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Case Report

Cannabinoid Hyperemesis Syndrome Mimicking Superior Mesenteric Artery Syndrome: A Case of Delayed Diagnosis in an Adolescent

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Abstract

Cannabinoid hyperemesis syndrome is an underrecognized cause of recurrent vomiting, weight loss, and abdominal pain in adolescents, often overlooked due to its nonspecific presentation and overlap with other gastrointestinal conditions. This case report highlights a 13-year-old female who presented with significant weight loss and postprandial bilious vomiting initially attributed to superior mesenteric artery syndrome. Persistent symptoms, despite surgical removal of an incidental ovarian dermoid cyst, prompted reevaluation after nondiagnostic imaging and lack of improvement. Further history two weeks later revealed daily cannabis use, confirmed by a positive urine toxicology screen for tetrahydrocannabinol. Following supportive care and cannabis cessation, her symptoms resolved. This case illustrates how incomplete social histories and incidental findings can delay the identification of cannabinoid hyperemesis syndrome and lead to unnecessary procedures. Early use of urine toxicology screening and validated substance use tools (CRAFFT, BSTAD, S2BI) in adolescents with persistent vomiting and abdominal pain can facilitate timely recognition, reduce hospital length of stay, and improve outcomes.

Introduction

Over the past decade, cannabis use has risen dramatically across the United States. Recent analyses show continued increases, with cannabis use rising from around 7.6% in 2013 to 15.11% in 2021–2022 among adults.[1] The legalization and medicalization of cannabis have increased its accessibility and affordability, contributing to higher consumption and, in some cases, harmful overuse with associated health consequences. According to the 2013–2022 National Survey on Drug Use and Health, cannabis use has become increasingly prevalent among teenagers, with nearly 53% of current or prior marijuana users aged 18 or older reporting that they first used marijuana between the ages of 12 and 17. This pattern has significant implications for pediatric health care providers, given the adverse effects associated with adolescent cannabis use, including poor brain development; deficits in attention, memory, and executive function; increased school dropout rates; poorer academic performance; limited postsecondary education; and higher risks of unemployment.[2,3]

Although cannabis-related neuropsychiatric effects are well documented, gastrointestinal manifestations such as cannabinoid hyperemesis syndrome (CHS) remain underrecognized, and for some patients, it may take up to nine years for clinicians to identify cannabis use as the cause of recurrent gastrointestinal symptoms.[4,5] CHS is characterized by recurrent episodes of intense nausea, abdominal pain, and intractable vomiting lasting 24–48 h in the context of chronic cannabis use. These symptoms are often alleviated temporarily by hot showers, a hallmark feature. Despite being commonly reported in adult populations, CHS is frequently overlooked in adolescents, often resulting in extensive and costly diagnostic workups for alternative etiologies, such as superior mesenteric artery (SMA) syndrome, gastrointestinal obstruction, or intracranial pathology. The nonspecific presentation of CHS, coupled with adolescents' reluctance to disclose substance use and clinicians' under recognition of the disease, frequently leads to substantial delays in diagnosis and treatment.

We report the case of a 13-year-old female who presented with bilious vomiting and was initially suspected to have SMA syndrome based on her rapid weight loss, imaging, and other clinical findings. However, after a prolonged hospital course, coupled with an extensive workup and re-questioning, the underlying etiology

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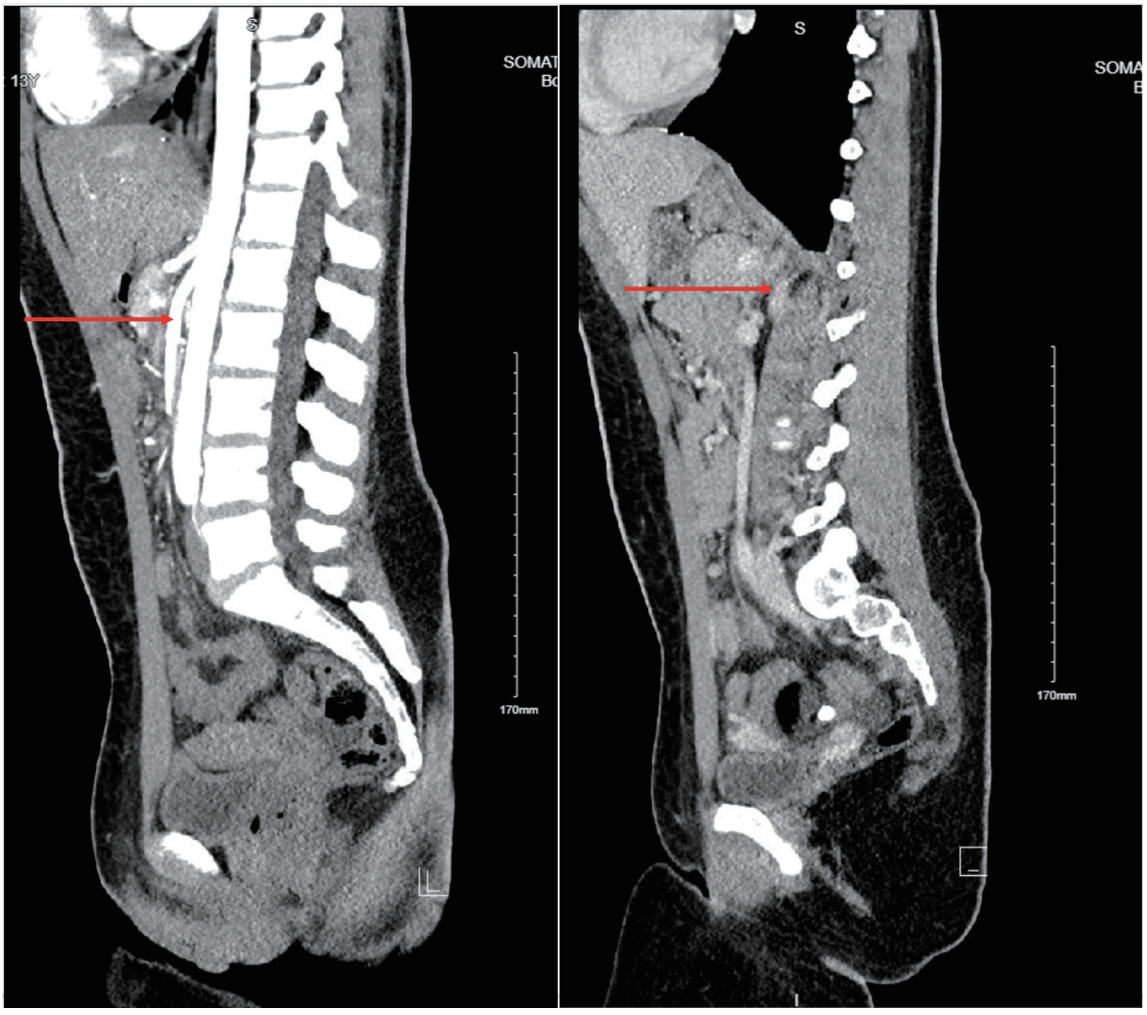

Fig. 1. CT abdomen, sagittal views. Sagittal CT images of the abdomen demonstrating findings suggestive of SMA syndrome. The aortomesenteric angle (red arrow) and distance of the SMA, measuring 24° and 4 mm, respectively, are both below the normal range (38–65° and 10–28 mm). The celiac trunk, SMA, bilateral renal arteries, and inferior mesenteric artery are patent. The stomach and duodenum are under distended, with mild fullness of the distal second portion of the duodenum; however, no definite dilatation of the duodenum or stomach is identified. CT, computed tomography; SMA, superior mesenteric artery.

was found to be CHS due to chronic cannabis use. Ultimately, this case underscores the critical need for routine substance use screening in adolescents presenting with vague gastrointestinal symptoms, and it highlights the potential for diagnostic delay when CHS is not promptly considered.

Case presentation

A 13-year-old female with a past medical history of asthma and no prior surgical history presented to the emergency department with two weeks of postprandial bilious vomiting, intermittent epigastric pain, and a 31-pound weight loss over the last few months. Her history was notable for a restrictive eating pattern, consisting of a single high-protein, low-carbohydrate meal per day at 7 p.m. She denied prior abdominal surgeries, trauma, or similar symptoms in the past.

On presentation, she appeared fatigued but was hemodynamically stable, with epigastric tenderness on examination and no peritoneal signs. Initial laboratory evaluation demonstrated leukocytosis with a white blood cell count of 20.6 K/μL and an absolute neutrophil count of 19.07 K/μL. The comprehensive metabolic panel was notable for mild hyperkalemia (5.2 mmol/L) and low bicarbonate (CO_2 17.9 mmol/L). Nutritional assessment revealed low vitamin D and mildly decreased prealbumin (16 mg/dL), with normal ferritin and iron studies. Gonadotropins (luteinizing hormone 4.2 mIU/mL, follicle-stimulating hormone 4.8 mIU/mL) and thyroid function tests were within normal limits. Inflammatory markers, including C-reactive protein and procalcitonin, were normal, as were lipase, magnesium, and phosphorus levels. Urinalysis showed trace leukocyte esterase with nine white blood cells per high-power field, while electrocardiogram, urine pregnancy test, and abdominal radiography were unremarkable.

Imaging and hospital course

On length of stay (LOS) day 1, an abdominal computed tomography (CT) scan was obtained due to persistent bilious emesis and weight loss. The CT raised concern for SMA syndrome (Fig. 1), demonstrating possible compression of the third portion of the

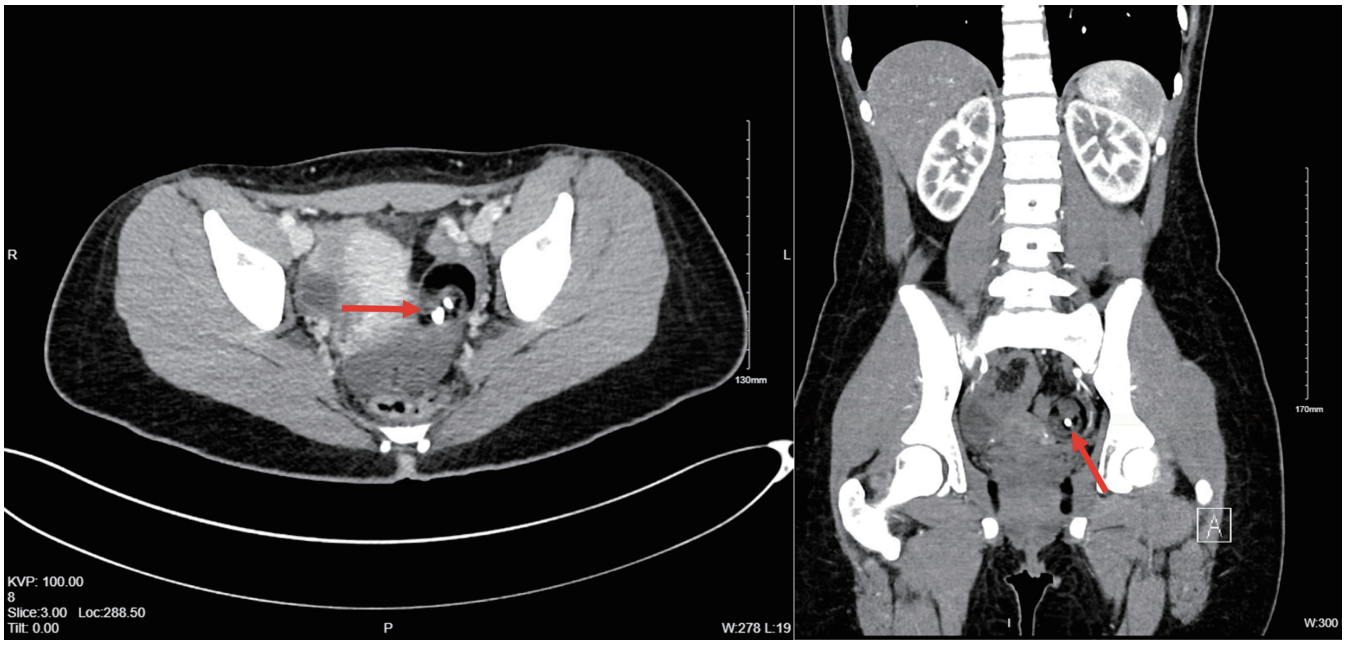

Fig. 2. Pelvic ultrasound images demonstrating a 3.4 × 2.9 × 3.2 cm left ovarian dermoid cyst (red arrow) and a 2.1 cm simple right ovarian cyst (red arrow). Mild pelvic free fluid is also noted.

duodenum between the aorta and the SMA, identified by a reduced aortomesenteric angle (23°) and aortomesenteric distance (4 mm). However, these findings were not accompanied by definitive evidence of obstruction, as there was no significant dilation of the stomach or proximal duodenum. Therefore, an upper gastrointestinal series was performed for further evaluation on LOS day 3, which did not demonstrate any obstruction or features consistent with SMA syndrome. Furthermore, ultrasound of the right upper quadrant on LOS day 3 was within normal limits, and repeat CT imaging on LOS day 10 showed normalization of the aortomesenteric angle (58°) and distance (9 mm), suggesting that the initial findings were transient and may be related to positional factors rather than true vascular compression or may have resolved due to dilation of the duodenum from placement of a nasoduodenal tube for nutrition earlier that same day. Since SMA syndrome is typically diagnosed based on a combination of reduced aortomesenteric angle (<22–25°), decreased distance (<8–10 mm), and radiologic evidence of duodenal compression with proximal dilation, the upper gastrointestinal and repeat CT findings ultimately argued against a diagnosis of SMA syndrome. The CT on LOS day 1 also incidentally identified a 3.4 × 2.9 × 3.2 cm left pelvic dermoid cyst (Fig. 2), which remained a significant finding in the context of her persistent abdominal pain, vomiting, and inability to tolerate oral intake. After a multidisciplinary discussion, the decision was made to proceed with surgical management.

Surgical intervention and postoperative course

On LOS day 6, the patient underwent laparoscopic removal of the 3.4 cm left ovarian dermoid cyst. The procedure was uncomplicated, and the cyst was successfully excised. Postoperatively, the patient was initiated on peripheral parenteral nutrition due to poor oral intake, though she tolerated only minimal clear liquids.

Following laparoscopic excision of a 3.4-cm left ovarian dermoid cyst, the patient had a complicated postoperative course with persistent gastrointestinal symptoms. She developed recurrent bilious emesis, dizziness, and diffuse abdominal pain.

Antiemetic and analgesic therapy were initiated postoperatively; the regimen is listed below in Table 1. Given persistent nausea and emesis despite standard antiemetics for a week, haloperidol injections 2 mg q6h PRN were subsequently initiated, with reported improvement in nausea. Capsaicin cream was applied topically for symptomatic relief, consistent with transient receptor potential vanilloid 1 (TRPV1)-mediated mechanisms implicated in CHS pathophysiology.

Abdominal examination revealed epigastric tenderness with guarding, while the abdomen remained soft with normal bowel sounds. Given the lack of clinical improvement following ovarian surgery, further evaluation was pursued, including an abdominal X-ray imaging (Fig. 3) to assess for bowel obstruction and a contrast-enhanced CT of the abdomen and pelvis (Fig. 4) to evaluate for suspected SMA syndrome. The patient was subsequently transferred to the surgical service for continued management.

Her postoperative course was further complicated by persistent intolerance of oral intake, necessitating placement of a peripherally inserted central catheter by Interventional Radiology for nutritional support. Ongoing bilious emesis and moderate epigastric pain raised concern for pancreatitis or an alternative gastrointestinal etiology contributing to her prolonged postoperative course, while ovarian pathology, including visceral pain–mediated autonomic stimulation or intermittent torsion or inflammatory irritation from the dermoid cyst, remained in the differential diagnosis. Due to continued emesis, a Dobhoff tube was placed into the fourth portion of the duodenum under fluoroscopic guidance. Although enteral feeds were initiated, they were discontinued because of nausea and throat discomfort, and the patient remained largely nil per os, requiring intravenous fluids, parenteral nutrition, and acid suppression.

Severe epigastric pain with vomiting persisted, and the patient intermittently used hot showers for symptomatic relief. Social work was consulted due to weight loss, restrictive eating behaviors,

Table 1. Pharmacological interventions during hospitalization

Medication	Dose, route, frequency & duration	Clinical indication
Antiemetics		
Ondansetron	8 mg IV q8h PRN, 2 doses administered	Postoperative nausea and vomiting
Ondansetron ODT	4 mg oral dissolving tablet q8h PRN, up to 7 days	Nausea and vomiting
Metoclopramide	10 mg IV q6h PRN, as needed	Nausea refractory to ondansetron
Haloperidol	2 mg IV q6h PRN, as needed	Refractory CHS-associated hyperemesis (off-label)
Scopolamine	1 patch transdermal, continuous, as needed	Nausea and vomiting
Fosaprepitant (Emend)	Single dose IV, 1 dose administered	Refractory nausea and vomiting
Analgesics		
Ketorolac	15 mg IV q6h PRN, 3 doses administered	Postoperative pain
Morphine	2 mg IV q4h PRN, 1 dose administered	Breakthrough postoperative pain
Acetaminophen	15 mg/kg IV q6h scheduled, continued throughout	Scheduled analgesia
Anti-inflammatory		
Dexamethasone	4 mg IV once, single dose	Postoperative nausea prophylaxis
Acid suppression		
Famotidine	20 mg IV q12h scheduled	Gastroprotection and acid suppression
Pantoprazole	20 mg IV q12h scheduled	Acid suppression during NPO/TPN
Topical/Adjunct		
Capsaicin cream	0.025–0.075% topical, applied as needed	CHS symptom relief via TRPV1-mediated mechanism
Bowel regimen		
Polyethylene glycol 3350	17 g oral daily, scheduled	Constipation management
Nutritional support		
TPN (Pediatric 2-in-1)	95 mL/hr IV continuous, until enteral tolerance achieved	Nutritional support during NPO period
Fat emulsion 20% (Intralipid)	14.85 mL/hr (297 mL) IV continuous, co-administered with TPN	Lipid supplementation
Respiratory (chronic medications continued)		
Fluticasone (Flovent)	110 mcg/actuation, 2 puffs inhaled BID, continued throughout admission	Asthma maintenance
Albuterol	2 puffs inhaled q4h PRN	Asthma rescue therapy

BID, twice daily; CHS, cannabinoid hyperemesis syndrome; IV, intravenous; NPO, nil per os; ODT, orally disintegrating tablet; PRN, as needed; TPN, total parenteral nutrition; TRPV1, transient receptor potential vanilloid 1.

and psychosocial stressors. On LOS day 12, the patient reported daily marijuana use prior to admission for appetite suppression and weight loss, confirmed by a urine drug screen performed using a standard immunoassay panel that was positive for tetrahydrocannabinol (>50 ng/mL). Based on chronic cannabis use, symptom relief with hot showers, and clinical features, a diagnosis of CHS was made, in addition to anorexia nervosa, restrictive type. Supportive care for CHS treatment included continuation of peripheral parenteral nutrition due to poor oral intake, along with close monitoring of electrolytes (CMP, magnesium, phosphorus), complete blood count, and liver function tests. Symptom control was achieved using a stepwise antiemetic regimen, including intravenous haloperidol as first-line therapy, followed by metoclopramide and ondansetron as needed, with electrocardiographic monitoring for QTc prolongation. Adjunctive therapy with topical capsaicin cream applied three times daily was initiated for nausea relief. The patient was also encouraged to utilize hot showers for symptomatic relief. Multidisciplinary care included consultation with psychiatry, social work, and adolescent medicine to manage comorbid eating disorders and substance use. The patient was counseled on cannabis cessation and referred for outpatient substance use treatment, as this represents the definitive management of CHS. Prior

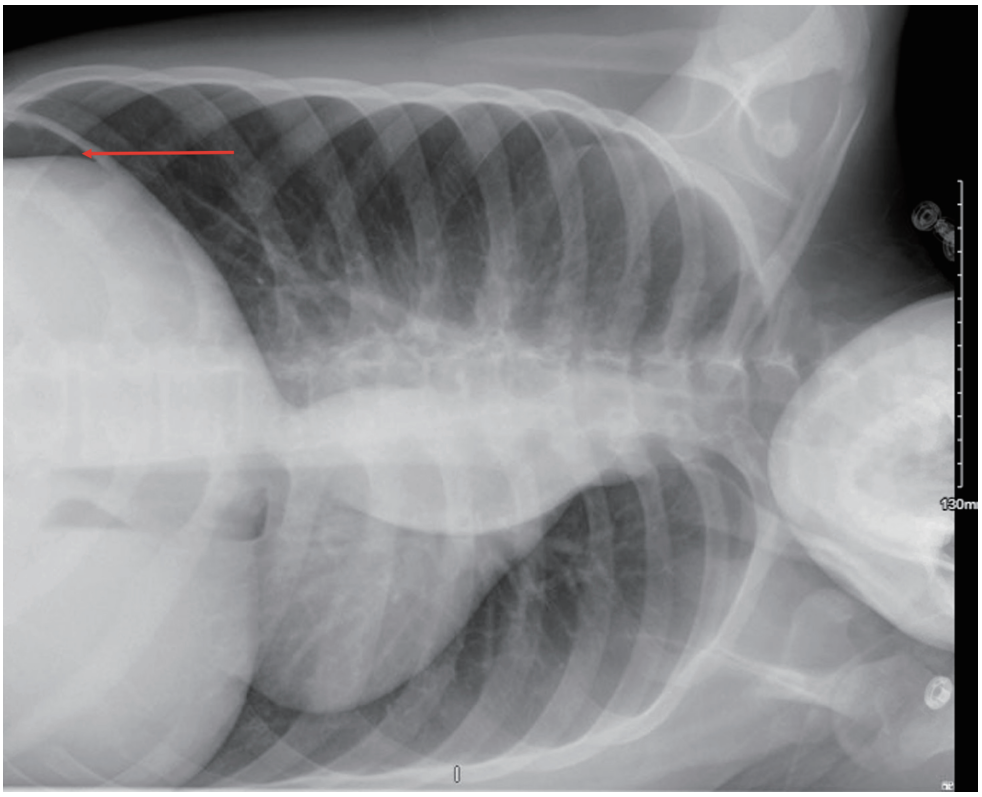

Fig. 3. Postoperative chest X-ray demonstrating residual pneumoperitoneum following laparoscopic procedure. Chest X-ray obtained on postoperative day 1 for evaluation of abdominal pain. A small amount of nondependent air is identified beneath the diaphragm (red arrow) (left lateral decubitus position demonstrating free air above the liver), consistent with residual pneumoperitoneum following a recent laparoscopic procedure.

to discharge, parenteral nutrition was discontinued, and she was transitioned to nasoduodenal feeds, which were later advanced to an oral diet (Fig. 5).

Discharge follow-up course

The patient was discharged on LOS day 24 after successfully meeting a 2,000 kcal/day caloric goal, advanced by 200 kcal every one to two days per eating disorder protocol, for three consecutive days without emesis or enteral support. Table 2 displays the full objective clinical course before and after CHS-directed management. She was cleared by pediatric psychiatry and adolescent medicine and outpatient follow-up was arranged given insurance barriers to inpatient eating disorder unit admission.

At her two-week postoperative surgical follow-up, examination revealed a possible suture granuloma at the umbilical incision site with minimal serous drainage and no surrounding cellulitis, managed with a five-day course of amoxicillin-clavulanate. She was cleared to return to full activity. Final pathology confirmed a benign mature teratoma with no evidence of malignancy or dysplasia (alpha fetoprotein <1.9, human chorionic gonadotropin <1).

At six-week follow-up, she reported complete resolution of abdominal pain, nausea, and vomiting. Secondary amenorrhea was noted, attributed to cumulative weight loss, prolonged hospitalization, and prior cannabis use; return to baseline was anticipated with continued nutritional recovery, and pelvic ultrasound was scheduled.

Two months after discharge, she was readmitted to the emergency department and was re-admitted with recurrent intractable vomiting and inability to tolerate oral intake. She reported a total 31-pound weight loss since her initial presentation (approximately 158 to 127 lbs) and acknowledged recent cannabis use, with self-reported cessation one month prior; hot showers continued to be her primary source of symptomatic relief. She was managed with antiemetics, electrolyte repletion, nasoduodenal tube feeds, and a single dose of fosaprepitant, with clinical improvement allowing discharge approximately two weeks later.

At a three-month outpatient visit for weight loss and malnutrition, she formally met DSM-5 criteria for atypical anorexia nervosa and was enrolled in a multidisciplinary program comprising weekly psychotherapy, nutritional rehabilitation with a registered dietitian, and ongoing adolescent medicine follow-up. By four months after initial hospitalization, she confirmed two months of cannabis abstinence and reported open communication with her mother regarding both her substance use and eating disorder.

At her five-month follow-up, she remained abstinent from cannabis, was actively engaged in therapy, and denied any recurrence of nausea, vomiting, or abdominal pain, representing sustained clinical remission at approximately six months post-cessation.

Discussion

CHS remains underdiagnosed and frequently overlooked in pediatric patients, often leading to delayed diagnosis and misattribution to other gastrointestinal or surgical conditions. This is largely due to its nonspecific presentation, coupled with challenges in obtaining a complete substance use history. Together, these factors con-

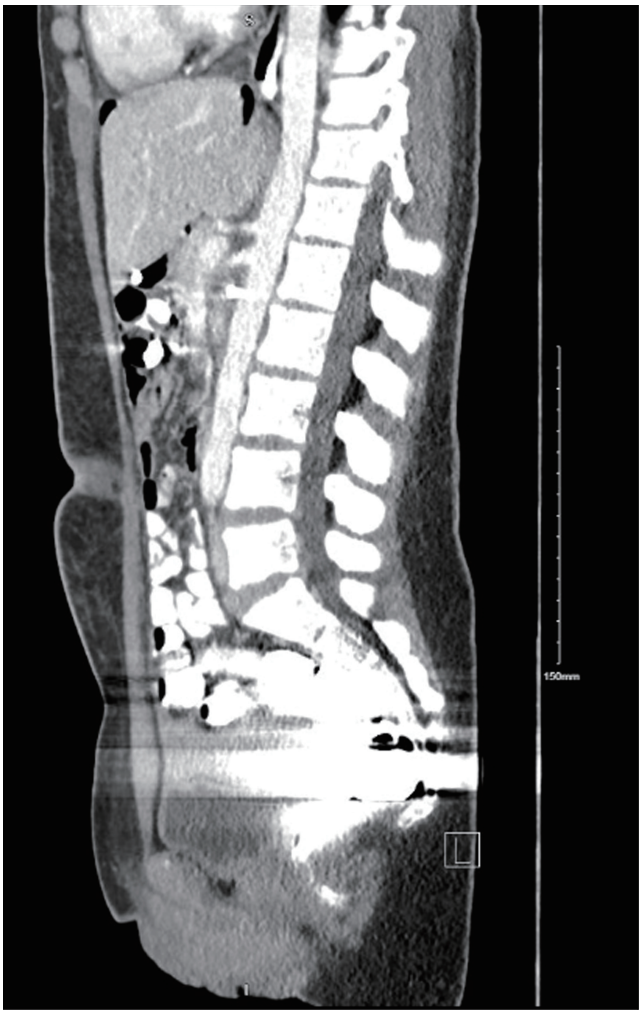

Fig. 4. Sagittal view CT abdomen and pelvis for re-evaluation of SMA syndrome on postoperative day 4. Repeat CT abdomen and pelvis obtained on postoperative day 4 for persistent vomiting. Compared to prior imaging, the aortomesenteric angle improved from 23° to 58° and the aortomesenteric distance from 4 mm to 9 mm, likely reflecting duodenal distention following nasoduodenal tube placement. No bowel obstruction was identified. Mild pneumoperitoneum is consistent with recent surgery. CT, computed tomography; SMA, superior mesenteric artery.

tributed to delays in appropriate diagnosis and treatment.

The pathophysiology of CHS remains incompletely understood, but current evidence suggests that chronic cannabis exposure disrupts normal endocannabinoid signaling. Acute cannabis use typically produces antiemetic effects through activation of cannabinoid receptor type 1 (CB1) in central emetic pathways. However, chronic stimulation may lead to CB1 receptor desensitization or downregulation, resulting in paradoxical nausea and vomiting.[6] Additional proposed mechanisms include disruption of CB1-mediated gastrointestinal motility and dysregulation of hypothalamic and autonomic thermoregulatory pathways. Symptom relief with hot showers is thought to involve activation of the transient receptor potential vanilloid 1 (TRPV1) receptor, which is influenced by both heat and cannabinoids and plays a role in pain and nausea pathways.[6–8]

Clinically, CHS often presents with recurrent vomiting and abdominal pain, symptoms that are common and nonspecific and therefore insufficient to establish a definitive diagnosis. As a result, clinicians frequently rely on supplemental imaging and laboratory evaluation, which may delay recognition of the underlying cause. In this case, early diagnostic focus was directed toward a structural cause, specifically suspected SMA syndrome based on CT findings. However, discordance between imaging findings and the patient's overall clinical presentation made SMA syndrome less likely early in the course. More common etiologies, including gastroenteritis, gastritis, and ovarian pathology, were initially considered. The persistence of bilious emesis despite appropriate management and repeatedly nondiagnostic studies ultimately led SMA syndrome to be considered as a diagnosis of exclusion. The incidental identification of an ovarian dermoid cyst further complicated clinical reasoning, shifting attention toward a surgical etiology and contributing to the decision to proceed with operative management. Despite successful excision, the patient's symptoms persisted, prompting reconsideration of alternative diagnoses. Ultimately, the continued vomiting, reported relief with hot showers, and later disclosure of chronic cannabis use led to the diagnosis of CHS.

This sequence highlights how incidental findings with plausible symptom overlap may shift diagnostic focus away from functional or toxicologic causes. In adolescents presenting with unexplained vomiting and abdominal pain, clinicians should therefore maintain a broad differential diagnosis and remain attentive to potential substance-related etiologies, even in the presence of structural abnormalities.

Despite the nonspecific nature of CHS symptoms, up to 98% of patients report symptomatic relief with hot showers.[9] This characteristic feature can provide important clinical insight and should be routinely assessed when obtaining a pediatric history. Additionally, prior studies suggest that SMA syndrome and CHS may present concurrently, with overlapping features such as weight loss and recurrent vomiting. Therefore, in patients presenting with rapid weight loss and gastrointestinal symptoms, SMA syndrome should not be considered the sole diagnosis.[10] A thorough evaluation for chronic cannabis use is essential to avoid missing CHS, particularly given its increasing prevalence among adolescents.

The diagnosis of CHS in adolescents is primarily clinical and may be supported by established frameworks such as the Rome IV criteria, which state that a new diagnosis of CHS must include all 3 of the following criteria stereotypical episodic vomiting resembling cyclic vomiting syndrome in terms of onset, duration, and frequency; presentation after prolonged cannabis use; and relief of vomiting episodes with sustained cessation of cannabis use.[11,12] In terms of pattern recognition, CHS follows a characteristic progression through prodromal, hyper-emetic, and recovery phases. The prodromal phase involves nausea and discomfort that may be temporarily relieved by continued cannabis use. This phase is followed by a hyper-emetic phase marked by severe cyclic vomiting, dehydration, and temporary relief with hot showers, which progresses to a final recovery phase with resolution of symptoms after cessation of cannabis.[11] Multiple diagnostic frameworks described in the literature demonstrate substantial overlap, consistently identifying core features such as chronic cannabis use, cyclic severe nausea and vomiting, hot shower relief, abdominal pain, and symptom resolution with cessation. Additional features such as symptom recurrence with cannabis resumption, normal bowel habits, negative workup, and significant weight loss were inconsistently reported and thus less reliable for diagnosis.[6] A large case series of 98 patients further supports these clinical features,

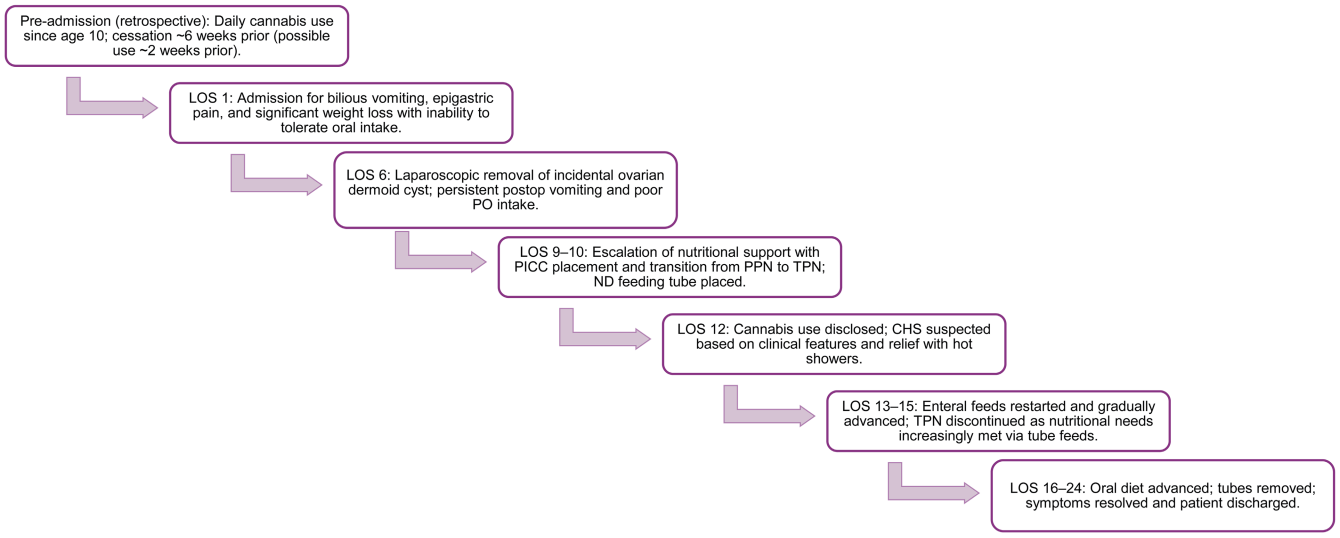

Fig. 5. Clinical timeline of disease progression and management. LOS, length of stay; ND, Nasoduodenal; PICC, peripherally inserted central catheter; PO, per os, by mouth; PPN, peripheral parenteral nutrition; TPN, total parenteral nutrition.

demonstrating that most individuals with CHS are younger than 50 years with chronic cannabis use, recurrent cyclic vomiting, and abdominal pain, with many reporting symptomatic relief from hot showers.[13] Importantly, among patients who discontinued cannabis, the majority experienced resolution of symptoms, reinforcing cessation as the cornerstone of management.[13] Consistent with pediatric clinical pathways,[14] diagnosis is based on clinical features with targeted evaluation, including laboratory studies, urine toxicology, and selective imaging, while management emphasizes supportive care (intravenous fluids, electrolyte correction) and CHS-directed therapies such as haloperidol and topical capsaicin, given the limited efficacy of traditional antiemetics. Definitive treatment remains strict cannabis cessation, supported by multidisciplinary care including psychiatry, social work, and substance use programs.

The patient in this case demonstrated several hallmark features consistent with CHS, including cannabis use, recurrent intractable vomiting, and symptomatic relief with hot showers, supporting CHS as a key contributor to the clinical presentation despite coexisting anorexia nervosa.

These findings underscores the importance of maintaining a broad differential diagnosis, particularly in adolescent patients, in whom functional, toxicologic, and behavioral etiologies are frequently underrecognized. To contextualize this diagnostic course, it is important to consider the clinical relevance of the ovarian dermoid cyst identified on imaging. Most dermoid cysts are asymptomatic and are frequently discovered incidentally during imaging performed for unrelated indications.[15] However, dermoid cysts can overlap significantly conditions as they may also cause nausea, vomiting, and abdominal pain, when complicated by ovarian torsion, a surgical emergency, as the size and weight of the cyst can increase ovarian mobility and predispose the ovary to twisting

Table 2. Objective clinical course before and after CHS-directed management

Parameter	Admission (LOS day 1)	After CHS-directed management (LOS day 12)	At discharge (LOS day 24)
Emesis episodes/day	15	5–8	0
Antiemetic use	Zofran, Reglan	1st line: Haldol; 2nd line: Reglan; 3rd line: Zofran. Capsaicin cream TID	None
Oral intake	Unable to tolerate PO	Meeting 50% of goal tube feeds	Full PO intake
Nutrition support	PPN required	Transitioned to TPN	Oral diet
Weight (kg)	57.7 kg	59.1 kg	58.1 kg
Electrolytes	Abnormal (↓K (3.3))	Improving	Normalized
Urinalysis	Cloudy, 2+ ketones,	THC positive	Not performed due to clinical improvement
Cannabis use	Stopped using 6 weeks prior to admission, but undeclared	Disclosed prior heavy cannabis use and subsequent hiatus	Abstinent
Other symptoms	Vomiting, abdominal pain, 31 lb recent weight loss,	Improved (particularly with capsaicin, Haldol and hot showers)	Resolved

CHS, cannabinoid hyperemesis syndrome; LOS, length of stay; PO, per os, by mouth; PPN, peripheral parenteral nutrition; THC, tetrahydrocannabinol, psychoactive compound in cannabis; TID, three times a day.

around its vascular.[16,17] In this patient, the presence of a dermoid cyst in the setting of persistent abdominal pain, bilious vomiting, and inability to tolerate oral intake raised concern for a potential gynecologic etiology and contributed to the decision to pursue surgical management. Although the cyst was successfully excised, the patient's symptoms persisted postoperatively, prompting reconsideration of alternative diagnoses. Ultimately, the continued episodes of vomiting, the patient's report of symptomatic relief with hot showers, and the later disclosure of chronic cannabis use led to the diagnosis of CHS.

This case emphasizes the importance of routine, targeted screening for substance use in adolescents. In this patient, urine toxicology screening was not performed until LOS day 12, despite persistent unexplained symptoms. Earlier use of structured screening approaches recommended by the American Academy of Pediatrics (AAP), including validated questionnaires such as CRAFFT (Car, Relax, Alone, Forget, Friends, Trouble), BSTAD (Brief Screener for Tobacco, Alcohol, and Other Drugs), or S2BI (Screening to Brief Intervention), which can guide both education and clinical management,[18–20] may have facilitated earlier identification of cannabis use. Urine toxicology is not a recommended screening tool and should be used when unexplained clinical signs and symptoms persist after questionnaire-based screening, when history is unreliable, or when a previously known substance use history is indicated. In this case, if screening had been applied early in the hospital course and yielded an unreliable or negative self-report in the setting of persistent unexplained symptoms such as nausea and vomiting, urine toxicology would have been clinically warranted as a confirmatory adjunct consistent with AAP guidance.[21] This stepwise model emphasizes patient confidentiality and an adolescent–provider trust approach while also enabling timely laboratory confirmation to address unexplained clinical symptoms.[20,22]

Although limited by its single-patient design, this case aligns with existing literature supporting routine screening for substance use in adolescents and timely recognition of CHS. Some limitations include that these findings may not apply across diverse pediatric populations, especially since CHS presentation might differ by age, sex, ethnicity, and cannabis use patterns. Additional limitations include the disruption of the hospital course by the incidental finding of a dermoid cyst and subsequent surgical intervention. Furthermore, the patient's coexisting anorexia nervosa complicates attribution of symptoms solely to CHS, making causality less clear. However, the presence of recurrent cyclic episodes of severe emesis, confirmed cannabis use on urine toxicology, and symptomatic relief with hot showers and capsaicin, all of which are characteristic of CHS and not typical of restrictive eating disorders, support CHS as the primary diagnosis. In contrast, the patient's weight loss and reduced oral intake are more consistent with anorexia nervosa and may have contributed to baseline gastrointestinal symptoms. However, since both cannabis cessation and nutritional rehabilitation were initiated during her hospitalization, the relative contribution of each intervention to symptom resolution cannot be definitively distinguished.

Building upon these limitations, future research must prioritize the unique needs of adolescent and young adult populations, who remain underrepresented in current CHS literature. Most available data are derived from adult cohorts, limiting generalizability to younger patients. With rising cannabis use among adolescents, there is an urgent need for multicenter studies to accurately characterize the prevalence, age of onset, clinical presentation, and hospital course of CHS in pediatric and young adult populations. Additionally, studies should evaluate whether the implementation of standardized urine toxicology screening protocols in adolescents presenting with unexplained vomiting leads to earlier CHS diagnosis, reduces unnecessary imaging and surgical interventions, and shortens hospital stays. The utility of substance use screening tools such as CRAFFT and BSTAD in identifying cannabis-associated gastrointestinal symptoms also warrants validation in this demographic. Finally, further investigation is needed to assess the long-term outcomes of early CHS diagnosis in youth, including its impact on gastrointestinal health, cannabis cessation success, and recurrence rates. These efforts are critical to improving timely recognition, appropriate management, and long-term outcomes for this increasingly vulnerable population.

Take-home messages

CHS should be routinely considered in adolescents presenting with intractable vomiting, abdominal pain, relief after hot showers, or rapid weight loss, especially given the rising prevalence of cannabis use in this population.

Implementing protocolized urine toxicology screening early in the evaluation of adolescents with unexplained gastrointestinal symptoms can prevent diagnostic delays, unnecessary procedures, and prolonged hospital stays.

Per AAP recommendations, universal screening for substance use in adolescents using tools like CRAFFT, BSTAD, or S2BI should be standard practice to guide clinical decision-making and support early intervention.

CHS and other conditions, like SMA syndrome, may present overlapping symptoms. A thorough evaluation, including substance use history, is critical to avoid missing concurrent or alternative diagnoses.

Conclusions

This case of CHS misdiagnosed as SMA syndrome emphasizes the need to broaden differential diagnoses early in the clinical course and to routinely include CHS when evaluating adolescents with intractable vomiting. Early implementation of urine toxicology screening protocols may prevent unnecessary diagnostic procedures, decrease hospital LOS, and enable more timely management.

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Conflict of interest

The authors have no conflict of interest related to this publication.

Author contributions

Study concept and design (PR, SB, DS, SK, SB), acquisition of data (PR, SB, DS, SK, SB), analysis and interpretation of data (PR, SB, DS, SK, SB), drafting of the manuscript (PR, SB, DS, SK), critical revision of the manuscript for important intellectual content (PR, SB, DS, SK, SB), administrative, technical, or material support (SB), and study supervision (SB). All authors have made

a significant contribution to this study and have approved the final manuscript.

Ethical statement

The study was performed in accordance with the ethical standards of the institutions to which we are affiliated and with the Declaration of Helsinki (as revised in 2024). Ethical approval was not required for this retrospective case report. Written informed consent was obtained from the patient's parent/guardian for publication of this case report. All imaging and pathology materials were fully de-identified in accordance with journal guidelines and ethical standards.

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