EAS Journal of Anaesthesiology and Critical Care
Abbreviated Key Title: EAS J Anesthesiol Crit Care
ISSN: 2663-094X (Print) & ISSN: 2663-676X (Online)
Published By East African Scholars Publisher, Kenya

OPEN ACCESS

Volume-8 | Issue-5 | Sep-Oct-2026 | DOI: https://doi.org/10.36349/easjacc.2026.v08i05.001

Case Report

Human Icterohemorrhagic Leptospirosis or Weil's Disease in the Intensive Care Unit of Dalal Jamm Hospital, Guediawaye: A Case Report

Niass E.H.T[1]*, Gaye I[1], Ndiaye A.P.N[1], Thiom C.O.L[1], Faye A[1], Camara L[1], Toure M.S[1], Leye P.A[1], Beye M.D.[1]

[1]Department of Anesthesiology and Intensive Care, Dalal Jamm Hospital Center, Dakar

Article History
Received: 25.07.2026
Accepted: 12.09.2026
Published: 21.09.2026

Journal homepage:
https://www.easpublisher.com

Quick Response Code

Abstract: ***Introduction*:** Leptospirosis is a bacterial zoonosis of worldwide distribution. Its clinical presentation is highly variable in terms of signs and severity. Weil's disease is characterized by the association of conjugated hyperbilirubinemia, sometimes severe, with hemorrhagic manifestations including hemoptysis and gastrointestinal bleeding. ***Case presentation*:** We report the case of a 27-year-old woman admitted to the intensive care unit for management of a hemorrhagic syndrome. She initially presented with an influenza-like illness treated by self-medication with paracetamol. On admission, the patient presented with febrile hepatonephritis associated with multiorgan failure, together with the classic triad of Weil's disease: conjugated hyperbilirubinemia, and hemorrhagic manifestations including hemoptysis and gastrointestinal bleeding. Leptospira IgM serology was positive, confirming the diagnosis. She was treated with ceftriaxone and supportive care. The clinical course was favorable, with discharge from the intensive care unit after 10 days of hospitalization. ***Conclusion*:** The presence of a hemorrhagic syndrome in the setting of febrile hepatonephritis should raise suspicion of human leptospirosis. Patient history-taking is essential and may reveal exposure to animals capable of transmitting the pathogen to humans. Definitive diagnosis relies on isolation of the organism or rapid IgM serological testing.

Keywords: Human Leptospirosis, Weil's Disease, Hepatonephritis, Jaundice, Hemorrhage.

INTRODUCTION

Leptospirosis is a bacterial zoonosis of worldwide distribution. It has long been referred to by several names reflecting its clinical spectrum: "swamp fever," "autumn fever," "rat-catcher's fever," and "hemorrhagic jaundice." Its first clinical description dates back to 1886, by Adolf Weil, a German physician who lent his name to the severe form of the disease. The bacterium responsible for the disease was first isolated and reported in 1915 in Japan by Inada *et al*., [1]. Its clinical presentation is highly variable in terms of signs and severity. Numerous differential diagnoses exist that, in the absence of an identified exposure risk for leptospirosis, can mislead the clinician and result in a potentially harmful delay in treatment [2]. The relative rarity of overt leptospirosis, particularly its florid form with hepatic, renal, or neurological involvement, nonetheless remains well established. With only 7 typical cases diagnosed in Dakar during the 1960s, the disease appears considerably less prevalent than in tropical regions of America or Asia [3].

We report a case of Weil's disease in a 27-year-old woman

CASE REPORT

A 27-year-old woman, with a known history of asthma since childhood and a history of dermocorticoid-induced depigmentation, presented with a symptomatology evolving over 10 days prior to hospitalization, marked by the onset of severe headaches associated with fever, relieved by oral paracetamol intake. Two days after the onset of headaches, vomiting appeared, associated with diffuse abdominal pain, without cessation of bowel movements or flatus, and without diarrhea.

On admission to the intensive care unit of Dalal Jamm Hospital in Guédiawaye, the patient presented with the following vital signs: blood pressure of 120/69 mmHg, heart rate of 86 bpm, tachypnea with a respiratory rate of 33 cycles/min and a pulse oxygen saturation (SpO2) of 94% on room air, temperature of

***Corresponding Author:** Niass E.H.T
Department of Anesthesiology and Intensive Care, Dalal Jamm Hospital Center, Dakar

37.2°C, capillary blood glucose of 1.42 g/L, and dark urine following urinary catheter placement.

Her general condition was classified as WHO 1, with icteric conjunctival mucosae, supple calves, and no lower limb edema. Consciousness was clear, with a Glasgow Coma Scale score of 15/15; pupils were isochoric and reactive, with no signs of meningeal irritation. Cardiovascular examination was normal. On respiratory examination, she presented with shallow tachypnea without signs of respiratory distress and clear lung fields. Cutaneous examination revealed purpura located on the flexural areas of the limbs and the external surface of the arm. Genital examination revealed endovaginal bleeding with dark blood clots.

History-taking revealed that the patient lived in a livestock-farming family, with sheep as livestock, and dogs present in the household.

Laboratory tests performed on admission revealed: hemoconcentration (hemoglobin 13.8 g/dL and hematocrit 42.6%), thrombocytopenia with a platelet count of 81,000/µL, functional acute kidney injury with blood urea of 1.27 g/L and serum creatinine of 42.6 mg/L, a nonspecific biological inflammatory syndrome (hyperleukocytosis at 49,000/µL, C-reactive protein greater than 200, and elevated erythrocyte sedimentation rate), procalcitonin at 54.31, marked hepatic cytolysis (AST 1670 [41×N] and ALT 1780 [50×N]), and biological cholestasis (gamma-GT = 242). The serum electrolyte panel was normal. Abdominopelvic ultrasonography showed homogeneous hepatomegaly with portal trunk flow not modulated by respiration, associated with moderate-volume ascites. The kidneys were enlarged, hyperechogenic, and poorly differentiated.

The diagnostic hypotheses considered in this setting were toxic and/or infectious hepatonephritis, arboviral infections, and thrombotic microangiopathies.

On admission, the patient was started on antibiotic therapy with ceftriaxone 2 g/day and metronidazole 1.5 g/day, vascular fluid resuscitation with crystalloids, base supplementation, and gastric protection with omeprazole.

Etiological work-up ruled out malaria, with a negative thick blood smear, and arboviral testing was negative. Leptospira IgM serology was positive at 55 IU/mL. Human leptospirosis in its icterohemorrhagic form was therefore diagnosed, and C3G (third-generation cephalosporin)-based treatment was maintained.

The clinical course was marked, on the 2nd day of hospitalization, by the onset of bicytopenia consisting of anemia of mixed inflammatory pattern with microcytosis and hemolytic features, with a schistocyte count of 2.2% without platelet aggregates, and thrombocytopenia at 66,000/µL. By the 5th day of treatment, regression of symptoms was observed, with cessation of bleeding, regression of jaundice, and resumption of diuresis marked by polyuria. After 7 days of treatment, clinical examination was normal. After one week of hospitalization with a favorable clinical course, our patient was transferred to the nephrology department for continued management.

Table I: Evolution of Biological Parameters during Hospitalization

Parameter	Two days before admission	On admission	Day 2	Day 5	Month 4 post-discharge
Sodium (mmol/L)	136	131	127	138	—
Potassium (mmol/L)	4.7	4.0	3.8	4.3	—
Chloride (mmol/L)	102	98	98	102	—
Hemoglobin (g/dL)	14.8	10	10	8	12.21
Hematocrit (%)	42.6	29	29	23	38.5
Leukocytes (/µL)	31,340	49,000	45,000	43,000	6,430
Platelets (/µL)	81,000	40,000	77,000	58,000	398,000
aPTT (s)	—	40	—	—	21
Prothrombin time (%)	—	66	—	—	98
Urea (g/L)	1.27	1.72	1.8	0.8	0.14
Creatinine (mg/L)	42.6	74	86	34	7
C-reactive protein (mg/L)	200	—	108	—	5
Procalcitonin (ng/mL)	—	54	—	—	0.05
Direct bilirubin	—	—	4.7×N	—	Normal
γ-GT	242	—	—	—	—
AST	41×N	—	2.5×N	—	Normal
ALT	50×N	—	8.5×N	—	Normal

DISCUSSION

The clinical presentation of leptospirosis is polymorphic and often nonspecific; however, certain patterns are more typical among patients admitted to intensive care units and should prompt consideration of the diagnosis—even in regions of low prevalence.

Typical features include febrile hepatorenal syndrome, multiorgan failure, and pulmonary involvement resembling acute respiratory distress syndrome (ARDS) with intra-alveolar hemorrhage (IAH). Organ involvement may also occur in isolation; thus, the presence of either acute renal failure with hypokalemia, conjugated hyperbilirubinemia with normal hepatic imaging, or thrombocytopenia should alert the clinician [5].

In our case, the diagnosis of leptospirosis was oriented by the discussion of etiologies of febrile hepatorenal syndrome in a context of multiorgan failure. The respiratory distress was moderate, and criteria for ARDS were not met.

Human infection mainly results from indirect contact with an infected animal, which carries leptospires in the renal tubules—particularly along the brush border—and contaminates the environment via urine [6]. Our patient lived in a family of livestock breeders (sheep) with dogs as domestic animals, consistent with a potential zoonotic exposure.

Humans may also be exposed directly to the secretions (urine, saliva) of infected animals through occupational contact—such as in farmers, veterinarians [15, 16], or slaughterhouse workers [17], or, less commonly, through recreational activities (hunting, farm visits) or animal bites [18].

The diagnosis of leptospirosis relies on a combination of clinical, epidemiological, and laboratory criteria. Clinical findings are highly variable due to the wide range of organ involvement by the spirochete. Epidemiological data help identify risk factors such as contact with contaminated animals or environments, or exposure common to previously documented cases. Laboratory confirmation depends on various techniques that can demonstrate the organism through culture, specific antibodies, or bacterial components such as DNA, housekeeping genes, or species-specific genes [4].

The classical triad defining Weil's disease includes conjugated hyperbilirubinemia (often marked), hemorrhagic manifestations (hemoptysis, gastrointestinal bleeding), and acute renal failure. This severe form may appear during the second week of illness or earlier, affecting approximately 10–15% of patients. Mortality rates vary depending on available resuscitation resources but typically exceed 10% **[7].** Our case was a typical form of Weil's disease presenting with the complete triad.

The recommended antibiotic for severe leptospirosis is penicillin G. However, from a practical standpoint, its narrow spectrum makes its empirical use difficult, as the initial presentation often mimics other acute bacterial infections requiring broader coverage (e.g., hepatobiliary infection, pneumonia, or typhoid fever). In severe cases, ceftriaxone [11], or cefotaxime [12], should therefore be preferred. For mild forms, oral doxycycline (100 mg twice daily) is recommended due to its efficacy in reducing both symptom duration and urinary bacterial excretion.

Macrolides (particularly azithromycin), amoxicillin, or ciprofloxacin are alternative options, although clinical data regarding fluoroquinolones remain limited. The WHO recommends a treatment duration of 5–7 days regardless of severity; however, some studies suggest that a 3-day regimen may be sufficient in rapidly improving cases [13, 14]. In our patient, ceftriaxone (2 g/day) was initiated prior to ICU admission and maintained due to favorable evolution of clinical and inflammatory parameters.

Classically, the disease course is biphasic. The first phase (bacteremic, anicteric) presents as a flu-like illness lasting about one week, which may resolve spontaneously or progress—after a symptom-free interval of 2–3 days—to a second immunologic phase characterized by icteric or icterohemorrhagic manifestations (in 10–15% of patients) or, occasionally, by a recurrence of fever, headache, and immune complications such as aseptic meningitis or uveitis**.** Progression to severe icteric forms may occur more rapidly. Complete recovery, including normalization of hepatic and renal abnormalities, may take up to 12 weeks [10].

In our case, the clinical course followed this classical pattern: an initial pseudo-flu-like phase, transiently controlled by self-medication with paracetamol, followed by an icterohemorrhagic phase prompting medical consultation. Reported mortality varies widely, from less than 5% to over 30%, depending on context and available medical resources [8].

The main risk factors for mortality described in the literature include altered mental status, renal failure (oliguria, hyperkalemia, creatinine >265 µmol/L), hypotension, advanced age (>30–40 years), and clinical or radiographic pulmonary involvement [9]. Our patient showed a favorable evolution under etiotropic therapy (ceftriaxone) and supportive treatment, consistent with outcomes reported in most published cases.

CONCLUSION

The occurrence of a hemorrhagic syndrome in the context of febrile hepatorenal failure should prompt consideration of human leptospirosis. A detailed medical history remains crucial, as it helps identify potential exposure to domestic or wild animals capable of transmitting the pathogen to humans. Definitive diagnosis relies on either isolation of the organism or rapid serological testing for anti-Leptospira IgM antibodies. Early recognition and prompt initiation of appropriate antibiotic therapy significantly improve

prognosis and reduce the risk of multiorgan failure and mortality.

REFERENCES

1. Inada R, Ido Y, Hoki R et al. (1916). The etiology, mode of infection, and specific therapy of Weil's disease (spirochaetosis icterohaemorrhagica). J Exp Med 23:377–402.
2. Ko AI, Galvão Reis M, Ribeiro DCM et al. (1999) Urban epide mic of severe leptospirosis in Brazil. Salvador Leptospirosis Study Group. Lancet 354:820–5.
3. Sankale M, Ruscher H, & Sarrat H. (1974) Nouvelle enquête sérologique, clinique et biologique sur la leptospirose à Dakar. Médecine et Maladies Infectieuses ; Volume 4, Issue 1, January, Pages 15-16, 19-20
4. A. Prisacariu, M. Fastenaekels, & J. Jobe. (2021) Leptospirose sévère compliquée de choc septique : à propos d'un cas. Journal Européen des Urgences et de Réanimation 33 ;3, pages 173 - 177
5. Grateau A, Allyn J, Gaüzère BA et al. (2023) Leptospirose en réanimation Méd. Intensive Réa. 32 (in press)
6. Le Turniera P, & Epelboin L. (2018) Mise au point sur la leptospirose. REVMED-5655 ; Pages 7
7. McBride AJA, Athanazio DA, Reis MG et al. (2005) Leptospirosis. Curr Opin Infect Dis;18:376–86.
8. World Health Organization ILS. (2003) Human leptospirosis guidance for diagno sis, surveillance and control. Geneva: World Health Organization
9. Haake DA, & Levett PN. (2015) Leptospirosis in humans. Curr Top Microbiol Immunol; 387:65–97.
10. Izurieta R, Galwankar S, & Clem A. (2008) Leptospirosis: the "mysterious" mimic. J Emerg Trauma Shock; 1:21–33.
11. Panaphut T, Domrongkitchaiporn S, Vibhagool A et al. (2003) Ceftriaxone compared with sodium penicillin g for treatment of severe leptospirosis. Clin Infect Dis; 36:1507–13.
12. Suputtamongkol Y, Niwattayakul K, Suttinont C et al. (2004) An open, randomized, controlled trial of penicillin, doxy cycline, and cefotaxime for patients with severe leptospirosis. Clin Infect Dis; 39:1417–24.
13. Faucher J-F, Chirouze C, Hoen B et al. (2013) Short-course treatment with ceftriaxone for leptospirosis: a retrospec tive study in a single center in Eastern France. J Infect Chemother; 21: 227–8.
14. Phimda K, Hoontrakul S, Suttinont C et al. (2007) Doxycycline versus azithromycin for treatment of leptospirosis and scrub typhus. Antimicrob Agents Chemother ; 51:3259–63.
15. Suwanpakdee S, Kaewkungwal J, White LJ et al. (2015) Spatio-temporal patterns of leptospirosis in Thailand: is flooding a risk factor? Epidemiol Infect; 143:2106–15.
16. Garcia-Ramirez LM, Giraldo-Pulgarin JY, Agudelo-Marin N et al. (2015) Geographical and occupational aspects of leptospirosis in the coffee-triangle region of Colombia, 2007-2011. Recent Pat Antiinfect Drug Discov;10:42–50
17. Dreyfus A, Wilson P, Collins-Emerson J et al. (2015) Risk factors for new infection with Leptospira in meat workers in New Zealand. Occup Environ Med; 72:219–25.
18. Gollop JH, Katz AR, Rudoy RC et al. (1993) Rat-bite leptospirosis. West J Med ; 159:76–7.

Cite this article: Niass, E. H.T., Gaye, I., Ndiaye, A. P. N., Thiom, C.O.L., Faye, A., Camara, L., Toure, M.S., Leye, P.A., & Beye, M.D. (2026). Human Icterohemorrhagic Leptospirosis or Weil's Disease in the Intensive Care Unit of Dalal Jamm Hospital, Guediawaye: A Case Report. *EAS J Anesthesiol Crit Care, 8*(5), 242-245. https://doi.org/10.36349/easjacc.2026.v08i05.001