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Bitter Bottle Gourd Toxicity

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Abstract

Bottle gourd (*Lagenaria siceraria*), colloquially known as "lauki" or "doodhi" in India, is an edible plant in the Cucurbitaceae family. Consumption of raw bottle gourd juice is common practice in India. However, little known is the fact that ingestion of raw bottle gourd juice that is extremely bitter tasting is associated with cucurbitacin toxicity. Cucurbitacin is a pheromone produced by the plant as a defense mechanism against insects and herbivores. It is responsible for imparting the bitter taste, and the associated toxicity. The exact mechanism of cucurbitacin toxicity is not well understood, however it is suspected to result in gastric erosions and increased capillary permeability, resulting in rapid onset of vomiting, diarrhea, gastrointestinal bleeding, hypotension and shock. As serum and urine concentration measurements of cucurbitacin are not readily available, bottle gourd toxicity remains a clinical diagnosis based largely on the temporal association of ingestion of unusually bitter tasting vegetable or its juice with rapid onset of symptoms. Physician awareness is therefore of paramount importance in the diagnosis of this unusual entity. We present the case of a 78-year-old woman, with no comorbid conditions, who presented to the Emergency Department (ED) with three episodes of vomiting and multiple episodes of watery loose stools soon after ingestion of extremely bitter tasting, fresh bottle gourd juice. A prompt diagnosis of cucurbitacin toxicity was made in the ED and fluid resuscitation was commenced expeditiously. However, it was noted that most physicians were unfamiliar with this entity, and therefore skeptical of the diagnosis. Our rationale in presenting this clinical case report is three-fold: 1) To create physician awareness regarding this unique clinical entity, especially among emergency physicians, as they are the first point of contact for a critically ill patient. 2) To highlight the rapid onset and progression of symptoms in cucurbitacin toxicity. 3) To emphasize aggressive fluid resuscitation and symptomatic management as the mainstay of clinical treatment.

Keywords

Bottle Gourd, Cucurbitacin, Tetracyclic Triterpenoid Compounds

1. Introduction

Bottle gourd (Lagenaria siceraria) is native to tropical Africa, but is cultivated globally in areas with warm climates. It is an edible plant in the Cucurbitaceae family [1].

Other members of the gourd family include watermelon, cucumber, gherkin, squash, pumpkin, and zucchini [2].

Cucurbitacins are tetracyclic terpenes with steroidal structures that are isolated from parts of the plants of the Cucurbitaceae family. It is a pheromone which is produced as a defense mechanism of the plant against insects and herbivores, and is responsible for imparting the bitter taste, and the associated toxicity [3] [4].

The highest concentration of cucurbitacin is found in the roots of the plant, however, it is also found in the seeds, leaves and fruit of the plant.

Patients who present with bottle gourd toxicity will unequivocally give a history of onset of symptoms soon after consumption of unusually bitter tasting fruit or raw juice [5].

On presentation, these patients usually complain of severe abdominal pain, vomiting and diarrhea. Gastrointestinal bleeding is also often seen on presentation. Hypotension followed by renal and hepatic failure is seen in 50% - 75% of patients [6] [7].

Symptoms have a rapid onset and progression, typically starting within 30 minutes and may continue up to 2 to 7 days [8].

We present the case of an elderly lady who, soon after consuming bitter raw bottle gourd juice, developed severe abdominal cramps and bloody diarrhea, with vomiting. Shock followed by syncope soon ensued. We detail the clinical presentation of this lesser-known toxin, and its successful resuscitation at our institute.

2. Case Summary

2.1. Clinical Presentation

A 78-year-old woman presented to the Emergency Department (ED) with colicky abdominal pain, multiple episodes of loose motions (watery, moderate quantity, no mucus in stool, last 2 episodes were blackish, as per the patient) and 3 episodes of vomiting, associated with profuse diaphoresis and an episode of syncope. History elicited was that the symptoms started soon after consumption of one-third of a glass of extremely bitter tasting fresh, raw bottle gourd juice.

On arrival, the woman was profusely diaphoretic with cold, clammy extremities, systolic blood pressure of 70 mm Hg, heart rate of 128 beats per minute (si-

nus tachycardia), respiratory rate of 30 breaths per minute, GCS—14 (E3V5M6), random blood sugar 180 mg/dl. Screening of the inferior vena cava (IVC) revealed a completely collapsed IVC. Urinary bladder screening showed that it was empty.

2.2. Point-of-Care Investigations

Blood gas on arrival showed pH—7.32, pCO_2—26.9, bicarbonate—15.4, base excess—negative 9.3 mmol/L, lactate—4.24 mmol/L, anion gap 14.

Blood gas after 24 hours showed pH—7.34, pCO_2—39.9, bicarbonate—21.8, base excess— negative 3.8, lactate—1.91, anion gap 8.

2.3. Differential

Enterohemorrhagic enterocolitis with septic shock.

2.4. Treatment and Outcome

Rapid volume resuscitation was initiated with 0.9% saline, of which a 3000 ml bolus was given. The patient was fluid-responsive, with the heart rate decreasing to 108 beats per minute and the systemic systolic pressure coming up to 96 mm Hg. Intravenous fluid was continued at 100 ml/hour, and a pantoprazole infusion was initiated at 8 mg/hour. Broad-spectrum empirical antibiotic was initiated in the ED (as septic shock was also a consideration).

Three hours after commencing fluid resuscitation, urine output was 60 ml/hour. The tachycardia resolved, and the blood pressure stabilized. The patient was shifted to the Intensive Care Unit (ICU) for continuum of care. Preliminary investigations: hemoglobin 11.1 g/dl, white cell count 12,300, platelet count 210,000/ml, creatinine 0.72 mg/dL, BUN 16 mg/dL, sodium 120 mmol/L, potassium 3.9 mmol/L, chloride 96 mmol/L, calcium 5.3 mmol/L, phosphorus 4.5 mmol/L, albumin 2.1.

Serum amylase and lipase were within normal range (61 and 75 respectively).

The abdominal ultrasound showed fatty liver with a distended gall bladder. Serum high-sensitive troponin was 75.4. Repeat troponin after 12 hours was 115.7, which then showed a decreasing trend. The elevated troponin was attributed to myocardial stress. 2-dimensional echocardiogram showed no regional wall motion abnormality, left ventricle ejection fraction of 50%. Supportive management was continued. The patient had a favourable outcome and was discharged on Day-6.

3. Discussion

The exact mechanism of cucurbitacin toxicity is poorly understood, however it is suspected to be caustic to the gastric mucosa leading to vomiting, diarrhea, gastric erosions and gastrointestinal bleeding. Cucurbitacins have also been shown to have cytotoxic effects with increased capillary permeability, leading to rapid onset of hypotension and shock [4].

The range of toxicity of cucurbitacin based on few *in-vivo* toxicity reports has been found to vary between 2 - 12.5 mg per kg body weight.

About 50 - 300 ml of juice can cause symptoms, and the larger the quantity of the juice or raw fruit ingested, the more severe the symptoms.

Note that fruits containing a higher level of cucurbitacin will show no difference in appearance, colour, odour, or texture; but will have a significantly bitter taste. In our report, it was important to elicit the history not only of the consumption of bottle gourd juice, but the fact that it was bitter; possibly indicating involvement of the outer peel of the fruit, since bottle gourd juice is tasteless.

There is no antidote for bottle gourd toxicity, and treatment is mainly supportive management, which includes aggressive fluid resuscitation, vasopressor support as needed, proton pump inhibitors in case of gastrointestinal bleeding, and dialysis in case of acute renal injury.

As serum and urine concentrations of cucurbitacin are not readily available, bottle gourd toxicity remains largely a clinical diagnosis. Community sensitization and physician awareness regarding this clinical entity is therefore imperative to aid in prevention as well as prompt diagnosis and timely interventions.

Some of the earliest published work on cucurbitacin poisonings in humans was in 1983 by Ferguson *et al.* who reported a number of cases of human poisoning over the period of a year from the consumption of commercially produced zucchini in Australia [8].

Ho *et al.* have reported 5 adult cases of bottle gourd toxicity in 2014, which at the time, was the first reported group of patients with toxicity due to ingestion of bottle gourd in the United States. All patients presented with nausea, vomiting, and diarrhea within 5 to 25 minutes of ingestion, with one patient developing hematemesis and hypotension [4].

In India, Puri *et al.* have reported 15 cases of gastrointestinal toxicity due to bitter bottle gourd. Verma and Jaiswal also reported a case of bottle gourd juice poisoning in 2015 [7] [9].

The incidence of bottle gourd toxicity reporting is gradually on the rise in India, though still uncommon in Europe. Toxic squash syndrome is not unheard of worldwide, with Le Roux *et al.* reporting a retrospective series of 353 patients from French Poison Control Centers [10], however, to the best of our knowledge, bottle gourd toxicity itself, is very rare in the United States and in Europe, therefore making our report relevant to clinicians dealing in global acute care and toxicology centers, with an emphasis on the varied causes of cucurbit poisoning, bitter bottle gourd being one of them.

Our patient had a positive outcome in the hospital, and improved with fluid resuscitation and symptomatic therapy. Fortunately, there was no overt gastrointestinal bleeding.

4. Conclusions

Cucurbitacin toxicity is a rapidly progressive toxidrome. Primary pathophysiol-

ogy appears to be vasodilation leading to distributive shock. Since laboratory investigations are unavailable to confirm the diagnosis, it remains a clinical diagnosis, relying mainly on the history.

There is no available antidote for bottle gourd toxicity, and treatment is mainly supportive. Rapid, aggressive fluid resuscitation is the mainstay of therapy.

Emergency physicians need to be well-versed with cucurbitacin toxicity in order to establish an early clinical diagnosis, and initiate treatment accordingly. Confusion over the diagnosis may lead to indecision while commencing treatment, and valuable time may be lost if spent on imaging, which has no role in diagnosis/treatment and outcome.

Consent

The authors of this article attest to the fact that patient identification has been kept entirely confidential and with complete anonymity. In the absence of informed consent, the primary author (also identified as the head of department) takes full responsibility to maintain confidentiality and autonomy of the patient described in this clinical case report.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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