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Case Report

Paraplegia following elective endovascular aneurysm repair of a common iliac-dissecting aneurysm

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Abstract: This is the report of a case of an 82-year-old gentleman who developed paraplegia after an elective endovascular aneurysm repair for a right common iliac-dissecting aneurysm. Postoperative magnetic resonance imaging (MRI) of the spine showed an infarct at the T10-L1 level (which has been acknowledged as Syringomyelia). The complication was not predicted as the patient did not have the classic aortic aneurysm with thrombus and had preserved perfusion of both internal iliac artery (IIA) after the operation. In our opinion, this suggests intraoperative and postoperative hemodynamics play a unique role in the development of spinal cord ischemic injury.

Keywords: Abdominal aortic aneurysm, endovascular aneurysm repair, paraplegia, spinal cord ischemia

Introduction

Postoperative paraplegia secondary to spinal cord ischemia (SCI) is a rare and devastating complication after endovascular abdominal aortic aneurysm repair (EVAR). The reported incidence of paraplegia after EVAR is only 0.21%, as high as after open treatment [1]. The occurrence of paraplegia after implantation of endovascular aneurysm devices may be associated with several etiologies. The mechanism involved in SCI remains not completely understood. It is thought to be multifactorial, and more attention may be necessary during perioperative management. This is a case of a patient with a right common iliac artery dissection who developed paraplegia after elective endovascular aneurysm repair. When reviewing the root cause analysis, hemodynamic control was identified as the most likely cause. The case brings up an important issue regarding paraplegia after EVAR and raises the possible implications for EVAR procedures as to preventing SCI. Informed consent was obtained for the case report, images, and publication.

Case report

The 82-year-old man had a significant past medical history for severe chronic bronchitis and presented with swelling and pain at the right abdomen. The patient's history of tuberculosis extended for 50 years. The patient had no prior thoracic (or abdominal) aortic surgery and his left subclavian artery patency was unobstructed. After failure of symptomatic treatment by the general surgery team, the patient was sent to Department of Vascular Surgery after a finding of a right common iliac artery dissecting aneurysm on computerized tomography (CT) scan. Aortography revealed a dissecting-aneurysm measuring 2 cm ("double chamber" sign) at root of the right common iliac artery and normal aorta wall without mural thrombus. The Department of Vascular Surgery decided that an EVAR under local anesthesia should be used given the patient's pulmonary function test revealed severe mixed ventilation dysfunction. His intravenous access included 20 gauge IV. The patient underwent a successful endovascular repair with Bifurcated Endovascular Grafting System (Lifetech Scientific, Shenzhen, China) performed below the renal arteries. The distal attachment sites were the common iliac arteries bilaterally, with maintenance of perfusion of bilateral internal iliac arteries. The completion angiography revealed that the stent graft was well-positioned without any endoleak and bilateral renal arteries and internal iliac

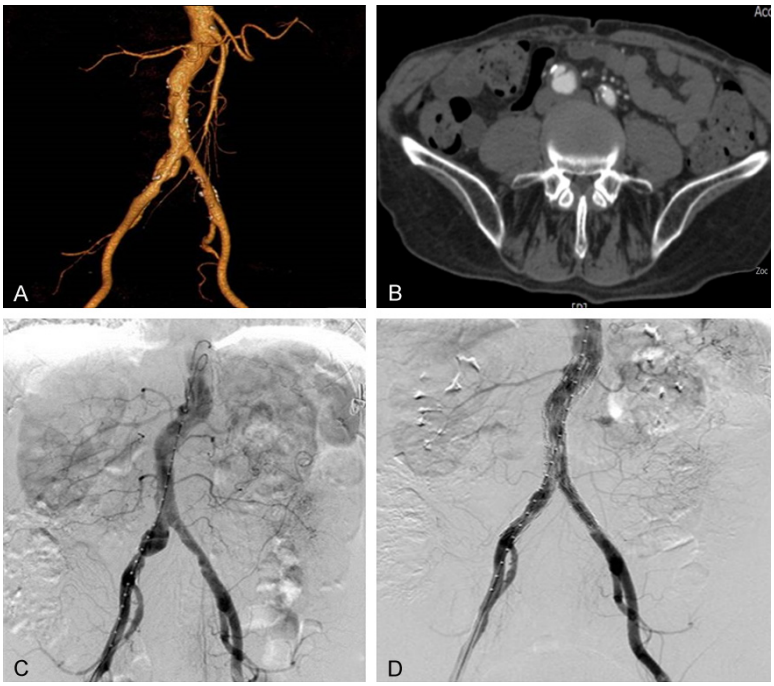

Figure 1. A. Pretreatment Angio-CT: MIP reconstruction of aorto-iliac vessels; B. Pretreatment Angio-CT showing a dissecting-aneurysm measuring 2 cm ("double chamber" sign) at root of the right commoniliac artery and normal aorta wall without mural thrombus; C. Intraoperative DSA showed aneurysmal dilatation of the proximal root of the right common iliac artery, and we decided to have endovascular treatment to repair the dissecting-aneurysm using bifurcated stent graft (Lifetech Scientific Corp, Shenzhen); D. The completion angiography revealed that the stent graft was positioned well without any endo-leak and preservation of the renal arteries and both internal iliac arteries.

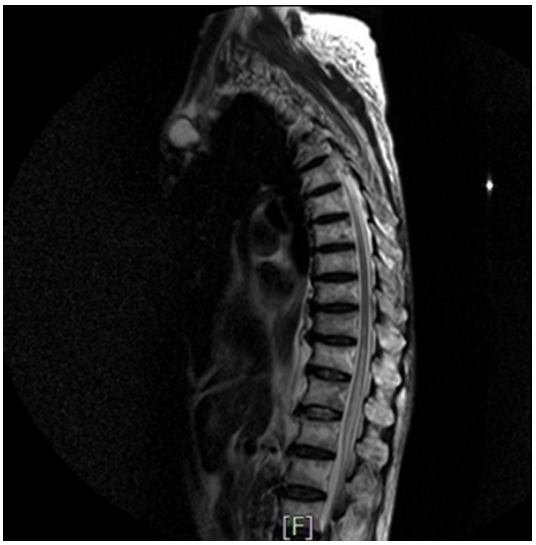

Figure 2. Magnetic resonance image showing spinal cord infarction at T10-L1 level.

arteries were well preserved (**Figure 1**). The procedure lasted 2 hours. The patient's vital signs were stable throughout the procedure except towards the end of the surgery when there was acute bleeding of the patient's wound due to the lack of cooperation at that time because he wasn't able to tolerate pain. The venous pathway was not well prepared before operation (no deep vein catheterization was performed), and hypotension (mean arterial pressure less than 70 mmHg) lasted a period of time (more than 30 minutes). The patient had inadequate IV access. During this period of acute bleeding, the patient was hypotensive for more than 30 minutes. The patient had stable baseline neurologic exam immediately after surgery. The patient's vital signs were adequate throughout the postoperative and he was not anemic (Hemoglobin of at least 100 g/L).

On the morning of postoperative day 1, the patient was found to have severe paraparesis and bladder dysfunction. With neurology consultation, he was found to have lost sensory and motor function below T10, with leg paralysis and bladder incontinence. The manual muscle test of the lower extremities was 0/5 bilaterally. Emergency magnetic resonance imaging (MRI) showed long T_2 signals in the spinal cord with multiple post contrast enhancements and cytotoxic edema below the T10 level, similar to the recent infarction of the spinal cord found on the previous MRI, which had been mistaken for syringomyelia (**Figure 2**). Cerebrospinal fluid (CSF) drainage was not performed due to patient's refusal. Instead, he was treated with methylprednisolone and hyperbaric oxygen therapy for 1 week. He showed no improvement on the physical examination and was then transferred to a rehabilitation center. At the 12-month follow-up, the patient remained paraplegic. He was unable to walk or stand independently and was confined to a wheelchair. He had no sensation below the T4 level, he had lower-extremity muscle atrophy, and bladder and bowel inconti-

nence. The manual muscle test of the lower-extremity was 0/5 bilaterally.

Discussion

Spinal cord ischemia following elective endovascular aneurysm repair (EVAR) for abdominal aortic aneurysm is an extremely rare condition. The mechanism involved in this condition is multifactorial, and is not fully understood. It is known that spinal cord ischemia can be attributed to changes in the spinal cord perfusion pressure. There are several predictive factors leading to this condition.

Obstruction of blood supply to the spinal cord

Perfusion to the spinal cord is provided by the segmental intercostal and lumbar arteries, especially variants of the great radicular artery (Adamkiewicz artery) and internal iliac artery (IIA). This can often be obstructed during open or endovascular repair of abdominal aortic disease leading to spinal cord injury. Nevertheless, there are different views on this question at the same time that a corresponding artery occlusion is often found in clinic without SCI. Metha et al. reported that no case of paraplegia was found in a series of 107 intentional hypogastric interruptions (eight bilateral and 99 unilateral) during EVAR [2]. Lee et al. also did not observe neurologic complication in 28 patients with HA (hypogastric artery) obstruction [3]. Morever, Freyrie et al. observed one case of paraplegia even after surgical revascularization of one HA [4]. In addition, it is of little value to evaluate the anatomic variation of the Adamkiewicz artery before the operation since the positive rate of preoperative locating the great radicular artery is low. As for our case, it was a common iliac-dissecting aneurysm, not a typical abdominal aortic aneurysm. There were no serious wall thrombus or other embolism factors, and bilateral internal iliac arteries were well preserved during EVAR. The duration of aortic occlusion during EVAR was short (actual 3-5 minutes), and spinal cord injury under local anesthesia is rare. However, severe and irreversible SCI was found in the patient postoperatively. As a result of the foregoing, we have to question the validity of the view.

Systemic hypotension resulting in cord hypoperfusion

The incidence of neurologic complications after elective EVAR is quite low, compared to the higher incidence of SCI observed in ruptured AAA (rEVAR) using an AUI device. The reason was supposed lied in an extended period of functional aortic occlusion and hemorrhagic shock or hypotension [5]. Chiesa et al. found that a perioperative mean arterial pressure less than 70 mmHg was a significant predictor of SCI [6]. Perioperative hypotension is a well recognized caused of SCI. The patient underwent local anesthesia because of his poor pulmonary function during the operation, which resulted in the acute bleeding of the wound due to the lack of cooperation in the operation because he wasn't able to tolerate pain. He had less than optimal intravenous access, and he experienced profound hypotension to as low as 55/40 for more than 30 minutes. Therefore, it is reasonable to believe that perioperative hypotension played an important role in the development of this patient's SCI.

High central venous pressure (CVP)

Spinal cord perfusion pressure decreases with increasing CVP. The CVP in patients with chronic obstructive pulmonary disease (COPD) tends to be high, which leads to a higher pressure in the extensive vertebral venous plexuses. A high CVP would impede spinal cord perfusion and ultimately lead to spinal cord ischemia [7]. A high CVP in addition to a lower mean arterial blood pressure would compromise spinal cord perfusion. This patient has a clear history of COPD and a drop in blood pressure during the operation. Although there were no traditional risk factors to the patient, such as long aortic coverage or major branch vascular occlusion, severe SCI still occurred after surgery.

Although various factors contributed to the occurrence of SCI, the ultimate cause of SCI is the decrease of the spinal cord perfusion pressure. Since the spinal cord has multiple vascular collaterals, the probability of SCI arising from an interruption of a certain blood supply is low unless obstruction to blood supply occurred on multiple levels. The decrease in spinal cord perfusion pressure caused by the rapid change of hemodynamics likely caused extensive spinal cord ischemia, in a similar manner to ruptured abdominal aortic aneurysm, which may lead to serious consequences if no effective corrections are made promptly.

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Disclosure of conflict of interest

None.

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