

A Diagnostic Dilemma Involving Dabs in First-Episode Psychosis

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CASE DESCRIPTION

An 18-year-old female with no prior psychiatric history presented to a psychiatric unit with a self-inflicted neck stab wound secondary to experiencing command auditory hallucinations to inflict self-harm. In the initial interview, she had short, vague responses. She reported dabbing cannabis daily for 2 months before her presentation. Recent psychosocial stressors included a breakup with a boyfriend, the death of her dog, and unstable housing. She had no family history of psychiatric disorders. Her mother reported that 2 months ago she was at baseline attending high school and functioning independently, but more recently she began socially withdrawing, not attending school, and demonstrating speech latency. A video showed the patient internally preoccupied.

Baseline serum laboratory results were fairly unremarkable with hypokalemia that resolved without intervention within 1 day (Table 1). A urine toxicology screen was positive for cannabis only. On presentation, she was managed with counseling and observation without an antipsychotic medication in hope that her symptoms would improve with cannabis abstinence. However, this approach was unsuccessful, and she was prescribed quetiapine, to which she responded poorly. She was transitioned to risperidone but continued to have

evidence of psychosis marked by auditory hallucinations, delusions, thought insertion, speech latency, paranoia, and blunted affect. She required reminders to perform activities of daily living. After transitioning her to olanzapine, she had less prominent auditory hallucinations and was able to perform self-care unassisted. She was discharged to her mother's care after 1 month in the psychiatric unit.

DISCUSSION

Dabbing is a new method of cannabis inhalation. Dabs can be synthesized by the user or purchased from local marijuana dispensaries (depending on state law). Names for dabs are based on their wax or glass-like consistency and include "butane hash oil," " Δ^9 -tetrahydrocannabinol (THC)³ concentrate," "honey," "glass," "wax," "shatter," and "amber" (1). A common method of consumption involves heating a piece of metal with a blow torch then placing dabs on the heated metal surface, which vaporizes the product into a modified water pipe that serves as a conduit for the vapor to be inhaled. Alternatively, some users place dabs into electronic cigarettes. Local marijuana dispensaries report the average cost of dabs is \$20–\$60 per gram and the average consumption is one-tenth of a gram per use. Users prefer dabs to traditional marijuana because they require less inhalations to feel high, the

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³ Nonstandard abbreviations: THC, Δ^9 -tetrahydrocannabinol THC; CBD, cannabidiol.

Table 1. Baseline laboratory results.

Test	Result (reference interval)
Cannabinoids, urine	Positive (negative)
Phencyclidine, urine	Negative (negative)
Barbiturate, urine	Negative (negative)
Cocaine, urine	Negative (negative)
Amphetamine/ methamphetamine, urine	Negative (negative)
Opiates, urine	Negative (negative)
Benzodiazepines, urine	Negative (negative)
Alcohol, urine	Negative (negative)
Sodium	139 (135–145) mmol/L
Potassium	3.0 repeat 4.0 (3.5–5.5) mmol/L ^a
Chloride	103 (95–110) mmol/L
Carbon dioxide	27 (18–32) mmol/L
Anion gap	12 (5–16) mmol/L
Protein, total	7.8 (6.0–8.6) g/dL
Albumin	4.6 (3.2–5.5) g/dL
Bilirubin, total	0.6 (0.1–1.2) mg/dL
Alkaline phosphatase	35 (25–165) U/L
Aspartate transaminase	17 (0–60) U/L
Alanine transaminase	24 (0–50) U/L
Total immunoglobulin	3.2 (1.8–3.5) g/dL
Glucose, mg/dL	114 repeat 92 (60–99) mg/dL ^a
Urea nitrogen	6 (5–26) mg/dL
Creatinine	0.7 (0.5–1.5) mg/dL
Calcium	9.5 (8.5–10.6) mg/dL
Thyroid-stimulating hormone	1.706 (0.400–5.000) μ IU/mL
White blood cell	9.18 (4.00–10.50) $10^3/\mu$ L
Hemoglobin	15.7 (11.5–15.0) g/dL
Hematocrit	43.9% (34.0%–44.0%)
Platelets	215 (140–415) $10^3/\mu$ L
Human chorionic gonadotropin, serum	Negative (negative)

^a Repeat laboratory results after 1 day.

effects are perceived as stronger, the duration of effects is longer, the high feels “different,” and there is no ash product (2).

The amateur manufacturing process of dabs is dangerous. A common form of home extraction is an open system that involves butane to extract cannabinoids (1). Explosions can occur from something as simple as a spark if the extraction is performed in a poorly ventilated area because butane is highly flammable. In a study of Colorado burns

related to manufacturing dabs, 29 cases of hydrocarbon burns from butane hash oil occurred in 2008–2014 (1). Another concern is contamination of dabs by solvents from the extraction process because these substances may be toxic or psychoactive. A study of California medical cannabis uncovered that 80% of THC concentrate samples were contaminated by solvents. This study did not quantify the concentration but detected isopentane, butane, heptane, hexane, isobutane, isopropyl alcohol, neopentane, pentane, and propane in studied samples. The pesticide paclobutrazol was detected in 22.8% of samples (3). Repeat inhalation of paclobutrazol in rats, dogs, and mice is associated with hepatocyte steatosis (4). Developmental studies of rats exposed to paclobutrazol demonstrated an association of increased cleft palates in offspring (4). As described, one of the most common methods of home extraction uses butane as the solvent. A case report of a young teen who inhaled butane for 1 year listed visual hallucinations, irritability, and social withdrawal as side effects (5). This scenario is a similar presentation to the patient evaluated in this case report. Could chronic exposure to butane from dabs contribute to psychotic symptoms?

There are numerous compounds in cannabis, but THC and cannabidiol (CBD) are the most frequently studied. Recreational use of marijuana is controversial because the negative side effects of THC include increased anxiety, induced psychosis, and impaired learning, but CBD has anxiolytic and antipsychotic effects (6). Interestingly, even in traditional leaf marijuana, THC concentrations have been increasing. In 1995, leaf marijuana had an average THC concentration of 4% and in 2014 it was 12% (7). While the data regarding the THC concentration in dabs is quite limited, the studies show a significantly higher THC concentration than traditional leaf marijuana. A study of California medical cannabis found that dabs have THC concentrations ranging from 23.7% to 75.9% (3). Per the 2014 National Drug Threat Assessment Summary, dabs have an average THC concentration

of 52%, with some samples having >80% (8). THC is the focus of most studies, but the question becomes, what other psychoactive compounds are concentrated in dabs and could these compounds be harmful?

Indeed, use of THC concentrates has been associated with psychotic symptoms. A case control study of first-onset psychosis of inpatient psychiatric patients compared to population controls showed individuals using THC concentrates daily had an odds ratio of 12.1 (95% CI, 3.7–37.3) of having a psychotic episode compared to those who had never used cannabis (9). There could be several reasons for the association of increased psychosis with THC use. A few possibilities include the THC and CBD concentrations, genetic predisposition, or contamination of THC concentrates during the manufacturing process.

A possible reason for the association of increased psychosis with THC concentrates is that they commonly have less CBD than leaf marijuana (6). European THC concentrates have <0.1% CBD (6); concentrations in American concentrates have not been published. A prior study looking at THC and CBD concentrations in hair shows the patients with hair samples positive for THC only had more schizophrenia-like symptoms than patients who had both THC and CBD detected (10). Because it is thought that CBD decreases the psychotic effects of THC and generally there is minimal CBD in THC concentrates, there could be increased risk of psychosis with concentrated THC products. The mechanisms by which THC and CBD interact are an area of active, innovative research.

Marijuana is commonly used, but only a subset of individuals experience psychotic symptoms (11). One possible explanation for this condition is a difference in genetic susceptibility to psychosis. Recently, a single-nucleotide polymorphism in the gene AKT serine/threonine kinase 1 (*AKT1*) was associated with increased psychosis in daily cannabis users. *AKT1* encodes for a protein kinase that is activated by THC and results in increased dopa-

mine in the striatum, which is associated with psychosis. A case-controlled study of patients with first episode psychosis demonstrated that daily users of cannabis with a C/C genotype at locus rs2494732 had an odds ratio of 7.23 (95% CI, 1.37–38.12) of psychosis compared to a T/T genotype (11). One could hypothesize that the THC exposure imposed by dabbing would lead to increased psychotic symptoms in patients who have a genetic predisposition to psychosis with cannabis use.

Marijuana consumers should be informed of the possible risks associated with THC concentrate consumption. A concern is that patrons may think THC concentrates are equivalent to other cannabis products. The prior safety research for marijuana may not apply to the cannabis products that are available today because the chemical composition varies. The limited data regarding THC concentrates is concerning, especially given the associations to increased psychosis. Psychotic episodes can be distressing for patients, families, and communities. As THC concentrates become more readily available, the major concern is for youth, because disruptions at an early age could make it more difficult for them to achieve important long-term goals.

In this case, we speculated that the patient had a substance-induced psychosis that would resolve with abstinence from cannabis because she had no family history of psychosis and a history remarkable for daily dab use. However, her symptoms were treatment-resistant and over time became more concerning for a primary psychotic disorder. There are no data describing if THC remains psychoactive in chronic dab users after discontinuation. Because THC can deposit into the fat and tissues to be released over time, she may have had a substance-induced psychotic disorder, making diagnosis and treatment complicated. Patients with cannabis-induced psychosis may take longer for symptoms to resolve with concentrated THC products compared to traditional cannabis. Studies investigating the serum concentrations of THC

in people consuming dabs compared to traditional cannabis are needed. Furthermore, observations of elimination pharmacokinetics of THC in daily dabs users would be intriguing.

Summary

Dabbing is a new and different method of cannabis inhalation with higher THC concentrations than traditional marijuana. Because of a lack of research, we do not understand the risks associated with its use. More research of these products is needed to help educate patients about safety, contamination, and side effects. Additionally, more studies regarding those who are susceptible to psychosis with cannabis would be helpful to inform patients regarding the risk of cannabis use. The difference in chemical composition with the increase in THC concentration in traditional leaf marijuana brings into question the applicability of prior studies regarding marijuana safety and effects compared to current cannabinoid products. Patients should be warned of the lack of data and the potential for psychosis.

TAKEAWAYS

- THC concentration of leaf marijuana is 12% while THC concentrates are higher at 23.7%–75.9%.
- THC concentration in leaf marijuana has been increasing. Prior studies regarding safety and side effects may not apply to current cannabinoid products.
- Use of THC concentrates on a daily basis has an association with psychosis compared to those who never used cannabis.
- THC concentrates show contamination with solvents used for extraction and pesticides.
- A single nucleotide polymorphism at gene *AKT1* has an association of increased first-episode psychosis with daily cannabis use.

Additional Content on this Topic

Free and Glucuronide Whole Blood Cannabinoids' Pharmacokinetics after Controlled Smoked, Vaporized, and Oral Cannabis Administration in Frequent and Occasional Cannabis Users: Identification of Recent Cannabis Intake

Matthew N. Newmeyer, Madeleine J. Swortwood, Allan J. Barnes, et al. Clin Chem 2016;62:1579–92

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REFERENCES

1. Bell CJ, Slim HK, Flaten G, Linderg W, Arek W, Monte AA. Butane hash oil burns associated with marijuana liberalization in Colorado. *J Med Toxicol* 2015;11:422–5.
2. Loflin M, Earlywine M. A new method of cannabis ingestion: the dangers of dabs? *Addict Behav* 2014;39:1430–3.
3. Raber JC, Elzinga S, Kaplan C. Understanding dabs: contamination concerns of cannabis concentrates and cannabinoid transfer during the act of dabbing. *J Toxicol Sci* 2015;40:797–803.
4. European Food Safety Authority. Conclusion on the peer review of the pesticide risk assessment of the active substance paclobutrazol. *EFSA Journal* 2010;8:1876–1936.
5. Mathew B, Kapp E, Jones TR. Commercial butane abuse: a disturbing case. *Br J Addict* 1989;84:563–4.
6. Curran HV, Freeman TP, Mokrysz C, Lewis DA, Morgan CJ, Parsons LH. Keep off the grass? Cannabis, cognition and addiction. *Nat Rev Neurosci* 2016;17:293–306.
7. ElSohly MA, Mehmedic Z, Foster S, Gon S, Chandra S, Church JC. Changes in cannabis potency over the last 2 decades (1995–2014): analysis of current data in the United States. *Biol Psychiatry* 2016;79:613–9.
8. National Drug Threat Assessment Summary. Washington (DC): U.S. Department of Justice, DEA Drug Enforcement Administration (DEA); 2014.
9. Di Forti M, Morgan C, Dazzan P, Pariante C, Mondelli V, Marques TR, et al. High-potency cannabis and the risk of psychosis. *Br J Psychiatry* 2009;195:488–91.
10. Morgan CJ, Curran HV. Effects of cannabidiol on schizophrenia-like symptoms in people who use cannabis. *Br J Psychiatry* 2008;192:306–7.
11. Di Forti M, Iyegbe C, Sallis H, Kolliakou A, Falcone MA, Paparelli A, et al. Confirmation that the AKT1 (rs2494732) genotype influences the risk of psychosis in cannabis users. *Biol Psychiatry* 2012;72:811–6.