American Journal of Ophthalmology Case Reports 25 (2022) 101374

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: www.ajocasereports.com/

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

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ARTICLE INFO

Keywords:
Toxic optic neuropathy
Peripapillary hemorrhages
Drugs abuse
Nutritional deficiency

ABSTRACT

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.

1. Case report

A 36-year-old man with a three-day history of subacute bilateral painless vision loss reported sporadic intranasal cocaine consumption three days before evaluation, and a calculated ethanol abuse of approximately 150 g daily for the preceding eight months. He had a medical history of alcoholic liver disease. His familiar and personal ophthalmological records were unremarkable.

Visual acuity (VA) was determined as count fingers in both eyes (OU) and fundoscopy revealed bilateral peripapillary hemorrhages, limited to the area of the papillo-macular bundle (PMB), as illustrated in Fig. 1A and B. A macular optical coherence tomography (OCT) scan (Fig. 2) revealed a normal foveal contour and no signs of edema or ischemia within the inner layers. The patient could not distinguish any Ishihara plates.

Emergency room evaluation dismissed cerebral or cardiovascular events after obtaining a contrast-enhanced cranio-orbital computed tomography scan (Fig. 3) and an electrocardiogram, both without remarkable findings. His blood pressure was 151/112 mmHg. The coagulation function and hemogram showed a subtle increase in the mean corpuscular volume to 98.8 fL (normal range 80–94 fL). Liver enzyme tests revealed hypertransaminasemia of 223 lU/L serum glutamic oxaloacetic transaminase (SGOT) (10–38 lU/L) and 79 lU/L serum glutamic pyruvic transaminase (SGPT) (10–37 lU/L). The SGOT/SGPT ratio was 2.82. Serum gamma-glutamyl transferase was increased at 1210 lU/L (7.0–50) and alkaline phosphatase to 270 UI/L (40–145 lU/L). Inflammation marker levels were elevated: C-reactive protein was 29.88 mg/L (1.00–10.0 mg/L) and erythrocyte sedimentation rate was 63 mm/h (0–10 mm/h).

Blood tests for B vitamins detected normal levels of thiamine (B1; 105.5 nmol/L, [66.5–200 nmol/L]) and cyanocobalamin (B12; 841 pg/mL [180–914 pg/mL]), but folic acid deficiency (B6; 1.56 ng/mL [(4–16 ng/mL]).

Toxic-nutritional optic neuropathy was diagnosed. An ethanol discontinuation program and daily 5-mg folic acid supplementation were initiated.

After one week, VA improved to 20/125 in the right eye and 20/100 in the left eye, and progressive reabsorption of the hemorrhages was observed (Fig. 1C and D). A first visual field test (VFT) was performed, which revealed bilateral central scotomas, but the poor VA suggested low test reliability (Fig. 4A and B).

VA recovered to 20/20 OU at week three, along with complete resolution of the hemorrhages. Reliable perimetry examinations confirmed the bilateral central scotoma. The optic nerve (ON) OCT scan is shown in Fig. 5A.

After three months of follow-up, progressive bitemporal pallor of the

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https://doi.org/10.1016/j.ajoc.2022.101374
Received 19 January 2021; Received in revised form 26 January 2022; Accepted 26 January 2022
Available online 3 February 2022

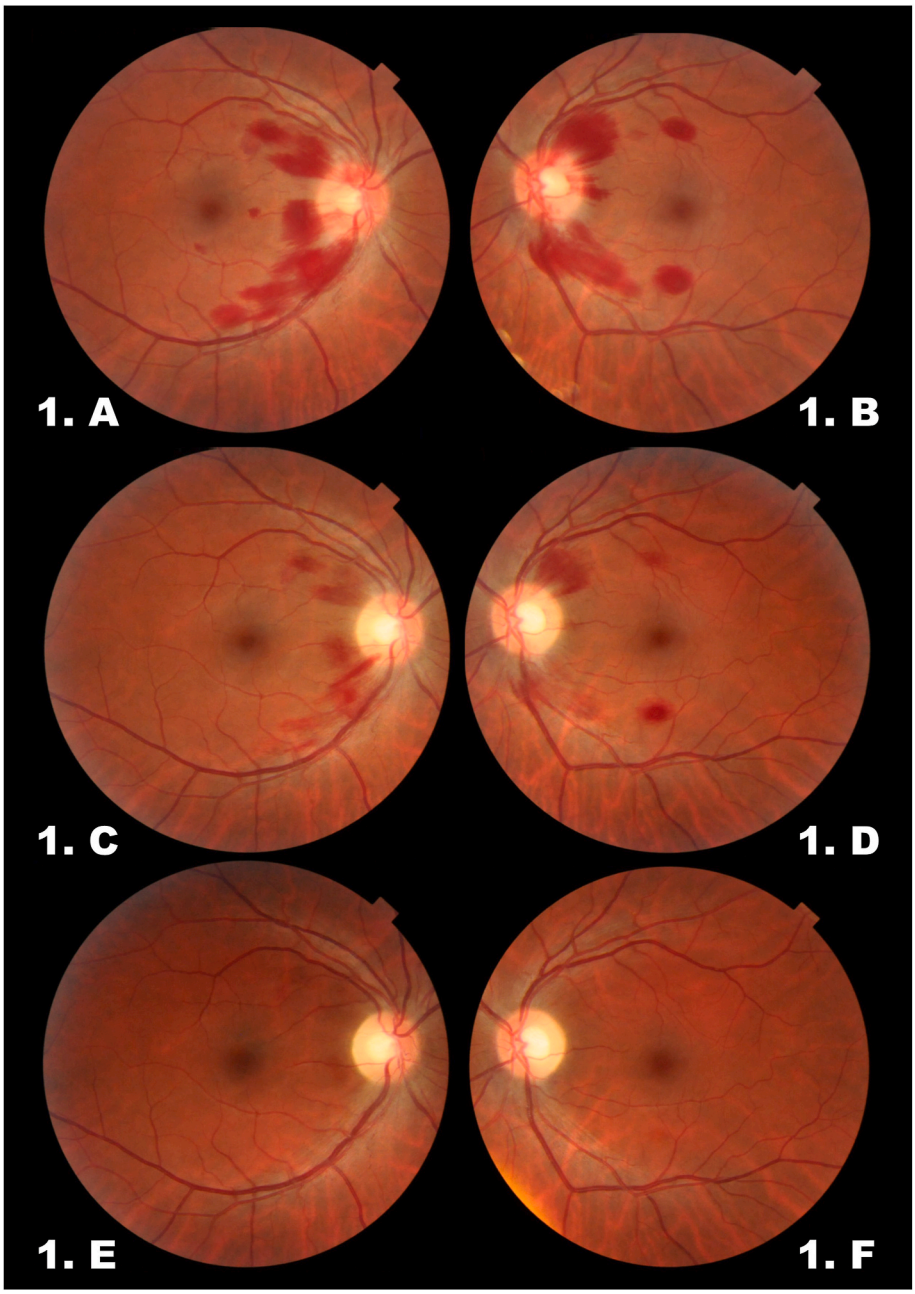

Fig. 1. Retinographies. A and B: Bilateral peripapillary hemorrhages of the right eye and left eye, respectively. C and D. Week 1 follow-up visit, exhibiting progressive reabsorption of the hemorrhages. E and F: Month 3 follow-up visit, showing absence of bleeding component, but bitemporal pallor of the optic discs.

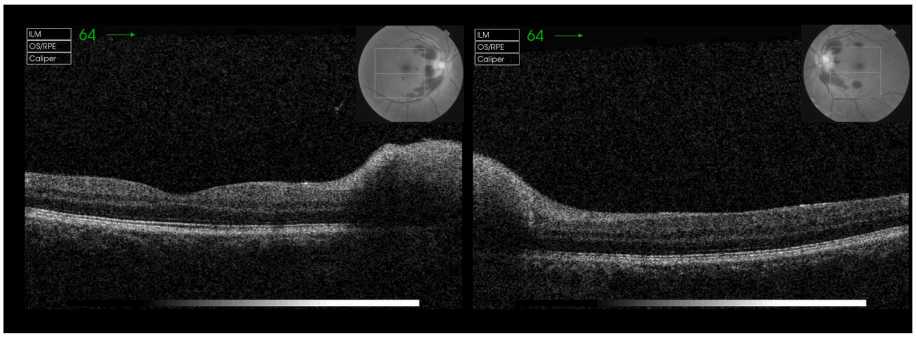

Fig. 2. Spectral domain optical coherence tomography (SD-OCT). A. Macular OCT at onset: normal foveal contour. No edema nor increased reflectivity from the outer plexiform layer to the ganglion cells layer were observed on the b-scans.

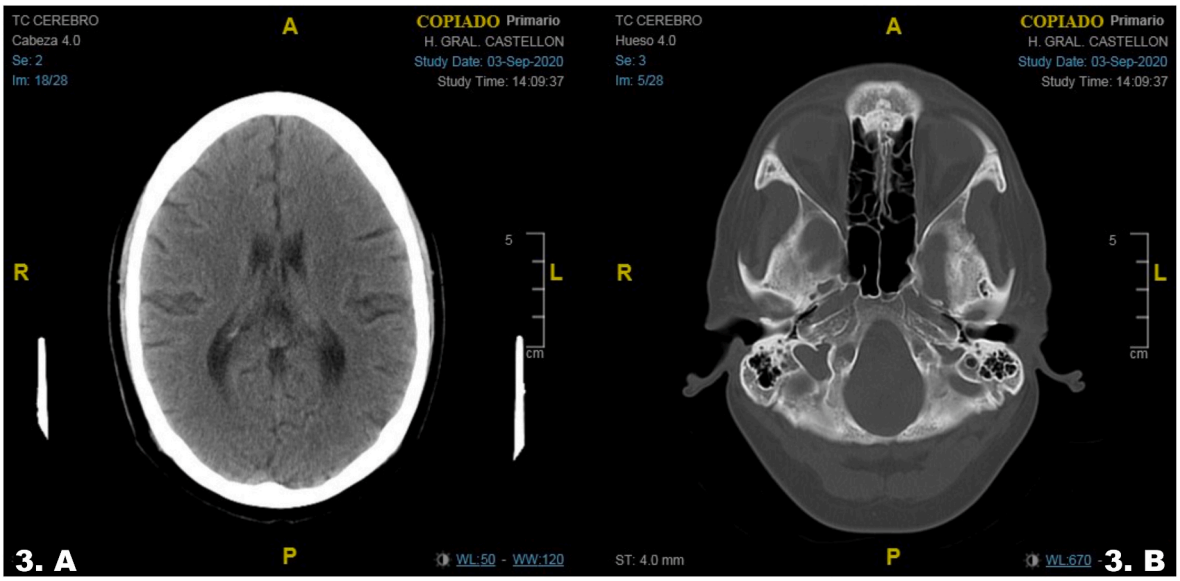

Fig. 3. Cranio-orbital computed tomography. A. Brain window image showing an axial plane with normal cerebral tissue. B. Bone window image showing an orbital axial section, dismissing compressive masses or abnormal optic nerves' anatomy.

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.[1] 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.[1]

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.[1]

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.[2]

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.[3]

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.

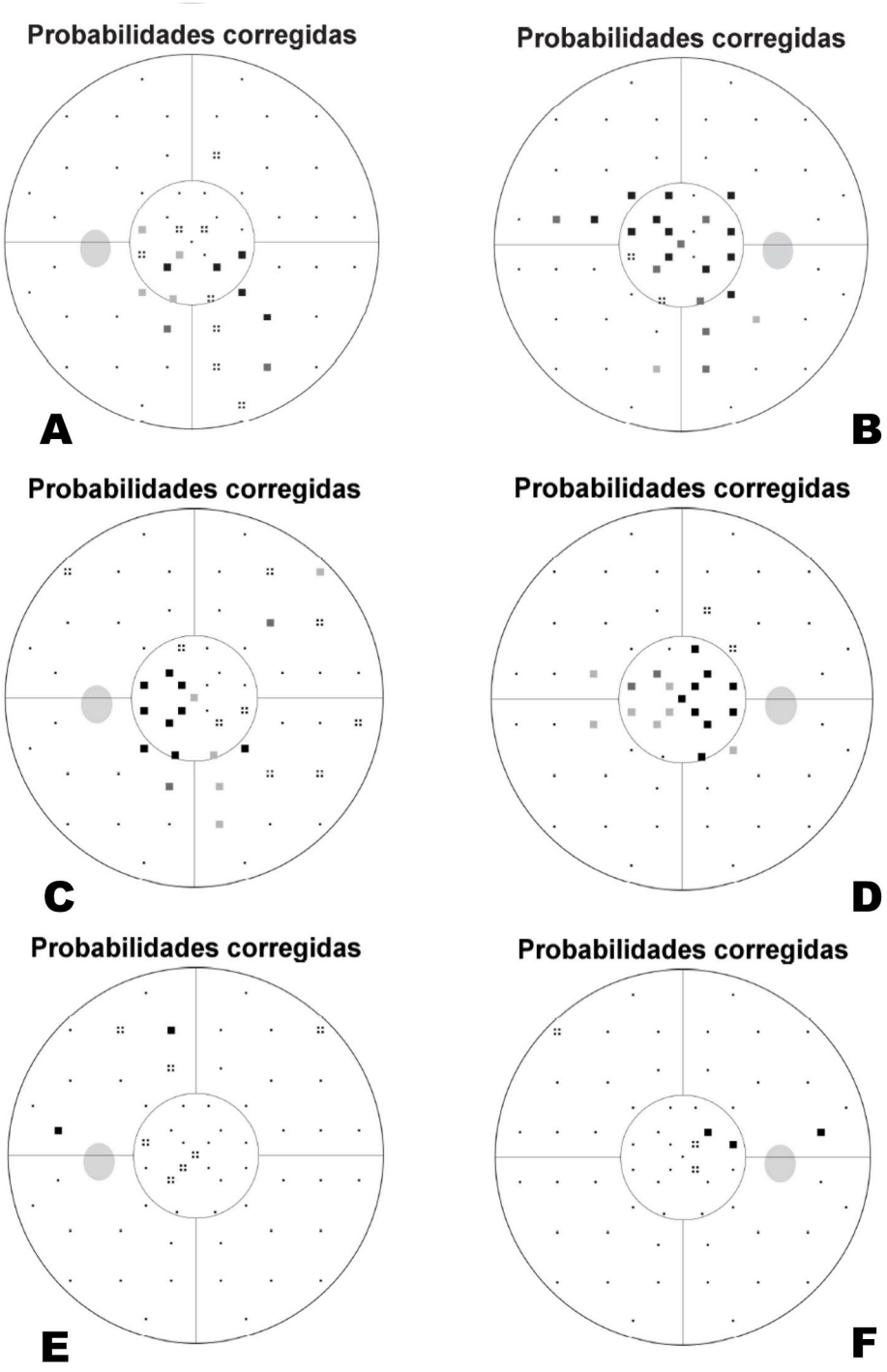

Fig. 4. Perimetry exams. A and B. Visual field test (VFT) for left eye (OS) and right eye (OD), respectively, performed after one week, detected central scotomas with a mean deviation (MD) of 4.1 dB OS and 5.7 dB OD. Low reliability (33% false negatives and 33% false positives OU) was attributed to poor 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.

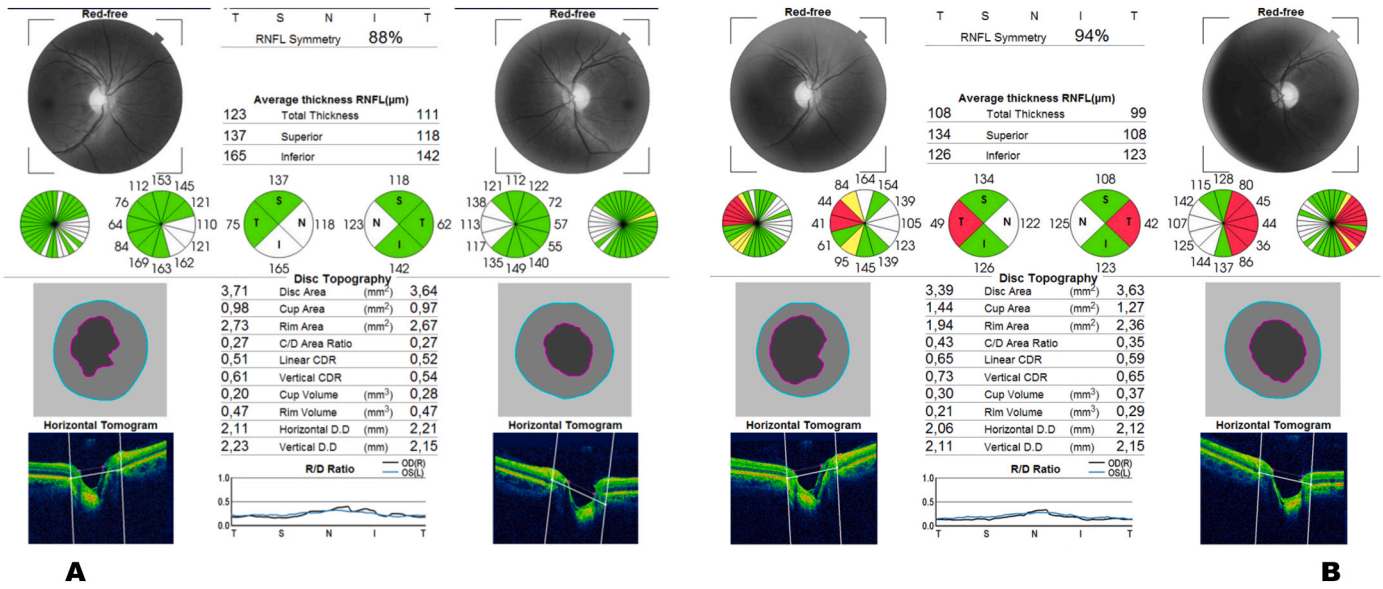

Fig. 5. Optic nerve OCT. A. Retinal nerve fiber layer (RNFL) thickness in range after three weeks of follow-up. B. Progressive loss of bitemporal peripapillary RNFL thickness after three months of follow-up.

Funding

No funding or grant support has been obtained for this work.

Authorship

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

Declaration of competing interest

The following authors have no financial disclosures: B–S, R. and S–P, ML.

Acknowledgements

None.

References

1. Grzybowski A, Zülsdorff M, Wilhelm H, Tonagel F. Toxic optic neuropathies: an updated review. *Acta Ophthalmol*. 2015;93(5):402–410.
2. Siemerink MJ, Freling NJM, Saeed P. Chronic orbital inflammatory disease and optic neuropathy associated with long-term intranasal cocaine abuse: 2 cases and literature review. *Orbit*. 2017;36(5):350–355. https://doi.org/10.1080/01676830.2017.1337178. Available from:.
3. Bahaya Alvarez Y, Acosta Acosta B, Rodríguez Gil R, Álvarez Marín J, Abreu Reyes P. Edema y hemorragia peripapilar tras consumo de cocaína. *Arch la Soc Canar Oftalmol*. 2008;19:68–71.