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Plasma Lipid Levels in Relation to Disease Severity in Sickle Cell Anaemia in Abakaliki, Southeast Nigeria

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Abstract

Background: Dyslipidaemia has been implicated in the pathophysiology of sickle cell disease (SCD) complications; hence its role requires further elucidation.
Objectives: To investigate the relationship between disease severity and plasma lipid levels of patients with sickle cell anaemia.
Methods: A cross-sectional study design was used for the survey. A total of 50 patients with sickle cell anaemia and 50 controls without SCD were recruited for the study. The clinical data and plasma lipid levels of lipids and haemoglobin parameters were analysed.
Results: The majority of the participants were aged 18-25 years. Total plasma cholesterol and HDL-C were significantly lower in individuals with SCA compared with the controls (3.3±1.2 vs 4.2±1.2; p<0.001) and (1.3±0.5 vs 1.5±0.4; p = 0.038) respectively. Most patients with SCA had moderate disease severity (24; 48%). There was no statistically significant difference in the plasma levels of total cholesterol and HDL-C across the disease severity groups of SCA (p = 0.694 and 0.262). There was also no significant correlation between total cholesterol, HDL-C, and markers of haemolysis, haemoglobin F, and haemoglobin S levels.
Conclusion: SCA is characterised by lower mean plasma TC and HDL than controls. However, no relationship was found between TC, HDL levels and SCD disease severity, markers of haemolysis, HbF and HbS levels. Further studies are required to ascertain the implications of plasma lipid levels in SCD.

Keywords: Cholesterol, Haemoglobinopathy, High-Density Lipoprotein-Cholesterol, Sickle Cell Disease, Total Cholesterol.

Introduction

Sickle cell anaemia (SCA) is a form of haemoglobinopathy due to substituting glutamic acid with valine at position 6 of the β-globin chain, leading to the synthesis of haemoglobin S (HbS). [1] The disease is characterised by red cell rigidity, poor tissue perfusion with attendant hypoxia, haemolysis, chronic inflammation, and multiple organ damage. [2-3] It is a known cause of reduced life expectancy in developing countries due to the lack of resources for adequately
managing this disorder. The underlying pathophysiology in sickle cell anaemia, which leads to diverse clinical presentations, is the polymerisation of HbS, which distorts the red cell membrane architecture. Other mechanisms include impaired biorheology and increased adhesion-mediated vaso-occlusion, haemolysis-mediated endothelial dysfunction, and sterile inflammation with elaboration of inflammatory molecules.

Cholesterol is critical for steroid synthesis, cellular membrane formation and bile acid synthesis. Cholesterol is a red cell membrane component, contributing to its flexibility. Chylomicrons (CM), very low-density lipoprotein cholesterol (VLDL-C), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) are produced as a result of cholesterol being delivered to tissues in apolipoprotein-packaged form. Dyslipidaemia refers to lipid metabolism disorders, which could be an elevation or decrease in plasma concentration of lipoproteins. They are related to various pathogenic states with particular affectation of the cardiovascular system. Dyslipidaemia may be primary from a genetic cause or secondary to systemic disease, diet or drugs. Abnormal increase in LDL-C is a known risk factor in cardiovascular disease as it leads to premature atherosclerotic changes of vessels. At the same time, HDL-C is anti-atherogenic and anti-inflammatory and primarily functions to transport cholesterol from the peripheral tissues to the liver, playing a role in the biodistribution of lipids. Cholesterol has been established to play a central role in atherosclerosis, which has mechanisms similar to those observed in vasculopathy of sickle cell anaemia, including prevention of the release of nitric oxide, dysfunctional endothelial function, platelet aggregation and activity. Atherosclerosis is characterised by increased accumulation of cholesterol in arterial wall macrophages, but atheromas are not found in SCD; instead, the vasculopathy in SCD is due to lack of nitric oxide following intravascular haemolysis and release of arginase. The features of SCD with haemolysis-vasculopathy-associated complications include pulmonary hypertension, stroke, and priapism, which are manifestations of SCA associated with vasculopathy.

Disease severity in SCA helps to prognosticate and stratify patient management. Some plasma lipids have been identified in previous studies to impact significantly on the severity of SCD. Dyslipidaemia has been associated with sickle cell anaemia as a possible causative factor in the haemolysis and inflammatory processes observed in the condition, with attendant morbidity and mortality. The lipid profile in sickle cell anaemia is typified by low levels of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) but increased levels of triglycerides (TG) levels. Dyslipidaemia is a risk factor for coronary artery disease and pulmonary hypertension in sickle cell disease and requires more evaluation.

Literature on the status of plasma lipids in patients with SCD needs to be improved in southeast Nigeria, while global data are conflicting. In some studies, the severity of anaemia in SCA has been associated with the level of HDL-C. The level of HDL-C correlates well with the level of haemoglobin F (HBF), which is essential in reducing untoward consequences of SCD. Moreover, HDL-C levels are inversely proportional to lactate dehydrogenase (LDH) level, a biomarker of haemolysis in SCA. Higher levels of LDH are associated with the severity of haemolysis. Different studies have also noted low levels of total cholesterol and elevated triglycerides to influence the severity of anaemia and associated effects in SCD. However, another study
reported no significant correlation between lipid levels and disease severity in patients with SCA. [9]

Given these discrepancies, this study was carried out to determine the blood lipid profile in SCA patients in Abakaliki, southeast Nigeria. The study evaluated the relationship between the blood lipid profile and disease severity in SCA.

**Methods**

This was a cross-sectional study conducted over five months (August to December 2022).

**Study participants**
Fifty subjects with HbSS attending the Sickle Cell Centre at the Alex Ekwueme Federal University Teaching Hospital Abakaliki (AEFUTHA), Southeast Nigeria while the controls were fifty age- and sex-matched controls aged 18 years and above. The controls were randomly selected from voluntary blood donors attending the blood donor clinic were recruited for this study.

**Ethical considerations**
Ethical approval for the study was obtained from the Hospital Research and Ethics Committee (NHREC 16/05/22/127). Informed written consent was obtained from the subjects. Patients' hospital records and a structured, interviewer-administered questionnaire were used to collect the participants' demographic and relevant clinico-laboratory data.

**Inclusion criteria**
Adult patients with HbSS confirmed with High-Performance Liquid Chromatography. Additional inclusion criteria for the cases included no history of crises in the last month, no history of blood transfusion in the preceding three months and an overnight fasting of about 12 hours duration. The controls were characterised by HbAA phenotype by haemoglobin electrophoresis and aged 18 years and above.

**Exclusion criteria**
Patients with recent history of crises, history of hypertension, and diabetes mellitus were excluded from the study. Moreover, those who did not consent to the study, those who were currently on lipid-lowering medications and those whose haemoglobin phenotype was not HbSS were excluded.

**Evaluation of disease severity**
Disease severity was determined and graded using the protocol recommended by Okocha et al. for SCD patients. [15] The patients were allocated scores for the following parameters: total white blood cell count, haemoglobin levels, transfusion data, and number of lifetime complications. The total severity scores were then stratified as <3 for mild disease, 3-5 for moderate disease, while scores >5 defined severe disease.

**Laboratory analysis**
Eight millilitres of venous blood were drawn from each participant at enrolment following a 12-hour fast. Three and five millilitres were dispensed into commercially prepared sodium ethylenediaminetetraacetic acid (EDTA) and lithium heparin sample tubes to analyse haematological and biochemical parameters, respectively. Haematological parameters, including full blood counts, were carried out using BC 5300 Mindray Haematology Analyzer. Haemoglobin genotyping was performed by high-performance liquid chromatography on an HPLC/Variant II haemoglobin testing system (Bio-Rad, Hercules, California, USA) to confirm the diagnosis of SCA. Biochemical assays of total cholesterol, high-density lipoprotein, and lactate dehydrogenase were performed using Selectra pro-XS biochemistry analyser (Elitech Group, Netherlands). Haematological and biochemical tests were conducted at the Research Laboratory of AEFUTHA.
**Statistical analyses**

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) version 23 software (IBM, Armonk, New York, USA). Continuous variables, including age, haematological parameters, Hb variants, total cholesterol, HDL, and LDH, were summarised as mean, standard deviation, range and 95% confidence interval. Categorical variables, including frequency of crises, blood transfusions, and organ complications, were summarised as frequency and percentages. The differences in the mean of continuous variables between the SCD and control population were tested using the Student’s t-test. In contrast, analysis of variance (ANOVA) was used to compare mean values of categorical variables between SCD severity groups. The differences in the proportion of categorical variables were tested using the Chi-Square test. Pearson’s correlation test was used to test the relationship between total cholesterol, HDL and LDH with haemoglobin, levels of Hb variants, and SCD severity score with age, Hb and Hb variants. P value ≤ 0.05 was considered statistically significant.

**Results**

Fifty patients with SCA and 50 age and sex-matched HbAA controls participated in the study. The majority of the participants were students (Table I).

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>SCA n (%)</th>
<th>Controls n (%)</th>
<th>Statistics</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age group</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18 – 24</td>
<td>33 (66.0)</td>
<td>41 (82.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25 – 29</td>
<td>9 (18.0)</td>
<td>6 (12.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 – 34</td>
<td>5 (10.0)</td>
<td>1 (2.0)</td>
<td>Fishers</td>
<td>0.174</td>
</tr>
<tr>
<td>35 – 39</td>
<td>0 (0.0)</td>
<td>1 (2.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥40</td>
<td>3 (6.0)</td>
<td>1 (2.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>25 (50.0)</td>
<td>23 (46.0)</td>
<td>χ² = 0.160</td>
<td>0.689</td>
</tr>
<tr>
<td>Female</td>
<td>25 (50.0)</td>
<td>27 (54.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary</td>
<td>21 (42.0)</td>
<td>0 (0.0)</td>
<td>Fishers</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Undergraduate</td>
<td>27 (54.0)</td>
<td>45 (90.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postgraduate</td>
<td>2 (4.0)</td>
<td>5 (10.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>44 (88.0)</td>
<td>48 (96.0)</td>
<td>Fishers</td>
<td>0.269</td>
</tr>
<tr>
<td>Married</td>
<td>6 (12.0)</td>
<td>2 (4.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Occupation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Students</td>
<td>34 (68.0)</td>
<td>45 (80.0)</td>
<td>Fishers</td>
<td>0.194</td>
</tr>
<tr>
<td>Business/trading</td>
<td>6 (12.0)</td>
<td>5 (10.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Civil servants</td>
<td>3 (6.0)</td>
<td>3 (6.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Artisan/ Unemployed</td>
<td>4 (8.0)</td>
<td>2 (4.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corp member</td>
<td>3 (6.0)</td>
<td>0 (0.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In Table II, the mean red cell count, haemoglobin levels and haematocrit of the SCA patients were significantly lower than those of the control group (3.2±0.7×10¹²/L vs. 4.8±0.7×10¹²/L,

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7.5±1.4 g/dL vs. 12.3±1.2 g/dL, 23.8±4.1% vs 37.2±3.7%; p<0.001 in each case). The SCA patients also had significantly higher mean LDH levels than the control group (759.5 ±379.6iu/L vs. 417.6±495.1iu/L; p <0.001). Total cholesterol and HDL were significantly lower in SCA patients compared to HbAA controls (3.3±1.2mmol/L vs. 4.2±1.2mmol/L; p<0.001) and (1.3±0.5 mmol/L vs. 1.5±0.4mmol/L; p = 0.038) respectively.

Table II: Mean age, blood count, haemoglobin distribution, LDH, and cholesterol distribution of the study population

<table>
<thead>
<tr>
<th>Parameters</th>
<th>SCA</th>
<th>Range</th>
<th>Controls</th>
<th>Range</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>23.8 ± 6.9</td>
<td>18 – 47</td>
<td>22.7 ± 4.6</td>
<td>17 – 43</td>
<td>0.332</td>
</tr>
<tr>
<td>Blood Parameters</td>
<td>Mean ± SD</td>
<td>95% CI</td>
<td>Mean ± SD</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>RBC (cells/mcL)</td>
<td>3.2 ± 0.7</td>
<td>3.0 – 3.4</td>
<td>4.8 ± 0.7</td>
<td>4.6 – 5.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>7.5 ± 1.4</td>
<td>7.2 – 7.9</td>
<td>12.3 ± 1.2</td>
<td>12.0 – 112.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>23.8 ± 4.1</td>
<td>22.7 – 25.0</td>
<td>37.2 ± 3.7</td>
<td>36.1 – 38.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Haemoglobin variants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A₂</td>
<td>3.5 ± 1.2</td>
<td>0.0 – 6.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>5.5 ± 5.2</td>
<td>0.2 – 19.7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>82.5 ± 11.4</td>
<td>37.5 – 92.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A₄c</td>
<td>5.3 ± 4.1</td>
<td>0.0 – 13.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A₀</td>
<td>4.0 ± 4.7</td>
<td>0.0 – 38.1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>0.0</td>
<td>0.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>759.5 ± 379.6</td>
<td>651.7 – 867.4</td>
<td>417.6 ± 495.1</td>
<td>276.9 – 558.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lipid Parameters (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>3.3 ± 1.2</td>
<td>2.9 – 3.6</td>
<td>4.2 ± 1.2</td>
<td>3.9 – 4.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL</td>
<td>1.3 ± 0.5</td>
<td>1.2 – 1.4</td>
<td>1.5 ± 0.4</td>
<td>1.4 – 1.6</td>
<td>0.038</td>
</tr>
</tbody>
</table>


In Table III, the majority of the SCA patients, 24 (48%), had moderate disease severity. About 80% had 3 or fewer painful crises in the last year, while 17 (63%) have had only three or fewer blood transfusions during the previous 12 months. Avascular necrosis of the hip joints (3; 6%) and chronic pain syndrome (3; 6%) were the most common complications among the patients with SCA.

Table IV depicts that based on disease severity, there was no statistically significant difference in the levels of total cholesterol and HDL-C levels across the SCA disease severity groups (p = 0.694 and 0.262, respectively). Table V shows there was no significant correlation observed between total cholesterol and HDL-C and age (p = 0.776 and p = 0.316 respectively), markers of haemolysis (Lactate dehydrogenase p = 0.502 and p = 0.024 and Haemoglobin concentration (p = 0.012 and p = 0.933) respectively. There was also no significant correlation between Total cholesterol, HDL, and haemoglobin F and S levels (p = 0.758; p = 0.930) and (p = 0.964; p = 0.890), respectively.

Discussion

The present study hypothesised a relationship between the levels of some plasma lipids and the severity of SCD. The mean total plasma cholesterol (TC) was lower in SCA patients than in the control group. From previous studies, hypcholesterolaemia is a documented
biochemical abnormality in patients with SCA.\textsuperscript{16, 17} As seen in the general population, the relationship between low plasma cholesterol and increased mortality had been reported by Nago \textit{et al.} and Koton \textit{et al.}, whose studies concluded that cancer mortality, stroke mortality, and heart disease mortality were related to low plasma cholesterol.\textsuperscript{18, 19},

Table III: Disease severity of the HBSS study population

\begin{center}
\begin{tabular}{ |l | c |}
\hline
\textbf{Indices of disease severity} & \textbf{N (%)} \\
\hline
Number of crises in the last one year & \\
0 - 1 & 22 (44.0) \\
2 - 3 & 18 (36.0) \\
>3 & 10 (20.0) \\
History of blood transfusion & 27 (54.0) \\
\hline
Number of blood transfusions in the last one year & \\
≤3 & 17 (63.0) \\
>3 & 10 (37.0) \\
\hline
Organ complications & \\
AVN & 3 (6.0) \\
Chronic pain syndrome & 3 (6.0) \\
Ulcers & 2 (4.0) \\
Pneumonia & 2 (4.0) \\
Infertility & 2 (4.0) \\
Retinopathy & 2 (4.0) \\
Seizure disorder & 1 (2.0) \\
Priapism & 1 (2.0) \\
Massive splenomegaly & 1 (2.0) \\
\hline
Disease severity & \\
Mild & 14 (28.0) \\
Moderate & 24 (48.0) \\
Severe & 12 (24.0) \\
\hline
\end{tabular}
\end{center}

AVN – Avascular necrosis

Table IV: Distribution of body cholesterol and HDL based on the severity of SCD

\begin{center}
\begin{tabular}{ |l | c | c | c | c | c |}
\hline
 & \textbf{Mild} & \textbf{Moderate} & \textbf{Severe} & \textbf{F value} & \textbf{P value} \\
& \textbf{\textit{n = 14}} & \textbf{\textit{n = 24}} & \textbf{\textit{n = 12}} & & \\
\hline
Total cholesterol (mmol/L) & 3.1±1.0 & 3.4±1.3 & 3.2±1.1 & 0.368 & 0.694 \\
& 2.5 – 3.7 & 2.9 – 4.0 & 2.5 – 3.9 & & \\
HDL-C (mmol/L) & 1.2±0.3 & 1.4±0.6 & 1.1±0.3 & 1.377 & 0.262 \\
& 1.0 – 1.4 & 1.2 – 1.7 & 1.0 – 1.3 & & \\
\hline
\end{tabular}
\end{center}

HDL-C – High-Density Lipoprotein-Cholesterol

However, these studies could not interpret the exact relationship between low plasma cholesterol and these mortalities. Several hypotheses have been proposed to account for low cholesterol plasma levels among individuals with SCA. These include increased cholesterol
utilisation during the increased red blood cell synthesis in SCA, dilution by a decrease in erythrocyte bulk, and increased plasma volume and lower cholesterol production due to diminished liver function. In understanding the hypotheses of increased utilisation of cholesterol synthesis during erythropoiesis in SCA, cholesterol is known to be mainly conserved through enterohepatic circulation, and the synthesis of new RBC membranes will likely consume recycled cholesterol from haemolysed erythrocytes. There are also reports of the unknown mechanism of the haemoglobin S gene inducing hypocholesterolaemia. At the same time, it has been noted that SCA rarely co-exists with conditions associated with secondary hyperlipidaemia, like diabetes mellitus.

Table V: Correlations of TC, HDL-C with age, haematocrit, LDH and Hb variants

<table>
<thead>
<tr>
<th></th>
<th>TC</th>
<th>HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.041</td>
<td>-0.145</td>
</tr>
<tr>
<td>Hb</td>
<td>0.164</td>
<td>-0.012</td>
</tr>
<tr>
<td>PCV</td>
<td>0.146</td>
<td>-0.037</td>
</tr>
<tr>
<td>LDH</td>
<td>-0.097</td>
<td>0.024</td>
</tr>
<tr>
<td>A₂</td>
<td>-0.210</td>
<td>0.058</td>
</tr>
<tr>
<td>F</td>
<td>0.045</td>
<td>-0.013</td>
</tr>
<tr>
<td>S</td>
<td>0.007</td>
<td>-0.020</td>
</tr>
<tr>
<td>A₁c</td>
<td>-0.221</td>
<td>0.075</td>
</tr>
<tr>
<td>A₀</td>
<td>0.046</td>
<td>-0.041</td>
</tr>
<tr>
<td>C</td>
<td>0.076</td>
<td>-0.069</td>
</tr>
</tbody>
</table>

TC – Total Cholesterol; HDL-C – High Density Lipoprotein-Cholesterol; Hb – Haemoglobin concentration; PCV – Packed Cell Volume; LDH – Lactate dehydrogenase

The patients with SCA in the present study had significantly lower HDL-C levels than the control group. This observation is in agreement with previous studies. A low HDL-C level is undesirable because HDL-C possesses anti-inflammatory and vasoprotective functions. HDL-C can inhibit low-density lipoprotein cholesterol (LDL-C) oxidation and other inflammatory complex formation. Recent data have revealed the participation of HDL-C in the vascular environment regarding haemolysis and anaemia, thereby suggesting the potential involvement of HDL-C in modulating haemolysis and vascular dysfunction. In SCA, cell-free haemoglobin (Hb), which is released into the blood after haemolysis, might alter the inflammatory properties of high-density lipoprotein cholesterol (HDL-C). HDL-C turns pro-inflammatory from being anti-inflammatory; hence, HDL-C becomes pro-inflammatory. Conversely, some studies have reported higher HDL-C levels in SCA patients. This variation may be due to age, gender, body mass index, diet composition, smoking, sample size, disease severity, and other underlying diseases and treatment regimens.

Most of the patients with SCA in the current study had moderate disease. This is in keeping with a previous report in Nigeria. Similar to the opinion of Ebele et al., the present study did not find any significant relationship between TC, HDL-C plasma levels, and disease severity in SCA patients. Moreover, no correlation was found between total cholesterol and HDL-C plasma levels and markers of haemolysis (LDH
and haemoglobin concentration), Hbs S, and HbF levels. Low plasma lipid levels were not isolated to SCA but have been reported in other kinds of anaemia, both haemolytic and non-haemolytic. This biochemical abnormality may be a result of the impact of anaemia on lipid metabolism generally rather than a mechanism peculiar to SCA pathology. However, Conceição da Guarda et al. and Emokpae et al. reported relationships between these markers, disease severity, and lipid levels. [31, 32] The finding of Emokpae may be attributable to age differences in the study population and the larger sample size of their study.

Although the current study could not establish a significant correlation between the levels of some lipids and disease severity in SCA, it demonstrated lower mean levels of HDL-C and TC among individuals with SCA compared to the control group. This agrees with other studies that reported decreased total cholesterol, HDL-C, and LDL-C levels among SCA individuals. These observations may highlight the need for future research in this direction.

Limitations

The limitations of this study may include a small sample size, which may limit the power of the research and the inability to include the participants' dietary and exercise history in the study's scope.

Conclusion

This study found that plasma TC and HDL-C are lower in SCA compared to individuals with HbAA. However, the study did not show any relationship between these plasma lipids and disease severity, markers of haemolysis, Hb F and HbS levels.

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