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Prognostic significance of free triiodothyronine levels in alpine region patients undergoing drug-coated balloon therapy for coronary heart disease

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Abstract

Objective This study retrospectively analyzed the relationship between serum-free triiodothyronine (FT3) levels and the prognosis of coronary atherosclerotic cardiopathy (CHD) in patients from alpine regions treated with drug-coated balloons (DCB).

Methods Data from 201 CHD patients with DCB at Hulunbuir People's Hospital between September 2019 and August 2023 were included. Patients were divided into two groups based on the occurrence of major adverse cardiovascular events (MACE) after surgery. Univariate and multivariate logistic regression analyses were conducted to identify risk factors. The predictive efficiency of these risk factors for MACE was evaluated using the ROC curve.

Results The poor prognosis group had significantly higher ages, a greater proportion of patients with a history of previous coronary interventions, and elevated levels of N-terminal pro-B-type natriuretic peptide compared to the good prognosis group. In contrast, FT3 levels were significantly lower ($P < 0.05$). No significant differences were observed in surgical parameters such as DCB target lesion site, lesion length, or puncture approach between the groups ($P > 0.05$). Multivariate binary logistic regression analysis identified FT3 level as an independent predictor factor of MACE in CHD patients treated with DCB. The optimal cut-off value for FT3 in predicting adverse prognosis following DCB surgery was 3.30 pmol/L, with a sensitivity of 72.5%, specificity of 62.8%, and an area under the curve (AUC) of 0.741 ($P < 0.05$).

Conclusion Decreased FT3 levels serve as a biomarker for predicting the occurrence of MACE in patients from alpine regions undergoing DCB treatment for CHD. There is a significant correlation between reduced FT3 levels and the incidence of MACE in these patients.

Keywords Coronary atherosclerotic cardiopathy, Drug-coated balloons, Free triiodothyronine, Prognosis

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Introduction

Coronary atherosclerotic cardiopathy (CHD) currently affects an estimated 11 million individuals in China, with the prevalence to rise [1]. Established risk factors for CHD include advanced age, male gender, hypertension, diabetes, dyslipidemia, smoking, and a family history of early-onset CHD [2, 3]. Recent studies have highlighted the influence of environmental temperature on cardiovascular health, with fluctuation linked to CHD incidence and mortality rates, indirectly heightening health risks through various mechanisms [4]. During cold winters, cardiac death rates peak, particularly among elderly and male patients [5]. Hulunbuir City, located in Inner Mongolia in the northeastern China, experiences harsh winter with significant annual temperature fluctuations and frequent cold waves, representing a regional meteorological challenge [6]. As a result, investigating CHD prevention and treatment in this region provides valuable insights for managing CHD in the alpine areas of China.

Current CHD management includes both pharmacological and revascularization strategies, with percutaneous coronary intervention (PCI) being widely used in clinical settings [2]. The introduction of drug-eluting stents (DES) has greatly improved the symptoms and prognosis in CHD patients. However, challenges like small vessel disease (SVD), side branch lesions (SB), and in-stent restenosis (ISR) remain sub-optimally addressed by stent deployment [7]. Recently, drug-coated balloons (DCB) have emerged as a pivotal treatment modality in interventional cardiology, offering enhanced management for SVD, SB, and ISR, thereby benefiting a broader patient population [8]. Therefore, identifying high-risk groups for DCB treatment through laboratory testing and preemptively assessing postoperative complications in CHD patients treated with DCB is crucial.

Thyroid hormones (TH) play a crucial role in regulating growth, development, and metabolism, significantly impacting the circulatory system. Free triiodothyronine (FT3), the most potent hormone in the TH group, regulates cardiac contractions, heart rate, and blood pressure [9, 10]. Reduced FT3 levels have been associated with arteriosclerosis and an increased incidence of CHD, negatively affecting its prognosis [11]. Additionally, FT3 has been identified as a significant predictor of cardiac mortality [12]. Despite this knowledge, no domestic studies have yet assessed how variations in FT3 levels in alpine regions may impact the prognosis of patients undergoing DCB treatment for CHD.

Materials and methods

Study population

A retrospective analysis was conducted on 206 patients who underwent DCB treatment for CHD in the cardiology department of Hulunbuir People's Hospital from September 2019 to August 2023. Data were collected from 201 patients who met the inclusion and exclusion criteria (5 patients were excluded), with effective follow-up conducted in 191 cases (127 males and 64 females), while 10 cases were lost to follow-up.

Prior to the study, we conducted a small observational analysis, which suggested a possible correlation between decreased FT3 levels and MACE. Based on this observation, we calculated the required sample size using SPSS 21 software for comparison of means between two groups. The preliminary investigation showed that the mean FT3 levels in the two groups were 4.3 and 3.8, with standard deviations of approximately 0.9. Setting the type I error (α) at 0.05, the type II error (β) at 0.2, and an expected adverse event rate of 20%, the minimum required sample size was calculated to be 163 participants. Accounting for a potential 20% data loss, the final required sample size was determined to be 205 participants.

Inclusion and exclusion criteria

Inclusion criteria

1. Patients with a history of CHD or those diagnosed with CHD via coronary angiography who underwent DCB expansion surgery.
2. Intraoperative lesion treatment was deemed satisfactory, meeting the standards outlined in the Chinese Expert Consensus on Clinical Application of Drug-Coated Balloons [13].
3. All participants provided informed consent and signed consent form to participate in the study.
4. Participants were aged 18 years or older.

Exclusion criteria

1. Patients requiring remedial stent placement due to intraoperative dissection.
2. Patients diagnosed with subclinical hypothyroidism, subclinical hyperthyroidism, hyperthyroidism, or those receiving thyroid hormone medication and other iodine-containing preparations upon admission.
3. Patients with severe infections, significant cardiac, liver, or kidney dysfunction, hematological disorders, malignant tumors, or sepsis.
4. Patients with a life expectancy of less than one year.

Data collection

General information for the 191 patients was documented, including name, age, gender, contact details, hospital ID, diagnosis, medical history (including hypertension, diabetes, smoking, previous coronary

interventions, and prior myocardial infarction), as well as surgical dates. Examination data include thyroid function tests (TSH, FT3, and FT4), complete blood count, coagulation profile, comprehensive biochemistry, homocysteine levels, glycated hemoglobin (for patients with abnormal blood sugar or diabetes), cardiac biomarkers, N-terminal pro-B-type natriuretic peptide levels, and preoperative echocardiographic ejection fraction assessment. Surgical details comprised target vessel location, lesion length, percentage of lesion stenosis, puncture approach, DCB dimensions (length and diameter), and calculation of the Gensini score (GS score) for each patient [14].

Treatment standards

Preoperative examination and oral medication regimen

Before enrollment, the aforementioned pre-operative examinations were conducted. Patients were informed about their condition, surgical objectives, and associated risks before signing the informed consent form for surgery. All patients underwent a standardized dual antiplatelet therapy (DAPT) regimen pre-operatively, which included oral administration of enteric-coated aspirin 100 mg with a loading dose of 300 mg once daily before surgery, alongside clopidogrel bisulfate 75 mg once daily with a loading dose of 300 mg before surgery, or ticagrelor 90 mg twice daily with a loading dose of 180 mg before surgery.

Surgical procedure

During coronary angiography, patients suitable for DCB intervention are carefully selected. The initial step involves dilation using a traditional or semi-compliant balloon, maintaining a balloon-to-artery diameter ratio of 0.8 to 1.0 and applying moderate pressure (typically 8 to 14 atmospheres, where 1 atmosphere equals 101.325 kPa) to minimize the risk of dissection. If initial dilation is inadequate, a non-compliant or cutting balloon may be used for further pre-dilation. After achieving sufficient pre-dilation, the outcome is assessed to determine eligibility for DCB treatment. DCB intervention proceeds under the following conditions: absence of dissection or presence of type A or B dissection; achievement of TIMI flow grade 3; residual stenosis $\leq$30%. The selected DCB diameter should match the vessel diameter (recommended diameter ratio of 0.8-1.0), and dilation maintained for 60 s at 10 atmospheres of pressure. To ensure adequate coverage between the treated area or stent and the DCB, the DCB must extend 2 to 3 mm beyond each end of the pre-treated area. Additionally, the DCB must reach the lesion site within 2 min of entering the body to prevent inadequate treatment.

Postoperative management and oral medication regimen

Following surgery, patients receive pressure bandaging at the radial artery puncture site for 4 to 6 h, with close monitoring for hematoma formation and any improvements in preoperative CHD symptoms, as well as monitoring for bleeding or melena. All patients are required to continue DAPT for at least one month post-surgery, as recommended by expert consensus (oral intake is recommended for 12 months) to prevent thrombosis formation [13]. Postoperative electrocardiograms and urinalysis are also performed to ensure comprehensive post-surgical care.

Follow-up records

Follow-up for patients undergoing DCB treatment for CHD is conducted through three methods: phone calls, outpatient clinic visits, and readmission records. The follow-up period ranges from six months to two years post-surgery. Patients are categorized based on prognosis: those readmitted for recurrent angina requiring further coronary imaging assessments (including coronary angiography or coronary artery enhanced CT) to evaluate restenosis at the target lesion site post-DCB are classified into the poor prognosis group; if no restenosis requiring intervention is observed during follow-up imaging assessments, they are categorized into the good prognosis group. Patients experiencing other major adverse cardiovascular events (MACE)-such as recurrent acute myocardial infarction (AMI), post-DCB target lesion revascularization (TLR), cardiac death, malignant arrhythmia, and heart failure-are also categorized into the poor prognosis group (n=40). The remaining patients are defined as the control group or the good prognosis group (n=151). Statistical analysis is conducted using R software version 4.2.0 to compare the correlation between FT3 levels and prognosis in the poor and good prognosis groups.

Statistical methods

Data analysis and graph plotting are conducted using R software version 4.2.0. For categorical data such as age, TSH, FT3, FT4, and platelet count, c2 tests are used. For measurement data that follows a normal distribution, such as gender, history of hypertension, diabetes, and previous coronary interventions, t-tests are applied. For data that does not follow a normal distribution, such as the Gensini score, the Kruskal-Wallis test (a non-parametric test) is employed. Both univariate and multivariate logistic regression analyses are conducted to assess the observed indicators, and ROC curves are plotted to predict the potential impact of these indicators on patient prognosis. A P-value of <0.05 is considered statistically significant.

Results

Comparison of general clinical data between groups

Patients were classified into a good prognosis group (151 cases) and a poor prognosis group (40 cases) based on the occurrence of MACE following DCB treatment for CHD. Statistically significant differences between the groups were observed for age, history of coronary intervention, FT3 levels, N-terminal pro-B-type natriuretic peptide measurements, and GS scores ($P<0.05$). However, no significant differences were found between the groups regarding gender, presence of hypertension, diabetes, history of myocardial infarction, smoking history, diagnostic results, platelet count, TSH and FT4 levels, fibrinogen, high-density lipoprotein, low-density lipoprotein, uric acid, triglycerides, total cholesterol, cardiac injury markers, homocysteine, glycated hemoglobin, and echocardiographic ejection fraction (Table 1).

Comparison of surgery-related data between the two groups

A univariate analysis comparing the surgical data of the two groups revealed no significant differences in lesion length, lesion stenosis percentage, puncture approach, DCB length, and DCB diameter (Table 2).

Logistic regression analysis of risk factors in the poor prognosis group

A univariate binary logistic regression analysis was conducted, with the occurrence of MACE post-DCB as the dependent variable. The analysis showed significant differences in age, history of coronary intervention, FT3 level, N-terminal pro-B-type natriuretic peptide measurement, diagnosis, and GS scores ($P<0.05$) (Table 3). Subsequent multivariate logistic regression analysis indicated that the FT3 level and history of coronary intervention of a patient independently predict the risk of MACE following DCB treatment for CHD (Table 4).

Comparison of adverse prognosis events between high FT3 level group and low FT3 level group

An ROC model was constructed using FT3 as a key biomarker (Fig. 1). The results indicated that the cut-off value for FT3 in predicting the occurrence of MACE post-DCB treatment for CHD is 3.30 pmol/L, with a

Table 1 A comparison of general clinical data and biochemical markers between groups with good and poor prognosis

Observational indicator	Good prognosis group ($n=151$)	Poor prognosis group ($n=40$)	t/X^2	*P*
Age (years, mean ± s)	59 ± 9.7	65 ± 14	-2.483	0.017
Gender [*n* (%)]			0.624	0.430
Male	103(68.2%)	24(60.0%)		
Female	48(31.8%)	16 (40.0%)		
History of high blood pressure [*n* (%)]	111(73.5%)	34(85.0%)	1.698	0.193
History of diabetes [*n* (%)]	55(36.4%)	21(52.5%)	2.773	0.096
Previous coronary intervention [*n* (%)]	45(29.8%)	21(52.5%)	6.236	0.013
Previous myocardial infarction [*n* (%)]	30(19.9%)	10(25.0%)	0.241	0.624
Smoking history [*n* (%)]	98(64.9%)	23(57.5%)	0.461	0.497
Diagnosis [*n* (%)]			7.063	0.070
Acute non-ST elevation myocardial infarction	22(14.6%)	7(17.5%)		
Acute ST elevation myocardial infarction	30(19.9%)	1(2.5%)		
Stable angina	20(13.2%)	7(17.5%)		
Unstable angina	79(52.3%)	25(62.5%)		
TSH (uIU/ml, mean ± *s*)	2.8 ± 2.6	5.3 ± 15	− 1.053	0.299
FT3(pmol/L, mean ± *s*)	4.4 ± 0.72	4. 1 ± 0.90	2.013	0.049
FT4(pmol/L, mean ± *s*)	16 ± 2.5	15 ± 3.0	0.540	0.592
Platelet count (× 10^9/L, mean ± s)	210 ± 52	220 ± 70	-0.646	0.521
Fibrinogen (g/L, mean ± s)	3. 1 ± 0.90	3.2 ± 0.70	-0.721	0.473
Uric acid (μmol/L, mean ± s)	340 ± 85	330 ± 91	0.290	0.773
Triglycerides (mmol/L, mean ± s)	1.9 ± 1.2	1.8 ± 0.78	1.007	0.317
Total cholesterol (mmol/L, mean ± s)	4. 1 ± 1. 1	4. 1 ± 1.3	0.040	0.969
High-density lipoprotein (mmol/L, mean ± s)	1.0 ± 0.29	1.0 ± 0.20	-0.458	0.648
Low-density lipoprotein (mmol/L, mean ± s)	2.5 ± 0.95	2.5 ± 0.97	0.329	0.743
Cardiac injury markers (ng/L, mean ± s)	320 ± 810	200 ± 360	1.314	0.191
N-terminal pro b-type natriuretic peptide (pg/ml, mean ± s)	330 ± 570	900 ± 1400	-2.476	0.017
Homocysteine (μmol/L, mean ± s)	16 ± 10	16 ± 8.0	0.067	0.947
Glycated hemoglobin (%, mean ± s)	2.9 ± 4.0	3.9 ± 3.7	− 1.468	0.147
Ejection fraction (%, mean ± s)	62 ± 7.4	61 ± 7.8	0.743	0.460
Gensini score (mean ± s)	40 ± 25	44 ± 22	-0.986	0.032

Table 2 Comparison of surgery-related data between the good and poor prognosis groups

Observational indicator	Good prognosis group ($n=151$)	Poor prognosis group ($n=40$)	t/X^2	P
Target vessel [*n* (%)]			3.9367	0.268
Left anterior descending	34(22.5%)	15(37.5%)		
Left circumflex	1(0.7%)	0(0%)		
Right coronary artery	17(11.3%)	4(10.0%)		
Other	99(65.6%)	21(52.5%)		
Lesion length (mm, mean ± s)	24 ± 4.6	25 ± 4.3	-0.953	0.344
Lesion stenosis percentage (%, mean ± s)	96 ± 5.8	96 ± 5.0	-0.312	0.756
Puncture approach [*n* (%)]			0.000	1
Right radial artery	150(99.3%)	40(100%)		
Left radial artery	1(0.7%)	0(0%)		
DCB length (mm, mean ± s)	24 ± 4.6	24 ± 5.5	-0.049	0.961
DCB diameter (mm, mean ± s)	2.4 ± 0.39	2.4 ± 0.36	− 1.134	0.261

Table 3 Univariate binary logistic regression analysis of observational indicators

Observational indicator	Reference indicator	B	Wald	OR value 95% confidence interval	P
Gender (female)		0.358	0.952	1.431(0.688 ~ 2.923)	0.329
Age (years)		0.054	8.698	1.056(1.02 ~ 1.097)	0.003
History of high blood pressure	No	0.714	2.215	2.042(0.847 ~ 5.724)	0.137
History of diabetes	No	0.657	3.351	1.929(0.955 ~ 3.927)	0.067
Previous intervention history	No	0.957	6.941	2.604(1.279 ~ 5.347)	0.008
History of myocardial infarction	No	0.296	0.501	1.344(0.571 ~ 2.985)	0.479
Smoking history	No	0.312	0.743	0.732(0.361 ~ 1.504)	0.389
		0.045	1.284	1.046(0.995 ~ 1. 171)	0.257
		-0.83	11.886	0.436(0.265 ~ 0.685)	< 0.001
		-0.041	0.356	0.96(0.838 ~ 1.098)	0.550
Platelets ($\times 10^9$/L)		0.002	0.587	1.002(0.996 ~ 1.008)	0.443
Fibrinogen (g/L)		0.126	0.392	1. 135(0.754 ~ 1.673)	0.531
Uric acid (μmol/L)		-0.001	0.091	0.999(0.995 ~ 1.003)	0.762
Triglycerides (mmol/L)		-0. 144	0.645	0.866(0.589 ~ 1.201)	0.422
Total cholesterol (mmol/L)		-0.007	0.002	0.993(0.728 ~ 1.339)	0.966
High-density lipoprotein (mmol/L)		0.23	0.139	1.259(0.335 ~ 4.04)	0.710
Low-density lipoprotein (mmol/L)		-0.063	0. 112	0.939(0.642 ~ 1.353)	0.738
Cardiac injury markers (ng/L)		0	0.733	1(0.999 ~ 1)	0.392
N-terminal pro B-type natriuretic peptide (pg/mL)		0.001	10.436	1.001(1 ~ 1.001)	0.001
Homocysteine (μmol/L)		-0.001	0.003	0.999(0.957 ~ 1.033)	0.953
Glycated hemoglobin (%)		0.061	1.918	1.063(0.974 ~ 1. 158)	0.166
Ejection fraction (%)		-0.018	0.589	0.983(0.94 ~ 1.03)	0.443
Gensini score		0.006	0.842	1.006(0.992 ~ 1.02)	0.035
Stable angina	Acute non-ST elevation myocardial infarction	-2.256	4.166	0. 105(0.005 ~ 0.648)	0.041
Acute ST elevation myocardial infarction		0.095	0.024	1. 1(0.323 ~ 3.753)	0.877
Unstable angina		-0.005	0	0.995(0.393 ~ 2.76)	0.991

sensitivity of 72.5%, specificity of 62.8%, and an AUC of 0.741. Patients with FT3 levels above 3.30 pmol/L were classified into the high-level group, while those with levels below this threshold were placed in the low-level group. A statistically significant difference in MACE occurrence post-DCB was observed between the high and low-level groups ($P<0.05$) (Table 5).

Discussion

FT3 is the primary active hormone among TH. In many non-thyroidal illnesses, a decrease in FT3 while TSH and FT4 levels remain normal is known as low triiodothyronine syndrome (LT3S), with its incidence increasing with age [15]. Over the past decades, numerous studies have explored the relationship between LT3S and the cardiovascular disease prognosis; however, research on the

Table 4 Multifactorial binary logistic regression analysis of observational indicators

Observational indicator	Reference indicator	B	Wald	OR value 95% confidence interval	*P*
Age (years)		0.034	3.116	1.034(0.997 ~ 1.076)	0.078
Previous coronary intervention history	No	0.956	5.307	2.602(1.153 ~ 5.924)	0.021
FT3(pmol/L)		-0.582	4.176	0.559(0.311 ~ 0.967)	0.041
N-terminal pro B-type natriuretic peptide (pg/mL)		0	2.994	1(1 ~ 1.001)	0.084
Stable angina	Acute non-ST elevation myocardial infarction	– 1.633	1.965	0. 195(0.009 ~ 1.429)	0.161
Acute ST elevation myocardial infarction		-0.261	0. 124	0.77(0.169 ~ 3.239)	0.724
Unstable angina		0.387	0.457	1.473(0.5 ~ 4.863)	0.499

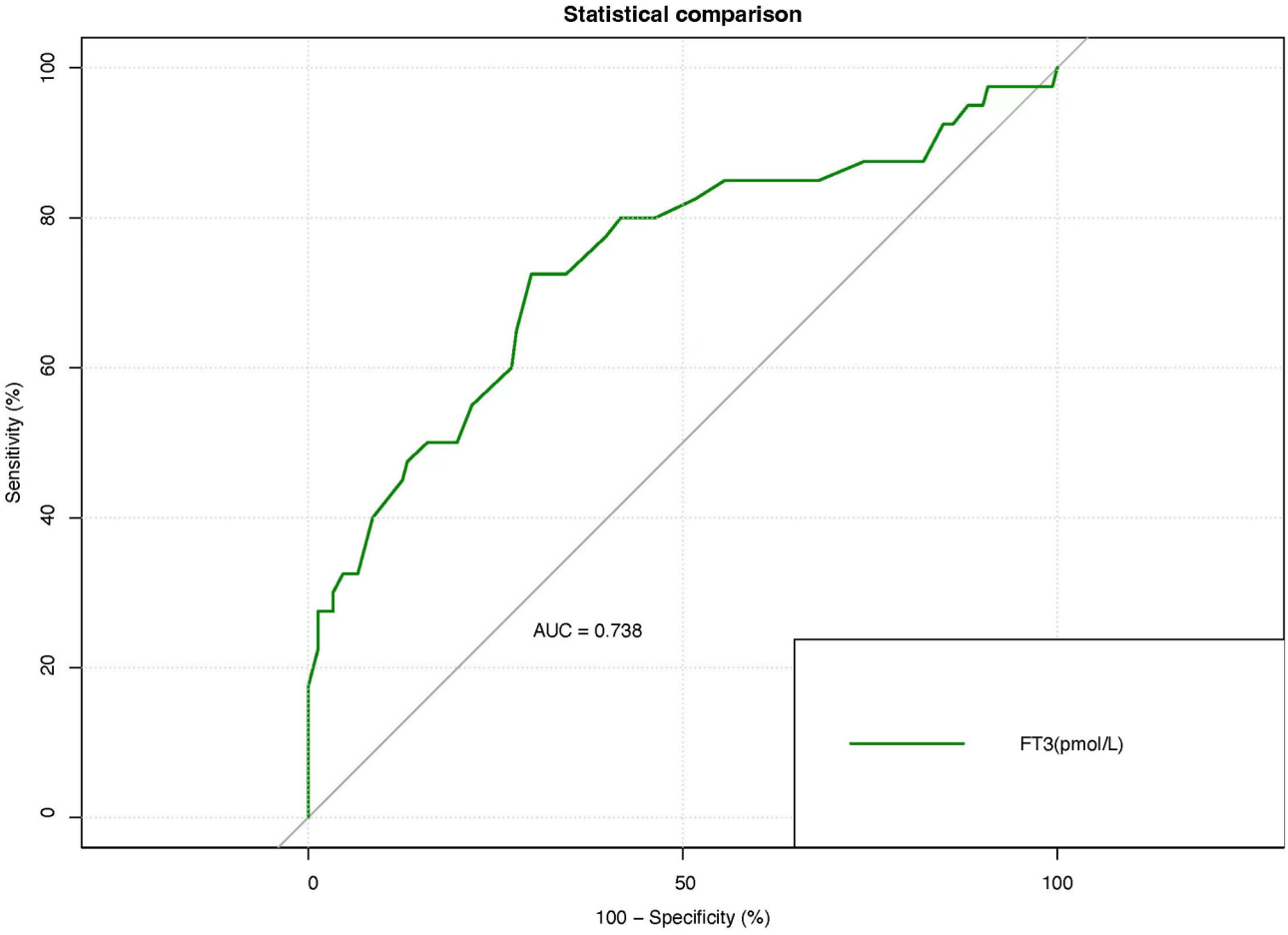

Fig. 1 The ROC curve for FT3 predicting adverse prognosis events following DCB treatment

Table 5 Comparison of adverse prognosis events in high and low FT3 level groups

	High FT3 level group (*n* = 111)	Low FT3 level group (*n* = 90)	X^2	*P*
Poor prognosis group/*n* (%)	21(18.9)	19(21. 1)	4.086	0.043

impact of reduced FT3 on the outcomes of DCB treatment for patients with CHD is still relatively limited. Studies have indicated that decreased FT3 may elevate the risks of restenosis and thrombosis following DCB treatment and is closely related to the severity of coronary stenosis [16–21]. These findings suggest that a reduction in FT3 may be a risk factor for complications after arterial balloon dilation treatment for CHD. Additionally, a decrease in FT3 can promote arteriosclerosis through various mechanisms, including effects on lipid metabolism, alterations in arterial smooth muscle structure, vascular wall thickening, decreased compliance, endothelial dysfunction, a hypercoagulable state, impaired fibrinolytic function, hyperhomocysteinemia, systemic

inflammation, and platelet dysfunction [2–27]. Furthermore, lower FT3 levels are associated with an increased risk of cardiac death and serves as an important predictive marker [28, 29]. To validate the impact of decreased FT3 on the efficacy of DCB treatment, this study established a predictive model for FT3 levels and the risk of MACE post-DCB treatment for CHD. The study confirmed that the risk of MACE significantly increases in a patient when FT3 levels fall below 3.30 pmol/L. This finding aligns with previous studies and provides strong support for predicting major adverse events postoperatively in CHD patients within clinical settings.

Severe fluctuations in environmental temperature can affect FT3 levels and contribute to the development of various diseases, including cardiovascular disease. Some studies suggest that short-term exposure to cold can elevate TH and FT3 levels, viewed as a compensatory response to increase heat production [30, 31]. However, observations of long-term changes in endocrine levels in individuals living in polar environments have found a decrease in FT3, with no significant changes detected in FT4 and TSH levels. Pathogenic analysis indicates that prolonged exposure to cold significantly raises both the internal production and clearance rates of FT3 [32–34]. The study area, Hulunbuir City, situated near the inland arctic region of China, frequently experiences polar cyclones, suggesting that residents exposed to cold conditions may similarly experience fluctuations in FT3 levels.

The impact of temperature changes on the circulatory system primarily manifests through alterations in vasomotor status, leading to fluctuations in blood pressure and contributing to cardiovascular diseases. Research reveals that while cardiovascular events can occur at any time, they are more likely to be triggered by extreme weather conditions like cold or hot temperatures [35]. Studies from various regions show that the highest incidence rates of acute coronary syndrome occur during the cold winter season [36, 37]. However, cold temperatures are not the only significant factor affecting cardiovascular health, and their impact remains controversial. Some studies report varying degrees of cardiovascular effects due to cold, with more pronounced impact observed in the incidence of cardiac arrest and acute myocardial infarction (AMI), suggesting the influence of additional related variables [38–43]. Further research indicates that human blood pressure, along with HDL, LDL, and glucose, tends to be slightly elevated in winter compared to summer [44, 45]. Additionally, research shows that individuals with dyslipidemia treated with statin medications achieve target LDL levels more readily in summer than in winter, suggesting that seasonal changes may affect lipoprotein metabolism [46]. Beyond hemodynamic changes caused by temperature shifts, cold weather can also alter arteriosclerotic risk factors, potentially destabilizing vulnerable plaques in individuals at high risk for cardiovascular disease. This increases the likelihood of plaque rupture and occlusive thrombosis, thus precipitating acute cardiovascular events.

Residents of alpine regions not only experiences endocrine changes triggered by temperature fluctuations but also increased cardiovascular risks due to vasoconstriction directly caused by cold exposure. Furthermore, the tendency to consume higher-fat diets during winter to combat severe colds, combined with reduced physical activity due to limited outdoor engagement, further heightens the risk of CHD. Therefore, comprehensive strategies for CHD prevention and control in alpine regions should address climate influences, lifestyle habits, and social adaptation. Implementing measures such as intensified health education, promoting healthier dietary choices, encouraging lifestyle modifications, and advocating for moderate indoor exercise during winter months are crucial. The results from this study on patients residing in Hulunbuir City demonstrate that decreased FT3 levels are associated with an increased incidence of MACE following DCB treatment in patients living in alpine regions. In future clinical practice, ongoing monitoring of FT3 levels in similar patients could serve as a valuable prognostic indicator, complementing the aforementioned measures aimed at mitigating the progression of stenotic lesions. Such efforts are essential for enhancing survival rates and improving the quality of life for patients with CHD residing in alpine regions.

The objective of this study was to retrospectively assess the relationship between FT3 levels in the alpine region and prognosis after DCB treatment for CHD. However, several limitations should be acknowledged. First, the sample size is relatively small due to regional restrictions, and the follow-up observation period for patients is limited. Second, the study involved only a single measurement of thyroid function at the time of patient admission, lacking dynamic observation of FT3 levels over time. The research team plans to address these limitations in future studies by continuously monitoring the prognosis of enrolled patients, particularly through coronary angiography to assess progression at the DCB target lesion site and corresponding changes in FT3 levels. Third, the study cannot conclusively determine a causal relationship between elevated FT3 levels and the occurrence of MACE following DCB treatment for CHD. To investigate this further, the team intends to conduct mechanistic animal model studies using mouse models lacking the FT3 factor. Finally, the current clinical study primarily analyzed the correlation between FT3 and various factors post-DCB treatment for CHD, without comparing results to a healthy (negative control) group or further stratifying patient risk factors for comparison. Future research

will aim to fill these gaps by including a larger group of clinical patients and incorporating comprehensive comparative analyses. Despite these limitations, our study is one of the first to focus on FT3 levels as a predictor of adverse outcomes in patients from alpine regions undergoing DCB treatment, offering valuable insights into better identifying and managing high-risk patients in this unique setting.

Conclusion

Reduced FT3 levels are identified as a biomarker for predicting adverse prognostic events in patients from alpine regions undergoing DCB treatment for CHD. There is a significant correlation between decreased FT3 levels and the incidence of MACE following DCB treatment for CHD in patients from alpine regions.

Abbreviations

DCB	Drug-coated balloons
FT3	Free triiodothyronine
CHD	Coronary heart disease
AMI	Acute myocardial infarction
TLR	Target lesion revascularization
MACE	Major adverse cardiovascular events
ROC	Receiver operating characteristic
AUC	Area under curve
PCI	Percutaneous coronary intervention
PTCA	Percutaneous transluminal coronary angioplasty
BMS	Bare-metal stents
DES	Drug-eluting stents
SVD	Small vessel disease
SB	Side branch
ISR	In-stent restenosis
TH	Thyroid hormones
DAPT	Dual antiplatelet therapy
GS	Gensini score
LT3S	Low triiodothyronine syndrome

Author contributions

Qin-Bao Zhang: Conceptualization, Data curation, Formal Analysis, Writing – original draft, Writing – review & editing. Gang Wu: Conceptualization, Validation, Writing – original draft, Writing – review & editing. Ze-Ying Wang: Data curation, Formal Analysis, Software. Zhi-Liang Cui: Formal Analysis, Software, Visualization. Hong-Xia Zhang: Data curation, Formal Analysis, Investigation. All authors read and approved the final draft.

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Data availability

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

Declarations

Ethics approval and consent to participate

This study was conducted with approval from the Ethics Committee of Hulunbuir People's Hospital (Approval Number: 2023SYY-11). This study was conducted in accordance with the declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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