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Case Study

Severe Leptospirosis with Suspected Disseminated Intravascular Coagulation, Hepatorenal Dysfunction, and Severe Thrombocytopenia: A Case Report

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Severe leptospirosis is an uncommon but potentially fatal infectious disease that may present with multiorgan involvement. The coexistence of disseminated intravascular coagulation (DIC), hepatorenal dysfunction, and severe thrombocytopenia represents a complex and clinically challenging presentation. A 43-year-old male school teacher with no previously known comorbidities presented with a one-week history of fever, one day of vomiting and generalized weakness, dark-coloured urine for 2 days, and five episodes of loose, blackish stool. Peripheral smear study showed a normocytic normochromic blood picture with neutrophilia and severe thrombocytopenia, deranged liver and renal function, and coagulation abnormalities consistent with clinically suspected DIC. Leptospira IgM ELISA was positive (1.77), while dengue and malaria were excluded. Abdominal ultrasonography showed mild hepatomegaly and mild bilateral renomegaly. Multidisciplinary management by intensive care, nephrology, gastroenterology, and hematology teams included intravenous antibiotics, vitamin K, corticosteroids, transfusion of 4 units of cryoprecipitate and 14 units of platelets, and supportive care. The patient improved clinically, with marked recovery of platelet counts and improvement in liver and renal parameters, and was discharged in stable condition on hepatoprotective medication. Severe leptospirosis can present with life-threatening multi-organ dysfunction, including clinically suspected DIC. Early clinical suspicion, serological confirmation, and prompt multidisciplinary supportive care are essential for a favorable outcome.

Keywords: Leptospirosis; Weil's disease; Disseminated intravascular coagulation; Thrombocytopenia; Hepatorenal dysfunction; Case report.

INTRODUCTION

Leptospirosis is a globally distributed zoonotic spirochetal infection caused by pathogenic Leptospira species, transmitted to humans through direct or indirect contact with the urine of infected animal reservoirs, most commonly rodents [1]. The disease is endemic in tropical and subtropical regions, particularly in coastal and monsoon-affected areas, where flooding and occupational exposure to contaminated water facilitate transmission [1,2]. Clinical presentation is highly variable, ranging from a self-limiting febrile illness to severe, life-threatening multi-organ dysfunction [1,2]. Approximately 5–10% of symptomatic cases progress to the severe icteric form known as Weil's disease, characterized by the triad of jaundice, acute kidney injury, and hemorrhagic manifestations, with reported mortality of 5–15% [1,3]. Coagulopathy, including disseminated intravascular coagulation (DIC), is an under-recognized but potentially fatal complication of severe leptospirosis whose pathophysiology remains incompletely understood [4,5]. We report a case of severe leptospirosis, clinically suspected DIC, hepatorenal dysfunction, and thrombocytopenia, a combination that raises important diagnostic and therapeutic considerations.

CASE PRESENTATION

A 43-year-old male school teacher with no known comorbidities presented to the emergency department with a one-week history of fever, associated with one day of vomiting. He also reported severe generalized weakness, dark-coloured urine for 2 days, and five episodes of loose, blackish stools. He had been evaluated at an outside facility and was referred for further management. There was no history of similar illness in the past.On examination at presentation, the patient was conscious, oriented, and hemodynamically stable. He was afebrile at the time of triage. A working diagnosis of sepsis due to severe leptospirosis with severe thrombocytopenia, coagulopathy with suspected DIC, and hepatorenal dysfunction was made. Although D-dimer and fibrinogen levels were not available, the presence of severe thrombocytopenia, prolonged coagulation parameters, and active gastrointestinal bleeding in the setting of severe leptospirosis raised clinical suspicion of disseminated intravascular coagulation (DIC). Therefore, DIC was considered clinically suspected rather than laboratory-confirmed. The patient was managed jointly by intensive care, nephrology, gastroenterology, and hematology teams.

Table 1. Serial Laboratory Investigations During Hospitalization

Parameter	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Hemoglobin (g/dL)	12.9	11.9	10.4	10.7	11.7	11.9	10.7
Total WBC (×10³/µL)	12.28	14.05	13.66	15.34	21.58	31.31	35.50
Platelet count (×10³/µL)	8.0	10.0	13.0	11.0	17.0	52.0	132.0
PT (sec)	16.6	17.3	12.6	29.8	12.5	ND	ND
INR	1.22	1.28	0.90	2.76	1.09	ND	ND
aPTT (sec)	55.0	36.9	ND	35.3	ND	ND	ND
Serum Creatinine (mg/dL)	3.3	5.1	6.6	7.4	7.2	6.3	4.9
Total Bilirubin (mg/dL)	10.1	16.9	24.5	56.6	41.5	31.2	14.5
Direct Bilirubin (mg/dL)	7.2	12.3	19.1	34.6	39.1	26.9	9.5
AST (U/L)	178	173	140	35	93	97	40
ALT (U/L)	109	120	100	35	105	105	82

Table 2. Important Baseline Diagnostic Investigations

Investigation	Result / Interpretation
C-reactive protein (CRP)	206.4 mg/L (markedly elevated)
Ferritin	873.4 ng/mL (markedly elevated)
Leptospira IgM ELISA	Positive (index 1.77)
Dengue NS1 / IgM / IgG	Non-reactive
Peripheral smear for malarial parasite	Negative
Stool occult blood	Positive
Peripheral blood smear	Normocytic normochromic red cell picture with neutrophilia and severe thrombocytopenia
Blood culture	No growth after incubation
Urine culture	Heavy growth of mixed bacterial flora
Ultrasonography (Abdomen)	Mild hepatomegaly, possible right renal, mild bilateral renomegaly
Transthoracic echocardiography	Normal study

Treatment Given

The patient was managed with a multidisciplinary approach, including intravenous broad-spectrum antibiotics with piperacillin-tazobactam 2.25 g thrice a day, which were subsequently de-escalated to oral doxycycline 100 mg twice daily following clinical stabilization and positive Leptospira IgM ELISA [1,3]. Supportive management comprised intravenous fluid resuscitation, intravenous vitamin K 10 mg once daily, IV pantoprazole 40 mg once a day, and oral ursodeoxycholic acid 300 mg twice daily for cholestatic liver dysfunction. Owing to profound thrombocytopenia complicated by active

gastrointestinal bleeding and coagulation abnormalities suggestive of DIC, the patient received IV dexamethasone 4 mg thrice a day under hematology guidance [4,5], in addition to transfusion support with 4 units of cryoprecipitate and 14 units of platelets.

Table 3. Clinical Course and Timeline of Key Parameters Over the seven-Day Admission

Day of admission	Platelet count ($\times 10^3/\mu L$)	Serum creatinine (mg/dL)	Remarks
Day 1	8	3.3	Admission; Leptospira IgM positive
Day 2	10	5.1	Continued supportive care
Day 3	13	6.6	Continued supportive care
Day 4	11	7.4 (peak)	Stool occult blood positive; active GI bleed
Day 5	17	7.2	Transfusion of 14U platelets and 4U cryoprecipitate
Day 6	52	6.3	Hemorrhage controlled; platelet rebound
Day 7	132	4.9	Marked clinical recovery; no further bleeding

Over the course of admission, the patient remained hemodynamically stable, urine output remained adequate, and no further overt bleeding manifestations occurred after the initial presentation. Platelet counts recovered progressively, and renal function improved. At discharge, the patient was prescribed oral doxycycline 100 mg twice daily to complete the antileptospiral course, pantoprazole 40 mg once daily, a multivitamin supplement, ursodeoxycholic acid 300 mg, ademethionine 400 mg, and antifungal mouth paint for oral candidiasis noted during admission. He was advised to maintain adequate hydration and attend a follow-up visit in the outpatient department after 12 weeks.

DISCUSSION

This case illustrates several features that make severe leptospirosis a diagnostically and therapeutically challenging condition. First, the initial presentation with fever, gastrointestinal symptoms, and dark urine was nonspecific and could have been mistaken for several other tropical febrile illnesses, including dengue, malaria, enteric fever, or viral hepatitis [1,2]. Systematic exclusion of these differentials, alongside a high index of suspicion based on epidemiological context (a coastal, monsoon-prone region), was essential to reaching the correct diagnosis [2,3].

Second, the combination of severe thrombocytopenia, prolonged coagulation parameters, and gastrointestinal bleeding in this patient raised concern for disseminated intravascular coagulation (DIC). However, D-dimer and fibrinogen levels were not available; therefore, DIC could not be confirmed using the complete laboratory criteria. The diagnosis was considered clinically suspected based on the available clinical and laboratory findings. Management of clinically suspected DIC requires treatment of the underlying infection and appropriate blood component support guided by active bleeding and coagulation parameters, alongside prompt antimicrobial therapy [4,5].

Third, the Leptospira IgM ELISA was positive (1.77). These serological findings, together with the clinical presentation and laboratory abnormalities, supported the diagnosis of severe leptospirosis [1,3]. Serological results should be interpreted together with the clinical presentation and timing of illness [1,3].

Fourth, the hepatorenal involvement observed in this patient is consistent with the classical description of Weil's disease, in which Leptospira-mediated renal tubular injury and centrilobular hepatic injury coexist [1,3]. Disproportionately elevated bilirubin relative to modest transaminase elevation was noted. Renal recovery in leptospirosis-associated acute kidney injury is typical with supportive care [1,3].

CONCLUSION

Severe leptospirosis should be considered in the differential diagnosis of undifferentiated febrile illness with thrombocytopenia and hepatorenal dysfunction, particularly in endemic regions, even

when initial serology is only weakly reactive. Clinicians should be aware that severe leptospirosis can be complicated by coagulopathy. A multidisciplinary approach combining early antimicrobial therapy, appropriate blood component support and organ-specific management resulted in a favorable outcome, highlighting the value of coordinated care in severe tropical infectious disease.

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