Case Report

Perioperative mortality in patients with acute aortic dissection and coronary artery problems: report of 3 cases and literature review

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Background: Acute type A aortic dissection is a critical condition associated with elevated mortality and disability rates, influenced by numerous risk factors. One significant contributor to intraoperative mortality is intraoperative myocardial ischemia or infarction, which can result in cardiac dysfunction following cardiac reperfusion.

Case Description: Case 1 was a 46-year-old male who was admitted for 22-hour sudden chest distress and pain. Intraoperatively, myocardial swelling and cyanosis were observed in the proximal-middle right coronary artery (RCA). Dissection extended to the right and non-coronary sinuses and the aortic annulus, resulting in complete avulsion of the RCA ostium. Aortic total arch replacement and RCA bypass grafting were performed. Following cardiac reperfusion, the patient developed bradycardia and hypotension, ultimately succumbing despite resuscitative efforts, with an estimated blood loss of approximately 2,000 mL. Case 2 was a 55-year-old male who presented with 3-hour sudden chest pain. Intraoperative exploration revealed aortic true and false lumens, along with a longitudinal intimal tear at the anterior wall of the sinotubular junction. There was also distal displacement of the bilateral coronary ostia, and a bicuspid aortic valve exhibiting thickening, calcification, and severe stenosis. Postoperatively, the patient was transferred to the Intensive Care Unit (ICU) but developed hypotension and poor circulation, leading to death after unsuccessful reperfusion. Case 3 was a 25-year-old female who was admitted for a 2-day sudden chest distress radiating to the shoulders. No RCA ostium was identified during the operation. Aortic declamping triggered ventricular fibrillation; sinus rhythm was restored after multiple defibrillations, but unstable circulation and hypotension followed. She died despite resuscitative attempts, with an estimated blood loss of approximately 2,000 mL.

Conclusions: This article reports 3 cases of cardiac dysfunction following cardiac reperfusion due to coronary artery issues, aiming to provide insights and caution for the intraoperative management of similar patients.

Keywords: Aortic dissection; coronary artery stenosis; coronary artery malformation; case report

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Introduction

Acute type A aortic dissection (ATAAD) is a vascular condition associated with high mortality and disability rates. Patient prognosis is influenced by the severity of the affected blood vessels and organs involved in the aortic dissection. Preoperative causes of death primarily include dissection rupture, myocardial infarction caused by involvement of the coronary arteries, and cerebral hemorrhage. In contrast, postoperative mortality is often linked to organ failure. Retrograde dissection extending to the coronary artery orifice can lead to acute coronary artery insufficiency or myocardial infarction. Myocardial ischemia or infarction significantly contributes to the following cardiac dysfunction after cardiac reperfusion. This dysfunction is a critical factor in intraoperative mortality, with a notably high mortality rate once it occurs. This article summarizes the clinical data of 3 patients who died during or shortly after surgery. These cases involved aortic dissection involving the coronary arteries, preoperative concurrent coronary artery stenosis, and a congenital anomaly characterized by a single opening of the coronary artery, all of which led to cardiac dysfunction after reperfusion and ultimately resulted in death. This analysis provides valuable insights for intraoperative management. We present this article in accordance with the CARE reporting checklist (available at https://acr.amegroups.com/article/view/10.21037/acr-2025-205/rc).

Highlight box

Key findings

- The primary cause of intraoperative and early postoperative mortality in patients with acute type A aortic dissection is cardiac dysfunction after reperfusion, which is induced by coronary artery-related issues. Both pre-existing coronary artery disease and congenital coronary artery anomalies can increase the risk of intraoperative and postoperative death in these patients.

What is known and what is new?

- Acute type A aortic dissection is a dangerous disease with high mortality and disability rate, and there are many risk factors related to its death. Intraoperative myocardial ischemia or infarction leading to cardiac dysfunction after cardiac reperfusion is an important cause of intraoperative death.
- In patients with acute type A aortic dissection, coronary artery-related cardiac dysfunction after reperfusion constitutes the primary cause of intraoperative and early postoperative mortality, which clarifies the core pathological link between coronary artery anomalies and poor surgical outcomes in this patient population.

What is the implication, and what should change now?

- By detailing three typical cases, this study emphasizes the critical importance of preoperative coronary artery status for patient prognosis and puts forward corresponding warnings and strategies for intraoperative management. These findings are of great significance for improving the surgical success rate and survival rate of such patients. Future research should conduct more in-depth exploration in terms of preoperative assessment and intraoperative monitoring, so as to further reduce the perioperative mortality of patients with acute type A aortic dissection.

Case presentation

All procedures performed in this study were in accordance with the Declaration of Helsinki and its subsequent amendments. This study was approved by the ethics committee of The Second Hospital of Hebei Medical University (No. 2026-P005). Written informed consent was obtained from the patients for the publication of this case report. A copy of the written consent is available for review by the editorial office of this journal.

Case 1: aortic dissection involving the right coronary ostium

The patient was a 46-year-old male with height of 180 cm and weight of 100 kg. Diagnosis of death included (I) low cardiac output syndrome; (II) DeBakey type I aortic dissection with hypertension grade 3, extremely high risk. The primary cause of death was identified as severe low cardiac output syndrome. The patient presented to the hospital after experiencing sudden chest tightness and pain lasting 22 hours. There were no reports of radiating pain in the lower limbs, dizziness, fainting, hemoptysis, nausea, or vomiting. The patient had a 10-year history of hypertension with a maximum recorded systolic blood pressure of 190 mmHg and no diabetes, coronary heart disease, or infectious diseases. Upon examination, his temperature was 36.7 ℃, pulse was 83 beats/min, respiration rate was 21 breaths/min, and blood pressure was recorded at 159/75 mmHg. The diagnoses included type A aortic dissection and hypertension.

Initial examination

Computed tomography angiography (CTA) and 3D reconstruction of thoracic and abdominal aorta showed that the origins of the brachiocephalic artery, left common carotid artery and left subclavian artery were normal. A double lumen shadow was observed at the origins of the

innominate artery and left common carotid artery. Both the ascending aorta and aortic arch appeared widened, exhibiting true and false lumen formations in the ascending aorta, aortic arch, innominate artery and left common carotid artery; the false lumen was larger and slightly lower density. A low-density inner membrane shadow was noted, with the intimal tear located in the ascending aorta. The right vertebral artery originated from the true lumen, while the right common carotid artery displayed both true and false lumens. The distal aorta appeared normal without any double lumen shadow. The starting positions of celiac trunk, superior mesenteric artery and bilateral renal arteries were normal, and no contrast filling defect shadows were observed. The imaging diagnosis was aortic dissection, with the intimal tear extending downward to the level of the thoracic aorta. The right brachiocephalic artery, left common carotid artery, left subclavian artery and right common carotid artery exhibited both true and false lumens, while the right vertebral artery originated from the true lumen.

The maximum width of the aortic sinus and ascending aorta was about 46 mm (24 rings, 38 sinus, 46 mm). Echoes from the stripped inner membrane were detectable in the aortic arch. The interventricular septum and posterior wall of the left ventricle were thickened, however, the amplitude of motion remained within normal limits, and no segmental wall motion abnormalities were noted. The aorta showed trifoliate opening and closing, with slightly diminished valve development. An echo loss of approximately 2 mm was observed at the crown and root, while the morphology and function of the remaining membrane appeared normal. The origins of the left (LCA) and right coronary arteries (RCA) were unremarkable, and the inner diameters of the opening were about 4 mm and 4 mm, respectively.

Color Doppler flow imaging (CDFI) showed a small amount of regurgitation in the mitral and tricuspid valves during systole, as well as a minor amount of regurgitation in the aortic inlet during diastole. The ultrasound diagnosis was aortic dissection (DeBakey type I). There was no coronary ostial involvement and mild to moderate aortic insufficiency. Mild mitral and tricuspid valve insufficiencies were noted, alongside thickening of the left ventricular wall.

Preoperative examination

Preoperative laboratory results indicated hemoglobin (Hb) at 12.1 g/dL, K at 3.55 mmol/L, Ca^{2+} at 1.88 mmol/L, magnesium (Mg) at 0.53 mmol/L, and glucose (Glu) at 9.9 mmol/L.

Intraoperative findings

The ascending aorta exhibited enlargement with a diameter of 4.5 cm. There was elevated tension in the right atrium, dilation of the right ventricle with reduced activity, and evidence of myocardial swelling and cyanosis in the proximal and middle segments of the right main coronary artery. The dissection involved both the right coronary sinus and noncoronary sinus and the aortic annulus proximally. This resulted in avulsion at the junction between the right coronary sinus and noncoronary sinus, aortic valve insufficiency, complete disruption of the right coronary ostium, with no involvement of the left coronary ostium. The left coronary opening was perfused with cardioplegia, and the outflow segment of the autologous great saphenous vein was anastomosed to the coronary artery in an area exhibiting normal myocardial color. The right coronary opening was subsequently sutured. Cold cardioplegic solution was infused under direct visualization through the inflow end of the great saphenous vein to protect the myocardium of the RCA. The dissection opening was situated in the anterior wall of the middle and distal segments of the aortic arch, with approximately 60% of the aortic intima excised. A total aortic arch replacement was performed in conjunction with RCA bypass surgery. Internal drainage was established between the aortic root and the right atrial appendage. Following the resumption of cardiac activity, the patient exhibited bradycardia and hypotension. Despite the administration of dopamine, hydrocortisone, and norepinephrine, the effects of the vasoactive agents were minimal. A pacemaker was implemented; however, multiple attempts to deactivate it were unsuccessful. The blood pressure remained low. Severe low cardiac output syndrome ensued, and despite extended mechanical circulatory support and various vasoactive agents, the therapeutic response was inadequate, ultimately resulting in the patient's death. Approximately 2,000 mL of blood was lost.

Timeline

The patient presented on admission with sudden chest tightness and pain for 22 hours; underwent surgery 13 hours post-admission, and died nearly 17 hours after arriving in the operating room (16 hours from surgical start).

The patient's condition was critical and deteriorated rapidly. Although the coronary artery was not involved during the CTA at another hospital, significant aggravation of aortic root involvement occurred at the onset of the operation. The included complete disruption of the right

coronary orifice, right ventricular dilation, and right heart failure, which developed 20 hours after, despite planned coronary artery bypass grafting and myocardial perfusion protection by graft. Unfortunately, the patient ultimately experienced low cardiac output syndrome and subsequently passed away.

Case 2: aortic dissection with preoperative coronary artery stenosis

A 55-year-old male patient was admitted to the hospital with sudden chest pain that persisted for 3 hours. Diagnosis of death included (I) low cardiac output syndrome, respiratory cardiac arrest; (II) aortic dissection, Stanford type A; (III) hypertension; (IV) acute coronary syndrome. The primary cause of death was determined to be low cardiac output syndrome. Upon examination, the patient's temperature was 37.5 ℃, pulse was 109 beats/min, respiration rate was 19 beats/min, and blood pressure was recorded at 159/99 mmHg.

Initial examination

Stanford type A aortic dissection, which involved the ascending aorta, aortic arch, descending aorta, abdominal aorta, bilateral common iliac arteries, upper segments of the bilateral external iliac arteries, brachiocephalic trunk, origins of the left common carotid artery, and left subclavian artery, celiac trunk, superior mesenteric artery, and right renal artery. A 9 mm × 12 mm tear was identified in the ascending aorta. The intimal avulsion at the junction of the ascending aorta-aortic arch was annular with the false lumen encircling the true lumen. The true lumen was smaller, the false lumen was larger, the left renal artery opened in the false lumen, and the degree of the right kidney was diminished. Multiple calcifications and mixed plaques were observed in the wall of the thoracoabdominal aorta, with no significant or mild stenosis detected in the corresponding lumen. The right internal iliac artery was underdeveloped, and multiple calcifications were present in the coronary arteries. Extremely high density with artifacts noted in the region of the aortic valve. A comprehensive medical history is recommended. CTA diagnosis showed that thoracoabdominal aorta CTA and three-dimensional reconstruction confirmed the presence of aortic dissection (Stanford type A) with intimal intussusception at the ascending aorta-aortic arch junction.

Preoperative examination

Preoperative laboratory investigations revealed the following results: hs-cTnI 0.020 ng/mL; D-Dimer >12 mg/L FEU; Hb 16.1 g/dL; K 3.18 mmol/L; myoglobin 80.0 ng/mL; PT 10.8 s; Fib 1.93 g/L.

Intraoperative findings

There was a medium amount of light yellow fluid within the pericardial cavity, severe enlargement of the left ventricle, enlargement of the aortic sinus with a diameter of about 4.5 cm, and enlargement of the ascending aorta with a diameter of about 6.0 cm. The aorta was divided into true and false chambers, and with a longitudinal intimal tear located in the anterior wall of the aorta, measuring approximately 4.0 cm at the junction of the sinus and tube. The dissection extended proximally, involving non-coronary sinus and the right coronary sinus, resulting in the avulsion of both the right non-coronary and left non-coronary commissures. The left and right coronary openings were displaced distally, and the aortic valve was bicuspid, with thickened, calcified leaflets and severe stenosis. Cold cardioplegic solution was perfused under direct vision from the left and right crown openings. During the operation, a 25 mm valved conduit was used to replace the aortic valve and ascending aorta, the left and right coronary openings were transplanted to the corresponding position of the artificial vessel, a 28-mm Maquet quadrifurcated graft was used to replace the aortic arch, and a 28 mm minimally invasive stent elephant trunk was implanted in the descending aorta. After the calcification of the right coronary artery was explored, the aortic-great saphenous vein-posterior descending artery bypass was performed, and the patient still had difficulty in hemostasis. No obvious bleeding was found after re-exploration of the aortic root, and the ventilator was gradually stopped after a large amount of vasoactive drugs. Low cardiac output syndrome was considered to be the underlying cause. After surgery, the patient returned to ICU, and his blood pressure was low and his circulation was poor after returning to the room, and he died after ineffective rescue.

Timeline

The patient presented on admission with sudden chest pain for 13 hours; underwent surgery 1 hour post-admission; and died 17 hours after arriving in the operating room (17 hours 30 minutes from surgical start).

ICU examination after surgery

Postoperative laboratory investigations upon ICU admission revealed markedly elevated cardiac biomarkers and coagulation abnormalities: WBC 6.4×10^9/L; Hb 102 g/L; Plt 61×10^9/L; PT 17.5 s; INR 1.57; Fib 1.62 g/L; troponin I 41.87 ng/mL; high-sensitivity C-reactive protein 38 mg/L; myoglobin 7,267 ng/mL; creatine kinase 1,290 U/L; creatine kinase isoenzymes-MB 153 U/L; lactate dehydrogenase 774 U/L; aspartate aminotransferase 194.7 U/L.

Preoperative CTA for Case 2 indicated concomitant coronary artery calcification, and ST-T wave changes observed on two preoperative electrocardiograms suggested the potential presence of preoperative myocardial ischemia. Combined findings from the CTA and intraoperative exploration confirmed the presence of coronary artery stenosis. Intraoperatively, the aortic dissection was observed to extend proximally, involving the non-coronary sinus and the right coronary sinus, with detachment at the right coronary-non coronary commissure and the left coronary-non coronary commissure. The ostia of the left and right coronary arteries were displaced distally, further compromising myocardial perfusion. The patient ultimately succumbed to postoperative low cardiac output syndrome.

Case 3: congenital anomalous single opening of coronary artery

The patient was a 25-year-old female with height of 167 cm and weight of 67 kg. Diagnosis of death included (I) Aortic root aneurysm with aortic dissection, DeBakey type I, moderate aortic valve insufficiency. (II) The origin of the right crown was abnormal. The cause of death was identified as low cardiac output syndrome. The patient presented with sudden chest tightness radiating to the shoulder, which had persisted for 2 days prior to hospital admission. She had dizziness, fainting, nausea and vomiting, and had no history of hypertension, diabetes, coronary heart disease or infections. The temperature was 36.2 ℃, the respiratory rate was 20 breaths per minute, the pulse was 101 beats per minute, and the blood pressure was recorded at 101/74 mmHg. The final diagnosis was aortic root aneurysm with dissection and moderate aortic valve insufficiency.

Initial examinations

CTA found that the root of the ascending aorta was dilated with a transverse diameter of 6.8 cm. Heterogeneous density was observed in the aortic lumen, beginning at the aortic origin, accompanied by a linear hypodense shadow indicative of an intimal tear. The dissected intimal flap partitioned the aorta into a true lumen and a false lumen. The false lumen, larger in size, occupied the lateral aspect and exhibited a lenticular shape, whereas the true lumen was smaller and positioned medially. The distal extent of the lesion reached the proximal bifurcation of the left and right common iliac arteries, involving the brachiocephalic trunk, left common carotid artery and left subclavian artery. An intimal tear was identified at the origin of the ascending aorta. The maximum transverse diameter of the aortic arch measured approximately 3.2 cm, and the transverse diameter at the intimal tear was about 2.3 cm. The common hepatic artery, as well as the superior and inferior mesenteric arteries, and right renal artery, all originated from the true lumen of the abdominal aorta; the splenic artery and left renal artery arose from the false lumen of the abdominal aorta, resulting in reduced perfusion in the right kidney compared to the left kidney. The bilateral internal and external iliac arteries exhibited good opacification, characterized by smooth inner margins, uniform calibers and normal courses. Notable bilateral pleural thickening was observed, along with fine reticular opacities in the dorsal aspects of the bilateral lower lobes of the lungs, and scattered patchy opacities in the left lower lobe.

Aortic CTA revealed (I) aortic dissection (Stanford Type A); the splenic artery and left renal artery originated from the false lumen of the abdominal aorta, with reduced perfusion in the right kidney relative to the left kidney; (II) aneurysmal dilatation or aneurysm of the aortic root aneurysm; dependent changes in the dorsal aspects of both lower lung lobes, inflammation of the left lower lobe; bilateral pleural thickening.

Ultrasound found that left ventricular dilatation was noted, with dilatation of the aortic sinus and ascending aorta. The maximum diameter was observed at the aortic sinus, measuring approximately 64 mm (24 rings, 64 sinus, 35 ascending). An echo of the dissected intimal flap was detected in the right coronary sinus, adjacent to the ostium of the RCA. The dimensions of the remaining cardiac chambers were within normal limits, and both the thickness and motion amplitude of the interventricular septum and left ventricular posterior wall showed no abnormalities. No segmental ventricular wall motion abnormalities were identified, and the morphology and movement of all cardiac valves appeared unremarkable. A small floccular anechoic area was noted in the pericardial cavity, with a maximum depth of about 2 mm. CDFI indicated a moderate to large

amount of regurgitation at the aortic valve during diastole.

Echocardiographic findings were consistent with DeBakey Type I aortic dissection, ascending aortic aneurysm, moderate-to-severe aortic valve insufficiency, and a small pericardial effusion.

Preoperative examination

Preoperative laboratory investigations revealed the following results: troponin I 0.03 ng/mL; high-sensitivity C-reactive protein 22.8 mg/L; myoglobin 50.2 ng/mL; creatine kinase 39.0 U/L; creatine kinase-MB 7.0 U/L; lactate dehydrogenase 1,940 U/L; aspartate aminotransferase 79.1 U/L; WBC 10.5×10^9/L; Hb 66 g/L; Plt 263×10^9/L; PT 11.6 s; INR 1.04; APTT 27.7 s; Fib 3.61 g/L; pH 7.465; pCO_2 32.7 mmHg; pO_2 99.9 mmHg; K 3.7 mmol/L; Na 134 mmol/L; Glu 5.48 mmol/L; Lac 0.7 mmol/L; NT-ProBNP 2,211.73 pg/mL.

Intraoperative findings

Right femoral and right axillary intubation; Pericardial effusion, 6.5 cm in the aortic sinus, 3.5 cm in the ascending aorta. Aortic dissection was characterized by the division of the aorta into true and false lumens, with two lacerations identified. One laceration was located in the anterior wall of the ascending aorta, encompassing approximately 60% of its circumference, while the other extended from the middle of the left coronary sinus to the right non-junction, accompanied by an enlarged aortic annulus and severe aortic insufficiency. No right coronary opening was observed. Cold cardioplegic solution was perfused under direct vision via the left coronary ostium, followed by the performance of a Bentall procedure in conjunction with aortic arch replacement and elephant trunk stent implantation. After aortic unclamping, ventricular fibrillation was converted to sinus rhythm following multiple defibrillations; however, circulatory instability and low blood pressure persisted, and the administration of vasoactive drugs proved challenging. Given the inadequate coronary blood supply, aortic-great saphenous vein-left anterior descending artery-diagonal artery bypass grafting and aortic-great saphenous vein-RCA bypass grafting were undertaken. Despite restoration of perfusion, circulatory instability, low blood pressure, and arrhythmia continued, and the patient could not be weaned from the CPB. The administration of a large volume of vasoactive drugs was gradually halted due to persistent low blood pressure and bradycardia, ultimately leading to death despite resuscitation efforts. The estimated blood loss was approximately 2,000 mL.

Timeline

The patient presented on admission with sudden chest tightness and radiating pain in the shoulder for 2 days; underwent surgery 24 hours and 30 minutes post-admission, and died 16 hours and 50 minutes after arriving in the operating room (16 hours and 50 minutes from surgical start).

Case 3 was a single ostium anomaly with coronary artery involvement. Congenital anomalies of the coronary arteries can result in abnormal coronary blood supply during or after surgery. When both the LCA and RCA share the same ostium, the risk of myocardial infarction and global ischemia increases significantly. Although single coronary artery is a very rare anomaly, recent years have seen several reports of successful treatment for this condition in conjunction with Stanford type A acute aortic dissection (AAD) (1-4).

Discussion

Post-aortic unclamping cardiac dysfunction represents a significant complication during total aortic arch replacement. This condition is primarily characterized by challenges in restoring cardiac rhythm, often necessitating multiple electrical defibrillations or the recurrence of ventricular fibrillation shortly after the heart resumes beating. It is frequently caused by severe hypotension, which may require substantial doses of inotropic agents to sustain circulation or, in some cases, the initiation of extracorporeal membrane oxygenation (ECMO) support. Cardiac dysfunction resulting from myocardial ischemia or infarction should be considered only after excluding acid-base disorders, electrolyte imbalances and volume depletion. The majority of patients experience mortality during or shortly after the procedure.

Preoperative coronary hypoperfusion caused by retrograde aortic dissection and preoperative coronary stenosis constitutes the primary contributors to intraoperative cardiac dysfunction. Severe CAD and AAD that involve the coronary arteries lead to obstruction of coronary blood flow while hemodynamic instability further diminishes coronary perfusion pressure for a period following cardiac arrest. At the same time, an elevated heart rate increases myocardial oxygen consumption, which can result in damage and necrosis of the ischemic myocardium. In addition, patients with congenital anomalies such as abnormal coronary artery origins, narrowed coronary ostia, and single coronary arteries face an elevated risk of dissection involvement and subsequent cardiac dysfunction. In

individuals with normal heart function and rhythm, the onset of sudden arrhythmias, hypotension, electrocardiographic signs of myocardial ischemia or infarction, insensitivity or poor tolerance to volume expansion, and diminished cardiac contractility observed during hemostasis after CPB should prompt consideration of coronary stenosis or obstruction. In such cases, active treatment measures must be implemented, and if necessary, CPB should be reestablished to investigate the underlying cause.

Case 1 exhibited type C coronary artery involvement. In this patient, elevated myocardial enzyme levels and ischemic or infarction-related changes were observed on the electrocardiogram prior to surgery. Following thoracotomy, the contraction of ischemic region of the heart was either weakened or absent, and the myocardium displayed signs of ischemia. Examination after aortic root dissection confirmed significant coronary involvement, characterized by compression or avulsion. Clinically, it has been noted that such patients frequently present with right ventricular contraction disorders following opening the pericardium, right ventricular distension after resumption of heartbeat, or poor tolerance to volume increases. Additionally, an increase in preload initially manifests as cardiac arrhythmia, which is eliminated upon reduction of preload.

Coronary involvement is characterized by acute myocardial ischemia and infarction resulting from the extension of AAD into the coronary ostium or any disruption of coronary flow due to dissection (5,6). The types of coronary artery involvement are classified as type A, which involves coronary ostial dissection; type B, characterized by false coronary artery dissection; and type C, which entails circumferential coronary artery detachment. Type A represents a coronary occlusion caused by compression from an anatomical false lumen bulge or by secondary extravasation of blood into the pericardium or perivascular tissue. Type B refers to the retrograde extension of the coronary artery wall dissection, where occlusion results from the compression of the false lumen within the enlarged coronary artery. Type C is the most severe form of coronary artery dissection, wherein the coronary artery is separated from the aortic root, leading to direct occlusion and malperfusion. Clinically observed type C lesions are predominantly in the RCA, as the RCA originates from the right anterior aspect of the ascending aorta, with the coronary artery orifice typically located below the sinus-tube junction. When the aortic root undergoes retrograde dissection towards the coronary artery orifice, the RCA is usually the first to be affected. Some patients may also exhibit varying degrees of left coronary involvement (7). It is uncommon for patients undergoing surgical repair of aortic dissection to present with bilateral coronary artery involvement (8).

Coronary artery involvement significantly elevates morbidity and mortality, with a mortality rate of 1–2% per hour following symptom onset. Some patients may succumb at the time of onset due to myocardial infarction resulting from dissection affecting the coronary arteries (9). Even when coronary artery repair is undertaken, the intraoperative and postoperative mortality rates for these patients are markedly higher than those for individuals without coronary artery involvement, with in-hospital mortality exceeding 40% (10). For patients experiencing preoperative myocardial infarction, intraoperative myocardial protection through ischemia or infarction becomes increasingly critical. Assessing the extent of myocardial infarction during CPB presents challenges, as hemodynamic changes and alterations in myocardial motion, such as weakening or absence, may not manifest until cardiac perfusion is restored. Consequently, maintaining myocardial perfusion and achieving a balance between myocardial oxygen supply and demand may prove more difficult once cardiac activity resumes in these patients.

Case 2 presented with coronary artery stenosis prior to surgery. The age of onset for coronary heart disease is progressively decreasing, with the prevalence of chronic CAD among patients experiencing acute type A dissection ranging from 8% to 41%. The presence of concurrent CAD significantly elevates the risk of adverse outcomes following surgical intervention and endovascular aortic repair, thereby increasing the likelihood of intraoperative or postoperative myocardial infarction (11,12).

Case 3 presented a single ostium anomaly with involvement of the coronary artery. Congenital anomalies of the coronary artery can result in abnormal coronary blood supply during or following surgical procedures. When both the LCA and RCA share the same ostium, the risk of myocardial infarction and global ischemia significantly increases. A single coronary artery represents a rare anatomical variation. Notably, emergency coronary artery bypass surgery has been successfully conducted in case of single coronary artery associated with Stanford type A AAD (13).

The incidence of congenital coronary aortic anomalies in adults is 0.27%, with RCA being the most frequently affected (14). Intraoperative findings warrant careful consideration. When such anomalies arise during surgery,

they are typically associated with severe malperfusion of the left or right ventricle, which can lead to mechanical failure. This situation often results in an inability to wean the patient from CPB and may culminate in mortality (15,16).

All three patients exhibited signs of arrhythmia and/or myocardial ischemia during the weaning process from CPB, characterized by diminished or absent myocardial motion, poor volume tolerance, challenges in achieving cessation of support, and ultimately, death. Regardless of the underlying cause of cardiac dysfunction, the primary therapeutic measures involve repairing the compromised aortic root structure and restoring coronary blood flow to mitigate myocardial ischemic injury (17,18). Surgical interventions encompass coronary artery reconstruction or local repair, coronary artery bypass grafting, and preoperative percutaneous coronary intervention (PCI) (19). Coronary artery bypass grafting (CABG) serves as a prevalent treatment for type C coronary artery dissection, significantly enhancing the patient's condition. For patients who undergo CABG achieve hospitalization survival, their subsequent conditional survival rates closely resemble those of individuals without coronary malperfusion (20). CABG poses substantial risks for patients with coronary artery involvement and preoperative coronary artery stenosis (21). This procedure may induce retrograde blood flow to the aorta at the anastomosis site of the coronary artery, leading to diminished or absent perfusion of the distal myocardium. This does not alleviate myocardial ischemia or infarcted myocardium, while increased myocardial oxygen consumption following resumption of cardiac activity further exacerbates myocardial infarction. Identifying the site of coronary artery stenosis during surgery presents a significant challenge. Prophylactic (CABG) is not recommended unless distinct features, such as epicardial color changes or localized ventricular wall motion abnormalities, can clearly indicate the target location for the procedure (22).

Some studies have indicated that CABG may be associated with an increased risk of postoperative mortality (23), suggesting that myocardial infarction can still occur in certain patients following the procedure. However, CABG serves as an effective treatment that enhances the prognosis for patients with CAD. The mortality of patients after CABG should be primarily attributed to the failure of coronary recanalization or the inability of CABG to improve myocardial perfusion in case of pre-existing myocardial ischemia or infarction, rather than to CABG itself being a risk factor for death.

Angiography serves as the gold standard for assessing coronary artery anatomy and differentiating acute CAD resulting from involvement of the coronary ostia from chronic CAD. The assessment of coronary artery anatomy does not influence the survival rates of patients with aortic dissection (24). The poor distribution of the contrast agent at the distal end of coronary artery ostium following compression or transection, leads to ambiguous development of the unclear coronary artery tree. Furthermore, angiography prolongs the time to surgical intervention and heightens mortality caused by aortic rupture (25). Preoperative coronary angiography is rendered a non-essential and selective examination, while intraoperative coronary angiography to evaluate coronary recanalization should be routinely employed during surgery for these patients.

The retrospective design of this case series inherently introduces selection bias; specifically, all cases included were fatal, which may not represent the full spectrum of disease, and limits the generalizability of our findings to non-fatal cases. The small number of cases limits our ability to draw generalizable conclusions or identify broader risk patterns. Incomplete preoperative clinical data (e.g., baseline inflammatory markers, functional status scores) for certain cases may have compromised the thoroughness of our outcome assessment. As a single-center study, our results may be influenced by the institutional treatment protocols and characteristics of the patient population, which may not be applicable to other medical centers.

Conclusions

This article delineates three primary causes of cardiac dysfunction in patients undergoing total arch replacement, aiming to inform intraoperative management and reduce patient mortality. The predominant factor contributing to intraoperative and early postoperative mortality in individuals with ATAAD is cardiac dysfunction after cardiac reperfusion, which is often precipitated by abnormalities related to the coronary arteries. Both pre-existing CAD and congenital coronary artery anomalies significantly increase the risk of intraoperative and postoperative death in these patients. Through the presentation of three illustrative clinical cases, this article underscores the critical importance of preoperative coronary artery assessment in predicting patient prognosis, and provides practical precautions and strategies for optimizing intraoperative decision-making. These findings are of great value for improving the surgical success rate and survival rate of such patients.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://acr.amegroups.com/article/view/10.21037/acr-2025-205/rc

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the Declaration of Helsinki and its subsequent amendments. This study was approved by the ethics committee of the Second Hospital of Hebei Medical University (No. 2026-P005). Written informed consent was obtained from the patients for the publication of this case report. A copy of the written consent is available for review by the editorial office of this journal.

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