ISSN: 2692-5400 DOI: 10.33552AJGH.2026.04.000595

Academic Journal of Gastroenterology & Hepatology

Iris Publishers

Case Report

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Cannabis Hyperemesis Syndrome provoking Mallory Weiss Tear: A Domino Effect

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Received Date: September 07, 2026

Published Date: September 24, 2026

Abstract

Cannabinoid hyperemesis syndrome (CHS) is clinic disorder that occurs in the setting of chronic, heavy cannabis use and is characterized by recurrent, stereotypical episodes of nausea, vomiting. It mainly transpires due to dysregulation of the endocannabinoid system secondary to prolonged Δ9-tetrahydrocannabinol exposure. When vomiting is severe and protracted, CHS can precipitate secondary complications such as superficial tears and complete rupture of the esophagus. We present the case of a young woman with a decade of daily cannabis use and prior concern for CHS who developed coffee-ground emesis and an endoscopically confirmed Mallory-Weiss tear in the setting of intractable vomiting. The case is further complicated by several concurrent contributors to upper gastrointestinal injury, recent nonsteroidal anti-inflammatory drug and high-dose aspirin exposure, erosive gastritis, duodenitis, and a small hiatal hernia—as well as a recent ruptured ectopic pregnancy, illustrating both the diagnostic challenge of attributing a Mallory-Weiss tear to CHS and the importance of considering CHS in the differential for recurrent, otherwise unexplained hyperemesis. The syndrome is frequently misdiagnosed, leading to extensive and repeated workups, and its recognition matters because sustained cannabis cessation is the only established route to resolution.

Introduction

Cannabis hyperemesis syndrome a complex clinical syndrome that materializes due to action of cannabinoid and its active metabolites on the central nervous system and gut in patients with specific genetic polymorphisms [1]. These patients present with cyclic vomiting syndrome characterized by 4 episodes of vomiting per hour to atleast 15 episodes per day [2]. With repeated episodes of vomiting, these patients are predilected to develop complications such as hypovolemia, acute renal failure, pneumomediastinum, pneumopericardium, and pneumo-retroperitoneum, MWT, Boerhaave syndrome [3-6]. Dopamine antagonism in the medulla and serotonergic antagonism in the gastrointestinal tract has afforded clinical efficacy for suppressing symptoms [2]. Prompt in identification, fluid repletion, antiemetics, benzodiazepines, anti-psychotics and counselling to prevent future drug usage will shorten the clinical course of this hyperemesis syndrome and associated complications [6]. It is not uncommon for repeated vomiting episodes to suddenly raise the intrathoracic and intraabdominal pressure. When this transpires, partial tear (MWT) and complete rupture of the esophagus (Boerhaave syndrome) will ensue [7-9]. We present a rare clinical case where patient presented with CHS which was later complicated by MWT. Our clinical case demonstrates the cause-effect relationship between cannabis hyperemesis syndrome (CHS) and Mallory Weiss Tear (MWT) which transpires in rare clinical scenarios. This case report discusses the

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pathophysiology, clinical features, diagnosis and treatment of both cannabis hyperemesis and MWT.

Clinical Case

The patient is a 33-year-old female with history of anxiety, GERD, borderline personality disorder, PTSD, prior right salpingectomy, and recent ruptured left ectopic pregnancy status post laparoscopic left salpingectomy on 08/23/2026. She presented with persistent nausea and vomiting, poor oral intake, coffee-ground/blood-streaked emesis, and burning epigastric abdominal pain. She also reported lower abdominal discomfort, constipation, and continued vaginal bleeding.

Her recent history was notable for prolonged daily marijuana use for approximately 10 years with prior concern for cannabinoid hyperemesis syndrome/cyclic vomiting syndrome. She reported cessation of marijuana approximately one week prior to this admission. She also had recently been taking ibuprofen following surgery and received high-dose aspirin in the emergency department.

Given the history of persistent retching and coffee-ground emesis, there was concern for an upper gastrointestinal bleed, including possible Mallory-Weiss tear and NSAID-related gastritis. She was started on IV pantoprazole 40 mg twice daily and sucralfate 1 g three times daily. Hemoglobin was trended and remained stable without evidence of significant ongoing blood loss. EGD demonstrated a Mallory-Weiss tear, erosive gastritis, duodenitis, and a small hiatal hernia (Figure 1). Gastroenterology recommended continuation of PPI therapy, anti-reflux lifestyle modifications, and outpatient follow-up in approximately 2 weeks. Gastric biopsies were obtained and remained pending at discharge, with plan to treat for H. pylori if positive.

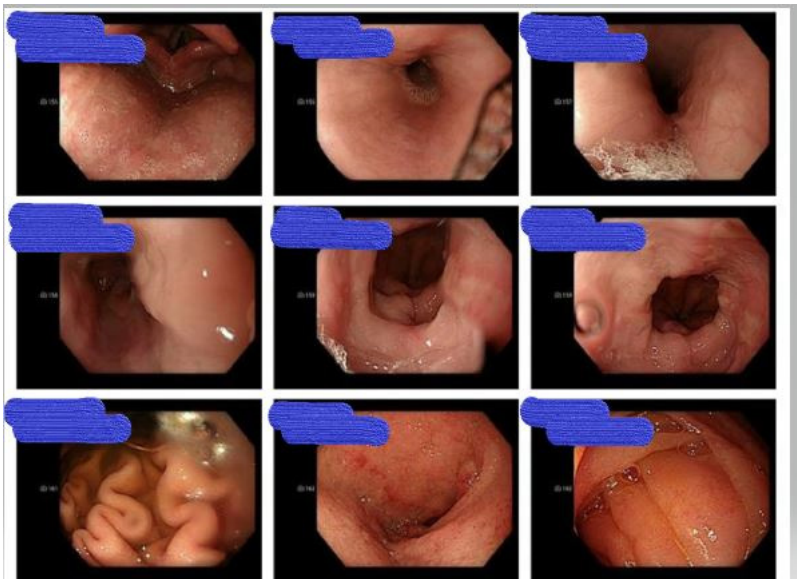

Figure 1:

Her nausea and vomiting were felt to be multifactorial, with suspected contribution from cannabinoid hyperemesis/cyclic vomiting syndrome as well as the acute gastrointestinal process. She was treated with IV ondansetron as needed, with subsequent clinical improvement and improvement in oral intake. Continued complete marijuana cessation was strongly encouraged.

Regarding her recent ruptured ectopic pregnancy, she had initially been admitted on 08/21/2026 with a left adnexal mass and rising beta-hCG and received methotrexate. She subsequently developed a ruptured left ectopic pregnancy with significant hemoglobin decline and underwent laparoscopic left salpingectomy on 08/23/2026. During this admission, CT demonstrated moderate pelvic free fluid, which was felt to be likely postoperative. Beta-hCG remained mildly elevated at 128.6. She continued to report vaginal bleeding and lower abdominal discomfort. OB/GYN evaluated her and initiated tranexamic acid for reported heavy vaginal bleeding. Her hemoglobin remained stable. Outpatient OB/GYN follow-up was recommended.

There was also concern for possible acute cholecystitis based on CT findings of gallbladder wall thickening, pericholecystic fluid, and surrounding inflammatory changes. HIDA scan on 08/24/2026 was negative. Repeat right upper quadrant ultrasound demonstrated persistent gallbladder wall thickening and positive sonographic Murphy sign without gallstones, with borderline prominence of the CBD (Figure 2). There was no definitive evidence of acute cholecystitis, and she was managed conservatively with clinical monitoring.

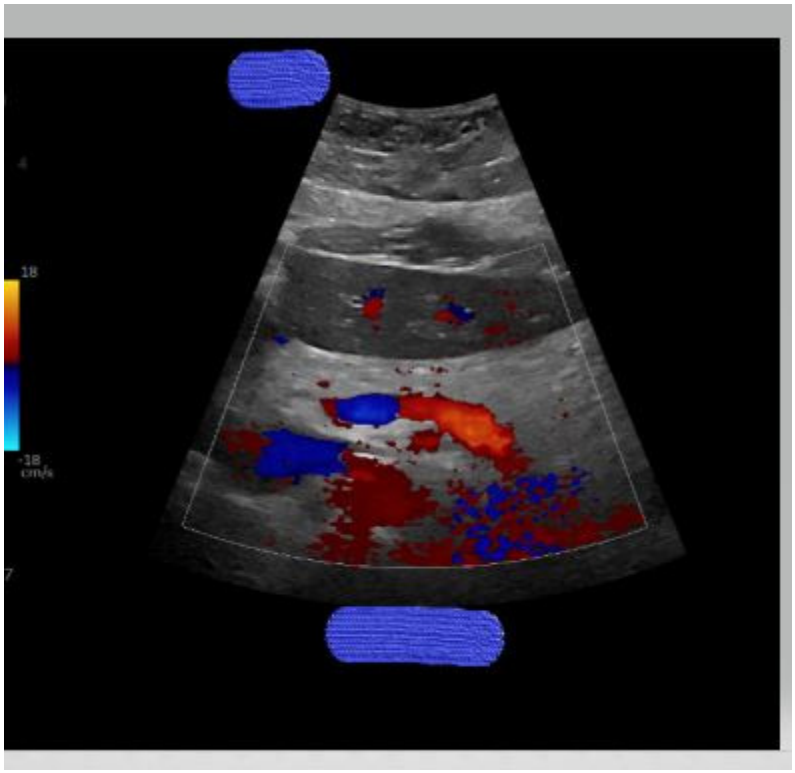

Figure 2:

She had hypokalemia attributed to persistent vomiting and poor oral intake. Potassium was repleted as needed and electrolytes were monitored. Constipation was managed with MiraLAX 17 g daily and bowel regimen as tolerated.

At the time of discharge, nausea and vomiting had improved, oral intake was improving, and hemoglobin remained stable without recurrent significant hematemesis or melena. She was clinically stable for discharge with outpatient follow-up.

Discussion

Cannabis usage can result in dependence thus increasing the chances of drug abuse in at least 9% of the patients using it [10,11]. Mallory Weiss Tear (MWT) is most common non-variceal hemorrhage and its incidence is estimated to be 10-20% [12]. MWT is commonly prevalent in men as compared to women [12]. The potential risk factors that can be attributed for the causation of MWT include prolonged vomiting, coughing, cardiopulmonary resuscitation and endoscopy [12].

Various mechanisms have been proposed for the occurrence of hyperemesis syndrome in the setting of cannabis abuse. The main active ingredient of cannabis is 9-tetrahydrocannabinol (THC). In chronic abuse of cannabis, THC is bound to accumulate in the fat tissues and eventually gets disengaged into the blood circulation in slow bursts in chronic abusers [13,14]. Due to above-mentioned preferential volume of distribution, its half-life tends to be prolonged. Specifically, its early and late half-life within first 8 hours and 4 weeks is estimated to be 2h and 12.6 days [15]. As a result, chronic abusers are more predilected to have protracted systemic effects. THC mainly acts on the G-protein coupled receptors CB1 and CB2 [14]. CB1 receptors are predominantly present in the central nervous system and gut. THC activates the CB1 receptors and mediated decreases gastric emptying, which partially accounts for hyperemesis syndrome [14].

Low doses of THC are bound to have antiemetic effect due to partial agonistic action of CB1 receptors [16]. However, with higher doses or chronic cannabis use, THC will have an antagonist effect on the CB1 receptors due to desensitization, internalization and dysregulated signaling, thus giving rise to increased emetic effect [10,16]. Moreover, chronic use of cannabis was also shown to increase the synthesis and secretion of other neurotransmitters in the synaptic cleft including serotonin, substance P and dopamine [10,16].

Another receptor that is involved in the pathogenesis of cannabis induced hyperemesis is Transient receptor potential vanilloid 1 (TRPV1) receptor [10]. This receptor is primarily expressed in the chemo-receptor trigger zone of the brain as well as myenteric plexus and epithelial cells of the gastrointestinal tract [10]. Physiologically, activation of this receptor promotes antiemetic effect, partly mediated by downregulation of the P-substance within the neural circuits of nucleus tractus solitarius [10]. Chronic abuse

of cannabis has been documented to suppress the TRPV1 receptors and signaling cascade, thus accounting haphazard gastric motility, emptying and eventually giving rise to hyperemesis [10].

During stress and starvation, increased ACTH release from the pituitary leads to heightened lipolysis and release of THC from the adipocytes, thus providing justification for emetic effect rapid heartbeat, sweating, tremors and high blood pressure [10]. Evidence of sympathetic dysfunction also contributes to hyperemesis with chronic cannabis use as patients usually present with tremors, agitation, hypertension and sweating [10].

In our patient, cannabis abuse induced hyperemesis syndrome can regarding as a triggering point for precipitating MWT. Various mechanisms have been for speculated for transpiration of this clinical entity. Repeated and persistent vomiting raises the intra-gastric pressure [12]. To counterbalance this, strong gastric muscular contractions will transmit the shear forces toward lower end of esophagus, where superficial mucosal lacerations ensue [12]. Notably, most of the lacerations are confined to the superficial mucosal layers, although incrimination of deeper submucosal and muscular layers might result in upper gastrointestinal bleeding [12].

The clinical presentation of cannabis hyperemesis includes epigastric abdominal pain associated with nausea, vomiting. In few instances, vomiting episodes start and increase in intensity in few hours. The vomiting episodes can be as frequent as 5 times per hour. The severity of vomiting can precipitate upper abdominal pain.

The upper abdominal pain in this clinical scenario can be become more diffuse to involve other areas of the abdomen. Thar being said, differential diagnosis should include pancreatitis, cholecystitis and choledocholithiasis, and gastritis, all of which should be ruled with out with appropriate investigations. Excess vomiting episodes can trigger hypovolemia, hypotension, acute renal failure and electrolyte disturbances [17,18]. As vomiting episodes progress in in frequency and severity can ultimately culminate in MWT, Boerhaave's syndrome and pneumomediastinum [19].

Patients with CHS usually present with repeat episodes of vomiting that can be as high 15 episodes per day, which can be triggering factor for developing complications such as hypovolemia, hypotension, pneumomediastinum and MWT [6,8]. In patients with CHS, MWT should be suspected when non-bloody vomitus which is closely followed by transformation into bloody vomitus [20]. The clinical spectrum for MWT includes hematemesis, melena hematochezia and anemia if bleeding is excessive [12].

Diagnosis of cannabis hyperemesis syndrome is mostly based on clinical presentation. Thorough clinical history particularly type and duration of cannabis use should be clearly documented. Urine drug screen for cannabis, amphetamines and cocaine should be ordered promptly on suspicion of cannabis abuse. Upper endoscopy is warranted when MWT is suspected. The hallmark sign of MWT during endoscopy is the appearance of superficial mucosal tear at the gastroesophageal function, most commonly at the cardia along the less curvature or posterolateral wall of distal esophagus [12,21]. Although single mucosal tear is most common, it is not uncommon to witness more than one tear 35% of the cases [20,22].

Cannabis hyperemesis syndrome is usually self-limiting and usually resolves spontaneously. Some patients get relief from hyperemesis syndrome with compulsive hot showers due to fact that temperature above 41F will stimulate the cutaneous TRPV1 channels, thence altering the emetic pathways in combination with reduced sympathetic activity and cutaneous steal syndrome [6,1,23]. Adjuvant management of fluid losses with fluid repletion, antiemetics, with attention to nutrition and pain is necessary [6]. In cases where, it is refractory, usage of benzodiazepines is most commonly associated with success, although haloperidol, antipsychotics and capsaicin have been used previously [24,25]. Sometimes, consultation with psychiatrist is mandatory to manage cannabis induced behavioral side effects [25]. The underlying mechanistic basis for clinical efficacy of antipsychotics in CHS is due their antagonistic effect on the dopamine receptors 2 in the chemo trigger zone (CTZ), which primarily regulates nausea and vomiting [1]. Counselling and referral to drug cessation programs to avoid future drug consumption becomes very much part of the treatment paradigm.

Most of the Mallory Weiss Tears are uncomplicated and resolve spontaneously with conservative therapy [20]. In occasional cases where there is massive bleeding, injecting vasoactive substances into celiac or gastric arteries will stop the bleeding and will prevent the need for surgical intervention [20]. Need for aggressive intervention in MWT include ongoing severe bleeding, visible vessels, presence of clots and severe comorbidities [12]. The Glasgow-Blatchford bleeding score is generally used to predict the need for aggressive intervention [26]. A Glasgow-Blatchford bleeding score less than 1 indicates that interventions are less likely to be necessary and patients can be managed with conservative management [26]. First line intervention for bleeding MWT is endoscopic injection with epinephrine, sclerosants, bipolar electrocoagulation probes, and heater probes [27]. If these interventions do not control bleeding, then endo-clips, and band ligation can be pursued [27-29]. If these strategies fail to control the bleeding, then endoscopic arterial embolization and surgery can be relied upon, although the prior has increased rebleeding rates as compared to former [30]. Concurrently, high dose proton pump inhibitor should be started for 72 h after bleeding episodes [26]. Risk factors for high rates of rebleeding including coagulopathy, portal gastropathy, anticoagulant use, advanced age and presence of comorbidities such as coronary artery disease, & chronic kidney disease should be recommended to continue oral proten pump inhibitors for 14 days to reduce the risk of rebleeding [12,26].

Take Home Messages

a. Patients Cannabis abuse is at higher risk of developing hyperemesis syndrome.

b. The principal ingredient of cannabis THC primarily acts of CB1, CB2 and TINV receptors in brain and gut through coordinated action predisposing them to hyperemesis syndrome.

c. Increases bouts of vomiting episodes raises the intraabdominal pressure and counteractive muscular contractions provoke the onset of MWT.

d. This is one of the rare cases where Cannabis hyperemesis syndrome has ultimately resulted in the MWT through repeated vomiting episodes.

e. Mallory Weiss Tear usually presents with hemetemsis melena and hematochezia

f. Upper endoscopy is usually diagnostic for demonstration of tear in the gastroesophageal junction.

g. Most MWTs resolve spontaneously with conservative management.

h. In MWTs with significant bleeding, endoscopic therapies and surgery might be necessary

i. Counselling to avoid cannabis consumption is very much emphasized to avert the future complications including MWT

Declarations

a. Ethical Approval and Consent to participate: Not Applicable

b. Consent for publication: Consent taken

c. Availability of data and materials: Not Applicable

d. Competing interests: Not Applicable

e. Funding: Not Applicable

f. Acknowledgements: Not applicable.

Authors' Contributions

Conceptualization, S.H.K&JT; Methodology, S.H.K& JT; Software, N.G.; Validation, N.A; Formal Analysis, N.A.; Investigation S.H.K, JT.&AKC; Resources, N.A.; Data Curation, N.A.; Writing– Original Draft Preparation, S.H.K, JT.&AKC; Writing– Review & Editing, S.H.K, JT, AKC, KR, & AR.; Visualization, S.H.K & BC.; Supervision, S.H.; Project Administration, S.H.

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