Hematemesis due to bitter melon (*Momordica charantia*) extract-induced gastric ulcerations

Bitter gourd also known as bitter melon or *Momordica charantia* L. (Family: Cucurbitaceae) or *karela* (Indian name) is a herb widely grown in tropical countries like India. As an unripe fruit, it is commonly eaten as a vegetable. Bitter gourd juice either alone or admixed with extracts of spinach, carrot and other vegetables is consumed as health drink for its presumed blood purifying effects. It constitutes a major portion of complementary and alternative medicine (CAM) and is used for treatment of skin infections, diarrhea, piles, tumors, asthma and hypertension, to name a few. Bitter gourd contains *gurmarin*, a polypeptide, which has anti-diabetic properties; hence is consumed by a large diabetic population. In addition, it has other alkaloids like momordicine, lectin or charantin.

Despite its large-scale consumption there is paucity of data on the side effect profile of bitter gourd. The hepatotoxic effects of bitter gourd have been demonstrated in animals. In children it has been shown to induce hypoglycemia. The seed constituent, vicine may induce “favism,” an acute condition characterized by headache, fever, abdominal pain, and coma. However gastrointestinal side effects have not been described. Here we describe acute gastric ulceration presenting as hematemesis as direct toxic effect of this herbal extract.

A 40-year-old man who consumed around half a liter of concentrated homemade liquid extract of bitter gourd, on the pretext of purifying his blood, presented to the emergency with history of severe epigastric pain and hematemesis (around 200-300 mL) within half an hour following this binge. There was no history of using any additive substance while preparing this extract. There was no history of prior alcohol abuse, non steroidal anti-inflammatory drugs or any other drug intake. There was no antecedent history of epigastric pain or discomfort in the past. On examination, he was pale, had a pulse rate of 120/min and blood pressure of 80/60 mmHg. Rest of the general physical and systemic examination was normal. His hemoglobin was 8.0 g% and hematocrit 23%, with normal leukocyte and platelet counts. His serum amylase was normal as were his liver and renal function tests. Ultrasound abdomen was unremarkable for evidence of portal hypertension or pancreatitis. Upper gastrointestinal endoscopy showed normal esophagus; the gastric mucosa was hyperemic, and there were ulcerations in the distal body and antrum (Fig. 1). There were no esophageal or gastric varices. Rapid urease test of the gastric biopsy was negative. The patient was managed with intravenous fluids, blood transfusion and intravenous rabeprazole. He improved and a repeat endoscopy showed normal stomach mucosa with no sequelae.

Bitter gourd-induced acute gastric ulceration has not been described earlier. On the contrary there is a single study demonstrating the healing effects of methanolic extract of bitter gourd on gastric and duodenal ulcers in rats. However there are no such data in humans. Humans consume bitter gourd as a vegetable or its extract in the form of juice generally mixed with other vegetables. Considering the widespread use of this vegetable in various preparations in India in the form of cooked vegetable or raw juice, this side effect is unusual. We speculate that cooking and admixing of bitter gourd extract with other vegetables possibly dilutes the toxic effect. Since our patient had consumed a large amount of pure concentrated bitter gourd extract possibly a direct toxic corrosive like effect led to gastric ulceration and upper gastrointestinal bleed. We postulate that alkaloids like momordicine or constituents like...
lectin or charantin which are present in bitter gourd may be responsible for this corrosive effect.

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References

Seroprevalence of hepatitis C virus in rural population of Bangladesh

Hepatitis C virus (HCV) is a predominant cause of chronic hepatitis and cirrhosis and hepatocellular carcinoma worldwide. HCV infection is endemic in many countries, with an estimated approximately 170 to 200 million HCV-infected persons world-wide. However, there is considerable geographical variation in the incidence and prevalence of HCV infection. Much of this variability can be explained by the frequency of different risk factors for HCV infection, such as injectable drug use, blood transfusions and organ transplantation, unsafe injections and other healthcare related procedures, occupational exposure, and unsafe sexual practices.

Bangladesh is a densely populated country with no population-based data on seroprevalence of hepatitis C virus (HCV). This study assessed the prevalence of HCV in a rural area of Bangladesh.

During July 2007 to June 2008, serum specimens were collected from 1508 subjects (mean age 32.9 [17.8] years, 790 [52.3%] women, 1144 married) residing in a rural area of Bangladesh, located about 60 kilometers from Dhaka city.

The study subjects were selected using systematic random sampling. A structured questionnaire was used to gather relevant data. A blood specimen (5 mL) was collected from each subject and transported using a cold chain to our institution, where serum was separated. Specimens were stored at -70°C; a commercial third-generation anti-HCVchemiluminescence-based enzyme immunoassay (Vitros Eci, Johnson & Johnson, USA) was used for detection of anti-HCV antibodies.

Of the study subjects, 639 (42.4%) were housewives, 296 (19.6%) were students, 209 (13.9%) were businessmen and 175 (11.6%) were farmers. History of surgery, blood transfusion, dental procedures or hospitalization was available in 285 (18.9%), 22 (1.5%), 245 (16.2%) and 272 (18 %) subjects, respectively. In addition, 4 (0.3%) had history of multiple sexual exposures, 6 (0.4%) were alcoholic and none gave history of intravenous drug abuse. Most of the subjects (1300 [86.0%]) belonged to middle socioeconomic class and only 35 (2.3%) to high socioeconomic class.

Only 7 (0.5%; 3 men) subjects tested positive for anti-HCV antibodies. HCV positive subjects were older (mean age 48.6 [12.5] years in HCV positive subjects vs. 32.8 [14.8] years in HCV negative subjects; p=0.005). All the HCV positive subjects belonged to middle class society; of them, one had diabetes mellitus, two had history of dental procedures, two had history of surgery and three had history of hospitalization. None of the HCV-positive subjects had history of multiple sexual exposure or blood transfusion.

Our data show that the prevalence of HCV infection in a rural community in Bangladesh is relatively low, with only about 0.5% showing evidence of HCV infection. This suggests that nearly 0.7 million persons in the country have had HCV infection. No clear mode of HCV transmission could be identified. Larger seroprevalence studies are needed to further clarify the seroprevalence of HCV infection and risk factors for this infection in Bangladesh.