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The Case | The smoker and the nephrologist

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Table 1 | Serum studies on patient at presentation

Sodium (135–145 mmol/l)	133
Potassium (3.5–5.5 mmol/l)	2.9
Chloride (97–105 mmol/l)	70
Bicarbonates (23–30 mmol/l)	40
Creatinine (40–120 μmol/l)	398
Blood urea nitrogen (3–7 mmol/l)	24
Protein (65–75 g/l)	90
Phosphorus (0.8–1.5 mmol/l)	1.3
LDH (125–250 UI)	567
CPK (10–180 UI)	228
CRP (<10 mg/l)	3
Uric acid (<240 μmol/l)	560
pH (7.38–7.42)	7.69
CO_2 (40–45 mm Hg)	33
PO_2 (90–100 mmHg)	100
Bicarbonates (25–28)	40
Hemoglobin (12.5–17 g/l)	17.1
Lactacidemia (<2.5 mmol/l)	2.4

Abbreviations: CPK, creatinine phosphokinase; CRP, C-reactive protein; LDH, lactate dehydrogenase.

A 28-year-old man, a native of Morocco, was hospitalized for acute renal failure associated with persistent vomiting. The patient first presented at age 16 with cyclical episodes of abdominal pain with vomiting, relieved transiently by compulsive hot baths. He had continuously smoked 15 cigarettes and 5 cannabis 'joints' a day since the age of 14 years. He denied consumption of medication and other illicit drugs or alcohol.

Table 1 shows serum studies during one of numerous presentations. His liver function tests, amylase, and lipase were normal. He was hospitalized five times during 2009–2010 with acute renal failure (Table 1). Urine sediment was normal. Urinary (U) sodium was 53 mmol/l, U potassium 66 mmol/l, U chloride <20 mmol/l, U urea 500 mmol/l, U protein <0.1 g/l, and U creatinine 12 mmol/l. Myoglobinuria was negative. Clinical examination was suggestive of hypovolemia (tachycardia with orthostatic hypotension). Colonoscopy and endoscopy of the upper digestive tract were normal. He was treated with intravenous fluids and antiemetic with spontaneous improvement of symptoms, and the creatinine level dropped to a normal value (75 μmol/l).

What is your diagnosis?

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | Cannabinoid hyperemesis syndrome

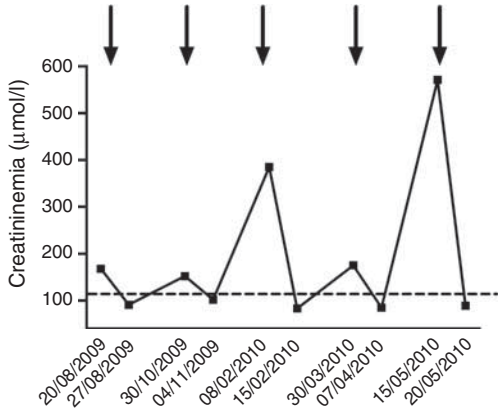

Figure 1 | Description of recurrent episodes of acute renal failure during a 1-year period. Arrows indicate each episode of vomiting associated with compulsive hot-bath behavior. Every episode resolves with restoration of extracellular volume.

We made the diagnosis of cannabinoid hyperemesis syndrome associated with acute renal failure following intractable vomiting. Serum and urinary electrolytes status show metabolic alkalosis, hemoconcentration, and extracellular dehydration (Figure 1).

The patient was told that he had to quit smoking cannabis, and we prescribed benzodiazepines for 1 week. After discharge, he was subsequently hospitalized five additional times, each time with reversible functional renal failure shown in the figure. He failed to stop his marijuana use despite several hospitalizations.

Cannabis is a highly consumed drug. Global annual cannabis use prevalence is estimated between 2.9 and 4.3% of the population aged 15–64 years. It is the third most commonly used drug after tobacco and alcohol. Traditionally, cannabinoids have been utilized for recreational purposes or therapeutically as antiemetics; however, recent reports suggest that chronic or 'excessive' use can result in a toxic effect causing cyclic vomiting, which has been termed 'cannabinoid hyperemesis syndrome'.[1–3]

The syndrome is characterized by a recurrent initial stage with nausea and abdominal discomfort. Next, intractable vomiting with thirst and polydypsia occurs. Sontineni and colleagues suggest the following diagnostic criteria: history of regular cannabis use for years, severe nausea and vomiting, vomiting that recurs in a cyclic pattern over months, and resolution of symptoms after stopping cannabis use, supportive features are compulsive hot baths with symptom relief (which ceases as soon as the sufferer stops bathing with a total duration of bathing averages 5 ± 5.1 h per day), colicky abdominal pain, and no evidence of gall bladder or pancreatic inflammation.[1,4]

Some patients lose more than 5 kg of weight. There is no fever, and patients have no other demonstrable etiology for vomiting. Abdominal ultrasounds as abdominal and cerebral scan were always unremarkable. All patients obtain resolution of their symptoms by cannabis cessation, while those who do not quit continue to have this syndrome.[5]

It seems important to inform nephrologists about this syndrome because of its frequency and the considerable associated health-care costs. The syndrome is stereotyped and can be easily recognized.

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