

Assessment of Platelet Count and Platelet Indices among Sudanese Patients with Dengue Fever in Khartoum State

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ABSTRACT

Background: Dengue fever is a viral infection transmitted primarily by the *Aedes aegypti* mosquito and poses a major public health concern in tropical and subtropical regions. One of the key hallmarks of dengue infection, particularly in severe cases, is thrombocytopenia, or a significant reduction in platelet count. Understanding the alterations in platelet counts and indices among dengue patients is important for the early detection of severe disease forms and for improving clinical outcomes. This study aims to assess platelet counts and platelet indices (MPV, PDW, and PCT) among patients with dengue fever. **Material and methods:** This is a case-control study conducted in Khartoum state -Sudan during the period from September 2024 to October 2024, involving 100 participants. 50 samples from patients with dengue fever as cases and 50 healthy individuals as controls. Blood samples were analyzed by Mindray hematology analyzers (BC2800). The ethical committee of Gharb EL-Niel approved the research, and informed consent was obtained from all participants. The statistical analyses were performed using SPSS, t-test, and Pearson correlation. **Result:** The comparison of platelet counts and platelet indices between dengue cases and healthy controls revealed significant differences. The mean $\pm$ SD of platelet count in dengue cases was $197.5 \pm 22.6 \times 10^9/L$, significantly lower than the control group's count of $269.2 \pm 38.4 \times 10^9/L$ ($p < 0.05$). Mean platelet volume (MPV) was also reduced in dengue patients, with a mean of 8.4 ± 0.8 fL compared to 9.8 ± 0.4 fL in controls ($p < 0.05$). Platelet distribution width (PDW) showed similar trends, with dengue cases averaging 13.4 ± 0.7 fL versus 15.2 ± 1.2 fL in controls ($p = 0.001$). Lastly, plateletcrit (PCT) was also significantly lower in dengue cases (0.17 ± 0.04) compared to controls (0.21 ± 0.05 , $p = 0.001$). These findings suggest that dengue fever substantially impacts platelet counts and indices. **Conclusions:** The results of this study show that dengue fever has a considerable impact on platelet counts and indices. Compared to healthy controls, dengue patients had significantly lower platelet counts, mean platelet volume (MPV), and platelet distribution width (PDW). These data show how much dengue fever inhibits platelet production and function, highlighting the necessity of monitoring these indicators for early detection, severity evaluation, and clinical care of affected individuals.

Keywords Dengue Fever, Platelet Count, Platelet Indices, Sudanese Patients, Thrombocytopenia

INTRODUCTION

Dengue fever is a viral infection transmitted primarily by the *Aedes aegypti* mosquito and poses a major public health concern in tropical and subtropical regions [1]. With an estimated 390 million infections annually worldwide, dengue has a significant burden on healthcare systems, especially in endemic areas [2]. The disease manifests in a range of symptoms, from mild flu-like symptoms to severe complications such as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS), which can be life-threatening [3]. One of the key hallmarks of dengue infection, particularly in severe cases, is thrombocytopenia, or a significant reduction in platelet count [4]. Platelet indices such as mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) have been increasingly studied as potential markers for dengue severity, as they provide insight into platelet production and destruction dynamics during the infection [5]. Monitoring platelet counts and indices in dengue patients is essential for understanding the progression of the disease and guiding clinical decisions [6]. A decline in platelet count is frequently observed in patients with DHF and DSS, leading to bleeding complications and poor outcomes if not properly managed [7]. Meanwhile, alterations in platelet indices have been linked to disease severity and may offer valuable prognostic information [8]. This has prompted researchers to investigate the role of platelet counts

and indices as reliable predictors of dengue severity and to explore their utility in the early diagnosis and management of the disease [9]. Dengue fever is a mosquito-borne viral disease caused by the dengue virus (DENV), which belongs to the Flavivirus genus. There are four closely related but distinct serotypes of the virus (DENV-1, DENV-2, DENV-3, and DENV-4). Infection with one serotype provides lifelong immunity to that specific strain but not to others, which means a person can be infected multiple times. Dengue is primarily transmitted through the bites of *Aedes* mosquitoes, particularly *Aedes aegypti* and *Aedes albopictus*, which are most active during daylight hours. Dengue fever can range from mild illness to severe disease, and in some cases, it can progress to dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS), both of which are life-threatening [10]. The symptoms of dengue fever typically begin 4 to 10 days after being bitten by an infected mosquito. The most common symptoms include high fever, severe headache, pain behind the eyes (retro-orbital pain), muscle and joint pain (often referred to as "breakbone fever"), nausea, vomiting, swollen glands, and rash. In severe cases, the disease can progress to dengue hemorrhagic fever, characterized by bleeding, blood plasma leakage, and low platelet counts. Dengue shock syndrome, a more severe form, leads to dangerously low blood pressure. Symptoms usually last for 2 to 7 days, but recovery from

severe forms requires prompt medical attention [11]. Several risk factors increase the likelihood of contracting and experiencing severe dengue. Living or traveling in tropical and subtropical areas, especially in regions where dengue is endemic (Southeast Asia, Latin America, and the Pacific Islands), significantly raises the risk. Repeated exposure to *Aedes* mosquitoes is another key risk factor, as is having had a prior dengue infection, which raises the risk of developing severe dengue if infected with a different serotype. Other risk factors for severe dengue include age (young children and older adults are more vulnerable), weakened immune systems, and coexisting medical conditions such as diabetes or asthma [12]. Dengue fever is a global public health concern, especially in tropical and subtropical climates. The World Health Organization (WHO) estimates that there are around 390 million dengue infections annually, with approximately 96 million showing clinical symptoms. Dengue is endemic in over 100 countries, with the highest prevalence in Southeast Asia, the Americas, and the Western Pacific. Urbanization, climate change, and increased global travel have contributed to the expanding reach of dengue. In recent decades, there has been a dramatic increase in dengue cases, with some countries experiencing explosive outbreaks. Seasonal variation, typically during rainy seasons, can also affect the frequency and severity of outbreaks [13]. Dengue fever primarily affects the immune system and vascular structures. Once the virus enters the bloodstream through a mosquito bite, it targets and infects immune cells, such as macrophages and dendritic cells. The body's immune response produces a high viral load and releases various cytokines and chemokines, leading to systemic inflammation. In severe dengue, capillary permeability increases, allowing fluid to leak from blood vessels into surrounding tissues, leading to hypovolemia (decreased blood volume) and shock. Platelet destruction and impaired blood clotting mechanisms result in the hemorrhagic manifestations of the disease. The pathophysiology is still not completely understood, particularly why some individuals develop severe disease, but prior immunity to a different serotype is believed to play a key role [14]. The diagnosis of dengue fever is based on clinical symptoms and confirmed by laboratory tests. In areas where dengue is endemic, a presumptive diagnosis can be made based on the presence of classic symptoms during the early febrile phase. However, confirmatory diagnostic tests include detection of the virus, viral RNA, or specific antibodies. Tests such as polymerase chain reaction (PCR) and enzyme-linked immunosorbent assay (ELISA) can identify dengue early in the course of the disease. A complete blood count is also commonly used to monitor changes in white blood cells and platelets, which typically decrease as the disease progresses. Early and accurate diagnosis is crucial, especially in severe cases, to manage the condition appropriately [15]. There is no specific antiviral treatment for dengue fever, so management focuses on supportive care. Mild cases are managed with rest, hydration, and pain relief (usually with acetaminophen, as nonsteroidal anti-inflammatory drugs like aspirin are avoided due to the risk of bleeding). Patients with more severe forms, such as dengue hemorrhagic fever or dengue shock syndrome, may require hospitalization for intravenous fluid replacement and careful monitoring of vital signs, blood pressure, and blood clotting status. In severe cases, blood transfusions and intensive care support may be necessary. Prevention through mosquito control measures, such as eliminating breeding sites, using insect repellent, and employing mosquito nets, is crucial. The WHO also recommends the use of the dengue vaccine, Dengvaxia, for individuals in endemic areas who have already been exposed to the virus [16].

Platelet and Dengue Fever

Platelets, also known as thrombocytes, are small blood cells that play a critical role in blood clotting and stopping bleeding. In dengue fever, a marked drop in platelet count (thrombocytopenia) is one of the hallmark complications, particularly in severe forms of the disease like dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). Thrombocytopenia in dengue is the result of multiple factors, including the destruction of platelets, impaired production in the bone marrow, and increased consumption in the body's immune response. Dengue virus directly affects the bone marrow, which is responsible for producing blood components, including platelets. The virus's attack on the bone marrow reduces platelet production, while the body's immune response further damages platelets, leading to their destruction. Additionally, the virus and immune reactions cause the lining of blood vessels to become more permeable, allowing plasma to leak into surrounding tissues [17]. A case-control study by Wiwanitkit [18] investigated the mean platelet volume (MPV) among dengue hemorrhagic fever patients, with a sample size of 64 patients. The study reported a significant reduction in MPV in dengue patients (mean $\pm$ SD: 7.8 ± 1.4 fL) compared to the control group, indicating the utility of MPV as a marker for early diagnosis and monitoring of dengue hemorrhagic fever [18]. Bashir et al. [19] conducted a case-control study on 157 patients in Sudan to explore platelet indices in dengue infections. They found that mean platelet counts were significantly lower in dengue patients with severe thrombocytopenia (mean $\pm$ SD: $56.3 \pm 15.6 \times 10^9/L$) compared to non-severe cases. Platelet distribution width (PDW) and MPV were also elevated in severe cases [20]. Sharma and Yadav [20] analyzed platelet volume indices in 150 dengue patients. They found a significant decrease in mean platelet counts (mean $\pm$ SD: $72.4 \pm 25.8 \times 10^9/L$) and an increase in MPV (mean $\pm$ SD: 10.6 ± 1.7 fL) in patients with severe forms of dengue compared to those with milder infections. A systematic review and meta-analysis conducted in 2021 included 42 case-control studies across various countries. It analyzed the coagulation abnormalities in dengue fever, revealing a pooled mean platelet count in thrombocytopenic dengue patients of $89.7 \times 10^9/L$ with significant variability. Subgroup analyses confirmed higher rates of thrombocytopenia in severe cases [21]. Understanding the alterations in platelet counts and indices among dengue patients is important for early detection of severe disease forms and for improving clinical outcomes. Platelet indices, which reflect platelet size and variability, may serve as non-invasive and cost-effective biomarkers for assessing disease progression and the likelihood of complications [10]. This study aims to contribute to the growing body of evidence on the role of platelet counts and indices in dengue and their potential use in clinical settings for better disease management.

MATERIAL AND Method

This study is a case-control study conducted in Khartoum state - Sudan during the period from September 2024 to October 2024. The study population consisted of patients diagnosed with dengue fever. A total of 50 samples were from patients with dengue fever cases and 50 control samples were from healthy individuals. Patients diagnosed with dengue fever based on clinical symptoms and confirmed laboratory results, aged 18 and above were included in this study. Patients with coexisting conditions that can independently affect platelet counts or function, such as hematological disorders, chronic liver diseases, recent blood transfusion, or patients on medications

known to alter platelet function (e.g., antiplatelet or anticoagulant therapy) were excluded from this study. Data collection involved gathering both clinical and laboratory information from each participant. Clinical data such as age, sex, medical history, and symptoms were obtained through structured interview questionnaires and medical records.

Sample Collection

Blood samples were collected from each participant in the study. Venous blood was drawn in EDTA using sterile techniques, and samples were processed in an accredited laboratory. A portion of the blood was used for routine complete blood count (CBC) analysis, including platelet count and platelet indices (MPV, PDW, and PCT), performed using an automated hematology analyzer.

Platelet Count and Indices by Mindray Analyzer

Mindray hematology analyzers (BC2800) use the impedance method in conjunction with light scattering and fluorescence technologies to perform platelet counting and calculate platelet indices, including mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT). The principle behind the impedance method is based on the detection of changes in electrical resistance as blood cells pass through an aperture [22].

Procedure

The process begins with the preparation of an anticoagulated blood sample, which is loaded into the analyzer. The analyzer mixes the sample to ensure an even distribution of cells, preparing it for analysis. The cell counting and sizing phase involves three technologies. First, the impedance method is employed, where the blood sample flows through an aperture while an electric current is continuously monitored. Each cell's passage momentarily disrupts the current, generating a pulse; the pulse height and width indicate the cell's volume and allow for platelet counting. Following this, light scattering technology is used; as cells pass through a laser beam, they scatter light at specific angles based on their size and structure, which distinguishes platelets from other cell types. Finally, fluorescence detection may be used, where specific dyes stain cellular nucleic acids, helping differentiate platelets from other cells or fragments, particularly useful in cases of immature or fragmented platelets.

Data Analysis

Data was analyzed by using the SPSS computer program, the t-test was used to compare the mean and STD, p-value of less than 0.05 is considered significant.

Ethical Consideration

The research was approved by the ethical committee of Gharb EL-Niel, and informed consent was obtained from all participants.

Results

Demographic Data

This is a case-control study involving 50 patients with dengue fever and 50 healthy controls, the age of dengue cases ranged from 20 to 65 years, with a mean $\pm$ SD of 30 ± 12 years, while the control group had an age range of 18 to 70 years, with a mean age of 35 ± 15 years.

The duration of dengue fever among the cases ranged from 3 to 14 days, with a mean duration of 7 ± 2.5 days. Regarding gender distribution, 60% of the cases were male (30 males, 20 females), and the control group had 55% males (28 males, 22 females). Table 1.

Platelet Count and Indices in Dengue Cases and Healthy Controls

The comparison of platelet counts and platelet indices between dengue cases and healthy controls revealed significant differences. The mean platelet count in dengue cases was $196.4 \pm 23.9 \times 10^9/L$, significantly lower than the control group's count of $269.2 \pm 38.4 \times 10^9/L$ ($p < .001$). Platelet volume (MPV) was also reduced in dengue patients, with a mean of 8.3 ± 0.8 fL compared to 9.8 ± 0.4 fL in controls ($p = 0.00$).

Platelet distribution width (PDW) showed similar trends, with dengue cases averaging 13.4 ± 0.7 fL versus 15.2 ± 1.2 fL in controls ($p = 0.00$). Lastly, plateletcrit (PCT) was also significantly lower in dengue cases (0.17 ± 0.04) compared to controls (0.21 ± 0.05 , $p = 0.05$). These findings suggest that dengue fever substantially impacts platelet counts and indices, with notable reductions across all measured parameters. As seen in table 2.

Correlation Between Platelet Parameters, Age, and Duration of Disease

The correlation analysis between platelet counts and platelet indices with age and duration of disease in dengue cases showed varied associations. Platelet count exhibited a weak negative correlation with age ($R = -0.11$, $p > 0.05$) and a stronger negative correlation with the duration of disease ($R = -0.20$, $p > 0.05$), though neither was statistically significant. Mean platelet volume (MPV) had a moderate negative correlation with age ($R = -0.27$, $p > 0.05$) but showed almost no correlation with disease duration ($R = -0.02$, $p > 0.05$). PDW had a slight negative correlation with age ($R = -0.18$, $p > 0.05$) and a weak positive correlation with disease duration ($R = 0.09$, $p > 0.05$). Interestingly, PCT displayed moderate positive correlations with both age ($R = 0.31$, $p > 0.05$) and duration of disease ($R = 0.32$, $p > 0.05$), though these correlations did not reach statistical significance. This suggests that while there may be trends in platelet parameters relative to age and disease duration, the relationships are not strong or significant in this study. Table 3.

Table 1: Descriptive Statistics of the Study Group

	N	Minimum	Maximum	Mean $\pm$ SD
Age in Cases	50	20	65	30 ± 12
Age in Control	50	18	70	35 ± 15
Duration of Dengue Fever	50	3	14	7 ± 2.5

Table 2: Mean and SD of Platelet Count and Platelet Indices among the Study Group.

	Dengue Cases	Healthy Controls	P-Value
Platelet Count	196.4 ± 23.9	269.2 ± 38.4	0.01
MPV	8.3 ± 0.8	9.8 ± 0.3	0.01
PDW	13.5 ± 0.7	15.2 ± 1.1	0.01
PCT	0.17 ± 0.04	0.22 ± 0.04	0.01

Table 3: Correlation between Platelet Count and Platelet Indices with Age and Duration of Disease.

	Age	Duration of Disease
Platelet Count	(R=-0.11) p >0.05	(R=-0.20) p >0.05
MPV	(R=-0.27) p >0.05	(R=-0.02) p >0.05
PDW	(R=-0.18) p >0.05	(R=0.09) p >0.05
PCT	(R=0.31) p >0.05	(R=0.32) p >0.05

Discussion

When comparing the current study's results with previous case-control studies on dengue fever and platelet indices, both similarities and differences are evident. In this study, the mean platelet count in dengue cases was $197.5 \pm 22.6 \times 10^9/L$, significantly lower than that of the controls ($269.2 \pm 38.4 \times 10^9/L$), reflecting the typical thrombocytopenia observed in dengue patients. However, this value is higher than the platelet counts observed in studies by Bashir et al. [19] and Sharma and Yadav [20], where platelet counts were notably lower in severe dengue cases, at $56.3 \pm 15.6 \times 10^9/L$ and $72.4 \pm 25.8 \times 10^9/L$, respectively (19,20). The higher platelet counts in the current study could indicate a less severe manifestation of dengue among the cases, or it may reflect differences in the study populations, with other studies possibly focusing on more critically ill patients. Similarly, Wiwanitkit [18] found significantly lower MPV (7.8 ± 1.4 fL) in severe dengue cases compared to controls, which aligns closely with this study's findings of reduced MPV. However, Sharma and Yadav [20] reported a much higher MPV (10.6 ± 1.7 fL) in their severe dengue patients, which may suggest that MPV could increase in very advanced stages of the disease, as elevated MPV is often associated with greater platelet destruction and turnover.

In contrast, the current study's PDW values (13.4 ± 0.7 fL in cases) were lower than those reported in previous studies where severe dengue patients had higher PDW, reflecting increased platelet size variability [20]. The systematic review from 2021, which pooled 42 case-control studies, highlighted wide variability in platelet counts and indices across different geographic regions, reinforcing that platelet behavior in dengue can differ depending on severity, population demographics, and local disease characteristics. Overall, while the current study's results are consistent with other research showing reduced platelet counts and altered platelet indices in dengue, the variations across studies highlight the importance of context, disease severity, and population in interpreting these markers [21].

Conclusion

The results of this study show that dengue fever has a considerable impact on platelet counts and indices. Compared to healthy controls, dengue patients had significantly lower platelet counts, mean platelet

volume (MPV), and platelet distribution width (PDW). These data show how much dengue fever inhibits platelet production and function, highlighting the necessity of monitoring these indicators for early detection, severity evaluation, and clinical care of affected individuals.

Study Limitations

This study was limited by a small sample size of 100 participants and a brief data collection period (September to October 2024), which may not adequately reflect broader population trends or account for seasonal fluctuations in dengue transmission.

Additionally, the absence of patient stratification by dengue virus serotype and clinical severity (e.g., dengue fever, dengue hemorrhagic fever, or dengue shock syndrome) restricts the interpretation of platelet parameter variations. The lack of longitudinal follow-up further limits insights into the progression and recovery phases of thrombocytopenia.

Recommendations

To enhance the generalizability and clinical relevance of future research, studies should:

- Enroll larger, more diverse populations across multiple dengue transmission seasons to capture temporal and epidemiological variability.
- Incorporate dengue virus serotyping and classify cases by disease severity to better understand differential impacts on platelet indices.
- Conduct serial assessments of platelet count, mean platelet volume (MPV), and platelet distribution width (PDW) throughout the course of illness and recovery to elucidate dynamic changes in platelet kinetics.
- Evaluate the prognostic value of platelet indices in predicting severe dengue outcomes and explore regional differences in hematological responses.
- Integrate platelet monitoring into routine clinical protocols to support early identification and management of patients at risk for complications.

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