

Selected Topics: Toxicology

BITTER BOTTLE GOURD (*LAGENARIA SICERARIA*) TOXICITY

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Abstract—Background: Bottle gourd (*Lagenaria siceraria*) is an edible plant in the *Cucurbitaceae* family. When extremely bitter, ingestion of bottle gourd can cause rapid onset diarrhea, vomiting, gastrointestinal bleeding, and hypotension due to release of a substance named cucurbitacin. **Objective:** Our aim was to increase physician awareness of cucurbitacin poisoning in order to facilitate accurate diagnosis and appropriate management. **Case Report:** Five adult patients presented with nausea, vomiting, and diarrhea within 5 to 25 min of ingesting cooked bitter bottle gourd. One patient developed severe diarrhea, hematemesis, and hypotension requiring hospitalization. All patients improved within a few days with intravenous fluids and proton pump inhibitors. To our knowledge, this is the first reported group of patients with toxicity due to ingestion of bottle gourd in the United States (US). **Conclusions:** Physicians should be suspicious of cucurbitacin toxicity in patients who present with symptoms within minutes of ingestion of a plant in the *Cucurbitaceae* family. Patients should be asked if the plant tasted unusually bitter. The most common symptoms include diarrhea and hematemesis. More than half of patients develop hypotension. There is no known antidote for bottle gourd poisoning; treatment is supportive. Proton pump inhibitors should be given to patients with gastrointestinal mucosal injury. © 2014 Elsevier Inc.

Keywords—*Cucurbitaceae*; cucurbitacin; hematemesis; food-borne illness; food poisoning

INTRODUCTION

Bottle gourd (*Lagenaria siceraria*) is a plant widely grown and ingested in India and Sri Lanka. Practitioners of *Ayurveda*, or “life-knowledge” in Sanskrit, believe that drinking fresh bottle gourd juice, called *lauki* in Hindi, can treat hyperlipidemia, liver disease, depression, hair loss, and constipation. Indeed, studies of animal models suggest that bottle gourd may have a potential role in the treatment of cancer, depression, toxin-induced liver injury, and dyslipidemia (1–4).

In the United States (US), bottle gourds are primarily sold to crafting enthusiasts who use the gourds to make bowls, birdhouses, containers, and decorative figurines. With the increasing practice of complementary and alternative medicine, many Americans are treating their ailments with plant and herb preparations (5). Although many of these preparations have potential therapeutic properties, controversy surrounds *Ayurveda* because of adulteration of preparations, lack of standardized dosing, and improper identification of plants.

Our case report represents the first known cases of toxicity due to ingestion of bitter bottle gourd in the US.

CASE REPORT

A family of five adults presented to the emergency department (ED) within 30 min of eating cooked bottle

gourd. The family, first-generation Chinese-American, consisted of a 64-year-old woman, her 64-year-old husband, 74-year-old brother-in-law, 72-year-old sister, and 52-year-old sister-in-law. During dinner, they ingested a dish containing cooked bottle gourd. The bottle gourd was grown in their backyard from seeds purchased at a craft store (Figure 1). Everyone noted the extremely bitter taste of the bottle gourd after one bite of the dish. All family members were healthy and denied history of previous gastrointestinal illness, anticoagulant, or nonsteroidal anti-inflammatory drug use.

Within 3 to 25 min of consuming the bottle gourd, all five adults developed a sensation of gastric fullness and nausea, followed by sudden onset abdominal cramping, fecal urgency, tenesmus, and diarrhea. Diarrhea was described as watery for three patients. Two patients developed bloody diarrhea. Two patients developed vomiting that progressed to hematemesis within several hours. All five patients were managed supportively with intravenous crystalloids. After 3 h, they were discharged home with ondansetron as needed for nausea and vomiting and instructions to continue oral rehydration.

One day after eating the bottle gourd, all patients continued to experience anorexia and frequent watery diarrhea, more than 30 episodes each. They returned to

the ED for worsening nausea and dehydration. Clinical features are shown in Table 1. Patient 1, 2, 4, and 5 were treated in the emergency department. Over 6 h, they were each given 2–3 L of intravenous 0.9% sodium chloride solution with improvement of symptoms and the ability to tolerate a liquid diet.

Patient 3 ate the largest quantity of bitter bottle gourd. Interestingly, despite noting the bitter taste of the gourd, he reported eating several bites because he did not want to insult his sister-in-law, who had prepared the meal. He was hospitalized for severe dehydration, hypotension, and persistent tachycardia that continued despite fluid resuscitation with 3 L 0.9% NaCl solution. In our cases, the severity of illness was dose dependent and correlated with the amount of cucurbitacin ingested.

On arrival to the hospital ward, Patient 3's vital signs were as follows: temperature 36.9°C (98.4°F), pulse 121 beats/min, blood pressure 93/66 mm Hg, respirations 20 breaths/min, and oxygen saturation 98%. He appeared awake and oriented but fatigued and had several episodes of vomiting and diarrhea during the initial examination. He had mild generalized edema, most prominent in his face and extremities. Abdomen was mildly tender on palpation.

Laboratory values were as follows: white blood cells 13,100/mm³, hemoglobin 14.8 g/dL, platelets 164,000/mm³, sodium 142 mEq/L, potassium 3.4 mEq/L, chloride 114 mEq/L, bicarbonate 19 mEq/L, creatinine 0.7 mg/dL, aspartate aminotransferase (AST) 24 U/L, alanine aminotransferase (ALT) 20 U/L, and albumin 3 g/dL. During his hospital stay, he received a total of 6 L intravenous crystalloids and 60 mEq potassium supplementation. Vomiting subsided by the second day of illness but diarrhea continued for 5 days. He was discharged after 2 days in the hospital. He was seen by a gastroenterologist and was advised to complete a 2-week course of omeprazole.

DISCUSSION

Bottle gourds are members of the Cucurbitaceae family of plants. Other plants in the family include cucumber, zucchini, eggplant, squash, pumpkin, and bitter melon. These plants produce cucurbitacin, a pheromone that protects the plants from insects and herbivores (6). The bitter taste of the substance prevents poisoning in humans. When bottle gourds are grown under environmental stress, such as extreme temperatures and poor soil quality, they produce higher levels of cucurbitacins. Plants that are over-ripened also produce more cucurbitacins (7). Compared with other Cucurbitaceae, bottle gourds have unusually elevated levels of cucurbitacins, in particular, types B, D, G, and H.

Cucurbitacins are triterpenoid substances; they are hydrophobic derivatives of triterpenes (C₃₀H₄₈), which are



Figure 1. Bottle gourd, “speckled swan” variety, ingested by our patients.

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