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Case Report

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An Atypical Case of Catatonia, Abnormal Gait and Psychosis in an Adolescent with Chronic Cannabis Use

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ABSTRACT

An association between cannabis and psychosis is well established. Cannabis use is known to precipitate schizophrenia in vulnerable individuals and it can exacerbate existing psychosis. However, there is a paucity of knowledge about the emergence of catatonia and abnormal gait associated with cannabis use. History of cannabis use is associated with long-lasting changes in open-chain elements of walking gait. Cannabinoid receptors are located in movement-related brain regions and cannabis use is suggested to affect gait. This report is a unique case of a 16-year-old male with no known past medical history, no past psychiatric history, with family history significant for psychosis who presented to the hospital for bizarre, disorganized behavior, and decreased oral intake following heavy cannabis use over the past three months in the context of a break-up. Upon arrival, he was found to be in a catatonic state with an impaired gait. As catatonia resolved with treatment, he was able to speak and the psychosis surfaced which was addressed with antipsychotics. This case report highlights an atypical but serious clinical picture observed to be precipitated by chronic, heavy cannabis use.

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Introduction

Cannabis is one of the most commonly used drugs among adolescents and young adults. Recreational and medicinal cannabis use has gained widespread popularity in recent years with the legalization of cannabis use in various states of the USA. Some consider it a "safe" drug and some perceive it as a "soft" drug but evidence-based data suggests otherwise. Different psychiatric diseases including schizophrenia, bipolar disorder, anxiety, and suicidality are found to be higher in cannabis users compared to non-users [1]. Cannabis can cause an acute psychotic reaction at high doses in non-schizophrenic cannabis users and worsen the illness in schizophrenic patients [2]. We understand very little about the earliest effects of cannabis use because most research is conducted in adults with a heavy pattern of lifetime use [3]. There is sparse research related to its use in the child and adolescent population. Recent brain imaging studies have found neuroanatomic alterations across brain regions with high cannabinoid receptor type 1 mostly characterized by a decrease in volume of the hippocampus, amygdala and prefrontal cortex. These alterations were more pronounced in people consuming higher doses at an earlier age [4,5]. Catatonia is seen in various psychiatric, medical, neurological disorders and even with substance intoxication/withdrawal. Cannabis is a serious public health concern our society is facing currently [6]. This article emphasizes the psychotic potentiality of cannabis and its hazardous consequences, especially in the vulnerable young population.

Case Description

Mr. "R" is a 16-year-old Hispanic male, with no known past medical and past psychiatric histories, no prior suicide attempts or self-injurious behavior who presented to the hospital brought in by his mother for bizarre, disorganized behavior, staying to himself with decreased oral intake for the past few days. Per patient's mother, he was well until 3 weeks prior to admission when he started "acting very strange". He would stare at the walls for hours, laugh at her at times, and would not interact appropriately. On one occasion, he had walked out of the house in the middle of the night wearing pajamas without shoes and started knocking on neighbor's doors because he wanted to read the Bible to them and became sad when nobody opened doors. Mother reported family history of a psychotic disorder in his cousin. She informed that patient had recently completed summer school for 11th grade after failing a class and failing to take regents exam. The patient was initially admitted to inpatient pediatrics for poor oral intake and was transferred to our inpatient adolescent psychiatric unit after being medically stabilized. Urine toxicology screen on admission was positive for cannabinoids and head CT was negative for intracranial pathology. Rest of the routine workup on admission was unremarkable. Patient presented to our unit in a catatonic state with mutism, immobility, rigid posturing, grimacing, waxy flexibility, and catalepsy. On the first encounter, his left lower extremity was contracted with foot dorsiflexed, and left upper extremity was rigidly extended in the air for several minutes.

He kept his eyes closed during evaluation, was guarded, and minimally verbal with sporadic incoherent mumbling in response to questions. He was observed to have slowed thinking with possible thought blocking and poverty of content. He scored high (31/69) on the Bush-Francis Catatonia Rating scale (BFCRS) which is a tool routinely used in clinical practice to screen and diagnose catatonia [7].

Treatment/ Management

The patient was placed on continuous observation for catatonia and disorganized behavior. To address catatonia, he was started on lorazepam 1 mg/day. Over the next day, he was noted to have unsteady, dragging gait without any musculoskeletal abnormality. Neurology was consulted to assess gait and his neurological exam was unremarkable. All neurological workup including ESR, CRP, TSH, creatine kinase, RPR, EBV, Lyme titers, and head CT were insignificant. His speech became louder with the treatment but was disorganized. He was noted to be psychotic, internally preoccupied, talking about seeing lasers "little flash dots" that may hurt him. Risperidone 1 mg /day was initiated to address psychosis. Risperidone was titrated to 4 mg/day and lorazepam to 3mg/day over the next 5 days to target the symptoms. His gait improved over the following week. As his thought process became more coherent, he reported heavy cannabis use in the context of feeling "down" in the previous month due to the break-up with his girlfriend of 1 year. He admitted to increasing cannabis use over the past 3 months. He started using it at age13 years because it helped him relax and later acknowledged using "multiple joints everyday" in the month prior to the hospitalization. He reported occasional alcohol use and denied using other substances. During the second week of hospitalization, his symptoms fluctuated. He became more psychotic, staring out of the window and became paranoid about being watched, followed on the unit. He endorsed hallucinations of seeing angels and demons that giggled in his ears. He also complained of something picking at his legs, trying to pull his heart out of his chest and his stomach moving in a wave. He reduced his oral intake in order to fast for Jesus to return. Risperidone was then titrated to 6 mg/day leading to significant improvement in psychosis. His lorazepam was slowly tapered down over 2 weeks and discontinued with the successful treatment of catatonia. He was monitored using serial mental status exams and the BFCRS. His BFCRS score eventually diminished to 0/69. He was discharged from the hospital after a 2.5-month stay on the regimen of risperidone with a referral to a dual diagnosis program.

Discussion

Mr. "R" presented with psychosis, abnormal gait, and catatonia in the context of heavy cannabis use. Our working diagnosis was cannabis induced psychotic disorder. Given that he did not meet the duration criteria yet, early onset schizophrenia was a differential diagnosis. Chronic cannabis use, especially in adolescence, in association with genetic predisposition to psychosis and exposure to environmental stressors like childhood trauma increases the risk of psychotic outcomes in later life [8]. This supports the two-hit hypothesis for schizophrenia. Our patient reported recent stressors: failing the regents exam and a break up with his girlfriend. Also, his family history is positive for psychosis in a cousin suggesting probability of genetic loading in our patient.

Cannabinoid receptors are located in movement-related brain regions and a recent study found cannabis users exhibited lower limb abnormalities during gait [9]. It suggests history of cannabis use led to motor deficits acutely and is associated with long-lasting changes in open-chain elements of walking gait [9]. There were no abnormal findings in the meticulous neurological workup in the patient, which led to the conclusion that cannabis could be a risk factor responsible for his altered gait. However, it is hard to distinguish if the gait abnormality was a result of cannabis led insult to movement-related brain regions or due to the catatonia. Nevertheless, it is vital to note that his gait improved with the period of abstinence and treatment during the hospital stay.

Abrupt cannabis cessation can dysregulate GABA/glutamate balance contributing to catatonia [10]. Interestingly, GABAergic regulating drugs such as selective GABA-B agonist Baclofen is postulated in the management of cannabis dependence [10]. Our patient was unable to recall if he suddenly stopped using cannabis in the days leading to his admission. Another hypothesis postulates that prostaglandins are involved in the catatonia produced by tetrahydrocannabinol (THC), the main psychoactive compound of cannabis [11]. A report highlighted a case of periodic catatonia with an increase in cannabis usage in an individual with cannabis dependence [12].

Long-term, chronic cannabis use is implicated in reduced hippocampal, amygdala and cerebellar white-matter volumes similar to that observed in schizophrenia [13,14]. Exposure to exogenous cannabinoids in adolescence can lead to a disturbance of the endocannabinoid system in the brain and interfere with neurodevelopmental processes like neurogenesis, neural maturation, and specification [8]. It has been suggested that adolescents are more susceptible to the adverse effects of cannabis use due to ongoing brain development and may result in poorer executive functioning [15]. Upon a 6-month and 1-year follow up by the team, our patient had remained psychotic. He was attending outpatient treatment and had received the diagnosis of schizophrenia.

Conclusion

Psychiatric conditions such as psychosis and catatonia are complex and multifactorial. This case provides the first report of an adolescent with an emergence of catatonia and gait abnormality along with psychosis associated with chronic cannabis use. The working hypothesis is that his presentation was a result of the cumulative insults to developing brain due to repeated cannabis use. Longitudinal studies to establish this causative relationship should be held in the future. Further research into the potential safety concerns surrounding cannabis use in adolescents is urgently needed. When treating new-onset psychosis in adolescents, clinicians need to be watchful for cannabis-induced psychosis and any physical, motor effects as well. Apt diagnosis and treatment can prevent enduring and devastating outcomes in the susceptible adolescent population.

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