Can cerebral microbleeds cause an acute stroke syndrome?

Giridhar P. Kalamangalam, MD, DPhil, University of Texas Health Science Center, Houston: I read with interest the report of Teo et al.¹ on acute vascular syndromes attributed to cerebral microbleeds (CMBs). Assessing neurologic presentations in these patients can be challenging; we recently encountered a patient who illustrated the diversity of presentation of this condition.

A 53-year-old man with previous whole brain irradiation for metastatic cancer reported repeated episodes of slowly progressive (over 5–60 minutes) unilateral weakness, proceeding to right or left hemiplegia lasting several hours. There was a visual disturbance at symptom onset and a unilateral headache at some stage. Neurologic examination and routine EEG were normal; brain MRI demonstrated multiple widespread punctuate CMBs (figure). Complex secondary migraine was diagnosed and prophylactic treatment suggested. Months later the patient presented to his local hospital with a further such episode progressing to complete flaccid right hemiplegia and global aphasia. Neuroimaging showed no new changes. He was transferred to our center after a week of failing to improve. Though the clinical suspicion for seizures was low, continuous portable EEG monitoring was commenced, which revealed the diagnosis: repeated prolonged nonconvulsive left hemispheric seizures. The patient improved rapidly on institution of anticonvulsants.

The rapidity of progression of neurologic symptoms is the classic clinical differentiator of migraine and seizure.² However, this patient manifested his seizures in a hemiplegic migraine fashion, for reasons we have not been able to ascertain. On a practical level, we have learned circumspection in the interpretation of neurologic symptoms in patients with CMBs.

Disclosures: The author reports no disclosures.

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Authors Respond: James Teo, PhD, MRCP, Institute of Neurology, London: We thank Dr. Kalamangalam for demonstrating that it is often difficult to attribute neurologic symptoms to CMBs. However, his case is different on several points.

Our cases had an immediate onset without fluctuation suggesting a vascular cause, unlike Dr. Kalamangalam's case, which was fluctuant. Dr. Kalamangalam's case had many subcortical and juxtacortical CMBs (probably from radiation vasculopathy³), so it is difficult to attribute a single causational CMB for the initial presentation. Our 2 cases were selected to be illustrative, so had fairly limited CMBs (our first patient had only 1 CMB throughout his cerebral vasculature). As such we can reliably attribute the CMB to the clinical symptoms. In Dr. Kalamangalam's case's second presentation, it would not be realistic that a single CMB would cause complete right hemiplegia and aphasia, which clearly prompted Dr. Kalamangalam to investigate further.

We agree that one should be cautious in attributing acute neurologic syndromes (vascular or ictal) to CMBs especially where there is gradual onset, fluctuant symptoms, and difficulty associating clinical symptoms to a single CMB. Nonetheless, it remains important to appreciate that CMBs can be acutely symptomatic.

Disclosures: See original article for full disclosure list.

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Let live or let die after traumatic coma: Scrutinizing somatosensory evoked potentials

Nitin K. Sethi, MD, New York–Presbyterian Hospital: I read with interest the recent article by Luauté et al.¹ critically analyzing 3 cases of traumatic comatose patients who awoke despite bilateral abolition of somatosensory evoked potentials (SEPs). Recent reviews indicate that accurate prognostication of neurologic outcome after hypoxic ischemic encephalopathy (HIE) is best accomplished by a multimodality approach involving repeated clinical examinations, presence or absence of N20 on SEPs, presence or absence of reactivity and malignant patterns on EEG, and neuron-specific enolase levels.^{2–4} This is especially true in the setting of induced hypothermia, which is now also induced in traumatic brain injured patients. Neurologic prognostication is exceptionally difficult in this group of patients, in whom the insult is either focal or multifocal and rarely global as in HIE. In these patients the ideal time after the insult when these testing modalities should be employed is also not clear. A strategically placed focal lesion may selectively abolish one testing modality while leaving others relatively intact. A longer period of clinical observation should be correlated to data from multiple neurophysiologic modalities prior to prognostication. There is an urgent need for standardized guidelines for performing and interpreting these studies. This shall help avoid the false positive cases that recently have gained infamy in the national and international media.

Disclosures: Dr. Sethi is an associate editor for the *Eastern Journal of Medicine*.

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Authors Respond: Jacques Luauté, MD, PhD, Catherine Fischer, MD, PhD, University of Lyon, France: We agree with Dr. Sethi, who highlights the utility of a multimodality prognostic approach for comatose patients especially in the case of induced hypothermia. We also agree that the prognostication of awakening is exceptionally difficult for traumatic comatose patients. However, the term neurologic prognostication is inappropriate given that this term encompasses regaining of awareness, neuropsychological impairment, and functional outcome, all of which are frequently fuzzy at the initial stage.

Finally, it should be clarified that the need to avoid false positive is true for traumatic coma whereas for hypoxic encephalopathy, the pejorative prognostic value of SEPs has been repeatedly demonstrated without controversy.

Disclosures: See original article for full disclosure list.

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