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Case Report

Acute Kidney Injury Following Ingestion of Averrhoa bilimbi Juice

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Abstract :

Bilimbi/belembu fruit belongs to the family of Oxalidacae, species Averrhoa bilimbi. Freshly prepared concentrated juice of bilimbi has very high oxalic acid content and consumption carries a high risk of developing acute kidney injury (AKI) by deposition of calcium oxalate crystals in renal tubules. AKI due to Averrhoa bilimbi juice injestion is very rare. We report the case history of a 60-year-old hypertensive, dyslipidemic patient, with normal renal function, who ingested around 600ml of juice in fasting state for treating dyslipidemia. The patient developed AKI and required three sessions of hemodialysis. Her renal function returned to normal after five weeks.

Key Words: *Acute kidney injury, Averrhoa bilimbi, Hemodialysis*

Introduction :

The bilimbi fruit *(Averrhoa bilimbi)* belongs to the family of *Oxalidaceae* and grows in tropical countries. The fruits are cylindrical with five longitudinal lobes and measures from 4 to 10 cm [Figure I]. Probably it originates from the Moluccas Indonesia and is grown in India, Malaysia, Thailand and Bangladesh.[1] Bilimbi (commonly known as bilimbi, belembu, cucumber tree, tree sorrel, Irumban Puli, or Chemmeen Puli) is a plant with several suggested medicinal properties. However, it has a high content of oxalic acid which could contribute to nephrotoxicity.[2] By literature search we found only few cases of AKI due to bilimbi juice ingestion.[3] In our country to the best of our knowledge no such reports has been published. We report a case in which a patient with previously normal renal function had AKI after bilimbi juice ingestion.

Figure: *Averrhoa bilimbi* fruit and tree

Case report:

A 60-year-old hypertensive lady presented with bilateral pedal edema, facial puffiness, anorexia, nausea and vomiting of six-day duration following ingestion of about 600 ml of bilimbi juice. She had no history of hematuria or foamy urine, fever, dysuria, shortness of breath. She was detected to have dyslipidemia on routine evaluation one year back and was advised life style modification along with atorvastatin 10 mg once daily. She was on regular follow-up and was documented to have normal renal function and high cholesterol level two months back.

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On examination, she had periorbital puffiness along with pitting bi-pedal edema and high blood pressure (200/105 mm Hg). She did not have retinopathy and other systemic examination including precordium and abdomen revealed normal finding.

Investigations showed serum creatinine of 5.5 mg/dl, with normal hemogram and liver function tests. Urine analysis showed no albuminuria, haematuria and microscopy showed oxalate crystals. Ultrasound of abdomen revealed normal sized kidneys with normal cortical echogenicity and cortico-medullary differentiation. Other investigations are showed in table I.

After admission patient became oliguric (urine output 300ml/24 hours) and serum creatinine was increased to 7.1 mg/dl. She underwent three sessions of hemodialysis. Her urine output gradually increased to 3000 ml/day six days after admission. She was discharged ten days later with serum creatinine of 2.0 mg/dl and three antihypertensive drugs (amlodipine 5 mg daily, atenolol 50 mg twice daily and prazosin 2 mg thrice daily). She was on close monitoring & her serum creatinine returned to normal by 5 weeks.

Table I: Laboratory investigations

Investigation	Result	important reference
Urine Protein	Nil	
Deposits in Urine		
Red blood cells (RBC	0-1/hpf	
White blood cells (WBCS)	5-6/hpf	
Calcium oxalate crystal	+	
Blood		
Hemoglobin	10.9 g/L	
Total white blood cell count	10300 cells/mm^3	
Differential white blood cell count	P 70 L 23 E 7	
ESR	17 mm I first hour	
Platelet count	2.7 lakhs/mm^3	
Random blood sugar	5.5 mmol/L	
Blood urea	92 mg/dl	
Serum creatinine	5.5 mg/dl	0.6-1.4 mg/dl
Serum sodium	128 mg/dl	135-145 mo/l
Serum potassium	4.3 mg/dl	3.5-5 mmo/l
Serum bilirubin total	0.4 mg/dl	
Serum alanine transaminase	19 units/L	
Serum aspartate transaminase	34 units/L	
Total protein	68 mg/dl	
Serum albumin	38.5mg/dl	
Serum alkaline phosphatase	69 u/l	
Serum calcium	8.5 mg/dl	8.5-10.5 mg/dl
Serum phosphorus	4.0 mg/dl	2.4-4.3 mg/dl
Serum uric acid	7 mg/dl	2-6 mg/dl

Discussion:

A. bilimbi had been advocated for the treatment of hyperlipidemia, hypertension and diabetes by different communities. In Indonesia, the fruit is used to treat hypertension and the decoction of the leaves is used for the management of diabetes.[4] *A. bilimbi* is used as traditional medicine for treating cough, cold, itches, boils, rheumatism, syphilis, diabetes, whooping cough, and hypertension in Asia.[5] Earlier studies showed that ethanolic leaf extract of *A. bilimbi* and its semi–purified fractions possesses hypoglycemic and hypolipidemic properties in type I diabetic rats when administered both intraperitoneally[4] as well as orally.[6,7] Oxalic acid content of bilimbi fruit has been reported to range between 8.57 and 10.32 mg/g with highest levels seen in half ripe fruit in rainy season and lowest levels in ripe fruits in dry season (25.1 mg/100 g).[2] Various renal and neurological side effects like muscle weakness, intractable hiccups, mental confusion, seizures after ingestion of star fruit (*A. carambola*) have been documented.[8-10] *A. bilimbi*, which belongs to the same family, causing acute tubular necrosis due to high oxalate content has few case reports. Oxalic acid has a direct toxic effect on the renal tubules and interstitium, facilitates the development of oxalate crystals which are endocytosed by renal epithelial cells and promote a proliferative response. These crystals also stimulate specific genes in renal tubular cells, including the connective tissue growth factor gene leading to interstitial fibrosis.[3]

There is several case reports of acute oxalate nephropathy due to several agents described in the literature. Bakul et al had reported a series of cases from five hospitals in the state of Kerala, India who developed AKI due to acute oxalate nephropathy after consumption of bilimbi juice. In that series 7 out of 10 patients had dialysis requiring renal failure after intake of juice but fortunately all had renal recovery.[3] Nair S et al had reported 2 cases; both patients had complete renal recovery though one required dialysis support.[11]

Our patient was treated with 3 session of hemodialysis and her renal function returned to normal within 5 weeks. Renal biopsy may show oxalate crystal in renal tubule[3,11] but in this case as patient present lately and patient didn't comply with medical decision of renal biopsy so we couldn't go for renal biopsy. Serum oxalate levels could not be assessed in our patient as the facility was not available in our hospital.

Conclusion:

Bilimbi when used in high concentrations especially in empty stomach or dehydrated state, the fruit juice can lead to AKI due to acute tubular necrosis, owing to its high oxalate content, which results in intratubular oxalate crystal deposition. We conclude that patients with CKD not yet on dialysis and all patients on dialysis should be warned to avoid eating bilimbi. Individuals with normal renal function should be warned to avoid ingestion of large amounts of the bilimbi or juice especially in an empty stomach.

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