PPID | Private Practice Infectious Disease

MIDC METRO INFECTIOUS DISEASE CONSULTANTS

Cat-Scratch Disease—Uncovering the Cause of Progressive Visual Loss in a 56-Year-Old Man

Morgan Scully [1,*]

[1] Metro Infectious Disease Consultants, Huntsville, AL 35801, USA
* Corresponding author: morgan.c.scully@gmail.com

Submitted: 23 April 2025, accepted: 20 May 2025, published: 30 September 2025

Abstract: Cat-scratch disease (CSD) is a zoonotic illness caused by the bacterium *Bartonella henselae* that is transmitted to humans via cats, fleas, and lice. Cat-scratch disease often presents as localized lymphadenitis, but occasionally can also cause neurologic, hepatosplenic, valvular and ocular disease. Identification of infection can be challenging due to nonspecific presentation of these syndromes. We offer a case of a 56-year-old male who presented with prolonged fevers and new vision loss in the setting of exposure to cats and kittens and was found to have *Bartonella* neuroretinitis. We discuss the clinical presentation, relevant epidemiology, and diagnostic and treatment challenges. This case highlights important considerations regarding high clinical suspicion for *Bartonella* neuroretinitis.

Keywords: cat-scratch disease; neuroretinitis; *Bartonella henselae*

How to cite: Scully, M. Cat-Scratch Disease—Uncovering the Cause of Progressive Visual Loss in a 56-Year-Old Man. *Priv. Pract. Infect. Dis.*, 2025, 5(3): 8; doi:10.55636/ppid05030008.

1. Introduction

Cat-scratch disease (CSD), caused by *Bartonella henselae*, is primarily associated with localized lymphadenitis and, less commonly, ocular manifestations such as neuroretinitis. *Bartonella* neuroretinitis is a rare but well-documented cause of vision loss, often misdiagnosed as other conditions like optic neuritis due to its nonspecific presentation [1]. In the current literature, early diagnosis and prompt antibiotic treatment are emphasized to prevent irreversible vision loss and expedite recovery.

This case emphasizes the diagnostic complexities of *Bartonella* neuroretinitis in a 56-year-old male who presented with fever, headaches, and progressive visual impairment over a several-week

period. The patients history of a recent kitten adoption provided potential clues to the underlying etiology. However, the lack of lymphadenitis on exam and the predominant presenting symptom of headache prompted an initial clinical suspicion of temporal arteritis. Empiric treatment with steroids and antibiotics were initiated, and the patient did have improvement in his symptoms. A thorough infectious disease workup, including serology testing, confirmed the diagnosis of *Bartonella* neuroretinitis. This condition demands a broader differential diagnosis in similar presentations of fever and visual disturbances, particularly in patients with relevant exposure histories.

The patients successful treatment with a combination of doxycycline and rifampin, resulting in gradual improvement in vision, aligns with existing recommendations in the literature. However, the case demonstrates the importance of considering *Bartonella* neuroretinitis and emphasizes the role of serologic testing in confirming the diagnosis. Furthermore, it highlights the potential benefit of combining antibiotics with corticosteroid therapy in achieving visual recovery, while also raising awareness of the potential for incomplete visual recovery despite appropriate treatment. This case illustrates the need for continued research on the optimal management of *Bartonella* neuroretinitis as well as increased clinical suspicion in patients presenting with systemic symptoms and vision loss.

2. Case Presentation

A 56-year-old presented to the hospital with a one-month history of fevers. Over the prior one to two weeks, he had experienced worsening bilateral frontotemporal headaches accompanied by lethargy, malaise, and night sweats. Four days prior to his presentation to the emergency department, he began noticing floaters in his right eye that progressively impaired his central vision, which prompted him to seek medical attention at the emergency room.

The patient had no significant past medical or surgical history and was not on any home medications. He worked as an engineer and resided in the southern United States. He traveled to Denver four months prior to attend a funeral. As a Desert Storm veteran, he retired from the military 15 years prior. He led an active lifestyle, frequently biking and hiking outdoors, and did recall a tick bite six months prior to hospitalization. He fostered cats and dogs at home. He denied the use of alcohol, tobacco, or illicit drugs.

On presentation, the patients vital signs included an oral temperature of 39.5 °C, heart rate of 125 beats per minute (peripheral), respiratory rate of 18 breaths per minute, blood pressure of 119/68 mmHg, and oxygen saturation of 97% on room air.

Initial physical examination revealed a well-developed, well-nourished male in no acute distress. The head and neck exam showed no masses, thyromegaly, or lymphadenopathy, and no nuchal rigidity, though there was mild bilateral scalp tenderness. The eyes showed no redness or drainage. The chest wall showed no tenderness or deformities. Lungs were clear to auscultation bilaterally, and heart examination revealed a regular rate and rhythm with physiologic S1 and S2, without murmurs. The abdomen was soft and non-tender to palpation, with no palpable masses and positive bowel sounds. The musculoskeletal exam demonstrated no deformities, with the patient moving all four extremities without difficulty. Peripheral pulses in the lower extremities were normal bilaterally, and there was no clubbing, cyanosis, or edema noted in the extremities. Neurologically, the patient had no focal deficits, and his skin was warm and dry, without any rashes, ulcerations, or lesions.

Laboratory data results revealed levels of sodium of 136 mmol/L, chloride of 97 mmol/L, blood urea nitrogen of 16 mg/dL, potassium of 4.7 mmol/L, bicarbonate of 26 mmol/L, creatinine of 0.9 mg/dL, and glucose of 91 mg/dL. The complete blood count showed a white blood cell count of 12,000 cells/mm^3, hemoglobin of 13.5 g/dL, hematocrit of 39.2%, and platelets of 546,000 units/mcl. Liver function tests revealed a total bilirubin of 0.4 mg/dL, direct bilirubin of 0.2 mg/dL, total protein of 6.8 g/dL, albumin of 3.5 g/dL, alkaline phosphatase of 262 U/L, alanine aminotransferase of 48 U/L, and aspartate aminotransferase of 25 U/L. Inflammatory markers were elevated, with a C-reactive protein (CRP) of 13.3 mg/L and an erythrocyte sedimentation rate (ESR) of 49 mm/h. Both COVID-19 polymerase chain reaction (PCR) and influenza antigen tests were negative. A viral hepatitis panel

was negative, and HIV antigen/antibody testing was also negative. Blood cultures returned negative as well. Cerebrospinal fluid analysis revealed two total nucleated cells, less than one red blood cell, glucose of 60 mg/dL, and protein of 35 mg/dL, all within normal limits. A computed tomography scan of the chest, abdomen, and pelvis with intravenous contrast showed no acute abnormalities. Transthoracic echocardiogram demonstrated no valvular abnormalities and no evidence of vegetation.

Given the fevers, vision loss and headaches, the initial clinical concern was for temporal arteritis, and rheumatology and ophthalmology were consulted. A fundoscopic examination revealed bilateral optic nerve disc edema with blurred disc margins and a pale appearance in both eyes. The patient was started on high-dose intravenous methylprednisolone 250 mg and initiated on ceftriaxone and doxycycline. He underwent a temporal artery biopsy. Within 24 hours of starting steroids and antibiotics, the patient experienced significant improvement, with resolution of fevers. He was subsequently discharged on a steroid taper alone. As an outpatient, his vision began to improve. The temporal artery biopsy returned negative and the diagnosis of temporal arteritis was reconsidered.

Initial infectious serology testing on presentation revealed negative results for cytomegalovirus, Epstein–Barr virus, *Ehrlichia*, anaplasmosis, Rocky Mountain spotted fever, murine typhus, and Q fever. Additional testing showed negative serologies for *Histoplasma*, *Blastomyces*, *Coccidioides*, and *Cryptococcus*, as well as negative urine *Histoplasma* antigen and beta-D-glucan assays. Serologies for *Aspergillus*, *Brucella*, and *Leptospira* were also negative. Syphilis serology testing was negative. However, the patients initial serology showed a *Bartonella henselae* IgG titer of 1:512 and IgM of less than 1:20, as well as a *Babesia microti* IgG titer of 1:256.

Repeat serology performed four weeks later demonstrated a negative *Babesia microti* IgG, while the *Bartonella henselae* IgG titer decreased to 1:256, and the *Bartonella henselae* IgM remained less than 1:20. *Babesia microti* and *Bartonella henselae* PCR tests were negative on repeat testing.

The patient was diagnosed with *Bartonella* neuroretinitis. The *Babesia microti* serology was thought to be a false positive. He completed a 6-week course of rifampin and doxycycline, with gradual improvement in vision, although some visual defects persisted. Additionally, he was slowly tapered off oral steroids. There was no recurrence of fevers or other systemic symptoms after the initial presentation. Repeat inflammatory markers, including CRP and ESR, returned to normal. Notably, further probing revealed that the patient had adopted a new kitten a few months prior to the onset of his symptoms, which is a known risk factor for *Bartonella* infection.

3. Discussion

Bartonella henselae is a gram-negative intracellular bacillus responsible for approximately 22,000 cases per year in the United States. Infection typically results from contact with a young cat, often a kitten, through a scratch, lick, or bite, or from a flea or louse. Over 90% of cats under one year of age have positive serology for *Bartonella*, though they are usually asymptomatic. The bacterium is spread among cats via fleas, which bite an infected cat and ingest the bacteria. They then shed bacteria in their feces onto the cats fur or onto a human wound. Cats become infected when grooming their fur with their teeth and claws [2].

In humans, the most common clinical presentation is self-limited regional lymphadenitis, but the infection can disseminate, leading to more severe forms, including hepatosplenic cat-scratch disease and neurologic cat-scratch disease. Neurologic manifestations can include encephalopathy, transverse myelitis, radiculitis, and cerebellar ataxia. *Bartonella henselae* can also cause culture-negative endocarditis. Ocular involvement occurs in 5% to 10% of infected individuals, which can lead to conditions such as Parinauds ocular glandular syndrome, optic neuritis, and neuroretinitis [1].

Ocular cat-scratch disease commonly presents as Parinauds ocular glandular syndrome. This condition is characterized by granulomatous conjunctivitis on one side, accompanied by swollen lymph nodes in front of the ear on the same side. Cat-scratch disease is the most frequent cause of

Parinauds syndrome, though it can also be associated with herpes simplex virus type 1, *Francisella tularensis*, and *Paracoccidioides brasiliensis* infection [3].

Another ocular manifestation of CSD is neuroretinitis, which often occurs without lymphadenopathy and is the leading cause of infectious neuroretinitis. It typically presents as painless vision loss and is the most common infectious cause of this condition. Neuroretinitis is often mistaken for optic neuritis, a more common cause of vision loss overall, due to the similarities in their clinical presentation [4,5].

The treatment of cat-scratch disease neuroretinitis typically involves a combination of doxycycline 100 mg twice daily and rifampin 300 mg twice daily for four to six weeks. This recommendation is based on a small case series involving seven patients. Alternative regimens include rifampin combined with either azithromycin or trimethoprim–sulfamethoxazole for four to six weeks [1].

During treatment, there is a possibility of a Jarisch–Herxheimer reaction, an inflammatory response to the rapid killing of bacteria. The use of corticosteroids remains a consideration in treatment as well, though their role in managing neuroretinitis has not been definitively established and may vary depending on the severity of the condition [1].

In a retrospective case series of 86 patients with ocular CSD, the use of corticosteroids in combination with antibiotics showed improved outcomes in vision recovery. Specifically, 87% of patients treated with both antibiotics and steroids experienced an improvement in vision loss, compared to 50% of those treated with antibiotics alone. Combination therapy was more frequently administered to patients with more severe visual acuity loss. Additionally, three patients who received steroids without antibiotics also showed improvement in vision, suggesting that steroids may play a beneficial role in managing neuroretinitis from CSD, particularly in severe cases [5].

4. Conclusions

This case emphasized several things in the diagnosis and treatment of *Bartonella* neuroretinitis. Clinicians must maintain a high index of suspicion for *Bartonella* infections, particularly in patients with relevant animal exposure. The diagnosis of neuroretinitis should prompt thorough infectious serologic testing to rule out less common causes of vision loss, such as *Bartonella henselae*. The case also reinforces the role of combination antibiotic therapy in treating *Bartonella* neuroretinitis, with studies showing improved visual outcomes when antibiotics are used alongside corticosteroids in severe cases. However, more research is needed to optimize treatment regimens, including the potential benefits of corticosteroids, as outcomes can vary and some patients may experience incomplete visual recovery despite timely therapy. Future studies should explore the long-term visual prognosis in presentations of *Bartonella* neuroretinitis and further investigate the role of combination therapies in enhancing patient outcomes.

Funding: This research received no external funding.

Conflicts of Interest: The author declares no conflict of interes.

References

1. Ksiaa, I.; Abroug, N.; Mahmoud, A.; Zina, S.; Hedayatfar, A.; Attia, S.; Khochtali, S.; Khairallah, M. Update on Bartonella neuroretinitis. *J. Curr. Ophthalmol.* **2019**, *31*, 254–261. [CrossRef] [PubMed]
2. Klotz, S.A.; Ianas, V.; Elliott, S.P. Cat-scratch Disease. *Am. Fam. Physician* **2011**, *83*, 152–155. [PubMed]

3. Habot-Wilner, Z.; Trivizki, O.; Goldstein, M.; Kesler, A.; Shulman, S.; Horowitz, J.; Amer, R.; David, R.; Ben-Arie-Weintrob, Y.; Bakshi, E.; et al. Cat-scratch disease: Ocular manifestations and treatment outcome. *Acta Ophthalmol.* **2018**, *96*, e524–e532. [CrossRef] [PubMed]
4. Messina, F.; Doria, R.; Gabriellini, G.; Sartini, M.S.; Tascini, C.; Menichetti, F. *Bartonella henselae* neuroretinitis. *Clin. Microbiol. Infect.* **2001**, *7*, 387–388. [CrossRef] [PubMed]
5. Saxena, R.; Gajjar, N.; Karnath, B.; Zhou, Y. Bartonella Neuroretinitis: There Is More to Cat Scratch Disease than Meets the Eye. *Am. J. Case Rep.* **2023**, *24*, e938380-1–e938380-5. [CrossRef] [PubMed]