Response to bolus methylprednisolone in Pulmonary Leptospirosis: Case Report

Amrit Ghimire[1], MD; Sanjay Raj Thapa[1], MD
[1]Department of Pediatrics, Grande International Hospital, Kathmandu Nepal

ABSTRACT

Pulmonary complications in leptospirosis are often unrecognized in a non-endemic area. We report here a patient with leptospirosis and severe pulmonary involvement who was treated with corticosteroids. This case report shows the beneficial effect of methylprednisolone in pulmonary leptospirosis, which usually has an aggressive course and grave outcome.

Keywords: Leptospirosis; Pulmonary; Acute Respiratory Distress Syndrome (ARDS); Methylprednisolone

Introduction

Leptospirosis is a widespread zoonosis caused by aerobic, motile spirochetes of the genus Leptospira. Pulmonary manifestations of leptospirosis are frequent, and now being increasingly reported.[1] When a patient from a non-endemic area presents with pulmonary involvement without the classical symptoms, leptospirosis is not often suspected.[2] We present a case with severe lung involvement in leptospirosis who responded to parenteral methylprednisolone. It also prevented the need for ventilator support. Use of bolus methylprednisolone was found to play an important role in infrastructure limited settings.

Case Report

A 16 year old boy from Butwal of Nepal was admitted in the month of August with fever for 12 days and increased shortness of breath for 3 days. He was admitted in the local hospital for 5 days where he received Inj Ceftriaxone and Inj Amikacin for 3 days followed by Inj Meropenem and Inj Vancomycin for 2 days. Relevant prior investigation were suggesting raised liver enzymes.Dengue IgM and Leptospira IgM were negative. On examination, he was febrile (102°F), tachypnoeic and tachycardic. Blood pressure was normal. On chest auscultation, there was diffuse crepitations all over the chest. Liver was palpable 3 cm below right costal margin.

His hemoglobin was 11.5gm/dl with total leucocyte count of 23800/cu mm (neutrophil 25.2%, lymphocyte 59.8%), platelet 1.96 thousand/µl, C reactive Protein (CRP) 40mg/L, Total Bilirubin 2.1mg/dl, SGOT 605U/L, SGPT 296U/L. Blood culture was sterile. Chest x-ray revealed bilateral symmetrical patchy opacities (Figure 1). Contrast enhanced computerized tomography of chest was suggestive of extensive consolidation in both

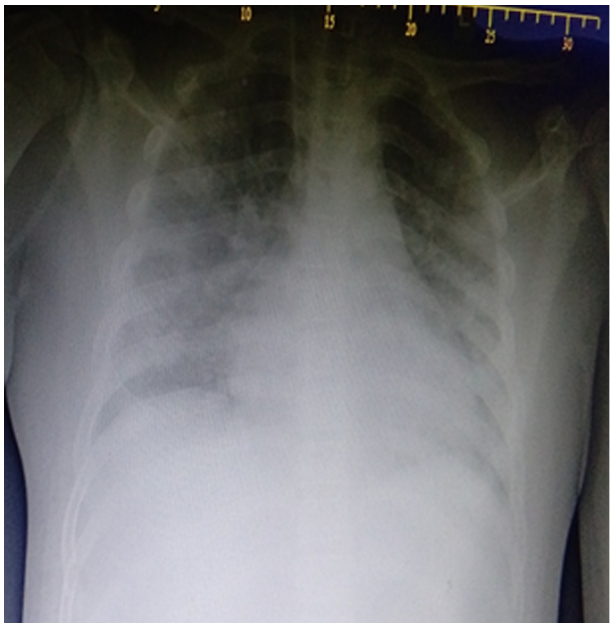

Figure 1: Chest radiograph on admission showing bilateral uniform opacities with a peripheraldistribution

Corresponding author:
Amrit Ghimire, MD
Email: amrit_sathi@yahoo.com
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lung fields with minimal bilateral pleural effusion (Figure 2). Arterial Blood gas report showing PaO2:FiO2=152. Severe Pneumonia with Acute Respiratory Distress Syndrome (ARDS) was the provisional diagnosis. He was admitted in Intensive Care Unit (ICU) and Inj Meropenem, Inj Vancomycin and Inj Azithromycin were started. Oxygen via ventury mask was adjusted at FiO2 60%.

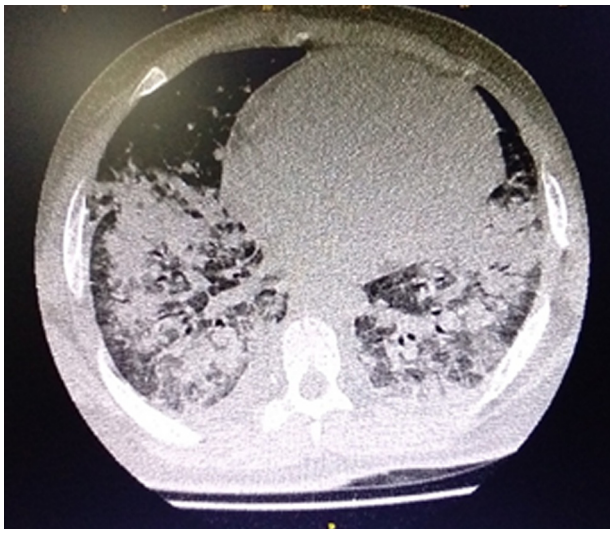

Figure 2: Computed tomography (CT) image on admission showing bilateral symmetrical opacities

On 2nd Day of our hospital admission and almost 2 weeks after the onset of symptoms, the serology (IgM) for leptospirosis was reported positive. Since a microscopic agglutination test was not available, detection of IgM antibodies was taken as evidence of recent infection with *Leptospira* in this clinical context. Considering the diagnosis of Pulmonary manifestation of Leptospirosis, he was pulsed with 2mg/kg/day of methylprednisolone once a day for 3 days followed by oral prednisolone. The oxygen requirement came down and he was weaned from oxygen in next 2 days. In the next 5 days the patient showed definite signs of improvement and was transferred to the general ward. Resolution of opacities was seen in chest X-ray on 6th day (Figure 3). Oral prednisolone was continued for 10 days and stopped. Liver enzymes gradually decreased.

Discussion

Leptospirosis is the infectious disease caused by pathogenic organisms belonging to the genus Leptospira. Pathogenic leptospires are maintained in nature in the renal tubules and genital tract of certain animals. Cattle, buffaloes, horses, sheep, goat, pigs, dogs and rodents are common reservoirs of leptospires. Leptospirosis is most common in

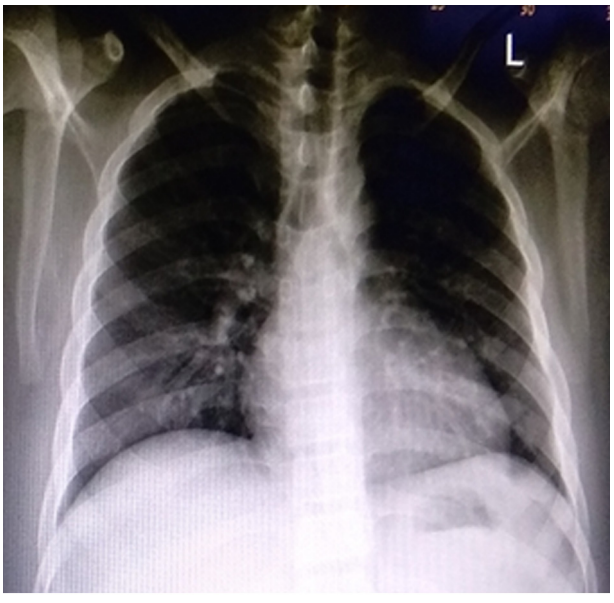

Figure 3: Chest radiograph on 6th day of admission showing total resolution of the opacities

tropical and subtropical areas with high rainfall. Most common cases have been reported from India, Indonesia, Thailand and Sri Lanka.

The first publication of lung involvement in leptospirosis is attributed to Moeschlin in 1943.[3]The spectrum of pulmonary manifestations is wide, ranging from mild respiratory symptoms to the presence of acute respiratory distress syndrome (ARDS). Cough, haemoptysis, and different grades of dyspnoea are the most common pulmonary symptoms. Chest radiographs usually show bilateral lower lobe involvement with a peripheral distribution. Nodular or reticulonodular infiltrations, consolidations, and a groundglass appearance are the most common patterns seen.[4]

Alveolar haemorrhage and ARDS are two of the most fatal conditions associated with leptospirosis.[5-7] Our case presented with the features of ARDS. In the most severe pulmonary form of leptospirosis, where mortality rates can be as high as 30–60%, respiratory symptoms usually appear between the 4th and 6th day of disease. [7,8] In our case respiratory symptoms appear on 9th day of illness. Although the pathogenesis of pulmonary manifestations is poorly understood, vasculitis mediated by toxins and an exaggerated immune response in the host are believed to be responsible.[8]

Postitive serology is not always proof of current infection. It is the seroconversion or a four fold rise in titre in consecutive serum samples to be diagnostic proof of recent or current infection. IgM

class antibody may remain detectable for several months or years. In our patient seroconversion was reported at 12th day of illness. Antileptospires IgM may be detected 4 to 5 days after the onset of symptoms, before detection of IgG and agglutinating antibodies, and persist at least 5 months in patients.[9]

A recent review showed that the benefit of antibiotic therapy in the treatment of leptospirosis, particularly for severe disease, remains unclear and the choice of penicillin, doxycycline, or cephalosporin did not affect mortality rates nor the duration of fever.[10] Although benefits of corticosteroids in ALI is known, evidence for use of corticosteroids in pulmonary leptospirosis is confined to occasional case reports or small studies.[11,12] In a series of 30 patients, Shenoy *et al*. demonstrated that corticosteroids reduce mortality and change outcomes significantly.[11]In this study, Inj Methylprednisolone was given for first 3 days followed by oral prednisolone for next 7 days. Similar regimen was used in our case. Trivedi *et al*. found that mortality was higher in patients with pulmonary involvement who did not receive steroids and concluded that high dose glucocorticoids should be given as early as possible after the onset of dyspnoea to all the patients with pulmonary involvement.[13] Our patient received methylprednisolone and the outcome improved significantly.

Conclusion

In this case, the administration of corticosteroids led to clinical improvement and complete recovery. The clinical response suggests the need for the use of corticosteroids in pulmonary involvement of leptospirosis.

References:

1. Simpson FG, Green KA, Haug GJ, et al. Leptospirosis associated with severe pulmonary haemorrhage in Far North Queensland. Med J Aust 1998;169:151–3.
2. Assimakopoulos SF, Fligou F, Marangos M, Zotou A, Psilopanagioti A, Filos KS. Anicteric leptospirosisassociated severe pulmonary hemorrhagic syndrome: A case series study. Am J Med Sci 2012; 344:326–9.
3. Carvalho CR, Bethlem EP. Pulmonary complications of leptospirosis. Clin Chest Med 2002; 23:469–78.
4. Tanomkiat W, Poonsawat P. Pulmonary radiographica findings in 118 leptospirosis patients. Southeast Asian J Trop Med Public Health 2005; 36:1247–51.
5. Marchiori E, Lourenco S, Setubal S, Zanetti G, Gasparetto TD, Hochhegger B. Clinical and imaging manifestations of hemorrhagic pulmonary leptospirosis: A state-of-the-art review. Lung 2011;189:1–9.
6. Clavel M, Lheritier G, Weinbreck N, Guerlin A, Dugard A, Denes E, et al. Leptospirosis: An unusual cause of ARDS. Crit Care Res Pract 2010; 2010:408365.
7. Dolhnikoff M, Mauad T, Bethlem EP, Carvalho CR. Leptospiral pneumonias. Curr OpinPulm Med 2007;13:230–5.
8. Silva JJ, Dalston MO, Carvalho JE, Setubal S, Oliveira JM, Pereira MM. Clinicopathological and immunohistochemical features of the severe pulmonary form of leptospirosis. Rev Soc Bras Med Trop 2002; 35:395–9.
9. Silva MV, Camargo ED, Batista L, Vaz AJ, Brandão AP, Nakamura PM, Negrão JM. Behaviour of specific IgM, IgG and IgA class antibodies in human leptospirosis during the acute phase of the disease and during convalescence. *J Trop Med Hyg* 1995;98(4):268-272.
10. Brett-Major DM, Coldren R. Antibiotics for leptospirosis. Cochrane Database Syst Rev 2012; 2:CD008264.
11. Shenoy VV, Nagar VS, Chowdhury AA, Bhalgat PS, Juvale NI. Pulmonary leptospirosis: An excellent response to bolus methylprednisolone. Postgrad Med J 2006; 82:602–6.
12. Minor K, Mohan A. Severe leptospirosis: Casedreport of treatment with intravenous corticosteroids and supportive care. Am J Emerg Med 2013; 31:449.
13. Trivedi SV, Chavda RK, Wadia PZ, Sheth V, Bhagade PN, Trivedi SP, et al. The role of glucocorticoid pulse therapy in pulmonary involvement in leptospirosis. J Assoc Physicians India 2001; 49:901–3.