

‘Scromiting’ and compulsive hot showering in cannabis users

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Abstract

‘Scromiting’ is the colloquial term for the simultaneous screaming and vomiting that characterizes the extraordinary intensity of pain experienced during acute hyperemetic episodes that occur in some long-term users of high doses of cannabinoids. It is recognized as a distinct clinical entity with a characteristic triad of features: prolonged history of daily or near-daily cannabis use, recurrent episodes of severe nausea and vomiting, and compulsive use of hot baths or showers, which patients have found to give temporary symptomatic relief. The intent of this review is to present a succinct overview of what (little) is known about postulated mechanisms, overlap with relationship to pain, and aspects of the clinical presentation.

Key words: scromiting; hot shower hydrotherapy; cannabinoid hyperemesis syndrome; endocannabinoid system; TRPV1; Δ^9 -THC.

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Introduction

Cannabinoid hyperemesis syndrome (CHS)^{1,2} is a paradoxical and increasingly prevalent clinical entity, partly attributable to proliferation of high-potency cannabis products,³ that is characterized by recurrent cycles of severe nausea, intractable vomiting, and cramping abdominal pain in the setting of chronic heavy cannabis use.^{1,2,4} CHS was first formally described in 2004,⁵ and can only be reliably resolved by cessation of cannabis use. It can be underdiagnosed because of clinician unfamiliarity, its paradoxical relationship with a substance long known as anti-emetic (cannabinoids),⁶ and the absence of definitive biomarkers.⁷ Despite growing prevalence, CHS can easily be misdiagnosed on initial presentation, leading to repeated emergency department (ED) visits, unnecessary diagnostic investigations, and inappropriate therapeutic interventions.⁷ The delay from symptom onset to diagnosis (up to years) incurs unnecessary medical testing or surgical treatment, and healthcare expenditure.⁸ As cannabis legalization broadens access and the rising Δ^9 -tetrahydrocannabinol (Δ^9 -THC) concentrations in commercial products increases the cumulative cannabinoid burden experienced by regular users, the burden of CHS on healthcare systems is expected to intensify. Although the pathophysiology of CHS is incompletely understood, it is possibly a consequence of chronic overstimulation and dysregulation (physiologic rebound?) of the endocannabinoid system, particularly cannabinoid receptor type 1 (CB1R), with additional involvement of the transient receptor potential vanilloid type 1 (TRPV1) receptor in both the hyperemetic mechanism and the hot water respite.⁹ A synopsis of the sparse literature on the clinical and basic science aspects of CHS is presented, along with reference to the overlap with pain mechanisms.

Methods

Sources were identified through targeted searches of PubMed and Google Scholar (through Aug 2026) using combinations of the search terms “cannabinoid hyperemesis syndrome”, “cannabis hyperemesis”, “scromiting”, “TRPV1”, “CB1”, “CB2”, “GPR55”, “endocannabinoid”, and “hot water hydrotherapy”, supplemented by searching the reference lists of identified reviews, case series, and the 2024 American Gastroenterological Association Clinical Practice Update.¹⁰ Foundational descriptions, large case series, systematic reviews, and the recent (2024-2026) literature were prioritized over isolated case reports where both were available, given the rapidly evolving epidemiology of CHS. Because ‘scromiting’ itself has not been the subject of dedicated mechanistic study, the discussion of its pathological basis is necessarily inferential, extrapolated from the broader CHS and nociception literature rather than from direct experimental evidence.

Clinical presentation

CHS is usually described as having three phases.¹ The ‘prodromal’ phase, which might last months or even years, is characterized by morning nausea, early satiety, fear of vomiting, and mild abdominal discomfort. During this phase, people often increase cannabis use in an attempt to self-medicate the nausea. But this is a counterproductive behavior that instead accelerates endocannabinoid system dysregulation.¹ The ‘hyperemetic’ phase is the acute presentation most commonly encountered in clinical settings and is defined by recurrent episodes of overwhelming nausea, profuse vomiting, and severe cramping abdominal pain.^{2,3}

Retching may occur up to five times per hour, and episodes typically last 24 to 48 h, but may persist even longer.² The ‘recovery’ phase follows cessation of cannabis use, and is characterized by slow progressive resolution of symptoms over days to weeks, with eventual complete remission only following sustained abstinence.^{4,5} Scromiting occurs during the acute hyperemetic phase of CHS,¹¹ when the occurrence and severity of pain, nausea, and retching is maximal. The timing of the three CHS phases, and the transitions between them, is clinically important because it shapes how patients present and how readily the diagnosis is missed. The prodromal phase is typically the longest, often spanning months to several years of intermittent morning nausea and early satiety before a first hyperemetic episode occurs, during which patients are rarely evaluated for a cannabis-related cause. Once established, hyperemetic episodes tend to recur every few weeks to a few months, with each discrete episode lasting 24 to 48 h, though individual episodes have been reported to persist for up to a week in severe cases;^{3,5} between episodes, most patients are entirely asymptomatic, a cyclical pattern that closely mimics cyclic vomiting syndrome and contributes to diagnostic delay. The recovery phase, once cannabis use is stopped, generally begins within days, with progressive resolution of nausea and nonspecific abdominal discomfort over one to two weeks; however, complete and durable remission, confirmed by absence of recurrence on re-exposure testing in some case series,^{3,5,9} may take up to six months,¹⁰ and premature reintroduction of cannabis before this window elapses is a common cause of perceived treatment failure.⁵

The pathological basis of scromiting is not yet formally studied, but it is believed to reflect the convergence of severe visceral pain arising from gastric dysmotility, intestinal cramping, and esophageal irritation.^{12,13} The intensity of scromiting is sufficient to cause soft tissue injuries and, in rare cases, esophageal mucosal (Mallory-Weiss) tears, which can progress to esophageal perforation (Boerhaave syndrome) and aspiration.¹⁴ For ED emergency clinicians, the phenomenon of scromiting serves as a diagnostic signal: while nausea and vomiting are among the most common ED presentations and have a broad differential diagnosis, the combination of screaming-level pain with vomiting in a young patient with a history of daily cannabis use should prompt consideration of CHS, particularly when standard antiemetics fail to provide relief.¹⁵

Compulsive hot showering

The compulsive use of hot baths or showers is among the most unusual characteristic features of CHS, occurring in the vast majority of cases. One early literature review found the behavior was present in 29 of 31 reported cases, and it is now incorporated into diagnostic criteria.¹⁶ Patients typically report that prolonged exposure to hot water is the only intervention that reliably attenuates their nausea and vomiting during acute episodes. Relief is characteristically rapid, temperature-dependent, and transient, symptoms returning within minutes of exiting the water.¹⁷ The short duration of relief drives patients to spend *hours per day* in the shower or bath during hyperemetic episodes, with water temperatures approaching or exceeding the thermal pain threshold. Thus, the behavior can produce significant burning pain in its own right. Case reports have documented severe scalding burns,¹⁸ hyponatremia from prolonged hot water exposure and concurrent vomiting-associated fluid loss, plus cardiovascular complications in patients with underlying comorbidities.

Epidemiology

The true prevalence of CHS is difficult to ascertain given diagnostic delays and underreporting, but available data suggest it is far from rare. A recent population-based survey of cannabis users in the United States estimated a prevalence of about 6% past-year CHS;¹⁹ a study in an urban New York City ED consisting of 2,127 patients reported that 32.9% of patients who smoked marijuana 20 days or more per month met the criteria for CHS.²⁰ Emergency department data corroborate a significant and growing burden: CHS accounts for a substantial proportion of hyperemesis-related presentations in states where cannabis is legal, with visit rates increasing in parallel with cannabis legalization. CHS is predominantly reported in young adults, with a median age of onset in the late 20s to early 30s, and it appears to be more common in males, though this may reflect higher rates of daily heavy cannabis use in that demographic. A critical risk factor is the duration and intensity of cannabis use: most CHS patients report years of daily use, often a decade or more, before symptom onset, and many use high-potency cannabis products including concentrates and oils with THC concentrations well above those present in traditional cannabis preparations.²¹ However, the escalating potency of commercially available cannabis products in legalized markets raises concern that the latency to CHS onset may shorten in current cohorts of heavy users.

Whether CHS risk differs between medically authorized and recreational cannabis use is directly relevant to pain physicians, oncologists, and palliative care specialists, given the expanding use of cannabinoids for chronic pain, chemotherapy-induced nausea, and symptom control in serious illness. CHS has, in fact, been reported in patients using cannabinoids under medical supervision: a palliative care patient with small-cell lung cancer developed CHS after five years of prescribed nabilone, a synthetic Δ^9 -THC analog, used to manage neuralgia, illustrating that a legitimate, physician-directed indication and formulation do not confer immunity from the syndrome.²² This evidence, however, remains at the level of isolated case reports rather than a dedicated epidemiological comparison; no study to date has directly compared CHS incidence between medically authorized and recreational cannabis cohorts matched for dose and duration of exposure. Clinical guidance for cannabis-based treatment of neuropathic pain generally recommends low-THC formulations (under 5-10%) specifically to limit psychoactive and other dose-related effects, which would theoretically place appropriately dosed medical use below the potency threshold empirically associated with CHS in heavy recreational users. In practice, this assumption may not hold: a survey of online dispensary listings found that advertised THC concentrations in medical cannabis programs (mean 19.2%) were statistically indistinguishable from those in recreational programs, with 70% to 91% of products in both categories exceeding 15% THC, concentrations well above those generally recommended for neuropathic pain.²³ The duration-of-use criterion central to CHS, typically years of daily or near-daily use, is also commonly met by legitimate long-term medical cannabinoid therapy for chronic pain or palliative symptom control. Taken together, current evidence suggests that CHS risk tracks more closely with cumulative THC exposure, product potency, and duration of use than with whether or not that use is medically authorized, and clinicians managing pain, oncologic, or palliative-care patients on long-term cannabinoid therapy should maintain the same index of suspicion for CHS as they would for recreational users presenting with treatment-refractory nausea, vomiting, or abdominal pain.

CHS is not confined to adults. A single-institution 10-year case

series identified 34 adolescent patients, aged 13 to 20 years, that met CHS criteria, with clinical features broadly similar to adult presentations, including cyclic vomiting after at least three months of regular cannabis use, abdominal pain, and hot-bathing behavior, although follow-up and documentation of drug history were frequently incomplete.²⁴ Pediatric CHS carries distinct diagnostic and complication considerations: adolescents may conceal cannabis use from parents or guardians, so a detailed drug history taken in private, ideally corroborated by urine drug screening, is recommended; pediatric case reports have also documented severe barotraumatic complications, including pneumomediastinum, arising from forceful repetitive retching, underscoring that scromiting-related tissue injury is not limited to adult patients.¹⁴ Diagnostic criteria validated in adults have not been formally confirmed in children and adolescents, and the true pediatric incidence remains poorly defined.

Surveillance of CHS has itself been limited by the absence of a dedicated diagnostic code; prior to October 2025, CHS-involved encounters could only be estimated indirectly by combining codes for cyclical vomiting or nonspecific emesis with a separate cannabis-related diagnosis code. A dedicated ICD-10-CM code for cannabis hyperemesis syndrome, R11.16, became effective October 1, 2025. In the first eight months following implementation, national emergency department surveillance data showed CHS-involved visit proportions roughly 3.7-fold higher than the average observed under the prior indirect coding approach, with the highest proportions among patients aged 15 to 24 years and among females, an increase the surveillance authors attribute largely to improved case ascertainment and coding rather than a true abrupt rise in incidence.²⁵ The new code should substantially improve the accuracy of future CHS surveillance and is a useful documentation tool for clinicians managing this population.

Thermoregulatory dysregulation in CHS

The current neurobiological explanation for hot water hydrotherapy in CHS begins with the observation that chronic heavy THC exposure dysregulates thermoregulatory centers in the hypothalamus through overstimulation of CB1 receptors. The endocannabinoid system plays a physiological role in body temperature regulation, with CB1R activation normally associated with hypothermia.²⁶ Chronic desensitization and downregulation of CB1R following prolonged heavy THC exposure is hypothesized to shift the hypothalamic temperature setpoint, producing a state of relative central hypothermia that is subjectively experienced as feeling cold and that sensation drives patients to seek external heat. Hot showers temporarily correct the perceived thermoregulatory deficit, with the resolution of the abnormal thermal state coinciding with symptomatic relief of nausea and vomiting.

A second mechanistic pathway through which hot water is thought to relieve CHS symptoms involves the transient receptor potential vanilloid type 1 (TRPV1) receptor, a polymodal cation channel that serves as a molecular integrator of thermal, chemical, and mechanical nociceptive stimuli.²⁷ TRPV1 was first cloned and characterized as the molecular receptor for capsaicin, the pungent principle of chili peppers, and was shown to be gated by noxious heat in approximately the 43°C range, establishing it as the principal molecular transducer of painful thermal stimuli and providing the mechanistic foundation for its proposed role in both hyperemetic visceral pain and hot-water-induced counter-stimulation in CHS.^{12,27} TRPV1 is activated by temperatures exceeding 43°C, low pH, and

endovanilloids, and is expressed on sensory nerve endings in the skin as well as on neurons of the gastrointestinal tract, including vagal afferents innervating the stomach. Importantly, TRPV1 is anatomically and functionally linked to the endocannabinoid system: anandamide, an endogenous cannabinoid, is also a TRPV1 agonist, and chronic THC exposure alters the expression and sensitivity of TRPV1 in conjunction with CB1R. In the context of CHS, it is proposed that hot shower-induced thermal activation of cutaneous TRPV1 receptors generates afferent neural signals that compete with and suppress the gastrointestinal nociceptive and emetic signals driving nausea and vomiting.¹² This counter-stimulation mechanism may exploit gate-control principles of pain modulation, with the intense thermal input from hot water overriding or attenuating the visceral hyperemetic drive. Additionally, repeated TRPV1 activation in response to high thermal stimuli leads to receptor desensitization and the depletion of neuropeptide stores (particularly substance P and calcitonin gene-related peptide) from sensory nerve endings, a phenomenon that may partly explain the relief, albeit temporary, experienced during hot showering.¹² The TRPV1 mechanism is further supported by the therapeutic efficacy of topical capsaicin, a pharmacological TRPV1 agonist and desensitizer, in acute management of CHS. The connection between hot shower response and capsaicin pharmacology strengthens the hypothesis that TRPV1-mediated counter-stimulation and desensitization are involved in symptomatic relief of CHS.

A third proposed mechanism is that hot water exposure produces peripheral vasodilation, redistributing blood flow from the splanchnic circulation to the skin and musculature. This vascular redistribution reduces visceral perfusion and may attenuate the gastrointestinal afferent signaling driving emesis. In a patient with CHS-related gastrointestinal dysmotility and visceral hypersensitivity, even a transient reduction in splanchnic blood flow and mucosal perfusion could meaningfully reduce the peripheral emetic drive. This proposed mechanism is speculative, but plausible given the known vascular effects of heat-induced cutaneous vasodilation.^{10,12}

Pathophysiology of CHS

CHS presents an apparent paradox: cannabis and its principal psychoactive constituent, Δ^9 -THC, possess well-known antiemetic properties mediated through CB1R agonism in the brainstem dorsal vagal complex and nucleus tractus solitarius. However, chronic high-dose Δ^9 -THC exposure produces a dose-dependent transition in the endocannabinoid system from *anti*-emetic to *pro*-emetic; the most widely invoked mechanism involving progressive downregulation and desensitization of CB1R throughout the enteric nervous system and brainstem emetic circuits following sustained receptor overstimulation.¹² In the gastrointestinal tract, CB1R activation normally inhibits the release of emetic neurotransmitters and suppresses intestinal motility. Following chronic Δ^9 -THC exposure, downregulated CB1R in the intestinal nerve plexus loses this inhibitory tone, resulting in disinhibition of gastrointestinal motility, heightened visceral sensitivity, and pathological gastric dysmotility.¹² Simultaneously, toxic accumulation of cannabinoids in the gastrointestinal mucosa, facilitated by cannabis' high lipid solubility and the large body-fat stores that accumulate with chronic use, is proposed to contribute to mucosal irritation and prolonged dysregulation even after acute use ceases.¹²

Not all heavy chronic cannabis users develop CHS, so genetic susceptibility likely plays a role. Polymorphisms in several candi-

date genes have been identified in small CHS cohorts, including genes encoding TRPV1, catechol-*O*-methyltransferase (COMT), the CYP2C9 cytochrome P450 enzyme responsible for Δ^9 -THC metabolism, the dopamine D2 receptor (DRD2), and the ATP-binding cassette transporter ABCA1.^{12,28} A genomic survey of self-reported CHS-affected patients compared to asymptomatic cannabis users identified variation in cannabinoid- and xenobiotic-metabolizing genes.²⁸ These associations, if replicated in larger studies, would help explain the inter-individual variability in CHS susceptibility and could eventually inform genetic risk stratification in clinical practice.

Although the discussion above has centered on CB1R, the cannabinoid receptor family includes a second, immunologically oriented receptor, CB2, together with the less-characterized 'orphan' (i.e., unknown role) receptor GPR55, both of which may contribute a neuroimmune dimension to CHS pathophysiology that has received comparatively less attention.^{1,9} Unlike CB1R, which is concentrated in myenteric and submucosal enteric neurons, CB2R are located predominantly in inflammatory and immune cells, including intestinal macrophages, with a smaller contribution from sensory neurons, and they are implicated in modulating inflammation and nociception rather than motility.^{1,9} Therefore, sustained high-dose Δ^9 -THC exposure could plausibly alter CB2-mediated immune signaling within the gut wall in parallel with CB1R down-regulation,^{1,9,13} potentially contributing to the low-grade mucosal inflammation and heightened visceral sensitivity observed in CHS, though this remains a mechanistic hypothesis rather than a directly demonstrated pathway in humans.^{4,10} GPR55 is raised here as a potential candidate for future mechanistic study rather than an established contributor. Clarifying whether CB2- and GPR55-mediated neuroimmune signaling meaningfully contributes to CHS, together with the better-characterized CB1R and TRPV1 pathways, represents an important gap for future basic-science research.^{1,4,10}

Diagnosis and management

CHS remains largely a clinical diagnosis of exclusion, and the time from symptom onset to correct diagnosis can exceed years. The diagnosis rests on three pillars: a history of chronic heavy cannabis use (typically daily or near-daily for at least one year), recurrent cyclical episodes of nausea and vomiting, and compulsive hot bathing or showering behavior with documented symptomatic relief. Key differential diagnoses that need to be excluded include cyclic vomiting syndrome (CVS), gastroparesis, peptic ulcer disease, biliary pathology, and, in women of reproductive age, hyperemesis gravidarum.⁷ CVS in particular shares clinical features with CHS, including recurrent stereotypic episodes and relief with hot bathing in some patients, and the two conditions may coexist or be causally related in cannabis-using patients.¹³ An important distinguishing feature of CHS is its direct relationship to cannabis use: cessation of which invariably leads to symptom resolution (typically within days to weeks), and rechallenge reproduces the syndrome. No specific laboratory or imaging findings are pathognomonic, though dehydration, electrolyte disturbances, and elevated amylase or lipase may be present secondary to repeated vomiting and reduction of food or water intake.¹³

The only definitive curative intervention for CHS is complete and sustained abstinence from cannabis use.²⁹ This information should be communicated to patients, as many are unaware of the causal relationship between their cannabis use and their symptoms.

Some patients may have been reassured by prior clinicians that cannabis is harmless or even beneficial, which it might be at lower amounts and in less chronic use, creating a therapeutic barrier. Recurrence of CHS is universal upon cannabis resumption, typically within days to weeks, and patients who return to use after a period of abstinence-related symptom resolution constitute a significant proportion of CHS-related ED recidivism. Acute management of CHS in the ED focuses on symptom relief, rehydration, and electrolyte correction.¹² Conventional antiemetics such as ondansetron, metoclopramide, promethazine, and antihistamines, are largely ineffective in CHS, a clinical observation that, when present, supports its diagnosis. The pharmacological agents with the strongest evidence for acute symptom relief are haloperidol and droperidol, plus topical capsaicin.³⁰ Haloperidol and droperidol are dopamine D2 receptor antagonists with action at the chemoreceptor trigger zone, a mechanism well-suited to the dopamine dysregulation implicated in CHS.³¹ Haloperidol was shown in the only randomized controlled trial conducted to date in CHS (the HaVOC trial) to produce better symptom control than ondansetron, with an associated shorter length of hospital stay.³² Droperidol demonstrates comparable efficacy.³³ An important safety consideration is the risk of QTc prolongation associated with both agents, of particular relevance given the hypokalemia commonly present from prolonged vomiting in patients who are already at risk due to concurrent cannabis-related effects.

Because pharmacologic therapy only addresses acute symptoms, durable resolution of CHS depends on sustained cannabis cessation, and structured counseling support materially improves the likelihood of achieving it. Validated screening instruments such as the Cannabis Use Disorder Identification Test-Revised can identify patients with a comorbid cannabis use disorder who warrant referral to addiction medicine or behavioral health specialists rather than discharge advice alone.³⁴ Motivational enhancement therapy, cognitive behavioral therapy, and contingency management have each shown benefit in reducing cannabis use and relapse in observational and controlled studies, and peer-support models analogous to twelve-step facilitation have also been used as adjuncts. Counseling should set realistic expectations regarding relapse: outpatient studies of cannabis cessation more broadly suggest that roughly half of patients achieve two weeks of abstinence, and most who relapse do so within six months, underscoring the value of scheduled follow-up rather than a single discharge conversation. Patients should also be counseled explicitly that even small amounts of cannabis can precipitate recurrence, and that heavy, long-term users attempting abrupt discontinuation may experience concurrent cannabis withdrawal symptoms; a gradual, supervised reduction, ideally coordinated with an addiction medicine or primary care clinician, may be better tolerated than abrupt cessation in this subgroup and can reduce the likelihood of a return to prior use patterns.³⁴

Topical capsaicin cream (0.025–0.1%), applied to the abdomen during acute CHS episodes, has shown efficacy in reducing nausea and vomiting in multiple case series and a systematic review, with a rapid onset of action and a favorable safety profile.^{30,35} Its mechanism of action, TRPV1 agonism and desensitization of cutaneous and visceral afferents, mirrors one of the proposed mechanisms of hot shower relief, providing a pharmacological analog to the serendipitous patient-discovered behavioral remedy. Topical capsaicin is now incorporated into expert consensus guidelines as a reasonable first-line treatment in the ED, with benzodiazepines, which through their sedating, anxiolytic, and muscle-relaxant properties, provide symptomatic relief, are among the most consistently report-

ed effective agents in pediatric and adult CHS case series.³⁶ In contrast, opioids should be avoided due to their emetogenic potential and the higher risk of opioid use disorder in this population

Conclusions

Cannabinoid hyperemesis syndrome, with its characteristic manifestations of ‘scomiting’ and compulsive hot showering, represents a clinically important and growing result of chronic heavy use of cannabis. Its paradoxical occurrence due to a substance widely perceived as antiemetic has contributed to delays in diagnosis and suboptimal management in some ED and outpatient settings. The neurobiological underpinnings of both the hyperemetic episodes, postulated to be driven by chronic CB1R downregulation and enteric nervous system dysregulation, and the compulsive hot shower behavior, presumably mediated through TRPV1 thermosensitive counter-stimulation and hypothalamic thermoregulatory dysregulation, are likely involved, though significant gaps remain.

The convergence of rising cannabis potency, expanding legalization, and increasing rates of daily high-potency cannabis use among young adults portends a significant increase in CHS incidence. Clinician education regarding the diagnostic criteria, paradoxical pharmacology, and appropriate treatment of CHS, including the importance of cannabis cessation as the only intervention that is curative. Future research priorities include prospective epidemiological studies to define the true incidence and risk factors for CHS, clinical trials of promising pharmacological agents, and mechanistic studies to elucidate the mechanistic interactions that drive both the hyperemetic syndrome and its unusual hot-water showering remedy.

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