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The Preoperative Uric Acid-to-Albumin Ratio as a New Indicator to Predict Long-Term Prognosis After Surgery for Patients with Acute Type A Aortic Dissection

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ABSTRACT

Background: The long-term prognosis of patients with acute type A aortic dissection (AAD) is poor, despite emergency surgical treatment. Therefore, it is imperative to evaluate patient risk factors to improve the prognosis. The aim of this study was to analyze the ability of the uric acid-to-albumin ratio (UAR) to predict the long-term mortality of patients with type A AAD after surgery.

Methods and results: A total of 289 patients with type A AAD who had received surgical treatment was enrolled in this study. Peripheral blood samples were collected before anesthesia induction. All patients were divided into the UAR < 9.875 group and the UAR ≥ 9.875 group, and mortality significantly differed between the two groups. The patients were further divided into survival and non-survival groups, according to whether death occurred after the procedure based on a one-year follow up. Factors, including age, hypertension, albumin, UAR, and D-dimer, differed significantly between the survival and non-survival groups. The independent risk factors for long-term death in patients with type A AAD were analyzed by univariable and multivariable COX regression analyses, and the predictive value of these indices for postoperative mortality was assessed based on the receiver operating characteristic (ROC) curves. Preoperative UAR (HR 1.904, 95% CI, 1.097 to 3.305; $P < 0.05$), D-dimer (HR, 1.991,95% CI, 1.116 to 3.554; $P < 0.05$), and age (HR 2.216, 95% CI, 1.287 to 3.815; $P < 0.05$) were identified as independent risk factors for one-year mortality in patients with Type A AAD. The area under the ROC curve (AUC) of UAR was 0.618 [95% (0.544, 0.693)], and the sensitivity and specificity were 69.6% and 51.8%, respectively ($P = 0.003$). The AUC for albumin was 0.349 [95% (0.274, 0.425)], and the sensitivity and specificity were 26.1% and 51.8%, respectively ($P = 0.000$), The AUC for uric acid was 0.544 [95% (0.470, 0.619)], and the sensitivity and specificity were 78.3% and 34.5%, respectively ($P = 0.265$). The AUC for UAR + age + D-dimer was 0.751 [95% (0.681, 0.821)], and the sensitivity and specificity were 76.8% and 68.2%, respectively.

Conclusions: UAR in patients with type A AAD may be used as a new independent risk factor for long-term mortality. Its predictive value is superior to that of albumin or uric acid alone. The combination of UAR, age, and D-dimer provide good prognostic value.

INTRODUCTION

Acute type A aortic dissection (AAD) is the most common and catastrophic presentation of acute aortic syndrome. Despite aggressive surgical treatment, the prognosis of some patients remains poor, and further exploration of the factors associated with the prognosis of aortic dissection is needed [Pape 2007; Trimarchi 2005; Tolis 2016]. Low serum albumin (SA) level can increase blood viscosity, damage endothelial cells, and aggravate primary cardiovascular disease [Zhang 2002; Ronit 2020; Arques 2018; Liao 2020; Nagai 2018]. Elevated serum uric acid level can cause a cascade reaction of inflammation and oxidative stress, damaging vascular endothelial cells [Maruhashi 2018; Papezikova 2009]. Uric acid is considered to be an independent factor in the occurrence and development of cardiovascular events [Cai 2019; Virdis 2020; Kleber 2015]. Çakmak et al. reported that the uric acid-to-albumin ratio (UAR), a novel inflammatory marker, might be used reliably to predict the extent of coronary artery disease (CAD) in non-ST elevation myocardial infarction (NSTEMI) patients [Çakmak 2021]. However, no studies have evaluated the ability of UAR to predict the prognosis of patients with type A AAD. In this study, we investigated the predictive value of preoperative UAR for the prognosis of patients with type A AAD to provide clinical basis for advanced clinical intervention.

PATIENTS AND METHODS

All patients were from the Cardiovascular Surgery Department of the First Affiliated Hospital of Xi 'an Jiaotong University from January 2019 to September 2020. These patients were diagnosed with type A AAD by computed tomography. Dissection was classified, according to the Stanford criteria. The primary inclusion criteria were patients with Stanford Type A AAD within two weeks after symptom onset and

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aged 18 to 75 years. A total of 289 patients with type A AAD were admitted to our study. The follow-up period of a year started on the day after surgery, including the patients who died in the hospital. The exclusion criteria were as follows: (1) a history of cardiogenic shock or pericardial tamponade; (2) iatrogenic aortic dissection; (3) traumatic aortic dissection; (4) severe valve disease; (5) congenital heart disease; (6) severe organ dysfunction, such as liver and kidney failure; (7) metabolic diseases; (8) malignant tumors; (9) gout or thyroid disease; (10) severe gastrointestinal diseases; and (11) recent use of drugs affecting uric acid and albumin. Patients were followed up between September and October 2021. The primary end-point event was death. Clinical data of the enrolled patients were obtained by consulting their medical records. Demographic information, medical history, vital signs, laboratory test were recorded. Complications and outcomes were recorded during the one-year follow-up period. All data were analyzed in a blinded manner. All study protocols were followed in the guidelines of the Research Committee of Human Investigation of Xi'an Jiaotong University Health Science Center. All selected candidates signed informed consent, and this study was approved by the Ethics Committee (Approval No.: XJTU1AF2019LSK-110).

Surgical methods: All patients with type A AAD were treated by surgery within one day after admission. A stentgraf (MicroPort Medical Company Limited, Shanghai, China) and 4-branched prosthetic graf (Vascutek Limited 4 Branch Graf, Newmains Avenue, Inchinnan, Renfrewshire, Terumo) were used in total arch replacement combining with stented elephant trunk implantation. Patients underwent a median sternotomy and total cardiopulmonary bypass.

Methods for detecting clinical indicators: Platelets, white blood cells, red blood cells, and hemoglobin were detected using an automatic blood cell analyzer (Mindray, model BC6800Plus, Shenzhen Mindray Bio-Medical Electronics Co., LTD., China). C-reaction protein (CRP) was detected using another automatic blood cell analyzer (Mindray, model CRP-M100, Shenzhen Mindray Bio-Medical Electronics Co., LTD., China). Total bilirubin, albumin, globulin, total cholesterol, urea, creatinine, uric acid, and CK-MB were detected using an automatic biochemical analyzer (HITACHI, Model: 008AS, Japan). D-dimer and fibrinogen degradation product (FDP) were detected using a hemagglutination analyzer (SYSMEX, model Cs-5100, Japan).

Statistical analysis: Summary statistics were presented as frequencies and percent ages or as means ± SD. Differences between two groups were compared using unpaired Student's t tests or Mann-Whitney U test for continuous variables and χ^2 test for categorical variables. The Cox proportional hazards model was used to identify predominant predictors for aortic events throughout the entire follow-up period with the use of univariate and stepwise multivariate analyses. The prediction ability of the combined index was obtained by Logistic regression analysis, and then the combining indexes were included in the ROC analysis for statistics. Survival rate, dissection-related death-free rate, and aortic event-free rate were computed, according to the Kaplan-Meier technique, and event-free curves were compared with the use of the log-rank test.

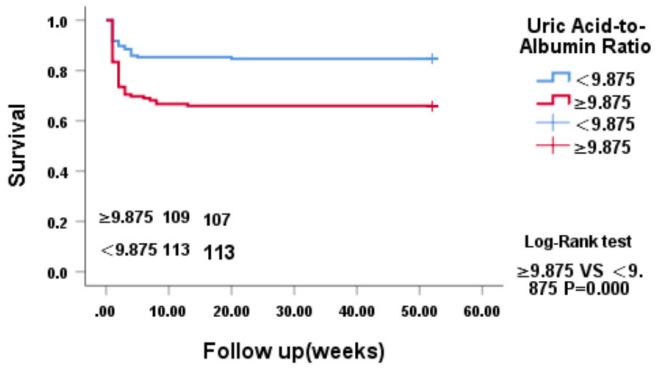

Figure 1. Cumulative survival in each group

For laboratory results, we also assessed whether measurements were outside the normal range. All statistical analyses were performed using IBM Statistics 26. A two-tailed value of $P < 0.05$ was considered statistically significant.

Consent for publication: Our study does not contain any individual person's data in any form. All authors signed a consent form for publication in case of acceptance.

RESULTS

Basic and clinical characteristics: A total of 289 patients (213 men and 76 women) were included in this study. Each patient received a surgical operation between January 2019 and September 2020. The average age was 52 ± 11 years old. All patients were divided into the UAR < 9.875 group (135 cases) and the UAR ≥ 9.875 group (154 cases), according to the optimal critical value of UAR in ROC analysis. Mortality was significantly different between the two groups. The indicators assessed included age, sex, BMI, globulin level, total bilirubin level, blood urea nitrogen (BUN) level, creatinine (Cr level), creatinine level, clearance, hemoglobin (HGB) level, white blood cells (WBC) level, red blood cell (RBC) level, total cholesterol level, creatine kinase-MB (CK-MB) level, hypersensitive troponin T level, ventilator-assisted time, and length of stay in the ICU. The patients also were divided into the survival and non-survival groups, according to whether death occurred after the procedure during follow up. Age, hypertension, albumin, UAR, Cr, creatinine, clearance, RBC, platelets (PLT), systolic pressure (SBP), CK-MB, D-dimer, and FDP were significantly different between the two groups. As shown in Figure 1, in type A AAD, Kaplan–Meier survival analysis showed that the mortality was higher in patients with high UAR (≥ 9.875 mg/L) compared with those with low UAR (< 9.875mg/L; log-rank X2 =20.68; $P < 0.001$). (Figure 1)

Predictive factors of death: Cox regression multivariate analysis after univariate analysis of all-cause mortality showed that UAR (hazard ratio [HR], 1.904; 95% confidence interval [CI], 1.097 to 3.305; $P < 0.05$), was independent risk factors for mortality. (Table 3) Patients in the UAR ≥ 9.875 group had significantly higher death rates than those in the UAR < 9.875 group ($P < 0.001$; Figure 1).

Table 1. Baseline and clinical characteristics between the UAR ≥ 9.875 group and the UAR < 9.875 group

Variable	All (*N* = 289)	UAR<9.875 group (*N* = 135)	UAR≥9.875 group (*N* = 154)	*P*-value
Age (years)	52 (45-59)	54 (47-61)	51 (42-57)	0.012
Men, n (%)	213 (74)	81 (60)	132 (86)	0.000
BMI (kg/m^2)	25.83 (22.86-28.15)	24.80 (22.22-26.24)	26.24 (24.08-29.33)	0.002
Smoking index (per year)	0 (0-400)	0 (0-200)	80 (0-400)	0.004
History of CHD (%)	11 (4)	4 (3)	7 (5)	0.483
Hypertension, n (%)	170 (59)	81 (60)	89 (58)	0.704
Diabetes, n (%)	5 (2)	2 (1)	3 (2)	0.762
EuroSCORE II (%)	4.65 (3.77-5.64)	4.65 (3.77-5.62)	4.38 (3.77-6.16)	0.668
Globulin (g/L)	20.5 (17.2-24.1)	19.1 (16.6-21.7)	22.2 (17.9-25.3)	0.000
Total bilirubin (μmol/L)	30.0 (18.1-47.4)	38.5 (25.9-36.1)	21.1 (15.3-35.2)	0.000
BUN (mmol/L)	7.13 (5.64-9.04)	6.61 (5.11-8.44)	7.61 (5.93-9.99)	0.000
Cr (μmol/L)	78 (58-105)	60 (44-82)	95 (71-125)	0.000
Creatinine clearance (ml/min)	99.40 (69.85-134.25)	111.50 (79.70-151.70)	86.10 (57.90-114.70)	0.000
HGB (g/L)	128±22	123 (112-139)	133 (119-146)	0.002
WBC (×10^9/L)	10.76 (8.32+14.43)	9.73 (7.56-12.72)	12.18 (9.48-15.28)	0.000
RBC (×10^{12}/L)	4.17±0.63	4.07±0.52	4.26±0.90	0.003
PLT (×10^9/L)	142 (1017-185)	147 (110-197)	140 (105-180)	0.293
CRP (mg/L)	16.6 (10.0-50.4)	13.5 (10.0-42.2)	22.8 (10.0-56.33)	0.086
Total cholesterol (mmol/L)	2.76 (2.10-3.49)	2.27 (1.89-2.77)	3.26 (2.49-3.96)	0.000
SBP (mmHg)	135 (118-155)	136 (120-155)	135 (114-179)	0.522
DBP (mmHg)	78 (65-89)	80 (66-89)	76 (62-88)	0.108
HR (times/min)	80±17	78 (66-89)	76 (62-88)	0.278
CK-MB (U/L)	12.0 (7.7-23.0)	10.7 (6.4-14.0)	16.8 (9.0-30.3)	0.000
Hypersensitive troponin T (ng/ml)	0.015 (0.010-0.042)	0.012 (0.010-0.021)	0.018 (0.011-0.102)	0.003
Ventilator-assisted time (d)	2 (1-6)	2 (1-4)	3 (1-8)	0.014
Length of stay in ICU (d)	6 (4-11)	6 (4-9)	7 (4-12)	0.113
Length of time (d)	18 (14-22)	18 (14-21)	18 (13-22)	0.530
Infection, n (%)	84 (29)	29 (21)	55 (36)	0.008
Ventricular arrhythmias, n (%)	15 (5)	4 (3)	11 (7)	0.110
Atrial arrhythmia, n (%)	10 (3)	4 (3)	6 (4)	0.665
Secondary tracheal intubation, n (%)	47 (16)	17 (13)	30 (19)	0.113
Secondary surgical hemostasis, n (%)	11 (4)	3 (2)	8 (5)	0.188
Cerebral infarction, n (%)	18 (10)	5 (4)	13 (8)	0.096
Cerebral hemorrhage, n (%)	10 (3)	2 (1)	8 (5)	0.085
Gastrointestinal dysfunction, n (%)	53 (18)	14 (10)	39 (25)	0.001
Death, n (%)	69 (24)	21 (16)	48 (31)	0.002

Data are mean ± SD or median (interquartile range), n (%). BMI, body mass index; CHD, coronary heart disease; BUN, urea nitrogen; Cr, creatinine; HGB, hemoglobin; WBC, white blood cells; RBC, red blood cells; PLT: platelet; CRP, C-reactive protein; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; CK-MB, creatine kinade MB; ICU, intensive care unit

The predictive value UAR for death was evaluated based on the receiver operating characteristic (ROC) curves, as shown in Table 4. (Table 4) The area under the ROC curve (AUC) of UAR was 0.618 [95% (0.544, 0.693)], and the sensitivity and specificity were 69.6% and 51.8%, respectively (P = 0.003). As shown in Figure 2, of the 135 patients in the low UAR group, 21 (13.6%) died during follow up. (Figure 2) Of the 154 patients in the high UAR group, 48 (31.2%) died during follow up. The remaining indicators are detailed in Figure 2.

The combined predictive value of UAR, age, and D-dimer for deaths in patients with type A AAD was evaluated based on the ROC curves. (Table 5) The results show that UAR + D-dimer is more valuable in predicting deaths in comparison to UAR + D-dimer and UAR + age.

DISCUSSION

Type A AAD is one of the most common and critical diseases in cardiovascular surgery. Early identification of risk factors in patients with Type A AAD may help reduce the risk of death in these patients. At present, older age, history of hypertension, cardiac tamponade, shock, coronary tear, acute renal failure, acute liver failure, stroke, mesenteric ischemia, and other risk factors have been considered independent predictors of type A AAD death [Evangelista 2018], but these risk factors still cannot meet the needs of clinical practice. The mortality of patients remains high after active corrective and preventive measures, and postoperative complications are common. The early identification of risk factors in type A AAD patients may help reduce their risk of death.

Many studies have shown that SA is likely protection factors to be the type A AAD, type A AAD occurs, a large number of inflammatory cytokines release, increase SA decomposition, led to lower SA, blood vessels in the colloid osmotic pressure drop, cause oedema after large amounts of fluid leakage, causing the blood volume is reduced, viscera perfusion decreased, AKI, pulmonary edema, cerebral edema and other serious complications can further affect the postoperative outcome of patients with Type A AAD. Low SA reduces the ability of scavenging oxygen-free radicals and inhibiting inflammatory response, and endothelial cells are damaged, and the elasticity of aortic vascular wall decreases, ultimately affecting the outcome of Type A AAD [Keskin 2021; Gao 2019; Zhao 2020; Weiner 2011].

Uric acid is the final product of purine metabolism formed via a xanthine oxidoreductase-catalyzed oxidation reaction. The serum uric acid level is related to the total antioxidant capacity and aortic dilation [Esen 2011]. The serum uric acid level also is related to pathological processes, such as endothelial dysfunction, oxidative stress, systemic inflammation, and renin-angiotensin system activation [Mercuro 2004; Corry 2008; Ruggiero 2006; Mehta 2015]. These pathologic processes further lead to endothelial dysfunction, vascular smooth muscle cell proliferation, and increased arterial stiffness. Uric acid can increase the serum levels of IL-1β and nucleotide binding oligomerization domain like receptor 3 and increase blood pressure variability [Balakumar 2008;

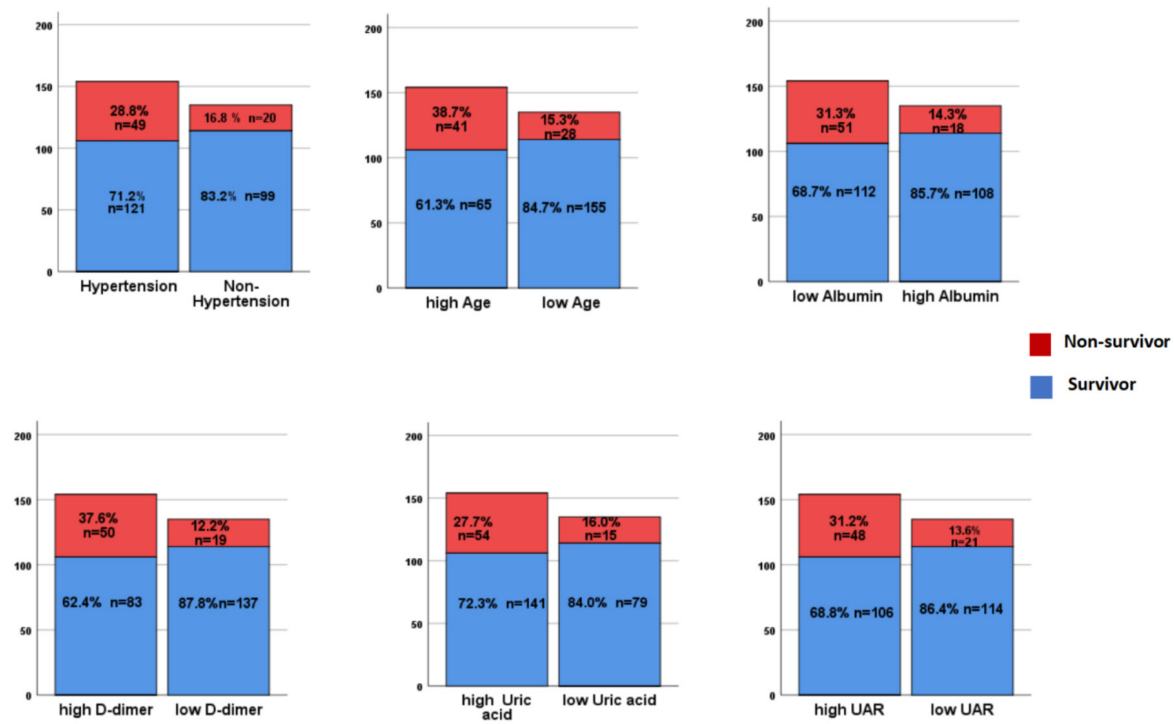

Figure 2. Distribution of mortality rate in patients with type A AAD, according to categories based on UAR, D-dimer, age, hypertension, albumin, and uric acid.

Hong 2012]. Hyperuricemia can participate in aortic vascular endothelial injury through oxidative stress pathway, resulting in increased serum ET-1 and ICAM and decreased carbon monoxide NO. Increasing the uric acid concentration was found to decrease the expression of ENOS, increase the expressions of ET-1 and ICAM, and significantly increase the apoptosis rate of aortic cells [Kang 2005]. Zhang et al. [Zhang 2020] found that preoperative uric acid is an independent risk factor for postoperative in-hospital death of Type A AAD. However, that study only included a few high-risk factors for type A AAD and did not exclude confounding factors. Moreover, the number of patients was small, and only in-hospital death was considered without long-term follow up. In clinical practice, many factors affect the production and excretion of SA and UA [So 2010; Arques 2018], making it difficult to use SA and UA parameters as markers for CAD.

Based on a one-year follow up, age, preoperative UAR, SA, and D-dimer were closely related to one-year mortality. Moreover, UAR can better predict the one-year mortality than SA or uric acid alone, and the combination of UAR, age, and D-dimer provides good prognostic value.

Albumin can be used to evaluate the nutritional status and severity of inflammation in patients with type A AAD. Due to endovascular tears, inflammatory cascade, systemic arteriolar spasms, and even liver and kidney involvement, the liver and kidney function are impaired in type A AAD,

Table 2. Comparison of the baseline and clinical characteristics between the survival and non-survival groups. Data are mean ± SD or median (interquartile range), n (%). FDP, fibrinogen degradation product.

Variable	All (*N* = 289)	Survival (*N* = 220)	Non-survival (*N* = 69)	*P*-value
Age (years)	52 (44-59)	51 (43-57)	58 (50-67)	0.000
Men, n (%)	213 (74)	168 (76)	45 (65)	0.067
BMI (kg/m^2)	25.83 (22.86-28.15)	25.51 (22.86-28.07)	26.67 (22.79-28.44)	0.234
Smoking index (per year)	0 (0-400)	0 (0-400)	0 (0-400)	0.727
Body mass index (kg/m2)	22 (8)	18 (8)	4 (6)	0.515
History of CHD (%)	11 (4)	7 (3)	4 (6)	0.322
Hypertension, n (%)	170 (59)	121 (60)	49 (71)	0.018
Diabetes, n (%)	5 (2)	4 (2)	1 (1)	0.838
EuroSCORE II (%)	4.65 (3.77-5.64)	3.88 (3.77-5.04)	5.57 (3.77-7.87)	0.000
Uric acid (μmol/L)	421.29±88.02	365.68±138.64	382.62±119.83	0.362
Albumin (g/L)	35.7 (33.0-38.6)	36.2 (33.6-38.9)	34.2 (31.5-36.8)	0.000
Globulin (g/L)	23.6 (20.7-26.5)	23.6 (20.9-26.6)	23.6 (18.5-26.4)	0.222
Total bilirubin (μmol/L)	18.0 (14.4-26.9)	18.00 (14.7-27.2)	17.40 (13.8-24.4)	0.290
UAR (μmol/g)	10.24 (7.96-12.54)	9.65 (7.60-11.96)	11.03 (8.98-13.08)	0.003
BUN (mmol/L)	7.13 (5.64-9.04)	7.06 (5.32-8.74)	7.47 (6.20-9.84)	0.011
Cr (μmol/L)	78 (58-105)	73 (54-99)	91 (68-122)	0.000
Creatinine clearance (ml/min)	99.4 (69.9-134.3)	107.3 (79.0-147.2)	71.8 (55.5-103.6)	0.000
HGB (g/L)	129 (116-142)	131 (118-142)	123 (109-141)	0.083
WBC (×10^9/L)	10.76 (8.32-14.43)	10.65 (8.28-14.34)	11.73 (9.13-14.95)	0.131
RBC (×10^{12}/L)	4.52±0.86	4.21±0.63	4.06±0.63	0.039
PLT (×10^9/L)	142 (107-185)	154 (113-190)	118 (86-155)	0.000
CRP (mg/L)	10.0 (8.0-12.0)	14.9 (10.0-48.10)	23.7 (10.0-73.0)	0.091
Total cholesterol (mmol/L)	3.44 (2.89-4.05)	3.50 (2.96-4.06)	3.23 (2.53-3.95)	0.065
SBP (mmHg)	135 (118-155)	138 (120-158)	124 (110-148)	0.010
DBP (mmHg)	78 (65-89)	79 (68-89)	75 (62-85)	0.051
HR (times/min)	80 (68-90)	80 (68-90)	80 (67-70)	0.634
CK-MB (U/L)	12.0 (7.7-23.0)	12.0 (7.0-20.0)	18.0 (9.2-32.5)	0.002
Hypersensitive troponin T (ng/ml)	0.015 (0.010-0.042)	0.013 (0.010-0.410)	0.018 (0.012-0.044)	0.256
D-Dimer (mg/L)	8.86 (3.14-20.33)	7.20 (2.67-17.08)	18.50 (8.12-30.93)	0.000
FDP	26.43 (9.63-61.78)	22.70 (7.52-47.49)	57.98 (27.43-107.47)	0.000

Table 3. Univariate and multivariate predictor analyses of all-cause death

Variables	Univariate analysis			Multivariate analysis		
	HR	95% CI	*P*-value	HR	95% CI	*P*-value
UAR	2.4000	1.462-3.941	0.001	1.904	1.097-3.305	0.022
Age (years)	2.870	1.784-4.618	0.000	2.216	1.287-3.815	0.004
Hypertension (%)	1.707	1.022-2.851	0.041	1.825	1.077-3.094	0.025
Albumin (g/L)	2.192	1.003-4.789	0.049	1.660	0.734-3.754	0.224
CRP (mg/L)	1.723	1.059-2.805	0.028	1.663	0.998-2.773	0.051
Creatinine (μmol/L)	2.198	1.316-3.672	0.003	1.602	0.905-2.838	0.106
RBC ($\times 10^{12}$/L)	1.664	1.022-2.708	0.041	1.364	0.772-2.409	0.285
Platelets($\times 10^{9}$/L)	1.824	1.137-2.925	0.013	1.096	0.633-1.898	0.744
CK-MB (U/L)	1.717	1.028-2.868	0.039	1.035	0.587-1.824	0.906
D-dimer (mg/L)	2.780	1.684-4.590	0.000	1.991	1.116-3.554	0.020
FDP (mg/L)	3.543	2.083-6.028	0.000	1.811	0.934-3.512	0.079

Table 4. Diagnostic value of UAR, D-dimer, age, hypertension, albumin, and uric acid for mortality

Variables	AUC	Cut-off value	SE	95% CI	Sensitivity	Specificity	*P*-value
UAR	0.618	>9.875	0.038	0.544-0.693	0.696	0.518	0.003
Age (years)	0.685	>56	0.039	0.609-0.761	0.565	0.741	0.000
Hypertension (%)	0.573	>0.5	0.039	0.497-0.649	0.696	0.450	0.068
Albumin (g/L)	0.349	<36.45	0.039	0.274-0.425	0.261	0.518	0.000
D-dimer (mg/L)	0.701	>9.19	0.035	0.632-0.770	0.725	0.600	0.000
Uric acid (μmol/L)	0.544	>305	0.038	0.470-0.619	0.783	0.345	0.265

Table 5. Diagnostic value of D-dimer + age, UAR + age + D-dimer, UAR + D-dimer, and UAR + age for mortality

Variables	AUC	SE	95% CI	Sensitivity	Specificity	*P*-value
UAR+age	0.714	0.038	0.640-0.787	0.594	0.759	0.000
UAR+D-dimer	0.727	0.034	0.660-0.794	0.855	0.518	0.000
UAR+age+D-dimer	0.751	0.036	0.681-0.821	0.768	0.682	0.000
D-dimer+age	0.731	0.037	0.659-0.803	0.708	0.655	0.000

leading to impaired albumin synthesis, secretion, and absorption. Some type A AAD patients experience severe hypoalbuminemia earlier than inflammatory biomarkers and visceral injury can indicate. Thus, significantly lowered SA may be a prodrome of type A AAD. The uric acid level reflects the state of oxidative stress in the body to a certain extent. As the degree of illness worsens, oxidative damage increases, which affects uric acid synthesis. Moreover, disease progression affects the kidneys, resulting in disordered uric acid excretion, further increasing the level of uric acid in the blood [Zeng 2016; Artigas 2016; Li 2016; Abe 2015]. Therefore, the blood level of uric acid is likely related to disease severity.

In this study, the SA level in type A AAD patients was significantly lower in the non-survival group than in the survival group, while the serum uric acid level was higher in the non-survival group. The correlation between prognosis and the serum SA level or the serum uric acid level alone was not strong. Therefore, UAR was evaluated as a prognostic factor for type A AAD patients. We found a statistically significant difference in UAR between the non-survival and survival groups. Moreover, UAR increased with increasing disease severity. UAR can reflect the severity of AAD and the prognosis of type A AAD patients with good sensitivity. UAR values exceeding 9.875 indicate the likelihood of poor prognosis in type A AAD patients.

CONCLUSIONS

UAR in patients with type A AAD may be used as a new independent risk factor for long-term mortality. The predictive value of UAR is superior to those of albumin and uric acid. Thus, the use of UAR may improve the accuracy of clinical prognostic assessment. UAR shows great potential in clinical application because it can be determined early, simply, and quickly with low cost. Therefore, UAR is valuable for prognostic evaluation in type A AAD patients. Further, the combination of UAR, age, and D-dimer has good prognostic value and can be used to guide clinical research and treatment strategies.

Limitations: This study was a single-center, observational study. Although follow up was conducted, this study did not include any further review. Therefore, a prospective, large-scale multi-center study is required to confirm our conclusions. In addition, the collection of inflammation index was not comprehensive, and we could not confirm the direct relationship between inflammation and albumin/uric acid. Moreover, we considered only preoperative UAR. Future research should consider a larger number of patients in a prospective study with dynamic UAR calculation and monitoring over time.

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