

Case Reports

A case of star fruit induced acute oxalate nephropathy in a previously healthy child

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Introduction

Acute oxalate nephropathy is a known complication of consumption of food items rich in oxalate¹. Star fruit, locally named “Kamaranka” is a popular food item in Sri Lanka, rich in oxalate. In addition, the fruit contain antioxidants, ascorbic acid and potassium¹. There are several reported cases of acute kidney injury following consumption of star fruit especially when there is underlying renal damage^{2,3}. However, acute oxalate nephropathy (AON) is very rare in an otherwise healthy individual¹. We present a case of AON in a previously healthy child following ingestion of star fruit.

Case report

An 8-year-old previously healthy child presented to a District General Hospital (DGH) with a history of vomiting and abdominal pain of 12-hours duration. He has consumed six “Kamaranka” fruit four hours prior to the onset symptoms. He has taken his usual breakfast in the morning and had a snack before the consumption of Kamaranka. By the time of admission, he had anuria for 18 hours and had one episode of watery stools. There was no history of haematuria, oedema or headache.

Examination was unremarkable except for having elevated blood pressure. His serum creatinine and blood urea levels were 282 μ mol/L and 130.5mg/dl respectively. Urine full report showed moderately field full red cells and pus cells. Ultrasound examination of the abdomen showed swollen hyperechoic pyramids and thickened cortex

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suggestive of acute renal parenchymal disease. Rest of his basic haematological and biochemical investigations were normal. At the DGH, he was given two intravenous fluid boluses and he has passed urine at a rate of 0.2ml/kg/hr.



Figure 1: Star fruit

The child was transferred to Teaching Hospital Karapitiya for specialized paediatric nephrologist opinion. On arrival, his serum creatinine and blood urea levels had risen to 345 μ mol/L and 141 mg/dl respectively. Following admission, he was overhydrated and was kept under observation for monitoring of vital signs. He was started on metolazone and subsequently his urine output became normal. Blood pressure control was achieved with nifedipine. The diagnosis of AON was made after detecting elevated oxalate (oxalate: creatinine ratio 0.08) in urine.

He did not have a history of chronic diarrhoea or a family history of nephrolithiasis. There was no history of long term consumption of ascorbic acid. His creatinine level declined gradually and he did not require any form of renal replacement therapy. The child made an uneventful recovery and was discharged after 11 days of hospital stay with normal renal functions. His follow up ultrasound scan was normal.

Discussion

Hyperoxaluria in children occur either due to inherited metabolic conditions or secondary to ingestion of oxalate rich food or excessive consumption of vitamin C. In familial hyperoxaluria, there is a family history of ureteric stones due to calcium oxalate. In addition, hyperoxaluria causes tubule-interstitial nephritis which leads to acute or chronic kidney disease¹. There were several reported cases of AON following ingestion of star fruit and Bilimbi^{2,3,4}. Though, in the majority of cases, pre-existing kidney injury was detected, in this case there was no underlying renal pathology. AON following ingestion of star fruit is common when the patient is dehydrated but, in this case the child did not have any history of dehydration prior to the consumption of the fruit.

Clinical features of AON start with gastrointestinal symptoms such as nausea, vomiting, diarrhoea and upper abdominal pain within a few hours of ingestion³. This child had most of these symptoms within four hours of consumption. These symptoms are thought to be due to local irritation of the food rather than due to uraemia since it takes some time to develop². Like in this scenario, in most other reported cases, patients had presented with oliguria after 12-18 hours of ingestion.

Acute kidney injury (AKI) following hyperoxaluria could be multifactorial. It may be due to tubular obstruction with oxalate crystals or direct cellular toxicity⁵. Histopathological changes in AON include acute tubular necrosis, mesangial proliferation, interstitial oedema and deposition of oxalate crystals⁶. However, the diagnosis of AON can be made by demonstrating hyperoxaluria in the presence of a relevant history without doing a renal biopsy. Renal biopsy was not carried out in this patient since the diagnosis was straightforward and the child recovered well.

The mainstay of management of AON is the management of AKI. It is important to hydrate the patient with fluid exceeding 1.5L/m² despite oliguric renal failure⁷. Moreover, measures should be adopted to prevent hypercalciuria in order to inhibit calcium oxalate crystal formation. Therefore, in this patient we did not use furosemide since it causes hypercalciuria, instead he was given metolazone, a diuretic which causes hypocalciuria. In resistant cases, haemodialysis remains the last option⁷.

References

1. Glew RH, Sun Y, Horowitz BL, Konstantinov KN, Barry M, Fair JR, et al. Nephropathy in dietary hyperoxaluria: A potentially preventable acute or chronic

kidney disease. *World Journal of Nephrology* 2014; **3**(4):122.

<https://doi.org/10.5527/wjn.v3.i4.122>

PMid: 25374807 PMCID: PMC4220346

2. Neto MM, Robl F, Netto JC. Intoxication by star fruit (*Averrhoa carambola*) in six dialysis patients? (Preliminary report) *Nephrology Dialysis Transplantation* 1998; **13**:570–2.
<https://doi.org/10.1093/ndt/13.3.570>
PMid: 9550629
3. Neto MM, da Costa JA, Garcia-Cairasco N, Netto JC, Nakagawa B, Dantas M. Intoxication by star fruit (*Averrhoa carambola*) in 32 uraemic patients: Treatment and outcome. *Nephrology Dialysis Transplantation* 2003; **18**:120–5.
<https://doi.org/10.1093/ndt/18.1.120>
4. Bakul G, Unni VN, Seethaleksmy NV, Mathew A, Rajesh R, Kurien G, et al. Acute oxalate nephropathy due to 'Averrhoa bilimbi' fruit juice ingestion. *Indian Journal of Nephrology* 2013; **23**(4):297.
<https://doi.org/10.4103/0971-4065.114481>
PMid: 23960349 PMCID: PMC3741977
5. Jonassen JA, Cao LC, Honeyman T, Scheid CR. Mechanisms mediating oxalate-induced alterations in renal cell functions. *Critical Reviews in Eukaryotic Gene Expression* 2003; **13**:55–72.
<https://doi.org/10.1615/CritRevEukaryotGeneExpr.v13.i1.50>
PMid: 12839097
6. Barman AK, Goel R, Sharma M, Mahanta PJ. Acute kidney injury associated with ingestion of star fruit: Acute oxalate nephropathy. *Indian Journal of Nephrology*. 2016; **26**(6):446.
<https://doi.org/10.4103/0971-4065.175978>
PMid: 27942177 PMCID: PMC5131384
7. Glew RH, Sun Y, Horowitz BL, Konstantinov KN, Barry M, Fair JR, et al. Nephropathy in dietary hyperoxaluria: A potentially preventable acute or chronic kidney disease. *World Journal of Nephrology* 2014; **3**(4):122.
<https://doi.org/10.5527/wjn.v3.i4.122>
PMid: 25374807 PMCID: PMC4220346