CASE REPORT

Anuria, an Atypical Presentation of Leptospirosis: A Case Report

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ABSTRACT

Leptospirosis, an underreported disease, is a highly prevalent spirochaetal zoonotic disease in both tropical and temperate climates. Symptoms can range from mild illness to potentially life-threatening infection. Laboratory tests are nonspecific. Microbiological confirmation is not widely available in endemic developing countries like Nepal. We need to rely on the serologic test, which has its own pitfalls in the initial days of illness. Here, we report a case of 56 years old female from the western region of Nepal who presented with fever, jaundice and anuria. She initially tested negative for leptospirosis but was later found to be positive in the second week of illness. Unlike the usual non-oliguric renal failure in leptospirosis, she presented with anuria requiring haemodialysis and subsequently had a good recovery with treatment. We highlight the importance of clinical suspicion and logical interpretation of serologic tests based on its timing from the onset of illness.

Keywords: anuria; case report; leptospirosis; renal insufficiency; zoonoses.

INTRODUCTION

Leptospirosis is a spirochaetal zoonotic disease prevalent in both tropical and temperate climates. The most notable source of infection for humans is brown rat, highly contagious, especially in rainy seasons in areas of poor sanitation after direct or indirect exposure to infected animals shedding Leptospira in their urine.1 Patients present with the self-limiting anicteric, nonspecific flu-like illness presenting as fever, chills, headache, lethargy, and myalgia in 90% of the cases whereas jaundice, acute renal failure, and respiratory distress in about 10% of the cases.2 Leptospirosis needs to be strongly suspected if farmers presents with these symptoms from endemic area.

CASE REPORT

A 56-year-old female from western Terai presented to our emergency department in September with a history of fever for five days, the maximum temperature documented 102°F Fahrenheit, associated with chills, rigor, backache and polyarthralgia. She also complained of progressive yellowish discoloration of eyes, darkening of the colour of urine and mild to moderate abdominal pain on the right upper quadrant region. It was not associated with itching or clay coloured stool. Her urine output decreased amounting to oliguria, three days after the onset of fever which then progressed to anuria in the following days.

She did not have a history of use of Non-steroidal anti-inflammatory drugs (NSAIDs) or alternative medicines, prior history of nausea, vomiting, loose motions, neck stiffness or altered mental status.

On her initial presentation, she was ill-looking and had pallor as well as icterus. There were no signs of clubbing or lymphadenopathy. She had a firm, tender liver measuring three centimetres below the right subcostal margin in the midclavicular line. There was no splenomegaly. The rest of the systemic examinations were unremarkable.

Her laboratory parameters at the time of admission are given below. Her liver function test showed conjugated hyperbilirubinemia (Table 1).

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Table 1. Laboratory parameters of the patient at the time of admission and discharge with the reference value.

| Parameters                  | Obtained value (at the time of admission) | Obtained value (at the time of discharge) | Reference value |
|-----------------------------|------------------------------------------|------------------------------------------|-----------------|
| Total leucocyte count       | $30 \times 10^3$/mm$^3$                  | $12.7 \times 10^3$/mm$^3$                | 4,000-11,000/mm$^3$ |
| Neutrophils                 | 70%                                      | Not available                            | <60%            |
| Lymphocytes                 | 20%                                      | Not available                            | <20%            |
| Hemoglobin                  | 8.3g/dl                                  | 8.54g/dl                                 | 14-18g/dl       |
| Platelet count              | $78 \times 10^3$/mm$^3$                  | $420 \times 10^3$/mm$^3$                | 150,000-450,000/mm$^3$ |
| Creatinine                  | 692µmol/L                                | 355µmol/L                                | 62-115µmol/L    |
| Blood Urea Nitrogen (BUN)   | 20mmol/L                                 | 15mmol/L                                 | 8 - 20mmol/L    |
| Sodium                      | 123meq/L                                 | 139meq/L                                 | 136 - 145meq/L  |
| Potassium                   | 3.6meq/L                                 | 3.7meq/L                                 | 3.5 - 5meq/L    |
| Total bilirubin             | 620µmol/L                                | 46µmol/L                                 | 5.1 – 17.1µmol/L |
| Direct Bilirubin            | 210µmol/L                                | 21µmol/L                                 | 1.7 – 5.1µmol/L |
| Aspartate transaminase      | 149U/L                                   | 19U/L                                    | 10 – 40U/L      |
| Alanine transaminase        | 94U/L                                    | 6U/L                                     | 10 – 40U/L      |
| Alkaline Phosphatase        | 505U/L                                   | 258U/L                                   | 30 – 120U/L     |
| Albumin                     | 25g/L                                    | Not available                            | 35 – 55g/L      |

DISCUSSION

Leptospirosis is a neglected zoonotic disease, caused by aerobic spirochete namely Leptospira sp. and is endemic in tropical countries. Rodents are the natural reservoirs and source of transmission while humans are the accidental hosts. The portals of entry are cut or abrasion on the skin and mucous membrane of the conjunctiva and oral cavity. Leptospira disseminates via the bloodstream in the body without forming any skin lesions unlike other spirochetes and settles in renal tubules and peritubular capillaries because of a special affinity for them. Evidence shows that the cytokine storm with the release of IL-10, TNF-alpha occurs in severe leptospirosis when there is a high level of leptospiraemia. The high bacterial load and dysregulated host response explain the multiorgan failure and sepsis in leptospirosis. The incubation phase averages from seven to twelve days, though it can be as short as three days or as long as a month. The patient usually present with wide range of symptoms ranging from mild fever, headache, back pain, lethargy, and myalgia, to severe symptoms presenting with multiorgan failure with a major target of kidney, liver, and lungs, popularly known as Weil’s syndrome. Conjunctival suffusion is a pathognomonic feature of leptospirosis.

Leptospirosis has a special predilection for proximal renal tubules, which has been attributed to the peculiar characteristic of non-oliguric renal failure with or without hypokalemia. Acute renal failure in leptospirosis usually takes five to eight days to recover. Significant improvement in urine output...
after adequate hydration is observed in oliguric renal failure. Seguro et al showed that 46% of oliguric patients at admission will become non-oliguric after volume replacement and use of furosemide. Similarly, patients with non-oliguric renal failure had a better prognosis than oliguric renal failure in leptospirosis. The common contributing factors for the renal failure are dehydration, rhabdomyolysis and high bilirubin. However, the renal function is restored. Dialysis dependence is very rare and only nine percent of cases show features of early-stage CKD. It takes usually six months for full recovery of GFR, whereas, urine concentrating ability may take a longer time. In contrast to usual findings, our patient had anuria at the time of presentation, required haemodialysis for life support and eventually had a full recovery of GFR in three months.

IgM ELISA is used for screening and confirmation of acute leptospiral infection but requires an adequate number of detectable antibodies which are produced after five to seven days. The molecular tests such as PCR are better at detecting leptospirosis in acute septicaemic phase, which occurs in three to seven days. In severe leptospirosis, combined treatment with doxycycline and cephalosporin, cefotaxime would be the better choice than doxycycline alone. Besides, doxycycline is a cheap and effective antibiotic for an empirical treatment in endemic areas where leptospirosis is a common clinical suspicion. Leptospirosis is an underappreciated and neglected tropical disease. It should be a common and foremost differential diagnosis in an endemic area when a patient presents with fever, jaundice, and anuria even if the antibody based serologic test is negative.

Conflict of Interest: None.

Consent: JNMA Case Report Consent Form was signed by the patient and the original article is attached to the patient’s chart.

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