Recent data suggest that use of simple MRI techniques can characterize plaque, based on composition, with a high degree of specificity. Indeed, though the authors found large nonstenotic plaques ipsilaterally in 35% of patients, such plaques were also found in the contralateral carotid artery in 15% of patients. In similarly designed MRI studies evaluating plaque composition rather than size, the prevalence of high-risk plaque ipsilateral to cryptogenic stroke was similar to the prevalence of large ipsilateral plaques found by Coutinho et al. (ranging from 22% to 38%), but the prevalence of high-risk plaque contralateral to cryptogenic stroke in these MRI studies was considerably lower (essentially 0% in these studies).7–9 Similarly, recent CTA data have emerged to suggest simple plaque density and thickness measurements may also provide insight into plaque stability.10 The study by Coutinho et al. has built upon these MRI and CTA data and provides a simple and rapid means of measuring plaque thickness on routinely acquired axial CTA images, in a method that takes a reader no more time than it does to make a NASCET stenosis measurement. This approach therefore offers a compelling alternative to more complex CTA or MRI methods.

What are the clinical implications of these findings? The authors have reported an association between large nonstenotic carotid plaques and ipsilateral acute cerebral infarction, “suggesting that nonstenotic plaque is an underrecognized cause of stroke.” The presence of an associated factor does not prove causality and, as we have noted above, causality is challenging to prove, especially in any specific patient. However, proof of causality is not needed to establish clinical benefit. All we really need to demonstrate is that there are strokes with specific associated factors for which specific treatments are of benefit as proven by clinical trials, such as endarterectomy for carotid stenosis and anticoagulation for atrial fibrillation.12,13 Thus, the value of the nonstenotic carotid plaques
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observation by Coutinho et al. will be demonstrated if it identifies a group of patients who could benefit from intensification of medical therapy, such as dual antiplatelet therapy or use of emerging cholesterol modulating agents. Demonstrating such a clinical benefit would then warrant further clinical trials evaluating carotid endarterectomy vs medical therapy alone. The potential appropriateness for endarterectomy as a treatment for nonstenosing plaques depends primarily on the stroke risk in a medically treated group, given that the surgical complication rates and subsequent stroke rates were similar for all degrees of stenosis, including <30%, in the trials for symptomatic carotid disease. Data showing a high recurrence rate on maximal modern medical therapy would provide the basis for treatment trials that could test whether patients harboring such plaques could benefit from surgical intervention.

DISCLOSURE

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