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Severe Leptospirosis (Weil's Disease) Complicated by Septic Shock: A Case Report

Melvin Andrean[1*], Calvin Sasongko[2], Nabil Mubtadi Falah[3]
[13]Rumah Sakit Umum Daerah Cengkareng, Jakarta, Indonesia
[2]Rumah Sakit Umum Daerah Leuwiliang, Bogor, Indonesia

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ABSTRACT

Introduction: *Weil's disease is the severe manifestation of leptospirosis, characterized by multiorgan dysfunction and high mortality risk without timely diagnosis and treatment. Early clinical suspicion and prompt intervention are critical for favorable outcomes.* ***Case Illustration****: a 51-year-old male with fever, left leg myalgia, jaundice, hypotension, and tachycardia consistent with septic shock. Laboratory evaluation revealed leukocytosis, acute liver injury, and severe acute kidney injury. Additionally, chest imaging revealed bilateral pulmonary infiltrates. Initial management included fluid resuscitation, vasopressors, and broad-spectrum antibiotics. Leptospira IgM serology confirmed the presence of leptospirosis on day 2. Despite severe acute kidney injury, renal function improved without dialysis, and vasopressors were discontinued by day 3. The patient was discharged on day 11, having made a full recovery.* ***Discussion:*** *Severe leptospirosis can present with nonspecific symptoms and progress to septic shock and multiorgan dysfunction. In this case, the diagnosis was supported by IgM-ELISA and a history of exposure to floodwater. Early treatment with broad-spectrum antibiotics and supportive care led to a full recovery.* ***Conclusion:*** *This case highlights the uncommon presentation of septic shock as the dominant feature of Weil's disease, together with rapid renal recovery without dialysis, underscoring the reversibility of multiorgan dysfunction when promptly managed.*

1. INTRODUCTION

Leptospirosis is a globally distributed zoonotic infection caused by pathogenic spirochetes of the genus *Leptospira*.[1,2] Humans are primarily infected through exposure to water or soil contaminated with the urine of infected animals, particularly in tropical and subtropical regions.[2,3] According to the World Health Organization (WHO), 920 cases of leptospirosis were reported in Indonesia in 2019, with 122 deaths.[4] However, the true burden is often underestimated, as recent estimates suggest an annual morbidity of 39.2 per 100,000 population.[4]

The clinical spectrum of leptospirosis can vary widely, ranging from a mild, nonspecific febrile illness observed in dengue fever, viral hepatitis, and malaria to a severe, life-threatening manifestation.[3,5] The severe form of leptospirosis is also known as Weil's Disease, classically defined by the triad of jaundice, hemorrhage, and acute kidney injury.[6,7] This wide variety of symptoms can lead to diagnostic confusion due to overlapping clinical features.[6] The overlap often delays appropriate management, thereby increasing the risk of severe complications, such as septic shock and multiorgan dysfunction, that further increase mortality rates.[5–8] The critical role of early recognition and treatment is paramount. Timely intervention significantly improves outcomes, especially for severe cases with renal, pulmonary, or neurological complications.[9,10]

This case report aims to present a rare and severe manifestation of Weil's disease, as well as to provide insights for physicians in Indonesia regarding the importance of early detection and timely treatment to prevent mortality and improve patient outcomes. In particular, we report the

*Corresponding author
E-mail addresses: melvinandrean@gmail.com

uncommon presentation of septic shock in severe leptospirosis, along with rapid recovery of renal function without dialysis, which has not been widely documented. It is hoped that this case can serve as a reminder that leptospirosis presents with diverse manifestations and varying degrees of severity, and can provide a significant contribution to the advancement of health and medical practice in Indonesia.

2. CASE ILLUSTRATION

A 51-year-old man arrived at the Emergency Department (ED) with a 2-day history of fever, left leg pain, and jaundice. The medical history was unremarkable for chronic illness, urinary/bowel issues, or high-risk behaviors such as IV drug use or promiscuity. Further questioning revealed recent exposure to floodwater, raising clinical suspicion of leptospirosis.

Upon arrival, vital signs revealed hypotension (73/44 mmHg), tachycardia (102 bpm) with a weak, regular pulse, a normal respiratory rate (20/min), normothermia (36.7°C), and an oxygen saturation of 98% on room air, consistent with early septic shock. Physical examination showed scleral icterus without hepatosplenomegaly, peripheral edema, or extremity jaundice. Cardiac and pulmonary auscultation was unremarkable.

Initial laboratory findings revealed leukocytosis ($28.6 \times 10^3/\mu L$), severe hyponatremia (121 mmol/L), and evidence of hepatic involvement (total bilirubin, 2.01 mg/dL; SGOT, 80 U/L; SGPT, 32 U/L). Marked renal impairment was observed with urea 264 mg/dL, creatinine 6.6 mg/dL, and estimated GFR of 9.5 mL/min/1.73 m^2 (Table 1), consistent with Stage 3 Acute Kidney Injury (AKI). Chest radiography showed bilateral pulmonary infiltrates (Figure 1), and abdominal ultrasonography revealed the absence of hepatic abnormality with a left kidney cyst (Figure 2). Based on these findings, the working diagnosis was septic shock secondary to suspected Weil's disease, complicated by severe hyponatremia and AKI.

Emergency department management included fluid resuscitation with crystalloids at 1000cc over 1 hour, norepinephrine infusion at 0.1 μg/kg/min to maintain mean arterial pressure ≥ 65 mmHg, systolic blood pressure ≥ 90 mmHg, and urine output ≥ 0.5 mL/kg/h, and empirical broad-spectrum antibiotics (cefoperazone 3g/day) for the initial treatment of septic shock. Hypertonic saline (3% NaCl, 500 mL over 24 hours) was administered for severe hyponatremia. The patient was admitted to the high care unit (HCU) for further monitoring and treatment.

By the second hospital day, the patient had shown clinical improvement, with hemodynamic targets and urine output achieved through fluid and vasopressor therapy. Leptospira IgM serology was positive, confirming the diagnosis of leptospirosis (Weil's disease). Despite severe AKI, renal function improved rapidly, and vasopressors were discontinued by day 3 as blood pressure stabilized. The patient was transferred to the general ward in stable condition. Over the following days, laboratory parameters progressively normalized, including leukocyte count, liver enzymes, and bilirubin. The patient was discharged on day 11 with a full clinical recovery, having avoided the need for dialysis.

Table 1.
Laboratory Test Result

Examination	Result						
	2/2	**5/2**	**6/2**	**7/2**	**8/2**	**9/2**	**10/2**
Hemoglobin (g/dL)	12.6	-	-	11.6	-	-	13.0
Hematocrit (%)	34	-	-	33	-	-	38
Leucocyte (/uL)	28.6	-	-	13.9	-	-	15.3
Thrombocyte (/uL)	175	-	-	541	-	-	379
Natrium (mmol/L)	121	129	-	-	-	-	-
Kalium (mmol/L)	3.8	3.2	-	-	-	-	-
Chloride (mmol/L)	88	107	-	-	-	-	-
ALT/SGPT (g/dL)	52	-	-	-	-	-	44
AST/SGOT (g/dL)	80	-	-	-	-	-	57
Urea (mg/dL)	264	194	127	68	67	64	73
Creatine (mg/dL)	6.6	3.5	2.3	1.4	1.3	1.2	1.2
eGFR (mL/min/1.73m2)	9.5	19.8	32.1	56.9	62.0	68.0	68.0
Total Bilirubin (mg/dL)	2.01	-	-	-	-	-	0.84
Conjugated Bilirubin (mg/dL)	1.56	-	-	-	-	-	0.52
Unconjugated Bilirubin (mg/dL)	0.45	-	-	-	-	-	0.32
Gamma GT (U/L)	-	105	-	-	-	-	-
Total Protein (g/dL)	-	5.4	-	-	-	-	-
Albumin (g/Dl)	-	2.6	-	-	-	-	-
HBsAg	-	Non-reactive	-	-	-	-	-
IgM Anti HAV	-	Negative	-	-	-	-	-
Anti HCV	-	Non-reactive	-	-	-	-	-
IgM Anti-Leptospira	-	Positive	-	-	-	-	-

g = grams; dL = deciliter; uL = microliter; mm = millimeter; U = unit; L = liter; mEq = milliequivalent, min = minutes

Figure 1.
Chest X-Ray shows bilateral infiltrates in lungs

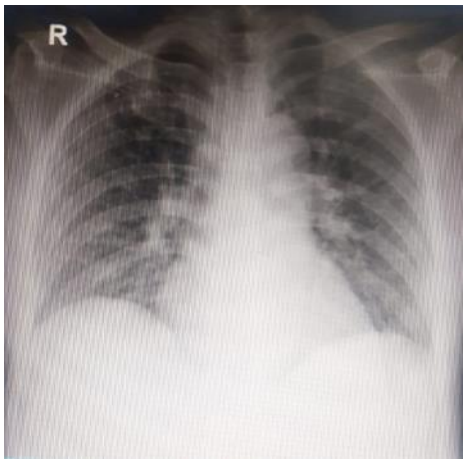
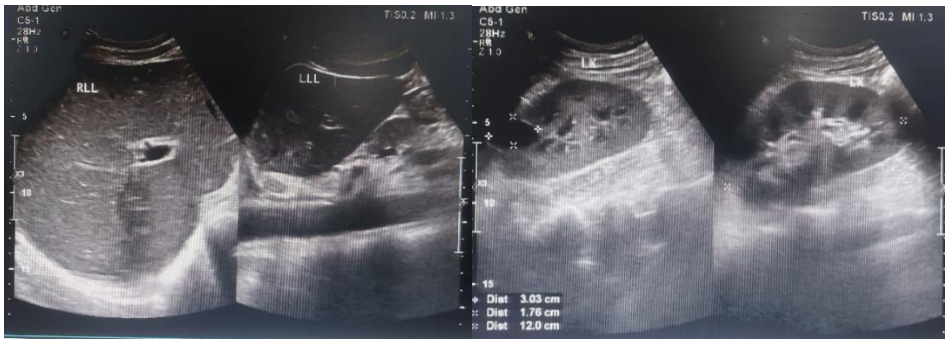


Figure 2.
Abdominal ultrasonography shows no abnormalities in liver and left kidney cyst



3. DISCUSSION

Diagnosis of Leptospirosis and Weil's Disease

Leptospirosis presents a diagnostic challenge due to its wide clinical spectrum and nonspecific early symptoms. In the initial phase, patients often exhibit flu-like symptoms, including fever, myalgia, and headache.[3,5] This initial presentation is sometimes indistinguishable from other infectious diseases, such as malaria, hepatitis, and dengue fever, all of which are common in tropical regions. The nonspecificity often leads to misdiagnosis or delays in diagnosis, particularly in areas with limited access to diagnostic testing.[3,5]

In this case, the patient presented with nonspecific symptoms of fever, leg myalgia, and jaundice. Although the presentation may overlap with other tropical infectious diseases, a key diagnostic clue was the history of floodwater exposure, a recognized risk factor for Leptospira transmission in endemic areas, such as Indonesia.[2,3]

While most patients present with mild symptoms, approximately 10% develop severe disease.[11] This severe form, known as Weil's Disease, is characterized by jaundice, acute kidney injury (AKI), and hemorrhagic manifestations.[6,7] The current patient met these criteria, with overt jaundice, significant renal dysfunction, and laboratory evidence of hepatic involvement. The diagnosis was confirmed via a positive Leptospira IgM serology using enzyme-linked immunosorbent assay (ELISA). While the Microscopic Agglutination Test (MAT) remains the gold standard, its limitations in acute settings make IgM-ELISA a more practical choice for early and definitive diagnosis.[12]

Septic Shock with Overlapping Hepatic and Renal Dysfunction

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.[13,14] According to the third international consensus on sepsis and septic shock (Sepsis-3), sepsis should be suspected in patients with signs of infection from any source.[13] A diagnosis is confirmed when the Sequential Organ Failure Assessment (SOFA) score is ≥ 2.[13] Septic shock is defined by the need for vasopressors to maintain a mean arterial pressure (MAP) ≥ 65 mmHg and a serum lactate level ≥ 2 mmol/L in the setting of sepsis.[13]

At initial presentation, the patient exhibited hemodynamic instability, characterized by hypotension, tachycardia, and a weak pulse, indicating shock. Given the clinical signs of infection and supporting laboratory results, sepsis was suspected. The patient's SOFA score of 11 confirmed the diagnosis, further supported by the presence of shock and the requirement for vasopressors.

Septic shock in severe leptospirosis, a relatively rare manifestation of Weil's disease, was a notable observation in this case. The patient's age (51) is consistent with the typical 40–70-year range for such presentations. This shock results from severe Leptospira infection, which triggers vasculitis and a systemic inflammatory response. Acute surges of cytokines and chemokines further contribute to the development of sepsis by dysregulating immune responses.[12]

This patient presented with jaundice and severe acute kidney injury (AKI), a combination that may resemble hepatorenal syndrome (HRS) in clinical practice. Leptospirosis-induced liver dysfunction, causing jaundice, may result from ischemic hepatic necrosis and architectural

disruption. Renal injury in leptospirosis can range from prerenal azotemia to severe AKI requiring dialysis. Inflammatory responses in the renal proximal tubules are mediated by activation of Toll-like receptor 2, nuclear factor-κB, and mitogen-activated protein kinase.[5]

HRS is typically diagnosed in patients with cirrhosis or acute liver disease with portal hypertension and a marked decline in glomerular filtration rate (GFR) in the absence of intrinsic renal disease.[15] In this patient, there were no clinical, laboratory, or radiological signs of cirrhosis. Therefore, a definitive diagnosis of HRS could not be established. Differentiating it from true HRS is essential due to differences in management. True HRS requires specific therapies, including intravenous albumin and treatment of the underlying cirrhosis.[16] Whereas AKI, because of leptospirosis, is primarily driven by infection-induced inflammation and is best managed with early antibiotics, fluids, and vasopressors.[12]

Similar overlaps of hepatic and renal dysfunction in leptospirosis have been described in previous reports. For example, Johri and Maheshwari[5] reported a case of pseudo-hepatorenal syndrome in leptospirosis, highlighting how combined hepatic and renal dysfunction can mimic HRS. Similarly, Dursun et al.[17] described fulminant leptospirosis complicated by severe pulmonary hemorrhage and hepatorenal failure, illustrating the poor outcomes often associated with such presentations. Earlier, Madle-Samardzija et al.[18] documented two cases of leptospirosis presenting with hepatorenal syndrome, further supporting that this overlap, although uncommon, has long been recognized in the literature.

Treatment and Outcome of Severe Cases

Timely diagnosis is critical for initiating appropriate interventions in multiorgan failure. Bauer et al. found an average 30-day septic shock mortality of 34.7% (38.5% at 90 days). Reducing this mortality hinges on effective fluid resuscitation (guided by responsiveness and tolerance), early vasopressor use, and suitable antibiotic therapy.[13,19]

Leptospirosis treatment involves antibiotics and supportive therapy. Severe cases may require intravenous penicillin G, third-generation cephalosporins, or erythromycin. In this instance, cefoperazone, a third-generation cephalosporin, was selected for its broad-spectrum coverage of Gram-negative bacteria and favorable pharmacokinetics. It is primarily biliary excreted, requiring no dosage adjustment in renal dysfunction.[20,21]

This case also notably featured a rapid improvement in renal function. A Brazilian study found that daily dialysis benefited leptospirosis patients with acute respiratory distress syndrome and AKI by reducing mortality.[19] However, the STARRT-AKI (Standard versus Accelerated Initiation of Renal-Replacement Therapy in Acute Kidney Injury) trial found that initiating renal replacement therapy within 12 hours of AKI diagnosis did not reduce mortality compared to standard timing.[22]

In the current case, the uncommon presentation of septic shock as the dominant feature of severe leptospirosis, together with rapid renal recovery without the need for dialysis, makes this report noteworthy. To our knowledge, such a combination has been rarely documented in the literature. This fact underscores the potential reversibility of multiorgan dysfunction in leptospirosis when managed promptly, highlighting that early recognition and intervention remain key factors in improving patient outcomes.

4. CONCLUSION

In conclusion, early recognition and treatment are crucial in managing severe leptospirosis with septic shock. This case highlights the uncommon presentation of septic shock as the dominant feature of Weil's disease, together with rapid renal recovery without the need for dialysis. Prompt antimicrobial therapy and supportive care can lead to full recovery, emphasizing the importance for clinicians in endemic regions to remain vigilant and intervene early to improve patient outcomes.

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