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Case Report

A Case of Paraplegia Treated with Cerebrospinal Fluid Drainage and Permissive Hypertension after Graft Replacement of the Ascending Aorta and the Total Aortic Arch for Acute Aortic Dissection Stanford Type A

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Paraplegia after an operation for acute aortic dissection Stanford type A (AADA) is fairly uncommon, and there is no consensus about optimal treatment. We present a case in which cerebrospinal fluid drainage (CSFD) and permissive hypertension were used for treatment of paraplegia. When the patient showed complete bilateral paraplegia after operation for AADA, we immediately began CSFD and maintained mean arterial blood pressure at over 90 mmHg. His neurological deficit gradually recovered, and he was eventually able to walk without support. The combination of CSFD and permissive hypertension could be a first-line emergent treatment for postoperative paraplegia after AADA surgery.

Key words: paraplegia, acute aortic dissection, cerebrospinal drainage, permissive hypertension

Paraplegia is one of the potentially devastating complications after aortic surgeries [1-3]. However, there is no consensus about optimal treatment for paraplegia after an operation for acute aortic dissection Stanford type A (AADA). We present a case in which cerebrospinal fluid drainage (CSFD) and maintenance of high blood pressure were successfully used for treatment of paraplegia associated with repair of an AADA. Written consent was obtained from the patient, and our institutional ethics committee approved the publication of this case report.

Case Report

A 69-year-old male (167 cm, 60.5 kg) was admitted to the hospital 1 h after the onset of chest pain. Emergent computed tomography (CT) showed an AADA from the ascending aorta extending into the bilateral common iliac arteries. We also confirmed the dissection of the right brachiocephalic artery, the bilateral subclavian arteries, and the bilateral common carotid arteries. Several left intercostal arteries branched from the false lumen. In addition, the celiac artery had blood flow from the false lumen, and the contrast effect of the pancreas and spleen was attenuated. The superior mesenteric artery was also dissected at its base. Despite these downstream compromises, the entry tear to the aneurysm was identified on the lesser curvature side of the ascending aorta and was thus considered a Stanford type A dissection.

The patient underwent emergent ascending aorta and arch replacement with placement of a stent graft into the descending aorta. Prior to surgery, he showed no neurological deficit in the extremities. General anesthesia was induced with 10 mg midazolam, 0.5 mg fentanyl, and 70 mg rocuronium and was maintained with sevoflurane, propofol, remifentanil, and rocuro-

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nium. A transesophageal echocardiography probe was inserted for intraoperative cardiac monitoring. BIS™ (Covidien, Minneapolis, MN, USA) and regional cerebral oxygen saturation (rSO_2) were monitored throughout the operation.

We obtained a surgical site through a median sternotomy and established a cardiopulmonary bypass (CPB) through cannulation from the right common iliac and right brachiocephalic arteries and bicaval drainage. After ascending aorta clamping, the heart was arrested with selective antegrade and intermittent retrograde cardioplegia. Simultaneously, the patient was subjected to systemic hypothermia at 28 degrees Celsius which is considered moderate hypothermia as a protective measure against ischemic injury. High blood flow through the CPB circuit with cooling temperatures at about 25 degrees Celsius achieved core cooling of the patient. After the body temperature reached 28 degrees Celsius, we started antegrade cerebral perfusion through the right brachiocephalic artery at a flow rate of 700 ~ 800 ml/min followed by additional selective perfusion through the left branches (left common carotid artery and left subclavian artery) at a flow rate of 500 ~ 600 ml/min. A stent graft (J Graft FROZENIXFRZX-25090 25 mm × 90 mm; Japan Lifeline, Tokyo, Japan) was inserted into the true lumen of the descending aorta and it was anastomosed end-to-end with the 4-branched graft (J Graft Branched Spiral 28 mm; Japan Lifeline, Japan). From the middle of the anastomosis, perfusion in the lower body was gradually resumed. After the completion of the anastomosis, antegrade perfusion through the extra branch of the 4-branched graft was launched (2,600 ml/min). Next, proximal aortic repair was performed in a standard fashion as the patient was rewarmed. Each branch was finally reconstructed after declamping (Fig. 1). The operation time was 8 h and 45 min, the aortic clamp time (myocardial ischemic time) was 2 h and 16 min, and the extracorporeal circulation time was 3 h and 26 min. The circulatory arrest time was 1 min for the brain and 58 min for the lower extremities. We maintained mean blood pressure between 50 ~ 60 mmHg. Blood loss was 2,530 ml, and we transfused 6 units of red blood cell, 4 units of fresh frozen plasma, and 10 units of platelet concentration during the surgery. rSO_2 was mostly maintained at around 50% without a significant bilateral difference. After completion of the surgery, the patient was transferred to the ICU under deep sedation.

We were unable to confirm his neurological condition on the first postoperative day (POD). Forty hours after the operation, he became conscious and exhibited complete bilateral paraplegia. Emergent CT showed no brain infarction or spinal hematoma. We suspected that ischemia of the spinal cord may have occurred during the surgery, and thus began CSFD to lower the pressure of the cerebrospinal fluid (CSF). We set the initial CSF pressure to 12 cmH_2O and maintained it between 12 and 26 cmH_2O to limit the amount of discharge below 15 ml/h (Fig. 2). At the same time we maintained the mean arterial blood pressure at over 90 mmHg with norepinephrine and fluid administration.

Twelve h after the initiation of CSFD, the patient was able to move his right lower limb at the MMT 1/5 level. On POD 4, we also confirmed motion of his left lower limb. Gradual recovery from the paraplegia ensued, and at 70 h after the start of drainage, the CSFD tube was clamped for 24 h to assess the change in neurological status of the lower limbs. Since no deterioration in the absence of drainage was observed, we removed the tube on POD 6. A total volume of 880 ml of clear CSF was drained in 70 h. He was extubated on POD 6, and he was discharged to the general ward on POD 8. Postoperative enhanced CT on POD 13 revealed delayed enhancement of the false lumen that was not thrombosed in the descending aorta, and most of the intercostal arteries were revealed to be open (Figs. 3, 4). The lower edge of the stent graft was at the Th6 level. By POD 23, he was able to walk with support. He was finally transferred to another hospital for additional rehabilitation on POD 32. After 3 months, he could walk without any support.

Discussion

Paraplegia is well known as a dreaded complication with a high incidence during descending thoracic or thoracoabdominal aortic aneurysm repair [1-3]. Paraplegia after an operation for AADA is less common, but it has been reported that any new postoperative neurological disorder after AADA surgery is associated with an increased rate of mortality [4, 5]. Spinal ischemia in patients who have undergone AADA may be caused by several factors: prolonged circulatory arrest, extensive replacement of the descending aorta, segmental artery obstruction by a stent graft, and spinal cord hypoperfusion following aortic false lumen clo-

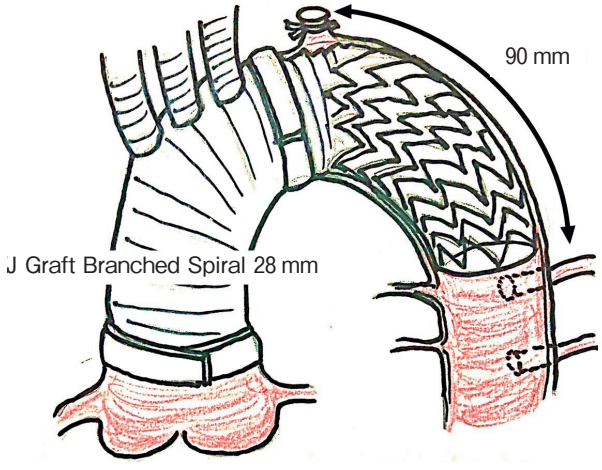

Fig. 1 Schema of the surgery. A stent graft (J Graft FROZENIXFRZX-25090 25 mm × 90 mm; Japan Lifeline, Japan) was inserted into the true lumen of the descending aorta and was anastomosed end-to-end with the 4-branched graft (J Graft Branched Spiral 28 mm; Japan Lifeline). The proximal aortic repair and each branch reconstruction were performed in a standard fashion.

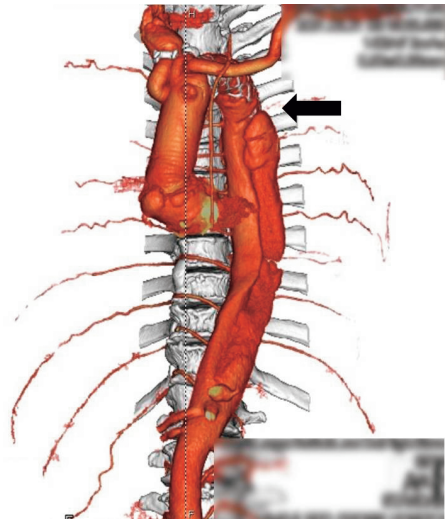

Fig. 3 Postoperative intercostal arteries and the stent graft. Postoperative enhanced CT revealed that most of the intercostal arteries were open and that the distal edge of the stent graft was at the Th6 level (closed black arrowhead).

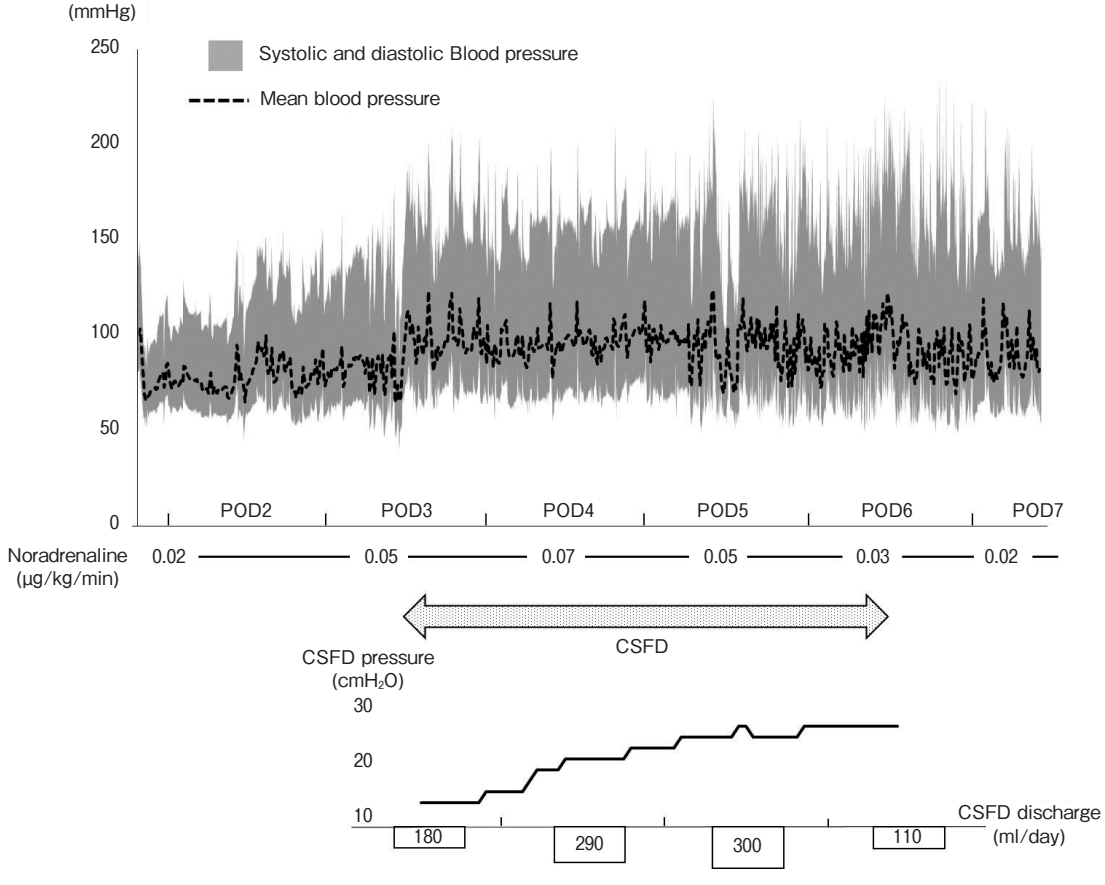

Fig. 2 Clinical course after the operation in ICU. CSFD, cerebrospinal fluid drainage.

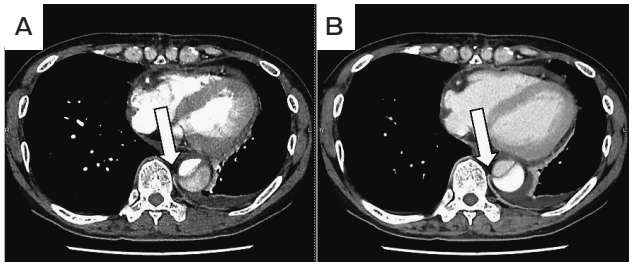

Fig. 4 Postoperative enhanced CT in the descending aorta. Postoperative enhanced CT revealed delayed enhancement of the non-thrombosed false lumen in the descending aorta. **A**, arterial phase; **B**, late phase; white arrowhead, left intercostal artery at the Th9 level from the false lumen.

sure. However, there is no consensus about optimal treatment, and only a few successful cases have been reported [4,6,7]. We describe a case of paraplegia that occurred after ascending aorta and arch replacement for AADA and was successfully resolved by CSFD and maintenance of high blood pressure.

We confirmed preoperative lower extremity motion in our patient and noted that both the true and false lumens were open on enhanced CT, suggesting no preoperative deficit of spinal cord blood flow. In addressing his postoperative paraplegia, we first considered that resection of the entry portion may have completely sacrificed the blood flow from the false lumen. However, postoperative CT showed that the left-sided lower intercostal arteries from the false lumen were still open, although their perfusion was delayed and flow might be retrograded from the distal re-entry portion. We also found that the stent graft was not disturbing the flow from the major intercostal and lumbar arteries. We finally speculated that the postoperative paraplegia was due to hypoperfusion without complete arterial occlusion, temporary spasm, micro thrombosis, or edema [2,8].

It is necessary to adopt a multimodal approach to minimize the ischemic insults in aortic surgery patients during the entire perioperative period. Before surgery, it is often difficult to confirm the presence of paraplegia of emergent ADAA patients because of unstable general conditions with hypotension, respiratory failure, severe pain, or sedation. Detection of the relationship of the Adamkiewicz artery, which supplies the anterior spinal cord, and the dissection area might be an effective target for predicting the risk of spinal ischemia. It is generally assumed that a shorter ischemic duration, especially in the lower body, can reduce the risk of neurological deficit including paraplegia [5,9]. Using distal clamping and early distal aortic perfusion is effective in maintaining spinal perfusion through intercostal arteries in total arch replacement [10,11]. During the CPB period in aortic surgeries, we usually implement moderate systemic hypothermia for attenuating ischemic injuries, including that to the spinal cord [10,11]. Neurophysiological monitoring of the spinal cord, like motor evoked potentials (MEP) during aortic surgeries, is supportive of the detection of spinal cord ischemia. Although reconstruction of intercostal arteries is difficult in AADA patients with a median sternotomy, hemodynamics can be optimized if the MEP indicates impairment [10,11]. A conservative or gradual approach to rewarming can avoid a mismatch between oxygen supply and demand in the spinal cord [12,13]. However, there is a delicate balance between prolonged CPB time and slow rewarming.

Recently, based on the concept of the collateral network, maintenance of spinal perfusion pressure at a high level has become widely accepted as a method of preventing spinal cord ischemia in patients with acute paraplegia [11,14-16]. Moreover, postoperative hypotension has been reported to be a potential cause of delayed paraplegia after aortic replacement [17]. We considered that permissive hypertension to maintain perfusion pressure in the feeding arteries was important in this case. We also considered that high blood pressure might be more effective for the delayed perfusion of left-sided intercostal arteries from the false lumen. However, it is unclear exactly how or why maintenance of high blood pressure is necessary for paraplegia patients. In addition, anesthesiologists have to guard against early bleeding after major vascular surgeries. We used 90 mmHg as the lower limit of mean arterial pressure based on previous reports [6,18].

We also considered that CSFD is effective for maintaining optimal spinal perfusion through collateral circulation to the site of the spinal cord's reversible ischemia. The spinal cord perfusion pressure can be defined as the difference between the mean arterial pressure and the CSF pressure. CSFD can lower the CSF pressure and thereby increase the spinal cord perfusion pressure. CSFD, especially during the repair of the descending or thoracoabdominal aorta, has been reported to reduce postoperative neurological complications [3,19,20]. We believe that prompt CSFD induction can contribute to the recovery of injured spinal cords even after AADA

repair, as some case reports have described post-surgical paraplegia successfully treated by CSFD [6,7]. Although we initially aimed to maintain CSF pressure below 10 mm Hg (13.6 cmH_2O), we maintained the pressure higher than this target because of the large amount of drained fluid, keeping in mind that excessive drainage of CSF poses a risk for intracranial hemorrhage [21-24]. More drainage might have been optimal to maintain proper CSF pressure and to hasten the neurological recovery.

Preoperative CSFD induction and intraoperative permissive hypertension are not practical for emergent AADA cases, although they might be effective as planned measures for elective surgeries. We think it is reasonable to intervene as early as possible after confirming postoperative paraplegia. Forty hours had passed before we detected the symptoms in this case, and we could have evaluated the movement of the lower limbs earlier to shorten that time. An animal study suggested that optimizing cerebrospinal blood flow and decreasing spinal cord metabolism postoperatively for 72 h could prevent paraplegia. There is reason to believe that treatment against postoperative paraplegia is best implemented within 72 h [25].

It is not unclear to what degree our strategy contributed to the patient's recovery from paraplegia in this case. However, we believe that the combination of CSFD and permissive hypertension could be a first-line treatment for postoperative paraplegia after AADA surgery because both can be performed quickly at the bedside.

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