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Cannabinoid Hyperemesis Syndrome in a Resource-Limited Setting: Clinical and Biochemical Insights from an Unusual Water-Triggered Presentation

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Abstract: Cannabinoid hyperemesis syndrome (CHS) is a paradoxical disorder of chronic, heavy cannabis use, defined by recurrent nausea and intractable vomiting that resolve after cannabis cessation. In settings where toxicological screening is unavailable, the diagnosis rests on a careful drug history, the pattern of illness, and the exclusion of alternative causes. A 22-year-old male university student with a 15-month history of daily cannabis use (about three wraps per day, escalating during academic and emotional stress) presented with a three-day history of persistent nausea and recurrent vomiting occurring predominantly after water intake. He was clinically dehydrated but haemodynamically stable. Two months earlier, he had received empirical treatment for Helicobacter pylori at a pharmacy for intermittent nausea, without confirmatory testing. Laboratory evaluation showed hypokalaemia (potassium 3.1 mmol/L) with otherwise preserved renal function; liver enzymes, lipid profile and abdominal ultrasonography were unremarkable. Ondansetron and metoclopramide did not relieve symptoms. Intravenous fluids and potassium replacement were given, and marked symptomatic improvement followed diazepam administration. The patient was counselled on complete cannabis cessation. Vomiting resolved, oral intake was tolerated before discharge, and the patient remained free of nausea and vomiting at one-month follow-up after cannabis abstinence. In the absence of toxicological confirmation, the combination of chronic heavy cannabis use, a characteristic vomiting pattern, exclusion of major alternatives, poor response to conventional antiemetics, and sustained resolution after abstinence supported a clinical diagnosis of CHS.

Keywords: Cannabinoid Hyperemesis Syndrome, Cannabis, Cyclic Vomiting Syndrome, Hypokalaemia, Diazepam, Case Report, Resource-Limited Settings.

Case Report

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1. INTRODUCTION

Cannabinoid hyperemesis syndrome (CHS) is a paradoxical consequence of chronic cannabis use, characterised by recurrent episodes of nausea and intractable vomiting that resolve after cannabis is withdrawn. It was first described by Allen and colleagues in 2004 in a group of chronic cannabis users with cyclical vomiting, and reports have increased as cannabis use has become more prevalent (Allen *et al*., 2004; Perisetti, Goyal, & Tharian, 2020). Because its features overlap with commoner disorders, CHS is frequently mislabelled as cyclic vomiting syndrome (CVS), gastritis, or gastroenteritis, which delays recognition and exposes patients to repeated investigation and ineffective treatment (Abalo *et al*., 2018; Simonetto *et al*., 2012; Sorensen *et al*., 2017).

Cannabis acts principally through Δ9-tetrahydrocannabinol (THC), which binds cannabinoid receptors type 1 (CB1) and type 2 (CB2) distributed across the central nervous system, the enteric nervous system, and peripheral tissues (Abalo *et al*., 2018; Darmani, 2010; Sharkey & Wiley, 2016). Acute cannabinoid exposure is antiemetic, an effect exploited clinically for chemotherapy-induced nausea. The paradox of CHS is that sustained, heavy exposure is associated with the opposite phenotype. The prevailing explanation is that chronic CB1 stimulation leads to receptor desensitisation and downregulation, with altered autonomic and gastrointestinal signalling; additional roles have been proposed for the hypothalamic–pituitary–adrenal (HPA) axis and for transient receptor potential vanilloid 1 (TRPV1) channels, the latter offered as an explanation for the symptomatic relief many patients report with hot bathing (DeVuono & Parker, 2020; Richards, 2017; Richards *et al*., 2018; Sorensen *et al*., 2017). These mechanisms are discussed, with their varying levels of support, later in this report.

There is no confirmatory biomarker for CHS. Urine or blood toxicology can document recent cannabis exposure, but this capability is often absent in rural and resource-limited facilities (Loganathan *et al*., 2024; Simonetto *et al*., 2012). In such settings, a structured drug history and recognition of the clinical pattern carry most of the diagnostic weight. We describe a young man with clinically diagnosed CHS in a Nigerian centre without access to cannabinoid toxicology, in whom vomiting was triggered predominantly by water intake, conventional antiemetics failed, and diazepam produced marked relief. We use the case to examine the biochemical reasoning behind both the syndrome and the observed treatment response, and to consider what can and cannot be concluded from a single clinical observation.

2. CASE PRESENTATION

2.1 Patient Information

A 22-year-old male university student presented to the accident and emergency unit with a three-day history of persistent nausea and recurrent vomiting. He had no documented history of peptic ulcer disease, metabolic, hepatobiliary, neurological or psychiatric disorders, no previous hospitalisation for similar symptoms, no prior surgery, and no known drug allergies.

2.2 Presenting Complaint

Vomiting occurred approximately five times per day, each episode producing an estimated 350 mL of gastric contents. The vomiting was triggered predominantly by water intake, and was non-projectile, non-mucoid, non-bloody and non-bilious; episodes resolved spontaneously without post-episode fatigue or syncope. There was no abdominal pain, fever, diarrhoea, haematemesis, weight loss, or change in bowel habit, and no dizziness, headache, photophobia or altered mental status. He denied recent travel, ingestion of contaminated food or water, and sick contacts.

2.3 Cannabis and Substance History

The patient reported daily cannabis use of about three wraps per day for the preceding 15 months, with escalation to five or more wraps per day during examination periods and emotionally stressful events. He denied alcohol, tobacco, amphetamines, opioids, hallucinogens, and other recreational drugs. There was no family history of gastrointestinal disorders, cyclic vomiting syndrome, or substance use disorder. He identified academic pressure as his principal stressor and reported no significant financial or other psychosocial difficulties.

2.4 Past Medical History

Two months before presentation, he had received empirical treatment for Helicobacter pylori infection at a pharmacy outlet after reporting intermittent nausea, although he had not had epigastric pain, bloating, or other classical dyspeptic features. Treatment consisted of standard triple therapy (a proton pump inhibitor with clarithromycin and amoxicillin or metronidazole). No test of eradication was performed, and the symptoms settled without hospital admission.

2.5 Clinical Findings

On examination he was conscious but weak and clinically dehydrated, with dry lips, dry buccal mucosa and reduced skin turgor. He was not pale, was anicteric and acyanosed, and was afebrile (36 °C). There was no finger clubbing, peripheral lymphadenopathy, or pedal oedema. The pulse was 110 beats per minute, small volume and regular; blood pressure was 95/60 mmHg; respiratory rate was 20 breaths per minute with normal vesicular breath sounds; and random blood glucose was 103 mg/dL. Pupils were equal and reactive, and he was fully oriented with no focal neurological deficit. The abdomen was soft and non-distended, with no tenderness, mass or organomegaly; the liver span was 8 cm, the spleen was not palpable, and the kidneys were non-ballotable. There was no costovertebral angle tenderness, bowel sounds were normoactive, and digital rectal examination was unremarkable.

2.6 Investigations

The laboratory findings are summarised in Table 1. The clinically important abnormalities were hypokalaemia (potassium 3.1 mmol/L) and a microcytic red-cell picture (mean corpuscular volume 69 fL, mean corpuscular haemoglobin 22 pg) with a preserved haemoglobin concentration. Renal function, electrolytes other than potassium, liver enzymes, total protein and albumin, and the lipid profile were within reference limits. Urinalysis showed amber, relatively concentrated urine with trace ketones and 2+ protein. Rapid viral screening was non-reactive, and abdominopelvic

ultrasonography was normal. Cannabinoid toxicology was not available at the facility.

Table 1: Laboratory investigations at presentation. Reference ranges are those of the reporting laboratory. Globulin was recalculated as total protein minus albumin (7.1 − 4.1 = 3.0 g/dL); the value originally recorded (1.0 g/dL) was arithmetically inconsistent and has been corrected

Investigation	Patient value	Reference range	Interpretation
Haemoglobin	13.4 g/dL	13–18 g/dL	Normal
Mean corpuscular volume (MCV)	69 fL	80–100 fL	Low — microcytosis (incidental)
Mean corpuscular haemoglobin (MCH)	22 pg	27–32 pg	Low — hypochromia (incidental)
White cell count	7.0×10^9/L	$4–11 \times 10^9$/L	Normal
Monocytes	14%	2–10%	Mildly raised; non-specific
Platelets	259×10^9/L	$150–400 \times 10^9$/L	Normal
Total protein	7.1 g/dL	6.4–8.3 g/dL	Normal
Albumin	4.1 g/dL	3.8–4.5 g/dL	Normal
Globulin (calculated)	3.0 g/dL	2.0–3.5 g/dL	Normal
Alkaline phosphatase	84 U/L	60–170 U/L	Normal
Aspartate transaminase (AST)	10 U/L	0–12 U/L	Normal
Alanine transaminase (ALT)	7 U/L	0–12 U/L	Normal
Sodium	135 mmol/L	135–155 mmol/L	Normal (lower limit)
Potassium	3.1 mmol/L	3.6–5.5 mmol/L	Low — hypokalaemia
Chloride	97 mmol/L	97–102 mmol/L	Normal (lower limit)
Urea	23 mg/dL	10–55 mg/dL	Normal
Creatinine	0.9 mg/dL	0.6–1.2 mg/dL	Normal
eGFR (CKD-EPI 2021)	123.8 mL/min/1.73 m²	≥ 90 (normal)	Normal; no demonstrable AKI
Total cholesterol / LDL / HDL / TG	174 / 102 / 57 / 104 mg/dL	See laboratory ranges	Within reference limits
Urinalysis	Amber; protein 2+; ketones trace; pH 7.0; SG 1.025; nitrite/leucocyte/blood negative	—	Concentrated urine; changes compatible with dehydration and reduced intake
Viral screen (RVS, VDRL, HBsAg, anti-HCV)	Non-reactive / negative	—	No serological evidence of screened infections
Abdominopelvic ultrasonography	Normal	—	No structural abnormality

2.7 Differential Diagnosis

The initial working diagnosis was acute gastritis, prompted by the recent empirical H. pylori treatment and the persistent vomiting. Several features argued against this and other common causes: there was no epigastric pain, dyspepsia or gastrointestinal bleeding; abdominal ultrasonography and biochemistry gave no evidence of hepatic or pancreatic disease; and there were no features of infection or obstruction. The main differentials and the case-specific features for and against each are set out in Table 2. Chronic heavy cannabis use, the water-triggered vomiting pattern, and the failure of conventional antiemetics moved CHS to the front of the differential.

Table 2: Differential diagnosis, with features supporting and opposing each based on this patient's findings

Differential	Features supporting (this patient)	Features against (this patient)
Cannabinoid hyperemesis syndrome (favoured)	Chronic daily heavy cannabis use with dose escalation; severe recurrent vomiting; poor response to ondansetron and metoclopramide; symptom resolution after diazepam and abstinence; symptom-free at 1-month follow-up	No toxicological confirmation available; hot-water bathing behaviour not documented
Cyclic vomiting syndrome	Stereotyped vomiting episodes; no structural abnormality on imaging	Chronic heavy cannabis use present; documented history did not establish three or more discrete prior episodes with well-defined symptom-free intervals
Gastritis / peptic ulcer disease	Recent empirical H. pylori treatment	No epigastric pain, dyspepsia or postprandial discomfort; no haematemesis or melaena; normal examination
Gastroenteritis	Vomiting	No diarrhoea, fever, sick contacts, or exposure to contaminated food or water; three-day course without infective features
Pancreatitis	Vomiting	No abdominal or back pain; normal abdominal ultrasonography; no biochemical pointer

Differential	Features supporting (this patient)	Features against (this patient)
Intestinal obstruction	Recurrent vomiting	Non-bilious vomitus; soft, non-distended, non-tender abdomen; normoactive bowel sounds; normal imaging
Metabolic causes (e.g. uraemia, hyperglycaemia)	Vomiting with dehydration	Normal renal function and glucose; hypokalaemia explained by vomiting and volume depletion

2.8 Treatment

Management is presented chronologically and is summarised in the clinical timeline (Table 3). Acute management comprised intravenous fluid resuscitation for clinical dehydration, potassium replacement for hypokalaemia, and antiemetic therapy with ondansetron and metoclopramide, which did not relieve symptoms. Following the failure of conventional antiemetics, diazepam was administered, after which there was marked symptomatic improvement.

Definitive management centred on complete cannabis cessation. The patient received counselling on the relationship between his cannabis use and his symptoms and on the importance of sustained abstinence, and follow-up was arranged. Cannabis cessation, rather than any single pharmacological agent, was framed as the cornerstone of long-term management.

2.9 Clinical Course and Outcome

Vomiting resolved with the measures above, and the patient tolerated oral intake without recurrence before discharge. He was discharged in a stable condition on advice of complete cannabis abstinence.

2.10 One-Month Follow-Up

At review one month after discharge, the patient reported continued abstinence from cannabis and remained free of nausea and vomiting. The sustained resolution of symptoms after cessation was consistent with the expected course of CHS and, taken with the rest of the clinical picture, supported the diagnosis.

Table 3: Clinical timeline, constructed from information documented in the case record

Time point	Event
≈ 15 months before presentation	Onset of daily cannabis use (about three wraps/day), escalating during examinations and emotional stress
≈ 2 months before presentation	Intermittent nausea; empirical H. pylori triple therapy obtained at a pharmacy without confirmatory testing; symptoms settled
3 days before presentation	Onset of persistent nausea and recurrent vomiting (~5 episodes/day) triggered predominantly by water intake
Day of presentation	Attendance at accident and emergency; clinical dehydration; hypokalaemia (K^+ 3.1 mmol/L); normal ultrasonography and routine biochemistry
During admission	Ondansetron and metoclopramide given without benefit; intravenous fluids and potassium replacement commenced
During admission	Diazepam administered, followed by marked symptomatic improvement
Before discharge	Vomiting resolved; oral intake tolerated; counselling on complete cannabis cessation
1 month after discharge	Continued cannabis abstinence; symptom-free (no nausea or vomiting)

3. DISCUSSION

3.1 Pathophysiological and Biochemical Basis of CHS

The pathophysiology of CHS is incompletely understood, and much of what is written about it is mechanistic hypothesis rather than established fact. What is well established is that THC acts through CB1 and CB2 receptors, that CB1 receptors are densely expressed at sites relevant to emesis, including the hypothalamus, brainstem, enteric neurons and vagal afferents, and that acute cannabinoid exposure is antiemetic (Abalo *et al*., 2018; Aloisio Caruso *et al*., 2025; Darmani, 2010; Sharkey & Wiley, 2016). The central question in CHS is how chronic exposure produces the opposite effect.

The most widely cited explanation is that sustained CB1 stimulation causes receptor desensitisation and downregulation, with a shift in downstream signalling that disturbs autonomic regulation and gastric motility (Camilleri & Zheng, 2023; DeVuono & Parker, 2020; Sorensen *et al*., 2017). Acute cannabinoids slow gastric emptying centrally, whereas chronic use has been associated with dysregulated motility and, in some reports, a gastroparesis-like state; the direction of this effect and its contribution to symptoms remain uncertain (Abalo *et al*., 2018; Camilleri & Zheng, 2023). CB2 receptors, expressed in immune cells and the gut wall, may modulate inflammatory and autonomic tone, but their specific role in CHS is not established (Darmani, 2010; Sharkey & Wiley, 2016).

Two further mechanisms are frequently proposed. First, chronic THC exposure may disturb hypothalamic regulation of emesis and activate the HPA axis and sympathetic nervous system, increasing sensitivity to emetic stimuli during symptomatic episodes; this is the basis of the stress-response model of CHS and is supported mainly by preclinical work on THC-induced nausea and by clinical hypothesis rather

than by direct human confirmation (DeVuono *et al*., 2020; DeVuono *et al*., 2025; Richards, 2017). Second, upregulation or overstimulation of TRPV1 channels, which are involved in nociception and thermoregulation, has been proposed to explain the relief many patients obtain from hot bathing, with the idea that cutaneous thermal stimulation transiently desensitises TRPV1 and dampens nausea (Richards *et al*., 2018; Sorensen *et al*., 2017). This mechanism is plausible and clinically useful but remains unproven, and hot-water bathing behaviour was not documented in the present patient. Figure 1 summarises the sequence from chronic exposure through proposed signalling changes to the clinical events observed in this case, and is intended as a conceptual overview rather than a claim that any single pathway is the confirmed mechanism of CHS.

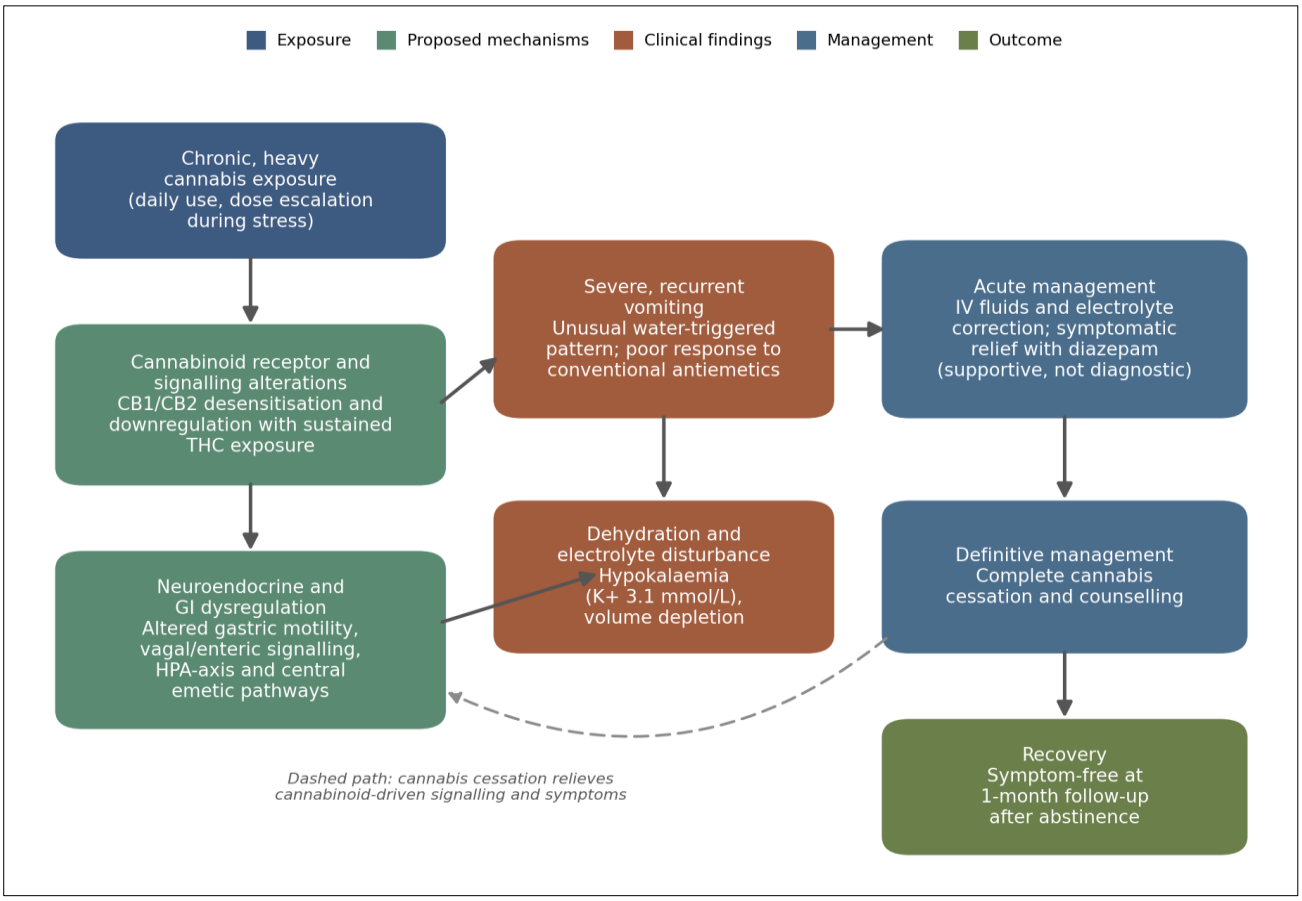


Figure 1: Conceptual overview linking chronic cannabis exposure to the clinical course in this case

Chronic heavy exposure is associated with proposed alterations in cannabinoid receptor signalling and in neuroendocrine and gastrointestinal regulation, which manifest clinically as severe vomiting (here with an unusual water-triggered pattern) and consequent dehydration and hypokalaemia. Acute management provides symptomatic support, while cannabis cessation is the definitive step associated with recovery, shown at one-month follow-up. Colour coding distinguishes exposure, proposed mechanisms, clinical findings, management and outcome; the mechanistic elements are hypotheses of varying strength rather than confirmed pathways.

The demographic and behavioural profile in this case is typical of reported CHS: a young adult man with prolonged, near-daily use and progressive escalation, often in response to stress or the mistaken belief that more cannabis will settle the nausea (Andrews *et al*., 2022; Galli *et al*., 2011; Rotella *et al*., 2022; Russo & Whiteley, 2024; Simonetto *et al*., 2012). CHS is reported more often in males and in the 18–35 year age group, and both duration and intensity of use appear relevant (Andrews *et al*., 2022; Hinton *et al*., 2016; Seabrook *et al*., 2025). Genetic susceptibility has also been proposed but is not established (Russo & Whiteley, 2024).

3.2 Clinical Diagnostic Reasoning

We regarded this as a clinical diagnosis of CHS, made on the balance of evidence rather than on any single confirmatory finding. The supporting elements were: chronic daily and heavy cannabis exposure over 15 months with dose escalation; severe, recurrent and intractable vomiting; the absence of structural gastrointestinal abnormality on examination and imaging; the absence of a convincing infective or metabolic alternative; poor response to conventional

antiemetics; marked improvement after diazepam as a therapeutic observation; cannabis cessation; and the absence of recurrent symptoms at one-month follow-up after abstinence (Gajendran *et al*., 2020; Simonetto *et al*., 2012; Sorensen *et al*., 2017). Because cannabinoid toxicology was unavailable, recent exposure could not be confirmed biochemically, so the diagnosis is best regarded as a probable clinical diagnosis. We have therefore avoided overstating diagnostic certainty, while noting that the convergence of history, illness pattern, exclusion of alternatives, and the response to abstinence made CHS the most coherent explanation.

3.3 CHS versus Cyclic Vomiting Syndrome

CVS was the most important differential because it shares the phenotype of stereotyped, severe vomiting with symptom-free intervals and no structural cause (Hasler *et al*., 2019; Venkatesan *et al*., 2019). The distinguishing feature in favour of CHS is chronic heavy cannabis use, which was prominent here. An earlier version of this case applied the Rome IV criteria for CVS and treated them as fulfilled; on review, the documented history did not clearly establish every criterion, in particular, at least three discrete prior episodes within the preceding year, each with well-defined asymptomatic intervals, were not demonstrated. It is also important to be clear that the Rome IV criteria define CVS and are not diagnostic criteria for CHS; satisfying them would not, by itself, establish CHS, and CHS is distinguished chiefly by the relationship to cannabis and to abstinence (Blumentrath *et al*., 2017; Hawatmeh *et al*., 2017; Stjepanović *et al*., 2024; Venkatesan *et al*., 2019). We therefore present CVS as a genuine differential rather than a diagnosis that was met, and rest the diagnosis of CHS on the totality of the evidence set out above.

3.4 Water-Triggered Vomiting

The most unusual feature of this case was vomiting triggered predominantly by water intake. Vomiting in CHS is usually spontaneous or associated with oral intake generally, and a discrete trigger of this kind has rarely been described. We can only offer hypotheses for it, and none can be confirmed from a single patient. Possible explanations include altered gastric motility and gastric emptying, visceral or gastrointestinal hypersensitivity, an exaggerated gastric reflex to distension, or an abnormal interaction between gastric distension and central emetic pathways in the context of cannabinoid-related gastrointestinal dysregulation (Abalo *et al*., 2018; Camilleri & Zheng, 2023). Each of these is speculative here. The observation is worth recording as a possible addition to the clinical spectrum of CHS, but its mechanism remains uncertain and would need systematic study, ideally with objective assessment of gastric function, before any firm inference could be drawn.

3.5 Interpretation of the Biochemical Abnormalities

The hypokalaemia (potassium 3.1 mmol/L) is best explained by the illness itself: repeated vomiting with gastrointestinal potassium loss, reduced oral intake, and volume depletion, the last also reflected in the low-normal sodium and chloride and the clinical signs of dehydration (Perisetti, Gajendran, *et al*., 2020; Sorensen *et al*., 2017). Hypokalaemia of this kind is commonly reported during the hyperemetic phase of CHS and is a marker of severity rather than a specific finding. The urinalysis, amber, concentrated urine (specific gravity 1.025) with trace ketones and transient 2+ proteinuria, is compatible with dehydration and reduced intake and should not be overinterpreted; the proteinuria in particular is most plausibly transient and functional in this context, though it was not re-checked. Renal function was preserved, with a normal creatinine and eGFR, indicating that significant acute kidney injury was not demonstrated despite the dehydration.

Two limitations of the biochemical assessment deserve emphasis. First, acid–base status was not measured; without venous or arterial bicarbonate or blood-gas data, the metabolic consequences of repeated vomiting (for example, a hypokalaemic, hypochloraemic metabolic alkalosis, which would be the expected pattern) cannot be characterised, and this gap limits the biochemical description of the case. Second, the microcytosis and hypochromia (MCV 69 fL, MCH 22 pg) with a preserved haemoglobin were an incidental finding. Iron studies and haemoglobin electrophoresis were not performed, so this red-cell picture, which in this population could reflect iron deficiency or a thalassaemia trait, among other causes, was not further characterised, and doing so was outside the scope of the acute presentation. It is noted here for completeness and does not bear on the diagnosis of CHS. Notably, the liver enzymes, total protein and albumin were normal, and there was no biochemical evidence of hepatobiliary dysfunction in this patient.

3.6 Diazepam and its Neuropharmacological Basis

The patient's lack of response to ondansetron and metoclopramide and his subsequent improvement after diazepam were clinically striking and warrant a careful rather than an overstated interpretation. Diazepam is a benzodiazepine that acts as a positive allosteric modulator of the GABA-A receptor, increasing the frequency of chloride-channel opening in response to GABA and thereby enhancing inhibitory neurotransmission in the central nervous system. Several mechanisms could plausibly link this action to symptomatic relief in CHS: enhanced GABAergic inhibition may dampen activity in central emetic pathways; it may reduce the anxiety and stress-related amplification of nausea and vomiting, which is relevant given the proposed involvement of the HPA axis and sympathetic activation in CHS; and it may modulate the central neurotransmitter activity implicated in cannabinoid-associated vomiting (Burillo-Putze *et al*., 2022; DeVuono *et al*., 2025; Leu & Routsolias, 2021; Richards, 2017, 2018). These are mechanistic

possibilities rather than demonstrated pathways in this patient.

The diagnostic weight of this response must be qualified. A benzodiazepine response is not pathognomonic of CHS and is not independently diagnostic; benzodiazepines relieve nausea and agitation in a range of conditions, and the effect here is a therapeutic observation, not a diagnostic test. Its value in this case is corroborative: interpreted alongside the chronic cannabis exposure, the characteristic vomiting pattern, the exclusion of major alternatives, and the sustained resolution after cannabis cessation, the response to diazepam was consistent with therapeutic responses reported in CHS and added to the overall clinical support for the diagnosis. It is important to keep the distinction between diagnostic evidence and therapeutic response clear.

3.7 Management

Acute management of CHS is supportive: rehydration and correction of electrolyte disturbance, together with symptomatic control of nausea and vomiting. Conventional antiemetics such as ondansetron and metoclopramide are commonly tried but frequently disappoint, as they did here (Perisetti, Gajendran, *et al*., 2020; Razban *et al*., 2022). Agents reported to give better symptomatic control in CHS include dopamine antagonists such as haloperidol and, as in this case, benzodiazepines, both thought to act on central neurotransmitter pathways involved in emesis (Burillo-Putze *et al*., 2022; Leu & Routsolias, 2021; Richards, 2018; Stubbs & McCallum, 2023). Topical capsaicin, a TRPV1 agonist that may reproduce the relief associated with hot bathing, is another option, though its availability varies and it was not used here (Moon *et al*., 2018; Stumpf & Williams, 2020). The definitive treatment, and the only intervention that addresses the underlying cause, is complete cannabis cessation; continued use perpetuates the syndrome, so counselling and support for abstinence are central to long-term management (Lapoint *et al*., 2018; Reinert *et al*., 2021). The favourable one-month outcome after abstinence in this patient is consistent with this principle.

3.8 Relevance to Resource-Limited Settings

This case illustrates the particular challenge of diagnosing CHS where cannabinoid toxicology and advanced investigation are unavailable, and where awareness of the syndrome among clinicians may be limited (Attout *et al*., 2020; Hernández Liebo *et al*., 2024; Simonetto *et al*., 2012). In such settings, the diagnosis depends on a structured drug history, recognition of the illness pattern, and disciplined exclusion of alternatives, much as occurred here. Stigma around cannabis use can also make patients reluctant to disclose their use, which further complicates recognition (Peixoto *et al*., 2023; Stjepanović *et al*., 2024). A higher index of suspicion for CHS in young patients with unexplained recurrent vomiting, and routine enquiry about cannabis use, can reduce repeated investigation and shorten time to appropriate management and counselling (Cholette-Tétrault & Grad, 2024; Pietrantoni *et al*., 2025; Seabrook *et al*., 2025). Reports of CHS from a range of practice settings, including emergency and primary care, reinforce that the diagnosis is accessible on clinical grounds when the syndrome is considered (Peixoto *et al*., 2023; Senderovich *et al*., 2021).

3.9 Strengths and Limitations

The main strength of this report is a documented course consistent with CHS, including failure of conventional antiemetics, response to diazepam, and, importantly, sustained symptom resolution after cannabis cessation confirmed at one-month follow-up, which strengthens the clinical diagnosis. The report also adds an unusual water-triggered vomiting pattern to the described spectrum of the syndrome. The limitations are equally clear. Cannabinoid toxicology was unavailable, so recent exposure could not be confirmed biochemically and the diagnosis remains a probable clinical one. This is a single case, which limits generalisability. The mechanism of the water-triggered vomiting is uncertain and hypothesis-generating only. The response to diazepam, while clinically useful, is non-specific and not diagnostic. Finally, the absence of acid–base measurement limits the biochemical characterisation of the consequences of repeated vomiting.

4. CONCLUSION

This report describes a clinically diagnosed case of cannabinoid hyperemesis syndrome in a resource-limited setting, in a young man with chronic heavy cannabis use who presented with severe vomiting, an unusual water-triggered pattern, hypokalaemia and dehydration, failure of conventional antiemetics, marked improvement after diazepam, and sustained symptom resolution after cannabis cessation documented at one-month follow-up. In the absence of toxicological confirmation, the diagnosis rested on the convergence of history, illness pattern, exclusion of alternatives, and response to abstinence. The case supports a high index of suspicion for CHS in young patients with unexplained recurrent vomiting and chronic cannabis use, particularly where confirmatory testing is unavailable, and it underlines cannabis cessation as the cornerstone of definitive management. The biochemical discussion offers plausible explanations for both the paradoxical hyperemetic effect of chronic cannabis exposure and the response to diazepam, while keeping established mechanisms distinct from those that remain uncertain.

5. Learning Points

- Consider cannabinoid hyperemesis syndrome in young patients with recurrent, unexplained, intractable vomiting and a history of chronic heavy cannabis use.
- Poor response to conventional antiemetics such as ondansetron and metoclopramide should raise, rather than lower, suspicion of CHS.

- Hypokalaemia and dehydration are common during the hyperemetic phase and reflect repeated vomiting and reduced intake rather than a specific diagnostic marker.
- Where toxicology is unavailable, CHS is a clinical diagnosis based on history, illness pattern, and exclusion of alternatives; the Rome IV criteria define cyclic vomiting syndrome and are not diagnostic criteria for CHS.
- Symptomatic relief may follow benzodiazepines or other centrally acting agents, but such a response is supportive rather than diagnostic; complete cannabis cessation remains the definitive treatment.
- Sustained symptom resolution after abstinence, confirmed on follow-up, is among the strongest clinical arguments for the diagnosis.

Ethics Statement

This report describes a single anonymised clinical case and did not require formal ethical approval under institutional and international guidance for case reports. All identifying information has been removed to protect confidentiality.

Written Informed Consent: Written informed consent for publication of this case, including the clinical details reported here, was obtained from the patient.

Conflict of Interest: The authors declare that they have no conflicts of interest relevant to this case report.

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