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CLINICAL REPORT

Extensive spinal cord ischemia following endovascular repair of an infrarenal abdominal aortic aneurysm: a rare complication

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Abstract Postoperative paraplegia secondary to spinal cord ischemia (SCI) is an extremely rare and devastating complication of endovascular repair in abdominal aortic aneurysm (AAA) surgery. The reported incidence is only 0.21 % worldwide. This case of postoperative paraplegia occurred in a 60-year-old man immediately following endovascular repair of an infrarenal AAA. Postoperative magnetic resonance imaging showed multiple foci of SCI involvement from C5 to L1. However, neither cerebral spinal fluid drainage nor steroid therapy was effective; he was eventually admitted with no improvement in his neurological status. The mechanism remains multifactorial until now and needs more attention in perioperative management. We report the first case involved in the most significantly extensive SCI after endovascular repair of an infrarenal AAA.

Keywords Spinal cord ischemia · Endovascular surgery · Aortic aneurysm · Postoperative complication

Introduction

Postoperative paraplegia secondary to spinal cord ischemia (SCI) is an uncommon and devastating complication following elective repair of an infrarenal abdominal aortic aneurysm (AAA) [1]. In the past, open surgery was the only treatment for an aortic aneurysm. However, with advancements in surgical techniques, endovascular repair for aortic aneurysms has become an alternative treatment. A recent report showed the 30-day operative mortality for endovascular surgery was significantly lower than that for open surgery (1.8 versus 4.3 %) [2]. Endovascular therapy is less invasive and causes less blood loss, fewer complications, and fewer mortalities, contributing to shorter hospital stays than open surgery [3]; however, some complications, such as artery rupture, endo-leak, stroke, renal failure, infection, and SCI, may occur [4]. One of the most severe complications of aortic aneurysm surgery is SCI, with an incidence of 0.25–0.3 % [5]. Here, we report a rare case of immediate-onset paraplegia and extensive SCI following endovascular repair of infrarenal AAA.

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Case report

A 6-cm-wide infrarenal AAA was found incidentally by abdominal ultrasonography during the health checkup of a 60-year-old man with a history of hypertension under medical control. The abdominal computed tomography angiography showed a 16×6 cm infrarenal AAA, extending from the infrarenal abdominal aorta to the end of the abdominal aorta with partial thrombus formation. Subsequently, a bare metal stent was inserted in the left anterior descending coronary artery because of coronary artery disease, which was shown in the coronary angiography workup for the elective endovascular surgery. Antiplatelet drug therapy, including aspirin and clopidogrel, was prescribed after coronary angioplasty. One month later, the elective endovascular surgery was performed under general anesthesia. The patient's blood pressure was initially 135/94 mmHg after induction for anesthesia with fentanyl, propofol, and rocuronium and was maintained by sevoflurane throughout the entire procedure. The systolic

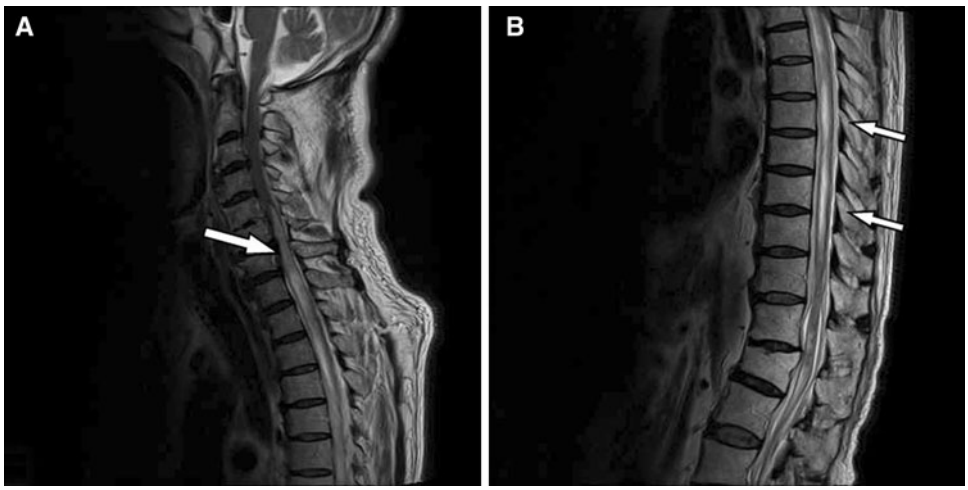

Fig. 1 Magnetic resonance imaging (MRI) scan showed spinal cord ischemia (SCI) in this patient postoperatively. **a**, **b** Sagittal T_2-weighted image demonstrated pencil-like lesions of multiple extensive intramedullary hyperintensities from C5 to L1 corresponding to cytotoxic edema with blood–brain barrier breakdown below T1 level (*white arrows*). These imaging findings are consistent with ischemic changes rather than degenerative, inflammatory, or neoplastic processes

pressure was kept above 100 mmHg during the operation, and urine output was maintained at no less than 1 ml/kg/h. Following systemic heparinization, the active clotting time was kept above 200 s. Endovascular repair was carried out with a left leg stent graft deployed initially, but the angiogram revealed a mild type I endo-leak. An extensor cuff was used for the stent graft sealing area. However, a type III endo-leak was suspected from the left distal stent graft overlapping site, so a contralateral limb stent graft was used to recover the leak site. The completion angiography revealed that the stent graft was positioned well without any endo-leak, and the whole procedure lasted 5 h.

Immediately after the patient regained consciousness, he stated that he could not move his legs and had lost sensation below the clavicles. The neurological examination showed full muscle power over the upper limbs but no sensory or motor function below the T1 level, with leg paralysis and loss of anal tone. Emergent magnetic resonance imaging (MRI) showed long T_2 signals in the spinal cord as myelopathy at the C5–L1 level, with multiple postcontrast enhancements and cytotoxic edema below the T_1 level, such as recent infarction of the spinal cord (Fig. 1). Although the patient underwent cerebrospinal fluid (CSF) drainage, which was maintained at a constant pressure of 10 cm H_2O with methylprednisolone (30 mg/kg bolus and 5.4 mg/kg drip for 23 h) treatment for 72 h, his neurological condition did not improve. Therefore, rehabilitation was initiated on the 4th postoperative day. As of the writing of this report, the patient is still paralyzed in both legs, needs a wheelchair for mobility, and has had a pressure sore over the sacral area for 10 months.

Discussion

Immediate- and delayed-onset paraplegia have been reported as rare complications after endovascular aortic repair [6, 7]. An analysis of the EUROSTAR database, including 2,862 patients who had undergone endovascular AAA, revealed an incidence of 0.21 % for SCI [8]. The mechanism involved in SCI is not fully understood, but it is thought to be multifactorial. Even though endovascular repair of an aortic aneurysm does not require large incisions or aortic cross-clamping and does not cause any significant blood loss, the maintenance of vital organ perfusion, which depends on stable perioperative hemodynamics, becomes a more important concept. The interruption of the spinal arteries, prolonged aortic occlusion, intraoperative hypotension, atheromatous embolization, and interference with collateral circulation arising from the internal iliac artery circulation have all been suggested as possible causes of SCI [9, 10].

In most people, the spinal cord is supplied by two posterior spinal arteries and one anterior spinal artery. The posterior spinal arteries supply the sensory tracts in the spinal cord and receive flow from posterior and inferior cerebellar arteries, vertebral arteries, and posterior radicular arteries. The largest radicular artery, which is called the artery of Adamkiewicz, perfuses the anterior two-thirds of the spinal cord and originates variably from an intercostal artery from T5 to L3 in 75 % of cases [11]. Additional supplies to the distal cord arise from the iliolumbar arteries, the lateral sacral arteries, and branches of the hypogastric arteries. In particular, the spinal cord from C1 to T3 is supplied at the C3 level from the vertebral arteries, which receive their blood supply from the costocervical trunk; this is a branch of the subclavian artery, which is less likely to undergo atheromatous change [12]. With the occlusion of the artery of Adamkiewicz, an increased amount of perfusion pressure is considered necessary along with the collaterals to maintain adequate perfusion pressure to the spinal cord. In a recent study, McGarvey et al. [13] recommended early intervention and aggressive blood pressure augmentation by increasing the mean arterial pressure

by 10 mmHg every 5 min until the weakness is resolved or an unacceptably high blood pressure is reached postoperatively. Chiesa et al. [14] showed that a perioperative mean arterial pressure less than 70 mmHg was a significant predictor of SCI. We tried to maintain the mean arterial pressure above 70 mmHg during the whole procedure, but SCI still occurred. Preserving the mean arterial blood pressure to control hemodynamic status aggressively may be the optimal spinal cord protection strategy.

SCI can also be prevented by minimizing the duration of the surgery, preventing hypotension, limiting manipulation of a catheter, and optimizing spinal cord perfusion [15]. In an analysis of the EUROSTAR registry, Berg et al. [8] described the clinical factors associated with intraoperative microembolization, including prolonged procedure (>150 min), extensive intravascular handling, and preoperative or perioperative embolization of the hypogastric and lumbar arteries. Although the procedure involves routine coverage of the inferior mesenteric artery (IMA) and lumbar arteries with the instrumentation of the aneurysmal vasculature, the stent graft adjustment may have contributed to excessive intravascular manipulation, prolonged the length of the procedure, and disseminated the emboli to the spinal cord circulation as well. In our case, it is notable that the computed tomography-angiography images indicated that there was thrombus formation in the abdominal aorta and the atheroembolic materials may decrease the collateral circulation. Therefore, we should avoid high risks such as delivery problems, anatomically difficult procedures, or an intraoperative coil to cause the embolization of aortic branches.

The SCI can be developed gradually during surgery in most cases, indicating that physicians should pay more attention to intraoperative neurological signs. It is vitally important to detect SCI as soon as possible. Motor evoked potential (MEP) and somatosensory evoked potential (SSEP) are useful methods for monitoring the spinal cord to help guide management during the procedure [16]. Jacobs et al. [17] reported that MEP could reduce the likelihood of SCI by the adjustment of hemodynamic strategies in most patients. When a mean distal aortic pressure of 60 mmHg was maintained, MEP was adequate in 82 % of patients, and SSEP had the same effect as MEP. A previous study has suggested that routine SSEP monitoring, serial neurological assessment, arterial pressure augmentation, and CSF drainage may aid in early detection and intervention to benefit patients at risk for paraplegia [18]. The immediate detection of SCI by intraoperative neurological monitoring and examination can be effective in treating neurological deficits after endovascular repair.

In conclusion, SCI after endovascular repair is an unpredictable complication. Our case is a rare, unique, and complicated case of postoperative paraplegia because of extensive and multiple foci of SCI involvement from C5 to L1. The mechanism remains unclear, and physicians should be aware of this complication in patients with comorbidities of atherosclerosis, coronary artery disease, or peripheral artery occlusive disease, or difficulties with device deployment. Continuous perioperative neurological monitoring and early intervention can help us to detect neurological changes and avoid critical complications.

Conflict of interest None.

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