



● ORIGINAL ARTICLE

Assessment of Platelet Count and Platelet Distribution Width among Sudanese Patients with Dengue Fever in Kassala State, Sudan

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Abstract

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BACKGROUND

Dengue fever is a mosquito-borne viral infection and a significant public health concern in Sudan, including Kassala State, where outbreaks recur. The infection is often associated with a decline in platelet count and changes in platelet distribution width (PDW), both used as candidate diagnostic and prognostic markers of disease severity. This study assessed platelet count and PDW among Sudanese patients with dengue fever in Kassala State and compared these parameters with those of healthy controls.

METHODS

This comparative analytical cross-sectional, laboratory-based study included 50 dengue fever patients and 50 age- and sex-matched apparently healthy controls from the same source population. Platelet count and PDW were measured using a Sysmex KX-21N hematology analyzer.

RESULTS

The mean age of patients was 38.5 ± 9.8 years, and 68.0% were male. Disease severity was classified as mild (32.0%), moderate (38.0%), and severe (30.0%). Platelet count was significantly lower in dengue patients than in controls (95.5 ± 59.5 vs $250.9 \pm 70.1 \times 10^9/L$, $P < 0.001$), while PDW was significantly higher in dengue patients than in controls ($24.8 \pm 5.7\%$ vs $20.6 \pm 5.5\%$, $P < 0.001$). No significant correlation was observed between age and either platelet count or PDW. However, platelet count decreased, and PDW increased significantly as disease severity increased ($P < 0.001$).

CONCLUSION

Platelet count was significantly lower in dengue patients than in healthy controls and decreased further with increasing disease severity, while PDW was significantly higher than in controls and increased further with increasing severity.

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KEYWORDS

Dengue fever · Hematology analyzer · Platelet count · Platelet distribution width · Sudan · Thrombocytopenia



Introduction

Dengue fever is the most rapidly spreading mosquito-borne viral infection worldwide, with a more than 30-fold rise in incidence over the past decade and expansion into new geographic regions.¹⁻⁴ More than 100 countries are now considered endemic, and the World Health Organization (WHO) estimated a record 14 million cases and over 9,000 deaths globally in 2024 alone.^{5,6} Although dengue was historically uncommon in Africa, large outbreaks have occurred across the continent since the year 2000, and the virus is increasingly recognized as an emerging public health threat in the region.⁷ In Sudan, recurrent outbreaks have been documented, particularly in the Red Sea State, underscoring the need for region-specific hematological data to guide clinical management.⁸ Cross-sectional serosurveys have further confirmed active dengue transmission and identified associated risk factors among residents of eastern Sudan, including Kassala and neighboring El-Gadarif state.^{9,10}

Dengue virus is transmitted primarily by *Aedes* mosquitoes that breed readily in small collections of stagnant water; infection typically presents as an acute febrile illness that can progress, in a minority of cases, to severe disease with plasma leakage or hemorrhagic manifestations.^{2,5} Kassala State, in eastern Sudan bordering Eritrea, combines a hot climate, seasonal rainfall, and population movement that favor sustained vector transmission, making it one of the areas repeatedly affected by dengue outbreaks.⁸

Dengue virus (DENV) comprises four antigenically related but genetically distinct serotypes (DENV-1–DENV-4); secondary infection with a different serotype carries a higher risk of severe disease through antibody-dependent enhancement.¹ Local surveillance in Kassala has confirmed simultaneous circulation of all four DENV serotypes during a recent outbreak at Kassala Teaching Hospital,¹¹ while an earlier assessment of the 2019 Kassala outbreak identified important gaps in surveillance and laboratory response capacity that continue to hamper timely case detection in the state.¹²

Dengue virus infection frequently causes thrombocytopenia, and platelet indices derived from the routine complete blood count, platelet count, mean platelet volume, plateletcrit (PCT), and platelet

distribution width (PDW) have attracted growing interest as diagnostic and prognostic tools.^{13,14}

Platelet distribution width reflects variability in platelet size and is regarded as a marker of platelet activation and turnover; several authors have proposed it as a low-cost adjunct to platelet count for assessing dengue severity.¹⁴ The mechanisms underlying dengue-associated thrombocytopenia are multifactorial and include direct viral suppression of bone-marrow megakaryocytes, increased peripheral platelet destruction through immune-mediated mechanisms, and platelet consumption secondary to endothelial activation and mild coagulopathy.^{13,15} As platelets are cleared and replaced during infection, the circulating population becomes more heterogeneous in size, a change that can, in principle, be captured by platelet distribution width. Because platelet indices are generated automatically with every complete blood count at negligible additional cost, they are attractive candidate biomarkers for resource-limited settings such as Kassala State, provided that their diagnostic performance is locally validated rather than extrapolated from other populations.¹⁴

Previous studies have reported inconsistent findings regarding PDW in dengue infection. Wayez et al. observed progressively higher PDW values with more severe thrombocytopenia,¹⁶ and Mukker and Kiran reported significant PDW alterations among patients with markedly reduced platelet counts.¹⁷ Zeb et al. similarly described altered platelet indices among Pakistani dengue patients.¹⁸ Elsewhere, Khatri et al. and Nehara et al. reported significantly higher PDW among Indian dengue patients with thrombocytopenia compared with those without,^{19,20} while Navya et al. and Kantharaj found platelet indices useful for guiding clinical decisions, including transfusion requirements, in dengue-positive patients.^{21,22} In Sudan, Bashir et al. reported that low platelet count, low mean platelet volume, and high PDW could serve as probable indicators of dengue infection in Red Sea State,⁸ and a subsequent Sudanese study similarly documented pronounced thrombocytopenia and bleeding manifestations among dengue patients in Port Sudan,²³ while a more recent study assessed these indices in Khartoum State.²⁴ However, no prior study conducted in Kassala State has directly compared platelet count and PDW between dengue patients and healthy controls, or

examined these parameters across clinical severity categories, in this specific population.

The present study therefore aimed to assess platelet count and PDW among Sudanese patients with dengue fever in Kassala State and to compare these parameters with those of healthy individuals, in order to establish locally relevant reference data rather than assuming that findings from other Sudanese states apply directly to Kassala.

Materials and Methods

Study Design and Setting

This was a comparative analytical cross-sectional, laboratory-based study conducted at Kassala Teaching Hospital, Kassala State, Sudan. Data were collected between September and October 2025.

Study Population and Sampling

Participants comprised Sudanese patients aged ≥ 18 years with a clinical diagnosis of dengue fever (cases, per WHO criteria),²⁵ recruited from among outpatients and inpatients presenting to Kassala Teaching Hospital during the study period, and age- and sex-matched apparently healthy controls with no history of dengue fever or other hematological disorders, recruited by convenience sampling from hospital staff, patient companions, and community volunteers drawn from the same catchment area and time frame as the cases. Age- and sex-matching between cases and controls was performed by frequency matching: controls were recruited so that the overall age-group and sex distribution of the control set matched that of the cases, without pairing individual controls to individual cases.

Sample Design

The minimum sample size required for the study was calculated using the formula for comparison of two independent means:

$$n = 2(Z\alpha/2 + Z\beta)^2 (\sigma^2/d^2)$$

where $Z\alpha/2 = 1.96$, $Z\beta = 0.84$ (80% power), σ is the control-group standard deviation of platelet count reported in a recent Sudanese case-control study of dengue-associated thrombocytopenia (Ahmed et al., 2025),²⁴ and d is the minimum clinically important difference in platelet count. A value of $d = 40 \times 10^9/L$ was chosen because it approximates one standard deviation of the reference control-group platelet count and was

therefore considered the smallest difference likely to be clinically meaningful; this calculation was based on platelet count alone.

Using $\sigma \approx 38.4 \times 10^9/L$, the minimum calculated sample size was 15 per group; this was increased to 50 per group to allow for possible data exclusion.

Inclusion and Exclusion Criteria

For cases, Sudanese patients aged 18 years and above who were diagnosed with dengue fever and who agreed to take part in the study were included. Individuals with bleeding disorders, thrombocytopenia unrelated to dengue infection, chronic diseases (diabetes mellitus, hypertension, cardiovascular, hepatic, or renal disease), pregnant women, and patients who had received a platelet transfusion within the preceding six months were excluded from both the case and control groups. Both patients and control subjects were asked to sign informed consent documents after due explanation.

Sample Collection and Laboratory Methods

A total of 100 participants were enrolled: 50 cases and 50 controls by convenience sampling.

Dengue fever was diagnosed clinically based on the WHO case definition (fever plus two or more of the following: nausea/vomiting, rash, headache, myalgia/arthralgia, positive tourniquet test, leukopenia), and confirmed by NS1 antigen/IgM serology.²⁵ Three milliliters of venous blood were then collected under sterile conditions from each participant into EDTA anticoagulant tubes and processed promptly, or stored at -20°C when immediate analysis was not possible. Platelet count and PDW were measured using an automated Sysmex KX-21N hematology analyzer, which determines platelet number and size distribution by electrical impedance sensing across a calibrated aperture. The manufacturer's reference ranges were $150\text{--}400 \times 10^9/L$ for platelet count and 8.3–56.6% for PDW.

Quality Control

Internal quality control was performed daily using commercial control blood samples run on the Sysmex KX-21N analyzer before testing of study samples, in accordance with the manufacturer's recommendations. Results falling outside the manufacturer-defined control range triggered recalibration before analysis resumed.

Data and Sample Collection

Data collection encompassed both the laboratory blood sampling procedure described above and questionnaire-

based data. Demographic data (age, sex, residence) and clinical information, including clinician-assessed disease severity (mild, moderate, and severe) based on WHO dengue case assessment criteria, were collected using a structured questionnaire administered to each participant.²⁵

Statistical Analysis

Data were analyzed using SPSS version 26. The normality of continuous variables was assessed and homogeneity of variance was checked prior to parametric testing; Spearman's correlation was used in place of Pearson's correlation where these assumptions were not met. Continuous variables were summarized as mean $\pm$ standard deviation and compared between cases and controls using the independent-samples t-test. The age and sex distributions of cases and controls were compared (Table 1a) to confirm that matching was achieved at the group level.

Comparison of platelet count and PDW across the three severity categories was performed using one-way ANOVA, with Bonferroni-corrected pairwise post-hoc comparisons between severity groups where the overall ANOVA was significant. Categorical variables were expressed as frequencies and percentages. Pearson's (or Spearman's, as applicable) correlation coefficient was used to assess the relationship between age and platelet parameters. A P-value ≤ 0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the Research Ethics Committee of Kassala Teaching Hospital, in coordination

with the Kassala State Ministry of Health. Written informed consent was obtained from all participants after the study objectives and any risks associated with sampling were explained. All participant information was kept confidential.

Results

A total of 100 participants (50 dengue fever patients and 50 healthy controls) were analyzed. Among the patients, the mean age was 38.5 ± 9.8 years; the largest proportion were aged 31–40 years (32.0%), followed by 20–30 years (28.0%), 41–50 years (24.0%), and 51–60 years (16.0%). Sixty-eight percent ($n = 34$) of patients were male, and 32.0% ($n = 16$) were female. Regarding disease severity, 38.0% ($n = 19$) had moderate disease, 32.0% ($n = 16$) had mild disease, and 30.0% ($n = 15$) had severe disease (Table 1).

Table 1. Demographic and Clinical Characteristics of Dengue Fever Patients ($n = 50$).

Characteristic	Category	n	%
Age (years)	20–30	14	28.0
	31–40	16	32.0
	41–50	12	24.0
	51–60	8	16.0
Sex	Male	34	68.0
	Female	16	32.0
Disease severity	Mild	16	32.0
	Moderate	19	38.0
	Severe	15	30.0

Table 1a. Demographic Comparison between Dengue Fever Patients and Healthy Controls ($n = 50$ each).

Characteristic	Category	Cases (n)	Controls (n)	P-value
Age (years)	20–30	14	12	0.966
	31–40	16	17	
	41–50	12	10	
	51–60	8	7	
Sex	Male	34	32	0.943
	Female	16	17	

Comparison of platelet parameters between dengue fever patients and healthy controls showed a significant reduction in platelet count among patients (95.5 ± 59.5 vs $250.9 \pm 70.1 \times 10^9/L$, $P < 0.001$), while PDW values were also significantly higher among dengue patients than controls ($24.8 \pm 5.7\%$ vs $20.6 \pm 5.5\%$, $P < 0.001$).

The mean difference in platelet count between dengue patients and controls was $-155.4 \times 10^9/L$ (95% CI: -180.9

to -129.9 ; independent-samples t-test, $P < 0.001$). The mean difference in PDW was 4.2% (95% CI: 2.0 to 6.4; $P < 0.001$), consistent with a significant between-group difference. Confidence intervals were calculated from the reported means, standard deviations, and group sizes using the standard formula for the difference between two independent means (Table 2).

Table 2. Comparison of Platelet Count and PDW between Dengue Fever Patients and Healthy Controls.

Parameter	Dengue patients (n = 50)	Healthy controls (n = 50)	P-value
Platelet count ($\times 10^9/L$)	95.5 $\pm$ 59.5	250.9 $\pm$ 70.1	< 0.001
PDW (%)	24.8 $\pm$ 5.7	20.6 $\pm$ 5.5	< 0.001

The mean platelet count decreased progressively from mild to severe dengue (167.6 $\pm$ 28.2, 87.3 $\pm$ 22.4, and 28.9 $\pm$ 11.1 $\times 10^9/L$, respectively), while PDW increased significantly with disease severity (21.2 $\pm$ 3.5%, 24.2 $\pm$ 4.6%, and 29.3 $\pm$ 5.9%, respectively). One-way ANOVA confirmed a significant overall difference in platelet count across severity groups ($F(2,47) = 156.4$, $P < 0.001$) and in PDW ($F(2,47) = 11.6$, $P < 0.001$). Bonferroni-corrected pairwise comparisons ($\alpha = 0.0167$) showed that platelet count differed significantly between every pair of severity groups (mild vs moderate: mean difference 80.3 $\times 10^9/L$, 95% CI 62.4 to 98.2, $P < 0.001$;

moderate vs severe: 58.4 $\times 10^9/L$, 95% CI 46.3 to 70.5, $P < 0.001$; mild vs severe: 138.7 $\times 10^9/L$, 95% CI 122.8 to 154.6, $P < 0.001$), confirming a stepwise decline across all three categories. For PDW, pairwise comparisons showed significant differences between mild and severe dengue (mean difference -8.1%, 95% CI -11.7 to -4.5, $P < 0.001$) and between moderate and severe dengue (-5.1%, 95% CI -8.9 to -1.3, $P = 0.011$), while the difference between mild and moderate dengue did not reach the Bonferroni-corrected threshold (-3.0%, 95% CI -5.8 to -0.2, $P = 0.036$), indicating that the rise in PDW was most pronounced at the transition to severe disease (Table 3).

Table 3. Comparison of Platelet Count and PDW between Dengue Fever Patients According to Disease Severity

Parameter	Mild Dengue (n = 16)	Moderate Dengue (n = 19)	Severe Dengue (n = 15)	P-value
Platelet count ($\times 10^9/L$)	167.6 $\pm$ 28.2	87.3 $\pm$ 22.4	28.9 $\pm$ 11.1	< 0.001
PDW (%)	21.2 $\pm$ 3.5	24.2 $\pm$ 4.6	29.3 $\pm$ 5.9	< 0.001

No significant correlation was found between age and platelet count ($r = -0.137$, $P = 0.342$) or between age and PDW ($r = -0.031$, $P = 0.832$) among dengue fever patients (Table 4).

Table 4. Correlation between Age and Platelet Parameters among Dengue Fever Patients.

Parameter	r	P-value
Age vs platelet count	-0.137	0.342
Age vs PDW	-0.031	0.832

Discussion

This study assessed platelet count and PDW among Sudanese patients with dengue fever in Kassala State and compared these parameters with healthy controls. The findings confirm that dengue infection was associated with a marked and statistically significant reduction in platelet count, consistent with the well-established role of thrombocytopenia as a hallmark of dengue infection and a criterion used in clinical severity assessment.^{8,16-18} Most patients in this study were male and aged between 31 and 40 years; male predominance has similarly been reported in other dengue series, but as occupational exposure was not directly measured in this study, differential outdoor exposure to mosquito vectors among men remains only a plausible hypothesis rather than a demonstrated explanation. Disease severity was distributed relatively evenly across mild, moderate, and

severe categories in this sample, although the convenience sampling approach means this distribution should not be assumed to represent the true clinical spectrum of dengue presentation in Kassala State more broadly. Consistent with platelet count, PDW was also significantly higher among dengue patients than healthy controls in this study, in agreement with reports by Wayez et al.¹⁶ and Mukker and Kiran,¹⁷ who described significant elevations in PDW with worsening thrombocytopenia, and with Bashir et al.,⁸ who proposed high PDW as a probable indicator of dengue infection in Red Sea State, Sudan. This finding is also broadly consistent with the more recent Khartoum State study by Ahmed et al.,²⁴ reinforcing PDW's potential value as a low-cost adjunct marker of dengue infection across different Sudanese settings. Similarly, Zeb et al. reported measurable alterations in platelet indices among dengue patients in Pakistan,¹⁸ and larger hospital-based cohorts by Asha et al. and Antony Gnana Shahila and Jothilingam have likewise shown significant differences in platelet indices between dengue patients and controls, though the specific parameters affected varied across studies.^{26,27} Some variation in effect size across these studies may still relate to differences in the day of illness at which blood was sampled relative to the onset of fever, the proportion of patients with severe versus mild disease, sample size, and possible platelet clumping or analyzer-related variability.

In the present study, platelet count decreased significantly with increasing disease severity, whereas PDW increased progressively from mild to severe dengue cases ($P < 0.001$). Our findings are consistent with those of Wayez et al.,¹⁶ who reported increased PDW in dengue patients with thrombocytopenia and highlighted the clinical value of platelet indices during disease progression. Similarly, Bashir et al.⁸ found significantly higher PDW and lower platelet counts in Sudanese dengue patients compared with healthy controls.

These findings suggest that both platelet count and PDW were consistently and significantly altered among dengue patients compared with controls in this population, supporting a potential complementary role for PDW alongside platelet count in dengue assessment in similar endemic settings. However, because this study did not evaluate sensitivity, specificity, or predictive values, these findings describe an association rather than an established diagnostic performance for either parameter.

The absence of a significant correlation between age and either platelet count or PDW indicates that age alone does not predict the degree of thrombocytopenia or platelet size variability in this cohort, suggesting that other factors such as viral load, infecting serotype, timing of presentation, or host immune response may play a more prominent role and warrant further investigation. Because platelet count declined consistently and significantly in this cohort, it remains a simple, inexpensive, and widely available parameter that was associated with dengue infection in this population.

Conclusion

Platelet count was significantly reduced among Sudanese dengue fever patients in Kassala State compared with healthy controls and decreased further with increasing disease severity, supporting an association between thrombocytopenia and dengue infection and severity in this population. PDW was also significantly higher in dengue patients than in healthy controls overall, and increased further with increasing dengue severity. These findings should be interpreted as cross-sectional associations rather than evidence of diagnostic or prognostic accuracy, and platelet count and PDW should continue to be monitored as part of the routine hematological assessment of suspected dengue patients pending further validation.

Future Research

Future studies should adopt multicenter, prospective designs with larger and randomly selected samples across different regions of Sudan to improve generalizability and allow assessment of platelet count and PDW trends across the course of illness. Incorporating dengue serotype identification and viral load quantification would help clarify their contribution to the variability in platelet indices observed across studies, while formal evaluation of the sensitivity, specificity, and predictive value of platelet count and PDW is needed before these parameters can be recommended as diagnostic or prognostic markers of dengue severity.

Limitations

This study was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings to other regions of Sudan. Participants were recruited by convenience rather than random sampling, which may have introduced selection bias and further limits generalizability of the findings. The comparative analytical cross-sectional design and single-timepoint sampling did not allow assessment of changes in platelet parameters throughout the course of illness or according to the day of illness, and platelet count and PDW were measured concurrently with dengue status rather than before disease onset, so a causal or temporal interpretation of the association is not possible.

Conflict of Interest

No conflict of interest.

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