

A Case Series of Leptospirosis

Dr. Kaverikannan .R MD¹, Dr. Vignesh S²

Abstract: *Leptospirosis is a zoonotic disease caused by bacteria belonging to the genus Leptospira. It is transmitted through direct contact with urine, blood, or infected tissue of animals. Leptospira species are highly motile, coiled, thin, helical organisms with periplasmic flagella. They stain poorly but can be visualized using dark field microscopy. Leptospirosis has a wide range of manifestations, from asymptomatic illness to severe complications. The classification of Leptospira species based on serology is valuable for clinical, epidemiological, and diagnostic purposes. This article presents a case series discussing various clinical presentations of leptospirosis. The cases include diffuse alveolar hemorrhage, necrotizing pancreatitis, acute myocarditis, Guillain-Barré syndrome, and sepsis with multiorgan failure. Each case describes the patient's clinical features, laboratory findings, imaging studies, and treatment outcomes. The first case involves a young female with fever, breathlessness, and hemoptysis, diagnosed with leptospirosis-associated diffuse alveolar hemorrhage. The second case highlights an agricultural worker presenting with fever, abdominal pain, and necrotizing pancreatitis. The third case presents a male patient with fever, chest discomfort, and acute myocarditis. The fourth case describes a male with weakness in limbs progressing to Guillain-Barré syndrome. The fifth case involves a dehydrated male farmer with sepsis and multiorgan dysfunction syndrome. Leptospirosis can present with a wide spectrum of manifestations, and early diagnosis is crucial for appropriate management. Laboratory investigations, including serological tests, can aid in confirming the diagnosis. Treatment involves antibiotics, supportive care, and specific interventions based on the organ involvement. Prompt intervention can prevent further organ damage and improve outcomes in severe cases of leptospirosis. In conclusion, this case series provides valuable insights into the diverse clinical presentations of leptospirosis. Increased awareness and early recognition of the disease can aid in timely diagnosis and appropriate management, ultimately reducing morbidity and mortality associated with this infectious disease.*

Keywords: Leptospira, Spirochaetales, Leptospiraceae, L. interrogans, L. biflexa, Pathogenic leptospira, Serovars, Serogroups, Clinical manifestations, Diffuse alveolar hemorrhage, Necrotizing pancreatitis, Acute myocarditis, Guillain-Barre syndrome, Sepsis with multiorgan failure, Diagnosis, Epidemiology, Diagnostic tests, Hemoptysis, Radiological manifestations, Treatment, Antibiotics, Fluid management, Hemodialysis, Complications, Mortality, Public health

1. Introduction

Leptospira species belong to order spirochaetales, and family leptospiraceae. The genus leptospira contains two species -L. interrogans and L. biflexa. Twenty two leptospira species have been described with 10 pathogenic species, 5 intermediate, 7 nonpathogenic species. classification based on serology better serves clinical, epidemiological and diagnostic purposes. Pathogenic leptospira are divided into serovars based on antigenic classification. More than 250 serovars and 26 serogroups have identified. Leptospira are coiled, thin, helicate highly motile organisms with hooked ends and periplasmic flagella. They stain poorly, but can be seen by dark field microscopy. Leptospira requires special media for growth. Transmitted by direct contact with urine, blood or infected tissue of infected animal. Survive in humid environment, hence contaminated water is important source of infection. Vast majority of infections range from asymptomatic illness to fulminant complications. Leptospirosis is also a traveler's disease, who acquires infections from tropical countries. In this series, wide manifestations of leptospirosis have been documented

2. Objectives

We are going to discuss about the different case presentation of leptospirosis. They are diffuse alveolar hemorrhage, necrotising pancreatitis, acute myocarditis, Guillain barre syndrome, sepsis with multiorgan failure

Case 1

A 18 year old female, college going student admitted with complaints of fever for 3 days and breathlessness since 1 hour and one episode of hemoptysis. She had a no previous

medical history. Physical examination revealed high body temperature (38.4degree celcius), patient is dyspneic and tachypneic Her respiratory rate was 30/min, oxygen saturation was 90% with room air. Chest xray showed, bilateral diffuse opacification pattern, CT shows-bilateral perihilar and central ground glassing. Arterial blood gas analysis at room air revealed a Ph of 7.35, Pco2 of 40mmhg, spo2-90. Laboratory investigation revealed hemoglobin-10 concentration. 2, total wbc count-11100, platelet-98000, urea-35, creatinine-1.0, total bilirubin-0.6, liver enzymes were normal. leptospirosis IgM were positive with 26.15 units. dengue and mp/mf were negative. in view of covid pandemic rt pcr for covid were done-negative. Prothrombin time (PT)-13.6 (9.8-12.8) mild degree of coagulopathy, INR-1.2, aPTT (activated) thromboplastin time)-31, sputum investigations were done.

At admission for 2 hours her hypoxemia progressively worsened. she was started on intravenous antibiotics & corticosteroids and mechanical ventilation with continue positive end expiratory pressure and fresh frozen plasma in view of mild coagulopathy. After two days patient symptomatically improved and weaned from cpap and started on nasal oxygen. her coagulopathy also gets corrected her platelet also improved. Finally she weaned off from Oxygen. In the present report, we report a case of leptospirosis with diffuse alveolar hemorrhage. Her life threatening condition was successfully treated with corticosteroids and respiratory support. leptospirosis should be considered when patients presents with fever and pulmonary manifestation. Pulmonary manifestations was found to occur independently without renal and hepatic impairments.

Case 2:

A 55 year female, agricultural worker by occupation

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presented with complaints of fever and chills for 1 week, abdominal pain for 3 days. On examination pt conscious, oriented, no pallor, no icterus, no pedal oedema, no generalized lymphadenopathy. Her vitals bp 100/70 mmHg, pr 90/min spo₂ 98 in room air. CVS s1s2+, RS b/l entry +, nvbs, per abdomen-soft, tenderness present in epigastrium and umbilical region, bowel sounds heard, her blood investigations showed hb 10.5, total counts 12,500, differential counts -75% neutrophils, 23% lymphocytes, 2% eosinophils, haematocrit 31, platelets 76,000, urea 25, creatinine 0.9 and total bilirubin 1.1, direct bilirubin 0.5, indirect bilirubin 0.6, AST 90, ALT 78, with serum amylase 1402 serum lipase 405. Her chest x ray normal study, x ray abdomen erect normal study with no features suggestive of perforation. Her ct abdomen with contrast showed necrosis of both pancreatic parenchyma and peri pancreatic tissues with peri pancreatic fluid collections. Her serial blood reports showed thrombocytopenia and raised renal parameters with urea 65 creatinine 4.8. Due to her occupation and high index of suspicion Igm leptospira sent showed high titres of 55 units. She was treated with iv fluids, iv higher antibiotics, analgesia, and other supportive measures. Started on intermittent hemodialysis. Later, she underwent surgical management for necrotizing pancreatitis. Patient condition improved after effective case management.

Case 3

A 45 year male presented with history of fever on and off for 10 days, chest discomfort for 5 days. On examination pt is conscious, oriented dyspneic, tachypneic. cvs s1s2+ rsb/l air entry+, nvbs, bilateral crepitation present, per abdomen soft, CNS no focal neurological deficits, no neck stiffness bilateral crepitations+. His vitals bp 150/70 mmHg, pr 100/min spo₂ 93 in room air. Blood reports showed elevated total counts, platelets 1.2 lakh, with normal renal and liver parameters. His ecg showed diffuse ST-T changes with no reciprocal changes. His cpk 355, cpk MB 195. ECHO showed no regional wall motion abnormality. His chest x ray showed bilateral perihilar opacities. CT chest revealed features suggestive of pulmonary oedema. A diagnosis of acute myocarditis suspected. On searching for underlying etiology leptospiral IgM positive with significant titre (20) pt treated with intravenous steroids, iv antibiotics, iv fluids and other supportive measures. pt improved well with

Case 4

A 37 year male presented with complaints of weakness in both lower limbs for 5 days, which is progressive with weakness in both upper limbs for 1 day. He had a history of fever before 14 days lasting for 3 days, for which he took treatment from nearby clinic, and not turned up for further checkups. On examination patient is mild tachypneic. no pallor, icteric +, no generalized lymphadenopathy, no pedal oedema. cvs s1s2+ rs b/l entry + nvbs, per abdomen soft, CNS-hypotonia in all four limbs, power in both lower limbs 2/5; power in both upper limbs 3/5; with are flexia both upper limbs and lower limbs, with no sensory involvement and no involvement of bowel and bladder. His blood reports hb 13.5, total counts 12,450, differential counts-70 neutrophils, 35 lymphocytes, 5 eosinophils, haematocrit 32.5, platelet 55,000. Renal parameters deranged with urea 77, creatinine 3.5. mri cervical spine with whole spine screening normal, mri brain normal study.

CSF analysis showed elevated protein 98 mg/dl, with cell count 5-7 cells. (albuminocytologic dissociation). His serial renal parameters worsened. Investigation for fever workup showed leptospiral igm positivity with significant titre 25 units. patient treated with iv higher antibiotics, iv fluids, other supportive measures. Started on intermittent haemodialysis for acute kidney injury. iv immunoglobulin initiated but with no response plasma pheresis initiated with approximately 7 cycles patient neurological condition improved. High index of suspicion needed to identify leptospirosis as a cause for Guillain barre syndrome.

Case 5

A 70 year male, farmer by occupation presented in a drowsy and dehydrated state with fever for 4 days, vomiting for 2 days, reduced urine output since 1 day. on examination pulse feeble with bp-80/50 mmHg. no pallor, icterus+, no generalized lymphadenopathy, no pedal oedema. cvs s1s2+, rs b/l entry + per abdomen soft, no organomegaly, CNS-no focal neurological deficits. Investigations elevated total counts, dearranged liver function test and increased creatinine levels. Pt started on iv fluids, iv higher antibiotics, inotropic support, ursodeoxycholic acid, doxycycline and other supportive measures. pt started on haemodialysis and series of 7 cycles done. after which patient improved. Sepsis presenting as multiorgan dysfunction syndrome in one of the dreaded complications of leptospirosis. Early diagnosis and adequate treatment initiation can prevent the mortality.

3. Discussion

Leptospirosis, a zoonotic disease presents in a wide spectrum of manifestations. Transmission occurs through cuts, abraded skin, mucous membrane. Two phases bacteremic phase and immune phase. Immune phase is associated with the appearance of antibody. Organisms have been isolated in autopsy from heart, lung, kidney, brain, liver. Renal pathology shows both acute tubular damage and interstitial nephritis. Deregulation of several transporters contribute to impaired sodium absorption, tubular potassium wasting, and polyuria. Histopathology of liver shows focal necrosis, foci of inflammation and plugging of bile canaliculi. petechiae and hemorrhages are observed in lung, kidney, brain, adrenal, muscle, prostate, testis, gastrointestinal tract. Several studies showed association between hemorrhage and thrombocytopenia. Platelet consumption have been important mechanism for thrombocytopenia, although other mechanisms are involved. Consumption coagulopathy have been reported. Several studies showed occurrence of disseminated intravascular coagulation (DIC). Endothelial injury by leptospira species have been reported, which is also involved in dissemination. Virulence factors include lipopolysaccharide antigens, outer membrane proteins. Clinical manifestations vary from asymptomatic anicteric illness to acute fulminant complications. Some serovars are associated with more severe disease than others. Mild symptoms include a flu like illness, headache, myalgia, nausea, vomiting, conjunctival suffusion (redness without exudate), muscle pain is typical and involves calves, back and abdomen. Aseptic meningitis is more common in children than adults. The natural course of mild leptospirosis lasts for 7-10 days which is followed by recovery. Severe leptospirosis is associated with a case

fataity rate 1-50%. High mortality rates are associated with age, altered mental status, hypotension, acute renal failure, respiratory insufficiency, arrhythmias. The classic presentation-weils syndrome is characterised by triad of hemorrhage, jaundice, acute renal failure. Pulmonary hemorrhage is now recognised as a widespread public problem. Acute kidney injury is common in severe leptospirosis, which presents after several days of illness that can be nonoliguric or oliguric. Typical electrolyte abnormality includes hypokalemia, hyponatremia. Loss of magnesium in urine is uniquely associated with leptospiral nephropathy. Hypotension is associated with acute tubular necrosis, oliguria, and anuria. leptospiral meningitis can be missed, since some presents in anicteric state and not associated with any clinical manifestations. Neurologic sequelae are described until months after acute illness. Other manifestations include necrotising pancreatitis, cholecystitis, skeletal muscle involvement, rhabdomyolysis, myocarditis, arrhythmias. Rare hematological manifestations include hemolysis, thrombotic thrombocytopenic purpura (TTP), hemolytic uremic syndrome (HUS). Autoimmune associated uveitis is a well recognized sequale of leptospirosis

The clinical diagnosis of leptospirosis should be based on appropriate exposure history and the clinical manifestation. Biochemical investigations are non specific with leucocytosis, elevated acute phase reactants. Total bilirubin elevated grossly with amino transferases and alkaline phosphatases levels moderate rise. the most common radiological manifestations scattered alveolar hemorrhage predominately affecting lower lobes, pleura based opacities, diffuse ground glass opacities typical of acute respiratory distress syndrome (ARDS)

Definitive diagnosis of leptospirosis is based on isolation of organisms from culture, PCR positive result or seroconversion or fourfold rise in titres. Microscopic agglutination tests (MAT) and ELISA are the standard serological tests for leptospirosis. MAT is useful for epidemiological studies.

Dual or mixed infections are common. This includes coinfection with other viral fevers like dengue fever, chiknunya fever, rickettsia, malaria and other viral hemorrhagic fevers. Therefore it is advisable, to conduct serological tests for rickettsia, dengue virus, hanta virus when leptospirosis is detected.

Treatment for severe leptospirosis iv penicillin. Other antibiotics like cephalosporins, macrolides, aminoglycosides can be used. Early initiation of antibiotics minimizes the mortality from organ damage. Azithromycin or doxycycline is drug of choice in areas where rickettsia infections are endemic. Patients with non oliguric renal dysfunctions require aggressive fluid requirement to prevent oliguric renal failure. Rapid initiation of hemodialysis is associated with reduced mortality and require only for short periods. Patients with pulmonary hemorrhage have reduced lung compliance which may benefit from mechanical ventilation with low tidal volume. The use of steroids, desmopressin as an adjunct for pulmonary hemorrhage is contradictory.

4. Conclusion

Early intervention can prevent development of major organ system failure in leptospirosis. Antibiotics are less likely to benefit, when organ damage occurred. This case series reported a wide spectrum of manifestations in leptospirosis, which will be useful for earlier diagnosis.

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