Treatment of Delayed Neurological Deficits after Surgical Repair of Thoracic Aortic Aneurysm

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Delayed neurologic deficits, paraplegia and paraparesis, are devastating complications after repair of a descending thoracic and thoracoabdominal aortic aneurysm (TAAA). A treatment protocol has not been established, although strategies such as cerebrospinal fluid (CSF) drainage, maintaining blood pressure and medication have been described. Cerebrospinal drain status/oxygen delivery/patient status (COPS) therapy for delayed neurological deficit can improve spinal cord ischemia through reducing intraspinal pressure, improving oxygen delivery and maintaining high blood pressure. We describe one patient (Case 1), in whom descending thoracic and abdominal aortic aneurysms were treated by endovascular aortic repair, and another (Case 2) with Crawford type II TAAA, who developed delayed neurological deficits that were treated with immediate COPS therapy (Modified Tarlov scale; Case 1, improved from 2 to 4; Case 2, from 0 to 4). These findings indicate the benefit of COPS for treating delayed neurological deficits after descending thoracic and TAAA.

Keywords: thoracoabdominal aneurysm, paraplegia, endovascular procedure

Introduction

Delayed neurological deficits, paraplegia and paraparesis, are the most devastating complications after repair of a descending thoracic and thoracoabdominal aortic aneurysm (TAAA). A treatment protocol has not been established, although strategies such as cerebrospinal fluid (CSF) drainage, maintaining blood pressure and medication have been described. Estrera et al. recently described that cerebrospinal drain status/oxygen delivery/patient status (COPS) therapy can improve spinal cord ischemia after delayed neurological deficits occur. This therapy reduces intraspinal pressure via CSF drainage, improves oxygen delivery by hemoglobin transfusion and maintains high blood pressure. We describe two patients with delayed neurological deficits that were treated with COPS (Fig. 1).

Case 1

A 71-year-old man with gastric and lung cancer was referred to our department to treat saccular descending thoracic aortic (maximum diameter, 48 mm) and abdominal aortic (maximum diameter, 54 mm; Fig. 2a) aneurysms. We selected endovascular aortic repair to treat the aortic aneurysm to minimize risk. A Talent® (Medtronic AVE, Santa Rosa, CA, USA) and a Zenith AAA Endovascular graft® (Cook, Bloomington, IN, USA) was deployed under general anesthesia. Preoperative enhanced computed tomography (CT) revealed that the branch level of the preserved Adamkiewicz artery (AKA) was right Th12. Intraoperative angiography and postoperative status (COPS) therapy can improve spinal cord ischemia after delayed neurological deficits occur. This therapy reduces intraspinal pressure via CSF drainage, improves oxygen delivery by hemoglobin transfusion and maintains high blood pressure. We describe two patients with delayed neurological deficits that were treated with COPS (Fig. 1).
enhanced CT (Fig. 2b) confirmed the absence of an endoleak and the patient could move the lower extremities. However, COPS was immediately started to treat paraparesis that occurred on post-operative day 1 (Modified Torlov scale2; 2). First day: 70 cc/day, second day: 10 cc/day and third day: 30 cc/day of the CSF was drained. The CSF drainage tube was removed on the fourth day because of risk for meningitis. The patient was transfused with red cell concentrates (RCC) for anemia (Hemoglobin: 8.6 g/dl). Mean blood pressure (MAP) was approximately 80 mmHg, so we used catecholamine support, and MAP was increased to >90 mmHg. The SpO2 was maintained 96%–100% in the perioperative period. He could draw up his knees on the following day and could walk with assistance (Modified Torlov scale: 4) on postoperative day 17.

Case 2

A 35-year-old man with Marfan syndrome had undergone surgical aortic repair of a total arch replacement, the Bentall procedure, and repair of an abdominal aortic aneurysm. However, a Crawford type II TAAA persisted and had expanded (maximum diameter, 56 mm; Fig. 2c). We performed TAAA repair. The aorta was exposed via a thoracoabdominal incision and circumferential division of the diaphragm under general anesthesia. An aortic aneurysm had replaced the artificial graft and was supported by distal perfusion, selective visceral perfusion and selective intercostal artery perfusion. Preoperative enhanced CT showed that the branching level of the AKA was left Th9 and that the intercostal arteries (left Th8 and Th9) were reattached. Motor-evoked potentials (MEP) remained constant during the procedure, after which the patient was admitted to the intensive care unit. He regained consciousness 4 hours later, and movement of the lower extremities was confirmed. Mean blood pressure was maintained at >80 mmHg. He was extubated at 13 hours after the operation, but required infusion with Flurbiprofen axetil (50 mg) 8 hours later to relieve pain sufficiently to sleep. Mean blood pressure at this point had decreased to 60 mmHg. One hour later he awoke with leg weakness that was confirmed as paraplegia (Modified Torlov scale: 0) and COPS therapy was immediately started. First day: 100 cc/day and second day: 12 cc/day of the CSF was drained. The CSF drainage tube was removed on the third day. The patient was transfused with RCC for anemia (Hemoglobin: 10.0 g/dl). The MAP was increased to >90 mmHg with catecholamine support. The SpO2 was maintained 98%–100% in the perioperative period. 5 hours later occurrence of the event, he could draw up his knees. By postoperative day 7, he could walk with assistance (Modified Torlov scale: 4). Post operative enhanced CT showed that the reattached intercostal arteries were occluded (Fig. 2d).

Discussion

Despite the use of various strategies for the prevention of spinal cord ischemia, paraplegia and paraparesis continue to occur after open and endovascular thoracoabdominal aneurysm repair. The reported incidences of delayed neurological deficits after TEVAR and TAAA repair are 0%–12%3) and 4%–16%,4,5) respectively. Several adjuncts, such as reattached intercostal arteries, distal perfusion, CSF drainage, maintaining high blood pressure, cooling the spinal cord and drugs such as Naloxone6) and steroids,7) can help protect the spine. Improving spinal cord perfusion is important for recovering neurological deficits since spinal ischemia can cause paraplegia/paraparesis.8) Estrera et al. described COPS as a protocol for treating delayed neurological deficits (Fig. 1)
through improving spinal cord perfusion and other parameters.\textsuperscript{19} The protocol contains several criteria including mean blood pressure $>90$ mmHg and hemoglobin $>12.0$ g/dL. Though there are no new adjuncts in the protocol, the significance of the protocol is the organization of several adjuncts, and it includes clear criteria in the treatment protocol for delayed neurological deficit.

We discussed which factors caused the delayed neurological deficits in our patients. The patient in Case 1 had a malignancy, and we immediately performed simultaneous EVAR and TEVAR. Delayed neurological deficits occurred despite preservation of the Adamkiewicz artery. Seriously reduced spinal cord perfusion caused by EVAR and TEVAR might have been the cause. Anemia induced by the malignancy could also have caused spinal cord ischemia. The patient in Case 2 had undergone the Bentall procedure and had been implanted with a mechanical aortic valve. Heparinization for surgical preparation was necessary, and CSF drainage was associated with a risk of subdural hematoma. The risk of a delayed neurological deficit was high for this patient, and intercostal artery reattachment was important because of the Crawford type II thoracoabdominal aortic aneurysm. Preoperative CT revealed the Adamkiewicz artery at left Th9 and the left Th8 and Th9 intercostal arteries were reattached. Technical errors did not arise during surgery, since the MEP remained constant. However, the postoperative pain killer caused a fall in blood pressure, which might have been associated with the delayed neurological deficit. Postoperative CT showed occlusion of reattached intercostal artery, which might also have been a factor. However, Shiya et al. reported that the patency of a reattached intercostal artery is $42\%$\textsuperscript{9}, which is insufficient. Christian et al. reported that routine surgical reattachment of intercostal artery is not indicated\textsuperscript{10}, as occlusion of only a reattached intercostal artery does not cause delayed neurological deficits.

The time from the onset of delayed neurological deficit to COPS therapy might influence the likelihood of recovery. Although the duration of spinal cord tolerance of ischemia is unknown, immediate therapy seems important for recovery. Delayed neurological deficit is expected to recover if appropriate treatment is performed at an early stage. Since most cases of delayed neurological deficit occur within 12–24 hours after surgery, attention must be paid to sigh of neurological disorders in that period.
Postoperative sedation should be avoided to achieve early awakening. Acute postoperative sensory perception and lower extremity movement should be monitored, and if neurological deficits are identified, then immediate COPS therapy will be critical to a good outcome.

Finally, we describe the strategy for prevention of neurological deficit in our hospital. (a) Identification of the Adamkiewicz artery (AKA): We identify the AKA by computed tomographic angiography or magnetic resonance angiography before surgery. In open surgery (e.g., descending thoracic aortic replacement, thoracoabdominal aortic replacement), the intercostal artery related to the AKA is reconstructed if the AKA exists at the level of aortic replacement. In endovascular surgery, a CSF drainage tube is inserted previously if occlusion of the AKA is expected.

(b) Cerebral spinal fluid (CSF) drainage: The CSF drainage tube is inserted on the day before the operation. However, some patients are administered anticoagulant or antiplatelet drugs, and we do not insert a CSF drainage tube in such patients because of the risk of hemorrhage.

(c) Monitoring of motor evoked potentials (MEP): During open surgery, we monitor MEP and check that spinal cord perfusion is adequate. (d) COPS therapy: Since COPS therapy may be useful for delayed neurological deficit, it will also be effective for preventing delayed neurological deficit. Hemoglobin is increased by blood transfusion and mean blood pressure is maintained at a high level in accordance with criteria of the COPS protocol. However, maintaining high blood pressure is a risk of bleeding in patients with hypertension, and adequate hemostasis should be performed during surgery.

We think that the strategy described above and COPS therapy will reduce delayed neurological deficit.

Disclose Statement

We declare that there are no conflicts of interest associated with this report.

References