Chapter 4
Cannabis Use Disorders and Related Emergencies

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Introduction

Cannabis has been used both recreationally and therapeutically since early recorded history, with the earliest use of cannabis for medical purposes documented in a Chinese book of herbal remedies from about 2700 BC [1]. Evidence of cannabis use extends from these early reports to the modern day [1, 2]. Cannabis is a species of plant that has both psychoactive and non-psychoactive properties. There are two subspecies: sativa, commonly called hemp, which has few psychoactive properties but has been used extensively for fiber and rope production, and indica, which has a much higher concentration of psychoactive compounds.

Cannabis use is prevalent throughout the world and is thought to be the most commonly used illicit psychoactive substance. Estimates suggest that up to 4% of the adult population worldwide has used cannabis in the past year [3]. Its use does, however, vary widely in different geographic areas. Approximately 12% of the adult populations of North America and Africa use cannabis annually, compared to 0.6% of the population in South and East Asia [4]. At the time of this writing, the legal status of cannabis is in transition in the United States. Despite remaining an illegal substance under federal law, many states have legalized the therapeutic use of cannabis, and multiple states have legalized recreational use, including Colorado, California, Washington, Oregon, Alaska, Maine, Nevada, Vermont, and Massachusetts. The health impact of these changes remains to be seen.

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The main psychoactive compound in marijuana is delta-9-tetrahydrocannabinol (THC), although the cannabis plant contains over 60 different cannabinols, some with psychoactive properties. THC is a partial agonist of endogenous cannabinoid receptors. There are two forms of the receptor: CB1, which is primarily found in the CNS, and CB2, which is found in immune tissue, as well as in the CNS. These proteins are G-protein-linked, inhibit adenylyl cyclase, and result in increased potassium conductance. The endogenous cannabinoid neurotransmitter is formed in the postsynaptic cell and released and then binds to presynaptic receptors, resulting in presynaptic inhibition [2]. The psychoactive effects of marijuana are mediated by THC binding to the endogenous cannabinoid receptor CB1 [5]. The other most abundant cannabinoid in marijuana is cannabidiol (CBD). CBD does not have the same psychoactive effects as THC. There is some emerging evidence suggesting CBD has both anxiolytic and antipsychotic properties [6, 7].

Other cannabis derivatives, such as synthetically derived cannabinoids, include a number of man-made compounds that interact with the endocannabinoid system. There are currently two FDA-approved synthetic cannabinoids, nabilone and dronabinol, which both act as THC analogs. Nabilone is approved for refractory chemotherapy-induced nausea and vomiting. Dronabinol is approved for AIDS-associated anorexia and chemotherapy-induced nausea and vomiting. Epidiolex, a plant-derived cannabidiol, has also been FDA approved for the treatment of seizures associated with Dravet syndrome and Lennox-Gastaut syndrome. Although the term "synthetic cannabinoids" technically refers to any man-made cannabinoids, the term often refers to illicit synthetic cannabinoids that are frequently abused. Recently, there has been a large increase in the production and illegal distribution of synthetic cannabinoids with psychoactive properties. These substances are primarily used for their cannabis-like psychoactive effects [8].

Cannabis is derived primarily from two sources. The first is marijuana, derived from the cannabis plant. There are many street names for marijuana, including pot, grass, dope, Mary Jane, weed, ganja, and hashish. Solvent-extracted concentrates of cannabis are called hash oil dabs, wax, and honey oil, among other names. Marijuana can be smoked, vaporized, or ingested. The dried flower heads can be smoked, or more concentrated preparations can be vaporized and inhaled. The onset of action is rapid, with psychotropic effects occurring within minutes and lasting about 2–3 hours. Alternatively, cannabis can be ingested for medicinal or recreational use. The psychoactive effects after oral ingestion are delayed compared to inhalation, with onset of action between 30 minutes and 3 hours, and duration up to 12 hours. The bioavailability of ingested cannabis is low because of extensive first-pass metabolism in the liver and inactivation by the acid environment in the stomach.

The second source of cannabis is synthetic cannabinoids. While technically outlawed by the Drug Abuse Prevention Act of 2012, cannabimimetic substances are often produced covertly, and legality is circumvented by altering the chemical compound slightly, thus making it a new substance not technically covered by the Drug Abuse Prevention Act. Typically, synthetic cannabinoids are dissolved in a solvent, applied to plant material, and smoked. They may be preferred over marijuana due to the relative ease of procurement, usually lower price, increased potency, limited

detection in drug screening, and perceived safety of use [9, 10]. Street names for these products are numerous and include K2, Spice, White Rhino, Bliss, Blue Bombay, Genie, Zoh, or Scooby Snacks [11]. Illicit synthetic cannabinoids are largely potent agonists of CB1 receptors and are often far more dangerous than marijuana, with risks of severe tachycardia, hypertension, seizures, and acute kidney failure [12]. Synthetic cannabinoids are most often smoked and have a rapid onset of action. The duration of intoxication is variable, reflecting the large heterogeneity of synthetic cannabinoids, but it typically ranges from 1 to 24 hours [13].

Cannabis use disorders are comorbid with multiple psychiatric illnesses, including bipolar disorder, several personality disorders, and other substance use disorders [14]. Among patients with anxiety disorders, cannabis is the most commonly used substance [15]. Individuals with schizophrenia also have high rates of cannabis use, which has been associated with increased rates of psychotic symptoms [16]. Synthetic cannabinoids have been reported in subgroups of individuals with schizophrenia, including those in transitional housing shelters [9]. Cannabis use is also highly comorbid with tobacco use and is associated with increased risk of developing alcohol use disorder and worse clinical outcomes [17].

DSM-5 Criteria

In a discussion of cannabis use disorders, it is important to have a clear understanding of the diagnostic criteria. The DSM-5 includes three diagnoses related to cannabis with criteria listed below [18].

I. Cannabis intoxication:

   (i) Recent use of cannabis
   (ii) Clinically significant problematic behavioral or psychological changes that developed during or shortly after cannabis use
   (iii) At least two of the following signs developing within 2 hours of cannabis use:

      1. Conjunctival injection
      2. Increased appetite
      3. Dry mouth
      4. Tachycardia

II. Cannabis use disorder (mild, moderate, or severe):

   (i) Cannabis is often taken in larger amounts or over a longer period than was intended.
   (ii) There is a persistent desire or unsuccessful effort to cut down or control cannabis use.
   (iii) A great deal of time is spent in activities necessary to obtain cannabis, use cannabis, or recover from its effects.
   (iv) There is craving or a strong desire or urge to use cannabis.

(v) Recurrent cannabis use results in a failure to fulfill major role obligations at work, school, or home.
(vi) There is continued cannabis use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of cannabis.
(vii) Important social, occupational, or recreational activities are given up or reduced because of cannabis use.
(viii) There is recurrent cannabis use in situations in which it is physically hazardous.
(ix) Cannabis use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by cannabis.
(x) There is tolerance, as defined by either (1) a need for markedly increased cannabis to achieve intoxication or desired effect or (2) markedly diminished effect with continued use of the same amount of the substance.
(xi) There is withdrawal, as manifested by either (1) the characteristic withdrawal syndrome for cannabis or (2) taking of cannabis to relieve or avoid withdrawal symptoms.
(xii) The severity of cannabis use disorder can be graded as mild with 2–3 symptoms, moderate with 4–5 symptoms, or severe with the presence of 6 or more symptoms.

III. Cannabis withdrawal:

(i) Cessation of cannabis use that has been heavy and prolonged (i.e., usually daily or almost daily use over a period of at least a few months)
(ii) Three (or more) of the following signs and symptoms develop within approximately 1 week after the above criteria:

1. Irritability, anger, or aggression
2. Nervousness or anxiety
3. Sleep difficulty (e.g., insomnia, disturbing dreams)
4. Decreased appetite or weight loss
5. Restlessness
6. Depressed mood

Cannabis Intoxication

The clinical manifestations of cannabis intoxication are broad and vary according to a number of clinical and substance-related factors, including formulation of cannabis, potency, method of administration, age of user, co-ingestion with other substances, and comorbid psychiatric illnesses.

Cannabis affects multiple organ systems with prominent neuropsychiatric effects, including changes in mood, cognition, perception, and psychomotor performance.

Cannabis can produce a euphoric effect that can be achieved at low dosages and is often described as the marijuana "high." Users may also report a feeling of decreased anxiety, decreased tension, and increased sociability [19]. Dysphoric reactions may occur with increases in anxiety or transient psychotic symptoms, including paranoia, thought broadcasting, hallucinations, and depersonalization. These symptoms generally occur at higher dosages, or in users with pre-existing psychiatric illnesses, such as psychotic or anxiety disorders. Cannabis can also heighten sensory experiences, making colors appear more vivid and tactile perceptions more intense [19]. Temporal perception may also be affected, with time appearing to progress more slowly. Cannabis intoxication has effects on cognition and psychomotor performance, including decreased concentration and impairment in reaction time and short-term memory. Long-term memory is not typically affected [20]. The cognitive effects of cannabis can lead to difficulty in completing tasks requiring divided attention, such as driving or operating heavy machinery [19, 21, 22].

Synthetic cannabis intoxication can cause different neuropsychiatric symptoms and medical complications. Clinical effects of synthetic cannabinoids are often unpredictable, due to inconsistent dosing, variable potency within each product, and wide variation in the specific chemical make-up of synthetic cannabinoids with differing cannabinoid receptor affinities. Users describe positive effects of relaxation, increased sociability, and increased laughter, while negative effects may include nausea, vomiting, aggression, hallucinations, chest pain, and palpitations [9]. Synthetic cannabinoids can also cause severe psychiatric effects, including agitation, delirium, and psychosis [12]. Conversely, CNS depression, lethargy, and disorientation have also been observed [23].

In some individuals, cannabis intoxication, either from marijuana or synthetic agents, can precipitate a substance-induced psychotic disorder in which psychotic symptoms persist beyond the acute intoxication period up to several months (see Chap. 7 for further details) [24]. In individuals with underlying psychotic disorders, the use of cannabis or synthetic cannabinoids can exacerbate symptoms or trigger recurrence of psychosis [25].

In children, intoxication most commonly occurs after accidental ingestion of edible forms of marijuana intended for adult use, including cookies, candies, or brownies, although direct administration from parents and caregivers has also been reported [26]. Symptoms of acute intoxication in children vary depending on the degree of exposure, but can include neurologic symptoms, such as lethargy, ataxia, and seizures [27, 28]. If large amounts of cannabis are ingested, prolonged lethargy, coma, or hypoxia may occur [29–31].

Withdrawal

While the syndrome of cannabis withdrawal has been supported by clinical observation and in research studies, it was first recognized as a distinct clinical syndrome only in the latest *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5).

Cannabis withdrawal, as defined in DSM-5, is the development of at least three signs or symptoms within 1 week after cessation of heavy and prolonged cannabis use, including (1) irritability, anger, or aggression; (2) nervousness or anxiety; (3) sleep difficulty (e.g., insomnia, disturbing dreams); (4) decreased appetite or weight loss; (5) restlessness; (6) depressed mood; and (7) at least one of the following physical symptoms causing significant discomfort: abdominal pain, shakiness/tremors, sweating, fever, chills, or headache [18].

Acute cannabis withdrawal occurs most commonly in heavy users and has been observed in more than half of individuals presenting for the treatment of cannabis use disorder [32]. Symptoms of cannabis withdrawal typically emerge 1–3 days after cessation of use and peak after 2–6 days. Withdrawal symptoms typically last for 1–2 weeks, although symptoms can persist up to 4 weeks in some individuals [33]. Biologically, chronic cannabis use leads to a downregulation of CB1 receptors, due to increased agonist activation with exogenous cannabinoids. When cannabis use is discontinued, exogenous agonist activation is removed, leaving CB1 receptors in a hypoactive state, which presumably mediates withdrawal symptoms. As cessation from cannabis continues, CB1 receptors return to the pre-cannabis state within 4 weeks, and withdrawal symptoms abate [34].

Relative to other substance withdrawal syndromes, cannabis withdrawal is comparable to tobacco withdrawal in magnitude [35]. Although relatively mild, cannabis withdrawal can lead to functional impairments in daily activities and is associated with relapse of cannabis use [36]. Furthermore, cannabis withdrawal can serve as a negative reinforcement for sobriety, as attempts at abstinence are frequently followed by uncomfortable withdrawal symptoms [37].

Due to the psychiatric and neuro-vegetative symptoms associated with cannabis withdrawal, it can be challenging to differentiate symptoms from potentially co-occurring mood and anxiety disorders from those of the withdrawal state. Longitudinal history and observation over time can be useful in making this diagnostic distinction. Synthetic cannabinoids can have more severe withdrawal symptoms, with agitation, irritability, anxiety, mood swings, and insomnia, and may emerge as quickly as 15 minutes after use [38].

Medical Comorbidities of Cannabis Use

Presentations to the emergency department (ED) for symptoms associated with acute intoxication from cannabis alone are uncommon. Life-threatening complications are infrequent, although fatalities have been reported after cannabis use due to acute coronary syndrome and tachyarrhythmias [39]. Among patients who do present to the ED, common complaints or findings include agitation and aggressive behavior (22%), psychosis (20%), anxiety (20%), and vomiting (17%) [40].

Cannabis hyperemesis syndrome (CHS) is a cluster of symptoms characterized by cyclic vomiting, nausea, and abdominal pain, with no clear etiology, other than a presumed, paradoxical effect of chronic cannabis use. Symptoms typically improve with cessation of cannabis use or with hot showers and baths [41]. Acute treatment

consists of administration of antiemetics, intravenous fluids, and benzodiazepines. In treatment refractory CHS, case reports indicate that using intravenous haloperidol at doses of 5 mg can be successful in reducing symptoms [42].

Patients using inhaled preparations of cannabis can develop pulmonary complications from the irritation of the inhaled smoke and increased intrathoracic pressures secondary to breath-holding during inhalation. Acute asthma exacerbation, bronchospasm, pneumothorax, and pneumomediastinum have all been reported after inhalation of marijuana. Unless obtained from an authorized dispensary, marijuana may contain contaminants, as well as other psychoactive compounds, and may induce allergic or bronchospastic reactions when the smoke is inhaled. In immunocompromised patients, inhalation of biologically contaminated marijuana may inadvertently lead to pulmonary infections [43].

Although rare, ischemic chest pain and myocardial infarction have been reported after marijuana use [39]. Again, if the preparation is contaminated with other compounds that are sympathomimetic, such as cocaine and methamphetamine, coronary ischemia can result from ingestion [44].

Children can present after cannabis ingestion with altered mental status that can progress to somnolence and coma. Depressed respirations and, in rare cases, apnea have been seen [27]. Accidental oral ingestions are more common in states where cannabis has been legalized for recreational use. These states have products available that mimic candies, cookies, and brownies, which children can ingest accidentally if the products are not carefully secured [45].

Patients who use synthetic cannabinoids are at risk of more serious medical complications. Synthetic preparations may be adulterated with other psychoactive chemicals, including amphetamines, cathinones, 3,4-methylenedioxymethamphetamine (MDMA), and other ingredients, all with significant sympathomimetic action. The most common complications from synthetic cannabinoid use include neuropsychiatric symptoms, including agitation, coma, psychosis, seizures, and delirium (66%); cardiovascular changes, including hypertension, tachycardia, or bradycardia (17%); and other systemic symptoms, including rhabdomyolysis (6%) and acute kidney injury (4%) [46].

Cannabis withdrawal is rarely life threatening; however, synthetic cannabinoid withdrawal may lead to seizures, kidney failure, and dyspnea [47, 48]. Synthetic cannabinoid withdrawal can also cause sympathetic autonomic hyperactivity, with associated hypertension, tachycardia, and diaphoresis. It may be difficult to differentiate if the physiologic effects are due to synthetic cannabinoid intoxication or withdrawal, as symptoms often overlap. Assessing for psychiatric symptoms of synthetic cannabinoid intoxication and determining time of last use may be helpful in differentiating the two.

Assessment and Management in the ED

The ED evaluation of the cannabis-using patient should clarify the history of cannabis use, including time of last use, chronicity and quantity of use, route of administration, formulation or type of cannabis, and co-ingestion with other substances.

The DSM-5 criteria previously discussed can help the clinician assess the potential presence and severity of a cannabis use disorder. Evaluation of psychotic, anxiety, or mood symptoms (either due to or comorbid with cannabis use) should also occur. All patients must be assessed for suicidality and homicidality. The mental status exam may be significant for conjunctival injection, odor of cannabis, disorganized thought process, persecutory delusions, thought broadcasting, referential thinking, anxiety, or euphoric mood. Collateral information from friends or family may be helpful, especially if the patient's ability to give a reliable history is compromised.

All patients presenting to the ED with cannabis-related complaints will benefit from a targeted medical evaluation. Obtaining vital signs, a physical examination, and routine laboratory studies, including urine toxicology screening, can aid in ascertaining medical stability.

In emergency settings, there are diagnostic challenges to identifying the patient who is using cannabis but not reporting it. Unfortunately, there is no readily available test for synthetic cannabinoids. The most commonly available urine toxicology screens are specific for THC and do not detect the other synthetic cannabinoids. In addition, for chronic cannabis users, a positive screen for THC can persist for days or weeks after use and long after the acute psychoactive effects have worn off. Thus, a patient's presenting symptoms may be attributed to cannabis use but, in fact, be unrelated. It is important to think critically about diagnosing a patient with cannabis intoxication based on a positive THC screen alone, especially when the clinical findings are atypical. A thorough history, physical exam, and observation over time may aid in clarifying acute intoxication versus prior cannabis exposure.

Laboratory Testing and Imaging

The most common screening tool for cannabis is the urine drug screen, consisting of an immunoassay to detect the metabolites of THC, primarily 11-*nor*-Δ-THC-9-carboxylic acid (THC-COOH). The detection time for cannabis in the urine is variable and is related to amount ingested, route of administration, frequency of use, THC content of cannabis, and cut-off value of the urine drug test. THC is highly lipophilic and can be stored in adipose tissue, where it is released into circulation over time. The mean detection time for smoking one marijuana cigarette is 1–2 days, while heavy users may be positive for THC up to 27 days [49]. As a result, a positive test may be helpful in identifying past use but does not always reflect recent cannabis exposure. False positives for cannabinoids with current immunoassays are typically rare but may include the synthetic cannabinoid dronabinol.

Detection of illicit synthetic cannabinoid exposure can be challenging, as synthetic cannabinoids do not typically share similar structures with THC or THC metabolites that are detected in standard immunoassays. While some immunoassays are available to detect common synthetic cannabinoids and their parent compounds, availability in most ED settings is limited [50]. As a result, the diagnosis of synthetic cannabinoid exposure is often based on clinical history.

If symptoms are atypical or if the diagnosis is unclear, evaluation for other medical or substance-related etiologies may be indicated. The differential diagnosis of patients presenting with signs and symptoms of acute cannabis intoxication is wide and can include blood sugar abnormalities (either hypoglycemia or hyperglycemia), metabolic disarray, CNS infection, carbon monoxide poisoning, other drug ingestions (sedatives, opioids, ethanol, etc.), or covert brain injury. Laboratory studies, including CBC, electrolytes, liver function tests, and urinalysis, can help exclude other medical etiologies. Imaging studies including head CT or MRI are not typically indicated, unless there is a suspicion for underlying neurologic injury.

Management of Intoxication

Cannabis intoxication rarely requires medical treatment. In more severe cases of intoxication, distressing psychiatric symptoms can occur and can lead to presentations for psychosis, mood disturbances, and anxiety symptoms. Management of the psychiatric effects of cannabis intoxication is largely supportive, and, depending on comorbidities, pharmacologic intervention is often unnecessary. If psychiatric symptoms are mild to moderate, behavioral and environmental interventions can be used, such as keeping the patient in a quiet environment with supportive reassurance.

More severe cases of intoxication with marked agitation or psychosis are more likely to be seen in emergency settings. In these cases, benzodiazepines or antipsychotics can be administered PO, IM, or IV for behavioral disturbances [51]. While there is a lack of vigorous clinical studies supporting a clear pharmacologic choice, benzodiazepines are generally preferred to antipsychotics for milder symptoms of anxiety or agitation, while antipsychotics are used to treat more overt psychotic symptoms or severe behavioral dysregulation. Psychotic symptoms associated with cannabis intoxication typically respond to low-dose first- or second-generation antipsychotics, including haloperidol, risperidone, or olanzapine. Hospital police and security presence can be helpful in managing violent behavior, and mechanical restraints can be used as a last resort to protect the safety of patients and others. If behavioral symptoms are severe and persist despite adequate trials of benzodiazepines and antipsychotics, sedation with other agents, including ketamine or dexmedetomidine, can be used with caution [52].

Management of Withdrawal

Cannabis withdrawal is rarely life threatening and does not usually require medical attention. In contrast, synthetic cannabinoid withdrawal can cause dyspnea, sympathetic autonomic hyperactivity with associated hypertension and tachycardia, seizures, and kidney failure [47, 48]. Medical management is dictated by the severity of the presenting symptoms.

Cannabis withdrawal can produce psychiatric symptoms of anxiety, insomnia, loss of appetite, irritability, and mood changes. Synthetic cannabinoids can have more severe withdrawal symptoms, with agitation, irritability, anxiety, mood swings, and insomnia. In the emergency setting, low-dose benzodiazepines can be used for withdrawal symptoms causing severe anxiety or distress.

Disposition Considerations

Cannabis intoxication or withdrawal by itself rarely requires inpatient admission. During periods of intoxication, patients may present with a variety of psychiatric concerns, including psychosis, mood disturbances, and anxiety symptoms, that typically resolve with sobriety. When clinically sober, the patient should be re-evaluated, to determine which, if any, symptoms remain, as these remaining symptoms will influence ultimate disposition. A thorough safety assessment should be performed prior to discharge in all patients presenting with suicidality, homicidality, psychosis, or significant behavioral disturbances. Furthermore, cannabis intoxication can affect cognition, psychomotor performance, and judgment and may affect the patient's ability to be safely discharged if driving a car or traveling alone [19, 21, 22]. The time needed to achieve sobriety can vary significantly and depends on a number of factors, including potency of cannabis product used, mode of ingestion, and co-ingestion with other substances.

Psychiatric admission should be considered for those unable to be safely discharged due to ongoing suicidality, homicidality, or persistent psychosis leading to inability to care for themselves. Additionally, if the patient had severe agitation requiring large amounts of sedating medications or restraints, psychiatric admission may be needed to ensure safety and further stabilization. For individuals with psychotic disorders, such as schizophrenia, worsening of psychosis can occur after cannabis or synthetic cannabinoid use. If acute psychotic symptoms persist beyond intoxication, psychiatric admission may be warranted. Cannabis withdrawal in the absence of other significant psychopathologies is largely managed in an outpatient setting. Admission for cannabis detoxification is rarely indicated.

When assessing the need for referral to outpatient treatment, a careful assessment of cannabis use and potential comorbid psychiatric illness should be performed. Individuals with cannabis use disorder or psychiatric illness can be provided with referrals to outpatient substance use disorder and/or psychiatric treatment. The referral should be appropriate to the patient's level of motivation and interest in treatment. Individuals with more severe cannabis use disorders can be referred to partial hospitalization or intensive outpatient programs for substance use disorder treatment where available. Information on self-help groups, such as Marijuana Anonymous or SMART Recovery, can be provided, although their effectiveness has not been rigorously studied.

Rarely do patients require hospital admission for medical complications of cannabis. Possible medical complications prompting admission may include

myocardial infarction, cannabis hyperemesis, seizures, renal failure, and pneumothorax. In pediatric cannabis exposure, observation or medical admission may be necessary, given potential risks of coma, seizures, and hypoxia [28–31].

Conclusion

In summary, cannabis has a long history of human consumption, and it is thought to be the most commonly used illicit psychoactive substance worldwide. The cannabis plant is composed of a number of cannabinoids that interact with the body's endogenous cannabinoid system. The most abundant psychoactive cannabinoid in cannabis is THC, which is responsible for its psychoactive effects. Synthetic cannabinoids also interact with the endogenous cannabinoid system and have a variety of psychoactive and physiologic effects, reflecting the large heterogeneity in compounds. Intoxication with cannabis often produces euphoria, perceptual changes, but also distressing symptoms of anxiety, paranoia, or worsening of underlying psychiatric illness. In the ED, patients may present with the more distressing symptoms of intoxication, prompting psychiatric assessment. Intoxication with cannabis is often self-limited and improves with supportive treatment or limited use of benzodiazepines or antipsychotics. In some individuals, including those with psychotic disorders, cannabis or synthetic cannabinoid intoxication may lead to worsening of underlying psychiatric illness. Medical consequences of cannabis or synthetic cannabinoid intoxication are rare and not often life threatening. As the legal status of marijuana continues to be in transition, clinicians may begin to encounter an increasing number of individuals with cannabis intoxication and its related emergencies.

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