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Research Article

Alterations in Renal and Hepatic Function Tests in Patients with Leptospirosis: A Clinical Correlation

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A B S T R A C T

Introduction: Leptospirosis is a significant zoonotic infection caused by Leptospira species, presenting with a wide clinical spectrum that ranges from mild febrile illness to severe multiorgan failure. Hepatic and renal dysfunctions are common complications that significantly influence prognosis. This study aimed to evaluate alterations in renal and hepatic function tests in confirmed cases of leptospirosis and correlate them with disease severity.

Methods: This prospective observational study was conducted in the Department of Medicine at a tertiary care teaching hospital. A total of 100 confirmed leptospirosis patients were enrolled based on clinical features and laboratory confirmation using IgM ELISA, MAT, or PCR. Routine haematological, renal, and hepatic parameters were analysed. Data were statistically evaluated using SPSS version 26.0, and correlations between biochemical markers and disease severity were assessed.

Results: The mean age of patients was 38.7 ± 12.4 years, with a male predominance (68%). Renal dysfunction was noted in 58% and hepatic dysfunction in 64% of cases, while 46% had combined hepatorenal involvement. Mean serum creatinine and total bilirubin were significantly higher in patients with severe disease ($p < 0.001$). A strong positive correlation ($r = 0.68$, $p < 0.001$) was observed between serum creatinine and bilirubin levels. The overall mortality rate was 5%, primarily in patients with multiorgan failure.

Conclusion: Hepatic and renal involvement are common in leptospirosis and correlate strongly with disease severity. Early recognition and timely management of hepatorenal dysfunction can significantly improve patient outcomes.

Keywords: Leptospirosis, hepatic dysfunction, renal dysfunction, hepatorenal involvement, liver function test, kidney function test

Introduction

Leptospirosis is a globally important zoonotic infection caused by pathogenic spirochetes of the genus *Leptospira*.[1] Transmission to humans typically occurs through direct or indirect contact with the urine of infected animals, particularly rodents, or with contaminated soil and water.[2] The disease is highly prevalent in tropical and subtropical regions where flooding, heavy rainfall, and poor sanitation favour the organism's survival and transmission.[3] India remains an endemic country, with frequent outbreaks reported from states such as Tamil Nadu, Kerala, Karnataka, Maharashtra, and the Andaman and Nicobar Islands. Despite its preventable nature, leptospirosis continues to be under recognised and underreported because of its nonspecific clinical presentation and low index of suspicion among healthcare providers.[4]

The clinical manifestations of leptospirosis range from a mild, self-limiting febrile illness to a severe, potentially fatal form characterised by multi organ dysfunction. The severe form, known as *Weil's disease*, presents with jaundice, renal failure, haemorrhagic manifestations, and cardiovascular collapse.[5] Hepatic involvement is a hallmark of the severe form and is characterised by hepatocellular injury, cholestasis, and hyperbilirubinemia disproportionate to the degree of transaminase elevation.[6] The jaundice in leptospirosis is typically non-obstructive and results from hepatocellular dysfunction and capillary leakage rather than massive hepatic necrosis. Common biochemical abnormalities include elevated serum bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP), which may help distinguish leptospirosis from viral hepatitis and other causes of febrile jaundice.[7]

Renal involvement is one of the most serious and frequent complications of leptospirosis, contributing significantly to morbidity and mortality.[8] The reported incidence of acute kidney injury (AKI) in leptospirosis varies widely from 10% to 60%, depending on geographical and population differences.[9] The pathogenesis of renal injury is multifactorial, involving both direct and indirect mechanisms. Direct tubular invasion by *Leptospira* leads to cytotoxic injury and tubulointerstitial nephritis, while indirect mechanisms include dehydration, rhabdomyolysis, immune-mediated inflammation, and haemodynamic instability, causing renal hypoperfusion.[10] The organism preferentially colonises the proximal tubular epithelial cells, triggering interstitial inflammation, acute tubular necrosis, and, in severe cases, fibrosis.[11] Laboratory derangements typically include elevated serum creatinine and blood urea nitrogen (BUN) levels, as well as electrolyte imbalances such as hypokalaemia and hyponatraemia.[12,13] Renal dysfunction in leptospirosis may manifest as either oliguric or non-oliguric AKI, with the degree of renal impairment correlating closely with disease severity and prognosis.[14] In some patients, severe AKI necessitates renal replacement therapy (RRT), reported in up to one-third of hospitalised cases.[15] Although renal function recovery is common, it may be delayed for several weeks or months, and recent evidence indicates that leptospiral AKI can predispose survivors to chronic kidney disease (CKD).[16]

Simultaneous hepatic and renal dysfunction, referred to as hepatorenal involvement, is a characteristic feature of severe leptospirosis and serves as a critical determinant of outcome.[6,8] This dual involvement arises from systemic endothelial injury, capillary leakage, and immune-mediated damage induced by leptospiral toxins. Understanding the spectrum and magnitude of hepatic and renal biochemical alterations is therefore vital for early diagnosis, risk stratification, and clinical management. Correlation of these laboratory findings with disease severity can also provide valuable prognostic information and guide timely intervention.

Despite numerous global reports, data regarding the pattern of biochemical abnormalities in leptospirosis remain limited in many Indian and tropical regional settings.[9] Variability in host response, infecting serovar, and environmental factors may influence the degree of hepatic and renal dysfunction. Thus, this study was undertaken to evaluate alterations in renal and hepatic function tests among patients with confirmed leptospirosis and to establish their correlation with clinical severity. The findings are expected to enhance understanding of disease progression and assist clinicians in prognostic evaluation and management of affected individuals.

Materials and Methods

Study Design and Setting

This study was designed as a prospective observational study conducted in the Department of Medicine at a tertiary care teaching hospital.

Study Population

A total of 100 patients who presented during the study period with clinically suspected leptospirosis were screened. The diagnosis of leptospirosis was confirmed based on compatible clinical features along with positive laboratory confirmation by Leptospira-specific IgM enzyme-linked immunosorbent assay (ELISA), microscopic agglutination test (MAT), or polymerase chain reaction (PCR). All confirmed cases satisfying the inclusion criteria were enrolled for detailed evaluation.

Inclusion Criteria

Patients aged ≥18 years with a confirmed diagnosis of leptospirosis established by:

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- Positive *Leptospira* IgM ELISA, or
- MAT titre ≥1:400, or
- Detection of *Leptospira* DNA by PCR.
- Patients providing written informed consent.

Exclusion Criteria

- Patients with known chronic liver disease (e.g., viral hepatitis, alcoholic liver disease, cirrhosis) or chronic kidney disease.
- Patients with other causes of febrile illness, such as dengue, malaria, enteric fever, or sepsis of non-leptospiral origin.
- Patients receiving hepatotoxic or nephrotoxic drugs prior to admission.
- Pregnant or lactating women.

Clinical Evaluation

All patients underwent a detailed history and physical examination at admission. Clinical information included duration of fever, myalgia, jaundice, vomiting, oliguria, breathlessness, and bleeding manifestations. A history of occupational exposure, contact with contaminated water, or recent rainfall/flooding was documented. A comprehensive physical examination was conducted, with special attention to vital signs, pallor, icterus, hepatomegaly, splenomegaly, oedema, and signs of haemorrhage. Disease severity was graded as mild, moderate, or severe based on WHO clinical criteria and extent of organ involvement.

Laboratory Investigations

All enrolled patients underwent a series of routine and specific laboratory investigations. Haematological parameters included haemoglobin, total leukocyte count, platelet count, and haematocrit estimation. Renal function tests (RFT) comprised measurement of serum creatinine, blood urea nitrogen (BUN), serum electrolytes such as sodium and potassium, and urine analysis for albumin, sugar, and microscopic examination. Hepatic function tests (LFT) included assessment of total and direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), total protein, and serum albumin levels.

For diagnostic confirmation, a Leptospira-specific IgM ELISA was performed in all patients, while the microscopic agglutination test (MAT) or polymerase chain reaction (PCR) was utilised in selected cases when available. Renal dysfunction was defined as serum creatinine ≥1.5 mg/dL or BUN above the normal reference range, whereas hepatic dysfunction was defined as total bilirubin >1.2 mg/dL or transaminase (AST/ALT) levels exceeding twice the upper limit of normal. Hepatorenal involvement was considered when both hepatic and renal parameters were simultaneously deranged.

Data Collection and Follow-Up

All clinical and biochemical data were recorded at admission, during hospitalisation, and at discharge using a predesigned proforma. The duration of hospital stays, requirement of intensive care, need for renal replacement therapy (RRT), and outcome (recovery or death) were documented. Patients who developed AKI were managed according to institutional protocols, including intravenous fluids, antibiotics, and dialysis when indicated.

Statistical Analysis

Data were compiled and analysed using Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range), while categorical variables were represented as frequency and percentage. Comparisons between groups (e.g., hepatic vs. non-hepatic involvement, renal vs. non-renal involvement) were made using the chi-square test or Fisher's exact test for categorical data and the Student's t-test or Mann–Whitney U test for continuous data as appropriate. Correlation between hepatic and renal function test parameters with disease severity was assessed using Pearson's or Spearman's correlation coefficients. A p-value of <0.05 was considered statistically significant.

Results

Demographic profile

A total of 100 patients with confirmed leptospirosis were included in the study. The mean age of the study population was 38.7 ± 12.4 years (range: 18–70 years). The majority of patients belonged to the 31–50 years age group (46%), followed by 18–30 years (28%) and those aged above 50 years (26%). Out of 100 patients, 68 (68%) were males, and 32 (32%) were females, with a male-to-female ratio of 2.1:1, indicating a male predominance. Most patients were from rural backgrounds (72%) and engaged in agriculture or field-related occupations, suggesting greater exposure to contaminated water and soil. The distribution of demographic and clinical features is shown in Table 1.

Clinical presentation

Fever was the most common presenting symptom, observed in all patients (100%), followed by myalgia (82%), headache (65%), and jaundice (60%). Other frequent symptoms included vomiting (54%), abdominal pain (48%), oliguria (42%), and breathlessness (28%). Conjunctival suffusion was

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noted in 40% of patients, while bleeding manifestations such as petechiae or haematuria were observed in 12%. Based on clinical severity, 64 (64%) patients had mild disease, 26 (26%) had moderate disease, and 10 (10%) presented with severe leptospirosis requiring intensive care management (Table 1).

Renal function abnormalities

Renal involvement was detected in 58% of patients. The mean serum creatinine level among those with renal dysfunction was 3.12 ± 1.24 mg/dL, compared to 0.94 ± 0.28 mg/dL in patients without renal involvement ($p < 0.001$). The mean blood urea nitrogen (BUN) level was 89.5 ± 28.6 mg/dL in patients with renal involvement, compared to 28.4 ± 10.2 mg/dL in those with normal renal function ($p < 0.001$). Electrolyte disturbances were frequent, with hyponatraemia seen in 38% and hypokalaemia in 42% of patients. Oliguria occurred in 40% of cases, and anuria in 6%. Renal replacement therapy (RRT) was required in 8% of patients with severe acute kidney injury. The comparative renal function parameters are summarised in Table 2.

Hepatic function abnormalities

Hepatic dysfunction was observed in 64% of the study population. The mean total bilirubin level among patients with hepatic involvement was 6.84 ± 3.15 mg/dL, significantly higher than 1.02 ± 0.36 mg/dL in those without hepatic derangement ($p < 0.001$). The mean AST and ALT levels were 118.6 ± 52.3 U/L and 102.4 ± 46.7 U/L, respectively, while the mean ALP level was 216.8 ± 88.4 U/L. The pattern of hyperbilirubinemia was predominantly conjugated, and none of the patients developed hepatic encephalopathy. Hypoalbuminemia (<3.5 g/dL) was found in 46% of cases. These findings are detailed in Table 2.

Combined hepatorenal involvement

Concurrent hepatic and renal dysfunction (hepatorenal involvement) was observed in 46% of patients. These patients had a significantly longer mean hospital stay (9.8 ± 3.4 days) compared to those with isolated hepatic or renal involvement (5.6 ± 2.8 days, $p = 0.004$). Among patients with combined organ involvement, 7 (15.2%) required dialysis, and 2 (4.3%) succumbed to multiorgan failure despite intensive management. A comparison of clinical outcomes based on the pattern of organ involvement is presented in Table 3.

Correlation between biochemical parameters and disease severity

A statistically significant positive correlation was found between serum creatinine and total bilirubin levels ($r = 0.68$, $p < 0.001$), indicating that rising renal parameters were associated with worsening hepatic dysfunction. Similarly, elevated AST and ALT levels were significantly correlated with disease severity ($p < 0.05$). Patients with severe leptospirosis demonstrated markedly higher mean values of serum creatinine (4.2 ± 1.3 mg/dL) and total bilirubin (8.7 ± 3.8 mg/dL) compared to those with mild or moderate disease.

Outcome

Out of 100 patients, 95 (95%) recovered completely with appropriate management, while 5 (5%) died due to disease-related complications, primarily multiorgan failure and severe pulmonary involvement. The mean duration of hospitalisation was 7.8 ± 3.2 days, with longer stays observed among patients with hepatorenal dysfunction (Table 3).

Table 1.Demographic and clinical profile of patients with leptospirosis

(n = 100)

Variable	Category	Number of patients (%)
Age (years)	18–30	28 (28%)
	31–50	46 (46%)
	>50	26 (26%)
Gender	Male	68 (68%)
	Female	32 (32%)
Residence	Rural	72 (72%)
	Urban	28 (28%)
Occupation	Agricultural/field worker	59 (59%)
	Industrial/other	25 (25%)
	Unemployed/student	16 (16%)

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Presenting symptoms	Fever	100 (100%)
	Myalgia	82 (82%)
	Headache	65 (65%)
	Jaundice	60 (60%)
	Vomiting	54 (54%)
	Abdominal pain	48 (48%)
	Oliguria	42 (42%)
	Breathlessness	28 (28%)
Disease severity	Mild	64 (64%)
	Moderate	26 (26%)
	Severe	10 (10%)

Table 2.Comparison of renal and hepatic function parameters among patients with and without organ involvement

Parameter	Without Organ involvement	Organ involvement	p-value
Serum creatinine (mg/dL)	0.94 ± 0.28	3.12 ± 1.24	<0.001
BUN (mg/dL)	28.4 ± 10.2	89.5 ± 28.6	<0.001
Sodium (mEq/L)	136.2 ± 3.5	132.8 ± 5.1	0.021
Potassium (mEq/L)	4.0 ± 0.3	3.4 ± 0.6	0.012
Total bilirubin (mg/dL)	1.02 ± 0.36	6.84 ± 3.15	<0.001
AST (U/L)	38.2 ± 11.4	118.6 ± 52.3	<0.001
ALT (U/L)	35.7 ± 10.8	102.4 ± 46.7	<0.001
ALP (U/L)	124.8 ± 34.6	216.8 ± 88.4	0.002
Serum albumin (g/dL)	4.1 ± 0.4	3.2 ± 0.5	0.008

Table 3.Clinical outcomes based on organ involvement

(n = 100)

Organ involvement	Number of patients (%)	Mean hospital stay (days)	ICU admission (%)	RRT required (%)	Mortality (%)
Hepatic only	18 (18%)	6.2 ± 2.1	11.1%	0	0
Renal only	20 (20%)	7.0 ± 2.8	15.0%	2 (10.0%)	0
Combined hepatorenal	46 (46%)	9.8 ± 3.4	41.3%	7 (15.2%)	2 (4.3%)
None	16 (16%)	4.5 ± 1.9	0	0	0
Total / mean	100 (100%)	7.8 ± 3.2	–	–	5 (5.0%)

Discussion

In the present study involving 100 confirmed leptospirosis patients, the mean age was 38.7 ± 12.4 years, and most cases (46%) were within the 31–50 years age group. A male predominance (68%) was noted, consistent with the occupational risk associated with outdoor exposure to contaminated water. Similar demographic patterns have been reported globally. Abgueguen et al. (2008)[17] conducted a retrospective analysis of 134 adult patients in France and found a mean age of 43 years, with males comprising 72% of the cohort, attributing this to occupational exposure during agricultural and industrial work. Our study findings closely parallel these results, suggesting similar demographic susceptibility patterns across diverse geographic settings.

Fever was universal in our study (100%), followed by myalgia (82%), headache (65%), and jaundice (60%). These data align with Cruz et al. (2009)[18], who reported fever in 100% and myalgia in 84% of cases in their multicentric review of leptospirosis in developing nations. The frequency of conjunctival suffusion (40%) in our study was comparable to

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the 35% reported by Abgueguen et al. (2008)[17], reinforcing its diagnostic value though it is often underrecognised.

Regarding disease severity, 64% of our patients had mild disease, 26% moderate, and 10% severe. This pattern is similar to that reported by Pappas and Cascio (2006)[19], who noted severe disease in approximately 9–12% of hospitalised cases. They emphasised that delayed presentation and comorbidities were significant predictors of severe outcomes—findings echoed in our cohort, where late presentation correlated with higher complication rates.

Overall, the demographic and clinical spectrum of our patients corresponds well with previously published literature, supporting the observation that leptospirosis predominantly affects working-age men engaged in high-risk occupations and manifests initially as a nonspecific febrile illness with variable hepatic and renal involvement.

Renal involvement

In our study, renal dysfunction was observed in 58% of cases. The mean serum creatinine was 3.12 ± 1.24 mg/dL, and the mean blood urea nitrogen (BUN) was 89.5 ± 28.6 mg/dL. Oliguria was noted in 40% and anuria in 6% of patients, while renal replacement therapy (RRT) was required in 8%.

These findings correspond closely with those of Daher et al. (2009) [20], who analysed 196 patients and found acute kidney injury (AKI) in 54.5% of cases, with oliguric AKI in 38% and dialysis required in 12.8%. They identified hyperbilirubinemia, hypotension, and late initiation of antibiotics as predictors of oliguric renal failure. Our lower dialysis rate (8%) might reflect early diagnosis and timely intervention.

Visith and Kearkiat (2005)[21] studied 87 patients in Thailand and reported renal involvement in 60%, with serum creatinine ranging from 2 to 10 mg/dL and BUN up to 120 mg/dL. They highlighted that non-oliguric renal failure was more common (70%) than the oliguric type (30%), suggesting that leptospiral nephropathy often presents with preserved urine output. Our observation that most cases were non-oliguric supports their findings.

In the prospective study by Basu et al. (2011)[22], 36% of patients with tropical acute febrile illness developed AKI (defined by RIFLE criteria), with 24% requiring dialysis. Their reported incidence of AKI was slightly lower, which they attributed to early hydration and aggressive management. These variations across studies likely reflect differences in population characteristics and access to healthcare.

Cruz et al. (2009)[18] described leptospirosis as a leading cause of AKI in tropical countries, with 40–60% of severe cases developing renal failure. They emphasised that hypovolemia, rhabdomyolysis, and tubular dysfunction are key pathogenic factors—mechanisms that are also evident in our cohort, where 38% had hyponatraemia and 42% hypokalaemia, reflecting tubular involvement.

Hepatic involvement

Hepatic dysfunction was present in 64% of our patients, with a mean total bilirubin of 6.84 ± 3.15 mg/dL, AST of 118.6 ± 52.3 U/L, ALT of 102.4 ± 46.7 U/L, and ALP of 216.8 ± 88.4 U/L. Hyperbilirubinemia was predominantly conjugated, and no patients developed hepatic encephalopathy.

Pappas and Cascio (2006)[19] reported that 60–70% of leptospirosis patients develop hepatic involvement, with bilirubin often exceeding 10 mg/dL but only moderate transaminase elevation (<200 U/L). They concluded that hepatic dysfunction in leptospirosis is primarily caused by capillary leakage and intrahepatic cholestasis, rather than hepatocellular necrosis. Our results (bilirubin 6.84 mg/dL; AST 118.6 U/L) support this biochemical pattern.

Visith and Kearkiat (2005)[21] similarly observed mean bilirubin levels of 7.1 mg/dL and AST/ALT around 110 U/L, indicating mild-to-moderate hepatocellular injury. Abgueguen et al. (2008)[17] reported jaundice in 58% of their patients, with an average bilirubin level of 8.5 ± 3.9 mg/dL and modest enzyme elevations—findings strikingly similar to our data.

Thus, the hepatic involvement in our series aligns closely with international studies, demonstrating that leptospirosis typically produces disproportionate jaundice with mild-to-moderate elevation of enzymes and preserved hepatic synthetic function.

In the present study, combined hepatic and renal dysfunction was seen in 46% of patients. This subgroup had prolonged hospital stays (mean 9.8 ± 3.4 days), higher ICU admissions (41.3%), and increased mortality (4.3%) compared to those with isolated organ involvement.

Daher et al. (2009)[20] reported that patients with both hepatic and renal dysfunction had a threefold higher risk of death compared to those with single-organ involvement. They also found that oliguria, hyperbilirubinemia, and thrombocytopenia were significant predictors of mortality. Our data corroborate this observation, as patients with dual organ failure exhibited greater severity and required more intensive management.

Hurst et al. (2009)[23] studied 12 cases of anicteric leptospirosis complicated by renal failure requiring haemodialysis. They concluded that renal injury can occur independently of hepatic involvement, often due to direct tubular invasion by *Leptospira* species. Similarly, in our cohort, a subset of patients with normal bilirubin values had significant renal dysfunction, reinforcing the concept of independent renal pathogenicity.

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Cruz et al. (2009)[18] emphasised that multiorgan involvement, particularly hepatorenal syndrome, is the primary determinant of poor outcomes in developing nations. Basu et al. (2011)[22] and Abgueguen et al. (2008)[17] also reported that patients with concurrent organ involvement had higher mortality and longer recovery times. These conclusions align with our findings, which show that the combined involvement group had worse outcomes than those with isolated hepatic or renal disease.

The overall mortality in our study was 5%, which is lower than the 8–10% mortality observed by Abgueguen et al. (2008) [17] and comparable to the 5% reported by Visith and Kearkiat (2005).[21] Pappas and Cascio (2006)[19] noted that early antibiotic therapy significantly reduces mortality and organ dysfunction, a finding consistent with our results, where early management correlated with improved outcomes.

Conclusion

In this study, hepatic dysfunction was observed in 64% and renal dysfunction in 58% of leptospirosis patients, with 46% showing combined hepatorenal involvement. Elevated bilirubin and creatinine levels correlated with greater disease severity and prolonged hospitalisation. Early detection and management of hepatorenal dysfunction are crucial for improving outcomes and reducing mortality.

Limitations

The present study had certain limitations. It was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalisability of the findings. Molecular confirmation tests like PCR and MAT were not performed for all patients due to resource constraints. Additionally, long-term follow-up to assess residual hepatic or renal impairment was not undertaken, restricting evaluation of chronic sequelae.

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