



Kidney Transplants from a Donor with Systemic Lupus Erythematosus: A Clinical Case Report

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ABSTRACT

Background: Kidney transplantation (KT) remains the optimal therapy for patients with end-stage kidney disease (ESKD), offering improved survival and quality of life compared to dialysis. Due to the global shortage of organ donors, there is a growing trend toward expanding selection criteria, including acceptance of marginal or previously contraindicated donors. Lupus nephritis (LN) in deceased donors has traditionally raised concerns regarding transplant viability.

Case Presentation: We report the successful transplantation of both kidneys from a 34-year-old female donor with systemic lupus erythematosus (SLE) and biopsy-proven Class V lupus nephritis. Following brain death due to neuropsychiatric lupus, intraoperative biopsies confirmed minimal chronic damage. Recipient 1, a 51-year-old male with diabetes and hypertension, experienced delayed graft function (DGF), nosocomial pneumonia, and acute T cell-mediated rejection (Banff 1A). He responded to high-dose steroids, achieved partial renal function recovery, and was discharged without further dialysis. Recipient 2, a 39-year-old female with ESKD and a complex vascular access history, also experienced DGF but had progressive improvement in renal function post-discharge, with creatinine stabilizing at 1.4 mg/dL.

Conclusion: Limited data exist on transplantation outcomes from donors with active lupus nephritis. Prior case reports have shown variable outcomes, ranging from resolution of histological findings to allograft failure. These cases illustrate the potential utility of donor kidneys with pre-existing glomerulonephritis when carefully selected and monitored. Donor kidneys with lupus nephritis may be viable for transplantation under selected conditions. Enhanced donor screening protocols and recipient-specific risk stratification are essential to optimize outcomes and expand the donor pool.

KEYWORDS: Kidney transplantation, Lupus nephritis, Tissue donors

INTRODUCTION

Kidney transplantation (KT) is the preferred treatment for end-stage kidney disease (ESKD). However, the demand for kidneys far surpasses the available supply, leading to an expansion in donor selection criteria. In Mexico, 16,675 patients were on the waiting list for a kidney transplant at the end of 2024, whereas only 2,723 kidney trans-

plants were performed during that year [1]. To address this, centers are increasingly accepting kidneys from marginal donors. Here, we describe a case of an uncommon donor and the outcomes of two kidney recipients from that donor with lupus nephritis.

CASE PRESENTATION

The Donor

A 34-year-old female with a history of systemic lupus erythematosus (SLE) since the age of 16, previously manifested with cutaneous, articular, neurological, and hematological in

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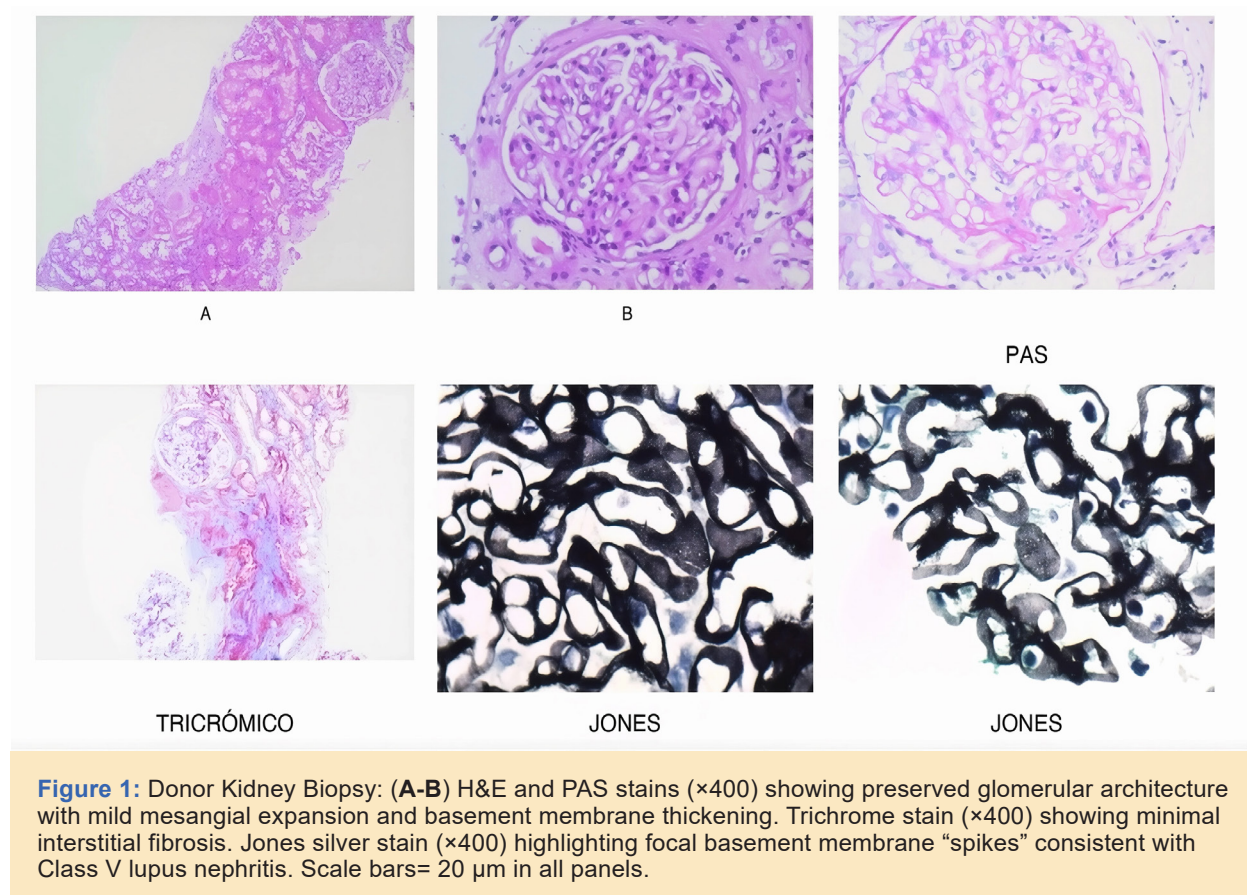


Figure 1: Donor Kidney Biopsy: (A-B) H&E and PAS stains ($\times 400$) showing preserved glomerular architecture with mild mesangial expansion and basement membrane thickening. Trichrome stain ($\times 400$) showing minimal interstitial fibrosis. Jones silver stain ($\times 400$) highlighting focal basement membrane “spikes” consistent with Class V lupus nephritis. Scale bars= 20 μm in all panels.

volvement. She had been off immunosuppressive therapy since January 2024, previously treated with DMARDs and steroids, which were gradually tapered off. She was admitted with a 4-day history of intense holocranial headache, accompanied by nausea, vomiting, and photophobia. On physical examination, she was conscious with a painful facial expression, neck stiffness, a positive Kernig’s sign, and a negative Brudzinski’s sign. A malar rash was present; cardiopulmonary examination was unremarkable. Cranial CT demonstrated posterior fossa edema. Given the high suspicion for lupus activity, a lumbar puncture confirmed active SLE. Methylprednisolone 1 g was administered after excluding infectious causes.

Laboratory findings included hemoglobin 10.1 g/dL, hematocrit 29.8%, WBC $7.74 \times 10^3/\mu\text{L}$ (neutrophils $6.62 \times 10^3/\mu\text{L}$, lymphocytes $0.46 \times 10^3/\mu\text{L}$), and platelets $347 \times 10^3/\mu\text{L}$. Serum creatinine was 0.47 mg/dL, BUN 12.9 mg/dL, Na 135 mmol/L, Cl 99 mmol/L, K 3.04

mmol/L, and P 1.4 mg/dL. Urinalysis revealed proteinuria (1.9 g/L), occult blood (++) and 1–3 RBCs and 3–5 WBCs per high-power field. ESR was 54 mm/h and CRP 54 mg/L; complement levels were low (C3= 43 mg/dL, C4 = 8.2 mg/dL).

Despite treatment, the following day the patient experienced abrupt neurological decline with respiratory arrest. Endotracheal intubation was performed and within 12 hours developed fixed and dilated pupils. Follow-up CT revealed diffuse cerebral edema. On hospital day 4, the patient was declared brain dead and the family consented to organ donation.

She was blood group A (+), seronegative for hepatitis B/C and HIV, and CMV IgG positive. The Kidney Donor Profile Index (KDPI) was 46%, and the Kidney Donor Risk Index (KDRI) was 0.96. The graft had one artery and one vein, and end-to-side anastomosis was performed during transplantation. A wedge biopsy of the donor kidney showed membranous lupus nephritis (LN) (ISN/RPS Class

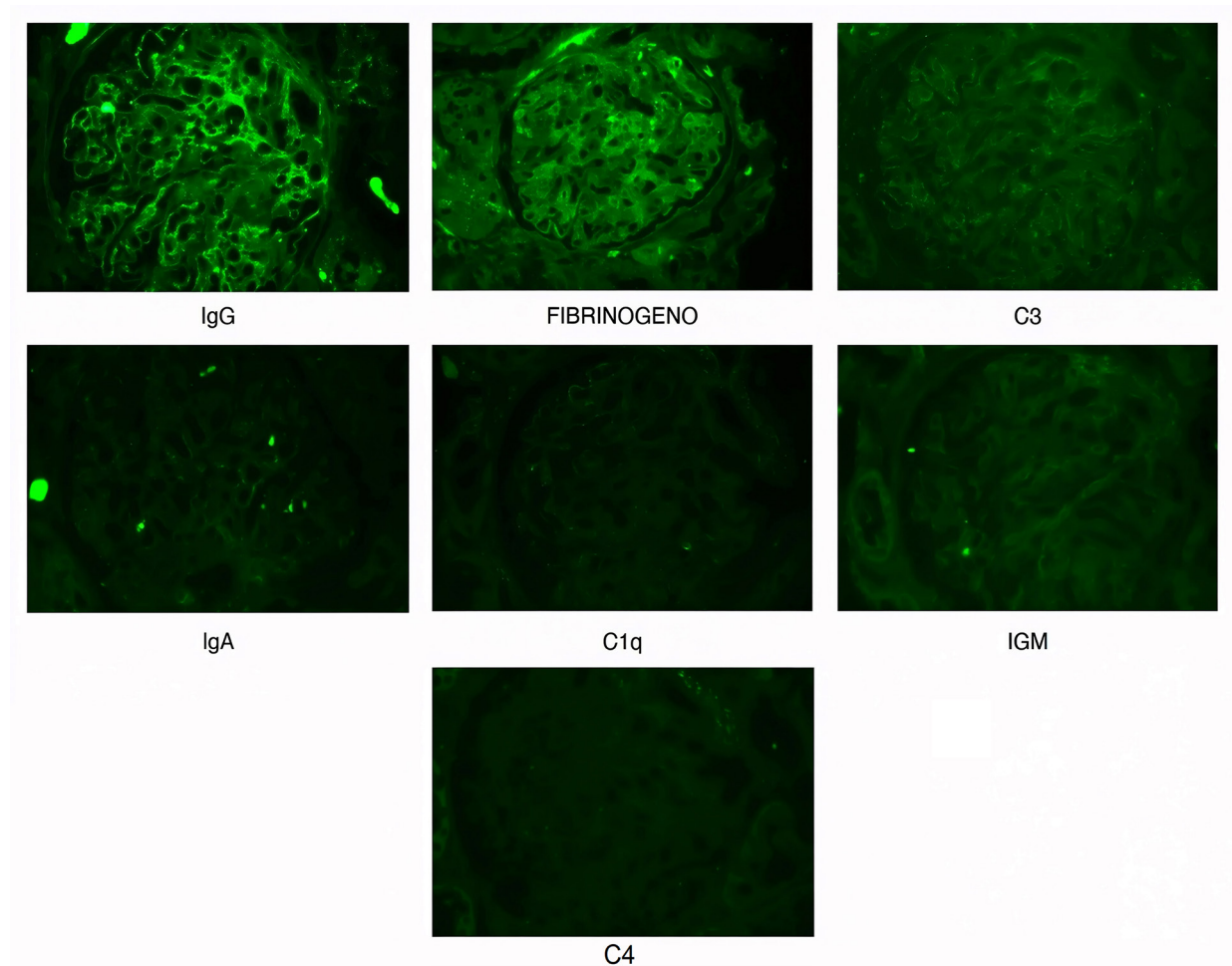


Figure 2: Donor Kidney Biopsy: Representative immunofluorescence staining (FITC, $\times 400$) shows diffuse granular capillary wall positivity for IgG (++), fibrinogen (++), and C3 (+), with focal mesangial IgA (+) and trace C1q reactivity. IgM and C4 are negative. The overall pattern is consistent with inactive immune-complex deposition characteristic of membranous lupus nephritis (ISN/RPS Class V). Scale bars= 20 μ m.

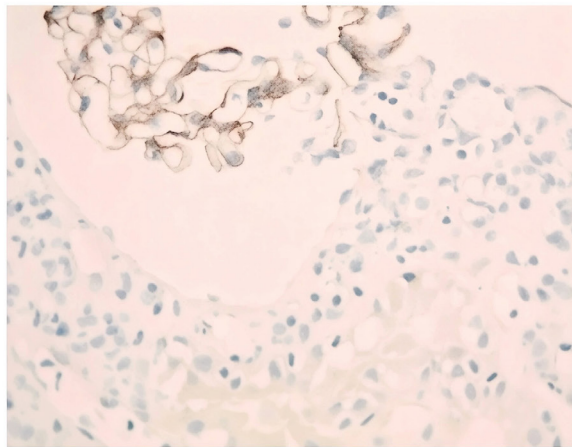
V) with mild mesangial expansion and focal basement membrane spikes. Interstitial fibrosis and inflammation were mild ($\approx 10\%$), with tubular atrophy $< 5\%$ and no endocapillary proliferation or crescents. Immunofluorescence demonstrated diffuse granular capillary staining for IgG (++), fibrinogen (++), and C3 (+), with trace IgA, C1q, C4, and IgM, consistent with inactive immune-complex deposition (Fig. 1 and Fig. 2).

The Recipient #1

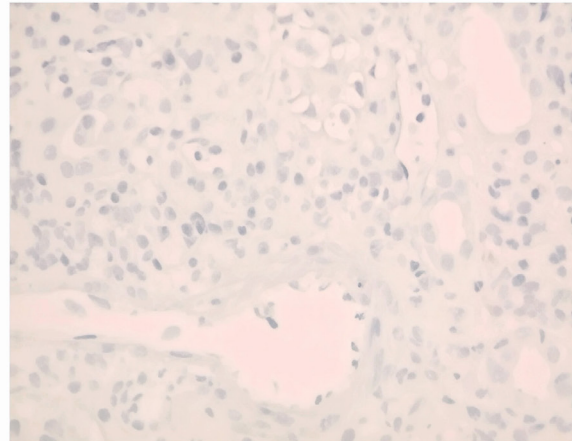
A 51-year-old male with a history of type 2 diabetes mellitus, arterial hypertension, and ESKD (G5), had been on hemodialysis (HD) three times per week for the past six years. The patient had no history of autoimmune diseases. At admission for KT, laboratory results

were as follows: hemoglobin 14.2 g/dL, WBC $19.8 \times 10^3/\mu\text{L}$ (neutrophils $19.35 \times 10^3/\mu\text{L}$, lymphocytes $0.2 \times 10^3/\mu\text{L}$), and platelets $165 \times 10^3/\mu\text{L}$. Biochemical profile: sCr 8.4 mg/dL, BUN 42.3 mg/dL, urea 90.6 mg/dL, Na 133 mmol/L, K 6.0 mmol/L, Cl 100.4 mmol/L, and P 5.7 mg/dL. Blood group A (+).

Induction therapy included basiliximab 20 mg IV on days 0 and 4, and methylprednisolone 500 mg during surgery. Basiliximab was chosen due to the patient's significant sarcopenia and overall clinical frailty, which placed him at higher risk for infectious complications. The surgery was uneventful. Cold ischemia time was 3 hours and warm ischemia time was 35 minutes. On days 1 and 2, the patient received 250 mg methylprednisolone post-transplant.



C4D



SV40

Figure 3: Recipient 1 Biopsy: Immunohistochemistry. Paraffin-embedded sections were processed using a polymer-based detection system with specific antibodies and appropriate positive and negative controls. SV40 (clone MRQ-4) showed no nuclear staining (negative), excluding BK virus infection. C4d (polyclonal) demonstrated negative staining in peritubular capillaries but positive subepithelial deposits along glomerular capillary walls.

Oral immunosuppression included mycophenolate mofetil 720 mg every 12 hours, prednisone 60 mg tapered, and tacrolimus 2 mg every 12 hours. On postoperative day 1, intermittent HD was required for oliguria, hyperkalemia, and metabolic acidosis secondary to delayed graft function (DGF); sCr was 7.80 mg/dL, BUN 54.1 mg/dL, and K 6.47 mmol/L.

On day 7 post-KT, the patient developed nosocomial pneumonia that progressed to acute respiratory distress syndrome (ARDS). Empiric meropenem and linezolid were administered for 3 days, and later adjusted to levofloxacin, given every 48 hours and adjusted for renal function, for a total of 18 days, after bronchoscopy identified *Legionella pneumophila* by PCR. He remained on intermittent HD with 188 mL/24 h of urine output, sCr 9.20 mg/dL, and BUN 109.2 mg/dL. Immunosuppression was tapered due to critical illness; tacrolimus and mycophenolate mofetil were discontinued, while IV hydrocortisone 200 mg/day was maintained. Testing for CMV, *Mycobacterium tuberculosis*, *Pneumocystis jirovecii*, and BK virus was negative.

On day 26 post-transplant, a renal biopsy

showed active T cell-mediated rejection, Banff Grade 1A (t2 + i3); C4d was negative in peritubular capillaries, indicating no evidence of antibody-mediated rejection, while SV40 was negative for nuclear staining, excluding BK polyomavirus nephropathy (Fig. 3). The immunofluorescence profile demonstrated persistent granular C1q/C3 deposits along the capillary walls, consistent with residual lupus membranous nephropathy rather than new immune complex activity (Fig. 4). No histologic features of drug-induced nephrotoxicity were identified; there was no evidence of arteriolar hyalinosis, striped interstitial fibrosis, or tubular isometric vacuolization, which would suggest calcineurin inhibitor toxicity. The observed interstitial inflammation was 80% with eosinophils and tubulitis was consistent instead with acute T-cell-mediated rejection (Banff grade IA). Methylprednisolone 300 mg IV every 24 hours for three days was administered. The patient improved clinically, with reduced oxygen requirements. Intermittent HD was required from POD 1, two to three times weekly during the first two weeks, and twice more before discharge (≈10 sessions total), after which renal function progressively recovered.

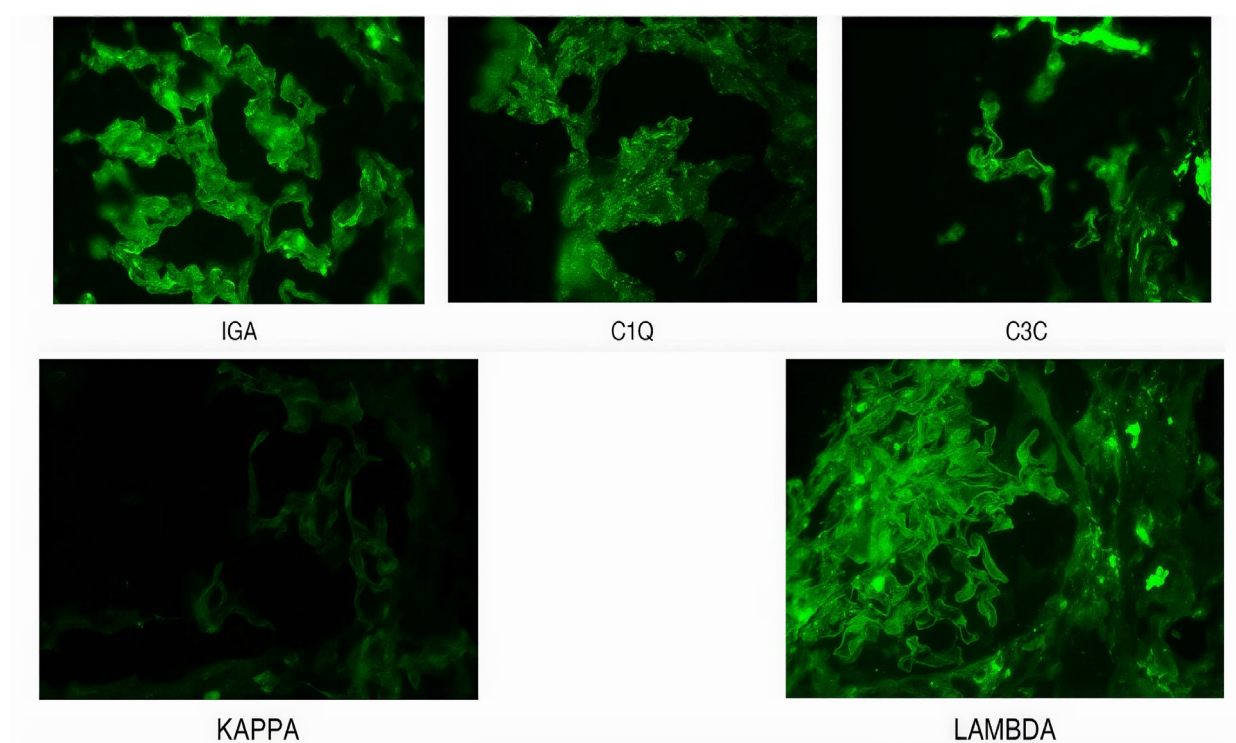


Figure 4: Recipient 1 Biopsy: Direct immunofluorescence. Frozen 4- μ m sections incubated with FITC-labeled antibodies (IgG, IgM, IgA, C3, C1q, fibrinogen, κ , λ). Three non-sclerotic glomeruli were evaluated: IgG/IgM non-assessable, IgA negative, C1q and C3 with trace granular epimembranous deposits, fibrinogen and light chains negative.

He was discharged with mycophenolate mofetil 360 mg PO BID, tacrolimus 3 mg PO BID, prednisone 10 mg PO daily, valganciclovir 450 mg BID twice weekly, bumetanide 2 mg every 12 hours, and erythropoietin 4,000 IU three times per week. At discharge, sCr was 3.08 mg/dL, BUN 70.4 mg/dL, urine output 2,300 mL/day, and estimated glomerular filtration rate (eGFR) was 24 mL/min. No further dialysis was required. At 10 months post-transplant, he maintains stable graft function (sCr 1.26 mg/dL, eGFR 65mL/min1.73m², CKD-EPI). The 24-hour Cr clearance, measured from a urine volume of 2050 mL, was 41 mL/min, with albuminuria of 176 mg/24 h. Tacrolimus trough level is therapeutic at 7.0 ng/mL, and genetic rejection assay was negative. No clinical or biochemical evidence of recurrent or progressive glomerular disease has been observed to date.

The Recipient #2

A 39-year-old female with type 2 diabetes mellitus (insulin-treated), hypothyroidism,

hypertension, and obesity (284 pounds, status post-gastric sleeve surgery; weight at transplant was 198 pounds), had ESKD on HD for six years. The patient had no history of autoimmune diseases. She had no viable vascular access, with three thrombosed AV fistulas (including two femoral) and multiple catheter placements. Central venous stenosis had been treated by angioplasty.

At admission, laboratory results were as follows: hemoglobin 12.8 g/dL, white blood cell count $5.99 \times 10^3/\mu\text{L}$ (neutrophils $4.38 \times 10^3/\mu\text{L}$), and platelets $234 \times 10^3/\mu\text{L}$. Biochemical profile: glucose 144 mg/dL, sCr 10.30 mg/dL, BUN 71.5 mg/dL, urea 153 mg/dL, uric acid 6.1 mg/dL, Na 133.3 mmol/L, K 5.22 mmol/L, Cl 97.3 mmol/L, Ca 9.5 mg/dL, and P 4.4 mg/dL. Blood group A (+); HIV negative.

Induction therapy included thymoglobulin (ATG) 125 mg IV for three days, and methylprednisolone 1 gram IV intraoperatively, followed by 500 mg and 250 mg on the first

Table 1: Side-by-side comparison of both recipients from the same SLE donor.

Parameter	Recipient #1	Recipient #2
Age/Sex	51-year-old male	39-year-old female
Comorbidities	Type 2 DM, HTN, ESKD on HD (6 yrs)	Type 2 DM (insulin-treated), hypothyroidism, HTN, obesity (post-gastric sleeve)
CIT/WIT	3 h/35 min	5 h/40 min
Induction IS	Basiliximab	Thymoglobulin
Major complications	Nosocomial Legionella pneumonia (POD7) → ARDS; temporary IS taper; acute cellular rejection	Two episodes of graft pyelonephritis and a CMV infection occurred after hospital discharge.
Biopsy findings	Acute T-cell-mediated rejection (Banff 1A, t2 + i3); residual lupus deposits (C1q/C3 granular)	Not performed
Urine output	POD1: oliguria → discharge 2.3 L/d; 10 mo: 2.05 L/24 h	POD4: 1.29 L/24 h → stable ~2.5 L/d
Creatinine (mg/dL)	Pre-Tx 8.4 → POD1 7.8 → POD7 9.2 → 10 mo 1.26	Pre-Tx 10.3 → POD4 5.5 → 1 mo 1.4 → 11 mo 0.9
Albuminuria	Albuminuria 176 mg/24 h (10 mo)	Microalbuminuria 10 mg/dL (11 mo)
eGFR (CKD-EPI)	24 mL/min (discharge) → 65 mL/min/1.73 m ² (10 mo)	49 mL/min/1.73 m ² (1 mo) → 83 mL/min/1.73 m ² (11 mo)
Dialysis sessions (total)	HD maintained 2–3×/wk. for first 2 wks. + 2 more before discharge	3 sessions (POD1, POD3, 2 ambulatory)

Abbreviations: DGF, delayed graft function; HD, hemodialysis; CIT, cold ischemia time; WIT, warm ischemia time; IS, immunosuppression; POD, postoperative day; ARDS, acute respiratory distress syndrome; Cr, creatinine; Tac, tacrolimus; eGFR, estimated glomerular filtration rate; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration.

and second postoperative days, respectively. ATG was selected as the induction agent because the graft was obtained from a deceased expanded-criteria donor, where lymphocyte-depleting induction is preferred to reduce the risk of acute rejection and delayed graft function. Surgery was uneventful; cold ischemia was 5 hours and warm ischemia 40 minutes. A double J catheter was placed during uretero-vesical anastomosis.

On the first post-transplant day, she developed DGF and underwent HD for 2 h and 45 min. Mycophenolic acid and acyclovir were initiated. A second HD session was needed on day 3. By day 4, urine output was 1,290 mL/24 h, and sCr was 5.51 mg/dL. She was discharged the following day on trimethoprim/sulfamethoxazole 800/160 mg orally (Monday–Wednesday–Friday), acyclovir 400 mg twice daily, levothyroxine 100 µg

daily, enoxaparin 40 mg daily, mycophenolic acid 360 mg twice daily, furosemide 40 mg twice daily, and erythropoietin (8,000 IU on Wednesday and 4,000 IU on Saturday). Two additional ambulatory HD sessions were required. Subsequently, urine output stabilized at ~2.5 L/day and sCr dropped to 1.1 mg/dL. One month post-transplant, the double J stent was removed. At that time, sCr was 1.4 mg/dL, tacrolimus trough level was 11.4 ng/mL, and microalbuminuria was 55.2 mg/dL. CMV PCR and BK virus screening remained negative, and a control biopsy was scheduled at three months post-transplant. However, the planned biopsy was not performed because the patient developed two episodes of graft pyelonephritis after discharge, which precluded the procedure. She also experienced a CMV infection that resolved with appropriate antiviral therapy. At 11-month follow-up, laboratory results were as follows: tacrolimus trough

Table 2: Comparative summary of donor-derived lupus nephritis cases reported in the literature and present dual-recipient case.

Study/Year	Donor LN Class (ISN/RPS)	Activity/Chronicity	Induction/ Maintenance IS	Early Complications	Function (follow-up)	Key Notes
Lipkowitz et al, 2000	Class IV-V	Inactive; no chronic changes	ATG → Tac, Pred, MMF	Recipient 1: None Recipient 2: acute cellular rejection	Both with normal renal function + urinalysis at 2 yrs.	Resolution of tubular reticular structures and immune deposits
Schwartzman et al, 2005	Class III	Mild activity, minimal chronicity	Alemtuzumab → Tac, Pred, MMF	None	6 mo: sCr 1.5–1.7 mg/dL, GFR 46–49 mL/min	Resolution of immune deposits and improving Class III
Magoon et al, 2010	Class IV	Active; chronicity not emphasized	ATG → Pred, Tac, MMF	DGF; occipital infarcts (POD5)	4 mo: resolution of proliferative lesions; 5.5 yrs: sCr 2.0 mg/dL	Donor LN resolved over time
Zhou et al, 2024	Class V	Mild activity, minimal chronicity	ATG → Tac, MMF, Pred	Renal hemangioma	1 yr: Cr 1.3 mg/dL, proteinuria 135 mg/24 h; normal Doppler	Resolution of lupus IF markers (IgG-, C3-, C4d+ only)
Present case (Recipient #1)	Class V	Inactive/mild chronicity	BAS → Tac, MMF, Pred	DGF, Banff IA rejection	10 mo: sCr 1.26 mg/dL, eGFR 65 mL/min 1.73 m ²	Granular C1q/C3 deposit, no new immune complex activity
Present case (Recipient #2)	Class V	Inactive/mild chronicity	ATG → Tac, MMF, Pred	DGF	11 mo: sCr 0.9 mg/dL, eGFR 83 mL/min 1.73 m ²	Pyelonephritis post discharge and CMV infection that resolved with medical therapy

Abbreviations: LN, lupus nephritis; IS, immunosuppression; ATG, Antithymocyte globulin; DGF, delayed graft function; BAS, basiliximab; Tac, tacrolimus; MMF, mycophenolate mofetil; Pred, prednisone; sCr, creatinine

level 7.5 ng/mL, sCr 0.9 mg/dL, BUN 23 mg/dL, and uric acid 6.4 mg/dL. BK virus testing was negative. Urinary microalbumin was 10 mg/dL, with urine albumin-to-creatinine ratio of 100 mg/g. Urinalysis showed 3–5 leukocytes per high-power and moderate bacteriuria. A comparative summary of both recipients, including demographic data, induction regimens, ischemia times, complications, and short-term outcomes, is presented in Table 1.

Ethical Considerations

This study was conducted in accordance with the ethical standards of the institutional and national research committees and with the 2013 Declaration of Helsinki. Approval for this donation was granted by the Organ Donation Committee of Christus Muguerza Hospital Alta Especialidad. Both the donor and the recipients provided written informed consent before organ procurement and transplantation. Given the presence of an active autoimmune condition in the donor, the recipients were explicitly informed of the potential risks associated with donor pathology, including the presence, persistence, or recurrence of immune complex-mediated nephropathy in the graft. The transplantation proceeded after multidisciplinary review and in compliance with current national transplant regulations and institutional policies regarding donor-derived risk assessment.

DISCUSSION

This dual-recipient case supports the emerging evidence that kidneys from donors with SLE and biopsy-proven LN may be transplanted successfully when activity and chronicity are minimal and perioperative conditions are well controlled. The procurement biopsy showed class V LN with low activity and chronicity and favorable KDPI/KDRI. Although both recipients developed early DGF—one complicated by infection and acute rejection—renal recovery and stable function were achieved, suggesting that donor-derived immune-complex deposits can persist histologically but remain clinically inactive.

Previous reports have shown variable outcomes. Lipkowitz et al. described successful use of kidneys from a donor with class IV–V LN in remission [2]; Schwartzman et al. reported persistent but nonprogressive deposits in a class III LN donor [3]; Magoon et al. observed histological resolution of class IV LN with crescents after initial DGF [4]; and Zhou et al. documented disappearance of class V LN deposits after one year [5]. A detailed comparison of previously published cases of kidney transplantation from donors with lupus nephritis, including donor class, activity/chronicity, immunosuppressive regimen, complications, and outcomes, is provided in Table 2.

Our findings align with these observations, emphasizing that short ischemia times, low chronicity, and tailored induction favor recovery. Residual subepithelial deposits do not necessarily imply active disease, reinforcing the value of proteinuria monitoring and biopsies to differentiate donor-derived from recurrent glomerulopathies. When considering kidneys from donors with LN, clinicians must ensure informed consent that extends beyond routine discussion of transplant risks, clearly addressing the potential for persistent immune-complex disease, uncertain long-term outcomes, and the balance between risks and the urgent need to expand organ availability.

In conclusion, aligned with the KDIGO Clinical Practice Guideline on the Evaluation and Management of Kidney Transplant Candidates [6], this report reinforces that donor selection should be guided by the degree of histologic chronicity and preserved functional reserve rather than categorical exclusion based on disease etiology. Our experience demonstrates that kidneys from donors with SLE and class V LN can achieve successful outcomes under carefully controlled conditions. As highlighted by Mulley and Kanellis [7], such donors may be ethically integrated into expanded-criteria programs when organ function, chronicity indices, and ischemia times remain favorable. In this case, short cold and warm ischemia times, tailored induction therapy, and structured post-transplant monitoring were decisive in achieving renal recovery despite early DGF.

Overall, this experience highlights that a multidisciplinary and pathology-guided approach can safely expand donor acceptance criteria while preserving graft performance and recipient safety. Such strategies are essential to responsibly broaden the donor pool and help meet the increasing global demand for KT.

ACKNOWLEDGMENTS

We extend our sincere thanks to the Christus Muguerza Health team for the support in the recovery of medical records. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

FINANCIAL SUPPORT: None.

CONFLICTS OF INTEREST: None declared.

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