



CASE REPORT

Bilateral Neuro-retinitis with Sudden Loss of Vision as the Initial Presentation of Measles

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Abstract

Neuroretinitis (NR) depicts a clinical presentation of optic disc edema with subsequent development of macular edema. Pathogenesis is directly linked to inflammation of the optic nerve and early clinical diagnosis may be challenging. NR may be idiopathic, infectious, inflammatory or mimic other ophthalmic conditions and identification of etiology is crucial. We report a patient of bilateral neuroretinitis as the initial presentation of measles. Laboratory tests and ancillary ophthalmic investigations using scan ultrasonography and optical coherence tomography provided supportive evidence of measles retinopathy. This case underscores the likelihood of ophthalmic disease such as neuroretinitis as an early manifestation of measles. Multi-disciplinary involvement enables early diagnosis and holistic management.

Key Words

Neuroretinitis, Measles, Idiopathic Intracranial Hypertension

Introduction

Neuro-retinitis (NR), presents as an inflammation of the optic disc and surrounding retina. It clinically appears as disc edema and macular exudates arranged in a star shaped fashion. It can be a diagnostic challenge due to aextensivespectrum of etiological agents.^[1] The term indicates the appearance, however does not provide a clue to the precise etiology. A knowledge on the etiology, course, severity, resolution and recurrence guides our management. Measles or Rubeola is a contagious infection caused by an RNA virus belonging to the paramyxoviridae family. It is characterized by exanthematous fever and has a good prognosis when the course is uncomplicated.^[2] We herein report our findings in a 54-year-old lady whose initial presentation was of sudden painless bilateral loss of vision associated with

headache. Ophthalmic complaints were followed two weeks later by full blown measles. This is the first report of a patient with of bilateral neuro retinitis as the initial presentation of measles.

Case Report

A 54-year-old lady presented with defective vision in the right eye and headache for 2 days. She had a history of measles in childhood but had been healthy otherwise. There was no contact with pets or any other significant past history. On initial presentation, her best corrected visual acuity was 6/24, N36 in the right eye and 6/9, N8 in the left eye. A sluggish pupillary reaction was noted in her right eye. Anterior segment was normal. Fundus examination showed vitritis, hyperemic disc edema and an abnormal macula contour more in the right than left eye

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Manuscript Received: 08.12.2025; Revision Accepted: 10.02.2026

Published Online First: 10th April, 2026

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Cite this article as: Mahesh A, Avadhani D, Ranganathan LN, Bhattacharya S, Singh M, Annamalai R. Bilateral Neuro-retinitis with Sudden Loss of Vision as the Initial Presentation of Measles. JK Science 2026;28(2):124-6.

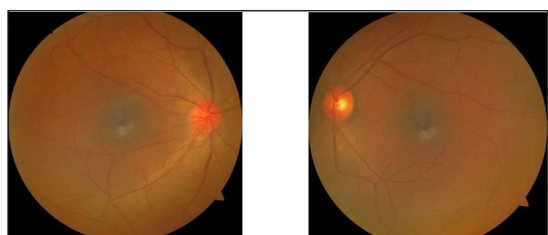


Figure 1 showing disc edema in both eyes.



Figure 2: B Scan shows widening of the optic nerve sheath with elevation of the optic disc.

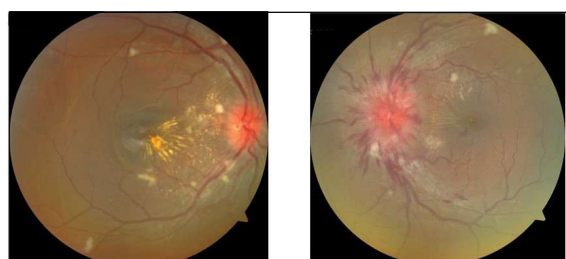


Figure 3: Neuroretinitis in both eyes.

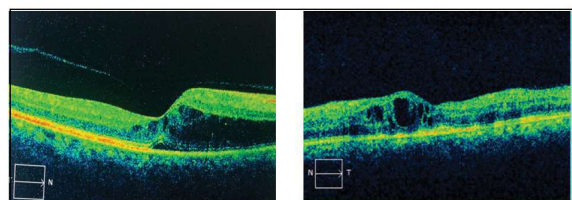


Figure 4: OCT showing macular edema.

(Figure 1). Extraocular movements and intraocular pressures were normal. VEP showed decreased amplitude and increased latency. B scan ultrasound on axial view showed widening of the optic nerve sheath with 1-2 mm elevation of the optic disc (Figure 2). Neurological examination, MRI of the brain and CSF analysis were normal. Complete blood counts revealed leucopenia of 3500 cells/mm³ (normal) with lymphopenia and an elevated ESR of 88 mm at the end of 1 hour by Westergren method. A differential diagnosis of idiopathic intracranial hypertension and posterior uveitis was considered at this point. Further work up showed a negative

serology for posterior uveitis. She reviewed after 1 week with sudden loss of vision in both eyes, severe headache, fever and rashes. Visual acuity was 6/60, N 36 in her right eye and limited to finger counting at 2 meters in her left eye. Examination of the fundus showed hyperemic disc with blurred margins, cotton wool spots, hemorrhages and hard exudates at the macula with macular edema of about 2- disc diameters (Figure 3). Color vision was impaired in both eyes at based on the test using Ishihara chart. Optical coherence tomography (OCT) showed macular edema (thickness of 350 microns), subretinal and intraretinal fluid with hypo-reflective spaces and complete posterior vitreous detachment (Figure 4). Laboratory tests revealed high titres of anti-measles antibodies with serum values greater than 270AU/ml. Supportive therapy was given. A diagnosis of bilateral neuro-retinitis likely due to measles was made. A repeat OCT at the end of 3 weeks from the date of diagnosis showed resolution of disc and macular edema with recovery of visual acuity to 6/6, N6 in her right eye and 6/9, N6 in her left eye.

Discussion

We report a case of neuroretinitis due to relapse of measles which diagnosed on the basis of a history, clinical and laboratory correlation. NR is a localised inflammation of the optic disc and the macula. The underlying etiology may be infectious, immune-mediated or an idiopathic disease. The most common cause is cat-scratch disease due to the bacterium *Bartonella henselae*.^[3] Other causes such as tuberculosis, Lyme disease, syphilis, toxoplasmosis, herpes virus, sarcoidosis, SLE, Behcet disease, and polyarteritis nodosa have been reported.^[4] Inflammatory cells due to measles can affect the optic nerve head. Macular edema and cotton wool spots occur as a consequence of ischemia. Macular exudates are essentially lipid (hard exudates) with extravasation of fluid and are a late feature. In the early stages, OCT will help to identify vitreous cells, intraretinal exudates, subretinal and intraretinal fluid. These observations may suggest NR due to an evolving systemic infection before the classical clinical presentation develops. OCT is an important tool especially when the patient presents with isolated disc oedema.^[5]

Our patient initially presented with disc oedema and developed NR in both eyes within one week. OCT of both eyes showed extensive destruction of the retinal architecture with ballooning of intact internal limiting membrane (ILM). Such ballooning of ILM is highly characteristic of measles retinopathy.

Multiplication of the measles virus causes extensive destruction within the layers of the retina. Measles retinopathy presenting with clinical features of haemorrhagic necrotising retinitis^[6] or a focal retinitis^[7] have been reported. NR in subacute sclerosing panencephalitis^[8] has been described and can be distinguished into subtypes based on aetiology and recurrence.

Onset of ocular inflammation prior to neurological and systemic manifestation is not typical. Macular exudates develop after a week of papillitis. In the initial stages when NR has not evolved fully, ocular signs may resemble idiopathic intracranial hypertension, hypertensive retinopathy (malignant hypertension) or posterior uveitis. Isolated disc oedema may be mistaken for papilledema, optic neuritis or anterior ischemic optic neuropathy. Imaging characteristics help exclude the above differentials. Splinter haemorrhages indicate severe inflammation. Various cases showing bilateral neuroretinitis following disseminated TB, zoster infection and anti-rabies vaccination have been reported. Measles retinopathy is more likely an immune mediated reaction to viral RNA rather than direct viral invasion and the severity depends on the immune response of the host.^[9]

Conclusion

Measles is a highly prevalent infection in our demographic. Although relapses of measles infections are rare, they can present with neuro-retinitis and ophthalmologists may be the first point of contact for such patients. In these cases, multidisciplinary involvement and ophthalmic screening guided by clinical suspicion is

imperative in preventing sequelae and in improving prognosis.

Financial Support and Sponsorship : Nil
Conflict of Interest: Nil

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