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Case Report

Brown–Séquard Syndrome after Thoracic Endovascular Aortic Repair for a Stanford Type B Aortic Dissection

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We present a case of Brown–Séquard syndrome (BSS) after thoracic endovascular aortic repair (TEVAR) to treat Stanford type B aortic dissection. A 49-year-old male presented to the emergency department with acute tearing pain between the scapulae, connected to respiratory movements. Computed tomography showed Stanford type B aortic dissection from the left subclavian artery to the level of the 11th thoracic vertebra. Conservative treatment was initiated with intravenous antihypertensives. However, due to persistent pain and an increase in the aortic diameter with an intramural hematoma, TEVAR was performed. The patient developed symptoms suspicious of spinal cord ischemia postoperatively. A lesion limited to the left-sided spinal cord was observed on magnetic resonance imaging at the level of the 4th to 5th thoracic vertebra. BSS after TEVAR is a rare phenomenon with a fairly good prognosis, depending on the initial injury severity.

Key Words: Aortic dissection, Endovascular procedures, Vascular surgery, Spinal cord ischemia

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INTRODUCTION

Brown–Séquard syndrome (BSS) is a rare neurological condition caused by a unilateral spinal cord lesion and was first described in the 1800s [1-3]. It is normally characterized by two aspects: 1) ipsilateral loss of motor function and proprioceptive sensation; and 2) contralateral loss of temperature and pain sensation. BSS is often associated with trauma and can also be iatrogenic after endovascular surgery [1,2,4,5]. Recently, multiple methods have been developed to protect against spinal cord injury during endovascular aortic surgery; however, neurological symptoms remain a major concern. Cerebrospinal fluid drainage, distal aortic perfusion, or a combination of both has been recommended to decrease the incidence of postoperative neurological deficits [6-8]. These neurological deficits are usually bilateral and can result in paraplegia, with an incidence of 5% to 30%, depending on the extent of the surgery and the use of the previously mentioned preventative strategies [6,8]. Pathophysiologically, the mechanisms of BSS include spinal cord hypoperfusion and ischemia, intercostal artery embolization, thrombosis, and post-ischemic inflammatory processes [2,4,6-8].

Here, we present a case of BSS after thoracic endovascular aortic repair (TEVAR) for Stanford type B aortic dissection. The medical ethics committee waived formal Institutional Review Board approval, and the patient provided written informed consent to publish this case report.

CASE

A 49-year-old male presented to the emergency department with acute tearing pain between the scapulae, connected to respiratory movements. His medical history

included hypertension treated with several oral antihypertensive drugs and smoking.

At presentation, the patient had stable respiration, 95% blood oxygen saturation without additional oxygen, and there was no tachypnea. Auscultation revealed clear lungs. His blood pressure and heart rate were 171/105 mmHg and about 80 bpm, respectively. He, however, reported pains in the left hypochondriac region.

The patient was suspected of having a pulmonary embolism. Surprisingly, computed tomography (CT) showed no pulmonary embolism but a Stanford type B aortic dissection from the left subclavian artery to the level of the 11th thoracic vertebra. The patient was initially admitted to the intensive care unit for conservative treatment with intravenous labetalol, nicardipine, and additional oral antihypertensives to regulate his blood pressure. Intravenous sufentanil was also administered to the patient for analgesia. The target systolic blood pressure was <110 mmHg with adequate urinary output (0.5 mL/kg/h). Despite this, one day later, he experienced more abdominal pain. A repeat CT scan revealed an increase in the diameter of the thoracic aorta from 32 to 39 mm and an intramural hematoma at the level of the 5th thoracic vertebra (Fig. 1).

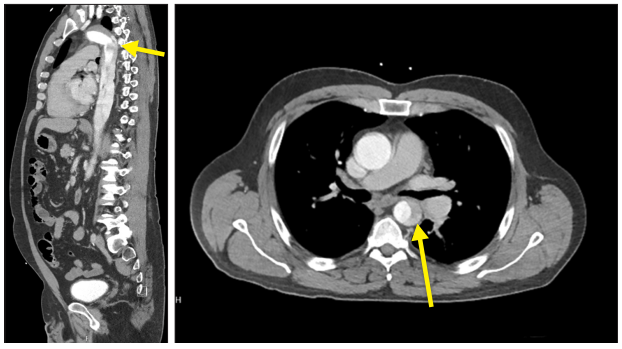

Fig. 1. Computed tomography showed a Stanford type B aortic dissection. The arrow indicates the entry tear.

Shortly after the CT angiography, TEVAR was performed. The anesthesiologist performed a spinal catheterization for eventual postoperative drainage. General anesthesia was then induced for the operation. Percutaneous access was performed using ultrasonography. Two Proglide vascular closure systems (Bard Medical, Covington, GA, USA) were inserted before introducing a 22-Fr DrySeal sheath (W.L. Gore and Associates, Phoenix, AZ, USA). First, C-tag conformable 343415 (W.L. Gore and Associates) was introduced and deployed cranially from the mesenteric artery under fluoroscopy. Subsequently, a second C-tag conformable 373720 was deployed over the left subclavian artery just distal to the left common carotid artery. Ballooning was not performed. Completion angiography showed a successful placement of the TEVAR system with patent aortic, mesenteric, and renal arterial flows (Fig. 2).

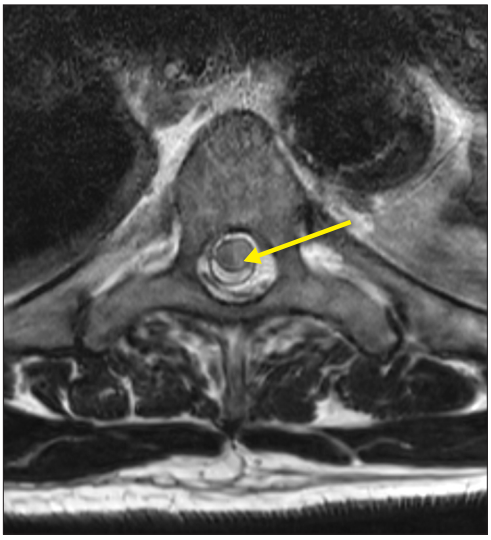

Fig. 3. Axial magnetic resonance imaging showed a left-sided lesion at the level of the 4th to 5th thoracic vertebra. The arrow points at the lesion, which is lighter than the right side, compatible with spinal cord ischemia.

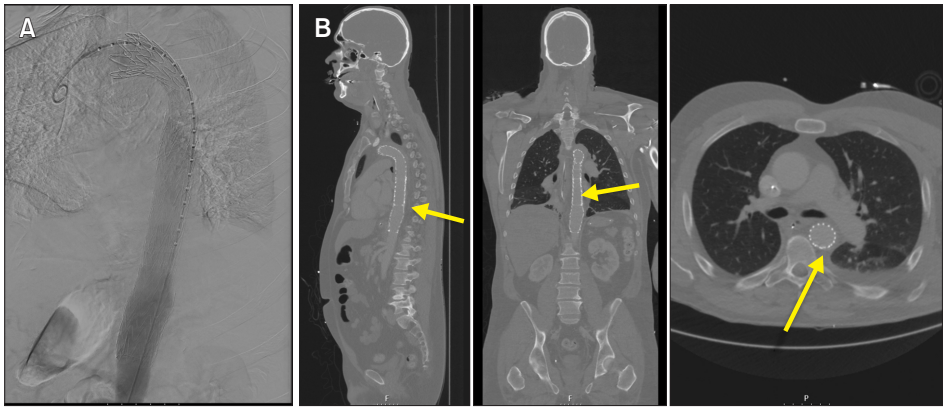

Fig. 2. Intraoperative and postoperative images after a successful thoracic endovascular aortic repair (arrow). (A) Intraoperative angiography. (B) Postoperative computed tomography scan.

When the patient awoke after surgery, he appeared to have left leg paralysis. There was impairment of tactile sensation and hypoesthesia in the left leg and lower abdomen. On the right side, there was an impairment in the temperature sensitivity. The movement of the right leg was normal. Patellar and Achilles tendon reflexes were absent in both legs, and bilateral Babinski signs were observed. The movement, sensation, and reflexes of the arms were normal. Magnetic resonance imaging of the spinal cord confirmed the suspicion of spinal cord ischemia. It revealed a unilateral ischemic lesion limited to the left-sided spinal cord at the level of the 4th to 5th thoracic vertebra, which explained the clinical BSS (Fig. 3) and was treated for induced hypertension (systolic blood pressure >180 mmHg or a mean arterial pressure >80 mmHg and spinal fluid drainage at 10 cm H_2O above the heart level). However, this did not improve the paralysis. After 4 days, the spinal catheter was removed, and the patient was transferred to a rehabilitation clinic specializing in spinal cord injuries. One year after the initial TEVAR, he was able to walk again with support and carry out activities of daily living again.

DISCUSSION

BSS is seen in patients with incomplete spinal cord injury and is confined to one-half of the spinal cord. The neurological signs are bilateral because some spinal tracts cross within the cord while others cross in the brainstem. There is ipsilateral paralysis and loss of proprioception, touch and vibration, and contralateral loss of pain and temperature sensation below the lesion [1-5,9-11]. BSS is most often caused by penetrating injuries, multiple sclerosis, or lateral compression from tumors. A vascular cause is very rare because large vascular territories are not hemi-laterally divided. Few cases with a vascular origin have been described [9-11], mostly after endovascular procedures [1,3,5], including 1 case after the TEVAR procedure [2].

Endovascular surgery has developed significantly and has been used for various indications. TEVAR is especially suitable for high-risk patients with multiple comorbidities. Its effectiveness and minimal invasiveness have now been well recognized [6-8,12]. Despite the advantages of the endovascular approach, major complications occur in approximately 5% to 25% of all cases, including mortality, spinal cord ischemia, and vascular injury [13]. Spinal cord injury after TEVAR has a 3.6% to 12% incidence and usually presents with paraplegia or paraparesis [12]. Of the full spectrum of spinal cord injuries, BSS after TEVAR is very rare [2]. Potential risk factors for spinal cord ischemia include: 1) extensive coverage of the aorta; 2) iliofemoral vessel injury; 3) sacrifice of the left subclavian artery; and 4) systemic arterial thromboembolism and previous repair of an abdominal aortic aneurysm [12,14]. In our patient, the most likely pathogenesis of BSS was hypoperfusion or micro-embolisms during the TEVAR, resulting in a spinal cord infarction involving the territory of one of the sulcocommisural arteries penetrating branches of the anterior spinal artery.

Treatment of spinal cord ischemia after TEVAR remains controversial. The patient was treated for induced hypertension because of the suggested spinal cord hypoperfusion. In general, the prognosis for the recovery of motor function in BSS is moderate to good. Approximately 50% to 66% of one-year motor function recovery occurs in the first 2 months after injury [15]. The initial severity of the impairment seems to be the best predictor of functional recovery [2,15].

In summary, spinal cord injury after TEVAR usually presents with paraplegia or paraparesis. BSS after TEVAR is a rare phenomenon with a good prognosis depending on the initial injury severity.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

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