Brazilian Journal of TRANSPLANTATION

https://doi.org/10.53855/bjt.v29i1.794_ENG

CASE REPORT

Late Diagnosis of Primary Hyperoxaluria Type 1 After Kidney Transplant

Sabrina Maria Araujo Sobreira[1*], Beatriz de Oliveira Neri[1], Vanessa Gurgel Adeodato[1], Arthur Holanda Dantas[1], Sebastiao Alves Sobreira Neto[1], André Costa Teixeira[1], Ronaldo de Matos Esmeraldo[1]

1.Hospital Geral de Fortaleza – Fortaleza (CE) – Brazil.

*Corresponding author: sabrina_sobreira@hotmail.com

Seccion editor: Ilka de Fátima Santana F. Boin

Received: Feb. 2, 2026 | Approved: Mar. 3, 2026

ABSTRACT

Primary hyperoxaluria type 1 is a rare and often unrecognized cause of renal allograft dysfunction and loss, particularly when renal disease is attributed to recurrent nephrolithiasis. We report the case of a 62-year-old woman with a history of bilateral nephrolithiasis and prolonged use of anti-inflammatory drugs who underwent kidney transplantation with immediate graft function and an initially favorable course. After an extended period of stability, late and progressive graft dysfunction occurred. Allograft biopsy demonstrated tubular injury associated with extensive calcium oxalate crystal deposition, suggesting oxalosis. Serum oxalate was 18.6 µmol/L, and urinary oxalate was 94.9 mg/24 h. Genotyping identified a homozygous pathogenic variant in the AGXT gene, confirming the diagnosis of primary hyperoxaluria type 1. Review of the medical history revealed nephrocalcinosis in native kidneys, and family screening identified a sibling with nephrolithiasis. Pharmacological-dose pyridoxine was initiated as first-line therapy based on the responsive genotype, followed by RNA interference therapy with lumasiran due to the high risk of graft loss, resulting in initial stabilization of graft function. This report emphasizes that primary hyperoxaluria type 1 should be considered in transplant recipients with a history of nephrolithiasis, even in adulthood, and that post-transplant identification has direct implications for graft prognosis, therapeutic approach, and family screening, highlighting the need for metabolic and genetic investigation in selected cases before transplantation.

Descriptors: Primary Hyperoxaluria; Kidney Transplantation; Nephrolithiasis; Kidney Failure, Chronic.

Diagnóstico Tardio de Hiperoxalúria Primária Tipo 1 Após Transplante Renal

RESUMO

A hiperoxalúria primária do tipo 1 é uma causa rara e frequentemente não reconhecida de disfunção e perda do enxerto renal, sobretudo quando a doença renal é atribuída à nefrolitíase recorrente. Relata-se o caso de uma mulher de 62 anos, com história de litíase bilateral e uso prolongado de anti-inflamatórios, submetida a transplante renal com função imediata e evolução inicial favorável. Após período prolongado de estabilidade, ocorreu disfunção tardia e progressiva do enxerto. A biópsia do enxerto demonstrou lesão tubular associada a depósitos intensos de cristais de oxalato de cálcio, sugerindo oxalose. A pesquisa do oxalato sérico foi de 18,6 µmol/L, e o urinário, de 94,9 mg/24 h. Estudo de genotipagem identificou variante patogênica em homozigose no gene AGXT, confirmando diagnóstico de hiperoxalúria primária tipo 1. A revisão da história médica evidenciou nefrocalcinose em rins nativos, e o rastreamento familiar permitiu identificar um irmão com nefrolitíase. Instituiu-se piridoxina em dose farmacológica como primeira linha terapêutica, com base no genótipo responsivo, seguida de terapia de interferência de RNA com lumasiran, diante do alto risco ao enxerto, com estabilização inicial da função do órgão transplantado. Este relato reforça que a hiperoxalúria primária do tipo 1 deve ser considerada em receptores com histórico de nefrolitíase, mesmo em idade adulta, e que a identificação pós-transplante tem implicações diretas no prognóstico do enxerto, na abordagem terapêutica e no rastreamento familiar, destacando a necessidade de investigação metabólica e genética em casos selecionados antes do transplante.

Descritores: Hiperoxalúria Primária; Transplante de Rim; Nefrolitíase; Falência Renal Crônica.

INTRODUCTION

Primary hyperoxaluria type 1 (PH1) is a rare, autosomal recessive metabolic disorder caused by a deficiency of the peroxisomal hepatic enzyme alanine-glyoxylate aminotransferase (AGT)[1]. This enzymatic defect results in the overproduction of oxalate, which, when combined with calcium, forms insoluble calcium oxalate (CaOx) crystals that are deposited in the renal parenchyma (nephrocalcinosis) and in the collecting system (urolithiasis)[1]. Although the clinical presentation is heterogeneous, PH1 frequently progresses to stage 5 chronic kidney disease (CKD)[2]. In many cases, the absence of systematic metabolic investigation in patients with recurrent kidney stones means that the etiology of renal failure remains undetermined until the time of transplantation[2]. In Brazil, late diagnosis is a challenge, especially when progression to dialysis occurs in adulthood, mimicking other common kidney diseases[2].

The contemporary relevance of this topic is amplified by the introduction of RNA interference (RNAi) therapies, such as lumasiran, which reduce hepatic production and urinary excretion of oxalate, modifying the natural course of the disease and opening the possibility of isolated kidney transplantation instead of combined liver-kidney transplantation in many cases[3-6]. This advancement is particularly important in the context of organ shortages for transplantation, making this pharmacological alternative potentially beneficial for both the individual and the healthcare system, thus highlighting the relevance of pharmacological alternatives in public health[7].

National case reports are fundamental for documenting the Brazilian experience with PH1, the use of new technologies such as RNAi, and kidney transplantation, contributing to the consolidation of local evidence and to the discussion on the incorporation of these therapies into the health system. In the context of transplantation, PH1 that is not recognized early represents a critical risk, as it can lead to oxalate deposition in the graft and accelerated loss of renal function, in addition to the inevitable recurrence after isolated kidney transplantation[8,9].

The authors report the case of a 62-year-old patient with a late diagnosis of PH1 in the post-renal transplant context, with laboratory confirmation in 24-hour urine, histopathological and genetic testing, and direct implications for graft management. Furthermore, screening first-degree relatives identified an affected family member, enabling earlier intervention and genetic counseling. By documenting an atypical presentation in an adult and the use of specific therapies in the national setting, this report contributes to the discussion on diagnostic and therapeutic strategies in renal transplant recipients with a history of recurrent kidney stones.

Ethical approval and consent

This study was approved by the Research Ethics Committee of the General Hospital of Fortaleza, under opinion number 8.149.811 (CAAE 95115826.1.0000.5040).

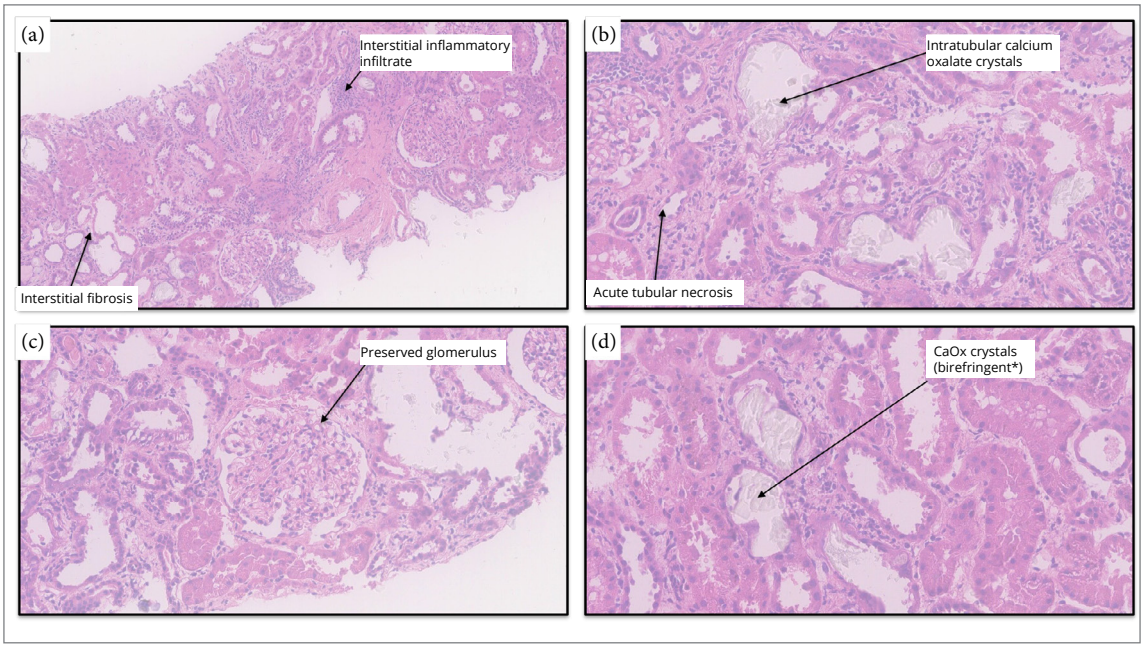
The patient described in this report provided written informed consent for the publication of her clinical information and the results presented. All data were anonymized to preserve the patient's confidentiality and privacy.

Case report

A 62-year-old female patient with a history of bilateral nephrolithiasis and prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs). She had a history of hepatitis C virus infection, previously treated with direct-acting antivirals, achieving a sustained virological response, and without clinical evidence of chronic liver disease. She progressed to stage 5 CKD, starting hemodialysis (HD) in September 2016. Her panel-reactive antibody (PRA) was 12.5% for Class I and 0% for Class II. She underwent a kidney transplant in 2019 from a 32-year-old deceased male donor (cause of death: traumatic brain injury). The donor's serum creatinine was 0.7 mg/dL (initial) and 1.6 mg/dL (final), with three HLA mismatches (1 in A, 1 in B, and 1 in DR). Immunosuppressive induction was performed with anti-thymocyte globulin (4.5 mg/kg), and maintenance therapy consisted of tacrolimus and sirolimus.

On the first postoperative day, the graft's Doppler ultrasound showed good intrarenal perfusion, patent vessels, and no hydronephrosis or calculi, with intrarenal resistivity indices ranging from 0.71 to 0.78. The graft functioned immediately, with hospital discharge on the fifth postoperative day and a baseline creatinine of 1.0-1.2 mg/dL.

After 14 months of stable graft function, the patient developed progressive dysfunction of the transplanted kidney, with a serum creatinine of 2.8 mg/dL, while remaining asymptomatic. A graft biopsy was performed (Fig. 1), which showed no signs of acute rejection but revealed acute tubular necrosis with abundant deposition of CaOx crystals within the tubules, along with interstitial fibrosis and tubular atrophy involving approximately 20% of the sampled parenchyma. Assessment according to the Banff Classification (2018) showed mild interstitial inflammation and absence of tubulitis (i1/t0), with mild interstitial fibrosis and tubular atrophy (ci1/ct1), and no evidence of glomerulitis, peritubular capillaritis, or vasculitis (g0/ptc0/v0). Focal minimal C4d staining was observed (C4d1, in about 10% of peritubular capillaries), and SV40 testing was negative, ruling out polyomavirus nephropathy.



Source: Elaborated by the authors

Figure 1. Histopathological findings (optical microscopy) of renal oxalosis. a) Panoramic view demonstrating interstitial inflammatory infiltrate of mononuclear pattern and areas of interstitial fibrosis, indicating chronicity of the process. b) CaOx crystals inside the tubular lumens with characteristic "envelope" morphology, associated with acute tubular necrosis, evidenced by epithelial flattening. c) Glomerulus with preserved architecture, demonstrating the predominantly tubulointerstitial pattern of renal lesion in hyperoxaluria, with CaOx crystals in adjacent tubules. d) Image obtained under polarized light, showing CaOx crystals with typical birefringence*. Staining: hematoxylin-eosin.

The diagnostic hypothesis of PH1 was raised after detecting significant hyperoxaluria (94.9 mg/24 h), a value well above the reference range for women (4-31 mg/24 h), and the presence of nephrocalcinosis in native kidneys, evidenced by tomography. Serum oxalate, measured by spectrophotometry, was 18.61 µmol/L (reference value: < 26.6 µmol/L), remaining within the normal range, despite the patient having a GFR of 19 mL/min/1.73 m² – a context in which an elevated plasma level would be expected. This apparently discordant finding may reflect variability in the laboratory method or the timing of sample collection and raises questions about the sensitivity of serum oxalate as an isolated diagnostic tool, even in patients with advanced CKD.

The definitive diagnosis was confirmed by genetic sequencing, which identified the pathogenic variant c.508G>A: p.(Gly170Arg) in homozygosity in the AGXT gene. Following diagnostic confirmation, genetic screening of first-degree relatives was performed. The test confirmed the diagnosis of PH1 in her 57-year-old brother, who carried the same variant in homozygosity and had a history of recurrent nephrolithiasis and stage G3a CKD.

Following confirmation of PH1 by genetic sequencing, the therapeutic approach was guided by the identified genotype. Since the p.Gly170Arg variant is among the genotypes with the highest potential for vitamin B6 responsiveness, pharmacological doses of pyridoxine (120 mg/day; ≈ 2.0 mg/kg/day, based on a body weight of 59 kg) were initiated as first-line therapy for both the patient and her brother in mid-2024. During this initial phase, therapeutic response was monitored using serum creatinine and estimated glomerular filtration rate (eGFR), as technical difficulties with 24-hour urine collection prevented serial oxaluria measurements. Despite continued pyridoxine use, graft function continued to decline. In February 2025, the dose was escalated to 200 mg/day (≈ 3.4 mg/kg/day) for the transplant recipient.

Given the progressive graft dysfunction – with serum creatinine reaching a peak of 4.3 mg/dL – and the active oxalate deposition documented in the biopsy, configuring a high-risk scenario for the graft, RNA interference therapy with lumasiran (3 mg/kg, subcutaneously) was instituted in March 2025 as an intensification to reduce hepatic oxalate production. After 7 months of combined treatment (pyridoxine + lumasiran), renal function stabilized, with serum creatinine remaining around 3.3 mg/dL. In a laboratory reassessment with a 24-hour urine collection, performed in September 2025, oxaluria had decreased from 94.9 to 40.1 mg/24 h (reference value: 4-31 mg/24 h), representing a drop of approximately 58%, accompanied by serum oxalate of 13.9 µmol/L (reference value: < 26.6 µmol/L). Due to administrative obstacles in accessing the drug, treatment with lumasiran was suspended in September 2025.

Given the progression of renal dysfunction and the interruption of RNAi therapy due to access difficulties, the team has been seeking alternatives to restore metabolic control, including new attempts to obtain the drug. Considering the risk of progression of graft dysfunction, the clinical team is evaluating the possibility of combined liver-kidney transplantation as a therapeutic alternative. Lumasiran represents an innovative RNAi-based technology that reduces hepatic oxalate production and, according to evidence from the ILLUMINATE studies, can modify the natural course of the disease and potentially avoid the need for liver transplantation – a particularly relevant aspect in the context of organ shortage. The decision to incorporate this therapy into the Brazilian Unified Health System (Sistema Único de Saúde-SUS) rests with the National Commission for the Incorporation of Technologies (Comissão Nacional de Incorporação de Tecnologias - CONITEC), following criteria of scientific evidence and cost-effectiveness analysis. Determining the best therapeutic strategy for the patient remains under multidisciplinary discussion, taking into account available options and the specific circumstances of the case.

DISCUSSION

PH1 is an inborn error of glyoxylate metabolism, with excessive endogenous oxalate production and deposition of CaOx crystals, culminating in recurrent nephrolithiasis, nephrocalcinosis, and progression to CKD in a significant proportion of patients[1,2]. Although classically described in childhood, late-onset forms are recognized, with diagnosis frequently delayed when nephrolithiasis is interpreted as "idiopathic" or attributed to environmental/medication factors, which can delay metabolic and genetic investigation[1,2]. In this scenario, the case presented exemplifies a particularly relevant outcome in transplant recipients: the identification of the disorder only after late graft dysfunction, when the biopsy revealed massive oxalate deposition and tubular damage, retrospectively changing the interpretation of the etiology of kidney disease and redefining prognosis and therapeutic strategy.

The importance of early diagnosis in PH1 is twofold. First, it allows for the implementation of measures to reduce urinary supersaturation and crystal formation (vigorous hydration, alkalinization/citrate, dietary guidance and, in specific genotypes, pyridoxine), potentially delaying the loss of renal function[1,2]. Secondly – and especially in the context of transplantation – the pre-transplant diagnosis guides the planning of the type of transplant and perioperative management, since kidney transplantation alone does not correct the continuous hepatic production of oxalate[1,8-10]. Thus, when primary hyperoxaluria is not recognized before transplantation, the graft may be exposed to recurrent oxalate deposition, with a risk of tubular injury and accelerated functional decline, which becomes even more critical in the presence of limited functional reserve or additional insults (ischemia-reperfusion, nephrotoxicity, infections)[8].

Post-transplant diagnosis presents specific challenges, as graft dysfunction is often initially attributed to more frequent causes such as acute rejection, calcineurin inhibitor toxicity, infection, or polyomavirus nephropathy[9]. The Kidney Disease: Improving Global Outcomes guidelines emphasize that recurrent disease should be included in the differential diagnosis of graft dysfunction and that the suspected etiology[9] should guide investigations. In this situation, graft biopsy plays a central role: the identification of acute tubular necrosis associated with abundant deposition of CaOx crystals, coupled with the absence of evidence of active rejection and the exclusion of viral infection, strongly supports the hypothesis of oxalic nephropathy as the predominant mechanism of dysfunction[8-10]. It is important to recognize that oxalate in the graft can result from both primary and secondary hyperoxaluria and/or from mobilization of the systemic pool accumulated during dialysis, so that etiological confirmation requires clinical-laboratory correlation and targeted investigation[8,10]. In this case, the history of recurrent nephrolithiasis, evidence of nephrocalcinosis in native kidneys, and sustained hyperoxaluria make primary hyperoxaluria a pathophysiologically plausible hypothesis, later confirmed by genetic testing[1,2,9,10].

A relevant finding in this case was the normal serum oxalate level (18.61 µmol/L; reference value: < 26.6 µmol/L), despite the patient presenting with an eGFR of 19 mL/min/1.73 m^2—a context in which plasma oxalate elevation would be classically expected. Although the literature suggests that serum oxalate tends to rise in patients with a GFR below 30 mL/min/1.73 m^2, reaching levels above 30-50 µmol/L when systemic supersaturation occurs[1,2], this case demonstrates that normal values do not exclude the diagnosis of PH1, even in advanced CKD. This observation may be related to methodological limitations of spectrophotometry, pre-analytical variations, or the fact that the plasma elevation threshold varies between individuals[13]. Therefore, the absence of hyperoxalemia should not discourage genetic investigation when the clinical picture – recurrent nephrolithiasis, nephrocalcinosis, hyperoxaluria, and suggestive histopathological findings – points to PH1.

Genetic confirmation is important for diagnosis and for defining therapeutic management. Identification of the pathogenic variant c.508G>A (p.Gly170Arg) in homozygosity in the AGXT gene establishes the etiological diagnosis. It allows stratification of therapeutic options, including the usefulness of pyridoxine in subgroups with potential for response, as described in some patients with PH1[1,2]. This mutation, the most common in Western populations, is associated with incorrect mitochondrial targeting of the AGT enzyme, yet preserves residual catalytic activity. This molecular phenotype explains the patient's late clinical presentation (stage 5 CKD in the sixth decade) and the potential responsiveness to pyridoxine (vitamin B6), which acts as a chemical chaperone, improving enzymatic stability[1,2].

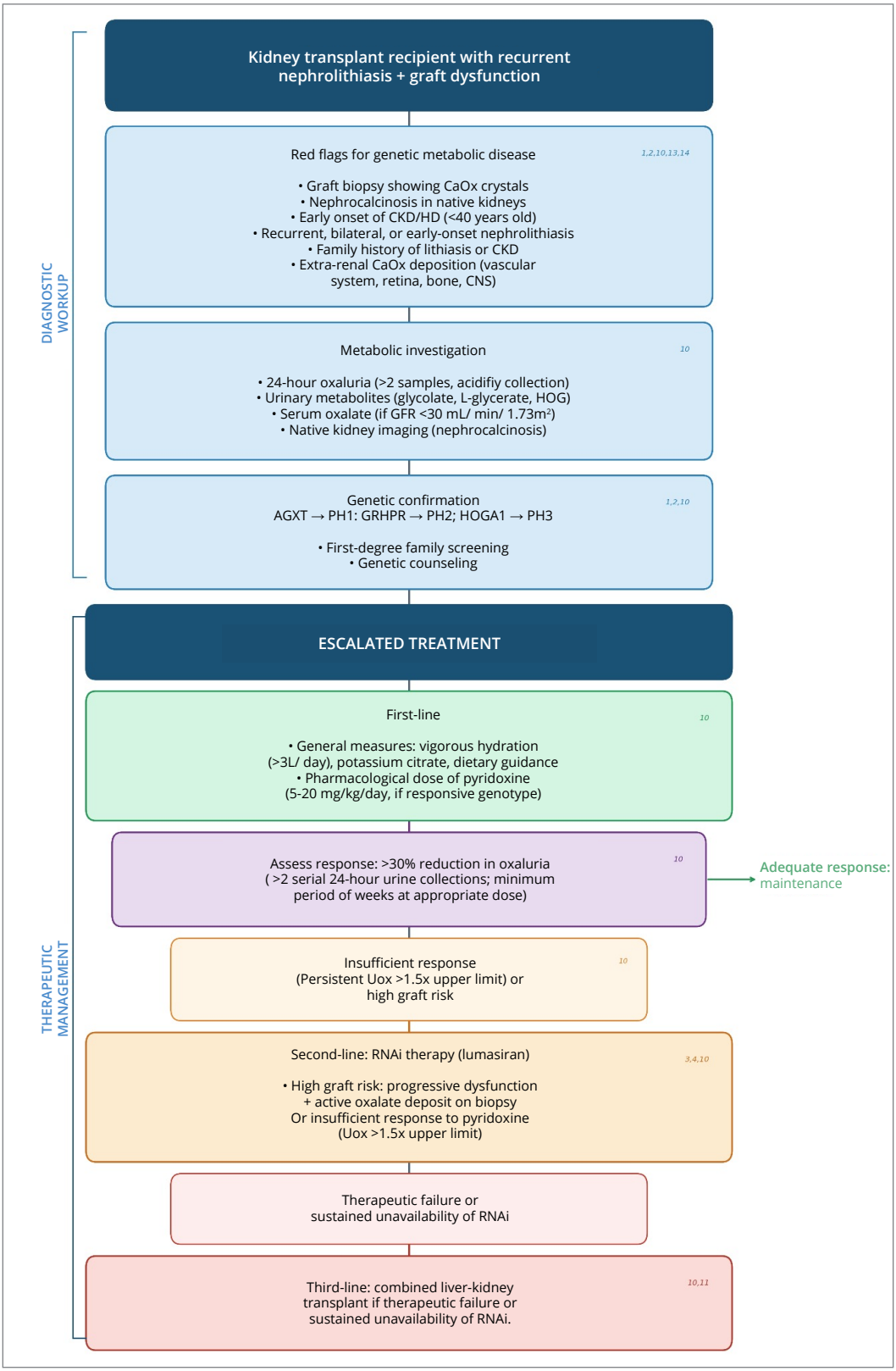
In this case, the identification of the homozygous p.Gly170Arg genotype directly guided the therapeutic strategy, following a stepped approach. According to the ERKNet/OxalEurope consensus, a therapeutic trial with pyridoxine should be the first step in potentially responsive genotypes, with objective evaluation through serial 24-hour oxaluria (Uox) measurements; a positive response is considered to be an average reduction of ≥ 30% in urinary oxalate excretion after a minimum period of several weeks of use at an appropriate dose[10]. Pharmacological doses are recommended (5-20 mg/kg/day, starting with 5 mg/kg/day and titrating according to response), with attention to periodic monitoring of signs and symptoms of peripheral neuropathy, given the potential for neurotoxicity with prolonged use[10]. In transplant patients, when there is evidence of active oxalate deposition in the graft with progressive dysfunction – posing a high risk to the graft – the addition of RNA interference therapy (lumasiran) is justified as an enhancement, regardless of the completion of the therapeutic trial with pyridoxine. Lumasiran acts by inhibiting the glycolate oxidase enzyme and reducing hepatic oxalate synthesis, with efficacy demonstrated in the ILLUMINATE studies[3,4]. In the case described, pyridoxine was initiated as first-line therapy (120 mg/day; ≈ 2.0 mg/kg/day), with subsequent escalation to 200 mg/day (≈ 3.4 mg/kg/day), but progressive graft dysfunction, with active oxalate deposition in biopsy, determined the introduction of lumasiran as an intensification measure, aiming at graft preservation.

In patients with an insufficient response to pyridoxine or at high risk for graft failure due to active oxalate deposition, RNA interference therapies, such as lumasiran, represent the main intensification strategy. By reducing hepatic oxalate production, these therapies have altered the traditional view of the need for combined liver-kidney transplantation in all scenarios, thereby modifying the natural course of the disease[3,4,11]. In selected cases, this control can increase the viability of strategies involving isolated kidney transplantation, provided that oxalate control is sustained and rigorous monitoring is in place[3,4,11]. The possibility of avoiding liver transplantation is particularly relevant given the global organ shortage. In Brazil, the incorporation of new technologies into the SUS (Unified Health System) follows technical criteria of scientific evidence and pharmacoeconomic evaluation established by CONITEC[5]. International data also demonstrate heterogeneity in access to diagnosis and treatment of PH1, reinforcing that the effectiveness of therapeutic strategies depends on the organization of the health system and the sustainability of treatment[12]. In this sense, national reports have value not only scientifically but also for public health, by documenting clinical experience with this rare condition and contributing to the debate on the organization of care pathways[5-7,12].

One of the most relevant aspects of this report is family screening following confirmation of the index case. In autosomal recessive diseases, confirmation in an individual should trigger systematic investigation of first-degree relatives, with testing targeted for the variant identified in the family, clinical evaluation for nephrolithiasis/nephrocalcinosis, and monitoring of renal function[1,2,10]. This approach has a direct impact because it allows for diagnosis at earlier stages, when conservative interventions and specific therapies have a greater potential to modify the prognosis, and avoids diagnostic delays in family members who might otherwise be labeled as having "idiopathic lithiasis"[1,2,10]. Furthermore, in transplant programs, family screening influences the eligibility of potential living donors and provides a basis for genetic counseling and reproductive planning[1,2,10].

Based on the above and the recommendations of the ERKNet/OxalEurope consensus[10], the screening score of Ferraro et al.[13] and the diagnostic algorithm of Michael et al.[14], a diagnostic-therapeutic algorithm adapted for renal transplant recipients with a history of recurrent nephrolithiasis is proposed, integrating the clinical warning signs, diagnostic investigation steps and therapeutic escalation discussed throughout this report (Fig. 2).

In summary, this case reinforces that PH1 should be considered in the differential diagnosis of candidates and recipients of kidney transplants with a history of recurrent nephrolithiasis and uncertain etiology of kidney disease[1,2,9,10]. A graft biopsy, showing oxalate deposition associated with tubular injury and absence of rejection, can be the sentinel event that triggers the correct etiological investigation[8,9]. Finally, genetic confirmation has repercussions that extend beyond the transplant recipient, enabling family screening and early interventions[1,2,10].



Source: Elaborated by the authors.

Figure 2. Proposed diagnostic-therapeutic algorithm for the investigation and management of PH1 in kidney transplant recipients with a history of recurrent nephrolithiasis. Red flags were compiled from the ERKNet/OxalEurope consensus[10] and validated screening scores,*. Criteria for pyridoxine response (≥ 30% reduction in oxaluria) and RNAi indication (Uox > 1.5 × upper limit) follow the European consensus recommendations[10]. HOG = 4-hydroxy-2-oxoglutarate; CNS = central nervous system. *Ferraro et al.[13]; **Michael et al.[14]

CONCLUSION

This case report highlights that PH1 should be considered in the differential diagnosis of renal graft dysfunction in patients with a history of recurrent nephrolithiasis, even in adulthood. Graft biopsy proved fundamental in distinguishing oxalate nephropathy from immunological causes of dysfunction, as the identification of CaOx crystals in histology points to a metabolic etiology and guides further investigation. Confirmation by genetic analysis, in addition to being definitive, has implications that extend beyond the transplant recipient, supporting genetic counseling and enabling family screening, with the potential to preserve renal function in possibly affected family members.

Finally, this case demonstrates that RNA interference-based therapies, such as lumasiran, can enable more conservative clinical management of the graft and stabilize the transplanted organ's function. However, interruptions or difficulties in accessing these technologies can compromise the benefits achieved and highlight the need for definitive strategies to control endogenous oxalate production – including, when clinically indicated, combined liver-kidney transplantation – aimed at patient and graft survival.

CONFLICT OF INTEREST

Nothing to declare.

AUTHOR'S CONTRIBUTION

Substantive scientific and intellectual contributions to the study: Sobreira SMA, Neri BO, Esmeraldo RM; **Conception and design:** Sobreira SMA, Neri BO, Sobreira Neto SMA; **Data analysis and interpretation:** Sobreira SMA, Adeodato VG, Dantas AH, Sobreira Neto SA, Teixeira AC; **Article writing:** Sobreira SMA; **Critical revision:** Neri BO, Esmeraldo RM, Teixeira AC, Sobreira Neto SA; **Final approval:** Sobreira SMA.

DATA AVAILABILITY STATEMENT

All dataset were generated or analyzed in the current study.

FUNDING

Not applicable.

DECLARATION OF USE OF ARTIFICIAL INTELIGENCE TOOLS

The authors declare that the ChatGPT tool (OpenAI) was used as an auxiliary resource in translating sections of the manuscript and in creating figures and the diagnostic-therapeutic algorithm. All generated content was critically reviewed by the authors, who assume full responsibility for the accuracy and integrity of the information presented.

ACKNOWLEDGEMENT

Not applicable.

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