Motor evoked potential-guided segmental artery revascularization during open thoracoabdominal aortic aneurysm surgery after coil embolization as a part of the minimally invasive staged segmental artery coil embolization concept

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ABSTRACT

Sacrifice of the segmental arteries during thoracoabdominal aortic repair carries the risk of spinal cord injury. Staged embolization of segmental arteries has been discussed as an option for preconditioning the spinal cord vascular network. In the present case, periprocedural monitoring of motor-evoked potentials detected spinal cord ischemia after aortic cross-clamping, although embolization of eight segmental arteries had been performed in advance. Implantation of an intercostal artery bypass restored spinal cord perfusion and normalized the motor-evoked potentials. Thus, the preconditioning strategy to stimulate creation of a spinal cord collateral network as an adjunctive method to prevent paraplegia is not perfect. (J Vasc Surg Cases Innov Tech 2022;8:206-9.)

Keywords: Aortic surgery; Embolization; Evoked potentials; Spinal cord ischemia; Thoracoabdominal aortic aneurysm

A formidable complication of treatment of thoracoabdominal aortic aneurysms (TAAAs), both open and endovascular, is spinal cord injury (SCI) with subsequent paraplegia. SCI has been understood to be mainly caused by acute disruption of segmental artery (SA) perfusion during aortic repair.1 Conventional, protective measures to maintain spinal cord function, including precise blood pressure management, routine application of cerebrospinal fluid drainage, and left heart bypass to preserve inline flow to the internal iliac arteries, have long been established. However, such protective measures have not eliminated the occurrence of SCI. In cases of open surgical repair, monitoring of motor-evoked potentials (MEP) has been proved to provide accurate information regarding spinal cord function and guiding the surgical strategies to restore SA perfusion, reducing the risk of postoperative paraplegia.”2 Another potential and currently discussed option for preventing SCI during endovascular and open TAAA repair is to activate arteriogenesis of the spinal cord vascular network through staged embolization of the SAs before aortic reconstruction.3

In the present case, implantation of an intercostal artery bypass became necessary after periprocedural MEP monitoring detected significant spinal cord ischemia, despite spinal cord preconditioning through staged embolization of eight SAs. The MEP had normalized after restoration of spinal cord perfusion, and patient’s clinical outcome was uneventful. The patient provided written informed consent for the report of his case details and imaging studies.

CASE REPORT

In 2017, a 54-year-old male patient had undergone ascending aorta and arch repair, including frozen elephant trunk (FET) reconstruction, to treat an acute type A aortic dissection. The brachiocephalic trunk and left carotid artery were reimplanted in the graft. The left subclavian artery had received an extra-anatomic bypass. The remaining dissection involved the entire descending and abdominal aorta, with distal extension into the left external iliac artery. Four years later, the maximal aortic diameter had increased to 6 cm, requiring surgical treatment. Open type II repair using the Crawford classification was planned. Before aortic reconstruction, the patient was included in the PAPA-ARTIS study (paraplegia prevention in aortic aneurysm repair by thoracoabdominal staging) and was randomized to staged coil embolization of the SAs. Because of the extent of the planned reconstruction, coiling of all accessible SAs was attempted. In three sessions, a total of eight SAs was successfully occluded, including the right-side T8 and T9 and bilateral T11, L2, and L1 (Fig 1). To allow for spinal cord recovery and avoid periinterventional SCI, each embolization session was performed after a 14-day interval, in accordance with MIS2ACE (minimally invasive staged segmental artery coil embolization) protocol.

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No peri-interventional complications were observed, and the patient’s neurologic assessment findings were normal. His type II open aortic repair was performed 4 months after the last coil embolization session. Perioperative monitoring of the MEP was performed, and a cerebrospinal fluid drain was placed. The thoracoabdominal aorta was exposed from the FET graft to the iliac bifurcation. Extracorporeal circulation was established via femorofemoral cannulation to provide retrograde perfusion of the visceral arteries, internal iliac arteries, and legs after proximal cross-clamping. After cross-clamping of the FET graft, the left kidney artery received an 8-mm Dacron bypass graft and was infused with the Custodiol HTK solution (Essential Pharmaceuticals, LLC, Durham, NC). The ostia of the superior mesenteric artery and the celiac trunk were anastomosed to a 12-mm Dacron bypass graft. The ostia of the superior mesenteric artery and the celiac trunk were anastomosed to an 8-mm polyester graft in an end-to-end fashion. Subsequently, a 15F selective perfusion catheter provided 100 mL/min of blood flow through the graft, and, after 10 minutes, the MEP were restored, finally reaching 80% of the preoperative value at the end of the procedure. The reconstruction was completed with distal anastomosis of the 28-mm Dacron graft at the iliac bifurcation and the end-to-side anastomoses of the renal artery grafts, superior mesenteric artery and celiac trunk bypass and bypass for the two SAs (Fig 3).

The early neurologic examination revealed no deficits, and the patient was mobilized at 72 hours postoperatively. During a follow-up of 8 months, no neurologic alterations were observed.

**DISCUSSION**

Preserving adequate perfusion to the spinal cord during and after open TAAA repair has remained the key aspect to avoiding SCI. However, establishing a sufficient collateral network, with consideration of the individual anatomic variations of the patient, has been challenging. The use of perioperative MEP monitoring allows for accurate detection of spinal cord ischemia, guiding the surgeon in performing revascularization of excluded critical SAs. However, this strategy can prolong the aortic cross-clamp duration, which could pose a further risk for the development of SCI. In animal experiments, staged occlusion of the SAs has been proved to fortify the spinal collateral network and lessen the severity of effects of periprocedural SA sacrifice. In the era of endovascular aortic repair, which can result in the obliteration of all SAs, a spinal network that does not depend on direct aortic perfusion is essential. Thus, our patient, within the PAPA-ARTiS trial, had undergone embolization of eight SAs in three sessions before undergoing open TAAA repair. Nevertheless, revascularization of two SAs became mandatory to restore spinal cord function. The main question raised from our experience...
is why the spinal cord collateral network was not adequate enough to maintain spinal cord function, despite sufficient time for spinal preconditioning. Thus, the theory of collateral network protection against ischemia will not apply to all patients requiring TAAA repair. The reports of spinal cord dysfunction due to
compromised radiculomedullary perfusion resulting from compressive mechanisms have underlined the dependence of spinal cord territories on dominant intersegmental arteries. Theoretically, watershed structures, anatomically known in brain and spinal cord vasculature, will not allow for cross shed blood flow. The zone at risk of ischemic injury mediated through the watershed mechanism is located at the junction of the anterior and posterior spinal arteries—the anastomotic basket of the conus medullaris. Segmental perfusion of the anterior and posterior spinal arteries will be rendered, in most cases, predominantly through the artery of Adamkiewicz and the great posterior radiculomedullary artery, respectively. However, the small caliber of these arteries (range, 0.5-1 mm) and their substantial anatomic variability have made their identification using preoperative imaging studies practically unattainable and, therefore, poses a challenge for their successful embolization. However, their functional importance can be reliably assessed intraoperatively using MEP monitoring, which was demonstrated in the present patient. The remaining two patent small arteries at T12 still contributed significantly to spinal cord perfusion, which had been proved by restoration of spinal cord function after revascularization of these two arteries.

CONCLUSIONS
Perioperative neurologic monitoring of the spinal cord remains an essential tool to safeguard spinal cord perfusion during open TAAA reconstruction. As demonstrated in our report, MEP-guided reimplantation of intercostal arteries could help to prevent postoperative paraplegia even for patients who had previously undergone staged segmental artery coil embolization.

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