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Original Research Article

Study of Correlation of Platelet Indices for Prognostication In Viral Hemorrhagic Fever In Pediatric Age Group

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Abstract:

Background: Viral hemorrhagic fevers (VHFs), caused by Arenaviruses, Filoviruses, Bunyaviruses, and Flaviviruses, are globally distributed. In India, Dengue fever, Dengue hemorrhagic fever (DHF), and Kyasanur Forest Disease (KFD) are endemic, with Dengue being hyperendemic in many regions. VHFs are highly infectious and potentially fatal, presenting with symptoms such as fever, vomiting, bleeding, edema, and hypotension. Diagnosis of Dengue fever primarily relies on NS1 antigen detection, while thrombocytopenia is a common finding due to bone marrow suppression, platelet destruction, and viral replication. Platelet indices, including mean platelet volume (MPV) and platelet distribution width (PDW), serve as important markers for assessing platelet activation and bleeding risk.

Methods: A prospective observational study at Ballari Medical College and Research Center (June 2022–December 2024) includes 200 febrile thrombocytopenia patients in the early defervescence stage. EDTA blood samples are analyzed for hematological parameters, including hemoglobin, leukocyte count, red cell indices, and platelet count. Platelet indices (MPV, PDW, PCT) are assessed within two hours of venipuncture. Serological tests (dengue NS1, IgM, IgG) and additional renal and liver function tests are also conducted.

Results: In this prospective study of 200 pediatric patients with viral hemorrhagic fever, the most affected age group was 6–10 years, with a male predominance (59.5%). Fever was universal, and common symptoms included abdominal pain (90%), right hypochondrial tenderness (81.5%), vomiting (68.5%), and nausea (64%). Most children were hemodynamically stable at presentation, though 68% had hepatomegaly. Platelet indices showed significant trends over 11 days: platelet count and plateletcrit progressively increased, while mean platelet volume (MPV) and platelet distribution width (PDW) decreased ($p<0.001$), indicating hematological recovery. Liver dysfunction was common (57%), but renal involvement was rare (1%). NS1 antigen was positive in 91%, confirming dengue as the major etiology. Confirmed diagnoses included classic dengue fever (74%), dengue hemorrhagic fever (9%), Dengue Shock syndrome(7%) and rare cases of dengue encephalitis (1%).

Conclusion: This study demonstrates that serial monitoring of platelet indices—rising platelet count, Plateletcrit and declining MPV & PDW and plateletcrit—effectively correlates with disease severity and recovery in pediatric viral hemorrhagic fever. The findings justify the study's objective, highlighting these indices as reliable, non-invasive prognostic tools that aid in clinical assessment, timely intervention, and improved outcomes in affected children.

Keywords: Viral hemorrhagic fevers (VHFs); Dengue hemorrhagic fever (DHF); Kyasanur Forest Disease (KFD); Mean platelet volume (MPV); Platelet distribution width (PDW); Hepatomegaly.

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Introduction

Viral hemorrhagic fevers (VHFs) are caused by viruses from four families: Arenaviruses, Filoviruses, Bunyaviruses, and Flaviviruses. Humans are not the natural reservoirs for these viruses and typically become infected through contact with infected hosts. However, human-to-human transmission has been documented for viruses such as Ebola, Marburg, and Crimean-Congo hemorrhagic fever, primarily through direct contact with infected blood and bodily fluids. VHF-causing agents are distributed worldwide. In India, Dengue fever, Dengue hemorrhagic fever (DHF), and Kyasanur Forest Disease (KFD) are endemic, with Dengue hyperendemic in many regions[1]. The incubation period for VHFs ranges from 2 to 21 days[2].

VHFs are highly infectious and potentially fatal, characterized by symptoms such as fever, malaise,

vomiting, mucosal and gastrointestinal bleeding, edema, and hypotension. An essential pathological defect in VHF is increased vascular permeability. The viruses primarily target the vascular system, causing early symptoms like flushing, conjunctival injection, petechial hemorrhages, fever, and myalgia. Advanced stages often lead to mucous membrane hemorrhages, hypotension, shock, and circulatory collapse[2].

Dengue fever is caused by one of four serotypes of the dengue virus (DEN-1 to DEN4) belonging to the Flaviviridae family. Both Aedes aegypti and Aedes albopictus mosquitoes are competent vectors, with the close association of A. aegypti with humans driving transmission in tropical urban areas[1]. Dengue was first reported in Bangladesh in 1964 as "Dacca fever." Its incidence increases during the monsoon and post-monsoon seasons[3]. Globally, dengue affects 390 million individuals annually, with 96 million manifesting clinical symptoms[4]. India reported an average of 1.93 lakh cases in 2021[5].

Dengue presents a wide disease spectrum, from dengue fever characterized by biphasic fever, myalgia, arthralgia, leukopenia, and lymphadenopathy to severe syndromes like DHF and dengue shock syndrome (DSS), which can be fatal[1]. DHF is marked by high fever, hemorrhagic phenomena, hepatomegaly, circulatory disturbances, and shock. Initial symptoms include a sudden rise in temperature, facial flushing, anorexia, vomiting, severe frontal headache, retro-orbital pain, nausea, and skin eruptions. Severe muscle and bone pain ("break-bone fever") is typical. Biphasic fever patterns may occur, with temperatures reaching up to 40°C[1].

The detection of the non-structural protein 1 (NS1) antigen is a sensitive and specific method for diagnosing dengue[11]. Thrombocytopenia is a common laboratory finding in dengue, resulting from mechanisms such as direct bone marrow suppression by the virus, antibody-mediated platelet destruction, peripheral consumption of platelets, or isolated viral replication within platelets[3].

Novel platelet indices, including mean platelet volume (MPV) and platelet distribution width (PDW), are potential markers of platelet activation. MPV reflects platelet volume and is associated with marrow suppression and increased bleeding risk. Changes in platelet morphology due to activation are evaluated through MPV and PDW, while plateletcrit (PCT), a parameter combining MPV and platelet count, provides a reliable measurement of platelet biomass[3].

Methodology

Setting of the Study: This prospective observational study is being conducted at Ballari Medical College and Research Center, a tertiary care referral center, from June 2022 to December 31, 2024. The study includes 200 patients diagnosed with febrile thrombocytopenia who present to the hospital during the early defervescence stage. Informed consent is obtained from all participants, and the study protocol has received approval from the institutional ethics committee.

Study Design: An Observational Prospective Study

Method of Collection of Data:

Inclusion Criteria: Patient with age group between 1 month – 14 yrs admitted with clinical diagnosis of viral hemorrhagic fever with varying severity and thrombocytopenia (Platelet count - < 1.5lakh/cumm).

Exclusion Criteria: Patients with viral hemorrhagic fever with co-morbidities like congenital heart disease, cerebral palsy, co – infection.

Patient with viral hemorrhagic fever and received platelet transfusion.

Results

Table 1: Distribution of the study subjects based on age (N=200)

Age group (N=200)	Frequency (N)	Percentage (%)
1 month to 1 year	30	15.0%
1 to 5 years	56	28.0%
6 to 10 years	69	34.5%
10 to 14 years	45	22.5%
Total	200	100%

Among the 200 pediatric patients included in the study, the majority belonged to the 6 to 10 years age group, comprising 34.5% of the total sample. This was closely followed by the 1 to 5 years group, which made up 28% of the subjects. Children aged between 10 to 14 years constituted 22.5%, while infants in the 1 month to 1 year group accounted for the lowest proportion at 15%. The dominance of children in the 6 to 10 years category suggests that this age group might be more susceptible to viral hemorrhagic fevers, potentially due to increased exposure to outdoor environments, vector-prone zones, or lower immunity compared to older children. The relatively lower representation among infants might reflect both lower exposure risk and possibly under-detection. This age-based pattern emphasizes that mid-childhood (6–10 years) is a critical window for both infection risk and intervention strategies in viral hemorrhagic fevers

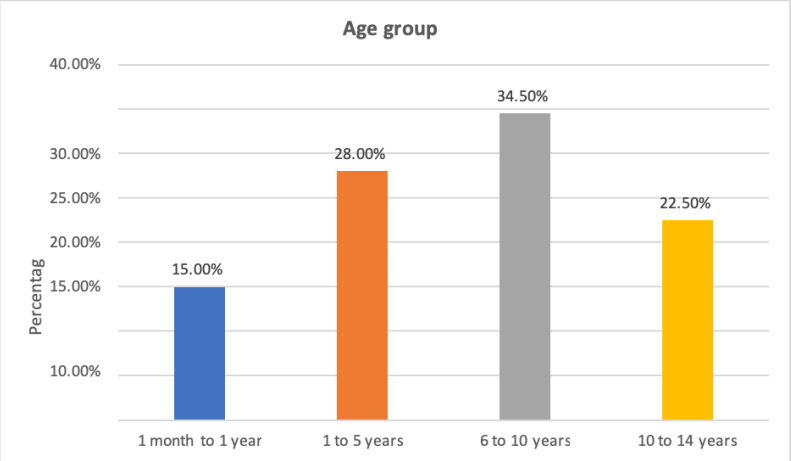

Figure 1: Distribution of the study subjects based on age (N=200)

Table 2: Distribution of the study subjects based on gender (N=200)

Gender (N=200)	Frequency (N)	Percentage (%)
Male	119	59.5%
Female	81	40.5%

The gender-wise distribution reveals a higher prevalence of male patients, with 119 out of 200 (59.5%) being male and the remaining 81 (40.5%) being female. This male preponderance may reflect behavioral, cultural, or possibly biological differences in susceptibility or care-seeking practices in the pediatric population. Males in certain sociocultural contexts are more likely to be brought to healthcare facilities, which could skew the observed figures. Additionally, increased outdoor exposure in male children may also increase contact with vectors such as mosquitoes, enhancing the likelihood of contracting viral hemorrhagic fevers. This implies that males formed the majority of the cases, suggesting a gendered pattern in either exposure or health system utilization that warrants further exploration

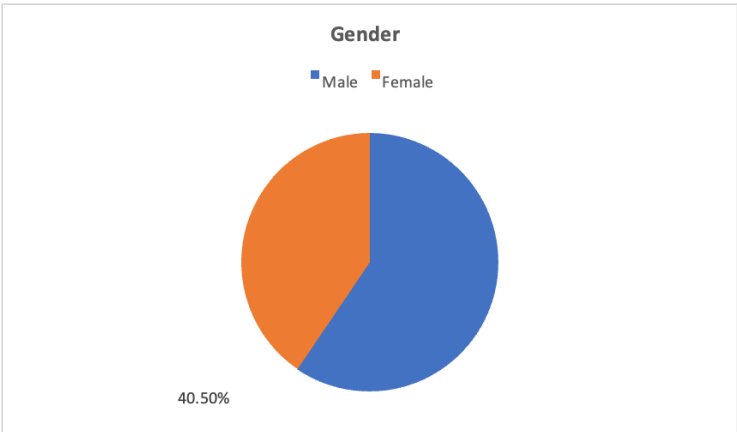

Figure 2: Distribution of the study subjects based on gender (N=200)

Table 3: Frequency of presenting complaints among the study subjects (n=200)

Clinical features (N=200)	Frequency (N)	Percentage (%)
Fever	200	100.0%
Cold	135	67.5%
Headache	89	44.5%
Joint Pain	78	39.0%
Myalgia	93	46.5%
Pain Abdomen	180	90.0%
Nausea	128	64.0%
Vomiting	137	68.5%
Right Hypochondrial Tenderness	163	81.5%
Decreased Urine Output	108	54.0%

Fever was the universal symptom among all subjects (100%), affirming it as the hallmark of viral hemorrhagic fevers. A significant majority also experienced pain abdomen (90%), right hypochondrial tenderness (81.5%), vomiting (68.5%), cold (67.5%), and nausea (64%). Decreased urine output was seen in 54% of the patients, suggesting renal involvement in a considerable portion. Myalgia and headache were observed in 46.5% and 44.5% respectively, while joint pain was noted in 39%. The clustering of gastrointestinal and constitutional symptoms in a large segment of the cohort indicates multisystem involvement. Abdominal symptoms like pain and vomiting appear to be dominant alongside fever, which may help in early clinical suspicion. The inference here is that the majority of patients presented with a characteristic constellation of fever with gastrointestinal and systemic symptoms, underscoring the typical multisystem presentation in pediatric viral hemorrhagic fever cases

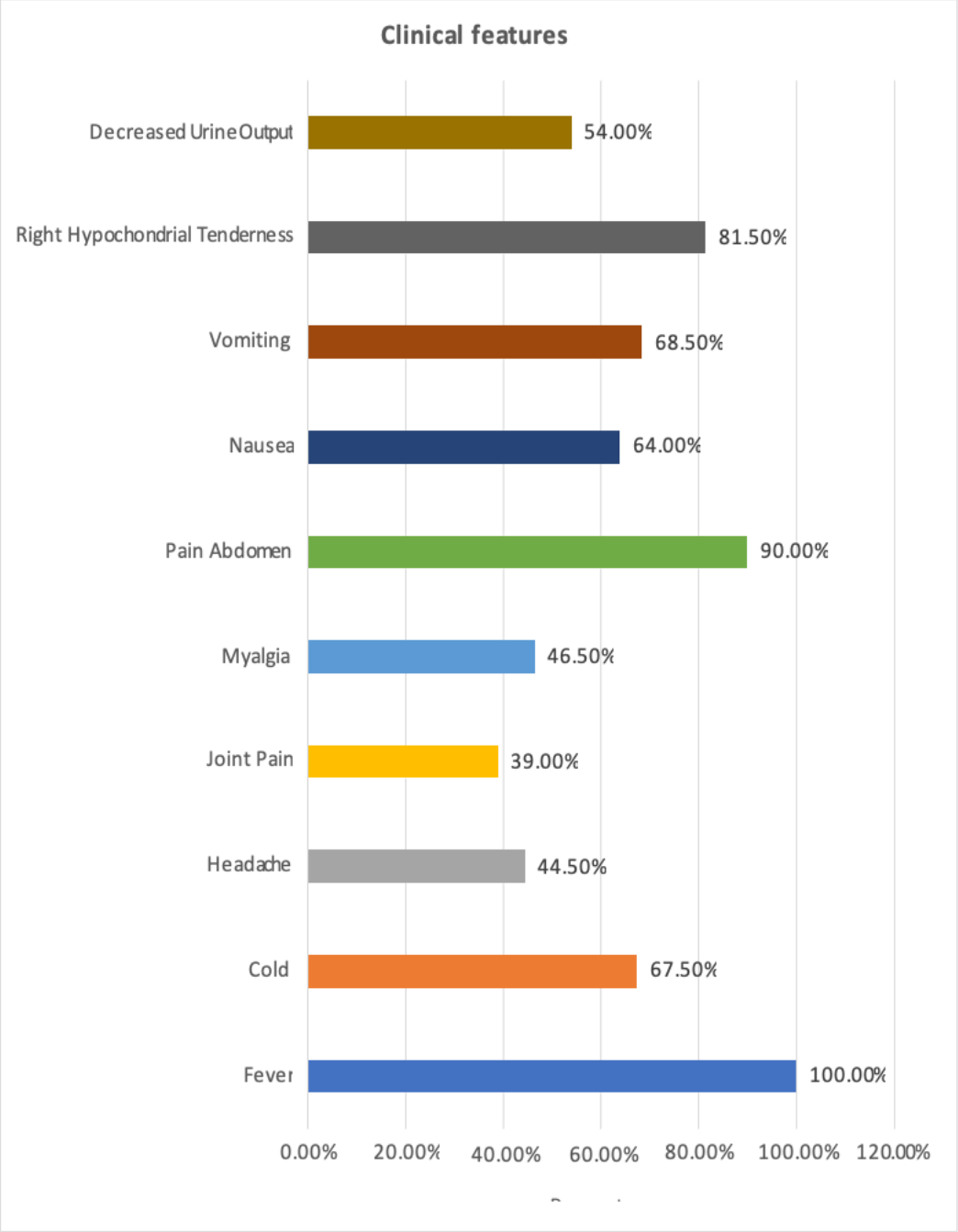

Figure 3: Frequency of presenting complaints among the study subjects (n=200)

Table 4: Baseline vitals of the study subjects (N=200)

Vital parameters (N=200)		Frequency (N)	Percentage (%)
MAP <80 mmHg	Yes	38	19.0%
	No	162	81.0%
Hepatomegaly	Yes	136	68.0%
	No	64	32.0%

(MAP – Mean arterial pressure)

Vital parameters (N=200)	Mean	SD	Minimum	Median	Maximum
PR (in bpm)	93.68	18.35	68.00	89.00	154.00

(PR – Pulse pressure)

A striking 81% of children presented with a mean arterial pressure (MAP) above 80 mmHg, suggesting that shock or hypotension was not prevalent at admission in most cases. Hepatomegaly was observed in 68% of the patients, indicating significant hepatic involvement—often seen in dengue and similar infections. The mean pulse rate (PR) was 93.68 bpm, with a wide range (68–154 bpm), and a median of 89 bpm, denoting variable hemodynamic responses. The substantial majority with normal MAP and high hepatomegaly frequency implies a compensatory phase in most children, where systemic manifestations were present without overt circulatory collapse. Therefore, it is clear that while hepatomegaly was a common clinical feature, most children were hemodynamically stable on presentation, pointing to early or moderate stages of illness in a majority

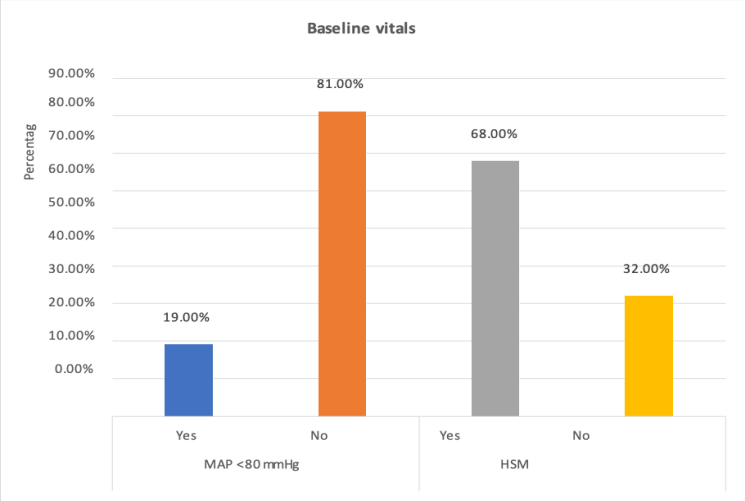

Figure 4: Baseline vitals of the study subjects (N=200)

Table 5: Variation in the platelet count among the study subjects (N=200)

Platelet count (N=200)	Mean	SD	Minimum	Median	Maximum
Day 1 (x1 lakh/µl)	0.55	0.35	0.07	0.48	1.94
Day 2 (x1 lakh/µl)	0.66	0.38	0.12	0.60	1.97
Day 3 (x1 lakh/µl)	0.78	0.40	0.15	0.70	1.98
Day 4 (x1 lakh/µl)	0.93	0.45	0.20	0.84	2.24
Day 5 (x1 lakh/µl)	1.08	0.46	0.27	1.02	2.42
Day 6 (x1 lakh/µl)	1.26	0.47	0.25	1.24	2.52
Day 7 (x1 lakh/µl)	1.44	0.44	0.52	1.40	2.68
Day 8 (x1 lakh/µl)	1.60	0.44	0.62	1.52	2.82
Day 9 (x1 lakh/µl)	1.72	0.47	0.76	1.71	3.00
Day 10 (x1 lakh/µl)	1.87	0.52	0.90	1.92	3.20
Day 11 (x1 lakh/µl)	2.03	0.58	1.00	2.14	3.82

Normal Platelet count – 1.5 to 4.5 lakh/µl
Repeated measures ANOVA
p-value <0.001 (Statistically significant)

The trend in platelet counts across 11 days revealed a consistent and statistically significant ($p<0.001$) upward trajectory. On day 1, the mean platelet count was 0.55 lakh/µl, progressively increasing to 2.03

lakh/µl by day 11. Each day marked a rise from the previous, with the median values closely mirroring the mean trend. This steady improvement in platelet count across the majority of the cohort indicates recovery from the thrombocytopenic phase characteristic of viral hemorrhagic fevers, particularly dengue. The gradual restoration of platelet counts over time reinforces the utility of serial platelet monitoring as a prognostic tool. The inference drawn is that the majority of patients demonstrated progressive normalization of platelet count, highlighting a positive trajectory in the disease course under appropriate management.

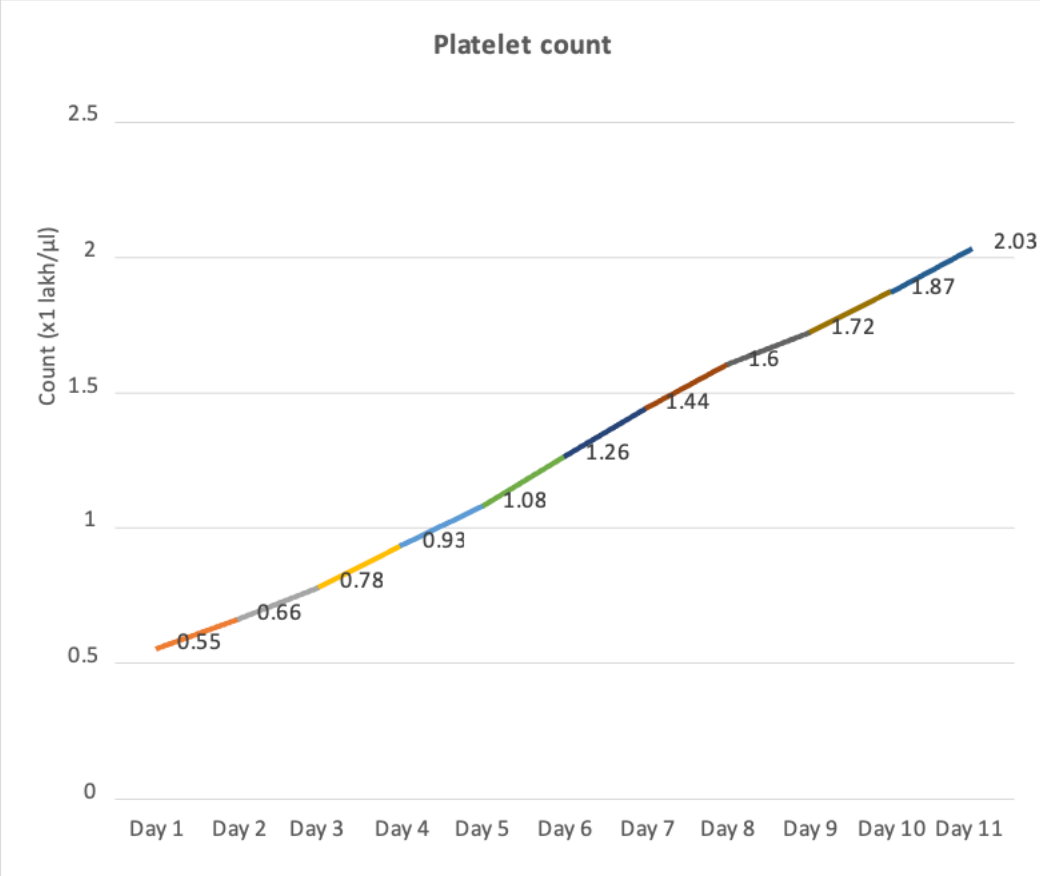

Figure 5: Variation in the platelet count among the study subjects (N=200)

Table 6: Variation in the mean platelet volume among the study subjects (N=200)

MPV (N=200)	Mean	SD	Minimum	Median	Maximum
Day 1 (fL)	11.54	0.69	9.20	11.80	12.50
Day 2 (fL)	11.23	0.77	8.90	11.55	12.40
Day 3 (fL)	10.95	0.81	8.60	11.30	11.90
Day 4 (fL)	10.69	0.85	8.10	10.90	12.20
Day 5 (fL)	10.44	0.86	7.90	10.70	11.70
Day 6 (fL)	10.18	0.84	7.70	10.30	11.70
Day 7 (fL)	10.00	0.83	7.40	10.10	11.70
Day 8 (fL)	9.79	0.93	7.10	9.80	11.80
Day 9 (fL)	9.56	0.96	7.10	9.50	11.70
Day 10 (fL)	9.32	1.05	7.00	9.30	11.60
Day 11 (fL)	9.10	1.17	7.00	8.90	11.90

Normal Mean platelet volume (MPV) - 7.2 and 11.7 fL
Repeated measures ANOVA
p-value <0.001 (Statistically significant)

Mean Platelet Volume (MPV) showed a consistent decline over the 11-day observation period, from a mean of 11.54 fL on day 1 to 9.10 fL by day 11, with the trend being statistically significant ($p<0.001$). The majority of patients followed this decreasing pattern, indicating that initially large-sized platelets

(which are young and more active) were gradually replaced by smaller, mature and more stable platelets as recovery ensued. The reduction in MPV aligns with the increase in platelet count seen concurrently, signifying a shift from a reactive thrombopoietic state to normalization. The decreasing standard deviation over time also suggests convergence towards more uniform platelet characteristics. This observation shows that most patients experienced a normalization of platelet production and morphology during recovery, confirming MPV as a dynamic marker of disease progression and hematological recovery.

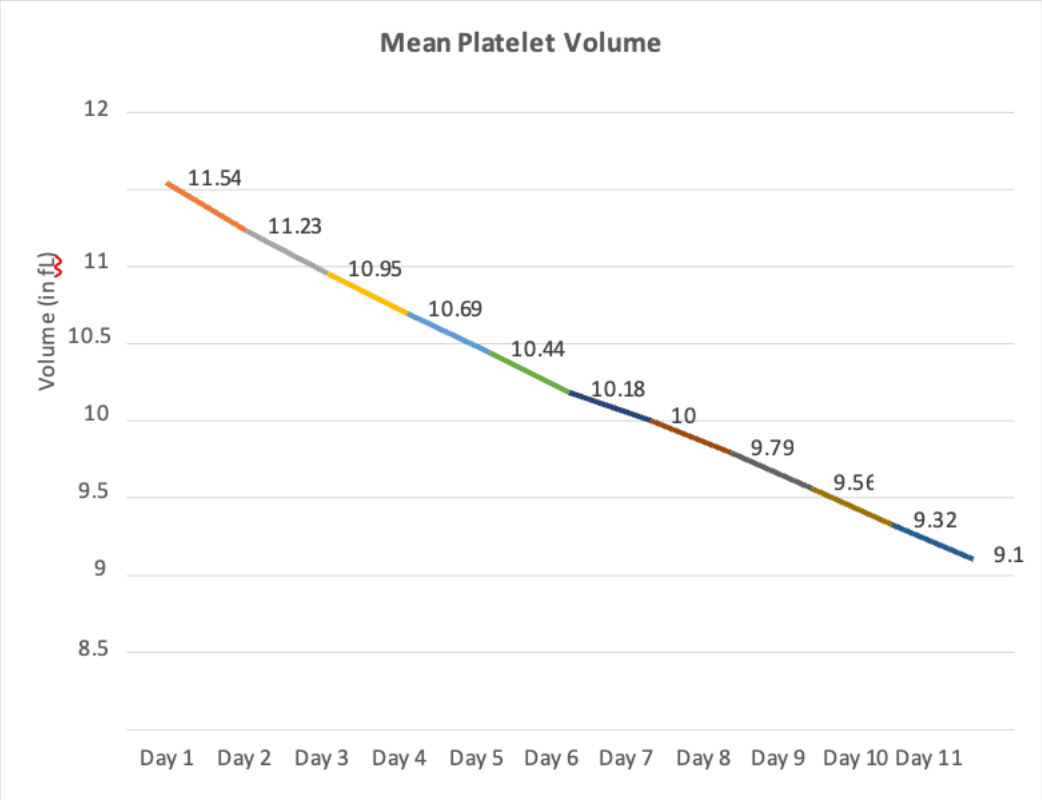

Figure 6: Variation in the mean platelet volume among the study subjects (N=200)

Table 7: Variation in the platelet distribution width among the study subjects (N=200)

PDW (N=200)	Mean	SD	Minimum	Median	Maximum
Day 1 (%)	16.09	1.41	10.60	16.00	19.20
Day 2 (%)	15.82	1.26	11.20	15.90	18.80
Day 3 (%)	15.55	1.18	10.90	15.60	18.80
Day 4 (%)	15.18	1.22	10.10	15.50	17.60
Day 5 (%)	14.94	1.29	9.90	15.30	17.20
Day 6 (%)	14.62	1.32	9.90	14.90	16.70
Day 7 (%)	14.32	1.41	9.50	14.70	16.40
Day 8 (%)	13.95	1.44	9.10	14.10	16.30
Day 9 (%)	13.62	1.55	9.50	13.85	16.40
Day 10 (%)	13.31	1.66	9.30	13.60	16.20
Day 11 (%)	13.08	1.70	8.90	13.30	15.90

Normal Platelet distribution width(PDW) - 10.0% to 17.9%
Repeated measures ANOVA
p-value <0.001 (Statistically significant)

Platelet Distribution Width (PDW) progressively declined from day 1 to day 11, with a statistically significant trend ($p<0.001$). On day 1, the mean PDW was

16.09 fL, which gradually dropped to 13.08 fL by day 11. The highest variability was noted at the beginning, with a wider range and higher standard deviation, which narrowed consistently over the subsequent days. This pattern reflects that initially, a majority of patients had heterogeneous platelet populations, typically observed during acute thrombo-

cytopenia when the bone marrow responds with a surge of immature and variably sized platelets. As the patients began to recover, the platelet population stabilized, leading to a reduced PDW. The convergence in values indicates restoration of normal platelet morphology in most patients. Hence, the implication drawn is that PDW reliably mirrored disease recovery in the majority, with decreasing anisocytosis of platelets indicating resolution of the acute hemorrhagic state.

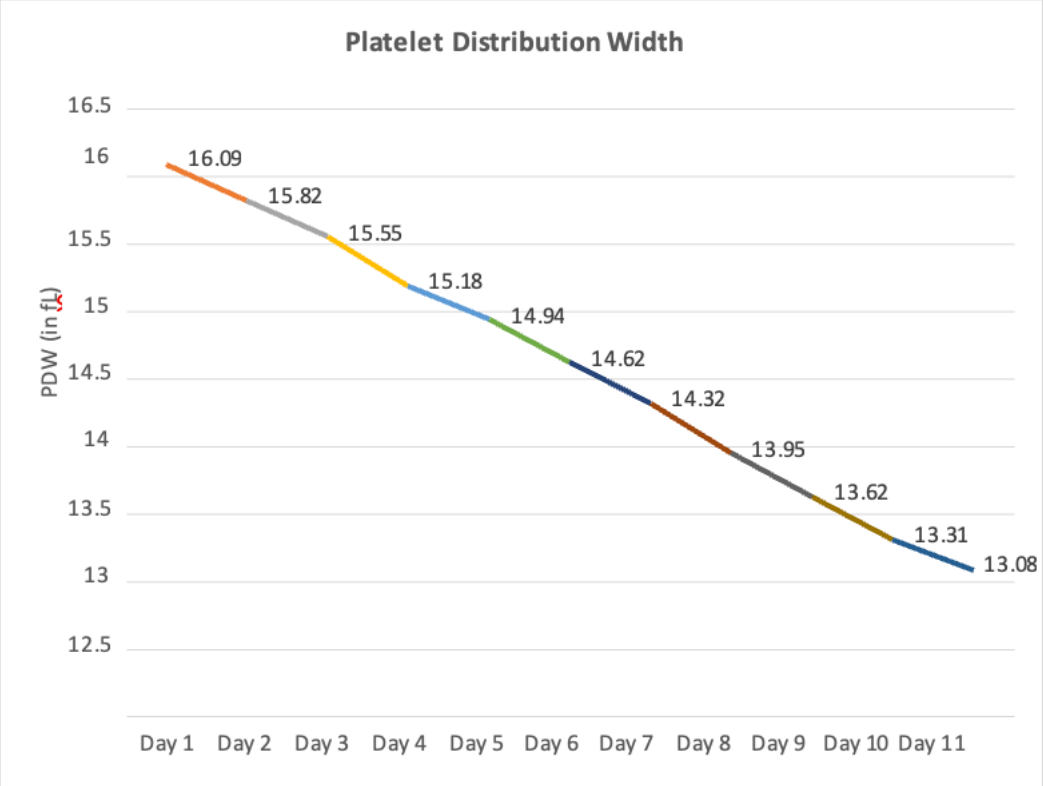

Figure 7: Variation in the platelet distribution width among the study subjects (N=200)

Discussion

This prospective observational study was conducted over 18 months in a tertiary care hospital, enrolling pediatric patients aged 1 month to 14 years diagnosed with viral hemorrhagic fever (VHF) and thrombocytopenia (platelet count <1.5 lakh/cumm). Patients were included based on clinical suspicion and laboratory confirmation via serological tests like NS1 antigen. Exclusion criteria comprised neonates, children above 14 years, those with co-morbidities (e.g., congenital heart disease, cerebral palsy, co-infections), and those who had received platelet-rich plasma transfusions.

Eligible patients presenting to the pediatric outpatient or inpatient departments were evaluated clinically and underwent serial monitoring of platelet indices using an automated analyzer. Confirmatory tests for VHF were performed in relevant cases. Platelet indices including platelet count, mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit were recorded daily for 11 days. The study aimed to correlate these indices with the clinical course—improvement or deterioration—of VHF.

Primary outcomes included assessment of the variability in platelet indices and their association with disease severity. Secondary outcomes focused on demographic trends such as age, gender, regional, and socioeconomic factors. Data were analyzed using repeated measures ANOVA to determine statistically significant changes in platelet parameters over time.

Characteristics of the study subjects

Table 8: Comparison of age of the study subjects with other studies

Age	Present study	Jayashree et al.[4]	Sontakke et al[5]	Makwana et al.[6]	Bashir et al.[7]	Sharma et al.[8]
1 month to 1 year	15%	11%	-	-	-	-
1 to 5 years	28.0%	24%	13.7%	-	-	-
6 to 10 years	34.5%	48%	37%	-	-	-
10 to 14 years	22.5%	17%	20.5%	-	-	-
14 to 18 years	-	-	28.8%	-	-	-

The present study reported that children aged 6–10 years were most commonly affected, a finding that is strongly supported by previous studies conducted by Jayashree et al. and Sontakke et al. both of which identified peak incidence in children between 6–10 years. However, the study by Makwana et al. found a peak incidence between 10–18 years in the pediatric population, though the lack of specific age group categorization in their analysis limits a deeper understanding of narrower age ranges.

In contrast, studies by Bashir et al. and Sharma et al., which focused on adult cohorts, indicate that age distribution patterns may differ between children and adults. These findings suggest that while certain age-related trends may be consistent within specific groups, the age distribution of the condition can vary depending on the population, setting, or age group being studied

Table 9: Comparison of gender of the study subjects with other studies

Gender	Present study	Jayashree et al.[4]	Makwana et al.[6]	Bashir et al.[7]	Sharma et al.[8]
Male	59.5%	58%	52%	65%	63.5%
Female	40.5%	42%	48%	35%	36.5%

The present study, conducted in a pediatric age group, reported a male predominance of 59.5%, a finding that is consistent with previous studies by Jayashree et al. and Makwana et al., which reported male predominance rates of 58% and 52%, respectively, in pediatric populations. Similarly, studies by Bashir et al. and Sharma et al., though conducted in broader or adult populations, also demonstrated a male preponderance (65% and 63.5%, respectively), further supporting the male predominance observed in the present study. These findings suggest that male gender predominance is consistent across different age groups, potentially reflecting underlying behavioral, biological, or care-seeking differences.

Presenting complaints

Table 10: Comparison of presenting complaints with other studies

Clinical features (N=200)	Freqeuncy Percentage (%) of present study	Freqeuncy Percentage (%) of Bashir et al.[7]	Freqeuncy Percentage (%) of Makwana et al.[6]	Freqeuncy Percentage (%) of Sharma et al.[8]	Freqeuncy Percentage (%) of Jayashree et al.[4]	Freqeuncy Percentage (%) of Arora et al.[9]	Freqeuncy Percentage (%) of Mukker et al.[10]	Freqeuncy Percentage (%) of Manoharan et al.[11]
Fever	100.0%	100%	100%	100%	100%	-	-	-
Cold	67.5%	-	-	-	-	-	-	-
Headache	44.5%	84.4%	-	-	-	-	-	-
Joint Pain	39.0%	78.2%	-	-	-	-	-	-
Myalgia	46.5%	46.7%	-	-	-	-	-	-
Pain Abdomen	90.0%	-	-	-	-	-	-	-
Nausea	64.0%	-	-	-	-	-	-	-
Vomiting	68.5%	-	-	-	-	-	-	-
Right Hypochondrial Tenderness	81.5%	-	-	-	-	-	-	-
Decreased Urine Output	54.0%	-	-	-	-	-	-	-

In the present study, fever was reported in 100% of cases, with a high frequency of gastrointestinal and systemic symptoms such as abdominal pain and vomiting. Fever was universally present in nearly all previous studies, including Bashir et al., Makwana et al., Sharma et al., and Jayashree et al., indicating

its role as the hallmark symptom of dengue. Bashir et al. also noted joint pain and headache, while Makwana et al. highlighted bleeding (32.85%) and neurological symptoms, demonstrating broader multisystem involvement. While Arora et al. and Mukker et al. did not list individual symptoms, their alignment with WHO clinical definitions implies similar presentations. Jayashree et al. and Sharma et al. affirmed fever as a consistent complaint but did not provide symptom frequency data. Manoharan et al. focused on bleeding in critically ill patients, and Elsewefy et al. did not address clinical symptoms, focusing instead on hematological indices. The convergence across studies reinforces fever as a universal finding and supports the inclusion of gastrointestinal and systemic complaints as common clinical indicators in dengue.

Baseline vitals

Table 11: Comparison of baseline vitals with other studies

Vital parameters (N=200)		Present study Percentage (%)	Percentage (%) Makwana et al.[6]	Percentage (%) Jayashree et al.[4]	Percentage (%) Manoharan et al.[11]
MAP <80 mmHg	Yes	19.0%	16%	9.5%	-
	No	81.0%	84%	91.5%	-
Hepatomegaly	Yes	68.0%	-	-	-
	No	32.0%	-	-	-

The present study revealed that 19% of patients had a mean arterial pressure (MAP) below 80 mmHg, and 68% had hepatomegaly. While most previous studies did not report MAP directly, Makwana et al. documented that 16% of patients developed shock and 22% required ICU admission, indirectly supporting the hemodynamic findings of the present study. Jayashree et al. applied WHO criteria to identify DSS cases (9.5%), which involve circulatory failure, aligning conceptually with the use of MAP in the present study. correlated high MPV with critical illness and bleeding, indirectly linking it to hemodynamic compromise. Hepatomegaly was not evaluated in other studies. Therefore, while direct comparison is limited, studies like those by Makwana et al. and Jayashree et al. support the relevance of hemodynamic indicators as markers of disease severity, underscoring the prognostic utility of early vital sign monitoring observed in the present study.

Platelet count over time

Table 12: Comparison of platelet count over time with other studies

Mean platelet count	Present study	Makwana et al.[6]	Manoharan et al.[11]	Arora et al.[9]	Bashir et al.[7]	Mukker et al.[10]	Sharma et al.[8]
Admission to Discharge	0.55 to 2.03 (x1 lakh/µl)	0.64 to 1.07(x1 lakh/µl)	0.20 to 1.2(x1 lakh/µl)	-	-	0.15 to 0.885(x1 lakh/µl)	-

The present study showed a progressive increase in platelet counts from 0.55 lakh/µL on day 1 to 2.03 lakh/µL on day 11, with a statistically significant trend ($p<0.001$), indicating recovery. This observation is strongly supported by previous studies by Makwana and Mukker et al., which demonstrated that platelet recovery corresponded with clinical improvement. Manoharan et al. tracked post-transfusion platelet increments, finding minimal responses in some patients with high MPV, further reinforcing the relevance of platelet dynamics. Although Arora et al. and Bashir et al. did not monitor platelet counts over time, they confirmed the association of thrombocytopenia with disease severity. Sharma et al. reported that 98% of dengue patients had thrombocytopenia but did not track platelet count changes longitudinally. These collective findings affirm that thrombocytopenia is a key diagnostic and prognostic feature, and its recovery serves as a useful clinical marker, as highlighted in the present study.

Conclusion

The present study highlights the prognostic significance of platelet indices (like platelet count, MPV, PDW and PCT) and hemodynamic indicators in assessing disease severity and recovery in pediatric viral hemorrhagic fever, particularly dengue. Mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) demonstrated dynamic associations with clinical outcomes, reinforcing their role as reliable biomarkers. Lower MPV and PDW correlated with severe disease, while their normalization aligned with recovery. Additionally, lower PCT values were linked to worse outcomes, emphasizing its utility beyond platelet count alone. Elevated liver function test levels further indicate hepatic dysfunction, serving as a marker of dengue severity.

These findings underscore the importance of routine monitoring of platelet indices and hemodynamic parameters for early risk stratification. Their systematic assessment can aid in guiding clinical decision-making and optimizing the management of pediatric viral hemorrhagic fever, particularly dengue

References

1. Parvaiz A Koul, Nargis K Bali;Viral hemorrhagic fever in india David C. Pigott, MD; Hemorrhagic Fever Viruses; Crit Care Clin 21 (2005) 765 – 783.
2. Debatosh paul, Amit K Pramanik, Sanchita Biswas, Chita das et al, International journal of advances in medicine, volume 10, issue 3,2023; 223-227
3. Jayashree K, Manasa GC, Pallavi P, Manjunath GV. Evaluation of platelets as predictive parameters in dengue fever. Indian J Hematol Blood Transfus. 2011;27(3):127–30
4. Sontakke RA, Aglave NR, Dua H. Correlation of Platelet Parameters With the Severity of Thrombocytopenia in Dengue Fever in Children Aged Less Than 18 Years at a Tertiary Care Centre: A Cross-Sectional Study. Cureus. 2024 Mar 24;16(3).
5. Makwana M, Kumari S, Mourya HK, Mitharwal R, Ram S. A study to find out the relationship between various platelet indices and morbidity profile of dengue fever in pediatric patients on admission in Western Rajasthan, India. Pediatr Infect Dis.2020;2(2):43–50.
6. Bashir AB, Mohammed BA, Saeed OK, Ageep AK. Thrombocytopenia and bleeding manifestations among patients with dengue virus infection in Port Sudan, Red Sea State of Sudan. J Infect Dis Immun. 2015;7(2):7–13.
7. Sharma K, Yadav A. Association of mean platelet volume with severity, serology and treatment outcome in dengue fever: Prognostic utility. J Clin Diagn Res. 2015;9(11):EC01–3
8. Arora S, Gupta M, Tahlan A, Gupta S, Jesrani G. Role of mean platelet volume and platelet distribution width in assessing the severity of dengue fever: An observational study. J NTR Univ Health Sci. 2022;11(4):328–32
9. Mukker P, Kiran S. Platelet indices evaluation in patients with dengue fever. Int J Res Med Sci. 2018;6(6):2054–9.
10. Manoharan A, Sinha P. Role of mean platelet volume in dengue fever - a prognostic marker. Int J Res Rev. 2017;4(6):72–6.