involvement of the optic chiasm, resulting in a junctional scotoma—a description that we have not seen to date in the English literature.

Medical management involves antiplatelet treatment, although the evidence supporting this practice is limited. Surgical revascularization is offered as well and is often pursued in pediatric patients who are more likely to have the progressive form of the disease.[4] Revascularization can be either direct or indirect. Direct involves anastomoses created between the superficial temporal artery and the middle cerebral artery. Indirect involves the use of implanted vascular tissue to promote angiogenesis in the implanted area.[5]

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Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this article.

Crossed-quadrant homonymous hemianopsia in a monocular patient

A 75-year-old male presented to the clinic reporting that upon waking up from a nap 4 days earlier, he noticed he could "only see in certain spots." He has an ocular history of moderate-stage primary open-angle glaucoma in both eyes, dry eye syndrome, and pseudophakia. He had previously undergone enucleation of his blind, painful right eye due to neovascular glaucoma from a previous central retinal vein occlusion. He was a former smoker, and his medical history is significant for multiple myeloma, bladder cancer, pulmonary embolism, hypertension, and hyperlipidemia.

On initial examination, his visual acuity was 20/25-2 OS with intraocular pressure of 13 mm Hg OS and 3 out of 11 colour plates. He wore a prosthesis OD. Examination of the anterior segment and fundus of his left eye was stable from previously documented examinations, including careful examination of the optic nerve. Optical coherence tomography imaging of the retina was within normal limits. Confrontational visual field testing with a colored target revealed possible left hemi-field loss. Humphrey Central 24-2 Threshold visual field testing revealed new deficits that were not present on testing performed 3 months prior, as seen in Figure 1.

Despite the patient's monocular status, it was believed that the automated perimetry demonstrated a combination of a left superior quadrantanopsia with a right inferior quadrantanopsia resembling a crossed-quadrant homonymous hemianopsia (CQHH). The patient was sent to the emergency department for urgent stroke evaluation and neurology consultation. Magnetic resonance imaging of the brain revealed bilateral acute occipital lobe infarcts—one infarct located along the left superior calcarine bank, and one infarct located along the right inferior calcarine bank, as demonstrated in Figure 2.

Discussion

The unique entity of CQHH or "checkerboard visual field defect" is exhibited when consecutive or simultaneous occipital lobe lesions occur superior to the calcarine fissure and inferiorly on the contralateral hemisphere.[1] Automated perimetry in our monocular patient disclosed a pattern resembling a CQHH or the striking "checkerboard visual field defect"—in this case, the juxtaposition of a left superior quadrantanopsia and a right inferior quadrantanopsia. This pattern is essentially pathognomonic for bilateral occipital lesions.

A suspicion of CQHH should prompt urgent stroke evaluation, particularly in an elderly patient such as ours with vasculopathic risk factors as well as multiple myeloma and bladder cancer, further increasing his risk of thromboembolism. The most common cause of CQHH, as in our patient, is an ischemic or hemorrhagic infarct that may be associated with a thromboembolic event, dissection, or cervical vertebral trauma, but CQHH has also been reported with tumors, migraine, syphilis, necrotizing ependymomyelitis, and demyelinating disease.[1–4] Most of the cerebral infarcts that cause CQHH are derived principally by embolization of the calcarine arteries from the P3 branch of the posterior cerebral artery.[2] Our patient had bilateral occipital lobe infarcts

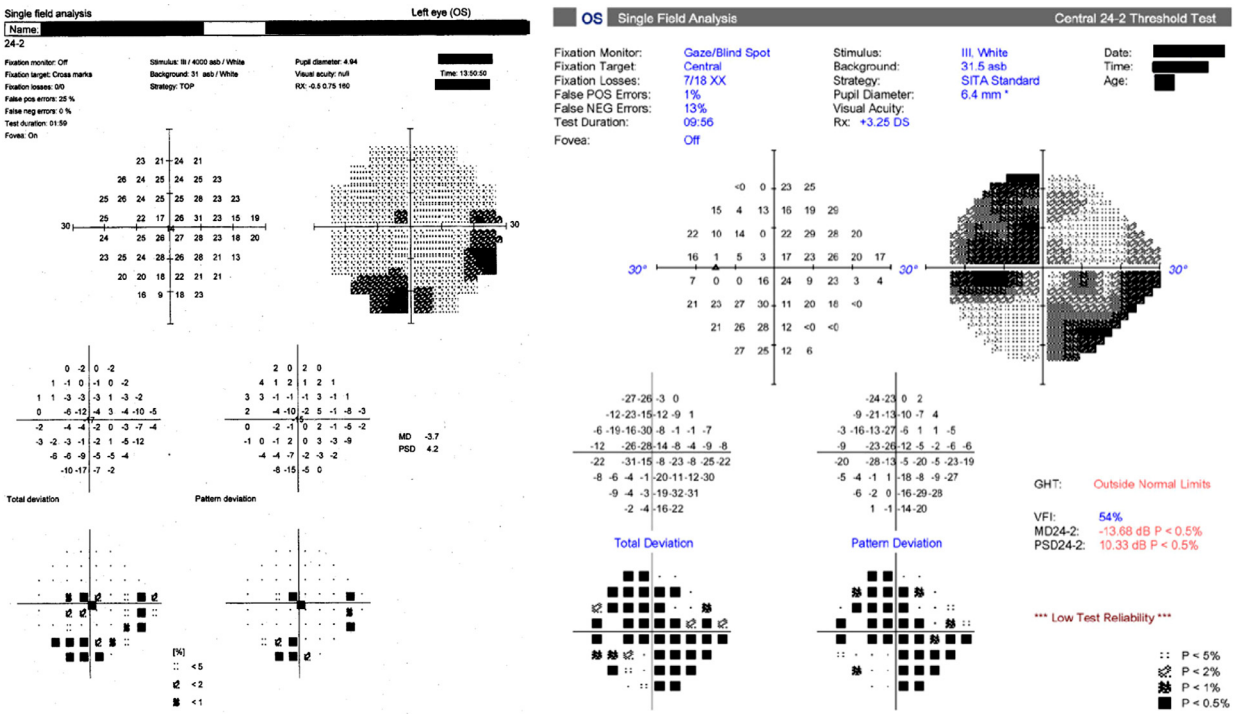

Fig. 1—Humphrey Central 24-2 Threshold visual field testing of the left eye from 3 months prior to presentation (left) and day of presentation (right). The visual field demonstrated the juxtaposition of a left superior quadrantanopsia and a right inferior quadrantanopsia resembling a crossed-quadrant homonymous hemianopsia.

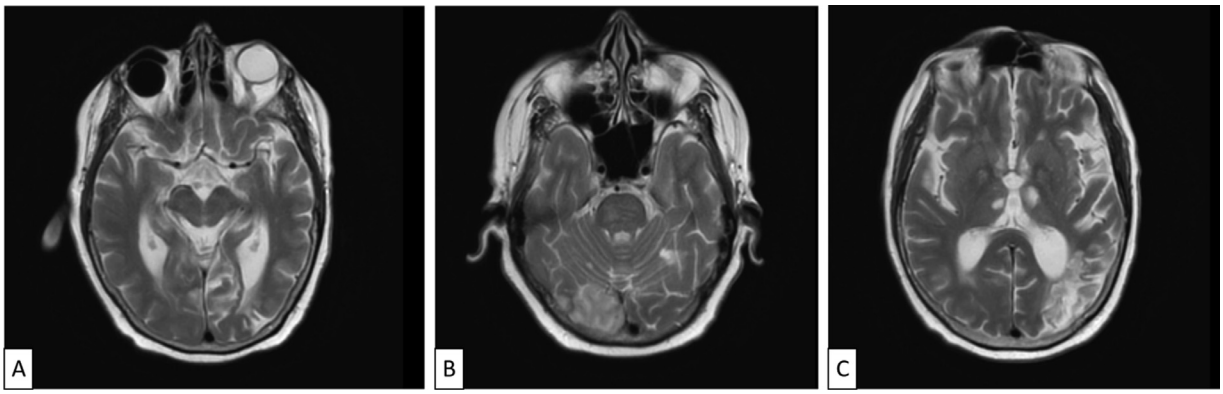

Fig. 2—Magnetic resonance imaging shows bilateral occipital lobe infarctions. The left infarct is superior to the calcarine fissure (A) and the right infarct is inferior to the calcarine fissure (B). The patient was also found to have a remote infarct of the left parieto-occipital lobe (C).

located along the left superior calcarine bank and right inferior calcarine bank.

To the best of our knowledge, we describe the first report of a CQHH diagnosed in a monocular patient. From this case, several important clinical pearls should be considered. First, the possibility of a homonymous field defect should always be considered in monocular patients with new visual field deficits. Second, in patients with glaucoma who present with new visual field loss, the clinician should always consider whether the visual field deficit corresponds with the appearance of their optic nerve. Third, visual field deficits can be detected with increased sensitivity on confrontational visual field testing by using a red target instead of a white target or counting fingers. This is consistent with the assertion by Pandit et al. that use of a small red target is the most sensitive method for examination of central 20° visual field and was crucial to the prompt diagnosis in this patient.[5] Lastly, the clinician should have a low threshold for obtaining formal visual field testing in an expedited fashion, particularly in patients with significant vasculopathic risk factors and when the ophthalmologic exam cannot explain the visual field deficit.

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Footnotes and Disclosure

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Written patient consent was obtained, and all patient identifiers were removed from the submission. Full adherence to the Declaration of Helsinki and all Federal and State laws.

Murine typhus presenting as pseudotumor cerebri

Murine typhus is an acute infection transmitted by rodent or cat fleas carrying gram-negative, obligate intracellular bacteria, *Rickettsia typhi*. This vector for transmission is most often carried on rodents, but opossums are thought to be a reservoir in suburban settings.[1] Associated with overcrowding, pollution, and poor hygiene, murine typhus has been documented worldwide, but the majority of cases in the United States are in Texas and California.[2]

Symptoms of murine typhus can be nonspecific, and they typically appear 7–14 days after infection. Patients most often present with recurrent fever, maculopapular rash, and headache, but arthralgia, cough, and abdominal pain can also be present.[2,3] Neurological symptoms are extremely rare, occurring in only 2%–5% of cases, and historically include altered mental status, seizures, or aseptic meningitis.[3]

We present a rare case of intracranial hypertension, papilledema, and visual disturbance secondary to *Rickettsia typhi* infection. To our knowledge, only three cases of this nature have been reported to date.

Case Report

A 31-year-old African American woman presented with acute, painless, binocular loss of vision. She also had a ten day history of acute intractable vomiting with nausea, chills, recurrent fevers up to 103°F, neck stiffness, myalgias, diarrhea, and left-upper-quadrant abdominal pain. She reported headaches behind the left eye with radiation to the left occipital region, which was noticeably different from her usual migraine pattern. On examination, neck pain was elicited in the occipital area, but there was no neck stiffness or Brudzinski sign. Past medical history was significant for hypertension, diabetes mellitus type II, migraines, and obesity (body mass index 34.3). She had no recent travel, animal exposure, or sick contacts.

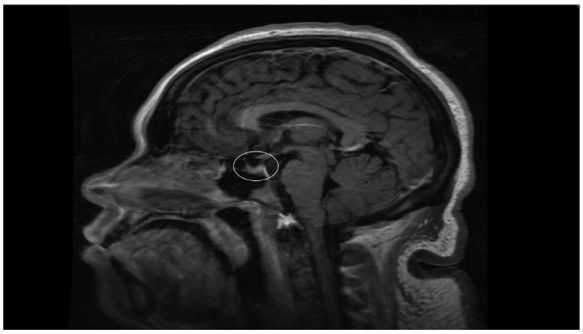

Fig. 1—Sagittal T1 Fluid Attenuated Inversion Recovery (FLAIR) MRI of the brain showing a partially empty sella (encircled).

Cranial magnetic resonance imaging (MRI) of the head and magnetic resonance venography with contrast were normal (Figs. 1 and 2). MRI of the spine was normal. Laboratory testing revealed an elevated C-reactive protein of

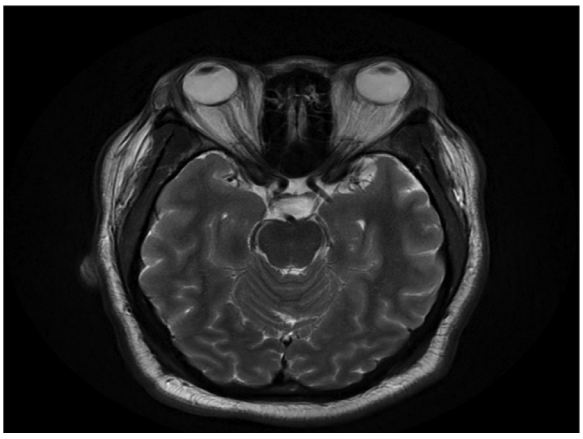

Fig. 2—Axial T2 Fast Spin Echo (FSE) MRI of the brain showing lack of optic nerve tortuosity.