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Haematological Changes in the Different Hemoglobin Genotypes in Enugu Metropolis

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Abstract

The study assessed haematological changes among individuals with different haemoglobin genotypes in Enugu Metropolis. A total of 90 participants, comprising 43 males and 47 females, were enrolled and grouped into HbAA, HbAS, and HbSS, with 30 participants in each group. Blood samples (4.0 mL) were collected from each participant; 2.0 mL was dispensed into an EDTA container for haematological analysis, while the remaining 2.0 mL was used for haemoglobin electrophoresis using standard procedures. The results showed variations in haematological parameters among the genotype groups. Individuals with HbAA had mean haemoglobin, RBC count, and haematocrit values of 13.21 ± 1.16 g/dL, 4.53 ± 0.36 × 10^{12}/L, and 39.30 ± 4.05%, respectively. Corresponding values for HbAS individuals were 12.24 ± 1.18 g/dL, 4.03 ± 0.42 × 10^{12}/L, and 36.78 ± 4.12%, while HbSS individuals had markedly lower values of 8.66 ± 1.25 g/dL, 2.56 ± 0.40 × 10^{12}/L, and 26.01 ± 4.88%, respectively. The HbSS group also demonstrated higher mean WBC (11.72 ± 4.23 × 10^{9}/L), platelet (416.70 ± 116.44 × 10^{9}/L), and reticulocyte counts (7.84 ± 2.94%) than the other groups. The study revealed more pronounced anaemia among HbSS individuals, with a prevalence of 100%, followed by HbAS (60%) and HbAA (30%).

Keywords: Haematological Parameters, Haemoglobin Genotypes, HbSS, Anaemia, Sickle Cell Disease

Introduction

Haemoglobin, an iron-containing protein within red blood cells, plays a central role in oxygen transport from the lungs to peripheral tissues and in returning carbon dioxide for exhalation. Structurally, normal adult haemoglobin (HbA) consists of two alpha (α) and two beta (β) globin chains, each bound to a heme group that facilitates oxygen binding. Fetal haemoglobin (HbF), consisting of $\alpha_2\gamma_2$, predominates in utero and is gradually replaced by HbA after birth [1].

Genetic mutations affecting globin-chain structure or synthesis result in diverse haemoglobin genotypes. These include HbAA, the normal adult genotype; HbAS, the sickle cell trait; HbSS, sickle cell disease; and other variants like HbAC, HbSC, and HbE.

Although more than a thousand haemoglobin variants have been described worldwide, only a subset cause significant clinical manifestations [2].

Haemoglobinopathies are among the most common inherited disorders globally [2]. Certain variants, such as HbS and HbC, are particularly prevalent in malaria-endemic regions due to selective evolutionary advantages. For example, individuals heterozygous for HbAS are protected against severe Plasmodium falciparum malaria, explaining the high trait frequencies across sub-Saharan Africa [3]. Compound heterozygotes, such as HbS/β-thalassemia, are also observed in Asia and other regions, with phenotypes ranging from mild to severe [4].

The clinical consequences of these variants arise because structural changes or reduced production compromise erythrocyte stability, oxygen affinity, and lifespan. For instance, the HbS mutation, caused by the substitution of valine for glutamic acid at position 6 of the β-globin chain, results in reduced solubility of haemoglobin and polymerization under low oxygen conditions, leading to sickling [2]. Inheritance patterns follow Mendelian autosomal recessive laws, with two abnormal alleles required for disease expression, while heterozygous carriers are usually asymptomatic but can show subtle haematological differences [2].

Globally, haemoglobin disorders continue to impose a substantial disease burden. The World Health Organisation (WHO) estimates that over 300,000 infants are born annually with haemoglobin disorders, with sickle cell disease accounting for about 70% of these cases [6]. A recent meta-analysis highlights that accurate prevalence and birth-rate data remain incomplete despite improvements in surveillance [7].

Sub-Saharan Africa carries the highest share of this burden, with Nigeria consistently cited as one of the most affected countries. It is estimated that approximately 150,000 affected newborns are delivered annually, and over 40 million Nigerians carry the sickle cell trait [8]. Research indicates that among individuals with sickle cell disease, the HbSS genotype is predominant, accounting for 97.5% of cases, while HbSC accounts for the remaining 2.5% [9]. These figures indicate that nearly one-quarter of the population carries the sickle trait. Sickle cell disease remains a major cause of childhood morbidity and mortality in the country, demanding urgent attention to preventive, diagnostic, and therapeutic measures.

The clinical expression of haemoglobin variants is usually accompanied by characteristic haematological alterations. In HbSS, findings include chronic hemolytic anemia, elevated white blood cell counts, high reticulocyte counts, and other markers of hemolysis. Carriers of HbAS usually have near-normal indices, though subtle deviations in red cell distribution width or mean corpuscular haemoglobin have been reported [10]. Thalassaemia traits are also present with microcytic hypochromic anaemia and distinct red cell indices, which help in differentiating them from iron deficiency anaemia.

Understanding genotype-specific haematological profiles is important because it improves diagnostic accuracy, guides transfusion protocols, and helps tailor clinical interventions, particularly in resource-limited settings [10]. From a public health perspective, genotype-informed data support screening campaigns, health education, and genetic counselling. These interventions empower communities to make informed reproductive and health decisions while reducing the overall burden of disease. Awareness of prevalent genotypes also informs blood donor selection and compatibility testing, reducing risks such as alloimmunization in transfusion-dependent patients.

In South-East Nigeria, including Enugu Metropolis, the prevalence of haemoglobin variants remains high, contributing to significant public health concerns. Enugu, a rapidly urbanizing region with both rural and urban populations, provides a unique setting for studying haematological changes across genotypes. The presence of tertiary hospitals and diagnostic laboratories also makes it a suitable environment for such investigations. Recent data from Abakaliki, also in Southeastern Nigeria, underscores the high prevalence of the sickle cell trait, reporting that 35% of a study cohort were carriers (HbAS) [12]. These data highlight the need for a deeper understanding of the associated haematological implications in this population.

Despite advances in diagnosis, the Nigerian diagnostic landscape is limited by infrastructure. High-performance liquid chromatography (HPLC) and capillary electrophoresis are available mainly in tertiary centers, while most facilities still rely on basic haemoglobin electrophoresis and full blood count analysis. Misdiagnosis remains common, with reports suggesting error rates as high as 32% among parents of children with sickle cell disease in Lagos [11]. Local factors such as malaria co-infection and nutritional deficiencies further influence haematological parameters, making it necessary to conduct context-specific studies in regions like Enugu [3].

Haemoglobinopathies, especially sickle cell disease and related variants, represent a major public health problem in Nigeria. The high prevalence of these conditions and their significant impact on morbidity and mortality call for continued research. Despite the existence of screening and diagnostic programs, the lack of localized genotype-specific haematological data in Enugu limits effective intervention.

Previous studies have shown that haemoglobin variants influence red cell indices, white blood cell counts, and platelet levels [13]. However, most of these studies focus on hospital-based or paediatric populations. There is still a knowledge gap in understanding how these changes manifest in the general urban population of Enugu, where demographic diversity and varying access to healthcare could influence expression.

Establishing baseline haematological reference values for different haemoglobin genotypes in this locality will provide a useful resource for clinicians, researchers, and policymakers. It will also inform targeted health education, screening programs, and pre-marital counselling, ultimately contributing to reduced morbidity and mortality. This study is justified because it is timely, locally relevant, and has the potential to inform both clinical practice and public health strategy. This study aims to investigate the haematological alterations associated with different haemoglobin genotypes in Enugu Metropolis.

Materials and Methods

Study Area

This study was carried out at Mother of Christ Hospital, Ogui, located in Enugu Metropolis, Enugu State, Nigeria. The hospital is a missionary institution that attends to over 10,000 patients monthly. Laboratory analyses were conducted in the Haematology Laboratory of the Department of Medical

Laboratory Science, University of Nigeria, Enugu Campus.

Subjects
A total of 90 apparently healthy subjects comprising 42 males and 48 females who presented for haemoglobin genotype screening at Mother of Christ Hospital, Ogui participated in the study. Participants were grouped according to their confirmed haemoglobin genotypes (HbAA, HbAS, HbSS).

Inclusion Criteria
- Individuals aged 18 years and above.
- Individuals with confirmed haemoglobin genotype (HbAA, HbAS, or HbSS).
- Individuals who gave informed consent.

Exclusion Criteria
- Individuals below 18 years of age.
- Pregnant women.
- Individuals with known co-morbidities that could affect haematological parameters.
- Individuals not in a steady state at the time of sampling.

Sample Size Determination
Sample size formula for comparing two means:

$$n = 2 * (Z_{\alpha/2} + Z_\beta)^2 * (\sigma / \Delta)^2$$

Where:
$Z_{\alpha/2} = 1.96$ (for $\alpha = 0.05$)
$Z_\beta = 0.84$ (for 80% power)
$\sigma = 1.5$
$\Delta = 1.2$

Calculation:

$$n = 2 * (1.96 + 0.84)^2 * (1.5 / 1.2)^2$$

$$= 2 * (2.80)^2 * (1.25)^2$$

$$= 2 * 7.84 * 1.5625$$

$$= 24.5 \approx 25$$

Therefore, 25 participants per group were required. Allowing for dropouts, 30 participants were selected per group (total N = 90).

Study Design
A prospective cross-sectional study design was employed. Participants were consecutively recruited during the study period. Ethical approval was obtained from the Health Research Ethics Committee of the University of Nigeria Teaching Hospital (UNTH). Written informed consent was obtained from all participants after the objectives and procedures of the study were explained to them.

Sample Collection
Venous blood sample (4.0 ml) was collected aseptically from each participant through the antecubital vein using sterile disposable syringes. The samples were dispensed into dipotassium EDTA (K_2-EDTA) anticoagulant bottles, gently inverted several times to ensure proper mixing, and labelled accordingly.

Materials and Equipment
The following materials and equipment were used during the study:
- K_2-EDTA bottles
- Tourniquet
- Syringes and needles
- Sterile swabs
- Gloves
- Universal containers

Laboratory Analysis
Haemoglobin Genotype Confirmation
Venous blood (2 ml) was collected into EDTA tubes, stored at 2–8 °C, and analyzed within 24 hours. Red cells were washed with isotonic saline and lysed to prepare haemolysates. Haemoglobin electrophoresis was carried out on cellulose acetate strips using Tris-EDTA-borate buffer at alkaline pH (8.6). Each electrophoresis run included known genotype controls (AA, AS, SS, AC) for reference. After separation, the strips were stained with Ponceau S, destained in 5% acetic acid, and air-dried. Bands were identified by comparing their mobility with the controls, and samples with ambiguous patterns were re-run for confirmation.

Determination of Haematological Parameters
All haematological parameters were determined using standardized manual techniques to ensure accuracy and reliability.

1. Packed Cell Volume (PCV/Haematocrit): The Packed Cell Volume was determined using the microhaematocrit method. Capillary tubes were filled with well-mixed EDTA-anticoagulated blood and sealed at one end with cristaseal. The tubes were then centrifuged in a microhaematocrit centrifuge at 11,000 rpm for 5 minutes. The PCV was read directly using a microhaematocrit reader and expressed as a percentage.
2. Haemoglobin Concentration (Hb) Haemoglobin concentration was estimated using the Sahli's acid haematin method. A known volume of blood was mixed with 0.1N Hydrochloric acid (HCl) in a Sahli's haemoglobin tube to convert haemoglobin to acid haematin. After 10 minutes, the solution was diluted with distilled water drop by drop until its colour matched the permanent brown glass standard. The haemoglobin value was read directly from the scale and expressed in grams per decilitre (g/dL).
3. Red Blood Cell (RBC) and White Blood Cell (WBC) Counts Erythrocyte and leucocyte counts were performed using the manual haemocytometer method. For the RBC count, blood was diluted 1:200 with Hayem's fluid. For the WBC count, blood was diluted 1:20 with Türk's solution (glacial acetic acid with a dye). The diluted samples were charged into a Neubauer improved haemocytometer chamber and cells were counted under a light microscope at 40x magnification. The counts were calculated and expressed as cells per litre (x 10^{12}/L for RBCs and x 10^9/L for WBCs).
4. Red Cell Indices: MCV, MCH, MCHC The red cell indices were derived mathematically from the primary measurements:
   - Mean Corpuscular Volume (MCV) was calculated as: (PCV / RBC count) x 10, expressed in femtolitres (fL).

- Mean Corpuscular Haemoglobin (MCH) was calculated as: (Hb / RBC count) x 10, expressed in picograms (pg).
- Mean Corpuscular Haemoglobin Concentration (MCHC) was calculated as: (Hb / PCV) x 100, expressed in grams per decilitre (g/dL).

5. Platelet Count (PLT) Platelets were counted manually using a haemocytometer. Blood was diluted 1:20 with 1% ammonium oxalate solution. The diluted sample was charged into the haemocytometer and platelets were counted under the microscope using the high-power objective (40x). The count was calculated and expressed as platelets per litre (x 10^9/L).
6. Quality Assurance All manual counts were performed in duplicate, and the average was recorded. Stained peripheral blood films were prepared, fixed with methanol, and stained with Giemsa stain for microscopic examination. These films were reviewed to confirm cell morphology and to verify any abnormal findings from the manual counts.

Blood Film Preparation and Microscopy

Peripheral blood smears were prepared within 2 hours of collection. Thin films were made on grease-free slides at a 30–45° angle, air-dried, and fixed briefly in methanol. Staining was done with Leishman's stain: the undiluted stain was applied for 2 minutes for fixation, then diluted with twice the volume of buffered distilled water (pH 6.8) and left for 5–7 minutes. Slides were gently washed, dried, and examined microscopically under oil immersion (×100).

During review, the following were assessed:

- Red cell morphology which includes anisocytosis, poikilocytosis, hypochromia, target cells, sickle cells.
- White blood cell differential counts.
- Platelet distribution and clumping.

At least 100 cells were evaluated per film, and findings were documented according to haematology guidelines.

Quality Control

Daily quality control was carried out on the automated analyzer using commercial control samples at low, normal, and high levels, as provided by the manufacturer. Patient samples were only analyzed when control results fell within the acceptable ranges.

Data Collection

Data were collected using a structured proforma. Information recorded included study identification number, age, sex, haemoglobin genotype, and all haematological parameters (from analyzer printouts and blood film review).

Statistical Analysis

Data were first entered into Microsoft Excel and then exported into the Statistical Package for the Social Sciences (SPSS) version 25.0 for analysis. Descriptive statistics (mean, standard deviation, frequencies, and percentages) were used to summarize the data. Haematological parameters were compared across genotype groups using Analysis of Variance (ANOVA) for normally distributed data and the Kruskal–Wallis test for non-normally distributed data. Post-hoc analysis (Tukey's HSD) was applied to identify specific group differences. Statistical significance was considered at $p < 0.05$.

Results

Descriptive Characteristics of the Study Participants

A total of 90 participants were enrolled in the study, with 30 individuals in each haemoglobin genotype group (HbAA, HbAS, HbSS). In the HbAA group, there were 14 males (46.7%) and 16 females (53.3%). The HbAS group showed a slight male predominance, consisting of 16 males (53.3%) and 14 females (46.7%). Conversely, the HbSS group had more females [17 (56.7%)] compared to males [13 (43.3%)]. The mean ages of the participants were comparable across the groups: 40.7 ± 11.7 years for HbAA, 41.1 ± 12.0 years for HbAS, and 40.8 ± 12.9 years for HbSS, indicating a relatively uniform age distribution.

Thus, the study population was well distributed by sex and age across the different genotypes, as presented in Table 1.

Table 2 summarizes the mean haematological parameters of participants across the three haemoglobin genotypes (HbAA, HbAS, HbSS).

The haemoglobin concentration was highest in the HbAA group (13.21 ± 1.16 g/dL), followed by HbAS (12.24 ± 1.18 g/dL), and markedly reduced in HbSS (8.66 ± 1.25 g/dL). A similar trend was observed in red cell indices: the RBC count was highest in HbAA (4.53 ± 0.36 ×10^{12}/L), slightly lower in HbAS (4.03 ± 0.42 ×10^{12}/L), and lowest in HbSS (2.56 ± 0.40 ×10^{12}/L). Haematocrit values followed this same pattern, with HbAA at 39.30 ± 4.05%, HbAS at 36.78 ± 4.12%, and HbSS significantly lower at 26.01 ± 4.88%.

For the red cell indices, mean corpuscular volume (MCV) ranged narrowly across the groups, with HbAS recording the highest (90.19 ± 6.19 fL). The mean corpuscular haemoglobin (MCH) was slightly higher in HbSS (30.25 ± 2.34 pg) compared to HbAA (29.71 ± 1.96 pg) and HbAS (28.89 ± 2.40 pg). MCHC values were similar across the groups, ranging from 33.48 to 33.78 g/dL. Red cell distribution width (RDW) was elevated in HbSS (17.00 ± 1.82%) relative to HbAA (13.82 ± 1.68%) and HbAS (14.13 ± 1.90%).

Regarding leukocyte parameters, total WBC count was highest in HbSS (11.72 ± 4.23 ×10^9/L), compared to HbAS (7.47 ± 2.22 ×10^9/L) and HbAA (6.75 ± 1.58 ×10^9/L). Neutrophils and lymphocytes were also increased in HbSS (6.87 ± 2.69 ×10^9/L and 3.53 ± 1.66 ×10^9/L, respectively) compared to HbAA and HbAS.

Platelet count was elevated in HbSS (416.70 ± 116.44 ×10^9/L), intermediate in HbAS (304.13 ± 84.38 ×10^9/L), and lowest in HbAA (270.87 ± 62.78 ×10^9/L). Reticulocyte count was markedly higher in HbSS (7.84 ± 2.94%) compared to HbAS (2.00 ± 0.85%) and HbAA (1.24 ± 0.36%).

Overall, HbSS participants consistently showed features of anaemia (low Hb, RBC count, and Hct), increased red cell turnover (high RDW and reticulocyte count), and bone marrow response with elevated WBC and platelet counts, as presented in Table 2.

Prevalence of Anaemia by Genotype

Anaemia prevalence was assessed using WHO standards (Hb < 12.0 g/dL for females, Hb < 13.0 g/dL for males).

Among the participants with HbAA genotype, 9 out of 30 (30.0%) were anaemic. In the HbAS group, 18 of the 30 participants (60.0%) had anaemia. All individuals with HbSS genotype were anaemic, representing a prevalence of 100.0%.

This shows a clear gradient in the burden of anaemia, being

lowest in HbAA, higher in HbAS, and universal in HbSS, as presented in Table 3.

Table 1: Age and Sex Distribution of Participants by Genotype

Genotype	Total Number	Male n (%)	Female n (%)	Mean Age (Years) ± SD
HbAA	30	14 (46.7%)	16 (53.3%)	40.7 ± 11.7
HbAS	30	16 (53.3%)	14 (46.7%)	41.1 ± 12.0
HbSS	30	13 (43.3%)	17 (56.7%)	40.8 ± 12.9

Table 2: Mean Haematological Parameters of Participants by Genotype

Parameter	HbAA (Mean ± SD)	HbAS (Mean ± SD)	HbSS (Mean ± SD)
Haemoglobin (g/dL)	13.21 ± 1.16	12.24 ± 1.18	8.66 ± 1.25
RBC Count ($x10^{12}$/L)	4.53 ± 0.36	4.03 ± 0.42	2.56 ± 0.40
Haematocrit (%)	39.30 ± 4.05	36.78 ± 4.12	26.01 ± 4.88
MCV (fL)	87.46 ± 5.64	90.19 ± 6.19	88.16 ± 5.77
MCH (pg)	29.71 ± 1.96	28.89 ± 2.40	30.25 ± 2.34
MCHC (g/dL)	33.48 ± 1.15	33.69 ± 1.09	33.78 ± 0.86
RDW (%)	13.82 ± 1.68	14.13 ± 1.90	17.00 ± 1.82
WBC Count ($x10^9$/L)	6.75 ± 1.58	7.47 ± 2.22	11.72 ± 4.23
Neutrophils ($x10^9$/L)	3.65 ± 0.95	3.96 ± 1.32	6.87 ± 2.69
Lymphocytes ($x10^9$/L)	2.38 ± 0.68	2.68 ± 1.17	3.53 ± 1.66
Platelet Count ($x10^9$/L)	270.87 ± 62.78	304.13 ± 84.38	416.70 ± 116.44
Reticulocyte Count (%)	1.24 ± 0.36	2.00 ± 0.85	7.84 ± 2.94

Table 3: Prevalence of Anaemia among Participants by Genotype

Genotype	Total Participants	Number with Anaemia	Percentage with Anaemia (%)
HbAA	30	9	30.0%
HbAS	30	18	60.0%
HbSS	30	30	100.0%

Discussion and Conclusion

Discussion

This study explored how different haemoglobin genotypes affect blood parameters in individuals within Enugu metropolis. The findings revealed marked differences, most notably between HbSS and HbAA genotypes. Individuals with HbSS had significant changes in their blood profile, the most obvious being severe anaemia. These findings, when considered together, suggest a chronic disease state in HbSS. In contrast, HbAS although slightly lower than HbAA, was still within normal limits, supporting the classification of HbAS as a largely benign carrier state.

The anaemia in HbSS observed in this study is mainly due to chronic haemolysis, which is already known to be the hallmark of sickle cell disease. Our finding is in line with the established pathophysiology of the disorder, where polymerization of haemoglobin S under low oxygen tension produces rigid, sickled red cells that are prematurely destroyed [14]. The mean haemoglobin value recorded here agrees with previous Nigerian studies that reported values between 5.2–9.2 g/dL in steady-state HbSS patients [15], and also matches [16], who reported values often below 8 g/dL. The normocytic and normochromic pattern of the anaemia confirms the haemolytic nature rather than a deficiency anaemia [17]. In addition, the elevated RDW and high reticulocyte count observed in our study further emphasize that the bone marrow was actively producing new red cells in response to the rapid destruction of mature ones. This compensatory erythropoiesis is well documented in HbSS patients, whose red cell lifespan is reduced from 120 days to 10–20 days [18, 10].

The raised white blood cell and platelet count also provide insight into the abnormal state in HbSS. The leucocytosis recorded in our participants, particularly the neutrophil elevation, is consistent with earlier reports. Shet *et al*. (2020) [19] highlighted that activated neutrophils adhere to vascular endothelium and interact with platelets through adhesion molecules such as P-selectin, directly promoting vaso-occlusion and painful crises. Our findings support this explanation, since the leucocytosis in HbSS participants clearly adds to the inflammatory background of the disease. Functional asplenia may further contribute, as it predisposes patients to infections, which in turn stimulates higher white blood cell production.

Similarly, the high platelet count we found in HbSS patients supports the work of Obiechina *et al*. (2020) [20], who reported that over half of HbSS patients in Abuja had platelet counts above 450 × 10^9/L [20]. This thrombocytosis is usually linked to autosplenectomy, where the spleen becomes fibrotic and loses its filtering function [21]. Since platelets are no longer adequately sequestered, their levels remain high. In addition to the raised counts, studies also show that these platelets are hyperactive, increasing the risk of clot formation and complications such as stroke [19]. Our study, therefore, confirms that thrombocytosis in HbSS is not just a numerical abnormality but contributes to the hypercoagulable state of the disease.

In contrast, the haematological profile of HbAS individuals in this study was mostly comparable to that of HbAA. Although their mean haemoglobin was slightly lower than that of HbAA, it was still within the normal physiological range for Nigerians. Ajayi *et al*. (2017) [22] reported similar results, with HbAS individuals having 12.67 g/dL on average. This agrees with the known carrier status of HbAS, where the presence of more than 50% normal haemoglobin A prevents the widespread sickling seen in HbSS [18]. In line with Fasola [10], our findings confirm that HbAS individuals under steady conditions remain clinically stable and asymptomatic. It is, however, important to note that under extreme hypoxic stress, complications can occasionally occur, though they are rare.

Conclusion

The findings from this study have reinforced the heavy haematological burden carried by individuals with homozygous sickle cell disease (HbSS). Severe haemolytic anaemia, together with leucocytosis and thrombocytosis, confirms the chronic, inflammatory, and hypercoagulable state of the condition. In contrast, the HbAS genotype showed near-normal findings, highlighting its benign nature. Overall, these results not only agree with established knowledge but also provide data specific to Enugu Metropolis. This underlines the importance of genetic counselling, prenatal screening, and routine haematological monitoring as essential aspects of managing sickle cell disease in Nigeria.

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