Dissertation on

EFFICACY OF INTRAVENOUS IRON SUCROSE IN TREATING IRON DEFICIENCY ANAEMIA IN ANTENATAL PATIENTS"

Submitted in partial fulfillment for

M.D. DEGREE EXAMINATION

BRANCH - II

OBSTETRICS AND GYNAECOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003

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APRIL 2011

CERTIFICATE

This is to certify that the dissertation titled **"Study of Efficacy of Intravenous Iron sucrose in Treating Iron Deficiency Anaemia in Antenatal Patients"** submitted by Dr. G.V. Preetha to the Faculty of Obstetrics and Gynaecology, Madras Medical College, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree (Obstetrics and Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance.

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DECLARATION

I Dr. G.V. Preetha solemnly declare that the dissertation titled "**Efficacy of Intravenous Iron sucrose in Treating Iron Deficiency Anaemia in Antenatal Patients"** has been prepared by me.

This is submitted to the Tamilnadu Dr. MGR Medical University, Chennai in partial fulfillment of the rules and regulation for MD Degree Examination in obstetrics and Gynaecology. This has not been previously submitted by me for the award of any degree or diploma from any university.

Place : Chennai **Dr. G.V. Preetha**

Date :

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I wish to express my deep gratitude to Prof Dr.Baby Vasumathi MD DGO for her valuable guidance, support and encouragement throughtout my study.

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I will be ungrateful if I don't thank my patients who have given me excellent co-operation all through my study.

I thank my family members very much for their support and encouragement at every step.

ETHICAL COMMITTEE CERTIFICATE

I **Dr. G.V. Preetha** apply for the ethical committee certificate for the project **"Efficacy of intravenous iron sucrose in treating iron deficiency anemia in antenatal patients"** under the guidance of Dr. Baby Vasumathi M.D, D.G.O Institute of Obstetrics and Gynaecology, Egmore, Chennai – 8.

I understand the implications of doing research with human subjects and will fully comply with the regulations and keep the dignity and protect the health of subjects at all costs.

Signature of Postgraduate student

I have no objection to guide this postgraduate student in the project mentioned above. I shall supervise that all the human rights are protected and research is carried on with the utmost humanitarian principles.

Signature of the guide **Seal of guide**

I certify that this project has been presented in front of the Ethical Committee, duly formatted in this institution and that all the members of the Ethical Committee have given permission to conduct this research.

Chairman of Ethical Committee **Seal of Chairman**

Date:

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INTRODUCTION

Anemia is one of the major public health problems in the developing world. More then 70% of pregnant women in South-East Asia region suffer from nutritional anemia.(1)

Anemia is the direct cause of maternal death in 10-15% of cases but is an associated cause in many maternal deaths due to haemorrhage, sepsis and cardiac failure.(2)

Anemia in pregnancy is associated with an adverse obstetric outcome in form of spontaneous abortions, preterm labor, low birth weight babies and intrauterine growth retardation.

It is paradoxical but true that though the cause of pregnancy anemia is known and iron therapy is cheap, anemia continues to take a heavy toll of maternal lives in most developing countries.

The World Health Organisation (WHO) defines anemia in pregnant women as hemoglobin level below 11 g / dl.

Iron deficiency is the most common hematinic deficiency in pregnancy, followed by folate deficiency.

Prevention or early treatment of pregancy anemia is the best prophylaxis against maternal mortality.

Supplementation with iron and folic acid during pregnancy is an effective method for preventing mortality and morbidity associated with anemia and improving the outcome of pregnancy.

One of the primary aims of antenatal care is to prevent and treat anemia during pregnancy, since the safety of labour and the puerperal state, to say nothing of the future health, depend upon the state of the patient's hemoglobin reserve

Iron is an essential component of hemoglobin in blood. The pregnant women needs 1000 mg of iron all through her pregnancy to maintain iron balance.

Traditional iron therapy which is based on either oral administration of iron or blood transfusion has many drawbacks. Now parenteral administration of new well tolerated iron preparations like iron sucrose which has been successfully used in treatment of anemia has revolutionised the treatment of anemia. (3)

This study was done to find out the efficacy and safety of intravenous iron sucrose in treatment of iron deficiency anemia in second trimester of pregnancy.

REVIEW OF LITERATURE

HISTORICAL ASPECTS OF ANEMIA

The work of Hedin and Wintrobe in assessing the volume of packed red calls by various types of hematocrit and work of Keith and Garothy in estimating blood volume lead to accurate laboratory definition of the presence or absence of anemia.

Pierre Blaud in 1832, discovered that ferrous sulphate tablets were effective therapy for iron deficiency anemia.

In 1932, Wintrobe deviced the concept of red cell indices.

In 1992 Price quantitated the variation in red cell size seen in various types of anemia.

Review of Literature

- **Bayoumeu F, Subiran - Buisset C, Baka NE, Legagneur H, Monnier - Barbarino P, Laxenaire MC** - France, 2002 March conducted a randomised control study in 50 patients with hemoglobin 8 to 10g/dl and ferritin <50 microg/L.

In intravenous group, iron dose was calculated from the formula: weight before pregnancy (kg) x (120g/l - actual hemoglobin g/l). Oral group received 240 mg of iron sulphate per day for 4 weeks. Treatment efficacy was assessed by measurement of hemoglobin and reticulocytes on days 8,15, 21 and 30 and at delivery and of ferritin on day 30 and at delivery.

Results:- An increase in hemoglobin rising from 9.6±0.79 g/dl to 11.11±1.3 g/dl on day 30 in IV group and from 9.7±0.5g/dl to to 11 ±1.25g/dl on day 30 in per oral group. On day 30 and at delivery ferritin was higher in IV group. A mean higher birthwegiht of 250 g was noted in IV group (not significant).

- **Bencaiova G. Von Mandach U, Zimmermann R Zurich** University Hospital, Switzerland 2009 Jun; 144(2) : 135-9. Epub 2009 Apr 29 have done a study to assess and compare the efficacy and safety of two or three doses of intravenous

iron sucrose with daily oral ferrous sulphate in the prophylaxis of iron deficiency anemia in pregnant women 260 women between 21st and 24th week with singleton pregnancy were randomised into either the intravenous iron group or oral iron group. Of 130 women in IV iron group 75 received 2 doses of 200mg iron sucrose and 55 received 3 doses of 200mg iron sucrose.

The first dose was administered between 21st and 24th weeks, second dose between 28th and 32nd week and third between 35th and 37th week. Women of oral group were given oral tablets of 80mg ferrous sulphate daily, beginning on day of study enrolment and stopping on day of delivery.

Conclusion:- There was no significant difference in the haematological, maternal and fetal outcomes in the parenteral route of iron prophylaxis in pregnant women.

- **AI RA, Unlubilgin E, Kandemir O, Yalvac S, Cakir L, Haberal A** – Ankara Maternity Hospital, Turkey 2005 December conducted a randomised controlled study to compare the efficacy of intravenous iron to oral iron in treatment of anemia in pregnancy. 90 women with hemoglobin between 8 and 10.5g/dL and ferrtin <13 microg/L received either oral iron polymaltose complex

(300mg elemental iron per day) or IV iron sucrose → dose calculated as follows:

Calculated: Weight before pregnancy (kg) x (110g/L – actual hemoglobin g/L) x 0.24 +500 mg

Results:- The change in hemoglobin from baseline was significantly higher in intravenous group than oral group.

Ferritin values were higher in IV iron group with no serious adverse reactions.

- **Perewusnyk G, Huch R, Huch A, Breymenn C** – Zurich University, Switzerland , 2002 July.

Parenteral iron therapy in obstetrics – 8 years experience with iron – sucrose complex.

Results:- Fe- sucrose complex has become a major interest to prevent functional Fe deficiency. Good tolerance to this formulation is partly due to low allergenic effect, partly due to slow release of elementary Fe from the complex, incorporation into bone marrow for erythropoiesis is faster.

By using parenteral iron sucrose in cases of severe iron deficiency, anemia during pregnancy is treated efficiently and rate of blood transfusion could be reduced to below 1% of patients per year.

- **Wali A, Mushtaq A, Nilofer J** Pak Med assoc 2002 Sep 52 (9) did a prospective comparative study – efficacy, safety and compliance of intravenous iron sucrose and intramuscular iron sorbitol in iron deficiency anemia of pregnancy.

A total number of 60 pregnant women with gestation age 12-34 weeks who had iron deficiency anemia. They were divided into 3 groups. Group A (n=15) received intravenous iron sucrose according to recommended dose containing 500mg of iron sucrose for storage, in group B (n=20) iron sucrose was administered according to deficit calculated as per formula but 200mg of iron was given for storage. Group C received intramuscular iron sorbitol in dose used as practice.

Conclusion:- Intravenous iron therapy is safe, convenient and more effective than intramuscular iron therapy in treatment of iron deficiency anemia during pregnancy and can replace blood transfusion in antenatal period.

- A study was conducted by **Divakar hospitals** Bangalore

A cohort of 96 women were recruited if their Hb <11g/dl, IV iron sucrose 200mg was given and their Hb estimated after 4 weeks of therapy.

A rise of >2g/dl was seen in 17.2%.

A mean rise of 1.31g/dl in 92%.

- **Al Momen et al** 1996 Nov conducted a prospective, open label controlled trial in 111 pregnant women with iron deficiency anemia, with Hb <9 g/dL and divided into 2 groups. Intravenous iron sucrose was administered as an infusion of single 100mg dose in normal saline every 1 to 3 days. Controls receive intramuscular iron dextran (100mg on alternate days) till the calculated dose was reached.

Intravenous iron therapy resulted in higher levels of Hb, with time to achieve maximum Hb in shorter period compared with controls. No serious adverse effects were noted in iron sucrose group whereas 6% of patients could not tolerate intramuscular iron dextran, who were excluded from study. 30% patients in control group had disturbing gastrointestinal symptoms and 32% were non-compliant.

- **Lipincott William**, Pakistan institute of Medical Science conducted a randomised controlled study in 80 patients with gestational age 12-36 weeks from antenatal clinic and 20 patients after postpartum haemorrhage with anemia.

Group A received intravenous iron sucrose

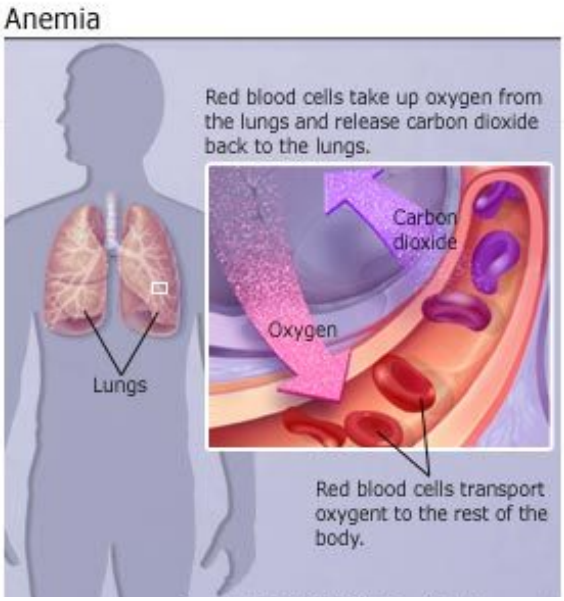
Group B received intramuscular iron sorbitol.

Conclusion:- IV iron sucrose is safe, convenient and more effective than intramuscular iron therapy in treatment of iron deficiency anemia during pregnancy. It can minimise blood transfusion in postnatal women. Increase in Hb was noted 1 week after iron sucrose administration.

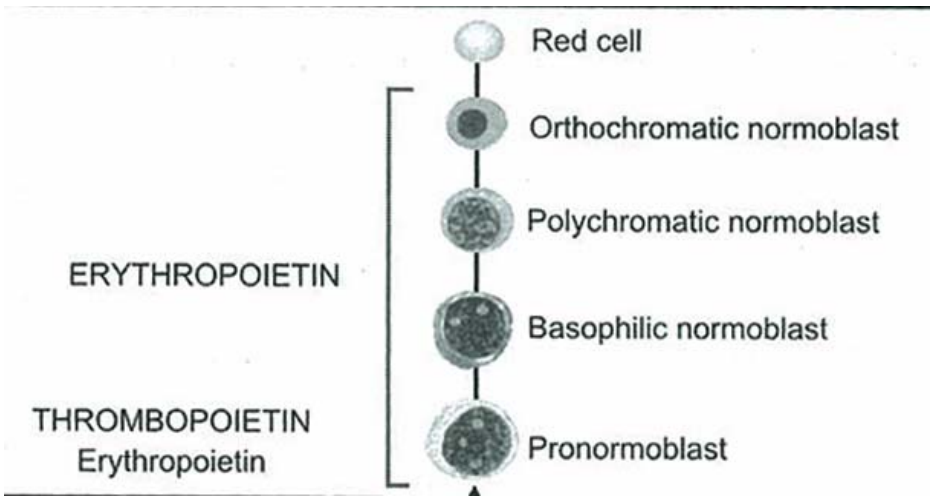
ANEMIA

Definition

Anemia is a reduction in RBC mass and hemoglobin content of blood due to diminished production or increased destruction or loss of RBC. This results in reduced oxygen carrying capacity of blood and inadequate oxygen supply to tissues.



ERYTHROPOIESIS



Erythropoiesis

After birth red cells normally develop only in red bone marrow.

Erythropoietic stem cell (>20 µm)

↓

Proerythroblast (15-20µm)

↓

Early normoblast (Basophil) (14-17µm)

↓

Intermediate normoblast (Polychromatophil (10-15µm)

(Hb appears)

↓

Late normoblast [Orthochromatic and non-dividing] (7-10µm)

↓

Reticulocyte (7.3 µm)

↓

Mature RBC (7.2µm)

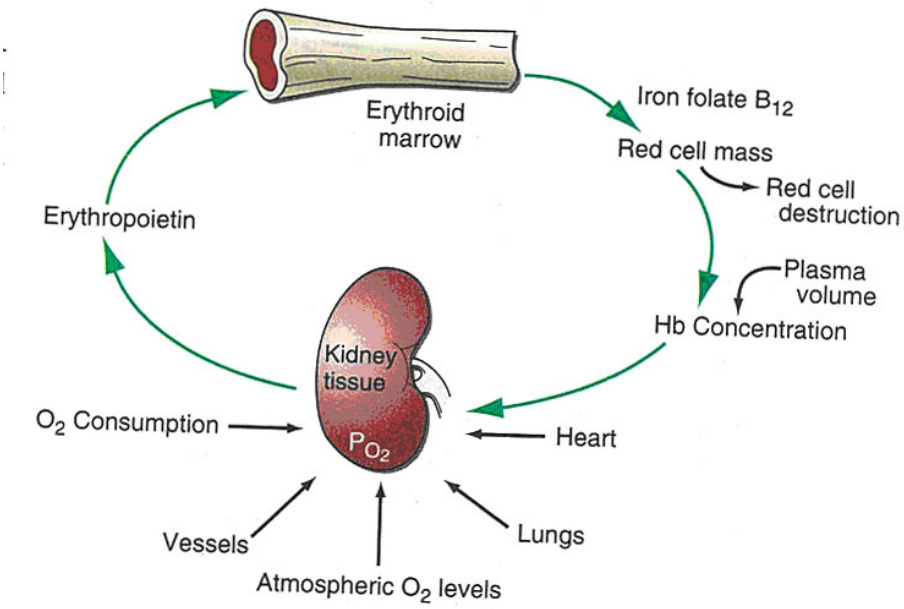
FACTORS REQUIRED FOR RBC FORMATION (4)

1. Proteins

2. Minerals – Iron, copper, zinc and cobalt.

3. Vitamins – Vitamin B12, folic acid, riboflavin and Vitamin C

4. Erythropoietin – Principal stimulus for red cell production in hypoxia.

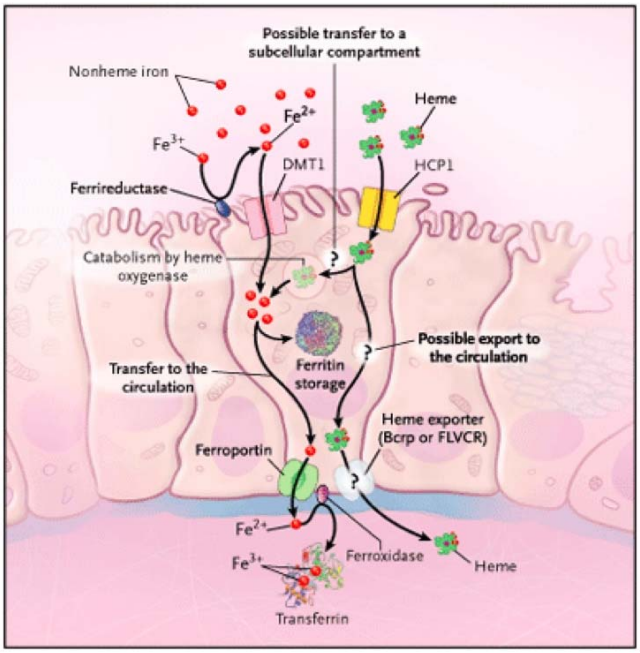


The physiologic regulation of red cell production by tissue oxygen tension. Hb, hemoglobin.

IRON METABOLISM

Body iron distribution

	Iron content, mg (female 60 kg)
Hemoglobin	1700
Myoglobin / enzymes	300
Transferrin iron	3
Iron stores	0 – 300



IRON CYCLE

Bilirubin (excreted)

Macrophage

Degrading Hb → Free iron

Tissues

Ferritin

Hemosiderin
Heme
Enzymes

Free iron

Red Cells

Hb

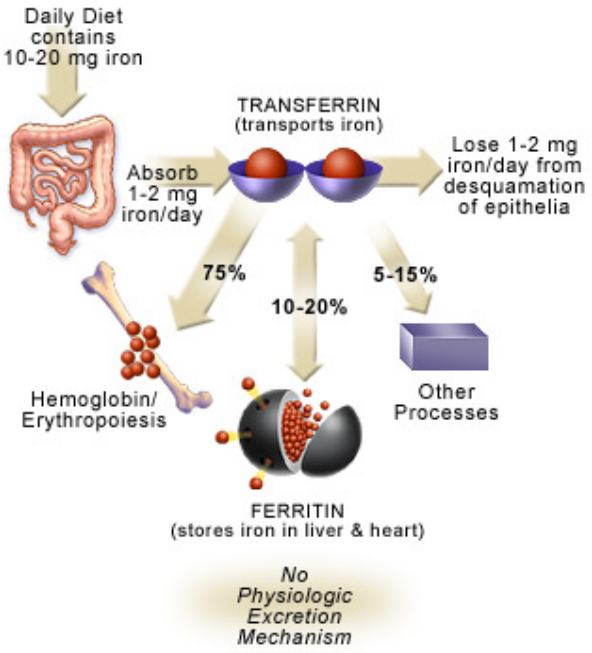
Plasma

Transferrin - FE

Blood loss 0.7 mg
Fe in menses/day

Fe++ absorbed
(small intestine)

Fe excreted
0.6mg daily



The Iron Cycle

ABSORPTION OF IRON

Iron absorption mostly occurs in duodenum and proximal jejunum. Mostly dietary iron is in ferric form which is not easily absorbed. Iron has to be reduced to ferrous form to be transported across the intestinal epithelium.

Iron Transport : Iron is transported by 2 stages

a) Brush border

b) Serosal surface

Brushborder of mucosal cell has mobilferrin which binds to iron and causes it to enter mucosal cell. The mucosal cell has apoferritin which binds iron and iron may be stored in mucosal epithelial cell or may be transported to blood. The regulation of iron absoption is by equilibrium between ferrous iron in mucosal cell, circulating iron levels and stored form of iron in mucosal cell

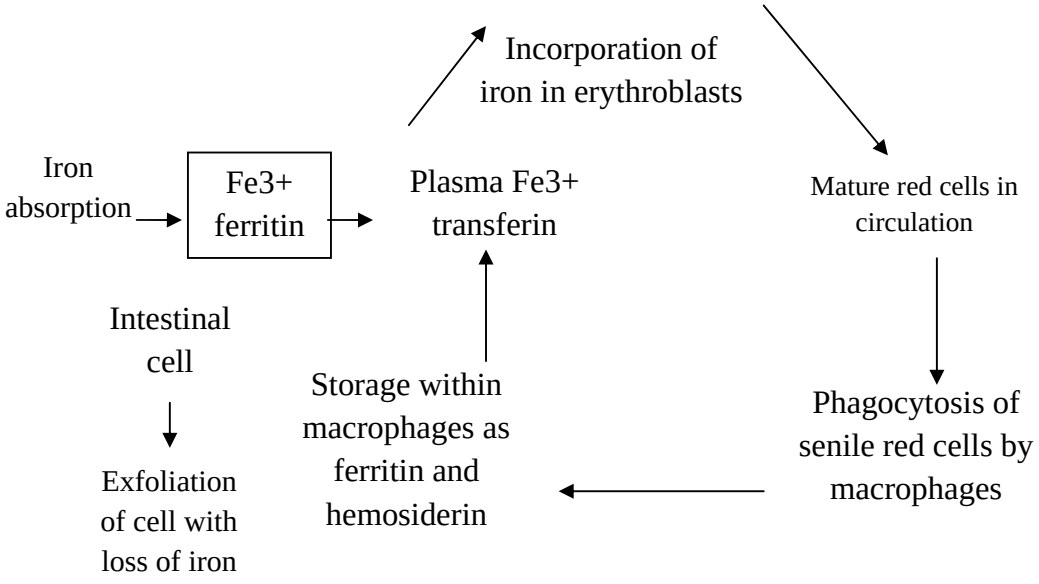
Mucosal Block

The gut has a mechanism to prevent entry of excess iron in body. Iron reaching inside mucosal cell is either transported to plasma or oxidised to ferric form and complexed with apoferritin to form ferritin. This ferritin is generally stored in mucosal cell and is lost when they are shed (life span of mucosal cell is 2-4 days). This is called “FERRITIN CURTAIN”

Transferrin is the protein involved in iron transport. Each transferrin is capable of binding one atom of iron. The transferrin-receptor – iron complex moves inside the cell by an endosome and iron is released and taken up by DMT-1 (divalent metal transporter), that carries iron through endosome membrane to be incorporated intro iron containing proteins or to be stored as ferritin.

The reticuloendothelial system phagocytoses old red blood cells and hemoglobin is liberated. Heme is further converted to biliverdin and iron is released and incorporated into ferritin or transported back to plasma.

Iron Metabolism



Nutritional Iron Balance (5)

The balance of iron in humans is tightly controlled and designed to conserve iron for reutilization there is no regulated excretory pathway for iron, and only mechanism by which iron is lost from the body are blood loss and loss of epithelial cells from the skin, gut and genito urinary tract. This margin between the amount of iron available for absorption and the requirement of iron, this accounts for the great prevalence of iron deficiency worldwide.

The amount of iron required from the diet to replace losses averages 15% in women of child bearing age. An individual with iron deficiency can increase iron absorption to about 20% of iron present in meat containing diet, but only 5-10% of iron in vegetarian diet, because certain food stuffs that include phytates & phosphates reduce iron absorption by 50% when ionizable iron salts are given together with food, the amount of iron absorbed is reduced, this is true with iron in

ferric state. In pregnancy, during the last two trimesters, daily iron requirement increase to 5 to 6mg. That is the reason why iron supplements are strongly recommended for pregnant women.

IRON DEFICIENCY ANEMIA IN PREGNANCY

Definition

According to WHO, anemia in pregnancy is present when the hemoglobin concentration of peripheral blood is 11g/dl or less. Anemia is responsible for 17% of maternal deaths in developing countries.

PHYSIOLOGICAL ANEMIA OF PREGNANCY (6)

Disproportionate increase in plasma volume, RBC volume and hemoglobin mass during pregnancy. In addition there is marked demand of extra iron during pregnancy.

ICMR CATEGORIES OF ANEMIA

Category	Severity (Hb level in g/dl)
Mild	10-10.9
Moderate	7-10
Severe	<7
Very severe	<4

IRON REQUIREMENT IN PREGNANCY

Demands for iron in pregnancy come to a total of about 900mg (range 700 – 1400mg) of which about

- 500 to 600mg goes to the uterus and its contents
- Around 150 to 200 mg are lost in the average blood loss at delivery and a similar amount is expended in lactation.
- In addition, there is an increased maternal hemoglobin mass which consumes about 500mg, but this iron is returned to the stores after delivery.(7)
- On the credit side, there is an average saving of about 225mg as a result of ammenorrhoea throughout pregnancy. (7)
- This leaves a likely iron deficit of 600 to 700mg.

In terms of daily needs, this approximates

4-6 mg/day – Second trimester

6-8 mg/day – Third trimester

Prevalence of iron deficiency anemia in pregnancy

Exact data on prevalence of anemia in women is not available but a crude estimate is that 500 million women between 15 and 49 yrs of age worldwide are anemic.(8)

According to World Health Organization estimates, upto 56% of all women living in developing countries are anemic.(9)

In India, National Family Health survey-2, shows that 54% of women in rural and 46% of women in urban areas are anemic.(10)

Prevalence of anemia during pregnancy is much higher. It is estimated that 60 million pregnant women world wide are anemic. In developing countries, the prevalence of anemia in pregnant women varies between 50 and 90%.

In a steering committee report from India, 13% women were reported to have hemoglobin <5g% and 34% had hemoglobin less then 8gm%.(11)

Nutritional anemia is a serious problem in pregnancy which affects 60-70% of pregnant women.

CAUSES OF INCREASED PREVALENCE OF ANEMIA IN TROPICS

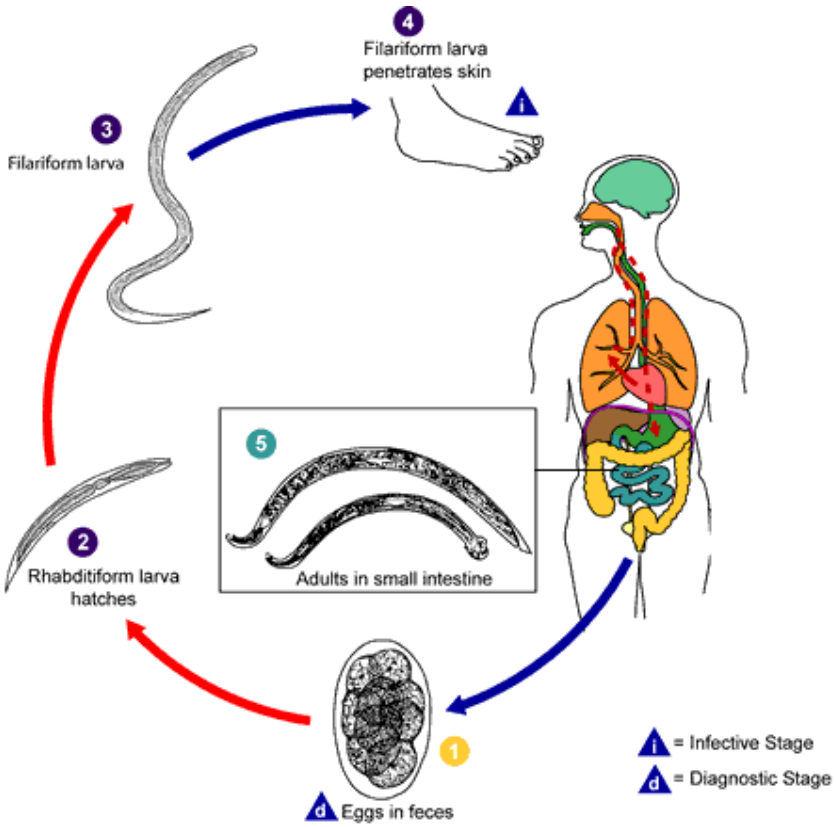
1. Faulty dietetic habit – High phosphate and phytates decrease absorption

2. Faulty absorption mechanism – Due to intestinal infestation

3. Iron loss – (i) Excess blood loss during menstruation

   (ii) Repeated pregnancies at short intervals

   (iii) Hook worm infestation



Hook worm Life Cycle

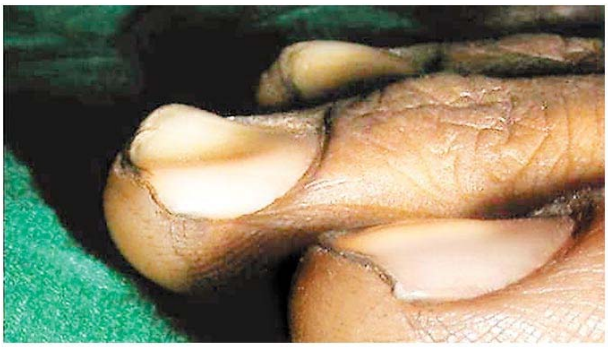
DURING PREGNANCY

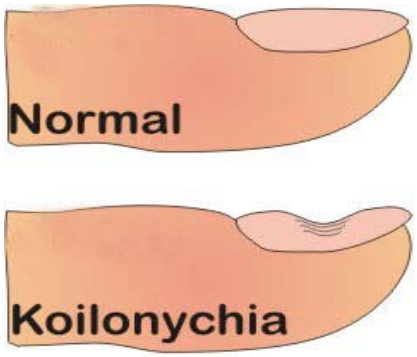
1. Increased demand (13)
2. Diminished intake
3. Disturbed metabolism

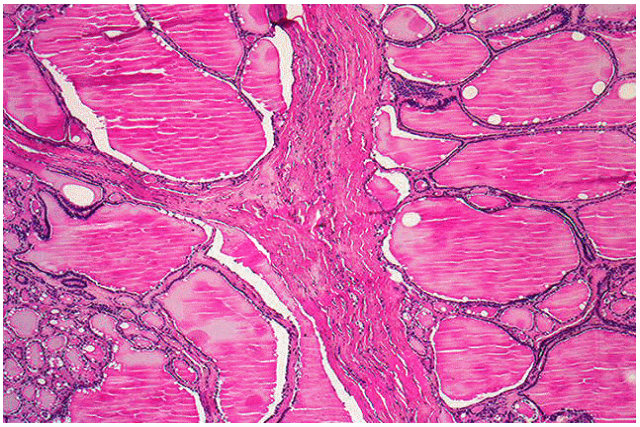
CLINICAL FEATURES

Symptoms	Signs
• Lassitude	• Pallor , Koilonychia
• Palpitation	• Glossitis and stomatitis
• Dyspnoea	• Tachycardia
• Giddiness	• Systolic heart murmur
• Pica	• Oedema (due to hypoproteinemia)

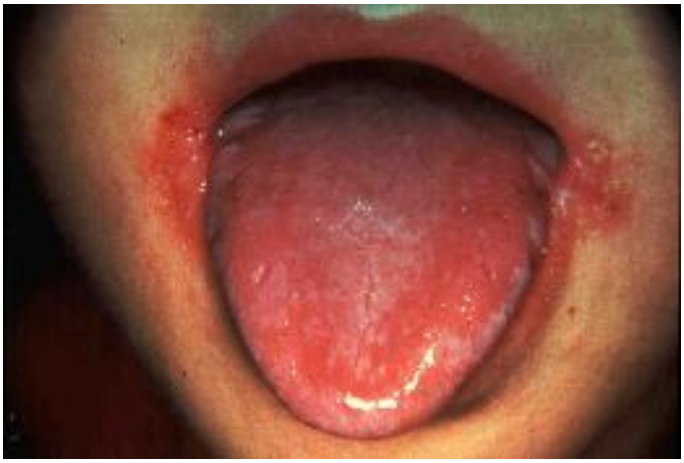
Koilonychia



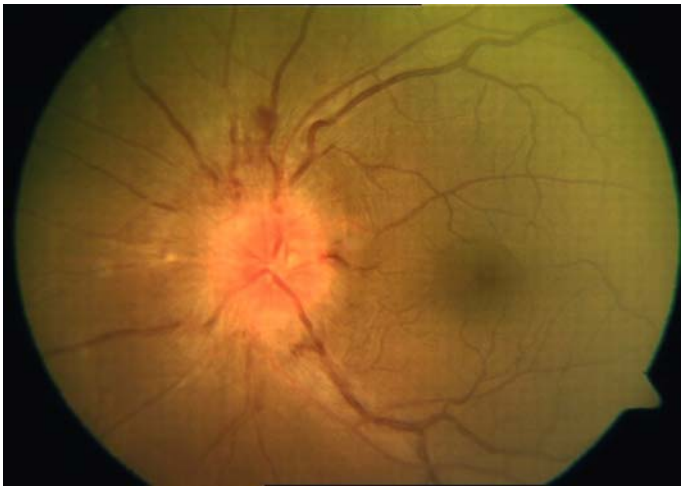




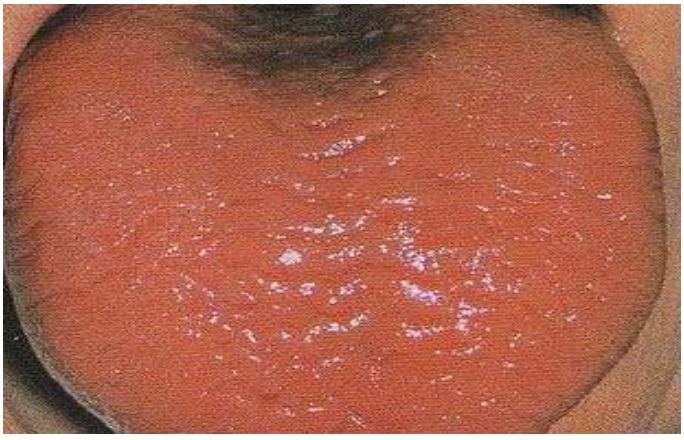
Angular Stomatitis



Eyes – Pallor



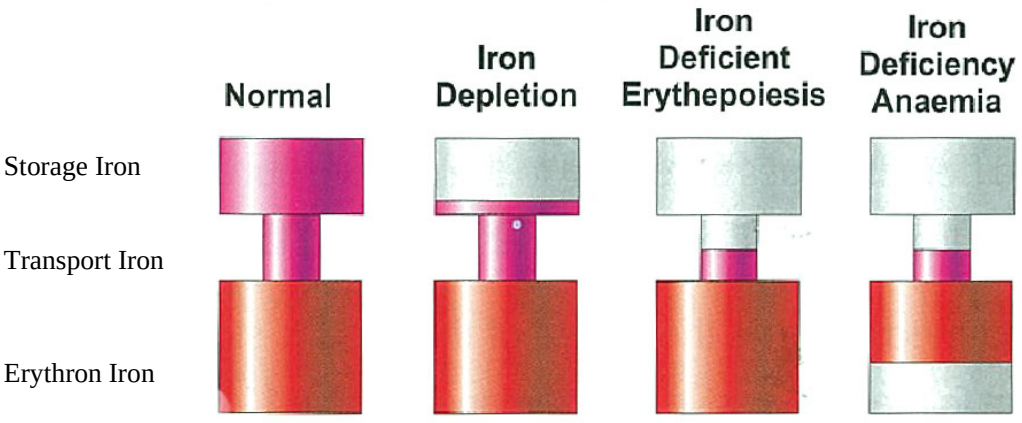
Smooth Bald Tongue



STAGES OF IRON DEFICIENCY

		Normal	Negative Iron balance	Iron deficient erythropoiesis	Iron deficiency anemia
1.	Marrow iron stores	1-3+	0-1+	0	0
2.	Serum ferritin (µg/L)	50-200	<20	<15	<15
3.	TIBC (µg/dl)	300-360	>360	>380	>400
4.	Serum iron (µg/dl)	50-150	NL	<50	<30
5.	Saturation (%)	30-50	NL	<20	<10
6.	Marrow Sideroblasts	40-60	NL	<10	<10
7.	RBC protoporphyrin (µg/dl)	30-50	NL	>100	>200
8.	RBC morphology	NL	NL	NL	Microcytic hypochromic

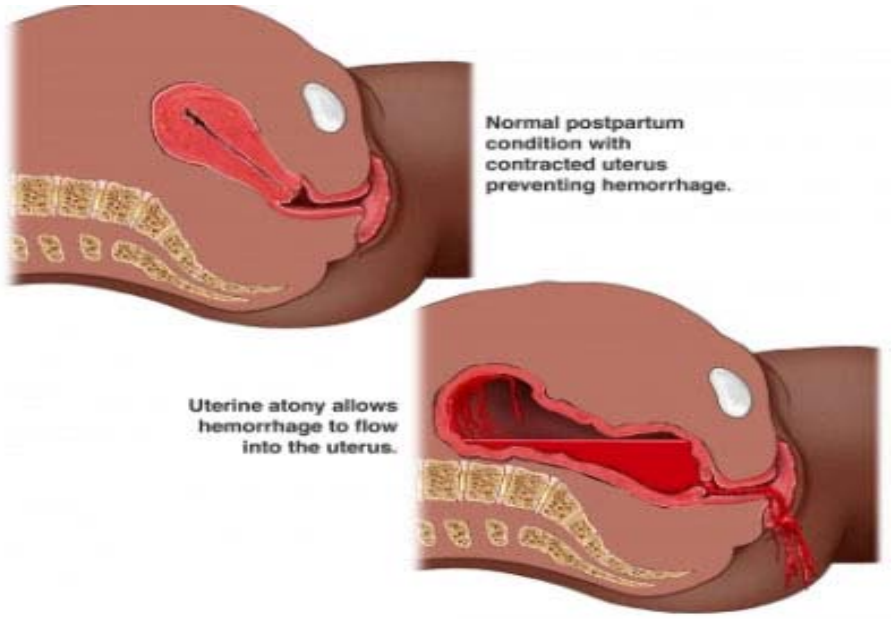
* NL - Normal Limits



COMPLICATIONS OF SEVERE ANEMIA

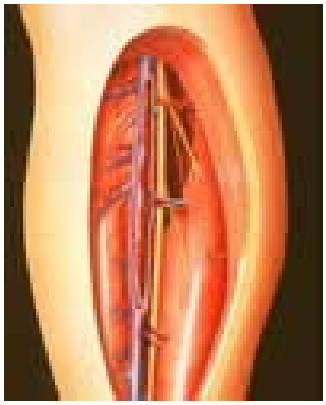
During pregnancy	Labour	Puerperium	Fetal effects
Pre-eclampsia	Uterine inertia	Pureperal sepsis	Low birth weight
Intercurrent infection	Postpartum haemorrhage	Subinvolution	Intrauterine death (anoxemia)
Heart failure at 30-32 weeks of gestation	Cardiac failure	Puerperal venous thrombosis	
Preterm labour	Shock	Pulmonary embolism	

Postpartum Haemorrhage

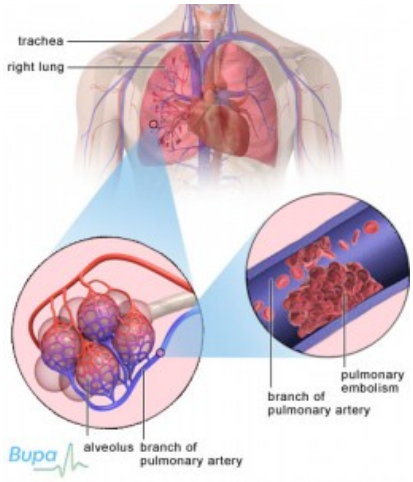


Cardiac failure

Puerperal venous thrombosis



Pulmonary embolism



MANAGEMENT OF IRON DEFICIENCY ANEMIA

Investigations

I. To know degree of anemia

Determination of hemoglobin concentration:·

a) Tallquist method

b) Sahli's acid hematin method

c) Alkali – hematin method

d) Cynmethhemoglobin method – most accurate

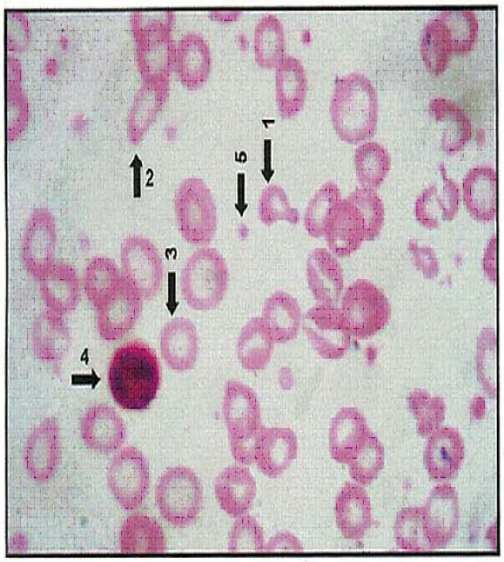
II. To assess type of anemia

a) **Peripheral smear:-** Single best investigation

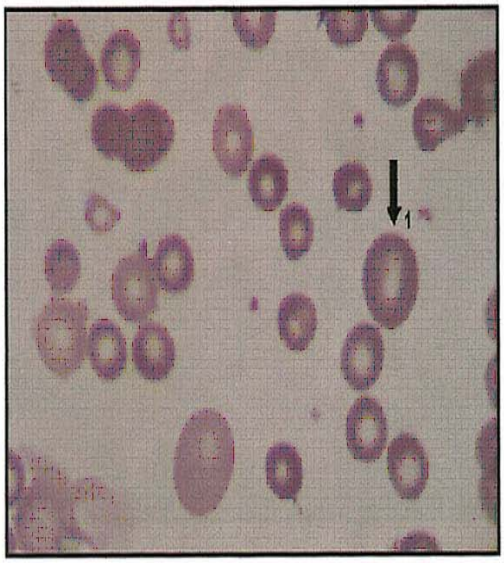
The differential RBC morphology like micro or macrocytosis, hypo or normochromia, anisocytosis, poikilocytosis, howel jolly bodies, heinz bodies, presence of target cells and schizocytes serve as important clues to the aetiology.

It also provides information about white blood cells (WBC), differential count and morphology, platelet count, presence of parasites like malaria and kala-azar, and toxic granules in case of chronic infections.

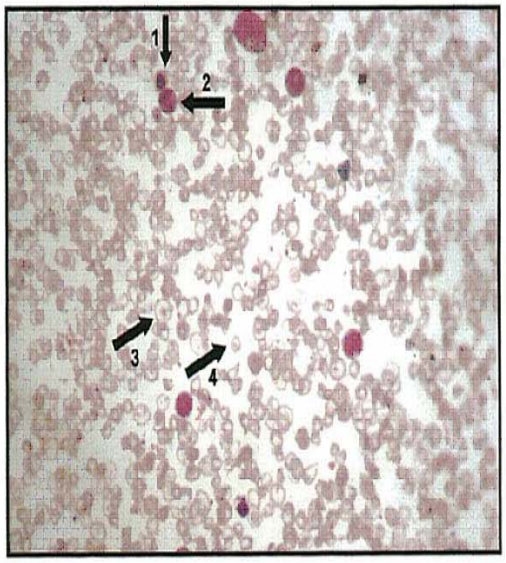
The reticuloyte count requires supravital staining of the peripheral smear.



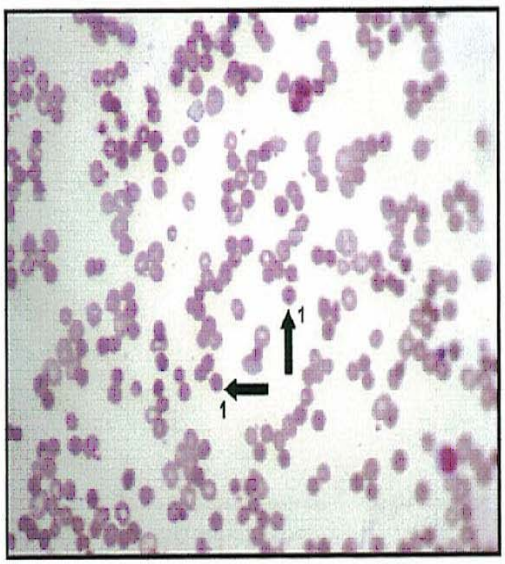
Microcytic hypochromic anemia
1. Tear drop cell **2.** Elliptocyte **3.** Microcytic hypochromic cell **4.** Lymphocyte **5.** Platelet



Macrocytic anemia **1.** Macrocyte

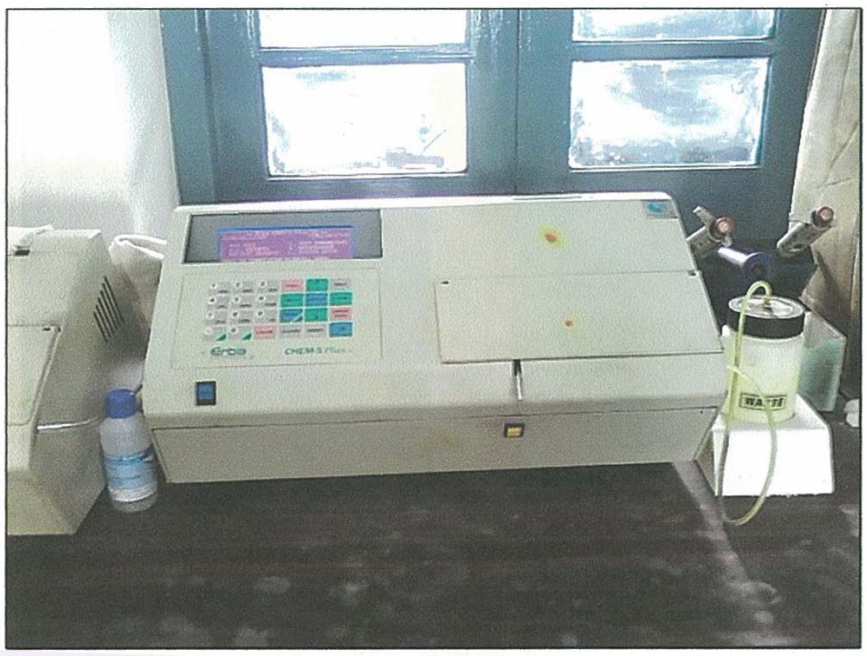


Thalassemia major (marked aniso poikilocytosis)
1. nRBC (nucleoted RBC) **2.** Neutrophil **3.** Target cell **4.** Microcytic hypochromic cell



Hereditary spherocytosis **1.** Spherocyte

SEMI AUTO ANALYSER –ERBA CHEM PLUS V2



CELL COUNTER

Hematological indices (15)

a. MCV (Mean corpuscular volume)

$$MCV = \frac{\text{Packed cell volume \%}}{\text{RBC count in millions/cu.mm}} \times 10$$

Normal range : 80-95 fl

b. MCH (Mean corpuscular hemoglobin)

c. $$MCH\ (Picograms) = \frac{\text{Hemoglobin g/dl}}{\text{RBC count in millions/cu.mm}} \times 10$$

Normal range : 27-32 pg

d. MCHC (Mean Corpuscular hemoglobin concentration)

$$MCHC\ (g/dl) = \frac{\text{Hemoglobin g/dl}}{\text{Packed cell volume}} \times 100$$

Normal range : 34 – 37 g/dl

III. Other Blood values

1. Serum iron: 50-150 µg/L

2. TIBC (Total Iron Binding Capacity)

Normal 325-400 µg/dl

Increased in iron deficiency anemia

3. Transferrin saturation

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

Normal : 20-50%

Decreased in iron deficiency anemia

4. Serum ferritin :- Normal value 50 – 200 μg/ l

- Most sensitive and specific test for iron deficiency anemia

- It correlates with body iron stores

5. Soluble transferrin receptor assay (TFR). (16)

o Indicates cellular iron status

o Increased in iron deficiency anemia.

6. Free erythrocyte protoporphyrin:- Increased in iron deficiency anemia.

7. Red cell distribution width – It is the degree of variation of red cells size.

It is increased in iron deficiency anemia and helps in distinguishing from beta-thalassemia, where red cell distribution width is normal.

IV. Examination of stool: To detect helminthic infection (especially hookworm)

V. Examination of urine – for protein, sugar and pus cells.

VI. Bone marrow aspiration and examination

INDICATION

a) Cases not responding to therapy

b) To diagnose hypoplastic anemia

c) To diagnose kala-azar

TREATMENT OF IRON DEFICIENCY ANAEMIA

PROPHYLACTIC SUPPLEMENTATION

An additional 500-600mg iron is required in pregnancy at a rate of 4-6 mg/day increasing upto 8mg/day in later stages of pregnancy. The average Indian diet seldom contains more than 15mg of iron per day, of which only a fraction (about 10%) is available for absorption.

So there is a definite role of prophylactic supplementation in pregnant women in developing countries.

Natural foods like liver, meat, eggs, peas, green leafy vegetables, sprouts, jaggery and certain dried fruits are sources of iron.

- WHO recommendation based on the prevalence of anemia is 60 mg elemental iron with 400 microgram of folic acid for 6 months, where prevalence of anemia is <40% and this dose to be

supplemented for another 3 months postpartum in areas where the prevalence >40%. (17)

- The National Nutritional Anaemia control programme of India recommends 100mg elemental iron and 500 microgram of folic acid for prophylactic supplementation for a minimum of 100days starting in second trimester. (18)

- Double this dose for the treatment of anemia that is 200mg elemental iron and 1000 microgram of folic acid. These cases need to continue the iron supplementation in postpartum period for 3-6months to replenish stores

Treatment

Oral therapy

There must be atleast 10 weeks for delivery to obtain satisfactory result with oral iron.

Iron is best absorbed in the ferrous form

Ferrous sulphate is cheapest of these and is suitable for most patients.

Other expensive forms like ferrous fumarate, gluconate and succinate may produce less epigastric discomfort.

Iron preparations should preferably be taken on an empty stomach to prevent dietary factors from interfering with its absorption.

Recently two newer preparations namely carbonyl iron and iron polymaltose complex have been shown to be effective with lesser gastrointestinal side effects. (19)

Treatment should be continued till the blood picture becomes normal, thereafter a maintenance dose is to be continued for atleast 100 days following delivery to replenish the iron stores.

Drawbacks

1. Intolerance – Evidenced by epigastric pain, nausea, vomiting and diarrhoea or constipation.

2. Unpredictable absorption rate – Antacids, oxalates and phospates will reduce absorption.

3. With the therapeutic dose, the serum iron may be restored but there is difficulty in replenishing the iron stores.

Rate of improvement

The improvement should be evident within three weeks of therapy.

Hemoglobin concentration is expected to rise at the rate of 0.7gm/100ml per week.

Contraindications of oral therapy

1) Intolerance to oral iron.

2) Severe anemia in advanced pregnancy

PARENTERAL THERAPY

Indications:- 1. Intolerance to oral iron

2. Patient is not co-operative to take oral iron

(A) Intravenous route

(i) Total dose infusion:- The deficit of iron is first calculated and total amount of iron required to correct the deficit is administered by a single sitting intravenous infusion. The compound used is iron dextran. 1 ml of which contains 50 mg elemental iron.

Advantage:- It eliminates repeated and painful intramuscular injections.

Estimation of the total Iron requirement (20)

0.3 x W (100-Hb%)

W → patient's weight in pounds;

Hb – hemoglobin

Additional 50% is to be added for replenishment of the body iron store.

B) **Intra muscular therapy:-** The compounds used are

(i) Iron dextran (Imferon)

(ii) Iron – sorbitol – citric acid complex (Jectofer)

Both preparations contain 50 mg elemental iron in one milli litre.

Oral iron should be suspended atleast 24 hours prior to therapy to avoid reaction

Both compounds require a test dose to be given

The injections are given into upper outer quadrant of the buttock

Drawbacks

1) Injections are painful

2) Chance of abcess formation and discolouration of skin over the injection site

3) Reactions - Pyrexia, Lymphadenopathy, headache, nausea, vomiting and allergic reactions are infrequently met with.

Rate of improvement : The expected rise in hemoglobin concentration is 0.7 gm / 100ml per week The rate of response to iron therapy is the same whether given orally or parenterally

Newer injectable iron preparations now available are iron polymaltose for intramuscular and intra venous use and sodium ferric gluconate for intravenous use. (21)

Place of Blood Transfusion

Indications

1) To correct anemia due to blood loss and to combat postpartum hemorrhage

2) Patients with severe anemia seen in later months of pregnancy

3) Refractory anemia – not responding to either oral or parenteral therapy

4) Associated infection

Advantages

1) Increases oxygen carrying capacity of the blood

2) Stimulates erythropoiesis

3) Supplies the natural constituents of blood like proteins, antibodies, etc

4) Improvement is expected after 3 days

Draw backs

1) Premature labour

2) Increased chance of cardiac failure with pulmonary edema because of over loading of heart

3) Transfusion reactions

Erythropoietin

Recently this has been tried in resistant cases of anemia during pregnancy and anemia associated with end stage renal disease.

- It acts by stimulating erythropoiesis and therefore may be an alternative to blood transfusion
- Recombinant human erythropoietin (rHu Epo) given at a dose of 150 IU / kg subcutaneously thrice a week.(22)
- Administration of rHuEpo seems to be safe for the fetus since it does not cross the placental barrier due to its high molecular weight
- Adequate iron stores need to be present for the treatment to be effective

Side Effects

Hypertension and thrombosis in mother, more often in women with end stage renal disease.

IRON SUCORSE

- It is a sterile complex of polynuclear iron (III) hydroxide for intravenous use with molecular weight of approximately 60,000 daltons.

- Each ml contains 20mg elemental iron as iron sucrose in water for injection.
- The drug product contains 30% sucrose and pH 10.5-11.1
- It has no preservatives and the osmolality 1.250 mOsmoL/L.

MECHANISM OF ACTION

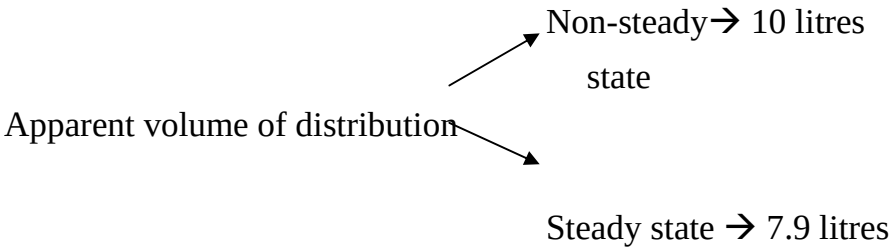
After iv injection, iron sucrose mix with plasma, then enter reticuloendothelial system directly from intravascular fluid compartment. Resident phagocytes of liver, spleen and bone marrow remove iron agents from circulatory plasma. Within phagocytes, iron is released from the iron sucrose compound into iron pool. Iron is either incorporated by ferritin into intracellular iron stores or released from the cell to be taken up by the extracellular iron binding protein transferrin. Iron-transferrin supplies iron for hemoglobin synthesis and maturation of red blood cells.

PHARMACOKINETICS

Iron component exhibits first order kinetics

Elimination t ½ : 6 hrs

Total clearance 1.2 l/hr



Iron sucrose mainly distributes in blood and to some extent in extra vascular fluid.

Elimination

Sucrose component and some iron is eliminated by urinary excretion.

Side effects

- Hypotension
- Cramps (Leg cramps)
- Nausea
- Headache
- Vomiting
- Diarrhoea

METHOD OF ADMINISTRATION

1. SLOW IV INJECTION

100mg to be administered undiluted over a period of 2-5 minutes.

2. IV INFUSION

100mg to be diluted with 100ml of normal saline immediately prior to infusion and infused over a period of at least 15 minutes.

Maximum 100-200mg given as single dose repeated upto 3 times a week on alternate days.

Storage: stored at 25°C

Excursions permitted 15-30°C

SALIENT FEATURES OF IRON SUCROSE

1. It is safe and effective in pregnancy anemia.
2. It has convenient dosage and administration.
3. It is a stable high molecular complex which does not cause transferrin saturation.
4. It does not cause oxidative cell toxicity due to free iron generation.
5. It has a safety profile established worldwide.
6. It is safe for patients intolerant to other IV iron preparations.
7. No parenchymal damage.

AIM OF STUDY

AIM OF STUDY :

1. To determine efficacy of iron sucrose in treating iron deficiency anemia in antenatal patients

2. To determine safety of iron sucrose in treatment of iron deficiency anemia in antenatal patients

MATERIALS AND METHODS

STUDY PLACE : Institute of Obstetrics and Gynaecology

STUDY PERIOD : 2009 to 2010

STUDY DESIGN : Prospective Study

50 antenatal patients in second trimester with hemoglobin > 6 grams % and < 11 grams % were selected.

INCLUSION CRITERIA

a) Gestational age 13 to 28 weeks

b) Hemoglobin > 6 gm % to < 11 gm %

EXCLUSION CRITERIA

1) Patients in first and third trimester of pregnancy.

2) Bleeding disorders.

3) Patients who had known allergy to parenteral iron.

METHOD

Iron sucrose was given as an intravenous infusion.

No test dose required

200mg iron sucrose was diluted with 100 ml of normal saline immediately prior to infusion and is to be infused over a period of atleast 30 minutes to 1 hour. The same dose repeated after 2 days.

We did not calculate the optimal dose of iron sucrose required by each women based on her pre – treatment hemoglobin; we studied the response to a uniform dose over a range of pre – treatment hemoglobin values.

Hemoglobin, packed cell volume were analysed by automatic cell counter. Serum iron, total iron binding capacity was calculated using semi auto analyser.

An informed consent obtained from patient and a detailed history taken.

OBSERVATION

- The following investigations were done before starting therapy.

1. Hemoglobin
2. Packed cell volume
3. Peripheral smear
4. Serum iron
5. Total iron binding capacity

6. Iron saturation

7. Serum ferritin

During therapy the following were monitored

1. Vitals - Pulse rate, Blood Pressure, Temperature.

2. Anaphylactic reactions

3. Adverse effects - Nausea, Vomiting, Chills & Rigors, Abdominal Pain, Headache etc.

RESULTS

I . AGE DISTRIBUTION

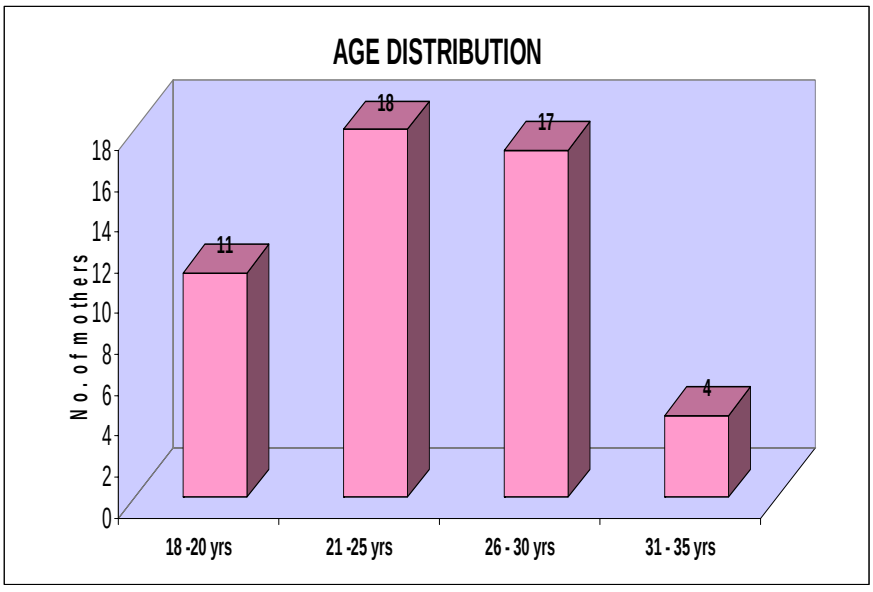
Age group	No. of Mothers	%
18 – 20 Yrs	11	22.0%
21 – 25 Yrs	18	36.0%
26 – 30 Yrs	17	34.0%
31 – 35 Yrs	4	8.0%
Total	**50**	**100**

Among the fifty patients 22% (11/50) were in the age group 18 – 20 yrs.

* 36% (18/50) were in age group 21 – 25 yrs.

* 34% (17/50) were in age group 26 – 30 yrs

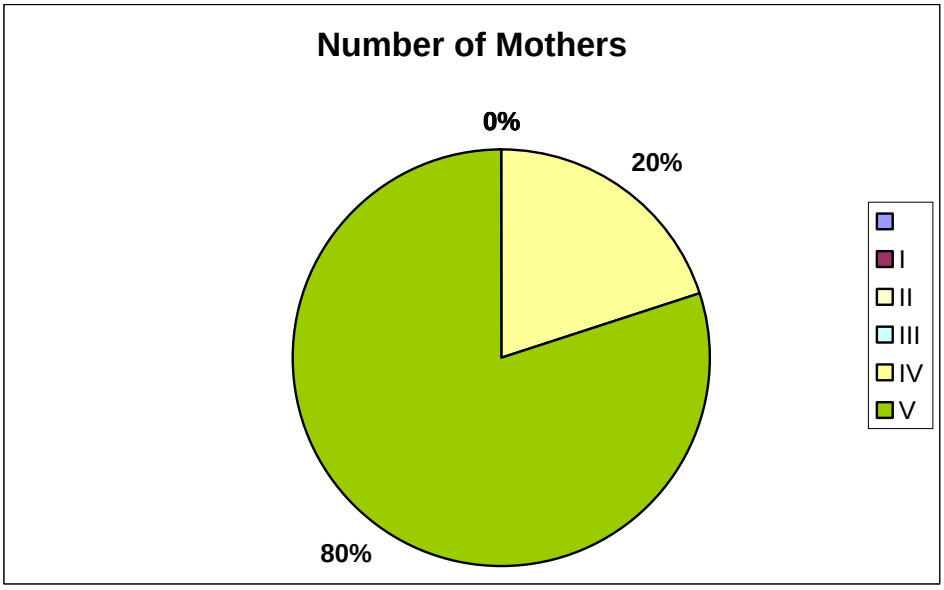
* 8% (4/50) were in age group 31 – 35 yrs.



II. SOCIOECONOMIC STATUS

Socio Economic Status	Number of Mothers	%
I	-	-
II	-	-
III	-	-
IV	10	20
V	40	80
Total	**50**	**100**

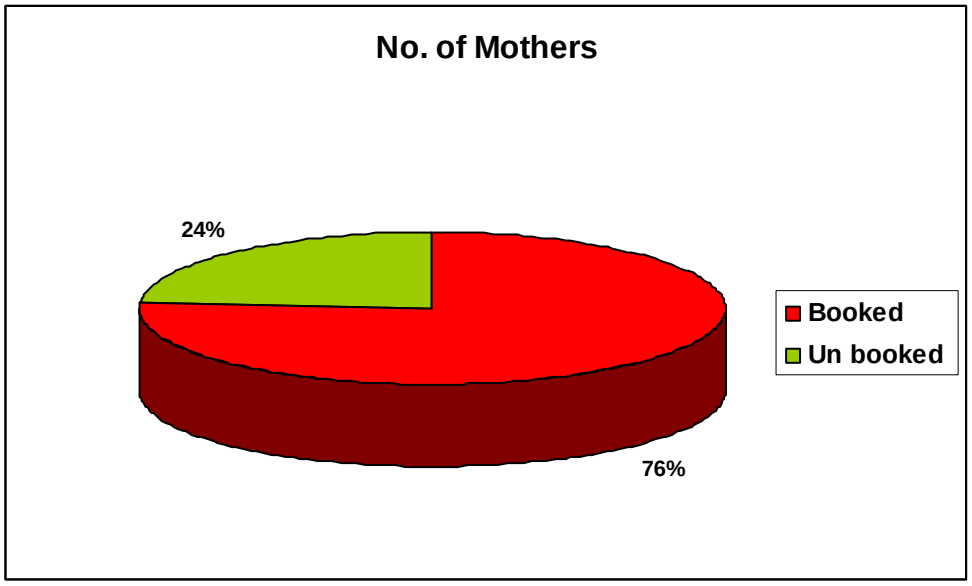
Among the fifty patients, 20% (10/50) are in class IV Socio – economic status. 80% (40/50) are in Class V socio economic status.



III. BOOKING STATUS

Booking Status	No. of Mothers	%
Booked	38	76%
Un booked	12	24%
Total	**50**	**100**

Among the fifty patients, 76% (38/50) were booked and 24% (12/50) were un-booked.

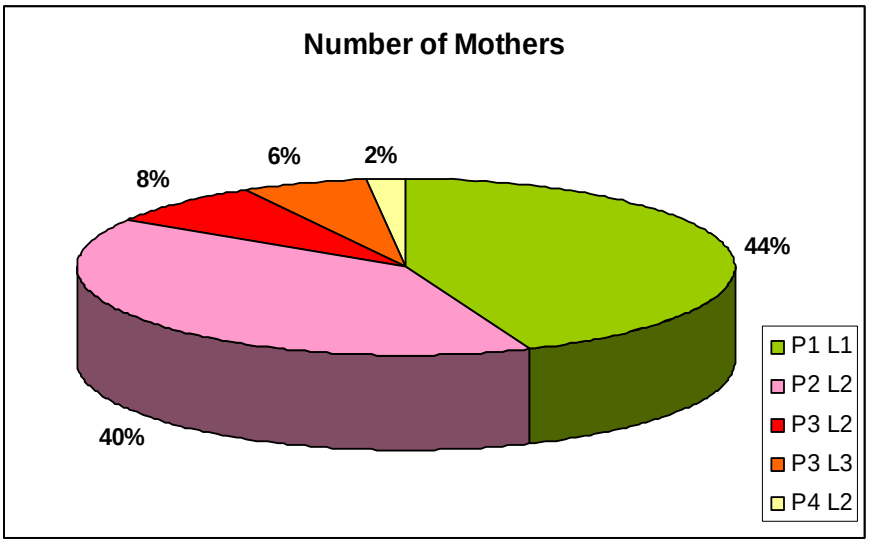


IV. OBSTETRIC CODE

Obstetric Code	Number of Mothers	%
$P_1 L_1$	22	44%
$P_2 L_2$	20	40%
$P_3 L_2$	4	8%
$P_3 L_3$	3	6%
$P_4 L_2$	1	2%
Total	**50**	**100%**

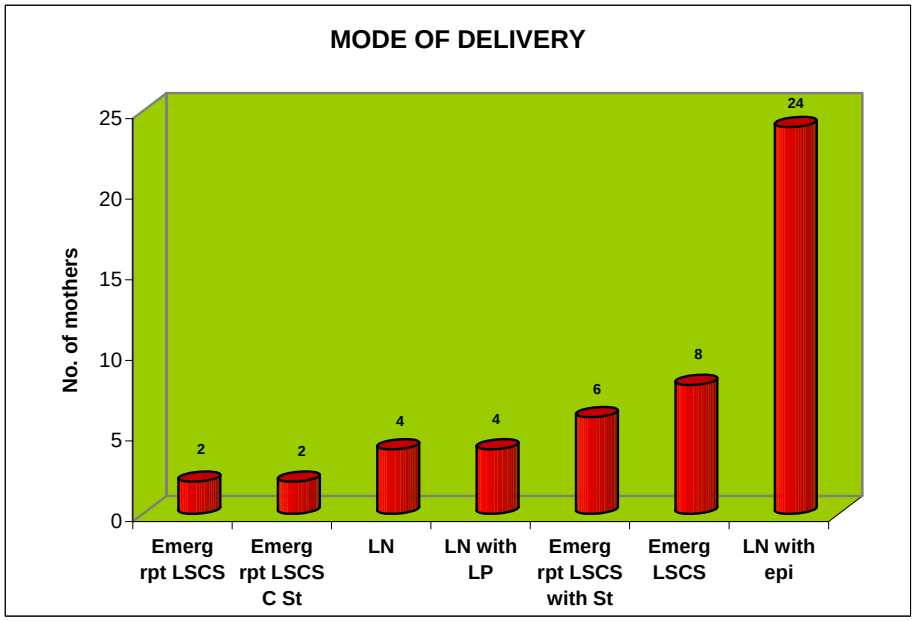
Among the fifty patients

* 44% (22/50) were $P_1 L_1$
* 40% (20/50) were $P_2 L_2$
* 8% (4/50) were $P_3 L_2$
* 6% (3/50) were $P_3 L_3$
* 2% (1/50) were $P_4 L_2$



V. Mode of Delivery

Mode of Delivery	No. of Mothers	%
Emergency LSCS	8	16.0
Emergency repeat LSCS	2	4.0
Emergency repeat LSCS with sterilization)	6	12.0
Labour Natural	4	8.0
Labour natural with LP	4	8.0
Labour natural with episiotomy	24	48.0
Total		



VI. Hemoglobin

	Mean (g/dl)	SD	Repeated Measures Anova F Test
Before iron sucrose	7.70	0.76	
After Month	10.32	0.74	F = 8694.2
At delivery	11.43	0.89	P = 0.001

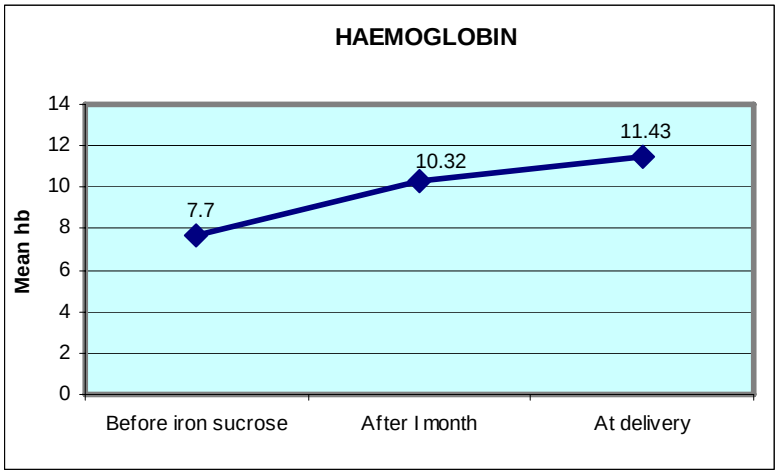
The change in hemoglobin level was significant

Before iron sucrose administration - Mean hemoglobin was 7.70mg / dl

1 month after iron sucrose administration, mean hemoglobin was 10.32 g / dl mean

At delivery, hemoglobin 11.43 g / dl.

p value 0.001 which was significant.



Multiple comparison using Bonferroni t-test

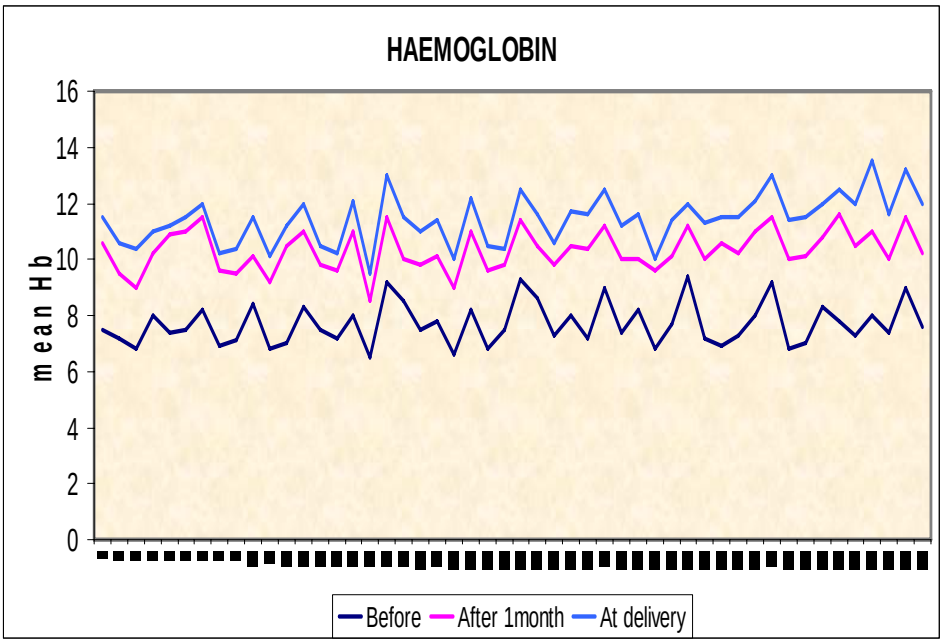
I	J	Mean Difference (I-J)	Significance	95% Confidence Interval for Difference(a)	
				Lower	After
Before iron sucrose	After I month	-2.618(*)	.001	-2.803	-2.433
	At delivery	-3.732(*)	.001	-3.938	-3.526
After I month	Before	2.618(*)	.001	2.433	2.803
	At delivery	-1.114(*)	.001	-1.256	-.972
At delivery	Before	3.732(*)	.001	3.526	3.938
	After I month	1.114(*)	.001	.972	1.256

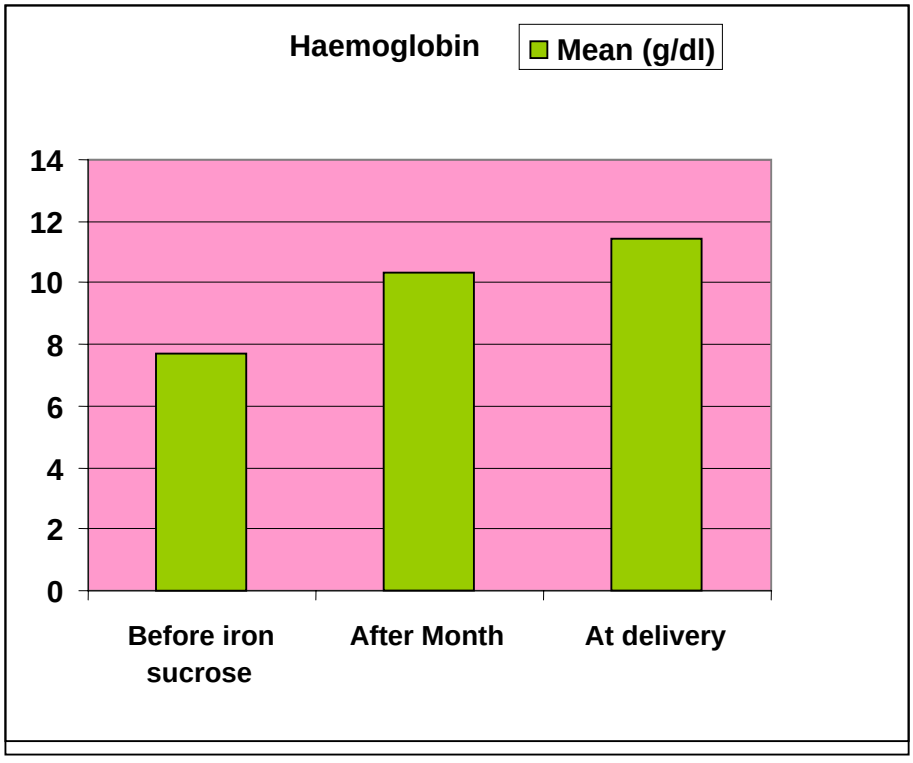
Based on estimated marginal means

* The mean difference is significant at the .05 level.

Hemoglobin

Hb	Mean difference (g/dl)	Std. Deviation	Paired t - test	Significance (2 – tailed)
Pair 1 After 1 month – Before	2.618	0.52	35.095	0.001
Pair 2 – At delivery Before	3.732	0.58	44.950	0.001
Pair 3 At delivery – After	1.114	0.40	19.470	0.001


HAEMOGLOBIN
mean Hb
16
14
12
10
8
6
4
2
0
Before
After 1month
At delivery


Haemoglobin
Mean (g/dl)
14
12
10
8
6
4
2
0
Before iron sucrose
After Month
At delivery

VII. SERUM FERITIN

	Mean	SD	Repeated meaures ANOVA F-test
Before iron sucrose	21.40	7.00	F=1816.1 P=0.001
After 1 month	85.03	15.96	
At delivery	177.29	27.65	

Mean Serum ferritin before iron sucrose administration was 21.40 μg /l

After 1 month of iron sucrose administration it was 85.03 μg/l

At delivery serum ferritin was 177.29 μg/l

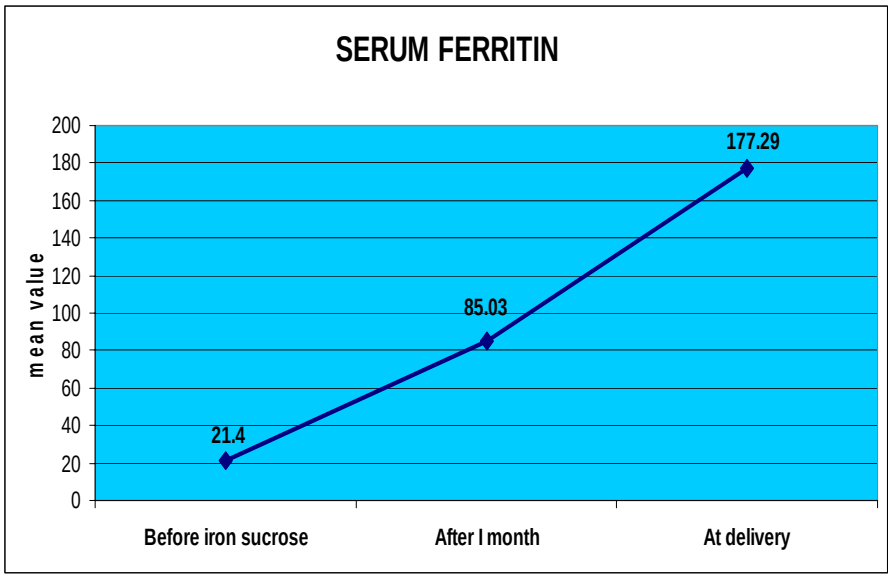
P value 0.001 & was significant

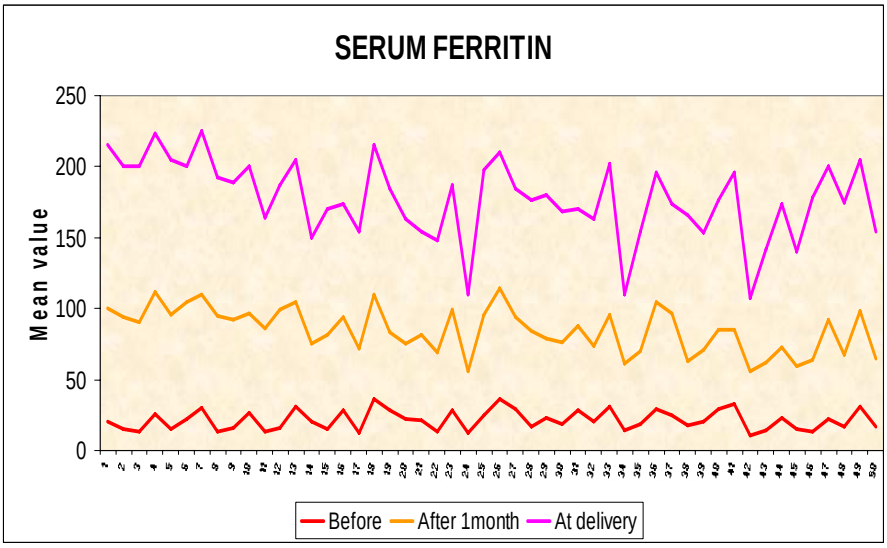
SR. FERRITIN		Mean difference	Std. Deviation	Paired t-test	Sig. (2-tailed)
Pair 1	After 1 month – Before	63.63	0.52	34.789	.001
Pair 2	At delivery - Before	92.26	0.58	45.530	.001
Pair 3	At delivery - After 1 month	92.26	0.40	41.671	.001

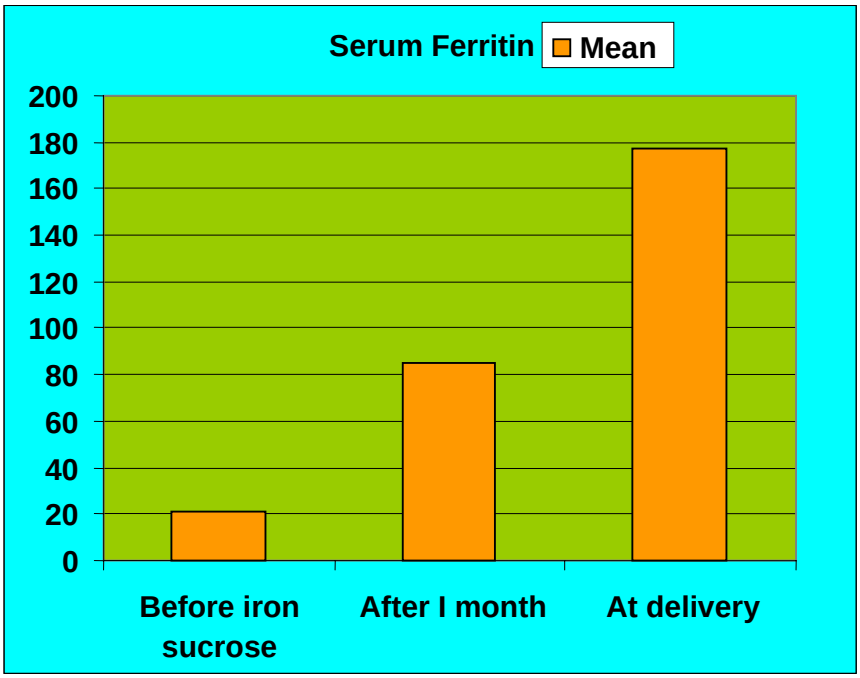
Multiple comparison using Bonferroni t-test

I	J	Mean Difference (I-J)	Significance	95% Confidence Interval for Difference(a)	
				Lower	After
Before iron sucrose	After I month	-63.630(*)	.001	-68.042	-59.218
	At delivery	-155.890(*)	.001	-164.378	-147.402
After I month	Before	63.630(*)	.001	59.218	68.042
	At delivery	-92.260(*)	.001	-97.748	-86.772
At delivery	Before	155.890(*)	.001	147.402	164.378
	After I month	92.260(*)	.001	86.772	97.748

* The mean difference is significant at the .05 level.




SERUM FERRITIN
Mean value
250
200
150
100
50
0
Before
After 1month
At delivery


Serum Ferritin
Mean
200
180
160
140
120
100
80
60
40
20
0
Before iron sucrose
After I month
At delivery

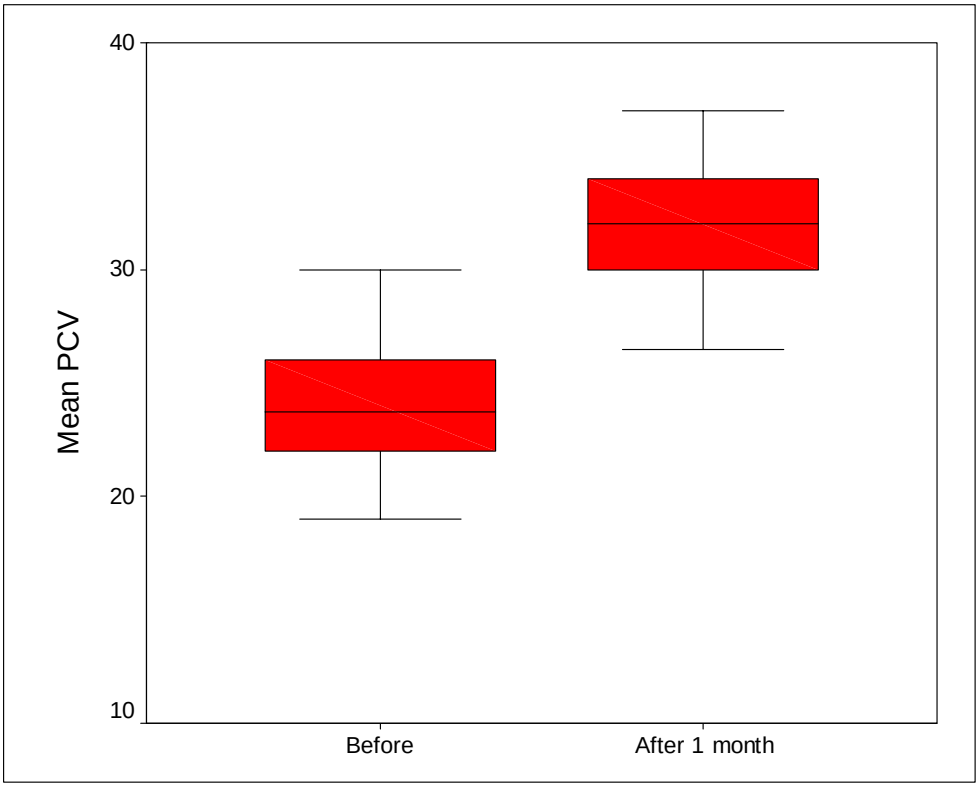
VIII. PCV – PACKED CELL VOLUME

	Mean	Std. Deviation	Paired t-test
Before iron sucrose	24.08	2.54	t=25.13
After one month	31.91	2.63	P=0.001***

* significant at p<0.05 ** significant at p<0.01 *** significant at p<0.001

- Mean PCV before administration of iron sucrose 24.08
- After one month mean PCV 31.91

  P value 0.001 & was significant



IX. SERUM IRON

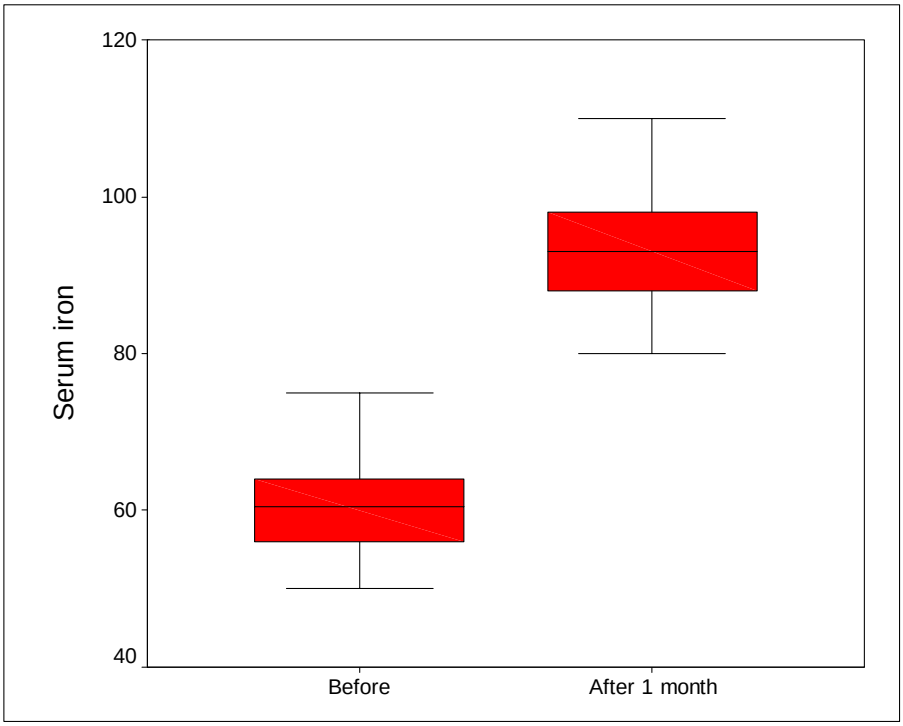
	Mean	Std. Deviation	Paired t-test
Before iron sucrose	60.82	6.140	t=34.78 P=0.001***
After one month	94.64	9.253	

significant at p<0.05 ** significant at p<0.01 *** significant at p<0.001

Mean serum iron before administration of iron sucrose 60.82 µg/dl.

1 Month after iron sucrose administration 94.64 µg/dl

P value 0.001 & was significant



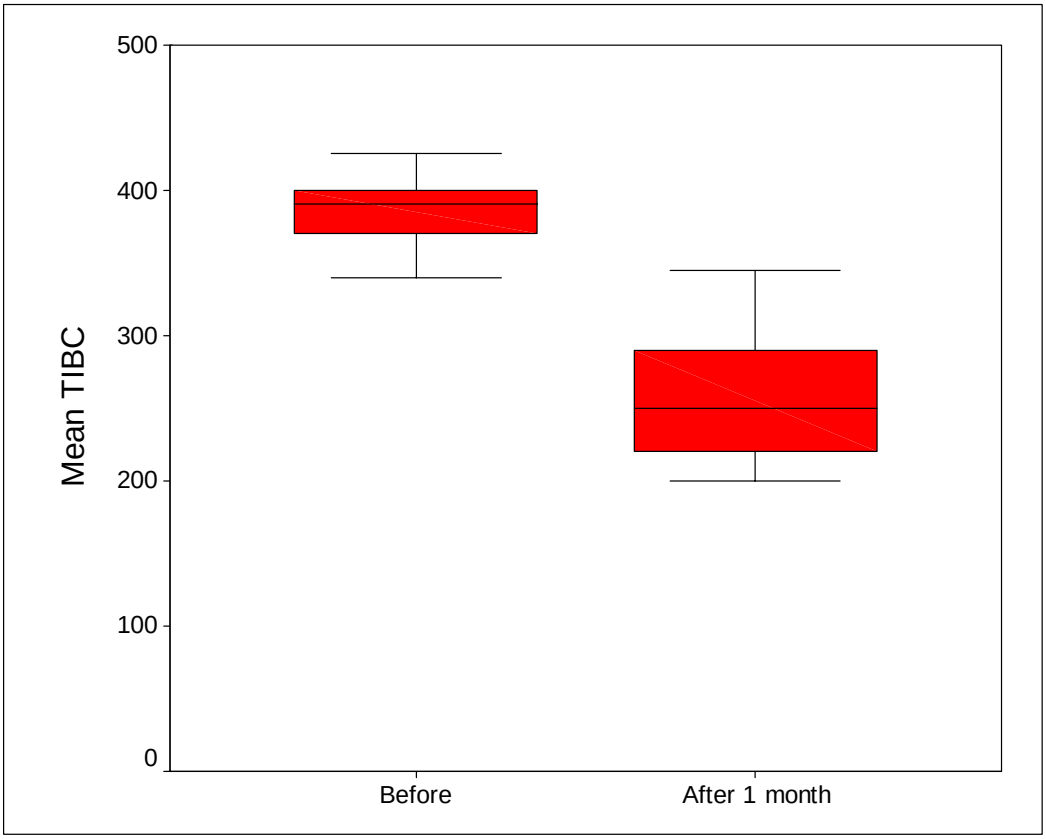
X. TOTAL IRON BINDING CAPACITY (TIBC)

	Mean	Std. Deviation	Paired t-test
Before iron sucrose	375.82	60.835	t=11.96
After one month	256.32	37.507	P=0.001***

* significant at p<0.05 ** significant at p<0.01 *** significant at p<0.001

Mean TIBC before iron sucrose administration was 375.82.

Mean TIBC 1 month after iron sucrose administration was 256. 32.

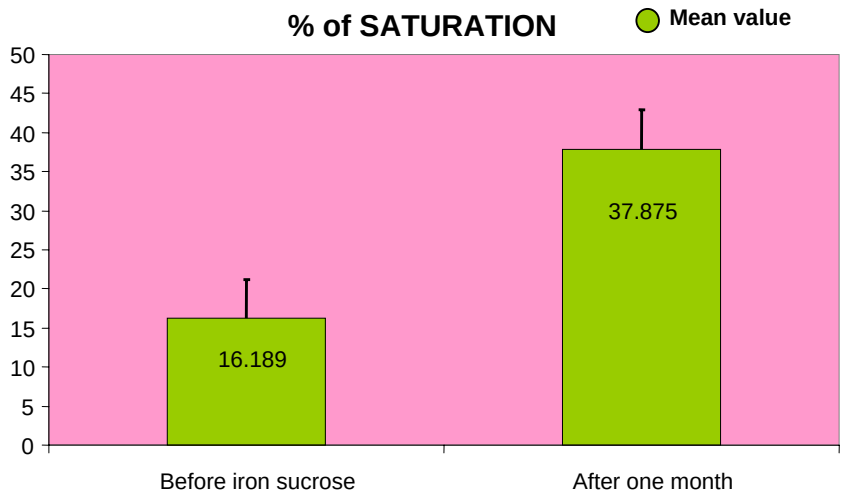


XI. PERCENTAGE SATURATION OF IRON

	Mean	Std. Deviation	Paired t-test
Before iron sucrose	16.189	3.5137	t=26.46
After one month	37.875	7.8960	P=0.001***

* significant at p<0.05 ** significant at p<0.01 *** significant at p<0.001

- Mean percentage saturation of iron before iron sucrose administration was 16.189%.
- Mean percentage saturation of iron 1 month after iron sucrose administration was 37.875%.

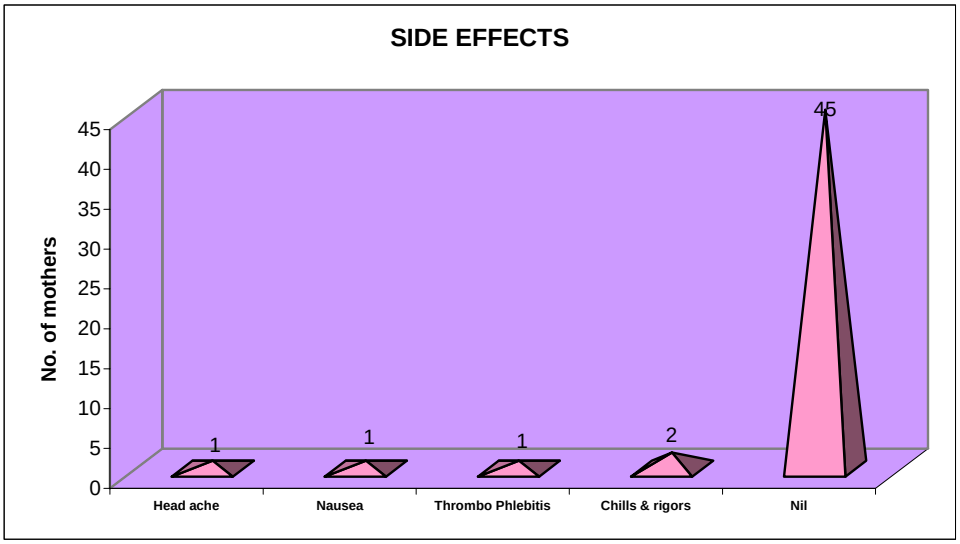


XII. SIDE EFFECTS

Side Effects	No. of mothers	%
Nil	45	90.0
Chills & rigors	2	4.0
Headache	1	2.0
Nausea	1	2.0
Thrombophlebitis	1	2.0
Total	**50**	**100.0**

Of the fifty patients 90% (45/50) had no side effects.

- 4% (2/50) had chills & rigors
- 2% (1/50) had headache
- 2% (1/50) had nausea
- 2% (1/50) had thrombophlebitis



DISCUSSION

In this study, 50 antenatal patients with iron deficiency anemia were selected according to the inclusion and exclusion criteria stated.

200 mg iron sucrose was given intravenously 2 doses 2 days apart and was followed up after 1 month interval & again at delivery.

- In our study, among the fifty patients 36% (18/50) were in age group 21 – 25 yrs.

  22% (11/50) were in age group 18 – 20 yrs.

  34% (17/50) were in age group 26 – 30 yrs

  8% (4/50) were in age group 31 – 35.

- In our study, among the fifty patients

  20% (10/50) were in class IV socio economic status

  80% (40/50) were in class V socio economic status

- Among the fifty patients in our study, 76% (38/50) were booked 24% (12/50) were unbooked.

- Obstetric code among the fifty patients, was
  * $P_1 L_1$ 44% (22/50)
  * $P_2 L_2$ 40% (20/50)
  * $P_3 L_3$ 6% (3/50)
  * $P_4 L_2$ 2% (1/50)

COMPARISON OF THE OUTCOME OF THE PARAMETERS

1. CHANGE IN HEMOGLOBIN:-

In our study of 50 antenatal patients, mean hemoglobin before starting treatment was 7.70 g/dl.

- 1 Month after iron sucrose administration mean hemoglobin was 10.32 g /dl.
- At delivery, mean hemoglobin was 11.43 g/dl.

The average raise is 3.73 g/dl with a p value < 0.05 which is statistically significant.

Change in Hemoglobin

Study	Rise in hemoglobin
Bayoumeu et al	1.6 $\pm$ 0.79 g/ dl
AI RA, Unlubilgin et al	2 g/dl
Divakar	0.9 – 2 g/dl
Wali A et al	1.9 – 2.8 g/dl
Krafft A et al	1.1 – 2 g/dl
Bhandal R Russel et al	3.7 g / dl
A. Dede et al	4.3 $\pm$ 0.6 g/dl
This study	3.73 g/dl

This study is similar to Bhandal R Russel et al study.

2. CHANGE IN FERRITIN

- In our study of 50 antenatal patients, mean serum ferritin was 85.03 μg/l.
- 1 Month after iron sucrose administration, mean serum ferritin was 85.03 μg/l.
- At delivery, mean serum ferritin was 177 – 20 μg/l.

The average rise is 155.89 μg/l. with p value < 0.05 is statistically significant.

Change in Ferritin:

Study	Rise in serum ferritin
Bayoumeu et al	Ferritin was higher after iron sucrose
AI RA, unlubilgin et al	Ferritin values were higher in intravenous iron sucrose group
Our study	Mean rise in serum ferritin 155.89 μg/L

3. CHANGE IN PACKED CELL VOLUME

- Mean packed cell volume before starting treatment 24.08.
- 1 Month after iron sucrose administration mean packed cell volume is 31.91.

The average rise in mean PCV is 7.83 with p value < 0.05 and is statistically significant.

4. CHANGE IN SERUM IRON

Mean serum iron before iron sucrose administration is 60.82 μg/dl.

Mean serum iron 1 month after iron sucrose administration is 94.64 μg/dl.

The average rise in mean serum iron is 33.82 μg/dl, with p value < 0.05 & is statistically significant.

Change in serum iron:

Study	Rise in serum iron
A. Dede, D.uygut et al	43.4 μg/dl
This study	33.82 μg/dl

Our study correlated with A.Dede et al and all other studies and showed rise in serum iron after intravenous iron sucrose.

5. CHANGE IN TOTAL IRON BINDING (TIBC) CAPACITY

Mean TIBC before iron sucrose administration is 375.82.

Mean TIBC 1 month after iron sucrose administration is 256.32.

The average decrease in mean TIBC is 119.5 with p value < 0.05 and is statistically significant.

Change in TIBC:

Study	Decrease in TIBC
A Dede et al	123.8
This study	119.5

6. CHANGE IN PERCENTAGE SATURATION OF IRON

Mean percentage saturation of iron before iron sucrose administration

16.189%.

Mean percentage saturation of iron 1 month after iron sucrose administration is 37.875%.

The average rise in mean percentage saturation of iron is 21.686% with p value < 0.05 and is satistically significant.

7. SIDE EFFECTS

90% (45/50) had no side effects.

4% (2/50) had chills & rigors

2% (1/50) had headache

2% (1/50) had nausea

2% (1/50) had thrombophalebitis.

Adverse reactions:

Study	Incidence of adverse reactions
N Bhandal, R Russel et al	Nil
Giannoulis et al	0.002%
Khurshid et al	Nil
Our study	10%

SUMMARY

In our study of 50 antenatal patients with iron deficiency anemia, were selected according to inclusion & exclusion criteria mentioned.

METHOD

200 mg of iron sucrose was given intravenously 2 doses 2 days apart, & followed up 1 month after administration of iron sucrose and at delivery.

The following parameters were assessed

1. Hb in g/dl
2. PCV
3. Serum ferritin in μg/l
4. Serum iron in μg/dl
5. Total iron binding capacity
6. Percentage saturation of iron.

THE RESULTS OF THE STUDY ARE AS FOLLOWS

1. Majority of patients around 36%, were in age group 21 – 25 yrs.
2. 80% of patients were in class V socio economic status.
3. 76% patients were booked
4. Majority of patients were multiparous
5. Average rise in hemoglobin after treatment was 3.73 g/dl with p value < 0.05, statistically significant.
6. Average rise in serum ferritin after treatment was 155.89 μg/l with p value < 0.05, statistically significant.
7. Average rise in packed cell volume after treatment was 7.83 with p value < 0.05, statistically significant.
8. Average rise in serum iron after treatment was 33.82 μg/ l with p value < 0.05, statistically significant.
9. Average rise in total iron binding capacity after treatment was 119.5 with p value < 0.05, statistically significant.
10. Average rise in percentage saturation of iron after treatment was 21.686% with p value < 0.05, statistically significant.

11. Side effect profile were very minimal with 90% patients (45/50) had no side effects

    2 patients had chills & rigors

    1 patients had headache

    1 patients had nausea

    1 patients had thrombophlebitis

    No anaphylactic reactions occurred.

CONCLUSION

1. Intravenous iron sucrose has become a major interest to prevent functional iron deficiency.

2. Iron sucrose has been found to be effective in improving hemoglobin, hematocrit, serum iron & ferritin values significantly in antenatal women with iron deficiency anemia

3. There is good tolerance to this formulation partly due to low allergenic effect and partly due to slow release of elementary iron from the complex.

4. By using intravenous iron sucrose to treat iron deficiency anemia in antenatal patients, the rate of blood transfusions could be reduced.

To conclude intravenous iron sucrose is safe, convenient and more effective therapy for treatment of iron deficiency anemia in antenatal patients requiring shorter period to achieve maximum hemoglobin concentration. It has convenient dosage and administration. It can be used to replace blood transfusion in antenatal period.

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PROFORMA

Study of efficacy of intravenous iron sucrose in treating anemia in antenatal women

Name **Occupation**

Age **Address**

IP Number **Phone Number**

Income **Socio-Economic Class**

Obstetric Score

Para

Live

Abortions

Presenting Complaints	Yes	No
H/o. easy fatigablility		
H/o breathlessness		
H/o swelling of legs		
H/o puffiness of face		
H/o haemetemesis / malena		
H/o passing worms in stool		
History suggestive of iron intolerance		

Past H/o

- H/o Diabetes mellitus, hypertension bronchial asthma, epilepsy, tuberculosis.
- H/o blood transfusion

Menstrual H/o

Age at menarche:

Cycles	Regular		Irregular
Flow	a)	Moderate	Excess
	b)	Lasting for	Days

Marital H/o

Married Since :

Obstetric H/o

Details of previous pregnancy / pregnancies:				
I. Any antenatal complication	**1**	**2**	**3**	**4**
1) APH 2) PIH 3) GDM 4) Others				
II. Outcome of pregnancy				
1. Spontaneous abortion 2. Induced abortion 3. Pre-term delivery 4. Term delivery a) Normal vaginal b) Vaginal instrumental c) LSCS				
III Place of delivery:				
Hospital / Nursing Home				
SC/ PHC / CHC				
IV Any Complication during Labour, delivery, puerperium a) No b) PPH c) Sepsis				
V) Baby's Status at birth Healthy live birth				

Birth weight Low birth weight Asphyxiated Still birth Congenitally malformed Large birth weight				
Present Pregnancy	Yes		No	
H/o antepartum haemorrhage H/o heart disease H/o diabetes H/o PIH H/o GDM				
General Examination	Yes		No	
Features of anemia 1. Pallor 2. Glossitis 3. Facial puffiness 4. Koilonychia				
Vital Signs				
Temperature Pulse rate Blood Pressure CVS RS				

INVESTIGATIONS		
1. Urine : Albumin Sugar Deposits 2. Blood Sugar Urea 3. Serum : Creatinine 4. Hemoglobin 5. Packed cell volume (Hematocrit) 6. Peripheral Smear 7. Serum iron 8. TIBC 9. Percentage saturation 10. Ferritin		
Parameters Monitored during therapy	Yes	No
1. Anaphylactic reaction (Hypotension) 2. Nausea/vomiting 3. Thrombo phlebitis 4. Chills / rigors 5. Joint pain 6. Headache		

Post therapy assessment		
a) The following parameters assessed 1 month after therapy		
1. Hb 2. Hematocrit 3. Serum iron 4. TIBC 5. Percentage Saturation 6. Ferritin		
b) The following parameters were assessed at delivery		
1) Hb 2) Ferritin		
Details of delivery		
1. Delivery	a) Term / Preterm b) Vaginal / Assisted vaginal / Caesarean section	
2. Complications during delivery If yes, specify 3. Post delivery complication If yes, specify		

ABBREVIATIONS

IV	Intravenous
IM	Intramuscular
Fe	Iron
Hb	Hemoglobin
PCV	Packed Volume
TFR	Transferrrin Receptor
rHuEPO	Recombinant Human Erythropoietin

BEFORE IRON SUCROSE												AFTER 1 MONTH						AT DELIVERY		
S.No.	**NAME**	**AGE**	**OS**	**MOD**	**HB**	**PCV**	**SR. IRON**	**TIBC**	**% SAT**	**PS**	**S.FER**	**HB**	**PCV**	**SR. IRON**	**TIBC**	**% SAT**	**S.FER**	**SE**	**HB**	**S.FER**
1	Egavalli	26	P_2L_2	6	7.5	21.5	63	395	15.9	MH	20	10.6	32	98	230	42.6	100	NO	11.5	215
2	Vennila	20	P_1L_1	2	7.2	24	54	3.95	13.6	MH	15	9.5	26.5	80	340	23.5	94	NO	10.6	200
3	Latha	19	P_2L_2	6	6.8	22	50	412	12.1	MH	13	9.0	28.2	83	345	24	90	NO	10.4	200
4	Jothi	23	P_1L_1	2	8.0	25.5	64	400	16.0	MH	26	10.2	32	92	290	31.7	112	NO	11.0	223
5	Thilaga	22	P_2L_2	2	7.4	22	60	380	15.7	MH	15	10.9	35	98	214	45.7	96	NO	11.2	205
6	Vimala	24	P_2L_2	2	7.5	23	61	370	16.4	MH	22	11.0	36	94	220	42.7	105	NO	11.5	200
7	Kala	25	P_2L_2	2	8.2	27	60	400	15.0	MH	30	11.5	37	96	290	33.1	110	NO	12.0	225
8	Nagammal	30	P_3L_3	3	6.9	23	67	410	16.3	MH	13	9.6	30	100	270	37	95	NO	10.2	192
9	Prema	20	P_1L_1	4	7.1	21	59	400	14.75	MH	16	9.5	29	90	296	30.4	92	NO	10.4	189
10	Lalitha	24	P_2L_2	6	8.4	26	62	410	15.1	MH	27	10.1	32	105	230	45.6	97	NO	11.5	200
11	Kavitha	23	P_2L_2	5	6.8	23	52	415	12.5	MH	13	9.2	28	85	215	39.5	86	NAU	10.1	164
12	Jaya	18	P_1L_1	4	7.0	21	61	375	16.2	MH	16	10.5	33	98	215	45.5	98.5	NO	11.2	186.5
13	Anitha	26	P_1L_1	2	8.3	27	64	352	18.1	MH	31	11.0	34	85	230	36.9	105	NO	12.0	205
14	Jeyanthi	27	P_2L_2	3	7.5	24	62	380	16.3	MH	20	9.8	30	80	250	32	75	NO	10.5	150
15	Devi	21	P_2L_2	2	7.2	23	58	400	14.5	MH	15	9.6	31	84	275	30.5	82	NO	10.2	170
16	Jothi	22	P_1L_1	2	8.0	25.1	54	392	13.7	MH	28	11.0	34	87	270	32.2	94	NO	12.1	174
17	Poongodi	19	P_1L_1	2	6.5	19	65	400	16.2	MH	12	8.5	24	88	290	30.3	72	NO	9.5	154

BEFORE IRON SUCROSE												AFTER 1 MONTH						AT DELIVERY		
S.No.	NAME	AGE	OS	MOD	HB	PCV	SR. IRON	TIBC	% SAT	PS	S.FER	HB	PCV	SR. IRON	TIBC	% SAT	S.FER	SE	HB	S.FER
18	Prabha	32	P_2L_2	3	9.2	28.5	76	325	23.3	MH	36	11.5	34	100	210	47.6	110	NO	13.0	215
19	Uma	25	P_2L_2	6	8.5	26	60	390	15.3	MH	28	10.0	31	92	215	42.7	83	T	11.5	184
20	Meena	27	P_2L_2	2	7.5	23	52	406	12.8	MH	22	9.8	30	88	275	32	75	NO	11.0	163
21	Priya	27	P2L2	2	7.8	23	62	400	15.5	MH	21	10.1	32	96	295	32.5	82	NO	11.4	154
22	Latha	23	P1L1	4	6.6	20.5	61	410	14.8	MH	13	9.0	28	90	290	31.03	69	NO	10.0	148
23	Elizabeth	18	P1L1	2	8.2	25.5	64	380	16.8	MH	28	11.0	34	94	240	39.1	99	NO	12.2	187
24	Ramya	29	P3L2	6	6.8	20.5	58	340	17.05	MH	12	9.6	29	89	220	40.4	56	H	10.5	110
25	Vimala	23	P1L1	2	7.5	23	50	400	12.5	MH	25	9.8	29.5	85	285	29.8	96	NO	10.4	198
26	Malathi	28	P1L1	2	9.3	28	70	320	21.8	MH	36	11.4	35	115	215	53.48	114	NO	12.5	210
27	Sheela	20	P2L2	3	8.6	26	66	340	19.4	MH	29	10.5	32	88	210	41.9	94	NO	11.6	184
28	Megala	30	P4L2	1	7.3	23	54	395	13.6	MH	17	9.8	29.5	92	250	36.8	84	NO	10.6	176
29	Jaya	24	P1L1	2	8.0	25	58	410	14.1	MH	23	10.5	32	98	310	31.6	79	NO	11.7	180
30	Sumathy	22	P1L1	4	7.2	22	62	385	16.1	MH	19	10.4	31.5	110	300	36.6	76	NO	11.6	168
31	Nithya	27	P2L2	5	9.0	28	70	380	18.42	MH	28	11.2	34	115	200	57.5	88	NO	12.5	170
32	Jothi	32	P3L2	1	7.4	23.5	54	400	13.5	MH	20	10.0	31.5	90	2800	32.1	74	NO	11.2	163
33	Kaa	19	P1L1	4	8.2	25	64	350	18.2	MH	31	10.0	32	106	246	43.08	96	NO	11.6	202
34	Nagammal	24	P1L1	2	6.8	20	65	425	15.2	MH	14	9.6	28.5	90	300	30	61	NO	10.0	110

BEFORE IRON SUCROSE												AFTER 1 MONTH						AT DELIVERY		
S.No.	NAME	AGE	OS	MOD	HB	PCV	SR. IRON	TIBC	% SAT	PS	S.FER	HB	PCV	SR. IRON	TIBC	% SAT	S.FER	SE	HB	S.FER
35	Indhira	28	P2L2	6	7.7	25	54	400	13.5	MH	19	10.1	32	87	290	30	70	NO	11.4	154
36	Prabha	29	P2L2	6	9.4	30	72	325	22.1	MH	29	11.2	35	110	210	52.3	105	NO	12.0	196
37	Poongodi	19	P1L1	2	7.2	21.5	56	410	13.6	MH	25	10.0	31.5	90	280	32.14	97	NO	11.3	174
38	Meena	34	P3L3	1	6.9	22.5	64	410	15.3	MH	18	10.6	32.5	104	290	35.8	63	C	11.5	166
39	Manimegalai	20	P1L1	4	7.3	23	60	390	15.38	MH	20	10.2	33.5	98	210	46.6	71	NO	11.5	153
40	Faizana	26	P2L2	2	8.0	26	62	410	15.12	MH	29	11.0	35	105	295	35.5	85	NO	12.1	176
41	Priya	26	P_1L_1	4	9.2	28.5	72	320	32.5	MH	33	11.5	35	115	200	57.5	85	NO	13.0	196
42	Kamul Nisha	32	P_3L_3	1	6.8	21	56	410	13.6	MH	11	10.0	32	90	280	32.1	56	NO	11.4	107
43	Jasmine	24	P_1L_1	2	7.0	22	58	400	14.5	MH	14	10.1	30.5	96	275	34.9	62	NO	11.5	142
44	Mariam	27	P_2L_2	2	8.3	26.5	56	385	14.54	MH	23	10.8	35	98	255	38.4	73	NO	12.0	174
45	Ponni	25	P_1L_1	4	7.8	24.5	54	390	13.8	MH	15	11.6	36	96	250	38.4	59	NO	12.5	140
46	Geetha	20	P_1L_1	2	7.3	23.5	60	385	15.5	MH	13	10.5	32	85	250	34	64	NO	12.0	178
47	Meena	23	P_2L_2	3	8.0	25.5	66	340	19.4	MH	22	11.0	34	97	215	45.1	92	C	13.5	200
48	Rajeswari	26	P_3L_2	2	7.4	24	56	365	15.3	MH	17	10.0	31.5	83	245	33.8	67	NO	11.6	175
49	Shanthi	30	P_3L_2	6	9.0	29	75	320	23.4	MH	31	11.5	33.5	110	225	48.8	98	NO	13.2	205
50	Mary	24	P_1L_1	2	7.6	24	58	380	15.2	MH	17	10.2	31.5	87	235	37.02	65	NO	12.0	154

KEY TO MASTER CHART

MOD	**-**	**Mode of Delivery**
1	-	Labour Natural
2	-	Labour Natural with Episiotomy
3	-	Labour Natural with Lacerated Perineum
4	-	Emergency LSCS
5	-	Emergency repeat LSCS
6	-	Emergency repeat LSCS with Sterilisation
OS	-	Obstetric Score
PS	-	Peripheral Smear
MH	-	Microcytic Hypochromic picture
% SAT	-	Percentage Saturation
S. FER	-	Serum Ferritin
TIBC	-	Total Iron Binding Capacity
SE	-	Side Effects
N	-	No Side Effects
NAU	-	Nausea and Vomiting
H	-	Headache
T	-	Thrombophlebitis
C	-	Chills and Rigors