ischemia. Thyroid hormones play a critical role in cardiovascular physiology. Triiodothyronine (T3), the biologically active thyroid hormone, regulates myocardial contractility, heart rate, diastolic relaxation, systemic vascular resistance, and lipid metabolism. At the cellular level, T3 modulates the expression of genes involved in calcium handling and myocardial energy utilization. Consequently, even subtle alterations in thyroid hormone levels can have significant cardiovascular consequences. In patients with acute illness, abnormalities in thyroid function tests are frequently observed in the absence of pre-existing thyroid disease, a condition commonly referred to as euthyroid sick syndrome or low T3 syndrome. Low T3 syndrome is characterized by reduced serum T3 levels with normal or low-normal thyroxine (T4) and thyroid-stimulating hormone (TSH) concentrations. This condition has been widely reported in critically ill patients, including those with sepsis, stroke, heart failure, and acute myocardial infarction. The underlying mechanisms are multifactorial and include impaired peripheral conversion of T4 to T3 due to reduced deiodinase activity, increased inflammatory cytokines, altered binding of thyroid hormones to plasma proteins, and dysregulation of the hypothalamic-pituitary-thyroid axis during acute stress [1,2]. Several studies have demonstrated that low T3 syndrome is associated with increased disease severity and poor clinical outcomes in cardiovascular disorders. In the setting of ACS, reduced T3 levels may exacerbate myocardial dysfunction, promote adverse ventricular remodeling, impair endothelial function, and increase the risk of arrhythmias and heart failure [3]. Subclinical thyroid dysfunction, particularly subclinical hypothyroidism, has also been implicated in the progression of atherosclerosis and adverse cardiovascular events [4]. Although growing evidence suggests a potential prognostic role of thyroid hormone abnormalities in ACS, routine assessment of thyroid profile is not universally practiced, and data from South Asian populations remain limited. Given the high burden of coronary artery disease in this region and the biological plausibility linking thyroid hormones to cardiovascular outcomes, further evaluation is warranted. The present study was therefore designed to assess thyroid function status in patients presenting with acute coronary syndrome and to evaluate the role of thyroid profile, particularly low T3 syndrome, as a predictor of poor in-hospital prognosis.

Methods & Materials

Study Design and Setting

This was a prospective observational study conducted in the Department of Cardiology, Faridpur Medical College and Hospital from July 2024 to June 2025.

Study Population

A total of 120 consecutive patients admitted with a diagnosis of ACS over the study period were included.

Inclusion Criteria

- Age ≥ 18 years
- Diagnosis of ACS (ST-elevation myocardial infarction [STEMI], non-ST elevation myocardial infarction [NSTEMI], or unstable angina) based on clinical features, ECG changes, and cardiac biomarkers

Exclusion Criteria

- Known thyroid disease or current thyroid hormone therapy
- Use of drugs affecting thyroid function (e.g., amiodarone, steroids)
- Severe hepatic or renal failure
- Sepsis or chronic inflammatory disease

Data Collection

Demographic data, cardiovascular risk factors, clinical presentation, ECG findings, echocardiographic parameters, and laboratory investigations were recorded. Thyroid function tests (TSH, free T3, free T4) were measured within 24 hours of admission.

Definitions

- **Low T3 syndrome:** Reduced free T3 with normal TSH and free T4
- **Subclinical hypothyroidism:** Elevated TSH with normal free T3 and free T4
- **Poor prognosis:** Occurrence of in-hospital MACE (death, cardiogenic shock, acute heart failure, life-threatening arrhythmias, or recurrent ischemia).

Statistical Analysis

Data were analyzed using standard statistical software SPSS v25. Continuous variables were expressed as mean ± SD and categorical variables as percentages. Comparisons were made using Student's t-test or chi-square test as appropriate. Multivariate logistic regression analysis was used to identify independent predictors of poor prognosis. A p-value < 0.05 was considered statistically significant.

Results

The study population consisted of 120 patients with acute coronary syndrome. The mean age was 56.4 ± 10.8 years, with males comprising 71.7% of the total cohort, indicating a marked male predominance. Hypertension (56.7%) and smoking (51.7%) were the most prevalent cardiovascular risk factors, followed by diabetes mellitus (45.0%) and dyslipidemia (40.8%). Regarding the clinical spectrum of acute coronary syndrome, ST-elevation myocardial infarction (STEMI) was the most common presentation (54.2%), while NSTEMI and unstable angina accounted for 28.3% and 17.5% of cases respectively (*Table I*).

Table I
Baseline Demographic and Clinical Characteristics of the Study Population (*n* = 120).

Variable	Number (%) / Mean ± SD
Age (years)	56.4 ± 10.8
Sex	
- Male	86 (71.7%)
- Female	34 (28.3%)
Cardiovascular Risk Factors	
- Hypertension	68 (56.7%)
- Smoking	62 (51.7%)
- Diabetes mellitus	54 (45.0%)
- Dyslipidemia	49 (40.8%)
Clinical Presentation (ACS type)	
- STEMI	65 (54.2%)
- NSTEMI	34 (28.3%)
- Unstable angina	21 (17.5%)

Normal thyroid function (euthyroid state) was observed in 53.3% of patients. Thyroid dysfunction was present in 46.7% of cases, with low T3 syndrome being the most common abnormality (31.7%). Subclinical hypothyroidism was detected in 10.0% of patients, while subclinical hyperthyroidism was observed in 5.0%, indicating that thyroid hormone alterations are frequent during the acute phase of coronary events (*Table II*).

Table II
Thyroid Profile Distribution in ACS Patients.

Thyroid Status	Number (n)	Percentage (%)
Euthyroid	64	53.3
Low T3 syndrome	38	31.7
Subclinical hypothyroidism	12	10.0
Subclinical hyperthyroidism	6	5.0

Overall, 28.3% of patients developed at least one MACE during hospitalization. Acute heart failure was the most frequent complication (24.2%), followed by significant arrhythmias (17.5%) and cardiogenic shock (15.0%). Recurrent ischemia occurred in 13.3% of patients. The overall in-hospital mortality rate was 11.7%, reflecting substantial disease severity in the study population (*Table III*).

Table III
In-Hospital Outcomes (Major Adverse Cardiac Events).

Outcome	Total (n=120)	Percentage (%)
Mortality	14	11.7
Acute heart failure	29	24.2
Cardiogenic shock	18	15.0
Significant arrhythmias	21	17.5
Recurrent ischemia	16	13.3
Any MACE	34	28.3

Patients with euthyroid status had a significantly lower incidence of adverse events (15.6%) compared to those with thyroid dysfunction. The highest rate of MACE was observed in patients with low T3 syndrome, where 50.0% experienced adverse outcomes, showing a statistically significant association ($p < 0.01$). Subclinical hypothyroidism was also associated with increased MACE (33.3%), whereas subclinical hyperthyroidism did not show a statistically significant relationship with poor outcomes (*Table IV*).

Table IV
Association Between Thyroid Status and In-Hospital MACE.

Thyroid Status	MACE Present n (%)	MACE Absent n (%)	p-value
Euthyroid (n=64)	10 (15.6%)	54 (84.4%)	
Low T3 syndrome (n=38)	19 (50.0%)	19 (50.0%)	<0.01
Subclinical hypothyroidism (n=12)	4 (33.3%)	8 (66.7%)	0.04
Subclinical hyperthyroidism (n=6)	1 (16.7%)	5 (83.3%)	NS

Low T3 syndrome emerged as a strong independent predictor of adverse in-hospital outcomes (OR 3.6; 95% CI 1.8–7.2; $p < 0.001$). Reduced left ventricular ejection fraction (<40%) and STEMI presentation were also independently associated with poor prognosis. Age above 60 years showed a modest but significant association, while male sex did not independently predict adverse outcomes (*Table V*).

Table V
Multivariate Logistic Regression Analysis for Predictors of Poor Prognosis.

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Age > 60 years	1.9	1.1 – 3.6	0.03
Male sex	1.3	0.7 – 2.8	NS
STEMI	2.4	1.3 – 4.5	0.01
LVEF < 40%	3.1	1.6 – 6.0	<0.001
Low T3 syndrome	3.6	1.8 – 7.2	<0.001

Discussion

The present study highlights the high prevalence of thyroid hormone abnormalities in patients admitted with acute coronary syndrome and demonstrates a significant association between altered thyroid profile and adverse in-hospital outcomes. Nearly half of the patients exhibited some form of thyroid dysfunction, with low T3 syndrome emerging as the most common abnormality. These findings reinforce the growing body of evidence suggesting that thyroid hormone alterations are not merely biochemical changes but are clinically relevant markers of disease severity in ACS. The predominance of low T3 syndrome observed in this study is consistent with previous reports in patients with acute myocardial infarction and other critical illnesses [3,5]. Acute stress, systemic inflammation, and reduced activity of type-1 deiodinase during myocardial ischemia result in decreased peripheral conversion of T4 to T3, leading to low circulating T3 levels. Since T3 is essential for maintaining myocardial contractility and vascular tone, its deficiency may contribute directly to hemodynamic instability and impaired cardiac performance during the acute phase of ACS. In the present study, patients with low T3 syndrome experienced significantly higher rates of major adverse cardiac events, including acute heart failure, cardiogenic shock, and mortality. Similar findings were reported by Iervasi et al., who identified low T3 as a strong independent predictor of mortality in patients with heart disease [6]. Reduced T3 levels have been associated with impaired left ventricular systolic function, increased systemic vascular resistance, and unfavorable ventricular remodeling, all of which adversely influence prognosis following an acute coronary event [7]. Subclinical hypothyroidism was also associated with a higher incidence of adverse outcomes, although the strength of association was less pronounced than that observed with low T3 syndrome. Even mild thyroid hormone deficiency has been linked to endothelial dysfunction, pro-atherogenic lipid abnormalities, and increased cardiovascular risk [4,8]. In contrast, subclinical hyperthyroidism did not show a significant association with poor prognosis in the current study, which may be attributable to the relatively small number of affected patients. Multivariate logistic regression analysis confirmed that low T3 syndrome remained an independent predictor of poor in-hospital prognosis after adjusting for established risk factors such as age, type of ACS, and left ventricular ejection fraction. Reduced left ventricular ejection fraction and STEMI presentation were also identified as strong independent predictors, consistent with previously published literature [9]. These findings suggest that assessment of thyroid profile, particularly free T3 levels, may

provide incremental prognostic information beyond traditional risk markers. The clinical implications of this study include the potential utility of routine thyroid function testing for early risk stratification in ACS patients. However, the role of thyroid hormone replacement therapy in patients with euthyroid sick syndrome remains controversial. Current evidence does not support routine treatment, and large randomized controlled trials are required to determine whether correcting low T3 levels can improve clinical outcomes [10]. This study has limitations, including its single-center design, modest sample size, and short-term follow-up. Thyroid function was assessed only during the acute phase of illness, and long-term outcomes were not evaluated. Nevertheless, the findings provide meaningful insight into the prognostic significance of thyroid hormone abnormalities in ACS. In conclusion, low T3 syndrome is common in ACS patients and serves as an independent predictor of poor in-hospital outcomes, highlighting its potential role as a valuable prognostic biomarker.

Limitations

- Single-center study with a relatively small sample size
- Short-term (in-hospital) follow-up only
- Thyroid function was assessed only once during acute illness

Conclusion

Thyroid hormone abnormalities are common in ACS patients. Low T3 syndrome is significantly associated with poor in-hospital prognosis and may serve as an independent predictor of adverse outcomes. Incorporating thyroid profile assessment into routine evaluation of ACS patients may enhance risk stratification.

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References

1. Klein I, Danzi S. Thyroid disease and the heart. Circulation. 2007;116(15):1725–1735.
2. Wartofsky L, Burman KD. Alterations in thyroid function in patients with systemic illness: The “euthyroid sick syndrome”. Endocr Rev. 1982;3(2):164–217.
3. Iervasi G, Pingitore A, Landi P, et al. Low-T3 syndrome: A strong prognostic predictor of death in patients with heart disease. Circulation. 2003;107(5):708–713.
4. Rodondi N, den Elzen WPJ, Bauer DC, et al. Subclinical hypothyroidism and the risk of coronary heart disease and mortality. JAMA. 2010;304(12):1365–1374.
5. Friberg L, Werner S, Eggertsen G, Ahnve S. Rapid down-regulation of thyroid hormones in acute myocardial infarction: Is it cardioprotective in patients with angina? Arch Intern Med. 2002;162(12):1388–1394.
6. Pingitore A, Galli E, Barison A, et al. Acute effects of triiodothyronine replacement therapy in patients with chronic heart failure and low-T3 syndrome: A randomized, placebo-controlled study. J Clin Endocrinol Metab. 2008;93(4):1351–1358.
7. Gerdes AM, Iervasi G. Thyroid replacement therapy and heart failure. Circulation. 2010;122(4):385–393.
8. Biondi B, Cooper DS. The clinical significance of subclinical thyroid dysfunction. Endocr Rev. 2008;29(1):76–131.
9. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Circulation. 2018;138(20): e618–e651.
10. Fliers E, Bianco AC, Langouche L, Boelen A. Thyroid function in critically ill patients. Lancet Diabetes Endocrinol. 2015;3(10):816–825.