Global Hemispheric CT Hypoperfusion May Differentiate Headache With Associated Neurological Deficits and Lymphocytosis From Acute Stroke

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Headache with associated neurological deficits and lymphocytosis (HaNDL) is characterized by temporary recurrent neurological deficits, moderate–severe headache, cerebrospinal fluid lymphocytosis, elevated protein, and increased opening pressure. Although CT and MRI should be normal, single photon emission CT may indicate focal hypoperfusion and electroencephalogram may reveal slowing or even epileptiform activity resolving once the patient is symptom-free (3 months). Catheter angiography also yields normal results but may trigger an acute neurological episode.

Because HaNDL is a benign, self-limited syndrome, it is important to differentiate it from cerebrovascular disease to avoid unnecessary interventions such as catheter angiography and thrombolysis. We present a case initially thought to be acute stroke in which the patient was considered for thrombolysis. CT perfusion changes atypical for stroke made stroke diagnosis questionable and thrombolysis was withheld.

Case Report
A 31-year-old man with hypertension, dyslipidemia, and sleep apnea (treated with continuous positive airway pressure), but no history of prior migraine, was brought into our emergency department by ambulance with suspected acute ischemic stroke. He had become aphasic at work so his coworkers called 911. On examination, he was globally aphasic without focal weakness or obvious sensory changes aside from questionable mild right facial paresis (National Institutes of Health Stroke Scale score 6). A noncontrast CT and CT angiogram (CTA) were normal, but CT perfusion revealed a striking pattern of decreased and delayed perfusion to the entire left hemisphere (Figure A). Compared with the right hemisphere, there was a reduction in cerebral blood flow and volume to 80% and 95%, respectively, and the mean transit time was prolonged to 136%, all consistent with a pattern of benign oligemia rather than ischemia. Given these findings, he was not offered thrombolysis. Over the subsequent hour, his aphasia began to improve and he reported an evolving severe headache that was alleviated by acetaminophen. By 8 hours postonset, his symptoms had resolved. He experienced a second episode of global aphasia the next day accompanied by right arm paresthesias, which was followed by a severe, albeit gradual-onset, bilateral throbbing headache lasting for several hours. A third episode occurred while the patient was on a day pass 2 days later. He developed a recurrent severe headache with associated nausea/vomiting and concomitant confusion (he ran away from the ambulance attendants) that resolved within 12 hours while back in the hospital.

Routine blood work, toxicology, and MRI, including diffusion-weighted imaging, MR angiography, and fluid-attenuated inversion recovery (Figure B), were normal. A lumbar puncture revealed elevated opening pressure of 270 mm H20, increased protein (834 mg/L), and pleocytosis (447×106/mL) with 96% lymphocytes. Glucose and Gram’s stain/culture were normal as was a complete viral workup, including herpes simplex, West Nile, cytomegalovirus, syphilis, and HIV. An electroencephalogram revealed intermittent delta wave slowing and transient sharp waves in the left temporal lobe.

The patient was discharged on carbamazepine as a precaution for possible seizures. He had no further neurological episodes or headaches and returned to work. Repeat clinical examination, CT perfusion (see Figure C), electroencephalogram, and formal detailed neuropsychological testing all performed 4 to 7 months later were within normal limits. The carbamazepine was tapered and he remains symptom-free. A final diagnosis of HaNDL was given because his presentation met the diagnostic criteria in the absence of a more likely diagnosis.

Discussion
It is important for neurologists to be aware of HaNDL syndrome. The presentation of sudden-onset neurological deficits and lymphocytosis may be atypical for an acutely ischemic stroke and catheter angiography and thrombolysis are not indicated. It is important to consider HaNDL in the differential diagnosis of acute stroke in patients with a transient neurological deficit.
This case because it is routinely used at our center for the evaluation of suspected acute stroke due to its rapid acquisition and processing characteristics. However, it is likely that MR perfusion would have yielded similar hemispheric hypoperfusion findings. We conclude that CT perfusion may help differentiate stroke from the more benign HaNDL syndrome in the acute setting.

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Disclosures

None.

References


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