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MIGRAINE-LIKE HEADACHE WITH VISUAL DEFICIT AND PERFUSION ABNORMALITY ON MRI

Migraine with prolonged aura can mimic an acute ischemic stroke (AIS), as each may present with accompanying headache and focal neurologic deficits. During the acute episode, perfusion imaging like perfusion-weighted MRI (PWI) can show focal abnormalities corresponding to the neurologic deficits in both entities, making clinical distinction challenging, especially considering the time urgency of AIS treatment. In this case report, we discuss how we utilized hyperacute PWI in conjunction with clinical reasoning to propose the diagnosis of migraine-like headache with prolonged aura.

Case report. A 46-year-old woman presented to the emergency department within 1 hour of sudden onset of bilateral tunnel vision. Over several minutes, this evolved into a right upper quadrant arc-shaped scotoma and then to right homonymous hemianopsia. She also developed a severe nonthrobbing right retro-orbital headache, which progressed to the bilateral occipital area with right face and arm paresthesias. Past history included allergy to iodinated contrast, Ehlers-Danlos type IV, and several years of frequent severe unilateral throbbing headaches with photophobia/phonophobia and nausea as a teenager. She had to miss school, had no aura, and had not been diagnosed with migraine, but her mother carried this diagnosis.

Examination revealed right homonymous hemianopsia, decreased pinprick over the right face and arm, with no motor deficits, nuchal rigidity, or bruits (NIH Stroke Scale = 3). Vital signs and noncontrast head CT were normal. To exclude AIS, we obtained an emergent gadolinium-enhanced MRI with diffusion-weighted imaging, PWI, and magnetic resonance angiogram (MRA) of the head and neck, 150 minutes after symptom onset (figure). The MRI did not demonstrate any area of reduced diffusion, vascular occlusion, abnormal arterial caliber, or dissection. Thrombolytic was not administered and the remaining magnetic resonance sequences revealed normal fluid-attenuated inversion recovery (FLAIR). After the MRI, the patient's headache severity increased, with development of photophobia, severe

nausea, and vomiting. These were treated with metoclopramide and naproxen, with resolution of all symptoms except hemianopsia over the next hour. Meanwhile, the raw perfusion images were processed to provide segmented and thresholded maps, facilitating better quantitative and qualitative assessment of any perfusion abnormality. While relative cerebral blood flow (rCBF) and relative cerebral blood volume (rCBV) maps were near normal, mean transit time (MTT) was slightly prolonged in the left occipital lobe. This focal hypoperfusion was best appreciated on the maximum time to peak of the residue function (Tmax) map. Substantial territory exhibited a Tmax >4 seconds, but minimal territory had a delay of ≥6 seconds. Dilated pial vessels were seen over the same area on contrast-enhanced T1 (T1C+), without any parenchymal or meningeal enhancement (figure). The patient's vision finally returned to normal 14 hours after onset and she subsequently remained asymptomatic. A repeat MRI done 12 days later was normal (figure).

Discussion. The distinction among migraine, AIS, and TIA in our patient was initially challenging due to the lack of prior similar migraine attacks with aura, the prolonged presence of focal deficits mimicking left posterior cerebral artery (PCA) territory ischemia, and a risk factor for arterial dissection due to Ehlers-Danlos syndrome. However, the bilateral onset of visual symptoms, their occipitofugal progressive nature, and the distribution of visual and sensory deficits involving face and arm suggested involvement beyond the PCA territory, and could be explained by migrainous cortical spreading depression.¹ When blood flow to a region is reduced due to narrowed vessel caliber, there is delayed contrast arrival time. Tmax and MTT maps are exquisitely sensitive to hemodynamic changes and mild increases in these, along with normal rCBF and rCBV, likely indicate benign hypoperfusion (or oligemia) without implying that brain tissue is at immediate risk of infarction.² This pattern of benign focal unilateral oligemia, typically in the occipital cortex, followed by pial vessel dilatation,³ as seen in our patient, has been reported on PWI and PET during migraine with^{4,5} and without aura^{5,6} and is thought to be due to corti-

Figure MRI illustrating pertinent findings during the attack of headache and right homonymous hemianopsia (A and B) and at 2-week follow-up (C and D)

The baseline MRI is done at 1.5 T and the follow-up MRI at 3 T. (A) At 150 minutes after symptom onset, axial isotropic diffusion-weighted imaging ($b = 1,000 \text{ s/mm}^2$) (a) is normal without ischemic injury. The circle of Willis on the time-of-flight magnetic resonance angiogram (MRA) (b) 155 minutes after symptom onset is also normal with symmetric arterial calibers. Axial fluid-attenuated inversion recovery (FLAIR) sequence at 170 minutes (c) is also normal. At 205 minutes, the T1 postcontrast "equilibrium" sequence (d) shows pial vessels dilatation (arrow), overlying the left occipital area. (B) Perfusion images at 210 minutes after symptom onset show symmetric relative cerebral blood volume (rCBV) map (a) and relative cerebral blood flow (rCBF) map (b) through the occipital lobes. The mean transit time (MTT) map (c) and the maximal time to peak of the residue function (T_{max}) map (d) show mild delay in contrast arrival time in the left occipital pole. Using the RAPID automated software, the post-processed thresholded and segmented T_{max} map (e) highlights 9.5 mL of tissue in blue with T_{max} > 4 seconds, indicating mild tissue hypoperfusion (benign oligemia). Only scattered and patchy areas within the area of mild hypoperfusion have a T_{max} ≥ 6 seconds (currently accepted threshold^{2,7} for significant tissue ischemia) on the T_{max} map (d) and these represented less than 3 mL of volume per RAPID (e). This pattern of normal rCBV, rCBF, mildly prolonged MTT and T_{max} indicates only mild hypoperfusion without risk of progressing to infarction.^{2,7} Similar perfusion changes were seen in 3 additional contiguous slices through the left occipital pole. (C) Follow-up MRI at day 12 shows a normal axial isotropic diffusion-weighted imaging ($b = 1,000 \text{ s/mm}^2$) (a), normal time-of-flight MRA (b), and normal axial FLAIR (c), without any resultant tissue injury. The improved conspicuity of arteries on this MRA is due to higher magnet strength (3T). The T1 postcontrast sequence (d) shows complete resolution of the prior pial vasodilation. (D) On perfusion imaging, the rCBV (a) and rCBF (b) maps continue to be normal. MTT (c), T_{max} (d), and RAPID (e) maps show complete resolution of the left occipital hypoperfusion.

cal spreading depression. This is distinct from stroke-related hypoperfusion, which is seen as changes on all the perfusion maps.⁷

Based on the above clinical reasoning with supporting changes on perfusion MRI, we assumed migraine-like headache with prolonged aura. Our impression was later confirmed by resolution of PWI, MRA, and T1C+ abnormalities without residual tissue injury on subsequent imaging. We emphasize that, when interpreting hyperacute perfusion imaging, caution should be exercised to avoid misinterpreting areas of focal benign oligemia as ischemic penumbral tissue. Finally, it is to be noted that our patient did not meet the International Headache Society criteria for a definite diagnosis and that migraine with prolonged aura increases future risk of stroke.

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