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Case Report

Internal Medicine Section

Bottle Gourd (*Lagenaria Siceraria*) Toxicity: A "Bitter" Diagnostic Dilemma

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ABSTRACT

Consumption of a glass of bottle gourd juice is thought to work as a health "tonic" and part of traditional healthy living practices in India. The juice may in certain circumstances turn bitter with increased levels of the cytotoxic compound called Cucurbitacins. If the bitter juice is consumed it causes a toxic reaction in the gut, leading to abdominal discomfort/pain, vomiting, hematemesis, and hypotension which may be rarely fatal, especially in persons with pre-existing illness. In the absence of clear cut history regarding the consumption of the bitter bottle gourd juice and the initiation of symptoms, the differential diagnosis for the above symptoms will include diseases causing gastrointestinal bleed with hypotension and/or shock.

We report a case of bitter bottle gourd poisoning presenting with abdominal symptoms, hematemesis and shock and with an initial differential diagnosis of septicemia with septic shock and multi-organ involvement. We conduct a literature review and ponder the various differential diagnoses of this clinical scenario.

Keywords: Bottle gourd, Cucurbitacins, Food-borne illness

CASE REPORT

A 55-year-old gentleman was referred to our hospital in a state of shock. He was admitted to a nursing home, a day prior, with history of sudden onset of profuse vomiting, bloody diarrhea and altered sensorium and developed hypotension and oliguria, after a couple of hours. He was treated with intravenous fluids, antibiotics and vasopressors. He did not show much improvement over the day & hence was referred to our hospital.

On evaluation of the symptoms, in detail, he was asymptomatic until 7 am, at which time he consumed a glass of bottle gourd juice as was his daily routine. He noticed the juice to be unusually bitter, and developed symptoms after 30 minutes. He had past history of Diabetes and Ischemic heart disease. He was on oral hypoglycemic drugs, antiplatelets and statins.

On examination, the patient was conscious, confused, with cold and clammy extremities. The peripheral pulse was feeble with tachycardia (rate 130 beats/ minute). Blood pressure was 70/40 mm of Hg, respiratory rate was 32 breaths/ minute with oxygen saturation on room air was 97%. There was no edema, cyanosis, clubbing or rash. Ryle's tube was inserted and gastrointestinal (GI) aspirate was coffee brown in colour. He had oliguria (urine output over last 24 h < 500 cc).

Systemic examination revealed diffuse abdominal tenderness with no organomegaly, a S_3 gallop with clear chest. The investigations on admission [Table/Fig-1] showed impaired renal & liver function, leukocytosis and hyperkalemia. Screening for tropical diseases was negative. Urine examination and Ultra-sonography of abdomen & pelvis was normal. Electrocardiogram showed sinus tachycardia. He was resuscitated with intravenous fluids and first dose of broad-spectrum antibiotic (Carbapenem) was injected. Right internal jugular vein was cannulated, with central venous pressure of 5cm of H_2O. A provisional diagnosis of GI sepsis with multi organ involvement (renal, hepatic, hematologic) was made. The possibility of GI bleed and cardiac cause of shock was also considered. Once the history of consumption of bitter bottle gourd juice was revealed, toxicity due to this was considered. He was also treated with proton pump inhibitors, hydrocortisone and other supportive measures.

His blood pressure improved to 100/60 mm Hg with mean arterial pressure of 80 mm of Hg, after he was adequately hydrated and started on an infusion of noradrenaline. His urine output improved to 100 cc per hour over next few hours. 2-D Echocardiogram done showed mild distal septal & localized hypokinesia with LV ejection fraction of 60 %. He was treated with supportive medications & antibiotics for next few days. The serial laboratory investigations showed rapid improvement [Table/Fig-1&2]. He was off vasopressors the next day. The patient's further course in hospital was uneventful & he was discharged on the fifth day.

DISCUSSION

Drinking of bottle gourd juice in the morning on empty stomach is considered a remedy for diabetes, heart disease, hypertriglyceridemia, constipation, liver diseases, urinary problems, depression, etc. by practitioners of Ayurveda and alternate medicine [1,2]. In this era of rapid media communications, such practices are increasing, fueled by increasing use of Ayurvedic, complementary or alternate therapies to treat ailments. However, there is lack of standardization in the preparation of such juices and sometimes, especially when the juice is bitter, such medications can become dangerous and life threatening. A handful of reports have been published regarding clinical presentation, endoscopic appearance and treatment of bottle gourd toxicity in humans [3-6]. We report a case of bottle gourd juice poisoning with shock and multi-organ system failure, and review the literature in general and discuss the differential diagnosis in particular. To our knowledge, this is the first case to be reported from Western India.

Bottle gourd (*Lagenaria siceraria*) belongs to the Cucurbitaceae family which has nearly 100 genera and over 750 species. It includes plants such as cucumber, colocynth, bitter gourd, zucchini, eggplant, squash, pumpkin and water-melon. It is commonly known as lauki in Hindi and is a common vegetable grown and eaten all over India, and the tropical and sub-tropical world. It is the unripe fruit which is most commonly utilized, after it is cooked. The fruits are believed to have cardioprotective, diuretic and nutritive properties by practitioners of Ayurveda and alternate therapy [4,6].

TEST	Day 1	Day 2	Day 3	Day 4	Day 5
Hemoglobin gm%	16	11.5	10.5	10.3	12.1
Packed Cell Volume %	48.7		40		
White Blood Cells / cumm	35,400	30,900	30,000		18,800
Neutrophils %	88	90	90		90
Lymphocytes %	6	8	8		8
Eosinophils %	0				1
Monocytes %	6	2	2		1
Platelets lacs/cumm	3.12				
International Normalised Ratio	1.69	1			
Blood Sugar mg/dl	122	277			135
Blood Urea mg/dl	50	22	20	20	17
Serum Creatinine mg/dl	3	2.4	2	2.1	1.8
Serum Bilirubin mg/dl		0.6	0.5		0.8
SGOT IU/L		97	39		14
SGPT IU/L		224	118		83
Alkaline Phosphatase IU/L		68	82		112
Serum PROTEIN gm/dl		4.8	4.2		5.4
Serum ALBUMIN gm/dl		2.4	2.6		2.9
Albumin:Globulin ratio		2			
Serum Sodium meq/L	153	140	144		143
Serum Potassium meq/L	6.2	5.6	4.4		3.7
Serum Chloride meq/L	113	108	113		106
Serum Uric Acid mg/dl		6			
Serum Lactate Dehydrogenase		34			

[Table/Fig-1]: Serial investigations results

Parameter	Day 1	Day 2	Day 3	Day 4
pO_2	82.2	94	89.3	88.9
pCO_2	19.2	29	32.7	34.5
pH	7.326	7.339	7.389	7.427
HCO_3	13.5	17.2	20.8	23.2
BE	-13.6	-9	-4.4	-1.4

[Table/Fig-2]: Serial ABG reports

As a defense mechanism against insects and herbivore animals, the plant produces Cucurbitacin, a pheromone. Cucurbitacins, which impart the bitter taste, are tetracyclic triterpenoids, hydrophobic derivatives of triterpenes, of various types (B, D, G and H), increased in concentration within the fruits of plants grown under stressful conditions like dehydration, extreme temperatures, poor soil quality. Cucurbitacins have shown promising pharmacological properties in animals such as anti-tumorigenic effects (in-vivo, in-vitro), cytotoxic effects, and increased capillary permeability leading to hypotension and ascites and pleural effusions [3-6]. Cucurbitacins are lethal in small doses in mice (1.2 mg cucA/kg) and fruits containing more than 130 ppm will cause symptoms if consumed [3,4]. About 50-300 ml of juice can cause symptoms and the larger the quantity of the juice /fruit ingested the more severe the symptoms [4-6].

Humans who consume the bitter juice of bottle gourd present with abdominal pain (often severe), vomiting, and diarrhoea in the majority. Evidence of gastrointestinal bleed in the form of hematemesis, malena, hematochezia, hypotension followed by oliguria, and elevated liver enzymes are seen in 50-75% of patients [4,5]. Symptoms start usually within 30 min (range 3 min to 9 h) of the consumption of the bitter bottle gourd juice and may continue for 2 to 7d [4-6].

The differential diagnoses we considered were:

i) Sepsis with multi- organ dysfunction with septic shock with the focus of infection in the gastrointestinal tract: due to presence of abdominal symptoms with bleeding, presence of SIRS, leucocytosis, hypotension progressing to shock, and associated hepato-renal abnormalities with metabolic acidosis. But there was no history of fever and urine, stool and blood culture did not show any growth. As fever may sometimes be absent in some case of severe sepsis and also the culture results would be available after three days of incubation, the patient was started on a course of broad spectrum antibiotics [7].

ii) Food poisoning or foodborne illness due to preformed toxins of *Staphylococcus aureus* or *Bacillus cereus*: the symptoms start typically 1 to 6 h after ingestion of food in infections with *Staphylococcus aureus* and after 10 to 16 h in infections with *Bacillus cereus*. Moreover, symptoms will resolve faster (24-48 h) [8].

iii) GI bleed (bleeding peptic ulcer, esophageal variceal bleeding, etc.) with haemorrhagic shock and organ dysfunction: there was no history of any predisposing factors for GI bleed except for consumption of aspirin, and presence of leucocytosis with lactic acidosis indicate some other aetiology [4,7].

iv) Cardiac cause for the hypotension/shock: absence of a history of chest pain and good ejection fraction on echocardiography.

The possibility of bottle gourd poisoning was considered in our patient because of earlier reports of adverse reactions after consumption of vegetables belonging to Cucurbitaceae family. The Naranjo adverse drug reaction probability scale [9] guided us towards a probable diagnosis of bottle gourd toxicity in our patient. The following points were in favour: i) Adverse reactions appearing within 30 min after consumption of the bottle gourd juice which was bitter in taste, ii) improvement in symptoms after discontinuation of the juice and iii) response to treatment with intravenous fluids, steroids, vasopressors and other supportive therapy. Re-challenge was not practical, so it was not done. Cytotoxic effect may have been responsible for renal, myocardial and hepatic dysfunction.

CONCLUSION

Toxicity with bottle gourd, which is bitter tasting, can occur in some as it is a commonly consumed vegetable and it may be actually more common than reported in literature. It presents with abdominal discomfort, hematemesis, hypotension with or without organ dysfunction which starts within approximately 30 min after consumption of the bottle gourd or its juice. It may be easily confused with other illnesses like GI sepsis which have similar presentation. Increasing public awareness about the dangerous effects of consumption of bitter bottle gourd will prevent further cases.

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