

CASE REPORT

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Fulminant diffuse alveolar hemorrhage in Weil's disease: A case report

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ABSTRACT

Introduction: Leptospirosis is a zoonosis with worldwide distribution. Its severe form (Weil's disease) is characterized by jaundice, renal dysfunction, and hemorrhagic diathesis. Hemorrhagic pulmonary involvement is a lethal, often underdiagnosed complication.

Case Report: A 34-year-old male farmer was admitted for fever, jaundice, and progressive symmetric muscle weakness in the lower limbs. Initial laboratory tests showed severe thrombocytopenia (5000/ μ L), acute kidney injury (creatinine 5.9 mg/dL), and hyperbilirubinemia. Despite empirical antibiotic management (doxycycline/ceftriaxone), the patient developed acute respiratory failure secondary to diffuse alveolar hemorrhage confirmed by bronchoscopy and imaging. Mechanical ventilation and hemodialysis were required. Serology (microscopic agglutination test) confirmed leptospirosis. The patient died due to pulmonary complications and multiorgan failure.

Conclusion: Leptospirosis should be considered in the differential diagnosis of patients with a febrile syndrome and muscle weakness in endemic areas. The onset of respiratory symptoms should raise concern for the development of alveolar hemorrhage, which carries an ominous prognosis.

Keywords: Acute respiratory distress syndrome, Leptospirosis, Spirochetal jaundice, Weil's disease

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INTRODUCTION

Leptospirosis is a zoonotic disease caused by spirochetes of the genus *Leptospira* [1–3]. Although most cases are mild, a proportion of patients develop the severe icterohemorrhagic form known as Weil's disease [2, 4, 5]. In recent years, a clinical phenotype characterized by Diffuse Alveolar Hemorrhage (DAH) has been recognized; it has mortality rates above 50% and often occurs before the classic renal failure develops [6–9]. We present the case of a young patient with occupational exposure who developed a fulminant course of leptospirosis with severe pulmonary involvement.

CASE REPORT

Subject background and present illness: A 34-year-old male farm worker, resident of a rural area in Veracruz (endemic zone), with no relevant past medical history or substance use, presented with a 7-day illness. The onset was marked by a sensation of an insect bite on the scalp, accompanied by fever up to 39 °C. Over the following four days, he developed progressive, symmetric weakness in the lower limbs that gradually limited his ability to ambulate. Given the neurological presentation, Guillain-Barré syndrome was initially suspected, prompting an emergency visit and subsequent hospital admission.

Physical examination and diagnostic studies: Physical examination was notable for generalized jaundice. Laboratory panel performed at admission revealed pancytopenia with critical thrombocytopenia, meeting multiple organ failure criterion:

- Complete Blood Count (CBC): Hb 11.6 g/dL, platelets 5000/μL, leukocytes 8900/μL.
- Renal function and electrolytes: Creatinine 5.9 mg/dL, urea 175 mg/dL, potassium 3.3 mmol/L, sodium 130 mmol/L.
- Liver function: Total bilirubin 10.2 mg/dL (direct 8.6 mg/dL), Aspartate Aminotransferase/Transaminase Glutamic-Oxaloacetic (AST/TGO) 93 U/L, Alanine Aminotransferase/Transaminase Glutamic-Pyruvic (ALT/TGP) 93 U/L, alkaline phosphatase 143 U/L.

Therapeutic approach and clinical course: Given the epidemiological context and clinical findings, the working differential included rickettsiosis, Lyme disease, and leptospirosis (Weil's disease). Empiric antimicrobial coverage was initiated with doxycycline (100 mg every 12 hours) and ceftriaxone (2 g every 12 hours), and a microscopic agglutination test (MAT) was requested to confirm etiology.

By hospital day 3, the patient's condition deteriorated acutely. New-onset dyspnea and oxygen desaturation prompted an urgent chest X-ray, which revealed bilateral radiopacities consistent with diffuse alveolar hemorrhage (DAH) (Figure 1). Non-contrast chest computed tomography (CT) confirmed a bilateral ground-glass pattern with hyperdensity in the left bronchus (Figure 2). Arterial blood gas analysis demonstrated severe metabolic acidosis (pH 7.2, PaCO₂ 35 mmHg, HCO₃⁻ 14 mEq/L) with a PaO₂/FiO₂ ratio of 130, necessitating orotracheal intubation and initiation of invasive mechanical ventilation in Assist-Control Volume Control (ACVC) mode, with a positive end-expiratory pressure (PEEP) of 12 cmH₂O and FiO₂ of 0.80. Organ dysfunction severity was reflected by a Sequential Organ Failure Assessment (SOFA) score of 15. Given progressive anuria and refractory azotemia, a *Mahurkar* catheter was placed for renal replacement therapy via hemodialysis.

Outcome: The clinical trajectory was unfavorable, with evolution toward severe acute respiratory distress

syndrome (ARDS). On the hospital day 13, bronchoscopy confirmed the presence of diffuse active bleeding (Figure 3). On the hospital day 20, the positive report for leptospirosis was received (Table 1). The patient died on hospital day 21 due to mixed shock and multiorgan failure.

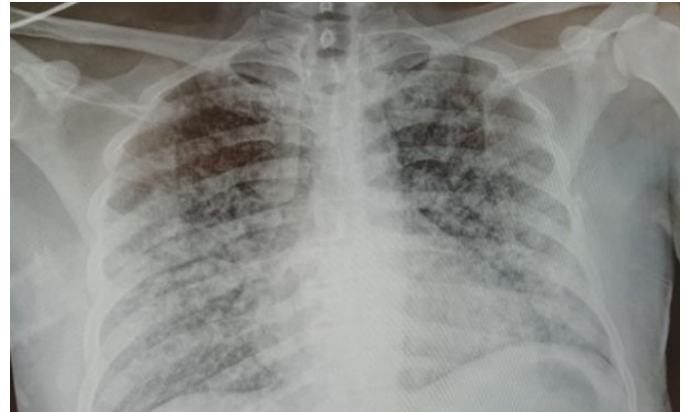


Figure 1: Chest radiograph: anteroposterior view with bilateral nodular radiopacities.

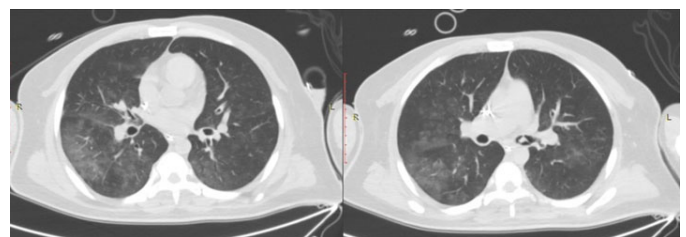


Figure 2: Non-contrast chest CT, lung window: bilateral ground-glass pattern, as well as hyperdensity in the left bronchus.

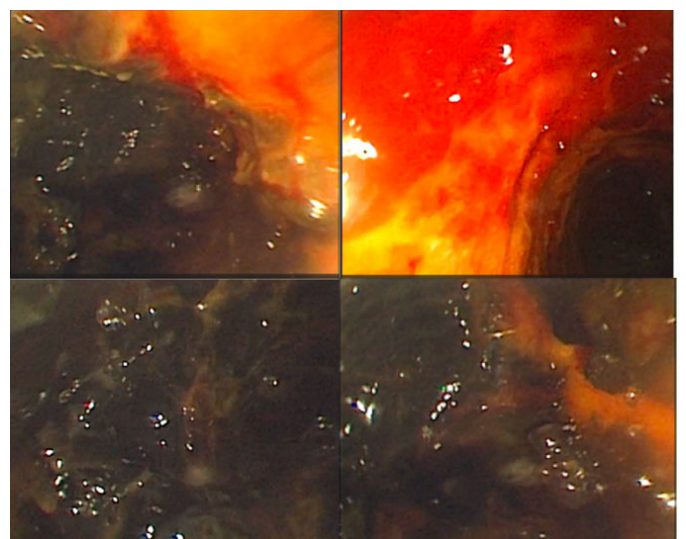


Figure 3: Bronchoscopy showing erythematous lesions in the mucosa and a clot in the left bronchus.

DISCUSSION

Leptospirosis is a globally distributed zoonosis with a pleomorphic clinical presentation, posing a significant

diagnostic challenge in endemic areas. The case presented illustrates the most severe form of the disease (Weil’s disease), characterized by the triad of jaundice, renal dysfunction, and hemorrhagic diathesis, culminating in fatal Diffuse Alveolar Hemorrhage (DAH) [1, 3, 6, 7, 8, 10, 11].

A distinctive feature of this case was the initial neurological presentation, which mimicked Guillain–Barré syndrome (GBS) due to progressive symmetric weakness in the lower limbs that impaired ambulation. While leptospirosis can cause neurological complications such as aseptic meningitis, flaccid paraparesis is less common and is usually secondary to severe myositis, rhabdomyolysis, or severe hypokalemia. Although our patient had mild hypokalemia (3.3 mmol/L), this alone does not fully account for the magnitude of weakness observed. A direct inflammatory muscle component may have contributed, though the precise pathophysiological mechanism in this case remains uncertain and cannot be established definitively from the available clinical data [2, 7, 8, 10, 11, 12, 13].

Progression to severe pulmonary involvement determined the fatal outcome. Pulmonary manifestations of leptospirosis occur in 20–70% of cases, but massive DAH is an uncommon complication, with case-fatality rates that may exceed 50% even with advanced ventilatory support [1, 11, 12]. In this patient, the sudden onset of

dyspnea and bilateral radiopacities on the third hospital day coincides with the immune phase of the disease, in which immune complex deposition and systemic vasculitis play a crucial pathogenic role. Despite management with protective mechanical ventilation and hemodynamic support, progression to refractory hypoxemia (PaO₂/FiO₂ 130) and pneumothorax underscores the aggressiveness of the hemorrhagic pulmonary form [12, 13].

From a hematologic standpoint, the critical thrombocytopenia on admission (5000/μL) is notable. Although thrombocytopenia is a classic finding in leptospirosis, levels below 20,000/μL are exceptional and are strongly associated with a worse prognosis and a higher risk of alveolar bleeding. This finding, together with marked jaundice (total bilirubin 10.2 mg/dL) and acute kidney injury (creatinine 5.9 mg/dL), confirms the clinical diagnosis of a complete Weil’s disease [14, 15].

It is relevant to mention the epidemiological context and the initial confusion with rickettsiosis due to the history of a presumed scalp bite. In rural areas of Mexico, coinfection or clinical overlap among leptospirosis, dengue, and rickettsiosis requires a broad syndromic approach. However, the presence of a marked cholestatic pattern with moderately elevated transaminases (<200 U/L) favors leptospirosis over viral hepatitis or fulminant rickettsiosis [16, 17].

Table 1: Microscopic agglutination test result for *Leptospira*

Leptospira determination			
Microagglutination test		Antibodies titration	Antibodies titration
<i>Leptospira interrogans</i> serovar Australis		1:80	<i>Leptospira interrogans</i> serovar Canicola
<i>Leptospira interrogans</i> serovar Hardjo		<1:80	<i>Leptospira interrogans</i> serovar Hebdomadis
<i>Leptospira interrogans</i> serovar Copenhageni		1:640	<i>Leptospira borgpetersenii</i> serovar Castellonis
<i>Leptospira interrogans</i> serovar Autumnalis		1:320	<i>Leptospira borgpetersenii</i> serovar Javanica
<i>Leptospira interrogans</i> serovar Pyrogenes		<1:80	<i>Leptospira noguchil</i> serovar Panama
<i>Leptospira borgpetersenii</i> serovar Sejroe		<1:80	<i>Leptospira interrogans</i> serovar Bratislava
<i>Leptospira interrogans</i> serovar Icterohaemorrhagiae		1:320	<i>Leptospira borgpetersenii</i> serovar Ballum
<i>Leptospira kirshneri</i> serovar Cynopteri		<1:80	<i>Leptospira santarosai</i> serovar Georgia
<i>Leptospira interrogans</i> serovar Pomona		<1:80	<i>Leptospira santarosai</i> serovar Borincana
<i>Leptospira borgpetersenii</i> serovar Tarassovi		1:80	<i>Leptospira interrogans</i> serovar Djasiman
<i>Leptospira kirshneri</i> serovar Grippothyphosa		1:160	<i>Leptospira santarosai</i> serovar Alexi
<i>Leptospira interrogans</i> serovar Wolffi		<1:80	<i>Leptospira interrogans</i> serovar Mankarso
<i>Leptospira interrogans</i> serovar Bataviae		1:640	
Final result: Positive			

CONCLUSION

This case highlights the importance of considering leptospirosis in the differential diagnosis of patients with fever and acute muscle weakness, beyond the classic suspicion of Guillain–Barré syndrome. It also underscores the need for strict respiratory monitoring in patients with severe thrombocytopenia, since diffuse alveolar hemorrhage may occur suddenly and catastrophically, and is the leading cause of death in modern icterohemorrhagic leptospirosis.

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Artificial intelligence (AI) use in the article:

Anthropic Claude Sonnet 4.6 (Released on 17-Feb-2026) was used to verify the translation and language used in the manuscript text.

Author Contributions

Héctor Escobedo-López – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guillermo Sánchez-López – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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