Science Journal of Public Health
2017; 5(5): 388-391
http://www.sciencepublishinggroup.com/j/sjph
doi: 10.11648/j.sjph.20170505.15
ISSN: 2328-7942 (Print); ISSN: 2328-7950 (Online)

Review Article

Antioxidant Vitamins A and E in Relation to BMI in Steady State Sickle Cell Anaemia and Normal Controls in North Eastern Nigeria

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To cite this article:

Tukur Maisaratu Aminu, Salami Hamza Adegoke, Ibrahim Bilal Muhammad, Abubakar Abdulwasi'u, Shehu Binta Baba, Ambe Jose Pwavimbo. Antioxidant Vitamins A and E in Relation to BMI in Steady State Sickle Cell Anaemia and Normal Controls in North Eastern Nigeria. *Science Journal of Public Health*. Vol. 5, No. 5, 2017, pp. 388-391. doi: 10.11648/j.sjph.20170505.15

Received: April 28, 2017; **Accepted**: July 17, 2017; **Published**: August 14, 2017

Abstract: Sickle cell anaemia is one of the commonest causes of anaemia in sub-Saharan Africa. It causes significant morbidity and mortality, commoner in black Africa, but due to increase medical care, the life expectancy is on increase. Several studies have been carried out on sickle cell anaemia (SCA) nationally and internationally. This present study determined the BMI of SCA patients in the steady state compared to normal control in the north-eastern Nigeria. A cross-sectional study was carried out at the University of Maiduguri Teaching Hospital (UMTH) as a referral center. Undergraduate students, secondary and primary school students of the University of Maiduguri Borno state were incorporated in the study as controls. A total number of 120 subjects were enrolled into the study constituting 60 subjects with homozygous SS, and 60 controls who are homozygous AA. Random sampling technique was employed in the selection of the subjects that attends the sickle cell haematology clinics both in adults and paediatrics that were at their steady state. BMI of the SCA were found to be either normal weight (18.5-24.9kg/m^2) or underweight (<18.5kg/m^2), while the subject with normal haemoglobin genotype showed overweight (25-29.9kg/m^2) and obese (>29.9kg/m^2) in addition to underweight and normal weight. Antioxidant vitamins A and E were also found to be low in SCA patients compared to the normal controls In conclusion, we therefore concluded that overweight and obese is very rare in patients with sickle cell anaemia.

Keywords: Vitamin A, Vitamin E, Basal Metabolic Index, Sickle Cell Anaemia

1. Introduction

Sickle cell disease (SCD) is an inherited disorder of haemoglobin synthesis that is associated with significant morbidity and mortality due to sequelae of episodic vaso-occlusive events: pain crisis and multi organ damage [1]. Sickle cell aneamia (SCA) results from the substitution of valine residue for glutamic acid at position 6 of the beta sub unit haemoglobin [2]. People with only one gene for HbS are phenotypically normal (sickle cell trait) [3] while those who inherited two HbS genes from both parents have SCA. The common theory is the association of this disease in the tropics with malaria. In 1949, Beet, a British medical officer stationed in Northern Rhodesia (now Zimbabwe), observed that blood from malaria patients who had sickle cell trait had fewer malaria parasites than blood from other patients. In Nigeria, heterozygous (AS) carrier rates is about 25% in the south and 18-32.6% in the North. In the North East the highest frequencies of Hb AS have been recorded among the Kanuris, (27.9%) of Borno state and Bades (32.67%) of Yobe

state while in the North West the Garkis (28.9%) of Kano state. The Ibos (24.3%) in the South East and the Yorubas (23.7%) in the South West, having the lowest frequencies [4], [5], [6], [7], [8].

Sickle cell anaemia is a systemic disorder with clinical features of acute episodes of pain, stroke, priapism and acute chest syndrome and chronic organ damage, e.g osteonecrosis, renal failure and chronic haemolytic anaemia [9]. The term sickle cell disease (SCD) is used in a generic sense to refer to all the clinically severe haemolytic syndromes [10]. The sickle cell gene is fairly evenly distributed throughout.

The molecular basis of sickling is that the deoxygenation of HbS leads to a conformational change that exposes a hydrophobic patch on the surface of the β-globin chain at the side of β valine. Binding of this side to a complementary hydrophobic site on a β-subunit of another haemoglobin tetramer triggers the formation of large polymers [11]. The blockade of blood flow produces areas of tissue ischaemia, leading to myriad of clinical problems seen with SCA. Also the consequences of the formation of these rigid cells include increased mechanical fragility and increased destruction of the RBCs predominantly in the extravascular sites [12]. The erythrocyte of these patients undergo a series of sickling-unsickling cycles before it becomes irreversibly sickled which usually occurs at the venous end of capillaries while unsickling occurs at the arterial end [12].

Several efforts have been made since this disease was described in 1910 in terms of clinical care (hospital admission, painful crisis, acute chest syndrome etc.), the under nutrition and weight as a complication SCA was not given attention until late 1980's [13], [14], [15]. Under nutrition was identified as a critical feature of sickle cell anaemia [16], [17], [18], [19], [20], [21], and the mechanism is being a decreased of intestinal malabsorption and increased catabolism [22]. In addition to the adequacy of diet that decrease with age [16], high level of IL-6 in sickle cell patients which adversely act on the brain to cause appetite suppression and decrease food intake [23], [24], [25]. Recently increase metabolic requirement (hyper metabolism) and relative nutrient shortage found in HbSS can be accounted for by the characteristic haemolytic anaemia [26]. Low circulating level of antioxidant vitamin A and E have been reported in SCA patients [27], [28], [29]. Despite that no clinical benefit recorded for supplementing of these micronutrients. This study was conducted at the University of Maiduguri teaching hospital and state specialist hospital Borno state of Nigeria with residents of north east. Also the environment is less friendly due to extreme temperature. Though this work did not dwelled on effect of diet on sickle cell patient, we compared basal metabolic index (BMI) of sickle cell patient socio-economic status of the family and anti-oxidant vitamin in a view to obtain correlation in this regard.

2. Methodology

A cross sectional study of steady sickle cell anaemia patients (HbSS) at University of Maiduguri Teaching Hospital (UMTH) and state specialist hospital was carried out. The subjects were from Haematology/Paediatric sickle cell anaemia clinic. The non-sickle cell subjects (HbAA) were from University of Maiduguri pre-primary, primary, secondary and undergraduate levels. A total number of 120 subjects were enrolled into the study constituting 60 subjects with sickle cell anaemia (homozygous with SS) who were in the steady state, and 60 controls who were homozygous AA, of both sexes (males and females) with age range between 1.3 to 35 years. The sickle cell group comprised 29 male (48.3%) and 31 females (51.66%) with age range of one year three months and 33 years, while the normal group comprised 40 males (66.6%) and 20 females (33.3%) with their age range between 3 years and 35 years.

Determination of BMI

The weight and height of the patients were measured and BMI was calculated using the formula. BMI= Weight /Height^2.

The data was collected and collated into a statistical package for social sciences (SPSS) version 16 for the analysis of the various parameters. All values were expressed as the mean ± SD and Z-test was used to obtain relationship between individual parameters in relation to experimental and control groups. The results obtained are presented in tables. Values less than 0.05 were considered significant while values greater than 0.05 were considered insignificant at a confidence level 95%.

3. Results

The results from this study showed that SCA subjects have underweight and normal weight while the control group in addition have overweight and obese. The Vitamin A of sickle cell subjects with normal weight was 0.049mg/ml ± 0.003 and underweight was 0.050 ± 0.002. The difference between these two values was not statistically significant (P>0.05) while the vitamin A of control group with the normal weight and obese were 0.070 mg and 0.090mg respectively. (Table 1).

The serum vitamin E level of SCA patients with normal weight was 0.066 ± 0.044mg/ml, while underweight was 0.073 ±0.033 mg/ml. the difference between this two were not statistically significant (P>0.05). Vitamin E level was significantly higher (P<0.05) in the control with normal weight (0.103 ± 0.009 mg/ml) and underweight (0.085 ± 0.002 mg/ml) respectively, when compared with SCA subjects with normal weight (0.066 ± 0.002 mg/ml) and underweight (0.073 ± 0.009 mg/ml) respectively. Over weight and obese were not recorded in SCA patients (Table 1).

4. Discussion

It has been advocated that growth and development is a complex process that is influenced by factors such as genetics, environmental and abnormal growth secondary to some chronic disease. The BMI of SCA patients were either underweight or had normal weight. The present result is in

agreement with other workers who observed that in SCA the incidence of underweight is more prominent [30], [31]. Prevalence of underweight may be attributed to repeated episodes of crisis, excessive RBC haemolysis and anaemia. Anaemia stimulates more production of RBC's, which are dependent on retinoid for normal differentiation, so most of the substances that need to increase body weight would be utilized for purpose of production of RBC to compensate for excessive lost and more energy will be wasted. Anaemia can also aggravate a disease condition and brings down the body weight. The present study also showed that there was no statistically significant difference in serum Vitamin E of SCA patient with normal weight and underweight (0.066 ± 0.044 and 0.073mg/ml respectively). It has also being observed that adequacy of diet decrease with age [16], increase high level of IL-6 found in sickle cell patient adversely act on the brain cell thereby suppressing appetite center and decrease food intake [23], [24] supporting the inability to get SCA patient with overweight and obesity in the present study rather most subject were underweight. Studies have also shown that children with SCA have elevated metabolic rates that can result in protein-energy deficits [32] and deficiencies in micronutrients, especially zinc [33]. Antioxidant vitamins A and E were also found to be low in SCA patients compared to the normal controls [28], [29]. High resting metabolic rate of sickle anaemia patients was reported compared to their counterpart's age and sex marched control subjects with a normal haemoglobin genotype [34].

Analysis of present results based on BMI showed that SCA subjects are either underweight or normal weight, while overweight and obese were not recorded when compared to control group. The probable reason for decrease in BMI may be attributed to many complications, including growth retardation (decreased height and weight compared to their peers), chronic hemolytic anemia, high resting metabolic rate, suppression of appetite in the brain by high level of IL-6 [24], while Zinc, folic acid, and vitamins A, C and E could be contributing factors [33], [35], [36], [37], [38], [39]. The present study also in agreement with the work of Chijioke 2009, who observed that the SCA, 92.5% were underweight and only 5.7% were overweight. It was also observed that lower BMI in SCA subjects is a reflection of the severity of the disease and the quality of care available to the SCA subjects.

5. Conclusion

In conclusion, the study showed that vitamin A and E levels were lower in SCA subjects compared to normal control groups, the BMI in SCA showed either underweight or normal weight while the control have in addition, showed overweight and obese.

Table 1. *BMI in relation to Mean Vitamin A level (mg/ml) and Mean Vitamin E level (mg/ml) in both SCA patients and normal subjects without SCA.*

BMI	Mean Vitamin A (mg/ml) ± SEM		Mean Vitamin E (mg/ml) ± SEM	
	Control (n=60)	SCA (n=60)	Control (n=60)	SCA (n=60)
Underweight (<18.5kg/m^2)	0.060 ± 0.002	0.050 ± 0.002	0.085 ± 0.003	0.073 ± 0.003*
Normal weight (18.5-24.9kg/m^2)	0.070 ± 0.005	0.049 ± 0.003*	0.103 ± 0.005	0.066 ± 0.004**
Overweight (25-29.9kg/m^2)	0.067 ± 0.001	-	0.082 ± 0.001	-
Obese (>29.9kg/m^2)	0.094 ± 0.001	-	0.143 ± 0.001	-

* Significance relative to control, *P < 0.05, Z-test.

References

[1] Wood, K. C. and Granger, D. N. (2007). Sickle cell disease; the role of reactive oxygen and nitrogen metabolites. Clin. Exp Pharmacol Physiol., 34 (9), pp. 926-32.

[2] Ingram, V. M. (1956). A specific chemical difference between the globin of normal human and sickle cell anaemia haemoglobin. Nature, 178, pp. 792-794.

[3] Misaki, W. (2008). Bone marrow transplantation (BMT) and gene replacement therapy (GRT) in sickle cell anaemia. niger. J. med, pp. 251-6.

[4] Jellife, D. B. and Humpteys J. (1952). The sickle cell Trait in Western Nigeria. Brit. Med. J., 1, pp. 405-6.

[5] Walters, J. H. and Lehmann H. (1956). Distribution of the S and C haemoglobin Variants in two Nigerian communities. Trans. Roy. Soc. Trop. Med. Hyg., 50, pp. 204-8.

[6] Fleming, A. F., Story, J., Molineaux, L., Iroko, F. A., and Attai D. E. (1979). Abnormal haemoglobin in Sudan Savanna of Nigeria. Trop. Med. Parasitol., 73, pp. 161-71.

[7] Kaine, W. N. and Udeozu, O. K. (1981). Incidence of Sickle cell trait and anaemia Ibo pre-school children. Nig. J. Paed., pp. 87-9.

[8] Khalil, M. I., Padonu, M. K., Omatara, B. A., and Ezimah, A. C. (1992). Evaluation of population genetics of haemoglobin (s) in a rural population genetics of North East Nigeria. Medicare J., 5, pp. 16-20.

[9] Ohene-Frempong, K., Steinberg, B. G., Forget, B. G., Higgs, D. R., and Nagel, R. L. (2001). Disorders of Haemoglobin: Genetics, Pathophysiology, and Clinical Management. Cambridge University Press; MH. Clinical Aspects of Sickle Cell Anemia in Adults and Children. In Steinberg, pp. 611-670.

[10] Desai, D. V. and Heren, D. (2004). Sickle cell disease history and origin the intern jour. Haem, 1 (2), pp. 1540-2649.

[11] Ashutosh, L. and Elliote P. V. (2007). Sickle cell disease in; Victor H., Daniel C., Edward, C. G., ed. Postgraduate haematology 5th ed.

[12] Olanrewaju, D. M. (2002). Sickle cell disease; lecture delivered at update course of WACP (paed).

[13] de Franceschi, L., Bachir, D., Galacteros, F. *et al*., (2000). Oral magnesium pidolate: effects of long-term administration in patients with sickle cell disease. Br J Haematol, 108 (2), pp. 284–9. [PubMed]

[14] Heyman, M. B., Katz, R., Hurst, D. *et al.,* (1985). Growth retardation in sickle-cell disease treated by nutritional support. The Lancet, 325 (8434), pp. 903–6. [PubMed]

[15] Tomer, A., Kasey, S., Connor, W. E. *et al.*, (2001). Reduction of pain episodes and prothrombotic activity in sickle cell disease by dietary n-3 fatty acids. Thromb Haemost., 85 (6), pp. 966–74. [PubMed]

[16] Al-saqladi, A. A., Cipolotti, R., Fijinvandraat, K. (2008). Growth and Nutritional status of children with homologous sickle cell disease. Ann. Tropical pediatric international child health, 28, pp. 165-189.

[17] Badaloo, A., Jackson, A. A., Jahoor, F. (1989). Whole body protein turnover and resting metabolic rate in homozygous sickle cell disease. Clin Sci, 77 (1), pp. 93–7. [PubMed]

[18] Barden, E. M., Zemel, B. S., Kawchak, D. A. *et al*., (2000). Total and resting energy expenditure in children with sickle cell disease. The J Pediatr, 136 (1), pp. 73–9. [PubMed]

[19] Khan, S., Steven, J. T., and Dinko, N. (2009). Zinc deficiency causing hyperammonemia and encephalopathy in a sickle cell patient. Chest {meeting abstract}, 136 (4), pp. 37S–7d.

[20] Natta, C. L. and Reynolds, R. D. (1984). Apparent vitamin B6 deficiency in sickle cell anemia. Am J Clin Nutr., 40, pp. 235–39. [PubMed]

[21] Soliman, A. T., El-Zalabany, M., Amer, M., *et al*., (1999). Growth and pubertal development in transfusion-dependent children and adolescents with thalassaemia major and sickle cell disease: A comparative study. J Trop Pediatr., 45 (1), pp. 23–30. [PubMed]

[22] Hyacinth, H. I., Gee, B. E., and Hibbert, J. M. (2010). The role of nutrition in sickle cell disease. Nutrition and Metabolic Insights, (3), pp. 57-67.

[23] Hibbert, J. M., Hsu, L. L., Bhathena, S. J., (2005). Pro-inflammatory cytokine and the hypermetabolism of children with sickle cell disease. Experimental biological medicine, 230 (1), pp. 68-74.

[24] Okpala, I. (2006). Leucocyte adhesion and the pathophysiology of sickle cell disease. Curr. Opin Hematology, (13) 1, pp. 40-4.

[25] Belcher, J. D., Bryant, C. J., and Ngugen, J. (2003). Transgenic sickle mice have vascular inflammation. Blood, 101 (11), pp. 3953-9.

[26] Hibbert. J. M., Creary, M. S., Gee, B. E., *et al*., (2006). Erythropoiesis and myocardial energy requirements contribute to hypermetabolism of childhood sickle cell anemia. J. pediatr Gastroenterol and Nutr, 43 (5), 680-7.

[27] Natta, C. L., Tatum, V. L., Chow, C. K. (1992). Antioxidant status and free-radical induced oxidative damage of sickle erythrocytes. Ann NY Acad Sci., 669, pp. 365–7. [PubMed]

[28] Tukur, M. A., Odeh, S. O., Ambe, J. P., Eyinkwola, O. and Salami, H. A., (2015). Vitamin A status of steady state sickle cell anaemia patients compared to normal control in Maiduguri north eastern Nigeria. British Journal of Medicine and Medical Research, 10 (5), pp. 1-6.

[29] Tukur, M. A., Odeh, S. O., Ambe, J. P., Eyinkwola, O., and Mojiminiyi, F. O., (2015). Vitamin E status of steady state sickle cell anaemia patients compared to normal controls. Internal J. Med. Med. Sci. Res., 3 (1), 006-012.

[30] Ozigbe C. J. and Nkanginiema K. E. (2003). Body Mass Index and sexual Maturation in adolescent patients with Sickle cell Anaemia. Nig. J. Paed., 30 (2), pp. 39-44.

[31] Chijioke A., (2009). The longevity and clinical pattern of adult sickle cell anaemia in Ilorin. Europ J of Scien Rear; vol 32 (4): 528-530.

[32] Singhal, A., Stephany, P., Louise, L., and Serjeant G. R. (2002). Energy intake and resting metabolic rate in preschool Jamaican children with homozygous sickle disease. Am J Clin Nutr, 75, pp. 1093-7.

[33] Pelligrini, B., Kerbauy, J., and Fisberg, M. (1995). Zinc, Copper and iron and their interrelations in the growth of sickle cell patients. Arch Latinoam Nutr., 45 (3), pp. 198-203.

[34] Singhal, A., Davies, P., Sahota, A., Thomas, P. W. and Graham S. (1993). Resting membrane rate in homozygous sickle cell disease. Am J. Clin. Nutr, 57, pp. 32-34.

[35] El-Hazmi, M. A. F. (1979). On the nature of sickle cell disease in the Arabian Peninsula. Hum Genet, 52, pp. 323-335.

[36] Powars, D. R., (1975). Natural History of sickle cell disease - the first ten years. Sem Hemat., 12, pp. 48-50.

[37] Leonard, M. B., Zemel, B. S., Kawchak, D. A., Ohene-Frempong, K., Stallings, V. A. (1998). Plasma zinc status, growth and maturation in children with sickle cell disease. J Pediatr, 132, pp. 467-71.

[38] Williams, R., George, E. O., Wang, W., (1997). Nutrition assessment in children with sickle cell disease. J Assoc Acad Minor Phys, pp. 844-8.

[39] Finan, A. C., Elmer, M. A., Sasanow, S. R., McKinney, S., Russell, M. O., Gill, F. M., (1988) Nutritional factors and growth in children with sickle cell disease. Am. J Dis Child, 142 (2). pp. 237-40.