# A Case of Stanford Type A Acute Aortic Dissection With Preoperative Extensive Cerebral Infarction Successfully Treated Surgically

**Shun Hiraga, MD**, Tomoaki Hirose, PhD[1], Ryohei Fukuba, MD[1], Junichi Takemura, MD[1], Rei Tonomura, MD[1], Sayaka Tamada, MD[1], Kazuhiro MitanI, MD[1], Shinya Yokoyama, PhD[1]

[1]Department of Thoracic and Cardiovascular Surgery, Nara Medical University School of Medicine, Kashihara, Japan

## ABSTRACT

Patients with organ malperfusion from acute aortic dissection (AAD) have poor outcomes, and the surgical indications for patients with AAD complicated by extensive cerebral infarction have not been established. Here, we report a successfully treated surgical case of a patient with cerebral infarction and Stanford type A, AAD. A 77-year-old man was admitted to the hospital with a chief complaint of left paresis. After confirming that there was no cerebral hemorrhage with a head computed tomography and an incision in the right neck, and the right internal carotid artery was ligated and closed, emergency surgery was performed with a 24 mm Triplex® raft. The ascending aorta was replaced, and a bypass was performed with a prosthetic graft from the right axillary artery. No cerebral hemorrhage or neurological issues were observed postoperatively, which indicates the possibility of surgical intervention as a treatment strategy for this disease.

## INTRODUCTION

The incidence rate of acute aortic dissections (AADs) is 2.5 per 100,000 person-years, and 66% of those AADs are classified as Stanford type A (Melvinsdorittir 2016). Among AADs, treatment outcomes of patients with organ malperfusion due to dissection before surgery is poor and surgical indications for patients with extensive cerebral infarction have not been established. We report a patient's case of AAD that was complicated by an extensive cerebral infarction. The surgery was performed in the acute phase of the infarction, and a positive outcome was observed.

## CASE REPORT

A 77-year-old man who had a left retinal detachment surgery performed 2 years ago was referred to our hospital. He fell at home and had difficulty walking. He visited a nearby doctor the day after his fall with a chief complaint of left paresis. The cerebral magnetic resonance imaging (MRI) illustrated a right internal carotid artery obstruction, and the right cerebral hemisphere displayed an extensive acute phase infarction. Thoracic and abdominal contrast-enhanced computed tomography (CT) was used to demonstrate the characteristics of Stanford type A and AAD (AAAD) and a right internal carotid artery obstruction due to a dissection (Figure 1). Upon immediate admission to our hospital, the patient was 145 cm tall and weighed 42 kg. On presentation,
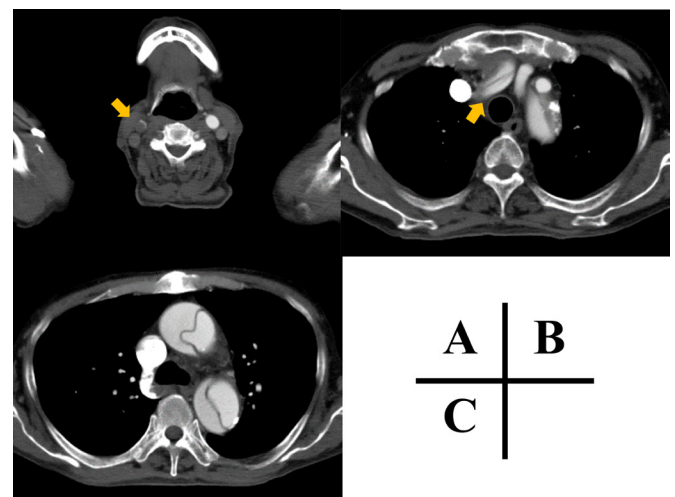
Figure 1. (A) Contrast-enhanced computed tomography shows the right internal carotid artery obstruction due to an aortic dissection. (B) An aortic dissection extends to the brachiocephalic artery. (C) An aortic dissection exists from the ascending aorta to the abdominal aorta.
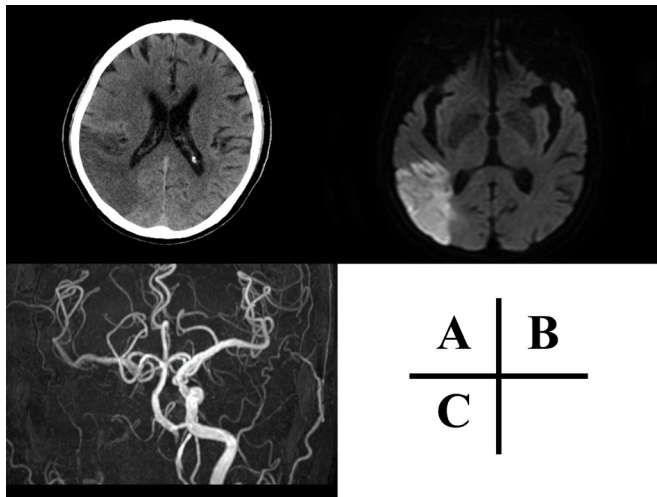
Figure 2. (A) The cerebral computed tomography shows an extensive acute phase ischemic stroke in the right cerebral hemisphere. (B) The cerebral magnetic resonance imaging shows (a) high signal in the right middle cerebral artery region in the diffusion-weighted image. (C) The magnetic resonance angiography shows a right internal carotid artery occlusion and a slight collateral network through the circle of Willis.
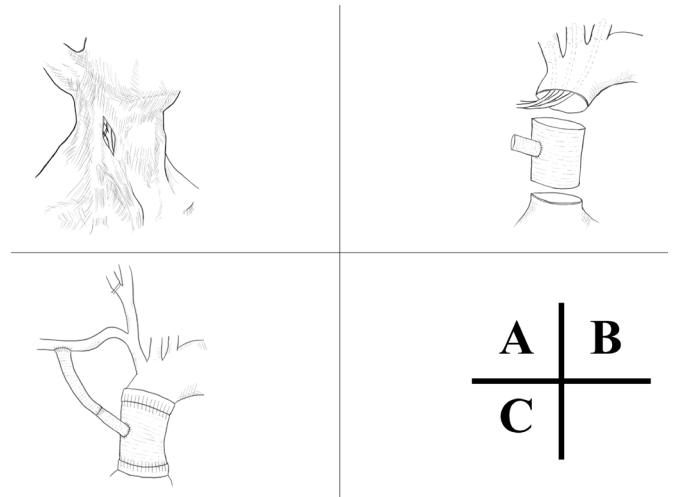


Figure 3. (A) A skin incision was made in the right neck, and the right internal carotid artery was doubly ligated with 2-0 silk. (B) Catheters were inserted into the cervical branches to perform selective cerebral perfusion. A 24-mm Triplex® was selected as the artificial blood vessel, and an 8-mm Gelsoft® was anastomosed for the right axillary artery bypass. (C) An 8-mm Gelsoft® was anastomosed for the right axillary artery. A graft was guided into the mediastinum, where the Gelsoft® was anastomosed to the Triplex®.
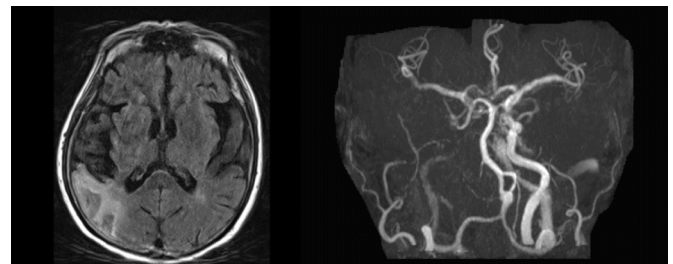


Figure 4. The magnetic resonance imaging performed 22 days after surgery shows no expansion of the cerebral infarction area, and cerebral arteries had collateral circulation.

the patient's vital signs included a blood pressure of 124/62 mmHg on his left upper limb, systolic blood pressure in the 60 mmHg-range on his right upper limb, and lateral blood pressure of his upper limbs. His heart rate was 79 beats per minute, and his right common carotid artery was difficult to palpate. The carotid echography revealed severe stenosis in his right common carotid artery and occlusion of his right internal carotid. He showed symptoms of left hemispatial neglect, mild left facial paralysis, and articulation disorder. He had pain in his right upper limb, and neurological evaluation was difficult to perform. The peripheral blood examination showed a high white blood cell count (11900 per µL), low hemoglobin count (11.2 g per dL), and normal platelet count ($20.9 \times 10^4$ per µL). High prothrombin time, an international normalized ratio of 1.19, high fibrinogen levels (586 mg per dL), high fibrin/fibrinogen degradation products (25.7 µg per dL), and low D-dimer levels (9.8 µg per dL) were all observed in the blood coagulation test. No notable findings were found in the biochemical tests. No renal dysfunction was observed as blood urea nitrogen (16 mg per dL) and creatine levels (0.61 mg per dL) were within the normal range.

Abdominal branches, including the celiac, superior mesenteric, and right renal artery, showed perfusion through the true lumen, while the left renal artery had perfusion through the false lumen. Transthoracic echocardiography showed no cardiac effusion or trivial aortic regurgitation. A decrease in cardiac contractility was also not observed. The cerebral CT showed an extensive acute phase ischemic stroke in the right cerebral hemisphere (Figure 2A). No hemorrhagic lesion was observed. The cerebral MRI showed a high signal in the right middle cerebral artery region with the diffusion-weighted image (Figure 2B). Blood flow was impeded in the right internal carotid artery and was supplemented through the circle of Willis in the right middle cerebral artery, but the signal was faint (Figure 2C). This suggests the possibility of a relative decrease in blood flow.

AAAD was suitable for emergency surgery, but there was a risk of cerebral hemorrhage due to anticoagulant therapy associated with extracorporeal circulation in acute phase surgery with acute ischemic stroke. However, the patient was unable to perform essential activities of daily living after the fall. Therefore, if conservative treatment was selected for AAD, long-term rest would result in irreversible symptom fixation. After intense discussions, a cerebral CT was performed the next day, and surgery was immediately performed after confirming the absence of lesions. The patient was under general anesthesia during the operation. A skin incision was made in the right neck. The right internal carotid artery was exposed and doubly ligated with 2-0 silk (Figure 3A). Subsequently, a 28 Fr cannula and an 18 Fr cannula were inserted into the superior vena cava and the ascending aorta using the Seldinger method, respectively, and the pump was

started. A 36 Fr cannula was inserted into the inferior vena cava. A venting catheter was placed in the left ventricle from the right superior pulmonary vein, and core cooling started. After the superior and inferior vena cava were secured with tape and a tourniquet and shifted to complete extracorporeal circulation, the right atrium was incised, and a retrograde myocardial protective catheter was inserted into the coronary sinus under direct vision. The right atrium was closed with a 4-0 polypropylene, and the circulation was arrested at a pharyngeal temperature of 25°C. The ascending aorta was incised, and the primary entry tear was confirmed in the ascending aorta. After inserting catheters into the cervical branches, selective cerebral perfusion of a flow rate of 10 mL/kg was started. The left ventricular outflow tract diameter was measured with a bougie, and a 24 mm Triplex® was selected as the artificial blood vessel.

On the back table, an 8 mm Gelsoft® was anastomosed to the 24 mm Triplex® with 6-0 polypropylene for the right axillary artery bypass. A 12 mm wide felt strip was applied to the outer circumference of the peripheral aorta, fixed with 3-0 Ethibond needles, and then anastomosed to the 24 mm Triplex® with 4-0 Polypropylene continuous suture. The lower body perfusion resumed from the side branch of the 24 mm Triplex®. The ascending aorta was transected 15 mm on the peripheral side of the sinotubular junction, and a Bioglue® was injected into the pseudolumen and adhered. Ten-millimeter width felt strips were applied on the inner and outer circumference of the proximal aortic wall, and a proximal anastomosis was performed with a 4-0 Polypropylene horizontal mattress. Terminal warm blood cardioplegia was injected through a retrograde myocardial protective catheter. Self-heartbeat resumed regularly. A skin incision was made on the lateral side of the right clavicle to expose the right axillary artery. After the arterial clamp, the right axillary artery was incised, and the intima was dissected. The intima was fixed with a 7-0 Polypropylene horizontal. An 8 mm Gelsoft® was anastomosed with a 7-0 Polypropylene continuous suture, and the graft was guided into mediastinum where the Gelsoft® was anastomosed with the Triplex® with a 6-0 Polypropylene continuous suture (Figure 3C). The duration of the entire operation was 480 minutes, the total extracorporeal circulation was 159 minutes, the cardiac arrest was 100 minutes, and the selective cerebral perfusion was 49 minutes. The postoperative course was uneventful.

The patient regained consciousness a day after the surgery, and no exacerbation of neurological abnormalities was observed. A cerebral CT was performed 4 days after surgery. No intracranial hemorrhagic lesion was observed, and the extent of the cerebral infarction was not expanded. Extubation was performed 6 days after the surgery. A cerebral MRI was performed 22 days after the surgery. No expansion of the cerebral infarction area was observed. The right middle and posterior cerebral arteries had collateral circulation (Figure 4). The patient was rehabilitated in another hospital 31 days after surgery. At the time of the transfer, his left facial nerve palsy and dysarthria improved, and his manual muscle test score was 5 for both his upper limbs. The patient could walk and was discharged from the hospital 50 days after the

transfer. Eight months post-surgery, he is currently visiting our outpatient department, but his daily life is independent, and no neurological deficits were observed.

## DISCUSSION

Surgical outcomes for AAAD have improved over the recent years but remain fatal. The incidence rate of AAD is 2.5 / 100,000 person years, of which 66% are Stanford type A. 17.6% of them die before arriving at the hospital, 21.4% die within 24 hours, and 45.2% die within 30 days (Melvinsdorittir 2016). Neurological complication accounts for 17% to 40% of AADs; however, half of them had an infarction in the brain or spinal cord (Charly 2007). A report states that postoperative complications, such as hypotension, coma, and malperfusion, are common in patients with preoperative cerebral infarction. However, the prognosis after discharge did not worsen (Eusanio 2013). According to a study using the International Registry of Acute Aortic Dissection (IRAD) data, surgical indication seemed to be affected by preoperative complications of cerebral nerve damage (Eusanio 2013). Surgical mortality was 11% in cases without cerebral nerve damage, 24.1% in cases with cerebrovascular events, and 33.3% in cases with preoperative coma (Eusanio 2013). It is difficult to decide on surgical treatment for AAD with preoperative widespread cerebral infarction, as reported in this case study.

In a comparative study of treatments for AAAD with preoperative neurological deficit, the conservative treatment showed poor outcomes. The in-hospital mortality rate of a conservative-treated group with a preoperative cerebrovascular accident was 76.2%, and preoperative coma was 100% (Eusanio 2013). In comparison, the in-hospital mortality rate of the surgically treated group with a preoperative cerebrovascular accident was 49.6%, and for patients who were in a coma was 55.6% (Eusanio 2013). Through logistic regression analysis, surgical treatment was an improving factor for the mortality rate of patients with preoperative neurological deficits. The 5-year survival rate in the group receiving conservative treatment for patients with preoperative cranial nerve damage and coma was 23.8% and 00.0%, respectively. At the same time, those in the surgically treated group with said damage and coma were 67.1% and 57.1%, respectively. Thus, the surgically treated group had an improved mortality rate after 5 years compared to the conservative treatment group (Eusanio 2013). Thus, it is likely that the patient's prognosis will improve through surgical intervention.

The greatest concern when performing the surgical treatment in the acute phase for AAD is hemorrhagic complications due to systemic heparinization associated with extracorporeal circulation. A report states that 6 out of the 7 patients with AAAD complicated by preoperative cerebral infarction died from postoperative cerebral infarction (Cambria 1988). However, another study showed that, among 14 cases of AAAD with preoperative cerebral infarction, the in-hospital mortality rate was 7.0%, and remission of neurologic status was observed in 14%, improved in 43%, unchanged in 43%.

No exacerbations were observed (Estrera 2006). In another study, 27 patients with AAAD who had preoperative coma showed an in-hospital mortality rate of 14% and complete remission of consciousness in 86% when surgical treatment was performed in the acute phase (Tsukube 2011). Furthermore, 52% of the patients showed independence in their daily life 3 years post-surgery (Tsukube 2011). Using IRAD data, a third research team reported that of the patients with AAAD presenting preoperative cerebral nerve damage that underwent surgical treatment, consciousness improved in 80.4% of the ones with a preoperative cerebral infarction and 74.2% of the ones with a preoperative coma (Eusanio 2013). In recent years, only 6% to 7% of patients with acute cerebral infarction had cerebral hemorrhage after thrombectomy. A previous clinical trial concluded that no significant difference in the conservative treatment group persisted (Albers 2018, Nogueria 2018). Furthermore, recent reports showed that prolonging the golden time in a thrombectomy for an acute cerebral infarction and an acute cerebral infarction caused by AAD may improve patients' neurological outcomes (Albers 2018, Nogueria 2018).

Previous studies examined the time between symptom onset of cerebral infarction to surgery and stated that patients with greater than 10 hours of symptom onset might have poor outcomes (Estrera 2006, Tsukube 2011). Moreover, endovascular treatment for acute cerebral infarction 7.3 hours after the onset of cerebral infarction was reported to have a poor outcome (Saver 2016). However, a study examining 50 patients with AAD, who had a preoperative cerebral infarction, showed no association between surgery time and neurological outcome (Chiu 2019). In our case, more than 24 hours passed from the onset of cerebral infarction to the start of surgery; however, no postoperative bleeding complications were observed. Improvement of the patient's neurological symptoms was observed at discharge. We believe the elapsed time from the onset of infarction has little effect on the judgment of the surgical indication, except for cases with preoperative cerebral bleeding where we have to perform surgical treatment for patients with AAAD who have preoperative cerebral neuropathy. However, in most cases of AAAD, a preoperative coma is not indicated for surgery after consultation with the patient's family.

In our case, we performed a right internal carotid artery closure to avoid cerebral hemorrhage from revascularization in the cerebral infarction area. Still, as far as we could investigate, there are no similar case reports. Cerebral hemorrhage can be avoided if the blood flow in the true lumen could be improved via the carotid artery without a right internal carotid artery closure and a right axillary artery bypass. However, the time for the procedure was short, and we believe that our patient's condition was suitable for an intraoperative concomitant procedure.

## CONCLUSION

We reported a surgical case for AAAD with a right internal carotid artery occlusion due to preoperative dissection and extensive cerebral infarction. A right internal carotid artery closure and a right axillary artery bypass were performed in the acute phase. A positive outcome was observed, indicating the possibility of surgical intervention as a treatment strategy for AAAD complicated by cerebral infarction.

## REFERENCES

Albers GW, Marks MP, Kemp S, et al. 2018. Thrombectomy for stroke at 6 to 16 hours with selection by perfusion imaging. N Eng J Med 378:708-18.

Cambria RP, Brewster DC, Gertler J, et al. 1988. Vascular complications associated with spontaneous aortic dissection. J Vasc Surg 7:199-209.

Charly G, Dietrich W, Friedrich I, et al. 2007. Neurological symptoms in type A aortic dissections. Stroke 38:292-7.

Chiu P, Rotto TJ, Goldstone AB, et al. 2019. Time-to-operation does not predict outcome in acute type A aortic dissection complicated by neurologic injury at presentation. J Thorac Cardiovasc Surg 158:665-72.

Estrera AL, Garami Z, Miller CC, et al. 2006. Acute type A aortic dissection complicated by stroke: can immediate repair be performed safely? J Thorac Cardiovasc Surg 132:1404-8.

Eusanio MD, Patel HJ, Nienaber CA, et al. 2013. Patients with type A acute aortic dissection presenting with major brain injury: Should we operate on them? J Thorac Cardiovasc Surg 145:S213-22.e.1.

Melvinsdottir IH, Lund SH, Agnarsson BA, et al. 2016. The incidence and mortality of acute thoracic aortic dissection: results from a whole nation study. Eur J Cardiothorac Surg 50:1111-7.

Nogueria RG, Jadhav AP, Haussen DC, et al. 2018. Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct. N Eng J Med 378:11-21.

Saver JL, Goyal M, van der Lugt A, et al. 2016. Time to treatment with endovascular thrombectomy and outcomes from ischemic stroke: a meta-analysis. JAMA 316:1279-88.

Tsukube T, Hayashi T, Kawahira T, et al. 2011. Neurological outcomes after immediate aortic repair for acute type A aortic dissection complicated by coma. Circulation 124:S163-7.