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Case Report

Pseudo-Foster Kennedy Syndrome Secondary to Multiple Sclerosis with Hypertensive and Ischemic Overlap: A Rare Neuro-ophthalmic Presentation

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ABSTRACT

Background: Pseudo-Foster Kennedy Syndrome (PFKS) represents an uncommon neuro-ophthalmic condition distinguished by the presence of ipsilateral optic atrophy alongside contralateral papilledema, occurring in the absence of an intracranial mass. This syndrome is generally attributed to a series of injuries to the optic nerve resulting from ischemic or demyelinating events. **Case Presentation:** A 39-year-old male patient exhibited a gradual decline in vision accompanied by headaches. Upon examination of the fundus, right optic atrophy and left disc oedema were observed. MRI scans of the brain and spine revealed numerous periventricular and juxtacortical plaques indicative of Multiple Sclerosis (MS), in addition to acute ischemic infarcts. Notably, severe hypertension was recorded at the time of presentation. Analysis of cerebrospinal fluid indicated a slight increase in protein levels without any mass effect. The interplay of chronic demyelinating optic neuropathy, a hypertensive crisis, and acute ischemic damage resulted in a clinical picture resembling that of PFKS. **Management and Outcome:** The patient received treatment involving intravenous antihypertensives, antiplatelet therapy, statins, and immunomodulation using interferon-β. The papilledema resolved following the management of blood pressure; however, optic atrophy remained. **Conclusion:** This seems to be the initial recorded instance of PFKS in India, resulting from a combination of multiple sclerosis, hypertensive crisis, and ischemic stroke. It emphasises the significance of linking fundus observations with neuroimaging to differentiate PFKS from tumour-associated FKS and stresses the necessity of acknowledging multisystem vascular-inflammatory interactions to avert permanent visual consequences.

Keywords: Pseudo-Foster Kennedy Syndrome, Multiple Sclerosis, Optic Atrophy, Papilledema, Hypertensive Encephalopathy, Ischemic Optic Neuropathy, Neuro-ophthalmology.

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INTRODUCTION

The classical Foster Kennedy Syndrome (FKS) is characterised by a triad consisting of ipsilateral optic atrophy, contralateral papilledema, and usually a mass lesion located in the frontal lobe, most frequently an olfactory groove or anterior cranial fossa meningioma that exerts pressure on one optic nerve. Concurrently, elevated intracranial pressure results in disc swelling in the contralateral eye (Musa & Zeppieri, 2023; Pastora-Salvador & Peralta-Calvo, 2011) This compressive mechanism is fundamental to the condition: direct injury to the optic nerve on the side of the lesion results in atrophy, while increased intracranial pressure due to mass effect causes papilledema in the opposite eye (Bansal *et al*., 2008). The defining imaging characteristic of FKS is the presence of an intracranial space-occupying lesion, typically situated in the frontal or olfactory regions. Consequently, FKS is considered rare, with an estimated occurrence of only about 1% to 2.5% of intracranial tumours (Musa & Zeppieri, 2023; Pastora-Salvador & Peralta-Calvo, 2011). Due to its striking presentation, it underscores the intricate relationship between central nervous system pathology and neuro-ophthalmic manifestations.

In contrast, Pseudo Foster Kennedy Syndrome (PFKS) exhibits similar optic nerve characteristics, specifically optic atrophy in one eye accompanied by contralateral disc swelling, yet occurs without any identifiable compressive intracranial lesion (EyeWiki, 2025; Petramfar *et al*., 2016). The recognised etiologies of PFKS encompass sequential Non-Arteritic Anterior Ischemic Optic Neuropathy (NAION), demyelinating optic neuritis, trauma, Idiopathic Intracranial Hypertension (IIH), optic nerve

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hypoplasia, and various other vascular or inflammatory processes (EyeWiki, 2025; Bansal *et al*., 2008). In contrast to true Foster Kennedy Syndrome (FKS), the mechanism underlying PFKS is non-compressive and frequently multifactorial, indicating asymmetric optic nerve injury and varying vulnerability to elevated intracranial pressure or vascular damage. This clinical differentiation is vital, as the absence of a mass necessitates a comprehensive differential diagnosis and may hinder the timely recognition of underlying systemic conditions (Desai *et al*., 2015).

The demyelinating condition known as Multiple Sclerosis (MS) is widely recognized for its impact on the optic nerve: optic neuritis frequently serves as an initial symptom and can manifest at any point during the progression of the disease, with estimates indicating that as many as 50% of individuals with MS may experience involvement of the optic nerve throughout the course of their illness (Pandit, 2011). Limited studies conducted in India by Zahoor *et al.* in 2017 (Zahoor & Haq, 2017) and Bhatia *et al.* in 2015 (Bhatia *et al*., 2015) validate that MS, which has historically been regarded as uncommon in the subcontinent, is exhibiting a rising prevalence: data from hospitals indicate that earlier incidence rates ranged from 0.17 to 1.33 per 100,000, which have escalated in recent years to 8.35 per 100,000 in certain areas. In a global context, the prevalence of MS is significantly higher, with estimates suggesting approximately 30 to 40 cases per 100,000 individuals in many Western populations (Bottaro, 2025). In rare instances, MS has been identified as a cause of PFKS, typically through asynchronous bilateral optic neuritis that results in sequential optic atrophy in one eye and disc swelling in the other, a clinical presentation that resembles the traditional FKS pattern (Mukhtar *et al*., 2024). This overlap highlights the possibility for demyelinating conditions to present as optic nerve compression syndromes, especially when they occur alongside other optic neuropathic injuries.

In addition to demyelination, vascular and hypertensive mechanisms may exacerbate the neuro-ophthalmic presentation and contribute to a PFKS-like manifestation. Accelerated hypertension can result in increased intracranial pressure accompanied by papilledema (Tajunisah & Patel, 2012), ischemic damage to optic nerve fibres leading to optic atrophy (notably in NAION) (Kaur & Margolin, 2025), and hypertensive retinopathy may further alter the appearance of the optic disc (Hayreh, 2015). In these circumstances, the atrophy of the optic nerve may indicate a prior vascular injury, while the swelling of the contralateral disc could signify persistent elevated pressure or acute ischemia (Behbehani *et al*., 2021). The simultaneous presence of demyelinating, hypertensive, and ischemic conditions in a single patient is exceedingly uncommon in the literature, particularly within the Indian context, and poses a diagnostic challenge due to the interplay of mechanisms that may obscure the primary pathology.

We therefore report this case of pseudo-Foster Kennedy Syndrome in which multiple sclerosis, hypertensive crisis, and ischemic infarction overlapped in a single patient. The purpose of presenting this case is to highlight the diagnostic challenge posed by overlapping neuro-inflammatory and vascular pathology, emphasise the importance of a multidisciplinary approach involving neuro-ophthalmology, neurology and vascular-risk control, and to contribute to the scarce Indian literature on this triple-mechanism interaction in PFKS. This case is reported in accordance with prevailing clinical practice in India, where overlapping demyelinating and vascular disorders are increasingly recognised, yet resource limitations may delay definitive diagnosis. Documenting such atypical neuro-ophthalmic presentations is essential to improve clinical awareness and diagnostic accuracy in similar settings.

CASE REPORT

A 39-year-old male exhibited a sudden onset of painless blurring of vision in the left eye for a duration of fifteen days, accompanied by a holo-cranial headache and weakness in the left upper and lower limbs over the past five days. There was no indication of fever, vomiting, trauma, nausea, or loss of consciousness. The patient had previously experienced a transient visual impairment in the right eye approximately two years ago, which was diagnosed as non-arteritic anterior ischemic optic neuropathy, with partial recovery of vision following treatment. He had a known history of hypertension for five years and reported irregular adherence to antihypertensive medications. There was no family history of diabetes, smoking, alcohol use, or comparable neurological or ophthalmic disorders.

Upon admission, the patient exhibited alertness, orientation and was afebrile. His blood pressure measured 200/110 mmHg, while his pulse rate was recorded at 98 beats per minute. The patient appeared moderately built and well-nourished, showing no signs of pallor, icterus, or pedal oedema. A neurological assessment indicated normal muscle tone and strength rated at 5/5 across all limbs, accompanied by brisk reflexes and bilaterally absent plantar responses. Examination of the cranial nerves revealed bilaterally mid-dilated pupils that reacted sluggishly to light. Evaluation of the fundus indicated optic atrophy in the right eye and papilledema in the left eye, along with a positive relative afferent pupillary defect, a combination that is suggestive of pseudo-Foster Kennedy Syndrome. Visual acuity was measured at 6/9 in the right eye and 6/12 in the left eye, while perimetry showed bilateral inferior field restriction. Visual evoked potential and optical coherence tomography corroborated the presence of optic atrophy in the right eye and disc swelling in the left eye, findings that align with the diagnosis.

Magnetic resonance imaging of the brain identified small acute infarcts affecting the left thalamus and internal capsule area, chronic lacunar infarcts in the bilateral lentiform nuclei and

corona radiata, as well as multiple ill-defined T2 and FLAIR hyperintensities in the periventricular white matter, centrum semiovale, and pons, suggesting an underlying demyelinating process. No mass lesion, midline shift, or compressive pathology was observed. MRI of the spine displayed patchy T2-weighted hyperintense lesions from C2 to C7 and at D2, D4, D6, D7, D9, and D11 vertebral levels, indicative of a demyelinating disorder, most likely multiple sclerosis. Analysis of cerebrospinal fluid showed elevated protein levels (44 mg/dL) and low glucose levels (15 mg/dL), which are consistent with central nervous system inflammation. Oligoclonal band testing was advised but was not available. A two-dimensional echocardiogram indicated mild concentric left ventricular hypertrophy with a normal ejection fraction (60%), ruling out a cardioembolic source. Renal Doppler studies revealed small bilateral cortical cysts without signs of renal artery stenosis.

In light of these findings, a diagnosis of pseudo-Foster Kennedy Syndrome secondary to multiple sclerosis, compounded by ischemic insult and accelerated hypertension, was made. The patient commenced treatment with intravenous labetalol at a dosage of 20 mg three times daily for the management of blood pressure, in conjunction with oral telmisartan 40 mg once daily, atorvastatin 40 mg at bedtime, and a dual antiplatelet regimen consisting of aspirin 75 mg and clopidogrel 75 mg once daily. Additionally, pantoprazole 40 mg was prescribed daily for gastroprotection, while paracetamol 650 mg was provided as needed for headache relief. Despite the rigorous antihypertensive treatment, blood pressure levels remained consistently elevated, fluctuating between 190/100 and 200/110 mmHg during the initial days of hospitalisation. This necessitated an increase in the telmisartan dosage to 80 mg once daily, along with the introduction of nifedipine 20 mg twice daily and oral labetalol at 100 mg three times daily. Over the subsequent week, a gradual improvement was noted, with blood pressure stabilising around 150/80 mmHg by the tenth day of hospitalisation. The headache diminished, and the patient reported a slight enhancement in vision in the left eye.

A repeat examination of the fundus revealed a decrease in disc oedema on the left side, whereas the right optic disc continued to appear pale and atrophic. The patient's overall neurological condition showed improvement, evidenced by a partial alleviation of limb weakness and enhanced visual function. Upon discharge, the conclusive diagnosis encompassed pseudo-Foster Kennedy Syndrome as a consequence of multiple sclerosis, an acute left thalamic and capsular infarct, chronic bilateral lacunar infarcts, hypertensive retinopathy, optic atrophy in the right eye, disc oedema in the left eye, and accelerated hypertension. The patient was prescribed oral telmisartan 80 mg once daily, nicardipine retard 20 mg twice daily, atorvastatin 40 mg at bedtime, clopidogrel 75 mg once daily, aspirin 150 mg once daily, and pantoprazole 40 mg before breakfast, along with recommendations for consistent neurological and ophthalmological follow-up. The initiation of immunomodulatory therapy with interferon beta-1a was advised following further assessment.

Management Rationale

Immediate and aggressive blood pressure control was prioritised to reduce intracranial pressure, prevent further ischemic injury, and facilitate resolution of papilledema, which is consistent with established management principles for hypertensive neuro-ophthalmic complications. Dual antiplatelet therapy and statin treatment were initiated for secondary prevention of ischemic cerebrovascular events, given the presence of acute and chronic infarcts and significant vascular risk factors. Disease-modifying therapy with interferon beta was advised following stabilisation in line with standard management of multiple sclerosis, to reduce the risk of future demyelinating relapses and further optic nerve damage. While papilledema showed clinical improvement following treatment, optic atrophy persisted, reflecting irreversible axonal loss and underscoring the importance of early recognition and intervention in asymmetric optic neuropathies.

This case illustrates an uncommon and intricate manifestation of pseudo–Foster Kennedy Syndrome, arising from a blend of demyelinating and ischemic mechanisms. The presence of multiple sclerosis alongside hypertensive and ischemic alterations highlights the diagnostic difficulties created by the intersection of neurovascular and neuroinflammatory processes. Timely neuroimaging and ophthalmologic assessment were essential in recognising the fundamental pathology. Swift intervention with antihypertensive treatment, dual antiplatelet medications, and a structured immunomodulation approach resulted in stabilisation and partial restoration of vision, underscoring the significance of a multidisciplinary approach in managing such unusual cases.

DISCUSSION

The current case outlines a 39-year-old male patient who exhibited unilateral optic atrophy alongside contralateral papilledema, occurring in the context of poorly managed hypertension, acute thalamic infarcts, and magnetic resonance imaging (MRI) results indicative of Multiple Sclerosis (MS). This combination of clinical findings resulted in a pseudo-Foster Kennedy Syndrome (PFKS) presentation. The observation of optic atrophy in the right eye, coupled with disc oedema in the left eye, in conjunction with demyelinating lesions in both the brain and spinal cord, as well as hypertensive and ischemic alterations, highlights a distinctive interaction between neuro-inflammatory and vascular processes. Although the clinical presentation resembled classical Foster Kennedy Syndrome, the absence of a frontal or olfactory groove mass lesion on neuroimaging excluded a compressive aetiology. The asymmetric optic disc findings in this patient are better

explained by sequential and multifactorial optic nerve injury, consistent with pseudo–Foster Kennedy Syndrome.

The prior episode of non-arteritic anterior ischemic optic neuropathy likely contributed to chronic optic atrophy, while long-standing poorly controlled hypertension predisposed the patient to acute vascular and pressure-related optic disc changes. This historical background provides an important context for understanding the asymmetric optic nerve involvement observed in this case.

In FKS, the defining triad of ipsilateral optic atrophy, contralateral papilledema, and a frontal-lobe mass lesion (commonly an olfactory groove meningioma) results from direct compressive optic neuropathy accompanied by contralateral elevated intracranial pressure (Musa & Zeppieri, 2023). In PFKS, analogous optic nerve manifestations occur in the absence of compression, primarily due to sequential ischemic or demyelinating events (Petramfar *et al*., 2016 & Bansal *et al*., 2008). In the current case, a history of Non-Arteritic Anterior Ischemic Optic Neuropathy (NAION) likely led to chronic optic atrophy in the right eye, while uncontrolled hypertension, coupled with an acute ischemic stroke and MS-related inflammatory activity, resulted in disc oedema in the opposite eye. This dual mechanism elucidates the PFKS-like presentation: chronic optic nerve atrophy resulting from ischemia or demyelination in one eye, and acute papilledema or inflammatory swelling in the other (Costello, 2014).

The demyelinating aspect of Multiple Sclerosis (MS) is a recognised contributor to optic neuritis, resulting in demyelination and ultimately leading to optic atrophy due to axonal degeneration (David *et al*., 2016). Approximately 50% of individuals diagnosed with MS experience involvement of the optic nerve throughout the progression of their illness (Pandit, 2011). In contrast, hypertensive crises can raise intracranial pressure and result in papilledema, while ischemic damage to small vessels can affect the optic nerve head, presenting as Non-Arteritic Anterior Ischemic Optic Neuropathy (NAION) and optic disc pallor (Tajunisah & Patel, 2012 & David *et al*., 2016) When both demyelinating inflammation and vascular ischemia are present, as observed in this scenario, a "dual-pathway" model is proposed, where demyelination leads to chronic atrophy on one side, while hypertensive vascular mechanisms cause papilledema and acute ischemic damage on the other (David *et al*., 2016).

The differential diagnosis encompassed true FKS, NAION, Idiopathic Intracranial Hypertension (IIH), compressive orbital lesions, and sequential optic neuritis. True FKS was ruled out as MRI did not show any intracranial mass lesion. The likelihood of IIH was diminished due to the normal size of the ventricles and the lack of dural venous sinus obstruction. Radiological assessments excluded compressive orbital or sinus lesions. Although sequential optic neuritis, a recognised pattern in Multiple Sclerosis (MS), was taken into account, the presence of fundus asymmetry and a concurrent hypertensive crisis suggested a vascular-inflammatory overlap instead of a purely demyelinating sequential neuritis. The analysis of Cerebrospinal Fluid (CSF) indicated central nervous system inflammation, and when combined with the presence of multiple periventricular and spinal demyelinating lesions, it confirmed the MS component. Additionally, hypertensive retinopathy and infarcts provided evidence for vascular involvement.

Table 1: WHO-UMC causality assessment summary for pseudo-Foster Kennedy Syndrome.

WHO-UMC Criterion	Assessment in the Present Case
Temporal relationship	A prior episode of optic nerve involvement was followed by the acute onset of contralateral disc oedema during a hypertensive crisis and ischemic cerebrovascular event, establishing an appropriate temporal sequence.
Clinical plausibility	Multiple sclerosis-related demyelination, uncontrolled hypertension, and ischemic infarction provide a biologically plausible mechanism for asymmetric optic nerve injury leading to pseudo–Foster Kennedy Syndrome.
Alternative explanations	Classical Foster Kennedy Syndrome was excluded due to the absence of an intracranial mass lesion on MRI. Idiopathic intracranial hypertension, compressive orbital pathology, and isolated sequential optic neuritis were considered but deemed unlikely based on imaging, cerebrospinal fluid findings, and vascular risk profile.
Objective evidence	Fundus examination demonstrated optic atrophy in one eye with contralateral papilledema. MRI of the brain and spine revealed demyelinating plaques consistent with multiple sclerosis and acute and chronic ischemic infarcts, with no evidence of compressive pathology.
Response to intervention	Disc oedema showed improvement following blood pressure control and vascular risk management, supporting the contributory role of hypertensive and ischemic mechanisms; optic atrophy persisted, consistent with irreversible axonal damage.
Consistency with the literature	Previous reports describe pseudo-Foster Kennedy Syndrome arising from demyelinating or ischemic optic neuropathies, supporting the observed mechanism in this case.
WHO–UMC causality category	Probable / Likely

Only a limited number of instances of PFKS secondary to MS have been documented in the literature (Mukhtar *et al.*, 2024 & David *et al.*, 2016) and to our knowledge, none have described the simultaneous occurrence of hypertensive crisis and ischemic stroke within the same clinical presentation. Therefore, this case may signify the first documented occurrence of PFKS resulting from a triad of demyelination, hypertension, and ischemia. The diagnostic difficulty resided in differentiating between pseudotumor cerebri, hypertensive papilledema, and MS-related optic neuritis, all of which can yield similar fundus findings. The integrated application of Visual Evoked Potentials (VEP), Optical Coherence Tomography (OCT), and MRI was crucial in distinguishing chronic from acute optic nerve damage.

Management concentrated on the meticulous control of acute blood pressure to avert further ischemic optic damage, alongside the implementation of dual antiplatelet therapy and statin treatment to mitigate the risk of additional cerebrovascular incidents, as well as long-term immunomodulation utilising interferon-β for multiple sclerosis. Even though optic atrophy persisted as irreversible, prompt intervention led to improvements in papilledema and the preservation of remaining vision. This case underscores the necessity for clinicians to recognise that PFKS should not be automatically equated with a space-occupying tumour; it is essential to take into account the interactions among neuro-immune and vascular systems. In settings with limited resources, particularly in India, where the availability of oligoclonal band testing is not widespread, the correlation between MRI and cerebrospinal fluid remains the cornerstone for the identification of such hybrid pathologies. The documentation of additional cases of this nature will contribute to a better understanding of the relationship between demyelinating and vascular mechanisms in pseudo-Foster Kennedy presentations.

Given the multifactorial nature of the presentation, a structured causality assessment was undertaken to evaluate the relationship between demyelinating disease, hypertensive crisis, ischemic insult, and the development of pseudo-Foster Kennedy Syndrome, which is summarised in Table 1.

CONCLUSION

This case highlights a unique and diagnostically complex intersection of demyelinating, hypertensive, and ischemic mechanisms that result in a pseudo-Foster Kennedy Syndrome presentation, notably in the absence of a compressive lesion. This case highlights that pseudo-Foster Kennedy Syndrome should not be presumed to indicate an intracranial tumour and emphasises the need to consider combined inflammatory and vascular mechanisms, particularly in patients with multiple systemic risk factors. The simultaneous occurrence of multiple sclerosis, hypertensive crisis, and ischemic stroke exemplifies how separate neuro-inflammatory and vascular injuries can amalgamate to replicate the classical features of Foster Kennedy Syndrome. Our case report emphasises the urgent need for prompt neuroimaging and comprehensive evaluation, including fundus imaging, OCT, VEP, and MRI, to distinguish between pseudo variants and true ones and prevent irreversible visual impairment. Acknowledging such hybrid neuro-ophthalmic presentations enhances clinical awareness within the Indian context. It necessitates systematic documentation of analogous cases to further clarify their underlying pathophysiology and improve multidisciplinary management strategies.

PATIENT PERSPECTIVE

The patient conveyed his contentment regarding the enhancement of his vision and overall health following prompt diagnosis and treatment. He valued the collaborative efforts between the neurology and ophthalmology departments and acknowledged the importance of maintaining long-term blood pressure management along with consistent neurological monitoring. He agreed to allow his case to be utilised for academic and educational objectives, recognising its uniqueness and the possible benefits it could provide to medical education.

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AUTHOR CONTRIBUTIONS

SS played a significant role in gathering clinical data, monitoring patient follow-up, and drafting the initial version of the manuscript. PKY was responsible for conceptualising the case report, overseeing the clinical analysis, enhancing the manuscript, and managing communication with the journal. JG contributed to the literature review, data analysis, and editing of the manuscript. DSKJ was involved in documenting the fundus, formatting the manuscript, and compiling references. RRN offered academic advice, conducted a critical review, and granted final approval of the manuscript. BDM was engaged in diagnostic assessments, making treatment decisions, and revising the clinical information before submission. All authors have reviewed and approved the final manuscript and accept responsibility for all facets of the work.

CONFLICT OF INTEREST

The authors affirm that there are no conflicts of interest associated with the publication of this case report.

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ETHICAL APPROVAL AND CONSENT

This case report has been compiled in alignment with the ethical standards set forth by the institution and the Declaration of Helsinki. The patient provided written informed consent for the publication of this case report along with any related clinical details or images. The patient has been assured that all identifying information will be anonymised to ensure confidentiality. Institutional permission was obtained for publication of this anonymised case report, and written informed consent was secured from the patient before submission.

ABBREVIATIONS

FKS: Foster Kennedy Syndrome; **PFKS:** Pseudo-Foster Kennedy Syndrome; **MS:** Multiple Sclerosis; **NAION:** Non-Arteritic Anterior Ischemic Optic Neuropathy; **MRI:** Magnetic Resonance Imaging; **FLAIR:** Fluid-Attenuated Inversion Recovery; **CSF:** Cerebrospinal Fluid; **IIH:** Idiopathic Intracranial Hypertension; **OCT:** Optical Coherence Tomography; **VEP:** Visual Evoked Potential; **BP:** Blood Pressure; **CNS:** Central Nervous System; **LVH:** Left Ventricular Hypertrophy.

SUMMARY

Pseudo-Foster Kennedy Syndrome is an uncommon neuro-ophthalmic presentation in which optic atrophy in one eye is associated with papilledema in the other, without evidence of an intracranial mass. We describe a 39-year-old male who presented with visual complaints and headache in the setting of long-standing, poorly controlled hypertension. Fundus examination showed right optic atrophy with left disc oedema. Neuroimaging ruled out a space-occupying lesion and instead demonstrated features suggestive of multiple sclerosis along with acute and chronic ischemic infarcts. Considering the clinical course, imaging findings, and exclusion of classical Foster Kennedy Syndrome, a diagnosis of pseudo-Foster Kennedy Syndrome due to overlapping demyelinating and vascular pathology was made. Blood pressure control and secondary stroke prevention led to the resolution of disc oedema, although optic atrophy persisted. This case highlights the need to consider combined inflammatory and vascular mechanisms when evaluating asymmetric optic disc findings and underscores the importance of early recognition to prevent permanent visual impairment.

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