

WHEN CARDIAC DRUGS THREATEN SIGHT: A CASE OF AMIODARONE-ASSOCIATED OPTIC NEUROPATHY

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INTRODUCTION

Cardiovascular disease remains a leading cause of mortality worldwide¹. Since its introduction in the 1970s, amiodarone has become a widely used anti-arrhythmic and is included in resuscitation guidelines for shockable cardiac arrest². Despite its efficacy, it has an extensive side effect profile requiring specialist initiation and monitoring. Toxicity can affect multiple organs, including the lungs, thyroid, liver, and gastrointestinal tract. Ocular side effects are common but usually not sight-threatening, with verticillate keratopathy occurring in up to 70–100% of patients³. Rarely, amiodarone can cause optic neuropathy, first described in 1987⁴, which can mimic non-arteritic ischaemic optic neuropathy.

CASE

History of present illness

- 66-year-old Caucasian male presented to clinic with progressive painless blurred vision in both eyes, worse in the left eye
- Denied head injury, headache, facial droop, weakness, fever

Pertinent ocular history

- Blepharitis and dry eye
- Known colour blindness

Pertinent Medical History

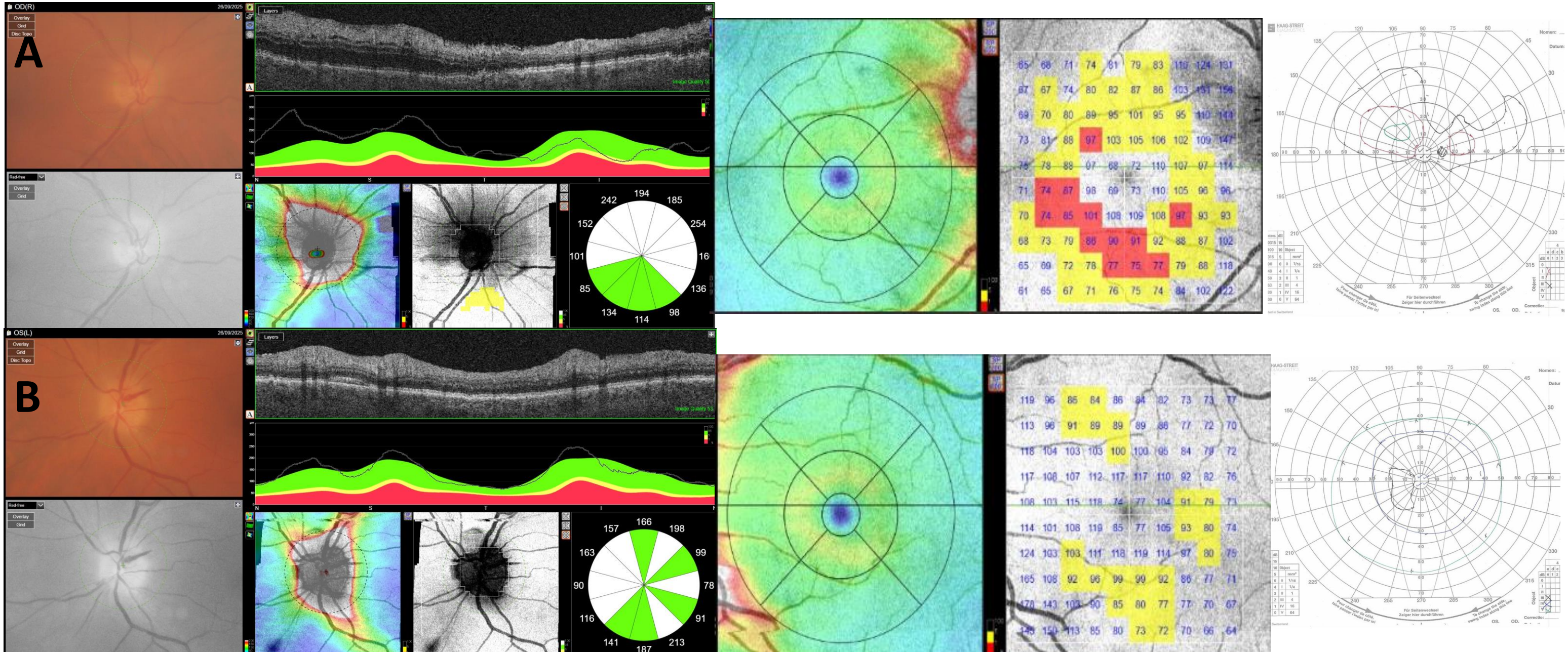
- Atrial fibrillation- started amiodarone 200mg OD March 2025, stopped September 2025

Clinical Findings

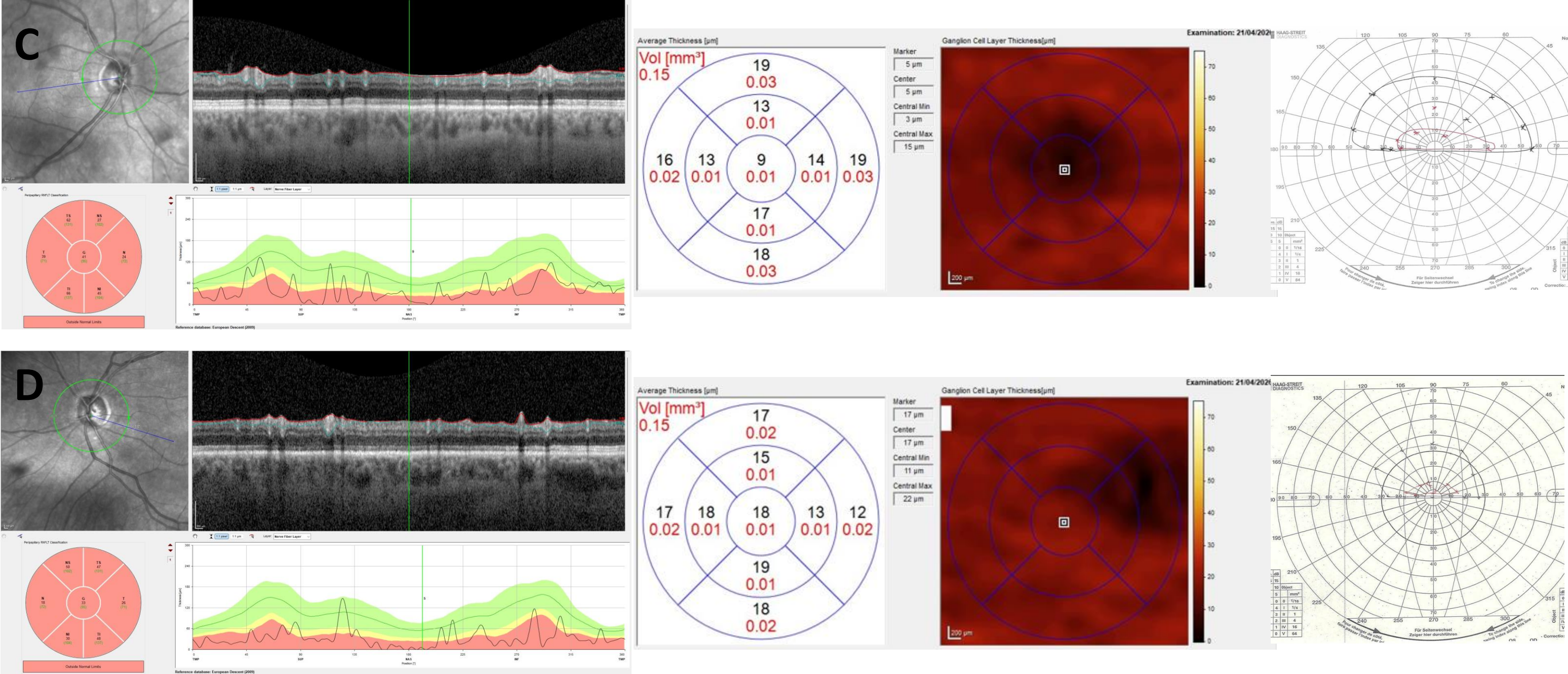
- VA: OD 0.62 OS CF
- IOP: OD 13mmHg OS 15mmHg
- Pupils: OD PERLA OS RAPD
- Anterior segment: OU blepharitis, corneal verticillata, 1 + NS cataract
- DFE: OU Bilateral optic disc swelling, flame haemorrhages
- VF: OD Inferior altitudinal defect OS Full field defect

Laboratory Data/ Imaging

- CT head: unremarkable intracranial appearance
- MRI head: no acute infarct; mild small vessel disease
- FBC, CRP, ESR, AQP4, MOG IgG, syphilis, autoimmune serology NAD
- VEP: Bilateral optic nerve dysfunction. Demyelination not demonstrated.



(A) Right before (B) Left before (C) Right after (D) Left after optic nerve appearance, macular ganglion cell loss and Goldmann visual field demonstrating constricted visual fields in Amiodarone Associated Optic Neuropathy



Clinical Course

He had stopped taking amiodarone, under cardiologist advice, the week he presented to ophthalmology.

Due to progression of visual decline in the right eye to counting fingers, the patient was given a short course of oral Methylprednisolone 500mg once a day for 5 days.

He eventually underwent uncomplicated right eye cataract surgery on request to improve visual brightness; he was aware of guarded visual prognosis.

His final visual outcome was OD 0.94 (0.82 pinhole) and counting fingers in the left eye. He was eventually issued a certificate of visual impairment.

DISCUSSION

Amiodarone-associated optic neuropathy (AAON) is a rare but important differential in patients presenting with visual decline associated with optic disc swelling and visual field defect, especially those receiving amiodarone therapy. This case highlights the characteristic insidious onset, slow progression, bilateral involvement and male predominance often seen in AAON⁵.

Differentiation from non-arteritic anterior ischaemic optic neuropathy (NAION) is challenging due to overlapping features. Furthermore, patients taking amiodarone often have risk factors for vascular disease. However, Wang and Cheng propose a diagnosis of AAON should be considered when the following criteria are present: (1) a clear history of taking amiodarone, (2) insidious onset, (3) slow progression, (4) bilateral visual loss, (5) protracted disc swelling⁶.

Diagnosis is still one of exclusion, supported by normal inflammatory markers, negative infectious and autoimmune workup, and unremarkable neuroimaging, as seen in our case. Only following cessation of amiodarone, and later gradual resolution of disc swelling and visual improvement, can a diagnosis of AAON be made for certain.

Amiodarone is amphophilic, has a high bioavailability and large volume of distribution. These properties are thought to contribute to the accumulation of the drug in lysosomal inclusion bodies in the optic nerve, disrupting axoplasmic flow⁷. Furthermore, with a long half-life of approximately 100 days, the effects of amiodarone on the optic nerve can persist even after drug discontinuation⁶. This is further exacerbated by prolonged use of up to 12 months as per NICE recommendations post-cardioversion⁸.

Prognosis can be variable following cessation. Passman et al reviewed 296 cases of AAON and showed 58% patients with improved visual acuity, 21% with unchanged, and 21% with further visual impairment⁹. Unfortunately, our patient fits into the latter categories.

CONCLUSION

In conclusion, AAON is a diagnostic challenge due to its overlap with other optic neuropathies including NAION. Clinical features in favour of AAON include, slow onset, bilateral involvement, optic disc swelling, and resolution that can take months. A high index of suspicion is needed in patients taking amiodarone as prompt cessation of amiodarone can provide the best visual prognosis for patients. However, this must be done safely and under the supervision of a cardiologist. Furthermore, we propose patient counselling at the initiation of treatment with recommendations of yearly eye tests and OCT scans which can be performed in the community.

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