Coinheritance Of Glucose-6-Phosphate Dehydrogenase Deficiency In Sickle Cell Anemia Patients: Impact On Clinical, Biochemical And Hematological Parameters

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Key words:

G6PD deficiency, Hemoglobin electrophoresis, Bilirubin, Saudi Arabia, Co-inheritance, Red blood cell disorders, Genetics.

Abstract

Background: Sickle cell disease (SCD) and glucose-6-phosphate dehydrogenase (G6PD) deficiency are among the most common hereditary hemolytic disorders globally, often co-existing in populations where malaria was historically endemic. The co-inheritance remains controversial, with limited data from regions with high prevalence such as the Eastern Province of Saudi Arabia. This study aimed to evaluate the clinical, hematological, and biochemical differences between patients with SCD alone and those with co-inherited G6PD deficiency (SCD/G6PDD), with a focus on fetal hemoglobin (Hb F), hemolysis markers, and disease severity indicators.

Methods: A cross-sectional analytical study was conducted at the Center of Genetic Blood Diseases, Al-Ahsa, Saudi Arabia, including 169 confirmed SCD patients—98 with SCD only and 71 with SCD/G6PDD. Clinical data were collected via validated questionnaires and chart reviews. Hematological and biochemical analyses included complete blood count, hemoglobin electrophoresis, liver enzymes, and bilirubin levels. Statistical analysis was performed using SPSS version 28, with significance set at $p<0.05$.

Results: No significant differences were observed between the two groups regarding vaso-occlusive crisis frequency, hospitalizations, or hydroxyurea usage. Hematological indices such as hemoglobin, hematocrit, MCV, and platelet counts were comparable. However, the SCD/G6PDD group exhibited significantly higher total bilirubin levels ($p=0.013$) and Hb F levels (mean 37.32% vs. 27.06%, $p=0.018$), while hemoglobin S was significantly lower ($p=0.008$). Liver enzymes and minor hemoglobin (HbA1, HbA2) showed no significant variation.

Conclusion: Co-inheritance of G6PD deficiency in SCD patients does not substantially alter clinical severity but is associated with increased Hb F and bilirubin, possibly indicating a protective hematologic effect. These findings highlight the need for genotype-based management strategies and underscore the importance of further genomic and longitudinal studies in similar populations.

Introduction

Sickle cell disease (SCD) is a common hereditary disorder of hemoglobin, especially prevalent in sub-Saharan Africa, the Middle East and parts of the Indian subcontinent [1]. It is caused by a single nucleotide substitution in the β-globin gene, resulting in the synthesis of hemoglobin S (Hb S). In low oxygen conditions, Hb S molecules tend to aggregate, distorting red blood cells into a sickle shape. These abnormally shaped cells contribute to chronic hemolysis, increased blood viscosity, and recurrent episodes of vaso-occlusion, which are key features of the disease and lead to intense pain, multiorgan complications, and a reduced lifespan [2]. At the same time, glucose-6-phosphate dehydrogenase deficiency(G6PDD) is the most prevalent enzyme

disorder worldwide, affecting over 400 million individuals [3]. G6PD is vital for generating NADPH via the pentose phosphate pathway, which helps neutralize oxidative damage in red blood cells. In G6PD-deficient individuals, exposure to oxidative agents such as specific medications, infections, or fava beans can result in red cell destruction and hemolytic episodes [4]. Both SCD and G6PD deficiency are thought to provide partial protection against malaria, which may explain their high frequencies in regions where malaria has historically been endemic [5,6]. The co-inheritance of these two distinct yet clinically overlapping hemolytic disorders presents a unique challenge [7]. Both conditions independently contribute to red blood cell destruction, raising questions about whether their combined presence exacerbates clinical severity, alters hematological parameters, or modifies biochemical markers of hemolysis. However, the existing literature on this co-inheritance is complex and often contradictory. Some studies suggest a more severe clinical course with increased hemolytic crises and transfusions [8], while others report a milder phenotype, possibly due to the protective effect of increased fetal hemoglobin (HbF) production or a subtle modulation of red cell lifespan [9, 10]. These discrepancies are likely attributable to variations in study populations, genetic backgrounds (different G6PD variants and SCD genotypes), environmental factors, and healthcare access [11]. The Eastern Province of Saudi Arabia is recognized as a region with a remarkably high prevalence of both SCD and G6PD deficiency, making its population an ideal cohort to study their co-inheritance [12, 13]. Despite this epidemiological significance, there is a relative paucity of local studies that have systematically investigated the clinical, hematological, and biochemical ramifications of G6PD deficiency in Saudi patients with SCD. Understanding these interactions is crucial for optimizing patient management and developing region-specific clinical guidelines. Therefore, this study was designed to rigorously explore the impact of co-inherited G6PD deficiency on the clinical presentation, biochemical profile, and hematological indices of Saudi patients diagnosed with SCD. By providing detailed insights into this dual burden, we aim to contribute to a more nuanced understanding of SCD pathogenesis and inform more individualized therapeutic approaches in this specific patient population.

Methods

A cross-sectional analytical study was carried out at the Center of Genetic Blood Diseases in Al-Ahsa, Eastern Province, Saudi Arabia. The research spanned from June to November 2023 and received ethical clearance from the Research Ethics Committee of the College of Medicine at King Faisal University (KFU), under approval number KFU-REC-2023-MAR-ETHICS689. All methodologies adhered to institutional ethical standards. The study population included patients with Sickle Cell Anemia (SCA), either alone or in combination with Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency. Participants were recruited via simple random sampling to reduce the potential for selection bias. The required sample size was calculated based on the prevalence of SCA using the conventional formula for cross-sectional studies. A total of 169 patients were ultimately enrolled: 98 with isolated SCA and 71 with coexisting SCA and G6PD deficiency. Inclusion criteria encompassed individuals diagnosed with SCA, with or without G6PD deficiency, and free from other chronic illnesses or additional hemoglobinopathies. Exclusion criteria included the presence of thalassemia, other hemolytic anemias, hemoglobin variants, or any chronic condition that might confound laboratory findings. Data collection included demographic, clinical, and laboratory parameters, using a pre-validated structured questionnaire. This form was completed via direct interviews or by reviewing patients' clinical records. All participants, or their legal representatives, signed written informed consent before inclusion in the study.

Venous blood samples (5 mL) were collected under aseptic conditions, following site disinfection using 70% isopropyl alcohol. Collected blood was divided into two tubes based on intended analyses. EDTA tubes were utilized for Complete Blood Count (CBC) and hemoglobin electrophoresis, while serum separator tubes were designated for liver function and bilirubin testing. All samples were processed using standard laboratory protocols. EDTA specimens were stored at 4°C and analyzed promptly. CBC was performed using the Sysmex

automated hematology analyzer to assess red and white cell parameters, hemoglobin levels, and platelet counts. Hemoglobin fractionation was determined using the Bio-Rad Variant II system. Serum samples were allowed to clot for 30 minutes at room temperature and then centrifuged at 4,500 RPM for 10 minutes to obtain clear serum for testing. Liver enzyme levels—including AST, ALT, and ALP as well as total, direct, and indirect bilirubin concentrations were measured using the HITACHI COBAS C 311 automated analyzer. G6PD enzyme activity was assessed through a qualitative manual assay, and results were validated using the UVitec gel documentation system for clarity and accuracy. All data were first entered into Microsoft Excel 2019, where initial validation and cleaning were conducted to ensure consistency and reliability. The final dataset was then imported into IBM SPSS Statistics version 28 for analysis. Descriptive statistics were used to summarize continuous variables as means ± standard deviations. Group comparisons were performed using independent t-tests, and Pearson correlation was applied to evaluate associations between hematological and biochemical markers. A p-value below 0.05 was considered statistically significant for all analyses. Confidentiality of participants' information was strictly maintained, and voluntary participation was ensured through the use of signed consent forms. Ethical oversight was maintained by the Ethics Committee of King Faisal University (KFU-REC-2023-MAR-ETHICS689).

Results

This study analyzed demographic, clinical, hematological, and biochemical differences between patients with sickle cell disease (SCD) and those with coexisting glucose-6-phosphate dehydrogenase deficiency (SCD/G6PDD). A total of 169 patients were evaluated, with findings summarized across four tables and four figures to highlight key trends and statistical comparisons. Table 1 revealed a predominance of males in the study population, accounting for 57.9%, while females comprised 42%. Within the SCD-only group, male patients made up 88.7%, and in the SCD/G6PDD group, males constituted 81.6%, emphasizing gender predominance across both cohorts. Clinical characteristics between the two groups showed no statistically significant differences. Vaso-occlusive crises (VOC) occurred in 52.6% of the SCD group and 54% of the SCD/G6PDD group. Other clinical factors such as recent blood transfusions, hospital admissions, and hydroxyurea therapy usage were also similar ($P > 0.05$), as detailed in Table 2. Hematological evaluations presented in Table 3 showed no meaningful differences between the groups. Parameters including total white blood cell count, red blood cell count, hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and platelet count were comparable across both populations ($P > 0.05$), suggesting uniform hematological profiles. Biochemical assessments in Table 4 highlighted largely comparable liver function parameters between groups, with the exception of total bilirubin (TB), which was significantly elevated in the SCD/G6PDD group ($P = 0.013$). Other markers such as AST, ALT, conjugated bilirubin (CB), and unconjugated bilirubin (UNCB) did not differ significantly ($P > 0.05$). Figure 1 illustrated that fetal hemoglobin (Hb F) levels were notably higher in the SCD/G6PDD group, with a mean of 37.32 compared to 27.06 in the SCD group ($P = 0.018$). The SCD/G6PDD group also exhibited a more consistent distribution, whereas the SCD group showed broader variability and several outliers. Conversely, as shown in Figure 2, hemoglobin S (Hb S) levels were significantly reduced in the SCD/G6PD group (mean 57.69) relative to the SCD group (mean 69.65; $P = 0.008$). The SCD group showed greater spread and higher maximum values. Figures 3 and 4 demonstrated that hemoglobin A1 (HbA1) and hemoglobin A2 (HbA2) levels were not significantly different between the two groups ($P > 0.05$). Both groups exhibited nearly identical means, medians, and interquartile ranges, with minimal variability and only occasional outliers. This indicates that G6PD deficiency had no significant effect on HbA1 or HbA2 concentrations.

Discussion

This study assessed and compared the clinical, hematological, and biochemical characteristics of patients with sickle cell disease (SCD) and those with co-inherited glucose-6-phosphate

dehydrogenase (G6PD) deficiency. While the majority of findings showed comparable profiles between the two groups, certain distinctions emerged that may have biological and clinical implications. The overall male predominance in both cohorts especially pronounced among those with SCD/G6PD can be attributed to the X-linked inheritance pattern of G6PD deficiency, where males carrying the mutated gene are typically affected, while females are often asymptomatic carriers unless homozygous or compound heterozygous [14]. This sex-linked transmission explains the skewed gender distribution consistently observed in similar studies conducted in malaria-endemic regions, where the selective pressure has maintained high frequencies of both SCD and G6PD deficiency [15]. Clinically, both groups demonstrated similar frequencies of vaso-occlusive crises (VOC), recent transfusions, hospital admissions, and hydroxyurea therapy. This aligns with previous investigations that found G6PD deficiency does not necessarily worsen the acute clinical complications of SCD, particularly in patients not exposed to oxidative stressors such as certain infections or drugs [16,17]. However, it is important to consider that while VOC frequencies were not significantly different, this does not rule out subtle variations in pain severity or frequency that may not have been captured in routine clinical assessments [18]. Hematological indices including red blood cell counts, hemoglobin concentration, hematocrit, and red cell indices (MCV, MCH, MCHC) did not differ significantly between the groups, suggesting that G6PD deficiency, under non-stress conditions, does not produce overt hematological derangements in SCD patients [19]. These findings are consistent with other cross-sectional studies that reported stable hematologic profiles in co-inherited patients during the steady state [20]. Biochemically, total bilirubin levels were significantly higher among SCD patients with G6PD deficiency. This elevation likely reflects a subclinical increase in hemolysis due to the vulnerability of G6PD-deficient red blood cells to oxidative stress, even under baseline conditions [21]. In contrast, liver enzymes (ALT and AST) were comparable between groups, reinforcing that the observed hyperbilirubinemia is hemolytic rather than hepatic in origin. Similar biochemical trends have been noted in populations with concurrent hemoglobinopathies and G6PD deficiency, particularly in African and Middle Eastern regions [22]. A key observation in this study was the significantly higher fetal hemoglobin (Hb F) levels in the SCD/G6PD group. Hb F is known to mitigate the polymerization of hemoglobin S, thereby reducing sickling, hemolysis, and clinical severity in SCD [23]. Higher Hb F levels in the co-inherited group may suggest the presence of genetic modifiers or epigenetic regulation that favors gamma-globin gene expression. Several studies have identified loci such as BCL11A, HBS1L-MYB, and KLF1 as influential in regulating Hb F levels, and it is possible that these or unidentified interactions contribute to the elevated Hb F observed in our cohort [24,25]. The reduced levels of hemoglobin S (Hb S) further support this protective effect, potentially contributing to a milder disease phenotype in some individuals, although longitudinal data are needed to validate this hypothesis. Conversely, hemoglobin A1 (HbA1) and A2 (HbA2) levels remained unchanged across both groups. This is expected, as these minor hemoglobins are typically not involved in the pathophysiology of homozygous sickle cell disease, and G6PD deficiency is not known to impact their expression directly [26]. These findings carry potential implications for clinical management. Elevated Hb F in co-inherited individuals could influence their eligibility or response to treatments such as hydroxyurea, which is used to pharmacologically increase Hb F levels [27]. Moreover, the presence of G6PD deficiency should be considered when prescribing drugs or when anticipating hemolytic risk, even in SCD patients with seemingly stable disease. Nevertheless, this study has some limitations. Being cross-sectional, it does not account for seasonal variations, oxidative challenges, or longitudinal changes in clinical severity. Additionally, genotyping for G6PD variants and Hb F modifier loci was not performed, which could have provided more mechanistic insights into the observed patterns.

Conclusion

In conclusion, while SCD patients with or without G6PD deficiency demonstrate broadly similar clinical and laboratory features, the presence of G6PD deficiency is associated with

higher total bilirubin levels and significantly increased fetal hemoglobin. These changes may have protective hematological effects and could influence the clinical trajectory of affected individuals. Understanding these interactions is essential in regions with high prevalence of both conditions, and future genomic and prospective studies are warranted to clarify the biological mechanisms and therapeutic implications.

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Authors Contributions

Ahmed Mohamedain: Conceptualization, visualization, supervision and writing-original manuscript. Khawlah Abdullah AlQabi: Data collection, investigations, laboratory work and results compilation. Ahmed Abdul qader and Nawaf Alkhashram: Research Administration and laboratory setting supervision. Omer Musa and Habib Qureshi contributed to the writing of the draft. Hamdan Z. Hamdan, revision of the methodology and critical reviewing and editing of the final manuscript. Maujid Masood Malik, supervised the laboratory work. Rabab Abbas: Set the criteria for diagnosing and selecting patients. All authors reviewed and approved the final manuscript.

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Table 1: Patients demographic data

Demographic data	Age n(%)	Patients (n=169)	
		SCD n(%)	SCD/G6PD n(%)
Male	98(57.9%)	87(88.7%)	58(81.6%)
Female	71(42.01%)	11(11.2%)	13(18.3%)
Total	169	98	71

SCD: sickle cell disease, G6PD: glucose 6 phosphate deficiency

Table 2: The clinical characteristics of patients

Clinical data	Study groups n(%)		P value
	SCD 98(57.9%)	SCD/G6PD 71(42.01%)	
VOC on OPD presentation	51(52.6 %)	38(54%)	0.850
Blood Transfusion in the last admission	11(11.2%)	13(18.3%)	0.210
Admission in the last year	14(14.28%)	10(14.08%)	0.971
Number of Medications Taken(Hydroxy Urea)	62	43	0.723

P. value ≤ 0.05 is considered to be significant , SCD; sickle cell disease, G6PD; glucose 6 phosphate deficiency, VOC; vaso-occlusive crisis, OPD; Outpatient department

Table 3: The hematology parameters analysis among patients

Laboratory data	Study groups Mean± SD		95% CI	P value
	SCD	SCD/G6PD		
TWBCs /µ	8.1 ±3.1	7.7±3.2	(-0.597-1.371)	0.439
RBCs /µ	3.8 ±0.8	3.7±0.9	(-0.171-0.367)	0.473
HB g/dl	10.3 ±1.7	10.3± 1.6	(-0.497-0.492)	0.993
HCT %	28.9 ±4.5	28.8 ±4.5	(-1.325-1.440)	0.935
MCV /Fl	77.2 ±13.5	80.0±10.3	(-6.565-0.682)	0.111
MCH / Pg	28.0±5.16	28.9±4.4	(-2.383-0.542)	0.216
MCHC %	35.7±1.5	35.7 ±1.8	(-0.562-0.499)	0.908
PLT /µ	350.0±232.9	313.6 ±190.1	(-27.988-100.772)	0.266

P. value ≤ 0.05 is considered to be significant, SCD: sickle cell disease, G6PD: glucose 6 phosphate deficiency, WBCs; white blood cells count, RBCs; red blood cells count, HB; hemoglobin, HCT; hematocrit, MCV; mean cell volume, mean cell concentration, MCHC; mean cell hemoglobin concentration.

Table 4: The biochemistry parameters analysis among patients

Laboratory data	Study groups Mean± SD		95% CI	P. value
	SCD	SCD/G6PD		
AST U/L	31.2± 13.2	33.6 ± 15.3	(-6.908-1.980)	0.275
ALT U/L	19.6± 11.1	24.8± 29.3	(-12.428-2.087)	0.160
TB mg/dL	32.9± 21.5	44.5± 34.3	(-20.751- -2.458)	0.013
CB mg/dL	10.3± 9.2	12.4± 11.9	(-5.503-1.223)	0.12
UNCB mg/dL	30.8± 30.8	42.1± 40.9	(-22.649-0.191)	0.054

P. value ≤ 0.05 is considered to be significant, SCD: sickle cell disease, G6PD: glucose 6 phosphate deficiency, AST; Aspartate Aminotransferase, ALT; Alanine Aminotransferase, TB; Total Bilirubin, CB; Conjugated (Direct) Bilirubin, UNCB; Unconjugated (Indirect) Bilirubin

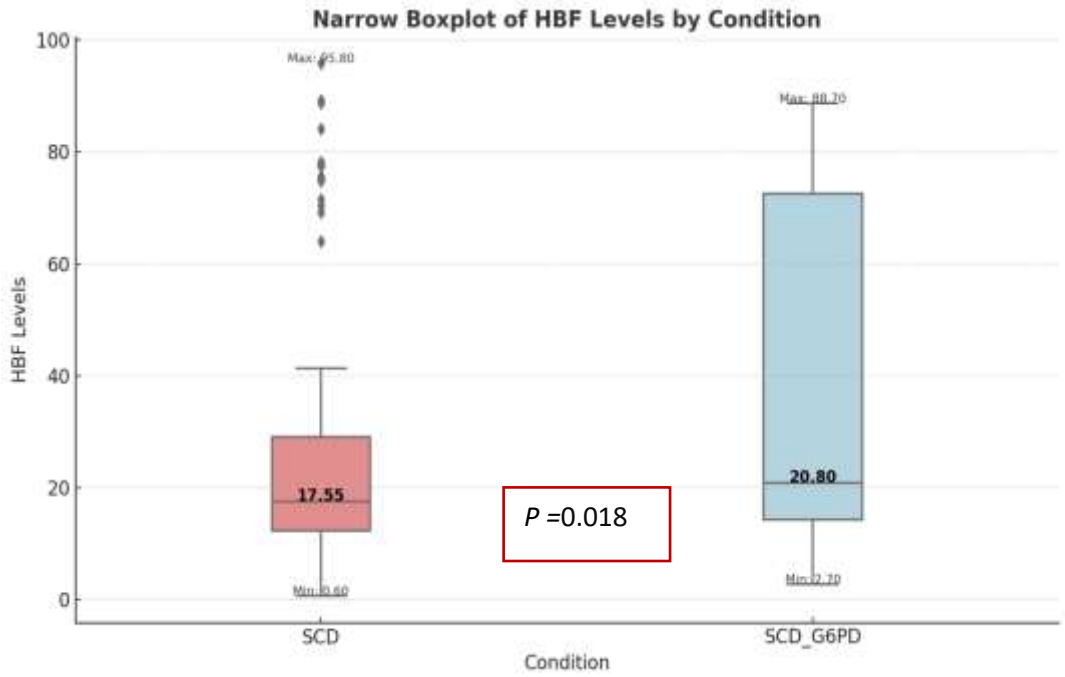

Figure 1: The association of SCD and SCD/G6PD with Hb F levels

This figure illustrates a statistically significant difference (P = 0.018) in fetal hemoglobin (Hb F) levels between individuals with sickle cell disease (SCD) and those with both SCD and glucose-6-phosphate dehydrogenase (G6PD) deficiency.

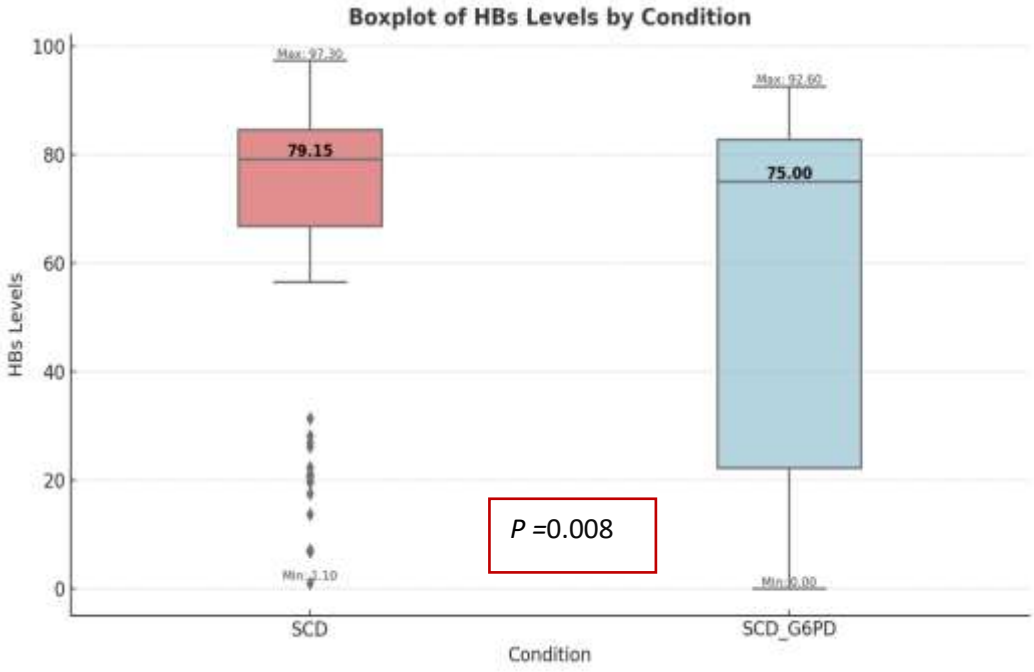

Figure 2: The association of SCD and SCD/G6PD with Hb S levels

This figure shows a statistically significant association (P = 0.008) between hemoglobin S (Hb S) levels in individuals with SCD compared to those with concurrent G6PD deficiency.

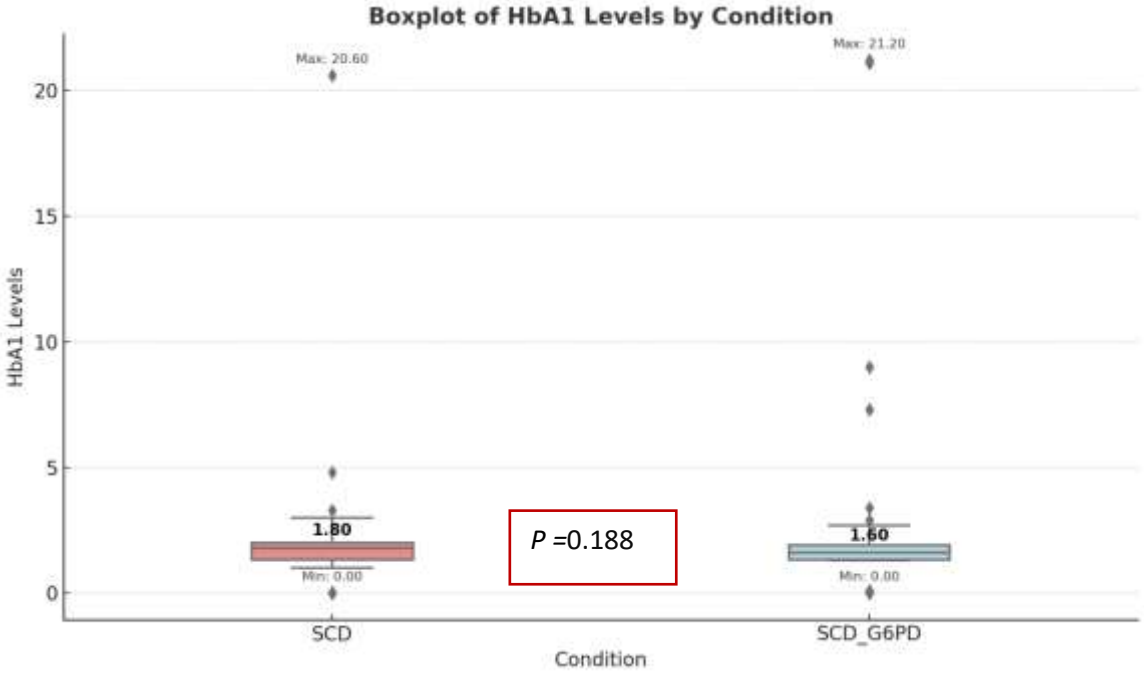

Figure 3: The association of SCD and SCD/G6PD with Hb A1 levels

This figure presents data on hemoglobin A1 (Hb A1) levels in both groups. The difference is not statistically significant (P = 0.188).

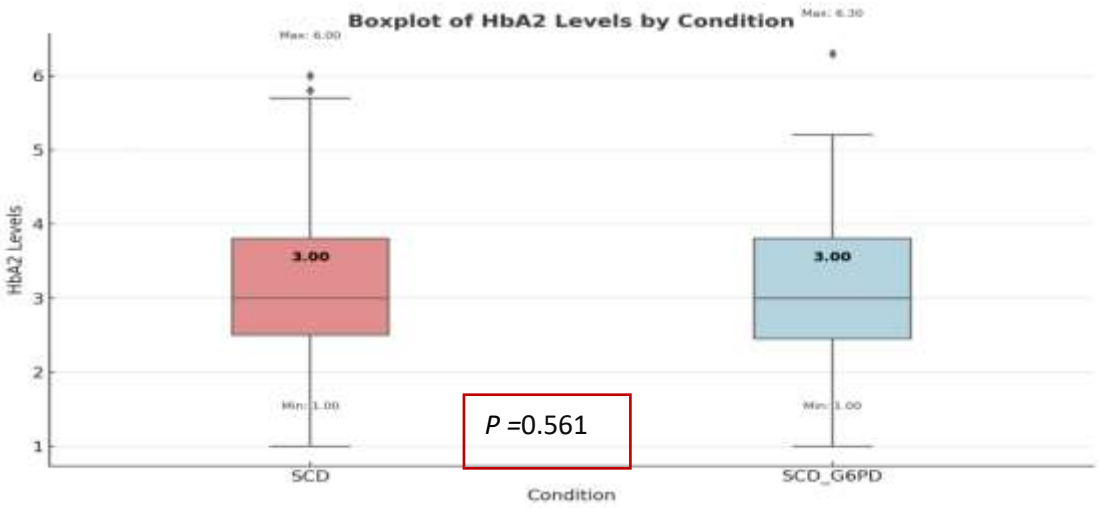

Figure 4: The association of SCD and SCD/G6PD with Hb A2 levels

This figure compares hemoglobin A2 (Hb A2) levels between SCD and SCD/G6PD groups, showing no significant difference (P = 0.561).