Recurrent Focal Neurological Symptoms and Cortical Siderosis

The Neurohospitalist
2017, Vol. 7(2) 104-105
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DOI: 10.1177/1941874416656732
journals.sagepub.com/home/NHO

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Keywords

angiopathy, siderosis, amyloid, MRI

Figure 1. A, Brain magnetic resonance imaging (MRI) revealing subarachnoid hemorrhage on both parietal convexities. B, Dark signal on gradient echo sequences with gyriform distribution involving the frontoparietal region, consistent with cortical siderosis. C, Positron emission tomography scan with ¹⁸florbetapir demonstrating diffuse cortical radiotracer uptake.

Case

A 90-year-old male presented with 2 episodes of reversible left-hand weakness, each lasting approximately 15 minutes. He had a previous history of multiple episodes of stereotyped, reversible right-sided paresthesias followed by hemiparesis and dysarthria lasting 1 minute in duration, interpreted as transient ischemic attacks (TIAs) and treated with clopidogrel. The patient was in no acute distress at the time of presentation and was without any cognitive deficits. Subsequent brain magnetic resonance imaging revealed subarachnoid hemorrhage on both parietal convexities (Figure 1A, arrows). Dark signal on gradient echo sequences with gyriform distribution involving the frontoparietal region was observed (white arrowheads), consistent with cortical siderosis (Figure 1B). Positron emission tomography scan with ¹⁸florbetapir demonstrates diffuse cortical radiotracer uptake (Figure 1C, black arrowheads). In addition to these radiographic findings,

further subsequent evaluation revealed elevated cerebral spinal fluid β -amyloid.

Discussion

The presence of transient focal neurological deficits in conjunction with cortical siderosis on imaging is suggestive of cerebral amyloid angiopathy (CAA) as opposed to other causes of hemorrhage.^{1,2} Cerebral amyloid angiopathy is an angiopathic disorder in which amyloid deposits in the blood

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vessel walls of the central nervous system. It may present with cognitive impairment or without as was the case with this patient.³ Intracerebral microhemorrhages in a distribution primarily involving the cerebral cortex are also commonly present in CAA. Lumbar puncture with elevated cerebral spinal fluid β -amyloid is also consistent with CAA as the underlying etiology.³ In context, the frequent stereotypical symptoms suggest simple partial seizures with transient motor and sensory phenomena secondary to hemorrhage, rather than TIA. An initial diagnosis of TIA led to antiplatelet therapy, which likely contributed to the observed subarachnoid hemorrhage,⁴ emphasizing the importance of CAA as a central part of the neurohospitalist's differential diagnosis.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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