Presumed Optic Neuropathy Secondary to Intranasal Cocaine Use: A Case Report

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Abstract

Ocular side effects of intranasal cocaine use are rare, but can include corneal epithelial changes, retinal vascular complications, and orbital inflammation. This case report describes a 54-year-old Black female who was a habitual cocaine user and presented with acute, painless vision loss of the right eye and right optic disc edema. Cocaine's sympathomimetic properties can lead to optic neuropathy; therefore, a patient's social history of recreational drug use should not be dismissed when considering potential etiologies.

KEY WORDS
Cocaine, optic neuropathy, optic nerve head edema, intranasal inhalation

INTRODUCTION

The 2021 National Survey on Drug Use and Health reported that 4.8 million people aged 12 or older in the United States had used cocaine in the previous 12 months.[1] One of the most common routes of cocaine administration is intranasal. While systemic and ophthalmic complications related to midline destruction from intranasal cocaine use have been widely studied, optic neuropathy is rarely mentioned.[2] The presence of optic nerve head edema with associated unilateral blurred vision and visual field defects indicates the presence of optic neuropathy. A differential diagnosis can be extensive and include inflammatory, ischemic, toxic, traumatic, and compressive causes.[3] This case report describes a patient with vision loss in the right eye (OD), several hours after ipsilateral intranasal cocaine insufflation, and her subsequent diagnosis of optic neuropathy.

CASE REPORT

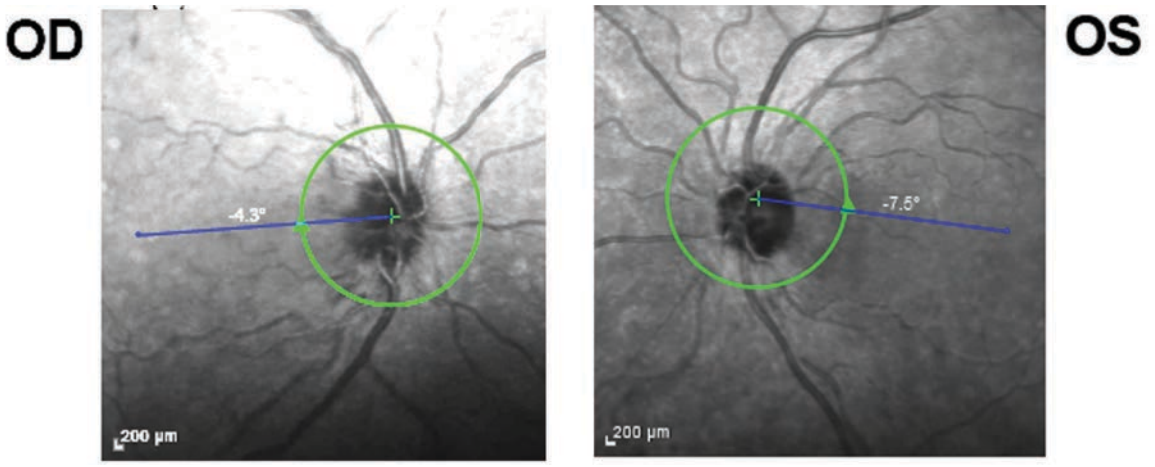
A 54-year-old Black female presented to the Veterans Affairs (VA) optometry service as a triage patient with a chief concern of central blurry vision OD and associated headache starting four days prior. She denied amaurosis fugax, scalp tenderness, jaw claudication, weight loss, or fever. Her ocular history included low myopia in both eyes (OU) and dry eyes that were managed with over-the-counter artificial tears. Her medical history included rhinitis, gastroesophageal reflux disease, and chronic back pain, which were treated with azelastine, pantoprazole, prazosin, baclofen, and gabapentin, respectively. She had a history of habitual cocaine use since 2004. Her last eye exam was one month prior at the VA optometry service, where she was documented to have a best corrected visual acuity (BCVA) of 20/20 in her right and left eyes (OS) and an unremarkable ocular health exam.

Her BCVA decreased to 20/60 OD and remained 20/20 OS at the subsequent triage exam. Extraocular motility and confrontation vision fields were full, and pupils were equal, round, and reactive to light with no obvious relative afferent pupillary defect (RAPD).

Anterior segment exam was unremarkable. Intraocular pressures by Goldmann applanation tonometry were 15 mmHg OD and 15 mmHg OS. Dilated

fundus examination revealed a 0.25 cup-to-disc ratio with an inferior disc hemorrhage and 2+ disc edema OD, based on the Frisen scale (Figure 1). A 0.25 cup-to-disc ratio with a flat and distinct disc margin was observed OS. All other structures were unremarkable OU.

Figure 1: *Black and white images of the optic nerves at the initial encounter.*



Spectral domain optical coherence tomography (SD-OCT) of the retinal nerve fiber layer (RNFL) demonstrated thickening of the RNFL OD, especially in the temporal quadrant (Figure 2). SD-OCT RNFL OS was within normal limits. Macular SD-OCT was unremarkable with normal central thickness OU. A 10-2 Humphrey visual field (HVF) test was initially ordered due to the patient's visual complaint of "central blur" and revealed an inferior altitudinal and superior paracentral defect OD with no abnormalities OS (Figure 3).

Her blood pressure was 108/71 mmHg, which was measured at the right arm, sitting. Lab testing, including erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), rapid plasma reagin (RPR), micro-hemagglutination treponema pallidum (MHA-TP), QuantiFERON-TB gold, angiotensin-converting enzyme (ACE), Bartonella, and drug analysis, was ordered. Magnetic resonance imaging (MRI) of the brain and orbits, magnetic resonance venography (MRV) of the brain, and chest X-ray were also performed. All lab results were unremarkable, except that C-reactive protein (CRP) was slightly elevated at 0.62 mg/dL (a range of 0.3-1.0mg/dL indicates minor elevation), suggestive of an acute inflammatory or infectious process.[4] The drug urinalysis was positive for cocaine; therefore, the patient was questioned about any contributing factors or recent lifestyle changes that may have been helpful in determining a cause. The patient acknowledged intranasal cocaine use several hours prior to the episode of vision loss.

The patient was admitted to the hospital for a neurology assessment. Lab work-up results for infectious etiologies (CRP, RPR, MHA-TP, QuantiFERON-TB gold, ACE, and Bartonella) were all negative. MRI of the brain and orbits, MRV of the brain, and chest X-ray were unremarkable. Lumbar puncture was aborted due to the patient's intolerance to the procedure.

The patient was re-evaluated two weeks after hospital discharge. Visual acuity was 20/50 OD with a reduction of RNFL thickening (Figure 4e) and improved optic nerve head edema OD (Figure 4b). The OS remained unaffected and stable.

At the final follow-up, one month post-discharge, visual acuity was 20/40 OD, with crisp distinct optic nerve head margins and trace temporal pallor on dilated fundus examination (Figure 4c). SD-OCT RNFL demonstrated temporal thinning OD (Figure 4f) and 24-2 HVF testing revealed an inferior central scotoma OD, and full field OS (Figure 5). Since the initial encounter, the patient was actively receiving biweekly mental health therapy and reported a more constructive and positive mindset. She denied any use of alcohol, illegal substances, or any abuse of prescription drugs since the initial triage eye exam.

Figure 2: *Spectral Domain Optical Coherence Tomography (SD-OCT) of the retinal nerve fiber layer (RNFL) at the initial encounter.*

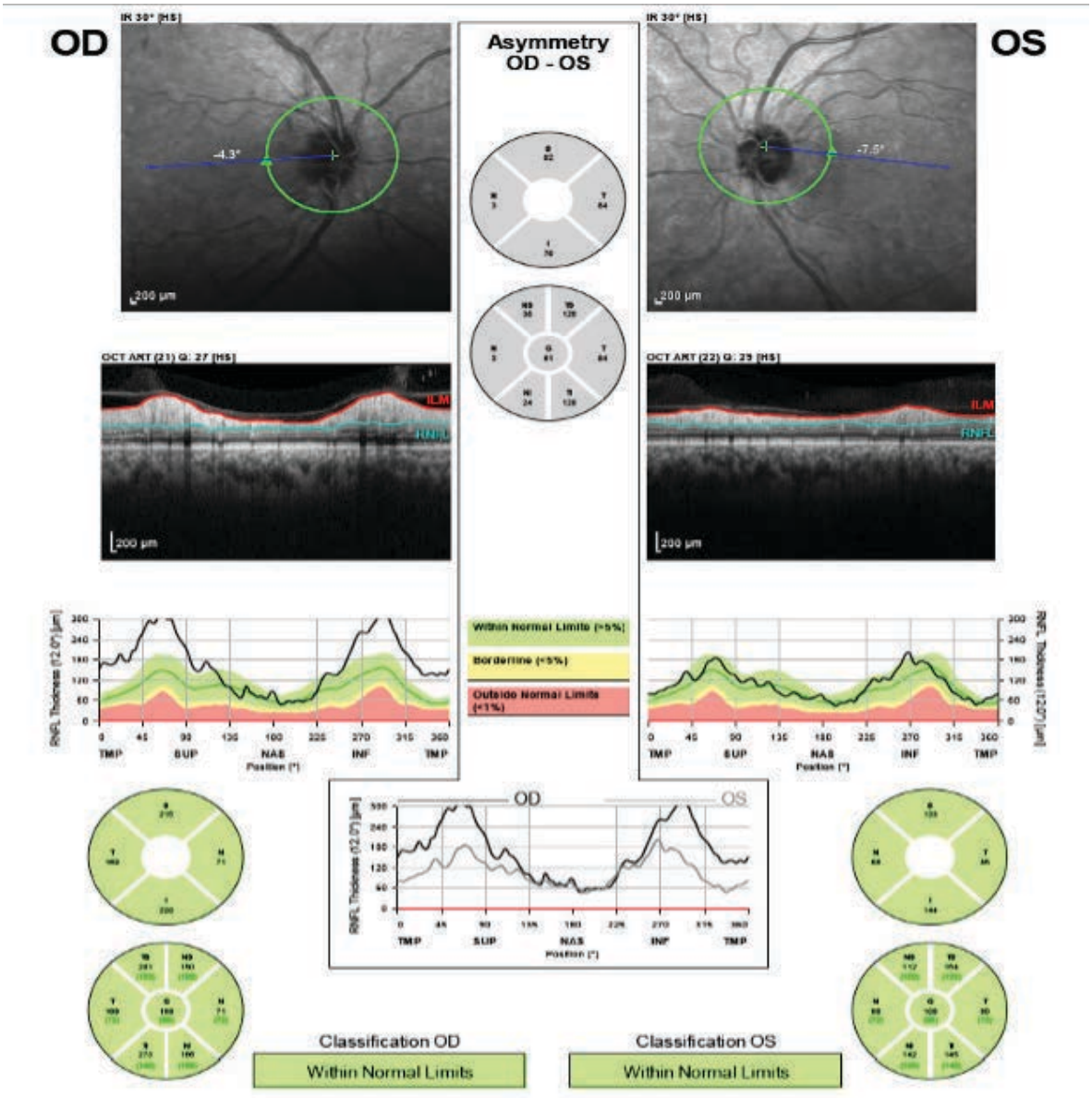


Figure 3: *Humphrey Visual Field 10-2 testing results at the initial encounter.*

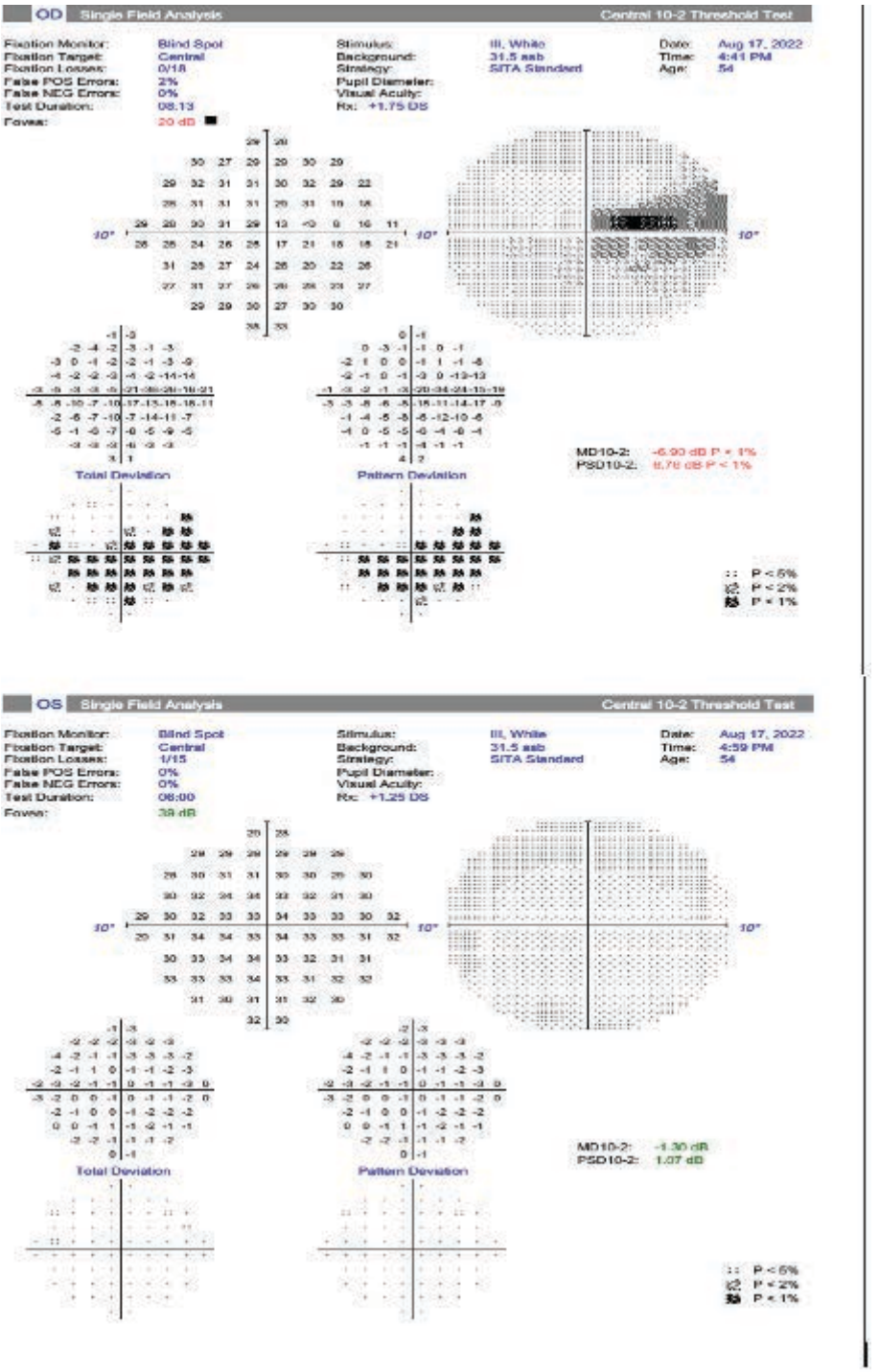


Figure 4: *Progression of SD-OCT analysis scans across patient visits. Depicted are the fundus photos of the right optic nerve head at the (a) first encounter, (b) second encounter (two weeks after the initial visit), and (c) third encounter (one month after the initial visit); and SD-OCT RNFL thickness scans of both eyes at the (d) first, (e) second, and (f) third encounters. The SD-OCT RNFL scans depict a temporal thickness of 169, 73, and 34 at the first, second, and third visits, respectively.*

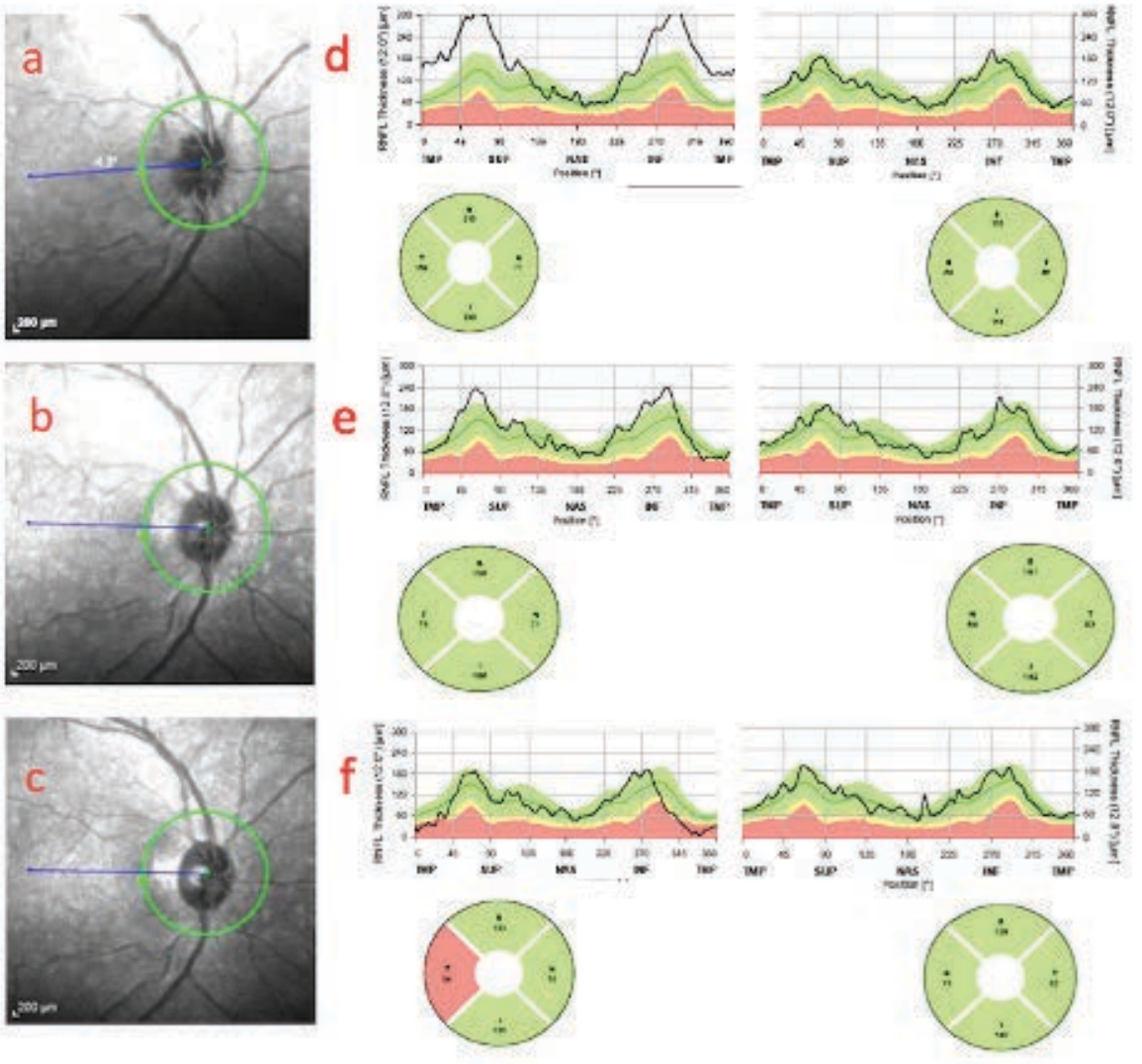
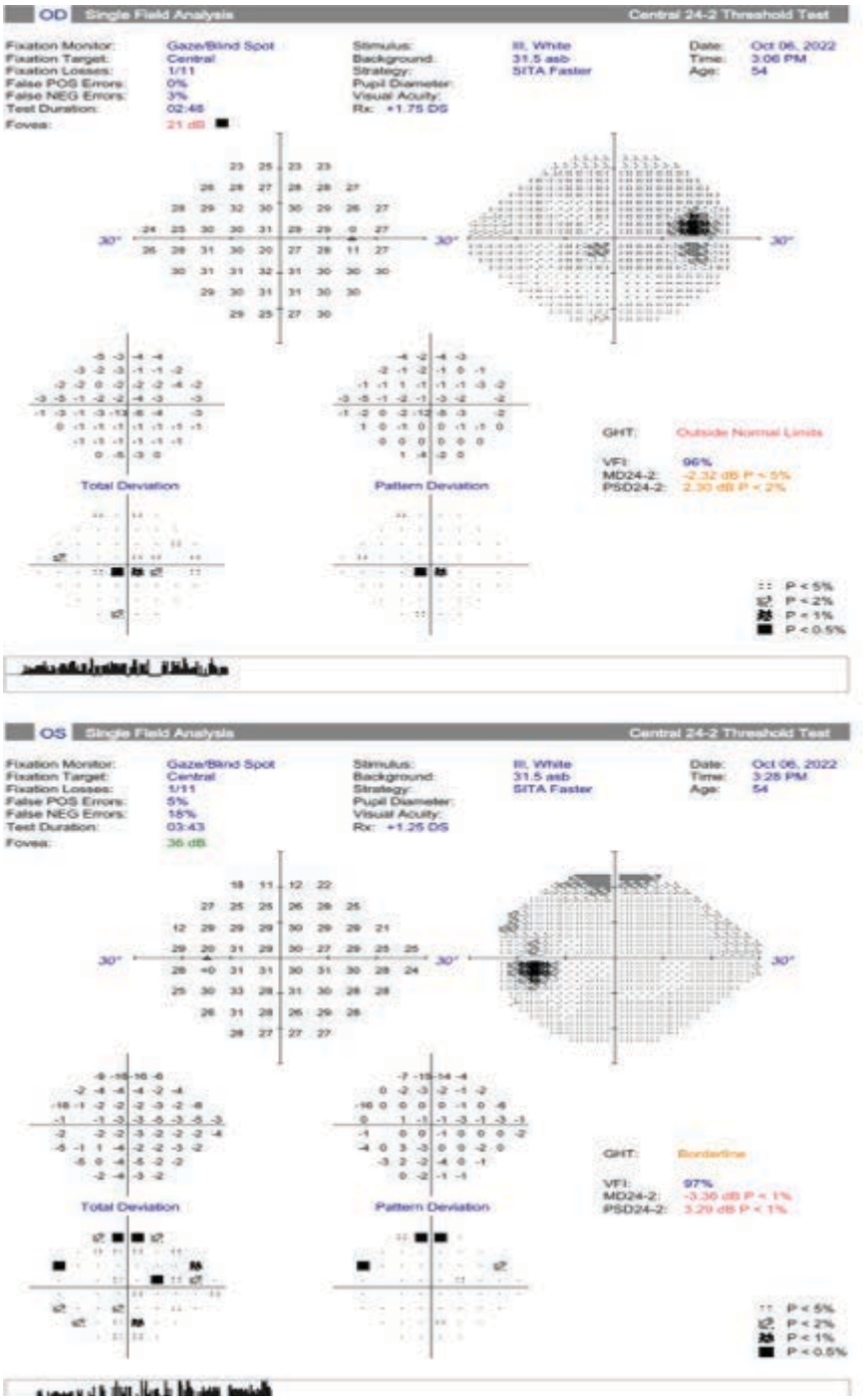


Figure 5: *Humphrey Visual Field 24-2 at the third follow-up visit, one month after the initial visit.*



DISCUSSION

Background

Ischemia of the optic nerve can involve either the anterior or posterior portion of the optic disc. Anterior ischemic optic neuropathy (AION) can be subclassified as non-arteritic (NAION) or arteritic (AAION). The most common cause of optic nerve-related acute unilateral vision loss over the age of 50 is NAION.[5] The pathophysiology of NAION is controversial; it is presumed to result from decreased circulatory function within the anterior optic nerve supplied by the short posterior ciliary arteries.[5,6]

NAION is a rare complication of intranasal cocaine use and considered a diagnosis of exclusion; therefore, demographic risk factors and etiologies such as demyelinating, inflammatory, ischemic, traumatic, pharmacological, compressive, toxic/nutritional, hereditary, and vasculopathic conditions should be investigated.[3] Common risk factors for NAION include hypertension, diabetes, hyperlipidemia, obstructive sleep apnea, and phosphodiesterase-5 inhibitor use.[5] Patients generally present with acute, unilateral vision loss, dyschromatopsia, RAPD, optic nerve edema, an inferior altitudinal or central visual field defect, and disc hemorrhage.[6]

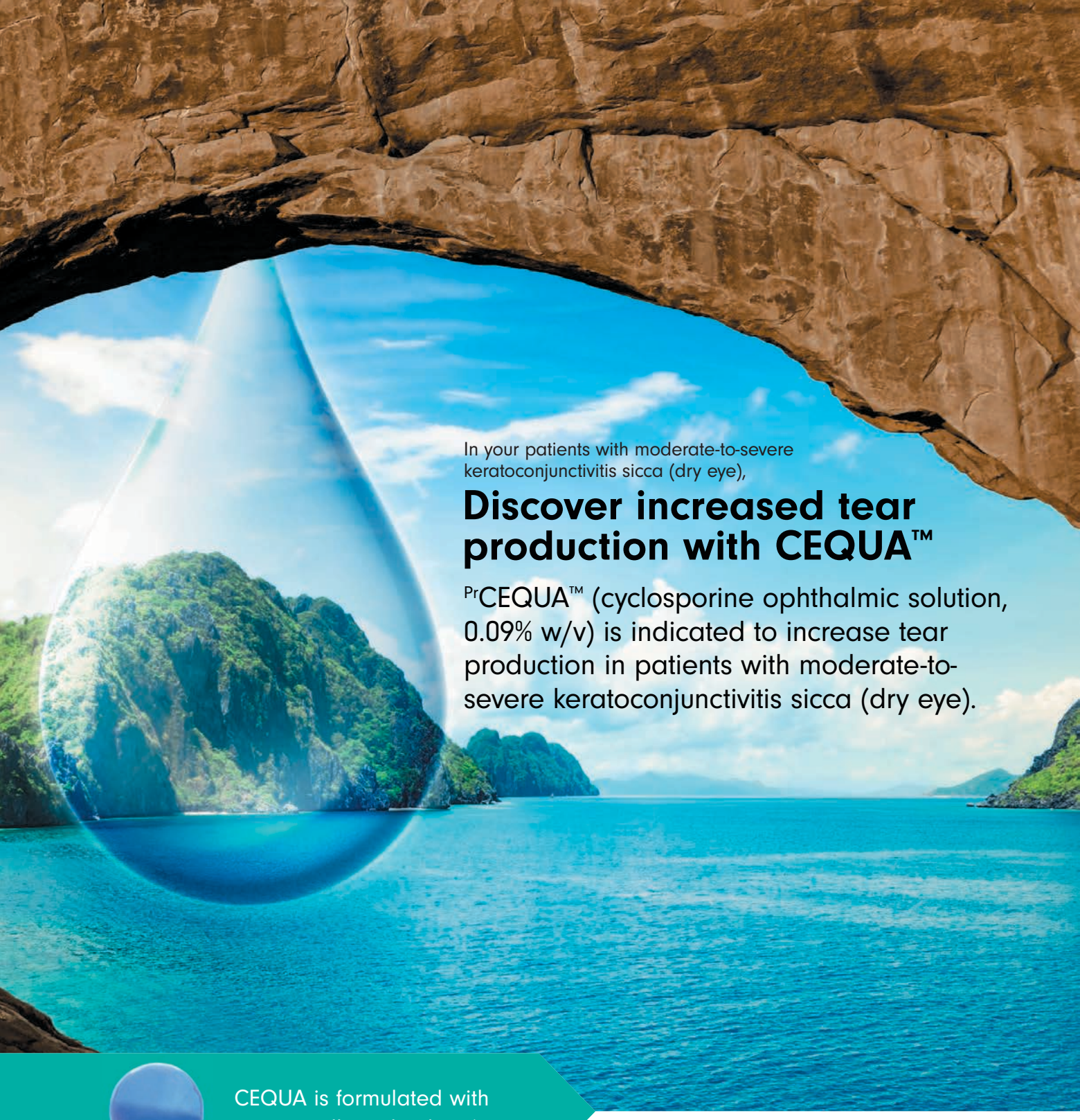
Several ophthalmic diagnostic tests can be used to help detect disease progression and monitor for improvement in NAION management. SD-OCT of the RNFL will initially show RNFL thickening, which subsequently decreases with atrophic changes.[7] Although optical coherence tomography angiography (OCT-A) was not performed in our patient's case, it can be used to discern early capillary and retinal ganglion cell changes that impact visual recovery.[8] Fluorescein angiography can detect leakage in cases of optic disc edema to differentiate it from pseudopapilledema.[9] HVF testing is also important as it is used to measure visual function and track progression while managing patients with NAION. 24-2 and 10-2 HVF are both appropriate tests in these cases. The area of complaint will usually determine the type of HVF testing the patient will undergo. The 24-2 evaluates the central 24 degrees of the visual field with testing points that are six degrees apart while the 10-2 measures the central 10 degrees, with testing points that are two degrees apart.[10] Since the 24-2 HVF has limited testing points centrally, a 10-2 HVF was more appropriate in this case given the patient's complaint of a central blur. Stereo disc images can also be helpful for following the optic nerve appearance over time.

It is critical to differentiate NAION from AAION associated with giant cell arteritis (GCA), since the latter requires time-sensitive management with high-dose corticosteroid therapy to prevent permanent vision loss. Considerations for GCA include: patients greater than 50 years of age who present with acute, unilateral vision loss, disc edema accompanied by ipsilateral scalp tenderness, and jaw claudication.[11] Immediate lab testing (ESR, CRP, and platelets) should be ordered, and a temporal artery biopsy is indicated if lab results are suspicious for GCA.[11]

Clinical case discussion

The differential diagnoses for disc edema include optic neuritis, AAION, idiopathic intracranial hypertension (IIH), compressive optic nerve tumor, and NAION.[6] Optic neuritis involves gradual, unilateral vision loss with pain on eye movement and demyelinating lesions on MRI images if associated with multiple sclerosis.[12] The patient's laboratory tests and neuroimaging ruled out potential inflammatory, infectious, compressive, and neurological risk factors for optic neuropathy. She had no risk factors, including cardiovascular disease, hypertension, or diabetes. Although our patient was unable to tolerate lumbar puncture, increased intracranial pressure is unlikely because IIH results in bilateral disc edema.[13] Furthermore, the patient's cup-to-disc ratio was greater than 0.2; thus, the nerve is less likely to be susceptible to ischemic injury from a mechanical axoplasmic flow obstruction.[6,14] Ultimately, the patient was diagnosed with presumed cocaine-induced NAION based on her comprehensive workup and the onset of symptoms several hours after cocaine inhalation.

Cocaine, a monoamine reuptake inhibitor, causes monoamines to accumulate in the synaptic cleft, resulting in enhanced vasoconstriction and subsequent impaired perfusion of surrounding structures.[15] Various intranasal cocaine-induced ophthalmic complications have been reported, such as cocaine-induced midline destructive lesion (CIMDL) and naso-orbital mass formation. Both of these conditions result in significant erosion of the nasal septum and surrounding areas, facilitating the development of preseptal cellulitis, nasolacrimal duct scarring, and other sequelae.[16,17] When managing patients who are dependent on cocaine, it is important to recognize that toxic additives can contribute to adverse effects.[18] Over the years, cocaine has been widely researched regarding its potential for inducing complications such as tremor, tachycardia, necrosis of the bone tissues of the nasal cavity, and local vasoconstriction of the nasal passage.[3,19] Although, ocular complications related to cocaine abuse are rare, side effects such as corneal epithelial damage, orbital inflammation, retinal vascular sequelae, diplopia, myokymia and optic neuropathy have all been documented.[3,20-22]



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The close proximity of the orbital cavity to the paranasal sinuses makes the optic nerve susceptible to damage from intranasal cocaine use. There are a myriad of pathways through which this damage can occur, including:

- inflammation of the paranasal sinuses, causing mechanical compression of the optic nerve vascular bundle and subsequent optic nerve head edema[3]
- infection transmission through breaks in the posterior paranasal sinuses or osteomyelitis in the nasal cavity[2]
- ischemia of surrounding tissues due to chronic sinonasal inflammatory disease[23]
- vasoconstriction of the ophthalmic artery and its branches with subsequent tissue ischemia due to cocaine's sympathomimetic effects[24]
- intra-orbital spread of inflammation through an open wall between the orbit and the nasal cavities secondary to ischemic destruction.[23]

Other cases of ophthalmic artery occlusion secondary to intranasal cocaine use have been reported previously.[25] These patients present with profound vision loss and optic disc edema, along with signs of thrombotic occlusion of the internal and external carotid arteries on computed tomography angiography (CTA).[25] Although rare, since imaging for this patient did not reveal any sinus inflammation or dehiscence in the nasal cavity, it can be speculated that the unilateral optic nerve edema may be due to local vasoconstriction of the ophthalmic artery and/or its branches as a result of cocaine's sympathomimetic effects.

The temporal RNFL thinning seen at the one-month follow-up suggests damage to the papillomacular bundle, causing central scotoma on HVF testing.[26] The clinical presentation of our patient was devoid of RAPD. Studies have suggested that superior and inferior RNFL injury may contribute the most to the degree of RAPD. Previous reports have also mentioned that, for RAPD to be present, there must be about 25% of RNFL loss in the affected eye compared to the unaffected eye.[27]

Management and prognosis

There is no proven effective therapy for NAION. Controlling known risk factors and medication reassessment is the widely accepted approach to the management of NAION.[6] It is unknown if the visual prognosis differs for intranasal cocaine versus vasculopathic-induced NAION. The visual prognosis of NAION is variable. Approximately 50% of patients with NAION have an initial visual acuity of 20/30 or better and in nearly 25% this is 20/200 or worse.[28] Acuity improvement of at least 3 lines is seen in about 13-42.7% of patients and disc edema resolution and subsequent atrophy are seen within 6-11 weeks.[29] Forty-one percent of patients with 20/70 or worse acuity showed improvement at 6 months.[28,30] Patients should be followed one month after the initial onset to monitor for disc edema resolution. Further testing is indicated if edema persists at this one-

month follow-up.[29] Patients should be educated on the 15% chance of contralateral eye involvement over 5 years and seek immediate medical attention if NAION symptoms are present.[31]

When addressing sensitive topics such as substance misuse disorders, it is imperative to use mindful non-verbal and verbal communication techniques to minimize patient anxiety and doctor-patient discord.[32] Building a respectful rapport will allow optometrists to effectively educate their patients on the risks associated with illicit drug use and thereby improve patient outcomes.

CONCLUSION

A patient's social history is important when considering the differential diagnosis for optic neuropathy. Optic neuropathy is a rare yet significant sequela of long-term cocaine abuse and should not be overlooked as a potential cause of NAION in the absence of other risk factors. Determining the underlying cause of optic neuropathy is essential for mitigating potential risk factors that may hinder the visual prognosis. Furthermore, as optometrists, we must be cognizant and considerate when broaching sensitive issues like substance abuse to increase a patient's trust, transparency, and accuracy of conveyed information. Patients should be educated on the importance of illicit drug use cessation in a non-judgmental manner. ●

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