

Antioxidant vitamins and glyatedhaemoglobin status in sickle cell anaemia

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Abstract: *Background:* Sickle cell anemia is a genetic disorder that is characterized by chronic anemia and oxidative stress. *AIM:* This study was investigated to evaluate the level of antioxidant vitamins C and E, and glyatedhaemoglobin in patients with homozygous sickle cell disorders (HbSS in steady state and HbSS in crisis) and normal healthy individuals (HbAA). *Material and Methods:* 100 sickle cell patients aged 5-30 years were included in this study while 100 normal healthy individuals served as the control. Also 30 sickle cell patients in crisis were involved. *Results:* This results obtained showed that the level of vitamins C and E were significantly depleted in sickle cell disease when compared with healthy individuals at $p < 0.05$. In the same vein, the level of glyatedhaemoglobin was decreased in sickle cell disease when compared with normal healthy control. Also the levels of vitamins and glyatedhaemoglobin were significantly decreased in sickle cell crises ($P < 0.05$). *Conclusion:* This shows that depleted antioxidant vitamins could be an important factor in sickle cell crisis. Hence, supplementation of sickle cell patients with vitamins can improve their health.

Keywords: Vitamins; glyatedhaemoglobin; sickle cell anemia.

Introduction

The physiological manifestation of sickle cell disorder increase geometrically with the sickle cell gene quantity [1]. Sickle cell anemia is the prominent cause of morbidity and mortality in Igbo land in early 19th century. Our fore fathers called it "Ogbanje". It is a genetic disorder in which valine is substituted for glutamic acid in the sixth position of the β -chain forming sickle cell haemoglobin [2].

Sickle cell disease patients have painful episode which last from hours to days. The crises among sickle cell anemia can result to pain in the bones and chest [3]. It is quite pertinent to note that individuals with sickle cell disease are highly prone to oxidative damage as a results of chronic redox imbalance in erythrocytes. Evidence exists to indicate that sickle cell red cells generates twice as much as hydrogen peroxide superoxide,

and hydroxyl radicals [4-6]. Studies have shown that a variety of pathophysiological conditions for instance dehydration, hypoxia, infection and reduced antioxidant vitamins are observed as a result of excessive production of reactive oxygen species [7-9].

Vitamins are Vitamins are organic substances present in minute quantities in natural foods, that are essential for health and which results in characteristic abnormalities when absent from diet or when present in insufficient amounts, due to the malfunctioning of a particular biochemical and metabolic process[10]. They serve the need for growth, differentiation and maintenance of normal cellular function. Vitamins are classified into fat-soluble vitamins which includes vitamin A (Retinol), vitamin D (Calciferol), vitamin E (a-tocopherol), vitamin K (2 methyl 1, 4-naphthoquinone) and water soluble vitamins

which includes vitamin B complex and vitamin C (Ascorbic acid). However, there is little or no study on the applicable value of vitamins as well as glycatedhaemoglobin in sickle cell anemia.

Material and Methods

Subjects: 230 subjects age 5 to 30 years were selected for the study (100 HbSS, 100 HbAA and 50 HbSS in crisis). Patients with any metabolic disease were excluded. Their consent was obtained as well as ethical approval from the ethical committee of the hospital (General Hospital Owerri and ABIC Medical Diagnostic Lab.).

Blood Collection: In all subjects 4ml of fasting venous blood was collected into plain and EDTA bottle. The serum was separated by centrifuging the whole blood in westerfuge (model 684) centrifuge at 5,000g for 5 minutes.

Biochemical Assay: Plasma vitamin C was assayed by the 2, 4-dinitrophenyl hydrazine method described by Tietz [11]. The vitamin E was done by the method of Tietz [12] in which vitamin E caused the reduction of ferric to ferrous ion which then forms a red complex with α -dipyridyl. Glycatedhaemoglobin was estimated by the method of Trivelliet al. [13], in which a haemlysed preparation of the whole blood is mixed continuously with a weak binding cation-exchange resin. Glycatedhaemoglobin binds to the resin.

Statistical Analysis: The results were expressed as mean \pm standard deviation and student t-test was used to calculate the level of significance at $p < 0.05$.

Results

Group	Vitamin C (mg/dl)	Vitamin E (mg/dl)	Glycated H. (%)
HbAA	0.97 \pm 0.06	1.45 \pm 0.10	6.67 \pm 0.40
HbSS-Steady state	0.81 \pm 0.09*	1.07 \pm 0.06*	4.82 \pm 0.34*
HbSS-crisis	0.74 \pm 0.07*	1.07 \pm 0.07*	4.31 \pm 0.23*

*Significantly different from control at $P < 0.05$

Discussion

Sickle cell anemia is a disorder caused by inheritance of a pair of abnormal haemoglobin genes. It is associated with various clinical symptoms such as jaundice, skeletal changes, intravascular sickling and thrombosis leading to painful crisis [13-16]. In this study, it was observed that vitamin C and E were depleted in HbSS steady state when compared with the control HbAA.

Also, the level of vitamin C and E were more depleted in HbSS –crisis when compared with the control. This is in line with the work of Debes et al [10]. Vitamins play an important role in the antioxidant defence system. Vitamin C is a water soluble vitamin which possibly may have protective role against oxidative membrane damage [17]. However, the depletion of vitamins

C and E could worsen the state of sickling as observed in the individual with sickle cell crisis. This is in line with the work of Behera et al [5]. In fact, the sickling may be more susceptible to oxidative stress [18-20] hence, it requires more vitamin C and E to neutralize reactive oxygen species. The depletion in the level of antioxidant vitamins is observed more in sickle cell crisis. Hence, they are subjected to higher level of oxidative stress. Also, the reduced level of antioxidant vitamins can trigger dehydration which leads to sickling leading to more sickle cell crises as observed in the study.

In the same vein, the level of glycatedhaemoglobin was significantly decreased in HbSS steady state when compared with the control. This is in line with the work of Atabani et al [21] in which

glycosylated haemoglobin was decreased in Sudanese sickle cell anaemia. However, it was more decreased in HbSS-crisis. This shows that they may unlikely be prone to diabetes.

Therefore, people with sickle cell disease should be supplemented with food, fruits or drug containing antioxidant to improve their health status.

References

1. Osuagwu CG and Mbeyi CU. Altered plasma hexose sugar metabolism in sickle cell anemia. *African Journal of Biochemistry* 2007; 1(3):037-040.
2. Nnodim JK. In vitro effect of Allopurinol on sickling rate and uric acid level in sickle cell erythrocyte. *Asian journal of Medical Science* 2003; 4(2):30-32.
3. Steinberg MH. Pathophysiologically based drug treatment of sickle cell disease. *Trends Pharmacol Sci.* 2006; 27:204-210.
4. Jyoti T, Suresh C, Madhur G and Nitin PJ.(2004).Pro-oxidant and antioxidant status in patients of sickle cell anaemia. *Indian J Clin Biochem*, 2004; 192:168-172.
5. Behera S, Dixit S, Bulhyya G and Kar SK. Vitamin A status and hematological values in sickle cell disorder cases. *India J. med. SC.* 2012; 66(8):169-173.
6. Klings ES and Farber HW. Role of free radicals in the Pathogenesis of acute Chest syndrome in sickle disease. *Respir Res.* 2001; 2:280-285.
7. Mauro S. The role of antioxidants in disease prevention. *Medicine* 2006; 34(12): 533-535.
8. Uwakwe AA, Onwuegbuke C and Nwinuka NM. Effect of caffeine on the Polymerization of Hbs and sickling rate, osmotic Fragility of Hbs and sickling rate of Hbs erythrocytes. *Nig J of Biochem Mol Biol.* 2002;11:27-32
9. Abiodun ME, Uadia PO and Kuliya GA. Antioxidant enzymes and acute phase proteins correlate with marker of lipid peroxidation in adult Nigerian sickle cell disease patients. *Iranian Journal of basic Medical Sciences* 2010; 13(4):177-182.
10. Debes D, Kalyan G and Neelam G. Antioxidant vitamin levels in sickle cell disorders. *The National Journal of India* 2007; 20(1):11-13.
11. Tietz W. Ascorbic acid. In: fundamental of clinical chemistry. *W.B. Saunder Company Britain.*1976; 422-429.
12. Tietz W. Clinical guide to laboratory tests. *W.B. Saunders Company Philadelphia.* 1976; 543-551
13. Trivelli LA, Ranney PH and Lai HT. Glycatedhaemoglobin estimation. *New Eng. J. med.* 1971; 284:353.
14. Prakash H, Adinath S, Aarti K, Rahul G and Maya V. Antioxidnt status and lipid peroxidation in sickle cell anaemia, *Biomedical Research*, 2010; 21(4): 46-464.
15. Mmeremiukwu M. Sickle cell disease. *Clin. Evid.* 2005; 14:15-28.
16. Nitin J. A review of clinical profile in sickle cell traits. *Oman Medical Journal.* 2010; 25(1):3-8.
17. Nwanjo HU, Ojiako OA. Effect of Vitamin E and C on exercise induced oxidative stress. *Global J. Pure Applied Sci*, 2005; 12:199-202.
18. Stuart MJ and Nagel RL. Sickle cell disease. *Lancet.* 2004; 364:1343-1360.
19. Switzer JA, Hess DC, Nichols FT and Adams RJ. Pathophysiology and treatment of stroke in sickle cell disease. *Lancet Neurol* 2006; 6:501-512.
20. Ugonabo MC, Onwuamaeze IC, Okafor EN and Ezeoke ACJ. Plasma cholesterol of sickle cell anaemia patients in Enugu Nigeria. *Bio Research*, 2007; 5(2):241-243.
21. Atabani GS, Hassan DA, Abdul R and Saeed BO. Glycosylated haemoglobin levels in Sudanese sickle cell anaemia. *Acta Haemtologica* 1989; 81(3):140-142.

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