Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Anesthesia

Acute Respiratory Distress Syndrome as a Rare Manifestation of Leptospirosis: A Case Report

Grine Ali[1*], Belghiti Amine[1], Abdellaoui Mohamed[2], Boucaid Abdelhalim[3], Doghmi Naoufal[1], Balkhi Hicham[1]

[1]Medical Intensive Care Unit, Department of Anesthesia and Critical Care, Mohammed V Military Teaching Hospital, Rabat, Morocco
[2]Radiology Department, Mohammed V Military Teaching Hospital, Rabat, Morocco
[3]Pneumology Department, Mohammed V Military Teaching Hospital, Rabat, Morocco

DOI: https://doi.org/10.36347/sjmcr.2024.v12i12.025 | **Received:** 09.11.2024 | **Accepted:** 12.12.2024 | **Published:** 16.12.2024

***Corresponding author:** Grine Ali
Medical Intensive Care Unit, Department of Anesthesia and Critical Care, Mohammed V Military Teaching Hospital, Rabat, Morocco

Abstract **Case Report**

Pulmonary leptospirosis is rare and causes alveolar hemorrhages that may be complicated by acute respiratory distress syndrome (ARDS). We report a case of leptospirosis revealed by severe febrile pneumonia complicated by acute respiratory distress. Its diagnosis is challenging when pulmonary involvement predominates, but it often reflects multivisceral involvement that may be occult and must be investigated to avoid diagnostic delays potentially endangering the patient's life.

Keywords: Pulmonary leptospirosis, Acute respiratory distress syndrome (ARDS), Alveolar hemorrhage, Febrile pneumonia, Sub-Saharan Africa.

INTRODUCTION

Icterohemorrhagic leptospirosis typically manifests as a triad of hepatic, renal, and meningeal involvement. Rarely, it can present with pulmonary manifestations, leading to alveolar hemorrhages and potentially progressing to ARDS [1].

We report a case of leptospirosis revealed by severe febrile pneumonia complicated by ARDS.

CASE PRESENTATION

A 31-year-old man, without a medical history, was admitted to the emergency department for dyspnea, coughing, and moderate hemoptysis, with a fever of 39°C. Symptoms began six days earlier, shortly after his return from a mission in sub-Saharan Africa, where he reported swimming in freshwater. Initial symptoms included an abrupt onset of flu-like febrile syndrome characterized by intense myalgia, arthralgia, asthenia, and headaches. Two days before admission, his condition worsened with progressive respiratory distress, coughing, and moderate haemoptysis.

Upon admission, the physical examination revealed a conscious, febrile (39°C) patient, pale but without overt jaundice, normotensive (105/70 mm Hg), tachypneic with a respiratory rate of 26 breaths/min, oxygen saturation at 88% on room air, and pulmonary crackles on auscultation. Due to the severity of his condition, he was transferred to the intensive care unit, where an initial biological and radiological workup was performed.

Arterial blood gases revealed a PaO_2 of 69 mm Hg. Blood tests showed hypochromic microcytic anemia (10 g/dL), leukocytosis (14,000/mm³ with 70% neutrophils), and moderate thrombocytopenia (74,000/mm³). C-reactive protein was elevated at 135 mg/L. Liver function tests indicated moderate cytolysis and cholestasis (AST 80 IU/L, ALT 98 IU/L, total bilirubin 19 IU/L). Additional findings included rhabdomyolysis (CK 1400 IU/L), proteinuria (250 mg/24h), normal creatinine levels, and preserved diuresis. Electrolytes were normal.

A chest CT scan revealed diffuse interstitial ground-glass opacities indicative of intra-alveolar haemorrhage (IAH), confluent alveolar consolidations consistent with pulmonary oedema, and low-volume bilateral pleural effusion (Figure1). No mediastinal lymphadenopathy was observed.

Treatment included tracheal intubation, colloid fluid resuscitation, hemostatic therapy, injectable proton pump inhibitors, and empiric antibiotics (amoxicillin 3 g/day and ofloxacin 400 mg/day). Clinical improvement

in respiratory and hemodynamic status was observed by day three.

Serology using the Martin and Pettit method confirmed IgM positivity for Leptospira interrogans serovar Icterohaemorrhagiae (SIH) on day eight after symptom onset, with a fourfold increase in titers between two samples, confirming the diagnosis. Blood and urine cultures were negative for spirochetes under dark-field microscopy, and PCR testing was not performed. Tests for Mycobacterium tuberculosis and malaria were negative.

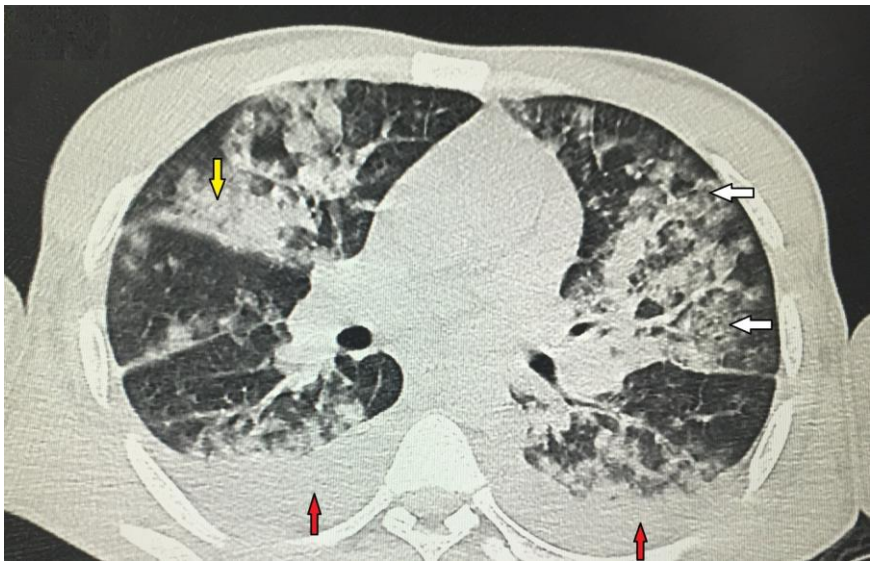

Figure 1: Axial chest CT scan: diffuse ground-glass opacities (white arrow), confluent alveolar consolidations (yellow arrow), and low bilateral pleural effusion (red arrow)

DISCUSSION

Leptospirosis is a ubiquitous bacterial zoonosis caused by spirochetes of the genus Leptospira and the species Leptospira interrogans. It was first described in the early 1880s and later by Weil in 1886 [2, 3].

In Africa, leptospirosis is recognized in North Africa. In sub-Saharan Africa, many countries have bioclimatic conditions favorable for Leptospira transmission, but the incidence and prevalence remain difficult to evaluate, making mapping impossible.

Globally, leptospirosis is estimated to account for more than 500,000 severe cases annually, with a fatality rate of around 20%. Its frequency is undoubtedly underestimated among tropical travelers [3].

The reservoir is primarily animal-based, including asymptomatic rodents. However, most domestic and wild mammals can be infested. Transmission is usually occupational and predominantly affects humans, with an average age of onset of 44 years. It occurs either directly through exposure to the urine of infected animals or indirectly through water contaminated with urine. A resurgence of the disease is currently observed in patients engaged in recreational freshwater activities (swimming, water sports, fishing), as seen in our patient [3, 4].

Although Leptospira were long considered strictly extracellular bacteria, their intracellular penetration capacity, particularly of the SIH serovar, is now proven [3].

Among pathogenic serogroups, the serovar Icterohaemorrhagiae remains predominant and is responsible for the most severe cases, which aligns with our case. However, severe forms can also occur with all other serogroups [3, 4].

Leptospirosis causes highly variable manifestations, ranging from anicteric flu-like syndrome (80% of cases) to icteric multivisceral forms or Weil's disease, which can be potentially fatal (20% of cases) [3]. In severe cases, all target organs are affected, albeit with varying intensity. The reason for this variability in intensity remains unexplained [5]. Hepatic involvement is nearly constant and appears as a diagnostic clue, seen in 70–80% of cases [4]. Our patient presented moderate cholestasis and cytolysis without jaundice. Renal involvement is common, linked to acute non-specific tubulointerstitial nephritis. It is clinically insignificant in 70–80% of cases, revealed by simple proteinuria, microscopic hematuria, or leukocyturia. Acute renal failure is a severe complication aggravated by rhabdomyolysis, requiring dialysis in 10–15% of cases [3, 4]. Our patient presented proteinuria but no renal failure. Rhabdomyolysis is explained by the direct pathogenic effect of Leptospira, which causes necrosis of infected muscle cells [4], as observed in our case.

Pulmonary forms were first reported by Silverstein in 1953 [6]. Pulmonary involvement occurs

in 20–40% of cases [4]. It involves endothelial lesions of the capillaries, resulting in diffuse or focal intra-alveolar hemorrhages (IAH) associated with pulmonary edema, which may progress to ARDS [3, 4]. Symptoms vary depending on the severity of the involvement: cough, dyspnea, chest pain, and hemoptysis, which is rarely a revealing symptom, as shown in our observation. Imaging reveals interstitial ground-glass opacities associated with IAH and extensive pulmonary consolidations [3, 7], as clearly demonstrated on the CT scan of our patient. Mortality doubles in cases of pulmonary involvement, exceeding 60% in ARDS cases [4, 7]. Severe pulmonary forms may predominate and occur even in the absence of jaundice [7], as illustrated by our observation, complicating diagnosis. Furthermore, our patient returned from a malaria-endemic area, initially suggesting severe malaria. Pulmonary radiological signs or IAH may be detected without respiratory symptoms, contributing to the underestimation of minor pulmonary forms overshadowed by more severe involvement of other organs [5, 7]. This suggests that IAH is constant in leptospirosis but may remain asymptomatic in milder cases. Performing bronchoalveolar lavage and CT scans could detect IAH regardless of severity [5].

Confirmation of the diagnosis is based on serology and bacteriology. Serology is difficult to interpret, with positivity often delayed (15 days or more), necessitating repeated sampling, especially in severe forms. The reference test is the Martin and Pettit microagglutination test (MAT). Direct examination does not allow for rapid diagnosis, and culture is slow and difficult. PCR, however, allows for a quick and early diagnosis from the first day of illness [3, 4].

Treatment is classically based on penicillin G. Other effective antibiotics include beta-lactams (ampicillin, amoxicillin, and third-generation cephalosporins) and tetracyclines [3, 8]. Our patient received empiric antibiotic therapy also covering Leptospira.

Due to the severity of the disease, vaccination is recommended for at-risk professions. The vaccine (SPIROLEPTR) is only effective against the SIH serovar and is rarely used [3, 4]. In cases of risk of exposure to contaminated water, chemoprophylaxis with doxycycline 200 mg per week is 95% effective [3, 8].

CONCLUSION

Leptospirosis's clinical variability poses diagnostic challenges, particularly when pulmonary involvement predominates. Recognizing the potential for multivisceral involvement is essential to avoid diagnostic delays that could endanger the patient's life.

Conflicts of Interest: The authors declare no conflicts of interest.

REFERENCES

1. Luks, A. M., Lakshminarayanan, S., & Hirschmann, J. V. (2003). Leptospirosis presenting as diffuse alveolar hemorrhage: case report and literature review. *Chest*, *123*(2), 639-643.
2. Nardone, A., Capek, I., Baranton, G., Campèse, C., Postic, D., Vaillant, V., ... & Desenclos, J. C. (2004). Risk factors for leptospirosis in metropolitan France: results of a national case-control study, 1999–2000. *Clinical Infectious Diseases*, *39*(5), 751-753.
3. Leptospiroses-actualités. (2016). Médecine tropicale. Centre René Labusquière, Institut de Médecine Tropicale, Université de Bordeaux, 33076 Bordeaux (France), www.medecine tropicale.com (Mise à jour le 03/02/2017).
4. Sekkach, Y., Qaçif, H., Jira, M., El qatni, M., El omri, N., & Ghafir, D. (2007). Acute respiratory distress revelated a sever pulmonary leptospirosis. *La revue de médecine interne*, 28, 48-51.
5. Paganin, F., Bourdin, A., Borgherini, G., Dalban, C., Poubeau, P., Tixier, F., ... & Arvin-Berod, C. (2009). Manifestations pulmonaires de la leptospirose. *Revue des maladies respiratoires*, *26*(9), 971-979.
6. Silverstein, C. (1953). Pulmonary manifestations of leptospirosis. *Radiology*, *61*(3), 327-334.
7. Rakotomalala, R., Tiaray, M., Borgherini, G., Andrianarisoa, A., & Rasamindrakotroka, A. (2010). Should leptospirosis be suspected in several feverish pneumopathy? *Revue d'anésthésie-réanimation et de médecine d'urgence, 2*(3), 1-3.
8. Panaphut, T., Domrongkitchaiporn, S., Vibhagool, A., Thinkamrop, B., & Susaengrat, W. (2003). Ceftriaxone compared with sodium penicillin G for treatment of severe leptospirosis. *Clinical Infectious Diseases*, *36*(12), 1507-1513.