

CASE REPORT

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Abrupt left coronary artery malperfusion secondary to acute type A aortic dissection after weaning from cardiopulmonary bypass: a case report

Myungsoo Jang^{1*}, Sang Beom Nam¹, Youn Jin Kim¹ and Suk-Won Song²

Abstract

Introduction Acute Stanford type A aortic dissection (ATAAD) is a lethal emergency. However, even with instant surgical repair, early mortality is up to 20%. ATAAD complicated by coronary artery involvement is considered rare but life-threatening because this can cause coronary artery malperfusion which results in acute myocardial infarction. In particular, left coronary artery malperfusion can bring worse outcomes than right coronary artery malperfusion, but there are few reports of left coronary artery involvement secondary to ATAAD.

Case presentation We present a case of a woman who got emergency open heart surgery due to ATAAD. After the hemiarch replacement, the first weaning from bypass was relatively smooth. However, as soon as starting infusion protamine, we found out sudden regional wall motion abnormality at the diffuse anteroapical to the lateral wall on echocardiography and ST depression on leads II and V5 electrocardiogram after several ventricular fibrillation. We recognized by echocardiography that intimal dissection flap extended to the left coronary artery ostium and dynamically obstructed left coronary artery blood flow, because the true lumen collapsed dynamically during the diastolic phase. Upon re-establishing bypass, proximal aortic false lumen was obliterated with BioGlue again. Smooth weaning from bypass proceeded at last. Finally, the blood flow to the left coronary artery ostium was good, and the wall motion abnormality was improved.

Conclusion Our report suggests the importance of the degree of myocardial damage caused by coronary artery malperfusion which is a major predictor of patient outcome. To reduce complications and minimize the mortality rate, an instant treatment plan is needed. However, limited options for exact surgical treatment directions or guidelines for coronary artery malperfusion secondary to ATAAD are available so far. We emphasize that we should not neglect any signs indicative of coronary artery malperfusion appear such as changes of electrocardiogram and echocardiography. Moreover, our report contributes to a profound understanding among clinicians regarding the necessity of practical treatment guidelines about coronary artery malperfusion due to ATAAD based on various surgical experiences and studies.

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Keywords Acute stanford type A aortic dissection, Coronary artery involvement, Coronary artery malperfusion, Left coronary artery malperfusion

Background

Acute Stanford type A aortic dissection (ATAAD) is a lethal emergency [1, 2]. According to statistical reports, the early mortality rate increases 1% per hour after the symptom onset of abrupt incisive chest pain spreads to the back [3]. The incidence of ATAAD, for which hypertension is well known to be a common risk factor, is estimated to be 5 to 30 cases per million people per year [1, 3]. Emergency open repair surgery is required for ATAAD, but even with surgical repair, early mortality is up to 20% [4]. Males are more commonly affected than females, and it occurs mainly in people aged 50–70 years, although patients with Marfan syndrome, a bicuspid aortic valve, or Loeys-Dietz syndrome can present at younger ages [5, 6]. ATAAD complicated by coronary artery involvement (CAI) is considered rare but life-threatening. This is because the intimal dissection flap extends to right or left coronary artery and can cause coronary malperfusion (CM) by either static or dynamic obstruction of coronary blood flow, which leading to acute myocardial infarction (MI) and a poor prognosis [7–9]. In this respect, rapid diagnosis and optimal treatment of CM is vital to survival. We report a dangerous experience for a 75-year-old female with a sudden obstruction of the left coronary artery (LCA) ostium due to the intimal flap immediately after ATAAD open repair surgery.

Case presentation

A 75-year-old woman was taken to a hospital with a chief complain of a sudden stuporous mentality. Because she showed left upper side weakness, she underwent brain computed tomography (CT), which revealed right carotid artery dissection secondary to ATAAD (Fig. 1).

Because the right innominate artery collapsed due to dissection, which resulted in right brain hemisphere malperfusion, she was immediately transferred to our hospital by helicopter right after taking a triphasic contrast enhanced aorta CT (Fig. 2) for emergency surgery.

Her medical history included hypertension, diabetes, and an infrarenal abdominal aortic aneurysm of 3.2 cm. Her initial vital signs when she arrived in our emergency department were a blood pressure of 134/52 mmHg, pulse rate of 51 beat per minute, temperature of 35.5 °C, and respiratory rate of 20 breaths per minute. The initial laboratory results were hemoglobin of 11.1 g/dL, platelets of 148,000/μL, fibrinogen of 85.8 mg/dL, and coagulation within normal limits. Her renal function test and liver function test results were also normal. Her initial eletrocardiogram (ECG) revealed a normal sinus rhythm,

and cardiac markers such as creatine kinase, creatine kinase-MB, and troponin T were in the normal ranges. There was no specific finding in chest radiology except mild pulmonary congestion. She was taken to the operating room immediately after obtaining informed consent from her spouse. Both radial arterial cannulations with five lead ECG monitoring were followed by general anesthesia. Pre-bypass transesophageal echocardiography (TEE) showed an intimal dissection flap just above the right coronary artery (RCA) ostium but good blood flow to both coronary ostia in the mid-esophageal aortic short and long axis views, without prominent pericardial effusions (Video 1, 2). In addition, left ventricular contractility and valvular functions were judged to be normal including trivial aortic regurgitation. A Hemashield Platinum (8 mm diameter) was grafted to the right axillary artery to establish a cardiopulmonary bypass (CPB) pump. After that, the median sternotomy was performed, and her pericardium was opened. Her right atrium cannulation was followed by starting the CPB pump, and 15-minute systemic cooling was done. The ascending aorta was cross clamped proximal to the right innominate artery, and surgeon transected the aortic root above the sinotubular junction to infuse cardioplegia solution directly through the coronary ostium and completely stop her heart. We recognized through the operation field that the primary entry tear was just above the RCA ostium and extended to the ascending aorta. Surgeon checked the both coronary artery ostia and they were not involved by the dissection flap. For proximal arch replacement, the hypothermic circulatory arrest with unilateral antegrade selective cerebral perfusion was done for 23 min at 29.8 °C. She received a hemiarch replacement with a Vascutek 30 graft. Distal anastomosis was zone 0, just before the right innominate artery, and proximal anastomosis was sinotubular junction. As rewarming was finished, gradual weaning from CPB was proceeded. Total aortic cross clamping time was 46 min, and whole pump time was 128 min. There was no arrhythmia or other adverse complications until protamine infusion was started. At that time, sudden ventricular fibrillation occurred, and the surgeon immediately defibrillated several times. We promptly discovered a new regional wall motion abnormality (RWMA) at the diffuse anteroapical to the lateral wall in the TEE (Video 3), and the ECG monitor showed deep ST depression at leads II and V5. To treat her cardiogenic shock, a dobutamine infusion (5 mcg/kg/min) was added to a norepinephrine infusion (0.1 mcg/kg/min). During our investigation to discover the cause of cardiogenic shock, we carefully observed the TEE and

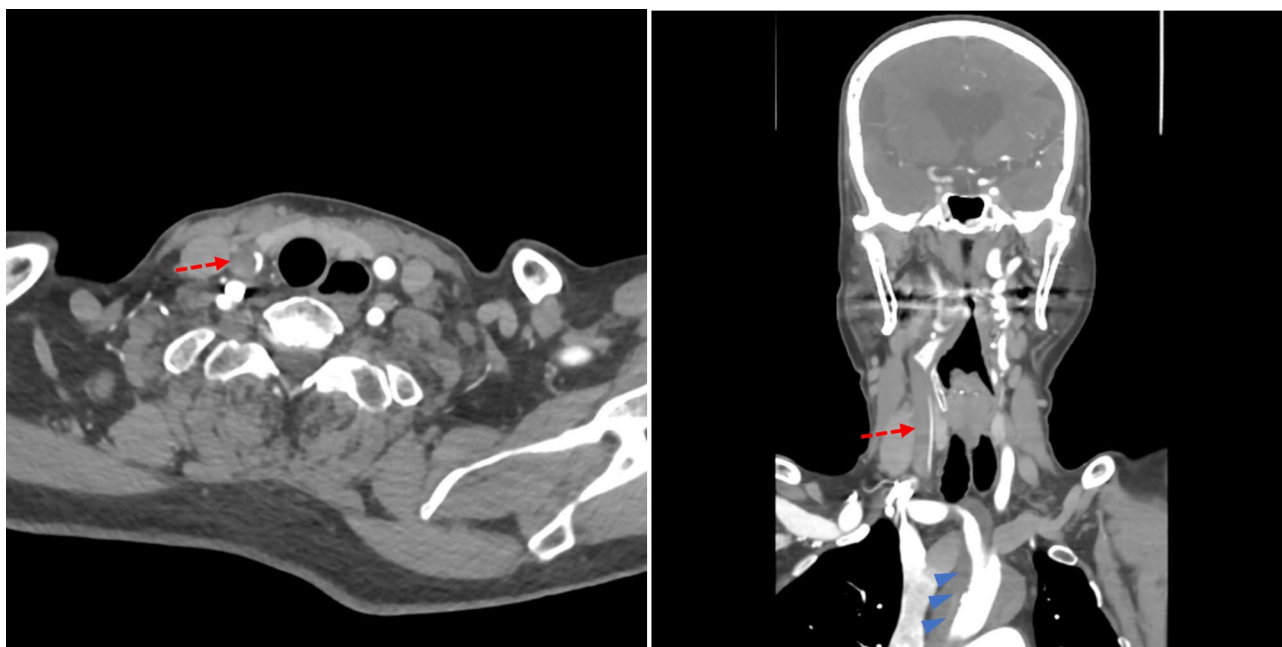


Fig. 1 Brain CT angiography. **1a** and **1b** show collapse (red arrow) of the right innominate artery and the right carotid artery. This caused the patient's mental change to stupor with left weakness due to the brain malperfusion. Also, a coronal view (**1b**) presents ATAAD (blue arrowheads)

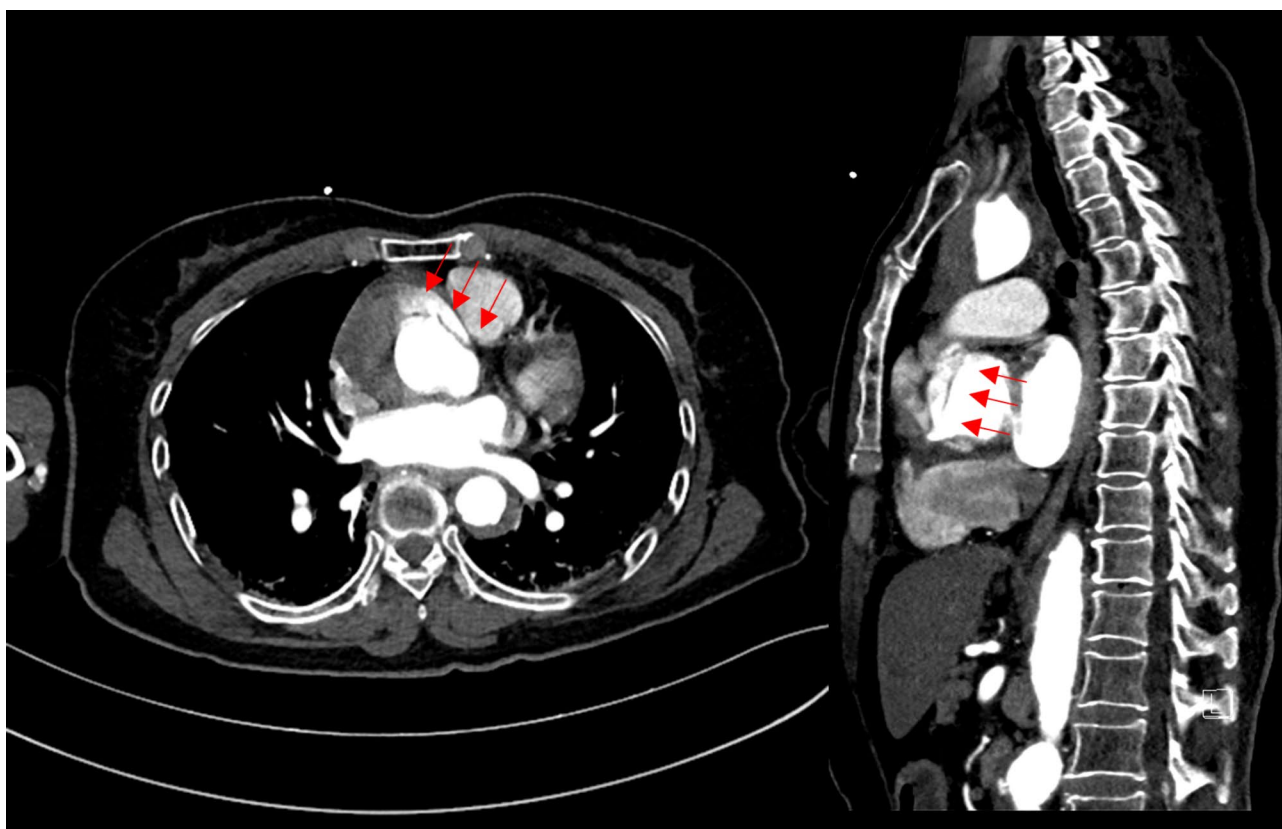


Fig. 2 Axial and sagittal views of aorta CT indicate the dissection flap in ascending aorta (red arrows)

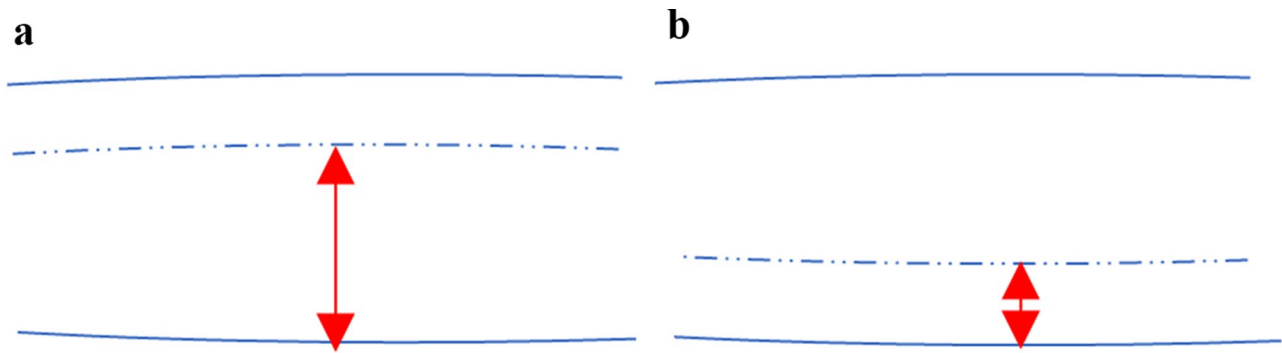


Fig. 3 At first, the shape of the flap was convex like **a**, and the blood flow seemed to be not so bad. However, when this shape of flap was to be concave during the diastolic phase like the right **b**, the true lumen was almost collapsed. Red arrow indicates the true lumen

found that the intimal dissection flap extended to the LCA ostium (Video 4). This dynamically disturbed the LCA blood flow because the LCA true lumen collapsed during the diastolic phase (Fig. 3).

As soon as the graft ascending aorta was again cannulated with heparinization, full CPB was re-established. After arresting the heart with cardioplegia, the surgeon meticulously checked around the LCA ostium and tightly obliterated the false lumen once again using BioGlue for LCA reperfusion. Despite another episode of ventricular fibrillation at the beginning of the second weaning from CPB, the LV hypokinesia and ST depression gradually improved, and the patient was successfully removed from CPB completely. At that time, her vital signs were stable. We reconfirmed that the TEE color doppler showed good blood flow through the LCA ostium and that the false lumen was thoroughly clogged (Video 5). She showed gradual recovery and stabilized condition during her hospital stay. Transthoracic echocardiography 2 weeks after the operation presented a satisfactory ejection fraction without any wall motion abnormality. Her limb power returned to normal except for her left arm grab power, which decreased mildly (grade 4/5). She was discharged 22 days after surgery with a plan for repetitive rehabilitation and brain imaging follow-up in our outpatient department.

Discussion

ATAAD is a devastating, life-threatening medical event [1, 4]. Without rapid diagnosis and treatment, happy endings are rare. The symptoms of ATAAD can be variable, and patients can present neurologic deficits with or without mental changes, as in our case. About 6% of ATAAD complications show these conditions, which are associated with cerebrovascular malperfusion secondary to dissection involving the carotid artery. Symptoms can also mimic acute MI with sharp chest or back pain [3, 10, 11]. Moreover, about 20% of patients with ATAAD show ECG signs suggesting myocardial ischemia due to coronary malperfusion. Sometimes patients with ST elevation

MI but finally diagnosed with ATAAD, which may have catastrophic effects because taking antiplatelet drugs for planned coronary angiography can cause uncontrolled bleeding and the rupture or dissection expansion [12–15]. Triphasic contrast enhanced aorta CT is the standard modality for diagnosing ATAAD, though TEE or TTE and magnetic resonance imaging can also be included [16, 17].

CAI secondary to ATAAD is rare, but it can worsen the prognosis. CAI presents in 10 to 15% of ATAAD patients. It is associated with younger age at presentation, and its in-hospital mortality is 20 to 30% [7–9]. It has been reported the RCA is involved much more often than the LCA because the false lumen mainly develops in the right anterior aspect of the ascending aorta [18]. However, recent multicenter research demonstrated that the involvement of the RCA and LCA in ATAAD was similar [19–21]. Our case showed a primary tear just above the RCA ostium in both direct visualization and TEE. CM is a deadly complication secondary to ATAAD [22]. In particular, LCA malperfusion causes worse outcomes than RCA malperfusion [7, 12]. In-hospital mortality from CM is 20–30%, and it is more than 90% when it is treated only medically. The reported incidence of CM among ATAAD patients was 6.1%, 9%, and 11.3% in different studies, and reports have been increasing [9, 12, 22, 23]. However, it can't be simply recognized, even in the surgical field [9, 18]. In this regard, TEE is an essential diagnostic tool during the ATAAD operation even though there are limitations for accuracy according to proficiency and limited time allocated before starting CPB or during weaning from CPB. TEE can evaluate not only the extension of the dissection flap and aortic regurgitation, but also the dynamic changes of RWMA. This information plays a crucial role to how to treat the patient. Our patient experienced smooth weaning from CPB at first, without any signs of CM. Then ECG ST segment changes and RWMA anteroseptal to the lateral wall with unstable vital signs suggestive a coronary perfusion problem occurred, which was when we finally found the LCA ostial malperfusion

in the TEE. Preoperatively, we concluded the dissection flap was not extended to the LCA ostium based on several reasons. Firstly, even though the flap was visualized near both coronary ostia in prebypass TEE, the blood flow using color doppler to the both coronary arteries was good. Secondly, the surgeon judged with direct vision that both coronary artery ostia were intact as well. Next, preoperatively, there were no myocardial ischemic signs. Preoperative TEE showed no regional wall motion abnormality, preoperative ECG showed no ST segment abnormalities, and preoperative cardiac markers were within normal ranges. Therefore, we thought the left coronary involvement was sudden event which occurred right after the first weaning from CPB. The abrupt new regional wall motion abnormality at the left coronary artery territories and sudden ST segment depression in ECG were the evidence. Finally, we tightly obliterated the false lumen once again using BioGlue for LCA reperfusion. There are ongoing debates about using BioGlue especially in aortic surgery. Lots of studies report the risk of pseudoaneurysm formation, tissue necrosis, and embolization due to BioGlue application [24–26]. On the other hand, some studies said careful use of BioGlue caused no obvious adverse problems, and it is a safe and effective adjunctive to thoracic surgery [27, 28]. In addition, Song S-W, one of our authors reported that when BioGlue was used properly, it significantly helps reduce bleeding and stabilize aortic tissue. This makes it an invaluable tool especially in high-risk cases where time is of the essence [29]. CAI of ATAAD can result in MI due to either static or dynamic obstruction of coronary arterial blood flow. The static mechanism is suggested by an expanding hematoma that results in arterial luminal narrowing. The dynamic mechanism indicates that the extended dissection flap compresses the true lumen of the coronary arteries, which makes the false lumen enlarge in the diastolic phase [12–14, 30]. We became certain of the CM only after seeing the dynamic narrowing of the patient's LCA ostium caused by the waving flap in the TEE. Unfortunately, both the static and dynamic physiology can induce the catastrophic symptoms of acute MI [31]. Neri's classifications have been used to assess the severity of CAI, but they cannot be the key for guiding surgical direction. To date, the optimal treatment modality remains unclear, and limited surgical guidelines exist. In the literature, Neri et al. suggested direct local repair of the involved coronary artery, whereas Kawahito proposed coronary artery bypass grafting (CABG) for all types of CAI [9, 32]. Those differences in opinions among studies can be confusing for surgeons, and the proposals are not completely practical [22, 30, 32]. Therefore, some studies have recommended therapeutic options [9, 30, 32]. As one of them, the Neri classifications were divided into 4 groups according to the degree of CAI. Neri A

indicates coronary ostial involvement by dissection, Neri B means dissection extending into the actual body of the coronary artery (CA), Neri C indicates circumferential detachment or complete avulsion of the CA, and the fourth category indicates a coronary orifice intimal tear (COIT). That study recommended repair in Neri A and most type B cases and suggested that Neri C be treated by CABG. In COIT, although its management should be individualized, the author said that orifice repair could be tried [30]. In those terms, our case was equivalent to Neri A and B, and local orifice repair, false lumen obliteration using glue, was successful. A retrospective study suggested that preoperative coronary CT angiography (CCTA) gives valuable information that could allow the surgeon to make a quick decision for combined CABG or not to improve the outcome [33]. This study said that the degree of luminal stenosis and orifice disruption in preoperative CCTA images is important for deciding the therapeutic direction. Even though CCTA avoids untimely determination by the surgeon in the surgical field, it difficult to proceed with CCTA after already performing contrast CT to diagnose ATAAD.

Conclusions

Recently, the surgical prognosis of ATAAD has been improving. Nevertheless, the degree of associated myocardial damage caused by CM is a major predictor of in-hospital death. Early diagnosis of CM thus plays a key role in patient outcomes. Most of all, LCA ostial involvement can bring about more devastating results than RCA involvement. However, it is challenging to recognize the degree of CAI preoperatively, and even during the operation. The important point is that if any signs indicative of CM appears, such as ST elevation or depression in ECG, RWMA, or CAI in intraoperative TEE or direct visualization in the field, an instant treatment plan is needed to reduce complications and minimize the mortality rate. Unfortunately, few exact surgical treatment directions or guidelines for CM secondary to ATAAD are available to improve patient outcomes. Therefore, not only are surgeons' instrumental in making rapid decisions to treat coronary artery perfusion, but anesthesiologists should also be proficient at handling TEE so they can provide information about the degree and aspect of CAI. Finally, it cannot be overemphasized that practical treatment guidelines based on various clinical experiences and studies is needed in the near future.

Abbreviations

ATAAD	Acute Stanford type A aortic dissection
CAI	Coronary Artery Involvement
CM	Coronary Malperfusion
MI	Myocardial Infarction
LCA	Left Coronary Artery
CT	Computed Tomography
ECG	EletroCardioGram

TEE	TransEsophageal Echocardiography
RCA	Right Coronary Artery
CPB	CardioPulmonary Bypass
RWMA	Regional Wall Motion Abnormality
CABG	Coronary Artery Bypass Grafting
CA	Coronary Artery
COIT	Coronary Orifice Intimal Tear
CCTA	Coronary CT Angiography

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12872-025-04556-4>.

Supplementary Material 1: **Video 1** Intimal dissection flap was just above the RCA ostium in the mid-esophageal aortic short and long axis views of the intraoperative TEE.

Supplementary Material 2: **Video 2** Color flow doppler shows good blood flow through the ostia of the LCA and the RCA in the aortic short axis view of the TEE.

Supplementary Material 3: **Video 3** The TEE shows sudden RWMA at the anterosseptal to the lateral wall in the 4 chamber views after several defibrillation due to ventricular fibrillation.

Supplementary Material 4: **Video 4** This video shows that the intimal dissection flap was extended to the LCA ostium.

Supplementary Material 5: **Video 5** Video 5. After weaning from the 2nd CPB, the final ejection fraction (5a) and blood flow of the LCA ostium (5b) was completely recovered.

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Author contributions

Suk-Won Song performed surgery, SangBeom Nam supervised whole anesthetic managements, and Younjin Kim arranged and handled the situations of operating room in case of emergency surgery.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Clinical trial number

Not applicable.

Consent for publication

Our institute has a Aorta surgery one-step system called 'Express'. The purpose of this 'Express' system is prevention of administrative delay and improvement of survival rate of aortic dissection. Here is the process of the 'Express'. If an emergency transfer occurs, information of patient such as symptoms and onset time, vital signs, neurologic examination, vasopressor infusion state, central and arterial line state, age, height and weight, ABO blood type, medical and surgical histories, history of taking antithrombotics, departure and arrival time, and CT image was shared among surgeons, anesthesiologists, nurses through phone message. Then, as soon as a patient arrives, he or she directly moves to the operating theater without any delay, and simultaneously we received an informed consent from his or her family protector. Also, our patient had mental change before surgery and we could not have a conversation with her at all. Also, she wanted to be communicated certainly to her protector, her spouse, about any information after surgery. Therefore, written informed consent was obtained from the patient's spouse for the publication of this case report and accompanying images.

Competing interests

The authors declare no competing interests.

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