Journal of Medicine and Health (JMH) **e-ISSN: 2442-5257**
Vol. 8 No. 2 August 2026 **https://doi.org/10.28932/jmh.v8i2.12685**

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Optic Neuritis Mimicking as Idiopathic Intracranial Hypertension in a Child: A Case Report

Neuritis Optik dengan Gambaran Menyerupai Hipertensi Intrakranial Idiopatik pada Anak: Laporan Kasus

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Received: July 17, 2025
Accepted: July 2, 2026

Abstract

Sudden visual loss in a child can be caused by optic neuritis or idiopathic intracranial hypertension (IIH), the latter being strongly associated with obesity. This report aims to describe a pediatric case with overlapping clinical features of optic neuritis and IIH, highlighting a diagnostic and therapeutic challenge. An 11-year-old girl presented with sudden bilateral visual loss. Right eye vision was light perception, while left eye vision was hand movement. A neuro-ophthalmic examination confirmed optic disc edema and increased retinal nerve fiber layer thickness on an optical coherence tomography scan. Her body mass index was in the obese range. Treatment initially involved a high-dose intravenous steroid injection for optic neuritis. MRI scans were normal, but after three days of treatment, there was only minimal improvement. Acetazolamide was then added for two weeks due to suspicion of IIH. After oral steroid continuation for three months, there was significant visual improvement. Follow-up showed good visual acuity. This case underscores the importance of recognizing optic neuritis as the primary cause of acute visual loss in children, even in the presence of confounding features such as obesity.

Keywords*: child; optic neuritis; sudden visual loss; idiopathic intracranial hypertension; steroid.*

How to Cite:
Abdullah MR, Mustikasari RI. Optic neuritis mimicking as idiopathic intracranial hypertension in a child: a case report. Journal of Medicine and Health. 2026; 8(2): 184-90. DOI: https://doi.org/10.28932/jmh.v8i2.12685.

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Introduction

A child may experience acute visual loss due to a number of conditions, including optic neuritis (ON), intracranial mass, or idiopathic intracranial hypertension (IIH). Up to 25% of children who arrive with an independent ocular symptoms develop ON. With an annual incidence rate of 0.15–0.57 per 100,000 person-years in children and 5.1 per 100,000 person-years in adults, pediatric ON is uncommon.[1–4] Decreased visual acuity (VA), dyschromatopsia, and impairments in the visual field are the main characteristics of ON. It usually takes hours to days for visual loss to start, and it usually gets worse a few days afterwards.[5] According to a study by Shah et al., 72% of children under the age of ten had bilateral ON, whereas 70% of children over the age of ten had unilateral ON.[6] The Optic Neuritis Treatment Trial (ONTT) evidence-based recommendations reveal that high-dose steroid intravenous injection leads to a faster recovery than oral steroids in the first 15 days after onset, and is currently used to treat ON in the pediatric population. For three to five days, children are treated with IV methylprednisolone at a dose of 30 mg/kg per day, up to a maximum of 1 g per day.[5,6]

IIH is defined as elevated intracranial pressure of unknown cause. New onset headaches accompanied by visual abnormalities are among the clinical characteristics. It is associated with papilledema. Brain MRI is used in the diagnosis process to rule out venous sinus thrombosis or mass. If a lumbar puncture reveals pressure more than 250 mmH_2O with normal composition, it is a significant marker of IIH. Obesity, which is often associated with excess androgen, is a well-recognized risk factor for IIH. Acetazolamide, to lower the production of cerebrospinal fluid (CSF), is widely used to control IIH in adult and children.[7]

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An 11-year-old girl was admitted to the hospital after experiencing unexpected blurriness in both eyes for a week. Associated complaints included pain upon eye movement and mild headache, without systemic symptoms such as fever, vomiting, or recent infections. Birth and developmental history were unremarkable, and immunizations were complete. Her anthropometric profile revealed obesity.

Initial ophthalmic evaluation showed severely impaired visual acuity (OD: light perception, OS: hand movement) with bilateral optic disc swelling (Figure 1). Color vision and visual field testing were significantly affected, and OCT revealed increased RNFL thickness in both eyes (Figure 2). Despite these findings, head MRI showed no enhancement of the optic nerve or its sheath, and there were no focal lesions or midline structural deviations (Figure 3). Serologic workup indicated elevated ESR, reactive IgG against Toxoplasma, and Rubella, and CMV

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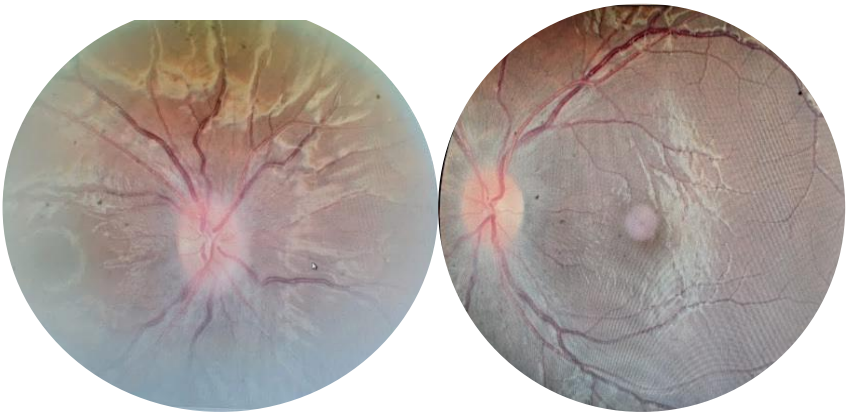
(TORCH), with profoundly high insulin levels. A TORCH-type workup is performed in young patients with optic neuritis to rule out infectious causes. The patient was diagnosed with pre-diabetic insulin resistance and obesity following a pediatric consultation. Initial diagnosis was atypical bilateral optic neuritis with papilledema as a differential consideration.



A **B**

Figure 1 Fundus Photo of Right Eye (A) and Left Eye (B) at Initial Examination Showed Optic Disc Swelling

She was admitted for intravenous methylprednisolone therapy (4×250 mg during three consecutive days), continued with a tapered-dose of oral corticosteroids. Supportive treatment included omeprazole, mecobalamin, and vitamin D3. Acetazolamide was added later, considering suspected IIH. The first three days after methylprednisolone IV, there was very minimal progression in visual acuity. The normal MRI and obesity status resembles with IIH. The family of the patient declined the lumbar puncture. Acetazolamide was given for two weeks. During the first week of combined steroid and acetazolamide treatment, there was notable visual acuity improvement. The acetazolamide was stopped after two weeks, and the oral steroid was continued up to three months.

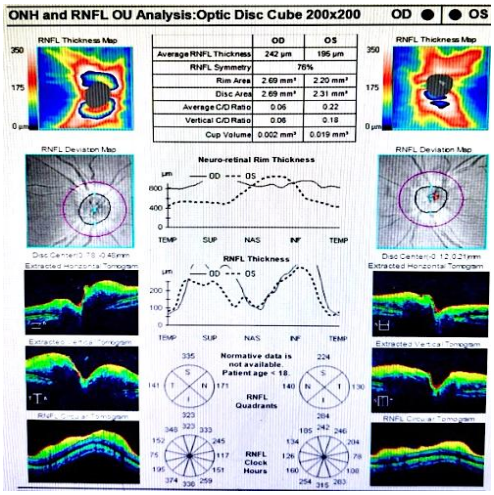


Figure 2 OCT showed increased retinal nerve fiber layer thickness at initial examination

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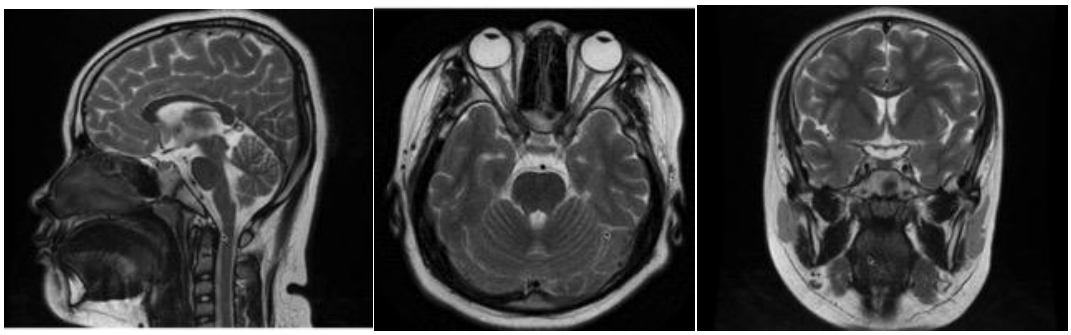

Figure 3 Brain and orbit MRI showed no optic nerve enhancement, mass, nor aneurysm

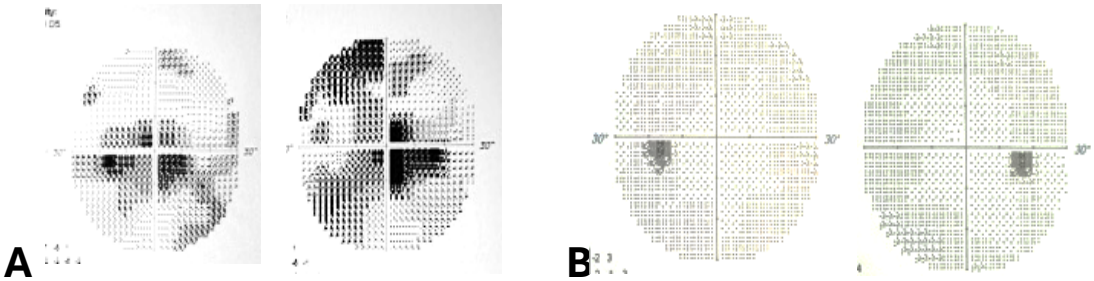

Figure 4 Visual Field (HFA, Zeiss) revealed improved condition of visual field shown by (A) at two weeks after treatment and (B) at three-month follow-up

Progress over three months showed steady visual recovery. Visual acuity improved to 5/5 OU, and OCT revealed normalization of RNFL metrics. Serial Humphrey visual field tests demonstrated resolving defects (Figure 4), although low reliability due to young age was noted in some sessions.

Discussion

This case illustrates the diagnostic challenges in pediatric neuro-ophthalmology. While optic neuritis is rare in children, its presentation can mimic other conditions such as IIH, especially in patients with comorbid obesity and endocrine abnormalities. Imaging and serology play crucial roles in excluding infectious and demyelinating etiologies. The absence of optic nerve enhancement on MRI initially argued against classical optic neuritis, yet clinical features and steroid responsiveness favored a diagnosis within its spectrum.

The therapeutic response emphasizes the importance of timely corticosteroid administration and the need for pediatric input regarding metabolic and endocrine aspects. This case also highlighted how atypical features such as obesity and absence of systemic signs might mask serious neuro-ophthalmologic disease.

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Up to 25% of children have an independent ocular illness or in association with a central nervous system inflammatory condition such as multiple sclerosis (MS). Reduced visual acuity (VA), dyschromatopsia, and deficits in the visual field are the main characteristics of ON. The beginning of visual loss normally takes hours to days, and it usually gets worse in a few days. Bilateral involvement may follow unilateral presentation at the beginning.[5]

ON was known to be associated with demyelinating disease. MS, neuromyelitis optica (NMO), myelin oligodendrocyte glycoprotein antibody-associated encephalomyelitis (MOG-EM), and acute disseminated encephalomyelitis (ADEM) are the four main categories of demyelinating illnesses. The most prevalent of these four types was MS, a chronic inflammatory illness that has a high correlation with acute vision loss from optic neuritis. The MRI typically shows an area of focal hyperintensity, which was not shown in the case presented. The McDonald Criteria, which depend on MRI, CSF measurement, and clinical characteristics, are used to diagnose MS.[8]

MOG-Ab has caused a fundamental shift in the field of pediatric ON. Aquaporin-4 antibody-positive neuromyelitis optica spectrum disorder (AQP4+NMOSD) and MS are clinically and radiologically different from a wide range of demyelinating illnesses linked to this serological marker. With the exception of the subset that displays a recurring phenotype, MOG-Ab seropositivity prevalence is higher in the pediatric population and indicates a better prognosis than MS or AQP4+NMOSD.[9]

Many signs and symptoms of ON in children are uncommon in adults, resembling the "atypical features" that prompt thorough investigation when seen in adult patients. Differential diagnoses include IIH, sarcoidosis, infectious neuroretinitis, papilledema, leukaemia, systemic lupus erythematosus, and ON caused by a virus or immunisation should be taken into account. ON may present as a recurrent disease with no additional symptoms or as an isolated, single-phase disorder.[9] Following the recommendations of the Optic Neuritis Treatment Trial, the patient's vision gradually improved.

Fontanelle bulging in healthy infants with normal neuroimaging should always be regarded as a late indicator of IIH, whereas restlessness and feeding difficulties, particularly with vomiting, may be an early warning sign. Infantile or pediatric IIH is incredibly uncommon.[10–12] Lumbar punctures should always be performed in these situations, ideally with cerebrospinal fluid opening pressure measurement, which is one of the standardised diagnostic criteria. Nevertheless, since there are numerous variables that might alter the outcome, lumbar punctures may be problematic in newborns. Affected newborns can avoid visual loss with proper diagnosis, prompt treatment, and rigorous follow-up.[13-14]

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A case study reported infantile IIH with visual recovery using steroids and acetazolamide.[15] Other case reports present IIH with limited ocular motility examination.[16] It is commonly related to obesity.[17] Age-related changes in usual clinical presentations and atypical symptoms in newborns may result in a delayed diagnosis. Diagnosis typically relies on papilledema, a normal brain MRI, and elevated opening pressure on lumbar puncture. Acetazolamide, the base of treatment, can alleviate symptoms and prevent irreversible vision loss if given on time.[18-20]

Our case highlights the importance of close observation during recovery, given the slow visual improvement seen with steroid treatment as the mainstay therapy. This might be due to metabolic disorders in the children, which might result in a slower consumption of the steroid effect for the optic nerve inflammation. IIH is diagnosed by the Friedman criteria, with lumbar puncture being one of the key diagnostic criteria.[11] We could not diagnose this patient as IIH due to no data on lumbar puncture, and the trial of acetazolamide was not effective either, since the visual progress was mainly a result of steroid treatment. ON is the clinical diagnosis demonstrated in this case. With corticosteroid therapy, most patients will see immediate improvement, but they are more susceptible to relapses during tapering or withdrawal. As oral prednisolone is tapered below 10 mg daily or within two months of stopping steroids, over 70% of patients relapse. The family and child were also advised to participate in a weight loss program, as it could potentially reduce the risk of developing metabolic diseases.[13-15]

Conclusion

Acute vision loss in children warrants a comprehensive neuro-ophthalmologic assessment. Despite atypical imaging findings, the clinical course supported a diagnosis of steroid-responsive optic neuritis. Multidisciplinary care, including pediatric endocrinology, contributed to favorable outcomes. It is also crucial to closely monitor and observe a child after initiating initial treatment, as optic neuritis tends to progress more slowly in children, particularly when associated with metabolic disorders.

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