Carotid Artery Stenting: Verdict Still Pending

To the Editor:

In their recent article, Derdeyn et al1 reviewed existing data for carotid artery stenting (CAS) in patients with asymptomatic carotid stenosis and concluded that there is no role for CAS in patients with low surgical risk and no proven benefit from carotid endarterectomy (CEA). These conclusions are categorical and strong against CAS without any possibility of “apellation” but are based on confusing data selected from literature. There are many unresolved questions in this article.

The authors exclusively analyzed results from nonrandomized industry-sponsored investigational device exemption (IDE) studies, most of which specifically focusing on high-risk subgroups (ARCHeR, BEACH, SECurity). These studies were reported as existing data about CAS in “low surgical risk patients” and they are potentially biased by nonrandomized design or commercial interest. The only data on real low-risk patients were those reported from the CREST study. Unfortunately, these are related exclusively to the lead-in phase of this large ongoing trial and, consequently, reliability is poor. Waiting for the results of ongoing randomized trials, it is more realistic to assess the risk of CAS in low-risk patients by reporting the evidence from the several published large observational case series, multicentric experiences, or nonsponsored registries rather than by analyzing industry-sponsored studies on high-risk patients asymptomatic or not. Approximately two-thirds of the CAS population addressed in almost all studies today involve asymptomatic patients with reported stroke rates of 0% to 3%.2,3

The authors should be aware of the difficulty to generalize results of studies with selection bias. In defining high surgical risk patients, the authors arbitrarily assumed for CAS most of the same known risk factors for CEA. To this regard, most of these risk factors have been defined in trials performed in the 1990s and are today overcome by advances in surgical techniques and medical therapy. The same criteria to define most of these risk factors poorly apply to currently treated CAS patients.

The authors assumed the same degree of risk reduction by CEA in asymptomatic patients obtainable by smoking cessation or modest control of hypertension (number needed to treat=18). However, these degrees of risk reduction are not comparable since detected in observational studies on patients without carotid stenosis.4 Misleading conclusions could be drawn by suggesting low risk in asymptomatic patients with contralateral carotid occlusion on medical therapy; stroke risk as high as 33% at 5 years was reported.5

Differently from CEA, there are no data to support the high CAS risk in women. The only study showing worse outcome in women for CAS is the SPACE trial in which females represented less than one-third (28%) of symptomatic patients.6 The lack of gender differences in stroke risk after CAS is largely supported by literature, including the same high-risk nonrandomized registries analyzed by the authors and the recent 2007 consensus document on carotid stenting.6

The challenge of CAS as a less invasive technique in patients with carotid stenosis is still a matter of debate and cannot be judged by this incomplete literature analysis. CAS outcome is strongly affected by patient selection, material evolution, and operators training.7 These points are not outlined in the article, which exclusively focused on high-risk asymptomatic patients undergoing CAS. There are ongoing trials evaluating CAS versus CEA in symptomatic carotid stenosis and another trial (ACST II) had been designed in asymptomatic carotid stenosis. Considering that this unsponsored large multicentric trials requires an immense time and study effort of the investigators, CAS cannot be judged guilty before these results are diffused. The “verdict” is still open.

Disclosures

None.

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