Case Report

Leptospiral pulmonary hemorrhagic syndrome as a severe complication of Weil's disease: A case report

Welly Salutondok[1], Karlos Berlusconi Sihaloho[2], Tiroy Sari Simanjuntak[1], Hildebrand Hanoch Victor[1], Donnie Lumban Gaol[1], Kurniyanto[1], Achnes Pangaribuan[1], Daniel Reinaldo Parsaoran Situmorang[1]

From [1]Internist, Department of Internal Medicine, Faculty of Medicine, [2]General Hospital, Department of General Hospital, Universitas Kristen Indonesia, Jakarta, Indonesia

ABSTRACT

Leptospirosis is a globally distributed zoonotic disease caused by *Leptospira* spirochetes, often leading to severe complications, such as Weil's disease and leptospiral pulmonary hemorrhagic syndrome (LPHS). LPHS is particularly lethal, with mortality rates ranging from 40% to 60% due to acute respiratory failure. A 41-year-old male presented with a 4-day history of high fever, severe myalgia (notably in the calves), headache, and nausea following recent exposure to floodwaters and rodents. Physical examination revealed jaundice, tachycardia, tachypnea, and gastrocnemius tenderness. Initial laboratory findings showed leukocytosis, severe thrombocytopenia (34,000/µL), and significant hepatorenal dysfunction (creatinine 6.7 mg/dL, elevated liver enzymes). Despite aggressive therapy, including broad-spectrum antibiotics, hemodialysis, and blood product transfusions, the patient's condition deteriorated. He developed bilateral subconjunctival hemorrhage and pulmonary edema. Diagnostic confirmation was achieved through a positive Leptospira IgM and a microscopic agglutination test titer of 1:640. The patient ultimately succumbed to septic shock and multi -organ failure on the 11th day of hospitalization. This case underscores the rapid and often fatal progression of LPHS in the context of Weil's disease. Early clinical suspicion based on epidemiological factors (e.g., flood exposure) and prompt initiation of intensive supportive care are critical, although the prognosis remains poor once severe pulmonary hemorrhage and multi -organ dysfunction develop.

Key words: Leptospiral pulmonary hemorrhagic syndrome, Leptospirosis, Weil's disease

Leptospirosis is a zoonotic disease caused by infection with spirochete bacteria of the genus Leptospira. The disease has a wide global distribution, with higher incidence rates in tropical and subtropical regions due to environmental conditions that support the survival and transmission of the bacteria. Overall, leptospirosis is one of the most common zoonoses in the world, with a mortality rate comparable to or even higher than that of hemorrhagic fevers of other etiologies. It is estimated that approximately 1.03 million cases and 58,900 deaths occur globally each year. Open wounds, such as cuts and abrasions, as well as mucous membranes, for example, the conjunctiva, oral cavity, or vagina, can serve as entry points for infection. Indirect exposure through Leptospira-contaminated media is far more common, typically associated with recreational or occupational activities. In addition to risks associated with outdoor work, professions, such as sewer workers, military personnel, and those in the agricultural sector in tropical regions with high rainfall also face high risks, with the agricultural sector being the largest contributor in terms of case numbers. The most severe manifestation is pulmonary hemorrhagic leptospirosis (SPHL), which is the leading cause of death. Patients in this condition often require mechanical ventilation, yet the prognosis remains poor despite intensive supportive therapy and antibiotics. In cases involving pulmonary hemorrhage, approximately 40–60% of patients may die rapidly due to severe acute respiratory failure [1-4].

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CASE REPORT

A 41-year-old man presented to the Emergency Department of UKI General Hospital with a 4-day history of intermittent high-grade fever. The fever temporarily subsided after antipyretic administration

Correspondence to: Welly Salutondok, Department of Internal Medicine, Faculty of Medicine, Universitas Kristen Indonesia, Jakarta, Indonesia. E-mail: welly.salutondok@uki.ac.id

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but subsequently recurred. The patient also experienced generalized weakness, particularly in both legs, which had persisted for 3 days, as well as continuous bilateral calf pain. He complained of a severe frontal headache associated with nausea and vomiting. His appetite was markedly reduced, and he was unable to finish half of a usual meal. Approximately 2 weeks before admission, the patient's home had been flooded. During the flood, he did not wear appropriate protective footwear and was unable to maintain adequate hygiene because the floodwater remained in the house for approximately 12 h. After the flood receded, numerous rats entered his home. This history provided a relevant epidemiological exposure to potentially contaminated water and an environment contaminated by rodents.

On admission, the patient appeared moderately ill and was alert and oriented. His blood pressure was 118/67 mmHg, pulse rate was 119 beats/min, respiratory rate was 30 breaths/min, temperature was 39°C, and oxygen saturation was 100% on room air. Physical examination revealed bilateral gastrocnemius tenderness without peripheral edema.

Initial laboratory examination demonstrated hemoglobin of 10.3 g/dL, leukocytosis of 20,400/µL, severe thrombocytopenia of 34,000/µL, hematocrit of 28%, lymphocytopenia, and neutrophilia. Liver enzymes were elevated, with alanine transaminase of 290 U/L and aspartate transaminase of 129 U/L. Renal function was markedly impaired, with urea of 253 mg/dL and creatinine of 6.7 mg/dL. Serum sodium was 139 mmol/L, potassium was 2.7 mmol/L, and chloride was 102 mmol/L. The initial chest radiograph demonstrated a fine infiltrate in the right lung apex.

At this stage, the patient was initially suspected of having expanded dengue syndrome with bacterial coinfection, acute kidney injury, and hypokalemia. Fluid therapy, moxifloxacin, paracetamol, and ondansetron were administered. On the 2nd day of hospitalization, bilateral scleral icterus developed, accompanied by a marked reduction in urine output to 115 mL over 15 h. The patient remained febrile. Hemoglobin decreased to 8.6 g/dL, while leukocytosis and thrombocytopenia persisted. Total bilirubin was 13.8 mg/dL, direct bilirubin 12.6 mg/dL, and indirect bilirubin 1.2 mg/dL. Serum albumin was 2.3 g/dL. Hepatitis B surface antigen and anti- hepatitis C virus testing were non-reactive. Arterial blood gas analysis showed compensated metabolic acidosis.

On the 3rd day of hospitalization, the patient's condition deteriorated. He developed anemia, jaundice, hypotension, and coarse bilateral wet rales (Fig. 1a). Urea increased to 271 mg/dL and creatinine to 7.5 mg/dL, while sodium decreased to 122 mmol/L. Leptospira IgM was positive. Chest radiography demonstrated cardiomegaly and pulmonary edema (Fig. 1b). Because of worsening renal dysfunction, oliguria, and fluid overload, hemodialysis was initiated. Laboratory tests showed leukocytosis, elevated urea and creatinine levels (271 mg/dL and 7.5 mg/dL), hyponatremia (122 mg/dL), compensated metabolic acidosis, and positive Leptospira IgM.

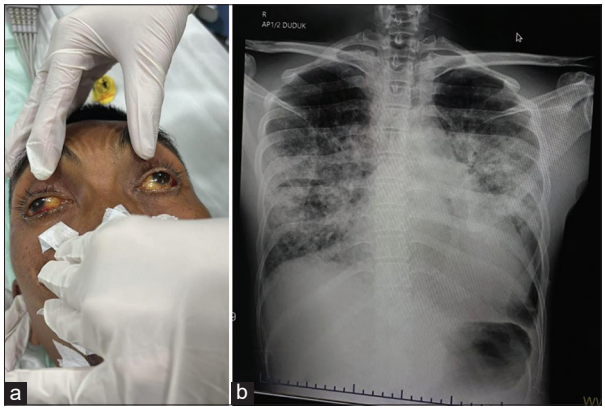


Figure 1: (a) Patient showing subconjunctival hemorrhage; (b) Chest X-ray showing cardiomegaly and pulmonary edema

On the 4th day, bilateral subconjunctival hemorrhage was documented. The patient remained icteric and had bilateral wet rales. Coagulation studies demonstrated prolonged prothrombin time and activated partial thromboplastin time. Dengue IgM was negative, and dengue IgG was positive. Following hemodialysis, hemoglobin was 9.9 g/dL, leukocyte count remained elevated at 31,600/µL, thrombocytopenia persisted, and urea and creatinine decreased to 202 mg/dL and 4.1 mg/dL, respectively. Peripheral blood examination showed hypochromic microcytic erythrocytes, leukocytosis, and thrombocytopenia. The patient received fresh frozen plasma, tranexamic acid, vitamin K, methylprednisolone, norepinephrine, and doxycycline. On the 5th day, chest radiography demonstrated bilateral paracardial infiltrates and a left-sided pleural effusion. On the 6th day, the patient developed rapid and deep breathing, leg edema, and supraventricular tachycardia with a heart rate of 168 beats/min. Electrical cardioversion at 100 kJ was performed, resulting in conversion to sinus rhythm.

On the 7th day, sputum culture and susceptibility testing identified *Klebsiella pneumoniae*. The organism demonstrated resistance to several antimicrobial classes, including penicillins, cephalosporins, carbapenems, aminoglycosides, quinolones, and tigecycline, with the susceptibility pattern documented in Table 1. The patient's eventual septic shock, cardiovascular instability, coagulopathy, acute kidney injury, hepatic dysfunction, and pulmonary abnormalities indicate severe multiorgan disease. Despite intensive supportive treatment, including vasopressors, oxygen therapy, blood products, antimicrobial therapy, and hemodialysis, the patient died on hospital day 11.

DISCUSSION

This report describes a case of leptospiral pulmonary hemorrhagic syndrome (LPHS). LPHS, the most severe complication of leptospirosis, is characterized by endothelial damage resulting from toxin effects

Table 1: Culture and antibiotic susceptibility testing for Tn.A

Group	Antibiotics	Result
Penicillins	Piperacillin/Tazobactam	R
	Amoxicillin	R
	Ampicillin	R
	Ampicillin/Sulbactam	R
Cephalosporin	Ceftriaxone	R
	Cefepime	R
	Cefazolin (urine)	R
	Cefazolin (other)	R
	Ceftazidime	R
Carbapenem	Meropenem	R
	Ertapenem	R
Monobactam	Aztreonam	R
Aminoglycosides	Amikacin	R
	Gentamicin	R
Quinolone	Ciprofloxacin	R
Antiseptik Urine	Nitrofurantoin	-
Antibiotik Lain	Tigecycline	I
	Trimethoprim/Sulfametho Xazole	R

Specimen: Sputum; Microorganism: *Klebsiella pneumoniae*

and a cytokine storm, which triggers diffuse alveolar hemorrhage and refractory hypoxemia, with a mortality rate that can exceed 50% [1,2,5,6]. Leptospirosis is a zoonotic disease commonly found in tropical regions and represents a significant public health problem, with an estimated annual morbidity rate in Indonesia of approximately 39.2 cases per 100,000 people. The disease is also known by various names, such as mud fever, swamp fever, mucous fever, autumn fever, infectious jaundice, field fever, and sugarcane fever. It is caused by spirochete bacteria of the genus Leptospira [4,7].

Transmission to humans can occur through direct contact with bodily fluids from infected animals (such as blood or urine), or indirectly through a contaminated environment, particularly standing water, rivers, lakes, ditches, or soil contaminated with animal urine. These bacteria can enter the body through intact mucous membranes and then spread through the bloodstream, causing systemic vasculitis in both large and small blood vessels and involving various organs [1,8-10]. For example, exposure can occur when a person sustains an injury while working, such as while cleaning a ditch, and the injured body part comes into contact with contaminated water. Most cases of leptospirosis are mild and resolve spontaneously without specific treatment. However, in some cases, multi-organ involvement occurs, increasing the risk of death.

One classic clinical form is Weil's disease, characterized by a combination of jaundice and kidney failure, first described in 1886. Initial symptoms typically appear suddenly as fever, chills, myalgia, and headache, with characteristic muscle pain in the calves and lower back. In severe cases, multiorgan dysfunction is frequently observed. In addition, in recent years, a rarer but extremely severe form has emerged: SPHL, characterized by massive pulmonary hemorrhage and often clinically unexpected [1,11-14]. The course of leptospirosis involves two main clinical phases: The septicemic phase and the immune phase. In the early phase, individuals exposed to the bacteria typically experience flu-like symptoms within 7–14 days of exposure. At this stage, conjunctival hyperemia may appear, a relatively characteristic sign rarely found in other tropical febrile infections, such as dengue fever, typhus, or malaria. In addition, fever often accompanies this phase [5,6,15,16].

About 1 month after exposure, the disease may enter its second phase, the immune phase. At this stage, the immune system responds to the infection by regulating cytokine release. In mild cases, this response is well-controlled, allowing patients to recover without residual symptoms. However, in severe cases, the immune response can become excessive and trigger a cytokine storm, leading to multi-organ damage, including the liver, kidneys, and heart. This occurs because large amounts of cytokines are released simultaneously. Laboratory results may show elevated bilirubin levels, elevated liver enzymes, impaired kidney function, and thrombocytopenia.

This case illustrates severe leptospirosis complicated by acute kidney injury, hepatic dysfunction, thrombocytopenia, coagulopathy, pulmonary involvement, cardiovascular instability, and septic shock. The epidemiological history was particularly relevant. The patient had been exposed to floodwater without adequate protective footwear approximately 2 weeks before symptom onset, and numerous rats subsequently entered his home. Flooding can facilitate exposure to water and soil contaminated with urine from infected animals. The clinical syndrome that followed abrupt fever, severe calf myalgia, headache, thrombocytopenia, jaundice, renal dysfunction, and hepatic abnormalities – is characteristic of severe leptospirosis. World Health Organization (WHO) guidance recognizes flooding and exposure to contaminated environments as important epidemiological contexts for leptospirosis.

The renal involvement was severe. At presentation, the patient's urea was 253 mg/dL, and creatinine was 6.7 mg/dL, increasing to 271 mg/dL and 7.5 mg/dL, respectively, by the 3rd hospital day. Oliguria and fluid overload subsequently developed, necessitating hemodialysis. Acute kidney injury is a recognized major complication of severe leptospirosis and may occur as part of the broader multiorgan dysfunction associated with severe disease. Hepatic involvement was also prominent. The patient developed jaundice with marked direct hyperbilirubinemia, elevated transaminases, and hypoalbuminemia. The combination of jaundice and renal failure is characteristic of severe icterohemorrhagic leptospirosis, historically referred to as Weil's disease. In this patient, the simultaneous occurrence of renal dysfunction, jaundice, thrombocytopenia, and coagulopathy was consistent with severe systemic disease. Pulmonary involvement was an important

component of the clinical deterioration. The initial chest radiograph showed a fine infiltrate in the right lung apex. This was followed by bilateral wet rales and radiographic pulmonary edema on the 3rd day, followed by bilateral paracardial infiltrates and a left pleural effusion on the 5th day.

These findings demonstrate progression from a relatively focal radiographic abnormality to more extensive pulmonary involvement. However, pulmonary edema and bilateral infiltrates are not synonymous with pulmonary hemorrhage. Pulmonary edema may occur in the context of renal failure and fluid overload, while pulmonary infiltrates may have multiple causes, including infection and inflammatory lung injury. Consequently, the available evidence supports pulmonary involvement in severe leptospirosis, but does not conclusively establish pulmonary hemorrhage.

This distinction is important because subconjunctival hemorrhage should not be considered equivalent to pulmonary hemorrhage. The former is an ocular hemorrhagic manifestation, whereas the latter requires evidence of bleeding into the pulmonary alveolar space. The patient's subconjunctival hemorrhage therefore supports the presence of a hemorrhagic tendency but does not independently establish pulmonary hemorrhage. The pulmonary findings were also potentially influenced by concomitant bacterial infection. Sputum culture on the seventh day isolated *K. pneumoniae*. This finding supports the presence of a concomitant bacterial respiratory infection, particularly in the setting of pulmonary infiltrates and progressive respiratory abnormalities. However, sputum isolation alone does not establish whether *K. pneumoniae* was the primary cause of the pulmonary radiographic abnormalities or whether it represented superimposed infection in a patient whose pulmonary condition was already affected by severe leptospirosis and fluid overload.

The WHO-recommended Faine criteria for diagnosing leptospirosis consist of three main components: Clinical data (Section A), epidemiological factors (Section B), and laboratory findings (Section C). Over time, these criteria have shown improved diagnostic sensitivity [1,8,9,16,17] (Table 2). The Faine score in the original manuscript also required correction. The modified Faine criteria allocate points across clinical, epidemiological, and laboratory domains. The original table produced a score of 56, but this appears to have resulted partly from counting both positive IgM and microscopic agglutination test (MAT) results separately. The modified criteria specify that only one positive serological test should be counted. In addition, the patient's clinical findings should be distinguished from findings that were not documented. For example, bilateral subconjunctival hemorrhage should not automatically be recorded as conjunctival suffusion, because these represent different clinical findings. Using the documented clinical information, the revised score is 34. The score exceeds the diagnostic threshold for presumptive leptospirosis when Parts A+B+C are combined. The positive Leptospira IgM and subsequent MAT titer of 1:640 further support the diagnosis. The MAT result was obtained on day 10 of hospitalization, and because only a single titer is reported, it should not be described as seroconversion or a fourfold rise unless paired sera demonstrating such a change are available.

Table 2: Faine patient score

Component/Criterion	Score	Patient Score	Patient finding
Part A: Clinical data			
Headche	2	2	Present
Fever	2	2	Present
Fever >39°C	2	2	39°C
Conjungtival suffusion	4	-	Not documented
Meningismus	4	-	Not documented
Myaliga	4	4	Bilateral calf pain
Conjungtival suffusion+meningismus+myalgia	10	-	Criteria not fulfilled
Jaundice	1	1	Developed during hospitalization
Albuminuria/Nitrogen Retention	2	-	Markedly elevated urea/creatinine
Hemoptisis/dispnea	2	2	Dyspnea/pulmonary involvement documented
Part B: Epidemiological factors			
Rainfall	5	5	Not specifically documented
Contact with contaminated environment	4	4	Recent flood-water exposure
Animal Contact	1	-	Rats entered the home; direct contact not documented
Part C: Bacteriological and laboratory findings			
Leptospira culture	Diagnostic	0	Not performed/documented
PCR	25	0	Not documented
ELISA IgM positive	15	15	Positive
MAT single high titer	15	15	1:640
Total Score		34	

PCR: Polymerase chain reaction, Enzyme-linked immunosorbent assay, MAT: Microscopic agglutination test

CONCLUSION

A 41-year-old man has been reported to have LPHS as a complication of Weil's disease. Leptospirosis in this case was diagnosed using the gold standard diagnostic test, the MAT, supplemented by the modified Faine criteria, which indicated a strong likelihood of infection. Although rare, leptospirosis can progress to pulmonary hemorrhagic syndrome, which has a high mortality rate. If there is a history of exposure supported by appropriate clinical and laboratory findings, antibiotic therapy should be initiated immediately. This is particularly important once respiratory symptoms begin to appear.

REFERENCES

1. Dong WH, Chen Z. Leptospirosis with pulmonary haemorrhage and multiple organ failure: A case report and literature review. J Int Med Res 2021;49:1-3.
2. Yang LW, Tang XG, Xiong B, Zhang Y. A case report of leptospirosis complicated by severe pulmonary hemorrhage treated with venovenous extracorporeal membrane oxygenation. Front Med (Lausanne) 2025;12:1696005.
3. Vaishnavi K, Geetha NK, Mariraj I, Balaganapathi P, Amin FM, Shankar AG. An unusual case of hemoptysis: Pulmonary hemorrhage in leptospirosis. Ro J Infect Dis 2024;27:214.
4. Hermawati BD, Hapsari BD, Wulandari EL, Prabowo NA, Sukmagautama C, Putri DP, *et al*. Weil's disease with multiple organ dysfunction, community-acquired pneumonia and septic shock: The role of rapid diagnosis and management. Narra J 2024;4:e587.
5. Galdino GS, Bezerra Da Silva Junior G, De Francesco Daher E. Leptospirosis and Weil's syndrome. In: Bezerra Da Silva Junior G, De Francesco Daher E, Barros E, editors. Tropical Nephrology. Cham: Springer International Publishing; 2020. p. 263-76.
6. Petakh P, Behzadi P, Oksenych V, Kamyshnyi O. Current treatment options for leptospirosis: A mini-review. Front Microbiol 2024;15:1403765.
7. Croda J, Neto AN, Brasil RA, Pagliari C, Nicodemo AC, Duarte MI. Leptospirosis pulmonary haemorrhage syndrome is associated with linear deposition of immunoglobulin and complement on the alveolar surface. Clin Microbiol Infect 2010;16:593-9.
8. Loulas A, Tran T, Meurant V. Severe leptospirosis (Weil's disease) presenting with fulminant alveolar hemorrhage and Multiorgan failure: A case report. Cureus 2026;18:e113916.
9. Brett-Major DM, Coldren R. Antibiotics for leptospirosis. Cochrane Database Syst Rev 2012;12:CD008264.
10. Petakh P, Oksenych V, Kamyshnyi O. Corticosteroid treatment for leptospirosis: A systematic review and meta-analysis. J Clin Med 2024;13:4310.
11. Sarkar U, Nascimento SF, Barbosa R, Martins R, Nuevo H, Kalofonos I, *et al*. Population-based case-control investigation of risk factors for leptospirosis during an urban epidemic. Am J Trop Med Hyg 2022;66:605-10.
12. Davenport C, Stawkowski LJ, Fernandez Felix D, Weaner S, Strow J. Leptospira(ls) to pulmonary hemorrhage. Am J Respir Crit Care Med 2025;211:A7836.
13. Tyas BD, Lestari P, Akbar MI. Maternal perinatal outcomes related to advanced maternal age in preeclampsia pregnant women. J Family Reprod Health 2019;13:191-200.
14. Adler B, De La Peña Moctezuma A. Leptospira and leptospirosis. Vet Microbiol 2010;140:287-96.
15. Escobedo-López H, Sánchez-López G, Cruz-Sánchez S, Gutiérrez-Campos ND, Gómez-Zamora CA, Terrones-Lozano A. Fulminant diffuse alveolar hemorrhage in Weil's disease: A case report. J Case Rep Image Infect Dis 2026;4:15-7.
16. Bandara K, Weerasekera MM, Gunasekara C, Ranasinghe N, Marasinghe C, Fernando N. Utility of modified Faine's criteria in diagnosis of leptospirosis. BMC Infect Dis 2016;16:446.
17. Saputra RC, Mahmuda IN, Utami MB, Pinakesty A, El Mubaraq S, Srirojanakul S. Correlation between probable or non-probable leptospirosis with laboratory findings: Based on leptospirosis case definition and faine criteria. Indones J Trop Infect Dis 2025;13:123.

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