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A case of failure to treat cannabinoid hyperemesis syndrome with topical capsaicin

Vishal Patel*; Elaine Smith

***Vishal Patel**

Department of Pharmacy, Community Medical Center, USA

Email: Vishal.Patel@rwjbh.org

Abstract

Cannabinoid hyperemesis syndrome is a disorder associated with cannabis use resulting in cyclic vomiting. There is a lack of strong evidence to guide treatment. Recently, there have been many cases reporting the efficacy of topical capsaicin cream completely resolving the symptoms of the syndrome. The published evidence would seem to indicate a nearly 100% resolution rate for the cream with cases of failures difficult to find. We present a failure of topical capsaicin cream to manage the symptoms of a patient with a recurrent episode of cannabinoid hyperemesis syndrome.

Keywords

cannabinoid hyperemesis syndrome; capsaicin cream; cannabis

Abbreviations

CHS: Cannabinoid hyperemesis syndrome; CT: Computed tomography; TRPV1: Transient receptor potential vanilloid-1 receptor; TCA: Tricyclic antidepressants

Introduction

Cannabinoid hyperemesis syndrome (CHS) is a cyclic vomiting syndrome that can occur in patients who use cannabis chronically [1,2]. CHS is associated with episodes of nausea, vomiting and abdominal pain, which is contrary to the proposed therapeutic role of cannabinoids as anti-emetics [1,3]. Management is not well defined, with medications such as antipsychotics and tricyclic antidepressants (TCA) showing some efficacy in limited studies and traditional treatments for nausea and vomiting such as antihistamines and serotonin antagonists showing less efficacy [4,5]. New evidence has been emerging regarding the use of topical capsaicin cream for treatment of CHS in both pediatric and adult patients, providing a potentially safer alternative for medical management [4,6,7]. While many of the current case series published report resolution rates of 100%, we present a case of CHS which topical capsaicin cream failed to resolve [6,8,9].

Case Presentation

A 25-year-old male presented to the emergency room with complaints of non-bloody nausea and vomiting starting 3 hours prior to arrival. Patient denied abdominal pain, diarrhea, fever, headache, gastric bleeding, visual disturbances, chest pain, dyspnea, recent travel or sick contacts. The patient had a past history of cannabis use leading to nausea and vomiting. On the day of presentation, his symptoms started shortly after smoking cannabis; he was unsure of the strain involved in this or prior uses. The patient reported that his current symptoms were similar to past episodes of nausea and vomiting after smoking cannabis, often lasting 12 hours or more. Initial vital signs included a blood pressure of 144/75, heart rate of 80 beats/minute, respiratory rate of 20 breaths/minute, temperature of 97.2°F and pulse oxygen saturation of 99% on room air. Laboratory testing included a complete metabolic panel, complete blood count with differential, lipase, and liver function tests. Potentially significant results included a white blood cell count of 13000/mcL with neutrophils of 87%, alanine aminotransferase of 141 units/L and aspartate aminotransferase of 108 units/L. Additional testing included a computed tomography (CT) scan of the chest with contrast and a CT scan of the abdomen and pelvis with contrast. The CT chest was positive for bilateral patchy ground glass airspace opacification. The CT abdomen/pelvis was positive for patch airspace disease in the right lung base. These findings were thought to suggest aspiration from vomiting as symptoms were not consistent with pneumonia or other causes of right sided basilar opacities. On serial abdominal exams, the patient's right upper quadrant was nontender suggesting against cholelithiasis, cholecystitis, or choledocholithiasis as the cause of elevated liver function testing. The patient denied concurrent alcohol use, recent alcohol use, or daily alcohol use. He denied daily medication use, including over-the-counter drugs. The managing physician believed his lab abnormalities to be caused by the patient's recurrent vomiting and not due to other pathology.

Given the patient's history of identical symptoms after smoking cannabis in combination with his history of present illness, cannabinoid hyperemesis syndrome became the working diagnosis. After discussing the risks and benefits with the patient, he was initially treated with a 1-millimeter-thick coating of capsaicin cream 0.025% in a circle around the periumbilical region. Upon application, the patient noted a burning sensation. The patient continued to experience repeated episodes of nausea and vomiting and fifteen minutes later, required ondansetron 4 mg IV for unmitigated symptoms. The patient received a reapplication of the capsaicin cream an hour after the first application with no additional relief or improvement. The patient was then treated with additional ondansetron 4mg IV, metoclopramide 10 mg IV and diphenhydramine 25 mg IV before beginning to develop abdominal pain. Further treatment with promethazine 25 mg IV followed by haloperidol 2mg IV provided complete and lasting resolution of the patient's symptoms 10 hours after initial presentation. No additional capsaicin cream was applied after the first two doses due to a lack of perceived benefit per patient and treatment team. Patient was moved to the emergency department observation unit and upon discharge received counseling, famotidine 20 mg and ondansetron 4 mg for maintenance of relief, outpatient follow-up with a primary doctor and gastroenterology specialist if symptoms persisted.

Discussion/Conclusions

Within the United States, there is an increasing movement towards cannabis law reform. Multiple states have decriminalized the use of cannabis for medical or recreational purposes. With this, there may be increases in adverse reactions presenting to an emergency department, such as CHS. This is particularly concerning as there is little evidence for why these adverse effects occur or how best to manage them. Treatment for CHS ranges from supportive therapy with intravenous fluids or hot showers to utilizing medications such as ondansetron, metoclopramide, diphenhydramine, promethazine, haloperidol and capsaicin cream [1,7].

While the pathophysiology of CHS is not fully understood and even counter-intuitive to the proposed use of cannabinoids as antiemetics, it is hypothesized that the transient receptor potential vanilloid-1 receptor (TRPV1) may play a role [7]. TRPV1 is a centrally and peripherally located nociceptor involved in both pain relief and body heat regulation [10,11]. TRPV1 has been found to be stimulated by both cannabinoids and heat such as hot baths and capsaicin [10-12]. A theorized mechanism of action is that TRPV1 causes symptoms of emesis with low-level stimulation, but anti-emetic effects during high stimulation due to receptor desensitization and depletion of substance P [13].

Multiple case reports and case series have demonstrated the potential benefits of capsaicin cream in managing CHS. Dezieck et al. had 13 patients who reported complete resolution of symptoms with capsaicin cream after failing other therapies [8]. Graham et al. reported two patients with improved symptoms and Lapoint et al. described another 5 patients with symptomatic relief [6,9]. It is important to note that between these reports, there is very little description given in regards to dosing. Strengths of the cream are not consistent, varying between 0.025% to 0.075%. Application amounts are vaguely stated as a thin layer, with only two reports stating a 1mm thick coating was applied [6]. The only consistent factor is location of application to the periumbilical region.

We consider our case a failure of capsaicin cream due to the need for multiple other agents before any significant relief of symptoms was achieved despite two applications of the cream within a narrow time period. Previous cases report fairly rapid improvement in symptoms, ranging from 30-45 minutes after application, which did not occur for our patient who has an established prior diagnosis of CHS and in whom no other cause of symptoms was found [6,14]. Our dosing and application technique were similar to the successful cases described by Graham, however our outcome was not [6]. Of note, the cream was utilized in our patient as a first line treatment.

While many published case reports of small numbers of patients suggest value in capsaicin cream for the management of CHS, it is important to understand that neither this therapy nor the mechanism of CHS has been fully studied or understood. When using capsaicin, other therapies should be readily available to relieve the symptoms if therapy is not successful to prevent delays in patient care as relief from capsaicin cream alone is not assured. Although utilization a topical agent may be a safer alternative to intravenous medication and the majority of the side effects of topical capsaicin are non-severe and localized to the application site, systemic side effects can occur especially if there is damage to the skin [15]. Stronger

studies are needed to fully assess the benefits of this medication in CHS.

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Authors Infomation: Vishal Patel*[1]; Elaine Smith[2]
[1]Department of Pharmacy, Community Medical Center, USA
[2]Department of Emergency Medicine, Community Medical Center, USA

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