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Case report

Spinal cord infarction six months after thoracic endovascular aortic repair–A case report

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ABSTRACT

Spinal cord infarction is reported to account for less than 1% of all strokes and is a relatively rare disease. In recent years, thoracic endovascular aortic repair (TEVAR) has become a common treatment for aortic aneurysms, and spinal cord ischemia is one of its complications. Most cases occur in the perioperative period; however, a few cases have been reported in the chronic stage. Here, we report a case of spinal cord infarction, 6 months after TEVAR. A 77-year-old man experienced sudden onset paraparesis following dumbbell exercises and defecation. He had a history of an infectious thoracoabdominal aortic aneurysm treated by TEVAR 6 months prior. Paralysis and disturbance of the thermal pain and tactile sensations of the lower limbs were observed, but proprioception and deep sensation were preserved. Computed tomography (CT) showed no evidence of intraspinal hemorrhage, new aortic dissection, or endoleak around the aortic stent placed from Th11 to L3. Magnetic resonance imaging (MRI) showed intramedullary hyperintensity from Th11 to the conus 2 days after onset. Anticoagulant therapy and rehabilitation were performed, and the lower-limb muscle strength gradually improved. After aortic stenting, particularly including the level of the Adamkiewicz artery, the risk of spinal cord ischemia must be monitored, because spinal circulation depends on collateral circulation.

1. Introduction

Spinal cord infarction is reported to account for less than 1% of all strokes and is a relatively rare disease. Causes include aortic dissection, aortic aneurysm surgery, and post cardiopulmonary arrest, and it may also be idiopathic [1]. Recently, thoracic endovascular aortic repair (TEVAR) has become common for thoracic abdominal aortic aneurysms. Spinal cord ischemia is one of the complications of this procedure, mostly occurring in the perioperative period, with few cases being reported in the chronic stage [2]. Here, we report a case of spinal cord infarction 6 months after TEVAR.

2. Case presentation

A 77-year-old man experienced sudden onset paraparesis after performing dumbbell exercises and defecation and was brought to our hospital. His past history was lumbar pyogenic spondylitis treated 7 years ago and infectious thoracoabdominal aortic aneurysm treated by TEVAR 6 months prior (Figure 1). Physical examination revealed complete paralysis, with disturbance of thermal pain and tactile sensation of the lower limbs below Th10, but proprioception and deep sensation were preserved. Urinary retention and constipation were observed. Computed tomography (CT) showed no evidence of intraspinal hemorrhage, new aortic dissection, or endoleak around the aortic stent placed from Th11 to L3. Magnetic resonance imaging (MRI) showed intramedullary hyperintensity from Th11 to the conus was observed 2 days after onset (Figure 2). Hematological and cerebrospinal fluid findings were unremarkable.

After admission, anticoagulant therapy and rehabilitation were started. The lower-limb muscle strength gradually improved (from American Spinal Injury Association [ASIA] grade B to C) and urination was observed with a decreased amount of retention. He was transferred to a rehabilitation hospital on the 14th day. Four months after onset, he was able to walk using a walker and was discharged (ASIA grade D).

This case report was approved by the ethics committee of Tokyo Women's Medical university. Written informed consent was obtained from the patient for publication of this case report and the use of accompanying images.

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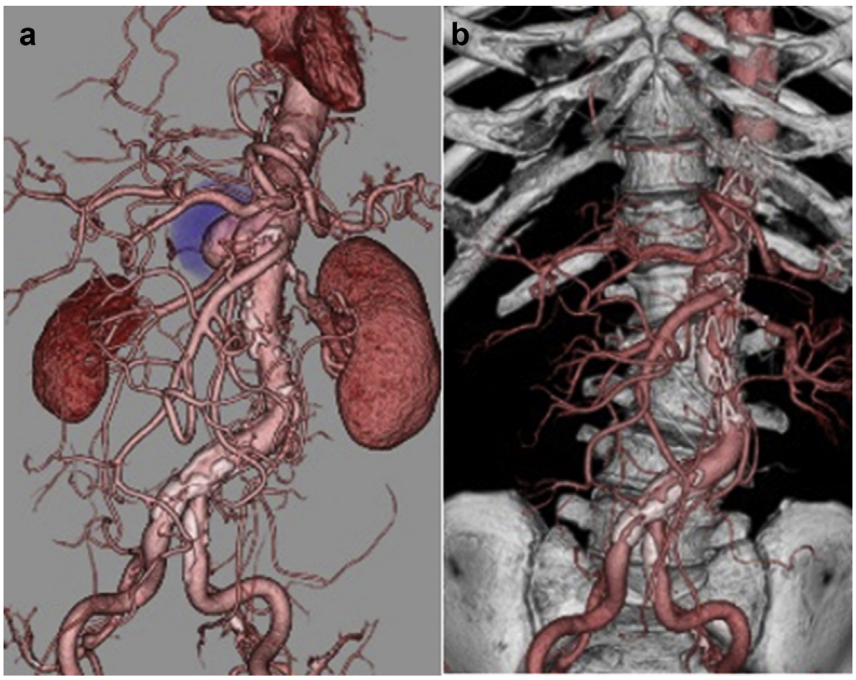

Figure 1. Three-dimensional contrast-enhanced computed tomography showing a 50-mm aortic aneurysm at L1 (a). The enhancement of the aneurysm disappeared after thoracic endovascular aortic repair between Th11 and L3 (b).

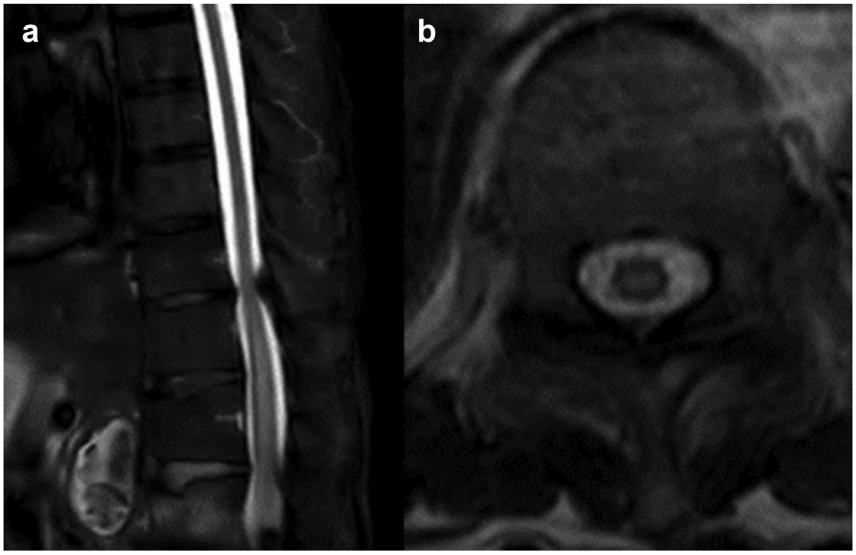

Figure 2. Sagittal (a) and axial (b) images of T2-weighted magnetic resonance imaging performed 2 days after onset showing an intramedullary high intensity area below Th11.

3. Discussion

Spinal cord infarction is less frequent than cerebral infarction due to the abundant collateral networks. However, the thoracic-spine blood flow to the anterior spinal artery originates from the Adamkiewicz artery, which derives from one segmental artery between Th8 and L3 [3]. The incidence of spinal cord ischemia after TEVAR is reported to be about 4.5% on average [4]. If TEVAR is performed, including the Adamkiewicz artery, the blood supply of the spinal cord will depend on collateral circulation. The left subclavian artery and the internal iliac artery are considered to be important for collateral circulation when the Adamkiewicz artery is occluded [5, 6].

Since a decrease in spinal cord perfusion pressure causes spinal cord infarction, maintaining mean blood pressures and controlling cerebrospinal pressures with drainage are common measures for perioperative spinal cord ischemia in cardiovascular surgery [7, 8]. Conversely, collateral circulation is considered to develop over time after TEVAR [9].

The risk of spinal cord ischemia is the highest during the 30-day perioperative period [10]. Since few reports on spinal cord ischemia after the perioperative period exist, its incidence and timing are still unclear.

Mendes reported a case of delayed paraplegia eight months after TEVAR [11]. Yamauchi reported a case of delayed paraplegia eight months after TEVAR [12]. Cho reported a case of delayed paraplegia ten

months after TEVAR [10]. They cautioned that the vulnerability to spinal cord ischemia may remain after TEVAR in the chronic phase. These case reports had episodes of hypotension triggered by general anesthesia, gastrointestinal bleeding, and diarrhea. Therefore, in order to prevent delayed paralysis, it is important to be aware of avoiding hypotension not only postoperative acute phase but also in the chronic phase.

In this case, the patency of celiac, superior mesenteric and renal arteries was confirmed at the time of TEVAR. The level of the Adamkiewicz artery was unknown, but it is highly likely that it was within the stent range. Deterioration in the spinal cord blood supply due to dehydration and hypotension was suspected as a cause for the episode of onset after exercise and defecation, and the strong atherosclerotic change of the aorta may have also reduced collateral flow.

Although it is not possible to determine whether the history of TEVAR was associated with the onset, these conditions are considered to be potential triggers of spinal cord infarction.

After aortic stenting, particularly when including the level of the Adamkiewicz artery, the vulnerability to spinal cord ischemia must be considered because spinal circulation depends on collateral circulation.

Declarations

Author contribution statement

All authors listed have significantly contributed to the investigation, development and writing of this article.

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Competing interest statement

The authors declare no conflict of interest.

Additional information

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