https://doi.org/10.48047/AFJBS.6.15.2024.3086-3090

African Journal of Biological Sciences

Journal homepage: http://www.afjbs.com

Research Paper Open Access

Analysis of WBC and Platelet Count among Patients with Sickle Cell Disease in the Waghodia Region, Vadodara, Gujarat

Charmi C. Thakkar[1], Dr. Inampudi Sailaja[2]*,Dr. Nisarg Savjiani[3]

[1]PhD Scholar, Department of Biochemistry, Parul Institute of Applied Sciences, Parul University, Vadodara-391760, Gujarat, India

[2]Associate Professor, Department of Biotechnology, Swaminarayan Sciences College (SSSC), Swaminarayan university, Ahmedabad-Mehsana Highway, at & P.O. Saij, Taluka-Kalol-382725, Dist-Gandhinagar, Gujarat, India

[3]Associate Professor, Department of Pathology, Parul Institute of Medical Sciences& Research, Parul University, Vadodara-391760, Gujarat, India

Corresponding author- **Dr. Inampudi Sailaja***

Article History

Volume 6, Issue 15, Sep 2024

Received: 15 July 2024

Accepted: 25 Aug 2024

Published: 05 Sep 2024

doi:10.48047/AFJBS.6.15.2024.3086-3090

Abstract: ***Introduction*:** Haematological parameters are extremely useful markers for effective disease management. Gujarat has seen little research on the haematological characteristics of sickle cell disease, yet.***Objective*:** The study aimed to observe the alterations in haematological results among sickle cell disease patients. ***Materials and Methods*:** The overall number of sickle cell anaemia patients registered in our institute for this prospective study is 150. A complete blood count (CBC) and the sickling test were performed on each individual. The outcomes of sickle cell trait and sickle cell illness were examined. ***Results:***The SCA patients' average age was 25.54±10 years. Age groups 25–30 years old (n = 35) account for the majority of participants, followed by 20–25 years old (n = 30). Out of the 150 SCA patients, there were 89 men (59.33%) and 61 females (40.66%), respectively. The platelet count (lacs/cumm) in the SCT and SCD groups is 3.75±1.10 and 4.04±0.94, respectively, whereas the mean value of the WBC count (/cumm) in the SCT group is 10611±3015 and the SCD group is 14427±3693. ***Conclusions*:** The current study discovered that SCD patients had higher WBC and PLT levels, which may have been caused by a spleen effect in these individuals. In addition to providing additional descriptive information about SCD patients in Gujarat, these haematological markers may also be a helpful tool for doctors managing SCD patients in India.

Keywords: Sickle cell, WBC (White blood cell), Platlet count, vasoocclusion crisis (VOC).

INTRODUCTION

An altered kind of haemoglobin in the blood causes the genetic illnesses known as sickle cell disease (SCD) and its variants 1, 2]. Renal sickness is one of the most common consequences, and kidney damage can cause significant difficulties as it can begin in childhood and increase throughout life. I. According to estimates, India is dwelling to more than half of all SCD patients globally. Scheduled tribal and other backward caste groups, as well as scheduled tribal states (Madhya Pradesh, Orissa, Chhattisgarh, Jharkhand, Gujarat, Andhra Pradesh, and Kerala), have higher carrier frequencies of the HbS gene [3, 4].

Complications resulting from a vasoocclusive crisis (VOC) characterize the clinical picture of sickle cell disease (SCD) VOC is produced as a result of a complicated process.

MATERIALS AND METHODS

In cooperation with ParulSevashram Hospital in Vadodara, Gujarat, this cross-sectional study was carried out at Parul Institute of Applied Sciences in 2017–2018.

The criteria for inclusion: Patients with sickle cell disease who have been confirmed to have the condition by any confirming tes t, such as Hb gel electrophoresis, capillary electrophoresis, and genetic analysis, as well as who have had their complete blood cou nt (CBC) examined and are at least five years old, will be eligible for enrollment.

Size of sample: There will be 150 patients with sickle cell disease who have already received a diagnosis.

Data collection

Data collection of following parameters will be done:

1. Age, sex and weight of the patients
2. BMI (Body mass index)
3. Hemogram (CBC)

Specimens and Investigations

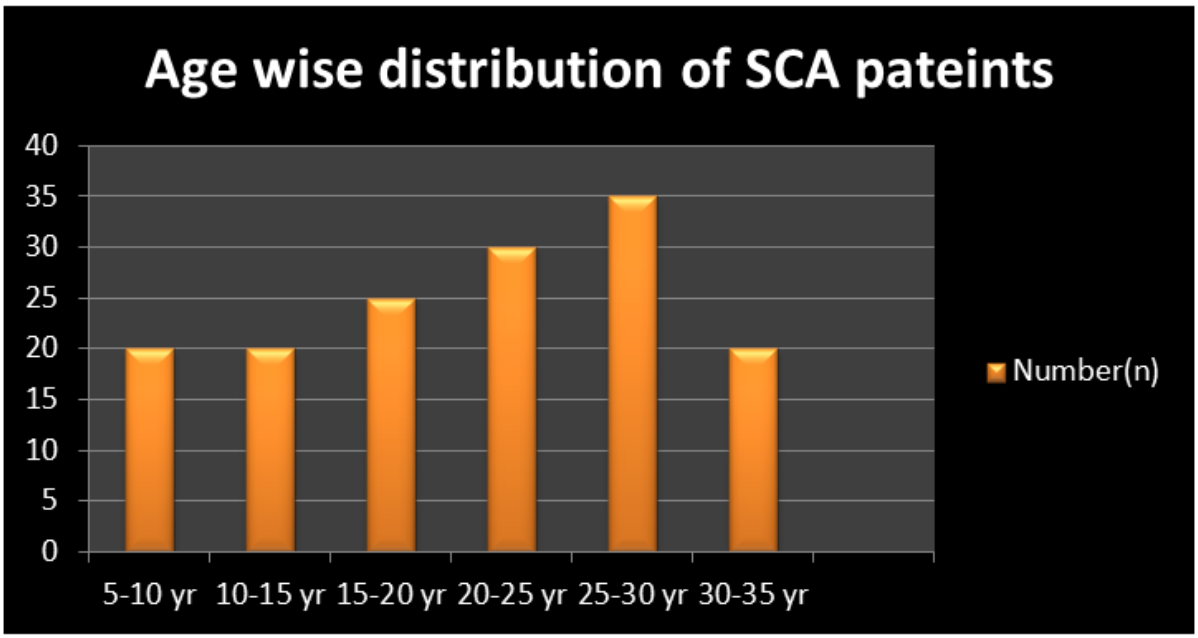
Blood samples for Complete blood count was collected aseptically in 5 ml EDTA vaccutainers. AnUniq ID was mentioned in all samples to hidden the identity of patients. WBC and platelet count estimation done by using 6 part cell counter (Impedence Method) at central laboratory of our hospital. Results of all collected samples were analysed statistically and calculate the Mean, SD and CV.

RESULTS

The mean age of the SCA patients was 25 54±10 years (Table 1). Maximum participants are found to be from age group 25-30 yr (n=35) followed by 20-25 yr (n=30) of the150 SCA patients, 89 (59 33%), and 61 (40 66%) were males and females, respectively.

Table 1: Demographic Characteristics of participants

Age Group(yr)	Number(n)
5-10 yr	20
10-15 yr	20
15-20 yr	25
20-25 yr	30
25-30 yr	35
30-35 yr	20
Total	150



Graph 1: Graphical Distribution of participants according to Age group

Table 2: Distribution of participants Based on type of sickle cell anemia (SCA)

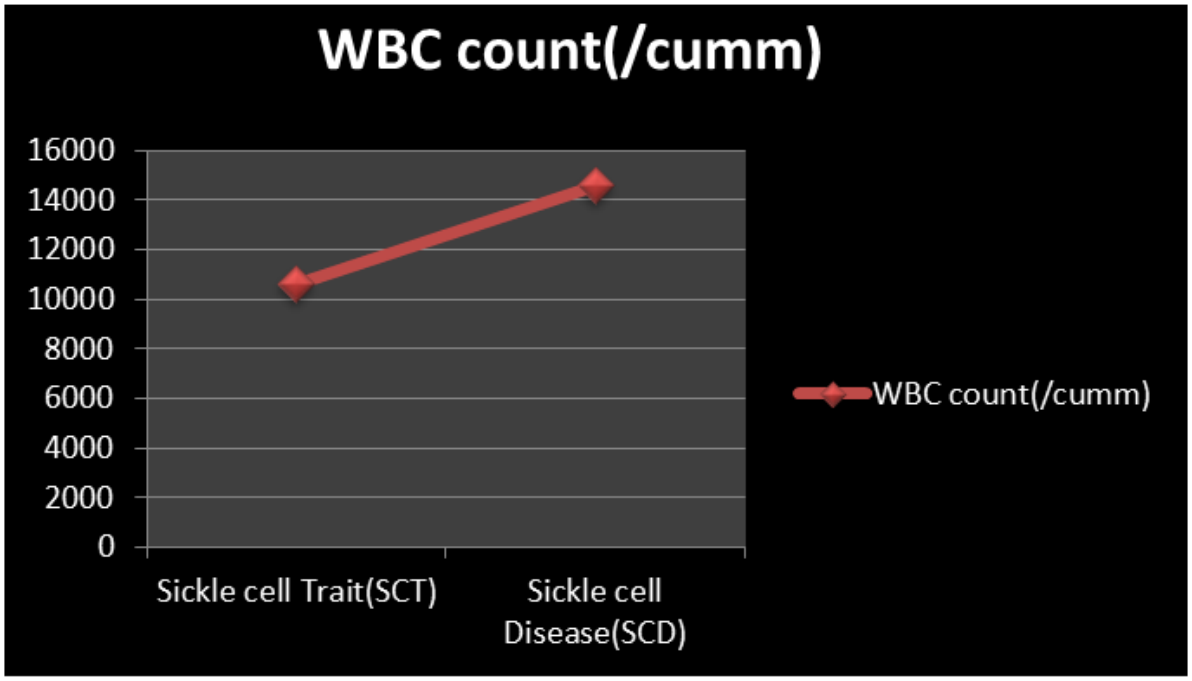
Total	Sickle cell trait(SCT)	Sickle cell disease(SCD)
150	92(61.33%)	58(38.66%)

Table 3: Gender wise distribution of participants

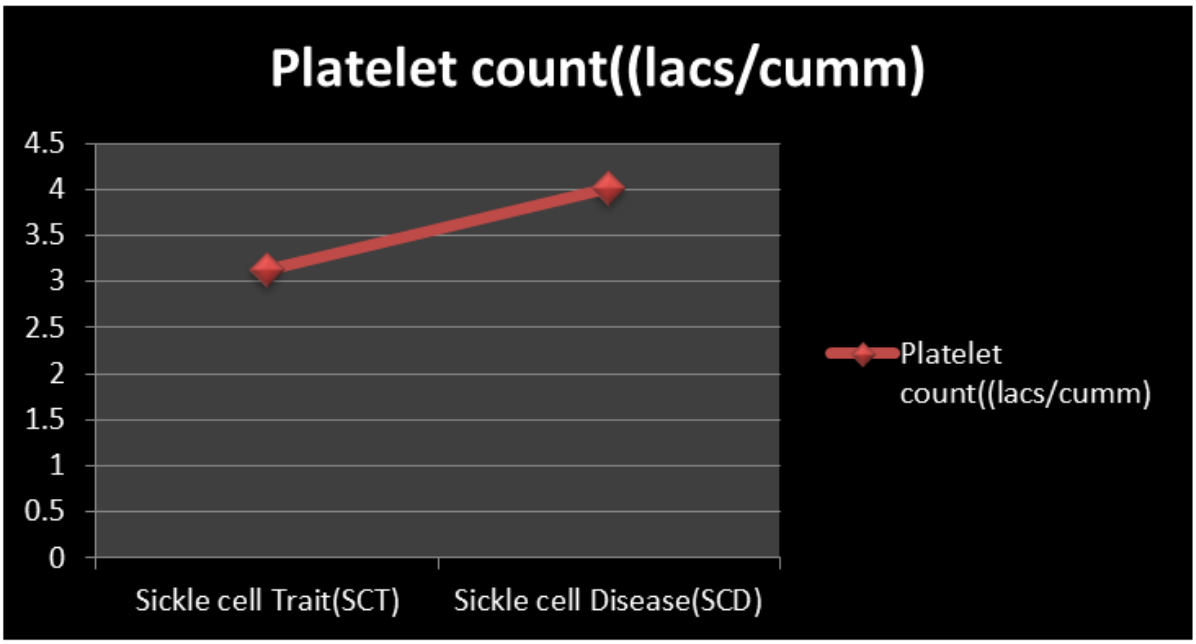
Total	Gender	Ratio
150	Male: female	89:61

Table 4: Hematological changes in SCT and SCD patients

	WBC count(/cumm)	Platelet count ((lacs/cumm)	P-value
Sickle cell Trait (SCT)	10611±3015	3.75±1.10	<0.05
Sickle cell Disease (SCD)	14427±3693	4.02±0.94	<0.05



Graph 2: Showing correlation of WBC count with SCT and SCD group



Graph 3: Showing correlation of Platlet count with SCT and SCD group

The Mean value of WBC count (/cumm) in SCT group is 10611±3015 and SCD is 14427±3693, the difference among them was found to be significant, p value is less than 0 05 while the Mean value of Platelet count ((lacs/cumm)is 3 75±1 10 and 4 04±0 94 SCT and SCD Group respectively and the difference among them was found to be significant, p value is less than 0 05 (Table 4).

DISCUSSION

The hematological features and clinical severity of sickle cell disease (SCD) are influenced by environmental, genetic, and gender variables. The existence of α-thalassemia, differences in Hb F levels, and haplotype background associated with the β

globin gene all affect how severe the condition is [13, 14]. This study highlights the association between hematological parameters and vaso-occlusion (VOC) in SCD patients in India. A plethora of research indicates that changes in hematological parameters may be the cause of the clinical problems that SCD patients experience [15, 16]. Therefore, an effective approach to managing sickle cell disease (SCD) can be achieved through the routine assessment of hematological parameters and the resolution of the underlying causes of these alterations.

Francis RB *et al*., conducted a similar study in India and found that SCD patients had elevated PLT levels [17]. This finding was likewise obtained in our study. Previous research has also shown that SCD patients have elevated WBC. Therefore, as shown in the current study, WBC and PLT counts are anticipated to rise in all patients who may present with any kind of SCD-related morbidity. Specifically, the inclusion of individuals with VOC (HbSS VOC and HbSC VOC), the hallmark of sickle cell disease, and the larger subject sample size were important aspects of this study.

The underlying chronic inflammation and potential splenic sequestration, decrease, or lack of spleen due to hyposplenismin SCD or autosplenectomy could be the cause of the increased PLT count observed in SCD patients. The increased PLT counts among SCD patients in this study are supported by reports that there is a link between PLT count and SCD [18], albeit this correlation was not shown in this investigation. The results of Freedman and Karpatkin's research are consistent with our investigation on elevated stable PLT counts in SCD patients [19, 20].

Comparing platelet counts in crisis with thrombocytosis, a common observation in our study is in line with a previous study by Okpala I *et al*., [21]. When it comes to the severity or consequences of the disease, there is inconsistent data about the predictive value of elevated baseline platelet counts. Few studies have been conducted in the literature on the relationship between thrombocytosis in sickle cell crises and outcome. The findings of our study indicate that reduced illness severity scores and a greater chance of survival are linked to increased platelet counts during times of crisis.

According to a prior study, patients with sickle cell disease (SCD) exhibit increased amounts of white blood cells (WBC), activated granulocytes, monocytes, and endothelial cells, as well as greater expression of endothelial cell adhesion markers, cytokine levels, and acute-phase reactants. Furthermore, a different study found that using medications like Hydroxyurea lowers WBC counts and enhances SCD patients' clinical outcomes. Anemia, which is typically seen in SCD patients, is a sign of the disease's overall severity. While SCD patients with greater Hb counts or values report higher frequencies of acute pain [22], lower steady-state Hb typically explains the higher risk of stroke in these same patients. High leukocyte count seems to be associated with some danger, as previous reports have shown.

Previous studies have shown that a high leukocyte count may be associated with an increased risk of many severe sickle cell disease (SCD) consequences, including rates of acute chest syndrome, severe pain, and mortality. Increased white blood cell counts have been linked to cerebrovascular accidents, according to research by Balkaran*et al*., [23, 24]. High PLT counts were also seen by Omotiet colleagues in their investigation in both steady state and vaso-occlusion SCD patients. WBC counts in SCD patients were typically raised, however it's important to note that patients with HbSS VOC had a much larger disparity in counts. When a result, an increased WBC count is anticipated in these patients when their SCD condition worsens and becomes hemolytic [25].

CONCLUSION

The current study discovered that SCD patients had higher WBC and PLT levels, which may have been caused by a spleen effect in these individuals. In addition to providing additional descriptive information about SCD patients in Gujarat, these haematological markers may also be a helpful tool for doctors managing SCD patients in India.

Acknowledgement: We acknowledge the all staff of our institute for their support.

REFERENCES

1. Platt, O. S., Brambilla, D. J., Rosse, W. F., Milner, P. F., Castro, O., Steinberg, M. H., & Klug, P. P. (1994). Mortality in sickle cell disease--life expectancy and risk factors for early death. *New England Journal of Medicine*, *330*(23), 1639-1644.
2. Ashley-Koch, A., Yang, Q., &Olneyi, R. S. (1998). Hemoglobin S Allele and Sickle Cell Disease. *American Journal of Epidemiology*, *151*(9), 839-845.
3. Steinberg, M. H. (2005). Predicting clinical severity in sickle cell anaemia. *British journal of haematology*, *129*(4), 465-481.
4. Alkjaersig, N., Fletcher, A., Joist, H., & Chaplin, H. (1976). Hemostatic alterations accompanying sickle cell pain crises. *The Journal of laboratory and clinical medicine*, *88*(3), 440-449.
5. Billett, H. H., Nagel, R. L., &Fabry, M. E. (1988). Evolution of laboratory parameters during sickle cell painful crisis: Evidence compatible with dense red cell sequestration without thrombosis. *The American journal of the medical sciences*, *296*(5), 293-298.
6. Allen, U., MacKinnon, H., Zipursky, A., & Stevens, M. (1988). Severe thrombocytopenia in sickle cell crisis. *Pediatric hematology and oncology*, *5*(2), 137-141.
7. Rowley, P. T., & Jacobs, M. (1972). Hypersplenic thrombocytopenia in sickle cell-bata thalassemia. *The American journal of the medical sciences*, *264*(6), 489-493.
8. Goldstein, A. R., Anderson, M. J., &Serjeant, G. R. (1987). Parvovirus associated aplastic crisis in homozygous sickle cell disease. *Archives of Disease in Childhood*, *62*(6), 585-588.

9. Ahmed, S. G., Ibrahim, U. A., & Hassan, A. W. (2006). Hematological parameters in sick cell anemia patients with and without priapism. *Annals of Saudi medicine*, *26*(6), 439-443.
10. Ahmed, A. E., Ali, Y. Z., Al-Suliman, A. M., Albagshi, J. M., Al Salamah, M., Elsayid, M., ...& Al-Jahdali, H. (2017). The prevalence of abnormal leukocyte count, and its predisposing factors, in patients with sickle cell disease in Saudi Arabia. *Journal of blood medicine*, 185-191.
11. Freedman, M. L., &Karpatkin, S. (1975). Elevated platelet count and megathrombocyte number in sickle cell anemia. *Blood*, 46, 579-582.
12. Ohene-Frempong, K., Weiner, S. J., Sleeper, L. A., Miller, S. T., Embury, S., Moohr, J. W., ... & Sickle Cell Disease, T. C. S. O. (1998). Cerebrovascular accidents in sickle cell disease: rates and risk factors. *Blood, The Journal of the American Society of Hematology*, *91*(1), 288-294.
13. Castro, O., Brambilla, D. J., Thorington, B., Reindorf, C. A., Scott, R. B., Gillette, P., ...& Levy, P. S. (1994). The acute chest syndrome in sickle cell disease: incidence and risk factors. The Cooperative Study of Sickle Cell Disease.*Blood*, *84*(2), 643-649.
14. Adegoke, S. A., &Kuti, B. P. (2013). Evaluation of clinical severity of sickle cell anemia in Nigerian children. *Journal of applied hematology*, *4*(2), 58-64.
15. Öner, C., Dimovski, A. J., Olivieri, N. F., Schiliro, G., Codrington, J. F., Fattoum, S., ...&Huisman, T. H. (1992). Beta S haplotypes in various world populations. *Human Genetics*, *89*(1), 99-104.
16. Kenny, M. W., George, A. J., & Stuart, J. (1980). Platelet hyperactivity in sickle-cell disease: a consequence of hyposplenism. *Journal of clinical pathology*, *33*(7), 622-625.
17. Francis Jr, R. B. (1991). Platelets, coagulation, and fibrinolysis in sickle cell disease: their possible role in vascular occlusion. *Blood coagulation & fibrinolysis*, *2*(2), 341-354.
18. Nagose, V., &Rathod, S. (2018). Hematological profile of sickle cell anemia subjects in central India: a cross-sectional analysis. *Ann Pathol Lab Med*, *5*(1), A87-A91.
19. Platt, O. S., Brambilla, D. J., Rosse, W. F., Milner, P. F., Castro, O., Steinberg, M. H., & Klug, P. P. (1994). Mortality in sickle cell disease--life expectancy and risk factors for early death. *New England Journal of Medicine*, *330*(23), 1639-1644.
20. Okpala, I. (2004). The intriguing contribution of white blood cells to sickle cell disease–a red cell disorder. *Blood reviews*, *18*(1), 65-73.
21. Okpala, I. (2002). Steady-state platelet count and complications of sickle cell disease. *The Hematology Journal: the Official Journal of the European Haematology Association*, *3*(4), 214-215.
22. Krishnan, S., Setty, Y., Betal, S. G., Vijender, V., Rao, K., Dampier, C., & Stuart, M. (2010). Increased levels of the inflammatory biomarker C-reactive protein at baseline are associated with childhood sickle cell vasocclusive crises. *British Journal of Haematology*, *148*(5), 797-804.
23. Al-Basheer, G., Abdelgadir, O., Mustafa, M. E. A., Muddathir, M. R. A., &Abdelgadir, E. R. (2015). C: reactive protein level and WBC count as biomarkers for vaso-occlusive crisis among patients with sickle cell disease. *Am J Med MedSci*, *5*(6), 283-286.
24. Balkaran, S. H., Hebbel, R. P., Mohandas, N., & Steinberg, M. H. (1994). Sickle cell disease: Basic Principles and Clinical Picturei New York: Raven Press, 311–311.
25. Omoti, C. E. (2005). Haematological values in sickle cell anemia in steady state and during vasoocclusive crisis in india. *Ann Afr Med*, *4*(2), 62–67.
26. Colah, R. B., Mukherjee, M. B., Martin, S., &Ghosh, K. (2015). Sickle cell disease in tribal populations in India. *Indian Journal of Medical Research*, *141*(5), 509-515.