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Case Report

Ophthalmology Section

Bilateral Vision Loss due to Fulminant Hypertensive Optic Neuropathy Mimicking Cortical Blindness in a Patient with Co-existing Vestibular Schwannoma: A Case Report

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ABSTRACT

Malignant hypertension is a hypertensive emergency characterised by acute target-organ damage involving the cardiovascular, renal, neurological and ocular systems. Although hypertensive retinopathy is a well-recognised manifestation of malignant hypertension, profound bilateral visual loss secondary to hypertensive optic neuropathy is exceedingly rare and may mimic cortical blindness, posing a significant diagnostic challenge. Here, the authors report a case of a 66-year-old male with poorly controlled systemic hypertension who presented with sudden painless bilateral visual loss progressing to no perception of light in both eyes. Fundus examination revealed bilateral grade 4 hypertensive retinopathy with marked optic disc oedema. Pupillary reactions were sluggish but preserved without a relative afferent pupillary defect. Optical coherence tomography demonstrated relatively preserved retinal architecture, whereas Magnetic Resonance Imaging (MRI) revealed a vestibular schwannoma without occipital lobe involvement or diffusion restriction, effectively excluding cortical causes of blindness. The patient was managed as a hypertensive emergency with gradual blood pressure reduction. At one-month follow-up, visual acuity improved to finger counting at one foot in both eyes with mild reduction in optic disc oedema. This case highlights the importance of clinicoradiological correlation in patients presenting with profound bilateral visual loss and emphasises the diagnostic limitations of relative afferent pupillary defect in bilateral symmetric optic nerve disease.

Keywords: Hypertensive emergency, Malignant hypertension, Neuroimaging, Optic disc oedema, Visual pathway disorders

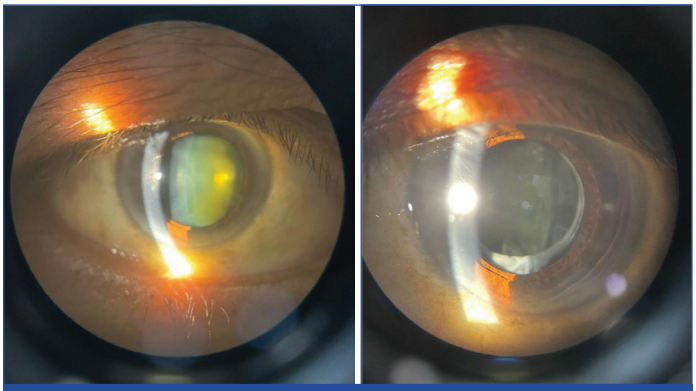
CASE REPORT

A 66-year-old male presented with sudden onset painless diminution of vision in both eyes for eight days, which rapidly progressed to complete loss of light perception. The visual loss was associated with severe persistent headache and multiple episodes of vomiting. The patient was a known hypertensive for the past ten years and admitted to irregular adherence to antihypertensive medications. There was no history of diabetes mellitus, cerebrovascular accident, seizures, trauma or anticoagulant usage. He reported tinnitus and episodic vertigo over the preceding several months, suggestive of vestibular involvement.

On admission, the patient was conscious, oriented and afebrile. Blood pressure was markedly elevated at 210/140 mmHg. Neurological examination revealed a positive Romberg test without focal neurological deficits. Best corrected visual acuity at presentation was no perception of light in both eyes. Pupillary reactions were sluggish but present bilaterally without a relative afferent pupillary defect. Extraocular movements were full and painless with no evidence of cranial nerve palsy.

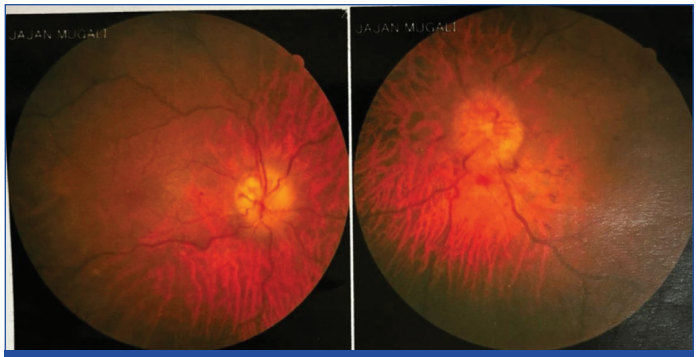
Anterior segment examination of the right eye revealed grade 2 nuclear sclerosis with posterior subcapsular cataract, whereas the left eye showed a well-centred posterior chamber intraocular lens. There was no anterior chamber inflammation and intraocular pressures were within normal limits in both eyes [Table/Fig-1,2].

Dilated fundus examination revealed bilateral marked optic disc oedema characterised by hyperaemia, elevation and blurred disc margins. Generalised arteriolar attenuation, arteriovenous crossing changes, flame-shaped retinal haemorrhages and hard exudates forming an early macular star configuration were noted in both eyes, consistent with grade 4 hypertensive retinopathy according to the Keith-Wagener-Barker classification [Table/Fig-3] [1].

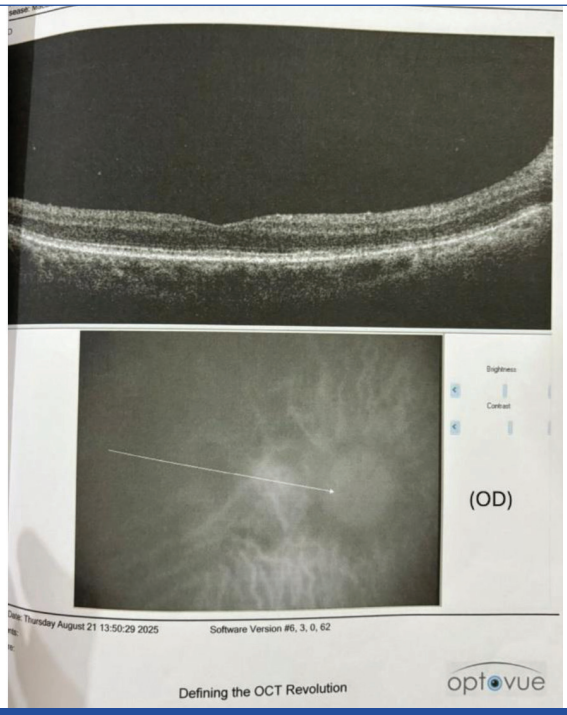


[Table/Fig-1]: Slit lamp photograph of the right eye demonstrating grade 2 nuclear sclerosis with posterior subcapsular cataract, with clear cornea and quiet anterior chamber. **[Table/Fig-2]:** Slit lamp photograph of the left eye showing a well-centred posterior chamber intraocular lens (pseudophakia) with quiet anterior segment. (Images from left to right)

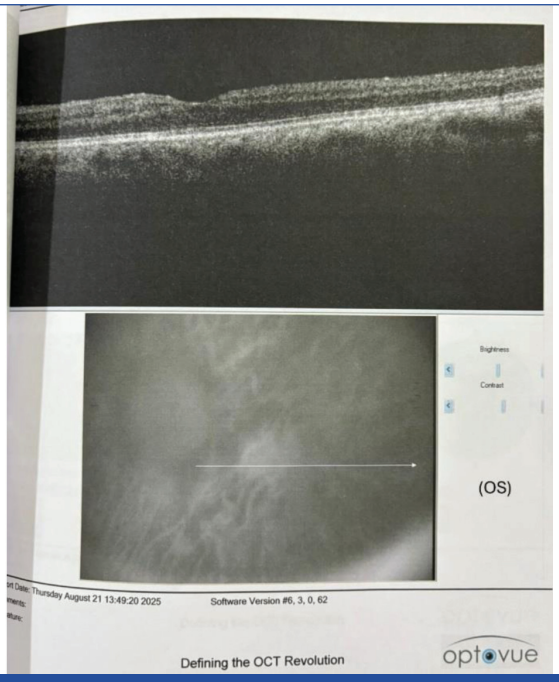
Macular Optical Coherence Tomography (OCT) demonstrated relatively preserved retinal architecture without significant intraretinal or subretinal fluid accumulation. Optical coherence tomography of the optic nerve head and Retinal Nerve Fibre Layer (RNFL) demonstrated increased RNFL thickness corresponding to optic disc oedema without evidence of established optic atrophy [Table/Fig-4-7].



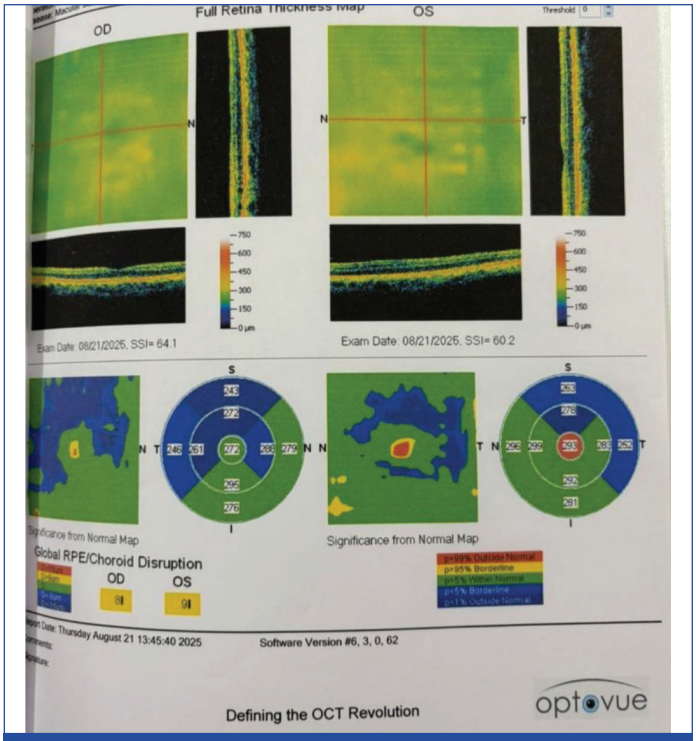
[Table/Fig-3]: Colour fundus photograph of the right eye demonstrating marked optic disc oedema with blurred margins and hyperaemia, associated with arteriolar attenuation, retinal haemorrhages, and hard exudates suggestive of grade 4 hypertensive retinopathy (Keith-Wagener-Barker classification).



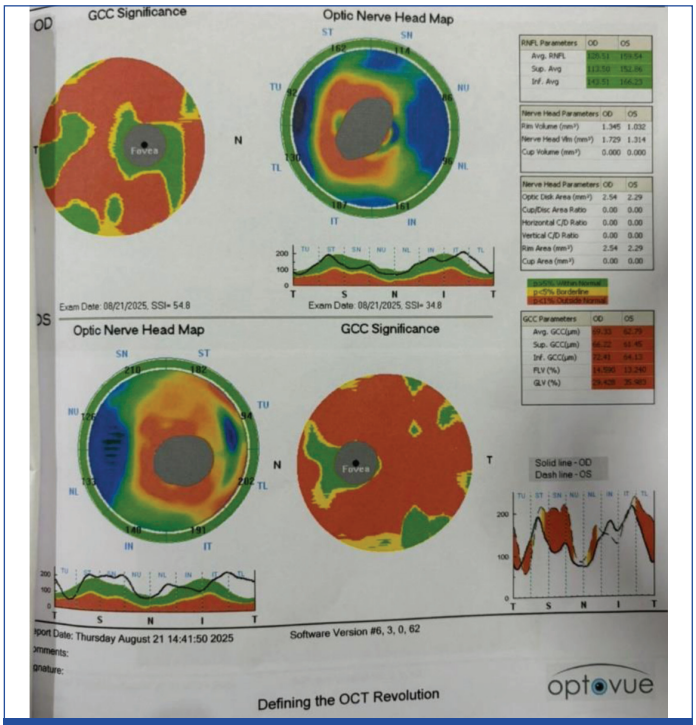
[Table/Fig-4]: Optical Coherence Tomography (OCT) macula of right eye (OD) demonstrating relatively preserved retinal architecture and foveal contour without significant intraretinal or subretinal fluid, not commensurate with the severity of visual loss.



[Table/Fig-5]: Optical Coherence Tomography (OCT) macula of left eye (OS) demonstrating relatively preserved retinal architecture and foveal contour without significant intraretinal or subretinal fluid.



[Table/Fig-6]: Optical Coherence Tomography (OCT) macular thickness and RPE/choroid map of both eyes demonstrating localised areas of increased retinal thickness with preserved overall macular contour.

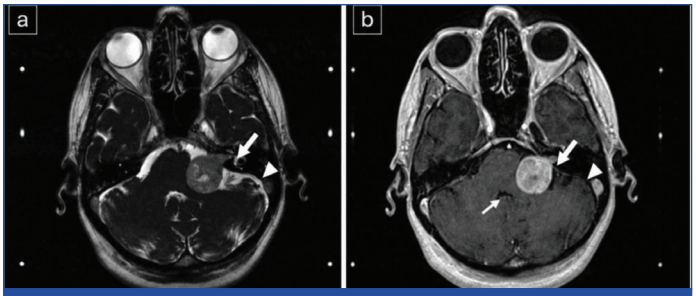


[Table/Fig-7]: Optical Coherence Tomography (OCT) optic nerve head and Retinal Nerve Fibre Layer (RNFL) analysis demonstrating increased thickness corresponding to optic disc oedema without evidence of established optic atrophy.

Laboratory investigations revealed mildly deranged renal function tests suggestive of hypertensive end-organ involvement. Electrocardiography demonstrated hypertensive strain pattern. Echocardiography was advised but could not be performed because the patient declined further cardiac evaluation.

Formal visual field assessment could not be performed because of bilateral no perception of light vision at presentation. MRI of the brain and orbits demonstrated a 3.5×2.8×1.8 cm extra-axial heterogeneously enhancing lesion in the left cerebellopontine angle extending into the internal auditory canal, consistent with vestibular schwannoma [Table/Fig-8a,b].

The lesion exerted mass effect on the adjacent pons with contralateral displacement. There was no evidence of vasogenic oedema,



[Table/Fig-8]: MRI brain: a) Contrast-enhanced T1-weighted image; b) Axial T2-weighted.
(a) Axial T1-weighted MRI image demonstrating a well-defined hyperintense extra-axial lesion in the left cerebellopontine angle (white arrow) with extension into the internal auditory canal, suggestive of vestibular schwannoma; (b) Contrast-enhanced T2-weighted MRI image demonstrating homogeneous enhancement of the left cerebellopontine angle lesion (white arrow) with mild mass effect on the adjacent pons (arrowhead), without evidence of occipital lobe involvement or hydrocephalus

diffusion restriction, hydrocephalus or occipital lobe involvement. Lumbar puncture and cerebrospinal fluid opening pressure measurements were not performed because the patient declined the procedure.

Treatment and Outcome

The patient was admitted under the Department of General Medicine and managed as a hypertensive emergency. Blood pressure was gradually reduced using intravenous labetalol followed by oral antihypertensive medications. Rapid blood pressure reduction was avoided to minimise the risk of further optic nerve ischaemia. Serial blood pressure measurements during admission demonstrated gradual reduction to 200/120 mmHg, 200/120 mmHg, 190/110 mmHg, 180/100 mmHg, 180/90 mmHg and 160/90 mmHg.

The patient was closely monitored for systemic and ophthalmic complications during admission. Conservative ophthalmic management with serial fundus examinations and optical coherence tomography imaging was undertaken. Blood pressure on discharge was 150/90 mmHg, consistent with appropriate management of hypertensive emergency.

At one-month follow-up, visual acuity improved to finger counting at one foot in both eyes. Repeat optical coherence tomography demonstrated mild reduction in optic disc oedema with persistent RNFL thickening. At two-month follow-up, both visual acuity and optical coherence tomography findings remained stable without further significant improvement.

The patient was referred to the department of Neurosurgery for evaluation of vestibular schwannoma. An initial period of observation for one month was advised. Following reassessment, the patient was counselled regarding definitive management options including microsurgical excision and stereotactic radiosurgery. At three-month follow-up, surgical intervention was recommended.

Although partial visual recovery was noted following blood pressure control, the visual prognosis remained guarded, suggesting irreversible optic nerve damage secondary to fulminant hypertensive optic neuropathy.

DISCUSSION

Malignant hypertension is characterised by a sudden and severe elevation in blood pressure resulting in acute target-organ damage involving the cardiovascular, renal, neurological and ocular systems [2,3]. Hypertensive optic neuropathy is an uncommon but potentially devastating ocular manifestation that results from failure of vascular autoregulatory mechanisms, fibrinoid necrosis of optic nerve arterioles and axoplasmic flow stasis leading to optic disc oedema and optic nerve ischaemia [4,5]. Severe bilateral involvement causing profound visual loss is exceedingly rare and remains sparsely reported in the literature.

The present case is clinically significant because the patient initially mimicked cortical blindness. The absence of a relative afferent pupillary defect despite profound bilateral visual loss may be misleading in cases of bilateral symmetric optic nerve involvement. Relative afferent pupillary defect is inherently a relative measure and may be absent when optic nerve dysfunction is equal in both eyes [6]. Consequently, its absence should not exclude severe bilateral optic neuropathy.

Differentiating hypertensive optic neuropathy from cortical causes of blindness is critical in patients presenting with malignant hypertension and bilateral visual loss. Posterior reversible encephalopathy syndrome and bilateral occipital infarction are important differential diagnoses that should be considered [7,8]. In the present case, the presence of bilateral optic disc oedema and grade 4 hypertensive retinopathy together with preserved retinal architecture on optical coherence tomography and normal occipital neuroimaging effectively localised the pathology to the pre-geniculate visual pathway [9].

The co-existence of vestibular schwannoma further complicated the diagnostic process. Vestibular schwannomas may rarely present with papilloedema secondary to raised intracranial pressure when associated with hydrocephalus or significant posterior fossa mass effect [10]. Although MRI demonstrated pontine mass effect, there was no radiological evidence of hydrocephalus or occipital lobe involvement. Furthermore, partial visual improvement following blood pressure control strongly supports fulminant hypertensive optic neuropathy as the primary aetiology of visual loss. Nevertheless, raised intracranial pressure could not be completely excluded because lumbar puncture was declined by the patient and therefore represents a limitation of this report.

Partial visual recovery from no perception of light to finger counting at one foot following treatment of hypertensive emergency highlights the importance of prompt diagnosis and multidisciplinary management. Despite visual improvement, long-term prognosis remains guarded once severe optic nerve involvement has occurred.

This case emphasises the importance of comprehensive clinicoradiological correlation when evaluating profound bilateral visual loss. Clinicians should recognise that hypertensive optic neuropathy may occur in the absence of a relative afferent pupillary defect and may mimic cortical blindness, particularly in the presence of co-existing intracranial pathology.

CONCLUSION(S)

Fulminant hypertensive optic neuropathy is a rare but sight-threatening manifestation of malignant hypertension that may present as bilateral profound visual loss and mimic cortical blindness. Bilateral symmetric optic nerve involvement may occur without a relative afferent pupillary defect, posing a significant diagnostic challenge. Careful clinical examination, multimodal ocular imaging and neuroimaging are essential for accurate localisation of pathology. Co-existing intracranial lesions such as vestibular schwannoma may further confound the diagnosis and should be interpreted within the broader clinical context. Early diagnosis and prompt management of hypertensive emergency may permit partial visual recovery, although visual prognosis remains guarded in advanced disease.

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