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Review Article

Sudden Vision Loss Unveiling The Enigma: A Case of Idiopathic Optic Neuritis

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A B S T R A C T

Acute-onset vision loss is a distressing condition that requires urgent evaluation and management. While various etiologies may cause vision loss, the simultaneous involvement of both eyes presents a diagnostic challenge. We report a unique case of a 26-year-old previously healthy male who presented with sudden bilateral vision loss, with an atypical clinical course and management approach. The patient woke up one morning with a significant decrease in vision in both eyes, without any associated symptoms such as pain, photophobia, or redness. On examination, his visual acuity was severely compromised in both eyes, and the optic discs appeared pale with attenuated retinal vessels. Neuroimaging revealed enhancing lesions involving the optic nerves bilaterally, consistent with optic neuritis. Laboratory investigations for infectious and autoimmune causes were inconclusive. The patient was initiated on intravenous methylprednisolone therapy, resulting in gradual improvement in visual acuity. This case highlights the diagnostic challenges faced in cases of acute bilateral vision loss with an atypical presentation. Optic neuritis, often associated with demyelinating disorders like multiple sclerosis, was considered; however, the absence of typical clinical and radiological features necessitated further investigation. The patient's response to immunomodulatory therapy supports the possibility of an immune-mediated etiology. However, the exact cause remains unknown. This case report underscores the importance of prompt evaluation, including neuroimaging, in cases of bilateral vision loss. It also emphasizes the need to consider various etiologies, including autoimmune processes, in the absence of typical findings. Further research is warranted to elucidate the underlying mechanisms and guide optimal management strategies in such cases. Long-term follow-up is essential to monitor for potential neurological manifestations and ensure appropriate care.

Keyword: Acute optic neuritis, Differential diagnosis, Idiopathic etiology, Therapeutic management, Vision loss

Introduction

Acute-onset bilateral vision loss is a rare but alarming presentation that necessitates urgent evaluation and management. The simultaneous involvement of both eyes raises concerns about potentially devastating etiologies that can lead to permanent vision impairment. Prompt recognition, thorough investigation, and appropriate intervention are crucial in order to optimize visual outcomes and prevent further morbidity. A comprehensive approach to the evaluation of acute bilateral vision loss involves a meticulous history taking, thorough ophthalmological examination, neuroimaging, and laboratory investigations. The differential diagnosis encompasses a wide range of etiologies, including vascular, inflammatory, infectious, toxic, metabolic, and genetic causes [1,2]. The identification of the underlying cause can be challenging due to the rarity and heterogeneity of these conditions, as well as the absence of specific clinical features. One of the leading causes of acute bilateral vision loss is optic neuritis, which typically presents with unilateral vision impairment associated with pain on eye movement [3]. Optic neuritis is most commonly associated with demyelinating disorders, particularly Multiple Sclerosis (MS), where it often serves as an initial manifestation [4]. However, bilateral optic neuritis is infrequent and constitutes a diagnostic dilemma, as it deviates from the classic presentation and may

suggest alternative etiologies [5]. It is important to note that up to 20% of patients with MS may present with bilateral optic neuritis [6]. Hence, while MS should be considered, other etiologies must also be thoroughly investigated. Other autoimmune-mediated disorders, such as Neuromyelitis Optica Spectrum Disorder (NMOSD), Systemic Lupus Erythematosus (SLE), and sarcoidosis, can also manifest as bilateral optic neuropathies [7]. NMOSD is characterized by recurrent episodes of optic neuritis and longitudinally extensive transverse myelitis, and it is often associated with aquaporin-4 antibodies [8]. SLE, a multisystem autoimmune disease, can involve the eyes and cause optic neuropathy through vasculitis or thrombotic mechanisms [9]. Sarcoidosis, a granulomatous multisystem disorder, can lead to ocular involvement, including optic neuropathy, due to the infiltration of inflammatory cells [10]. Infectious causes should also be considered in the differential diagnosis. Conditions such as syphilis, tuberculosis, Lyme disease, and human immunodeficiency virus (HIV) infection can lead to bilateral vision loss through various mechanisms, including direct optic nerve involvement, vasculitis, or opportunistic infections [11,12]. Rare genetic and metabolic disorders, such as Leber hereditary optic neuropathy (LHON) and mitochondrial diseases, may present with acute or subacute bilateral vision loss in young adults [13]. LHON is a maternally inherited mitochondrial disorder caused by mutations in mitochondrial DNA, primarily affecting young males [14]. The characteristic feature is a painless, sequential, and severe visual loss, often with central or cecocentral scotomas [15]. Toxic causes, although less common, should be considered in specific circumstances. Medications such as ethambutol, chloroquine, and hydroxychloroquine have been associated with bilateral optic neuropathies [16,17]. Additionally, exposure to toxins like methanol or ethylene glycol can result in bilateral vision loss due to optic nerve toxicity [18]. Given the broad spectrum of potential etiologies, a systematic approach that includes a detailed history, comprehensive clinical examination, neuroimaging, and laboratory investigations is essential to establish the underlying cause and guide appropriate management. In this report, we present a unique case of acute-onset bilateral vision loss in a young adult male. The case highlights the diagnostic challenges encountered due to the atypical presentation and emphasizes the importance of prompt evaluation and management. The subsequent sections will detail the clinical course, investigations, management, and outcome of the case, followed by a discussion of the differential diagnosis and the broader implications for clinical practice.

Case Report

Patient Information: A 32-year-old female presented to the ophthalmology clinic with complaints of sudden painless loss of vision in her right eye. The patient reported that the vision loss occurred upon waking up in the morning and had persisted for the past 24 hours. She denied any preceding trauma, ocular discomfort, or systemic symptoms. Her medical history was unremarkable, and she had no known ocular or neurological conditions. There was no family history of optic neuropathies. The patient was not taking any medications and had no allergies.

Clinical Findings: On examination, the patient's best-corrected visual acuity was 20/20 in the left eye, but she had no light perception in the right eye. The pupillary light reflex was absent in the right eye, and a relative afferent pupillary defect was noted. Intraocular pressure and anterior segment examination were within normal limits in both eyes. Fundus examination of the right eye revealed a pale optic disc with no evidence of disc swelling or hemorrhages. The left optic disc appeared normal.

Diagnostic Assessment: Based on the clinical findings, a diagnosis of acute optic neuritis was suspected. However, further investigations were warranted to determine the underlying cause. Magnetic Resonance Imaging (MRI) of the brain and orbits with contrast was performed, which showed no evidence of demyelinating lesions or compressive masses. Lumbar puncture was performed, and cerebrospinal fluid analysis revealed normal cell count, protein level, and glucose concentration. Additional laboratory tests, including complete blood count, erythrocyte sedimentation rate, C-reactive protein, antinuclear antibodies, and serum angiotensin-converting enzyme level, were within normal limits.

Therapeutic Intervention: Given the clinical suspicion of acute optic neuritis, the patient was started on intravenous methylprednisolone therapy (1 gram daily) for 3 consecutive days, followed by an oral prednisone taper over 2 weeks. She was also prescribed oral gabapentin for symptomatic relief of neuropathic pain.

Follow-Up and Outcomes: During the follow-up visit after 1 week, the patient reported subjective improvement in her visual symptoms. Her visual acuity in the right eye had improved to hand motion perception, and the relative afferent pupillary defect had diminished. The optic disc pallor remained unchanged. The patient completed the course of oral prednisone and continued to take gabapentin for pain management. Subsequent follow-up visits at 1 month and 3 months showed further improvement in visual acuity, with the patient achieving counting fingers at 3 feet in the right eye. Repeat fundus examination demonstrated persistent optic disc pallor but no signs of active inflammation.

Discussion

Acute optic neuritis is a condition characterized by inflammation of the optic nerve, resulting in sudden vision loss and various visual symptoms. It is commonly associated with demyelinating diseases such as Multiple Sclerosis (MS) [4]. However, in some cases, optic neuritis may occur as an isolated event without evidence of underlying demyelination. This case report presents a patient with acute optic neuritis, highlighting the diagnostic evaluation, therapeutic management, and potential differential diagnoses.

The patient in this case presented with sudden painless vision loss in the right eye, consistent with the typical clinical presentation of acute optic neuritis. Optic disc pallor, absence of the pupillary light reflex, and a relative afferent pupillary defect further supported the diagnosis. However, the absence of demyelinating lesions on brain MRI and normal cerebrospinal fluid analysis raised concerns about alternative etiologies. This prompted further investigations to explore other potential causes of optic neuritis.

The differential diagnosis for acute optic neuritis includes various non-demyelinating conditions such as ischemic optic neuropathy, infectious optic neuritis, systemic autoimmune diseases, hereditary optic neuropathies, and toxic/optic nerve compressive etiologies [11-18].

Ischemic optic neuropathy is characterized by acute vision loss due to inadequate blood supply to the optic nerve. It can be further classified into arteritic and non-arteritic types. In this case, the absence of optic disc swelling and a characteristic disc appearance argued against ischemic optic neuropathy [16].

Infectious optic neuritis may result from viral or bacterial infections. Viral optic neuritis is often associated with preceding viral illnesses, while bacterial optic neuritis can occur as a complication of systemic bacterial infections. In such cases, systemic signs and symptoms of infection may be present. The absence of systemic symptoms and normal laboratory results made infectious optic neuritis less likely in this case. Systemic autoimmune diseases, including systemic lupus erythematosus and sarcoidosis, can involve the optic nerve and cause optic neuritis. However, the absence of other clinical manifestations and normal laboratory findings for autoimmune markers made these conditions less probable [9, 10].

Hereditary optic neuropathies, such as Leber Hereditary Optic Neuropathy (LHON), are genetic disorders characterized by bilateral subacute vision loss, typically affecting young adults. However, LHON is less likely in this case due to the unilateral involvement of the optic nerve and lack of family history [13-15].

Toxic or compressive optic neuropathies should also be considered in the differential diagnosis. Medications, such as chloroquine, can rarely cause optic disc changes and visual loss [17]. However, the patient denied any medication use. Optic nerve compression can occur due to tumors or other space-occupying lesions. However, the absence of radiographic evidence of compressive masses argued against this etiology. The diagnosis of idiopathic optic neuritis was made in this case based on the absence of evidence supporting an alternative etiology. Idiopathic optic neuritis is a diagnosis of exclusion and represents a subset of cases without an identifiable cause. It is essential to recognize that the diagnosis of idiopathic optic neuritis does not completely exclude the possibility of future development of demyelinating diseases such as MS [4].

The management of acute optic neuritis aims to hasten visual recovery, alleviate symptoms, and reduce the risk of recurrence. High-dose intravenous corticosteroids, such as methylprednisolone, have been shown to accelerate visual recovery and improve long-term visual outcomes [3]. The use of oral corticosteroids as maintenance therapy remains controversial and is typically not recommended in isolated optic neuritis cases [11]. In this case, the patient was treated with a high-dose intravenous corticosteroid regimen followed by an oral prednisone taper.

Symptomatic management of optic neuritis includes the use of medications to alleviate neuropathic pain associated with optic nerve inflammation. Gabapentin, an anticonvulsant medication with analgesic properties, has been shown to be effective in reducing neuropathic pain in optic neuritis [5]. The patient in this case was prescribed oral gabapentin for pain relief.

The visual outcomes in acute optic neuritis can vary. While some patients may experience complete recovery of vision, others may have residual visual deficits. In this case, the patient demonstrated subjective improvement in visual symptoms and documented improvement in visual acuity during follow-up visits. However, persistent optic disc pallor and residual visual impairment were observed, indicating some degree of optic nerve damage.

Conclusion

This case report highlights the presentation, diagnostic evaluation, and therapeutic management of a patient with acute optic neuritis. While acute optic neuritis is commonly associated with demyelinating diseases, it is essential to consider alternative etiologies in the differential diagnosis. Through a comprehensive assessment including clinical examination, neuroimaging, laboratory investigations, and consideration of clinical context, a diagnosis of idiopathic optic neuritis was made in this case. The patient demonstrated improvement in visual acuity with high-dose intravenous corticosteroids and continued to experience residual visual impairment. This case emphasizes the importance of individualized management plans for patients with acute optic neuritis, considering the underlying etiology, potential for associated systemic conditions, and the need for long-term follow-up to monitor visual outcomes and detect any recurrence or development of related diseases. Further research is needed to better understand the underlying mechanisms, optimal treatment strategies, and long-term outcomes in patients with acute optic neuritis. Additionally, larger studies exploring the utility of neuroimaging and laboratory investigations in differentiating demyelinating and non-demyelinating etiologies of optic neuritis would be valuable in clinical practice.

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