

Vertical Meridian Sparing Homonymous Hemianopia due to an Occipital Infarct

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A previously healthy 20-year-old man developed acute visual field loss followed by a severe headache, dizziness, and nausea that resolved after few hours. The patient presented to the emergency department 10 hours later. Ophthalmic examination showed a visual acuity of 20/20 in each eye (both eyes). The pupils were normally reactive in both eyes, and there was no anisocoria or relative afferent pupillary defect. Automated perimetry (Humphrey visual field) showed a right congruous and vertical meridian sparing homonymous hemianopia (Fig. 1A, B). The remainder of the eye examination was normal in both eyes.

A computed tomography scan of the head without contrast showed hypodensity in the left occipital region. MRI scan of the brain including diffusion-weighted imaging (DWI) showed a subacute posterior cerebral artery (PCA) ischemic stroke of the left medial occipital lobe, splenium of the corpus callosum, and dorsal left thalamus (Fig. 2). A full neurologic examination was normal.

Cardiac evaluation showed a right-to-left shunt through a patent foramen ovale (PFO), and he was started on dual antiplatelet therapy and a statin medication. Two months later, he underwent endovascular cardioform occlusion therapy to close the PFO. The remainder of the stroke evaluation was unremarkable including carotid, hypercoagulable state, and vasculitis evaluation. The visual field defect remained stable, the patient developed no recurrent symptoms or signs, and he was referred to low-vision rehabilitation.

DISCUSSION

The vertical meridian sparing homonymous hemianopia has classically been described as localizing to the lateral

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geniculate nucleus (LGN). The unique blood supply and anatomy of the LGN account for the localizing nature of the visual field defect. The LGN, located posterolaterally in the thalamus, is the main input to the visual cortex and receives crossed nasal and uncrossed temporal visual field input from the optic tract. The LGN is a wedge-shaped nucleus comprising 6 layers with fibers from the ipsilateral retina ending in layers 2, 3, and 5 and fibers from the contralateral retina ending in layers 1, 4, and 6 (1). In addition, the LGN has a dual blood supply from both the anterior choroidal artery (AChA) and lateral posterior choroidal artery (LPChA). The AChA arises from the internal carotid artery, and LPChA arises from the P2 segment of the PCA. The lateral and medial portions of the LGN, supplied by the AChA, correspond to the superior and inferior (i.e., wedge sector sparing) hemifields. Lesions (usually ischemic infarction) of the AChA, typically result in loss of vision of these hemifields, producing a horizontal meridian sparing or “quadruple” homonymous hemianopia (Fig. 1). By contrast, the hilum of the LGN is supplied by the LPChA (also referred to as the lateral

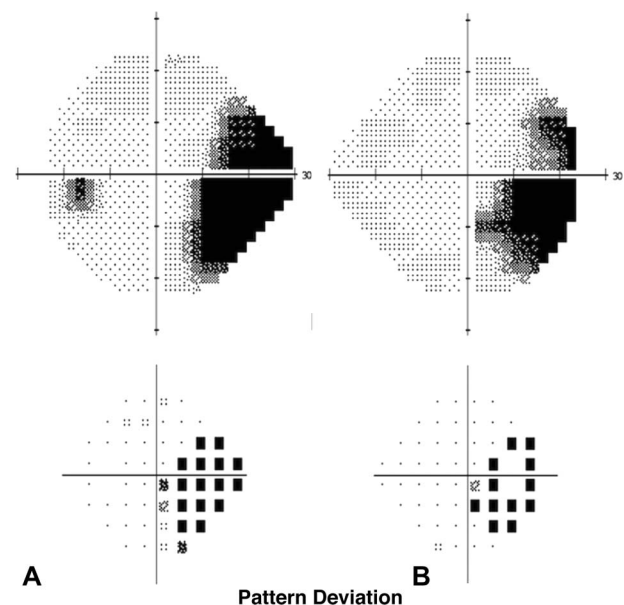


FIG. 1. (A, B) Humphrey visual field testing with 24-2 threshold shows right congruous, vertical meridian sparing homonymous hemianopia.

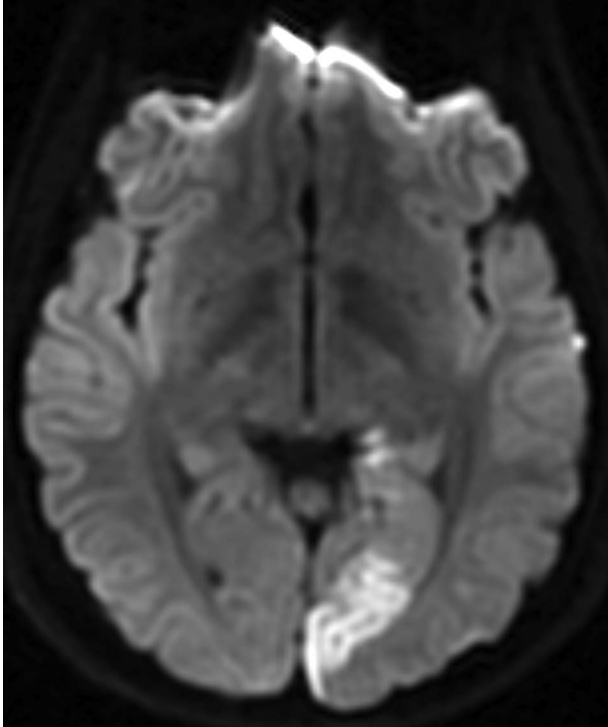


FIG. 2. Axial diffusion-weighted image. MRI of the brain shows the subacute posterior cerebral artery (PCA) ischemic stroke involving the left medial occipital lobe.

choroidal artery), and damage in this location corresponds to the vertical sparing or “wedge-shaped” homonymous hemianopia (as seen in our patient) with sparing of the superior and inferior portions of the hemifield (supplied by the AChA) (2).

In addition, patients with LGN damage often have additional localizing neurological signs and symptoms (e.g., contralateral hemisensory or hemiparetic complaints). Also, patients with LGN involvement may develop a localizing form of optic atrophy (band atrophy). Retinal nerve fiber layer atrophy can occur from retrograde degeneration. For example, a lesion in the right LGN may produce a left homonymous hemianopia (a left eye temporal hemianopic visual field defect combined with a right eye nasal hemi-

anopic visual field defect). The eye with the temporal hemianopic defect (in this example, the left eye) would have nasal fiber loss and demonstrate band atrophy. Likewise, the eye with the nasal hemianopic defect would have temporal fiber loss and more diffuse (nonband type) optic atrophy (3). In contrast to an LGN lesion, an occipital lobe lesion would not produce band optic atrophy.

Although there have been previous reports of occipital lobe (4–7) and temporoparietal lobes (8), this is an example of an acute homonymous hemianopia with sparing of the vertical meridian due to an occipital “stroke in the young” patient due to an underlying PFO. These reports, including our patient, challenge the traditional notion that the vertical meridian sparing homonymous hemianopia is absolutely localizing to the LGN. Nevertheless, clinicians should be aware of the unique anatomy of the LGN in this setting, however.

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