Cecocentral scotoma as the initial manifestation of subacute bacterial endocarditis

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Introduction: We report a case of a 67-year-old male who presented with a cecocentral scotoma caused by a septic embolus from subacute bacterial endocarditis (SBE).

Methods: A 67-year-old man presented with sudden, painless decreased vision in the left eye. A dilated fundoscopic exam, Humphrey visual field test, transthoracic echocardiogram, abdominal computed tomography (CT), and blood cultures were all performed.

Results: A dilated fundoscopic exam revealed temporal segmental optic disc pallor on the left, and Humphrey visual field testing demonstrated a dense left cecocentral scotoma. When the patient developed fever (103.9°F) and palpitations, transthoracic echocardiogram revealed valvular vegetations, and contrast CT of the abdomen revealed an abscess in the dome of the liver likely due to an infectious thrombus. Blood cultures grew viridians group streptococci in three separate peripheral collections.

Conclusion: This case illustrates that a sudden cecocentral scotoma may be the initial manifestation of SBE.

Keywords: endocarditis, scotoma, streptococcal infections, visual fields

Introduction
Subacute bacterial endocarditis (SBE) is an indolent microbial infection of the endocardium with the potential for systemic dissemination by way of septic emboli. We report the case of a 67-year-old male who presented with a cecocentral scotoma caused by an infectious embolus from SBE.

Case report
A 67-year-old man presented to our emergency department (ED) complaining of sudden, painless decreased vision in the left eye for 3 days. He reported no other systemic or ocular symptoms, including no fever, weight loss, pain on chewing, joint pain, or skin rashes. Other than treatment for dental caries 1 week prior to presentation, he had not sought medical attention for over 10 years. On examination, his visual acuity was 20/20 on the right and count fingers on the left. Extraocular movements were full bilaterally. Pupillary examination revealed a 2+ afferent pupillary defect on the left. The patient correctly identified nine of nine Ishihara color plates on the right but only four of nine color plates on the left. Anterior segment examination was otherwise within normal limits, as were the intraocular pressures. Dilated fundoscopic exam revealed sharp, nonedematous, nonglaucomatous-appearing optic disks bilaterally but with temporal segmental pallor on the left. The remainder of the peripheral posterior exam was within normal limits, including no Roth or cotton wool spots,
no Hollenhorst plaques, no retinal edema or hemorrhage, and no neovascularization. Other than a mildly elevated temperature to 100.1°F, all other vital signs were within normal limits, and there were no focal neurologic findings. Laboratory testing revealed a sedimentation rate of 80 and a white blood cell count of 11,000/µL with 78% neutrophils. Platelet count was 225,000/µL, and the hematocrit and basic metabolic panel were within normal limits.

As computed tomography (CT) and/or magnetic resonance imaging (MRI) were not acutely available, we performed Humphrey visual field testing, which demonstrated a dense left cecocentral scotoma (Figure 1). The differential diagnosis of a pale nerve with a cecocentral scotoma includes an embolic event, optic neuritis, toxic optic neuropathy, and nutritional deficiency. The patient was admitted to the intensive care unit for further medical workup, including further imaging and blood studies. However, he left the hospital against medical advice within 1 day of admission.

Seventy-two hours later, the patient returned to the ED with increased temperature (103.9°F), palpitations, and stable decreased visual acuity of the left eye and was therefore readmitted. Transthoracic echocardiogram revealed valvular vegetations (not shown), and contrast CT of the abdomen revealed an abscess in the dome of the liver that was likely due to an infectious thrombus (Figure 2). Chest X-ray and CT of the head showed no focal lesions. The patient’s blood cultures grew *Streptococcus viridans* in three separate peripheral collections. Following intravenous antibiotic treatment, his systemic condition steadily improved over 1 week, warranting discharge from the hospital. However, his visual acuity and field deficits remained unchanged. The patient continued oral antibiotic therapy for 3 additional weeks until repeat blood cultures showed no evidence of residual infection.

On serial yearly follow-up examinations almost a decade after the original insult, the patient’s low vision status on the left persists with decreased Snellen vision, reproducible red desaturation, and decreased color plates on that side. Yearly follow-up Humphrey visual field testing has remained unchanged. Follow-up intravenous fluorescein angiography has remained unremarkable, with recent fundus photos of the left eye (Figure 3) showing inferotemporal disk pallor. Follow-up MRI was not acquired, as the patient’s examination and visual defect remained stable over many years. We postulate that an infectious embolus from endocarditis impeded circulation, causing ischemia and segmental infarction of the left optic nerve and affecting the maculopapillary bundle.

**Discussion**

We report the first case of a cecocentral scotoma presenting as the initial manifestation of SBE. It is important for both
Cecocentral scotoma as initial manifestation of endocarditis

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