CASE REPORT

Acute oxalate nephropathy following kidney transplantation: Report of three cases

Diana Taheri[1,2], Alaleh Gheissari[3], Pooria Shaabani[4], Seyed Reza Tabibian[4], Mojgan Mortazavi[2], Shiva Seirafian[5], Alireza Merrikhi[3], Mehdi Fesharakizadeh[6], Shahaboddin Dolatkhah[4]

Departments of [1]Pathology and [2]Nephrology, Isfahan Kidney Diseases Research Center, Departments of [3]Pediatric Nephrology and [5]Nephrology, Isfahan University of Medical Sciences, [4]Faculty of Medicine, Isfahan University of Medical Sciences, [6]Department of Surgery, School of Medicine, Islamic Azad University, Najaf Abad Branch, Isfahan, Iran

Calcium oxalate (CaOx) crystal deposition is a common finding immediately after kidney transplantation. However, small depositions of CaOx could be benign while extensive depositions lead to poor graft outcome. Here we report three cases with end-stage renal disease (ESRD), bilateral nephrolithiasis, and unknown diagnosis of primary hyperoxaluria (PH) who underwent a renal transplant and experienced an early-onset graft failure. Although an acute rejection was suspected, renal allograft biopsies and subsequent allograft nephrectomies showed extensive CaOx deposition, which raised a suspicion of PH. Even though increased urinary excretion of CaOx was found in all patients, this diagnosis could be confirmed with further tests including genetic study and metabolic assay. In conclusion, massive CaOx deposition in kidney allograft is an important cause of poor allograft survival and needs special management. Furthermore, our cases suggest patients with ESRD and a history of nephrolithiasis should be screened for elevated urinary oxalate excretion and rule out of PH.

Key words: Acute oxalate nephropathy, calcium oxalate deposition, kidney transplantation, nephrocalcinosis, primary hyperoxaluria

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INTRODUCTION

Oxalate (Ox) is produced as an end product in metabolic reactions, but it must be excreted through glomerular filtration by kidneys.[1] Recently, calcium Ox (CaOx) deposition has been considered as a cause of early allograft dysfunction. Acute Ox nephropathy (AON) is recognized by CaOx deposition in allograft, high serum Ox level, and massive deposition within renal parenchyma.[2,3] One of the causes of CaOx deposition in renal allograft is primary hyperoxaluria type 1 (PH1) which is a rare inborn metabolic disorder with autosomal recessive inheritance.[4] It is the most common form of primary hyperoxaluria (PH),[5,6] and is caused due to absolute or functional deficiency or mistargeting of peroxisomal alanine: Glyoxylate aminotransferase (AGT) in hepatic cells.[7,8]

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This deficiency results in oxidation of enhanced amounts of glyoxylate to poorly soluble Ox.[5,8] Hyperproduction and urinary excretion of Ox lead to recurrent urolithiasis, nephrocalcinosis, and finally to early end-stage renal disease (ESRD).[6,9,10] The disease has an approximate prevalence ranging between 1 and 3 per million populations and an approximate incidence rate of 1:100,000 live births per year in Europe.[11]

PH is a heterogeneous disease presented in five categories:

1. Infantile form with early nephrocalcinosis and progression to end-stage renal failure (ESRF);
2. Juvenile form with abdominal pain secondary to recurrent urolithiasis or nephrocalcinosis and progressive renal failure leading to diagnosis of PH1 in adolescence;
3. Adult or late-onset form with occasional stone passage in adulthood;

Address for correspondence: Dr. Seyed Reza Tabibian, Faculty of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran.
E-mail: srt_1371@yahoo.com
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4. Diagnosis given after transplantation; and
5. Presymptomatic subjects with family history of PH1.[7,12,13]

CASE REPORTS

Case 1

A 24-year-old man was admitted to our hospital with the signs of advanced chronic renal failure (CRF) in 2006. In his past history, he had developed kidney and bladder stones since he was 5-year-old. In his family history, his sister exhibited an elevated urine Ox concentration and his brother presented with kidney stones and renal failure. Unfortunately, they refused to cooperate with physicians to perform required tests until transplantation. Immediately in 1st day of admission, blood urea nitrogen (BUN) and serum creatinine (Cr) were determined 165 and 16.5 mg/dl, respectively, and also protein and glucose were detected in urine. The diagnostic findings indicate shrunken hyper-echogenic kidneys with hydronephrosis. Multiple stones with different sizes were observed in calyces of both kidneys. The bladder wall was thickened (4.7 mm), and urinalysis did not reveal any crystals. The patient underwent hemodialysis (HD) three times per week for 4 months. Four months after admission, the patient underwent bilateral nephrectomy. Based on the findings obtained in bilateral radical nephrectomy, chronic pyelonephritis with nephrolithiasis was confirmed. Microscopic examination revealed glomerulosclerosis and tubulointerstitial fibrosis. About 2 years later (September 2008), the patient underwent living kidney transplantation with a diagnosis of ESRD. The primary graft function was acceptable? but 3 days after transplantation, the urine output suddenly dropped to 30-50 cc a day. Therefore, a renal biopsy was taken. Tissue sections containing glomeruli and tubules were obtained. The following histopathological data were gathered: The glomeruli showed no conspicuous changes, mild to moderate atrophy with mononuclear inflammatory cells and occasional eosinophilic infiltration of interstitium, some tubules contained marked Ox crystals, and some contained granular and white blood cells casts. Following the acute rejection, anti-thymocyte globulin (ATG) was administered for the patient. After few days of ATG administration, the drug was discontinued because of fever. The possibility of PH1 was considered when a lot of Ox crystals were observed in the renal allograft biopsy and also by ruling out the other causes of hyperoxaluria. Therefore, pyridoxine therapy was commenced (dose, interval, and route of admin).

In January 2009, the patient's serum Cr and BUN levels were 4.8 and 38 mg/dl, respectively. The patient's computed tomography (CT) scan and radiography demonstrated cardiomegaly, bilateral pleural effusion with congestion, and calcification at the right pulmonary hilum and parenchyma. The molecular diagnostic polymerase chain reaction test revealed the persistence of cytomegalovirus. After few months, the patient refused to continue the treatment and finally he died 1-year later.

Case 2

A 13-year-old boy was admitted to our hospital in July 2008 with renal stones in superior and inferior calyces and pelvis. Bilateral nephrocalcinosis and decreased corticomedullary contrast was observed. Furthermore, both kidneys had smaller sizes than normal. Hence, percutaneous nephrolithotomy was conducted. In his past medical history, he had urolithiasis and had undergone extracorporeal shock wave lithotripsy twice to remove the Ox stones in his childhood. When he was eight, due to the bilateral kidney stones, polycitrate, and vitamin B6 therapy was initiated for him and consequently the serum Cr decreased considerably. His parents were cousins but none of them had renal failure and his aunt had renal stones. When he was 11, he was admitted to the hospital again. Ultrasonography of urinary system revealed multiple bilateral renal stones and small right kidney with signs of diffused bilateral renal parenchyma disease. Hydronephrosis was obscured by the shadow of stones. The laboratory tests showed the values of BUN, blood Cr, urine Cr, urine Ox, and urine volume to be 26.6 mg%, 1.1 mg%, 775 mg/24 h, 1240 mg/24 h, and 3100 ml/24 h, respectively. The patient also had ureter stone. Since Cr level raised and glomerular filtration rate (GFR) decreased in the following, HD was performed. Due to unavailability of definite diagnostic tests for PH1 in our center and also the normal ranges of 24 h urine Ox in frequent tests; isolated renal transplantation was performed due to the clinical features for management of his unknown primary disease. Before transplantation, BUN and Cr levels were 168 and 10.1 mg/dl, respectively. The 24 h urine Cr level was 1.7 mg/kg/24 h. The hematocrite was 31.9% and red blood cells showed poikilocytosis and anisocytosis. In September 2010, kidney transplantation was conducted. After transplantation, lymphedema of lower limb and scrotum occurred. Ten days after transplantation, Cr level again increased to 7.5 mg/dl. Renal allograft biopsy performed on day 12, posttransplantation showed acute tubular necrosis (ATN). The microscopic findings of graft biopsy on day 35 after transplantation were nonsignificant pathological finding of glomeruli, while interstitium showed diffused moderate edema, mild patchy mononuclear and occasional polymorphism infiltration, and some scattered CaOx crystals. Renal allograft biopsy on day 43, posttransplantation showed extensive deposition of CaOx; suggestive of PH recurrence. The patient's clinical manifestations were severe anemia, increased serum Cr, and hypertension. Plasmapheresis performed for two times a day for first 3 days of treatment and then daily plasmapheresis to

reverse severe hemolytic uremic syndrome signs (normalize lactate dehydrogenase levels, decrease the percentage of fragmented red blood cells, and increase platelet count) and also to prevent allograft rejection. However, considering the patient's clinical features, nephrectomy was performed. In the specimen sections obtained, the tubules contained extensive CaOx depositions; suggestive of primary disease (PH1) recurrence. The perfusion lung scintigraphy performed 22 days after nephrectomy was suggestive of the pulmonary thromboembolic disorder. The serum urea and Cr levels were 82 and 3.3 mg/dl, respectively. He is now on HD and peritoneal dialysis (PD) and he is the candidate for the measurement of AGT catalytic and immunoreactivity in a liver biopsy specimen before combined liver-kidney transplantation.

Case 3

A 15-year-old girl, who was suffered from ESRD since she was thirteen, was admitted to our hospital with dyspnea, chest pain, cellulitis, and osteomyelitis. Two months after admission, the patient had an operation with the diagnosis of septic knee arthritis. In the sonography performed 1 month later, renal stones were observed. The patient underwent HD and PD. Two days before transplantation, the BUN and Cr levels were 73 and 5.9 mg/dl, respectively. Before the operation, the patient had received methylprednisolone and vitamin B6. She underwent kidney transplantation from an independent living donor. Two days after transplantation, BUN and Cr levels were 24 and 1.4 mg/dl, respectively. The patient developed dyspnea and severe ascites and the urine volume decreased. Five days after transplantation, BUN and Cr levels increased (66 and 4 mg/dl, respectively). The leukocyte count also increased. Furthermore, renal transplant scintigraphy showed mild increased perfusion with reduced function of transplanted kidney with the evidence of dilated ureter (ureterovesical junction stenosis). Therefore, core needle biopsy of the transplanted kidney was done. Histopathologic view of the biopsy showed nonsignificant changes in glomeruli, moderate infiltration of lymphocytes accompanying edema in the interstitium, and marked Ox crystal deposition in some tubules and endarteritis consistent with type II acute T-cell-mediated rejection. Two months after transplantation, she developed avascular necrosis of the head of the femur. Finally, 9 months after transplantation, the nephrectomy was conducted due to graft failure. Renal transplant nephrectomy revealed excessive CaOx deposition. Subsequently, the peritoneal catheter was implanted which was functional for 1 month. At the end, she was again admitted with hyperkalemia and the patient died [Figures 1 and 2].

DISCUSSION

AON could be considered as a cause of early graft dysfunction and acute tubular injury. It is defined as extensive Ox deposition in renal tubules and parenchyma, which can damage tubular epithelial cells, promote tubulointerstitial injury and necrosis, and finally provoke renal dysfunction. Deposition of CaOx may develop in different clinical contexts:

1. Early posttransplant accumulation of CaOx, which is usually characterized by few foci of CaOx and background of acute rejection and ATN;
2. CRF, which is usually characterized by few foci of CaOx and background of chronic allograft nephropathy;
3. Recurrent PH, which is characterized by extensive renal deposition of CaOx and chronic tubulointerstitial injury; and is associated with graft failure; and
4. Secondary hyperoxaluria due to elevated enteric absorption of Ox or drugs that contain Ox.[2-4,14]

The approach for PH1 diagnosis consists of five steps including measurement of urinary Ox, calculation of urinary Ox over Cr ratio in screening children, determination

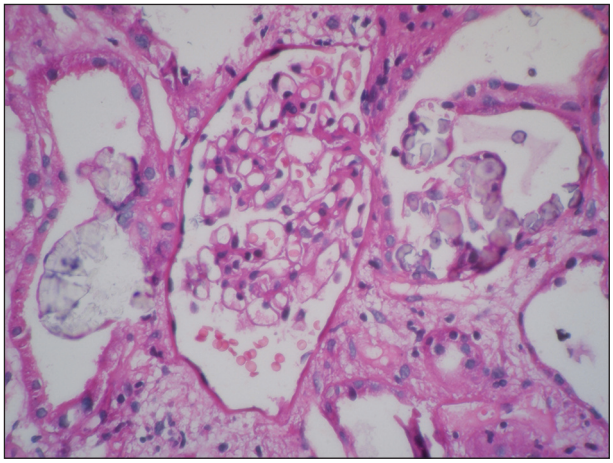

Figure 1: Renal allograft biopsy. Renal tubule is filled with oxalate crystals (H and E, ×400)

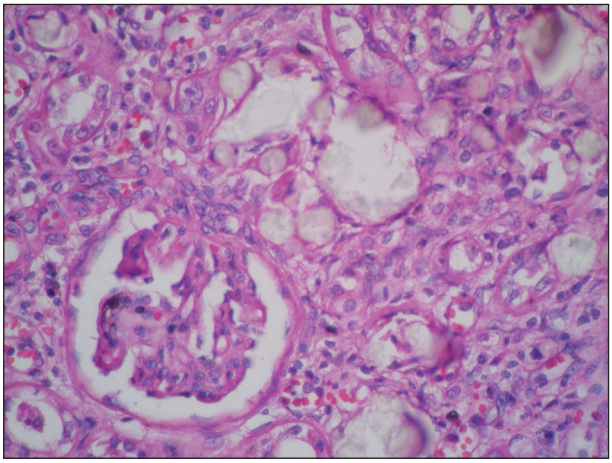

Figure 2: Transplant nephrectomy. Renal tubules are filled with oxalate crystals (H and E, ×400)

of plasma Ox, liver biopsy for enzymatic analysis, and deoxyribonucleic acid (DNA) analysis. Plasma Ox level is determined when the values of urinary Ox are falsely low in patients with renal impairment due to Ox retention and systemic deposition as CaOx; and the normal value are in the range of 0.5-7.5 umol/l. Liver biopsy for enzymatic analysis is required if liver transplantation is considered and DNA analysis is used only in populations having a high frequency for a specific mutation.[15] So far, more than 60 mutations (mainly point missense or nonsense mutations) and polymorphisms have been identified and it has been shown that selective exon sequencing allow a definitive diagnosis in half patients within 2 weeks.[16,17]

There are few reports in the literature presented the AON cases with different causes. Spasovski *et al*. reported a 48-year-old female with CRF. Her clinical manifestations were a bilateral flank pain, hyper-echogenic renal parenchyma, and multiple small stones in the calyces. The patient underwent transplantation and kidney biopsy performed 4 months posttransplantation showed calcifications (oxalosis). The PH1 diagnosis was established just after graft failure and confirmed by mutational analysis of the AGXT gene.[9] Shang *et al*. reported a 14-year-old girl who had recurrent kidney stones for 7 years. She received a living kidney transplant from her mother. Seven months after transplantation, second allograft biopsy revealed extensive CaOx crystals in the tubule lumens, but there was no evidence of chronic or acute rejection. The case was diagnosed as allograft Ox nephropathy. The possibility of PH1 was considered by reviewing the findings of the patient's X-ray before transplantation, which revealed diffuse bony changes that were ignored, abdominal plain radiography, nonenhanced CT scan, and a plain radiography of pelvic bone.[12] Malakoutian *et al*. reported a 22-year-old patient with kidney failure, who was received kidney transplantation from a living donor. Swelling of transplanted kidney with multiple echogenic shadows in both native kidneys were indicative for calculus; increased the suspicion of metabolic cause for recurrent nephrolithiasis. Percutaneous kidney allograft biopsy was performed. There were large refractile, acellular deposits in several tubular lumens resembling Ox crystals that caused tubular destruction and injury. The final diagnosis was Ox crystal nephropathy. The genetic study revealed a mutation in exon 5, leading to substitution of thymidine in place of guanine at nucleotide 584 that confirmed the diagnosis of PH1.[5] Parasuraman *et al*. reported a 49-year-old male with ESRD secondary to autosomal dominant polycystic kidney disease. Posttransplant, the patient developed delayed graft function. CaOx deposition with no evidence of rejection was observed in the biopsy. In the view of CaOx deposition, clinical evaluations for the underlying causes of secondary hyperoxalosis were performed. Potential causes of secondary hyperoxalosis could be enteric hyperoxalosis, MMF-induced diarrhea, excessive oral intake of Ox-containing foods as vitamin C, ethylene glycol intoxication, or the use of methoxyflurane. The evaluation results were negative. Finally, nephrectomy was performed with the diagnosis of primary nonfunction of renal allograft. The diagnosis of AON was made based on CaOx deposition in allograft, high serum Ox level, and CaOx deposition in collecting tubules in the absence of other reasons for allograft dysfunction (2).

CaOx deposition may commonly be observed in allograft biopsy and has different manifestations. PH1 recurs immediately after transplantation with massive CaOx deposition associated with acute and chronic renal tissue injury that causes graft failure. Usually, after renal transplantation, excessive Ox will be observed in urine and elevated plasma Ox level decreases to normal values between 3 days and 3 weeks posttransplantation. This will be observed in all the patients except those suffering from PH1. Recurrent nephrolithiasis and nephrocalcinosis in childhood and its exposition after transplantation are distinguished by massive CaOx deposition. This leads to chronic injury of tubules and parenchyma that finally causes renal dysfunction and allograft failure, which is not reversible and specifies PH1 from other AON causes.[2,4,12,14] Another presentation of PH1 that differentiates it from the other causes of ESRD is that the bone Ox content among PH1 patients is higher than that in ESRD patients without PH1 (15-910 vs. 2-9 mmol Ox/g bony tissue).[18] The clinical manifestation of PH1 varies dramatically even among the family members who have the same AGXT mutations. The presentation could be in a severe form in some patients while other could suffer from an asymptomatic disease for a long time with normal urinary excretion.[9] Due to the scarcity of the disease and insufficient information on inherited urolithiasis, the disease in some patients is diagnosed when the patient has reached ESRF. For instance, in a Dutch study, 59% of the patients older than 18 years were had ESRF at the time of PH1 diagnosis.[6,15]

The suspicion of infantile form of PH1 could be considered as a cause of AON in our second and third cases and the first case is considered to be the juvenile form. Measurement of plasma Ox is not performed in our hospital. Moreover, liver biopsy for enzymatic analysis and DNA analysis are not commonly available and are very expensive. Hence, the PH1 diagnosis was not confirmed for our patients, as other causes of AON could not be excluded completely. For example, secondary hyperoxaluria, which is increasingly recognized in the recent years, could not be ruled out. Fecal excretion of Ox depends on its binding to calcium in the intestine. The most common mechanism that leads to enteric hyperoxalosis is that intestinal fat malabsorption decreases

the availability of intestinal calcium; thus, Ox absorption will be increased. Fat malabsorption due to pancreatic calcification causes AON with consequent loss of the first allograft.[19] Rankin *et al*. reported a 70-year-old male in the UK who was developed diabetes mellitus secondary to idiopathic chronic pancreatitis progressed to ESRD. AON diagnosis secondary to enteric hyperoxaluria was made in the patient based on intraluminal CaOx crystals. The patient quitted taking pancreatic supplements as there was no evidence-based on supplemental vitamin C and other Ox precursors or calcium salt usage in his past medical history.[14]

CONCLUSION

The three cases reported here reiterates the importance of evaluation patients with acute graft dysfunction for all causes. AON is an important cause, which requires an appropriate attention and all the causes of AON must be considered. PH1 is one of the causes of AON. Of our three cases, two patients died due to complications of their renal insufficiency and the other one traveled abroad for his treatment. In patients with normal or significant residual GFR, concomitant hyperoxaluria (urine Ox >1 mmol/1.73 mBSA per day, normal <0.5), and hyperglycoluria (urine glycolate >0.5 mmol/1.73 m^2) are indicative of PH1. However, some patients do not present with hyperglycoluria. In children with ESRD, plasma Ox:Cr ratio and Ox measurement in dialysate might be helpful for screening of PH1.[20] Normal values (mmol/mol) of plasma Ox: Cr ratio is <300 for age <2 years, <130 for age 2-5 years, <70 for age 5-15 years, and <40 for age >16 years. However, such ratios need to be confirmed by complete urine collection. Urinary Ox excretion is falsely low in patients with decreased GFR owing to Ox retention and systemic deposition as CaOx.[15] Because our cases admitted with progressive renal failure and ESRD, we could not measure the 24 h Ox urine. As it was mentioned, plasma Ox measurement was not available; so, we could not diagnose the cases as PH1 due to the lack of definite diagnostic tests. However, their clinical data and also rule out of other PH causes strongly suggested the PH1 diagnosis. Based on the low age of our three cases and the past history of nephrolithiasis and nephrocalcinosis in their childhood; the possibility of PH1 as the cause of AON drastically raised. In this regard, the physicians and nephrologists should consider the diagnosis of PH1 before kidney transplantation; especially, in patients with nephrolithiasis during their childhood. However, other causes of AON could not be overlooked and the additional tests and clinical examinations must be conducted to confirm the diagnosis.

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Conflicts of interest
There are no conflicts of interest.

AUTHOR'S CONTRIBUTIONS

SRT contributed in the conception of the work, conducting the study, revising the draft, approval of the final version of the manuscript, and agreedfor all aspects of the work MM contributed in the conception of the work, drafting and revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work. PSH contributed in the conception of the work, conducting the study, revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work DT contributed in the conception of the work, revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work. AGH contributed in the conception of the work, conducting the study, revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work. AM contributed in the conception of the work, revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work. MF contributed in the conception of the work, revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work. SHD contributed in the design of the work, revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work.

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