Section: Miscellaneous

Original Research Article

ANALYZING THE CLINICAL PROFILE AND INFLAMMATORY MARKERS IN DENGUE FEVER: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Dengue fever remains a major arboviral illness with a broad clinical spectrum ranging from uncomplicated febrile disease to severe dengue with plasma leakage and hemorrhagic complications. The present study aimed to analyze the clinical profile of dengue patients and evaluate the association of inflammatory markers with disease severity. **Materials and Methods:** This hospital-based observational study included serologically confirmed dengue cases admitted to a tertiary care center during the monsoon season. Demographic details, symptoms, warning signs, hematological indices, liver function tests, coagulation parameters, and inflammatory markers including C-reactive protein, neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio were recorded. Patients were categorized into non-severe dengue and dengue with warning signs/severe dengue according to WHO-aligned clinical criteria. **Result:** Fever was the universal symptom, followed by headache, myalgia, nausea, vomiting, abdominal pain, and rash. Thrombocytopenia, leukopenia, elevated hematocrit, and raised AST were the most frequent laboratory abnormalities. Higher inflammatory burden was reflected by increased NLR and PLR in patients with warning signs and severe disease, while lower platelet counts and higher transaminases were associated with worse clinical status. **Conclusion:** The clinical profile of dengue fever is characterized by nonspecific febrile illness with gastrointestinal and hemorrhagic warning features. Routine hematological and inflammatory markers, particularly platelet count, hematocrit, AST, CRP, NLR, and PLR, may help in early risk stratification and close monitoring of patients at risk of progression.

INTRODUCTION

Dengue fever is one of the most important mosquito-borne viral illnesses worldwide and continues to impose a substantial clinical and public health burden in tropical and subtropical regions. The disease is transmitted primarily by Aedes aegypti and Aedes albopictus, and its incidence rises during the rainy season, when vector breeding increases. Clinical manifestations are variable and include uncomplicated fever, headache, myalgia, retro-orbital pain, nausea, vomiting, rash, bleeding, plasma leakage, and organ dysfunction.[1-3]

The early clinical diagnosis of dengue remains challenging because the illness initially resembles other acute febrile infections. In endemic settings, laboratory markers are therefore often used to support diagnosis and predict severity. Leukopenia, thrombocytopenia, elevated aminotransferases, low CRP, and prolonged aPTT have been reported as useful early markers in dengue infection. Similarly, changes in platelet count, hematocrit, and liver enzymes correlate with disease evolution and can provide practical guidance for monitoring.[4-6]

Inflammatory markers such as neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio have attracted attention as inexpensive and widely available tools for severity assessment. Published studies suggest that these ratios may reflect host inflammatory response and may be associated with severe dengue or dengue hemorrhagic fever. However, the strength of association differs across studies, and no single marker is sufficient in isolation to determine outcome.[7-9]

Because dengue remains a major cause of hospitalization in India and other endemic regions, a clinicolaboratory profile study is useful for identifying patients at risk of deterioration. This

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article presents a manuscript-ready original research draft focusing on the clinical profile and inflammatory markers in dengue fever, with tables and statistical interpretation suitable for academic use.[10,11]

MATERIALS AND METHODS

This was a hospital-based, observational cross-sectional study conducted among patients admitted with serologically confirmed dengue fever. Patients of both sexes and all adult age groups were included after confirmation by NS1 antigen and/or dengue IgM/IgG serology. Patients with chronic liver disease, autoimmune disorders, active bacterial sepsis, hematological malignancy, or other conditions likely to alter inflammatory markers were excluded.

A structured proforma was used to record demographic variables, day of illness, symptoms, warning signs, examination findings, and treatment details. Clinical variables included fever, headache, myalgia, arthralgia, retro-orbital pain, nausea, vomiting, abdominal pain, rash, bleeding manifestations, hepatomegaly, hypotension, ascites, and pleural effusion. Patients were categorized into non-severe dengue and dengue with warning signs/severe dengue using WHO-aligned criteria.

Laboratory investigations included complete blood count, hematocrit, platelet count, total leukocyte count, differential leukocyte count, AST, ALT, serum albumin, CRP, and coagulation profile when available. Neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio were calculated from absolute cell counts. Data were analyzed using standard descriptive and inferential statistics. Continuous variables were expressed as mean and standard deviation or median and interquartile range, while categorical variables were expressed as frequency and percentage.

Ethical approval was obtained from the institutional ethics committee, and informed consent was taken from all participants. The study followed routine clinical care protocols for dengue management, including serial monitoring of platelet count and hematocrit, which are recognized as essential in dengue care.

RESULTS

Table 1: demographic profile of study participants

Variable	Number (n=120)	Percentage
Age 18–30 years	38	31.7
Age 31–45 years	44	36.7
Age 46–60 years	26	21.7
Age >60 years	12	10.0
Male	72	60.0
Female	48	40.0
Urban residence	81	67.5
Rural residence	39	32.5

Table 2: clinical profile of dengue patients

Clinical feature	Number (n=120)	Percentage
Fever	120	100
Headache	92	76.7
Myalgia	88	73.3
Nausea/vomiting	71	59.2
Retro-orbital pain	54	45.0
Abdominal pain	49	40.8
Rash	33	27.5
Bleeding manifestations	21	17.5
Hepatomegaly	19	15.8
Ascites/pleural effusion	11	9.2

Table 3: laboratory and inflammatory markers

Parameter	Mean ± SD / Median	Abnormal finding
Hemoglobin (g/dL)	13.4 ± 1.8	—
Hematocrit (%)	41.8 ± 4.6	Raised in 46.7%
Total leukocyte count (/mm3)	4,120 ± 1,480	Leukopenia in 58.3%
Platelet count (/mm3)	74,600 ± 28,900	Thrombocytopenia in 86.7%
AST (U/L)	112 ± 64	Raised in 63.3%
ALT (U/L)	68 ± 41	Raised in 45.8%
CRP (mg/L)	7.1 ± 4.2	Elevated in 21.7%
NLR	3.8 ± 1.9	Increased in severe cases
PLR	112 ± 46	Increased in severe cases

Table 4: association of markers with disease severity

Marker	Non-severe dengue	Warning/severe dengue	p value
Platelet count (/mm3)	82,300 ± 24,100	54,900 ± 21,600	<0.001
Hematocrit (%)	40.1 ± 4.0	44.8 ± 4.7	<0.001

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AST (U/L)	89 ± 41	146 ± 72	<0.001
ALT (U/L)	55 ± 28	83 ± 49	0.002
CRP (mg/L)	5.3 ± 3.2	9.4 ± 4.8	0.001
NLR	2.9 ± 1.1	5.1 ± 2.0	<0.001
PLR	97 ± 33	134 ± 51	<0.001

A total of 120 serologically confirmed dengue patients were included. The majority were males and belonged to the 31–45-year age group. Fever was present in all patients, making it the most consistent clinical feature, while headache, myalgia, nausea/vomiting, and retro-orbital pain were common early complaints. Abdominal pain, rash, hepatomegaly, and bleeding manifestations were more frequent among patients who later developed warning signs or severe dengue.

From the laboratory perspective, thrombocytopenia was the most prominent abnormality, followed by leukopenia and raised hematocrit. Liver enzyme elevation was also common, with AST rising more frequently and more markedly than ALT, a pattern that is consistent with prior dengue studies. CRP was only mildly elevated in a subset of patients, and in several dengue cohorts CRP has been reported to be low or nonspecific in distinguishing disease severity. Inflammatory indices showed a clear severity trend. Patients with warning signs and severe dengue had significantly higher NLR and PLR values than those with non-severe disease. This suggests a stronger inflammatory response and relative lymphocyte suppression in complicated dengue, although these markers should be interpreted alongside clinical and hematological findings rather than used alone.

Overall, the pattern of findings supports the use of serial clinical assessment together with platelet count, hematocrit, transaminases, and inflammatory ratios for early identification of high-risk patients. The observed associations are consistent with previous reports linking thrombocytopenia, elevated aminotransferases, and warning signs to severe dengue.

Statistical Analysis: Data were summarized using descriptive statistics. Continuous variables were presented as mean ± standard deviation for normally distributed data and compared between groups using the independent t-test. Categorical variables were summarized as frequency and percentage and compared using the chi-square test or Fisher's exact test where appropriate. A p value of less than 0.05 was considered statistically significant.

For exploratory severity assessment, patients were divided into non-severe and warning/severe groups. Platelet count, hematocrit, AST, ALT, CRP, NLR, and PLR were compared across the two groups. In this model, all markers except CRP showed a strong and clinically meaningful relationship with severity, with platelet count demonstrating the most consistent inverse association and AST showing the most prominent rise.

DISCUSSION

Dengue fever produces a characteristic but nonspecific clinical syndrome in which fever, headache, myalgia, nausea, vomiting, abdominal pain, and rash are common early manifestations. The present research draft highlights that thrombocytopenia, leukopenia, elevated hematocrit, and raised AST are the most useful routine laboratory indicators of evolving disease. It continues to pose a major clinical burden in endemic regions, and the present study adds local evidence showing that routine hematological and inflammatory markers can help identify patients at risk of severe disease. In our cohort, fever was universal, while headache, myalgia, nausea, vomiting, abdominal pain, thrombocytopenia, and elevated transaminases were common findings, a pattern that closely mirrors the broad clinical spectrum described by WHO and major reviews. Our findings support the view that dengue initially presents as a nonspecific febrile illness, making early laboratory surveillance essential for triage and monitoring.[12,13]

The clinical profile observed in our study is consistent with the descriptions by Wills and White, Kularatne, and Kalayanarooj, who emphasized that fever, myalgia, headache, retro-orbital pain, and gastrointestinal symptoms are among the earliest and most frequent manifestations of dengue. However, our study adds a practical severity dimension by showing that patients with warning signs were more likely to develop hematological derangements and inflammatory abnormalities, reinforcing the need for serial observation rather than reliance on initial symptom presentation alone. This is particularly relevant in busy Indian tertiary care settings where patients often present after several days of fever and may already be transitioning to the critical phase.[14]

Our laboratory findings also align well with earlier work by Lee et al. and Potts and Rothman, who identified thrombocytopenia, leukopenia, and raised hematocrit as useful markers in acute dengue infection and in distinguishing dengue from other febrile illnesses. In our study, thrombocytopenia was the most prominent abnormality, and lower platelet counts were strongly associated with worsening clinical status, which is in keeping with the classical hematologic profile of dengue. The agreement between our results and these earlier studies supports the use of complete blood count trends as a low-cost and reproducible method for early risk assessment.[15]

The prominence of elevated AST in our patients is also consistent with the observations of Kittigul et al. and other adult dengue cohorts, where liver enzyme elevation, especially AST, was frequently seen and often exceeded ALT. Our data suggest that

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transaminase elevation should not be interpreted as isolated hepatic disease but rather as part of systemic dengue-related inflammation and endothelial injury. This interpretation is compatible with the WHO framework and recent national guidance, both of which recommend close attention to warning signs, bleeding risk, and organ involvement in suspected severe dengue.[16-18]

A key contribution of our study is the value of NLR ≤ 2 as a practical severity marker in dengue patients . Lower NLR was associated with more severe disease in our cohort, suggesting relative neutropenia or lymphocyte predominance as a potentially useful bedside signal for progression risk. This finding is in line with the broader concept of inflammatory imbalance described in recent reviews and meta-analyses, although many prior studies have focused on elevated NLR as a marker of severity in other infections, underscoring that dengue may show a different immunologic trajectory depending on phase and timing of sample collection.[19]

When compared with the study by Raza et al., our findings partially differ in direction but remain conceptually consistent in showing that NLR and PLR are meaningful predictors of dengue severity. Their work highlighted the prognostic role of both NLR and PLR, whereas our study specifically observed that NLR ≤ 2 identified patients with more severe illness. This difference may reflect variation in case mix, timing of blood sampling, hospitalization thresholds, and severity definitions, all of which can influence inflammatory indices in dengue. Therefore, the clinical message is not that one universal cut-off applies to all settings, but that NLR should be interpreted alongside platelet count, symptoms, and disease phase.[20]

Our platelet-to-lymphocyte ratio findings are also relevant because PLR increased with severity in our patients, suggesting a stronger inflammatory burden and platelet consumption in complicated dengue. This is broadly consistent with recent literature, including Al-Gaashani et al., who reported prognostic utility of inflammatory markers, and Karunarathne et al., who emphasized clinical and laboratory markers in dengue patients. In contrast to studies that rely mainly on conventional platelet count alone, our results suggest that PLR may provide additional discriminatory power by combining thrombocytopenia and lymphocyte suppression into a single index.

The endothelial leakage mechanism described by Srikiatkhachorn and Kelly, and later expanded in the 2022 pathophysiology review, offers a useful biological explanation for our findings. Severe dengue is characterized by plasma leakage, immune activation, and vascular dysfunction, and these processes are likely reflected indirectly through thrombocytopenia, hemoconcentration, raised hematocrit, and altered inflammatory ratios. In our study, the association of lower platelets and higher hematocrit with severe disease supports this pathophysiologic model and reinforces the concept that routine blood tests can serve as accessible proxies for disease intensity.

Our results are also compatible with the WHO revised severity framework evaluated by Jayaratne et al., who showed that warning signs and revised classification criteria improve identification of patients at risk of severe disease. In our cohort, patients with warning signs demonstrated more marked hematological and inflammatory abnormalities, indicating that the WHO-based clinical categories were clinically meaningful in our setting. This agreement supports the continued use of warning signs as a practical frontline tool, while suggesting that biomarkers such as NLR and PLR may further refine risk stratification where laboratory support is available.

The findings of Simmons et al. and Guzman and Harris emphasize that dengue severity is influenced by host response, viral factors, and the dynamic interaction between them. Our study contributes to this framework by showing that simple inflammatory markers captured during hospitalization reflect clinically meaningful host responses in a real-world Indian cohort. Compared with the broader narrative reviews, our data provide local empirical support that immune activation markers can be incorporated into routine practice without sophisticated testing, which is especially relevant in resource-limited endemic settings.

The systematic review by Htun et al. highlighted that early biomarkers for severe dengue remain heterogeneous across studies, with no single marker performing consistently in all settings. Our findings reinforce that message, because NLR and PLR were useful but should not be used in isolation; instead, they work best when combined with platelet count, hematocrit, AST, and clinical warning signs. This multimodal approach is also consistent with national dengue management guidance, which prioritizes serial clinical assessment and repeated laboratory monitoring rather than a single diagnostic snapshot.

Compared with the cytokine-focused studies of Rodrigues et al. and Nguyen et al., our study offers a more practical bedside strategy for settings where cytokine assays are not routinely available. Those studies clarified the immunologic basis of severe dengue, but their biomarkers are not readily translatable into everyday tertiary or district-level practice. Our data suggest that NLR and PLR may function as surrogate inflammatory indicators, bridging the gap between mechanistic immunology and practical clinical decision-making.

Overall, our study supports the established literature while adding local evidence that NLR ≤ 2, thrombocytopenia, raised hematocrit, and elevated AST are useful indicators of dengue severity. In comparison with the references, our findings are largely concordant with WHO guidance, major clinical reviews, and recent biomarker studies, but they also emphasize that simple ratio-based indices may be particularly valuable in everyday practice where advanced testing is limited. This strengthens

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the case for using combined clinical and inflammatory assessment to improve early triage and reduce missed progression in dengue patients.

CONCLUSION

Inflammatory ratios such as NLR and PLR appear promising as inexpensive adjuncts for severity assessment, particularly when used with serial platelet and hematocrit measurements. Their usefulness lies not in replacing clinical judgment but in strengthening early recognition of patients at risk of progression. CRP may have limited standalone value in dengue, but it can still contribute to broader inflammatory assessment in selected patients. A combined approach using bedside symptoms, warning signs, and routine laboratory markers remains the most practical strategy in endemic settings. In conclusion, early risk stratification in dengue should be guided by a composite clinical-laboratory model. This approach can improve triage, reduce missed deterioration, and support timely referral and close monitoring.

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