Pneumomediastinum in a Patient with Cannabinoid Hyperemesis Syndrome

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**CASE PRESENTATION**

A 23-year-old man with a past medical history of cannabinoid hyperemesis syndrome presented to the emergency department with 1 week of nausea, emesis and poor oral intake. Prior to presentation, the patient had been treated in the emergency department several times for intractable vomiting. The patient reported he was a daily long-term user of marijuana cigarettes.

On presentation, the patient was afebrile with a pulse of 117 beats per minute, respiratory rate of 20 per minute, blood pressure of 111/79 and oxygen saturation of 99% on room air. Physical examination revealed a thin man with eructation and subcutaneous crepitation over the neck and thorax. Lung sounds were clear to auscultation bilaterally. Laboratory testing revealed a pH of 7.26, anion gap of 33, blood-urea nitrogen of 107 mg/dL and a newly elevated creatinine of 13.01 mg/dL. Notably, the patient had normal labs with a creatinine of 0.84 mg/dL during a similar presentation for intractable vomiting one month prior to presentation. Chest X-ray showed evidence of subcutaneous gas and pneumomediastinum. Computed tomography (CT) of the chest and abdomen with intravenous contrast revealed pneumomediastinum and pneumoretroperitoneum with extension into the spinal canal ([Figure 1](#)). Repeat CT imaging of the chest with oral contrast was performed and did not show extraluminal oral contrast extravasation into the mediastinum. The patient was evaluated by cardiothoracic surgery, who recommended conservative management with close monitoring of symptoms. Urinalysis demonstrated muddy brown casts and renal tubular epithelial cells, suggesting pre-renal azotemia secondary to volume depletion. Treatment with 3 days of intravenous fluids and anti-emetics resulted in normalization of the patient’s creatinine ([Figure 2](#)). The patient was subsequently discharged without further complication.

Two months after discharge, the patient presented with a subsequent episode of intractable vomiting from cannabinoid hyperemesis syndrome. CT of the chest with intravenous contrast revealed complete resolution of the patient’s prior findings of pneumomediastinum and pneumoretroperitoneum.

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**Figure 1.** (A) Red arrows illustrating extensive pneumomediastinum and pneumoretroperitoneum; (B) illustrating air extending into the neck and spinal canal.
DISCUSSION

Definitive diagnosis of cannabinoid hyperemesis syndrome (CHS) has proven to be challenging, as there is no standardized diagnostic criteria. This is likely due to the elusive nature of the syndrome’s pathophysiology. It has been hypothesized to involve a complex interaction between the endogenous CB$_1$ and CB$_2$ cannabinoid receptors (with CB$_1$ responsible for the majority of deleterious clinical effects) and tetrahydrocannabinol (THC) in marijuana.\textsuperscript{1,2} In an effort to increase the sensitivity for diagnosis, a recent systematic review evaluated several case reports to determine which symptoms were most frequently encountered in patients with CHS. The symptoms with the highest sensitivity included severe nausea and vomiting in a cyclical pattern over several months, a minimum of weekly cannabis use for longer than 1 year, symptom relief with hot baths or showers and resolution of symptoms after cessation of cannabis use.\textsuperscript{3} There is a male predominance and episodes usually last 1–2 days, but can extend up to 10 days.\textsuperscript{4} While the symptoms of CHS may appear benign, intractable vomiting from CHS can result in severe complications including acute renal failure, esophageal perforation, severe electrolyte derangement, pneumomediastinum, and death.\textsuperscript{5,6} While our patient did not end up having esophageal perforation, his acute renal failure, electrolyte derangements and pneumomediastinum demonstrate the potential life-threatening complications of CHS. This case illustrates the importance of monitoring for these complications in patients who present with intractable vomiting as a manifestation of CHS.

References


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