Dystonia after Shooting Street Heroin: An Underreported Matter of Concern

Recent World Drug Report mentions opioids as the most harmful drugs in terms of health effects.[1] Neurological sequelae have been reported in a few cases of inhalational heroin use ('chasing the dragon'). It is conjectured that vapours (pyrolysate) produced after heating black market heroin on aluminium foil, rather than pure pharmaceutical diamorphine, are responsible for the brain damage, although the incriminated adulterant has not been isolated till now.[2] The brain pathology associated with such use is spongiform leukoencephalopathy which can lead to long-term consequences or can even be fatal.[3] Structural brain imaging of these spongiform leukoencephalopathy cases showed the involvement of the posterior fossa, pallidum, corpus callosum and supratentorial white matter tract.[4] In this case series, we shall discuss six patients with acute onset transient dystonia following injection heroin use. To the best of our knowledge, no such cases have been described until date in the literature.

CASE SERIES

Case identification was retrospective and based on the patient self-report and informant description. The patients reported to us in a short span of time, i.e., between July to October 2017 and belonged to the same locality or adjacent districts. Here, we include six cases, of which five were inpatients and one outpatient. All the cases were dependent on injection heroin. We assessed the cases with a thorough general physical examination, including neurological examination, relevant investigations and brain magnetic resonance imaging (MRI).

All patients reported experiencing dystonic symptoms within minutes of injecting heroin which developed

to full in a few hours. All patients reported that the heroin they used had a little effect and instead they developed opioid withdrawals in addition to the dystonic symptoms. All the patients approached medical emergency settings for treatment. Two of the patients reported to our emergency department and were examined thoroughly by the internal medicine team, and consultations were taken from the neurology and psychiatry teams also. Routine emergency tests including serum electrolytes were within normal limits. They were administered injection promethazine 50 mg intravenous which gave complete relief in 10–15 min. The remaining four patients received treatment in the emergency departments of other hospitals. One patient reported being given intravenous calcium without any relief, but later, in another hospital, some intravenous drug was given which gave complete relief. For the rest, treatment details were not available, but they received some intravenous drugs that produced complete relief of dystonia within a few minutes. There was no confusion at the time of dystonia, and patients had sufficient awareness of the symptoms. There was no report of tonic-clonic movement or tremors. All patients developed cervical dystonia with variable involvement of the tongue, face and upper limb. The details of demographics, case description and investigations are provided in Table 1.

None of the patients could produce a sample of the heroin used by them before developing dystonia. As per the patients, they had either finished their dose or discarded it after recovering from dystonia. Despite reassurance of confidentiality and anonymity, inability to provide the sample could also be due to fear of being prosecuted. Three of the patients recalled that the texture and colour of the heroin sample were different from usual. Laboratory investigations included complete blood count (CBC), renal function tests (RFT), liver function tests (LFT), fasting blood sugar (FBS) and lipid profile. Except for the reports mentioned in the table, rest of the values were normal in all subjects. Ultrasonography (USG) whole abdomen, chest X-ray and electrocardiography (ECG) were normal in all patients. 3T MRI brain was also normal in all the patients. All the patients received treatment for opioid dependence in our opioid substitution therapy (OST) clinic. They received individualised doses of Tab buprenorphine/naloxone 2.5 mg combination sublingually.

At the time of writing this case series in October 2017, we have not received any new patients with a dystonic reaction after injection heroin use. All patients are in follow-up at our OST clinic without any recurrence of dystonia, and four of them were drug-free at last follow-up.

DISCUSSION

In this cases series, we described acute dystonia in dependent heroin users. There was a clear temporal relationship with the use of injection heroin and the appearance of predominantly localised cervical dystonias. The absence of any other neurological

Table 1: Clinical profile, laboratory investigations and clinical features of dystonia of cases

Clinical profile and laboratory investigations of cases				
Initials, Age (years)	**Other substance dependence**	**Physical illness**	**Psychiatric illness**	**Significant reports**
M 24	Tobacco dependence (ST)	HCV+	None	HCV RNA-TND, LDL-167 IU/L, TG-186 IU/L
H 27	Cannabis and tobacco dependence (ST)	None	None	None
I 26	Tobacco dependence (SLT), alcohol and cannabis dependence currently abstinent	None	None	AST/ALT-86/42 IU/L
A 30	Cannabis, alcohol and tobacco dependence (ST)	Seizure disorder	Bipolar affective disorder	TG-204 IU/L
S 20	Tobacco dependence (ST), cannabis dependence currently abstinent	None	None	None
L 20	Tobacco dependence syndrome (SLT)	None	None	None

Clinical features of dystonia as described by patients and informants							
Initials Age (years)	**Heroin amount used (g)**	**Time to onset of dystonia (minutes)**	**Duration of symptom (hours)**	**Distribution of muscular involvement**			
				Retrocollis/torticollis (side)	**Tongue protrusion**	**Grimacing and trismus**	**Ipsilateral upper limb involvement**
M 24	½	30	3	Y/Y Right	Y	N	Y, wrist flexion
H 27	1	120	2	Y/Y Right	Y	Y	Y, wrist flexion and locked elbow
I 26	½	20	6	Y/Y Left	Y	N	N
A 30	3	30	2	Y/Y Right	Y	N	N
S 20	½	180	1	Y/N Midline	N	N	N
L 20	½	30	3	Y/Y Right	Y	N	Y, wrist flexion and locked elbow

Y=Yes, N=No, ST/SLT=Smoked tobacco/smokeless tobacco, HCV=Hepatitis C virus, LDL=Low Density Cholesterol, TND=Target not detected, ALT=Alanine aminotransferase, AST=Aspartate aminotransferase, TG=Triglycerides

abnormalities in the past, at the time of presentation or in subsequent follow-ups rule out the possibility of any underlying neurological disorder. There was no discernible electrolyte abnormality. None of the patients had received any other drug (over the counter or prescription medication) prior to the onset of dystonia. Hence, temporal connection, specificity of presentation and lack of any other apparent cause strongly suggest the role of injection heroin in producing acute dystonias. Next question is whether dystonias in these patients occurred as a result of direct effect of heroin or due to the adulterants (or cutting agents) mixed with it.

As already mentioned, incidents of acute dystonia following heroin use has never been reported. The reports published so far have described long-lasting features of residual central nervous system (CNS) damage, almost always following inhalational heroin use (rather than injection).[3] Hence, the atypical presentation prompted us to look into the possibility of dystonia induced by adulterants. Reporting of cases in a short span of time, from the same locality, and a similar subjective experience following heroin use too support the adulterant hypothesis. Moreover, being a transit area for South-West Asian heroin, the northern part of India (from where the cases were reported) is no doubt vulnerable to be exposed to the adulterated street heroin as the small-time local drug dealers try to compound their profit.[5] If the adulterant hypothesis is convincing, the nature of the adulterant needs to be speculated.

On the basis of a review of the literature, we speculate the involvement of a couple of offending agents, namely strychnine and chloroquine. A 2005 report by the United Nations Office on Drugs and Crime (UNODC) listed a range of alkaloidal impurities and adulterants isolated from street heroin samples. A thorough check of the list revealed that a probable candidate could be strychnine, which is a non-opiate cutting agent with pharmacological effects. Strychnine blocks the inhibitory action of glycine at interneuron-motor axon synapses and causes exaggerated motor activity.[6] In 1974, analyses of street heroin samples from Amsterdam were reported to be containing strychnine. Though the samples contained less than the lethal dose, low dose strychnine can produce dystonic reactions.[7,8] In another UNODC report (2009), chloroquine was identified as one of the cutting agents present in heroin manufactured in Afghanistan.[9] Though chloroquine is considered a non-toxic drug, there are five reported cases of chloroquine-induced extrapyramidal symptoms, including cervical dystonias from India. The exact mechanism of chloroquine-induced dystonia has not been identified, but chloroquine is believed to cause an imbalance in the neurochemical control of psychomotor activity in the basal ganglia.[10] So, chloroquine could be the second candidate which can produce dystonia.

A major limitation is not being able to gather any sample of the used street heroin. Hence, it is impossible to assert about the chemical nature of the adulterant. There are several learning points. In addition to obvious dangers of heroin, the adulterants or cutting agents too are a matter of real concern. We also reiterate the need for improved forensic capacities to identify specific adulterants and periodic monitoring for the level and nature of impurities. Like the United States had a Drug Abuse Warning Network (DAWN) been present in India, a detailed account of the source, nature, cause and extent of these events would have been generated, which in a way would have helped in shaping a public health response.

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Conflicts of interest
There are no conflicts of interest.

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