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A RARE CASE REPORT OF BOTTLE GOURD POISONING PRESENTATION IN THE EMERGENCY DEPARTMENT

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Abstract

Bottle gourd (*Lagenaria siceraria*) popularly known as lauki or ghiya in India is known to be one of the healthiest vegetables and its juice, as per Ayurveda, is sometimes used as an adjunct to medical treatment for diabetes mellitus, hypertension, weight loss, liver diseases and many other ailments. But intake of juice which has turned bitter with large amounts of a tetracyclic triterpenoid toxin – Cucurbitacin can cause a reaction to the gut mucosa leading to acute onset abdominal pain, blood mixed vomiting (hematemesis), blood mixed loose stools, severe hypotension which can be fatal with a patient having known comorbidities. Furthermore, it can lead to septicaemia with septic shock and multi organ involvement. This kind of toxicity is very rare and only a few cases have been reported. We as emergency physicians must be aware of such toxicities especially in India where this bottle gourd is consumed a lot.

Keywords: Poisoning, Bottle Gourd, Cardiac Arrest.

CASE REPORT

A 46-year-old known diabetic patient presented to ED with complaints of persistent loose watery stools which were sudden in onset, gradually progressive, blood tinged associated with vomiting around 4-5 episodes. He also complained of excessive sweating for 2 hours. Patient had one episode of blood-tinged vomitus in the Red Zone of ED. The episodes had begun half an hour after he had consumed bottle gourd (ghiya) juice at around 8:00 P.M. He presented with feeble pulse around 113/minute, blood pressure- non-recordable, respiratory rate around 18/minute, saturation of oxygen around 98% on room air, temperature 98.6^{o}F and random blood sugar 270 mg/dL. His chest was bilaterally clear with soft abdomen having mild tenderness in the epigastric and umbilical region. The patient was conscious and oriented to time, place and person with a GCS 15/15. An arterial blood gas was done which showed severe high anion gap metabolic acidosis (HAGMA) with a pH-7.11, pCO_2- 40, pO_2-96, $HCO3^-$ -10, anion gap- 18 and lactate- 10.2. Electrocardiogram showed sinus tachycardia with normal axis. He was given intravenous pantoprazole, ondansetron, metoclopramide, drotaverine and octreotide followed by infusion of pantoprazole and octreotide and tablets racecadotril and doxycycline. He was given 2 litres of isotonic normal saline and 2 litres of lactated ringer's solution. After 4 litres of fluid resuscitation, blood pressure was recorded as 110/70 mmHg. He was referred to general medicine doctor on duty and shifted to the MICU, with a provisional diagnosis of bottle gourd poisoning with upper gastrointestinal bleeding with shock with severe metabolic acidosis with severe dehydration. Initial blood counts showed haemoglobin 20.10 g/dL, total leucocyte count 29300, red blood cells 7.05 million/mm3, packed cell volume 67%,

platelet count 1.48 L, SGOT 249 U/L, SGPT 105 U/L, ALP 207 U/L and Albumin 2.40 g/dL. Renal function tests were within normal limits.

After around 1 hour patient again had a non-recordable blood pressure. An infusion of nor-adrenaline (8/50) was started at a rate of 20 ml/hr. But the blood pressure still remained non recordable and patient started to desaturate and developed tachypnoea. The patient was intubated for above mentioned indications with ET tube of size 8.5. At 6:55 A.M the patient became unresponsive with non-palpable carotid pulse. High quality CPR was initiated as per latest ACLS guidelines. CPR was continued for 15 minutes and return of spontaneous circulation (ROSC) was achieved but the blood pressure was still non recordable. Patient was started on adrenaline infusion. At 8:25 A.M the patient monitor started showing asystole and high-quality CPR was initiated again. ROSC was achieved after 4 cycles and infusion dopamine was started at 10 mcg/kg/hr. At around 3:00 P.M monitor started showing ventricular fibrillations and again CPR was given for next 30 minutes but the patient could not be revived. ECG showed flatline (asystole). The patient was declared dead at 3:30 P.M.

DISCUSSION

Intake of bottle gourd juice on an empty stomach early in the morning is considered to be a cure for hypertension, diabetes mellitus, constipation, liver diseases, heart diseases, hypertriglyceridemia, urinary issues, etc. by Ayurvedic practitioners [1,2]. Misinformation on social media has caused a surge in such malpractices. But because of a lack of proper procedure for preparation of such juices, and sometimes when juice is bitter, this can turn to be life threatening. Some publications discuss the clinical presentation, endoscopic vision and management of bottle gourd poisoning in human beings [3-6]. We hereby present a case of bottle gourd poisoning with severe shock, multi-organ disfunction and sepsis leading to mortality.

Bottle gourd (*Lagenaria siceraria*) belongs to Cucurbitaceae family having around hundreds of genera and over 700 species such as plants like cucumber, sponge gourd. The fruits are known to have protective properties for heart, kidneys and nutritional in Ayurveda and alternate therapy [5,7]. To protect themselves from animals and insects, they produce a bitter tasting toxin, Cucurbitacin, which is a tetracyclic triterpenoid, which has shown promising pharmacological properties in animals such as anti-tumorigenic effects, cytotoxic effects, and increased capillary permeability causing third space losses leading to hypotension, ascites and pleural effusions [4-7]. Around 60- 250 mL of juice is known to cause clinical features and a larger quantity of juice /fruit taken leads to more severe symptoms [5-7].

Humans ingesting bitter juice of bottle gourd complain of severe pain per abdomen, vomiting, and loose stools classically. Clinical features appear within the initial half hour (range 3 minutes to 9 hours) of the ingestion of bitter bottle gourd juice and may continue for next 7 days[5-7].

CONCLUSION

We considered our patient to have Bottle Gourd Toxicity because of similar reports of such an event due to ingestion of vegetables from Cucurbitaceae family. Cytotoxic effects may have led to multi organ dysfunction causing sepsis, septic shock and consequently death. It is one of the rare case reports been published.

References

1) BVS Lakshmi, PU Kumar, N Neelima, et al. Hepatoprotective effects of L siceraria. *Res J Pharmaceut Biol Chem Sci.* 2011; 2:130–37.

2) BV Ghule, MH Ghanti, PG Yeole, AN Saoji. Diuretic activity of Lagenaria siceraria fruit extracts in rats. *Indian J Pharm Sci.* 2007; 69:817–19.

3) Khatib KI, Borawake KS. Bottle gourd (Lagenaria siceraria) toxicity: a "bitter" diagnostic dilemma. J Clin Diagn Res. 2014 Dec;8(12):MD05-7.

4) A Sharma, JP Sharma, R Jindal, RM Kaushik. Bottle gourd poisoning. *JK Sci.* 2006;8:120–21.

5) R Puri, R Sud, A Khaliq, M Kumar, S Jain. Gastrointestinal toxicity due to bitter bottle gourd (Lagenaria siceraria)- a report of fifteen cases. *Indian J Gastroenterol.* 2011; 30:233–36.

6) Indian Council of Medical Research Task Force. Assessment of effects on health due to consumption of bitter bottle gourd (Lagenaria siceraria) juice. *Indian J Med Res.* 2012; 135:49–55.

7) CH Ho, MG Ho, S Ho, HH Ho. Bitter Bottle Gourd (Lagenaria Siceraria) toxicity. *J Emerg Med.* 2014;46(6):772–75.