Recently, reports have suggested that chronic cannabis abuse can result in cyclical vomiting, or cannabinoid hyperemesis syndrome. With the increasing prevalence of cannabis use in the United States, this syndrome may be encountered in the emergency department. The authors describe a case of a 30-year-old man who presented to the emergency department with diffuse abdominal pain, nausea, and intractable vomiting. He reported symptomatic relief with prolonged hot showers. Results of a urine drug screen were positive for cannabis, and the patient admitted to chronic cannabis use for years. Results of the drug screen, combined with the patient’s symptomatic relief with hot showers, led to the diagnosis of cannabinoid hyperemesis syndrome. The patient was admitted to the hospital and underwent pharmaceutical treatment. However, hot showers continued to be the mainstay of the patient’s symptomatic relief. Four days after presentation, the patient’s symptoms resolved and he was discharged from the hospital.

Several mechanisms have been proposed to explain the pathophysiology of cyclical vomiting, but the etiology remains unclear. Although cannabinoids have long been recognized for their therapeutic potential as antiemetics, chronic cannabis use has recently been associated with recurrent vomiting. Patients with this disorder, termed cannabinoid hyperemesis syndrome, also experience abdominal pain, polydipsia, and a desire to take repeated hot showers.

The diagnosis and management of cannabinoid hyperemesis syndrome is of clinical importance because cannabis use is so widespread. Marijuana is the most commonly used illegal drug in the United States, with a prevalence rate of roughly 4%. In 2009, 16.7 million US individuals aged 12 years or older had used marijuana at least once in the month prior to being surveyed. In the absence of other possible causes, including pancreatitis, gastroenteritis, nephrolithiasis, pyelonephritis, cholecystitis, peptic ulcer disease, pyloric obstruction, gastroparesis, and pregnancy, this syndrome should be considered in treating patients with cyclical vomiting as it may be underrecognized.

We describe the case of a man who sought emergency care for uncontrolled nausea and vomiting. We also provide a brief review of the literature on cannabinoid hyperemesis syndrome.

**Report of Case**
A 30-year-old previously healthy man with a white collar job presented to the emergency department with diffuse abdominal pain accompanied by nausea, intractable vomiting, and decreased oral intake of food and drink. The patient was dehydrated at presentation and indicated his symptoms had lasted for 3 days. The patient denied any previous episodes and reported symptomatic onset with ingestion of “several Budweisers.” He also reported symptomatic relief with prolonged hot showers. He denied eating anything out of the ordinary, encountering other unhealthy individuals, traveling recently, or feeling sick prior to symptom onset. His past medical and surgical histories were unremarkable, and he had no reported allergies. He denied tobacco or other illicit drug use. He noted a subjective weight loss of 10 lbs.

On physical examination, the patient’s vital signs included a temperature of 35.2°C, heart rate of 52 beats per minute, respiratory rate of 16 breaths per minute, blood pressure of 135/76 mm Hg, oxygen saturation of 98% on room air, and weight of 80 kg. He was irritable and agitated with intractable hiccupping. He had mild flushing of the integument on the anterior thorax. His pupils were equal in diameter and reactive to light, and extraocular movements were intact. Examination of the patient’s mouth and oropharynx revealed moist mucous membranes. No discernable lymphadenopathy or thyromegaly was present. The patient’s cardiovascular examination was unremarkable and his chest was clear on auscultation. Examination of the abdomen revealed soft tissue with diffuse ten-
deness, positive bowel sounds, and no signs of organomegaly, rebound tenderness, or guarding. Extremities showed no signs of clubbing, cyanosis, or edema. Findings of a neurologic examination were unremarkable.

Laboratory findings revealed an elevated anion gap, hyponatremia, hypochloremia, elevated bicarbonate levels, and acute renal insufficiency with elevated blood urea nitrogen (Table). Fractional excretion of Na was 2.27%. Liver transaminase, lipase, and amylase levels were normal. Urinalysis results were positive for trace ketones from decreased food intake. Ultrasonography of the kidneys showed no evidence of hydronephrosis or obstruction.

Results of the patient’s urine drug screen were positive for cannabis. When confronted with the positive urine drug screen, the patient admitted to last using cannabis 3 days prior to admission, with history of chronic daily use for years. Results of the drug screen, combined with the patient’s description of symptomatic relief with hot showers, led to the diagnosis of cannabinoid hyperemesis syndrome due to chronic cannabis abuse.

The patient was admitted to the hospital and immediately treated with intravenous fluids, including normal saline (2 L bolus and then 200 mL/hr) and famotidine (20 mg). Within the next 2 hours, the patient experienced minimal relief as we administered ondansetron hydrochloride for nausea and vomiting (4 mg as needed), morphone for pain (4 mg as needed), droperidol (5 mg), diphenhydramine hydrochloride (50 mg), promethazine hydrochloride (12.5 mg as needed), chlorpromazine hydrochloride for resolution of intractable hiccups (25 mg as needed), and acetaminophen for headache (650 mg as needed). Except for the acetaminophen, all drugs were administered intravenously.

The patient vomited several times overnight and nausea was still present the next morning. However, the patient reported that his headache had resolved and abdominal pain was improving. In addition, the patient’s dehydration, relative polycythemia, and leukocytosis had improved, and his hiccups had resolved. The patient took multiple hot showers on this day.

The next day, the patient continued to vomit despite continued treatment with ondansetron hydrochloride. He also reported continued abdominal pain. At this point, we switched his nausea treatment to promethazine hydrochloride (first line, 12.5 mg as needed), lorazepam (second line, 1 mg every 4 hours as needed), and ondansetron hydrochloride (third line, 4 mg as needed). We administered another 1 L bolus and resumed intravenous fluids at 150 mL per hour. We let the patient continue to take hot showers as needed.

By the next morning (the fourth day of hospitalization), patient felt much better. His abdominal pain had resolved and he denied any nausea or vomiting; the last episode of vomiting had occurred the previous night. Pain was controlled with morphine, and nausea was controlled with promethazine hydrochloride and lorazepam. Ondansetron hydrochloride was not administered, despite being available in the instance that the patient needed it.

Hot showers—19 throughout the course of hospitalization—were the mainstay of the patient’s symptom relief. He had episodes of perspiration, extreme thirst, flushing, and episodes of low-grade fever after showering. The patient did not respond well to antiemetics but intravenous fluids and hot showers seemed to calm the active phase of his disease process. Results of his blood and chemistry panels further supported this improvement. Liver, pancreas, and renal pathologies were dismissed as potential causes because of normal enzyme levels and ultrasonography readings.

We conducted research simultaneously with presentation of this obscure case, which helped us determine the patient’s treatment. The patient was discharged on the fourth day of hospitalization, at which point his symptoms had resolved. Before discharge, the patient was educated about symptoms of cannabinoid toxicity and how abstaining from cannabis could resolve his symptoms. He was also informed that, while the disease takes years to develop, it would resurface within weeks of resuming cannabis, even after considerable periods of abstinence.1,8 The patient was not followed up.

### Clinical Presentation

Cannabinoid hyperemesis syndrome is a new and emerging clinical diagnosis that is often overlooked in the emergency

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**Table. Laboratory Findings for a 30-Year-Old Man With Cannabinoid Hyperemesis Syndrome**

<table>
<thead>
<tr>
<th>Laboratory Test</th>
<th>Finding</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>White Blood Cell Count, × 10⁶/μL</td>
<td>19.0</td>
<td>4.0-11.0</td>
</tr>
<tr>
<td>Red Blood Cell Count, × 10⁶/μL</td>
<td>5.89</td>
<td>4.60-6.20</td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>17.7</td>
<td>14.0-18.0</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>49.1</td>
<td>42.0-52.0</td>
</tr>
<tr>
<td>Blood Platelet Count, × 10⁹/μL</td>
<td>492</td>
<td>150-450</td>
</tr>
<tr>
<td>Segmented Neutrophils, K/μL</td>
<td>14.3</td>
<td>1.8-8.0</td>
</tr>
<tr>
<td>Anion Gap, mmol/L</td>
<td>24</td>
<td>8-16</td>
</tr>
<tr>
<td>Osmolality, mmol/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium, mmol/L</td>
<td>130</td>
<td>132-146</td>
</tr>
<tr>
<td>Chloride, mmol/L</td>
<td>67</td>
<td>99-109</td>
</tr>
<tr>
<td>Bicarbonate, mmol/L</td>
<td>39</td>
<td>20-31</td>
</tr>
<tr>
<td>Nitrogen, mg/dL</td>
<td>67</td>
<td>9-23</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>3.2</td>
<td>0.5-1.3</td>
</tr>
</tbody>
</table>

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department because adverse effects of chronic cannabinoid use are not always recognized. The syndrome, characterized by a triad of chronic cannabis use, cyclical vomiting, and compulsive hot bathing, is divided into 3 phases: prodromal, recurrent vomiting, and recovery.2

Prodromal phase2—The prodromal phase is variable in duration and manifests with nausea, fear of vomiting, and abdominal discomfort. Symptoms are most common in early middle-aged adults who have consistently been using cannabis since adolescence.

Vomiting phase2—The vomiting phase is characterized by intense, persistent nausea, vomiting, and retching that can occur up to 5 times per hour and is often described by patients as overwhelming. In addition, patients experience concomitant abdominal pain and a compulsive need to bathe in hot water. Patients typically visit hospitals numerous times throughout this phase, which can lead to escalating healthcare costs. Results of esophagogastroduodenoscopy, colonoscopy, radiologic abdominal imaging, ultrasonography, and further workup are generally unremarkable. Routine blood tests are often unrevealing as well.

Recovery phase2—The recovery phase begins with cannabis cessation. Within 1 week, patients will experience a substantial decrease in and eventual resolution of vomiting. Symptoms are further alleviated with 24 to 48 hours of intravenous fluid administration. Despite reports of patients with this condition being refractory to supportive antiemetic regimens (such as in the present report), pharmaceutical management can relieve symptoms of pain and nausea in some cases. The tendency to take hot showers also subsides during the recovery phase. Return to cannabis use at any time will lead to recurrence of cannabinoid hyperemesis syndrome.

Etiology

Despite proposed mechanisms of cannabinoid hyperemesis syndrome, the etiology remains unclear.1,2 Cannabis has been used to treat chemotherapy-induced nausea and vomiting, anorexia, anxiety, and glaucoma.9 The antiemetic effect of cannabinoids is mediated by cannabinoid type 1 receptors in the brain by means of Δ⁸-tetrahydrocannabinol, the active compound in cannabis.4,10 Proposed mechanisms of cannabinoid hyperemesis syndrome include toxicity because of marijuana’s long half-life, cumulative lipophilic effects in the brain, delayed gastric emptying, and thermoregulatory and autonomic disequilibrium in the limbic system.8,4,10

Vomiting is coordinated by the brainstem in response to noxious stimuli involving many neurotransmitters. Metoclopramide, an antiemetic agent, acts as an antagonist to the same chemoreceptor trigger zone in the brainstem that inhibits the vomiting reflex. It also increases gastric motility, further decreasing the emetic response.9,11,12 In patients who develop hyperemesis, the enteric emetic effects of cannabis, such as decreased gastrointestinal motility, may promote emesis by overriding antiemetic effects mediated by the central nervous system.2,13

As previously mentioned, patients with cannabinoid hyperemesis syndrome shower or bathe in hot water to decrease the intensity of the nausea and vomiting. A series of cases in South Australia, Australia, demonstrated that simultaneous with hot bathing, patients lost 10 to 15 lbs of body weight, which they regained after they abstained from cannabis and their symptoms resolved. They also displayed a variety of autonomic symptoms including sweating, flushing, thirst, alteration in body temperature, and colicky abdominal pain.5 This effect might arise from the modulation of the hypothalamic-pituitary-adrenal axis by endocannabinoids.8,14 Cannabis toxicity may disrupt the balanced equilibrium of satiety, thirst, digestion, and the thermoregulatory systems of the hypothalamus.8 This disruption may be settled with hot bathing or showering.8

Conclusion

Cannabinoid hyperemesis syndrome is a newly recognized diagnosis related to cannabis toxicity. The adverse effects of chronic cannabis use are still under investigation, and the mechanism of cannabis leading to intractable nausea and vomiting is still unclear.1,2 With the widespread use of this substance, both recreationally and therapeutically, the paradoxical effect of cannabinoid hyperemesis syndrome deserves further attention.

The goal of the present case report is to raise awareness of the potential adverse effects of cannabis and the importance of obtaining a thorough history, including questions regarding illicit substance abuse. When questioning a patient’s social history, it may be worthwhile to inquire about hot showering patterns, especially in those who deny or minimize their use of illicit drugs. Clinicians should have heightened awareness when intractable nausea and vomiting is unresponsive to antiemetics but relieved with hot showering. When encountering this unusual presentation, clinicians should consider illicit cannabis abuse in addition to organic disease as a possible cause. This consideration may prevent further unnecessary workup and healthcare costs for patients with cannabinoid hyperemesis syndrome.

References


(continued)


