A 67-year-old man developed visual loss. His visual field shows a right homonymous hemianopia. Are there characteristics of the field defect or any associated symptoms that might provide a clue as to the localization of the lesion? What type of imaging study should be performed and how urgently should it be done? Is there any proven treatment for homonymous visual field defects?

Homonymous hemianopia refers to a field defect caused by a lesion of the visual system posterior to the optic chiasm. When discovered as an acute finding, as in your patient, it is a sign of serious intracranial disease that requires emergency evaluation.

The decussation of nasal retinal fibers at the optic chiasm unites in the visual pathway, each eye’s representation of the contralateral hemifield of vision (Figure 36-1). Consequently, damage to the optic tract, lateral geniculate nucleus, optic radiations, or visual cortex produces an overlapping pattern of visual field loss in each eye, which usually respects the vertical meridian. Patients are sometimes unaware of a fresh hemianopia or erroneously attribute their symptoms to monocular visual loss. Careful testing of the visual fields is the crux of accurate diagnosis. Perimetry should be obtained in any patient complaining of unexplained visual loss. My preference is the Humphrey 24-2 SITA program because it provides threshold data in only a few minutes per eye. Although it does not test the peripheral field, the risk of missing a hemianopia is small because the representation of the central field is highly magnified in the brain. In fact, the central 24 degrees occupy 70% of the postchiasmal visual pathway. If computerized perimetry is not available immediately, the visual fields should be tested manually, at least by finger confrontation.
Hemianopia is usually incomplete; the amount of similarity between the pattern of visual field loss in each eye is referred to as the “congruity.” The degree of congruity is not a reliable criterion for localizing the site of pathology, perhaps because many lesions involve both the optic radiations and visual cortex. In any case, localization based on exam findings is moot because all patients with hemianopia require neuroimaging. I would refer this patient to the hospital for urgent brain scanning and inpatient neurological evaluation.

Any process that can injure the brain can give rise to a homonymous hemianopia (Table 36-1). The most common etiology is stroke. It can be caused by hemorrhage, embolus, thrombosis, dissection, or vasculitis. In patients with stroke involving the middle cerebral territory, there are often accompanying signs, such as hemiplegia, hemianesthesia, dysphasia, or stupor. In contrast, patients with stroke from involvement of the posterior cerebral artery may have no findings except for hemianopia. In this setting, the diagnosis can be missed by the ophthalmologist who neglects visual field testing. Sometimes the patient may report an episode of acute vertigo, numbness, or diplopia, suggesting an embolus that has become lodged in a posterior cerebral artery after traveling up the basilar artery.

Patients who are evaluated within 3 hours of the onset of symptoms may be candidates for treatment with intravenous tissue plasminogen activator. In practice, few patients with isolated hemianopia receive medical care within this time frame because they do not realize the seriousness of their predicament. Nonetheless, urgent neurologic evaluation is still advisable to reduce the likelihood of stroke progression or occurrence of a second event. In fact, even if hemianopia is not present on an office exam, a reliable history of a transient ischemic attack that produced temporary hemianopic visual loss should trigger prompt referral.

What type of imaging study should be obtained? Noncontrast computerized tomography is low cost, rapid, and available in any emergency room. It is excellent for exclusion of hemorrhagic stroke and especially suitable in the setting of head trauma. However, magnetic resonance imaging (MRI) is more sensitive for the detection of nonhemorrhagic...
ischemia from stroke, as well as identification of other lesions such as demyelinating plaques, tumors, and infections. Figure 36-2 shows the visual fields from a patient who reported a 10-day history of a shadow in her vision on the left side. Computed tomography revealed no abnormality. Regular T2-weighted MRI sequences were also normal (Figure 36-3A). However, fluid-attenuated inversion recovery (FLAIR) sequences showed an infarct involving the lower, right calcarine visual cortex in the territory of the lingual

Table 36-1

Causes of Homonymous Hemianopia*

- Stroke (embolic, thrombotic, hemorrhagic, vasculitic, dissection)
- Tumor (primary or metastasis)
- Trauma
- Infection (bacterial, fungal, viral, parasitic)
- Arteriovenous malformation
- Nonorganic visual loss
- Demyelination
- Migraine
- Congenital malformation
- Perinatal hypoxic or hemorrhagic injury
- Posterior reversible leukoencephalopathy
- Neurosurgery; occipital lobe retraction syndrome
- Eclampsia
- Dementia
- Epilepsy
- Mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke (MELAS)
- Drug toxicity (cyclosporine, tacrolimus, sirolimus)
- Nonketotic hyperglycemia

*Stroke, tumor, and trauma account for more than 95% of cases.
artery (Figure 36-3B). MR angiography showed partial occlusion of the proximal right posterior cerebral artery (Figure 36-3C). Another advantage of MRI is that fresh infarcts can be detected sooner than by computed tomography. Diffusion-weighted MRI demonstrates cytotoxic edema within 30 minutes of stroke onset. Computed tomography, as well as standard T1 and T2 MRI sequences, require many hours before any abnormality becomes visible.

If noncontrast studies show no evidence of stroke, gadolinium should be administered to the patient. It increases the sensitivity of MRI for the detection of tumors and infections. Once the etiology of the homonymous hemianopia is established through review of the patient’s history, findings, and neuroimaging studies, a focused laboratory evaluation is appropriate. A stroke work-up usually includes echocardiography, vascular imaging, outpatient electrocardiographic monitoring, and selected hematological studies. For tumor, a biopsy is usually performed. Removal of a solitary metastasis may be advisable, depending on the patient’s overall prognosis.

Naturally, the prognosis for homonymous hemianopia depends on its etiology. For a few months after trauma or a stroke, improvement can occur spontaneously, especially in patients with an incomplete hemianopia. Surgery for a tumor, arteriovenous malformation, or infectious lesion sometimes worsens a hemianopia by damaging surrounding brain tissue. Occasional patients with homonymous hemianopia have macular sparing, usually from collateral blood flow via the middle cerebral artery to the occipital pole, where central vision is represented. These individuals are able to read normally and adapt better to their handicap than patients with macular splitting. Unfortunately, rehabilitation strategies for patients with hemianopia based on prisms, visual exercises, or computer training are not effective. Patients with a complete hemianopia must be told explicitly by the ophthalmologist that it is not safe or legal to drive. Patients with a partial hemianopia should be retested by the state motor vehicle division to assess their ability to drive.

Figure 36-3. (A) Coronal T2-weighted MR scan from the patient with the partial left homonymous hemianopia illustrated in Figure 36-2. The study appears virtually normal. (B) MR FLAIR image showing an ischemic stroke (arrow) involving the lower bank of the calcarine fissure, where the upper quadrant of the visual field is represented in the primary (striate) visual cortex. (C) MR angiogram showing partial occlusion (arrow) of the right posterior cerebral artery, presumably from an embolus.
Summary

- The prognosis for homonymous hemianopia depends on the specific underlying etiology.
- Post-traumatic, hemorrhage related, or ischemic stroke patients may demonstrate spontaneous improvement over time especially in incomplete hemianopias.
- Surgery for a tumor, arteriovenous malformation, or infectious lesion sometimes worsens a hemianopia by damaging surrounding brain tissue.
- Rehabilitation strategies for patients with homonymous hemianopia may be considered but prisms, visual exercises, or computer training have to date not been proven to be effective.

References
