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Hypoperfusion of the deep capillary plexus associated with acute on chronic cocaine use

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ABSTRACT

Purpose: To illustrate a case of decreased perfusion in the deep capillary plexus seen on ocular coherence tomography angiography in a patient with acute on chronic cocaine use.

Observations: A 69-year-old male who presented with a sudden loss of vision in his right eye following recent use of cocaine. Ocular coherence tomography of the right eye revealed hyperreflectivity within layers of the retina and ocular coherence tomography angiography showed decreased vascular density and flow in the deep capillary plexus.

Conclusions and importance: Cocaine use has been associated with systemic and cardiac effects, as well as ocular sequelae. It has been hypothesized to have a role in the pathogenesis of acute macular neuroretinopathy. Here we present the first case of ocular coherence tomography angiography findings of hypoperfusion of the deep capillary plexus in a patient with acute on chronic cocaine use.

1. Introduction

Recreational drug use is exceedingly common with 28.6 million Americans having reported illicit drug use in the preceding 30 days according to the National Survey on Drug Use and Health in 2016.[1] This survey also demonstrated that 1.9 million individuals admitted to being current cocaine users.[1] Cocaine is well described in the cardiac literature for its effects on vasoconstriction and thrombophilic effects precipitating myocardial ischemia.[2] Cocaine use has been associated with severe ocular sequelae including acute angle closure glaucoma,[3] central retinal artery occlusion,[4,5] and retinal hemorrhage.[6] To the authors' knowledge, there has only been one report of two cases in the literature demonstrating spectral domain – ocular coherence tomography (OCT) findings of acute macular neuroretinopathy (AMN) associated with cocaine use.[7] We report the first case of OCT angiography (OCT-A) demonstrating acute deep capillary plexus (DCP) hypoperfusion in the setting of chronic cocaine vasculopathy.

1.1. Case report

A 69-year-old male presented to ophthalmology urgent care with a complaint of sudden onset vision loss in his right eye two days prior to presentation. He reported the loss of vision as seeing a "jigsaw puzzle with pieces missing" that lasted 36–48 hours followed by "a grey donut" in the center of his right eye. His medical history was notable for hypertension, diabetes, hyperlipidemia, renal artery stenosis, and silent lacunar infarcts. He also endorsed a 40-pack-year smoking history and frequent cocaine use for "multiple decades" with most recent use 1 week prior to presentation. His ophthalmic history was notable for a non-ischemic central retinal vein occlusion (CRVO) in his right eye in 2014 with recovery of vision to 20/20.

On initial presentation, visual acuity (VA) was counting fingers at five feet in the right eye and 20/30 + 2 in the left eye. The anterior segment exam was notable only for nuclear sclerotic cataracts in both eyes. Fundus exam of the right eye revealed a few dot blot hemorrhages within the macula and a mild epiretinal membrane (ERM). The arteries were attenuated with venous engorgement similar to comparison photographs from prior CRVO. The left eye revealed arteriolar attenuation. OCT of the right eye demonstrated hyperreflectivity within the outer plexiform layer (OPL), outer nuclear layer (ONL), external limiting membrane, and ellipsoid zone (EZ) surrounding the fovea (Fig. 1). An ERM with a pseudohole configuration was also noted. OCT of the left revealed a partially detached hyaloid and trace ERM.

Upon evaluation by the retina service five days later, VA had improved to 20/40 in the right eye, and the fundus exam remained unchanged. OCT was notable for improvement in the hyperreflective

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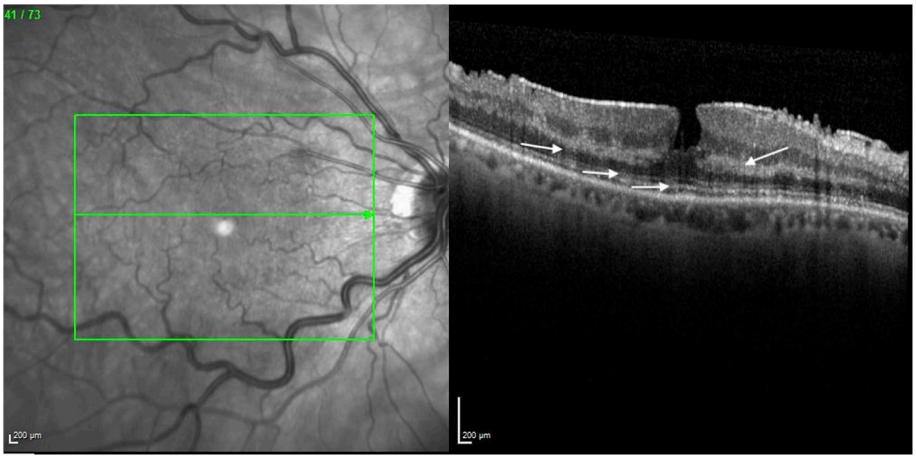

Fig. 1. Ocular coherence tomography (OCT) demonstrating hyperreflectivity within the outer plexiform layer (OPL), outer nuclear layer (ONL), external limiting membrane, and ellipsoid zone (EZ).

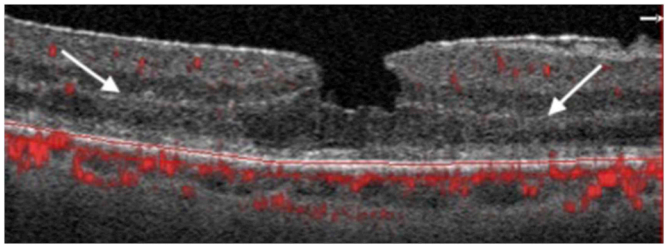

Fig. 2. Ocular coherence tomography angiography (OCT-A) of the right eye at presentation revealing hypoperfusion in the deep capillary plexus (DCP).

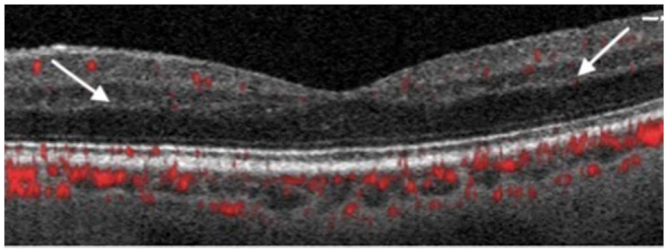

Fig. 3. OCT-A of the left eye at presentation revealing hypoperfusion in the DCP.

changes in the OPL. OCT-A of the right eye showed vascular remodeling of the superficial vessels and decreased vascular density and flow in the DCP (Fig. 2). OCT-A of the left eye also showed some hypoperfusion in the DCP (Fig. 3). Attenuation of the DCP was noted on the en-face OCT-A for both eyes (Fig. 4). Fluorescein angiography (FA) revealed a mild delay in arterial filling time, delayed transit times, and vascular remodeling consistent with prior CRVO. The patient was referred urgently to his established stroke neurologist for systemic evaluation and treatment.

At 1-month follow-up, VA was 20/25–2 in the right eye, with resolution of the intraretinal hemorrhages on fundus exam. OCT of the right eye revealed continued decrease of hyperreflectivity in the OPL with some attenuation of the ellipsoid zone (EZ) (Fig. 5). OCT-A showed improved flow in the DCP in both eyes (Fig. 5). Computed tomography angiography ordered by neurologist showed chronic lacunar infarcts but no acute cerebrovascular accident and no significant carotid stenosis was identified.

2. Discussion

Initially reported by Bos and Deutman in 1975, AMN is an uncommon disease, affecting one or both eyes, typified by paracentral scotomata which tend to correspond with flat, dark, intraretinal wedge shaped lesions whose apex typically points towards the fovea.[8] The etiology of AMN is hypothesized to be attributable to DCP ischemia.[9] Post-partum hypotension,[10] oral contraceptives,[8] flu-like syndrome,[8] epinephrine,[11] and others have been implicated as potential etiologic agents. Classic multimodal imaging findings include hyperreflectivity of the ONL and OPL, thinning of the ONL, and disruption of the EZ on OCT, and dark sharply demarcated lesions on near-infrared reflectance.[12]

As a sympathomimetic, cocaine has been associated with physiologic effects such as increased heart rate and increased systolic blood pressure when administered intranasally or intravenously.[13,14] Cocaine has been associated with numerous clinical conditions including aortic dissection,[15] myocardial infarction,[16] cardiomyopathy,[17] and prothrombotic states.[18] Ocular involvement is primarily due to ischemia from vasoconstriction induced by cocaine. Retinal artery occlusion,[4] talc retinopathy,[19] and AMN[7] secondary to cocaine use have been reported. Retinal artery spasm[20] and decreased perfusion in the DCP[7] following cocaine use have been theorized to potentially contribute to the pathogenesis of AMN.

While our case does not show the classic wedge shaped lesion on near-infrared, it does demonstrate hyperreflective changes within the ONL and OPL with subsequent thinning of the ONL and disruption of the EZ consistent with AMN.[21] As relatively few cases of cocaine related AMN have been reported, this case is notable for substantial recovery of vision over a brief period of time.[7] The attenuation of DCP on the en-face view and decreased flow on b-scan OCT-A lend support to the effects of acute on chronic insult secondary to cocaine use.

Very little is known about the chronic effects of cocaine use on the microvasculature of the retina. This case demonstrates that chronic usage may lead to the loss of complex microvascular networks over time and this loss may be further precipitated by multiple discrete episodes of acute transient ischemia. These factors are likely compounded by the patient's overall vasculopathic status and may be additive to other vasoconstrictive agents such as nicotine. Our patient's past history of CRVO in his right eye may have led to chronic vascular compromise, including change in capillary morphology and density as shown in several studies.[22,23] This could also explain the slight asymmetry in the hypoperfusion seen on b-scan OCT-A, with the right eye showing less perfusion compared to the left. Although decreased perfusion was noted in both eyes, it is possible that our patient presented with symptoms acutely in the right due to this previous history. Without this prior vascular insult, it is difficult to say if our patient would have presented differently or presented at all. Prior frequent exposures to cocaine, may have afforded this retina greater resilience in recovering from transient bouts of ischemia.

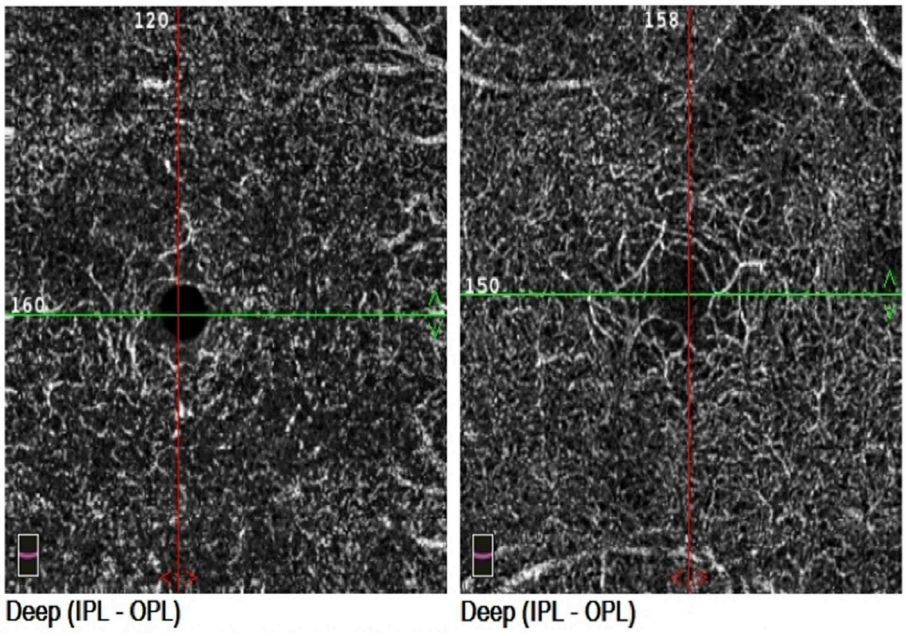

Fig. 4. Attenuated DCP on en-face OCT-A; right and left eye.

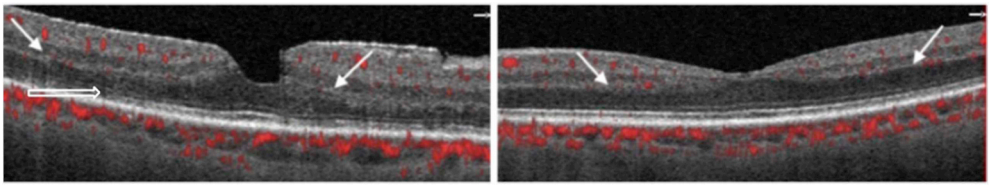

Fig. 5. OCT-A at 1-month follow-up showing improved perfusion in the DCP of the right and left eye (solid arrows) and decrease in hyperreflectivity of the OPL with some attenuation of the EZ in the right eye (open arrow).

3. Conclusions

OCT-A may prove to be an essential tool to further elucidate the complex effects of cocaine on the DCP with its intermittent periods of transient ischemia and decrease in DCP flow. Further study of the acute and long-term effects of cocaine on the microvascular physiology and the associated OCT-A findings may be warranted.

Patient consent

Consent to publish this case report was obtained from the patient in writing.

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