Toxoplasma Neuroretinitis

BRIAN MAC GRORY, MB, BCh, BAO, MRCP; DERRICK CHENG, BSc, BA; ADENIYI FISAYO, MD

A 38-year-old female presented with painless monocular vision loss in her left eye. She was otherwise entirely healthy and took no medications. Physical examination revealed a visual acuity of 20/20 in her right eye and 20/50 in her left eye. Her pupils constricted from 4mm to 3mm bilaterally and there was a left relative afferent pupillary defect. Her visual fields were full on the right side and she had a constricted superonasal and inferonasal visual field. She exhibited dyschromatopsia being able only to see the test plate on Ishihara testing (scored 0/14). Dilated ophthalmoscopy revealed severe swelling and hemorrhage of the left optic disc with surrounding subretinal swelling extending to the macula (**Figure 1**).

Examination of the contralateral eye was entirely within normal limits including no evidence of remote infection. Serum testing for inflammatory etiologies (including ACE, anti-aquaporin-4 antibody, ANCA and serum protein electrophoresis) and infectious etiologies (including bartonella antibodies, HIV antibodies, VDRL, lyme antibodies, leptospira antibody, Quantiferon Gold and mumps antibodies) was normal. Cerebrospinal fluid analysis demonstrated no leukocytosis or oligoclonal banding. Magnetic resonance imaging (MRI) of her brain performed with intravenous gadolinium administration demonstrated no enhancement of her left optic nerve or evidence of demyelination in her brain. She was discharged with a diagnosis of idiopathic neuroretinitis.

When seen in follow-up 18 days later, her visual acuity had worsened – she was only able to count fingers in the left eye. Fundoscopic examination demonstrated vitreous cells, optic disc whitening, serous retinal detachment and phlebitis along the superior temporal arcade. An anterior chamber tap was performed. Clindamycin, trimethoprim-sulfamethoxazole and prednisone were commenced for empiric treatment of toxoplasma neuroretinitis. Toxoplasma PCR testing was positive in her aqueous fluid. Of note, she had no systemic manifestations of toxoplasmosis, including no fever, chills, malaise, headache, myalgia, rash or lymphadenopathy. Additionally, she ate only fully cooked meat and did not consume untreated water or raw vegetables. She did not have a cat in her home. At a follow-up visit 2 weeks later, her visual acuity had improved and fundoscopic examination was notable for a "macular star" (**Figure 2**).

Figure 1. Fundoscopic examination on the date of initial presentation. This image depicts severe swelling and hemorrhage of the left optic disc with surrounding subretinal swelling extending to the macula.

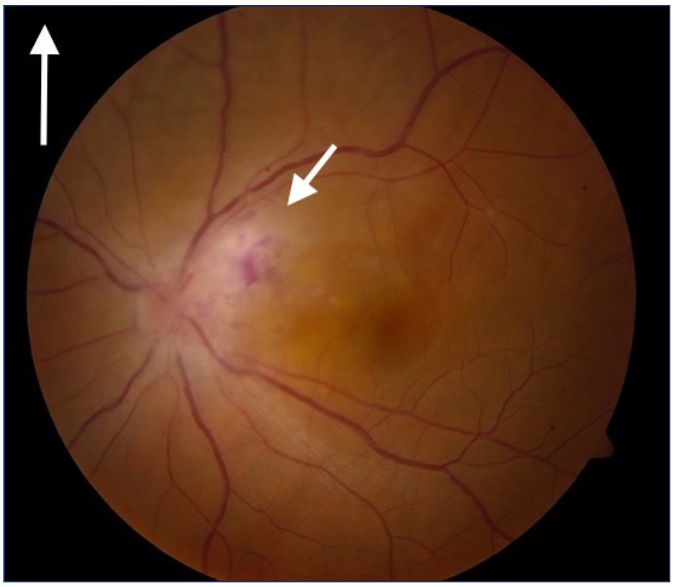
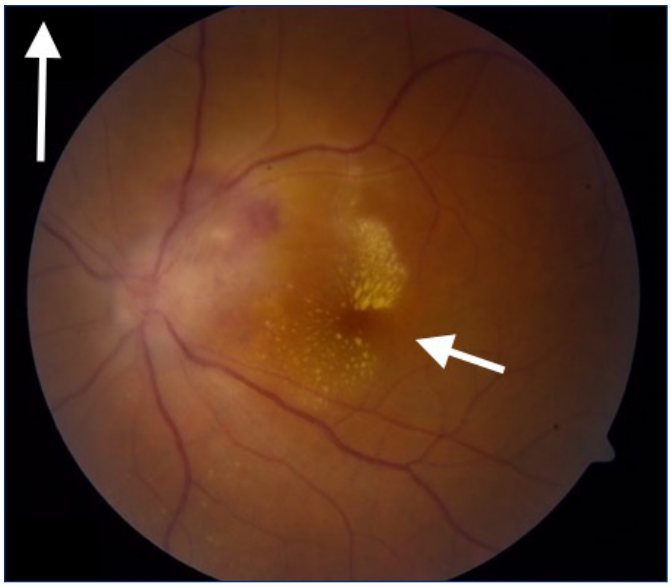


Figure 2. Fundoscopic examination at the time of last follow-up. This image depicts an exudative material in a radial pattern around the fovea. This so-called "macular star" is a characteristic sequel of toxoplasma neuroretinitis.



This case illustrates the importance of recognizing that significant optic disc swelling is atypical in demyelinating optic neuritis and the need to consider neuroretinitis in the work-up of painless monocular visual loss. Neuroretinitis describes inflammation of the vasculature in the region of the optic disc with ensuing retinal edema.[1] The findings on ophthalmoscopy are often difficult to distinguish from other cases of optic disc edema – such as raised intracranial pressure. There is no relationship between the development of neuroretinitis and a future risk of multiple sclerosis.[2]

The differential etiology of neuroretinitis includes bacterial, viral, fungal, protozoal and spirochaetal infection as well as systemic vasculitidies and autoimmune disorders (such as sarcoidosis and polyarteritis nodosa). There are also a minority of cases in which recurrent idiopathic neuroretinitis occurs. Bartonella henselae ("cat scratch disease") accounts for two-thirds of all cases attributed to infection. Reactivation of toxoplasma gondii is a recognized cause of neuroretinitis and can be seen even in the immunocompetent[3] and is known for causing bilateral and, in some cases, recurring neuroretinitis.

References

1. Purvin V, Sundaram S, Kawasaki A. Neuroretinitis: review of the literature and new observations. *Journal of Neuroophthalmology*. 2011; 31:58-68.
2. Parmley VC, Schiffman JS, Maitland CG, Miller NR, Dreyer RF, Hoyt WF. Does neuroretinitis rule out multiple sclerosis? *Arch Neurol*. 1987; 44:1045-1048.
3. Rayess N, Rahimy E, Dunn JP. Bilateral Neuroretinitis and Retinal Hemorrhages in an Immunocompetent Patient. *JAMA Ophthalmology*. 2014;132(10):1243-1244.

Authors

Brian Mac Grory, MB, BCh, BAO, MRCP, Department of Neurology, Warren Alpert Medical School of Brown University, Providence, RI.

Derrick Cheng, BSc, BA, Warren Alpert Medical School of Brown University, Providence, RI.

Adeniyi Fisayo, MD, PharmD, Departments of Neurology and of Ophthalmology and Visual Science, Yale University School of Medicine, New Haven, CT

Correspondence

brian_macgrory@brown.edu

IMAGES IN CLINICAL MEDICINE

Original, high-resolution images which have not been published elsewhere will be considered for publication. Submit 2–4 images.

Submissions should include:

Brief title: 8 words or less

Content: Relevant clinical information, findings, clinical course, and response to treatment if initiated. Limit: 400 words

Legends: All labeled structures in the image should be described and explained in the legend. Any identifying information should be removed from the image.

Author information: Names, professional degree, academic/hospital affiliations, address, email and telephone number.

Send to:

RIMJ editors-in-chief

William Binder, MD
william_binder@brown.edu

Edward Feller, MD
edward_feller@brown.edu

Cc: Mary Korr, managing editor
mkorr@rimed.org