Central African Journal of Public Health
2026, Vol. 12, No. 6, pp. 333–340
https://doi.org/10.11648/j.cajph.20261206.12

Research Article

Persistence of Fetal Hemoglobin and Clinico-biological Complications in Homozygous Sickle Cell Patients

Mvogo Manga Charly[1, 2, 3], Manga Sipewa Marie Julie[1], Gueguim Cedric[4, 7, *], Atangana Ngono Murielle[1], Messina Nkama Ines[5], Stephane Fargeon Massin Mbouna[1], Bidi Abada Protais[1], Etame Sone Lucien[1, 6]

[1]Higher Institute of Medical Technology (ISTM), Department of Biomedical Sciences, Yaounde, Cameroon

[2]Faculty of Medicine and Pharmaceutical Sciences, University of Douala (UD), Douala, Cameroon

[3]Doctoral Training Unit, School of Health Sciences, Catholic University of Central Africa, Yaounde, Cameroon

[4]Higher Institute of Agriculture Wood Water and Environment, University of Bertoua, Bertoua, Cameroon

[5]Central Hospital of Yaounde (HCY), Laboratory Services, Yaounde, Cameroon

[6]Institute of Medical Research and Medicinal Plants Studies, Ministry of Scientific Research and Innovation, Yaounde, Cameroon

[7]Faculty of Science, University of Bertoua, Bertoua, Cameroon

Abstract

Homozygous sickle cell disease is the most common hemoglobinopathy worldwide, particularly in Africa where it remains a major cause of morbidity and mortality. Fetal hemoglobin (HbF) plays a recognized protective role by inhibiting the polymerization of hemoglobin S. The aim of this study was to evaluate the association between HbF levels and the occurrence of clinical and biological complications in homozygous sickle cell patients. This was a prospective descriptive and analytical study conducted from September 2022 to August 2023. A total of 114 patients aged between 6 and 63 years were included. Socio-demographic and clinical data were collected using a standardized survey form. Biological parameters (Complete Blood Count, urea, creatinine, transaminases, C-Reactive Protein, and hemoglobin electrophoresis) were analyzed from blood samples. Statistical analysis was performed using CSPro 7.0 and Excel software. Among the 114 patients, 57.9% were female and 42.1% male, with a mean age of 21 years. The mean HbF level was 7.04%, ranging from 0 to 11.7%. Only 13% of patients had an HbF level ≥ 10%. The mean hemoglobin concentration was 7.6 g/dL, with 28.9% of cases presenting severe anemia. Vaso-occlusive crises (VOC) were the most frequent clinical complication (72.68%). Biologically, elevated CRP (≥ 6 mg/L) was found in 94.7% of patients. The overall frequency of clinical complications (VOC, septicemia, pneumonia, malaria, acute chest syndrome, polyarthritis, osteomyelitis, stroke, priapism, epigastric pain, and osteoarticular involvement) was higher in patients with HbF < 10%. A statistically significant association was observed between elevated HbF (≥ 10%) and reduced frequency of certain clinical complications, notably polyarthritis, osteomyelitis, stroke, priapism, and osteoarticular involvement ($p < 0.05$). Furthermore, higher HbF levels were significantly associated with a reduction in severe anemia ($p = 0.03$). Regarding the biological complications studied (transaminases > 40 IU/L, CRP ≥ 6 mg/L, hyperleukocytosis > 120,000/mL, urea > 0.50 g/L, creatinine > 13 g/dL), CRP was the only abnormality more frequently observed in patients with HbF ≥ 10%. In conclusion, fetal hemoglobin persists at variable levels in homozygous sickle cell patients, regardless of age and sex. Elevated HbF levels (≥ 10%) are significantly associated with a decrease in clinical and biological complications, confirming its protective role in sickle cell disease.

*Correspondence: Gueguim Cedric (guecedricfr@yahoo.fr)

Received: 28 July 2026; **Accepted:** 8 August 2026; **Published:** 26 November 2026

Copyright: © The Author(s), 2026. Published by Science Publishing Group. This is an **Open Access** article, distributed under the terms of the Creative Commons Attribution 4.0 License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution and reproduction in any medium, provided the original work is properly cited.

Keywords

Homozygous Sickle Cell Disease, Fetal Hemoglobin, Clinico-biological Complications

1. Introduction

Hemoglobinopathies are autosomal recessive hereditary diseases [1, 2]. They are divided into two groups: quantitative abnormalities (thalassemias), resulting from partial or complete defects in hemoglobin synthesis, and qualitative abnormalities (sickle cell disease), characterized by abnormal hemoglobin structure. Hemoglobinopathies are further classified into two categories: major (β-thalassemia and homozygous sickle cell disease) and minor (α-thalassemia and heterozygous sickle cell disease) [3].

Homozygous sickle cell disease is the most widespread major hemoglobinopathy worldwide. It is caused by a genetic mutation on chromosome 11 [4], affecting the gene encoding the β-globin chain [3, 5]. The resulting hemoglobin is highly prone to polymerization, deforming red blood cells into a sickle shape when the partial pressure of oxygen is low [4].

Sickled red blood cells lose their deformability, become rigid, and obstruct small blood vessels. This vaso-occlusion leads to various acute and chronic complications such as vaso-occlusive crises, acute chest syndrome, priapism, strokes, severe anemia, infections, and renal failure [4].

These complications are modulated by internal and external factors, notably fetal hemoglobin (HbF). HbF, composed of α and γ chains, has a higher affinity for oxygen than hemoglobin A because it interacts weakly with 2,3-diphosphoglycerate (2,3-DPG), a glycolysis metabolite that regulates hemoglobin's affinity for oxygen. HbF inhibits the polymerization of abnormal hemoglobin (HbS) and reduces red blood cell sickling, which is beneficial for patients [3, 6].

More than fifty (50) million people worldwide are affected by homozygous sickle cell disease [6, 7]. Africa is the most impacted continent, with 38 million cases and over 300,000 births recorded annually [7]. Infant mortality is extremely high, with 50-70% of children with sickle cell disease dying before the age of five [6, 9]. In Cameroon, more than 2 million patients are reported [6, 8] with approximately 4,000 new births each year [9].

The high morbidity and mortality, along with the complexity of management, make sickle cell disease a public health priority. Research in this field is vast, yet data remain limited and scarce in Cameroon. In this context, we conducted a study aimed at evaluating the correlation between HbF levels and clinico-biological complications in homozygous sickle cell patients.

2. Materials and Methods

2.1. Type of Study

The study was a prospective, cross-sectional, and analytical study was conducted as part of this work.

2.2. Study Sites

Samples were collected at the Central Hospital of Yaoundé. Analyses were performed in the laboratory of the Central Hospital of Yaoundé (CBC, urea, CRP, creatinine, and transaminases), as well as at the Centre for Social and Health Animation (CSHA) in Nkolndongo (hemoglobin electrophoresis).

2.3. Study Duration

The study was carried out from September 10, 2022 (protocol drafting) to August 3, 2023 (thesis defense), covering a one-year period. Fieldwork, including the collection of clinical, sociodemographic, and biological data, was conducted over three months and nine days, from April 3, 2023, to July 12, 2023.

2.4. Study Population

Homozygous sickle cell patients of all ages, both sexes, and all origins, followed at the Sickle Cell Center of the Central Hospital of Yaoundé.

2.5. Sampling

A non-probabilistic consecutive exhaustive sampling method was used. The sample size was calculated using Lorentz's formula, based on the prevalence of sickle cell disease in Cameroon: 8.34% [12], with a 95% confidence level (Z = 1.96) and a 5% margin of error. The calculation yielded a sample size of 126 patients.

2.6. Ethical Considerations

The study was conducted in accordance with the principles of ethics and human research as outlined in the Declaration of Helsinki (1964) and its subsequent revisions. Ethical clearance was obtained from the Institutional Ethics Committee for Human Health Research of the University of Douala, as well as research authorization from the Director of the Central Hos-

pital of Yaoundé. Prior to inclusion, informed consent was obtained from adult patients, and assent from parents or guardians for minors.

2.7. Data Collection and Analysis

Sociodemographic and clinical data were collected using standardized survey forms. Biological data were obtained after sample analysis and recorded on the same forms. Data processing and analysis were performed using CSPro 7.0, PowerPoint, and Microsoft Word software.

3. Results

3.1. Sociodemographic Characteristics

A total of 114 patients aged between 6 and 63 years were included in the study, comprising 66 females and 48 males (57.9% and 42.1%, respectively). The sex ratio (F/M) was 1.3. The mean age of participants was 21 years, with a 95% confidence interval of 19.55-23.71 years, and extremes of 6 and 63 years. The most represented age group was 20-29 years, accounting for 34.2% of the study population. The population was predominantly young (0-30 years), representing 81.6% of the total sample. A schooling rate of 78.1% was observed, and 96.5% of the study population resided in urban areas.

Table 1. *Distribution by sex, age, occupation, and living environment.*

Variable	Numbers (N=114)	Frequency (%)
Sex		
Male	48	42.1
Female	66	57.9
Age group (years)		
[0-9]	18	15.7
[10-19]	36	31.6
[20-29]	39	34.2
[30-39]	10	8.8
[40 ≥]	11	9.7
Occupation		
Students (primary and secondary)	63	55,3
Students (unversity)	26	22.8
Workers	5	4.4
Unemployed	20	17.5
Living environment		
Urban	110	96,5
Rural	4	3,5

3.2. Frequency of Clinical Complications

Among the 114 patients included in the study, 84 presented at least one clinical complication, representing 73.68% of the total population. Vaso-occlusive crisis is by far the most common complication in the population, with a rate of 72.68%.

Table 2. *Frequency of Clinical Complications.*

Complication	Number	Frequency among with complications (n=84)	Frequency in the total population (N=114)
VOC (Vaso-occlusive Crisis)	83	98.80%	72.68%
Septicemia	10	11.90%	08.77%
Pneumonia	4	7.89%	4.76%
Malaria	09	10.71%	7.89%
Acute Chest Syndrom (ACS)	06	7.14%	5.26%
Polyarthritis	02	2.38%	1.75%
Osteomyelitis	05	5.95%	4.39%
Osteoarthritis	01	1.19%	0.87%
Stroke	02	2.38%	1.75%
Priapism	01	1,19%	0,87%
Epigastric pain	01	1,19%	0,87%

3.3. Biological Characteristics

3.3.1. Electrophoretic Data

Out of 114 patients, 113 had persistence of fetal hemoglobin, representing a persistence rate of 99.12%. Only 1 patient showed no persistence of fetal hemoglobin, corresponding to a non-persistence rate of 0.88%. The extreme values ranged from 0.0% to 11.7%, with a mean rate of 7.04%. The distribution frequencies of fetal hemoglobin according to whether the rate was below 10% or equal to/above 10% were 87% (99 patients) and 13% (15 patients), respectively. Fetal hemoglobin persists at variable levels regardless of age groups.

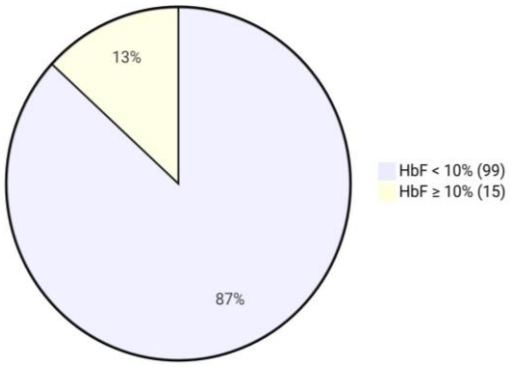

Figure 1. Distribution Frequency of Fetal Hemoglobin Levels.

Table 3. Electrophoretic Data.

Indicator	Value
Main	7,04%
Minimum	0,0%
Maximum	11,7%

Table 4. Mean Fetal Hemoglobin Levels According to Age Groups.

Age group (years)	Total number	Main HbF (%)
[0-9]	18	6,8
[10-19]	36	6,8
[20-29]	39	7,3
[30-39]	10	6,5
[40-49]	6	7,4
[50-59]	3	8,5
[60-69]	2	5,5
Overall mean 7,04%		

3.3.2. Other Biological Data

Table 5. Other Biological Data.

Paramètres	Moyenne ± ET
Hemoglobin level (g/dL)	7,6 ± 1,4
MCV (fL)	84,6 ± 10,9
White Blood Cell count (/mm^3)	12 328 ± 5 122
CRP (mg/L)	104,8 ± 67,52
Urea (g/L)	0,33 ± 0,19
Serum creatinine (mg/L)	9,9 ± 3,7
AST (U/L)	31,1 ± 49,0
ALT (U/L)	28,8 ± 38,0

3.3.3. Frequency of Biological Complications

Anemia is present in 100% of the population. However, the critical threshold (hemoglobin level < 7 g/dL) is observed in 28.94%. C-reactive protein (CRP) is the most frequent biological complication in the population, with a rate of 94.73%.

Table 6. Frequency of Biological Complications.

Parameter	Abnormal threshold	Abnormal cases	Frequency (%)
CRP (C-reactive Protein)	≥ 6 mg/L	108	94.73%
Hemoglobin (Hb)	< 07 g/dL	33	28.94%
White Blood Cell (WBC)	> 12 000/mm^3	51	45.94%
Urea	> 0,50 g/L	08	7.01%
Creatinine	> 13mg/L	19	16.67%
ALT	> 40 UI/L	03	2.68%

Parameter	Abnormal threshold	Abnormal cases	Frequency (%)
AST	> 40 UI/L	06	5.26%

3.4. Evaluation of the Correlation Between the Persistence of Fetal Hemoglobin and Clinical-biological Complications

3.4.1. Evaluation of the Correlation Between the Persistence of Fetal Hemoglobin and Clinical Complications

The study of clinical complications according to different fetal hemoglobin levels is presented in Table 7. The frequency of clinical complications is elevated to 100% among subjects with HbF levels < 10%. Several significant correlations were found between the persistence of HbF ≥ 10% and the occurrence of clinical complications, notably stroke, priapism, epigastric pain, polyarthritis, and osteomyelitis ($p < 0.05$).

Table 7. *Correlation Between the Persistence of HbF and Clinical Complications.*

Clinical complications	Percentage in total sample		Odds Ration	95% Confidence Interval	p-value
	HbF < 10%	HbF≥10%			
CVO	72%	11%	1,48	0,61-3,58	0,38
Septicemia	8%	2%	0,68	0,14-3,21	0,62
Pneumonia	4%	0%	0,39	0,02-7,47	0,55
Malaria	7%	3%	0,40	0,10-1,60	0,21
Acute Chest Syndrom	4%	3%	0,21	0,04-1,09	0,07
Polyarthritis	2%	0%	0,48	0.02-10,6	< 0,05
Osteomyelitis	5%	0%	0,30	0,02-5,49	< 0,05
Stroke	2%	0%	0,48	0,02-10,6	< 0,05
Priapism	1%	0%	0,96	0,03-30,5	< 0,05
Epigastric pain	1%	0%	0,96	0,03-30,5	< 0,05
Osteoarthritis	1%	0%	0,96	0,03-30,5	< 0,05

3.4.2. Persistence of Fetal Hemoglobin and Biological Complications

The study of biological complications according to different fetal hemoglobin levels is presented in Table 8. Anemia was present in 100% of the study population. However, severe anemia (hemoglobin < 7 g/dL) was observed in 33 patients, representing 28.94% (see Table 6). Severe anemia was significantly correlated with HbF ≥ 10%. Overall, the high frequency of biological complications was correlated with HbF < 10%, and inversely when HbF ≥ 10%.

Table 8. *Correlation Between the Persistence of HbF and Biological Complications.*

Biological Complications	Percentage in the total sample		Odds Ratio	95% Confidence Interval	p-value
	HbF <10%	HbF≥10%			
Severe anemia (Hb <7g/dL)	96.97%	3.03%	5.5	0.65-46.5	0.03
CRP ≥ 6 m/dL	88.89%	96.97%	1.25	0.58-2.70	0.56

Biological Complications	Percentage in the total sample		Odds Ratio	95% Confidence Interval	p-value
	HbF <10%	HbF≥10%			
Leukocytosis (>12000 mg/L)	88.89%	19.61%	0.72	0.31-1.65	0.47
Creatinine > 13 mg/L	88.89%	11.11%	0.85	0.30-2.10	0.68
Urea > 0.5 g/L	75.00%	25.00%	0.60	0.12-2.85	0.52
AST > 40 UI/L	83.33%	16.67%	0.90	0.10-7.80	0.91
ALT> 40 UI/L	100%	0%	0.94	0.04-20.5	0.21

4. Discussion

Homozygous sickle cell disease is the most widespread form of major sickle cell syndrome worldwide. It mainly affects Africa, with more than 38 million patients. It is a systemic disease requiring multidisciplinary management and involving several medical specialties due to its numerous acute and chronic complications. However, fetal hemoglobin, whose structure differs from hemoglobin A, appears to exert a protective effect against complications when present at high levels. It is in this perspective that the present study was conducted, aiming to investigate the correlation between HbF persistence and the occurrence of clinico-biological complications in sickle cell patients.

Thus, a prospective, cross-sectional, and analytical study was carried out. A total of 114 patients were included, among whom 66 were women (57.9%) and 48 men (42.1%), with a sex ratio of 1.3 (F/M). The mean age of participants was 21 years, ranging from 6 to 63 years. For patients whose life expectancy was previously considered limited due to a high infant mortality rate of 50-70% before the age of 5, the results of this study highlighted a significant increase in survival age among those benefiting from regular and adequate medical follow-up. This finding is consistent with that of Thabet Mohsen and al. [10] in Senegal (2020), who reported a life expectancy of 57 years and a female predominance (57%) similar to ours. Conversely, MI. Kueviakoe and al. [11] (Togo, 2011) observed a male predominance (54%), with a mean age of 18.93 years and a life expectancy of 75 years, confirming that disease transmission is not sex-dependent.

The study population was predominantly young, with 82.5% aged between 0-30 years. It included 63 school pupils and 26 students, representing a total schooling rate of 78.1%. This rate once again confirms a visible improvement in patient care. Since sickle cell disease is disabling, adequate management enables patients to pursue education and professional activities. However, this does not fully address the issue of schooling level relative to age, raised by Minto'o Rogombe and al. [13] in Gabon (2018), who concluded that one-third of children had delayed schooling, while two-thirds had normal or advanced schooling.

The mean fetal hemoglobin level was 7.04%, with extremes ranging from 0.0 to 11.7%. This rate is comparable to the 7.2% reported by Tshilolo L. and al. [14] (Congo-Kinshasa, 2010), and the 7.4% found by Nagel RL and al. [17] for the Benin and Bantu haplotypes. Literature indicates that HbF decreases slowly in sickle cell patients after the age of 5, but remains low for the Benin, Cameroon, and Bantu haplotypes.

Analysis of clinical complications revealed a higher frequency among patients with HbF < 10%. Vaso-occlusive crises were elevated (72%) in patients with HbF < 10%. However, studies by Eng and al. [15] (Cameroon, 2022) and Thabet Mohsen and al. [10] observed high rates of VOC in patients with HbF ≥ 10%, suggesting that HbF does not systematically influence the occurrence of VOC, priapism, or osteomyelitis. Conversely, strokes and acute chest syndrome (ACS) were found predominantly in patients with HbF ≥ 10% in this study and in that of Thabet Mohsen and al. [10].

On the biological level, anemia was present in all participants (100%), with a mean hemoglobin level of 7.6 g/dL. Similar results were reported by Thabet Mohsen and al. [10] (97% anemia, mean Hb 8.5 g/dL) and Tshilolo L. and al. [14] (mean Hb 7.2 g/dL). Severe anemia (Hb < 7 g/dL) was found in 28.94% of the study population (33 patients), mainly among those with HbF < 10% (96.97% of severe anemia cases). Higher frequencies were also reported in studies by Eng and al. [15] and Thabet Mohsen and al. [10], confirming the correlation between low HbF levels and anemia severity.

Additionally, 51 cases of leukocytosis (WBC > 12,000/mm^3) were noted. The leukocytosis observed in this study may result from hyperproduction in the bone marrow, associated with demargination linked to stress, infections, or inflammation. Systematic analysis of blood smears excluded false leukocytosis due to erythroblasts, as supported by Chies and al. [16] and Doupa D. and al. [15] The highest frequency of leukocytosis was observed in patients with HbF < 10% (88.89%). Thabet Mohsen et al. [10] also found elevated rates in patients with HbF < 10%.

Finally, C-reactive protein (CRP ≥ 6 mg/L) was positive in 88.89% (108 patients), reflecting the inflammatory state associated with VOC, although HbF ≥ 10% did not appear to influence this parameter. Overall, the frequency of biological

complications remained higher in patients with HbF < 10%. This result is corroborated by previous studies, notably those of Thabet Mohsen and al. [10], Eng and al. [15], Tshilolo L. and al. [14], and Kueviakoe and al. [11].

5. Conclusion

Fetal hemoglobin is an important factor influencing the severity of homozygous sickle cell disease. It persists in almost all patients in this study (99.12%), at variable levels regardless of age and sex. An HbF level ≥ 10% is correlated with a lower frequency of clinical and biological complications, and conversely, when HbF < 10%. Therefore, fetal hemoglobin plays a protective role in homozygous sickle cell patients.

Abbreviations

VOC	Vaso-Occlusive Crises
CBC	Complete Blood Count
CRP	C-Reactive Protein
WBC	White Blood Cell
AST	Aspartate Aminotransferase
ALT	Alanine Aminotransferase
HbF	Fetal Hemoglobin
SCD	Sickle Cell Disease
2,3-DPG	2,3-Diphosphateglycerate
HbS	Hemoglobin S
CSHA	Center for Social Health Animation
F/M	Female/Male
ACS	Acute Chest Syndrom

Acknowledgments

We extend our gratitude to all the medical staff of the Hematology-Oncology Department at the Central Hospital of Yaoundé for their support in data collection for this study. We also thank the staff of the Central Laboratory of the Central Hospital of Yaoundé and the laboratory of CSHA of Nkolndongo for their assistance and expertise during sample analysis. Finally, we sincerely thank all the patients who voluntarily agreed to participate in our study.

Author Contributions

Mvogo Manga Charly: Writing – review & editing, Ressources

Manga Sipewa Marie Julie: Project administration

Gueguim Cedric: Writing – review & editing

Atangana Ngono Murielle: Investigation

Messina Nkama Ines: Investigation

Stephane Fargeon Massin Mbouna: Data curation, Formal Analysis

Bidi Abada Protais: Data curation, Formal Analysis

Etame Sone Lucien: Supervision

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Elion J, Laurance S, Lapouméroulie C. Physiopathology of sickle cell disease. Medecine Tropicale. 2010: 450–458.

[2] Philippes Connes. Physiopathology of sickle cell disease. In: Sickle cell disease in children and adolescents. Chapter 1. Elsevier Masson SAS; 2020. Available at: www.elsevier-masson.fr

[3] Bain BJ. Haemoglobinopathy diagnosis. 2nd ed. Oxford: Blackwell Publishing; 2006. p. 2. Available at: www.lab-cerba.com

[4] Evan M. Branstein. Sickle cell disease (HbS hemoglobinopathy). Johns Hopkins University School of Medicine. Medical review. Updated June 2022.

[5] Pierre Aubry, Bernard-Alex Gauzère. Tropical medicine: hemoglobinopathies. Updated April 19, 2022. Available at: www.medecinetropicale.com

[6] Genevieve Côté, and al. Transfusion particularities in pediatrics. Published February 25, 2015. Available at: www.anesthesiologie.umontreal.ca

[7] World Health Organization. Sickle cell disease: Report of the 59th World Health Assembly, April 24, 2006. Available at: www.who.int

[8] Arnaud Ntchapda. Sickle cell disease: progress in Cameroon. Written June 18, 2020; updated November 23, 2021. Available at: ALLO DOCTEURS AFRICA.

[9] Drepavie: Sickle cell disease: the world's most common genetic disorder. Available at: http://www.drepavie.org

[10] Thabet Mohsen, and al. Influence of fetal hemoglobin persistence on the occurrence of clinico-biological complications in homozygous sickle cell disease. Published 2020. Available at: www.bu.ucad.sn

[11] MI. Kueviakoe, V. N'Zouonto, K. Agbtiafa, and al. Influence of fetal hemoglobin levels on the profile of homozygous sickle cell disease in Togo. Published 2010.

[12] Njifon H. Nsangou, Scelles R. Sickle cell disease and future perspectives: lived experience of a sister of a child with sickle cell disease in Cameroon. Elsevier; 2020. Available at: www.elsevier.com/open-access/userlicence/1.0

[13] Minto'o Rogombé, Kuissi Kamgaing, Minko JI, Mayi MM, Nkoghe VC, Koko J, and Ategbo S. Medical follow-up and schooling of children with sickle cell disease in Gabon. Health Sci Dis. 2018; 19(1). Available at: www.hsd-fmsb.org

[14] Tsilolo L, Wembonyoma S, Summa V, and Avvisati G. Hemogram of Congolese children with sickle cell disease in steady state. Published 2010. Available at: www.semanticscholar.org

[15] Eng YCM, Nko'o Amvene M, Awana AP, and al. Fetal hemoglobin levels in homozygous sickle cell children and effect on clinical severity. Health Sci Dis. 2022; 23(3 Suppl): 34–38. Available at: www.hsd-fmsb.org

[16] Chies JA, Nardi NB. Sickle cell disease: a chronic inflammatory disorder. Med Hypotheses. 2001; 51(1): 46–50. Available at: www.pubmed.ncbi.nlm.nih.gov (pubmed.ncbi.nlm.nih.gov in Bing)

[17] Nagel RL, Erlingsson S, Fabry MOI, and al. The Senegal DNA haplotype is associated with improved anemia in African-American sickle cell patients. Blood. 1991; 77(6): 1371–1375. Published March 15, 1991. Available at: www.pubmed.ncbi.nlm.nih.gov (pubmed.ncbi.nlm.nih.gov in Bing)