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Leptospirosis-Associated Acute Kidney Injury Presenting with Polyuria and Hypokalemia-Induced Flaccid Quadriparesis in a Young Farmer: A Case Report

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ABSTRACT

Leptospirosis is a globally distributed zoonotic infection that can cause acute kidney injury (AKI), electrolyte disturbances, and rare neuromuscular complications. Non-oliguric AKI accompanied by polyuria and hypokalemia represents an important but often underrecognized manifestation of leptospiral nephropathy. This case report aimed to describe the clinical presentation, diagnostic evaluation, management, and outcome of leptospirosis-associated AKI complicated by severe hypokalemia and flaccid quadriparesis. A 36-year-old male farmer was evaluated through clinical examination, serial laboratory assessments, serologic testing, urinalysis, electrocardiography, abdominal ultrasonography, urine output monitoring, and muscle strength evaluation. The patient developed marked polyuria of 7,000 mL/24 hours, severe hypokalemia with serum potassium levels decreasing to 2.3 mmol/L, azotemia, and acute flaccid quadriparesis characterized by grade 2 muscle strength and absent deep tendon reflexes. Leptospira IgM was positive, while ultrasonography findings suggested bilateral acute tubulointerstitial nephritis. The findings indicated that renal tubular dysfunction and potassium wasting were key mechanisms contributing to polyuria and neuromuscular impairment. Management included intravenous fluid therapy, controlled potassium and sodium replacement, ceftriaxone administration, and continuous electrolyte and electrocardiographic monitoring. Muscle strength gradually recovered to grade 5, serum potassium normalized to 3.6 mmol/L, urine output decreased, and renal function improved within six days. Early recognition and systematic correction of fluid and electrolyte disturbances can lead to complete recovery from this severe complication of leptospirosis.

INTRODUCTION

Leptospirosis is a bacterial zoonotic infection caused by pathogenic spirochetes of the genus Leptospira, with worldwide distribution and particularly high prevalence in tropical regions. It is estimated to cause approximately 1 million cases and nearly 60,000 deaths annually worldwide. Severe complications include acute kidney (Chancharoenthana et al., 2022) injury (AKI) (Sethi et al., 2025), electrolyte disturbances, and significant neuromuscular manifestations (Atlani et al., 2025).

AKI is a major complication of leptospirosis (Rajapakse, 2022), with incidence varying considerably across geographic regions. A recent systematic review and meta-analysis of studies published between early 2020 and 2025, involving 48 cohorts, reported that the pooled prevalence of AKI among patients with leptospirosis was 49.2%. This burden demonstrated

substantial geographic variation, with the highest proportion reported in South America (67.4%), followed by Europe (48.8%), South Asia (48.3%), and Southeast Asia (45.1%). Mortality among patients with leptospirosis-associated AKI is consistently higher than among those without renal involvement (Faubel et al., 2014). A 2025 meta-analysis reported an overall mortality rate of 8.8% among all leptospirosis (Lin, 2019) cases, increasing to 12.5% among patients who developed AKI, highlighting AKI as an important marker of disease severity and poor prognosis (Younes-Ibrahim et al., 1997).

More detailed analyses have shown that leptospiral AKI commonly presents with oliguria and reduced urine output, occurring in approximately 31.5% of cases, while the requirement for dialysis reaches approximately 14.4%. This pattern represents the typical presentation of severe AKI with oliguria and hyperkalemia, which may require intensive care and renal replacement therapy in endemic regions. However, leptospirosis is also recognized for a relatively "paradoxical" presentation characterized by polyuria and hypokalemia. Recent systematic reviews (2025) and narrative reviews (2023) indicate that non-oliguric AKI is one of the most common patterns in leptospirosis, accounting for approximately 40–70% of AKI cases. The combination of polyuria and hypokalemia is considered a highly suggestive clinical indicator of leptospiral nephropathy (Andrade et al., 2008).

Previous studies have confirmed that leptospirosis (McElroy et al., 2024) causes complex electrolyte disturbances through inhibition of Na^+/K^+-ATPase activity and renal tubular dysfunction. The primary mechanisms underlying neuromuscular manifestations include inhibition of Na^+/K^+-ATPase by leptospiral outer membrane proteins, renal potassium wasting through kaliuresis, increased aldosterone and cortisol activity that promotes potassium loss, and proximal tubular dysfunction. These mechanisms may result in severe hypokalemia, with potassium levels decreasing to approximately 2.0 mEq/L, accompanied by flaccid quadriparesis and areflexia (Mahendran et al., 2014).

The combination of polyuria and hypokalemia, which is strongly suggestive of leptospiral nephropathy, may lead to significant clinical consequences, including hypokalemia-induced flaccid quadriparesis. Case reports and case series have described leptospirosis-associated hypokalemia with flaccid quadriparesis as a rare but well-characterized clinical manifestation. Diagnostic features include rapidly progressive flaccid weakness affecting all four limbs, markedly reduced muscle tone and strength, predominant proximal muscle involvement, generalized loss of deep tendon reflexes without sensory impairment, and rapid clinical progression over a short period. Laboratory findings typically demonstrate low serum potassium levels (often <3.0 mmol/L) associated with acute muscle weakness, increased urinary potassium and sodium excretion reflecting kaliuresis, and electrocardiographic (Wang et al., 2020) abnormalities such as U waves and ST–T changes. Other common causes of acute flaccid paralysis (Araujo et al., 2010), including familial periodic paralysis, severe diarrhea, diuretic use, and overt thyrotoxicosis, should be excluded. Neurological and clinical recovery generally occurs without long-term sequelae when patients receive timely fluid management, appropriate oral and intravenous potassium replacement to restore serum potassium levels to the normal range (3.5–5.5 mEq/L), and appropriate antimicrobial therapy for leptospirosis. Continuous monitoring of serum potassium levels is essential to prevent overcorrection and secondary hyperkalemia.

METHOD

This case report described the clinical course, diagnostic evaluation, and therapeutic management of a 36-year-old male farmer who was admitted with leptospirosis-associated acute kidney injury complicated by non-oliguric polyuria, severe hypokalemia, and flaccid quadriparesis. Data were obtained from the patient's medical records, including clinical history, physical examination findings, and serial laboratory, serologic, and imaging evaluations conducted during hospitalization.

The diagnostic evaluation at admission included complete blood count, serum electrolyte analysis, and electrocardiography (ECG) to assess hypokalemia-related cardiac abnormalities. Serial electrolyte measurements, urinalysis, and serologic testing for Salmonella IgM and Leptospira IgM were performed during hospitalization to identify the causative infection and monitor renal and electrolyte status. Abdominal ultrasonography (Moses & Fernandez, 2022) was conducted to evaluate renal involvement. Twenty-four-hour urine output was monitored serially to characterize the polyuric phase, while serum potassium levels were regularly assessed with continuous ECG monitoring during electrolyte correction.

Therapeutic management was implemented based on the patient's clinical progression and included intravenous fluid therapy, sodium correction using appropriate saline administration, oral and intravenous potassium chloride replacement, and antimicrobial therapy with ceftriaxone for suspected leptospirosis. Potassium replacement was adjusted according to electrolyte monitoring and clinical response. Patient outcomes were evaluated through serial electrolyte measurements, urine output monitoring, and muscle strength assessment using the Medical Research Council (MRC) scale throughout the hospitalization period.

RESULTS AND DISCUSSION

Case Presentation

A 36-year-old male farmer presented with a 4-day history of fever. He reported epigastric abdominal pain with a sensation of fullness, nausea, and frequent vomiting, to the point that he could no longer count the number of episodes per day. The vomitus consisted of yellowish fluid and partially digested food. He also complained of dizziness, generalized weakness, myalgia and arthralgia, and throbbing headache. He denied dysuria, changes in urination, or bowel habit disturbances.

On admission, the patient appeared weak but was fully conscious (compos mentis). Vital signs showed a high-grade fever of 39°C. Physical examination revealed localized tenderness over the left gastrocnemius muscle and epigastric tenderness on palpation, without hepatosplenomegaly or other organomegaly (Chou et al., 2023). Initial laboratory investigations included complete blood count, serum electrolytes, and electrocardiography (ECG). Results demonstrated leukocytosis with a white blood cell count of 19,600/µL, mild hyponatremia, and mild hypokalemia. The ECG showed characteristic U waves and QT interval prolongation in precordial leads V3–V6, findings consistent with hypokalemia (Figure I). Based on the initial clinical and laboratory data in the emergency department, the working diagnosis (Levtchenko et al., 2025) was febrile illness under observation, suspected typhoid fever versus leptospirosis, accompanied by azotemia and electrolyte imbalance.

The patient received intravenous Ringer's lactate for fluid resuscitation and symptomatic medications including intravenous paracetamol, ketorolac, esomeprazole, and ondansetron, along with oral sucralfate syrup. He was admitted to the general medical ward for further evaluation and management.

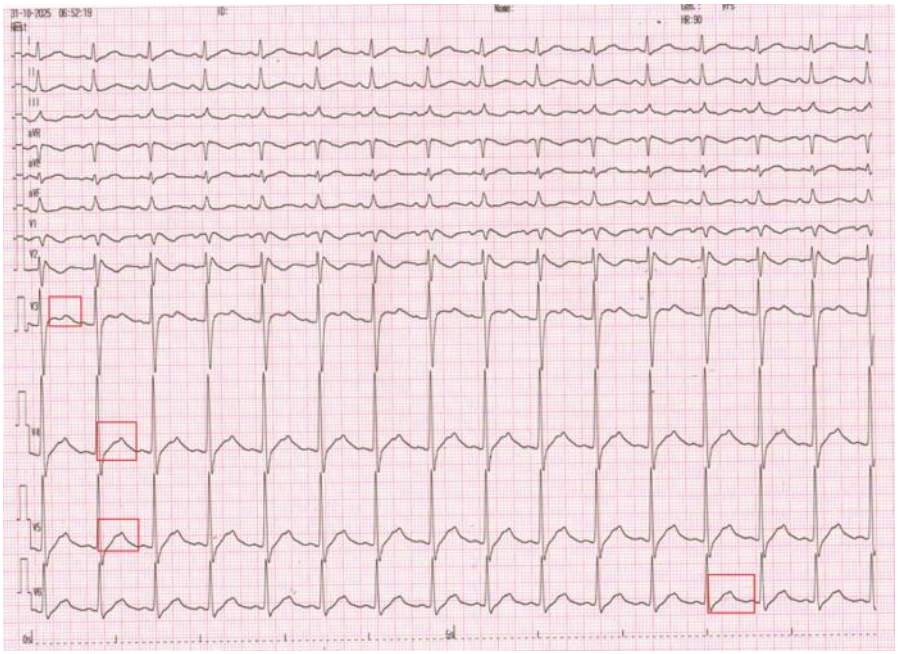

Figure 1. Electrocardiogram demonstrating U waves and QT prolongation consistent with hypokalemia

During the first 24 hours of hospitalization (Zou et al., 2022), the patient's epigastric pain, myalgia, arthralgia, and throbbing headache persisted without significant improvement. On repeat examination, he continued to exhibit tenderness over the left gastrocnemius muscle and epigastric tenderness, but his body temperature had normalized, and no organomegaly was detected. Additional investigations on hospital days 2 and 3 included repeat serum electrolytes, urinalysis, immunology–serology testing for *Salmonella* IgM and leptospira IgM, and abdominal ultrasonography.

On day 2, laboratory tests revealed moderate hyponatremia with serum sodium 123.3 mmol/L, mild hypokalemia with potassium 3.0 mmol/L, hypocalcemia with calcium 1.8 mmol/L, and azotemia with urea 51.7 mg/dL and creatinine 2.93 mg/dL (Table I). Fluid therapy was continued using 3% NaCl 500 mL, and broad-spectrum intravenous ceftriaxone 2 g once daily was initiated (Uribe-Restrepo et al., 2023). Oral electrolyte replacement was started with sodium chloride capsules 500 mg three times daily, potassium supplement (KSR) 1 tablet twice daily, and oral bicarbonate 500 mg three times daily. Urine output monitoring on day 2 showed a 24-hour urine volume of 1,600 mL.

Table 1. Complete blood count, serum biochemistry, renal function, and electrolyte profile (H: high, L: low)

Pemeriksaan	**Hasil**	**Satuan**	**Nilai Rujukan**
Hemoglobin	12.0	g/dl	12 – 16
Leukosit	**19.6 (H)**	**10^3/ mikroliter**	**3.8 – 10.6**
Hematokrit	49.6	%	40 - 52
Eritrosit	4.76	10^6/ mikroliter	4.40 – 5.90
Trombosit	271	10^3/ mikroliter	150 - 440
MCH	29.5	pg	26 - 34
MCHC	35.5	g/dL	32 - 36
MCV	83.2	fL	80 - 100
Pemeriksaan	**Hasil**	**Satuan**	**Nilai Rujukan**
GDS	98	mg/dL	80 - 110
Ureum	**51.7 (H)**	**mg/dL**	**10 - 50**
Creatinine	**2.93 (H)**	**mg/dL**	**0.9 – 1.3**
Kalium	**3.0 (L)**	**mmol/L**	**3.5 – 5.3**
Natrium	**123.3 (L)**	**mmol/L**	**135 - 147**
Chlorida	**72.3 (L)**	**mmol/L**	**98 - 107**
Calcium	**1.8 (L)**	**mmol/L**	**2.15-2.55**

In addition to oral correction, intravenous treatment of hyponatremia was undertaken to safely restore serum sodium toward the normal range. For this 70-kg patient with serum sodium 123.3 mmol/L and a target of 135 mmol/L, hypertonic 3% NaCl was used based on the estimate that 1 mL/kg of 3% NaCl increases serum sodium by approximately 1.6 mEq/L. Accordingly, 500 mL of 3% NaCl was administered at 12 drops per minute, calculated to raise serum sodium by about 11.4 mEq/L. Hyponatremia in this range (120–129 mmol/L) is classified as moderate and requires cautious correction to avoid osmotic demyelination syndrome (ODS). Current recommendations limit sodium increase to 10–12 mEq/L over 24 hours in patients at normal ODS risk, and 4–8 mEq/L per day in high-risk patients (e.g., malnutrition, chronic liver disease, alcoholism).

Immunology–serology testing subsequently showed positive leptospira IgM (Table II), and urinalysis demonstrated pyuria with leukocytes +2 (Table III). Based on these results, symptomatic treatment was continued and ceftriaxone 2 g intravenous once daily was maintained as definitive therapy targeting leptospira.

Table 2. Serologic results for Salmonella IgM and leptospira IgM

IMMUNOLOGI - SEROLOGI

PEMERIKSAAN *Test*	**HASIL** *Result*	**NILAI NORMAL** *Reference*	**SATUAN** *Unit*	**METODE** *Method*
Leptospira IgM	Positif *	Negatif		Immuno Kromatografi
IgM Salmonella	0	**< 2 : Negatif** Tidak menunjukkan infeksi typoid **3 : Borderline** Sulit disimpulkan (meragukan) ulangi pemeriksaan. Jika masih meragukan ulangi pemeriksaan di lain hari. **4-5 : Positif Lemah** Mengindikasikan infeksi typoid akut **6-10 : Positif Kuat** Indikasi kuat untuk infeksi typoid akut.		Tubex Test

On hospital day 3, the patient's condition acutely deteriorated. He developed chills, worsening throbbing headache, nausea, and vomiting of gastric contents, along with profound weakness of all four limbs. Physical examination revealed tachypnea, tachycardia, signs of dehydration, and markedly reduced muscle tone and strength in all extremities, graded as 2 on the Medical Research Council scale (movement possible with gravity eliminated but not against gravity). He was only able to slide his arms along the bed surface and could not lift them against gravity. Deep tendon reflexes were absent globally. There were no urinary or bowel disturbances, no diarrhea, and no signs of respiratory failure. Remarkably, his 24-hour urine output was 7,000 mL, indicating marked polyuria.

Given the combination of fever, positive leptospira serology, severe hypokalemia, polyuria, and acute flaccid quadriparesis, the diagnosis was revised to febrile illness due to leptospirosis, complicated by severe hypokalemia-induced flaccid quadriparesis. Management included aggressive intravenous fluid resuscitation with Ringer's lactate 1,000 mL over 3 hours, followed by maintenance at 40 drops per minute, targeting a 24-hour fluid balance of approximately 0 mL. Fluid administration was adjusted continuously based on clinical assessment for dyspnea or peripheral edema. Symptomatic medications and ceftriaxone 2 g intravenous once daily were continued.

Abdominal ultrasonography showed slightly increased renal cortical echogenicity with preserved corticomedullary differentiation, consistent with bilateral acute nephritis or acute tubulointerstitial nephritis (Figure II).

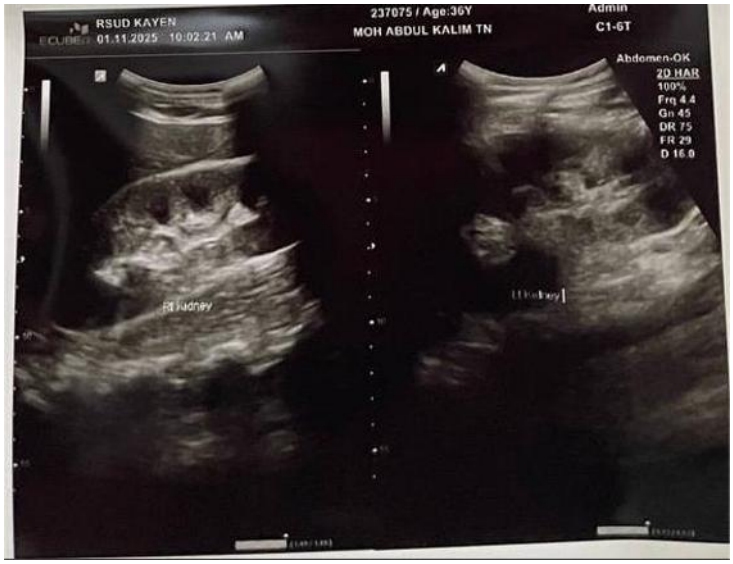

Figure 2. Abdominal ultrasonography demonstrating mildly increased cortical echogenicity with preserved corticomedullary differentiation, suggesting bilateral acute tubulointerstitial nephritis.

Repeat electrolyte testing revealed a further drop in serum potassium to 2.3 mmol/L, classified as severe hypokalemia. This prompted urgent potassium replacement via both oral and intravenous routes. Total potassium deficit was estimated using a standard formula, which recognizes that serum potassium reflects only about 2% of total body stores and that correction must proceed gradually under close monitoring. Intravenous replacement was planned at 10–20 mEq/hour (up to 40 mEq/hour in critical situations, via central access with strict monitoring), along with correction of any associated hypomagnesemia and serial electrolyte checks.

For this 70-kg patient with serum potassium 2.3 mmol/L and a target of 3.5 mmol/L, the total potassium deficit was calculated as:

Deficit = (K target − K measured) × body weight × 0.4

= (3.5 − 2.3) × 70 × 0.4 ≈ 33.6 mEq.

Daily physiological potassium requirement was estimated at 1 mmol/kg, or 70 mEq. Thus, total potassium needed over 24 hours (deficit plus daily requirement) was approximately 103.6 mEq.

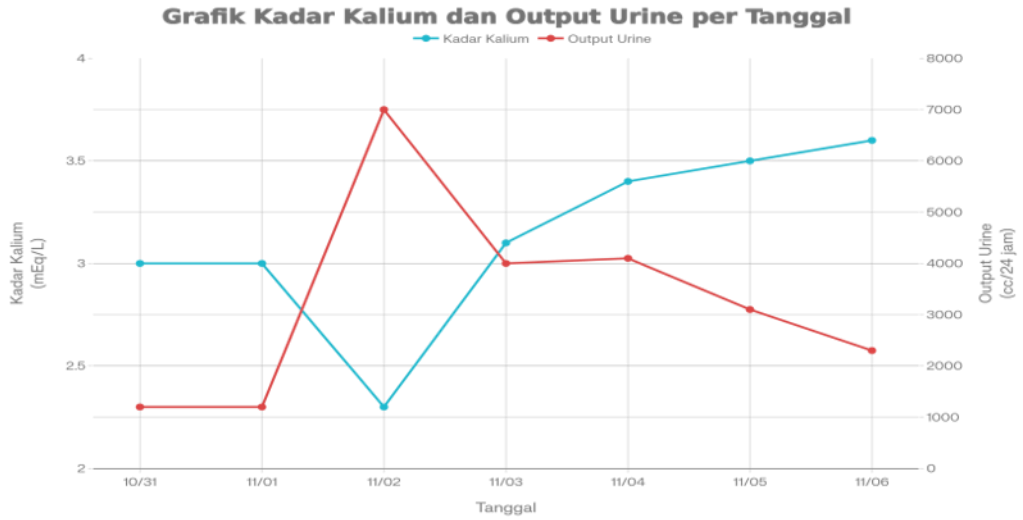

Figure 3. Trend of serum potassium levels and 24-hour urine output from day 1 to day 6, pre- and post-correction.

Intravenous potassium chloride (KCl) was administered at a standard rate of 10 mEq/hour via peripheral venous access. The infusion solution was prepared with 40 mEq KCl in 1,000 mL of 0.9% NaCl, given over 5 hours. This rate was chosen to address the deficit while minimizing the risk of venous irritation, phlebitis, and abrupt potassium shifts that could trigger arrhythmias. Serum potassium was rechecked 4 hours after starting the infusion, accompanied by continuous ECG monitoring. After 4 hours, serum potassium had risen to 3.0 mmol/L without arrhythmias on ECG, and intravenous KCl (2 × 25 mL) was subsequently discontinued. Oral supplementation was continued with potassium chloride 600 mg twice daily and sodium chloride 500 mg three times daily.

Table 3. Urinalysis showing leukocyturia consistent with urinary tract inflammation

Pemeriksaan	**Hasil**	**Nilai Normal**	**Satuan**	**Metode**
Makroskopis	**Result**	**Reference**	**Unit**	**Method**
Makroskopis				
Warna	Kuning Tua	Kuning Muda – Kuning Tua		
Kekeruhan	**Agak Keruh***	**Jernih**		
Berat Jenis	1.025	1.005 – 1.030		
Protein	Negatif	Negatif		
Glukosa	Negatif	Negatif		
Keton	Negatif	Negatif		
Bilirubin	Negatif	Negatif		
Urobilinogen	Normal	Normal		
Nitrit	Negatif	Negatif		
Eritrosit	Negatif	Negatif		

Leukosit	**Positif (++) 2***	**Negatif**	
Ascorbic Acid	Negatif	Negatif	
Mikroskopis			
Epitel Gepeng	Positif (+) 1*	Positif (+) 1	/Lpk
Epitel Bulat	Positif (+) 1*	Positif (+) 1	/Lpk
Epitel Transisional	Negatif	Negatif	/Lpk
Lekosit	**42 – 46**	**< 5**	**/Lps**
Eritrosit	2 – 4*	< 4	/Lps
Kristal Ca Oxalat	Negatif	Negatif	
Kristal Tripel Fosfat	Negatif	Negatif	
Kristal Na Urat	Negatif	Negatif	
Kristal Amorf	Negatif	Negatif	
Bakteri	Positif (+) 1	Positif (+) 1	
Jamur	Negatif	Negatif	
Parasit	Negatif	Negatif	

Urinalysis during this period showed slightly turbid yellow urine, leukocyturia, and elevated microscopic leukocytes (42–46 per high power field), with bacteria present and other parameters within or near normal limits, consistent with tubulointerstitial involvement and infection (Table III).

By hospital day 4, the patient reported improvement in headache, nausea, vomiting, and limb weakness. He was able to lift his arms, and muscle power improved to grade 4 in all extremities, indicating full range of movement against gravity with some resistance but less than normal strength. Serum potassium increased to 3.1 mmol/L, in line with the clinical improvement (Table IV). The 24-hour urine output was 4,100 mL with a fluid balance of −280 mL (Figure III). Fluid and electrolyte management continued with Ringer's lactate at 30 drops per minute, aiming for neutral fluid balance, along with ongoing oral potassium and sodium supplementation and intravenous ceftriaxone 2 g once daily.

On day 5, the patient experienced a brief febrile episode at night, which resolved after intravenous paracetamol. Overall weakness in the arms and legs continued to improve. The 24-hour urine output was 3,100 mL with a fluid balance of −360 mL (Figure III). Serum potassium rose to 3.5 mmol/L. Liver function tests showed normal bilirubin values (total 0.44 mg/dL, direct 0.19 mg/dL, indirect 0.25 mg/dL). Follow-up laboratories demonstrated improving leukocytosis (11,900/µL), correction of hyponatremia (sodium 135.1 mmol/L), normalization of potassium (3.5 mmol/L), normalization of calcium (2.1 mmol/L), and improved renal function (urea 25.2 mg/dL, creatinine 1.64 mg/dL) (Table IV).

Table 4. Serial electrolytes and renal markers from day 1 to day 6, before and after potassium correction

Pemeriksaan elektrolit serial	Hasil	Satuan	Keterangan
K+ hari ke-1	3,0	mmol/L	**Koreksi K+ (per oral) KSR 2x 600 mg**
K+ hari ke-2	3,0	mmol/L	**Koreksi K+ (per oral) KSR 2x 600 mg**
K+ hari ke-3	**2,3**	**mmol/L**	**Koreksi K+ (IV) 10,4 meq/jam**
6 jam post Koreksi	**2,8**	**mmol/L**	**Koreksi K+ (per oral) KSR 2x 600 mg**
K+ hari ke-4	3,1	mmol/L	-
K+ hari ke-5	3,5	mmol/L	-
K+ hari ke-6	3,6	mmol/L	-

By hospital day 6, the patient's clinical condition had stabilized. Muscle strength was fully restored (grade 5) in all extremities, and he could perform normal movements without residual weakness. The 24-hour urine output was 2,300 mL with a fluid balance of −53 mL (Figure 3). Electrolytes showed potassium 3.6 mmol/L, sodium 135.1 mmol/L, and calcium 2.1 mmol/L, with further improvement of renal markers (urea 25.2 mg/dL, creatinine 1.64 mg/dL). After completion of six days of inpatient therapy—including intravenous ceftriaxone 2 g daily, structured potassium and sodium replacement, fluid resuscitation, and supportive care—the patient was discharged home in good clinical condition.

This case highlights a characteristic yet clinically under-recognized form of leptospirosis-associated acute kidney injury (Sethi et al., 2025) (AKI) non-oliguric AKI with marked polyuria, severe hypokalemia, and hypokalemia-induced flaccid quadriparesis. The patient's presentation demonstrates how leptospira-related tubular dysfunction can produce a paradoxical pattern of excessive urine output and potassium wasting, in contrast to the more typical oliguric, hyperkalemic AKI seen in other causes of acute renal failure.

Leptospirosis-related polyuria arises from several interacting pathophysiological mechanisms. First, leptospiral glycolipoproteins act as potent inhibitors of renal Na^+/K^+-ATPase. When leptospira proliferate and are subsequently killed by the host immune response, glycolipoproteins are released into the circulation and reach the kidney, where they inhibit Na^+/K^+-ATPase activity throughout the nephron. This impairs active sodium transport in proximal tubular cells and disrupts the osmotic gradient that normally drives water reabsorption via aquaporin-1. As a result, substantial amounts of sodium and water remain in the tubular lumen and are excreted, contributing to osmotic diuresis and polyuria.

Immunohistochemical studies from human and experimental leptospirosis have shown reduced expression of the proximal tubular sodium–hydrogen exchanger NHE3 and aquaporin-1, along with increased expression of the Na-K-2Cl cotransporter (NKCC2) in the thick ascending limb of the loop of Henle. This constellation produces severe impairment of sodium and water handling, elevates fractional excretion of sodium and potassium, and leads to high-volume, dilute urine. In the present case, this mechanism is reflected by profound polyuria (7,000 mL/24 h) and significant urinary electrolyte losses.

A second major mechanism involves inflammatory injury and acute tubulointerstitial nephritis. Leptospiral outer membrane proteins such as LipL32 activate Toll-like receptor-2 on tubular epithelial cells, triggering intracellular (Gonçalves-de-Albuquerque et al., 2023) inflammatory cascades with production of cytokines including TNF-α, IL-1, IL-6, and IL-10. This leads to tubulointerstitial nephritis characterized by mononuclear cell infiltration, tubular damage, and mixed patterns of acute tubular necrosis and interstitial inflammation. In this patient, abdominal ultrasound demonstrating increased cortical echogenicity with preserved corticomedullary differentiation, together with leukocyturia on urinalysis, supported the diagnosis of bilateral acute tubulointerstitial nephritis. Tubular damage further impairs reabsorptive capacity, amplifying polyuria and electrolyte wasting.

A third mechanism is acquired nephrogenic (Etish et al., 2014) diabetes insipidus (NDI) with vasopressin resistance. Experimental studies in animal models have shown that leptospiral glycolipoproteins reduce aquaporin-2 expression in the inner medullary collecting duct and decrease osmotic water permeability by up to 40%. Even in the presence of elevated endogenous vasopressin, collecting duct cells respond poorly, leading to excretion of large volumes of hypotonic urine. Clinically, this manifests as non-oliguric AKI with hyposthenuria, polyuria, polydipsia, and progressive electrolyte depletion, as seen in this patient.

The severe hypokalemia observed in this case is a direct consequence of renal potassium wasting superimposed on high urinary flow rates. Leptospirosis increases distal potassium secretion through several mechanisms, inhibition of Na^+/K^+-ATPase alters tubular sodium handling, activation of mineralocorticoid pathways (aldosterone and cortisol) promotes kaliuresis, and tubular dysfunction reduces potassium reabsorption. The combination of increased distal sodium delivery and aldosterone-mediated potassium secretion markedly enhances urinary potassium loss. In this patient, serum potassium fell to 2.3 mmol/L with high urinary output, consistent with severe renal potassium wasting rather than extrarenal loss.

Neuromuscular manifestations such as flaccid quadriparesis represent a rare but well-described complication of hypokalemia in leptospirosis. Extreme reductions in serum potassium disrupt membrane potential and impair action potential generation in skeletal muscle, leading to symmetric flaccid weakness, predominantly affecting the proximal muscles, with loss of deep tendon reflexes and preserved sensation. Electrocardiographic changes, including U waves and QT prolongation, reflect concomitant myocardial involvement and signal increased risk of arrhythmia. In this case, acute onset generalized flaccid quadriparesis, absent reflexes, severe hypokalemia, high urinary potassium loss, and typical ECG findings, together with positive leptospira serology and exclusion of alternative etiologies, fulfilled criteria for hypokalemia-induced flaccid quadriparesis due to leptospirosis.

Management of such cases must focus on four pillars aggressive but carefully monitored fluid resuscitation, structured potassium and electrolyte replacement, definitive antimicrobial therapy, and close cardiorenal surveillance. In this patient, potassium deficit was calculated and corrected with a combination of intravenous and oral supplementation, at a controlled rate of approximately 10 mEq/hour via peripheral access to minimize the risk of arrhythmias and phlebitis. Serial electrolyte measurements and continuous ECG monitoring allowed timely adjustment of therapy and prevention of rebound hyperkalemia. Targeted antibiotic therapy with intravenous ceftriaxone, along with supportive measures, addressed the underlying leptospiral infection.

The clinical course—with gradual improvement in muscle strength from grade 2 to full recovery (grade 5), normalization of potassium from 2.3 to 3.6 mmol/L, and progressive recovery of renal function—illustrates that even severe neuromuscular and renal complications of leptospirosis can be fully reversible when recognized early and managed systematically. This is particularly relevant in resource-limited settings, where access to dialysis and intensive care may be constrained and optimal fluid–electrolyte management can significantly influence outcomes.

This case adds several important insights to the existing literature. First, it reinforces that polyuria with hypokalemia in a febrile patient from an endemic area should strongly raise suspicion for leptospiral nephropathy, even before serologic confirmation. Second, it shows that severe hypokalemia-induced flaccid quadriparesis can be a dramatic but reversible neuromuscular manifestation of leptospirosis-associated AKI, provided that potassium deficit is promptly recognized and systematically corrected. Third, it offers a practical example of structured potassium replacement (calculation of total deficit, controlled infusion rates, and close monitoring) that can be applied in district hospitals with limited resources, potentially avoiding progression to life-threatening arrhythmias and permanent neurological disability.

For clinicians practicing in leptospirosis-endemic regions, this case underscores the importance of routinely assessing urine output, serum electrolytes, and ECG in patients with suspected leptospirosis, and of considering non-oliguric AKI with polyuria as a frequent and clinically meaningful pattern.

CONCLUSION

This case illustrates a distinctive presentation of leptospirosis-associated acute kidney injury characterized by non-oliguric AKI with marked polyuria, severe hypokalemia, and hypokalemia-induced flaccid quadriparesis in a young farmer from an endemic area. The combination of polyuria, electrolyte loss, and flaccid paralysis reflects underlying leptospiral tubular dysfunction, impaired urinary concentrating ability, and renal potassium wasting.

Timely recognition of this clinical pattern, combined with appropriate potassium replacement, careful fluid and electrolyte management, and ceftriaxone therapy, resulted in complete recovery of muscle strength and substantial improvement in renal function within six days of hospitalization. This favorable outcome demonstrates that severe neuromuscular complications of leptospirosis can be reversible when diagnosed and managed promptly with systematic supportive care.

This report highlights the importance of clinical awareness regarding non-oliguric AKI and hypokalemia in patients with leptospirosis and provides a practical management approach applicable to resource-limited healthcare settings.

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