International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Leptospirosis: A Case Report

Dr. Vidhya Lakshmi M A[1], Dr. Sarath bhaskar S[2], Dr. Jayanthi N[3]

Junior Resident, Department of General Medicine, ACS Medical College
Email: *saividhi15[at]gmail.com*

Senior Resident, Department of General Medicine, ACS Medical College
Email: *sarath_bhaskar[at]yahoo.com*

Associate Professor, Department of General Medicine, ACS Medical College
Email: *drjayanthin[at]gmail.com*

Abstract: ***Leptospirosis control, management, monitoring, and epidemiology in people and other animals depends on reliable diagnostics that are easy to get and use. Exposure risk, clinical presentation, and various direct and indirect diagnostic methods should all be considered in a comprehensive diagnosis. Culture, immunostaining, histology, and clinical specimens, as well as nucleic acid amplification tests (NAATs), are some of the ways that Leptospira spp. may be directly detected. Macroscopically agglutination test (MAT), enzyme - linked immunosorbent assay (ELISA), and lateral flow procedures are examples of indirect serologic methods that may detect leptospiral antibodies. In order to better understand the environmental stage of leptospirosis transmission, this study emphasised the importance of first assessing the physical characteristics and biogeochemical processes that allow Leptospira to survive in the environment for an extended period of time. Then, using epidemiological methods, we should evaluate the transmission and movement of these same strains in current wildlife and livestock.***

Keywords: leptospirosis; Leptospira; environmental zoonoses; neglected tropical diseases; one health

1. Introduction

The Leptospira bacteria cause Leptospirosis, a disease that can spread from animals to humans.[1]

During the leptospirosis phase, the leptospira infects blood vessels and cerebrospinal fluid. It also generates particular IgM and IgG antibodies, which are used as a first line of defense against Leptospirosis. Acute leptospiral infections cause a number of disorders during the leptospirosis phase when the infection spreads to several organs, including the lungs, liver, kidneys, heart, skin, muscles, and CSF fluid in the brain[2]. As far as leptospirosis therapy goes, the gold standard is the use of antibiotics such as doxycycline, erythromycin, and benzylpenicillin.

Fish, amphibians, birds, reptiles, and mammals are just a few of the many animal types that may carry leptospira. The primary risk factor for human infection is environmental exposure to disease - causing agents, such as polluted water or soil, diseased animal tissue, or urine [3].

Adolph Weil first described Leptospirosis in 1866 as a feverish illness in people who had come into touch with water; symptoms included jaundice, an enlarged spleen, kidney failure, and inflammation of the eyes. Thus, Weil's disease—the severe version of the illness—was named after him. Spirochaeta interrogans was formally named in 1907 from the shape it resembled under a microscope—a question mark—and because it is the causal organism behind leptospirosis[4]. The Andaman and Nicobar Islands were the site of the first documented case of Leptospirosis in India in 1929, according to Taylor and Goyal[5]. Both sporadic and pandemic cases have been reported in mainland India. Since the 1980s, the number of leptospirosis cases reported from India has risen significantly. More and more reports of epidemics have come in from the Indian states of Orissa, Maharashtra, Karnataka, Tamil Nadu, and Kerala[6]. An estimated 19.7 cases per 100, 000 population[7] are reported in India each year for morbidity. Here, the southern and western states are endemic to the illness.[8]

The fast and asymptomatic spread of Leptospirosis through the proximal tubule of the kidneys makes this disease notoriously difficult to contain in rodent hosts. While most infections begin in the tubular lumen, the bacteria can affect the kidneys in rare instances by releasing themselves into the urine[9]. A severe form of the infection called Weil's disease (W. D.) can develop if not treated or misdiagnosed.

Acute febrile illness is the most common symptom, and most cases are mild, rarely fatal. The most common bacterial infection causing Leptospirosis with secondary hemophagocytic lymphohistiocytosis (sHLH) is Mycobacterium tuberculosis, which is a rare and deadly disease. Immunosuppressive treatment (kind, dosage, or duration) has to be patient - specific since the pathophysiology of various secondary HLH subtypes may differ.[10]

The lack of distinct symptoms makes the identification of sHLH in critical care units challenging for clinicians. Multiple symptoms might overlap with other illnesses, such as sepsis, multiorgan dysfunction syndrome, or other cytokine storm syndromes, and sometimes various disorders can coexist[11]. Additionally, sepsis is a potential precipitating factor for sHLH. Late diagnosis, delayed initiation of HLH - specific treatment, and organ failure all increase the risk of catastrophic outcomes in HLH patients requiring critical care.

When compared to children, adults had a worse prognosis from leptospirosis - induced sHLH. Although other health conditions may hasten organ failure and change the immune response, these numbers are mostly attributable to inadequate diagnosis and care that does not comply with

current standards. Because of the similarity in symptoms and signs between sHLH and Leptospirosis, including chronic fever, organomegaly, and cytopenia, the former may go unrecognized and the latter underreported.[12]

The complicated epidemiology of Leptospirosis, the management of acute infection, and the ability to monitor and control transmission from reservoir hosts that are subclinically infected all need immediate advances in diagnostics. Nucleic acid amplification tests (NAATs), immunostaining of tissues or clinical specimens, histopathology, and culture are all ways to directly diagnose a current infection. The microscopic agglutination test (MAT), enzyme - linked immunosorbent assay (ELISA), and lateral flow techniques are indirect serologic approaches that may detect antibodies to Leptospira spp. Potential exposure, clinical manifestations, laboratory values, and the outcomes of many diagnostic tests should all be considered in a comprehensive leptospirosis diagnosis rather than depending on a single test alone[13]. There is a strong relationship between the functions of direct and indirect methods in diagnosis. In the early stages of an infection, direct detection methods tend to be more accurate, but indirect serologic methods show higher sensitivity as the illness progresses. If we want to know how common an infection is in a certain host species, we need to employ both direct and indirect approaches. However, understanding which Leptospira species and serovars are important to a given epidemiologic context and host species is essential for tailoring serologic and molecular methodologies, such as whole - genome sequencing, to that context and species. Because of factors such as the intricacy of leptospiral media, the relative sluggish development of leptospiral and contaminating organisms, and the need for darkfield microscopy for determining culture positive, the technical recovery of leptospiral isolates from clinical specimens is challenging. Selective media [14] and media that enable picky organisms to thrive are examples of recent advancements in culture techniques [15.] Because antimicrobials and supportive care work better when started early in the course of illness, better quick diagnostic tests are required to identify acute infections. The efficacy of antimicrobial treatment in eradicating renal tubular and vaginal infections in animals, as well as other measures of infectious disease transmission, can only be determined with better diagnostic tools for monitoring and management [16]. This study aims to provide a synopsis of important leptospiral diagnostics and the functions they play from a One Health standpoint, including aspects such as people, domestic animals, and cattle. Another crucial objective is to push for better diagnostic tools that can fill in the gaps and overcome the limits of the ones that are now available.

Case Report 1:

A 60 - year - old man who was otherwise healthy presented to the clinic complaining of a persistent high fever that had persisted for seven days, along with a strong cough and shortness of breath that had persisted for four days. At first admittance, the patient was completely alert and aware of the time, date, location, and identification—SpO2 - 93% at room temperature, 99% with 10 liters of oxygen. S1 and S2 of the cardiovascular system are at rest, with bilateral inspiratory cracks apparent; P/A is soft and normal, and the central nervous system is not functioning normally.[3]

The patient began intravenous therapy after receiving oxygen support and following up on Piptaz's co - administration and creatinine clearance, as well as other supportive treatments. The first evaluation revealed a slightly elevated urea level and normal white blood cell count (WBC) with thrombocytopenia. Notably, total bilirubin was almost four times greater. Citation: Cushman et al., 2009.02. The patient tested positive for lepto IgM after the fever panel.640 tigers were found to have Leptospirosis, a bacterial illness caused by bacteria belonging to the genus Leptospirosis. The MAT titer for this disease was 1. Not COVID - related. Afterward, the patient was put on injections. A severe case of Leptospirosis followed by pulmonary hemorrhage was clinically suspected, leading to the empirical administration of Doxycycline and Inj. Dexamethasone. The binomial micropattern was seen on the HRCT chest, and there were centrilobular nodules and several patchy regions of consolidation on the left side. The patient had renal impairment on day five, which manifested as a urea level of 137, severe leucopenia, abnormal P. T., and aPTT. Over three days in the hospital, the patient had extreme drowsiness, was experiencing oxygen saturation levels below normal, and ultimately required intubation. The patient was found to have significant hypoxemia, according to the results of the arterial blood gas analysis. A probable clinical diagnosis of sHLH was established on day 7 when the USG abdomen revealed hepatosplenomegaly, while BRA, CBC, and urine revealed lymphopenia, anemia, and thrombocytopenia. Following the execution of the methodology, the patient's elevated ferritin and triglyceride levels were verified by the bone marrow biopsy. Intravenous infusions of methyl pred and dexamethasone were started. Following that, DIC was used to do a serial complete blood count, and Fresh Frozen Plasma was used to treat the clotting profile. On the fifth day of ventilator support, the patient had a cardiac arrest and passed away.

Case Report 2

A 57 - year - old male patient with a history of coronary artery disease presented to the hospital with a 4 - day history of intermittent fever, chills, rigors, nausea, vomiting, abdominal distention, shortness of breath, and yellowing of the eyes. The patient had been experiencing these symptoms for the past three days. The patient was restless, feverish, and tachypneic on admission day. The patient was discovered to be icteric, with an unrecordable blood pressure and the presence of PR - tachycardia. Creps were detected during auscultation. Ionotropic support was initiated for the patient, and empirical intravenous antibiotics doxycycline and 2 grams of ceftriaxone were administered. The entire blood picture and first blood examination both showed normal white blood cell counts with thrombocytopenia. As a result of a cholestatic pattern of increase, total bilirubin was elevated to 17.3, while AST and ALT were determined to be within normal ranges. Also, creatinine was 5.7, and urea was 370. Ultrasound of the abdomen and chest (USG) revealed moderate hepatic steatosis as part of the diagnostic imaging workup. A positive result for Lepto IgM was detected in the fever profile. The patient was released from the hospital on

the fifth day of treatment due to an improvement in his clinical presentation.

Case Report 3

The patient, a 31 - year - old woman, came to the emergency room complaining of left - sided radiating discomfort to her left shoulder along with sweat and chest pain that had persisted for one day. One week ago, the patient had a high temperature without fluids. The patient's vital signs were steady on the day of arrival. During the first assessment, the electrocardiogram (ECG) showed a significant increase in cardiac biomarkers and a broad S. T. elevation. There was a little increase in aspartate aminotransferase, but otherwise, the CBC was normal. The electrocardiogram revealed hypokinesia in the anteroseptal area. Coronary angiography was performed due to persistent chest discomfort and showed normal coronary arteries. It was determined that Leptospirosis had occurred by a microscopic agglutination test.

Her clinical and electrocardiogram (ECG) results improved after receiving intravenous ceftriaxone.

2. Discussion

A patient is suspected of having Leptospirosis if they have had a fever (>380 C) with two or more of the following symptoms: headache, myalgia, jaundice, arthralgia, conjunctivitis, vomiting, diarrhea, back pain, reticular pain, or rash in the last three weeks. Factors related to epidemiology are also taken into account, such as being in close proximity to diseased animals or rodents, being in an area with heavy seasonal rainfall, and working in potentially dangerous jobs like garbage collection, cleaning streams or streets, raising livestock, butchering, or farming (e. g., rice farming).

The most reliable method of diagnosis is the serological microscopic agglutination test, often known as MAT. Finding serovars of epidemiological significance is the key benefit of MAT.[17]

Early involvement of a panel of subspecialties, intensified investigations and monitoring, targeted therapy with corticosteroids and immunoglobulin, and a high index of suspicion for sHLH are warranted in leptospirosis patients who do not respond to optimal antibiotics, progress rapidly, and have multiorgan dysfunction. These measures may change the outcome of the patient's condition. Fever and multiorgan failure are common symptoms of secondary HLH, making it more difficult to identify. Endemic infections have a significant role as initiators.
The majority of cases of HLH in adults are secondary HLH, which may be caused by viral, autoimmune/inflammatory, or cancerous diseases [18].

Infections, cancers, rheumatologic diseases, pregnancies, and medications are the most common causes of secondary HLH, which is also known as sHLH, acquired HLH, or reactive HLH.1[9] Although infectious triggers may cause bacterial, fungal, or parasitic pathogenesis, viral infections (especially CMV and EBV) are more common. In nations in East and South Asia, sHLH is often linked to bacteria that do not have a full range of symptoms.2[0]

References

[1] Rajapakse S. Leptospirosis: clinical aspects. Clin Med (Lond).2022; 22: 14 - 7.
[2] SY Kocak, K Aysegul, A Kayalar et al. Leptospirosis With Acute Renal Failure and Vasculitis: A Case Report. Arch Rheumatol, 2019; 34 (2): 229.
[3] Fentahun T and Alemayehu M. Leptospirosis and its public health significance: a review. Eur J Appl Sci, 4 (6), 2012; 238 - 44.
[4] Rajapakse S. Leptospirosis: clinical aspects. Clin Med (Lond).2022, 22: 14 - 7
[5] Faines S. Guidelines for control of Leptospirosis. Geneva, World Health Organisation offsetPublication 1982; 67.
[6] John T J. Emerging and re - emerging bacterial pathogens in India. Indian J Med Res 1996; 103: 4 - 18. and Leptospirosis. India. Report of the investigation of a post - cyclone outbreak in Orissa. November, 1999. Wkly Epidemiol Rec 2000; 75: 217).
[7] Saha R, Sil S, Thakurta RG. Incidence of Leptospirosis in India: A cross - sectional study. Eur J Pharm Med Res.2016; 3 (7): 502 - 04].
[8] Chaudhry R, Das A, Premlatha MM, et al. Serological & molecular approaches for diagnosis of Leptospirosis in a tertiary care hospital in north India: A 10 - year study. Indian J Med Res.2013; 137 (4): 785.
[9] Petakh P, Isevych V, Kamyshnyi A, Oksenych V. Weil's disease - immunopathogenesis, multiple organ failure and potential role of gut microbiota. Biomolecules.2022, 12: 1830, and Marshall RB. The route of entry of leptospires into the kidney tubule. J Med Microbiol.1976; 9: 149 - 52.
[10] La Rosée P, Horne A, Hines M et al. Recommendations for the management of hemophagocytic lymphohistiocytosis in adults. Blood 2019; 133 (23): 2465–2477.
[11] Rajagopala S, Singh N, Agarwal R et al. Severe hemophagocytic lymphohistiocytosis in adults - experience from an intensive care unit in North India. Indian Journal of Critical Care Medicine: Peer - Reviewed, Official Publication of Indian Society of Critical Care Medicine, 2012; 16 (4): pp.198–203.
[12] Munasinghe BM, Arambepola AG, Pathirag N et al. Case Report Haemophagocytic Lymphohistiocytosis with Leptospirosis: A Rare but Devastating Complication. Case Reports in Infectious Diseases, 2021.
[13] Philip, N.; Affendy, N. B.; Masri, S. N.; Yuhana, M. Y.; Than, L. T. L.; Sekawi, Z.; Neela, V. K. Combined PCR, and MAT improves the early diagnosis of the biphasic illness leptospirosis. PLoS ONE 2020, 15, e0239069
[14] Chakraborty, A.; Miyahara, S.; Villanueva, S. Y.; Saito, M.; Gloriani, N. G.; Yoshida, S. - I. A novel combination of selective agents for isolation of Leptospira species. Microbiol. Immunol.2011, 55, 494–501.

[15] Hornsby, R. L.; Alt, D. P.; Nally, J. E. Isolation and propagation of leptospires at 37 ◦C directly from the mammalian host. Sci. Rep.2020, 10, 9620.
[16] Ellis, W. A.; Montgomery, J.; Cassells, J. A. Dihydrostreptomycin treatment of bovine carriers of Leptospira interrogans serovar hardjo. Res. Vet. Sci.1985, 39, 292–295
[17] Shivakumar S, Krishnakumar B. Diagnosis of Leptospirosis - Role of MAT. Assoc Physicians India.2006; 54: 338 - 339.
[18] La Rosee P, Horne A, Hines M, et al. Recommendations for the management of hemophagocytic lymphohistiocytosis in adults. Blood 2019; 133 (23): 2465–2477.
[19] Yildiz H, Neste EVD, Defour JP et al. Adult haemophagocytic lymphohistiocytosis: a review. QJM: An International Journal of Medicine, 2020.
[20] Wong KF and Chan JKC. Reactive hemophagocytic syndrome clinicopathologic study of 40 patients in an oriental population. American Journal of Medicine, 1992; 93 (2): pp.177–180.