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Case Report

Acute ischemic optic neuropathy in a case of heroin overdose ☆,☆☆

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ABSTRACT

Presence of acute optic disc and optic nerve infarction in a young man is uncommon finding. This is most commonly seen in the setting of vasculitis and infection. Ischemic optic neuropathy has been reported with cocaine use, amlodipine and alcohol use. To our knowledge there is no reported case of ischemic optic neuropathy in the setting of heroin / opioid use. MR imaging findings in the setting of substance use are similar to other etiologies of ischemic optic neuropathy, with high T2/FLAIR signal, diffusion restriction and abnormal gadolinium enhancement. Here we report a case of 23-year-old man with heroin use disorder presenting with optic nerve infarct resulting in acute painless monocular vision loss.

Background

Ischemic syndromes of the optic nerve are referred to as Ischemic optic neuropathy (ION). ION is a visually crippling disease of the middle-aged population, which results from an acute interruption of the blood flow in the ophthalmic artery or its tributaries [1–4,5]. ION produces a spectrum of degrees of blindness. ION are classified according to the location of the ischemic damage of the nerve and according to the etiological factor for the ischemia. Anterior ION (AION) involves optic nerve head and optic disc, whereas, posterior ION (PION) involves retrobulbar optic nerve [6–8].

Based on underlying etiology for ischemia, ION is classified into 2 categories: Arteritic and nonarteritic ION. Arteritic ION is secondary to blood vessel inflammation and nonarteritic results from non-inflammatory conditions. Nonarteritic ION is most often idiopathic, but systemic hypotension and radiation injury have been identified as specific etiologies. The arteritic form has a poor prognosis, compared to nonarteritic ION [3,6–8].

Abbreviations: ION, ischemic optic neuropathy; AION, anterior ischemic optic neuropathy (AION); PION, posterior ischemic optic neuropathy (PION).

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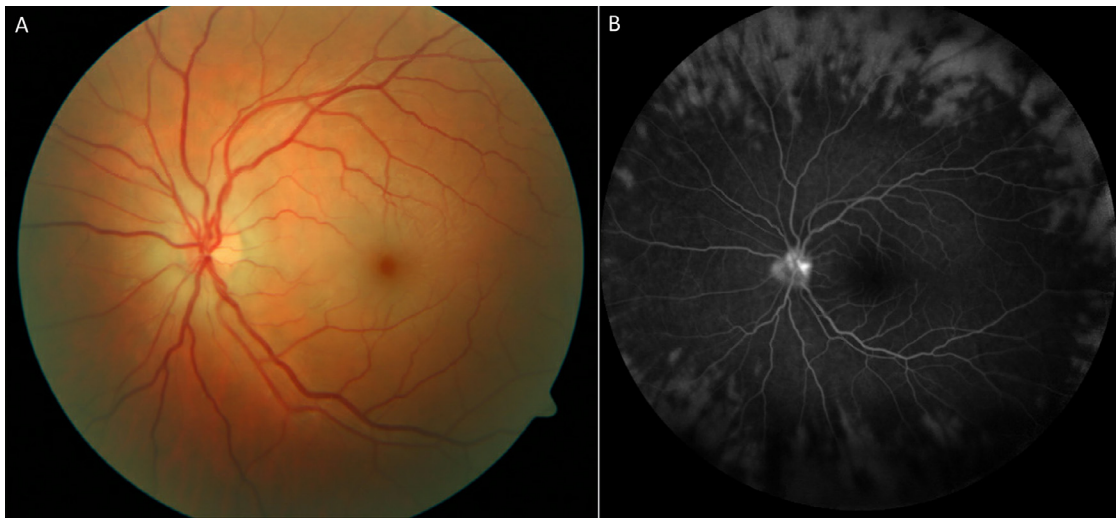

Fig. 1 – Fundus photograph (A) and the fluorescein angiograph (B) image of the left eye, demonstrates optic disc and peripapillary edema, cherry red spot in the macula with peripheral choroidal infarcts, findings compatible with central retinal artery occlusion.

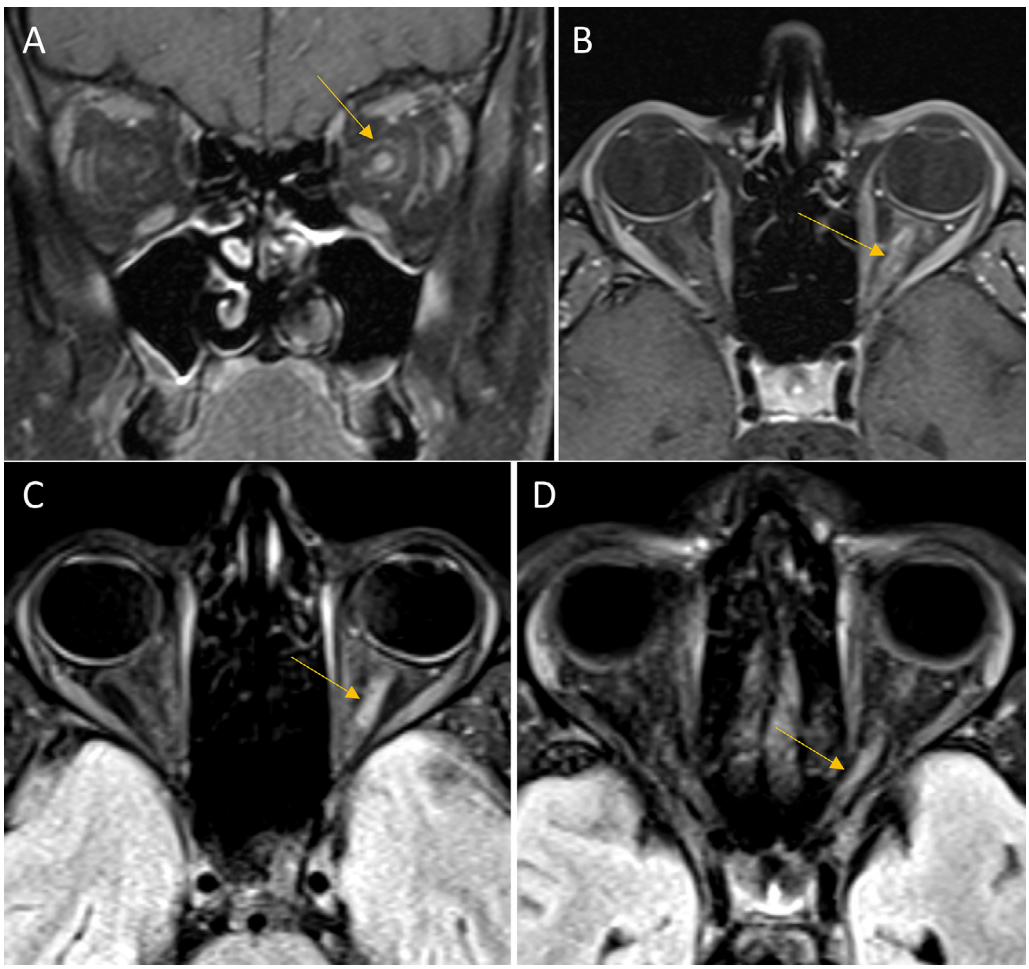

Fig. 2 – Coronal T1 fat-suppressed postcontrast (A), Axial T1 fat-suppressed postcontrast (B), and axial T2 FLAIR images (C and D). High T2 signal and abnormal nerve and nerve sheath enhancement involving the intraorbital and anterior intracanalicular segments of the left optic nerve was noted.

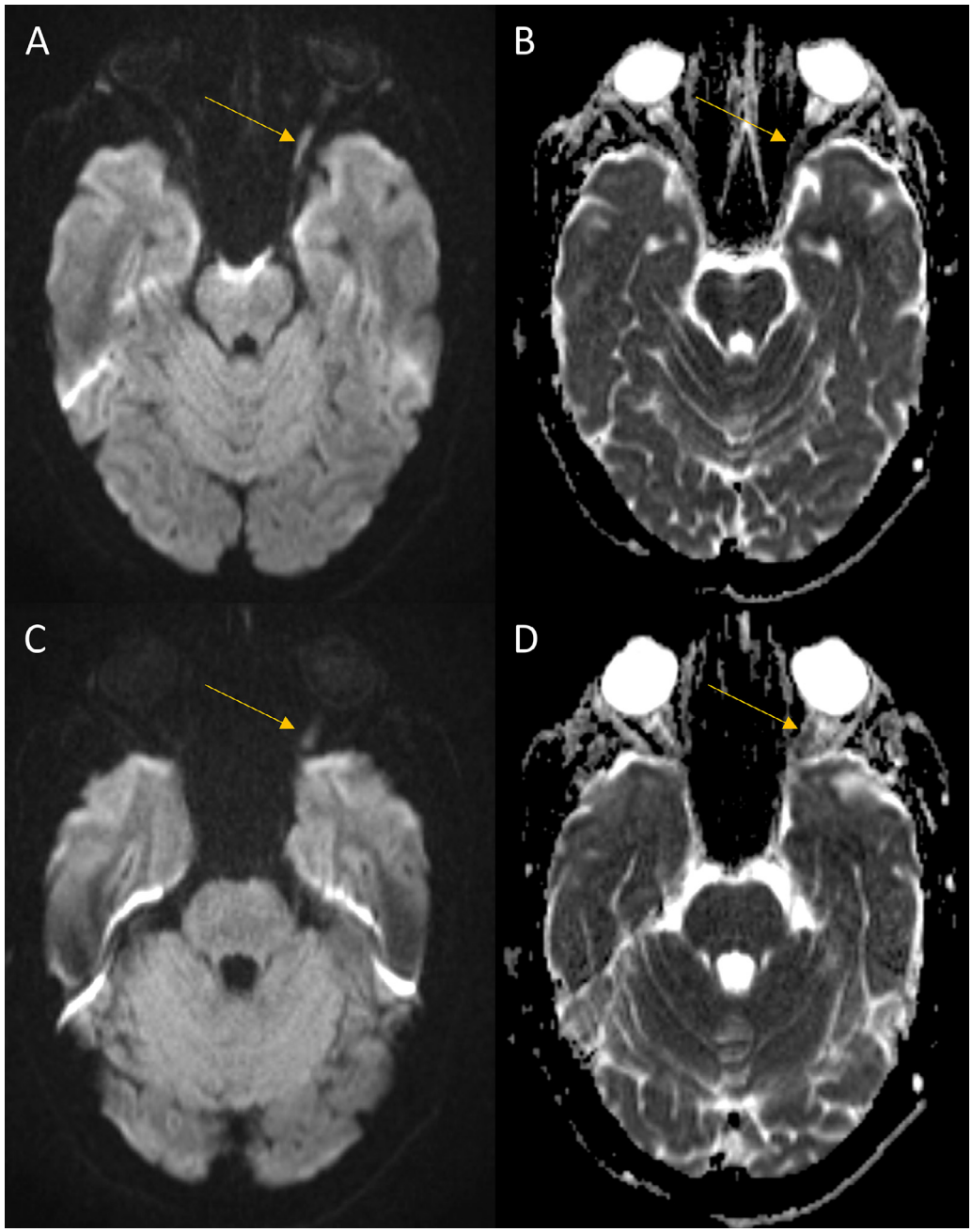

Fig. 3 – Diffusion-weighted images (DWI) (A and C) and apparent diffusion coefficient (ADC) (B and D) shows restricted water diffusion in the intraorbital and anterior intracanalicular segments of the left optic nerve corresponding to signal abnormality and enhancement seen on Fig. 2. The findings are compatible with acute infarction of the left optic nerve.

Here we describe a case of anterior and posterior optic nerve ischemia in a 23-year-old man from heroin overdose. ION has not been described in the literature with heroin or opioid overdose.

Case report

A 23-year-old man with a history of heroin use disorder (on Suboxone) presents to the emergency department after being found unresponsive and unarousable in front of his apartment complex via EMS. On arrival, pertinent examination findings included respiratory rate of 6 breaths per minute and pinpoint pupil. Resuscitation protocol was initiated and 2 mg intravenous Naloxone was administered with presumptive diagnosis of heroin overdose, which was subsequently confirmed on urine drug screen. After the regimen, he responded appropriately and after hemodynamic stability was established, he was admitted to the hospital for further workup.

The next day following stabilization, the patient developed new onset left monocular painless vision loss. He denied any

history of trauma, pain or prior episode of transient vision loss. CT brain and cervical spine without contrast along with CT chest, abdomen and pelvis were negative on admission and ruled out solid organ injury, intracranial hemorrhage or significant trauma.

The ophthalmology service was consulted for acute left vision loss. Slit lamp and Fluorescein angiographic examination (Fig. 1) was performed which showed disc and peripapillary edema, along with a cherry red spot in the macula with peripheral choroidal infarcts, compatible with central retinal artery occlusion.

MRI of the brain and orbit with and without contrast was performed for further evaluation (Figs. 2 and 3). The intraorbital and anterior intracanalicular segments of the left optic nerve showed high T2 signal, and diffusion restriction, findings compatible with acute infarction of the left optic nerve. The diseased segment of the optic nerve along with optic sheath demonstrated abnormal gadolinium enhancement (Fig. 2).

Lumbar puncture was performed and cerebrospinal fluid studies were normal. Initial blood workup was also normal. Patient was diagnosed as left AION and left PION in the setting of acute opioid overdose. Leading hypothesis for optic nerve infarction includes systemic hypotension, with drug induced vasculitis being an additional consideration. He was continued with monocular precautions and referred to institution's comprehensive opioid addiction treatment program.

Discussion

AION typically presents with rapid onset painless unilateral visual loss developing over hours to days. The optic disc is most often diffusely swollen. Estimated annual incidence of AION is 0.57 per 100,000 over age of 60, with mean age of 70 years [9,10]. Most common cause of visual loss in giant cell arteritis is arteritic AION [11–13]. Arteritic AION has female predisposition, whereas no gender discrepancy is noted in nonarteritic AION. Arteritic AION requires management of underlying vasculitis [14–16]. Nonarteritic AION encompasses majority (94.7%) of AION cases, with an estimated annual incidence of 2.3–10.2 per 100,000 population [9,17,18]. In nonarteritic AION, neuroimaging is not performed unless there is an atypical clinical course, such as prolonged optic disc edema, severe pain or continued progressive or recurrent visual loss more than 2 months after initial presentation. There is no proven effective therapy or proven prophylactic measures for nonarteritic AION [1–5].

Ischemia of retrobulbar optic nerve occurs in many settings and can independently affect each segment, due to separate arterial supply of anterior and posterior optic nerve. Anterior optic nerve is supplied by short posterior ciliary artery and choroidal circulations. Retrobulbar optic nerve depending on the location is supplied by pial plexus of ophthalmic artery (Intraorbital segment) and branches of internal carotid artery, anterior cerebral and anterior communicating arteries (Intracranial segment) [9–15]. PION is characterized by acute visual loss with characteristics of optic neuropathy, typically without initial disc edema, with subsequent development of optic nerve atrophy. PION is most often seen in the setting of: 1) Giant Cell Arteritis or rarely other vasculitis; 2) Nonarteritic PION and 3) Related to surgery (coronary artery bypass and lumbar spine procedures), or severe hypotension [19,20]. In PION neuroimaging is indicated to rule out these possibilities.

T1-weighted MR imaging, with and without gadolinium has shown utility in assessment of ischemic injury to the optic nerve, showing gadolinium enhancement in the injured nerve. Diffusion imaging is widely used to assess intracranial acute ischemia, however its application in the setting of ION is not studied [8]. Since, optic nerve is an extension of the central nervous system as is the spinal cord, the Pathophysiology of acute optic nerve ischemia is expected to be similar in the setting of cell swelling and restriction of water diffusion leading to low ADC and high DWI signal [8,19–21].

Our patient presented with findings most supportive of AION and PION in the setting of heroin overdose. Ischemic optic neuropathy has been reported in cases with methamphetamine use [22], amlodipine overdose [23], and cocaine use [24]. But there is no reported case of ION in the setting of heroin / opioid use.

Patient consent

Consent was obtained for publication of this case report. No patient identifier is either recorded or published.

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