CASE REPORT

Star fruit intoxication leading to acute kidney injury

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Abstract

There are few case reports regarding star fruit's nephrotoxicity and neurotoxicity in chronic kidney disease patients. Recently cases are found in people with normal renal function Star fruit nephrotoxicity is believed to be due to its high oxalate content which causes acute obstructive oxalate nephropathy. A neurotoxin (caramboxin) present in the fruit is responsible for neurotoxic features. Here we present a young male who developed acute kidney injury following star fruit ingestion in empty stomach. After admission, patient was treated conservatively and recovered completely.

Introduction

Carambola, also known as star fruit, is the fruit of Averrhoa carambola species of tree native to the Philippines, Indonesia, Malaysia, India, Bangladesh and Sri Lanka. Star fruit (Fig. 1) is one of the plant sources that contain highest concentration of oxalic acid; 100g of fresh fruit contains 50,000-95,800 ppm of oxalic acid. There are two main types of the fruit: the small sour (or tart) type and the larger sweet type. The sour varieties have higher oxalic acid content than the sweet type.¹

The maximum recommended volume of star fruit juice or amount of fruit one can ingest safely has not been determined yet. The literature contains cases of fasting individuals developing nephropathy after having 300 ml of pure star fruit juice.² The amount of fruit ingested which causes toxicity can be as low as approximately 25 ml or half a fruit.³ The levels above which star fruit intake can cause acute oxalate nephropathy have to be defined and considered along with the individual risk factors that may increase the chance of adverse events.² ⁴ The risk factors of getting star fruit intoxication are fasting state, dehydration, large amount of juice and poor renal function.² ⁵

Two distinct effects may be triggered nephrotoxicity and neurotoxicity. Star fruit nephrotoxicity is believed to be due to its high oxalate content.² Oxalic acid and its soluble salts are potentially harmful to humans and animals, contrary to insoluble oxalic acid calcium and magnesium salts.² ⁵ When individuals ingest, free oxalic acid binds to calcium or magnesium in the gastrointestinal tract to form insoluble complexes that cannot be reabsorbed. Free reabsorbed oxalate binds to circulating calcium and precipitates in the renal tubules, causing acute obstruction and tubular injury.⁴ Experimental study in rats has suggested that oxalate crystals may provoke Acute Kidney Injury (AKI) by inducing apoptosis of renal epithelial cells.⁶ ⁷

The neurotoxicity is due to caramboxin. Healthy people clear out this agent through the kidneys without problems. In those with kidney disease, however the toxin accumulates and can eventually enter the brain. Caramboxin is a molecule similar to phenylalanine and a strong glutamate receptor which produces cerebral hyperexcitability. Caramboxin acts on AMPA and kainate receptors, two important glutamate controlled neurotransmitter receptors of the central nervous system. This causes hyperexcitability in the brain. There are a variety of symptoms: from intractable hiccups, vomiting, weakness, mental confusion, and psychomotor agitation, to unusually long lasting epileptic seizures, coma, and death.⁸ ⁹

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Case Report
A young farmer of 30 years hailing from Bagerhat was admitted on 24/08/2014 in Shaheed Sheikh Abu Naser Specialized Hospital, Khulna with the history of abdominal pain, back pain, vomiting, hiccup and reduced urine volume following ingestion of 3-4 medium to large sized star fruits in empty stomach. There was no history of acute watery diarrhea, blood loss and ingestion of any nephrotoxic drugs. The time between ingestion and development of symptoms was about 3-4 hours. He had no history of renal disease. On admission he was confused and mildly edematous (Fig. 2). Vital parameters were within normal range. Serum creatinine was raised. He was diagnosed as a case of acute kidney injury probably due to star fruit intoxication. After admission he was followed up closely and treated conservatively. He recovered completely. Dialysis was not required.

Investigation reveals initial serum creatinine as 2.9mg/dl. USG showed right kidney (BPL-11.4 cm), left kidney (BPL-11.7cm). Renal parenchyma was hypoechoic. Complete blood count, liver function test was normal. During discharge his serum creatinine was 1.2mg/dl. Kidney biopsy was done and histopathology report was in light microscopy; most of the glomeruli reveals segmental increase of mesangial cells and matrix. Diffuse interstitial infiltration of lymphocytes, eosinophils and neutrophils was present. There was marked edema in the interstitium. Some renal tubules contain pus casts in the lumen with shedding and necrosis of the tubular epithelium in some renal tubules. No crystal of any form was present in the lumen. Blood vessels were normal. In direct immunofluorescence study no deposit of IgA, IgG, IgM, or C3 was present. Histological diagnosis was acute tubulointerstitial nephritis with mild tubular necrosis.

Discussion
This patient showed acute renal failure and neurotoxic symptoms (persistent hiccups and mild confusional state) following ingestion of star fruits in empty stomach. Patient was treated conservatively without dialysis and recovered completely. This feature is similar to the findings of Moyses Neto et al. who described case series of 5 patients with previously normal renal function. The patients with normal renal function developed acute nephrotoxic and neurotoxic feature after ingestion of Star Fruits or its juice. In three of five patients kidney biopsy was not done. The diagnosis was done on the basis of history, clinical finding and biochemical parameters.

There is no baseline data in this patient. But recovery and normal renal function after treatment give clue to his normal kidney status. Kidney biopsy shows all histological features similar to oxalate nephropathy except oxalate crystals deposition. This type of histological features may also be present in pre renal AKI or in acute tubular necrosis. In this case AKI may be due to pre renal cause. But if we consider all the scenario of the patient the diagnosis goes more in favor of carambola intoxication. The patient took star fruits in empty stomach and developed AKI with CNS features. But the failure to detect oxalate, crystal in biopsy does not rule out carambola induced AKI. Rather it demands exploration of more cases. It may be associated with severity of renal involvement. To detect oxalate crystals, other investigations like polarized microscopy and von Kossa stain could be helpful. Most of the literatures have focused on patients...
with renal impairment who became ill after taking star fruit. There are limited reports on cases with normal renal function. The particular interest of this paper is to present a rare case of AKI due to star fruit intoxication that can be easily missed unless such history of star fruit intake is specifically sought to rule out the existence of this disease and raise new speculations in this epidemiological area.

Reference