Section: Medicine

Systematic Review Article

DYNAMIC CHANGES IN HEMATOLOGICAL PARAMETERS DURING DENGUE INFECTION: A SYSTEMATIC REVIEW

Ayman Ahmed[1], *Ibrahimova Gulnara*[2]

[1]*Medical graduate, Azerbaijan Medical University, Baku, Azerbaijan.*
[2]*Candidate of Medical Sciences, Associate Professor, Department of Infectious Diseases, Azerbaijan Medical University, Baku, Azerbaijan.*

Received *: 15/01/2026*
Received in revised form *: 20/02/2026*
Accepted *: 11/03/2026*

Corresponding Author:
Dr. Ibrahimova Gulnara,
Candidate of Medical Sciences, Associate Professor, Department of Infectious Diseases, Azerbaijan Medical University, Baku, Azerbaijan.

Email: ayman.ahmed2k@gmail.com

DOI: 10.70034/ijmedph.2026.1.461

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (1); 2681-2687

ABSTRACT

Background: Dengue remains a significant public health problem in tropical and subtropical regions and is marked by dynamic hematological changes such as thrombocytopenia, hemoconcentration, leukopenia, and altered differential counts. These parameters vary across disease phases and may indicate severity and prognosis. However, a comprehensive synthesis of their temporal trends and clinical relevance is lacking. This review aims to systematically summarize the progression and prognostic significance of hematological changes in dengue infection. **Objectives:** The objective of this study is to systematically review and synthesize the available evidence on dynamic hematological changes occurring during different phases of dengue infection, evaluate the association between key hematological parameters such as platelet count, hematocrit, and leukocyte count with disease severity, and identify potential hematological markers that may serve as predictors of complications and clinical outcomes.
Material and Methods: This study was conducted as a systematic review in accordance with PRISMA guidelines. A comprehensive search of PubMed, Scopus, and Web of Science was performed using predefined inclusion and exclusion criteria to identify relevant observational and clinical studies on hematological parameters in laboratory-confirmed dengue cases. Data on platelet count, hematocrit, leukocyte indices, and related markers were extracted and analyzed across disease phases and severity levels. Study quality was critically appraised, and findings were synthesized qualitatively.
Results: Thrombocytopenia was the most consistent finding and was frequently associated with disease severity, bleeding, and progression to dengue hemorrhagic fever (DHF), with platelet count showing predictive value for severe outcomes. Hemoconcentration and elevated hematocrit were linked to plasma leakage, while leukopenia and lymphopenia were common and associated with warning signs and severity. Additional markers, including Non-Structural Protein 1 (NS1)-mediated complement activation, altered T-cell subsets, elevated transaminases, and viral load, were related to severe disease. Overall, dynamic hematological changes across disease phases underscored their prognostic significance in dengue infection.
Conclusion: Current evidence suggests that dengue severity is influenced by complex immune and viral factors, with warning signs, hematological changes, Sequential Organ Failure Assessment (SOFA) score, transaminase elevation, and persistent fever serving as key predictors of complications—underscoring the importance of integrated clinical and laboratory monitoring for early risk stratification.
Keywords: Dengue, Hematologic Tests, Thrombocytopenia, Leukopenia, Hematocrit, Disease Severity, Prognosis.

INTRODUCTION

Dengue is a rapidly expanding mosquito-borne viral disease caused by the dengue virus and transmitted primarily by Aedes aegypti mosquitoes.

Dengue is an acute, self-limiting systemic viral illness transmitted by mosquitoes. Its rapidly increasing global spread poses a major public health and economic challenge, compounded by the limited availability of effective vaccines, specific antiviral therapies, and fully successful vector-control measures (Simmons CP et al., 2012).[1]

Dengue has expanded markedly in both incidence and geographic distribution, now affecting many regions that were previously untouched. It continues to be the leading arthropod-borne viral infection impacting humans worldwide (WHO, 2009).[2]

The rapid growth of international air travel and maritime trade has facilitated the long-distance movement of insect disease vectors, bypassing natural geographic barriers. Using a human–environment framework that integrates global transport data with climatic suitability, researchers have shown that the spread of Aedes albopictus—a laboratory-confirmed vector of multiple arboviruses—closely follows major shipping and air traffic routes. Its expansion has been largely predictable based on climate compatibility and transportation intensity, with higher traffic volumes linked to newly established regions. In contrast, Anopheles gambiae has shown limited spread beyond Africa, likely due to comparatively lower outbound sea traffic and historical air travel patterns (Tatem AJ et al., 2006).[3]

Dengue continues to expand in both geographic reach and disease burden, yet its true global distribution remains uncertain. Variations in diagnostic practices, surveillance quality, and healthcare infrastructure limit the reliability of individual data sources, leading to inconsistencies in national dengue reporting across international health agencies. By integrating multiple sources of evidence, researchers have developed a consensus-based map to better define the global range of dengue and identify countries where its status remains unclear (Brady OJ et al., 2012).[4]

Dengue is caused by four related serotypes transmitted by Aedes aegypti. While most infections result in mild dengue fever, severe forms such as dengue hemorrhagic fever and shock syndrome can occur, particularly during secondary infection with a different serotype. Increased vascular permeability and coagulation abnormalities characterize severe disease. Antibody-dependent enhancement, along with host immune and genetic factors, plays a key role in determining disease severity (Halstead SB, 1988).[5]

Dengue is a major global mosquito-borne illness with presentations ranging from mild fever to severe disease characterized by plasma leakage, bleeding, and shock. The lack of specific treatment is due to incomplete understanding of its pathogenesis. Severe manifestations are linked to immune activation and cytokine release, highlighting the need to better define these mechanisms to develop targeted therapies (Srikiatkhachorn A, et al., 2017).[6]

In acute Dengue, neutrophils become activated and form neutrophil extracellular traps (NETs), which can reduce viral infectivity. However, elevated NET components and pro-inflammatory cytokines are associated with severe disease, suggesting that neutrophils have a dual role in both antiviral defense and disease pathogenesis (Opasawatchai A et al., 2019).[7]

In a prospective study of schoolchildren in Northern Thailand, marked spatial and temporal variability was observed in the symptomatic-to-inapparent (S:I) dengue illness ratio across schools and transmission seasons. The ratio was primarily influenced by the current year's dengue incidence and infection rates from the preceding year, while no association with age was identified. These findings suggest that population-level virus–host interactions, potentially mediated by heterotypic cross-reactive immunity, may modulate disease severity, carrying important implications for dengue vaccine development (Endy TP et al., 2011).[8]

Although numerous individual studies have evaluated hematological alterations in dengue, a comprehensive synthesis focusing on their temporal trends and clinical implications is lacking. Therefore, this systematic review aims to summarize the dynamic changes in hematological parameters during dengue infection and to evaluate their diagnostic and prognostic significance in improving patient management.

Objectives

This study aims to systematically review and integrate existing evidence on the dynamic hematological changes across various phases of dengue infection, examine the relationship between key parameters—such as platelet count, hematocrit, and leukocyte count—and disease severity, and identify hematological markers that may predict complications and clinical outcomes.

MATERIALS AND METHODS

This systematic review was conducted in accordance with PRISMA guidelines. A comprehensive literature search was performed in PubMed, Scopus, Web of Science, and Google Scholar using the following keywords: Dengue, Hematologic Tests, Thrombocytopenia, Leukopenia, Hematocrit, Disease Severity, and Prognosis. Eligible study designs included randomized controlled trials, cohort studies, case–control studies, cross-sectional studies, meta-analysis and relevant review articles. Non-English publications, studies without extractable data or clearly defined outcome measures, as well as editorials, commentaries, and conference abstracts, were excluded.

Titles, abstracts, and full texts were independently screened by two reviewers, with disagreements resolved by consensus. Data extraction and quality assessment were performed independently using standardized tools appropriate to study design. Due to heterogeneity among studies, a qualitative synthesis was conducted.

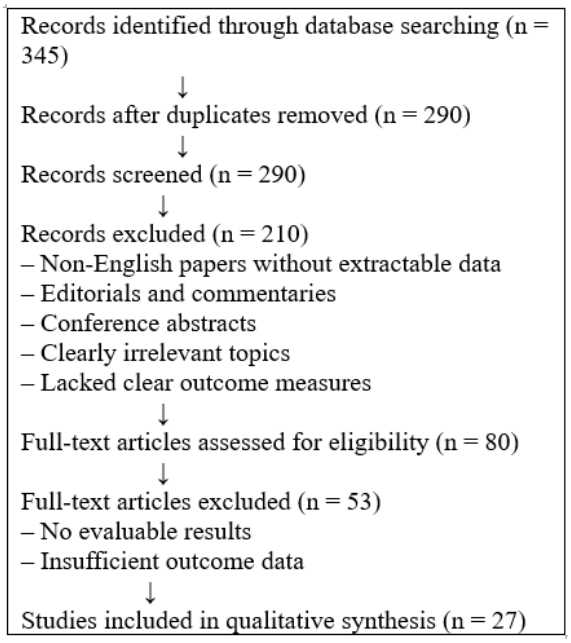

Figure : PRISMA Flow chart of Study Selection Process

RESULTS

Platelet Count

John KJ et al., 2019, reported that among 159 admitted dengue patients, 60.4% were male, with a mean age of 31.3 ± 13.5 years; 10.7% had diabetes and 2.5% were on immunosuppressants. Common symptoms included vomiting (70.4%), myalgia (60.4%), headache (42.1%), abdominal pain (38.4%), bleeding (38%), and rash (37.1%). Median admission values were hemoglobin 144 g/L, platelets 31 × 10^3/µL, WBC 4.9 × 10^9/L, creatinine 64.05 µmol/L, bilirubin 0.6 mg/dL, SGOT 149 U/L, and SGPT 92 U/L; the mean SOFA score was 3.27. Hepatitis (65.8%), encephalitis (6.2%), and renal dysfunction (5.6%) were present at presentation.[9]

Logia P et al., 2023, reported that during the study period, 9,817 patients were hospitalized with dengue. Among 120 ICU patients with thrombocytopenia (<100,000 cells/mm^3), 31.6% developed clinically significant bleeding (CSB). On multivariate analysis, independent predictors of CSB were higher Sequential Organ Failure Assessment (SOFA) score (Adjusted Odds Ratio [aOR] 1.52), fever >38.3°C (aOR 2.71), and prolonged aPTT >40 seconds (aOR 4.66). A predictive score based on these factors showed good accuracy (Area Under the Curve [AUC] 0.81), with 81% sensitivity and 77% specificity.[10]

The findings by Ojha A et al., 2017, indicate that platelet activation plays a key role in the development of thrombocytopenia in dengue. Targeted modulation of platelet activation may help prevent their accelerated clearance during Dengue Virus (DENV) infection.[11]

A cross-sectional study of 130 confirmed dengue cases in Pakistan identified 23 severe and 107 nonsevere infections. Severe cases commonly presented with mucosal bleeding, fluid accumulation, shock, and gastrointestinal bleeding. Key hematological abnormalities associated with severity were thrombocytopenia, leukopenia, and elevated hematocrit ($P < 0.001$), with severe cases showing marked thrombocytopenia (mean platelet count ~50 × 10^9/L). Clinical features combined with hematological parameters are essential for assessing dengue severity and guiding management (Haq FU et al.,2023).[12]

Table 1: Summary of evidence on hematological abnormalities and severity indicators in Dengue

Author (Year)	Key Findings
John KJ et al., 2019	159 patients; majority male. Common symptoms: vomiting, myalgia, headache. Marked thrombocytopenia (31 × 10^3/µL); mean SOFA 3.27. Hepatitis, encephalitis, and renal dysfunction observed.
Logia P et al., 2023	31.6% ICU thrombocytopenic patients developed significant bleeding. Predictors: higher SOFA, fever >38.3°C, prolonged aPTT. Predictive score AUC 0.81.
Ojha A et al., 2017	Platelet activation contributes to dengue thrombocytopenia; modulation may reduce platelet clearance.
Haq FU et al., 2023	Severe dengue linked to thrombocytopenia, leukopenia, elevated hematocrit; severe cases had ~50 × 10^9/L platelets.

Hematocrit and Hemoconcentration

Plasma leakage in dengue results from interactions among the virus, host immune responses, and endothelial cells that disrupt vascular integrity. Recent research highlights the influence of angiogenic factors and virus-induced endothelial changes. Understanding these mechanisms is essential for developing effective preventive and therapeutic strategies (Srikiatkhachorn A et al., 2009).[13]

In a study by Chaloemwong J et al., 2018, among 154 dengue patients and 146 controls, dengue cases more commonly had headache, nausea, anorexia, and bleeding. They showed higher hemoglobin and hematocrit (days 3–10), lower WBC (days 1–10) and platelets (days 3–10), with early monocytosis, later atypical lymphocytosis, and late eosinophilia. The neutrophil-to-lymphocyte ratio was initially >1 but reversed after day 5, unlike controls where it remained >1.[14]

Among 515 patients, 333 had dengue hemorrhagic fever (DHF) and 182 had dengue fever (DF). During the acute phase, patients who progressed to DHF showed significantly lower platelet counts and

different hemoglobin levels compared to non-leakers, while WBC, neutrophil, lymphocyte, and hematocrit values were similar. By day 5, leakers had higher WBC and hemoglobin but lower platelet counts. Receiver Operating Characteristic (ROC) analysis demonstrated that platelet count, particularly during the acute phase, had good predictive value for identifying DHF in both males and females (Ralapanawa U et al., 2018).[15]

Among 150 suspected cases tested within days 1–5 of illness, 78 were NS1-positive; 37 were in the febrile phase and 41 in the critical phase. Patients in the critical phase showed leukopenia and thrombocytopenia. Nine developed plasma leakage, accompanied by elevated hemoglobin, hematocrit, and liver transaminases, indicating more severe disease (Jayadas TTP et al., 2019).[16]

Table 2: summary of laboratory findings associated with severity and plasma leakage in Dengue

Author (Year)	Key Findings
Srikiatkhachorn A et al., 2009	Plasma leakage results from virus–immune–endothelial interactions disrupting vascular integrity; angiogenic factors and endothelial changes contribute to severity.
Chaloemwong J et al., 2018	Higher hemoglobin and hematocrit, lower WBC and platelets; early monocytosis, later atypical lymphocytosis; neutrophil-to-lymphocyte ratio reversed after day 5 in dengue.
Ralapanawa U et al., 2018	DHF cases had lower platelet counts in acute phase; platelet count showed good predictive value for identifying DHF.
Jayadas TTP et al., 2019	Critical phase showed leukopenia and thrombocytopenia; plasma leakage associated with elevated hemoglobin, hematocrit, and transaminases.

Leukocyte and Differential Counts

Oliveira EC et al., 2009, studied that in 543 DENV-3 cases from a 2007 outbreak, most had mild classic dengue. Common findings were leukopenia, thrombocytopenia, lymphocytopenia, and atypical lymphocytosis. Dengue hemorrhagic fever showed more prolonged thrombocytopenia and higher atypical lymphocyte counts, with hematological changes correlating with disease severity.[17]

A retrospective study conducted at Aga Khan University Hospital, Karachi (2001–2006) included 210 dengue IgM–positive patients (male: female ratio 1.6:1; mean age 29.7 years). On admission, 9% had elevated hemoglobin/hematocrit, persisting in 2.1% at discharge. Leukopenia with neutropenia was observed in 26.6%, and thrombocytopenia in 77.1%. Prolonged PT and APTT were noted in 2.5% and 16.7% of patients, respectively, while atypical lymphocytes were present in 52%. Platelet transfusion was required in 22.1% of cases. Most patients (93.3%) recovered fully, whereas 3.3% died due to dengue shock syndrome. The findings highlight the importance of hematological parameters in the evaluation and management of suspected dengue cases (Ali N et al., 2007).[18]

In a multi-centre study by Gupta BP et al., 2025, found that leukopenia (21.5%) was significantly associated with dengue with warning signs, while thrombocytopenia (62.1%) was more common in milder cases and showed only a weak link to severity. Leukopenia correlated with joint pain, nausea, and rash, and thrombocytopenia with retro-orbital and abdominal pain. No association was found with age or sex.[19]

Among patients, 65.9% had dengue without warning signs, 26.9% had warning signs, and 7.1% had severe dengue. Leukopenia (<4000 cells/mm³) occurred in 64.7% (lowest 1,440), and thrombocytopenia (<150,000 cells/mm³) in 40.5% (lowest 26,000). Both were significantly associated with increasing disease severity ($p \leq 0.001$) (Thapa B et al., 2025).[20]

Table 3: Summary of hematological patterns and severity correlation in Dengue infection

Author (Year)	Key Findings
Oliveira EC et al., 2009	Most cases had mild classic dengue; common findings were leukopenia, thrombocytopenia, lymphocytopenia, and atypical lymphocytosis. DHF showed more prolonged thrombocytopenia and higher atypical lymphocyte counts, correlating with severity.
Ali N et al., 2007	Thrombocytopenia (77.1%) most common, followed by leukopenia with neutropenia (26.6%) and atypical lymphocytosis (52%); elevated hemoglobin/hematocrit in 9%. Most recovered; mortality 3.3% due to dengue shock syndrome.
Gupta BP et al., 2025	Leukopenia (21.5%) significantly associated with warning signs; thrombocytopenia (62.1%) more common in milder cases with weak link to severity. No association with age or sex.
Thapa B et al., 2025	Leukopenia (64.7%) and thrombocytopenia (40.5%) were significantly associated with increasing disease severity ($p \leq 0.001$).

Other Hematological Parameters

Soluble Non-Structural Protein 1 (NS1) triggered full complement activation, which was further amplified by NS1-specific antibodies; cell-associated NS1 also activated complement in their presence. Higher plasma levels of NS1 and terminal complement complex, Soluble C5b-9 (SC5b-9) were associated with greater disease severity. Elevated NS1, C5a, and SC5b-9 were detected in pleural fluid of patients with dengue shock syndrome (Avirutnan P et al., 2006).[21]

Decreased levels of CD3, CD4, CD8, and CD5 cells were significantly associated with the severity of thrombocytopenia in dengue hemorrhagic fever ($p < 0.05$), indicating their possible involvement in a shared pathogenic pathway (Fadilah SA et al., 1999).[22]

In the prospective cohort study by Sigera PC et al., 2019, only 70% of clinically suspected cases were confirmed as dengue, indicating overdiagnosis based on symptoms alone. Reverse Transcription Loop-Mediated Isothermal Amplification (RT-LAMP) showed diagnostic accuracy comparable to or better than Reverse Transcription Quantitative Polymerase Chain Reaction (RT-qPCR) and NS1 testing. Early dengue was characterized by lower lymphocyte counts and higher Aspartate Aminotransferase (AST) levels, and younger age with elevated AST predicted later plasma leakage.[23]

A 2024 prospective cohort study of 135 confirmed dengue patients (with 2014–2023 retrospective data) demonstrated a biphasic platelet decline, reaching a nadir on day 6 and recovering by day 9. Thrombocytopenia (50% intermediate-low; 14.8% very low) was significantly associated with lymphocytosis and neutropenia ($p < 0.001$). Platelet counts correlated with viral load, hematocrit, and platelet indices including Mean Platelet Volume (MPV), Platelet Large Cell Ratio (P-LCR), and Plateletcrit (PCT). No patients developed severe dengue despite marked thrombocytopenia (Guo L et al., 2025).[24]

The retrospective descriptive study by Azin FR et al., 2012 included 154 serologically confirmed dengue patients admitted to a tertiary referral hospital in Fortaleza (January–May 2008), categorized as <15 years (n = 66) and ≥15 years (n = 88). Laboratory evaluation showed thrombocytopenia and elevated Aspartate Aminotransferase (AST) and Alanine Aminotransferase (ALT) in classic dengue, while dengue hemorrhagic fever and severe forms additionally demonstrated hemoconcentration. Abnormalities typically emerged by day 3, peaked around day 5, and normalized by day 11, with more pronounced changes in children and severe cases.[25]

A retrospective cross-sectional study using Kairuki Hospital laboratory data evaluated 255 suspected dengue patients tested for dengue IgM and complete blood count. Among them, 73.7% were dengue-positive. Dengue patients had significantly lower total white blood cell and lymphocyte counts compared to non-dengue cases ($p \leq 0.007$), while other hematological parameters showed no significant differences. Lymphopenia independently predicted dengue positivity (adjusted Odds Ratio [OR] 5.26; 95% Confidence Interval [CI]: 2.28–12.2; $p < 0.001$) (Kalabamu FS et al., 2021).[26]

CONCLUSION

Logia P et al., 2023, concluded that fever >38.3°C, prolonged aPTT, and higher SOFA scores independently predicted clinically significant bleeding, and a score based on these factors can help identify high-risk patients.[10]

Dengue mainly affected young males, with vomiting and myalgia as common symptoms; 44.7% had bleeding. Most cases occurred in October, predominantly from Vellore. The mean SOFA score was 3.27. Secondary infection and hepatitis were common, and most patients had warning signs or severe dengue. Elevated transaminases, severe and secondary dengue were associated with major bleeding, while older age, abnormal laboratory parameters, and higher SOFA scores predicted mortality. Overall mortality was 2.5% (John KJ et al., 2019).[9]

Severe dengue results from dysregulated immune activation involving innate and adaptive responses. The lack of targeted therapies and clear severity predictors reflects incomplete understanding of key mediators. Stage-specific approaches—early antiviral therapy and later immunomodulation—may be beneficial, but progress requires robust clinical studies with advanced integrated analyses (Srikiatkhachorn A et al., 2017).[6]

Leukopenia may serve as an early marker of dengue severity, whereas thrombocytopenia showed limited predictive value. Further longitudinal studies are needed to validate these findings and strengthen risk models (Gupta BP et al., 2025).[19]

Close monitoring of warning signs and clinical parameters is essential to detect progression to the critical phase of dengue, which typically emerges around defervescence. Leukopenia and thrombocytopenia are common findings and may signal worsening disease at the time of emergency presentation (Thapa B et al., 2025).[20]

The study by Guo L et al., 2025, highlights viral load–associated thrombocytopenia in dengue, with predominantly moderate platelet reduction challenging conventional risk assessment. Integrated monitoring of platelet indices and viral replication may improve risk prediction and understanding of platelet–virus interactions.[24]

Dengue patients demonstrated significantly lower total white blood cell and lymphocyte counts than non-dengue febrile cases. Lymphopenia emerged as a key hematological predictor of dengue, and when combined with clinical features, may help guide decisions for confirmatory testing (Kalabamu FS et al., 2021).[26]

In a study of 100 patients with dengue hemorrhagic fever/dengue shock syndrome, 7 had concurrent bacteremia while 93 had dengue alone. Dual infection was associated with older age, prolonged fever, acute renal failure, gastrointestinal bleeding, altered consciousness, and shock. Acute renal failure and fever lasting more than five days were identified as independent predictors of bacteremia. These findings emphasize the need for heightened clinical suspicion and prompt management of possible concurrent bacterial infection in high-risk dengue patients (Lee IK et al., 2005).[27]

Overall, current evidence indicates that dengue severity and complications are driven by complex immune and viral dynamics, with clinical warning signs, leukopenia, thrombocytopenia patterns, platelet indices, SOFA score, transaminase elevation, and persistent fever serving as important predictors

of bleeding, mortality, and co-infection—highlighting the need for integrated clinical and laboratory monitoring to enable early risk stratification and timely management.

Strengthening translational research that bridges laboratory findings with clinical outcomes will be crucial to improving prevention, early detection, and mortality reduction in dengue.

Acknowledgement

The authors sincerely acknowledge all researchers and investigators whose published work formed the foundation of this systematic review. Special thanks to our mentors and colleagues for their valuable guidance, critical feedback, and continuous support throughout the review process. Their contributions greatly enhanced the quality and rigor of this work. The authors declare that there are no conflicts of interest and that no funding was received for this study.

REFERENCES

1. Simmons CP, Farrar JJ, Nguyen vV, Wills B. Dengue. N Engl J Med. 2012 Apr 12;366(15):1423-32. doi: 10.1056/NEJMra1110265. PMID: 22494122.
2. Dengue: Guidelines for Diagnosis, Treatment, Prevention and Control: New Edition. Geneva: World Health Organization; 2009. PMID: 23762963.
3. Tatem AJ, Hay SI, Rogers DJ. Global traffic and disease vector dispersal. Proc Natl Acad Sci U S A. 2006 Apr 18;103(16):6242-7. doi: 10.1073/pnas.0508391103. Epub 2006 Apr 10. PMID: 16606847; PMCID: PMC1435368.
4. Brady OJ, Gething PW, Bhatt S, Messina JP, Brownstein JS, Hoen AG, Moyes CL, Farlow AW, Scott TW, Hay SI. Refining the global spatial limits of dengue virus transmission by evidence-based consensus. PLoS Negl Trop Dis. 2012;6(8):e1760. doi: 10.1371/journal.pntd.0001760. Epub 2012 Aug 7. PMID: 22880140; PMCID: PMC3413714.
5. Halstead SB. Pathogenesis of dengue: challenges to molecular biology. Science. 1988 Jan 29;239(4839):476-81. doi: 10.1126/science.3277268. PMID: 3277268.
6. Srikiatkhachorn A, Mathew A, Rothman AL. Immune-mediated cytokine storm and its role in severe dengue. Semin Immunopathol. 2017 Jul;39(5):563-574. doi: 10.1007/s00281-017-0625-1. Epub 2017 Apr 11. PMID: 28401256; PMCID: PMC5496927.
7. Opasawatchai A, Amornsupawat P, Jiravejchakul N, Chan-In W, Spoerk NJ, Manopwisedjaroen K, Singhasivanon P, Yingtaweesak T, Suraamornkul S, Mongkolsapaya J, Sakuntabhai A, Matangkasombut P, Loison F. Neutrophil Activation and Early Features of NET Formation Are Associated With Dengue Virus Infection in Human. Front Immunol. 2019 Jan 11;9:3007. doi: 10.3389/fimmu.2018.03007. PMID: 30687301; PMCID: PMC6336714.
8. Endy TP, Anderson KB, Nisalak A, Yoon IK, Green S, Rothman AL, Thomas SJ, Jarman RG, Libraty DH, Gibbons RV. Determinants of inapparent and symptomatic dengue infection in a prospective study of primary school children in Kamphaeng Phet, Thailand. PLoS Negl Trop Dis. 2011 Mar 1;5(3):e975. doi: 10.1371/journal.pntd.0000975. PMID: 21390158; PMCID: PMC3046956.
9. John KJ, Gunasekaran K, Prasad JD, Mathew D, Das S, Sultan N, Abraham AM, Iyyadurai R. Predictors of Major Bleeding and Mortality in Dengue Infection: A Retrospective Observational Study in a Tertiary Care Centre in South India. Interdiscip Perspect Infect Dis. 2019 Sep 4;2019:4823791. doi: 10.1155/2019/4823791. PMID: 31565054; PMCID: PMC6746148.
10. Logia P, Selvam V, Parasuraman V, Renuka MK, Rajagopalan RE. Predictors of Clinically Significant Bleeding in Thrombocytopenic Dengue Patients Admitted to Intensive Care Unit: A Retrospective Study. Indian J Crit Care Med. 2023 Dec;27(12):888-894. doi: 10.5005/jp-journals-10071-24574. PMID: 38074960; PMCID: PMC10701565.
11. Ojha A, Nandi D, Batra H, Singhal R, Annarapu GK, Bhattacharyya S, Seth T, Dar L, Medigeshi GR, Vrati S, Vikram NK, Guchhait P. Platelet activation determines the severity of thrombocytopenia in dengue infection. Sci Rep. 2017 Jan 31;7:41697. doi: 10.1038/srep41697. PMID: 28139770; PMCID: PMC5282509.
12. Haq FU, Imran M, Aslam Z, Mukhtar F, Jabeen K, Chaudhry M, Rahman SU, Muhammad N. Severity of Dengue Viral Infection Based on Clinical and Hematological Parameters among Pakistani Patients. Am J Trop Med Hyg. 2023 Oct 23;109(6):1284-1289. doi: 10.4269/ajtmh.23-0309. PMID: 37871589; PMCID: PMC10793050.
13. Srikiatkhachorn A. Plasma leakage in dengue haemorrhagic fever. Thromb Haemost. 2009 Dec;102(6):1042-9. doi: 10.1160/TH09-03-0208. PMID: 19967133; PMCID: PMC5527705.
14. Chaloemwong J, Tantiworawit A, Rattanathammethee T, Hantrakool S, Chai-Adisaksopha C, Rattarittamrong E, Norasetthada L. Useful clinical features and hematological parameters for the diagnosis of dengue infection in patients with acute febrile illness: a retrospective study. BMC Hematol. 2018 Aug 29;18:20. doi: 10.1186/s12878-018-0116-1. PMID: 30181881; PMCID: PMC6114047.
15. Ralapanawa U, Alawattegama ATM, Gunrathne M, Tennakoon S, Kularatne SAM, Jayalath T. Value of peripheral blood count for dengue severity prediction. BMC Res Notes. 2018 Jun 20;11(1):400. doi: 10.1186/s13104-018-3505-4. PMID: 29925425; PMCID: PMC6011352.
16. Jayadas TTP, Kumanan T, Arasaratnam V, Gajapathy K, Surendran SN. The clinical profile, hematological parameters and liver transaminases of dengue NS1 Ag positive patients admitted to Jaffna Teaching Hospital, Sri Lanka. BMC Res Notes. 2019 Sep 23;12(1):604. doi: 10.1186/s13104-019-4655-8. PMID: 31547852; PMCID: PMC6755686.
17. Oliveira EC, Pontes ER, Cunha RV, Fróes IB, Nascimento Dd. Alterações hematológicas em pacientes com dengue [Hematological abnormalities in patients with dengue]. Rev Soc Bras Med Trop. 2009 Nov-Dec;42(6):682-5. Portuguese. doi: 10.1590/s0037-86822009000600014. PMID: 20209355.
18. Ali N, Usman M, Syed N, Khurshid M. Haemorrhagic manifestations and utility of haematological parameters in dengue fever: a tertiary care centre experience at Karachi. Scand J Infect Dis. 2007;39(11-12):1025-8. doi: 10.1080/00365540701411492. Epub 2007 May 30. PMID: 17852892.
19. Gupta BP, Uranw S, Gupta VP, Deuba E, Sah AK, Chaudhary S, Wagle C. Leukopenia and thrombocytopenia in dengue patients: a cross-sectional study from a tertiary hospitals in Koshi Province, Nepal. BMC Infect Dis. 2025 May 26;25(1):753. doi: 10.1186/s12879-025-11126-8. PMID: 40419985; PMCID: PMC12105263.
20. Thapa B, Lamichhane P, Shrestha T, Lamichhane S, Karki S, Pradhananga S, Batajoo KH, Pudasaini P. Leukopenia and thrombocytopenia in dengue patients presenting in the emergency department of a tertiary center in Nepal: a cross-sectional study. BMC Infect Dis. 2025 Jan 11;25(1):56. doi: 10.1186/s12879-025-10486-5. PMID: 39815245; PMCID: PMC11734494.
21. Avirutnan P, Punyadee N, Noisakran S, Komoltri C, Thiemmeca S, Auethavornanan K, Jairungsri A, Kanlaya R, Tangthawornchaikul N, Puttikhunt C, Pattanakitsakul SN, Yenchitsomanus PT, Mongkolsapaya J, Kasinrerk W, Sittisombut N, Husmann M, Blettner M, Vasanawathana S, Bhakdi S, Malasit P. Vascular leakage in severe dengue virus infections: a potential role for the nonstructural viral protein NS1 and complement. J Infect Dis. 2006 Apr 15;193(8):1078-88. doi: 10.1086/500949. Epub 2006 Mar 9. PMID: 16544248.
22. Fadilah SA, Sahrir S, Raymond AA, Cheong SK, Aziz JA, Sivagengei K. Quantitation of T lymphocyte subsets helps to distinguish dengue hemorrhagic fever from classic dengue fever during the acute febrile stage. Southeast Asian J Trop Med Public Health. 1999 Dec;30(4):710-7. PMID: 10928365.

23. Sigera PC, Amarasekara R, Rodrigo C, Rajapakse S, Weeratunga P, De Silva NL, Huang CH, Sahoo MK, Pinsky BA, Pillai DR, Tissera HA, Jayasinghe S, Handunnetti S, Fernando SD. Risk prediction for severe disease and better diagnostic accuracy in early dengue infection; the Colombo dengue study. BMC Infect Dis. 2019 Aug 1;19(1):680. doi: 10.1186/s12879-019-4304-9. PMID: 31370795; PMCID: PMC6676631.
24. Guo L, Gu Y, Zhang Y, Zhang H, Weng W, Wu S, Yuan J. Platelet dynamics and thrombocytopenia in dengue fever: A prospective cohort study from Shenzhen, China. New Microbes New Infect. 2025 Aug 23;67:101624. doi: 10.1016/j.nmni.2025.101624. PMID: 40919228; PMCID: PMC12409373.
25. Azin FR, Gonçalves RP, Pitombeira MH, Lima DM, Branco IC. Dengue: profile of hematological and biochemical dynamics. Rev Bras Hematol Hemoter. 2012;34(1):36-41. doi: 10.5581/1516-8484.20120012. PMID: 23049382; PMCID: PMC3459605.
26. Kalabamu FS, Maliki S. Use of Haematological Changes as a Predictor of Dengue Infection among Suspected Cases at Kairuki Hospital in Dar Es Salaam, Tanzania: A Retrospective Cross Sectional Study. East Afr Health Res J. 2021;5(1):91-98. doi: 10.24248/eahrj.v5i1.655. Epub 2021 Jun 11. PMID: 39432750; PMCID: PMC8291204.
27. Lee IK, Liu JW, Yang KD. Clinical characteristics and risk factors for concurrent bacteremia in adults with dengue hemorrhagic fever. Am J Trop Med Hyg. 2005 Feb;72(2):221-6. PMID: 15741560.