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Clinical Findings and Management of Opiate Withdrawal Related Acute Onset Esotropia: A Case Report

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ABSTRACT

Acute onset comitant esotropia (AOCE) is a rare ophthalmic complication associated with opiate withdrawal. As the prevalence of opioid use disorder remains high, it is critical for clinicians to recognize this condition as a differential diagnosis for acute diplopia in adults, especially after ruling out emergent neurological pathologies. This case report details the clinical presentation, diagnostic workup, and management of AOCE secondary to acute heroin withdrawal in a 34-year-old male. The patient reported the sudden onset of constant, horizontal diplopia three days after stopping heroin use. An initial outside consultation three weeks after onset measured a 25 prism diopter (PD) esotropia. Upon presentation to our clinic, his deviation had reduced, and a comprehensive workup, including magnetic resonance imaging (MRI) and angiography (MRA), was performed to rule out intracranial pathology. All were found to be unremarkable. The patient's symptoms were managed with a temporary 12 PD base-out Fresnel prism, and his esotropia and diplopia spontaneously and completely resolved over the following three weeks. Opiate withdrawal-induced AOCE is theorized to result from a reversible disuse atrophy of the motor fusion system. Chronic opiate-induced miosis decreases accommodative demand; acute withdrawal causes a relative parasympatholytic state (mydriasis), increasing accommodative demand that the atrophied motor fusion system cannot meet with appropriate divergence, resulting in esotropia. This patient's history of uncorrected hyperopia and convergence excess likely predisposed him to this condition. This case highlights that acute esotropia following opiate withdrawal is a transient condition where a thorough history is paramount, allowing for conservative management and avoiding unnecessary procedures.

INTRODUCTION

Opioids are a class of naturally occurring or synthetically produced drugs used for the treatment of pain. Its use as a pain reliever has seen a decline in the past decade, with prescriptions reduced by 44% from 2011 to 2020.[1] Despite this, illicit, recreational use has increased. In 2021, there were over 80,000 overdose deaths due to opioids, accounting for over 75% of total overdose deaths.[2] As the prevalence of this addiction remains high (4.8 million people)[3], it becomes more important to be aware of and manage both the adverse effects related to the drug itself as well as withdrawal complications upon cessation.

Withdrawal symptoms of opioids are variable based on length of use, type of opioid, and amount used. Somatic symptoms can include bone or muscle pain, changes in body temperature, insomnia, nausea, vomiting, and weakness. Affective symptoms can include anxiety, irritability, malaise, and stress. A less studied and rare ophthalmic complication associated with opiate cessation is acute onset comitant esotropia (AOCE).[4] However, despite the rise of opiate illicit use, AOCE associated with opiate withdrawal continues to be an under investigated and seldomly reported condition with only 16 cases documented. Due to the limited clinical information available, the underlying pathogenetic mechanisms involved in opiate withdrawal induced AOCE remain poorly understood. Consequently, treatment for this condition continues to be unsubstantiated.

Clinical providers should be aware of and be able to recognize AOCE associated with opiate withdrawal as a differential diagnosis for acute esotropia, especially in young adults. As with any sudden onset esotropia, the concern for an intracranial pathology, some of which can be life-threatening, should be explored.[5] It is critically important to perform a thorough case history of the medical, social and illicit drug history, clinical examination and supplementary testing for drug use and withdrawal symptoms to narrow down the diagnosis and possibly avoid costly and unnecessary workups. This case further highlights the appropriate work-up for a patient who presents with AOCE related to opiate withdrawal.

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Prior History

A 34-year-old Caucasian male has been seen in the West Haven Veteran Affairs Medical Center Acquired Brain Injury (ABI) Eye Clinic for the past 6 years. During that time, his clinical exam was stable and consistent with the diagnoses of convergence excess and accommodative insufficiency. However, within the past year his lateral posture at both distance and near was now exophoric, while the accommodative insufficiency remained stable.

The patient has had a long history of substance abuse with opiates, starting with oxycodone, and most recently with heroin. He had gone through several cycles of rehabilitation before relapsing following his discharge.

Prior to his most recent visit, the patient entered into a heroin treatment and recovery center in Florida. He self-reported severe withdrawal symptoms as he declined methadone maintenance therapy during his stay. Three days following admission to the center, he reported awakening with constant diplopia at both distant and near. He reported that it resolved when one eye was closed, was horizontally oriented, and was worse at distance than at near. He declined an urgent referral for an eye exam as he was overwhelmed by the systemic withdrawal symptoms he was experiencing. Two weeks later, he agreed to have a full ocular health evaluation, which was found to be unremarkable. The ophthalmologist then referred the patient for a strabismus consultation. He was seen for the strabismus consultation one week later. A partial 9-position gaze test was completed (Figure 1) and revealed a comitant esotropia of 25 prism diopters (PD) in magnitude. It was unclear whether the testing to measure the deviation was done at distance or near, or if the esotropia was monocular or alternating. Unfortunately, no further information could be obtained from the office. As treatment, the ophthalmologist applied a 25 base out Fresnel prism OD. He reported that the Fresnel prism resolved the diplopia at that time.

CASE REPORT

The patient presented to the ABI Eye Clinic three days following his strabismus consultation. The patient reported resolution of diplopia at near with residual constant diplopia at distance. In addition, he reported discomfort with the 25 PD Fresnel prism and had removed it himself prior to the visit.

The patient's medical history was remarkable for headaches, depression, insomnia, and obstructive sleep apnea. He had a history of a TBI during deployment nine years prior. The only medication prescribed was quetiapine for his depression and insomnia. He was allergic to penicillin. Systemic family history was remarkable for hypertension (mother) and coronary artery disease (father). Ocular family history was unremarkable.

All examination findings can be found in Table 1. Testing in the 9 cardinal positions of gaze was performed with the patient wearing his fully corrected distance prescription and with the Maddox Rod over OD (Figure 2). With a 12 PD Fresnel prism applied over OD, the patient reported resolution of the diplopia in primary gaze at distance as well as all other gazes except for extreme right and left gaze. Although there did not appear to be a palsy, the sudden onset of the deviation and the new, slight inconcomitancy noted (Figure 2) made an imaging consult appropriate. The patient was recommended to return to the ABI clinic in one week.

Table 1: Examination Findings

Subjective Refraction		
Right Eye	+1.25-1.75x162	DVA: 20/20; NVA: 20/20
Left Eye	+1.25-1.50x017	DVA: 20/20; NVA: 20/20
Cover Test		
Distance	5 PD Constant Right Esotropia	
Near	10 PD Esophoria	
Extraocular Motilities (EOMs)	Full, complete range of motion (+) diplopia: extreme right, left and down gazes (-) restriction or pain	
Pupils	Equal and reactive to light (-) APD	
Confrontation Visual Fields (CVF)	Full to finger counting OD and OS	
Fixation	Stable for 10 seconds OD and OS	
Saccades	Slight undershoot in all gazes	

Magnetic resonance imaging (MRI) scans, with and without contrast, of the brain and orbits were completed. The scan was essentially unremarkable with no evidence of acute, subacute, or chronic brainstem or cerebral infarction. There was no evidence of intracranial mass or mass effect. The ventricular system was normal in size and symmetric. There was no evidence of an intra-orbital mass, and the extraocular muscles appeared normal. An MRA of the circle of Willis was unremarkable for aneurysms.

One week later, the patient presented for his follow-up examination. He reported a resolution of diplopia at distance and near in primary gaze for the past two days. He removed the 12^ Fresnel prism at that time because he no longer needed it. On examination, the subjective refraction did not change, and BCVA remained 20/20 OD and OS. Distance cover test was ortho both laterally and vertically. Near cover test revealed a 6 esophoria with no vertical deviation. EOMs remained full without restriction or pain. He reported diplopia only in extreme down-right gaze. 9 positions of gaze testing were completed and can be seen in Figure 3. Since the patient reported no diplopia and was able to easily perform his activities of daily living, no Fresnel prism was given. He was recommended to return for a follow-up exam in 2 weeks.

Two weeks later, the patient presented for his second follow-up exam. He reported a complete resolution of diplopia at distance, even in extreme gazes. BCVA remained stable at 20/20 in each eye, and EOMs remained full without restrictions, pain, or diplopia in any gaze. The distance cover test was unchanged at ortho laterally and vertically. At near, 3-4 esophoria was noted without a vertical deviation. Since the patient now had no diplopia at distance or near and had no functional limitations, the patient was discharged for 4 months with instructions to return to the clinic sooner should the diplopia recur.

Figure 1. 9 Positions of Gaze (Strabismus Consultation)

-	25 eso	-
25 eso	25 eso	25 eso
-	25 eso	-

Partial 9 positions of gaze testing completed by another eye care provider.

Figure 2. 9 Positions of Gaze (First Visit to VA)

12 eso	10 eso	12 eso
12 eso	8 eso	16 eso
20 eso	18 eso	20 eso

9 positions of gaze testing completed on initial encounter with CA at West Haven VA Eye Clinic. Note mild incomitancy.

DISCUSSION

An acute onset, symptomatic esotropia in a healthy young adult male is an important clinical finding that necessitates a thorough work-up. The first and foremost serious differential diagnosis to rule out is an extraocular muscle palsy. In the case of an esotropia without a vertical component, such as with the patient, the most likely EOM to be affected is the lateral rectus.

A lateral rectus palsy is due to either a lesion along the tract of the nerve or a microvascular etiology. Patients will usually present with an incomitant esotropic deviation that is worse on ipsilateral gaze and improves with contralateral gaze. EOM testing will show gaze palsy on the ipsilateral movement laterally. The location of a lesion can be further localized by assessing whether the palsy is occurring in isolation or with other deficits. MRI scans are standard for younger patients. For patients greater than 50 years old with vasculopathic risk factors such as diabetes or hypertension, a microvascular etiology should be considered as well. There is some debate as to whether older patients with vasculopathic risk factors should also be scanned. At a minimum, a sixth nerve palsy that has not resolved within 3 months should be scanned.[5] Unfortunately, an acute onset of esotropia with comitant deviations does not completely terminate the risk of intracranial lesions. Several conditions have been linked to AOCE, such as hydrocephalus, midbrain lesions, or cerebellar lesions.[6] A key differentiating clinical feature, although not universal, is the presence of an abduction nystagmus or other neurological abnormalities in cases with an intracranial condition.[6] As for the patient, his EOM's were full, decreasing the likelihood of a sixth nerve palsy as a diagnosis. In addition, he was in good cardiovascular health, without any vasculopathic risk factors. Finally, the MRI was unremarkable for infarctions or intracranial masses. With this evidence in mind, it was concluded that a neurological origin was unlikely.

After a neurological cause has been ruled out, the remaining etiologies for AOCE are often divided into 3 sub-categories. The first of the 3 sub-categories for AOCE is known as the Swan type.[6] This can be due to either occlusion or damage to the eye, causing it to stay in its phoric position. The second sub-category of AOCE, known as Franceschetti type,[6] is precipitated by an esophoric deviation that over time becomes esotropic – it is generally a larger angle deviation. The pathogenesis for this category is unknown. The 3rd sub-group, known as Bilschowsky type, involves individuals with myopia greater than 5 D.[6] Although controversial, Bielschowsky reported that the etiology stemmed from uncorrected myopes holding reading material excessively close to the eyes.[6] This would lead to a dysfunction of the convergence system. Treatment for any of the sub-categories can include occlusion, prism, botulinum toxin injections, and strabismus surgery.[7] These groups comprise the majority of non-neurological AOCE. This patient did not fit the identifying characteristics of any of the three sub-categories.

A relatively rare cause for AOCE and binocular diplopia is opiate withdrawal, which typically occurs immediately following drug cessation. Opioids are a class of naturally occurring or synthetically produced drugs used for the treatment of pain. They act on the mu receptor found primarily in the brainstem and medial thalamus.[8] Activation of this receptor elicits analgesia, sedation, euphoria, and physical dependence.[8] The latter two effects, euphoria and physical dependence, lead to a high rate of addiction and overuse.8 Heroin is an illicit opioid that is not available by prescription. It is considerably cheaper compared to prescription opioids and, therefore, readily utilized illegally. During regular sustained use, most patients have an exophoric posture. Following detoxification, the posture shifts towards eso without a noticeable change in sixth nerve function.[9] Acute onset esotropia during heroin or opiate withdrawal has also been reported in literature.[10-15] Based on a review of the available literature, the onset of the esotropia is on average at 3 days[11,12,14] following the last dose of the opioid; however, it can range from two[11] to eight[10] days. The magnitude of deviation is not readily reported. During the initial exam, the esotropia can range from 30[12] to 40[10] PD. Since the condition is transient, the deviation may change depending on the day of evaluation. Resolution of the esotropia occurs on average at about 50 days,[10-15] following onset but has a wide range from as little as 7 days[13] to as long as 150 days.[11] Patients are generally either hyperopic10 or mildly myopic.[13]

The pathophysiology of this condition is not well understood. It is theorized to be possibly due to a reversible disuse atrophy of the motor fusion system.[16] During active opioid use, the parasympathetic system is stimulated, leading to pupillary constriction. Miotic pupils naturally increase the depth of focus, lowering the accommodative demand. The decrease in accommodative demand reduces the need for active motor fusion. Over time, with the lack of use, the motor fusion system temporarily atrophies. Consequently, with acute discontinuation of opioids, the system goes into a relative parasympatholytic state where the pupil is dilated. The loss of the miotic pupil decreases depth of focus, requiring greater accommodation and accommodative convergence and with it, a demand for better motor fusion. Unfortunately, due to the atrophy, the motor fusion system cannot diverge appropriately to maintain single vision and esotropic posture with diplopia ensues.[16] It is important to note that not all individuals undergoing heroin withdrawal develop acute esotropia. AOCE and diplopia after discontinuation of heroin have been associated with underlying hyperopic refractive errors that preexisted prior to drug use and had previously been compensated for with accommodation.[16] Furthermore, Kowal indicated several risk factors that may make individuals more prone to this condition: uncorrected hyperopia, convergence excess, accommodative spasm and a high accommodation/accommodative convergence ratio.[16] In this particular case, a thorough chart review was completed and revealed that during remissive periods (verified by negative lab testing for opioids), the patient was a hyperope, had reduced amplitudes of accommodation, and was diagnosed as convergence excess. As can be seen, he had several risk factors for eliciting an acute esotropia during his withdrawal period. He did report that in general, he rarely wore his spectacles as he did not need them often. Without corrective lenses to compensate for his hyperopia, he was utilizing his accommodative system with greater effort at both distance and near. This increase in accommodation and accommodative convergence required a compensatory increase in motor fusional divergence. Moreover, with atrophy of the motor system, compensation was not likely and may explain the esotropia at both distance and near. It seems that after a period of time following the discontinuation of heroin, the motor fusion system is restored and returns to a normal functional state, which accordingly causes the esotropia to subside.

This patient fit the criteria for heroin withdrawal related sudden onset esotropia. His onset was within 3-4 days following discontinuation of heroin, with the first measured magnitude of his deviation being 25 PD, nearly 3 weeks following onset. One could speculate that he started at a higher magnitude and had already been improving by the time of that visit. He also reported complete resolution of the diplopia at 48 days. This time frame was consistent with the average resolution of 50 days reported in literature.[10-15] Of interest, the magnitude of the eso deviation was greater in right gaze on both follow-up visits at the VA (Figures 3 and 4). It is unclear why this apparent inconcomitancy was noted. Although the literature has indicated that heroin withdrawal induced esotropia is generally concomitant,[11,17] to the author's knowledge, no 9 positions of gaze testing have been reported or published. It can be speculated that recovery of the accommodative and convergence system may be asymmetric and/or imperfect, leading to confounding test findings. As would be expected in cases with incomitant deviations, brain imaging was completed.

Figure 3. 9 Positions of Gaze (Follow-up 1 week later)

9 eso	8 eso	2 eso
9 eso	1 eso	2 eso
10 eso	6 eso	3 eso

9 positions of gaze testing completed at first follow-up.

Figure 4. 9 Positions of Gaze (Follow-up 2 weeks following 1st follow-up)

4 eso	1 eso	2 eso
4 eso	1 eso	1 eso
5 eso	1 eso	2 eso

9 positions of gaze testing completed at second follow-up.

Several treatment options have been discussed, including pilocarpine[12], occlusion[13], botulinum toxin and even surgical intervention in cases that do not resolve.[17] Firth discourages the use of occlusion as temporary treatment due to the potential to precipitate swan type AOCE.[17] With this patient, the treatment option was the use of a Fresnel prism. It was thought to be the best option as the prism magnitude could easily be altered as the esotropic posture reduced in magnitude. Furthermore, the Fresnel prism is non-invasive and easy to apply in the clinic. The prism magnitude given at the ABI eye clinic was 12^ base out. This may seem large as the reported primary gaze deviation at distance was only 5 eso. Both 8^ and 10^ base out were trialed as well, but the patient was still reporting diplopia in certain gazes. With 12^ base out, the patient reported resolution of the diplopia in all gazes, apart from the extreme right gaze. He was comfortable with this, as functional gaze changes remained single. Additionally, since the distance base out compensating range was measured as 16(blur)/30(break)/20(recovery), it seems that even if he were to become exophoric in primary gaze, the compensating range would be adequate to prevent symptoms. A limitation of this report is the lack of compensating BI ranges that could provide further information on the state of the binocular system both during the initial insult and following resolution.

CONCLUSION

Heroin withdrawal-related sudden onset esotropia is a relatively unknown and pathophysiologically confounding entity. Its presenting symptoms may at first seem alarming, as ominous sixth nerve palsies may manifest the same. As with any difficult diagnosis, careful patient history and examination are paramount for identifying this condition. With the unfortunate rise in opioid and heroin addiction in recent years, this condition will likely become more prevalent, and a proper diagnosis will prove invaluable for better patient care.

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