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SUDDEN BILATERAL VISION LOSS IN A YOUNG PATIENT WITH SYSTEMIC AUTOIMMUNE DISEASE AND A NORMAL BRAIN MRI, WHICH RESPONDED TO STEROID TREATMENT

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ABSTRACT

Background: Sudden bilateral visual loss is a rare but alarming manifestation, particularly in young patients with systemic autoimmune disorders.

Case report: We describe the case of a 26-year-old woman with systemic lupus erythematosus, juvenile idiopathic arthritis, and Sjögren's syndrome, who presented with sudden bilateral vision loss following headache, vomiting, and diarrhoea. Ophthalmologic evaluation excluded hydroxychloroquine toxicity. Brain magnetic resonance imaging revealed no acute ischemic or demyelinating lesions. High-dose intravenous methylprednisolone was initiated, leading to near-complete recovery of vision.

Conclusion: Autoimmune optic neuropathy should be considered in young patients with sudden bilateral vision loss, even in the absence of neuroimaging abnormalities. Prompt recognition and empirical corticosteroid therapy may prevent irreversible disability.

KEYWORDS

Bilateral vision loss, systemic lupus erythematosus, autoimmune optic neuropathy, corticosteroids, neuro-ophthalmology

LEARNING POINTS

- Sudden bilateral vision loss in young patients with autoimmune diseases should raise suspicion for immune-mediated optic neuropathy.
- Normal magnetic resonance imaging and fundoscopy findings do not exclude serious inflammatory pathology.
- Early empirical corticosteroid therapy can be vision-saving.

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INTRODUCTION

Sudden bilateral vision loss is a rare but clinically significant presentation. In patients with systemic autoimmune diseases such as systemic lupus erythematosus (SLE) and Sjögren's syndrome, the differential diagnosis includes optic neuritis, vasculitis, posterior reversible encephalopathy syndrome, and drug-induced toxicity[1,2]. Prompt recognition is critical, as delayed treatment may result in irreversible visual impairment. This report presents a case of acute bilateral vision loss in a young woman with multiple autoimmune conditions, highlighting the challenges of diagnosis and the importance of early intervention.

CASE DESCRIPTION

A 26-year-old woman with SLE, juvenile idiopathic arthritis, and Sjögren's syndrome presented to the emergency department with 5 episodes of watery diarrhoea and more than 15 episodes of bilious vomiting. Within hours, she developed a severe occipital headache (10/10 intensity) and awoke with complete bilateral vision loss.

Her medications included hydroxychloroquine (200 mg every 12 hours), prednisolone (10 mg daily), and mycophenolate mofetil (500 mg every 8 hours). She denied recent trauma, infections, or medication changes.

On physical examination, her vital signs were stable. Neurological examination revealed intact pupillary light reflexes, no motor or sensory deficits, and no signs of intracranial hypertension. Fundoscopy demonstrated preserved optic disc margins without papilledema or retinal haemorrhage *(Fig. 1)*. Visual acuity was reduced to no light perception in both eyes.

Ophthalmological evaluation excluded hydroxychloroquine-induced retinopathy. Magnetic resonance imaging (MRI) of the brain with gadolinium contrast showed no optic nerve enhancement, demyelination, or infarction, but revealed multiple hyperintense areas in the white matter, suggestive of lupus activity, without involvement of the optic pathways *(Fig. 2 and 3)*. Laboratory studies ruled out infections, electrolyte imbalance, or systemic inflammation.

Given the clinical suspicion of autoimmune optic neuropathy, empirical intravenous methylprednisolone (1 g/day for 3 days) was administered, while hydroxychloroquine and mycophenolate were temporarily suspended. Supportive care with hydration and antiemetics was provided.

The patient showed partial recovery of vision by day 3 and near-complete recovery within one week. At one-month follow-up, visual acuity was normal, and no recurrence was observed. Maintenance immunosuppression was resumed under rheumatology and neurology supervision.

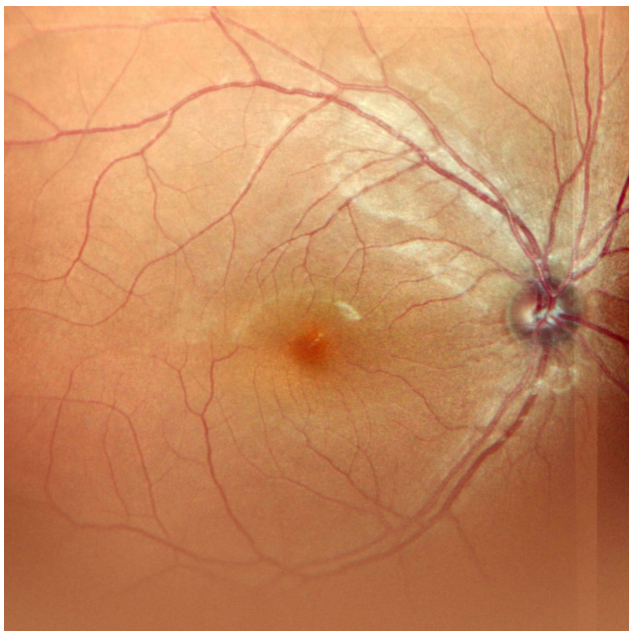


Figure 1. Fundoscopy showing preserved optic disc margins and absence of papilloedema or retinal hemorrhage.

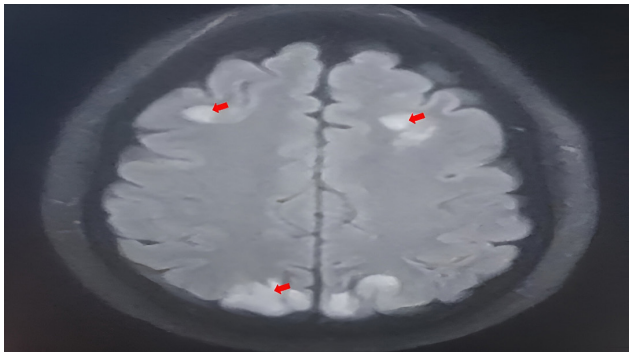


Figure 2. Brain magnetic resonance imaging with gadolinium, axial T1 projection: multiple irregular hyperintense lesions in occipital and frontal lobes, right caudate nucleus, internal capsule, and left thalamus (red arrows).

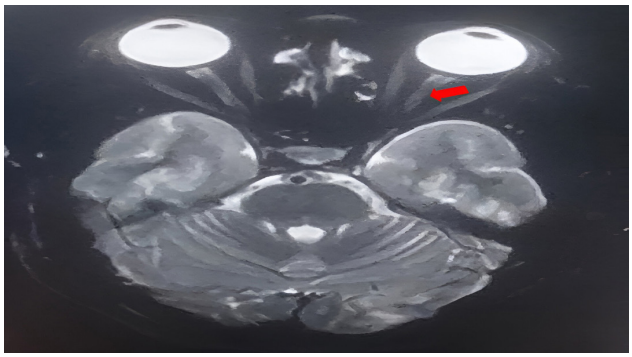


Figure 3. Brain magnetic resonance imaging, axial T2-weighted with fat suppression: optic nerves of normal caliber and signal intensity, without enhancement or demyelinating lesions, (red arrow).

DISCUSSION

Acute bilateral vision loss in young adults is an uncommon event that requires immediate and thorough evaluation. In patients with autoimmune diseases, this presentation may correspond to a neuro-ophthalmic manifestation such as optic neuritis or posterior reversible encephalopathy syndrome (PRES)[3]. Previous studies have demonstrated that these entities may respond favourably to treatment with high-dose corticosteroids[3].

In the case described the absence of pathological findings on MRI and fundoscopic examination initially complicated the diagnosis. Nevertheless, the clinical course was highly

suggestive of an autoimmune optic neuropathy. Similar reports in patients with lupus have documented reversible optic neuropathies with good steroid response, emphasizing the importance of initiating early empirical treatment when clinical suspicion is high. Delayed therapy may result in permanent visual disability. Therefore, a multidisciplinary approach involving neurology, ophthalmology, and rheumatology is essential to ensure timely diagnosis, appropriate management, and prevention of relapses.

Autoimmune optic neuritis may present with a normal MRI scan in a proportion of cases, although this is not the most frequent finding. The presence or absence of abnormalities depends on the underlying aetiology and the intensity of the inflammatory process. In isolated optic neuritis, without involvement of other regions of the central nervous system, MRI may be normal in approximately 20–30% of patients, particularly when inflammation is mild or does not produce detectable structural lesions[4].

There are specific entities in which MRI findings may consistently remain unremarkable:

- *Chronic recurrent inflammatory optic neuropathy (CRION):* characterized by recurrent inflammation of the optic nerve, without visible changes on conventional MRI, particularly during remission phases.
- *Non-arteritic ischemic optic neuropathy (NAION):* associated with comorbidities such as hypertension or diabetes but usually lacks MRI abnormalities since it does not involve acute inflammatory or demyelinating processes.
- *Optic neuropathies secondary to non-demyelinating autoimmune or infectious diseases (e.g., sarcoidosis, toxoplasmosis):* may cause chronic inflammation without detectable structural changes, in contrast to multiple sclerosis.
- *Metabolic or toxic optic neuropathies:* such as those related to vitamin B12 deficiency or drug-induced toxicity, which can lead to visual dysfunction without characteristic MRI alterations[5].

Adjacent infections (e.g., sinusitis, mucoceles) and space-occupying processes such as primary optic nerve tumours, meningiomas, pituitary adenomas, aneurysms, bone lesions, or metastatic disease are significant causes of optic neuropathy due to compression or infiltration[6].

The effects of systemic inflammation on the central nervous system are well characterized in neuropsychiatric SLE (NPSLE), one of the most severe forms of the disease[7]. Approximately 40% of patients with SLE present with neuropsychiatric manifestations, according to the American College of Rheumatology criteria[8].

Ocular involvement in lupus affects structures such as the retina, choroid, and optic nerve. It is estimated that about one-third of patients present with some degree of ocular involvement, with lupus retinopathy being the most common manifestation (≈29% during active disease phases). This condition ranges from mild changes, such as exudates and haemorrhages, to severe forms such as vaso-occlusive retinopathy, which is associated with a poor prognosis for both visual function and systemic disease course[9]. In contrast, lupus-related optic neuropathy is less common, with a prevalence of approximately 1%. Its pathophysiology is associated with inflammatory or ischemic mechanisms.

Diagnosis requires a combination of clinical examination and ancillary studies, including ophthalmoscopy, fluorescein angiography, optical coherence tomography, and, in selected cases, MRI. Treatment necessitates close collaboration between neurology, rheumatology, and ophthalmology, with systemic corticosteroids and immunosuppressive agents representing the cornerstone therapies, both of which have demonstrated efficacy in preserving visual function[10].

In the early management of optic neuritis, classification of the clinical subtype is essential, as therapeutic approaches may differ. In autoimmune or inflammatory forms, acute treatment with high-dose intravenous methylprednisolone followed by oral prednisone has been shown to accelerate visual recovery, although without significantly improving long-term functional outcomes. Equivalent doses of 1 g intravenous methylprednisolone are associated with a faster recovery[11].

Early corticosteroid use in optic neuritis is associated with common adverse effects such as sleep disturbances, mild mood changes, gastrointestinal symptoms, facial flushing, and weight gain. Less frequently, acne, headache, transient diarrhoea, and, in rare cases, severe complications such as acute pancreatitis have been reported[12].

CONCLUSION

Sudden bilateral vision loss in young patients with autoimmune diseases should prompt suspicion for autoimmune optic neuropathy. Even in the absence of structural abnormalities on imaging, empirical corticosteroid therapy can result in significant recovery and prevent long-term disability.

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