

A case report on leptospirosis with pseudohepatorenal syndrome

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ABSTRACT

The spirochete *Leptospira interrogans* causes leptospirosis, a zoonosis. There are numerous clinical symptoms of leptospirosis. Clinical manifestations might range from a subclinical infection with a mild fever to severe clinical symptoms with jaundice and renal failure. Here, a case of leptospirosis with acute renal failure, thrombocytopenia, and hyperbilirubinemia is presented. A 51-year-old man presented with fever, body aches, muscle aches, colored urine, and jaundice for 2 days. On serology report, *L. interrogans* AB IGM-CARD was weekly positive. In the Renal function test, urea and creatinine values were high as well as the liver function test was abnormal. The patient was given 1 g of ceftriaxone intravenously and 100 mg of doxycycline orally. Hepatorenal treatment was supportive and the outcome was positive. It may have been overlooked when the diagnosis of leptospirosis was confirmed.

Key words: AB IGM-CARD, Acute renal failure, Hyperbilirubinemia, Leptospirosis, Thrombocytopenia, Zoonosis

The bacterium *Leptospira interrogans* belonging to the genus *L. interrogans* causes leptospirosis. Previously, leptospirosis was considered an under-reported disease but now, it has emerged as the most prevalent zoonosis globally. This zoonotic disease is more pervasive in tropical regions of the world when compared to temperate regions [1]. According to the Centers for Disease Control and Prevention, the worldwide approximate annual occurrence of leptospirosis is >1 million, with around 59,000 deaths. Epidemics of zoonotic disease are known particularly in disasters and extreme weather. The regions with the highest rates of leptospirosis are South and Southeast Asia [2]. Leptospirosis has a clinical manifestation that is similar to that of common tropical illnesses such as dengue fever, scrub typhus, viral hepatitis, and malaria [3]. The illness can manifest itself in a variety of clinical symptoms, leading to diagnostic confusion and further aggravating the clinical situation. The disease can be spread by many kinds of animals but the rat is considered as the biggest vector of the bacterium. When humans come into touch with contaminated water, soil, urine, or animal tissue infected with *L. interrogans*, they become infected. This disease is most common in people who have their occupations more oriented toward animals, soil, and water like farmers and paddy workers [4,5].

There has been an exponential growth in leptospirosis cases in recent years, particularly during the rainy season. The primary goal of this report is to present a case of leptospirosis with pseudo-hepatorenal syndrome and to emphasize the relevance of incorporating this disease in the differential diagnosis of more prevalent diseases.

CASE REPORT

A 51-year-old patient, a previously healthy man, a farmer by profession, was admitted to the hospital with complaints of generalized body ache, high-grade fever (39.4°C), myalgia for the past week, pain for 1 week in thigh and calf muscles, and yellowish urine for the past 3 days. He has no other comorbidities.

The patient was hospitalized and an investigation showed thrombocytopenia, acute kidney injury (AKI), and hyperbilirubinemia. His physical examination shows that the patient was febrile and icteric.

On chest X-ray, it shows minimal bilateral infiltrate presence, his blood pressure was 110/80 mmHg, and his heart rate was 90 beats/min. Serology report shows that *L. interrogans* AB IgM card was weekly positive. The initial laboratory examination showed a progressive decline in renal function. On the initial day, creatinine was 5.1 mg/dl then after it elevated to 7.2 mg/dl, day by day the patient's creatinine increased. Initially, the urea

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level was 114 mg/dl, after 5 days, it elevated to 267 mg/dl. The patient also developed thrombocytopenia ($25,000 \text{ cells} \times 10^9/\text{L}$), hyponatremia (127 mmol/L), and hypokalemia (2.43 mmol/L). The liver function test (LFT) was abnormal, serum glutamic-oxaloacetic transaminase was 199 IU/L, serum glutamic pyruvic transaminase was 91 IU/L, total bilirubin was 25.1 mg/dl, direct bilirubin 26.08, mg/dl, total protein 5.8, and albumin was 2.9 g/dl. The daily blood pressure and pulse rate were normal. The temperature was 102.9 F. Urine analysis shows the presence of albumin and a trace amount of glucose.

On the 2nd day, the patient had three episodes of loose stools and periabdominal examination shows mild epigastric tenderness, day-by-day, patient had decreased urine output.

The patient was shifted to the high dependency unit ward and his blood pressure was elevated to 180/100 mmHg. After elevation of blood pressure, the patient's troponin I HS and creatinine kinase levels were monitored. Troponin I HS (286.9 pg/ml) and creatinine kinase (5445 u/l) levels were extremely elevated (Table 1).

During the 14 days of hospital stay, the patient was treated for leptospirosis with the pseudohepatorenal syndrome with inj. ceftriaxone 1 g IV BD, tablet doxycycline 100 mg BD, and inj. pantoprazole 40 mg. The patient's potassium level was low, so the patient was treated with IVF, normal saline 0.9% 500 ml iv bolous with KCL 20 mEq @100ML/HR. For metabolic acidosis, the patient was treated with tablet sodium bicarbonate for 3 days, along with renal and liver-protective medications. Elevated blood pressure was managed with T. amlodipine 5 mg OD. After the therapy, the patient improved symptomatically. Glomerular filtration rate and hyperbilirubinemia showing an improving trend, hence, discharged to continue to follow-up in outpatient department with LFT/Serum creatinine/complete blood count (CBC) report.

Table 1: Laboratory findings during case management

Test	1 st time	2 nd time (after 7 days)	Reference
Hemoglobin (g/dl)	13	12.4	12.9–15.9
Platelet (cells $\times 10^9/\text{l}$)	25000	35000	155–366
Urea (mg/dl)	267	258	16–48
Creatinine (mg/dl)	7.2	6.9	0.7–1.2
Potassium (mmol/l)	2.43	3.19	3.3–5.1
Sodium (mmol/l)	127	125	136–145
Total bilirubin (mg/dl)	22.1	19.9	1.1
Direct bilirubin (mg/dl)	19	26.12	1.2
SGOT (iu/l)	199	169	0–32
SGPT (iu/l)	91	83	0–32
Total protein (g/dl)	5.8	5.8	6.4–8.3
Albumin (g/dl)	2.9	2.9	3.5–5.2
Troponin I HS (pg/ml)	286.9	286.9	24–30
Creatinine kinase (u/l)	5445	5445	22–198

SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase

DISCUSSION

Leptospirosis is a condition with a varying degree of severity and involvement of organs. The consequences of severe leptospirosis highlight the disease's multisystemic nature [6]. It occurs worldwide, but in tropical regions, it is more common. It is transmitted by wild or domestic animals such as rats, mice, sheep, cattle, pigs, dogs, and goats. The severe form of leptospirosis is linked with renal failure and jaundice. In nearly all severe leptospirosis instances, jaundice with elevated bilirubin concentrations is present and leads to severe AKI [7]. Leptospirosis can manifest itself in two stages. Symptoms of fever, chills, severe headache, nausea, vomiting, myalgia, and diarrhea are manifested in the initial stage. The second stage, also known as Weil's disease, is more severe, with symptoms such as sepsis, multi-organ failure, jaundice, and bleeding that can result in cardiogenic shock and increased mortality [8].

In leptospirosis, renal impairment can vary from prerenal azotemia to severe acute renal failure that necessitates dialysis. The occurrence of acute renal failure is widespread, ranging from 40 to 60% in severe leptospirosis. The inflammatory response is induced in renal proximal tubule cells when Toll-like receptor 2, nuclear factor (κ) B, and mitogen-mediated protein kinases are activated [9]. Hypokalemia also occurs due to tubular dysfunction and the major renal lesion causing renal failure is tubulointerstitial nephritis. Furthermore, severe hypotension is a leading sign of the onset of renal and pulmonary problems. Non-oliguric acute renal injury, hypokalemia, and hyponatremia are common clinical manifestations of leptospirosis [10]. In leptospirosis, tubular dysfunction is due to proximal tubular dysfunction, leading to increased distal sodium and ultimately potassium excretion through the intact distal tubule [10]. Thrombocytopenia can develop in the presence of acute renal failure without the presence of disseminated intravascular coagulation and it is linked to significant leptospirosis endotoxin damage. As a result, thrombocytopenia might be a sign of kidney injury caused by acute leptospirosis.

Leptospirosis is diagnosed by serological methods (microscopic agglutination test) for *L. interrogans* antibody detection, polymerase chain reaction for *L. interrogans* DNA identification, and microbe cultivation. Due to the difficulties of cultivating *L. interrogans*, the microscopic agglutination test, is the most often used diagnostic approach. For quick acute leptospirosis screening, the immunoglobulin M assay has recently been employed [6].

Antimicrobial treatment for the severe forms of leptospirosis is primarily recommended although its use in moderate form is contentious. Antibiotics such as Doxycycline, Amoxicillin, and Ampicillin are used to treat mild leptospirosis. For many years, intravenous penicillin G was the preferred therapy for severe leptospirosis, but third-generation cephalosporins such as Cefotaxime and Ceftriaxone are now widely used. Gomes *et al.* in a recent study uncovered a novel avenue for treating leptospirosis using antibodies. They observed that the administration of

anti-LipL32 mAbs before infection significantly protected animal models and prevented the growth of *L. interrogans in vitro* [11].

In this case, the use of Ceftriaxone and Doxycycline helped to reduce the severity, duration of hospital stay, and provided a better quality of life. Supportive care, close monitoring, and the management of renal, hepatic, hematologic, and central nervous system problems are pivotal in severe instances. Patients should be treated in a supervised environment since their condition can quickly advance to CVS collapse and shock. The function of the kidneys should also be monitored routinely and dialysis should be considered in cases of renal failure. The routine monitoring of CBC, LFT, kidney function test, and initiation of hemodialysis have significantly assisted to reduce the severity of renal failure in this case.

CONCLUSION

Leptospirosis has lately gained worldwide recognition as a globally major re-emerging epidemic disease that affects both developing and developed countries. Leptospirosis can cause a variety of symptoms, including severe renal and liver failure. This zoonotic disease should be regarded as a primary diagnosis in individuals with hepatorenal dysfunction, and empiric treatment should begin before laboratory test confirmation. The current case of fulminant leptospirosis should serve as a cautionary tale to health-care practitioners and the general public about the clinical significance of this potentially lethal disease if left untreated. Early identification of leptospirosis is critical since, in this case, prompt antibiotic treatment reduced the severity of the infection.

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