CORRELATION OF SERUM MYOGENIN WITH DISEASE SEVERITY IN SUBJECTS WITH SICKLE CELL DISEASE IN STEADY STATE

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and body mass index (BMI) calculated. Disease severity in HbSS participants was evaluated using the Adegoke and Kuti sickle cell disease severity scoring system. Individuals with cardiovascular disease, inflammatory disorders, renal disease, obesity, pregnancy, or metabolic conditions were excluded. Statistical analyses included ANOVA and correlation testing to evaluate group differences and relationships between variables.

Results: HbSS participants had significantly lower BMI compared to HbAA and HbAS groups ($p = 0.001$), despite no significant difference in age across groups. However, serum myogenin levels did not differ significantly among HbAA, HbAS, and HbSS participants ($p = 0.967$). Correlation analysis showed no significant association between myogenin and BMI ($r = 0.017$, $p = 0.931$) or age ($r = 0.003$, $p = 0.986$), although BMI was strongly positively correlated with age ($r = 0.801$, $p = 0.001$). Notably, there was a statistically significant moderate inverse correlation between myogenin levels and disease severity in HbSS patients ($r = –0.387$, $p = 0.035$).

Conclusion: This study demonstrated that while individuals with SCD exhibit significant nutritional deficits, as reflected by reduced BMI, these do not directly influence circulating myogenin levels. The observed inverse relationship between myogenin and disease severity may suggest that myogenin may serve as a marker of disease-related muscle dysfunction rather than genotype or nutritional status.

Recommendations: Further longitudinal and mechanistic studies are needed to validate myogenin as a biomarker and explore its potential role in therapeutic strategies targeting muscle health in SCD.

Key words: Sickle cell anaemia, Sickle cell disease, myogenin, biomarkers, body mass index.

INTRODUCTION

Sickle cell disease (SCD) represents one of the most prevalent monogenic disorders globally, affecting approximately 50 million people worldwide, with Nigeria bearing the highest burden of 4-6 million cases.[1] The disease results from a point mutation in the β-globin gene, leading to the production of abnormal hemoglobin S (HbS), which polymerizes under deoxygenated conditions, causing red blood cells to assume a characteristic sickle shape.[2]

The clinical manifestations of SCD extend beyond hematological complications, significantly impacting multiple organ systems, including the musculoskeletal system.[3] While vaso-occlusive crises and chronic hemolysis are well-documented pathophysiological mechanisms, the role of muscle-specific factors in disease progression and severity remains poorly understood.[4] This knowledge gap is particularly significant given that muscle wasting, and reduced muscle mass are common complications in SCD patients, contributing to reduced quality of life and increased morbidity.

Myogenin, a muscle-specific transcription factor belonging to the MyoD family, plays a crucial role in skeletal muscle development, repair, and regeneration. Previous studies have demonstrated elevated

myogenin expression in various muscle-wasting conditions and skeletal myopathies, suggesting its potential role as a compensatory mechanism in muscle repair.[5] However, despite the prevalence of muscle-related complications in SCD, no previous studies have investigated the relationship between myogenin level and disease severity in SCD patients.

This study aimed to bridge this critical knowledge gap by examining the relationship between serum myogenin level and disease severity in patients with homozygous sickle cell disease (HbSS). Understanding this relationship could provide new insights into disease progression mechanisms and potentially identify novel therapeutic targets for managing SCD-related muscle complications.

MATERIALS AND METHODS

Study Site This research was carried out in Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi, Anambra State, Nigeria.

Study Design: A cross-sectional comparative study was carried out to evaluate the serum level of myogenin and its correlation with disease severity in subjects with sickle cell disease in their steady state. Selection of sickle cell subjects as well as the control group (HbAS and HbAA) was done by simple random sampling method. A total of 90 subjects were recruited for the study which includes 30 homozygous sickle cell (HbSS) subjects, 30 heterozygous sickle cell (HbAS) and 30 normal subjects (HbAA). The selection of the steady state group was dependent on subjects not experiencing crisis for at least 2 weeks; not receiving blood transfusion for at least 3 months, and not having fever for at least 2 weeks prior to the study. Full blood count results of the subjects were used alongside other criteria, to estimate severity scoring in homozygous sickle cell disorder in steady state and crisis.

Sample Size Determination

Sample size was calculated using the Cochran's Formula as given below:

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Where n= sample size; Z = 1.96 (95% confidence level); p = 0.04 (SCD prevalence in Nigeria = 4.0% [6] and d = 0.05 (absolute error or precision level).

$$n = \frac{1.96^2 * 0.04(1 - 0.04)}{0.05^2}$$

n= 59.00

However, in order to increase the statistical power of the study, a total of 90 participants were recruited into the study using simple random sampling method. Participants were comprised of 30 homozygous sickle cell (HbSS) subjects, 30 heterozygous sickle cell (HbAS) and 30 normal subjects (HbAA).

Inclusion Criteria: Homozygous sickle cell disease (HbSS) subjects in steady state, heterozygous sickle cell subjects (HbAS) and normal healthy subjects (HbAA) all within the age range 10-50 years.

Exclusion Criteria: Subjects outside the age range of 10-50 years, individuals with known cardiovascular and inflammatory disease (autoimmune disease), obesity, renal disease, pregnancy, metabolic disorders such

as diabetes, subjects on medication such as non-steroidal anti-inflammatory drugs (NSAIDs).

Ethical Approval: The ethical approval for this research was obtained from Nnamdi Azikiwe University Teaching Hospital Ethics Committee in agreement with the Helsinki declaration by the World Medical Association (WMA) on the ethical principles for medical research involving human subjects.[7]

Informed consent: Prior to the study, written informed consent from the subjects and or their guardians (for minors) was obtained.

Sample Collection Blood samples were collected aseptically, and serum was separated and stored at -20°C until analysis. Hemoglobin genotype was determined using cellulose acetate electrophoresis.[8]

Parameters Assayed: Serum myogenin levels were measured using an enzyme-linked immunosorbent assay (ELISA). Disease severity in HbSS participants was evaluated using the Adegoke and Kuti sickle cell disease severity scoring system based on anemia, complications, white blood cell counts, transfusion rates, and crisis frequency in line with prior studies.[9]

Body mass index (BMI) measurement: BMI was calculated using the following formula[10,11] as given below:

$$BMI = \frac{\text{Weight}(kg)}{Height^2\,(m^2)}$$

A measuring tape was fastened to a piece of wood to determine height, and an electronic weighing scale was used to estimate the body weight.

Statistical Analysis The data were presented as mean±SD and the mean values of the control and test group were compared by Analysis of Variance (ANOVA), posthoc test and Pearson's correlation coefficient using Statistical package for social sciences (SPSS) (Version 26.0) software. Statistical significance was set at $p < 0.05$.

RESULTS

Table 1 show the mean anthropometric values in different blood genotype groups (HbAA, HbAS and HbSS). The One-way ANOVA revealed no statistically significant difference in the mean age between groups ($F = 0.525$; $p = 0.593$). However, a significant difference was observed in the BMI ($F = 16.927$; $p=0.001$) when compared across the groups. The BMI did not differ significantly in the HbAA participants when compared to the HbAS group ($p = 0.906$), but the BMI was significantly decreased in sickle cell patients when compared with HbAA and HbAS subjects ($p = 0.001$), respectively.

Table 2 shows the mean level of Myogenin in subjects with homozygous sickle cell disease (HbSS) and the control groups (HbAA and HbAS). There was no significant difference in the mean serum level of myogenin in the different hemoglobin phenotype groups ($F = 0.034$; $p = 0.967$). No significant difference was observed in the mean level of myogenin between HbAA and HbAS subjects ($p = 0.963$), between HbAA and HbSS ($p = 0.989$) and between HbAS and HbSS ($p = 0.992$).

Table 3 shows the correlation of BMI, age and Myogenin with disease severity in subjects with sickle cell anemia in steady state. No correlation was observed between BMI and Myogenin in sickle cell anemic patients (r = 0.017, p = 0.931). A positive correlation was observed between BMI and age in sickle cell anemic patients (r = 0.801, p = 0.001). No correlation was observed also between age and Myogenin in sickle cell anemic patients (r = 0.003, p = 0.986).

Table 4 shows the correlation of BMI, age and Myogenin with disease severity in subjects with sickle cell anemia in steady state**.** No correlation was observed between disease severity and Age (r = -0.068, p = 0.719) and between disease severity and BMI (r = -0.174, p = 0.356) in sickle cell anemic patients. A negative correlation was observed between disease severity and Myogenin level (r = -0.387, p = 0.035).

Table 1: Anthropometric values in different blood genotype (HbSS, HbAA and HbAS)

Groups	N	Age(years)	BMI (kg/m^2)
AA	30	25.03 ± 5.72	23.81 ± 2.77
AS	30	23.50 ± 4.54	23.49 ± 2.19
SS	30	24.93 ± 8.52	19.90 ± 3.52
F-value		0.525	16.927
P-value		0.593	0.001*
AA vs AS (p-value)		0.632	0.906
AA vs SS (p-value)		0.998	0.001*
AS vs SS (p-value)		0.669	0.001*

**P*-value is statistically significant at <0.05.

Table 2: Mean level of Myogenin in different haemoglobin phenotype groups (HbSS, HbAA and HbAS)

Groups	Myogenin
AA (n=30)	31.70 ± 9.13
AS (n=30)	31.97 ± 6.00
SS (n=30)	31.47 ±6.98
F-value	0.034
P-value	0.967
AA vs AS (p-value)	0.963
AA vs SS (p-value)	0.989
AS vs SS (p-value)	0.992

**P*-value is statistically significant at <0.05.

Table 3: Correlation of anthropometric values (BMI and Age) with Myogenin in subjects with sickle cell anaemia in steady state

Variable	N	r- value	p-value
BMI VS MYOGENIN	30	0.017	0.931
BMI VS AGE	30	0.801**	0.001*
AGE VS MYOGENIN	30	0.003	0.986

**P*-value is statistically significant at <0.05.

Table 4: Correlation of Myogenin, BMI and Age with disease severity in subjects with sickle cell anaemia in steady state

Variable	N	r- Value	p-value
Disease Severity Vs Myogenin	30	-0.387*	0.035*
Disease Severity Vs Age	30	-0.068	0.719
Disease Severity VS BMI	30	-0.174	0.356

**P*-value is statistically significant at <0.05.

DISCUSSION

Sickle cell disease (HbSS) is an autosomal recessive genetic disorder of the red blood cell.[12] affecting approximately 1-3% of populations within the states in Nigeria.[13] Increasing evidence points towards oxidative stress, vasooclusion and the resulting inflammatory response as responsible for the increased organ and system involvement in sickle cell disease.[14] The activity and expression of myogenin has been shown to be controlled by the balance between the pro-inflammatory and anti-inflammatory cytokines and these factors are

shown to be induced in sickle cell anemia. Skeletal muscle hypertrophy and hypotrophy have been documented to occur due to imbalance between these group of cytokines.[15]

This study examined anthropometric characteristics, myogenin levels, and their association with disease severity in individuals with sickle cell disease (SCD) in steady state. While our findings provide useful insights, a more critical evaluation highlights both agreements and inconsistencies with existing literature, particularly regarding the complex regulation of muscle regeneration in chronic disease.

The anthropometric findings (Table 1) showed that individuals with HbSS had significantly lower BMI compared to HbAA and HbAS groups ($p = 0.001$), despite similar age distribution. This is consistent with established evidence that SCD is associated with chronic undernutrition, increased resting energy expenditure or metabolic demand, and recurrent illness, all of which contribute to reduced body mass and impaired growth. However, a critical observation is that despite this significant reduction in BMI, there was no corresponding alteration in myogenin levels (Table 2) or correlation with BMI (Table 3). This contrasts with evidence from muscle-wasting conditions where nutritional deficits and catabolic states are often associated with dysregulation of myogenic regulatory factors, including myogenin.[16, 17] The lack of association observed in this study suggests that BMI may be an insufficient proxy for muscle mass or muscle metabolic activity, and therefore may not adequately reflect the biological processes regulating myogenin expression. It also raises the possibility that in steady-state SCD, nutritional deficits alone are not sufficient to disrupt muscle differentiation pathways.

The lack of significant difference in myogenin levels across haemoglobin phenotypes (Table 2) also warrants critical consideration. While this suggests that genotype alone does not influence myogenin expression, it contrasts with findings in chronic inflammatory and degenerative muscle conditions, where myogenin is often altered as part of a regenerative response.[18,19] One possible explanation is that the study population was in a steady state, where acute inflammatory or hypoxic triggers that typically activate myogenic pathways are minimal. Another possibility is methodological, suggesting that circulating myogenin may not accurately reflect tissue-level activity, where most myogenic processes occur. This limitation has been highlighted in studies showing that myogenin expression is tightly regulated within muscle tissue and may not be reliably detected in systemic circulation.[20]

The correlation analysis (Table 3) revealed no relationship between myogenin and BMI or age, despite a strong correlation between BMI and age. This finding reinforces the concept that myogenin reflects localized cellular processes such as satellite cell differentiation rather than systemic or demographic variables.[20] However, this also limits its clinical applicability, as it suggests that myogenin cannot be easily inferred from routine clinical parameters. Furthermore, ageing is known to impair muscle regenerative capacity and reduce

expression of myogenic markers under stress conditions.[21,22] The absence of such an effect in this study may reflect the relatively young population studied of our participants, or maybe due to the insufficient age variability in our sampled population.

The most significant finding of this study is the inverse relationship between myogenin and disease severity (Table 4). This suggests that as disease severity increases, myogenin levels decrease, indicating impaired muscle regenerative capacity. This is supported by mechanistic studies showing that chronic inflammation and inflammatory mediators such as TNF-α can suppress myogenin expression and inhibit myogenic differentiation.[23] Similarly, reduced myogenin expression has been associated with impaired muscle repair and muscle wasting in chronic disease states.[16,24]

However, this finding is not entirely consistent across the literature. Some studies report increased myogenin expression in response to muscle injury, reflecting an adaptive or compensatory mechanism response.[18,25] This apparent contradiction may be explained by disease stage. For example, in early or moderate disease, myogenin may be upregulated to promote repair, whereas in chronic or severe disease, persistent inflammation and repeated injury may exhaust regenerative capacity, leading to reduced expression. This concept is supported by studies showing that in prolonged disease states, muscle regeneration becomes ineffective despite ongoing damage.[26,27] Therefore, the negative correlation observed in this study likely reflects advanced impairment of muscle regenerative signalling in more severe SCD.

Another critical limitation is that myogenin is primarily a tissue-specific marker, and its measurement in serum may not accurately represent its functional activity within muscle. This methodological issue may partly explain the weak or absent associations with BMI, age, and genotype. This is because some studies have shown that direct tissue-level assessment provides more reliable insight into myogenic activity[20], suggesting that future research should incorporate muscle biopsies or even more sensitive molecular techniques.

Generally, our findings indicate that while HbSS individuals exhibit clear nutritional deficits (Table 1), these do not directly influence myogenin levels, and genotype alone is not a determinant of myogenin expression (Table 2). Instead, myogenin appears to be more closely linked to disease burden and chronic pathophysiological stress, as reflected in its inverse association with disease severity (Table 4). This suggests that myogenin may serve as a marker of functional muscle impairment rather than structural or nutritional status.

Conclusion

This study provides important insight into the relationship between myogenin and disease severity in sickle cell disease. While significant reductions in BMI were observed in HbSS individuals, these did not correspond to changes in myogenin levels, highlighting the limitation of anthropometric measures in reflecting muscle biology. Critically, the study demonstrates a significant inverse relationship between myogenin and disease severity, suggesting that worsening SCD is associated with

impaired muscle regenerative capacity. When interpreted alongside existing evidence, our finding suggests progressive exhaustion of muscle repair mechanisms under chronic inflammatory and hypoxic stress. Hence, our study challenges the assumption that myogenin is influenced by genotype or nutritional status alone and instead positions it as a potential marker of disease-related muscle dysfunction. However, its clinical utility is limited by methodological constraints and variability in its expression across disease states. Therefore, we recommend that future studies should adopt longitudinal designs and integrate molecular, functional, and inflammatory markers to better define the role of myogenin in SCD progression and its potential as a therapeutic target.

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