Case Report

Severe Leptospirosis With non-Oliguric Renal Failure With 'Myocarditis' Mimicking Acute Coronary Syndrome: A Rare Presentation From Northern India

Amit Kumar[1], Majaz Ahmad[2], Desai Vishal Jakshibhai[3]

[1]Senior Resident, Department of Medicine, Pt. BD Sharma Pgims Rohtak, [2]Resident, Department of Medicine, Pgims Rohtak, [3]Resident, Department of Medicine, SGT Hospital, Gurugram.

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Abstract

Leptospirosis is a globally distributed zoonosis with a broad clinical spectrum. Thisthe disease mostly affects liver and kidney tissues. Other organs can be affected by leptospirosis-induced vasculitis. In addition, cardiac manifestations are common, and the presence of transient ECG abnormalities can be found in 50-60 % of the patients(1). Early diagnosis and adequate supportive therapy are crucial for the appropriate management of symptoms. In this article, we present a case of leptospirosis with myocarditis with hypokalemia and non-oliguric acute renal failure. All clinical findings gradually regressed after treatment.

Keywords: Leptospirosis, Myocarditis, Nonoliguric renal failure.

Introduction

Leptospirosis is one of the most common and important zoonotic infections worldwide. Leptospirosis generally presents with features of bacterial infection in the acute phase followed by multi-organ complications and may be complicated by jaundice and renal failure, pulmonary hemorrhage, acute respiratory distress syndrome, myocarditis, rhabdomyolysis, and uveitis. Myocarditis and acute pancreatitis are very rare manifestations of leptospirosis. In this article, we present a case of leptospirosis with myocarditis with hypokalemia and non-oliguric acute renal failure.[1]

Case Report

A 45-year female patient, with a known case of seizure disorder for 1o years, on tablet sodium valproate 500 mg BD, presented in a medical emergency with chief complaints of chest pain associated with shortness of breath. The patient gave a history that she had a fever which appeared 10 days before the present episode. The fever was continuous without any associated chills and rigors but was accompanied by generalized body aches including retro-orbital pain. The fever lasted for 6 days and the patient was febrile for last4days before the above-mentioned presentation. The current complaint of chest pain was acute in onset, in the left side of the chest, non-radiating, aggravating on exertion, and associated with difficulty in breathing. There was no history of cough, expectoration, trauma to the chest, nausea vomiting. No history or decreased urination. On examination, she was mildly tachypneic (Respiratory rate: 22) with oxygen saturation of 98% at room air and blood pressure of 140/86, pulse rate -86/minute regular rhythm. On examination, she had bilateral equal air entry with a clear chest.

Corresponding Author: Amit Kumar, Senior Resident, Department Of Medicine - Pt. BD Sharma Pgims Rohtak.

E-mail: Amitgaider390@Gmail.Com

Her cardiac, and abdominal examination were also normal. Her CNS examination was normal with no focal neurological deficit.

Investigations done in emergency revealed electrocardiography showing generalized T wave inversions with normal sinus rhythm (with normal QTc interval). In light of generalized T wave inversion, a bedside 2D echo was done in which there were no regional/global/valvular or pericardial abnormalities. Her complete hemogram revealed Hb -11.3, TLC-13000/mm^3, and a platelet count of 3.1lakhs. The urine examination revealed hematuria (RBCs-10-12) along with mild proteinuria (2+). Blood biochemistry revealed blood urea-177 mg/dl, serum creatinine 7.0 mg/dL with serum sodium-130mEq/L, and potassium-2.8mEq/L. Her LFT was within normal limits and her creatinine kinase was found to be elevated. Her chest radiography was within normal limits and also her USG abdomen revealed no abnormality her bilateral kidney size and echo texture were normal.24-hour urinary protein test revealed 1.3 g/day of proteinuria.

In the background of pyrexia rapid test for dengue and malaria were done and was found to be negative. Serology for scrub typhus and rickettsia was also negative. But her IgM leptospirosis was found to be positive by ELISA. ANA and viral serology (HIV/HBsAg/HCV) were negative. Based on the clinical profile and positive report for leptospirosis, the patient was hospitalized. The patient had high blood urea with raised serum creatinine with low potassium and adequate urine output and thus she was diagnosed with a case of Leptospirosis with acute myocarditis with acute non-oliguric hypokalemic renal failure. The patient was given the antibiotic Tab Azithromycin along with supportive care and vital monitoring.

The patient's symptoms gradually improved and her renal parameters returned to normal by the 5th day of hospitalization. Her ECG changes of T wave inversion also reverted within one week of the hospital stay. Her Creatine kinase also returned to normal range. The patient was discharged in an asymptomatic state after having recovered from acute illness.

Table 1: Laboratory parameters of our reported case with leptospirosis

Test	Parameters at admission	Parameters at discharge	Reference value
Haemoglobin (g/dL)	11.3	9.0	12.9-15.9
Leucocytes (cells x10^9/L)	13000	6900	3.7-10.1
Platelets (cells x10^9/L)	310	210	155-366
Urea (mg/dL)	177	36	10-50
Creatinine (mg/dL)	8.0	2.3	0.7-1.3
K^+ (mEq/L)	3.6	4.6	3.5-5.5
Na^+ (mEq/L)	143	136	135-155
Uric acid (mg/dL)	6.1	5.6	3-7
Total bilirubin (mg/dL)	0.58	0.4	0.2-0.8
Direct bilirubin (mg/dL)	0.2	0.1	0-0.2
Aspartate aminotransferase (IU/L)	23	16	0-40
Alanine aminotransferase (IU/L)	37	12	0-40
Total protein (g/dL)	7.6	7.2	6-8
Albumins (g/dL)	4.3	3.2	3.5-5 .2
PT/INR	14/1.04		11-13.5/0.8-1.1
CPK total (IU/L)	346		0-170
CRP mg/L	21		<5.0
Urinary Albumin	2+	1+	negative
Urinary RBCs/hpf	10-12	3-4	0-2
Urine for dysmorphic RBCs	negative		negative
Urine for myoglobin	negative		negative
24 hr urinary protein (g/day)	1.2 g		0.04-0.15

Discussion

Leptospirosis, essentially a zoonotic disease that is bacterial in origin, is caused by pathogenic strains of Leptospira and is prevalent worldwide. Human infection occurs by direct contact or with exposure to soil or water contaminated by the urine of the reservoir hosts like cattle, dogs, pigs, and rodents Outbreaks of leptospirosis have been increasing in India for the past three decades. The positivity rate for the disease is notable in the southern part of India at 25.6%, followed by 8.3%, 3.5%, 3.1%, and 3.3% in northern, western, eastern and central India, respectively, where heavy monsoon, animal rearing practices, unplanned urbanization and agrarian way of life predispose to this infection.[2]

More than ten genetic types of Leptospira cause disease in humans. Both wild and domestic animals can spread the disease, most commonly rodents. The bacteria can spread through contact with animal urine, water, or soil contaminated with animal urine, coming into contact with breaks in the skin, eyes, nose, and mouth. In our country, farmers, people working in cleaning sewage, and low socioeconomic strata with poor sanitation are at high risk. It is a biphasic illness. Acute phase (first week of illness), clinical features include abrupt onset of fever, rigors, myalgias (especially in the calves and lower back), and headache; these symptoms occur in 75 to 100 percent of patients. Approximately half of the patients experience nausea, vomiting, and diarrhea, and non-productive cough occurs in 25 to 35 percent of cases. Less common symptoms include arthralgias, bone pain, sore throat, and abdominal pain. Acalculous cholecystitis and pancreatitis have been described in children.[3]

In some around 20%, symptoms resume after one to three days, initiating the immune phase of the disease, which last for 4 to 30 days. "Immune" phase (delayed phase of illness), the second phase thus can be complicated by acute renal failure, jaundice, myocarditis, pulmonary hemorrhage, meningitis, uveitis, and optic neuritis. IgM antibodies are commonly found in that phase, and the severity of leptospirosis is associated with the intensity of the humoral immune response of the host. Serological tests are used most frequently for the diagnosis of leptospirosis. MAT is still the golden standard in the diagnosis of leptospirosis. In our case, diagnosis of leptospirosis was established with positive serology and IgM ELISA test for Leptospira.

Although cardiac involvement is common in leptospirosis but severe cardiac dysfunction is rare. Myocarditis usually occurs during the 5–7th day of leptospiral infection. This coincides with the 'immunogenic' phase of the disease. A study done by TRIVEDI et al in India in 2003 revealed that the incidence of cardiac involvement in leptospirosis in India is 56% and out of these 52%patients had ECG changes. ECG changes in leptospirosis may present as sinus tachycardia to relative Bradycardia, bundle branch block, low voltage QRS complex, Intraventricular conduction defects, atrial fibrillation,1st and 3rddegree A-V block, and nonspecific ventricular repolarization. Pathogenesis of myocarditis and ECG changes is still not clear. The ECG Changes may be the result of electrolyte abnormalities associated with leptospiroses like hypokalemia or hyperkalemia, hypocalcemia, or hypomagnesemia. These ECG changes are associated with lower potassium levels, decreased platelet counts, and transaminitis with raised bilirubin

Myocarditis involvement in leptospirosis is the result of the complex intersection between host immunity and organism. Systemic vasculitis has been postulated to be one of the mechanisms of organ dysfunction in leptospirosis. Widespread activation of the immune system may result in sepsis-like syndrome which may result in direct myocardial involvement or it may be a result of systemic vasculitis caused by leptospirosis. Autopsy study in these patients shows mononuclear infiltration in the epicardium, interstitial myocarditis with pericardial effusion, and coronary arteritis.CK-MB levels may be elevated in the patient but it does not appear as a reliable marker for cardiac involvement. So is with elevated troponin level as there is no pathological or prognostic significance associated. Echocardiography is also not a reliable tool for determining early involvement. For the treatment part, the role of immunomodulatory therapy in myocarditis of any cause is inconclusive. Methylprednisolone although showing some mortality benefit in the presence of less severe leptospirosis failed to improve survival in leptospirosis with MODS.[4]

Conclusion

Most cases of leptospirosis are mild and resolve spontaneously. Early initiation of antimicrobial therapy may prevent some patients from progressing to severe disease. Empirical treatment should be as soon as the diagnosis of leptospirosis is suspected. it is shown in two open-label studies that ceftriaxone, cefotaxime, or doxycycline is a satisfactory alternative to penicillin for the treatment of severe leptospirosis. Aggressive fluid and electrolyte therapy requires in nonoliguric renal dysfunction patients to prevent dehydration and precipitation of oliguric renal failure. Peritoneal dialysis or hemodialysis should be provided to patients with oliguric renal failure. There is no specific treatment identified for myocarditis in leptospirosis. To prevent the precipitation of arrhythmia electrolyte abnormalities should be corrected as soon as possible. Although vasculitis is the main pathogenesis in cardiac involvement studies showed that there is no role of corticosteroids and immunomodulators in management. Our patient was treated with a tablet of azithromycin 500 mg OD, an injection of ceftriaxone 1 gm BD, and IV fluids with supportive care. Because the clinical features and diagnostic findings of leptospirosis are not specific, a high index of suspicion must be maintained for the diagnosis. Early clinical suspicion and laboratory confirmation of leptospirosis are essential since delayed diagnosis may increase mortality.

In conclusion, leptospirosis should be considered as a preliminary diagnosis in our country during the tropical fever season and a person's belonging from high risk in the community. We recommend starting empiric treatment before confirmation of laboratory tests in patients with suspected Leptospirosis to prevent them from landing into severe leptospirosis and thus decrease the mortality associated with it.

Informed Consent: written informed consent was taken from patients.

Ethical Approval: ethical committee approval was not required from the committee as per protocol for case reports.

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References

1. Pushpakumara J, Prasath T, Samarajiwa G, Priyadarshani S, Perera N, Indrakumar J. Myocarditis causing severe heart failure--an unusual early manifestation of leptospirosis: a case report. BMC Res Notes. 2015 Mar 13;8:80.
2. Kamath S. Leptospirosis In: Das S Ed. API Medicine Update, Mumbai. 2003;13:1008-11. In.
3. Sohan L, Shyamal B, Kumar TS, Malini M, Ravi K, Venkatesh V, et al. Studies on leptospirosis outbreaks in Peddamandem Mandal of Chittoor district, Andhra Pradesh. J Commun Dis. 2008 Jun;40(2):127–32.
4. Vilaichone RK, Mahachai V, Wilde H. Acute acalculous cholecystitis in leptospirosis. J Clin Gastroenterol. 1999 Oct;29(3):280–3.