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Article

Association between Thyroid Function and Postoperative Outcomes in Patients with Acute Coronary Syndrome after Treatment with PCI: A Retrospective Study

Dan Xu[1], Jiawei Zhang[1], Jing Qu[1], Yan Wang[1,*]

[1]Department of Cardiovascular Medicine, Qingdao Central Hospital, University of Health and Rehabilitation Sciences (Qingdao Central Hospital), 266000 Qingdao, Shandong, China

*Correspondence: 18561858396@163.com (Yan Wang)

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Abstract

Objective: This study aims to investigate the relationship between thyroid function and prognosis in acute coronary syndrome (ACS) patients after percutaneous coronary intervention (PCI). **Methods**: According to the thyroid function status of the patients at admission, they were divided into the euthyroid group (Group A), subclinical hyperthyroidism group (Group B), subclinical hypothyroidism group (Group C), and low T3 group (Group D). The clinical data of the four patient groups were compared. The logistic regression model was used to analyze the relationship between thyroid function and postoperative outcomes in patients with ACS undergoing PCI. **Results**: A total of 200 ACS patients who underwent PCI treatment in our hospital from January 2022 to October 2023 were selected as the research population. The incidences of subclinical hyperthyroidism, subclinical hypothyroidism, and low T3 syndrome were 17.00% (34/200), (36/200), and 29.50% (59/200), respectively. Univariate analysis of variance showed significant differences in age, diabetes, smoking history, systolic pressure and heart rate at admission, shock index, blood urea nitrogen (BUN), Cr, glucose (GLU), fibrinogen (FIB), glycosylated hemoglobin (HbA1c), creatine kinase isoenzyme (CK-MB), and CRP among all groups ($p < 0.05$). Thyroid function markers differed significantly across the groups ($p < 0.001$). Specifically, Group B exhibited the lowest thyroid stimulating hormone (TSH) levels (0.23 $\pm$ 0.09 mIU/L), whereas Group C displayed the highest (8.05 $\pm$ 3.72 mIU/L). Group D showed the lowest free triiodothyronine (FT3) levels (2.74 $\pm$ 0.51 pmol/L), and Group B had the highest free thyroxine (FT4) levels (16.31 $\pm$ 2.62 pmol/L). A significant difference in the incidences of cardiovascular death and heart failure was observed among the four groups ($p < 0.05$). Notably, Group D showed higher incidences of cardiovascular death (11.86%) and heart failure (50.85%). Similarly, at 1-year follow-up, a significant difference in the incidences of cardiovascular death and heart failure were found among the four groups ($p < 0.05$). Specially, Group D exhibited higher rates of cardiovascular death (15.25%) and heart failure (27.12%). Logistic regression analyses highlighted FT3 as a significant predictor of adverse events during hospitalization (OR = 0.335, 95% CI: 0.222–0.506, $p < 0.001$), while TSH emerged as an independent risk factor for adverse events during 1-year follow-up (OR = 1.136, 95% CI: 1.040–1.240, $p = 0.005$). **Conclusion**: Patients with ACS have a higher incidence of mild thyroid dysfunction. Compared with patients with euthyroid function, patients with low triiodothyronine (T3) syndrome have a worse prognosis after PCI treatment. Serum FT3 concentration and TSH can be used as predictors of poor prognosis in ACS patients.

Keywords

serum FT3; thyroid stimulating hormone; acute coronary syndrome; percutaneous coronary intervention

Introduction

Acute coronary syndrome (ACS) is a common type of coronary heart disease, including unstable angina (UA), non-ST segment elevation myocardial infarction, and ST segment elevation myocardial infarction (STEMI). They account for 30% to 40% of coronary heart diseases, and the incidence of ACS has gradually increased in recent years [1,2].

Percutaneous coronary intervention (PCI) has become the first choice for the treatment of ACS due to its simple surgical operation, small trauma, fast postoperative recovery, and rapid opening of diseased blood vessels. It significantly reduces cardiac damage and death risk in patients with ACS [3], but some patients may still suffer from restenosis after surgery [4].

In recent years, subclinical thyroid dysfunction has been supposed to be the cause of the worsening of cardiovascular disease [5]. Changes in thyroid stimulating hormone (TSH) levels within the normal range are closely related to changes in blood lipids and blood pressure [6].

Thyroid hormone has various effects on the human cardiovascular system and can directly act on cardiomy-

ocytes [7]. It also binds to thyroid hormone receptors to promote the release of calcium ions from the sarcoplasmic reticulum of cardiomyocytes, activate myocardial contraction-related proteins, and improve myocardial contractile function [8]. When hypothyroidism occurs, the excitability of the sympathetic nervous system decreases, which leads to bradycardia, exacerbates atherosclerosis, and possibly increases the risk of myocardial infarction [9].

The cardiovascular system is the main target organ for the action of thyroxine. About 80.0% of active triiodothyronine (T3) is composed of deiodination of thyroxine (T4), which can exert good biological effects. Free T3 can regulate heart rate and myocardial contractility in the human body. Abnormal thyroid hormone metabolism can cause various heart diseases. The incidence is negatively correlated with coronary heart disease, and hypothyroidism can easily increase the incidence of coronary heart disease [10]. Low T3 syndrome can increase the mortality rate of patients with myocardial infarction and is an independent risk factor for cardiac death. Patients with hypothyroidism are prone increased incidence of angina pectoris on the basis of coronary artery stenosis. In severe cases, hypothyroidism will cause myocardial infarction and threaten the patient's life [11].

Mild thyroid dysfunction includes subclinical hypothyroidism, subclinical hyperthyroidism, and low T3 syndrome. Thyroid hormone has many effects on the cardiovascular system. Current research [12] has confirmed that subclinical hypothyroidism is related to hypercholesterolemia, hypertension, and cardiovascular disease. At present, whether the risk factors affecting the prognosis of PCI in ACS patients are related to the thyroid gland is still unclear. Therefore, strengthening the research on the relationship between thyroid function and PCI prognosis in patients with ACS is important for taking effective measures to deal with and reduce clinical mortality [13].

However, the impact of thyroid function conditions, e.g., disorders on the prognosis of PCI in ACS patients is still uncertain. On this basis, this study aims to analyze the thyroid function and prognosis of ACS patients after PCI and explore their relationship. The goal is to explore its clinical significance, predict the poor prognosis of ACS patients after surgery, and improve the poor prognosis.

Information and Methods

Normal Information

This study is a retrospective study. Specifically, clinical data of 200 ACS patients who underwent PCI in our hospital from January 2022 to October 2023 were collected. According to the thyroid function status of the patients at admission, they were divided into Group A (euthyroid group, n = 71), Group B (subclinical hyperthyroidism group, n = 34), Group C (subclinical hypothyroidism group, n = 36), and Group D (low T3 Group n = 59). All patients were followed up until February 2024. This study complied with the minimum sample size calculation. All patients signed informed consent forms. This study obtained ethical approval from our hospital (ethical approval number: 20231106) and was conducted in line with the Declaration of Helsinki principles.

Inclusion Criteria

Age >18 years old.

According to the symptoms, electrocardiogram changes, and myocardial enzyme spectrum, the patient was diagnosed as ACS according to the clinical diagnostic criteria of ACS [14].

Patients who underwent PCI in our hospital.

TSH level testing was completed after admission.

The exercise treadmill test result was positive.

Clinical and follow-up data are complete.

Exclusion Criteria

Stable angina or angina due to other causes or STEMI.

Patients who develop severe complications such as cardiogenic shock during PCI.

Patients with organic disorders of liver and kidney function.

Patients with malignant tumors.

Patients with cardiomyopathy, congenital heart disease, and heart valve disease.

People with autoimmune dysfunction.

People with connective tissue diseases.

Patients taking oral iodine, thyroxine tablets, or other drugs that affect thyroid function.

No history of abnormal thyroid function in the past.

Patients after thyroidectomy or partial thyroidectomy.

Patients with pituitary-hypothalamic lesions.

Pregnant or breastfeeding women.

Patients with incomplete clinical data.

Methods

(1) Investigation method: The research subjects were asked in detail about their medical history, including age, past medical history, family history, and smoking history. Their body mass index was also calculated to complete the comparison of the patient's general information.

(2) Description of the examination indicators and indicator detection methods in the collected patient data: 3 mL of venous blood was collected from all patients on an empty stomach the day after admission. After rapid centrifugation, the levels of free triiodothyronine (FT3), free thyroxine (FT4), and high-sensitivity thyrotropin (TSH) were measured using the Beckman Coulter Unicel DxL800 (Beckman Coulter, Inc., 20083402447, Brea, CA, USA)

Table 1. Comparison of general data of patients.

Index	Group A (n = 71)	Group B (n = 34)	Group C (n = 36)	Group D (n = 59)	F/χ^2	*p*
Average age	64.54 ± 13.37	67.32 ± 11.91	72.83 ± 9.79	75.12 ± 10.33	10.193	<0.001
Male [n (%)]	41 (57.75)	18 (52.94)	19 (52.78)	32 (54.24)	0.360	0.948
Diabetes [n (%)]	21 (29.56)	11 (32.35)	14 (3.89)	32 (54.24)	9.039	0.029
Hypertension [n (%)]	48 (67.61)	21 (61.76)	24 (66.67)	36 (61.02)	0.797	0.850
History of smoking [n (%)]	42 (59.15)	20 (58.82)	12 (33.33)	23 (38.98)	10.133	0.017
Systolic pressure (mmHg)	137.31 ± 26.20	125.85 ± 28.39	143.53 ± 20.69	133.75 ± 27.21	2.906	0.036
Heart rate (BPM)	87.4 ± 25.5	91.2 ± 17.7	81.4 ± 21.5	78.3 ± 26.2	2.747	0.044
Shock index	0.6 ± 0.2	0.7 ± 0.1	0.6 ± 0.1	0.6 ± 0.2	3.978	0.009
BMI (kg/m^2)	25.48 ± 3.96	25.99 ± 4.60	25.20 ± 2.02	25.12 ± 5.51	0.317	0.813
BUN (mmol/L)	6.6 ± 2.9	6.2 ± 2.2	7.9 ± 6.1	10.1 ± 6.1	25.425	<0.001
Cr (umol/L)	75.5 ± 10.5	71.4 ± 7.3	71.2 ± 13.7	95.3 ± 13.5	50.432	<0.001
UA (umol/L)	358.3 ± 101.0	336.7 ± 96.9	373.3 ± 112.1	402.5 ± 170.3	2.284	0.080
FIB (g/L)	3.1 ± 0.9	3.3 ± 0.8	3.5 ± 0.9	4.1 ± 1.3	10.672	<0.001
cTnI (pg/mL)	20.4 ± 23.5	28.9 ± 22.8	19.9 ± 20.5	22.9 ± 21.5	1.315	0.271
HbA1c (%)	7.3 ± 1.7	8.1 ± 2.6	7.1 ± 1.1	7.9 ± 1.8	2.387	0.049
GLU (mmol/L)	6.9 ± 3.9	7.6 ± 4.2	6.8 ± 2.1	9.6 ± 3.9	7.110	<0.001
CK-MB (U/L)	65.3 ± 35.8	125.1 ± 45.1	43.10 ± 9.50	66.4 ± 30.5	39.879	<0.001
TC (mmol/L)	4.9 ± 2.5	4.6 ± 1.2	4.8 ± 1.2	4.7 ± 1.1	0.284	0.837
TG (mmol/L)	2.1 ± 1.7	1.8 ± 1.0	1.9 ± 1.2	1.6 ± 0.7	2.108	0.101
HDL (mmol/L)	1.0 ± 0.3	1.1 ± 0.3	0.9 ± 0.3	1.0 ± 0.3	1.925	0.127
LDL-C (mmol/L)	2.9 ± 0.8	2.7 ± 1.0	3.1 ± 0.9	3.0 ± 0.9	1.562	0.200
hs-CRP (mg/L)	6.7 ± 2.2	15.0 ± 5.3	15.5 ± 11.5	41.4 ± 19.7	94.932	<0.001

*Note: CA199 is carbohydrate antigen 199 and CEA is carcinoembryonic antigen. BMI, body mass index; BUN, blood urea nitrogen; Cr, creatinine; UA, unstable angina; FIB, fibrinogen; cTnI, cardiac enzymes such as troponin I; HbA1c, glycosylated hemoglobin; GLU, glucose; CK-MB, creatine kinase isoenzyme; TC, cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL-C, low-density lipoprotein-C; hs-CRP, hypersensitive C-reactive protein.

immunoassay analyzer. At the same time, the Abbott C16000 (Abbott Laboratories; 20162223123; Chicago, IL, USA) fully automatic biochemical analyzer produced in the United States was used to detect blood lipid levels. Heart rate (HR), blood pressure (BP), and shock index (SI) = heart rate/blood pressure were collected. Kidney function was assessed using blood urea nitrogen (BUN) and uric acid (UA). Blood lipids, including cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL), were evaluated. Blood glucose (GLU), fibrinogen (FIB), a blood routine examination, glycosylated hemoglobin (HbA1c), and cardiac enzymes such as troponin I (cTnI) and creatine kinase isoenzyme (CK-MB) peak were measured. The inflammatory marker high-sensitivity serum C-reactive protein was detected using Siemens BN-II fully automatic specific protein analyzer.

Diagnostic Standards for the Definition of Thyroid Function Status

Thyroid hormone was measured on the second day of admission. Specifically, blood was collected at 7 am and tested using an AIA600 machine (Tosa Corporation, Japan; 20113401158; Zhounan City, Yamaguchi Prefecture; Tampa, FL, USA). Normal thyroid function was defined as levels of TSH, FT4, and FT3 within the reference range. The normal range of data was as follows: FT3: 2.8–6.3 pmo/L, FT4: 11.5–22.7 pmol/L, TSH: 0.35–5.50 mIU/L. Subclinical hypothyroidism was defined when serum TSH was above the reference range (TSH >5.50 mIU/L) and serum FT3 and FT4 were within the normal range. Subclinical hyperthyroidism was defined as serum TSH below the reference range (TSH <0.35 mIU/L) and serum FT3 and FT4 were within the normal range. Low T3 syndrome was defined as low serum FT3 levels (FT3 <3.1 pmol/L) and FT4 and TSH were within the normal range.

Table 2. Comparison of two indexes between two groups ($\bar{x} \pm s$).

Index	FT3 (pmol/L)	FT4 (pmol/L)	TSH (mIU/L)
Group A (n = 71)	4.15 ± 0.56	15.45 ± 2.53	1.44 ± 0.71
Group B (n = 34)	3.92 ± 0.73	16.31 ± 2.62	0.23 ± 0.09
Group C (n = 36)	4.07 ± 0.64	15.18 ± 2.24	8.05 ± 3.72
Group D (n = 59)	2.74 ± 0.51	14.83 ± 2.36	1.86 ± 0.78
F	70.920	2.706	163.692
p	<0.001	0.047	<0.001

FT3, triiodothyronine; FT4, free thyroxine; TSH, thyroid stimulating hormone.

Table 3. Short- and long-term follow-up of patients in each group.

Index	Group A (n = 71)	Group B (n = 34)	Group C (n = 36)	Group D (n = 59)	*p*
Adverse events during hospitalization (cases/%)					
Cardiovascular death [n (%)]	1 (1.41)	4 (11.76)	0 (0.0)	7 (11.86)	0.014
Heart failure [n (%)]	9 (12.68)	10 (29.41)	10 (27.78)	30 (50.85)	<0.001
Non-fatal myocardial infarction [n (%)]	1 (1.41)	0 (0.00)	2 (5.56)	3 (5.08)	0.340
Severe angina [n (%)]	1 (1.41)	2 (5.88)	4 (11.11)	3 (5.08)	0.186
Bleeding [n (%)]	1 (1.41)	1 (2.94)	2 (5.56)	4 (6.78)	0.431
Adverse events at 1-year follow-up [n (%)]					
Cardiovascular death	1 (1.41)	1 (2.94)	2 (5.56)	9 (15.25)	0.011
Heart failure	5 (7.04)	2 (5.88)	9 (25.00)	16 (27.12)	0.002
Non-fatal myocardial infarction	2 (2.86)	3 (8.82)	2 (5.56)	2 (3.39)	0.530
Severe angina	2 (2.86)	2 (5.88)	4 (11.11)	1 (1.69)	0.147
Bleeding	1 (1.41)	0 (0.00)	1 (2.78)	0 (0.00)	0.530

Table 4. Results of logistic regression analysis.

Index	B	SE	Wald	*p*	OR	95% CI
Adverse events during the hospitalization as the dependent variable						
BUN	0.125	0.043	8.617	0.003	1.133	1.042–1.232
FT3	−1.092	0.210	27.135	0.000	0.335	0.222–0.506
FT4	0.130	0.058	4.973	0.026	1.139	1.016–1.277
Adverse events during the 1-year follow-up period as the dependent variable						
TSH	0.127	0.045	8.055	0.005	1.136	1.040–1.240

SE, Standard Error; OR, odds ratio; Cl, confidence interval.

Statistical Analysis

The data in this study were analyzed and processed by SPSS21.0 (IBM Corporation, Armonk, NY, USA) statistical software. For continuous variables, data were expressed as mean ± SD for normally distributed variables and median (P25, P75) for non-normally distributed variables, with comparisons between groups conducted using analysis of variance and the Kruskal–Wallis test, respectively. For categorical variables, data were expressed as numbers (percentages) with comparisons between groups performed using the chi-square test. The logistic regression model was used to analyze the relationship between thyroid function and postoperative outcomes in patients with ACS undergoing PCI. $p < 0.05$ means the difference was statistically significant.

Results

Comparison of General Data

A total of 200 ACS patients in our hospital from January 2022 to October 2023 were finally included. According to the thyroid function status, they were divided into four groups: Group A: normal thyroid function group (71 cases), Group B: subclinical hyperthyroidism group (34 cases), Group C: subclinical hypothyroidism group (36 cases), and Group D: low T3 syndrome group (59 cases) (Table 1). The incidences of subclinical hyperthyroidism, subclinical hypothyroidism, and low T3 syndrome were 17.00%, 18.00%, 29.50%, respectively. Univariate analysis of variance showed significant differences in age, diabetes, smoking history, systolic pressure and heart rate at admission, shock index, BUN, Cr, GLU, FIB, HbA1c, CK-MB, and CRP among all groups ($p < 0.05$). Moreover, other data among four groups were not statistically different ($p > 0.05$).

Comparison of TSH, FT3, and FT4 in the Four Groups

We measured TSH, FT3, and FT4 levels among the four groups. The results showed (Table 2) that TSH levels were 1.44 ± 0.71 mIU/L, 0.23 ± 0.09 mIU/L, 8.05 ± 3.72 mIU/LL, and 1.86 ± 0.78 mU/L for the four groups, respectively ($p < 0.001$). FT3 levels were 4.15 ± 0.56 pmol/L, 3.92 ± 0.73 pmol/L, 4.07 ± 0.64 pmol/L, and 2.74 ± 0.51 pmol/mL ($p < 0.001$). FT4 levels were 15.45 ± 2.53 pmol/L, 16.31 ± 2.62 pmol/L, 15.18 ± 2.24 pmol/L, and 14.83 ± 2.36 pmol/L ($p < 0.05$).

Comparison of Short- and Long-Term Follow-up of Patients in Each Group

During hospitalization, the incidences of cardiovascular death were 1.41%, 11.76%, 0%, and 11.86% in Group A, B, C, and D, respectively ($p = 0.014$). The occurrence of heart failure varied significantly among groups, with rates

of 12.68% in Group A, 29.41% in Group B, 27.78% in Group C, and 50.85% in Group D ($p < 0.001$). No significant differences were found between groups in other adverse events such as non-fatal myocardial infarction, severe angina, and bleeding. At 1-year follow-up, significant differences persisted among groups in cardiovascular death ($p = 0.011$) and heart failure ($p = 0.002$) (Table 3).

Logistic Regression Analysis of Risk Factors Related to Long- and Short-Term Adverse Events in Patients

The occurrence of adverse events during hospitalization was the dependent variable. The variables (e.g., systolic blood pressure, heart rate, shock finger, BUN, Cr, GLU, FIB, HbA1c, and CK-MB) with statistical differences among the groups of normal thyroid function, subclinical hyperthyroidism, subclinical hypothyroidism, and low T3 syndrome were used as independent variables for stepped-regression analysis. After confounding factors were adjusted, FT3, FT4, and BUN were entered into the regression equation. Among them, FT3 was highly correlated with the incidence of adverse events during hospitalization (OR = 0.335, 95% CI: 0.222–0.506, $p < 0.001$) (Table 4).

The occurrence of adverse events during 1-year follow-up was used as the dependent variable, and Cr, GLU, FT3, FT4, TSH, and other statistically significant indicators were selected as independent variables for stepwise regression analysis. The results showed that TSH was an independent risk factor for predicting adverse events in ACS patients within 1 year of follow-up (OR = 1.136, 95% CI: 1.040–1.240, $p = 0.005$) (Table 4).

Discussion

Non-thyroid abnormal systemic diseases can cause abnormalities in thyroid dysfunction, which is generally called “euthyroid sick syndrome (ESS)” Surgery and fasting may cause ESS, and ACS patients often accompany varying degrees of thyroid hormone changes [15,16]. In this study, the incidences of subclinical hyperthyroidism, subclinical hypothyroidism, and low T3 syndrome were 17.00%, 18.00%, and 29.50%, respectively.

ACS is a common clinical disease. The disease is a syndrome of coronary atherosclerotic plaque rupture, bleeding, and thrombosis, which induce angina pectoris and even infarction. If the patient does not take positive and effective treatment after the onset, then other diseases will be induced, and severe cases will threaten the patient’s life. At present, patients with simultaneous coronary heart disease and normal thyroid hormones, have high levels of coronary heart disease, disease mortality, and heart-based death. However, the results of previous research are controversial. Studies have shown that the metabolic deformation of coronary heart disease can cause changes in thyroid hormone levels. However, the conclusions differ due to the different standards of research samples [17–20].

Basic data after admission showed that the heart rates of patients in the three groups of subclinical hyperthyroidism, subclinical hypothyroidism, and low T3 syndrome were higher than those in the euthyroid group. Heart rate is a predictor of morbidity and mortality related to coronary heart disease, as supported by earlier studies and substantial research evidence. Thyroid hormones can act on the cardiovascular system directly or indirectly by affecting the patient’s heart rate levels. Given that hyperthyroidism can cause atrial fibrillation, thyroid hormone physiology also affects the electrophysiological properties of the heart [21].

Statistical analysis in this study found that the HGB levels in the three groups of patients with thyroid dysfunction were significantly lower than those in the euthyroid group ($p < 0.05$). A previous study [22] emphasized that anemia is also related to a decrease in FT3 levels. Another study [23] showed that anemia promotes the development of non-thyroid disease syndromes by blunting the pituitary response to TRH and reducing peripheral conversion of FT3.

A study [24] reported that, among people with coronary heart disease, changes in TSH levels within the normal range are closely related to cardiovascular death. Another study [25] also found that changes in thyroid hormone levels are related to the occurrence of atherosclerosis and myocardial infarction. At the same time, they can regulate multiple cell functions and affect cell metabolism, protein synthesis, and material transport. However, the relationship between TSH and adverse events after PCI in patients with non-ST-segment elevation ACS is still unclear.

This study found that Cr and UA and other indicators reflecting renal function in the patient group with low T3 syndrome were significantly higher than those in the three other groups. A review on the impact of low T3 syndrome on cardiorenal syndrome in patients also showed that, as renal function declines, the incidence of low T3 syndrome gradually increases [26].

Analysis of the basic data of this study showed that the proportion of patients with diabetes history in the low T3 syndrome group was clearly higher than that of the three other groups. Notably, patients in the subclinical hyperthyroidism group have a higher smoking history than other patients, and diabetes, smoking, and drinking can lead to an inflammatory state in the body. Therefore, thyroid dysfunction affects the short- and long-term prognosis of AMI patients through the inflammatory mechanism [27].

The follow-up data during hospitalization showed that patients in the low T3 syndrome group had significantly higher adverse events of heart failure during hospitalization than the three other groups, and their cardiovascular death rate was significantly higher. In logistic regression analysis, FT3 had the highest correlation with the incidence of adverse events during hospitalization (OR = 0.335, 95% CI:

0.222–0.506, $p < 0.001$). This finding is consistent with the findings in the study of Wang *et al*. [28]. Comparing adverse events during the 1-year follow-up period showed that the proportion of non-fatal myocardial infarction in patients with subclinical hyperthyroidism during the follow-up period was significantly higher than that of the three other groups. In logistic regression analysis, TSH was an independent risk factor for predicting adverse events in ACS patients within 1 year of follow-up, and TSH was an independent risk factor for adverse events during 1-year follow-up (OR = 1.136, 95% CI: 1.040–1.240, $p = 0.005$). Thus, we speculate that FT3 can be used as a predictor of short-term adverse prognosis, while TSH can be used as a predictor of long-term prognosis. This view requires further research for confirmation.

This study also has certain limitations. First, this study is a single-center study with a limited number of participants. Thus, the results are more of a baseline study and supplement to the field, and larger studies are needed. Second, various biases are inevitably observed in retrospective studies. In our work, potential selection bias may be present. Third, current studies have shown that thyroid hormones decrease within a short time after the occurrence of ACS. This study did not detect thyroid hormones during patient follow-up, and further analysis was not conducted. Fourth, the expression and activity of various abzyme subtypes of thyroxine deiodinase activity, activation complexes, and nuclear thyroid hormone receptor subtypes differ between tissues and organs [29] and may affect the results of the study. Finally, variables such as comorbidity and drug use were excluded in this study. For example, some cardiac drugs such as amiodarone also affect the occurrence of thyroid disease [30], and this study did not further determine the impact of these drugs on thyroid hormone levels. In future studies, we will include additional variables for a more comprehensive analysis.

Conclusion

Patients with ACS have a higher incidence of thyroid dysfunction. Compared with patients with euthyroid function, patients with low T3 syndrome have significantly higher myocardial injury markers and inflammatory markers, and the prognosis after PCI is poor. Effective measures should be made to improve the prognosis of patients. Serum FT3 concentration and TSH can be used as predictors of poor prognosis in ACS patients.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

DX designed the study; all authors conducted the study; JQ and YW collected and analyzed the data; DX and JZ participated in drafting the manuscript, and all authors contributed to critical revision of the manuscript for important intellectual content. All authors gave final approval of the version to be published. All authors participated fully in the work, take public responsibility for appropriate portions of the content, and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or completeness of any part of the work are appropriately investigated and resolved.

Ethics Approval and Consent to Participate

This study was approved by the ethics committee of Qingdao Central Hospital (Approval No.: 20231106). Data were anonymized to protect patient privacy and all study subjects gave informed consent.

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Conflict of Interest

The authors declare no conflict of interest.

References

[1] Han K, Shi D, Yang L, Wang Z, Li Y, Gao F, *et al*. Prognostic value of systemic inflammatory response index in patients with acute coronary syndrome undergoing percutaneous coronary intervention. Annals of Medicine. 2022; 54: 1667–1677.

[2] Buccheri S, Capodanno D, James S, Angiolillo DJ. Bleeding after antiplatelet therapy for the treatment of acute coronary syndromes: a review of the evidence and evolving paradigms. Expert Opinion on Drug Safety. 2019; 18: 1171–1189.

[3] Arambam P, Kaul U, Ranjan P, Janardhanan R. Prognostic implications of thyroid hormone alterations in acute coronary syndrome-A systematic review. Indian Heart Journal. 2021; 73: 143–148.

[4] Jabbar A, Ingoe L, Thomas H, Carey P, Junejo S, Addison C, *et al*. Prevalence, predictors and outcomes of thyroid dysfunction in patients with acute myocardial infarction: the ThyrAMI-1 study. Journal of Endocrinological Investigation. 2021; 44: 1209–1218.

[5] Abdu FA, Mohammed AQ, Liu L, Zhang W, Yin G, Xu

B, *et al*. Low Free Triiodothyronine as a Predictor of Poor Prognosis in Patients With Myocardial Infarction With Non-Obstructive Coronary Arteries. Frontiers in Endocrinology. 2021; 12: 681978.

[6] Cao Q, Jiao Y, Yu T, Sun Z. Association between mild thyroid dysfunction and clinical outcome in acute coronary syndrome undergoing percutaneous coronary intervention. Cardiology Journal. 2020; 27: 262–271.

[7] Tian XX, Zheng SF, Liu JE, Wu YY, Lin L, Chen HM, *et al*. Free Triiodothyronine Connected With Metabolic Changes in Patients With Coronary Artery Disease by Interacting With Other Functional Indicators. Frontiers in Molecular Biosciences. 2021; 8: 681955.

[8] Rasool R, Unar A, Jafar TH, Chanihoon GQ, Mubeen B. A Role of Thyroid Hormones in Acute Myocardial Infarction: An Update. Current Cardiology Reviews. 2023; 19: e280422204209.

[9] Senguttuvan NB, Reddy PMK, Shankar P, Abdulkader RS, Yallanki HP, Kumar A, *et al*. Trans-radial approach versus trans-femoral approach in patients with acute coronary syndrome undergoing percutaneous coronary intervention: An updated meta-analysis of randomized controlled trials. PLoS ONE. 2022; 17: e0266709.

[10] Zhuang Q, Shen C, Chen Y, Zhao X, Wei P, Sun J, *et al*. Association of high sensitive C-reactive protein with coronary heart disease: a Mendelian randomization study. BMC Medical Genetics. 2019; 20: 170.

[11] Bergmark BA, Mathenge N, Merlini PA, Lawrence-Wright MB, Giugliano RP. Acute coronary syndromes. Lancet. 2022; 399: 1347–1358.

[12] Rosário PWS, Calsolari MR. Subclinical Hypothyroidism with TSH$>$7 mIU/l and$\leq$10 mIU/l and Coronary Artery Disease. Hormone and Metabolic Research. 2020; 52: 85–88.

[13] Hajian-Tilaki K, Heidari B, Bakhtiari A. Triglyceride to high-density lipoprotein cholesterol and low-density lipoprotein cholestrol to high-density lipoprotein cholesterol ratios are predictors of cardiovascular risk in Iranian adults: Evidence from a population-based cross-sectional study. Caspian Journal of Internal Medicine. 2020; 11: 53–61.

[14] Kimura K, Kimura T, Ishihara M, Nakagawa Y, Nakao K, Miyauchi K, *et al*. JCS 2018 Guideline on Diagnosis and Treatment of Acute Coronary Syndrome. Circulation Journal. 2019; 83: 1085–1196.

[15] Kong S, Chen C, Zheng G, Yao H, Li J, Ye H, *et al*. A Nomogram Based on Lactate to Predict Major Adverse Cardiovascular Events for Acute Coronary Syndrome after Percutaneous Coronary Intervention. 2021. (preprint)

[16] Kong S, Chen C, Zheng G, Yao H, Li J, Ye H, *et al*. A prognostic nomogram for long-term major adverse cardiovascular events in patients with acute coronary syndrome after percutaneous coronary intervention. BMC Cardiovascular Disorders. 2021; 21: 253.

[17] Yu N, Wang L, Zeng Y, Zhao Y, Chen S, Pan H, *et al*. The Association of Thyroid Hormones with Coronary Atherosclerotic Severity in Euthyroid Patients. Hormone and Metabolic Research. 2022; 54: 12–19.

[18] Gürdoğan M, Altay S, Korkmaz S, Kaya Ç, Zeybey U, Ebik M, *et al*. The Effect of Thyroid Stimulating Hormone Level Within the Reference Range on In-Hospital and Short-Term Prognosis in Acute Coronary Syndrome Patients. Medicina. 2019; 55: 175.

[19] Yuan D, Zhang C, Jia S, Liu Y, Jiang L, Xu L, *et al*. Predictive value of free triiodothyronine (FT3) to free thyroxine (FT4) ratio in long-term outcomes of euthyroid patients with three-vessel coronary artery disease. Nutrition, Metabolism, and Cardiovascular Diseases. 2021; 31: 579–586.

[20] Abdel-Moneim A, Gaber AM, Gouda S, Osama A, Othman SI, Allam G. Relationship of thyroid dysfunction with cardiovascular diseases: updated review on heart failure progression. Hormones. 2020; 19: 301–309.

[21] Gencer B, Cappola AR, Rodondi N, Collet TH. Challenges in the Management of Atrial Fibrillation With Subclinical Hyperthyroidism. Frontiers in Endocrinology. 2022; 12: 795492.

[22] Tomoda F, Takata M, Izumino K, Oh-hashi S, Ueno H, Iida H. Effects of erythropoietin treatment on thyroid dysfunction in hemodialysis patients with renal anemia. Nephron. 1994; 66: 307–311.

[23] Adams KF, Jr, Patterson JH, Oren RM, Mehra MR, O'Connor CM, Piña IL, *et al*. Prospective assessment of the occurrence of anemia in patients with heart failure: results from the Study of Anemia in a Heart Failure Population (STAMINA-HFP) Registry. American Heart Journal. 2009; 157: 926–932.

[24] van Vliet NA, Bos MM, Thesing CS, Chaker L, Pietzner M, Houtman E, *et al*. BBMRI Metabolomics Consortium. Higher thyrotropin leads to unfavorable lipid profile and somewhat higher cardiovascular disease risk: evidence from multi-cohort Mendelian randomization and metabolomic profiling. BMC medicine. 2021; 19: 1–13.

[25] Lerner RK, Gruber N, Pollak U. Congenital Heart Disease and Thyroid Dysfunction: Combination, Association, and Implication. World Journal for Pediatric & Congenital Heart Surgery. 2019; 10: 604–615.

[26] Xiong H, Yan P, Huang Q, Shuai T, Liu J, Zhu L, *et al*. A prognostic role for non-thyroidal illness syndrome in chronic renal failure:a systematic review and meta-analysis. International Journal of Surgery. 2019; 70: 44–52.

[27] Ni WC, Kong ST, Lin K, Huang YH, Li JF, Shi SL, *et al*. Normal thyroid stimulating hormone is associated with all-cause mortality in patients with acute myocardial infarction after percutaneous coronary intervention. European Journal of Medical Research. 2023; 28: 199.

[28] Wang B, Liu S, Li L, Yao Q, Song R, Shao X, *et al*. Non-thyroidal illness syndrome in patients with cardiovascular diseases: A systematic review and meta-analysis. International Journal of Cardiology. 2017; 226: 1–10.

[29] De Luca R, Davis PJ, Lin HY, Gionfra F, Percario ZA, Affabris E, *et al*. Thyroid Hormones Interaction With Immune Response, Inflammation and Non-thyroidal Illness Syndrome. Frontiers in Cell and Developmental Biology. 2021; 8: 614030.

[30] Wang J, Zhang R. Evaluation of ^{99m}Tc-MIBI in thyroid gland imaging for the diagnosis of amiodarone-induced thyrotoxicosis. The British Journal of Radiology. 2017; 90: 20160836.