Outcome Predictors in Patients Presenting With Acute Aortic Dissection

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Objective: **To investigate the role of thyroid hormones and other factors in acute aortic dissection and an association with in-hospital adverse events.**

Design: **A retrospective analysis.**

Setting: **A university-affiliated cardiac center.**

Participants: **A total of 151 patients with aortic dissection admitted to the authors' hospital between January 2011 and May 2015.**

Intervention: **None.**

Measurements and Results: **The total in-hospital mortality rate was 12.6%. Triiodothyronine (T3) level was lower in nonsurviving than surviving patients (0.8 ± 0.3 v 1.0 ± 0.4 nmol/L, $p < 0.05$). T3 independently predicted in-hospital mortality (hazard ratio [HR] 0.07, 95% CI 0.01-0.43, $p < 0.01$) and in-hospital acute renal failure (HR 0.22, 0.05-0.89, $p < 0.05$) for all patients. Other independent predictors of in-hospital mortality were pericardial effusion (HR 8.18, 2.11-31.67, $p < 0.01$), conservative treatment (HR 82.12, 12.49-540.09, $p < 0.01$) and Stanford type-A aortic dissection (HR 3.86, 1.06-14.09, $p < 0.05$). Inpatient conservative treatment, T3 (HR 0.01, 0.00-0.18, $p < 0.01$) as well as pericardial effusion (HR 11.80, 2.46-56.59, $p < 0.01$), Stanford type-A dissection (HR 22.35, 3.15-158.40, $p < 0.01$), and in-hospital acute renal failure (HR 16.95, 2.04-140.86, $p < 0.01$) were predictors for in-hospital mortality. In nonconservatively treated patients, T3 (HR 0.02, 0.00-0.88, $p < 0.05$) as well as cardiac care unit stay (HR 0.74, 0.59-0.94, $p < 0.01$) and postoperative acute renal failure (HR 21.32, 3.07-147.88, $p < 0.01$) were predictors for in-hospital mortality.**

Conclusion: **T3 was downregulated in acute aortic dissection. Low T3 level was a risk factor for in-hospital death and acute renal failure in patients with acute aortic dissection.**

KEY WORDS: triiodothyronine, acute renal failure, acute aortic dissection

INTRODUCTION

ACUTE AORTIC DISSECTION is a catastrophic cardiovascular emergency; its in-hospital mortality rate is as high as 52%.[1] However, in "stable" patients, usually those in the chronic phase, 30-day and 5-year mortality could be as low as 5.3% and 18%, respectively.[2,3] Acute renal failure with acute aortic dissection, which is caused by either general circulatory collapse or renal ischemia due to renal artery dissection,[4] significantly increases in-hospital mortality for patients with acute aortic dissection. To distinguish high-risk patients and improve clinical outcomes, numerous risk factors have been identified by previous researchers.[5] Some risk factors are related to patients' general condition, such as age, hypotension, and past medical history; some are related to vital organ malperfusion or systemic circulatory collapse, such as stroke, myocardial infarction, and pericardial tamponade. Some are biomarkers reflecting stress status and inflammatory reaction,[6] including matrix metalloproteinases and soluble elastin fragments; however, these biomarkers are nonspecific and cannot be tested in the clinical laboratory.

Triiodothyronine (T3) is a well-known risk factor in multiple clinical scenarios. It was first reported in critically ill patients admitted to intensive care units (ICUs)[7] and now can be found in various life-threatening conditions, such as respiratory failure, acute myocardial infarction, severe sepsis, trauma, etc.[8-12] Under such critical conditions, thyroid hormone metabolism can be profoundly changed in patients without previously diagnosed intrinsic thyroid disease, including low serum triiodothyronine (T3) level, normal or low thyroxine (T4) level, and normal or low thyroid-stimulating hormone (TSH) level.[13,14] The magnitude of those changes has been associated with disease severity and clinical outcome.[15,16] The pathophysiologic mechanism underlying the association between T3 and critical illness has not been elucidated fully; it generally is considered an adaptive self-protective physiologic response rather than a maladaptive response requiring treatment.[7]

Nevertheless, T3 and acute aortic dissection outcomes have not been reported previously. Gutierrez et al[17] reported that T3 level was not changed in aortic dissection patients, but both acute and chronic aortic dissection patients were enrolled in this small sample size ($n = 28$) research, and the relationship between T3 and clinical outcome was not investigated. Therefore, the authors investigated T3 level in patients with acute aortic dissection and its predictive value for in-hospital outcome as well as other risk factors.

MATERIALS AND METHODS

Subjects and Data Collection

Between January 2011 and May 2015, patients diagnosed with acute aortic dissection at the First Affiliated Hospital of Wenzhou Medical University were enrolled, and their data were analyzed retrospectively. Inclusion criterion was acute aortic dissection confirmed by computed tomography

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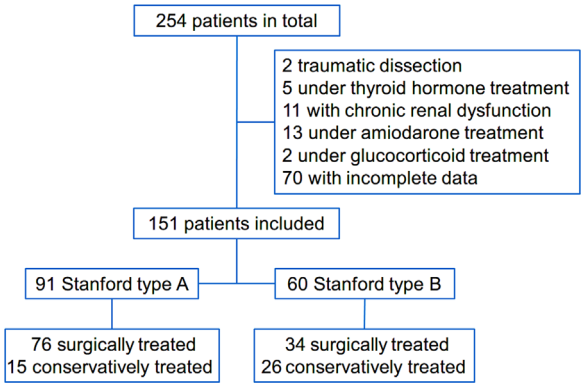

Fig 1. Flowchart of patients enrolled in the study.

angiography (CTA) (Fig 1). "Acute" referred to aortic dissection diagnosed within 2 weeks of symptom onset. The exclusion criteria were history of overt thyroid disease and thyroid hormone-interfering drug therapy, such as amiodarone, thyroid hormone, dopamine, or glucocorticoid. The authors also excluded patients with traumatic dissection and those with a history of severe liver diseases or chronic renal failure.

Serum thyroid hormones, including thyroid stimulating hormone (TSH), triiodothyronine (T3), free triiodothyronine (fT3), thyroxine (T4), and free thyroxine (fT4) in addition to other blood variables (eg, creatinine, serum low-density lipoprotein, D-dimer) were measured on the second morning following patient admission. The reference ranges of thyroid hormones in the authors' laboratory were as follows: TSH, 0.34-5.60 mIU/L; T3, 1.34-2.73 nmol/L; fT3, 3.8-6.0 pmol/L; T4, 78.38-157.40 nmol/L; and T4, 7.86-14.41 pmol/L. Echocardiography was performed within 2 days of admission for

Table 1. Characteristics of Patients With Acute Aortic Dissection

		Stanford Type A (n = 91)		Stanford Type B (n = 60)		
	Total (N = 151)	Surgery (n = 76)	Conservative (n = 15)	Stent-graft (n = 34)	Conservative (n = 26)	p
Age (years)	50.3 ± 13.5	60.9 ± 14.5	52.8 ± 15.1	61.5 ± 12.7	53.8 ± 14.4	>0.05
Male (n, %)	106 (70.20)	47 (61.84)	12 (80.00)	29 (85.29)	18 (69.23)	>0.05
Hypertension (n, %)	99 (65.56)	48 (63.16)	11 (73.33)	21 (61.76)	19 (73.08)	>0.05
Diabetes (n, %)	12 (7.95)	6 (7.89)	1 (6.67)	2 (5.88)	3 (11.54)	>0.05
Current smoker (n, %)	35 (23.18)	17 (22.37)	6 (40.00)	6 (17.65)	6 (23.08)	>0.05
Coronary heart disease (n, %)	3 (1.99)	1 (1.32)	0 (0.00)	0 (0.00)	2 (7.69)	>0.05
History of stroke (n, %)	8 (5.30)	4 (5.26)	4 (26.67)	0 (0.00)	0 (0.00)	<0.05
History of heart failure (n, %)	4 (2.65)	3 (3.95)	1 (6.67)	0 (0.00)	0 (0.00)	>0.05
Marfan's syndrome (n, %)	18 (11.92)	14 (18.42)	1 (6.67)	1 (2.94)	2 (7.69)	>0.05
Thyroid hormones						
T3 (nmol/L)	1.0 ± 0.4	1.0 ± 0.4	1.1 ± 0.3	1.1 ± 0.3	1.0 ± 0.3	>0.05
T4 (nmol/L)	97.9 ± 28.5	98.3 ± 28.5	97.9 ± 32	92.4 ± 25.5	103.9 ± 30.5	>0.05
fT3 (pmol/L)	3.8 (3.3, 4.2)	3.8 (3.2, 4.2)	4.1 (3.8, 4.4)	3.9 (3.5, 4.3)	3.8 (3.3, 3.9)	<0.05
fT4 (pmol/L)	11.3 ± 3	11.9 ± 3.2	10.7 ± 3.1	10.5 ± 3	10.8 ± 2.1	<0.05
TSH (mIU/L)	0.83 (0.49, 1.49)	0.8 (0.5, 1.5)	0.8 (0.4, 2.2)	0.8 (0.5, 1.3)	0.8 (0.5, 1.5)	<0.05
Troponin I (μg/L)	0.02 (0.01, 0.36)	0.15 (0.01, 2.02)	0.03 (0.01, 0.21)	0.01 (0.01, 0.01)	0.02 (0.01, 0.02)	<0.05
LDL (mmol/L)	2.5 ± 0.9	2.2 ± 0.8	2.7 ± 1.0	2.8 ± 0.9	2.7 ± 1.1	<0.05
Creatinine on admission (μmol/L)	75 (60, 102)	75 (57, 108)	70 (61, 114)	69.5 (60.8, 79.3)	84.5 (65, 106)	>0.05
D-Dimer (mg/L)	5.16 (1.96, 13.72)	5.2 (2.8, 17.1)	8.7 (2.2, 15.8)	3.5 (1.9, 7.9)	2.1 (1.1, 9.1)	<0.05
Platelet count ($\times 10^9$/L)	175.8 ± 63.8	172.7 ± 69.5	184.7 ± 53.7	184.6 ± 60.0	168.4 ± 57.6	>0.05
Symptom duration (days)	1 (1, 2)	1 (1, 2)	1 (1, 2)	1 (1, 3)	1 (1, 1.3)	>0.05
Preoperative duration (days)*	2.5 (1, 10)	2.5 (1, 4.8)	N/A	10 (3.8, 12.3)	N/A	<0.05
Artery dissection involved to (n, %)						
Carotid artery	48 (31.79)	39 (51.32)	6 (40.00)	1 (2.94)	2 (7.69)	<0.05
Spinal cord vascular	3 (1.99)	2 (2.63)	0 (0.00)	0 (0.00)	1 (3.85)	>0.05
Coronary artery	13 (8.61)	12 (15.79)	1 (6.67)	0 (0.00)	0 (0.00)	<0.05
Renal artery	37 (24.50)	23 (30.26)	5 (33.33)	4 (11.76)	5 (19.23)	>0.05
Mesenteric artery	19 (12.58)	11 (14.47)	3 (20.00)	4 (11.76)	1 (3.85)	>0.05
Pericardial effusion	53 (35.10)	43 (56.58)	5 (33.33)	2 (5.88)	3 (11.54)	<0.05
Acute kidney injury (n, %)	98 (64.90)	67 (88.16)	7 (46.67)	17 (50.00)	7 (26.92)	<0.01
Stage 1	65 (43.05)	40 (52.63)	7 (46.67)	14 (41.18)	4 (15.38)	<0.01
Stage 2	11 (7.28)	10 (13.16)	0 (0.00)	1 (2.94)	0 (0.00)	
Stage 3	22 (14.57)	17 (22.37)	0 (0.00)	2 (5.88)	3 (11.54)	
Postoperative renal failure needing CRRT (n, %)*	9 (5.96)	8 (10.53)	N/A	1 (2.94)	N/A	<0.05
CCU stay (days)	7 (3, 10)	7 (6, 13)	2 (1, 6)	6 (3.8, 10)	5 (1, 6)	<0.05
In-hospital mortality (n, %)	19 (12.6)	6 (7.9)	8 (53.3)	0 (0.0)	5 (19.2)	<0.05

Abbreviations: CCU, cardiac care unit; CRRT, continuous renal replacement therapy; LDL, low-density lipoprotein; fT3, free triiodothyronine; fT4, free thyroxine; T3, triiodothyronine Cardiac care unit; T4, thyroxine; TSH, thyroid-stimulating hormone.

*For patients who underwent surgical treatment.

preoperative evaluation as well as diagnosis of pericardial effusion or tamponade. All patients received appropriate treatment with statins in addition to individualized maximal doses of β-blockers by oral or intravenous infusion.

The rationale and strategy for treatment were determined by thoracic surgeons according to the 2010 American Association for Thoracic Surgery guidelines for the diagnosis and management of thoracic aortic disease.[18]

This study was approved by the ethical committees of the First Affiliated Hospital of Wenzhou Medical University. Patient information was anonymized and deidentified prior to analysis.

Outcome Measurement

Data on in-hospital adverse events were collected, including death from aortic dissection; rupture of dissecting aneurysm; brain, spinal, coronary, or mesenteric artery dissection indicated by CTA; acute organic dysfunction due to artery dissection such as renal failure, stroke, or myocardial infarction; and pericardial effusion or tamponade found by CT or echocardiography. Acute kidney injury (AKI) and AKI staging were defined as follows: AKI stage 1: increase in serum creatinine $\geq$1.5 times baseline or $\geq$26.5 μmol/L; stage 2: increase in serum creatinine $\geq$2.0 times baseline; stage 3: increase in serum creatinine $\geq$3 times baseline or $\geq$353.6 μmol/L). These definitions followed the Kidney Disease Improving Global Outcomes (KDIGO) clinical practice guideline for acute kidney injury.[19]

Statistical Analysis

Statistical analysis involved use of SPSS v18.0 for Windows (SPSS Inc., Chicago, IL). Continuous variables were expressed as mean ± SD or median (25th-75th percentile); categorical variables were expressed as a number (percentage). Data were compared by Student *t*, ANOVA, rank-sum, or chi-square test, as appropriate. The association of T3 level and in-hospital adverse events was ascertained by binary logistic regression and Cox multivariate survival analysis. Statistical significance was set at $p < 0.05$.

RESULTS

Patient Characteristics

One hundred three patients who met exclusion criteria or with incomplete data were excluded; 151 patients ultimately were included in this investigation. CTA scans were performed in all patients to confirm Stanford type of aortic dissection. Patient enrollment and group are shown in Figure 1. Overall, the mean age was 50.3 (± 13.5) years, and there were significantly more men than women (70.2%, $p < 0.05$). More than half of patients had a history of hypertension (65.56%). Age, sex, symptom duration, and previous medical history were not different among the 4 groups, except for history of stroke. Thirty-eight patients had elevated troponin I, and all of these had Stanford type-A dissections. D-dimer was also higher in patients with Stanford type-A dissections compared with patients who had Stanford type-B dissections.

On the basis of the normal ranges described previously, T3 was lower in 123 (81.5%) patients. T3 level did not differ between Stanford type-A and -B dissections (0.99 ± 0.39 *v* 1.03 ± 0.29 nmol/L, $p > 0.05$), nor did it differ between conservatively and nonconservatively treated patients (1.04 ± 0.28 *v* 0.99 ± 0.38 nmol/L, $p > 0.05$). However, T3 levels were lower in nonsurvivors compared with survivors (0.8 ± 0.3 *v* 1.0 ± 0.4 nmol/L, $p < 0.05$). The baseline characteristics of patients are shown in Table 1.

Table 2. Predictors of In-hospital Mortality by Cox Analysis

	Univariate				Multivariate			
		95% CI				95% CI		
Predictors	HR	Low	Upper	p	HR	Low	Upper	p
T3	0.27	0.07	0.94	0.04	0.07	0.01	0.43	0.00
TSH	1.27	1.12	1.43	0.00	0.99	0.84	1.16	0.86
D-Dimer	1.09	1.02	1.16	0.01	1.05	0.99	1.12	0.12
Pericardial effusion	4.26	1.62	11.22	0.00	8.18	2.11	31.67	0.00
Conservative treatment	7.31	2.76	19.38	0.00	82.12	12.49	540.09	0.00
Stanford type A	1.89	0.68	5.25	0.22	3.86	1.06	14.09	0.04

Abbreviations: CI, confidence interval; HR, hazard ratio; T3, triiodothyronine; TSH, thyroid-stimulating hormone.

Table 3. Predictors of In-hospital Mortality by Cox Analysis for Patients Who Received Conservative Treatment

	Univariate				Multivariate			
		95% CI				95% CI		
Predictors	HR	Low	Upper	p	HR	Low	Upper	p
T3	0.26	0.03	2.00	0.20	0.01	0.00	0.22	0.00
D-Dimer	1.10	1.03	1.18	0.01	1.02	0.95	1.11	0.57
Pericardial effusion	8.80	2.85	27.17	0.00	10.06	2.25	44.87	0.00
Stanford type A	4.11	1.33	12.71	0.01	10.74	2.28	50.70	0.00
Acute kidney injury	1.84	1.15	2.93	0.01	2.29	1.06	4.92	0.03

Abbreviations: CI, confidence interval; HR, hazard ratio; T3, triiodothyronine.

Treatment and In-hospital Outcome

Among patients with Stanford type-A dissections, 76 (50.3%) underwent surgery, and 15 (9.9%) received conservative treatment. Surgical procedures included 43 (28.5%) aortic replacements with or without descending aorta stent graft; 18 (11.9%) aortic valve, root, and ascending aorta replacements with coronary artery re-implant; and 15 (9.9%) aortic valve, root, and ascending aorta replacement with coronary artery bypass. Among patients with Stanford type-B dissections, 34 (22.5%) were treated with stent-graft placement, and 26 (17.2%) received conservative treatment.

The total in-hospital mortality rate was 12.6%. Mortality was higher in conservatively versus nonconservatively treated patients (31.7% *v* 5.5%, $p < 0.01$) but did not differ between Stanford type-A and -B dissections (15.4% *v* 8.3%, $p = 0.20$). Mortality among patients who received conservative treatment for Stanford type-A dissections was significantly higher than that of patients treated surgically for Stanford type-A dissections, as well as for patients treated conservatively and

Table 4. Predictors of In-hospital Mortality by Cox Analysis for Patients Who Received Nonconservative Treatment

	Univariate				Multivariate			
		95% CI				95% CI		
Predictors	HR	Low	Upper	p	HR	Low	Upper	p
T3	0.06	0.01	0.71	0.03	0.02	0.00	0.88	0.04
CCU stay	0.78	0.61	1.00	0.05	0.74	0.59	0.94	0.01
Postoperative renal replacement treatment	18.05	3.00	108.52	0.00	21.32	3.07	147.88	0.00

Abbreviations: CCU, cardiac care unit; CI, confidence interval; HR, hazard ratio; T3, triiodothyronine.

nonconservatively for Stanford type-B dissections (53.3% *v* 7.9%, 0.0%, 19.2%, respectively, $p < 0.05$). No difference in mortality was found among those 3 groups.

Ninety-eight patients (64.90%) experienced AKI during hospitalization, including 22 patients with AKI stage 3 and 9 patients with postoperative acute renal failure and need for continuous renal replacement therapy. The incidences of AKI, AKI stage 3, and postoperative continuous renal replacement therapy were significantly higher in the surgically treated Stanford type-A dissection group than in other groups.

Risk Factors for In-hospital Outcomes

On univariate Cox proportional hazards analysis, in-hospital death was associated with T3 (HR 0.27, 95% CI 0.07-0.94, $p < 0.05$), TSH (HR 1.27, 1.12-1.43, $p < 0.01$), D-dimer (HR 1.09, 1.02-1.16, $p = 0.01$), pericardial effusion (HR 4.26, 1.62-11.22, $p < 0.01$), and conservative treatment (HR 7.31, 2.76-19.38, $p < 0.01$). After adjustment for the above risk factors as well as Stanford type, multivariate Cox analyses confirmed that low T3 independently predicted in-hospital death (HR 0.07, 95% CI 0.01-0.43, $p < 0.01$). Other risk factors were pericardial effusion (HR 8.18, 2.11-31.67, $p < 0.01$), conservative treatment (HR 82.12, 12.49-540.09, $p < 0.01$), and Stanford type-A aortic dissection (HR 3.86, 1.06-14.09, $p < 0.05$) (Table 2).

In patients who received conservative treatment, T3 (HR 0.01, 0.00-0.22, $p < 0.01$) as well as pericardial effusion (HR 10.06, 2.25-44.87, $p < 0.01$), Stanford type A (HR 10.74, 2.28-50.70, $p < 0.01$), and AKI (HR 2.29, 1.06-4.92, $p < 0.05$) were predictors for in-hospital mortality (Table 3). In nonconservatively treated patients, T3 (HR 0.02, 0.00-0.88, $p < 0.05$), cardiac care unit stay (HR 0.74, 0.59-0.94, $p < 0.01$), and postoperative renal replacement treatment (HR 21.32, 3.07-147.88, $p < 0.01$) were predictors for in-hospital mortality (Table 4).

T3 level was lower for patients with AKI stage 3 (0.72 ± 0.38 nmol/L) than stage 1 (1.06 ± 0.34 nmol/L), stage 2 (1.12 ± 0.43 nmol/L) or patients without AKI (0.72 ± 0.38 nmol/L) ($p < 0.01$). T3 level was associated with AKI stage 3 (OR 0.05, 95% CI 0.01-0.24, $p < 0.01$). Other significant risk factors found by univariate logistic regression included D-dimer, pericardial effusion, and low-density lipoprotein. After adjustment for the above risk factors as well as Stanford type and conservative treatment, multivariate logistic regression indicated T3 (OR 0.1, 95% CI 0.02-0.51, $p < 0.01$), and D-dimer (OR 1.09, 95% CI 1.02-1.18, $p < 0.05$) were associated with in-hospital acute renal failure (Table 5).

Table 5. Predictors of AKI Stage 3 by Logistic Regression

	Univariate				Multivariate			
		95% CI				95% CI		
Predictors	OR	Low	Upper	p	OR	Low	Upper	p
T3	0.05	0.01	0.24	0.00	0.1	0.02	0.51	0.01
D-Dimer	1.11	1.04	1.18	0.00	1.09	1.02	1.18	0.02
Pericardial effusion	3.21	1.27	8.13	0.01	1.47	0.45	4.86	0.53
LDL	0.47	0.26	0.85	0.01	0.77	0.4	1.47	0.42
Conservative treatment	0.38	0.11	1.35	0.13	0.55	0.13	2.41	0.43
Stanford type	2.53	0.88	7.27	0.09	0.92	0.22	3.89	0.91

Abbreviations: CI, confidence interval; LDL, low-density lipoprotein; OR, odds ratio; T3, triiodothyronine.

DISCUSSION

The major finding and innovation of the present study were that the authors found that T3 was lower in nonsurvivors versus survivors of acute aortic dissection. T3 independently predicted in-hospital mortality and acute renal failure for patients with acute aortic dissection.

In the present study, in-hospital mortality due to acute aortic dissection varied from 0% to 53.3%, mainly depending on whether surgical or medical treatment was performed. Risk factors for in-hospital mortality differed between surgically and medically treated patients. Nevertheless, in both medically and surgically treated patients, univariate and multivariate analyses demonstrated that T3 was an independent risk factor for in-hospital mortality. To the best of the authors' knowledge, no study has investigated thyroid hormones in acute aortic dissection outcome. This research provided a new biomarker for risk classification and an outcome predictor.

Previous studies have proven that surgical management could greatly decrease mortality compared with medical treatment.[20,21] Thus, it generally was advocated that acute aortic dissection should be managed surgically to improve outcomes. In the current study, mortality of surgically treated Stanford type-A dissection was similar to that of Stanford type-B dissection, and Stanford type-A dissection was not a significant risk factor for surgically treated patients, which also agreed with previous studies. In addition, T3 independently predicted in-hospital mortality despite treatment strategy.

The pathophysiologic mechanism underlying the association between lowered T3 and critical illness has not been fully elucidated, but several theories have been proposed. Some researchers suggested that T3 was lowered by thyroid hormone-interfering drugs such as glucocorticoids, dopamine,

and amiodarone or propranolol,[22-25] which were not found in patients in the current study. Some researchers suggested that in the setting of infectious or inflammatory diseases, T3 synthesis was inhibited by free fatty acids or cytokines,[26,27] which needs further investigation in the setting of aortic dissection.

The authors also found low T3 associated with AKI and AKI staging. AKI could be caused by general circulatory collapse or renal malperfusion due to renal artery dissection. In the present study, renal artery dissection was found in only about one fifth of all included patients and nearly two thirds of all included patients experienced AKI. It was conceivable that quite a few instances of AKI were caused by hypotension, systemic malperfusion, and shock. Previous studies associated low T3 with vulnerable renal function and worsened renal disease outcome; however, these studies all focused on chronic subclinical or clinical hypothyroidism in chronic renal failure or kidney transplantation.[28,29] More investigations are needed to reveal the underlying association between acute low T3 and acute renal injury.

Some limitations of the current study need to be mentioned. First, T3 could be significantly lower in patients with pre-existing subclinical hypothyroidism, which the authors were not able to exclude. Given that the prevalence of subclinical hypothyroidism in the healthy population ranges from 2.8% to 7.5%,[30,31] the incidence of patients with both acute aortic dissection and subclinical hypothyroidism should be quite small, and the authors assumed the effect was negligible. Second, because all patients underwent CTA, the authors could not exclude AKI caused by contrast-induced nephropathy (CIN), although the incidence of CIN was quite low in patients without chronic renal disease. Last, this was a single-center retrospective study with a relatively small study population. Therefore, a prospective large-scale multicenter study is required to confirm the authors' results as well as to predict the role of T3 in long-term survival.

CONCLUSIONS

Serum T3 provides an additional risk factor to predict acute renal failure and in-hospital mortality in patients with aortic dissection.

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