Clusters of Microembolic Signals: A New Form of Cerebral Microembolism Presentation in a Patient With Middle Cerebral Artery Stenosis

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Background—Middle cerebral artery (MCA) stenosis is a rare occlusive disease in western populations, with a high risk of stroke recurrence. An artery-to-artery embolic mechanism has been argued. We report the detection of a new pattern of microembolic signals (MES) in the MCA poststenotic segment in a patient with multiple recurrent transient ischemic attacks.

Case Description—A 75-year-old man was admitted to our hospital with a transient leg monoparesis on the left side. Right MCA stenosis was detected by transcranial Doppler ultrasonography (TCD). In spite of aspirin treatment, the patient had a recurrent right hemispheric transient ischemic attack, and anticoagulant therapy was started. A new, similar event happened after correct anticoagulation. The bigated TCD monitoring study of both MCAs disclosed clusters of MES at the poststenotic MCA segment. Each cluster contained between 12 and 45 embolic signals. The patient became asymptomatic and clusters of MES disappeared, coinciding with the combination of ticlopidine and oral anticoagulants.

Conclusions—We describe a new presentation of MES, ie, grouped in clusters of signals, that may be associated with a high risk of stroke recurrence. (Stroke. 1998;29:722-724.)

Key Words: cerebral embolism ■ middle cerebral artery ■ stenosis ■ ultrasonography

S tenosis of the MCA causes less than 5% of the ischemic strokes in western populations.1-2 Ischemic events in the hemisphere distal to the stenosis could occur because of hemodynamic insufficiency or by embolic artery-to-artery mechanism, but the fact that stroke recurrence is not reduced in these patients after extracranial-intracranial bypass surgery strongly suggests the latter.3,4

We report the case of a patient with recurrent TIAs and MCA stenosis in whom TCD monitoring showed several clusters of MES at the poststenotic segment of the affected MCA.

Case Report
A 75-year-old man was admitted to our hospital because of sudden lower left limb paresis. On the previous day, the patient had suffered two events of acute transient paresthesia, each lasting for about 30 minutes in the same zone, but he did not consult his general practitioner. His medical history was remarkable only for arterial hypertension and mild hypercholesterolemia. On admission he was alert, oriented, and with normal speech; mild paresis in the leg and a Babinski sign were observed. Arm strength, sensitivity, and cerebellar functioning were normal. Results of chest x-ray and ECG and biochemical and hematologic parameters were normal. Ten hours after admission the patient became asymptomatic. Cranial CT showed a right lenticular hypodensity that was interpreted as an old lesion. Cervical color-coded duplex sonography revealed a low-grade (<30%) bilateral carotid artery stenosis, and there were no ulcerated plaques. The patient was treated with aspirin (500 mg/d). The day after admission, the TCD study showed a high-grade right MCA stenosis, with a mean velocity of 272 cm/s. The stenosis was confirmed through cranial helical CT angiography (Fig 1). Forty-one hours after admission the patient suffered a new TIA with left brachiocephalic hemiparesis that lasted for 8 hours; anticoagulant therapy was started with sodium heparin. Five days after admission the patient had a new, brief TIA recurrence (only 5 minutes), in spite of correct anticoagulation (activated partial thromboplastin time was three times the control value). TCD monitoring of both MCAs was performed for 1 hour with a pulsed-Doppler machine (DWL Multidop X4) provided with two bigated probes and special software for the detection of MES. These probes, which are capable of simultaneous insonation at two different depths of the same vessel, were placed over each MCA. We placed one gate receiving flow signals from the stenotic segment at the right artery and the second gate at the greatest possible distance from the center of stenosis. The sample volume of probe insonation was only 5 mm because only a short poststenotic segment of the right MCA was detected in the same plane. Data from both channels was recorded on digital audiotape (Sony SLV-425) to confirm visual and acoustic criteria for MES. During the monitoring we detected MES only on the right side. Grouped MES were...
detected at the rate of eight clusters per hour, and each cluster contained between 12 and 45 embolic signals (Fig 2). Only about 50% of the proximal MES could also be recognized at the distal gate, probably as a result of a partial lumen insonation at the depth of 40 mm; alternatively, MES could have escaped from the second gate by traveling through lenticulostriate branches. A further study with the two gates located in the prestenotic segment did not disclose MES. Ticlopidine (250 mg/12 h) was added to the anticoagulant treatment. Forty-eight hours later the patient remained asymptomatic and a new TCD monitoring did not show clusters of signals, although there were 18 ungrouped MES per hour. The patient was discharged 4 days later, free of TIA recurrences and on a regimen of ticlopidine and acenocoumarol (INR 2.6). Three months later, the TCD study showed a high-grade MCA stenosis with a mean velocity of 226 cm/s, but again no MES were detected. At the last check-up, 5 months after discharge, the patient was still healthy and receiving acenocoumarol (INR 2.5) and ticlopidine. A new helical CT did not reveal new cerebral infarcts, and the degree of the stenosis was similar to that of the first examination.

**Discussion**

It has been shown during the last 7 years that TCD can detect cerebral microemboli. These microemboli were originally identified during monitoring of carotid and cardiac surgery and described as HITS over the background blood flow Doppler signal. Subsequently, studies with in vitro models suggested that these signals could be caused by air bubbles and fat or platelet-fibrinogen embolic particles. There has been considerable discrepancy between different investigators about the frequency of MES in the different pathologies. To address this problem, the Consensus Committee of the 9th International Cerebral Hemodynamic Symposium established as embolic signals only the HITS that are unidirectional, high intensity, of short duration, and accompanied by a characteristic “clicking” sound. Any MES should be recorded as HITS in TCD monitoring. Although the definition of HITS did not mention the temporal profile of the signal distribution, our own experience and previous reports found HITS with an aleatory but homogeneous distribution over the monitoring time. MES have been detected in a variety of conditions, including prosthetic valve replacement, high-grade carotid stenosis, atrial fibrillation, and acute cerebral ischemia, but only a few studies have reported MES in relation to intracerebral artery stenosis. MCA stenosis is a relatively rare disease in the Caucasian population, but it is strongly associated with a high rate of stroke recurrence. An embolic artery-to-

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**Selected Abbreviations and Acronyms**

- **HITS** = high-intensity transient signals
- **INR** = international normalized ratio
- **MCA** = middle cerebral artery
- **MES** = microembolic signals
- **TCD** = transcranial Doppler ultrasonography
- **TIA** = transient ischemic attack

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**Figure 1.** Helical CT angiography of the intracranial arteries. The arrow indicates a high-grade right MCA stenosis. The CT image is a multiprojection volume reconstruction.

**Figure 2.** Example of a cluster of MES. The upper panel shows a sequence of MES in a very high time resolution spectral display. In each spectral window, the calculated transit distance (lower right), the absolute dB value (lower left), and the depth of insonation (upper left) are displayed. The relative dB level and the time are given under the spectrum. Nearly thirty MES can be identified in a 3-second period of time. The lower panel shows a raw Doppler signal of one of the individual microemboli. The time delay of the episode at each insonation depth is displayed in milliseconds. The TCD software uses the delay in the detection of particles to automatically distinguish between emboli (particles in movement) and artifacts.
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artery mechanism has been suggested for stroke in these patients, and it is supposed that the emboli spring from the stenotic segment to reach the distal field of the artery. Navabi et al. found MES in the poststenotic segment in 14% (2 of 14) of MCA stenoses but all occurred in the acute phase of stroke in patients not treated with anticoagulants. However, Sliwka et al. could not detect any MES in 58 patients with chronic stenosis who received different treatments. This could result either from MES never having been present or from their disappearance during the natural course of symptomatic MCA stenosis.

In this article we describe a new pattern of clusters of MES at the poststenotic segment of the MCA in a patient with ipsilateral recurrent ischemic events, a finding that to the best of our knowledge has not been previously reported. In our patient, the disappearance of clusters of MES in the early acute phase may reflect the natural course of the MCA stenosis or a treatment effect of the acenocoumarol and ticlopidine combination. Although at present the best treatment for intracranial artery stenosis remains uncertain, it seems that there is a favorable risk-to-benefit ratio for anticoagulant compared with aspirin therapy for prevention of major vascular events in symptomatic patients. We think that MES detection in the acute phase of symptomatic MCA stenosis is probably a marker for partial recanalization and speculate that the new form of MES presentation we describe is due to the release of multiple particles of microaggregates owing to turbulent flow at the level of the stenosis. Our results suggest that clusters of MES could be associated with a high risk of recurrence and that anticoagulant along with antiplatelet drugs could be a treatment option in these cases.

References

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