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Posterior Ischaemic Optic Neuropathy (Pion) Secondary To Distal Segment Stenosis Of The Left Ophthalmic Artery: A Rare Case Report

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Abstract

Ischaemic optic neuropathy (ION) is a vision-threatening condition caused by impaired arterial blood supply to the optic nerve. Posterior ischaemic optic neuropathy (PION) is a rare subtype that often presents without funduscopic abnormalities in the acute phase, making diagnosis challenging. We report a case of a 50-year-old man who presented with sudden, painless monocular visual loss in the left eye, accompanied by hemisensory deficits. Ophthalmic examination revealed no optic disc abnormalities, while automated perimetry demonstrated an inferior arcuate visual field defect. Brain magnetic resonance imaging showed a right corona radiata infarction, and digital subtraction angiography confirmed stenosis of the distal branches of the left ophthalmic artery. These findings supported the diagnosis of PION secondary to ophthalmic artery stenosis, an uncommon etiology of ION. The patient had uncontrolled hypertension as a major vascular risk factor and was treated with antiplatelet therapy, resulting in clinical improvement of visual acuity. This case highlights the importance of comprehensive vascular evaluation in patients with suspected PION and emphasizes ophthalmic artery stenosis as a rare but significant cause of ischaemic optic neuropathy. Early recognition of the underlying vascular pathology may facilitate appropriate management and help prevent further ischemic complications.

Keywords: Anopia, Anticoagulant, Ischemic Optic Neuropathy, Occlusion.

INTRODUCTION

Ischaemic optic neuropathy (ION) is a vision-threatening disorder characterized by sudden, painless monocular vision loss caused by impaired arterial blood supply to the optic nerve, leading to ischemia and axonal injury. Ischemic damage may occur along any segment of the optic nerve and is clinically classified into anterior ischaemic optic neuropathy (AION), which involves the optic nerve head and typically presents with optic disc edema, and posterior ischaemic optic neuropathy (PION), which affects the retrobulbar segment and shows normal funduscopic findings in the acute phase. Based on etiology, ION is further divided into non-arteritic and arteritic forms; non-arteritic ION is commonly associated with systemic vascular risk factors such as hypertension, diabetes mellitus, hyperlipidemia, and obstructive sleep apnea, whereas arteritic ION is most often related to giant cell arteritis (Kang et al., 2022; Li & Bhattacharya, 2025; Patil et al., 2022).

Epidemiologically, AION represents the vast majority of ION cases. In a previous study analyzing 1,400 patients with ischaemic optic neuropathy, the relative frequencies of AION and PION were reported to be 96% and 4%, respectively. Typical clinical features of ION include sudden painless monocular vision loss, the presence of a relative afferent pupillary defect (RAPD), and characteristic visual field defects, most commonly inferior altitudinal defects. Although AION and PION share overlapping clinical manifestations, PION is considerably rarer and remains a diagnosis of exclusion, underscoring the importance of a thorough understanding of optic nerve vascular anatomy and pathophysiology to ensure accurate diagnosis and appropriate management(Liu et al., 2021; Patil et al., 2022).

In addition to clinical symptoms and signs, the diagnosis of ION also relies on confirmation of stenosis or occlusion of the arteries supplying the optic nerve. Neuroanatomically, the anterior portion of the optic nerve is supplied by the central retinal artery and posterior ciliary arteries, whereas the

posterior segment receives its blood supply from several branches of the ophthalmic artery(Flaxel et al., 2020). Ischaemic optic neuropathy caused by stenosis or occlusion of the ophthalmic artery is rarely reported. Therefore, we present this case to highlight its rarity and to discuss the diagnostic approach and management strategies in patients with ION secondary to ophthalmic artery occlusion.

RESULTS AND DISCUSSION

A 50-year-old man presented with a sudden onset of vision loss in the left eye, beginning in the peripheral visual field. The visual disturbance was painless and was not accompanied by ocular redness. The symptoms had persisted for two weeks prior to hospital admission. The visual complaint was associated with numbness on the left side of the body and face. The patient had a history of uncontrolled hypertension. He had previously been treated as an outpatient by an ophthalmologist and received high-dose steroid therapy, with no clinical improvement over the following week. Subsequently, brain magnetic resonance imaging (MRI) was performed, and the patient was referred to the neurology department for further evaluation to determine the underlying cause of his condition.

On physical examination, the patient was hypertensive, with a blood pressure of 145/97 mmHg. Visual acuity assessment revealed no light perception in the left eye. Neurological examination demonstrated paresthesia and hypoesthesia involving the left hemibody. Laboratory investigations, including complete blood count, blood glucose, renal function tests, liver function tests, and electrolyte levels, were within normal limits. Funduscopic examination showed no significant abnormalities. Automated perimetry revealed an absolute inferior arcuate visual field defect in the left eye (**Figure 1**). Contrast-enhanced brain magnetic resonance imaging revealed an infarction in the right corona radiata, with no evidence of mass lesions or signs of increased intracranial pressure (**Figure 2**). Digital subtraction angiography subsequently demonstrated occlusion of the distal segment of the left ophthalmic artery (**Figure 3**).

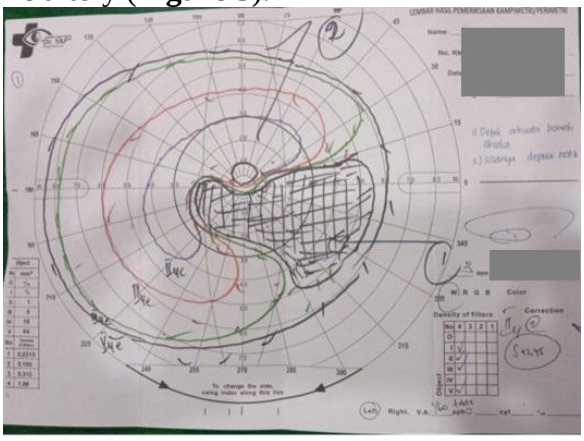

Figure 1. Automated perimetry of the left eye showing an absolute inferior arcuate defect

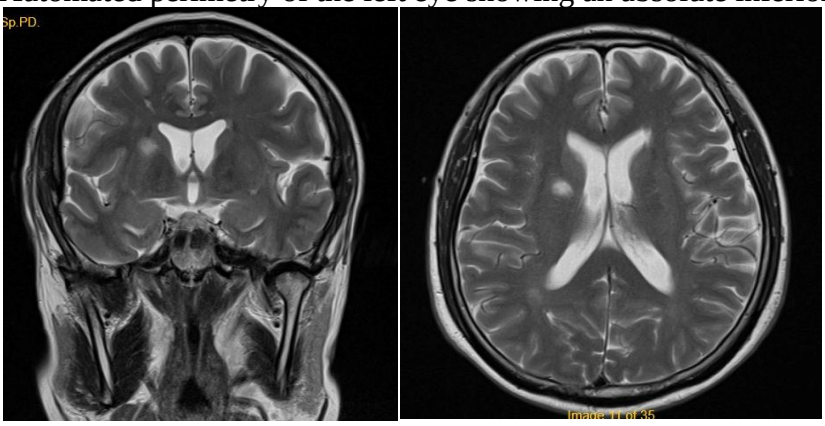

Figure 2. Contrast-enhanced brain MRI showed an infarction in the right corona radiata

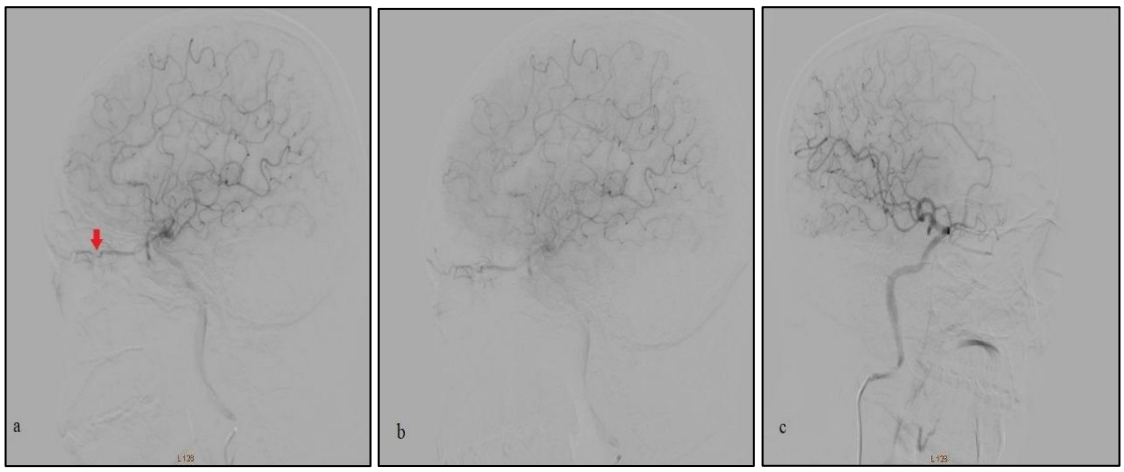

Figure 3. Digital subtraction angiography (DSA) showing: (**a, b**) occlusion of the distalbranches of the left ophthalmic artery (red arrows); (**c**) normal right ophthalmic artery.

According to the findings, posterior ischaemic optic neuropathy (PION) was highly suspected related to his poor control hypertension. The patient was hospitalized for three days and received the following treatments: intravenous ceftriaxone 1 g every 12 hours, intravenous hydrocortisone 100 mg every 12 hours, intravenous omeprazole 40 mg once daily, intravenous citicoline 500 mg every 12 hours, intravenous mecobalamin 500 mg every 12 hours, oral atorvastatin 40 mg once daily, oral clopidogrel 75 mg once daily, and oral aspirin 80 mg once daily. On the third day of hospitalization, the patient's visual acuity showed improvement, and he was discharged for outpatient care. The outpatient treatment regimen included oral atorvastatin 40 mg once daily, oral omeprazole 20 mg twice daily, oral citicoline 500 mg twice daily, oral aspirin 80 mg once daily, oral mecobalamin 0.5 mg twice daily, and oral edoxaban 60 mg once daily at night. The patient was scheduled for regular follow-up at the neurology outpatient clinic for symptom monitoring.

Discussion

This case describes posterior ischaemic optic neuropathy (PION) secondary to distal ophthalmic artery stenosis, an uncommon cause of ischaemic optic neuropathy. The diagnosis was supported by the acute onset of painless monocular visual loss, absence of funduscopic abnormalities in the acute phase, and definitive vascular evidence of distal ophthalmic artery stenosis on digital subtraction angiography (DSA). The coexistence of cerebral infarction and hemisensory deficits further suggests a systemic vascular pathology involving the internal carotid artery circulation.

Ischaemic optic neuropathy (ION) represents optic nerve dysfunction caused by transient or permanent impairment of blood supply to any segment of the optic nerve(Bernstein & Miller, 2015). Clinically, ION typically presents with sudden, painless visual loss accompanied by characteristic visual field defects. Anterior ischaemic optic neuropathy (AION) is associated with acute optic disc edema due to ischaemia of the optic nerve head supplied by the posterior ciliary arteries, whereas PION affects the retrobulbar segment of the optic nerve and usually lacks funduscopic abnormalities in the acute stage, making diagnosis more challenging(Li & Bhattacharya, 2025). Owing to overlapping clinical features, ION may mimic other optic neuropathies such as glaucoma or myelin oligodendrocyte glycoprotein antibody associated optic neuritis, necessitating careful differential diagnosis(Patil et al., 2022).

From an anatomical perspective, the optic nerve can be divided into anterior and posterior segments based on its vascular supply. The anterior portion, or optic nerve head, is primarily supplied by the posterior ciliary arteries, whereas the posterior segment is perfused by the circumferential pial capillary plexus derived from distal collateral branches of the ophthalmic artery and other arterial sources(Hayreh, 2011; Zheng et al., 2020) . Disruption of posterior ciliary artery circulation led to the original description of AION in the early 1970s, while PION was later recognized as a distinct clinical

entity resulting from ischaemia of the posterior optic nerve segment not supplied by the posterior ciliary arteries(Hayreh, 2013).

Posterior ischaemic optic neuropathy is a rare but severe condition, most commonly caused by hypoperfusion or infarction of the pial capillary plexus, which receives blood supply from branches of the ophthalmic artery, internal carotid artery, and vertebrobasilar system(Yang & Lin, 2022). Etiologically, PION is classified into arteritic PION related to giant cell arteritis, non-arteritic PION associated with systemic vascular risk factors, and perioperative PION occurring after prolonged surgical procedures(Li & Bhattacharya, 2025). Non-arteritic PION is considered a multifactorial disease. Hayreh reported a higher prevalence of systemic and vascular risk factors such as hypertension, diabetes mellitus, ischemic heart disease, cerebrovascular disease, carotid and peripheral vascular disease, migraine, and gastrointestinal ulcer disease among patients with non-arteritic PION compared with age-matched controls, suggesting their contributory role in disease development(Hayreh, 2013, 2014). PION remains a diagnosis of exclusion and must be differentiated from retrobulbar optic neuritis, toxic or compressive optic neuropathies, and retinal or macular disorders, with central visual loss being the most frequently reported visual field abnormality(Yang & Lin, 2022).

The diagnosis of ION relies not only on clinical findings but also on confirmation of stenosis or occlusion of arteries supplying the optic nerve. In the present case, DSA demonstrated stenosis of the distal branches of the ophthalmic artery, providing objective vascular evidence of an ischaemic mechanism. Stenosis or occlusion of the ophthalmic artery may compromise blood flow to both the central retinal and posterior ciliary arteries, resulting in impaired choroidal perfusion and visual dysfunction(Flaxel et al., 2020). Preservation of optic disc appearance in this patient supports predominant involvement of the retrobulbar optic nerve segment. Similar cases of ION secondary to ophthalmic artery occlusion have been reported previously, further supporting this vascular etiology(Newman, 2008). Taken together, the clinical, ophthalmologic, and radiologic findings in this patient are highly consistent with PION in the setting of distal ophthalmic artery stenosis.

The exact pathophysiological mechanisms underlying ION remain incompletely understood, although vascular insufficiency is widely regarded as the primary process(Dearaini et al., 2022; Rao et al., 2022). Several studies have suggested shared pathogenic pathways between cerebral infarction and ION, particularly involving hypoperfusion and thromboembolic mechanisms(Aftab et al., 2016). ION is a multifactorial disease associated with systemic risk factors including hypertension, diabetes mellitus, nocturnal hypotension, sleep apnea, cardiovascular disease, and hypercoagulable states(Dearaini et al., 2022). In this case, the presence of uncontrolled hypertension likely contributed to optic nerve hypoperfusion and increased susceptibility to ischaemic injury, consistent with previous reports demonstrating hypertension as a significant risk factor for ION(Lee et al., 2011).

Therapeutic strategies for ION remain limited and largely empirical due to the incomplete understanding of its pathophysiology, with most interventions aimed at preserving residual vision and preventing involvement of the fellow eye(Li & Bhattacharya, 2025). The majority of proposed therapies include agents targeting thrombosis, vascular function, optic disc edema, or neuroprotection(Dearaini et al., 2022). In arteritic forms of ION, high-dose systemic corticosteroids remain the mainstay of treatment, while steroid-sparing agents such as tocilizumab have emerged as effective alternatives. In contrast, the role of corticosteroids in non-arteritic ION remains controversial, and no therapy has been conclusively shown to restore vision in PION, particularly in perioperative cases(Badla et al., 2024; Hayreh, 2011). Management therefore focuses largely on prevention and optimization of systemic vascular risk factors. In the present case, antiplatelet therapy was administered and was followed by improvement in visual acuity, supporting the potential role of antithrombotic strategies in selected patients with underlying vascular risk factors, as suggested in previous reports(Aftab et al., 2016). Despite increasing interest in neuroprotective approaches, their clinical application remains investigational, highlighting the need for further large-scale controlled trials.

CONCLUSION

Ischaemic optic neuropathy (ION) results from inadequate arterial perfusion of the optic nerve and may involve either the anterior or posterior segment depending on the vascular territory affected. In addition to characteristic clinical features, accurate diagnosis requires confirmation of stenosis or occlusion of the arteries supplying the optic nerve. This case highlights distal ophthalmic artery stenosis as a rare but important etiology of posterior ischaemic optic neuropathy (PION), leading to hypoperfusion of the retrobulbar optic nerve. Comprehensive vascular evaluation using digital subtraction angiography was essential in establishing the diagnosis. Early recognition of the underlying vascular pathology and appropriate antithrombotic management were associated with clinical improvement and may help reduce the risk of further ischemic complications, including cerebral infarction, in patients with ION.

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