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Uric acid concentration in sickle cell disease patients in owerri metropolis

*** Obeagu,Emmanuel Ifeanyi[1], Okoroiwu,I.L.[2], Daniel-Igwe,Gloria[3] , Queen Braxton N.[4] ,Ogwuegbu,Victoria Onyemakeya[5]**

[1.] Diagnostic Laboratory Unit,Department of University Health Services,Michael Okpara University of Agriculture,Umudike,Abia State,Nigeria.
[2].Departemnet of Medical Laboratory Science,Imo State University,Owerri,Nigeria.
[3.]Department of Veterinary Pathology,College of Veterinary Medicine, Michael Okpara University of Agriculture,Umudike,Abia State,Nigeria.
[4.] Department of Accident and Emergency Lagos State University Teaching Hospital,Nigeria.
[5].Department of Public Health and Private Medicine College of Veterinary Medicine, Michael Okpara University of Agriculture,Umudike,Abia State,Nigeria.
*Corresponding Author : *emmanuelobeagu@yahoo.com,obeagu.emmanuel@mouau.edu.ng*

Keywords

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arthritis
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Abstract

The serum uric acid levels of some patients suffering from sickle cell disease, attending clinic at Federal Medical Centre Owerri were determined. A total of 50 subjects suffering from sickle cell disease and 50 apparently healthy subjects were analysed using standard laboratory procedure. The test subjects were classified as adults and children. The values obtained were statistically analysed and compared with normal published values using mean and standard deviation. The result obtained revealed an increase in the uric acid value of test subjects which is equally statistically significant (6.8±1.5) P >0.05. Also statistically analysed were values of uric acid between adult sicklers and children. Significant statistical difference between the two was observed, children (4.6 ± 1.4), adult (6.3 ± 1.2). the results obtained from this work has revealed that apart from gout, arthritis and some kind of leukaemia where high uric acid level has been incriminated, sickle cell disease equally trigger increase in high uric acid value.

Introduction

Sickle cell disease is a collective name for a group of conditions causing clinical symptoms which are characterized by the formation of Sickled red cell (Lewise et al., 2004). Sickle cell disease can also be described as a group of haemoglobin disorders in which abnormal beta-globin gene is inherited with the homozygous Sickle cell anemia (HbSS) being the most common, while the heterozygous condition of HbSC and HbS that may also cause Sickle disease (Hoffbrand et al., 2004).The disease can therefore be classified into; Sickle cell anaemia, sickle HbC disease (HbSC), sickle beta-plus thalasenia and sickle beta-zero thalasemia. Sickle cell disease can be described further as an important hereditary haemoglobmoputhy; a type of disease characterized by production of defective haemoglobin (Kumar et al., 2004).

Haemoglobin (Hb) is a chromo protein consisting of a globin molecule attached to four red coloured haem molecules. The globin molecule consist of two alpha and two beta polypeptide chains.

Haem is a metal complex containing an iron atom in the centre of a prohyrin structure (Ochei and Kolhatkar, 2000). There are proximately 280 million Hb molecules in each red blood cell. The beta-globin is a major component of adult haemoglobin (HbA). The gene for beta-globin is located on chromosome 11 and there are 475 allelic variants. One of

these variants is responsible for the leritance of sickle cell disease. Sickle cell disease is regarded as first genetic disorder whose molecular basis is known (Balgir, 2007).

Uric acid (UA) is a white, tasteless, orduorless, crystalline product of protein (purine) metabolism found in blood and urine. It is a nitrogenous compound present as the principal nitrogenous component of the excrement of reptiles and birds. It is found in minutes amount in the mammalian urine. Uric acid has the molecular formular C5 H4 N4 O3/2, 6, 8- trihydroxypurine. It is a weak acid with pka of 5.75 and 10.3 (Wortmann, 1994). Purines such as adenosine and guanine result from the breakdown of nucleic acids. They are either ingested or come from the destruction of tissue cells, and are converted mainly in the liver into uric acid (Ochei and Kolhatkar, 2000). Daily synthesis rate of uric acid is approximately 400mg, dietary intake contributes 300mg urates,the ionized forms of uric acid predominate in plasma, extracellular fluid and synovial fluid (Wortmann, 1994). Normal plasma uric acid level is 1.5-7.0mg/dl (Nwanjo et al., 2005), but may vary according to different laboratories and locations. Uric acid is quite insoluble in water, and it also readily forms an insoluble sodium salt known as sodium urate. Gout is a joint inflammation of sudden onset characterized by elevated levels of uric acid in body fluids (Matta et al.,1996).

JUSTIFICATION

The study was carried out because uric acid associated with arthritis is known to cause severe pain and most often, sicklers when in crises complain of pain, therefore, this project work is carried out to find out if the increase in pain in arthritic patients is also seen in sticklers.

AIM

To assess serum uric acid concentration in patients with sickle cell disease.

Materials and Methods

Study Area

The study was conducted in Owerri, the capital of lmo State Nigeria.

Ethics:

Oral consents were sought from the subjects prior to sample collection.

Subject Recruitment

A total of 100 subjects comprising 50 sickle cell patients and 50 apparently subjects were recruited for this research work

Sample Collection

Fasting blood samples (3ml) were collected by venipuncture from the anticubital vein of each patient with a sterilized 5ml syringe and transferred into a plain container, and the test were performed in Federal Medical Center Laboratory Owerri.

Uric Acid Estimation by Spectrophotometrically (Ochei and Kolhatkar, 2000) Method(caraway)

Principle

Uric acid is converted to allantoin and hydrogen peroxide by uricase, which under the catalytic influence of peroxidase, oxidizes 3,5-dichloro-2-hydoxybcnzcncsulfonic acid and A-amino phenazone to form a red violet quinoneimine compound (Ochei and Kolhatkar, 2000).

Three test tubes were prepared and labeled 'Test, 'standard' and blank' and reagents were delivered into each of the test tube with aid of an automatic pipette as follows.

	Test	Standard	Blank
Phosphotungstic acid reagent	2ml	2ml	2ml
Sample	0.2ml		
Working uric acid std 5mg/di	0.2ml	-	
Distilled water	-	0.2ml	-

Each tube was thoroughly mixed and incubated at room temperature for 15 minutes.

All the tubes were centrifuged at 3000rpm for 10 minutes.

The supernatant was decanted directly into three separate corresponding labeled tubes. 1ml of sodium carbonated solution was added to each tube. Incubated at room temperature for 15 minutes

The absorbance was read at 650-700nm, setting the zero with blank.

Results

The test subjects were made up of 25 males and 25 females with age bracket of 1-35 and 4-47 years respectively and mean age of 27.2±10.1 with mean value of 6.8± 1.5

Table 4.1 below shows the mean and standard deviation of serum uric level from subjects with sickle cell disease.

	±SD
Subjects mean age	
mean (mg/dl)	
	6.8
	1.5
	(N=50) 27.2±10.1

The serum uric acid level showed a significant increase in sickle cell patients when compared with normal published uric acid level from normal individual (2 -7.2mg/dl normal individual).

Table 4.2 below shows the mean and standard deviation of serum acid level from adult sickle cell patients.

Mean	Standard
6.3	1.2

The serum uric acid level showed a significant increase when compared to normal published uric acid level from normal adults (.normal adult = 2.6 - 7.2mg/dl).

Table 4.3 below, shows the mean and standard deviation of serum uric acid level from children suffering from sickle cell disease.

Mean	Standard deviation
4.6	1.4

The serum uric acid level showed a significant increase when compared with normal published uric acid level from normal children (normal children = 2-5,5 mg/dl).

Table 4.4 shows the comparism of results from children and adults sickle cell patients with the P value.

Children	Adults	P-level
4.6+1.4	6.3+1.2	P>0.05

The serum uric acid level from the adult sickle cell patients showed a significant increase when compared with that of the children. This could be because of the tubular and structural abnormalities as a result of vaso-occlusion, ischemia and infarction of the medullary circulation of the kidneys. It may also be due to age, body physiology, biochemistry and body response, working conditions, disease management, and prophylaxis, hygiene and environment.

Discussion

Uric acid is an organic acid that is the end product of nucleic acid metabolism. It is insoluble in water and can form crystals that lodge in the joints and skin. It is more toxic to tissue than xanthine or hypoxanthine. In this study uric acid of 30 subjects was determined with the aim of accessing the uric acid concentration in sickle cell patients. The results showed increase in serum uric acid as seen in table 4.1 (6.8 ± 1.5), this could be as a result of renal structural and functional abnormalities associated with sickle cell disease, which has also altered renal physiology of the sickle cell disease patients and predilection to nephropathy.This agrees with the work of Yardim -Akaydin and Sepici (2004), that increased allantoin level, the oxidation product of uric acid is suggestive of the possible involvement of free radicals in sickle cell patients. A positive relationship between uric acid and allantoin in plasma was also carried out by Tsahar et al. (2006), where they suggested that there is an increase in an in-vivo marker for oxidative stress in humans. The result of uric acid level in adult suffering sickle cell disease was also increased as seen in table 4.2 (6.3±1.2). There was also an increase level of uric acid in children suffering from sickle cell disease as seen in table 4.3 (4,6±1.4). There was a significant increase of uric acid level in adult sicklers when compared with that of children sicklers as seen in table 4.4 (adult = 6.3±1.2), (children = 4.6±1.4). This could be because of the tubular and structural abnormalities as a result of vaso-occlusion, ischemia and infarction of the medullary circulation of the kidneys. It could also be due to age, working condition, disease management, hygiene, environment, body physiology, biochemistry and body response. It has been established that increased concentration of uric acid cause crystals to form in the joints, which lead to the joint inflammation and pain characteristics of gout. This may not be for fetched from the joint inflammation seen in sickle cell patients. This further finds support from the observation reported by Becker (1993), that particularly elevated levels of uric acid may be a marker of oxidative stress.

Some researchers however propose that hyperuricaemia-induced oxidative stress is a cause of metabolic syndrome. A persistently high titre of uric acid in sickle cell patients may imply that there is an impaired excretion of uric acid or an increase production of uric acid. In addition, the tissue destruction and muscle wasting in sickle cell patients may be concomitant with the elevated uric acid levels as seen on this study. Currently high dose of Asprin can be used to Idwer the concentration of uric acid in sickle cell patients.

Conclusion

Findings from this work has really revealed that apart from some kind of arthritis and leukaemia where high uric acid level has been incriminated, increase in uric acid level is equally observed in sicklers which could be a contributing factor to joint pain which usually go with crises in sicklers.The management of sicklers should include periodic evaluation of their uric acid level.

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