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Review on cannabinoid hyperemesis syndrome

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Abstract
The cyclic vomiting illness known as cannabinoid hyperemesis syndrome (CHS) is linked to cannabis consumption. Our goal is to compile the information that is currently accessible on the pathogenesis, diagnosis, and therapy of CHS. After being first reported in 2004, cannabinoid hyperemesis syndrome (CHS) is still a clinical condition that affects chronic heavy marijuana users but is not well understood. Treatment for the hyper emetic phase includes supportive therapy, fluid resuscitation, and antiemetic medicines; this phase usually ends in 48 hours. Individuals frequently exhibit the acquired habit of taking frequent hot baths, which results in a brief improvement in nausea, vomiting, and abdominal pain. Furthermore, research on humans and animals indicates that prolonged cannabis use may be necessary for the degree of emesis rather than being a requirement for the induction of vomiting.

Keywords: Cannabis, marijuana, cyclic vomiting, nausea, cannabinoid hyperemesis syndrome

1. Introduction
Cannabinoid hyperemesis is characterized as excessive marijuana usage along with cyclical vomiting and gastrointestinal pain. It was first reported by Allen1 in a 2004 case study including nine individuals in Australia [1]. Patients with cannabinoid hyperemesis syndrome (CHS), a paradoxical side effect of cannabis usage, frequently come with cyclical nausea, vomiting, and abdominal discomfort without any other apparent cause. As a result, they are discharged from the hospital with a variety of incorrect diagnoses [2]. After alcohol and tobacco, cannabis is the third most widely used narcotic and has been used recreationally for millennia [3]. Cannabis offers many medicinal benefits, such as hunger stimulation, pain relief, and anti-emesis. Due to these benefits, cannabis is now being used by patients who suffer from severe peripheral neuropathy, AIDS-related cachexia, chemotherapy-induced nausea and vomiting, and multiple sclerosis-related muscle spasms [4]. According to the Rome IV criteria, CHS is defined as stereotyped episodic vomiting that resolves with a sustained cannabis cessation following protracted, excessive cannabis usage with symptom onset more than six months [5]. As of June 2023, adult non-medical cannabis use is legal in 23 states, 2 territories, and DC; medicinal cannabis use is permitted in 38 states, 3 territories, and DC [6]. Over the past few years, an increasing number of publications detailing suspected occurrences of Cannabinoid Hyperemesis Syndrome (CHS) have been published globally. Patients with chronic cannabis usage who experience recurrent vomiting without a clear organic reason are said to have CHS [7]. According to UN estimates, 277 million people worldwide roughly 4.9% of the entire population have used cannabis [8]. Both the general people and the medical community typically believe that marijuana has anti-emetic qualities. Because so few medical professionals are familiar with CHS, it is uncommon to draw the link between persistent marijuana use and cyclical vomiting. Before the connection between extensive marijuana use and CHS is discovered, patients with the condition frequently endure years of suffering through multiple invasive procedures and medical tests [9]. Cannabis is FDA-approved for treating chemotherapy-related nausea and vomiting in cases where conventional therapy is ineffective. It has long been used as an appetite stimulant and antiemetic medication. It affects the gastrointestinal tract in a number of ways, including motility, secretions, appetite stimulation, and inflammatory modulation [10]. Previously believed to be uncommon, cannabinoid hyperemesis syndrome (CHS) is now observed nearly every day. Though the precise cause of the symptoms is unknown, the GI tract's endocannabinoid receptors are likely to be the culprit.

Those who smoke 20 or more times a month, or heavy users, are most frequently impacted [11]. Considering the widespread use of cannabis globally, the relatively recent identification of CH, and the dearth of research on the subject, it seems probable that this illness is not well understood and underdiagnosed. Patients and doctors may become frustrated because of intrusive and expensive diagnostic tests brought on by a lack of knowledge about the illness. To increase awareness and comprehension of this illness, we carried out the largest patient case series to date, Identified as having CH at our facility [12].

1.1 The classic features of CHS are the following: [13].

- Heavy, ongoing cannabis use,
- Frequent bouts of intense nausea and uncontrollably vomiting,
- Pain in the abdomen,
- Short-term symptom alleviation from hot baths or showers,
- The issue is resolved if cannabis use is ceased. There are milder forms of the disease, and a case without traits 3 and 4 has been reported.

1.2 Clinically there are three phases of CHS: [14].

1. **Prodromal phase:** In this phase patient presents with nausea without vomiting, anorexia, abdominal discomfort.
2. **Hyperemesis phase:** This phase can last for many days and is characterized by intractable vomiting, flushing, diaphoresis, abdominal pain. Weight loss is common. Hot showers are believed to relieve the symptoms. It is in this phase that more ED visits occur.
3. **Recovery phase:** Complete resolution of symptoms occurs during this phase and patient returns to baseline.

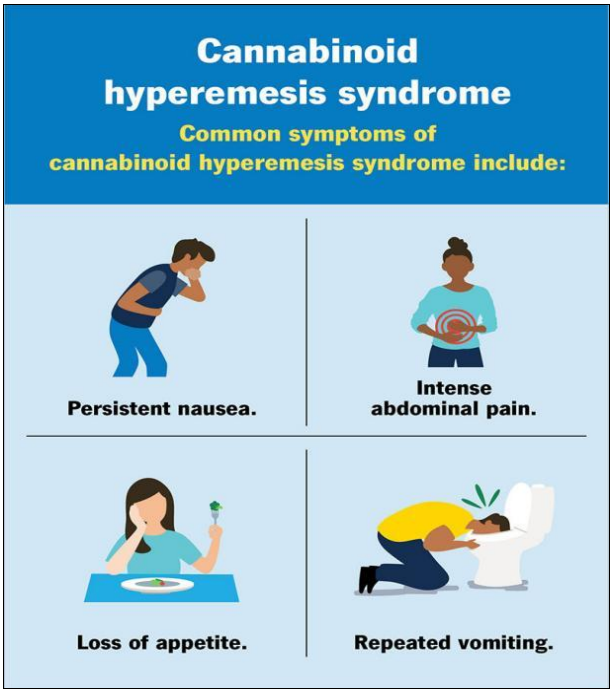

Fig 1: Common symptoms of CHS [15]

2. Causative agent

Cyclic vomiting syndrome, or CHS, is a largely unknown condition that affects long-term cannabis users. Patients are mostly male, have used cannabis for more than a year, and use it at least once a week, with daily users being most prevalent [16].

Fig 2: Cannabis (marijuana) [17].

3. Adverse Effect

Panic attacks are the emergency brought on most frequently by marijuana consumption. Dizziness, dry mouth, nausea, euphoria, disorientation, confusion, drowsiness, elevated heart rate, and breathing difficulties are among the most frequent side effects linked to marijuana usage.

1. Marijuana usage is linked to acute reversible psychotic responses and is responsible for 24% of newly diagnosed cases of psychosis in teenagers. Moreover, it has been shown to increase the likelihood of psychotic illnesses and exacerbate symptoms or cause relapses in people who already have psychotic disorders [18].
2. A number of research point to a link between marijuana inhalation and spontaneous pneumothorax as well as an increased risk of lung cancer from the drug. Furthermore, connected to bullous emphysema and chronic obstructive pulmonary disease consequences, such as increased wheezing, coughing, and phlegm production, are these conditions [18].
3. Chronic marijuana usage has also been linked to periodontal disease, a higher chance of preterm delivery if used before 20 weeks of pregnancy, and a higher frequency of pain episodes in sickle cell patients [18].
4. While drowsiness is a common side effect, cannabinoid formulations that just include CBD have less potential to trigger behavioural reactions. Since CBD is a CYP3A4 inhibitor, it may cause toxicities and medication interactions in substances that the CYP3A4 system metabolizes [19].
5. The benefits of topical capsaicin cream have been shown to be similar to those of hot water hydrotherapy. It is thought to reroute blood flow from the gastrointestinal system to the skin when applied to the belly. But the only way to completely alleviate the symptoms of hyperemesis is to stop using cannabis [20].
6. Another negative effect or side effect linked to long-term cannabis use is a motivational syndrome, which is clinically similar to depression plus a lack of motivation. There is a link between indifference and cannabis abusers, according to a new large-scale study [21].

4. Pathophysiology

Many theories that make use of cannabis pharmacokinetics and pharmacodynamics have been put forth to explain the pathophysiology of CHS. First, the primary active ingredient

in cannabis, delta 9-tetrahydrocannabinol (THC), tends to sequester in fat and slowly diffuse back into the serum [21]. Because THC binds to CB1 receptors in the central nervous system (CNS), which regulate sensory, somatic, and cognitive perception, it has impacts on emesis, appetite, and pain [20]. Preclinical research indicates that at least two G-protein-coupled cannabinoid receptors exist, CB1 and CB2, the functions of which are still being fully understood. CB2 is primarily found in peripheral lymphoid tissues, whereas CB1 is expressed in neurons of the central nervous system (CNS) [22]. At least 66 different cannabinoids have been identified in cannabis, with delta-9 tetrahydrocannabinol (THC) being the primary active component. In human tissues, THC binds to cannabinoid type 1 (CB1) and type 2 (CB2) receptors. The CB2 receptor is typically present outside the central nervous system, in immune tissues like the spleen, thymus, and different types of immune cells. The CB1 receptor is primarily restricted to neuronal tissue [4].

CB1 receptors are found in the basal ganglia, hippocampal regions, anterior cingulate gyrus, hypothalamus, and cerebral cortex of the brain. CB1 receptors are present on intrinsic and extrinsic neurons in the gastrointestinal tract, with the enteric nervous system acting as the primary site of action [23]. As an antagonist to the same chemoreceptor trigger zone in the brainstem that suppresses the vomiting reflex, metoclopramide is an antiemetic medication [24]. According to some research, hyperemesis may be caused by a slowdown in stomach motility caused by disruption of peripheral cannabinoid receptors in the enteric neurons [25]. The mechanism of cannabinoid hyperemesis is unknown. Marijuana, whether taken as a pill or smoked, has been used to treat chemotherapy-induced nausea and vomiting, glaucoma, anorexia, anxiety, and for purposes of muscle relaxation. Some known adverse reactions include altered sensorium, blurred vision, decreased coordination, dry mouth and eyes, hypotension or hypertension, sedation, somnolence, rash, urinary retention, and nausea and vomiting [26].

Heat and capsaicin activate the non-selective cation channel known as transient receptor potential vanilloid 1 (TRPV1). This receptor can be found in several sensory neurons, enteric neurons in the myenteric plexus, and gastric mucosal epithelial cells throughout the gastrointestinal tract [27]. Additionally, the central nervous system contains it, particularly in the postrema region, which is also referred to as the chemoreceptor trigger zone. The strong antiemetic action of activating this receptor is believed to be mediated by substance P being depleted from neuronal circuits that route to the nucleus tractus. Individuals without a job [27].

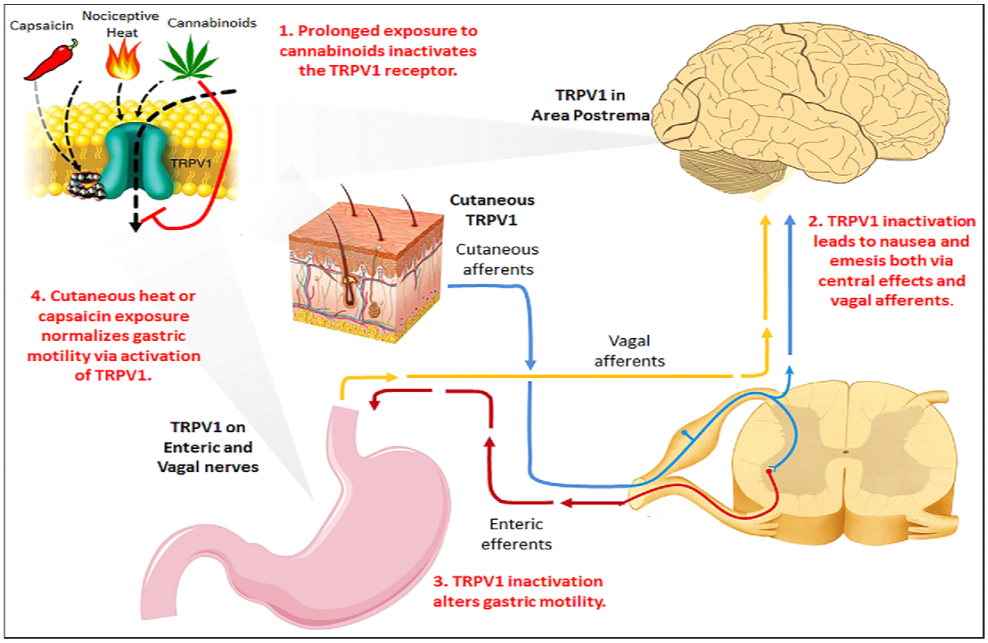

Fig 3: Diagrammatic representation of the pathophysiology of cannabinoid hyperemesis syndrome (CHS), TRPV1, transient receptor potential vanilloid 1 [28].

Uncertainty surrounds the pathophysiology of uncontrollably vomiting with CUD. The compulsive hot bathing habit observed in CHS patients may be explained by one of two possible processes. The patient may be forced to take hot showers because of the cannabinoid's ability to first induce a dose-dependent disequilibrium of the hypothalamic digestive and thermoregulatory systems. Secondly, this behavior may be the consequence of a rise in body temperature at the core combined with a fall in skin temperature, which triggered the hypothalamus and caused the patient to take frequent hot showers [29].

5. Diagnosis

CHS has typically been defined as the presence of Hyperemesis is related to chronic cannabis use. with abnormal hot bathing behavior. Nonetheless, there has Previous studies have shown significant heterogeneity when categorizing patients with this diagnosis. CHS involves three phases: prodromal, hyperemetic, and recovery. The prodromal phase involves nausea, stomach discomfort, and anxiety about vomiting [5]. Sustained monitoring of patients following cessation of cannabis use might aid in a precise diagnosis [30]. An important characteristic of CHS that is not highlighted in Rome IV is abdominal pain that usually originates in the epigastrium and radiates widely. It also usually occurs with the commencement of vomiting. Hot showers can help with both vomiting and stomach pain, maybe because they are relaxing and distracting. In between assaults, there are times of wellbeing or remission that might continue for days or weeks. If the entity is not treated, these periods of well-being eventually converge [31]. Even if there is no life-threatening diagnosis, the healthcare provider should ask the patient if his

or her symptoms of nausea, vomiting, and abdominal discomfort improve after having a hot bath or shower [32].

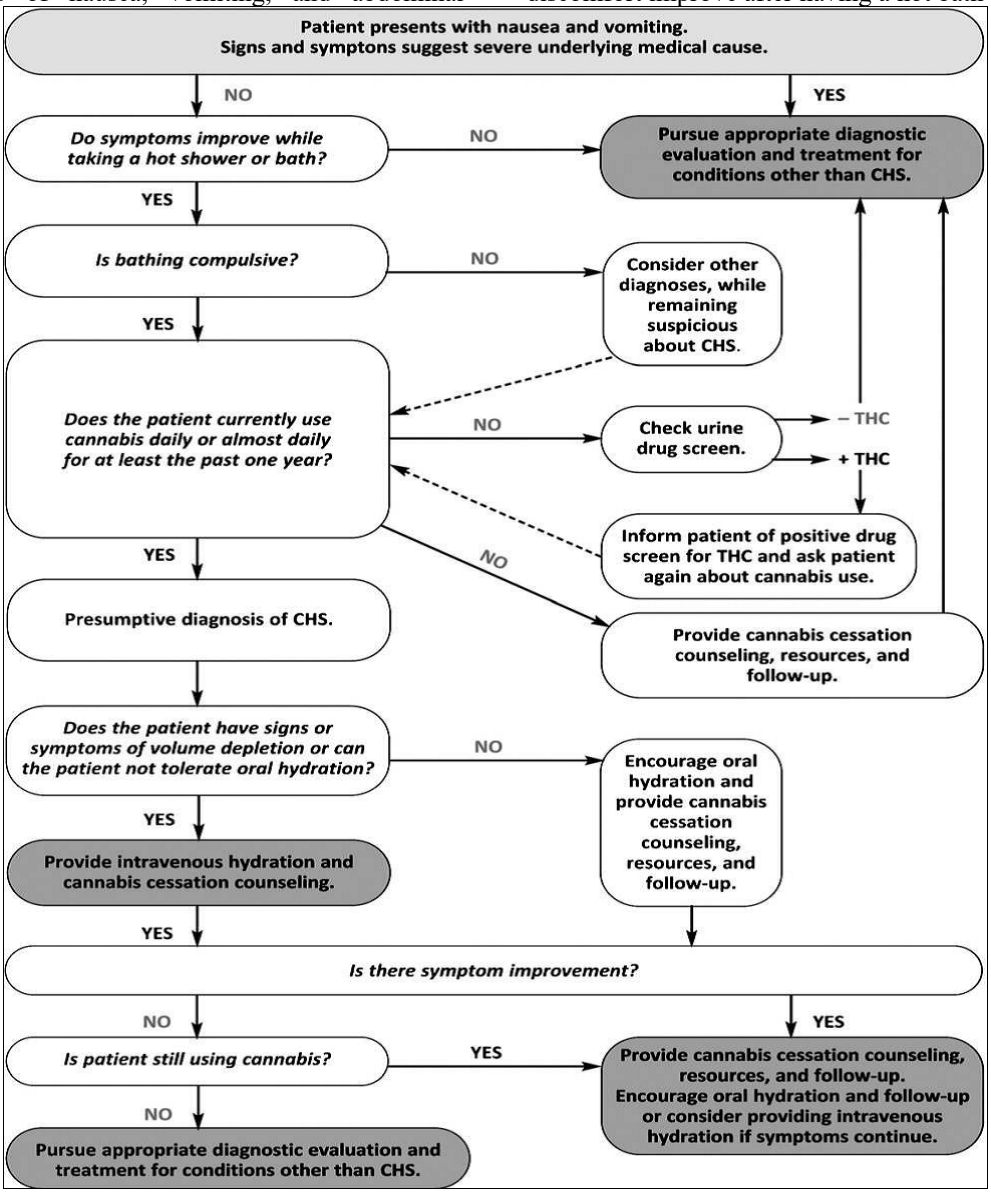

Fig 4: Identification of cannabis hyperemesis syndrome and its treatment protocol. Acute liver disease, adrenal insufficiency, bowel perforation, bowel obstruction, cholangitis, cholecystitis, diverticulitis, ectopic pregnancy, gastroparesis, myocardial infarction, nephrolithiasis, pancreatitis, pelvic inflammation, pregnancy, and ruptured or dissecting aortic aneurysm are among the diagnoses to take into consideration when a patient presents with nausea [32].

A patient may be diagnosed with CHS if they have used cannabis for a long time and have any of the following symptoms: [33].

1. Feeling queasy or throwing up
2. Having stomach discomfort
3. Having symptoms that go away when you take a hot bath or shower
4. Having symptoms that go away when you stop using cannabis
5. Using cannabis at least once a week.

The following features have also been proven to be useful:

1. Being under 50 years of age
2. At least 5 kg of weight reduction combined with symptoms
3. Symptoms in the morning
4. Regular procedures for the bowels
5. Negative test findings from radiography, endoscopy, and laboratories.

Table 1: Proposed new diagnostic criteria for cannabinoid hyperemesis syndrome [34].

Clinical features	Stereotypical episodic vomiting resembling CVS in terms of onset and frequency ≥3 episodes a year
Cannabis-use patterns.	Duration of use >1 y preceding onset of symptoms Frequency of use >4 times a week on average
Cannabis cessation	Resolution of symptoms should follow a period of cessation from cannabis for a minimum of 6 more at least equal to a duration that spans three typical cycles in an individual patient

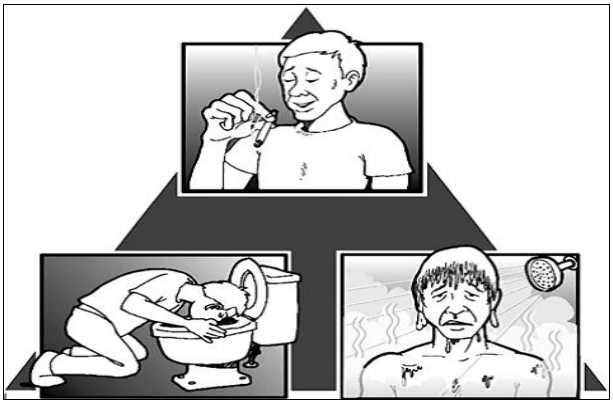

Fig 5: Three characteristics strongly point to the diagnosis of CHS: frequent (and frequently heavy) marijuana uses over an extended period of time; episodes of nausea and vomiting that are intractable and last for hours or days and do not improve with standard antiemetic therapy; and relief from symptoms from extremely hot baths or showers [22].

6. Management/Treatment

Problematic use of another substance (i.e., alcohol use) significantly increased the risk for cannabis use problems. Future work could usefully consider these clinically relevant findings when developing treatment for cannabis users; such considerations may help inform more personalized treatment and increase treatment efficacy [35]. Numerous therapeutic approaches have been investigated to address symptoms and manage the illness. Intravenous hydration, electrolyte replacement, and antiemetics are first used for symptom alleviation in the ED context. This helps CHS patients who repeatedly vomit to correct their volume and electrolyte deficiencies. Ondansetron, promethazine, prochlorperazine, and metoclopramide are examples of antiemetics that are regularly used; however, they are rarely successful in reducing CHS patients' nausea and vomiting [36]. Antiemetic, amnestic, and anxiolytic characteristics of lorazepam have made it useful in treating anticipated nausea and vomiting that occurs in chemotherapy patients. The therapy of cyclic vomiting syndrome has also been proposed to use lorazepam [37]. Other effective treatments mentioned only once included, fentanyl, promethazine, methadone, nabilone, levomepromazine, piritramide, and pantoprazole. hot showers and bathing were mentioned either by the authors or patients in all level-4 and -5 articles as universally effective at ameliorating CHS [38]. The care of CHS is not well understood, other from treating the consequences of frequent vomiting, which call for fluid resuscitation. The greatest alleviation from symptoms is obtained by taking a hot bath, how youever this is frequently impractical and has transient effects. In CHS, antiemetics have not demonstrated significant effectiveness, while not being discouraged [39]. This systematic review identified low-quality evidence from case reports, case series, and abstracts to suggest that capsaicin may be an effective treatment for CHS [40]. A documented limitation of capsaicin use is the patient's insight and compliance to capsaicin application. It is reportedthat some patients could not relate their relief of symptoms to the use of topical capsaicin [41].

Treatment of the acute phase of nausea and vomiting: [42].

- Intravenous application of lorazepam, alprazolam or (second line) haloperidol
- Intravenous application of sodium chloride 0.9% (1-2 bolus followed by 150-200 ml/h for 24-48 h)
- Intravenous application of proton pump inhibitors
- Cannabis cessation in patients with chronic marijuana abuse

Follow-up treatment of patients suffering from CVS: [42].

- Application of preventive medication, e.g. amitriptyline, propranolol, sumatriptane
- Application of medication capable to abort an episode during the prodromal phase, e.g. metoclopramide, ondansetron, lorazepam, oxycodone
- Psychosocial care
- Help identify and avoid trigger-factors

Follow-up treatment of patients suffering from CHS: [42].

- Primary treatment goal: cure due to cannabis cessation
- Provide access to psychiatric treatment for cannabis cessation
- Consider follow-up treatment as recommended for patients suffering from CVS in case of insufficient resolution of symptoms after cannabis cessation or if the patient refuses cannabis cessation.

One of the biggest challenges in managing CHS is its traditional resistance to standard antiemetic medications like metoclopramide and ondansetron, which are used in both emergency and general care settings. Over the past few years, a range of medical treatments have been recommended by case studies and reviews; these include short-term benzodiazepines, long-term tricyclic antidepressants, antiepileptics like levetiracetam, and antipsychotics like haloperidol. However, the recommendations are based on small case studies [43]. Admission is not necessary for the majority of adult cannabis toxicity patients, who recover on their own with observation and minimal intervention. To ensure safety in the home, pediatric patients, however, could need more time for monitoring and assistance, as well as the engagement of social services [44].

There are currently no consistently successful treatment plans, including traditional antiemetics, for CHS patients who refuse to give up cannabis use. Studies on pharmacology suggest that haloperidol may alleviate nausea, vomiting, and abdominal pain by acting indirectly on CB1 receptors. Case reports from the emergency department setting align with our case study findings [45]. Furthermore, splanchnic vasodilation, which results in the redistribution of blood from the gut to the skin and the development of "cutaneous steal syndrome," may be induced by hot bathing and potentially alleviate CHS symptoms [46].

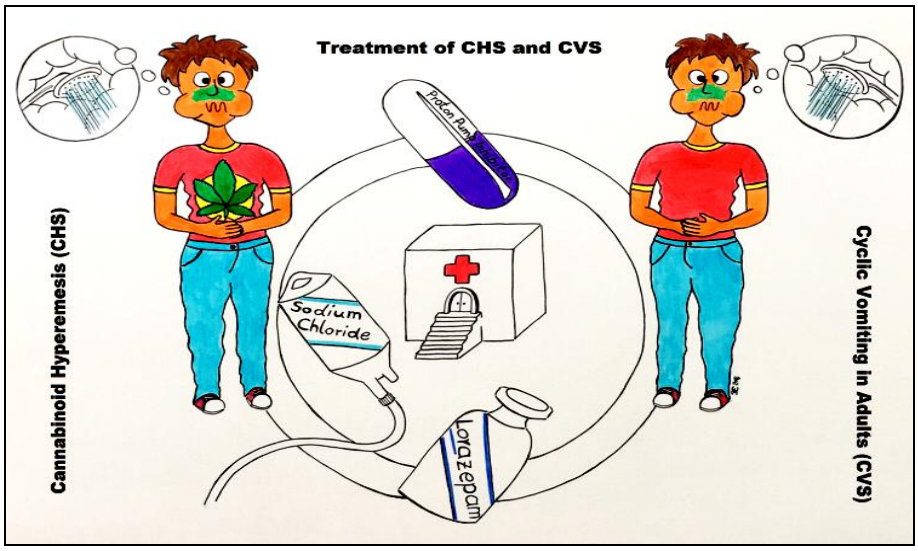

Fig 6: Patients often have severe nausea, vomiting, and stomach pain when they first arrive [42]

About half of the patients exhibit mandatory bathing behavior because taking a hot shower relieves their symptoms. It is necessary to overuse marijuana continuously to suspect CHS. When a vomiting episode is promptly and appropriately treated, recovery time is shortened and the inter-episodic period of relative wellbeing is extended. Intravenous lorazepam, proton pump inhibitors, and sodium chloride solution are used to treat the acute phase. comprehensive details on recommended treatments and diagnostic standards [42].

The compulsive "hot bath (shower) behavior" has also been implicated in cannabinoid hyperemesis syndrome and has been suggested to occur via "disequilibrium of the thermoregulatory system of the hypothalamus" which could settle with hot baths or showers have suggested that the desire for hot showers is either to counteract the cannabis-induced decrease in core body temperature or is a direct response to CB1 receptor activation in the hypothalamus. In fact, the medial pre optic/anterior hypothalamic area (POA) is the primary thermo sensitive site of the CNS [47]. These modalities can be effective treatment of the symptoms associated with cannabis hyperemesis syndrome; however, the best cure would be complete abstinence from cannabis use. Another theory of the relief brought on by hot showers is the concept of peripheral vasodilation and redistribution of blood flow from the splanchnic circulation to muscle that has been demonstrated during exercise and warm bathing [48]. The goal of CHS treatment has been to help patients stop using cannabis so that their symptoms will go away. Since patients in Allen *et al*.'s most extensive series were monitored for over two years, we can assume that stopping cannabis use did, in fact, cause the vomiting episodes to stop. Nevertheless, a lot of studies discuss brief cannabis-free periods that are as short as a few days spent in the hospital. These accounts are meaningless and call into question the reliability of the data used to establish a CHS diagnosis [49].

The focus of treatment is typically on helping the patient stop using marijuana. A common reason for using intravenous fluids is dehydration from vomiting. Antiemetics such 5-HT3 (ondansetron, for example), D2 (prochlorperazine, for example), H1 (promethazine), or neurokinin-1 receptor antagonists (aprepitant, for example) can be used, however these treatments are frequently unsuccessful. As the patient is able, the diet might be increased. It may be necessary to utilize proton pump inhibitors or H2 blockers since many patients have minor gastritis. Since marijuana is the only medication that has been proven to provide long-lasting relief from the hyper emetic phase, extensive counselling on quitting is required [50].

7. References

1. Nicolson SE, Denysenko L, Mulcare JL, Vito JP, Chabon B. Cannabinoid hyperemesis syndrome: a case series and review of previous reports. Psychosomatics. 2012;53(3):212-219.
2. Patterson DA, Smith E, Monahan M, Medvecz A, Hagerty B, Krijger L, *et al*. Cannabinoid hyperemesis and compulsive bathing: a case series and paradoxical pathophysiological explanation. J Am Board Fam Med. 2010;23(6):790-793.
3. Sorensen CJ, DeSanto K, Borgelt L, Phillips KT, Monte AA. Cannabinoid hyperemesis syndrome: diagnosis, pathophysiology, and treatment-a systematic review. J Med Toxicol. 2017;13:71-87.
4. Hopkins CY, Gilchrist BL. A case of cannabinoid hyperemesis syndrome caused by synthetic cannabinoids. J Emerg Med. 2013;45(4):544-546.
5. Perisetti A, Gajendran M, Dasari CS, Bansal P, Aziz M, Inamdar S, *et al*. Cannabis hyperemesis syndrome: an update on the pathophysiology and management. Ann Gastroenterol. 2020;33(6):571-578.
6. Ukaigwe A, Karmacharya P, Donato A. A gut gone to pot: a case of cannabinoid hyperemesis syndrome due to K2, a synthetic cannabinoid. Case Rep Emerg Med. 2014;2014(1):1-3.
7. Wallace EA, Andrews SE, Garmany CL, Jelley MJ. Cannabinoid hyperemesis syndrome: literature review and proposed diagnosis and treatment algorithm. South Med J. 2011;104(9):659-664.
8. Simonetto DA, Oxentenko AS, Herman ML, Szostek JH. Cannabinoid hyperemesis: a case series of 98 patients. Mayo Clin Proc. 2012;87(2):114-119.
9. Senderovich H, Patel P, Jimenez Lopez B, Waicus S. A systematic review on cannabis hyperemesis syndrome and its management options. Med Principles Pract. 2022;31(1):29-38.
10. Sun S, Zimmermann AE. Cannabinoid hyperemesis syndrome. Hosp Pharm. 2013 Aug;48(8):650-655.
11. Galli JA, Sawaya RA, Friedenberg FK. Cannabinoid hyperemesis syndrome. Curr Drug Abuse Rev. 2011

Dec;4(4):241-249.
12. Hernandez JM, Paty J, Price IM. Cannabinoid hyperemesis syndrome presentation to the emergency department: A two-year multicentre retrospective chart review in a major urban area. Can J Emerg Med. 2018;20(4):550-555.
13. Lapoint J, Meyer S, Charles KY, Koenig KL, Lev R, Thihalolipavan S, *et al*. Cannabinoid hyperemesis syndrome: public health implications and a novel model treatment guideline. West J Emerg Med. 2017;19(2):380-386.
14. DeVuono MV, Parker LA. Cannabinoid hyperemesis syndrome: a review of potential mechanisms. Cannabis Cannabinoid Res. 2020;5(2):132-144.
15. Gajendran M, Sifuentes J, Bashashati M, McCallum R. Cannabinoid hyperemesis syndrome: definition, pathophysiology, clinical spectrum, insights into acute and long-term management. J Invest Med. 2020;68(8):1309-1316.
16. Darmani NA. Cannabinoid-induced hyperemesis: a conundrum-from clinical recognition to basic science mechanisms. Pharmaceuticals. 2010;3(7):2163-2177.
17. Khattar N, Routsolias JC. Emergency department treatment of cannabinoid hyperemesis syndrome: a review. Am J Ther. 2018;25(3):357-361.
18. Richards JR, Gordon BK, Danielson AR, Moulin AK. Pharmacologic treatment of cannabinoid hyperemesis syndrome: a systematic review. Pharmacotherapy. 2017;37(6):725-734.
19. Sontineni SP, Chaudhary S, Sontineni V, Lanspa SJ. Cannabinoid hyperemesis syndrome: clinical diagnosis of an underrecognised manifestation of chronic cannabis abuse. World J Gastroenterol. 2009;15(10):1264-1266.
20. McConachie SM, Caputo RA, Wilhelm SM, Kale-Pradhan PB. Efficacy of capsaicin for the treatment of cannabinoid hyperemesis syndrome: a systematic review. Ann Pharmacother. 2019;53(11):1145-1152.
21. Cox B, Chhabra A, Adler M, Simmons J, Randlett D. Cannabinoid hyperemesis syndrome: case report of a paradoxical reaction with heavy marijuana use. Case Rep Med. 2012;2012:1-3.
22. Mahmad AI, Jehangir W, Littlefield II JM, John S, Yousif A. Cannabis hyperemesis syndrome: a case report review of treatment. Toxicol Rep. 2015;2(1):889-890.
23. Pergolizzi Jr JV, LeQuang JA, Bisney JF. Cannabinoid hyperemesis. Med Cannabis Cannabinoids. 2019;1(2):73-95.
24. Elnagar A, Kgomo M, Mokone M, Yousif B. Cannabinoid hyperemesis syndrome. BMJ Case Rep CP. 2024;17(4):1-3.
25. Jaishi PP, Neupane SK, Joshi K, Shrestha N, Khatri SS, Lamichhane S, *et al*. Case report: a case of undiagnosed cannabinoid hyperemesis syndrome in rural part of Nepal. Ann Med Surg. 2022;84:1-3.
26. Cha JM, Kozarek RA, Lin OS. Case of cannabinoid hyperemesis syndrome with long-term follow-up. World J Clin Cases. 2014;2(12):930-933.
27. Chang YH, Windish DM. Cannabinoid hyperemesis relieved by compulsive bathing. Mayo Clin Proc. 2009;84(1):76-78.
28. Iacopetti CL, Packer CD. Cannabinoid hyperemesis syndrome: a case report and review of pathophysiology. Clin Med Res. 2014;12(1):65-67.
29. Bajgoric S, Samra K, Chandrapalan S, Gautam N. Cannabinoid hyperemesis syndrome: a guide for the practising clinician. Case Reports. 2015;23(1):1-3.
30. Jones JL, Abernathy KE. Successful treatment of suspected cannabinoid hyperemesis syndrome using haloperidol in the outpatient setting. Case Rep Psychiatry. 2016;2016:1-3.
31. Collins AB, *et al*. "I still partly think this is bullshit": A qualitative analysis of cannabinoid hyperemesis syndrome perceptions among people with chronic cannabis use and cyclic vomiting. Drug Alcohol Depend. 2023;246:1-6.
32. Figueroa-Rivera IM, Estremera-Marcial R, Sierra-Mercado M, Gutiérrez-Núñez J, Toro DH. Cannabinoid hyperemesis syndrome: a paradoxical cannabis effect. Case Rep Gastrointest Med. 2015;2015:1-3.
33. Blumentrath CG, Dohrmann B, Ewald N. Cannabinoid hyperemesis and the cyclic vomiting syndrome in adults: recognition, diagnosis, acute and long-term treatment. GMS German Med Sci. 2017;15:1-8.
34. Venkatesan T, Levinthal DJ, Li BU, Tarbell SE, Adams KA, Issenman RM, *et al*. Role of chronic cannabis use: cyclic vomiting syndrome vs cannabinoid hyperemesis syndrome. Neurogastroenterol Motil. 2019;31(2):1-14.
35. Price SL, Fisher C, Kumar R, Hilgerson A. Cannabinoid hyperemesis syndrome as the underlying cause of intractable nausea and vomiting. J Osteopath Med. 2011;111(3):166-169.
36. Fig:1 - https://images.app.goo.gl/MyVLiV49vbLE8CFN6.
37. Manning K, Rogers AH, Bakhshaie J, Hogan JBD, Buckner JD, Ditre JW, *et al*. The association between perceived distress tolerance and cannabis use problems, cannabis withdrawal symptoms, and self-efficacy for quitting cannabis: The explanatory role of pain-related affective distress. Addict Behav. 2018;85(1):1-7.
38. Madireddy S, Patel RS, Ravat V, Ajibawo T, Lal A, Patel J, *et al*. Burden of comorbidities in hospitalizations for cannabis use-associated intractable vomiting during post-legalization period. Cureus. 2019 Aug 27;11(8):1-10.
39. Fig:2 - https://cdn.britannica.com/39/123539-050-837C3C7C/Hemp.jpg.
40. Fig;3- https://images.app.goo.gl/CUVQkH1z7Am5L6As6.
41. Fleming JE, Lockwood S. Cannabinoid hyperemesis syndrome. Fed Pract. 2017;34(10):33-36.
42. Soh J, Kim Y, Shen J, Kang M, Chaudhry S, Chung TH, *et al*. Trends of emergency department visits for cannabinoid hyperemesis syndrome in Nevada: An interrupted time series analysis. PLoS One. 2024;19(5):1-8.
43. Arendash JM, Chiu C, Wang J, Mihm F. Cannabinoid hyperemesis and pheochromocytoma hypertensive urgency: a case report. J Med Case Reports. 2024;18(1):1-5.
44. Ng T, Keshock MC. Tetrahydrocannabinol (THC). In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. 2023 Nov 12. PMID: 33085321.
45. Aziz A, Waheed T, Oladunjoye O, Oladunjoye A, Hanif M, Latif F. Topical capsaicin for treating cannabinoid hyperemesis syndrome. Case Rep Gastrointest Med. 2020;2020:1-4.
46. Randall K, Hayward K. Emergent medical illnesses related to cannabis use. Missouri Med. 2019;116(3):226-228.
47. Kelly BN, Nappe TM. Cannabinoid Toxicity. In: StatPearls. Treasure Island (FL): StatPearls Publishing;

2024 Jan. 2023 Jul 10.
48. Cue L, Chu F, Cascella M. Cannabinoid Hyperemesis Syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan. 2023 Jul 3. PMID: 31751105.
49. Turner AR, Patel P, Agrawal S. Marijuana. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan. 2024 Feb 2. PMID: 28613552.
50. Taylor BN, Mueller M, Sauls RS. Cannaboinoid Antiemetic Therapy. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan. 2023 Aug 14. PMID: 30571051.