Case Report

Catatonia in mixed alcohol and benzodiazepine withdrawal

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ABSTRACT

Catatonia is mostly caused by different neuropsychiatric conditions. We report a case of a 30 year old man suffering from both alcohol and benzodiazepine dependence who exhibited catatonic features soon after stopping the intake of substances. This case will help clinicians to recognize catatonic features within the varied symptomatology of substance withdrawal and thereby helping in its early diagnosis and management.

Key words: Alcohol, benzodiazepine, catatonia, withdrawal

INTRODUCTION

Catatonia is a syndrome of specific motor abnormalities closely associated with disorders in mood, affect, thought and cognition. Its principal signs are mutism, stupor, and posturing.[1] Formulated by Kahlbaum in the nineteenth century, the concept of catatonia had a fluctuating course across the twentieth century–initially disappearing and again being revived lately and is finally posed for a paradigm shift in the twenty-first century.[2] Aetiologically, apart from psychiatric disorders, medical causes like neurological and metabolic diseases are common. Among substances, alcohol and benzodiazepines withdrawal have been reported to be associated with catatonia. Though initially reported among laboratory animals, only a few of such cases have been reported in relation to alcohol use as shown in [Table 1].[3-5] For benzodiazepines, there is more available literatures. Initially, Rosebush *et al*. reported five patients who became catatonic after abrupt withdrawal of benzodiazepines used by them chronically for a period from 6 months to several years.[6] Thereafter, several similar cases of benzodiazepine withdrawal catatonia have been reported and all common benzodiazepines like alprazolam, clonazepam, lorazepam, diazepam, oxazepam, and temazepam have been implicated. We report a patient, who, to the best of our knowledge, is one of the first cases to exhibit catatonic features in relation to mixed alcohol and benzodiazepine dependence and withdrawal.

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CASE REPORT

A 30 year old married male from rural background in northern India, agricultural labourer by occupation, presented with history of alcohol use for last 15 years. He had no past or family history of any neuropsychiatric or major medical illness. Since last 6 years he had been drinking regularly country made liquor daily with history of tolerance, withdrawal, having a strong desire for substance, negligence of alternative pleasures and use despite adequate knowledge about the harm related to substance use leading to a diagnosis of alcohol dependence as per International Classification of Diseases,

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Table 1: Alcohol withdrawal catatonia

Author	Age/ gender	Duration of alcohol dependence (years)	Clinical features
Muralidharan *et al.*[3]	35 years male	5	72 hours after cessation he developed signs of negativism, mutism and psychomotor agitation lasting 2 days followed by psychomotor retardation, stereotypies and retardation over next 2 days
Narayanswamy *et al.*[4]	30 years male	15	After 2 days of abrupt cessation, he developed symptoms of decreased sleep and psychomotor agitation lasting for 1 day, followed by stupor, mutism, negativism, rigidity, posturing, echopraxia and autonomic disturbances. (Bush Francis catatonia rating, BFCRS score of 18)
Geoffroy *et al.*[5]	65-year-old woman	15	Episodes characterised by psychomotor agitation, disorientation in time and space, fluctuating mutism, akinesia, negativism, stereotypy, verbigeration, rigidity, posturing and autonomic disturbances (BFCRS score of 43/69) several times during alcohol withdrawal treatment

BFCRS=Bush francis catatonia rating scale

10th revision (ICD-10). Three years ago to control excessive alcohol use, his wife gave disulfiram tablets surreptitiously for few days. He experienced a few instances of mild DER (disulfiram ethanol reaction) and thereafter in lieu of alcohol he started consuming alprazolam (0.5 mg) tablets on and off to enjoy its pleasurable experience and subsequently developed dependence on these tablets. Since last 3 months he had been taking ten tablets of 0.5 mg alprazolam everyday along with frequent alcohol consumption (about once or twice weekly).

Due to his increased substance use and inability to quit despite several attempts he was admitted in a private unauthorised de-addiction centre where he remained forcefully abstinent for about a week. There he developed fearfulness, suspiciousness with complaints of transient auditory hallucinations for which he was referred to our hospital. No records were available related to his stay in the de-addiction centre. On presentation he was oriented though fearful. No history suggestive of any organic condition like seizures, fluctuating sensorium, and prolonged fevers was obtained and a general physical examination including neurological examination was normal. Routine investigations were advised and a provisional diagnosis of substance induced psychotic disorder was considered. Patient was advised in-patient care for observation and monitoring. However, due to some personal reasons his accompanying family members declined admission. So he was advised oral tablet olanzapine 10 mg on out-patient basis which he took for 15 days and there was complete remission of symptoms. Thereafter, he left medications, dropped out of treatment, restarted consumption of alprazolam tablets (5 mg/d) and continued it for one and half months. Six days prior to presentation in our clinic he stopped taking alprazolam tablets and started consuming alcohol–12–15 units of country-made liquor every day. He continued alcohol consumption for 3 days and then he stopped suddenly under family pressure. There was no history of any other substance use in the recent past.

When he was presented to our clinic he was completely abstinent from all substances for 3 days as he was under constant supervision by family members. At the time of presentation he was fearful, would speak minimally, be excited at times, show unprovoked aggression towards attendants, exhibit staring, ambitendency (motorically 'stuck' in hesitant, indecisive movements), motor stereotypy (repetitive non-goal directed motor activity), and posturing. He would have sleeplessness and exhibit negativism, thereby refusing food or water intake. He was conscious, oriented, and a general physical examination including a neurological examination was non-contributory except some withdrawal features (Clinical Institute for withdrawal of alcohol rating scale, CIWA, score on day 1 was 18). His routine investigations like hemogram, blood sugar, renal function, liver function and electrolytes were within normal limits. A toxicological screen, thyroid profile, vitamin B12, folic acid, computed tomography (CT) scan (head), electroencephalography (EEG), HIV, and Venereal Disease Research Laboratory (VDRL) were planned of which last two were within normal limits whereas the rest could not be done due to lack of resources. Bush Francis Catatonia Rating Scale (BFCRS) was applied, score was found to be 23 and parenteral lorazepam on the first day showed spectacular response and the score came down significantly to below 5 [Table 2]. For detoxification he was started on oral clonazepam 5 mg/d as equivalent dose of the amount of alprazolam last used. Along with this, other supportive treatment like parenteral multivitamins (including thiamine 100 mg) and adequate nutrition and hydration was ensured. Throughout the next 2 days his BFCRS was consistently low as shown in [Table 2] as also his withdrawal symptoms [Table 3]. From the fourth day of admission the patient was asymptomatic. There were no physical or psychological symptoms and he regained full socio-occupational functioning with no cognitive deficits. Clonazepam was gradually tapered and stopped within a week. However, he was very poorly motivated to quit substances and his motivation could not be improved even with regular sessions. He was discharged after 10 days of inpatient care. But he relapsed within a month of discharge and followedup only once–thereafter dropping out of treatment. During the month when he was abstinent he did not exhibit any of his initial presenting symptoms.

Table 2: Bush francis catatonia rating scale scores of patient

	Day 1	Day 2	Day 3
Excitement	2	1	0
Immobility/stupor	0	0	0
Mutism	2	0	0
Staring	3	2	1
Posturing/catalepsy	2	2	0
Grimacing	0	0	0
Echopraxia/echolalia	0	0	0
Stereotypy	2	0	0
Mannerisms	0	0	0
Verbigeration	0	1	0
Rigidity	0	0	0
Negativism	1	0	1
Waxy flexibility	0	0	0
Withdrawal	2	0	0
Impulsivity	2	0	0
Automatic obedience	0	0	0
Mitgehen	0	0	0
Gegenhalten	0	0	0
Ambitendency	3	0	0
Grasp reflex	0	0	0
Perseveration	0	0	0
Combativeness	1	0	0
Autonomic abnormality	3	0	0
Total score	23	6	2

Table 3: Clinical institute withdrawal assessment of alcohol scale, revised

	Day 1	Day 2	Day 3
Nausea/vomiting	1	0	0
Tactile disturbance	0	0	0
Tremors	4	2	0
Auditory disturbance	3	0	0
Paroxysomal sweats	0	0	0
Visual disturbance	0	0	0
Anxiety	5	2	2
Headache	0	0	0
Agitation	5	2	0
Orientation and clouding of sensorium	1	0	0
Total score	18	6	2

DISCUSSION

This case brings us at the crossroads of a number of diagnoses. As indicated in [Table 2], the patient evidently had catatonia as he exhibited more than two of its classic features namely mutism, stupor, posturing, automatic obedience, and stereotypy for more than one day.[7] Catatonia is commonly expressed in patients with structural neurologic conditions, abnormal metabolic states, epilepsy, parkinson's disease, and different psychiatric conditions like mood disorders, psychosis, pervasive developmental disorder and different drug related conditions.[1] Lack of any pointers towards organicity in history, normal detailed neurological examination, and routine investigations ruled out most of the common organic conditions.

After ruling out medical causes of catatonia, we considered the psychiatric conditions. As per ICD-10, catatonia is considered a psychotic symptom.[8] Mood disorder and schizophrenia are its common causes. Since he did not exhibit any mood or schizophrenic symptoms we considered the conditions which might give rise to psychotic disorders in relation to alcohol/benzodiazepine use–the most relevant are substance withdrawal, substance induced psychotic disorder, Wernicke's, and related encephalopathy due to vitamin deficiencies, and alcoholic dementia.[9] In such a situation the key to successful differential diagnosis is the sensorium–clear consciousness ruled out delirium tremens and other organic encephalopathy.[10] He developed only transient nonprogressive cognitive deficits which were totally reversible thereby excluding dementia as a possibility. This leaves us with only substance induced psychotic disorder and substance withdrawal as possible diagnosis. Now, since he did not exhibit any other psychotic features like hallucinations and delusions, the question arises whether the catatonia is a part of the substance induced psychotic disorder or a component of the withdrawal syndrome as both are temporally related and both would respond to benzodiazepines. Since the classical description of alcohol or benzodiazepine withdrawal does not consist of catatonia, we diagnosed this as substance induced psychotic disorder.

After establishing the diagnosis we need to address the neurobiology underlying this clinical condition. This exuberance of catatonia in organic conditions has led to the motor circuitry dysfunction model which proposes important role of frontal lobe basal ganglia circuitry abnormalities in the mechanism of catatonia. Other important associated portions involved are thalamic, parietal lobe, cerebellar, or limbic regions.[11] Apart from this, epilepsy, genetic, endocrine, and immune models find mention in literatures. Whatever be the case, the aetiological model involved the final common pathway is mediated by the neurotransmitters dopamine and gamma-aminobutyric acid (GABA).[1] Since both alcohol and benzodiazepine influence the GABA-ergic pathway, they have some relationship with the development of catatonia. It is a well known that repeated withdrawal and binge drinking makes an already neurochemically altered 'alcoholic' brain vulnerable to different complicated withdrawal.[12] The episode of possible withdrawal psychosis previously, made this patient vulnerable to a similar episode. Thereafter, in the current episode the

sudden cessation of benzodiazepine might have made his brain more susceptible to the alcohol withdrawal related catatonia. The associated benzodiazepine use might have added more to the complications.

Viewed from an epidemiological perspective, catatonia might not be a very uncommon disorder restricted to schizophrenia only and it may be more frequent than presently reported in alcohol or benzodiazepine withdrawal where the symptoms of delirium, agitation, anxiety masks its features.[13] Hence, this report will caution clinicians against underestimating catatonia in nonpsychiatric medical and substance related conditions. In this respect the caveat remains that due to scarcity of resources we could not perform the range of investigations necessary to rule out all possible medical causes of catatonia particularly any post ictal catatonia related to substance withdrawal; however, lack of other organic signs and symptoms and continued remission from catatonic features even after stopping benzodiazepines almost ruled out underlying medical causes and established the catatonia to be related to substance use only.

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