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Case Report
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Amiodarone-Associated Simultaneous Bilateral Ischemic Optic Neuropathy: A Case Report

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Abstract

Introduction: *Amiodarone is a widely used antiarrhythmic agent associated with multiple systemic adverse effects, including rare but potentially vision-threatening optic neuropathy. Amiodarone-associated optic neuropathy (AAON) remains a controversial entity, with clinical features that may overlap with nonarteritic anterior ischemic optic neuropathy (NAION).*

Case Report: *We report the case of a 56-year-old man with arterial hypertension who developed painless, bilateral sequential visual loss three months after initiation of amiodarone therapy for arrhythmia control. Ophthalmologic examination revealed reduced visual acuity, bilateral optic disc edema with peripapillary splinter hemorrhages, and inferior altitudinal visual field defects with relative macular sparing. Neuroimaging, cerebrospinal fluid analysis, and inflammatory markers were unremarkable. Given the temporal association between drug exposure and symptom onset, amiodarone was discontinued. Optic disc edema persisted for approximately three months, followed by bilateral optic disc pallor. At six-month follow-up, visual acuity and visual field defects remained unchanged, without improvement or further deterioration.*

Discussion: *Amiodarone is a highly lipophilic drug that accumulates within lysosomes, interferes with lipid metabolism, and may disrupt axoplasmic flow through biochemical and mechanical mechanisms. Duration of exposure appears to be a more relevant risk factor than daily dosage. Visual outcomes after drug discontinuation are variable, ranging from improvement to permanent visual impairment.*

Conclusion: *This case highlights the importance of considering AAON in patients receiving amiodarone who present with optic disc edema and visual field defects. Early recognition and appropriate management are essential to optimize visual outcomes.*

Keywords: Optic Neuropathy, Amiodarone, Anterior Ischemic Optic Neuropathy, Amiodarone Associated Optic Neuropathy, Optic Disc Edema.

Introduction

Amiodarone is a di-iodinated benzofuran derivative classified as a class III antiarrhythmic agent. It was originally developed for the treatment of angina pectoris and is currently widely used in the management of atrial fibrillation, ventricular and supraventricular arrhythmias [1]. Numerous systemic adverse effects associated with amiodarone use have been described, including thyroid dysfunction, peripheral neuropathy, interstitial pneumonitis, and ocular abnormalities [2].

Amiodarone is also associated with relevant ocular manifestations, with corneal vortex keratopathy being the most common ophthalmic adverse effect. However, although less frequent, optic nerve involvement represents one of the most concerning

complications, as it may lead to permanent unilateral or bilateral visual loss. Studies on amiodarone-associated optic neuropathy (AAON) have reported a mean duration of exposure of approximately 9 months before the onset of the visual symptoms, with most cases occurring within the first 12 months of therapy, a median daily dose of 200 mg, and initial visual acuity ranging from 20/15 to light perception [3, 4]. Despite these reports, the existence of AAON as a distinct clinical entity remains controversial. Nevertheless, given the large population of patients receiving amiodarone therapy, clarifying and recognizing the potential association between amiodarone use and optic neuropathy is clinically relevant [5].

Case Report

A 56-year-old man with a history of arterial hypertension treated with β-blockers for five years presented with painless, sequential visual loss over a three-day period. Three months prior to symptom onset, amiodarone therapy had been initiated for arrhythmia control.

On examination, best-corrected visual acuity was 20/50 in the right eye (OD) and hand motion in the left eye (OS). Pupillary responses were sluggish bilaterally, without a relative afferent pupillary defect. Intraocular pressure was 15 mmHg in both eyes. Dilated fundus examination revealed bilateral optic disc edema associated with peripapillary splinter hemorrhages (Figure 1). Automated visual field testing demonstrated inferior altitudinal defects in both eyes (Figure 2).

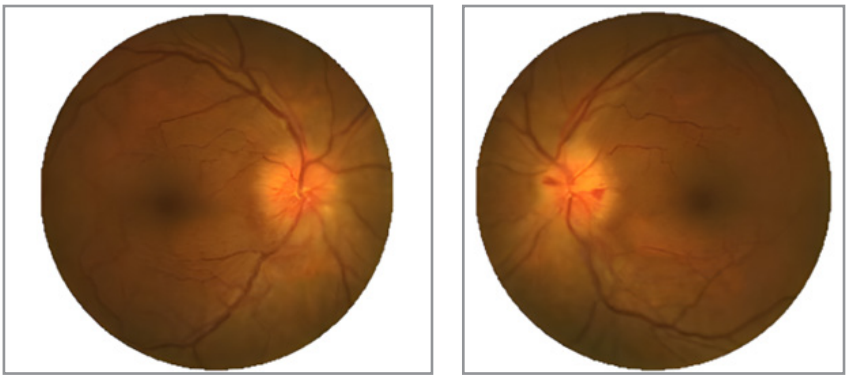

Figure 1: Color fundus Photographs. Color fundus photographs of the right and left eye show bilateral optic disc edema with blurred margins and associated peripapillary splinter hemorrhages.

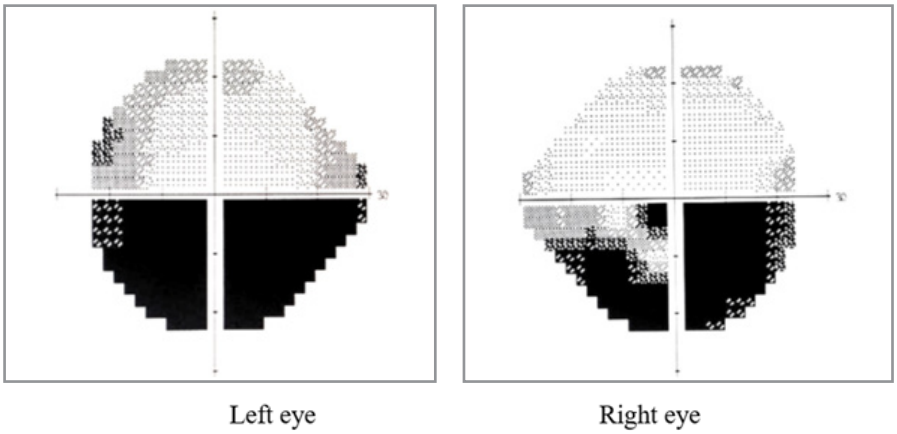

Figure 2: Automated Visual Field Testing. Humphrey visual field testing demonstrates bilateral inferior altitudinal defects.

Neuroimaging with contrast-enhanced magnetic resonance imaging of the brain and orbits demonstrated normal optic nerves and no evidence of compressive or inflammatory pathology. Cerebrospinal fluid analysis, erythrocyte sedimentation rate, and C-reactive protein levels were within normal limits.

Given the temporal relationship between amiodarone use and the onset of visual symptoms, the medication was discontinued in coordination with the patient's cardiologist. Optic disc edema persisted for approximately three months, followed by the development of bilateral optic disc pallor. At six-month follow-up, visual acuity and visual field defects remained unchanged, with no evidence of improvement or further deterioration.

Discussion

Amiodarone is a highly lipophilic drug capable of penetrating lysosomes and interfering with their metabolic function [6]. According to Palmeri et al., amiodarone interferes with cholesterol transport, promoting intracellular lipid accumulation. Histopathologic analyses of ocular tissues have revealed lysosome-like intracytoplasmic membranous lamellar bodies [7]. It has been hypothesized that amiodarone-induced lipidosis may interfere with axoplasmic flow through biochemical or mechanical mechanisms3. In addition, amiodarone may promote vasodilation and induce oxidative stress, which could further contribute to the pathogenesis of optic neuropathy [8].

The estimated incidence of AAON ranges from 0.36% to 2% among patients receiving amiodarone therapy [9]. Studies have demonstrated a higher incidence of amiodarone-associated optic neuropathy among male patients, which may be related to differences in body composition, fat distribution, enzymatic activity, and hormonal influences .

Considerable debate remains regarding whether AAON represents a distinct clinical entity or a variant of nonarteritic anterior ischemic optic neuropathy (NAION), the most common cause of optic nerve–related visual loss in individuals over 50 years of age. These conditions share similar funduscopic findings and systemic risk factors, making differentiation challenging .

The markedly prolonged half life of amiodarone, reported to reach up to 100 days, is considered a principal factor underlying the extended course of optic nerve head edema observed in AAON. In this context, optic disc edema has been documented to persist for 1 to 8 months (with a median duration of 3 months) [10]. This temporal profile contrasts sharply with that of NAION, in which optic disc swelling generally resolves within 6 to 8 weeks, which can reflect the different pathophysiologic mechanisms between AAON and NAION [11].

In an attempt to distinguish these entities, clinical parameters were proposed by Macaluso et al. [12]. The classic presentation of AAON has been described as an insidious, slowly progressive, simultaneous, bilateral optic neuropathy, with visual acuity ranging from 20/20 to 20/200 and prolonged optic disc edema that tends to stabilize within several months after discontinuation of the drug. However, AAON demonstrates considerable clinical variability, further complicating diagnosis.

Duration of amiodarone exposure appears to be more strongly associated with optic neuropathy than daily dosage. A meta-analysis demonstrated that the use of amiodarone longer than 12 months increased the odds of ocular adverse effects, even at relatively low doses (152–330 mg/day) [13].

Following identification of the condition, management typically involves discontinuation of the drug. In one study, visual acuity improved in 58% of patients after amiodarone cessation, whereas 21% progressed to legal blindness at one-year. Nevertheless, visual acuity can remain stable despite cessation of therapy [6].

The other relevant and most frequently encountered ocular manifestation associated with the systemic amiodarone therapy is the corneal vortex keratopathy, which can occur in nearly all patients (more than 95%) receiving amiodarone at doses as low as 200 mg/day [14]. Although epithelial deposits may appear within the first week of treatment initiation, they more typically become evident after one to three months of continuous exposure. The degree of keratopathy demonstrates a clear relationship to both daily dosage and cumulative drug burden, with higher total exposure corresponding to more pronounced corneal involvement. Visual acuity is generally preserved, although patients may experience photophobia, halos, and ocular irritation [15]. Because corneal deposits are seldom symptomatic, treatment is generally not discontinued on this basis.

In patients receiving amiodarone who develop optic disc swelling or optic neuropathy, the presence of corneal vortex keratopathy may serve as supportive evidence of drug exposure, given its frequency and early onset. Clinical decision making must account for the substantial cardiovascular comorbidities common in this population, which often complicate considerations regarding medication cessation. In the present case, amiodarone was discontinued in coordination with cardiology, yet no improvement in visual function was observed. Although the optic disc edema resolved over three months, the patient subsequently developed bilateral optic disc pallor, suggesting that structural optic nerve injury had already occurred.

Conclusion

Amiodarone-associated optic neuropathy is a severe adverse effect with multifactorial pathophysiology. Its clinical presentation may overlap significantly with nonarteritic anterior ischemic optic neuropathy, complicating diagnosis. Duration of exposure appears to be a more relevant risk factor than daily dosage. Although discontinuation of amiodarone may result in visual improvement in some patients, visual function may remain unchanged or progress to permanent impairment, underscoring the need for early recognition. Cardiology consultation is advised before any therapeutic modification is undertaken.

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Conflicts of Interest

None.

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Ethics Approval and Informed Consent

Informed consent was obtained from the patient.

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