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Cannabis Induced Periodic Catatonia: A Case Report

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Abstract Catatonia is a syndrome of specific motor abnormalities closely associated with disorders in mood, affect, thought and cognition. The principal signs of the disorder are mutism, immobility, negativism, posturing, stereotypy and echo phenomena. Catatonia is commonly seen in various psychiatric disorders, neurological disorders and certain medical conditions. It has also been reported in individuals with substance withdrawal. But we are presenting the case of a patient with cannabis dependence, who presented with symptoms of catatonia preceded by an increase in the amount of cannabis intake and resolution of the catatonia when he abstained from the substance. Literature review did not show any case revealing association between cannabis to catatonia.

Keywords Cannabis · Catatonia

Cannabis is obtained from cannabis sativa and cannabis americana. Different preparations of this plant like marihuana, hashish, charas, ghanja, bhang, kef, and dagga are available in India. There are various pharmacologically active compounds in cannabis, important being the oily cannabinoids, the chief compound being 9-δ-tetrahydrocannabinol [9-δ-THC]. The mechanism of action of cannabinoids is by inhibition of adenylate cyclase activity centrally. It produces euphoriant effect on mood, perceptual changes, impairs cognitive and psychomotor performance.

Catatonia has been variously described as a disease (Kahlbaum 1874), a syndrome (Gelenberg 1976), a nonspecific symptom (Peralta et al. 1997), a subtype of schizophrenia (Kraepelin 1919) and as a symptom seen more commonly in mood disorders (Abrams and

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Taylor 1976). Catatonia is known to occur in various psychiatric disorders, neurological disorders and general medical conditions and substance dependence. Catatonia is well known to occur in substance withdrawal, but there is no report of cannabis induced catatonia till now in literature.

Case Summary

A 30 year old patient with cannabis dependence presented to de-addiction OPD of IHBAS with mutism, negativism, posturing and poor oral intake for last 10 days. The patient's vitals were stable with no obvious signs of any systemic disease. There was history of smoking cannabis in increased quantities over the last 3 weeks. All investigations including X-ray chest, ECG, complete hemogram, liver and renal function tests, and serum electrolytes were sought immediately and were found to be within normal limits. Fundoscopy and MRI were done and did not reveal any abnormality. There was history of five previous episodes of catatonia during last seven years with no psychiatric symptom other then this. Each of these episodes according to the family members was preceded by an increase in the cannabis intake by the patient. Some of these episodes lasted up to 2 months and on all occasion, patient showed improvement with medication and abstinence from cannabis. The patient was admitted and supportive management was started. He was started on oral lorazepam 4 mg/day and his catatonic symptoms resolved after 2 days. Serial MSE and ward observation did not reveal any psychopathology. The patient was discharged after complete resolution of catatonia. He continues to follow up in OPD.

Discussion

Initially conceptualized as a subtype of schizophrenia, catatonia is now recognized to occur not only with other psychiatric conditions like mood disorders, drug intoxication and withdrawal but also with medical conditions like epilepsy, parkinson disease, abnormal metabolic states and structural neurological conditions. Different pathophysiological mechanisms have been described for catatonia namely deficits in fetal cortical development, dopaminergic blockade, glutaminergic dysfunction, dysfunction in neurotransmission of noradrenaline and serotonin. Fairbairn and Pickens (1979, 1980) suggested that THC increased the availability of arachidonic acid, possibly from the gut, with a subsequent increase in the level of prostaglandins. They postulated that PGE2 was involved with the catatonia produced by THC. Coupar and Taylor (1982) injected tethahydrocannabinol in rats resulting in catatonia. The results of the methods employed in this study did not support the suggestions that THC increases the level of PGE2-like material. However, they did not exclude the possibility that prostaglandins were involved in the catatonia produced by THC. Association of increased cannabis use and catatonia across several episodes was present in our patient, the etiology of which remains hypothetical. In the end, we would like to highlight the fact that cannabis intoxication can be one of the possible causes of catatonia and should be kept in mind while treating patients of catatonia.

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