Malaysian Journal of Medical Research, 10(2), 62-67
https://doi.org/10.31674/mjmr.2026.v010i02.07

MJMR
Malaysian Journal of Medical Research

Malaysian Journal of Medical Research

Online ISSN: 2550-1607

www.mjmr.com.my

Case Study

Severe Leptospirosis (Weil's Disease): An Interesting Case Report

Sk Hasan Habib[1], Md. Salamat Ali[2], Waliza Ansar[3*]

[1]Krishnanagar Medical College, Krishnanagar, Larica Tolly, Kolkata 741102, India

[2]Department of ICU, Fleming Hospital, Mirania Gardens, East Topsia, Topsia, Kolkata, West Bengal 700046, India

[3]Department of Zoology, Behala College (Autonomous), Dhopapara, Parnasree Palli, Behala, Kolkata, West Bengal 700060, India

Corresponding Author's Email: waliza_ansar@yahoo.co.in

Abstract

Leptospirosis is underrated zoonotic disease caused by pathogenic *Leptospira* species in warm climate with poor sanitation. It is characterized by varying asymptomatic infection to mild Leptospirosis with severe fulminant disease. Misdiagnosis and management increase the risk of mortality. A rare case study of human Leptospirosis with serum bilirubin over 42 mg/dl, high urea (155 mg/dl), high creatinine (6.5 mg/dl), pulmonary hemorrhage, deranged coagulation profile (INR 3), with severe metabolic acidosis (pH 7.2 in ABGA) in pertinent laboratory findings were noted. Our aim is to present awareness through this unique case of severe human leptospirosis (Weil's disease) here. Patient is 31 y old male (with occupation history) working in New Delhi, came to hometown Kolkata, India with history of drowsiness (Glasgow Coma Scale, GCS 7), scanty urine (anuria) and respiratory distress. During admission, on examination, the GCS (E2V2M3), heart rate (126/min), blood pressure (78/44 mm of Hg), respiratory rate (38/min), oxygen saturation (SpO2 :86%) capillary blood glucose (CBG; 132 mg/dl) were noted. Apart from clinical examinations, blood/serum reports show hyperbilirubinemia. Urea, creatinine and potassium were very high, indicating acute renal failure. Pulmonary hemorrhage, haematemesis (blood vomit) and melena (gastrointestinal, GI hemorrhage, blood in stool), coagulopathy was noticed after admission. Hemoglobin dropped minimally to 5.7g/dl during hospital stay. All the clinical presentations and biochemical reports revealed high bilirubin, urea, potassium and creatinine; pulmonary hemorrhage, GI bleeding and coagulopathy. Positive *Leptospira* antibody (IgM) detected in ELISA. All these tests made a clear diagnosis of severe form of Leptospirosis or Weil's disease in this patient.

Keywords: *Anuria; Coagulopathy; Hyperbilirubinemia; Leptospirosis (Weil's Disease); Pulmonary Hemorrhage; Zoonosis*

Introduction

Leptospira species are Spirochaetes belonging to the Order Spirochaetalis and the family Leptospiraceae. Total 22 *Leptospira* species with 7 non-pathogenic, 5 intermediate and 10 pathogenic species are described based on virulence and phylogenetic analysis. *Leptospira* species are divided into serovars according to their antigenic composition. More than 250 pathogenic serovars make up the 26 serogroups (Papadakis et al., 2025).

Leptospirosis occurs most commonly in the tropical and sub-tropical regions because of favorable climate, high rainfall and poor hygienic conditions. Tropical climate favors the pathogen distribution and

Received: 12thMarch 2026; Revised version received on: 30th April 2026; Accepted: 10th May 2026

survival. Most incidences peak during the summer and reported cases occurred mostly in males. According to WHO reports, globally the incidence ranges from approximately 0.1–10 per 100 000 per year with almost 60,000 deaths. In high-exposure risk groups and during outbreaks, disease incidence may reach over 50 per 100, 000. Only 100-150 cases are reported annually, according to the Centers for Disease Control and Prevention (CDC) report (Torgerson et al., 2015; WHO, 2009; CDC, 2025).

As a major direct zoonotic disease, leptospirosis affects almost all mammalian species. Rodents (especially rats) are the most important reservoir (Torgerson et al., 2015; Samrot et al., 2021). Human to human transmission is rare. The vast majority of infections with *Leptospira* cause no or only mild disease in humans. A small percentage of infection (~1%) lead to severe potentially fatal complications (Haake & Levett, 2015).

Leptospirosis may be asymptomatic, mild, moderate and severe. The disease usually present with flu-like illness, fever, chills, headache, nausea, vomiting, abdominal pain, conjunctival redness, myalgia, photophobia with altered mental status, acute renal failure, respiratory insufficiency, hypotension, arrhythmia, severe bleeding and complications like pulmonary hemorrhage, hematemesis, melena, significant hematuria, hemoptysis, and skin manifestations (like ecchymosis, petechiae, jaundice and bleeding from venipuncture sites)(Prinsen et al., 2023; Muñoz-Zanzi et al., 2025).

Weil's disease is the severe form of Leptospirosis (5%-10% of Leptospirosis cases), characterized by hemorrhage, jaundice and acute kidney injury (AKI), progressive multisystem failure, and is associated with 5%-15% mortality rate. It is often characterized by fever, marked jaundice, high serum aminotransferase and bilirubin levels (Samrot et al., 2021).It is listed as a "rare disease" by the Office of Rare Diseases of the National Institutes of Health of America (Bracho et al., 2010; Chakrabarti et al., 2014).Through this case presentation, we want to create awareness for this disease.

Case Description

31 y old male patient, non-alcoholic, working in New Delhi (as slaughterhouse worker for the past 10 years with poor sanitation and hygiene) came to Kolkata, his hometown prior to presentation. He suddenly deteriorated and was admitted within 12 h in Fleming Hospital, Kolkata in August, 2025. His occupation history raised concerns. His family members complained that he had fever for last 8 days and he was only taking oral anti-pyretics without relief of symptoms. He was not previously hospitalized. The patient's family history is unknown. He was admitted with altered mental status (drowsy and sometimes restless), anuria, not responding to verbal comment, only some movements observed on painful stimulation (Glasgow Coma Scale, GCS: E2V2M3). He had no significant past medical history or any comorbidities.

On his initial physical examination, he had palor (++), cervical lymphadenopathy, pedal edema, midline trachea, abdominal distention with hepatomegaly (4 cm below right costal margin at right mid clavicular line), spelenomegaly (near umbilicus), rhonchi and crepitations on chest auscultation. At presentation, patients had body temperature of 104.40F, heart rate was 126/min and BP was 78/44 mm of Hg. The patient had gasping respiration, ABGA showed severe metabolic acidosis with hypoxemia. He was immediately intubated and put on mechanical ventilator with pressure regulated volume control mode. Oxygen was given by mask.

After intubation, he received bolus isotonic IV fluid albumin followed by maintenance fluid to fill the vascular compartment and maintain the normal central venous pressure. Central venous line was established. Foley's Catheter was inserted. Patient's urine was deep yellow colored with total volume of 40 ml. The patient continued to be hypotensive and vassopressor (nor adrenaline) infusion started.

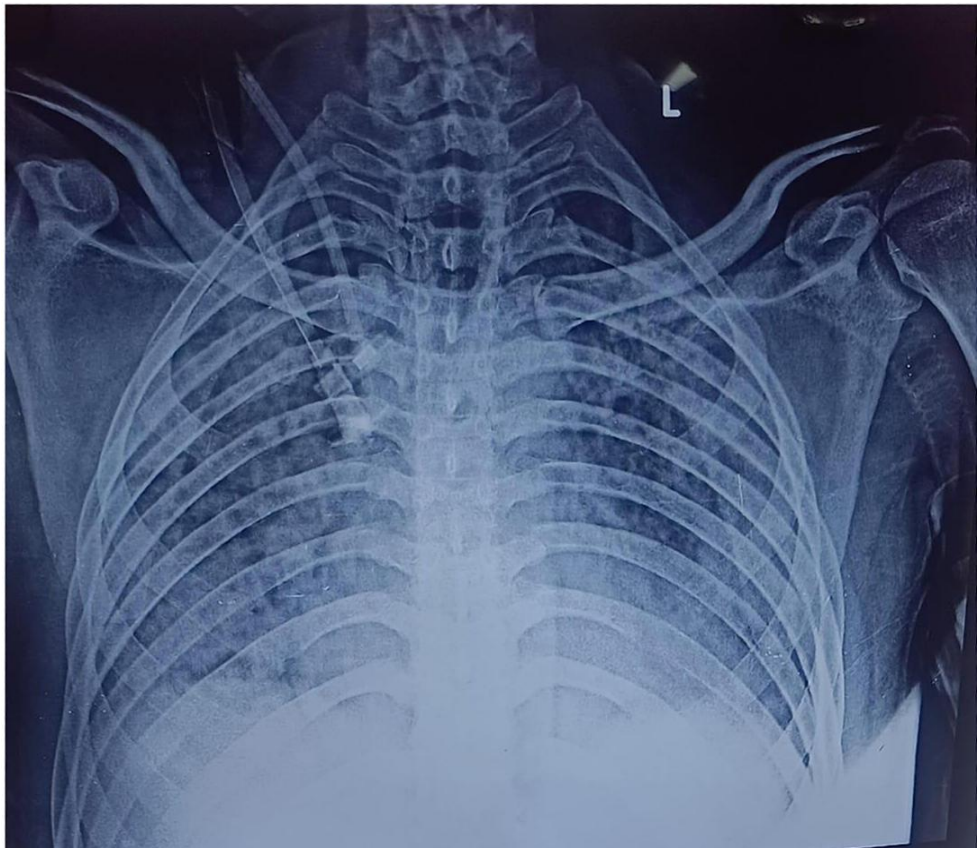

Figure 1: Lung Consolidated Seen in Chest X-Ray of the Patient

Initially nutrition was started by enteral route (naso-gastric tube) but due to intolerance to enteral feeding, parenteral nutrition started after few days. The initial blood/laboratory report was severely deranged. The day-wise comparative chart of laboratory parameters was provided in Table 1. Patients had high WBC count, elevated urea, creatinine and bilirubin values. Right lower zone and left upper zone infective changes noted in X-ray. Coarse thick wall bronchovasculatures were seen in both sides of lungs (Figure 1). Ultrasound of the abdomen showed hepatomegaly, thick-walled gall bladder,empty urinary bladder, edematous kidney suggestive of AKI. During his stay, the patient's serum bilirubin peaks to 42 mg/ dl. His kidney was worsened with peak creatinine of 6.38 mg/ dl. Blood and urine routine cultures showed no growth of any pathogenic bacteria. The patient was *Leptospira* IGM antibody positive in serum ELISA. All tests for dengue, malaria (dual antigen) and typhoid were negative. The patient was diagnosed with Weil's disease. Doxycycline antibiotics for Leptospirosis were started after admission.

In treatment discourse, for AKI, hemodialysis was started and continued several times. For coagulopathy, fresh frozen plasma was transfused. For anemia, several units of packed red cell were transfused. Hypoalbuminemia was corrected by albumin infusion. Hematemesis and melena (GI bleeding) were managed by Proton pump inhibitor infusion. Due to pulmonary hemorrhage endo-tracheal tube was changed 5 times during hospital stay.

Gradually, lung function of patient improved, started weaning trial and he was successfully ex-tubated. Deep vein prophylaxis was given in non-pharmalogical way (stockings and pneumatic compression device). Patients responded well and his clinical status improved. He became conscious, hemodynamically stable, started urine formation, tolerating enteral feeding, so we sequentially withdrew the supports of mechanical ventilator, vasopressors and hemodialysis. Physiotherapy started after 20 days and he started sitting and walking, became stable. After 26 days of hospitalization, he was discharged in good clinical condition. At discharge, patient was advised to be for Thiamine, folate, and vitamin B12 supplementation as he was found to be severely deficient.

Table 1: Day Wise Values of Different Laboratory Parameters of the Patients During His Hospital Stay

Parameters	Day 1	Day 4	Day 16	Day 26
Total Leucocyte count(/dl)	42700	45700	14900	7500
Neutrophils (%)	84	85	87	53
Lymphocytes (%)	11	12	9	42
Hemoglobin (g/dl)	8.6	5.7	10.2	7.6
Platelet count (lac/ mm^3)	1.37	1.75	1.64	2.75
Creatinine (mg/dl)	6.38	4.05	3.81	1.0
Urea(mg/dl)	139.7	178.2	145.2	32.4
Sodium (mEq/L)	126.9	134.7	140.6	138.1
Potassium(mEq/L)	6.31	3.65	2.55	5.02
Total bilirubin(mg/dl)	40.7	39	8.5	1.5
Direct bilirubin (mg/dl)	28.3	31	6.5	0.4
Indirect bilirubin(mg/dl)	12.4	8	2.0	1.1
SGOT (Aspartate transaminase) (U/L)	206	109	40	35
SGPT (Alanine Aminotransferase) (U/L)	115	37	36	32
Alkaline Phosphatase(U/L)	594	528	449	278
Albumin(g/dl)	2.5	2.6	2.52	3.5
Globulin(g/dl)	2.6	2.4	2.4	3.1
INR	1.5	1.5	1.2	1.0
pH	7.2	7.27	7.41	7.5
HCO_3^-(mmol/L)	9.4	23.4	25.1	26.1
Base deficit (mmol/L)	17.11	3.51	0.1	0.1

SGOT: Serum Glutamic-Oxaloacetic Transaminase, SGPT: Serum Glutamic-Pyruvic Transaminase, INR: International Normalized Ratio, HCO3-: bicarbonate ions

Discussion

Leptospirosis is an underreported zoonotic spirochaetal (aerobic *Leptospira* sp) disease highly prevalent in temperate and tropical regions. Weil's disease, an aggressive disease (a severe icteric form) accounts for 5%-10% of all leptospirosis cases with characteristic fever, renal failure, respiratory distress, jaundice and hemorrhage. Pathogens are transmitted indirectly or directly to humans from animals (rodents, dogs etc) or by inoculation with water or food. Mammals are the most common reservoirs. The common source of infection for humans was in indirect or direct contact with infected animals shedding *Leptospira* sp in their urine.

Our patient could possibly be infected from his contact with urine or mice (or dog) contaminated food or water in his workplace with poor sanitation condition. In the second phase of this disease, serum bilirubin rarely approaches 30 mg/ dl. Our patient liver function tests showed hyperbilirubinemia (much above 30 mg/dl, Table 1). High bilirubin level is due to hepatocellular damage (evident from liver function tests) and leakage of bilirubin from bile ducts. The main factors for renal failure are high bilirubin and dehydration. The patient was given hemodialysis due to his anuric condition and high creatinine level. He showed improvement in his liver function test at the time of discharge. Hyperbilirubinemia and hypoalbuminemia were due to liver failure. AKI was represented by anuria with high urea level. Severe metabolic acidosis was also successfully treated by hemodialysis. Anemic condition was resolved during the hospital stay.

The diagnosis of leptospirosis is made via direct diagnostic methods, serological tests, culture methods and PCR. In our patient, we tested Leptospirosis positive via IgM antibody titers for screening and confirmation. Molecular tests like PCR are not widely available in most hospitals. Doxycycline is effective (and cheap) when treated within few days of illness. Our patient responded well with doxycycline (Samrot et al., 2021).

A unique aspect of our case report is that the patient's total serum bilirubin level spiked much higher than 30 mg/ dl. This is very rare report; mostly mild to moderate rise of bilirubin is reported before (Puca et al., 2020; Haake and Levett, 2015).

This report emphasized timely diagnosis, management, intervention and treatment in intensive care. There is no standard care for this disease, detailed patient history and patient-centric treatment is the hallmark.

Conclusion

We present a unique case of Weil's disease, a severe form of leptospirosis. Misdiagnosis and confusion with other diseases are very common in Leptospirosis. A detailed case history is needed when patients come from poor sanitary background like our patients. It is known that those who live in poor sanitary conditions are at risk for leptospirosis. It is always necessary to avoid fatal diseases like Weil's disease. When clinical suspicion for leptospirosis is high, early antibiotic administration is lifesaving and should be initiated empirically.

CRediT Authorship Contribution Statement

S.H.H: Supervision, drafting of manuscript. M.S.A: Supervision. W.A: Concept and design, drafting of manuscript.

AI Assistance Declaration

The author hereby declares that, during the preparation of this manuscript, generative AI tools such as ChatGPT, Microsoft Copilot, and Google Gemini were utilized to assist with language enhancement and grammar correction. Following the use of these tools, the author thoroughly reviewed and revised the content and takes full responsibility for the final version of the manuscript, ensuring its accuracy and adherence to the required academic standards.

Conflict of Interest

The authors declare that they have no conflicts of interest.

Acknowledgement

The authors would like to express their sincere gratitude to all authors and their institutions who contributed to work.

References

Bracho, G., Varela, E., Fernández, R., Ordaz, B., Marzoa, N., Menéndez, J., ... & Campa, C. (2010). Large-scale application of highly-diluted bacteria for Leptospirosis epidemic control. *Homeopathy, 99*(03), 156-166. https://doi.org/10.1016/j.homp.2010.05.009

Centers for Disease Control and Prevention (CDC) (2025). *About Leptospirosis.* https://www.cdc.gov/leptospirosis/about/index.html

Chakrabarti, A., Nandy, M., Pal, D., & Mallik, S. (2014). A rare case of Weil's disease with alveolar haemorrhage. *Asian Pacific Journal of Tropical Biomedicine, 4*, S66-S69. https://doi.org/10.12980/APJTB.4.2014D126

Haake, D. A., & Levett, P. N. (2014). Leptospirosis in humans. *Leptospira and leptospirosis*, 65-97. https://doi.org/10.1007/978-3-662-45059-8_5

Muñoz-Zanzi, C., Dreyfus, A., Limothai, U., Foley, W., Srisawat, N., Picardeau, M., & Haake, D. A. (2025, February). *Leptospirosis—Improving healthcare outcomes for a neglected tropical disease. In Open Forum Infectious Diseases* (Vol. 12, No. 2, p. ofaf035). US: Oxford University Press. https://doi.org/10.1093/ofid/ofaf035

Papadakis, M.A., Rabow, M W., McQuaid, K. R., Gandhi, M (2025). *Current Medical Diagnosis & Treatment. McGraw Hill* (64th edns). ISBN-13. 978-1266266232. https://accessmedicine.mhmedical.com/book.aspx?bookid=3495

Prinsen, G., Baker, M., Benschop, J., Collins-Emerson, J., Douwes, J., Fayaz, A., ... & Yeung, P. (2023). "We don't really do doctors." messages from people diagnosed with occupational leptospirosis

for medical professionals on infection, hospitalisation, and long-term effects. *Heliyon, 9*(9). https://doi.org/10.1016/j.heliyon.2023.e19303

Puca, E., Abazaj, E., Pipero, P., Harxhi, A., Ferizaj, R., Como, N., & Puca, E. (2020). A case with high bilirubinemia and hemolytic anemia during leptospirosis and a short review of similar cases. *Caspian Journal of Internal Medicine, 11*(4), 441. https://doi.org/10.22088/cjim.11.4.441

Samrot, A. V., Sean, T. C., Bhavya, K. S., Sahithya, C. S., Chan-Drasekaran, S., Palanisamy, R., ... & Mok, P. L. (2021). Leptospiral infection, pathogenesis and its diagnosis—a review. *Pathogens, 10*(2), 145. https://doi.org/10.3390/pathogens10020145

Torgerson, P. R., Hagan, J. E., Costa, F., Calcagno, J., Kane, M., Martinez-Silveira, M. S., ... & Abela-Ridder, B. (2015). Global burden of leptospirosis: estimated in terms of disability adjusted life years. *PLoS Neglected Tropical Diseases, 9*(10), e0004122. https://doi.org/10.1371/journal.pntd.0004122

World Health Organization (WHO) (2009). *Leptospirosis: Fact sheets.* https://www.who.int/publications/i/item/B4221