Bitemporal peripapillary hemorrhages: Toxic-ischemic optic neuropathy caused by ethanol and cocaine abuse

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A 36-year-old man presented complaining of bilateral painless vision loss. He was admitted with chronic ethanol abuse, as well as sporadic cocaine consumption three days before symptom onset. General medical evaluation dismissed cerebral and cardiovascular events. Blood tests revealed folic acid deficiency. Visual acuity (VA) was count fingers in both eyes (OU). Fundoscopic findings included remarkable bitemporal peripapillary hemorrhages. A diagnosis of toxic-ischemic optic neuropathy was made. The hemorrhages resolved after three weeks, with VA recovering to 20/20 OU. The sequelae included bitemporal peripapillary retinal fiber loss on optical coherence tomography, and central scotomas observed on visual field tests. This is the first report of cocaine as a triggering agent for a classical presentation of toxic optic neuropathy and the presence of peripapillary hemorrhages in its initial stages.
Fig. 1. Retinographies. A and B: Bilateral peripapillary hemorrhages of the right eye and left eye, respectively. C and D. Week 1 follow-up visit, exhibiting progressive reabsorption of the hemorrhages. E and F: Month 3 follow-up visit, showing absence of bleeding component, but bitemporal pallor of the optic discs.

Fig. 2. Spectral domain optical coherence tomography (SD-OCT). A. Macular OCT at onset: normal foveal contour. No edema nor increased reflectivity from the outer plexiform layer to the ganglion cells layer were observed on the b-scans.
discs was detected (Fig. 1E and F). The central scotomas deepened (Fig. 4C and D), and the ON OCT revealed gradual loss of bitemporal peripapillary retinal nerve fiber layer (RNFL) thickness (Fig. 5B). Folic acid levels had increased to 5.01 ng/mL (normal range 4–16 ng/mL). Supplementation was maintained.

The patient presented with a favorable evolution in terms of VA, color vision test, and perimetry, after eight months of follow-up. The VAs were 20/25 and 20/20 in the right and left eye, respectively. He could identify 16/16 of the Ishihara color plates OU and showed scotoma improvement in the VFT (Fig. 4E and F).

2. Discussion

Toxic optic neuropathy (TON) results from progressive bilateral injury of the PMB, which manifests as painless vision loss, dyschromatopsia, and central or cecocentral scotomas. Bitemporal disc pallor is the most common finding once toxicity is established. Nevertheless, swelling and peripapillary hemorrhages may occur in the initial stages. The onset is usually gradual, except in methanol intoxication, wherein symptoms occur acutely.

Oxidative damage is considered as the physio-pathological mechanism, since PMB fibers have the highest energy requirements. Several types of TON have been reported. One group comprises those purely derived from medication intake (ethambutol, linezolid, chloramphenicol, and methanol, among others). Another comprises a combination of toxic exposure and secondary nutritional deficiencies, which would worsen oxidative damage, as observed in ethanol or tobacco abuse.

The sympathomimetic substance cocaine is responsible for inflammation, arterial vasoconstriction, and platelet aggregation. To date, unilateral compressive optic neuropathies related to chronic intranasal abuse have been described. There are some reports of ON compression due to chronic sinusitis and contiguous midline destructive masses observed on magnetic resonance imaging scans that exhibit focal inflammatory granulomatous tissue on biopsy. Moreover, a purely ischemic entity was reported by Bahaya Álvarez Y. et al. (2008) in a sporadic cocaine abuser. They observed a unilateral hyperemic disc with a remarkable hemorrhage, leading to the suggestion of induced vasospasm of the short posterior ciliary arteries.

Herein, we describe a case of TON that presented with a classical bitemporal pattern but started clinically with a marked decrease in VA and a notable hemorrhagic component. We hypothesize the confluence of two factors. First, this patient presented with a pre-existing vulnerability of the axons of the PMB due to chronic ethanol exposure. Swelling of these axons could affect the regional capillaries, making them more susceptible to rupture under high blood pressure. Therefore, the cocaine-induced arterial hypertension in this individual resulted in focal retinal bleeding within the PMB.

Cocaine may have acted as an accelerating agent of PMB damage concurrent with ethanol abuse, represented by posterior gradual bitemporal RNFL loss on OCT.

3. Conclusions

This report illustrates the initial stage and evolution of bitemporal peripapillary hemorrhages in the context of TON and is the first reported case of cocaine as a potential triggering agent.

Patient consent

Written consent to publish this case has not been obtained. This report does not contain any personal identifying information.
Fig. 4. Perimetry exams. A and B. Visual field test (VFT) for left eye (OS) and right eye (OD), respectively, performed after one week, detected central scotomas with a mean deviation (MD) of 4.1 dB OS and 5.7 dB OD. Low reliability (33% false negatives and 33% false positives OU) was attributed to poor initial visual acuity. C and D. Reliable VFT performed at month three exhibited persistence of bilateral deep central scotomas, with an MD of 6.7 dB OS and 3.5 dB OD. E and F. VFT at month eight demonstrated scotomas improvement, with an MD of 0.9 dB OS and 1.4 dB OD.
Fig. 5. Optic nerve OCT. A. Retinal nerve fiber layer (RNFL) thickness in range after three weeks of follow-up. B. Progressive loss of bitemporal peripapillary RNFL thickness after three months of follow-up.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

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