Plaque Imaging to Decide on Optimal Treatment

Medical Versus Carotid Endarterectomy Versus Carotid Artery Stenting

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KEYWORDS

• Carotid artery disease • Optimal medical therapy • Carotid endarterectomy • Carotid artery stenting • MR imaging

KEY POINTS

- Advancements in optimal medical therapy, comprising antiplatelet agents, statin therapy, and aggressive risk factor control, have substantially reduced the risk of stroke in individuals with carotid artery disease.
- Compared with the current clinical standard of attributing risk based on carotid stenosis, plaque imaging may be better able to stratify the risk for stroke, and identify those most likely to benefit from carotid endarterectomy (CEA) and carotid artery stenting (CAS).
- Although CAS is considered as an alternative to CEA, it currently has higher rates of periprocedural stroke and clinically silent hemispheric infarction. Preliminary results suggest that vessel wall imaging of plaque morphology, composition, and activity may be beneficial in planning CAS procedures, and thereby reduce periprocedural stroke.
- While evidence is increasing, larger outcome studies and randomized clinical trials are needed to confirm the value of carotid plaque imaging in deciding between optimal medical therapy, carotid endarterectomy, and CAS.

CURRENT CLINICAL CHALLENGES

Prevalence of Carotid Artery Disease

Carotid artery disease, a local manifestation of systemic atherosclerosis, is not an uncommon finding in clinical practice. Indeed, a recent study pooling individual participant data from 4 large population-based studies estimated that the prevalence of asymptomatic moderate carotid stenosis, defined as 50% or more based on duplex ultrasonography, was as high as 7.5% in the general population depending on subjects' age and sex.[1] Specifically, 2.3% of men and 2.0% women in their 60s had carotid stenosis on ultrasonographic examination, which increased to 7.5% and 5.0% for individuals aged 80 years or older. In subgroups with cardiovascular risk factors or those with established coronary or peripheral artery disease, the prevalence of carotid stenosis is increased by severalfold compared with the general population.[2,3]

In addition, the prevalence of carotid artery disease that is significant, from a histopathologic point of view, may be underestimated. Nonstenotic or minimally stenotic carotid plaques can

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harbor substantial amounts of lipid and necrotic debris as a result of outward remodeling and the geometry of the carotid bulb. Furthermore, high-risk plaque features such as intraplaque hemorrhage (IPH), thin or ruptured fibrous cap, and plaque inflammation have been found in carotid plaques that are otherwise morphologically indistinct (**Fig. 1**).[4,5] In a 2008 study, 192 subjects underwent ultrasonography to quantify the degree of carotid stenosis, and MR imaging to identify the presence of complex plaques.[6] Complex plaques were defined as having IPH or luminal surface defect on multicontrast MR images and were more frequently found in plaques with higher grades of stenosis. Of note, however, complex plaques were detected in 21.7% of arteries with only 16% to 49% luminal stenosis. Increasingly, the advent of improved techniques for vessel wall imaging demonstrates that diagnostic imaging methods that only measure stenosis underestimate plaque burden, plaque complexity, and, hence, the true prevalence of histologically significant carotid artery disease.[4,5]

Clinical Outcomes with Medical Therapy Alone

Most carotid plaques have a relatively benign clinical course. However, once carotid artery disease becomes symptomatic, it often leads to disabling or devastating stroke without preceding signs. In the Asymptomatic Carotid Surgery Trial (ACST), half of the ipsilateral ischemic strokes in patients with asymptomatic carotid stenosis greater than 60% were disabling or fatal.[7] Carotid artery disease is a major source of ischemic stroke, accounting for approximately 15% of the 795,000 incident strokes that occur annually in the United States.[8] Less known is the role of nonstenotic but high-risk carotid plaques in cryptogenic strokes, of which a significant portion may be attributable to large-artery atherosclerosis,[9,10] as discussed elsewhere in this issue.

Medical therapy plays a pivotal role in the primary and secondary prevention of ischemic strokes ascribed to carotid artery disease. The benefit of aspirin has long been recognized, and

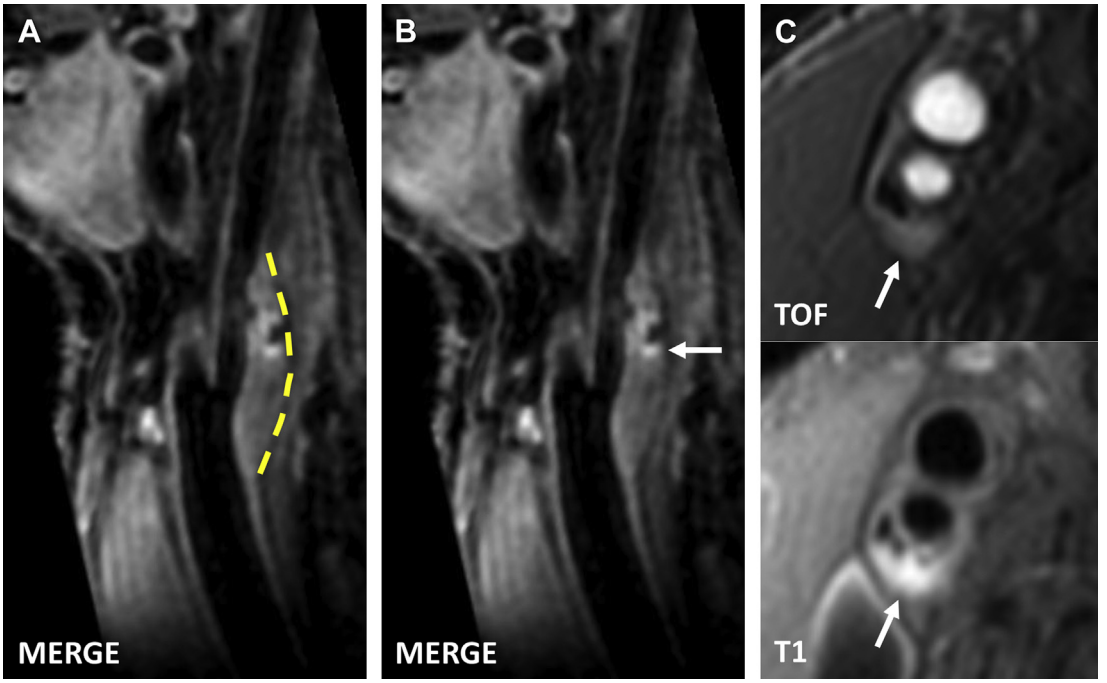

Fig. 1. Mild luminal stenosis as "the tip of the iceberg" in carotid artery disease. (*A*, *B*) Multiplanar reformat (MPR) of 3-dimensional volume data acquired using the motion-sensitized driven equilibrium prepared rapid gradient echo sequence (3D-MERGE)*, with centerlines in the common and internal carotid arteries. (*A*) A large plaque in a carotid artery with minimal luminal stenosis. The outer boundary of the plaque is delineated by the dotted yellow line. (*B*) Presence of intraplaque hemorrhage (*white arrow*) located near the outer boundary of the plaque. (*C*) Corresponding time-of-flight (TOF, *upper*) and T1-weighted (*lower*) images at the same level, confirming the presence of intraplaque hemorrhage. Plaque burden and complexity are disproportionate to the mild luminal narrowing. (* Balu N, Yarnykh VL, Chu B, et al. Carotid plaque assessment using fast 3D isotropic resolution black-blood MRI. Magn Reson Med 2011;63:627–37.)