Delayed paraplegia after endovascular repair of an abdominal aortic aneurysm after lumboperitoneal shunting

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ABSTRACT

Spinal cord ischemia is a rare but catastrophic complication of elective endovascular abdominal aortic aneurysm repair. We report a case of delayed spinal cord ischemia after the elective endovascular repair of an infrarenal aortic aneurysm in a patient who previously underwent lumboperitoneal shunting. This case demonstrates that spinal cord ischemia could cause the inability to control spinal cord pressure and that patients who undergo endovascular aortic repair with lumboperitoneal shunting may be more vulnerable to spinal cord ischemia. This case report also suggests that spinal cord pressure can be a major contributor to spinal cord ischemia. (J Vasc Surg Cases Innov Tech 2023;9:1-3.)

Keywords: Spinal cord ischemia; Lumboperitoneal shunting; Abdominal aortic aneurysm; Endovascular procedures; Stents

Spinal cord ischemia (SCI) remains a rare but catastrophic complication of elective endovascular abdominal aortic aneurysm (AAA) repair. Although the risk factors and complex pathophysiology of SCI are being elucidated, the triggering factors remain unidentified.

Here, we describe delayed SCI after endovascular aortic repair (EVAR) in a patient who had undergone lumboperitoneal shunt (LPS) implantation 18 years ago. Patients with LPS have limited spinal cord pressure (SCP) regulation, indicating that higher SCP can impair spinal cord blood flow and increase the risk of delayed SCI.

CASE DESCRIPTION

Written informed consent was obtained from the patient for the publication of this case report. An 81-year-old woman with a history of subarachnoid hemorrhage, normal pressure hydrocephalus, and deep vein thrombosis presented with a 5-cm asymptomatic infrarenal AAA that grew by 10 mm in 4 months. Surgery was recommended as the AAA had grown by 5 mm in 6 months. We decided to conduct surgery for the patient. The patient had undergone LPS implantation 18 years ago to improve normal pressure hydrocephalus. Furthermore, this patient was prescribed direct oral anticoagulant for deep vein thrombosis.

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Author conflict of interest: none.

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The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2468-4287

https://doi.org/10.1016/j.jvscit.2022.11.015

Preoperative computed tomography revealed a fusiform aneurysm starting approximately 3 cm below the lowest (left) renal artery. The infrarenal aortic neck—considered an acceptable proximal fixation site—was approximately 30 mm and 18-19 mm in length and diameter, respectively (Fig 1). The neck angle was >60°; the aortic region from the top of the right renal artery to the bottom of the superior mesenteric artery orifice was 18 mm in length, and AAA was associated with thrombosis. Furthermore, both the left subclavian and the hypogastric arteries were patent.

As the proximal neck of the AAA was highly flexed, EVAR was initially deemed not suitable, and open surgery was considered; however, open surgery was also ruled out because of sarcopenia and poor condition of the patient.

Under general anesthesia, bilateral oblique groin incisions were made to expose the common femoral arteries, and the inferior mesenteric artery was occluded using embolization coils (Tornade 3*2*1, C-stopper 2.3*60*4). A 24 × 18 × 174 Aorfix stent graft (Lombard Medical) was deployed starting proximally at the orifice of the left renal artery and landing distally in the bilateral common iliac artery. The endograft was extended using 12-mm limb extenders to land in the right common iliac artery to secure the landing zone on the distal side. After the procedure, no endoleaks were observed, and pedal pulses were present in both extremities (Fig 2). The entire procedure lasted approximately 2 hours but intraoperative systolic blood pressure (sBP) remained low (80 mm Hg).

The patient complained of incomplete paraplegia in both limbs on postoperative day 2 (POD2) but an abdominal computed tomography scan was unremarkable and pedal pulses remained normal. A neurologist observed muscle weakness of both limbs on examination, and both lower extremities had lost muscle strength by grade 1 in manual muscle testing. As SCI was suspected, cerebrospinal fluid (CSF) drainage was considered, but placing a spinal drain would have been challenging because the patient was using direct oral anticoagulant.

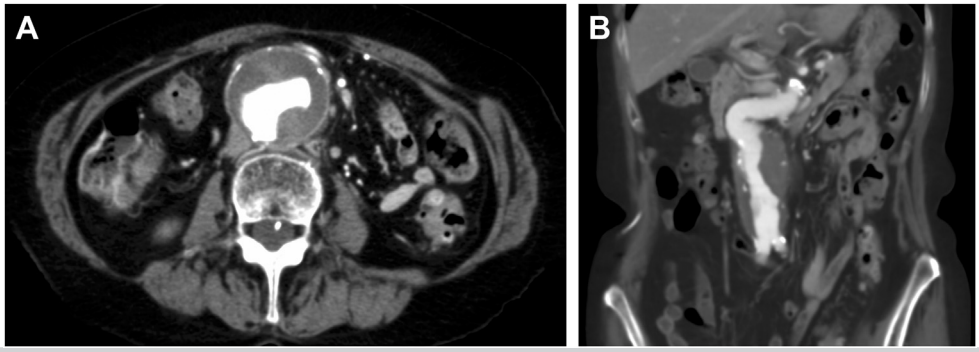

Fig 1. Preoperative computed tomography: **(A)** axial and **(B)** coronal.

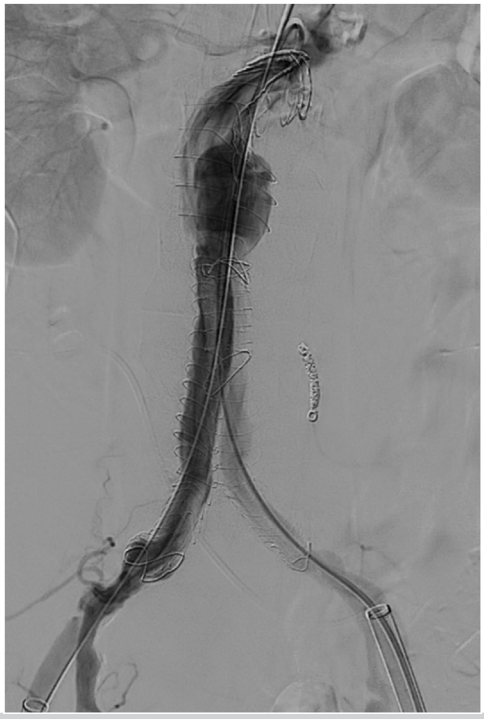

Fig 2. Completion angiogram after the insertion of the stent graft device.

In addition, as an LPS was present, the neurosurgeon recommended CSF drainage by reducing shunt pressure. We could successfully reduce shunt pressure from 80 to 60 mm Hg in the fluoroscopy room by noninvasively modifying the pressure setting of the changeable valve of the LPS using a special transmitter, which is connected to a programmer that defines the pressure of the LPS, against the LPS from the body surface.

Dramatic improvement in the neurological deficit was observed after the procedure, and muscle strength was comparable to that before the surgery. Nonetheless, for safety, we administered high-dose steroids and naloxone and used noradrenaline to maintain adequate mean blood pressure. Transfusion was not needed as her hemoglobin level was 12 g/dL. She was discharged on POD12 and has remained deficit free.

DISCUSSION

This report suggested that EVAR in patients with LPS may be associated with a higher risk of SCI and that controlling SCP is effective in treating SCI after EVAR.

SCI remains a rare but catastrophic complication of EVAR, and an analysis of the EUROSTAR database of 2862 patients undergoing EVAR showed an SCI incidence of 0.21%.[1]

Mechanisms leading to SCI after elective surgical management of infrarenal AAAs are poorly understood but are thought to be the result of several complex interactions. As the arterial network that supplies the spinal cord with blood is interrupted, the spinal cord is injured by ischemia, which increases the risk of SCI after EVAR. Further, on AAA rupture, intraoperative sBP of $<$90 mm Hg is associated with SCI risk.[2]

SCI after AAA repair is classified as immediate or delayed. The delay was reported to be 1 to 21 days postoperatively.[3] The mechanisms of delayed SCI are reperfusion injuries with the influx of inflammatory and neurochemical mediators, spinal cord edema, and arterial hypotension.[4] Moreover, temporary ischemia can cause delayed SCI in mice.[5]

In this case, SCI onset on POD2 was delayed, and as intraoperative sBP was low and AAA was associated with thrombosis, the presence of microemboli cannot be ruled out. Thus, blood supply to the spinal cord may have been decreasing, and the ischemia-reperfusion injury may have increased SCP.

LPS is a technique for diverting CSF from the lumbar thecal sac to the peritoneal cavity, and the shunt is inserted to treat the symptoms of hydrocephalus and idiopathic intracranial hypertension. To the best of our knowledge, no previous studies have described SCI after EVAR in a patient with LPS. Notably, as spinal fluid flow is regulated by a flow control valve connected between the tubes, patients with an LPS have difficulty self-regulating SCP.[6] Regarding the development of delayed SCI, in this

case, we hypothesized that the volume of draining CSF defined by the LPS would be insufficient to decrease the SCP when the SCP increased after EVAR, which subsequently led to SCI.

Thus, given the inability to autoregulate SCP, which may not be sufficiently responsive to reduced blood flow to the spinal cord, patients with LPS may have a higher risk of SCI. The resulting spinal cord edema may have reduced the spinal cord blood supply, resulting in delayed SCI.

When treating SCI, a multimodality approach is crucial, and spinal drainage is among its most critical components.[7] Moreover, several studies have suggested maintaining a high mean postoperative pressure to support blood flow to the spinal cord; hence, intravenous steroid therapy and hyperbaric oxygen therapy must be considered.[8,9] In this case, SCP was reduced after SCI onset by lowering LPS pressure and draining more CSF into the peritoneal cavity, which resulted in a dramatic improvement in symptoms. Other treatments included the use of intravenous steroid therapy, maintaining higher mean pressure, and administration of naloxone. This report showed that postoperative muscle weakness in the lower extremities should be closely monitored in patients with a history of LPS to prepare for the possibility of postoperative paraplegia and promptly adjust LPS pressure if present.

CONCLUSIONS

Patients undergoing EVAR with LPS may be more susceptible to SCI; however, as drainage via LPS improves SCI, this case indicates that SCP can be a major contributor to SCI.

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Submitted Aug 25, 2022; accepted Nov 9, 2022.