



VITAMIN C INDUCED OXALATE NEPHROPATHY : A RARE CASE REPORT

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ABSTRACT

Oxalate nephropathy induced by high dietary intake of oxalate rich food is rare cause of acute kidney injury and end stage renal disease. We describe 42 year old male with adequate baseline renal function who developed severe AKI after diarrheal episodes of 2 days which was not resolving after volume repletion and hemodialysis. On renal biopsy patient found oxalate crystals and gave history of chronic vitamin C tablet consumption from 2 years. After 2 month Patient's renal function normalize with normal RFT values after stopping vitamin C tablets and conservative management.

KEYWORDS : Oxalate Nephropathy, Vitamin C, Acute Kidney Injury

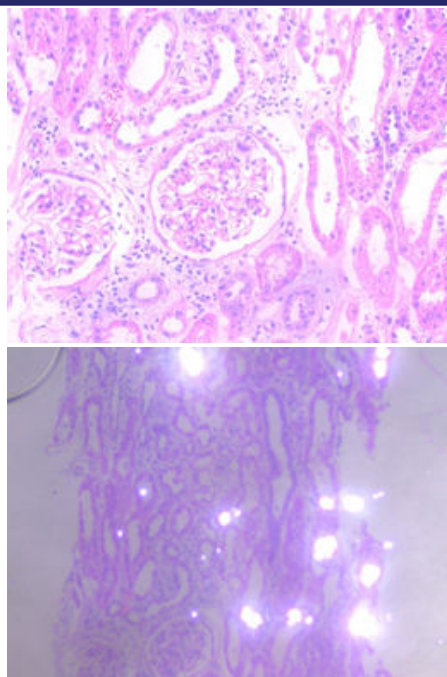
INTRODUCTION

Acute renal failure can occur as a result of acute tubular necrosis secondary to acute oxalate nephropathy due to the deposition of calcium oxalate crystallization within the renal tubules [1]. Oxalate nephropathy can occur in both primary and secondary hyperoxaluria. Primary hyperoxaluria is a group of autosomal recessively inherited enzymatic deficiencies that lead to the increased urinary excretion of oxalate. Secondary hyperoxaluria can occur due to increased dietary oxalate intake, increased absorption of oxalate from the bowel, and increased production of oxalate. Increased dietary intake of oxalate is fairly uncommon; however, there have been case reports in the literature describing excessive star fruit juice ingestion [2,3] and peanut intake [4] leading to oxalate nephropathy. Increased production of oxalate is mainly due to increased levels of oxalate precursors, more commonly glyoxylate, which is associated with ethylene glycol ingestion, and less commonly ascorbic acid [5]. We report, in detail, a case of a patient who presented with this rare occurrence of excessive vitamin C-induced oxalate nephropathy.

Case Report :

42 Year male Health care professional landed up at local emergency with decreased Urine Output(300ml/day) following episode of GI Upset after eating some outside food whose Stool frequency was 4 times/day for 2 days which was watery , no blood in stool, Voluminous. There was No Documented Hypotension / loss of consciousness & patient is Known smoker from 10 years & Non Alcoholic & having no comorbidities. Patient Self medicated with ORS and 2 Dose of OTC oral Antibiotics and ample amount of Oral Fluid for 2 days. Patient had asymptomatic Covid 19 infection 1.5 Year ago. Patients creatinine was 9.2mg/dl and urea was 178mg/dl, Hb was 11.2mg/dl, WBC-5700 & APC-2.1 lac/mm3. Urine RE- +1 Protein & 10-15 RBC/HPF, 1-2 Pus cell/HPF. Autoimmune profile was negative, LDH was in normal range, Procalcitonin was negative & USG suggested normal sized kidney with normal cortical echo & normal Cortico-medullary differentiation. Patient Started on HD through right IJV. After 10 days patients UO improved to 2000ml/day but creatinine was in rising titer on holding HD. After 17 days patient done renal biopsy which suggest Oxalate crystals & Acute Tubular Necrosis

On repeated asking patient gave history of Ingestion of vitamin C tablets daily of 1 gram from 1.5 years. Patient was given HD for 25 days along with stoppage of vitamin C tablets & discharged with 2-2.5L UO/day & 2.5mg/dl creatinine which reduced to 1.0mg/dl in follow up.

**DISCUSSION :**

As described by Ralli et al. in the 1930s, vitamin C is excreted by the kidneys by filtration and active tubular reabsorption. The metabolism of vitamin C to oxalate was later described in the 1960s by other investigators. Since then, there have been several case reports in the literature mentioning acute oxalate nephropathy in association with excessive vitamin C intake. Wong et al. reports a patient with metastatic carcinoma of the prostate with underlying obstructive renal insufficiency who received a 60 gm bolus of IV vitamin C as an alternative therapy and developed anuric renal failure. Two days later on renal biopsy, the patient had similar findings to our patient.

Lawton et al. also describes a patient with similar findings after a single administration of 45 gm of IV ascorbic acid as adjuvant therapy for primary amyloidosis with nephrotic syndrome.

CONCLUSIONS

Use of vitamin C must be scrutinized closely both in patients with normal renal function and with underlying renal insufficiency. Although vitamin C induced hyperoxaluria is not as common as ingestion of ethylene glycol leading to oxalate

nephropathy, clinicians must consider the potential dangers of large dose ingestion of vitamin C in some individuals, especially those with underlying kidney disease or advanced age or precipitating condition

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