Review Article

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Illicit drugs: Effects on eye

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There is a myriad of changes that can be produced in the eye by toxic drugs ranging from mild/no symptoms to severe loss of vision from endophthalmitis. The routes of administration include oral ingestion, smoking, nasal inhalation, intravenous injection, topical application or application to other mucosal surfaces. It is important to recognize certain clinical signs and symptoms in the eye produced by these toxins. This article describes in brief some of the ocular effects of commonly abused drugs. For identification of a particular poisoning, in addition to the clinical presentation, pulse, blood pressure, respiration and body temperature, pupillary size, pupillary reaction to light, ocular convergence and nystagmus can be useful indicators of the type of drug the patient is exposed to. Unmasking these features help the clinician in an early and accurate diagnosis of the offending drug as well as timely management.

Key words Alcohol - blurred vision - cannabinoids - illicit drugs - methanol - ophthalmology - opiates - retinopathy - smoking - toxins

Introduction

There are numerous illicit drugs or chemicals causing unwanted physiological changes in our body. Several of these may have ophthalmic effects. It is important from an ophthalmologist's point of view to have knowledge regarding the effects of these illicit drugs on the eye. Understanding their adverse effects on the eye can aid in early diagnosis and initiating appropriate treatment. The routes of administration of these drugs include oral ingestion, smoking, nasal inhalation, intravenous injection, topical application or application to other mucosal surfaces[1]. The changes that can be produced in the eye by toxic drugs range from mild/no symptoms to severe loss of vision and endophthalmitis resulting in a permanent loss of sight.

Intravenous drug abuse can lead to microemboli in retinal microcirculation leading to retinal ischaemia[2,3]. Spread of microorganisms (including *Candida*, *Aspergillus*, *Bacillus*, *Staphylococcus*, *Pseudomonas*, *Klebsiella, etc*.) to the eye through blood stream due to contaminated needles can lead to endogenous endophthalmitis[2,3]. Several drugs which dilate the pupil can lead to angle-closure glaucoma in predisposed patients with narrow angles[4]. Often such patients present to the emergency team or to a physician in the acute setting. Recognition of subtle ophthalmic signs in a patient who does not have the whole manifestation of symptoms due to a particular drug abuse can be beneficial. This review describes some of the ocular effects of commonly abused drugs.

Alcohol

Alcohol abuse is emerging as a major public-health problem in India and more than half of all alcohol drinkers fall into the category of hazardous drinking[5]. Alcohol intake in short term leads to dilated pupils, slower pupillary reaction, diplopia, night vision disturbances[6,7], decreased contrast sensitivity, congested eyes, twitching of eyelid (myokymia) due to excessive intake and nystagmus. Alcohol intake may impair the vision or orientation to visuospatial stimuli due to the various mechanisms. Alcohol intoxication can also impair mesopic rod and cone temporal processing pathways[8]. The mean subfoveal choroidal thickness increases during the first hour after alcohol consumption and decreases during the next two hours[9].

Chronic intake of alcohol can cause external ophthalmoplegia (due to thiamine deficiency), toxic amblyopia and age-related macular degeneration (ARMD). Ethanol is detected in tears, and it decreases tear film volume, disturbs tear film structure[10], increases tear hyperosmolarity[11], induces increased expression of inflammatory cytokines[12], and vitamin A deficiency and all these factors combined lead to dry eyes. Chronic alcoholism changes the conjunctival flora by increased colonization of *Staphylococcus aureus* bacteria which along with associated dry eye is responsible for higher rates of keratitis in alcoholics[13].

Intake of alcohol with other abusive agents is also common, and a cocktail of drugs can lead to a multitude of changes in the eye which are often unpredictable (Fig. 1). Alcohol dependence can be associated with other addictive disorders, among which nicotine dependence is most common in about 80-90 per cent of the patients[14]. Coexistent use of smoking and alcohol is known to cause tobacco-alcohol amblyopia, the cause of which is hypothesized to be either because of these substances themselves or nutritional deficiency associated with the abuse. The prevalence of toxic optic neuropathy among alcohol addicts might be underestimated. A pilot study on a group of alcoholic patients reported 13 per cent prevalence of bilateral typical optic neuropathy in males, but about 20-40 per cent of patients had incomplete forms of optic neuropathy with or without visual impairment which was unilateral and caused impaired colour vision[15]. Hence, screening of these patients for early detection of toxic optic neuropathy is essential. Early identification can lead to timely preventive measures and abstinence from drug abuse as well as vitamin supplementation[15].

Eye thermal signatures as a test to detect alcohol drunkenness

In a sober person, temperature of sclera and iris is the same, but with alcohol intoxication, temperature of sclera increases compared to the iris because of denser blood vessel network over the sclera, and thermal signature of eye with infrared imaging may provide first assessment tool to detect alcohol drunkenness[16].

Treatment

The acute effects of alcohol seldom need treatment as these subside with time. The treatment of Wernicke's encephalopathy is a medical emergency, and mainstay of treatment is intravenous hydration and thiamine.

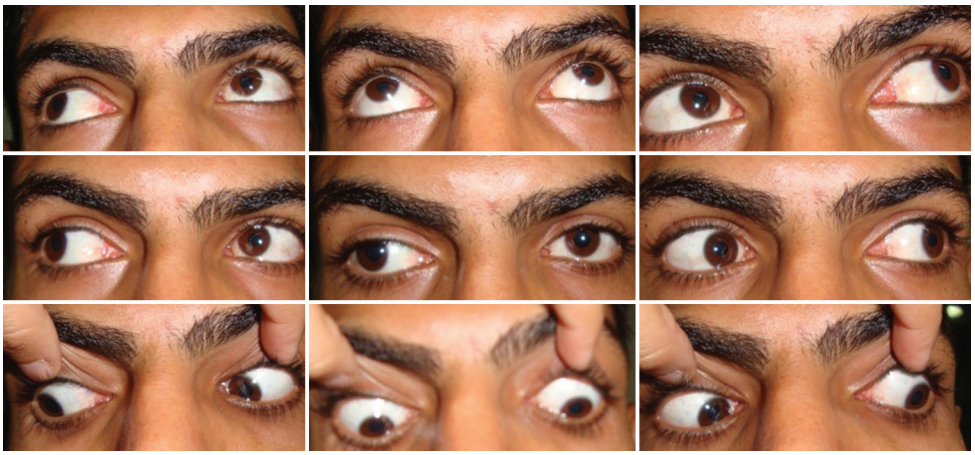

Fig. 1. Unpredictable and unwanted consequences of combination of alcohol with other drugs. A 24 yr old male with a visual acuity of 6/6 in both eyes with normal pupillary reactions but 45 prism dioptre of exotropia in the right eye with full extraocular movements in all gazes. Patient presented with binocular diplopia for 4-5 months following intake of marijuana and alcohol. On the basis of multiple infarcts on magnetic resonance imaging brain, a diagnosis of reversible cerebrovascular spasm syndrome was made.

Magnesium sulphate is also used to reduce the potential for seizures. Chronic thiamine supplementation may be required to reverse the external ophthalmoplegia[17].

Nicotine

Cigarette smoking is one of the most common and the most alarming health problem today. It can affect multiple structures in the body as well as the eye due to chemical toxicity and free radical-related oxidative damage. Nicotine has been reported to cause alteration of the conjunctival flora, irritation, redness, dry eye, ocular surface inflammation and meibomian gland dysfunction[18-20]. Tear film breakup time is decreased which is suggestive of unstable tear film. Schirmer's test may be normal, lower or even higher because of unstable tear film and reflex tearing[18,21]. Smoking also increases the risk of squamous metaplasia of bulbar conjunctiva and conjunctival intraepithelial neoplasia[21,22]. Corneal wound healing is delayed in smokers, increasing the risk of keratitis and poor healing of epithelial defect or ulcer[23]. Some studies have shown reduced endothelial cell count or a decrease in hexagonality of endothelial cells[24,25]. The only beneficial effect on the cornea has been an accelerated collagen cross-linking leading to improved corneal biomehanics[26].

Smoking increases the risk of cataract formation including nuclear and posterior subcapsular cataract (PSC)[27]. It has a higher association with nuclear cataract compared to PSC, but it does not increase the risk of cortical cataract[28,29]. The City Eye Study[29], a nine year prospective study conducted to know the association between lens opacities and risk factors has shown the relative risk for nuclear lens opacity of 2.6 for past heavy smokers and 2.9 for present heavy smokers.

Risk of development of ARMD is also increased in genetically susceptible individuals who smoke. Smoking has been shown to increase the risk of polypoidal choroidal vasculopathy (3 times more than non-smokers) and choroidal neovascular membrane due to ARMD (4 times more than non-smokers)[30]. According to The Blue Mountains Eye Study[31], current smokers have four times greater risk of late ARMD than non-smokers. Smoking along with alcohol also predisposes the patient to ARMD.

Regarding effects of smoking on intraocular pressure (IOP), studies have shown variable results. The Blue Mountains Eye Study showed no relationship of IOP with smoking[32]. However, some studies have shown higher mean IOP in smokers independent of corneal biomechanics[33]. The mechanism of raised IOP has been proposed to be vasoconstriction and rise in episcleral venous pressure due to smoking, thus increasing the risk of glaucoma[32]. Smoking increases the risk of development of thyroid-associated orbitopathy, and it is associated with its progression and poor response to treatment[34,35]. Ophthalmologists should advise patients with thyroid-associated orbitopathy to abstain from smoking.

There is doubtful association between smoking and non-arteritic anterior ischemic optic neuropathy (NAION). One study has shown that smoking increases, and its cessation reduces the risk of NAION[36]. Hayreh *et al*[37] did not show any association with smoking. Smoking with/without alcohol leads to dysfunction of electron transport chain of mitochondria leading to cell death with damage to papillomacular bundle because of combined toxic cyanide and formic acid levels along with nutritional deficiency of vitamin B12, folate, thiamine, *etc*[38,39]. It is initially manifested as a change in colour vision and later on causes progressive decline in visual acuity leading to a central fixation scotoma. However, visual loss usually does not go beyond 20/400 and does not lead to blindness. Optic nerve may be normal or hyperemic in early stages, and later, there may be temporal pallor of disc[40]. Visual field shows central or centrocaecal scotoma.

Methanol

Methanol intake in the form of an adulterated drink can lead to metabolic acidosis (due to toxic metabolite formic acid). Symptoms include headache, dizziness, nausea, vomiting, abdominal pain and blurred vision. The onset of symptoms is usually delayed for 12-24 h. In severe poisoning, dyspnoea, coma, convulsion and blindness may occur[41]. Fatal dose can be as little as 30 ml and blindness can occur with as little as 10 ml[42]. Fundus may be normal in case with visual impairment and retrobulbar neuritis. Optic neuropathy can be present in the form of hyperemic optic disc oedema followed by optic disc pallor and cupping if not treated on time. The cause of optic neuropathy is considered to be mitochondrial dysfunction and progressive demyelination in acute stage followed by retrograde degeneration of optic nerve axons leading to optic disc cupping and pallor[43]. In a case report, methanol intoxication has been shown to cause bilateral multifocal extrafoveal retinal pigment epithelial detachments along with optic neuritis[44]. Methanol has also been shown to cause effects on outer retina including retinal pigment epithelium and photoreceptors[45].

Treatment

It includes gastric lavage, ethanol/fomepizole (aldehyde dehydrogenase enzyme inhibitors), haemodialysis to remove toxic metabolites, folinic acid to enhance the metabolism of formic acid, sodium bicarbonate for acidosis, correction of vitamin deficiencies and use of intravenous steroids for optic disc oedema[46]. A retrospective case series of 37 patients with visual disturbance after methanol poisoning reported that 62 per cent patients completely recovered, 14 per cent recovered partially, 11 per cent had partial recovery followed by deterioration to blindness and 14 per cent had complete blindness[47]. A study on predictors of visual outcome in methanol poisoning has found acidosis at presentation to be a stronger predictor of final visual acuity[48]. An inverse relationship has been found between serum methanol levels at presentation and final visual acuity. Initial *p*H <7.2 was associated with lesser improvement in visual acuity. Early presentation and treatment may not affect the visual outcome, especially in a case of severe poisoning[48].

Role of erythropoietin

There are many studies on the use of erythropoietin for methanol poisoning because of its ability to reduce the neuronal apoptosis, reduction in inflammatory response and its neuroregenerative properties. Intravenous erythropoietin 10000 IU twice a day for three days has been shown to improve the visual outcome dramatically in patients already receiving supportive measures and intravenous steroids. However, whether it is efficacious when given alone, needs further studies[49]. A study evaluated the effect of steroids plus erythropoietin versus steroids alone and found that patients with steroids plus erythropoietin showed deterioration in visual acuity at two months. The conclusion is that protective effect of erythropoietin may be strong at the beginning of intervention, but it is probably transient[50]. Another study on efficacy of intravitreal erythropoietin in late-stage optic neuropathy did not find any beneficial or detrimental effect, but its effect in early stage is still to be determined[51].

Cannabinoids

The active compound is tetrahydrocannabinol (THC), and route of intake can be smoking or oral ingestion. The effects of smoked cannabinoid begin within minutes and usually last for 1-3 h. It leads to euphoria, short attention span and red eyes. With oral ingestion, concentration peaks occur at about 1-3 h[52]. Cannabis intake leads to conjunctival injection[53], dilated pupils[54], reduced accommodation amplitude[55,56] and impaired oculomotor function in chronic users. Impaired oculomotor function can manifest in the form of increase in latency to initiate saccades, impairment in processing of saccades and impaired visuospatial working memory[57], and smooth pursuit eye tracking performance[58]. Because of high lipid solubility of THC, it accumulates in fat cells. When used continuously, it is slowly distributed out of the cells. Its metabolites can be detected in urine for one day to a week or longer after acute use, depending on the amount smoked[59]. A case of conjugate deviation of the eyes due to cannabis intoxication was reported which lasted for six weeks[60]. The reported effects of the drugs can last longer sometimes without detectable levels in urine. Fig. 2 highlights the pupillary side effects of marijuana. Cannabis decreases IOP, but it is not suitable for medical purpose for glaucoma because of short duration of action (3-4 h) and its addiction properties. For being effective, it must be smoked 6-8 times a day which can lead to dependence and patients develop tolerance with time[61].

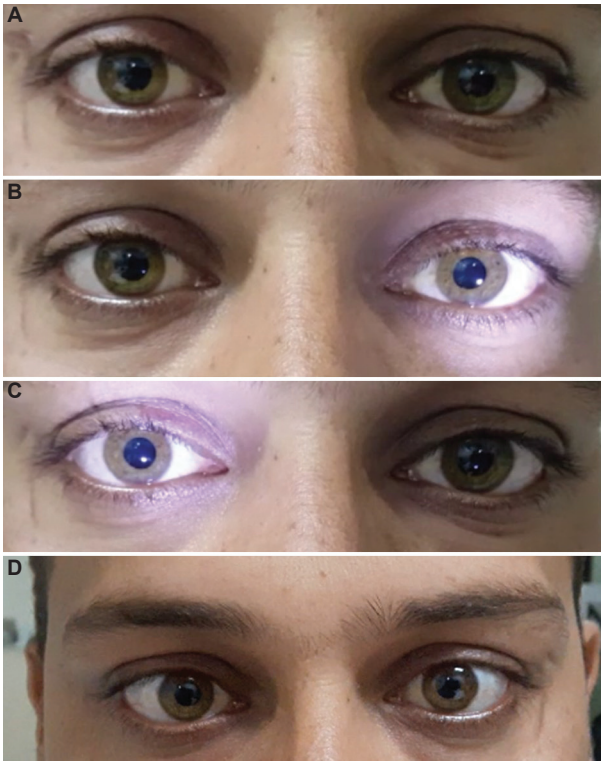

Fig. 2. Dilated pupils in both eyes (**A**) and sluggishly reacting on direct and consensual reflexes (**B** and **C**) as well as near reflex (**D**) in a 25 yr old male patient with a history of smoking marijuana. Patient presented with a one week history. Pilocarpine one per cent drops were described to relieve symptoms.

Opiates

Opiates include numerous substances such as morphine (naturally occurring), heroin (semisynthetic), meperidine and methadone (synthetic derivatives) and prescription opioids including hydrocodone, oxycodone, pentazocine and fentanyl.

Routes of intake

The routes of intake for morphine include oral, intravenous, intramuscular, rectal, epidural and intrathecal. Morphine tablets can be injected, and opium can be smoked. Heroin intake can be in the form of smoking, snorting, or intravenous and subcutaneous administration. Black tar heroin is usually dissolved, diluted and injected. These drugs act through the opioid receptors μ, κ, δ, and cause a decrease in the pupillary size and in the velocity of constriction to light stimulus, and dilatation after the light stimulus is removed[62]. The effect usually starts in 15-60 min and lasts for 3-5 h. Miosis by morphine is due to an excitatory action on the Edinger-Westphal nucleus of the oculomotor nuclear complex[63]. The effect on pupil diameter in dependent and non-dependent individuals varies because of the development of tolerance in dependents. A study on the effect of heroin has found that pupillary constriction starts in 15 min and persists for at least two hours in non-dependent individuals whereas dependent individuals show recovery from pupillary constriction after 15 min[64]. Therapeutic doses of morphine increase accommodative power and decrease IOP in normal and glaucomatous eyes[65,66].

The triad of coma, pupillary constriction and depressed respiration suggests opioid poisoning. Intravenous heroin and morphine abuse can cause downbeat nystagmus, transient disturbance of eye fixation, saccadic intrusions and oscillations, lasting for 10-15 min[67]. Intravenous abuse can also lead to microembolism in the retinal vasculature and endophthalmitis. Acute-onset esotropia can be seen in about 30 per cent of individuals following withdrawal of heroin which is manifested as an acute onset of binocular diplopia with impaired convergence[68]. Neurological investigations are normal in such patients. Hence, clinicians should be aware of this condition due to drug use and avoid unnecessary and extensive neurological investigations[68].

Diffuse retinal ischaemia and disc neovascularization with intravenous use of crushed oxymorphone have been reported which is intended to be used as an oral opioid analgesic. It has also been associated with thrombotic thrombocytopenic purpura (TTP). Hence, any patient with TTP like illness and retinal findings should be questioned regarding drug abuse, and urine testing should be done[69]. To detect recent use of opioids, a useful test is the nalorphine test[70]. Two to four milligrams of nalorphine is injected subcutaneously, and the pupillary dilation is observed within 30 min. Narcotic users show dilation of pupils whereas in patients who are non-opiate users or have not used narcotics recently, pupillary constriction will be observed[70]. During the withdrawal states of opioids, mydriasis or anisocoria can occur.

Stimulants

Commonly abused stimulants include cocaine, amphetamine, methamphetamine, and 3,4-methylenedioxymethamphetamine (MDMA, ecstasy); in increasing order of potency cocaine < amphetamines < methamphetamine < MDMA. Other psychostimulants include cyclazodone, 4-methylaminorex and prescription stimulants.

Cocaine

Cocaine can be ingested orally, and combined opioid and cocaine abusers, use it as intravenous injection. It is often used with alcohol. With freebase inhalation, effects occur within 4-6 sec and lasts for 5-7 min only. When the powder is sniffed, effects are produced within 1-3 min and last for about 30 min. Cocaine causes dilated pupils because of inhibition of reuptake of norepinephrine. In high concentrations, it may cause cycloplegia, and in chronic users, exophthalmos and retraction of upper eyelid can occur[56]. A case of severe sinusitis following intranasal cocaine abuse was reported which spread to the orbit leading to optic neuropathy and orbital apex syndrome[71]. Cocaine users can also present with complications like superficial punctate keratitis, epithelial defects and ulcers because of contamination through eye rubbing or retrograde passage of the substance through the nasolacrimal duct by sniffing, as well as direct toxic effects from substance smoke[72]. Conjunctival lesions and chronic red eye have been reported with the transconjunctival use of crystallized heroin[73].

Methamphetamine

It increases the production of dopamine in the brain and activates reward centres of the brain giving a sense of euphoria soon after taking the drug and causes aggressiveness, anxiety and dilated pupils. It is known to cause crystalline retinopathy by

intranasal methamphetamine use[74]. Retinal vascular occlusive disease can also occur with cocaine and methamphetamine[75,76]. Psychostimulants act on dopamine receptors in the brain. These enhance the activity of sympathetic nervous system leading to increased pulse rate, respiratory rate and blood pressure.

Cyclazodone (n-cyclopropylpemoline)

It is a novel stimulant drug which produces stimulating and focus-enhancing effects similar to dexamphetamine by increasing release of dopamine, noradrenaline and serotonin. Its ocular and visual effects are less consistent and usually occur at higher doses in the form of pupillary dilatation and brightness alterations which manifest as change in the level of perceived brightness *i.e.,* surroundings may appear darker and gloomier or brighter[77]. Transformations may also occur rarely with high doses which manifest as smooth and fluid-like transitions of an object in various shapes.

4-Methylaminorex

It is a stimulant drug with its action similar to amphetamine and available in powder and tablet forms. It is abused because of its stimulant and euphoric effects. The unwanted effects include agitation, nausea, tachycardia, restlessness and dilated pupils[78]. Currently, its availability is limited, and hence, it is abused less frequently.

Prescription stimulants

Prescription stimulants include amphetamines, methylphenidate for attention-deficit hyperactivity disorder and nasal decongestants such as pseudoephedrine, phenylephrine, promethazine, phenylpropanolamine and oxymetazoline. These drugs can also be abused/misused, and drug effects on eye can occur in the form of pupil dilatation and precipitation of angle-closure glaucoma in predisposed individuals with narrow angles[79]. Anecdotal case of anisocoria in a patient on oral decongestant pseudoephedrine for sinusitis in which anisocoria occurred because of the absence of pupil dilatation in one eye with latent form of Horner's syndrome has been described[79]. Promethazine because of anticholinergic property and phenylpropanolamine due to sympathomimetic activity have been reported to cause acute angle-closure glaucoma[80].

Saturday night retinopathy

It has been described in patients with heavy drug abuse (alcohol, iv heroin/ methadone) which is characterized by unilateral vision loss along with proptosis and ophthalmoplegia after heavy intravenous drug abuse. It occurs because of unconsciousness following heavy drug abuse and patient sleeping in abnormal posture with continuous pressure on the orbit leading to orbital congestion and ophthalmic/central retinal artery occlusion. It may also be associated with peroneal nerve palsy of lower limb[81]. Orbital congestion and proptosis improve with time, but visual prognosis is poor.

Hallucinogens

This group includes lysergic acid diethylamide (LSD), psilocybin, phencyclidine (angel dust) and mescaline. These drugs can cause hallucinations, recklessness, sleeplessness, slurred speech, hyperarousal of the central nervous system (CNS), loss of coordination and pupil dilation. LSD 'trip' typically lasts for 6 to 18 h. The effects of psilocybin and mescaline are similar to those of endogenous serotonin and can last for 8 to 12 h[82]. There have been no systematic studies on dynamic measures of light reflex after the intake of these drugs.

Phencyclidine does not cause changes in pupil size but often causes horizontal and vertical nystagmus in intoxicated states[83]. A case of phencyclidine-induced oculogyric crisis with involuntary conjugate upwards deviation of eyeballs was reported, the rest of the ocular and systemic examinations were normal and patient improved with diphenhydramine[84]. Dystonia can be improved with anticholinergics/antihistaminics.

Poppers maculopathy

Poppers belong to a group of alkyl nitrites and are used as recreational drugs in the form of inhalation. Popper use has been found to cause visual impairment due to photoreceptor damage because of an increase in cyclic guanosine monophosphate (cGMP) leading to disruption of inner segment-outer segment (IS-OS) junction in the fovea[85]. Some improvement of vision may occur on cessation of drug use and using oral lutein. It has been found more in HIV patients/who are on sildenafil/proteinase inhibitor. The pathogenesis is not exactly known. It has been postulated to be due to sildenafil/proteinase inhibitor use which also increases cGMP similar to poppers and probably enhances the photoreceptor damage[86]. Another study suggested that poppers maculopathy might be caused by photic injury because of its similarity on clinical examination as a small yellow spot at the fovea and IS-OS junction disruption[87].

Central nervous system (CNS) depressants

Barbiturates and benzodiazepines

Benzodiazepines are commonly self-administered by addicts, sometimes to ameliorate withdrawal from heroin, alcohol or to weaken the side effects of cocaine or methamphetamine intoxication. Addicts may also combine these drugs with heroin, marijuana or alcohol to enhance their effects. The short- and intermediate-acting barbiturates are lethal if taken more than 10 times a single therapeutic dose. Benzodiazepines are non-lethal unless combined with alcohol or other CNS-depressant drugs. The symptoms of benzodiazepine overdose include drowsiness, slurred speech, ataxia, horizontal gaze nystagmus, hypotension, coma, respiratory depression and cardiorespiratory arrest[88]. Some patients can have severe allergies such as anaphylaxis and angioedema[89,90]. Flumazenil is indicated for reversing the sedative effect of benzodiazepines and for treatment in a benzodiazepine overdose[91]. The treatment has to be done under the supervision of a psychiatrist. Large doses of barbiturates can cause decreased pulse rate, shallow breathing and dilated/normal pupils. Common ocular manifestations are disturbances of ocular movements, including decreased convergence, paresis of extraocular muscles or nystagmus. Pupillary response is variable although hippus and sluggish pupillary reaction can be seen[92]. Subnormal vision or bilateral blindness has been reported in patients recovering from coma caused by barbiturates[93]. Ptosis is common in habitual barbiturate users[94]. There is no antidote to reverse barbiturates action.

Methaqualone

It is a CNS depressant with sedative-hypnotic action on gamma aminobutyric acid (GABA) A receptor and is used for insomnia. It has addiction potential, but unlike barbiturates, it does not cause respiratory depression. It can have visual and ocular effects at high doses. Visual effects can occur in the form of double vision, hallucinations and visual disconnection. It is known to cause generalized purpura due to thrombocytopenia, and its ocular effects can include conjunctival[95] and retinal haemorrhages[96]. Pupil size and reaction usually are not affected, but dilated pupils can occur at very high doses[97].

Gamma hydroxybutyrate (GHB)

It is a depressant drug with its actions on its own receptor in brain and GABA B receptor and is used by club goers for its euphoric action, body builders for probable action on growth hormone boost and as a date rape drug. It is available as powder and taken in liquid/oral form and can be mixed with any alcoholic/non-alcoholic drink. It can cause blurred vision, visual disturbances with difficulty in focusing. Due to its tendency to cause dependency, withdrawal symptoms have been reported in the form of 6th nerve palsy, nystagmus and Wernicke-Korsakoff syndrome[98]. Wernicke-Korsakoff is proposed to be due to thiamine deficiency and gets ameliorated with thiamine supplementation.

In utero use of abusive drugs

A study on infants born to drug-misusing mothers who were prescribed methadone in pregnancy for opioid dependence, found abnormal visual development in infants in the form of strabismus (25%, 10-fold higher than in normal children), decreased visual acuity (22%), nystagmus (11%) and five-fold higher risk of failing in six-month visual assessment[99]. Exposure to opiates and/or benzodiazepines during pregnancy may cause infantile nystagmus in child[100]. Foetal alcohol syndrome is associated with optic nerve hypoplasia, strabismus and decreased saccadic velocity[83]. Opioids and polysubstance abuse in mothers has been found to be associated with poor visual acuity and binocular visual functions compared to control groups even if they are detoxified during pregnancy[101].

Trauma

Intoxicated patients are more prone to road side accidents/assaults. Alcohol-related ocular injuries have been found to be associated with severe globe rupture with high incidence of adnexal injuries and were associated with worse visual outcome and higher rates of evisceration[102]. Among addicts, polydrug use is very common; so, variable ocular and systemic effects can be seen due to combined mechanisms. Treatment from ophthalmologist point of view is as per the effect of drug, *e.g.,* for pupillary effects - mydriatic or miotics; for ocular surface diseases including dry eyes and conjunctival hyperaemia - tear substitutes and topical steroids may be required; and for accommodation - convergence dysfunction, refractive correction to be prescribed until the effect of abusive drug subsides.

Tables I and II summarize the ocular manifestations of commonly abused drugs. For identification of poisoning, clinical presentation, pulse, blood pressure, respiration, body temperature, pupillary size, pupillary reaction to light, ocular convergence and nystagmus

Table I. Frequent ophthalmic or visual manifestations and most commonly abused drugs causing them

Ocular/visual manifestations	Suspected drugs
Diplopia	Alcohol, phencyclidine, barbiturates, heroin
Corneal anaesthesia, keratitis	Phencyclidine, cocaine
Dry eye	Alcohol, nicotine
Mydriasis	Cocaine, methamphetamine, lysergic acid diethylamide, marijuana
Miosis	Heroin
Hippus/indistinct pupillary response	Barbiturates
Non-arteritic optic neuropathy	Nicotine
Age-related macular degeneration	Alcohol, nicotine
Retinal venous occlusion, intraretinal haemorrhage	Cocaine, methamphetamine, opioids
Talc retinopathy	Methamphetamine, heroin
Toxic optic neuropathy	Nicotine, methanol
Nystagmus	Phencyclidine, barbiturates, morphine
Impaired oculomotor function	Cannabinoids
Palinopsia	Lysergic acid diethylamide
Thyroid orbitopathy	Nicotine
Raised intraocular pressure	Smoking nicotine
Decreased intraocular pressure	Cannabinoids

Source: Refs 1, 20, 21, 34, 35, 67

Table II. Summary of effects of abusive drugs on ocular motility and pupil

Ocular adverse effects	Marijuana	Narcotic analgesics	Hallucinogens	CNS depressants	CNS stimulants	Phencyclidine
Pupils	Dilated/normal	Constricted	Dilated	Normal	Dilated	Normal
Pupillary reaction to light	Normal	Slow/none	Normal	Slow	Slow	Normal
HGN	Not present	Not present	Not present	Present	Not present	Present
VGN	Not present	Not present	Not present	Possibly present	Not present	Usually present
Lack of ocular convergence	Present	Not present	Not present	present	Not present	Present

CNS, central nervous system; HGN, horizontal gaze nystagmus; VGN, vertical gaze nystagmus
Source: Refs 54, 62, 66, 103

can be useful indicators of the type of drug the patient is exposed to[103] (Table II).

Conclusion

The use of illicit drugs is a public health concern. Identification of ophthalmic side effects of these drugs is crucial for timely diagnosis and management of these cases. Not only is it essential for the general physician to use the ophthalmic signs for early diagnosis but also for the ophthalmologist to timely refer and treat the patient. Early recognition can go a long way in visual rehabilitation of these patients.

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