PREVALENCE OF ANAEMIA,IRON DEFICIENCY AND IRON DEFICIENCY ANAEMIA IN NINE MONTH OLD INFANTS ATTENDING WELL CHILD CLINIC IN NAIROBI.

A dissertation submitted in part fulfillment of the requirements for the degree of Masters of Medicine (Paediatrics and Child Health) in the University of Nairobi.

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DECLARATION

This dissertation is a result of my original work and has not been presented for a degree in any other university.

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This work is dedicated to my dear wife Jackie and son Ashivira.

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LIST OF ABBREVIATIONS

DNA	- Deoxyribonucleic acid
dL	- Decilitre
DDST	- Denver Development Screening Test
fl	- Femtolitre
gms	- Grammes
Hb	- Haemoglobin
K.N.H	- Kenyatta National Hospital
L	- Litre
mg	- Milligrammes
ml	- Millilitre
MCV	- Mean Corpuscular Volume
mcm	- Micromoles
ng	- Nanogrammes
PCV	- Packed cell volume
pg	- Picogrammes
sTfR	- Serum Transferrin Receptor
TIBC	- Total Iron Binding Capacity
UNICEF	- United Nations Children's Fund
WHO	- World Health Organization
ZPP	- Zinc Protoporphyrin

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SUMMARY

Background

Iron deficiency is the most common micronutrient deficiency worldwide, with the highest prevalence found in developing countries. Consequences of iron deficiency include developmental delay, behavioural disturbances, growth retardation, increased susceptibility to infection, lead poisoning and poor response to iodine prophylaxis. There is very little local data on prevalence of iron deficiency with or without anaemia in an urban setting. In addition there is no national programme for screening for anaemia, iron deficiency and iron deficiency anaemia in infants.

Objectives

The primary objective of this study was to determine the prevalence of anaemia, iron deficiency and iron deficiency anaemia in nine month old infants attending immunization clinic

Secondary objectives were to determine the association between these parameters and developmental status and dietary habits.

Study design

This was a cross sectional descriptive study carried out over a five month period (4th August 2008 to 27th January 2009).

Setting

Immunization clinics in Kenyatta National Hospital, Riruta and Waithaka health centers. These health facilities all lie within the Nairobi west district, Nairobi province.

Subjects

Nine month old infants

Results

Overall 23 children (6.2%) were classified as normal, 175(47.2%) had iron deficiency, 127(34.2%) iron deficiency anaemia and 46(12.4%) anaemia due to other causes. This means that 81.4 % of the study population were iron deficient and 46.6% were anaemic.

Iron deficiency was the most common cause of anaemia in the population studied accounting for 74% of all causes. Amongst the population which had iron deficiency (n=302), 42.1% had developed iron deficiency anaemia.

Diet and psychomotor development were not associated with anaemia and iron status.

Conclusion

There is a high prevalence of iron deficiency and iron deficiency anaemia in the population studied. In addition, iron deficiency anaemia is the most common type of anaemia. There is need for universal provision of iron in the population studied.

BACKGROUND AND LITERATURE REVIEW

EPIDEMIOLOGY

Iron deficiency is the most common micronutrient deficiency worldwide, with the highest prevalence found in developing countries [1]. Children less than 2 years of age are considered as one of the highest-risk groups [1]. The World Health Organization (WHO) estimates that some two billion people are anaemic. [1] An estimated 10-20% of preschool children in developed countries, and an estimated 30-80% in developing countries, are anaemic at 1 year of age. [1] Anaemia is defined as haemoglobin levels that are below two standard deviations of the distribution mean in an otherwise normal population of the same gender and age at the same altitude. [1] In a normal population, two point five percent of the population would be expected to be below this threshold. Hence, anaemia would be considered a public health problem only when the prevalence of haemoglobin concentration exceeds five percent of the population. [1]

Data collected in United States national surveys revealed that thirty to forty percent of children under five years of age who had evidence of iron deficiency, were also anaemic [2]. In developing countries, it is estimated that the frequency of iron deficiency is two to five times that of iron deficiency anaemia. [1] Up to a prevalence of iron deficiency anaemia of forty percent, the prevalence of iron deficiency will be about two and a half times that of anaemia. [1]. A decision analysis using the US national survey data reached a similar conclusion [4]. The same analysis also concluded that screening becomes ineffective by the time the prevalence of anaemia is lower than five percent, because most of the cases are not related to iron deficiency. Screening for programmatic purposes should be considered for anaemia prevalences between five and twenty percent. When the prevalence of iron deficiency anaemia reaches the twenty to thirty percent level in the age-gender group under evaluation, it may be more effective and possibly more efficient to provide universal supplementation to that entire group than to screen for individual case-management purposes. [1]

A Nigerian study of iron status of fifty randomly selected infants attending an infant welfare clinic for routine immunization found that forty percent of the infants had packed cell volumes below 0.32, forty eight percent had haemoglobin below 10g/dL and twenty seven percent had mean corpuscular volume less that 70fL. [5]

A micronutrient survey done in Kenya found that among children six to seventy two months of age, prevalence was17.1, 41.5 and 11 percent for mild, moderate and severe anemia, respectively. [6]. A study in a Kenyan peri-urban health facility showed that iron deficiency anaemia had a prevalence of seven point four percent and was predominantly mild. [7]Age was found to be significantly associated with iron deficiency anaemia with a prevalence of fourteen point six percent in infants. This study recommended further investigation of iron deficiency anaemia amongst infants.

In order to plan effective interventions to combat both iron deficiency and anaemia there is an urgent need to have better information on the iron status of populations. This will enable the right interventions to be chosen in the first place and then, once programmes are in place, to have the right indicators to monitor their impact. [2]

BODY IRON COMPARTMENTS

There are three body iron compartments;

- functional
- storage
- transport

1. Functional

This is the iron involved in cellular metabolism. It includes iron contained in:

- hemoglobin and myoglobin (for transport and storage of oxygen),
- components of the electron transport chain, cytochromes and other enzyme systems,
- ribonucleotide reductase system required for the production of DNA,
- fenton catalyst system in the production of free radicals.

Iron deficiency is the state in which there is insufficient iron to maintain the normal physiological function of tissues such as the blood, brain, and muscles. Iron deficiency can exist in the absence of anaemia if it has not lasted long enough or if it has not been severe enough to cause the haemoglobin concentration to fall below the threshold for the specific sex and age group .[2] Evidence from animals fed on iron-deficient diets indicates that iron deficiency becomes detectable at about the same time in the blood, brain, and tissue enzyme systems.[2] The major liabilities of iron deficiency are associated only with depletion of this compartment .[8]

2. Storage

This consists of iron sequestered in relatively safe and non toxic form as ferritin and hemosiderin. It serves as a repository from which iron, lost from the functional compartment in excess of absorption, is replenished. This pool of iron is not being used by tissues. Iron depletion is the state in which storage iron is absent or nearly absent but the tissues that need iron are able to maintain normal physiological functions. [2]

3. Transport

This consists of the carrier molecule transferrin which links the storage and functional compartments.

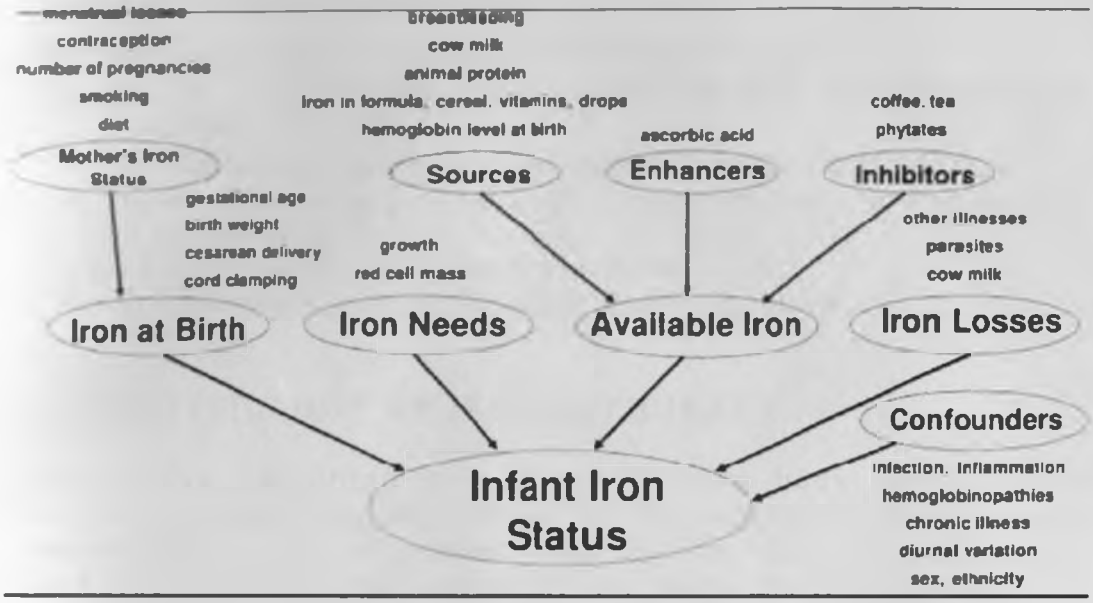
IRON BALANCE DURING INFANCY [9]

In the third trimester of pregnancy the foetus accumulates iron at a rate that is proportional to the increase in body weight, maintaining a total iron content of approximately seventy five milligrammes per kilo body weight. After birth, erythropoiesis falls as haemoglobin concentration tends to return to normal levels around twelve grammes per decilitre instead of the neonatal levels around seventeen grammes per decilitre .The iron derived from physiologic breakdown of red cells is accumulated in iron stores and subsequently used for new haemoglobin production. Thus the iron reserves of a healthy term infant may preserve it from iron deficiency anaemia for several months, even if the nutritional iron supply is nil.

Human milk iron content is low (zero point two to point four milligrammes per litre), but an estimated half of breast milk iron is absorbed. Thus in full-term infants, exclusive breastfeeding is sufficient to maintain an optimal iron balance for at least the first six months of life. Cow's milk iron is low in both concentration (around zero point four milligrammes per litre) and bioavailability because of the high calcium and phosphorus and low ascorbic acid content of cow's milk. In contrast to human milk, cow's milk and cow's milk formulas unfortified with iron cannot supply the infant with sufficient amounts of iron to maintain an optimal iron balance during the first 6 months of life.

After 6 months of life, iron body content increases substantially and the infant iron balance becomes critically dependent on dietary iron, provided by complementary foods containing highly bioavailable iron. Unless born prematurely or with low birth weight, most infants are at low risk of iron deficiency before 6 months of age because their iron stores are usually still adequate from the perinatal period. Accordingly, the earliest age to begin assessment of iron status is normally between 6 and 9 months; assessment may begin earlier in communities with low iron status

RISK FACTORS FOR IRON DEFICIENCY



Physiological model of iron status in infancy [10]

1. Diet

Dietary risk factors include;

- Dietary deficiency of iron; In plant based diets in developing countries most dietary iron is non-haem iron and its absorption is often less than ten percent [11] In contrast, in thirty to seventy percent of iron in meat is in haem form of which fifteen to thirty five percent is absorbed [11]. The bioavailability of non-haem iron is highly variable and influenced by several factors, including current diet and the amount of iron already present in the body. Bran, dietary fiber, calcium, tannins (in tea and coffee), and oxalates, phytates (e.g. sorghum, millet and maize), and polyphenols are all inhibitors iron absorption. Absorption is enhanced by reducing substances such as hydrochloric acid and ascorbic acid. The consumption of haem iron, even in small amounts, enhances the absorption of non-haem iron.
- Breastfeeding for less than six months.[12]
- Use of non iron fortified infant formula .[12]
- Excessive early consumption of cows milk (before one year of age) and consumption of greater than six hundred and eighty millilitres of whole cows milk per day after first year of life.[12]

2. Gastrointestinal blood loss.

This may be due to a number of causes including;

- Cow's milk which may cause occult gastrointestinal bleeding [12].
- Parasites e.g. Trichuris trichuria, Necator americanus.
- Other causes e.g. Meckel's diverticulum, inflammatory bowel disease.

3. Low birth weight and premature birth.
4. Severe maternal iron deficiency during pregnancy.

PATHOPHYSIOLOGY OF IRON DEFICIENCY

Quantitative phlebotomy to slowly reduce body iron content has shown that initially there is reduction of the storage compartment with a proportional decline in serum ferritin. Once the stores are exhausted ferritin concentration plateaus at approximately twelve nanogrammes per millilitre showing little further decline as functional compartment depletion progresses. [13] However as the functional compartment declines, the serum transferrin receptor concentration shows a highly consistent and progressive increase in proportion to the magnitude of the iron deficit [13] Further decline of the functional compartment results in anaemia. Between the point where storage iron depletion occurs and the development of anaemia the only measurement to accurately reflect the iron deficit is the serum transferrin receptor concentration. [13] Evidence from animals fed on iron-deficient diets indicates that iron deficiency becomes detectable at about the same time in the blood, brain, and tissue enzyme systems. [2]

CONSEQUENCES OF IRON DEFICIENCY

Iron deficiency with or without anaemia has important consequences for child health and development. These include;

1. Developmental

Iron Deficiency Anaemia and Infant Development

Iron deficiency anaemia has been seen to delay psychomotor development and impair cognitive performance of infants in Chile [14], Costa Rica [15], Guatemala [16], and Indonesia [17]. Neurological malfunction (as determined by electrophysiological measurements) has also been documented as being associated with iron deficiency [18].
These effects of iron deficiency anaemia on psychomotor development in infancy and early childhood (i.e. less than two years of age) are not likely to be corrected by subsequent iron therapy. [19, 20, 21, 22,]

Marginal Iron Deficiency and Infant Development

Mild iron deficiency without anaemia may also result in developmental abnormalities, these children improve significantly after iron therapy and thus it appears that if treatment is to be effective, it must be before anaemia develops. [21, 22] Children with marginal iron deficiency who did not improve with iron therapy had lower achievement scores when tested at five years of age. [9]

Although direct evidence demonstrating an effect of iron deficiency without anaemia on cognitive, behavioral, or other brain functions is limited, until shown otherwise it seems wise to assume that a gradation of effects of iron deficiency occurs in the brain, with milder anaemia and iron deficiency without anaemia resulting in more subtle but still potentially adverse brain effects, particularly if they occur during sensitive periods of development i.e. less than two years of age. [22]

Children and Adolescents with Iron Deficiency

Preschool children with iron deficiency with or without anaemia have been shown to have problems with inattention resulting in difficulties with higher cognitive function. Other studies have demonstrated lower scores on cognitive testing of iron deficient anaemic adolescents. [23, 24]

2. Behavioral

Iron deficient children tend to be tired and clingy with reduced interaction with other children. [25]

3. Growth

Children who are iron deficient tend to be shorter than non-iron deficient children.[26] Growth was shown to improve in iron-deficient children who were given supplementary iron in Indonesia [37], Kenya [28], and Bangladesh [29], as well as in the United Kingdom [30] and the United States [23].

4. Infections

Increased susceptibility to infections mainly of the upper respiratory tract which happen more often and have a longer duration in iron deficient anaemic than healthy children [31].

5. Response to Iodine Prophylaxis

Response to iodine prophylaxis is reduced in goitrous children with deficiencies of both iodine and iron. This is probably due to impairment of the heme-dependent enzyme thyroid peroxidase. [32, 33]

6. Lead Poisoning

Iron deficiency may increase the risk for chronic lead poisoning in children exposed to environmental lead. Both problems tend to be concentrated in children from lower socioeconomic strata residing in urban environments. [34]

INDICATORS OF IRON STATUS [12]

I. Hematological

Marker	Normal	Iron Depletion	Iron Deficiency Without anemia	Iron Deficiency Anaemia
Haemoglobin (hb)(g/dl)	> 11	> 11	> 11	< 11*
Mean Corpuscular Volume(MCV) (fL)	N 70 to 100	N 70 to 100	N 70 to 100	D < 70*
Red Blood Cell Distribution Width (%)	N < 15	N < 15	N < 15	I > 15
Reticulocyte Haemoglobin Content (pg)	N > 29	N > 29	N < 29	N < 29
Reticulocytes	N	N	I	I

N _ normal, I _ increased, D _ decreased
*Values for ages 6 mo to 2 y.

Comments

- **Haemoglobin** when used alone has low specificity and sensitivity [35]. This is simple to measure, and has functional and public health consequences.
- **Mean corpuscular volume** is a reliable but late indicator of iron deficiency, low values can also be due to thalassaemia. [35]
- **Mean corpuscular haemoglobin concentration**; can be characteristic of type of anaemia but it is slow to respond to iron deficiency.
- **Red blood cell distribution width** has low specificity therefore its use in screening is limited [12]
- **Reticulocyte hemoglobin content** has been shown to be an early indicator of iron deficiency in healthy subjects receiving recombinant human erythropoietin. [12] False normal values can occur when MCV is increased and in thalassaemia.
- **Reticulocytes** decrease with iron deficiency. However, the reticulocyte count increases with blood loss.[12]

2. Biochemical

Marker	Normal	Iron Depletion	Iron Deficiency Without Anaemia	Iron Deficiency Anaemia
Serum ferritin (mcg/dL)	N 100 ± 60	D < 20	D <10	D <10
Serum Iron (mcg/dL)	N 115 ± 50	N <115	D < 60	D <40
Total Iron-binding Capacity	N 330 ± 30	N 360 - 390	N/I 390 - 410	I > 410
Transferrin Saturation (%)	N 35 ± 5	N < 30	D <20	D < 10
Serum Transferrin Receptor (nmol/L)	N < 35	I >35	I >35	I >35
Zinc Protoporphyrin	N	N	I	I
Heme (mcmol/mol)	< 40	< 40	< 40	> 70

N _ normal, I _ increased, D _ decreased
mcg/dl;microgrammes per decilitre
nmol/l; nanomoles per litre

Comments [12]

- **Serum ferritin** It is a measure of the iron storage compartment. It is also an acute-phase reactant that can become elevated in the setting of inflammation, chronic infection, or other disease. When infection is present the concentration of ferritin may increase even if iron stores are low; this means that it can be difficult to interpret the concentration of ferritin in situations in which infectious diseases are common.
- **Serum iron** may not reflect iron stores accurately because it is influenced by several additional factors, including iron absorption from meals, infection, inflammation, and diurnal variation.

- **Total iron binding capacity(TIBC)** is affected by factors other than iron status. For example it is decreased with malnutrition, inflammation, chronic infection and cancer.
- **Transferrin saturation** influenced by the same factors that affect TIBC and serum iron concentration and is less sensitive to changes in iron stores than is serum ferritin
- **Serum transferrin receptor (sTfR)** this receptor is present on reticulocytes and is shed from the membrane as the reticulocyte matures. Its primary function is to bind to differic transferrin and to internalize it by receptor mediated endocytosis. With tissue iron deficiency, there is a proportional increase in the number of transferrin receptors. As it is not substantially affected by the acute-phase response, sTfR is therefore useful as an early marker of iron deficiency, but it also may differentiate between iron deficiency anemia and anemia of chronic disease. [12]The sTfR is directly correlated with the total mass of erythroid precursors; the only other determinant is tissue iron deficiency which increases the sTfR in proportion to the severity of the iron deficit. [13].The concentration of sTfR is also increased in haemolytic anaemia and thalassaemia. [2]This is the single most sensitive indicator of functional iron depletion and it is also the functional depletion marker most likely to become abnormal early in deficiency. [13] There is no universal reference value for sTfR but the WHO recommends application of thresholds recommended by manufacturer of assay until an international reference standard is available. [2]
- **Zinc protoporphyrin(ZPP)/heme** reflects iron status during hemoglobin synthesis and detects iron deficiency before the onset of anemia. ZPP reflects a shortage in the supply of iron in the last stages of making haemoglobin so that zinc is inserted into the protoporphyrin molecule in the place of iron. Although ZPP and erythrocyte protoporphyrin can be measured by using affordable, clinic-based methods, both are elevated with lead poisoning and chronic disease, making them less useful for the diagnosis of anemia.

ASSESSING THE IRON STATUS OF POPULATIONS [2]

The concentration of haemoglobin should be measured, even though not all anaemia is caused by iron deficiency. Measurements of serum ferritin and transferrin receptor provide the best approach to measuring the iron status of populations. In places where infectious diseases are common, serum ferritin is not a useful indicator because inflammation leads to a rise in the concentration of serum ferritin as a result of the acute phase response to disease. If infectious diseases are seasonal, then the survey should be done in the season of lowest transmission. In general the concentration of transferrin receptor does not rise in response to inflammation so that, when combined with the concentration of serum ferritin, it is possible to distinguish between iron deficiency and inflammation.

INTERVENTION STRATEGIES

1. Iron Supplementation

This is the provision of iron usually in doses higher than fortification but without food. [35]

2. Education and Dietary Modification or Diversification. [35]

This is the most sustainable intervention but change of dietary practices and preferences is difficult and foods that provide bioavailable iron e.g. meat are expensive.

3. Iron fortification of food

This is the public health policy of adding iron to foodstuffs to ensure that minimum dietary requirements are met. Prerequisites for implementation of this strategy include the identification of an appropriate food vehicle that reaches the target population, that is centrally processed, and that is widely available and consumed in relatively predictable amounts by vulnerable population group.

JUSTIFICATION

The prevalence of anaemia and iron deficiency varies in different populations [11].There is very little local data on prevalence of iron deficiency with or without anaemia in an urban setting. In addition there is no national programme for screening for iron deficiency and iron deficiency anaemia.

The adverse effects of iron deficiency anaemia on infant development might be only partially reversible [19].There are about two to five times more iron-deficient than anaemic individuals [1] and it is also recognized that even without anaemia, mild to moderate iron deficiency has adverse functional consequences [22].

The earliest age to begin assessment of iron status is normally between six and nine months as most infants are at low risk before six months of age because their iron stores are usually still adequate from the perinatal period. [1] The nine month age group is accessible, vulnerable and representative as they attend child health clinics (for measles vaccination) where assessments can be conducted. [1]

OBJECTIVES

Primary Objective

1. To determine the prevalence of anaemia, iron deficiency and iron deficiency anaemia in nine month old infants attending immunization clinic in Kenyatta National Hospital, Riruta and Waithaka health centres.

Secondary Objectives

1. To determine what proportion of those with anaemia and those with iron deficiency have iron deficiency anaemia.
2. To asses the influence of diet on anaemia, iron deficiency and iron deficiency anaemia.
3. To determine the association between infant development, anaemia, iron deficiency and iron deficiency anaemia in this setting.

METHODOLOGY

a) STUDY SITES

Immunization clinics at Kenyatta National Hospital (KNH), Riruta and Waithaka health centres. KNH is a state corporation at the top of the referral system in the health sector in Kenya. The total bed capacity is 1800. However on any given day the hospital hosts in its wards between 2500 and 3000 patients. On average the hospital caters for over 80,000 in-patients and over 500,000 out-patients annually. Riruta and Waithaka health centres are primary care facilities run by the Nairobi city council serving a peri-urban population. They offer outpatient services only. Riruta health centre serves the Riruta and Kawangware divisions of Nairobi with a combined catchment population of 140,000. Waithaka health centre serves Waithaka division with a catchment population of 46,000.

b) STUDY POPULATION

Nine month old infants attending immunization clinic in Kenyatta National Hospital, Riruta and Waithaka health centres.

c) STUDY DESIGN

Cross sectional descriptive study.

d) SAMPLE SIZE ESTIMATION

Using Fischer's formula for sample size estimation in prevalence studies;

$$n = \frac{Z^2 (1-\alpha / 2) P (1-P)}{d^2}$$

Where;

n = sample size

p = estimated prevalence of anaemia in infants; 40 percent from a Nigerian study (5)

d = precision (five percent)

$Z^2 (1-\alpha/2)$ = the square of the standard normal deviation corresponding to a confidence interval of ninety five percent i.e. 1.96^2

Power; eighty percent

n = 369

e) SAMPLING METHOD

Proportionate allocation of sample size was done based on the average number of clients in each health facility over the previous one year. A total of 3371 infants received measles immunization in the three facilities between June 2007 and May 2008.In KNH, 608 infants (18.1% of the total) were seen with 668 (23.2%) and 1981 (58.8%) in Waithaka and Riruta respectively. Subsequently consecutive sampling was done in each facility until the sample size was achieved over a four month period (3rd August to 27th January 2009). A total of 371 subjects were thus recruited, 218 from Riruta, 86 from Waithaka and 67 from KNH.

f) INCLUSION AND EXCLUSION CRITERIA

Inclusion;

- nine month old infants coming for measles vaccine
- weight for age greater than eighty percent.
- weight for length greater than eighty percent.
- Parental/guardian informed consent.

Exclusion:

- infants born prematurely or with low birth weight.
- infants known to have hereditary anaemia.
- family history of hereditary anaemia.
- known bleeding tendency.
- history of blood transfusion or supplementation/treatment with iron.

g) DEFINITIONS

- Anaemia : haemoglobin concentration of less than 11.0 g/dL.[1]
- Iron deficiency as serum transferrin receptor (sTfR) concentration of greater than 8.3 ug/ml. The normal range for serum sTfR as measured by the Ramco sTfR assay has been determined to be 2.9 - 8.3 ug/ml.
- Iron deficiency anaemia as haemoglobin, less than 11g/dL plus mean corpuscular volume less than 70 fL and serum transferrin receptor greater than 8.3ug/ml.
- Normal; no anaemia, iron deficiency or iron deficiency anaemia.

f) PROCEDURES

Recruitment

Patients were recruited by the reception nurse using the inclusion and exclusion criteria.

History

Demographic information was collected using questionnaires (appendix 1) which included the study subjects' name, age, and sex. Infant feeding practices were evaluated using the twenty four hour dietary recall and seven day food frequency techniques (appendix 1).

Physical Examination

A complete physical examination was carried out by the investigator and the presence or absence of palmar pallor recorded (appendix 1). Nutritional status was assessed by measuring weight and length. Weight for age and weight for height was subsequently obtained from WHO charts [36] (appendix 3).

Developmental Screening.

Development screening was done by the investigator using the Denver Developmental Screening Test(DDSTII, Appendix 4).The (DDST) was standardized and published in 1967. It was subsequently revised and re-standardized in 1992 i.e. the DDST II.DDST II contains 125 test items in four developmental sectors i.e. gross motor , language, fine motor–adaptive and personal social.

The child's test behavior was also assessed and rated. The test behavior may affect test scores but this is not factored in the final scoring and is thus a limitation of this screening test.

Laboratory Investigations

Full Blood Count

One milliliter of venous blood was collected in an EDTA vacutainer and a full blood count done by the MS4 machine in the department of Paediatrics laboratory University of Nairobi to obtain haemoglobin and mean corpuscular volume.

Serum Transferrin Receptor

One ml whole blood was collected aseptically, the blood was allowed to coagulate and the clot separated from the serum by centrifugation. Serum transferrin receptor concentration was determined using the Ramco's transferrin receptor (TfR) assay. This is an enzyme immunoassay based upon the double antibody sandwich method (appendix 5) this assay was carried out in the Immunology department University of Nairobi

h) DATA MANAGEMENT AND STATISTICAL ANALYSIS

Data collected was entered into a Microsoft Access database then cleaned and verified. The data was then subsequently stored in compact discs and a flash disc. Data analysis was done using SPSS version 11.5 and Epinfo. Descriptive analysis for categorical variables was done using frequencies distributions. For continuous variables, mean, standard deviation median and range were computed. Inferential statistics for categorical variables were computed using Chi Square statistics for measure of associations. For continuous variables, if the variable was normally distributed T test technique was used, if skewed Man Whitney U test was used.

i). ETHICAL CONSIDERATIONS

The study was undertaken after approval by the Department of Paediatrics University of Nairobi and the Ethical review committee Kenyatta National Hospital. Permission was sought from the Nairobi City Council to collect data from Waithaka and Riruta Health centres.

Parents/Guardians were given full explanation of the study and written consent was sought from them. Costs of laboratory tests were borne by the investigator. All patient information was treated with strict confidentiality. Results of the assessments were communicated to the Parents/Guardians. Prescriptions for folate and iron were given for all patients with iron deficiency and iron deficiency anaemia together with nutritional counseling.

RESULTS

DEMOGRAPHIC CHARACTERISTICS

Table 1: Demographic Characteristics of the study population

A total of 371 subjects were recruited. All the infants were nine months old. All infants had a weight for length and age greater than 2 standard deviations above the mean. Kenyatta National hospital, Riruta and Waithaka are located within Nairobi West district in Nairobi province

Demographic Variables	Riruta (n=218)		KNH (n=67)		Waithaka (n=86)		Total (n=371)	
	n	%	n	%	n	%	n	%
Sex								
Male	119	54.6	38	56.7	55	64.0	212	57.1
Female	99	45.4	29	43.3	31	36.0	159	42.9
Maternal marital status								
Married	197	90.4	57	85.1	75	87.2	329	88.7
Single	21	9.6	9	13.4	10	11.6	40	10.8
Separated	0	0.0	1	1.5	1	1.2	2	0.5
Maternal education								
Primary	122	56.0	6	9.0	47	54.7	175	47.2
Secondary	85	39.0	33	49.3	33	38.4	151	40.7
Tertiary	9	4.1	28	41.8	5	5.8	42	11.3
No Formal	2	0.9	0	0.0	1	1.2	3	0.8
Maternal employment status								
Employed	21	9.6	24	35.8	10	11.6	55	14.8
Unemployed	158	72.5	22	32.8	56	65.1	236	63.6
Self employed	39	17.9	21	31.3	20	23.3	80	21.6
Maternal Age (Years)								
Mean	25.68		28.43		24.76		25.96	
Standard deviation	5.00		5.25		4.79		5.14	
Infant Age (mths)								
Mean	9		9		9		9	

There was no significant difference in distribution of sex and marital status by site (p=0.330 and 0.423 respectively). There was a significant difference (p< 0.0010) in terms of maternal occupation by site with K.N.H having the largest proportion of employed (35.8%) followed by Waithaka (11.6%) and Riruta (9.6%).The same distribution was seen with self employment; K.N.H (31.3%), Waithaka (23.3%), Riruta (17.2%).

There was a significant difference in distribution of education levels by site with all the mothers in K.N.H having received formal education compared to 99.1% in Riruta and 98.8% in Waithaka. There was significant difference in mean maternal age between K.N.H on one hand and Riruta and Waithaka on the other hand. However, there was no significant age difference between Riruta and Waithaka.

SUMMARY DISTRIBUTION OF HAEMOGLOBIN, MEAN CORPUSCULAR VOLUME and SERUM TRANSFERRIN RECEPTOR

Table 2: Distribution of Hb, sTFR and MCV

Variable	n	Mean	SD	95% C.I
Hb (g/dl)	371	10.8	1.7	10.53-10.87
sTFR (ug/ml)	371	16.7	8.1	15.88-17.52
MCV (fl)	371	68.2	7.6	67.43-68.97

.Overall mean haemoglobin level in all sites was 10.8 ± 1.7 g/dl (95% C.I 10.53-10.87). The overall mean MCV in all sites was 68.2 ± 7.6 fl (95% C.I 67.43-68.97). Overall mean sFTR level was 16.7 ± 8.1 ug/ml (95% C.I 15.88-17.52).

Table 3: Distribution of Hb, MCV and sTFR by study sites

Variable	Riruta (n=218)		KNH (n=67)		Waithaka (n=86)	
	Mean (SD)	95% C.I	Mean (SD)	95% C.I	Mean (SD)	95% C.I
Hb	10.6 (1.7)	10.37-10.83	11.1 (1.7)	10.69-11.51	10.8 (1.7)	10.44-11.16
MCV	67.9 (6.9)	66.98-68.82	68.4 (8.4)	66.39-70.41	68.6 (8.5)	66.80-70.4
sTFR	16.2 (7.9)	15.15-17.25	19.7 (8.7)	17.62-21.78	15.7 (7.7)	14.07-17.33

There was between sites variation of haemoglobin ranging from 10.6 g/dl (Riruta) to 11.1 g/dl (K.N.H). The lowest MCV was in Riruta (67.9fl) and highest in Waithaka 68.6fl. All the three sites had the mean sTFR higher than the upper limit of 8.3ug/ml as shown in table 3.

Table 4: Comparison of mean Hb, MCV and STFR by study sites

Variables	Study Site	P value
Hb(g/dl)	Riruta vs. KNH	0.035
	Waithaka vs. KNH	0.224
	Riruta vs. Waithaka	0.415
MCV(fl)	Riruta vs. KNH	0.650
	Waithaka vs. KNH	0.931
	Riruta vs. Waithaka	0.539
SFTR(ug/ml)	Riruta vs. KNH	0.005
	Waithaka vs. KNH	0.004
	Riruta vs. Waithaka	0.596

Mean haemoglobin comparison between Riruta (10.6 g/dl) and KNH (11.1 g/dl) revealed a significant difference; (P=0.035). However, There was no significant difference in the mean Hb between Riruta (10.6 g/dl) and Waithaka (10.8 g/dl); (P=0.415). Likewise there was no significant difference between Waithaka (10.8 g/dl) and KNH (11.1 g/dl); (P=0.224). There was no statistically significant difference between the mean MCV as shown in table 4.There was a significant difference in mean sTFR between Riruta and KNH (P=0.005). The same was seen between Waithaka and KNH (P=0.0004). However, there was no significant difference between Riruta and Waithaka (P=0.596).

PREVALENCE OF ANAEMIA, IRON DEFICENCY AND IRON DEFICENCY ANAEMIA

Table 5: Distribution of Iron and Anaemia Status

Iron and Anaemia status	n	%	95% C.I
Normal	23	6.2	3.82-8.58
Iron deficiency	175	47.2	43.51-50.89
Iron deficiency anaemia	127	34.2	30.29-38.11
Other Anaemia	46	12.4	9.26-15.54
Total	**371**	**100**	

Overall 23 children (6.2%, 95% C.I 3.82-8.58) were classified as normal, 175(47.2%, 95% C.I 43.51-50.89) had iron deficiency without anaemia, iron deficiency anaemia 127(34.2% 95% C.I, 30.29-38.11) and anaemia due to other causes 46(12.4%, 95% C.I 9.26-15.54).This means that 81.4 % of well nine month old infants attending the clinics are iron deficient and 46.6% are anaemic.

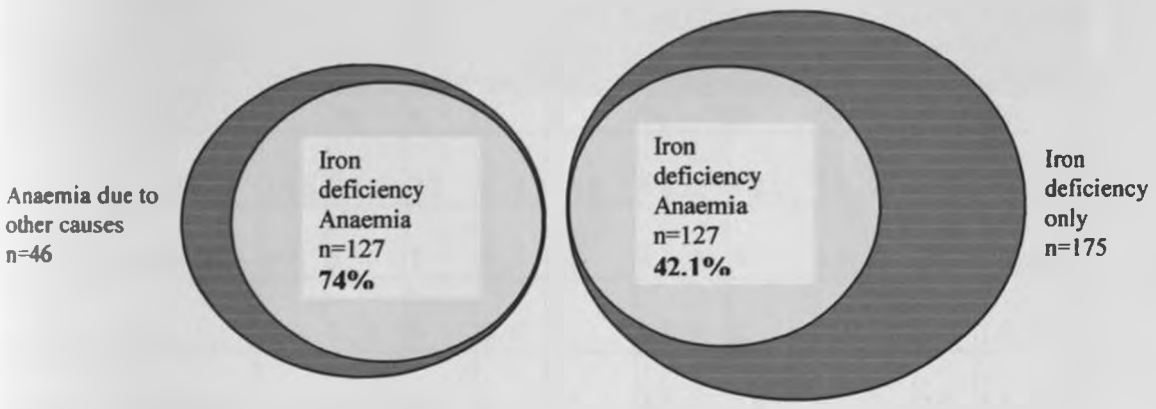
Table 6: Distribution of Iron and Anaemia status by study site

Iron and Anaemia status	Riruta		KNH		Waithaka		Total	
	n	%	n	%	n	%	n	%
Normal	11	5.0	3	4.5	9	10.5	23	6.2
Iron deficiency	97	44.5	41	61.2	37	43.0	175	47.2
Iron deficiency anaemia	78	35.8	17	25.4	32	37.2	127	34.2
Other Anaemia	32	14.7	6	9.0	8	9.3	46	12.4

The prevalence of iron deficiency anaemia was highest in Waithaka(37.2%). Iron deficiency was highest in KNH (61.2%) and anaemia caused by other factors was highest in Riruta (14.7%) There was no significant difference in the distribution of iron and anaemia status by sites (P=0.092).

PROPORTION OF IRON DEFICIENCY ANAEMIA IN INFANTS WITH IRON DEFICIENCY AND ANAEMIA

Figure 1: Iron deficiency anaemia in infants with iron deficiency and anaemia



In total 173 infants were anaemic, out of this 127 had iron deficiency anaemia meaning iron deficiency was the most common cause of anaemia in population studied accounting for 74%.There were 302 infants with iron deficiency and 127(42.1%) of them had iron deficiency anaemia. This means the prevalence of iron deficiency without anaemia (n=175) is1.4 times that of iron deficiency anaemia.

ANAEMIA AND IRON STATUS BY BREASTFEEDING PRACTICE

Table 7: Distribution of Iron and Anaemia status by breastfeeding practice

POPULATION SAMPLED	Total		Normal		Iron deficiency		Iron deficiency anaemia		Other anaemia	
	n	%	n	%	n	%	n	%	n	%
Currently breastfeeding	346	93.3	22	6.4	160	46.2	122	35.3	42	12.1
Stopped breastfeeding	19	5.1	1	5.3	12	63.2	3	15.8	3	15.8
Never breastfed	6	1.6	0	0.0	3	50.0	2	33.3	1	16.7
Exclusive breastfeeding	150	41.1	11	7.3	70	46.7	49	32.7	20	13.3
Not exclusive breastfeeding	215	58.9	12	5.6	102	47.4	76	35.3	25	11.6

Breastfeeding practices were classified as those currently breastfeeding, those who had breastfed for any duration but now stopped and those who had never breastfed. Those who were currently breastfeeding or had done so and stopped were further categorized into exclusively breastfed for six months and not exclusively breastfed

There was no significant difference in distribution of Iron and Anaemia status by breastfeeding practice overall (P=0.704). The prevalence of iron deficiency without anaemia amongst those who had stopped breastfeeding was 63.2 %(95% C.I 50.04-76.36) compared to 50% (95% C.I 21.71-78.29) of those that never breastfed, 47.4% (95% C.I 42.56-52.24) of those that did not exclusively breastfeed, 46.7 %(95% C.I 40.87-52.53%) of those that exclusively breastfed and 46.2% (95% C.I 42.35-50.05) of those that are currently breastfeeding. Anaemia is most prevalent in those that never breastfed (33.3+16.7=50%).

There was no significant difference in distribution of Iron and Anaemia status by breastfeeding practices in all the three sites. Analysis of association between Iron and Anaemia status by whether the patients were breastfeeding or not revealed no significant association; Riruta (P=0.454), KNH (P=0.766) and Waithaka (P=0.794.

The same scenario was seen when association between Iron and Anaemia status by whether the patient did exclusive breastfeeding or not was analysed; Riruta (P=0.252), KNH (P=0.569) and Waithaka (P=0.440).

ANAEMIA AND IRON STATUS BY IRON CONTENT OF CURRENT DIET

Table 8: Distribution of Iron and Anaemia status by iron intake (24 hour dietary recall)

Population Sampled	Iron poor diet (n=336, 90.57%)	Iron rich diet (n=35 9.43%)	Odds ratio (95% C.I)	P value
1. **OVERALL**				
Normal	22 (95.6%)	1 (4.4)	1	
Iron deficiency	157 (89.7)	18 (10.3)	0.4 (0.02-3.06)	0.594
Iron deficiency anaemia	117 (92.3)	10 (8)	0.53 (0.02-4.52)	0.871
Other anaemia	40 (86.9)	6 (13.1)	0.3 (0.01-2.56)	0.481
2. **RIRUTA**				
Normal	11 (100%)	0	1	
Iron deficiency	88(90.72)	9(9.27)	<0.01 (<0.01-5.51)	0.631
Iron deficiency anaemia	70 (89.75)	8(10.25)	<0.01 (<0.01-5.04)	0.968
Other anaemia	29(90.63)	3(9.37)	<0.01 (<0.01-7.13)	0.737
3.**K.N.H**				
Normal	2 (66.6%)	1 (33.3)	1	
Iron deficiency	34 (82.93)	7(17.07)	2.43 (<0.01-43.71)	0.944
Iron deficiency anaemia	15 (88.23)	2(11.76)	3.75 (<0.01-38.7)	0.930
Other anaemia	5 (83.33)	1(16.67)	2.5 (<0.01-195.3)	0.777
4.**WAITHAKA**				
Normal	9 (100%)	0	1	
Iron deficiency	35(94.6)	2(5.4)	<0.01 (<0.01 (562.16)	0.042
Iron deficiency anaemia	32 (100)	0	undefined	0.538
Other anaemia	6 (75)	2(25)	<0.01(<0.01-107.98)	0.280

Diet was described as being iron rich if any haem iron was eaten in the last seven days using the 24 hour dietary recall and the seven day food frequency techniques. Iron poor diet was defined as absence of any haem iron in the diet during the same period.

There was no statistically significant difference in distribution of iron and anaemia status by iron content of current diet in the previous 24 hours. Overall, 95.6% of those who were classified as normal had an iron poor diet compared to 89.7% of iron deficiency, 92.3% of iron deficiency anaemia and 86.9% of anaemia due to other causes.

In K.N.H, those with iron deficiency were more likely to have had an iron poor diet in the last 24 hours compared to those classified as normal (odds ratio 2.43,p=0.94). The same applies to iron deficiency anaemia (odds ratio 3.75, p=0.930) and anaemia due to other causes (odds ratio 2.5, p=0.777) compared to the normal infants. However these are not statistically significant differences. In Riruta and Waithaka the normal infants were more likely to have had an iron poor diet but this was not statistically significant.

Table 9: Distribution of Iron and Anaemia status by iron intake within 7 days (7 day food frequency).

Population Sampled	Iron poor diet (n=256, 69%)	Iron rich diet (n=115, 31%)	Odds ratio (95% C.I)	P value
1. **OVERALL**				
Normal	17 (73.9%)	6 (26.1)	1	
Iron deficiency	120 (77.4)	55 (22.6)	0.77 (0.26-2.23)	0.6
Iron deficiency anaemia	92 (72.44)	35 (27.55)	0.93 (0.30-2.77)	0.8
Other anaemia	27 (58.7)	19 (41.3)	0.5 (0.14-1.69)	0.21
2. **RIRUTA**				
Normal	8 (72.72%)	3 (27.27)	1	
Iron deficiency	73 (75.26)	24 (24.74)	1.14 (0.22-5.31)	0.854
Iron deficiency anaemia	53 (67.95)	25 (32.05)	0.8 (0.15-3.72)	0.582
Other anaemia	18 (56.25)	14 (43.75)	0.48 (0.08-2.59)	0.714
3. **K.N.H**				
Normal	1 (33.33%)	2 (66.67)	1	
Iron deficiency	21 (51.21)	20 (48.78)	2.1 (0.13-63.81)	1.00
Iron deficiency anaemia	10 (88.23)	7 (11.76)	2.86 (0.15-99.48)	0.897
Other anaemia	3 (50)	3 (50)	1.5 (0.25 -8.98)	0.813
4. **WAITHAKA**				
Normal	8 (88.89%)	1 (11.11)	1	
Iron deficiency	26 (70.27)	11 (29.72)	0.3 (0.01-2.92	0.473
Iron deficiency anaemia	29 (90.63)	3 (9.38)	1.21 (<0.01-16.91)	0.631
Other anaemia	6 (75)	2 (25)	0.38 (0.01-7.72)	0.910

There was no statistically significant difference in distribution of iron and anaemia status by iron content of current diet in the last seven days. Overall, 73.9% of those who were classified as normal had an iron poor diet compared to 77.4% of iron deficiency, 72.44% of iron deficiency anaemia and 58.7% of anaemia due to other causes.

In K.N.H, those with iron deficiency were more likely to have had an iron poor diet in the last seven days compared to those classified as normal (odds ratio 2.1,p=1.00). The same applies to iron deficiency anaemia (odds ratio 2.86, p=0.897) and anaemia due to other causes (odds ratio 1.5, p=0.813) compared to the normal infants. However these are not statistically significant differences. In Riruta, the normal infants were more likely to have had an iron poor diet but this was not statistically significant. On considering Waithaka, there was a statistically insignificant increased chance of an iron poor diet amongst those with iron deficiency anaemia compared to the normal infants.

Table 10: Distribution of Iron and Anaemia status by consumption of cow milk

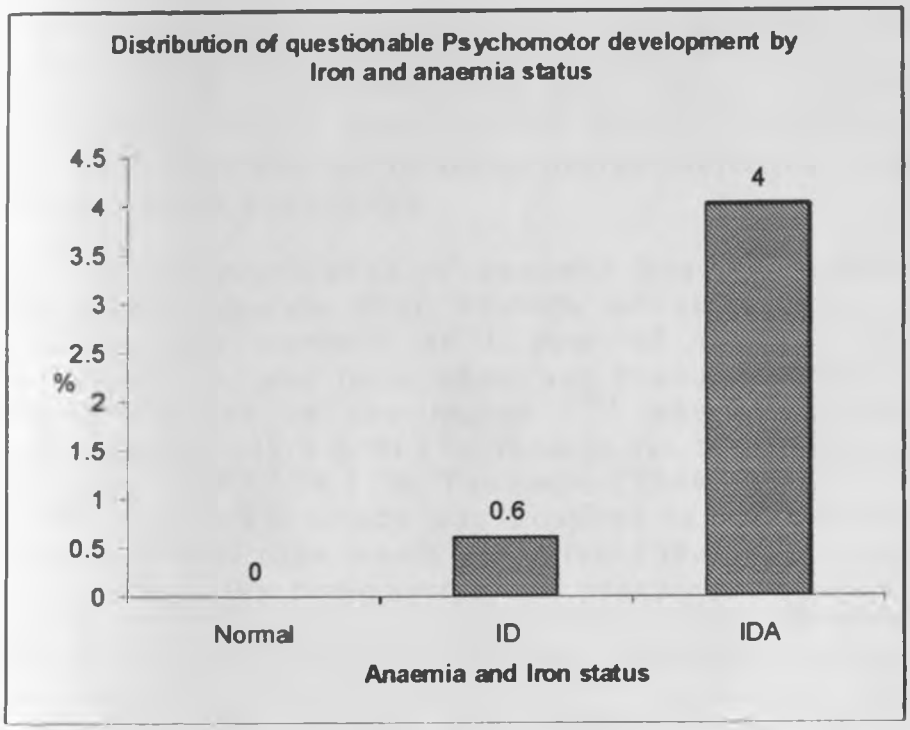
Population Sampled	Fed cow's milk (n=112)	Not fed cow's milk (n=259)	Odds ratio (95% C.I)	P value
1. **OVERALL**				
Normal	8	15	1	
Iron deficiency	44	131	0.63 (0.23-1.75)	0.462
Iron deficiency anaemia	43	84	0.96 (0.35-2.70)	0.878
Other anaemia	17	29	1.1 (0.34-3.56)	0.929
2. **RIRUTA**				
Normal	7	4	1	
Iron deficiency	67	30	1.28 (0.29-5.39)	0.980
Iron deficiency anaemia	48	30	0.91 (0.2-3.91))	0.844
Other anaemia	19	13	0.84 (0.55-1.59)	0.914
3. **K.N.H**				
Normal	3	0	1	
Iron deficiency	36	5	<0.01 (<0.01-23.2)	0.764
Iron deficiency anaemia	10	7	<0.01 (<0.01-4.59)	0.470
Other anaemia	3	3	<0.01 (<0.01-5.52)	0.453
4. **WAITHAKA**				
Normal	5	4	1	
Iron deficiency	28	9	2.49 (0.43-14.42)	0.430
Iron deficiency anaemia	26	6	3.47 (0.55-22.63)	0.252
Other anaemia	7	1	5.60 (0.35-181.22)	0.363

Any cow's milk feed in the last seven days was considered significant since any consumption of cow's milk in the first year of life is a risk factor for development of iron deficiency [12].

Overall, the odds of having fed on cow's milk were less amongst infants with iron deficiency (odds ratio 0.63, p=0.46), and iron deficiency anaemia (odds ratio 0.96, p=0.878) compared to the normal infants. Infants with other anaemia (odds ratio 1.1, p=0.46) were however more likely to have been fed cow's milk. These differences are statistically insignificant.

In Riruta the iron deficient infants were more likely to have been fed cow's milk compared to the normal infants (odds ratio 1.28, p=0.98). In K.N.H, normal infants were more likely to have been fed cow's milk compared to the iron deficient (odds ratio<0.01, p=0.764), iron deficient anaemic odds ratio (<0.01, p=0.470), and other anaemia (odds ratio<0.01, p=0.764). In Waithaka those with iron deficiency were more likely to have been fed cow's milk compared to those classified as normal (odds ratio 2.49, p=0.43). The same applies to iron deficiency anaemia (odds ratio 3.47, p=0.252) and anaemia due to other causes (odds ratio 5.60, p=0.363) compared to the normal infants. None of these differences in the three sites is statistically significant (p>0.05)

Figure 2



Amongst the infants with iron deficiency (n=175), 0.6% (n=1) had "questionable" score on DDSTII. On the other hand 4% (n=5) of the infants with iron deficiency anaemia scored questionable. The trend is towards more likely hood of having questionable development with worsening iron deficiency.

DISCUSSION

Kenyatta National Hospital, Riruta and Waithaka health centres are all situated in Nairobi West district. K.N.H is a national teaching and referral hospital while Riruta and Waithaka are pimary health care facilities run by the Nairobi city council. The well baby clinics in these health facilities serve an urban and periurban population.

The overall prevalence of anaemia was 46.6%.This is within the WHO estimate that 30-80% of children in developing countries are anaemic at 1 year of age [1]. However this prevalence is less than what was found in Demographic and Health surveys in the region [37] where the prevalence of anaemia (Hb <11.0 g/dL) in infants six to nine months of age: Ethiopia (2005) 76.3 %, Tanzania (2004) 83.2% and Uganda (2006) 92.2%.The study was hospital based and only included otherwise well nine month old infants therefore prevalence may be higher in the community. The prevalence of iron deficiency anaemia in the study population was 34.2%.WHO recommends that when the prevalence of iron deficiency anaemia reaches the 20-30% percent level in the age-gender group under evaluation, it may be more effective and possibly more efficient to provide universal supplementation to that entire group than to screen for individual case-management purposes. [1]

The cut off haemoglobin level of 11.0g/dl is based on two standard deviations of the distribution mean in an otherwise normal population of the same gender and age. However, there is disagreement about the appropriate cut-off values for the diagnosis of anaemia in infants [37]. The commonly used value for diagnosis recommended by WHO is Hb, 11.0 g/dL, use of this value allows comparison with other studies. Alternative cut-off level for insufficient Hb has been proposed as 10.0 g/dL for infants 9 mo of age [37]. Use of this conservative level will result in lower prevalence of anaemia but anaemia still remains the most prevalent nutritional problem [37]. In addition, the prevalence of iron deficiency and potential consequences remain the same.

Iron deficiency was the most common cause of anaemia in the population studied accounting for 74% of all causes. This prevalence of iron deficiency anaemia is more than the WHO estimate that IDA represents 50% of all anaemias [1]. The prevalence of iron deficiency (81.4%) was 1.75 times the prevalence of all anaemias (46.6%) which is less than the WHO estimate of 2.5 times the prevalence of anemia[1].

There was no association between diet and anaemia and iron status. In terms of breast feeding practice only a small number of infants (1.6%) had never been breastfed, and only 5.1% had been breastfed but stopped, this may account for the apparent absence of association. The same is seen in iron content of current diet as the majority had an iron poor diet in the last 24 hours and seven days (90.57%, 69% respectively). However, meeting iron requirements through food alone is nearly impossible particularly between 6 and 12 months of age when requirements remain very high and infants consume relatively small amounts of food [37]. In addition, in low income countries there may be inadequate maternal iron status, and inadequate birth practices (i.e. delayed cord clamping for two minutes) to promote the transfer of a portion of the birth iron via placental blood [37] .The possible contribution by these two factors to infant iron status was not assessed in this study.

There was no association found between anaemia, iron status and psychomotor development. This may be due to fact the development assessment tool has not been adapted for use in the population studied.

Strategies for prevention of iron deficiency at community level include dietary modification, food fortification and iron supplementation. Dietary modification may not be optimal for the infants as it has been shown that it is very difficult to provide adequate iron to infants through diet alone [37]. Iron fortification has been done locally where consumption of whole maize flour fortified with NaFeEDTA caused modest, dose-dependent improvements in children's iron status [38]. Iron supplementation may be associated with increased risk of malaria in endemic regions [39]. In such areas a cautious approach to supplementation based either on screening out iron-replete children or combining iron administration with effective disease control strategies is required .The study areas however do not have intense and all year round malaria transmission.

Other ways of preventing iron deficiency specifically in infants include prenatal iron supplementation and delayed cord clamping. Delayed cord clamping for two to three minutes transfers 35–40 mL of blood per kilogramme body weight to the infant [40]. This has been shown to have a beneficial effect through to six months of age [40].

STUDY LIMITATIONS

- The study was not adequately powered to allow sub-group analysis of the secondary objectives by study site.
- Iron content of current diet was not quantified and therefore the dietary assessment tool may not have been sensitive enough to pick up important differences.
- The Denver development screening tool has not been validated for use in the population studied.

CONLUSIONS

There is a high prevalence of anaemia, iron deficiency and iron deficiency anaemia in the study population since;

- 46.6% are anaemic
- 81.4% are iron deficient.
- 34.2% have already developed iron deficiency anaemia.
- 42.1% of infants with iron deficiency have iron deficiency anaemia.

In this population iron deficiency anaemia is the most common type of anaemia accounting for 74% of the study population with anaemia.

Diet and psychomotor development were not associated with anaemias and iron status.

RECOMMENDATIONS

In order to help prevent iron deficiency and iron deficiency anaemia amongst infants in the population studied two strategies are recommended;

- Reinforce exclusive breastfeeding to six months.
- Promote use of iron rich foods and iron fortified locally available foods e.g. maize flour for complementary feeding from age of six months.

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APPENDIX 1

DATA COLLECTION TOOL

1. DEMOGRAPHICS

Name

Study Number Study site
Age (months)
Sex
- Male [1]
- Female [2]

Maternal age (yrs)

Maternal marital status (tick)
- Married [1]
- Single [2]
- Separated [2]

Maternal level of education (tick highest level)
- Primary school [1]
- Secondary school [2]
- Tertiary education [3]
- Adult education [4]
- No formal education [5]

Maternal employment status (tick)
- Employed [1]
- Unemployed [2]
- Self employed [3]

2. BREASTFEEDING PRACTICES

2.1. Have you ever breastfed this child? ...
- Yes [1]go to 2.2
- No [2] go to 3.1

2.2 Are you currently breastfeeding this child?
- Yes [1]go to 3.1
- No [2]go to 2.3

2.3 How old was your child when you stopped breastfeeding?
months

3. COMPLEMENTARY FEEDING

3.1 How old was your child when you introduced other foods apart from breast milk? months.

3.2 Which food(s) apart from breast milk did you introduce first and at what age?

Food group	Code	Age (months)
Cows' Milk	1	
Animal Products(Meat, Fish, Liver, Chicken)	2	
Cereals(Maize, Millet, Sorghum)	3	
Cereal mixture (Maize, Millet, Sorghum with peanuts, beans ,fish)	4	
Legumes(Beans, Peas, Green-Grammes)	5	
Fruit (Mangoes, Bananas, Oranges, Lemons)	6	
Vegetables (Green Leafy, Cabbage, Cauliflower)	7	
Roots /Tubers(Irish Potatoes, Sweet Potatoes, Arrow Roots, Cassava)	8	
Infant Formula	9	
Water or glucose water	10	
Other (Specify)	11	

3.3 What have you fed your child since yesterday morning? (24 hour dietary recall AND 7 day food frequency)

Food group	Number Of Feeds Since Yesterday Morning	Number of days fed in the last one week
Breast milk		
Cows' Milk		
Animal Products(Meat, Fish, Liver, Chicken)		
Cereals (Maize, Millet, Sorghum)		
Cereal mixture (Maize, Millet, Sorghum with peanuts, beans ,fish)		
Legumes(Beans, Peas, Green-grams)		
Fruit (Mangoes, Bananas, Oranges, Lemons)		
Vegetables (Green Leafy, Cabbage, Cauliflower		
Roots /Tubers(Irish Potatoes, Sweet Potatoes, Arrow Roots, Cassava)		
Infant Formula		
Other (Specify)		

KEY

a) Food group; see above

b) Iron content

- Iron rich (haem-iron);animal products [1]
- Iron poor (non-haem) cows' milk, cereals, cereal mixture, legumes, fruit, vegetables, roots, tubers, [2]

c). Inhibitors of iron absorption; cereals, tea. [3]

4. DENVER DEVELOPMENT SCREEN II SCORING

	Gross motor	Language	Fine motor –adaptive	Personal – social
Number passed				
Number failed				

- Abnormal [1]
- Questionable [2]
- Normal [3]

2. ANTHROPOMETRIC MEASURES

Weight 1 Weight 2 Average Weight for age

Length 1 Length 2 Average Weight for length

5. PHYSICAL EXAMINATION

Palmar pallor

Present [1]

Absent [2]

6. LABORATORY INVESTIGATION RESULTS

6.1 Haemoglobin(g/dl).....

>11 [1] <11 [2]

6.2 Mean Corpuscular Volume (fl).....

>70 [1] <70 [2]

6.3 Serum Transferrin Receptor (ug/ml) .....

>8.3 [1] 2.9-8.3 [2]

APPENDIX 2

CONSENT FORM

Dr.Aluvaala Jalemba
P.O Box 301973
Department of Paediatrics and Child Health,
University of Nairobi,
Kenyatta National Hospital.
Tel; 0722-217034

Dear Parent/Guardian,

RE: PREVALENCE OF ANAEMIA, IRON DEFICIENCY AND IRON DEFICIENCY ANAEMIA IN NINE MONTH OLD INFANTS

Anaemia is insufficient amounts of haemoglobin in the blood. Haemoglobin is used to transport oxygen in the blood. Iron is a micronutrient used by the body to make haemoglobin. Iron deficiency is therefore the presence of inadequate amounts of iron in the body which can therefore lead to iron deficiency anaemia. Iron deficiency anemia is the most common micronutrient deficiency worldwide, with the highest prevalence found in developing countries. It is estimated that the frequency of iron deficiency in developing counties is two to five times that of iron deficiency anaemia. Iron deficiency and iron deficiency anaemia may cause irreversible delays in brain development.

This study aims to find out how common anaemia, iron and iron deficiency anaemia are amongst nine month old babies in nine month old babies attending immunization clinic in Kenyatta National Hospital, Riruta and Waithaka health centres. 3 millilitres of blood will be drawn to determine the presence of anaemia, iron deficiency and iron deficiency anaemia. A test of the child's development will also be carried out and assessment of diet done using a questionnaire. All patient information will be treated with the strictest confidentiality. Results of the assessments will be communicated to the Parents/Guardians.

The benefits of participating in this study are;

- Early detection of anaemia iron deficiency and iron deficiency anaemia and appropriate treatment prescribed.
- Early detection of developmental delay.
- There will be no charge for the tests carried out

There is a risk of pain and bruising when blood is being drawn. No more than three attempts at drawing blood will be made.

I agree to be part of this study

Name; Signature Date

Witness; Signature Date

A5.2 Calculating the child's weight-for-length

Determining child's % weight-for-length or SD weight-for-length

Refer to Table 35 on page 365.

- Locate the row containing the child's length in the central column of Table 35.
- Look to the left in that row for boys, and to the right for girls.
- Note where the child's weight lies with respect to the weights recorded in this row.
- Look up the adjacent column to read the weight-for-length of the child.

Example 1: Boy: length 61 cm, weight 5.3 kg;

this child is -1SD weight-for-length (90% of the median).

Example 2: Girl: length 67 cm, weight 4.3 kg;

this child is less than -4SD weight-for-length (less than 60% of the median).

Table 35. WHO/NCHS normalized reference weight-for-length (49–84 cm) and weight-for-height (85–110 cm), by sex

Boys' weight (kg)						Girls' weight (kg)				
-4SD 60%	-3SD 70%	-2SD 80%	-1SD 90%	Median	Length (cm)	Median	-1SD 90%	-2SD 80%	-3SD 70%	-4SD 60%
1.8	2.1	2.5	2.8	3.1	**49**	3.3	2.9	2.6	2.2	1.8
1.8	2.2	2.5	2.9	3.3	**50**	3.4	3	2.6	2.3	1.9
1.8	2.2	2.6	3.1	3.5	**51**	3.5	3.1	2.7	2.3	1.9
1.9	2.3	2.8	3.2	3.7	**52**	3.7	3.3	2.8	2.4	2
1.9	2.4	2.9	3.4	3.9	**53**	3.9	3.4	3	2.5	2.1
2	2.6	3.1	3.6	4.1	**54**	4.1	3.6	3.1	2.7	2.2
2.2	2.7	3.3	3.8	4.3	**55**	4.3	3.8	3.3	2.8	2.3
2.3	2.9	3.5	4	4.6	**56**	4.5	4	3.5	3	2.4
2.5	3.1	3.7	4.3	4.8	**57**	4.8	4.2	3.7	3.1	2.6
2.7	3.3	3.9	4.5	5.1	**58**	5	4.4	3.9	3.3	2.7
2.9	3.5	4.1	4.8	5.4	**59**	5.3	4.7	4.1	3.5	2.9
3.1	3.7	4.4	5	5.7	**60**	5.5	4.9	4.3	3.7	3.1
3.3	4	4.6	5.3	5.9	**61**	5.8	5.2	4.6	3.9	3.3
3.5	4.2	4.9	5.6	6.2	**62**	6.1	5.4	4.8	4.1	3.5
3.8	4.5	5.2	5.8	6.5	**63**	6.4	5.7	5	4.4	3.7
4	4.7	5.4	6.1	6.8	**64**	6.7	6	5.3	4.6	3.9
4.3	5	5.7	6.4	7.1	**65**	7	6.3	5.5	4.8	4.1
4.5	5.3	6	6.7	7.4	**66**	7.3	6.5	5.8	5.1	4.3
4.8	5.5	6.2	7	7.7	**67**	7.5	6.8	6	5.3	4.5
5.1	5.8	6.5	7.3	8	**68**	7.8	7.1	6.3	5.5	4.8
5.3	6	6.8	7.5	8.3	**69**	8.1	7.3	6.5	5.8	5
5.5	6.3	7	7.8	8.5	**70**	8.4	7.6	6.8	6	5.2
5.8	6.5	7.3	8.1	8.8	**71**	8.6	7.8	7	6.2	5.4
6	6.8	7.5	8.3	9.1	**72**	8.9	8.1	7.2	6.4	5.6
6.2	7	7.8	8.6	9.3	**73**	9.1	8.3	7.5	6.6	5.8
6.4	7.2	8	8.8	9.6	**74**	9.4	8.5	7.7	6.8	6
6.6	7.4	8.2	9	9.8	**75**	9.6	8.7	7.9	7	6.2
6.8	7.6	8.4	9.2	10	**76**	9.8	8.9	8.1	7.2	6.4
7	7.8	8.6	9.4	10.3	**77**	10	9.1	8.3	7.4	6.6
7.1	8	8.8	9.7	10.5	**78**	10.2	9.3	8.5	7.6	6.7
7.3	8.2	9	9.9	10.7	**79**	10.4	9.5	8.7	7.8	6.9
7.5	8.3	9.2	10.1	10.9	**80**	10.6	9.7	8.8	8	7.1