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A case of branch retinal vein and artery occlusion following intranasal cocaine abuse

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ABSTRACT

The known ocular effects of cocaine abuse include nystagmus and retinal vascular occlusions, among others. Retinal vascular occlusions are rarely seen in young adults and are thus generally associated with hypercoagulable disorders. Increasing rates of cocaine abuse in Western countries have led to increased reports of vascular complications. In addition to common vascular complications of myocardial infarction and stroke, cocaine-induced vasospasm can manifest in visual impairment. Previously, only two case reports of central retinal artery occlusion have been reported in intranasal cocaine abusers. This case report details combined branch retinal artery occlusion and branch retinal vein occlusion in one eye of an intranasal cocaine abuser. A 40-year-old man presented to our clinic with sudden painless nasal field loss of his left eye. The patient reported a recent history of intranasal cocaine use. Fundus examination showed intraretinal hemorrhage in the superior papillary, superior retina, temporal macula, and retinal thickening. We present a case of a patient with unilateral branch retinal vein and artery occlusions, possibly associated with induction of vascular spasm following cocaine abuse.

KEY WORDS: cocaine abuse; retinal vascular occlusion; imaging; retina

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INTRODUCTION

Retinal vascular occlusion is the blockage of retinal arteries or veins that carry blood to or away from the retina. Blockage of the vessels can lead to hemorrhages or leakages, which may compromise eyesight. Since retinal vein occlusions occur when the venous flow is obstructed, they are more likely to occur in older patients and patients with diabetes, hypertension, glaucoma, or other preexisting conditions [1]. As such, retinal vascular occlusions are rarely seen in young adults. A recent rise in cocaine use increases the likelihood of reports of vascular complications due to cocaine abuse [2]. Cocaine use causes vasospasm, which leads to vascular complications such as myocardial infarction, stroke, and cardiomyopathy, and can also manifest in unilateral visual impairment. Here, we report a case in which intranasal cocaine abuse led to branch retinal artery occlusion (BRAO) and branch retinal vein occlusion (BRVO) in one eye that manifested in nasal field loss in his left eye.

CASE PRESENTATION

A 40-year-old man presented to the hospital with a 2-week history of sudden painless vision loss

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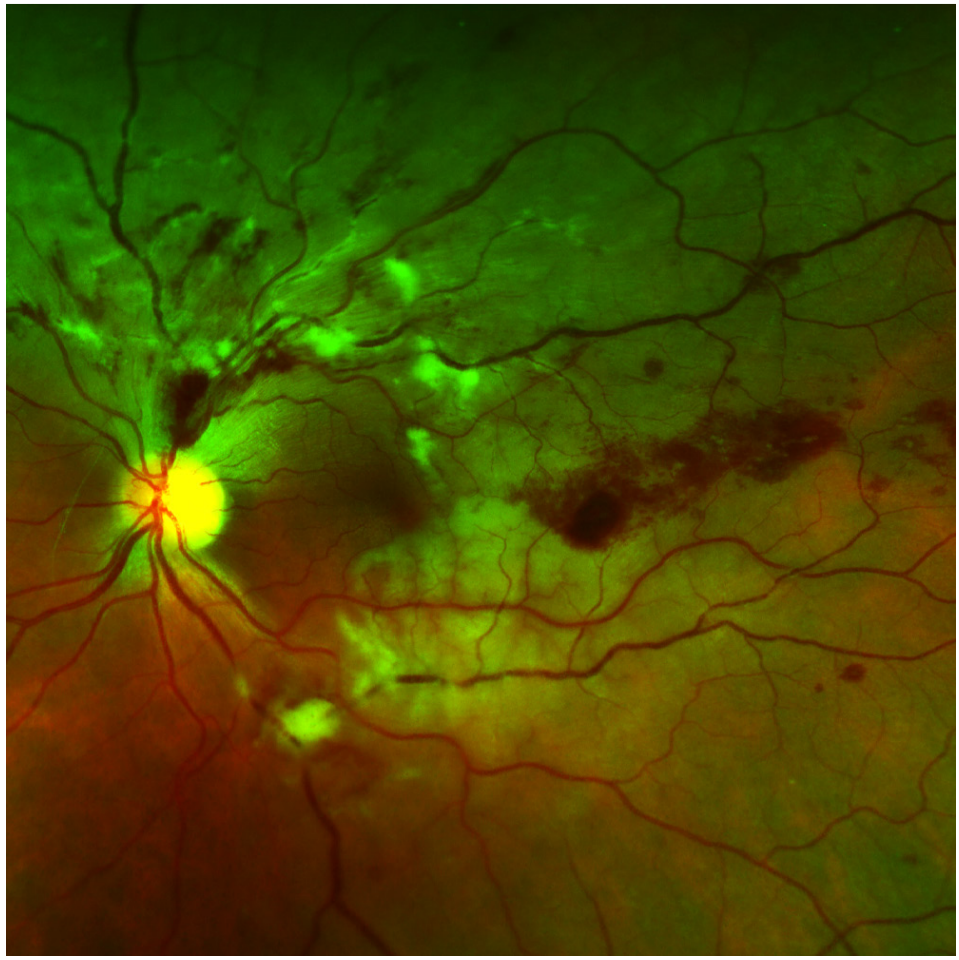

FIGURE 1. Fundus photography of the left eye showing cystoid macular edema, retinal thickening, branch retinal artery occlusions, branch retinal vein occlusions, and retinal hemorrhage

from the nasal visual field of his left eye. The patient was not using any medications and denied any previous medical conditions. He was a light tobacco smoker and used marijuana and cocaine. The patient reported that his visual symptoms started after a recent bout of cocaine use. Visual acuity in the left eye was 20/40 on the temporal visual field, but he was unable to count fingers on the nasal visual field of his left eye. Visual acuity in the right eye was 20/20. There was a chalazion on the left eye. Intraocular pressure of both eyes was within normal limits. Examination of the anterior segments of both eyes was unremarkable.

Examination of the left fundus showed beaded and enlarged venules superiorly and inferiorly (Fig. 1). The left macula had temporal retinal whitening along the superior and inferior arcade. There was also retinal sparing along the left cilioretinal artery. There were dot blot hemorrhages in the near superior arcade and temporal macular and cotton wool spots near the arteriovenous junction of the superior and inferior arcades. The left periphery showed a temporal hemorrhage and inferior vitreous opacities but there were no retinal tears or detachments. The right fundus showed retinal pigment epithelium changes in the near periphery but was otherwise within normal limits.

Fluorescein angiography (FA) showed delayed transit in the temporal arterioles/venules (Fig. 2). There was also vessel staining/leakage and a hyperfluorescent optic disc. FA of the right eye was within normal limits.

Optical coherence tomography (OCT) of the left macula showed temporal parafoveal inner retina thickening and possible cystoid macular edema (Fig. 3).

Immediate treatment constituted 1.25 mg of intravenous bevacizumab given the cystoid macula edema. He was advised that his visual prognosis may be guarded given the ischemia that involved his

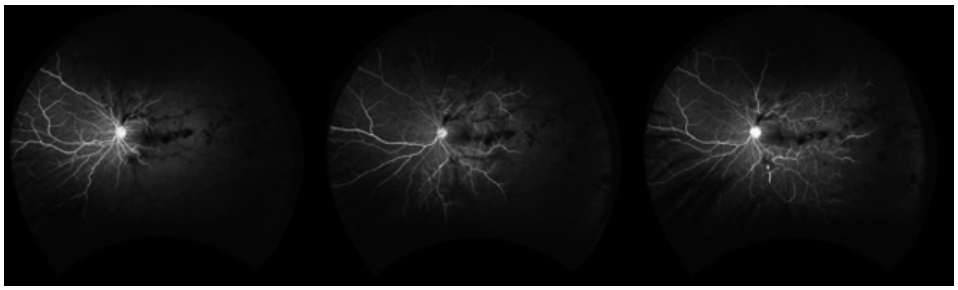

FIGURE 2. **A.** Fluorescein angiography of the left eye in 20 seconds showed delayed transit in the temporal arterioles/venules; **B.** Fluorescein angiography of the left eye in 30 seconds showing gradual perfusion of the temporal vasculature; **C.** Fluorescein angiography of the left eye in 1 minute showing hyperfluorescence/leakage of the optic nerve, vessel staining, and persistent non-perfusion of the temporal periphery

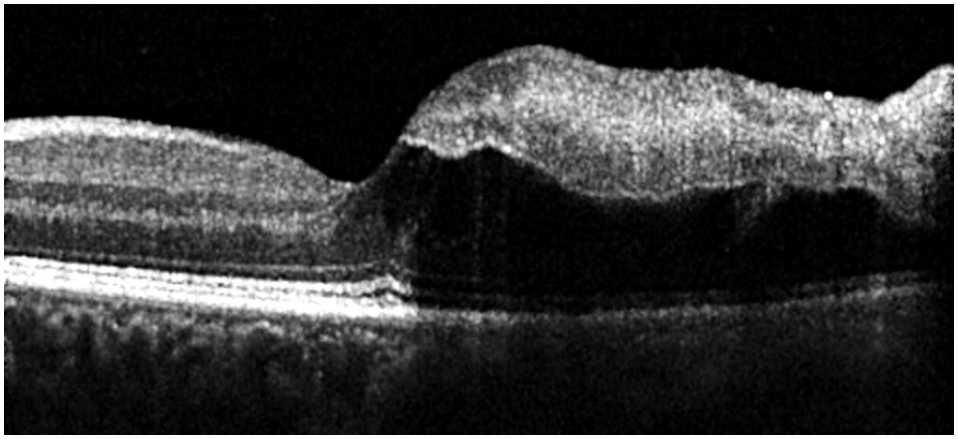

FIGURE 3. Optical coherence tomography of the left eye showing temporal parafoveal inner retina thickening and cystoid macular edema

central macula and severity. He was advised to obtain a hypercoagulable and autoimmune work-up to ensure there were no other potential etiologies for his presentation, but the patient refused. He was advised drug cessation as well. He did not present for a follow-up visit.

DISCUSSION/CONCLUSION

This case report of combined BRAO and BRVO associated with intranasal cocaine abuse in a previously healthy young man has not been seen in existing literature describing central vascular occlusion [3, 4]. Cocaine stimulates the central nervous system by blocking dopamine and serotonin reuptake at synapses and also causes a release of epinephrine and norepinephrine from the adrenal medulla into the blood. The epinephrine and norepinephrine release mimics a sympathetic nervous system response, making cocaine a potent sympathomimetic drug that induces vasoconstriction and increases blood pressure. Cocaine use leads to a multitude of vascular complications in a dose-dependent fashion, including significantly reduced coronary diameter, decreased myocardial oxygen supply, and increased risk of acute myocardial infarction [5–7].

BRAO and BRVO are typically present in older populations, with risk factors such as hypertension, atherosclerosis, coronary artery disease, diabetes mellitus, and smoking [8]. BRAO and BRVO in younger patients are extremely rare and are more likely to be associated with trauma and hypercoagulable states that lead to thrombosis [9]. The resultant vasospasm from cocaine use may explain the presence of arterial and venous occlusions, specifically in younger patients.

Cocaine has been established to induce vascular spasms, which may account for the decrease in ocular perfusion seen in ischemic damage to the retina. Hypercoagulable states and other systemic conditions should be evaluated in young patients pre-

senting with vascular occlusions to assess the risk of developing thrombosis.

This case demonstrates the effects of cocaine on the vasculature of the eye and potential consequent visual impairment, sometimes permanently.

Overall, visual impairment should be included in lists of ischemic complications of cocaine abuse, and cocaine abuse may serve as an explanation for retinal vascular occlusions in young adults in the absence of hypercoagulable states.

Statement of ethics

For solely reporting of cases, ethical approval was not required in accordance with local/national guidelines. Written informed consent was obtained from the patient for the publication of relevant medical data and any accompanying images.

Conflict of interest

The authors have no conflicts of interest to declare.

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Author contributions

Both authors, I.Z. and Y.J. have made a significant contribution to this paper. Y.J. has seen and treated the patient that was mentioned in this report. I.Z. wrote the initial draft, which was expanded upon by Y.J.

Data availability

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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