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Kidney Injury in Patients with Leishmaniasis Treated with Amphotericin B in Northern Paraguay: A Series of Four Cases

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Abstract

Leishmaniasis and amphotericin B treatment can both be associated with kidney injury. This retrospective series describes four men aged 31–61 years assessed between 2023 and 2025 in northern Paraguay: one with visceral and three with mucocutaneous leishmaniasis, including one patient with newly diagnosed HIV infection. The patient with visceral disease had kidney dysfunction, polyuria and electrolyte abnormalities before amphotericin B and later required maintenance haemodialysis. Among the patients with mucocutaneous disease, creatinine increased during treatment; two were dialysis-independent at last follow-up and one required maintenance haemodialysis. In that patient, a transfer note contained an unusual record of amphotericin administration, but the cumulative dose could not be reconstructed. These cases illustrate the difficulty of distinguishing disease-related kidney injury, drug toxicity and other contributors. Careful verification of amphotericin formulations and doses, monitoring of kidney function and electrolytes, and follow-up after acute kidney injury may help guide care. Clinical details and reported outcomes require confirmation against source records.

Keywords: Leishmaniasis; Amphotericin B; Acute kidney injury; Kidney failure; Renal dialysis; Paraguay

Introduction

Leishmaniasis is endemic in Paraguay: between 2017 and 2020, 278 cases of tegumentary and 101 cases of visceral leishmaniasis were notified, and San Pedro was the department with the most tegumentary cases [1]. Pan American Health Organization guidance includes amphotericin B formulations among the treatments for visceral and mucosal leishmaniasis, with the liposomal formulation preferred in patients at higher risk of toxicity and in those with HIV co-infection [2].

Both the disease and its treatment can injure the kidney. Visceral leishmaniasis causes proteinuria, haematuria, tubular dysfunction, and acute kidney injury (AKI) [3,4]; in a Brazilian cohort, AKI affected 33.9% of patients, and amphotericin B use was strongly associated with AKI (odds ratio 18.4) [5]. Amphotericin B alters tubular cell membrane permeability and causes renal vasoconstriction, leading to potassium wasting, distal renal tubular acidosis, and a fall in glomerular filtration; sodium loading attenuates this toxicity [6,7]. Lipid formulations are less nephrotoxic but do not eliminate AKI [8]. We describe four patients with leishmaniasis and kidney injury before, during or after amphotericin B treatment who were assessed by the nephrology service of a regional hospital in northern Paraguay, following the CARE guidelines [9,10].

Case Report

Design and data source. This retrospective case series is based on the progress notes and consultation records of the Nephrology Service, Hospital Regional de Concepción, a public referral hospital in northern Paraguay. All 1146 progress-note documents recorded between October 2021 and October 2026 were screened with the Spanish terms for leishmaniasis, amphotericin, and rK39, followed by manual review. The included patients were first assessed between April 2023 and June 2025.
Selection criteria. We included patients with a confirmed diagnosis of visceral or tegumentary leishmaniasis who had kidney involvement before, during, or after treatment with amphotericin B, and who had at least two nephrology assessments or one assessment with a documented outcome. Of nine patients identified, four were excluded because leishmaniasis had been ruled out (negative rK39 or anti-Leishmania serology), and one with visceral leishmaniasis and HIV infection was excluded because he was assessed only once and his outcome was not recorded.

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Variables and definitions. We recorded age, place of residence, clinical form and diagnostic method of leishmaniasis, comorbidities, HIV status, amphotericin B formulation and dose, serial laboratory results, urine output, imaging, need for dialysis, and outcome. AKI was assessed with reference to the KDIGO criteria [11]; staging requires verification against baseline values and the specified time windows. Baseline creatinine was the last value before amphotericin B, when available. Dialysis independence and renal function were reported separately, and creatinine at last follow-up is reported. Laboratory values are given in the units used at our centre: to convert creatinine from mg/dL to µmol/L, multiply by 88.4; to convert urea from mg/dL to mmol/L, multiply by 0.166.

The original draft reports institutional ethics approval; the approval and its scope require confirmation against the original documentation (see Ethics and consent).

All four patients were reported to be men. Three lived in rural communities in Concepción department and one in San Pedro department. Table 1 summarises the available baseline data.

Visceral leishmaniasis (D1)

Case D1. A 38-year-old man with hypertension and chronic alcohol use from a rural community in Concepción department was admitted through the emergency department in December 2023 with AKI of undetermined cause, anaemia, haematuria, and bilateral leg oedema. On day 0, creatinine was 3.6 mg/dL, urea 96 mg/dL, haemoglobin 85 g/L, leucocytes 2.7 × 10⁹/L, and platelets 213 × 10⁹/L; HIV, hepatitis B, and hepatitis C tests were negative. With intravenous fluids, creatinine fell to 1.42 mg/dL on day 2, but he became polyuric (5.2 L/24 h), with sodium 124 mmol/L and potassium 2.75 mmol/L. On day 3, ultrasonography showed hepatosplenomegaly, ascites, and kidneys with diffuse parenchymal changes, and the rK39 antibody test was positive, a result later confirmed by Leishmania amastigotes on bone marrow aspirate at the referral hospital. Pancytopenia worsened (haemoglobin 78 g/L, leucocytes 2.1 × 10⁹/L, platelets 145 × 10⁹/L).

On day 4, creatinine had risen again to 3.3 mg/dL. Liposomal amphotericin B was recommended after a test dose, with an explicit warning about its tubular toxicity, together with transfusion and an immunological screen. By day 6, urine output had reached 9.9 L/24 h, sodium had fallen to 119 mmol/L, and creatinine was 4.0 mg/dL; he was referred to a tertiary hospital. According to the patient and his family, he was treated there for leishmaniasis and received several haemodialysis sessions before discharge. The discharge summary documented liposomal amphotericin B 3 mg/kg/day on days 1–5, 14, and 21 (cumulative dose 21 mg/kg), six haemodialysis sessions, and a discharge creatinine of 2.3 mg/dL; no nephrology follow-up was arranged.

Twelve months later (December 2024), he returned with uraemic symptoms: creatinine 11.2–13.1 mg/dL, urea 157–178 mg/dL, sodium 106 mmol/L, potassium 2.26 mmol/L, haemoglobin 70 g/L, leucocytes 3.7 × 10⁹/L, and platelets 144 × 10⁹/L, with a bland urinalysis, AST/ALT 94/70 U/L, alkaline phosphatase 307 U/L, and albumin 35.5 g/L. He declined haemodialysis and received intravenous fluids with sodium and potassium supplements. Three weeks later (January 2025), he was readmitted disoriented and agitated, with uraemic syndrome, creatinine 25.9 mg/dL, urea 286 mg/dL, and haemoglobin 59 g/L, and heparin-free haemodialysis was started in intensive care through a femoral catheter. He was anuric by day 9.

The admission was complicated by Staphylococcus haemolyticus bacteraemia, Acinetobacter baumannii hospital-acquired pneumonia, pain and bleeding at the femoral catheter site requiring catheter replacement, and later a catheter exit-site infection treated with vancomycin and catheter replacement. Anti-HBs was reactive, with an undetectable hepatitis B viral load; ferritin exceeded 1000 ng/mL (transferrin saturation 62.4%). An immunological screen was requested repeatedly and was negative (ANA, anti-DNA, ANCA, antiphospholipid antibodies, C3, and C4), and visceral leishmaniasis was recorded as cured. After 44 sessions in 12 weeks, the National Institute of Nephrology allocated him a maintenance haemodialysis place outside the city, which he and his family declined; he therefore continued thrice-weekly haemodialysis at our centre. In April 2026 he had a catheter-related bacteraemia due to Enterobacter sp., without vegetations on echocardiography (ejection fraction 56%). In October 2026, 21 months after starting dialysis, he remained anuric on maintenance haemodialysis; as of July 2026, he was still dialysed through a temporary jugular catheter.

Mucocutaneous leishmaniasis (D2–D4)

Case D2. A 47-year-old man with chronic alcohol use from a

Table 1: Baseline characteristics of the four patients.

Case	Age (years)	Year	Clinical form	Diagnosis	Comorbidities	Amphotericin B formulation	Admitting unit
D1	38	2023	Visceral	rK39 positive; amastigotes on bone marrow aspirate	Hypertension; chronic alcohol use	Liposomal (at the referral hospital)	ED, then referral hospital
D2	47	2023	Mucocutaneous	Nasal mucosal biopsy	Chronic alcohol use	Conventional, switched to liposomal	Medical ward
D3	31	2023	Mucocutaneous (nasal lesion)	Nasal mucosal biopsy	HIV infection, newly diagnosed (viral load 83,400 copies/mL)	Conventional (deoxycholate)	Medical ward
D4	61	2025	Mucocutaneous	Nasal mucosal biopsy	Diabetes; hypertension; mixed dyslipidaemia	Conventional (deoxycholate); '34 doses in 7 days'	ICU

a) ED, emergency department; ICU, intensive care unit.

Table 2: Timeline of events (D, day relative to first assessment or admission; M, month).

Case	Events
D1	D0 (December 2023) creatinine 3.6, pancytopenia · D2 creatinine 1.42, polyuria 5.2 L/24 h, sodium 124, potassium 2.75 · D3 rK39 positive, hepatosplenomegaly · D4 creatinine 3.3; liposomal amphotericin B recommended · D6 polyuria 9.9 L/24 h, sodium 119, creatinine 4.0; referral · referral hospital: liposomal amphotericin B (21 mg/kg), six haemodialysis sessions, discharge creatinine 2.3 · M12 creatinine 13.1, sodium 106; haemodialysis declined · M13 uraemic syndrome, creatinine 25.9; haemodialysis started · M16 44 sessions; place outside the city declined · M34 on maintenance haemodialysis
D2	D0 admission; conventional amphotericin B; creatinine 0.9 · D11 creatinine 1.9; switch to liposomal amphotericin B with saline loading · D50 readmission to complete the liposomal course · D60 creatinine 2.1; ultrasonography showing acute nephropathy · 3 months creatinine 1.4
D3	D0 admission; amphotericin B deoxycholate; creatinine 0.8 · D6 creatinine 5.9 · D7 amphotericin-associated AKI diagnosed; transfer requested · D8 creatinine 4.1, urine output 2.9 L/24 h · D9 creatinine 2.8; conservative management · D12 liposomal amphotericin B · D14 antiretroviral therapy · D20 discharge, creatinine 1.3 · 3 months creatinine 1.0
D4	Before transfer '34 doses of amphotericin in 7 days'; normal ultrasonography on D−8 · D0 anuria, haematuria, creatinine 7.0 · D1 creatinine 10.4; haemodialysis started · D5 urine output 3.15 L/24 h; peak creatinine 10.5 · D13 creatinine 5.6 · D14 diagnosis revised to stage 5 diabetic CKD · D15 session 10; referral for maintenance haemodialysis · D30 maintenance haemodialysis at a satellite unit · 3 months on haemodialysis

a) Creatinine and urea in mg/dL; sodium and potassium in mmol/L. AKI, acute kidney injury; CKD, chronic kidney disease.

Table 3: Diagnostic assessment and outcomes.

Case	Creatinine, baseline/peak	AKI stage (KDIGO)	Other findings	Haemodialysis	Last follow-up	Outcome
D1	Not available/4.0 (2023); 25.9 (2025)	Requires verification	Polyuria up to 9.9 L/24 h; sodium 119 mmol/L; potassium 2.75 mmol/L; pancytopenia; hepatosplenomegaly	Six sessions at the referral hospital (2023); maintenance since January 2025	Oct-26	Kidney failure on maintenance haemodialysis
D2	0.9/2.1	Requires verification	Ultrasonography showing acute nephropathy	No	3 months, creatinine 1.4	Partial recovery; lesions healed
D3	0.8/5.9	3	HIV viral load 83,400 copies/mL; haemoglobin 67 g/L; post-void residual 172 mL	No (transfer requested)	3 months, creatinine 1.0	Recovered; on antiretroviral therapy
D4	Not available/10.5	3	Haematuria; enlarged right kidney; ejection fraction 41%; magnesium 1.4 mg/dL	Yes, from D1 (10 sessions by D15)	3 months, on haemodialysis	Dialysis-dependent

a) Creatinine in mg/dL. AKI, acute kidney injury; D, day. Staging for D1 and D2 requires confirmation using the appropriate baseline and time window.

rural community in Concepción department was admitted to the medical ward in April 2023 for treatment of mucocutaneous leishmaniasis diagnosed by biopsy of the nasal mucosa with conventional amphotericin B deoxycholate 50 mg/day. On day 11, creatinine was 1.9 mg/dL (0.9 mg/dL on admission), urea 51 mg/dL, uric acid 6.9 mg/dL, sodium 137 mmol/L, potassium 3.8 mmol/L, and haemoglobin 129 g/L, with spontaneous diuresis and no oedema. On the advice of the infectious diseases team, conventional amphotericin was replaced by liposomal amphotericin B on alternate days, infused over 10 h, with 300 mL of Ringer's lactate before and after each infusion, daily electrolyte monitoring, and avoidance of nephrotoxic drugs. He was readmitted in June 2023 to complete the liposomal course (cumulative dose 1.5 g); on day 10 of that admission, creatinine was 2.1 mg/dL, urea 49 mg/dL, uric acid 7.8 mg/dL, and potassium 3.9 mmol/L, and ultrasonography showed features of acute nephropathy. Intravenous saline (300 mL every 8 h) and oral fluids were prescribed. At 3 months, creatinine was 1.4 mg/dL, and the mucosal lesions had healed.

Case D3. A 31-year-old man from a rural community in Concepción department, with mucocutaneous leishmaniasis involving the nostril and HIV infection diagnosed earlier that month (viral load 83,400 copies/mL; CD4 count 180 cells/µL), was admitted to the medical ward in October 2023 and treated with amphotericin B deoxycholate 1 mg/kg/day. By day 6, creatinine had risen to 5.9 mg/dL from 0.8 mg/dL on admission, with urea 171 mg/dL, sodium 140 mmol/L, potassium 3.6 mmol/L, and haemoglobin 85 g/L. At nephrology assessment on day 7, he was haemodynamically stable (blood pressure 120/70 mmHg) and had no oedema, with a spontaneous urine output of 750 mL in 6 h; ultrasonography showed normal-sized kidneys without hydronephrosis and a post-void residual volume of 172 mL (27%). AKI secondary to amphotericin B was diagnosed, and the drug was withheld. Transfer to a centre with a dialysis machine reserved for patients with blood-borne viral infections was requested, because our unit could not admit new patients. With intravenous fluids and furosemide, urine output rose to 2.9 L/24 h, and creatinine fell to 4.1 mg/dL on day 8 and

to 2.8 mg/dL on day 9 (urea 75 mg/dL), so conservative management was continued. Haemoglobin fell to 67 g/L on day 8, and leucocytes to 2.9 × 10⁹/L on day 9. He was transfused, and liposomal amphotericin B was started on day 12, once creatinine was below 2 mg/dL. Antiretroviral therapy with dolutegravir, abacavir, and lamivudine was started on day 14. He was discharged on day 20 with creatinine 1.3 mg/dL; at 3 months, creatinine was 1.0 mg/dL, and the nasal lesion had healed.

Case D4. A 61-year-old man with diabetes, hypertension, and mixed dyslipidaemia from San Pedro department, who was being treated for mucocutaneous leishmaniasis at another hospital, was transferred to our intensive care unit in June 2025 with hospital-acquired pneumonia, bilateral pleural effusions, anasarca, and anuria. The transfer note recorded that he had received '34 doses of amphotericin in 7 days'; the formulation, dose per administration, and cumulative dose were not documented. The referring hospital later confirmed that conventional amphotericin B deoxycholate had been used for a mucosal lesion diagnosed by nasal biopsy, but the cumulative dose could not be reconstructed. An abdominal ultrasound scan 8 days earlier had been normal, and no previous creatinine measurement was available. On day 0, creatinine was 7.0 mg/dL, urea 95 mg/dL, haemoglobin 126 g/L, leucocytes 9.5 × 10⁹/L, and platelets 323 × 10⁹/L; he passed only small volumes of haematuric urine despite a furosemide infusion and required oxygen at 8 L/min. Ultrasonography showed a right kidney larger than the left and bilateral pleural effusions; rapid tests for HIV, hepatitis B, hepatitis C, and syphilis were negative.

On day 1, with creatinine 10.4 mg/dL and urea 112 mg/dL, a right femoral catheter was inserted and heparin-free haemodialysis was started; imipenem was continued, and methyldopa and amlodipine were prescribed. Echocardiography showed moderate left ventricular systolic dysfunction (ejection fraction 41%) with regional hypokinesia, moderate mitral and severe tricuspid regurgitation, severe pulmonary hypertension, and a small pericardial effusion. Urine output increased from 670 mL/24 h on day 3 to 3150 mL/24 h on day 5, when haematuria resolved and the furosemide infusion was stopped. Creatinine peaked at 10.5 mg/dL on day 5 and, on haemodialysis, fell to 5.6–5.9 mg/dL on days 13–14, with magnesium 1.4–1.8 mg/dL. On day 14, the diagnosis was revised to stage 5 chronic kidney disease (CKD) of diabetic origin, and after ten sessions he was referred to the National Institute of Nephrology for maintenance haemodialysis; erythropoietin was unavailable at our centre. HbA1c was 8.9%. He started maintenance haemodialysis at a satellite unit on day 30 and, at 3 months, remained on thrice-weekly haemodialysis with a residual urine output of approximately 1.5 L/day.

Kidney injury was reported before treatment in D1 and during treatment in D2–D4. The clinical timelines are summarised in **Table 2** and the reported findings and outcomes in **Table 3**. The chronology alone does not establish causation.

Discussion

In this series, kidney injury in leishmaniasis followed two patterns. In the three patients with mucocutaneous disease it developed during amphotericin B treatment, whereas in the patient with visceral leishmaniasis it preceded treatment and progressed to kidney failure. Both patterns are recognized: visceral leishmaniasis causes glomerular and tubular involvement and AKI [3, 4], and amphotericin B was the factor most strongly associated with AKI in a large Brazilian cohort [5].

Case D1 had pancytopenia, hepatosplenomegaly, polyuria of up to 9.9 L/day, hyponatraemia, and hypokalaemia within days of admission, before amphotericin B was given. Tubular dysfunction, including distal renal tubular acidosis, is common in chronic visceral leishmaniasis [3], and kidney disease predicts death even in patients treated with liposomal amphotericin B [12]. His progression to kidney failure within a year, without nephrology follow-up, has several possible contributors – the infection, amphotericin B, alcohol use, and unrecognized pre-existing disease – and the absence of a kidney biopsy precludes a definitive explanation. Nevertheless, his course suggests that patients with kidney involvement during visceral leishmaniasis need structured renal follow-up after cure.

Amphotericin B increases tubular cell membrane permeability and causes renal vasoconstriction; the resulting fall in glomerular filtration is partly reversible, and sodium loading is the preventive measure with the best supporting evidence [6, 7]. Case D2 illustrates the management recorded in the notes: switching to the liposomal formulation, prolonging the infusion, and loading with saline. Lipid formulations are less nephrotoxic than the deoxycholate formulation, which was withdrawn from a Brazilian randomized trial in visceral leishmaniasis because of toxicity [13], but liposomal amphotericin B was still associated with AKI in a cohort of patients with mucosal leishmaniasis [8]. In case D2, creatinine had not returned to its initial value 2 months later.

In case D3, AKI coincided with a new diagnosis of HIV infection. Amphotericin B formulations are recommended for leishmaniasis in people living with HIV, in whom relapse is common [2, 14]. Access to dialysis was an additional barrier, because our unit could not admit a new patient with a blood-borne viral infection. The rapid fall in creatinine once the drug was withheld is consistent with the haemodynamic component of amphotericin B nephrotoxicity [6].

Case D4 raises the greatest concern. A record of '34 doses in 7 days' does not match any standard regimen, and the cumulative dose could not be established. Doses differ between conventional and liposomal amphotericin B, and confusion between the two formulations has caused AKI and deaths [15, 16]. Whatever the mechanism, a normal ultrasound scan 8 days earlier, an enlarged right kidney, haematuria, and recovery of urine output point to severe AKI rather than established kidney failure. Labelling him as having stage 5 diabetic CKD after 2 weeks of dialysis, without a baseline creatinine, risked overlooking renal recovery; KDIGO recommends re-evaluating patients 3 months after AKI for resolution or progression to CKD [11].

The series also reflects structural barriers: the lack of dialysis places for patients with blood-borne viral infections (D3), the unavailability of erythropoietin (D4), the absence of nephrology follow-up after referral (D1), and refusal of maintenance dialysis far from home, which prolonged hospitalization (D1). Strengths and limitations. The series documents the renal course in detail and includes 34 months of follow-up for one patient. However, it is retrospective and single-centre and relies on nephrology notes. The formulation and cumulative dose of amphotericin B were not consistently recorded, magnesium and urinary electrolytes were rarely measured, no patient un-

derwent kidney biopsy, and the method used to diagnose mucocutaneous leishmaniasis was not documented in the nephrology notes; it was obtained from the referral records.

Conclusion

The reported cases illustrate kidney injury occurring before or during treatment for leishmaniasis, with diverse subsequent courses. They support careful documentation of drug formulation and dosing, assessment of renal function and electrolytes, and follow-up after acute kidney injury. The contribution of infection, treatment and pre-existing disease cannot be determined definitively from this series.

Ethics and consent: Approval no. 048/2026 dated November 2026 and written publication consent from all four patients.

Authorship Criteria: Raúl Desvars: conceptualization, methodology, original draft, supervision and review and editing; Elena Schupp, Jorge Rojas Godoy, María José López Otaño and Oscar Maciel: data curation and investigation. Guarantor: Raúl Desvars.

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