One potential cause of flow voids is shadowing from overlying structures such as retinal layers and vitreous or lens opacities; however, there was no apparent OCT or OCTA signal attenuation in the retina or underlying choriocapillaris at presentation (Fig 2 A2, C2), making this possibility unlikely. Another cause for flow voids is the low velocity of flow such that it falls under the OCTA decorrelation threshold for detection (less than 0.3 mm/s in this case). This is also unlikely given the clinical setting and significant improvement of signal after treatment using the same machine. One may test this possibility using a custom variable interscan time analysis hardware—software framework to detect slower flow.

This case signifies the value of multimodal imaging and particularly OCTA not only to diagnose a life-threatening condition but also to utilize this imaging technique to determine the extent of choroidal infiltration at a microscopic level and to monitor the course of ocular disease with restoration of choroidal vascular flow indicating a response to treatment.

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Late progression of visual loss from ocular quinine toxicity

A 64-year-old Caucasian man was admitted to hospital with acute loss of vision after ingesting at least 3 grams of quinine, risperidone, and paracetamol. His medical history was significant for schizophrenia and nocturnal leg cramps. His usual medications were risperidone and quinine as required.

No ophthalmic history was noted. An inpatient examination revealed bilateral no-light-perception vision and grossly constricted visual fields. Electrocardiogram showed sinus rhythm and blood tests, including glucose, haematology, liver, renal, coagulation profiles, and paracetamol levels were normal. The patient was given supportive therapy and empirical high dose vitamin C and E supplements. No gastrointestinal decontamination was attempted given the
timing of his presentation (>24 hours). He reported partial a return of vision 10 days after overdosing. At the 3-month review, visual acuities (VA) were near normal: 6/5 OD and 6/7.5 OS. Pupillary responses were sluggish. Anterior segment examinations were normal, with no abnormalities of iris structure detected in either eye. Fundus examination revealed optic atrophy, nerve fibre layer loss, and retinal arteriole attenuation. Colour vision was 0/15 bilaterally (Ishihara plates). Intraocular pressures were 10 mm Hg OD and 12 mm Hg OS. Goldman visual fields showed asymmetric constriction, worse in the right eye (Fig. 1 (A)).

For the first 7 years, he was reviewed annually and maintained stable VA, visual fields, and colour vision. After this time, he did not attend regular ophthalmology care for 12 years. Nineteen years after diagnosis, he re-presented with deteriorating vision. VA was 6/12 OD and 6/7.5 OS.
Ocular examination findings were unchanged. Automated visual field analysis (Humphrey, Zeiss, Meditec, Germany) showed mean deviation of −27.20 decibels (dB) OD and −28.26 dB OS and binocular Esterman an efficiency of 34%. Optical coherence tomography (Spectralis, Heidelberg, Germany) showed inner retinal atrophy (Fig. 2 (B)). At the most recent review, 25 years after initial presentation, he complained of progressive dimming of vision, poor contrast sensitivity, and episodes of visual loss of 5–10 seconds. VA was 6/15 OD, 6/12 OS. Clinical examination and ancillary testing were unchanged, and fundus photography shows expected optic nerve pallor, attenuated retinal arteriolar vasculature (Fig. 2 (A)). Binocular Esterman efficiency had reduced to 24% (Fig. 1 (E) and

Fig. 2—25 years after toxic insult: (A) Fundus photography demonstrating bilateral optic nerve head pallor and retinal vessel attenuation, (B) Optical coherence tomography of bilateral posterior poles demonstrating inner retinal thinning.
Quinine is derived from cinchona trees and was historically utilised for the prevention of malaria and as a muscle relaxant. It has a narrow therapeutic index. It is most regularly taken by patients off-label for leg cramps, with limited evidence for efficacy. Symptoms of quinine toxicity include visual disturbances, tinnitus, headaches, nausea, abdominal pain, acute cardiac dysrhythmias, and convulsions. Toxicity appears to be dose related, occurring in 80% of individuals ingesting over 5 grams (16.5 tablets) and 1% in patients ingesting less than 1 gram (3 tablets). Plasma concentration greater than 10–15 mg/mL is associated with ocular toxicity.1 Although systematic toxicity usually subsides once plasma concentration falls, ocular toxicity, in contrast, has long lasting and sometimes permanent effects. In the acute setting, gastric lavage can be attempted, but quinine and its metabolites are unsuitable for hemodialysis. Trial treatments for ocular toxicity have included retrobulbar vasodilators, anterior chamber paracentesis, stellate ganglion block, and hyperbaric oxygen, with little evidence for efficacy. Clinically, important differentials to exclude are other toxic optic neuropathies, nutritional optic neuropathy (vitamin B1, B2, B6, B12), infectious or inflammatory optic neuropathy, and normal tension glaucoma.

Many patients with quinine ocular toxicity typically present with poor vision and a relatively normal fundus appearance within hours after ingestion. This is followed by a variable recovery of vision, with the appearance of optic disk pallor and retinal arteriole attenuation, usually evident within a few days to weeks. This is often described to as a “paradox of quinine toxicity”. Iris muscle atrophy is common and pupils may remain dilated and nonreactive. There should be a variable recovery in colour vision, visual fields and pupil responses over time. However, permanent blindness can occur in 27% of cases.2 The mechanism for vision loss is incompletely understood and has been classically theorised as toxic optic neuropathy. More recently, a contribution from inner retinal cell layer loss has been proposed, supported by optical coherence tomography scanning, showing early inner retinal edema with late atrophy; full-field and multifocal electoretinography (outside the central 5 degrees) showing reduced b-waves; and automated visual fields showing generalised constriction rather than centrocaecal scotoma.3,4

Over the 25 years of follow-up, our case illustrates an unusual natural history with delayed progressive visual field loss, as in most cases the visual dysfunction is stable over time. We postulate that this pattern of visual field loss may be attributed to inner retinal cell layer loss reaching a “threshold level,” from initial the quinine toxicity and subsequent normal age-related losses. Beyond this threshold, more rapid and symptomatic visual field loss can occur, and retinal fibre nerve layer imaging does not correlate well with visual field function.5 From this, we draw parallels to glaucomatous optic neuropathy, which can progress in a similar fashion. This potential for progression highlights the importance of routine clinical reviews, even after a diagnosis of stable ocular quinine toxicity.


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