Case report

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Frozen after the smoke: A case report on cannabis induced catatonia

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Abstract

Catatonia is a syndrome of psychomotor disturbance occurring in association with various psychiatric, medical, or substance-related conditions. In this report we describe a 31-year-old male with no past psychiatric history who presented with features of catatonia in the context of heavy cannabis use with recent escalation. This case highlights cannabis as a potential precipitant of catatonia and emphasizes the importance of considering substance use in the differential diagnosis of acute catatonic presentations. To our knowledge, this is the first reported case of cannabis-induced catatonia from Sri Lanka.

Keywords: catatonia, cannabis-induced, case report

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Introduction

Catatonia is a syndrome of psychomotor disturbances characterized by the presence of several increased, decreased, or abnormal psychomotor activity (1). It comprises a constellation of motor, behavioural, and autonomic abnormalities including stupor, mutism, posturing, waxy flexibility, and echo-phenomena. Although catatonia was historically associated with schizophrenia it is now being described across a wide range of psychiatric and medical disorders including psychotic and mood disorders, various neurological and metabolic conditions and related to psychoactive substance use. ICD 11 recognizes catatonia as a separate disorder and further categorizes it as catatonia associated with another mental disorder, catatonia induced by substances or medications, secondary catatonia syndrome, and catatonia unspecified (1). A systematic review conducted in 2017 revealed that the mean prevalence of catatonia was 9% (2). Timely recognition and management is crucial as it is a potentially life-threatening condition which can be treated efficiently with benzodiazepines and electroconvulsive therapy (3). Although less frequently reported, cannabis has emerged as a potential precipitant of catatonia. Several case reports have documented cannabis-associated catatonic presentations. A review done in 2021 has identified 11 publications describing 14 cases of catatonia precipitated by cannabis use and has concluded that cannabis may be related to catatonia in a manner similar to its established association with psychosis (4).

In Sri Lanka, psychoactive substance use is an increasing public health concern, with a documented rise in cannabis and synthetic drug use in recent years and with cannabis being the most used illicit substance (5). Despite this, a review of published literature revealed no prior case reports from Sri Lanka describing cannabis-induced catatonia. Hence, this makes the present case noteworthy, as it raises awareness among local clinicians about cannabis as a potential trigger for catatonia while adding to the global literature.

Case report

Mr. D, a 31-year-old unemployed, single male with no previous contact with mental health services, was referred from a medical ward due to reduced responsiveness of several hours' duration. He had initially presented following an episode of fainting. Upon regaining consciousness, he has remained mute, had no interactions, and refused food.

According to the collateral history from his parents he had completed education up to the Advanced Level and subsequently held several short-term private sector jobs. His most recent employment as a call centre operator ended one month back due to performance-related concerns. Since then, he had been largely confined to his home, with limited social interactions except for occasional visits from a friend. Over the preceding week, his parents observed increasing social withdrawal,

reduced speech, poor sleep with wandering off at night, and intermittent abnormal posturing. They denied observing any hallucinatory behaviours or aggression. Parents were not aware of any psychoactive substance use other than nicotine.

Examination revealed complete mutism, posturing, catalepsy, and withdrawal. The Bush-Francis Catatonia Rating Scale (BFCRS) score was 14/69. Routine investigations including full blood count, liver and renal function tests, serum electrolytes, C-reactive protein, non-contrast CT brain, and EEG were normal. Urine drug screen was positive for cannabis.

A diagnosis of catatonia was made, and he was taken over to the psychiatric ward after excluding medical causes. As lorazepam was unavailable, clonazepam 0.5 mg twice daily dose was initiated. However, due to persistent refusal of meals and medication, electro-convulsive therapy (ECT) was commenced on the third day. He demonstrated a rapid response, with improved speech and oral intake after the first session of ECT. A total of seven ECT sessions were administered, with cautious refeeding under consultant nutritionist supervision.

As rapport improved, he disclosed a 5-year history of heavy cannabis use with recent significant increase in quantity of use over the past month. He denied depressive symptoms. Initially, he had difficulty expressing his thoughts, but he later described ideas of reference and thought echo. He was in the precontemplation stage regarding cannabis use.

He was treated with olanzapine, titrated to 20 mg nocte to which his psychotic symptoms responded. Motivational interviewing was continued throughout the ward stay. Following a 22-day inpatient stay, he was discharged to a substance rehabilitation centre for ongoing care.

Discussion

Catatonia is increasingly recognized as a syndrome occurring across a spectrum of psychiatric and medical disorders, and psychoactive substance use has emerged as a significant but under-recognized precipitant with the increasing substance use. Studies examining catatonic episodes related to substance use have found cannabis to be most frequently linked to catatonia among the psychoactive substances (6).

The pathophysiology of catatonia remains incompletely understood, but dysregulation of γ-aminobutyric acid (GABA)-A, glutamate, and dopamine neurotransmitter systems is implicated (7). Dysregulation of the endo-cannabinoid system with a reduction in the GABAergic inhibition is one suggested mechanism of catatonia linked with cannabis use (8).

This case highlights the importance of collateral history and routine screening investigations in evaluating catatonia. Despite the absence of a disclosed history of cannabis use, it was detected as routine urine drug screening was carried out due to clinical suspicion. Even though the causal inference is limited as other confounding factors, including concurrent psychiatric vulnerability, cannot be fully excluded, the temporal relationship with heavy cannabis use and absence of alternative precipitants strengthen the association between cannabis use and catatonia in this patient.

Benzodiazepines and electroconvulsive therapy are considered the main treatment modalities of catatonia, regardless of its aetiology (9). Benzodiazepines are the first-line treatment with a 60% to 80% response rate and with lorazepam being used most commonly (9). clonazepam was used for this patient due to the unavailability of lorazepam. Even though most guidelines and literature specifically mention lorazepam as treatment, there are case reports of catatonia successfully being managed with clonazepam and diazepam as well (10). This awareness that other benzodiazepines can be effective helps avoid moving prematurely to ECT and hence is particularly important in resource-limited settings, where lorazepam may be unavailable.

Conclusions

Given the rise in cannabis and other psychoactive substance use locally and globally, this case highlights the importance of considering cannabis as a potential precipitant when evaluating catatonia.

Declaration of Interest

There are no conflicts of interest.

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