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EFFECTS OF SELENIUM SUPPLEMENTATION ON APOLIPOPROTEINS AND LIPID PROFILE IN STEADY-STATE SICKLE CELL DISEASE PATIENTS IN SOUTHEASTERN NIGERIA

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ABSTRACT

Sickle Cell Disease (SCD) is the most prevalent hereditary hemoglobinopathy characterized by chronic oxidative stress, inflammation and metabolic disturbances that causes long-term disability requiring daily care. Sickle cell disease affects about 4.4 million people globally. Selenium (Se), an essential micronutrient with antioxidant properties, may influence oxidative balance and lipid metabolism in SCD. However, there is paucity of data, especially in sub-Saharan Africa, on the effect of Se supplementation on apolipoproteins and lipid profile in SCD. This study assessed the effect of Se supplementation on levels of serum Se, apolipoproteins A1 and B (apo A1 and apo B), and other lipid profile markers among individuals with different HB genotype blood groups. One hundred and fifty (150) aged-matched adult (18-60years) attending routine clinic in the Hematology department of the Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria, were randomly recruited and grouped according to hemoglobin genotype: group 1= HbAA (n=50), group 2= HbAS (n=50) and group 3= HbSS (n=50). Participants in group 3 received oral Se supplementation (200 µg/day) for 90 days. Blood samples were collected at baseline and after 90 days (post supplementation). Serum Se was measured using atomic absorption spectrophotometry method (AAS) while apolipoproteins and lipid profile were determined using enzymatic spectrophotometric method. Questionnaires were administered to participants for relevant data. At baseline, the study showed that there were significant variations in the levels of apo A1, Apo B, TG, HDL-C, LDL-C and VLDL-C concentrations among the three groups ($p< 0.001$). However, after Se supplementation in the HbSS group, apo A1 and B levels increased significantly ($p= 0.003$ and 0.001, respectively). Likewise, HDL-C and TG concentrations were significantly elevated ($p < 0.001$), whereas total cholesterol and LDL-C levels showed no significant changes post-supplementation. The findings from this study suggest that dyslipidemia and oxidative stress persist in steady-state SCD. Low level of Se is indicative of a weakened antioxidant defense even in individuals with steady-state SCD. Selenium supplementation in SCD patients in this study appears to improve selected lipid and apolipoprotein parameters, suggesting its potential therapeutic value in the management of SCD.

KEY WORDS: *HDL-C High density lipoprotein-cholesterol, LDL-C low density lipoprotein-cholesterol, Se Selenium, TC Total cholesterol, TG Triglyceride, VLDL-C Very low density lipoprotein cholesterol, Apo A-1 Apolipoprotein A-1, Apo B Apolipoprotein B, SS Homozygous sickle cell hemoglobin, AA Homozygous Adult hemoglobin, AS Heterozygous sickle disease carrier hemoglobin.*

INTRODUCTION

Sickle-cell anemia (SCA) results from a point mutation, in which an adenine nucleobase in the sixth codon of the β-globin gene is replaced by a thymine (GAG → GTG). The molecular translation replaces glutamic acid with valine, thereby producing an abnormal form of hemoglobin called hemoglobin S (20,22). Although the molecular lesion is limited to a single nucleotide, the SCA gene is pleiotropic and leads to multiple phenotypic expressions. SCA patients may present with various complications, such as recurrent episodes of vaso-occlusion, acute chest syndrome (ACS), stroke, infections, and priapism. These complications vary considerably among patients and over time (16,23). Sickle cell anemia is an autosomal recessive disorder affecting the function of haemoglobin. In order for full disease symptoms to manifest in an individual, they must carry two copies (homozygous genotype = SS) of the HbS gene. However, individuals who are heterozygous (genotype = AS) have what is referred to as sickle cell trait, a phenotypically dominant trait. Although AS individuals are clinically normal, their red blood cells can sickle under very low oxygen pressure, e.g., when at high altitudes in airplanes with reduced cabin pressure. Because of this phenomenon, AS individuals exhibit phenotypic dominance yet are recessive genotypically (23). Mutations in the globin genes that alter the protein composition, but not necessarily the amount of expression are referred to as qualitative mutations. Of the mutations leading to qualitative alterations in hemoglobin, the missense mutation in the β-globin gene that causes sickle cell anemia is the most common (16)

The form of hemoglobin in persons with sickle cell anemia is referred to as HbS. The nomenclature for normal adult hemoglobin protein is HbA Adult red blood cells also carry another minor form of adult haemoglobin (about 2% of the total) identified as HbA_2. The HbA_1 heterotetramer is composed of two α-globin peptides and two β-globin peptides. The HbA_2 heterotetramer is composed of two α-globin peptides and two δ-globin peptides. The human β-globin gene cluster contains several genes whose 5' to 3' orientation on chromosome 11 reflects the ontogeny of their expression from embryonic globin epsilon (ε) gene], to fetal β-globin gammaG (γG) and γA genes], to adult β-globin weakly the delta (δ) gene followed by the β gene. The β-globin gene (HBB) is located at 11p15.5 and is composed of 3 exons that encode a 147 amino acid protein (18). The underlying problem in sickle cell anemia is that the valine for glutamic acid substitution results in hemoglobin tetramers that aggregate into arrays upon deoxygenation in the tissues. This aggregation leads to deformation of the red blood cell into a sickle-like shape making it relatively inflexible and unable to traverse the capillary beds. This structural alteration in the red blood cell can easily be seen under light microscopy and is the source of the name of this disease. Repeated cycles of oxygenation and deoxygenation lead to irreversible sickling (18). Sickle cell anaemia is characterized by persistent episodes of haemolytic anaemia and the occurrence of acute episodes referred to as sickling crises. The sickling red cells result in clogging of the fine capillary beds. Individuals with SCA are subjected to increased oxidative stress, (17), particularly during vaso-occlusive crises and acute chest pain, (17). Several aspects of the abnormalities in SCA are thought to result from the oxidative stress of RBCs, white blood cells (WBCs) and endothelial cells and activation of platelets, (20,22). Oxidative stress represents the imbalance between enhanced generation of reactive oxygen species and low cellular content of antioxidants, (1,2,19). While continuously subjected to oxidative stress in cellular environment, the red cell possesses various antioxidant systems for its protection. Reactive oxygen species can cause damage to biological macromolecules such as proteins, lipids and DNA, (19,24). The unsaturated chain of membrane fatty acids can readily react with free radicals and undergo peroxidation. This process can become autocatalytic after initiation and yields lipid peroxides, lipid alcohols and aldehydic byproducts, such as, 4-hydroxynonenal (4-HNE) and malondialdehyde (MDA), (Antunies et al 1996). Oxidative damage can also alter membrane permeability and lead to hemolysis. In addition, oxidative insult can result in immune recognition of RBC, (17). Other reactions of reactive oxygen species with proteins result in the oxidation of various amino-acid side chains, often leading to a loss of function, (Aslan et al., 2021). Sickle cell disease (SCD), is the most common blood disorder in the world. It can cause major problems and long-term disability. Thus, it requires daily care and management. Africa has the highest prevalence of the carrier rates with 20-30% in countries such as Nigeria, Cameroon, Republic of Congo, Gabon, and Ghana (WHO Africa, 2020). Sub Saharan Africa accounts for the highest percentages of various indices associated with sickle cell disorder. It accommodates 75% of all patients with sickle cell disease and 70% of all sickle cell disease births globally, with some affected children dying before the age of 5 years, (Stephen et al., 2018). Nigeria has the highest number of people suffering from sickle cell disease in the world, (Adigwe et al., 2023). As the most sickle cell endemic country in Sub-Saharan Africa with between 2% and 3% of the total population affected, (Nwongoh et al., 2022). In Caucasians, this inherited disorder of haemoglobin (Hb), occurs in 70000 to 80000 Americans of African Mediterranean or Middle Eastern region, (25). In a study conducted by Delesderrier, et al. 2019 it was observed that, among the antioxidant micronutrients studied, selenium was found to play the most important role in hemolysis in SCD subjects (19). Previous studies have considered the levels of some of the inflammatory markers in healthy subjects and SCD subjects when in their steady state, (21). In addition, much is still unknown about the possible or exact role of inflammatory markers, lipid profile, apoA1, apo B in steady state SCD subjects, their potential as biomarkers of SCD (13).

AIM

This study is aimed at measuring the serum levels of Apo-A1, Apo B, lipid profile markers and Se across H.B genotype, and blood groups of people on selenium supplements.

METHODS

A total of 150 age-matched participants (18-60 years) visiting the Haematology Clinic at Nnamdi Azikiwe University Teaching Hospital in Nnewi, Nigeria, were recruited and categorized into three groups based on their hemoglobin genotype: HbAA (n = 50), HbAS (n = 50), and HbSS (n = 50). Participants with HbSS were given oral selenium supplementation (100 µg/day) for 90 days. The sickle cell phenotype in sickle cell anaemia (SS) patients was assessed by Hb electrophoresis and confirmed by restricted fragment length probe polymerase chain reaction (RFLP PCR). Concerning the control subjects, the absence of a sickle cell trait was confirmed by the presence of a normal Hb electrophoresis.

Blood samples were taken both before and after the selenium supplement intervention. Serum selenium levels were determined using atomic absorption spectrophotometry, Apolipoproteins and lipid profile markers were evaluated using the enzymatic spectrophotometric method. Ethical approval for this study was obtained from the Nnamdi Azikiwe University Teaching Hospital Ethics Committee.

RESULTS

Table 1: Levels of (MEAN±SD) Apo A1, Apo B, TC, TG, HDL-C AND LDL-C and VLDL-C among the three HB genotype groups at baseline

Group (n=150)	Apo A1(g/l)	Apo B(g/l)	TC (mmol/l)	TG (mmol/l)	HDL-C (mmol/l)	LDL-C (mmol/l)	VLDL-C (mmol/l)
SS (n=50)	166.938±6.96	85.30±10.31	3.49±0.55	0.95±0.75	0.89±0.85	2.10±0.38	0.20±0.04
AS (n=50)	177.54±4.39	95.26±10.97	3.46±0.37	0.92±0.79	1.00±0.14	2.39±0.59	0.19±0.02
AA (n=50)	170.36±7.63	90.26±4.27	4.42±0.53	1.15±0.79	1.06±0.93	2.86±0.43	0.21±0.02
f-value	34.620	15.128	62.462	124.879	27.944	32.709	16.56
p-value	0.001*	0.001*	0.001*	0.001*	0.001*	0.001*	0.001*
SS vs AS	0.000*	0.000*	1.000	0.375	0.000*	0.011*	0.058
SS vs AA	0.030*	0.021*	0.000*	0.000*	0.000*	0.000*	0.003*
AS vs AA	0.000*	0.020*	0.000*	0.000*	0.053	0.000*	0.000*

Apo A_1 = Apolipoprotein A

Apo B = Apolipoprotein B

HDL-C = High Density Lipoprotein-Cholesterol

LDL-C = Low Density Lipoprotein-Cholesterol

TC = Total Cholesterol

TG = Triacylglycerol

SS= Homozygous sickle cell anemia genotype, AS= Heterozygous sickle cell carrier genotype, AA= Normal Healthy individual genotype.

Where p is significant at:

***p < 0.001 level

**p

*P < 0.05 level

Table 2: Levels of (MEAN±SD) apolipoproteins and lipid markers before and after selenium supplement intervention in SS HB genotype group.

Parameter	Before	After	t-test	p-value
Apo A_1 (g/l)	166.93±6.96	173.81±14.20	-2.800	0.003*
Apo B (g/l)	85.31±10.31	97.08±10.88	-5.383	0.001*
HDL-C (mmol/l)	0.89±0.85	0.97±0.18	-2.439	0.018*
LDL-C (mmol/l)	2.10±0.39	2.01±0.38	1.278	0.207
TC (mmol/l)	3.49±0.55	3.53±0.59	-0.454	0.552
TG (mmol/l)	0.95±0.75	1.05±0.20	-3.468	0.001*
Se (mcg/l)	6.83±1.34	18.07±2.70	-25.264	0.001*

APO A_1 =Apolipoprotein A

Apo B= Apolipoprotein B

HDL-C =High Density Lipoprotein-Cholesterol

LDL-C = Low Density Lipoprotein-Cholesterol

TC = Total Cholesterol

TG =Triacylglycerol

SS= Homozygous sickle cell anaemia genotype.

*P < 0.05 level

**p

***p < 0.001 level

Table 3: Levels of (MEAN±SD) Apo A1, Apo B, TC, TG, HDL-C and LDL-C among the three HB genotype groups and gender.

Gender/Genotype	N	APO-A1 (g/l)	APO B(g/l)	TC (mmol/l)	TG (mmol/l)	HDL-C (mmol/l)	LDL-C (mmol/l)
AA female	25	171.65±7.85	90.70±2.64	4.35±0.52	1.15±0.91	1.04±0.10	2.75±0.44
AA male	25	169.08±7.34	89.83±5.47	4.50±0.53	1.14±0.65	1.07±0.08	2.97±0.39
AS female	27	177.59±5.05	96.74±9.72	3.43±0.41	0.93±0.07	1.00±0.16	2.30±0.58
AS male	23	177.51±3.60	93.52±12.32	3.50±0.32	0.91±0.05	1.01±0.12	2.48±0.60
SS female	20	165.59±6.63	83.20±10.93	3.40±0.55	0.95±0.07	0.90±0.09	2.12±0.46
SS male	30	167.91±7.13	86.71±9.83	3.55±0.56	0.95±0.08	0.90±0.82	2.09±0.34
f-value		14.599	6.774	25.339	49.271	11.315	14.152
p-value		0.001*	0.001*	0.001*	0.001*	0.001*	0.001*
AA female vs AS female	50	0.018*	0.259	0.001*	0.001*	1.000	0.012*
AS female vs SS female	50	0.001*	0.001*	1.000	1.000	0.054	1.000
AA female vs SS female	50	0.033*	0.095	0.001*	0.001*	0.001*	0.001*
AA male vs AS male	50	0.001*	1.000	0.001*	0.001*	0.697	0.006
AS male vs SS male	50	0.001*	0.110	1.000	1.000	0.003*	1.000
AA male vs SS male	50	1.000	1.000	0.001*	0.001*	0.001*	0.001*

APO A_1 = Apolipoprotein A

Apo B = Apolipoprotein B

HDL-C = High Density Lipoprotein-Cholesterol

LDL-C = Low Density Lipoprotein-Cholesterol

TC = Total Cholesterol

TG = Triacylglycerol

SS= Homozygous sickle cell anaemia genotype.

AS= Heterozygous sickle cell carrier genotype

AA= Normal Healthy individual genotype.

*P < 0.05 level

**p

***p < 0.001 level

DISCUSSION

In this study the mean serum levels of ApoA1, Apo B, TC, TG, HDL-C and LDL-C were significantly different amongst the group (F=11.718, 33.645, 16.558, 11.277 and 18.958) ($p<0.05$), respectively. The mean Apo A-1 level was significantly lower in the SS group when compared to the AS group ($p=0.000$) but was not significantly different compared to the values observed in the AA group ($p=0.595$). Serum concentration of APOA1 was significantly higher in the AS group in comparison to the AA group ($p=0.003$). However, there were significant differences observed in the mean serum APOB, TC, TG, HDL-C and LDL-C levels in the SS group than in the AS group ($p < 0.05$). Serum TC, TG, HDL-C and LDL-C levels and Apo B in the SS group were significantly lower compared to AA group respectively ($p<0.05$). Moreover, the mean serum TC, TG, HDL-C and LDL-C levels in the AS group were significantly lower when compared to the observed values obtained in AA group ($p<0.05$) respectively. Lipid metabolism disorders, especially hypocholesterolemia and hypertriglyceridemia, are linked to clinical events observed in SCA, suggesting they play a relevant role in the multifactorial pathogenesis of this disease (Dantas et al., 2022). However, it might be hypothesized that SCD hypocholesterolemia results from cholesterol utilization during increased erythropoiesis of SCD. Cholesterol is largely conserved through enterohepatic circulation at least in healthy individuals. In this study, participants from the SS and AS groups had lower total cholesterol (TC), HDL-C, TG, and LDL-C values than those from the AA group. One study which was carried out in Lagos south west Nigeria reported low TC, LDL-C and HDL-C levels in SCD patients compared with local laboratory reference values (Ebele et al., 2018) which agrees with the findings of the present study. Nearly every study that looked at lipids in SCD adults found that they had lower TC and LDL-C levels hence this may be suggestive for the lower levels of these biochemical parameters in this study. Samarah et al. also observed that total cholesterol and LDL-C were significantly lower in SS and sickle β-thalassemia patients compared to AS individuals and AA controls which is in corroboration with the present findings whereas they noted that the HDL-C was significantly higher in AS individuals compared to AA controls which is in contrast with the current reports (Samarah et al., 2021). Decreased TC and LDL-C in SCD have been reported in many previous studies that examined lipids in SCD patients (8,9 Guarda et al., 2020) while some other similar studies showed lower HDL-C and TC levels among the SCD individuals than in controls (Gupta et al., 2020). We found significantly lower levels of cholesterol, LDL-C, and HDL-C, apo A and apo B in SCD cases, which are consistent with earlier research by other investigators (15, Gupta et al., 2020). Previous study has shown that cholesterol is closely related to haematocrit values, and hypocholesterolemia is also commonly seen in different types of anaemia (Ephrahim et al., 2016). The observed hypocholesterolemia may be due to decreased reservoir storage of cholesterol related to the decreased total red cell mass in SCD anaemia (14, Dantas et al., 2022). Hypocholesterolemia have been documented in SCD worldwide for over forty years, yet the mechanistic basis and physiological aspects of these altered lipid levels have yet to be fully elucidated (8,9). The low levels of LDL-C in SCD are consistent with low levels of TC and the absence of atherosclerosis among SCD individuals (Guarda et al., 2020). The present study result also clearly shows a decrease in HDL-C in SCD (SS and AS) vs Controls (AA). Lower HDL-C in SCD has been documented in some, but not all previous studies (5,6,8,9). In studies on Lipid in which HDL-C is low, this may be suggestive for inconsistencies between studies, including differences in age, diet, bodyweight, smoking, gender, different ranges of disease and severity of other diseases and other diseases and treatments (1). Decreased HDL-C is a known risk factor endothelial dysfunction in the general population and in SCD (15). Furthermore, this study also shows that TG level was significantly lower in SS and AS compared to AA. In normal individuals. TG levels are determined to a significant degree by bodyweight, physical exercise, diet. Mechanisms for SCD specific risk factors for delayed TG clearance are not clear. In SCD the rate of TG synthesis from glycerol is elevated up to four-fold in sickled reticulocytes (8,9), but SCD patients have defects in postabsorptive homeostasis of fatty acids (4). Some Previous studies have recorded mixed results regarding the levels of TG in SCD. Increased TG levels have been reported in several studies of SCD adults (25) and this corroborates with the findings in this study. Lipolysis of TG present in TG-rich lipoproteins releases neutral and oxidized free fatty acids that induce endothelial cell inflammation. However, two studies (8,9,) did not find increase TG levels in

SCD adults but the findings differ from that of the present study. The mean value observed in apolipoprotein A-I in the AA and AS groups (female and male) were higher compared to that of the SS group (female 165.59±6.63 and 167±7.13) respectively p= 0.001, f value=14.599. The post hoc analysis was statistically significant in all the variables except AA male vs SS male p=1.000. Apo B showed a higher mean value in the AA and AS groups (female and male) compared with the mean value obtained in the SS group (83.20±10.93 and 86.71±9.83) and statistically significant p value = 0.001. In the post hoc study apo B showed statistically difference only in AS female vs SS female ($p < 0.001$). Consequently, total cholesterol level was lower in the SS group (3.55±0.46 and 3.40±0.55) female and male and higher in the AA group (4.35±0.52 and 4.50±0.53) respectively ($p <0.001$). Post hoc analysis showed a statistical difference in all the variables except AS vs SS female and male. However, TG was elevated in the AA group (1.15±0.91 and 1.14±0.65) compared with the mean level observed in the SS group (0.95±0.07 and 0.95±0.08) p value = 0.001. Post hoc analysis was statistically significant in all except the AS vs SS variable (female and male.). In addition, HDL-C was higher in the AA group (female and male) (1.04±0.10 and 1.07±0.08) compared with the observed mean values in the SS group (0.90±0.89 and 0.90±0.82) $p<0.001$. The mean value obtained from the post hoc analysis of the variables statistically significant are 0.003, 0.001 and 0.001 respectively. LDL-C showed a higher mean value in the AA group (2.75±0.44 and 2.97±0.39) and lower in the SS group (2.12±0.46 and 2.09±0.34) respectively with f value14.152 ($p <0.001$). Conversely, post hoc analysis was significant in AA female vs AS female (0.012) and AA male vs SS male (0.001). Hypocholesterolemia is the most frequently encountered lipid abnormality in SCD (15). Lower levels of LDL-C, TC, HDL-C, Apo A-1 and Apo B in SCD compared to controls. Hemolysis was associated with decreased HDL-C and inflammation was linked to decreased Apo A-1 levels in SCD patients (15) which is in tandem with the findings in this study. The altered HDL in SCD may become dysfunctional and result with slowing down of the reverse cholesterol transport. In a study conducted by Oktaz et al 2013 hypocholesterolemia was observed in SCD individuals compare with controls while TG levels were significantly increased in SCD individuals, Apo A-1 lower and no significant difference was observed in Apo B levels compared to controls (24). Soupene et al reported a reduction in Apo A and B levels in SCD (8,9). A few studies have reported reduced Apo A-1 levels at steady state in individuals with SCD (5,6,15,8,9). Severe hemolysis causes Apo A-1 to become open to constant oxidative stress in the circulation of individuals with SCD. The loss of defence against oxidative damage maybe a mechanism that caused the relationships between oxidative/inflammatory parameters and the levels of Apo A-1 and HDL-C (15). Seixas et al, previously suggested that SCD patients with low HDL and relatively increased TG might have a specific dyslipidemic phenotype of the disease (1). They suggested that proinflammatory transformation of HDL particle in SCD due to oxidative damage (7) causes lipid peroxidation and significantly influences all facets of lipoprotein and apolipoprotein function (12) this may be the basis of low levels seen in HDL-C and Apo A-1 levels and their relationships with oxidative and inflammatory parameters (12). Se plays a significant role in the prevention of oxidative modification of lipids and reduction of inflammation. Proteasa and colleagues suggested that reduced serum concentrations of Se may be associated with impaired antioxidant capacity of erythrocytes caused by reduced activity of GPx and other selenoenzymes as shown by Natta and Colleagues (SS female 28 and male 23) who observed low serum concentrations and lower GPx activity in SCD individuals (10,11). The findings in their studies were in corroboration with what was observed in this study. They also suggested that a reduced serum concentration of Se may impair the body's antioxidant capacity resulting in higher ROS formation and subsequent oxidative stress.

CONCLUSION

The observed hypocholesterolemia may be due to decreased reservoir storage of cholesterol related to the decreased total red cell mass in SCD anaemia (14). Hypocholesterolemia have been documented in SCD worldwide for over forty years, yet the mechanistic basis and physiological aspects of these altered lipid levels have yet to be fully elucidated (Ndrepepa et al., 2020). The low levels of LDL-C in SCD are consistent with low levels of TC and the absence of atherosclerosis among SCD individuals (14). In studies on Lipid in which HDL-C is low, this may be suggestive for inconsistencies between studies, including differences in age, diet, bodyweight, smoking, gender, different ranges of disease and severity of other diseases and other diseases and treatments (2). Decreased HDL-C is a known risk factor endothelial dysfunction in the general population and in SCD (15). In normal individuals TG levels are determined to a significant degree by bodyweight, physical exercise, diet. Mechanisms for SCD specific risk factors for delayed TG clearance are not clear. In SCD the rate of TG synthesis from glycerol is elevated up to four-fold in sickled reticulocytes (1), but SCD patients have defects in postabsorptive homeostasis of fatty acids (4). Lipolysis of TG present in TG-rich lipoproteins releases neutral and oxidized free fatty acids that induce endothelial cell inflammation. Low serum selenium levels observed in this study may also suggest that a weakened antioxidant potential may be associated with sickle cell disease patients. Low blood Se levels observed in this research suggest that a weakened antioxidant potential may be associated with SCA patients. There is also an increased resting metabolic rate and resting energy expenditure in SCA with subsequent increased turnover of macro- and micronutrients and subsequent depletion of micronutrients like selenium which is an important antioxidant. These variations in the serum selenium levels reported in all these studies may be a reflection of the fact that serum selenium levels are influenced by intake in the diet which varies based on geographical location.

CONFLICT OF INTEREST

The Authors declare no conflict of interest.

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AUTHORS CONTRIBUTION.

CUJ, WSN: contributed with Recruitment of Participants and Laboratory Analysis. OEC and WSN : contributed greatly with Laboratory and Statistical Analysis. SSM : helped in Conceptualising the title and abstract While OMJ and OAJ : contributed by editing the manuscript. WSN: contributed immeasurably via Conceptualisation, Laboratory analysis and Drafting of Manuscript.

CKE: contributed by editing of the manuscript

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