A Case of Traumatic Retrograde Type A Aortic Dissection Accompanied by Multiorgan Injuries

Katsuaki Tsukioka, MD, PhD,1 Tetsuya Kono, MD, PhD,2 Kohei Takahashi, MD, PhD,1 Hiromu Kehara, MD, PhD,2 Shuichi Urashita, MD,2 and Kazunori Komatsu, MD, PhD2

A 75-year-old woman was involved in a traffic accident and suffered retrograde type A aortic dissection, multiple rib fractures, and grade II hepatic injury accompanied by intraperitoneal bleeding. We performed total arch replacement using an open stent graft with cardiopulmonary bypass and circulatory arrest. This procedure requires anticoagulation and hypothermia, which are principally contraindicated in severe trauma patients. However, this situation was resolved by managing the patient non-operatively for 7 days, confirming the stabilization of other injured organs, and then performing the surgery. She required prolonged postoperative rehabilitation; however, she recovered steadily.

Keywords: open stent graft, aortic dissection, hepatic injury

Introduction

Blunt aortic injury (BAI) in a patient with multiorgan injuries requires immediate open or endovascular aortic repair because of a high mortality rate of 55% within 2 days.1) We managed a patient with retrograde type A aortic dissection (RAAD) accompanied by multiorgan injuries, including hepatic injury. The open repair procedures for BAI warrant anticoagulation, which worsens bleeding from other injured organs; these challenges have been resolved in similar cases by performing thoracic endovascular repair (TEVAR).2) However, TEVAR is not performed at all institutions, and its application in RAAD has not been established. Partial cardiopulmonary bypass (CPB) with full heparinization is reportedly possible in a patient with only grade I or II solid organ injury3); however, RAAD repair in our patient required both total CPB and deep hypothermia for circulatory arrest, which should ideally be avoided in trauma patients.

Here, we report a successful delayed total aortic arch replacement with open stent grafting in a patient with traumatic RAAD and multiorgan injuries after successful stabilization of other injured organs and non-operative management (NOM) for 7 days.

Case Report

A 75-year-old woman was driving alone and was involved in a traffic accident. She was transferred to our hospital half an hour after the accident because of chest pain. Her medical history included only an orthopedic surgery. Upon arrival, her systolic blood pressure (sBP) was 80 mmHg, and she exhibited peripheral sweating, flail chest on the right side, and a minor contusion on the left lower leg. Focused assessment with sonography for trauma (FAST) did not reveal any intra-abdominal bleeding. Computed tomography (CT) was performed 40 min after her arrival following the achievement of a stable hemodynamic status by initial fluid resuscitation. CT revealed an aortic dissection with thrombosed false lumen in the ascending aorta (Fig. 1a) and primary entry in the descending aorta (Fig. 1b) with multiple rib fractures on both sides and a left hemothorax. A subcapsular grade II hepatic injury accompanied by intraperitoneal bleeding (Fig. 1c) was also observed on the initial CT image. The celiac, superior mesenteric, and left renal arteries were perfused from the true lumen, whereas the right renal artery was perfused from the false lumen. No pericardial effusion was observed; however, the large thrombosed false lumen present in the ascending aorta was likely to compress the right coronary orifice despite no ischemic change visible on the electrocardiogram. Echocardiography showed slight aortic valve regurgitation. The injury severity score, revised
trauma score, and probability of survival were 33, 7.11, and 0.70, respectively. Her hemodynamic status remained stable; therefore, we initially elected to perform medical therapy for RAAD. We maintained her stabilized condition under general anesthesia with mechanical ventilation, maintaining sBP < 110 mmHg and repeating the FAST examinations, which did not show any positive findings. A CT scan on post-injury day (PID) 3 showed disappearance of the intra-abdominal hematoma without progression of RAAD. However, the true lumen of the abdominal aorta was depressed, and an intrahepatic pseudoaneurysm was present (Fig. 2a) that placed the patient at the risk of life-threatening abdominal bleeding, which might have required interventional radiology (IVR). The right coronary orifice remained likely to undergo sudden occlusion (Fig. 2b). However, the serum aspartate transaminase (AST)
and alanine transaminase (ALT) concentrations decreased from 401 IU/L and 195 IU/L to 105 IU/L and 150 IU/L, respectively, indicating hepatic injury recovery. The prothrombin time/international normalized ratio (PT/INR) also decreased from 1.34 to 0.93, indicating recovery from the trauma-induced coagulopathy. Thus, we increased the sBP threshold from 110 mmHg to 130 mmHg to facilitate peripheral organ perfusion. On PID 6, her renal function began to decline, as evidenced by a gradual elevation in the serum creatinine kinase concentration (0.90 mg/dL on admission to 1.24 mg/dL on PID 6). This change was attributed to the decreased left renal perfusion secondary to the decreased flow in the compressed true lumen and decreased blood flow in the false lumen under strict sBP control, which resulted in decreased right renal perfusion. Therefore, we decided to discontinue the medical therapy for RAAD and performed total aortic arch replacement using a 26-mm J-Graft (Japan Lifeline, Tokyo, Japan) on PID 7. CPB was established by the cannulation of the right axillary artery and left subclavian artery interposed by a 9-mm J-Graft under systemic heparinization. Peripheral insertion of a J-Graft open stent with a 33-mm diameter and 120-mm length was performed under a temporary circulatory arrest with mild hypothermia of 26.0°C and selective antegrade cerebral perfusion. Intraoperatively, an intimal tear was observed at the distal portion of the left subclavian artery. The patient required postoperative transit hemodialysis. On postoperative day (POD) 2, a resternotomy was performed because of the worsening of the left hemothorax. In addition to postoperative flabbiness, her concomitant flail chest necessitated continuous positive pressure ventilation for internal fixation rather than rib fixation to prevent the worsening of general mobilization due to surgical invasion. Her effortive respiration gradually improved, and she was weaned from mechanical ventilation by POD 70. During this period, she underwent tracheostomy on POD 16, which spontaneously closed after disuse. CT scan on POD 16 showed expansion of the true lumen and promotion of thrombosis in the false lumen of the descending aorta (Fig. 3a). Although the false lumen in the abdominal aorta remained patent due to persistent reentry, the true lumen was enlarged with all abdominal branches, including the right renal artery, being patent and the intrahepatic pseudoaneurysm had spontaneously thrombosed (Fig. 3b). One and a half years after the surgery, she was discharged with improved mobilization.

**Discussion**

In previous studies, BAI constituted only 0.3% of all trauma cases requiring admission to the hospital. Of these BAI patients, 23% could not survive transport or triage and 55% died within 2 days; therefore, urgent repair was required. Considering these facts, RAAD of our patient also warranted immediate surgical, endovascular, and medical interventions to prevent adverse consequences. Lee et al. reported good outcomes of TEVAR in patients with BAI and multiorgan injuries. They found that the omission of systemic anticoagulation during TEVAR is occasionally possible, and this is particularly beneficial in patients with concomitant intracranial or abdominal bleeding. TEVAR seemed a promising procedure for immediate intervention if only the descending aorta was affected; however, in our patient, the whole aortic arch was involved, and the landing of the proximal site of the stent graft in the dissecting aortic arch could induce an additional intimal tear because of the fragility of the aortic wall secondary to the fresh dissection. However, in one study, systemic anticoagulation for open repair of BAI did not disturb NOM of grade I or II hepatic injury. Although our patient had grade II hepatic injury, she also had intraperito-
neal bleeding, and aortic arch repair required hypothermic circulatory arrest in addition to systemic heparinization. We considered this combination of anticoagulation and hypothermia to be lethal, especially in the early phase of NOM of hepatic injury. Symbas et al.\(^4\) reported better outcomes with delayed rather than immediate repair of BAI for appropriately-selected patients whose BP was strictly controlled. In addition, Kim et al.\(^5\) reported that the early mortality rate with medical therapy for RAAD was 6.1%, significantly lower than that with medical therapy for antegrade dissection (58.5%). Thus, an open aortic repair was less preferable, and medical treatment for RAAD might also have been a potential therapeutic option in this case. While the CT study on PID 3 showed no improvement in the ascending aorta, threatened myocardial infarction due to coronary artery compression and an additional pseudoaneurysm in the liver despite the disappearance of the intraperitoneal hematoma raised concerns about a life-threatening delayed bleeding that might necessitate IVR. We also considered that the narrowed true lumen of the abdominal aorta, especially at the level of the celiac artery, shown on CT would compromise IVR in case of pseudoaneurysm rupture. Therefore, permanent medical therapy for RAAD in our patient did not seem optimal. The development and prevalent use of open stent grafting has enabled not only the repair of intimal tears of the proximal descending aorta in patients with RAAD but also the exclusion of all antegrade blood flow in the false lumen, which could otherwise cause malperfusion syndrome or dilatation of the descending aorta in the long term.\(^6\) Therefore, we performed open stent grafting in our patient to facilitate IVR therapy by expanding the true lumen and to prevent dilation of the descending aorta.

The optimal time for attempting an open aortic repair in patients undergoing NOM of blunt hepatic injuries remains unclear. As Kaplan et al.\(^7\) reported, delayed hemorrhage generally occurs during the first 72 h. Repair within this period did not seem inevitable for our patient who was stable. However, Tiberio et al.\(^8\) reported that the median healing time of grade II subcapsular hematomas in the liver was 16 days, and Streit et al.\(^9\) reported tricuspid valve repair using CPB 17 days after NOM for concomitant blunt hepatic injury. Takahashi et al.\(^10\) also reported successful repair of an ascending aortic dissection caused by blunt trauma accompanied by multiorgan injuries 10 months after the incident. However, our patient was at high risk owing to hepatic pseudoaneurysm and renal dysfunction, which appeared on PID 6 due to malperfusion. Moreover, she was at risk of deep venous thrombosis and pneumonia due to the prolonged bed rest during NOM. Therefore, we could not continue NOM for several months, as reported in the aforementioned studies.\(^10\) However, no re-elevation in AST and ALT levels and PT/INR was observed, and we decided to perform the surgery on PID 7. The open stent grafting showed optimal results on postoperative CT that revealed closure of the entry in the descending aorta and good expansion of the true lumen of the abdominal aorta, especially at the level of the celiac branch, thus, facilitating IVR for the delayed hepatic bleeding. However, we were required to perform the open repair on PID 7 and could not confirm that this was the optimal period for the procedure. Thus, the optimal timing of aortic repair in such cases remains unclear and should be individualized.

**Conclusion**

Despite the conflicting coexistence of the demands for an imminent aortic arch repair and NOM for other severe injuries, we resolved this issue in the present case by delaying the aortic procedures for 7 days. The open stent grafting in the traumatic RAAD of our patient not only excluded antegrade flow in the false lumen, which could cause a late dilatation of the descending aorta, but also might have facilitated IVR for the possible late hepatic hemorrhage by expanding the true lumen in the abdominal aorta.

**Disclosure Statement**

The authors have no conflict of interest.

**Author Contributions**

Study conception: KT
Data collection: KT
Investigation: KT
Writing: KT
Critical review and revision: all authors

**References**