Limb-Shaking Transient Ischemic Attack with Distal Micro-Embolic Signals and Impaired Cerebrovascular Reactivity Using Transcranial Doppler

Dear Editor,

We present a patient with limb-shaking transient ischaemic attack (TIA) associated with extracranial carotid occlusion and suggest that the pathophysiological mechanism is hypoperfusion, as evidenced by impaired cerebrovascular reactivity.

A 52-year-old man presented with 10 episodes of left-sided involuntary movements over 1 week, each lasting less than 5 minutes. There was no head/eye version, incontinence or impaired consciousness. The symptoms were not associated with positional change or exertion. He was a smoker and had no past medical history of note. There were no neurological deficits on examination. The pulse was regular at 68/minute and there was no heart murmur.

Brain CT showed an old left occipital infarct. Ultrasound studies showed right internal carotid artery (ICA) occlusion. Transcranial doppler (TCD) studies revealed blunted right middle cerebral artery (MCA) waveforms with 5 micro-embolic signals detected over 30 minutes, during which the patient was asymptomatic. Diffusion-weighted magnetic resonance imaging (MRI) showed no acute infarction. Brain magnetic resonance angiography (MRA) revealed some signal in the right MCA, possibly supplied from a patent anterior communicating artery and retrograde flow in the right anterior cerebral artery (ACA). Catheter angiography confirmed right ICA occlusion. TCD monitoring in response to carbon dioxide revealed impaired cerebrovascular reactivity of the right MCA (Table 1). Electrocardiogram and transthoracic echocardiogram were normal.

Double antiplatelet therapy with aspirin and clopidogrel, proven for micro-embolic signal reduction,[1,2] was started. Three days later, only 1 micro-embolic signal was detected in the right MCA over 30 minutes. After 2 weeks, aspirin was ceased and clopidogrel continued. The patient remained asymptomatic over the following 7 months. Repeat carotid ultrasound showed persistent right ICA occlusion. Four micro-embolic signals were detected in the right MCA over 60 minutes. There was persistent cerebrovascular reactivity impairment of the right MCA.

The clinical picture was consistent with limb-shaking TIA.[3,4] The microembolic signals were likely asymptomatic artery-to-artery embolism as the patient had no symptoms when the micro-embolic signals were detected.

This is the first case report to describe impaired cerebrovascular reactivity to carbon dioxide using TCD studies in limb-shaking TIA. We postulate that the pathophysiological mechanism for the limb-shaking TIA in this patient was hypoperfusion due to carotid disease, which has been previously proposed.[5,6] A possible reason for the lack of symptoms despite persistently impaired cerebrovascular reactivity is the development of tolerance to hypoperfusion. Perfusion CT has been used to demonstrate impaired cerebrovascular reactivity in limb-shaking TIA.[7] We have shown that cerebrovascular reactivity to carbon dioxide with TCD studies can also assess for haemodynamic compromise in suspected limb-shaking TIA.

Table 1. Transcranial Doppler Studies Results of Cerebrovascular Reactivity Testing with Inhalation of Carbon Dioxide

		Left MCA		Right MCA	
At presentation	**End-tidal CO_2 (mmH)**	**Mean velocities (cm/s)**	**CVR Index**	**Mean velocities (cm/s)**	**CVR Index**
Baseline 1	4.6	47.9	-	36.5	-
6% CO_2 in air	6.6	71.2	24.3%	42.8	8.6%
Baseline 2	4.2	47.8	-	33.2	-
8% CO_2 in air	8.2	76.0	14.7%	43.1	7.5%
4 months after presentation					
Baseline 1	5.0	56.2	-	35.1	-
6% CO_2 in air	7.2	83.4	22.0%	38.7	4.7%
Baseline 2	5.0	48.9	-	35.4	-
8% CO_2 in air	7.6	81.0	25.2%	41.7	5.6%

CO_2: carbon dioxide; CVR: cerebrovascular reactivity; MCA: middle cerebral artery

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