Editorial

Spinal cord protection: lessons learned from endovascular repair

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Multi-disciplinary expertise is required for both open and endovascular repair of extensive thoracoabdominal aortic aneurysms (TAAAs) to minimize mortality and disabling complications. Regardless of the approach, these procedures are technically challenging. Spinal cord injury (SCI) is among the most feared post-procedural complications, especially when resulting in permanent paraplegia, which can significantly reduce patient quality of life and survival. The risk of SCI following fenestrated-branched endovascular aortic repair (FB-EVAR) varies widely in the literature, with some reports showing rates as high as 40% (1). The extent of aortic coverage directly relates to the risk of SCI. Still, potential reasons for the variation in reported rates include the lack of reporting standards, patient heterogeneity, and differences in the use of preventive strategies. The consideration of pathophysiology is crucial in SCI, as it encompasses various factors. Although hemodynamic compromise is undoubtedly a significant factor, other factors, such as embolization and hemorrhagic complications resulting from spinal drain placement also contribute significantly to SCI.

Standardized preventive protocols that include staging, cerebrospinal fluid (CSF) drainage (routine or rescue), lower limb reperfusion, and temporary aneurysm sac perfusion (TASP) have been the primary focus of contemporary reports (2). The goal of these management protocols is threefold: (I) to optimize spinal cord perfusion while reducing metabolic and oxygen demands; (II) to detect neurologic deficits early on (patients should have clinical examination every 4–6 hours for the first 24–48 hours. Any changes in lower extremity strength/sensibility should be reported immediately); and (III) to enable the prompt rescue of patients with established SCI. To increase spinal cord collateral network perfusion pressure, permissive hypertension is commonly employed. Additionally, CSF drainage is utilized to reduce the CSF pressure that may arise due to cord edema.

CSF drainage practices are constantly changing across different medical centers. These practices range from non-selective prophylactic use in all patients to selective use in higher-risk patients and no prophylactic drainage with therapeutic drainage for postoperative SCI rescue. Despite significant variations in physician practice, the European and American TEVAR guidelines currently favor the selective use of CSF drainage (3-5). In the case of TAAAs, recommendations are evolving and based on practices of large volume centers. However, there is an argument for avoiding routine CSF drainage due to the increased risk of drain-related complications among endovascular repair patients, such as intracranial hemorrhage and spinal cord hematomas (6-8). A small multi-center pilot study is currently ongoing to determine the feasibility and processes for a larger, prospective randomized controlled trial comparing prophylactic with therapeutic CSF drainage (ClinicalTrials.gov Identifier: NCT04941157). Hopefully, the trial will provide answers on the efficacy of prophylactic drainage in preventing SCI and therapeutic drainage in rescuing SCI symptoms. Our current practice has progressed to utilize therapeutic drainage rather than prophylactic CSF drainage. The use of therapeutic drainage

is reserved for patients experiencing progressive SCI symptoms that do not improve with simple blood pressure augmentation. It is crucial in these cases to have a team immediately available to place a CSF drain promptly.

The staging was justified by translational and clinical research that initially considered the spinal cord's perfusion to be mainly influenced by terminal segmental aortic branches like the artery of Adamkiewicz. However, a deeper comprehension of the intricate and dynamic collateral network emerged from animal studies and clinical anatomical imaging following extensive open surgical repair (9). The collateral network's comprehensive elements rely on various factors, such as the axial network of small arteries in the spine canal, paravertebral tissue, and musculature, which interconnect and provide tributaries to the spinal cord. Additionally, contributions from not only segmental intercostal and lumbar arteries but also vertebral (cephalic input) and hypogastric arteries (distal input), as well as vessel remodeling and reorientation of flow within the collateral network from one source to another upon reduction of selective inflow source, play a crucial role. Consequently, the collateral network offers some degree of adaptation to the loss of individual contributors to perfusion, leading to dysfunction only when a certain threshold is exceeded (10).

In the clinical setting, multiple strategies have been suggested to achieve staged occlusion of segmental aortic branches, such as proximal thoracic aortic repair (TEVAR), temporary aneurysm sac perfusion (TASP), and Minimally Invasive Staged segmental Artery Coil Embolization (MIS^{2}ACE), which consist of a first stage based on embolization of the segmental arteries of the thoracic or abdominal levels (2). However, a drawback of these approaches are the potential risks of intervening rupture between procedures and morbidity associated with multiple procedures. In a comprehensive multi-center (for consistency) observational study of patients who received treatment for Extent I to III TAAAs, it was observed that elective FB-EVAR resulted in a low overall composite all-cause mortality and/or permanent paraplegia (5%) during the first 30 days of hospitalisation (11). Additionally, a multistage approach led to a 52% reduction in the primary endpoint. After adjusting for variations in baseline clinical and anatomical characteristics, staging improved patient survival by 29% at the 3-year follow-up (11). Our preference is to proceed with coverage of the proximal thoracic aorta from the landing zone to just above the celiac axis, leaving a distal Ib endoleak with completion repair in 6–8 weeks or earlier in patients with rapid expansion of very large aneurysms that are suitable candidates for off-the-shelf devices. If there are persistent changes in neuromonitoring upon completion of the procedure and the restoration of the lower extremity and pelvic flow, we use the strategy of TASP by leaving the contra-lateral gate of the bifurcated device or one of the mesenteric branches unstented.

In conclusion, SCI remains a devastating complication of aortic surgery, even in the endovascular era. Several approaches have been described to mitigate these risks, including some that are applicable to endovascular procedures. Both experimental models and clinical scenarios suggest that staging repairs of TAAA provide a protective advantage against the development of SCI. Moreover, meticulous attention to perioperative management, including intraoperative neuromonitoring, may help prevent or limit the severity of this complication. Finally, we emphasize the importance of a dedicated institutional protocol and adequate critical care to facilitate prompt diagnosis and intervention, which can help reverse or limit the severity of neurological deficits.

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Footnote

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