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Acute Kidney Injury in 43 Years Old Male Patient Caused by Severe Leptospirosis

. **Ajeng Sulistianing Utami[1], Amanukarti Resi Oetomo[2*]**

[1] General Practicioner, Regional General Hospital of Mataram City, Indonesia.

[2] Nephrology and Hypertension Division of Internal Medicine Department, Regional General Hospital of Mataram City, Indonesia.

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Abstract: Leptospirosis is a zoonotic infectious disease commonly found in tropical regions, caused by *Leptospira interrogans*, a pathogenic spirochete. The main reservoir is rodents, especially rats. Human transmission occurs either directly through inoculation from infected animal tissues, body fluids and urine, or indirectly through contact via mucosal surfaces or abraded skin. The excretion of organisms through the urine of infected animals represents the most common source of Leptospira infection. As of mid-2024, 367 cases of leptospirosis with 42 deaths had been reported in Indonesia. However, the true incidence is likely underreported due to diagnostic challenges. In West Nusa Tenggara (NTB), no formal report exists; however, a data from Mataram City Hospital between 2013-2025 recorded three cases of leptospirosis involving renal impairment requiring hemodialysis (HD). The clinical course of leptospirosis is typically acute, following an incubation period of five to fourteen days. Most patients present with mild, anicteric febrile, but a smaller subset develop severe multiorgan involvement known as Weil's Disease. The severe form is characterized by acute high-grade fever, acute kidney injury, hepatic failure, pulmonary involvement, cardiovascular instability, neurological deficits, and hemorrhagic diathesis.

Keywords: Leptospirosis, Weil's Disease, Acute Kidney Injury, Hemodialysis

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Introduction

Leptospirosis is an endemic zoonotic disease caused by Gram-negative bacteria of the genus Leptospira, capable of infecting both humans and animals. It is one of the most common zoonotic disease in worldwide, particularly in archipelagic countries with high rainfall and frequent flooding, such as Indonesia (Ningsih et al., 2022). Humans may acquire the infection through exposure to the urine of infected animals, either via direct contact or through contact with contaminated soil or water (Wang et al., 2023). As of mid-2024, 367 leptospirosis cases with 42 deaths had been reported in Indonesia by Ministry of Health of the Republic of Indonesia; however, the actual number is likely higher due to underreporting, as the disease remains challenging to diagnose (Pusat Analisis Keparlemenan Badan Keahlian Setjen DPR RI, 2024). Leptospirosis is often left untreated or misdiagnosed for other conditions due to its overlapping symptoms with other acute febrile illness like malaria and dengue fever (Pinto et al., 2022). In West Nusa Tenggara (NTB), no formal report exists; however, a data from General Regional Hospital of Mataram City between 2013-2025 recorded three cases of leptospirosis involving renal impairment requiring hemodialysis (HD). The clinical manifestations of leptospirosis are broad and non-specific, ranging from mild, self-limiting symptoms to severe and potentially fatal presentations. Kidney is one of the most common organs—beside liver, that is affected by leptospirosis which can lead to acute kidney

Email: ajengtmtami@gmail.com *(*Corresponding Author)*

injury. Treatment strategies vary depending on disease severity and the extent of organ involvement in affected patients (Sukma et al., 2021). This article reported a case in 43 years old male patient with acute kidney injury caused by severe leptospirosis.

Case Presentation

A 43-year-old male presented to the emergency department with incoherent speech and excessive somnolence for one day. Four days prior to admission, he developed persistent high-grade fever associated with headache, nausea, vomiting, and abdominal pain. Additional symptoms included jaundice (figure 1), calf muscle tenderness, absence of defecation for two days, decreased urine output, and dark-colored urine. The patient denied any similar illness in himself or his family. He worked as a construction laborer in flood-prone areas since the onset of the rainy season. On physical examination, the patient appearance was severely ill with GCS E3V3M5 (somnolent), blood pressure 120/71 mmHg, pulse 116 bpm, respiratory rate 21/min, temperature 38.5°C, $SpO_2$98% with 3 L/min oxygen via nasal cannula. Conjunctival suffusion was found bilaterally. Chest auscultation revealed coarse wet rales bilaterally. Abdominal examination showed tenderness, especially in the right hypochondrium, with liver palpable two fingers below the costal margin. Calf tenderness was present. ECG was sinus rhythm. The laboratory test was done to the patient on the first day of admission, the result showed that hemoglobin 13.0 g/dL, thrombocytopenia (123,000/µL), leukocytosis (22,840/µL), elevated liver enzymes (AST 77.9 U/L, ALT 168.1 U/L), and renal dysfunction (urea 357.4 mg/dL, creatinine 5.62 mg/dL). Arterial blood gas analysis showed metabolic acidosis (pH 7.30, HCO_3^- 20 mEq/L, pCO_2 20.6 mmHg). The patient was scheduled to undergo urinalysis, blood culture, malaria blood smear, and IgM anti-Leptospira testing. Urinalysis revealed protein +1, bilirubin +1, blood +3, numerous erythrocytes, and presence of crystals and bacteria. Malarial smear was negative. Chest X-ray suggested bronchitis. Based on clinical presentation, laboratory, and radiologic findings, the patient was diagnosed with uremic encephalopathy with severe sepsis, acute kidney injury (AKI), acute hepatic injury, consistent with the clinical presentation of Weil's Disease. The patient was given IV NaCl 0.9% (1000 mL/24 hours), IV Ceftriaxone 1 g once daily, IV Pantoprazole 40 mg daily, renal diet via NGT (6 × 200 mL), and hemodialysis (HD). The blood culture test was done, it showed that it was negative for bacterial growth; however, serology showed positive for IgM anti-Leptospira antibodies. After the first HD session on May 1st, 2025, the patient showed no clinical improvement and continued to complain of dyspnea, restlessness, and general weakness. As the dyspnea worsened, oxygen therapy was initiated using a simple mask at 6 L/min, and the patient remained tachycardic. The patient was subsequently scheduled for a second HD session. However, following the second HD session on May 3rd, 2025, the patient's clinical condition remained poor, with no significant improvement. Due to the persistent lack of clinical progress, a third HD session was planned. After the third session on May 5th, 2025, the patient's condition still showed no improvement, his level of consciousness was still somnolent. Laboratory findings continued to demonstrate markedly elevated renal function parameters, and the patient developed hypotension. As a result, vasoconstrictor therapy with a norepinephrine infusion at 0.1 mcg/kg/min was administered for three days in the ICU. Laboratory evaluations continued to show elevated renal function markers, and the patient was scheduled for another HD session once blood pressure is stabilized. The patient developed a polyuric phase post third HD (urine output >5000 mL/24h), which was managed by fluid balance regulation. Following the fourth HD session (day 12), Pre-HD laboratory tests revealed mild anemia (Hb 9.0 g/dL) and persistently elevated renal function parameters (Urea 222.1 mg/dL; Creatinine 3.11 mg/dL). Following the HD session, the patient demonstrated some clinical improvement; however, the patient complained of fatigue, and physical examination revealed pale conjunctivae. Laboratory studies showed a significant decline in hemoglobin level (8.8 g/dL), leukocytosis, and partial improvement in renal function parameters (Urea 93.4 mg/dL; Creatinine 1.43 mg/dL). On the following day, the patient reported worsening fatigue and overall clinical deterioration. No signs of spontaneous or occult bleeding were identified. Blood tests revealed severe anemia (Hb 7.2 g/dL), reduced serum iron (31 µg/dL), decreased TIBC (190 µg/dL), elevated ferritin (990.9 µg/mL), and normal transferrin saturation (16.2%). The patient subsequently received two units of packed red blood cells, which increased the hemoglobin level to 9.0 g/dL, followed by an additional unit of transfusion. The patient's last recorded hemoglobin level before hospital discharge was 10.0 g/dL. The progression of the patient's laboratory findings is summarized in Table 1. One week after being discharged from the hospital, patient returned for follow-up at the nephrology outpatient clinic for a clinical and laboratory evaluation. The clinical condition had significantly improved and the laboratory results also showed improvement (Hb: 10.5 g/dL, PLT: 334.000/uL, WBC: 9.130/uL, Urea 32,2 mg/dL, Creatinine 0,72 mg/dL).

Figure 1. Patient's Clinical Appearance

Table 1. Serial Laboratory Findings

Parameters	01/05 Pre HD 1	05/05 Post HD 2	08/05 Post HD 3	09/05 Pre HD 4	09/05 Post HD 4	10/05	11/05	13/05
Hb (g/dL)	13.0	10.3	15.0	9.0	8.8	7.2	9.0	10.4
PLT (10^3/uL)	123	218	309	278	277	396	446	426
WBC (10^3/uL)	22.84	15.98	7.27	10.31	20.26	11.26	13.52	9.75
Urea (mg/dL)	357.4	337.0	232.8	222.1	93.4	-	60.7	30.4
Creatinine (mg/dL)	5.62	6.79	3.63	3.11	1.43	-	1.09	0.83
AST (U/L)	77.9	-	30.8	-	31.8	-	-	-
ALT (U/L)	168.1	-	35.6	-	28.4	-	-	-
Total Bilirubin (mg/dL)	9.02	-	-	-	5.09	2.42	-	-
Direct Bilirubin (mg/dL)	3.44	-	-	-	3.83	1.79	-	-
Indirect Bilirubin (mg/dL)	5.58	-	-	-	1.26	0.63	-	-
Blood Glucose (mg/dL)	114	105	-	-	-	-	-	-
Na (mmol/L)	135.0	140.0	-	-	-	-	-	134.0
K (mmol/L)	4.0	5.40	-	-	-	-	-	3.30
Cl (mmol/L)	103	110.0	-	-	-	-	-	99.0
HBsAg	Neg.							-
Anti-HCV	Neg.							-
IgM Anti-Leptospira			Positive					-

Discussion

Leptospirosis is a bacterial zoonosis acquired through contact with environments contaminated by the urine of infected animals. The pathogen colonizes the renal tubules of reservoir hosts and is shed into water and moist soil, facilitating transmission particularly in tropical settings. Predisposing factors include occupational exposure, inadequate sanitation, contact with rodents or livestock, and immersion in contaminated floodwater. In the present case, a recent history of flood exposure likely contributed to the development of infection (Ningsih et al., 2022; Wang et al., 2023). Most cases of leptospirosis manifest as mild, anicteric fever, but a minority progress to severe, multiorgan involvement termed Weil's Disease (NCDC, 2015). In this case, the patient showed the features of severe leptospirosis: acute fever, jaundice, myalgia, decreased consciousness, oliguria, leukocytosis, thrombocytopenia, severe uremic syndrome, hepatic jaundice, hepatomegaly, right hypochondrial tenderness, and gastrocnemius muscle pain. Renal involvement represents a major complication of severe leptospirosis, characterized by a >3 fold elevation of serum creatinine above normal—classified as stage 3 AKI—requiring renal replacement therapy (Sukma et al, 2022; Ministry of Health, Nutrition & Indigenous Medicinine of Sri Lanka, 2016). The diagnosis of AKI was based on clinical presentation and laboratory evidence. The uremic and septic encephalopathy were inferred from central nervous system disturbances secondary to azotemia and sepsis. The probable diagnosis of severe leptospirosis was supported by positive IgM anti-leptospira serology. During hospitalization, the patient also developed progressive anemia, with hemoglobin significantly decreasing from 15.0 g/dL to 7.2 g/dL, PRC transfusion was given. Anemia is a recognized feature of leptospirosis, likely due to erythropoiesis suppression and hemolysis during the acute phase (Gangula et al., 2019). The patient subsequently entered a polyuric recovery phase prior to discharge. Primary therapy included IV fluids, antibiotic Ceftriaxone, and hemodialysis. The drugs of choice for moderate to severe leptospirosis are Penisilin G IV, Ampicilin IV, Azitromicin dihidrat, Cefotaxim IV, and Ceftriaxone IV. Ceftriaxone was chosen for this patient, it is because its availability and its efficacy is comparable to intravenous penicillin G against *Leptospira interrogans*. In addition, the use of Ceftriaxone is more convenient for healthcare providers, as it is administered once to twice daily, unlike penicillin, which must be given every 6-8 hours. Its pharmacokinetic profile has excellent tissue penetration, including renal parenchyma, and relatively long half-lives (Cantwell et al., 2017; Ji et al., 2024; Sukma et al., 2021). By day 12, the patient showed clinical and laboratory improvement following four HD sessions. The prognosis for AKI secondary to severe leptospirosis is generally favorable when adequate supportive therapy—including fluids, antibiotics, and renal support—is initiated (Sukma et al., 2022).

Conclusion

Leptospirosis is a common zoonotic disease in tropical countries. Its clinical presentation ranges from mild, self-limiting illness to severe, life-threatening

multiorgan failure (Weil's Disease). In Indonesia—particularly in West Nusa Tenggara—underreporting remains an issue due to diagnostic limitations. Data from Regional General Hospital of Mataram City (2013–2025) revealed only three cases with renal involvement requiring hemodialysis. Early recognition and adequate supportive management can significantly improve the outcomes in patients with severe leptospirosis.

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