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Case Report

Bottle Gourd – A Poison in Disguise

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Abstract

Background: Bottle gourd (*Lagenaria siceraria*) is widely promoted in traditional Indian medicine for its purported health benefits, including use in managing diabetes, hypertension, liver disorders, and gastrointestinal ailments. However, under certain conditions, the juice can turn bitter due to elevated concentrations of cytotoxic compounds known as cucurbitacins. These compounds are highly toxic to humans and can cause severe gastrointestinal and systemic symptoms.

Case Presentation: We report the case of a 45-year-old male who routinely consumed bottle gourd juice. On the day of presentation, he consumed an unusually bitter juice. Within 15 minutes, he developed severe vomiting, epigastric discomfort, headache, and profuse sweating. He arrived at the Emergency Department hemodynamically stable but soon developed altered sensorium, hypotension, and tachycardia. ECG changes (S1Q3T3 pattern) and elevated D-dimer raised concerns for pulmonary embolism, and a CTPA was planned. However, the scan was aborted due to sudden hematemesis. Initial labs showed leukocytosis and deranged liver function. Endoscopy revealed diffuse gastric erythema, fluid accumulation, and a suspicious ulcer with a clot on the lesser curvature. The patient was managed conservatively with IV fluids, inotropes, PPIs, antiemetics, and antibiotics. He improved over the next few days and was discharged in a stable condition.

Conclusion: This case underscores the potential lethality of consuming bitter bottle gourd juice. Physicians should suspect cucurbitacin poisoning in patients presenting with acute GI symptoms, hypotension, and recent consumption of Cucurbitaceae family plants. There is no antidote; early recognition and supportive care remain the mainstay of treatment. Public awareness is crucial to prevent further morbidity and mortality.

Keywords: *Bitter bottle gourd, Gastrointestinal toxicity, Hematemesis, Traditional medicine complications, Endoscopy findings, Public health awareness.*

Introduction

Bottle gourd (*Lagenaria siceraria*), a Cucurbitaceae family vegetable, is widely consumed in India and other tropical regions. Its juice is promoted in traditional medicine for managing diabetes, hypertension, and gastrointestinal disorders due to its nutrient content and low caloric value [1,2]. However, under conditions like environmental stress or genetic variations, bottle gourd can accumulate cucurbitacins, cytotoxic tetracyclic triterpenoids causing a bitter taste [3,4]. These compounds, notably types B, D, G, and H, are potent gastrointestinal irritants and systemic toxins, leading to symptoms like vomiting, hematemesis, hypotension, and potentially death [5,6]. Despite its preventable nature, cucurbitacin poisoning is underrecognized, often mimicking gastrointestinal sepsis or foodborne illness [3,7]. Limited awareness among the public and healthcare providers, combined with the cultural use of bottle gourd, contributes to delayed diagnosis [8,9]. This case report highlights the clinical features and management of cucurbitacin toxicity to enhance awareness and prevent morbidity.

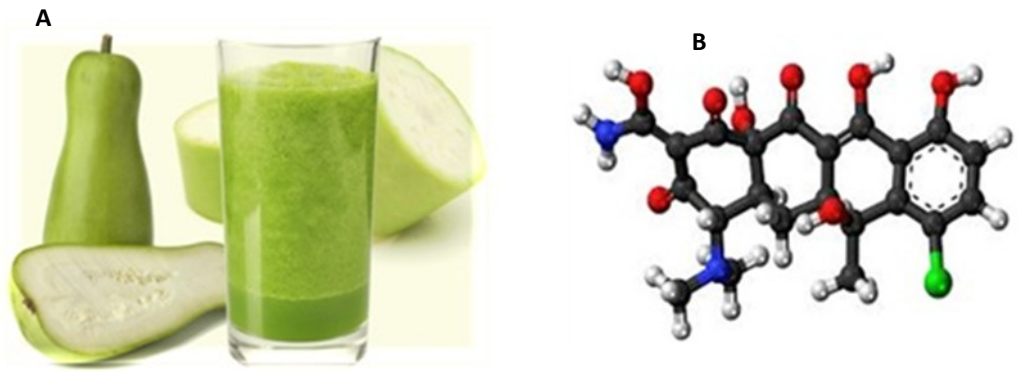

Figure 1. A. *Bottle gourd (Lagenaria siceraria) commonly used in Indian cuisine.*
B. *Tetracyclic Triterpenoid Cucurbitacin's*

Case Presentation

A 45-year-old male and his wife habitually consumed bottle gourd juice every morning. One morning, however, they noticed the juice was unusually bitter. His wife did not consume it further due to the bitter taste, but he finished the glass. Within 15 minutes of consumption, he experienced multiple episodes of vomiting, profuse sweating, mild epigastric discomfort, and a headache.

On arrival in the Emergency Department, the patient was hemodynamically stable, but within 15 minutes, he suddenly became restless with altered sensorium. He became hypotensive, tachycardic, with cold and clammy extremities and feeble peripheral pulses. Systemic examination was unremarkable except for mild epigastric tenderness on abdominal examination. The patient was started on IV crystalloids and inotropes and shifted for a CTPA due to S1Q3T3 changes on ECG and raised D-dimer. In the CT gantry, the patient had an episode of large hematemesis, and the CT was abandoned.

Initial blood work showed leukocytosis and deranged LFTs, with renal and coagulation profiles within normal limits. Upper GI endoscopy revealed diffuse erythema in the stomach with fluid collection in the fundus and a suspicious ulcer with a clot on the lesser curvature. The patient was managed with intravenous fluids, inotropic support, antibiotics, antiemetics, and proton pump inhibitors, showing steady improvement over 2 days. Repeat endoscopy on day 4 showed reduced erythematous changes compared to the initial findings. The patient recovered well and was discharged.

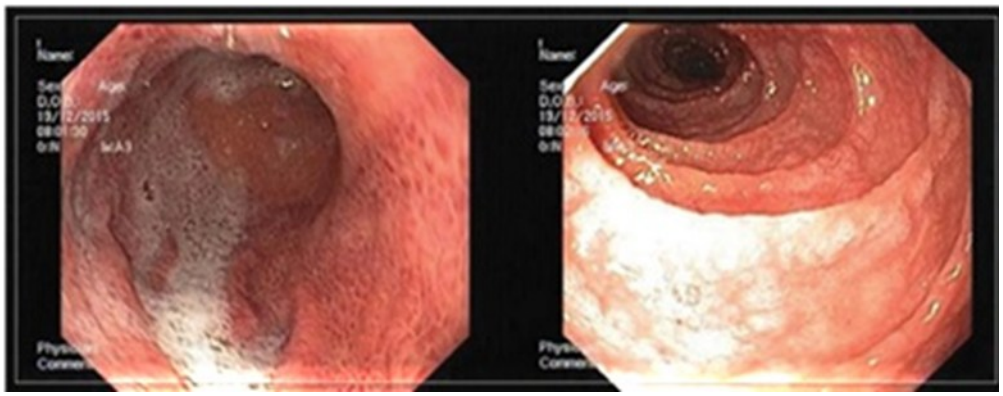

Figure 2. *Upper gastrointestinal endoscopy showing patchy erythema in fundus, body and antrum of stomach and first and second part of duodenum.*

Discussion

Cucurbitacins, tetracyclic triterpenoids causing bitterness in bottle gourd, are highly toxic, particularly types B, D, G, and H [4,5]. These compounds, studied for anti-tumorigenic effects against cancers by inducing apoptosis [10], cause severe toxicity in acute exposure. Cucurbitacin B inhibits STAT3 signaling, while cucurbitacin D increases capillary permeability, leading to hypotension and capillary leak syndrome, as seen in our patient's rapid deterioration [6,10]. Both inhibit cortisol binding to glucocorticoid receptors, exacerbating hypotension [5]. Gastrointestinal damage results from direct toxicity and hypoperfusion, with endoscopic findings like gastric erythema and ulcerations consistent with prior reports [3,7,11].

The presentation mimics sepsis, foodborne illness (*Staphylococcus aureus*, *Bacillus cereus*), or gastrointestinal bleeding [3]. The rapid symptom onset and bitter juice history strongly suggest cucurbitacin poisoning. The S1Q3T3 ECG pattern and elevated D-dimer, initially concerning pulmonary embolism, likely reflected systemic toxicity [11]. Supportive management with IV fluids, inotropes, proton pump inhibitors, antiemetics, and antibiotics stabilized the patient, aligning with the self-limiting nature of the condition [6]. Delayed recognition can be fatal [7]. Physicians should suspect cucurbitacin poisoning in patients with acute symptoms post-Cucurbitaceae ingestion, inquiring about bitter taste [8,9].

Conclusion

Toxicity from bottle gourd, a commonly consumed vegetable, may be more prevalent than reported. It presents with abdominal discomfort, hematemesis, hypotension, and potential organ dysfunction, with symptoms appearing within approximately 30 minutes of consumption. The clinical picture can mimic gastrointestinal sepsis or other acute conditions, complicating diagnosis. No antidote exists, and supportive management is the cornerstone of treatment, with most patients recovering within one week. Physicians should suspect cucurbitacin poisoning in patients with rapid-onset symptoms following ingestion of Cucurbitaceae family plants, particularly if a bitter taste is reported. Public awareness campaigns highlighting the dangers of consuming bitter bottle gourd are essential to prevent further cases.

Conflicts of Interest

The authors declare no conflict of interest.

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