



# Paraplegia after Open Repair of Suprarenal Abdominal Aortic Aneurysm

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Spinal cord ischemia after open repair of abdominal aortic aneurysm (AAA) is an unpredictable and devastating complication. We present a case report of a patient who developed paraplegia 6 hours after open repair of suprarenal AAA. A 74-year-old man presented with asymptomatic 5.5-cm suprarenal AAA, for which he underwent open repair under general anesthesia. The paraplegia was identified 6 hours after the operation. Postoperative magnetic resonance imaging showed T2 signal hyperintensity and swelling of the spinal cord, which were consistent findings with subacute spinal cord infarction. Although intravenous steroid was administered and lumbar cerebral spinal fluid drainage was instituted, his neurological outcome did not improve. He was discharged after vigorous rehabilitation but still has paraplegia and requires wheelchair for ambulation.

**Key Words:** Paraplegia, Spinal cord ischemia, Abdominal aortic aneurysm

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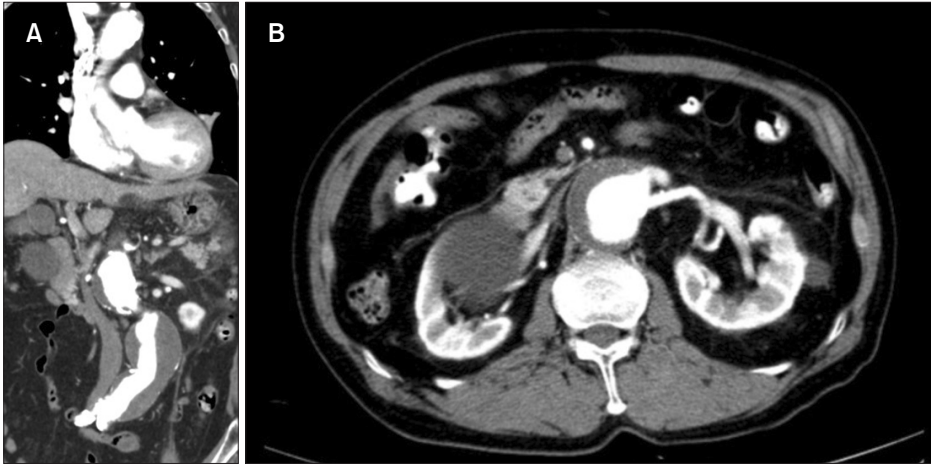
## INTRODUCTION

Postoperative paraplegia secondary to spinal cord ischemia (SCI) after abdominal aortic aneurysm (AAA) repair is an extremely rare but devastating complication. The incidence of SCI after conventional open surgery has been reported to range from 0.15% to 0.30% [1]. Although the exact mechanism is still unclear, several mechanisms of postoperative SCI after AAA repair have been proposed. These include surgical interruption of vascular supply to the spinal cord and hypotension-related SCI [1-4]. In this report, we present a very rare case of postoperative paraplegia after elective open repair of suprarenal AAA. The case report was approved by the Institutional Review Board of the Seoul National University Bundang Hospital (IRB No. B-2012-654-701).

## CASE

A 74-year-old man was admitted for elective repair of 5.5-cm suprarenal AAA. The patient had a history of 2-vessel coronary artery disease for which he had undergone percutaneous stent placement of the right coronary artery 3 years ago. A preoperative abdominal computed tomography angiography (CTA) showed AAA extending proximally up to the superior mesenteric artery (SMA) origin and distally to the aortic bifurcation. The bilateral renal arteries and the inferior mesenteric artery were patent, although the right renal artery had a severe stenosis (Fig. 1). Both internal iliac arteries were also patent without thrombus. After preoperative evaluation, the patient was deemed to be a candidate for an elective open repair.

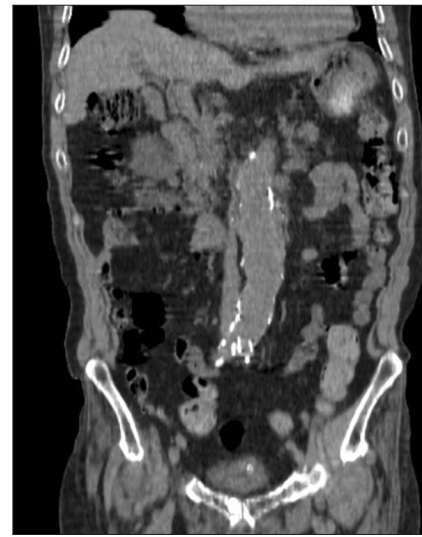
Under general anesthesia, a transperitoneal, mid-line approach was utilized. Mannitol (0.5 g/kg body weight) was administered before clamping of the aorta. Following



**Fig. 1.** Preoperative abdominal computed tomography (CT) angiography scan demonstrating suprarenal abdominal aortic aneurysm. (A) Coronal image. (B) Axial image.



**Fig. 2.** Magnetic resonance imaging scan demonstrating subacute spinal cord infarction.



**Fig. 3.** Postoperative computed tomography scan at 1-year follow-up showing no abnormal contour of aortic interposition graft.

systemic heparinization, control was obtained of the supra-SMA aorta. Bilateral renal arteries were perfused with 4°C lactated Ringer solution containing 500 mg solumedrol and 2,000 IU heparin, 240 mL to each kidney initially and then 120 mL every 15–20 minutes. Proximal anastomosis was effected with a 24 mm×30 mm Hemashield knitted polyester graft. Bilateral renal artery bypass grafting was performed using 6-mm knitted polyester grafts originating from the aortic graft. The distal aortic anastomosis was effected just above the aortic bifurcation. Heparin effects were reversed with protamine upon completion of the graft implantation. Supra-SMA aortic clamping time for proximal anastomosis was 30 minutes. Right renal artery and left renal artery anastomosis time was 15 minutes each. Distal anastomosis time was 30 minutes. Thus, right renal artery and left renal artery clamping time was 45 minutes and 60 minutes each.

Total clamping time was 1 hour and 30 minutes. The entire procedure took approximately 7 hours. The mean arterial pressure (MAP) was maintained above 70 mmHg throughout the procedure, although on occasion, the MAP dropped to as low as 55 mmHg which was treated with inotropic agent. Total estimated blood loss was approximately 3,500 mL including some low grade but persistent backbleeding from the lower lumbar arteries that was not recognized until just before the distal aortic anastomosis. After removing thrombus, we identified 4 backbleeding sites from lumbar arteries and suture ligated these vessels. A total 12 units of packed red blood cells and 10 units of fresh frozen plasma were transfused. Additionally, 1,500 mL of colloid and 6,580 mL of crystalloid were administered.

The patient was admitted to the intensive care unit under light sedation. After the sedatives wore off 6 hours

later, the patient was noted to have bilateral lower extremity motor loss, deep tendon reflex loss and sensory loss of the legs. Magnetic resonance imaging showed T2 signal hyperintensity on diffusion-weighted image and edema of the spinal cord from lower thoracic to conus medullaris (Fig. 2), consistent with spinal cord infarction. Intravenous dexamethasone, 5-mg qid, was administered immediately for 72 hours and lumbar cerebral spinal fluid (CSF) drainage instituted and maintained at 10-cm water for 5 days. However, the patient's neurological outcome did not improve and recurrent urinary tract infection occurred due to bladder dysfunction, requiring a suprapubic cystostomy. The patient was discharged to a rehabilitation facility on postoperative 4 months. Despite vigorous rehabilitation efforts after discharge, the patient never recovered from SCI and is wheelchair dependent. Postoperative CTA at 1-year follow-up showed widely patent renal and aortic grafts without any graft-related complications (Fig. 3).

## DISCUSSION

Postoperative paraplegia after suprarenal AAA repair is a very rare complication. Although there have been a number of reports on immediate and delayed postoperative paraplegia, the precise mechanism is yet to be established. It is thought to be multifactorial. Possible explanations of SCI include the interruption of spinal blood flow, especially the great radicular artery (also known as the artery of Adamkiewicz), prolonged aortic occlusion, intraoperative hypotension, atheromatous embolization, and interruption of the collaterals from internal iliac artery circulations [5,6].

In order to preserve spinal cord perfusion, it is of paramount importance to maintain stable perioperative hemodynamics. Previous studies have shown that perioperative systemic hypotension can lead to ischemic infarction of the spinal cord. Chiesa et al. demonstrated that perioperative MAP less than 70 mmHg was a significant predictor of SCI [7,8]. In our case, preoperative MAP of the patient was maintained above 80 mmHg. However, during the operation, there were intermittent periods of significant fluctuation of blood pressure. From time to time, the patient's MAP dropped to as low as 55 mmHg (69/45). It was soon recovered by intravenous injection of ephedrine by the anesthesiologist. The patient's postoperative MAP was strictly kept above 70 mmHg by continuous infusion of inotropic agents such as norepinephrine, but SCI could not be reversed. Intraoperatively, minimization of blood loss and intraoperative maintenance of stable hemodynamics with necessary blood products and avoidance of vasopressor also help mitigate the risk of SCI.

Other intraoperative factors that contribute to the de-

velopment of SCI include prolonged operative time and extended aortic clamping time, intraoperative microembolism to the spinal cord circulation [9]. Arterial and venous thrombosis of the spinal vascular tree may also cause SCI by causing the spinal cord congestion and swelling [9,10]. Although intravenous heparin is routinely administered during the procedure, the dosage and timing of the administration varies widely [11]. In our case, the initial dose was 100 IU/kg and ACT was kept above 250 seconds throughout the procedure.

Time is of essence in the detection and treatment of SCI. Few reports have addressed the importance of intraoperative neurological monitoring for early detection [12]. Jacobs et al. [13] demonstrated that motor evoked potential and somatosensory evoked potential are useful in monitoring spinal cord perfusion and adjust intraoperative strategies during thoracoabdominal aortic repair. During abdominal aortic surgery, this may not be practicable as there may be very little one can do other than revascularization of the hypogastric artery. As such, it is critical to assess the motor function of the lower extremities upon completion of the procedure before the patient is transferred out of the operating room. Even when the patient is not ready for extubation, it is essential to let patient emerge from anesthesia to rule out SCI. If SCI is suspected, 1) CSF drainage catheter may be placed immediately and kept at 10 cm of water or lower; 2) steroid may be administered; 3) hemoglobin is maintained above 10 g/dL; 4) MAP is kept above 80–90 mmHg; 5) coagulopathy should be corrected. In this case, the motor function could not be assessed for 6 postoperative hours. The window of opportunity may have been lost by the time CSF drainage and steroid were instituted.

In conclusion, SCI abdominal aortic surgery occurs rarely, the surgical team (both the surgeon and the anesthesiologist) must be cognizant of its potential intraoperatively and be ready to institute proper therapy. This includes minimizing blood loss, having blood products readily available in the operating room with rapid infuser and maintenance of normothermia to avoid coagulopathy. It is important to develop a comprehensive standardized protocol.

## CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Concept and design: HSP, TL. Analysis and interpretation: CSS, HJK. Data collection: CSS. Writing the article: HJK, TL. Critical revision of the article: HSP, JSC, TL. Final approval of the article: all authors. Statistical analysis: none. Obtained funding: none. Overall responsibility: TL.

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