Role of Antiplatelets in Carotid Artery Stenting

To the Editor:

We would like to compliment the authors of the article “The Role of Antiplatelet Therapy in Carotid Stenting for Ischemic Stroke Prevention” by Drs Chatuvedi and Yadav. They presented a much needed, comprehensive overview of the role of adjunct antiplatelet therapy in carotid artery stenting (CAS) and emphasized the proliferation of endovascular, stent-supported, carotid balloon angioplasty as a reasonable alternative to conventional surgery, carotid endarterectomy, in certain high-risk patients with both symptomatic and asymptomatic disease. Similarly, they projected, and we agree, this proliferation will soon superecede carotid endarterectomy surgery in the United States for stroke prevention because the numbers of trials have become extremely numerous since 2000.

The authors state that periprocedural dual antiplatelet therapy with aspirin (ASA) and clopidogrel has been accepted as the standard therapy in CAS mainly attributable to the experience gained in percutaneous coronary interventions in addition to the outcome noted in CARESS and CURE studies. This has occurred despite the former study including only 108 patients, and the latter actually had nonstatistical significance in the stroke outcomes.

Two observations within this article captured our attention. The first was the authors’ mention of ASA+extended release dipyridamole (ER-DP) as almost an aside as an antiplatelet agent that may be used periprocedurally to have a positive impact on 24-hour and 30-day neurovascular events. We have recently performed an analysis of our patients who had CAS and received ASA+ER-DP. All were high risk (diabetic, hypertensive, and/or dyslipidemic) symptomatic patients. At the time of this writing, we have identified 38 cases of CAS procedures with patients on ASA+ER-DP periprocedurally with up to 4 years of follow-up. There were no procedural-related deaths, strokes, or transient ischemic attacks noted at 24 hours and at 30 days. Although ASA and clopidogrel appear to be an acceptable adjunct antiplatelet regimen in CAS, we do not have any evidence-based data to suggest it is the most ideal regimen. ASA+ER-DP has shown superiority in reducing event rates in patients with acute ischemic neurovascular syndromes with few side effects. Based on our experience with CAS, this regimen certainly warrants some consideration as an alternative, if not the regimen of choice in CAS. Clearly a head-to-head prospective randomized clinical trial is warranted.

The second glaring point was that although intravenous GPIIb/IIIa inhibitor was reported as having some potential benefit in reduction of acute neurovascular events, its use was discouraged because of the reported increase in bleeding, particularly intracranial hemorrhages. We routinely use the intravenous GPIIb/IIIa inhibitor, eptifibatide, in all our cases. In over 400 cases, we have had only 1 intracranial bleed, and this was felt to be most likely secondary to cerebral hyperperfusion. We feel the authors may be misleading the audience in to believing that all GPIIb/IIIa inhibitors may be detrimental when actually there are some that may prove to be a definite asset if properly used. Again, there is need for a carefully designed, well controlled, prospective trial to clearly answer this question before abandoning a therapeutic, pharmacological strategy that may be of significant benefit.

Disclosures

None.

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*Stroke*. 2007;38:14; originally published online November 16, 2006;
doi: 10.1161/01.STR.0000251691.91522.04

*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/38/1/14

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