

A Case of Neuroretinitis in a Young Male Triggered by Cat Scratch Disease

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ABSTRACT

Neuroretinitis is a potentially vision-threatening condition characterised by the swelling of the optic disc, followed by the development of a macular star due to the spread of fluid to the surrounding peripapillary retina. The present case report describes a rare case of unilateral neuroretinitis in a 36-year-old male who presented with an acute, painless decrease in vision in the right eye. The patient exhibited classic signs of neuroretinitis, such as optic disc oedema and a macular star. A systemic work-up ruled out most infectious and inflammatory causes, while serological tests for Cat Scratch Disease (CSD) showed highly reactive results. He was immediately started on appropriate oral antibiotics and steroids for six weeks, after which he experienced significant visual recovery. Managing neuroretinitis involves early investigation, addressing the underlying infection and mitigating the inflammatory response. Challenges include the need for accurate and timely diagnosis, often requiring serological tests and imaging studies. Notably, the present case underscores the importance of considering *Bartonella henselae* infection in the differential diagnosis of neuroretinitis, particularly in patients with a history of cat exposure. The rarity of such presentations in otherwise healthy young individuals highlights the need for increased awareness and prompt diagnostic evaluation to ensure optimal outcomes and prevent long-term visual impairment.

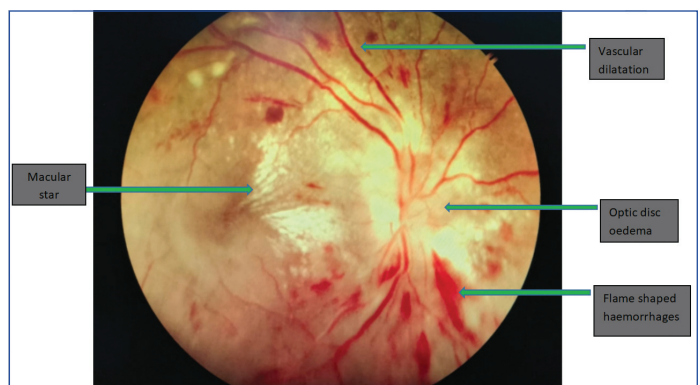
Keywords: *Bartonella henselae*, Macular star, Optic disc oedema, Stellate maculopathy, Visual impairment

CASE REPORT

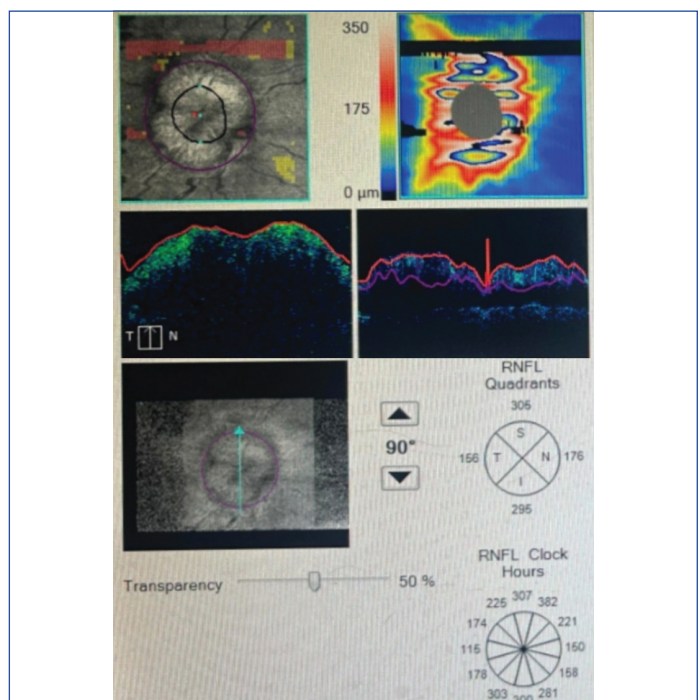
A 36-year-old male farmer from a rural area of western Maharashtra, India, came to the Ophthalmology Clinic with a three-day history of sudden vision loss in his right eye. Three weeks earlier, he had experienced symptoms of headache, fever and muscle pain but did not have vomiting, cold, cough, or any ocular problems at that time. He was treated symptomatically by a local physician and his symptoms resolved within 4-5 days. He reported having close contact with pet animals, mainly cats, on his farm. He has no significant medical history or chronic illnesses and denies any similar conditions in his family.

The vision loss in his right eye began suddenly three weeks after his initial prodromal symptoms, without ocular pain, redness, watering, discharge, floaters, or double vision. Upon examination, the patient exhibited a relative afferent pupillary defect in the right eye, with Best-corrected Visual Acuity (BCVA) of 6/36 in the right eye (OD) and 6/9 in the left eye (OS). He also demonstrated impaired colour vision in the right eye when using the pseudoisochromatic Ishihara chart. The intraocular pressure of both eyes was normal, as measured by a non contact tonometer. Extraocular movements were full and free in all directions.

The slit-lamp examination revealed a normal anterior segment of the right eye. However, an indirect ophthalmoscope examination of the dilated fundus in the right eye showed a highly oedematous optic disc with indistinct margins and the cup-to-disc ratio and neuroretinal rim could not be appreciated due to intense disc oedema [Table/Fig-1]. There were multiple flame-shaped haemorrhages along all four vascular arcades, extending up to 2-3 disc diameters from the optic disc. Three yellowish-white fluffy lesions were present along the superotemporal arcade, suggestive of cotton wool spots. Macular oedema with exudates extended from the foveal centre to the optic disc. There was marked dilatation of the first and second-order blood vessels. The retinal periphery appeared normal despite the significant findings in the posterior pole [Table/Fig-1]. The anterior and posterior segment evaluations of the left eye were normal. A Spectral Domain Optical Coherence Tomography (SD-OCT) of the optic disc of the right eye was performed [Table/Fig-2].



[Table/Fig-1]: Fundus photograph of right eye showing optic disc oedema and macular star formation.



[Table/Fig-2]: SD-OCT of right eye showing severe optic disc oedema.

A provisional diagnosis of right eye neuroretinitis was made. However, other causes of disc oedema, such as papilloedema, diabetic papillopathy, grade 4 hypertensive retinopathy and anterior ischaemic optic neuropathy, should be excluded. The differential diagnosis was based on history, epidemiological data, systemic manifestations and the pattern of ocular involvement. Laboratory investigations were conducted to rule out possible causative conditions such as tuberculosis, syphilis, sarcoidosis, CSD and viral serology. The complete blood count showed a haemoglobin level of 15 gm/dL and an elevated leucocyte count of 16,000 cells/mm³. The random blood sugar level was 99 mg/dL. Additionally, C-reactive protein was 25 mg/L and the erythrocyte sedimentation rate was 20 mm/hour. The tuberculin skin test and QuantiFERON Tuberculosis (TB) Gold test were negative. Serological tests for Human Immunodeficiency Virus (HIV), hepatitis B and hepatitis C antigens, as well as, the *Treponema pallidum* hemagglutination assay, were negative. However, the serologic test for *Bartonella henselae* using the indirect Immunofluorescent Assay (IFA) was highly positive (1:1190 UI/L) for Immunoglobulin G (IgG) [Table/Fig-3]. The chest X-ray showed no abnormalities.

Test	Result	Reference/Interpretation
Complete Blood Count (CBC)	Haemoglobin: 15 gm/dL Leucocyte count: 16,000 cells/mm ³	Normal range (male): 13-18 gm/dL Elevated (normal range: 4,000-11,000 cells/mm ³)
Random Blood Sugar (RBS)	99 mg/dL	Normal (70-100 mg/dL)
C-reactive Protein (CRP)	25 mg/L	Elevated (Normal: <3 mg/L)
Erythrocyte Sedimentation Rate (ESR)	20 mm/hour	slightly elevated (Normal: <20 mm/hour)
Tuberculin skin test	Negative	-
QuantIFERON TB Gold test	Negative	-
Chest X-ray (PA view)	Normal	-
HIV	Negative	-
HbsAg	Negative	-
HCV	Negative	-
<i>Treponema Pallidum</i> Haemagglutination Assay (TPHA)	Negative	-
<i>Bartonella henselae</i> Immunofluorescent Assay (IgG)	Highly positive (1:1190 IU/L)	Strongly indicative of recent <i>Bartonella</i> infection

[Table/Fig-3]: Laboratory investigations.

PA: Posteroanterior; HbsAg: Hepatitis B surface antigen; HCV: Hepatitis C virus

The diagnosis of CSD-related neuroretinitis was confirmed and the patient was treated with oral doxycycline (200 mg per day) and rifampicin (900 mg per day), along with oral prednisolone initially at 60 mg per day for two weeks, with progressive tapering of oral prednisolone for a total duration of six weeks [1]. The patient was monitored once weekly for the initial two weeks, then biweekly for the next two follow-ups. At the end of six weeks, the BCVA in the right eye improved to 6/18. Fundus examination revealed a significant reduction in disc oedema and macular exudates [Table/Fig-4].



[Table/Fig-4]: Fundus photograph showing reduction in macular exudates and optic disc oedema.

DISCUSSION

Neuroretinitis, characterised by optic disc oedema and macular star formation, often leads to acute vision loss and is an uncommon manifestation of CSD, a zoonotic infection caused by the bacterium *Bartonella henselae*. The incidence of neuroretinitis as a result of CSD varies, but research indicates that CSD may account for up to 1-2% of all neuroretinitis cases [2]. Initially described by Leber in 1916 as 'stellate maculopathy,' this definition was revised by Don Gass in 1977, who observed that disc oedema precedes macular exudates. Gass proposed the term 'neuroretinitis' based on optic disc leakage detected by fluorescein angiography [3,4].

Neuroretinitis is a rare form of optic neuritis, less frequently reported than retrobulbar neuritis and papillitis. It can affect both immunocompetent, as well as, immunocompromised individuals. It typically presents with acute visual loss and patients often report preceding systemic symptoms such as fever and lymphadenopathy. The classic triad of neuroretinitis includes reduced visual acuity, optic disc oedema and a macular star, which develops over several weeks [1]. The macular star results from lipid-rich fluid leaking from the optic disc vasculature into the outer plexiform layer, with the aqueous phase passing through the external membrane to beneath the neurosensory retina, leading to lipid deposition in a star-like pattern around the macula [3]. Atypical presentations can include vitreous inflammation, retinal haemorrhages, or serous retinal detachments. Skrehot HC et al., described such a presentation with haemorrhagic retinal features and serous retinal detachment, highlighting the varied spectrum of ocular manifestations [5].

Causes of neuroretinitis can be idiopathic or associated with infectious conditions like CSD, toxoplasmosis, toxocariasis, tuberculosis, syphilis, Lyme disease, rickettsiosis, dengue fever, sarcoidosis and Behçet's disease [4,6]. CSD in humans is contracted through contact with cats infected with Gram negative bacteria of the genus *Bartonella*. *Bartonella* species are fastidious bacteria, making them challenging to culture from clinical specimens [7]. Consequently, microbiological diagnosis is often based on positive serology for anti-*Bartonella* antibodies, as performed in the present case.

The prognosis for neuroretinitis associated with CSD is generally good, particularly in mild to moderate cases in immunocompetent patients. However, severe disease, especially in immunocompromised individuals, can lead to long-lasting ocular complications [8]. In the present case, the patient was treated with a combination of antibiotic therapy (doxycycline and rifampicin) and systemic corticosteroids for six weeks, resulting in a favourable visual outcome. Avaylon J et al., documented a similar case of cat scratch-associated neuroretinitis in a young female whose initial symptoms were misattributed to idiopathic intracranial hypertension. After subsequent serology confirmed a *Bartonella henselae* infection, treatment with doxycycline and rifampicin led to full visual recovery within three months [2]. Ksiaa I et al., highlighted that *Bartonella* neuroretinitis typically presents with optic disc oedema and a macular star, appearing approximately 1-2 weeks after the initial optic disc swelling. They emphasised the favourable prognosis in immunocompetent individuals, noting that systemic antibiotics combined with corticosteroids can enhance visual outcomes in severe cases [6]. Holdeman NR et al., presented a case of cat scratch neuroretinitis in a 13-year-old female, noting that visual recovery was observed within weeks of initiating antibiotic therapy, although lingering visual distortions were present upon resolution of systemic symptoms [8]. Mahjoub A et al., reported a case of neuroretinitis in a patient with CSD, which presented as unilateral optic disc oedema and macular exudates forming a partial star. The diagnosis was confirmed via positive serology for *Bartonella henselae* and the patient responded well to a combination of doxycycline, rifampicin and corticosteroids, achieving full visual recovery after one year [9]. CSD can be prevented by minimising exposure to cats, particularly in elderly individuals and

those with compromised immune systems, while promoting safe interactions with pets. If a scratch or wound occurs, it should be immediately cleaned with warm, soapy water and the cat should not be allowed to lick the injury. For individuals who become infected, especially those at higher risk of severe illness, it is recommended to test and treat any infected pets. Patients should be advised to consult their veterinarian for appropriate testing and treatment options for their pet cats [7].

CONCLUSION(S)

Neuroretinitis can arise from various infectious and non infectious aetiologies. Early serological testing should be considered alongside primary investigations whenever there is a high suspicion of a specific aetiological organism, given the hallmark signs of neuroretinitis. Accurate diagnosis and identification of the underlying cause, as well as, prompt initiation of targeted treatment, can mitigate several devastating visual impairments, as demonstrated in the present case.

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