Synthetic Cannabis induced Agitated Catatonia in Adolescents

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Introduction

- Catatonia is a psychomotor condition with manifestations, ranging from severe immobility to excessive psychomotor agitation. Roughly 90,000 people tend to suffer from catatonia each year, with a prevalence ranging between 7.6% to 38%. It is most often characterized by decreased motor activity, lack of responsiveness, and posturing. Its etiology can be related to general medical conditions or therapeutic and illicit drugs. Synthetic cannabinoids (SCs) have been commonly used as illicit drugs posing a serious public health concern due to their profound effects on physical and mental health. The toxicologic profile of SC use may vary, including changes in mental state, psychosis, and catatonia. Hence, when adolescents with no prior medical or mental health history present with catatonia, a thorough workup is warranted to investigate the cause. In such a situation, SC use as an etiological factor becomes an important consideration as routine drug testing cannot trace these substances. As the use of SCs increases in this population, it is essential for mental health providers to understand the role of SCs in the development of neuropsychiatric symptoms, especially agitated catatonia. This could facilitate early detection of SC as a potential cause of catatonia and can lead to timely intervention and management.

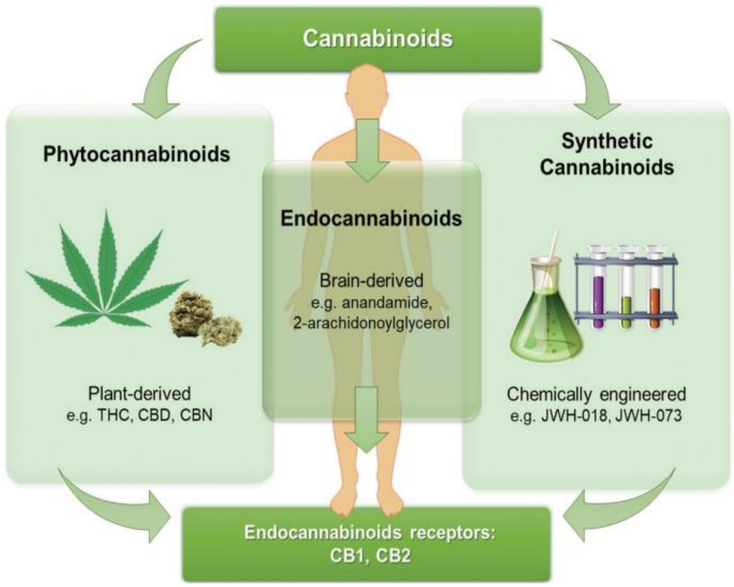

Fig 1: Types of cannabinoids: phytocannabinoids, endocannabinoids and synthetic cannabinoids

Case Presentation

- We present a case of a 17-year-old Hispanic male who developed agitated catatonia after SC use. The patient initially presented to the emergency department with sweating, insomnia, and auditory hallucinations. He had a history of THC, and alcohol use and reported "synthetic vaping" at the time of presentation with a UDS negative for THC. The medical workup was unremarkable; hence psychiatry was consulted due to concerns for first-episode psychosis and catatonia.

Case Presentation

- On psychiatric assessment, he was found to be distractible and exhibited grimacing, stereotypy, negativism, and ambitendency with a Bush Francis Catatonia Rating Scale (BFCRS) score of 11. A lorazepam challenge test was administered to investigate a diagnosis of catatonia. Catatonia was considered as the underlying cause of the patient's presentation, and he was started on lorazepam 0.5 mg by mouth twice a day. The lorazepam was increased to 1 mg by mouth twice a day. This regimen resulted in a significant improvement in his catatonia symptoms. After recovery, the patient admitted to smoking synthetic marijuana daily to cope with the stress in his life for the past year. Therefore, in the absence of any other etiology, it was concluded that the patient's catatonic presentation and psychosis were attributed to his cannabis use. At discharge, an Ativan taper was recommended, and a follow-up appointment was scheduled with a psychiatrist.

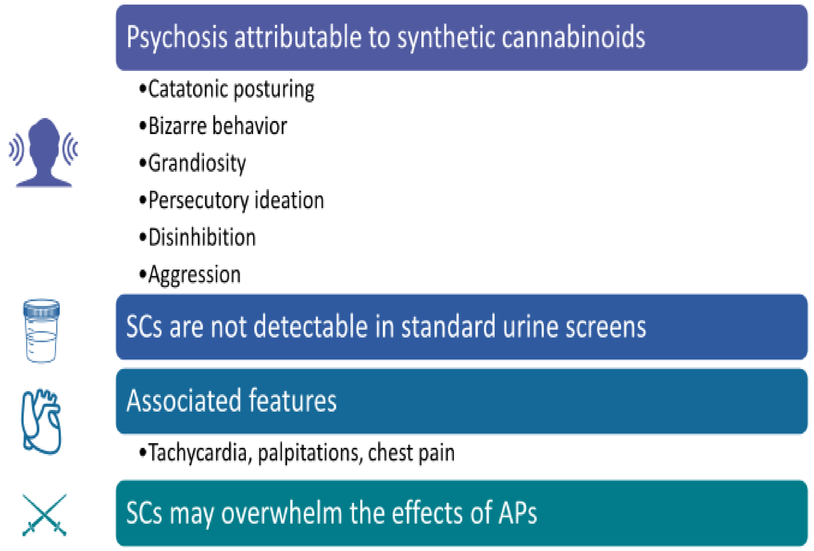

Figure 2. Key Symptoms of Synthetic Cannabinoids use

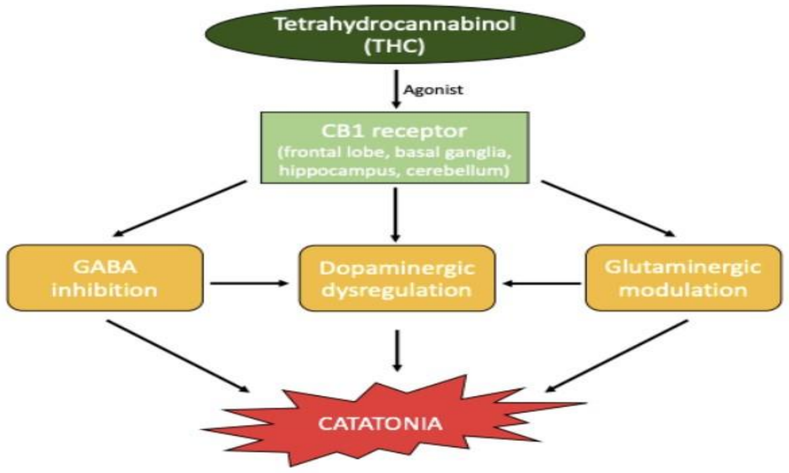

Figure 3. Neurotransmitter Systems Involved in Cannabis-Induced Catatonia

Discussion

- With increasing legalization and marijuana use worldwide, including in the United States, we are likely to see an increased prevalence of catatonia along with other psychiatric conditions. Although no precise pathophysiological mechanisms of catatonia have been identified, studies have reported that it occurs primarily due to dysregulation of several neurotransmitter systems—particularly the dopaminergic, gabaergic, and glutaminergic systems—which have been implicated in catatonia. There seems to be cortical-to-cortical and cortical-to-subcortical modulatory dysfunction in patients with catatonia. The cortical-to-cortical modulatory dysfunction refers to hyperactivity of the orbitofrontal cortex and other prefrontal cortex areas, with alterations in connections between these areas to motor and premotor areas of the cortex. The cortical-to-subcortical modulatory dysfunction refers to dysfunction of cortical areas, mostly in the frontal and parietal lobes, leading to alterations in connections to subcortical motor areas in the basal ganglia. The cortical dysfunction seen in catatonia is related to decreased gabaergic tone, which explains the response to lorazepam. The use of N-methyl-D-aspartate (NMDA) antagonists also has some efficacy in the treatment of catatonia, likely related to the relative glutaminergic hyperactivity seen in the condition. Gabaergic and glutaminergic system dysfunction are not only related to catatonia itself but are also related to cannabis use (figure 3).
- Consumption of synthetic cannabinoids, also known as "spice" or "k2," has also led to catatonic presentations. Synthetic cannabinoids have increased in popularity in recent years due to their ease of availability, low cost, and lack of detection by standard urine drug screens.
- Benzodiazepines, especially lorazepam, are found to be extremely beneficial in resolving symptoms of different catatonic presentations by increasing GABA and decreasing NMDA receptor activity and are generally used as the first line of treatment. Even though it is rare, the dosing of BZDs up to 20 mg to 30 mg per day is required for improvement or resolution of catatonic symptoms. In patients unresponsive to BZDs, ECT has shown to be efficacious as 2nd line.

Conclusion

- The use of SCs, especially among adolescents, seems to be an increasing public health concern and a serious issue. It is essential to identify and manage their notorious effects on the mental health of adolescents who are using it. The case discussed demonstrates the potentially severe impact of SCs and hence highlights the urgency with which the scientific community must address the many unknowns that surround them. Healthcare providers should consider SCs as an etiological factor of psychosis and catatonia, especially in the presence of red flags related to substance use.

References

References are available upon request