



A rare case of isolated lung involvement in leptospirosis

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Abstract

In tropical regions, leptospirosis is a prevalent zoonotic illness. The most prevalent reservoirs are rodents and other animals. Humans contracted this illness through coming into touch with water, dirt, or animal tissues that had been exposed to the sickness. It is distinguished by the participation of many systems. Given that it can appear as anything from an undetected infection to hepatocellular failure, early detection is crucial to halting the fulminant course. Here, we'll discuss a severe case involving a 25-year-old female patient who went to the hospital complaining of a fever with chills, a cough, shortness of breath, and bodily aches. Bilateral heterogenous opacity in the mid and lower zone was visible on the chest X-ray. A high resolution computed tomography (HRCT) scan of the chest revealed minor pleural effusion on both sides, as well as bilateral mid- and lower-zone alveolar opacities and a few regions of bronchial dilatation that may have been caused by an alveolar haemorrhage. Highly positive results from a serum IgM leptospira test indicated leptospirosis.

Keywords: acute respiratory distress syndrome, immunosuppressants, leptospirosis, noninvasive ventilation, pulmonary hemorrhage

Introduction

"Leptospira interrogans" is the cause of leptospirosis [1]. Weil's illness, which frequently involves numerous organs and has a high case fatality rate, typically results in moderate disease. The sickness has a biphasic pattern, with the acute or septicemic phase lasting one week and the immunological phase lasting an additional one. Jaundice, renal failure, hepatic necrosis, pulmonary involvement, and hemorrhagic diathesis are all symptoms of the severe type, which involves many organs. In cases of severe leptospira infection, isolated lung involvement with acute respiratory distress syndrome (ARDS) [4] is extremely uncommon. We recently managed a case of leptospirosis with isolated lung involvement in terms of alveolar haemorrhage and ARDS [3].

Case Report

A 28-year-old female paddy field worker from a remote area complained of having a high grade fever for six days, chills, and a terrible bodily ache, as well as a cough for three days that made her breathless. On the day of admission, she noticed a sudden rise in breathing trouble and one incident of blood mixed sputum. Upon examination, she showed signs of low grade fever, hypoxia (SpO₂ 62% on room air), sinus tachycardia, hypotension (blood pressure 90/50 mmHg), low blood pressure, and low blood sugar. Reduced bilateral air intake is audible, along with rough basal creeps. Type 1 respiratory failure was detected by the first arterial blood gas (pH 7.49, PCO₂ 21.6, HCO₃ 17, PO₂ 41). Laboratory tests showed that the RFT was normal, the serum total bilirubin was 0.8 mg/dl, the Hb was 7.3 g%, the platelet counts were 78,000/cmm, aspartate transaminase

was 93, and alanine transaminase was 56. An X-ray of the chest was taken in PA view, and it revealed bilateral heterogenous opacity in the mid and lower zones [Figure1]. In her two-dimensional echocardiogram, the right atrium and right ventricle are somewhat dilated, while the left ventricle is functioning normally. Blood procalcitonin levels were negative. Her H1N1 antigen nasal swab, antinuclear antibody, and Coomb's test results were all negative. She received high flow oxygen therapy, non-invasive breathing support, bilevel positive airway pressure (BiPAP), an injectable antibiotic (ceftriaxone), and vasopressors as her initial treatments. A high resolution computed tomography (HRCT) scan of the chest revealed minor pleural effusion on both sides, as well as bilateral mid- and lower-zone alveolar opacities and a few regions of bronchial dilatation that may have been caused by an alveolar haemorrhage. In light of the radiographic finding of an alveolar haemorrhage and the negative results of the procalcitonin test, H1N1 antigen from a nasal swab, antinuclear antibody, and Coombs test, serum IgM leptospira was performed using the ELISA method. High positive value was found in the serum IgM leptospira test (>100 u/ml, negative).

Discussion

The most severe type of leptospira infection, Weil's disease, as well as subclinical infection and an unexplained febrile sickness are all connected to various focal organ failure. Major hemorrhagic problems from the severe type are typically brought on by renal involvement, jaundice, and other symptoms. Lung involvement might range from non-fatal pulmonary haemorrhage and ARDS to severe clinical symptoms. In the immunological phase, pulmonary

involvement often manifests as overt pulmonary symptoms in 20–70% of individuals, the majority of which recover completely. [4] Rare isolated lung involvement causes a delayed diagnosis. Clinical signs of pulmonary involvement include cough, dyspnea, chest discomfort, and hemoptysis. The primary lung symptom is alveolar haemorrhage, which can range in severity from moderate to severe, with severity being correlated with death. [5] Toxin-mediated processes that result in vascular damage, particularly small vessel vasculitis, are one potential cause for alveolar haemorrhage. Membrane glycoproteins, lipopolysaccharides, outer membrane proteins, and hemolysins are examples of potential poisons. Leptospirosis' pathogenesis may potentially be influenced by cytokines like tumour necrosis factor alpha. [6] Vasculitis mostly impacts capillaries, which causes haemorrhage in alveoli. In addition to toxin-mediated processes, host immunological response to infection is another indirect pathogenic mechanism. [5] Circulating antibodies against the lipopolysaccharides of the pathogen cause the innate immune system to become activated, which serves as the catalyst for antibody production. [7] High fatality rates (30–60%) accompany the quick and severe course of acute pulmonary haemorrhage. [8] Leptospirosis patients hardly ever develop a cute respiratory distress syndrome, which has a significant death rate (up to 51%). [9] As a result of endothelial injury, ARDS is frequently accompanied with pulmonary haemorrhage. [8] In cases of severe lung involvement, microscopic examination reveals intraalveolar and interstitial haemorrhage, pulmonary oedema, fibrin deposition, hyaline membrane development, and proliferative fibroblastic response.

Most leptospirosis patients heal spontaneously and don't need any special treatment, according to management. Antibiotics can lessen the severity of the disease and stop the progression of mild disease, even if their effectiveness in treating leptospirosis is not well established. [11] The recommended antibiotics include penicillin, tetracycline, ceftriaxone, and doxycycline. The results of a Cochrane systematic review were insufficient to establish definitive recommendations for the usage of antibiotics.

The treatment of hypovolemia, hypotension, and electrolyte imbalances is necessary for patients with severe leptospirosis. To treat respiratory failure, early mechanical ventilation with positive end expiratory pressure, also referred to as NIV (BiPAP), and a high concentration of inspired oxygen should be employed. In situations of refractory hypoxia, extracorporeal membrane oxygenators have been utilised. In cases of severe leptospirosis, immunomodulation with the use of glucocorticoids, immunoglobulin, or plasmapheresis may be beneficial. The glucocorticoids use in leptospiral pulmonary haemorrhage has been validated by several Indian accounts. Shenoy *et al.* evaluated the effectiveness of methyl prednisone bolus and came to the conclusion that corticosteroids only had a mortality benefit when administered within the first 24 hours of the onset of pulmonary symptoms. Patients already receiving mechanical ventilation did not benefit from this reduction in the need for ventilator support. Trivedi *et al.* investigated the efficacy of plasma exchange and cyclophosphamide in treating patients with leptospiral lung haemorrhage. Activated factor VII, desmopressin, hemofiltration, and inhaled nitric oxide are a few of the cutting-edge treatments that have been tested on resistant leptospirosis patients.

Conclusion

In conclusion, leptospirosis cases in developing nations [1] like India are both underreported and under diagnosed. In cases of severe leptospirosis without jaundice and renal failure, localised lung involvement or ARDS [4] may result. People who work with diseased animals [5], either directly or indirectly through polluted water or soil, should be considered to be at high risk. On radiographic or bronchoscopic assessment, isolated lung involvement can manifest as an acute febrile illness with respiratory symptoms [4]. Complications and mortality can be avoided with early identification and treatment using oxygenation, antibiotics, and immunosuppressives.

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