Respir Case Rep 2026;15(1):28-31 DOI: 10.5505/respircase.2026.27870

A Case of Leptospirosis Causing Pulmonary Hemorrhagic Syndrome

Pulmoner Hemorajik Sendroma Neden Olan Leptospiroz Olgusu

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Abstract

Leptospirosis is a zoonotic infection caused by Leptospira spirochetes. We present here the case of a 31-year-old male with alveolar hemorrhage, renal, and hepatic dysfunction who reported abdominal pain and weakness after cleaning out a warehouse containing rats 2 weeks earlier. Physical examination revealed abdominal tenderness but no other significant findings. As his clinical condition worsened, he developed cough and shortness of breath. Chest radiography revealed bilateral consolidation, and computed tomography (CT) of the chest revealed ground-glass opacities. Bronchoalveolar lavage pathology identified hemosiderin-laden macrophages. Leptospirosis testing confirmed antibodies against L. icterohaemorrhagiae with a titer of 1/800. The patient was placed on antibiotic therapy, corticosteroids, and plasmapheresis. Following treatment, his symptoms and laboratory results improved, and he was discharged.

Keywords: *Pulmonary hemorrhage, leptospirosis, Weil Disease.*

Öz

Leptospiroz, leptospira cinsi spiroketlerin neden olduğu zoonotik bir hastalıktır. Bu yazıda, alveoler hemoraji, böbrek ve karaciğer fonksiyon bozukluğu olan bir olgu sunuldu. Otuz bir yaşında erkek hasta, karın ağrısı ve halsizlik şikayeti ile başvurdu. Anemnezinden 2 hafta öncesinde farelerin bulunduğu bir depoyu temizlediği öğrenildi. Fizik muayenesinde batında hassasiyet dışında bulgu saptanmadı. Klinik, radyolojik ve laboratuvar parametrelerinin takibi sırasında hastada öksürük ve nefes darlığı şikayetleri ortaya çıktı. Bilgisayarlı toraks tomografisinde her iki akciğerde dağınık yamasal buzlu cam dansiteleri saptandı. Bronkoalveolar lavaj patolojisinde hemosiderin yüklü makrofajlar izlendi. Hastadan leptospiroz için gönderilen tetkik sonucunda L. icterohaemorrhagiae antikoru 1/800 titrede pozitif saptandı. Multisistemik tutulumu olan hastanın tedavisinde antibiyoterapi, kortikosteroid ve plazmaferez uygulandı. Tedaviler sonrasında hastanın semptomları ve laboratuvar değerlerinde düzelme sağlanarak hasta taburcu edildi.

Anahtar Kelimeler: *Pulmoner hemoraji, leptospirozis, Weil Hastalığı.*

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Submitted (Başvuru tarihi): 27.08.2025 ***Accepted (Kabul tarihi):*** 15.12.2025

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Pleural effusion is characterized by the abnormal accumulation of fluid within the pleural space between the visceral and parietal pleura. It has many possible causes, including infective, malignant, and systemic inflammatory disorders, and congestive heart failure in particular. The accumulations of fluid can impair lung expansion during inspiration by preventing adequate lung expansion (1).

Leptospirosis is a zoonotic disease caused by spirochetes of the Leptospira genus. It is commonly observed in tropical regions and in our country (1). Infection typically occurs as a result of direct contact with infected animals or contaminated water or soil. The disease is most frequently seen among farmers, soldiers, miners, veterinarians, and sewer workers (2). The clinical presentation of leptospirosis can range from asymptomatic or mild cases in 90% of cases to severe forms such as Weil's disease or pulmonary hemorrhagic syndrome (PHS) with multi-organ involvement in the remaining 10%. Pulmonary hemorrhagic syndrome is one of the leading causes of death in leptospirosis and has a mortality rate of 40–60% (3). We present here a case of leptospirosis characterized by pulmonary hemorrhagic syndrome that was treated with plasmapheresis and high-dose steroids. Written informed consent was obtained from the patient prior to the preparation of this report.

CASE

A 31-year-old male patient presented to the emergency department with complaints of abdominal pain and weakness. No significant findings were noted in the patient's medical history. A detailed anamnesis revealed that the patient, who is a refugee in our country, had been employed to clean out a warehouse in which rats were present 2 weeks prior to the onset of symptoms.

Physical examination revealed no findings other than abdominal tenderness. Laboratory tests performed in the emergency department produced the following results: urea, 239.3 mg/dL (17–43); creatinine, 6.31 mg/dL (0.67–1.17); AST, 50.3 U/L (0-50); ALT, 52.3 U/L (0–50); CRP, 249.8 mg/L (0–5); WBC, 1900/mm^3; Neutrophils, 1500/mm^3; Hb, 15.2 g/dL (12–17); and Platelets, 39,000/mm^3. Arterial blood gas analysis indicated metabolic acidosis. After initial assessment, the patient underwent hemodialysis and was started on empirical ceftriaxone therapy. Following initial assessment and acute management in the emergency department, the patient was transferred to the Infectious Diseases Department for further inpatient care. While monitoring the clinical, radiological, and laboratory parameters, the patient developed cough and shortness of breath, along with elevated total bilirubin levels and decreased platelet count, leading to the administration of plasmapheresis. Therapeutic plasma exchange was conducted within the Infectious Diseases inpatient service throughout the patient's admission (Table 1).

Table 1: *Laboratory findings according to the patient's hospitalization days*

Lab Findings	Admission	Day 3	Day 5	Day 8	Day 10	End of therapy
HB (g/dL)	15.2	10.1	9.9	10.1	8.6	9.7
WBC (/mm^3)	1900	7820	12600	20500	9100	10600
Neutrophil (/mm^3)	1500	4800	8300	17000	7660	6500
Thrombocyte (/mm^3)	39000	27000	31000	305000	541000	374000
Urea (mg/dL)	239	132	129	26	36	35
Creatinine (mg/dL)	6.31	2.6	2	0.7	0.6	0.8
AST (U/L)	50	33	35	55	31	42
ALT (U/L)	52	41	39	55	56	73
Total Bilirubin (mg/dL)	2.64	10.6	14.6	25.4	6.6	2.8
CRP (mg/L)	249	137	43.6	31	7.8	4.1

Serological tests, including direct and indirect Coombs, Anti-HAV IgM, Anti-HCV, Anti-HIV, HBsAg, Brucella IgG, IgM, Anti-CMV IgM, and EBV IgM, were all negative. Peripheral blood examination showed no plasmodium infection. Cultures of blood, urine, sputum, and bronchoalveolar lavage revealed no growths. The bronchoalveolar lavage culture was negative for acid-fast bacilli. Reverse transcription PCR and a respiratory virus panel also returned negative results.

In the radiological findings, a chest X-ray at the time of admission was evaluated as normal, while a follow-up chest X-ray on the 5th day after the onset of respiratory symptoms revealed consolidation areas in all bilateral lung zones (Figure 1). A chest computerized tomography (CT) revealed scattered patchy ground-glass densities in both lungs (Figure 2).

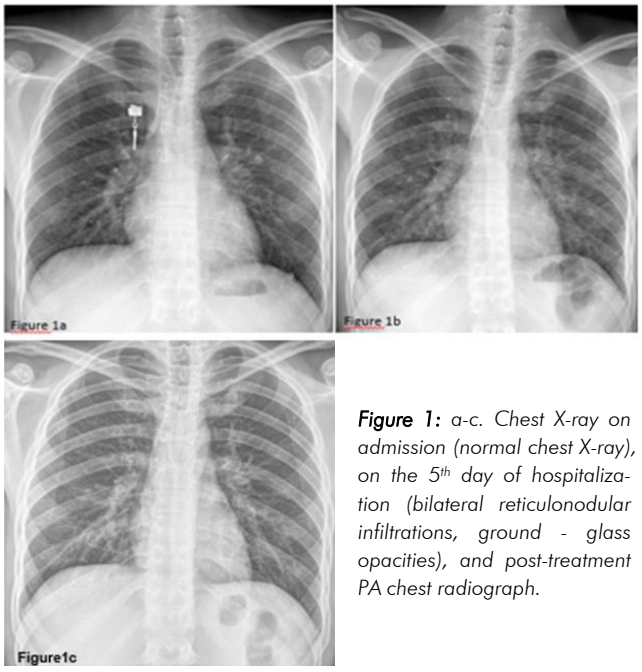

Figure 1: *a-c. Chest X-ray on admission (normal chest X-ray), on the 5th day of hospitalization (bilateral reticulonodular infiltrations, ground - glass opacities), and post-treatment PA chest radiograph.*

Magnetic resonance cholangiopancreatography (MRCP) was normal. Bronchoscopy revealed no endobronchial lesions, while hemosiderin-laden macrophages were noted in bronchoalveolar lavage pathology. The leptospirosis test results showed a positive antibody titer of 1/800 for *L. icterohaemorrhagiae*. The analysis was performed using the ELISA method.

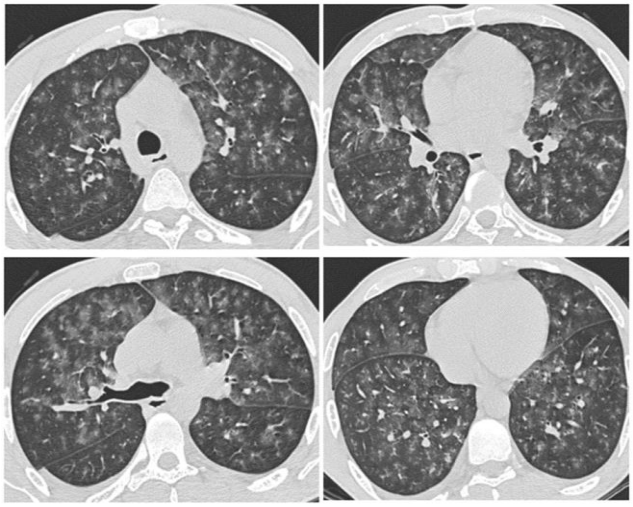

Figure 2: *Bilateral ground-glass opacities seen on thorax CT on the 5th day of hospitalization*

Alveolar hemorrhage due to leptospirosis was suspected based on anamnesis, clinical, laboratory, and radiological findings, for which the patient was started on ceftriaxone therapy. The patient was referred to the Pulmonology Department due to respiratory symptoms and radiologic abnormalities observed on chest imaging. After bronchoscopic lavage, high-dose methylprednisolone (500 mg/day) was added to the treatment, and the dosage was gradually reduced every 3 days, ultimately being discontinued after 15 days. Ceftriaxone therapy was completed to a total duration of 14 days and subsequently discontinued.

On the 3rd day of methylprednisolone treatment, the patient's respiratory symptoms started to decrease, bilirubin levels dropped, and platelet count returned to normal. Following treatment, the patient was discharged in a stable condition.

DISCUSSION

In cases of leptospirosis, pulmonary involvement may present with mild respiratory symptoms or pulmonary hemorrhage and acute respiratory distress syndrome (ARDS), accompanied by high fever, thrombocytopenia, renal failure, and jaundice (4,5). Clinical manifestations, including cough, dyspnea, and hemoptysis, typically emerge after the 4th day of illness. In pulmonary leptospirosis, reticular and nodular consolidations in the bilateral lower lung zones are revealed by chest radiography (6). High-resolution computed tomography (HRCT) typically demonstrates diffuse areas of consolidation with ground-glass opacities affecting all pulmonary lobes (7). In our patient, thrombocytopenia, hyperbilirubinemia, and renal failure were evident from the onset of illness. From the 5th day onward, pulmonary manifestations and radiographic abnormalities became more prominent, in line with previously reported findings in the literature. Clinical, laboratory, and radiological findings of leptospirosis are nonspecific and can be observed in many other diseases, and so differential diagnosis includes numerous infectious diseases, including influenza, HIV during the seroconversion period, dengue fever, viral hemorrhagic fevers, typhoid fever, malaria, brucellosis, rickettsioses, viral hepatitis, infectious mononucleosis, encephalitis, poliomyelitis, hantavirus infections, and respiratory infections such as viral pneumonia, bronchopneumonia, and tuberculosis (8). Leptospira infection is typically linked to exposure to contaminated water and may show jaundice and conjunctival suffusion, which are useful distinguishing signs. Hantavirus infection is associated with inhalation of rodent excreta and is characterized by either severe pulmonary edema (cardiopulmonary syndrome) or prominent renal involvement with thrombocytopenia (hemorrhagic fever with renal syndrome). Rickettsial infections are transmitted by ticks or lice and often present with an eschar or a petechial rash due to endothelial vasculitis. Differences in exposure history, presence of rash or eschar, and the pattern of organ involvement are central to the differentiation of these infections in clinical practice. In our case, the differential diagnosis from other diseases was established based on serological methods and cultures.

For the treatment of leptospirosis, antibiotic therapy should be initiated early to shorten the duration of symptoms, reduce morbidity and mortality, and decrease the urinary excretion of the microorganism. In addition to antibiotic therapy, patients should be closely monitored for such life-threatening complications as dehydration, hypotension, severe hemorrhage, and prolonged renal failure, and supportive treatments should be initiated.

In mild cases, oral doxycycline, azithromycin, and amoxicillin are preferred, while severe cases will require the parenteral administration of such agents as penicillin G, doxycycline, ceftriaxone, or cefotaxime (9–11). Previous studies in the literature include case reports documenting the use of corticosteroids, plasmapheresis, and extracorporeal membrane oxygenation (ECMO) for the management of leptospirosis-associated pulmonary hemorrhagic syndrome and acute respiratory distress syndrome (ARDS) (12–14).

Publications have reported that mortality can be reduced in affected patients through the early administration of high-dose methylprednisolone when respiratory symptoms become prominent, and plasmapheresis performed in conjunction with corticosteroid therapy (1,12,15). Our case was started on ceftriaxone for antibiotic therapy and underwent plasmapheresis. Subsequently, high-dose methylprednisolone was administered due to the development of pulmonary hemorrhage. Following treatment, a prompt resolution of clinical manifestations, radiological abnormalities, and laboratory findings was observed.

In conclusion, although rare, leptospirosis can cause pulmonary hemorrhagic syndrome with high mortality. In the presence of a relevant history and appropriate clinical and laboratory findings, the early initiation of antibiotic therapy is recommended. When respiratory symptoms develop, the addition of corticosteroids and plasmapheresis to the treatment should be considered as they may reduce mortality.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - E.A.A., L.Ö., S.G., M.U., O.G.; Planning and Design - L.Ö., E.A.A., S.G., M.U., O.G; Supervision - O.G, E.A.A., S.G., M.U., L.Ö.; Funding -; Materials - S.G., M.U., O.G; Data Collection and/or Processing - S.G., M.U.; Analysis and/or Interpretation - E.A.A.; Literature Review - E.A.A., L.Ö.; Writing - E.A.A.; Critical Review - L.Ö.

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