Case Report

Acute Oxalate Nephropathy in a Young Boy Due to Ingesting Averrhoa Bilimbi; Case Report and Literature Review of an Under-recognized Cause of tropical Renal Disease

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ABSTRACT

The occurrence of acute oxalate nephropathy (AON) as a result of consuming foods rich in oxalates is well -recognized. Although renal injury has been extensively recognized and described following the ingestion of star fruit (Averrhoa carambola), reports implicating 'bilimbi' (Averrhoa bilimbi), are far less common and were found in adults following ingestion of bilimbi juice. This report describes possibly the first case of AON occurring in an otherwise healthy eight year old boy following the consumption of several bilimbi fruit. Features which favoured dietary hyperoxaluria as the cause for his acute renal injury included the development of acute gastro-intestinal symptoms after ingesting the oxalate rich fruit, the presence of oxalate crystals in the urine full report and renal biopsy and the episodic reversible nature of his illness. Dehydration is likely to have contributed to his AON. The patient was managed medically, did not require dialysis and recovered completely within two weeks. It is important to note that dietary hyperoxaluria causing oxalate nephropathy may be an under-recognised cause of renal disease.

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Introduction

Acute oxalate nephropathy (AON) as a result of consuming foods rich in oxalates is well recognized [1]. However, the fact that commonly available fruit are rich in oxalates and therefore have the potential to cause renal damage, is less well known. Although renal injury has been described following the ingestion of star fruit (Averrhoa carambola), reports implicating 'bilimbi' or 'biling fruit' (Averrhoa bilimbi), are far less common. Moreover, in these reported cases, the renal injuries described had occurred in adults following the ingestion of bilimbi juice.

We describe what is possibly the first case of AON occurring in an otherwise healthy young boy, following the consumption of several bilimbi fruit, a common tropical fruit.

Case Report

A previously healthy 8 year old boy presented with acute onset vomiting, lower abdominal pain and gross haematuria. He had eaten six bilimbi fruit the same day, while playing cricket for about four hours in the hot sun on a humid day, without water.

He became anuric for more than 18 hours with the serum creatinine increasing to 508µmol/l. The urine full report revealed proteinuria, red blood cells and calcium oxalate crystals. A renal core biopsy stained with haematoxylin and eosin showed acute tubular destruction and inflammation with polymorphonuclear leucocytes on histologic sections (Figure 1). The renal tubules showed dilatation and destruction and were clogged by numerous fractured crystals which were polarisable (Figure 2). Interstitial fibrosis was notably absent. He developed acidosis, hyperkalaemia and hypertension which were managed medically. His urine output improved following a high dose of loop diuretics and he was started on steroids. His renal function improved gradually over the next two weeks and he recovered completely without the need for dialysis.

Discussion

Hyperoxaluria may be primary or secondary. Primary hyperoxaluria is metabolically inherited and characterized by the early appearance of renal failure and a family history of recurrent nephrolithiasis. Secondary hyperoxaluria may be due to increased oxalate absorption following the ingestion of oxalate-rich foods including herbs [1,2], due to Crohn's disease and follow gastric by-pass surgery or due to increased production of oxalate in the body as a result of prolonged Vitamin C ingestion or accidental ethylene glycol ingestion [3].

In Sri Lanka, two commonly consumed tropical fruit rich in oxalates are bilimbi and the star fruit. Ingesting star fruit juice has been well-documented as a cause of AON [4,5,6]. However, reports implicating bilimbi as a cause of AON due to hyperoxaluria are far less common and therefore not as well known.

Averrhoa bilimbi or the 'biling tree' is widely cultivated in Sri Lanka, India and Malysia [7,8]. Its sour fruit only rarely eaten raw, is yellowish –green in colour and contains a high oxalate concentration ranging from 8.57 to 10.32 mg/g. Pickling and dilution processes are known to reduce the oxalate content markedly [8]. A literature search for AON secondary to bilimbi consumption revealed only a few biopsy-proven cases, a series of ten patients from five hospitals and two similar cases, both reported from Kerala, India in 2014 and a third case from Bangladesh in 2015. All cases were described in adults secondary to consuming large amounts of

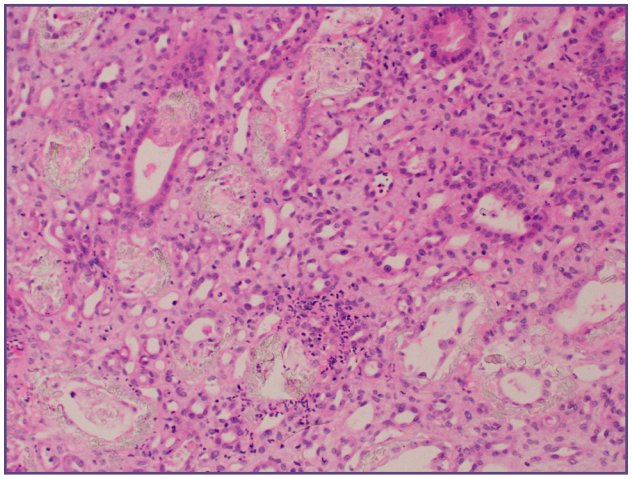

Fig. 1: Microphotograph showing acute tubular destruction and inflammation with polymorphs. Note the irregular oxalate crystals within and around tubules (H&Ex200).

Fig. 2: Numerous fractured deposits of oxalate crystals in the renal tissue under polarized light (x200)

fresh bilimbi juice as a presumptive, traditionally accepted herbal medication for the treatment of hypertension, diabetes and dyslipidaemia [9,10,11,12]. Many of these patients required dialysis during the acute stage of the disease.

Our patient was different as he was an otherwise healthy child, who had consumed six bilimbi fruit, (not juice) while playing cricket in the hot sun for several hours. Features which favoured dietary hyperoxaluria as the cause for his acute renal injury included the development of acute gastro-intestinal symptoms within several hours of ingesting the oxalate rich fruit, the presence of diagnostic oxalate crystals in both the urine full report and the renal biopsy (with accompanying tubular damage) and the episodic reversible nature of his illness.

While 'unbound oxalate' is readily absorbable in the gastrointestinal tract, the divalent cations calcium and magnesium present in other foods, form 'oxalate salts' which are poorly absorbable. [2,13]. A 'safe' dose of oxalate consumption has yet to be determined and is likely to vary among individuals [3]. Dehydration and fasting are recognized predisposing factors for hyperoxaluria, both of which were present in our patient [6,13].

Oxalate is eliminated almost exclusively by the kidneys and diuresis is known to promote urinary oxalate excretion in individuals with normal renal function. In hyperoxaluria, the crystals are thought to cause direct nephrotoxicity to renal tubular epithelial cells by inhibition of cell proliferation and by apoptosis . They are also thought to stimulate specific genes in renal tubular cells including the connective tissue growth factor gene, leading to interstitial fibrosis [3,9]. In renal biopsies, oxalate as a cause of acute or chronic nephropathy is diagnosed by identifying the characteristic intratubular polarizable crystals.

Conclusion

In Sri Lanka and other tropical countries where the bilimbi fruit is regularly consumed, identifying its potential as a cause of AON has many implications. As long term hyperoxaluria is known to cause nephrolithiasis, nephrocalcinosis, acute and chronic kidney disease and end stage renal disease, investigating the effects of chronic dietary hyperoxaluria on the kidney and assessing the extent, if any, of renal functional impairment becomes important. Dietary hyperoxaluria may well be an under-recognised cause of renal disease which requires careful epidemiological, biochemical and histologic consideration.

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Competing Interests

None Declared

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