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M.T. Agyei-Frempong, BSc, MSc, PhD, G. Asare, BSc, MSc, W.K.B.A. Owiredo, BSc, MSc, PhD and F.A. Yeboah, BSc, MSc, PhD, Department of Molecular Medicine, School of Medical Sciences, University of Science and Technology, Kumasi, Ghana

Request for reprints to: Dr. F.A. Yeboah, Department of Molecular Medicine, School of Medical Sciences, University of Science and Technology, Kumasi, Ghana.

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M.T. AGYEI-FREMPONG, G. ASARE, W.K.B.A. OWIREDU and F A. YEBOAH

ABSTRACT

**Objectives:** To compare the prevalence of iron deficiency among Ghanaian children in different residential settings and to see whether 200mg ferrous fumarate B.P. could correct iron deficiency anaemia in observed cases of iron deficiency.

**Design:** Prospective case-finding study using an iron-deficiency society questionnaire, laboratory data and general practice records. Crude prevalence was calculated using the hospital's mid-year estimates.

**Setting:** Nkoranza in the Brong Ahafo Region of Ghana, Komfo Anokye Teaching Hospital, Kumasi, Ghana

**Subjects:** Rural-dwelling children entering as out-patients, urban-dwelling children entering as controls and newly diagnosed iron-deficient children entering as in-patients.

**Main outcome measures:** Crude prevalence rates (per quinquennia) for three groups of children. Corrected deficiencies expressed as percentage after management. Age, haemoglobin, iron status, residential status, symptoms at entry and after therapy.

**Results:** Following a 30-day administration of ferrous fumarate, the mean serum iron for the rural children increased significantly by 3.3  $\mu\text{mol/l}$  representing an improved iron status of 20.0% ( $P < 0.0001$ ). Iron deficiency anaemia defined by serum  $\text{Hb} < 12.0\text{g/dl}$  and  $\text{Fe} < 12.5\text{mmol/l}$  decreased by 10% in the rural subjects. Comparatively, iron deficiency among the newly diagnosed anaemia group, fell by 17.6% whilst their ambulant urban counterparts employed as the control group had an iron deficiency anaemia of 0.0%.

**Conclusion:** The study clearly indicates that the prevalence of iron deficiency anaemia among children in rural Ghana is about ten times that of the urban-dwelling children and that iron-deficiency anaemia accounts for a greater percentage of all anaemic cases among children in our hospitals. It was also shown that taking appropriate iron supplements like 200 mg of ferrous fumarate for thirty days can substantially improve the iron status of iron-deficient children.

INTRODUCTION

Iron deficiency and its precise functional liabilities continue to interest researchers. The socioeconomic consequences of iron deficiency is of special importance in the light of its high global prevalence. Approximately two thirds of the world's population is believed to be suffering from iron deficiency anaemia(1). Infants fed on relatively low iron milk formulae and breast fed infants, fall short of the recommended requirement of iron intake(2). Studies have indicated that there is a direct relationship between iron deficiency, mental performance and irritable behaviour in children, resulting in lower values for the Barley Scale of Infant Development( BSID) and the Mental Development Index (MDI)(3). This has been substantiated by a study carried out in Indonesia where it was realised that individual children with iron deficiency anaemia tend to have an impaired scholastic performance(4). It has also been shown that serum iron levels of patients with sensorineural deafness are significantly lower than those of normal-hearing individuals thus emphasising the possible physiochemical

role of iron in hearing(5). Ironically, however, in most of these iron deficiency cases, the haemoglobin levels appear normal(6). These and other findings compelled us to investigate the iron status of children in a typical rural dwelling in sub-Saharan Ghana and to find out the underlying causes, if any, of iron deficiency anaemia.

MATERIALS AND METHODS

A total of 218 children were entered into the study. This included 57 newly diagnosed anaemic children at a local Teaching Hospital, 71 children from Koforidua, a typical rural setting in the Nkoranza District of Ghana and 90 primary school going children from the university primary school. All the subjects recruited for this study were by the consent of their parents and blood samples were collected according to the Helsinki Declaration. All the 218 subjects were investigated for their haemoglobin levels, serum iron concentration, total iron binding capacity (TIBC), percentage saturation (PSAT) and serum ferritin at the entry of the study. The rural and the newly diagnosed anaemic group was then administered with 200mg of ferrous fumarate (B.P.=65mg of iron/tablet) for 30 days and their iron status further investigated at the end of the treatment. All samples for the serum iron estimations were taken before 10.00am.

The serum iron levels were estimated by the ascorbic acid reduction test using Ferrozine as the chromogen at a pH of 4.5 and incubation temperature of 40°C. Readings were taken at 540nm in the visible region. Total iron binding capacity (TIBC) was estimated from a 0.5ml of serum saturated in 1.0ml of FeCl<sub>3</sub> in the presence of MgCO<sub>3</sub>. The serum transferrin determination was by the radial immunodiffusion technique based on the formation of immunoprecipitin rings after incubating 5 µl of serum at 20°C for 48hrs. Serum ferritin was estimated at 25°C by an enzyme immuno assay technique using a monoclonal anti-ferritin antibody immobilised to a polystyrene tube. The sickling status of all the subjects was performed by the standard sodium metabisulphite procedure.

RESULTS

The overall mean results of the various iron deficiency indices in the various groupings in this study is represented in Table 1. In the rural group, the mean serum iron obtained was 13.3 ± 4.3 µmol/l before the administration of the iron supplement. This value increased to 16.6 ± 6.0 µmol/l representing 20.0% (p<0.0001) improvement. In the newly diagnosed anaemic group the mean serum iron level before the administration of the iron supplements was 14.9 ± 5.6 µmol/l. This value improved remarkably to 17.0 ± 4.9 µmol/l after 30 days of iron supplement therapy. The urban control group however maintained a mean appreciable serum iron level of 22.8 ± 5.9 µmol/l. In a similar development the mean TIBC concentrations of the various groups were estimated. The rural group had a mean value of 44.3 ± 2.2 µmol/l before iron supplement administration and 56.3 ± 2.3 µmol/l after thirty days on iron supplements.

Table 1

Iron deficiency indices in various study groups

	Rural children		Urban children		Anaemic group	
	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment
Mean Hb (g/dl)	12.5 ± 1.9	13.5 ± 2.4	13.8 ± 0.8	-	5.7 ± 1.9	8.7 ± 1.9
Mean iron (umol/L)	13.3 ± 4.3	16.6 ± 6.0	22.8 ± 5.9	-	14.9 ± 5.6	17.0 ± 4.9
Mean TIBC (umol/L)	44.3 ± 2.2	56.3 ± 2.3	57.2 ± 1.2	-	59.1 ± 2.9	55.5 ± 1.4
Mean PSAT (%)	34.5	35	41.6	-	29.9	35.7

Figure 1

Mean distribution of haemoglobin (Hb) levels in the various children groups before and after treatment with iron supplements

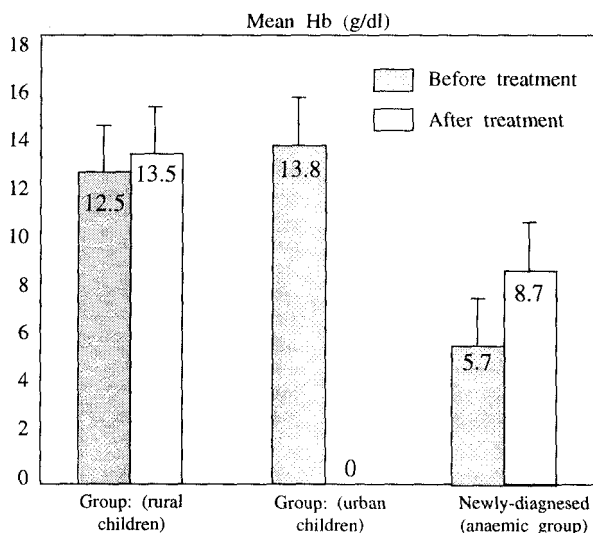


Figure 2

Mean distribution of the total serum iron in the various children groups before and after treatment with iron supplements

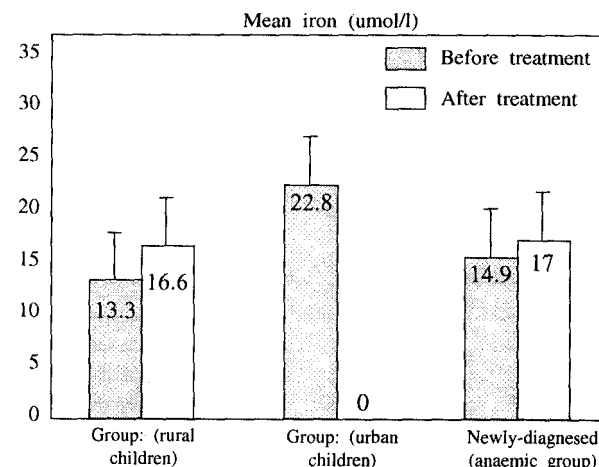


Figure 3

Mean distribution of the total binding iron capacity (TIBC) in the various groups before and after treatment

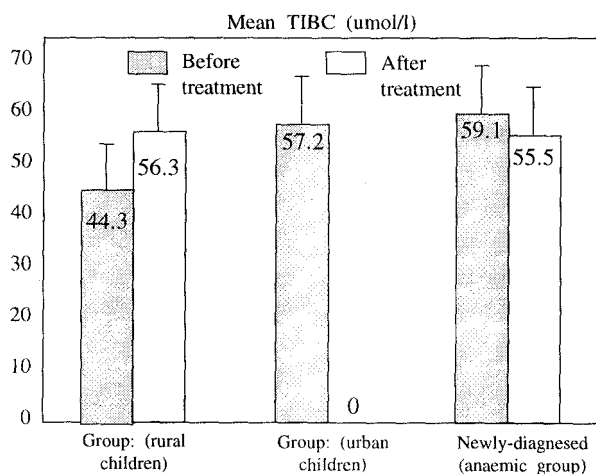
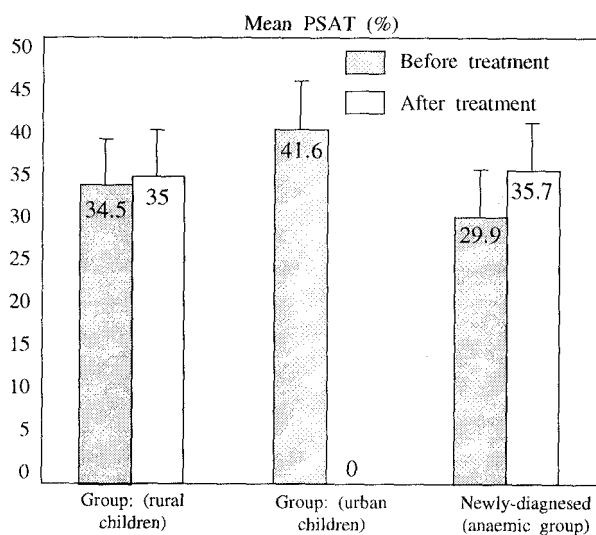


Figure 4

Mean distribution of percentage in Iron saturation (PSAT) in the various children groups before and after treatment with iron supplements



The newly diagnosed anaemic group had an entry TIBC value of  $59.1 \pm 2.9 \mu\text{mol/l}$  and  $55.5 \pm 1.4 \mu\text{mol/l}$  after supplementary therapy. Their urban control counterpart's TIBC level was estimated at  $57.2 \pm 1.2 \mu\text{mol/l}$ . Table 1 also depicts the serum ferritin level trends in the various groupings. The rural group had massively high levels of ferritin ( $290.6 \pm 31.7 \mu\text{g/ml}$ ) followed by the newly diagnosed anaemic group ( $95.6 \pm 12.1 \mu\text{g/ml}$ ). The urban control group had the least ( $45.8 \pm 6.2 \mu\text{g/ml}$ ) value as expected. The corresponding levels of haemoglobin of all the various groups investigated are similarly shown in

Table 1. Interestingly, the urban control group had the highest value of  $13.8 \pm 0.8 \text{ g/dl}$  followed by the rural group with a value of  $12.5 \pm 1.9 \text{ g/dl}$  before ferrous fumarate administration and  $13.5 \pm 2.4 \text{ g/dl}$  after ferrous fumarate supplements. The newly diagnosed anaemic group with an entry low Hb value of  $5.7 \pm 1.9 \text{ g/dl}$  improved to  $8.7 \pm 1.9 \text{ g/dl}$ . The over-all trends in the various serum analyses in this study is illustrated in Figures 1 - 4.

## DISCUSSION

Iron deficiency, the most common form of anaemia has a high prevalence among children of low socio-economic background. In a study by Murakawa *et al* iron deficiency anaemia was classified as  $\text{Hb} < 12.0 \text{ g/dl}$  and serum  $\text{Fe} < 12.5 \mu\text{mol/l}$ . Anaemia was classified as  $\text{Hb} < 12.0 \text{ g/dl}$ (7). Adopting this classification criterion in our study, 43.2% of all the subjects investigated were found to be iron-deficient. The incidence of iron deficient anaemia was 15.3% among the rural children, 0% among the urban children and 40.4% among the newly diagnosed anaemic children. On the other hand using the Hb index criterion, all (100%) of the newly diagnosed children were confirmed to have been anaemic, whilst 28.2% of the rural group were classified as anaemic and only 4.2% of the urban children were anaemic. These results confirm the observation made by Yip(9) that a good number of people who are iron deficient are not necessarily anaemic and that anaemia is present when iron deficiency is severe. A similar observation has been made that some school children who were found to be iron deficient had normal haemoglobin levels(6).

Haemoglobin estimation alone as a criterion for diagnosing iron deficiency anaemia is therefore misleading since as many as 33.3% of all the subjects investigated in this study would have been excluded from treatment. This study has also strongly indicated that of the total 57 newly diagnosed anaemic children who entered into the study, 23 were iron-deficient. This represents a 40.4% of all the reported cases suggesting that quite a significant proportion of anaemic cases are iron deficiency-related. However, it should be stressed that other causes such as Kwashiorkor, Marasmus among others may be the primary cause of the anaemia. Indeed, Kwashiorkor patients are known to have protein-energy malnutrition (PEM)(10). This is closely associated with folate deficiency which leads to megaloblastic anaemia. Nevertheless, there is an indication from this studies, that the diet of many children in the rural areas is quite deficient in iron and that some form of supplementation may provide them with sufficient iron to prevent deficiency. A similar assertion has been made by Palupi *et al*(10) who in their study, concluded that one-weekly iron supplementation was an effective, cheap and simple intervention which improved the iron status of children in an Indonesian community. The observed trends of ferritin as shown in Table 1 indicates that ferritin levels of the newly diagnosed anaemic group was twice as high as those of the urban group.

This trend possibly reflects iron maldistribution in which most of the iron is deposited in the iron stores rather than in the serum. The results from the iron binding studies indicated that there was no significant difference ( $p>0.05$ ) between the mean TIBC of the urban group and the newly diagnosed anaemic group suggesting anaemia alone does not necessarily affect the transport and tissue release of iron.

In conclusion, this study has shown that the diagnosis of iron deficiency anaemia is quite often different from the diagnosis of anaemia and therefore the appropriate indices and symptoms should be adopted in these two conditions since the inappropriate use of anaemic indices can be misleading and therefore lead to poor management of anaemia, iron deficient anaemia or even iron deficiency. The use of specific and sensitive indicators such as serum iron, total iron binding capacity (TIBC) and ferritin, should allow for the accurate evaluation of the iron status of an individual. This study has also shown that the incidence and prevalence of iron deficient anaemia is quite high in rural Ghana and that dietary iron supplements should be incorporated and enforced in major future nutritional policies if this condition is to be prevented.

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