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Research Paper

Unusual presentation of Cannabis use related Catatonic Schizophrenia and role of ECT in its management: A case report

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ABSTRACT:

- *Introduction: An acute and usually transient psychosis has been associated with prolonged cannabis use. Catatonic schizophrenia is a subtype of schizophrenia characterized by a psychomotor disturbance.*
- *Objectives: To discuss about the atypical presentation and difficulty in management of cannabis use related catatonic schizophrenia. To highlight on the use of ECT in the treatment outcome.*
- *Background: Cannabis is known to be associated with psychosis as a predisposing, precipitating and perpetuating factor. It also has a varied polymorphic presentation. One of the difficult case presentations for management is catatonia.*
- *Methodology: A 25 years single male belonging to Hindu family of rural background having a history of cannabis use since adolescence for past 8 years in a dependent pattern. He presented with abrupt onset continuous deteriorating course of 2month precipitated by increased use of cannabis, symptoms comprising of hallucinations, bizarre delusion and catatonia requiring hospitalization. He was given adequate trials with both typical and atypical antipsychotic but responded after MECT augmentation: - 28 therapeutic followed by maintenance MECTs for past 1year (48 total).*
- *Results: This case is reflexion of varied presentation of the cannabis related Schizophrenia and management difficulty; role of MECT in therapeutic and maintenance phase of treatment.*
- *Conclusion: Cannabis use is associated with poor prognostic presentation and difficulty in management; although ECT is a promising therapeutic measure in patients with Cannabis use related Catatonic Schizophrenia.*

***Key Words**: Cannabis, Catatonia, Schizophrenia, MECT*

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I. INTRODUCTION:

Cannabis is a psychoactive drug derived from the *Cannabis* plant used for medical or recreational purpose and its main psychoactive part is tetrahydrocannabinol (THC). Regular cannabis use and psychotic disorders (such as schizophrenia) are associated in the general population and heavy cannabis users are over-represented among new cases of schizophrenia[1]. Basu et al[2] has proposed four hypotheses depending upon the nature of the relationship between cannabis use and psychosis. That are: - cannabis use causes psychosis, cannabis use precipitates psychosis among vulnerable individuals, cannabis use worsens the prognosis of persons with schizophrenia and regular cannabis use is more likely among persons with psychosis.

A 15-year prospective investigation of cannabis use and schizophrenia in 50,465 Swedish conscripts found that those who had tried cannabis by age 18 were 2.4 times more likely to be diagnosed with schizophrenia than those who had not used cannabis[3]. A Dunedin cohort study reported that at age 18years or later showed only a small, non-significant increase in the risk of schizophrenia-like psychosis by age 26, but the risk increased fourfold among those starting at age 15 or earlier (Arseneaultet al., 2002)[4].

Case presentation:

One and a half year back, a 25-year-old single male of rural background literate, labourer living in a Hindu nuclear family was brought by his mother to the emergency department of a tertiary care hospital with

altered mental status and risk of harm to self. As reported by his mother he had an acute onset continuous deteriorating illness of 2 months. The symptoms comprised of- suspiciousness towards family member, hearing voices commanding and discussing about him and muttering to self, disorganised behaviour in form of undressing self, eating garbage, stool and drinking his own urine, saying that he could give birth to children despite being male by pressing button of stomach, poor self-care, poor social interaction and emotional expression with significant deterioration in occupational and biological functions.

S.NO.	DRUG	DOSE (mg)	DURATION (weeks)	RESPONSE	SIDE EFFECT
1	Haloperidol	5-40	2-3	Poor	EPS
2	Risperidone	2-8	2	Poor	EPS
3	Olanzapine	10-30	2	Initial partial response later developed catatonia	-

There was a history of cannabis and nicotine use for past 8 years. Cannabis use in form of Bhang and Ganja which increased in amount and frequency in the past 2-3 years and significantly increased for 4-5 months; current pattern of daily use of 3-4 joints per day and 18-20 bidi per day. There was craving, withdrawal symptoms (irritability, restlessness and weakness) and significant decline in other previously pleasurable activities. The last intake was 15 days back prior admission.

Patient was well adjusted pre-morbidly, had no past medical and psychiatric illness. The family history suggestive of psychotic illness in father of patient that lead to missing from home. His mother is the only primary caregiver for the patient

Clinical course during hospital stay:

Patient was admitted with consent of his mother as a nominated representative. Detail general and systemic examination showed no abnormality. On cross-sectional assessment he was ill kempt, poorly-groomed, withdrawn, not spontaneously interactive would maintain eye contact briefly though rapport was established, normal psychomotor activity and speech, blunt, restricted, non-reactive and inappropriate, though subjectively normal affect. He had bizarre delusion, delusion of persecution and thought broadcast; 2nd and 3rd person auditory hallucination and absent insight (Grade-1).

His routine investigation- hemogram, LFT, KFT, serum electrolyte, blood sugar levels- normal range; urine drug screening- negative and NCCT head had no abnormality detected. Patient was diagnosed as: Cannabis dependence syndrome, Tobacco dependence syndrome with Schizophrenia.

Patient was started with antipsychotics (Haloperidol, Risperidone) on which he had poor response. Later, oral Olanzapine was started. The response noticed was as shown in table 1.

During hospital stay, after one-and-a-half-month patient showed deterioration in form of development of catatonic symptoms: mutism, rigidity, posturing, negativism and staring (BFCRS=14). On evaluation, considering no autonomic instability, raised temperature, normal neutrophil and CPK levels, Malignant catatonia was ruled out.

Hence treatment was augmented with 2mg i.v. Lorazepam T.D.S dose over 72 hours catatonic signs and symptoms started resolving but there was exacerbation of positive and negative symptoms.

Thus, the patient was started with MECT and was response was noted as shown in table 2. During the initial phase of MECT there were recurrence of catatonic symptoms on MECT free days which slowly improved though brief period of 1-2 hours of relapse would be seen. Considering this presentation, patient was later continued with maintenance MECT.

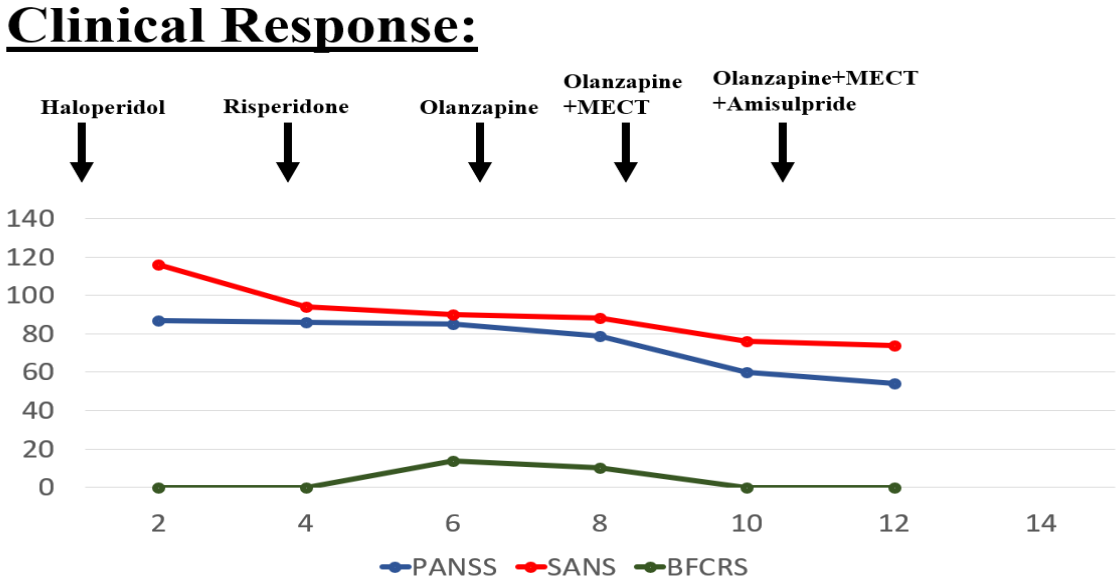
Table 1: Treatment and response with Antipsychotic drugs:

Table 2: Clinical response with MECT:

NO. OF MECT	RESPONSE / OUTCOME
0-7	Catatonic symptoms improving (BFCRS=10), other positive and negative symptoms remaining
13	Improvement in catatonia (BFCRS=0) and positive symptoms
13-15	Spacing of MECT started. Relapse in catatonia after a week MECT was withheld MECT restarted

22	Good response; negative symptoms present. Brief period of relapse of catatonia

Figure 1: Clinical response with treatment:



Follow-up and Outcome:

Patient was on a regular follow up in OPD. He received weekly MECT for a month and fortnightly MECT for 6 month and on monthly Maintenance MECT for past 5 months (48th MECT). Patient has no positive and catatonic symptoms, though negative symptoms in form of asociality and apathy are persistent at a plateau of response.

II. DISCUSSION:

Cannabis produces a psychosis with predominantly affective features and more of positive symptoms, violence and excitement and show polymorphic presentation of acute onset and transient course[6,7,8]. A possible explanation for this is that the brain is still developing when teenagers start cannabis[9]. Exposing the juvenile brain to cannabis might permanently impair the endocannabinoid system, and impact adversely on brain and neurotransmitter function (Volkow et al., 2016)[10], so was a depiction in our case.

Though in our case it had acute onset polymorphic picture continuous and gradually deteriorating course. Our case showed poor response to antipsychotic drugs and later good response to therapeutic as well as maintenance MECT.

III. CONCLUSION:

Unlike usual presentation of cannabis related schizophrenia our case had a varied presentation comprising of positive, negative and catatonic symptoms; and response for MECT with antipsychotic drugs over a longer course of time with intact cognition.

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Declaration of Interest: None.

Abbreviation:

BFCRS: Bush Francis Catatonia Rating Scale
ECT: Electro-Convulsive Therapy
EPS: Extra Pyramidal Side-effects
MECT: Modified Electro-Convulsive Therapy
PANSS: Positive and Negative Symptoms of Schizophrenia
SANS: Scale for the Assessment of Negative Symptoms
THC: Tetrahydrocannabinol

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