Catatonia as a Presenting Feature in a Case of Alcohol Withdrawal: Is There a Causal Link?

Sir,

Catatonia is a neuropsychiatric condition characterized by alterations in motor behavior, thought, affect, and vigilance.[1] Originally conceptualized by Kahlbaum, it is found to occur in a variety of medical and psychiatric conditions.[1] Among substances of abuse, benzodiazepine and alcohol withdrawal have been reported to present rarely with catatonia, both in rodent models and humans.[2–4] We report another case of catatonia that occurred during alcohol withdrawal and discuss the possible etiological link.

A 35-year-old Hindu married male, hailing from a rural background, premorbidly well-adjusted, with no significant family history, was brought by his brother who reported sudden onset, 2 days ago, of inability to talk and mimicking actions performed by others, along with insomnia, restlessness, and generalized tremulousness. The patient was otherwise able to interact with his immediate environment. He used to consume country-made alcohol (approximately 250–500 mL/day) for the last 4 years in a near-daily pattern, which he stopped 3 days back. He had initially started to drink once a week, but gradually developed tolerance and loss of control to his drinking pattern along with previous experiences of distressing withdrawals on abstaining from alcohol. He had no concomitant evidence of a mood disorder or a nonaffective psychosis, and no history of fever, head trauma, or use of other psychoactive substances at the index presentation. He had no past complicated withdrawal, no previous treatments for alcohol use, and no comorbid medical diagnosis.

Physical examination revealed fine bilateral tremors of outstretched hands, tachycardia, and increased perspiration, with a Clinical Institute Withdrawal Assessment of Alcohol Scale, Revised (CIWA-Ar) score of 12.[5] On mental status examination, he was found to be completely mute, with an indifferent affect and with behavior suggestive of negativism and echopraxia (Bush–Francis Catatonia Rating Scale [BFCRS][6] score of 16). There was no hallucinatory behavior (e.g., solitary talk), and his thought and perception were difficult to assess because of mutism. He appeared to have adequate comprehension of his personal and immediate environment and orientation on behavioral observation.

He was provisionally diagnosed as a case of alcohol withdrawal with catatonia according to the *Diagnostic and Statistical Manual for Mental Disorders* (*DSM-5*), with a differential of alcohol-induced psychosis.[7] On admission, he was treated with lorazepam 6 mg/day orally in divided doses, which was tapered later over 7 days, based on his withdrawal symptoms, along with parenteral thiamine as per hospital protocol. Routine blood investigations (complete hemogram; liver, renal, and thyroid function tests; and serum electrolytes) and brain imaging (magnetic resonance imaging) were all found out to be normal. His catatonia dramatically resolved (BFCRS score 0) on the next day of starting lorazepam. After detoxification (CIWA-Ar score 3), his treatment proceeded in lines of pharmacological and non-pharmacological management to maintain abstinence.

At index presentation, the patient developed catatonia after stopping alcohol intake. Other possible organic causes for catatonia, including dyselectrolytemia, structural brain lesions, infections, epilepsy, parkinsonism, and psychiatric conditions, such as mood disorders, psychosis, and drug overdose, were ruled out. The temporal relation between abrupt discontinuation of alcohol and the onset of catatonia (in the absence of delirium) and its resolution on detoxification with benzodiazepine all point to this catatonia to be an alcohol-withdrawal-related presentation.

There are reports of catatonia in alcohol withdrawal states, but what makes the current report stand out is the predominance of echopraxia, which is rarely reported in this context. Muralidharan et al. reported catatonia (negativism, stupor, and psychomotor agitation followed by retardation) in the absence of delirium, in a patient, 3 days after stopping alcohol, which remitted within 3 days of treating the withdrawal state.[4] Narayanaswamy et al. reported catatonia (with echopraxia as one of the presentations) that occurred 2 days after abrupt cessation of alcohol use, which was treated with lorazepam, which resulted in complete resolution of catatonia within 1 day.[3] Basu et al. reported catatonia (ambitendency, negativism, stereotypy, and posturing), which occurred after 3 days of complete abstinence from alcohol and benzodiazepine, both of which were taken in a dependence pattern. The catatonia completely resolved after adequately treating the withdrawal state.[8]

Neurobiologically, an abnormality in the GABA-ergic neuromodulatory system can be proposed, considering its role in both catatonia and alcohol withdrawal state. Catatonia is proposed to occur due to "top-down" alterations of the basal ganglia structures by GABA-mediated orbitofrontal cortex deficits.[9] The anteroinferior part of the frontal lobe (a part of the mirror neuron system) is also implicated for echo phenomena, such as echopraxia, as in the index patient.[10] We propose that abnormalities in these frontal lobe areas, on a background of an altered GABA-ergic neurotransmission, along with glutamatergic excitotoxicity due to alcohol withdrawal state, have led to catatonia (with predominant echopraxia) in our case. We also exercised caution in choosing an anti-craving agent in this patient because agents such as disulfiram and baclofen are known to cause catatonia and psychosis and might exacerbate his psychosis, considering his background vulnerability to psychosis.[11,12] Whatever the cause may be, this report points to the need to emphasize that clinicians should not consider catatonia to occur only in the context of psychiatric disorders—it can occur in a gamut of organic conditions and alcohol

withdrawal states too. These point to the need for further studies in understanding the neurobiological underpinnings of catatonia in substance use disorders that will facilitate a well-concerted management.

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Club Drugs in India: An Analysis of Newspaper Reports

Dear Sir,

Substance use and substance use related disorders posit a unique challenge in India given a large and growing vulnerable population, socio-economic transitions leading to changes in social dynamics and attributes, and geographical location making it a potential transit route.[1,2] Though there are well-established substances that have been in regular use among the Indian population, newer substances have also made inroads, especially in some populations.[3] In this context, club drugs represent a growing potential issue that primarily affects the younger population and may become a public health challenge over time.

Club drugs are psychoactive drugs that are usually used by young adults and adolescents at bars, nightclubs, concerts, and parties, to reduce fatigue and enhance the pleasure of the "clubbing" scene.[4] Club drug use has been associated with serious physical and psychiatric disorders, risky sexual behaviors, HIV infection, violence, and criminality.[5] Since users of club drugs do not generally need to seek treatment at healthcare facilities, the use is often concealed, and the consequences are under the surface. Population surveys generally are not able to reach the users of these substances effectively. Hence, newspaper reports are often the sources of some usable information.

In view of the lack of literature from India, we aimed to synthesize information from the newspaper reports on club drugs. Google News database was used to identify relevant online English language regional or national newspaper reports over a two year period (January 1, 2017 to December 31, 2018). The search keywords included "India" (and "Delhi" OR "Mumbai" OR "Kolkata" OR "Chennai" OR "Hyderabad" OR