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Original Research Article

Comparative Evaluation of Thyroid Hormone Status in Patients with Acute Coronary Syndrome: A Cross-Sectional Study

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Abstract

Background: Acute coronary syndrome (ACS) is a major cause of morbidity and mortality worldwide and includes clinical conditions such as ST-elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI). Thyroid hormones play a significant role in cardiovascular physiology by regulating myocardial contractility, heart rate, vascular resistance, and lipid metabolism. Alterations in thyroid hormone levels are frequently observed during acute systemic illnesses and may occur as part of Euthyroid Sick Syndrome (ESS). These hormonal changes may influence the clinical course and prognosis of patients with ACS.

Aims and Objectives: To evaluate thyroid hormone status in patients with acute coronary syndrome and compare the thyroid profile between STEMI and NSTEMI patients.

Setting and Design: This was a cross-sectional observational study conducted at P.D.U. Government Medical College and Civil Hospital, Rajkot, Gujarat, India.

Materials and Methods: A total of 100 patients diagnosed with acute coronary syndrome were included in the study. Serum levels of free triiodothyronine (fT3), free thyroxine (fT4), and thyroid stimulating hormone (TSH) were measured within 24 hours of hospital admission using the ELISA method. Statistical analysis was performed using Chi-square test and unpaired t-test, and a p-value < 0.05 was considered statistically significant.

Results: Among the 100 ACS patients studied, 67% had normal thyroid function, while 33% showed thyroid dysfunction. The most common abnormality was Euthyroid Sick Syndrome (17%), followed by subclinical hypothyroidism (11%) and subclinical hyperthyroidism (5%). Abnormal thyroid profiles were significantly more frequent in STEMI patients compared to NSTEMI patients ($p = 0.006$). However, comparison of mean fT3, fT4, and TSH levels between STEMI and NSTEMI groups did not show statistically significant differences ($p > 0.05$).

Conclusion: Thyroid dysfunction is relatively common in patients with acute coronary syndrome, with Euthyroid Sick Syndrome being the most frequent abnormality. A significantly higher prevalence of thyroid abnormalities was observed among STEMI patients, suggesting greater physiological stress in these individuals. Routine evaluation of thyroid hormone status in ACS patients may aid in early risk assessment and clinical management.

Keywords: Acute Coronary Syndrome, Thyroid Hormones, Euthyroid Sick Syndrome, ST-elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI), Thyroid Function Tests.

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Introduction

Acute Coronary Syndrome: Acute coronary syndrome (ACS) represents a spectrum of clinical conditions caused by sudden reduction in coronary blood flow leading to myocardial ischemia. [1] It includes ST-elevation myocardial infarction (STEMI), non–ST-elevation myocardial infarction (NSTEMI), and unstable angina, which differ in the degree of coronary artery obstruction and the extent

of myocardial injury. [1] ACS remains one of the leading causes of morbidity and mortality worldwide and continues to pose a major burden on healthcare systems. The most common pathophysiological mechanism underlying ACS is rupture or erosion of an atherosclerotic plaque within the coronary arteries. [1] This event exposes thrombogenic material that triggers platelet aggregation and thrombus formation, ultimately resulting in partial or complete occlusion of the coronary vessel. The consequent reduction in myocardial perfusion leads to ischemia and, if prolonged, irreversible myocardial necrosis. Several established risk factors contribute to the development of ACS, including hypertension, diabetes mellitus, dyslipidemia, smoking, obesity, advanced age, and sedentary lifestyle. Although these factors play a significant role in the pathogenesis of coronary artery disease, they do not fully explain the variability in disease severity and clinical outcomes observed among patients with acute coronary events. Consequently, increasing attention has been directed toward the role of endocrine and metabolic factors in cardiovascular diseases.

Role of Thyroid Hormones in Cardiovascular Function: The thyroid gland plays an essential role in regulating metabolic activity and maintaining cardiovascular homeostasis. [2] The principal hormones produced by the thyroid gland are thyroxine (T4) and triiodothyronine (T3), with T3 being the biologically active form responsible for most physiological effects. [2] Thyroid hormones exert significant influence on the cardiovascular system by regulating heart rate, myocardial contractility, diastolic relaxation, systemic vascular resistance, and cardiac output. [3] Adequate levels of thyroid hormones are necessary for maintaining normal cardiac performance. Even subtle alterations in thyroid hormone concentrations may produce significant changes in cardiovascular physiology. Both hypothyroidism and hyperthyroidism have been associated with adverse cardiovascular effects, including alterations in heart rate, arrhythmias, and changes in vascular resistance, and disturbances in lipid metabolism. [4] These effects highlight the close relationship between thyroid function and cardiovascular health.

Mechanisms of Thyroid Hormone Action on the Heart: Thyroid hormones influence cardiac function through genomic and non-genomic mechanisms. [2,3] Genomic actions occur when triiodothyronine enters cardiomyocytes and binds to nuclear thyroid hormone receptors, thereby regulating the transcription of genes responsible for myocardial contractility and calcium handling. These mechanisms enhance the synthesis of proteins involved in cardiac muscle contraction and relaxation, improving overall myocardial performance.

In addition to genomic effects, thyroid hormones also exert rapid non-genomic actions through interaction with membrane receptors and ion channels. These mechanisms influence electrical conduction in cardiac tissue, regulate heart rate through effects on the sinoatrial node, and contribute to the maintenance of normal cardiac rhythm. Thyroid hormones also promote vasodilation of peripheral blood vessels, thereby reducing systemic vascular resistance and facilitating efficient blood circulation.

Thyroid Hormone Alterations during Acute Illness: In critically ill patients, including those with acute myocardial infarction, abnormalities in thyroid hormone metabolism frequently occur even in the absence of primary thyroid disease. This condition is commonly referred to as Euthyroid Sick Syndrome (ESS) or Non-thyroidal Illness Syndrome.6 ESS is typically characterized by decreased circulating levels of triiodothyronine with normal or low, levels of thyroxine and thyroid-stimulating hormone.

The principal mechanism responsible for this condition is reduced peripheral conversion of T4 to T3 due to decreased activity of deiodinase enzymes. Systemic inflammation, metabolic stress, and the release of cytokines during severe illness interfere with normal thyroid hormone metabolism, leading to alterations in circulating hormone levels. [6]

Pathophysiological Basis of Thyroid Dysfunction in ACS: In patients with acute coronary syndrome, myocardial injury triggers inflammatory and metabolic responses that may significantly affect thyroid hormone metabolism. The release of inflammatory mediators during myocardial ischemia can inhibit deiodinase activity and disrupt the hypothalamic–pituitary–thyroid axis. [5] As a result, circulating levels of active T3 may decrease, while levels of inactive reverse T3 increase. Since T3 plays a crucial role in maintaining myocardial contractility and cardiac output, reduction in circulating T3 levels during acute illness may adversely affect cardiac performance. [6] Several studies have suggested that low T3 levels are associated with increased severity of myocardial injury, impaired left ventricular function, and poorer clinical outcomes in patients with acute coronary syndrome. [7]

Thyroid Disorders and Cardiovascular Risk: In addition to transient hormonal changes during acute illness, previously undiagnosed thyroid disorders may also influence cardiovascular risk.8 Subclinical hypothyroidism has been associated with dyslipidemia, endothelial dysfunction, and

accelerated atherosclerosis, which may contribute to the development of coronary artery disease.9 Conversely, hyperthyroidism may increase sympathetic activity and myocardial oxygen demand, predisposing individuals to tachyarrhythmias and ischemic cardiac events. [10]

Rationale of the Study: Given the significant influence of thyroid hormones on cardiovascular physiology, evaluation of thyroid hormone status in patients with acute coronary syndrome may provide valuable insights into metabolic alterations occurring during acute cardiac events. Understanding the prevalence and pattern of thyroid hormone abnormalities in ACS patients may also help identify individuals at higher risk and improve clinical management strategies.

Materials and Methods

The present study was conducted as a cross-sectional observational study in the Department of Physiology at P.D.U. Government Medical College and Civil Hospital, Rajkot, Gujarat, India. The study protocol received formal approval from the Institutional Ethical Committee, and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki principles.

A total of 100 patients diagnosed with acute coronary syndrome (ACS) who were admitted to the Department of Medicine at Civil Hospital, Rajkot were included in the study. Patients presenting with clinical features suggestive of ACS were evaluated and confirmed based on clinical presentation, electrocardiographic findings, and cardiac biomarker levels. [1]

The patients were subsequently classified into ST-elevation myocardial infarction (STEMI) and non–ST-elevation myocardial infarction (NSTEMI) groups.

A. Inclusion Criteria: All patients diagnosed with acute coronary syndrome (ACS) who provided informed consent were included in the study irrespective of gender, age, race, ethnic background, or clinical severity of the disease. Patients presenting with ST-elevation myocardial infarction (STEMI) or non–ST-elevation myocardial infarction (NSTEMI) and admitted to the hospital within 24 hours of onset of symptoms were considered eligible for inclusion in the study.

B. Exclusion Criteria: Patients with known thyroid disorders, those patients who failed to give consent, or those receiving thyroid hormone replacement therapy, or those taking medications known to influence thyroid function such as amiodarone, lithium, or corticosteroids were excluded from the study. In addition, patients with chronic systemic illnesses such as chronic kidney disease, chronic liver disease, or malignancy, as well as pregnant women, were excluded in order to avoid potential confounding factors that could affect thyroid hormone levels and interfere with the interpretation of results.

C. Procedure and Data Collection: A detailed clinical history and thorough physical examination were performed for all patients at the time of admission. Routine laboratory investigations were carried out as part of the standard diagnostic evaluation for acute coronary syndrome, including complete blood count, blood glucose levels, renal function tests, lipid profile, and cardiac biomarkers. Electrocardiography was also performed to aid in the diagnosis and classification of acute coronary syndrome.

Patients with acute coronary syndrome were categorized into two groups according to American Heart Association (AHA) criteria: non–ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI). [11] Patients were classified as NSTEMI when electrocardiographic findings showed ST-segment depression or T-wave inversion in the absence of persistent ST-segment elevation, along with elevated cardiac biomarkers indicating myocardial necrosis. Patients were classified as ST-elevation myocardial infarction (STEMI) when electrocardiography demonstrated new ST-segment elevation at the J point in at least two contiguous leads measuring ≥2 mm in men or ≥1.5 mm in women in leads V2–V3, or ≥1 mm in other contiguous chest or limb leads, along with elevated cardiac biomarkers such as CK-MB or cardiac troponin. [12] For assessment of thyroid hormone status, venous blood samples were collected from each patient within 24 hours of hospital admission. Serum levels of free triiodothyronine (fT3), free thyroxine (fT4), and thyroid stimulating hormone (TSH) were measured using the enzyme-linked immunosorbent assay (ELISA) method. The obtained thyroid hormone values were interpreted using the standard laboratory reference ranges, which are presented separately in Table 1. Based on these values, patients were categorized as having normal thyroid function, euthyroid sick syndrome, subclinical hypothyroidism, or subclinical hyperthyroidism.

Table 1: Reference ranges for thyroid hormone parameters

Parameter	Normal Range (Used in Study)	ESS	Subclinical Hypothyroidism	Subclinical Hyperthyroidism
fT3	1.4 – 4.2 pg/mL	Low (< 1.4)	Normal	Normal or slightly high
fT4	0.8 – 2.0 ng/dL	Normal	Normal	Normal or high
TSH	0.4 – 6.2 µIU/mL	Normal	High (> 6.2)	Low (< 0.4)

D. Statistical Analysis: All collected data were compiled and analyzed using Statistical Package for the Social Sciences (SPSS) software, version 28.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage.

Comparisons between groups were performed using the independent sample t-test for continuous variables and the Chi-square test for categorical variables. A 95% confidence interval (CI) was used for statistical analysis, and a p-value of less than 0.05 was considered statistically significant.

Results

A total of 100 patients diagnosed with acute coronary syndrome (ACS) were included in the present study. Among these, 40 patients were diagnosed with ST-elevation myocardial infarction (STEMI) and 60 patients were diagnosed with non–ST-elevation myocardial infarction (NSTEMI). The age distribution of the study population showed that the majority of patients belonged to the older age group, with 66% of patients aged more than 60 years, while 34% of patients were between 30 and 60 years of age. The gender distribution indicated a slightly higher prevalence of ACS among males, with 56% male patients and 44% female patients.

Thyroid Hormone Profile in ACS Patients: Evaluation of thyroid hormone status among patients with acute coronary syndrome revealed that 67% of patients had normal thyroid function, while 33% showed abnormal thyroid hormone profiles. Among the observed thyroid abnormalities, Euthyroid Sick Syndrome was the most common finding, followed by subclinical hypothyroidism and subclinical hyperthyroidism. The detailed distribution of thyroid hormone status among ACS patients is presented in Figure 1.

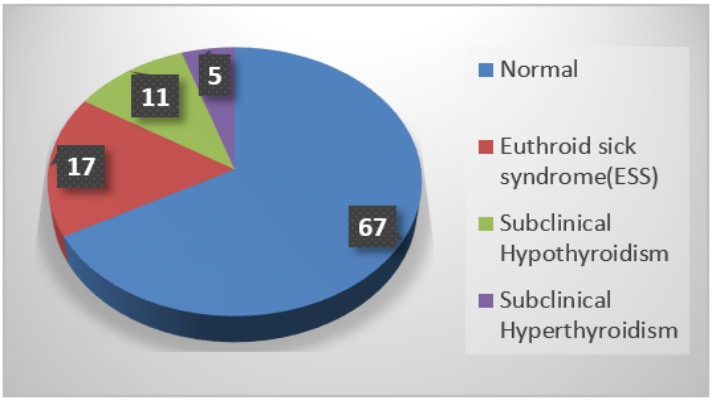

Figure 1: Thyroid Hormone Profile in ACS Patients

Association of Thyroid Dysfunction with Age and Gender: The association between thyroid dysfunction and demographic variables such as age and gender was also analyzed. Thyroid abnormalities were observed in 21 male patients and 12 female patients, while 35 males and 32 females had normal thyroid function. Statistical analysis showed no significant association between thyroid dysfunction and gender (p = 0.3868). Similarly, when thyroid dysfunction was compared across age groups, 10 patients aged 30–60 years and 23 patients older than 60 years showed abnormal thyroid profiles, while 24 and 43 patients respectively had normal thyroid function.

This association was also not statistically significant (p = 0.747). The detailed distribution is presented in Table 2.

Table 2: Association of Thyroid Dysfunction with Age and Gender

Parameter	Thyroid profile		Total	Chi-square test (χ^2)	P Value
	Abnormal	Normal			
Male	21	35	56	0.75	0.3868
Female	12	32	44		
30–60 years	10	24	34	0.10	0.747
>60 years	23	43	66		

Comparison of Thyroid Hormone Levels between STEMI and NSTEMI: The mean levels of thyroid hormones were compared between patients with STEMI and NSTEMI. The mean fT3 level was 2.32 ± 0.86 pg/mL in STEMI patients and 2.36 ± 0.71 pg/mL in NSTEMI patients (p = 0.776). The mean fT4 level was 1.41 ± 0.27 ng/dL in STEMI patients and 1.36 ± 0.24 ng/dL in NSTEMI patients (p = 0.376). The mean TSH level was 3.33 ± 2.51 μIU/mL in STEMI patients and 2.60 ± 1.88 μIU/mL in NSTEMI patients (p = 0.117). These differences were not statistically significant. The comparison of thyroid hormone levels between the two groups is shown in Table 3.

Table 3: Comparison of Thyroid Hormone Levels between STEMI and NSTEMI

Parameter	STEMI Mean ± SD	NSTEMI Mean ± SD	t-value	p-value
fT3 (pg/mL)	**2.32 ± 0.86**	**2.36 ± 0.71**	**-0.286**	**0.776**
fT4 (ng/dL)	**1.41 ± 0.27**	**1.36 ± 0.24**	**0.890**	**0.376**
TSH (μIU/mL)	**3.33 ± 2.51**	**2.60 ± 1.88**	**1.587**	**0.117**

Association between Thyroid Dysfunction and Type of ACS: When thyroid dysfunction was compared between the two ACS subtypes, 20 out of 40 STEMI patients (50%) showed abnormal thyroid profiles, whereas 13 out of 60 NSTEMI patients (21.7%) had thyroid abnormalities. Conversely, 20 STEMI patients and 47 NSTEMI patients had normal thyroid function. This difference was found to be statistically significant ($\chi^2 = 7.48$, p = 0.006). The distribution of thyroid dysfunction according to the type of ACS is presented in Table 4.

Table 4: Thyroid Profile between STEMI VS NSTEMI

ACS type	Thyroid profile		Chi-square test (χ^2)	P Value
	Abnormal	Normal		
STEMI	20	20	7.48	0.00624
NSTEMI	13	47		

Distribution of Thyroid Disorders According to Type of Acute Coronary Syndrome: The distribution of different thyroid abnormalities among patients with ST-elevation myocardial infarction (STEMI) and non–ST-elevation myocardial infarction (NSTEMI) was analyzed. Among the thyroid abnormalities observed, Euthyroid Sick Syndrome (ESS) was present in 10 STEMI patients and 7 NSTEMI patients. Subclinical hypothyroidism was identified in 7 STEMI patients and 4 NSTEMI patients, while subclinical hyperthyroidism was observed in 3 STEMI patients and 2 NSTEMI patients. Statistical analysis using the Chi-square test showed that none of these individual thyroid abnormalities had a significant association with the type of acute coronary syndrome. The p-values for ESS (p = 0.142), subclinical hypothyroidism (p = 0.171), and subclinical hyperthyroidism (p = 0.640) were all greater than 0.05, indicating that the differences observed between STEMI and NSTEMI groups were not statistically significant.

Table 5: Abnormal Thyroid Profile vs Types of Acute Coronary Syndrome

Thyroid Disorder	STEMI	NSTEMI	χ^2 Value	p-value
Euthyroid Sick Syndrome	10	7	2.15	0.142
Subclinical Hypothyroidism	7	4	1.88	0.171
Subclinical Hyperthyroidism	3	2	0.22	0.640

Discussion

The present study was conducted to evaluate thyroid hormone alterations in patients with acute coronary syndrome (ACS) and to determine the relationship between thyroid dysfunction and different types of myocardial infarction. In the present study, 33% of patients exhibited abnormal thyroid hormone profiles, while 67% had normal thyroid function. Among the thyroid abnormalities observed, Euthyroid Sick Syndrome (ESS) was the most common finding (17%), followed by subclinical hypothyroidism (11%) and subclinical hyperthyroidism (5%).

The overall prevalence of thyroid dysfunction observed in the present study (33%) is comparable with previously reported studies evaluating thyroid function in ACS patients. Sah VK et al. reported thyroid dysfunction in 25% of patients with acute coronary syndrome [13], while Chand et al. observed thyroid abnormalities in 27.3% of ACS patients [14]. The slightly higher prevalence observed in the present study may be attributed to differences in patient characteristics, sample size, and the timing of thyroid hormone assessment during the acute phase of myocardial infarction. Acute physiological stress and inflammatory responses during myocardial ischemia are known to

influence thyroid hormone metabolism, which may contribute to the occurrence of transient thyroid abnormalities.

In the present study, Euthyroid Sick Syndrome was identified in 17% of patients, making it the most common thyroid abnormality. Similar findings have been reported in previous studies. Sah et al. reported ESS in approximately 15% of patients with acute coronary syndrome [15], while Chand et al. observed ESS in nearly 17% of their study population [14]. The predominance of ESS in ACS patients may be explained by the metabolic and inflammatory changes that occur during acute myocardial injury. Systemic stress associated with myocardial infarction may suppress the peripheral conversion of thyroxine (T4) to triiodothyronine (T3), leading to reduced circulating T3 levels and the characteristic hormonal pattern observed in ESS.

Another important finding of the present study was the significantly higher prevalence of thyroid dysfunction among STEMI patients compared with NSTEMI patients. In our study, 50% of STEMI patients exhibited abnormal thyroid profiles compared with 21.7% of NSTEMI patients, and this difference was found to be statistically significant ($p = 0.006$). Similar observations have been reported in previous investigations. Sah VK et al. reported thyroid abnormalities in 72% of STEMI patients compared with 28% of NSTEMI/UA patients. [13] The higher prevalence of thyroid abnormalities among STEMI patients may be explained by the greater severity of myocardial injury associated with ST-elevation myocardial infarction. STEMI usually results from complete occlusion of a coronary artery and causes extensive myocardial damage, which triggers a stronger inflammatory and metabolic stress response that may significantly influence thyroid hormone metabolism.

In the present study, when individual thyroid abnormalities were analyzed separately, ESS, subclinical hypothyroidism, and subclinical hyperthyroidism did not demonstrate statistically significant differences between STEMI and NSTEMI groups. This finding may be related to the relatively small sample size and division of patients into multiple subgroups, which reduces the statistical power to detect significant differences. Subclinical hypothyroidism was observed in 11% of patients in the present study. A comparable prevalence has been reported in previous studies evaluating thyroid dysfunction in ACS patients. Sah VK et al. reported subclinical hypothyroidism in approximately 7% of patients with acute coronary syndrome, [13] while other studies have reported prevalence ranging between 7–12% among patients with cardiovascular disease. Subclinical hypothyroidism has been associated with metabolic abnormalities such as dyslipidemia, endothelial dysfunction, and increased systemic vascular resistance, which may contribute to the development and progression of coronary artery disease. [9]

Similarly, subclinical hyperthyroidism was observed in 5% of patients in the present study. Chand et al. reported a comparable prevalence of approximately 4–5% of subclinical hyperthyroidism among ACS patients, [14] which is consistent with the findings of the present study. Although relatively uncommon, subclinical hyperthyroidism may have important cardiovascular implications. Increased thyroid hormone activity may lead to tachycardia, increased myocardial oxygen demand, and a higher risk of arrhythmias, which may predispose susceptible individuals to ischemic cardiac events. [10]

In addition to its prevalence, thyroid dysfunction may also have important prognostic implications in patients with acute coronary syndrome. Several studies have reported that alterations in thyroid hormone levels are associated with worse clinical outcomes following myocardial infarction. [5] Iervasi G et al. demonstrated that patients with reduced T3 levels following acute myocardial infarction had significantly impaired left ventricular function [7], suggesting a relationship between thyroid hormone alterations and the severity of myocardial injury. Similarly, Friberg et al. reported a rapid decline in circulating T3 levels during the early phase of myocardial infarction, [6] indicating that thyroid hormone changes occur as part of the systemic response to acute cardiac stress. Furthermore, Collet et al., in a large pooled analysis involving 52,674 participants, reported that subclinical hyperthyroidism was associated with an increased risk of coronary heart disease events and cardiovascular mortality. [10] These findings suggest that thyroid hormone abnormalities are not only common during acute coronary events but may also contribute to adverse cardiovascular outcomes.

Overall, the findings of the present study demonstrate that thyroid hormone abnormalities are relatively common among patients presenting with acute coronary syndrome, with Euthyroid Sick Syndrome being the most frequent alteration. The significantly higher prevalence of thyroid dysfunction among STEMI patients suggests that thyroid hormone alterations may be associated with the severity of myocardial injury. Therefore, assessment of thyroid hormone profile in patients with ACS may provide useful clinical information and may help identify patients who require closer monitoring during the acute phase of the disease.

The present study has certain limitations that should be considered while interpreting the results. First, the sample size of the study was relatively small (n = 100), which may limit the generalizability of the findings to the wider population. A larger sample size could provide more robust statistical power and allow more accurate evaluation of individual thyroid abnormalities. Second, the study was conducted at a single tertiary care center, which may introduce selection bias and may not fully represent the overall population of patients with acute coronary syndrome. Third, thyroid hormone levels were measured only at the time of admission, and serial measurements were not performed during the course of hospitalization. Since thyroid hormone levels may change during the progression of acute illness, repeated measurements could provide better insight into the dynamic alterations of thyroid function during ACS. Finally, the present study did not evaluate the long-term clinical outcomes or prognostic implications of thyroid dysfunction in these patients. Future studies with larger sample sizes and long-term follow-up are required to better understand the clinical significance of thyroid hormone alterations in patients with acute coronary syndrome.

Conclusion

The present study demonstrates that thyroid hormone abnormalities are relatively common in patients with acute coronary syndrome, with Euthyroid Sick Syndrome being the most frequently observed alteration. The findings also suggest that thyroid dysfunction occurs more frequently in patients with ST-elevation myocardial infarction compared to those with non-ST-elevation myocardial infarction, indicating a possible relationship between thyroid hormone alterations and the severity of myocardial injury. These observations highlight the potential clinical importance of evaluating thyroid hormone status in patients presenting with acute coronary syndrome. Assessment of thyroid function may provide additional insight into the metabolic response associated with acute cardiac events and may assist clinicians in identifying patients who require closer monitoring. Further studies with larger sample sizes and long-term follow-up are required to clarify the clinical and prognostic significance of thyroid hormone alterations in acute coronary syndrome.

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