Mania Induced by a Single Dose of Synthetic Cannabinoid

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Abstract

Synthetic cannabinoids (SCs) are substances with chemical structures that comprise a family of compounds and have structures that can bind to cannabinoid receptors in human cells known as CB1 or CB2. Most SCs are stronger than tetrahydrocannabinol and pose a greater health risk. In this article, we present a mania case that was triggered by a single dose of the SC Bonzai and registered improvement after long-term inpatient treatment. In the literature, only one previous case has been reported of a manic attack induced by SC, and this previously reported case had manic symptoms that started after 6 months of SC use. In our case, we aimed to discuss the possible mechanisms of SC use, which may cause affective symptoms and a serious psychotic manic episode even in the absence of long-term exposure.

Key Words: mania, synthetic cannabinoids, substance abuse

CASE REPORT

A 25-year-old male patient was brought to the emergency room by his brother and father, exhibiting aggression, flood, the belief that he was a chosen person as a secret military agent, decreased sleep for the past 1 week, and a lot of jumping from one subject to another during conversation. During the patient’s admission to the clinic, the patient punched his father due to delusions of persecution. He was admitted to the psychiatry inpatient clinic from the emergency room.

In his first psychiatric examination, the patient was conscious and oriented. His psychomotor activity was increased, his hostility was evident, his affinity was irritable and good, and his affect was dysphoric. The amount and speed of his speech had increased, with abusive speech, grandiose delusions about being a chosen person, and delusions of persecution by family members. It was learned that his interest in religion had increased and that he had been praying and reading prayers when he was not sleeping. He was questioned about visual and auditory hallucinations, but he did not have insight.

In the anamnesis obtained from the patient’s family, it was determined that the patient had no previous history of psychiatric illness, except drug abuse. He had started drug use 5 years earlier with cannabis. He had been using methamphetamine for the past 2 years, and, 1 week before being admitted, he had started using Bonzai. After the manic symptoms started, the possibility of his having used Bonzai was questioned in

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detail. However, it was clear from the anamnesis, before using Bonzai, the patient’s sleep pattern had been normal, there had been no hostility, and there had been no delusions or other manic symptoms. Physical and neurological examinations (hemogram, electrolyte levels, kidney and liver function tests, electrocardiography, electroencephalography, and brain magnetic resonance imaging) revealed no pathological findings. A urine toxin screening test for K2-type SCs (including Bonzai) was positive, and the other measured parameters [amphetamine, methamphetamine, benzodiazepines, cocaine, methadone, tetrahydrocannabinol (THC), opioids, phencyclidine, and 3,4-methylenedioxymethamphetamine] were negative. The patient was diagnosed with bipolar disorder caused by substance use according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria and was followed up for treatment. Three ampoules of haloperidol, 1 ampoule of biperiden, and 1 ampoule of chlorpromazine were injected due to the patient’s marked increase in psychomotor activity and especially his aggression toward his family, who had brought him to the examination. In the initial assessment, the Young Mania Rating Scale (YMRS) score was 46/60, and the Positive and Negative Syndrome Scale score was 112/210.

Chemical determination of the patient was continued for 3 days due to continued aggression and elevation of mood state. His increased hostility and psychomotor activity did not decrease, despite intermittent chemical and physical fixation for 3 days. Risperidone (8 mg/d), diazepam (20 mg/d), olanzapine (20 mg/d), lithium (1200 mg/d), and quetiapine (900 mg/d) were started on the third day of treatment. Zuclopenthixol depot (200 mg injection) was added once every 2 weeks, after taking into consideration the difficulty of compliance with oral medications during follow-ups and long-term follow-up. At the 2-week follow-up, there was no regression in the findings for the patient, and he had a YMRS score of 42/60. At that time, his treatment was arranged as risperidone (12 mg/d), and chlorpromazine (300 mg/d) was added, and the other drugs were continued. At the end of the third week, the patient’s YMRS score was 38/60, psychological findings continued, and anesthetic electroconvulsive therapy was started 3 times a week with the consent of his parents. The patient responded dramatically to the treatment, as his YMRS score improved to 17/60. After 5 weeks of treatment, the patient had a YMRS score of 11/60, a Positive and Negative Syndrome Scale score of 41/210, with euthymic, normal speech at a regular rate. He had improved insight and complied with oral medication, and he was discharged with zuclopenthixol depot injection, risperidone (8 mg/d), biperiden (6 mg/d), olanzapine (20 mg/d), chlorpromazine (200 mg/d), quetiapine (900 mg/d), and lithium (1200 mg/d). The patient’s lithium blood level at discharge was 0.82 mmol/dL. The patient, whose treatment compliance continued after 1 month and subsequent controls and who stopped using SCs, was followed up for 2 years and remained in full health without another attack of mania.

DISCUSSION AND CONCLUSION

Long-term use of NC has been associated with the occurrence of psychotic symptoms, relapse of psychosis, and worsening of psychotic symptoms. Similarly, psychotic symptoms such as visual hallucinations, paranoid delusions, thought block, and irregular speech have been reported with prolonged use of SCs. In addition, the characteristic symptoms of cannabis-induced mania, hypomania, and agitation have been reported.

One study found that SC use has a stronger relationship with a wide range of mental health problems compared with NC use. In studies comparing NC and SC users, the use of SC was found to lead to psychotic symptoms, agitation, and longer hospitalizations than NC use. The pharmacological effect of SCs is similar to the main active ingredient of NC, Δ9-THC2, but SCs have a far greater binding affinity, especially for the CB1 receptor. As a potent CB1 receptor agonist, SC has been observed to cause synaptic dysfunction in users, potentially greater than that of the partial agonist Δ9-THC. SC blends and pure...
SCs do not contain cannabidiol, a compound found in NC that is associated with psychotic experiences and anxiety reduction and that can reduce the potential risk of mental health problems occurring after the use of SCs.\textsuperscript{8} SCs have been reported to cause manic episodes.\textsuperscript{2} In the reported cases, there is also a case of psychosis triggered by the use of Bonzai, but there was no manic episode resulting from a single dose use of Bonzai in our literature review.\textsuperscript{9}

In the presented case, it has been observed that the patient, who had been using cannabis for 5 years and amphetamine for 2 years, had not had any mood disorder attacks in the past 5 years and before, and there was no evidence of psychiatric illness in the anamneses of his family and himself. The clinical symptoms of the patient appeared to occur after Bonzai use; hence, it was thought that his manic attack was triggered by the use of SCs. Although the long-term history of cannabis and amphetamine use was also a risk factor for the occurrence of a mood disorder, it was still held that the main trigger of this emerging manic attack might have been the SC use.\textsuperscript{10} In the literature, it was seen that the patient had used SC for 6 months in the case of a manic episode induced by SC and that this was the only case reported on the subject.\textsuperscript{2} In our case it was observed that a manic episode period accompanied by serious and long-term psychotic symptoms was induced by a single dose use of SC. This suggests that there may not be a physiopathologic mechanism that develops after chronic exposure, and the use of synthetic drugs may stimulate a potential psychiatric trigger point in the patient as well as idiosyncratically. Of course, controlled studies are needed to illuminate this mechanism.

**REFERENCES**


