Hyperperfusion Syndrome After Intracranial Angioplasty and Stent Placement

To the Editor:

Rezende et al.1 incorrectly state that there is no prior report in the literature of hyperperfusion syndrome after stent revascularization of the intracranial vertebral artery. In fact, a nearly identical case was published with imaging proof of hyperperfusion based on cerebral blood flow (Figure) nearly 6 years ago in Neurosurgery.

In July 2000, authors working at the University of California, San Francisco, were the first to publish a series examining the incidence of intracranial hemorrhage and cerebral hyperperfusion syndrome after cervical and cerebral stent placement.2 In our series of 140 patients at that time, the incidence of cerebral hyperperfusion was 5%, whereas the incidence of intracranial hemorrhage was 1.4%, but with 0% mortality. We also reported on patients with clinical or radiological manifestations of cerebral hyperperfusion syndrome without intracerebral hemorrhage, something the current authors did not attempt. Indeed, we observed the major contribution to the 5% incidence of cerebral hyperperfusion syndrome were these cases.

The incidence of intracranial hemorrhage after surgical carotid endarterectomy is ~0.6%. In agreement with Rezende, this risk is increased by the presence of profound cerebral ischemia with impaired hemodynamic reserve.3 However, Rezende fails to indicate that other reports examining the incidence of cerebral hyperperfusion syndrome using perioperative transcranial Doppler studies have reported rates of up to 9%.4,5 Because the clinical findings in cerebral hyperperfusion syndrome may be subtle and ancillary investigations examining postprocedural cerebral blood flow, such as brain perfusion CT, nuclear medicine single-photon emission CT, xenon CT, and transcranial Doppler sonography, are not often performed, one may expect a significant reporting bias that can skew the published data.

Normal systemic blood pressure (“normotension”) after a revascularization procedure is often excessive blood pressure (effective “hypertension”) in the susceptible cerebral circulation. Already a low resistance system, the brain can sustain severe barotrauma because of the loss of cerebrovascular tone and reactivity. Our patient detailed in the Figure sustained a 100% increase in cerebral blood flow after stent revascularization meeting the specific definition of hyperperfusion. In general, pharmacologically induced hypotension is required in these cases while taking into account comorbidities which could be exacerbated by hypotension. In the future, imaging may better guide pharmacotherapy on an individualized patient basis.

Anticoagulation in the treatment of cerebral artery stenosis remains controversial, but one thing is clear: strong or multiple anticoagulants can cause cerebral hemorrhage. In both retrospective and prospective WASID studies, hemorrhagic complications substantially offset the benefit of anticoagulation for patients with posterior circulation stenosis.6,7 The WARSS trial found no benefit to warfarin over aspirin.8 Similarly, platelet inhibition must be used judiciously to balance the need to prevent stent thrombosis with the risk of hemorrhage. In carotid stent series using abciximab (a strong glycoprotein IIb/IIIa platelet inhibitor), there was a significant risk of cerebral hemorrhage.9,10 Although some authors advocate continued heparin infusion after neuroendovascular stent procedures, the coronary literature indicates little effect on the rate of stent thrombosis but a significant risk of hemorrhagic complications.11,12 Consequently, we use relatively smaller doses of heparin during revascularization procedures in which patients already take aspirin and clopidogrel, and we do not continue heparin after conclusion of the procedure.

With the advent of the Boston Scientific Wingspan system, and others like it, we are entering a time when routine stenting of cerebral arteries becomes possible. It is incumbent on the greater neurosciences community to maximize our knowledge based on the available case material in order to advance the field. We cannot afford to reinvent the wheel on a periodic basis. Our neurological colleagues luxuriate in a relative abundance of treatable disease. To their credit, however, cardiology has masterfully engineered trial after trial to evaluate drugs, devices, and techniques. Neurosciences need to follow this example to advance our discipline at a commensurate rate.

Disclosures

None.

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Cerebral hyperperfusion after stenting in a 77-year-old man with crescendo vertebrobasilar insufficiency, despite heparin anticoagulation and the use of antiplatelet agents. A, Angiogram, demonstrating preocclusive (>90%) narrowing of the right vertebrobasilar junction. The left vertebral artery was occluded in the neck, and it was reconstituted by retrograde filling from the vertebrobasilar junction. B, Angiogram, after stenting, showing that the vertebral and basilar arteries are widely patent. C, Xenon-enhanced CT scan, before treatment, demonstrating decreased cerebral blood flow of 28 to 30 mL/100 g tissue per minute (arrowheads). D, 36-Hour post-treatment xenon-enhanced CT scan, showing quantitatively elevated perfusion of the posterior circulation with relative cerebral blood flow of 61 to 66 mL/100 g tissue per minute (arrowheads).
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Stroke. 2006;37:2210-2211; originally published online August 3, 2006;
doi: 10.1161/01.STR.0000237208.77716.1c
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/37/9/2210