
CASE REPORT**A case of oxalate nephropathy presenting with acute kidney injury***Pradnya Mukund Diggikar¹, Mayank Mundada^{1*}, Raju Hansini Reddy¹, Tushar Pancholi¹**¹Department of General Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune -411018 (Maharashtra), India*

Abstract

Oxalate Nephropathy (ON) represents a serious condition characterized by a decline in renal function associated with calcium oxalate crystal deposition within renal tubules. It can arise from Primary Hyperoxaluria (PH) due to genetic defects or secondary causes such as increased dietary oxalate intake. We present a case of a 46-year-old male with facial puffiness, abdominal pain, and pedal oedema, diagnosed with acute kidney injury superimposed on probable chronic kidney disease. Diagnostic investigations revealed bilateral renal echogenicity and severe acute tubular injury with calcium oxalate crystal deposition on renal biopsy. A 24-hour urinary oxalate analysis confirmed ON. Despite the absence of identifiable risk factors, the patient was categorized as probable primary oxalate nephropathy, pending genetic testing results. Management included thrice-weekly haemodialysis and assessment for liver-kidney transplant. Early identification and intervention are crucial in ON to prevent progression to renal failure, emphasizing the need for heightened clinical suspicion and timely management strategies.

Keywords: Oxalate Nephropathy, Primary Hyperoxaluria, Liver-Kidney Transplant

Introduction

Oxalate Nephropathy (ON) is characterized by renal function decline due to calcium oxalate crystal accumulation in renal tubules, often leading to acute tubular injury and interstitial nephritis or fibrosis [1]. ON can be primary, from genetic glyoxylate pathway defects causing reduced oxalate metabolism, or secondary, due to factors like intestinal absorption or dietary oxalate intake [2]. Reports indicate occurrences of ON after ingestion of medicinal herbs like rhubarb and star fruit [3]. Hyperoxaluria from primary or secondary factors leads to calcium oxalate crystal formation, contributing to urolithiasis and renal parenchymal deposition, causing acute tubular injury or fibrosis [4]. On average, people consume about 80-130 mg of oxalate per day. Normally, only 5% to 15% of the oxalate through oral consumption gets absorbed by the body. This is because when oxalate is

bound to calcium in the gut, it gets removed in our stools. Also, certain bacteria in our intestines, like *Oxalobacter formigene*, break down oxalate [4]. ON can often cause progressive kidney dysfunction and carries a notable risk of End-Stage Kidney Disease (ESKD) [1].

Case Report

A 46-year-old male presented with a four-day history of facial puffiness and pedal oedema. Concurrently, he reported diffuse, dull aching abdominal pain, persisting for the same duration, coupled with non-projectile vomiting (1-2 episodes per day) accompanied by nausea and food particle content. The patient denied any comorbidities, current medications, any addictions, or previous hospital admissions. Clinical examination revealed a blood pressure of 150/100 mmHg, a pulse rate of

90 bpm, respiratory rate of 16 cpm, and an SPO₂ of 98% on room air. Mild pallor and grade 3 pitting pedal oedema were observed, while systemic examination revealed no abnormalities. Investigations including electrocardiography and chest X-ray

were unremarkable. Abdominal ultrasonography showed bilateral raised renal echogenicity with normal-sized kidneys. Laboratory investigations on presentation have been enumerated in Table 1.

Table 1: Laboratory investigations on presentation

Parameters [normal limit]	Reports on presentation
Haemoglobin (13.2-16.6 g/dl)	10.8 g/dl
Total leucocyte count (4,000-10,000 / μ L)	5600 / μ L
Platelet count (1,50,000-4,10,000 / μ L)	1,60,000 / μ L
Serum urea (17–49 mg/dL)	135 mg/dL
Serum creatinine (0.6–1.35 mg/dL)	11.8 mg/dL
eGFR by CKD-EPI	5 ml/min/1.73m ²
Serum bilirubin (0.2–1.2 mg/dL)	0.7 mg/dL
SGOT (8–48 IU/L)	45 IU/L
SGPT (7–55 IU/L)	60 IU/L
Random blood sugar level (up to 140 mg/dl)	123 mg/dl
Total protein (6.0-8.3 g/dl)	7.2 g/dl
Serum albumin (3.4-5.0 g/dl)	4.0 g/dl
Serum globulin (2.3-3.5 g/dl)	3.2 g/dl
Serum sodium (136-145 mmol/Lt)	131 mmol/Lt
Serum potassium (3.50-5.10 mmol/Lt)	5.8 mmol/Lt
Serum chloride (98-107 mmol/Lt)	91 mmol/Lt
Urine routine microscopy	Protein +2 No crystals, casts, sediments
Serum magnesium (1.8-2.4 mg/dl)	1.8 mg/dl
Serum calcium (8.6-10.2 md/dl)	7.7 mg/dl

Continued...

Parameters [normal limit]	Reports on presentation
Serum phosphorus (2.6-4.7 mg/dl)	6.5 mg/dl
Serum uric acid (3.7-8 mg/dl)	7.4 mg/dl
Parathormone intact molecule (15-65 pg/ml)	292 pg/ml
HbA1C (<5.7%)	5.4%
24-hour urine proteins (<149 mg/24 hours)	312 mg/24 hours
UACR (0-30) µg/mg	1334 µg/mg
UPCR (<0.2)	2.37
C3 complement(90-120 mg/dl) C4 complement (10-40 mg/dl)	100 mg/dl 24 mg/dl
Anti-nuclear antibody by immunofluorescence	Negative
Anti-nuclear antibody by blot (17 antigens)	Negative

eGFR: estimated Glomerular Filtration Rate, CKD-EPI: Chronic Kidney Disease Epidemiology Collaboration, SGOT: Serum Glutamic-Oxaloacetic Transaminase, SGPT: Serum Glutamate Pyruvate Transaminase, HbA1C: Glycosylated Haemoglobin, UACR: Urine Albumin-Creatinine Ratio, UPCR: Urine Protein Creatinine Ratio

Based on the above clinical presentation and laboratory findings, the patient was diagnosed with Acute Kidney Injury (AKI) with probable Rapidly Progressive Renal Failure (RPRF). Subsequently, the patient was considered for haemodialysis in view of grossly deranged renal function tests and hyperkalemia. To investigate the underlying cause of AKI on RPRF, the patient underwent a renal biopsy. Renal biopsy (Figures 1 and 2) unveiled histopathological findings consistent with non-proliferative morphology upon the examination of glomeruli. Notably, severe acute tubular injury was observed, accompanied by several foci of crystal deposition, indicative of calcium oxalate (likely). The presence of these histological features aligned with the clinical suspicion of oxalate nephropathy as the underlying cause of the AKI on RPRF.

Following the renal biopsy report, a 24-hour urinary oxalate analysis was conducted, revealing a concentration of 46 mg/24 hours (normal range: 7-24 mg/24 hours). Considering these findings, the patient was diagnosed as ON. After this diagnosis, we revisited the history (over consumption of green leafy vegetables, Vitamin C, ethylene glycol, oxalate rich nuts, herbal medications etc.) yielding no relevant information related to the diagnosis. Consequently, the patient was categorized as having probable primary ON. Genetic testing for Primary Hyperoxaluria (PH) was conducted, and the results are awaited. The discharge plan included a scheduled regimen of thrice-weekly maintenance haemodialysis, adherence to follow-up appointments, and simultaneous assessment for liver-kidney transplant.

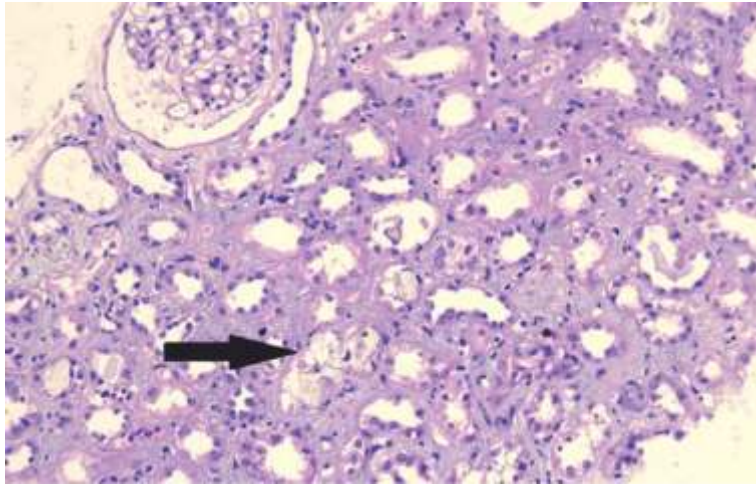


Figure 1: Haematoxylin and Eosin-stained sections of renal biopsy specimen showing fan shaped calcium oxalate crystals within the renal tubules (arrow) at 200× magnification.

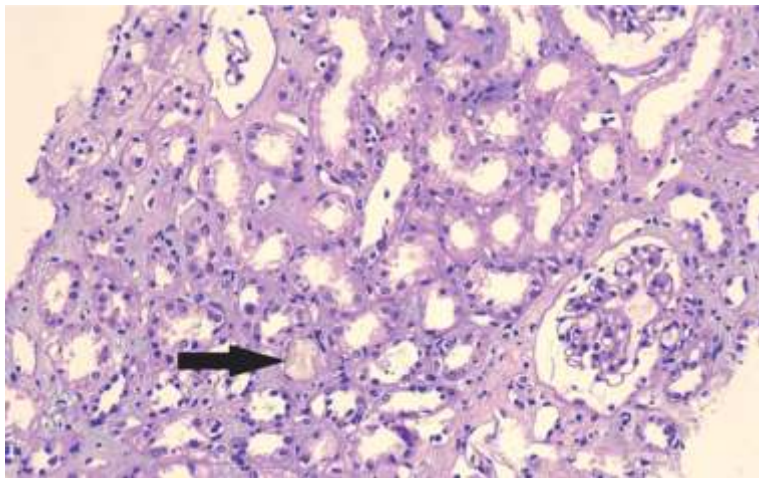


Figure 2: Haematoxylin and Eosin-stained sections from renal biopsy illustrating rhomboid shaped calcium oxalate crystals within the tubules (arrow) along with tubular inflammation and interstitial oedema at 200× magnification

Discussion

PH is an inherited condition characterized by abnormal glyoxylate metabolism, leading to increased oxalate production in the body. There are three distinct hereditary enzymatic deficiencies associated with PH, namely, PH type 1 (PH1), type 2 (PH2), and type 3 (PH3). Among these, PH1 is the most common and severe form [5]. In India and

other developing nations, diagnosing PH remains a significant challenge. The exact incidence and prevalence of this condition remain uncertain, largely due to its diverse range of clinical manifestations and its potential to emerge at any age of life. In a research study [6], it was found that the occurrence of oxalate deposition on kidney biopsy

was 4.07% (25 out of 615 cases), with a primary observation in 88% of instances.

Patients with PH often experience inflammation due to oxalate-induced tubular toxicity, along with nephrocalcinosis and renal obstruction caused by stone formation. These factors are believed to contribute to the progression of Chronic Kidney Disease (CKD) in PH patients [5]. Approximately 10% of patients are diagnosed with PH only upon recurrence of the disease following kidney transplantation [7]. As in our case, at the time of diagnosis, a significant proportion of patients (20 to 50%) present with advanced CKD or even End Stage Renal Disease (ESRD). It is important to note that patients with AKI who advance to CKD face a higher overall risk of developing ESRD. The estimated risk is 13%, but if the patients already had pre-existing CKD, this risk increases to 40% [8]. Persistently elevated excretion, indicated by levels exceeding 0.7 mmol per 1.73 m² body surface area per day or a urinary oxalate: creatinine ratio surpassing the age-specific reference range, alongside suggestive clinical symptoms of PH and the absence of secondary hyperoxaluria, necessitates further assessment. Efforts have been made to define a threshold plasma oxalate level for distinguishing PH from other kidney diseases, although notable overlap exists. Nevertheless, plasma oxalate levels exceeding 50 µmol per liter are indicative of PH [7]. Diagnostic criteria, according to research [4] for oxalate nephropathy include advancing kidney disease, oxalate crystal deposition with tubular injury, interstitial nephritis, and ruling out alternative causes of kidney disease. Additionally, identifying conditions facilitating hyperoxaluria is essential.

As expected, the main intervention in most cases involves intravenous or oral hydration to lower

urinary oxalate levels. Oral citrate is used to prevent crystallization in primary and secondary hyperoxaluria, but data on outcomes are limited. Pyridoxine, lumasiran, and nedosiran have proven effective in treating different PH types based on multiple studies [1]. Health specialists have recommended various non-medical strategies to lower the risk of developing and recurring urinary stones. Their dietary advice focuses on reducing the intake of factors that increase the risk of stone formation, such as calcium, oxalate, salt, and phosphate [9]. Pre-emptive liver transplantation to prevent systemic oxalosis complications before stage 4 CKD appears reasonable but raises ethical concerns due to procedural risks. Combined liver and kidney transplantation is preferred over kidney transplantation alone due to a higher 5-year survival rate of 64% versus 45%. Dual transplantation is advisable for stage 4 CKD due to rapid oxalate retention. Sequential transplantation, starting with the liver, may benefit stage 5 CKD patients by allowing aggressive dialysis and new kidney protection. Experience with organ transplantation in PH type 2 is limited, and type 3 has not led to ESRD [7].

Conclusion

Oxalate nephropathy is a serious condition, potentially more prevalent than previously acknowledged, with a significant risk of progression to renal failure. The imperative lies in maintaining a high index of suspicion, particularly in individuals with unexplained renal dysfunction. Early identification and intervention are pivotal, as renal function at presentation serves as a robust prognostic indicator. Heightened awareness and timely management strategies are paramount to improving outcomes and mitigating the burden of ON in affected individuals.

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