



Ocular manifestations of opiate and alcohol

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KEYWORDS

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Dear Editor

I read with great interest the recent publication by Khoshnazar et al. regarding the accommodative functions of opium users compared to non-users. They found significant changes in all baseline accommodative functions after the application of 5% phenylephrine eye drops in opium users, except for negative relative accommodation, which remained unchanged [1].

Additionally, I came across an insightful study published in your journal titled "Binocular vision parameters in chronic heavy alcoholics." The research found that, after one month of alcohol detoxification, there were no significant changes in the binocular vision parameters of chronic heavy drinkers. Most individuals exhibited non-strabismic binocular vision anomalies, and these conditions remained unchanged after rehabilitation. This suggests that short-term alcohol detoxification may not be effective in improving binocular vision parameters [2].

In this letter, I aim to summarize the ocular effects of alcohol and drug abuse and highlight the confounding factors that may impact study outcomes.

Illicit drug consumption is an increasing global public health concern, with its prevalence steadily on the rise. The effects of these substances on the eyes and visual pathway can vary, ranging from tear film instability to severe vision loss due to conditions such as endophthalmitis or occipital lesions. The existing literature on the ocular effects of drug abuse mainly consists of case reports and retrospective studies, with a noticeable lack of systematic reviews or cohort studies [3-5].

Drug abuse can result in various ocular manifestations. Cannabinoids may lead to conjunctival injection, mydriasis, reduced accommodative amplitude, and impaired oculomotor function. Opiates are associated with miosis, nystagmus, disturbances in eye fixation, saccadic intrusions, and oscillations. Intravenous abuse can result in retinal microembolisms and endophthalmitis. Acute-onset esotropia may occur during heroin withdrawal, along with retinal ischemia and disc neovascularization when using crushed oxymorphone intravenously. Cocaine use may cause mydriasis, cycloplegia, exophthalmos, upper eyelid retraction, optic neuropathy, orbital apex syndrome, corneal epitheliopathies, ulcers, and retinal vascular occlusion. Methamphetamine can lead to mydriasis, crystalline retinopathy, and retinal vascular occlusion. Barbiturates and benzodiazepines may cause mydriasis, decreased convergence, extraocular muscle paresis or nystagmus, and ptosis. Methaqualone is linked to diplopia, hallucinations, visual disconnection, retinal hemorrhage, and mydriasis. Gamma hydroxybutyrate is associated with accommodation abnormalities, blurred vision, sixth nerve palsy, nystagmus, and Wernicke-Korsakoff syndrome [3-5]. Recreational users of poppers who develop maculopathy often present with vision loss or visual disturbances due to foveal disruption, as well as a decline in electroretinogram readings [6, 7].

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Alcohol consumption leads to early and significant changes in the tear film and the ocular surface epithelia, which can cause visual disturbances [8]. Ingesting methanol may result in severe, irreversible bilateral toxic optic neuropathy, potentially leading to blindness. The effects of alcohol on the eyes can be both short-term and long-term, ranging from blurred vision to serious, vision-threatening conditions. Direct impacts include toxic neuropathy, fetal alcohol syndrome, diplopia, and Wernicke's encephalopathy. Indirectly, alcohol use can increase the risk of cardiovascular diseases and ocular ischemia. Furthermore, it may elevate the risk of ocular trauma and interact negatively with other medications [4, 9]. Heavy alcohol consumption is also significantly associated with a higher risk of developing age-related cataracts [10].

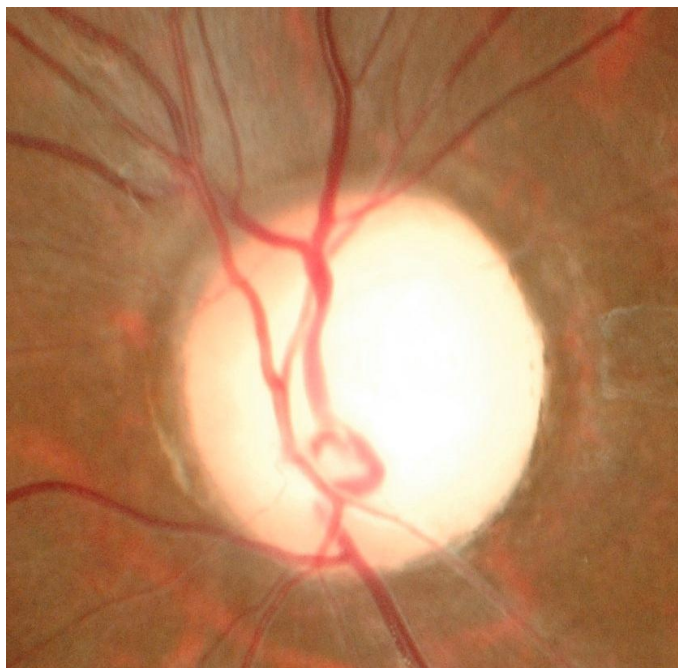


Figure 1. The left eye optic nerve head image of a patient with optic atrophy. This is characterized by an enlarged optic cup and a generalized narrowing of the peripapillary arteries, observed three months after the patient was diagnosed with methanol-induced optic neuropathy.

Acute alcohol intoxication can lead to abnormal eye movements, altered color perception, and decreased contrast sensitivity. Acute methanol poisoning may result in optic neuropathy (Figure 1), optic disc edema, damage to the retinal ganglion cells, and permanent scotomas or vision loss. The teratogenic effects of alcohol can include facial abnormalities such as short palpebral fissures, ocular hypertelorism, coloboma, epicanthus, strabismus, blepharoptosis, cataracts, microphthalmia, optic nerve hypoplasia, and tortuosity of the retinal vessels. On the other hand, chronic alcoholism is associated with a range of ocular issues, including cataracts, dry eye syndrome, corneal epitheliopathy, primary open-angle glaucoma, alcohol-induced optic neuropathy, age-related macular degeneration, retinal vein occlusion, central serous chorioretinopathy, functional retinal diseases, and asteroid hyalosis [9].

Some individuals who abuse drugs often consume multiple substances, frequently in combination with alcohol or tobacco. These substances can significantly affect ocular health, making it difficult to isolate the effects of a specific drug. Additionally, street drugs are rarely taken in their pure form; they are typically diluted or mixed with other substances. These additional components may have unknown effects, serving as further confounding factors. Moreover, drug abusers are often hesitant to provide complete information about their drug use to healthcare providers [3]. Illicit drugs can be administered through various routes, including oral ingestion, smoking, nasal inhalation, intravenous injection, topical application, or via mucosal surfaces [5]. For instance, Alipour et al. reported a case of a 16-year-old girl who developed atypical corneconjunctival lesions as a result of transconjunctival heroin abuse. The authors concluded that substance abuse should be considered in the differential diagnosis for patients presenting with atypical ocular symptoms [11]. Given the different routes of administration, it is crucial to differentiate these methods in studies, as they may lead to various adverse biological effects, particularly when combined with other substances.

There has been significant focus on the connections between substance use disorders and outbreaks of human immunodeficiency virus infections, as well as acute viral hepatitis [12]. In light of the persistent public health challenges, it is crucial to identify the causes of ocular manifestations that could be mistaken for confounding factors, especially in patients who are at risk for sexually transmitted diseases.

Research on the ocular effects of illicit drugs and alcohol has some limitations, such as focusing on specific age and sex groups, as well as certain dosages and brand names. To validate these findings, it is essential to conduct further studies that include a balanced distribution of doses, age groups, and sex ratios. Future research should also aim to minimize confounding factors to ensure more accurate and reliable results.

The eye care providers should consider the ocular effects of drug abuse and alcohol consumption. They should also be involved in patient education, early diagnosis, and appropriate management of potential vision-threatening conditions caused by substance abuse and heavy alcoholism.

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