

# Cessation of Microemboli in the Middle Cerebral Artery after a Single Dose of Aspirin in a Young Patient with Emboliogenic Lacunar Syndrome of Carotid Origin

Gregory Telman MD, Efim Kouperberg MD, Ilana Schlesinger MD and David Yarnitsky MD

Department of Neurology, Rambam Medical Center, Haifa, Israel

Affiliated to Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

**Key words:** transcranial Doppler, microemboli, drug abuser, young stroke, aspirin

*IMAJ 2--6;8:724-725*

The possible underlying mechanisms of stroke in drug abusers include: acute and severe elevation of blood pressure, cardiac dysrhythmias, cerebral vasospasm, vasculitis, embolization due to infective endocarditis, cardiomyopathy, and foreign material injected under non-sterile conditions. We describe a young drug abuser with lacunar syndrome originating from highly emboliogenic carotid plaque with cessation of emboli after the first dose of aspirin.

## Patient Description

A 36 year old, right-handed man, born in Ukraine, was hospitalized in the neurology department because of mild weakness in the right arm and leg that appeared 1 day prior to admission. Neurologic examination revealed right hemiparesis; the rest of the examination was normal. There was no history of cervical or other trauma, hypertension or diabetes mellitus. The patient gave a history of 23 pack-years of cigarette smoking and a 10 year history of injections of opioids daily. Additionally, he was known to be a carrier of hepatitis C and to suffer from iron deficiency anemia.

Computed tomography of brain on admission was interpreted as normal. A repeat CT scan 5 days later showed a hypodense area in the left centrum semiovale. Carotid duplex demonstrated a homogeneous hypoechoic plaque in the left carotid bulb causing a stenosis of 50–69%. CT angiography was subsequently performed and showed carotid stenosis of approximately 50%. A conventional angiography was suggested in order to rule out carotid dissection but the patient refused.

Transcranial Doppler (Pioneer, TC 8080, Viasys™, Nicolet) including microembolic signals monitoring was done 3 hours after the patient's admission. The monitoring of MES was performed according to criteria of international consensus group guidelines [1]. All the measurements were performed technically by one observer (E.K.), who was blinded to the clinical syndrome in the first transcranial Doppler investigation. We used a computed system of microemboli fixation with subsequent confirmation or rejection of MES by agreement between two observers (E.K. and G.T.). Routine TCD examination of cerebral blood flow velocities was normal. Transcranial Doppler monitoring was performed for 30 minutes and revealed 25 microemboli in the left middle cerebral artery. There were no emboli in the right MCA. Transesophageal echocardiography was interpreted as normal. Aspirin 500 mg once a day was initiated 5 hours after admission. TCDM was repeated twice, 24 hours and 48 hours after aspirin treatment was started. In both cases the monitoring lasted 1 hour (instead of the usual 30 minutes). In both examinations, 24 hours and 48 hours after aspirin treatment, no emboli were detected in both MCAs. The additional monitoring was performed 21 days after the first examination as follow-up and was also negative.

## Comment

This case emphasizes two important points. The first is the immediate effect of a single dose of aspirin 500 mg on complete cessation of microembolization to the MCA. It is generally accepted that prolonged antiplatelet medication with

aspirin prevents recurrent ischemic stroke. However, data on the immediate effect of aspirin on emboli rate in proximity of acute stroke are limited. Goertler et al. [2] examined 44 stroke patients with significant (> 50%) carotid stenosis and embolic signals at admission who showed rapid cessation of embolic signals under antithrombotic therapy (aspirin 500 mg in most patients) within 4 days. In contrast, Tytgat and co-workers [3] showed that a single dose of aspirin (120 mg) the night before carotid endarterectomy did not significantly reduce the emboli rate during CEA. Markus et al. [4], in the recently published CARESS trial, compared the effectiveness of aspirin alone (75 mg) versus aspirin in combination with clopidogrel in emboli reduction in patients with significant carotid stenosis. The authors concluded that combination therapy is more effective than aspirin alone (TCDM was done on admission, and on days 2 and 7). It should be emphasized that Tytgat and Markus et al., used small doses of aspirin, while Goetler et al. used high doses. Therefore, the reduction of microemboli rate may depend on the aspirin dosage.

The second point emphasized by this case is that lacunar stroke and lacunar syndrome are not the same thing. Lacunar stroke is the result of small vessel disease. Lacunar syndrome, on the other hand, may result from many etiologies including embolism. In our case, the

MES = microembolic signals

TCD = transcranial Doppler

MCA = middle cerebral artery

TCDM = transcranial Doppler monitoring

CEA = carotid endarterectomy

young patient without hypertension and diabetes mellitus suffered from pure motor stroke compatible with a typical lacunar syndrome with a probable emboliogenic origin.

The carotid plaque was homogeneous and hypoechoic and this is in concordance with the literature pointing to more emboliogenicity of such hypoechoic plaques [5]. The carotid plaque found in our patient may represent a fresh carotid thrombus, which might have resulted from carotid dissection. This possibility was not entirely ruled out because a conventional angiography was not performed, yet a negative duplex and CT angiography without a history of trauma make this diagnosis less likely.

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**Correspondence:** Dr. G. Telman, Dept. of Neurology, Rambam Medical Center, Haifa 31096, Israel.

Phone: (972-4) 854-2161

Fax: (972-4) 854-2944

email: g\_telman@rambam.health.gov.il