Early sign of hemodynamic insufficiency in the MCA watershed territories

Just the (finger)tip of the iceberg?

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In April 2009, a 68-year-old right-handed man presented hours after acute onset of left hand and leg weakness. During a 6-week period preceding admission he had experienced at least 2 sudden onset episodes of an isolated paresthesia of the left ring finger. Each persisted for approximately 2 minutes and the patient did not seek medical attention for it. He had a history of hypertension and hyperlipidemia, but no previous stroke. Clinical examination showed mild left hemiparesis, symmetric monosynaptic reflexes, and hypesthesia of the left hand, this time affecting all fingers. There were no signs of peripheral nerve lesion—sensitive or motor—and no change of or new paresthesia under provocation tests for entrapment neuropathies. MRI of the head and neck with diffusion-weighted sequences and angiography showed isolated right middle cerebral artery (MCA) stenosis and multiple acute infarctions in its watershed territories, involving the right corona radiata and cortical and subcortical white matter of the right fronto-parieto-occipital lobes (figure, A). Routine blood tests, chest radiography, ECG, long-term ECG, and EEG were normal. Trans-thoracic echocardiogram indicated beginning hypertensive heart disease, no cardiac thrombus or valvular dysfunction. The following day, he received successful angioplasty and stenting of the symptomatic MCA stenosis (figure, B and C). Treated before publication of the SAMMPRIS¹ trial data, follow-up of this patient to date is without further ischemic events or complications under antiplatelet therapy.

Cerebral watershed infarcts typically involve the junction of the distal fields of the nonanastomosing territories of the anterior cerebral artery (ACA), middle cerebral artery, and posterior cerebral artery and account for approximately 10% of cerebral ischemias.² Untreated MCA stenosis is associated with a high rate of stroke recurrence³ and warning TIAs are common.³ Recognized pathogenetic factors are hemodynamic impairment and microembolism.² We report a case of unilateral watershed infarction due to MCA stenosis with prodromal isolated paresthesia of 1 ring finger. Pseudoperipheral sensory-motor deficits due to CNS lesions were described as early as 1909.⁴ In a more recent MRI study on cortical infarction with weakness restricted to particular groups of fingers,⁵ predominant ulnar finger involvement was closely

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Figure Ischemic lesions in middle cerebral artery (MCA)-anterior cerebral artery (ACA) watershed territories are likely to cause sensorimotor deficits of the hand or of isolated fingers

(A) Axial fluid-attenuated inversion recovery (above) and diffusion-weighted imaging (below) MRI sequences of the head, day of admission: multiple cerebral infarction in the right MCA-ACA watershed territories. Anatomic landmarks and lesions responsible for sensory and motor deficits are highlighted. (B, C) Cranial digital subtraction angiography, contrast in right internal carotid artery, day after admission; MCA stenosis (red arrow) before (B) and after (C) successful angioplasty and stenting. (D) Schematic illustration, coronal view of the brain at the central sulcus; area supplied by the MCA (blue, left) and somatotopic cortical sensory homunculus (right).

associated with cerebral ischemia by a hemodynamic mechanism, radial finger involvement with normal angiograms and embolic mechanisms. As motor and sensory representation areas of particular fingers are reported to be closely related in individual subjects,⁶ this topical pattern might as well be valid for pure sensory symptoms.

Why is this exemplary? Quite often, even large cortical infarction goes undetected unless it crosses primary sensorimotor representations or its immediate afferences and efferences. Accordingly, our patient had been unaware of all the different lesions in the watershed area but the one touching primary sensory cortex (figure, A, blue circle) and the one affecting the pyramidal tract below primary motor cortex (figure, A, yellow circle). Due to the large cortical representation of the hand, spanning the ACA-MCA borderzone (figure, D), minor cerebral ischemia can affect single fingers. What appears to be a curious observation at first rather proposes a general rule: severe MCA stenosis causing watershed ischemia is likely to present with sensorimotor impairment of isolated fingers. This may be the only sign preceding devastating stroke due to MCA occlusion.

If a patient presents with hypesthesia/paresthesia in isolated fingers, we suggest considering internal carotid artery or intracranial artery occlusive disease along with the more common causes of fractional sensory-motor deficits, particularly if the patient has risk factors for cardiovascular disease and typical signs of peripheral nerve damage are absent. High availability of CT/MRI angiography and transcranial duplex/Doppler sonography facilitates differential diagnostics. Antiplatelet drug therapy and potentially revascularization procedures, including angioplasty or bypass surgery, improve the prognosis of patients with symptomatic MCA stenosis. By mistaking this valuable prodrome as peripheral nerve dysfunction, the opportunity to treat risk factors and prevent further stroke will be missed. Primary care physicians and patients at risk for further vascular complications should be aware of this particular prodrome.

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DISCLOSURES

The authors report no disclosures.