RESEARCH

Association of plasma free triiodothyronine levels with contrast-induced acute kidney injury and short-term survival in patients with acute myocardial infarction

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Abstract

Objective: Post-treatment contrast-induced acute kidney injury (CI-AKI) is associated with poor outcomes in patients with acute myocardial infarction (AMI). A lower free triiodothyronine (FT3) level predicts a poor prognosis of AMI patients. This study evaluated the effect of plasma FT3 level in predicting CI-AKI and short-term survival among AMI patients.

Methods: Coronary arteriography or percutaneous coronary intervention was performed in patients with AMI. A 1:3 propensity score (PS) was used to match patients in the CI-AKI group and the non-CI-AKI group.

Results: Of 1480 patients enrolled in the study, 224 (15.1%) patients developed CI-AKI. The FT3 level was lower in CI-AKI patients than in non-CI-AKI patients (3.72 ± 0.88 pmol/L vs 4.01 ± 0.80 pmol/L, $P < 0.001$). Compared with those at the lowest quartile of FT3, the patients at quartiles 2–4 had a higher risk of CI-AKI respectively (P for trend = 0.005). The risk of CI-AKI increased by 17.7% as FT3 level decreased by one unit after PS-matching analysis (odds ratio: 0.823; 95% CI: 0.685–0.988, $P = 0.036$). After a median of 31 days of follow-up (interquartile range: 30–35 days), 78 patients died, including 72 cardiogenic deaths and 6 non-cardiogenic deaths, with more deaths in the CI-AKI group than in the non-CI-AKI group (53 vs 25, $P < 0.001$). Kaplan–Meier survival analysis showed that patients at a lower FT3 quartile achieved a worse survival before and after matching.

Conclusion: Lower FT3 may increase the risk of CI-AKI and 1-month mortality in AMI patients.

Key Words

- acute myocardial infarction
- free triiodothyronine
- contrast-induced acute kidney injury
- propensity score matching analysis
- short-term survival

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Introduction

Thyroid hormone level changes with the development of acute myocardial infarction (AMI) in patients without prior thyroid disease (1, 2, 3). Low triiodothyronine (T3) syndrome (LT3S), manifested as serum T3 level and normal levels of thyroid-stimulating hormone (TSH) and thyroxine (T4), is a common thyroid dysfunction (4, 5). Free T3 (FT3) plays various roles in the cardiovascular system (6, 7). Many publications have reported that a low

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FT3 is associated with a poor prognosis in patients with heart disease (8, 9, 10, 11). This suggests that cardiovascular function declines as FT3 level drops in AMI patients.

In patients receiving percutaneous coronary intervention (PCI), 5.2–59% of them may suffer from contrast-induced acute kidney injury (CI-AKI) (12, 13, 14). CI-AKI is associated with increased dialysis odds and 1-year mortality (15, 16, 17).

However, no studies have been conducted to assess whether lower FT3 could predict CI-AKI risk in patients with aggressive AMI. In this study, we aimed to investigate the association of FT3 level with CI-AKI risk and short-term survival in AMI patients undergoing PCI or coronary arteriography (CAG).

Methods

Study participants

The research program was formulated in accordance with the ethical guidelines of the 1975 Declaration of Helsinki and approved by the Ethics Committee of the Affiliated Changzhou No. 2 People's Hospital of Nanjing Medical University. Each patient signed informed consent before enrollment in this study. This trial was registered in the Chinese Clinical Trials Registry (ChiCTR1800014583).

As shown in Fig. 1, a total of 2381 participants aged over 18 years and diagnosed with AMI between January 2010 and January 2018 in the Affiliated Changzhou Second People's Hospital of Nanjing Medical University were recruited. The diagnosis of AMI was based on symptom, cardiac troponin, renal dysfunction, and electrocardiographic changes (18). A total of 512 patients were excluded because they had (i) refused CAG or PCI therapy; (ii) been diagnosed with myocarditis, pericarditis, valvular disease, severe infection, or old myocardial infarction; (iii) received emergency coronary artery bypass grafting. Of the remaining 1869 patients, 389 patients were further excluded for (i) absence of postoperative creatinine, baseline creatinine, value of FT3; (b) FT3 >6.8 pmol/L; (iii) perioperative dialysis treatment; (iv) past history of thyroid diseases; (v) history of medication for thyroid diseases, or medication causing thyroid dysfunction (e.g. amiodarone, glucocorticoid, interferon-alpha). Finally, 1480 subjects were enrolled.

AKI definition

Baseline serum creatinine level was measured at 24 h before operation. AKI was defined as serum creatinine with an absolute increase ≥0.3 mg/dL (26.4 μmol/L) or ≥ 150% higher than the baseline level within 48 h after CAG or PCI treatment according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria (19). Serum creatinine was detected within 48 h after intervention treatment.

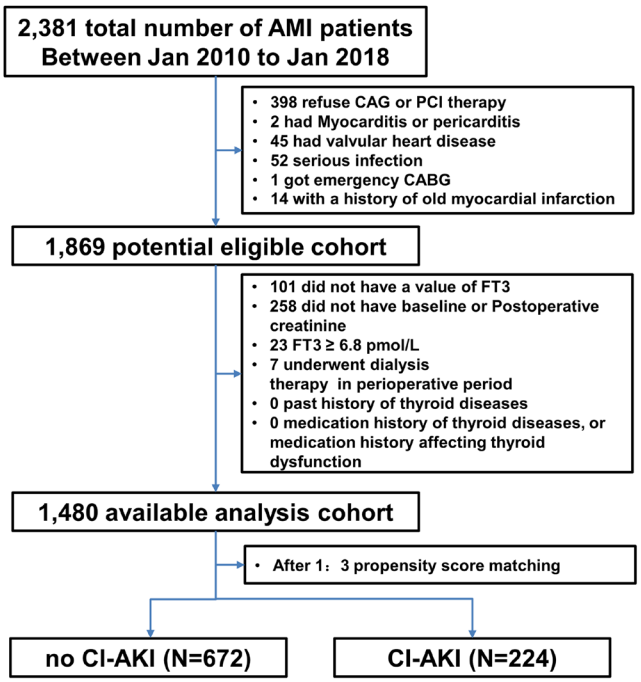

Figure 1
Study flow chart. AMI, acute myocardial infarction; CAG, coronary angiography; PCI, percutaneous coronary intervention.

PCI and CAG

For CAG or PCI, the Seldinger technique was conducted by physicians experienced in digital subtraction angiography. The procedural characteristics were recorded (20).

Blood collection and plasma thyroid hormone assays

Blood was sampled and tested for thyroid hormone within 24 h after AKI onset. Serum TSH level was tested by enhanced chemiluminescence assay (21). The reference range was 3.1–6.8 pmol/L for FT3, 12–22 pmol/L for free thyroxine (FT4), and 0.27–4.2 μIU/mL for TSH in adults. Hyperthyroidism was diagnosed as elevated FT3 and (or) FT4 levels with reduced serum TSH level; hypothyroidism as decreased FT3 and (or) FT4 levels with increased TSH level; subclinical hyperthyroidism as decreased serum TSH level and normal serum FT4 and FT3 levels (22); and subclinical hypothyroidism as elevated serum TSH level and normal FT3 and FT4 levels (23).

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Endpoints

According to the incidence of AKI, patients were divided into the CI-AKI group and the non-CI-AKI group with a propensity score (PS). The abbreviated MDRD equation was used to estimate glomerular filtration rate (eGFR) according to the baseline serum creatinine concentration (24). The primary endpoint was set as the development of CI-AKI after CAG or PCI, and the secondary endpoint was set as all-cause mortality within 30 days after coronary intervention.

Statistical analysis

Continuous variables were expressed as means with S.D. or median and interquartile range 25–75%. Discrete variables were expressed as percentage. Mann–Whitney *U*-test and Student's *t*-test were used to evaluate continuous variables, and chi-squared test and Fisher's exact test were used to evaluate categorical variables about demographic and clinical characteristics.

Restricted cubic spline regression models were used to determine the continuous changes in CI-AKI risk and FT3 level. Multivariate analysis (binary logistic regression), comprising factors of clinical interest and all significant covariates in univariate analysis, was performed to investigate the association between CI-AKI and FT3. The results were presented as odds ratios (OR) and 95% CIs.

After that, a PS-matched cohort was generated to minimize the impact of selection bias and control potential confounding factors. Two similar groups (CI-AKI group and non-CI-AKI group) were constructed. Since there were more patients in the non-CI-AKI group, each patient in the CI-AKI group was matched to three patients in the non-CI-AKI group (1:3 matching). The greedy nearest-neighbor method, with no replacement, was used with a caliper of 0.4 of PS in order to match. Absolute standardized difference (ASD) was used to evaluate the balance of baseline characteristics between the two groups. An ASD of ≤ 0.2 (20%) was regarded as a negligible difference in each covariate between the two groups. When the ASD was greater than 0.15 (15%), the covariate was included in the logistic regression model. The association between FT3 and short-term survival was analyzed by Kaplan–Meier survival analysis and log-rank test. PS-matching analysis was carried out using the statistical package R (the R Foundation; http://www.r-project.org; version 3.4.3).

To maximize the statistical ability and minimize bias that may occur in eliminating missing data in analysis, multivariate multiple imputation with chained equations was used to impute the missing values. Each analysis was repeated in the complete data cohort for comparison.

All statistical tests were two-sided, and *P* values less than 0.05 were considered to be statistically significant.

Results

Among the 1480 patients enrolled in the study, 224 (15.1%) developed new CI-AKI after CAG or PCI. Among the enrolled patients, 6 (0.4%) had hyperthyroidism, 24 (1.6%) had hypothyroidism, 84 (5.7%) had subclinical hyperthyroidism, and 66 (4.5%) had subclinical hypothyroidism. There were no significant difference in the incidence of thyroid and subclinical thyroid diseases between the CI-AKI group and the non-CI-AKI group ($P > 0.05$). Older age and more hypertensive patients were observed in the CI-AKI group than in the non-CI-AKI group ($P < 0.001$) (Table 1). More male patients were found in the non-CI-AKI group than in the CI-AKI group ($P < 0.001$). There were significant differences in heart rate, left ventricular ejection fraction (LVEF), number of smokers, Killip classification, neutrophil percentage, hemoglobin, serum album, and brain natriuretic peptide between the two groups (*P* all < 0.05). The FT3 level in the CI-AKI group was lower than that in the non-CI-AKI group (3.72 ± 0.88 pmol/L vs 4.01 ± 0.80, $P < 0.001$). After matching, variables except for Killip classification and FT3 level presented no significant differences between the two groups.

Table 2 shows medications and procedural characteristics in two groups. In the CI-AKI group, more patients used diuretics ($P < 0.05$) and fewer received PCI therapy ($P = 0.009$). There were significant differences in contrast volume and use of iso-osmolar contrast media (*P* all < 0.05). After matching, no significant differences in medications and procedural characteristics existed between two groups.

Table 3 shows the risk of CI-AKI in patients at different quartiles of FT3 level. Three adjusted logistic models were constructed: model 1 adjusted with age and gender; model 2 adjusted with age, gender, and all variables with $P < 0.1$ before matching in Table 1; model 3 adjusted with all variables in model 2 plus variables with $P < 0.1$ before matching in Table 2. According to FT3 quartiles, patients were divided into four groups. A lower FT3 level was associated with a higher risk of CI-AKI ($OR_{unadjusted} = 0.39$, 95% CI: 0.26–0.59 for quartile 4 vs quartile 1, $P_{trend} < 0.001$; $OR_{model\ 3} = 0.51$, 95% CI: 0.32–0.81 for quartile 4 vs quartile 1, $P_{trend} = 0.005$). To be specific, the risk of CI-AKI decreased

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Table 1 Baseline characteristics before and after matching. Mean ± S.D. or median and 25th and 75th percentiles were used to represent continuous variables. The categorical variable is represented by absolute value (percent).

Characteristics	Before matching			After matching		
	No CI-AKI (*n* = 1256)	CI-AKI (*n* = 224)	*P* value	No CI-AKI (*n* = 672)	CI-AKI (*n* = 224)	*P* value
Age, years	66.09 ± 13.73	69.58 ± 14.15	<0.001	69.07 ± 12.97	69.58 ± 14.15	0.62
Male, *n* (%)	912 (72.61%)	143 (63.84%)	0.008	437 (65%)	143 (63.84%)	0.809
Body mass index, kg/m^2	23.76 ± 3.78	23.32 ± 3.77	0.126	23.46 ± 3.79	23.32 ± 3.77	0.766
Hypertension, *n* (%)	819 (65.21%)	169 (75.45%)	0.003	493 (73.4%)	169 (75.45%)	0.598
Diabetes, *n* (%)	330 (26.27%)	68 (30.36%)	0.204	207 (30.8%)	68 (30.36%)	0.967
Current or former smoker, *n* (%)	642 (51.11%)	94 (41.96%)	0.012	296 44.0%)	94 (41.96%)	0.641
Alcohol consumption, *n* (%)	162 (12.90%)	19 (8.48%)	0.063	54 (8.0%)	19 (8.48%)	0.944
STEMI, *n* (%)	795 (63.30%)	138 (61.61%)	0.629	406 (60.4%)	138 (61.61%)	0.813
NSTEMI, *n* (%)	461 (36.70%)	86 (38.39%)	0.629	266 (39.6%)	86 (38.39%)	0.813
LVEF, %	49.92 ± 8.91	48.31 ± 8.93	0.013	48.45 ± 9.35	48.31 ± 8.93	0.847
Killip class III or IV, n%)	108 (8.60%)	46 (20.54%)	<0.001	97 (14.4%)	46 (20.54%)	0.04
SBP, mmHg	132.59 ± 24.67	131.28 ± 25.41	0.465	132.59 ± 25.06	131.28 ± 25.41	0.5
DBP, mmHg	79.41 ± 16.39	78.03 ± 17.33	0.248	79.36 ± 16.63	78.03 ± 17.33	0.305
Heart rate, bpm	80.14 ± 16.04	84.91 ± 20.43	<0.001	82.94 ± 16.75	84.91 ± 20.43	0.151
WBC, 10^9/L	8.93 (6.95, 11.47)	9.12 (7.22, 11.98)	0.202	9.80 ± 3.95	9.92 ± 3.96	0.708
Neutrophil percentage, %	75.30 ± 10.74	77.83 ± 10.83	0.001	77.44 ± 10.34	77.83 ± 10.83	0.634
Hemoglobin, g/L	134.60 ± 19.46	127.46 ± 22.87	<0.001	129.50 ± 19.80	127.46 ± 22.87	0.199
Serum creatinine, μmol/L	78.90 (65.20, 96.62)	75.60 (61.32, 103.20)	0.44	76.40 (62.45, 95.98)	75.60 (61.18, 103.60)	0.843
Serum albumin, g/L	37.90 ± 4.30	36.31 ± 5.00	<0.001	36.71 ± 4.04	36.31± 5.00	0.231
LogBNP	2.95 ± 0.75	3.14 ± 0.82	0.001	3.08 ± 0.76	3.14 ± 0.82	0.337
TNI, ng/mL	1.85 (0.43, 7.53)	1.50 (0.45, 4.70)	0.321	2.13 (0.50, 8.05)	1.52 (0.45, 5.06)	0.094
FT3, pmol/L	4.01 ± 0.80	3.72 ± 0.88	<0.001	3.87 ± 0.81	3.72 ± 0.88	0.021
FT4, pmol/L	15.43 ± 2.82	15.50 ± 2.85	0.713	15.35 ± 2.95	15.50 ± 2.85	0.5
TSH, μIU/mL	1.03 (0.60, 1.82)	1.13 (0.62, 2.12)	0.219	1.07 (0.60, 1.81)	1.13(0.62, 2,12)	0.313

AKI, acute kidney injury; BNP, brain natriuretic peptide; DBP, diastolic blood pressure; FT3, free triiodothyronine; FT4, free thyroxine; LVEF, left ventricular ejection fraction; NSTEMI, non-ST segment elevation myocardial infarction; SBP, systolic blood pressure; STEMI, ST segment elevation myocardial infarction; TNI, cardiac troponin I; TSH, thyroid-stimulating hormone; WBC, white blood cell.

by 18.9% when FT3 level increased by one S.D. (95%CI: 0.69–0.95, $P = 0.011$) after multivariable adjustment. Restricted cubic splines showed a similar result. The OR declined dramatically as FT3 rose to 4 pmol/L, but then flattened out (Fig. 2).

Finally, 672 patients without CI-AKI were PS-matched to 224 patients with CI-AKI. Between-group balance was checked (Fig. 3). After matching, standardized differences of all variables were less than 20%. Tables 1 and 2 show the data characteristics matched between the CI-AKI group and the non-CI-AKI group. Logistic regression analysis was then performed for paired groups. The risk of CI-AKI increased by 17.7% as FT3 level fell by one unit after PS matching ($OR_{adjusted}$: 0.823, 95% CI: 0.685–0.988, $P = 0.036$). The lower FT3 level was also associated with the higher risk of CI-AKI in the matched cohort ($OR_{adjusted}$= 0.618, 95% CI: 0.398–0.959 for quartile 4 vs quartile 1, $P < 0.05$, $P_{trend} = 0.025$) (Supplementary Table 1, see section on supplementary materials given at the end of this article).

After a median follow-up of 31 days (interquartile range: 30–35 days), 78 (6.93%) patients died, including 72 from cardiogenic and 6 from non-cardiogenic causes. The 78 deaths included 53 in the CI-AKI group and 25 in the non-CI-AKI group ($P < 0.001$). Kaplan–Meier survival analysis showed that patients at the lowest FT3 quartile displayed the worst survival than patients at other quantiles before and after matching (Q1 vs Q2 and Q3 and Q4; both $P < 0.05$; Fig. 4A and C). The prognosis was significantly worse in CI-AKI group than in the non-CI-AKI group (both $P < 0.05$; Fig. 4B and D).

Subgroup analysis was performed according to age, gender, eGFR, LVEF, and hypertension. The results are shown in Fig. 5. A high FT3 was further proved to be associated with a reduced risk of CI-AKI in each subgroup.

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Table 2 Medication and procedural characteristics in relation to CI-AKI before and after matching.

Characteristics	Before matching			After matching		
	No CI-AKI (n = 1256)	CI-AKI (n = 224)	P value	No CI-AKI (n = 672)	CI-AKI (n = 224)	P value
Medication before procedures, n (%)						
Aspirin	1217 (96.9%)	213 (95.1%)	0.168	653 (97.2%)	213 (95.1%)	0.198
Clopidogrel	451 (35.9%)	88 (39.3%)	0.333	235 (35.0%)	88 (39.3%)	0.278
Ticagrelor	805 (64.1%)	136 (60.7%)	0.333	435 (64.7%)	136 (60.7%)	0.345
ACEI/ARB	736 (58.6%)	137 (61.2%)	0.473	402 (59.8%)	137 (61.2%)	0.783
β-blocker	738 (58.8%)	144 (64.3%)	0.12	438 (65.2%)	144 (64.3%)	0.872
Statins	1127 (89.7%)	195 (87.1%)	0.232	599 (89.1%)	195 (87.1%)	0.466
Low molecular heparin	1233 (98.2%)	216 (96.4%)	0.094	653 (97.2%)	216 (96.4%)	0.735
Tirofiban hydrochloride	606 (48.2%)	112 (50.00%)	0.629	331 (49.3%)	112 (50.0%)	0.908
Digoxin	12 (1.0%)	4 (1.79%)	0.285	7 (1.0%)	4 (1.8%)	0.599
Diuretics	230 (18.3%)	55 (24.6%)	0.029	153 (22.8%)	55 (24.6%)	0.648
Procedural characteristics, n (%)						
Contrast volume >100 mL	382 (30.4%)	98 (43.8%)	<0.001	251(37.4%)	98 (43.8%)	0.105
Contrast exposure time >60 min	163 (13.0%)	34 (15.2%)	0.372	100 (14.9%)	34 (15.2%)	1
Use of IOCM	386 (30.7%)	50 (22.3%)	0.011	169(25.1%)	50 (23.3%)	0.445
Hydration therapy	291 (23.2%)	53 (23.7%)	0.872	143 (21.3%)	53 (23.7%)	0.514
PCI	1201 (95.6%)	205 (91.5%)	0.009	629 (93.6%)	205 (91.5%)	0.362
Only CAG	55 (4.4%)	19 (8.5%)	0.009	43 (6.4%)	19 (8.5%)	0.362
Number of stents with each vessel						
Left main coronary artery			0.005			0.093
0	1250 (99.5%)	218 (97.3%)		666 (99.1%)	218 (97.3%)	
≥1	6 (0.5%)	6 (2.7%)		6 (0.9%)	6 (2.7%)	
Left anterior descending artery			0.832			0.878
0	630 (50.2%)	112 (50.0%)		342 (50.9%)	112 (50.0%)	
≥1	626 (49.8%)	112 (50.0%)		330 (49.1%)	112 (50.0%)	
Left circumflex artery			0.338			0.063
0	1076 (85.7%)	185 (82.6%)		590 (87.8%)	185 (82.6%)	
≥1	180 (14.3%)	39 (17.4%)		82 (12.2%)	39 (8.5%)	
Right coronary artery			0.158			0.166
0	866 (69.0%)	165 (73.7%)		460 (68.5%)	165 (73.7%)	
≥1	390 (31.0%)	59 (26.3%)		212 (31.5%)	59 (26.3%)	

ACEI/ARB, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers; CAG, coronary angiography; IOCM, iso-osmolar contrast media; PCI, percutaneous coronary intervention.

Sensitive analysis

As five data sets were generated after multiple imputations, we performed multivariate regression analysis for the other four sets of data sets. In addition, we performed multivariate regression analysis for the entire set ($n = 1480$) without adjusting missing variables. The multivariate regression results were affected due to high values of some missing variables. After removing these variables, we re-analyzed the rest cohort ($n = 1256$), and the results remained consistent (Supplementary Table 2), confirming that a lower FT3 level was associated with a higher risk of CI-AKI.

Discussion

Our study confirmed that the incidence of AKI was 15.1% in AMI patients, higher than most in previous studies (25, 26). Meanwhile, a study has reported an AKI incidence of 26% in AMI patients (25). This inconsistency may arise from (i) differences in enrolled patients because we enrolled patients with ST segment elevation myocardial infarction and non-ST segment elevation myocardial infarction in our study; (ii) differences in baseline age and renal function of enrolled patients; (iii) differences in diagnostic criteria for AKI; (iv) differences in serum creatinine detection methods.

In this study, we found that low FT3 level was associated with elevated AKI risk and mortality after AMI. The association between FT3 level and prognosis of AMI has also been evaluated in several studies (27, 28, 29). Gulzar *et al* reported that FT3 level deceased in patients with AMI, and was related to the duration of illness (30). In a prospective cohort study, Yu *et al* reported that the FT3/FT4 ratio was an independent predictor of 1e year all-cause mortality. The prognostic performance of the FT3/FT4 ratio is similar to that of the GRACE score (31). In another PS-matching study in AMI patients, low FT3 was associated

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Table 3 FT3 level and risk of CI-AKI in entire population. Model 1 adjusted for age and sex; model 2 adjusted for covariates in model 1 plus current or former smoker, alcohol consumption, hypertension, heart rate, neutrophil percentage, hemoglobin, albumin, eGFR, HbA1c, LVEF, LogBNP, and Killip class III or IV; model 3 adjusted for covariates in model 2 plus left main coronary artery, contrast volume >100 mL, use of IOCM, PCI therapy, use of diuretics, and use of low molecular heparin.

	FT3 (range)	**Odds ratio** (95% CI) **and** *P* **value**			
		Unadjusted	Model 1	Model 2	Model 3
FT3 (per 1 S.D.)	(1.1–6.8)	0.704 (0.611–0.813) <0.001	0.754 (0.646–0.881) <0.001	0.807 (0.687–0.947) 0.009	0.811 (0.689–0.954) 0.011
FT3 quartiles					
Q1	≤3.5	1 Reference	1 Reference	1 Reference	1 Reference
Q2	3.5<FT3≤4.0	0.454 (0.308–0.668) <0.001	0.488 (0.329–0.723) <0.001	0.569 (0.377–0.857) 0.007	0.564 (0.371–0.856) 0.007
Q3	4.0<FT3≤4.5	0.484 (0.329–0.713) <0.001	0.550 (0.367–0.826) 0.004	0.651(0.426–0.993) 0.046	0.624 (0.406–0.961) 0.032
Q4	4.5<FT3≤6.8	0.388 (0.257–0.586) <0.001	0.458 (0.295–0.710) <0.001	0.504 (0.319–0.796) 0.003	0.507 (0.319–0.808) 0.004
P for trend		<0.001	<0.001	0.004	0.005

eGFR, estimated glomerular filtration rate.

with severe myocardial injury and high mortality. In addition, a combination of FT3 with TIMI risk score is more accurate to predict the risk of cardiovascular death in AMI backyard (9). Moreover, the lower FT3 level is significantly associated with the worse left ventricular mechanics in AMI patients (32).

In this study, we found that the risk of CI-AKI increased by 19.1% for each unit of FT3 decrease in the matched cohort. Survival analysis also showed that patients at a lower FT3 quartile achieved a worse survival rate than those at other quantiles before and after matching. Several biological mechanisms can explain the association between circulating FT3 and CI-AKI in AMI patients. As an adaptive response to acute diseases, the FT3 level drops to reduce catabolism and energy consumption (33). In a pilot study, supplementation of T3 improved cardiac function in AMI patients with LT3S (34).

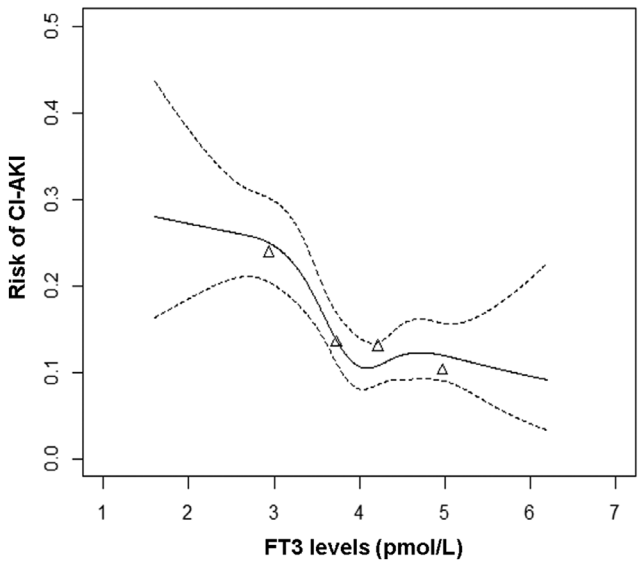

Figure 2
Restricted cubic spines analysis of the association of FT3 levels and risk of CI-AKI. X-axis represents plasma FT3 concentrations. Y-axis represents the probability of CI-AKI. Dashed lines indicate 95% CI. From left to right, the triangles indicate the 20th, 40th, 60th, and 80th percentile.

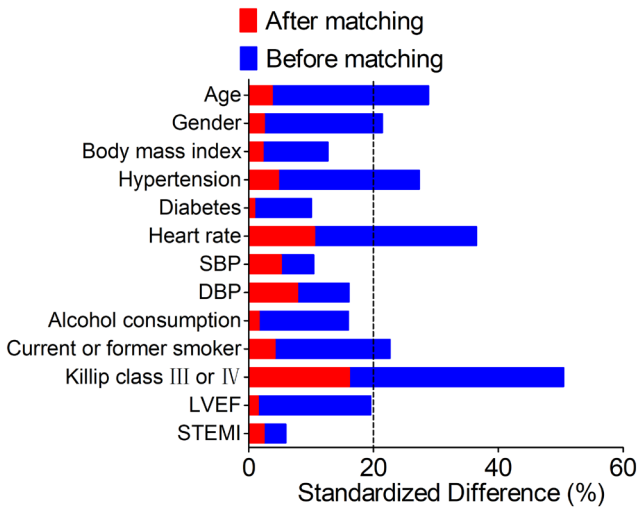

Figure 3
Balance checks of each variable after propensity score matching analysis. Standardized differences of all the variables were illustrated. AKI, acute kidney injury; BNP, brain natriuretic peptide; DBP, diastolic blood pressure; FT3, free triiodothyronine; FT4, free thyroxine; LVEF, left ventricular ejection fraction; NSTEMI, non-ST segment elevation myocardial infarction; SBP, systolic blood pressure; STEMI, ST segment elevation myocardial infarction; TNI, cardiac troponin I; TSH, thyroid-stimulating hormone; WBC, white blood cell.

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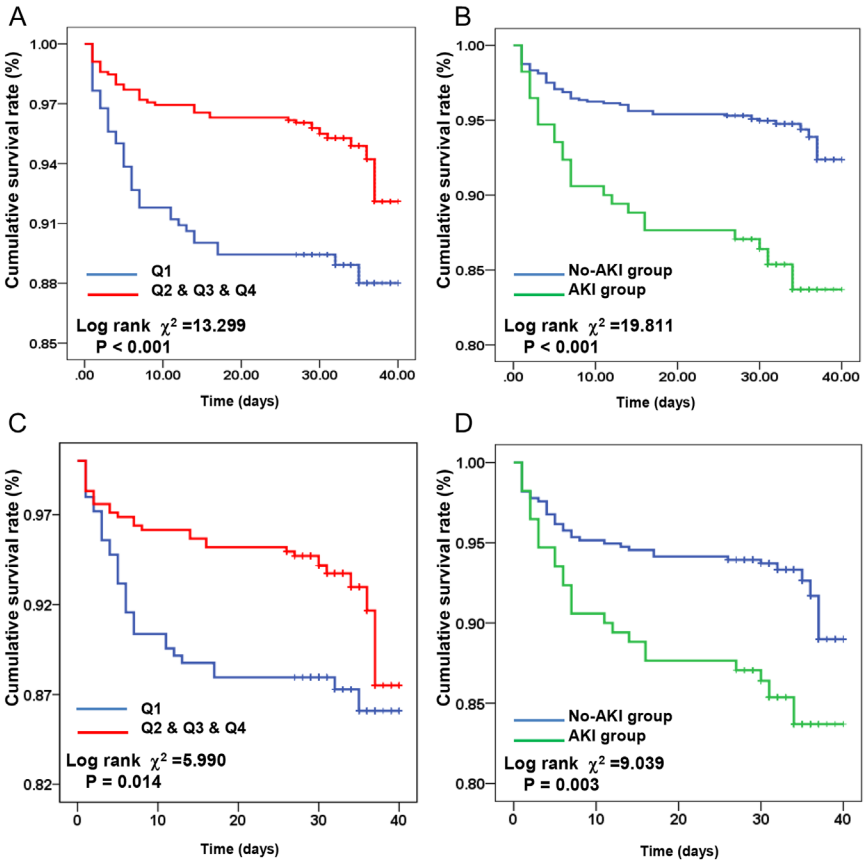

Figure 4
Survival analyses according to FT3 quartiles and the prevalence of CI-AKI before and after matching. (A) Short-term survival rate according to FT3 quartiles before matching (Q1 vs Q2 and Q3 and Q4); (B) Survival rate between CI-AKI group and non-CI-AKI group before matching; (C) Short-term survival rate according to FT3 quartiles after matching (Q1 vs Q2 and Q3 and Q4); (D) Survival rate between the CI-AKI group and the non-CI-AKI group after matching.

T3 and T4 are two main iodinated hormones that can regulate the activities of the cardiovascular system. T3 is transformed from T4 but has a higher affinity to thyroid hormone receptors than T4. In our study, we evaluated the clinical value of FTI in predicting the fate of AMI patients. However, the clinical effect and mechanism of T3-based therapy in patients with AMI need to be further studied.

Limitations

This retrospective study is mainly limited by the bias originating from confounding factors related to CI-AKI occurrence in patients with AMI. So, we tried to collect as many as factors which may influence the risk of CI-AKI and conducted logistic regression analysis. Moreover, we

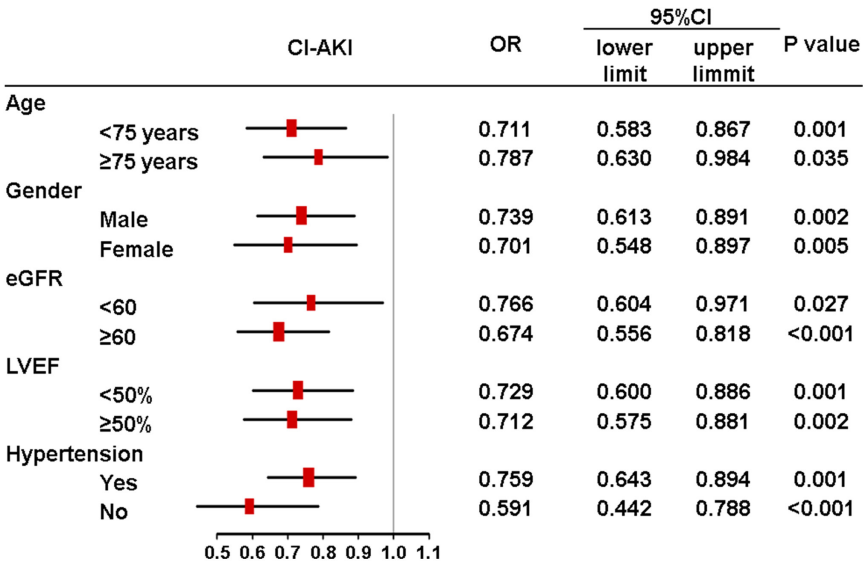

Figure 5
Subgroup analysis of the association between FT3 and CI-AKI. Odds ratios (OR) with 95% CI of CI-AKI per unit increase of FT3 concentration.

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conducted PS matching to balance the variables at baseline. After matching, a highly comparable control group was created. Nonetheless, a prospective, multicenter, larger-sample-size and long-term follow-up study is needed to assess the impact of plasma FT3 on CI-AKI and prognosis.

Conclusions

A low FT3 level was associated with an increased risk of CI-AKI and 1-month all-cause mortality in patients with AMI after coronary intervention.

Supplementary materials

This is linked to the online version of the paper at https://doi.org/10.1530/EC-22-0120.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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