International Journal of Research in Medical Sciences
Saravanamuthu PK et al. Int J Res Med Sci. 2026 Aug;14(8):3399-3405
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20262628

Original Research Article

Tropical-infection-precipitated acute-on-chronic liver failure: a distinct, lower-mortality phenotype in a Northeast Indian cohort

Ponn Karthikeyan Saravanamuthu[1]*, Anup Kumar Das[1], Rashmi Rajkakati[2], Aparna Sonowal[3]

[1]Department of Medicine, Assam Medical College and Hospital, Dibrugarh, Assam, India
[2]Department of Biochemistry, Assam Medical College and Hospital, Dibrugarh, Assam, India
[3]Department of Microbiology, Assam Medical College and Hospital, Dibrugarh, Assam, India

Received: 20 May 2026
Revised: 16 June 2026
Accepted: 18 July 2026

***Correspondence:**
Dr. Ponn Karthikeyan Saravanamuthu,
E-mail: sponkarthi@gmail.com

ABSTRACT

Background: Leptospirosis and scrub typhus are endemic tropical infections in northeast India capable of precipitating acute-on-chronic liver failure (ACLF). Both are doxycycline-responsive. Whether tropical-precipitated ACLF represents a distinct, lower-mortality phenotype amenable to targeted therapy is unknown.
Methods: Seventy consecutive adults with EASL-CLIF ACLF were prospectively enrolled at a tertiary teaching hospital in Northeast India (October 2024-September 2025). Leptospirosis was diagnosed by IgM ELISA and/or compatible clinical syndrome; scrub typhus by Orientia tsutsugamushi IgM ELISA or Weil-Felix titre ≥1:160. All clinically suspected tropical infections received empirical doxycycline 100 mg twice daily alongside standard broad-spectrum antibiotics. Tropical-precipitated versus non-tropical-precipitated subgroups were compared for severity, inflammatory markers, and 28-day mortality.
Results: Tropical infection precipitated 17 of 70 ACLF episodes (24.3%)- 10 leptospirosis (14.3%), 6 scrub typhus (8.6%), and 1 mixed Leptospira-Orientia infection (1.4%). The tropical subgroup had a higher proportion of grade 1 ACLF (41.2% versus 7.5%; p=0.003), lower admission PCT (2.54 versus 6.73 ng/ml), and significantly higher admission creatinine (5.91 versus 3.19 mg/dl; p=0.018). Twenty-eight-day mortality was 35.3% in the tropical subgroup versus 62.3% in non-tropical patients (OR 0.33; 95% CI 0.11-1.05; p=0.091). Compared with alcohol-precipitated ACLF specifically, tropical-precipitated mortality was significantly lower (35.3% versus 73.9%; OR 0.19, p=0.024).
Conclusions: Tropical-infection-precipitated ACLF is a distinct phenotype with lower presentation severity and a clinically important mortality advantage over alcohol-precipitated ACLF. Empirical doxycycline in ACLF patients with suspected leptospirosis or scrub typhus in endemic regions deserves routine inclusion in management protocols.

Keywords: Acute-on-chronic liver failure, Doxycycline, Leptospirosis, Mortality, Northeast India, Scrub typhus, Tropical infection

INTRODUCTION

Acute-on-chronic liver failure (ACLF) is the most lethal acute syndrome that complicates established cirrhosis, with 28-day mortality of 23%, 32%, and 74% across Grades 1, 2, and 3 respectively, in the EASL-CLIF construction.[1] The epidemiology of ACLF is geographically variable. Western European cohorts are dominated by alcohol-related cirrhosis with bacterial infection- particularly gram-negative bacteraemia and

spontaneous bacterial peritonitis- as the precipitant.[2] East Asian cohorts feature hepatitis B reactivation as a major driver.[3] In India, alcohol-related liver disease and bacterial infections remain the principal contributors, but the infection mix is regionally heterogeneous.[4]

Northeast India occupies a distinctive epidemiological position. The region has high per-capita alcohol consumption and an attendant burden of alcohol-related cirrhosis, but is simultaneously endemic for two tropical zoonoses- leptospirosis and scrub typhus- both of which peak during and after the monsoon season and both of which are recognised causes of acute hepatic injury.[5,6] In a cirrhotic patient, the systemic inflammation generated by leptospiral or rickettsial infection can precipitate multi-organ failure, closely mimicking bacterial-sepsis-precipitated ACLF.

The question matters clinically because both precipitants are doxycycline-responsive. If tropical-precipitated ACLF retains some reversibility- if outcome is meaningfully better when the precipitating infection is eliminated- then early empirical doxycycline becomes a potentially life-saving intervention on top of standard ACLF management, and the phenotype deserves distinct clinical recognition.[7,8]

Against this background, we examined a prospective single-centre cohort of 70 adults with EASL-CLIF ACLF in Northeast India. We aimed to: (i) define the proportion of ACLF episodes precipitated by tropical infection; (ii) compare the tropical and non-tropical subgroups in admission severity and inflammatory profile; and (iii) compare 28-day mortality between subgroups to test the hypothesis that tropical-precipitated ACLF is a distinct, lower-mortality phenotype.

METHODS

Setting and design

This was a prospective, single-arm, observational study conducted in the department of general medicine of Assam Medical College and Hospital (AMCH), a 1,250-bed tertiary teaching hospital in Dibrugarh, Assam, northeast India. Enrolment ran from October 2024 through September 2025, spanning a complete monsoon season during which tropical transmission peaks.

Participants

Consecutive adults admitted to the department of general medicine with ACLF fulfilling EASL-CLIF Consortium criteria were screened for eligibility.[1]

Inclusion criteria

The study included: (i) age ≥18 years; (ii) a diagnosis of ACLF as defined by the EASL-CLIF consortium criteria; and (iii) written informed consent provided by the patient or next-of-kin.

Exclusion criteria

The study excluded: (i) hepatocellular carcinoma or any other active malignancy; (ii) portal-vein thrombosis; (iii) pregnancy; (iv) congestive cardiac failure; (v) connective-tissue disease requiring biologic therapy; (vi) previous major abdominal surgery; and (vii) refusal or unwillingness to participate.

Sample size

The cohort size was determined a priori by a correlation-based power calculation (expected correlation coefficient r=0.33, two-sided α=0.05, statistical power 80%), which yielded a minimum requirement of 70 participants. Accordingly, 70 consecutive eligible patients enrolled during the study period constituted the analytical cohort, within which the tropical-precipitated and non-tropical-precipitated subgroups were compared.

Identification of tropical precipitants

All patients received a uniform infection screening bundle including peripheral blood cultures, urine culture, diagnostic paracentesis where indicated, and chest radiograph. Given regional epidemiology, IgM ELISA for leptospirosis and for scrub typhus (Orientia tsutsugamushi) were requested in any patient with unexplained fever, hepatorenal syndrome, or multi-organ involvement. Leptospirosis was diagnosed by a positive IgM ELISA or a compatible clinical syndrome. Scrub typhus was diagnosed by positive IgM ELISA, an eschar, or a Weil-Felix titre ≥1:160 against OXK antigen with a compatible clinical syndrome.

Empirical antimicrobial protocol

All patients received broad-spectrum empirical antibiotics per institutional protocol aligned with EASL 2023 recommendations.[9] Doxycycline 100 mg orally or intravenously twice daily was added empirically- pending serology- in any patient with clinical suspicion of leptospirosis (fever with myalgia, conjunctival suffusion, jaundice, acute kidney injury) or scrub typhus (fever with rash, eschar, lymphadenopathy, hepatic injury). Antibiotic regimens were narrowed, continued, or stopped after 48-72 hours based on culture and serology results.

Clinical assessment and data collection

Structured history, clinical examination, and a uniform admission laboratory panel- including haemogram, liver and renal biochemistry, INR, electrolytes, viral hepatitis serology, and abdominal ultrasonography- were obtained for every patient. Hepatic encephalopathy was graded by West Haven criteria. MELD-Na and CLIF-C ACLF scores were calculated at admission. C-reactive protein and procalcitonin were measured on day 1 and day 3. Twenty-eight-day vital status was ascertained by in-hospital review or structured telephone contact.

Statistical analysis

Continuous variables are summarised as mean±SD; categorical as N (%). Group comparisons used the Mann-Whitney U test for continuous variables and Fisher's exact test for categorical variables. Odds ratios (OR) with 95% confidence intervals were derived by logistic regression. Two-sided $p<0.05$ was the threshold for significance. All analyses were performed using Python 3 (SciPy, NumPy).

Ethics

Institutional human ethics committee approval was obtained before study initiation. Written informed consent was obtained from each participant or their next-of-kin. The study conformed to the Declaration of Helsinki. This study was an observational analysis and was reported in accordance with STROBE guidelines.

RESULTS

Cohort and tropical-precipitant frequency

Seventy adults with ACLF were enrolled. Mean age was 42.2±9.6 years; 88.6% were men. Alcohol-related cirrhosis was the dominant underlying aetiology (68.6%). Overall, 28-day mortality was 55.7% (39/70). Seventeen of 70 ACLF episodes (24.3%) were tropical-precipitated: 10 by leptospirosis alone (14.3%), 6 by scrub typhus alone (8.6%), and 1 by combined Leptospira-Orientia infection (1.4%). The dominant non-tropical subgroups were alcohol-precipitated (n=23, 32.9%), bacterial-sepsis-precipitated (n=23, 32.9%), and other precipitants (variceal bleeding, drug-induced liver injury, liver abscess; n=7, 10.0%).

Table 1: Baseline characteristics by precipitant subgroup.

Variables	Tropical (n=17)	Alcohol (n=23)	Bacterial (n=23)	Other (n=7)
Age (years, mean±SD)	42.8±9.8	39.8±10.6	45.2±7.3	39.0±11.1
Male, N (%)	16 (94.1)	21 (91.3)	19 (82.6)	6 (85.7)
Alcohol-related cirrhosis, N (%)	14 (82.4)	18 (78.3)	12 (52.2)	4 (57.1)
MELD-Na (mean±SD)	34.9±7.4	36.2±4.6	33.4±5.6	31.7±8.7
CLIF-C ACLF score (mean±SD)	52.1±7.7	52.4±6.8	51.6±6.3	48.4±6.2
ACLF grade 1, N (%)	7 (41.2)	1 (4.3)	1 (4.3)	2 (28.6)
ACLF grade 2, N (%)	3 (17.6)	6 (26.1)	14 (60.9)	2 (28.6)
ACLF grade 3, N (%)	7 (41.2)	16 (69.6)	8 (34.8)	3 (42.9)

MELD-Na- model for end-stage liver disease-sodium; CLIF-C ACLF- Chronic Liver Failure Consortium ACLF score. Values shown as mean±SD or N (%).

Table 2: Severity and inflammatory profile, tropical versus non-tropical precipitated ACLF.

Parameters	Tropical (n=17)	Non-tropical (n=53)	P value
Age (years, mean±SD)	42.8±9.8	42.0±9.6	0.587
Total bilirubin (mg/dl)	18.65±12.44	16.36±9.82	0.519
Creatinine (mg/dl)	5.91±4.01	3.19±2.11	0.018*
INR	1.98±0.53	2.38±0.93	0.174
CRP Day 1 (mg/dl)	3.81±0.90	4.40±2.95	0.951
PCT Day 1 (ng/ml)	2.54±1.89	6.73±13.02	0.529
MELD-Na (mean±SD)	34.9±7.4	34.4±5.8	0.794
CLIF-C ACLF score (mean±SD)	52.1±7.7	51.5±6.5	0.896
ACLF grade 1, N (%)	7 (41.2)	4 (7.5)	0.003*
28-day mortality, N (%)	6 (35.3)	33 (62.3)	0.091

Continuous comparisons by Mann-Whitney U test; categorical by Fisher's exact test. *$p<0.05$. CRP- c-reactive protein; PCT, procalcitonin; INR, international normalised ratio.

Baseline characteristics by precipitant subgroup

The tropical subgroup did not differ meaningfully from the non-tropical cohort in age (42.8±9.8 versus 42.0±9.6 years; p=0.587), sex distribution, or underlying aetiology; alcohol-related cirrhosis was the dominant background pathology in tropical-precipitated patients too (82.4%). Baseline demographics by precipitant subgroup are shown in Table 1.

Lower severity at admission in the tropical subgroup

Tropical-precipitated ACLF presented at lower severity grades. Grade 1 disease accounted for 7 of 17 tropical

patients (41.2%) versus 4 of 53 non-tropical patients (7.5%; Fisher's exact p=0.003). Grade 3 was less common in tropical patients (7/17, 41.2%) than in alcohol-precipitated patients (16/23, 69.6%). Mean MELD-Na (34.9 versus 34.4) and CLIF-C ACLF score (52.1 versus 51.5) were comparable between tropical and non-tropical groups (p=0.794 and 0.896), indicating that conventional severity scores overlapped even when grade distribution differed.

Inflammatory profile and creatinine

Admission inflammatory markers were numerically lower in the tropical subgroup: day-1 CRP 3.81 versus 4.40 mg/dl and day-1 PCT 2.54 versus 6.73 ng/ml; neither reached statistical significance, partly due to small subgroup size and wide PCT variance. In contrast, admission creatinine was significantly higher in the tropical group (5.91 versus 3.19 mg/dl; p=0.018), reflecting direct infection-associated acute kidney injury from both leptospirosis and scrub typhus superimposed on cirrhotic renal dysfunction.[10-12] This combination explains why tropical patients presented predominantly at grade 1-renal-dominant single-organ failure- with the potential to avert escalation when the infection is treated promptly. Severity and inflammatory data are shown in Table 2.

Twenty-eight-day mortality

Twenty-eight-day mortality was 35.3% in the tropical subgroup (6/17) versus 62.3% in the non-tropical group (33/53). The absolute risk difference was −27.0 percentage points; the unadjusted OR for death in the tropical subgroup was 0.33 (95% CI 0.11-1.05; Fisher's exact p=0.091). Grade-stratified mortality showed 0% deaths in grade 1, 66.7% in grade 2, and 57.1% in grade 3 tropical patients- compared with 0%, 40.9%, and 88.9% in the corresponding non-tropical grades (Table 3). The grade 3 difference (57.1% versus 88.9%) is particularly striking clinically.

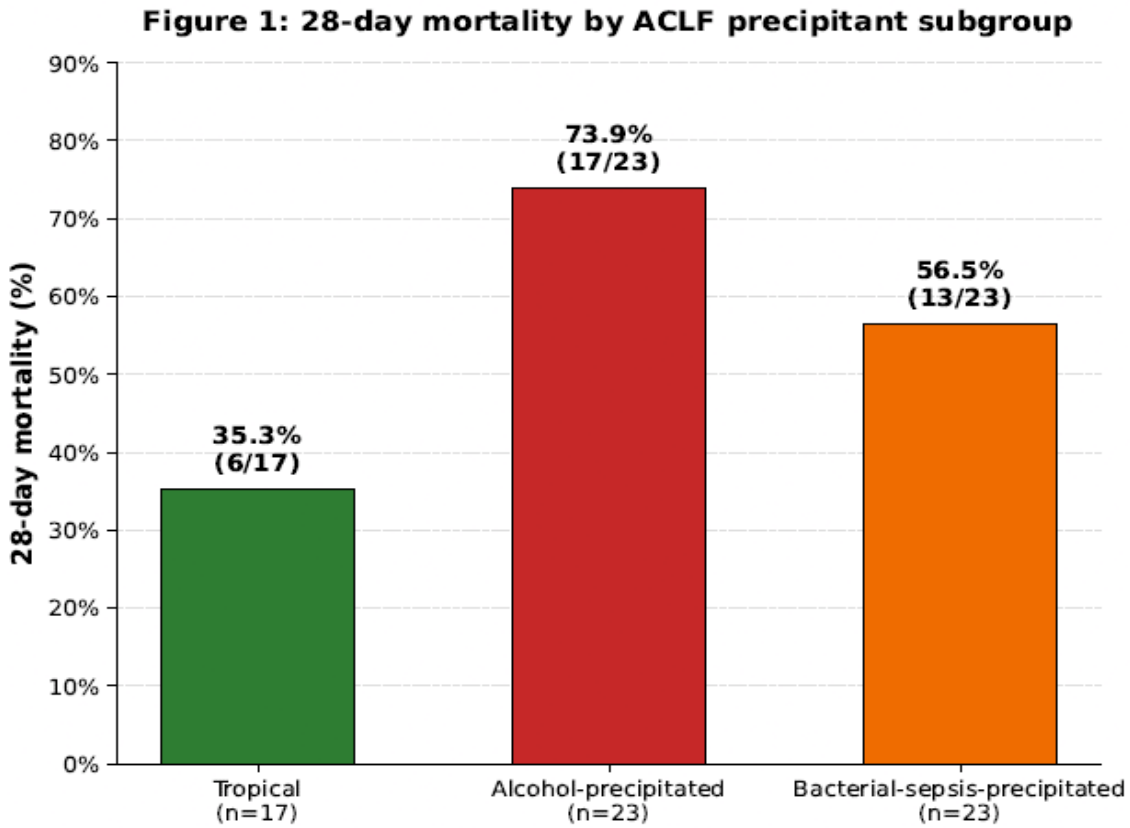

Figure 1: 28-day mortality by ACLF precipitant subgroup.

Table 3: Twenty-eight-day mortality stratified by ACLF grade.

ACLF grade	Tropical (n=17) deaths/N (%)	Non-tropical (n=53) deaths/N (%)	Overall cohort deaths/N (%)
Grade 1	0/7 (0.0)	0/4 (0.0)	0/11 (0.0)
Grade 2	2/3 (66.7)	9/22 (40.9)	11/25 (44.0)
Grade 3	4/7 (57.1)	24/27 (88.9)	28/34 (82.4)
Overall	6/17 (35.3)	33/53 (62.3)	39/70 (55.7)

Mortality versus alcohol- and bacterial-precipitated subgroups

Pairwise comparison showed 28-day mortality of 35.3% in tropical (6/17), 73.9% in alcohol-precipitated (17/23), and 56.5% in bacterial-sepsis-precipitated (13/23) subgroups (Figure 1). The pairwise OR for tropical versus alcohol-precipitated was 0.19 (95% CI 0.04-0.89; p=0.024) and for tropical versus bacterial-precipitated was 0.42 (95% CI 0.11-1.59; p=0.22). The tropical-versus-alcohol mortality difference reached formal statistical significance and the bacterial comparison was directionally consistent, supporting the hypothesis that tropical-precipitated ACLF carries a meaningfully better short-term prognosis than alcohol-precipitated ACLF in particular.

DISCUSSION

Three findings frame this analysis. First, tropical infections precipitated nearly a quarter (24.3%) of ACLF episodes in this northeast Indian cohort- a proportion substantially higher than in most published Indian or international ACLF series and consistent with prior regional reports.[8] Second, the tropical subgroup presented at lower severity grades, with lower inflammatory markers but significantly higher creatinine reflecting infection-associated acute kidney injury superimposed on cirrhosis. Third, 28-day mortality in the tropical subgroup was 35.3% versus 62.3% in non-tropical ACLF- a 27-percentage-point absolute advantage, with the comparison against alcohol-precipitated ACLF reaching formal significance (OR 0.19, p=0.024), and retained even within grade 3 disease (57.1% versus 88.9%).

The most likely explanation for the mortality advantage is the treatability of the precipitant. Doxycycline is highly effective against both Leptospira and Orientia tsutsugamushi, and early therapy substantially reduces mortality even in severe disease.[7,13] Because all clinically suspected tropical patients in our cohort received empirical doxycycline within the first 24-48 hours of admission alongside standard broad-spectrum antibiotics, the tropical subgroup effectively received targeted antimicrobial therapy alongside conventional ACLF management. By contrast, alcohol-precipitated ACLF has no specific antidote beyond abstinence and supportive care, and bacterial-sepsis-precipitated ACLF responds to antibiotics only as well as source control allows.

Several features of the tropical phenotype are biologically coherent with this interpretation. The lower admission inflammatory burden (particularly PCT) compared with non-tropical ACLF is consistent with the more selective inflammatory profile of tropical infections versus gram-negative bacteraemia.[10] The higher creatinine reflects direct tubular injury from both Leptospira and Orientia, superimposed on cirrhotic functional renal impairment- a combination that is partially reversible when the infection is treated.[11,12] The predominance of grade 1 in the tropical subgroup (41.2%)- renal-dominant single-organ failure- is consistent with the idea that early antimicrobial therapy can interrupt organ-failure cascade before progression to grade 3.

The clinical implication is direct. In any tropical endemic region during monsoon and post-monsoon seasons, a cirrhotic patient with acute decompensation, fever, acute kidney injury, and hepatic injury should receive empirical doxycycline 100 mg twice daily alongside standard broad-spectrum antibiotics, pending serology. The cost and toxicity of empirical doxycycline are negligible compared with the mortality risk of delayed treatment. A second implication concerns prognostication and transplant triage: a grade 3 ACLF patient with a tropical precipitant retains a substantially better survival probability (42.9% in our data) than the cohort-wide grade 3 mortality of 82.4% would imply, arguing against premature de-escalation of care.

The western ACLF literature- including CANONIC and the EASL 2023 guidelines- does not isolate a tropical-precipitated subgroup because the precipitant is rare in those settings.[2,9] The APASL framework recognises infection as a key ACLF driver in Asia but does not differentiate tropical from bacterial subgroups.[3] Medhi and colleagues from a neighbouring northeast Indian centre identified leptospirosis as a notable precipitant but did not report outcome stratification.[8] Our analysis adds the first outcome-stratified data characterising the tropical phenotype as clinically distinct and prognostically favourable relative to other precipitant categories.

These findings can be placed in the context of previous ACLF cohorts. In the multicentre Indian National Association for Study of the Liver (INASL) consortium experience, alcohol-related disease and active bacterial infection dominated the precipitant profile, with tropical zoonoses scarcely represented at the national level; the 24.3% tropical-precipitant burden observed here therefore reflects a genuinely region-specific epidemiology rather than a generalisable Indian pattern.[14] The comparable MELD-Na and CLIF-C ACLF scores between our subgroups, despite divergent mortality, echo the recognised limitation that prognostic scores derived predominantly from alcohol- and sepsis-precipitated cohorts may under-estimate survival when the precipitant is treatable; precipitant aetiology thus appears to carry prognostic information not captured by score magnitude alone.[15] Finally, the largely reversible, treatment-responsive nature of scrub-typhus- and leptospirosis-associated hepatic and renal injury described in dedicated tropical-infection series is consistent with our observation that the tropical subgroup, although presenting with higher creatinine, achieved substantially better 28-day survival once doxycycline was instituted.[16] Our results also align with the North American NACSELD experience, in which the number of extrahepatic organ failures- rather than the identity of the precipitant alone- governed survival in infection-related ACLF; the predominance of renal-dominant, single-organ (grade 1) failure in our tropical

subgroup is mechanistically consistent with its lower observed mortality.[17] The high admission creatinine in tropical-precipitated patients likewise echoes evidence that acute kidney injury complicating ACLF is pathophysiologically distinct from- and, when driven by a treatable infection, more readily reversible than- the acute kidney injury seen in cirrhosis with simple acute decompensation.[18]

Limitations must be acknowledged. The study is single-centre and the tropical subgroup is modest (n=17), limiting statistical power and producing wide confidence intervals around the mortality difference. The overall tropical-versus-non-tropical comparison did not cross the conventional 0.05 threshold (p=0.091), although the pairwise tropical-versus-alcohol comparison did (p=0.024). Serological diagnosis is timing- and operator-dependent, and one patient was classified on clinical grounds alone. Ninety-day mortality was not captured. The findings reflect a monsoon-spanning tertiary-care enrolment window and should be interpreted with caution outside comparable endemic settings. External validation in larger multicentre cohorts with prospective doxycycline-protocol enrolment is needed.

CONCLUSION

In a Northeast Indian tertiary cohort with monsoon-season enrolment, tropical infections- predominantly leptospirosis and scrub typhus- precipitated 24.3% of EASL-CLIF ACLF episodes. The tropical subgroup presented at lower severity grades, with significantly higher admission creatinine and numerically lower inflammatory markers, and carried a 27-percentage-point absolute mortality advantage over non-tropical ACLF (35.3% versus 62.3% at 28 days), with significance reached against alcohol-precipitated ACLF (OR 0.19, p=0.024). Because both precipitants are doxycycline-responsive, tropical-precipitated ACLF represents a distinct, partially reversible phenotype. Empirical doxycycline alongside standard broad-spectrum antibiotics in any ACLF patient with suspected leptospirosis or scrub typhus in endemic regions deserves prospective evaluation as a routine protocol.

ACKNOWLEDGEMENTS

The authors thank the patients and their families who consented to participate in this study.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee Assam Medical College and Hospital, Dibrugarh

REFERENCES

1. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, et al. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. Gastroenterology. 2013;144:1426-37.
2. Arroyo V, Moreau R, Jalan R. Acute-on-chronic liver failure. N Engl J Med. 2020;382:2137-45.
3. Sarin SK, Choudhury A, Sharma MK, Maiwall R, Al Mahtab M, Rahman S, et al. Acute-on-chronic liver failure: consensus recommendations of the APASL: an update. Hepatol Int. 2019;13:353-90.
4. Choudhury A, Jindal A, Maiwall R, Sharma MK, Sharma BC, Pamecha V, et al. Liver failure determines the outcome in patients of acute-on-chronic liver failure (ACLF): comparison of AARC and CLIF-SOFA models. Hepatol Int. 2017;11:461-71.
5. 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;9:e0003898.
6. Devasagayam E, Dayanand D, Kundu D, Kamath MS, Kirubakaran R, Varghese GM. The burden of scrub typhus in India: a systematic review. PLoS Negl Trop Dis. 2021;15:e0009619.
7. Charan J, Saxena D, Mulla S, Yadav P. Antibiotics for the treatment of leptospirosis: systematic review and meta-analysis of controlled trials. Int J Prev Med. 2013;4:501-10.
8. Medhi PK, Choudhury BN, Bhattacharyya M, Deka UJ, Baruah BJ. ACLF in Northeast India: underlying cause, precipitating factors and their effect on mortality. J Clin Exp Hepatol. 2024;14:102099.
9. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on acute-on-chronic liver failure. J Hepatol. 2023;79:461-91.
10. Bone RC, Grodzin CJ, Balk RA. Sepsis: a new hypothesis for pathogenesis of the disease process. Chest. 1997;112:235-43.
11. Daher EF, da Silva GB Jr, Karbage NN, Carvalho PR Jr, Kataoka RS, Silva EC, et al. Predictors of oliguric acute kidney injury in leptospirosis. Nephron Clin Pract. 2009;112:c25-30.
12. Vikrant S, Dheer SK, Parashar A, Gupta D, Thakur S, Sharma A, et al. Scrub typhus associated acute kidney injury- a study from a tertiary care hospital from western Himalayan State of India. Ren Fail. 2013;35:1338-43.
13. Varghese GM, Trowbridge P, Janardhanan J, Thomas K, Peter JV, Mathews P, et al. Clinical profile and improving mortality trend of scrub typhus in South India. Int J Infect Dis. 2014;23:39-43.
14. Shalimar, Saraswat V, Singh SP, Duseja A, Shukla A, Acharya SK, et al. Acute-on-chronic liver failure in India: the Indian National Association for Study of the Liver consortium experience. J Gastroenterol Hepatol. 2016;31:1742-9.
15. Jalan R, Saliba F, Pavesi M, Amorós A, Moreau R, Ginès P, et al. Development and validation of a prognostic score to predict mortality in patients with acute-on-chronic liver failure. J Hepatol. 2014;61:1038-47.

16. Chauhan R, Ahmad S, Goyal C, Tewatia P. Hepatopathy in scrub typhus: clinical presentation, association with morbidity and impact on outcome. Cureus. 2024;16:e52316.
17. Bajaj JS, O'Leary JG, Reddy KR, Wong F, Biggins SW, Patton H, et al. Survival in infection-related acute-on-chronic liver failure is defined by extrahepatic organ failures. Hepatology. 2014;60:250-6.
18. Maiwall R, Kumar S, Chandel SS, Kumar G, Rastogi A, Bihari C, et al. AKI in patients with acute on chronic liver failure is different from acute kidney injury in patients with cirrhosis and acute decompensation. Liver Int. 2015;35:2418-28.

Cite this article as: Saravanamuthu PK, Das AK, Rajkakati R, Sonowal A. Tropical-infection-precipitated acute-on-chronic liver failure: a distinct, lower-mortality phenotype in a Northeast Indian cohort. Int J Res Med Sci 2026;14:3399-405.