

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/319016903>

# EVALUATION OF SOME BIOCHEMICAL PARAMETERS OF SICKLE CELL DISEASE (SCD) PATIENTS IN BENIN CITY

Article · January 2017

CITATIONS

2

READS

376

3 authors:



**Johnson Jemikalajah**

Delta State University, Abraka

22 PUBLICATIONS 96 CITATIONS

[SEE PROFILE](#)



**Adu Matthew**

University of Delta

18 PUBLICATIONS 112 CITATIONS

[SEE PROFILE](#)



**Kester Digban**

Novena University

53 PUBLICATIONS 310 CITATIONS

[SEE PROFILE](#)

Some of the authors of this publication are also working on these related projects:



Biochemical Evaluation of Adiposity and Fat Distribution among Adult Obese subjects [View project](#)



Evaluation of some biochemical parameters among sickle cell disease patients in Benin City [View project](#)



## EVALUATION OF SOME BIOCHEMICAL PARAMETERS OF SICKLE CELL DISEASE (SCD) PATIENTS IN BENIN CITY

<sup>1</sup>Jemikalajah, D.J., <sup>2</sup>Adu, M.E.\* and <sup>3</sup>Digban, A.K.

<sup>1</sup>Department of Medical Microbiology and Parasitology, College of Medicine, Delta State University, Abraka.

<sup>2</sup>Department of Medical Laboratory Services, Antiretroviral Therapy Centre, Central Hospital, Agbor, Delta State.

<sup>3</sup>Department of Medical Laboratory Science, College of Health Sciences, Igbinedion University, Okada, Edo State.

\*Correspondence author

### ABSTRACT

**Background:** Sickle cell disease (SCD) is an autosomal inherited disorder of haemoglobin caused by a substitution of thymidine for adenine (GAG-GTG) of the  $\beta$ -chain that results in the amino acid valine instead of glutamic acid. This congenital haemoglobin mutation results in alteration in the biochemical parameters of these individuals.

**Aim:** Our aim is to assess some biochemical parameters (Glucose, Total protein, Albumin, Globulin and Creatinine) of sickle cell patients.

**Methods:** A total of one hundred and ten (110) subjects were recruited for this study which consists of fifty (50) sickle cell subjects and sixty (60) apparently healthy subjects which served as control. Out of the total fifty subjects 28 were males and 22 were females. The control subjects comprised of 21 males and 39 females. Blood samples were analyzed using standard spectrophotometric methods.

**Results:** All the estimated parameters were significantly higher in sickle cell patients except creatinine which was statistically lowered when compared to controls.

**Conclusion:** Therefore sickle cell patients have altered biochemical parameters which need to be assessed routinely to prevent morbidity and mortality.

**Keywords:** Sickle cell Disease, Total Protein, Glucose, Creatinine, Haemoglobin.

### INTRODUCTION

Sickle cell disease (SCD) is an inherited disorder of haemoglobin caused by a single nucleotide substitution of thymidine for adenine (GAG-GTG) of the  $\beta$ -chain that results in the amino acid valine instead of glutamic acid (Steinberg *et al.*, 2001, Ballas, 2002). This leads to alteration in the properties of the haemoglobin tetramer, with the tendencies to polymerize in the deoxygenated state. In homozygotes or when there is co-inheritance of a double heterozygous state (with haemoglobin C,  $\beta$ -thalassaemia, D or O), the normal  $\beta$ -chains are replaced by the modified form of the  $\beta$ -chain. Sickle Cell Disease is one of the commonest hereditary haemoglobinopathies experienced in Nigeria. Anaemia has been

observed as a common feature in Sickle Cell Disease which leads to an elevated production of haemopoietic cells due to chronic haemolysis and cell death. This results in hyper-metabolic rate and demand (Emokpae and Tijani, 2014). Edozien and his coworkers (1960) observed hyperproteinemia in sickle cell patients which Isichei (1979) in his report attributed to hyperglobulinemia that is present in Sickle cell patients. But Famodu and colleagues (1987) in their study observed a high level of albumin in Sickle cell patients. The Sickle cell gene has been observed to cause deleterious effects such as anaemia, renal dysfunction when it exists as homozygous state.

Good patient management depends on accurate and reliable laboratory results which ultimately lead to a better outcome. Sickle cell patients are fragile and vulnerable group of patients that need care at all times. There is paucity of data on the biochemical parameters of sickle cell disease in this locality. Therefore this study was undertaken to evaluate some biochemical parameters of sickle cell patients.

#### **MATERIAL AND METHODS**

This study was carried out among patients visiting Sickle Cell Centre, Benin-City Edo State. A total of one hundred and ten (110) subjects were recruited for this study which consists of fifty (50) sickle cell subjects and sixty (60) apparently healthy subjects which served as control. Out of the total fifty subjects 28 were males and 22 were females. The control subjects comprised of 21 males and 39 females. Ethical clearance was obtained from the ethical committee of Ministry of Health, Benin City and informed consent was obtained from participants after explanation of the purpose and procedures of the study.

#### **Sample Collection**

Blood samples (5mls) were collected by venepuncture into an accurately labeled fluoride oxalate and plain container for both subjects and control. The blood samples were centrifuged at 4000rpm for 10 minutes at room temperature within two hours of collection and the serum separated into clean plain containers, kept frozen until required for analysis. Analysis was carried out for serum Creatinine, Glucose, Total protein, Albumin and globulin.

#### **Biochemical Analysis**

Total serum protein was determined spectrophotometrically using Biuret method (Doumaset *al.*, 1981) while serum albumin was determined by bromocresol green

method (Doumaset *al.*, 1981). Serum globulin was calculated by subtracting albumin from total protein. Glucose was determined by glucose oxidase method (Panzer *et al.*, 2002) and serum creatinine by Picric alkaline method (Brod and Sirota 1948). All kits used were commercially available tests kits; products of Randox Laboratories, U.K. Laboratory analyses were carried out according to manufacturer's instructions.

#### **Statistical Analysis**

Data was expressed as mean and standard deviation. Comparative analysis of the two continuous variables was done using independent sample t-test. Statistical significance was set at  $P < 0.05$ . All statistics was done using statistical package for social sciences (SPSS) (version 21.0) statistical software.

#### **RESULTS**

Table 1 shows that the mean  $\pm$  SD for glucose in sickle cell subjects ( $7.05 \pm 1.12$ ) was significantly higher ( $P < 0.05$ ) when compared to the control ( $6.29 \pm 1.01$ ) subjects. Also there was a significant difference ( $P < 0.05$ ) in the mean  $\pm$  SD of serum total protein of sickle cell subjects ( $8.20 \pm 2.43$  g/dl) when compared to the control ( $7.45 \pm 1.24$ g/dl). There was no significant difference ( $P > 0.05$ ) in the mean  $\pm$  SD obtained for serum albumin in sickle cell subjects ( $4.36 \pm 0.91$ g/dl) when compared to the control ( $4.31 \pm 0.78$ g/dl). But there was a statistical significant difference ( $P < 0.05$ ) in the mean  $\pm$  SD of serum globulin in sickle cell subjects ( $3.88 \pm 2.13$  g/dl) when compared to the control ( $3.08 \pm 0.76$ g/dl). Also there was a statistical significant difference ( $P < 0.05$ ) in the mean  $\pm$  SD obtained for serum creatinine among sickle cell subjects ( $0.65 \pm 0.26$  mg/dl) when compared to the control ( $0.82 \pm 0.21$  mg/dl).

**Table I:** Mean  $\pm$ SD of Sickle Cell Disease (SCD) patients.

Parameters	SCD	Controls	t value	P value
Glucose (mmol/l)	7.05 $\pm$ 1.12	6.29 $\pm$ 1.01	4.806	0.000**
Total Protein (g/dl)	8.20 $\pm$ 2.43	7.45 $\pm$ 1.24	2.186	0.034**
Albumin (g/dl)	4.36 $\pm$ 0.91	4.31 $\pm$ 0.78	0.424	0.673*
Globulin (g/dl)	3.88 $\pm$ 2.13	3.08 $\pm$ 0.76	2.659	0.011**
Creatinine (mg/dl)	0.65 $\pm$ 0.26	0.82 $\pm$ 0.21	4.488	0.000**

\*\*Significant\*Not Significant

Table 2 shows the biochemical parameters obtained for sickle cell male subjects and its comparison with the male control. There was significant difference ( $P < 0.05$ ) when the mean  $\pm$  SD of serum glucose in sickle cell male subjects ( $7.29 \pm 1.20$ mg/dl) were compared to the male control ( $6.26 \pm 0.87$ mg/dl). The mean  $\pm$  SD of serum total protein of male subjects ( $8.13 \pm 2.58$ g/dl) was elevated but not statistically significant ( $P > 0.05$ ) when compared to the male control ( $7.75 \pm 1.40$ g/dl). There was no significant

difference ( $P > 0.05$ ) when the mean  $\pm$  SD of serum albumin in sickle cell male subjects ( $4.24 \pm 0.69$ g/dl) was compared to the male control ( $4.41 \pm 0.88$ g/dl). The mean  $\pm$  SD of serum globulin of male subjects ( $3.92 \pm 2.24$ g/dl) was not significant ( $P > 0.05$ ) when compared to the male control ( $3.37 \pm 0.87$ g/dl). The mean  $\pm$ SD of serum creatinine among sickle cell male subjects ( $0.68 \pm 0.30$ mg/dl) was significantly difference ( $P < 0.05$ ) when compared to the control ( $0.87 \pm 0.24$ mg/dl).

**Table: 2** Mean  $\pm$ SD of Male Sickle Cell Disease (SCD) patients.

Parameters	Male SCD	Male Controls	t value	P value
Glucose (mmol/l)	7.29 $\pm$ 1.20	6.26 $\pm$ 0.87	4.509	0.000**
Total Protein (g/dl)	8.13 $\pm$ 2.58	7.75 $\pm$ 1.40	0.773	0.446*
Albumin (g/dl)	4.24 $\pm$ 0.69	4.41 $\pm$ 0.88	1.322	0.197*
Globulin (g/dl)	3.92 $\pm$ 2.42	3.37 $\pm$ 0.87	1.196	0.242*
Creatinine (mg/dl)	0.68 $\pm$ 0.30	0.87 $\pm$ 0.24	3.380	0.002**

\*\*Significant\*Not Significant

Table 3 shows the mean  $\pm$ SD obtained for sickle cell female subjects and its comparison with the female control. There was significant difference ( $P < 0.05$ ) when the mean  $\pm$  SD of serum glucose of sickle cell female subjects ( $6.75 \pm 0.96$ ) was compared to the female control ( $6.30 \pm 1.08$ ). The mean  $\pm$  SD of serum total protein of female subjects ( $8.30 \pm 2.28$ ) was significantly higher when compared to the female control ( $7.28 \pm 1.13$ ). There was no significant difference ( $P > 0.05$ ) between the mean  $\pm$ SD

of serum albumin of sickle cell female subjects ( $4.53 \pm 1.11$ ) when compared to the female control ( $4.26 \pm 0.72$ ). The mean  $\pm$ SD of serum globulin of female subjects ( $3.84 \pm 1.75$ ) was significantly elevated when compared to the female control ( $2.93 \pm 0.64$ g/dl). The mean  $\pm$ SD obtained for serum creatinine among sickle cell female subjects and female control is  $0.62 \pm 0.21$  and  $0.80 \pm 0.18$  respectively were statistically significant difference ( $P < 0.05$ ).

*Evaluation of Some Biochemical*

Table 3: Mean  $\pm$ SD of Female Sickle Cell Disease (SCD) patients.

Parameters	Female SCD	Female Controls	t value	P value
Glucose (mmol/l)	6.75 $\pm$ 0.96	6.30 $\pm$ 1.08	2.198	0.039**
Total Protein (g/dl)	8.30 $\pm$ 2.28	7.28 $\pm$ 1.13	2.089	0.049**
Albumin (g/dl)	4.53 $\pm$ 1.11	4.26 $\pm$ 0.72	1.108	0.280*
Globulin (g/dl)	3.84 $\pm$ 1.75	2.93 $\pm$ 0.64	2.409	0.024**
Creatinine (mg/dl)	0.62 $\pm$ 0.21	0.80 $\pm$ 0.18	4.007	0.000**

\*\*Significant\*Not Significant

Table 4 shows the mean  $\pm$ SD obtained for male and female sickle cell subjects. There was a significant difference ( $P < 0.05$ ) when the mean  $\pm$  SD of serum glucose in sickle cell male subjects 7.29 $\pm$ 1.20 when compared to the female subjects 6.75 $\pm$ 0.96. The mean  $\pm$  SD of serum total protein of male subjects 8.13 $\pm$ 2.58 was lowered when compared to female subjects 8.30 $\pm$ 2.28 but not statistically significant ( $P > 0.05$ ). There was no significant difference ( $P > 0.05$ ) when the mean  $\pm$  SD of serum albumin of sickle cell

male subjects 4.24 $\pm$ 0.69 when compared to the female subjects 4.53 $\pm$ 1.11. The mean  $\pm$  SD of serum globulin of male subjects 3.92 $\pm$ 2.42 was not statistically significant ( $P > 0.05$ ) when compared to the female subjects 3.84 $\pm$ 1.75. The mean  $\pm$  SD of serum creatinine among sickle cell male subjects and female subjects were 0.68 $\pm$ 0.30 and 0.62 $\pm$ 0.21 respectively. When compared statistically there was no significant difference ( $P > 0.05$ ) between values obtained.

Table 4: Mean  $\pm$ SD of Female and Male Sickle Cell Disease (SCD) patients

Parameters	Female SCD	Male SCD	t value	P value
Glucose (mmol/l)	6.75 $\pm$ 0.96	7.29 $\pm$ 1.20	2.630	0.016**
Total Protein (g/dl)	8.30 $\pm$ 2.28	8.13 $\pm$ 2.58	0.342	0.736*
Albumin (g/dl)	4.53 $\pm$ 1.11	4.24 $\pm$ 0.69	1.192	0.247*
Globulin (g/dl)	3.84 $\pm$ 1.75	3.92 $\pm$ 2.42	0.223	0.826*
Creatinine (mg/dl)	0.62 $\pm$ 0.21	0.68 $\pm$ 0.30	1.323	0.200*

\*\*Significant\*Not Significant

## DISCUSSION

Sickle cell disorder is an autosomal recessive disease which results from point mutation in the  $\beta$ - chain of the globin chain. Its clinical severity varies whether it is heterozygous (sickle cell trait) or homozygous (sickle cell anemia) (Wang, 2004). The result of our study shows a significantly increased level of plasma glucose when compared to apparently healthy control subjects. There was significant increase in glucose when the Sickle cell male subjects were compared with the apparently healthy male subjects. Also, the female Sickle cell patients show a

significantly higher level of glucose when compared with the apparently healthy female individuals. This is in tandem with previous report on blood glucose level among Sickle cell patients (Adekile *et al.*, 1985, Osuagwu and Mbeyi, 2007). This increase glucose in Sickle cell patients has been attributed to presence of the Sickle cell gene which make carriers with it more susceptible to impaired glucose metabolism than non- carriers (Osuagwu and Mbeyi, 2007). This impaired glucose metabolism Sickle cell patient's lead to anergy as earlier observed by Markov *et al.*, (2001).

Ehtisham and Timothy (2004) in their study attributed this impaired glucose metabolism to microvasculature and microvasculature complications experience by Sickle cell patients. There was gender difference in the level of glucose with the male sickle cell patients having a significant higher level than the female sickle cell patients. Though, there is hyperglycemia among sickle cell disease patients due to impaired glucose tolerance but not diabetic because it is below the cut – off value of >7.7mmol/l established by Jouvenet *et al.*, (2005).

There was a significantly increase of serum total protein in sickle cell disease patients when compared with apparently healthy individuals. An increase serum total protein was observed when the male sickle cell disease patients was compared with the apparently healthy male subjects but not statistically significant.

Conversely, when the serum total protein of female sickle cell disease patients was compared with apparently healthy female, there was a significantly increase observed, this was in agreement with the reports of previous studies done on sickle cell patients (Edozienet *et al.*, 1960, Isichei 1979, Famoduet *et al.*, 1987, Adenikeet *et al.*, 1998, Adu *et al.*, 2012) but inconsistent with the report of Tripathiet *et al.*, (2011) who reported low levels of total protein in sickle cell disease patients. This hyperproteinemia may be due to hyperglobulinemia as observed in this study among sickle cell disease patients. Adenikeet *et al.* (1998) attributed this hyperglobulinemia to increased erythrocyte destruction during sickling. Adu *et al.*, (2012) attributed the hyperglobulinemia in sickle cell patients to the excessive production of gamma ( $\gamma$ ) globulin fraction which results from antigenic stimulation from the environment as observed by Johnson *et al.*, (1999).

Our results show, an increased levels of albumin in sickle cell disease patients but

not statistically significant when compared with the apparently healthy subjects. This is in tandem with the report of Adu and colleagues (2012) who reported similar trend of albumin in sickle cell disease subjects but in contrast with the report of Tripathiet *et al.*, (2011) who reported low levels of serum albumin in sickle cell disease patients. Famoduet *et al.*, (1987) in their report observed that higher levels of serum albumin in sickle cell disease patients rule out hepatic dysfunction. The increased levels of albumin may be attribution as a result of protein synthesis that occurs during sickling (Isichei 1979). There was no significant difference observed when the male and female sickle cell disease patients were compared with their respective controls. Also there was no gender difference observed in terms of albumin levels in sickle cell disease patients. Creatinine is a popular substance used routinely in the assessment of renal function. Increase in serum creatinine indicates renal impairment and its clearance are reduced. Our results show significant decreased levels of serum creatinine in sickle cell disease patients. Creatinine levels were observed to be significantly lowered when male and female sickle cell disease patients were compared with their respective controls. There was no gender difference between the male and female sickle cell subjects with respect to what???. This reduced levels of creatinine observed in sickle cell patients may be attributed to reduced muscle mass seen in Sickle cell anaemia patients (Odonkoret *et al.*, 1984).

Conclusively, it has been shown that glucose; total protein, albumin as well as creatinine are altered in sickle cell disease patients who need to be investigated on routine basis to prevent morbidity and mortality. We therefore advocate prompt diagnosis and as well as proper care for sickle cell patients at all times.

**Conflict of Interest:** None

**Source of funding:** None

Authors Contribution: JJD design the research and analysed samples as well as

proof read the manuscript, AME did statistical analysis and draft the manuscript and DAK analysed samples and proof read the manuscript.

## REFERENCES

- Adekile AD, Olusi SO and Oyebola DD (1985). Oral Glucose Intolerance Test in Children With sickle Cell Anaemia. *East African Medical Journal*, 62(3): 213-217.
- Adenike F A, Bakare A R, Fajimi JL, Ogunyemi EO, Obisesan KA and Sokoya G (1998). Serum protein levels in sickle cell disease. *African Journal Medicine & Pharmaceutical Science*. 1: 40-43.
- Adu EM, Okosun RE, Bini E N, and Ophori EA (2012). Effects of the sickle cell (s) gene on serum protein profile. *Continental Journal Biomedical Sciences* 6 (2): 1 – 5.
- Ballas VV, Kalinyak KA and Bean JA (2002): Hyperhomocysteine.nia is associated with low plasma pyridoxine levels in children 'with sickle cell disease. *Journal Pediatric Hematology Oncology*. 24(5):374-379.
- Brod J and Sirota JH (1948).The renal clearance of endogenous creatinine in man.*Journal Clinical Investigations*, 27(5): 645- 654.
- Doumas BT, Bayse D, Borner K, Carte RJ., Peters T. Jr., Schaffer R (1981). A candidate reference method for determination of total protein in serum: 1. Development and validation. *Clinical Chemistry*; 27: 1642.
- Edozien JC, Boyo AE and Morley DC(1960). The relationship of serum Gamma globulin concentration to malaria and sickling.*Journal Clinical Pathology*, 13: 118-123.
- Ehtisham S and Timothy GB (2004).Emergence of type 2 diabetes in childhood.*Annals Clinical Biochemistry*, 41:10-16.
- Emokpae MA and Tijani AD (2014).The impact of proteinuria on serum levels of trace elements in sickle cell disease patients.*Journal of Medical and Biomedical Sciences*, 3(3): 16-20
- Famodu A. A. Omodiale P. Adedeji M. O. and Reid H. L. (1987).Serum protein study in adult Nigerians with sickle cell anaemia.*Medical Science Research*, 15: 193-194.
- Isichie U P(1979). Serum proteins profile in sickle cell disease. *Journal Clinical Pathology*, 32: 171-121.
- Johnson AMRohif EM and Silverman LM(1999). Proteins.In Teltz Textbook of clinical chemistry (3<sup>rd</sup>ed).Ashwood E.R. Butris C. A. (eds) W .B Sanders Philadephia. Pp 350- 615.
- Jouven X, Rozenn NL, Thomas D, Rea NS, Jean PE and David SS(2005). Diabetes, glucose level and risk of sudan cardiac death. *European Heart Journal*, 26(20): 2142- 2147.
- Markov AK, Fox AW and Marangos PJ (2001). Treatment of Sickle Cell Anemia Crisis with Fructose-1, 6-Diphosphate As An Analgesic Drug; <http://freepatentsonline.com/6312707.html>. Questcor Pharmaceuticals Inc.
- Odonkor PO, Addae SK, Yamamoto S, Apatu RS (1984).Effect of dietary nitrogen on urinary excretion of non-protein nitrogen in adolescent sickle cell patients. *Human Nutrition & Clinical Nutrition*, 38(1):23-29.
- Osuagwu CG and Mbeyi CU (2007).Altered plasma hexose sugar metabolism in sickle cellanaemia. *African Journal of Biochemistry*,1 (3) : 37-40
- Panzer C, Lauer MS, Brieke A Blackstone E and Hoogwerf B (2002). Association of plasmagluose with heart rate recovery in healthy adults: A population based study of diabetes. *Diabetes*, 51: 803-807.

- Steinberg MH, Barton F, Castro O, Pegelow CH, Ballas SK, Kutlar A, Orringer E, Bellevue R, Olivieri N, Eckman J, Varma M, Ramirez G, Adler B, Smith W, Carlos T, Ataga K, DeCastro L, Bigelow C, Sauntharajah Y, Telfer M, Vichinsky E, Claster S, Shurin S, Bridges K, Waclawiw M, Bonds D, Terrin M (2003): "Effect of hydroxyurea on mortality and morbidity in adult sickle cell anemia: risks and benefits up to 9 years of treatment". *Journal of American Medical Association*; 289 (13): 1645-5116.
- Tripathi S, Dadsena R, and Kumar A (2011). Study of Certain Biochemical Parameters in Patients of Sickle Cell Anemia. *Advance Bioresearch*, 2(2):79-81.
- Wang W, Elliot-mills D. and Powars D. (2004). Renal failure in sickle cell Anemia. *Hematology & Oncology Clinics of North. America*, 10(6): 1321- 1331