

Severe Leptospirosis and Co-infections in North India: A Case Series of Paediatric and Adult ICU Patients Highlighting Diagnostic Challenges

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Abstract

Background: Leptospirosis, a zoonotic disease of global significance, is endemic in India and often presents as an acute febrile illness that can be difficult to distinguish from other tropical infections like enteric fever. Co-infections are increasingly recognized but pose a significant diagnostic and therapeutic challenge, potentially leading to severe complications and fatal outcomes, especially when diagnosis is delayed. **Methods:** This case series analyses six patients (ages 8–55 years) presenting to a tertiary care centre with severe febrile illness and diagnosed with leptospirosis, either alone or in combination with other pathogens (enteric fever, Hepatitis A). Diagnosis was confirmed through serology (Leptospira IgM ELISA), PCR, blood culture, and ancillary testing. Clinical presentation, laboratory parameters, management, and outcomes were meticulously reviewed. **Results:** The series highlights diverse and severe manifestations of leptospirosis, including Weil's syndrome with hepatic encephalopathy (Case 1), fatal ARDS with refractory shock (Case 2), and co-infection with hepatitis A (Case 3). Leptospirosis was also identified as a cause of acute decompensation in chronic liver disease (Case 4). Other presentations included reactive arthritis preceding fever (Case 5) and leptospiral encephalopathy with ARDS (Case 6). A critical finding was the presence of leptospirosis and enteric fever co-infection in four of the six cases. Thrombocytopenia, transaminitis, and elevated CRP were consistent laboratory features. Empiric treatment with doxycycline, initiated upon suspicion of zoonotic exposure or lack of response to first-line antibiotics, was associated with rapid clinical improvement in surviving cases. The fatal outcome (case 2) highlights that a delay in initiating appropriate antibiotic coverage for leptospirosis, often due to its non-specific early symptoms and overlapping presentation with other fevers, can rapidly progress to irreversible, life-threatening complications. **Conclusion:** This series highlights the high prevalence and clinical significance of leptospirosis and its co-infections, particularly with enteric fever, in endemic regions. A high index of suspicion is crucial in patients presenting with febrile illness, haematological abnormalities, and multi-organ dysfunction. History of rodent or animal exposure should be actively sought. Empirical doxycycline coverage for leptospirosis is recommended alongside standard therapy when co-infection is suspected, as prompt dual-pathogen treatment can be life-saving. Enhanced surveillance, improved diagnostics, and rodent control are essential public health measures.

Keywords: Leptospirosis, Enteric Fever, Co-infection, Zoonotic Disease, Weil's Disease, Doxycycline, ARDS, Multi-organ Failure.

Introduction

Leptospirosis is a zoonotic disease caused by *Leptospira* bacteria, which are spread through water or soil contaminated by the urine of infected animals, especially rodents.¹ Humans contract the infection through cuts or mucous membranes. The bacteria enter the bloodstream, damaging organs such as the liver, kidneys, and lungs, and can cause severe complications, including haemorrhage and organ failure. Leptospirosis causes an estimated 1.03 million cases and 58,900 deaths globally each year.² High-risk occupations include farming, sewer work, and veterinary medicine. Severe infections can result in jaundice, pulmonary hemorrhage, and multi-organ failure.³⁻⁴ Preventive measures like rodent control and protective equipment are essential for reducing transmission.⁵ Leptospirosis is significantly underdiagnosed in India.⁶ In Haryana, 30% of febrile patients tested positive for leptospirosis, with the highest prevalence among young adults and during the monsoon season.⁷ Co-infections with dengue and typhoid are frequently observed. Severe cases may require advanced treatments like plasma exchange.⁸ Research by Dr. N. Rathnapriya⁹ emphasizes the critical importance of investigating co-infections—particularly enteric fever—in leptospirosis patients who show poor response to therapy. These findings underscore the urgent need for enhanced surveillance, improved diagnostic capabilities, and targeted prevention strategies to address the substantial burden of leptospirosis and its co-infections in India.

Case 1- Severe leptospirosis with Weil's reaction, enteric fever coinfection, hepatic encephalopathy stage 1, anasarca, and shock

A 9-year-old female with normal anthropometric indices came with complaints of fever for 5 days, abdominal pain for 3 days, yellowish discoloration of eyes and high colored urine for 2 days, altered sensorium, and loose stools for 1 day. On admission child was sick with a confused mental status and GCS of 10/15 (E₃V₃M₄), tachycardia of 120/min, cold peripheries, feeble pulses, and prolonged CRT, BP-100/57 (Both SBP and DBP more than 50th centile), temperature of 97.2 degrees Fahrenheit. Following this IV fluid bolus was given. Respiratory rate of 32/min, abdominothoracic, with a saturation of 98% on 8 litres of oxygen via mask. Icterus was visible in the eyes. Given persistent signs of hemodynamic instability, inotropic support was also added, keeping the possibility of septic shock.

In the per abdomen examination, the abdomen was distended with tenderness in the epigastric and right hypochondrium, with the right lobe of liver 7 cm below the costal margin, firm margins with normal surface and a span of 11 cm, spleen 3.5 cm below the costal margin, and shifting dullness was present. In CNS examination, the child was lethargic and in stage 1 encephalopathy with a GCS of E₃V₃M₄, Brisk DTR, Extensor plantar, normal tone, and power of 3/5 as per MRC classification. Optic nerve sheath diameter showed early signs of raised ICT with borderline raised diameter in both eyes as

per age (4.6 and 4.8 mm). The respiratory system showed decreased air entry on the right side.

Initial investigations revealed thrombocytopenia (76,000/ μ L) and cholestasis, with elevated CRP, polymorphonuclear leukocytosis, direct hyperbilirubinemia, deranged transaminases (SGOT/SGPT), hypoalbuminemia (1.9 g/dL), and elevated urea (**Table 1**). Imaging demonstrated a right-sided pleural effusion on chest X-ray, while abdominal USG revealed gallbladder wall sludge, mild hepatic echotexture changes, and ascites.

The child was managed for septic shock with enteric encephalopathy, receiving fluid resuscitation, inotropic support, 3% saline for possible cerebral edema, and broad-spectrum antibiotics. N-acetylcysteine was added due to acute liver dysfunction, ascites, and encephalopathy. Given worsening hypoalbuminemia (1.9 g/dL) and generalized anasarca, 20% albumin infusion was administered, followed by packed red blood cell transfusion (Hb: 6.3 g/dL) for anemia.

Given the child's disproportionately severe illness relative to the short history, further history-taking revealed significant rodent exposure in the household kitchen, raising suspicion for leptospirosis. *Leptospira* PCR was sent, and IV doxycycline was empirically initiated on day 2 of admission.

The child showed progressive clinical improvement by days 4–5, with normalization of GCS, resolution of respiratory distress, and successful weaning off oxygen support. Confirmatory testing returned positive for *Leptospira* spp. by PCR, while blood culture grew *Salmonella* spp., indicating a concurrent (mixed) infection.

The child completed 14 days of IV antibiotics (covering *Salmonella*) and 7 days of IV doxycycline (for leptospirosis), with resolution of ascites, effusions, and hepatosplenomegaly by discharge. The child was discharged in stable condition with no residual deficits. This case highlights the importance of considering mixed infections (leptospirosis and enteric fever) in children presenting with septic shock, hepatic dysfunction, and encephalopathy, particularly in endemic areas with rodent exposure, where prompt diagnosis and dual antibiotic therapy can lead to complete recovery.

Case 2- Severe Leptospirosis with enteric fever, Acute respiratory distress syndrome (ARDS), respiratory failure, and refractory shock.

A 32-year-old female from Gurgaon presented with 5 days of fever with chills/rigors and 3 days of progressive dyspnea, having failed outpatient treatment. Initial labs showed leucopenia (TLC 3220/cumm), thrombocytopenia (98,000/ μ L), and positive Typhidot IgM (>1:160). On admission, she was in NYHA class IV orthopnea, pulse rate of 124/min, low volume and feeble pulse with cold peripheries, RR 44/min, abdominothoracic, temperature of 98 degrees Fahrenheit, hypotensive shock (BP 90/54), profound hypoxemia (SpO₂ 54%), and altered sensorium (GCS E2V3M4). Chest examination

revealed bilateral crepitus and reduced air entry. Per abdomen, soft and distended with hepatomegaly and liver span of 19 cm. CNS- GCS was E2V3M4 with normal tone/power and reflexes. Despite immediate intubation, aggressive fluid resuscitation, ionotropic support, and empirical antibiotics (doxycycline/ceftriaxone), her condition rapidly deteriorated. Confirmatory tests revealed concurrent leptospirosis (IgM+) and typhoid fever (IgM+), with CXR showing bilateral diffuse haziness and USG demonstrating pleural effusion. The patient expired within 4 hours of admission, illustrating the lethal potential of co-infections in endemic regions when presenting with multi-organ failure. This case highlights the critical importance of early recognition and aggressive management of zoonotic infections in febrile patients with haematological abnormalities and respiratory distress.

Case 3-Leptospirosis with enteric fever and viral hepatitis A coinfection.

A 22-year-old male presented to our outpatient department with a 3-week history of fever, abdominal pain, decreased appetite, nausea, and weight loss, followed by one day of haematochezia. Prior investigations had revealed a positive Typhi dot IgM, and the patient had been on oral cefixime for 5-7 days without clinical improvement. On admission, vital signs were stable with normal temperature (98°F), pulse (76/min), good volume, blood pressure (114/80 mmHg), and oxygen saturation (98% on room air). Physical examination demonstrated diffuse abdominal tenderness with otherwise normal cardiovascular, respiratory, and neurological findings. Initial management included intravenous ceftriaxone and azithromycin for suspected drug-resistant enteric fever. However, azithromycin was discontinued after 24 hours due to worsening liver function tests, and further testing was pursued for persistent fever. Subsequent investigations revealed concurrent infections with positive *Leptospira* IgM/IgG and Hepatitis A IgM serologies. Abdominal ultrasound showed interbowel fluid with increased echogenicity. The patient showed marked clinical improvement after initiating oral doxycycline for leptospirosis, with resolution of fever within 48 hours and no further hematochezia. After completing a 7-day antibiotic course and receiving dietary hygiene counseling, the patient was discharged in stable condition. This case highlights the diagnostic challenges in febrile illnesses with gastrointestinal manifestations in endemic regions. It emphasizes the importance of comprehensive serological evaluation when patients fail to respond to initial therapy.

Case 4- Decompensated Chronic liver disease with portal hypertension, spontaneous bacterial peritonitis, leptospirosis and enteric fever

A 41-year-old male with known alcohol-induced chronic liver disease and portal hypertension (on carvedilol 3.125 mg BD, spironolactone 25 mg OD, and ursodeoxycholic

acid 300 mg OD) presented with 4-5 days of abdominal distension, pain, and vomiting. On examination, he was hemodynamically stable with a pulse of 76/min, good volume, blood pressure of 100/56 mmHg, and oxygen saturation of 96% on room air. Physical findings included bilateral pedal edema, flapping tremors, mild pallor, and massive ascites with positive fluid thrill. Abdominal examination revealed a liver span of 10 cm, spleen 3 cm below the costal margins, smooth surface with firm consistency, with positive fluid thrill and diffuse tenderness. While cardiovascular and neurological examinations were unremarkable, respiratory assessment demonstrated decreased air entry on the left side.

Initial management included empirical intravenous cefotaxime for suspected spontaneous bacterial peritonitis, with a diagnostic ascitic tap performed. Subsequent investigations revealed positive *Leptospira* IgM antibodies, prompting the addition of doxycycline to the treatment regimen. Abdominal CT confirmed cirrhotic liver morphology with portal hypertension, peri-splenic collaterals, and gross ascites. The patient showed clinical improvement with the resolution of marked ascites and was subsequently discharged.

This case underscores the diagnostic challenge of acute decompensation in chronic liver disease patients and highlights leptospirosis as an important differential diagnosis that can exacerbate pre-existing hepatic dysfunction. The presentation emphasizes the need for a comprehensive infectious workup in cirrhotic patients presenting with acute clinical deterioration, particularly in endemic regions.

Case 5- Culture-positive enteric fever with leptospirosis co-infection with reactive arthritis

An 8-year-old girl with normal growth parameters presented with a 10-day history of bilateral knee pain followed by 4 days of high-grade fever and 2 days of upper respiratory symptoms (dry cough and coryza). On examination, she was febrile (105°F) with tachycardia (130 bpm), tachypnoea (28 breaths/min), and normal blood pressure (106/66 mmHg) for her age. Systemic examination revealed hepatomegaly (2 cm below the costal margin, span 8.5 cm) with otherwise normal cardiorespiratory and neurological findings (GCS 15/15).

Initial management with ceftriaxone (for Typhi dot IgM-positive enteric fever) failed to control persistent fever spikes, which were subsequently accompanied by diarrhoea and vomiting. Expanded history revealed cattle exposure at home, prompting leptospirosis testing (serology positive). The addition of doxycycline resulted in clinical improvement within 48 hours, with resolution of fever. Blood cultures later confirmed *Salmonella* Typhi infection. The child completed a 7-day course of doxycycline and a 14-day course of ceftriaxone and was discharged in stable condition.

Case 6- Leptospiral encephalopathy with ARDS and type 1 respiratory failure.

A 55-year-old male with a history of chronic smoking and alcohol use presented to the emergency department with a 2–4-day history of undocumented fever, accompanied by breathlessness and altered sensorium for the past 24 hours. His symptoms progressed to include slurred speech for 6 hours, followed by episodes of tongue biting and uprolling of the eyeballs.

On presentation, the patient was critically ill. His vitals were: pulse of 112/min (regular, good volume), respiratory rate of 40/min (abdominothoracic pattern), oxygen saturation of 84% on a 10L/min non-rebreathing mask, random blood sugar of 136 mg/dL, temperature of 100.8°F, and blood pressure of 102/72 mmHg.

A neurological examination revealed a drowsy patient with a Glasgow Coma Scale (GCS) of 10/15 (E4V2M4). There was no meningismus, and deep tendon reflexes were normal. Respiratory examination revealed bilateral crackles, more prominent on the left side, with bilateral reduced air entry. The cardiovascular and abdominal examinations were unremarkable except for sinus tachycardia.

The patient was immediately initiated on non-invasive mechanical ventilation for type 1 respiratory failure. Empirical treatment with intravenous ceftriaxone and levetiracetam was started, and a full panel of investigations for tropical fevers was sent.

HRCT of the thorax revealed bilateral ground-glass opacities with pleural effusions. Lumbar puncture showed an acellular fluid with elevated protein levels. A contrast-enhanced MRI of the brain was normal except for age-related atrophic changes.

Subsequent serological testing returned positive for *Leptospira*. In light of this diagnosis and his poor clinical response to cephalosporins, the antibiotic regimen was upgraded to include intravenous doxycycline and a broader-spectrum coverage with Meropenem.

After 48 hours of intensified treatment, the patient's condition began to improve. His ventilatory requirements decreased, along with a resolution of his respiratory distress and an improvement in his GCS. He was successfully weaned off ventilatory support after 7 days. He completed a 10-day course of antibiotics and was subsequently discharged in stable condition.

Discussion

Leptospirosis is a globally significant zoonotic disease caused by pathogenic *Leptospira* species. Humans acquire infection through exposure to contaminated water, soil, or animal tissues. Its nonspecific clinical presentation often mimics other tropical fevers like dengue and malaria, leading to frequent misdiagnosis. Delayed treatment increases the risk of severe complications, including renal failure, pulmonary hemorrhage, and multi-organ dysfunction. Early antibiotic therapy is critical to reducing morbidity and mortality ¹⁰

Case 1 highlights the diagnostic complexity of mixed infections (*Leptospira* and *Salmonella*) in a child presenting with septic shock, hepatic dysfunction, and encephalopathy. Rodent exposure history and multi-organ involvement (cholestasis, thrombocytopenia, ascites) prompted early empirical doxycycline alongside broad-spectrum antibiotics, later confirmed by PCR and blood culture. The dramatic response to dual therapy underscores the need for high clinical suspicion of zoonotic co-infections in endemic areas, where timely intervention can prevent fatal outcomes despite severe initial presentation. Rodent exposure in household kitchens poses a significant risk for paediatric leptospirosis, as contaminated surfaces or food storage areas can transmit *Leptospira* through urine. Children are particularly vulnerable due to frequent hand-to-mouth contact and proximity to floors where rodents may leave infected urine. This case underscores the importance of rodent control and hygiene awareness in homes to prevent zoonotic disease transmission.¹¹

This fatal leptospirosis-typhoid coinfection in case 2 illustrates how delayed diagnosis of zoonotic pathogens in febrile shock with ARDS can prove lethal. Thrombocytopenia, hepatomegaly, and profound hypoxemia should trigger empirical doxycycline alongside typhoid therapy in endemic regions. The case emphasizes that hemodynamic collapse (BP <90, GCS ≤8, SpO₂ <60%) in co-infections demands immediate dual-pathogen coverage to prevent irreversible multi-organ failure. Leptospirosis-induced ARDS has high mortality; radiological changes often precede clinical symptoms. Early high-dose steroids (administered within 12 hours of pulmonary involvement) and strict ARDS management (e.g., lung-protective ventilation) are crucial. The day 3 PaO₂/FiO₂ ratio predicts outcomes—aggressive rescue therapies (e.g., prone ventilation, ECMO) should be considered for ratios <150.¹²

Case 3 underscores that in endemic regions, a single positive serology can be misleading. The patient's lack of improvement on enteric fever treatment prompted further testing, which identified leptospirosis as the true cause. His rapid recovery on doxycycline highlights the vital importance of re-evaluating the diagnosis when therapy fails. Although hepatitis A and leptospirosis share common risk factors and are highly endemic in regions with poor sanitation, their concurrent infection in individuals is seldom described.¹³⁻¹⁴

Case 4 shows that a patient with chronic liver disease presented with acute decompensation and ascites. Leptospirosis was identified as the underlying cause. Successful treatment with doxycycline highlights the need to consider zoonotic infections in cirrhotic patients presenting with acute decline, particularly in endemic areas. A study investigated whether leptospirosis worsens pre-existing chronic liver disease (CLD) by comparing 31 cirrhotic patients with leptospirosis to 91 otherwise healthy individuals with the infection. The results showed that the cirrhotic patients had a significantly more severe clinical presentation, with much higher rates of jaundice, elevated blood urea, and

elevated serum creatinine. Crucially, there were four deaths due to hepatorenal dysfunction and coagulopathy in the cirrhotic group, while no deaths occurred in the control group. The study concludes that in endemic areas, leptospirosis is an important cause of acute-on-chronic liver failure and is associated with a poor outcome, often leading to fatal hepatorenal dysfunction.¹⁵

Case 5 presented with reactive arthritis (bilateral knee pain) followed by fever. She was diagnosed with a co-infection of typhoid fever and leptospirosis. Her persistent fever only resolved after doxycycline was added for leptospirosis to the ceftriaxone for typhoid. The initial arthritis was likely a reactive manifestation preceding the systemic infections.

Case 6 presented with fever, breathlessness, decreased level of consciousness, and seizures. He was in respiratory failure with ARDS. He was diagnosed with severe leptospirosis. After treatment with IV doxycycline and meropenem, he improved and was discharged. Neurological manifestations in leptospirosis have been reported only in 10–15% of cases, including aseptic meningitis, encephalitis, stroke, myelitis, and acutely flaccid paralysis.¹⁶ Another series from Assam of eight patients presenting with acute encephalitis syndrome (AES) was confirmed as neuro-leptospirosis through IgM ELISA and microscopic agglutination test (MAT) with most prevalent clinical features- fever (100%) and altered sensorium (62.5%).¹⁷

Meningitis is an uncommon but recognized manifestation of leptospirosis, typically associated with its anicteric form. It often presents in two phases: an initial septic phase with viral-like prodromal symptoms, during which *Leptospira* may be detectable in the CSF via culture or PCR, followed by a later immunological phase characterized by meningism and lymphocytic pleocytosis (typically <500 cells/ μ L). Diagnosis in this second phase relies on serological tests. Leptospirosis accounts for approximately 5%–13% of all cases of aseptic meningitis.¹⁸

Another case report from Albania describes a young patient with isolated encephalitis caused by *Leptospira*, where acute encephalitis was the initial presentation of the disease.¹⁹ Rapidly evolving, predominantly peripheral, diffuse nodular or confluent pulmonary lesions are typical of leptospirosis, while pleural effusions are less common, and subsegmental atelectasis is nonspecific.

HRCT revealed extensive ground-glass opacities, primarily in the peripheral and lower lungs, which were found to be caused by airspace haemorrhage upon histological comparison. These abnormalities, including consolidation and nodules, were more extensive on HRCT than on chest radiographs.²

Table 1

Parameter	Case 1	Case 2	Case 3
Hemoglobin	6.3→9.5 gm/dl	10.6 gm/dl	13.4 gm/dl
TLC	8000 cells/cumm	1700 cells/cumm	22370 → 6400 cells/cumm
DLC	Poly-82, lymphocytes-10→P58L36	P71L12EoM2	P82L13EoM5Bo→P55L39E1M5Bo
Platelet count	60,000→3 Lakh/cumm	1.5 lakhs/cumm	4.6 lakh/cumm
Hematocrit	20.2→28%	33.7%	41.8%
CRP	138→5.8 mg/L	96.6 mg/L	38.3 →4 mg/L
T. Bilirubin/Direct Bil/Indirect Bilirubin	10.3/6.9/3.4→2.5/1.7/0.8 mg/dl	0.5/0.3/0.2 mg/dl	2.4/0.9/1.5 →0.5/0.4/0.1 mg/dl
SGOT/SGPT	165/129→40/52 IU/L	208/46 IU/L	671/2458 →33/74 IU/L
ALP	573→263 IU/L	176 IU/L	160 →136 IU/L
T. Protein/S. albumin	3.8/1.9→5.7/3.7 gm/dl	4.8/2.69 gm/dl	6.7/3.75 gm/dl
Urea/creatinine	70/0.4→18/0.5 mg/dl	37/0.84 mg/dl	10 /0.91 mg/dl
Sodium/potassium	127/4.5→137/4.3 mmol/L	139/4.4 mmol/L	136/4.3 mmol/L
Typhifast Igm	Positive	Positive	Positive
Leptospira Igm By Elisa	Positive (PCR Also Positive)	Positive	Positive
Blood Culture	Salmonella	No Growth	Ng

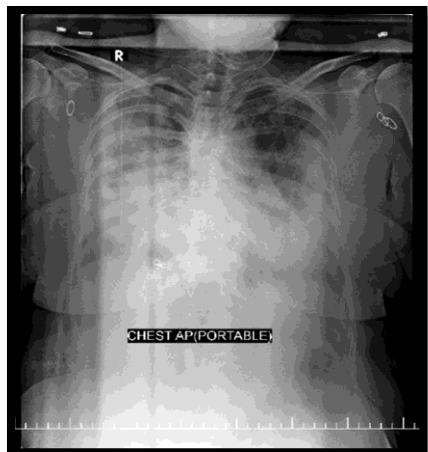
Dengue Nsiag/Igm	Negative	Negative	Negative
Scrub Typhus Igm	Negative	Negative	Negative
Viral Markers	Negative	Negative	Hav Igm Positive
Others	Retic, LDH Normal ANA, DCT, Ceruloplasmin Negative	PCT- 2.9 D-DIMER-800 Ng/ML	PT/APTT/INR-11.3 /29 Sec/o.9

Table 2

Test Name	Case 4	Case 5	Case 6
Haemoglobin	10.2 gm/dl	10.5 gm/dl	10.6 gm/dl
TLC	2680 → 6500 cells/cumm	11430 → 4530 cells/cumm	12500→6300 cells/cumm
DLC	P70L15E3M12Bo→ P55L39E1M5Bo	P79L17EoM4Bo→ P30L60E2M8Bo	P78L20M2→P60L33E1 M6
Platelet count	1.5 lakh/cumm	1.5 lakh/cumm	1.5 lakhs/cumm
Haematocrit	30.7%	32.8%	31.6%
T. Bil/D. Bil/In Direct Bil	6.23/3.34/2.8 →3/1.3/2.7mg/dl	2.4/0.9/1.5 →0.5/0.4/0.1 mg/dl	3.5/2.6/0.9→1.5/0.6/0.9 mg/dl
SGOT/SGPT	60/27→49/22 IU/L	210/168 →75/43 →48/18 IU/L	107/68→83/58 IU/L
ALP	206 →130 IU/L	182	408
Total protein/albumin	7.6/2.60 gm/dl	6.7/3.25 gm/dl	5.16/2.01→6.33/2.78
Urea/Creatinine	15/0.61 mg/dl	14/0.4 mg/dl	53/0.96→30/0.7 mg/dl
Sodium/Potassium	129/4 mmol/L	134/4.1 mmol/L	138/4.8 mmol/L
INR	1.4	1.13	1.03 sec
Leptospira Igm(Elisa)	Positive	Positive	Positive
Viral Markers	Negative	Negative	Negative
Scrub Igm	Negative	Negative	Negative

Crp	48→8 Mg/L	168.6→54→48 →5mg/L	120→5 Mg/L
Ps For Mp	Negative	Negative	Negative
Dengue Ns ₁ /Igm	Negative	Negative	Negative
Hbv/Hcv/Hiv	Negative	Negative	Negative
Typhi Dot Igm/Widal	Positive	Positive	Negative
Blood culture	NG	Salmonella typhi	NG
Ascitic Fluid Tap	Tlc-400 Cells/Cumm Dlc -Poly70 Mono 30% Sugar-136 Mg/Dl Protein-0.85 Gm/Dl Ada-84 U/L Gene Xpert Negative Culture Negative	Nad	Csf- 5 Cells/Cumm 95% Polymorphs 5% Mononuclear Csf Sugar -51 Mg/Dl Protein-75 Mg/Dl Ada-3.7 U/L Dark Field Microscopy, Gram Stain, & Culture-Negative

CASE 2



CASE 4 CT ABDOMEN



CASE 6 HRCT



References

1. Palaniappan RU, Ramanujam S, Chang YF. Leptospirosis: pathogenesis, immunity, and diagnosis. *Curr Opin Infect Dis.* 2007 Jun;20(3):284-92.

2. Costa F, Hagan JE, Calcagno J, Kane M, Torgerson P, Martinez-Silveira MS, et al. Global morbidity and mortality of leptospirosis: a systematic review. *PLoS Negl Trop Dis*. 2015 Sep;9(9):e0003898.
3. Adler B, de la Peña Moctezuma A. **Leptospira** and leptospirosis. *Vet Microbiol*. 2010 Jan;140(3-4):287-96.
4. Madhu G, Anjali V, Girish P, Abhishek TM. A case series and review of severe leptospirosis. *Int J Med Pharm Res*. 2023 Mar-Apr;4(2):564-6. NLM ID: 9918523075206676.
5. Chirathaworn C, Supputtamongkol Y, Lertmaharit S, Poovorawan Y. Cytokine levels as biomarkers for leptospirosis patients. *Cytokine*. 2016;85:80-2.
6. Mishra D, Singh A, Shewale AD, Tiwari S, Nale T. Prevention & Control of Leptospirosis in India: Human Health Initiatives. *Epi-Dis-Phere* (Publication of Health Resilience). 2025 Jan;1(1):[42-7]. Corresponding author: Tiwari S, Joint Director & Head, Centre for One Health, National Centre for Disease Control (NCDC), Delhi. Email: simmi.phs14@gmail.com.
7. Choudhary D, Khandait M, Singh A, Saha R. To study the seroprevalence of *Leptospira* infection at a tertiary care hospital. *Afr J Biol Sci*. 2024 Jul;6(13):4959-71.
8. Davood UB, Kaur P, Gupta A, Tripathi A, Chatterjee AK, Kumar A, et al. Hepatitis A and leptospirosis dual infection-induced fulminant hepatic failure successfully treated with standard volume therapeutic plasma exchange. *Transfus Clin Biol*. 2024 Nov;31(4):265-6.
9. Rejitha IM, Rathnapriya N. Coinfections of leptospirosis with dengue, malaria and enteric fever. *Int J Sci Res*. 2025 Jan;14(1):22-3.
10. Bharti AR, Nally JE, Ricaldi JN, Matthias MA, Diaz MM, Lovett MA, et al. Leptospirosis: a zoonotic disease of global importance. *Lancet Infect Dis*. 2003 Dec;3(12):757-71.
11. Döneray H, Keskin H, Akat H, Başaslan İH, Bağatır PŞ. Pediatric leptospirosis: a case report and review of literature. *Eurasian J Med*. 2023 Dec;55(1):150-6.
12. Kedia YS, Thorve SM, Waghmare SR, Mishra H, Karpe SP, Nair JP. Outcome of severe leptospirosis presenting with ARDS in respiratory ICU. *Lung India*. 2023 Nov-Dec;40(6):502-6.
13. Gadodia A, Ghosh I, Prakash G, Thulkar S, Raina V. Co-infection with hepatitis A and Leptospirosis in the Amazon region: Report of two cases. *Trop Gastroenterol*. 2011;32(3):234-6.
14. Gancheva G, Tzvetanova C, Ilieva P, Simova I. Comparative Study in Leptospirosis and Acute Viral Hepatitis. *J of IMAB*. 2007;13(1):25.

15. Somasundaram A, Loganathan N, Varghese J, Shivakumar S, Jayanthi V. Does leptospirosis behave adversely in cirrhosis of the liver? *Indian J Gastroenterol*. 2014 Nov;33(6):512-6.
16. Bismaya K, Dev P, Favas TT, Pathak A. Neuro-Leptospirosis: Experience from a tertiary center of North India. *Rev Neurol (Paris)*. 2023 Mar;179(3):238-43.
17. Khan SA, Dutta P, Borah J, Chowdhury P, Topno R, Baishya M, et al. Leptospirosis presenting as acute encephalitis syndrome (AES) in Assam, India. *Asian Pac J Trop Dis*. 2012 Apr;2(2):151-3.
18. Bhatt M, Rastogi N, Soneja M, Biswas A. Uncommon manifestation of leptospirosis: a diagnostic challenge. *BMJ Case Rep*. 2018 Oct 7;2018: bcr2018225281.
19. Puca E, Majko J, Puca E, Qyra E, Gega A, Pipero P. Acute encephalitis as initial presentation of leptospirosis. *J Infect Dev Ctries*. 2017 Apr 30;11(4):361-3.
20. Gulati S, Gulati A. Pulmonary manifestations of leptospirosis. *Lung India*. 2012 Oct;29(4):347-53.