INVITED COMMENTARY

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Management of the patient with asymptomatic carotid disease is approaching clinical equipoise. Our current guidelines are based on decades-old randomized level I trials, the Asymptomatic Carotid Atherosclerosis Study (ACAS) and Asymptomatic Carotid Surgery Trial (ACST-1), which enrolled patients between 1983 and 2003. Since then, medical therapy has intensified, particularly with the use of statins, dual antiplatelets, and more optimal antihypertensive medicines. At the same time, carotid interventions (carotid endarterectomy and carotid artery stenting) have become safer. The current Carotid Revascularization and Medical Management for Asymptomatic Carotid Stenosis (CREST-2) trial aims to answer some of these timely clinical questions.1

Increasing evidence suggests a more benign natural history for asymptomatic carotid disease than previously thought.2 An analysis of the asymptomatic carotid trials, ACAS and ACST-1, suggests that only 50 to 60 strokes are prevented at 5 years per 1000 carotid endarterectomies performed for asymptomatic disease.3 However, clinical experience shows us that a subset of patients with moderate or severe carotid disease still progress to plaque rupture and stroke in 2018.

Are there certain ultrasound characteristics that can reliably identify the asymptomatic carotid plaque at high-risk of rupture? Noninvasive imaging of plaque vulnerability remains an area of active investigation. Spanos et al associate the presence on ultrasound examination of a large juxtaluminal black area with a thin fibrous cap, a decrease in vascular smooth muscle cells, and increased macrophages, all known histologic changes of plaque vulnerability. Thinning of the fibrous cap, leading to plaque vulnerability and rupture, are events that likely occur owing to a complex interaction between local plaque and systemic inflammatory mediators, hemodynamic strain, and biomechanical forces on the plaque leading to material fatigue. Vascular smooth muscle cells volume loss and thinning of the fibrous cap likely occur owing to an array of molecular changes, which may in part be explained by a particular microRNA signature occurring both locally in the plaque and in the systemic circulation.4,5 All of these changes that seem to be associated with thinning of the fibrous cap will need to be further validated in future clinical studies. Lastly, it is possible that the significant protective effects of statin therapy against cardiovascular events6 may be due to thickening of the atherosclerotic fibrous caps.

Earlier identification of an asymptomatic patient at risk of plaque rupture could then be used to treat that patient with more intense medical therapy or with a prophylactic carotid endarterectomy (or carotid artery stenting in select cases). Can ultrasound features of plaque vulnerability, such as a juxtaluminal black area and the absence of a fibrous cap, be used to decide whether or not to intervene in a patient with asymptomatic carotid artery stenosis? Because there are no randomized studies to validate these studies, these observations are unlikely to currently be widely adopted into practice. However, this article elegantly correlates known ultrasound features with well-described histologic characteristics of plaque vulnerability. Carotid plaque noninvasive imaging will continue to evolve and help to discern the vulnerable plaque that places the patient at risk.

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REFERENCES