NEURORETINITIS FROM CAT SCRATCH DISEASE

Your patient's furry friend may be the source of sudden, painless vision loss.

BY ALDEN NEGAARD, OD

Neuroretinitis is a condition characterized by inflammation of the neurosensory retina and optic nerve. Clinical signs typically include optic nerve edema, along with edema of the peripapillary retina and macula, which classically present with a macular star pattern of hard exudates. The etiology of neuroretinitis can be split broadly into either infectious or noninfectious/inflammatory. The most common identifiable cause is cat scratch disease (CSD), which results from infection by the *Bartonella henselae* bacterium and accounts for roughly two-thirds of neuroretinitis cases.[1] Other potential infectious causes include syphilis, Lyme disease, tuberculosis, toxoplasmosis, toxocariasis, herpes simplex virus, and herpes zoster virus.[2] Potential inflammatory etiologies to consider include sarcoidosis, lupus, polyarteritis nodosa, and inflammatory bowel disease.[2,3] In some cases, serology testing is negative; imaging should be considered before labeling these as idiopathic neuroretinitis.

ABOUT CSD-NR

CSD affects about 22,000 individuals in the United States annually, or about 6.6 cases per 100,000 each year, with an average onset of 25 to 35 years of age.[4] Importantly, 5% to 13% of patients with CSD experience ocular involvement about 1 to 2 weeks following the fever and infection onset.[5] Ocular presentations of CSD include CSD neuroretinitis (CSD-NR), panuveitis, and Parinaud oculoglandular syndrome. Kittens are more likely than adult cats to be the source of transmission, and a scratch, bite, or saliva making contact with a wound can transmit the disease. It is not uncommon for cats to harbor the organism, as *Bartonella henselae* bacteremia has been documented in 30% to 40% of domestic and adopted shelter cats.[6]

In CSD-NR, inflammation and fluid emanate from the optic nerve, and hard exudates within the fluid are deposited into Henle layer in the characteristic macular star pattern due to radial arrangement of the nerve fibers. Disc edema is typically the first sign, followed by macular edema and star exudates, which usually present 1 to 2 weeks after nerve edema onset.[3] In addition to optic nerve edema and macular star, CSD-NR may also present with a focal or multifocal retinitis in the form of small, discrete yellow-white spots of retinal inflammation often outside of the posterior pole and, occasionally,

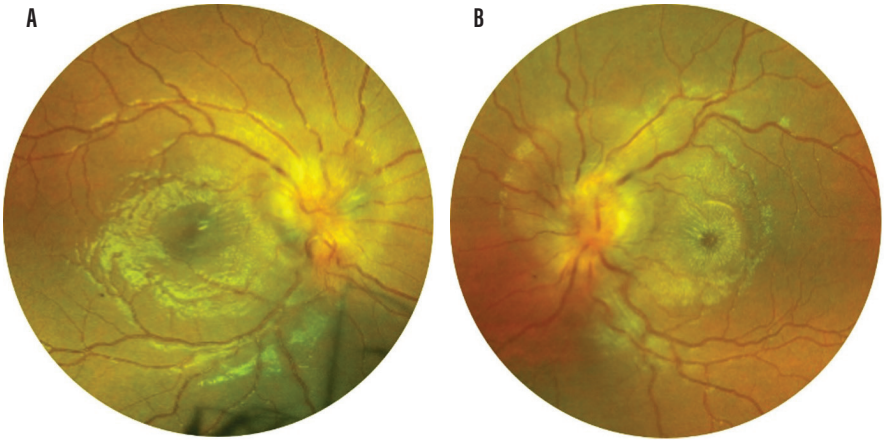

Figure 1. Color fundus photography of the right (A) and left (B) eye at the initial visit showed optic nerve edema and partial star exudate pattern in the macula.

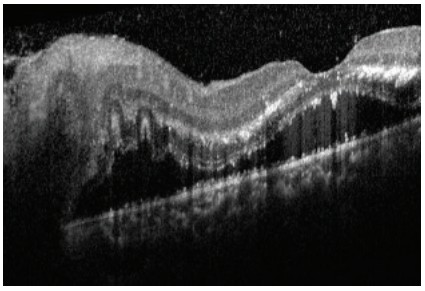

Figure 2. OCT of the left eye at initial presentation showed hard exudates in the outer plexiform and Henle fiber layer, subretinal fluid extending from the optic nerve through the fovea, and vitreous cell.

in the fellow eye.[7] This finding can be a useful clue that CSD is the cause. CSD-NR typically presents unilaterally with sudden-onset vision loss, and afferent pupillary defect is seen in two-thirds of cases.[8]

Vision in patients with CSD-NR varies depending on the amount of disc and macular edema and may be anywhere from 20/20 to light perception; when present, visual effects are often profound initially, as more than half of patients present with VA of 20/200 or worse.[5]

PROGNOSIS AND MANAGEMENT

Prognosis is typically quite good, with 94% of patients showing VA improvement to 20/40 or better.[3,5] However, resolution of macular edema can take weeks, and the exudates can often linger for months to years, causing vision issues if located centrally in the fovea. Patients may also be left with nerve pallor and reduced contrast sensitivity. Although more rare than unilateral cases of CSD-NR, bilateral cases are certainly possible. In such cases, the list of differentials must be expanded to include grade 4 hypertensive retinopathy and papilledema, both of which require a different, more emergent workup and treatment path. Therefore, be sure to check the patient's blood pressure in the office and ask about symptoms to help differentiate papilledema from CSD-NR, such as headache, diplopia, or transient vision dimming or loss lasting seconds, which would raise suspicion of elevated intracranial pressure.

Because neuroretinitis due to *Bartonella* is typically a self-limiting infection in patients who are immunocompetent, the decision to prescribe medication remains controversial. There is very little literature showing that treatment improves final visual outcome.[9] However, evidence does show that use of certain antibiotics, particularly doxycycline, rifampin, or azithromycin, can shorten the infection course and potentially speed up visual recovery. In theory, a shorter infection course should lessen the nerve edema course and, thus, reduce the risk of continued hard exudate deposition in the fovea, which can cause lasting visual issues. Some data show that a combination of antibiotics and corticosteroid was more effective than antibiotic alone, but the general consensus is more research is needed on the effectiveness of corticosteroids in such cases.[10] A 4- to 6-week treatment course with an antibiotic, such as doxycycline, particularly if implemented earlier in the disease process, should be

AT A GLANCE

- Neuroretinitis, a condition characterized by inflammation of the neurosensory retina and optic nerve, is considered either infectious or noninfectious/inflammatory.
- The most common identifiable cause of neuroretinitis is cat scratch disease (CSD-NR), which results from infection by the *Bartonella henselae* bacterium.
- Disc edema is often the first sign of CSD-NR, followed by macular edema and star exudates, which typically present 1 to 2 weeks after onset of optic nerve edema.
- Assuming the patient history supports a diagnosis of CSD-NR, initiating treatment with antibiotics can help speed up visual recovery while waiting on serology results to confirm.

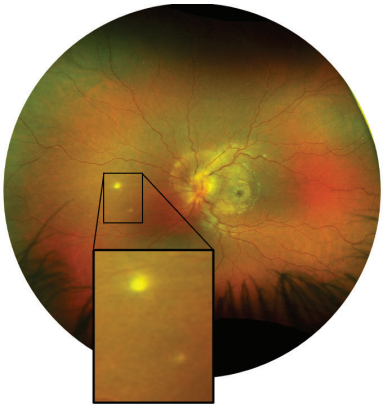

Figure 3. Fundus photography of the left eye at initial presentation showed multifocal lesions of retinitis.

considered in cases where there is prominent vision loss on presentation.[9]

Of note, continued treatment may be needed for up to 4 months for patients who are immunosuppressed, and a collaborative care approach with an infectious disease specialist would be wise.

CASE EXAMPLE

A 17-year-old Hispanic male presented with a chief complaint of sudden, painless profound vision loss OU that started 1 week earlier and was worse in his left eye. Review of systems revealed the patient had a low-grade fever 2 weeks prior. He was not on any systemic or ocular medications, and his medical history was unremarkable. His ocular history was remarkable only for myopia, and his family history was significant for diabetes and hypertension. He had no history of recent travel or trauma and no family history of ocular disease.

At presentation, the patient's BCVA was 20/60 OD and counting fingers at 5 ft OS. His IOP was 12 mm Hg OD and 14 mm Hg OS, and his blood pressure was 117 mm Hg/71 mm Hg. Examination of extraocular muscles revealed full range of motion without pain or diplopia. His pupils were equally round and showed sluggish reactions to light, as well as trace relative afferent pupillary defect OS. Confrontation fields revealed a central scotoma OS. Anterior segment examination was unremarkable.

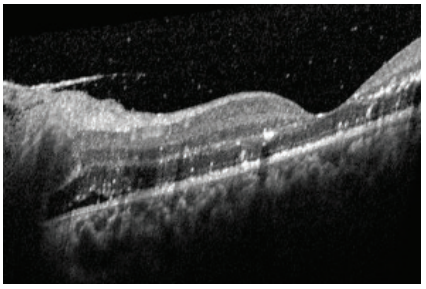

Figure 4. OCT of the left eye after 2 weeks on oral doxycycline 100 mg twice daily showed improvement in previous findings.

Posterior segment examination revealed disc edema that was greater in the left eye and macular edema with star pattern exudates OS and partial star pattern OD (Figure 1). OCT showed trace vitreous cell and subretinal fluid (Figure 2). Two multifocal white lesions of retinitis were noted OS nasally (Figure 3).

Getting to the Root

Upon further questioning, the patient reported that his family fostered cats and kittens, and he regularly played with the new kittens. He denied any headache, diplopia, tinnitus, or transient vision loss.

Labs ordered included complete blood count with differential, *Bartonella* species antibodies (ie, IgG and IgM) with reflex to titers, and rapid plasma reagin with reflex fluorescent treponemal antibody absorption test. Serologic testing revealed *Bartonella henselae* IgG and IgM levels of greater than 1:1024 (≥ 1:256 suggests current infection) and 1:128 (≥ 1:16 suggests current infection), respectively.

Management

We discussed the likely diagnosis of CSD-NR given the clinical picture and history, and the decision was made to start oral doxycycline 100 mg twice daily for 4 weeks while awaiting serology results. At the 2-week follow-up visit, the patient's vision had improved from 20/60 OD to 20/30 OD and from counting fingers OS to 20/60 OS with resolution of the relative afferent pupillary defect and subretinal fluid (Figure 4). He was seen again 2 weeks later after completing the course of doxycycline, at which point his nerve edema had resolved, although exudate deposits remained in the left fovea. His VA was 20/25 OD and 20/50 OS, likely due to remaining exudates. He was scheduled to return in 1 month but was lost to follow up.

PATIENT HISTORY IS KEY

Unilateral disc edema with macular star exudates is a classic characteristic of neuroretinitis. Keep CSD-NR high on the differentials list in cases of unilateral or bilateral neuroretinitis, especially in younger patients. Don't forget to look beyond the posterior pole for focal white chorioretinal lesions, signifying active *Bartonella* retinitis. When there is a supportive history of the patient being scratched or bitten by a cat, such as in our case, antibiotics can be initiated to speed up visual recovery while waiting on serology results to confirm the presence of elevated *Bartonella* antibodies. ■

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ALDEN NEGAARD, OD
- Optometrist and Residency Supervisor, Georgia Eye Partners, Atlanta, Georgia
- Adjunct Faculty Member, Southern College of Optometry, Memphis, Tennessee
- Alden.Negaard@gaeyepartners.com
- Financial disclosure: None