

Dynamic Renal Deterioration in Flood-Associated Severe Leptospirosis Multiorgan Dysfunction

Original Article

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Abstract

Severe leptospirosis remains an under-recognized cause of rapidly progressive multiorgan dysfunction in tropical, flood-prone regions, where delayed risk stratification contributes to preventable renal failure and mortality. Acute kidney injury (AKI) represents a pivotal determinant of outcome, yet its clinical trajectory may be dynamic and misleading. This case report aimed to characterize the temporal clinical and biochemical progression of severe leptospirosis complicated by Stage 3 AKI and to identify practical implications for early bedside recognition in endemic settings. We conducted a retrospective case report in accordance with CARE guidelines. Clinical, epidemiological, laboratory, and radiological data were systematically extracted from hospital records. Diagnosis was established based on compatible clinical manifestations, documented floodwater exposure with skin barrier disruption, positive serology, structured scoring using Modified Faine's Criteria, and AKI staging according to KDIGO guidelines. Serial laboratory monitoring enabled assessment of organ involvement and disease evolution. The patient developed cholestatic jaundice, thrombocytopenia, pulmonary inflammatory changes, profound electrolyte imbalance, and rapidly progressive renal dysfunction fulfilling KDIGO Stage 3 criteria. Notably, transient improvement in serum creatinine preceded abrupt deterioration requiring hemodialysis, underscoring the non-linear course of leptospirosis AKI. Integrated supportive management and tailored antimicrobial therapy resulted in subsequent stabilization. This report highlights the need for dynamic renal monitoring and early escalation strategies in high-risk patients with environmental exposure. Incorporating structured diagnostic scoring and continuous biochemical surveillance into frontline care may improve prognostic accuracy and reduce severe renal complications in climate-vulnerable populations.

Keywords: Acute Renal Failure, Environmental Exposure, Renal Replacement Therapy, Systemic Inflammation, Zoonotic Infection.

1. Introduction

Leptospirosis is a globally distributed, climate sensitive zoonotic infection caused by pathogenic *Leptospira* species. Despite being preventable and treatable, it remains a major cause of morbidity and mortality in tropical and subtropical regions. Recent global burden analyses confirm that leptospirosis continues to disproportionately affect low and middle income countries, where environmental exposure, limited sanitation infrastructure, and constrained diagnostic capacity amplify transmission and delay case recognition (Haake & Levett, 2014; Rajapakse et al., 2015). Leptospirosis incidence is strongly linked to environmental exposures such as floodwater contact and contaminated soil and water sources, particularly in tropical regions (Baharom et al., 2024). Severe disease manifestations



including jaundice, pulmonary hemorrhage, and acute kidney injury (AKI) are associated with significantly increased mortality, particularly when diagnosis and treatment are delayed.

In the past five years, emerging evidence has reinforced the role of climate variability and extreme weather events in shaping leptospirosis epidemiology. Systematic reviews and modeling studies demonstrate that heavy rainfall, flooding, and urban environmental degradation significantly increase the risk of outbreaks by facilitating contact between humans and contaminated water sources (Mwachui et al., 2015; Torgerson et al., 2015). These findings position leptospirosis as a sentinel disease within the broader context of climate change associated infectious threats Indonesia, as a tropical archipelagic country with high rainfall, recurrent flooding, and ongoing challenges in sanitation and waste management, faces a high risk of leptospirosis. The disease has been reported across multiple provinces, particularly in flood-prone areas (Yuniasih et al., 2022). According to a World Health Organization report in 2019, Indonesia recorded 920 cases of leptospirosis with 122 deaths across nine provinces. Data from the Early Warning and Response System indicate a consistent increase in suspected leptospirosis cases during the rainy season over the past three years, with a significant surge observed at the onset of the rainy season during the 2023-2025 period. In 2025, an increase in cases was also noted on Java Island, with the highest numbers reported in Central Java (1,273 cases), followed by the Special Region of Yogyakarta (876 cases), East Java (601 cases), West Java (371 cases), and Banten (186 cases) (Ministry of Health Republic of Indonesia, 2025).

Despite strengthened surveillance systems and laboratory capacity-building initiatives, early diagnosis remains difficult due to nonspecific clinical presentation. Early-stage leptospirosis often mimics dengue, malaria, or other tropical febrile illnesses, contributing to misclassification and delayed therapy (Haake & Levett, 2014). Delayed recognition increases the risk of progression to multiorgan dysfunction and Stage 3 AKI, a complication strongly associated with increased need for renal replacement therapy and mortality (Daher et al., 2010; Yang, 2018). In Brazil, flood events have been associated with significant increases in leptospirosis incidence and mortality, reinforcing the climate-disease linkage (Michelon et al., 2025). Long-term outcome studies suggest that leptospirosis-associated AKI can predispose patients to chronic kidney disease and increased long-term mortality (Chang et al., 2022). In Indonesia, where leptospirosis remains endemic and access to early diagnosis and long-term renal monitoring may vary, these outcomes could be more pronounced, although further local studies are needed to confirm this.

Thus, the core clinical and public health problem lies in the gap between epidemiological risk awareness and early bedside recognition of severe disease. Bridging this gap requires improved clinical vigilance, standardized risk stratification approaches, and strengthened integration between surveillance data and frontline care. This study describes the clinical manifestations, laboratory findings, multiorgan involvement, and outcomes of severe leptospirosis complicated by Stage 3 acute kidney injury, aiming to inform earlier diagnostic frameworks and optimized management strategies in climate-vulnerable endemic settings.

2. Case Presentation

A 28-year-old previously healthy male was admitted to the Emergency Department of K.R.M.T. Wongsonegoro Regional Hospital, Semarang, Indonesia, with a 14-day history of intermittent high-grade fever (maximum recorded temperature 39.5 °C), generalized weakness, and progressive headache. The symptoms were accompanied by abdominal pain, persistent nausea, and recurrent vomiting, which markedly worsened within 24 hours prior to

admission (>20 episodes, non-projectile, containing gastric contents). Four days before presentation, he developed jaundice and dark-colored urine with reduced output.

The patient reported occupational exposure to floodwaters approximately 15 days prior to symptom onset. He had worked in inundated areas without personal protective equipment. Notably, he had a pre-existing open wound on the right calf for one month prior to illness onset. The wound initially measured approximately 1 cm with purulent discharge and progressively enlarged. During flood exposure, the wound was covered only with a single-layer adhesive dressing. After removal of the dressing, he noticed wound enlargement and increasing localized pain (pain score 6/10), followed by the onset of fever.

He had sought medical care twice at primary healthcare facilities and received unspecified medications without clinical improvement. He denied previous similar illness, recent travel outside the region, or known sick contacts. There was no personal or family history of hypertension, diabetes mellitus, cardiovascular disease, or chronic kidney disease.

2.1. Physical Examination

On admission, the patient appeared acutely ill and markedly weak. Vital signs revealed blood pressure 119/55 mmHg, heart rate 100 beats per minute, respiratory rate 22 breaths per minute, body temperature 39.5 °C, and oxygen saturation 98% on room air. General examination demonstrated scleral icterus and generalized jaundice. Pulmonary auscultation revealed fine bilateral inspiratory crackles without signs of respiratory distress. Abdominal examination showed diffuse tenderness without guarding or organomegaly. No clinical hepatosplenomegaly was detected. Examination of the right lower extremity revealed a round ulcerative wound measuring 3 × 2 cm with a depth of approximately 0.5 cm, erythematous base, and no active purulent discharge. Peripheral perfusion was preserved, with warm extremities and capillary refill time <2 seconds.

2.2. Investigations

2.2.1. Radiological Findings

Anteroposterior chest radiography demonstrated normal cardiac size and position with a cardiothoracic ratio <50%. Increased bronchovascular markings and minimal bilateral patchy opacities were observed, suggestive of inflammatory or infectious pulmonary involvement. There was no radiographic evidence of pleural effusion. The diaphragms, costophrenic angles, bony thorax, and soft tissues were unremarkable.

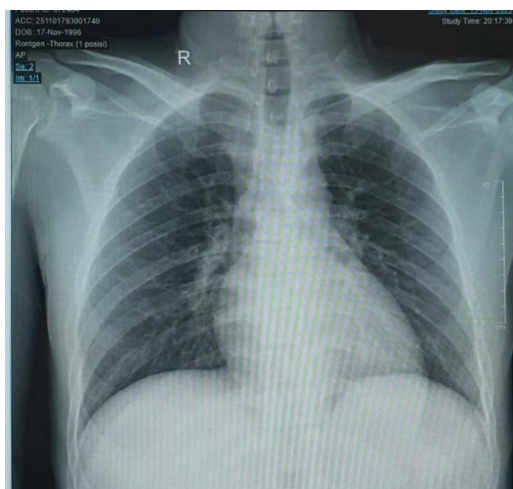


Figure 1. AP Chest X-Ray of the Patient

2.2.2. Laboratory Findings

Initial laboratory investigations revealed severe acute kidney injury, with markedly elevated blood urea nitrogen (297.7 mg/dL) and serum creatinine (8.02 mg/dL). Electrolyte disturbances included severe hyponatremia (116 mmol/L) and hypocalcemia (1.02 mmol/L). Thrombocytopenia was present ($55 \times 10^3/\mu\text{L}$). Liver function tests demonstrated pronounced direct hyperbilirubinemia (total bilirubin 23.31 mg/dL; direct bilirubin 17.35 mg/dL), consistent with a cholestatic pattern frequently observed in severe leptospirosis.

On hospital day 2, serological testing for *Leptospira* returned positive, confirming the diagnosis. Leukocytosis subsequently increased to $25.8 \times 10^3/\mu\text{L}$, indicating an escalating systemic inflammatory response. Despite transient biochemical improvement, renal function deteriorated again on day 6, with serum creatinine rising to 11.8 mg/dL, fulfilling the criteria for stage 3 acute kidney injury according to KDIGO classification.

Overall, the constellation of clinical, radiological, and laboratory findings was consistent with severe leptospirosis complicated by multiorgan dysfunction, including hepatic involvement, thrombocytopenia, pulmonary inflammatory changes, and stage 3 acute kidney injury.

Table 1. Patient Laboratory Results During Treatment

Parameter	Reference Value	Examination					
		Day					
		Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Glucose (POCT)	70 - 110 mg/dL	-	73	-	96	-	-
Random Blood Glucose	70 - 140 mg/dL	80	150	-	-	-	-
Calcium	1.35 - 1.15 mmol/L	1.02	1.22	1.18	1.23	-	1.18
Potassium	3.50 - 5.0 mmol/L	4.00	3.10	3.60	3.80	-	3.60
Sodium	135.0 - 147.0 mmol/L	116.0	132.0	133.0	132.0	-	133.0
Creatinine	0.6 - 1.1 mg/dl	8.02	3.53	-	0.99	1.46	11.8
Urea	17.0 - 43.0 mg/dl	297.7	161.4	-	41.9	55.5	26.20
Hemoglobin	13.2 - 17.3 mg/dl	10.6	10.3	11.8	9.4	-	101
Hematocrit	40 - 52 %	28.90	27.80	31.70	26.20	-	3.31
Thrombocyte	150 - 400 /uL	55	74	101	100	-	17.5
Erythrocyte	4.7 - 6.1 /uL	3.98	4.13	4.29	3.49	-	-
Leukocyte	3.8 - 10.6 /uL	8.8	10.9	16.9	25.8	-	-
Indirect Bilirubin	0.0 - 0.65 /uL	-	5.96	-	-	-	-
HIV I Reagent	Non reactive	-	Non reactive	-	-	-	-
Anti HCV	<1.00:Negative>=1	-	0.03	-	-	-	-
HBsAg Qualitative	Negative	-	Negative	-	-	-	-
Direct Bilirubin	0.0 - 0.35 mg/dL	-	17.35	-	-	-	-
Total Bilirubin	0.00 - 1.00 mg/dL	-	23.31	-	-	-	-
SGPT	0 - 50	-	120	-	-	53	-
SGOT	0 - 50	-	109	-	-	43	-
Leptospira Serology	Negative	-	Positive	-	-	-	-
Procalcitonin	-<0.5 ng/L: Normal	-	-	-	-	0.5ng/L	-

2.3. Therapy

Based on the patient's history, physical examination, and investigations, a diagnosis of severe leptospirosis (Weil's disease) was established, characterized by jaundice, acute kidney injury, and thrombocytopenia. Management consisted of supportive and specific therapies. The patient received intravenous fluids with 0.9% sodium chloride at a rate of 10 drops per minute, hypertonic saline (3% NaCl) administered in two cycles, and intravenous AHEP

infusion at one bag per day. Additional treatments included intravenous sodium bicarbonate (0.1 mEq), intravenous ranitidine 50 mg every 12 hours, oral paracetamol 500 mg every 8 hours, intravenous mecobalamin 500 µg once daily, and oral ursodeoxycholic acid 250 mg every 8 hours. A double-lumen hemodialysis catheter was inserted to facilitate renal replacement therapy.

Empirical antibiotic therapy was initiated with intravenous ceftriaxone at a dose of 2 g every 12 hours. However, the patient developed an allergic reaction manifested by blistering of the left hand and erythematous patches on the hands and feet, necessitating discontinuation of ceftriaxone. Antibiotic therapy was subsequently switched to meropenem 1 g every 8 hours, which was administered for two days. The patient underwent hemodialysis twice during the hospitalization period.

3. Methods

3.1. Study Design and Data Collection

This study was conducted as a single-patient case report in accordance with the CARE (CAse REport) reporting guidelines. Clinical data were obtained retrospectively from the patient's electronic and written medical records at K.R.M.T. Wongsonegoro Regional Hospital, Semarang, Indonesia. Data extraction included demographic characteristics, exposure history, clinical presentation, physical examination findings, laboratory results, radiological findings, therapeutic interventions, and clinical outcomes. All laboratory examinations were performed in the hospital's central clinical laboratory using standardized automated analyzers following institutional protocols. Radiographic imaging was interpreted by a certified radiologist. Serological testing for *Leptospira* was conducted using validated hospital laboratory procedures, including specify method, e.g., ELISA, Microscopic Agglutination Test (MAT), or rapid diagnostic test, to ensure reproducibility. Written informed consent was obtained from the patient for publication of clinical data and imaging findings.

Permission to conduct this study was obtained from RSD K.R.M.T Wongsonegoro, Semarang. As this report involves a single patient and does not include experimental intervention, formal ethical approval was waived. Written informed consent was obtained from the patient prior to data collection and publication.

3.2. Diagnostic Criteria and Clinical Definitions

The diagnosis of leptospirosis was established based on compatible clinical presentation and confirmed by positive serological testing for *Leptospira*, in accordance with World Health Organization (WHO) diagnostic recommendations for leptospirosis (Akbar et al., 2024). Severe leptospirosis (Weil's disease) was defined by the presence of jaundice, renal impairment, and hematologic abnormalities, consistent with established clinical criteria in the literature (Haake & Levett, 2014). Acute kidney injury (AKI) was classified according to the Kidney Disease: Improving Global Outcomes (KDIGO) Clinical Practice Guidelines, which define Stage 3 AKI as a ≥ 3 -fold increase in serum creatinine from baseline or serum creatinine ≥ 4.0 mg/dL, or initiation of renal replacement therapy. Electrolyte abnormalities were interpreted based on institutional laboratory reference ranges. Thrombocytopenia was defined as platelet count $< 150 \times 10^3/\mu\text{L}$.

3.3. Clinical and Laboratory Monitoring

Clinical and laboratory parameters were monitored daily throughout the 6 day hospitalization period. The measurements included:

1. Serum creatinine and blood urea nitrogen (renal function)
2. Serum electrolytes (sodium, potassium, calcium)
3. Complete blood count
4. Liver function tests (total and direct bilirubin, AST, ALT)
5. Inflammatory markers (leukocyte count, as part of the complete blood count, and procalcitonin (specify whether measured daily or selectively)

Radiological evaluation was performed using anteroposterior chest radiography on admission to assess pulmonary involvement. Laboratory trends were recorded chronologically from hospital day 1 to day 6, as summarized in Table 1.

3.4. Therapeutic Procedures and Outcome Evaluation

Management decisions were guided by current clinical recommendations for severe leptospirosis and AKI management (Haake & Levett, 2014). Empirical antibiotic therapy was initiated with intravenous ceftriaxone, consistent with standard treatment guidelines for severe leptospirosis. Following an allergic reaction, antibiotic therapy was escalated to meropenem based on clinical judgment and patient safety considerations.

Renal replacement therapy (intermittent hemodialysis) was initiated after fulfillment of KDIGO Stage 3 AKI criteria and worsening biochemical parameters. A double-lumen catheter was inserted under sterile conditions to facilitate dialysis access. Treatment response was evaluated based on:

1. Improvement or deterioration in serum creatinine and urea levels
2. Correction of electrolyte abnormalities
3. Resolution or progression of thrombocytopenia
4. Changes in bilirubin levels
5. Overall clinical stability

The primary clinical outcome assessed was the progression of renal dysfunction and need for renal replacement therapy. Secondary outcomes included hematologic recovery and improvement of hepatic dysfunction.

4. Results and Discussion

4.1. Findings

A 28-year-old previously healthy male was admitted with a 14-day history of fever, systemic symptoms, and progressive gastrointestinal complaints, followed by the development of jaundice and oliguria. A history of occupational exposure to floodwater and a pre-existing open wound suggested a high risk for leptospiral infection. On admission, laboratory evaluation revealed severe acute kidney injury, with markedly elevated blood urea nitrogen (297.7 mg/dL) and serum creatinine (8.02 mg/dL), accompanied by significant electrolyte disturbances, including severe hyponatremia (116 mmol/L) and hypocalcemia (1.02 mmol/L). Thrombocytopenia was also present ($55 \times 10^3/\mu\text{L}$). Liver function tests demonstrated marked direct hyperbilirubinemia (total bilirubin 23.31 mg/dL; direct bilirubin 17.35 mg/dL), indicating significant hepatic involvement.

Serological testing for *Leptospira* returned positive on hospital day 2, confirming the diagnosis. Leukocyte counts showed a progressive increase, reaching $25.8 \times 10^3/\mu\text{L}$, reflecting an escalating systemic inflammatory response. Chest radiography demonstrated mild bilateral patchy opacities, suggestive of pulmonary inflammatory involvement without pleural effusion. During hospitalization, renal function initially showed transient improvement, with serum creatinine decreasing to 0.99 mg/dL by day 4. However, this was followed by a marked deterioration, with creatinine rising to 11.8 mg/dL on day 6, fulfilling the criteria for stage 3 acute kidney injury according to KDIGO guidelines. The patient required renal replacement therapy and underwent hemodialysis twice during the hospital stay.

Hematologic parameters demonstrated partial recovery, with platelet counts increasing from $55 \times 10^3/\mu\text{L}$ to $100 \times 10^3/\mu\text{L}$ by day 4, although fluctuations were observed thereafter. Liver enzyme levels showed gradual improvement, with reductions in AST and ALT levels, while hyperbilirubinemia persisted. Overall, the patient exhibited a dynamic clinical course characterized by multiorgan involvement, including severe renal impairment, hepatic dysfunction, hematologic abnormalities, and mild pulmonary involvement. Temporal changes in laboratory parameters during hospitalization are summarized in Table 1.

4.2. Discussion

Leptospirosis remains a major climate-sensitive zoonosis with significant morbidity in tropical regions. Recent global analyses indicate that severe manifestations continue to be under-recognized in endemic low- and middle-income countries, particularly during periods of heavy rainfall and flooding (Costa et al., 2015; Haake & Levett, 2014). The present case illustrates a classical yet clinically severe presentation following direct floodwater exposure in a patient with an untreated open wound, a well-established portal of entry for pathogenic *Leptospira* species.

Pathophysiologically, *Leptospira* disseminates hematogenously during the leptospiremic phase, leading to endothelial dysfunction, capillary leakage, and systemic inflammatory activation. Host-pathogen interactions in leptospirosis involve innate immune modulation by bacterial outer membrane proteins, influencing disease severity (Varma et al., 2023). The immunopathogenesis of severe leptospirosis reflects both endothelial dysfunction and immune dysregulation (Anggraini & Lenggogeni, 2024). Recent evidence suggests that severe disease is driven by a combination of direct endothelial injury and dysregulated host immune responses, resulting in multiorgan dysfunction (Rajapakse et al., 2015). This mechanism explains the constellation of findings in our patient, including thrombocytopenia, hyperbilirubinemia, pulmonary inflammatory changes, and acute kidney injury (AKI). Pulmonary involvement is increasingly recognized as a key severe manifestation influencing mortality (Rajapakse et al., 2025). Clinical predictors of severe disease, such as thrombocytopenia and jaundice, have been validated in hospitalized cohorts (Wickramasinghe et al., 2025).

Kidney involvement, including AKI and the need for dialysis, remains a frequent and severe complication of leptospirosis, with pooled incidence rates nearing 50% in some cohorts (Sethi et al., 2025). Biomarker studies are exploring novel indicators for early detection of kidney injury in leptospirosis, although no biomarker is yet standard in clinical practice (Uribe-Restrepo et al., 2023). Acute kidney injury represents one of the most critical complications of severe leptospirosis. Contemporary studies emphasize that leptospiral AKI is multifactorial, involving tubular epithelial injury, interstitial nephritis, hypovolemia, rhabdomyolysis, and immune-mediated damage (Daher et al., 2010). Unlike classical ischemic AKI, leptospiral AKI frequently presents with non-oliguric patterns and electrolyte disturbances such as hyponatremia and hypokalemia, as observed in this patient. The

progression to KDIGO stage 3 AKI by day six, despite transient biochemical improvement, reflects the dynamic and unpredictable course described in recent nephrology-focused analyses (Yang, 2018).

The marked direct hyperbilirubinemia observed in this case is consistent with the cholestatic pattern characteristic of Weil’s disease. Severe leptospirosis-associated jaundice is not solely attributable to hepatocellular necrosis but is thought to result from intrahepatic cholestasis secondary to hepatocellular dysfunction and endothelial injury (Rajapakse et al., 2015). Severe leptospirosis cases often require intensive care and may be complicated by hepatic dysfunction and thrombocytopenia, supporting aggressive supportive management (Yuniasih et al., 2022). This pathophysiological distinction explains why transaminase elevations are often modest relative to bilirubin levels, a pattern also evident in our laboratory findings (Table 1).

Table 2. Modified Faine’s Criteria and Patient Clinical

A. Clinical feature	Score	Score patient
Headache	2	2
Fever	2	2
Fever > 39 °C	2	2
Conjunctival suffusion	4	0
Meningism	4	0
Myalgia	4	4
Conjunctival suffusion + meningism + myalgia	10	4
Jaundice	1	1
Albuminuria / nitrogen retention	2	2
Haemoptysis / dyspnoea	2	0
B. Epidemiological Factors		
Rainfall / heavy rain exposure	5	5
Contact with contaminated environment (water/soil)	4	4
Animal contact	1	0
C. Bacteriological and Laboratory Findings		
Isolation of <i>Leptospira</i> in culture – Diagnosis certain	–	-
PCR positive	25	-
ELISA IgM positive	15	-
SAT positive	15	-
Other rapid serological tests	15	15
MAT – single high titer	15	-
MAT – rising titer / seroconversion (paired sera)	25	-

Diagnostic confirmation in this case was supported by positive serology and a high Modified Faine’s Criteria score. As shown in Table 2, the total score of 42 significantly exceeded the ≥ 25 threshold suggestive of confirmed leptospirosis. The integration of epidemiological exposure (flood contact), compatible clinical features (fever, myalgia, jaundice, renal dysfunction), and positive serological findings strengthens diagnostic validity. Recent evaluations continue to support the utility of the Modified Faine’s Criteria in endemic settings, particularly where access to PCR or paired MAT testing is limited (Agampodi et al., 2012).

From a therapeutic perspective, early antibiotic administration remains the cornerstone of management. Antibiotic regimen comparisons highlight the need for tailored therapy in severe leptospirosis (Brett-Major & Coldren, 2012; Panaphut et al., 2003). Updated clinical

reviews recommend intravenous penicillin G, ceftriaxone, or cefotaxime for severe disease, with evidence suggesting reduced progression to organ failure when initiated promptly (Haake & Levett, 2015). In our patient, empirical ceftriaxone was initiated but discontinued due to allergic reaction, necessitating escalation to meropenem. Although carbapenems are not first-line therapy, they may be considered in cases with severe systemic inflammation or diagnostic uncertainty. Literature reviews demonstrate that adjunct therapies, including corticosteroids in select settings, have been explored but remain inconclusive (Petakh et al., 2024).

Renal replacement therapy (RRT) plays a pivotal role in severe leptospiral AKI. Contemporary cohort studies demonstrate that early initiation of hemodialysis in stage 3 AKI improves metabolic stabilization and may reduce mortality in severe cases (Daher et al., 2010). The need for two sessions of hemodialysis in this patient underscores the severity of renal involvement and aligns with reported patterns of severe multiorgan leptospirosis.

Importantly, this case also highlights the intersection between climate exposure and clinical severity. Increasing rainfall variability and recurrent flooding in tropical regions have been associated with rising incidence and severity of leptospirosis outbreaks (Costa et al., 2015). The presence of an untreated open wound during flood exposure likely amplified bacterial inoculation and systemic dissemination, emphasizing the importance of preventive strategies in climate-vulnerable communities. Overall, this case reinforces three critical clinical implications:

1. Flood exposure with skin breach should immediately raise suspicion for leptospirosis in endemic regions.
2. Early biochemical improvement does not preclude subsequent deterioration to stage 3 AKI.
3. Integration of epidemiological risk assessment and bedside recognition is essential to prevent delayed escalation of care.

5. Conclusion

This case highlights severe leptospirosis (Weil's disease) complicated by multiorgan dysfunction and progression to KDIGO stage 3 acute kidney injury following documented floodwater exposure and skin barrier disruption. The main finding of this report is the dynamic and unpredictable clinical course, characterized by transient biochemical improvement followed by rapid renal deterioration requiring renal replacement therapy. The constellation of epidemiological risk factors, cholestatic hyperbilirubinemia, thrombocytopenia, pulmonary inflammatory changes, and severe AKI underscores the complexity of severe leptospiral infection in climate-vulnerable endemic settings.

The contribution of this report lies in its integration of environmental exposure history, structured diagnostic assessment using Modified Faine's Criteria, and standardized AKI classification under KDIGO guidelines. By aligning epidemiological risk recognition with clinical progression patterns, this case reinforces the importance of early risk stratification and vigilant biochemical monitoring, even when initial laboratory parameters appear to improve.

Clinically, the findings emphasize that flood exposure combined with skin disruption should prompt early suspicion of leptospirosis in endemic regions. From a public health perspective, this case supports the need for strengthened integration between seasonal surveillance data and frontline clinical decision-making, particularly in regions experiencing increasing rainfall variability and flooding due to climate change.

Several limitations should be acknowledged, including the absence of molecular confirmation PCR or MAT titers, which may reflect resource constraints or institutional

limitations. As a single-case report, causal inference and generalizability are limited. Molecular confirmation with polymerase chain reaction (PCR) and paired microscopic agglutination testing (MAT) titers were not available, which may restrict microbiological characterization. Additionally, long-term renal outcomes after discharge were not assessed.

Future research should focus on prospective cohort studies evaluating early biomarkers predictive of progression to severe AKI in leptospirosis, and should also consider implications for hospital-level preparedness in flood-prone regions. Further investigation into host immune response profiles and endothelial injury markers may improve early severity stratification. In climate-endemic regions, implementation studies assessing the integration of environmental risk alerts into primary healthcare triage protocols may help bridge the gap between epidemiological awareness and early bedside recognition of severe disease.

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