DISSERTATION

TO COMPARE THE EFFICACY OF ORAL CARBONYL IRON VERSUS FERROUS SULPHATE IN MODERATELY ANEMIC PREGNANT WOMEN BELONGING TO 24-32 WEEKSOF GESTATION

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CERTIFICATE

This is to certify that this dissertation titled **"TO COMPARE THE EFFICACY OF ORAL CARBONLY IRON VERSUS FERROUS SULPHATE IN MODERATELY ANEMIC PREGNANT WOMEN BELONGING TO 24-32 WEEKS OF GESTATION".**

Submitted by **Dr. P. PRIYADARSENE** appearing for Part II M.D. Branch II Obstetrics and Gynecology Degree examination in March 2010 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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CONTENTS

INTRODUCTION

Anemia in pregnancy exists world over but is a very common problem in most of the developing countries. Anemia is major public health problem in economically disadvantaged segments of population in developing countries.

In country like India it is frequently severe and contributes to maternal morbidity and mortality. It deserves more attention than what it is currently receiving. Recently lot of programmes have been focused on safe motherhood but maternal anemia a problem of great concern.

Gender discrimination also plays a major role in increasing the prevalence of anemia in developing countries.

Magnitude of Problem

The world wide prevalence of anemia in pregnant women is estimated at 51% WHO states that 35% to 75% of pregnant women in developing countries and 18% of women in industralised countries are anemic.

In India, the incidence of anemia among expectant mothers is alarmingly high with hemoglobin level less than 10 gm%, 15 to 30% of maternal deaths are due to anemia.

In areas where malaria and hookworm infestation is endemic prevalence of anemia is high as 91%. The incidence of anemia is adolescent girls in slum areas estimated to 98% this adversely affect reproductive performance.

The prevalence of anemia has come down due to fortification, prophylactic iron supplementation and better health care programmes aimed at woman and children.

NEED FOR THE STUDY

Anemia is the most important complication in pregnancy in the developing countries not only because of its greatly increased incidence but also because of its severity **(Agarwal et al., 1999)**.

Several studies reveal that socioeconomic cultural factors influence dietary inadequacy during pregnancy which is attributed to poor purchasing power, literacy, ignorance, regarding nutritive value of readily available cheaper food stuffs, cultural taboos,

superstitions and large family ***(Menon Krishna et al., 1995; Ruth Bunnet and Brown Linding, 1993; Naik D. Jayashree, 1992)***. Nutritional iron deficiency anemia is a serious problem in pregnancy which affects 90% pregnant women.

Severe forms of anemia in the third trimester of pregnancy are invariably associated with cardiac failure. Highest maternal mortality rate in developing countries is due to severe anemia, contributing to 20% death both, directly and indirectly. It is also responsible for incidence of premature and low birth weight babies thus increasing perinatal and infant mortality and morbidity.

According to ***Hassan Masood (1991)***, anemia is prevalent among 50% - 90% of pregnant women especially in India, which is the number one killer. Despite considerable improvement and awareness in the antenatal care in developing countries and in spite of national anemia prophylaxis programme in India anemia remains a problem of great concern with regard to maternal morbidity, mortality and adverse outcome ***(Singh Kishore et al., 1995).***

Due to the above stated reasons this study emphasis on regular antenatal check up, to screen for anemia during every check up detect it earlier so that it can be treated adequately before the mother reaches term gestation. The goal of the treatment is to increase the hemoglobin status as rapidly as possible.

AIM OF THE STUDY

To Compare

- The response in each group by observing the rise in hemoglobin which can be a good predictor for oral iron therapy.
- The side effects in both groups of drugs
- The patient compliance in both groups, and then by assessing the efficacy of ferrous sulphate and carbonyl iron in moderately anemic pregnant women, belonging to 24 to 32 weeks of gestational age.

REVIEW OF LITERATURE

Definition of Anemia

Anemia is a condition of low circulating hemoglobin (Hb) in which the Hb concentration has fallen below a threshold lying at two standard deviations all below the median of healthy population of the same age, sex and stage of pregnancy.

WHO definition for diagnosis of anemia in pregnancy is a Hb concentration of less than 11 gm / dl and a hematocrit of less than 33%, although CDC (Centers for Disease control USA) proposes a cutoff point of 10.5 gm/dl during the second trimester.

Severity of Anemia

The Indian Council of Medical research uses four categories of anemia depending upon the hemoglobin levels in their study are:

S. NO	Category	Hb Status gm/dl
1.	Mild	10-10.9
2.	Moderate	7-10
3.	Severe	<7
4.	Very Severe (Decompensated)	<4

Causes of Anemia

I. Physiological Anemia or hydremia of pregnancy

II. Pathological Anemia

a. Nutritional – Iron, Folic Acid ,Vit B12, Protein.

b. Hemorrhagic.

Acute - Following bleeding in early pregnancy., H/o abortion or vesicular mole previously.

Chronic - Hookworm Infestation, bleeding hemorrhoids, ulcerative colitis, malignancies.

c. Hemolytic

Familial Hemoglobinopathies

Acquired Malaria, Severe Infection

d. Bone Marrow Insufficiency

Hypoplasia or aplasia due to radiation, drugs (aspirin, indomethacin)

CAUSES OF NUTRITIONAL ANEMIA DURING PREGNANCY

Nutritional Anemia is believed to be the most widespread nutritional disorders in the world. It essentially affects people in developing countries According to WHO 500 million to I billion individuals representing 15 to 20 percent of world's population is presently affected by this condition.

According to WHO definition, the "nutritional Anemia" encompasses all pathological conditions in which the blood hemoglobin concentration drops to an abnormally low level owing to a deficiency in one or several essential nutrients, regardless of the cause of this deficiency.

The etiology of nutritional deficiency of iron in developing countries is multifactorial. The causes include nutritional deficiency of iron, folate and vitamin B_{12}. Secondary effects of infections and parasitic infestation. An imbalance occur as a result of low nutrient intake, poor absorption, increased demand of nutrients during pregnancy, increased nutrient loss due to repeated pregnancies, short birth interval and menstruation. Dietary short coming are often associated with low socio economic status and related to cooking and dietary habits, local food taboos, ignorance, illiteracy and lack of knowledge regarding nutrition ***(Van De, 1998).***

The etiological factors of nutritional Anemia in pregnancy

a. Dietary factors.
b. Increased demand during pregnancy
c. Maternal causes
d. Sociocultural factors
e. Infections and infestation.

a) Dietary factors

Inadequate intake of food

a) Low nutrient intake is the commonest cause of nutritional anemia.

b) Main nutrients involved in synthesis of hemoglobin are iron, folic acid, Vit B12 and deficiency of these cause anemia.

Low iron absorption

Poor absorption and utilization of iron in the body is one of the causes of iron deficiency anemia during pregnancy.

Factors decrease Iron absorption

a) Phytates
b) Calcium
c) Tannins
d) Tea and coffee
e) Herbal drinks
f) Fortified iron supplements.

b) Increased Demand during pregnancy

Increased Demand for nutrients is one of the major of nutritional anemia during pregnancy ***(Bucksher et at., 1996).*** The increased blood volume during pregnancy results in dilution of red cells and a reduction in hemoglobin concentration. This dilutional anemia is potentially accentuated by the increased demands of iron folic acid leading to anemia.

Poor absorption of iron occurs due to the fact that Indian Diet is predominantly cereal based.

Though Indian diet has adequate iron content 20–25 mg/day several factors inhibits absorption, the most important being the phytate from the cereals. Deficiency of ascorbic acid and calcium inhibits iron absorption.

Factor Affecting Absorption

Factors increase absorption

a) Hydrochloric acid by favoring dissolution and reduction of ferric iron.

b) Reducing substance like ascorbic acid, amino acids containing sulphydryl radical, these reduce ferric to ferrous iron.

c) Meat contain organic iron and increased HCL secretion.

d) Food containing low phosphorus diet.

There is increased demand for iron as it is essential for the synthesis of increased hemoglobin required for the greater maternal blood as well as for the storage of iron in the fetus. When the mother does not meet this demand, she may develop features of iron deficiency anemia. It is need not only to compensate the usual losses in urine, faeces, cutaneous desquamation (around 0.5 – 1 mg per day) but also to cover the various pregnancy related expenditure. Total 1000 mg of iron is required. This represents an extremely large amount to meet. If not looked after it can result in iron deficiency anemia ***(Hercubury, 1991).***

The Requirements of iron is not confined only to those aspects of the fetus but also the placenta, the enlarged uterus and for increased volume that occurs during pregnancy.

The estimates of iron requirements are

Foetus	:	400 mg
Placenta	:	100 mg
Uterus	:	50 mg
Hb	:	320 mg
Total	:	870 mg

c) Maternal Causes

Depletion of nutrients occur in maternal body due to various reasons. The women may have entered pregnancy with compromised or absent iron stores. When further iron depletion occurs with advancing gestation anemia results ***(Zuspan and Huiligun, 1994).***

In tropics frequent pregnancies are the common cause of iron and folic acid deficiency anemia among pregnant mother ***(Buckshae et al., 1999)*** from depletion occurs due to

uncompensated menstrual loses and chronic blood loss due to repeated pregnancies.

Grand multipara is considered as one of the cause contributing to nutritional anemia during pregnancy as it is associated with increased incidence of disorders like hypertension, antepartum hemorrhage and others. The co-existing pathological condition like APH, PPH, abortions lead to iron deficiency results in iron deficiency anemia ***(Buckshae et al., 1996;*** **Modak et al., 1996).**

There are maternal causes which lead to nutritional anemia during pregnancy. These are:

a) Anemia in multigravida or multipara is mainly due to blood loss in previous deliveries or pregnancies.

b) Repeated abortions, APH, PPH.

c) Extra demand for iron, folic acid in multiple pregnancy.

d) Short interval between pregnancies.

i. Less storage of iron in the mothers body since it is already utilized in previous pregnancies.

ii. There is no time for storage of iron in the body for the next pregnancy.

d) Socio Cultural Factors

i) Poor socioeconomic Status.

As the women continue to be a socially disadvantaged group their poor status affects nutrition.

ii) Cultural belief, Cooking and Eating habits food taboos.

Cultural practices play an important role in cause of nutritional anemia in pregnancy. Because of taboos the pregnant mothers, avoid meat, fish, egg, jaggery, curds, milk and leafy vegetables. Over cooking of food, washing of vegetables several time may lead to loss of nutrients in the food. This may also lead to nutritional anemia in pregnancy ***(Vande 1998).***

e) Infection and infestation

i) Hookworm infestation

Hookworm infestation is considered as one of the cause of nutritional anemia among the pregnant women ***(Agarwal et al., 1999).***

Infestation with hookworm, roundworm and whipworm cause or aggravate iron deficiency anemia ***(Sephension, 1994).***

Hookworm infestation also leads to the deficiency of folic acid in pregnant women ***(Buckshae et al., 1996).***

ii) Malaria

Plasmodium falciparam cause profound anemia during an acute infection among pregnant woman ***(Agarwal et al., 1999).***

Erythropolesis

In human embryo hemopoiesis is first evident in yolk sac at 2 months, liver at 3rd month and to some extent spleen between

3rd and 6th month. Bone marrow activity begins by 4th and 6th month, it completely take over the process of hemopoiesis.

At birth bones are filled with red marrow, by 7 years marrow in long bones is less active, by age of 10 to 14 years fatty marrow extends proximally and 18 years normal adult distribution of marrow is in the skull, axial skeleton, pelvis shoulder, sternum ribs and with extension of proximal ends of long bones.

Stages of Erythropoiesis

i) Basophyllic proerythroblast

ii) Early Normoblast

iii) Intermediate normoblast

iv) Late normoblast

v) Reticulocyte

vi) Erythrocyte

Kinetics of Erythropoiesis

The time taken for erythropoiesis in human beings is about 7 days. The first 5 Days are occupied by division and maturation upto non nucleated reticulocytes in the marrow and the

reticulocytes normally mature for another 24 hours in the peripheral blood and spleen, after which it circulate for 120 days. Since about 1% circulating erythrocytes are destroyed each day, same number are formed and released. Factors regulating erythropoiesis.

a) Androgen stimulates, esterogen suppresses erythropoiesis,

b) Thyroid hormone and adrenocortical steroids stimulates erythropotesis

c) Nutritional factors like proteins. Vit B_{12} folic acid , Vit $B_{6,}$ riboflavin. Pantothenic acid, nicotinic acid, ascorbic acid, Vit E, mineral like copper cobalt, zinc and iron.

IRON COMPARTMENTS IN HUMAN

Hemoglobin: 1ml of packed RBC contain 1mg of iron and size of this compartment changes in anemia.

Storage: Ferritin and haemosiderin.

Ferritin: It is a water insoluble complex of ferric hydroxide and protein apoferritin. Uptake and release of iron by ferritin is very rapid. Ferritin occurs in all cells of the body. Its concentration correlates with iron store. Hemosiderin water soluble compound found predominantly in cells of monocytes macrophage system.

Myoglobin: Present in all skeletal and cardiac muscles cells.

Transport Iron: Active transport process explained by “Mucosal Block Theory”. According to this theory ferrous form absorbed in small intestine converted to Ferric form in the intestinal villi combine with apoferritin in the villous epithelial cell to form ferritin. Absorption of iron is controlled by the availability of apoferritin. If this is fully saturated no more iron is absorbed, and this protects from excessive absorption in anemia. In anemia excess ferritin is broken down to make more apoferritin available and thereby augments iron absorption. This intracellular ferritin is eventually excreted following desquamation of epithelial cells of intestine.

IRON TRANSPORT AND UTILIZATION

Iron after absorption circulate in the blood bounded to a β globulin transferrin which is normally 1/3 saturated with iron. Normal iron varies from 66 -140 microgram / 100 mg plasma.

Physiological changes in Hemoglobin during pregnancy

In pregnancy there is progressive increase in circulating blood volume due to increase in plasma and RBC volume.

Increase blood volume ranges from 30 – 70% of the non pregnant level.

Increase blood volume due to maternal size, level of placental and extra placental hormones and erythropoietin, influence the increase in blood volume.

The increase in plasma volume is 40 – 50%

The increase in RBC volume is 20%.

Anemia during pregnancy is a consequence of primarily of expansion of plasma volume without normal expansion of maternal hemoglobin.

This is called physiological anemia because

i) It begins in second trimester when the iron needs are fully met.

ii) Occurs in well nourished women

Physiological Anemia Occurs Because

i) Increase in plasma volume and RBC volume occur at different periods of pregnancy.

ii) Plasma volume begins to rise at 6 -8 weeks of pregnancy reaches peaks at about 32 weeks after it plateaus.

iii) The rise in RBC volume begins at 20 weeks and continue till term. There is trend for mean Hb fall in the first and second trimester and little rise later in the third trimester ***(Peck and Ariyes, 1979.)***

Hematological parameters

Normal blood values in non pregnant status.

RBC - 4.8 - 6 million / cumm

Hb - 12 – 14 gm / dl

PCV - 40 – 45%

Life span of RBC is 110 -120 days.

Normal blood values during pregnancy

RBC	-	4.5 million / cumm
PCV	-	37 – 42%
MCV	-	80 – 90 cubic microns
MCHC	-	28 – 34%
MCH	-	27 – 32 picogram
Colour Index	-	0.9 – 1

$$\text{Mean corpuscular Hb MCH} = \frac{\text{Hb in gm per 1000ml blood}}{\text{RBC in million / cumm}}$$

Normal = 27 - 32 Picogram

$$\text{Mean corpuscular Hb MCV} = \frac{\text{Volumes of packed cell per 1000ml}}{\text{RBC in million / cumm}}$$

Normal = 80-90 cubic microns

Mean corpuscular Hb = Hb in gm per 100 ml

$$\frac{\text{Concentration MCHC}}{\text{Volumes of packed cell per 100ml}} \times 100$$

Normal = 34 percent

$$\text{Colour Index} = \frac{\text{Hb (percentage of normal)}}{\text{RBC (percentage of normal)}}$$

Normal = 0.9 – 1

Symptoms and signs

Symptoms: complaints of fatigue, poor tolerance of exertion, weakness and lassitude. Other features are anorexia and indigestion, palpitation caused by ectopic beats, dyspnoea, giddiness and swelling of legs.

Signs

Pallor

Pallor of varying degree is evident. The colour of the lips, tongue, inner side of the lower eyelid, palms, and sole are pale. If

the colour of palmar creases are as pale as the surrounding skin the hemoglobin is usually less than 7 gm/dl.

Nailbed should be examined for pallor, the rapidity of capillary refill is an indicator of integrity of the cardiovascular system.

Koilonychia

Koilonychia is strongly suggestive of Iron deficiency and is demonstrated as spoon shaped nails.

Glossitis

Ulceration in the mouth and tongue. The painful dry tongue of glossitis is helpful in diagnosing Vit B12 deficiency anemia. Angular stomatitis is also associated.

Oedema legs

This is due to hypoproteinemia.

Cardiovascular Changes

Cardiovascular signs reflect decompensation, a soft systolic murmur may be heard in mitral area due to mitral incompetence. Crepitations may be heard at the base of lungs due to congestion.

Investigation

1. Maternal condition.
2. Fetal Condition.

Maternal condition: a)Type and degree of severity.

The investigations include,

a) Hb%

b) RBC Count.

c) Blood indices.

MCV – Normal 78 -92 cu micron.

<78 microcytic suggest iron def.

>92 macrocytic follate def.

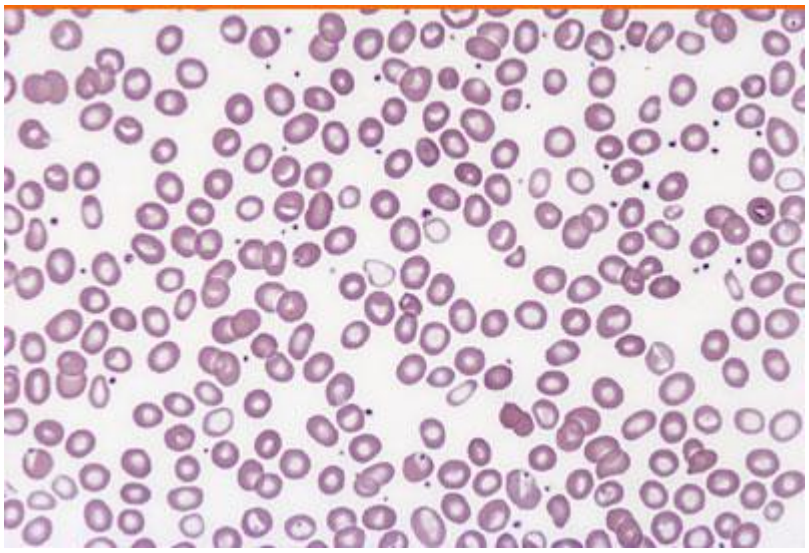
MCH – Normal 28 -33 pgm.

↓ In iron deficiency anemia

MCHC –33 – 36 %

↓ In iron deficiency anemia

Peripheral smear



MICROCYTIC HYPOCHROMIC BLOOD PICTURE

This is simple investigation often throws light on the etiology

a) Microcytic hypochromic cells.

b) Macrocytic hyperchromic cells.

c) Normocytic normochromic cells.

1) Microcytic hypochromic calls are seen in

   a) Iron deficiency.

   b) Thalessemia.

   c) Sideroblastic anemia.

   d) Anemia of chronic disease.

   e) Lead poisoning.

   f) Copper poisoning.

2) Macrocytic hyperchromic cells.

   a) Follate/ vit B12 deficiency.

3) Normocytic normochromic.

   a) Other causes or early nutritional deficiency

4) Schisto / aniso and poikilocytes:

   Hemolytic anemia.

5) Target cells:

   Thelessemia.

6) Sickle cells.

   Sickle cell anemia.

7) Hypersegmented polymorphs.

Seen in megaloblastic anemia.

8) Platelet:

May be decreased.

9) Parasites:

Such as malaria and leishmania may be seen.

B) INVESTIGATION FOR ETIOLOGY

Urine examination :

Urine albumin, sugar and deposits, and urine culture and sensitivity to rule out infection.

Stool examination :

- Parasites (ova of hookworm)
- Occult blood loss.

C) SPECIAL INVESTIGATIONS

1. Ferrokinetics:

These are tests that confirm the diagnosis and severity of iron deficiency anemia.

a) Serum ferritin

Normal - 15-300 μg/l

Abnormal when <12 μg/l

b) Serum transferrin

Normal 300 – 360 g/ml value increases with severity of anemia.

c) Serum iron

Normal 65 – 165 μ gm/dl. It is decreased in iron deficiency anemia.

d) Percentage saturation of transferring -

$$\frac{\text{Serum iron}}{\text{Total iron binding capacity}}$$

Normal 35-50%. This value is 15% in iron deficiency anemia.

e) RBC protoporphyrin – 30 gm/dl. This value is found doubled or tribled.

f) Total iron binding capacity

Normal 350 – 400 μg / 100 ml

Increased in iron deficiency anemia.

g) Free erythrocyte protoporphyrn (FEP): substrate used for heam synthesis

Increased in iron deficiency anemia

Mild IDA - zinc protoporphyrin concentration between 40–70 μ mol/ml

More advance IDA – ZPP is > 70 μ mol/ml

h) Serum Transferrin receptor measured by ELISA

This is new method to assess cellular iron status. It is particularly valuable in identifying iron deficiency anemia in pregnancy, since it is the only measurement to accurately reflect the iron deficit between the point of storage iron depletion and development of anemia.

2. Investigations in magaloblastic anemia.

a) MCV 110 – 140 cubic microns.

b) MCH 33 – 36 picograms.

c) Serum follate <3 ng/ml.

d) RBC follate <150 nm/ml.

e) Serum vit B12.

f) Peripheral smear macrocytic and hypersegmented neutrophil.

g) Serum B12.

h) Buffy cost.

i) FIGLU test.

3. Investigations in hemolytic anemia.

a) Osmotic fragility.

b) Coombs test.

c) Hemoglobin electrophoresis.

4. Tests to ensure therapy.

1. Reticulocyte count – increases.
2. Hemoglobin – increases.
3. Peripheral smear - decrease in abnormal cell type.

5. Screening test

Hemoglobin must be determined by Sahils hemoglobin method at the first antenatal visit. It should be repeated in all antenatal visit ideally at 32 weeks gestation when their haemodilution is at its peak.

Effects of nutritional anemia during pregnancy

Nutritional anemia among pregnant women is associated with adverse consequences to both the mother and the foetus. These adverse effects vary upon the severity of anemia and presence of other obstetric or systemic problems in anemic women **(Lewis and Chamberlin 1999).**

Effects on the mother

1) Iron deficiency anemia among pregnant women leads to diminished work capacity, physical performance and neurological dysfunction.

2) More prone for infections.

3) Antepartum and postpartum haemorrhage are more common in anemic mothers.

4) Antenatal mothers go for cardiac failure during labour.

5) Puerperal pyrexia.

6) Puerperal sepsis.

7) Subinvolution of uterus.

8) Lactational failure

9) Episiotomy wound gaping and abdominal wound gaping.

10) Abortion.

11) Folate deficiency leads to PIH, abruptioplacenta

12) Maternal and perinatal morbidity and mortality increased in anemic mother

Effect on fetus

Nutritional anemia due to iron and folic acid deficiency is the cause of abortion, premature birth, low birth weight, foetal wastage like abortion, intrauterine death, and stillbirth occur in

about 20% of the conception due to anemia **(Sethi et al., 1991; Kelton et al., 1988)** IUGR, impaired learning ability in children.

- Folic acid deficiency has been implicated as a cause of foetal malformation especially neural tube defects.
- Maternal iron deficiency anemia leads to lower iron rescue in infancy.
- Severe nutritional anemia among pregnant mother leads to increased risk of intrauterine hypoxia, higher perinatal and neonatal mortality and morbidity.

PREVENTION OF IRON DEFICIENCY ANEMIA

Prophylaxis of non pregnant women

Girls in India are deprived of good diet. As most women start their pregnancy with anemia or low iron stores, prevention should start even before pregnancy. The women of childbearing age should receive 60mg of iron daily for 2 – 4 months along with folic acid to prevent neural tube defects.

Iron supplementation during pregnancy

The ministry of health government of India now recommended intake of 100mg of elemental iron 500 µgm folic acid in the second half of pregnancy for a period of at least 100 days.

Treatment of hookworm infestation

Single dose albendazole 400mg or mebendazole 100 mg twice daily for 3 days with iron supplementation should be given to all anemic pregnant women.

4. Improvement of dietary habits

Pregnant woman should eat foods rich in iron jaggery, green leafy vegetables like spinach, mustard leaves, turnip green, cereals, sprouted pulses. Avoid tea and coffee intake, too much cooking should be avoided.

5. Proper antenatal care

a) Antenatal mothers should have regular antenatal checkup which should begin in first trimester of pregnancy.

b) Hb% estimation should be done during every A.N. visit.

c) Iron and folic acid should be consumed daily.

d) Proper treatment of infection.

6. Health education

1. By limiting number of children 1 to 2.
2. The ideal interval between 2 successive pregnancy should be 3 year.
3. Ideal age for child bearing is 20 – 25 yrs.

TREATMENT OF IRON DEFICIENCY ANEMIA

Oral iron therapy

If the women presents in mid trimester or early third trimester with anemia oral iron is started. Plenty of iron preparation are available each with own advantages and disadvantages.

Dosage

Ferrous sulphate tablets are used in all government hospital for oral treatment of anemia. For treatment more than 180 mg of elemental iron per day is required. Two tablets of ferrous sulphate each containing 100 mg of elemental iron per day is required for treatment.

Side effects

Upto 10% of women may have side effects with oral iron in the form of gastrointestinal symptoms such as nausea, vomiting, constipation, abdominal cramps and diarrhea, which are dose related, side effects can be minimized by advising to take the tablet with food.

Response to treatment

The patient's response to 180 mg elemental iron per day is fast with significant increase in Hb from 0.3 to 1 gm per week. Reticulocytosis occur within 5 – 10 days of treatment.

Duration of treatment

Treatment should be continued until the blood parameter become normal after which a maintenance dose of 1 tablet per day should be continued for at least 3 months after delivery.

Disadvantage of oral treatment

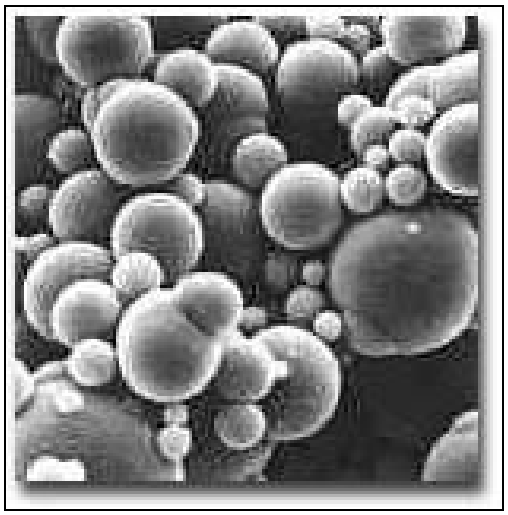
a) Intolerance of medication.

b) Noncompliance.

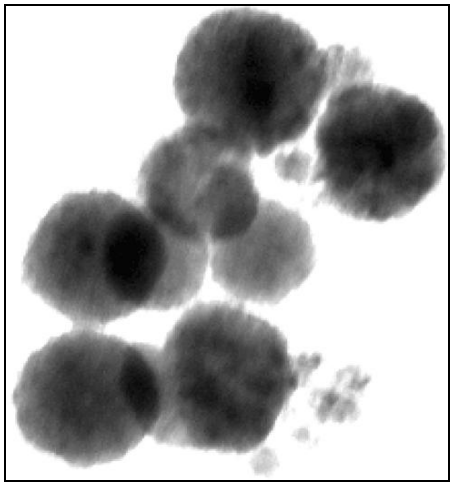
c) Unpredictable absorption.

d) Hemoglobin concentration restored with therapeutic dose but replenishing iron stores required treatment for longer periods.

If there is no significant clinical or hematological improvement within 8 weeks diagnostic reevaluation is needed. Reasons for failure to oral therapy involve inaccurate diagnosis of other causes of microcytic anemia such as thalessemia, phyridoxine deficiency, lead poisoning, continuous loss of blood through hookworm infestation, co-existing infection, faulty iron absorption concominant folate deficiency.



Carbonyl iron



Conventional iron

a) Ferrous sulphate

Ferrous sulphate available in government hospital contains 100 mg of elemental iron.

b) Carbonyl iron

The name Carbonyl iron comes from its process of manufacturing. This is a pure form of elemental iron that contains 98% mineral iron which is a small particle preparation of highly purified metallic iron. It is a form of elemental iron produced by a chemical carbonyl decomposition process. Carbonyl describes the process of manufacture of iron particles from iron penta carbonyl gas.

Features

Carbonyl iron is the purest form of iron; it is inert and hence cannot be chelated.

Bioavailability

Carbonyl particles are small < 5µ and have large surface areas which results in improved bioavailability.

Carbonyl iron has been shown to be well absorbed and utilized in Hb synthesis. Its advantage includes environmental stability.

Safety Profile

Carbonyl iron is much less toxic than ionized forms of iron. In humans lethal dose of ferrous sulphate is 200 mg/kg. In recent study no serious toxicity was reported in all patients with mean carbonyl iron ingestion of 11.2 gm/kg.

Side effects

Carbonyl iron does not have many of the side effects associated with other iron product particularly ferrous sulphate such as GI irritation, nausea, constipation or diarrhea. There was no evidence of hematologic, hepatic or renal toxicity.

Advantages of carbonyl iron

i) It is 98% pure form. The percentage of elemental iron in carbonyl iron is 100%.

ii) Unlike other preparation it is inert and incapable of reacting with chelators of iron such as transferrin and desferraxamine.

iii) It leads to rapid rise in hemoglobin.

iv) It is a wide margin of safety.

v) It is free from gastric side effect.

vi) It is safest in pregnancy.

MANAGEMENT OF LABOUR IN ANEMIC PATIENTS

First Stage

Patient should be in a comfortable position. Sedation and pain relief should be given. Oxygen should he kept ready and is given if dyspnoea. In case of preterm labour, betamimitics and steroids should be given with caution to avoid risk of pulmonary oedema. Digitalization may be required in cardiac failure due to anemia. The aim is to deliver vaginally.

Second Stage

The second stage is very stressful as the patients may go in for cardiac failure. Prolongation of second stage can be curtailed by forceps.

Third Stage

Third stage must be energetically treated as these patients tolerate bleeding very poorly. Prophylactic methergin should be given. Oxytocin 20 U infusion slowly given. Episiotomy wound should be sutured faster.

Puerperium

During puerperium mother should have adequate rest. Iron and folate therapy should be continued for at least 3 months. Any infection must be energetically treated. The anemic patient must use an effective method of contraception and should not conceive for at least 2 years until the iron stores are replenished. Sterilisation is preferred if the family is completed.

Several studies were done to compare the efficacy of different treatment using oral and parentral iron.

A prospective partially randomized study of pregnancy outcomes and hematologic responses to oral carbonyl iron treatment in moderately anemic pregnant women This was done in Moulana Azed Medical Collage, New Delhi, conducted by Jaib Sharma, Sandya Jain, Venkatesan Malika, Tejinder Singh, Ashok Kumar, This study compared the safety and efficacy in treating pregnancy anemia with carbonyl iron and ferrous sulphate. Changes in haemoglobin iron indicators, pregnancy outcomes and birth weight were compared between the 2 groups. The result obtained were serum ferritin concentration rise after carbonyl iron treatment better than oral ferrous sulphate group.

Similar other studies

1. “A novel regime carbonyl iron therapy” Srivastava Aarthi, Tanden Prabha, Darvineta conducted in ICMR Deparment of Obstetric and Gynaecology KGMU Lucknow.
2. carbonyl iron the treatment of iron deficiency anemia of pregnancy stein ML. Gunston KD, Mey RM.
3. New Schedule of carbonyl iron administration for pregnant women Mahale AR. Shah SH.

Bioavailability of carbonyl iron a randomized double blind study Devasthali SD. Gordeuk VR. Brittenham GM. Bravo JR, Compared ferrous sulphate with carbonyl iron in blood donors. The Study Showed bio availability of carbonyl iron was better with carbonyl iron.

High does carbonyl iron in iron deficiency anemia a randomized double blind trial Gordeuk, Britterbharm. The study concluded that high dose carbonyl iron was well tolerated than standard ferrous sulphate in higher dosage.

MATERIALS AND METHODS

The study was conducted from April 2009 – September 2009 at Government RSRM Lying – Hospital attached to Stanely Medical College, Chennai.

The antenatal women attending the antenatal OPD between the gestational age of 24 – 32 weeks were screened for Hb status and 200 antenatal women who were having Hb between 8 – 9.5 gm% were selected for the study.

Exclusion Criteria

1. Gastrointestinal bleeding or mal-absorption, Bleeding piles and bleeding from any other site is excluded.
2. Previous blood transfusion
3. Recent administration of iron for treatment of anemia
4. Woman with parasitic infection
5. Multiple pregnancy

6. Woman with diabetes, hypertension significant medical complication such as heart disease or any other systematic disease are excluded.

INCLUSION CRITERIA

- ❖ Gestational age 24 – 32 weeks of pregnancy
- ❖ Moderately anemic (Hb 8 to 9.5 gms %) pregnant woman attending OPD at RSRM

INVESTIGATION PERFORMED

1. Hb estimation by Sahlis hemoglobinometer
2. Peripheral smear with Leishman Stain
3. Packed cell volume
4. Mean corpuscular volume
5. Mean corpuscular hemoglobin concentration
6. Motion ova and cyst
7. Bleeding time
8. Clotting time

METHODOLOGY

In all pregnant woman included in the study a written informed consent was obtained.

Both the groups were allocated in such a way that body mass index, gestational age and hemoglobin status initially was equal in both treatment groups.

After selection patient was subjected to detailed history & physical examination.

All patient subjected to investigation like hemoglobin, peripheral smear, packed cell volume, mean corpuscular volume, mean corpuscular hemoglobin concentration, platelet count, bleeding time, clotting time and motion ova cyst.

THE TWO TREATMENT GROUPS ARE

100 of selected Group A received ferrous sulphate one tablet of 100 mg of elemental iron thrice a day with folic acid 0.5 mg one tablet per day.

100 of selected Group B received carbonyl iron one tablet of 100 mg of elemental iron twice a day with folic acid 0.5 mg one tablet per day.

Patient evaluated apart from baseline at 2 weeks interval. Clinical evaluation for anemic improvement like improvement in appetite and well being was enquired. Patient was also enquired about the drug complications like gastritis and about the colour of the stool. Adverse effect if any reported, were noted and vital like blood pressure and pulse rate were noted. Patient should be bring back the used empty pack.

At the end of 4 weeks, repeat hemoglobin and hemotocrit estimation was done. The results and data was analyzed with statistical test.

OBSERVATION AND RESULTS

Table 1

Initial Hb gm %	Group A Ferrous Sulphate		Group B Carbonyl Iron	
	No. of Patients	Percentage	No. of Patients	Percentage
8 to 8.5	23	23%	25	25%
8.5 to 9	28	28%	28	28%
9 to 9.5	49	49%	47	47%

In this study in Group A ferrous sulphate 23% of patient had initial Hb 8 - 8.5 gm%, 28% had 8.5 - 9 gm% and 49% had 9-9.5gm%.

In Group B carbonyl iron 25% of patient had initial Hb of 8-8.5 gm%, 28% had 8.5-9 gm% and 47% had 9-9.5 gm%.

Table 2

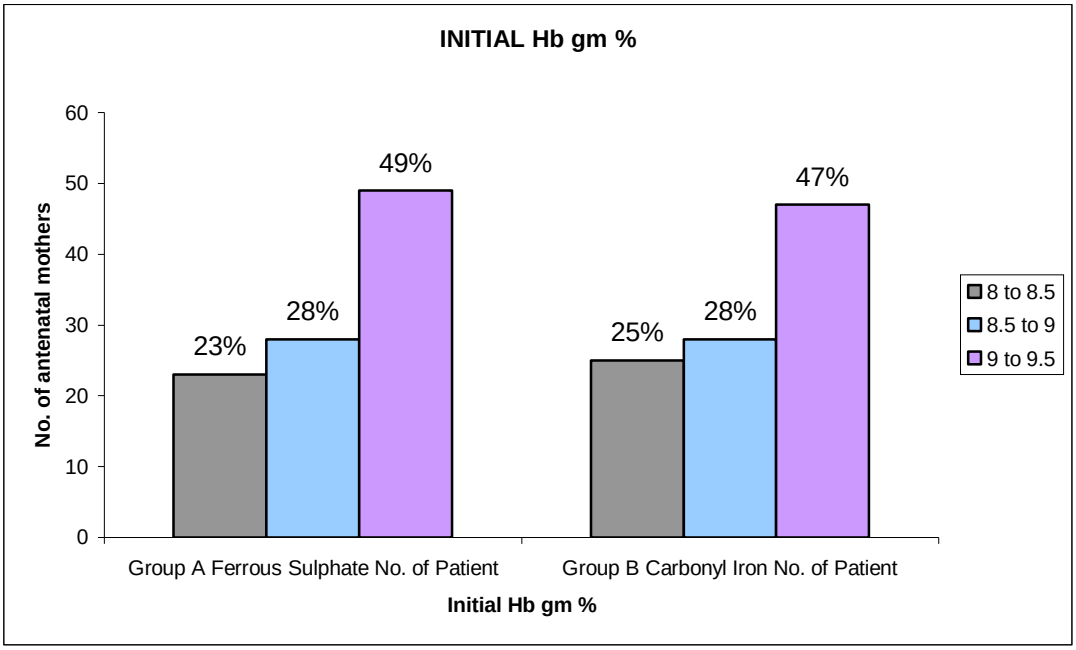
Rise in Hb gm %	Group A Ferrous Sulphate		Group B Carbonyl Iron	
	No. of Patients	Percentage	No. of Patients	Percentage
0.5 to 1	12	12%	-	
1 to 1.5	66	66%	34	34%
1.5 – 2	22	22%	54	54%
> 2	-		12	12%

The rise in Hb in ferrous sulphate group is 12% between 0.5 - 1 gm%, 66% had rise of 1 - 1.5 gm% and 22% had rise of 1.5 - 2 gm%.

In Group B carbonyl iron 34% of patient had rise of 1 - 1.5 gm%, 54% of patient had rise of 1.5 - 2 gm% and 12% showed a rise of > 2 gm%.

Conclusion

P value of less than 0.001 indicates the difference in “Rise in Hb” between ferrous sulphate group and carbonyl iron group is highly significant (Significant at 1%).


INITIAL Hb gm %
No. of antenatal mothers
60
50
40
30
20
10
0
49%
47%
28%
28%
23%
25%
8 to 8.5
8.5 to 9
9 to 9.5
Group A Ferrous Sulphate No. of Patient
Group B Carbonyl Iron No. of Patient
Initial Hb gm %

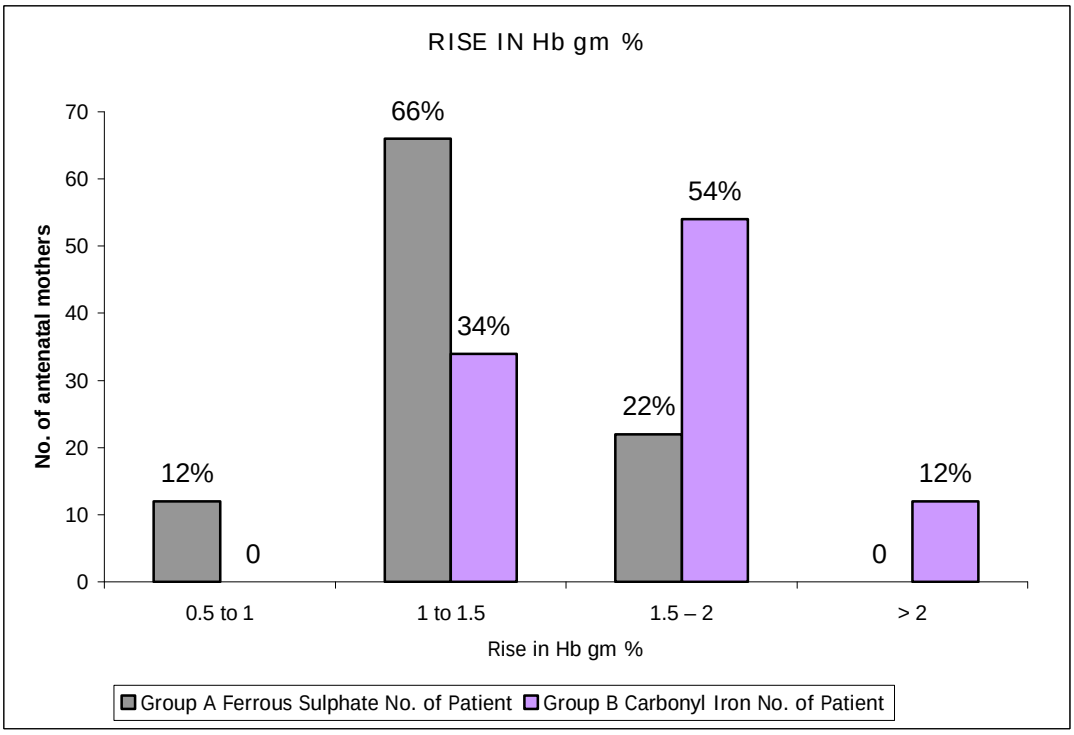

RISE IN Hb gm %
No. of antenatal mothers
70
60
50
40
30
20
10
0
66%
54%
34%
22%
12%
12%
0
0
0.5 to 1
1 to 1.5
1.5 – 2
> 2
Rise in Hb gm %
Group A Ferrous Sulphate No. of Patient
Group B Carbonyl Iron No. of Patient

Table 3

Initial Hematocrit in %	Group A Ferrous Sulphate		Group B Carbonyl Iron	
	No. of Patients	Percentage	No. of Patients	Percentage
23 to 26	23	23%	25	25%
26 to 29	77	77%	75	75%

In this study 23% of patient had initial Hct of 23 - 26% and 77% of patient had Hct of 26 - 29% in ferrous sulphate group.

In carbonyl iron group 25% of patient had initial Hct of 23 - 26% and 75% of patient had 26 - 29%.

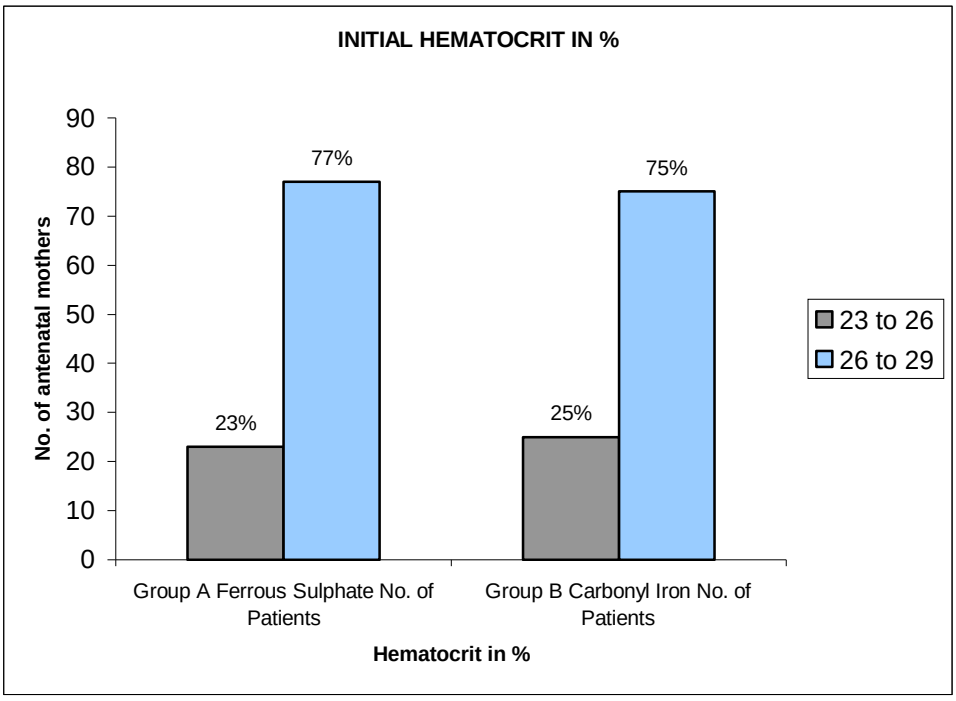


Table 4

Rise in Hematocrit in %	Group A Ferrous Sulphate		Group B Carbonyl Iron	
	No. of Patients	Percentage	No. of Patients	Percentage
1% to 4%	47	47%	12	12%
4% to 7%	53	53%	88	88%

In this study 47% of patient had rise in Hct of 1 - 4% and 53% of patient had rise of 4 - 7% of Hct in ferrous sulphate group.

In carbonyl iron group 12% had rise of 1 - 4% Hct and 88% had rise of 4 - 7% of Hct.

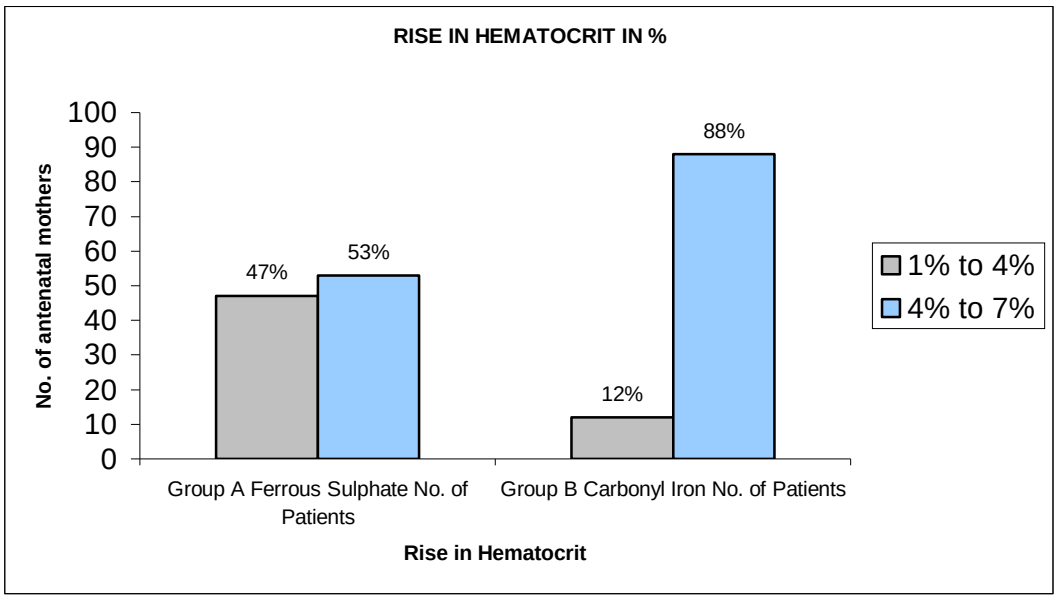


Table 5

RISE IN Hb gm % IN PATIENT WITH INITIAL Hb 8 TO 8.5 gm%

Hb Rise in gm %	Group A – Ferrous Sulphate	Group B – Carbonyl Iron
0.5 to 1	6%	0
1 to 1.5	15%	12%
1.5 to 2	2%	11%
> 2	0	2%

This table shows in ferrous sulphate group 15% had a rise of 1 - 1.5 gm%, 2% of the patient had rise of 1.5 - 2gm% and none had rise of > 2 gm%.

In carbonyl group 12% had rise of 1 - 1.5 gm%, 11% had rise of 1.5 - 2 gm% and 2% had rise of > 2 gm%.

Table 6

RISE IN Hb gm % IN PATIENT WITH INITIAL Hb 8.5 TO 9 gm%

Hb Rise in gm %	Group A – Ferrous Sulphate	Group B – Carbonyl Iron
0.5 to 1	3%	0
1 to 1.5	23%	10%
1.5 to 2	2%	17%
> 2	0	1%

In this table in ferrous sulphate group 23% of patient had rise of Hb 1 - 1.5 gm% and 17% of patient in carbonyl iron had rise of 1.5 - 2 gm% and 1% of the carbonyl group had rise of > 2 gm%.

Table 7

RISE IN Hb gm % IN PATIENT WITH INITIAL Hb 9 TO 9.5 gm %

Hb Rise in gm %	Group A – Ferrous Sulphate	Group B – Carbonyl Iron
0.5 to 1	3%	0
1 to 1.5	28%	12%
1.5 to 2	18%	26%
> 2	0	9%

In ferrous sulphate group 28% had rise of Hb 1 - 1.5 gm%, 18% had rise of 1.5 – 2 gm% and none had rise of > 2 gm%.

In carbonyl iron group 12% had rise of 1 – 1.5 gm%, 26% of patient had rise of 1.5 - 2 gm% and 9% of patient had rise of > 2 gm%.

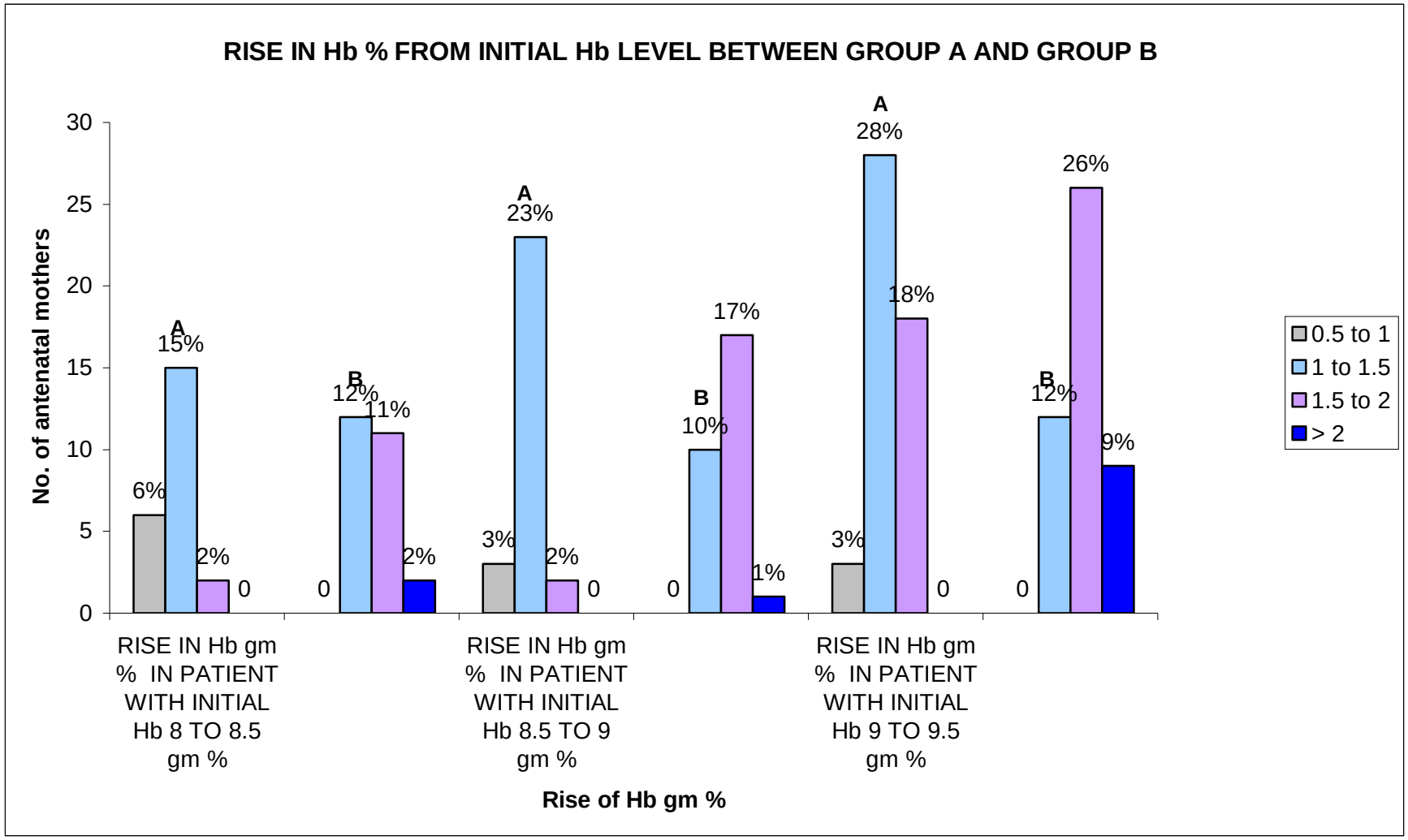

RISE IN Hb % FROM INITIAL Hb LEVEL BETWEEN GROUP A AND GROUP B
No. of antenatal mothers
30
25
20
15
10
5
0
A
15%
6%
2%
0
B
12%
11%
0
2%
A
23%
3%
2%
0
B
10%
17%
0
1%
A
28%
3%
18%
0
B
12%
26%
0
9%
0.5 to 1
1 to 1.5
1.5 to 2
> 2
RISE IN Hb gm % IN PATIENT WITH INITIAL Hb 8 TO 8.5 gm %
RISE IN Hb gm % IN PATIENT WITH INITIAL Hb 8.5 TO 9 gm %
RISE IN Hb gm % IN PATIENT WITH INITIAL Hb 9 TO 9.5 gm %
Rise of Hb gm %

Table 8

Adverse effect	GROUP A - Ferrous Sulphate		GROUP B - Carbonyl Iron	
	No. of Patients	Percentage	No. of Patients	Percentage
Constipation	7	7%	4	4%
Diarrhea	3	3%	2	2%
Epigastric Pain	16	16%	8	8%
Metallic taste	8	8%	12	12%
Nil	62	62%	72	72%
Vomiting	4	4%	2	2%

The adverse effect in group A ferrous sulphate as follows:

16% had epigastric pain, 8% had metallic taste, 7% had constipation, 3% had diarrhea and 4% had vomiting and 62% had no side effects.

In Group B carbonyl iron 12% had metallic taste, 8% had epigastric pain, 4% had constipation, 2% had vomiting and diarrhea and 72% had no adverse effects.

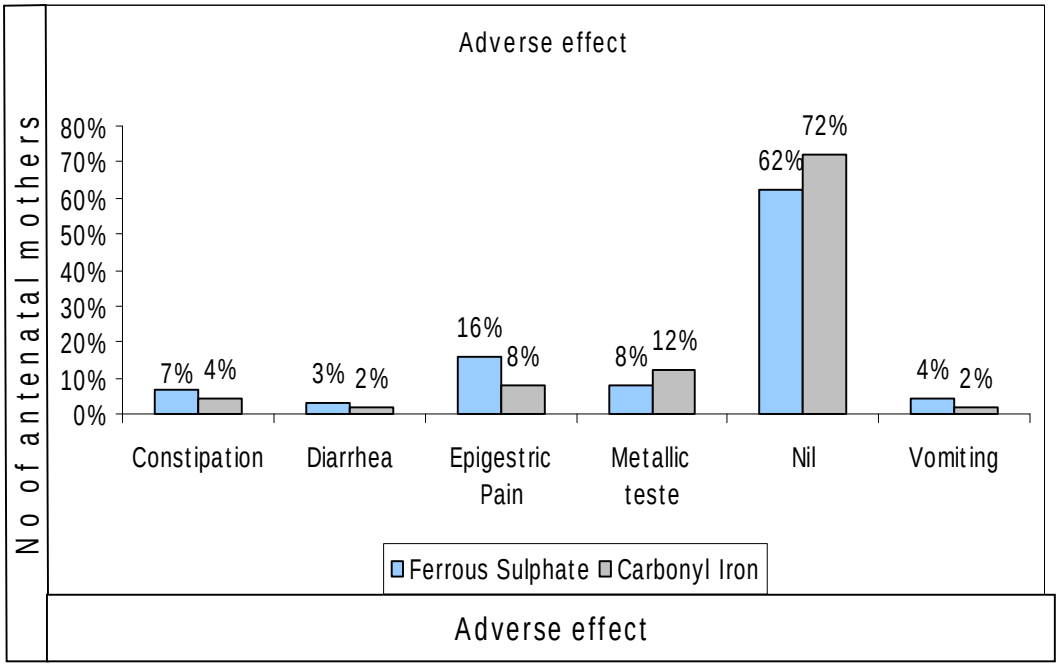
Table 9

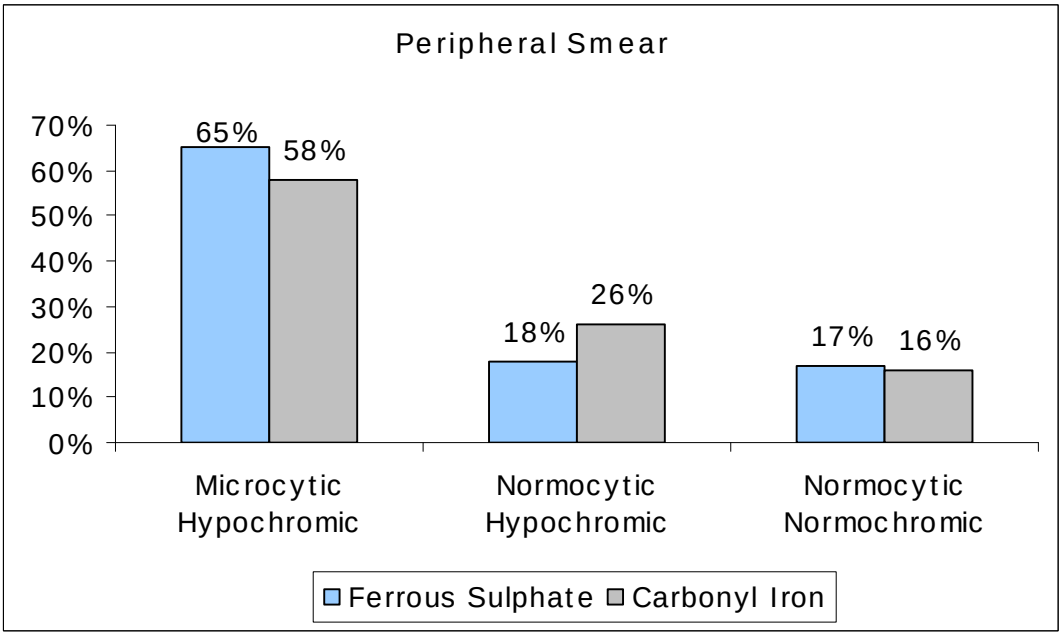
Peripheral Smear	GROUP A - Ferrous Sulphate		GROUP B - Carbonyl Iron	
	No. of Patients	Percentage	No. of Patients	Percentage
Microcytic Hypochromic	65	65%	58	58%
Normocytic Hypochromic	18	18%	26	26%
Normocytic Normochromic	17	17%	16	16%

65% of ferrous sulphate group and 58% of carbonyl iron group had microcytic hypochromic anemia blood picture.

18% of ferrous sulphate and 26% of carbonyl iron had normocytic hypochromic blood picture.

17% of ferrous sulphate and 16% of carbonyl iron had normocytic normochromic blood picture.


Adverse effect
No of antenatal mothers
80%
70%
60%
50%
40%
30%
20%
10%
0%
7% 4%
3% 2%
16% 8%
8% 12%
62% 72%
4% 2%
Constipation
Diarrhea
Epigestric Pain
Metallic teste
Nil
Vomiting
Ferrous Sulphate
Carbonyl Iron
Adverse effect


Peripheral Smear
70%
60%
50%
40%
30%
20%
10%
0%
65% 58%
18% 26%
17% 16%
Microcytic Hypochromic
Normocytic Hypochromic
Normocytic Normochromic
Ferrous Sulphate
Carbonyl Iron

Bivariate Analysis

Comparison of Age between Ferrous Sulphate Group and Carbonyl Iron Group

Group Statistics

	GROUP	Mean	Std. Deviation
AGE	Ferrous Sulphate	25.1500	4.48651
	Carbonyl Iron	24.5600	4.57114

Independent Samples Test

	t-test for Equality of Means					
					95% Confidence Interval of the Difference	
	t	df	P-value	Mean Difference	Lower	Upper
AGE	.921	198	.358	.5900	-.67308	1.85308

Conclusion: P-value of 0.358 indicates the difference in Age between Ferrous Sulphate Group and Carbonyl Iron Group is not significant.

Comparison of Parity between Ferrous Sulphate Group and Carbonyl Iron Group

PARITY_ * GROUP Crosstabulation

			GROUP		
			Ferrous Sulphate	Carbonyl Iron	Total
PARITY_	G2	Count	34	27	61
		% within PARITY_	55.7%	44.3%	100.0%
		% within GROUP	34.0%	27.0%	30.5%
	G3	Count	36	38	74
		% within PARITY_	48.6%	51.4%	100.0%
		% within GROUP	36.0%	38.0%	37.0%
	G4	Count	7	7	14
		% within PARITY_	50.0%	50.0%	100.0%
		% within GROUP	7.0%	7.0%	7.0%
	Primi	Count	23	28	51
		% within PARITY_	45.1%	54.9%	100.0%
		% within GROUP	23.0%	28.0%	25.5%
Total		Count	100	100	200
		% within PARITY_	50.0%	50.0%	100.0%
		% within GROUP	100.0%	100.0%	100.0%

Conclusion: P-value of 0.718(insignificant) indicates the distribution of parity does not differ across Ferrous Sulphate Group and Carbonyl Iron Group

Comparison of Gestational Age between Ferrous Sulphate Group and Carbonyl Iron Group

Group Statistics

	GROUP	Mean	Std. Deviation
GES_AGE	Ferrous Sulphate	27.7200	2.34448
	Carbonyl Iron	28.2814	2.55750

Independent Samples Test

	t-test for Equality of Means					
					95% Confidence Interval of the Difference	
	t	df	P-value	Mean Difference	Lower	Upper
GES_AGE	-1.618	198	.107	-.5614	-1.24559	.12279

Conclusion: P-value of 0.107 indicates the difference in gestational age between Ferrous Sulphate Group and Carbonyl Iron Group is insignificant.

Comparison of Initial Hb between Ferrous Sulphate Group and Carbonyl Iron Group

Group Statistics

	GROUP	Mean	Std. Deviation
INIT_HB	Ferrous Sulphate	8.7790	.38620
	Carbonyl Iron	8.8660	.35653

Independent Samples Test

	t-test for Equality of Means					
					95% Confidence Interval of the Difference	
	t	df	P-value	Mean Difference	Lower	Upper
INIT_HB	-1.655	198	.099	-.0870	-.19065	.01665

Conclusion: P-value of 0.099 indicates the difference in Initial Hb between Ferrous Sulphate Group and Carbonyl Iron Group is insignificant

Comparison of Final Hb between Ferrous Sulphate Group and Carbonyl Iron Group.

Group Statistics

	GROUP	Mean	Std. Deviation
FINAL_HB	Ferrous Sulphate	10.0950	.54576
	Carbonyl Iron	10.4590	.55489

Independent Samples Test

	t-test for Equality of Means					
					95% Confidence Interval of the Difference	
	t	df	P-value	Mean Difference	Lower	Upper
FINAL_HB	-4.677	198	.001	-.3640	-.51748	-.21052

Conclusion: P-value of less than 0.001 indicates the difference in Final Hb between Ferrous Sulphate Group and Carbonyl Iron Group is highly significant(significant at 1%).

Comparison of Rise in Hb between Ferrous Sulphate Group and Carbonyl Iron Group.

Group Statistics

	GROUP	Mean	Std. Deviation
RISE_HB	Ferrous Sulphate	1.3190	.30141
	Carbonyl Iron	1.6080	.28909

Independent Samples Test

	t-test for Equality of Means					
					95% Confidence Interval of the Difference	
	t	df	P-value	Mean Difference	Lower	Upper
RISE_HB	-6.920	198	.001	-.2890	-.37136	-.20664

Conclusion: P-value of less than 0.001 indicates the difference in "Rise in Hb" between Ferrous Sulphate Group and Carbonyl Iron Group is highly significant (significant at 1%).

DISCUSSION

Anemia is the commonest medical disorder in pregnancy. Iron deficiency can be corrected by administration of iron. The goal of treatment is to identify anemia at an earlier gestational age and treatment should be aimed at increasing hemoglobin level as rapidly as possible so as to decrease the maternal and fetal morbidity and mortality.

Ferrous sulphate iron tablet is available in all government hospitals and hence the result obtained with Ferrous sulphate and corbonyl iron were compared.

In my study only moderately anemic pregnant patients were selected due to ethical reasons that severely anemic pregnant women needs blood transmission.

In both groups majority of the patients had microcytic hypochromic blood picture followed by normocytic hypochromic blood picture.

In my study Carbonyl Iron showed a better rise in Hb percentage than ferrous sulphate (P value 0.001) which is significant at 1%. This is consistent with the observation made by Adul BB, Desai A, Gawde A, Baliga B in which there was a significant increase in average Hb level ($P<0.05$) which was significant at 1% from J. Indian Medical Association June 2005; 103 (6).

The better rise of Hb in Carbonyl Iron group could also be attributed to increase in Bioavailability of Carbonyl Iron as suggested by Devasthali SD, Godeuk VR, Brittenham GM from American Journal of clinical nutrition 1987 that overall bio availability of Carbonyl iron was high above 70% of ferrousulphate.

In my study the rise of Hb with short term therapy (4 weeks) was better than ferrous sulphate (P value 0.001). This is similar to the study by GOR Godeuk VR, Brittenhan Bravo J, Hughes MA, Keating LJ 1990 March which concluded that short terms iron supplement was effective against iron deficiency anemia with (P value 0.001).

In my study rise in Hb was a good predictor for the response of iron treatment in anemic pregnant women. This is similar to the observation made by Freire WB Am J Clin Nutr 1989 Dec.

The adverse effect profile was in favor of carbonyl iron in my study with less gastrointestinal toxicity this was similar to the study by Gordeuk VR Brittenham GM, Mclaren CE, Hughes Ma, Keating LJ March 1986.

With respect to cost benefit analysis the cost of ferrous sulphate tablets accounts to Rs. 75/- for 1 month. The cost of carbonyl iron for 1 month amounts ti Rs. 240/- for 1 month. Hence in affordable group carbonyl iron is preferred.

SUMMARY

In my study 54% of Carbonyl iron group showed a rise of 1.5-2 gm % and 12% showed a rise of more than 2 gm % of hemoglobin whereas in ferrous sulphate group 66% showed a rise of 1-1.5 gm % and 22% showed a rise of 1.5-2gm % and none showed a rise of more than 2 gm % hemoglobin

- Rise in hemoglobin percentage was better with carbonyl iron than ferrous sulphate.
- Carbonyl iron had less toxic to gastrointestinal mucosa.
- Patients compliance was better with carbonyl iron.
- It is safer than ferrous sulphate as there is less risk of causing poisoning due to over dosage.
- Carbonyl iron is inert and consists of small particles of metallic iron.

CONCLUSION

This study shows that the efficacy of carbonyl iron is significantly more than ferrous sulphate, even though the relative cost is more.

Affordable persons can be offered carbonyl iron.

In resource poor settings effective treatment of moderate anemia is possible with ferrous sulphate if diagnosed at an earlier stage.

Further large scale studies will raise the curtain for a better understanding of the effect of different iron preparation on different types of anemia.

MASTER CHART

GROUP - A - FERROUS SULPHATE

S.NO	Name	Age	Parity	BMI kg/mg2	Gestational age in weeks	initial Hb in gml%	Final Hb in gml%	Rise in Hb in gml%	Rise in Hct %	Peripheral Smear	Adverse Effect
1	Sarala	26	G2A1	18.4	32	9	10.3	1.3	4.0	Normocytic Hypochromic	Epigestric Pain
2	Sathiya	17	Primi	24	30	8.5	9.7	1.2	3.5	Microcytic Hypochromic	Nil
3	Roja	21	G3A2	22.8	28	9.5	10.7	1.2	3.5	Normocytic Normochromic	Nil
4	Uma	22	G2P1L1	22.8	28	9	10	1.2	3.5	Microcytic Hypochromic	Constipation
5	Savithiri	24	G3P2L1	23.3	26	9	10	1.2	3.5	Microcytic Hypochromic	Nil
6	Laskhmi	30	G3A2	22.2	28	8.4	9.7	1.3	4.0	Microcytic Hypochromic	Vomiting
7	Anandhi	32	G3P1L1A1	18.7	24	8.5	9.8	1.3	4.0	Microcytic Hypochromic	Nil
8	Latha	30	G4P2L2A1	26.14	24	9.2	10.8	1.6	5.0	Normocytic Hypochromic	Diarrhea
9	Barathy	17	Primi	24.14	30	9	10.6	1.6	5.0	Normocytic Hypochromic	Epigestric Pain
10	Kalai	18	Primi	20.95	30	8.8	10	1.2	3.5	Normocytic Hypochromic	Metallic taste
11	Anushya	22	G3P2L2	24	28	8.3	9	0.7	2.0	Microcytic Hypochromic	Nil
12	Padma	24	G4A3	22.5	28	9	11	2	6.0	Microcytic Hypochromic	Nil
13	Jothi	27	G2P1L1	22.4	30	8	10.2	1.2	3.5	Microcytic Hypochromic	Epigestric Pain
14	Vannila	28	Primi	23	32	9	8.9	0.9	2.5	Microcytic Hypochromic	Nil
15	Rajini	30	Primi	20	30	9	10.2	1.2	3.5	Microcytic Hypochromic	Nil
16	Jayashree	21	G2P1L1	19.5	28	8	10	2	6.0	Microcytic Hypochromic	Metallic taste
17	Jayalakshmi	24	G3A2	20.2	24	8.5	9.7	1.2	3.5	Microcytic Hypochromic	Constipation
18	Fathima	26	G2P1L1	23	28	8.7	9.9	1.2	3.5	Microcytic Hypochromic	Nil
19	Sheela	27	G4A3	21	24	8.2	9	0.8	2.5	Microcytic Hypochromic	Epigestric Pain
20	Egavalli	28	Primi	22	30	8.4	9.4	1	3.0	Microcytic Hypochromic	Nil
21	Anandhi	30	G4P2L2A1	18.3	24	9	10.1	1.1	3.0	Microcytic Hypochromic	Nil
22	Ramani	31	Primi	21.35	28	8	9.2	1.2	3.5	Microcytic Hypochromic	Nil
23	Bagyam	32	Primi	24.2	28	8.2	10.2	2	6.0	Microcytic Hypochromic	Nil
24	Rama	34	G3P2L2	18.6	30	9.2	10.5	1.3	4.0	Normocytic Normochromic	Nil
25	Ammu	35	G3A2	20	32	9	10.3	1.3	4.0	Normocytic Normochromic	Nil
26	Amudha	36	G2P1L1	21.6	28	8.5	9.7	1.2	3.5	Microcytic Hypochromic	Nil
27	Sumathy	27	G3P1L1A1	24.7	28	8.8	10	1.2	3.5	Microcytic Hypochromic	Epigestric Pain
28	Mala	28	G2P1L1	22	26	9.2	10.8	1.6	5.0	Normocytic Normochromic	Nil
29	Sarala	30	G2P1L1	21	26	9	10.4	1.4	4.0	Normocytic Hypochromic	Constipation
30	Mala	30	G3P1L1A1	18	26	8.4	9.3	0.9	2.5	Microcytic Hypochromic	Nil
31	Manjula	22	G3A2	18.6	30	8.2	9.3	1.1	3.0	Microcytic Hypochromic	Nil
32	Vatchala	21	Primi	20	32	9	10.2	1.2	3.5	Normocytic Hypochromic	Nil
33	Savithiri	17	Primi	21.6	28	8.6	9.6	1	3.0	Microcytic Hypochromic	Epigestric Pain
34	Sujatha	24	G3P1L1A1	24.7	28	9	10.2	1.2	3.5	Normocytic Hypochromic	Metallic taste
35	Kuppu	18	Primi	22	26	9.2	10.4	1.2	3.5	Microcytic Hypochromic	Nil
36	Rajeswari	22	G4A3	21	28	9	10.1	1.1	3.0	Microcytic Hypochromic	Vomiting
37	Vaniotha	21	G3P1L1A1	18	24	8.2	9.1	0.9	2.5	Microcytic Hypochromic	Nil
38	Sonia	30	G2P1L1	18	28	8.6	9.7	1.2	3.5	Normocytic Normochromic	Nil
39	Alamelu	17	Primi	26.1	30	9	11	2	6.0	Microcytic Hypochromic	Epigestric Pain
40	Jothi	28	G3P1L1A1	24.6	32	9.2	10.5	1.3	4.0	Microcytic Hypochromic	Diarrhea
41	Jenifer	31	G2A1L1	20.95	24	9.3	10.5	1.2	3.5	Microcytic Hypochromic	Nil
42	Kavitha	27	G2A1L1	24	24	8.5	9.4	0.9	2.5	Normocytic Hypochromic	Epigestric Pain
43	Latha	23	G3P1L1A1	22.5	28	8.6	9.9	1.3	4.0	Normocytic Normochromic	Metallic taste
44	Anuesya	22	G2P1L1	23	26	8.7	10	1.3	4.0	Normocytic Hypochromic	Nil
45	Kala	21	G3A2	20	30	9.2	10.8	1.6	5.0	Microcytic Hypochromic	Nil
46	Latha	21	G2P1L1	22.4	32	9	11	2	6.0	Microcytic Hypochromic	Constipation
47	Lavanya	19	Primi	24.2	28	8.2	9.8	1.5	4.5	Normocytic Normochromic	Nil
48	Ammani	21	Primi	18.4	24	8.8	10	1.2	3.5	Microcytic Hypochromic	Nil
49	Anitha	26	G2P1L1	24	26	9.2	10.5	1.3	4.0	Normocytic Normochromic	Nil
50	Sasikala	29	G3P1L1A1	22.8	24	9	10.8	1.8	5.5	Microcytic Hypochromic	Nil

MASTER CHART

GROUP - A - FERROUS SULPHATE

S.NO	Name	Age	Parity	BMI kg/mg2	Gestational age in weeks	initial Hb in gml%	Final Hb in gml%	Rise in Hb in gml%	Rise in Hct %	Peripheral Smear	Adverse Effect
51	Vidya	30	G3A2	22.8	26	9.2	10.4	1.2	3.5	Normocytic Normochromic	Epigestric Pain
52	Vinodha	31	G4A3	23.3	28	8.4	10	1.6	5.0	Microcytic Hypochromic	Epigestric Pain
53	Vijayalakshmi	30	G2P1L1	22.2	26	9	10.2	1.2	3.5	Normocytic Hypochromic	Nil
54	Arasi	29	G2A1	18.7	24	9.2	11	1.8	5.5	Normocytic Hypochromic	Constipation
55	Booma	27	Primi	26.1	28	8.8	9.9	1.1	3.0	Microcytic Hypochromic	Nil
56	Deepa	28	G3P1L1A1	24.1	28	9	10.8	1.8	5.5	Microcytic Hypochromic	Nil
57	Divya	29	G2A1	18.6	30	9.4	10.5	1.1	3.0	Normocytic Normochromic	Nil
58	Aruna	30	G2P1L1	20	32	8.4	9.8	1.4	4.0	Microcytic Hypochromic	Vomiting
59	Angel	26	G3A2	21.6	30	8.8	10.2	1.4	4.0	Microcytic Hypochromic	Nil
60	Shanthi	27	G2A1	24.7	26	8.6	10	1.4	4.0	Microcytic Hypochromic	Epigestric Pain
61	Divya	28	G3P2L2	22	24	9	10.6	1.6	4.8	Normocytic Hypochromic	Nil
62	Selvi	21	G3A2	21.6	26	8.1	9.8	1.7	5.0	Microcytic Hypochromic	Metallic taste
63	Dharani	18	Primi	24.7	30	9	11	2	6.0	Normocytic Hypochromic	Nil
64	Sujatha	22	G3P1L1A1	22	24	9.1	10.8	1.7	5.0	Normocytic Normochromic	Nil
65	Vijayalakshmi	23	G3A2	21	26	9.1	10.3	1.2	3.5	Normocytic Hypochromic	Nil
66	Nirmala	27	G2P1L1	18.7	26	8.2	9	0.8	2.5	Microcytic Hypochromic	Metallic taste
67	Deepa	28	G2P1L1	18	28	9.3	10.5	1.2	3.5	Normocytic Normochromic	Nil
68	Dhanalakshmi	32	G2P1L1	26.1	28	8.4	9.2	0.8	2.5	Microcytic Hypochromic	Nil
69	Sheela	31	G3A2	24.1	28	9.2	10.8	1.6	5.0	Normocytic Normochromic	Nil
70	Asha	28	G2A1	29.95	26	8.3	9.5	1.2	3.5	Microcytic Hypochromic	Vomiting
71	Eswari	29	G4A3	23	28	9	10.3	1.3	4.0	Normocytic Hypochromic	Nil
72	Sony	18	Primi	20	30	8.8	10	1.2	3.5	Microcytic Hypochromic	Metallic taste
73	Rani	27	G2A1	24.2	28	9.1	10	0.9	2.5	Normocytic Hypochromic	Nil
74	Chella	25	G3A2	18.4	26	8.9	10.3	1.4	4.0	Microcytic Hypochromic	Constipation
75	Nisha	17	Primi	24	30	9	10.5	1.5	4.5	Normocytic Hypochromic	Nil
76	Rajeswari	24	G2P1L1	22.8	28	8.8	10.1	1.3	4.0	Microcytic Hypochromic	Nil
77	Baby	23	G3P1L1A1	22	28	8.7	10	1.3	4.0	Microcytic Hypochromic	Nil
78	Shanthi	19	Primi	24	32	9.2	10.5	1.3	4.0	Normocytic Normochromic	Metallic taste
79	Mala	20	Primi	27.1	30	8.5	9.4	0.9	2.5	Microcytic Hypochromic	Nil
80	Nithya	24	G2A1	26	26	8.6	10	1.4	4.0	Microcytic Hypochromic	Epigestric Pain
81	Jeyalakshmi	26	G3P1L1A1	24.1	28	9.5	10.8	1.3	4.0	Normocytic Normochromic	Nil
82	Gowri	32	G3P2L0	20	28	8.7	10	1.3	4.0	Microcytic Hypochromic	Nil
83	Veni	32	Primi	18	30	8.2	9	0.8	2.5	Microcytic Hypochromic	Nil
84	Neela	26	G3P2L2	22	24	9.4	11.2	1.8	5.5	Normocytic Normochromic	Nil
85	Nalini	30	G2P1L1	24	28	8.4	9.7	1.3	4.0	Microcytic Hypochromic	Constipation
86	Ruckmani	31	G2P1L0	20	26	8.5	9.4	0.9	2.5	Microcytic Hypochromic	Epigestric Pain
87	Eswari	24	G3A2	24.7	30	8.6	10.6	2	6.0	Microcytic Hypochromic	Nil
88	Mahitha	25	G2P1L1	26	26	9	10.4	1.4	4.0	Normocytic Normochromic	Nil
89	Egavalli	29	G2A1	24	26	8.7	10	1.3	4.0	Microcytic Hypochromic	Epigestric Pain
90	Mala	31	G2A1	24.1	28	9	10.8	1.8	5.5	Normocytic Hypochromic	Nil
91	Margret	28	G2P1L1	22	28	8.8	10	1.2	3.5	Microcytic Hypochromic	Nil
92	Roja	26	G3P1L1A1	18.6	24	8.4	9.6	1.2	3.5	Microcytic Hypochromic	Nil
93	Raji	27	G3A2	20	26	8.4	9.7	1.3	4.0	Microcytic Hypochromic	Diarrhea
94	Salomi	28	G3P1L1A1	21.6	30	9.1	10.5	1.3	4.0	Microcytic Hypochromic	Nil
95	Savitha	18	Primi	24.7	32	8	9.4	1.4	4.0	Microcytic Hypochromic	Nil
96	Sharmila	19	Primi	18.6	30	9	10.6	1.6	5.0	Microcytic Hypochromic	Epigestric Pain
97	Sasikala	20	G2A1	24.2	28	8.8	10	1.2	3.5	Microcytic Hypochromic	Nil
98	Tamilselvi	24	G2P1L1	21.35	26	9.2	10.5	1.3	4.0	Microcytic Hypochromic	Nil
99	Tharani	23	G3P1L1A1	18.3	28	8	9.2	1.2	3.5	Microcytic Hypochromic	Epigestric Pain
100	Tamil	25	G3A2	22.4	26	9.3	10.5	1.2	3.5	Microcytic Hypochromic	Nil

MASTER CHART

GROUP - B - CARBONYL IRON

S.NO	Name	Age	Parity	BMI kg/mg2	Gestational age in weeks	initial Hb in gm%	Final Hb in gml%	Rise in Hb in gml%	Rise in Hct %	Peripheral Smear	Adverse Effect
1	Mahalakshmi	21	G2P1L1	18.3	30	9	10.6	1.6	5.0	Normocytic Hypochromic	Nil
2	Poovarasi	24	G2A1	21.35	32	8.5	10.1	1.6	5.0	Normocytic Normochromic	Nil
3	Gowri	25	Primi	24.2	30	9	10.2	1.2.	3.5	Normocytic Hypochromic	Metallic taste
4	Anbu	19	G3P1L1A1	18.6	26	8.4	9.6	1.2	3.5	Normocytic Normochromic	Nil
5	Charumathy	25	G3P2L2	20	28	9.1	10.8	1.7	5.0	Normocytic Normochromic	Nil
6	Kalaiselvi	24	G2P1L1	24.7	24	8.2	9.5	1.3	4.0	Microcytic Hypochromic	Nil
7	Uma	28	G3A2	21.6	32	9.2	11	1.8	5.5	Normocytic Hypochromic	Epigestric Pain
8	Reena	29	G2P1L1	24.7	24	8	9.2	1.2	3.5	Microcytic Hypochromic	Nil
9	Parameswari	31	Primi	22	32	9	10.4	1.4	4.0	Normocytic Hypochromic	Constipation
10	Vanitha	34	G3P1L1A1	21	28	8.6	10.2	1.6	5.0	Microcytic Hypochromic	Nil
11	Jothi	26	G2P1L1	20.1	24	9.2	10.8	1.6	5.0	Normocytic Hypochromic	Nil
12	Sonia	21	G3A2	18.4	28	8.8	10.1	1.3	4.0	Microcytic Hypochromic	Metallic taste
13	Alamelu	29	G3P1L1A1	24	26	9	10.4	1.4	4.0	Normocytic Hypochromic	Nil
14	Sujatha	22	Primi	22.8	32	8.7	10.3	1.6	5.0	Microcytic Hypochromic	Nil
15	Uma	17	Primi	22.9	24	9.2	11.5	2.3	7.0	Microcytic Hypochromic	Epigestric Pain
16	Poovarasi	28	G2P1L0	23.3	28	8.5	10.1	1.6	5.0	Microcytic Hypochromic	Nil
17	Renuka	20	G3P1L1A1	22.2	28	9.3	10.5	1.2	3.5	Normocytic Normochromic	Metallic taste
18	Rajeswari	24	G2A1	18.17	30	8.6	10.2	1.6	5.0	Microcytic Hypochromic	Nil
19	Kuppu	23	G3P2L1	19.3	28	9	10.7	1.7	5.0	Normocytic Hypochromic	Nil
20	Mageswari	20	G2P1L1	26.14	28	8.8	10.5	1.7	5.0	Microcytic Hypochromic	Diarrhea
21	Kasthuri	24	G4A2P1L1	24.14	26	9.2	11	1.8	5.5	Normocytic Hypochromic	Nil
22	Jeniffer	21	Primi	20.95	30	9	10.4	1.4	4.0	Microcytic Hypochromic	Nil
23	Pavithra	22	G2P1L1	24	28	9.1	10.7	1.6	5.0	Microcytic Hypochromic	Epigestric Pain
24	Kanmani	28	G2P1L1	22.5	26	8.4	10.2	1.8	5.5	Microcytic Hypochromic	Nil
25	Vani	27	G3P2L1	22.4	28	9	10.3	1.3	4.0	Microcytic Hypochromic	Nil
26	Alamelu	33	Primi	23	30	8.2	10	1.8	5.5	Microcytic Hypochromic	Nil
27	Vatchala	26	G4P1L1A2	20	26	9	10.7	1.7	5.0	Normocytic Hypochromic	Metallic taste
28	Sarasu	20	G2A1	19.5	28	8.6	10	1.4	4.0	Microcytic Hypochromic	Nil
29	Latha	18	Primi	20.2	32	8.7	10.4	1.7	5.0	Microcytic Hypochromic	Nil
30	Kavitha	21	Primi	23	30	9.1	11.4	2.3	7.0	Microcytic Hypochromic	Nil
31	Raji	21	G3A2	18.3	26	9.2	10.8	1.6	5.0	Normocytic Normochromic	Metallic taste
32	Kamala	19	Primi	21.35	32	8.8	10.5	1.7	5.0	Microcytic Hypochromic	Nil
33	Aruna	24	G3P1L1A1	24.2	28	9	10.6	1.6	5.0	Normocytic Hypochromic	Nil
34	Muniamma	28	Primi	18.6	30	9.1	11.4	2.3	7.0	Microcytic Hypochromic	Nil
35	Amudha	17	Primi	20	30	8.2	9.8	1.6	5.0	Microcytic Hypochromic	Nil
36	Bagya	20	G2P1L1	24.7	32	9	10.2	1.2	3.5	Normocytic Hypochromic	Metallic taste
37	Catherine	22	G3P1L1A1	21.6	26	8.4	9.6	1.2	3.5	Microcytic Hypochromic	Nil
38	Durga	24	G2A1	24.7	28	9.1	10.5	1.4	4.0	Microcytic Hypochromic	Nil
39	Deepa	30	Primi	22	24	8.4	10.1	1.7	5.0	Microcytic Hypochromic	Epigastric pain
40	Geetha	32	G3P2L1	21	26	9.2	11.4	2.2	6.5	Normocytic Normochromic	Nil
41	Hemalatha	26	Primi	21.1	28	8.8	10.4	1.6	5.0	Microcytic Hypochromic	Nil
42	Amsa	27	G2A1	18.4	26	9.4	11.5	2.1	6.5	Microcytic Hypochromic	Nil
43	Indira	29	G3P2L0	24	30	9	10.6	1.6	5.0	Normocytic Hypochromic	Nil
44	Indu	27	G3A2	22.8	32	8.9	10.6	1.7	5.0	Microcytic Hypochromic	Vomiting
45	Jamuna	27	G3P2L2	22.9	24	9.1	10.5	1.4	4.0	Normocytic Hypochromic	Nil
46	Jansi	20	G2P1L1	23.2	26	9.2	10.5	1.3	4.0	Microcytic Hypochromic	Nil
47	Kamala	17	Primi	22.2	28	8.5	10.1	1.6	5.0	Microcytic Hypochromic	Nil
48	Karthika	19	Primi	18.17	32	9.5	11	15	4.5	Normocytic Normochromic	Consitipation
49	Kamatchi	20	G2A1	19.3	26	9.5	11.6	2.1	6.5	Normocytic Normochromic	Nil
50	Leela	22	G4P1L1A2	26.14	26.14	24	8.6	1.6	5.0	Microcytic Hypochromic	Nil

GROUP - B - CARBONYL IRON

S.NO	Name	Age	Parity	BMI kg/mg2	Gestational age in weeks	initial Hb in gm%	Final Hb in gml%	Rise in Hb in gml%	Rise in Hct %	Peripheral Smear	Adverse Effect
51	Latha	23	G2P1L1	24.14	26	9	10.6	1.6	5.0	Normocytic Hypochromic	Nil
52	Meena	26	G3A2	20.95	28	8.5	10.1	1.6	5.0	Normocytic Normochromic	Nil
53	Mala	30	G3P3L2	24	28	9	10.2	1.2	3.5	Normocytic Hypochromic	Metallic taste
54	Noorjakhan	32	G3P2L1	22.5	28	8.4	9.6	1.2	3.5	Normocytic Normochromic	Nil
55	Padma	36	Primi	22.4	30	9.1	10.8	1.7	5.0	Normocytic Normochromic	Nil
56	Palaniammal	18	Primi	23	30	8.2	9.5	1.3	4.0	Microcytic Hypochromic	Nil
57	Pavithra	22	G3A2	20	28	9.2	11	1.8	5.5	Normocytic Hypochromic	Epigestric Pain
58	Reena	24	G2P1L1	19.5	28	8	9.2	1.2	3.5	Microcytic Hypochromic	Nil
59	Rajathi	18	Primi	20.2	30	9	10.4	1.4	4.0	Normocytic Hypochromic	Constipation
60	Rama	19	G2P1L1	23	30	8.6	10.2	1.6	5.0	Microcytic Hypochromic	Nil
61	Kaveri	21	G2P1L1	23	26	9.2	10.8	1.6	5.0	Normocytic Hypochromic	Nil
62	Kasthuri	24	G3P1L1	20.2	28	8.8	10.1	1.3	4.0	Microcytic Hypochromic	Metallic taste
63	Vidhya	25	G4P1L1A2	19.5	28	9	10.4	1.4	4.0	Normocytic Hypochromic	Nil
64	Latha	19	G3A2	20	30	8.7	10.3	1.6	5.0	Microcytic Hypochromic	Nil
65	Veena	25	G2P1L1	23	32	9.2	11.5	2.3	7.0	Microcytic Hypochromic	Epigestric Pain
66	Vijayalakshmi	24	G3A2	22.4	26	8.5	10.1	1.6	5.0	Microcytic Hypochromic	Nil
67	Sarasu	20	G3P2L1A1	22.5	28	9.3	10.5	1.2	3.5	Normocytic Normochromic	Metallic taste
68	Uma	22	Primi	24	32	8.6	10.2	1.6	5.0	Microcytic Hypochromic	Nil
69	Parvathy	19	G2P1L1	18.3	32	9	10.7	1.7	5.0	Normocytic Hypochromic	Nil
70	Mumtaj	17	Primi	21.35	32	8.8	10.5	1.7	5.0	Microcytic Hypochromic	Diarrhea
71	Kanmani	22	G3P1L1A1	24.2	24	9.2	11	1.8	5.5	Normocytic Hypochromic	Nil
72	Aruna	32	G3A2	18.6	26	9	10.4	1.4	4.0	Microcytic Hypochromic	Nil
73	Jayalaskhmi	34	G3P1L1A1	20	26	9.1	10.7	1.6	5.0	Microcytic Hypochromic	Epigestric Pain
74	Kanchana	20	G3P1L1	24.7	28	8.4	10.2	1.8	5.5	Microcytic Hypochromic	Nil
75	Monica	26	G3A2	21.6	30	9	10.3	1.3	4.0	Microcytic Hypochromic	Nil
76	Mahalaskhmi	27	G3P1L1A1	24.7	24	8.2	10	1.8	5.5	Microcytic Hypochromic	Nil
77	Manjula	33	Primi	2	28	9	10.7	1.7	5.0	Normocytic Hypochromic	Metallic taste
78	Karthika	31	G4P1L0A2	21	28	8.6	10	1.4	4.0	Microcytic Hypochromic	Nil
79	Malar	27	G3P2L2	20.1	24	8.7	10.4	1.7	5.0	Microcytic Hypochromic	Nil
80	Sundari	20	Primi	18.4	30	9.1	11.4	2.3	7.0	Microcytic Hypochromic	Nil
81	Rekha	20	Primi	24	32	9.2	10.8	1.6	5.0	Normocytic Normochromic	Metallic taste
82	Banumathy	23	G4P1L1A2	22.8	24	8.8	10.5	1.7	5.0	Microcytic Hypochromic	Nil
83	Anandhi	25	G3A2	22.9	28	9	10.6	1.6	5.0	Normocytic Hypochromic	Nil
84	Anjalai	27	G3P1L1A1	23.3	30	9.1	11.4	2.3	7.0	Microcytic Hypochromic	Nil
85	Aarthi	30	G4P3L3	22.5	24	8.2	9.8	1.6	5.0	Microcytic Hypochromic	Nil
86	Deepa	18	Primi	24	28	9	10.2	1.2	3.5	Normocytic Hypochromic	Metallic taste
87	Fathima	20	G3A2	20.95	30	8.4	9.6	1.2	3.5	Microcytic Hypochromic	Nil
88	Gayathiri	20	G2P1L1	24.14	30	9.1	10.5	1.4	4.0	Microcytic Hypochromic	Nil
89	Kanmani	19	Primi	26.14	32	8.4	10.1	1.7	5.0	Microcytic Hypochromic	Epigastric pain
90	Sheela	18	Primi	19.3	30	9.2	11.4	2.2	6.5	Normocytic Normochromic	Nil
91	Mahalaskhmi	21	G2A1L1	18.3	30	8.8	10.4	1.6	5.0	Microcytic Hypochromic	Nil
92	Poovarasi	24	G2A1	21.35	32	9.4	11.5	2.1	6.5	Microcytic Hypochromic	Nil
93	Gowri	25	Primi	24.2	30	9	10.6	1.6	5.0	Normocytic Hypochromic	Nil
94	Anbu	19	G3P1L1A1	18.6	26	8.9	10.6	1.7	5.0	Microcytic Hypochromic	Vomiting
95	Charumathy	25	G3P2L2	20	28	9.1	10.5	1.4	4.0	Normocytic Hypochromic	Nil
96	Kalaiselvi	24	G2P1L1	24.7	24	9.2	10.5	1.3	4.0	Microcytic Hypochromic	Nil
97	Uma	28	G3A2	21.6	32	8.5	10.1	1.6	5.0	Microcytic Hypochromic	Nil
98	Reena	29	G2P1L1	24.7	24	9.5	11	1.5	4.5	Normocytic Normochromic	Consitipation
99	Parameswari	31	Primi	22	32	9.5	11.6	2.1	6.5	Normocytic Normochromic	Nil
100	Vanitha	34	G3P1L1A1	21	28	8.6	10.3	1.6	5.0	Microcytic Hypochromic	Nil

PROFORMA

PATIENT NAME : AGE:

ADDRESS : OBSTETRIC STATUS

LMP

EDD

DISSERTATION REF NO:

SOCIOECONOMIC STATUS

HT WT BMI GA

TYPE OF IRON DATE OF STARTING TREATMENT

PAST H/O

H/O ANY DISEASES IN THE PAST

IF YES, SPECIFY THE DISEASE

CLINICAL EXAMINATION USG

INVESTIGATION

DAY – 0	DAY – 30
Hb gm%	Hb gm%
Hct %	Hct %
Peripheral Smear	
MCV	MCV
MCHC	MCHC
BT / CT	
Motion - Ova / Cyst	

SIDE EFFECTS NOTED ON 15th DAY

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