

Acute Kidney Injury Due to Star Fruit Ingestion: A Case Report

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Abstract:

Star fruit (*Avarrhoa carambola*) is a fruit from oxalidace family. It is found in many countries of the world including Bangladesh. But its ingestion or drinking star fruit juice may lead to intoxication especially in patients with chronic kidney disease and manifestations might be neurological or nephrological. It may also cause acute kidney injury in patients with previously normal renal function. Here we are presenting a case who presented with acute kidney injury after star fruit ingestion with previously unknown renal function impairment. The etiology was confirmed by histopathological examination after doing renal biopsy. This renal function impairment is mainly due to oxalate crystal induce nephropathy which is richly abundant in star fruit. His renal function was improved with conservative management. Physicians should be alert to consider the ingestion of star fruit as a cause of acute kidney injury in a patient even in the absence of previous renal function impairment.

Key Words : Acute Kidney Injury, Star Fruit.

[BSMMU J 2014 ; 7 (2) : 151-153]

Introduction:

Star fruit, *Avarrhoa carambola*, is from Oxalidace family (Figure 1). It is also known as belimbing beti (Malaysia), yang tao (China), five fingers (Australia) and Kamranga (Bangladesh). Ceylon and the Molusacus are thought to be areas where star fruit first originated. But it is cultivated in South East Asia including Malaysia, Thailand, Hong Kong, China, Taiwan, Brazil and other places for many centuries.^{1,2} It is also grown in the Indian subcontinent including Bangladesh. It is a very popular fruit in this part of the world.

It has been recently known that patients of chronic kidney diseases are susceptible to intoxication from eating star fruit or drinking its juice and this toxic effect is independent of dialysis. Star fruit intoxication mainly shows neurological manifestations in this type of patients e.g. hiccough, vomiting, seizures, coma and death³. But star

fruit nephrotoxicity in patients with previous normal renal function has been rarely reported. There are few publications reporting cases of acute renal failure due to acute oxalate nephropathy in patients with previously normal renal function. Here we are presenting a case with previous normal renal function developing acute kidney injury after ingestion of star fruit.

Case reports:

A 41-year-old diabetic, normotensive man, presented with several episodes of vomiting and hiccough associated with upper abdominal pain which started 2 hours after ingestion of about half kilogram of star fruit. Vomiting was non-projectile; the vomitus contained ingested fruits and was not foul smelling. Abdominal pain was mild, colicky in nature and confined to the upper abdomen having no radiation. Initially he was treated conservatively, resulting in improvement of his vomiting and abdominal pain. But subsequently he developed anorexia accompanied by scanty urine despite being

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hemodynamically stable with normal pulse and blood pressure. Though his previous renal function was normal, at this time his serum creatinine was raised (3.2mg/dl) . At this stage he was referred to nephrology unit for specialized management. On query, he denied any other systemic illness or ingestion of any NSAIDs or other nephrotoxic and/or herbal medication.

At the time of admission in nephrology unit he was conscious and oriented. He was not dehydrated and was euvolumic. His pulse rate was 80 beats/min , blood pressure was 130/80 mm of Hg and temperature was normal. Other systemic examinations including optic fundi were normal. Bed side urine was negative for albumin, acetone and sugar. The white cell count was $8.5 \times 10^9/L$, haemoglobin 12.5 gm/dl, and platelet count $370 \times 10^9/L$. His serum creatinine and blood urea were 7.0 mg/and 116mg/dl respectively. Urine RME showed pus cell 4-6 /HPF, epithelial cell 2-4/HPF, RBC 2-4/HPF with oxalate crystaluria and urine for phase contrast microscopy yielded 10% dysmorphic RBC. His serum electrolytes revealed hypokalaemia with acidosis (Na- 137 mmol /l, K- 3.1mmol/l, Cl- 100 mmol/l , CO₂ - 20mmol/L). His liver function tests ,other biochemical reports including S Ca, S Mg , S PO₄, fasting lipid profile, ECG ,CXR (PA view) were essentially normal but USG of whole abdomen showed swollen kidneys bilaterally(right kidney 111mm, left kidney 107mm) with decreased cortical echogenicity.

During his hospital stay his serum creatinine was gradually rising and it was 8.5mg/dl after two days of his admission in renal unit. His urine output was still 200-250ml/day. To establish the definite cause of his acute kidney injury, renal biopsy was done for histopathological examination which showed global hyalinization of glomeruli with dilated tubules lined by flattened epithelial cells. Some tubules showed cytoplasmic swelling and vacoulation of the lining cells. Few tubules contained eosinophilic debris often mixed with degener-

ated cells and one tubule showed crystalline deposit. Interstitium revealed marked oedema with focal aggregates of lymphocytes. (Figure 2) All these findings established the diagnosis of acute tubular necrosis as a cause of his acute kidney injury.

With conservative managements his renal function was improving gradually and the patient went into the diuretic phase from the 4th day of admission. Urine output was raised to 3 litres/day(from 250ml/day on admission) and serum creatinine came down to 1.2 gm/dl after eight days of his admission and he was discharged with a diagnosis of acute kidney injury due to star fruit toxicity with diabetes. On follow up visits at two and six weeks after discharge his renal function was found stable within normal range.

Discussion:

Our patient presented initially with vomiting, hiccough and abdominal pain without any other types of neurological manifestations and subsequently developed acute kidney injury which improved with conservative management. There are few case reports and studies which showed neurological toxicities were the main presentation in patients with pre-existing chronic kidney disease specially in stage 3-5 without any correlation whether they are on dialysis or not, leading coma and status epilepticus a refractory to conventional therapy⁴ and death. Neto et al. performed a study on the treatment and outcome in a number of uremic patients who were intoxicated with star fruit. All of them presented with different types of neurological manifestations like hiccough, mental confusion, seizures and coma.⁵ In Bangladesh, a case of star fruit intoxication in a patient with pre-existing chronic kidney has been reported who presented with hiccough and disorientation.⁶

In addition to causing neurological symptoms as described, star fruit may cause reversible acute nephrotoxic effects. Chen et al. first reported two cases of acute

oxalate nephropathy in patients with previous normal kidney function in 2003 and found no neurological manifestations in those patients. In 2009, for the first time, Neto et al. published a study including five patients with normal renal function, who developed nephrotoxicity after ingestion of star fruit. However they reported simultaneous neurological as well as nephrotoxic effects after ingestion of star fruit in patients with normal renal function. They noticed mild mental confusion and hiccoughs in their patients in addition to acute renal failure⁷. Our patient also had pre-existing normal renal function prior to ingestion of star fruit and developed hiccough after intoxication which might be due to neurological toxic effects. Though star fruit, like all other fruits contains probably high potassium content, but in previous case reports no changes in potassium level was noticed⁸. We also did not find any rise in the potassium level in our case rather the potassium level was below than the normal range, which probably was caused by vomiting.

There have been few studies about the mechanism of nephrotoxic effects of star fruit. It has been postulated that star fruit induces acute renal injury not only through obstructive effect of calcium oxalate crystals, but also by inducing apoptosis of renal epithelial cells, which may be caused by the levels of oxalate in the fruit.⁹ However, this acute nephrotoxic effect is reversible as have been reported in few case reports.⁸ Our patient also had complete reversal of his renal injury with only conservative management.

Conclusion :

Large amount of star fruit ingestion or even smaller amounts on an empty stomach, may provoke acute oliguric renal failure even in people with normal renal function. Physician in alert of the possibility of acute kidney injury when a patient with previously normal renal function presents with one or more specific symptoms like hiccoughs, back pain or mental confusion after ingestion of star fruit.

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