Lacunar Infarction due to Middle Cerebral Artery Stenosis

To the Editor:

Some cases of lacunar infarction with middle cerebral artery (MCA) stenosis may be explained by the Bernoulli principle. In a constant flow system, the smaller the cross-sectional area, the greater the flow velocity, and the lower the perfusion pressure. If the flow were turbulent, the pressure would be lower still. Thus, if the origin of a lenticulostriate artery were within a stenotic segment of MCA, the perfusion pressure in this segment would be reduced, compared to that in normal MCA segments, perhaps sufficiently to produce ischemia in the supply territory of the lenticulostriate artery. Such a hemodynamic mechanism might explain progressive lacunar infarction, which until now has not been satisfactorily explained.4

As a limited test of this hypothesis, we have reviewed 62 cases of acute hemiplegia in whom transcranial Doppler was performed within 48 hours of onset. These included 10 cases of lacunar stroke, in five of whom MCA stenosis was presumed by the criterion of mean velocity 20–60% greater on the symptomatic segment of MCA, but is paradoxically associated with thrombotic events such as deep venous thrombosis, cerebral and extracerebral arterial disease, and cerebral venous thrombosis. Few angiographic results have been reported. We have encountered a patient who gave a history of two spontaneous abortions, the most recent one 2 months before, she had noted numbness of the right hand followed 2 months later by a left cerebral infarction caused by an intraluminal thrombosis in the left internal carotid artery. He improved after surgical excision of the thrombus.5

Two additional case reports were found. One patient was a 30-year-old man with a vasculitis who suffered infarctions in the left cerebral hemisphere caused by an intraluminal thrombus in the left internal carotid artery. He improved after surgical excision of the thrombus.5 Another patient was a 38-year-old man with a diagnosis of systemic lupus erythematosus, who was hypertensive and a smoker.6 Angiography disclosed an ulcerated, nearly occluded left internal carotid artery.

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Our patient had a characteristic history for stroke associated with the lupus anticoagulant. She was young and suffered two spontaneous previous abortions, the most recent one 2 months before the stroke. There was neither a history of head or neck trauma nor use of birth control pills. She did not have systemic lupus erythematosus nor a cardiac source for embolism. Risk factors of smoking, migraine, and hypercholesterolemia were present, but none were likely to be the etiology of an extracranial internal carotid artery occlusion in a 24-year-old woman.

References


Lupus Anticoagulant Associated With Extracranial Internal Carotid Artery Occlusion

To the Editor:

Stroke and transient ischemic attacks have been associated with the lupus anticoagulant and anticardiolipin antibodies.1–3 The lupus anticoagulant prolongs the partial thromboplastin time (PTT), but is paradoxically associated with thrombotic events such as deep venous thrombosis, cerebral and extracerebral arterial disease, and cerebral venous thrombosis. Few angiographic results have been reported. We have encountered a patient who gave a history of two spontaneous abortions, with the most recent one 2 months before, she had noted numbness of the right hand followed 2 months later by a left cerebral infarction caused by an occlusion near the origin of the left internal carotid artery.

A 24-year-old Mexican woman was seen for neurologic evaluation because of impaired speech and right-sided weakness. Two months before, she had noted numbness of the right hand followed within 3 hours by inability to move the right arm and leg and difficulty expressing herself. By the next morning, she was unable to speak. She was initially evaluated in Mexico City, where computed tomography (CT) disclosed a large infarction in the left middle cerebral artery distribution. Digital subtraction angiograms demonstrated an extracranial left internal carotid artery occlusion.

Language and motor function improved slowly up until the time of this evaluation.

The patient gave a history of classic migraine and one episode of prolonged amnesia associated with migraine. She had had two spontaneous abortions, the last one 2 months prior to this stroke. There was a history of a pituitary microadenoma for which she was treated with bromocriptine. She smoked one pack of cigarettes per day. There was no history of head or neck trauma, and she had not used birth control pills.

General examinations disclosed a blood pressure of 108/64 and a regular heart rate of 68 beats per minute. There were no cardiac murmurs. She had dysarthria, nonfluent speech, poor repetition, and made numerous paraphasias. There was severe dysoria and acalulia. A right facial weakness, severe spastic right hemiparesis, right hyperreflexia, and Babinski sign were present.

Pertinent laboratory data included a normal complete blood count and platelet count. Fasting blood sugar, electrolytes, renal, liver, and thyroid functions were normal. Cholesterol was 297 mg/dl and triglycerides 150 mg/dl. Plasma reagin test was nonreactive. An erythrocyte sedimentation rate was 21 mm/hr. An antinuclear antibody was 1:8 when performed initially and 1:640 with a speckled pattern at the time of this evaluation. DNA antibody was negative. Electrocardiogram and two-dimensional echocardiogram were normal.

Coagulation studies included anticardiolipin antibodies, which were not elevated. The activated partial thromboplastin time was 41 seconds (23–36 seconds was the normal range). The partial thromboplastin time did not correct with a 1:1 mix with normal plasma. A thromboplastin inhibition test was done and was positive for the lupus anticoagulant at a dilution of 1:500. At that dilution, the control was 105 seconds and the patient 152 seconds for a ratio of 1.45 (a ratio greater than 1.3 is considered a positive result). The platelet neutralization test was also positive for the lupus anticoagulant. The PTT with platelets corrected to 38.5 seconds, but remained elevated with saline alone at 45.5 seconds (test positive when the PTT with platelets is at least 5 seconds shorter than a PTT with saline).

The CT scan showed no evidence of a pituitary adenoma, but rather a partially empty sella. There was a left frontal infarction. A review of digital subtraction angiograms demonstrated a left internal carotid artery occlusion approximately 1 cm above the bifurcation.

A cardiology consultant examined the patient and her cardiac studies and found no basis for cardiogenic embolism.

Angiographic studies in patients with the lupus anticoagulant have been summarized by Levine and Welch,1 who reported a total of 17 patients. Six of these patients had an internal carotid artery stenosis or occlusion, but only one was reported to have been an extracranial internal carotid artery occlusion.4 Two additional case reports were found. One patient was a 30-year-old man with a vasculitis who suffered infarctions in the left cerebral hemisphere caused by an intraluminal thrombus in the left internal carotid artery. He improved after surgical excision of the thrombus.5 Another patient was a 38-year-old man with a diagnosis of systemic lupus erythematosus, who was hypertensive and a smoker.6 Angiography disclosed an ulcerated, nearly occluded left internal carotid artery.

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Lupus anticoagulant associated with extracranial internal carotid artery occlusion.
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