COMPARATIVE STUDY OF EFFICACY OF FERROUS SULPHATE AND CARBONYL IRON IN ANEMIA OF ANTENATAL WOMEN

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HOW TO CITE THIS ARTICLE:

ABSTRACT: Iron deficiency anemia is the most common and important public health problem all over the world in the risk group of antenatal women. Research is going on to improve the iron status of the pregnant women with different forms of iron available. In this regard, Carbonyl Iron is showing promising results in improving the red cell mass with better compliance. 120 antenatal women were recruited in this study. The study comprised of 6 weeks for each patient. They were given Carbonyl Iron 100 mg/day and FeS04 100gm/day. Before and after treatment all the baseline and specific investigations were done. Results were tabulated, comparison and significance were tested by unpaired student’s test and their ‘p’ value was calculated. Results were shown graphically also. Carbonyl Iron showed improvement in hemoglobin, PCV and better than ferrous Sulphate (P <0.001). Incidence of side effects were less with Carbonyl Iron than Ferrous Sulphate, better compliance was seen with Carbonyl Iron. In conclusion, the present study showed that Carbonyl Iron had better efficacy and safety in the management of Iron deficiency anemia in antenatal women than ferrous Sulphate.

KEYWORDS: Anemia, Ferrous sulphate, Carbonyl Iron.

INTRODUCTION: The global prevalence of anemia in 1980 was over 30% out of a population of 4.4 billion, 1.3 billion were anemic, out of which 1.2 billion lived in developing countries. The incidence of anemia ranges from 40 to 80% amongst pregnant women in India and our neighboring countries. The estimated prevalence in middle-income group is 20 to 30%. While it is almost 60-70% in pregnant women of lower socioeconomic groups. The prevalence is more in rural women than in urban women. In urban slums, where hookworm infestation and malaria are endemic, the incidence is as high as 90%, 13 million out of 22 million pregnant women in India are anemic and 25% of them suffer from moderate to severe anemia. It is these women who are responsible for the high maternal and prenatal morbidity and mortality. In India, which is a developing country, where population explosion is a major problem, the slogan of one or two children is very apt and therefore it is important to ensure that every fetus conceived is carried to viability and brought into this world under optimal conditions.

Anemia in pregnancy continues to play a major role in being responsible for maternal and fetal morbidity and mortality. The term 'Anemia in Pregnancy' refers to all types of anemia encountered during pregnancy. This term includes anemia’s occurring independently of pregnancy and also anemia’s precipitated or caused by pregnancy.[1]

Iron deficiency is the commonest cause of anemia all over the world. It is estimated that about 20% of women in child-bearing age group are iron deficient. Pregnancy causes a state of hydramic plethora, in other words, the total volume of blood is increased partly by dilution and
hemoglobin is consequently reduced to a varying extent, occasionally as low as 80%. Levels below this are pathological, and one should aim at raising the hemoglobin to 80% or more, if possible before delivery. The commonest source of trouble in anemic pregnancy is inadequate absorption of iron. The normal daily requirement for the gravid woman is about 20 mg of iron, even in cases in which the iron stores have not suffered depletion prior to pregnancy, the baby, especially in the later months of pregnancy, makes heavy demands upon maternal iron and average fetal requirements amount to about 375 mg. Unfortunately, the margin between the patients requirements and the quantity of iron normally available in a reasonably good diet is a very narrow one; in fact, the average diet seldom contains more than about 15 mg a day. Of the total amount of iron in food, only a fraction (about 10%) is available for absorption. So, Iron therapy is the sheet anchor in these women. Keeping the above view in mind, a prospective and comparative clinical study was carried out in antenatal women with moderate anemia i.e <8 g/ml. Two groups of each 50 antenatal women were given ferrous sulphate and Carbonyl Iron the response is seen after six weeks. This study carried out at the department of Obstetrics and Gynecology, Govt. General Hospital, Kurnool.

AIMS AND OBJECTIVES:

- To study and compare the efficacy and safety of Ferrous Sulphate and Carbonyl Iron in anemia of antenatal Women.
- To study the improvement of anemic status ill anemia of antenatal women.
- To study the compliance of drugs.

Anemia is defined as a hematological disorder characterized by a hemoglobin concentration of less than 11 gram% (W.H.O), (In developing countries: 10 gms%), resulting in reducing oxygen carrying capacity.

- A reduction in normal limits of the total circulating red cell mass.
- Anemia call be defined as a reduction below normal limits in the amount of hemoglobin (Hb) A or in the
- Volume of RBC’s (hematocrit) in a sample of peripheral venous blood.
- A decrease in the oxygen carrying capacity of the blood is termed as 'anaemia.'
- Anemia is defined as a condition of lower range of hemoglobin for the age and sex of the individual.

CLASSIFICATION OF ANEMIA: Several types of classifications of anemia have been proposed. Three of the widely accepted classifications are based on the pathophysiology, morphology and etiology.

ETIOLOGIC CLASSIFICATION: Anemias are classified according to their etiology as follows:

1. Anemias due to dietary deficiency or malabsorption of factors essential for normal blood formation, e.g. iron, folic acid,
2. Vitamin B12, vitamin C, and pyridoxine.
3. Anemias due to blood loss such as menorrhagia, GI loss and hook worm infestation.
4. Anemias due to excessive blood destruction, e.g. Thalassemia, sickle cell anemia and autoimmune hemolytic Anemia.

5. Anemias due to aplasia or hypoplasia of the bone marrow, such as idiopathic or following certain drugs such as anticancer drugs and chloramphenicol.

6. Anemia due to deficiencies of erythropoietin as in chronic renal diseases.

7. Anemias of uncertain origin, e.g. due to infection, rheumatoid arthritis, liver disease and widespread Malignant disease.

**PATHOPHYSIOLOGIC CLASSIFICATION**[^3]: Anemias are classified depending upon the pathophysiologic mechanism into following groups.

**I. Anemia due to blood loss:**
- a. Acute post-hemorrhagic anemia.
- b. Anemia of chronic blood loss.

**II. Anemia due to impaired red cell formation.** A disturbance due to impaired red cell production from various causes may produce anemia. These are as follows.
- a. Cytoplasmic maturation defects.
  - 1. **Deficient heme synthesis:** iron deficiency anemia.
  - 2. **Deficient globin synthesis:** thalassemia syndromes.
- b. Nuclear maturation defects: Vitamin BI2 and folic acid deficiency: megaloblastic anemia.
- c. Hematopoietic stem cell proliferation and differentiation abnormality e.g. aplastic anemia and pure red cell.

**Aplasia:**
- d. Bone marrow failure due to systemic diseases (anemia of chronic disorders) e.g.
  - 1. Anemia of infections.
  - 3. Anemia in liver disease.
  - 4. Disseminated malignancy.
  - 5. Endocrinopathies.
- e. Bone marrow Infiltration e.g.
  - 1. Leukemia’s.
  - 2. Lymphomas.
  - 3. Myelosclerosis.
  - 4. Multiple myeloma.

- f. Congenital anemia e.g.
  - 1. Sideroblastic anemia.
  - 2. Congenital dyserythropoietic anemia.
III. Anemia due to increased red cell destruction (hemolytic anemias): This is further divided into two groups.
   1. Intra corpuscular defect (hereditary and acquired).
   2. Extra corpuscular defect (acquired hemolytic anemias).

MORPHOLOGIC CLASSIFICATION[3]: Anemias are also classified based on the red cells size, hemoglobin content and red cell indices:
1. Microcytic, hypochromic: Mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration are all reduced in iron deficiency anemia and in certain non-iron deficiency anemias (sideroblastic anemia, thalassemia, anemia of chronic disorders).
2. Normocytic, normochromic: Mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration are all normal e.g. after acute blood loss, hemolytic anemias, bone marrow failure, anemia of chronic disorders.
3. Macrocytic: Mean corpuscular volume is raised e.g. in megaloblastic anemia due to deficiency of Vitamin Bl2 or folic acid.

GRADING OF ANEMIA[8]: Anemias are arbitrarily graded according to the level of hemoglobin estimation in the blood as follow.
   Mild anemia 8 – 10 gm%.
   Moderate anemia 6.5 – 8 gm%.
   Severe anemia <6.5 gm%.

Anemia is defined as decreased oxygen carrying capacity of the blood. Oxygen is carried through red blood cells. Red blood cells are produced in the bone marrow. The erythroid series is - pro norm oblast, basophilic (early) norm oblast, polychromatotic (intermediate) norm oblast, orthochromatic (late) norm oblast, reticulocyte and finally the mature red blood cell (RBC) is formed. This process of production of a red blood cell in marrow is called as Erythropoiesis. The normal life span of RBC is 120 days.[6]

New red cells are being produced each day for which the marrow requires certain essential substances.

These substances are as under:
1. Metals: Iron is essential for red cells production because it forms pal of the heme molecule in hemoglobin. Its deficiency leads to iron deficiency.
   Anemia- cobalt and manganese are certain other metals required for red cell production.
2. Vitamins: Vitamin B 12 and folate are essential for biosynthesis of nucleic acids. Vitamin C plays an indirect role in facilitating the iron turnover in the body. Vit. B6, vit. E, Riboflavin are the other essential vitamins required in the synthesis is of red cell.
3. Amino acids: Amino acids comprise the globin component of hemoglobin. Severe amino acid deficiency due to protein deprivation causes depressed red cell production.
4. **Hormones:** Erythropoietin plays a significant regulatory role in the erythropoietin activity besides erythropoietin; androgens and thyroxine also appear to be involved in the red cell production.

The mature erythrocytes of the human peripheral blood are non-nucleated cells and lack the usual cell organelles. It is a biconcave disc, 7.2µm at the periphery and 1µm in the center. The biconcave shape renders the red cells quite flexible so that they can pass through capillaries whose minimum diameter is 3.5µm. More than 90% of the weight of erythrocytes consists of hemoglobin. Hemoglobin consists of a basic protein, globin and iron-porphyrin complex, haem. The molecular weight of hemoglobin is 68000. Most of the hemoglobin (65%) is synthesized by the nucleated red cell precursors in the marrow, while the remainder (35%) is synthesized at the reticulocyte stage. Synthesis of haem occurs largely in the mitochondria by a series of biochemical reactions.

Coenzyme, pyridoxal - 6 - phosphate, derived from pyridoxine (vit B6) is essential for the synthesis of amino levulinic acid (ALA) which is the first step in the biosynthesis of protoporphyrin. The reaction is stimulated by Erythropoietin and inhibited by haem. Ultimately, protoporphyrin combines with iron supplied from circulating transferrin to form haem.[9]

Each molecule of haem combines with a globin chain synthesized by polyribosomes. A tetramer of 4 globin chains, each having its own haem group, constitutes the hemoglobin molecule. Thus, Iron is an essential Constituent of hemoglobin and its deficiency leads to iron deficiency anemia.

**ETIOLOGY OF IRON DEFICIENCY ANAEMIA:** Iron deficiency is a leading cause of anemia, affecting over one half billion people worldwide. Anemia in pregnancy, in India, ranges from 40-80%. In our country, Iron deficiency anemia is prevalent both in rural and Urban population, however, severity of anemia is more in lower-socioeconomic and high risk groups of children, pregnant women and elderly. The commonest causes of iron deficiency anemia are.[10]

1. **Dietary deficiency:**

2. **Malabsorption:**
   - Gluten induced Enteropathy.
   - Atrophic Gastritis. Hcl secretion reduced.
   - Following gastrectomy: Hcl secretion reduced.

3. **Increased blood loss:**
   A. **GI Causes:**
      - Peptic Ulcer.
      - NSAID Intake.
      - Hiatus Hernia.
      - Hook worm infestation.
      - Malignancies of stomach, colon.
• Haemorrhoids.
• Ulcerative colitis.
• Amoebic colitis.
• Diverticulosis.

B. Uterine causes:
• Menorrhagia.
• Repeated pregnancies.
• Increased requirements.
• Excessive blood loss.

C. Urinary tract causes:
• Hematuria due to renal, bladder, prostate lesions.
• Chronic dialysis.
• Paroxysmal Nocturnal Haemoglobinuria.

4. Increased physiological demands:
• In infancy.
• Adolescence.
• Pregnancy and lactation.

In pregnancy: The factors concerned III the production of Iron deficiency during Pregnancies is
• Pre pregnant anemic state of the patient.
• The intake of iron in the food during pregnancy.
• Absorption and utilization of iron.
• The iron demands of the fetus.

The iron requirements of pregnancy are not confined to those of the fetus alone. Additional iron is also required for the placenta, the enlarged uterus and for the increased blood volume that occurs in pregnancy\[11\] during puerperium, the iron stores may further suffer from blood loss in parturition. So, anemia incidence is more in multipara.

The growing fetus for its optimum nutrition and sustenance can draw the necessary essential factors only from the mother. Increasing metabolic demands and fetal needs may thus deplete the mother unless her haemopoietic reserve has been sufficiently strong or unless her dwindling stores are reinforced by supplementary quota. During normal pregnancy, a woman, loses approximately 550 mg of iron, 350-400 mg is taken up by the.

Fetus and about 150 mg goes towards the formation of placenta. Cessation of menstruation during pregnancy leads to conservation of about 300 mg, but again 100-200 mg may be lost in postpartum hemorrhage. Approximately 150 mg is also lost in milk during lactation. Conservation of iron is also seen during menopause. Daily requirement of iron in non-pregnant normal woman is 2.1 mg, increases to 3.8 mg during pregnancy and 2.8 mg during lactation.
These demands cannot be adequately met during pregnancy in most of the women due to the following reasons.

- Capricious appetite, nausea, vomiting - which are common in early pregnancy may further interfere with adequate intake of dietary protein, iron, folic acid, vitamin B12 and vitamin C.
- Hypo secretion of gastric-HCl and pepsin may sometimes occur temporarily during pregnancy. Occasionally this interferes with absorption and utilization of haemopoietic factors.
- Preexisting diarrheal and dysenteric conditions can flare up during pregnancy and cause serious interference with the intake and absorption of essential factors.
- Coincident infections inhibit hemoglobin synthesis.
- An inhibitory endocrinal influence has also been incriminated.\[1\]

**PATHOPHYSIOLOGY:** Despite the fact that iron is the second most abundant metal in the earth’s crust, iron deficiency is the world’s most common cause of anaemia. When it comes to life, iron is more, precious than gold.

Daily requirement of Iron.\[12\]
- Adult male 0.5 -1 mg (13, ug / kg).
- Adult female 1-2 mg (21, ug / kg).
- Infants 60, ug / kg.
- Children 25, ug / kg.
- Pregnancy 3 -5mg (80, ug / kg) and lactation.

**EFFECTS OF IRON DEFICIENCY ANAEMIA ON PREGNANCY:** According to WHO, anaemia has been implicated as contributed in up to 40 percent of maternal deaths in third world countries. In India, anaemia is the second most common cause of maternal deaths accounting for 20 percent of total maternal deaths.\[10\]

Anaemia is associated with adverse maternal outcome such as puerperal sepsis, antepartum haemorrhage, postpartum haemorrhage, and maternal mortality. It is also responsible for increased incidence of premature births, low birth weight babies and high perinatal mortality.\[11,12\] Liberman and collaborators (1987) found a positive association with low haematocrit and preterm birth in black women and suggested that anaemia was a marker of nutritional deficiencies. Anaemia may also be associated with foetal growth restriction.

**MATERIALS AND METHODS:** The study was comparative and randomized. Performed on 120 antenatal women in the outpatient department of obstetrics and Gynecology, Government General Hospital, Kurnool. Before starting this study, approval of ethical committee was obtained. Informed consent was obtained from each antenatal woman.

**INCLUSION CRITERIA:**
- Moderate anemia with Hemoglobin <8 g%.
- Antenatal women of gestational age <20 wks.
EXCLUSION CRITERIA:
- Medical and surgical causes of anemia.
- Medical and surgical diseases complicating pregnancy.

All antenatal women underwent routine antenatal checkups. Baseline investigations like urine for albumin, sugar and deposits, stool examination for ova and cysts, hemoglobin estimation, PCV (hematocrit), and ultrasonography were done before starting the study.

METHODS:
1. **Hemoglobin estimation by Sahli’s method:** Hemoglobin is converted into acid hematin.
   The brown color of the compound is compared against a brown glass standard in a comparator.

   **Procedure:** Venous blood (oxalated) is taken. Haemoglobinometer is filled up to mark 2 with 0.1 N Hcl by means of a dropper. Pipette is filled with blood exactly up to the 20 micro litres mark by gentle controlled suckling. This is blown into acid tube. Acid hematin will be formed after allowing it for 5 minutes. This solution IS diluted by adding distilled water, drop by drop, stirring the mixture all the while, with the glass rod, comparator is held against good day light and the addition of water continued till the colour of the solution matches, perfectly with that of standard. Bottom of the meniscus is read off in grams /dl.

   **Normal Range:**
   - Male 14-16 g /dl.
   - Female 12-14 g /dl.

2. **Hematocrit or packed cell volume (PCV):** Procedure: Wintrobes tube is filled with blood by means of a pasteur pipettee up to mark 10. This is done by passing the pipette to the bottom of the tube and raising it gradually so as to avoid air bubbles. It is then centrifuged at 3000 rpm for 30 minutes.

   **OBSERVATION:**
   1. After 30 minutes, packed red blood cells at the bottom of the tube reading10 times is PCV.
   2. Above the RBC is the buffy coat of white blood cells approximately.
   3. Pinkish white layer above the buffy coat represents layer of platelets.

   **Normal ranges of PCV Male 43 -54 No’s.**
   - Female 37-42 No’s.

   Among 120 Antenatal women, 20 Women discontinued treatment (due to noncompliance). Out of these 20 women 16 belong to Feso₄ group and 4 to Carbonyl Iron Group. In the remaining 100 women, 50 were given Ferrous Sulphate 100 mg per day and 50 women in the other group were given Carbonyl Iron 100 mg per day orally for a period of six weeks. These women were followed up for every two weeks and the routine clinical antenatal checkups were done and,
Improvements in the clinical symptoms were noted. After six weeks all the investigations were repeated for all the women. Results were compared, tabulated and statistical significance was tested by unpaired 't' test and their 'p' value was calculated. Results were represented graphically also.

RESULTS: Both regimens improved the anemic status at comparable rates but the compliance was good with Carbonyl Iron because of less side effects. Among 100 antenatal women, 88 were under low socio economic group and 12 were from middle income group. So, incidence of anemia is more in low socioeconomic group reflecting the poor nutritional status of that group. Among 100 antenatal women, 41 were primi gravida and 59 were second gravida and beyond. Incidence of anemia was more in the latter group.

The mean weight of the women in the FeS04 group before treatment (0 weeks) was 47.2 (±5.23) and after treatment.

(6 weeks) it was 48.52(±4.78). In the Carbonyl Iron before treatment (0 weeks) it was 48.08(±5.34) and after treatment (6 weeks) it was 49.72 (±5.12).

In FeS04 group before treatment (0 weeks) mean Hemoglobin concentration was 7.268 (±0.428) gm/dl and after treatment for 6 weeks it raised to 8.3 18 (±0.392) gm/dl. In Carbonyl Iron before treatment (0 weeks) mean hemoglobin concentration was 7.25 (±0.46) gm/dl and after treatment for 6 weeks it raised to 8.598 (±0.369) gm/dl. When compared between the two groups there was a significant improvement in Carbonyl Iron group (P <0.001).

Before treatment (0 weeks) the mean PCV in the FeS04 group was 29.1 (±2.125) and in the Carbonyl Iron group it was 30.04 (±1.948). After treatment (6 weeks) the mean PCV improved in the first group to 32.5 (±2.061) and in the latter group it was 34.24 (±1.656). While in the inter group comparison there was a significant improvement in the Carbonyl Iron group with P <0.001. In the Carbonyl Iron group there was a better patient compliance and tolerance when compared with FeS04. Side Effects: 9 antenatal women in the ferrous sulphate group complained of Diarrhea, 9 of epigastric distress, 7 of nausea, 2 of vomiting and 2 of constipation.

In Carbonyl Iron group 4 complained of nausea, 1 of epigastric distress, 2 of constipation, 1 of diarrhea.

DISCUSSION: The results in the present study suggest that Carbonyl Iron was more effective than FeS04 with better tolerance than the latter and hemoglobin and hematocrit improvement was significant (P <0.001). These results were consistent with those of Jacobs P, Wood L, Bird A.R. in their study "Better tolerance of Carbonyl Iron compared with Ferrous sulphate in the treatment of anemia" (hematology. 2000; 5 (I): 77_83).[13] In Carbonyl Iron group the improvement in hemoglobin was significant (P <0.001). This was similar to the studies done by V.R. Badhwar and et. al- "Carbonyl Iron - An effective Hematinic" (IIIM, April 2004; 102 (4) 225).[14]

Comparing the mean PCV's after six week treatment with Carbonyl Iron recorded higher and statistically significant improvement (P <0.00 I) than Fe S04. This is similar with the studies of Komalafe J. O, Kuti O., Ijadunola KT, Ogynmiyo 'A Comparative study between Carbonyl Iron

Clinical parameters like pallor, weakness, as well as biochemical parameters (Hb, PCV,) showed favorable changes with Carbonyl Iron. These results were similar to those of Reddy PS, Abdul BB, Gandewark, Korde KM, Desai A in.

"Evaluation of efficacy and safety of Carbonyl Iron Vs Ferrous Sulphate in female patients with anemia- JIMA, 2001 March; 99(3):154-526.In our study side effects were noted more in Ferrous Sulphate group than Carbonyl Iron group.

SUMMARY AND CONCLUSION: Iron deficiency anemia is the most common and important public health problem all over the world in the risk group of antenatal women. Research is going on to improve the iron status of the pregnant women with different forms of iron available. In this regard, Carbonyl Iron is showing promising results in improving the red cell mass with better compliance.120 antenatal women were recruited in this study. The study comprised of 6weeks for each patient. They were given Carbonyl Iron 100 mg/day and FeS04 100gm/day. Before and after treatment all the baseline and specific investigations were one. Results were tabulated, comparison and significance were tested by unpaired student 't' test and their 'p' value was calculated. Results were shown graphically also.

Carbonyl Iron showed improvement in hemoglobin, PCV and better than ferrous Sulphate (P < 0.001).

Incidence of side effects were less with Carbonyl Iron than Ferrous Sulphate, better compliance was seen with Carbonyl Iron. In conclusion, the present study showed that Carbonyl Iron had better efficacy and safety in the management of Iron deficiency anemia in antenatal women than ferrous Sulphate.

REFERENCES:
5. The care of the Medical patient by Fred F Furi, Chapter 7, page No. 364.


1. Details of study distribution of antenatal woman as per age in study group.

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<th>Sl. No.</th>
<th>Age in Years</th>
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<td>25-29</td>
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Graph 1
2. Distribution of antenatal women as per gravidity in the study group.

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<td>2.</td>
<td>Second</td>
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3. Distribution of antenatal women as per socioeconomic status.

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4. Distribution of side effects in two groups.

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<tr>
<td>2</td>
<td>Vomiting</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>Diarrhoea</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>Constipation</td>
<td>2</td>
<td>2</td>
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<tr>
<td>5</td>
<td>Epigastric Distress</td>
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Graph 4

5. Ferrous sulphate haemoglobin estimation carbonyl iron.

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<td>8.318</td>
<td>7.25</td>
<td>8.598</td>
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<tr>
<td>SEM</td>
<td>±0.428</td>
<td>±0.392</td>
<td>±0.46</td>
<td>±0.369</td>
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Graph 5
6. Ferrous sulphate packed cell volume carbonyl iron.

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<td>SEM</td>
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<td>± 2.061</td>
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<table>
<thead>
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<th></th>
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<tr>
<td>Mean</td>
<td>30.04</td>
<td>34.24</td>
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<tr>
<td>SEM</td>
<td>± 1.948</td>
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**Graph 6**

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