Neurologic Protection During TEVAR

Reducing the incidence of devastating neurologic complications
and minimizing their impact when they do occur.

BY SHARIF H. ELLOZY, MD

In recent years, thoracic endovascular aneurysm repair (TEVAR) has become the first-line therapy for fusiform aneurysms of the descending thoracic aorta. The development of adjunctive procedures, such as hybrid reconstructions, snorkels, chimneys, and fenestrated and branched devices, has allowed practitioners to extend the indications for therapy even further. TEVAR offers clear benefits over traditional open repair in terms of reduction in blood loss, decreased postoperative pulmonary dysfunction, and faster return to normal function. However, certain hurdles remain. Despite the less-invasive nature of endovascular therapy, stroke and spinal cord ischemia (SCI) continue to be a concern. The ability to preoperatively identify patients who are at risk for stroke and SCI and then institute measures to minimize the risk of these potentially devastating complications is central to optimizing TEVAR outcomes.

STROKE AFTER TEVAR

Stroke rates after TEVAR vary considerably. Most large series report an incidence ranging anywhere from 3% to 7%.[1] Strokes can present immediately or within the first few days after TEVAR.[2] The vast majority of strokes are ischemic, and most are believed to be embolic, resulting from the passage of large devices through the arch. Hypoperfusion from coverage of the arch vessels is a rare cause of stroke; however, certain anatomic variants need to be identified preoperatively if coverage of the left subclavian artery is planned. A dominant left

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vertebral artery or an incomplete circle of Willis should lead one to consider subclavian revascularization before TEVAR.

Ischemic embolic strokes can be seen in the anterior circulation, posterior circulation, or both.[2,3] Although the sample size reported by Ullery[2] et al was small (20 strokes), posterior strokes after TEVAR seem to have a worse prognosis than anterior strokes. Risk factors for stroke can be anatomic or systemic. Anatomic factors include the grade of the arch,[4] the location of proximal coverage, and the degree of atheroma in the arch.[5] Some authors have identified coverage of the left subclavian artery as a risk factor for stroke,[6] although this has not been universally reported.[7] Systemic risk factors for stroke after TEVAR include previous stroke history, age, chronic renal insufficiency, and female sex.[2,8] A review of the American College of Surgeons National Surgical Quality Improvement Program database has

shown that emergency procedures have a higher stroke risk than elective procedures. The same authors identified the need for a proximal cuff as a risk factor for perioperative stroke.[6]

Preoperative planning is central to decreasing the likelihood of stroke, as appropriate case selection is one of the most important factors in avoiding embolization. Assessment of the degree of atheromatous debris in the arch, the tortuosity of the arch, and the suitability of the proximal seal zone is central to choosing appropriate patients. Meticulous intraoperative technique, with a focus on minimizing unnecessary manipulation in the arch, is also essential. Simple maneuvers, such as the use of precurved stiff guidewires designed for the arch, may be helpful. Accurate imaging is necessary to allow for precise device deployment and to minimize the need for proximal extension cuffs.

The role of subclavian revascularization in preventing stroke is unclear. Although coverage of the left subclavian is a risk factor for stroke, a universal policy of subclavian revascularization has not been shown to clearly reduce the incidence of stroke. Most of the studies investigating the role of left subclavian revascularization have been retrospective, and there has been heterogeneity in terms of the use of a selective versus universal policy of subclavian revascularization.[1,6] As such, no clear benefit in terms of stroke prevention has been shown with left subclavian revascularization; however, many operators have a low threshold for subclavian revascularization, as it may offer protection against SCI.

SPINAL CORD ISCHEMIA
Pathogenesis
In contrast to stroke after TEVAR, the etiology of SCI is usually thought to be due to hypoperfusion. The concept of a single dominant artery to the anterior spinal cord was originally described in 1882 by Albert Adamkiewicz.[9] Classically, the artery of Adamkiewicz is described as arising directly off of the aorta in the lower thoracic or upper lumbar region, and it is identified by its characteristic hairpin turn. The importance of a single crucial artery has diminished, and the contribution of collateral vessels (particularly the vertebral artery and the internal iliac arteries) has been recognized. Animal studies have demonstrated the extent and plasticity of the collateral network to the spinal cord.[10,11] The perfusion at the level of the spinal cord can be measured directly in animal models as a collateral network pressure. Experimental models demonstrate that the collateral network pressure at baseline is a fraction of the mean arterial pressure.[12] As the spinal canal is an enclosed space, spinal cord perfusion pressure is equal to the collateral network pressure minus the cerebrospinal fluid (CSF) pressure. Measures designed to prevent SCI are predicated on that simple equation. Hence, this explains the potential impact of CSF drainage on maintaining spinal cord perfusion pressure.

Risk Factors for the Development of SCI
Current recommendations for the prevention of SCI are largely extrapolated from the experience with open thoracoabdominal aortic aneurysm repair. However, the risk of permanent ischemia after TEVAR appears to be less than that of comparable open repairs. In a recent meta-analysis comparing TEVAR with open surgery for descending thoracic aortic aneurysms, the overall incidence of SCI after TEVAR was 3.4% compared to 8.2% after open surgery.[13] As such, not all of the recommendations for open repair necessarily translate to TEVAR. In order to optimize outcomes, it is important to identify patients who are at high risk for SCI after TEVAR and then assess the efficacy of the different available treatments.

The extent of treatment and the number of stent grafts used are perhaps the most consistently identified risk factors for the development of SCI.[3,14] Coverage of the left subclavian artery without revascularization has been identified as a risk factor. A meta-analysis of subclavian artery coverage during TEVAR revealed that coverage of the left subclavian artery increased the risk of both stroke and SCI. Revascularization of the subclavian seems to offer some protection against SCI, but not against stroke.[6] Previous repair of the infrarenal aorta has been shown to be a risk factor for the development of SCI after TEVAR, with an incidence of 12% to 14%.[15,16] Preoperative renal insufficiency, older age, and longer operations may also contribute to the development of SCI.[14,16]

Preventive Measures
As previously mentioned, spinal cord perfusion pressure is the collateral network pressure minus the CSF pressure. Strategies to prevent SCI are based upon this equation and fall into two basic categories: (1) augmentation of the collateral network pressure and (2) CSF drainage in patients believed to be at risk for SCI. Care should be taken to avoid hypotension intraoperatively and postoperatively in all patients undergoing TEVAR. In patients thought to be at high risk for SCI (namely, those with extensive coverage of the aorta, previous replacement of the abdominal aorta, and preoperative renal insufficiency), further adjuncts may be necessary. Left subclavian and internal iliac artery perfusion should be preserved as much as possible, with subcla-

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CSF drainage should be considered, although unlike with open thoracoabdominal aortic aneurysm repair, there is no consensus concerning the routine use of CSF drainage in TEVAR. This is largely due to the heterogeneity of the literature reporting the use of CSF drainage in TEVAR. A recent Cochrane review of the available literature on CSF drainage and TEVAR found a pooled SCI rate of 3.2% with routine placement versus 3.47% with no drain placed. This can be compared with a rate of 5.6% in studies reporting a selective policy.[17] However, the authors believed that the quality of the reported literature was only moderate, and few data regarding complications of CSF drainage were reported. Thus, despite the fact that there is level I evidence supporting the use of CSF drainage in open thoracoabdominal aneurysm repair, they could not state with any certainty that CSF drainage was of benefit in TEVAR. On the basis of this lack of evidence, a policy of selective CSF drainage in patients who are at high risk for SCI is probably reasonable.

In patients who have concomitant thoracic and abdominal aortic aneurysms, the repairs should be staged. Experimental models have shown that the collateral network dilates within several days after segmental artery sacrifice.[10] This corroborates the clinical finding from open thoracoabdominal repairs that staged replacement of the thoracic aorta has a lower risk of SCI than replacement of the same extent of aorta all at once.[18] Experimental work in a porcine model has demonstrated that staging endovascular coverage of the thoracic aorta in pigs that have undergone infrarenal aorta replacement also reduces the risk of SCI over simultaneous repair.[19]

Should a patient develop SCI in the postoperative period, rapid diagnosis and treatment is essential. Raising the blood pressure pharmacologically and immediately placing a lumbar drain can often dramatically reverse the clinical findings. Correcting any anemia to increase the oxygen-carrying capacity of the blood, as well as optimizing the cardiac output, may be of benefit as well. The benefit of corticosteroids is not clearly defined, although most clinicians will use them to decrease the edema around the spinal cord.

CONCLUSION

Stroke and SCI continue to be some of the most devastating complications of TEVAR. The key to minimizing their impact is identifying high-risk patients and adopting strategies to decrease their occurrence. Operators should have a low threshold for subclavian revascularization in patients at risk for SCI, and a policy of selective CSF drainage is probably reasonable in high-risk patients. If a patient should develop delayed SCI, early recognition and intervention with CSF drainage and elevation of blood pressure can potentially reverse the clinical course. ∎

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