Leptospirosis Presenting as Acute Respiratory Distress Syndrome (ARDS) in Sub-Himalayan Region

V Chauhan*, DM Mahesh**, P Panda**, J Mokta##, S Thakur#

Abstract

Indira Gandhi Medical College, Shimla receives referred patients of pyrexia with multi-organ dysfunction during the monsoon season from all over the state of Himachal Pradesh. Most common etiologies of pyrexia are enteric fever, scrub typhus, malaria, viral, tubercular, and some patients of dengue fever from adjoining states. Leptospirosis has not yet been reported in sub-Himalayan state of Himachal Pradesh, India. We present here a case of leptospirosis presenting as ARDS, proven on IgM Elisa and confirmed by PCR. Leptospirosis is a new etiology in this region for patients presenting with pyrexia and ARDS.

Introduction

Leptospirosis is a zoonosis which in its milder form resembles any other viral illness and in its severe form needs to be differentiated from other common infection in tropical regions of India like scrub typhus, dengue, malaria, viral hepatitis, and Hantavirus infection. We are reporting here a case that presented with pyrexia for 9 days and shortness of breath proven to be ARDS on blood gas analysis. He was incidentally found positive for leptospirosis (IgM ELISA) and recovered fully with ventilatory support plus injectable ceftriaxone and oral doxycycline. The infection was later confirmed by the Regional Medical Research Centre (ICMR), Port Blair by PCR method. This is the first reported case of leptospirosis from this Sub-Himalayan region of North India.

Case Report

This is a report of 24 yr old male from Sub-Himalayan region of North India, District Bilaspur in Himachal Pradesh who presented with a history of pyrexia for 9 days. Patient had cough which was dry, accompanied by progressive shortness of breath for 4 days. He also had generalized bodyaches and conjunctival suffusion. Patient developed acute confusional state one day before admission. There was no rash, lymphadenopathy, diarrhea, or sore throat. There was no history of travel to any other place prior to pyrexial illness. There was history of contact with cattle, rodents, and patient was from rural background.

On examination patient had sinus tachycardia with a pulse rate of 120/min, blood pressure of 130/82 mm Hg. He had severe cyanosis and a respiratory rate of 32/min. There was no hepatosplenomegaly, and there were crepitation over bilateral bases. Patient was irritable and confused, there were no signs of meningism, no cranial nerve deficits and was moving all four limbs spontaneously. Plants were downgoing and deep tendon jerks were normally elicitable.

Patient was admitted with a possibility of pyrexia with acute confusional state and ARDS. The investigations showed hepatorenal dysfunction. His serum bilirubin, SGOT, SGPT, and alkaline phosphatase at admission were 2.18 mg%, 158 IU/L, 96 IU/L, and 912 IU/L respectively. The urea and creatinine at admission were 72 mg% and 2.3 mg% respectively. His total leucocyte count was 14,200/cumm. Chest X-ray showed bilateral diffuse chest infiltrates (fig. 1). His arterial blood gas showed severe acidosis with pH of 7.16 and pO\(_2\) of 29 mmHg, HCO\(_3\) was 10.9 mmol/L, pCO\(_2\) was 44.4 mmHg. Based on the chest x-ray and patient’s pO\(_2\)/FiO\(_2\) =138 [<200], he was labeled to be having Acute Respiratory Distress Syndrome (ARDS). Platelet count was 49,000/cumm and creatinine phosphokinase (CPK MB) levels were 11.0 IU/L (normal). Possibility of scrub typhus or leptospirosis was kept and patient treated with ceftriaxone and doxycycline. He was immediately given oxygen ventilatory support. His blood and urine culture were sterile, there were no malaria parasites on blood smear, and Weil Felix and widal were negative. IgM ELISA (Serion Virion Germany) was done for leptospira and was positive with a titre of 23. Patient gradually improved with the same treatment and was on spontaneous respiration after 5 days. He became afebrile after 6 days of treatment. At discharge the values of serum bilirubin, SGOT, SGPT, and alkaline phosphatase were 0.4 mg%, 157 IU/L, 125 IU/L, and 238 IU/L respectively. Platelet counts were normal and at discharge the urea and creatinine were 40 mg% and 1.0 mg% respectively. Patient was discharged after 14 days of admission with a final diagnosis of Pyrexia with multiorgan dysfunction including ARDS, hepatorenal dysfunction, and acute septic encephalopathy etiology for which was acute leptospirosis. His convalescent serum was sent to the research lab at ICMR Port Blair, on which microagglutination test (MAT) and PCR was done. MAT was inconclusive but the infection was confirmed positive by PCR method.
Discussion

Leptospirosis is a worldwide zoonosis caused by pathogenic species of the genus *Leptospira*. The initial diagnosis of leptospirosis depends on a high index of clinical suspicion, as routinely available diagnostic tests are unreliable in the initial period.

Exposure to moist soil has been found to be established risk factor in south Indian patients. Most of the cases occur in monsoon season in endemic areas of India and multi-organ dysfunction syndrome, primarily acute respiratory distress syndrome with thrombocytopenia and renal failure are the causes for mortality. Our patient also developed the infection in monsoon season, and belonged to a place which is warm and humid, but there was no water clogging in the area. The patient was from a rural setting where exposure to cattle and rodents is present. There are no seroprevalence studies available from this region till now either in animals or humans.

In 90% of cases, leptospirosis manifests as an acute febrile illness with a biphasic course and an excellent prognosis. Nonspecific signs and symptoms of leptospirosis (eg, fever, headache, nausea, vomiting) are often confused with viral illness.

In 10% of cases, the presentation is more dramatic, and the infection has a mortality rate of 10 percent. Known as Weil disease or icteric leptospirosis, the classic definition of this form of leptospirosis includes fever, jaundice, renal failure, and hemorrhage. Other organ systems (ie, pulmonary system, cardiac system, CNS) are also frequently involved. The patients of leptospirosis have elevated serum levels of bilirubin and alkaline phosphatase as well as mild increase in aminotransferases (up to 200 U/L). Levels of CPK are elevated in upto 50% during first week of illness and help to differentiate this from viral hepatitis. Mild thrombocytopenia occurs in upto 50% and is associated with renal failure. Pulmonary manifestations arise usually from 3-9 days after onset of illness. Our patient showed elevated alkaline phosphatase, mild elevation of serum bilirubin and aminotransferases, thrombocytopenia, acute renal failure and pulmonary involvement in form of ARDS.

Pulmonary manifestations occur in 20–70% of patients and in many patients can progress to ARDS. ARDS is usually seen in second week of illness in severe form of leptospirosis. Our patient showed pulmonary involvement in the form of ARDS, though there was no clinical evidence of pulmonary hemorrhage. An autopsy study in India on 62 leptospirosis patients showed massive intra-alveolar haemorrhage in 48 (77%) cases, acute interstitial nephritis or acute tubular necrosis 45 (72%) cases, and myocarditis 24 (32%) cases. The drug of choice is penicillin and ampicillin but doxycycline and ceftriaxone are equally effective.

The traditional gold standard for diagnosis is microagglutination test (MAT), which is available only in reference centres. Other tests done to confirm the diagnosis are PCR, Indirect hemagglutination assay (IHA) and IgM ELISA. IgM ELISA (Serion ELISA) done in our patient for leptospira is a rapid quantitative diagnostic test. Our patient was later tested and found positive by PCR method by the ICMR Port Blair. To the best of knowledge this is the first case of Leptospirosis to be reported from Himachal Pradesh presenting as ARDS.

References