

ORIGINAL ARTICLE

MICROEMBOLIC SIGNAL MONITORING IN PATIENTS WITH ACUTE STROKE

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Background- There are few data on the occurrence of microembolic signals (MES) in the acute phase of ischemic stroke. The aim of this study was to determine the frequency of MES in patients with middle cerebral artery (MCA) ischemic events and to evaluate the clinical value of MES.

Methods- Twenty patients with an ischemic infarction in the MCA territory were investigated by transcranial Doppler technology within 24 hours of the onset of symptom. One-hour recording of MES was performed using an ultrasound device with a 2 MHz probe (Vingmed 800, Oslo, Norway) from the affected MCA. The criteria of MES identification were short duration (100-300 ms), 9 dB above the Doppler background frequency, typical chirp sound, occurring random in cardiac cycle and unilateral visual appearance on the spectral display. Early recurrent stroke was investigated during the observation period.

Results- Among the 20 patients completing the study, three (15%) showed MES in their recording and had a definite cardioembolic source. One patient developed a minor ischemic event despite heparinization. In five patients with lacunar stroke and four patients with atherosclerotic carotid artery stenosis, no MES was found. Early recurrent stroke was absent in the patients without MES.

Conclusion- There seems to be an association between MES in the acute phase of stroke and a definite cardioembolic sources. Larger studies are needed to confirm these preliminary results.

Keywords • embolism • microembolic signals • stroke • ultrasonography

Introduction

Although special attention has been given to the detection of cerebral microemboli in recent years, the clinical relevance of microembolic signals (MES) remains controversial. Extracranial internal carotid artery (ICA) occlusive disease and cardiac disease are the main sources of embolization into the cerebral arteries and subsequent stroke. Acute stroke patients are prone to stroke recurrence or worsening of symptoms. Clinically, silent circulating cerebral microemboli can be detected noninvasively by means of transcranial Doppler (TCD) ultrasonography. They present as high-pitched signals within the frequency spectrum.^{1,2} The presence of MES is indicative of ongoing embolization in the cerebral arteries and provides

clinical information on emboligenic activity of the embolic source.

Several studies have investigated the prevalence and frequency of MES in stroke.³⁻⁵ The objective of this prospective study was to investigate the frequency of MES in patients with middle cerebral artery (MCA) ischemic events and their association with potential sources of embolism. Furthermore, we explored the relationship between MES detection and recurrent stroke during the observation period.

Patients and Methods

Thirty-eight patients who were admitted to our neurology ward with an MCA ischemic event with onset of symptoms within 24 hours prior to the investigation were included. To exclude intracranial hemorrhage, in the study all patients underwent cranial computed tomography (CT)

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Table. Review of the literature on microembolic signals (MES) studies.

Author, year	Number of cases	Maximal delay in recording (day)	Duration of recording (min)	MES positivity (%)	Repeated investigation
Daffertshoffer et al ³ 1996	280	28	30	9.3	No
Sliwka et al ⁴ 1997	78	7	30	51	Yes
Koennecke et al ⁵ 1998	145	16	30	24	No
Grosset et al ⁹ 1994	41	2	30	66	No
Del Sette et al ¹⁰ ,1997	90	3	30	12	Yes

following admission.

Standard hematologic and coagulation tests, standard electrocardiography, color-coded duplex sonography of the supra-aortic trunks, trans-thoracic echocardiography, TCD and cerebral microembolus monitoring were performed.

TCD ultrasound was completed using an ultrasound device with a 2 MHz probe (Vingmed 800, Oslo, Norway). The examination included the intracranial segments of the ICAs, the MCAs, the anterior and posterior cerebral arteries, the vertebral arteries, and the basilar artery.

For TCD embolus detection, the main stem of the MCA on the side of infarction was isolated through the temporal window. We placed the gate at a depth of 50 to 55 mm. A low gain provided a setting guaranteeing optimal embolus discrimination from the background spectrum.⁶ The patients lay in a recumbent position on a couch. A 2-MHz transducer was fixed in a temporal position using a head strap; the duration of recording was 1 hour. MES were identified according to their previously documented characteristics: short duration (100–300 msec), typical visual appearance on the spectral display with a typical sound (click, chirp, bloop) occurring at random in the cardiac cycle.^{1,2,7,8} The thresholds used to identify short microembolic ultrasound events was 9 dB above the Doppler background. Patients were monitored clinically for worsening or recurrence of symptoms on a day-by-day basis during the 2 weeks of observation.

Results

From the 38 patients who were eligible for the study, 18 patients were later excluded; four due to occlusion of the main stem of the MCA, thus, preventing detection of MES within the artery. Eight patients had an insufficient temporal bone window. In five patients, the time between the onset of symptoms and hospital admission turned out to be longer than 24 hours. One patient was

restless and uncooperative.

Evaluation was completed in 20 patients (age range, 26–85 years). An ischemic infarction in the MCA territory was confirmed in 19 patients. In the remaining patient, despite a normal CT scan, clinical presentation and high-grade carotid artery stenosis strongly supported the localization of the infarction in the MCA territory.

Five patients had experienced a previous cerebral ischemic event. MES were detected in 3 (15%) patients. One female patient (patient 8) showed 24 MES per hour. A small thrombus at the cardiac apex, mitral valve stenosis and regurgitation were detected as the cause of her stroke. Although she was heparinized, she experienced a minor ischemic event on the seventh day of hospitalization. Two patients (patients 11 and 14) who had atrial fibrillation and severe mitral valve stenosis showed 5 and 3 MES per hour, respectively. Thus, a potential cardioembolic source was detected in all three patients who showed MES. Early recurrent stroke was absent in patients without MES.

Discussion

The Table presents an overview of previous MES studies on stroke. These studies differ in two respects; first, the three studies performed by Daffertshofer,³ Sliwka⁴ and Koennecke⁵ and their colleagues did not focus on patients in the early phase of stroke as we did in this study. Second, the recording time was 30 minutes in previous studies and 60 minutes in our study.

Neither of our five patients with lacunar infarction, nor the four patients with atherosclerotic carotid artery stenosis, showed any MES. Lack of MES in lacunar strokes has already been reported by two other groups.^{3,9} One patient with MES (patient 8) developed an early recurrent ischemic event. Two other patients with MES (patients 11 and 14), and all patients without MES, did not show any further ischemic stroke

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during the observation period.

The use of anticoagulant or antiplatelet drugs in patients with acute stroke is controversial. With the exception of patient 8, none of our patients received anticoagulant or antiplatelet drugs during the observation period.

A shortcoming of our study is the small number of our patients, which does not allow us to make any statistically firm conclusions concerning MES as an indicator of early stroke recurrence. Nonetheless, we hope our findings urge the initiation of further definite measurements in larger numbers of patients with stroke in order to characterize the clinical significance of MES more fully. Our findings do not indicate how often a patient should be investigated in order to qualify him or her as embolus-negative, and this question remains unanswered.

In conclusion, our study emphasizes the importance of a cardioembolic source in patients with MES during their stroke. Microemboli detection helps to estimate the persisting emboligenic potential of an embolic source in the acute phase of stroke.

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