Research Square

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Lower Serum Free Triiodothyronine is Independently Associated with Isolated Left Anterior Descending Coronary Slow Flow：A Cross-Sectional Study

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Research Article

Keywords: Coronary slow flow, Free triiodothyronine, Microvascular dysfunction, TIMI frame count, Biomarker

Posted Date: July 7th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10230639/v1

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Additional Declarations: The authors declare no competing interests.

Abstract

Background

Coronary slow flow (CSF) is an angiographic phenomenon characterized by delayed distal vessel opacification without obstructive coronary disease. CSF is associated with angina and increased cardiovascular risk, yet its pathophysiology remains incompletely understood. This study aimed to investigate the association between serum free triiodothyronine (FT3) levels and CSF in the left anterior descending coronary artery (LAD), and also to evaluate its predictive value.

Methods

In this single-center, cross-sectional study, 77 patients undergoing coronary angiography without significant stenosis were categorized into LAD normal flow (n = 30, corrected TIMI frame count [CTFC] ≤ 27) and slow flow (n = 47, CTFC > 27) groups. Clinical, laboratory, and echocardiographic parameters were collected and compared.

Results

Univariate analysis revealed significantly lower FT3 levels in the CSF group (4.42 ± 0.80 vs. 5.06 ± 0.57 pmol/L, P < 0.001). Multivariate logistic regression confirmed FT3 as an independent protective factor against CSF (OR = 0.257, 95%CI: 0.103–0.639, P = 0.003). FT3 levels were inversely correlated with CTFC (r=-0.414, P < 0.001). Receiver operating characteristic curve analysis demonstrated that FT3 had a moderate predictive ability for CSF (AUC = 0.749, 95%CI: 0.641–0.858), with an optimal cutoff value of 4.69 pmol/L (sensitivity 83.3%, specificity 68.1%).

Conclusions

Lower serum FT3 is independently associated with LAD slow flow and shows promising predictive value, suggesting a potential role of thyroid hormone in coronary microvascular dysfunction. Further prospective and mechanistic studies are warranted to establish causality and explore therapeutic implications.

1. INTRODUCTION

Coronary slow flow (CSF) is defined as delayed distal vessel opacification in the absence of significant epicardial stenosis[1]. With a prevalence of 1–5%[3], CSF represents a prevalent clinical entity that often leads to recurrent angina, frequent hospitalizations, and adverse long-term cardiovascular outcomes[2, 3]. Despite its clinical significance, CSF remains under-recognized and lacks targeted therapeutic strategies,

largely due to an incomplete understanding of its pathophysiology. Although microvascular dysfunction is recognized as the central driver—encompassing endothelial dysfunction, abnormal vasoreactivity, and inflammation[1, 2]—the role of endocrine factors remains underexplored.

While previous studies have linked CSF to traditional risk factors and inflammatory markers, the impact of subtle variations in thyroid hormone levels within the normal reference range has not been elucidated. Specifically, free triiodothyronine (FT3) is crucial for regulating endothelial function and vascular tone[4–6]; however, its association with CSF remains unestablished. Given that a "low T3 state" portends poor prognosis in various cardiac conditions[7], we hypothesized that lower FT3 levels may be implicated in coronary microvascular dysfunction.

To address this critical gap, the present study employs a methodologically rigorous approach by focusing exclusively on isolated slow flow in the left anterior descending (LAD) artery. This specific focus is clinically justified, as the LAD is the most frequently affected vessel in CSF and supplies a substantial myocardial territory, rendering its functional status of paramount importancet[8].

2. MATERIALS AND METHODS

2.1 Study Population

77 patients undergoing coronary angiography in our hospital between 2024 and 2025 were enrolled in this study based on predefined inclusion and exclusion criteria. Inclusion criteria: Patients who underwent coronary angiography via radial artery pathway with high-quality images suitable for analysis. Exclusion criteria: Stenosis exceeding 50% in any major coronary artery including left main artery (LM), left anterior descending artery (LAD), left circumflex artery (LCX), and right coronary artery (RCA); acute myocardial infarction; left ventricular ejection fraction (LVEF) < 40%; hypertrophic cardiomyopathy; connective tissue diseases; history of percutaneous coronary intervention; congenital heart disease; hypothyroidism or hyperthyroidism. Based on the corrected thrombolysis in myocardial infarction (TIMI) frame count (CTFC)[9], patients were divided into two groups: the LAD normal flow group (n = 30, CTFC ≤ 27) and the LAD slow flow group (n = 47, CTFC > 27). This study was approved by the Institutional Ethics Committee of our hospital, and informed consent was obtained from all participants.

2.2 Clinical Data Collection

After admission, the patients information were collected, including gender and age. Height and weight were measured to calculate the body mass index (BMI). History of smoking and alcohol consumption was recorded, along with the presence of comorbidities such as diabetes mellitus, hypertension, chronic obstructive pulmonary disease (COPD), and atrial fibrillation. Blood pressure and heart rate were recorded at the commencement of coronary angiography for all patients.

2.3 Laboratory and Echocardiographic Assessments

Venous blood samples were drawn after an overnight fast of more than 8 hours to assess blood routine, lipid profile, renal function, and thyroid hormone levels. Samples were delivered to the laboratory within 2 hours.

Prior to coronary angiography, all subjects underwent transthoracic echocardiography performed by qualified and experienced sonographers. Morphological and functional parameters were measured in standard section views, including ascending aortic diameter (AAOD), left atrial diameter (LAD), left ventricular diameter (LVD), interventricular septal thickness (IVST), left ventricular long axis fractional shortening (FS), and left ventricular ejection fraction (LVEF).

2.4 Coronary Angiography Procedure

Selective coronary angiography was performed via the transradial approach using the standard Judkins technique on a Neusoft angiography system. The procedure involved radial artery puncture, insertion of a 6F radial sheath, administration of 3000 units of heparin, placement of guide wire and a 5F Tig catheter to engage the coronary ostia for manual contrast injection. The anteroposterior cranial and right anterior oblique projections were used for LAD angiography. Notably, nitroglycerin and other medications potentially affecting coronary blood flow were withheld for 1 hour prior to and during the angiographic procedure.

2.5 Definition of LAD Slow Flow

LAD slow flow was diagnosed using the TIMI frame count (TFC) method proposed by Gibson et al. The frame acquisition rate was standardized to 30 frames per second. The frame count was defined as the number of frames required for the contrast dye to travel from the coronary ostium to distal. The first frame was defined when contrast completely or nearly completely filled the proximal coronary segment, touching both vessel walls, with visible forward motion. The last frame was defined when contrast reached the specific distal anatomical landmark of the LAD (the apical bifurcation), characterized by the “fork,” “mustache,” or “whale tail” signs. Two experienced investigators, who were blinded to the study groups, measured the TFC for each subject, and the mean value was adopted for subsequent analysis. Given the typically longer length of the LAD, its TFC value was divided by 1.7 to obtain the corrected TFC (CTFC). CTFC > 27 frames was defined as LAD CSF.

2.6 Statistical Analysis

Data were analyzed using SPSS version 26.0. Continuous variables were tested for normality and homogeneity of variance. Data are presented as mean ± standard deviation (SD). Differences between groups were analyzed using the Student’s t-test or non-parametric tests. Categorical variables are expressed as counts (percentages) and were compared using the Chi-square test. Multivariate logistic regression analysis was employed to identify independent risk factors associated with LAD coronary slow flow. Correlations between variables were assessed using Spearman’s rank correlation coefficient. $P < 0.05$ was considered statistically significant.

3. RESULTS

3.1 Comparison of Baseline Characteristics

The comparison of baseline characteristics between the two groups is presented in Table 1. The results showed that the CTFC in the LAD normal flow group was 20.16 ± 4.02, while in the LAD slow flow group it was 41.60 ± 12.46, indicating a statistically significant difference ($P < 0.001$). The proportion of smokers was significantly higher in the slow flow group compared to the normal flow group [22 (46.8%) vs 7 (23.3%), P = 0.038]. No statistically significant differences were observed in other baseline characteristics between the two groups.

Table 1
Comparison of Baseline Characteristics Between LAD Normal Flow and Slow Flow Group

Variable	**Normal Flow Group (n = 30)**	**Slow Flow Group (n = 47)**	**Statistical indicator**	***P***
CTFC (frames)	20.16 ± 4.02	41.60 ± 12.46*	-7.368	0.000
Gender [male, n(%)]	12 (40.0)	28 (59.6)	2.811	0.094
Age (years)	58.0 ± 11.0	63.0 ± 7.8	-1.914	0.056
Height (cm)	165.8 ± 6.7	164.3 ± 7.5	0.877	0.383
Weight (kg)	66.3 ± 12.3	67.0 ± 11.6	-0.278	0.782
BMI (kg/m²)	24.0 ± 3.3	25.0 ± 4.0	-1.106	0.272
Hypertension [n(%)]	15 (50.0)	27 (57.4)	0.410	0.522
Diabetes Mellitus [n(%)]	4 (13.3)	8 (17.0)	0.189	0.756
COPD [n(%)]	2 (6.7)	3 (6.4)	0.002	1.000
Atrial Fibrillation [n(%)]	1 (3.3)	6 (12.8)	1.971	0.237
Smoking [n(%)]	7 (23.3)	22 (46.8)*	4.298	0.038
Alcohol Consumption [n(%)]	7 (23.3)	17 (36.2)	1.407	0.236
Systolic BP (mmHg)	127.3 ± 20.3	123.5 ± 14.8	0.923	0.359
Diastolic BP (mmHg)	84.0 ± 13.7	81.0 ± 11.6	1.000	0.320
Heart Rate (bpm)	71.2 ± 12.7	68.9 ± 10.3	-0.817	0.414

Note: CTFC, Corrected TIMI Frame Count; COPD, Chronic Obstructive Pulmonary Disease; BMI, Body Mass Index; BP, Blood Pressure . Indicates P < 0.05 compared with the normal flow group.*

3.2 Comparison of Laboratory and Echocardiographic Parameters

Comparisons of laboratory and echocardiographic parameters between the two groups are shown in Table 2. The results indicated that the basophil count (BASO) was significantly lower in the LAD slow flow group compared to the normal flow group (0.02 ± 0.01 vs 0.03 ± 0.01, $P = 0.031$). Additionally, free triiodothyronine (FT3) levels were significantly lower in the slow flow group than in the normal flow group (4.42 ± 0.80 vs 5.06 ± 0.57, $P < 0.001$). No statistically significant differences were found in other measured parameters between the two groups.

Table 2
Comparison of Laboratory and Echocardiographic Parameters Between LAD Normal Flow and Slow Flow Group

Variable	Normal Flow Group (n = 30)	Slow Flow Group (n = 47)	Statistical indicator	*P*
AAOD (mm)	32.0 ± 4.4	33.0 ± 3.0	-1.716	0.086
LAD (mm)	34.8 ± 4.3	35.8 ± 5.4	-0.341	0.733
LVD (mm)	46.4 ± 4.1	44.6 ± 4.5	-0.241	0.810
IVST (mm)	10.6 ± 1.5	10.5 ± 1.0	-0.105	0.916
FS (%)	34.8 ± 4.2	34.3 ± 5.7	0.330	0.742
LVEF (%)	64.2 ± 6.0	62.8 ± 8.0	0.739	0.462
WBC (×10⁹/L)	5.82 ± 1.51	5.79 ± 1.44	-0.147	0.883
RBC (×10¹²/L)	4.54 ± 0.56	4.52 ± 0.54	-0.424	0.671
PLT (×10⁹/L)	210.69 ± 48.07	194.61 ± 62.13	-1.861	0.063
HB (g/L)	138.00 ± 14.19	140.8 ± 15.8	-0.310	0.756
NEU (×10⁹/L)	3.69 ± 1.27	3.67 ± 0.94	-0.076	0.939
LYM (×10⁹/L)	1.57 ± 0.56	1.58 ± 0.58	-0.321	0.748
MONO (×10⁹/L)	0.41 ± 0.15	0.42 ± 0.14	-0.403	0.687
EOS (×10⁹/L)	0.15 ± 0.15	0.13 ± 0.10	-0.158	0.874
BASO (×10⁹/L)	0.03 ± 0.01	0.02 ± 0.01	-2.152	0.031
TG (mmol/L)	2.19 ± 1.40	1.71 ± 1.05	-1.229	0.219
TC (mmol/L)	4.68 ± 1.30	4.13 ± 1.24	1.778	0.080
HDL-C (mmol/L)	1.41 ± 0.32	1.39 ± 0.33	0.369	0.713
LDL-C (mmol/L)	2.24 ± 0.92	2.12 ± 0.83	0.581	0.563
UREA (μmol/L)	5.52 ± 1.76	7.06 ± 9.07	-0.875	0.382
CRE (μmol/L)	69.48 ± 20.56	77.17 ± 45.30	-1.019	0.308

*Note: AAOD, Ascending Aortic Diameter; LAD, Left Atrial Diameter; LVD, Left Ventricular Diameter; IVST, Interventricular Septal Thickness; FS, Fractional Shortening; LVEF, Left Ventricular Ejection Fraction; WBC, White Blood Cell Count; PLT, Platelet Count; HB, Hemoglobin; NEU, Neutrophil Count; LYM, Lymphocyte Count; MONO, Monocyte Count; EOS, Eosinophil Count; BASO, Basophil Count; TG, Triglycerides; TC, Total Cholesterol; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; UREA, Urea; CRE, Creatinine; UA, Uric Acid; FT3, Free Triiodothyronine; FT4, Free Thyroxine; TSH, Thyroid Stimulating Hormone. * Indicates P < 0.05 compared with the normal flow group.*

Variable	Normal Flow Group (n = 30)	Slow Flow Group (n = 47)	Statistical indicator	*P*
UA (μmol/L)	319.64 ± 79.97	324.56 ± 97.07	-0.224	0.823
FT3 (pmol/L)	5.06 ± 0.57	4.42 ± 0.80*	4.106	0.000
FT4 (pmol/L)	14.82 ± 2.08	14.61 ± 2.46	-0.339	0.734
TSH (μIU/L)	2.00 ± 1.56	2.20 ± 1.14	-1.463	0.144

*Note: AAOD, Ascending Aortic Diameter; LAD, Left Atrial Diameter; LVD, Left Ventricular Diameter; IVST, Interventricular Septal Thickness; FS, Fractional Shortening; LVEF, Left Ventricular Ejection Fraction; WBC, White Blood Cell Count; PLT, Platelet Count; HB, Hemoglobin; NEU, Neutrophil Count; LYM, Lymphocyte Count; MONO, Monocyte Count; EOS, Eosinophil Count; BASO, Basophil Count; TG, Triglycerides; TC, Total Cholesterol; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; UREA, Urea; CRE, Creatinine; UA, Uric Acid; FT3, Free Triiodothyronine; FT4, Free Thyroxine; TSH, Thyroid Stimulating Hormone. * Indicates P < 0.05 compared with the normal flow group.*

3.3 Risk Factor Analysis for LAD Slow Flow

Based on the results from Tables 1 and 2, variables with a $P < 0.1$ in the univariate comparison were selected as independent variables for multivariate logistic regression analysis. These included gender, age, smoking, AAOD, PLT, BASO, TC, and FT3, with the presence of slow flow as the dependent variable. The multivariate logistic regression analysis revealed that FT3 was negatively correlated with the occurrence of LAD slow flow, identifying it as a protective factor (Table 3).

Table 3
Logistic Regression Analysis of Factors Associated with LAD Slow Flow

Variable	Beta	Odds Ratio	P	95% CI
Gender	0.563	1.755	0.600	0.215–14.362
Age	-0.013	0.987	0.778	0.901–1.081
Smoking	-0.335	0.716	0.772	0.075–6.874
AAOD	0.153	1.165	0.139	0.951–1.428
PLT	0.003	1.003	0.679	0.990–1.015
BASO	-41.349	0.000	0.091	0.000-776.999
TC	-0.468	0.626	0.132	0.340–1.152
FT3	-1.359	0.257	0.003	0.103–0.639
Constant	5.344	209.403	0.201	-

Note: AAOD, Ascending Aortic Diameter; PLT, Platelet Count; BASO, Basophil Count; TC, Total Cholesterol; FT3, Free Triiodothyronine.

3.4 Correlation Analysis Between CTFC and FT3

Spearman correlation analysis was performed to evaluate the relationship between CTFC and FT3 levels. The results demonstrated a significant negative correlation between CTFC and FT3 (r = -0.414, *P* < 0.001).

3.5 ROC Curve Analysis for FT3 in Predicting LAD Slow Flow

A Receiver Operating Characteristic (ROC) curve was constructed to evaluate the sensitivity and specificity of FT3 in predicting LAD coronary slow flow (Fig. 1). The area under the curve (AUC) was 0.749 (95% CI: 0.641–0.858, *P* < 0.001). The optimal cutoff value for FT3 was determined to be 4.69 pmol/L, yielding a sensitivity of 83.3% and a specificity of 68.1%. These results suggest that plasma FT3 levels possess predictive capability for the occurrence of LAD coronary slow flow.

4. DISCUSSION

This study provides novel evidence of a significant independent association between serum free triiodothyronine (FT3) and the presence of CSF in left anterior descending artery (LAD). Our findings indicate that lower FT3 levels are a distinctive feature in LAD-CSF patients, even after adjusting for confounding risk factors.

Many factors are believed to involve in the formation of CSF in previous studies. Beyond traditional atherosclerotic risk factors, CSF has been consistently linked with endothelial dysfunction, as evidenced by impaired flow-mediated dilation and altered biomarkers like asymmetric dimethylarginine[10, 11]. Chronic, low-grade inflammation marked by elevated high-sensitivity C-reactive protein (hs-CRP) and interleukin levels is another cornerstone in CSF pathophysiology[12]. Furthermore, abnormalities in platelet function and coagulation, increased homocysteine levels, and autonomic nervous system dysregulation have been reported as associated factors[13–15]. Genetic predispositions and specific anatomical variations in coronary arteries may also contribute[1, 16]. The association of low FT3 with CSF introduces a novel endocrine-metabolic dimension to this multifactorial condition, suggesting a potential interplay between thyroid hormone status and these established pathways.

The potential link between thyroid hormones and coronary microvascular function offers a plausible biological framework for our observations. Thyroid hormones, particularly T3, exert profound effects on the cardiovascular system, including the regulation of vascular tone, endothelial function, and myocardial metabolism[17, 18]. FT3, the biologically active form, plays a crucial role in modulating nitric oxide (NO) synthesis and bioavailability in endothelial cells [19, 20]. A state of low FT3, often termed "low T3 syndrome" or euthyroid sick syndrome in non-thyroidal illnesses, may contribute to coronary microvascular dysfunction—a key pathological substrate of CSF[21]. The proposed mechanisms include: 1) Endothelial Dysfunction: Reduced FT3 can impair endothelial NO synthase (eNOS) activity, leading to decreased NO production and subsequent vasodilation impairment in the resistance arterioles[22]. 2) Increased Vascular Resistance: Thyroid hormones influence vascular smooth muscle cell relaxation.

Low FT3 levels may enhance vascular sensitivity to vasoconstrictors like endothelin-1 and attenuate beta-adrenergic receptor responsiveness, promoting microvascular constriction[17, 23]. 3) Systemic Inflammation and Oxidative Stress: Low FT3 is frequently associated with a pro-inflammatory state and increased oxidative stress, both of which are implicated in endothelial injury and microvascular dysfunction[24, 25]. These pathways collectively could slow the distal progression of contrast medium in the LAD, manifesting as CSF on angiography. Our findings extend previous research on CSF determinants.

Intriguingly, our data revealed that basophil count was higher in the LAD normal flow group compared to the slow flow group, a finding that contrasts with prior reports. While Soylu et al. demonstrated elevated basophil count in CSF patients ($\mathrm{n} = 78$) versus controls $(\mathrm{n} = 61, \mathrm{p} = 0.002)$. Memioğlu et al. found no significant intergroup difference in a larger cohort ($233 \text{ vs. } 229, \mathrm{p} = 0.401$).

Intriguingly, our data revealed that basophil counts were higher in the LAD normal flow group compared to the slow flow group. This finding contrasts with prior reports by Soylu et al.[26], who demonstrated elevated basophil counts in CSF patients. However, Memioglu et al.[27] found no significant intergroup difference in a larger cohort. These discrepancies may stem from variations in patient selection or sample size. Mechanistically, basophils are hypothesized to influence coronary blood flow through granule-derived mediators, immune activation, and viscosity-inflammation synergism[26, 27]. Given these conflicting results, further prospective studies are warranted to validate the precise role of basophils in CSF pathophysiology.

In this study the left anterior descending coronary artery was selected as the research subject, because it is the primary vessel supplying blood to the left ventricle with prevalence of 90% in CSF[8]. Furthermore, almost the anterior descending coronary artery over 80% of patients reaches the apex of the heart[28]. So, the length of the LAD exhibits relatively small variation among patients, regardless of whether the coronary circulation is right-dominant or left-dominant, which helps enhance the phenotypic homogeneity of our cohort, reducing confounding from diffuse or multi-vessel flow abnormalities. Second, to our knowledge, this is the first clinical report to identify a significant independent relationship between serum FT3 levels and isolated LAD-CSF, proposing a new potential player in its complex etiology. Third, this insight may open new therapeutic avenues. If confirmed, investigating whether thyroid hormone supplementation or modulation (in euthyroid individuals) could ameliorate microvascular dysfunction presents a compelling direction for future interventional studies[29].

This study has limitations. The cross-sectional design prevents establishing causality between low FT3 and LAD-CSF. Despite adjusting for confounders, residual bias from unmeasured variables (e.g., inflammation, insulin resistance) remains possible. The single-center design and reliance on TIMI frame count—an operator-dependent measure lacking coronary flow reserve data—limit the robustness of our conclusions. Furthermore, the absence of longitudinal follow-up restricts insights into the dynamic interplay between thyroid status and microvascular function. External validation in larger, multicenter cohorts is necessary to confirm these preliminary observations.

5. CONCLUSION

In conclusion, this study identifies lower serum FT3 as an independent correlate of isolated slow coronary flow in the LAD. While further large-scale, prospective, and mechanistic studies are required to validate causality, our findings highlight FT3 as a novel biomarker of interest and may inform future therapeutic strategies targeting coronary microvascular dysfunction.

Declarations

Consent for Publication

Not applicable.

CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest.

Ethics approval and consent to participate

Ethical approval was granted by the Ethics Committee of Xiaogan First People's Hospital. Informed consent was obtained from all participants. This study was performed in accordance with relevant guidelines and regulations.

FUNDING INFORMATION

Supported by the Research Projects of Xiaogan First People's Hospital (Grant No. XYKJ-2023-002).

AUTHOR CONTRIBUTIONS

Jinpeng Zhong: Data curation and analysis(equal); investigation (equal); project administration (equal); writing – original draft (lead). Liwu Dai: Data curation (equal); formal analysis (equal); project administration (equal). Xiaohong Lu: Data curation (equal); project administration (equal). Jianzhong Ruan: Data curation (equal); project administration (equal). Yazhou Li: investigation (equal); project administration (equal); Yan Lu: investigation (equal); project administration (equal). Yunqing Chen: writing – review and editing (equal).

DATA AVAILABILITY STATEMENT

All raw data are available upon request. For access to the data, please contact Jinpeng Zhong at *jpzhong2005_@126.com*.

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Figures

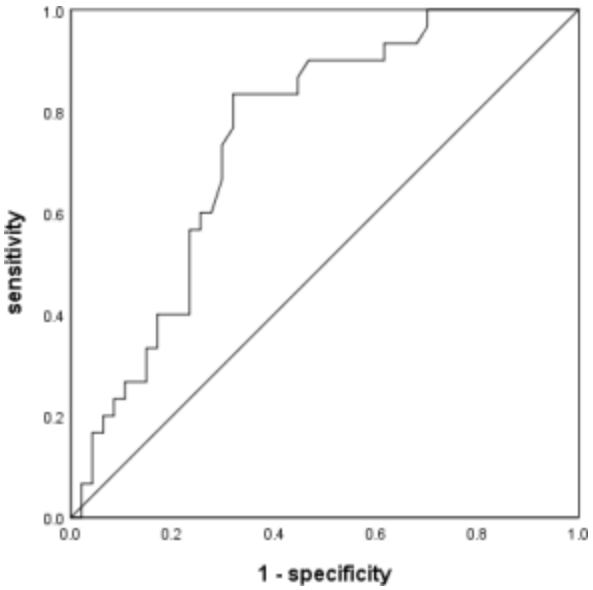


Figure 1

ROC Curve of FT3 for Predicting the Occurrence of LAD Slow Flow