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Research Article

Biochemical Predictors of New-Onset Atrial Fibrillation after Ascending Aorta Replacement Surgery in Acute Type A Aortic Dissection Patients

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Objective. This study aimed to determine the risk factors of new-onset postoperative atrial fibrillation after ascending aortic replacement in acute type A aortic dissection patients, with emphasis on biochemical parameters. *Methods*. From Jan 2020 to Dec 2021, a total of 435 acute type A aortic dissection patients who underwent ascending aortic replacement and without a history of atrial fibrillation were retrospectively analyzed in this study. Perioperative data of these patients were obtained from the hospital's database. The 30-day follow-up was via telephone interviews. The multivariate regression analysis was used to identify risk factors that may be predictive of postoperative atrial fibrillation. *Results*. 218 (50.1%) patients experienced postoperative atrial fibrillation after ascending aorta replacement surgery. Older age (OR = 1.081 (1.059–1.104), $p < 0.001$), higher total bile acid (OR = 1.064 (1.024–1.106), $p = 0.002$), glucose (OR = 1.180 (1.038–1.342), $p = 0.012$), and serum potassium (OR = 2.313 (1.078–4.960), $p = 0.031$) were identified by multivariate regression analysis as risk factors of postoperative atrial fibrillation. The multivariate regression analysis prediction model incorporating these four factors had a good prediction effect (AUC = 0.769 (0.723–0.816), $p < 0.001$). *Conclusions*. Older age, higher total bile acid, glucose, and serum potassium were risk factors of postoperative atrial fibrillation after ascending aortic replacement surgery in acute type A aortic dissection patients.

1. Introduction

New-onset postoperative atrial fibrillation (POAF) is one of the most common complications after cardiac surgery. The prevalence of POAF after cardiac surgery varies from 20% to 40%, according to different reports [1]. POAF is usually paroxysmal, recurrent, and occurs within 2 to 4 days after surgery [2]. Recent studies have shown that POAF after cardiac surgery would increase the risk of postoperative stroke and acute renal failure, and prolong the length of intensive care unit stay and hospital stay [3, 4]. POAF has been extensively studied in patients undergoing coronary artery bypass graft (CABG) and valve repair or replacement. However, the development of POAF after ascending aortic replacement (AAR) in acute type A aortic dissection (aTAAD) has received little attention. In this study, we will find risk factors for new-onset atrial fibrillation after AAR in aTAAD patients and analyze the relationship between POAF and clinical prognosis.

2. Methods

2.1. Patients. 435 patients with aTAAD in our center from Jan 2020 to Dec 2021 were retrospectively analyzed. All patients were diagnosed with enhanced CT scans by two experienced cardiovascular surgeons, and were finally confirmed by intraoperative exploration. The flow diagram including patients is shown in Figure 1.

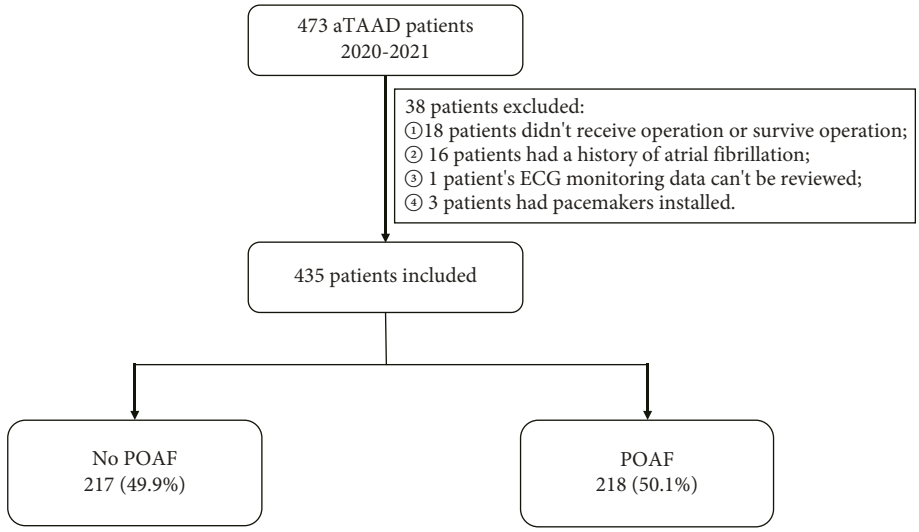

Figure 1: Schematic diagram of inclusion and exclusion criteria. aTAAD, acute type A aortic dissection; ECG, electrocardiograph; POAF, postoperative atrial fibrillation.

2.2. Surgical Treatments. All included patients underwent ascending aorta replacement surgery. Cardiopulmonary bypass (CPB) was established, and the cannulation approaches were chosen according to the surgical treatments, dissection lesion, and perfusion situation of the individual patient. Cardiac arrests in all patients were achieved with the cold-blood hyperkalemic cardioplegic solution. Deep hypothermic circulatory arrest (DHCA) was used in patients with the aortic arch repair. The patient was cooled down to 18–20°C if only deep hypothermia circulatory arrest (DHCA) was applied. While with the use of cerebral perfusion, the patient was cooled down to 24°C. The appropriate cerebral perfusion strategy was selected, including unilateral anterograde cerebral perfusion (UACP, 259 (60%)), bilateral anterograde cerebral perfusion (BACP, 18 (4.1%)), and retrograde cerebral perfusion (RCP, 34 (7.8%)). Root repair or replacement procedure was decided by the modified root classification of the aortic dissection [5]. Four kinds of aortic arch repair methods were in common use in our center, such as hemi-arch replacement, fenestrated stent implantation, modified "*in situ*" total arch replacement (MiTAR), and total arch replacement (TAR) [6–8]. According to the extension of the aortic arch resection, the hemi-arch replacement and fenestrated stent implantation are considered a limited arch replacement, and the others are considered an extended arch replacement.

2.3. Definition of Postoperative Atrial Fibrillation and Other Clinical Variables. POAF was defined as new-onset atrial fibrillation lasting more than 5 minutes as indicated by postoperative continuous telemetry monitoring or 12-leads ECG, which was confirmed by two experienced cardiologists. According to 2020 ESC guidelines, POAF was divided into paroxysmal or persistent atrial fibrillation [9]. Preoperative haemodynamic instability was defined as systolic blood pressure <80 mmHg and mean arterial pressure <50 mmHg. Pulmonary infection diagnostic criteria included lung infiltrate visible in chest X-ray and/or CT scan combined with respiratory symptoms, fever, leukopenia, or leukocytosis [10]. Patients who were still unconscious 6 h after surgery or with other neurological symptoms (seizure, hemiplegic, and memory impairment) would undergo a cranial CT [11], and perioperative stroke was defined as brain infarction confirmed by cranial CT scan that occurred within 30 days of surgery [12]. Acute renal failure was defined as requiring hemodialysis, peritoneal dialysis, hemofiltration, hemodiafiltration, or ultrafiltration, according to the NSQIP database.

2.4. Data Analysis. Preoperative, intraoperative data, and serological indicators on the first day after surgery were obtained from the hospital's database. The 30-day follow-up was via telephone interviews. Statistical analysis was performed with the SPSS version 20.0. The normality test was performed in continuous variables before difference tests. The normally distributed continuous variables were described as mean ± standard deviation (SD) and were compared by independent-samples T test. The non-normally distributed continuous variables were described as median with interquartile range and were compared by the Mann–Whitney test. Categorical variables were expressed as numbers with percentages and were compared by the Chi-squares test. Fisher exact test was performed when the sample size was less than five. Univariate regression analysis was used to identify risk factors that may be predictive for POAF, and these risk factors ($p < 0.05$) were included in multivariate regression analysis.

3. Results

3.1. Occurrence and Risk Factors of POAF after AAR Surgery. Among the 435 patients included in this study, 218 (50.1%) experienced POAF after surgery. Perioperative clinical data of these patients are shown in detail in Table 1. In general,

Table 1: Perioperative information of the 435 patients with or without POAF.

	No-POAF 217 (49.9%)	POAF 218 (50.1%)	p value
Preoperation			
Age (y)	49 ± 11	60 ± 13	**<0.001**
Male, *n* (%)	181 (83.5%)	164 (75.2%)	**0.035**
Body mass index (kg/m^2)	25.47 (23.25–28.06)	24.90 (22.86–27.35)	0.093
Hypertension, *n* (%)	160 (73.7%)	174 (79.8%)	0.133
Smoking history, *n* (%)	69 (31.8%)	57 (26.1%)	0.194
Alcohol consumption history, *n* (%)	54 (24.9%)	52 (23.9%)	0.802
Pericardial effusion, *n* (%)	169 (77.9%)	181 (83.0%)	0.176
Haemodynamic instability, *n* (%)	8 (3.7%)	13 (6%)	0.371
Intraoperation			
Operative time (h)	6.50 (6.0–7.8)	6.60 (5.6–8.0)	0.887
CPB time (min)	191.00 (161.00–230.75)	200.00 (165.25–240.00)	0.316
DHCA time (min)	26 (19–36)	25 (19–35)	0.629
Aortic clamp time (min)	142.0 (116.00–174.75)	142.5 (114.25–178.00)	0.432
Bentall procedure, *n* (%)	38 (17.5%)	39 (17.9%)	1.000
Wheat's procedure, *n* (%)	6 (2.8%)	7 (3.2%)	1.000
Aortic valve replacement, *n* (%)	44 (20.3%)	46 (21.1%)	0.906
Descending aorta stent implantation, *n* (%)	181 (83.4%)	179 (82.1%)	0.800
Extend arch repair, *n* (%)	60 (27.6%)	48 (22.0%)	0.184
Cerebral perfusion			0.141
No	61 (28.1%)	63 (28.9%)	
UACP	134 (61.8%)	125 (57.3%)	
BACP	11 (5.1%)	7 (3.2%)	
RCP	11 (5.1%)	23 (10.6%)	
LTN (°C)	22.95 (22.00–25.85)	22.70 (21.80–23.88)	0.309
RBC transfusion (U)	9.0 (5.5–12.5)	10.5 (7.5–15.0)	**0.002**
Postoperation			
Hemoglobin (g/L)	96 (84–107)	93 (85–105)	0.368
C-reactive protein (mg/L)	181.0 (145.8–218.1)	178.9 (140.9–218.1)	0.378
Apolipoprotein A (g/L)	0.62 (0.54–0.70)	0.61 (0.54–0.68)	0.191
Apolipoprotein B (g/L)	0.40 (0.32–0.51)	0.37 (0.30–0.45)	**0.008**
Adenosine deaminase (U/L)	7.2 (6.0–9.0)	8.0 (7.1–10.2)	**<0.001**
Leucine aminopeptidase (U/L)	40.50 (34.80–46.75)	40.00 (34.45–47.65)	0.877
Lactic dehydrogenase (U/L)	461 (370–651)	493 (382–798)	0.111
Aspartate aminotransferase (U/L)	48 (30.5–103.2)	54 (32.8–142.6)	0.159
Alanine aminotransferase (U/L)	21.6 (14.5–39.8)	21.5 (14.6–53.5)	0.47
Albumin globulin ratio	1.97 (1.78–2.24)	1.99 (1.78–2.26)	0.64
Cholesterol (mmol/L)	2.69 (2.33–3.05)	2.44 (2.14–2.9)	**0.001**
HDL (mmol/L)	0.69 (0.50–0.89)	0.68 (0.5–0.85)	0.624
LDL (mmol/L)	1.04 (0.81–1.35)	0.93 (0.69–1.16)	**0.001**
Cholinesterase (KU/L)	4.5 (3.9–5)	4.1 (3.6–4.7)	**0.002**
Triglyceride (mmol/L)	1.05 (0.79–1.43)	0.94 (0.68–1.4)	**0.049**
Total bile acid (umol/L)	3.4 (1.8–6.7)	4.1 (2.5–7.9)	**0.012**
Glucose (mmol/L)	6.68 (6.08–7.55)	7.17 (6.31–8.63)	**0.001**
Uric acid (umol/L)	295 (225–383)	309 (235–413)	0.291
Urea nitrogen (mmol/L)	9.8 (7.3–13.2)	11.6 (8.9–14.7)	**0.002**
Creatinine (umol/L)	95 (70–164)	110 (73–188)	0.134
Calcium (mmol/L)	2.12 (2.04–2.22)	2.12 (2.04–2.23)	0.95
Sodium (mmol/L)	146.8 (144–149.4)	147.2 (143.8–149.8)	0.447
Chlorine (mmol/L)	109 (105.9–111.8)	108.9 (106.4–112)	0.917
Phosphorus (mmol/L)	0.85 (0.68–1.07)	0.86 (0.71–1.12)	0.24
Potassium (mmol/L)	4.28 (4.11–4.47)	4.35 (4.18–4.52)	**0.015**

CBP time, cardiopulmonary bypass time; DHCA time, deep hypothermia circulatory arrest time; UACP, unilateral anterograde cerebral perfusion; BACP, bilateral anterograde cerebral perfusion; RCP, retrograde cerebral perfusion; LTN, lowest temperature of nasopharyngeal; RBC transfusion, red blood cell transfusion; HDL, high-density lipoprotein; LDL, low-density lipoprotein. The bold values in the p-value column are less than 0.05, which indicates statistical significance.

the age of the POAF group was higher than that of no-POAF (60 ± 13 vs. 49 ± 11, $p < 0.001$) and there were more females in the POAF group compared to the no-POAF group (24.8% vs. 16.5%, $p = 0.035$). There was no significant difference in body mass index, presence of hypertension, smoking history, alcohol consumption history, pericardial effusion, and haemodynamic instability between the two groups. Patients with and without POAF had similar duration of operative

time, cardiopulmonary bypass time, deep hypothermia circulatory arrest time, and aortic clamping time. There were no differences in the strategies of root management, arch management, cerebral perfusion, and percentage of descending aortic stent implantation between the two groups. The POAF group had more intraoperative red blood cell transfusion (10.5 vs. 9.0U, $p = 0.002$). Blood tests on the first day after surgery suggested that patients with POAF had higher levels of adenosine deaminase (8.0 vs. 7.2 U/L, $p < 0.001$), total bile acid (4.1 vs 3.4 umol/L, $p = 0.012$), glucose (7.17 vs. 6.68 mmol/L, $p = 0.001$), blood urea nitrogen (11.6 vs. 9.8 mmol/L, $p = 0.002$), and serum potassium (4.35 vs. 4.28 mmol/L, $p = 0.015$) and lower levels of apolipoprotein B (0.37 vs. 0.4 g/L, $p = 0.008$), cholesterol (2.44 vs. 2.69 mmol/L, $p = 0.001$), low-density lipoprotein (0.93 vs. 1.04 mmol/L, $p = 0.001$), cholinesterase (4.1 vs. 4.5 KU/L, $p = 0.002$), and triglyceride (0.94 vs. 1.05 mmol/L, $p = 0.049$). 12 risk factors were identified by univariate regression analysis (Table 2) and were included in multivariate regression analysis. Multivariate regression analysis revealed that older age (OR = 1.081 (1.059–1.104), $p < 0.001$), higher total bile acid (OR = 1.064 (1.023–1.106), $p = 0.002$), glucose (OR = 1.180 (1.038–1.342), $p = 0.012$), and serum potassium (OR = 2.313 (1.078–4.960), $p = 0.031$) were the risk factors of new-onset POAF after AAR in aTAAD patients (Table 3). These four factors were incorporated into a logistic regression prediction model, which had a good predictive effect (AUC = 0.769 (0.723–0.816), $p < 0.001$) and performed better than the prediction model that included age alone ($p = 0.01$) (Figure 2).

3.2. Features of POAF after AAR Surgery. Among the 218 patients with POAF, 131 (60.1%) patients experienced at least one recurrence during the postoperative period in the hospital. The first three days after surgery were the main time periods for the first episode of POAF (71.1%), with the highest incidence on the first day after surgery (21.56%) (Figure 3). Most POAFs were classified as paroxysmal atrial fibrillation (95.9%), with 9 patients (4.1% of POAF) developing into persistent atrial fibrillation. 19 patients (8.7% of POAF) maintained an atrial fibrillation rhythm at the time of discharge.

3.3. Outcomes. The clinical outcomes of patients with or without POAF are compared in Table 4. It shows that patients with POAF had a higher rate of reintubation (12.4% vs. 5.1%, $p = 0.007$), pulmonary infection (53.7% vs. 29.0%, $p < 0.001$), and acute renal failure (22.5% vs. 10.6%, $p = 0.001$). Patients with POAF had higher 30-day mortality (16.1% vs. 8.8%, $p = 0.021$) but not hospital mortality. The length of mechanical ventilation (34 vs 19 h, $p < 0.001$), intensive care unit stay (6 vs 4 d, $p < 0.001$), and hospital stay (18 vs 14 d, $p < 0.001$) were longer in POAF patients. The rate of reexploration and perioperative stroke did not differ between the two groups.

4. Discussion

4.1. POAF after AAR in aTAAD Patients. In our retrospective analysis, 50.1% of aTAAD patients had POAF after AAR surgery, which was higher than other cardiac procedures such as CABG (20–40%) [1]. The high incidence of POAF in aTAAD patients may be due to the severe systemic inflammatory response [13] and large surgical trauma. Similar to POAF after other kinds of surgery [14], POAF after AAR surgery usually developed in the early postoperative period, most in the first three days, and had a high recurrence rate (60.1%).

Postoperative atrial fibrillation leads to worse outcomes in patients after aortic surgery. Our data show patients with POAF after AAR surgery had increased 30-day mortality, increased rate of pulmonary infection, use of CRRT, and increased duration of mechanical ventilation, ICU, and hospital stay. Similar to our results, Dolapoglu and his colleagues [15] found patients who had cardiac arrhythmia, mostly POAF, after open thoracoabdominal aortic aneurysm (TAAA) repair, had higher 30-day mortality and longer ICU and hospital stay. Zhao et al. [16] noted that patients with POAF after aortic arch replacement had higher in-hospital mortality, higher hospitalization costs, longer hospitalization and ICU time, and higher postoperative complications, including postoperative stroke, sepsis, and lung infection.

4.2. Risk Factors. Advanced age, as a major risk factor for new-onset atrial fibrillation after cardiac surgery, has been revealed by several studies [1, 17]. In our study, advanced age was identified as a risk factor by both univariate and multivariate regression analysis. The mechanism may lie in that increased age provides a vulnerable atrial substrate for the development of POAF, including increased atrial fibrosis [18], decreased conduction velocity [19], and disturbed calcium homeostasis [20].

The relationship between laboratory tests after AAR and the occurrence of POAF has not been well investigated. Our study identified that elevated total bile acid, glucose, and serum potassium are the three serological risk factors of POAF after AAR surgery. These three factors have also been reported to be associated with atrial fibrillation occurring after other kinds of surgery or out of the setting of surgery.

Bile acids are the end products of cholesterol catabolism. Primary bile acids, such as cholic acid (CA) and chenodeoxycholic acid (CDCA), are synthesized by the liver and secreted into bile. When bile is secreted into the intestine, the bacterial 7α-dehydroxylase converts CA and CDCA to deoxycholic acid (DCA) and lithocholic acid (LCA), which are the secondary bile acids. The secondary bile acids are then reabsorbed by the intestine and returned to the liver through the portal vein system. During the enterohepatic cycle, some bile acids spill over into the systemic circulation [21]. The connection between increased total bile acids in the blood and the development of POAF may be related to the following three factors. First, the secretion of bile is regulated

Table 2: Univariate risk factors for POAF.

Risk factors	Univariate analysis		
	OR	95% CI	*p* value
Advanced age (y)	1.074	1.055–1.093	**<0.001**
Male gender	0.604	0.377–0.968	**0.036**
RBC transfusion (U)	1.054	1.021–1.088	**0.001**
Apolipoprotein B (g/L)	0.12	0.026–0.567	**0.007**
Adenosine deaminase (U/L)	1.133	1.054–1.217	**0.001**
Cholesterol (mmol/L)	0.573	0.397–0.826	**0.003**
Low-density lipoprotein (mmol/L)	0.484	0.294–0.797	**0.004**
Cholinesterase (KU/L)	0.724	0.575–0.912	**0.006**
Total bile acid (umol/L)	1.058	1.018–1.098	**0.004**
Glucose (mmol/L)	1.214	1.079–1.367	**0.001**
Urea nitrogen (mmol/L)	1.066	1.019–1.114	**0.005**
Potassium (mmol/L)	2.258	1.16–4.394	**0.017**

RBC transfusion, red blood cell transfusion. The bold values in the p-value column are less than 0.05, which indicates statistical significance.

Table 3: Multivariate risk factors for POAF.

Risk factors	Univariate analysis		
	OR	95% CI	*p* value
Advanced age (y)	1.081	1.059–1.104	**<0.001**
Total bile acid (umol/L)	1.064	1.023–1.106	**0.002**
Glucose (mmol/L)	1.180	1.038–1.342	**0.012**
Potassium (mmol/L)	2.313	1.078–4.960	**0.031**

Bold values in the p-value column are less than 0.05, which indicates statistical significance.

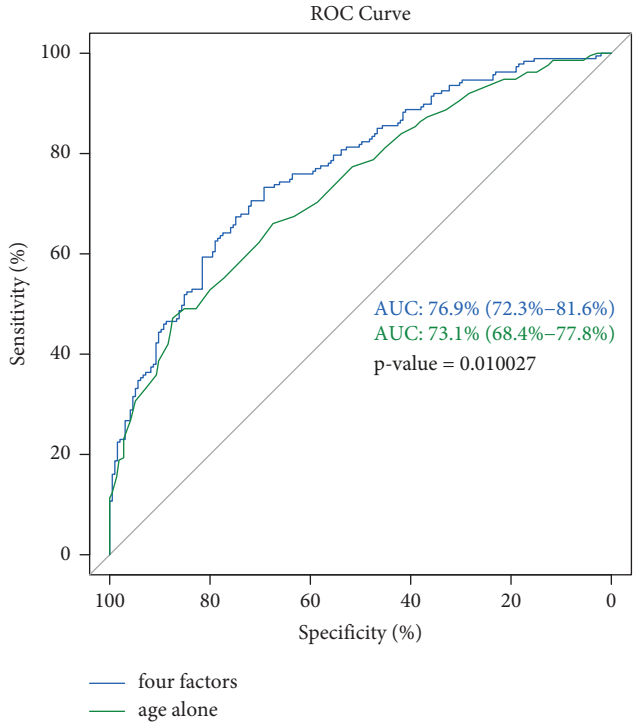

Figure 2: ROC curves for the multivariate regression analysis model and the age-only predictive model.

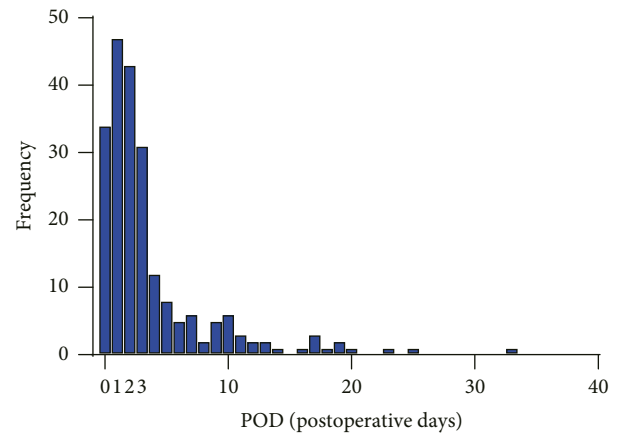

Figure 3: Temporal distribution of the first episode of atrial fibrillation after ascending aorta replacement surgery.

by the autonomic nerves. The sympathetic nerve increases secretin-induced bile secretion and Ca^{2+}/cAMP-dependent proliferation of cholangiocytes by stimulating $\alpha 1$ receptors on cholangiocyte cells [22], which could lead to cholestasis and an increase of total bile acids in the blood [23]. The autonomic nervous system is thought to be closely related to the development of POAF. The level of plasma noradrenaline is significantly increased in patients with POAF after cardiac surgery [24, 25], and prophylactic β-blockers are particularly effective in preventing POAF [26]. Based on the previously mentioned sympathetic activation after AAR surgery may link increased total bile acids to the development of POAF. The second point of relevance may be in terms of inflammation. Bile acids can induce endothelial cells to express adhesion molecules (ICAM-1 and VACM-1) [27] through the activation of reactive oxygen species, NF-kappa B, and p38. Meanwhile, levels of soluble VACM-1 are

Table 4: Clinical outcomes of the 435 patients with or without POAF.

	No-POAF 217 (49.9%)	POAF 218 (50.1%)	p value
Reintubation, n(%)	11 (5.1%)	27 (12.4%)	**0.007**
Re-exploration, n(%)	11 (5.1%)	18 (8.3%)	0.183
Pulmonary infection, n(%)	63 (29.0%)	117 (53.7%)	**<0.001**
Acute renal failure, n(%)	23 (10.6%)	49 (22.5%)	**0.001**
Perioperative stroke, n(%)	21 (9.7%)	30 (13.8%)	0.186
Mechanical ventilation time (h)	19 (12–43)	34 (17–99.5)	**<0.001**
Intensive care unit time (d)	4 (2.63–5.88)	6 (3.06–10)	**<0.001**
Hospital stay (d)	14 (11–18)	18 (13–24)	**<0.001**
Death in hospital, n(%)	16 (7.4%)	17 (7.8%)	0.867
Death within 30 days, n(%)	19 (8.8%)	35 (16.1%)	**0.021**

The bold values in the p-value column are less than 0.05, which indicates statistical significance.

significantly associated with new-onset AF [28]. However, due to the limitations of retrospective studies, this study did not include VACM-1 as a variable, and more studies are needed. Third, bile acids may directly regulate the electrophysiology of cardiomyocytes. Taurocholic acid, formed by CA conjugated to taurine, was proven to enhance Na^+/Ca^{2+} exchanger tail current density and induce afterdepolarisations of human atrial myocardium [29], which may play a role in the electrical remodeling of the atrium in POAF.

Higher blood glucose, the other risk factor in the present study, has been widely reported to be associated with various types of AF, such as age-related AF [30], diabetes-related AF [31], and POAF after CABG [32]. Hyperglycemia can induce endoplasmic reticulum stress, mitochondrial oxidative stress, and Ca^{2+} overload in atrial cardiomyocytes [33]. Ca^{2+} overload could cause ectopic trigger activity by delayed afterdepolarizations (DADs), which is typically needed for the initiation of AF.

Last but not least, potassium plays a pivotal role in atrial myocyte action potential duration and calcium handling, and dysregulation of both increases susceptibility to arrhythmias. Hypokalemia is traditionally believed to increase the risk of fatal ventricular arrhythmias after cardiac surgery. As a result, serum potassium at the upper half of the normal range (4.5–5.5 mmol/L) after cardiac surgery is more accepted [34]. In addition, our study showed that higher serum potassium, although within the normal range of values, was a risk factor for POAF after AAR surgery. Actually, low and high potassium can cause arrhythmias through different mechanisms. When the concentration of potassium outside the cell is low, the diastolic membrane potential is unstable, which leads to supernormal excitability and enhanced focal activity. On the other hand, hyperkalemia increases the conductivity of potassium current channels, also causing supernormal excitability and shortened action potential duration (APD) [35]. These changes may facilitate re-entry and increase the development of POAF.

4.3. Limitations. This study also has some limitations. First, the serum biochemical parameters we included in the multivariate regression analysis were detected on the first postoperative day, which delayed the time to identify patients at high risk for POAF. Second, this study only included patients from our single center, and the conclusions obtained need to be confirmed by data from other centers.

5. Conclusion

This study focused on the relationship between serum biochemical parameters after AAR surgery in aTAAD patients and the occurrence of POAF. Apart from older age, higher total bile acid, glucose, and serum potassium were identified as risk factors of POAF after AAR surgery. The occurrence of POAF was associated with short-term poor prognosis after AAR surgery, such as increased 30-day mortality.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Ethical Approval

The study was approved by the Institutional Review Board of Nanjing Drum Tower Hospital (2020-185-01).

Disclosure

Jian Shi and Yong-Qing Cheng shared the first authorship.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

Jian Shi and Yong-Qing Cheng contributed equally to the study.

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