



NEURORETINITIS: A COMPREHENSIVE ANALYSIS OF CLINICAL PRESENTATIONS AND ETIOLOGIES

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ABSTRACT

Purpose: To describe the clinical and etiological spectrum of neuroretinitis and treatment outcomes.

Methods: A case series of five patients with neuroretinitis was evaluated for clinical features, investigations, OCT findings, etiology, treatment, and visual outcomes.

Results: Patients aged 5–39 years presented with unilateral visual loss of varying severity. Optic disc edema with macular star was the predominant finding. Etiologies included presumed idiopathic/post-viral disease, sickle-cell disease, ocular tuberculosis, and Bartonella henselae infection. OCT demonstrated optic nerve head swelling and subretinal fluid in the Bartonella case. Etiology-directed antimicrobial therapy and/or corticosteroids resulted in visual improvement in most cases.

Conclusion: Neuroretinitis has a diverse clinical and etiological spectrum. Detailed history, targeted investigations, and multimodal imaging are essential for etiological diagnosis and appropriate management.

KEYWORDS: Neuroretinitis, Optic Disc Edema, Macular Star, Bartonella Henselae, Optical Coherence Tomography, Visual Outcome

INTRODUCTION

Neuroretinitis is a type of optic neuropathy characterized by optic disc edema that leads to the formation of a macular star of exudates. Although the funduscopy appearance is typical, the presentation is not always clear-cut, as it may lack a macular star at the outset. Neuroretinitis may present as an isolated optic neuritis or be part of an underlying infectious, inflammatory, or immune-mediated disease.

The cause of neuroretinitis is diverse. It may be associated with bacterial, viral, parasitic, or systemic infectious, inflammatory, or immune-mediated disease, or it may have an unknown cause. Cases of neuroretinitis associated with Bartonella infection are common,

but they can also be caused by a wide range of other pathogens. Diagnosis is important because treatment can vary from observation and corticosteroids to specific antibiotic therapy, depending on the cause.

The clinical features of neuroretinitis can range from mild vision impairment to loss of central vision. Visual acuity may be reduced with dyschromatopsia and central or paracentral scotomas, and patients may present with optic disc edema and vitreous inflammation and exudative involvement of the macula. Investigations such as optical coherence tomography, fundus fluorescein angiography, neuroimaging, and relevant tests can help determine the diagnosis and possible cause. There is considerable clinical overlap between the

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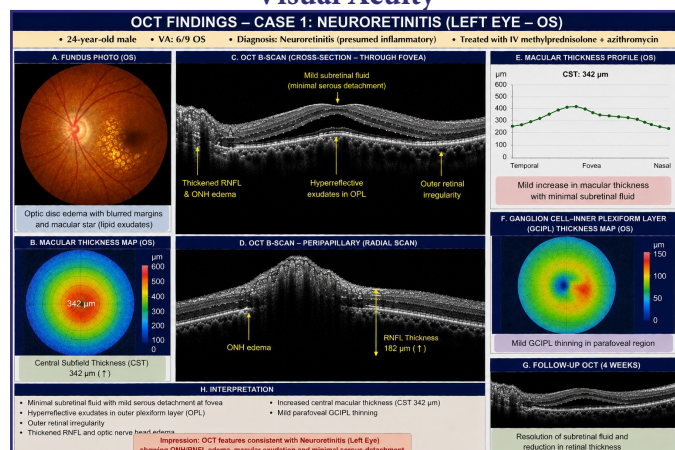
different causes of neuroretinitis, and diagnosis can be challenging.

The heterogeneity of clinical presentations and causative factors in neuroretinitis make it an important differential diagnosis for patients with optic disc edema and vision problems. Characterization of the range of clinical features and causes and their association with each other may help improve recognition and management of this condition.

This study aims to evaluate the range of clinical presentations and ophthalmic examination results and investigations and the relationship between them and the possible causes associated with neuroretinitis in the study cohort.

CASE STUDIES

Case 1: Unilateral Neuroretinitis With Preserved Visual Acuity



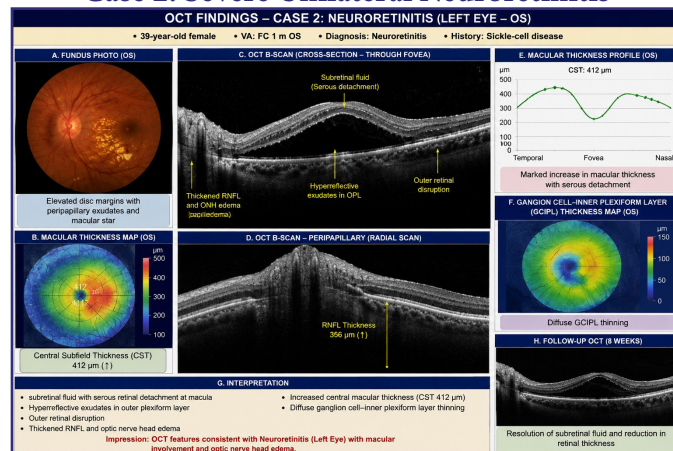
A 24-year-old male presented with unilateral headache and sudden, non-progressive blurring of vision in the left eye (OS). Best-corrected visual acuity (BCVA) was 6/9 OS, with a grade 1 relative afferent pupillary defect. Ocular motility was full and painless. Fundus examination revealed optic disc edema with blurred margins and a macular fan/star configuration. MRI of the brain and orbits showed no optic nerve lesion, with only mild paranasal sinus mucosal changes.

The patient was treated with intravenous methylprednisolone (1 g/day for 3 days), followed by oral azithromycin. Visual acuity improved to 6/6 on follow-up.

Teaching point: Neuroretinitis may present with

relatively preserved central visual acuity despite objective evidence of optic nerve dysfunction.

Case 2: Severe Unilateral Neuroretinitis

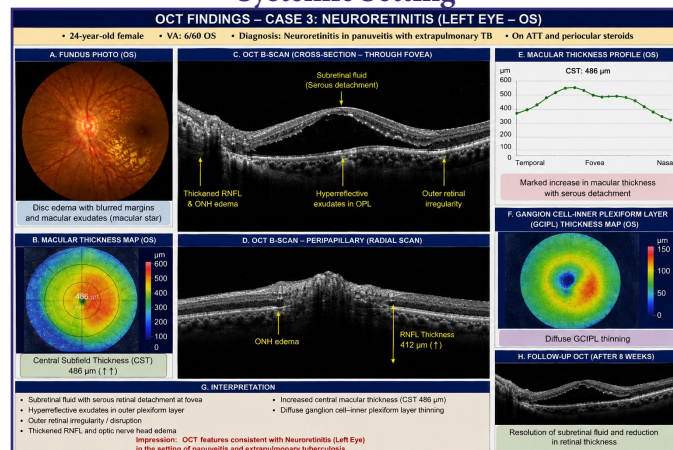


A 39-year-old female came in with sudden decrease in her left eye (OS) vision, associated with a headache and pain on looking to the left for 3 days. Her best corrected visual acuity was finger counting at 1 m OS and 6/6 RE. There was relative afferent pupillary defect in the left eye with eccentric pupil. On fundus examination, there was optic disc edema with surrounding exudates and macular star. The patient had history of sickle-cell disease. Routine investigations including sickling test were done.

The patient was treated with intravenous methylprednisolone (1gram/day) for 3 days along with supportive treatment.

Teaching point: Neuroretinitis can present with sudden severe loss of vision and requires consideration of systemic and infective causes and their mimics.

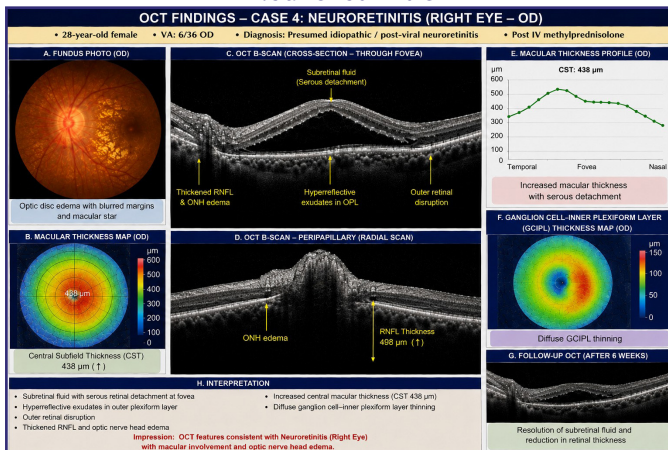
Case 3: Neuroretinitis in a Complex Ocular/ Systemic Setting



A 24-year-old female with history of panuveitis, bilateral disc edema and extrapulmonary tuberculosis presented with recurrent visual symptoms in the left eye (OS) after receiving periocular corticosteroids. The right eye (OD) had longstanding severe ocular involvement with retinal scarring and poor visual potential. On examination, the left eye had optic disc edema with associated macular exudative changes in the context of active/recurrent ocular inflammation. She had previously received anti-tubercular therapy and periocular corticosteroids.

Teaching point: Disc edema with macular exudation can be seen in complicated cases of infection and inflammation and careful evaluation of the systemic and ocular context is needed to reach a diagnosis taking into account the relevant infections and treatment.

Case 4: Presumed Idiopathic / Post-Viral Neuroretinitis

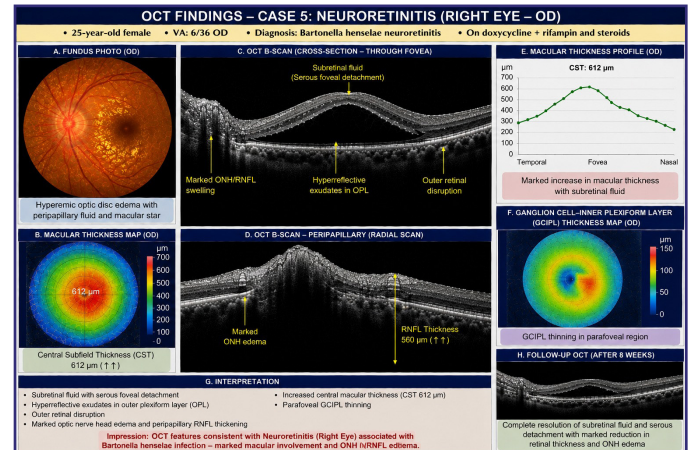


28-year-old female presented with painless progressive visual loss and central scotoma in the right eye for 5 days. History of low grade fever, malaise with self limited viral prodrome about 2 weeks back. Best corrected visual acuity was 6/36 OD with relative afferent pupillary defect and impaired colour vision. Fundus examination showed hyperemic optic disc edema with macular star. Magnetic resonance imaging (MRI) of brain and orbits was normal and the infective work up was negative.

The patient was managed with intravenous methyl prednisolone (1 g/day for 3 days). On follow up best corrected visual acuity improved to 6/9 with diminution of optic disc edema.

Teaching point: Neuroretinitis can present following viral prodrome and can mimic infectious causes with significant optic nerve involvement despite normal neuroimaging and negative infective work up.

Case 5: Confirmed Bartonella henselae Neuroretinitis



A 5-year-old female presented with sudden loss of vision in one eye and a central scotoma in the right eye for 5 days. The patient had a viral prodrome 3 weeks ago associated with fever, malaise, and right axillary lymphadenopathy. There was a history of exposure to stray kittens 1 month before presentation. Best-corrected visual acuity (BCVA) was 6/36 in the right eye with a relative afferent pupillary defect. Fundus examination showed hyperemic optic disc edema, peripapillary fluid, and an evolving macular star.

Optical coherence tomography (OCT) revealed subretinal fluid with serous foveal detachment, hyperreflective exudative foci at the level of the outer plexiform layer, and marked optic nerve head/retinal nerve fiber layer thickening. Serum Bartonella henselae antibody levels were positive (>1:256; IgM 1:64), and other serologic tests for infectious etiologies were negative.

The patient was treated with doxycycline (100 mg twice a day) and rifampin (300 mg twice a day) and received adjunctive corticosteroids after the start of antibiotic therapy. Visual acuity improved to 6/6 in the right eye (partial) at 8 weeks, with resolution of subretinal fluid and remarkable reduction of exudates.

Teaching point: Bartonella-associated neuroretinitis may show characteristic features on OCT, such as

optic nerve head edema, subretinal fluid, and serous foveal detachment. Awareness of the association between cat exposure and neuroretinitis allows timely and specific treatment.

SUMMARY

Certainly. For a **paper/manuscript**, I would keep the

table concise while retaining the clinically important differentiating features.

Yes. For a **journal manuscript**, I would reduce it to the variables that distinguish the five cases and avoid repeating detailed examination findings.

Table 1: Clinical Profile and Outcomes of Patients With Neuroretinitis

Case	Age/Sex	Presentation	Key Findings	Etiology	Treatment	Outcome
1	24/M	Blurring OS + headache	Disc edema + macular star; BCVA 6/9	Idiopathic/clinical	IVMP + azithromycin	BCVA 6/6
2	39/F	Severe visual loss OS + headache	Disc edema + macular star; FC 1 m	Sickle-cell disease	IVMP + supportive	—
3	24/F	Recurrent visual symptoms OS	Panuveitis + disc edema + macular exudates	Ocular TB	ATT + periocular steroid	Poor OD prognosis
4	28/F	Progressive visual loss OD + central scotoma	Disc edema + macular star; BCVA 6/36	Post-viral/idiopathic	IVMP	BCVA 6/9
5	5/F	Visual loss OD + central scotoma	Disc edema + macular star; SRF on OCT	<i>B. henselae</i>	Doxycycline + rifampin + steroid	BCVA 6/6(P); SRF resolved

Abbreviations: ATT – antitubercular therapy; BCVA – best-corrected visual acuity; FC – finger counting; IVMP – intravenous methylprednisolone; OD – right eye; OS – left eye; SRF – subretinal fluid; TB – tuberculosis.

DISCUSSION

Neuroretinitis is an inflammatory optic neuropathy associated with optic disc edema and subsequent macular star formation with a wide range of infectious, inflammatory, and idiopathic etiologies. Our series highlights the variable presentation, severity, and associations of this condition.

In our cases, the visual acuity ranged from relatively good to severely reduced, indicating that the degree of central vision loss may not directly correlate with the involvement of the optic nerve. The characteristic appearance of the macular star was present in most cases, although it might change over time. The OCT was particularly useful in the pediatric *Bartonella* case, as it showed optic nerve head edema, subretinal fluid, serous foveal detachment, and outer retinal exudative changes.

The etiology varied across our series, ranging from presumed idiopathic/post-viral disease, systemic

sickle-cell disease, ocular tuberculosis, and *Bartonella henselae* infection. The *Bartonella* case had a supportive exposure history, lymphadenopathy, positive serology, and characteristic ocular manifestations, illustrating the importance of a targeted systemic history and investigations. Likewise, the presence of systemic disease in other cases further emphasizes the need to consider neuroretinitis as an opportunity to identify associated infectious and systemic inflammatory conditions.

The treatment was individualized based on the suspected etiology and included corticosteroids, antimicrobial therapy, or both. Overall, the cases had a relatively good visual outcome; however, the severity and coexisting systemic disease affected the prognosis.

In summary, the cases highlight the heterogeneity of neuroretinitis. We have outlined some of the key diagnostic and therapeutic considerations for managing this condition.

CONCLUSION

Neuroretinitis is a broad clinical and etiologic category ranging from relatively mild loss of vision to total blindness and from isolated postviral syndrome

to systemic illnesses such as sickle-cell disease, tuberculosis, or *Bartonella henselae* infection. A comprehensive clinical and systemic evaluation, including ocular imaging such as optical coherence tomography, is required to delineate the underlying cause and guide management. Timely treatment may lead to visual recovery in most instances.

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