RESEARCH PAPER

Correlation Between Serum Lactate Dehydrogenase Levels and Disease Severity in Dengue: A Cross-Sectional Study from a Tertiary Care Center

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ABSTRACT

Background: Dengue shows a wide clinical spectrum, and early identification of patients who may deteriorate remains difficult in routine ward settings. Serum lactate dehydrogenase (LDH), a marker of cellular injury, has been explored as a pragmatic biomarker in dengue.

Methods: In this hospital-based cross-sectional study, 120 laboratory-confirmed dengue patients were enrolled. Severity was classified using the WHO 2009 dengue classification. Serum LDH was measured at admission. Group differences in LDH were analysed, and correlation between LDH and severity grade was assessed.

Results: Of 120 patients, 52 (43.3%) had dengue without warning signs, 39 (32.5%) had dengue with warning signs, and 29 (24.2%) had severe dengue. Mean LDH increased progressively across groups: 200.4 ± 43.2 IU/L (without warning signs), 285.7 ± 56.5 IU/L (with warning signs), and 430.7 ± 63.1 IU/L (severe dengue) ($p < 0.001$).

Conclusion: Serum LDH at admission was significantly associated with dengue severity. While LDH may aid early risk stratification, its clinical utility as a stand-alone prognostic tool requires confirmation in prospective studies with serial measurements.

Keywords: dengue; lactate dehydrogenase; severity; biomarker; cross-sectional study

How to cite this article: Raghavendra PB, Madhusudhan J, Shashikantha, Raghavendra PB. Correlation Between Serum Lactate Dehydrogenase Levels and Disease Severity in Dengue: A Cross-Sectional Study from a Tertiary Care Center. Int J Drug Deliv Technol. 2026;16(58s): 732-737. DOI: 10.25258/ijddt.16.58s.78

DOI: xxxx

Introduction

India sees dengue every year, but the clinical problem is rarely "just dengue". In a busy ward during monsoon season, the first question is simple: who will settle with fluids and observation, and who is quietly sliding towards plasma leakage, bleeding, or organ dysfunction? That uncertainty sits at the heart of dengue care in tertiary hospitals.

Dengue virus infection has expanded markedly in its global footprint over the last two decades, and India contributes a substantial share of reported and unreported disease because of dense urbanisation, vector ecology, and seasonal transmission patterns.[1][2] Even within the same outbreak, presentation ranges from an undifferentiated febrile illness to life-threatening shock and end-organ injury.[3][4] The World Health Organization's 2009 classification (dengue without warning signs, dengue with warning signs, and severe dengue) was designed to bring order to this heterogeneity and to support pragmatic triage at the bedside.[5]

Yet classification alone does not eliminate uncertainty. Warning signs improve decision-making, but their prognostic performance varies by setting, age group, and admission thresholds, particularly in adult cohorts where comorbidity and delayed presentation are common.[6]

Lactate dehydrogenase (LDH) is a plausible adjunct in this space. LDH is a ubiquitous cytosolic enzyme released with cellular injury, and in dengue it can rise with hepatocellular injury, skeletal muscle involvement, haemolysis, and hypoperfusion-associated tissue stress.[7]

Prior work has suggested that higher LDH levels are more frequent in patients with haemorrhagic manifestations and shock, and that values around the early critical phase may track more severe disease biology.[8]

Still, the practical question remains: in our hospital population, does LDH measured at presentation show a meaningful gradient across WHO 2009 severity categories? If it does, LDH might support triage decisions as a complement to clinical judgement, rather than a stand-alone "prognostic tool". The present study therefore assessed the association between admission LDH and dengue severity (WHO 2009 classification) in a tertiary-care cohort.

Materials and Methods

Study design and setting: A hospital-based observational cross-sectional study was conducted at Adichunchanagiri Hospital and Research Centre (tertiary care teaching

hospital). The study period spanned 18 months (May 2024 to October 2025).

Participants: Adults with laboratory-confirmed dengue infection (NS1 antigen and/or IgM antibody positive) admitted during the acute phase were included. Exclusion criteria were chronic liver disease, haemolytic disorders, malignancy, myopathies, coinfections, and medications known to influence LDH levels.

Severity classification: Patients were categorised as dengue without warning signs, dengue with warning signs, or severe dengue as per WHO 2009 dengue classification.

LDH measurement: Serum LDH was measured at the time of admission using the hospital laboratory's standardised assay and reported in IU/L.

Statistical analysis: Continuous variables were summarised as mean ± standard deviation, and categorical variables as frequency and percentage. Normality of LDH distribution within severity groups was assessed (Shapiro–Wilk). Homogeneity of variances was checked (Levene's test). If assumptions were satisfied, one-way ANOVA was used to compare mean LDH across severity groups; otherwise, the Kruskal–Wallis test was planned. Correlation between LDH and ordered severity grade was assessed using Pearson's correlation for approximately normal data (or Spearman's rank correlation if non-normal). A two-sided p value <0.05 was considered statistically significant.

Ethical considerations: The study was approved by the Institutional Ethics Committee of Adichunchanagiri Institute of Medical Sciences (AIMS/IEC/025/2024; dated 01-04-2024; IEC registration no. EC/NEW/INST/2023/KA/0382). Written informed consent was obtained from participants prior to enrolment.

Results

A total of 120 patients were included. Females constituted 55.8% (n=67) and males 44.2% (n=53). Fever was universal (100%). Icterus (52.5%) and abdominal pain/tenderness (35.8%) were frequent, while overt bleeding manifestations were uncommon (5.8%).

Table 1. Distribution of patients according to dengue severity (WHO 2009).

Severity category	n	%
Dengue without warning signs	52	43.3
Dengue with warning signs	39	32.5
Severe dengue	29	24.2
Total	120	100.0

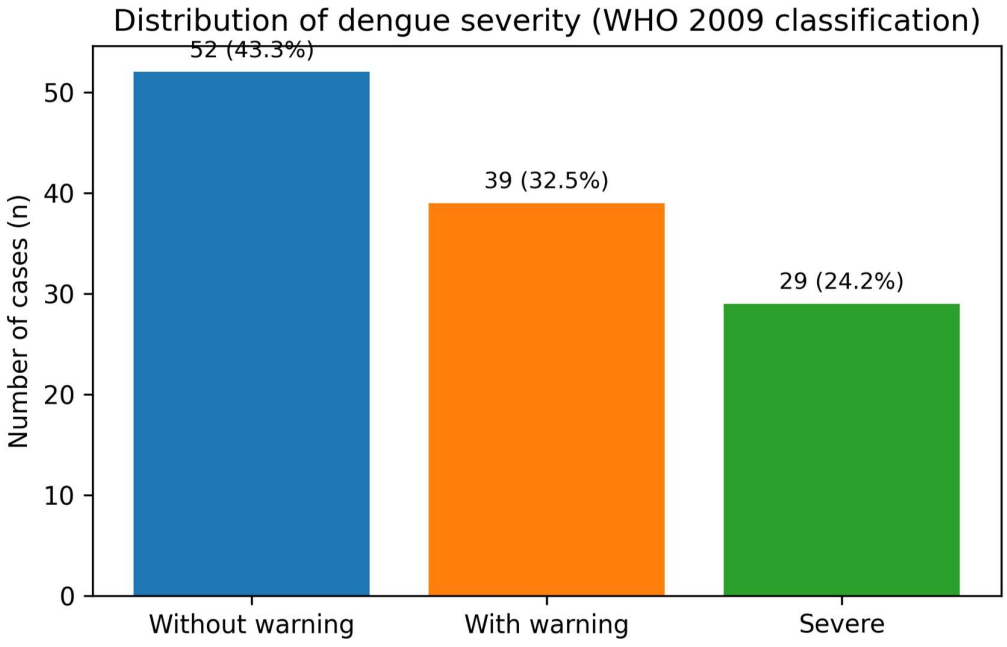

Figure 1. Distribution of dengue severity categories in the study cohort (values shown as n and %).

Table 2. Mean serum LDH levels across dengue severity groups.

Severity category	Mean LDH (IU/L)	SD
Dengue without warning signs	200.4	43.2
Dengue with warning signs	285.7	56.5

Severity category	Mean LDH (IU/L)	SD
Severe dengue	430.7	63.1

Mean LDH differed significantly across the three groups (overall $p < 0.001$).

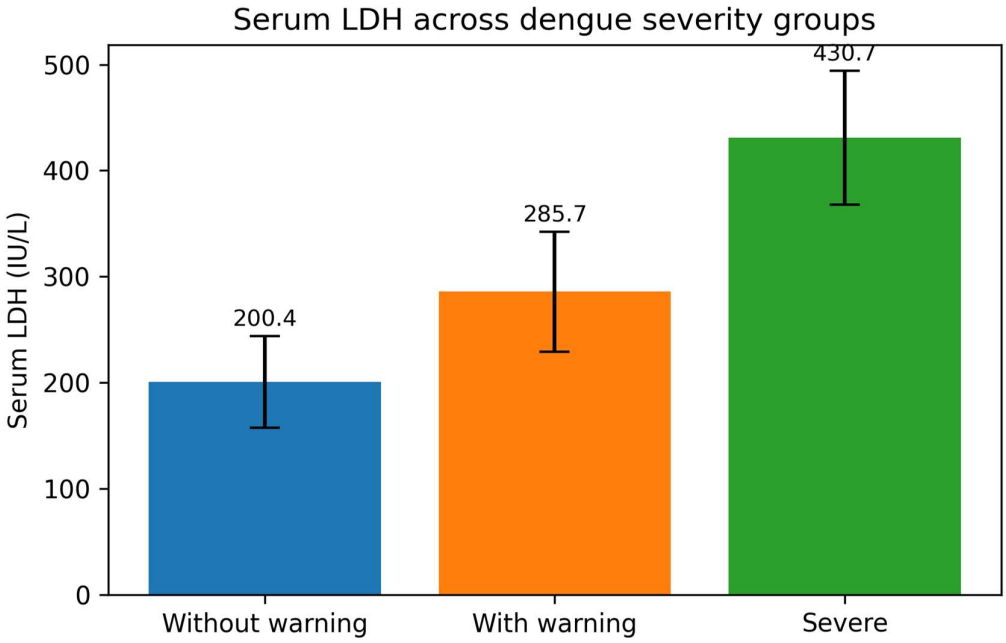

Figure 2. Mean serum LDH by dengue severity (error bars represent SD).

Additional laboratory associations are summarised below to strengthen clinical interpretability (Table 3–4; Figure 3–4).

Severity group	Platelets (cells/µL) Mean ± SD	PT (sec) Mean ± SD	aPTT (sec) Mean ± SD	Test	p-value	Interpretation
Without warning signs	85,660.59 ± 18,369.08	12.12 ± 1.20	34.90 ± 3.37	ANOVA	<0.001	Progressive thrombocytopenia and coagulopathy
With warning signs	71,133.69 ± 19,681.60	13.79 ± 1.70	40.08 ± 3.77	ANOVA	<0.001	Intermediate derangements
Severe dengue	62,553.42 ± 22,166.93	16.59 ± 1.38	46.66 ± 3.54	ANOVA	<0.001	Most pronounced prolongation

Table 3. Platelet and coagulation parameters across dengue severity groups.

Severity group	Bilirubin (mg/dL) Mean ± SD	SGOT (U/L) Mean ± SD	SGPT (U/L) Mean ± SD	Albumin (g/dL) Mean ± SD	p-value
Without warning signs	0.84 ± 0.20	58.60 ± 18.80	38.70 ± 12.40	3.92 ± 0.28	<0.001
With warning signs	1.61 ± 0.29	157.18 ± 41.47	83.13 ± 22.88	3.35 ± 0.26	<0.001
Severe dengue	4.14 ± 0.77	407.21 ± 120.55	223.28 ± 91.42	2.64 ± 0.32	<0.001

Table 4. Liver function markers across dengue severity groups.

Platelet counts declined stepwise from dengue without warning signs to severe dengue, while PT and aPTT rose in parallel, suggesting an advancing haemostatic disturbance with clinical worsening (Table 3). Figure 3 visualises the same gradient, with the lowest mean platelet count observed in the severe dengue group.

Biochemical liver involvement also tracked with severity. Mean bilirubin, SGOT, and SGPT values increased sharply in severe dengue, while albumin showed a progressive fall (Table 4). Figure 4 summarises these shifts across groups, underscoring a predominantly hepatocellular injury pattern rather than an isolated cholestatic picture.

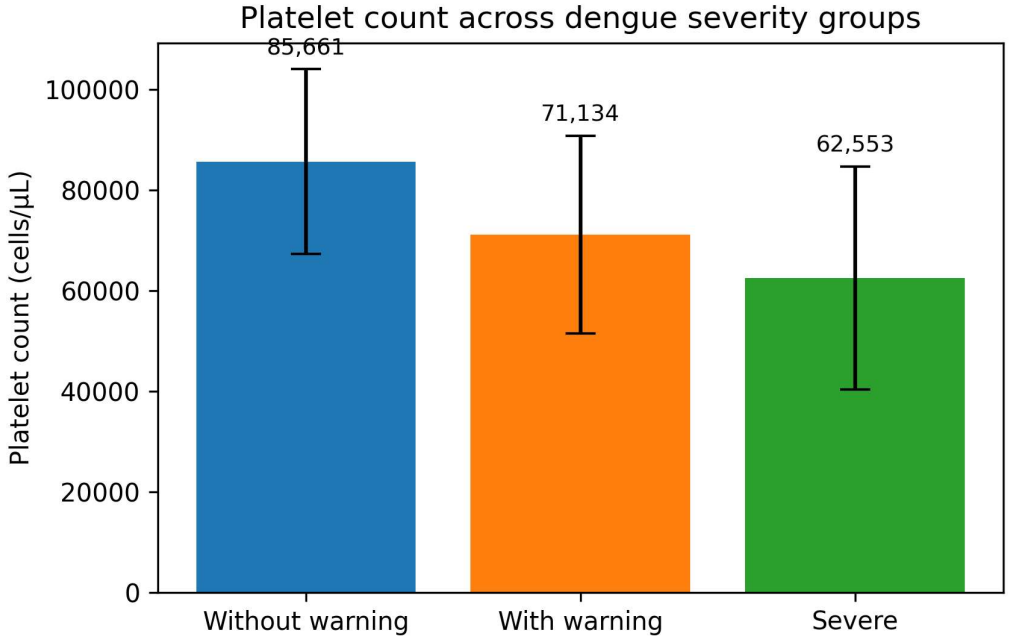

Figure 3. Platelet count (mean ± SD) across dengue severity groups.

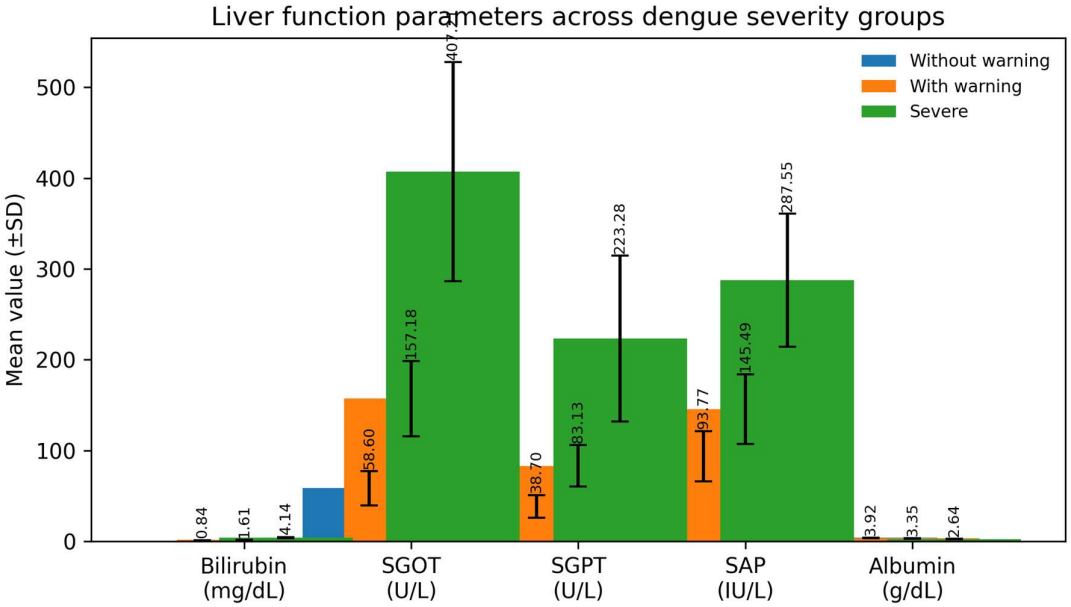

Figure 4. Liver function parameters (mean ± SD) across dengue severity groups.

Discussion

Our main observation is straightforward: mean LDH at admission increased stepwise across WHO 2009 severity categories, with severe dengue showing roughly a two-fold higher mean LDH than dengue without warning signs (430.7 vs 200.4 IU/L). In day-to-day clinical terms, that pattern matters because it suggests LDH is not behaving like random noise; it is moving in the same direction as clinical deterioration.

Mechanistically, this is plausible. Severe dengue is a multi-system process: vascular leak, inflammatory endothelial activation, and organ stress occur together, and LDH becomes a composite signal of cellular injury across tissues rather than a single-organ biomarker.[9] When hepatic injury is prominent, LDH can rise alongside aminotransferases, as shown in dengue-associated acute liver failure cohorts where LDH correlated strongly with AST and ALT.[10]

A numerical comparison with earlier studies helps place magnitude. In a Thai cohort, Sirikutt and Kalayanarooj reported higher LDH levels across classical severity groups and discussed practical cut-offs: LDH >500 IU/L to support early differentiation of dengue from other febrile illness, and values approaching ~1,000 IU/L near the start of leakage as a warning signal for severe physiology.[8] Our severe dengue mean (430.7 IU/L) sits below 500 IU/L. This does not necessarily contradict the biology. LDH values are sensitive to timing of sampling, assay platform, age structure, and case-mix. A cohort enriched with earlier admissions or less profound shock would be expected to show lower means even if the directionality remains intact.

More recent hospital-based work has also linked LDH with complications and thrombocytopenia, but effect sizes vary across centres. Kasarabada and colleagues (Medical Journal Armed Forces India) described LDH at presentation as a prognostic marker and demonstrated statistically significant associations between LDH and clinical course variables in hospitalized dengue patients.[11] Although their outcomes and modelling strategy differ from our cross-sectional approach, the shared theme is consistent: LDH tends to rise when disease becomes clinically complicated.

Our results sit within the broader literature on dengue triage. Systematic reviews of WHO severe dengue phenotypes highlight that higher-risk patients cluster with organ involvement, plasma leak manifestations, and marked laboratory derangements.[12] LDH is not part of the formal warning sign list, but in a crowded emergency bay it can still be useful as a "stress marker" when interpreted with vitals, warning signs, haematocrit trends, and platelet trajectory.

The design, however, sets clear limits. This is an observational cross-sectional analysis of admission values, so LDH should be interpreted as associated with severity at presentation, not as a validated prognostic tool of future progression. Before any threshold-based triage rule is suggested, prospective cohorts with serial LDH measurements through the febrile-to-critical transition and hard endpoints (shock, imaging-confirmed plasma leakage, ICU admission) are needed.[6][13]

Taken together, the findings support a cautious, clinically grounded message: in a tertiary-care Indian setting, higher admission LDH accompanies higher dengue severity category, and LDH may complement clinical assessment when deciding intensity of monitoring.

Conclusion

Admission LDH showed a graded association with dengue severity categories, rising from dengue without warning signs to severe dengue. In practice, LDH may function as a simple adjunct marker of systemic cellular injury that supports bedside risk stratification, particularly when combined with WHO warning signs and routine laboratory trends. Because this study is cross-sectional and uses a single-time-point measurement, LDH should not be framed as a definitive prognostic tool of progression; confirmation in prospective cohorts with serial testing and clinical endpoints is required.

Declarations

Ethical approval: The study was approved by the Institutional Ethics Committee, Adichunchanagiri Institute of Medical Sciences, B.G. Nagara (AIMS/IEC/025/2024; dated 01-04-2024; IEC Registration No. EC/NEW/INST/2023/KA/0382).

Funding: No external funding was received.

Conflict of interest: The authors declare no conflict of interest.

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