World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

Check for updates

ALTERATIONS IN PLATELET AND LEUCOCYTE PROFILES AS INDICATORS OF HYPERINFLAMMATION AND THROMBO-INFLAMMATORY STATE IN SICKLE CELL DISEASE

Vivian Akudo Maduka [1], Benjamin Onyema Eledo [1], Richard Ikechukwu Eze [2] and Emmanuel Chinedu Onuoha [1,*]

[1] Department of Haematology and Transfusion Science, Faculty of Medical Laboratory Science, Federal University, Otuoke, Bayelsa, Nigeria.
[2] Medical Laboratory Science department, Madonna University, Nigeria, Elele, River State, Nigeria.
* Corresponding Author

ORCID Details
Emmanuel Chinedu Onuoha: https://orcid.org/ 0000-0002-4446-7900

World Journal of Advanced Research and Reviews, 2026, 31(03), 415–423

Publication history: Received on 25 July 2026; revised on 02 September 2026; accepted on 08 September 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.31.3.2271

ABSTRACT

Background: Sickle cell disease (SCD) is a chronic inflammatory haemoglobin disorder characterized by vaso-occlusion, endothelial dysfunction, and abnormal activation of immune and haemostatic pathways. Leucocytes and platelets contribute significantly to the thrombo-inflammatory processes involved in disease progression and vaso-occlusive crisis. This study evaluated alterations in leucocyte and platelet profiles among patients with SCD during crisis and steady state compared with healthy controls in Bayelsa State, Nigeria.

Materials and Methods: A comparative cross-sectional study was conducted among One hundred and eighty (180) participants comprising Sixty-seven (67) SCD patients in vaso-occlusive crisis, Fifty-three (53) SCD patients in steady state, and Sixty (60) apparently healthy controls. Venous blood samples were collected into EDTA bottles and analysed using an automated haematology analyser. Leucocyte parameters (total white blood cell count, neutrophils, lymphocytes, monocytes, eosinophils, and basophils) and platelet parameters (platelet count and mean platelet volume) were evaluated. Data were analysed using appropriate statistical methods, and significance was set at $p<0.05$.

Results: The findings revealed significant alterations in leucocyte and platelet profiles among SCD patients compared with controls. Total white blood cell count and neutrophil levels were significantly elevated among patients in vaso-occlusive crisis, indicating enhanced inflammatory activation. Platelet count was significantly increased among SCD patients, suggesting increased platelet activation and thrombo-inflammatory activity. The neutrophil-to-lymphocyte ratio was also higher among crisis patients compared with steady-state patients and controls, reflecting increased systemic inflammatory burden.

Conclusion and Recommendation: Leucocyte and platelet abnormalities are important features of SCD and may serve as indicators of inflammatory activation and thrombo-inflammatory status. Routine assessment of leucocyte and

platelet-derived indices may improve monitoring and management of SCD patients, particularly in resource-limited settings. Further longitudinal studies are recommended to determine their predictive value for disease severity and vaso-occlusive complications.

Keywords: Sickle cell disease; Leucocytes; Platelets; Vaso-occlusive crisis; Thrombo-inflammation.

1 INTRODUCTION

One of the most prevalent genetic hemoglobin diseases in the world, sickle cell disease (SCD) continues to be a significant public health concern, especially in sub-Saharan Africa, where the disease burden is highest. A single nucleotide mutation in the β-globin gene causes aberrant hemoglobin S (HbS), which undergoes polymerization in deoxygenated environments and leads in erythrocyte deformation, hemolysis, and repeated vaso-occlusive episodes[1].SCD is a complex multisystem inflammatory disease with endothelial dysfunction, coagulation abnormalities, chronic immune activation, and cellular interactions involving leucocytes and platelets, despite being traditionally thought of as a disorder of red blood cells[2].

The classic clinical sign of sickle cell disease (SCD), vaso-occlusive crisis (VOC), is caused by intricate interactions between sickled erythrocytes, vascular endothelium, inflammatory cells, and platelets. Adhesion molecules and inflammatory mediators released by activated endothelial cells during VOC encourage the recruitment and activation of leucocytes, especially neutrophils, which cause tissue ischemia and vascular blockage[3]. The development of inflammation and vascular damage in SCD has been linked to increased leucocyte adhesion, neutrophil activation, and the creation of neutrophil extracellular traps[4].

Beyond their typical immune defense roles, leucocytes are crucial to the pathogenesis of sickle cell disease. In people with SCD[5-6], persistent elevation of neutrophilia and total white blood cell count has been linked to acute chest syndrome, numerous vaso-occlusive episodes, higher disease severity, and early mortality. By releasing cytokines, reactive oxygen species, proteases, and adhesion molecules, activated neutrophils promote endothelial activation and microvascular occlusion[7]. On the other hand, lymphocytes are involved in the regulation of adaptive immunity, and changes in the distribution of lymphocytes may indicate shifts in the balance of the immune system in both steady state and acute crises.

It is becoming more well acknowledged that platelets, or thrombocytes, play a significant role in the thrombo-inflammatory process in sickle cell disease. While normal hemostasis depends on platelet activation, excessive activation in sickle cell disease (SCD) increases platelet adhesion, aggregation, and contact with erythrocytes, leucocytes, and endothelial cells, which increases vascular inflammation and thrombosis[8]. Due to prolonged hemolysis, decreased platelet survival, and compensatory megakaryopoiesis brought on by splenic dysfunction, patients with sickle cell disease (SCD) have been found to have elevated platelet numbers and enhanced platelet activation markers[9].

An effective biomarker of platelet reactivity and inflammatory condition is mean platelet volume (MPV), which measures platelet size and activity. Bigger platelets are more thrombogenic because they have higher granule and physiologically active material concentrations. Therefore, variations in MPV and other platelet indices may shed light on thrombotic risk and platelet activation in SCD patients[10]. An essential part of thrombo-inflammation is the interaction between active platelets and leucocytes, where platelet-leucocyte aggregates encourage vaso-occlusion, inflammatory signaling, and endothelial damage[11].

The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR), two composite inflammatory indices derived from standard complete blood count values, have recently drawn interest as reasonably priced biomarkers for evaluating systemic inflammation and disease activity. These indicators provide more information than just cell counts by combining innate immune activity and platelet responses. In a number of chronic inflammatory illnesses, including SCD, elevated NLR has been linked to poor outcomes and inflammatory load[12].

Regular laboratory monitoring of leucocyte and platelet abnormalities is still crucial despite advances in our understanding of the molecular causes of sickle cell disease (SCD), particularly in settings with low resources where sophisticated inflammatory and molecular indicators may not be easily accessible. The comparative patterns of leucocyte and thrombocyte profiles among patients in vaso-occlusive crisis, those in steady state, and healthy individuals are still poorly understood in Nigeria, where sickle cell disease (SCD) is a major cause of morbidity and mortality.

The present study therefore evaluates alterations in platelet and leucocyte profiles as indicators of hyperinflammation and thrombo-inflammatory states among patients with SCD during crisis and steady state compared with healthy controls in Bayelsa State, Nigeria. Understanding these haematological alterations may provide valuable insight into

disease activity, inflammatory status, and potential biomarkers for improved clinical monitoring and management of SCD patients.

2 MATERIAL AND METHODS

2.1 Study area

Communities in Bayelsa State with coordinates of 4.8678°N, 5.8987°E were used for this investigation. With a total land area of roughly 10,773 km2 (4,159 sq mi), Bayelsa State is a state in Southern Nigeria located in the central Niger Delta Region between Delta State and River State. Yenagoa, its capital, is located at 4°55'29"N, 6°15'51"E. English is the official language, however Ijaw is the primary language spoken. With an estimated population of 1,704,515 based on the 2006 census, the state was formed in 1996 from a portion of Rivers State. Yenagoa serves as the capital of Bayelsa State, which contains eight local government areas. The research was carried out between June 2025 and February 2026.

2.2 Study population

The study population was made up of 180 participants within the age of 5 – 40 years for both male and female subjects which comprises 67 sickle cell subjects in crisis and 53 stable sickle cell subjects who were either admitted or attending their clinic days at Federal Medical Centre Yenagoa, Kaiama General Hospital, Diete Koki Memorial Hospital, and Niger Delta University Teaching Hospital Okolobiri, during the study period. 60 AA subjects were used as controls.

2.3 Selection Criteria

This was done using questionnaire and consent form.

2.3.1 Inclusion Criteria

Participants included in the study were confirmed sickle cell patients and AA controls, both male and female, aged 5 to 40years, as verified through laboratory screening.

2.3.2 Exclusion Criteria

The study excludes:

- Sickle cell patients and control subjects with co-morbidities, including hypertension, diabetes mellitus, chronic kidney disease, and known coagulation disorders.
- Individuals who refused to provide informed consent.

2.4 Sample size

The prevalence of sickle cell disease in the South-south region of Nigeria was reported to be 3.7% [13]. The sample size was calculated using the formula proposed by Oladimeji[14]

$$N= Z^2PQ/d^2$$

Where:

- N = required sample size
- Z^2 = critical value at 95% confidence level = 1.96
- P = estimated prevalence = 0.037
- Q = 1 − P = 0.963
- d^2 = absolute sampling error tolerated = 0.05

The calculations were done as follows:

- $Z^2 = 1.96^2 = 3.8416$
- P =3.7% =0.037
- Q =1-0.037 =0.963
- d^2=5% =0.05 =0.0025
- N =3.8416 X 0.037 X0.963 / 0.0025 =54.7
- The minimum sample size for this study was 55.

Using 10% attrition rate, the sample size increased to 60 participants each.

2.5 Collection of sample

Aseptically, using a 5 mL syringe, 3 mL of venous blood was collected and dispensed into an EDTA (Ethylenediaminetetraacetic Acid) tube for haematological analysis. Analysis was done within 2 hrs of sample collection using whole blood to avoid haemolysis and platelet breakdown.

2.6 Ethical approval

Ethical clearance was obtained from Bayelsa State Ministry of Health which control activities of all Bayelsa State health facility and from the Federal Medical Center Yenagoa research committee. Respect to participants' rights was observe including the right to refuse participation with explanation through participant's information form.

2.7 Statistical analysis

Data were entered, cleaned, and analyzed using the Statistical Package for the Social Sciences (SPSS) version 27.0. Continuous data were expressed as mean ± standard deviation (SD), and a p-value of less than 0.05 was considered statistically significant. Differences between crisis, stable and control groups were analyzed using the independent samples t-test. Comparisons among crisis, stable and control groups were carried out using one-way analysis of variance (ANOVA).

2.8 Method ofLaboratory analysis

Leucocytes and thrombocytes were done by 5-part Haematology Analyzer (wincom HA8520) while the electrophoresis were performed using the Model Techmel and Techmel 300 (USA) systems.

3 RESULTS

Table .1 presents a comparison of leucocytes and thrombocytes among control, stable sickle cell disease (SCD), and crisis SCD groups using one-way ANOVA with Tukey's post hoc test. Total white blood cell count (TWBC) showed a statistically significant difference ($F = 138.746$, $p < 0.001$), with the crisis group having the highest value (13.33 ± 3.52), followed by the stable group (7.21 ± 1.90) and control group (6.24 ± 1.38). Neutrophil percentage differed significantly among groups ($F = 31.210$, $p < 0.001$), with the crisis group (63.89 ± 7.98) higher than both stable and control groups. Lymphocyte percentage also showed a significant difference ($F = 34.624$, $p < 0.001$), with higher values in the stable (38.98 ± 5.58) and control (36.45 ± 3.76) groups compared to the crisis group (30.10 ± 7.34). Monocyte percentage showed a statistically significant difference ($F = 3.158$, $p = 0.045$), with higher values in the stable group (3.48 ± 1.76) compared to the crisis group (2.79 ± 1.31), while the control group (3.15 ± 1.14) showed an intermediate value. Eosinophil percentage did not differ significantly across the groups ($F = 2.124$, $p = 0.123$), with comparable values among control, stable, and crisis groups. Basophil percentage also showed no statistically significant difference ($F = 1.846$, $p = 0.161$) across the groups. Platelet count differed significantly among groups ($F = 64.248$, $p < 0.001$), with the highest values observed in the crisis group (420.96 ± 81.77), followed by the stable group (357.26 ± 89.96), and the lowest in the control group (260.42 ± 51.92). Mean platelet volume (MPV) also showed a significant difference ($F = 6.263$, $p = 0.002$), with the stable group showing the highest value (11.41 ± 1.21) compared to control (10.90 ± 0.87) and crisis (10.87 ± 0.36) groups. Neutrophil ratio showed a statistically significant difference ($F = 20.915$, $p < 0.001$), with the crisis group having the highest value (4.78 ± 3.06), followed by control (2.90 ± 1.68) and stable groups (2.38 ± 0.60). Lymphocyte ratio did not differ significantly among the groups ($F = 1.999$, $p = 0.139$), with comparable values across all groups. Neutrophil-to-lymphocyte ratio (N/L ratio) showed a significant difference ($F = 45.707$, $p < 0.001$), with progressive increases from control (1.30 ± 0.09) to stable (1.69 ± 0.45) and crisis (2.38 ± 0.96) groups.

Table 1: ANOVA comparison of some haematological parameters amongst crisis, stable and control subjects.

Parameter	Control	Stable	Crisis	F	p-value
TWBC($\times10^9$/L)	6.24 ± 1.38	7.21 ± 1.90[a]	13.33 ± 3.52[ab]	138.746	0.000
Neut(%)	57.75 ± 3.93	55.17 ± 5.14[a]	63.89 ± 7.98[ab]	31.210	0.000
Lym(%)	36.45 ± 3.76	38.98 ± 5.58[ab]	30.10 ± 7.34[a]	34.624	0.000
Mon(%)	3.15 ± 1.14	3.48 ± 1.76	2.79 ± 1.31[a]	3.158	0.045
Eos(%)	2.12 ± 0.81	2.04 ± 0.95[a]	2.42 ± 1.22[a]	2.124	0.123
Bas(%)	0.53 ± 0.19[a]	0.59 ± 0.19	0.56 ± 0.19	1.846	0.161
Platelet	260.42 ± 51.92	357.26 ± 89.96[a]	420.96 ± 81.77[ab]	64.248	0.000
MPV(fL)	10.90 ± 0.87	11.41 ± 1.21	10.87 ± 0.36	6.263	0.002
Neutrophil Ratio	2.90 ± 1.68	2.38 ± 0.60[a]	4.78 ± 3.06[ab]	20.915	0.000
Lymphocyte Ratio	2.47 ± 0.41	2.14 ± 0.51	2.29 ± 1.40	1.999	0.139
N/L Ratio	1.30 ± 0.09	1.69 ± 0.45[b]	2.38 ± 0.96[ab]	45.707	0.000

Footnote: Values are presented as mean ± standard deviation (SD). Statistical comparisons were performed among the various study groups, and a p-value < 0.05 was considered statistically significant. Different letters along the column indicate statistical variations according to Waller-Duncan test statistics; Abbreviations: PLT = Platelet count ($\times10^9$/L);MPV = Mean Platelet Volume (fL); WBC = White Blood Cell count ($\times10^9$/L);NEU = Neutrophils (%);LYM = Lymphocytes (%);MONO = Monocytes (%);EOSO = Eosinophils (%);BASO = Basophils (%).Keya=significantly different compared to control; b= significantly different compared to stable.

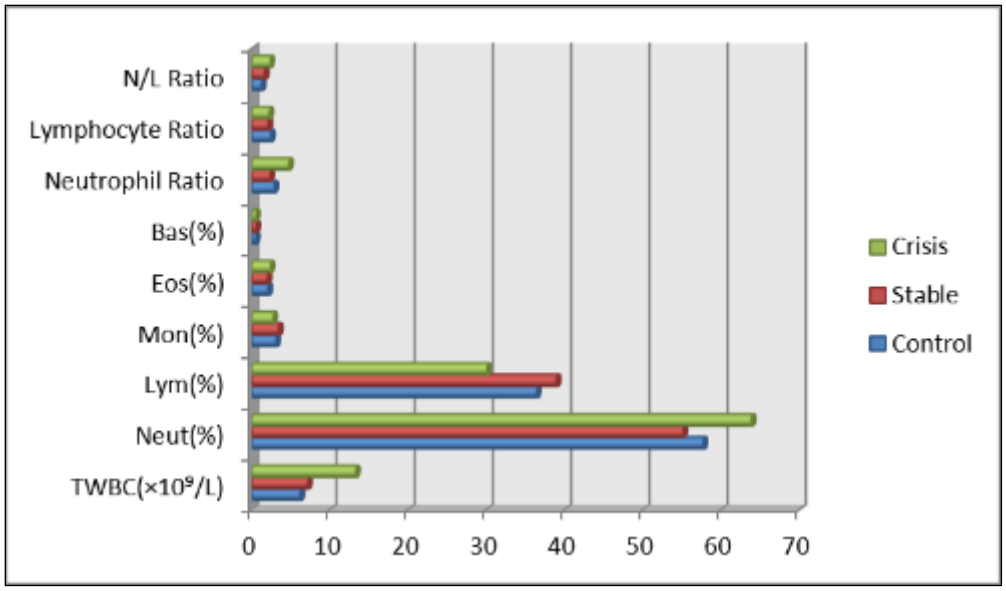

Figure 1: Comparative Leucocytes Profiles (Control, Stable, Crisis)

Figure 1 displays the distribution of leucocyte parameters across control, stable, and crisis groups. It highlights a marked rise intotal white blood cell count (TWBC)and neutrophil percentagein crisis patients compared to stable and controls, reflecting heightened inflammatory activation. Conversely**,** lymphocyte percentageis reduced in crisis, indicating immune imbalance. Ratios such as the neutrophil ratioandN/L ratio are significantly elevated in crisis, reinforcing their role as indicators of systemic inflammation in sickle cell disease.

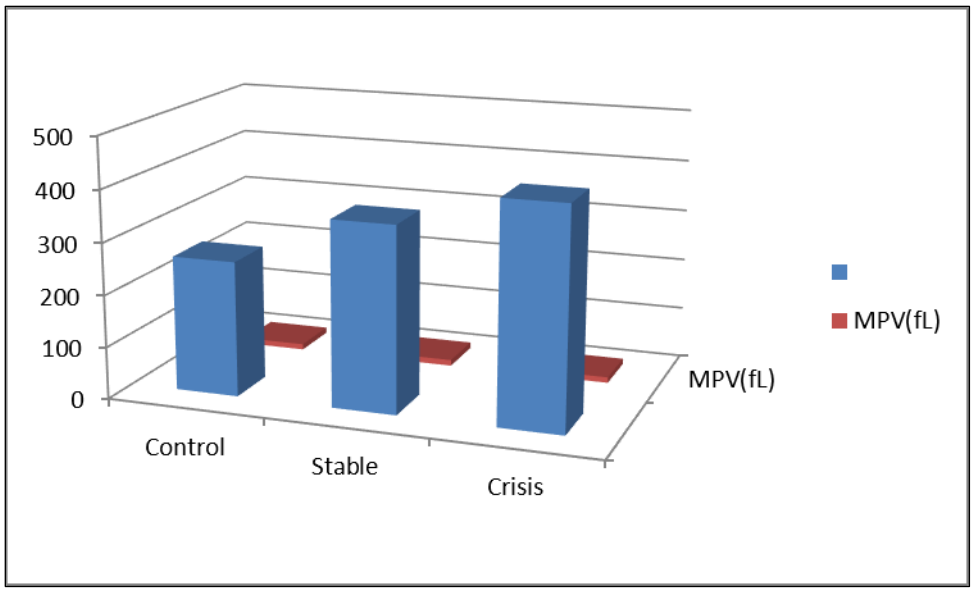

Figure 2: Platelet Count and Mean Platelet Volume (MPV) Across Groups

Figure 2 compares platelet indices among control, stable, and crisis subjects. Platelet count shows a progressive increase from control to stable and crisis, suggesting enhanced thrombopoiesis and platelet activation during vaso-occlusive episodes. Meanwhile, MPV peaks in the stable group, implying ongoing platelet reactivity even outside acute crises. These findings underscore the contribution of platelets to the thrombo-inflammatory state in sickle cell disease.

4 DISCUSSION

Erythrocytes, leucocytes, platelets, and vascular endothelial cells interact abnormally in sickle cell disease (SCD), a chronic inflammatory and thrombo-inflammatory illness that is becoming more widely acknowledged. The current investigation highlighted the role of immunological activation and platelet-mediated inflammation in SCD pathophysiology by showing notable changes in leucocyte and platelet profiles among SCD patients during vaso-occlusive crisis and steady state compared with healthy controls.The graphical representation of these parameters further reinforces the progressive inflammatory changes occurring from healthy controls to steady-state patients and reaching their peak during vaso-occlusive crisis, providing visual evidence of the dynamic nature of SCD-associated inflammation.

Increased inflammatory activation during vaso-occlusive events is shown by the substantial increase in total white blood cell count (TWBC) seen in crisis patients as compared to steady-state patients and healthy controls. Increased synthesis of inflammatory cytokines, endothelial activation, and immune cell migration into the bloodstream may all contribute to the rise in leucocyte counts during a crisis. Through enhanced adherence to vascular endothelium and interaction with sickled erythrocytes, activated leucocytes—neutrophils in particular—promote vaso-occlusion, impairing microvascular blood flow and causing tissue ischemia[2]. Leucocytosis is a significant marker linked to increased disease activity and consequences in SCD patients, according to similar findings published by other researchers[6].

The neutrophilia that was seen in crisis patients when compared to steady-state and control groups provides additional evidence that innate immune activation plays a part in acute SCD problems. By producing inflammatory mediators, reactive oxygen species, proteolytic enzymes, and neutrophil extracellular traps (NETs), which exacerbate endothelial damage and encourage coagulation activity, neutrophils contribute to vaso-occlusion. Thus, the higher neutrophil percentage in crisis patients in this study is in line with earlier findings that neutrophil activation is strongly linked to vaso-occlusive events and inflammatory burden in sickle cell disease[4].

In contrast, crisis patients had comparatively lower lymphocyte percentages than the other groups. Even while lymphocytes are necessary for adaptive immune responses, acute inflammatory circumstances can cause lymphocyte-mediated immune control to be suppressed or circulating lymphocytes to be redistributed to tissues. Thus, an imbalance between innate and adaptive immune responses during vaso-occlusive crises could be the cause of the observed decline. Similar changes in the distribution of lymphocytes have been reported in inflammatory conditions where the preponderance of neutrophils is indicative of acute inflammatory stress[12].

The current study also showed that SCD patients, especially those going through a crisis, had significantly higher platelet counts. Chronic hemolysis, inflammatory stimulation, and decreased splenic clearance of platelets because of functional asplenia are linked to increased platelet production and activation in SCD. By aggregating with leucocytes and sickled red blood cells, improving endothelial adhesion, and encouraging a prothrombotic state, activated platelets contribute to vaso-occlusion[8]. The study's noticeably higher platelet counts corroborate earlier findings that thrombocytosis is a prevalent haematological defect in SCD patients.

The substantial variation in mean platelet volume (MPV) between the study groups points to changed platelet activity in sickle cell disease (SCD). Larger platelets are typically thought to be more thrombogenic and metabolically active. MPV is a measure of platelet size and activation status. Increased MPV has been linked to increased inflammatory responses and platelet aggregation in a number of vascular disorders[10]. The idea that SCD is still a chronic inflammatory disease even during clinically stable times is supported by the higher MPV seen, especially in steady-state patients, which may suggest ongoing platelet activation even in the absence of acute vaso-occlusive episodes.

From healthy controls to steady-state and crisis patients, the neutrophil-to-lymphocyte ratio (NLR) showed a progressive increase, with crisis patients exhibiting the highest value. This result suggests that during vaso-occlusive events, the systemic inflammatory burden is elevated. Neutrophil-mediated innate immunological activation and lymphocyte-mediated immune control are two crucial aspects of inflammation that are combined by NLR. Therefore, its increase in SCD may offer a straightforward and reasonably priced marker for evaluating disease activity. Because NLR may be obtained from standard complete blood count values without incurring additional laboratory costs, prior research has demonstrated that it may be a valuable inflammatory biomarker in chronic inflammatory and haematological disorders[12].

The results of this investigation show that platelet and leucocyte abnormalities are interrelated parts of the thrombo-inflammatory cascade in sickle cell disease (SCD). Acute vaso-occlusion may be accompanied by enhanced immunological activation and platelet-mediated vascular inflammation, as seen by the elevated TWBC, neutrophils, platelet count, and NLR seen in crisis patients. These results provide credence to the growing theory that sickling alone is not the cause of SCD problems, but rather intricate interactions between inflammation, coagulation, and cellular adhesion[3].

These findings have therapeutic implications in that ordinary haematological parameters may offer useful information for tracking disease progress, especially in settings with limited resources when advanced inflammatory biomarkers and molecular investigations are not available. TWBC, platelet count, MPV, and NLR are examples of parameters that can be used as low-cost markers to identify patients who are more likely to experience vaso-occlusive problems.

5 CONCLUSION

In comparison to healthy individuals, this study shows notable changes in leucocyte and platelet profiles in sickle cell disease patients during vaso-occlusive crisis and steady state. Increased platelet count, neutrophil percentage, neutrophil-to-lymphocyte ratio, MPV, and total white blood cell count all point to increased thrombo-inflammatory and inflammatory activation in SCD. According to the results, routine haematological measures can be useful markers for tracking clinical development and evaluating disease activity. Leucocyte and platelet-derived indices may help identify patients at risk for vaso-occlusive problems earlier in the course of normal SCD therapy.

Significance of the study

The significance of the study lies in its ability to provide practical, affordable, and clinically relevant insights into sickle cell disease (SCD):

- It demonstrates that routine haematological parameters such as total white blood cell count, neutrophil percentage, platelet count, mean platelet volume, and neutrophil-to-lymphocyte ratio can serve as accessible biomarkers of inflammation and thrombo-inflammatory activity in SCD.
- The findings highlight the dynamic changes between steady state and vaso-occlusive crisis, offering clinicians a clearer picture of disease progression and systemic inflammatory burden.
- By emphasizing the role of platelet-leucocyte interactions in driving vaso-occlusion, the study expands understanding of SCD beyond being a red cell disorder to a complex inflammatory and vascular disease.
- Importantly, it provides local evidence from Nigeria, strengthening global literature while addressing gaps in resource-limited settings where advanced molecular markers are not readily available.
- In essence, the study is significant because it identifies low-cost, routine laboratory indices that can improve monitoring, risk assessment, and management of SCD patients, thereby enhancing patient care and outcomes.

Recommendations

- Routine monitoring of leucocyte and platelet parameters should be incorporated into the clinical management of patients with SCD, particularly during vaso-occlusive crises.
- Inflammatory indices derived from complete blood count parameters, such as neutrophil-to-lymphocyte ratio (NLR), should be considered as affordable biomarkers for assessing disease activity.
- Further longitudinal studies involving larger populations should be conducted to evaluate the predictive value of platelet and leucocyte indices for complications and mortality in SCD.
- Healthcare institutions in Nigeria should strengthen laboratory capacity for regular haematological monitoring of individuals living with SCD.
- Future studies should investigate the relationship between platelet-leucocyte interactions, inflammatory cytokines, and molecular markers of disease severity in SCD.

Contribution to knowledge

- This study provides local evidence demonstrating significant alterations in leucocyte and platelet profiles among SCD patients during crisis and steady state in Bayelsa State, Nigeria.
- The study highlights the importance of platelet and leucocyte interactions as indicators of the thrombo-inflammatory state associated with SCD progression.
- It establishes the potential usefulness of routine haematological indices, particularly NLR and platelet parameters, as affordable biomarkers for monitoring disease activity.
- The findings contribute to the growing understanding that SCD is not only a red cell disorder but also a complex inflammatory and vascular disease involving immune and haemostatic pathways.
- The generated dataset provides a foundation for future development of predictive models, including artificial intelligence-based approaches, for early identification of vaso-occlusive complications in SCD patients.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Acknowledgments

The authors sincerely acknowledge the Director of Scanex, Nikton Road, Yenagoa, Bayelsa State, Mr. Azubuike Ogechi, and the scientist at the facility, Mrs. Cynthia Onyegbula, for their valuable support and assistance throughout the study. The authors also appreciate Dr. Adaka Oluwayemisi and Mr. James Full James, both of the Federal University, Otuoke, Bayelsa State, for their contributions and support towards the successful completion of this work.

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical clearance was obtained from Bayelsa State Ministry of Health which control activities of all Bayelsa State health facility and from the Federal Medical Center Yenagoa research committee.

Statement of informed consent

Respect to participants' rights was observe including the right to refuse participation with explanation through participant's information form.

REFERENCES

[1] Kato, G. J., Piel, F. B., Reid, C. D., Gaston, M. H., Ohene-Frempong, K., Krishnamurti, L., Smith, W. R., Panepinto, J. A., Weatherall, D. J., Costa, F. F., & Vichinsky, E. P. (2018). Sickle cell disease. Nature Reviews Disease Primers, 4, 18010. https://doi.org/10.1038/nrdp.2018.10

[2] Sundd, P., Gladwin, M. T., & Novelli, E. M. (2019). Pathophysiology of sickle cell disease. Annual Review of Pathology: Mechanisms of Disease, 14, 263–292. https://doi.org/10.1146/annurev-pathmechdis-012418-012838

[3] Manwani, D., & Frenette, P. S. (2013). Vaso-occlusion in sickle cell disease: Pathophysiology and novel targeted therapies. Blood, 122(24), 3892–3898. https://doi.org/10.1182/blood-2013-05-498311

[4] Chen, G., Zhang, D., Fuchs, T. A., Manwani, D., Wagner, D. D., & Frenette, P. S. (2014). Heme-induced neutrophil extracellular traps contribute to the pathogenesis of sickle cell disease. Blood, 123(24), 3818–3827. https://doi.org/10.1182/blood-2013-10-529982

[5] Lubeck, D., Agodoa, I., Bhakta, N., Danese, M., Pappu, K., Howard, R., Gleeson, M., Halperin, M., & Lanzkron, S. (2019). Estimated life expectancy and income of patients with sickle cell disease compared with those without sickle cell disease. JAMA Network Open, 2(11), e1915374. https://doi.org/10.1001/jamanetworkopen.2019.15374

[6] Knisely, M. R., Tanabe, P. J., Walker, J. K. L., Yang, Q., & Shah, N. R. (2022). Severe persistent pain and inflammatory biomarkers in sickle cell disease: An exploratory study. Biological Research for Nursing, 24(1), 24–30. https://doi.org/10.1177/10998004211027220

[7] Taher, M., Aminondin, S., Nasir, N. A., Jasmadi, N. A., Nizam, N. I. N., Shahrul, I. S., Susanti, D., Khotib, J., Faiyazuddin, M., Widodo, R. T., & Haris, M. S. (2025). Sickle cell disease: Understanding pathophysiology, clinical features and advances in gene therapy approaches. Frontiers in Pharmacology, 16, 1630994. https://doi.org/10.3389/fphar.2025.1630994

[8] Hamali, H. A. (2023). Hypercoagulability in sickle cell disease: A thrombo-inflammatory mechanism. Hemoglobin, 47(6), 205–214. https://doi.org/10.1080/03630269.2023.2301026

[9] Vallelian, F., Schaer, D. J., Lechartier, B., Buehler, P. W., & Irwin, D. C. (2026). Sickle cell disease-associated pulmonary hypertension: An integrated framework linking pathologies, mechanisms, and clinical phenotypes. EBioMedicine, 128, 106288. https://doi.org/10.1016/j.ebiom.2026.106288

[10] Estevez, B., & Du, X. (2017). New concepts and mechanisms of platelet activation signaling. Physiology, 32(2), 162–177. https://doi.org/10.1152/physiol.00020.2016

[11] Morrell, C. N., Aggrey, A. A., Chapman, L. M., & Modjeski, K. L. (2014). Emerging roles for platelets as immune and inflammatory cells. Blood, 123(18), 2759–2767. https://doi.org/10.1182/blood-2013-11-462432

[12] Buonacera, A., Stancanelli, B., Colaci, M., & Malatino, L. (2022). Neutrophil to lymphocyte ratio: An emerging marker of the relationships between the immune system and diseases. International Journal of Molecular Sciences, 23(7), 3636. https://doi.org/10.3390/ijms23073636

[13] West, B. A., & Aitafo, J. E. (2023). Prevalence, pattern of disease and outcome of children with sickle cell disease admitted in a private health facility in Southern Nigeria. Asian Journal of Pediatric Research, 12(1), 17–27. https://doi.org/10.9734/ajpr/2023/v12i1230

[14] Bolarinwa, O. (2020). Sample size estimation for health and social science researchers: The principles and considerations for different study designs. Nigerian Postgraduate Medical Journal, 27, 67. https://doi.org/10.4103/npmj.npmj_19_20