Determinants of Microembolic Signals in Patients with Atherosclerotic Plaques of the Internal Carotid Artery

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Dr. Telman and colleagues are to be commended with a significant contribution to the intriguing field of microembolic signal (MES) registration in patients with atherosclerotic carotid disease.

Intriguing, because any first-time observer of a transcranial Doppler rendered microembolic shower in the middle cerebral artery is convinced, for a moment, that this patient will never wake up. Fortunately however, as so often, nature is mild and most such patients will 'recover' without any obvious sequelae to this so ominously displayed event.

The clinical importance of MES, therefore, has been subject to debate ever since the first report on this method, by Spencer et al. in 1969. Forty years later, this elegant monitoring technique still struggles to mature from a mere research tool to the ranks of evidence based, standard of care. The current publication, with all respect, does not help the issue by failing to confirm the well-established paradigm that the most embologenic period is close to the acute stroke, one of the pillars of the current practice of early surgical intervention for symptomatic carotid stenosis. As the authors state, the small sample size of patients with stroke in their population may be due to this conceived lapse.

Another shortcoming in this study is that alternative sources of emboli, such as the heart, aortic arch, or the intracranial vessels themselves, have not been ruled out. Interestingly, perhaps for the same reasons, also the degree of carotid stenosis did not relate to a higher presence of MES as has been suggested frequently in the past.

Plaque characteristics such as ultrasonic texture and density, in this study, were not associated with a higher frequency of MES. Apparently, soft vulnerable plaque is a liability to the patient only when doctors are around: evidence is accumulating that such plaque characteristics are strongly associated with increased microembolisation during carotid interventions, both carotid endarterectomy and stenting.

The authors did find a greater presence of MES in patients who suffered stroke, either in the past or within four days of examination, significantly differing from those who suffered TIA's alone or were asymptomatic.

From a clinician’s point of view, however, the question remains what to do with the current abundance of MES related data. Is MES detection of proven use for risk stratification, choice of medical therapy or choice of intervention? If a clearly observed storm of MES does not necessarily always lead to (temporary) paresis, then what do microemboli do to the brain? Should we look further than a patient’s arm in the air and focus more on subtler parameters such as memory and attention?

I am looking forward to future contributions by Dr. Telman and others in this intriguing field of research.

Reference