Case Report

Severe leptospirosis (Weil's disease) presenting with pulmonary hemorrhage syndrome and acute kidney injury

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ABSTRACT

Leptospirosis is a globally prevalent zoonotic infection with a wide clinical spectrum. Weil's disease represents a severe form of leptospirosis characterized by jaundice, acute kidney injury (AKI), and hemorrhagic manifestations. Pulmonary hemorrhage is an uncommon but potentially fatal complication. We report the case of a 62-year-old man who presented with acute febrile illness, hemoptysis, jaundice, and AKI. Investigations revealed thrombocytopenia, leukocytosis, severe azotemia, and positive Leptospira immunoglobulin M serology, while dengue and malaria were excluded. He was diagnosed with severe leptospirosis complicated by pulmonary hemorrhage syndrome. The patient was managed with doxycycline, broad-spectrum antibiotics, and supportive care, resulting in complete clinical recovery with normalization of renal function. This case highlights the importance of early recognition of pulmonary involvement in leptospirosis, as timely treatment and supportive care may improve outcomes.

Key words: Acute kidney injury, Leptospirosis, Pulmonary hemorrhage, Weil's disease, Zoonosis

Leptospirosis is an emerging zoonotic disease of global importance, particularly in tropical and subtropical regions [1]. Human infection occurs following exposure to water or soil contaminated with the urine of infected reservoir hosts, particularly rodents [2]. The clinical spectrum ranges from a mild, self-limiting, influenza-like illness to fulminant disease known as Weil's disease [3].

Weil's disease is classically characterized by jaundice, acute kidney injury (AKI), and hemorrhagic manifestations [4]. Although hepatic and renal involvement are well recognized, pulmonary involvement, ranging from mild respiratory symptoms to pulmonary hemorrhage, is an important cause of morbidity and mortality.

This case describes severe leptospirosis presenting with hemoptysis and features suggestive of pulmonary hemorrhage syndrome, highlighting the diagnostic challenge and favorable outcome following appropriate treatment and supportive care.

CASE PRESENTATION

A 62-year-old man presented to the emergency department with a 4-day history of high-grade continuous fever associated with chills and rigors. Two days before admission, he developed shortness of breath and a cough productive of dark-brown blood (hemoptysis). He also reported episodes of dark-colored vomiting, nausea, generalized body aches, and passage of dark-yellow urine. The patient had a history of hypertension treated with chlorthalidone (6.25 mg). There was no history of diabetes mellitus, thyroid disease, or previous tuberculosis. He denied recent travel to malaria-endemic areas.

On admission, the patient was conscious but appeared distressed. His blood pressure was 100/70 mmHg, pulse rate was 98–100 beats/min, and oxygen saturation was 95% on room air. Although he was afebrile at the time of examination (98.6°F), he reported high-grade fever before admission. Icterus and pallor were present. There was no lymphadenopathy or pedal edema. Respiratory examination revealed bilateral basal crackles. Cardiovascular examination revealed normal S1 and S2 without murmurs. Abdominal examination was soft, with mild hepatomegaly.

Initial laboratory investigations demonstrated features of multiorgan dysfunction. Hemoglobin was 11.0 g/dL, the total leukocyte count was 11,200 cells/mm^3, and the platelet count was 80,000 cells/mm^3. Severe AKI was evident, with blood urea nitrogen of 118.3 mg/dL and serum creatinine of 3.90 mg/dL. Liver function tests showed hyperbilirubinemia, with total bilirubin initially 1.8 mg/dL and a peak value of 8.57 mg/dL. Aspartate

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aminotransferase and alanine aminotransferase were 157.4 U/L and 116.6 U/L, respectively. Leptospira immunoglobulin M (IgM) serology was positive (47.5 units; reference value <9 units). Dengue NS1 antigen and IgM were non-reactive, and malaria antigen testing was negative. Echocardiography demonstrated normal left ventricular systolic function (68%) with grade I diastolic dysfunction, making primary cardiogenic pulmonary edema less likely. The chest radiograph was reported as normal. Sputum Gram staining showed Gram-positive cocci, but culture showed no growth. Sputum for acid-fast bacilli staining on 3 consecutive days and CBNAAT were negative.

A diagnosis of severe leptospirosis (Weil's disease) was made on the basis of the clinical presentation, including jaundice and AKI with hemorrhagic manifestations, together with positive Leptospira IgM serology and exclusion of other common tropical infections. The hemoptysis and bilateral basal crackles were considered suggestive of leptospirosis-associated pulmonary hemorrhage syndrome.

The patient was admitted to the intensive care unit. Empirical broad-spectrum antimicrobial therapy with piperacillin-tazobactam and meropenem was initiated to provide coverage for possible secondary bacterial sepsis, along with doxycycline for suspected leptospirosis. Intravenous fluids were administered cautiously to manage AKI while minimizing the risk of fluid overload in the setting of pulmonary involvement. Vitamin K was administered for suspected coagulopathy, and ursodeoxycholic acid was given for cholestatic liver dysfunction. Nebulization and supplemental oxygen were provided as required.

The patient responded favorably to treatment. Fever subsided within 48 h of initiation of antimicrobial therapy. Renal function improved, with serum creatinine decreasing from 3.90 mg/dL to 0.85 mg/dL during hospitalization. Bilirubin levels subsequently stabilized. Hemoptysis ceased, and oxygen saturation remained stable on room air. The patient was discharged in stable condition with advice for outpatient follow-up.

DISCUSSION

Leptospirosis is a systemic infection caused by pathogenic Leptospira species and can produce a wide spectrum of clinical manifestations. Severe disease may involve the kidneys, liver, lungs, and coagulation system. The pathogenesis of pulmonary involvement is thought to involve endothelial and alveolar-capillary injury, although the precise mechanisms remain incompletely understood.

The diagnostic presentation in this case was challenging because the patient had fever, thrombocytopenia, hemoptysis, and dark-colored vomiting, features that can mimic severe dengue or malaria in tropical settings [5]. However, leukocytosis, marked renal dysfunction, jaundice, and positive Leptospira IgM serology supported the diagnosis of severe leptospirosis.

Pulmonary involvement in leptospirosis can range from cough and dyspnea to life-threatening pulmonary hemorrhage [6]. In this patient, hemoptysis and bilateral basal crackles were clinically suggestive of pulmonary hemorrhage syndrome. However, the normal chest radiograph and absence of documented pulmonary infiltrates or computed tomography findings should be acknowledged when describing the pulmonary diagnosis. Early recognition of respiratory deterioration is important because severe pulmonary hemorrhage has been associated with high mortality [7].

Renal involvement is common in severe leptospirosis and may manifest as AKI [8,9]. Hemoptysis in a patient with suspected leptospirosis is a red flag for pulmonary involvement and warrants prompt respiratory assessment and imaging [10]. Leptospirosis may mimic other tropical infections such as dengue and malaria; appropriate microbiological and serological testing is therefore important. Severe leptospirosis can be reversible with timely antimicrobial therapy and appropriate supportive management [11,12].

The manuscript does not report dialysis, urine output, electrolyte abnormalities, or the exact duration of renal dysfunction; these details would strengthen the case report if available.

CONCLUSION

The patient's creatinine improved from 3.90 mg/dL to 0.85 mg/dL with treatment and supportive care, demonstrating substantial renal recovery. The patient's favorable outcome emphasizes the importance of early diagnosis, appropriate antimicrobial therapy, careful fluid management, and close monitoring for multiorgan involvement. Fever with jaundice and AKI should prompt consideration of severe leptospirosis, particularly in appropriate epidemiological settings.

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REFERENCES

1. Bharti AR, Nally JE, Ricaldi JN, Matthias MA, Diaz MM, Lovett MA, *et al*. Leptospirosis: A zoonotic disease of global importance. Lancet Infect Dis 2003;3:757-71.
2. Levett PN. Leptospirosis. Clin Microbiol Rev 2001;14:296-326.
3. National Centre for Disease Control. National Guidelines on Diagnosis, Case Management, Prevention and Control

of Leptospirosis. New Delhi: Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India; 2015.
4. Costa F, Hagan JE, Calcagno J, Kane M, Torgerson P, Martinez-Silveira MS, *et al*. Global morbidity and mortality of leptospirosis: A systematic review. PLoS Negl Trop Dis 2015;9:e0003898.
5. Adler B, De La Peña Moctezuma A. Leptospira and leptospirosis. Vet Microbiol 2010;140:287-96.
6. World Health Organization. Human Leptospirosis: Guidance for Diagnosis, Surveillance and Control. Geneva: World Health Organization; 2003.
7. Faine S, Adler B, Bolin C, Perolat P. Leptospira and Leptospirosis. 2nd ed. Melbourne: MediSci; 1999.
8. Kularatne SA, Budagoda BD, De Alwis VK, Wickramasinghe WM, Bandara JM, Pathirage LP. Severe leptospirosis mimicking dengue haemorrhagic fever: A case report. Ceylon Med J 2011;56:168-9.
9. Gouveia EL, Metcalfe J, De Carvalho AL, Aires TS, Villasboas-Bisneto JC, Queirroz A, *et al*. Leptospirosis-associated severe pulmonary hemorrhagic syndrome, Salvador, Brazil. Emerg Infect Dis 2008;14:505-8.
10. Dolhnikoff M, Mauad T, Bethlem EP, Carvalho CR. Pathology and pathophysiology of pulmonary manifestations in leptospirosis. Braz J Infect Dis 2007;11:142-8.
11. Sitprija V, Losuwanrak K, Kanjanabuch T. Leptospirosis and the kidney. Semin Nephrol 2003;23:53-9.
12. Suputtamongkol Y, Niwattayakul K, Suttinont C, Losuwanaluk K, Limpaiboon R, Chierakul W, *et al*. An open, randomized, controlled trial of penicillin, doxycycline, and cefotaxime for patients with severe leptospirosis. Clin Infect Dis 2004;39:1417-24.

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