Cannabinoid hyperemesis syndrome as an unusual cause of cyclic vomiting

Sir,

Cyclic vomiting syndrome is a disease of uncertain etiology seen more often in children. Several associations have been proposed including a link to migraine, sympathetic dysfunction, and in the last 10 years chronic cannabis use.[1,2]

Cannabis is a widely used recreational drug. Recent research has thrown light on the three major compounds of the exogenous cannabinoids found in cannabis and the two cannabinoid receptors in the body that they act on.[3] While cannabis in low doses has antiemetic effects, a distinct syndrome characterized by a triad of cyclic and recurrent vomiting refractive to standard therapy, abdominal pain, and frequent hot showers which help to relieve the symptoms is recognized in chronic cannabis users termed “cannabinoid hyperemesis syndrome (CHS).”[4,5]

A 21-year-old male presented with epigastric and retrosternal burning pain and nonbilious vomiting about 10 episodes per day for 3 days duration.

He had a past history of similar symptoms requiring multiple hospital admissions (5 in the last 1 year at different hospitals). He had lost 6 kg of weight in the last year despite a normal appetite. At the time of admission, his height was 167 cm, weight 63 kg with a body mass index of 22.6.

He was a smoker, smoking about 5–10 cigarettes per day and an occasional consumer of alcohol. He had no other chronic medical or psychiatric ailments such as anorexia or bulimia nervosa. He had been evaluated extensively outside, and other than an endoscopic evidence of reflux esophagitis and mild antral gastritis, all investigations including a computed tomography (CT) scan of the abdomen and brain were normal. He had been treated outside with proton pump inhibitors, antiemetics and had also received one course of anti-H. Pylori therapy for a duration of 14 days. While in hospital, he was started on pantoprazole and antiemetics with intravenous fluids. Baseline blood investigations were normal. Within 12 h of admission, he developed slight neck swelling and mild breathlessness after repeated retching and vomiting (nearly 25 episodes). There were clinical signs of subcutaneous emphysema. CT scan of the thorax showed evidence of small posterior wall tear of cervical esophagus with subcutaneous emphysema and pneumomediastinum. He was put on conservative therapy including nil per oral and supportive measures following which neck swelling resolved. While in the hospital, he was restless and agitated. On repetitive questioning, he admitted to smoking marijuana cigarettes (up to a maximum of 20 per day for nearly 2 years) which were consistent with cannabis dependence. A urine toxicology screen was subsequently asked for and was positive. He also admitted to taking frequent baths at home which helped relieve the symptoms. There was a definite temporal association of vomiting to the use of marijuana and following each hospitalization, when he temporarily stopped smoking, there was resolution of symptoms. Hence, a diagnosis of CHS was made. Once the acute issues resolved, a psychiatry opinion was sought, and he was put through a de-addiction program. He remained symptom-free for nearly 1 year but had another hospital admission after that due to restarting marijuana.

CHS was first described in Australia by Allen et al.[9] and since then is being increasingly recognized worldwide by emergency physicians and gastroenterologists.[11] The diagnostic criteria consist of essential criteria (long-term cannabis use), 5 major and 5 supportive criteria.[3] To the preexisting triad, weekly use of marijuana and resolution of symptoms with cannabis cessation have been added.[3] All the above were satisfied by our patient. Cannabis contains many substances chief among them being tetrahydrocannabinol (THC), cannabidiol (CBD), and cannabigerol (CBG).[3,6] There are also two distinct
cannabinoid receptors CB1 and CB2.\[3\] CB1 is located in several parts of the body including the cerebral cortex, hypothalamus, and basal ganglia. In the gut, they are located on the enteric nervous system. CB2 is linked to immune system.

THC has a psychotropic effect and is responsible for the addictive effect of cannabis.\[3\] It activates CB1 receptor in the dorsal vagal complex of brainstem. THC is stored in fat and gets released during lipolysis and stress and may play a role in CHS.

Activation of the CB1 receptors in the gut decreases acid secretion, lowers the lower esophageal sphincter tone, and reduces intestinal motility.

CBD enhances the expression of CB1 receptors in the hypothalamus and amplifies the hypothermic effect of THC. It causes antiemetic effect in low doses and emetic effect at high doses.\[6\] CBG is nonpsychotropic and antagonises the antiemetic effect of low-dose CBD.\[6\]

Together, the impaired gastric motility, lower sphincter tone, emesis effect of high-dose CBD, enhanced hypothermic effect of THC, which is relieved by hot water bath, all probably contribute to the clinical features of CHS.\[7\]

Goal of therapy is twofold—relief of hyperemesis and cannabis cessation. Intravenous fluid replacement and antiemetics with proton pump inhibitors are used.

Long-term cessation of cannabis is the only way to prevent relapse. Rehabilitation program, cognitive behavioral therapy, and motivational therapy help patients to abstain from use.

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