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Sickle Cell Anaemia: A Prevalence Study among the Children Attending Usmanu Danfodiyo University Teaching Hospital, Sokoto, North-Western Nigeria

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Authors' contributions

This work was carried out in collaboration between all authors. Authors NMJ and KKI designed the study, performed data analysis and wrote the first draft of the manuscript respectively. Authors KM, OE and ASM managed the data collection and compiled the data. Authors MAN, AG and AUM managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Aims: The aim of this study was to determine prevalence of Sickle Cell Anaemia (SCA) and other abnormal haemoglobin variants among the children attending the Paediatrics outpatients Department of Usmanu Danfodiyo University Teaching Hospital (UDUTH), Sokoto, North-Western of Nigeria.
Study Design: This was a cross-sectional study designed to investigate the prevalence of Sickle

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Cell Anaemia and other variants of abnormal haemoglobin among 395 children attending Paediatrics Outpatients Department of UDUTH, Sokoto.

Place and Duration of Study: Paediatrics Outpatients Department of UDUTH, Sokoto, North Western Nigeria, between January and April, 2016.

Methodology: A total of three hundred and ninety-five (395) children aged 6 months -15 years were recruited for this study. Their haemoglobin electrophoresis patterns were determined at alkaline pH (8.6) using the method described by the Helena Biosciences procedure.

Results: Of the three hundred and ninety-five (395) children recruited for this study, the prevalence of haemoglobin electrophoresis patterns recorded were HbAA, HbAS, HbSS, HbAF, HbSS+F, HbAC and HbSC, that is; 70.0%, 15.2%, 5.0%, 0.3%, 6.0%, 2.0%, and 1.5% respectively. Hence the prevalence of SCA among the study subjects was 11% (5.0% HbSS + 6.0% HbSS+F).

Conclusions: While HbAA is the predominant haemoglobin electrophoresis pattern in our environment, there is also a significant number of sickle cell anaemia that is of every 10 children in the study, one has SCA. It is necessary therefore, to keep abreast with developments in the area of its management in order to cope with the challenges.

Keywords: Sickle cell anaemia; prevalence; children; Sokoto; North-Western Nigeria.

1. INTRODUCTION

Haemoglobin genotypes are inherited blood characters. The inherited disorders of haemoglobin are the most common genetic disorders worldwide, with 7% of the world's population being carriers [1]. It is on record that about 300,000 children are born with sickle cell disease (SCD) globally every year [2]. The most common are the sickle cell disorder and the thalassaemias, occurring in people of African, Asian, South European and Middle Eastern descent. These sickling disorders include the heterozygous state for haemoglobin (HbAS), the homozygous for haemoglobins (HbSS) and the compound heterozygous state for haemoglobins together with haemoglobin C, D, E or other structural variants [3].

Haemoglobins S differs from haemoglobin A by the substitution of valine for glutamic acid at position 6th in the β chain ($\alpha_2\beta_2^{6\ Glu\ .....\ Valine}$) [3]. The Substitution of glutamic acid for lysine at position 6 of the globulin chain ($\alpha_2\beta_2^{6\ Glu.....Lys}$) also occurs in the haemoglobin (HbC) genotype [4]. In haemoglobin D-punJab (HbD) glycine is substituted for glutamic acid at position 121st in the β globulin chain ($\alpha_2\beta_2^{121\ Glu.....Gly}$) while in haemoglobin E (HbE) lysine is substituted for glutamic acid at the 26th position of the β globulin chain ($\alpha_2\beta_2^{26\ Glu.....Lys}$) [5].

Subjects with heterozygous state for Hb (HbAS) and (HbAC) are asymptomatic but may present with a mild microcytosis with relative red cell resistance to haemolysis and malaria. Subjects homozygous for HbC (HbCC) usually have compensated haemolysis with splenomegaly. There is an increased risk of hypersplenism, biliary lithiasis, folate deficiency and worsening of anaemia following some infections (parvovirus) [6]. The association of HbCC with β thalassaemia, especially with β^+ thalassaemia (more common than β^o thalassaemia) in the ethnic groups concerned by (HbCC) results in a clinical pictures similar to that of HbCC. Cβ^o thalassaemia is more severe and can mimic beta thalassaemia intermedia. Compound heterozygotes HbSC presents with a sickling disorder similar to sickle cell anaemia (SCA), although it is generally milder than in the HbSS form. However, 2% of HbSC patients have more severe disease with frequent vaso-oclusive crisis (VOC) and acute chest syndrome (ACS) [7].

Sickle cell anaemia is one of the most common single gene disorders in man with variable distributions in different parts of the world and variable clinical manifestation [8]. Sickle cell anaemia commonly affects growth, leading to low mean weight, low mean height and decreased height velocity [9].

Africa has 70% of the world's annual figure of 300,000 new births affected by SC [10]. In Nigeria the prevalence of sickle cell trait is about 25% while the homozygous state is found in about 3% of the population [11]. Nigeria has the largest population of people with sickle cell disorder, with about 150.000 births annually [12]. Sickle haemoglobin (HbS) is present at birth, but most infants don't show signs until they are six months or shortly before, because the predominant haemoglobin at this time is foetal haemoglobin (HbF) [8]. It has been earlier documented that high level of HbF inhibits

sickling in babies with SC [13]. This is because HbF has the ability to decrease the polymerization of deoxygenated HbS, hence preventing red blood cell from forming tactoids which lead to vaso-occlusion [14]. Sickle cell anaemia accounted for 8.2% of all admission and about 24.6% of those with severe anaemia, in a study at the children's emergency ward, university college hospital, Ibadan [10]. This confirmed that SCA is an important cause of severe anaemia [15]. The frequencies of these inherited characters have been reported to vary significantly in various populations and ethnic groups around the world. In Nigeria, few recent published data have been encountered, but none in Sokoto, North-West of Nigeria. This study was, therefore, designed to provide the frequencies of haemoglobin variants for reference purposes using children attending the Paediatrics Department of Usmanu Danfodiyo University Teaching Hospital (UDUTH) Sokoto, Nigeria. Dissemination of the study findings, screening, pre-marriage counselling, and pre-natal diagnosis are fundamental to preventing or lowering the birth of children with sickle cell anaemia in the affected populations [16].

2. MATERIALS AND METHODS

2.1 Study Area

This study was carried out in the Paediatrics Department of Usmanu Danfodiyo University Teaching (UDUTH) Sokoto, North-Western Nigeria. Sokoto State is located in the extreme North- West of Nigeria, near the confluence of the Sokoto River and the Rima River. The State is located between longitude 11'30°, 13'50° East and latitude 4'to 6'0° North of the Equator. It is bordered to the North by Niger Republic, Zamfara State to the East, while Kebbi State borders most of the South and Western parts [17]. The major indigenous tribes in the state are the Hausa and Fulani and other groups such as Gobirawa, Zabarmawa, Kabawa, Adarawa, Arawa, Nupes, Yorubas, Ibos and others. The Majority of the Hausas' are farmers while Fulanis are nomadic and are engaged in animal rearing [18]. Based on 2006 population census, Sokoto State had a population of 3,696,999 [19], with an average estimate of 4,806,098 in 2015 based on the population annual growth rate of 3% [20].

2.2 Study Population

A total of three hundred and ninety-five (395) children aged 6 months - 15 years were recruited for this study. The subjects were recruited from Paediatrics Outpatient Department of Usmanu Danfodiyo University Teaching Hospital, (UDUTH) Sokoto, North- Western Nigeria. The age of 15years is considered as the upper age limit for Paediatrics patients in UDUTH, Sokoto [21].

2.3 Study Design

This was a cross-sectional study designed to investigate the prevalence of Sickle Cell Anaemia and other variants of abnormal haemoglobin among 395 children attending the Paediatrics Outpatients Department of UDUTH, Sokoto, North Western Nigeria, between January and April, 2016.

2.4 Methods

About 3 millilitres (mL) of blood was collected from the subjects into the K_3 EDTA containers. Haemoglobin electrophoresis was carried out on the samples at an alkaline pH of about 8.6, using the method as described in the Helena BioSciences procedure by Monica Ceesbrough, 2008. Leishman's stained blood films were also examined for red cells morphology; these would give additional pictures of SCA (sickle cells in the blood film).

2.4.1 Inclusion criteria

The inclusion criteria for this study included; children aged (6 months to 15 years), attendance at the Paediatrics Wards of Usmanu Danfodiyo University Teaching Sokoto, North-Western Nigeria.

2.4.2 Exclusion criteria

The following exclusion criteria were used; individuals above 15 years, known sickle cell anaemic children, children less than five months and children whose parents/guardians refused to sign a written informed consent for their wards to participate in the study as subjects.

2.5 Statistical Analysis

The data collected were entered into the data editor of statistical package for social sciences (SPSS Version 22) software. Analysis was based on simple percentages, or proportions and values were expressed as Mean ± SD. A Chi-square test at a 95% confidence level was also used to test for association between aged

groups, anaemia and gender. A p-value of < 0.05 was considered as significant in all statistical analysis.

3. RESULTS

This research work examined haemoglobinopathies of the three hundred and ninety-five children attending the Paediatrics Department of UDUTH, Sokoto, North-Western Nigeria. In the Table 1 is shown that Out of the 395 children tested, 220 (55.7%) were male while 175 (44.3%) were female giving an M: F ratio of 2:1. The children included; pre primary school (6 months - 5.0 years), schooled (5.1-10.0 years) and post primary school (10.1-15.0 years).

The results in Table 2 indicated that the females were more affected with SCA than male children 24 and 20 respectively but there was no statistically significance difference.

Table 3 shows that the younger children (6 months - 5.0 years), still had the highest prevalence of SCA, which may be due to lack of awareness of sickle cells and the low level of education of the parents.

Table 1. Age and gender distribution of the study population

Age (years)	Gender		Total
	Male	Female	
6 months – 5.0	122(30.9%)	101(25.6%)	223(56.5%)
5.1 – 10.0	62(15.7%)	35(8.8%)	97(24.5%)
10.1 - 15.0	36(9.1%)	39(9.9%)	75(19.0%)
Total	220(55.7%)	175(44.3%)	395(100.0%)

X^2 = 4.47; df = 2 p-value = 0.107 df = degree of freedom

Table 2. Gender comparison of haemoglobinopathies among the study population

Haemoglobin	Gender		Total
	Male	Female	
AA	150(38.0%)	126(32.0%)	276 (70.0%)
AS	40 (10.1%)	20 (5.1%)	60 (15.2%)
AC	5 (1.2%)	3 (0.8%)	8 (2.0%)
AF	1 (0.2%)	0 (0%)	1(0.2%)
SC	4 (1%)	2 (0.5%)	6 (1.5%)
SS	12 (3%)	8 (2%)	20 (5.0%)
SS+F	8 (2%)	16 (4.1%)	24 (6.1%)
Total	220 (55.5%)	175 (44.5%)	395(100.0%)

X^2 = 10.782; df = 8, p-value = 0.214. A = Normal Haemoglobin, S = S Haemoglobin (trait), C = C Haemoglobin (trait), F = foetal Haemoglobin, AA = Healthy, AS = Carrier, SS = Sickle Cell Anaemia, SC = Sickle Cell Diseases

Table 3. Age comparison of haemoglobinopathies among the study population

Hb electro pattern	Age (months and Years)			Total
	6 months – 5.0	5.1 10.0	10.1 15.5	
AA	164(41.5%)	63(15.9%)	49(12.4%)	276(69.9%)
AS	28(7.1%)	20(5.1%)	12(3.1%)	60(15.1%)
AC	7(1.8%)	0	1(0.3%)	8(2.0%)
AF	1(0.3%)	0	0	1(0.3%)
SC	2(0.5%)	1(0.3%)	3(0.8%)	6(1.5%)
SS	7(1,8%)	8(2.0%)	5(1.2%)	20(5.1%)
SS+F	14(3.5%)	5(1.2%)	5(1.25%)	24(6.1%)
Total	223(56.5%)	97(24.5%)	75(19.0%)	395(100.0%)

X^2 = 20.01; df = 16, p-value = 0.220. Hb = haemoglobin, electro = electrophoresis, A = Normal Haemoglobin, S = S Haemoglobin (trait), C = C Haemoglobin (trait), F = foetal Haemoglobin, AA = Healthy, AS = Carrier, SS = sickle cells, SC = sickle Cell Diseases

Table 4. Age and gender comparison of haemoglobinopathies among the 139 anaemic children

Hb ELT	Gender	Age (years)			Total	X^2	Df	p-value
		6 months - 5.0	5.1-10.0	10.1-15.0				
AA	Male	29	12	8	49	0.61	2	0.780
	Female	14	4	5	23			
AC	Male	3	1	0	4	0.60	1	0.439
	Female	2	0	0	2			
AS	Male	5	7	1	13	0.94	2	1.00
	Female	1	2	1	4			
AF	Male	1	0	0	1			
	Female	0	0	0	0			
SC	Male	0	1	1	2	3.00	2	0.22
	Female	1	0	0	1			
SS	Male	4	5	2	11	1.18	2	0.55
	Female	3	2	3	8			
SS+F	Male	4	2	1	7	0.54	2	0.761
	Female	7	3	4	14			
Total		74	39	26	139			

Hb ELT = Haemoglobin Electrophoresis, A=Normal Haemoglobin, S = S Haemoglobin (trait), C = C Haemoglobin (trait), F = foetal Haemoglobin, AA = Healthy, AS = Carrier, SS = sickle cells, SC = sickle Cells Diseases

Table 4 (above) shows that female children aged 6 months -5.0 years had highest prevalence of sickle cell anaemia compared to the male anaemic children but the p values were not statistically significance ($p<0.05$).

4. DISCUSSION

Anaemia in this study was defined based on the World Health Organisation (WHO) criteria of haemoglobin values of less than 11g/dl; mild anaemia (9.0–10.9 g/dl), moderate anaemia (7.0–8.9 g/dl) and < 7.0 g/dl (severe anaemia) [22]. The global prevalence of anaemia in school-age and preschool-age children is 25.4% and 47.4% respectively [23]. It affects 293 million children globally with the highest prevalence found in Africa (67.6%) [24].

Three hundred and ninety-five subjects were tested for haemoglobinopathies In this study, the recorded frequencies of HbAA, HbAS, HbSS, HbAF, HbSS+F, HbAC and HbSC were 70.0%, 15.2%, 5.0%, 0.3%, 6.0%, 2.0%, and 1.5% respectively. These findings were consistent with the previous reports, in which the prevalence of 70.0%, 23.3%, 4.8%, 1.3%, 0.8% were observed for HbAA, HbAS, HbSS, HbAC, and HbSC respectively [25]. Similarly, a prevalence of 69.4%, 26.9%, 3.5% 0.1%, and 0.02% of HbAA, HbAS, HbSS, HbAC, and HbSC respectively were observed in Anambra [26], 71.0%, 22.1%, 0.5%, 5.3%, and 0.8% was observed for HbAA, HbAS, HbSS, HbAC, and HbSC respectively, in Ogbomoso [27]. The observed prevalence of HbAA is within the normal range of 55 – 75% earlier reported for blacks [28]. The results from these current findings revealed a prevalence of 5.0% HbSS + 6.0% HbSS+F (11.0%) among the study subjects. These findings are however, higher compared to the other published reports in Nigeria; 3.0% in the South-West region of Nigeria [29], 2% among undergraduate students in Bayelsa State [10,11, 30] and 3% in Rivers State [31] both in the South-South of Nigeria. These present findings are however, at variance with previous report in Kenya, East Africa [32] and among 620 University students in Port Harcourt Nigeria [33], which both obtained a 0% prevalence of HbSS. The zero frequencies observed in these studies, possibly imply that the sickling gene pool is gradually reducing in some African populations due to increase awareness and pre-marital counselling. The low prevalence of HbSS observed in those studies could be attributed to increased awareness of the disease, improved socio-economic conditions, improved pre-marital counselling, environmental and genetic factor which have an overall effect on the sickling gene pool. The zero prevalence may also be attributed to an active program of prenatal diagnosis among pregnant women in Nigeria and the survival rate of sickle cell anaemic children may decrease with age (newborn to adult).

The finding from the current study are consistent with prevalence of HbAS observed among the black population in the United States, which was reported to be 9% and 30%–40% generally for

Africans [34-35]. Sickle haemoglobin (HbS) is the most common and clinically significant haemoglobin structural variant. In this series the recorded zero percent of HbCC is slightly at variance with the previous findings: 0.24% in Akwa Ibom [36], 0.18% in Ogbomoso by Akhigbe *et al*., 2009 and 0.01% in Anambra by Uzoegwu and Onwurah, 2003. The zero frequencies observed in our studies, may be attributed to sample size and other environmental conditions.

The number of people with homozygous HbSS for both males and females, respectively in Sokoto is high. The reason for this high prevalence may be due to the absence of carrier testing programs and premarital counselling/ testing for prospective couples prior to marriage, in a bid to reduce the prevalence of haemoglobinopathies in the area. Other factors such as persisting high concentration of foetal haemoglobin could invariably influence the prevalence of SCA in a population. It can be an effective way to diagnose and monitor the trend of haemoglobinopathies in the state. Evidenced-based data from Belgium, a country with universal neonatal screening programme has shown that neonatal screening is an excellent health education tool [37].

5. CONCLUSION

In conclusion, the recorded prevalence of 11.0% SCA among the study children is high. The number of children with SCA in both male and female in Sokoto is also high. Sokoto State in particular and Nigeria in general, can benefit from universal neonatal screening program for sickle cell disease which may be an effective way to diagnose and monitor the trend of haemoglobinopathies in the state.

ETHICAL CONSIDERATION

This study was approved by Health Research and Ethics Committee of UDUTH, Sokoto, North-western Nigeria.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. World Health Organization (WHO). Evaluation (Phase 1) of the Uni-Gold™ HIV using Whole Blood Specimens. 2005; 5-12.
2. Bakare AA, Azeez MA, Agbolade JO. Gene frequencies of ABO and Rhesus blood groups and haemoglobin variants in Ogbomosho, South - West, Nigeria. Global Journal of Medical Science. 2004;3:17–22.
3. Fleming AF, Lehman H. Sickle cell disease: A handbook for general clinician. Edinburgh: Churchill Livingstone 1982:10-17.
4. Monica C. District laboratory practice. 2nd edition. Cambridge University Press. 2008;337-340.
5. Nwafor A, Bamigo BM. A comparison of measured and predicted hemoglobin genotype in a Nigerian population in Bonny, Rivers State, Nigeria. Journal of Applied Science and Environmental Management. 2001;5:79–81.
6. Okpala I, Thomas V, Westierdale N, Jegede T, Ray K. Comprehensive care of sickle cell disease. European Journal of Haemotology. 2002;68:157-162.
7. Richard AW. Textbook of black related diseases. New York, USA: McGraw Hill; Lewis RA. Sickle cell: Clinical features in West Africans. Accra, Ghana: Ghana University Press. 1970;5-15.
8. Serjeant GR, Serjeant BE. Sickle cell diseases 3rd edition. New York: Oxford University Press; 2001.
9. Ogunrinde GO, Yakubu AM, Akinyanju OO. Anthropometric measures and zinc status of children with sickle cell anaemia in Zaria. Nigerian Journal of Paediatrics. 2000;27:64-9.
10. Makani J, Konba AN, Cox SE, Oruo J, Mwamtemi K, et al. Malaria in patients with sickle cell anaemia: Burden, risk factors, and outcome at the outpatient clinic and during hospitalization. Blood. 2010;115-220.
11. Adekile AD, Adeodu OO. Haemoglobinopathies. In: Azubuike JC, Nkanginieme KEO. (editors). Textbook of Paediatrics and ChilHealth in a Tropical Region. 2nd ed. Owerri: African Educational Services. 2007;373-90.
12. World Health Organization. Report by the secretariat of the fifth-ninth World Health Assembly A59/9; 2006.
13. Anie KA, Egunjobi FE, Akinyanju OO. Psychosocial impact of sickle cell disorders; perspective from a Nigerian

setting. Globalization and Health. 2010;6: 1-6.

14. Edoh D, Antwi-Bosaikoc AD. Foetal haemoglobin during infancy and in sickle cell adults. African Health Science. 2006;6: 51-54.
15. Akodu SO, Indiaku-Akinwumi, Njokanma OF. Age at diagnosis of sickle cell anaemia in Lagos, Nigeria. Mediterrenian Journal of Haematological Infectious Diseases. 2013;5(1):e2013001.
DOI: 10.4084/MJHID.2013.001
Available:http://www.mjhid.org/article/View/10393
16. Yadav R, Lazarus M, Ghanghoria P, Singh M, Gupta RB, Kumar S, Sharma RK, Shanmugam R. Sickle cell disease in Madhya Pradesh, central India: A comparison of clinical profile of sickle cell homozygote vs. sickle-beta thalassaemia individuals. Hematology. 2016;21(9):558-63.
17. Pavord S, Myers B, Robinson S, Allard S, Strong J, Oppenheimer C. British Committee for Standards in haematology. UK guidelines of the management of iron deficiency in pregnancy. British Journal of Haematology. 2012;156(5):588-600.
18. Tower J, Butler R. Modern obstetric for midwives. 2th edition. Lloyd Duke Ltd., London. 2006;219-225.
19. Nemeth E. Ganz T. Regulation of iron metabolism by hepcidin. Annual Revision Nutrition. 2006;26:323-342.
20. Ernest SK, Anunobi NE, Adeniyi A. Correlates of emergency response interval and mortality from severe anaemia in childhood. West African Journal of Medicine. 2002;21:177-179.
21. Jiya NM, Isezuo KO, Waziri UM, Jangebe MA. Severe malaria anaemia in children. A 22 – month retrospective review; 2014.
Available:www.panconf2015.org/wp - Content/.../322 - ABSTRACT SMA
22. World Health Organization (WHO). The global picture of anaemia. Technical report series. 2001;15-23.
23. World Health Organization (WHO). Worldwide prevalence of anaemia 1993 – 2005. WHO global database on anaemia. Geneva Switzerland. 2005;71:16-24.
24. World Health Organization (WHO). Worldwide prevalence of anaemia 1993– 2005. Geneva. 2008;1-25.
25. Umar RA, Jiya NM, Ladan MJ, Abubakar MK, Hassan SW, Nata'ala U. Low prevalence of anaemia in a cohort of pre-school children with acute uncomplicated Falciparum malaria in Nigeria. Trends in Medical Research. 2007;2:95-101.
26. Mannir S. Anaemia as seen in Hassan Usman Hospital, Katsina. Biological Environment Science Journal Tropical. 2006;3:73-75.
27. deBenoist B. Focusing on anaemia - towards an integrated approach for effective anaemia control. 2004;10-12.
28. Akinkugbe FM. Prevalence of anaemia in the urban population (University College Hospital GOPD, Ibadan. Journal of Tropical Paediatrics. 1977;23:271-275.
29. Mousa ZA. A comparison of selected nutrient intakes in anaemic and non-anaemia adolescent girls in Kuwait. Journal of Nutrition Research. 2003;23: 425-433.
30. Yakubu A, Zama II, Osaro E, Sanusi BM, Frank PU, Ezimah AC, Yeldu M, Abdulahi, W, Emmanuel KU, Teddy CA, Iwueke IP, Dorcas I, Festus A, Osazuwa I, Isah AB. Haemoglobin electrophoretic pattern among resident in Sokoto, Nigeria. Journal of Medical Disorder; 2013.
DOI: 10.7243/2053-3659-1-2
31. Uzoegwu PN, Onwurah AE. Prevalence of haemoglobinopathy and malaria diseases in the population of Old Aguata Division, Anambra State, Nigeria. Nigeria Society for Environmental Biology. Biokemstr. 2003;15(2);57-66.
32. Akhigbe RE, Ige SF, Afolabi AO, Azeez OM, Adegunlola GJ, Bamidele JO. Prevalence of haemoglobin variants, ABO and rhesus blood groups in Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria. Trends in Medical Research. 2009;4:24-29.
33. Egesie UG, Egesie OJ, Usar I, John-bull TO. Distribution of ABO, Rhesus blood and haemoglobin electrophoresis among the undergraduate students of Niger Delta State University Nigeria. Nigerian Journal of Physiological Science. 2008;23:5-8.
34. Moormann AM, Embury PE, Opondo J, Sumba OP, Ouma JH, Kazura JW, John CC. Frequencies of sickle cell trait and glucose-6-phosphate dehydrogenase deficiency differ in highland and nearby lowland malaria-endemic areas of Kenya. Trans Royal Society of Tropical Medicine and Hygiene Journal. 2003;97:513-514.

35. Jeremiah ZA. Abnormal haemoglobin variants, ABO and Rh blood groups among student of African descent in Port Harcourt, Nigeria. African Journal of Health Science. 2006;6:177-81.
36. Gulbis B, Vertongen F, Ferster A. Neonatal screening and clinical care programmes for sickle cell disorders in Sub-Saharan Africa: Lessons from pilot studies. Journal of Public Health. 2009; 122:933-941.
37. Sinou MT. Antenatal screening of sickle cell disease. 8th postgraduate course for training in reproductive medicine and reproductive biology. Cameroon. 2003; 10-21.

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