IRON PROPHYLAXIS IN PREGNANCY-

INTRAVENOUS VERSUS ORAL ROUTE

DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT
FOR
M.D DEGREE
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OBSTETRICS & GYNECOLOGY,
MADRAS MEDICAL COLLEGE,
CHENNAI - 3.

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that the dissertation entitled "**IRON PROPHYLAXIS IN PREGNANCY – INTRAVENOUS VERSUS ORAL ROUTE"** AT ISO-KGH is a bonafide work done by Dr.R.DHEEBA JAYANTHI in the Institute of Social Obstetrics, Govt Kasturba Gandhi hospital(Madras Medical College) Triplicane , Chennai in partial fulfillment of the university rules and regulations for award of MD degree in Obstetrics and Gynecology under my guidance and supervision during the academic year 2010-2013.

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DECLARATION

I. Dr.R.Dheeba jayanthi , solemnly declare that the dissertation titled "**IRON PROPHYLAXIS IN PREGNANCY – INTRAVENOUS VERSUS ORAL ROUTE"** was done by me at The Institute of social obstetrics, Govt Kasturba Gandhi Hospital, Madras Medical College during 2010-2013 under the guidance and supervision of Prof. **Dr.P.M.GOPINATH MD,DGO.** This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in Obstetrics and Gynecology. This has not been submitted previously by me for the award of any degree or diploma from any other university.

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To
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Dear Dr. R. Dheeba Jayanthi

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Iron prophylaxis in Pregnancy : Intravenous versus oral route " No. 12012012.

The following members of Ethics Committee were present in the meeting held on 27.01.2012 conducted at Madras Medical College, Chennai -3.

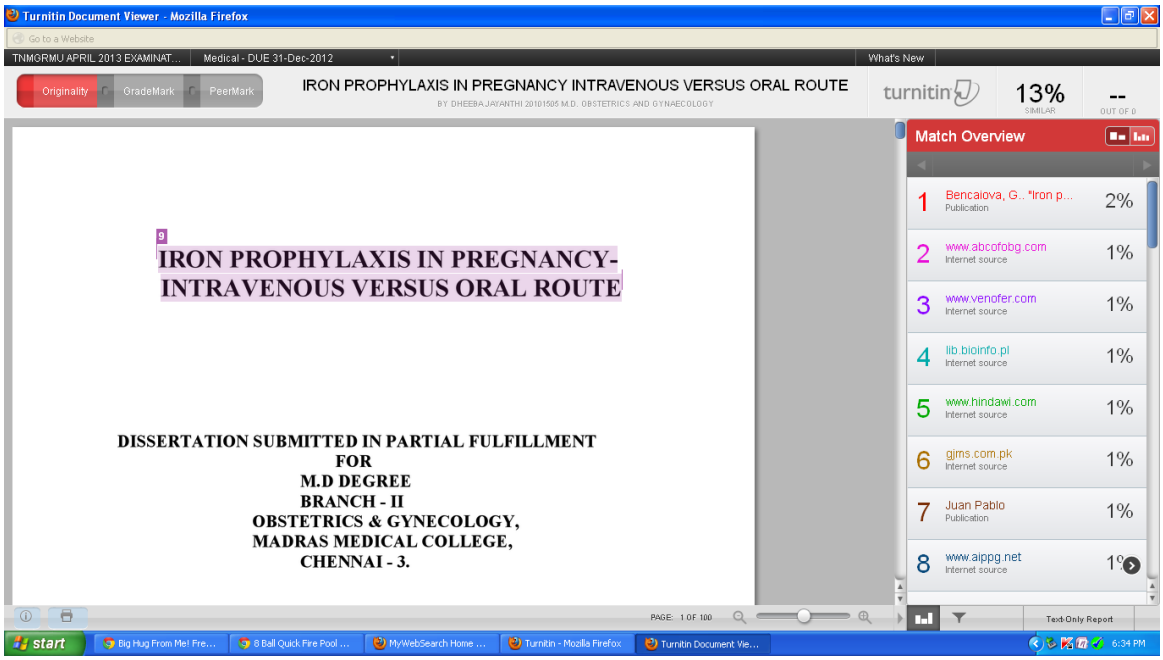
1.	Prof. S.K. Rajan. MD	-- Chairperson
2	Prof. Pregna B. Dolia MD Vice Principal, Madras Medical College, Chennai -3 (Director , Institute of Biochemistry, MMC, Ch-3)	-- Member Secretary
3.	Prof. B. Kalaiselvi. MD Prof of Pharmacology ,MMC, Ch-3	-- Member
4.	Prof. Shruti Kamal MS Prof of Surgery, Madras Medical College., Ch-3	-- Member
5.	Thiru. S. Govindsamy. BA BL	-- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE 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.

Member Secretary, Ethics Committee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INTRODUCTION

INTRODUCTION

Child birth should be a joyous event. Pregnancy is a physiological Phenomenon. The physiological effects of pregnancy should be taken care at the earliest and we should not let one die of physiological phenomenon.

Iron is very important for the normal function and development of the organs in the fetus. In order to prevent iron deficiency in the fetus, we have to ensure an adequate iron status in pregnancy.

Maternal mortality is around 350- 450/100000 live births in India. Anaemia contributes 20% of maternal deaths. Risk of perinatal mortality is nine times higher. Bayley mental development index shows poor performance in children born to mothers affected by iron deficiency anemia.

Most common nutritional disorder affecting majority of population in the world is Iron deficiency. About 25% of world population is affected by iron deficiency. Pregnant women are at high risk of iron deficiency anemia, because of increased iron needs during pregnancy.

Iron deficiency anemia in mother during pregnancy leads to Iron deficiency anemia in infancy and childhood. At two months of age and beyond the iron reserves in infants doubles in women taking iron when compared to unsupplemented mothers. The definite solution for this is to eradicate iron deficiency during pregnancy.

There is a wide geographical variation in the incidence of iron deficiency and iron deficiency anemia in pregnancy . Because of high levels of anemia in pregnancy, National Nutritional Anaemia control programme was started in 1970. This is now called as Iron and Folic acid program. According to NNACP "iron and folic acid tablets must be given to all antenatal mothers from second trimester to three months of lactation".

This Program has been taken up by the maternal and child health division of health and family welfare. It is now part of Reproductive and child health program. In this Pregnant women were recommended to have one tablet per day containing 100 mg of elemental iron and 500 μg folic acid for 100 days from second trimester of pregnancy till 3 months of lactation. Preschool children (aged 1–5 years) were recommended to take one tablet (pediatric tablet contains 20 mg iron and 100 μg folic acid) per day for 100 days every year.(5)

The rationale behind the IFA program was to decrease the incidence and Prevalence of anemia in the reproductive age group women (4).

Number of studies, surveys or audits was conducted to assess the prevalence of anemia in women in India, so as to assess the impact of IFA Program. The prevalence according to various studies is as follows.

According to NFHS I (1995) only 40% of rural women receive care from a physician during at least one antenatal visit and out of them Only 45% receive Iron – Folic acid tablets. (1)

According to NFHS- II (2002) In pregnant women the prevalence of anemia was 49.7% . In breast feeding women the prevalence is around 56.4%.Among non pregnant and non breast feeding women the prevalence is around 50.4%.(2)

NFHS – III (2008) was carried out in 2005-2006. According to NFHS –III prevalence of anaemia is 57.9 % in pregnant women, 54.6% in urban and 59% in rural.(3)

District Level Household Survey (DLHS 2, 2006) is one of the largest health surveys conducted, involving 700,000 households covering 563 districts of the country. According to this study70% of pregnant women and adolescent girls in the country were anemic.

After 30 years of starting IFA Program, still the prevalence of iron deficiency anemia in pregnancy is on the rising trend. The prevalence is in the range of 50- 60%.

The reasons for failure of IFA program are

- Partial coverage of population
- Defective absorption due to intestinal infestation
- Improper consumption
- Diet with high iron chelators
- Failure to replenish stores
- Because of lack of health education and supervision
- Failure with delivery system
- Failure to deworm prior to iron supplementation

The solution for the failure of delivery system is to include NGO, school system, women's club with community leaders who could distribute the tablets so that the iron tablets would reach the women at their door steps.

There is evidence to say that, even when the women receive the tablets, actual consumption is not ensured.

NFHS – II (1998-1999) conducted a study for urban slum dwellers in Madhya Pradesh. According to the study, only 61.1% of pregnant women consumed the required number of tablets (6)

According to RCH report from Government of India, even in areas of high coverage of 86.2%, only 11.5% of them actually consumed them for more than 3 months during their pregnancy.

According to Nutrition Foundation India Survey (2003) pregnant women who received IFA tablets were only 32% and out of them only 4.9% consumed it.

Indian Council of Medical Research conducted a study on why women would accept IFA tablets but failed to consume the required numbers (ICMR 1985, 1989) 7, 8

The reasons are as follows:

- ✓ Some women did not like the taste of the tablet.
- ✓ Some women found the tablets foul smelling.
- ✓ Some forgot to consume the tablet due to household workload.
- ✓ Some women believe that consumption of iron tablets results in big baby resulting in difficulty in delivery.

The side effects of oral iron supplements increases with higher dosages and leads to poor compliance. We can minimize this by prescribing single tablet at bedtime or by biweekly regimen.

According to World Health Organization (WHO), oral iron program have often failed to reduce frequency of iron-deficiency anemia. High levels of iron-deficiency anemia exist in pregnancy despite routine use of iron prophylaxis adopted by many centres in the developing world.

The WHO technical working group on the prevention and the treatment of severe anemia has stated that parenteral iron therapy produces a rapid and complete correction of iron deficiency, including replacement of iron stores producing a more rapid erythropoietic response than oral iron replacement.

Fresh thinking is therefore necessary to overcome the reasons for failure of IFA program. It has been widely studied that intravenous iron Supplementation is very effective in the treatment of iron deficiency anemia in pregnancy. There is strong evidence to show that intravenous iron sucrose show a rapid improvement in iron deficiency anemia when compared to oral iron**.**

As iron sucrose is given intravenously, it overcome the problem of poor compliance which is one of the most common reason for failure of IFA program. The side effects are minimal with iron sucrose, anaphylactic reactions are rare with iron sucrose when compared with intravenous iron dextran. Iron sucrose can be given to all pregnant women irrespective of iron

status at the time of booking and at the time of antenatal visit under the supervision. So all the women restore body iron stores more rapidly, and a prompt increase in hemoglobin is more likely to be achieved.

We conducted a study to assess the efficacy and safety of intravenous iron sucrose versus oral ferrous sulphate in the prophylaxis of gestational anemia

AIM OF THE STUDY:

A. To assess the response of intravenous iron sucrose with that of oral ferrous sulphate in the prophylaxis of Iron deficiency anemia in pregnancy.

B. To compare the efficacy and safety of two and three doses of intravenous iron sucrose with daily oral ferrous sulphate in the prophylaxis of gestational anemia in pregnant women.

C. To compare the acceptability, efficacy and side effects of injectable iron sucrose over oral ferrous sulfate in the prophylaxis of anemia in pregnancy.

PRIMARY OBJECTIVE:

Whether Iron sucrose can be used as an alternative in the prophylaxis of iron deficiency anemia in pregnancy, so that maternal morbidity and Mortality due to anemia can be reduced.

REVIEW OF LITERATURE

1. International journal of gynaec and obstetrics (2005) 90,238- 239. A.Dede etal D.Uygus, B.Yilmaz T.mungan, M.Ugur They have done a comparative study in the treatment of iron deficiency in postnatal women. 75 women whose Hemoglobin <=9g/dl were included in the study. They were allotted into one of two groups , 50 patients in intravenous group and 25 patients in oral group. Patients in intravenous group were given calculated dose of iron sucrose and patients in oral group received 300mg tablets of ferrous sulphate thrice daily one hour before meals. Blood was taken before starting treatment and at days 7 and 28.The following parameters were assessed Hb%, Hematocrit, C Reactive protein, serum ferritin, serum iron mean corpuscular volume (MCV) and serum iron binding capacity. They concluded that intravenous iron therapy with iron sucrose significantly raised the serum ferritin level within a short period of time with minimal side effects than oral iron therapy in postpartum iron deficiency anemia(53).

2. British journal of Obstetrics and Gynaecology, Sep 2006; 1248-1252. Bhandal.N et al .Russell. R This was a prospective randomized study in the

treatment of iron deficiency anemia in postnatal mothers. After 24-48 hours of delivery Hb was done and 44 women with Hb< 9 g/dl were included in the study. 22 women in group A were given 200 mg of iron sucrose on days 2 and 4. 22 women in oral group were given 200 mg tablet of ferrous sulphate twice a day for 6 weeks.

They found an average rise in Hb of 2.5gm/dl on day 5 in intravenous group and in the oral group average rise was 0.7gm/dl . There were no serious adverse effects in intravenous group .One third of women in oral group had gastrointestinal side effects.

They concluded that in women with postpartum iron deficiency anemia, higher level of blood Hemoglobin was achieved in iron sucrose group when compared to oral group(52).

3. C Giannoulis et al A Danniilides, T Tantanasis, K Dinas, and J Tzafettas. Hippokratia, 2009 Jan – Mar: 13(1): 38 – 40. The efficacy of intravenous versus oral iron was compared in 104 anaemic postpartum women. Women with hemoglobin of <8 g/dl and ferritin < 10μg/dl were included in the study.

They concluded that in intravenous group the mean rise in Hb level was 4.6g/dl and of mean rise in ferritin level was 105 mg/l. In oral group the mean rise in Hb level was2.3 g/dl and the rise in ferritin level was 68mg/dl.

They observed a Significant difference between the two groups, in the rise of hemoglobin level (p=0.0001) and also in the rise of ferritin level (p=0.000). The efficacy of intravenous iron sucrose was proved in the treatment of iron deficiency anemia in the postpartum period(54).

4. Khurshid shabbir Raja et al , (Rawal Medical Journal 2003;28:40-43). This study was carried out in anemic pregnant women to study the efficacy of intravenous iron sucrose. Fifty antenatal women with hemoglobin 8gm/dl were included in the study. Calculated dose of iron sucrose was given intravenously over several sessions. Hemoglobin (Hb) levels, serum ferritin, and mean corpuscular volume measured. They noted significant rise in Mean Hemoglobin of 3.5 g/dl. Significant rise in Serum ferritin and MCV were noted. Iron sucrose complex , significantly improved the hematological parameters(55).

5. Al-Momen Ak et al, al-Mechari A; al-Nuaime L in 1996 conducted a study comparing Intravenous iron sucrose and oral ferrous sulphate. Significantly higher Hb level ($p<=0.001$) were observed in iron sucrose group when compared to oral group.No major side effects were noted in the Iron sucrose group. In the oral group there was about 30% of poor compliance(50).

6. Scott B et al. silverstein and George M Rodgers According to them , the increased availability of parentral iron preparation should decrease the need to use red cell transfusion in patients with iron deficiency anemia. They observed that increase in Hb is observed after one week of iron sucrose administration and serious anaphylactic hypersensitivity of 0.002% in intravenous iron sucrose group compared to 0.6-0.7% in intramuscular iron dextran group.

7. Gravier A et al , Descargues G, Marpeace L et al (1999) conducted a study on how to avoid postpartum blood transfusions in Iron deficiency anemia patients, by treating with I.V Iron sucrose. They concluded that I.V Iron sucrose is effective in preventing unnecessary blood transfusions in postpartum patients(57)

8. Al Momen et al conducted a prospective study in 111 pregnant women with iron deficiency anaemia. Women with Hb <9gm/dl were included in the study and divided into 2 groups. Intravenous iron sucrose was given as an infusion in study group. 100mg of iron sucrose in 100ml of normal saline was given every 1 to 3 days. Controls were given 100mg of Iron dextran on alternate days till the calculated dose was given.

Intravenous iron sucrose group achieved a higher levels of Hb, with in short period of time when compared with controls. Adverse effects were less with iron sucrose when compared to iron dextran group. 32% of patient in iron dextran group were non compliant.

9. Catherine Gay et al (2005) concluded that although oral iron is the standard treatment for iron deficiency anemia, it is poorly tolerated and has low efficacy in rapid correction of anemia; But I.V iron sucrose is both quick and effective in treating anemia. The average mean rise of Hb was 0.8 gm/dl for oral iron, 3.5 gm/dl for blood transfusion and 3.1gm/dl for I.V iron after 14 days. No serious adverse effects were noted for I.V iron sucrose.

10. Bayoumeu Fet al, Subiran – Buisset E, Baka NE et al (2002) American Journal of obst. and Gynaecology also observed the effectiveness, safety and tolerability of I.V iron sucrose compared to oral iron for treatment of iron deficiency anemia in pregnant women.

11. Chamate E et al . conducted a study on treatment of iron deficiency anemia in pregnancy and immediate puerperium comparing I.V iron sucrose and oral ferrous sulphate and concluded that I.V iron sucrose is safe, convenient and more effective with less adverse effects and it can replace blood transfusion in antenatal period.

12. Gabriela Bancaiova et al Ursula Von Mandach, Roland Zimmerman- European journal of obstet and gynecology and reproductive biology, vol 144 issue2 – June 2009; 135-139- done a comparative study between iron sucrose and oral iron in the prophylaxis of gestational anemia in pregnant Women. Group A women were given two dose of 200mg iron sucrose Intravenously 4 weeks apart after 20 weeks . Group B women were given three doses of 200 mg of iron sucrose per dose intravenously 4 weeks apart. Group C women were given 100 tablets of ferrous sulphate for 100 days. There was no clinically significant difference in the hematological parameters in the iron sucrose group when given as prophylaxis in pregnant women.

13. Kochhar et al , P. K., Kaundal, A. and Ghosh, P. (2012), Intravenous iron sucrose versus oral iron in treatment of iron deficiency anemia in pregnancy: A randomized clinical trial. Journal of Obstetrics and Gynecology Research. doi: 10.1111/j.1447-0756.2012.01982.x This was a prospective study, where 100 antenatal women with hemoglobin 7–9 g/dL, MCV <85 fL and S. ferritin <15 ng/mL, were randomized into one of the two groups. For women belonging to Group A 200 mg tablets of ferrous sulphate were given three times daily for 4 weeks. 200 mg of iron sucrose was given on alternate days by slow intravenous infusion to the women belonging to group B. The parameters assessed were hemoglobin, reticulocyte count, red blood cell indices on days 7, 14, 21, and 30 and at delivery, and ferritin was measured on day 30 and at delivery. Side-effects of treatment and the neonatal outcome were studied as secondary outcome measures.

Results: **S**tatistically significant difference in rise of hemoglobin levels (3.1 g/dL in group A vs 5.1 g/dL in group B; $P = 0.002$) and ferritin levels were noted between the two groups on day 30 ($P = 0.005$).

The adverse effects were more in oral group when compared to intravenous iron sucrose group. Neonatal outcome was comparable in the two groups.

14. Gowda et al (2010) conducted a randomized prospective study to evaluate the safety and efficacy of intravenous iron sucrose. Antenatal women whose Hb less than 8 g/dl were included in the study. Hematological parameters were done on day 1,7,28. Calculated dose of iron sucrose 200mg/dose was given on alternate days. Iron sucrose was found to be safe and effective in the treatment of iron deficiency anemia in pregnancy. This study was sponsored by NRHM, Govt. of Karnataka, to study the efficacy and safety in pregnancy as a pilot project. After conclusion of this study, the Government made iron sucrose injection for use at Government hospitals. This will benefit the society at large.

15. Surriaya Halimi et al, Syed Muhammad Ashhad Halimi, Muhammad Shoaib conducted a comparative study to evaluate the efficacy of oral versus parenteral iron therapy for correction of anemia in pregnant women.

100 pregnant women with Hb less than 11gm/dl and heamatocrit less than 33% were selected and divided into two groups. Group-I for oral and Group-II for intravenous administration of iron. The treatment results were assessed on day 30 of the treatment.

Results: There was a significant rise in Hemoglobin in intravenous group on day 30 when compared to oral group.

They concluded that Parenteral iron therapy in form of iron sucrose is better choice to correct iron deficiency anaemia related to pregnancy.

16. Intravenous Iron Therapy for IDA in Pregnancy: (Divakar, Manyonda, *et al*. 2009) They conducted a preliminary study of the efficacy, safety, and feasibility of the use of IV iron sucrose in iron deficiency anemia of pregnancy. 96 women who were anemic were enrolled in the study. These women had been given IFA tablets from the time of booking and for a minimum period of 4 weeks. when their Hb had remained below 11 g/dL after 4 weeks of therapy they are declared as '' failed to oral therapy". Among them, those who were willing to receive IV iron sucrose were included in the study. The mean gestational age was 23.5 weeks (range 20–34 weeks gestation) and the mean Hb was 9.2 g/dL (range 6.8–10.8 gm/dL). All the participants were dewormed with two tablets of Mebex Plus, prior to intravenous iron therapy. These women received two doses of IV iron sucrose of 200 mg per sitting, 3–5 days apart, on an outpatient basis and their Hb was estimated before and 4 weeks after therapy.

The iron was administered either as a bolus push over 5 minutes or as an infusion over 30 minutes after dilution with 100 ml isotonic saline solution. Test dose was not given, and the patient was monitored for any adverse reactions over an hour following the first injection. The iron sucrose

was not calculated and given. They studied the response to a uniform dose of IV iron sucrose over a range of pretreatment Hb and found an optimal and quick response in all the patients

A Cochrane review conducted in Pakistan reported that compared to intermittent iron supplementation , daily iron supplementation increases the haemoglobin level at the time of delivery among pregnant women in developing countries .

According to a recently published randomized study there was no difference in pregnancy outcomes between daily and twice weekly iron supplementation. But the daily regimen improves the hemoglobin at term when compared to twice weekly regimen.

The timing and dosage of oral iron are also controversial as most studies have focused on preventive treatment from midpregnancy, at or before 20 weeks gestation.

Some researchers believe that both daily and weekly iron supplementation does not reduce the prevalence of anemia in pregnancy.

We have to pay attention before the women become pregnant in the adolescent girls and women of reproductive age and provide them with sufficient and necessary micronutrients and low dose iron supplements.

Experiences on parenteral iron use are from the developed world. The study on prophylactic use of parenteral iron in pregnancy is scanty , could be because of concerns about its adverse effects.

The WHO technical working group has documented the efficacy in the prevention and treatment of severe anemia . Parenteral iron therapy when compared to oral therapy produces

- rapid and complete correction of iron deficiency
- Rapid replacement of iron stores
- Rapid erythropoietic response .

The indications for parenteral iron therapy are

- Those who are unable to tolerate oral iron
- Those in whom oral iron therapy fails due to noncompliance.
- In Pregnancy For rapid restoration of hemoglobin in patients with severe anemia coming near term.

Based on ferritin levels in early pregnancy we can go for selective administration in patients with low iron stores.

WHO recommends iron supplementation in accordance to prevalence. “In areas with prevalence of less than 40% , we have to supplement with 60 mg of elemental iron daily for 6 months in pregnany. In areas with prevalence of more than 40% continue supplementation for 3 months postpartum”.

CDC recommends 30 mg of elemental iron supplementation.

Food and Agriculture Organization of the United Nations concludes: “Iron requirements in the second and third trimesters cannot be satisfied by dietary iron alone, even if it is of high bioavailability and, unless stores of about 500 mg are believed to exist before pregnancy, administration of iron supplements may be indicated if impairment of the expected increase in hemoglobin mass in the mother is to be avoided” (58).

Department of Health in United Kingdom states: “Ideally all women of childbearing age should have sufficient stores to cope with the metabolic demands made by pregnancy which can be met without further increase because of cessation of menstrual losses and by mobilisation of maternal iron stores and increased intestinal absorption. However, when iron stores are inappropriately low at the start of pregnancy, supplementation with iron may be necessary” (59).

The European Union states: “The physiologic solution for covering the high iron requirements in pregnancy is to use iron from stores. The problem, however, is that very few women, if any, have iron stores of this magnitude. Therefore, daily iron supplements are recommended in the latter half of pregnancy” (60).

The Nordic Nutrition Recommendations states: “An adequate iron balance during pregnancy implies iron reserves of at least 500 mg. The physiologic iron requirements in the second half of gestation cannot be fulfilled solely through dietary iron” (61)

OVERVIEW

Anemia is said to be present when the hemoglobin (Hb) falls below a defined level or range. Due to physiological changes in the Hb concentration during the course of a normal pregnancy in a nonanaemic Women ,there is no universally accepted definition of anemia in pregnancy.

WHO (1972): WHO defines "Anemia as the presence of a Hb level of less than 11 g/dL during pregnancy and less than 10 g/dL in the puerperium"(14)

US CDC (1989): CDC defines "Anemia as the presence of a hemoglobin level of less than 11 g/dL during the first and third trimesters (weeks 1–12, and 29–40 of pregnancy) and less than 10.5 g/dL during the second trimester (weeks 13–28 of pregnancy)"(15)

ICMR (1989): According to ICMR "Anemia is defined by an Hb of less than 11.0 g/dL" and the severity of the anemia categorized as follows

<4 g/dL = very severe

4–6.9 g/dL = severe

7–9.9 g/dL = moderate

10–10.9 g/dL = mild" **(16)**

Types of Anemia Based on Cause:

Anemia is most commonly caused by

1) Iron deficiency
2) vitamins B12 and folic acid deficiency.
3) Chronic renal disease, chronic infestations and chronic inflammatory conditions may also be associated with anemia.

Iron Deficiency and Iron-Deficiency Anemia:

There is a state of iron deficiency that precedes iron-deficiency anemia. Various iron deficiency states are as follows:

Absolute iron deficiency:

Serum ferritin will be less than 15 μg/L, regardless of whether anemia is present.

Iron deficiency anemia:

Serum ferritin will be less than 15 μg/L and Hb level fulfils the criteria of anemia.

Latent iron deficiency:

Serum ferritin will be less than 15 μg/L, but Hb levels are within the non-anemic range.

Functional iron deficiency:

Serum ferritin is normal or even elevated, but transferrin saturation is decreased or the hypochromic erythrocyte fraction is greater than 10%.

STAGES OF IRON DEFICIENCY ANAEMIA:

Stage I: Negative iron balance

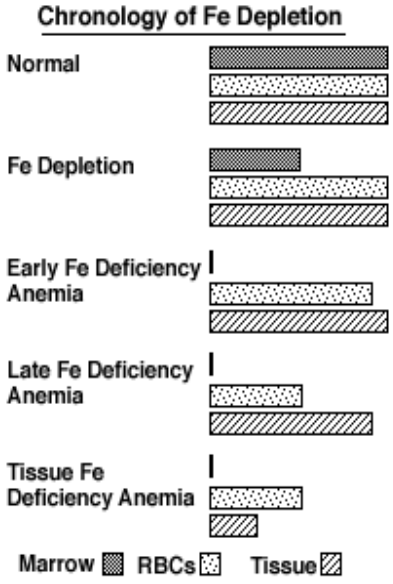
- Demands for iron exceed the iron absorbed from diet.
- Depletion of storage iron.
- Transport and functional iron is normal.
- Normal Hb / hematocrit level with Normal RBC indices.
- Serum Ferritin < 20ng/ml.

Stage II: Iron deficient Erythropoiesis

- Stored Iron become depleted.
- Transport iron is reduced .
- Serum iron begins to fall and total iron binding capacity rises gradually.
- When the transferrin falls to 15 to 20%, Hb synthesis becomes impaired.
- Increased erythrocyte protoporphyrin

Stage III: Iron deficiency anaemia:

- Peripheral smear reveals microcytic and hypochromic picture appearing as vacuolated red blood cells with reticulocytes in circulation.
- Gradually Hb and hematocrit begins to fall.
- Transferrin saturation < 15%



HAEMATOLOGICAL CHANGES IN STAGES OF IRON DEFICIENCY

	NORMAL	NEGATIVE IRON BALANCE	IRON DEFICIENT ERYTHROPOIESIS	IRON DEFICIENCY ANEMIA
Marrow iron stores	1-3+	0-1+	0	0
S. Ferritin(microgram/dl)	**50-200**	**<20**	**<15**	**<15**
TIBC(microgram/dl)	**300-360**	**>360**	**>380**	**>400**
SI mg/dl	**50-150**	**NL**	**<50**	**<30**
Saturation %	**30-50**	**NL**	**<20**	**<10**
Marrow sideroblasts%	**40-60**	**NL**	**<10**	**<10**
RBC protoporphyrin (microgram/dl)	**30-50**	**NL**	**>100**	**>200**
RBC morphology	**NL**	**NL**	**NL**	**Microcytic Hypochromic**

IRON METABOLISM IN PREGNANCY

Total body iron content of a pregnant woman is around 4 gms.

Out of this

Haemoglobin	–	70%,
Hemosiderin and ferritin	-	25%
Myoglobin	-	4.9%

Majority of iron within the body exist in the form of haem Proteins(25). Like Hemoglobin , Myoglobin is oxygen carrying component in skeletal muscle. Deficiency of iron also affects myoglobin which is one of the causes for atonic PPH in anemic pregnant women.

Enzymes involved in bactericidal action require iron containing enzymes. For optimum bactericidal action Myeloperoxidase in neutrophils requires iron. So, pregnant women who are anemic are thus prone for concurrent infections.

Iron is an important component of several respiratory proteins and enzymes and it serves as a carrier of oxygen.

One of the important enzymes involved in DNA synthesis is Ribonucleotide reductase , which requires iron for its optimum action. This is the reason for fetal growth restriction in pregnant women with iron deficiency anaemia.

In summary, the important bio-chemical functions of iron are

1. Cell division and differentiation,
2. Oxygen transport,
3. Electron transport and many other functions.
4. Catalyst for oxygenation and hydroxylation
5. Erythropoeisis

DAILY REQUIREMENT:

Adult male - 0.5- 1 mg

Adult female - 1-2 mg

Infants - 60 microgram/Kg

Children - 25microgram/Kg

Pregnancy - 3 to 5 mg

DIETARY SOURCES OF IRON:

RICH SOURCES:

Liver, egg yolk, oyster, dry beans, dry fruits, wheat germ, yeast

MEDIUM SOURCES:

Meat, chicken, fish, banana , spinach , apple

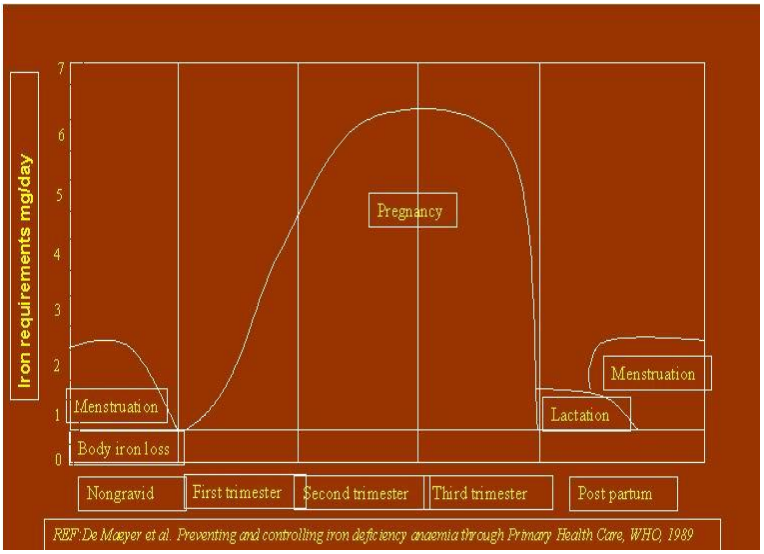
POOR SOURCES:

Milk and its products, root vegetables

IRON REQUIREMENTS DURING PREGNANCY:

The Iron requirement in pregnancy varies markedly in each trimester. Iron requirement decreases during first trimester due to stoppage of menstruation and account for saving of 0.56mg of iron/day. Iron requirements begin to increase in second trimester and it is on increasing trend throughout remainder of pregnancy.

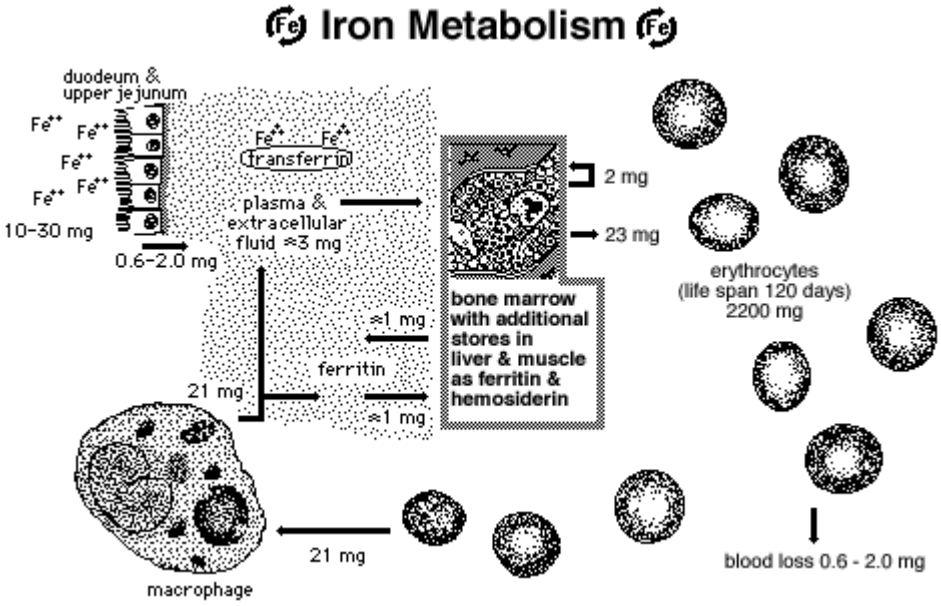
As the pregnancy progresses, the requirements of iron for the fetus steadily increases in proportion to the weight of the fetus. The iron content of 3 Kg fetus is approximately <270 mg



An iron loss that occurs during parturition must also be added which includes 150 mg iron at blood loss and 90 mg in placenta and umbilical cord.

According to Council on Food and Nutrition, the requirements have been quantified as follows:

External iron losses	- 170 mg
Expansion of red cell mass	-450 mg
Fetal iron	- 270 mg
Placenta and cord	- 90 mg
TOTAL	**- 980 mg**



IRON BALANCE IN PREGNANCY:

Iron requirements rise to 4 mg in the second trimester and 6 mg in third trimester. Even from most optimal diet, iron requirements cannot be met with from dietary absorption.

Iron absorption is usually 3-4 mg/day, if the diet contains large quantities of bioavailable iron . But in developing country like us where the staple food is cereal amount of iron absorbed is lower. **(26)**

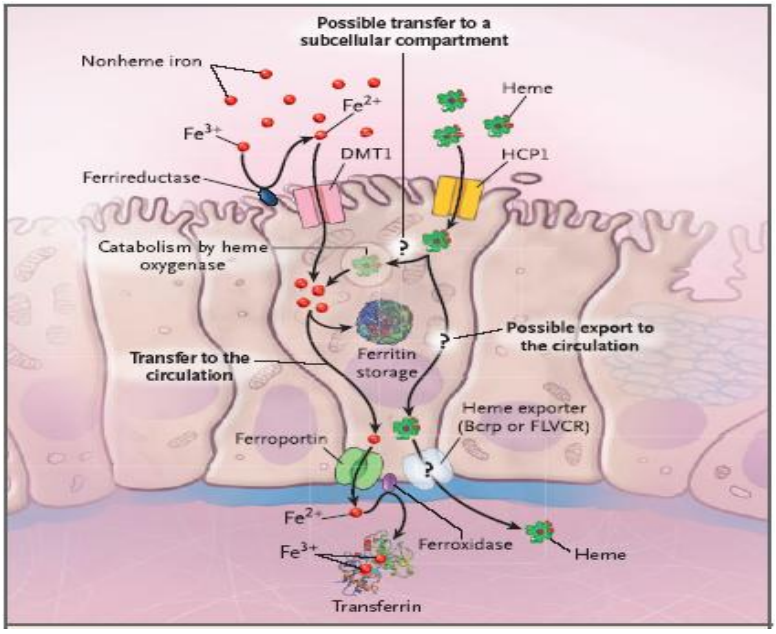
FACTORS FACILITATING IRON ABSORPTION:

The heme form of iron has better bioavailability and is absorbed in ferrous form.

- Ascorbic acid
- Fermented food items
- Gastric acidity
- Low iron stores
- Increased erythropoietic activity

FACTORS INHIBITING IRON ABSORPTION:

- Alkalis like Antacids
- Phosphates
- Phytates
- Tannin
- Tetracylines



TRANSPORT AND UTILIZATION:

Free iron is highly toxic. Free iron on entering the plasma is immediately converted to ferric iron and is complexed with transferrin. The total plasma iron content is about 3 mg and is recycled about 10 times everyday. so the turnover of iron per day is around 30 mg/day.

Iron is transported into erythropoietic and other cells through specific membrane bound transferrin receptors. The complex is engulfed by the process of endocytosis. In the acidic PH of intracellular vesicles, Iron dissociates from the complex. The iron thus released is utilized for hemoglobin synthesis, while the transferrin and transferrin receptors return to the cell surface to carry fresh iron loads.

STORAGE AND EXCRETION:

Iron is stored in reticuloendothelial cells in liver, spleen ,bone marrow, and also in hepatocytes and myocytes as ferritin and haemosiderin. Plasma iron derived from destruction of old RBCs , from stores and from intestinal absorption forms a pool that is available for erythropoiesis.

Iron is excreted mainly as exfoliated gastrointestinal mucosal cells, some RBCs, and in bile. Other routes are desqumated skin, very little in urine and sweat.

The upper limit for iron tolerance in pregnancy is 45mg/day of iron. The adverse effects are less likely at this level.(IOM 2001)

CLINICAL FEATURES:

1. Mild anaemia may not have any effect on pregnancy.
2. Moderate anaemia may cause increased weakness, fatigue, lack of energy, and poor work performance.
3. Severely anemic woman may have palpitations,tachycardia, breathlessness, increased cardiac output leading on to cardiac stress which can cause decompensation and cardiac failure which may be fatal.

SIGNS:

There may be no signs in mild anaemia. There may be pallor, glossitis and stomatitis. Patient may have edema due to hypoprotinemia. Soft systolic murmur can be heard in the mitral area due to hyperdynamic circulation. There can be fine crepitation at the base of lungs due to congestion.

DIAGNOSIS:

Hemoglobin:

Haemoglobin estimation is the most practical method of diagnosis of anemia. Though various methods like Taliquists method, copper sulphate method and sahli's method are available. Cyanmethhaemoglobin method appears to be the most accurate.

Peripheral smear:

Peripheral blood smear is another bedside indicator of diagnosis of anemia which will also differentiate between iron deficiency anemia, megaloblastic anemia and hemolytic anemia.

RBC Indices:

Mean corpuscular volume, Mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration are all low in the iron deficiency anemia.

Serum Ferritin:

- Serum Ferritin estimation refects iron stores
- Ferritin is a high molecular weight glycoprotein
- Normal level of S. Ferritin is 15-300 microgram/dl
- Level <12 microgram/dl indicates iron deficiency
- It is stable, unaffected by recent iron intake.
- It is the first abnormal laboratory test in iron deficiency.

Transferrin saturation:

- It is estimated from serum iron and total iron binding capacity
- Second measurement to be affected in iron deficiency anemia
- Serum iron varies from 60-120 microgram/dl and TIBC is 300-350 microgram /dl
- Transferrin saturation <15% indicates iron deficiency.

Free Erythrocyte Protoporphyrin:

- It rises with defective iron supply
- It takes 2-3 weeks to become abnormal after depletion of iron stores

Serum Transferrin Receptor:

- Specific and sensitive marker of iron deficiency
- Its levels are increased in iron deficiency anemia
- Its facilities are not routinely available

Bone Marrow Examination:

- Invasive test
- By staining with potassium ferrocyanate, the characteristic blue granules of stainable iron in erythroblasts can be seen.
- It is indicated in

1. Cases where there is no response to iron therapy after 4 weeks
2. Diagnosis of kala- azar
3. Aplastic anemia

Stool for Ova and cyst:

Should be done consecutively for 3 days

Peripheral smear:

For malarial parasite

Renal function Test:

For suspected renal disease

Serum Proteins- Hypoproteinemia

Urine Examination

In Non- pregnant state , iron deficiency can be measured using serum ferritin, Transferrin, Serum iron , Transferrin Saturation and Transferrin receptors.These tests have their own limitations and are not readily available. Interpretation is difficult in situations like Malaria, HIV/AIDS,

Vaginosis and during Pregnancy. Even in women ingesting high amount of iron , it is found that Serum ferritin and Bone marrow iron fall during pregnancy. This raised the doubt about their significance in pregnancy. (Puolakka1980; Romslo1983; Svan berg 1975).

WHO and CDC Technical Consultation on the Assessment of Iron status at population level concluded that serum ferritin and hemoglobin were the efficient combination of indicator for monitoring iron status.

CONSEQUENCE OF MATERNAL IRON DEFICIENCY IN CHILDREN:

" . Impaired mental and psychomotor function

- Impaired sleep patterns and affective relationship
- Limited attention span
- Capacity of immune system decreased
- Increased morbidity from infectious diseases
- More severe and longer lasting diarrhoea
- Signs and symptoms of cardiac failure" (*Verster A. WHO Guidlelines 1996)*

EFFECT OF ANAEMIA ON PREGNANCY

1) Antenatal period:

a. Early pregnancy loss (abortion).

b. Increased incidence of urinary tract infection.

c. Increased incidence of pregnancy induced hypertension

d. Abruptio placenta

e. Congestive cardiac failure

f. Fetal growth restriction

2) Labour

a. Labour dysfunction like uterine inertia, precipitate labour

b. Prolonged II stage

3) After delivery (immediate)

a. Postpartum haemorrhage

b. Congestive Cardiac failure

4) After delivery (Late)

a. Lactation failure

b. Puerperal sepsis

c. Deep vein thrombosis

d. Sub involution of uterus

e. Wound infection-for example episiotomy wound & Caeserean section wound

5) *Remote*

a. Genital prolapse

b. General debility

c. Menstrual abnormalities (41).

Anaemia in pregnancy causes certain problems like abruption, pregnancy induced hypertension, urinary tract infection, and thrombophlebitis. These are major causes of maternal and perinatal morbidity and mortality.

By correction of anaemia of pregnancy we can drastically reduce these problems. Post-partum haemorrhage is a leading cause of maternal death in our country because of increased prevalence of anaemia. The cause for post-partum haemorrhage in anaemic pregnant women is mainly due to decreased myoglobin in uterine musculature consequently leading to decreased oxygen perfusion to the uterus. So by correcting anemia, we can indirectly prevent postpartum hemorrhage and its attendant morbidity and mortality.

Prophylaxis:

Before a women marries and get pregnant she should have adequate iron stores. This can be achieved by

1) Routine screening for anemia for all adolescent girls

2) Eating iron rich foods

3) Food fortification with iron

4) Providing iron supplementation from school days

5) Annual screening for women with high risk factors.

ORAL IRON:

The preferred route of iron administration is oral. Ferrous salts are Inexpensive, have high iron content and better absorbed than ferric salts. The most common side effects of oral iron are gastric irritation and Constipation which are related to total quantity of elemental iron administered. The elemental iron content and not the quantity of iron Compound per dose unit is to be taken into consideration.

Sustained release preparations are more expensive and not rational because these preparations release part of the iron content lower down in the intestine where absorption does not take place. Liquid preparations may stain the teeth and they are less satisfactory. Maximal haemopoeitic response

is achieved when 200 mg elemental iron given daily in three divided doses. Absorption of oral iron is better when it is taken in empty stomach , but side effects are more. To overcome the side effects, some prefer to give larger amounts after meals, while others prefer to give smaller doses in between meals.

ADVERSE EFFECTS OF ORAL IRON:

The adverse effects are due to elemental iron content.

- Epigastric pain
- Heart burn
- Nausea
- Vomiting
- Staining of teeth
- Metallic taste
- Abdominal colic
- constipation due to astringent action of iron
- Diarrhoea due to reflect irritant action

AMOUNT OF IRON IN COMMON IRON PREPARATIONS:

PREPARATION	Molecular iron mg/tablet	Percentage of iron(%)	Elemental iron (mg/tablet)
Ferrous sulphate	300	20	60
Ferrous sulphate anhydrous	200	37	74
Ferrous sulphate, dessicated	200	30	60
Ferrous Fumerate	200	33	66
Ferrous Gluconate	300	12	36

PARENTERAL IRON:

INDICATIONS:

- When oral iron is not tolerated and when the bowel upset is more
- Failure to absorb oral iron in conditions like malabsorption and inflammatory bowel disease
- Non- compliance to oral iron.
- In presence of severe deficiency with chronic bleeding
- Parenteral iron is given along with erythropoietin to meet the demands of rapid erythropoiesis.

Parenteral iron are of two types

i)Intramuscular

ii)Intravenous

Intramuscular therapy is rarely used nowdays due to pain and discolouration. These are iron sorbitol citric acid complex and Iron dextran.

There are three types of I V iron preparations

TYPE I COMPLEXES:

- Iron Dextran and Iron Dextrin are Type I complexes.
- They are high molecular compounds.
- It has got very high stability.
- Type I Hypersentivity reaction is common.
- Total dose infusion may be given.

TYPE II COMPLEXES:

- These are iron hydroxide sucrose complex.
- It has got low molecular weight.
- Biological polymers are not formed in standard doses.
- Anaphylactic reactions are rare.
- Can be given up to 200 mg on alternate days.
- Ferric carboxymaltose can be given up to 1000mg over 15 minutes (not available in india)

TYPE III COMPLEXES:

- Iron gluconate, Iron Hydroxide sorbitol complex and Iron Ammonium citrate.
- Low molecular weight.
- Better side effect profile.
- Tissue toxicity is more due to more of free iron.

IRON SUCROSE

Iron sucrose is polynuclear iron (III)-hydroxide complex. It is a sterile brown coloured fluid for intravenous use.

- "Molecular weight about 34,000 - 60,000 daltons"
- "The osmolarity is around 1150 mOsmol/L to 1350 mOsmol/L".
- "Structural formula: $[Na_2Fe_5O_8(OH) \cdot 3(H_2O)]n \cdot m(C_{12}H_{22}O_{11})$"

COMPOSITION :

Each 5 ml contains 100 mg of iron sucrose

CLINICAL PHARMACOLOGY :

MECHANISM OF ACTION:

Following intravenous administration, iron sucrose dissociates into iron and sucrose by reticuloendothelial system.

Iron is transferred from the blood into iron pool in the liver and bone marrow.

Iron in a nonionic form is sequestered by ferritin from which iron is easily available.

Pharmacokinetics:

- Iron sucrose exhibits first order kinetics
- Half-life is 6 hours .
- Total clearance - 1.2 L/h.
- Steady state volume of distribution - 7.9 L
- Non steady state volume of distribution -10 L.
- Clearance of iron from the serum is rapid in patients with iron deficiency when treated with iron sucrose.

Metabolism and Elimination:

Iron sucrose dissociates into iron and sucrose by the reticulo endothelial system. The sucrose component is excreted mainly through urine.

CONTRAINDICATIONS:

• Anemia not due to iron deficiency.

• Hypersensitivity to drug.

• When there is evidence of iron overload.

SIDE EFFECTS:

Headache, fever, chills, rigor, pain, asthenia, malaise, abdominal pain, flushing metallic taste, nausea, vomiting .

Delayed reactions like joint pain , myalgia, skin rashes, lymphadenopathy and thrombophlebitis at the site of injection may occur.

INTERACTION:

Should not be administered with oral iron preparation because the absorption of oral iron may be reduced.

METHOD AND ROUTE OF ADMINISTRATION

No test dose is required

1. Slow IV injection

100mg (1 amp) to be given undiluted over a period of 2 – 5 min.

2. Slow IV infusion

100mg (1 amp) to be diluted with 100ml of normal saline immediately prior to infusion and to be infused over a period of atleast 15min .

DOSAGE FREQUENCY

100mg/day given on alternate days until the required dose is infused. Chandler et al observed that Doses of 200 – 300mg of iron sucrose when infused intravenously over 2 hours were well tolerated and safe. Those

Patients who received higher dose of 400 – 500mg intravenously over 2 hours experienced side effects like hypotension, nausea, and low back pain (56).

Storage/Stability

- Vials can be stored at room temperature (59° to 86°F).
- Protect from freezing.
- We have to use it immediately after dilution in saline.

Among all IV preparations iron sucrose is highly efficacious due to following reasons

- It is a nonionic iron complex Which leads on to low toxicity
- Anaphylactic reactions are rare because biological polymers are not formed
- Iron sucrose is immediately available for erythropoiesis
- Renal excretion is very low
- Tissue accumulation is low and so toxicity is less
- No oversaturation, so adverse reactions are minimal

Iron sucrose was used as hematinic therapy for past 50 years. It can be used for

i) Anemia of chronic kidney disease

ii) Anemia associated with pregnancy

iii) In the post surgical period.

In November 2000 , FDA approved the clinical use of iron sucrose in anemia of chronic hemodialysis patients. Approval for iron sucrose in pregnancy and puerperal period was given by FDA in 2005 Oral iron when given in appropriate doses correct iron deficiency anemia , but it requires high degree of motivation for the people to take it correctly to maintain the compliance. Poor compliance is one of the major disadvantage of oral iron. Intravenous iron sucrose overcome the problem of compliance and the side effects are also minimal. The main advantage is certainity of administration and the patient acceptability is also high. The drawback is its cost. But it is now made available in all Government institutions free of cost.

So in order to find an alternative for oral iron in the prophylaxis of iron deficiency anemia which has got many drawbacks, iron sucrose was used as prophylaxis in our study.

MATERIALS AND METHODS

This study was conducted in the department of obstetrics and gynecology at institute of social obstetrics, kasturba Gandhi hospital triplicane, Chennai - 5 from the period of December 2011-december 2012.

All Antenatal women attending antenatal OP who fulfill the inclusion / exclusion criteria were randomly selected and included in this study.

INCLUSION CRITERIA:

1. Women booked and immunized in KGH
2. singleton pregnancy
3. 20 to 36 weeks of gestation
4. hemoglobin >10g%
5. women age 20 to 35yrs

EXCLUSION CRITERIA:

1. History of allergic condition or bronchial asthma
2. History of allergy to iron
3. Multiple pregnancy
4. Cirrhosis, viral hepatitis.
5. History of hematological disease.

6. History of bleeding tendency.

During the Antenatal visit, for all the Women attending the outpatient department, the following investigations were done.

1. Hemoglobin
2. Packed cell volume
3. Mean corpuscular volume

Those who fulfill inclusion criteria were enrolled in the study after getting informed consent. A detailed history and complete clinical examination was done.

METHODS:

Antenatal mothers who were selected were allocated in to one of three groups

1. Group A (100) women were given only two doses of 200mg of iron sucrose between 20-24weeks & 28-32weeks and oral iron was not given to this group of patients.

2. Group B (100) women were given three doses of 200mg of iron sucrose at 20-24 weeks & 28-32 weeks &34-36 weeks and oral iron was not given to this group of patients.

3. Group C (100) women were given 100 tablets of oral ferrous sulphate100mg once daily for 3 months between 20 to 36 weeks.

This treatment was supplemented with folic acid , to all the three groups. All women were given Tab Albendazole 400 mg in second trimester before starting iron therapy.

Before starting therapy blood was taken for all women and the assessment of Hb , PCV ,MCV was done.

200mg of iron sucrose diluted in 100ml of normal saline(0.9%) was given intravenously over a period of 30 minutes as out patient procedure and followed up for two hours for any adverse drug events. Emergency drugs like steroids, antihistamines were kept ready. Adverse drug events if any were noted.

The parameters that are monitored during intravenous iron therapy are

1. Vitals – temperature, blood pressure, pulse rate
2. Adverse effects like nausea, abdominal pain,vomiting chills, etc
3. Anaphylactic reactions

The Response to treatment was monitored by doing the following hematological indices four weeks, eight weeks and at term .

1. Hemoglobin
2. Mean corpuscular volume
3. Packed cell volume

The results were statistically analysed using SPSS software.

RESULTS AND ANALYSIS:

CHARACTERISTICS OF CASES STUDIED:

***Table 1*: AGE DISTRIBUTION**:

	20-24 YEARS	**25-29 YEARS**	**30-35 YEARS**	**TOTAL**
A	54	34	12	100
B	55	37	8	100
C	48	39	13	100
TOTAL				300

AGE GROUP	**TOTAL**	**PERCENTAGE**
20-24 YEARS	157	52.3%
25-29 YEARS	110	36.7%
30-35 YEARS	33	11%
	300	100%

Among 300 Women studied, 157 Women (52.3%) belong to age group Between 20-24 years, 110 (36.7%) Women belong to age group between 25-29 years and only 13(11%) women belong to age group between 30-35 years.

Table 2: **SOCIO ECONOMIC STATUS:**

GROUPS	1	2	3	4	5	TOTAL
A	-	-	8	78	14	100
B	-	-	10	80	10	100
C	-	-	4	81	15	100
PERCENTAGE			7.3%	79%	13%	

Among three hundred Women in our study group, Majority of Women belong to Class IV Socioeconomic status which constitutes 239 (79%) women, 39 (13%) Women belong to Class V Socioeconomic status and only 22(7.3%) Women belong to Class III Socioeconomic status . None of them belong to Class I and Class II Socioeconomic status.

***Table*: 3: PARITY**

GROUPS	PRIMI	MULTI	TOTAL
A	63	37	100
B	65	35	100
C	57	43	100
TOTAL	185	115	
PERCENTAGE	61.7%	38.3%	

Among 300 Women in our study, 185 (61.7%) Women were primipara which constitutes major portion and 115(38.3%) Women were multipara.

Table :4: HEMOGLOBIN VALUES IN GROUP A:

HB	MEAN	SD
BEFORE	10.644	0.3942
4WEEKS	11.091	0.2896
8WEEKS	11.314	0.2425
AT TERM	11.613	0.1715

CHANGE IN HEMOGLOBIN :

HB	MEAN	STANDARD DEVIATION
BEFORE	10.644	0.3942
AT TERM	11.613	0.1715
CHANGE IN HB	0.969	P <0.005

Among 100 Women in group **A** , the Mean pretreatment Hemoglobin was 10.644 and the standard deviation was 0.3942 and at term the mean Hemoglobin was 11.613 with Standard deviation of 0.1715 after giving two doses of iron sucrose. There is a Mean rise in Hemoglobin of about 0.969. There is statistically significant rise in Hb with P Value <0.005

Table:5 : HEMOGLOBIN VALUES IN GROUP B

HB	MEAN	SD
BEFORE	10.756	0.4222
4WEEKS	11.208	0.3871
8WEEKS	11.444	0.3767
AT TERM	12.357	0.2717

CHANGE IN HEMOGLOBIN :

HB	MEAN	STANDARD DEVIATION
BEFORE	10.756	0.4222
AT TERM	12.357	0.2717
CHANGE IN HB	1.599	P<0.002

Among 100 Women studied in group **B** , the mean pretreatment Hemoglobin was 10.756, standard deviationwas0.4222 and after giving three doses of iron sucrose , the Hemoglobin at term was found to be 12.357 with standard deviation of 0.2717. The Mean rise in hemoglobin was 1.599.There is a statistically significant rise in Hb from the pretreatment value with P<0.002

Table: 6: HEMOGLOBIN VALUES IN GROUP C:

HB	MEAN	SD
BEFORE	10.705	0.3812
4WEEKS	11.062	0.3212
8WEEKS	11.274	0.3111
AT TERM	11.433	0.2756

CHANGE IN HEMOGLOBIN:

HB	MEAN	STANDARD DEVIATION
BEFORE	10.705	0.3812
AT TERM	11.433	0.2756
CHANGE IN HB	0.729	**P<0.001**

Among 100 Women in Group C who were taking oral iron tablets, the Mean pretreatment Hb was 10.705 with standard deviation of 0.3812 and at term the mean Hb was 11.433 with standard deviation of 0.2756. The mean rise in H b was about is 0.729. From the pretreatment value there is a significant rise in Hb with P<0.001.

Table: 7: PCV VALUES IN GROUP A

PCV	MEAN	SD
BEFORE	34.62	1.0324
4WEEKS	35.54	1.005
8WEEKS	35.28	0.9680
AT TERM	35.70	0.6540

CHANGE IN PCV

PCV	MEAN	STANDARD DEVIATION
BEFORE	34.62	1.0324
AT TERM	35.70	0.6540
CHANGE IN PCV	1.08	**P<0.047**

The Mean PCV before starting treatment and at term in Group A was 34.62 and 35.70 respectively. The Mean change in PCV is 1.08 which is statistically significant with P<0.047.

Table:8: PCV VALUES IN GROUP B

PCV	MEAN	SD
BEFORE	34.82	0.9974
4WEEKS	35.24	0.0064
8WEEKS	35.76	0.7328
AT TERM	36.20	0.3426

CHANGE IN PCV

PCV	MEAN	STANDARD DEVIATION
BEFORE	34.82	0.9974
AT TERM	36.20	0.3426
CHANGE IN PCV	1.28	**P<0.004**

The Mean PCV before starting treatment and at term in Group B was 34.82 and 36.20 respectively. The Mean change in PCV was 1.28 which is statistically significant with P<0.004.

Table :9: PCV VALUES IN GROUP C

PCV	MEAN	SD
BEFORE	34.76	1.0284
4WEEKS	34.24	0.1006
8WEEKS	35.54	0.7818
AT TERM	35.72	0.4326

CHANGE IN PCV

PCV	MEAN	STANDARD DEVIATION
BEFORE	34.76	1.0284
AT TERM	35.72	0.4326
CHANGE IN PCV	0.96	P=0.000

The Mean PCV before starting treatment and at term in Group C was 34.76 and 35.72 respectively. The Mean change in PCV is 0.96 which is statistically significant with P=0.000.

Table: 10: MCV VALUES IN GROUP A

MCV	MEAN	SD
BEFORE	85.29	1.9808
4WEEKS	85.73	1.6354
8WEEKS	86.05	1.4716
AT TERM	86.44	0.9628

CHANGE IN MCV:

MCV	MEAN	STANDARD DEVIATION
BEFORE	85.29	1.9808
AT TERM	86.44	0.9628
CHANGE IN MCV	1.15	P=0.03

The Mean MCV before starting treatment and at term in Group A was 85.29 and 86.44 respectively. The Mean change in MCV was 1.15 which is statistically significant with P=0.03.

Table: 11: MCV VALUES IN GROUP B

MCV	MEAN	SD
BEFORE	85.09	1.554
4WEEKS	85.40	1.426
8WEEKS	85.51	1.2815
AT TERM	86.91	0.8162

CHANGE IN MCV

MCV	MEAN	STANDARD DEVIATION
BEFORE	85.09	1.554
AT TERM	86.91	0.8162
CHANGE IN MCV	1.82	P=0.009

The Mean MCV before starting treatment and at term in Group B was 85.09 and 86.91 respectively. The Mean change in MCV was 1.82 which is statistically significant with P<0.009.

Table: 12: MCV VALUES IN GROUP C

MCV	MEAN	SD
BEFORE	85.44	1.5543
4WEEKS	85.40	1.4264
8WEEKS	85.51	1.2815
AT TERM	86.91	0.8162

CHANGE IN MCV

MCV	MEAN	STANDARD DEVIATION
BEFORE	85.44	1.5543
AT TERM	86.91	0.8162
CHANGE IN MCV	1.47	P=0.07

The Mean MCV before starting treatment and at term in Group C was 85.44 and 86.91 respectively. The Mean change in MCV is 1.47 with P<0.07.

***Table* :13: CHANGES IN PCV IN ALL GROUPS:**

	GROUP A		GROUP B		GROUP C	
	MEAN	**SD**	**MEAN**	**SD**	**MEAN**	**SD**
BEFORE	34.62	1.0324	34.82	0.9974	34.76	1.0284
4 WEEKS	35.54	1.0005	35.24	1.0064	34.24	0.1006
8 WEEKS	35.28	0.9680	35.76	0.7328	35.54	0.7818
AT TERM	35.70	0.6540	36.20	0.3426	35.72	0.4326
	P<0.047		P<0.004		P<0.000	

The mean change in PCV in Group A from the pretreatment value to PCV at term is 1.08, the mean change in PCV in Group B is 1.38 and the mean change in PCV in Group C is 0.96. The mean PCV within groups before starting therapy and at term is statistically significant P<0.047,P<0.004, P<0.000 respectively. when compared between groups,there is no statistically significant rise in PCV.

***Table* :14: CHANGES IN MCV:**

	GROUP A		**GROUP B**		**GROUP C**	
	MEAN	**SD**	**MEAN**	**SD**	**MEAN**	**SD**
BEFORE	85.29	1.9808	85.09	1.5543	85.33	1.4461
4 WEEKS	85.73	1.6354	85.40	1.4264	85.40	1.4264
8 WEEKS	86.05	1.4716	85.51	1.2815	85.40	1.4128
AT TERM	86.448	0.9628	86.91	0.8162	86.10	0.7221
		P=0.03		P=0.009		P=0.07

The Mean rise in MCV from pretreatment value to MCV at term is 1.15 in Group A, 1.82 in Group B and 0.76 in Group C.There is a statistically significant rise in MCV with intravenous groups (p=0.03, p=0.009 for Group A and Group B respectively) when compared between pretreatment and at term than oral group (p=0.07) .when analysis was done between groups there is no statistically significant rise in MCV.

***Table*: 15: CHANGES IN HB:**

	GROUP A		**GROUP B**		**GROUP C**	
	MEAN	**SD**	**MEAN**	**SD**	**MEAN**	**SD**
BEFORE	10.644	0.3942	10.756	0.4222	10.705	0.3812
4 WEEKS	11.091	0.2896	11.208	0.3871	11.062	0.3212
8 WEEKS	11.314	0.2425	11.444	0.3767	11.274	0.3111
AT TERM	11.613	0.1715	12.353	0.2717	11.433	0.2756
		P=0.005		P=0.0002		P=0.001

The mean pretreatment HB in all the three Groups is same. The mean degree of raise in HB in Group A is 0.969 , the mean HB rise in Group B is 1.599 and the mean HB rise in Group c is 0.729. ONE WAY ANOVA test was performed with in groups, which showed a statistically significant rise in HB from the pretreatment value to HB at term in all groups. Then student T test was performed to know the statistical significance between groups. There is statistically significant (P< 0.000) rise in HB in Group B , who were given three doses of intravenous iron sucrose when compared to Group C who were given oral iron supplementation and Group A who were given two doses of iron sucrose.

Table: 16: ADVERSE DRUG EVENTS:

ADE	INTRAVENOUS IRON SUCROSE	ORAL IRON
Local pain	2(1%)	-
Burning sensation at infusion	-	-
Giddiness	2(1%)	-
Rashes	1(0.5%)	-
Gastritis	-	15(15%)
Constipation	-	8(8%)
Diarrhoea	-	2(2%)
Chest discomfort	1(0.5%)	-
Palpitation	1(0.5%)	-
Anaphylaxis	-	-

Out of 200 Women who received intravenous iron sucrose, 3.5% of them had minor adverse drug events like local pain, giddiness, rashes , palpitation and chest discomfort which was treated symptomatically. There were no major Anaphylactic reactions noted during this study. On the contrary 25% of Women had minor gastrointestinal side effects in the oral group which is the common cause of non- compliance.

DISCUSSION

In our study, antenatal women attending the Antenatal Outpatient Department who fulfill the inclusion, exclusion criteria stated in the methodology were selected and assigned to one of the three groups. Women belonging to Group A and B were given two and three doses of iron sucrose respectively, each dose containing 200 mg of iron sucrose. Group C women were given 100 tablets of ferrous sulphate. Hemoglobin, Packed cell volume, Mean Corpuscular Volume were assessed 4 weeks,8 weeks and at term. Any adverse reaction during the therapy noted. The results were analysed.

Among 300 Women studied, 157 Women (52.3%) belong to age group Between 20-24 years, 110 (36.7%) Women belong to age group between 25-29 years and only 13(11%) women belong to age group between 30-35 years.

Among three hundred Women in our study group , 239 Women which constitutes 79% belong to Class IV Socioeconomic status, 39 (13%) Women belong to Class V Socioeconomic status and only 22(7.3%) Women belong to Class III Socioeconomic status. None of them belong to Class I and Class II Socioeconomic status.

Among 300 Women in our study, 185 (61.7%) Women were primipara. 115(38.3%) Women were multipara,this suggest that anemia is common in multipara due to previous pregnancy events ,short interdelivery interval.

COMPARISION OF OUTCOME PARAMETERS:

I) COMPARISION OF HEMOGLOBIN:

Among 100 Women in group **A** , the Mean pretreatment Hemoglobin was 10.644 and the standard deviation was 0.3942 and at term the mean Hemoglobin was 11.613 with Standard deviation of 0.1715 after giving two doses of iron sucrose. There is a Mean rise in Hemoglobin of about 0.969 which is statistically significant with p=0.005.

Among 100 Women studied in group **B** , the mean pretreatment Hemoglobin was 10.756 and after giving three doses of iron sucrose , the Hemoglobin at term was found to be 12.357. The Mean rise in hemoglobin was 1.599 which is statistically significant with p=0.0002

Among 100 Women in Group C who were taking oral iron tablets, the Mean pretreatment Hb was 10.705 with standard deviation of 0.381 and at term the mean Hb was 11.433 with standard deviation of 0.2756. The mean rise in H b was about is 0.729 which is statistically significant with p=0.001.

Gabriela Bencaivo et al conducted a study to assess and compare the efficacy of two and three doses of iron sucrose with daily supplementation of oral ferrous sulphate in the prophylaxis of iron deficiency anemia in pregnant women.There was a non significant trend to a higher frequency of responders in the intravenous iron group (75%vs 80%).

When the hemoglobin >11g/dl is considered as responders. Hb before delivery in the intravenous group was 12+/-0.9 and 12.4+/-1.1 in oral group with p value of 0.110. HCT and MCV in the intravenous group was 87.4+/-5.7 and 35.2+/-2.1 respectively and in the oral group it was 86.6+/-5.7, 35.6+/-3.1 with p value of 0.259, 0.222.

The efficacy of iron prophylaxis was demonstrated by an significant increase in Hb and HCT in the intravenous and oral iron group ($p<0.001$ and $P<0.01$).Serum ferritin , which is the best indicator of body iron stores showed a absolute rise in women with three doses of iron prophylaxis. Eighty percent of women with three doses of intravenous iron and 71% with two dosess of intravenous iron exhibited Hb >11g/dl after iron supplementation. Our study is comparable to this study.

ii) COMPARISION OF PCV AND MCV:

The mean change in PCV in Group A from the pretreatment value to PCV at term is 1.08, the mean change in PCV in Group B is 1.38 and the mean change in PCV in Group C is 0.96.There is a statistically significant rise in PCVwith in groups from the pretreatment to at term. Although the mean PCV between groups is not statistically significant, there is slight rise in PCV in Group B compared to other two groups.

The Mean rise in MCV from pretreatment value to MCV at term is 1.15 in Group A, 1.82 in Group B and 0.76 in Group C. There is statistically significant rise in MCV with in groups when compared to pretreatment to at term. There is no statistically significant rise in MCV in the intravenous group compared to oral group.

Our study could be compared to Gabrielo Bencaivo et al European journal of obstetrics and gynecology and reproductive biology 144 (2009) 135- 139. In this study iron prophylaxis was given as intravenous and oral and the efficacy compared.

According to this study HCT before delivery in intravenous group was 35.2+/-2.1 and 35.6+/-3.1 in the oral group. There is no significant change in HCT in both intravenous and oral group. MCV was 87.4+/-5.7 in

the intravenous group and 86.6+/-5.7 in the oral group. Our study is comparable to this study.

The mean pretreatment HB in all the three Groups is same. The mean degree of raise in HB in Group A is 0.969 , the mean HB rise in Group B is 1.599 and the mean HB rise in Group c is 0.729. ONE WAY ANNOVA test was performed with in groups, which showed a statistically significant rise in HB from the pretreatment value in each groups. Then student T test was performed to know the statistical significance between groups. There is statistically significant(P= 0.000) rise in HB in Group B , who were given three doses of intravenous iron sucrose when compared to Group C who were given oral iron supplementation and Group A who were given two doses of iron sucrose.

Out of 200 Women who received intravenous iron sucrose, 3.5% of them had minor adverse drug events like local pain, giddiness, rashes , palpitation and chest discomfort which was treated symptomatically. There were no major Anaphylactic reactions noted during this study. On the contrary 25% of Women had minor gastrointestinal side effects in the oral group which is common cause for non – compliance.

In our study we gave iron sucrose at 4 weeks interval as prophylaxis and found highly significant rise in hemoglobin concentration as also reported by Françoise B et al who gave iron at weekly interval. While Al-Memon et al reported no significant difference in the effectiveness of iron sucrose over oral iron for elevating Hb concentration during pregnancy.

Contrary to the conclusion of Reveiz et al regarding parenteral iron therapy about possible serious adverse effects, in our study compliance with iv iron therapy was good.In daily practice, physicians often face poor compliance with oral therapy because of digestive side effects which can lead to worsening of anemia. In these cases parenteral forms of administration are indicated as well as in those patients in whom oral treatment is ineffective.

Intravenous iron therapy was found safe, convenient and more effective than intramuscular iron therapy in treatment of iron deficiency anemia during pregnancy by Wali et al.The effects of parenteral iron therapy on the baby should be investigated in further studies.

Our study showed that three dose of intravenous iron sucrose therapy in the prophylaxis of iron deficiency anemia in pregnant women showed significantly raised hemoglobin levels when compared to two doses and oral iron.

Iron sucrose therapy as prophylaxis can reduce the maternal mortality and morbidity to a significant level. Iron sucrose therapy can be a first line of prophylaxis in view of its easy accessibility , safety and good efficacy compared to oral iron.

SUMMARY

In our study 300 Antenatal Women attending the Antenatal out Patient department who fulfill the inclusion criteria were randomly selected and divided into one of the three groups. Initially Hemoglobin, Packed cell volume, Mean corpuscular volume was done in all the Women. Group A and B Women were given two and three doses of intravenous iron sucrose respectively. Group C Women were given oral ferrous sulphate. The following parameters were analysed after 4 weeks, 8 weeks and at term.

1. Hemoglobin in g/dl.
2. Packed cell volume.
3. Mean corpuscular volume.

Statistical package for social science (SPSS) was used for statistical compilation and analysis. For Statistical analysis of difference between groups, analyses of covariance or t-test were applied when appropriate. Statistical significance was accepted as $P<0.05$.

The results of the study are tabulated, analysed and summarized as follows.

1. Majority of women 157 (52.3%) belong to the age group between20-24 years.
2. In this study Majority of women 239(79%) belong to Class IV Socioeconomic status.

3. Most of the Women 185 (61.7%) were primipara in our study.
4. The Mean PCV within groups before starting therapy and at term is statistically significant P<0.001. When compared between groups, there is no statistical signifance in PCV between intravenous and oral group.
5. The Mean MCV within groups before starting therapy and at term is statistically significant P<0.001. When compared between groups there is no statistically significance in MCV between intravenous and oral group.
6. Mean rise in HB at term is 0.969 in group A,1.599 in Group B, 0.729 in Group C. There is statistically significant rise in HB fro pretreatment value to HB at term in all the three groups. Group B is statistically significant P<0.000 when compared to Group A and Group C.
7. Only 3.5% of Women had minor drug events in intravenous iron sucrose group. No major Anaphylactic reactions noted during our study. But 25% of Women on Oral iron had side effects of gastrointestinal symptoms.

CONCLUSION

1. From our present study , Intravenous iron sucrose is highly efficacious in improving hemoglobin when compared to oral iron in the prophylaxis of iron deficiency anemia. There is no statistical difference between intravenous group and oral group in terms of packed cell volume, Mean corpuscular volume.
2. Three dose of iron sucrose improves hemoglobin significantly with no serious side effects, compared to two doses of iron sucrose and oral ferrous sulphate in the prophylaxis of iron deficiency anemia in pregnancy.
3. Intravenous iron sucrose was well tolerated and safe and there were no major adverse reactions.

To conclude three doses of intravenous iron sucrose is safe, convenient, more effective in the prophylaxis of gestational anemia compared to oral iron.

Intravenous Iron sucrose could be a better alternative to oral ferrous sulphate In the prophylaxis of iron deficiency anemia in pregnancy so as to reduce the maternal mortality and morbidity.

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PROFORMA

STUDY OF IRON PROPHYLAXIS IN PREGNANCY – INTRAVENOUS VERSUS ORAL ROUTE

Name: Age :

OPNo:

Address:

Occupation: Phone no:

Income: Group:

Socioeconomic Class:

Obstetric score:

Gravida: Para: Live: Abortions:

Presenting complaints:

H/o Easy fatiguability / giddiness/Hook worm infestation

H/o Bleeding per vaginum/ Hematemesis / malena

H/o Anorexia / indigestion/ Breathlessness/ Swelling of legs

H/o Multiple pregnancy /Puffiness of face /Iron intolerance

Past H/o:

H/o Blood loss in between pregnancy

H/o DM, HT, Asthma, epilepsy, TB

Menstrual H/o:

Age at menarche: Cycles: Regular/ Irregular flow:

Obstetric History:

H/o Antepartum Hemorrhage / Postpartum Hemorrhage/ Blood Transfusion

General Examination:

Features of anemia: Yes/ No
Pallor /Glossitis / Facial Puffiness/ Koilonychia

VITALS:

Temp: PR: BP:

CVS: RS:

Investigations:

To rule out iron deficiency anaemia:

1. Hb:

2. Urine: Albumin

Sugar, deposits

3. Peripheral smear

4. PCV:

5. MCV:

POST THERAPY ASSESSMENT:

	Pre Treatment	**4 Weeks**	**8 Weeks**	**At Term**
HB				
PCV				
MCV				

Parameters monitored during therapy:
Adverse effects Yes/ No

1. Anaphylactic reaction
(Shivering, Hypotension)

2. Nausea / Vomiting

3. Thrombophlebitis

4. Abdominal Pain/gastritis

5. Diarrhea/ constipation

6. Chills / Rigors

7. Joint pain

8. Giddiness

9. Palpitation

10. Chest discomfort

ABBREVATIONS

WHO	-World Health Organisation
CDC	- Center for Disease Control
ICMR	- Indian Council of Medical Research
HB	- Haemoglobin
MCV	- Mean Corpuscular Volume
PCV	- Packed Cell Volume
RBC	- Red Blood Corpuscles
I.V	- Intravenous
TIBC	- Total Iron Binding Capacity
NNACP	- National Nutritional Anemia Control Programme
NFHS	- National Family Health Survey
IDA	- Iron Deficiency Anemia
DLHS	- District Level Household Survey

MASTER CHART GROUP A

S.NO	NAME	AGE	OP NO	OBS CODE	SES	PRE-HB	PRE-PCV	PRE-MCV	4 WKS HB	4 WKS PCV	4 WKS MCV	8 WKS HB	8 WKS PCV	8 WKS MCV	TERM HB	TERM PCV	TERM MCV	ADV EFFECTS
1	Saraswathy	20	6000	G3P2L2	IV	10.2	34	86	10.2	34	87.2	10.8	35	88	11.8	34.1	86.4	0
2	Rekha	20	5883	G2P1L1	IV	10.6	35	87	10.8	36	87.1	11.2	35	88	11.6	35	86.2	0
3	Amudha	28	6201	Primi	IV	10.4	34	86	10.6	36	86.5	10.9	36	85	11.5	32.4	85.5	0
4	Nalini	28	6190	G2P1L1	IV	10.2	33	80	10.6	34	82.7	10.6	35	82	11.6	33.4	82.5	0
5	Indra	24	6232	Primi	IV	10.9	34	88	10.7	36	88.2	11	35	88	11.5	35	86.4	0
6	Rajalakshmi	27	6367	Primi	V	10.8	35	89	10.7	36	87.2	11.2	36	86	11.4	33.6	85.4	0
7	Ezhilarasi	21	1720	Primi	IV	10.5	35	84	10.8	38	87.2	11.2	36	86	11.8	34	84.1	0
8	Vishalakshi	22	6905	Primi	IV	10.4	34	85	10.8	35	85.6	11	35	86	11.6	34.2	84	0
9	Tamilselvi	31	6845	Primi	IV	10.8	34	86	11.2	35	86.1	11.3	34	85	11.5	34.2	84.5	0
10	Thenmozhi	28	6814	Primi	IV	10.4	34	85	10.8	35	85.9	11.2	33	86	11.7	34.6	85	0
11	Deepika	23	7151	Primi	IV	10.2	34	86	10.6	35	85	11.2	36	84	11.5	34.2	86.2	0
12	Maimoon	33	7173	G2A1	IV	10	33	81	10.8	35	82.3	11.1	35	83	11.4	33.8	83.2	0
13	Dhavamani	27	7196	G2P1L1	V	10.2	33	88	10.8	33	88.2	11	34	88	11.6	34	86.7	0
14	Lakshmi	25	7406	G2P1L1	III	10.2	33	86	10.8	34	87	11.1	34	87	11.6	34.2	84.6	P
15	Nithyapriya	22	7809	Primi	IV	10.7	34	84	10.8	36	84.2	11	35	86	11.6	34.2	84	0
16	Levinrani	24	8028	Primi	III	10.7	35	86	10.8	35	86.4	11.2	36	88	11.6	34.8	86.8	0
17	Parveen	25	8090	G3P1L1A1	IV	10.5	34	84	10.8	36	84	11	34	86	11.5	33.9	84.2	0
18	Janani	22	8503	Primi	IV	10.4	34	87	10.9	36	87.2	11	37	88	11.5	36.2	86	0
19	Nancy	20	9092	G3P1L1A1	IV	10.2	33	84	10.9	36	85	11	34	87	11.4	34.2	84.5	0
20	Lavanya	30	10031	Primi	IV	10.2	33	86	10.9	35	86.4	11.2	35	88	11.5	34.8	86	0
21	Uma	20	10025	G2A1	IV	10.6	35	84	10.9	37	86.5	11.2	36	85	11.4	33.7	85.2	0
22	Suganya	21	10544	G3PIL1A1	IV	10.6	35	86	10.9	36	85.9	11.1	34	88	11.3	32.8	85.4	0
23	Abirami	24	9437	G2A1	IV	10.4	34	82	10.9	35	84	11.2	36	84	11.5	34.7	84.1	0
24	Pavithra	24	10798	G2P1L1	III	10.6	35	86	10.9	36	87	11.2	35	86	11.5	34.9	86.2	0
25	Jamuna	24	11078	G2P1L1	V	11	36	85	11.1	36	85.6	11.4	37	85	11.8	33	85.1	0
26	Sharmila	27	11241	G1A1	IV	10.4	34	83	10.9	34	84.2	11.2	33	84	11.8	34	82	Pa
27	Radhika	28	11466	Primi	IV	10.7	35	86	10.9	35	87.3	11.2	35	87	11.7	35.6	86.4	0
28	Maheshwari	30	11640	Primi	IV	10.5	34	85	10.9	36	84	11.1	34	86	11.6	35.8	85.2	0
29	Nagammal	34	11852	G2P1L1	IV	10.2	33	87	10.9	35	82	11.1	33	88	11.5	35.2	84.2	0
30	Manimegalai	23	11992	G2P1L1	IV	10.6	34	84	10.9	35	84.6	11.2	35	84	11.4	35.1	84.2	0
31	Renuka	25	12161	Primi	IV	10.4	34	86	10.9	37	86.9	11	37	88	11.4	34.1	86.2	0
32	Kamatchi	35	12369	Primi	V	10.2	33	84	11	34	85	11.4	36	86	11.6	34.2	84.1	0
33	Reetamary	25	12444	Primi	IV	10.2	33	86	11	34	86.2	11.4	34	86	11.7	34.2	85.2	0
34	Revathy	27	12674	Primi	IV	10.1	33	87	11	35	86	11.3	35	86	11.6	35.4	86.3	0
35	Bhavani	20	12885	G2P1L1	III	10.2	33	82	11	35	84.3	11.3	35	83	11.6	35.3	85.5	0
36	Revathy	25	13245	Primi	IV	10.2	34	82	11	36	82	11.4	36	87	11.5	33.2	85.2	0
37	Prema	28	13421	Primi	IV	11.4	36	88	11.6	36	87	11.8	36	87	11.9	34.2	84	0
38	Gayathri	27	13562	G4P1L1A2	IV	10.4	34	84	11	35	84	11.3	36	87	11.6	35.8	84	0
39	Devi	21	13736	Primi	IV	10.2	33	85	11.1	34	84	11.2	36	86	11.5	33	85.2	0
40	Devi	22	14063	G2P1L1	III	10.4	34	84	11.1	36	84	11.5	36	85	11.8	33.1	84.2	0
41	Bhuvaneshwari	23	5724	G3P1L1A1	IV	10.4	35	84	11.1	36	85.6	11.4	33	85	11.6	33.4	84.5	0

42	Priya	20	14522	Primi	IV	10.6	34	82	11	35	84.2	11.4	35	83	11.5	34	85.2	0
43	Nirmala	23	14755	Primi	IV	10.6	35	89	11.1	36	88	11.4	36	86	11.6	35.4	86.2	0
44	Subhashini	21	15124	G2A1	IV	10.8	35	85	11.2	36	85	11.2	34	86	11.5	35.2	86.4	0
45	Shobana	20	15273	Primi	IV	10.4	34	84	11.2	35	83.9	11.4	35	83	11.4	36.3	83.2	0
46	Selvi	21	15590	Primi	V	10.4	334	85	11	35	85.6	11.4	34	86	11.6	35.6	84.6	0
47	Preethi	22	14658	Primi	V	10.5	36	84	11.2	36	85.7	11.4	36	86	11.8	35.8	86	0
48	Gowri	23	14662	G2P1L1	IV	10.4	34	86	11.2	35	85	11.4	35	88	11.6	34.7	85.2	0
49	Kalpana	25	15825	G2A1	IV	10.6	34	82	11.1	34	83.4	11.3	35	85	11.6	34	88.3	0
50	Lavanya	27	16288	Primi	IV	10.3	34	86	11	35	86.5	11.3	36	87	11.5	33.2	84.2	0
51	Jhansirani	28	16327	G2P1L1	V	10.9	35	82	11.2	35	83.2	11.3	36	84	11.6	34	83.4	0
52	Shanthi	33	16463	G3P1L1A1	IV	10.4	34	84	11.2	36	87	11.4	36	84	11.8	34.5	85.4	0
53	Menaga	21	16843	G2P1L1	IV	11.6	36	83	11.8	37	83	11.9	34	84	12	35.2	85	0
54	Sakunthala	22	16895	G2P1L1	V	10.6	35	85	11.1	36	85.4	11.3	36	86	11.5	35.4	84.6	0
55	Vanitha	23	17434	G2A1	IV	10.9	36	81	11.3	36	84.3	11.4	35	87	11.6	35.2	85	0
56	Thulasi	20	17669	Primi	V	10.8	35	84	11.2	36	85.7	11.4	36	86	11.5	34.2	84.9	0
57	Amala	22	17916	Primi	IV	10.9	36	84	11.3	36	88	11.5	36	87	11.6	35.3	84.5	0
58	Durga	24	18058	Primi	IV	10.8	35	87	11	37	84	11	35	86	11.5	35.4	87	0
59	Selvi	24	18110	G2P1L1	III	10.7	35	87	11.3	35	86.4	11.3	36	85	11.6	34.8	86.3	0
60	Backiyalakshmi	25	11176	Primi	IV	10.3	34	85	11	34	84.6	11.2	35	86	11.5	35.4	85.4	0
61	Shalini	27	18464	G2P1L1	IV	10.8	35	85	11.2	36	85.6	11.4	34	86	11.6	36.5	84.5	0
62	Clara	33	18417	Primi	V	11.4	36	87	11.4	36	87.2	11.6	33	87	11.8	35.1	87.1	0
63	Rajalakshmi	23	18680	Primi	IV	10.8	34	88	11.4	34	88	11.4	34	89	11.9	34.8	86.1	0
64	Priya	32	18707	Primi	IV	10.5	34	86	11.1	36	85.2	11.4	35	87	11.5	34.6	85.4	0
65	Selvi	28	18838	G2P1L1	IV	10.8	36	82	11.2	36	83.4	11.4	36	84	11.6	35.3	84.2	0
66	Priyadharshini	20	18821	Primi	IV	11	36	87	11.3	36	86.2	11.2	37	86	11.7	34.4	87	0
67	Jothilakshmi	20	18952	Primi	IV	10.9	36	85	11.3	36	86.5	11.4	36	88	11.4	35	84.3	0
68	Sindhuja	20	19091	Primi	IV	10.8	35	84	11.2	36	85.6	11.4	36	88	11.5	35.1	84.1	0
69	Sumalatha	20	19055	Primi	IV	10.5	34	89	11	36	87	11.4	36	84	11.6	35	86	0
70	Fathima	21	19402	Primi	IV	10.2	33	82	11	35	82.1	11.4	35	84	11.7	33.5	82.3	0
71	Mala	25	19484	Primi	V	10.8	36	84	11.3	36	83.8	11.5	36	84	11.8	34.6	85.6	0
72	Ramya	25	19715	Primi	IV	10.8	35	82	11.2	35	82.6	11.5	36	85	11.6	35	82	0
73	Aruldeepa	26	19987	Primi	IV	10.9	36	85	11.4	37	85	11.5	37	85	11.7	35.1	83.1	0
74	Renganayagi	28	19990	G2P1L1	V	10.4	34	83	11.4	36	11.6	10.6	35	87	11.8	35	83.4	0
75	Meena	21	20011	G2P1L1	IV	10.7	35	85	11.2	36	85.3	11.4	37	85	11.7	34.8	86.2	0
76	Chandra	28	20296	G2P1L1	IV	10.7	35	85	11.2	36	85.2	11.5	35	87	11.9	35.4	85	0
77	Ramya	32	20294	G2A1	IV	10.5	34	87	11	35	87.7	11.2	35	88	11.4	33.5	86.4	0
78	Revathy	25	20347	Primi	IV	10.5	34	85	11	36	85.9	11.2	35	86	11.4	34.5	86.4	Gi
79	Aishwarya	21	20377	G2P1L1	IV	10.5	35	85	11.5	38	86.2	11.6	36	86	11.7	33.9	84.1	0
80	Suguna	28	20576	Primi	IV	10.8	35	84	11.3	36	85.9	11.4	35	85	11.8	34.9	85.3	0
81	Manjula	20	20571	G3PIL1A1	IV	10.6	34	85	10.9	35	86	11.2	35	86	11.9	34	86	0
82	Geetha	20	20595	Primi	III	10.6	35	85	11.2	37	85.9	11.4	35	86	11.5	35.2	84.2	0
83	Jebina	22	20830	G2P1L1	IV	10.7	35	86	11.1	36	86.7	11.2	36	87	11.4	35.2	86.4	0
84	Sasikala	24	19707	Primi	IV	10.5	34	89	11	35	88	11.2	35	88	11.6	34.8	86.4	0
85	Dhakshayini	26	20952	Primi	IV	11	36	87	11.2	37	87.6	11.5	36	88	11.5	35.4	85.4	0
86	Ruby	27	21085	G2P1L1	IV	11.2	36	88	11.5	37	88.6	11.7	36	88	11.4	35.2	86.2	0
87	Malathy	20	21287	G3P2L2	IV	10.6	35	89	11.4	35	88	11.8	36	87	11.8	34.8	85.4	0
88	Malathy	21	21267	Primi	IV	11.4	36	86	11.6	37	87.6	11.8	37	88	11.8	33.9	85.4	0
89	Sathya	24	21351	Primi	III	11.8	34	86	11.8	38	87.2	12	36	86	11.8	35.4	85.2	0

90	Shenbagavalli	31	12409	Primi	IV	10.5	34	86	10.9	37	86.4	11.4	34	87	11.9	34.6	86.2	0
91	Riswana	27	21599	G2P1L1	IV	11.3	36	88	11.6	36	87.2	11.6	37	87	11.8	34.2	86.7	0
92	Jeyalakshmi	20	19531	G3P1L1A1	IV	10.9	35	84	11.2	37	84.6	11.4	37	86	11.5	35	84.2	0
93	Indhumathy	23	15425	G2P1L1	V	10.5	35	83	10.9	37	83	11.2	36	83	11.4	34.2	83.2	0
94	Renu	26	21858	Primi	V	10.6	34	89	11	35	87.9	11.2	35	88	11.4	35.1	86.6	0
95	Saridha	30	21908	G2P1L1	IV	10.8	36	89	11.3	34	87.5	11.5	36	86	11.6	35	82.3	0
96	Rajimunisha	20	22100	Primi	IV	10.4	35	89	10.9	37	88	11.6	36	87	11.7	34	84.2	0
97	Mariammal	26	22066	G3P1L1A1	IV	11.2	36	89	11.3	37	88	11.6	36	88	11.6	35.4	86.4	0
98	Vijayakumari	22	17370	Primi	IV	10.6	35	87	11	37	87	11.3	36	87	11.5	34.2	84	0
99	Shameemnisha	20	22267	Primi	IV	12.2	35	87	12.1	36	86.8	11.5	36	86	12.4	35.2	85.6	0
100	Priya	26	22243	Primi	IV	12	36	86	11.9	35	86.2	11.9	34	86	11.9	35.4	85.2	0

MASTER CHART GROUP B

S.NO	NAME	AGE	OP NO	OBS CODE	SES	PRE-HB	PRE-PCV	PRE-MCV	4 WKS HB	4 WKS PCV	4 WKS MCV	8 WKS HB	8 WKS PCV	8 WKS MCV	TERM HB	TERM PCV	TERM MCV	ADV EFFECTS
1	Razia	20	5944	IV	Primi	10	33	81	10	35	84.5	10.9	35	85	12.1	34.2	82.4	0
2	Radha	24	5894	IV	G2P1L1	10.5	34	85	11	35	85.6	10.9	35	86	12.3	34.2	86.9	0
3	Mubeena	22	6115	IV	G3P2L2	10.6	35	85	11	34	83	11.1	34	84	12.1	35.9	85.6	0
4	Kavya	30	6384	IV	G3A2	10.4	34	85	11	35	85.8	10.8	35	86	12.2	34.5	84.9	0
5	Manju	30	6380	IV	Primi	10.6	33	85	11	34	86.7	10.9	36	85	12.4	34.9	85	0
6	Ammu	27	6537	III	G2A1	10.8	36	84	11	35	85.3	11.2	35	85	12.1	36.8	84.8	0
7	Ambikadevi	27	6673	IV	G2A1	10.5	35	84	11	36	85.2	11.2	36	86	12.5	35.9	85.6	0
8	Kanniga	26	6703	V	Primi	10.5	34	85	11	36	84.6	11.2	36	84	12.6	35.7	86.4	0
9	Nandhini	21	6704	IV	G2A1	10.2	35	89	11	36	86.6	11.9	34	87	12.4	35.6	88.7	0
10	Saranya	20	6625	IV	G2P1L1	10.2	33	80	11	34	82.2	10.9	34	82	12.4	34.6	81.5	0
11	Selvarani	21	6954	IV	Primi	10.9	35	87	11	35	88	11.9	36	88	12.8	35.8	87	0
12	Shakila	27	6969	IV	G2A1	10.9	34	83	11	34	85.6	10.6	35	86	12.9	33.9	84.2	0
13	Bama	25	6992	IV	Primi	10.6	36	84	11	36	85.3	10.9	36	85	12.3	36	85.4	0
14	Sangeetha	21	7321	IV	G2	10.2	35	84	11	34	85.2	11.1	35	85	12.5	35.6	84.2	Gi
15	Renuga	25	3177	IV	G2P1L1	10.1	35	85	11	35	84.2	11	35	85	12.8	35.4	85.2	0
16	Karpagavalli	20	7630	V	G3P1L1A1	10.4	34	84	11	34	85.3	10.9	35	86	12.4	34.8	85.6	0
17	Nandhini	20	8050	IV	G3P1L1A1	10.6	34	82	11	34	84.5	11.1	35	83	12.8	35.2	83.2	0
18	Subha	21	8444	IV	Primi	10.6	36	82	11	37	83.2	10.9	37	83	12.9	36.1	83	0
19	Nithya	21	8973	IV	Primi	10.2	33	85	11	34	84	11.2	35	85	12.8	34.2	84.5	0
20	Revathy	22	10263	IV	G2P1L1	10.2	34	83	11	35	85.2	11.4	35	85	12.6	35.2	84	0
21	Mahalakshmi	25	10355	IV	G2P1L1	10.4	34	84	11	35	85	11.6	35	85	12.8	36	84.2	0
22	Vahidha	22	10632	III	G3P2L2	10.8	35	84	11	34	87	11.2	34	87	12.7	35.6	85.6	0
23	Kanimozhi	22	5962	IV	Primi	10.8	33	85	11	34	85.1	11.2	36	85	12.5	35.8	85.4	0
24	Sudha	27	10999	IV	Primi	10.5	34	87	11	34	85.6	11	35	86	12.7	33.7	87	0
25	Geetha	28	10922	V	Primi	10.2	33	82	11	34	84.4	11.1	35	86	12.6	35.6	83.2	0
26	Banumathy	26	11347	IV	G2A1	10.4	36	85	11	36	85.9	11.2	36	86	12.5	36.4	85.6	0
27	Nagadevi	24	11535	IV	G2P1L1	10.4	34	83	11	34	80	11.2	36	82	12.3	34.8	82.5	0
28	Belsi	24	11731	IV	G2P1L1	10.4	34	87	11	35	85.1	11.1	35	87	12.6	34.3	86.5	0
29	Lakshmipriya	25	11900	IV	Primi	10.8	34	84	11	35	84	10.9	36	84	12.4	34.6	84.2	0
30	Padmavathy	25	12212	III	G2A1	10.8	35	84	11	36	85.6	11.5	36	86	12.1	35.4	84.8	0
31	Saradha	26	12266	V	Primi	10.7	35	87	11	36	86.6	11.1	36	87	12.5	35.8	86.5	P
32	Mariam	25	12452	IV	G2A1	10.5	35	86	11	36	87	11	36	87	12.4	35.4	86.5	0
33	Nalini	27	12572	IV	G2P1L1	10.5	35	85	11	36	85.1	11.2	36	85	12.4	34.7	85	0
34	Vannamayil	24	12713	IV	G2A1	10.6	35	87	11	35	87.6	11.2	36	88	12.1	35.8	87	0
35	Komala	22	12921	IV	Primi	10.2	35	84	11	35	83.9	11.2	35	84	11.9	35.5	84.6	0

36	Jerinabegum	24	13329	IV	Primi	10.2	36	86	11	36	86.2	11.1	36	86	12.2	36.2	86.4	0
37	Sabiya	21	13461	IV	G2P1L1	10.4	34	86	11	37	87.2	11.6	37	87	11.8	34.7	86.7	0
38	Divya	22	13657	IV	G2P1L1	10.7	35	85	11	36	85.2	11.2	36	86	12.1	35.4	85.4	0
39	Kavitha	25	13743	IV	Primi	10.6	34	86	11	36	84.2	11.4	35	84	11.7	35.2	87	0
40	Padmavathy	23	14145	IV	G3P1L1A1	10.8	37	84	11	36	86.2	11.5	36	86	11.9	37	84.3	0
41	Devi	25	14306	IV	G2P1L1	10.4	35	85	11	36	85.2	11.2	36	86	12.5	34.9	84.8	R
42	Sasi	24	14337	IV	Primi	10.9	35	84	11	35	84.8	11.6	36	86	12.3	35.6	84.5	0
43	Shantha	24	14597	IV	Primi	10.8	35	84	11	36	85.2	11.9	37	86	12.5	36.4	85.4	0
44	ThelathBee	25	14777	IV	Primi	10.8	35	87	11	35	87	11.6	35	87	12.4	35.7	87.2	0
45	Sujatha	26	15210	IV	Primi	10.5	35	89	11	34	85.4	11.6	35	86	12.6	35.4	85	0
46	Prabhavathy	27	15352	IV	Primi	10.6	34	86	11	35	86	11.4	36	86	12.5	35.1	86.2	0
47	Sathya	21	15757	IV	Primi	10.8	34	84	11	34	85.5	11.4	35	83	12.3	34.7	84.2	0
48	Sathya	21	15925	IV	Primi	10.4	34	85	11	34	85.1	11.5	35	86	12.4	34.6	85.6	0
49	Divya	21	15972	IV	Primi	10.2	34	86	11	35	86.8	11.8	36	86	12.6	35.4	86.2	0
50	Jothi	22	16269	IV	G2P1L1	10.9	34	84	11	34	85.3	11.4	35	86	12.2	34.1	84.2	0
51	Sindhu	22	16632	IV	G2P1L1	10.5	34	86	11	36	84.3	11.1	36	84	12.4	35.6	86.5	0
52	Gomathy	23	16671	V	G2A1	11	36	86	11	37	86	11.6	36	86	12.1	35.7	86.4	0
53	Dhanalakshmi	30	16998	V	Primi	11.4	36	87	11	36	87	11	36	86	12.2	35.9	87.1	0
54	Supriya	24	17332	IV	G3P2L2	10.5	34	87	11	34	85.4	11.6	37	85	12.3	35.4	86.8	0
55	Janani	32	17537	III	Primi	10.5	36	85	11	35	85.1	11.6	35	85	12.4	35.8	85.4	0
56	Sharmila	20	17621	IV	Primi	11.2	36	85	11	36	87.4	12	37	87	12.4	35.9	85.4	0
57	Shanthi	24	18038	IV	G2P1L1	11	34	85	11	34	85.3	11.2	36	85	12.5	34.9	85.4	0
58	Mercy	27	18209	IV	G2P1L1	10.6	35	86	11	35	87.2	11.2	35	87	12.5	35.7	86.2	0
59	Tamilselvi	22	18468	IV	Primi	11	34	85	11	33	85.2	11.5	34	86	12.3	35	85.4	0
60	Sangeetha	25	11991	IV	Primi	10.8	34	87	11	37	87.6	11.4	36	86	12.4	34.8	86.9	0
61	Stella	23	16826	IV	Primi	11	36	87	11	36	86.2	11.6	36	86	12.3	36.4	86.5	0
62	Sofia	29	18531	IV	G2P1L1	10.6	37	85	11	36	83.8	11.5	35	84	12.5	36.1	84.2	0
63	Samantha	26	18527	IV	Primi	10.9	36	86	11	37	85.3	11.5	37	86	12.2	35	87.1	0
64	Mythili	31	18853	IV	G2P1L1	10.6	35	86	11	36	85.2	11.5	37	85	12.4	35.4	86.4	0
65	Saritha	23	18811	IV	Primi	10.8	35	87	11	36	86.9	11.8	36	85	12.3	35.4	87.2	0
66	Sarathy	20	18592	III	Primi	11.2	33	84	11	34	88.2	11.8	36	88	12.5	34.2	85	0
67	Vasanthi	21	18586	IV	Primi	10.9	35	86	11	36	86.3	11.5	37	86	12.1	36.4	86.4	0
68	Amutha	22	18978	IV	Primi	11	36	87	11	36	86.2	11.6	36	86	11.9	35.4	86.5	0
69	Ramya	23	19128	IV	Primi	10.8	34	89	11	35	84.6	11.6	36	85	11.8	35.2	87	0
70	Jansirani	23	19203	IV	Primi	10.9	34	84	11	35	85.2	11.7	35	86	12.2	35.8	85.4	0
71	Nirmala	24	19217	III	G3P1L1A1	11.2	37	89	11	36	87.2	11.8	36	87	12.4	36.8	88	0
72	Renuga	25	19616	IV	Primi	11	37	88	11	37	87	11.6	37	87	12.5	36.9	87.2	0
73	Vijayalakshmi	29	19641	IV	G2P1L1	10.9	36	82	11	36	83.1	11.6	36	83	12.4	35.8	82.3	0
74	Sathya	25	19654	V	G2A1	11	36	87	11	36	87.2	11.6	37	87	12.1	36.5	87.6	0
75	Priya	31	19886	IV	G3P2L2	10.6	35	85	12	36	86	10.9	36	86	11.9	35.8	85.6	0
76	Suguna	27	20111	IV	G2A1	10.5	34	84	12	38	85.4	11.6	37	85	11.9	34.6	86.4	0

77	Saridha	28	20129	III	Primi	10.9	36	85	12	36	85.6	11.8	36	87	12.2	36.5	84.6	0
78	Jabbuthin	21	20185	IV	Primi	10.9	35	82	12	34	83.2	11.5	36	83	12.4	35.2	83.4	0
79	Gomathy	21	20442	IV	Primi	11	36	85	12	36	87.1	11.6	36	87	12.6	35.4	85.4	0
80	Kosaladevi	27	20608	IV	Primi	10.9	34	86	12	34	88	11.8	36	88	12.4	35.4	86.2	CD
81	Sivasakthi	25	20628	IV	G3P1L1A1	10.3	34	85	12	37	83	11.4	37	84	12.8	34	85.3	0
82	Bhuvaneshwari	26	20603	V	G2P1L1	11.2	36	86	12	35	83	11.4	35	83	12.6	36.2	86	0
83	Renuga	28	20754	IV	G2A1	11.2	35	86	12	36	86.9	11.4	36	86	12.8	35.8	86.8	0
84	Sabana	20	20971	IV	Primi	10.7	35	86	12	36	86.5	11.4	36	87	12.4	35.8	87.2	0
85	Leelavathy	20	21074	IV	Primi	10.9	36	85	12	36	83.2	11.8	37	84	12.5	36.2	83	0
86	Mangai	23	21152	IV	Primi	10.8	34	86	12	35	86.3	11.8	36	87	12.6	35.2	86.2	0
87	Razia	24	21218	IV	G2P1L1	11.8	32	87	12	33	83.5	11.6	35	84	12.7	35.7	86.9	0
88	Ganga	28	21230	IV	G2P1L1	10.9	37	84	12	37	85	11.9	37	85	12.4	37	84.9	0
89	Mohana	20	21493	III	G2P1L1	10.5	35	84	12	35	84.2	12.1	37	85	12.5	33	85.2	0
90	Kamatchi	21	21519	IV	Primi	11.4	36	84	12	37	84	11.8	37	84	12.4	36.4	84.8	0
91	Anitha	22	21689	IV	Primi	10.9	35	84	12	36	85.1	11.4	36	85	12.1	35.4	85.1	0
92	Selvi	22	20657	IV	Primi	11.2	36	87	12	37	86.2	11.9	36	86	11.9	35.8	87.2	0
93	Velvizhi	24	21875	IV	G4P1P1A2	10.8	36	84	12	35	84.2	11.7	35	86	11.8	35.8	84.2	0
94	Kamatchi	30	21823	IV	Primi	11.8	34	85	12	34	88.2	12	37	88	11.8	36.4	86.2	0
95	Ruckmani	24	21985	III	Primi	10.8	34	84	12	34	84.2	11.6	35	84	12	35	85.1	0
96	Meena	25	22233	IV	Primi	10.5	34	84	12	35	83.4	11.9	36	83	12.2	35.4	83	0
97	Gayathri	24	22698	V	G2A1	12	37	85	12	36	85.2	12.2	37	86	12.1	37.2	85.1	0
98	Aswini	29	22791	V	G2P1L1	11.6	36	84	12	37	84.6	12.2	37	85	12.1	36.2	85.2	0
99	Devi	25	22972	IV	G3P1L1A1	11.6	35	86	12	36	85.6	12	37	86	12.2	34	85.6	0
100	Muniyammal	33	22623	III	Primi	12.6	35	87	13	35	86.7	12.9	36	87	12.5	35.4	86.2	0

MASTER CHART GROUP C

S.NO	NAME	AGE	OP NO	OBS CODE	SES	PRE-HB	PRE-PCV	PRE-MCV	4 WKS HB	4 WKS PCV	4 WKS MCV	8 WKS HB	8 WKS PCV	8 WKS MCV	TERM HB	TERM PCV	TERM MCV	ADV EFFECTS
1	Devika	24	5879	IV	G2P1L1	10.1	35	83	10	35	85.3	10.5	35	84	11	35.4	83.8	0
2	Vahitha	24	5980	IV	Primi	10.6	34	86	11	35	85.6	10.8	36	86	11.2	34.5	85.9	0
3	Mahalakshmi	29	6081	IV	G2P1L1	10.3	34	83	11	37	83	11.4	37	84	11.4	34.6	84.6	0
4	Yasmin	22	6065	IV	Primi	10.8	36	86	11	37	83.2	11	36	86	10.8	35.9	85.9	G
5	Padmavathy	20	6303	IV	Primi	10.1	34	82	11	35	84.5	10.8	35	83	10.8	33.2	83	0
6	Elavarasi	23	6357	IV	Primi	10.2	33	83	11	34	82.2	10.9	35	83	11.2	33	82.1	0
7	Saraswathy	25	6374	IV	Primi	10.2	35	86	11	35	85.8	10.9	35	86	11.1	34.2	84.6	0
8	Sivagami	31	6460	V	Primi	10.1	34	83	11	34	84.4	10.7	35	84	11	33.4	84.2	C
9	Yamini	20	6519	III	G2P1L1	10.6	35	84	11	34	83	10.9	35	85	11.2	35.9	85.4	0
10	Jothilakshmi	23	1792	IV	G2A1	10.2	33	83	11	35	83.9	11	34	83	11.2	34.2	83.5	0
11	Vidhya	26	6816	IV	G2P1L1	10.5	34	84	11	34	84.5	11.2	34	85	11.1	34.2	83.9	0

12	Rubeena	20	7015	IV	G2P1L1	10.5	34	84	11	34	85.6	10.9	36	84	11.2	34.1	83.6	C
13	Sheela	25	7060	IV	Primi	10.9	35	86	11	36	85.2	11.1	36	86	11.3	35.6	85.9	0
14	Esther	23	7157	IV	Primi	10.5	34	86	11	34	85.6	11.4	35	86	11.6	34.6	85.6	G
15	Sharmila	20	7235	IV	G2P1L1	10.8	33	86	11	34	86.7	11.1	35	86	11.4	33.1	85.4	0
16	Seethalakshmi	26	7345	IV	G2P1L1	10.2	33	85	11	36	86.6	11.1	36	84	11.2	34.2	85.1	0
17	Padmavathy	23	7544	IV	Primi	10	34	87	11	36	84.6	11	35	87	11.1	35.2	86.7	G
18	Sumithra	28	7586	IV	Primi	10.8	35	86	11	36	85.2	10.9	36	86	11.4	35.4	86.4	0
19	Megala	20	7587	V	G2P1L1	10.4	34	84	11	35	84	11.2	36	85	11.5	34.2	85	0
20	Vidhya	21	7686	III	G2P1L1	10.8	37	83	11	36	86.2	10.9	35	84	11.1	36.4	83.2	0
21	Vannapetchi	23	7980	IV	G2P1L1	10.4	34	85	11	37	87.2	11.2	36	86	11.5	34.2	85.2	D
22	Deepa	24	7931	IV	Primi	10.5	34	84	11	36	87.1	11	36	85	11.2	33.9	84.6	0
23	Lakshmi	25	8048	V	Primi	10.6	34	86	11	35	86	11	36	865	11.1	34.1	86.5	0
24	Suryakala	26	8112	IV	G2A1	10.4	34	86	11	36	85.2	11.4	36	86	11.8	33.4	85.4	D
25	Sangeetha	27	8265	IV	G2P1L1	10.2	33	84	11	34	84	10.8	35	85	10.9	35.4	83.6	0
26	Karpagam	19	8437	IV	G2P1L1	10.8	35	85	11	35	88	11.2	35	86	11.5	34.8	85.6	0
27	Kavitha	20	8633	IV	G2A1	10.2	34	83	11	35	83.4	11.2	36	84	11.2	34.8	83.2	0
28	Elakiya	24	8791	IV	Primi	10.4	34	85	11	34	85.3	11.2	36	87	11.4	35.2	85.4	G
29	Sivagami	29	8937	V	Primi	10.2	34	85	11	36	83.8	11	36	85	11.1	34.5	84.7	0
30	Fathima	30	9148	IV	Primi	10.1	33	87	11	35	84.2	11	36	86	11.1	33.6	86.3	0
31	Eswari	26	9326	III	G2P1L1	10.6	35	84	11	35	87	11.4	35	85	11.5	34.2	84.2	0
32	Janani	22	9546	IV	G2P1L1	10.2	35	84	11	34	85.2	10.9	34	86	11	34.2	84.1	0
33	Priya	23	9772	V	G2P1L1	10.4	34	87	11	36	85.9	11	36	86	11.2	35.2	87.2	C
34	Dhanalakshmi	25	9865	IV	Primi	10.4	34	83	11	34	80	11	35	84	11.1	34.1	84.2	0
35	Kalaivani	28	10317	IV	G2P1L1	10.5	33	86	11	36	85.1	11.4	36	86	11.4	33.9	86	G
36	Kala	30	10392	IV	Primi	10.6	34	86	11	34	85.1	11.2	34	86	11.5	34.1	86.4	0
37	Koteeshwari	21	10486	IV	Primi	10.8	35	83	11	34	87	11.1	35	85	11.3	34.8	84.1	G
38	Mumtaz	28	9382	IV	G2P1L1	10.9	36	86	11	36	85.6	11.2	37	86	11.5	35.8	86.2	0
39	Meera	33	10700	IV	G2P1L1	10.7	35	85	11	36	86.6	11.4	36	85	11.4	35.3	85.1	0
40	Manjula	30	10855	IV	Primi	10.8	36	87	11	37	85.3	11.4	37	87	11.6	35.4	86.4	0
41	Vijayalakshmi	34	10905	IV	Primi	10.5	36	87	11	35	87.6	11.6	35	86	11.8	35.2	86.4	0
42	Kavitha	21	11144	IV	G2A1	10.5	36	84	11	35	85.1	10.9	35	83	11.1	35.8	84.7	0
43	Ayesha	21	11258	IV	Primi	10.9	35	86	11	34	83.2	11.2	35	85	11.5	34.9	86.2	C
44	Rekha	25	11377	V	Primi	10.8	35	85	11	34	85.5	11.3	35	85	11.5	35	85.4	0
45	Rajeshwari	21	11580	IV	G2P1L1	10.5	34	85	11	36	84.2	11.3	36	86	11.6	34.2	85.4	0
46	Parvathy	22	11708	IV	G2P1L1	11.5	36	87	11	36	87	11.2	36	87	11.4	35.1	86.2	G
47	Gomathy	29	11758	IV	Primi	11.2	36	86	11	37	86.2	11.4	36	87	11.5	36.2	85	0
48	Aishwarya	24	11916	III	Primi	10.5	35	85	11	38	85.4	11.2	37	86	11.6	35.6	85.7	cons
49	Gowsalya	22	12025	IV	Primi	10.2	33	84	11	36	86.2	11.5	36	85	11.6	34.2	84.2	0
50	Parveen	20	12020	IV	G3P1L1A1	10.5	35	88	11	36	84.3	11.4	35	88	11.5	36.5	87.4	0
51	Praveena	23	11005	IV	Primi	10.7	36	83	11	36	83.1	11.2	36	83	11.1	35.9	83.2	0
52	Kodumpadi	27	12345	IV	Primi	10.6	34	86	11	34	84.2	11.2	35	87	11.4	34.2	86.3	0

53	Malliga	27	12365	V	Primi	10.6	33	85	11	35	86.8	11.2	36	85	11.1	34.1	84.7	0
54	Ramani	26	12410	IV	G2P1L1	11.1	36	88	11	37	86	11.2	37	87	11.5	35	87.6	gas
55	Shanthi	33	12536	IV	G2A1	10.9	36	85	11	35	84.8	11.3	35	85	11.5	35.4	84.6	0
56	Meenakshi	20	12574	IV	G3P1L1A1	11.2	37	87	11	37	87	11.4	36	86	11.5	36.4	87.2	0
57	Lakshmi	21	12691	IV	G2P1L1	10.8	34	86	11	36	85.2	11.4	36	85	11.5	34.2	85.7	0
58	Parameshwari	25	12761	IV	Primi	10.5	35	86	11	36	87	11.2	36	86	11.5	34.8	86.4	0
59	Sharmila	27	12877	IV	Primi	10.5	36	85	11	36	86	11.3	36	86	11.6	35.2	85.6	0
60	Devika	30	13031	IV	Primi	10.5	35	85	11	35	84.2	11.3	36	85	11.3	34.9	85.1	0
61	Meenakshi	23	13090	IV	G2A1	10.6	36	84	11	34	85.1	11.3	34	84	11.3	33.4	84.9	0
62	Selvi	25	13185	IV	G2P1L1	10.5	36	85	11	34	85.4	11.4	35	86	11.5	34.2	84.2	G
63	Saritha	26	13388	IV	G2P1L1	10.6	34	87	11	35	85.1	11.3	35	86	11.4	34.5	85.4	0
64	Yuvarani	30	13385	IV	Primi	10.9	36	88	11	35	84.6	11.4	35	88	11.6	36.1	87.9	0
65	Bharathi	21	13538	IV	G3A2	10.7	34	88	11	34	88	11.4	35	86	11.6	34.8	87.4	0
66	Sathyabama	30	13734	IV	Primi	10.7	34	85	11	34	85.4	11.3	35	86	11.5	34.2	84.9	0
67	Sathya	20	13877	V	Primi	10.6	35	85	11	36	85.2	11.5	37	86	11.6	35.4	85.4	0
68	Radhika	29	13992	IV	G2P1L1	10.4	34	84	11	35	85	11.4	35	84	11.2	36.2	84.5	0
69	Amudha	26	14074	IV	G3P2L2	10.8	35	83	11	35	83	11.4	36	85	11.5	35.4	83.9	C
70	Jothi	21	14280	IV	Primi	10.5	33	86	11	36	86.3	11.4	36	85	11.6	33.4	85.4	0
71	Geetha	30	14340	IV	G2P1L1	10.8	36	87	11	37	87.6	11.6	36	86	11.6	35.4	87.2	0
72	Radha	20	14460	IV	G2P1L1	11	36	87	11	36	86.2	11.4	37	87	11.2	36.4	87.2	D
73	Selvi	23	14498	IV	G2A1	11.6	33	87	11	33	83.5	11.2	34	87	11.2	34.8	86.4	0
74	Sumathy	24	14750	IV	Primi	11.6	34	87	12	34	88.2	11.8	34	87	12	33.9	86.4	0
75	Deepa	25	14934	V	Primi	10.5	37	87	11	37	85	11.4	37	87	11.5	35.9	86.4	0
76	Nagooramma	22	15223	V	G2P1L1	10.8	36	86	11	36	85.3	11.4	36	86	11.6	35.4	85.4	0
77	Varalakshmi	24	15300	IV	Primi	11.2	33	84	11	34	85.3	11.8	35	85	11.7	33.8	84.2	G
78	gnanavalli	25	15443	IV	G2P1L1	10.8	35	84	11	35	85.2	11.4	35	86	11.5	35.3	84.3	0
79	Sangeetha	26	15676	V	Primi	11	36	88	11	35	86.3	11.5	35	88	11.6	35	86.5	0
80	Gayathri	26	15764	IV	Primi	11.2	37	88	11	36	87.2	11.5	37	88	11.6	36.4	87.2	0
81	Usha	27	15824	V	G2P1L1	10.6	35	85	11	35	87.2	11	35	87	11.3	35.2	84.2	0
82	Radha	29	15961	IV	G2P1L1	11	37	87	11	33	85.2	11.4	34	86	11.8	36.5	86.5	0
83	Vijayalakshmi	31	16026	IV	G2P1L1	11.6	36	85	11	36	87.4	11.4	36	85	11.9	35.7	85.4	C
84	Kanagalakshmi	19	16053	IV	G2A1	11	36	85	11	36	87.2	11.7	35	86	11.7	35.2	85.4	0
85	Bagavathy	29	16152	IV	Primi	11.2	36	85	11	34	88.2	11.6	34	85	11.8	35.2	85.4	0
86	Vinothini	23	16306	IV	Primi	10.9	36	85	11	36	83.2	11	36	85	11.3	35.5	84.6	G
87	Pushpa	20	16375	IV	G3P1L1A1	11.1	36	88	12	36	85.6	11.2	36	88	11.4	35.4	87	0
88	Nandeeshwari	28	16423	IV	G2P1L1	10.9	35	87	11	36	86.9	11.4	36	86	11.6	35.2	85	0
89	Indrani	31	16575	IV	Primi	10.8	36	84	11	36	86.5	11.5	36	85	11.6	35	84.4	G
90	Laavanya	27	16597	IV	Primi	10.9	35	88	11	36	85.1	11.5	36	87	11.6	35.4	86.5	0
91	Backiyalakshmi	20	16751	V	Primi	11.2	35	84	12	36	86.9	11.6	36	85	11.8	35.2	84.3	0
92	Senthamarai	21	16928	IV	G2P1L1	10.9	35	84	12	35	85.2	11.6	35	86	11.4	35.6	84.6	0
93	Anitha	24	17218	IV	G2P1L1	10.9	34	83	11	34	85.3	11.4	34	85	11.4	34.8	83.4	0

94	Rekha	28	17277	IV	Primi	10.5	35	85	11	36	85.2	11.4	36	86	11.6	36.2	85.2	G
95	Varalakshmi	26	17537	IV	Primi	10.9	34	86	11	36	85.6	11.5	37	84	11.7	33.9	87	0
96	Gunasundari	22	17675	IV	G2P1L1	11.4	36	87	12	37	84	11.5	37	87	11.8	36.4	86	0
97	Anandhi	26	17728	V	G2A1	10.5	36	86	11	35	84.2	11.6	36	86	11.7	35.9	84.9	G
98	Parimala	29	17768	V	Primi	11.2	35	87	12	36	86.2	11.8	36	85	11.8	35.9	86	0
99	Vetriselvi	29	18013	IV	G2P1L1	11.6	37	86	12	37	86.2	11.9	35	85	12.2	36	85.5	0
100	Sabina	22	16976	IV	G2P1L1	11.8	35	87	12	35	86.7	12.2	37	88	12.4	35.4	86.5	G

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு: "கர்ப்பிணி பெண்களுக்கு ஏற்படும் இரத்தச் சோகையை தடுப்பதற்காக பயன்படும் மருந்து–மாத்திரைகள் மற்றும் ஊசி மருந்து மூலம் செலுத்தி அதன் வீரியத்தைக் கண்டறிவது"

ஆராய்ச்சி நிலையம் : சமூக மகப்பேறியல் மற்றும் அரசு கஸ்தூரிபாய் காந்தி தாய்–சேய் நல மருத்துவமனை
சென்னை மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை
சென்னை–600 003.

பங்பெறுபவரின் பெயர் :

பங்குபெறுபவரின் எண்:

மேலே குறிப்பிட்டுள்ள மருத்துவமனை ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் விளக்தகப்பட்டுள்ளது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கூட்டத்திலும் எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும், மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனத்துடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன், இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய் அறிகுறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி .....
கட்டைவிரல் ரேகை.

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
.....

சாட்சியின் கையொப்பம் ..... இடம் ..... தேதி .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி .....

ஆய்வாளரின் பெயர் .....

CONSENT FORM

STUDY TITLE : " IRON PROPHYLAXIS IN PREGNANCY: INTRAVENOUS VERSUS ORAL ROUTE "

STUDY CENTRE : Institute of Social Obstetrics and Govt. KGH, Chennai.

PARTICIPANT NAME : **AGE:** **SEX:** **J.D.NO.**

I confirm that I have understood the purpose of procedure for the above study, I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the procedure, I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

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

I hereby consent to participate in this study of " **IRON PROPHYLAXIS IN PREGNANCY: INTRAVENOUS VERSUS ORAL ROUTE "**

Signature of Investigator: Place :
Date :

Study Investigators Name Institution

Signature / Thumb Impression of patient Place :
Date :

Signature of Witness

Thanking you,

Yours faithfully,

KEY TO MASTER CHART

SE STATUS	- socioeconomic status
CD	- Chest discomfort
G	- Gastritis
C	- Constipation
D	- Diarrhoea
Pa	- Palpitation
R	- Rashes
Gi	- Giddiness
P	- Local Pain