

# Acute Psychosis Following Synthetic Cannabinoid (K2) Use in a 16-Year-Old Male: A Case Report

Megan Woods<sup>1</sup>, Bryce Tarno<sup>2</sup>, Faheem Arain<sup>3</sup>

<sup>1,2</sup>A.T. Still University – Kirksville College of Osteopathic Medicine

<sup>3</sup>Royal Oaks Hospital, Compass Health Network

**Abstract:** *Synthetic cannabinoids (SCs), commonly known as K2 or Spice, are increasingly used among adolescents and are associated with severe neuropsychiatric effects due to their high potency as full cannabinoid receptor agonists. This report describes a 16-year-old male with no prior psychiatric history who presented with an acute onset of psychosis characterized by paranoia, religious delusions, disorganized behavior, and visual hallucinations following suspected synthetic cannabinoid exposure via vaping. Routine toxicology detected THC but no other substances, complicating diagnostic confirmation. Medical evaluation was unremarkable, and the patient required inpatient psychiatric hospitalization. Treatment with olanzapine led to gradual symptom resolution by hospital day 7. This case highlights the diagnostic limitations of standard drug screening and underscores the need for clinical vigilance in adolescents presenting with abrupt psychosis. Early recognition and intervention are essential to reduce morbidity and potential progression to persistent psychiatric disorders.*

**Keywords:** synthetic cannabinoids, K2, adolescent psychosis, substance-induced psychosis, vaping

## 1. Introduction

Synthetic cannabinoids (SCs), commonly marketed under names such as Spice and K2, are a heterogeneous group of new psychoactive substances (NPS) designed to mimic the psychoactive effects of  $\Delta^9$ -tetrahydrocannabinol (THC), the primary psychoactive component of cannabis [1,2]. Unlike natural cannabis, which acts as a partial agonist at cannabinoid receptors, most synthetic cannabinoids act as full agonists at cannabinoid type 1 (CB1) receptors and often demonstrate substantially greater receptor affinity and potency, resulting in more intense and unpredictable neuropsychiatric effects [1,3].

Since the mid-2000s, synthetic cannabinoids have emerged as one of the largest and most rapidly evolving classes of NPS worldwide, with hundreds of structurally distinct compounds identified [2,3]. Their use is disproportionately observed among adolescents and young adults, driven by low cost, ease of access, misleading perceptions of safety, and the ability to evade detection on standard urine drug screens [2–5]. Products sold as synthetic cannabinoids often contain unknown and rapidly changing chemical compositions, with little to no quality control, contributing to marked variability in clinical presentations and toxicity [1,6].

From a pharmacologic perspective, synthetic cannabinoids differ from natural cannabis not only in receptor binding but also in their metabolism and interaction with drug-metabolizing enzymes, further contributing to variability in clinical effects and toxicity [7]. Many SCs act as substrates or inhibitors of cytochrome P450 enzymes, raising concerns about variability in potency for prolonged or exaggerated effects and unpredictable clinical courses, particularly in pediatric populations [6,7].

Accumulating evidence indicates that synthetic cannabinoids are associated with a significantly higher risk of severe adverse effects compared to natural cannabis [4,8]. Reported complications include agitation, autonomic instability,

seizures, acute kidney injury, and cardiovascular events; however, neuropsychiatric manifestations, particularly acute psychosis, are among the most concerning outcomes [4,8]. Compared with cannabis-associated psychosis, synthetic cannabinoid-induced psychosis has been shown to present with more severe positive symptoms, poorer functional outcomes, and increased suicidal ideation [5]. Individuals with a family history of psychotic disorders - including schizophrenia, schizoaffective disorder, mood disorder with psychotic features - have a two- to fivefold increased risk of developing psychosis among adolescents who regularly use cannabinoid products [9].

Adolescents may be especially vulnerable to the neuropsychiatric effects of synthetic cannabinoids due to ongoing brain development and heightened sensitivity of the endocannabinoid and dopaminergic systems during this critical period [3,10]. Preclinical and clinical studies suggest that synthetic cannabinoids may induce neurotoxic changes, including hippocampal injury, oxidative stress, neuroinflammation, and alterations in glutamatergic and dopaminergic signaling pathways implicated in the pathogenesis of psychosis [11]. These findings raise concern for both acute symptom severity and potential long-term neuropsychiatric sequelae following adolescent exposure.

Despite growing recognition of these risks, diagnosis of synthetic cannabinoid-induced psychosis remains challenging. Routine drug screens frequently fail to detect these compounds, and clinical presentations may closely resemble first-episode primary psychotic disorders, leading to potential misclassification and delayed intervention [1,4]. Furthermore, adolescent-specific data remains limited, with much of the existing literature derived from adult populations, retrospective analyses, or mixed-substance cohorts [3,5].

We present a case of a previously psychiatrically stable 16-year-old male who developed acute psychosis following synthetic cannabinoid use, highlighting diagnostic challenges and management considerations in adolescent populations.

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## 2. Case Presentation

### Patient Information

A 16-year-old African American male high school student with no prior psychiatric diagnoses or hospitalizations presented for evaluation of acute behavioral changes and psychotic symptoms. He was a sophomore at a public high school and resided at home with his mother and multiple siblings. There was no history of special education placement, and academic performance was described as average before symptom onset.

### Chief Complaint

“Mom thought I was under psychosis.”

### History of Present Illness

The patient was brought to the emergency department by his mother for an acute onset of psychotic symptoms, including paranoia, disorganized behavior, and perceptual disturbances. According to collateral information obtained from his mother, symptoms began abruptly several days before presentation and progressively worsened.

In the days preceding admission, the patient exhibited religious preoccupation and delusional beliefs, including referring to himself as a prophet, believing that downstairs areas of the home represented “hell,” and stating that he was “possessed by God.” He demonstrated increasing paranoia, reporting that people were shooting at the house, hiding in bushes, and communicating through video subtitles. He also endorsed visual hallucinations, describing seeing “spirits,” though he inconsistently acknowledged whether these entities were speaking to him.

Behavioral changes included marked social withdrawal, spending prolonged periods isolated in his room, irritability, episodes of staring with psychomotor freezing, refusal to shower due to fear that something bad would occur, and significant sleep disturbance, including near-total insomnia for at least 24 hours before admission. His mother also reported hypersexual behaviors, including excessive pornography use and masturbation, which represented a change from baseline.

Substance use history obtained from collateral sources revealed ongoing THC use via vape pens beginning several years ago; however, the duration of use was not precisely established. His mother discovered multiple THC and nicotine vape devices among his belongings and expressed concern that some cartridges likely contained synthetic cannabinoids (K2), although the exact substance and concentration were unknown. The patient reportedly used these products regularly, often in isolation, and the route of administration was vaping. The timing of the last use was unclear, but exposure was suspected shortly before symptom onset. There was no clear evidence of dose escalation, though the unregulated nature of the substances raised concern for variable potency.

The patient denied alcohol use and denied use of methamphetamine, cocaine, heroin, PCP, or other illicit substances. He demonstrated poor insight into his symptoms and did not recognize his experiences as pathological.

Psychosocial stressors included growing up in a neighborhood with increased exposure to violence, heavy engagement with online video games and Discord communities, and recent interpersonal conflict during online gaming interactions, which appeared to exacerbate paranoid ideation.

Due to the severity of psychosis, the patient was unable to provide a reliable self-history at the time of evaluation.

### Past Psychiatric History

The patient had no prior psychiatric diagnoses, outpatient psychiatric care, psychotropic medication trials, or psychiatric hospitalizations. There was no history of suicide attempts or self-harming behavior.

### Past Medical History

Medical history was unremarkable. He denied a history of seizures, traumatic brain injury, congenital heart disease, or chronic medical conditions. Immunizations were up to date. There were no known drug allergies.

### Medication History

The patient was not taking any prescribed medications before admission.

### Substance Use History

- Alcohol: denied
- Cannabis: THC use via vape pens; frequency and duration not precisely established
- Synthetic cannabinoids: Suspected exposure (K2), though not laboratory-confirmed
- Nicotine: regular vaping
- Other substances: Denied

### Family Psychiatric History

Family psychiatric history was notable for substance use disorders, including maternal history of cocaine, PCP, and THC use, currently in recovery. The patient’s biological father had a history of substance use, though details were limited. Two siblings reportedly had a history of suicidal ideation. There was no known family history of schizophrenia or bipolar disorder, though mental health discussions were limited within the family.

### Social History

The patient lived with his mother and siblings in a stable home environment. Parental supervision was present but strained by the patient’s increasing isolation and secrecy. He was not employed and had no legal history. Peer relationships were primarily online, with extensive screen time before admission. Academic performance was described as average, without prior disciplinary issues.

### Mental Status Examination

On examination, the patient appeared his stated age, was well-groomed, and demonstrated intermittent eye contact. Behavior was guarded and intermittently uncooperative. Speech was normal in rate and volume but disorganized at times. Mood was described as “fine,” with a labile and incongruent affect.

Thought processes were disorganized with loosening of associations. Thought content was notable for religious delusions, paranoid ideation, and referential thinking. The patient endorsed visual hallucinations and inconsistently reported auditory phenomena. Additionally, the patient had markedly impaired insight and judgement regarding illness awareness. He denied current suicidal or homicidal ideation.

### Physical Examination and Laboratory Findings

Vital signs on presentation were stable. Neurologic examination was grossly nonfocal. Laboratory evaluation, including complete blood count and comprehensive metabolic panel, was within normal limits. Urine drug screening was positive for THC and negative for other substances, including amphetamines, cocaine, opioids, and benzodiazepines. Creatine kinase levels were not significantly elevated. No acute abnormalities were identified on medical evaluation.

### Hospital Course and Clinical Management

The patient required inpatient psychiatric hospitalization for safety and stabilization. During the hospital course, he exhibited persistent psychotic symptoms and episodes of agitation, including an attempt to forcefully exit the unit, resulting in minor injury to the lower right extremity from hitting it on a wall. He required close observation and pharmacologic intervention. Treatment was initiated with olanzapine, titrated to 10 mg nightly. Due to ongoing agitation, PRN haloperidol and lorazepam were made available.

### Outcome and Follow-Up

The patient was diagnosed with first-episode psychosis, suspected substance-induced, with differential consideration given to cannabis or synthetic cannabinoid-associated psychosis. Over the course of hospitalization, psychotic symptoms gradually improved and resolved on hospital day 7. The patient was discharged home with recommendations for follow-up with primary care, a mental health counselor, and psychiatry in the outpatient setting. Continued antipsychotic treatment was encouraged in the outpatient setting along with strong recommendations for abstinence from all cannabinoid products. Substance use counseling and close family supervision were emphasized.

### Differential Diagnosis

- Substance-induced psychotic disorder (cannabinoids/synthetic cannabinoids)
- Primary psychotic disorder
- Mood disorder with psychotic features
- Delirium or medication-induced psychosis

## 3. Discussion

Synthetic cannabinoids are a growing public health concern, particularly among adolescents, due to their high potency, accessibility, and ability to evade standard toxicology screening. Unlike THC, which is a partial agonist at cannabinoid receptors, most synthetic cannabinoids act as full agonists at CB1 receptors, resulting in exaggerated and highly variable neuropsychiatric effects. This increased receptor activity is thought to contribute to significant downstream

dopaminergic dysregulation, which is a key mechanism in the development of psychosis.

In addition to receptor-level differences, synthetic cannabinoids may exert neurotoxic effects through oxidative stress, neuroinflammation, and disruption of glutamatergic signaling pathways. These mechanisms are particularly concerning in adolescents, whose brains are still undergoing critical neurodevelopment. Increased sensitivity of the endocannabinoid and dopaminergic systems during this period may increase vulnerability to both acute psychosis and long-term psychiatric disorders.

This case also illustrates the diagnostic challenges associated with synthetic cannabinoid exposure. Routine urine drug screens do not detect most synthetic cannabinoids, which can lead to under-recognition or misclassification as primary psychotic disorders. In this patient, the presence of THC on screening may have further confounded the clinical picture, as symptoms could be incorrectly attributed to natural cannabis use rather than more potent synthetic compounds. A high index of suspicion, particularly in adolescents presenting with abrupt and severe psychosis, is essential for accurate diagnosis.

Management of synthetic cannabinoid-induced psychosis is largely supportive and symptom driven. Antipsychotic medications, such as olanzapine, are commonly used and may be effective in controlling acute symptoms, as demonstrated in this case. Additional medications, including benzodiazepines, may be necessary for agitation. Close monitoring is critical due to the risk of complications such as severe agitation, autonomic instability, and potential progression to prolonged psychotic states.

Prognosis remains variable. While many patients experience resolution of symptoms with cessation of substance use and appropriate treatment, there is increasing evidence that synthetic cannabinoid exposure may precipitate persistent or recurrent psychosis, particularly in vulnerable individuals. Adolescents with substance-induced psychosis are at increased risk of later developing primary psychotic disorders, emphasizing the importance of close psychiatric follow-up and early intervention.

Overall, this case underscores the importance of clinician awareness of synthetic cannabinoids as a cause of acute psychosis in adolescents. Improved recognition, early intervention, and targeted education regarding the risks of these substances are critical steps in reducing morbidity and preventing long-term psychiatric outcomes.

## 4. Conclusion

This case illustrates the significant neuropsychiatric risks associated with synthetic cannabinoid use in adolescents, particularly the potential for rapid onset of severe psychosis. Limitations of routine toxicology screening complicate the diagnostic process and overlap with primary psychiatric disorders. Prompt recognition, thorough clinical assessment, and early initiation of antipsychotic treatment are critical for symptom resolution and prevention of adverse outcomes. Increased clinician awareness and targeted public health

strategies are essential to address the growing use of these substances and mitigate long-term psychiatric consequences in vulnerable populations.

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## References

- [1] Castaneto MS, Gorelick DA, Desrosiers NA, Hartman RL, Pirard S, Huestis MA. Synthetic cannabinoids: epidemiology, pharmacodynamics, and clinical implications. *Drug Alcohol Depend*. 2014;144:12-41.
- [2] Roque-Bravo R, Silva RS, Malheiro RF, et al. Synthetic cannabinoids: A pharmacological and toxicological overview. *Annu Rev Pharmacol Toxicol*. 2023;63(1):187-209.
- [3] Souza JDR, Madruga CS, Noto C. Emerging synthetic cannabinoids and the risk of psychosis: a growing concern for public health. *Rev Bras Psiquiatr*. 2023;45(4):306-307.
- [4] Taflaj B, La Maida N, Tittarelli R, Di Trana A, D'Acquarica I. New psychoactive substances toxicity: A systematic review of acute and chronic psychiatric effects. *Int J Mol Sci*. 2024;25(17):9484.
- [5] Ricci V, Ceci F, Di Carlo F, et al. First episode psychosis with and without the use of cannabis and synthetic cannabinoids: Psychopathology, global functioning and suicidal ideation and antipsychotic effectiveness. *Psychiatry Res*. 2023;320(115053):115053.
- [6] Alzu'bi A, Almahasneh F, Khasawneh R, Abu-El-Rub E, Baker WB, Al-Zoubi RM. The synthetic cannabinoids menace: a review of health risks and toxicity. *Eur J Med Res*. 2024;29(1):49.
- [7] Kong TY, Kim JH, Kim DK, Lee HS. Synthetic cannabinoids are substrates and inhibitors of multiple drug-metabolizing enzymes. *Arch Pharm Res*. 2018;41(7):691-710.
- [8] Prete MM, Feitosa GTB, Ribeiro MAT, Fidalgo TM, Sanchez ZM. Adverse clinical effects associated with the use of synthetic cannabinoids: A systematic review. *Drug Alcohol Depend*. 2025;272(112698):112698.
- [9] Samoon Ahmad KPH. *Medical Marijuana a Clinical Handbook*. Wolters Kluwer Health; 2020.
- [10] Hoch E, Volkow ND, Friemel CM, Lorenzetti V, Freeman TP, Hall W. Cannabis, cannabinoids and health: a review of evidence on risks and medical benefits. *Eur Arch Psychiatry Clin Neurosci*. 2025;275(2):281-292.
- [11] Alzu'bi A, Abu-El-Rub E, Almahasneh F, et al. Delineating the molecular mechanisms of hippocampal neurotoxicity induced by chronic administration of synthetic cannabinoid AB-FUBINACA in mice.