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Successful Management of Refractory Cannabinoid Hyperemesis Syndrome With Topical Capsaicin: A Case Report With Proposed Mechanism of Action and Literature Review

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Abstract

Introduction: Cannabinoid hyperemesis syndrome (CHS) is a debilitating disorder of chronic, heavy cannabis users characterized by cyclic severe nausea, vomiting, and abdominal pain that is classically relieved by hot baths. Standard antiemetics often fail, and dysregulation of endocannabinoid signaling with involvement of heat-sensitive TRPV1 channels has been proposed. Topical capsaicin - a TRPV1 agonist - has been reported in small series and case reports to reproduce the hot-water effect and rapidly relieve symptoms. **Case presentation:** A 28-year-old man with daily high-potency cannabis use (~2 g/day for 7 years) presented to the emergency department with 48 hours of intractable non-bilious vomiting (~20 episodes/day), severe periumbilical cramping (8/10), and a history of similar episodic flares relieved by prolonged hot showers. Initial ED therapy (IV fluids, ondansetron, metoclopramide, pantoprazole) produced minimal benefit; labs showed hypokalemic, hypochloremic metabolic alkalosis and pre-renal azotemia, with otherwise unremarkable imaging and enzymes. After informed consent, ~2 g of 0.1% topical capsaicin cream was applied across the abdomen. The patient experienced an acute burning sensation for ~15–30 minutes; retching ceased within 10 minutes, pain fell to 2/10 by 30 minutes and resolved by 90 minutes, and he tolerated oral intake. Vital signs normalized and electrolytes/renal function returned to baseline at 48-hour follow-up. He was discharged with counseling on cannabis cessation and a capsaicin tube for prodromal use. **Conclusion:** Topical capsaicin produced rapid, durable symptom resolution in this case of refractory CHS with only transient local discomfort. The effect is plausibly mediated by TRPV1 activation followed by peripheral desensitization, recapitulating the therapeutic hot-water response. Given consistent positive signals from case reports, series, and small pilot data, topical capsaicin is a low-risk, accessible adjunct for refractory CHS, but larger controlled studies are needed to define optimal dosing, duration, and long-term outcomes.

Keywords

cannabinoid hyperemesis syndrome, topical capsaicin, TRPV1

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Introduction

Cannabinoid hyperemesis syndrome (CHS) is a clinical disorder seen in long-term cannabis users, characterized by cyclical episodes of severe nausea, vomiting, and abdominal pain. Patients typically have a history of daily or near-daily cannabis use for years, and a main hallmark is that symptoms markedly improve during prolonged hot baths or showers.[1,2] First described in 2004, CHS is being increasingly recognized as marijuana use rises, yet its diagnosis can be challenging since routine evaluation (labs, imaging) is often unrevealing.[1-3]

Standard antiemetic therapies frequently provide little relief in CHS, prompting use of adjunctive treatments.[3] The underlying pathophysiology is not fully understood, but dysfunction of endocannabinoid pathways and thermosensitive TRPV1 (transient receptor potential vanilloid-1) channels has been implicated.[2] TRPV1 is a heat-activated nociceptor that, when stimulated (for example, by hot water or capsaicin), initially causes burning pain but subsequently leads to neuronal desensitization and depletion of excitatory neurotransmitters. Topical capsaicin, a chili-pepper extract and potent TRPV1 agonist, has been explored as a novel therapy: by activating the same receptors as heat, it can mimic hot-water bathing and, in case series, has yielded rapid symptom relief and tolerable side effects.[2,4]

Despite growing interest, reports of capsaicin use in CHS are still scarce. To our knowledge, topical capsaicin has been used successfully in only about 18 cases of CHS to date,[4] and contemporary analyses emphasize that the evidence remains limited to small series and case reports.[3] Refractory CHS that fails standard therapy is relatively uncommon, making novel treatment successes noteworthy. We present a case of otherwise-healthy young man with refractory CHS who experienced dramatic and sustained improvement after topical capsaicin application, underscoring both the rarity of this scenario and the therapeutic potential of TRPV1-targeted treatment.

Case Presentation

A 28-year-old male presented to the Emergency Department (ED) with a 48-hour history of intractable vomiting and epigastric pain. He reported approximately 20 episodes of non-bloody, non-bilious vomiting per day and described a constant, cramping periumbilical pain, rating it 8/10 in severity. He admitted to daily cannabis use (high-potency cannabis flower, ~2 grams/day) for over 7 years. He reported identical episodes every 2-3 months over the past two years, each relieved temporarily by prolonged hot showers. He had presented to another ED 24 hours prior, where he received intravenous (IV) fluids, ondansetron, metoclopramide, and pantoprazole with minimal improvement.

The patient was anxious, diaphoretic, and actively retching. Vital signs: temperature 37.1°C, heart rate 112 bpm, blood pressure 148/92 mmHg, respiratory rate 20/min, SpO2 99% on room air. Abdominal examination revealed hyperactive bowel sounds and mild, diffuse tenderness to palpation without guarding, rebound, or organomegaly. The remainder of the examination was unremarkable. Notable was erythematous skin on his trunk and limbs, consistent with recent hot bathing.

Due to refractory symptoms, comprehensive laboratory testing and imaging were performed to rule out alternative pathologies (Table 1). Results were significant for a mild hypokalemic, hypochloremic metabolic alkalosis consistent with vomiting, and elevated creatinine suggestive of pre-renal azotemia. Lipase, liver enzymes, and abdominal CT were normal.

After obtaining informed consent, a trial of topical capsaicin was initiated. Approximately 2 grams of 0.1% capsaicin cream (over-the-counter formulation) was applied to the patient's entire abdomen from the costal margins to the inguinal ligaments. The patient reported an initial intense warming and burning sensation, which peaked at 15 minutes and subsided by 30 minutes.

The patient's retching ceased within 10 minutes of application. At 30 minutes post-application, he reported his abdominal pain had decreased to 2/10. At 60 minutes, he tolerated sips of water without nausea. At 90 minutes, he consumed a full glass of juice and a cracker without incident. He denied any further urge to vomit. Repeat vital signs at 90 minutes were normalized: heart rate 78 bpm, blood pressure 128/84 mmHg. He was observed for an additional 2 hours, during which he remained symptom-free. He was discharged with a diagnosis of CHS, provided with counseling on cannabis cessation, and a tube of 0.1% capsaicin cream for potential future prodromal symptoms. Follow-up laboratory tests at 48 hours via outpatient clinic showed complete normalization of electrolytes and renal function (Table 2), (Figures 1-3).

Discussion

CHS occurs in chronic cannabis users and is characterised by cyclical nausea, vomiting and abdominal pain with symptoms alleviated by hot bathing. Extensive emergency department evaluations are often unrevealing. Our patient's heavy use (2 g/day for 7 years) and hot-shower relief support CHS; his recurrent vomiting produced hypokalaemic metabolic alkalosis and prerenal azotaemia. CHS is frequently missed at first, prompting repeated ED visits and normal investigations, and is classically refractory to standard antiemetics and IV fluids.[2,3]

Table 1. Comprehensive Laboratory and Imaging Data at Presentation

Test	Result	Reference range	Interpretation
CBC			
WBC	12.3 x10^3/μL	4.5-11.0	Mild Leukocytosis
Hemoglobin	15.2 g/dL	13.5-17.5	Normal
Hematocrit	46%	41-53%	Normal
Platelets	310 x10^3/μL	150-400	Normal
Chemistry			
Sodium	138 mmol/L	136-145	Normal
Potassium	3.1 mmol/L	3.5-5.1	Hypokalemia
Chloride	92 mmol/L	98-107	Hypochloremia
Bicarbonate	32 mmol/L	22-29	Metabolic Alkalosis
BUN	28 mg/dL	7-20	Elevated
Creatinine	1.4 mg/dL	0.7-1.3	Elevated
Glucose	102 mg/dL	70-100	Normal
Amylase/Lipase			
Lipase	45 U/L	13-60	Normal
Liver Function Tests			
AST	28 U/L	10-40	Normal
ALT	32 U/L	10-55	Normal
Alkaline Phosphatase	78 U/L	40-130	Normal
Total Bilirubin	0.8 mg/dL	0.2-1.2	Normal
Urinalysis			
Appearance	Clear		
Specific Gravity	1.025	1.005-1.030	Normal
Ketones	Moderate	Negative	Positive
Imaging			
CT Abdomen/Pelvis (w/IV contrast)	No evidence of obstruction, inflammation, appendicitis, or pancreatitis. Mild gastric distension.		

As CHS often fails to respond to first-line antiemetics, clinicians have turned to alternative treatments. Benzodiazepines, benzamides, and atypical antipsychotics (such as haloperidol) have been reported to help in refractory CHS, but such agents carry notable risks.[3,5] CHS is often refractory to standard treatment, and while haloperidol can relieve symptoms, even a single dose may cause serious adverse effects (dystonia, NMS).[5] In contrast, topical capsaicin has emerged as a promising option. Numerous case reports in adults (and more recently in adolescents) describe rapid symptom relief following abdominal capsaicin cream when usual antiemetics failed.[2,5] These successes underscore that our patient's response was consistent with emerging experience: after capsaicin application his intense retching and pain abated within minutes and he tolerated oral intake by 90 minutes.

The efficacy of capsaicin in CHS is hypothesized to stem from its action on the transient receptor potential vanilloid-1 (TRPV1) channel. TRPV1 is a heat-sensitive nociceptor expressed on peripheral sensory neurons that also modulates emesis pathways.[2,4] Capsaicin, a potent TRPV1 agonist, triggers a rapid influx of cations (Ca^2+ and Na+) and release of neuropeptides (such as substance P and CGRP) from these nerves.[4] This accounts for the acute burning sensation we noted after application. Crucially, sustained activation leads to a refractory state: prolonged TRPV1 opening causes desensitization (known as "defunctionalization") of the nerve endings, depleting substance P and other peptides.[6] In effect, the capsaicin

Table 2. Follow-Up Laboratory Data at 48 Hours Post-treatment

Test	Result	Reference range
Potassium	4.0 mmol/L	3.5-5.1
Chloride	101 mmol/L	98-107
Bicarbonate	26 mmol/L	22-29
BUN	12 mg/dL	7-20
Creatinine	0.9 mg/dL	0.7-1.3

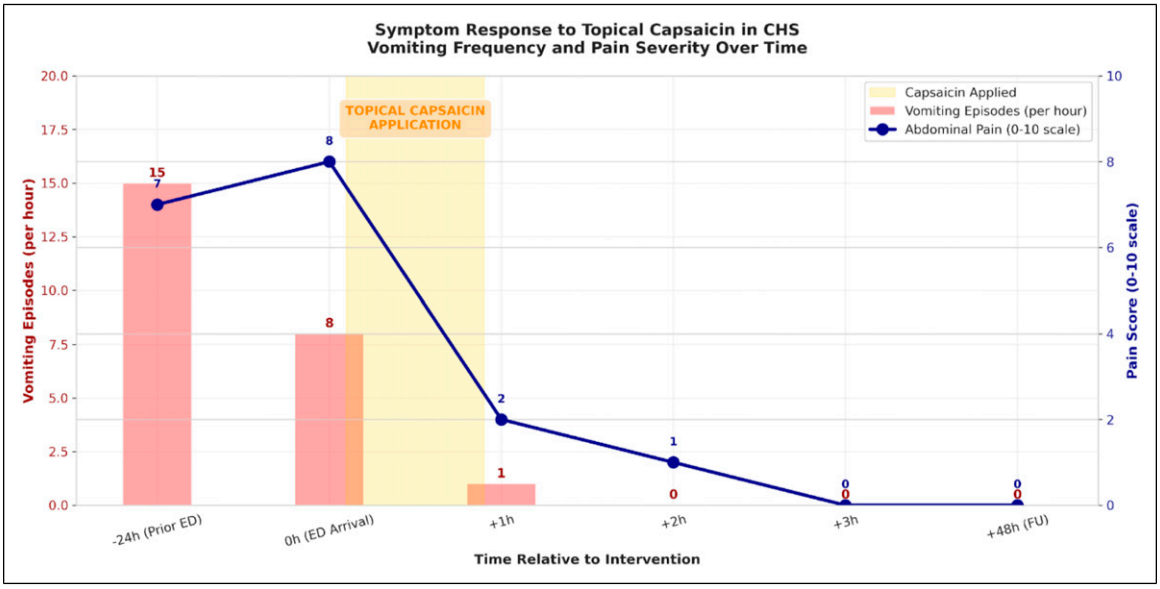

Figure 1. Graphical Timeline of Symptom Severity and Intervention. • **X-axis:** Time (Hours: -24, 0 [ED Arrival], +1, +2, +3, +48 [Follow-up]). • **Left Y-axis (Bar Graph):** Number of Vomiting Episodes per Hour. Shows high frequency at arrival, dropping to zero after capsaicin application. • **Right Y-axis (Line Graph):** Self-Reported Abdominal Pain (0-10 Numeric Rating Scale). Shows pain at 8/10 at arrival, precipitous drop after capsaicin application, sustained at 0/10. • **Annotation:** A clear vertical line and shaded area indicating the time of topical capsaicin application

induces a temporary disconnect of the aberrantly sensitized pain/emetic fibers. This has been described as a novel desensitization analgesia where TRPV1 hyperactivation by capsaicin yields downstream analgesic and antiemetic effects.[4] Capsaicin mimics the effect of the patient's hot-water bathing – activating TRPV1 on cutaneous afferents – but drives it further, producing longer-lasting receptor inactivation. Indeed, CHS patients universally report hot showers as palliative

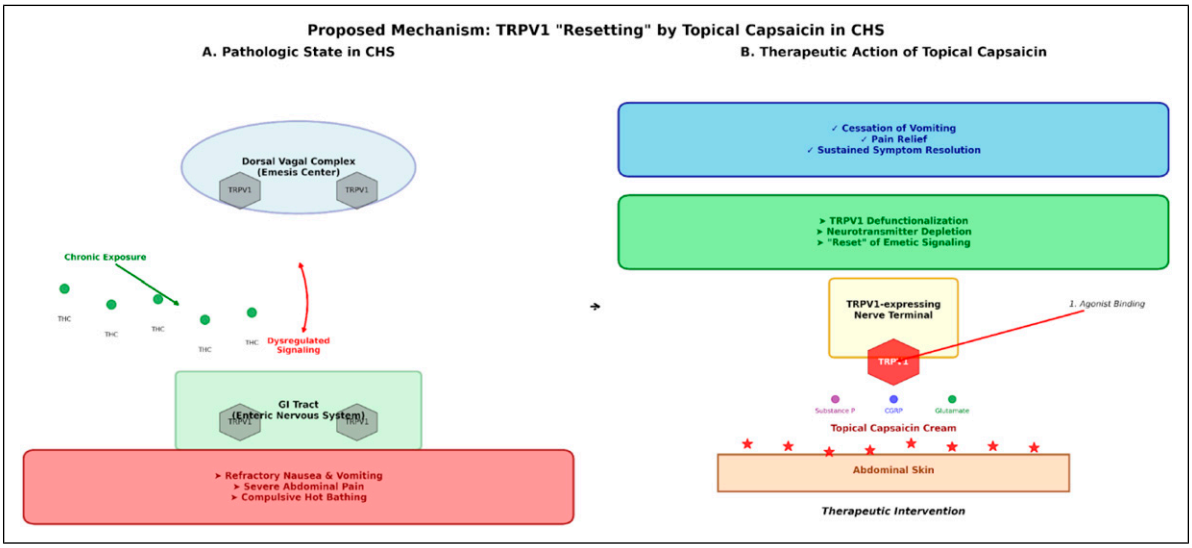

Figure 2. Proposed Mechanism of Topical Capsaicin in CHS. The figure illustrates the hypothesized "TRPV1 Resetting" mechanism: **1. Pathologic State (CHS):** Chronic cannabinoid exposure leads to downregulation and/or desensitization of TRPV1 receptors in the gut and CNS, disrupting normal emetic and thermoregulatory pathways. **2. Intervention (Topical Capsaicin):** High-concentration topical capsaicin acts as a "super-agonist" on cutaneous TRPV1 receptors. **3. Acute Effect:** Initial depolarization of afferent nerves causes burning sensation. **4. Therapeutic Effect:** Sustained application leads to defunctionalization of the TRPV1-expressing nociceptive nerve terminals. This involves depletion of substance P, CGRP, and other neurotransmitters, and temporary inactivation of the nerve terminal. This "resets" the aberrant signaling, mirroring the effect of a prolonged hot shower but in a more potent and sustained pharmacological manner

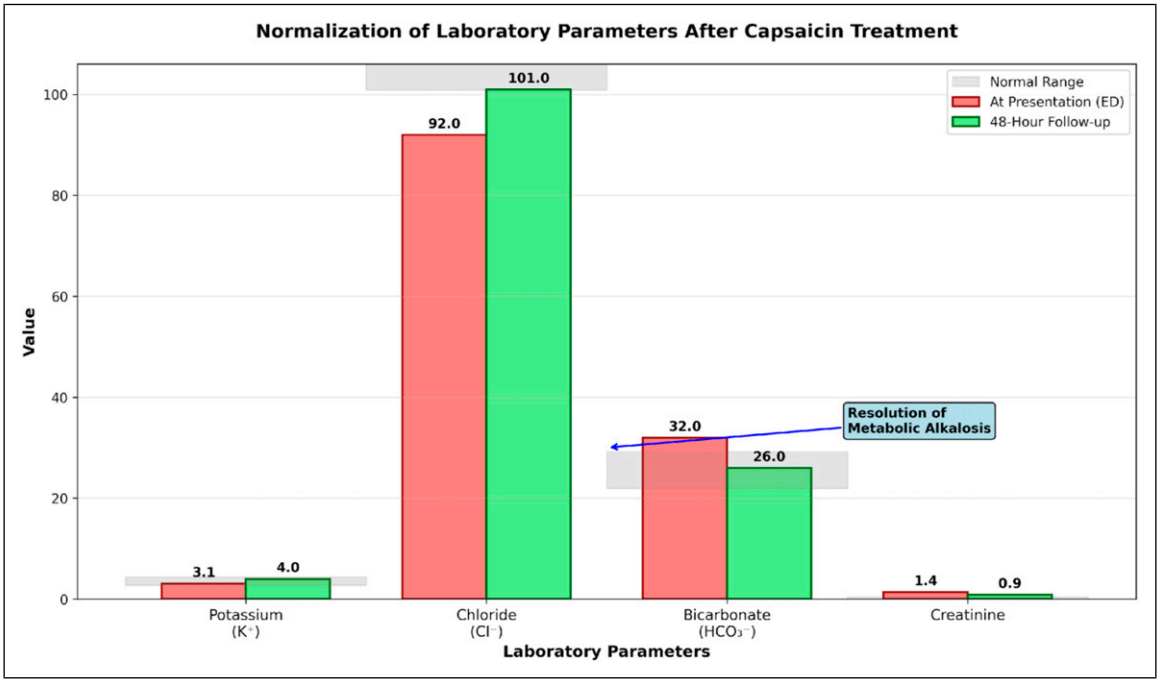

Figure 3. Resolution of Laboratory Abnormalities After Capsaicin Treatment. Bar chart showing normalization of serum electrolytes and renal function at 48-hour follow-up compared to initial presentation, demonstrating correction of the metabolic alkalosis and azotemia secondary to vomiting. The gray band indicates the normal reference range

(likely through TRPV1),[2,4] and topical capsaicin appears to recapitulate that benefit in a more potent way. Some experts have suggested that chronic cannabis may itself dysregulate TRPV1 signaling (perhaps via CB1-mediated hypofunction), so that CHS represents a state of TRPV1 downregulation that can only be reset by a super-agonist like capsaicin.[4,7]

While mechanistically appealing, the evidence base for capsaicin in CHS is still limited. Reports of cannabinoid hyperemesis syndrome treated with topical capsaicin are limited.[4] Available case series suggest potential benefit but lack a controlled study design. For instance, Wagner and colleagues described 43 CHS patient visits treated with capsaicin and found reduced symptom scores, but no change in ED length of stay – partly due to study limitations.[3,4] The first randomized data come from a small pilot trial (Dean et al., 2020) in which 0.1% capsaicin cream applied to the abdomen significantly lowered nausea scores at 60 minutes compared to placebo. In that study, about 30% of the capsaicin group had complete resolution of nausea by 60 minutes (versus none in placebo).[8] Thus, the literature – though not yet robust – consistently shows rapid symptomatic improvement with capsaicin in otherwise refractory CHS.[2,8]

Topical capsaicin has a favorable safety and convenience profile. It is inexpensive, available over-the-counter in various strengths (we used 0.1% cream), and its side effects are limited to local discomfort. The classic side effect is a burning or stinging sensation at the application site.[2,8] Our patient reported intense warmth for about 15–30 minutes, but tolerated it well. Unlike systemic antiemetics or sedatives, capsaicin has no significant central or cardiac effects.[3] This low side-effect burden and ease of use make it attractive as an adjunct in CHS management. In contrast, alternative therapies (including haloperidol, benzodiazepines and antipsychotics) carry risks of sedation, extrapyramidal reactions, or other systemic toxicity, making capsaicin a safer option when first-line treatments have failed.[5]

We performed a structured literature search of PubMed, EMBASE, and Google Scholar to identify published case reports and case series describing the use of topical capsaicin in patients with CHS. Eligible studies were peer-reviewed human reports of any age group in which CHS—defined as chronic daily cannabis use with cyclic vomiting and/or abdominal pain relieved by hot bathing—was treated with topical capsaicin for symptom control. Reviews, editorials, conference abstracts, and studies lacking original patient-level data were excluded, as were publications involving non-topical routes of capsaicin administration. No language or date restrictions were applied. Reference lists of included articles were hand-searched to identify additional relevant reports. The search strategy used combinations of the terms “cannabinoid hyperemesis syndrome,” “CHS,” “cannabis hyperemesis,” and “capsaicin,” using Boolean operators as appropriate. Final inclusion was limited to peer-reviewed articles reporting individual cases or case series. Across the peer-reviewed literature, 18 patients with cannabinoid hyperemesis syndrome treated with topical capsaicin have been reported. These patients were reported across six publications (3 case reports and 2 case series).[2,5,9-11] (Table 3).

Table 3. Summary of Published Case Reports and Case Series of Cannabinoid Hyperemesis Syndrome Treated With Topical Capsaicin

Study (Year)	Patients (age/Sex)	Cannabis use & CHS history	Capsaicin regimen	Outcome (time to relief)
Graham et al (Pediatrics 2017)[5]	2 adolescents (16–17 y, both)	Daily heavy cannabis (≥2 yrs)	0.025% cream applied periumbilically (1 mm thick)	Both had cessation of vomiting and pain within ~30 min
Dezieck et al (Clin Tox 2017)[9]	13 patients (8M/5F; 20–40 y)	Daily cannabis (years)	Topical capsaicin cream (concentration NR; abdominal)	All 13 reported symptom relief (time not specified)
Moon et al (ACG Case Rep J 2018)[10]	23 y male	Daily marijuana (≥5 yrs)	0.075% cream, 15×25 cm abdomen, reapplied q4h	Abdominal pain & nausea improved after 1st dose; nausea gone after 2nd dose; pain resolved after total of 4 doses
Aziz et al (Case Rep Gastrointest Med 2020)[2]	41 y female	Daily cannabis (periods of excess use)	0.1% cream, applied over epigastrium and abdomen, TID	Dramatic pain/vomiting relief within 24 h (near-complete resolution)
Huang et al (Pain Med Case Rep 2025)[11]	34 y pregnant female (11 wk)	Chronic cannabis use	Capsaicin cream (strength NR) applied abdominally	Significant improvement in nausea/pain shortly after application
Current Case (2025)	28 y male	Daily cannabis (~7 yrs, ~2 g/day)	0.1% cream, ~2 g over entire abdomen (single application)	Retching ceased in ~10 min; pain ↓ to 2/10 by 30 min; pain 0/10 by 90 min

All reported CHS cases involved long-term heavy cannabis use (typically daily use for years) and featured the classic hot-shower–relief phenomenon. In each case, standard antiemetics and IV fluids had failed to abort symptoms, prompting use of topical capsaicin. Symptom response was rapid in nearly all reports.[2,5,9-11] For example, a 41-year-old female with refractory CHS experienced near-complete symptom resolution within 24 hours of thrice-daily 0.1% abdominal capsaicin cream.[2] Similarly, a young adult male had marked improvement in pain and vomiting a few hours after two doses of 0.075% cream.[10] In Dezieck et al.'s series of 13 adult CHS patients, all 13 reported relief after capsaicin application.[9] In the only pediatric series, two adolescent CHS patients (ages ~16–17) whose symptoms were resistant to conventional therapy responded fully to 0.025% capsaicin cream.[5] Across studies, capsaicin concentrations ranged from 0.025% up to 0.1%. Higher-strength formulations (0.1%) were often associated with dramatic, rapid relief,[2] whereas even 0.025% cream was effective in adolescents.[5] Patients commonly reported an intense burning sensation initially (lasting ~15–30 minutes), but this was well-tolerated. No serious adverse effects were reported. In a large retrospective review (57 patients), capsaicin use yielded a median pain score drop from 8/10 to 5.5/10, and 42% required no further therapy afterwards; importantly no systemic side effects were observed.[12] Reported CHS cases varied in age, sex, and context, including pediatric, adult, and pregnancy-related presentations. Capsaicin concentrations and dosing regimens differed, but outcomes were consistently positive. Rare treatment failures were associated with low or infrequent dosing, whereas higher concentrations (0.1%) reliably produced effective relief. Overall, topical capsaicin rapidly suppresses symptoms in refractory CHS across diverse populations.[2,5,9-11]

Conclusion

Topical capsaicin produced rapid and sustained symptom relief in this case of refractory cannabinoid hyperemesis syndrome, likely via TRPV1-mediated desensitization mimicking the hot-water response. It is safe, inexpensive, and easily applied, with only transient local burning. Although evidence is limited to case reports, series, and a small pilot trial, capsaicin represents a promising adjunct for refractory CHS. Further research is warranted to optimize dosing and confirm efficacy in larger cohorts.

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Ethical Considerations

Ethical approval was not required for this case report, as it involves a single patient and does not meet the criteria for research requiring institutional review board (IRB) oversight. The use of authorized medications was consistent with clinical practice guidelines, and the management of this individual patient did not require IRB approval.

Consent for Publication

Written informed consent was obtained from the patient prior to publication of this case report and any accompanying images. This consent includes permission for the use of off-label medications as part of the treatment plan. The completed consent form is available to the Editor upon request and will be treated confidentially.

Author Contributions

Muhammad Ilyas: Conceptualization, study design, supervision, and writing – review & editing. Anushka Fernando: Critically reviewed the manuscript, addressed reviewers' comments, and contributed to final revision of the manuscript. Abubakir Choriyev: writing – original draft preparation, and writing – review & editing. Mohammad Adi: Methodology, data interpretation, supervision, and approval of the final manuscript. Ahmed Ashraf: Contributed to data interpretation and critical review of the manuscript. All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work.

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Declaration of Conflicting Interests

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Data Availability Statement

All data pertinent to this case report have been included in this article. Further inquiries can be directed to the corresponding author.

Supplemental Material

Supplemental material for this article is available online.

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