Dissertation on

"PROFILE AND OUTCOME OF NUTRITIONAL ANEMIA IN CHILDREN ATTENDING THE PEDIATRIC DEPARTMENT OF A TERTIARY CARE HOSPITAL IN CHENNAI"

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CERTIFICATE

Certified that this dissertation entitled **"PROFILE AND OUTCOME OF NUTRITIONAL ANEMIA IN CHILDREN ATTENDING THE PEDIATRIC DEPARTMENT OF A TERTIARY CARE HOSPITAL IN CHENNAI"** is a bonafide work done by **Dr.A.T.INDHUMATHI,** Post graduate, ESIC Medical College & PGIMSR, K.K. Nagar, Chennai, during the academic year 2013-2015

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DECLARATION

I solemnly declare that the dissertation entitled **"PROFILE AND OUTCOME OF NUTRITIONAL ANEMIA IN CHILDREN ATTENDING THE PEDIATRIC DEPARTMENT OF A TERTIARY CARE HOSPITAL IN CHENNAI"** is done by me at **ESIC Medical College & PGIMSR,** Chennai – 78 during 2013 to 2015 under the guidance and supervision of **Prof.Dr.SOWMYA SAMPATH, M.D., DNB.,** to be submitted to **The Tamilnadu Dr.M.G.R. Medical University** towards the partial fulfillment of requirements for the award of **M.D. DEGREE IN PEDIATRIC MEDICINE –BRANCH VII.**

Place: Chennai

Date: (**Dr. INDHUMATHI.A.T.)**

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CERTIFICATE OF APPROVAL

TO

DR. A.T. Indhumathi
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Dear Dr. A.T. Indhumathi,

The Institutional Ethics Committee of ESI PGIMSR reviewed and discussed you application for approval of the proposal entitled "Profile and Outcome of Anemia in Children Attending Pediatric Department in a Tertiary Care Hospital in Chennai" , No. 01/20/11/2013

The following members of the Ethics Committee were present in the meeting held on 20.11.2013 conducted at ESI PGIMSR, KK Nagar, Chennai-78.

S.No.	EC MEMBERS
1.	Dr. Saradha Suresh, Chairperson
2.	Dr.Kamalini Sridharan, Co-ordinator/ Prof. & HOD, Dept. of Anesthesia, ESI-PGIMSR.
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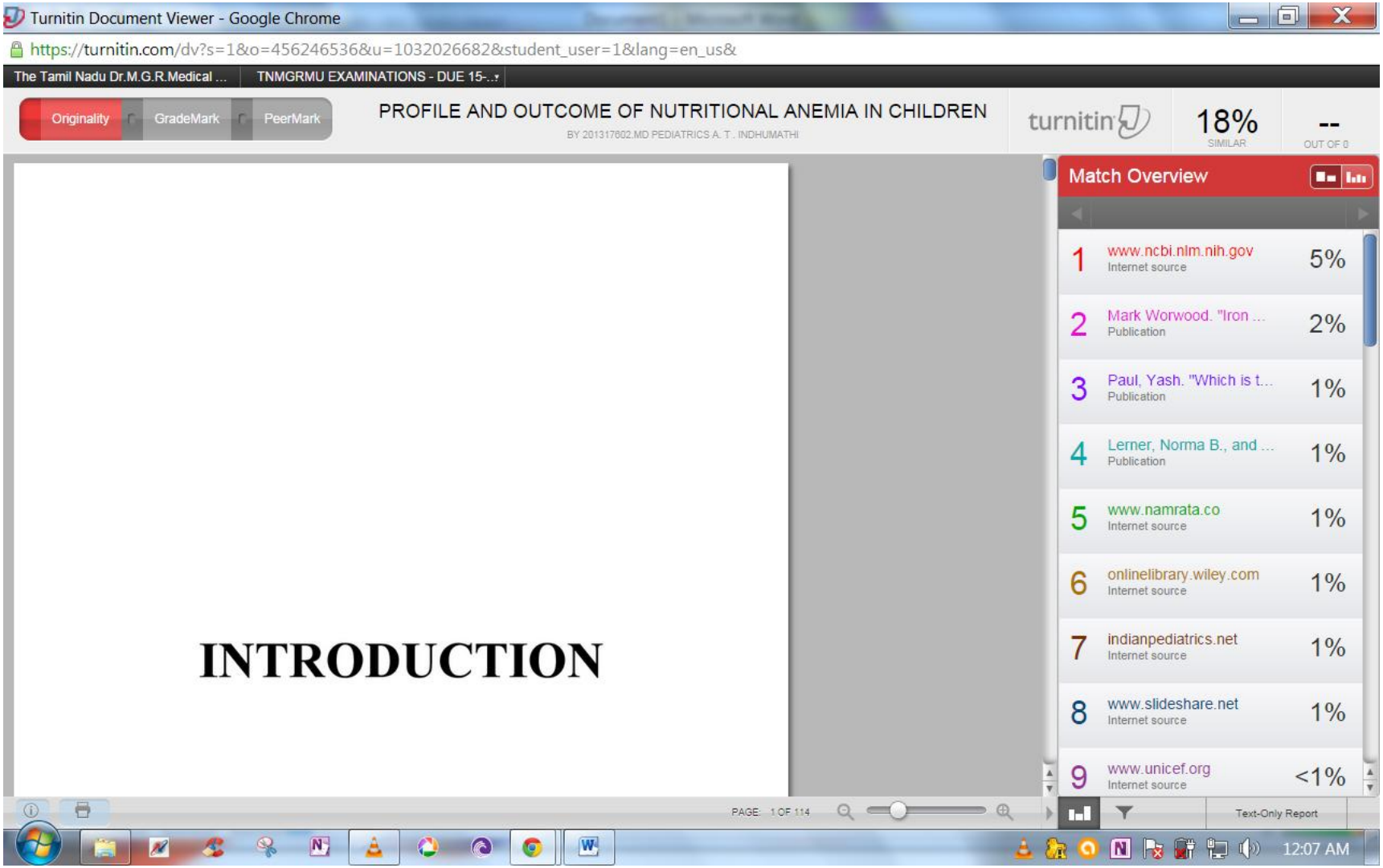
13.	Shri. K M Venugopal, Advocate, EC Member

The proposal is approved to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and significant adverse effects occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

[DR. SARADHA SURESH]
CHAIRPERSON
ETHICAL COMMITTEE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CONTENTS

ABBREVIATIONS

ANOVA	Analysis of variance
BMI	Body mass index
CRP	C-reactive protein
DALY	Disability Adjusted Life Years
DMT-1	Divalent metal transporter-1
DNA	Deoxy Ribonucleic acid
ESR	Erythrocyte sedimentation rate
EDTA	Ethyelene diamine tetra acetic acid
ESI	Employees state insurance
Hb	Hemoglobin
Hct	Hematocrit
IDA	Iron deficiency anemia
I-PMC	Iron poly maltose complex
LBW	Low birth weight
MCV	Mean corpuscular volume
MCH	Mean corpuscular hemoglobin
MCHC	Mean corpuscular hemoglobin concentration
MHA	Microcytic hypochromic anemia
NFHS	National Family Health Survey
NHA	Normocytic hypochromic anemia
NNA	Normocytic normochromic anemia

N	Number
PCV	Packed cell volume
RBC	Red blood corpuscle
RE	Reticulo-endothelial system
RDW	Red cell distribution width
SF	Serum ferritin
SD	Standard deviation
TIBC	Total iron binding capacity
UIBC	Unsaturated iron binding capacity
WHO	World Health Organization
WBC	White blood corpuscle
M	Male
F	Female

ABSTRACT

AIM: To study the profileof nutritional anemia in children and to determine the treatment outcome of iron deficiency anemia

METHODS: A total of 150 children with confirmed anemia were enrolled in this study. After a targeted history and clinical examination, hematological investigations were done. Children with microcytic hypochromic anemia were treated with oral iron and followed up after 1 and 3 months for improvement.

RESULTS: In the study population, 55 children were found to have microcytic hypochromic, 15 children had normocytic hypochromic and 80 children had normocytic normochromic anemia. Among the microcytic hypochromic group, the mean hemoglobin was found to be 8.22 ± 1.27 g/dL. Symptom analysis revealed 54.5% of children with iron deficiency anemia had lethargy and 34% had irritability. Pica was seen in 47.3% of children with microcytic hypochromic anemia. Hemoglobin levels correlated well with red cell indices, but it was found that 52.7% of children had normal serum ferritin levels. After treatment, it was found that the mean hemoglobin increased from 8.25 ± 1.30g/dLto 9.17 ± 1.36g/dLafter one month and after three months, there was a further increase z 10.39 ± 1.36g/dL.

CONCLUSION: In children with iron deficiency anemia, normal or high ferritin level should not distract the physician away from the diagnosis as co-existing infection and inflammation may confound the picture. In cases of normocytic, normochromic anemia, S. ferritin levels can mask a latent iron deficiency.Hence, a therapeutic trial of oral iron is more cost effective.

KEYWORDS: Children, Iron deficiency anemia, Red cell indices, Iron profile, Treatment outcome.

INTRODUCTION

1.1. History of iron deficiency:

Iron deficiency, described in various texts since ancient times, is 'probably the most frequent nutritional deficiency in the world'. Globally about 1.62 billion individuals are said to be suffering from iron deficiency anemia.[1] Around 1500 B.C, *Papyrus Ebers*, which is considered to be the oldest manual of therapeutics, has described a similar disease condition characterized by pallor, dyspnea, and edema.[2]

In 1713, Lemery and Geoffroy, and in 1747, Menghini found residual particles which were attracted to lodestone after burning blood to ash and thus presumed those particle to be made up of iron. The German biochemist Felix Hoppe-Seyler (1825–1895), using absorption spectrometry showed that hemoglobin is made up of a complex of haematin and protein. Stokes (1819–1903), a professor of mathematics at the University of Cambridge, showed that hemoglobin exists as two forms and demonstrated the changes between them in response to oxygen administration.

Bunge (1902, Basle) was the first to suggest the fact that iron deficiency caused hypochromic anemia. He also showed that iron was found in higher concentrations in the liver and kidneys of newborn

infants than older infants, children or adults. He also stated that iron content in human milk was very low and the iron derived from hemoglobin in meat was poorly absorbed. This fact was very much in contrast to today's evidence which suggests that haem iron is rapidly absorbed from the gastrointestinal tract. He was the first to recognize that a high concentration of iron was present in egg yolk, spinach, apples, lentils and beef. Koilonychia, a specific feature of iron deficiency also represented in the ancient literature as 'Lydney hand' was described in the twentieth-century by Kaznelson (1931).

In the middle of the 16th century, European physicians had treated a disease condition known as chlorosis or "green sickness". This condition had been treated with iron salts and other remedies (including, oddly enough, phlebotomy) in France, by the middle of the 17th century. Not long thereafter, Sydenham had also recommended iron as a specific remedy for chlorosis.[3]

In 1832, pills containing 1.39 g of ferrous sulphate and 0.1 g of potassium carbonate were widely recommended for chlorosis and other conditions. These pills were introduced by Blaud. The original pills contained 64 mg of iron. Later on, arsenic was often included to these

pills as many physicians thought iron combined with arsenic was more effective. This trend continued even into the 1930s.

Finally, the study of blood cell morphology was made possible by the pioneering work of Paul Ehrlich (1854–1915) who developed aniline dyes to stain blood films and lead to the birth of modern hematology.

Only after the 1932 studies by Heath, Strauss, and Castle that it was shown that the response of anemia to iron was stoichometrically related to the amount of iron given and that chlorosis was, indeed, iron deficiency.

1.2. Prevalence:

Iron deficiency is the most widespread nutritional disorder in the world. Anemia is the most common problem among micronutrients deficiency. Anemia affects health, education, economy and thereby, productivity of the entire nation. Thus, anemia constitutes a public health epidemic. The numbers are staggering. Over 30% of the world's population is anemic, mainly due to iron deficiency. This figure is frequently exacerbated by malaria and worm infections in developing countries.

Impact of anemia in South East Asia:

South East Asia has the largest number of anemic persons, including children contributing to 12,500,000 Disability Adjusted Life-Years (DALYs) and 324,000 deaths in this region, which is the highest in the world. More than 66% of the children in this region are anemic. In children aged less than two years, the prevalence of anemia may exceed 90%.[4]

Magnitude of Anemia in Indian Children:

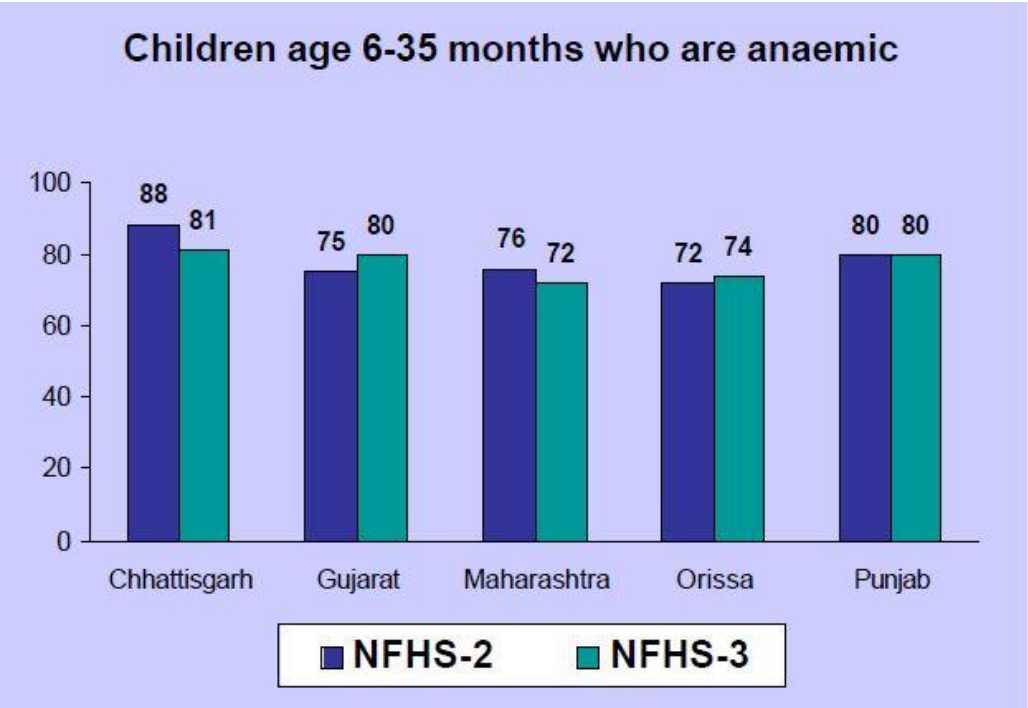


Fig.1 Comparison of anemia among 6 – 35 months children–NFHS 2 and NFHS 3

Anemia has been a big public health problem in India. The prevalence of anemia among children less than five years of age has been shown to be around 70% as per the data collected by National Family Health Survey (NFHS) III, with Bihar topping the list. The prevalence of anemia increases to 79% among children aged less than three years, with the highest prevalence among six to twenty-three months age group. This value, when compared to NFHS II survey done six years prior to the NFHS III survey, was five percent more. However, the prevalence of severe anemia had slightly reduced in the same period.[5]

Iron deficiency affects the activity of numerous enzymes adversely. Infants, whose organs are still developing, including the brain, are affected significantly by anemia resulting in growth retardation and impairment of intellectual development. In these modern times where we strive for millennium development Goal 4, this silent morbidity is completely unacceptable as children are denied of their right to full mental and emotional development, even before they reach a classroom.

At the same time, iron deficiency anemia can usually be prevented easily at a very low cost. The cost-benefit ratio of implementing preventive programs for iron deficiency is recognized as one of the highest in the realm of public health.

1.3. Nutritional anemia in children:

The term ‘nutritional anemia’ includes all pathological conditions characterized by low blood hemoglobin concentration, which may be due to a deficiency in one or several nutrients. Iron, folic acid and vitamin B_{12} are the main nutrients required for the synthesis of hemoglobin. By far, the first cause of nutritional anemia worldwide is iron deficiency. Folic acid deficiency is less common. Rarer among the main nutrient deficiencies is Vitamin B_{12} deficiency. Also folic acid deficiency is often observed with iron deficiency.[4]

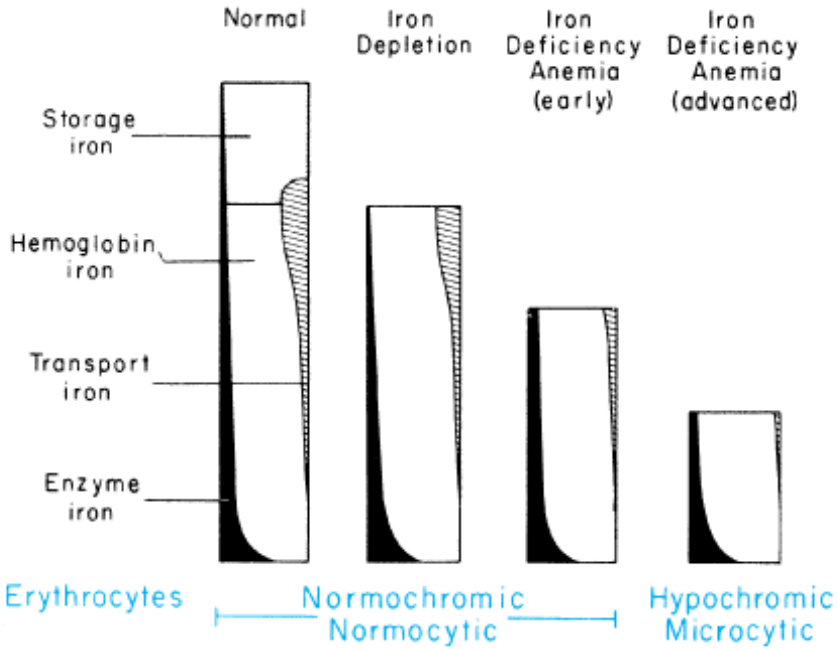
1.4. Iron deficiency:

Iron deficiency is defined as a condition characterized by signs of compromise in supply of iron to tissues including the erythrocyte, as there are no mobilizable iron stores. Iron deficiency is usually the result of inadequate bioavailable dietary iron, increased iron requirement during rapid growth, and increased blood loss for any reason.[6]

Iron deficiency anemia:

Anemia, when caused by severe iron deficiency is termed as iron deficiency anemia (IDA) and represents a very late stage of iron deficiency. Iron deficiency anemia is commonly associated with

decreased physical capacity, reduced immunity and cognitive impairment. Severe iron deficiency anemia also affects the capacity to maintain body temperature and is also life-threatening.



Source: Lichtman MA, Beutler E, Kipps TJ, Seligsohn U, Kaushansky K, Prchal JT: *Williams Hematology*, 7th Edition: http://www.accessmedicine.com

Diag. 1 Stages of depletion and status of compartments of iron[3]

Hemoglobin levels:

World Health Organization defined criteria, using hemoglobin levels for anemia classification[7]

	Hemoglobin (g/dL)			
Age Groups	**No anemia**	**Mild**	**moderate**	**severe**
6 -59 months of age	≥ 11	10 – 10.9	7 – 9.9	< 7
5 – 11 years of age	≥ 11.5	11 – 11.4	8 – 10.9	< 8
12-14 years of age	≥ 12	11 – 11.9	8 – 10.9	< 8

Table - 1 : Hemoglobin values

Causes of iron deficiency anemia:

Decreased intake or absorption	1. Insufficient dietary iron 2. Too early introduction or delayed introduction of complementary feeding without supplementation of iron 3. Poor bioavailability due to increased absorption inhibitors and decreased absorption enhancers
Increased requirement	Rapid growth phase in infancy and early childhood
Increased blood loss	1. Infections (hookworm,trichuris trichura, plasmodium, helicobacter pylori) 2. Gastrointestinal blood loss (Meckels diverticulum, peptic ulcer, esophageal varices, hemorrhoids, hereditary telangiectasia, ulcerative colitis, diverticulosis, angiodysplasia) 3. Other causes (Hemoglobinuria, Widespread bleeding disorders) 4. Malabsorption (Gluten-induced enteropathy, gastrectomy, atrophic gastritis, chronic inflammation) 5. Bovine milk allergy

Table 2 Causes of iron deficiency anemia[8]

Pathophysiology of anemia in children:

At birth, hemoglobin concentrations are normally higher than at any other time of life. This occurs as a result of the adaptation of the fetus to the hypoxic environment of the uterus. The neonatal reserves of storage iron are also relatively generous.

From two months of age, the hemoglobin concentration starts declining from a mean of 17.0 g/dL at birth and reaches a low level of 11.0 g/dL. This occurs due to breakdown of hemoglobin in order to meet the iron needs. This decrease in hemoglobin concentration is known as ‘early anemia of infancy’. This condition is usually unresponsive to iron treatment and hence, distinguished from the ‘late anemia of infancy’. Thus, in a term infant, the total body iron between birth and four months of age is almost not changed and there is only a modest need for exogenous iron during this period.

After about four months of age, the period of continued rapid growth begins and a gradual shift to marginal iron reserves occurs. Between four and twelve months, blood volume is rapidly expanding and a large amount of iron is needed in order to maintain a near constant mean hemoglobin concentration of 12.5 g/dL. The children are highly vulnerable to iron deficiency during this period.

By four to six months of age, dietary iron absorption becomes important as iron stores are usually depleted. Low birth weight babies have less iron stores and hence, they need extra iron as well as iron at an early age from the diet source.

During the first 15 years of life, an average of 0.8 mg of iron needs to be absorbed each day. In addition, a small amount is also needed to balance normal losses of iron caused by shedding of cells. Therefore, approximately 1 mg of iron is needed daily in order to maintain positive iron balance in childhood. This can be achieved by ensuring a dietary intake of 8-10 mg of iron daily, since only 10% of dietary iron usually is absorbed.

When growth is most rapid, approximately 1 mg/L of iron in bovine and breast milk makes it difficult to maintain the body iron. Breast-fed infants have an advantage because they absorb iron 2-3 times more efficiently than infants fed with bovine milk.

Dietary iron absorption:

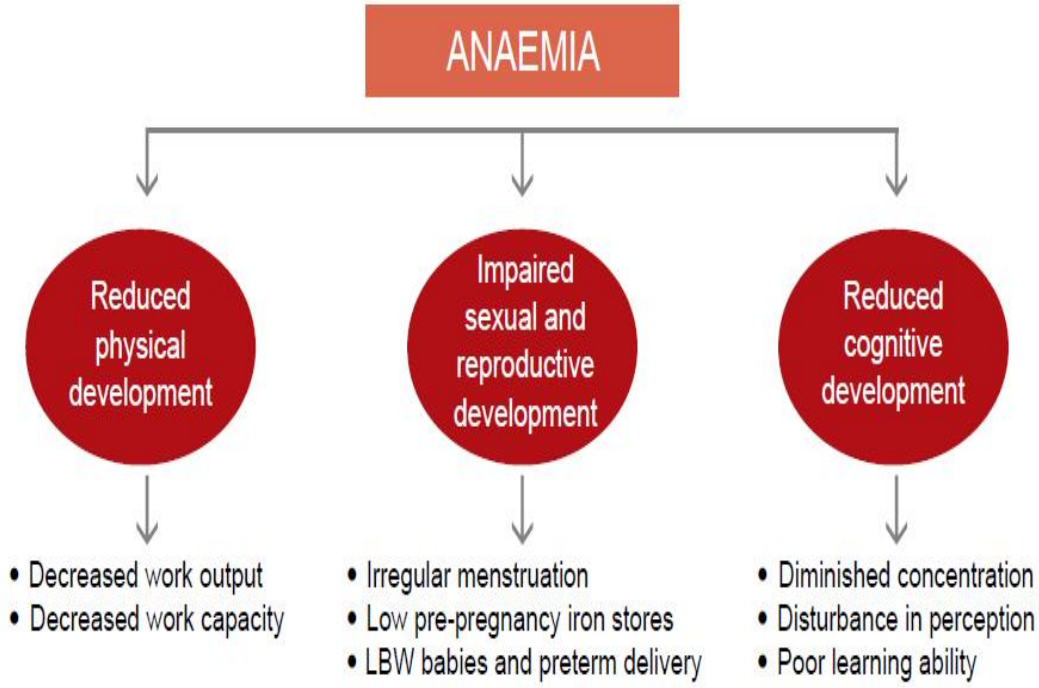
Haem iron absorption	☛ Iron status ☛ Haem iron in the diet • Fish, meat, poultry. • Constitutes less than 10% of the total intake in developing countries. • However, bioavailability is high. ☛ Calcium content in meal (e.g. milk, cheese) ☛ Method of food preparation (temperature, time)
Non-haem iron absorption	o Iron status o Dietary non haem iron • Cereals , pulses, vegetables, tubers • Bioavailability is low o Contamination iron • Soil, dust, water, iron pots

Table 3 Dietary iron absorption

Enhancers and inhibitors of iron absorption:

Enhancing factors	Ascorbic acid present in fruits, fruit juices, vegetables Fish, meat, chicken
Inhibiting factors	Phytates and other inositol phosphates - rice (unpolished), oats - breakfast cereals, - bread Calcium (e.g. milk, cheese) Iron binding phenolic compounds - tea, coffee, cocoa, certain spices, - certain vegetables - most red wines Nuts, peas, soybeans

Table 4 Enhancing and inhibiting factors[9]



Diag. 2 Adverse effects of anemia

Sequence of events in iron deficiency anemia

Depletion of iron stores:

This is the first event which occurs in a state of negative iron balance. Iron stores are depleted in order to enhance hemoglobin production. As a result, iron absorption is increased even when serum iron level is still normal and even before anemia develops. During this stage, the serum ferritin would have already fallen.

Iron-deficient erythropoiesis:

When the serum ferritin drops below 15ng/L, the serum transferrin saturation starts to fall to less than 15%. This occurs due to a fall in serum iron and a rise in transferrin concentration. At this stage, iron-deficient erythropoiesis occurs characterized by increasing concentrations of red cell protoporphyrin and serum transferrin receptors. But, the hemoglobin, mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) may still remain within the reference range, although the values may rise significantly after iron therapy is given.

Iron deficiency anemia:

Frank iron deficiency anemia develops when the negative balance continues. The red cells at this stage become obviously microcytic, hypochromic and poikilocytosis becomes more marked. The MCV and

MCH are also reduced. Target cells may be present in the peripheral smear. The reticulocyte count is also low for the degree of anemia. The serum iron falls and serum TIBC rises. As a result, the percentage saturation of the TIBC is usually less than 10%. This may be accompanied frequently by increase in platelet numbers.

Iron metabolism:

Iron is present in most living organisms and is critical for normal functioning by being part of electron transport in cytochromes and cytochrome oxygenase. Krebs cycle, in which heme containing iron acts as a coenzyme, is the active site of electron transport. Heme is also the site of oxygen uptake by myoglobin and hemoglobin. Circulating red cells in the human body has the highest concentration of iron, containing about 1 mg of iron per ml of packed cells. The storage form of iron is either ferritin or hemosiderin.[3]

At the same time, antioxidant mechanisms are needed by the body to protect against the damage by free iron in chemical reactions that generate free radicals such as singlet O_2 or OH.

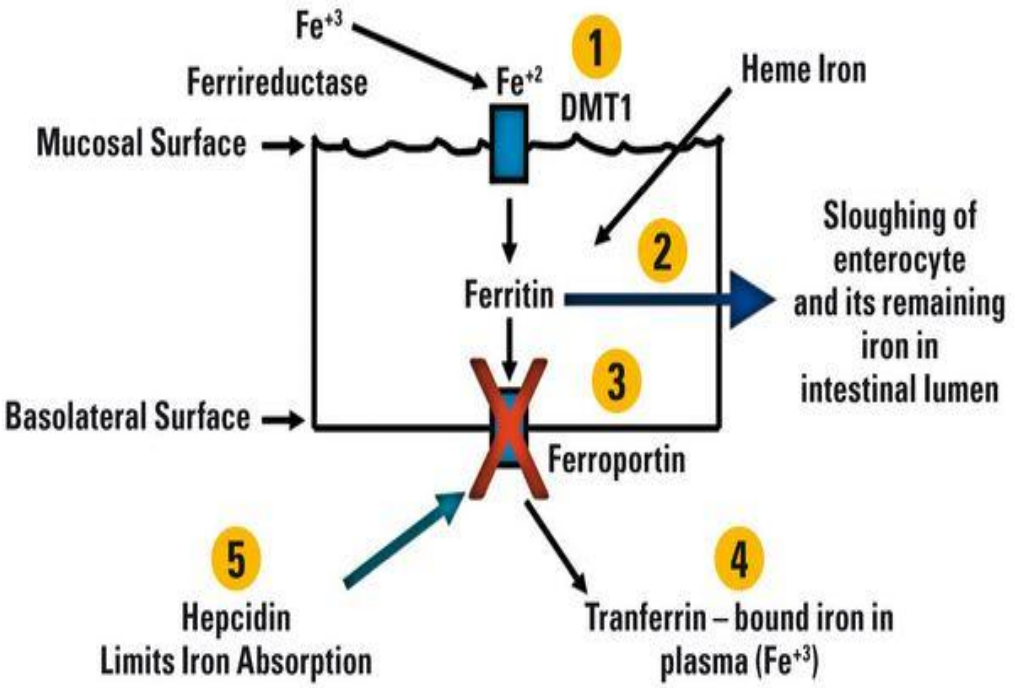
In a person with normal iron status, approximately 1 mg to 2 mg of iron (10% of the iron in a normal diet) is absorbed each day from the duodenal lumen into the intestinal mucosal cell (enterocyte), usually in the ferrous (Fe^{2+}) state. After iron is absorbed from the diet, it is either

transported by ferroportin into plasma or retained in the enterocytes as ferritin and shed later.

The main regulating mechanism of iron content in the body is iron absorption. The transport of iron from enterocyte to the plasma is tightly regulated by a peptide known as hepcidin. Hepcidin is produced in the liver and up regulated in states of inflammation. It is this process of intestinal iron absorption that serves as the primary mechanism of iron homeostasis.

When iron deficiency is present, hepcidin is decreased, thus fostering enhanced absorption of iron. In contrast, during states of iron excess or inflammation, hepcidin levels are usually increased, thereby blocking ferroportin and limiting its ability to mobilize iron into the plasma from enterocytes and storage sites.

Iron circulates in the plasma bound to transferrin, the iron transport protein until it interacts with specific transferrin receptors present on the surface of marrow erythroid cells. The iron-transferrin complex is internalized after the interaction via clathrin-coated pits. Once internalized, the complex is then transported to an acidic endosome, where low pH causes the iron to be released and made available for heme synthesis.



Diag.3 Iron absorption in the intestine

1. Iron is absorbed in the ferrous sate from the duodenal lumen into the enterocyte via divalent metal transporter 1 (DMT 1)

2. Iron is retained as ferritin and subsequently sloughed

3. Iron is transported across the basolateral membrane into the plasma via ferroportin

4. Iron is bound to transferrin and transported to marrow iron stores

5. During states of iron excess or inflammation, hepcidin increase and block ferroportin limiting the ability to mobilize iron into plasma

Iron is then incorporated into hemoglobin within the erythroid cell and subsequently enters the circulation. This form of iron will not become available for reutilization until the red cell dies. At the same time, excess iron is bound to a storage protein, apoferritin and forms ferritin. Hemosiderin is a protein aggregate formed within in the RE system when excess ferritin accumulates. Iron present in the hemosiderin is less readily available.

An average red cell life span is 120 days in a normal individual, which means about 0.8 to 1% red cells turnover each day. Once the red cell reaches the end of its life span, it undergoes phagocytosis after being recognized as senescent by reticulo-endothelial cells. Once the hemoglobin from the ingested red cell is broken down, the iron is shuttled back to the surface of the RE cell and presented to circulating transferrin.

1.5. Megaloblastic anemia:

Megaloblastic anemia is the most common manifestation of deficiency of vitamin B12 and folic acid. Vitamin B12 and folic acid deficiency primarily results in decreased capacity of DNA synthesis and manifests as reduction in number as well as enlargement of all rapidly proliferating cells of the body, including marrow cells. The commonest age is 3 to 8 months with maximum number of cases in 9 to 12 months.[10]

Animal products such as eggs, meat, milk and other dairy products are the main source of cobalamins. Intrinsic factor, a protein present in the gastric juice, is required for absorption. Once bound to intrinsic factor, the cobalamins are subsequently absorbed in the ileum and are taken up from circulation and stored in the liver. They are then released into circulation as and when needed. Vitamin B12 acts as a coenzyme in two important metabolic functions which are vital to DNA synthesis and normal cell growth. The two vital chemical reactions are concerned with methionine synthesis and formation of succinyl CoA from methylmalonyl CoA.

The most common cause of vitamin B12 deficiency is defective secretion of intrinsic factor which results in inadequate vitamin B12 absorption. This condition is known as pernicious anemia and is most common among people aged over 50 years. Other causes are malabsorption due to surgical resections, gastrectomy and bacterial or inflammatory diseases affecting the small intestine. There has been no known incidence of clinical problems caused by elevation in vitamin B12 levels.

Folic acid is present in foods such as dark leafy vegetables, milk, egg, yeast, beans and citrus fruits. It is also absorbed in the small intestine and stored in the liver. Folate deficiency can be caused by decreased

dietary intake, malabsorption or excessive utilization as in pregnancy, liver-damaging diseases. Folate deficiency is characterized by megaloblastic anemia and ultimately, severe neurological problems[14].

Some peculiar features to Megaloblastic anemia include hyperpigmentation of knuckles, terminal phalanges and mild enlargement of liver and spleen. Other less common manifestations include neurodevelopmental effects and abnormal movements.

1.6. Clinical features:

Iron deficiency anemia is asymptomatic in most cases. Pallor is the most common manifestation and is identified in children over palmar creases, palms, nail beds or conjunctivae. Pallor is associated with fall in hemoglobin levels to 7 to 8 g/dL. When the hemoglobin decreases further, child displays mild irritability.[8]

When hemoglobin decreases further to 5 g/dL, the children complain of headache, fatigue, dizziness, shortness of breath, palpitation, chest pain and manifest irritability, lethargy and anorexia. Hemic murmurs may be heard on auscultation. This stage is associated with significant physical, psychological and social consequences. Tachycardia and high output cardiac failure occurs, when hemoglobin continues to fall. .

Other features include pica, pagophagia (the desire to eat ice cubes) and plumbism, poor school performance, leg cramps, reduced resistance to infections. Children who are iron deficient suffer from growth impairment and tend to be shorter than non-iron deficient children. In early iron deficiency, even before iron deficiency anemia develops, the most important non hematological side effect is intellectual impairment, cognitive function and motor function.[11]These changes are not reversible with iron therapy and hence prevention is better than cure. Physical examination may reveal pallor, spoon shaped nails (koilonychia), leuconychia, platynychia, splenomegaly in severe and persistent untreated anemia.

1.7. Investigations

Investigations for iron deficiency anemia includes complete blood count, iron profile, peripheral smear and stool for occult blood and serum B12 and folic acid to look for other nutritional anemias.

Complete blood count:

Most clinical laboratories now employ automated machines to perform complete blood count to estimate hemoglobin, hematocrit (packed cell volume), and red blood cell count. MCV, MCH and MCHC are calculated values from hemoglobin, hematocrit, and red blood cell count.

Red cell indices are used in the morphologic classification of anemias based on the size of the red blood cell as being normocytic (normal MCV), macrocytic (increased MCV), or microcytic (decreased MCV). . Red cell indices were first introduced by Wintrobe in 1929.

$$\text{MCV} = \frac{\text{Volume of packed cells / 1000 mL of blood}}{\text{Red blood cell count (millions / mL)}} \quad \text{fL or } \mu m^3$$

$$\text{MCH} = \frac{\text{Hemoglobin in g / 1000 mL of blood}}{\text{RBC count (millions /mL)}} \quad \text{pg/cell}$$

$$\text{MCHC} = \frac{\text{Hemoglobin in g / 100 mL of blood X 100}}{\text{Volume of packed cells /100 mL of blood}} \quad \text{g/dL or \%}$$

MCV defines the size of the red blood cells and is expressed as femtoliters (10^{-15}; fL) or as cubic microns (μm^3). MCH quantifies the amount of hemoglobin per red blood cell. MCHC indicates the amount of hemoglobin per unit volume. In contrast to MCH, MCHC correlates the hemoglobin content with the volume of the cell. It is expressed as g/dl of red blood cells or as a percentage value.

Microcytic anemias were also often described as being hypochromic based on peripheral smear examination and MCHC when this value was determined manually.

Red cell distribution width (RDW), otherwise known as red cell morphology index represents the coefficient of variation in the volume distribution (size) of the red blood cell and is expressed as a percentage, thereby quantifies the variation in the size of red cells (anisocytosis).

Spurious results associated with Automated Cell Counters.

- In the case of red cell agglutination, larger clumps are not counted as red blood cells at all and doublet erythrocytes are counted as one. This leads to a "decrease" in red cell count and a falsely elevated MCV. Pre-warming the sample eliminates these spurious values.

- In hyperglycemic conditions, red cells are swollen as they are transiently hypertonic in relation to the isotonic diluting fluid resulting in elevated MCV. Providing some time to allow for equilibration after dilution can avoid this spurious result.

- Conditions such as hyperlipidemia, hyperbilirubinemia, a very high white blood cell count and high serum protein can interfere with absorption characteristics of hemoglobin. Since hemoglobin is quantified based on its absorption characteristics, this can result in falsely elevated hemoglobin values.

- Since MCH and MCHC are calculated and are not directly measured, these indices also become abnormal when the values of hemoglobin, red cell count, and MCV are affected.

The reference values of red cell indices and iron profile are given in Table 2 shown below:[8]

Parameters	**Reference range**
RBC count	6m to 2 years: 3.7 to 5.3 million/cu.mm 2 to 6 years: 4.9 to 5.3 million/cu.mm 6 to 12 years: 4 to 5.2 million/cu.mm
PCV	1 to 23 months: 32 -42 % 2 to 9 years : 33 -43% 10 to 17 years : 36 -45%
MCV	1 to 23 months: 72 to 88 fL 2 to 9 years : 76 to 90 fL 10 to 17 years : 78 to 95 fL
MCH	1 to 23 months: 24 – 30 pg 2 to 9 years : 25 – 31 pg 10 to 17 years : 26 – 32 pg
MCHC	32.0 – 36.0 %
RDW	10.0 – 15.0 %
Platelet count	154000 – 400000 cells/cu.mm
Reticulocyte count	<1%

Serum Iron	50.00 – 120.00 µg/dL
TIBC	250.00 – 400 µmol/L
Transferrin saturation	15 - 55%
Serum Transferrin	208 – 400 mg/dL
Serum Ferritin	6.0 – 67.0 µg/L
Serum B12	140.00 – 700.00 pg/mL
Serum Folic acid	1.8-9.0 ng/mL
ESR	5-15 mm/Hr
CRP	0.01-2.80mg/L

Table 5 Red cell indices and iron profile

Erythrocyte sedimentation rate

The rate at which red blood cells sediment in a period of one hour is called erythrocyte sedimentation rate. ESR is a non-specific indicator of inflammation. The high proportion of fibrinogen present during an inflammatory process results in red blood cells sticking to each other. This results in rouleaux formation and red blood cells settle faster.

Peripheral smear examination:

A blood smear is used to categorize the type of anemia based on the size, shape and color of RBC (indicators of hemoglobin content). It also helps in identifying conditions that affect one or more type of blood cells. Examples include anemia, myeloproliferative disorders, leukemia, and parasitic infections. If the result from an automated cell count or a differential count indicates the presence of abnormal WBC, RBC and/or platelets, it has to be verified and confirmed by a good peripheral smear examination, making it the most important investigation of hematology. The reticulocyte count is calculated manually and it is corrected for appropriate hemoglobin levels.

Diagnostic methods for investigating iron metabolism[12]

Serum iron and iron binding capacity:

The serum iron level indicates the amount of circulating iron bound to transferrin. The iron supply to the tissues is measured by serum iron and the saturation of the total iron-binding capacity of transferrin (TIBC). The serum iron shows a diurnal rhythm in normal subjects, the values being lower in the morning than in the evening. The TIBC is an indirect measure of the circulating transferrin. Serum transferrin is determined by adding Serum iron with TIBC. Transferrin saturation levels below 20%

are associated iron deficiency states. Transferrin saturation is obtained by the following formula:

$$\frac{\text{Serum iron}}{\text{Serum transferrin}} \times 100$$

Serum Ferritin:

Serum ferritin is a non-invasive measure of iron stores and the most convenient laboratory test to estimate iron stores as the levels correlate with total body iron stores. In neonates, over the first 2 months of life, the fetal hemoglobin is broken down and cord blood concentration (median approximately 100µg/L) rises further. Thereafter, throughout childhood and adolescence, serum ferritin levels fall to low levels (median 20–30 µg/L). Until the onset of puberty, there is no gender difference in ferritin values. Once puberty is attained, serum ferritin levels rise in males. The normal serum ferritin value varies according to the age and gender of the individual. In children less than 5 years, serum ferritin levels less than 12 µg/L and in children more than 5 years, levels less than 15µg/L is considered as depleted iron stores.[13]

Serum ferritin values falls to <15 µg/L as iron stores are depleted. Serum ferritin values below 15µg/L are virtually specific for storage iron depletion. However, normal values do not exclude storage iron depletion and values above 300µg/L do not necessarily indicate iron overload.

Confounding factors influencing Serum ferritin:

Serum ferritin is an acute-phase reactant and is increased in conditions like infection, inflammation or malignancy. Serum ferritin is released from the tissues when iron-rich organs are damaged as in hepatic necrosis, chronic liver disease and bone marrow or splenic infarction in sickle cell disease.

Serum transferrin receptors:

Serum transferrin receptor concentration reflects both iron supply to the bone marrow and the number of erythroid precursors. It is an important and invaluable indicator of iron deficiency in anemia of chronic disease.

Serum folic acid levels:

Folate status is assessed by measuring folate levels in both serum and red blood cells. Red blood cell (RBC) folate is the best indicator of long term folate stores whereas serum folate level is an indicator of recent folate intake. Prolonged folate deficiency is indicated by low RBC folate values. Folate uptake into red blood cells is also affected vitamin B12 deficiency, leading to low RBC folate values even after adequate folate intake.[14]

Serum vitamin B12 levels:

A deficiency of either vitamin B12 or folic acid disrupts the synthesis of methionine as both are linked to the reaction pathway. This leads to the same symptoms and medical problems. Therefore, in a clinical workup, measurement of both vitamins is often necessary and treatment varies depending on which vitamin is deficient.

C-reactive protein:

C-reactive protein is an acute phase reactant and is increased in processes such as infections, inflammatory diseases and malignant neoplasms. Clinical symptoms, including fever, are frequently preceded by the CRP response. Normal CRP values in healthy individuals have a range up to 5 mg/an L. Serum CRP value rise rapidly and extensively after onset of an acute phase response and are detectable within 6 to 8 hours and reaches the peak within 24 to 48 hours. CRP activates the classical complement pathway. CRP is an ideal tool for clinical monitoring as it has a half-life of only a few hours. Whenever high serum CRP concentration persists in a clinical condition, it generally indicates the presence of an uncontrolled infection.

1.8. Treatment of iron deficiency anemia

The objectives of therapy in IDA are

i) To restore the hemoglobin to normal

ii) Replenish the depleted iron stores

iii) Treat etiological factors and prevent their recurrence.

Severe iron deficiency anemia can be rapidly treated with the help of iron supplements in all sex and age groups. Whether iron supplementation can be given daily or twice or once per week without compromising the efficacy of supplementation is an ongoing debate and numerous studies have been conducted. However, daily supplementation for young children remains the current recommendation.

Oral iron supplements should be started as soon as a diagnosis of IDA is established. The standard dose is 3 mg/kg of elemental iron per day up to a maximum of 180 mg/day. Higher doses (6mg/kg body weight) were previously used; but were associated with more frequent gastrointestinal side effects. Single daily doses are effective, but 2-3 divided doses are better tolerated by children. Ideally, oral iron should be given at least half an hour before a meal to ensure maximal absorption. It can also be given 1-2 hours after meals.[1,15,16,17]

Oral iron therapy must continue for a period of at least three months to replenish depleted iron stores, if an adequate response to therapy is seen. Stoppage of iron, once anemia is corrected, is the most frequent cause for recurrence of IDA.

In 12 to 24 hours of starting therapy, there is replacement of intracellular iron enzymes leading to dramatic improvement in sense of wellbeing, decreased irritability and increased appetite. In 36 to 48 hours, the bone marrow responds by way of erythroid hyperplasia. An increase in reticulocyte count is observed in 48-72 hours and peaks in 5-7 days. The hemoglobin level rises at an average rate of 0.15 g/dL per day, usually commencing about one week after the institution of therapy. Repletion of iron stores occurs by 1 to 3 months.

Choice of iron preparations:

Various salts have been used. Ferrous sulphate is used most frequently, is least expensive and causes few side effects, if administered in appropriate doses. Ferrous sulphate tablets are available as pediatric iron tablets of 66 mg with an elemental iron content of 20 mg. Ferric hydroxide polymaltose complex was recently introduced salt in the market and has fewer gastrointestinal side effects. The non-ionic iron polymaltose, has been shown to have no oxidative potency on

lipoproteins in healthy subjects. Hence, a better compliance is expected than with ferrous sulphate. But, the data on efficacy are lacking.[18,19]

Activated hydroxyl radicals are released when ferrous compounds are oxidized in the lumen of the gut or within the gut mucosa. These molecules attack the gut wall resulting in a range of gastrointestinal symptoms and discomfort. In an attempt to avoid these problems, enteric coated ferrous formulations designed to minimize iron release in the stomach are available. But, this may compromise the absorption of iron. Therefore, enteric-coated tablets should not be used as maximum absorption of iron occurs in the duodenum and proximal jejunum.

Nausea, vomiting, abdominal discomfort, diarrhea and constipation occur in some patients. These side effects are dose related and can be minimized by adhering to standard doses, gradual hiking up of dose and administration of iron with or after meals.

Oral iron preparations:

Iron preparation	Percentage of elemental iron
Ferrous sulfate	20
Exsiccated ferrous sulfate	30
Ferrous fumarate	33
Ferrous succinate	23
Ferric ammonium citrate	18

Table 6 oral iron preparations[20]

Commercial iron preparation:

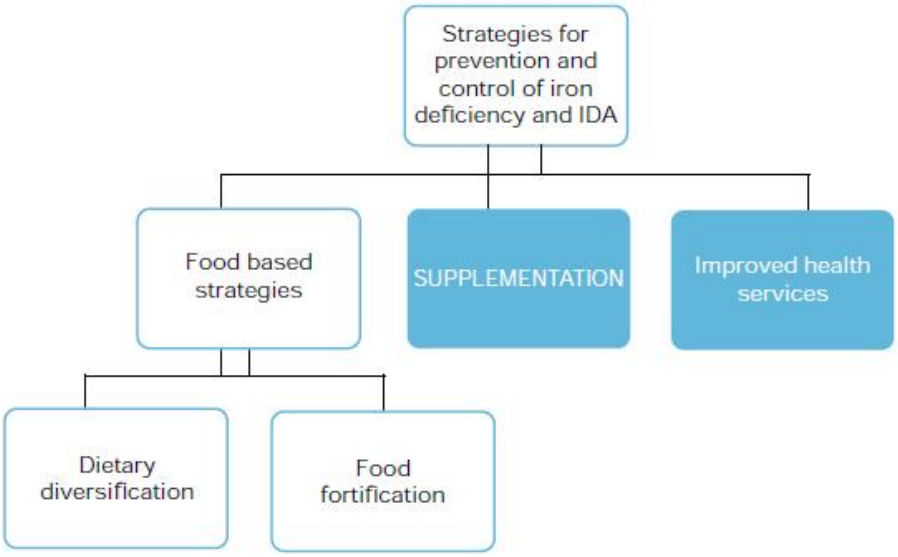
Iron preparation	Elemental iron (per tab/cap or per 5 ml)
Fersolate Tab	66 mg
Tonoferon pediatric syrup	80 mg
Vitcofol syrup, Hemsi syrup, Fesovit syrup	33 mg
Mumfer, Trifer	50 mg

Table 7 commercial oral iron preparations

Helminthic control:

Albendazole is given as a single dose (400 mg) in children more than 2 years and as a single dose of 200 mg in children less than 2 years. Other drugs used are single dose of Mebendazole (500 mg), single dose of Levamisole (2.5 mg/kg) or single dose of Pyrantel (10 mg/kg).

1.9. Prevention



Diag.4. Methods of prevention and correction of iron deficiency and IDA

Food-based Interventions:

Dietary diversification[1]

Improvement of nutritional status can be achieved by educating households on methods of food preparation, including minimizing the amount of inhibitors of iron absorption in the diet and recommending minimal cooking in small amount of water of vegetables rich in vitamin C, folate and other water soluble vitamins. Also, the bioavailability of iron is improved by adding a small portion of haem iron containing diets like meat or fish, since iron content is low in cereals and tuber based diets.

Food fortification

Food fortification is being done in many countries as a part of prevention of anemia. The best method is to fortify a staple food of the local population, since it will be consumed in significant quantities. Successful implementation of iron fortification of wheat flour has been achieved in several countries in the North America, Great Britain, South America, and Caribbean.[15]

Another method is to fortify a widely consumed condiment such as salt and sugar which have all been successfully fortified with iron. Both dried and liquid milk and dairy products such as yogurt have been fortified with iron in South America.

In the control of iron deficiency anemia in children receiving complementary foods, fortified infant foods are an especially important component. This method has been shown to be effective in preventing infant iron deficiency anemia in Latin America and United States.

In our country, this can be made possible by taking advantage of existing technology and health care network.

National health programs:

IFA supplementation for children has been recommended by the Ministry of health and family welfare. As part of this program, 20 mg elemental iron and 100µg folic acid per mL of liquid formulation for children aged 0 to 5 years and 30 mg elemental iron and 250 µg per day in children aged 6 to 10 years, both for a period 100 days with age appropriate deworming is recommended.

In the year 2013, a policy decision to develop the National Iron+ Initiative was taken by the Ministry of Health and Family Welfare. Existing national programs were brought together under this initiative and new age groups were introduced as given below.[1]

- Preschool children aged 6 months to 5 years are given iron supplementation of 20 mg elemental iron and 100 µg folic acid bi-weekly.

- Children from 1st to 5th grade (5 to 10 years of age) in Govt. & Govt. Aided schools are given weekly iron supplements containing 45 mg of elemental iron and 400 µg of folic acid by school teachers. Out of school children are given iron supplements at Anganwadi Centers.

- In the case of adolescents (10–19 years), weekly iron supplementation is given containing 100 mg elemental iron and 500 µg of folic acid through school teachers.

Review of Literature

REVIEW OF LITERATURE

2.1. Articles related to prevalence:

In the year 2011, Sudhagandhi et al. conducted a study among school children aged 8 to 16 years of Kattankulathur, Tamil Nadu to know the prevalence of anemia. A total of 900 children were included in this study and it was found that 52.88% were anemic and the prevalence of anemia among girls (67.77%) was significantly higher when compared to the boys (35.55%) and anemic children were underweight.[21]

Rayn et al., conducted a cross-sectional study in two rural districts of Karnataka, in the year 2010 among children aged 12 to 23 months. Out of the 401 children included in the study, anemia was detected in 75.3% children. It was also found that low ferritin levels, maternal anemia and food insecurity were associated with anemia. In this study, correlation statistics revealed a direct association between children's ferritin levels and their iron intake, CRP levels and the maternal hemoglobin levels. An inverse association was also demonstrated between children’s ferritin levels and history of continued breastfeeding and the child's energy intake.[22]

A prevalence study was conducted by Sabitha basu among school going adolescents girls of Chandigarh who were apparently healthy. Results of the study revealed that anemia is more common among rural

adolescents (25.4%) when compared to urban (14.2%) adolescents. Also, 81.7% and 41.6% of the adolescent girls and boys respectively had deficiency in Serum ferritin levels reflecting reduced iron stores.[23]

In the year 2010, a nationwide survey was done by Chellan et al., to find the prevalence of anemia and to assess the influences of socioeconomic and demographic factors on the degree of anemia. The prevalence of anemia was high not only in the low socioeconomic group, but also in the higher strata. But the percentage of severely anemic children declined with mother's educational level and standard of living.[24]

Gomber et al., in Indian Journal of Medicine in October 2003 conducted a study in corporation schools among children of age 5 to 11 years from an urban slum and it was found that prevalence of anemia as per WHO recommended cut-off values of hemoglobin was 41.8 per cent. The commonest type of anemia noted was pure or mixed iron deficiency anemia in 68.42% children followed by pure or mixed B12 deficiency noticed in 28.42% children. Iron deficiency was the commonest cause among the pure variety occurring in 41.05 % (39 of 95) children.[25]

Duque et al. conducted a study among Mexican children aged less than 2 years in rural as well as in urban areas to determine the prevalence of anemia as well as deficiencies of iron, folic acid and zinc. In this study, anemia was found in 20% of children. About 27.8% rural children and

32.6% urban children in the study had iron deficiency. Low serum zinc was noted in 28% urban and 13% rural children included in the study. Similarly, folic acid deficiency was noted in 10% children in the urban areas, and 8% children in rural areas. Thus, the principal micronutrient deficiencies noted among Mexican children in the age group of less than 2 years was iron and zinc deficiencies. More important was the fact that low ferritin concentrations were not seen in more than 50% of anemia.[26]

In January 2011, a study was done to determine the prevalence of iron deficiency anemia by Al-Sayes et al. among apparently healthy young female university students in Saudi and it was found that 50.2% of students were normal and 25.9% of students had deficient iron store and 23.9% of students had iron deficiency anemia. There was a significant correlation between iron deficiency and iron deficiency anemia with inadequate meat intake and impaired exercise capacity.[27]

2.2. Articles related to clinical profile:

The study by Jain et al. among school girls of age 8 to 11 years in 2012 regarding correlation between hematological and cognitive profile in anemia showed that among 111 children in the sample, 77% were anemic with low iron studies ,lower IQ, intellectual capacity and educational achievement than those of their non-anemic counterparts. These three parameters had significant correlation ($p<0.05$) with Hb and serum iron.[28]

Lukowski et al. found that frontostriatal-mediated executive functions like inhibitory control, set-shifting and planning were performed less well by infants with chronic, severe iron deficiency. The hippocampus-based recognition memory task was also impaired. These deficits suggest that early iron deficiency in the long-term may affect the hippocampus, dopamine system as well as their interaction.[29]

Peirano et al. conducted a study which showed affective differences among infants with iron deficiency anemia. Also, infants with iron deficiency anemia tested lower in mental and motor developmental assessments. It was found that even after iron therapy, they had persistent sleep disturbances and neurofunctional affects which lead to reduced behavioral and cognitive outcomes indicating that iron may have key role in the normal development and progression of several neurofunctional systems.[30]

Fretham et al. published an article regarding the role of iron in at least 3 major neurobehavioral domains in learning and memory, including learning, speed of processing and affect. Learning and memory, being particularly prominent, were affected by early life iron deficiency and were found to persist despite iron repletion. In the case of adults, interactions between direct and indirect effects of iron deficiency contributing to abnormal hippocampal structure and plasticity is the likely cause of learning deficits.[31]

In the year 2014, the relationship between pagophagia (ice pica) and iron deficiency anemia was studied by Uchida et al. Out of 81 patients with iron deficiency anemia, pagophagia was present in 13 patients (16.0%), and it was found that oral iron therapy can cure the pagophagia earlier than hemoglobin recovery and repair of tissue iron deficiency.[32]

2.3. Articles related to investigations:

Sazawal et al. conducted a study among 2091 children in the age group of 1–3 years from an urban low socio-economic population of Delhi to determine the role of RDW in discriminating IDA and published an article in the year 2014. The study found that in children with hemoglobin values ≤10.0 g/dL, RDW values greater than >15% identifies iron deficient anemia without the need for iron status.[33]

In the year 2009, Aulakh et al. of Christian medical college, Ludhiana conducted a study among 151 children aged 6 months-12 years with microcytic (MCV<75 fL) anemia to determine the usefulness of Red Cell Distribution Width (RDW) for the diagnosis of iron deficiency. On the basis of serum ferritin and total iron binding capacity (TIBC), Children with microcytic hypochromic anemia were classified into iron deficient and non-iron deficient anemia. It was concluded that the RDW had sensitivity and specificity of 81.0% and 53.4% respectively, in the

diagnosis of IDA. Similarly, the positive and negative predictive value of RDW in the diagnosis of IDA was 63.0% and 72.2% respectively.[34]

Lee et al., in 2001 investigated hematologic profiles of anemic children aged 5 to 36 months (198 patients) comparing them with the control group. The children were mainly brought to the clinic for infectious or inflammatory illness. Only 13.1% children with iron deficiency anemia had come directly for evaluation of pallor. Among the children with IDA, the correlation between hemoglobin and MCV was much more than the control group ($P < 0.001$). It was concluded in the study that the accuracy of diagnosis of IDA can be increased by combining hemoglobin, MCV, RDW as well as iron profile in screening for iron deficiency.35

In the year 2014, the HELENA study conducted among adolescent girls in 10 European cities to evaluate the usefulness of iron profile confirmed that serum ferritin and serum transferrin receptors can be used in assessing the iron status of adolescents and it was also found that adolescent girls are at high risk of iron deficiency.[36]

2.4. Articles related to various iron therapies:

Aggarwal et al., in 2004 conducted a study among predominantly breast fed term low birth weight (LBW) infants aged 50–80 days to evaluate the hematological effects of iron supplementation. Total 73

infants enrolled were randomized into two groups. One group received iron (3 mg/kg/day) and the other group received placebo drops. Baseline anthropometry and hematological parameters were measured. These measurements were repeated after four and eight weeks and no significant differences were found in serum ferritin and anthropometry. However, there was a significant positive change in hemoglobin levels in the iron supplementation group.[37]

In 2004, a review article published in Indian Pediatrics regarding iron supplementation recommended oral iron for treating iron deficiency anemia. According to the article, the type of iron salt to be used has to be based on the bioavailability of the iron salt, side effects and cost effectiveness.[38]

Ferrous salts, specifically ferrous sulfate, are preferred for treatment of IDA, since all iron needs to be reduced to ferrous form for absorption. As ferrous sulfate is not stable in liquid form, other ferrous salts are used in liquid formulations. One important side effect of ferrous sulfate is gastrointestinal intolerance. Various measures have been suggested to overcome this side effect. These measures include administration after meals and at bed time, since decreased intestinal motility during sleep may help improve absorption.

In the case of ferric salts, ferric ammonium citrate is used. The important drawback of these salts is the low bioavailability which is considered to be 3-4 times less than ferrous salts.

Better bioavailability is claimed with iron-amino acid chelators, which are other group of iron preparations, because absorption of this salt is said to be not interfered by phytates in the diet. However, when compared to ferrous sulfate in clinical studies in young children and infants, equal rise in hemoglobin has been reported.

Iron polymaltose complex (I-PMC) and carbonyl iron are two more types of iron preparations that deserve mention. The bioavailability of iron polymaltose complex (I-PMC) has been demonstrated to be similar to ferrous sulfate. Also, both ferrous sulfate and I-PMC in equivalent doses were found to have similar efficacy in a recent meta-analysis on iron polymaltose complex use in adults with IDA.

In the case of carbonyl iron, the main advantages are the small particle size of the molecule contributing to increased bioavailability as well as the considerably higher doses of administration. However, this fact needs further research as the result of two Indian studies are conflicting, one stating that the bioavailability to be higher and another study finding the bioavailability to be similar. Carbonyl iron has been mainly used in food fortification industry.

Conventionally, a dose of 4-6 mg/kg/day of elemental iron were recommended for treatment of IDA in children. However, it was found that smaller doses were also equally effective as well as better tolerated. Hence, currently a dose of 3 mg/kg/day of elemental iron is recommended. It is to be noted that approximately 25 mg of elemental iron saturates the absorptive capacity of iron in the duodenum. Therefore, absorption of iron will not be increased at higher doses.

Low et al. in a meta-analysis of randomized controlled trials on the effects of daily iron supplementation in 5-12 years old primary school children identified 16501 studies in which a total of 7089 children were evaluated. A total of 31 studies had been conducted in low or middle income settings. The review concluded that there was improvement in intelligence quotient ($p = 0.04$), global cognitive scores ($p = 0.01$) as well as measures of attention and concentration among anemic children on iron supplementation.[39]

An improvement in age-adjusted height and weight among anemic children on iron supplementation was also noted. According to this review, the risk of iron deficiency and iron deficiency anemia were 50% and 79% respectively by iron supplementation.

Desai et al. conducted a study in Western Kenya among children less than 2 years living in a malaria-endemic area, comparing the efficacy of supervised as well as unsupervised, daily and twice weekly iron

supplementation over a period of 6 weeks in the treatment of mild and moderate anemia after administering a single dose of sulfadoxine-pyrimethamine at enrollment. Daily supervised or unsupervised iron supplementation was given in the dose of 3–6 mg/kg/day and twice weekly supervised or unsupervised iron supplementation was given in the dose of 6–12 mg/kg/wk.[40]

Results from the study revealed that hemoglobin concentration in the daily supervised iron supplementation group was significantly higher than twice weekly supervised iron supplementation group. In case of unsupervised iron supplementation groups, hemoglobin concentrations were significantly higher at 12 weeks. But, the hemoglobin concentrations were not different at 6 weeks of iron supplementation.

It was concluded that, in the treatment of anemia in preschool children, regardless of compliance to therapy, hematological responses can be expected after 6 weeks of daily iron supplementation rather than twice weekly iron supplementation.

2.5. Articles related to treatment and improvement in hemoglobin:

Maheswari et al. conducted a study among a total of hundred anemic school children from three government schools of Raipur city. They were subjected to anthropometry, iron status and clinical examination before they underwent iron therapy and the response to iron

was assessed by estimation of hemoglobin after one month following iron therapy. In all the 100 children, clinical pallor was the most common finding (100%), followed by fatigue (54%), weakness (38%), anorexia (18%) and icterus (5%). The commonest blood picture was that of microcytic hypochromic.[41]

Age dependent MCV, MCH, MCHC and RDW showed that the maximum number of cases had below normal MCV, MCH and MCHC values. RDW values were increased in 74% cases. 82% cases had serum iron values below normal (<50μg/dl) and 91% cases had TIBC values above the normal range. The serum ferritin levels in 35% children was <15ng/ml (in 18% boys and in 17% girls). The mean and SD of Hb before therapy was 8.60 ± 2.05 and after therapy, it was 9.55±1.88 respectively. A significant increase in the hemoglobin ($p<0.009$) level was observed in both boys and girls after 30 days of iron supplementation.

A systematic review of randomized controlled trials in 2007 showed the effect of iron supplementation on hemoglobin response in children by Gera et al. He found that there is significant, but modest increase hemoglobin levels after iron supplementation. The review also concluded that children who were anemic at the beginning of the trial showed a greater increase in hemoglobin, whereas improvement in

hemoglobin was much lower in those children living in malarial hyper-endemic areas and in those consuming iron-fortified food.[42]

In the year 2010, Huang et al. in Taiwan conducted a study to determine the various causes of iron deficiency anemia and the treatment outcomes. It was found that the most common cause in children less than 2 years was inadequate intake and blood loss was common in children aged 2 to 10 years. Treatment with iron supplementation for 3 months resulted in improvement of hemoglobin levels in 78.9% of IDA patients.[43]

Paracha et al. conducted a study to determine the effects of iron supplementation on iron status in selected pre-adolescent school girls aged 8 to 11 years in North West Frontier Province, Pakistan by dividing them into treatment and control groups. The treatment group received 76 mg elemental iron per day over a period of 11 weeks. At the same time, both the groups received multivitamin tablets daily. It found that the 35% of the pre-adolescent schoolgirls had IDA. The improvement in hemoglobin (15g/dL), Hct (3%) and serum ferritin (20ng/mL) was significant ($P<0.05$) in the anemic treatment group when compared to the control group.[44]

STUDY JUSTIFICATION

3.1. Study justification:

Iron deficiency anemia is most prevalent among nutritional anemia and it constitutes more than 70%. Though there are many studies on prevalence in rural and in varied populations and in schools, the present study is done to assess the prevalence and profile of iron deficiency anemia among the children of working group mothers. Though children attending the ESI HOSPITAL belong to parents who are educated and are aware of nutritional importance of minerals, most of them are not able to take care of their children because of the work and hence my study is done to assess the clinical profile in this particular population, and also to test the correlation of various red cell indices with iron studies. Though there are lot of studies on iron therapies and comparison of various formulations there are not many studies on improvement in hemoglobin or follow up studies after oral iron supplementation. Hence, this study is intended to follow up children with iron deficiency anemia to know the improvement in hemoglobin.

Aim and Objectives

AIMS AND OBJECTIVES

4.0. Aim of the study:

The aim is to study the profile of nutritional anemia in children and to determine the treatment outcome of iron deficiency anemia.

4.1. Objectives:

- To assess the proportion of various types of anemia like microcytic hypochromic, normocytic hypochromic, normocytic normochromic or megaloblastic anemia.
- To analyze the various presenting symptoms in relation to anemia
- To assess the correlation of Red cell indices and peripheral smear with iron profile, folic acid and vitamin B12.

4.2. Secondary objective:

- To determine the treatment outcome of iron deficiency anemia in children after oral iron supplementation for 12 weeks.

Material and Methods

MATERIAL AND METHODS

5. Material and methods:

Study design:

Cross-sectional study and interventional study

Place of study:

ESIC Medical College & PGIMSR, KK Nagar, Chennai

Period of study:

9 months

Sample size:

150 children

5.1. Inclusion criteria:

Children in the age group of 1 to 10 years with hemoglobin levels less than the cut-off values for the age, as per WHO guidelines, were included in the study. Out of the 150 children, those with iron deficiency anemia were followed up for treatment outcome after iron therapy.

5.2. Exclusion criteria:

- Chronic diarrhea
- Children with renal disease
- Children with cyanotic heart disease
- Children with endocrine diseases

5.3. Methods:

150 children in the age group of 1 to 10 years with pallor were confirmed to have anemia by hemoglobin estimation.[45, 46] The criteria for diagnosing anemia was hemoglobin values less than 11 g/dL in children aged 12 months to 59 months, less than 11.5 g/dL in children aged 5 years to 11 years. Children who fulfilled the criteria were included in the study after obtaining informed written consent from their parents.

<table>
<tr><td>• Severe palmar pallor</td><td>SEVEREANAEMIA</td></tr>
<tr><td>• Some palmar pallor</td><td rowspan="2">ANAEMIA</td></tr>
<tr><td>• No palmar pallor</td></tr>
<tr><td></td><td>NO ANAEMIA</td></tr>
</table>

Fig. 2 IMNCI guidelines for assessment of pallor[45]

The history and examination was documented on a pre-designed proforma. A social demographic profile including parents' education, family structure and diet consumed (vegetarian, non-vegetarian) was noted. A detailed general physical examination was done to look for pallor, icterus, edema, lymphadenopathy, and signs of vitamin deficiency. The anthropometric measurements (weight, height) were made by a single observer and finally, a thorough systemic examination was also done.

The following investigations were done in all cases – complete blood count, red cell indices, peripheral smear study, reticulocyte count, RDW, ESR, CRP, stool for occult blood, serum ferritin, iron indices, vitamin B12 and folic acid assay.

Children with iron deficiency anemia were given oral iron syrup (iron polymaltose complex) in the dose of 3 mg/kg/day once daily, for 3 months and hemoglobin levels were checked at 1 and 3 months after starting iron supplementation. Reticulocyte count was also checked after one month of therapy.

Blood sampling:

Blood samples were collected under strict aseptic precautions. About 2 mL of blood was collected in EDTA tube for complete blood count and peripheral smear examination. Around 3 mL of blood was collected in 2 separate serum containers. One container was used for spectrophotometric assay of serum iron and TIBC and the other container for assessment of serum Ferritin, Vitamin B12 and folic acid by chemi luminescent assay

Stool sampling:

Patient was instructed to eat a well-balanced diet including fiber such as bran, cereal, fruits and vegetables. Red meat and excess of vitamin C and iron supplements in excess of 250mg per day was avoided 3 days before collection of sample.

5.4. Laboratory techniques:

Complete blood count was done using five part automated cell counter from Beckman Coulter with tri level Quality control from Bio-Rad for measuring the following values such as hemoglobin, red cell count, white cell count, PCV, MCV, MCH, MCHC and platelet count. A peripheral blood smear was prepared, stained using Leishman stain and examined under the microscope. Reticulocyte count was calculated manually after supravital staining.

The ferritin assay was done by using the technique of chemiluminescent immunoassay on Access 2 equipment from Beckman Coulter in human serum using monoclonal anti ferritin antibodies.

Serum iron and serum unsaturated iron binding capacities (UIBC) were quantitatively determined on Roche/Hitachi cobas c 311 systems using spectrophotometric immunoturbidimetric assay. TIBC was derived from the sum of serum iron and UIBC. Serum transferrin and serum transferrin saturation are derived values from serum iron and TIBC.

Vitamin B12 and serum folate were also determined using chemiluminescent immunoassay.

Erythrocyte sedimentation rate was estimated by automated infra-red based machines using Westergrens method. The C-reactive protein was determined in human serum done by immunoturbidimetric assay on Roche/Hitachi cobas c 311 systems.

Stool samples were tested for occult blood by hemospot test.

STATISTICAL ANALYSIS

6. STATISTICAL ANALYSIS

In the above study, the statistical methods used for quantitative data were descriptive statistics presented by N, Mean, Standard Deviation and Range. For qualitative data, frequency count, N and percentage were put in tabular columns.

To analyze the data, appropriate statistical tests were applied. To compare the difference between variances in the subjects, one way ANOVA was used. To find the correlation between hemoglobin, red cell indices & iron profile, Pearson's correlation coefficient was used.

All the statistical analysis had been done by using statistical software SPSS (version 16.0). Other data are displayed by various tables and charts by using Microsoft excel (windows 7).

* Significant at $p < 0.05$

** Very significant $p < 0.01$

*** Highly significant $p < 0.001$

Results

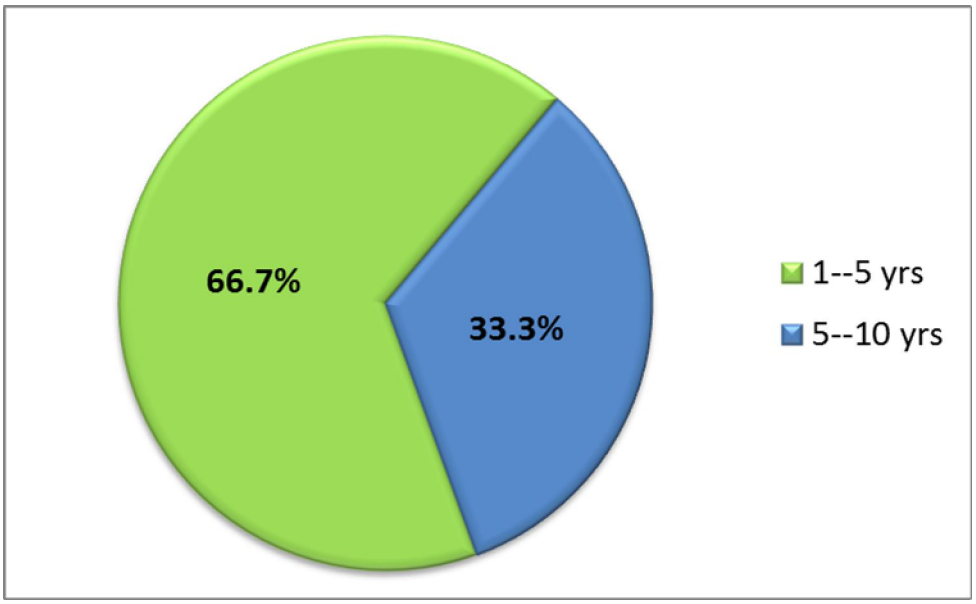
RESULTS

7. RESULTS

A total of 150 children were enrolled as study population and statistical analysis revealed the following observations.

Demographic profile:

Fig. 7.1 Age distribution of children



Among the 150 children, 66.7% (n= 100) were between 1 to 5 years of age and 37.3% (n=50) were between 5 to 10 years of age. The mean age of children with microcytic hypochromic anemia (MHA), normocytic hypochromic (NHA), and normocytic normochromic anemia (NNA) was 3.39 years ± 2.75, 5.2 years ± 3.67 and 5.05 years ± 3.22 respectively.

Fig. 7.2 : Age distribution among various types of anemia

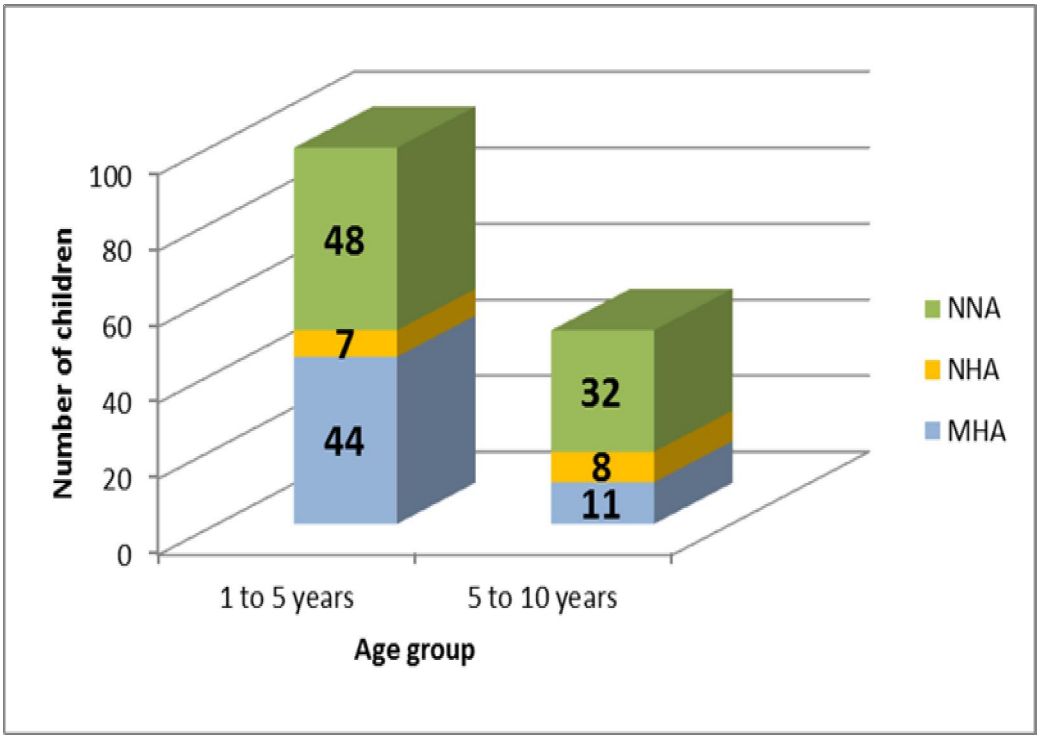
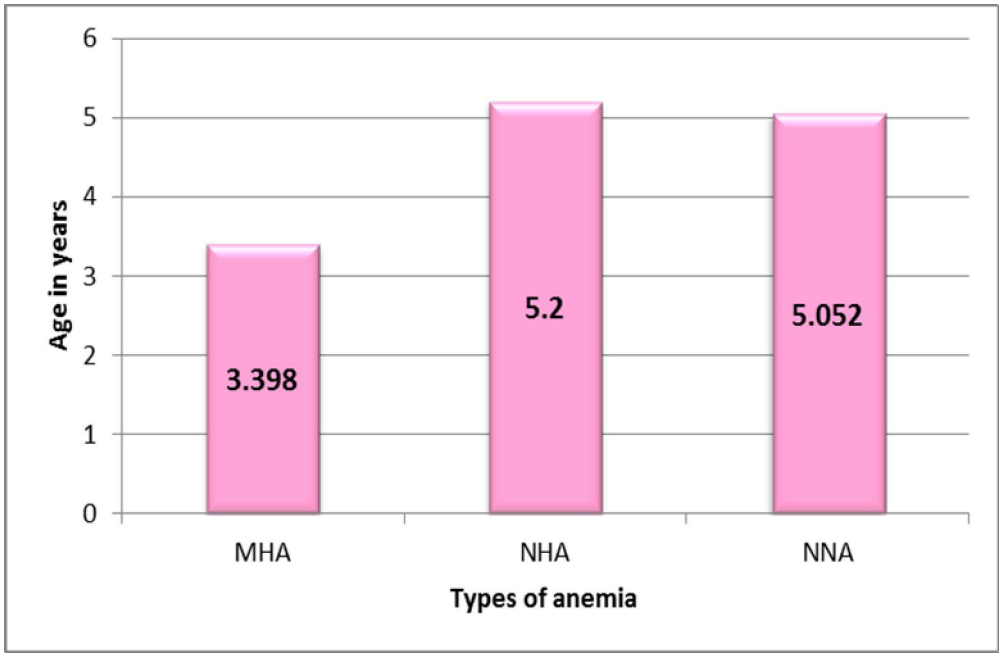


Fig. 7.3 : Mean age among various types of anemia



In children with MHA, 80% (n=44) were in the age group of 1 to 5 years and 20% (n=11) were in the age group of 5 to 10 years. In children with NHA and NNA, 53.30% (n=7) and 58.80% (n=48) were between 1 to 5 years of age respectively. Among 5 to 10 years age group, the children with NHA were 46.70% (n=8) and the children with NNA were 40% (n=32).

Fig. 7.4: Sex distribution of children

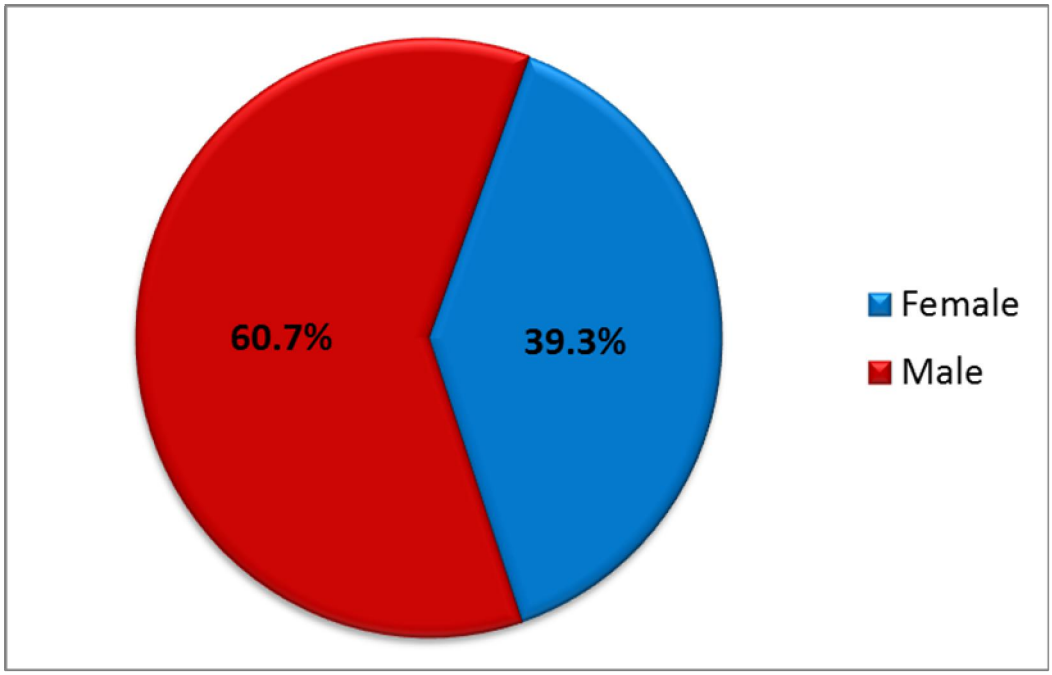


Fig. 7.5: Sex distribution among various types of anemia

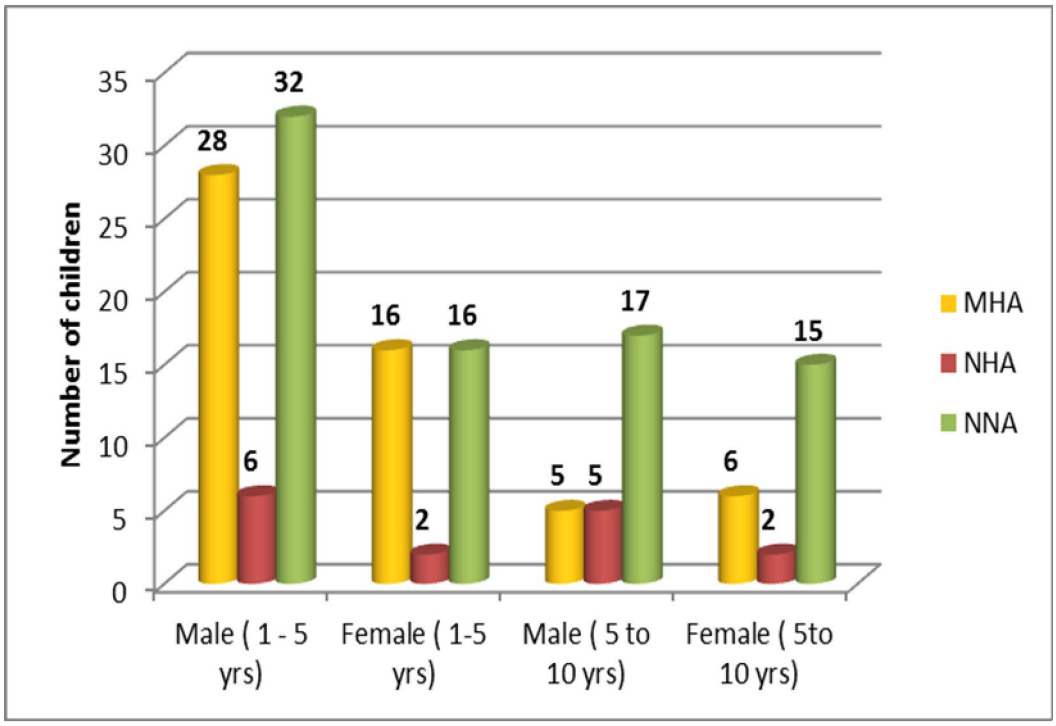


Table 7.1. Sex distribution among various types of anemia

Gender	MHA		NHA		NNA	
	N	%	N	%	N	%
Female	22	40.00%	4	26.70%	33	41.20%
Male	33	60.00%	11	73.30%	47	58.80%

Among the study population, 60.7% (n=91) were male and 39.3% (n=59) were female. In the study group with MHA, 60% (n=33) children were males and 40% (n=22) were females. In children with NHA, 73.30% (n=11) were male and 26.70% (n=4) were female. In children with NNA, 58.80% (n=47) were male and 41.20% (n=33) were female.

Fig. 7.6: Socioeconomic status of the families

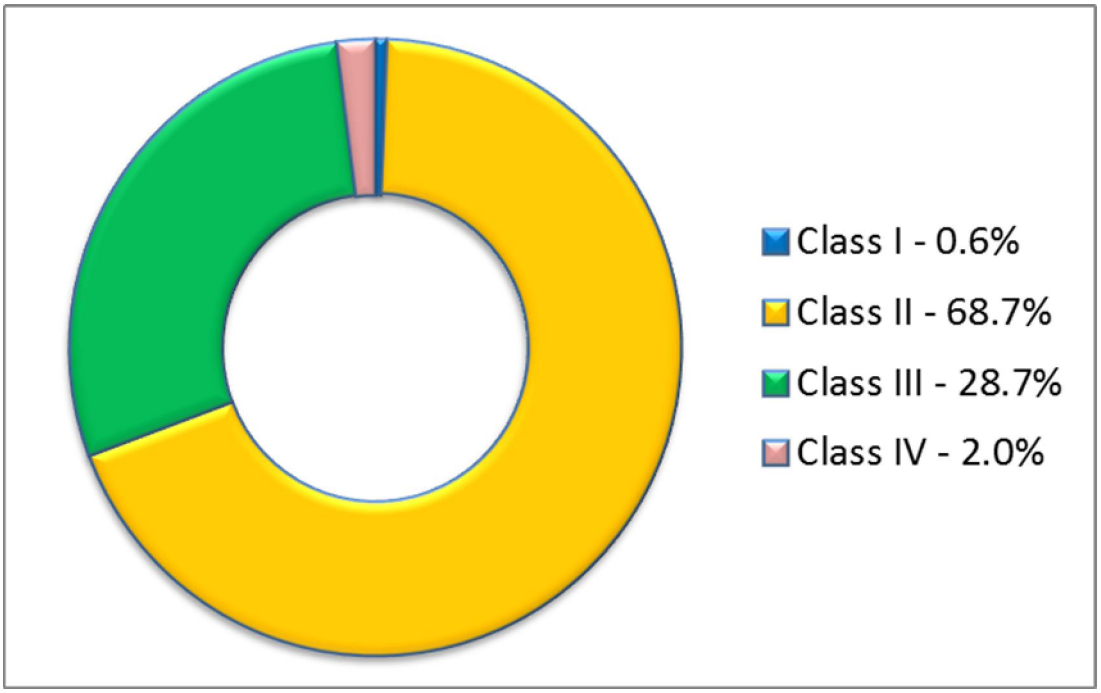
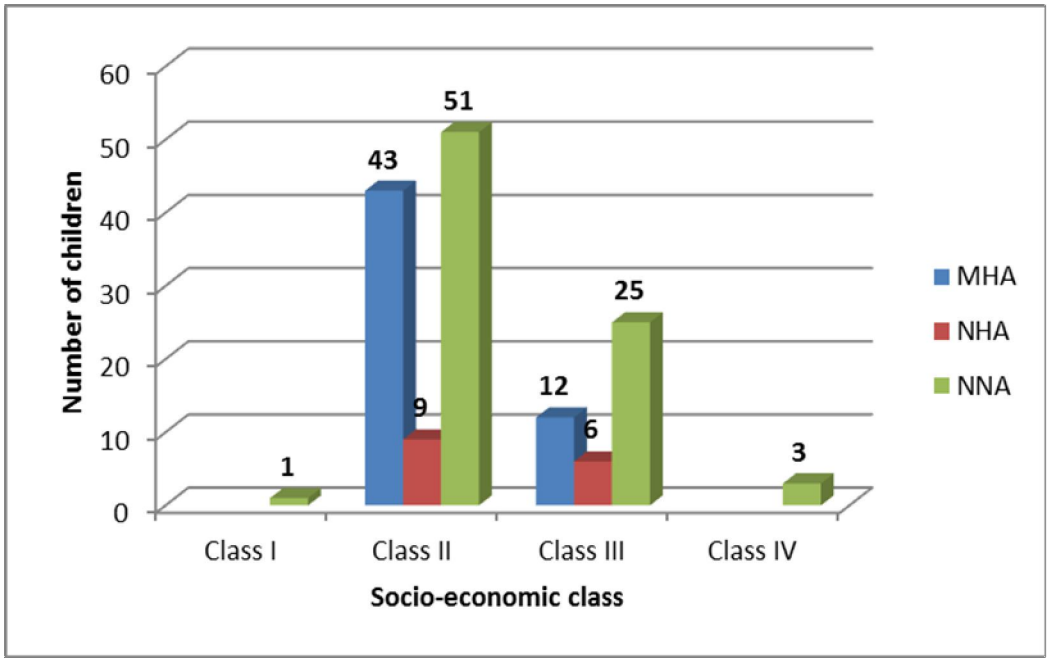


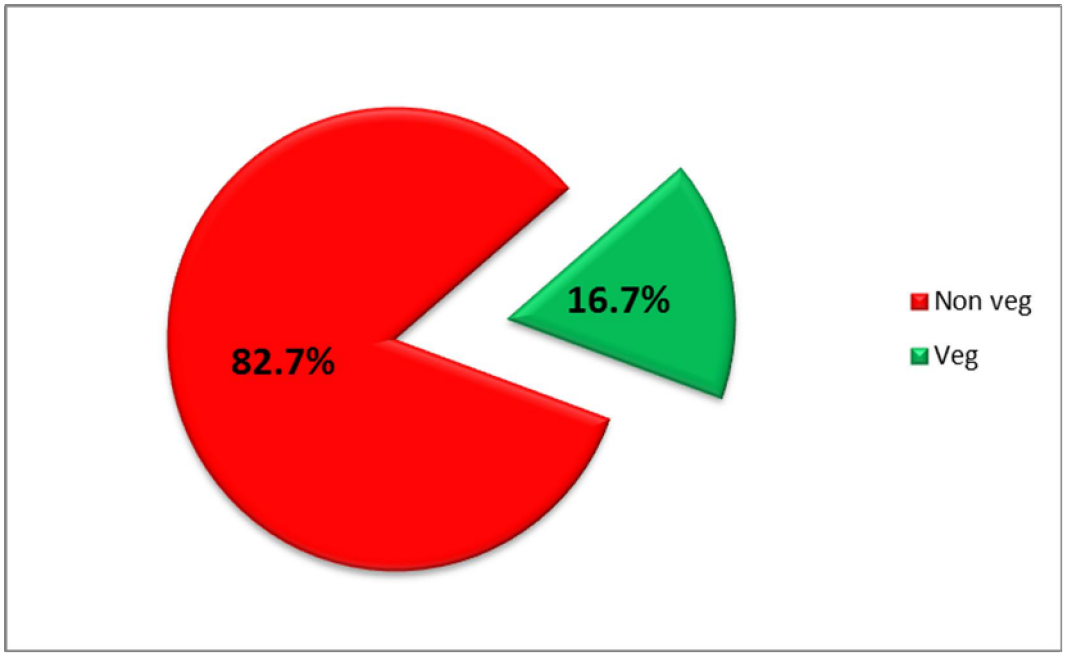
FIG. 7.7: Prevalence of anemia in various socioeconomic classes



The socio-economic status of the children was assessed by modified Kuppuswamy's scale[47] and it was found that majority of the families belonged to class II [68.7% (n=103)] followed by class III [28.7% (n=43)] and class IV [2% (n=3)] and class I [0.7% (n=1)]

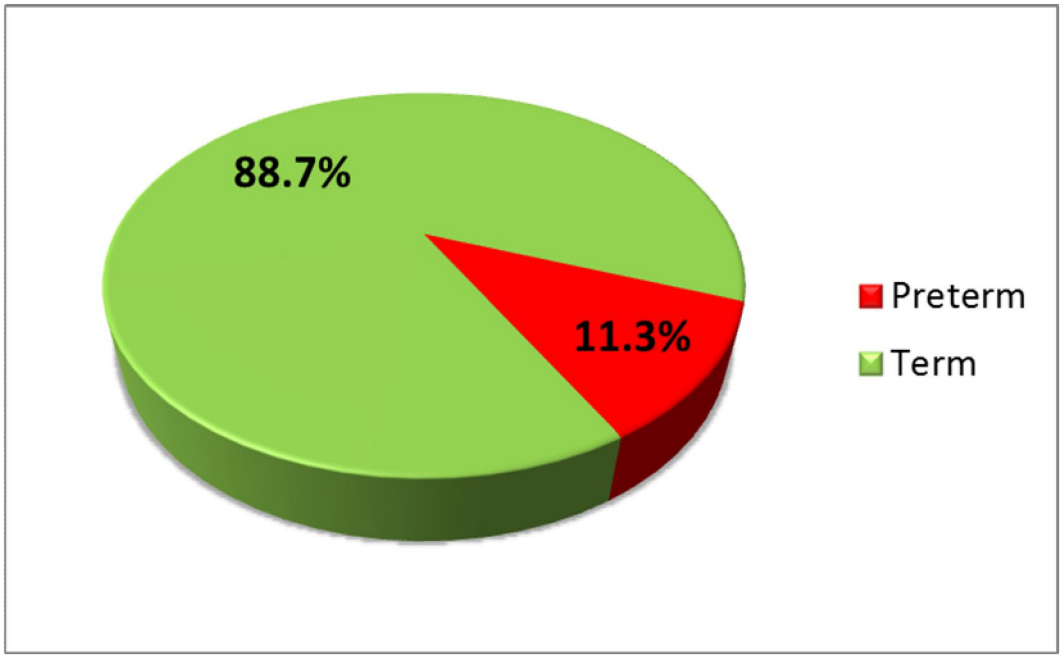
On analysing the socioeconomic class in children with MHA, it was found that 78.20% (n=43) belonged to class II socioeconomic status and 21.80% (n=12) belonged to class III.

Fig. 7.8: Dietary habits of families



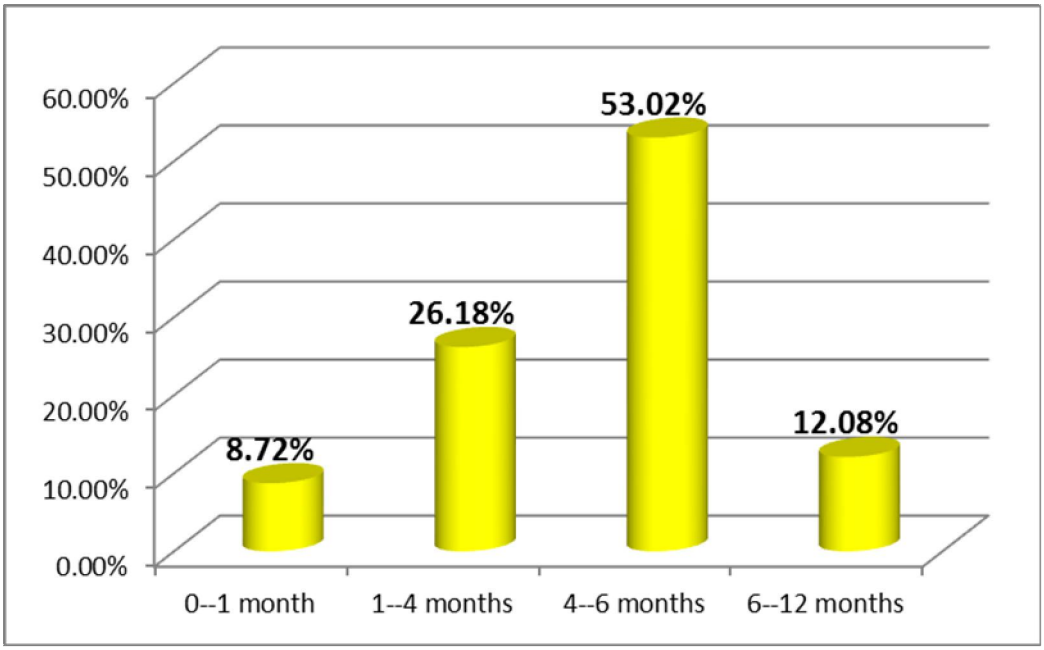
Majority of the families consumed non-vegetarian diet [82.7% (n=124)] and only 16.7% (n=26) consumed vegetarian diet.

Fig. 7.9: Gestational age at birth



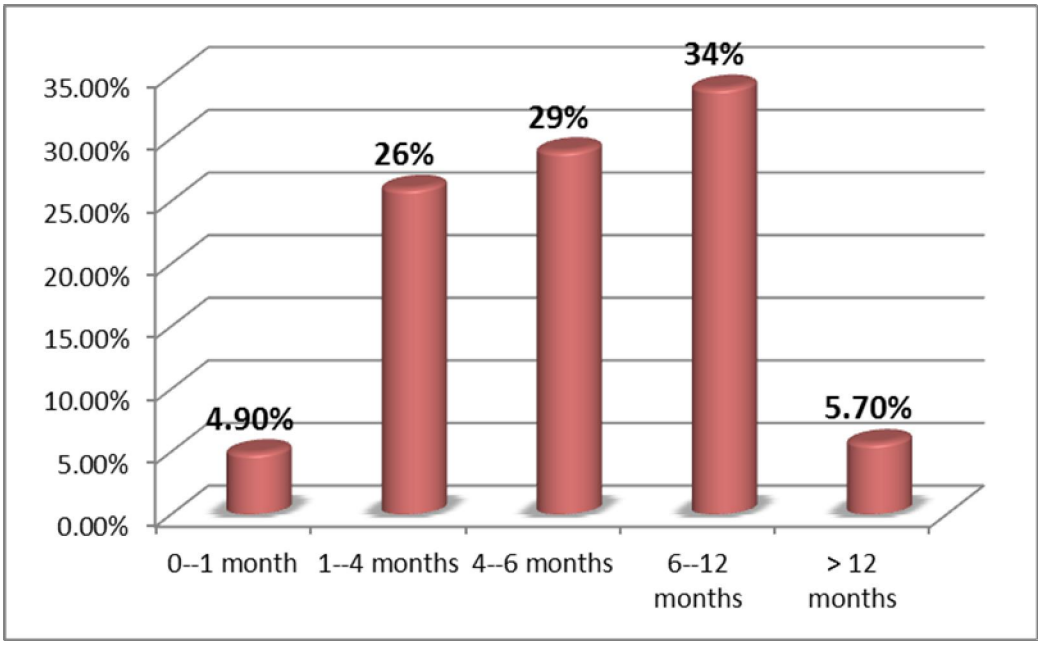
Out of the 150 children included in the study, 11.3% children were born preterm (n= 17) and the rest were born at term gestation.

Fig. 7.10: Duration of exclusive breastfeeding



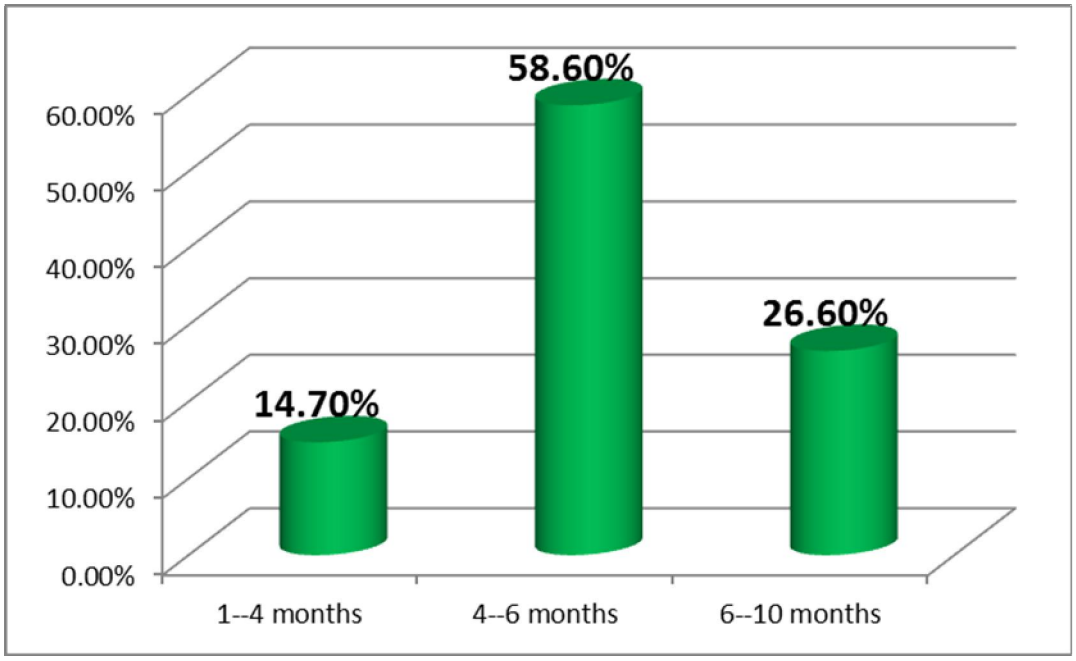
It was found that 53% (n=79) of the children were exclusively breast fed up to 6 months. In 26% (n=39) of children exclusive breast feeding was practiced only up to 4 months. In 12% (n=18) of cases, exclusive breastfeeding was given for more than 6 months. In 8.7% (n=13) of children exclusive breastfeeds were received only up to one month.

Fig. 7.11: Time of introduction of artificial feeding



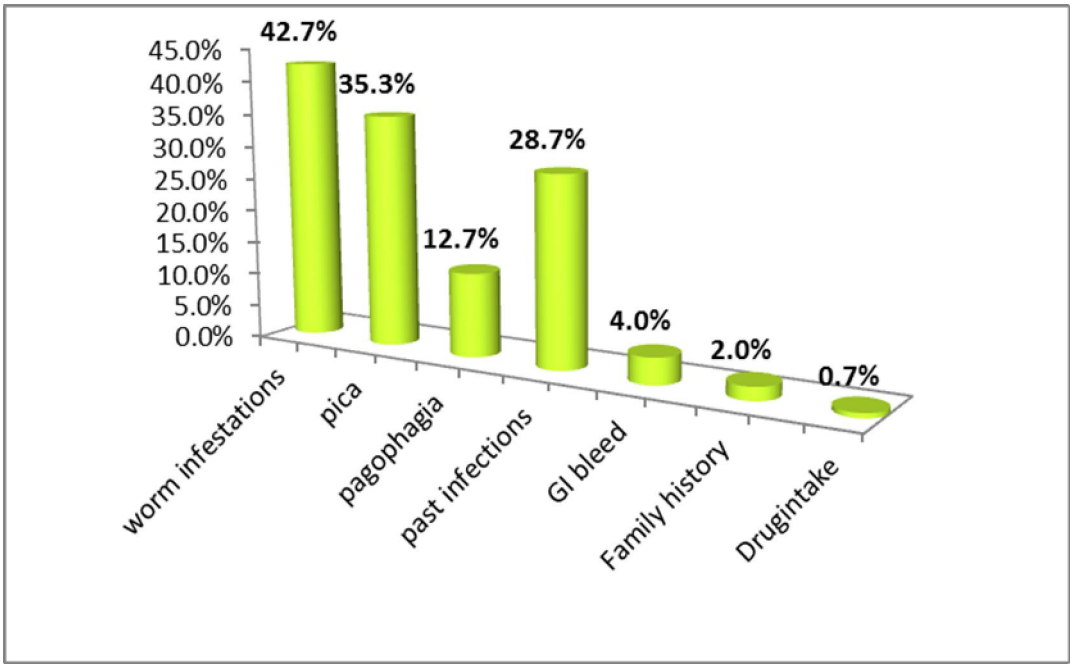
Early cow's milk/formula feeds were started in 26% (n= 32) of children between 1 to 4 months and 29% (n=36) between 4 to 6 months and 34% (n= 42) between 6 to 12 months. In 4.9% of children, artificial feed was introduced in the first month of life.

Fig. 7.12: Time of introduction of complementary feeding



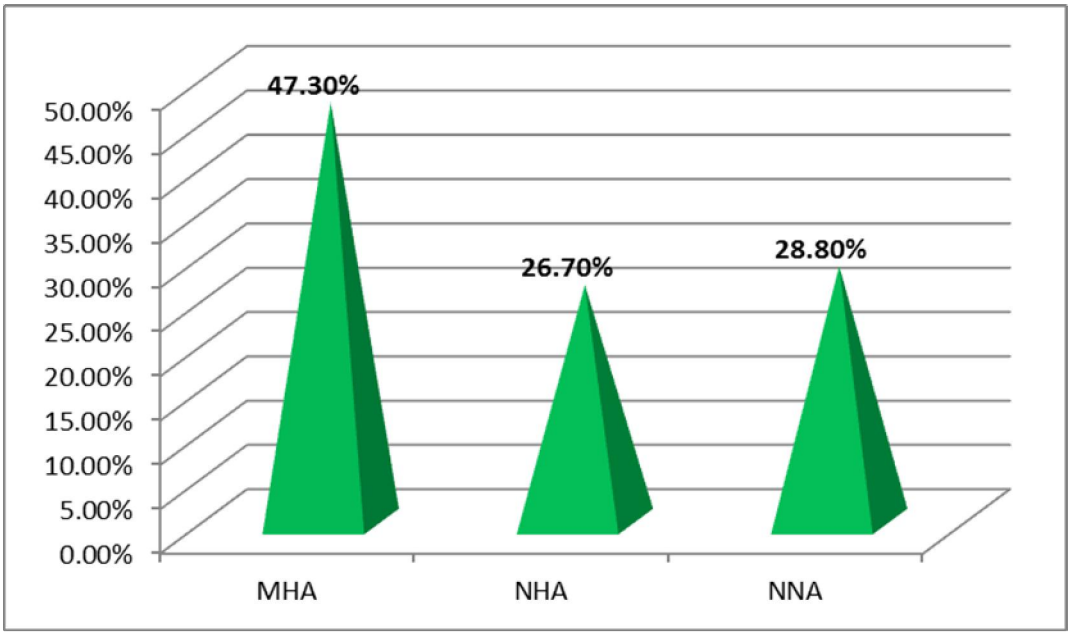
Majority of the children (58.6 %) received early complementary feeding by 4 to 6 months and in 26.6% , the children received complementary feeding between 6 to 10 months.

Fig. 7.13: History related to anemia



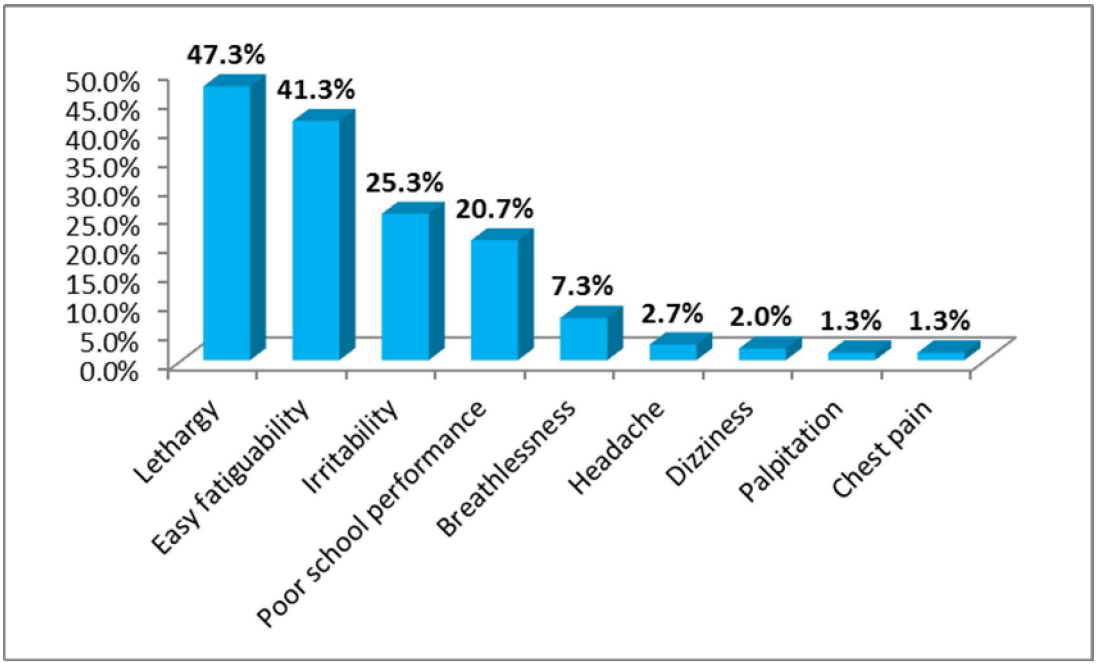
History of worm infestation was present in 42.7% (n=64) of children, pica in 35.3% (n=53), pagophagia in 12.5% (n=19), past infections in 28.7% (n=43), Gastrointestinal bleed in 4% (n=6), family history of hemolytic anemia in 2% (n=3) and drug intake in 0.7% (n=1) of children.

Fig. 7.14: Proportion of anemic children having pica



Pica was found to be more in children with MHA constituting 47.30% (n=26).

Fig. 7.15: Symptoms of anemia



Symptom analysis revealed that majority of children had lethargy which was found in about 47.3% (n=71) followed by easy fatigability in 41.3% (n=62), irritability in 25.3% (n=38), poor school performance in 20.7% (n=31), breathlessness in 7.3% (n=11), headache and dizziness in 2.7% (n=4) and 2% (n=3) respectively with palpitation and chest pain accounting for the least number in about 1.3% (n=2).

Fig. 7.16 Symptom analysis among various groups of anemia

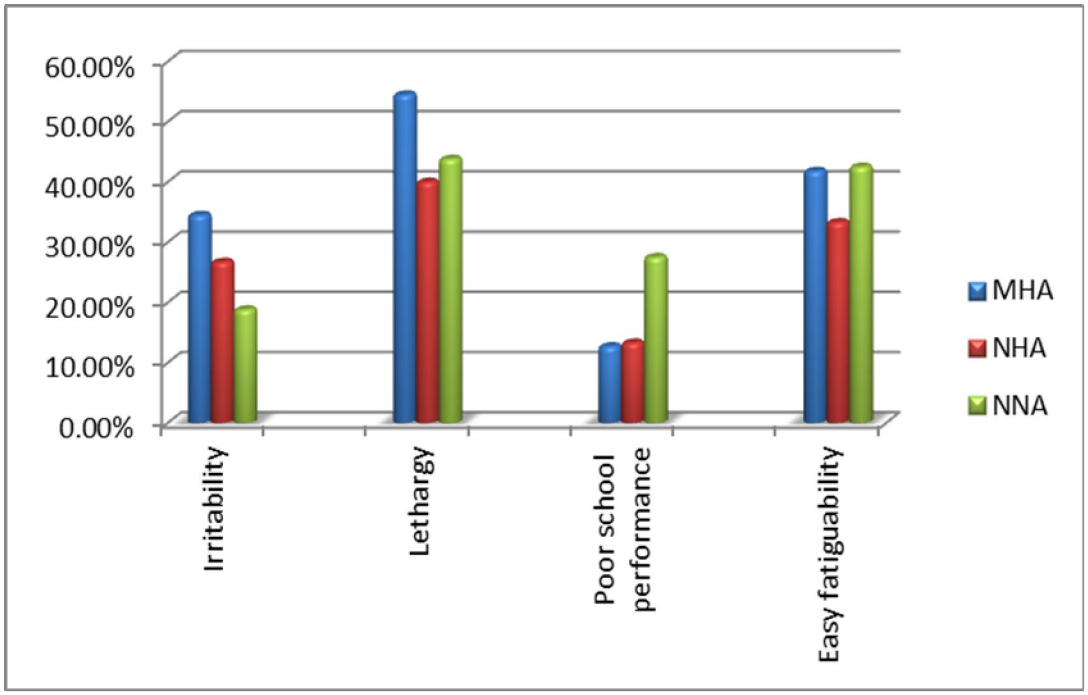
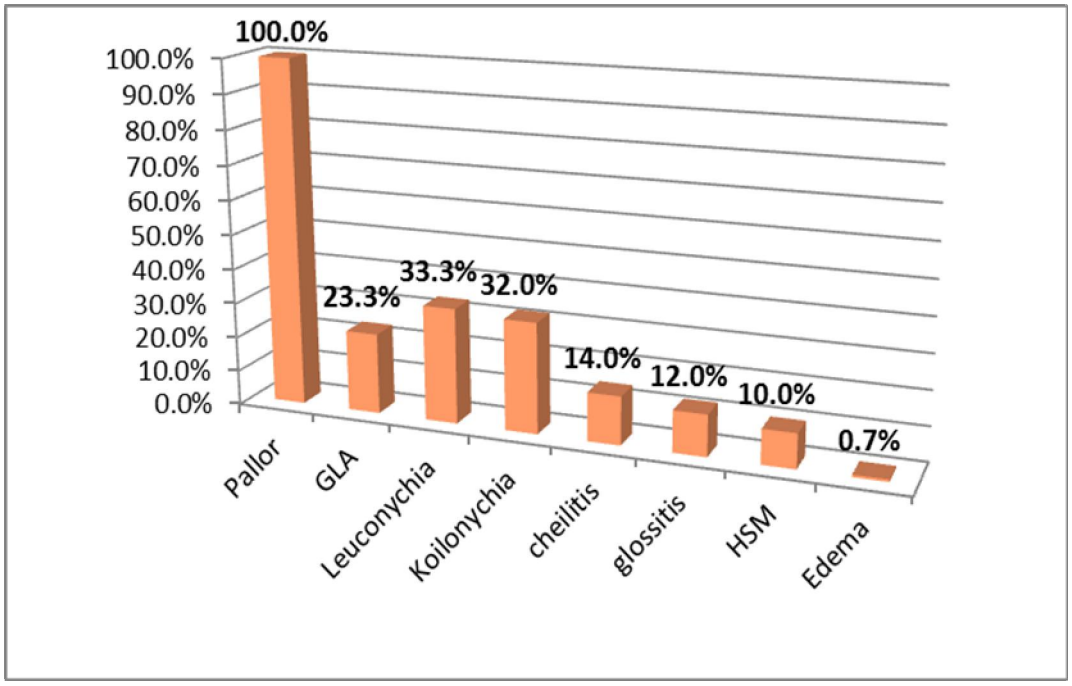


Table 7.2 Symptom analysis among various groups of anemia

Types of anemia	**MHA**		**NHA**		**NNA**	
Symptom	N	Mean	N	Mean	N	Mean
Irritability	19	34.50%	4	26.70%	15	18.80%
Lethargy	30	54.50%	6	40.00%	35	43.80%
Poor school perfomance	7	12.70%	2	13.30%	22	27.50%
Easy fatigability	23	41.80%	5	33.30%	34	42.50%

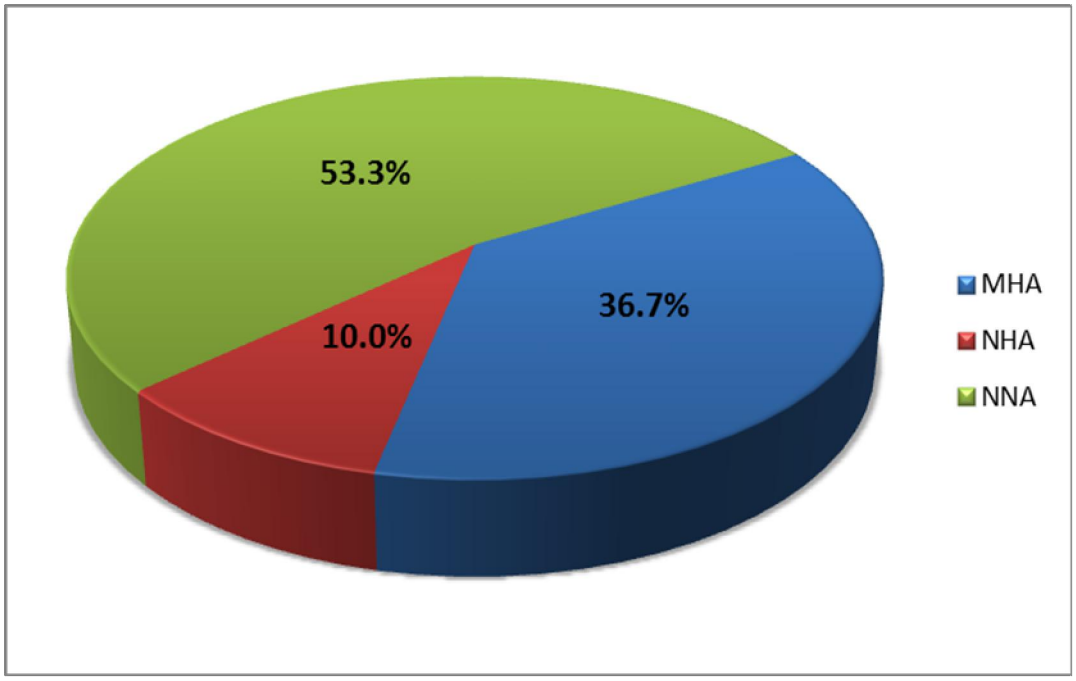
Among the 150 children with various types of anemia, irritability [34.50 % (n=19)] and lethargy [54.50% (n=30)] was more in children with MHA.

Fig. 7.17: Signs of anemia



Among the 150 children examined, leuconychia was present in 33.3% (n=50), koilonychia in 32% (n=48), generalized lymphadenopathy in 23.3% (n=35). Cheilitis, glossitis, hepatosplenomegaly was found in 14% (n=21), 12% (n=18) and 10% (n=15) respectively.

Fig. 7.18: Proportion of various types of anemia



It was noted from our observation that among the 150 children, 36.7% (n=55) had microcytic hypochromic anemia, 10% (n=15) had normocytic hypochromic anemia and 53.3% (n=80) had normocytic normochromic anemia.

Fig. 7.19: Descriptive statistics of CBC profile

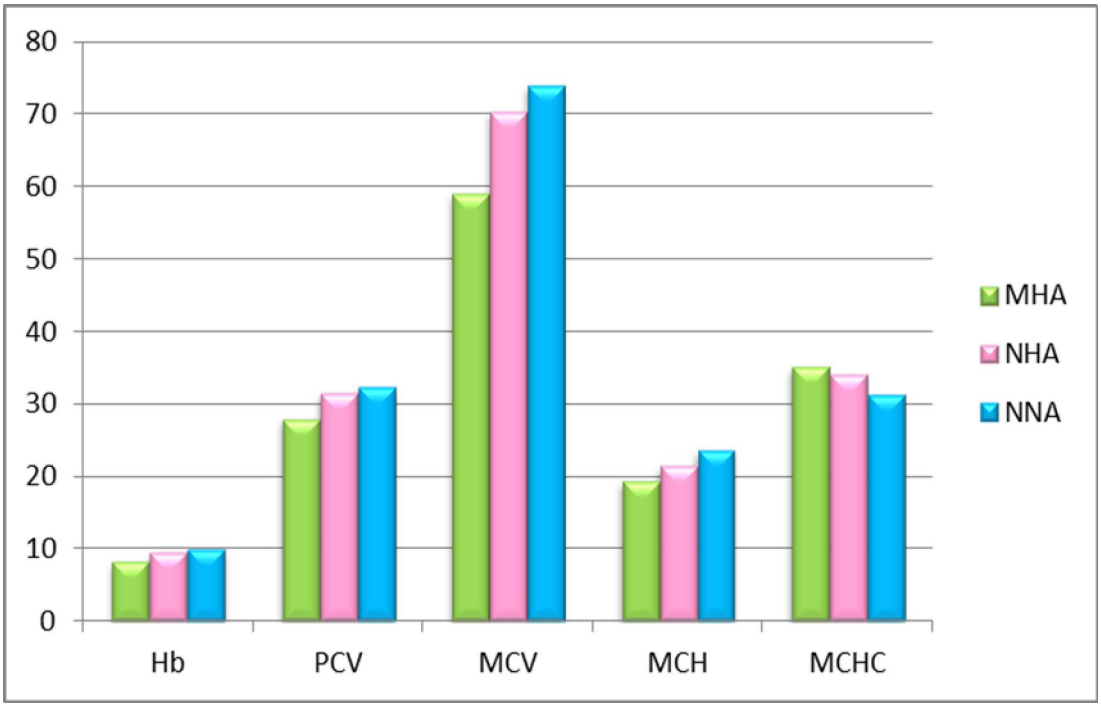
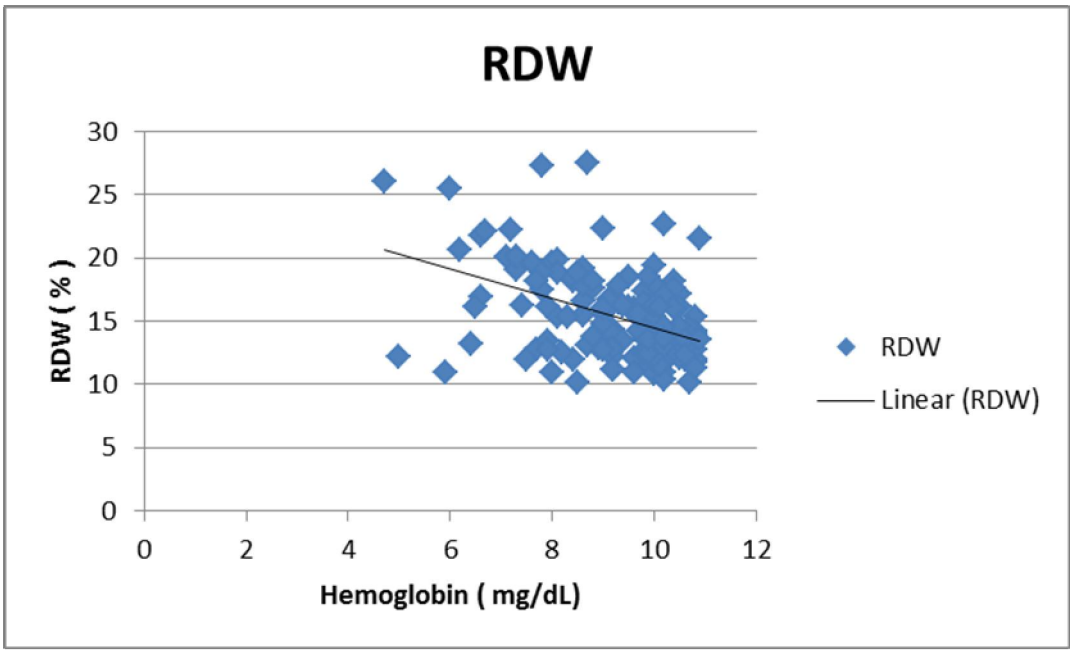


Table 7.3 Mean values of red cell indices

Types of anemia	MHA	NHA	NNA
Hb	8.22 ± 1.27	9.58 ± 0.91	9.925 ± 0.91
PCV	28 ± 9.22	31.43 ± 3.58	32.33 ± 3.58
MCV	59.1 ± 12.30	70.29 ± 7.06	73.98 ± 10.44
MCH	19.33 ± 5.96	21.39 ± 1.94	23.53 ± 2.95
MCHC	35.08 ± 36.18	34.1 ± 13.5	31.31 ± 2.50

Statistical analysis of complete blood count reveals the mean Hb is 8.22 ± 1.27 in children with MHA and in children with NHA and NNA, the mean Hb is 9.58 ± 0.91 and 9.925 ± 0.91 respectively. The mean values of PCV, MCV, MCH, are less in MHA when compared to NHA and in NNA.

Fig. 7.20: Scatter plot diagram showing correlation between hemoglobin and RDW



The mean RDW in children with MHA was 16.75 ± 3.96, 14.61 ± 2.44 in NHA and 14.50 ± 2.93 in NNA. The scatter plot diagram shows that, in our study, the red cell distribution width increases as hemoglobin values decrease.

Fig. 7.21: Descriptive statistics of iron profile

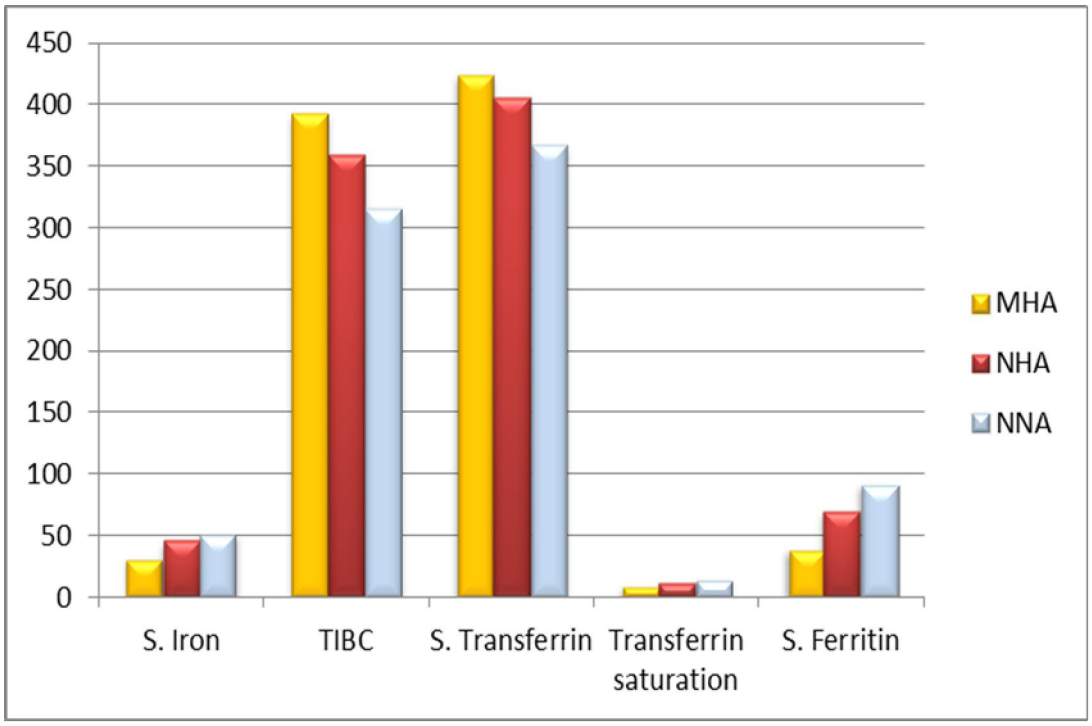


Table 7.4 Mean values of iron profile

Types of anemia	MHA	NHA	NNA
S. Iron (µg/Dl)	31.62 ± 23.90	46.22 ± 23.40	51.64 ± 22.52
TIBC (µmol/L)	392.82 ± 103.7	359.96 ± 79.11	315.8 ± 81.36
S. Transferrin (mg/dL)	424.44± 90.45	406.17 ± 83.63	367.44 ± 77.38
Transferrin saturation (%)	8.76 ± 11.25	11.45 ± 5.71	14.68 ± 7.63
S. Ferritin (ng/mL)	38.84 ± 49.75	69.97 ± 138.01	91.93 ± 203.90

The descriptive statistics of iron profile revealed that the mean serum iron levels were 31.62 ± 23.90 μg/dL in children with MHA, 46.22 ± 23.40 μg/dL in children with NHA and 51.64 ± 22.52 μg/dL in children with NHA. The mean TIBC was increased in children with MHA with a mean of 392.82 ± 103.7 μmol/L, and in children with NHA, the mean TIBC is 359.96 ± 79.11 μmol/L, and in NNA , the mean is 315.8 ± 81.36 μmol/L. Serum transferrin was high in children with MHA and in children with NHA, the mean transferrin levels were low when compared to MHA. The mean serum ferritin levels were 38.84± 49.75 ng/dL in children with MHA and in children with NHA the mean levels were 69.97 ± 138.01 ng/dL, and 91.93 ± 203.90 ng/dL respectively.

Table 7.5 Pearson's correlation statistics between hemoglobin, red cell indices and iron profile

Pearson Correlation	PCV	MCV	MCH	MCHC	RDW	S. Iron	TIBC	Transfer. Sat	S. Transferrin	S. Ferritin	Reticulocyte count
Hemoglobin	.366**	.316*	.308*	0.225	-.287*	0.259	-0.228	0.188	-0.193	0.125	-0.072
P value	0.006	0.019	0.022	0.099	0.034	0.057	0.094	0.169	0.158	0.362	0.603

On analyzing the data of MHA with Pearson correlation statistics of hemoglobin and red cell indices, it was found that hemoglobin had weak positive correlation with PCV, MCV, MCH and weak negative correlation with RDW. The correlation is at $p<0.05$ level significance. Hemoglobin has weak correlation with iron studies and it is not statistically significant.

Table 7.6 Comparison of mean differences between 3 groups of anemia

ANOVA test							
Types of anemia	**MHA**		**NHA**		**NNA**		**Sig**
Parameters	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**p value**
Hemoglobin	8.23	1.27	9.58	0.76	9.93	0.91	0.0001***
PCV	28.00	9.23	31.43	3.59	32.33	3.90	0.0001***
MCV	59.10	12.30	70.29	7.07	73.98	10.44	0.0001***
MCH	19.33	5.97	21.39	1.94	23.53	2.95	0.0001***
MCHC	35.09	36.18	34.10	13.52	31.32	2.50	0.617
RDW	16.75	3.96	14.61	2.44	14.50	2.93	0.001**
Reticulocyte count	1.08	1.19	0.55	0.26	0.84	0.36	0.045*
S. Iron	31.62	23.90	46.22	23.40	51.65	22.52	0.0001***
TIBC	392.82	103.70	359.96	79.11	315.80	81.36	0.0001***
Transferrin saturation	8.76	11.25	11.46	5.71	14.68	7.63	0.001**
S. Transferrin	424.44	90.45	406.18	83.63	367.45	77.38	0.001**
S. Ferritin	38.84	49.75	69.98	138.01	91.93	203.90	0.164

One way ANOVA test was used to compare the differences in mean of various indices among the children with MHA, NHA and NNA. The mean difference between groups with respect to hemoglobin, PCV, MCV, MCH and RDW was statistically highly significant (p=0.0001). The reticulocyte count also was compared and the difference was statistically significant (p = 0.045). It is seen from the table above that the mean difference in serum ferritin levels (p=0.164) and MCHC (p=0.617) was not statistically significant between the groups.

Table 7.7 Comparison of serum ferritin, ESR and CRP between the groups

Types of anemia	S. Ferritin		ESR		CRP	
Values	< 15 ng/mL	>15 ng/mL	< 20 mm/Hr	>20 mm/Hr	<10 mg/L	>10 mg/L
MHA	26 (47.27%)	29 (52.72%)	29 (52.72%)	26 (47.27%)	35 (63.63%)	20 (36.36%)
NHA	4 (26.6%)	11 (73.33%)	8 (53.33%)	7 (46.67%)	8 (53.33%)	7 (46.67%)
NNA	17 (21.25%)	63 (78.75%)	37 (46.25%)	43 (53.75%)	43 (53.75%)	37 46.25(%)

Table 7.8 Proportion of cases with elevated ESR/ CRP and normal serum ferritin levels

Types of anemia	ESR	CRP
MHA	15 (51.7%)	10 (34.48%)
NHA	5 (45.45%)	3 (27.27%)
NNA	38 (60.3%)	31 (49.20%)

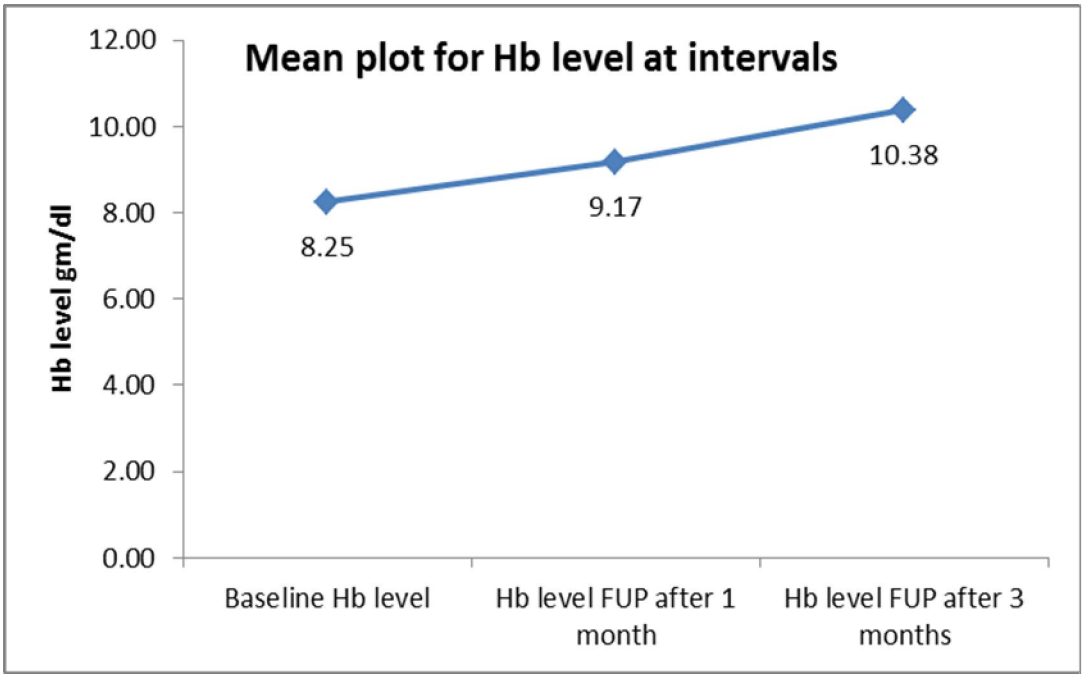
From the previous observations, it was obvious that serum ferritin was not reduced in all children with anemia. Hence, the correlation with ESR and CRP was done and it revealed that out of 29 children with MHA, 15 had elevated ESR and 10 children had elevated CRP. In NHA, 5 out of 11 children and in NNA, 38 out of 63 children had increase in ESR. In NHA, 3 out of 11 and 31 out 63 had increased CRP levels.

A total of 7 out of 29 children with MHA had elevations in both ESR and CRP and 3 out 11 children with NNA and 24 out of 63 children also had elevations in both ESR and CRP.

Treatment outcome (n=50):

Out of the 150 children, a total of 55 children had MHA. These children were given oral iron supplementation and were followed-up. 5 children were lost to follow-up. The results of the follow-up are shown below.

Fig. 7.22: Improvement in hemoglobin after iron therapy



The follow-up study after oral iron supplementation had shown that the mean hemoglobin increased from the baseline value of 8.25 ± 1.302 mg/dL to 9.17 ± 1.365 mg/dL after one month and it increased to 10.38 ± 1.368 mg/dL after 3 months.

Table 7.9 Follow-up hemoglobin and its significance

Paired Samples Test								
Parameters	**Paired Differences**			**95% Confidence interval of the Difference**				
	Mean	**Std. Deviation**	**Std. Error Mean**	**Lower**	**Upper**	**t test value**	**df**	**p value**
Hb level - Baseline and follow-up after 1 month	-0.916	1.100	0.156	-1.229	-0.603	-5.888	49	**0.0001*** **
Hb level - Baseline and follow-up after 3 months	-2.126	1.277	0.181	-2.489	-1.763	-11.771	49	**0.0001*** **
Hb level after 1 month and 3 months follow-up	-1.210	0.651	0.092	-1.395	-1.025	-13.137	49	**0.0001*** **
Reticulocyte count - Baseline and follow-up after 1 month	-0.272	1.232	0.174	-0.622	0.077	-1.56	49	0.125 (NS)

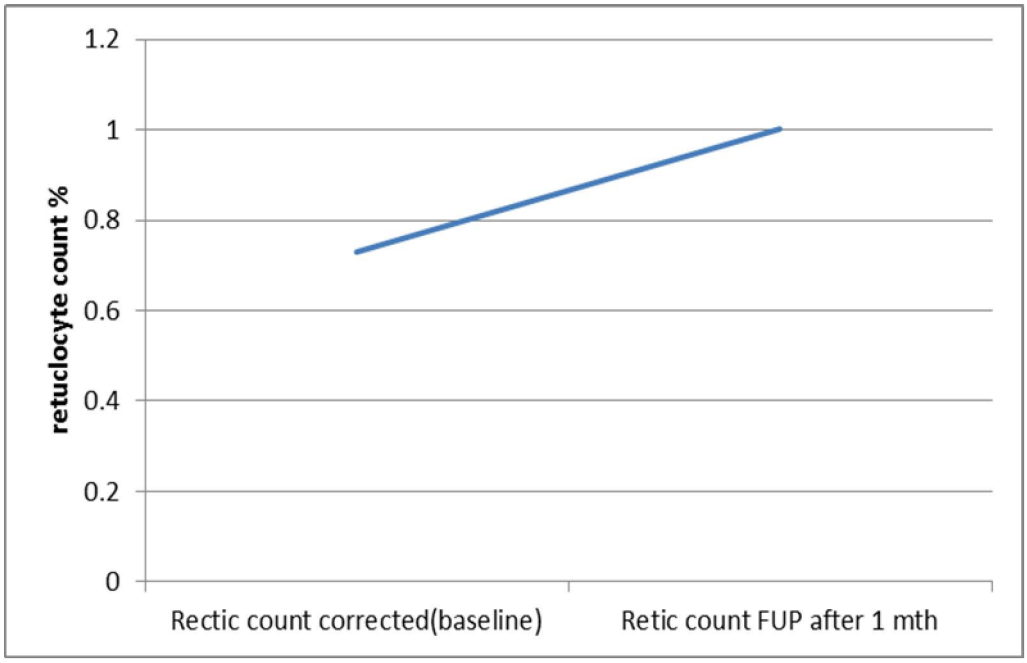
*** *Highly Significant, NS-Not Significant*

The mean increase in hemoglobin at 1 month from baseline month and then at 3 months was statistically significant (p=0.0001).

Table 7.10 Improvement in reticulocyte count after one month

Corrected reticulocyte count	**Mean**	**Std. deviation**	**Std. error**
Baseline	0.729	0.610	0.086
Follow-up after 1 month	1.001	0.968	0.137

Fig 7.23 Mean increase in reticulocyte count after treatment



It is seen from the observation that the corrected reticulocyte count has increased from the baseline of 0.7 to 1.0.Though there is an increase in reticulocyte count it has not shown any statistical significance (p=0.125).

Discussion

DISCUSSION

This study was primarily designed to determine the clinical and laboratory profile of nutritional anemia among children attending the Pediatric outpatient Department of a tertiary care hospital.

A total of 150 children were recruited, following confirmation of anemia with hemoglobin estimation. After a targeted history and detailed clinical examination, they were subjected to red cell indices and iron studies besides RDW, reticulocyte count and peripheral smear. Serum vitamin B12 and folic acid levels were estimated in all cases. An attempt was also made to look for laboratory evidence of inflammation by estimating ESR and CRP in all cases.

Based on the above investigations, anemia was grouped into microcytic hypochromic anemia (MHA, 55cases), normocytic hypochromic anemia (NHA, 15 cases) and normocytic normochromic anemia (NNA, 80 cases). There were no cases of macrocytic anemia on peripheral smear examination and serum B12 and folic acid levels were normal in all cases.

On analyzing the group characteristics of MHA, it was found that 80% of the children belonged to a younger age group (1-5 years). A similar observation was also noted in a study by Rayn et al. wherein 75%

of children with IDA were in the 1-2 years age group. However, there was no statistically significant difference in gender distribution.

On the socio economic front, most of the cases belonged to a relatively higher class (78% in Class II Kuppuswamy Scale). The presence of anemia, in this socioeconomic group, is explained by the fact that most of the mothers were working and hence the diet of the child probably remained unsupervised. Chellan et al. also observed a greater prevalence of anemia in higher socioeconomic strata.[24]

Worm infestation, which may have been a predominant contributor to IDA, was seen in up to 44% of cases and pica was seen in 47.3% of children with microcytic hypochromic anemia. Subjective symptoms (lethargy, easy fatigability, irritability) were seen in 55% of cases whereas objective symptoms like breathlessness and palpitations were seen in very few. The absence of objective symptoms in our series is presumably because of the lesser severity of anemia as evidenced by mean hemoglobin of 8 g/dL. Maheshwari et al. had observed fatigue in 54% and weakness in 38% of their cases.[41]

Among the 55 cases with MHA, though MCV and MCH values were suggestive of iron deficiency, serum ferritin values were normal in 53% of cases. This was similar to the observation made by Duque et al.[26] where >50% children in the age group of 1-2 years had no correlation between iron deficiency and serum ferritin levels. It was also seen that in

the study conducted by Maheshwari et al. only 35% had low ferritin levels.[41]

Ferritin is an acute phase protein that rises in response to inflammation. The presence of normal ferritin in cases of IDA can be explained by the fact that in a hospital based study like ours, subclinical infection or latent inflammation could well be the cause for such an elevation, thus masking the true size of the iron store.[13] This association with inflammation is also suggested by the elevated CRP in 36.36% of cases and elevated ESR in 47.3%.When such confounders are encountered; the estimation of RDW may help in identifying true IDA.

RDW is a quantitative measure of all the variation in the width of the RBCs. It is of great value in discriminating IDA from other causes of microcytic hypochromic anemia, but studies in children are few. Elevated RDW (>15%) was seen in 67% of our MHA cases. RDW was found to have a strong correlation with IDA in the present study. In studies by Sazawal et al and Aulakh et al. the sensitivity of RDW was found to be 81% in diagnosing IDA.[33,34]

A small proportion of patients (n=15) with NHA are probably patients with evolving iron deficiency as the average MCH value in this group was low at 21.9%. With time, these children would have evolved into frank hypochromic, microcytic anemia. In a resource poor setting, there is an urgent need to identify IDA in its earliest stage and replenish

stores to prevent cognitive impairment and growth failure. Probably, this group of children too will benefit from oral iron therapy.[28]

Among the 150 recruited cases, we were surprised to find that more than half the children (80 cases) belonged to the category of normocytic normochromic anemia, which is classically associated with chronic disease, even though we did not include any cases with overt systemic illness or inflammation in the study.[13] Symptomatology of these cases mimicked iron deficiency and the finding of pica and koilonychia in one-third of the cases also prompted us to study the parameters in greater detail. The ferritin levels were normal in the majority of this group (79%) and low in only one-fifth of the cases.

Inflammatory disorders cause elevation of circulating hepcidin, which causes a blocking effect on enterocytes and reticulo-endothelial system leading to iron deficient erythropoiesis. Since hepcidin levels cannot be assessed easily, an alternate and cheaper modality would be needed.

A widely used and easily available marker of inflammation is the CRP with values above 10 - 30mg/L being significant. On analysis of cases of NNA with normal ferritin, it was found that 39% had elevated CRP while 47% had increased ESR, again indicating transient infection or inflammation being the triggering agents. Thus, in anemic children with elevated CRP, concurrent iron deficiency must be considered despite

high ferritin levels. In such situations increased RDW has been seen to be a strong pointer towards iron deficiency.

In our study, RDW had a strong correlation with anemia in only 35% of children with normocytic normochromic anemia. It is unlikely that such a significant number of children would actually be suffering from anemia of chronic disease. It is likely that such children too may have latent iron deficiency anemia and it is this group that in a busy OPD, may get overlooked. It is important to note that they too may benefit from oral iron therapy and that further studies are needed to establish the same.

In terms of correlation of the hemoglobin with the red cell indices, we found a positive correlation between hemoglobin and MCV and MCH which was statistically significant and a statistically significant negative correlation with the RDW. This has been borne out amply in many studies wherein a high RDW has been found to be a sensitive marker of iron deficiency.

Treatment and follow up:

All cases with MHA were treated with iron supplements in therapeutic doses and hemoglobin and reticulocyte count were repeated at 1 month followed by hemoglobin alone at 3 months after starting treatment.

There was an increase in reticulocyte count as well as hemoglobin at 1 month follow up but the rise in the reticulocyte count was much lower than the expected i.e. from a mean count of 0.7% to 1 %. The mean increase in hemoglobin was only 2 g% over three months while the expected rise is about 1 – 2 g% per month[8]. This was attributed to issues related to compliance, GI intolerance and also to frequent inter current infections necessitating temporary iron withdrawal. The studies done by Maheshwari et al.[41] and Huang et al.[43] showed that only 78.9% cases improved with iron indicating that supplementation of a deficient nutrient may not result in 100% improvement due to multiple confounders.

In summary, in our study we found a major percentage of children with normocytic normochromic anemia rather than microcytic hypochromic anemia. These cases, as discussed earlier, are presumably cases with latent iron deficiency and will probably benefit with oral iron therapy followed by a meticulous follow up to detect other disorders. Though, in the formulation of the study design we had treated only established cases of MHA with iron supplements, the finding of such a large number of children with NNA, makes us postulate that it would be ideal to give a trial of oral iron to this group also and then estimate the response rather than go in for expensive investigations to definitely establish other causes.

Since the ferritin values were inconclusive in the diagnosis of anemia, it may not always be useful as a single parameter to either diagnose or exclude IDA. Probably RDW could have a better diagnostic value in IDA. Larger studies comparing RDW with anemia are needed in the future.

LIMITATIONS

1. Sample size should have been larger
2. The group with NNA and NHA could have been given a therapeutic trial of iron in order to validate our postulate

Conclusion

CONCLUSION

1. In children with IDA, normal or high ferritin level should not distract the physician away from the diagnosis as co-existing infection and inflammation may confound the picture.

2. In cases of normocytic, normochromic anemia, S. ferritin levels can mask a latent iron deficiency. In doubtful cases a therapeutic trial of oral iron is more cost effective. If red cell and iron indices do not improve with an adequate trial of iron, it may be worthwhile to go ahead with more sophisticated and expensive investigations to find alternate causes.

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S. N	Name	age	agegr	sex	Socstats	term_preterm	breastfed	dur_breastfeed	timeintrofeed_cm	time_intrcfeed	V_NV	worminfe	pica	pagophagia	pastinfec	Gibleed	Fami_Heanemia	irritability	lethargy	poorscperf	easyfatig	breathLN	palpitation	chestpain	dizzyness	headache	Drugintake	Ht	Ht_m	Wt	BMI	pallor	icterus	GLA	Edema	Koilonychia	Leuconychia	glossitis	cheilitis	hepatosp	Hb		WBC	RBC	PCV	MCV	MCH	MCHC	RDW	Plt_count	Retic_count	sIron	TIBC	TransfeSat	Stransferrin	SFerritin	SB12	SFolicacid	ESR	CRP	PS	FOBT	FUHB1	Ret_ct	FUHb2
1	Nithish	1.5	1--5	M	2	T	Y	6	1.5	7	NV	N	N	N	Vfever	N	N	N	N	N	N	N	N	N	N	N	N	80	0.8	9.3	14.53	Y	N	N	N	N	N	N	N	N	9.5	9--11	12900	4.36	29.2	67	21.7	32	16.2	2.74	0.5	28.4	414	6.42	442.09	3.9	196	80.31	10	0.69	NNA	N			
2	Srikanth	7	5--10	M	2	T	Y	6	0	6	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	108	1.08	17.8	15.26	Y	N	N	N	N	N	N	N	N	10	9--11	11700	4.42	33.2	75	22.7	30	16.1	2.2	0.8	34.36	310	9.98	344.16	31.9	315	4.67	31	13.08	NNA	N			
3	Mohammed Firadaus	5	1--5	M	2	T	Y	6	17	6	NV	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	100	1	15	15.00	Y	N	N	N	N	N	N	N	N	9	7--9	6000	5.24	29.4	56.2	17.7	32	15.6	4.06	0.5	115.6	152	43.25	267.19	131.9	651	15.92	59	1.04	MHA	N	10.0	1.0	11.2
4	Sivasakthi	5	1--5	M	2	T	Y	1	2	6	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	109	1.09	15.2	12.79	Y	N	N	N	N	N	Y	Y	N	6.6	>=7	7900	3.68	20.2	54.9	17.9	33	21.8	3.61	0.5	7.97	279	2.77	287.46	83.7	347	4.85	12	25.92	MHA	N	7.1	0.3	8.8
5	Vaishali	2.5	1--5	F	2	T	Y	5	12	5	NV	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	80	0.8	10	15.63	Y	N	N	N	N	N	N	N	N	9.6	9--11	6600	4.57	31.1	68	21	31	15.7	3	0.6	53.97	350	13.37	403.56	7.1	325	11.13	9	3.37	NNA	N			
6	Logesh	1.5	1--5	M	3	T	Y	10	12	10	NV	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	80	0.8	10	15.63	Y	N	N	N	N	N	N	N	N	10	9--11	11001	4032	32.7	75.7	24.9	33	14	3.63	1	87.65	327	21.13	414.88	23.6	937	25.2	31	3.11	NNA	N			
7	Tharani	2	1--5	F	3	T	Y	5	5	9	NV	N	Y	N	Walri	N	N	Y	N	N	N	N	N	N	N	N	N	78	0.78	8.3	13.64	Y	N	N	N	N	N	N	N	N	8	7--9	9000	4.31	25	57.9	18.6	32	19.6	2.79	1	21.75	424	4.88	445.79	25	200	180	43	6.27	MHA	N	9.0	1.0	9.5
8	Mohan kumar	5.5	5--10	M	2	T	Y	6	8	6	NV	N	Y	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	106	1.06	16.3	14.51	Y	N	Y	N	N	N	Y	N	N	7.1	7--9	5600	4.63	23.4	50.5	15.4	30	20.1	3.2	2	13.68	443	3.00	456.26	2.7	617	13.74	20	0.5	MHA	N	8.8	0.7	10.5
9	Dhanshika	2	1--5	F	3	PT	Y	6	0	6	NV	N	N	N	Atypicalfebrileillness	N	N	N	N	N	N	N	N	N	N	N	N	86.5	0.87	10.3	13.77	Y	N	Y	N	N	N	N	N	N	10.4	9--11	13600	4.74	31.3	66	22	33	18.2	3.6	1	29.31	394	6.92	423.4	37.4	270	11.97	41	6.31	NNA	N			
10	jayachandiran	2	1--5	M	3	T	Y	6	7	6	NV	N	N	N	Vfever	N	N	Y	N	N	Y	N	N	N	N	N	N	85	0.85	9.93	13.74	Y	N	Y	N	N	N	N	N	N	9.8	9--11	9300	4.33	29.7	68.6	22.7	33	14.2	2.79	0.3	92.64	342	21.32	434.42	17.3	274	8.82	15	1.25	NHA	P			
11	vidharth	2	1--5	M	2	T	Y	6	12	6	NV	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	91	0.91	13.6	16.42	Y	N	N	N	N	N	N	N	N	10	9--11	14400	5.76	38.5	66.8	20.4	31	15.1	2.72	0.1	79.62	410	16.25	490.02	13.7	194	14.78	13	0.24	NHA	N			
12	varnika	3	1--5	F	2	T	Y	6	7	6	NV	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	90	0.9	13	16.05	Y	N	N	N	N	N	N	N	N	8.1	7--9	8700	4.77	28.1	59	16.9	29	19.9	2.94	0.6	29.78	387	7.15	416.68	18.4	261	11.32	40	0.73	MHA	N	9.1	0.8	9.7
13	Hariharan	2	1--5	M	2	T	Y	6	7	7	NV	Y	N	N	loosestool	N	N	N	N	N	N	N	N	N	N	N	N	70	0.7	10	20.41	Y	N	N	N	N	N	N	N	N	10	9--11	27000	4.83	29.2	60.4	20.8	35	16.9	3.13	0.6	49.7	292	14.54	341.9	12	386	11.25	50	10.36	MHA	N	9.6	1.4	10.6
14	Sangeetha	8	5--10	F	3	T	Y	9	9	9	NV	N	N	N	Vfever	N	N	N	N	N	Y	N	N	N	N	N	N	180	1.8	17.8	5.49	Y	N	Y	N	N	N	N	N	N	9.8	9--11	11300	3.67	28.1	77.4	26.6	34	12	1.69	0.9	78.26	214	26.80	292.06	47	185	17.92	84	14.07	NNA	N			
15	Masthan	1.5	1--5	M	2	T	Y	6	7	7	NV	N	N	N	loosestool	N	N	N	N	N	N	N	N	N	N	N	N	78	0.78	8.7	14.30	Y	N	N	N	N	N	N	N	N	10.2	9--11	15800	5.33	35.2	66	19.1	29	22.7	3.96	0.5	49.44	375	11.64	424.63	10.9	635	10.96	1	0.75	NNA	N			
16	Santhosh Raj	2	1--5	M	3	T	Y	8	9	9	NV	Y	Y	N	loosestool	N	N	Y	N	N	N	N	N	N	N	N	N	75	0.75	9.2	16.36	Y	N	N	N	N	N	N	N	N	7.6	7--9	18600	4.87	25.4	52.2	55.6	30	19.6	4.8	2	20.94	435	4.59	455.81	2.2	269	5.38	10	6.53	MHA	N	7.9	1.0	8.5
17	Mohammed haroon	1.4	1--5	M	2	T	Y	3	7	3	NV	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	83	0.83	11.5	16.69	Y	N	N	N	N	N	N	N	N	9.8	9--11	32900	3.63	28.3	77.8	26.9	35	12.5	2.57	1.5	63.81	242	20.90	305.35	20.12	562	12.15	12	27.34	NNA	N			
18	Keerthirajan	3	1--5	M	2	T	Y	5	12	6	NV	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	97	0.97	16.1	17.13	Y	N	Y	N	Y	N	N	N	N	9.9	9--11	5700	4.13	30.6	74	22.1	30	12.9	2.47	0.8	24.64	306	7.45	330.93	62.8	325	17.24	62	9.88	NNA	N			
19	Rithikagowda	2.9	1--5	F	2	T	Y	1	2	6	NV	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	92	0.92	11.4	13.47	Y	N	Y	N	N	N	N	N	N	10.8	9--11	23600	4.59	32.1	70	23.5	34	15.3	2.65	1	16.93	299	5.36	315.73	76.1	315	17.04	28	77.19	NNA	N			
20	jonathan samuel	1	1--5	M	2	T	Y	6	0	7	NV	N	N	N	UTI	N	N	N	Y	N	N	N	N	N	N	N	N	75	0.75	8.48	15.08	Y	N	N	N	N	N	N	N	N	9	7--9	13800	4.22	21.3	64.6	21.3	33	14	6.8	0.6	20.38	429	4.53	449.76	16.9	211	22.43	68	57.03	NNA	N			
21	Gokulnath	2	1--5	M	2	T	Y	3	4	6	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	87	0.87	11.1	14.67	Y	N	Y	N	N	N	N	N	N	8.8	7--9	11800	4.29	26.5	61.8	20.5	33	17.6	2.75	0.4	20.99	301	6.52	321.99	65	325	17.82	25	224.47	NHA	N			
22	Chandreleka	2	1--5	F	3	T	Y	8	9	10	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	84	0.84	9.7	13.75	Y	N	Y	N	N	N	N	N	N	10	9--11	14500	4.64	31.2	67.1	22.9	34	19.4	5.82	1	47.94	440	9.83	487.89	48	173	13.68	8	0.27	NNA	N			
23	Goutam	9	5--10	M	2	T	Y	4	8	4	NV	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	120	1.2	25.8	17.92	Y	N	N	N	N	N	N	N	N	9.6	9--11	13400	4.46	29.3	65.7	21.6	33	12.1	4.86	0.6	14.77	433	3.30	447.77	3.7	483	5.27	21	1.25	NHA	N			
24	Harini	1.6	1--5	F	2	T	Y	6	6	7	NV	N	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	71	0.71	7.12	14.12	Y	N	N	N	N	Y	N	N	N	8.1	7--9	12900	4.71	28.2	59.9	17.3	29	15.3	5.5	1	22.07	526	4.03	548.07	2.12	275	9.75	12	3	MHA	N	9.2	0.8	10.0
25	Roshan	1	1--5	M	3	T	Y	6	0	6	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	70	0.7	8.6	17.55	Y	N	N	N	N	N	N	N	N	8.9	7--9	11100	3.67	26.7	72.8	24.3	33	12.9	3.5	5	54.38	386	12.34	440.57	2.19	492	17.82	11	0.44	MHA	N	10.6	1.0	11.9
26	Govardha harini	2.5	1--5	F	2	T	Y	0.1	5	6	NV	Y	Y	N	N	N	N	Y	N	N	Y	N	N	N	N	N	N	94	0.94	12.7	14.37	Y	N	N	N	N	N	N	N	N	8.6	7--9	13300	4.81	27.6	57.3	18.7	33	15.5	3.6	1	34.47	543	5.97	577.73	75	512	14.04	28	0.13	MHA	N	10.8	0.8	11.9
27	Yuvashree	4	1--5	F	2	T	Y	6	8	6	NV	Y	N	N	Vfever	N	N	Y	Y	Y	N	N	N	N	N	N	N	93	0.93	10.7	12.37	Y	N	Y	N	Y	N	N	N	N	9	7--9	11400	4.96	27.9	56.3	18.3	32	22.4	4.05	1	28.84	406	6.63	434.83	52	522	15.5	25	3.45	MHA	N	10.5	0.6	11.8
28	Varshini	6	5--10	F	2	PT	Y	2	2	6	NV	Y	N	N	Vfever	N	N	Y	Y	Y	N	N	N	N	N	N	N	102	1.02	14.4	13.84	Y	N	Y	N	N	Y	Y	Y	N	8.7	7--9	6500	3.91	26.9	68.9	22.4	32	27.5	2.2	0.5	155.4	46.5	76.96	201.96	220.1	317	5.16	100	15.84	MHA	N	9.0	0.6	9.4
29	Elavarasan	3	1--5	M	3	PT	Y	0.6	0.6	5	NV	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	95	0.95	12.3	13.63	Y	N	N	N	N	N	Y	Y	N	7.3	7--9	16500	5.09	24.4	47.9	14.3	30	20.1	3.43	1	31.3	454	6.45	485.6	4.5	915	12	20	10	NNA	N			
30	Yokesh	2	1--5	M	2	T	Y	4	7	4.5	NV	Y	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	78	0.78	11.5	18.90	Y	N	N	N	N	Y	N	N	N	10.9	9--11	7400	5.8	33.3	57.3	18.7	33	21.6	2.22	0.9	51.48	263	16.40	313.98	200.5	431	25.2	13	5.5	NNA	N			
31	maharaj	10	5--10	M	2	T	Y	6	0	7	NV	Y	N	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	127	1.27	30	18.60	Y	N	N	N	Y	Y	N	N	N	7.8	7--9	6300	4.44	27.5	62	20.1	32	27.3	5.5	1.6	41.64	415	9.12	456.44	21.1	559	25.2	25	6	NNA	N			
32	Thrishika	1.5	1--5	F	2	T	Y	3	4	5	v	Y	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	81	0.81	9	13.72	Y	N	N	N	Y	Y	N	N	N	6.7	>=7	9500	4.5	22.3	49.3	14.7	30	22.1	3.12	0.4	26.5	516	4.88	542.9	103	586	7.91	10	0.34	MHA	N	N	N	N
33	Suriya varman	1	1--5	M	2	T	Y	6	0	7	V	N	N	N	Vfever	N	N	N	N	N	N	N	N	N	N	N	N	68	0.68	7.45	16.11	Y	N	N	N	N	N	Y	Y	N	9.1	9--11	7300	3.87	27.8	72	23.6	33	16	2.22	0.8	33.75	308	9.89	341.25	199.3	463	13.42	35	1.38	NNA	N			
34	Divesh	1	1--5	M	2	T	Y	1.5	1.5	7	NV	N	Y	N	Dysentery	N	N	N	N	N	N	N	N	N	N	N	N	77	0.77	8.65	14.59	Y	N	N	N	N	N	N	N	N	7.8	7--9	12600	4.01	26.5	66	19.4	29	17.5	3.16	1	22	269	7.56	291.1	37.8	786	13.06	4	26.27	MHA	N	9.4	1.0	11.5
35	Pramila	6	5--10	F	3	T	Y	2.5	0	3	V	Y	N	N	Vfever	N	N	Y	N	N	N	N	N	N	N	N	N	100	1	16	16.00	Y	N	N	N	N	N	N	N	N	10.2	9--11	11800	4.03	31.2	77	25.2	33	11	2	0.4	25.12	248	9.20	273.02	52	425	4.25	37	63.71	NNA	N			
36	Jayashree	10	5--10	F	3	T	Y	6	6	6	NV	Y	Y	Y	N	N	N	N	N	Y	Y	N	N	N	N	N	N	131	1.31	27	15.73	Y	N	N	N	N	N	N	N	N	10.6	9--11	10500	4.06	32	79	26.2	33	12	3.02	1	83.56	269	23.68	352.86	59.3	343	5.58	28	1.38	NNA	N			
37	Swetha	9	5--10	F	2	T	Y	3.5	0	5	NV	N	N	N	N	N	N	N	Y	Y	Y	Y	N	N	Y	Y	N	138	1.38	33.2	17.43	Y	N	N	N	Y	N	N	N	N	10.7	9--11	7300	3.89	33.1	85	27.6	32	10.2	2.18	0.4	100.8	242	29.37	343.2	22	629	11.54	6	0.21	NNA	N			
38	Deenaprasandan	1	1--5	M	2	PT	Y	5	7	5	NV	N	N	N	Hypocalcemicseizures	N	N	N	N	N	N	N	N	N	N	N	N	72	0.72	7.5	14.47	Y	N	N	N	N	N	N	N	N	8.4	7--9	20000	4.51	28.6	63	18.7	30	12	4.68	0.8	34.79	328	9.60	362.49	115.2	540	20.31	76	320.3	MHA	N	9.6	0.6	11.5
39	Praveen	8	5--10	M	3	T	Y	6	6	6	NV	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	122	1.22	19.3	12.97	Y	N	N	N	N	N	N	N	N	10.6	9--11	11600	4.46	30.8	69.1	23.7	34	12.8	6.51	0.8	49.74	302	14.13	352.04	69.4	941	3.23	12	10	NNA	N			
40	Sairam	3	1--5	M	2	T	Y	6	6	6	V	N	Y	N	bronchopneumonia	N	N	N	Y	N	Y	N	N	N	N	N	N	90	0.9	10	12.35	Y	N	N	N	Y	N	N	N	N	10.3	9--11	8300	3.61	29.1	80.5	28.4	35	13.1	2.72	0.8	102.2	195	34.35	297.62	25	242	12.8	13	10	NNA	N			
41	Nilson	9	5--10	M	3	PT	Y	3	4	5	NV	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	121	1.21	18.7	12.77	Y	N	Y	N	N	N	N	N	N	9.6	9--11	7200	4.11	29.1	70.8	23.3	33	16	4.09	0.8	36.53	266	12.08	302.43	45.7	965	3.24	94	32.88	NNA	N			
42	Jaitheeasri	1.5	1--5	F	2	T	Y	3	4	5	NV	N	N	N	UTI	N	N	N	Y	N	Y	N	N	N	N	N	N	80	0.8	10	15.63	Y	N	N	N	N	Y	N	N	N	5.9	>=7	10500	3.19	18.4	58	18.5	32	11	6.88	0.4	47.76	365	11.57	412.96	11.6	451	9.53	74	10.87	MHA	N	8.3	0.3	9.0
43	kamesh	1	1--5	M	2	T	Y	6	0	7	NV	N	N	N	Atypicalfebrileillness	N	N	N	N	N	N	N	N	N	N	N	N	79	0.79	10.4	16.66	Y	N	Y	N	N	N	Y	N	N	8.4	7--9	7300	4.5	28	62	18.6	30	18.2	1.13	0.1	17.47	441	3.81	458.07	6.1	296	6.24	19	19.71	MHA	N	8.9	0.5	10.2
44	Monish	4	1--5	M	3	T	Y	5	7	5.5	V	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	93	0.93	11.5	13.26	Y	N	N	N	N	N	N	N	N	10.8	9--11	12200	4.15	31.8	76.6	26.1	34	14.2	3.25	2.2	50.64	343	12.85	393.94	18.2	524	17.55	17	1.44	NNA	N			
45	Gracy	6	5--10	F	2	T	Y	6	6	7	NV	Y	N	N	Bronchiolits	N	N	N	N	N	N	N	N	N	N	N	N	100	1	11.7	11.70	Y	N	N	N	N	N	N	N	N	9.6	9--11	13800	4.05	30.8	76	23.8	31	11	3.47	0.4	16.78	267	5.91	283.98	113.4	482	14.55	39	11	NHA	N			
46	Tamilselvan	10	5--10	M	3	T	Y	6	12	6	NV	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	121	1.21	18.6	12.70	Y	N	N	N	N	N	N	N	N	8.1	7--9	7900	4.78	26.7	55.8	17	30	18.9	3.45	0.8	46.73	417	10.08	463.42	1.6	###	8.46	24	0.43	NHA	N			
47	Naveen	5	1--5	M	2	T	Y	5	12	5.5	V	Y	Y	N	Walri	N	N	Y	Y	Y	N	N	N	N	N	N	N	108	1.08	16.9	14.63	Y	N	N	N	N	N	N	N	N	10.3	9--11	11800	4.38	35.4	80.9	28	35	13	3.25	1.6	33.12	363	8.37	395.72	42	392	12.5	49	11.57	NNA	N			
48	Nikilesh	3	1--5	M	2	T	Y	5	6	5	NV	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	94	0.94	13	14.71	Y	N	Y	N	N	N	N	N	N	8	7--9	4000	4.26	24.1	56.6	18	32	11	1.55	0.7	13.02	430	2.94	443.12	10.1	366	17.14	7	5	MHA	N	9.1	0.8	10.1
49	Kalaiselvi	2	1--5	F	2	T	Y	9	0	9	NV	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	78	0.78	7.2	11.83	Y	N	N	N	Y	Y	N	Y	Y	8.4	7--9	11200	4.31	26.6	61.7	19.5	32	18.4	3.05	0.8	14.77	435	3.28	449.77	194	###	6.35	23	90	MHA	P	9.5	1.0	11.2
50	Jayan	2	1--5	M	2	T	Y	1	2	5	NV	N	N	N	Atypicalfebrileillness	N	N	N	N	N	N	N	N	N	N	N	N	79.5	0.8	9	14.24	Y	N	N	N	N	N	N	N	N	10.5	9--11	7300	5.13	31.8	62	20.4	33	17.2	4.59	1	15.49	361	4.12	376.19	50	512	10.42	20	1.84	NNA	N			
51	Saran	10	5--10	M	2	PT	Y	6	10	6	V	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	70	0.7	6.87	14.02	Y	N	N	N	N	N	N	N	N	9.2	9--11	22700	4.57	32.6	71.4	24.2	34	14.3	6.75	1.5	55.85	423	11.67	478.63	10.1	130	17.34	11	0.31	NNA	P			
52	Dharun	2	1--5	M	2	PT	Y	7	0	7	V	Y	N	N	bronchopneumonia	N	N	N	N	N	N	N	N	N	N	N	N	82	0.82	8.3	12.34	Y	N	N	N	N	N	N	N	N	7.3	7--9	4500	3.2	24.1	75	22.7	30	19.1	14	0.2	14.42	240	5.67	254.12	11.1	575	12.21	80	27.59	NNA	N			
53	Rukesh	4	1--5	M	2	T	Y	7	12	7	NV	N	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	98.8	0.99	14	14.34	Y	N	Y	N	N	Y	N	N	N	10.1	9--11	12600	4.45	32.3	73	22.6	31	11.2	1.76	0.4	66.27	401	14.18	467.47	15.8	594	12.61	112	0.18	NNA	N			
54	Yuvaraj	9	5--10	M	2	T	Y	6	0	6	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	118	1.18	18.1	13.00	Y	N	N	N	N	N	N	N	N	9.8	9--11	5900	4.14	31.4	76	26.4	35	12.7	21.3	0.8	36.22	270	11.84	305.92	56	587	8.96	23	14.7	NNA	N			
55	Anisha	1	1--5	F	2	T	Y	5	5.5	5	V	Y	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	78	0.78	8.6	14.14	Y	N	N	N	N	N	N	N	N	10	9--11	12300	4.26	30.6	71.8	23.4	33	10.8	3.4	0.7	42.93	354	10.83	396.53	8.12	350	15.26	9	5.5	MHA	N	10.5	0.2	11.8

56	Saranya	10	5--10	F	4	T	Y	6	0	9	NV	Y	N	N	Vfever	Y	N	N	N	N	N	N	N	Y	N	N	N	146	1.46	30	14.07	Y	N	N	N	N	Y	N	N	N	10.8	9--11	8400	4	31.3	78.2	26.9	34	14	2.91	0.9	96.39	301	24.26	397.39	42	482	15.15	11	5	NNA	N			
57	Yokeshwari	9	5--10	F	2	T	Y	5	5	5	NV	Y	N	N	N	N	N	N	Y	N	Y	Y	Y	Y	Y	Y	Y	126	1.26	24	15.12	Y	N	Y	N	Y	Y	N	N	N	7.8	7--9	4800	2.88	22.7	79	27.3	35	18.9	4.44	0.4	44.81	194	18.75	239.01	25	161	16.5	4	5.3	MHA	N	9.1	1.0	9.8
58	Krish sudharsan	6	5--10	M	2	T	Y	6	0	6	NV	Y	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	107	1.07	14	12.14	Y	N	N	N	N	N	N	N	N	9.8	9--11	27200	3.62	29.6	81.9	26.9	33	13.7	2.46	0.8	52.16	251	17.20	303.26	32	738	17.82	31	14.8	NNA	N			
59	Johnjonathan	5	1--5	M	2	T	Y	6	7	6	NV	N	Y	Y	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	101	1.01	14.5	14.21	Y	N	Y	N	Y	N	N	N	Y	10.3	9--11	9300	4.36	33.9	78	23.7	31	12.2	3.07	0.6	62.23	292	17.55	354.49	139	760	6.1	29	12	NNA	N			
60	Prince Bhavya	5	1--5	F	1	T	Y	1	2	6	V	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	90	0.9	24	29.63	Y	N	N	N	N	N	N	N	N	10	9--11	5900	4.9	34.2	70	20.4	29	16.8	2.52	0.8	67.08	370	15.36	436.78	35.1	523	4.42	10	5.3	NNA	N			
61	Kamaleshwaran	1.5	1--5	M	2	T	Y	2	3	4	NV	N	N	N	Denguefever	N	N	N	N	N	N	Y	N	N	N	N	N	79	0.79	11	17.63	Y	N	N	N	N	N	N	Y	N	10.1	9--11	9700	4.37	33.4	76	23.2	30	11.4	1.25	1.6	34	287	10.61	320.6	12	452	12.32	22	40.7	NNA	N			
62	Ritesh	1	1--5	M	2	T	Y	5	7	5	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	73	0.73	8.5	15.95	Y	N	N	N	N	Y	N	Y	N	10.5	9--11	10600	4.74	31.7	67	22.7	34	16.1	5.1	0.5	52.76	534	9.00	586.47	86.9	217	12.37	27	2.21	NHA	N			
63	Yona	5	1--5	F	2	T	Y	4	4	6	NV	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	110	1.1	15	12.40	Y	N	Y	N	Y	Y	N	N	N	10.5	9--11	6900	4.43	33.4	75.4	24	32	16.2	4.33	0.6	32.99	284	10.39	317.39	29.4	188	18.53	43	12	NNA	N			
64	Mithra	1	1--5	F	2	T	Y	4	4	4	NV	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	67	0.67	7.2	16.04	Y	N	N	N	Y	Y	N	N	N	10	9--11	17000	3.8	30.9	81.3	26.6	33	13.5	63.02	1.1	52.7	169	23.74	222	32	234	17.82	11	26.3	NNA	N			
65	Kabilesh	4	1--5	M	2	T	Y	5	5	6	V	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	94	0.94	12	13.58	Y	N	N	N	Y	Y	N	N	N	10.4	9--11	9200	4.84	32.1	66.2	21.5	32	17.6	3.26	0.4	37.74	353	9.66	390.54	9.5	455	18.92	21	12	MHA	N	10.8	0.5	12.0
66	Mahalakshmi	10	5--10	F	2	T	Y	5	6	5	V	Y	N	N	Developmental delay	N	N	Y	N	Y	N	N	N	N	N	N	N	127	1.27	21	13.02	Y	N	N	N	Y	Y	N	N	N	6.4	>=7	5100	2.52	21.1	84	25.4	30	13.2	2.82	0.81	65.1	289	18.37	354.4	35.12	396	8.63	33	5.5	NNA	N			
67	Prabhu	10	5--10	M	2	T	Y	3	0	3	NV	N	Y	Y	N	N	N	N	Y	Y	N	N	N	N	N	N	N	138	1.38	20	10.50	Y	N	N	N	N	N	N	Y	N	9.7	9--11	4200	4.66	33.7	72.4	20.8	29	14.2	1.43	0.9	53.11	235	18.45	287.81	554	352	7.19	67	75.8	NHA	N			
68	Haroon raship	3	1--5	M	2	T	Y	6	6	6	NV	Y	N	N	Malaria	N	N	N	Y	N	Y	N	N	N	N	N	N	89	0.89	10.4	13.13	Y	N	N	N	N	Y	N	N	N	9.2	9--11	7800	5.73	35	60.1	16	27	12.2	2.92	0.5	24.18	395	5.77	419.18	98	572	7.5	18	12.9	MHA	N	9.0	2.0	10.2
69	Hariharan	1	1--5	M	3	T	Y	1	3	8	V	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	78	0.78	8.5	13.97	Y	N	N	N	N	N	N	N	N	8.3	7--9	7300	4.25	27.6	65	19.5	30	15.3	1.91	0.5	27.78	405	6.42	432.58	12.95	356	19.5	31	5.8	MHA	N	8.9	2.0	9.7
70	Karthik	10	5--10	M	3	T	Y	5	7	5	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	135	1.35	30	16.46	Y	N	N	N	N	N	N	N	N	10.8	9--11	7300	4.54	38.4	84.5	24	28	11.7	3.36	0.8	74.53	246	23.27	320.23	18.2	452	13.15	27	5	NNA	N			
71	Harish kumar	3	1--5	M	2	T	Y	3	3	7	NV	Y	Y	N	simplefebrileseizures	N	N	N	N	N	N	N	N	N	N	N	N	86.5	0.87	10.5	14.03	Y	N	Y	N	N	Y	N	N	N	8.5	7--9	15800	4.73	28	61	17.9	29	10.2	2.5	0.89	35.37	453	7.24	488.67	9.1	473	11.66	63	9.6	MHA	N	N	N	N
72	Bharathkumar	9	5--10	M	2	T	Y	6	6	6	NV	Y	N	N	Appendicectomy	N	N	N	N	Y	N	N	N	N	N	N	N	135	1.35	30	16.46	Y	N	N	N	N	N	N	N	N	10.3	9--11	6000	4.33	34.2	78.9	23.7	30	12.2	2.87	0.9	69.81	262	21.02	332.11	66.3	253	4.72	55	9.9	NNA	N			
73	Deepak	10	5--10	M	2	T	Y	3	4	6	NV	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	127	1.27	25.3	15.67	Y	N	N	N	N	N	N	N	N	10.7	9--11	13100	4.88	37.8	77.5	22	82	13.4	2.52	0.5	53.92	371	12.69	425.02	15.9	453	983	9	6.6	NHA	N			
74	Dharshini	8	5--10	F	3	PT	Y	5	11	5	NV	N	N	Y	Vfever	N	N	N	N	N	N	N	N	N	N	N	N	115	1.15	19	14.37	Y	N	N	N	N	Y	N	N	N	10	9--11	6000	4.84	35.4	73.2	20.7	28	13.9	2.24	0.5	70.64	320	18.07	390.94	55.5	711	6.68	21	30.8	NNA	N			
75	Joaatha leoma	3	1--5	F	2	T	Y	4	5	3	NV	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	85	0.85	8.4	11.63	Y	N	Y	N	Y	N	N	N	N	9.2	9--11	9800	4.32	32.4	75	21.4	28	14	2.5	1.2	36.62	233	13.56	270.02	1010	724	25.2	50	43.2	NNA	N			
76	Sandhya	6	5--10	F	2	T	Y	6	8	6	NV	Y	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	123	1.23	39.3	25.89	Y	N	N	N	N	N	N	N	N	10.3	9--11	1300	5.16	40.8	79	23.9	30	12.2	4	1.1	68.71	341	16.75	410.11	25.1	343	7.72	18	8.4	NNA	N			
77	Emancy	2	1--5	F	2	T	Y	5	10	5.5	NV	Y	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	80.7	0.81	9.6	14.74	Y	N	N	N	Y	N	N	N	Y	9.4	9--11	12200	4.39	32.5	73.9	21.4	29	13.4	6.19	0.8	39.35	308	11.34	346.85	25.2	285	11.45	61	20.7	MHA	N	10.6	0.8	11.2
78	Bharathkumar	2	1--5	M	2	T	Y	6	24	6	NV	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	79	0.79	8.5	13.62	Y	N	N	N	Y	Y	N	N	N	9.8	9--11	12900	4.5	32.2	68.1	20.8	31	17.4	4.16	0.6	33.47	292	10.28	325.67	58	505	12.5	44	90	MHA	N	10.2	1.0	11.5
79	Priyanka	10	5--10	F	2	T	N	0.5	1	3	NV	Y	Y	N	Vfever	N	N	N	N	Y	Y	N	N	N	N	N	N	136	1.36	24	12.98	Y	N	N	N	N	N	N	N	N	10.5	9--11	9800	4.04	35	8.7	25.9	30	12.1	1.95	0.7	56.99	337	14.46	393.99	28.2	227	12.5	5	5	NNA	N			
80	Sai sugeesh	1	1--5	M	2	T	Y	6	6	6	NV	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	67	0.67	8.3	18.49	Y	N	N	N	Y	N	N	N	N	9	7--9	8300	4.39	28.9	65.7	20.5	31	14.8	6.43	0.5	34.17	390	8.07	423.67	52	422	12.5	22	9.8	MHA	N	10.8	0.2	11.6
81	Thrisha	1	1--5	F	2	T	Y	5	0	5	NV	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	75	0.75	7	12.44	Y	N	N	N	Y	Y	N	N	N	10.7	9--11	10800	4.42	35	79	24.1	31	14.4	2.9	0.6	52.95	221	19.30	274.35	65	425	12.5	25	14	NNA	N			
82	Litheeswaran	4.5	1--5	M	2	T	Y	4	4	4	NV	N	Y	Y	N	N	N	Y	Y	Y	N	N	N	N	N	N	N	108	1.08	17.5	15.00	Y	N	Y	N	Y	Y	N	N	N	10.4	9--11	12800	4.53	34.5	76	23	30	13.9	4.5	0.8	20.14	335	5.67	355.14	45.8	222	12.89	63	18	NNA	N			
83	B/O Kalavathy	1	1--5	M	2	T	Y	3	3	6	NV	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	75	0.75	9.5	16.89	Y	N	N	N	N	N	N	N	N	7.7	7--9	9800	3.9	23.5	63.7	19.7	31	12.8	6.03	1.75	31.92	320	9.07	351.95	31.9	293	22	12	2.1	MHA	N	8.2	2.0	8.6
84	Bharathy	2	1--5	M	2	T	Y	7	8	7	NV	Y	N	N	N	Y	N	Y	Y	N	Y	N	N	N	N	N	N	93	0.93	11.4	13.18	Y	N	Y	N	N	N	N	N	N	10	9--11	10500	4.25	34.2	80.6	23.9	30	13.9	4.26	0.8	68.67	275	19.96	343.97	32.5	572	18.25	30	10.3	NNA	N			
85	Abishek	1	1--5	M	3	PT	Y	6	7	6	V	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	67	0.67	6	13.37	Y	N	N	N	N	N	N	N	Y	9.3	9--11	6800	4.97	32.4	65	18.7	29	16.5	2.68	0.5	25.97	591	4.21	616.97	7.86	756	20.24	35	22	MHA	N	9.8	0.5	11.0
86	Thanigaivel	1.5	1--5	M	2	PT	Y	3	3	5	V	Y	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	84.5	0.85	9.6	13.44	Y	N	N	N	Y	Y	N	N	N	6.6	>=7	12200	5.94	25.6	43	11.2	26	16.9	6.5	0.3	19.38	482	3.87	500.98	15.1	678	21.18	4	10	MHA	N	5.7	1.2	7.2
87	Prashanth	6	5--10	M	3	T	Y	3	0	3	V	N	N	N	Tb abd	Y	Y	Y	Y	N	N	N	N	N	N	N	N	108	1.08	13.7	11.75	Y	N	Y	N	Y	Y	N	Y	N	10.1	9--11	4900	4.51	33.6	74	22.3	30	16.9	1.48	0.6	71.28	261	21.45	332.38	36	476	16.75	14	50.5	NHA	N			
88	Pushpak	5	1--5	M	3	T	Y	6	6	12	NV	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	103	1.03	16	15.08	Y	N	Y	N	Y	Y	N	N	Y	10.8	9--11	9500	4.16	33.2	79.7	25.9	33	13.6	1.86	1.5	35.4	222	13.73	257.82	131.8	452	21.9	21	24.4	NNA	N			
89	Deepika	10	5--10	F	2	T	Y	1	1	3	NV	Y	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	148	1.48	27.5	12.55	Y	N	N	N	N	N	Y	N	Y	10.3	9--11	8000	4.32	36.5	84.5	23.8	28	12.4	2.24	1.2	12.83	254	4.81	266.93	1139	772	13.35	12	90	NNA	N			
90	Sanjay	3	1--5	M	3	T	Y	1	2	6	NV	N	N	N	Seizure disorder	N	N	Y	Y	N	N	N	N	N	N	N	N	106	1.06	16	14.24	Y	N	N	N	N	Y	N	N	N	10.2	9--11	7000	4.32	38.5	84.5	28.8	29	10.4	2.16	0.5	56.4	386	12.74	442.6	13.1	375	10.9	8	24	NNA	N			
91	Vishal	6	5--10	M	3	T	Y	4	5.5	4	NV	N	Y	N	bronchopneumonia	N	N	Y	Y	Y	N	N	N	N	N	N	N	104	1.04	14.8	13.68	Y	N	N	N	N	Y	N	N	N	9.8	9--11	11400	4.02	31.9	79	24.4	31	12.4	4.96	0.9	47.6	264	15.27	311.8	37	684	6.33	11	21	NNA	N			
92	Vimalraj	3	1--5	M	4	PT	Y	1	1	8	NV	Y	Y	N	simplefebrileseizures	N	N	Y	Y	N	Y	N	N	N	N	N	N	81	0.81	8.2	12.50	Y	N	N	N	N	Y	Y	Y	N	10.8	9--11	9600	4.52	36.4	80	24.1	30	12	2.97	0.7	24.87	293	7.83	317.67	51.3	492	11.85	22	58.9	NNA	N			
93	Kamalesh	10	5--10	M	3	T	Y	8	12	8	NV	N	Y	Y	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	135	1.35	22.5	12.35	Y	N	Y	N	Y	Y	N	N	N	6.2	>=7	10000	3.77	20.4	56.6	16.2	29	20.7	3.97	1	15.14	412	3.54	427.14	66	452	13.8	18	0.28	MHA	N	5.3	1.4	9.3
94	Elavarasan	3	1--5	M	3	PT	Y	8	9	8	NV	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	105	1.05	12.3	11.16	Y	N	N	N	N	N	Y	Y	N	7.3	7--9	16500	5.09	24.4	47.9	14.3	29	20.1	2.43	0.8	31.3	455	6.44	485.8	4.5	915	12	10	7	MHA	N	8.0	2.0	9.1
95	Rithika	2.5	1--5	F	2	T	Y	8	8	8	NV	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N	N	76	0.76	11.3	19.56	Y	N	N	N	N	N	N	Y	N	9.2	9--11	14500	4.46	29.7	6.7	19.6	30	12.8	3.19	0.5	22	408	5.12	429.9	65	452	14.2	15	4	MHA	N	9.8	1.0	11.5
96	Maana sree	1	1--5	F	2	T	Y	6	0	6	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	73	0.73	7.6	14.26	Y	N	Y	N	Y	N	N	N	N	8.7	7--9	18600	4.39	28	64	19.7	31	13.1	2.9	0.6	23.41	440	5.05	463.11	72	522	13.04	12	6.5	MHA	N	9.6	1.0	10.5
97	Ranjith	1.5	1--5	M	2	T	Y	7	12	7	NV	N	N	N	Vfever	N	N	N	N	N	N	N	N	N	N	N	N	85	0.85	9.1	12.60	Y	N	N	N	N	N	N	N	N	7.9	7--9	8200	4.52	27.2	65	22.2	28	13.4	4.3	0.6	27	452	5.64	479	10	652	20.2	6	12	MHA	N	9.4	2.0	11.5
98	Harini	10	5--10	F	2	T	Y	4	4	6	NV	Y	Y	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	132	1.32	25.6	14.69	Y	N	N	N	N	N	Y	Y	N	9.8	9--11	8900	4.46	29.2	65.5	21.9	33	16.5	3.26	0.8	15.8	380	3.99	395.5	2.8	743	5.4	21	0.13	MHA	N	11.6	0.8	12.0
99	Monika	1	1--5	F	3	T	Y	6	6	6	NV	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	94	0.94	8.4	9.51	Y	N	N	N	N	N	N	N	N	9.7	9--11	11500	4.2	28.5	67.9	23	34	15.4	3.44	0.7	31.46	412	7.09	443.46	57.8	228	9.29	22	4.02	NNA	N			
100	Jagatheesh	2	1--5	M	2	T	Y	3	3	7	NV	Y	N	N	Vfever	Y	Y	N	Y	N	N	N	N	N	N	N	N	91	0.91	14	16.91	Y	N	N	N	N	N	N	N	N	10.4	9--11	10000	4.8	34.2	75.4	21.6	29	16	3.2	0.7	56.12	390	12.59	445.62	28.1	695	10.72	2	12	NNA	N			
101	Mahathi	1.5	1--5	F	2	T	Y	6	6	6	NV	N	N	N	Vfever	N	N	N	N	N	N	N	N	N	N	N	N	76	0.76	7.8	13.50	Y	N	N	N	Y	Y	N	N	N	10.8	9--11	15500	4.69	37.7	80	23.3	29	12	1.87	0.8	48	317	13.16	364.7	33.4	625	11.83	19	9.4	NNA	N			
102	Sasikumar	2	1--5	M	3	T	Y	4	4	4	NV	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	127	1.27	25.3	15.67	Y	N	N	N	Y	Y	N	N	N	5	>=7	8600	3.47	16	46	11.6	25	12.2	4.28	1.1	17.13	500	3.32	516.63	2.9	834	6.29	9	5	MHA	N	6.7	2.0	7.5
103	Shanthi	10	5--10	F	3	T	Y	0	1	5	NV	N	N	N	simplefebrileseizures	Y	N	N	Y	Y	N	N	N	N	N	N	N	118	1.18	25.5	18.31	Y	N	N	N	Y	N	N	N	N	10.1	9--11	8000	3.75	32.9	88.8	26.8	31	11.5	1.46	1.7	110.4	120	47.95	230.34	54.5	505	4.54	23	5	NNA	N			
104	Karthikrraj	10	5--10	M	2	T	Y	6	0	6	NV	Y	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	142	1.42	33.6	16.66	Y	N	N	N	N	N	N	N	N	9	7--9	10500	3.76	29.9	80	23.9	30	12.8	3.3	0.4	39.27	243	13.93	281.87	132.8	325	3.47	85	32.3	NNA	N			
105	Shamini	2	1--5	F	3	T	Y	5	6	5	NV	Y	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	78	0.78	9	14.79	Y	N	N	N	Y	Y	N	N	Y	8.2	7--9	11100	4.2	27.9	66	19.5	29	12.5	2.5	1.1	20.36	380	5.09	400.06	18.6	485	7.59	15	12.3	NHA	N			
106	Bhakyalakshmi	8	5--10	F	2	T	Y	5	5	5	NV	Y	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	122	1.22	18.6	12.50	Y	N	N	N	Y	N	N	N	N	10.8	9--11	9700	4.76	40.6	85	26.1	31	12.8	3.98	0.4	80.74	350	18.74	430.74	28.7	247	4.76	12	6.6	NNA	N			
107	Swathi	8	5--10	F	2	T	Y	5	5	5	NV	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	122	1.22	24.5	16.46	Y	N	N	N	N	Y	N	N	N	10.5	9--11	6700	4.75	37.9	80	24.2	30	14	3.8	0.8	90.2	218	29.25	308.4	30.3	476	8.9	9	5	NNA	N			
108	Suryaprakash	8	5--10	M	3	T	Y	3	0	3	NV	Y	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	124	1.24	20	13.01	Y	N	Y	N	N	N	N	N	N	10.4	9--11	4300	4.41	34.1	77	23.7	31	12.6	1.82	0.9	80.45	233	25.63	313.85	55	321	12.4	5	5.3	NNA	N			
109	Sivasubavishnu	2	1--5	M	3	T	Y	5	0	5	NV	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	80	0.8	9	14.06	Y	N	N	N	N	N	N	N	N	9.2	9--11	6900	3.92	29.3	74	23.1	31	11.2	2.67	0.8	28.73	341	7.78	369.43	25.2	267	14.8	12	19.5	NHA	N			
110	Rabitha	10	5--10	F	3	T	Y	4	0	4	NV	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	131	1.31	33.6	19.58	Y	N	N	N	N	Y	N	N	N	10.5	9--11	8300	4.78	35.2	73.6	24.8	34	14.6	3.1	1	57.24	439	11.54	495.91	4.4	274	7.96	12	0.28	NNA	N			
111	Dhanyasree	1.5	1--5	F	2	T	Y	6	18	6	NV	N	Y	N	N	N	N	Y	N	N	Y	N	N	N	N	N	N	83	0.83	9.6	13.94	Y	N	N	N	Y	Y	N	N	N	10.2	9--11	10400	4.25	32.1	75.5	24	32	17.1	5.2	0.6	45.76	306	13.02	351.36	31.1	431	14.52	5	6	NNA	N			
112	Vidyut	2	1--5	M	2	T	Y	7	12	7	V	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	84	0.84	18.2	25.79	Y	N	N	N	N	N	N	N	N	9.2	9--11	5800	3.9	28.4	73	23.5	32	11.2	2.95	0.5	32	410	7.24	442	23.1	156	13.79	11	6.77	MHA	N	10.6	0.5	11.8
113	Mithun	4	1--5	M	2	T	Y	3	3	3	V	N	Y	Y	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	103	1.03	15.9	14.99	Y	N	N	N	Y	N	N	N	N	6.5	>=7	16400	5.45	24.7	45	12	27	16.2	5.75	1.8	10.1	587	1.69	597.1	2.6	461	12.21	15	1.58	MHA	N	7.8	1.0	10.2
114	Samreen fathima	5.5	5--10	F	2	T	Y	1	1	12	NV	Y	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	102	1.02	16	15.38	Y	N	N	N	Y	Y	N	N	N	9.1	9--11	8300	5.6	29.5	52.7	16.2	31	16.7	2.36	5	74.35	259	22.32	333.11	39.9	670	12.39	11	0.22	MHA	N	10.6	0.8	12.1

115	Divakar	6	5--10	M	3	T	Y	4	4	6	V	N	Y	Y	N	N	N	N	Y	N	Y	N	N	N	Y	Y	N	112	1.12	17.8	14.16	Y	N	N	N	Y	N	N	N	N	10	9--11	11100	4.41	30.6	69.4	22.8	33	15.4	3.44	0.8	20.11	382	5.00	402.01	4.5	436	8.15	17	0.43	MHA	N	10.4	0.5	11.7
116	Keerthirajan	7	5--10	F	2	T	Y	4	6	4	NV	Y	N	Y	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	108	1.08	18	15.43	Y	N	N	N	N	N	Y	Y	N	10.6	9--11	6500	4.29	34.3	80	24.7	31	12.9	2.33	1	50.21	428	10.50	478.21	8.2	172	28.2	14	18	NNA	N			
117	Subramani	10	5--10	M	3	T	Y	10	0	11	NV	N	Y	N	N	N	N	Y	Y	N	Y	Y	N	N	N	Y	N	131	1.31	24.3	14.16	Y	N	N	N	Y	Y	N	N	N	4.7	>=7	5800	3.88	18.2	46.8	12	26	26.1	3.5	0.9	19.43	486	3.84	505.66	1.3	295	13.3	41	0.25	MHA	N	10.5	1.3	11.9
118	Santhosh	5	1--5	M	2	T	Y	6	12	6	V	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	110	1.1	18.4	15.17	Y	N	N	N	N	N	N	N	N	10	9--11	12800	4.75	30.1	63	18.9	298	18	5.25	0.5	26.17	457	5.41	483.53	19	595	16.75	7	1.78	MHA	N	7.4	1.2	8.0
119	Pradeep	3.5	1--5	M	2	T	Y	5	6	5.5	NV	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	95.5	0.96	11.5	12.61	Y	N	N	N	Y	N	Y	Y	N	8.6	7--9	13000	4.42	26.3	59.5	19.4	33	16.6	3.13	1	38.2	384	9.05	422.2	12	482	14.8	12	6	NNA	N			
120	Narendar	5	1--5	M	2	T	Y	6	6	7	V	N	Y	Y	Vfever	N	N	N	N	Y	Y	N	N	N	N	N	N	101	1.01	14	13.72	Y	N	Y	N	N	N	Y	N	Y	10	9--11	5400	3.61	29	80.4	27.4	34	12.2	3.66	1	57.82	272	17.54	329.62	483	380	17.25	55	12	NNA	N			
121	Sahana	4	1--5	F	2	T	Y	5	5	5.5	NV	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	87	0.87	11	14.53	Y	N	N	N	Y	N	N	N	N	10.2	9--11	11900	4.59	30.3	76.8	20.2	26	14.6	2.74	0.4	53.92	371	12.69	425.02	71.2	431	10.65	36	8	NHA	N			
122	Shaheel	5	1--5	M	2	T	Y	6	6	6	NV	N	N	N	N	N	N	Y	Y	Y	Y	N	N	N	N	N	N	104	1.04	11.2	10.36	Y	N	N	N	N	N	N	N	N	10.6	9--11	8100	4.33	34.4	79.3	25.3	32	13.5	3.5	0.6	70.15	262	21.10	332.45	5	187	5.72	10	10.2	NNA	N			
123	Karthik	7	5--10	M	2	T	Y	6	0	6	NV	N	Y	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	109	1.09	16.3	13.72	Y	N	Y	N	N	N	N	Y	N	9.5	9--11	8500	4.33	30.5	70.5	22	31	18.5	5.73	1.5	37.46	306	10.90	343.66	62.9	360	12.82	40	12	MHA	N	10.6	2.0	11.4
124	Thangaraj	10	5--10	M	3	T	Y	1.5	1.5	3	NV	Y	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	128	1.28	22	13.43	Y	N	Y	N	N	N	N	N	N	9.9	9--11	6300	4.36	34.6	79.4	22.8	29	12.4	3.5	0.2	74.53	246	23.27	320.23	230.1	792	14.82	49	5	NNA	N			
125	Arulraj	2.5	1--5	M	3	T	Y	5	24	5	NV	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	91	0.91	12.1	14.62	Y	N	N	N	N	Y	N	N	N	9.7	9--11	10200	3.99	28.4	71.2	24.2	34	15.6	3.17	1	26.86	432	5.85	458.86	4.85	300	13.64	28	15.8	NNA	N			
126	Abishek	2.5	1--5	M	3	PT	Y	7	12	7	NV	Y	N	N	N	Y	N	N	Y	N	Y	N	N	N	N	N	N	83	0.83	10.2	14.81	Y	N	Y	N	N	Y	N	N	Y	7.4	7--9	20000	4.2	43	68	11.6	17	16.3	3.64	0.7	43.69	418	9.46	461.99	12.2	714	25.2	22	8.7	NNA	N			
127	Liza mondal	2	1--5	F	2	PT	Y	6.5	6.5	7	NV	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	84	0.84	10.2	14.46	Y	N	N	N	Y	Y	N	N	N	10	9--11	8700	5.06	32.8	65	19.8	31	16.3	3.43	0.6	71.15	320	18.21	390.65	8.6	852	16.4	25	1.25	NNA	N			
128	Vishnu	5	1--5	M	2	T	Y	6	30	6	NV	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	118	1.18	18	12.93	Y	N	N	N	N	N	N	N	N	10.1	9--11	8900	4.09	30.9	76	24.6	33	16.2	2.76	1	74.52	245	23.34	319.32	19.6	402	9.1	16	3	NNA	N			
129	Varunikasri	2.2	1--5	F	2	T	Y	6	6	6	NV	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	81	0.81	10	15.24	Y	N	N	N	N	N	N	N	N	9	7--9	10400	5.03	30.4	16.4	19.7	33	16	3.63	0.4	30.7	492	5.87	522.72	32	###	11.24	54	1.06	MHA	N	N	N	N
130	Hansika	1	1--5	F	2	PT	Y	3	0	3	NV	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	72	0.72	8	15.43	Y	N	N	N	Y	Y	N	N	N	7.9	7--9	7800	4.27	26.8	63	18.5	30	12.8	2.8	0.1	18.3	397	4.40	415.7	13.1	18	4.5	16	22	MHA	N	7.9	0.5	7.9
131	Balavignseh	8	5--10	M	2	T	Y	3	12	3	NV	N	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	102	1.02	18.6	17.88	Y	N	Y	N	N	N	N	N	N	10.1	9--11	8400	3.81	30.8	80.8	26.6	33	13.3	2.8	0.5	53.86	368	12.76	421.98	178	727	11.18	66	11	NNA	N			
132	Raagav	2.3	1--5	M	3	T	Y	3	3	6	NV	N	Y	Y	Walri	N	N	N	N	N	Y	N	N	N	N	N	N	86	0.86	11.5	15.55	Y	N	N	Y	Y	N	N	N	N	9.4	9--11	29100	5.19	31.2	60	18.1	30	16.3	4.14	0.3	53.96	411	11.61	464.77	12.6	740	3.8	13	0.6	NNA	N			
133	Sharon roselin	1.2	1--5	F	2	T	Y	6	6	6	NV	N	Y	Y	loosestool	N	N	Y	Y	N	Y	N	N	N	N	N	N	80	0.8	9	14.06	Y	N	N	N	Y	Y	N	N	N	7.9	7--9	11900	8.3	88.2	66	18.7	28	16.2	4.7	0.5	35.12	483	6.78	518.07	5.9	496	12.2	14	13	MHA	N	8.6	1.5	9.2
134	Swetha	10	5--10	F	3	T	Y	6	6	6	NV	N	N	N	N	N	N	N	Y	Y	Y	Y	N	N	N	N	N	138	1.38	25.8	13.55	Y	N	Y	N	N	N	Y	N	Y	7.2	7--9	4000	3.79	23.9	63.1	19.1	30	22.2	0.75	1	29.7	490	5.71	519.82	20.2	433	15.82	57	2	MHA	N	N	N	N
135	Raakesh	4	1--5	M	3	T	Y	6	6	7	NV	Y	N	N	bronchopne umonia	N	N	Y	Y	Y	Y	N	N	N	N	N	N	92	0.92	14.3	16.90	Y	N	N	N	Y	Y	N	N	Y	10.2	9--11	10200	4.22	34.8	82.7	26.4	32	13	2.28	0.6	19.8	206	8.78	225.6	1000	282	14.4	28	14	NNA	N			
136	Sathya	10	5--10	F	2	T	Y	5	0	5	V	Y	Y	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	N	116	1.16	19.5	14.49	Y	N	N	N	N	Y	Y	Y	N	6	>=7	12200	3.08	16	52.1	13.6	26	25.5	5.29	2.4	20.8	225	8.46	245.92	4.12	178	15.7	24	18	MHA	N	6.7	2.0	8.1
137	Samuel	1.3	1--5	M	2	T	Y	3	3	8	V	N	Y	N	Breathholdi ngspell	N	N	N	Y	N	N	N	N	N	N	N	N	74	0.74	10.4	18.99	Y	N	N	N	Y	N	N	N	N	7.7	7--9	9600	4.28	26.8	63	18	29	18.2	4.52	0.6	12.82	534	2.35	546.63	4.39	390	12.42	9	6	NNA	N			
138	Abishek	1.9	1--5	M	2	T	Y	3	4	4	NV	N	Y	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	82	0.82	10.2	15.17	Y	N	Y	N	N	Y	N	N	N	8.6	7--9	11000	4.53	30.3	67	19	28	17.8	4.12	1	27.78	406	6.41	433.38	12.89	482	11.4	31	6	MHA	N	9.8	0.5	11.8
139	Mohana	6.3	5--10	F	3	T	Y	6	6	6	NV	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	108	1.08	14.8	12.69	Y	N	Y	N	N	Y	N	Y	N	9.9	9--11	15200	4	31.6	79	24.7	31	18.5	2.78	0.5	74.35	253	22.75	326.85	65.5	276	10.39	12	18	NNA	N			
140	Karthik	2	1--5	M	2	T	Y	8	0	9	V	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	73	0.73	8.3	15.58	Y	N	N	N	Y	Y	N	N	N	9.9	9--11	15000	5.02	34	68	19.7	29	13.5	4.7	0.5	37.46	308	10.84	345.66	19.4	177	12.8	9	5	NHA	N			
141	Ramya	10	5--10	F	3	T	Y	7	7	8	NV	N	Y	Y	N	N	N	N	Y	N	Y	N	N	N	N	N	N	143	1.43	34	16.63	Y	N	N	N	N	N	N	N	N	9.3	9--11	10200	3.78	31.7	84	24.6	29	17.8	3.76	0.4	50.21	429	10.48	479.21	7.72	172	28.2	15	18	NHA	N			
142	Amit kumar	4	1--5	M	3	T	Y	5	6	5	NV	Y	N	N	N	N	N	Y	Y	Y	Y	N	N	N	N	N	N	105	1.05	14.7	13.32	Y	N	N	N	Y	Y	Y	Y	Y	8.6	7--9	8700	5.75	32	56	14.9	27	19.2	2.57	0.5	17.35	261	6.23	278.45	172	303	10.2	66	58.2	MHA	N	9.5	0.8	10.8
143	Smruti sikta veura	2.5	1--5	F	2	PT	Y	6	18	6	NV	Y	N	N	N	N	N	Y	Y	N	Y	N	N	N	N	N	N	89	0.89	10.9	13.76	Y	N	Y	N	N	Y	Y	Y	Y	10.2	9--11	9300	4.46	31.9	71.5	22.9	32	13.7	2.16	0.8	34.63	217	13.76	251.73	180	834	12.2	54	28.4	NNA	N			
144	Dhanasri	1.2	1--5	F	2	T	Y	5	5	8	V	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	71	0.71	16	31.74	Y	N	N	N	Y	N	N	N	N	10.9	9--11	6300	4.4	34.1	77.6	24.9	32	13.5	2.9	1	63.18	379	14.28	442.44	68.4	330	14.42	11	10	NNA	N			
145	Vishwa	1	1--5	M	2	T	Y	6	8	6	V	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	N	70	0.7	8.07	16.47	Y	N	N	N	N	N	N	N	Y	8.5	7--9	7800	4.51	26.9	59.6	18.9	32	18.9	4.75	0.7	26.17	457	5.41	483.53	6.45	214	16.75	24	7.2	MHA	N	9.5	0.5	11.2
146	Hendric paul	4	1--5	M	2	T	Y	4	4	6	NV	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	105	1.05	13.4	12.15	Y	N	N	N	Y	Y	N	N	N	8.8	7--9	8200	3.88	28.7	74	22.8	31	13.8	2.48	0.5	18.26	395	4.42	413.26	14.8	364	14.58	10	5.7	MHA	N	N	N	N
147	Abinaya	10	5--10	F	2	T	Y	1	2	5	NV	Y	N	N	N	N	N	N	Y	Y	Y	N	N	N	N	N	N	132	1.32	22.9	13.14	Y	N	N	N	N	N	N	N	Y	10.8	9--11	8600	4.36	36.9	84	24.6	29	11.3	3.39	1	52.76	533	9.01	585.58	130	303	12.37	27	26.4	NNA	N			
148	Krithika	2	1--5	F	2	T	Y	3	18	3	NV	N	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	80	0.8	9.1	14.22	Y	N	N	N	Y	Y	N	N	N	7.5	7--9	13600	4.69	26.5	56	15.9	28	12	5.42	2	15.73	426	3.56	442.03	20.6	427	6.8	12	6.26	MHA	P	8.2	1.5	9.6
149	Maariselvam	6	5--10	M	4	PT	Y	6	12	6	NV	N	Y	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	107	1.07	14.2	12.41	Y	N	N	N	N	N	N	N	N	10.6	9--11	14800	4.7	36.3	77	23.3	30	12.3	2.8	0.5	58.21	352	14.19	410.33	22.7	588	17.82	25	8	NNA	N			
150	John jeslin	1.8	1--5	F	2	T	Y	5	6	6	NV	N	N	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	79	0.79	9.5	15.22	Y	N	N	N	Y	N	Y	Y	N	8.8	7--9	12800	4.23	29.3	69	20.7	30	18.2	4.25	0.8	39.16	282	12.19	321.28	12.2	516	15.65	8	12	NNA	N			

Annexures

ANNEXURE-I

PROFORMA

- Name : Insurance No -
- Age :
- Sex :
- Informant :
- Reliability :
- Address :

- Socio –economic Details

Mother’s education	
Father’s education	
Annual Income	
Occupation	

- Socio –economic Status :
- Housing :

- History

Term / preterm	
Duration of breast feeding	
Timing of introduction of cow's milk / formula feeds	
Introduction of complementary feeds	
Diet	Veg ☐ Non-Veg ☐
	H/o Intake of red meat, liver and greens
Worm infestation	Yes ☐ No ☐
H/O Pica	Yes ☐ No ☐
H/O Pagophagia	Yes ☐ No ☐
H/O treatment for any infections in the past	Yes ☐ No ☐
H/O of GI bleed	Yes ☐ No ☐
Family history of hemolytic anemia	Yes ☐ No ☐

- Presenting complaints with duration:

  - H/o Irritability Yes ☐
    No ☐
  - H/o Lethargy Yes ☐
    No ☐

- H/o Poor school performance Yes ☐
  No ☐
- H/o Easy fatigability Yes ☐
  No ☐
- H/o Breathlessness Yes ☐
  No ☐
- H/o Palpitation Yes ☐
  No ☐
- H/o Chest pain Yes ☐
  No ☐
- H/o Dizziness Yes ☐
  No ☐
- H/o Headache Yes ☐
  No ☐

- Drug History-

EXAMINATION

ANTHROPOMETRY

Height	
Weight	
BMI	

VITAL SIGNS

Temperature	
Heart rate	
Respiratory rate	
BP mmHg)	

GENERAL EXAMINATION

Pallor	Yes ☐ No ☐
Icterus	Yes ☐ No ☐
Generalized lymphadenopathy	Yes ☐ No ☐
Edema	Yes ☐ No ☐
Koilonychia	Yes ☐ No ☐
Leuconychia	Yes ☐ No ☐
Glossitis	Yes ☐ No ☐
Cheilitis	Yes ☐ No ☐

SYSTEMIC EXAMINTION

CVS -

RS -

ABDOMEN -

CNS -

INVESTIGATIONS

Parameters	Values	Reference range
Hemoglobin		1 to 23 months: 10.5 -14 g/dl 2 to 9 years : 11.5 - 14.5 g/dl 10 to 17 years : 12.5 -15 g/dl
WBC count		1 to 23months: 6000 -14000 cells/cu.mm 2 to 9 years : 4000 – 12000cells/cu.mm 10 to 17 years : 4000 - 10500cells/cu.mm
RBC count		6m to 2 years: 3.7 -5.3 million/cu.mm 2 to 6 years: 4.9 – 5.3 million/cu.mm 6 to 12 years: 4 – 5.2 million/cu.mm
PCV		1 to 23 months: 32 -42 % 2 to 9 years : 33 -43% 10 to 17 years : 36 -45%
MCV		1 to 23 months: 72 – 88 fL 2 to 9 years : 76 – 90 fL 10 to 17 years : 78 – 95 fL
MCH		1 to 23 months: 24 – 30 pg 2 to 9 years : 25 – 31 pg 10 to 17 years : 26 – 32 pg
MCHC		32.0 – 36.0 %
RDW		10.0 – 15.0 %

Platelet count		154000 – 400000 /cu.mm
Reticulocyte count		<1%
Serum Iron		50.00 – 120.00 µg/dL
TIBC		250.00 – 400 µmol/L
Transferrin saturation		15 - 55%
Serum Transferrin		208 – 400 mg/dl
Serum Ferritin		6.0 – 67.0 ng/mL
Serum B12		140.00 – 700.00 pg/mL
Serum Folic acid		1.8-9.0 ng/mL
ESR		5-15 mm/Hr
HsCRP		0.01-2.80mg/l

- Peripheral smear study :
- Stool for occult blood :

DIAGNOSIS:

TREATMENT DETAILS:

FOLLOW U P DETAILS

DATE	Hb values	Remarks

ANNEXURE II

PATIENT CONSENT FORM

Study title: PROFILE AND OUTCOME OF NUTRIONAL ANEMIA IN CHILDREN ATTENDING THE PEDIATRIC DEPARTMENT OF A TERTIARY CARE HOSPITAL IN CHENNAI

Study center: ESI-PGIMSR, K.K.Nagar, Chennai

Participant name: Age: Sex:
I.P.No:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to clarify all my queries and doubts and they have been answered to my satisfaction. Investigator explained very well about the procedure and I am made aware of the safety, advantage and disadvantage of the technique.

I understand that my participation in the study is purely voluntary and that I am free to withdraw at any time without giving any reason.

I have understood that the investigator, regulatory authorities and the ethics committee will have access to my health records both in respect to current study and any further research that may be conducted in relation to it, even if I decide to withdraw from the study. I have understood that my identity will not be revealed in anyway and information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

Without any compulsion I am willing to give consent for the participation of my child in this study.

Date: Signature / thumb impression of patient

Place:

Patient name:

Signature of the investigator:

Name of the investigator:

ANNEXURE III

Sample size Calculation

The following were the statistical considerations.

Single Proportion – Absolute Precision	
Expected Proportion	0.75
Precision (%)	7
Desired confidence level (1-alpha) %	95
Required sample size (n)	147

The required sample size for this study was 147 participants. 3 cases were taken as lost to follow up. So, 150 participants were enrolled. Therefore, total sample size was 150 for this study.

The following were the sample size calculations for the interventional study.

Single Mean - Paired t-test	
Pre-test mean	8
Post-test mean	9
Standard deviation in Pre-test	2.2
Standard deviation in Post-test	2
Effect size	0.47619
Power (1-β)%	90
Alpha Error (%)	5
1 or 2 sided	2
Required sample size	48

2 sided: Null hypothesis: There is no difference between before and after treatment.

So, a total sample size of 50 was taken for follow up study including lost to follow up. The Master software (version 1.0) was used to calculate sample size.