

# Impact of unintentional coronary angiography on outcomes of emergency surgery in acute type A aortic dissection: a retrospective study

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## Research Article

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# Abstract

**Background:** This study investigated the impact of coronary angiography on outcomes of emergency operation in acute type A aortic dissection (ATAAD) patients who were initially misdiagnosed as the acute coronary syndrome.

**Methods:** From October 2016 to April 2019, 129 patients underwent surgery for ATAAD in our institution, including 21 patients (16.3%, CA group) who received preoperative coronary angiography without knowledge of the ATAAD, and the rest 108 did not (NCA group). Preoperative clinical characteristics, surgical mortality and postoperative complications were compared. Multivariable logistic regression was performed to confirm the independent prognostic factors for short-term and long-term outcomes.

**Results:** Patients undergoing coronary angiography had higher prevalence of preoperative hypotension or shock (61.9% vs 35.2%,  $P=0.022$ ), ischemic changes on electrocardiogram (66.7% vs 37.0%,  $P=0.012$ ), platelet inhibition (ADP-induced inhibition 92.0% vs 46.0%,  $P=0.001$ ), and coronary involvement (66.7% vs 30.6%,  $P=0.002$ ). 30-day/in-hospital mortality was 4.8% versus 9.3% ( $P=0.84$ ). CA group had more intraoperative bleeding (1900ml vs 1500ml,  $P=0.013$ ) and chest-tube drainage on the first postoperative day (1040ml vs 595ml,  $P=0.028$ ). However, preoperative coronary angiography was not independent risk factors for surgical mortality (OR 0.171, 95%CI 0.013-2.174,  $P=0.173$ ) and overall survival (HR 0.407; 95%CI 0.080-2.057;  $P=0.277$ ).

**Conclusion:** Patients undergoing coronary angiography carried a higher risk of preoperative hemodynamic instability, myocardial ischemia, and perioperative bleeding. However, unintentional coronary angiography did not have a significant impact on short-term and long-term outcomes of emergency surgery in ATAAD.

## Background

Acute type A aortic dissection (ATAAD) is one of the most lethal cardiovascular diseases, the post-onset mortality rate of which increased 1% to 2% per hour on the first day without surgical intervention [1]. Emergency surgical repair was proved to be the best therapeutic option in these circumstances [2,3]. Survival relies on the rapid accurate diagnosis and timely lifesaving emergency operation.

The clinical presentation of ATAAD and acute coronary syndrome (ACS) can be remarkably similar, making differential diagnosis very difficult [4,5]. Patients with ATAAD were occasionally misdiagnosed as ACS and transferred to a catheterization laboratory to undergo coronary angiography (CA) without knowledge of the ATAAD (unintentional CA) [6-8]. Unintentional coronary angiography might result in significant surgical delay, not only due to time consumption in search of coronary ostia in the diseased aortic root, but also the administration of overload dose of dual antiplatelet agents, inevitably resulting in coagulopathy and significant risk of perioperative bleeding, which makes the decision of emergency surgery very difficult [9-11]. Previous studies had shown that emergency operation was postponed or even concealed in ATAAD patients who undergoing preoperative CA [12,13] which undoubtedly increased the

risk of death. In addition, time consumed by angiography, the instrumentation of the diseased aorta, and the myocardial ischemia by coronary ostium involvement were also associated with increased risk of dissection progression, hemodynamic collapse, and aortic rupture [14-16].

There was only a little research about the effect of coronary angiography on the ATAAD, in most of which the angiography was performed with awareness of ATAAD to clarify concomitant coronary artery disease [12,14-16]. In comparison with unintentional coronary angiography, these intentional coronary angiographies had a relatively small effect on the prognosis of ATAAD patients because dual antiplatelet therapy was not prescribed and patients with unstable hemodynamics were excluded. Therefore, the aim of this study was to explore the impact of unintentional coronary angiography on the outcomes of emergency surgery in ATAAD patients.

## Methods

### Study population and study design

As the regional aortic center, our tertiary heart hospital (Shanghai DeltaHealth Hospital) received ATAAD patients for emergency surgical intervention from transferring hospitals where the primary diagnosis of aortic dissection was made. The clinical database was queried to include all patients admitted to our institution with a diagnosis of ATAAD and undergoing emergency operation between October 2016 and April 2019. The exclusion criteria were as follows: (1) the time from onset to admission longer than 7 days; (2) the time from admission to surgery longer than 24 hours; (3) emergency operation for giant true aneurysm or type B aortic dissection; (4) patients who were too sick for surgery or refused surgery; (5) patients died before emergency operation. According to whether preoperative coronary angiography was performed, the patients were divided into CA group and NCA group. The research protocol for this study was reviewed and approved by the Medical Ethics Committee of Shanghai DeltaHealth Hospital [No. SDH(2020) KYLWZQTYPJ001], and informed consent was approved to be waived for its retrospective nature by the Medical Ethics Committee of Shanghai DeltaHealth Hospital. Medical records were reviewed for data collection. Follow-up was performed by outpatient clinical visit and telephone contact. The last follow-up date was April 30<sup>th</sup>, 2020.

### Clinical Management

All operations were performed through a median sternotomy incision and on cardiopulmonary bypass (CPB). The hypothermic circulatory arrest was initiated at 25 °C and with unilateral selective cerebral perfusion in all patients. For proximal aortic repair, Bentall procedure was performed if with severe aortic sinus dilation ( $\geq 4.5\text{cm}$ ) and aortic valve insufficiency. Otherwise, aortic valve repair and replacement of the ascending aorta replacement were conducted. For distal aortic repair, total arch replacement and frozen elephant trunk procedures were routinely conducted if the aortic arch was involved by the dissection unless the patient was too frail to undergo such procedures. For those frail patients, partial replacement of the aortic arch was performed. The coronary repair was applied when the coronary ostium

was compressed by hematoma in the false lumen. If discontinuity of the ostium or coronary artery occurred, coronary artery bypass grafting would be performed. The autologous aortic wall was wrapped around the graft to create a 'perigraft to right atrial shunt' following aortic repair in all cases. Red blood cell was transfused when the blood hemoglobin level decreases to <70g/L in stable patients and <90g/L in patients with symptomatic anemia. Plasma was transfused in patients with ongoing bleeding and signs of impaired coagulation. Platelet was transfused in patients with ongoing bleeding and suspected platelet dysfunction if available. Re-operation was performed in case of chest tube drainage > 300 milliliters per hour, a persistent fall in hemoglobin and unstable hemodynamics, or suspected cardiac tamponade.

## **Data collection and outcome**

Clinical data were collected using a standardized data form and standardized definitions, including patient demographic and medical history, preoperative clinical characteristics, coagulation function, intraoperative data, perioperative bleeding, postoperative complications, and mortality. Patient demographic included age, sex, body surface area, and history of smoking. Medical history included a history of cardiac surgery, hypertension, type 2 diabetes mellitus, and coronary artery diseases. Preoperative clinical characteristics included hypotension or shock, cardiac tamponade, antiplatelet therapy, intramural hematoma, Marfan syndrome, time from onset to admission and operation. Coagulation function included international normalized ratio, activated partial thromboplastin time, fibrinogen, D-Dimer, platelet count and the results of thromboelastography. Intraoperative data included procedures performed for proximal and distal aortic repair and coronary interventions, operative time, and time of cardiopulmonary bypass. Perioperative bleeding was assessed by the volume of intraoperative bleeding, chest tube drainage on the first three postoperative days, blood transfusion, and the rate of re-exploration for bleeding. Postoperative complications included low cardiac output syndrome, cardiac tamponade, new-onset atrial fibrillation, respiratory insufficiency requiring tracheostomy, reintubation, acute renal failure requiring continuous renal replacement therapy (CRRT), and any neurological deficiency. The length of intensive care unit (ICU) stay and hospital stay, re-admission to ICU, and 30-day re-admission to the hospital were also documented to evaluate the postoperative recovery course.

The outcomes of this study were as follows: (1) operative mortality rate, including mortality in the hospital or within 30 days of surgery; (2) perioperative bleeding complications; (3) postoperative complications rate within 30 days; (4) overall survival (OS).

## **Statistical Analysis**

Statistical analysis was conducted using SPSS version 23.0 (SPSS, Chicago, Illinois, USA). Normally distributed continuous variables were expressed as mean  $\pm$  standard deviation (SD) and analyzed by unpaired Student's *t*-test. Non-normally distributed continuous data were expressed as median (M) and interquartile range (IQR: Q25%-Q75%) and analyzed by Mann-Whitney *U* test. Categorical variables were reported as frequency and analyzed using Chi-square analysis or Fisher's exact test. The Kaplan-Meier method was used to estimate the OS probabilities, and differences were compared using the log-rank

test. Univariate analysis was performed to examine the association of each variable with the outcome variable. Binary logistic regression was used to identify independent predictors of operative mortality. Survival prognostic factor analysis was performed using the Cox regression model. For both the binary logistic regression model and the Cox regression model, the candidate variables were selected by using stepwise variable selection ( $\alpha_{\text{ste}}=0.05$ ,  $\alpha_{\text{sls}}=0.1$ ). The 'preoperative CA' was then forced to enter the binary logistic regression and the Cox regression model, respectively, with independent predictors of operative mortality and overall survival, to identify the impact of preoperative CA on early mortality and long-term survival. Two-tailed  $P$ -values of  $<0.05$  were considered significant.

## Results

A total of 141 patients was identified to be admitted to our hospital with a diagnosis of ATAAD during this period. Of these, 12 patients (8.5%) were excluded because they received no surgery, including 7 patients who died suddenly before surgery. Of these excluded patients, 25% (3 patients) had undergone preoperative CA, all of whom died of aortic rupture. Finally, 129 patients were enrolled in this study. Of these patients, 21 (16.3%, CA group) received preoperative CA at local hospitals, all initially suspected of having ACS. The diagnosis of aortic dissection was identified during the angiography procedure and confirmed with the following computed tomography. The rest 108 patients (NCA group) were initially diagnosed with aortic dissection by computed tomography without coronary angiography.

### Preoperative clinical characteristics

Baseline demographic and clinical information is presented in Table 1. Patients in the CA group had a male predominance and received significantly more antiplatelet therapy and dual antiplatelet therapy than the NCA group (100% vs 0.9%,  $P<0.001$ ; 85.7% vs 0%,  $P<0.001$ ; respectively). Patients in the CA group were significantly more likely to experience preoperative hypotension or shock than those in the NCA group (61.9% vs 35.2%,  $P=0.022$ ).

An electrocardiogram (ECG) and plasma troponin test were performed on admission. Ischemic changes on preoperative ECG (including ST-segment elevation or depression, T-wave inversion, Q-wave and left bundle branch block) were almost double in the CA group compared to the NCA group (66.7% vs 37.0%,  $P=0.012$ ). The median plasma level of troponin I in the CA group was numerically higher than in the NCA group but was not statistically significant (0.048ng/ml vs 0.022ng/ml,  $P=0.266$ ).

The other demographics, medical history, and clinical presentations showed no difference between groups. The time from symptom onset to admission (CA 10.0hours vs NCA 11.0hours,  $P=0.297$ ) and that from admission to operation (CA 1.5hours vs NCA 1.5hours,  $P=0.926$ ) were similar between the two groups.

### Preoperative coagulopathy, perioperative bleeding, and blood transfusion

Platelet aggregation inhibition induced by adenosine diphosphate (ADP) and arachidonic acid (AA) was significantly higher in the CA group than in the NCA group (92.0% vs 46.0%,  $P=0.001$ ; 91.4% vs 71.0%,  $P=0.042$ ; respectively). Other indicators of coagulation were comparable between the two groups (Table 2).

Median intraoperative bleeding volumes in the CA group were significantly higher than in the NCA group (1900ml vs 1500ml,  $P=0.013$ ). Volumes of chest tube drainage were also significantly higher than those in the NCA group on the operative day (1040ml vs 595ml,  $P=0.028$ ). However, chest tube drainage was comparable on the first and second postoperative days between groups. There was no significant difference in re-operation for bleeding and postoperative cardiac tamponade between the two groups.

Volumes of platelet transfusion in the CA group were significantly more than NCA group (0U vs 1U,  $P=0.003$ ) whereas no difference was found in transfusion of red blood cell (RBC) (CA 8U vs NCA 6U,  $P=0.149$ ) and plasma (CA 18U vs NCA 12U,  $P=0.075$ ).

### **Intraoperative Interventions**

No differences were identified between the two groups in the proximal or distal aortic repair strategies (Table 3). Intraoperative exploration revealed that coronary involvement was much more prevalent in the CA group than in the NCA group (66.7% vs 30.6%,  $P=0.002$ ). The CA group had significantly more coronary repairs than the NCA group (61.9% versus 25.9%,  $P=0.001$ ), but coronary artery bypass grafting (CABG) was comparable between the groups. The operative time and time of cardiopulmonary bypass were similar among the groups.

### **Survival outcomes and postoperative complications**

Operative mortality was not significantly different between the CA and NCA groups (4.8% vs 9.3%,  $P=0.804$ ). The follow-up time was  $20.1 \pm 8.4$  months. Two patients were lost to follow-up after 30 days post discharge, with a follow-up rate of 98.4%. No significant differences in accumulative overall survival were found between the CA and NCA groups (90.5% vs 88.0%,  $P=0.739$ ) (Figure 1).

Overall, there were no significant differences in major postoperative complications. However, the incidence of intensive care unit (ICU) readmission (CA=30.0% vs NCA=7.7%,  $P=0.012$ ) and new-onset atrial fibrillation (CA=47.6% vs NCA=19.4%,  $P=0.006$ ) were significantly higher in the CA group than in the NCA group (Table 4). The median length of hospital stay was numerically much longer in the CA group than the NCA group, although did not reach statistical significance (18.0days vs 13.0days,  $P=0.056$ ). The echocardiography performed before discharge revealed no differences in the left ventricular ejection fraction, the fractional shortening, and the incidence of wall motion abnormalities between groups. The median brain natriuretic peptide (BNP) before discharge was also comparable across the groups (CA 329.0 pg/ml vs NCA 297.0 pg/ml,  $P=0.443$ ).

### **Multivariate analysis**

Multivariate analysis showed that intramural hematoma (OR 7.375; 95% CI 1.048-51.884, P=0.045), CPB time (OR 1.023; 95% CI 1.005-1.042; P=0.012), postoperative hemodialysis (OR 59.610; 95% CI 7.748-458.613; P<0.001), and cerebral hemorrhage (OR 63.181; 95% CI 4.067-981.416; P<=0.003) were independent risk factors for operative mortality. Coronary angiography was not a significant risk factor for operative mortality after adjusting for the above variables (OR 0.171, 95%CI 0.013-2.174, P=0.173).

Cox regression model showed that preoperative creatinine (HR 1.006; 95%CI 1.003-1.009; P<0.001), preoperative lactic acid (HR 1.487; 95%CI 1.260-1.756; P<0.001), intramural hematoma (HR 11.767; 95%CI 2.707-51.143; P=0.001), CPB time (HR 1.017; 95%CI 1.007-1.027; P=0.001), reintubation (HR 5.262; 95%CI 1.028-26.936; P=0.046), and postoperative cerebral hemorrhage (HR 27.164; 95%CI 3.312-222.826; P=0.002) were independent prognostic factors for OS. Preoperative coronary angiography was not a significant prognostic factor for OS after adjusting for the above variables (HR 0.407; 95%CI 0.080-2.057; P=0.277).

## Discussion

Angiography had once been considered useful for confirming the diagnosis and defining coronary anatomy in patients with aortic dissection [17,18]. With the development of noninvasive imaging techniques, this technique was no longer routinely recommended for patients with ATAAD in consideration of the increased risk of dissection progression, cardiac tamponade, and aortic rupture due to delay of surgery and instrumentation of the diseased aorta [14-16]. Nevertheless, owing to the high similarity of clinical presentation between the two diseases, patients with ATAAD might be transferred to the catheterization lab due to misdiagnosis of ACS. It was reported that the incidence of preoperative coronary angiography employment for patients with ATAAD was 9.3%-21.9% [6,12,19]. However, the diagnosis of ATAAD had either already been confirmed or not well documented when the coronary angiographies were performed in these studies. Thus, the present study focused on the impact of coronary angiography performed without knowledge of the ATAAD on outcomes of emergency surgery in ATAAD. Based on our results, 16.3% of ATAAD patients underwent coronary angiography due to misdiagnosis of ACS, indicating a relatively common scenario in this population.

There were significantly more popular ischemic changes on preoperative ECG and more coronary involvement in the CA group, indicating that patients undergoing unintentional coronary angiography had a higher risk of myocardial ischemia. Preoperative hypotension or shock was also more common in the CA group, reflecting the hemodynamic compromise in these patients. As the incidence of cardiac tamponade was comparable across groups, myocardial ischemia might be the dominant cause of hemodynamic changes. Interestingly, the value of troponin did not achieve a significant difference between groups. These results were consistent with the data obtained by Pourafkari, which showed that troponin increase was not different in ATAAD patients with acute ECG changes [19]. The possible mechanism of this phenomenon might lie in that compromise of the coronary perfusion would be intermittent, incomplete, and variable when it was caused by coronary ostium obstruction of the local dissection flap instead of complete coronary detachment [18,20]. This hypothesis was also supported by

our comparable incidence of CABG applied between the two groups, which was performed only in case of complete avulsion of the coronary artery [18,21].

In comparing with those who received angiography with awareness of ATAAD, patients undergoing unintentional coronary angiography were exposed to additional risk of perioperative coagulopathy and bleeding due to the administration of loading dose of antiplatelet drugs before angiography, which contributed mainly to the emergency operation delay or cancellation [22]. The higher inhibition of platelet aggregation and the more volume of intraoperative bleeding and chest tube drainage on the operative day demonstrated a higher risk of bleeding in the CA group in the early perioperative period. These findings were consistent with other studies showing the association of bleeding and antiplatelet therapy [23,24]. However, there was no significant difference between the two groups to the re-exploration for bleeding or the total amount of RBCs and plasma transfusion, suggesting the difference in bleeding had not resulted in severe clinical consequences. This might likely be attributed to our surgical strategy during emergency aortic operations. When vascular reconstruction had been done, the aneurysmal wall was routinely wrapped around the graft and a fistula was made on the right atrium to drain oozing blood from the aneurysmal sac, which significantly reduced the volume of chest drainage and the subsequent requirement of transfusion. In this way, we achieved good control of bleeding even in patients with antiplatelet therapy and a shortage of platelets in case of emergency.

Surprisingly, our results did not show significant time delay from onset to operation in the CA group, which differed from some published studies. It was reported that delays in correct diagnosis were quite common in patients with initial misdiagnosis of ATAAD [25,26], and delay to surgery was popular and associated with the poor prognosis in ATAAD patients undergoing intentional preoperative coronary angiography [15,16,27]. These differences could be explained in part by the strict time requirement for coronary angiography based on the current guideline for ACS [28], thus minimizing time consumption in the emergency room on detection of the definitive diagnosis. In addition, unintentional coronary angiographies were always be ceased immediately upon suspicion of ATAAD, which meant a much shorter time in the catheterization laboratory as compared to intentional coronary angiography. Furthermore, according to our institutional protocols, all the involved patients were transferred to the operating theater as soon as possible no matter the antiplatelet therapy was administrated or not (median time from admission to the operation were 1.5 hours in both groups), which avoided delayed treatment due to the surgical strategies.

Although unintentional coronary angiography was associated with the risk of hemodynamic instability, myocardial ischemia, and perioperative bleeding, the increase in surgical mortality was not significant, which proved the necessity and effectiveness of emergent surgical management in this scenario. The surgical delay was considered the main cause of poor hospital mortality in patients who underwent the coronary angiography performed with knowledge of the ATAAD diagnosis, which was not the case in the present study [16,27]. Antiplatelet therapy was also reported to be associated with increased early mortality in ATAAD patients with initial misdiagnosis, attributed to the increased perioperative bleeding and transfusion [23,25]. Although the early perioperative bleeding was greater in the CA group, the

transfusion of RBC and plasma were comparable between the groups, suggesting that perioperative bleeding in this study appears to have a relatively minor impact on clinical outcomes.

In our study, unintentional coronary angiography was associated with higher rates of ICU readmission (30.0% vs 7.7%,  $P=0.012$ ) and a trend toward the longer length of ICU stay (92.0hours vs 63.5hours,  $P=0.193$ ) and hospital stay (18days vs 13days,  $P=0.056$ ), reflecting a more arduous course of recovery. Previous studies had described that preoperative hypotension or shock and the major bleeding within the initial 24 hours were independent prognostic factors of in-hospital mortality, while no significant abnormal ECG findings predicted excellent in-hospital survival [15,29]. Considering that patients undergoing coronary angiography had been exposed to such risk factors, the slower recovery process was easy to understand, and the value of timely and appropriate operative interventions in this circumstance was confirmed.

We did not find the long-term effect of unintentional coronary angiography on prognosis. Ramanath and colleagues (2011) reported increased trends in long-term mortality among patients undergoing preoperative coronary angiography and speculated more preexisting coronary artery disease in this population as the potential underlying cause [12]. Patients in the present study underwent coronary angiography due to misdiagnosis instead of clarifying the coronary anatomy, thus they were younger and had fewer comorbidities such as diabetes mellitus and coronary artery disease. It was reasonable that they had similar long-term survival compared with patients without coronary angiography after the early postoperative phase of critical illnesses.

In conclusion, unintentional coronary angiography was not uncommon in patients with ATAAD and was associated with a higher risk of perioperative bleeding, myocardial ischemia, and hemodynamic instability. However, the surgical mortality and long-term mortality were not significantly influenced by unintentional coronary angiography given timely and appropriate surgical repairs.

## **Limitations**

The limitations of this study are its retrospective nature and a relatively small number of subjects. In addition, all patients were initially presented to their local hospitals and transferred to our center for further surgical treatment. There could be inherent selection bias, as critically ill patients could have no opportunity to be transferred or die before admission to our hospital. As mortality was proved better in high-volume centers compared to low-volume centers, and patients transferred to high-volume centers had similar outcomes as direct admits, transferring patients to the center of 'aortic teams' was common and recommended nowadays [3,30]. Our results reflected the prognosis of ATAAD patients in this 'real world' scenario.

## **Declarations**

### **Ethics approval and consent to participate**

All methods performed in this study were in accordance with the Declaration of Helsinki. The research protocol for this study was reviewed and approved by the Medical Ethics Committee of Shanghai DeltaHealth Hospital [No. SDH(2020) KYLWZQTYPJ001]. Informed consent was approved to be waived for its retrospective nature by the Medical Ethics Committee of Shanghai DeltaHealth Hospital.

### **Consent for publication**

Not applicable.

### **Availability of data and materials**

All data generated or analyzed during this study are included in this published article.

### **Competing interests**

All authors declare no competing interests.

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### **Authors' contributions**

YQM and LZS conceived and supervised the study. HP and YQM designed experiments; HP, WL, KTJ and YX collected data; HP and JSL analyzed data; HP wrote the manuscript; YQM and LZS made manuscript revisions. All authors read and approved the final manuscript

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## Tables

Table 1. Preoperative clinical characteristics

Variable	CA group (n=21)	NCA group [n=108]	P
Male [n (%)]	19 (90.5%)	72 (66.7%)	<b><u>0.030</u></b>
Age [years, mean±SD]	54.0±7.8	54.3±13.0	0.865
Body surface area [m <sup>2</sup> , mean±SD]	1.89±0.20	1.84±0.25	0.332
Cardiac surgery history [n (%)]	0 (0.0%)	5 (4.6%)	0.591
Hypertension [n (%)]	15 (71.4%)	75 (69.4%)	0.856
Diabetes mellitus [n (%)]	1 (4.8%)	3 (2.8%)	0.513
Coronary artery disease history [n (%)]	0 (0%)	2 (1.9%)	1.000
Smoking history [n (%)]	13 (61.9%)	44 (40.7%)	0.074
Intramural hematoma [n (%)]	5 (23.8%)	23 (21.3%)	1.000
Marfan syndrome [n (%)]	0 (0.0%)	10 (15.2%)	0.217
Any antiplatelet therapy [n (%)]	21 (100%)	1 (0.9%)	<b><u>&lt;0.001</u></b>
Dual antiplatelet therapy [n (%)]	18 (85.7%)	0 (0.0%)	<b><u>&lt;0.001</u></b>
Hypotension/shock [n (%)]	13 (61.9%)	38 (35.2%)	<b><u>0.022</u></b>
Cardiac tamponade [n (%)]	4 (19.0%)	25 (23.1%)	0.900
Ischemic changes on ECG [n (%)]	14 (66.7%)	40 (37.0%)	<b><u>0.012</u></b>
Troponin I [ng/ml, M (IRQ)]	0.048 (0.251)	0.022 (0.161)	0.266
Duration: onset to admission [hours, M (IRQ)]	10.0 (12.0)	11.0 (17.0)	0.297
Duration: admission to operation [hours, M (IRQ)]	1.5 (3.0)	1.5 (2.9)	0.926
Duration: onset to operation [hours, M (IRQ)]	12.0 (17.0)	14.0 (18.8)	0.442

Table 2. Preoperative coagulation function, perioperative bleeding, and blood transfusion.

Variable	CA group (n=21)	NCA group [n=108]	P
<b>Coagulation function</b>			
INR [M (IRQ)]	1.04 (0.14)	1.04 (0.15)	0.245
aPTT [seconds, M (IRQ)]	30.9 (7.6)	29.0 (8)	0.327
Fibrinogen [g/L, M (IRQ)]	2.00 (1.25)	2.20 (1.57)	0.318
D-Dimer [mg/L, M (IRQ)]	13.9 (26.8)	9.34 (17.9)	0.697
Platelet count (10 <sup>9</sup> /L, mean±SD)	168.6±62.3	157.5±66.6	0.482
<b>Thromboelastography</b>			
R [reaction time, min, M (IRQ)]	5.10 (3.37)	4.70 (2.00)	0.787
K [potentiation phase, min, M (IRQ)]	2.20 (1.10)	1.80 (0.85)	0.305
α [rate of clot, degree, M (IRQ)]	61.0 (21.7)	62.4 (17.5)	0.411
MA [maximum amplitude, mm, M (IRQ)]	60.0 (12.0)	61.3 (11.5)	0.679
LY30 [fibrinolysis, %, M (IRQ)]	0.00 (0.10)	0.00 (0.05)	0.822
AA-induced platelet inhibition [%, M (IRQ)]	92.0 (43.6)	46.0 (44.2)	<b>0.001</b>
ADP-induced platelet inhibition [%, M (IRQ)]	91.4 (36.4)	71.0 (55.4)	<b>0.042</b>
<b>Perioperative bleeding</b>			
Intraoperative Bleeding [ml, M (IRQ)]	1900 [1925]	1500 [1100]	<b>0.013</b>
Chest Tube Drainage [ml, M (IRQ)]			
Operative Day	1040 [1030]	595 [685]	<b>0.028</b>
1st Postoperative Day	460 [477.5]	320 [440] &	0.567
2nd Postoperative Day	150 [280]	130 [204] #	0.642
Re-exploration for Bleeding [n (%)]	3 (14.3%)	14 (13.0%)	1.000
<b>Blood transfusion</b>			
RBC [U, M (IRQ)]	8 (14)	6 (10)	0.149
Plasma [U, M (IRQ)]	18 (25)	12 (14)	0.075
Platelets [U, M (IRQ)]	1 (2)	0 (1)	<b>0.003</b>

INR, international normalized ratio; aPTT, activated partial thromboplastin time; ADP, adenosine diphosphate; AA, arachidonic acid. &Missing data due to death of patients, n=107. # Missing data due to death of patients, n=106.

Table 3. Surgical intervention

Variable	CA group (n=21)	NCA group (n=108)	P
Proximal aortic repair			
Bentall procedure [n (%)]	4 (19.0%)	34 (31.5%)	0.253
Aortic valvuloplasty [n (%)]	7 (33.3%)	25 (23.1%)	0.323
Ascending aorta replacement [n (%)]	17 (81%)	70 (64.8%)	0.205
Distal aortic repair			
Hemi-arch replacement [n (%)]	1 (4.8%)	16 (14.8%)	0.372
Sun's procedure [n (%)]	18 (85.7%)	73 (67.6%)	0.096
Coronary Intervention			
Ostium repair [n (%)]	13 (61.9%)	28 (25.9%)	<b>0.001</b>
CABG [n (%)]	1 (4.8%)	4 (3.7%)	1.000
Overall intervention [n (%)]	14 (66.7%)	32 (29.6%)	<b>0.006</b>
Operative time [minutes, M (IRQ)]	319 (187.5)	323.5 (112.3)	0.674
Time of CPB [minutes, M (IRQ)]	168.0 (47.5)	160.5 (47.6)	0.794
Time of ACC [minutes, M (IRQ)]	86.0 (30.0)	87.5 (24.0)	0.728
Time of DHCA [minutes, M (IRQ)]	17.0 (6.0)	18.0 (7.8)	0.627

CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; ACC, aortic crossclamping; DHCA, deep hypothermic circulatory arrest; RBC: red blood cell; Sun's procedure: total arch replacement + frozen elephant trunk procedure.

Table 4. Surgical mortality and postoperative complications

Variable	CA group (n=21)	NCA group (n=108)	P
surgical mortality [n (%)]	1 (4.8%)	10 (9.3%)	0.804
Cardiac complications			
Low cardiac output syndrome [n (%)]	0 (0.0%)	6 (5.6%)	0.588
Cardiac tamponade [n (%)]	2 (9.5%)	13 (12.0%)	1.000
New-onset atrial fibrillation [n (%)]	10 (47.6%)	21 (19.4%)	<b>0.006</b>
Echocardiogram before discharge			
LV ejection fraction [%, mean±SD]	60.9±6.9	61.3±7.8	0.834
LV fractional shortening [%, mean±SD]	32.7±4.8	32.9±5.5	0.860
LV Wall motion abnormality [n (%)]	2 (9.5%)	13(12.1%)	1.000
Re-intubation [n (%)]	2 (9.5%)	13 (12.0%)	1.000
Tracheotomy [n (%)]	2(9.5%)	12(11.1%)	1.000
Acute renal failure [n (%)]	2 (9.5%)	6 (5.6%)	0.845
Coma [n (%)]	1 (4.8%)	10 (9.3%)	0.804
Hypoxic-ischemic encephalopathy [n (%)]	1 (4.8%)	6 (5.6%)	1.000
Cerebral infarction [n (%)]	2 (9.5%)	13(12.0%)	1.000
Cerebral hemorrhage [n (%)]	1(4.8%)	3(2.8%)	0.513
ICU stay [hours, M (IRQ)]	92.0 (129.8)	63.5 (68,7)	0.193
ICU readmission* [n (%)]	6 (30.0%)	8 (7.7%)	<b>0.012</b>
Hospital stay [days, M (IRQ)]	18.0 (16.5)	13.0 (10.0)	0.056
30-day readmission* [n (%)]	2 (10%)	17 (16.3%)	0.702

ICU, intensive care unit. \*As patients died in ICU were excluded, 20 in the CA group and 104 patients in NCA group were included.

## Figures

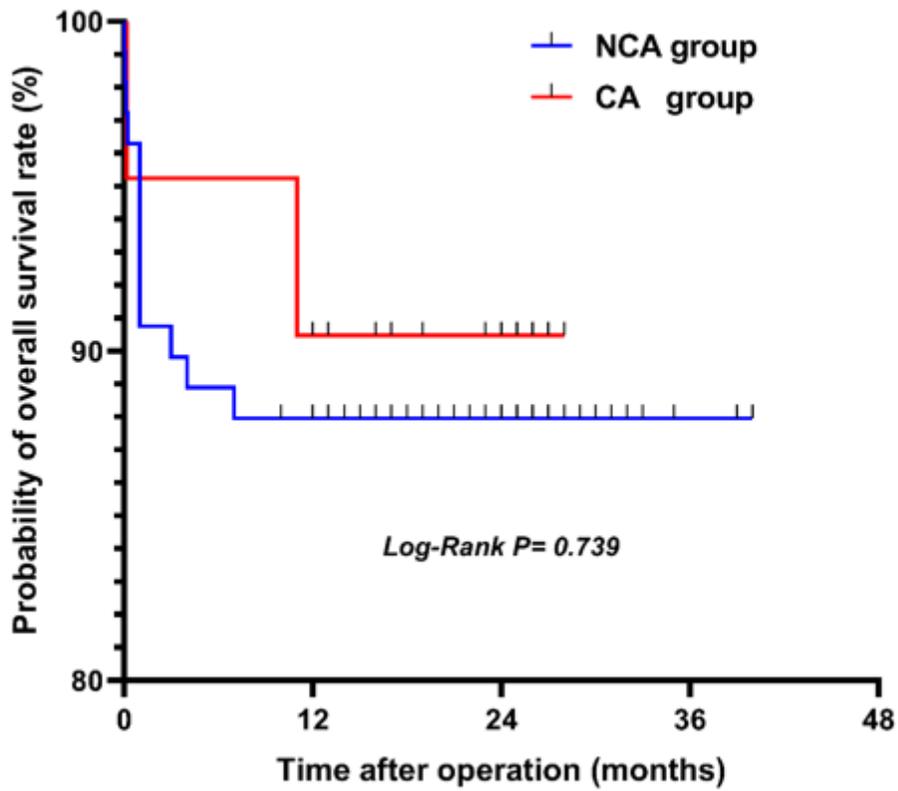


Figure 1

Survival analysis showed no significant difference in overall survival between the two groups