Beyond established criteria for carotid endarterectomy (CEA), additional plaque histologic features such as a large lipid core, fibrous cap rupture, surface ulceration, and intraplaque hemorrhage have been associated with atheroembolic stroke.1,2 It has become of great interest to identify which plaques are “vulnerable,” even though not yet ruptured (ie, at risk of becoming symptomatic or at a vulnerable stage).3 The 2-year risk of ipsilateral stroke for medically treated patients with ulcerated plaques was 73% for higher degrees of stenosis compared with 21% for all degrees.4

We report the imaging findings of high-grade symptomatic carotid stenosis. On admission, the ultrasound study did not show any feature suggestive of a vulnerable plaque except for the degree of stenosis. However, an ultrasound examination performed 24 hours later revealed acute changes in the plaque surface, leading to urgent CEA.

**Case Report**

A 66-year-old right-handed man was admitted for recurrent episodes of weakness in the left limbs and slurred speech. On examination, the patient was drowsy. He had a left facial droop and dysarthria. There was a left pronator drift, and he could not maintain the left leg against gravity. The National Institutes of Health Stroke Scale score was 12. He had diabetes, hypercholesterolemia, and bilateral internal carotid artery (ICA) atherosclerosis with an asymptomatic plaque growing on serial ultrasound studies. He was taking 20 mg of atorvastatin and 100 mg of aspirin once per day.

Head computed tomography ruled out intracranial bleeding. Diffusion-weighted 3-T magnetic resonance images (Achieva; Philips Healthcare, Bothell, WA) revealed multiple ischemic areas in the right middle cerebral artery territory suggestive of acute embolism. Three-dimensional
time-of-flight magnetic resonance angiography of the intracranial vessels was unremarkable. An ultrasound examination was performed with a Philips HD-11 system (5- to 7.5-MHz linear array probe). An extracranial color-coded duplex ultrasound examination showed a concentric isoechoic plaque with a regular surface in the right ICA and a Doppler-derived peak systolic velocity of up to 300 cm/s, suggestive of greater than 70% stenosis. The patient was clinically stable and started receiving 75 mg of clopidogrel and 40 mg of atorvastatin. The extracranial color-coded duplex ultrasound examination was repeated the following day, as routinely performed in our stroke unit for symptomatic plaque monitoring. Changes in the plaque surface on both longitudinal and axial sonograms were detected, suggestive of deep ulceration or free-floating atheroma.

Carotid computed tomographic angiography showed high-degree concentric stenosis, an irregular plaque surface, and an appendage projecting into the lumen, as suggested by lack of central contrast filling in the axial plane (Figure 1). The patient underwent emergent uncomplicated right CEA. A fibrous plaque with deep ulceration and a fresh thrombus attached was removed. Histologic examination showed fibrous cap disruption, ulceration, and infiltration of inflammatory cells dating no longer than 4 days, indicating a type 4 (complex) plaque (Figure 2). On discharge, the patient had mild dysarthria, a left pronator drift, and reduced finger dexterity. An ultrasound study confirmed patency of the right ICA.

**Discussion**

We have described a patient with recurrent episodes of left sensorimotor disturbances and high-grade right ICA stenosis on an ultrasound study performed early after admission. Treatment for secondary stroke prevention was started, and on the basis of recent data on the benefit of surgery in symptomatic carotid disease, CEA within a few days was considered. However, a 24-hour follow-up ultrasound study showed changes in the plaque surface and flow velocities suggestive of acute plaque ulceration or rupture. Thus, emergent CEA was performed without complications.

Physical disruption of a plaque, which involves tearing or fissuring of the fibrous cap, represents a critical step in the evolution of the plaque toward acute intravascular thrombus formation and sudden expansion of atheroma, eventually leading to clinical manifestations. Local inflammation in the vessel wall plays an essential role in plaque destabilization and rupture. Noninvasive imaging techniques allow in vivo identification and quantification of the characteristics of plaque instability with high sensitivity and specificity. Anechoic plaques have been associated with an increased stroke risk irrespective of the degree of carotid stenosis. Magnetic resonance imaging can define plaque surface morphologic characteristics and can distinguish between an intraplaque and a lipid-rich necrotic core with good accuracy. Computed tomographic angiography identifies plaque ulceration with high sensitivity and specificity; ulceration is more frequently detected proximally to the point of maximum stenosis, confirming the role of shear stress, as in our case. Computed tomographic angiography allows quick acquisition of the anatomic details required by surgeons, and it is well tolerated in the acute stroke setting.

In our case, histologic examination confirmed fibrous cap thinning and rupture. Combined serial ultrasound and computed tomographic angiographic studies showed an excellent correlation in the detection of acute plaque rupture and allowed us to forecast recurrent stroke and a worse prognosis. Ultrasound is well tolerated for daily monitoring of symptomatic atherosclerotic lesions in patients with recent ischemic stroke and candidates for early CEA.

Although the benefit of CEA performed within 2 weeks of symptoms onset has been widely reported, this case suggests that serial ultrasound imaging of carotid plaque morphologic characteristics in patients with acute carotid syndromes may improve patient selection and the timing of surgery. Whether appropriate triage of the patient and more rapid revascularization procedures may effectively reduce the risk of stroke recurrence remains to be clarified in large randomized controlled trials.
Figure 1. A, Extracranial color-coded duplex sonogram of the right ICA at admission showing a hypoechoic plaque (longitudinal section). B and E, Computed tomographic angiograms of the right ICA 24 hours later showing narrowing of the lumen with a protruding peduncle (arrows; axial and longitudinal). C and D, Extracranial color-coded duplex sonograms of the right ICA 24 hours later showing plaque rupture (longitudinal and transverse sections). The red line indicates the corresponding transverse plane.
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Figure 2. A, Specimen showing 70% cross-sectional narrowing of the ruptured plaque. The scant thrombotic material in the lumen suggested artery-to-artery embolization. B, Transverse section of the plaque showing ulceration of the fibrous cap (arrow). C, Micrograph showing plaque rupture (Ru) with an excavated necrotic core and discontinuation of the fibrous cap (Fc). The acute thrombus (Th) appears in contiguity with the underlying necrotic core (Movat, original magnification x2). D, High-magnification view of the site of plaque rupture (Movat, original magnification x10). E, Large inflammatory infiltrate mainly consisting of macrophagic foam cells in the fibrous cap (anti-CD68, antihuman monocytes and macrophages, original magnification x4). F, Detail of E showing numerous macrophagic foam cells at the site of fibrous cap rupture (anti-CD68, original magnification x10).
References


