PEDIATRIC ONCALL
CHILD HEALTH CARE

CASE REPORTS

LEPTOSPIROSIS PRESENTING WITH AUTOIMMUNE HEMOLYTIC ANEMIA AND ACUTE KIDNEY INJURY

Lam Le Vu Tung[1], Bay Huu Luong[2], Huong Nguyen Thu[2].

[1]VinUniversity, Hanoi, Vietnam,
[2]Department of Pediatric Nephrology, National Children's Hospital, Hanoi, Vietnam.

ABSTRACT

Leptospirosis is an infectious disease caused by pathogenic spirochetes from the Leptospira genus, which often leads to a wide range of clinical symptoms. These can vary from flu-like symptoms to more severe manifestations like multi-organ failure. While mild cases often resolve without complications, severe leptospirosis involves the failure of several organs, such as the liver, kidneys, lungs, heart and brain. The combination of jaundice and kidney failure, referred to as Weil's disease iss one of the most well-known forms of the illness. The presence of organ dysfunction signals a more progressed stage of the infection, which can emerge rapidly and is often found in many patients upon initial diagnosis.[1]

This report highlights autoimmune hemolytic anemia (AIHA) is a rare complication of leptospirosis in children, with few cases reported in the literature. This report presents an uncommon pediatric case of Weil's disease complicated by AIHA and extreme hyperbilirubinemia, adding to the limited evidence on atypical manifestations in endemic regions and highlighting diagnostic challenges.

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Case Presentation

A 14-year-old girl was admitted with a 5-day history of intermittent fever (without documented temperature), diarrhea, vomiting, and abdominal pain, for which she had received no specific treatment. On the third day of illness, she developed rapidly progressive jaundice and scleral icterus. Her urine also became progressively darker and less frequent. She had no significant past medical history and no known exposure to contaminated water or food.

At admission, her heart rate was 125 beats/min, blood pressure was 100/50 mmHg, temperature was 39°C (102°F), and oxygen saturation was 98% on room air. Physical examination showed jaundice, scleral icterus, pallor, fatigue, and dehydration. There was no hepatosplenomegaly or respiratory distress. She was anuric. Mild peripheral edema was present in the hands and feet, without periorbital edema. Abdominal examination was unremarkable. She passed loose yellow stools without blood. The remainder of the examination was unremarkable.

Initial laboratory evaluation showed elevated inflammatory markers, anemia, renal dysfunction, and hyperbilirubinemia (Table 1). The anemia was severe and gradually progressive. Peripheral blood smear demonstrated spherocytes, fragmented red blood cells, and evidence of hemolysis. Lactate dehydrogenase (LDH) was elevated, and the direct Coombs test was positive, supporting a diagnosis of autoimmune hemolytic anemia. Drug-induced hemolysis was excluded because she was not receiving any potentially causative medications.

Renal function was markedly impaired, with elevated urea and creatinine levels and an estimated glomerular filtration rate (eGFR) of 7.3 mL/min/1.73 m² calculated using the Schwartz formula. Comprehensive etiological investigations identified a markedly elevated Leptospira IgM level (>100 U/L). Other potential causes, including viral hepatitis, rickettsial infection, and mycoplasma infection, were negative. Additional investigations for autoimmune and other hemolytic causes were also negative, including normal C3 and C4 levels, negative autoimmune antibodies, and normal G6PD, pyruvate kinase, and hemoglobin electrophoresis results.

The patient was immediately started on meropenem and doxycycline when she was admitted to hospital. Hemodialysis was initiated to manage the AKI, and a packed red blood cell transfusion was given to address the severe anemia (see in table 2). During her first day, jaundice persisted, and she remained anuric. Following dialysis, the patient's urine output gradually improved to 1700 mL/24 hours by the fourth day of hospitalization.

The patient's clinical condition improved in the next two weeks. Her bilirubin, creatinine, and hemoglobin levels, began to normalize. By day 17, total bilirubin had decreased to 63.7 µmol/L, and hemoglobin had risen to 82 g/L. Continued monitoring showed stabilization of her renal function, with creatinine levels of 43 µmol/L on day 22. Despite significant clinical improvement, the patient remained mildly jaundiced at discharge. Bilirubin levels were expected to normalize within the next few weeks. A repeat bone marrow biopsy on day 25 demonstrated normal trilineage hematopoiesis. At a three-week follow-up visit, the patient had fully recovered clinically, with normal renal function and no residual anemia.

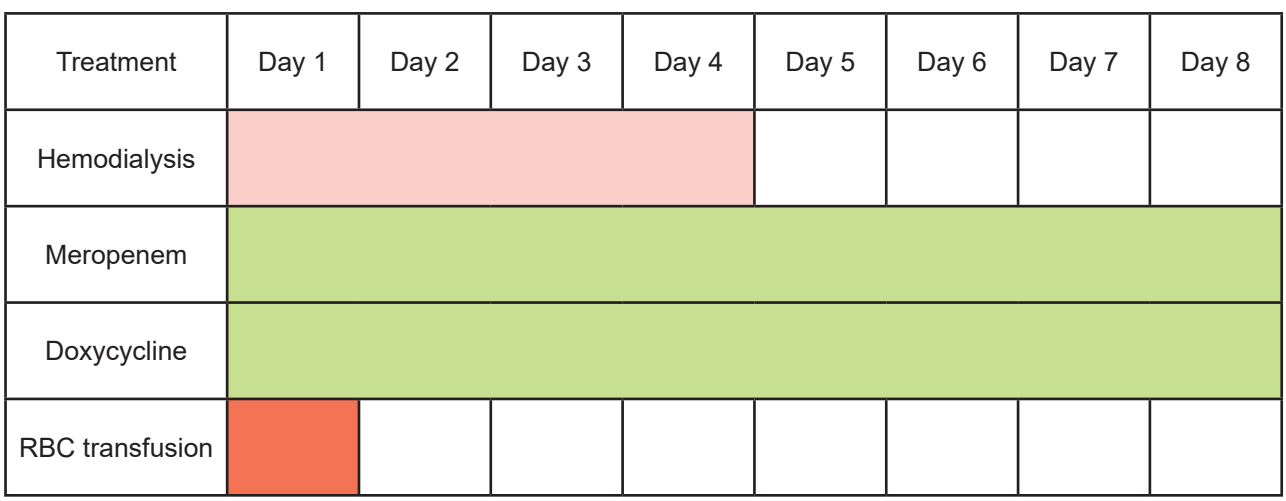
Address for Correspondance: Lam Le Vu Tung, Department of General Pediatrics, VinUniversity, Hanoi, Vietnam.
Email: vutunglamflbt@gmail.com

Table 1. Laboratory investigations

Investigation	On admission	4th day	7th day	8th day	17th day	On discharge
WBC (x109/L)	20.3	20.3	24.6	5.6	4.4	-
Neutrophil (%)	88	82.9	65.8	2.8	2.4	-
Lymphocytes (%)	4	6.6	10.7	2.2	1.6	-
RBC (x1012/ L)	3.6	4.3	3.9	3.9	4.1	
Hemoglobin (g/L)	56	83	78	68	84	-
MCV (fL)	50.8	61.8	56.2	-	67.1	-
MCHC	368	308	353			
Platelets (x109/L)	85	126	222	-	355	-
Serum urea (mmol/L)	54.7	19.7	11.1	7.9	5.6	4.4
Serum creatinine (µmol/L)	958.9	174.7	140	44.9	40.7	43
Serum albumin (g/L)	25.6	23.8	28.9	-	36.2	34.5
Total bilirubin (µmol/L)	483	335	162.5	-	63.7	44.4
Direct bilirubin (µmol/L)	276	199	127	-	28	18.4
GOT (U/L)	63	31.7	-	-		21.7
GPT (U/L)	58	39.9	-	-		15.5
CRP (mg/L)	73	26.8	14.5	-	0.4	5.5
LDH (U/L)	546	355.4	-	-	-	-
Direct Coombs Test	2+				+/-	
Leptospira IgM (U/L)	Positive >100					

WBC, White blood cell; RBC, Red blood cell; GOT, Glutamic oxaloacetic transaminase; GPT, Glutamic pyruvic transaminase; CRP, C-reactive protein, LDH, Lactate dehydrogenase.

Table 2. Management



Discussion

Leptospirosis is a disease caused by Leptospira, with Leptospira interrogans being the most common cause of Weil syndrome in humans.[2] The clinical presentation of leptospirosis ranges from mild, asymptomatic illness to severe Weil syndrome, which involves bleeding, kidney failure, and liver failure. Rare manifestations include rhabdomyolysis, pulmonary hemorrhage, myocarditis, thrombotic microangiopathy.[2,3]

In our case, the patient presented with severe jaundice, anemia, and acute kidney injury, which initially raised concern for other hemolytic disorders. Autoimmune hemolytic anemia (AIHA) is an uncommon but recognized complication of leptospirosis. AIHA associated with leptospirosis has been well-documented in animals[4], but it is rare in humans, with only a few cases reported in the medical literature.[5] Initially, it was believed that anemia in leptospirosis was primarily caused by suppressed erythropoiesis. However, studies involving bone marrow biopsies have shown that erythropoiesis is actually moderately activated, indicating a hyperproliferative state of the bone marrow rather than impaired function. These findings suggest that anemia in the acute phase is mostly due to hemolysis, whereas anemia in later stages is often linked to chronic renal failure.[6] In the case presented,

there are some unusual presentations that have to be taken into consideration. The first one was Jaundice which is a typical feature of Weil's disease, but such extreme bilirubin levels are rarely documented. A search on PubMed revealed only four publications reporting bilirubin levels higher than 30 mg/dL. Pothuri et al. and Covic et al. each documented a case with bilirubin exceeding 30 mg/dL.[7,8] Sing et al. described a case involving co-infection of hepatitis E virus (HEV) and leptospirosis, while Legris et al. reported a case involving both leptospirosis and amebiasis, with bilirubin levels over 29. 23 mg/dL.[9] Although jaundice is a key sign of liver dysfunction, its underlying mechanism in leptospirosis remains unclear. It is thought that hepatocellular damage and disruption of hepatocyte junctions lead to bile leakage into the bloodstream, causing elevated direct bilirubin levels.[10] In this case, however, liver enzymes were not significantly elevated, suggesting that hyperbilirubinemia might have been due to bile duct obstruction.

Additionally, the patient had severe anemia, which is atypical for early stages of leptospirosis. While thrombocytopenia and slight reductions in hematocrit and hemoglobin are common in the initial phase, the patient's anemia could not be solely explained by some bruise in the skin. Laboratory findings, including the presence of spherocytes, elevated lactate dehydrogenase (LDH), and a positive direct Coombs test, indicated that the anemia was immune-mediated. Hemolytic anemia is a rare complication of Weil's disease, and its mechanisms are not fully understood. Solmazgul et al. described a case of severe acute hemolytic anemia related to leptospirosis, while other researchers have suggested that hemolysins, specifically with phospholipase activity, may be responsible for such cases.[5] The role of leptospira hemolysins in the disease process is still not entirely clear.

One limitation of this case was the lack of specific diagnostic tests for leptospirosis, such as microscopic agglutination test or PCR instead of ELISA conducted.

In conclusion, leptospirosis can manifest with diverse clinical features. Clinicians should maintain a high index of suspicion for leptospirosis in patients presenting with jaundice, anemia, and acute kidney injury, particularly in endemic regions.

Compliance with Ethical Standards
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