Extracranial Carotid Artery Disease in Patients With Arterial Limb Embolism

To the Editor:
The relationship between embolism and thrombosis as a cause of ischemic stroke is far from established. Not all ischemic strokes in patients with a cardiac source are caused by cardiogenic embolism since cerebrovascular atherosclerosis often coexists. Thus, the clinical differentiation between thrombotic and embolic infarction is extremely difficult and often impossible. General hallmarks of cerebral embolic events have been outlined, with the presence of atherosclerotic lesions in the appropriate artery considered by most as a feature excluding cerebral embolism.

However, atherosclerotic lesions of the cervical carotid arteries have been found in 23 of 100 asymptomatic men aged 50–69 years, in 34 of 108 patients with familial hypercholesterolemia and coronary artery disease, and in 21 of 56 elderly adults with isolated systolic hypertension. Furthermore, in two different studies, the concomitance of appropriate carotid disease and potential cardiac sources of emboli illustrates the difficulties of diagnosing cardioembolism in an individual patient. In one study, the appearance together of arterial disease and potential cardiac sources of emboli was evaluated in 205 patients with transient ischemic attacks (TIAs) who underwent both angiography and echocardiography. Fifty patients had a potential source of emboli, and a carotid lesion appropriate to the TIAs was present in 38 of these 50 patients. In the work by Rem et al., in which 184 consecutive patients with TIA and stroke were studied, 59 patients with a possible cardiac source for cerebral emboli were detected. After cerebral angiography, 29 of these 59 patients also showed a vascular lesion in the appropriate carotid artery. The authors could not decide definitely which lesion was responsible for the cerebral embolus.

Since carotid atherosclerosis is so frequent in different populations, we must ask what is its frequency in patients with demonstrated cardioembolic emboli? We screened 25 consecutive patients without history of stroke admitted because of an acute arterial limb embolus for atherosclerotic lesions of the cervical carotid arteries using a high-resolution multi-gated pulsed Doppler system. There were nine men and 16 women aged 53–87 years (mean 68 years). The anatomic site of lodgement of the arterial embolus was femoral in 10 patients, popliteal in five, upper extremity in five, aortic saddle in three, and iliac in two. The sources of arterial embolism (as determined by two-dimensional echocardiography) were hypertensive heart disease with atrial fibrillation in eight patients, valvar heart disease in seven, dilated cardiomyopathy in two, mitral anulus calcification in one, and lone atrial fibrillation in seven.

Nine of 25 patients (36%) had an abnormal Doppler investigation in the common and/or internal carotid arteries. In two patients multivessel disease was observed. The degree of stenosis was considered to be important (>50% diameter reduction) in three patients.

If any of these nine patients had an arterial embolus lodged in the cerebral arteries, he or she would have been diagnosed for thrombotic stroke and no anticoagulant therapy would have been prescribed. We conclude that asymptomatic carotid artery disease is common in patients with cardioembolic events, and thus its detection should not be used as an argument against cerebral embolism diagnosis.

References

Multiple Small-Vessel Occlusions in Systemic Lupus Erythematosus

To the Editor:
There has been great interest over the past few years, recently highlighted by the papers of Levine and Welch and Levine et al., in the association of cerebrovascular diseases occurring not only in systemic lupus erythematosus (SLE) but also in lupus-like diseases and in the primary antiphospholipid syndrome with antibodies to phospholipids ("lupus anticoagulant," antibodies to cardiolipin [aCL], and the false-positive VDRL). Multi-infarct dementia accompanied by cerebral atrophy may result, often with other vascular occlusive events encountered in the same patients. We wish to document one such patient, a young man with SLE, in whom the only serologic evidence of antiphospholipid antibodies was a low positive (1:1) serologic test for syphilis. He had also developed retinal artery occlusions.

Our patient, a 25-year-old man, presented in 1981 with myalgias, Raynaud's phenomenon, and generalized lymphadenopathy. Following a lymph node biopsy, his condition deteriorated and he developed pleurisy, fever, and a malar rash. SLE was diagnosed and he was started on prednisolone therapy (5 mg/
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