

Iron Status of Pregnant Women Attending an Antenatal Clinic in Benin City, Nigeria.

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ABSTRACT

The federal ministry of health in Nigeria gives great attention to pregnant women in other to minimize morbidity and mortality, but some perennial problems including poor nutrition, position in polygamous homes, poor child spacing, non compliance to supplementation of hematinic and women entering pregnancy with iron depleted status. This study was aimed at determining the iron status of pregnant women in Benin City.240 pregnant women (80 in each trimester), aged between 18 and 42years and 80 non pregnant women (age and sex matched control) participated in the study. hemoglobin concentration was estimated using the cyanmethemoglobin method .Serum ferritin was determined by enzyme linked immuno sorbent assay technique. Serum iron was determined by spectrophometric method. It was observed that, there was a significant decrease (P < 0.05) in hemoglobin concentration, serum iron and serum ferritin at stage 1 when compared with state 2, 3 and 4. Pregnant women are prone to iron deficiency anaemia due to the increased physiological demand of the growing fetus and inadequate dietary iron.

Keywords: Iron; Anaemia; Nutrition; Pregnancy; Serum ferritin



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INTRODUCTION

Iron deficiency is the most common cause of anaemia worldwide ⁽¹⁾. What is referred to as physiologic anaemia of pregnancy is a dilution process secondary to an increase in plasma volume, during pregnancy the demand for micronutrients especially iron and folate are increased ⁽²⁾. The maternal body stores and dietary intake may be insufficient for adequate erythropoiesis (3-4). Severe anaemia causes intrauterine growth retardation, premature delivery, neonatal and perinatal death ⁽⁵⁻⁷⁾. A decrease in iron stores in pregnancy is accompanied by a significant rise in the reticulocyte production index, which are consistent with an increase erythropoietic activity⁽⁸⁾.

The federal ministry of health in Nigeria gives great attention to pregnant women in other to minimize morbidity and mortality. But some perennial problems including poor Nutrition, position in polygamous homes, poor child spacing, non compliance to supplementation of hematinic and women entering pregnancy with iron depleted status⁽¹⁾.

A typical western diet provides an average of 6mg of heme and non heme iron per 4120kj. The bioavailability of iron is both a function of its chemical from and the presence of food items that promote or inhibit its absorption. Ascorbic acid and meat are powerful enhancers of iron absorption whereas cellulose, pectin, phytic acid, bran, hemicelluloses which are found in wheat and soy products inhibits iron absorption ^{(1,8).} This study was aimed at determining the iron status of pregnant women in Benin City.

MATERIALS AND METHODS

Subjects aged between 18 and 42 years participated in the study. Patient consent form was obtained from 240 pregnant women on antenatal visit and 80 aged matched healthy individual on routine checkup. The pregnant women were divided into three groups by trimester. Ethical approval was obtained from Lahor medical and research laboratory, Benin City, Edo state.

Blood sample collection

6ml of venous bloods were taken from the antecubital vein by venapuncture. It was shared equally into ethylene diamine tetra acetic acid container and an anticoagulant free test tube, allow to clot and subsequently centrifuge at 750xg from 15minutes to obtain serum. The serum was immediately aliquoted into eppendorf tube and stored at -20^oC.

Blood Sample Processing

Sample Preparation and test performance for hemoglobin concentrations were done using the cyanmethemoglobin method described by [9]. 20 µl of blood was diluted in a 5 ml buffered solution of potassium Cyanide to yield the stable hemoglobin derivative cyanmethemoglobin. The potassium ferricyanide converts the Hemoglobin to methemoglobin by the action of potassium cyanide. This must be allowed to stand for at least three minutes, to allow for complete conversion of hemoglobin to cyanmethemoglobin, before the absorbance is measured against a reagents blank at a wavelength of 540 mm using a spectrophotometer.

Assessment of iron status:

Serum ferritin

A microtitre plates' containing 96 wells pretreated with antiferritin antibody was incubated with 0.05ml fresh unhaemolysed serum and left overnight at 4° c. The wells were washed three times with wash solution. Then 0.2ml of a substrate (chromogen) was added to the wells incubated for 30min. 0.05ml of hydrochloric acid was added to each well to stop the reaction. The absorbance was read using a microplate reader at 450nm. The colour change is proportional to the amount of ferritin in serum (⁹).

Serum iron

Iron in serum was dissociate from its complex fe^{3+} , 2.5ml of an acidic buffer containing hydroxylamine was added to four properly labeled test tubes blank, standard, test and control containing 0.5ml of fresh unhaemolysed serum. This addition caused a reduction the Fe³⁺ to Fe²⁺ 0.05ml of a chromogenic agent was then added which formed a highly coloured Fe²⁺ complex that was measured using a spectrophotometer at 560nm. ⁽⁹⁾

RESULTS

Table 1: The mean \pm standard deviation of hemoglobin concentration, serum iron and serum ferritin at stage 1 to 4. It was observed that, there was a significant decrease (P< 0.05) in hemoglobin concentration, serum iron and serum ferritin at stage 1 when compared with stage 2, 3 and 4.



Table 1: The mean ±standard deviation of hemoglobin concentration, serum iron and serum ferritin of pregnant women at
different trimesters and the control group.

	1	2	3	4
Parameters	N = 80	N = 80	N = 80	N= 80
Hemoglobin Concentration (Hb (g/dl)	13 ± 0.01	10.4 ±0.01 ^A	9 ±0.02 ⁸	8 ± 0.03 ^C
Serum iron (µg/l)	140 ± 10	100 ± 5^{A}	85 ±0.7 ⁸	$70 \pm 08^{\circ}$
Serum ferritin (µg/l)	18 ± 0.01	12 ± 0.02^{B}	8 ± 0.01 ^B	6 ± 0.02^{C}

Keys

1	=	Control group
2	=	1st trimester
3	=	2nd trimester
4	= []]	3rd trimester
Ν	=	Number of sample size
А		Significant (P < 0.05) comparison between stage 1 and 2
в	=	Significant (P < 0.05) comparison between stage 1 and 3
С	=	Significant (P < 0.05) comparison between stage 1 and 4

DISCUSSION

It was observed that, there was a significant decrease (P < 0.05) in Hemoglobin concentration, serum iron and serum ferritin at stage 1 when compared with state 2, 3 and 4. These could be attributed to a disturbance in metabolic process associated with iron deficiency anaemia occasioned by an increased physiological demand of the growing baby for iron or dietary intake or absorption of iron may be insufficient for adequate erythropoiesis. This is in line with these findings. Iron deficiency anaemia is the most common hematological problem in pregnancy (1). During pregnancy, the demand for micro-nutrients (iron) is increased and material body stores and dietary intake may be insufficient for adequate erythropoiesis (2,3,4). Severe anaemia increases prenatal morbidity and mortality causing intrauterine growth retardation, premature delivery, neonatal and prenatal death (5-8). The bioavailability of iron is both a function of its chemical from and the presence of food items that promote or inhibit its absorption. Ascorbic acid and meat are powerful enhancers or iron absorption whereas cellulose, pectin, phytic acid, bran, hemicelluloses which are found in wheat and soy products inhibits iron absorption (1,8).

Iron deficiency is associated with altered metabolic process including mitochondrial electron transport, neutronsmitter synthesis, protein synthesis and organogenesis. The overt physical manifestations of chronic iron deficiency are glossitis, angular stomatitis, koilonychia (spoon nails), blue sclera, esophageal webbing and anaemia. Behavioral disturbances such as pica (abnormal consumption of dirt (geophagia) and ice (pagophagia) are often present in persons with iron deficiency (10-13). A decrease in myoglobin and other iron containing proteins in the skeletal muscle of persons with iron deficiency anaemia contribute significantly to the decline in muscle contraction capacity. (14-15). Hemoglobin iron, when lacking can profoundly alter physical work performance via a decrease in oxygen transport. (15, 16, 17).

CONCLUSION

Pregnant women are prone to iron deficiency anaemia due to the increased physiological demand of the growing fetus and inadequate dietary iron intake. Enlightenment campaign on iron bioavailability should be organized and women of child bearing age should check their iron status before getting pregnant.

REFERENCES

- 1. Milmann N, Byg KE, Agger AO. (2006). Hemoglobin and erythrocyte Indices during normal pregnancy and post-partum in 206. Women with an without iron supplementation. Acta obstetrician et Gynecologica Scandinavica. 79:89-98
- Milman N, Bergholt T, Byg KE. (2007). Reference intervals for hematological variables during normal pregnancy and post-partum in 434 healthy Danish women. European journal of hematology. 79: 39 – 46.
- 3. Milman N. (2008). Prepartum anaemia: Prevention and treatment. Anneal of hematology. 87: 949 -959.



- Wheeler S. (2008). Assessment and Interpretation of micronutrient status during pregnancy. Proceeding of the nutrition society 67:437 – 450.
- 5. Hoffbrand AV, Lewis SM, Tuddenham EG. (1999). Postgraduate Haematology. Butterworth Heinemann. Oxford. Pp 14-127.
- 6. Allen LH. (1997). Pregnancy and iron deficiency unresolved issues. Nutritional Review. 55:91-101.
- 7. Scholl TO.(2005). Iron status during pregnancy: setting the stage for mother and infant. America journal of Nutrition. 81:12185-12225.
- 8. Howell MR, Jones SE, Nepier JAF, Saunders K. (1986). Erythropoiesis in Pregnancy. British Journal of haematology 64: 595 599.
- 9. Dacis J and Lewis D. (2006). Practical Hematology, 8th Edition, Churchill Livingstone, London, Pp. 27-30.
- 10. Tietz NW. (1984) Fundamentals of Clinical chemistry, Philadelphia, W.B Saunders, Pp. 923-929.
- 11. Weaver CM, Rajaram S.(1992). Exercise and iron status. Journal of Nutrition. 122:782-787.
- 12. Cook JD. (1994). The effect of endurance training on iron metabolism. Seminar in Hematology .31:146–54.
- 13. Dallman PR.(1986). Biochemical basis for the manifestations of iron deficiency. Annu Rev Nutr .6:13–40.
- 14. Tobin B, Beard JL. Iron and exercise. In: Wolinsky I, Driskell JA, eds. CRC handbook of sports nutrition: vitamins and trace minerals. Boca Raton, FL: CRC Press, 1996:137–156.
- 15. Hallberg L, Rossander T, Hulten L, Brune M, Gleerup A. (1993). Inhibition of haem-iron absorption in man by calcium. British Journal of Nutrition .69:533–540
- 16. Dallman PR. (1982). Manifestations of iron deficiency. Seminar Hematology .19:19-30.
- 17. Finch CA, Huebers MD. (1982). Perspectives in iron metabolism. New England Journal of Medicine .25:1520–1525.
- 18. Viteri FE. (1995). Iron deficiency in children: new possibilities for its control. International Child Health .6:49-61.

