Cannabinoid Hyperemesis Syndrome: Descriptive Overview of an Under-Recognized Diagnosis

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Cannabis is the most commonly used illegal drug worldwide. Its usage has short and long-term negative effects, including impairment of short-term memory and fluctuations in blood pressure [1,2]. With the growing spread of cannabis abuse, a novel clinical disorder has emerged, termed Cannabinoid Hyperemesis Syndrome (CHS), first described by Allen and colleagues in 2004 [3].

CHS is associated with heavy and chronic use of cannabis. It is characterized by recurrent attacks of severe nausea and vomiting and accompanying abdominal pain. Users compulsively take hot baths to ease the symptoms. Cessation of cannabis use will result in complete resolution of the symptoms [4,5]. We describe two patients in Israel who presented with symptoms suggesting CHS and discuss the clinical approach and management.

PATIENT DESCRIPTIONS

PATIENT 1
A 44 year old man presented to the emergency department (ED) with a chief complaint of abdominal pain and recurrent vomiting during the previous 5 days. At triage he was encoded as “drunk.” He was accompanied by his two sons who stated that their father has been a heavy marijuana user for at least 20–30 years. They also mentioned that in the previous few days he had been restless due to his complaints and took several pills of diazepam and clonazepam – with minimal relief of the symptoms. The only alleviation was achieved by taking repetitive hot baths for long periods. They also mentioned that in the past he had occasionally suffered similar complaints that had been treated with several medications and ceased only after he abstained from marijuana use for a few days. They also denied any febrile illness or alcohol consumption in the previous few weeks.

At admission the patient’s pulse was 43 beats/min, blood pressure 163/84 and oxygen saturation 100%. ECG showed sinus rhythm, with no ST or T wave deviations. Blood test results were all within normal limits.

In the ED the patient received a total of 2 L of IV fluids, metoclopramide and intramuscular thiamine, with minimal effect on his symptoms. After 9 hours in the emergency department he decided to leave the hospital and return home.

PATIENT 2
This was a 34 year old man who had developed post-traumatic stress disorder (PTSD) following his army service. He presented with undifferentiated abdominal pain for which he had been admitted repeatedly in the past and had undergone colonoscopies, computed tomography (CT) scans, endoscopy and even magnetic resonance imaging (MRI). No cause was found for the pain and vomiting and the patient was thought to be suffering from conversion disorder. When the physician came to examine the patient, he could not be found. A friend who accompanied him said he was taking a hot shower (in the ED). When questioned, he said that the patient took multiple hot showers a day, and that he smoked copious amounts of marijuana. He was counseled to stop but continued to present to the ED and was unable to cease his marijuana usage. He was then lost to follow-up.

COMMENT
Cannabis is a popular drug worldwide. Its popularity led to the recognition of an entity which has been associated with chronic cannabis abuse. Cannabinoid Hyperemesis Syndrome (CHS) includes cyclical events of hyperemesis among heavy cannabinoid abusers. A PubMed review revealed only a few dozen case reports on this syndrome, none of them from Israel.

The pathophysiology of CHS is not totally clear, yet it probably involves CB1 receptors, which are expressed mainly in the central nervous system and the intestine. It is believed that activation of CB1 receptors triggers marijuana’s effects, such as appetite stimulation and euphoria. The antiemetic effect of cannabinoids is well known, but has not been precisely characterized. Taking into account the paradoxical emetic effect of chronic cannabis usage and CHS suggests the involvement of the same cannabinoid receptors. Consistent chronic stimulation of CB1 receptors by Δ-9-tetrahydrocannabinol (THC) may lead to over-activation of these receptors and the presentation of CHS. THC activates CB2 receptors as well. CB2 receptors are expressed principally in immune cells. Their role is probably associated with pain reduction and inhibition of inflammation. CB2 receptors are believed not to be involved in the pathophysiology of CHS in chronic cannabinoid users [4,5].
The majority of patients with CHS are heavy users of cannabinoids, on a daily or almost daily basis for years. Moreover, several cases of CHS in adolescents with similar presentations and complaints have been published [3]. The hyperemetic events last from several hours to several days and might reoccur over several weeks or months. Other common complaints include abdominal pain and secondary fluid/electrolyte imbalances due to recurrent vomiting. The vomiting responds minimally to the common antiemetic treatments. The hallmark of those attacks is a compulsive need for a hot bath, which helps relieve the symptoms. There is no specific test to validate CHS, apart from the regular screening for cannabinoid usage combined with the collected clinical data.

The compulsive behavior of having a hot shower is seen repeatedly in patients presenting with CHS. This is a self-learned activity that alleviates the discomfort during CHS attacks. There is no single explanation for how these showers contribute to the relief of the vomiting. The main method to alleviate CHS is cessation of cannabis use. Haloperidol has also been suggested as an efficient antiemetic medication in CHS attack, with rapid response [4].

CONCLUSION
Underdiagnosis of CHS may result in recurrent visits to the hospital and the resultant unnecessary medical and surgical investigations. We believe that CHS is underdiagnosed by health care systems and hope this study will contribute to medical staff alertness to this clinical entity.

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References

Capsule

Branch-specific dendritic Ca2+ spikes cause persistent synaptic plasticity

The brain has an extraordinary capacity for memory storage, but how it stores new information without disrupting previously acquired memories remains unknown. Cichon and team show that different motor learning tasks induce dendritic Ca2+ spikes on different apical tuft branches of individual layer V pyramidal neurons in the mouse motor cortex. These task-related, branch-specific Ca2+spikes cause long-lasting potentiation of post-synaptic dendritic spines active at the time of spike generation. When somatostatin-expressing interneurons are inactivated, different motor tasks frequently induce Ca2+ spikes on the same branches. On those branches, spines potentiated during one task are depotentiated when they are active seconds before Ca2+ spikes induced by another task. Concomitantly, increased neuronal activity and performance improvement after learning one task are disrupted when another task is learned. These findings indicate that dendritic branch-specific generation of Ca2+ spikes is crucial for establishing long-lasting synaptic plasticity, thereby facilitating information storage associated with different learning experiences.

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Capsule

Complex archaea that bridge the gap between prokaryotes and eukaryotes

The origin of the eukaryotic cell remains one of the most contentious puzzles in modern biology. Recent studies have provided support for the emergence of the eukaryotic host cell from within the archaeal domain of life, but the identity and nature of the putative archaeal ancestor remains a subject of debate. Spang and colleagues describe the discovery of ‘Lokiarchaeota’, a novel candidate archaeal phylum, which forms a monophyletic group with eukaryotes in phylogenomic analyses, and whose genomes encode an expanded repertoire of eukaryotic signature proteins suggestive of sophisticated membrane remodeling capabilities. These results provide strong support for hypotheses in which the eukaryotic host evolved from a bona fide archaeon, and demonstrate that many components that underpin eukaryote-specific features were already present in that ancestor. This provided the host with a rich genomic ‘starter-kit’ to support the increase in the cellular and genomic complexity that is characteristic of eukaryotes.

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