Letter to the Editor

Acute Concomitant Esotropia during Heroin Detoxification

Dear Editor,

Although acute concomitant esotropia following heroin detoxification or withdrawal has been described in a few case reports in Europe and Australia,[1-3] it has not been reported elsewhere. Given the high prevalence of heroin use, however, this phenomenon may have been overlooked. "Diplopia/blurred vision" following heroin withdrawal has been described among Vietnam War veterans, but no attempts were made to elucidate the cause of these symptoms.[4] We report a case of acute concomitant esotropia for which the cause is most likely heroin detoxification.

A 31-year-old man complained of binocular horizontal diplopia upon awakening one morning, 7 days into an inpatient detoxification programme for heroin addiction. He first started using various painkillers following a sports-related injury, which left him with severe lower back pain. He took up to 20 oxycodone or hydrocodone with acetaminophen pills daily. He began smoking heroin (4 to 6 balloons a day) 2 years ago and had failed 2 previous detoxification regimens (which did not include the use of methadone) during which he did not experience any ocular problems. On the day of admission into the detoxification programme, he admitted to using heroin and hydrocodone. He rarely used cocaine, marijuana, and methamphetamines, and the last use had been at least a few months prior to the onset of diplopia. He drank alcohol occasionally and never used intravenous drugs or smoked cigarettes. On admission, toxicology screen showed only opiates and no benzodiazepines, cocaine, methamphetamines, or other illicit drugs. He was sequentially placed on lorazepam, clonidine, quetiapine, trazadone, divalproex, zolpidem, and hydroxyzine, along with lidocaine patch for the back pain during the detoxification programme. He denied trauma, recent viral infections, headache, blurry vision, or other neurological symptoms. He had no history of medical or ocular disease such as strabismus, diplopia, amblyopia, or neurological deficits.

Ocular exam revealed uncorrected vision of 20/20 in both eyes. There was no afferent papillary defect. Confrontational visual fields were normal in both eyes. Ductions were full in both eyes, demonstrating no apparent weakness or nystagmus in all directions of gaze. Anterior segment examination was unremarkable. Ophthalmoscopy revealed no abnormalities in the retina or optic nerve. There was, however, a 24-prism dioptre (PD) esotropia at distance (20 feet) and a 19 PD esotropia at near (approximately 3 feet) in the primary gaze. There was no difference in the deviation in all directions of gaze. There was no evidence of superior or inferior oblique dysfunction. Magnetic resonance imaging (MRI) of the brain and brainstem revealed only left maxillary sinusitis and no other abnormalities. He was advised to wear an occlusion patch on one eye. After an otherwise uneventful detoxification, he was discharged on daily divalproex and zolpidem as needed.

Ten days later in the outpatient clinic, he continued to complain of diplopia, although it was subjectively slightly improved. He had a 15 PD esotropia at distance and a 12 PD esotropia at near. Cycloplegic refraction revealed +1.00 +0.25 x 90 in the right eye and +0.75 +0.50 x 40 in the left eye. He was advised to wear +1.00 glasses to determine if the diplopia could be alleviated, although it did not affect the degree of esotropia in the clinic.

Thirty-seven days after the onset of symptoms, the patient still complained of diplopia. He had not tried the +1.00 glasses. He continued to have a 15 PD esotropia at distance and 12 PD at near. With +1.00 correction on both eyes, the distance esotropia was 12 PD. Ten minutes after pilocarpine 1% was administered in both eyes, his refraction was -1.75 in the right eye and -2.25 in the left eye. Esotropia at distance was 10 PD, but he was orthotropic at near. He was advised to wear +3.25 lenses for reading, with which he no longer experienced double vision at near. However, diplopia at distance could only be neutralised with prismatic correction.

Acute, late-onset esotropia is most likely caused by decompensated, uncorrected hypermetropia.[5,6] It may also be associated with neurological disease such as Chiari malformation.[7] Without neurological signs, however, further evaluation may not be necessary.[5] In our patient, a normal brain MRI and the lack of other symptoms suggested that neurological disease was unlikely. Maxillary sinusitis has been associated with hypotropia, probably from adjacent inflammation that affects the inferior rectus muscle.[8] It has not been associated with esotropia. Withdrawal from other addictive substances such as cocaine, marijuana, and methamphetamines also has not been associated with esotropia.

It is believed that the "sudden parasympatholytic state with pupillary dilation and paralysis of the ciliary muscle" associated with heroin withdrawal may precipitate decompensation of fusion, leading to esotropia.[3] In another case series, 4 out of 5 patients were found to have strabismogenic features such as hyperopia and oblique

dysfunction, and it was suggested that the "physiological turmoil in the withdrawing patient" could weaken the motor fusion system.[2] Esotropia has also been reported in the setting of diabetic ketoacidosis[9] and following "physical and psychic shock."[10] In our patient, it was possible that withdrawal from other substances may contribute to the "physiological turmoil", but heroin and other narcotic medications were the major drug of choice and thus the most likely culprit. Heroin also induces miosis, resulting in a pinhole effect which allows increased depth of focus in a hyperope with less accommodative effort and accommodative convergence. With heroin intake, exotropia may develop, possibly because of this decreased drive for accommodative convergence.[3] Heroin withdrawal may have the opposite effect. In addition to miosis, pilocarpine also induces myopia, which theoretically would alleviate accommodative esotropia. In our patient, pilocarpine alleviated the esotropia at near. It is believed that the mechanism of esotropia is unrelated to divergence insufficiency or sixth nerve palsy.[11]

Management of heroin withdrawal-related esotropia usually entails prismatic correction to facilitate binocular fusion.[12] Prism strength can be gradually tapered, but surgery may be necessary for intractable cases.[1] Pilocarpine was described as "subjectively helpful" in 1 hyperopic patient.[2] However, it causes blurry vision and the effects are too imprecise to be used chronically.

As heroin use is prevalent in many countries, physicians should be aware of strabismus associated with heroin use and withdrawal to avoid unnecessary diagnostic evaluations. Prompt and effective treatment for debilitating diplopia is also important to minimise relapse and detoxification failure.

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