A Dissertation on

COMPARATIVE STUDY OF INTRAVENOUS FERRIC CARBOXYMALTOSE AND IRON SUCROSE IN THE MANAGEMENT OF POSTNATAL IRON DEFICIENCY ANAEMIA

Submitted to

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STANLEY MEDICAL COLLEGE
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CERTIFICATE

This is to certify that the dissertation titled

"COMPARATIVE STUDY OF INTRAVENOUS FERRIC CARBOXYMALTOSE AND IRON SUCROSE IN THE MANAGEMENT OF POSTNATAL IRON DEFICIENCY ANAEMIA" submitted by **Dr. V. ARULMOZHI** to the faculty of Obstetrics and Gynaecology, Stanley Medical College, The TamilNadu Dr. M.G.R Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S Degree (Obstetrics and Gynaecology), is a bonafied research work carried out by her under our direct supervision and guidance.

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DECLARATION

I **Dr. V. ARULMOZHI**, solemnly declare that the dissertation titled **"COMPARATIVE STUDY OF INTRAVENOUS FERRIC CARBOXYMALTOSE AND IRON SUCROSE IN THE MANAGEMENT OF POSTNATAL IRON DEFICIENCY ANAEMIA"** is a bonafied work done by me at Govt. RSRM Lying in Hospital, Stanley Medical College, Chennai, under the guidance and supervision of **Prof. Dr. C.SUMATHY M.D.,D.G.O.**

This dissertation is submitted to The TamilNadu Dr. M.G.R Medical University in partial fulfilment of university rules and regulations for the award of M.S. Degree examination (Branch –II) in Obstetrics and Gynaecology.

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Date:

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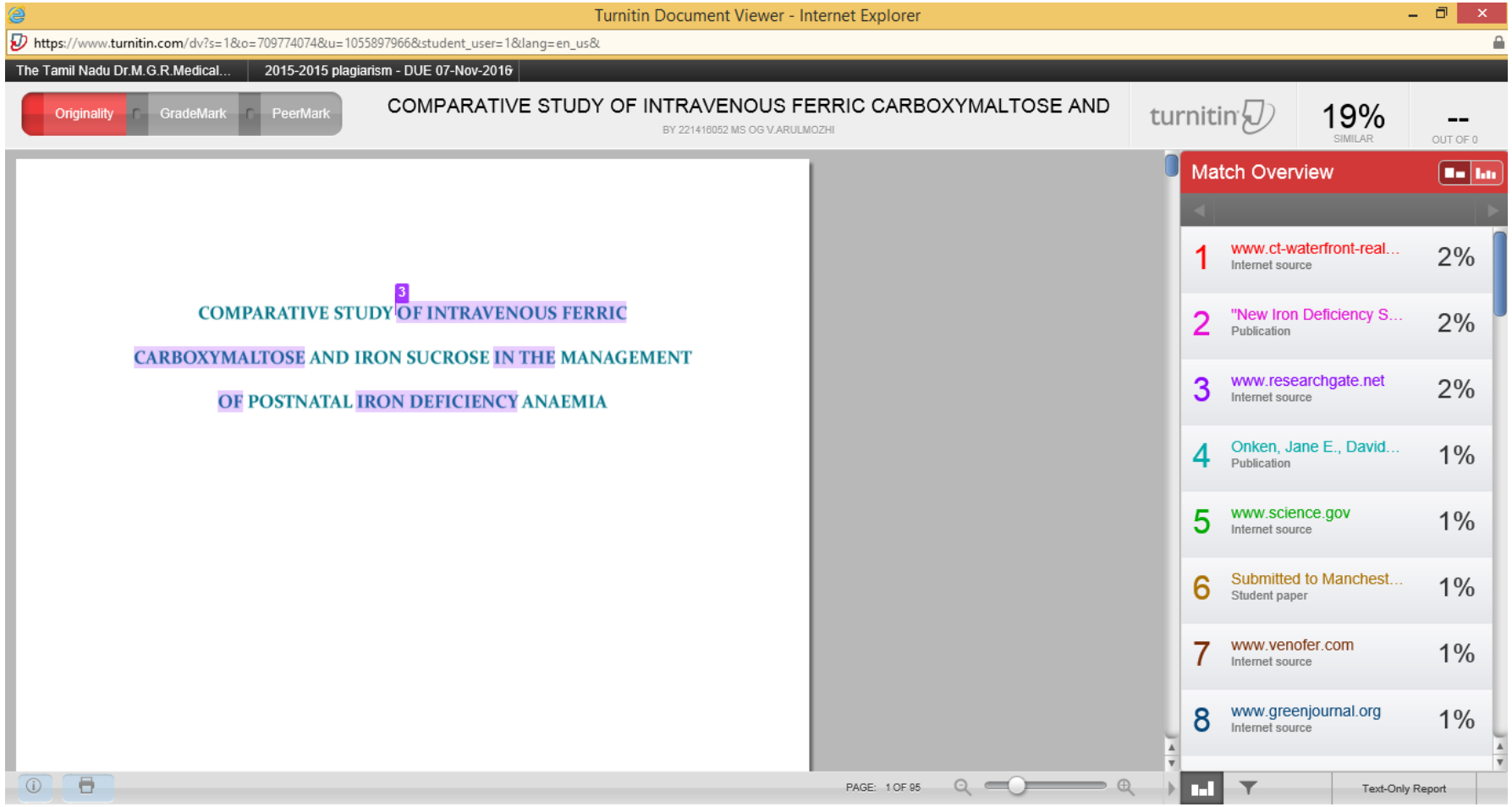
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INTRODUCTION

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Anemia is defined as decreased oxygen carrying capacity of blood. It is one of the major illness affecting more than 50% of antenatal and postnatal women in developing countries like India leading to increased maternal mortality and morbidity. Most common type is the nutritional anemia – IRON DEFICIENCY ANAEMIA. Menstruation and pregnancy put women at a higher risk for anemia.

Iron deficiency anemia in pregnant mother is mainly due to increased demand or due to poor absorption. This unmet need during antenatal period may lead to anemia in postnatal mother. Postnatal anemia may also result from acute blood loss during delivery and also even due to multiple birth without adequate spacing.

WHO defines postnatal anemia as hemoglobin less than 11gm. This anemia in postnatal mother may lead to several complications like depression, subinvolution of uterus, puerperal infection, sepsis, decreased milk production and its quality, lactation failure, deep vein thrombosis, poor wound healing and neurocognitive dysfunction in

infants. So this postnatal anemia should be diagnosed early and should be treated properly to avoid these complications.

Postnatal iron deficiency anaemia can be diagnosed clinically and confirmed by laboratory parameters like hemoglobin, peripheral smear, blood indices and serum ferritin.

Various modes of treatment are available to treat postnatal iron deficiency anaemia. They are

- Oral iron
- Parenteral iron
- Blood transfusion

Due to poor compliance to oral iron and its gastrointestinal side effects and because of inherent risks following blood transfusion, parenteral iron has gain more importance to treat iron deficiency anaemia in clinical practice. Among them second generation intravenous iron sucrose is most commonly used. Upcoming is the third generation injection ferric carboxymaltose.

AIM OF THE STUDY

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This study was conducted to compare the efficacy of ferric carboxymaltose and iron sucrose in the treatment of postnatal iron deficiency anaemia.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

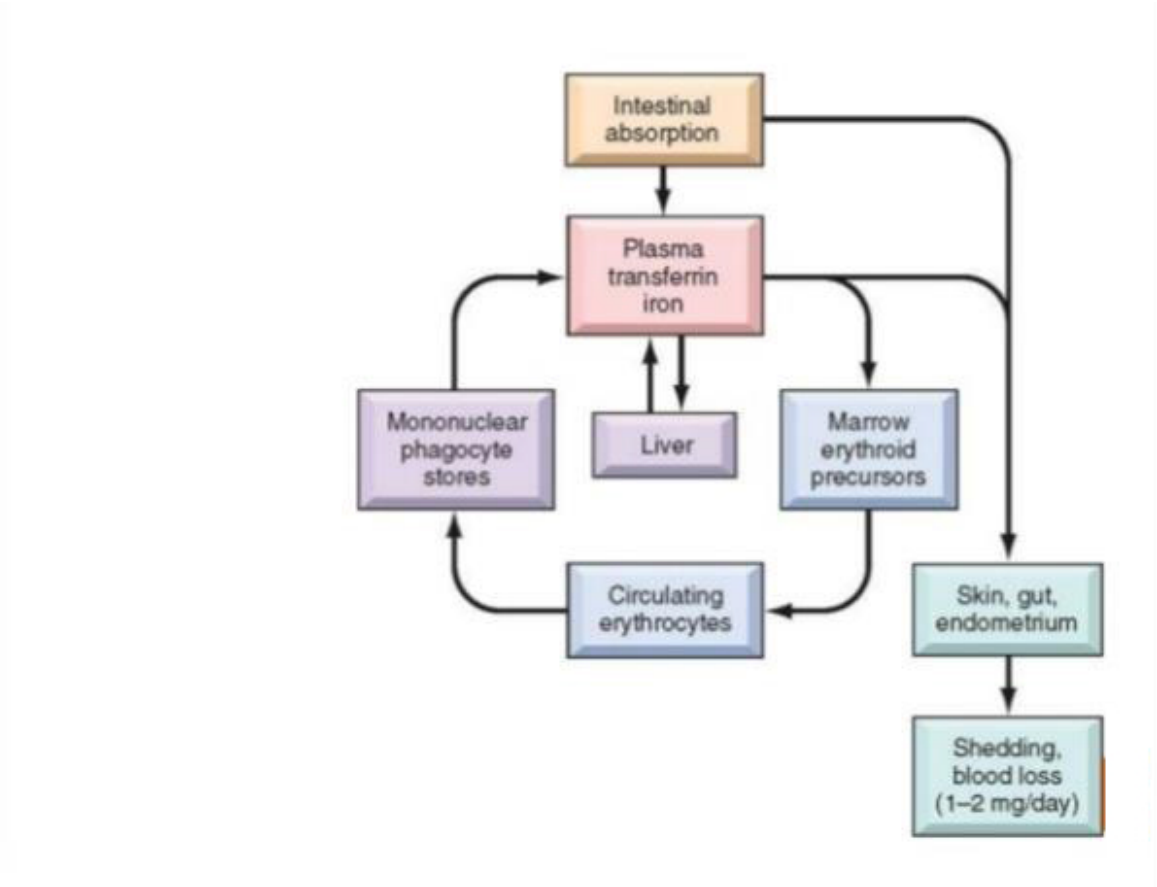
IRON METABOLISM:

It is a unique process where its homeostasis is maintained by absorption and not by excretion. Iron is a one way element. When iron stores are depleted absorption is increased and when adequate quantity of iron is stored absorption is decreased. This is referred to as mucosal block of regulation of absorption of iron. Iron is not actively excreted from the body, it is eliminated only through epithelial cells from the gastrointestinal tract, epidermal cells of skin, and in mensturating women red blood cells. The total average body loss of iron is 1-2mg in normal adult men and non mensturating women. Although iron is a physiological component of sweat only a tiny amount of iron (22.5µg/L) is lost by this route. Urinary[21] excretion amounts to <0.005 mg/day. Mensturating women lose an additionally high variable amount during each menstural cycle from 0.006 to more than 0.025 mg/kg/day.

IRON DISTRIBUTION: [3]

PROTEIN	FUNCTION	AMOUNT(g)	PERCENT
Haemoglobin	Erythrocyte O_2 transport	2.6	65
Myoglobin	Muscle O_2 storage	0.130	6
Transferrin	Plasma iron transport	0.003	0.1
Ferritin	Intracellular iron storage	0.520	13
Haemosiderin	Intracellular iron storage	0.480	12
Others(Enzymes)		0.150	3.9
Catalase peroxidase	H_2O_2 degradation		
Cytochromes	Electron transport		
Aconitase	Tricarboxylic acid		
Ferrochelatase	Heme biosynthesis		
Duodenal cytochrome	Intestinal iron absorption		

IRON HOMEOSTASIS:



IRON REQUIREMENT:

Total body iron content is about 3 to 5 gm, out of which 75% is in blood, the rest is in liver, bone marrow and muscles. Iron is present in all cells

Recommended daily allowance is 20mg out of which only 10% (1-2mg) is absorbed. In pregnant and lactating women iron demand increased to 30- 60 mg. Demand increases as trimester increases. During first and second trimester it is about 30-40mg (3-4mg is absorbed) and third trimester it is about 60-70mg (6-7mg is absorbed). Total[4] iron requirement during pregnancy is about 1000mg.

- fetus and placenta-300 mg
- maternal haemoglobin-500mg
- excretion-200 mg

IR0N ABSORPTION:

Iron is absorbed in the upper part of duodenum.

- Only ferrous form of iron is absorbed not the ferric form.
- Ascorbic acid, gastric Hcl, cysteine & SH group of protein favour iron absorption by reducing ferric to ferrous form.
- 50-60 mg of ascorbic acid is sufficient for normal absorption.
- Calcium, copper, lead, phosphates and phytates inhibit iron absorption by forming insoluble iron salts.
- In iron deficiency anaemia iron absorption is increased 10 times
- Iron absorption is also regulated by hepcidin a protein synthesized by liver in response to intrahepatic iron stores

TRANSPORT:

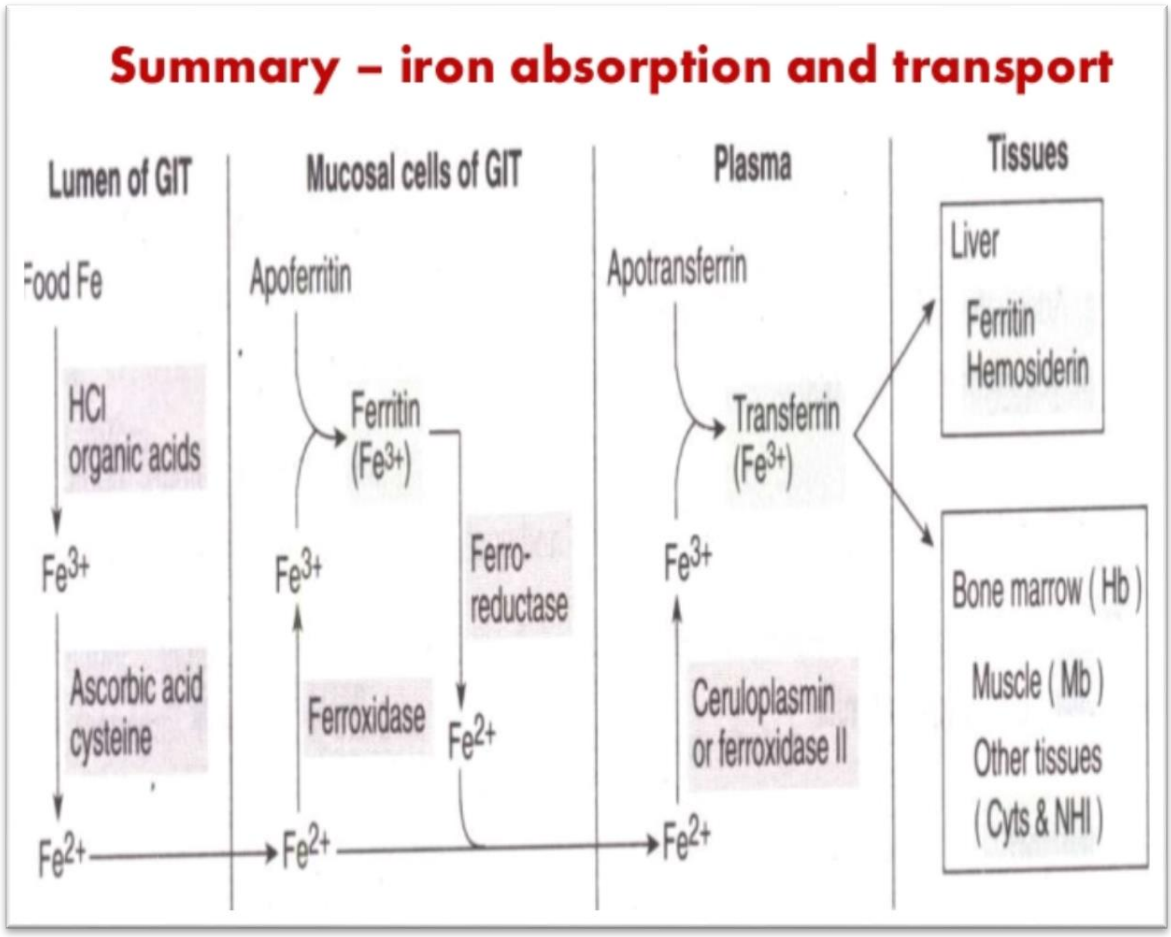
- Iron in ferrous form is oxidized to ferric form in mucosal cells
- It combines with Apo ferritin and forms ferritin.
- From the mucosal cells it enters the plasma in ferrous form
- It is oxidized to ferric form by ferroxidase.

- Ferric iron binds with the specific iron binding protein transferrin or siderophilin
- Transferrin is the major transport form of iron.
- Normal range of transferrin-250-450milligram/dl
- One molecule transports 2 ferric atoms and its half-life-7-10 days
- Total iron binding capacity of transferrin-250-450 µg/dl
- In iron deficiency anaemia TIBC is increased

STORAGE:

- Iron is stored in liver, spleen, bone marrow in the form of ferritin.
- Ferritin is the index of iron stores.
- It contains 23% of iron.
- Ferritin is also an acute phase reactant elevated in inflammatory condition.
- Normal range -15-150 ng/ml.
- In iron deficiency anaemia serum ferritin is decreased.

- Disorder of iron metabolism results in defective erythropoiesis leading to iron deficiency anaemia.



IRON DEFICIENCY ANAEMIA:

Defined by world health organisation as hemoglobin less than 11gm in postnatal women. Iron deficiency is the first common cause of anaemia in postpartum women, second is due to acute blood loss during delivery. Each ml of blood loss result in loss of 0.5mg iron. Prevalence in India varies from 50-70%. About 20% of maternal deaths worldwide is attributed to anaemia. In India 36% of maternal death is because of anaemia.

In healthy women with iron supplementation during pregnancy prevalence of anaemia in postpartum is about 14%.So without iron prophylaxis postpartum anaemia increased to about 24%.

PREVALENCE:

Different[1] regions of world are classified by WHO into different categories depending on the prevalence of anaemia.

- High prevalence - >40%
- Medium prevalence - 15-39%

- Low prevalence - 5-14.9%
- Not a problem - <5%

Currently Center for Disease control and Prevention (CDC; Atlanta, Georgia) recommends selective anaemia screening at 4-6 weeks postpartum for women who had anaemia continued through the third trimester, excessive blood loss during delivery, and multiple births.

HIGH RISKS GROUP:

- Uncorrected anaemia during antenatal period
- Teenage pregnancy
- Low socio economic status
- Multiple births without adequate spacing
- Multiple pregnancy
- Chronic liver disease
- Chronic kidney disease due to decreased erythropoietin
- Hemolytic disorder

CAUSES:

- Increased blood loss during delivery (In normal vaginal delivery >500ml, caesarean>1000 ml)
- Iron malabsorption due to vomiting, or due to gastrointestinal disease
- Due to intake of iron deficient foods.
- Due to hookworm infestations

STAGES OF IRON DEFICIENCY ANAEMIA:

There[2] are three stages of development of iron deficiency anaemia.

1. storage iron depletion
2. Iron deficient erythropoiesis
3. Iron deficiency anaemia.

STAGE IF STORAGE IRON DEPLETION:

Status of iron stores assessed by measuring the concentration of serum ferritin. Ferritin is a polypeptide of molecular weight 450000 kDa.. 1ng/ml of serum ferritin is equivalent to 8mg of

storage iron. Normal range 15-150 ng/ml levels less than 12 ng indicates complete depletion of iron stores. Low ferritin is a sensitive marker of iron deficiency but it is not very specific as it is an acute phase reactant elevated in infection and inflammation. Storage iron depletion leads to early functional iron deficiency which signifies that the iron supply to the bone marrow and the other tissue is marginally inadequate but there is still no anaemia.

CATEGORIES OF IRON DEFICIENCY:

Degree of iron deficiency	Haemoglobin	Serum ferritin
Iron deficient not anaemic	>11gm/dl	<12ng/ml
Iron deficiency anaemia	<11gm/dl	<12ng/ml

STAGE OF IRON DEFICIENT ERYTHROPOIESIS:

It is diagnosed by ferro kinetic studies a battery of tests that diagnose and differentiate it from other anaemic conditions. In this stage the peripheral smear shows normocytic cells. Laboratory test used to

detect this stage are serum transferrin saturation (percentage), total iron binding capacity (TIBC), erythrocyte zinc protoporphyrin concentration (ZPP), soluble transferrin receptor concentration assay. Transferrin saturation is calculated from the ratio of serum iron to TIBC. Free erythrocyte protoporphyrin represents the substrate for heme synthesis. Levels rise when there is defective iron supply for the developing red cell. Mild form of IDE is associated with ZPP-ratios between 40 and 70 μ mol/mol. Advanced IDE is associated with >70 μ mol/mol.

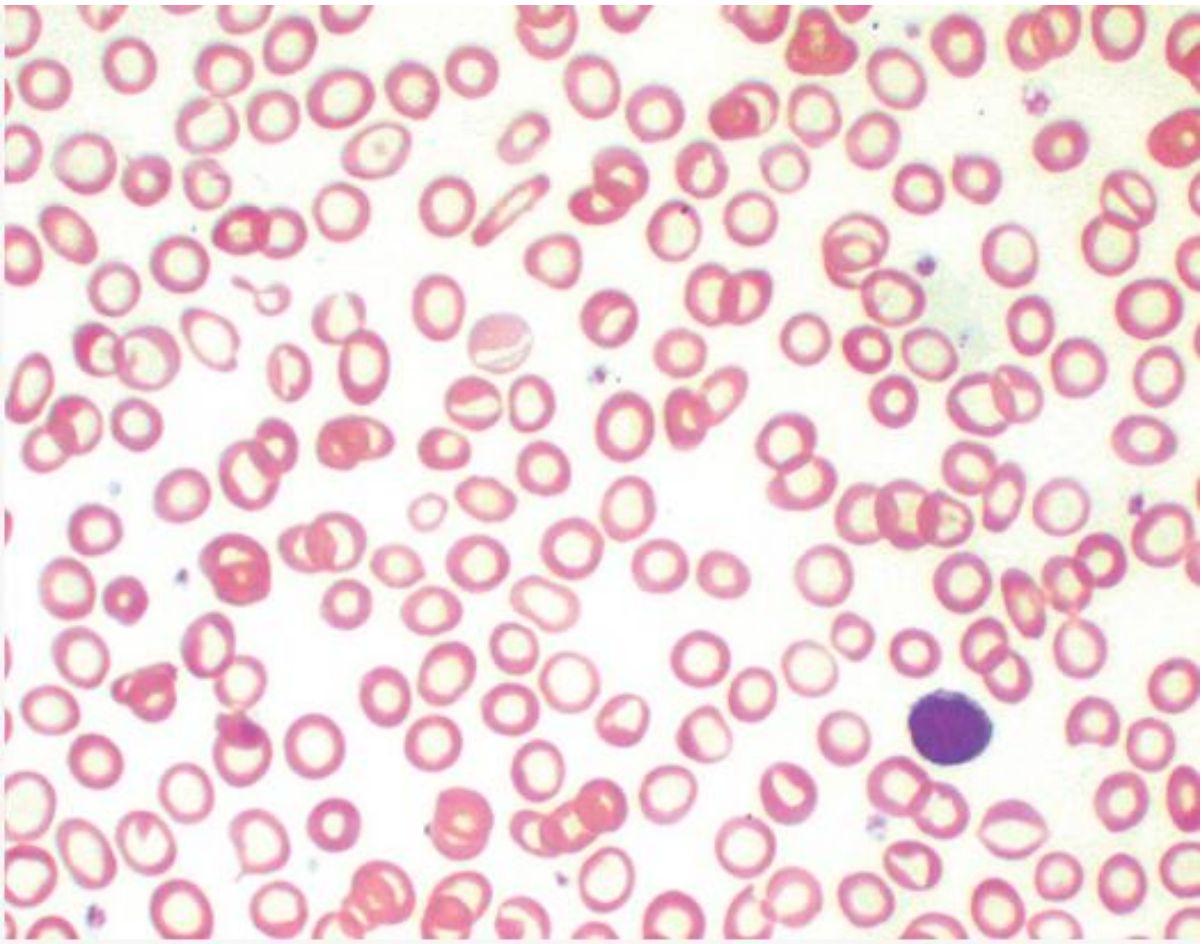
Serum iron transferrin receptor (Tfr) measured by ELISA is a reliable method for assessing cellular iron status. It is particularly valuable in identifying iron deficiency in pregnancy since it is the only measurement to accurately reflect the iron deficit between the points of storage iron depletion and development of anaemia. Serum Tfr increases progressively in direct proportion to the magnitude of iron deficiency. Iron deficiency is associated with 3-5 fold increase in the concentration of these receptors.

PARAMETERS	NORMAL RANGE	IDA
Serum iron µg/dl	60-120	<60
Serum ferritin ng/ml	15-150	<15
TIBC µg/dl	325-400	>400
Transferrin saturation	20-50%	<15%
Tfr mg/dl	5.8	8.8
ZPP µg/dl	<40	>70

STAGE OF IRON DEFICIENCY ANAEMIA:

It represents the tip of iceberg. Changes in the red cell indices is the good indicator of iron deficiency. Mean cell volume (MCV), Mean cell haemoglobin (MCH), and mean cell haemoglobin concentration are all reduced. There is also decreased packed cell volume. The peripheral smear picture is typically microcytic hypochromic. New cells are being generated at the normal rate and the reticulocyte shows little deviation from the normal and it is increased. The cells besides being smaller than normal, they are of unequal size and abnormal staining i.e. anisocytosis and polychromasia.

MICROCYTIC HYOCHROMIC PICTURE



HEMATOLOGICAL PARAMETERS IN DIFFERENT TYPES OF ANAEMIA:

TYPE OF ANAEMIA	**PCV**	**MCV**	**MCH**	**MCHC**	**RBC MASS**
Normocytic	Decreased	Normal or increased	Normal	Normal	Decreased
Microcytic hypochromic	Decreased	Decreased	Decreased	Decreased	Normal or decreased
macrocytic	Decreased	Increased	Increased	Decreased	Normal or decreased

GRADES OF ANAEMIA:

CATEGORY	HAEMOGLOBIN LEVEL(g/dl)
Mild	8.0-10.9
Moderate	5.0-7.9
Severe	Less than 5

SIGNS AND SYMPTOMS:

Postpartum women with iron deficiency anaemia have poor quality of life. They require longer hospitalisation. In mild anaemia they may be asymptomatic. Mostly they have,

- Easy fatigability
- breathlessness
- palpitation
- Decreased lactation
- Postnatal depression

They develop complications like,

- Subinvolution of uterus
- puerperal sepsis
- Deep vein thrombosis
- Cardiac failure in severe anaemia
- Cortical vein thrombosis

They have signs like facial pallor, koilonychia, and tachycardia, haemic (systolic) murmur due to increased haemodynamic circulation, facial puffiness and pedal edema. JVP is increased in cardiac failure.

DIAGNOSIS:

The Traditional method to diagnose anaemia is by hemoglobin estimation and by doing peripheral smear. Other parameters used are the packed cell volume, blood indices like MCV, MCH, MCHC, serum ferritin, serum iron, TIBC, Transferrin saturation.

PREVENTION:

Iron deficiency anaemia can be prevented by oral iron prophylaxis. So in order to prevent anaemia in antenatal and postnatal period[2] WHO recommends 60mg of elemental iron with 400 microgram of folic acid per day for 6 months prophylactically where the prevalence of anaemia in pregnancy is <40%.This dose is supplemented for another 3 months postpartum if the prevalence is > 40%.

In India where anaemia affects all age group, women are already deficient in iron store,so when they enter into pregnancy and puerperium where the iron demand is very high they are more prone to develop iron deficiency anaemia.So the National Nutritional anaemia control programme of India recommends 100mg of elemental iron plus 500 microgram of folic acid prophylactically for 100 days starting from the second trimester.

MANAGEMENT:

Iron deficiency anaemia is managed by oral iron, intramuscular iron, intravenous iron, and blood transfusion depending on the severity of anaemia. Oral iron has poor compliance of the patients, gastrointestinal side effects. Intramuscular iron is painful. They may develop anaphylactic reactions, arthralgia, dizziness, fever, headache, malaise, fever, headache and vomiting. They require test dose.

Intravenous iron-First generation is the iron dextran which require test dose and longer duration for administration and have side effects. The only advantage is possibility of giving large dose around 1000mg in single sitting.

Second generation is the Iron sucrose where test dose is not required and with fewer side effects. No severe anaphylactic reactions reported so far. Third generation intravenous iron is the ferric carboxymaltose, ferumoxytol, iron isomaltoside. Ferric carboxymaltose is approved by FDA and is upcoming in the management of iron deficiency in postpartum and in pregnancy as well in second and third trimester.

The response to iron therapy is monitored by improvement of symptoms and by the reticulocyte count. The absence of response warrants complete investigations, including bone marrow biopsy.

According to the 10th five-year plan (ICMR) oral iron in therapeutic dose should be administered in mild anaemia. However in moderate to severe anaemia second and third generation iron should be started as first line therapy irrespective of blood transfusion because of increased transfusion reactions and transfusion associated infections like HIV,Hepatitis B ,Hepatitis C,cytomegalovirus etc. Parenteral iron has several advantages because of less allergic reactions and fewer side effects. Parenteral iron has a high availability for erythropoiesis, little renal excretion and low tissue accumulation and toxicity

SETU RATHOD[5] et al conducted a study in SCB Medical college,Cuttack, between September 2010 and august 2012.The study included 366 women with postpartum anaemia with haemoglobin <10gm/dl and were randomly assigned to receive oral iron or iron sucrose or FCM. Changes in haemoglobin and serum ferritin at 2week and 6 week after treatment were measured and analysed using

ANNOVA. A statistically significant increase in Hb and serum ferritin were observed in all three groups but the increase in FCM group was significantly higher ($p<0.00001$) than conventional iron sucrose and oral iron group. The mean increase in Hb after 2nd week was 0.8;2.4;3.2gm/dl and 2.1;3.4 and 4.4 gm/dl at 6 weeks. The mean increase in serum ferritin after 2 weeks was 2.5;193.1;307.1 and 14.2;64;106.7ng/ml, after oral iron, iron sucrose and FCM groups respectively. Adverse drug reactions were significantly less ($p<0.001$) in FCM group when compared with other two groups. They concluded that FCM elevates Hb level and serum ferritin level faster than iv iron sucrose and oral iron.This study was published in International journal of applied and basic medical researchJan-april 2015,Vol 5,Issue 1.

DAVID B.VAN WYCK[6] et al conducted a randomized multicenter trial in 43 centers; 40 in United States, 3 in Mexico. They selected anaemic women with Hb equal to or less than 10gm within 10 days postpartum .174 patients received 350 iron doses of FCM (mean total dose 1403.1mg) in 3,2 or 1 injection (10.9%,79.3%,9.8% of patients respectively).178 received oral iron. Patients who received FCM

achieved Hb rise equal to or greater than 2 gm/dl earlier (7.0 days compared with 14 days $p<0.001$), were more likely to achieve a Hb rise greater than or equal to 3.0 gm /dl at any time (86.3% compared with 60.4%, $p<0.001$) and were more likely to achieve a Hb greater than 12 gm/dl (90.5% compared with 68.6%, $p<0.001$) when compared with oral iron.

CHRISTIAN BREYMANN[7] et al (International journal of Gynaecology and Obstetrics 2008) compared the efficacy of iron carboxymaltose with ferrous sulphate to treat iron deficiency anaemia in postpartum. Patients were randomized (2:1 ratio) to receive FCM 9upto 3 weekly doses of 1000mg maximum, applied in 15 minutes (n=227) or ferrous sulphate (100mg twice daily,12 weeks, n=117). Changes in Hb and iron stores upto 12 week analyzed. They concluded that parenteral FCM is a safe and effective treatment for postpartum anaemia with advantages of a shorter treatment period, better compliance, rapid return of iron stores and lower side effects.

MELVIN H SEID[8] et al, conducted a multicenter randomized trial at 28 centers in united states between May 9,2006 and

Dec 27,2006(AJOG 2008).291 women less than 10 days after delivery with haemoglobin 10gm /dl or less were randomized to receive the FCM (n=143),1000 mg or less intravenously over 15 minutes or less, repeatedly weekly to a calculated replacement dose (maximum 2500mg) or ferrous sulphate (n=148) 325mg orally thrice daily for 6 weeks. FCM patients achieved 1) a haemoglobin greater than 12g/dl in shorter time period with shorter time period with sustained haemoglobin greater than 12 g/dl at day 42.2) achieve Hb rise 3g/dl or greater more quickly. 3)attain higher serum transferrin saturation and ferritin levels.

Patel[10] et al conducted a clinical observational study at tertiary care teaching hospital (SBKS Medical Institute and Research Centre) for a period of 4 months in 30 pregnant and 30 post-partum women. The baseline hemoglobin and serum ferritin levels were recorded prior to treatment. After completion of the treatment the women were followed up for changes in hemoglobin and serum ferritin levels on day 8 and day 15. The mean rise of hemoglobin value was 5.2gm/l for ferric carboxymaltose and 4.1gm/l for iron sucrose in pregnant women. For postpartum women mean rise of hemoglobin was 4.9gm/l on the 15th

day of treatment. Side effects were reported in 40% among patients treated with iron sucrose as compared to 16.67% in case of ferric carboxymaltose. Ferric carboxymaltose administration in pregnant women is safe and well tolerated by postpartum women. Ferric carboxymaltose is associated with fewer side effects as compared to iron sucrose in present study. It also offers the advantage of a much higher iron dosage at a time reducing the need for repeated applications and increasing patients comfort.

Lysung -Williamson KA[12] et al reviewed many trials, patient received FCM equivalent to an iron dose of less than or equal to 1000 mg (or 15 mg/kg in those weighing <66 kg) administered over less than or equal to 15 minutes or 100 mg of iron twice daily in postpartum patients. FCM improves haemoglobin level and replenish depleted iron stores and improve health related quality of life in patients with iron deficiency anemia.

Bernd Froessler[14] et al conducted prospective observational study with 65 anaemic pregnant women who received ferric caroxymaltose upto 15mg/kg between 24 and 40 weeks of pregnancy.

The study showed significant increase in Hemoglobin values above baseline levels in all women. Ferritin levels increased significantly after the infusion.

Khalafallah[11] et al did a prospective randomized controlled study at two centers in Tasmania, Australia, in patients undergoing elective surgery with IDA. Between Dec 2014 and May 2015, 201 eligible patients are recruited. 103 were assigned to intravenous FCM and 98 to standard care. Baseline haemoglobin was 105.5g/L (SD 13.8) in the standard group versus 106.2g/L(11.9) in the FCM ,improving at 4 weeks to 121.5g/L(SD 14.5) in the standard group and 130.1g/L (11.3) in the FCM group(mean difference of 7.8g/L,95%CI 3.79-11.9;p<0.0001 in favour of FCM group). Significant improvements in serum iron (5.36micromol/L,95%CI 3.62-7.09;P<0.0001),iron saturation (11.4%,95% CI 8.33-14.50;P<0.0001) and serum ferritin saturation (468 microgram /L,95% CI 355-582;P<0.0001) were also noted in FCM group at 4 weeks compared with standard care, although no differences were noted in transferrin concentrations(0.06 g/L,95% CI0.01-0.85;P=0.O35). No adverse reactions with FCM noted. Postoperative

intravenous FCM is a feasible and pragmatic management approach in surgical patients with functional IDA.

Rognoni.C[13] et al performed a systematic literature review of published RCTs on the use of FCM in iron deficiency between July and October 2014. The initial search yielded 1027 citations, which was decreased to 21 studies eligible for inclusion in the review. Studies were heterogeneous in the number of patients randomised, iron deficiency-related conditions addressed, trial inclusion criteria, time horizon, treatment dosage and outcomes assessed. Six studies with the same time horizon (i.e. 6 weeks) were included in the network meta-analysis. Considering the differences between final and initial outcome values for each iron formulation, the mean difference of these differences (delta) was estimated for each couple of treatments involving ferric carboxymaltose. Significant improvements in serum ferritin (µg/l) were obtained with ferric carboxymaltose compared to oral iron (delta 172.8; 95 % CI 66.7–234.4) and in haemoglobin (g/dl) with respect to ferric gluconate (delta 0.6; 95 % CI 0.2–0.9), oral iron (delta 0.8; 95 % CI 0.6–0.9) and placebo (delta 2.1; 95 % CI 1.2–3.0).

Kuster M[15] et al retrospectively analyzed data from 173 patients Intravenous ferric carboxymaltose led to a significant increase in hemoglobin and serum ferritin levels. Side effects of intravenous treatment were found in 2% of all case.

Mahey R[16] eta al conducted randomized control trial between April 2013 and May 2014 in patients older than 18yrs of age presenting in a hospital in New Delhi with anemia due to abnormal uterine bleeding. One group received iron sucrose and other one received FCM. Increase in mean Hb from baseline is higher in FCM group at 6 weeks. P =.005. Treatment with FCM resulted in rapid increase in hemoglobin level in patents with anemia due to abnormal uterine bleeding.

Breymann C[17] compared the efficacy and safety of intravenous FCM with first line ferrous sulphate in pregnant women with iron deficiency anaemia. Pregnant women (n=252), with mean gestational age 16-33 weeks with iron deficiency anaemia were randomized 1:1 to FCM (1000-1500mg) or Ferrous sulphate(200mg of Fe/day) for 12 weeks. Significant women achieved anaemia correction

with FCM Vs Ferrous sulphate. (Hb ≥11gm/dl, 84% vs 70%; odds ratio 2.06; 95% confidence interval.1.07,3.97; p=.031) with in shorter time (3.4 Vs 4.3 weeks). FCM significantly improved vitality (p=0.025) and social functioning (p=0.049) prior to delivery. Adverse reactions were low with FCM when compared with ferrous sulphate.

Christoph P[19] performed a retrospective analysis of 206 pregnant women treated with either ferric carboxymaltose or iron sucrose for iron deficiency anaemia with intolerability to oral iron substitution or insufficient haemoglobin increase after oral iron treatment or need for rapid haemoglobin correction. The mean rise in haemoglobin was significantly higher in women who received FCM. The rise in haemoglobin was 15.4g/dl for FCM and 11.7 g/dl for iron sucrose.

ONKEN JE[18] evaluated FCM vs oral iron in iron deficiency anaemia patients. After 14 days of oral iron, 507 participants responding inadequately to oral iron (haemoglobin <10g/dl were assigned to group A (2 doses of FCM 750mg 1 week apart) or Group B (oral iron 325 mg thrice /day for 14 additional days. Another 504 participants who were not appropriate for oral iron were assigned as Group C to receive FCM

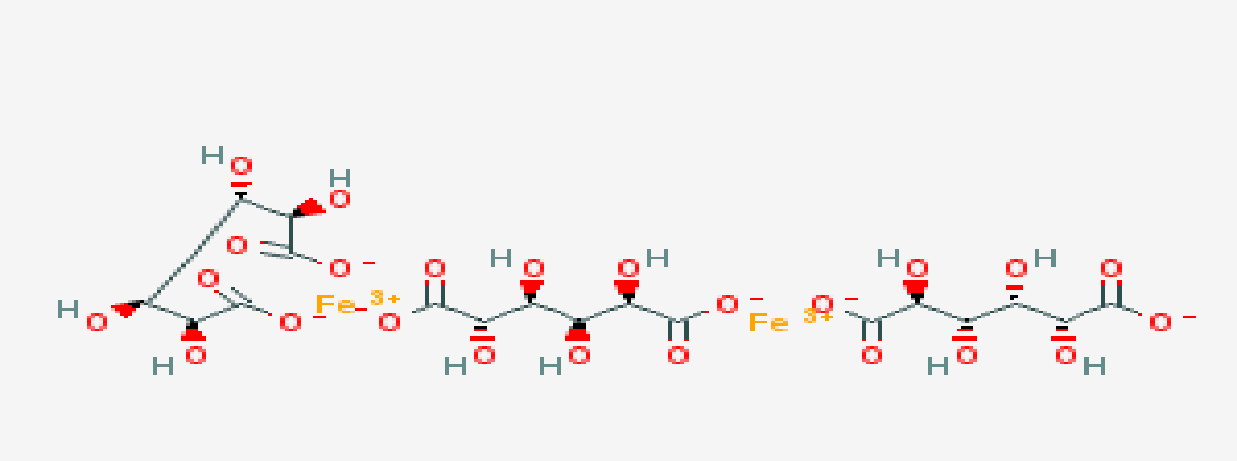
as above or Group D (standard care). Mean increase in Hb was significantly greater in Group A-FCM than Group B oral iron 1.57(±1.19)g/dl versus 0.80(±0.80)g/dl(p=0.001). Comparison of Group C vs Group D also demonstrated significant mean increase in Hb from baseline to highest value.2.9(±1.64)g/dl versus 2.16(±1.25)g/dl(p=0.001)

Dillion R[20] et al from a retrospective single center study compared the efficacy of three intravenous iron preparations (iron dextran, iron sucrose, and FCM) in 280 patients. While comparing the haemoglobin and other measures of Iron deficiency, after six weeks of treatement, they have showed statistically and clinically significant increase in haemoglobin level with FCM group.

IRON SUCROSE:

CHEMICAL FORMULA:

$C_{18}H_{24}Fe_2O_{24}$



Iron sucrose is a brown, sterile, aqueous complex of polynuclear iron (III) hydroxide in sucrose containing 20mg of elemental iron per ml.

The sterile solution has an osmolality of 1250mosm/l. the product does not contain preservatives.

Molecular wt: 34,000-60000 daltons.

pH:10.5 -11.1

MECHANISM OF ACTION:

Following intravenous administration, it is dissociated into iron and sucrose by reticuloendothelial system and iron is transferred from the blood into pool of iron in the liver and bone marrow. Ferritin sequesters iron in a nonionic form from which iron is easily available. Iron is transported as a complex with transferrin to target cells including erythroid precursor cells. The iron in the precursor cells is incorporated into haemoglobin as the cells mature into red blood cell.

PHARMACOKINETICS:

Its iron component exhibits first order kinetics.

Half-life: 6 hrs

Total clearance: 1.2 litres/hour

Non-steady state apparent volume of distribution : 10 litres

Steady state apparent volume of distribution: 7.9 litres.

DISTRIBUTION:

In healthy adults, its iron component appears to distribute mainly in blood and to some extent in extra cellular fluid.

ELIMINATION:

The sucrose component is eliminated mainly by urinary excretion. Some iron is also eliminated in the urine (approximately 5%) within 24 hrs.

SIDE EFFECTS:

Head ache, fever, pain, asthenia, malaise, abdominal pain.

INTERACTION:

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

Chloramphenical may decrease the effect of iron sucrose

CONTRA INDICATIONS:

1. Evidence of iron over load.

2. Anemia not caused by iron deficiency.

3. Known hypersensitivity to I.V iron sucrose (or) any of its inactive compounds.

METHODS AND ROUTE OF ADMINISTRATION:

No test dose is required.

1. Slow IV injection:

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

2. Slow IV infusion:

100mg (2amp) to be diluted with 100ml of normal saline immediately prior to infusion and to be infused over a period of atleast 15min(6mg/min).

DOSAGE FREQUENCY:

200mg/day given on alternate days until the required dose is infused.

Chandler et al observed that optimal doses of 200-300mg infused intravenously over 2 hours were well tolerated and safe. Patients who received a dose of 400-500mg intravenously over 2 hours experienced hypotension, nausea and low back pain.

SUPPLY AND STORAGE:

Supplied as 5 ml single dose vial. Each 5 ml vial contains 100mg of elemental iron (20mg/ml). Stored at 25°C. Excursions permitted to 15-30°C.

IRON SUCROSE INJECTION :

FERRIC CARBOXYMALTOSE:

Parenteral iron complex consisting of colloidal ferric III hydroxide core stabilized by carbohydrate shell so there is controlled delivery of iron to cells of reticuloendothelial system and subsequent delivery to the iron binding proteins ferritin and transferrin .It has low immunogenic potential, dextran free product and not predisposed to anaphylactic reaction.

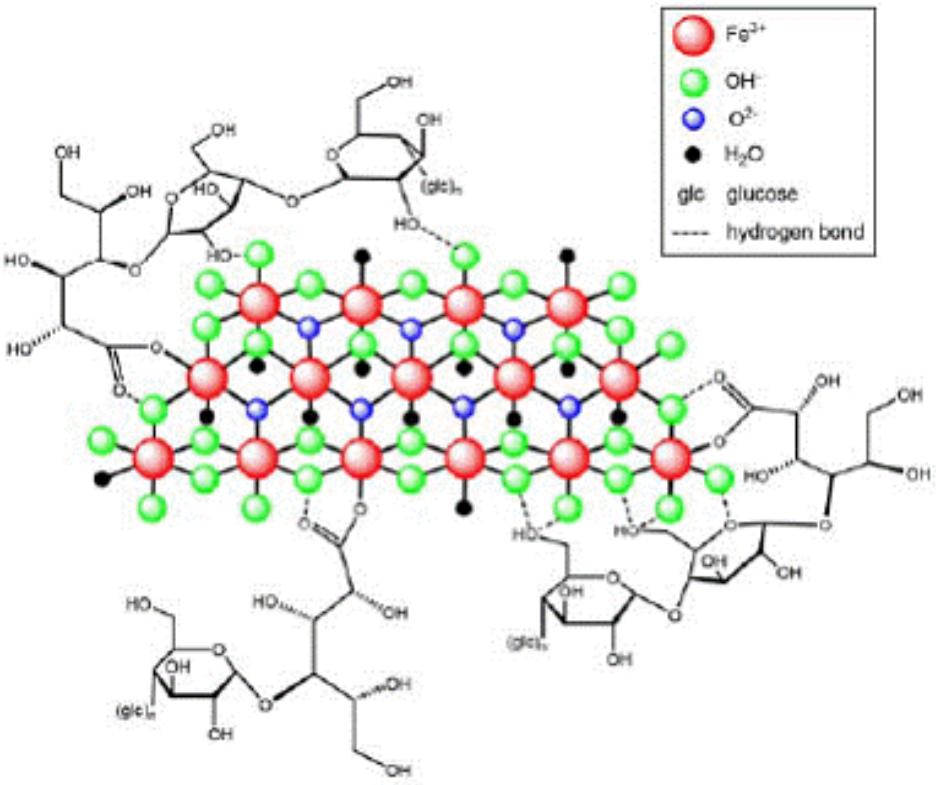
MECHANISM OF ACTION:

FCM results in transient elevations in serum iron, serum ferritin and transferrin saturation and ultimately the correction of haemoglobin and replenishment of depleted iron stores.serum iron concentrations increase in dose dependent manner after intravenous FCM.

CHEMICAL FORMULA:

$$[FeOx(OH)y(H2_2O)z]n\ [\{C_6H_{10}O_5)m(C_6H_{12}O_7)\}l]k$$

n=10^3; m=8; l=11; k=4 (l-represents the mean branching degree of the ligand)



Half-life :4-12 hours.

pH:5-7 (neutral)

Molecular weight: 150000 daltons

DISTRIBUTION:

FCM is cleared rapidly from the circulation and distributed mainly in the bone marrow (approximately 80%) and also to the spleen and bone marrow.

FCM INJECTION :

INDICATIONS:

- iron deficiency anemia
    - pregnancy
    - postpartum
- Anemia due to chronic kidney disease
- Anemia in abnormal uterine bleeding.
- Anaemia in inflammatory bowel disease

CONTRAINDICATIONS:

- Hypersensitivity to iron products.
- Iron overload

METHOD AND ROUTE OF ADMINISTRATION:

Slow intravenous infusion-Maximum of 1000 mg/week/dose diluted in 0.9 normal saline and infused over 15 minutes.

STORAGE:

Do not store above 30°C. Do not freeze

SHELF LIFE:

36 months.

EXCIPIENTS:

Sodium hydroxide and hydrochloric acid for PH adjustment

SIDE EFFECTS:

Headache, dizziness, nausea, abdominal pain, constipation, diarrhoea, and injection site reactions.

MONITORING PARAMETERS:

During transfusion the patient should be continuously monitored by blood pressure, pulse rate and for any adverse reactions. They should be monitored for 30-60 minutes following infusion for transfusion reactions.

MATERIALS AND METHODS

MATERIALS AND METHODS

The study was conducted in Govt RSRM Lying Hospital - Stanley Medical College, Chennai during year August 2015 – 2016.

100 Postnatal women with hemoglobin between 8-10gm% were selected and placed randomly into two groups. One group of 50 postnatal mother received iron sucrose while the other 50 postnatal mother received Ferric carboxymaltose.

INCLUSION CRITERIA:

Postnatal women with Iron deficiency anaemia with HB 8-10gm.

EXCLUSION CRITERIA:

- H/O Allergy to iron compound
- Chronic kidney disease
- Anaemia due to other causes(Including postpartum Haemorrhage)

- Hematological disorder
- Bronchial asthma
- Hepatitis
- HIV
- Heart disease
- H/O recent blood transfusion

DOSE CALCULATION:

In postnatal women 24 hrs after delivery Haemoglobin estimation, blood indices, peripheral smear and serum ferritin should be done. Patient with iron deficiency anaemia with HB between 8 to 10gm were selected. Required iron dose is calculated using the formula below. Target HB is 12 gm.

2.4 x Body weight in Kg x (Target HB - Actual HB) +500 gm.

IRON SUCROSE: (No test dose is required)

1. It is given by IV injection according to the iron dose calculated and rounded up to the nearest multiple of 100 for each individual.
2. 200 mg elemental iron diluted in 200 ml 0.9% normal Saline is the maximum dose given over 15 to 20 min and repeated on alternate days as required.

FERRIC CARBOXYMALTOSE: (No test dose is required)

1) It is given by IV injection according to the iron dose calculated and rounded up to the nearest multiple of 100 for each individual.
2) Maximum single dose of 1000 mg diluted in 250 ml of 0.9% normal Saline given over 15 minutes and not more than once a week.

DATA ANALYSIS AND INTERPRETATION:

Data analysis is a systematic organization and synthesis of research data and testing of research hypothesis using that data. Interpretation is the process of making sense of the study and examining the implication (Polite and Beck, 2004). In this study the data was collected, assembled, analyzed, tested individually.

Paired't' test:

This test was adopted to determine the effect of iron sucrose and FCM before, 2 weeks and 4 weeks after therapy.

Independent t test:

This test is used to identify whether there is significant difference among iron sucrose and FCM group.

INVESTIGATIONS:

- Hemoglobin,
- Peripheral smear,
- Mean Corpuscular Volume,
- Serum Ferritin

Pre-treatment and 2nd and 4th week post-treatment investigations are done for both the groups and results are compared.

MONITORING PARAMETERS:

- Blood pressure
- Pulse rate
- Temperature
- Allergic reactions
- Nausea
- Vomiting

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

IRON SUCROSE:

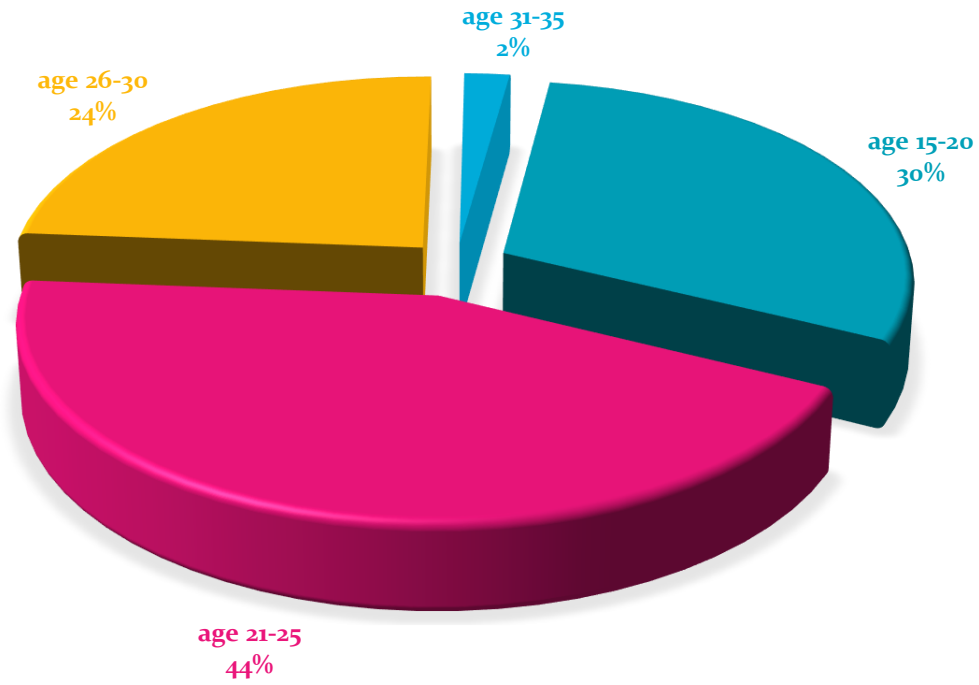
Following are the demography features of Iron Sucrose participants and their outcomes following Iron Sucrose administration.

AGE DEMOGRAPHY IN IRON SUCROSE:

Out of 50 women studied in iron sucrose group 30%(n=15) belong to age group 15-20yrs,44% belong to age group 21-25yrs(n=22),24% belong to 26-30 yrs(n=12),2% belong to 31-35 yrs(n=1).

AGE	Count	Percentage
age 15-20	15	30%
age 21-25	22	44%
age 26-30	12	24%
age 31-35	1	2%

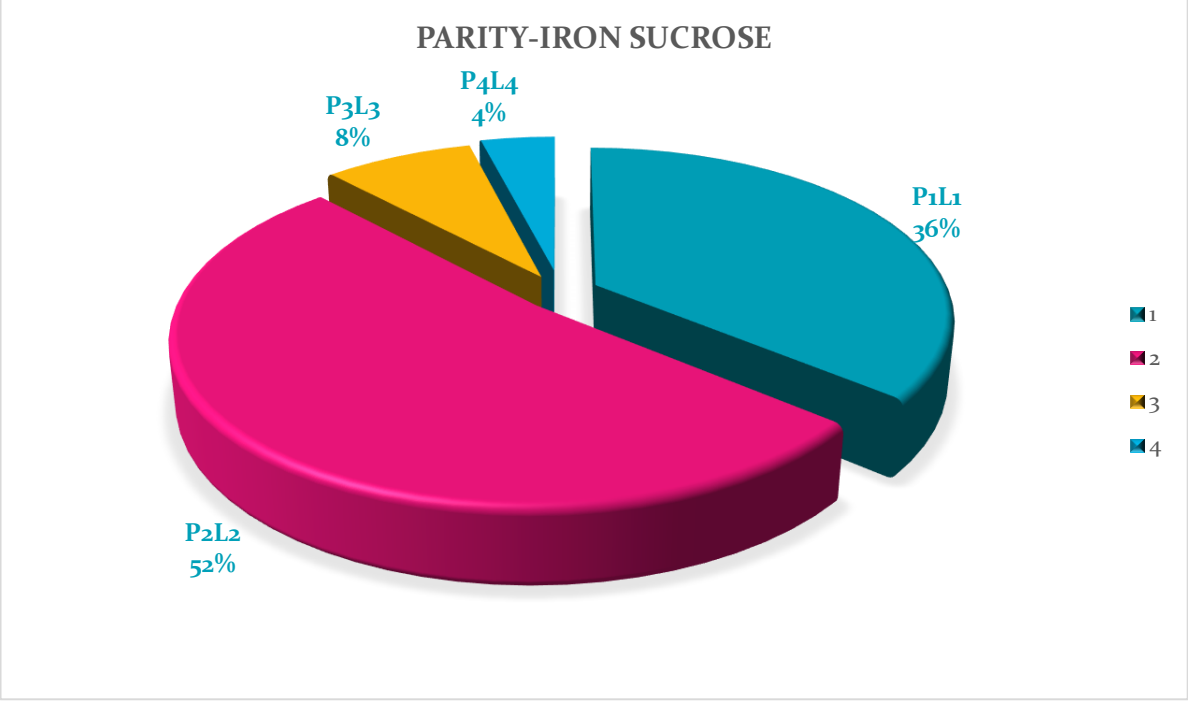
AGE DISTRIBUTION -IRON SUCROSE



PARITY IN IRON SUCROSE:

Among parity distributon 52% belong to P2L2,36% belong to P1L1 ,8% belong to P3L3 and 4% belong to P4L4 group.so prevalence of anaemia is more among multiparas.

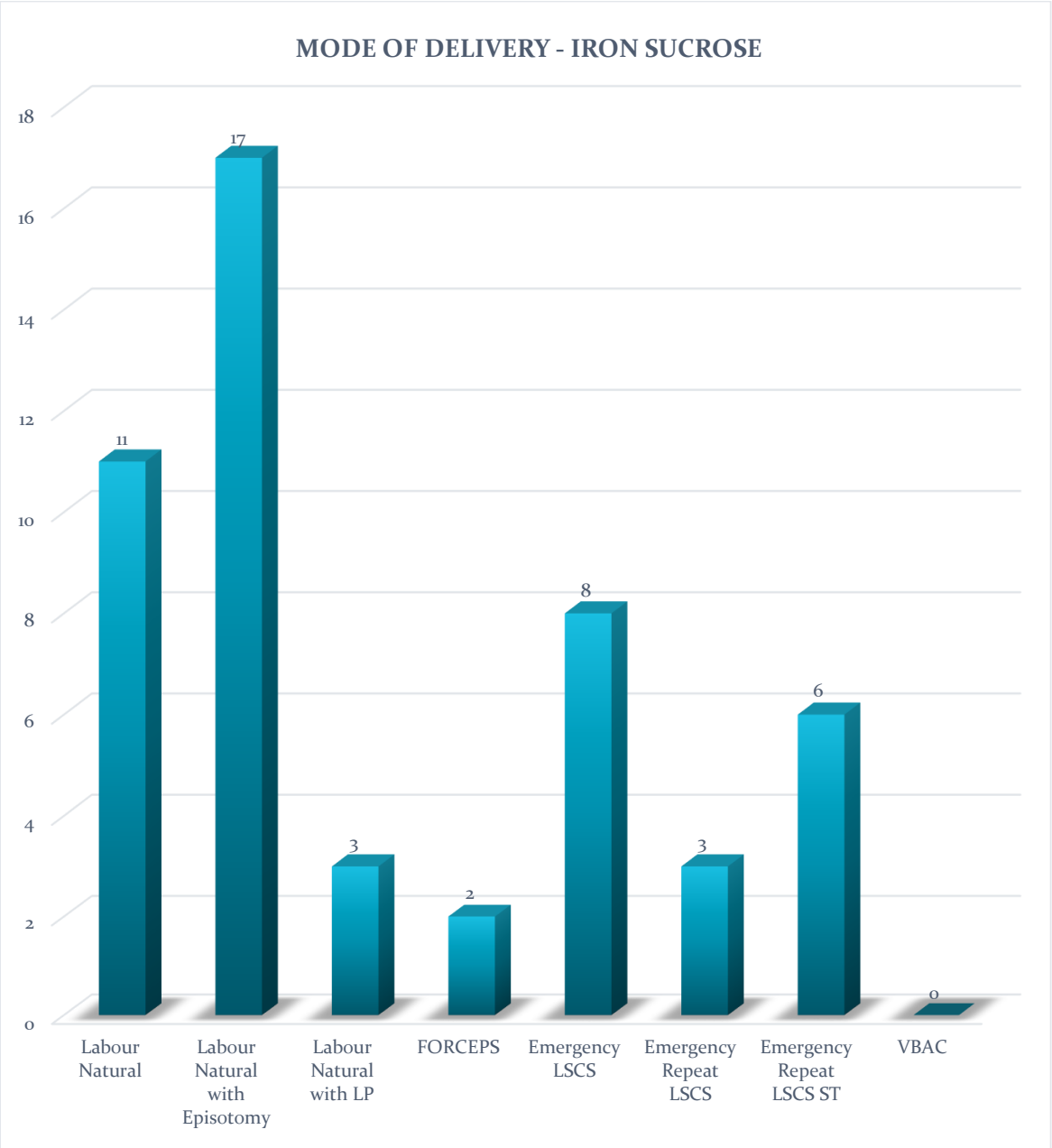
OB.Code	Parity	Count	Percentage
1	P1L1	18	36%
2	P2L2	26	52%
3	P3L3	4	8%
4	P4L4	2	4%



MODE OF DELIVERY (MOD) IN IRON SUCROSE:

Among mode of deliveries 31 deliveries were labour natural (includes with episotomy and with lacerated perineum), forceps delivery -2 and emergency lscs- 17(includes repeat lscs and repeat lscs with sterilization)

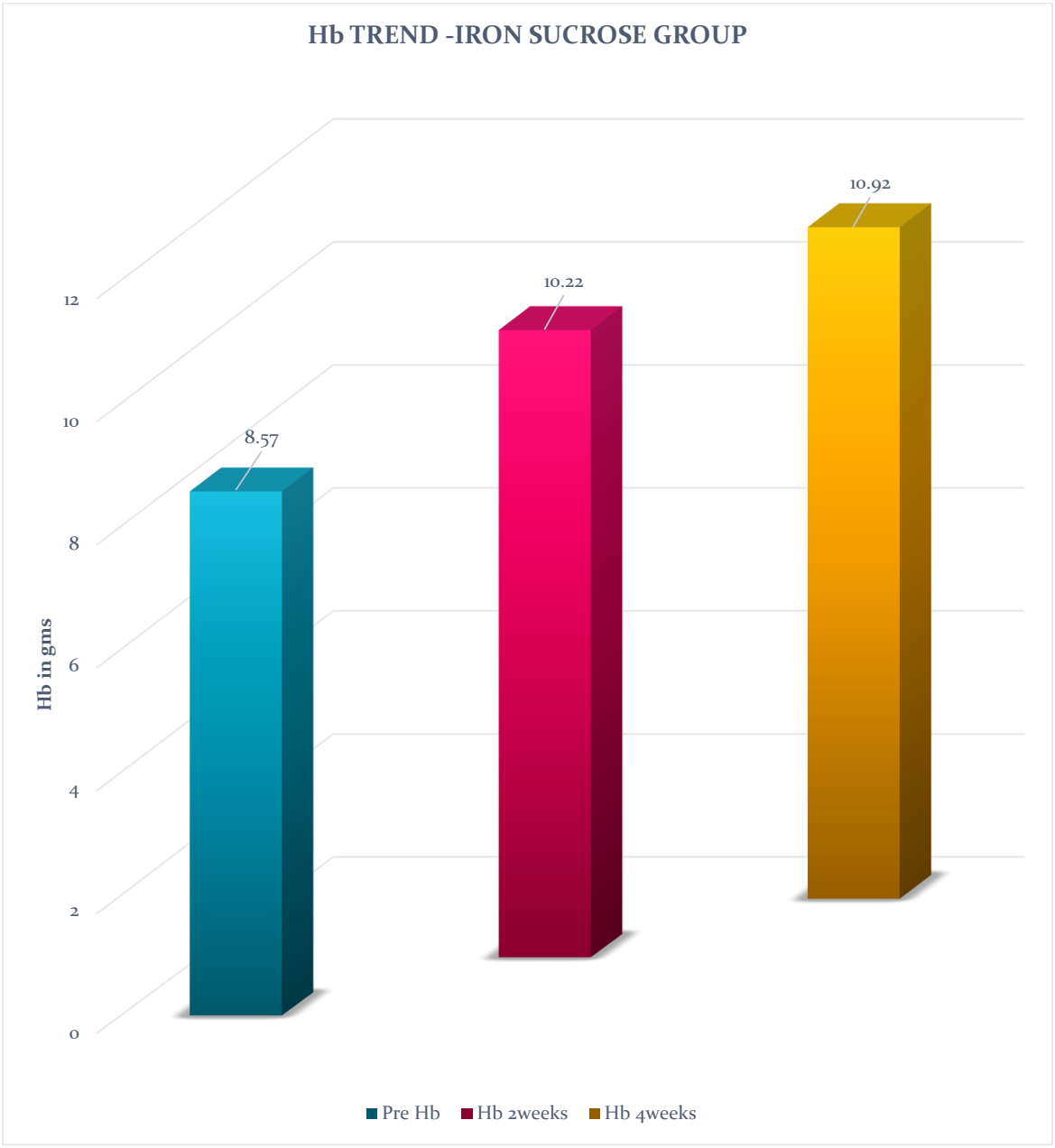
MOD.Code	MOD	Iron sucrose
1	Labour Natural	11
2	Labour Natural with Episotomy	17
3	Labour Natural with LP	3
4	FORCEPS	2
5	Emergency LSCS	8
6	Emergency Repeat LSCS	3
7	Emergency Repeat LSCS ST	6
8	VBAC	0


MODE OF DELIVERY - IRON SUCROSE
18
16
14
12
10
8
6
4
2
0
11
17
3
2
8
3
6
0
Labour Natural
Labour Natural with Episotomy
Labour Natural with LP
FORCEPS
Emergency LSCS
Emergency Repeat LSCS
Emergency Repeat LSCS ST
VBAC

MEAN HEMOGLOBIN TREND IN IRON SUCROSE GROUP:

Improvement of haemoglobin with iron sucrose from pretreatment haemoglobin (mean) 8.57 gm to 10.22(mean) at two weeks and to 10.92(mean) at 4 weeks. The average increase is about 1.65 gm at 2 weeks and 2.35gm at 4 weeks.

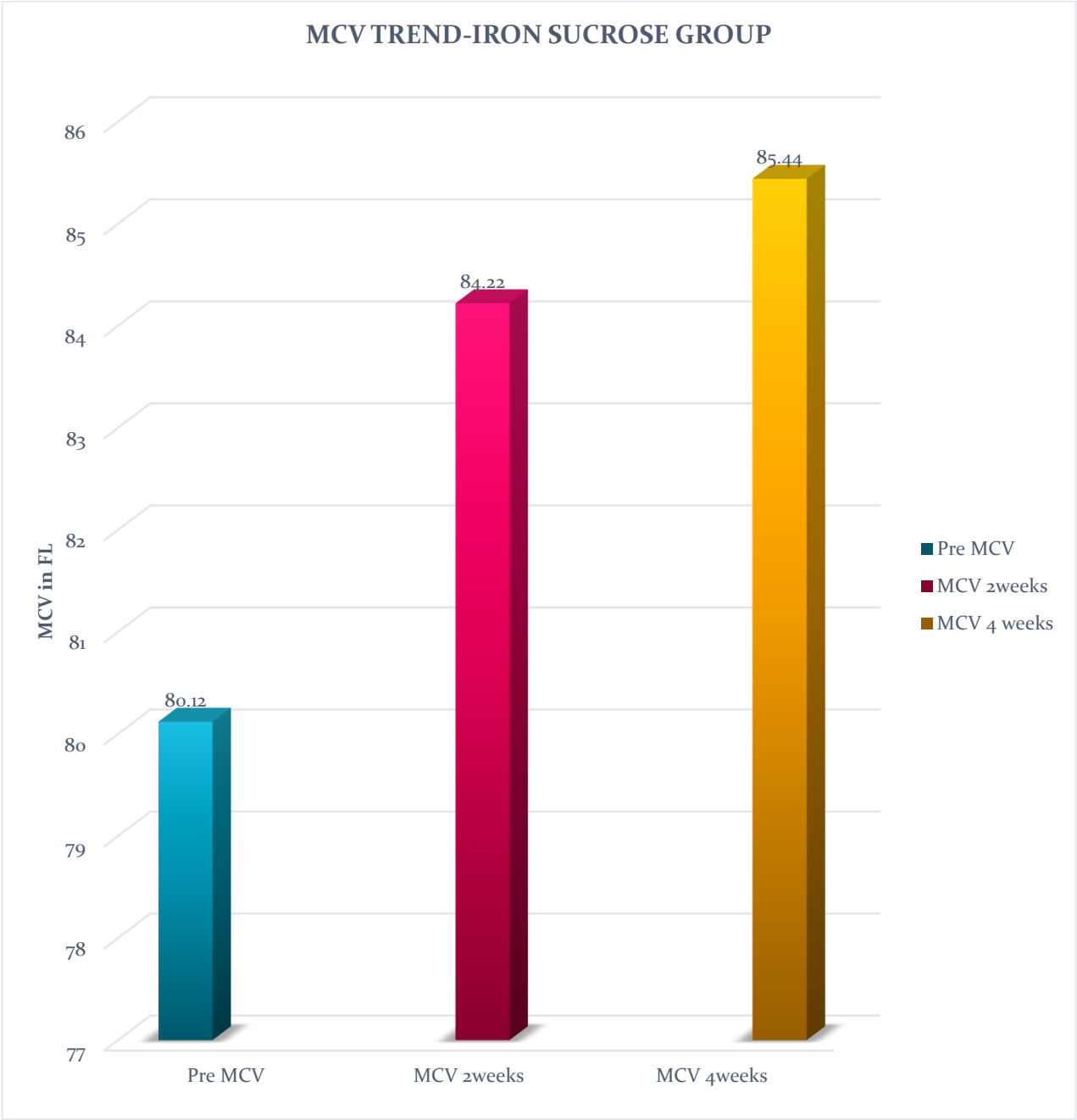
Iron Sucrose	Pre Hb	Hb 2weeks	Hb 4 weeks
Pre Hb	8.57		
Hb 2weeks		10.22	
Hb 4weeks			10.92


Hb TREND -IRON SUCROSE GROUP
Hb in gms
0
2
4
6
8
10
12
8.57
10.22
10.92
Pre Hb
Hb 2weeks
Hb 4weeks

MEAN MCV TREND IN IRON SUCROSE GROUP:

There is also significant increase in the MCV following iron sucrose treatment. Pretreatment MCV is about 80.12(mean value) and 84.22(mean) at two weeks and 85.449(mean) at 4 weeks.

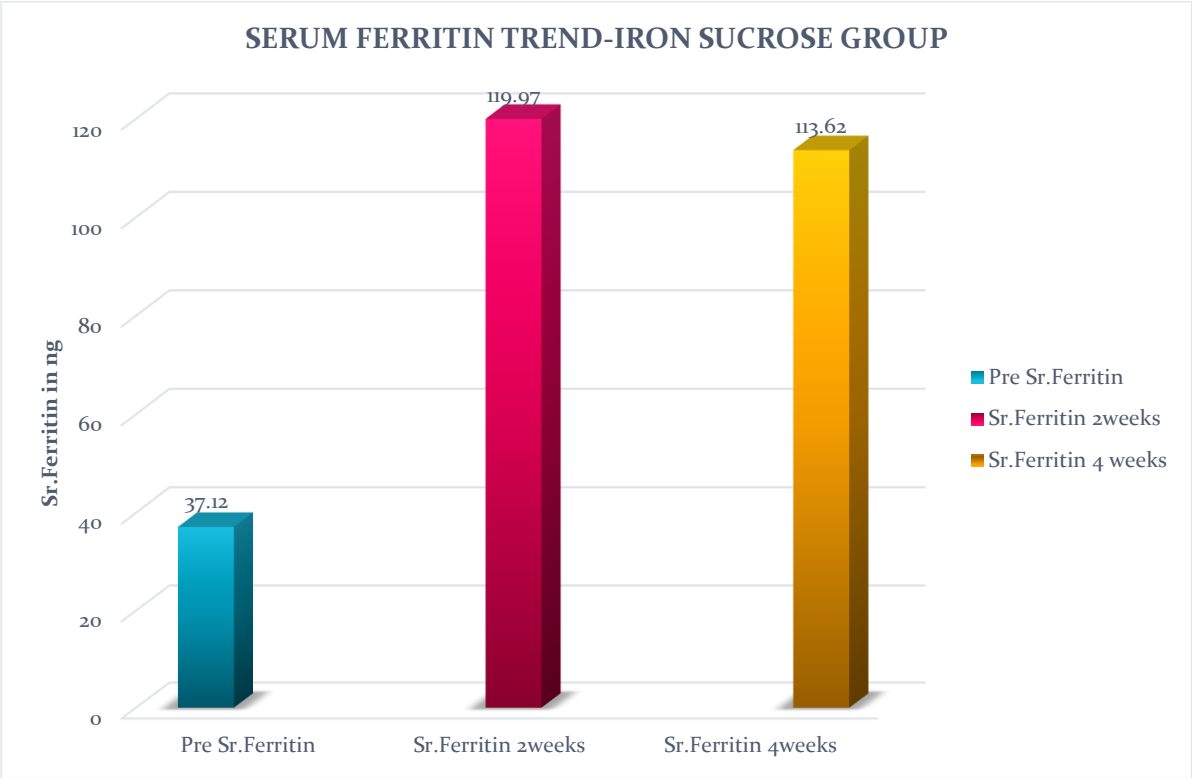
Iron Sucrose	Pre MCV	MCV 2weeks	MCV 4 weeks
Pre MCV	80.12		
MCV 2weeks		84.22	
MCV 4weeks			85.44


MCV TREND-IRON SUCROSE GROUP
MCV in FL
86
85
84
83
82
81
80
79
78
77
80.12
84.22
85.44
Pre MCV
MCV 2weeks
MCV 4weeks
Pre MCV
MCV 2weeks
MCV 4 weeks

MEAN SERUM FERRITIN TREND IN IRON SUCROSE GROUP:

Serum ferritin increases from average of 37.12 ng/ml to 119.97 ng/ml at two weeks and there is a fall at four weeks 113.62ng/ml following redistribution of iron.

Iron Sucrose	Pre Sr.Ferritin	Sr.Ferritin 2weeks	Sr.Ferritin 4 weeks
Pre Sr.Ferritin	37.12		
Sr.Ferritin 2weeks		119.97	
Sr.Ferritin 4weeks			113.62



SIDE EFFECTS:

Among iron sucrose group, 7 persons reported side effects.

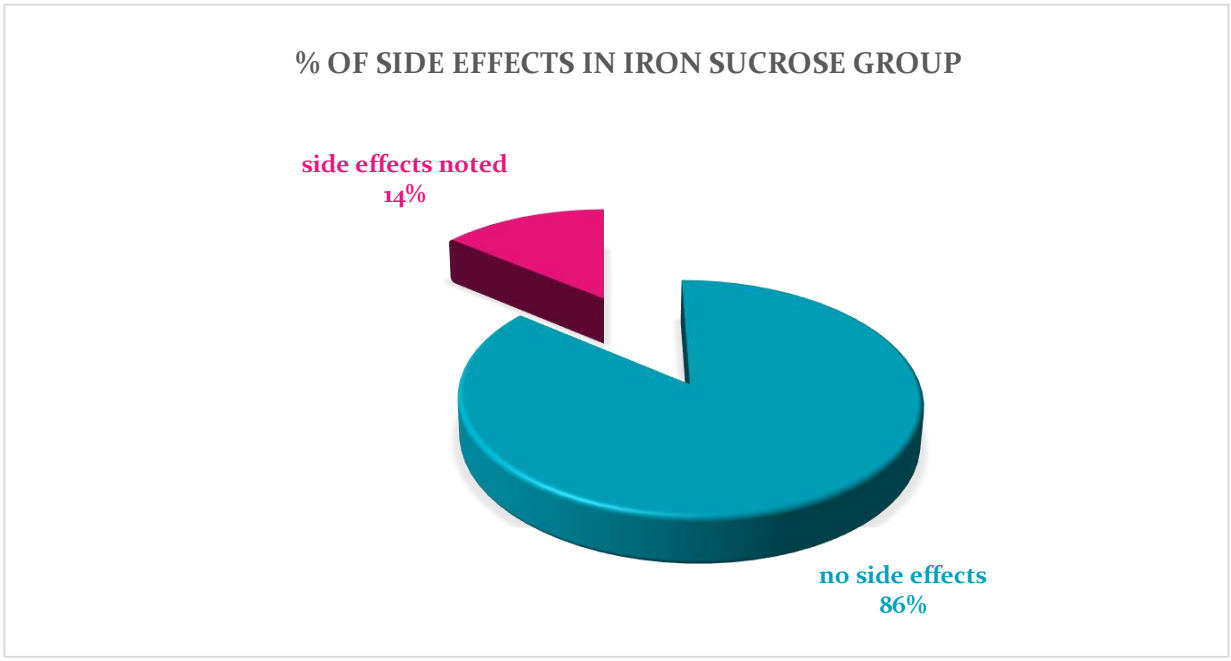
Among side effects:

4 reported nausea with or without vomiting

2 reported giddiness

1 reported urticarial

Total pts	side effects reported	Percentage
50	7	14%



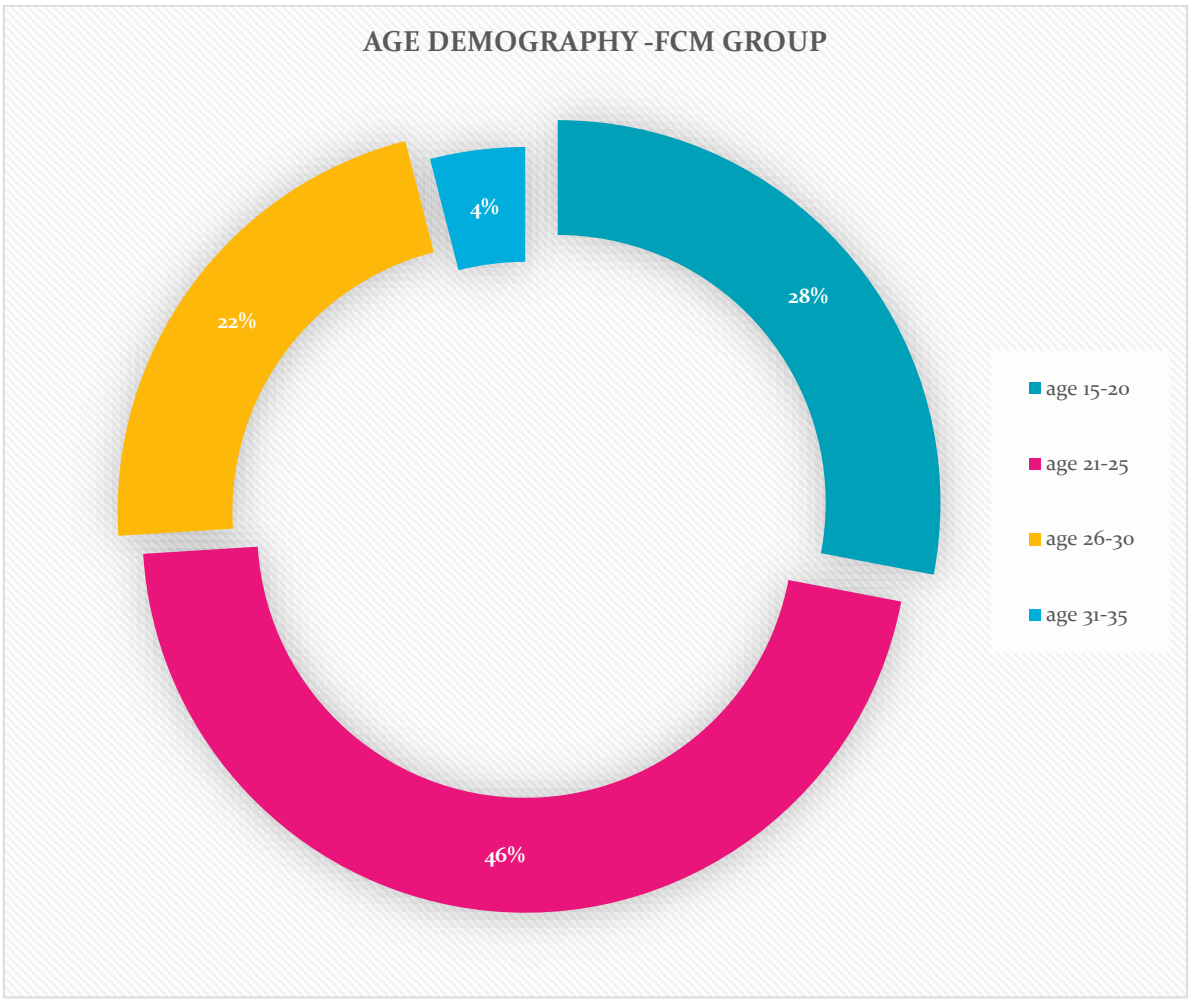
FERRIC CARBOXYMALTOSE (FCM):

Following are the demography features of Ferric Carboxymaltose participants and their outcomes following FCM infusion.

AGE DEMOGRAPHY IN FCM GROUP:

Out of 50 women studied in FCM group 28%(n=14) belong to age group 15-20yrs, 46% belong to age group 21-25yrs(n=23), 22% belong to 26-30 yrs(n=11), 2% belong to 31-35 yrs(n=1).

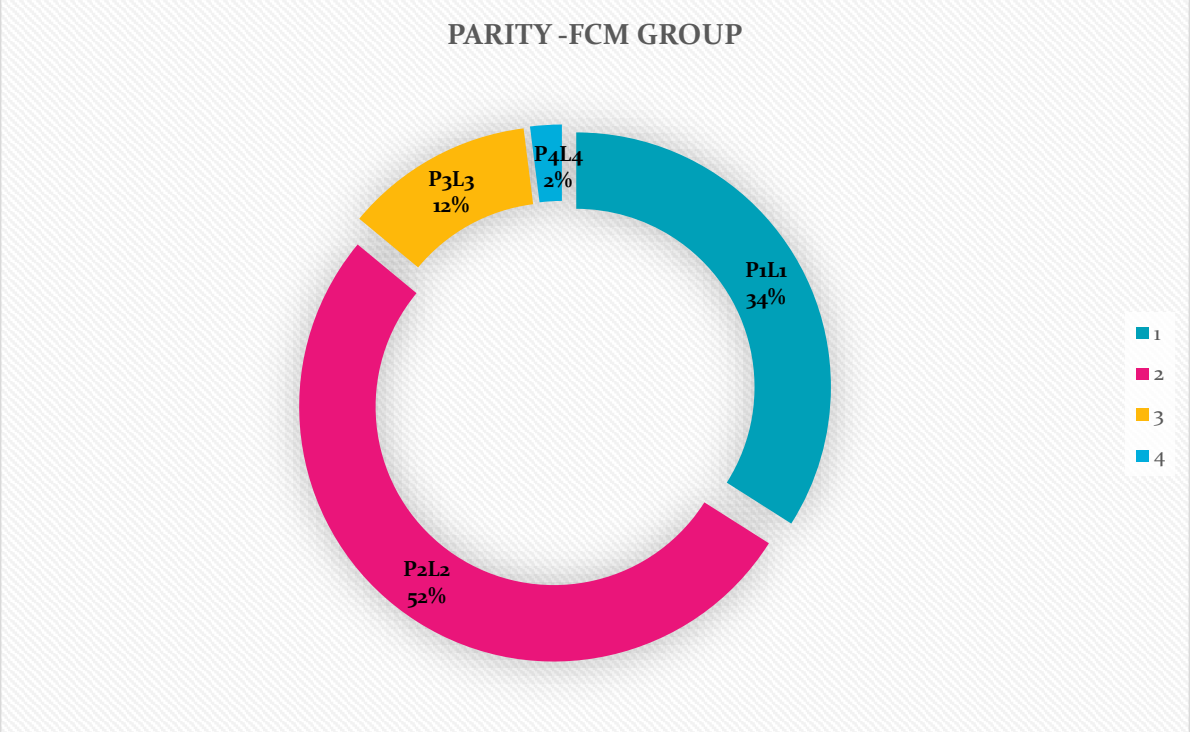
AGE	Count	Percentage
age 15-20	14	28%
age 21-25	23	46%
age 26-30	11	22%
age 31-35	2	4%


AGE DEMOGRAPHY -FCM GROUP
4%
28%
22%
46%
age 15-20
age 21-25
age 26-30
age 31-35

PARITY IN FCM GROUP:

Among parity distributon 44% belong to P2L2, 42% belong to P1L1, 12% belong to P3L3 and 2% belong to P4L4 group. So prevalence of anaemia is more among multiparas

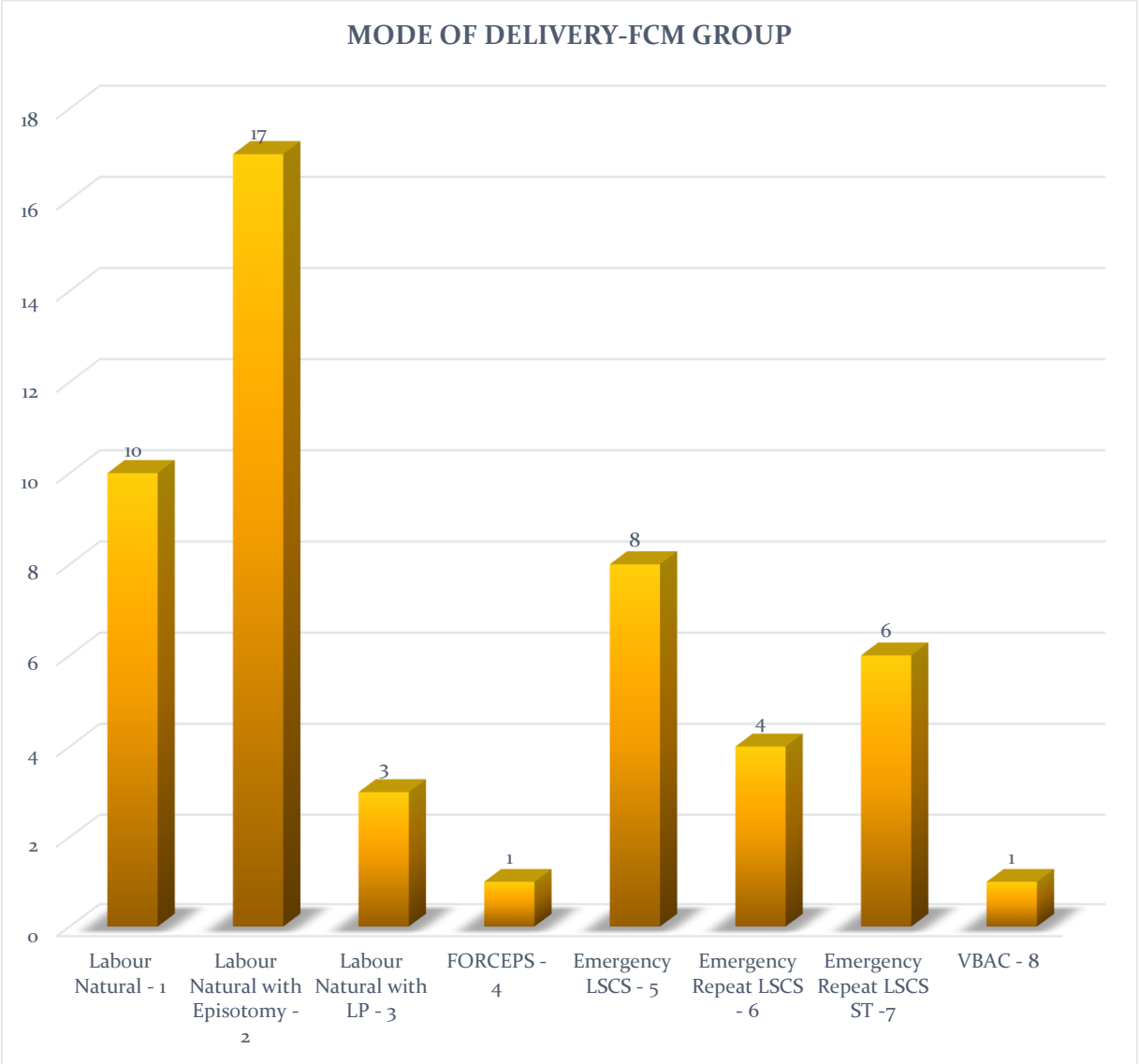
OB.Code	Parity	FCM	Percentage
1	P1L1	17	42%
2	P2L2	26	44%
3	P3L3	6	12%
4	P4L4	1	2%



MODE OF DELIVERY (MOD) IN FCM GROUP:

Among mode of deliveries 30 deliveries were labour natural (includes with episotomy and with lacerated perineum), forceps delivery -1 and emergency lscs- 18(includes repeat lscs and repeat lscs with sterilization) and VBAC 1.

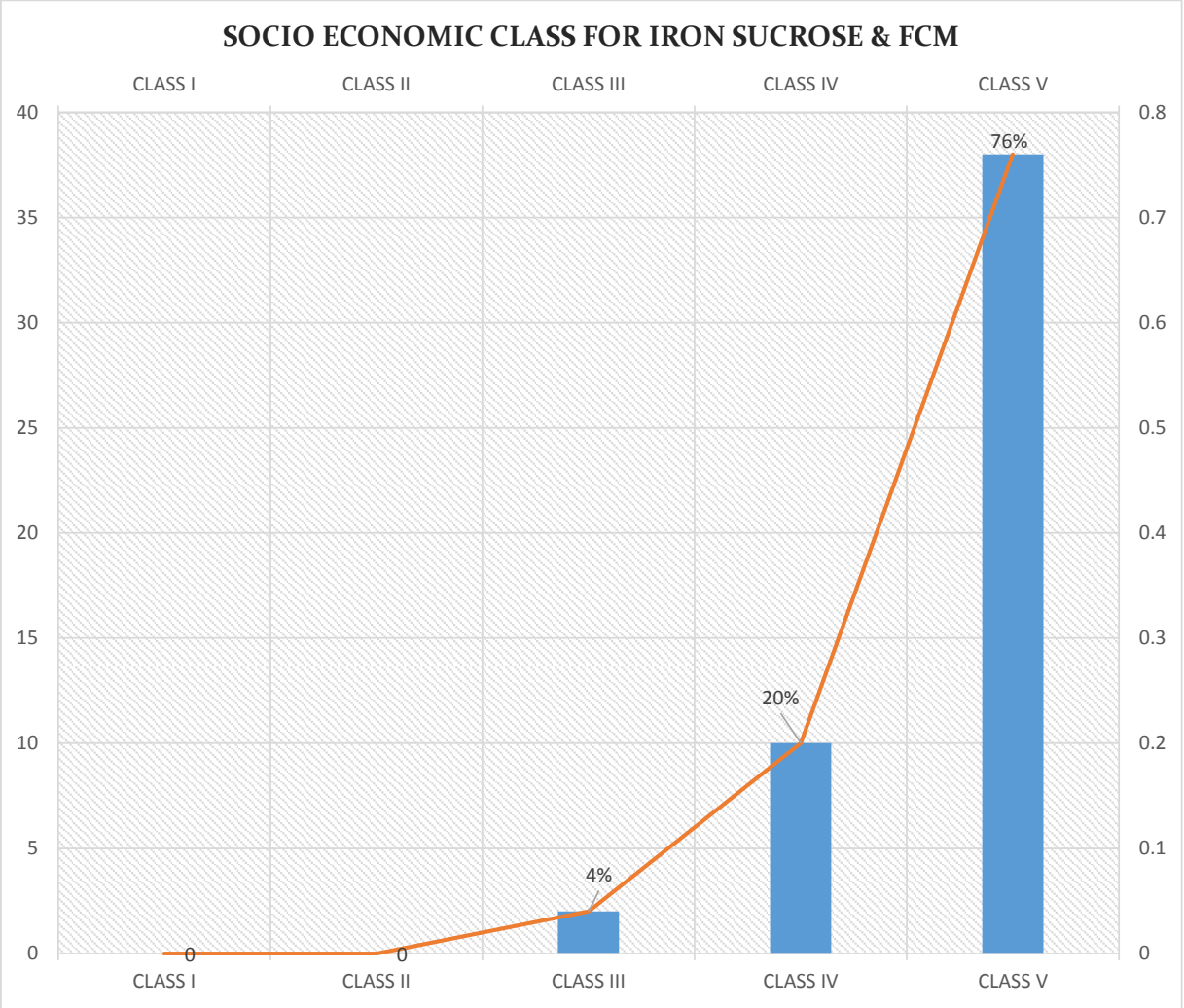
MOD.Code	MOD	FCM
1	Labour Natural	10
2	Labour Natural with Episotomy	17
3	Labour Natural with LP	3
4	FORCEPS	1
5	Emergency LSCS	8
6	Emergency Repeat LSCS	4
7	Emergency Repeat LSCS ST	6
8	VBAC	1


MODE OF DELIVERY-FCM GROUP
18
16
14
12
10
8
6
4
2
0
10
17
3
1
8
4
6
1
Labour Natural - 1
Labour Natural with Episotomy - 2
Labour Natural with LP - 3
FORCEPS - 4
Emergency LSCS - 5
Emergency Repeat LSCS - 6
Emergency Repeat LSCS ST -7
VBAC - 8

SOCIO ECONOMIC STATUS:

The incidence of iron deficiency anaemia is more among low socio economic group for both Iron sucrose and FCM recipients. 76% belong to class V and 20% belong to IV.

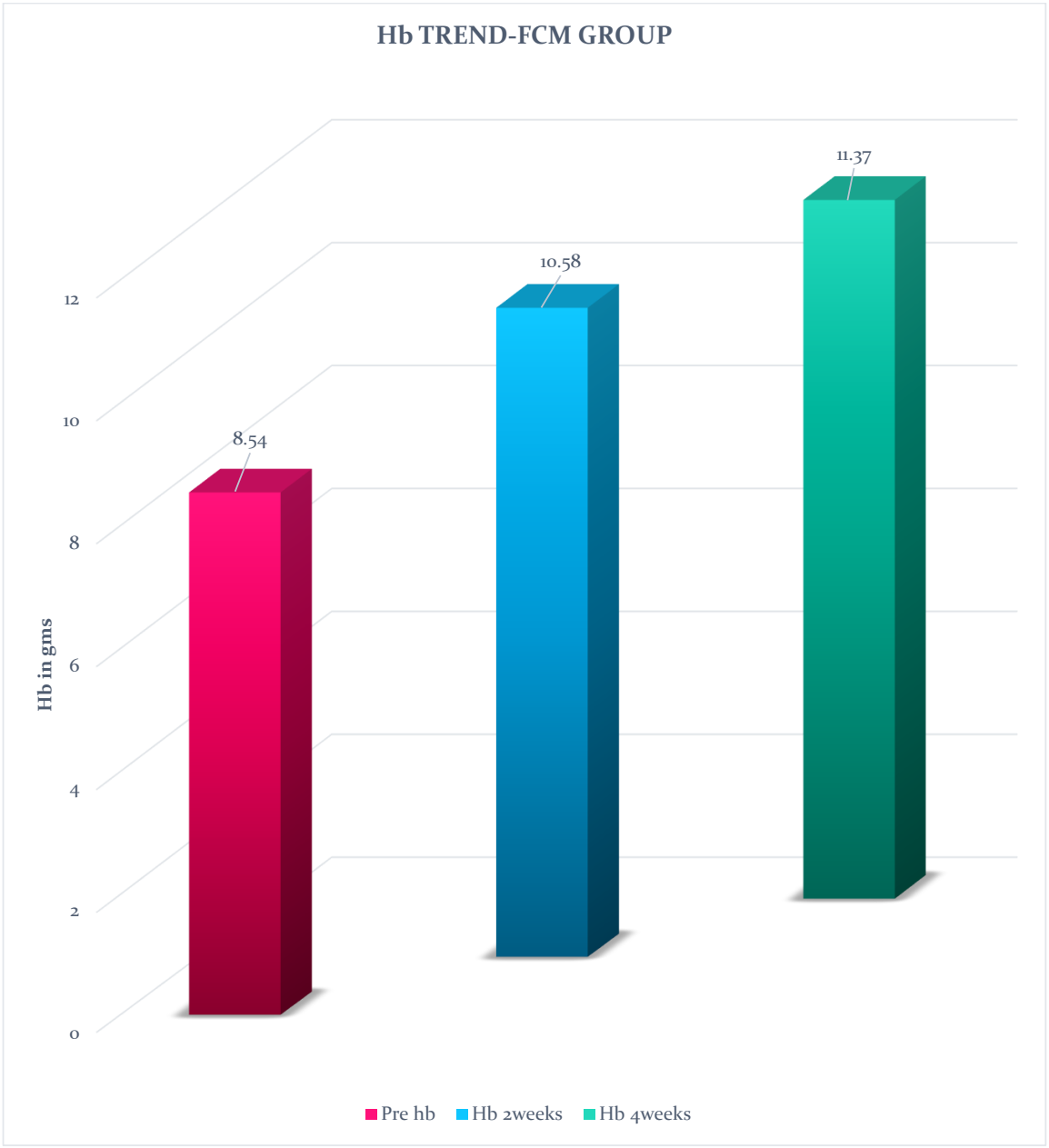
SOCIO ECONOMIC CLASS	Count	Percentage
CLASS I	–	–
CLASS II	–	–
CLASS III	2	4%
CLASS IV	10	20%
CLASS V	38	76%


SOCIO ECONOMIC CLASS FOR IRON SUCROSE & FCM
CLASS I
CLASS II
CLASS III
CLASS IV
CLASS V
40
35
30
25
20
15
10
5
0
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
0
4%
20%
76%
CLASS I
CLASS II
CLASS III
CLASS IV
CLASS V

MEAN HEMOGLOBIN TREND IN FCM GROUP:

Improvement of haemoglobin with iron sucrose from pretreatment haemoglobin (mean) 8.54 gm to 10.58(mean) at two weeks and to 11.37 (mean) at 4 weeks. The average increase is about 2.04 gm at 2 weeks and 2.83gm at 4 weeks.

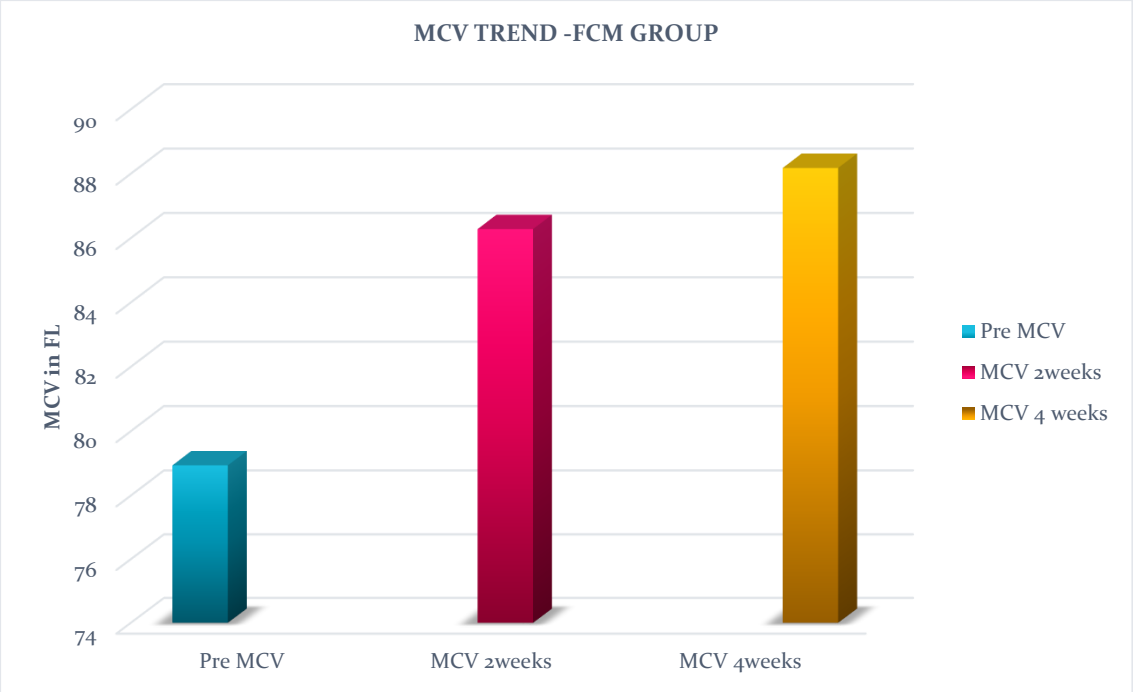
FCM	Pre Hb	Hb 2weeks	Hb 4weeks
Pre hb	8.54		
Hb 2weeks		10.58	
Hb 4weeks			11.37


Hb TREND-FCM GROUP
Hb in gms
0
2
4
6
8
10
12
8.54
10.58
11.37
Pre hb
Hb 2weeks
Hb 4weeks

MEAN MCV TREND IN FCM GROUP:

There is also significant increase in the MCV following iron sucrose treatment. Pretreatment MCV is about 78.94(mean value) and 86.28 (mean) at two weeks and 88.18(mean) at 4 weeks.

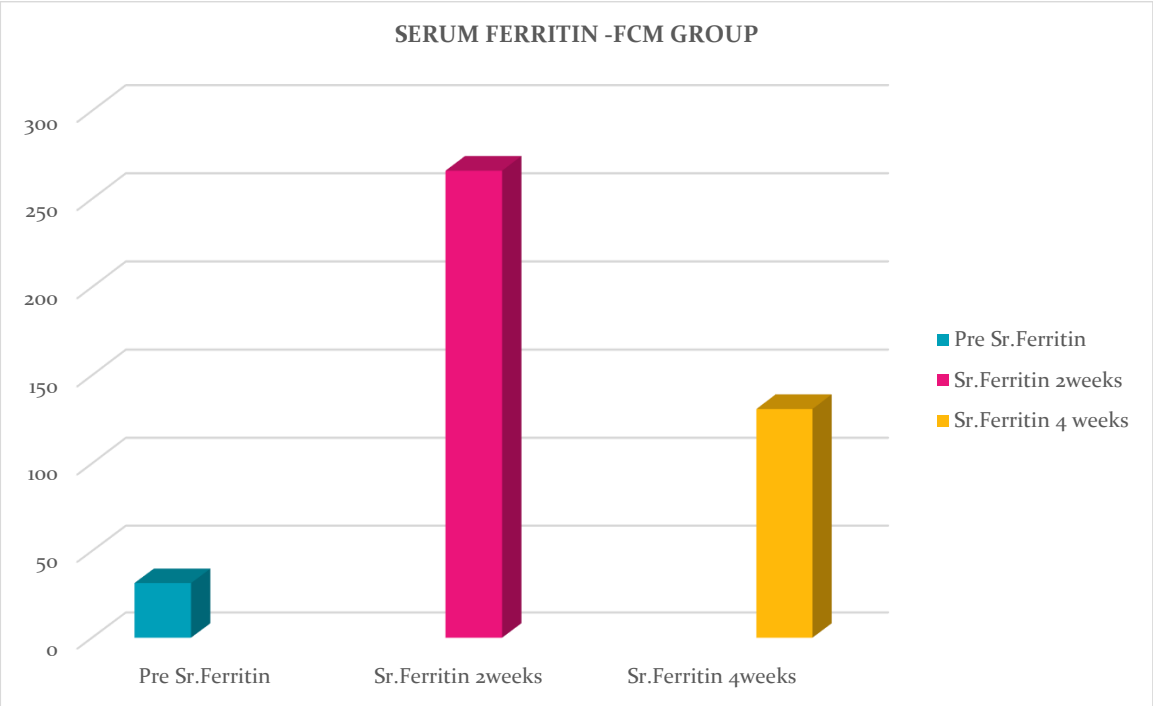
FCM	Pre MCV	MCV 2weeks	MCV 4 weeks
Pre MCV	78.94		
MCV 2weeks		86.28	
MCV 4weeks			88.18



MEAN SERUM FERRITIN TREND IN FCM GROUP:

Serum ferritin increases from average of 31.5 ng/ml to 266.02 ng/ml at two weeks and there is a fall at four weeks 130.86 ng/ml following redistribution of iron

FCM	Pre Sr.Ferritin	Sr.Ferritin 2weeks	Sr.Ferritin 4 weeks
Pre Sr.Ferritin	31.5		
Sr.Ferritin 2weeks		266.02	
Sr.Ferritin 4weeks			130.86



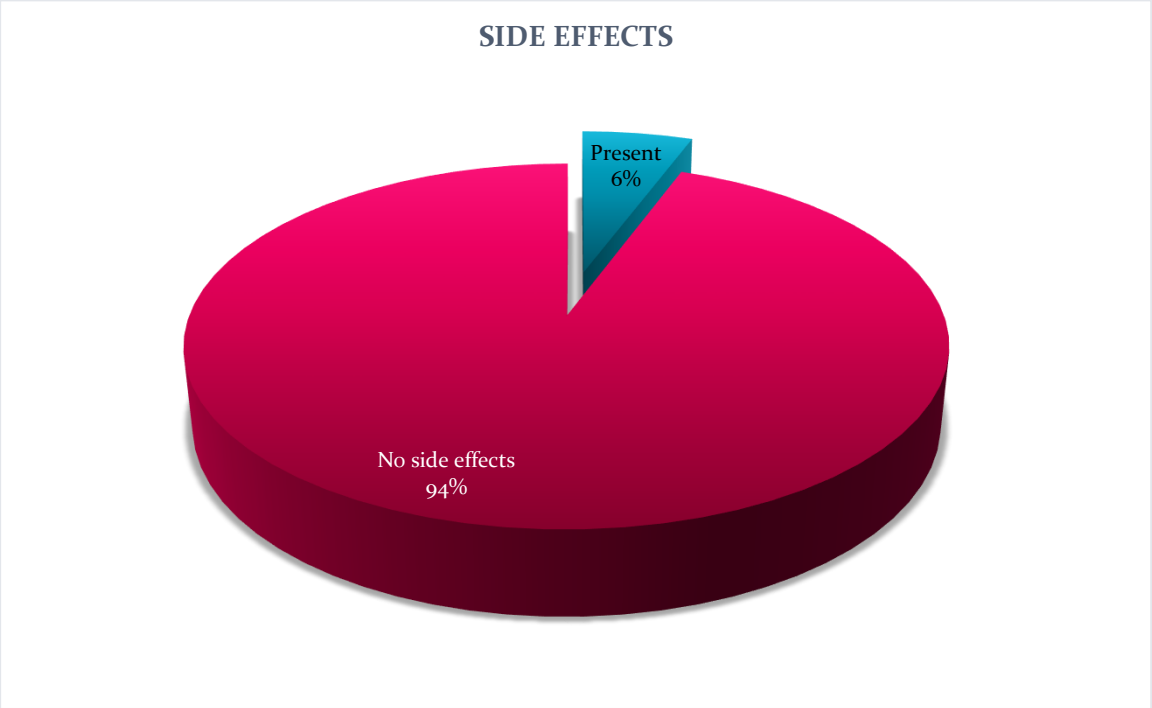
SIDE EFFECTS:

Among FCM group, 3 persons reported side effects.

Among side effects:

- 2 reported nausea
- 1 reported arthralgia

TOTAL	SIDE EFFECTS NOTED	Percentage
50	3	6%



Testing of hypotheses:

Iron sucrose treatment and Ferric carboxy maltose treatment are effective in increasing HB, MCV and Sr. Ferritin. For this I used paired test due to pre and post test scores.

Paired't' test:

This test was adopted to determine the effect of iron sucrose and FCM before and immediately after the intervention. It was calculated by using the formula

$$t = \left| \frac{\bar{d}}{SD/\sqrt{n}} \right|$$

$$SD = \sqrt{\frac{\sum(d-\bar{d})2}{n-1}}$$

d= Mean difference of parameters score before and after therapy.

SD=Standard deviation of individual parameters before and after iron sucrose and FCM

n= Number of sample

Null Hypothesis 1: The Iron sucrose treatment and Ferric carboxymaltose treatment are not effective in increasing HB, MCV and Sr. Ferritin during the first two weeks.

Alternative Hypothesis: The Iron sucrose treatment and Ferric carboxy maltose treatment are effective in increasing HB, MCV and Sr. Ferritin. during the first 2 weeks.

Level of significance α = .05

Test Statistic: $t = \left| \frac{\bar{d}}{SD/\sqrt{n}} \right|$

Calculations:

Paired Samples Statistics

Treatment Code			Mean	N	Std. Deviation	Std. Error Mean
Iron Sucrose	*Pair 1*	*Pre_Hb*	*8.572*	*50*	*.3933*	*.0556*
		Post2HB	*10.22*	*50*	*.548*	*.077*
	Pair 2	*Pre_MCV*	*80.12*	*50*	*3.081*	*.436*
		Posrt2MCV	*84.22*	*50*	*2.332*	*.330*
	Pair 3	*PreSr.Ferritin*	*37.118*	*50*	*9.5262*	*1.3472*
		Post2Sr.Ferritin	*199.9716*	*50*	*50.92968*	*7.20255*
FCM	Pair 1	Pre_Hb	8.536	50	.4746	.0671
		Post2HB	10.58	50	.448	.063
	Pair 2	Pre_MCV	78.94	50	4.460	.631
		Posrt2MCV	86.28	50	2.090	.296
	Pair 3	PreSr.Ferritin	31.504	50	12.6092	1.7832
		Post2Sr.Ferritin	261.0304	50	70.26405	9.93684

Paired Samples Test

TreatmentCode			Paired Differences					t	df	*Sig. (2-tailed)*
			Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
						Lower	Upper			
	Pair 1	*Pre_Hb - Post2HB*	*-1.6480*	*.5342*	*.0756*	*-1.7998*	*-1.4962*	*-21.813*	*49*	*.000*
Iron Sucrose	*Pair 2*	*Pre_MCV - Posrt2MCV*	*-4.100*	*2.073*	*.293*	*-4.689*	*-3.511*	*-13.988*	*49*	*.000*
	Pair 3	*PreSr.Ferritin - Post2Sr.Ferritin*	*-162.85360*	*41.6030*	*5.88356*	*-174.67706*	*-151.03014*	*-27.679*	*49*	*.000*
	Pair 1	Pre_Hb - Post2HB	-2.0420	.3671	.0519	-2.1463	-1.9377	-39.338	49	.000
FCM	Pair 2	Pre_MCV - Posrt2MCV	-7.340	3.514	.497	-8.339	-6.341	-14.768	49	.000
	Pair 3	PreSr.Ferritin - Post2Sr.Ferritin	-229.52640	58.2314	8.23517	-246.07560	-212.97720	-27.871	49	.000

Inference: The p- values for all the combinations are .000, Since p- values are less than the level of significance α=.05, Null hypotheses are rejected and Alternative hypotheses are accepted

Conclusion: This shows that the Iron sucrose treatment and Ferric carboxy maltose treatment are effective in increasing HB, MCV and Sr. Ferritin during the first two weeks.

A similar analysis is carried to know whether the same trend continues between the 2nd and the 4th week of the treatment.

Null Hypothesis 2: The Iron sucrose treatment and Ferric carboxymaltose treatment are not effective in increasing HB, MCV and Sr. Ferritin during the 2nd and the 4th week of the treatment.

Alternative Hypothesis: The Iron sucrose treatment and Ferric carboxic malthouse treatment are effective in increasing HB, MCV and Sr. Ferritin. during the the 2nd and the 4th week of the treatment.

Level of significance $\alpha = .05$

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Post2HB	10.40	100	.529	.053
	Post4Hb	11.145	100	.6445	.0645
Pair 2	Posrt2MCV	85.25	100	2.434	.243
	Post4MCV	86.81	100	2.286	.229
Pair 3	Post2Sr.Ferritin	230.5010	100	68.32905	6.83290
	Post4Sr.Ferritin	122.237	100	2.286	23.4

		Mean	Std. Deviation	Std. Error mean	t	df	Sig. (2-tailed)
Pair 1	Post2HB - Post4Hb	-.7460	.3421	.0342	-21.804	99	.000
Pair 2	Posrt2MCV - Post4MCV	-1.560	1.343	.134	-11.613	99	.000
Pair 3	Post2Sr.Ferritin - Post4Sr.Ferritin	108.26390	55.13744	5.51374	19.635	99	.000

Inference: The p- values for all the combinations are .000. Since p- values are less than the level of significance α=.05, Null hypotheses are rejected and Alternative hypotheses are accepted

Conclusion: This shows that the Iron sucrose treatment and Ferric carboxy maltose treatment are effective in increasing HB, MCV and Sr. Ferritin during the 2nd and the 4th week of the treatment.

Combining the results of these two tests we conclude that Iron sucrose treatment and Ferric carboxymaltose treatment are effective in increasing HB, MCV and Sr. Ferritin.

Independent t test: This test is used to identify whether there is significant difference in in Hb, MCV and Sr Ferritin between Iron sucrose treatment group and Ferric carboxymaltose treatment group. It is calculated by using following methods,

$$t = \frac{|x_1 - x_2|}{SD\sqrt{\frac{1}{n_1} + \frac{1}{n_2}}}$$

$$SD = \sqrt{\frac{(n_1 - 1)(SD_1)^2 + (n_2 - 1)(SD_2)^2}{n_1 + n_2 - 2}}$$

x_1 = Mean value of Iron sucrose treatment group

x_2 = Mean value of Ferric carboxymaltose treatment group

S = Standard deviation of Iron sucrose treatment group and Ferric carboxymaltose treatment group

n_1 = Number of samples in Iron sucrose treatment group

n_2 = Number of samples in the Ferric carboxymaltose treatment group

Null hypothesis: There is no significant difference in Hb, MCV and Sr Ferritin between Iron sucrose treatment group and Ferric carboxymaltose treatment group.

Alternative Hypothesis: Ferric carboxymaltose treatment is better than Iron sucrose treatment.

Level of significance $\alpha = .05$

Calculations:

COMPUTE Increase in HB = Post2Hb - Pre_Hb.

Increase in MCV2 = Posrt2MCV - Pre_MCV

Increase in changeSr.Fer2 = Post2Sr.Ferritin - Pre Sr.Ferritin

Group Statistics

	Treatment Code	N	Mean	Std. Deviation	Std. Error Mean
increase in HB 2weeks	Iron sucrose	50	1.6480	.53423	.07555
	FCM	50	2.0420	.36706	.05191
increase in MCV 2weeks	Iron sucrose	50	4.1000	2.07266	.29312
	FCM	50	7.3400	3.51446	.49702
increase in Sr Ferritin 2weeks	Iron sucrose	50	162.8536	41.603036	5.88356
	FCM	50	229.5264	58.231446	8.23517

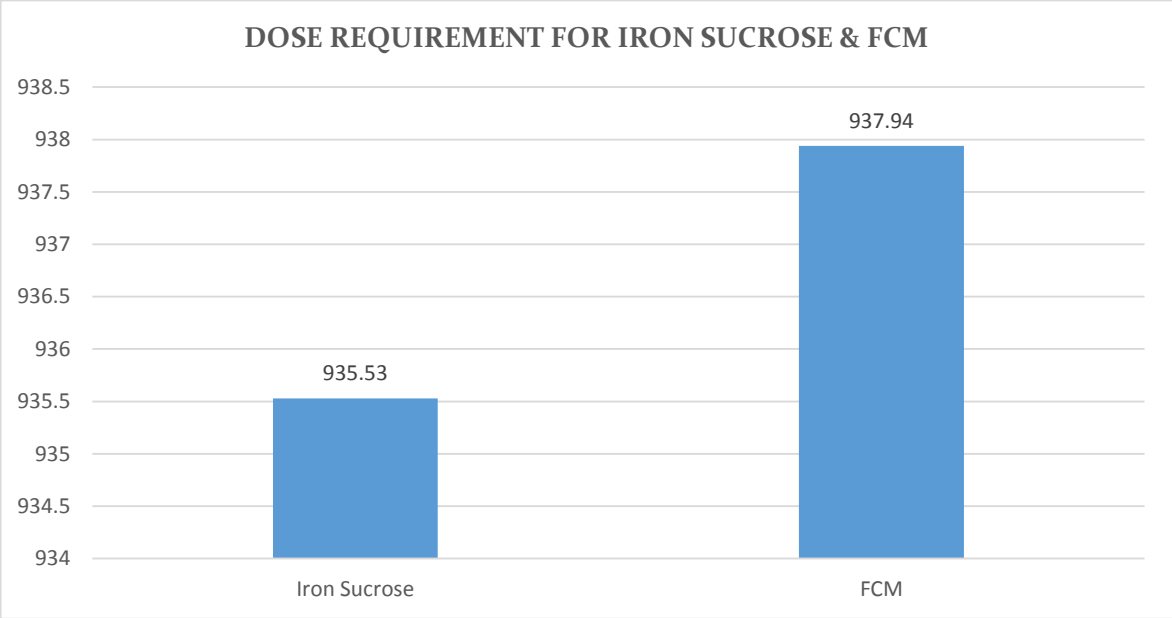
		Levene's Test for Equality of Variances				
		F	Sig.	t	df	Sig. (2-tailed)
increase in HB 2weeks	Equal variances assumed	7.416	.008	-4.298	98	.000
	Equal variances not assumed			-4.298	86.832	.000
increase in MCV 2weeks	Equal variances assumed	15.519	.000	-5.615	98	.000
	Equal variances not assumed			-5.615	79.407	.000
increase in Sr Ferritin 2weeks	Equal variances assumed	14.256	.000	-6.588	98	.000
	Equal variances not assumed			-6.588	88.683	.000

Inference: Since p- values (.000) are less than α (=.05), the null hypotheses are rejected consequently alternative hypotheses are accepted.

Conclusion: Ferric carboxymaltose treatment is better than Iron sucrose treatment. The treatments behave over the periods, after 2 weeks and after 4 weeks. This is done with the help of the technique **Mean plots.**

DOSE REQUIREMENT FOR IRON SUCROSE & FCM:

	IRON SUCROSE	FCM
DOSE	935.53	937.94



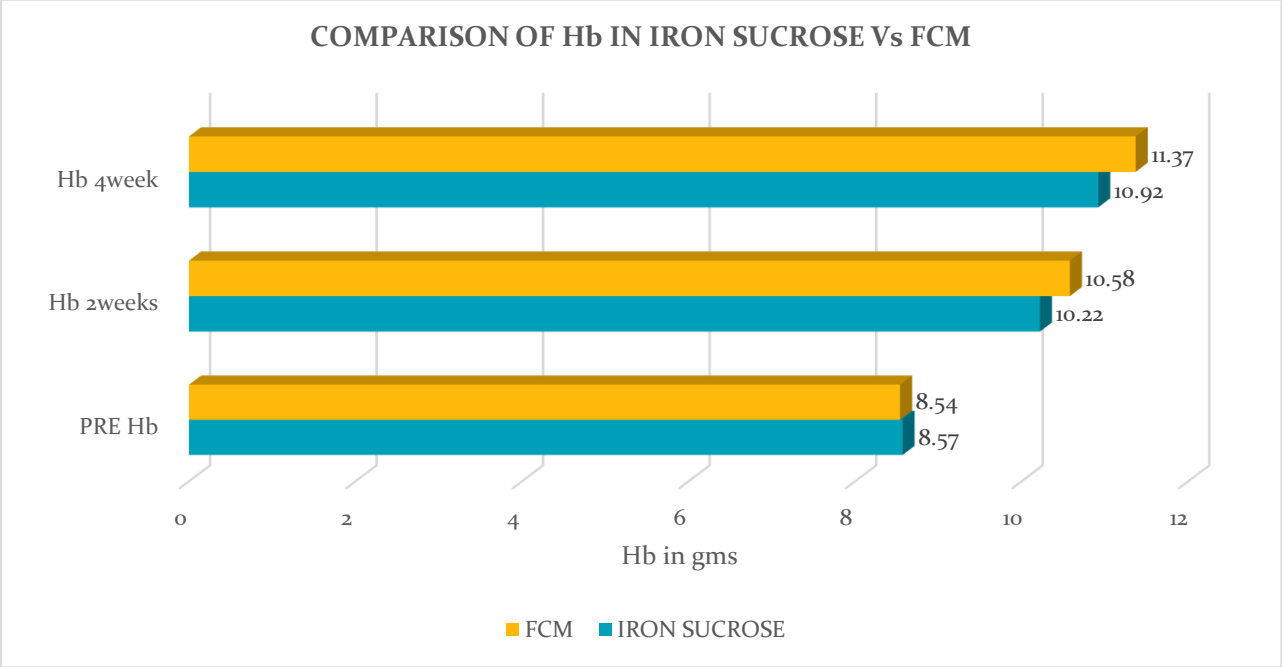
The mean iron dose required in both iron sucrose and FCM group is about 1000mg.

COMPARISON OF EFFICACY OF IRON SUCROSE vs FCM USING MEAN PLOT:

COMPARISON OF Hb IN IRON SUCROSE vs FCM:

In our study the trend of haemoglobin increase in FCM group is faster and greater .the haemoglobin increase is about 10.92 at 4 weeks in iron sucrose group from baseline value of 8.57g where as in FCM group Hb increase is about 11.37 from 8.54.

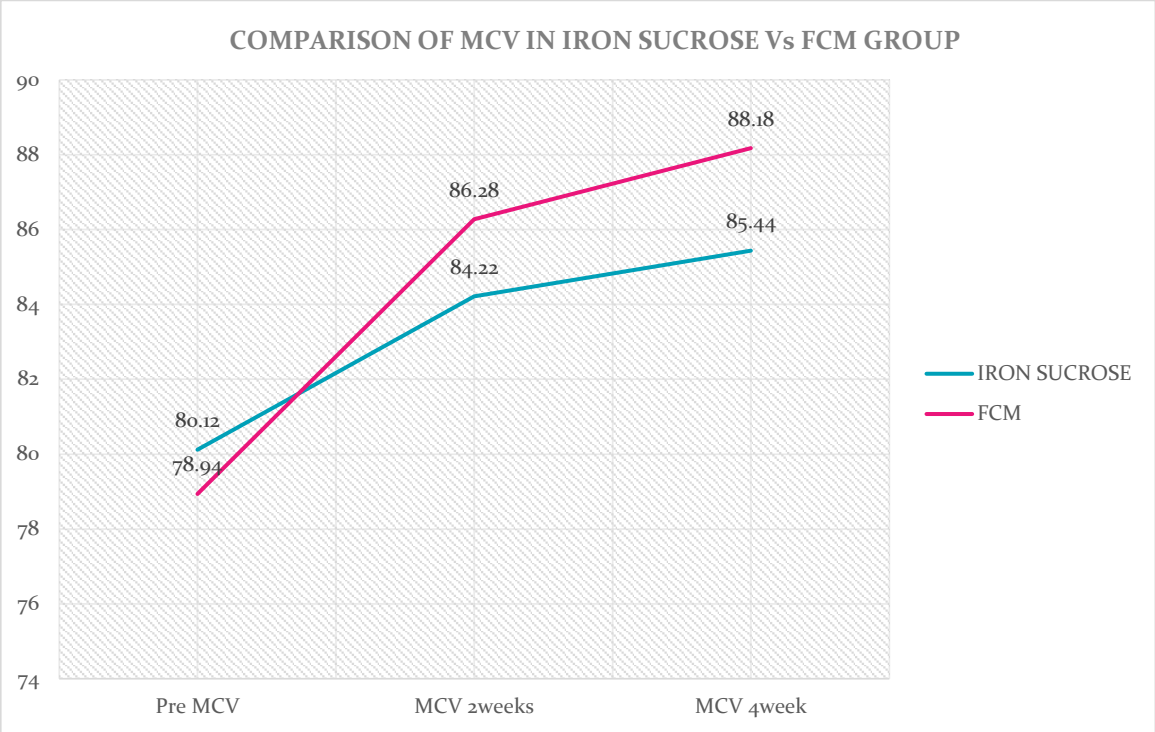
	IRON SUCROSE	FCM
PRE Hb	8.57	8.54
Hb 2weeks	10.22	10.58
Hb 4week	10.92	11.37



COMPARISON OF MCV - IRON SUCROSE Vs FCM:

Blood indices also improves following intravenous iron formulations. MCV increases about 85.44(fl) from baseline of 80.12(fl) in iron sucrose group whereas in FCM it increases to about 88.18(fl) from 78.94.so there is significant improvement in blood indices following FCM injection….

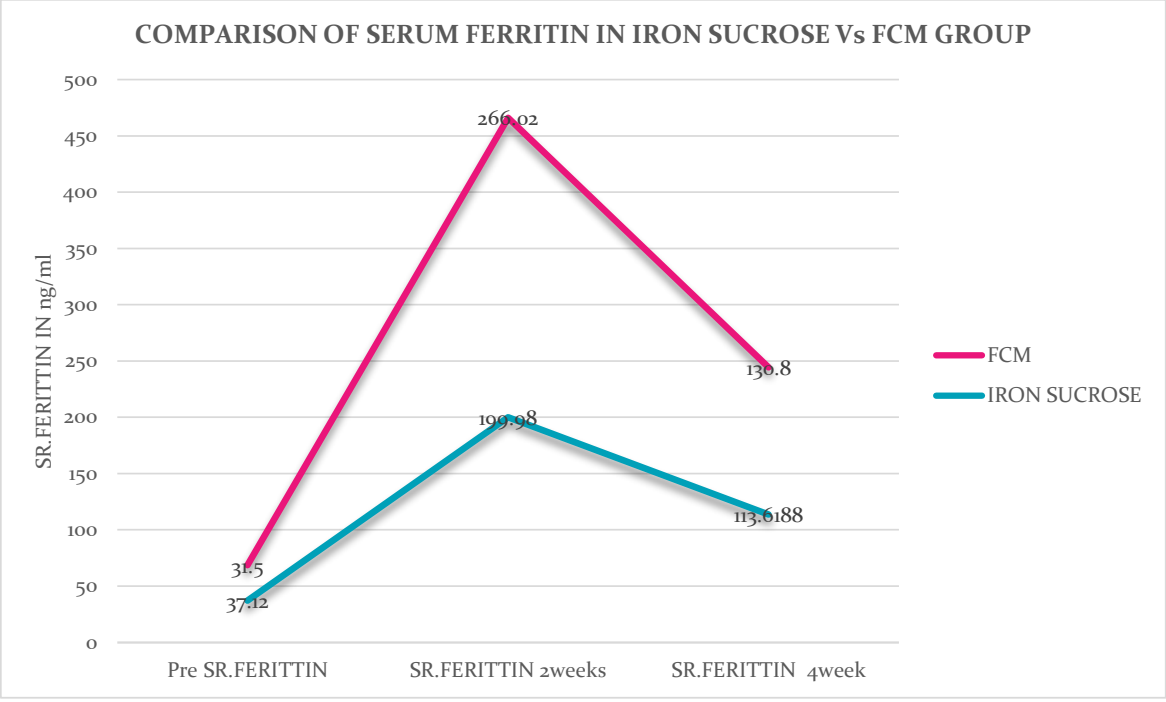
	IRON SUCROSE	FCM
Pre MCV	80.12	78.94
MCV 2weeks	84.22	86.28
MCV 4week	85.44	88.18



COMPARISON OF SR.FERRITIN - IRON SUCROSE Vs FCM:

In our study among iron sucrose group serum ferritin increases from baseline (37.2 to 199.98 at 2weeks; 113.61 at 4 weeks).But the inmprovement is greater among FCM GROUP.(31.5 to 266.02 at 2 weeks and 130.8 at 4 weeks).the ferritin fall at 4th week is due to iron redistribution in haemoglobin formation.

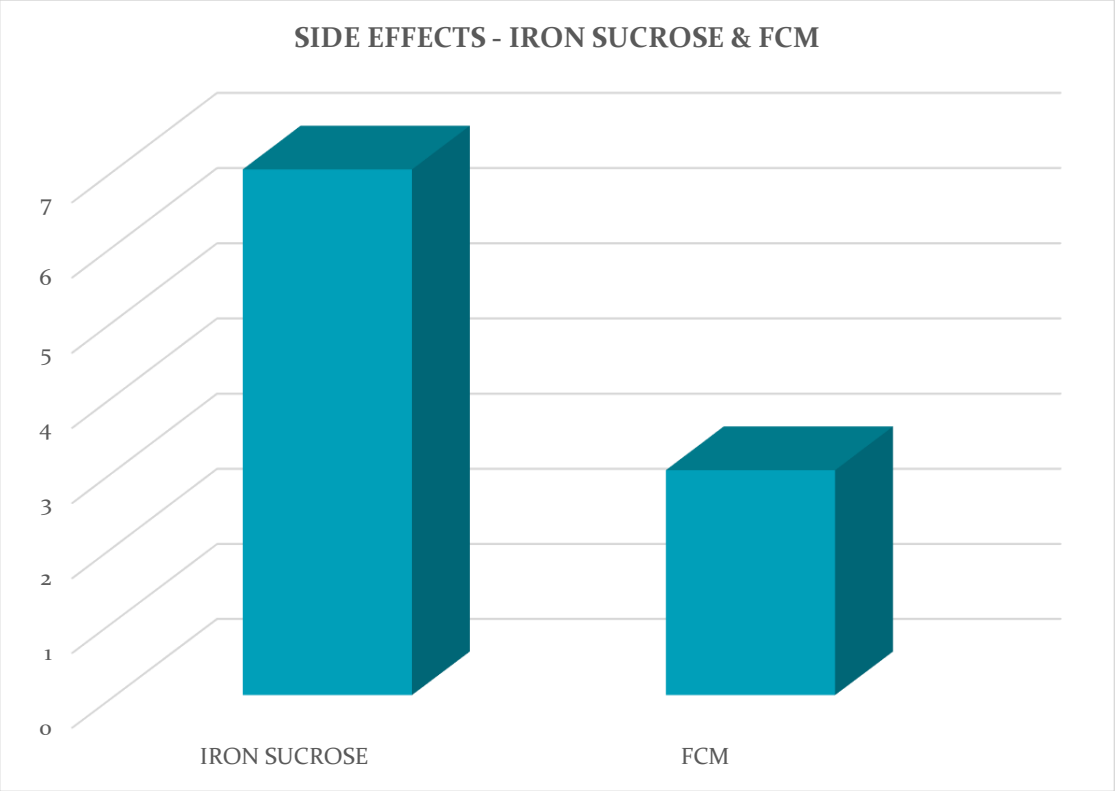
	IRON SUCROSE	FCM
Pre SR.FERITTIN	37.12	31.5
SR.FERITTIN 2weeks	199.98	266.02
SR.FERITTIN 4week	113.6188	130.8



COMPARISION OF SIDE EFFECTS:

Adverse reactions following both groups were milder .Following iron sucrose 14%(n=7) developed side effects like nausea, vomiting, urticaria whereas in FCM group only 6% developed nausea,giddiness and arthralgia.

IRON SUCROSE	FCM
7	3



DISCUSSION

DISCUSSION

Post-partum iron deficiency anaemia can be treated by oral and intravenous iron. With both of these iron therapies hemoglobin levels increase by 2.4 to 4.6 gm/dl. David B et al study has shown increase in hemoglobin levels by 2 gm/dl within 7 days and 4gm/dl within 2-4 weeks in patients receiving ferric carboxymaltose. Serum ferritin also increased promptly in IV FCM patients.

Giannoulis et al reported increase of hemoglobin by 4-6 gm/dl in patients receiving iron sucrose. Setu Rathod et al has showed increased in hemoglobin 0f about 2.4gm/dl and 3.4 gm/dl at 2 weeks and 6 weeks respectively. In our study hemoglobin level increased by 1.65 gm/dl;2.35g/dl in iron sucrose group and 2.04 gm/dl;2.83g/dl from FCM group at 2 weeks and 4 weeks of post treatment.

Seid et al reported that ferritin level increases in 6 weeks about 238 ng/ml in FCM group while there was reported that an increase in serum ferritin in oral iron group as 21ng/ml. Christian breymann et al serum ferritin from 39.9 ng/ml from baseline to 568.2

ng/ml at week 1, 161.2ng/ml at 12 weeks (p<.001 when compared to margin increase in ferritin level with ferrous sulphate group 32.4 from baseline to 34.8ng/ml and 43.3ng/ml at 2 weeks and 12 weeks respectively. Prasanth S Kharde et al showed that mean increase in serum ferritin level from11.47ng/ml from baseline to 47.69 ng/ml at 2 weeks and 53.47 ng/ml at 6 weeks. In our study the mean improvement in serum ferritin level from baseline of 37.12 ng/ml to 113.61 ng/ml at 4 weeks following iron sucrose and among FCM group there is improvement from31.5 ng/ml of baseline to 130.8 ng/ml at 4 weeks.

Van Wyck et al has shown a significant increase in MCV among FCM patients. Dede et al had shown increase in mean MCV of 33.3 fl from pretreatment following iron sucrose therapy. Khurshid shabir Raja et al had shown a mean increase in MCV 0f 10 fl from baseline. Our study shows mean improvement in MCV following iron sucrose therapy is from 80.12 fl to 85.44 fl at 4 weeks and among FCM group the mean increase is 88.18 fl at 4 weeks from 78.94 fl.

There is no serious adverse reactions among the iron sucrose as well as FCM groups. The incidence of adverse effects reported so far is between 6.8% and 24.2%. in our study there is milder adverse reactions like nausea, giddiness and urticarial was reported with incidence of 6% among FCM group and 14% in iron sucrose group.

SUMMARY

SUMMARY

In this study I randomly selected 100 postnatal women with IRON DEFICIENCY ANAEMIA with haemoglobin level between 8 to 10 gm. Iron deficit is calculated using formula with the aim of target haemoglobin 12gm.After getting the consent, and after explaining the risks and benefits, 50 postnatal women received iron sucrose and 50 women received FCM. The dose required is almost 1000mg in both the groups. For iron sucrose per sitting only 200mg can be administered. So patients in iron sucrose group needs at least five visits to receive the required dose. Whereas in FCM group 1000 mg can be administered as a single dose. The outcomes are compared and analysed using paired T test and by Independence T test. The investigations used are haemoglobin, serum ferritin, blood indices, and peripheral smear before and after treatment. The results are summarized below.

In age and parity the incidence of iron deficiency anaemia in both groups were comparable and is more among

young age group (15-25) and in multiparas .People belonging to low socioeconomic group is also at increased risk.

The average increase in haemoglobin in FCM group is faster and greater. The average haemoglobin increase is about 10.92(2.35g/dl) at 4 weeks in iron sucrose group from baseline value of 8.57g where as in FCM group Hb increase is about 11.37(2.83g/dl) from 8.54. MCV increases about 85.44(fl) from baseline of 80.12(fl) in iron sucrose group whereas in FCM it increases to about 88.18(fl) from 78.94.So there is significant improvement in blood indices following FCM injection.Among iron sucrose group serum ferritin increases from baseline (37.2 to 199.98 at 2weeks; 113.61 at 4 weeks). But the improvement is greater among FCM GROUP.(31.5 to 266.02 at 2 weeks and 130.8 at 4 weeks).the ferritin fall at 4^{th} week is due to iron redistribution in haemoglobin formation.

Adverse reactions following both groups were milder .Following iron sucrose 14%(n=7) developed side effects like nausea, vomiting, urticaria whereas in FCM group only 6% developed nausea, giddiness and arthralgia.

CONCLUSION

CONCLUSION

This study compared the efficacy of ferric carboxymaltose over iron sucrose in the management of postnatal iron deficiency anaemia.Though our results showed improvement in hemoglobin, serum ferritin, and blood indices in both iron sucrose and FCM group but it was faster and greater with ferric caboxymaltose when compared with iron sucrose. Other advantages are more dose can be administered at a single visit and the hospitalization duration of the patients are reduced greater. The quality of life is also better with FCM group. FCM lacks dextran and less immunogenic so adverse reactions are also low. So out of two intravenous iron FCM seems to be clinically better and statistically significant than iron sucrose in treatment of postnatal iron deficiency anaemia

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BIBLIOGRAPHY

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ANNEXURES

ANNEXURES

ABBREVIATIONS:

IS – Iron Sucrose

FCM – Ferric carboxymaltose

TIBC – Total iron binding capacity

Tfr – Transferrin

ZPP – Zinc protoporphyrin

MCV - Mean Corpuscular Volume.

MCH – Mean Corpuscular Haemoglobin

MCHC – Mean Corpuscular Haemoglobin Concentration.

PCV – Packed cell volume.

IDA – Iron Deficiency Anaemia.

Hb – Haemoglobin

JVP – Jugular Venous Pulse

ICMR – Indian Council of Medical Research

SD – Standard Deviation

LP – Lacerated Perineum

VBAC – Vaginal Birth After Caesarean

LSCS – Lower Segment Caserean Section

ST – Sterilisation

IV – Intravenous.

WHO – World Health Organisation.

KEY TO MASTER CHART:

MOD – Mode of Delivery

P1L1 – Para 1, Live 1

PS – Peripheral Smear

M.H.A-Microcytic Hypochromic Anaemia.

SE – Side Effects

P – Present

A- Absent

PROFORMA:

DATE:

NAME: AGE:

IP NO:

SOCIOECONOMIC CLASS:

RELIGION:

OCCUPATION:

DOA:

DOS:

DOD:

ADDRESS & CONTACT NO:

PRESENTING COMPLAINTS:

MENSTRUAL HISTORY:

MARITAL HISTORY:

OBSTETRIC HISTORY:

CONTRACEPTION HISTORY:

PAST MEDICAL HISTORY:

PAST SURGICAL HISTORY:

ANY MEDICATIONS:

FAMILY HISTORY:

GENERAL EXAMINATION:

VITALS: PULSE RATE

RESPIRATORY RATE

BLOOD PRESSURE

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

ABDOMINAL EXAMINATION:

PER VAGINAL EXAMINATION:

INVESTIGATIONS:

COMPLETE BLOOD COUNT:

PERIPHERAL SMEAR: SR.FERRITIN:

CONSENT FORM

I agree to participate in the study entitled "COMPARATIVE STUDY OF INTRAVENOUS FERRIC CARBOXYMALTOSE AND INTRAVENOUS IRON SUCROSE IN THE MANAGEMENT OF POSTNATAL IRON DEFICIENCY ANAEMIA"

I confirm that I have been told about this study in my mother tongue and have had the opportunity to clarify my doubts.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reasons and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from this study.

Name of the participant:

Sign / Thumb print:

Name of the investigator: Dr. V.Arulmozhi

Sign of Investigator:

<u>சுய ஒப்புதல் படிவம்</u>

இரத்த சோகை உள்ள பிரசவித்த தாய்மார்களுக்கு ∴ பெரிக் கார்பாக்ஸி மால்டோஸ் மற்றும் ஐயர்ன் சுக்ரோஸ் ஆகியவற்றின் ஒப்பீடு பற்றிய ஆய்வு.

ஆய்வாளர் **:மரு. வெ. அருள்மொழி**, முதுநிலை பட்ட மேற்படிப்பு மாணவர்,
மகப்பேறு மற்றம் பெண்கள் நலத்துறை
ஆர்.எஸ்.ஆர்.எம்.மருத்துவமனை,
ஸ்டான்லி மருத்துவ கல்லூரி - சென்னை

பெயர் : வயது : உள்ளிருப்பு எண் :

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது என்னுடைய சந்தேகங்களை தீர்க்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்டசிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை எனவும் அறிந்து கொண்டேன் என்னை பற்றிய தகவல்கள் ரகசியமாக பாதுகாக்ப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப பயன்படுத்திக் கொள்ளவும் அதனை பிரசுரிக்கவும் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன் எனக்கு கொடுக்கப்பட்டுள்ள அறிவுரைகளின்படி நடந்து கொள்வதுடன் ஆய்வாளருக்கு உண்மையுடன் இருப்பேன், என்றும் உறுதி அளிக்கிறேன்.

உடல்நலம் பாதிக்கப்பட்டாலோ வழக்கத்திற்கு மாறான ஏதேனும் நோய்குறி தென்பட்டாலோ அதனை தெரிவிப்பேன் என்றும் உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும் சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு,

ஆய்வாளரின் கையொப்பம் நோயாளியின் கையொப்பம்

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Comparative study of intravenous Ferric Carboxymaltose and Iron sucrose in the management of Postnatal Iron deficiency Anaemia.

Principal Investigator : Dr. V Arulmozhi

Designation : PG, MS (O & G)

Department : Department of O & G
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

MASTER CHART

S.NO	NAME	AGE	IP.NO	MOD	OB.CODE	WEIGHT	IRON DOSE	PRE TRANSFUSION				POST TRANSFUSION -2 WEEKS			POST TRANSFUSION-4 WEEKS			SE
								Hb	MCV	P.S	Sr.Ferritin	HB	MCV	Sr.Ferritin	Hb	MCV	Sr.Ferritin	
1	LALLI	23	3830	1	2	50	896	8.7	80	M.H.A	32.6	11	86	185.82	11.7	88	130.4	_
2	ASAMMA	20	3856	2	4	55	896	9	82	M.H.A	44.9	11.1	87	228.99	12.1	89	133	_
3	SUDHA	28	4029	2	3	60	990	8.6	80	M.H.A	37	10.3	84	199.8	11.3	86	135.3	_
4	PALANIAMMAL	20	4076	2	2	45	932	8	78	M.H.A	28	10.1	83	159.6	11	85	102	P
5	Deivanayaki	24	4514	1	2	52	949	8.4	82	M.H.A	35	10.5	86	178.5	10.9	86	140	_
6	MANJU	19	4766	5	1	58	945	8.8	86	M.H.A	37.2	10.1	87	200.88	11.1	87	121.9	_
7	ELIZABETH	27	4931	4	2	48	961	8	78	M.H.A	25.9	9.8	82	147.63	10.5	83	108.78	_
8	SANTHI	27	5244	6	1	40	865	8.2	80	M.H.A	27.3	9.3	84	139.23	10.7	86	95.55	_
9	DIVYA	19	5407	5	2	56	984	8.4	79	M.H.A	26	10.3	83	140.4	10.7	83	104	_
10	DURGADEVI	18	5449	2	2	60	874	9.4	85	M.H.A	48	10.3	88	273.6	11.3	88	138	P
11	NANDHINI	22	5553	3	2	54	902	8.9	81	M.H.A	42	10.5	84	214.2	11.2	84	109	_
12	SALAMA	19	5713	5	2	50	968	8.1	76	M.H.A	22	9.4	80	118.8	11.1	84	77	_
13	LATHA	21	6329	2	1	48	926	8.3	78	M.H.A	25.5	10.5	84	145.35	11.2	85	102	_
14	DILSATH	18	6948	7	1	65	1030	8.6	79	M.H.A	33	10.7	83	168.3	11.5	84	105	_
15	KUMARI	23	7090	1	4	50	896	8.7	80	M.H.A	40.6	10.1	84	219.24	11	85	104.7	_
16	BHUVANA	21	7119	7	1	52	899	8.8	81	M.H.A	38	10.7	83	216.6	11.1	83	133	_
17	NALINI	29	7229	2	2	53	958	8.4	80	M.H.A	27.5	10.1	86	140.25	10.9	88	110	_
18	VINITHA	28	7683	2	3	47	816	9.2	86	M.H.A	46	10.7	88	248.4	11.3	89	118.5	_
19	RAMANI	23	7765	5	1	49	853	9	86	M.H.A	44.8	9.5	87	255.36	9.9	88	132	_
20	SHARIKA	21	8194	1	1	52	937	8.5	78	M.H.A	40	10.1	82	204	11	84	107	P
21	RAJALAKSHMI	19	8244	3	2	58	1057	8	76	M.H.A	19	9.6	80	102.6	10.3	82	76	_
22	JANAKI	24	8327	2	1	50	848	9.1	82	M.H.A	43	10.8	86	245.1	11.5	87	142	_
23	LOGESWARI	18	8677	1	2	53	856	9.2	80	M.H.A	45.2	10.5	84	230.52	11.1	84	139	_
24	PAVITHRA	20	8804	5	2	52	962	8.3	79	M.H.A	29.4	10.1	88	158.76	10.7	88	80.9	_
25	SASIKALA	21	9235	2	2	57	992	8.4	80	M.H.A	37.1	10.3	84	211.47	11.3	86	148.4	_
26	AMEENA BEEVI	27	9350	1	2	53	907	8.8	81	M.H.A	41.4	9.3	82	211.14	9.7	83	137.6	_
27	SWETHA	22	9369	7	1	50	896	8.7	79	M.H.A	44.5	10.5	82	240.3	11.2	84	98.4	_
28	NADHIYA	20	9381	2	1	48	961	8	75	M.H.A	18	9.9	80	102.6	10.3	81	76.8	_
29	MANJU	22	9395	1	1	52	849	9.2	86	M.H.A	46	11	87	234.6	11.7	88	127.5	_
30	CHITRA	20	9396	1	1	51	965	8.2	78	M.H.A	22.5	10.3	84	121.5	10.9	85	68.9	_
31	SASIKALA	25	9404	7	2	55	988	8.3	76	M.H.A	33.7	10.4	80	192.09	11.1	83	127.3	_
32	NISHANTHI	24	9410	2	2	57	938	8.8	80	M.H.A	41.7	10.9	82	212.67	11.9	86	102.6	_
33	KALPANA	25	9412	2	2	46	875	8.6	80	M.H.A	38.2	9.1	81	206.28	9.5	81	128.5	_
34	PARVEEN	21	9452	2	2	48	903	8.5	81	M.H.A	32.6	10	86	185.82	10.7	87	123.88	P
35	MAHARAJOTHI	26	9508	2	2	51	867	9	86	M.H.A	43.8	9.4	86	223.38	9.8	87	138.9	_
36	SANDHYA	20	9568	4	1	58	862	9.4	87	M.H.A	52.3	10.3	88	282.42	11.7	90	101.6	_
37	CHITRAVALLI	23	9735	3	2	62	1095	8	76	M.H.A	20.2	9.3	80	115.14	9.9	81	73.83	_
38	NEELAVATHY	27	9828	6	3	50	944	8.3	78	M.H.A	44	9.1	82	224.4	9.7	83	117.6	_
39	KARTHIGA	21	9959	7	3	55	922	8.8	80	M.H.A	46.1	9.7	84	248.94	10.5	86	151.2	_
40	SATHYAKALA	30	10020	5	1	61	983	8.7	81	M.H.A	37	10.5	86	210.9	10.8	87	109	P
41	DHIVYA	20	10033	1	1	46	886	8.5	77	M.H.A	33	10.8	86	168.3	11.5	87	94.1	_
42	KATRINA	20	10108	5	2	57	1047	8	75	M.H.A	24.9	10.3	84	134.46	11	86	84	_
43	INDUMATHI	21	10383	2	2	59	967	8.7	79	M.H.A	38.5	11	85	219.45	11.5	87	108.4	_
44	JYOTHI	20	10503	5	1	53	945	8.5	81	M.H.A	42	10.5	87	214.2	10.9	86	117	_
45	GEETHAPRIYA	26	11133	1	2	55	922	8.8	83	M.H.A	58.1	11	86	313.74	11.5	87	149.1	_
46	GOMATHY	22	11138	7	1	50	968	8.1	78	M.H.A	52	10.4	85	296.4	11.1	86	127.9	_
47	SUDHA	28	11146	2	2	51	990	8	76	M.H.A	34	9.9	82	173.4	10.5	84	96.8	P
48	LALITHA	23	10549	1	2	68	1006	8.9	83	M.H.A	51.5	11.1	85	278.1	11.5	86	106.8	_
49	MADHAVI	22	10571	2	2	40	865	8.2	78	M.H.A	36.6	10	84	208.62	10.3	85	108.8	_
50	PREETI	31	9354	6	1	65	1030	8.6	80	M.H.A	48.3	9.9	84	246.33	10.5	84	111	_
	AVG	22.76					935.53	8.57	80.12		37.12	10.22	84.22	199.97	10.92	85.44	113.62	

S.NO	NAME	AGE	IP.NO	MOD	OB.CODE	WEIGHT	IRON DOSE	PRE TRANSFUSION				POST TRANSFUSION -2 WEEKS			POST TRANSFUSION-4 WEEKS			SE
								Hb	MCV	P.S	Sr.Ferritin	Hb	MCV	Sr.Ferritin	Hb	MCV	Sr.Ferritin	
1	AKSHAYA	18	3845	2	1	48	926	8.3	76	M.H.A	36	10.2	86	292.6	10.7	88	144	_
2	SANDHYA	18	3916	2	2	56	863	9.3	86	M.H.A	54	11.3	89	391.6	12	90	152	_
3	KALAIRANI	22	4044	2	4	48	961	8	76	M.H.A	22.1	9.8	88	216.65	10.5	88	92.82	_
4	SINDHU	15	4227	1	1	53	882	9	88	M.H.A	54	11.2	90	307.6	12.1	92	189	_
5	TAMILSELVI	20	4682	5	2	58	959	8.7	82	M.H.A	49	10.5	88	367	11.2	89	153.32	_
6	GNANSUNDARI	20	4788	8	3	61	954	8.9	86	M.H.A	52.8	10.7	87	295.8	11	88	138.6	_
7	GOWRI	21	5086	2	2	40	874	8.1	78	M.H.A	21.6	9.8	84	200.44	10.4	86	90.72	_
8	RAGINI	27	5398	1	1	52	924	8.6	80	M.H.A	37.1	10.7	86	314.15	11.3	89	129.85	_
9	SARANYA	20	5417	1	1	57	883	9.2	85	M.H.A	48.5	11	88	373.7	12.2	90	144.3	_
10	REVATHY	21	5481	3	2	44	922	8	75	M.H.A	17	10.2	89	175	11	89	104.8	_
11	INDUMATHI	22	5713	5	2	56	970	8.5	81	M.H.A	21.3	10.9	87	202.93	11.2	87	89.46	_
12	LAKSHMI	25	5932	5	2	57	1020	8.2	76	M.H.A	24.5	10.5	88	217.55	10.5	89	85.75	_
13	NANDHINI	26	6474	2	3	59	1052	8.1	77	M.H.A	22.4	9.8	86	218.6	10.4	90	89.6	P
14	INDUMATHI	26	7090	1	2	49	970	8	75	M.H.A	16.9	10.2	85	177.78	11	88	92.6	_
15	DEEPIKA	20	7150	2	2	50	884	8.8	82	M.H.A	32.8	10	88	269.8	10.5	89	137.76	_
16	ELAMMA	21	7179	1	2	53	843	9.3	86	M.H.A	43.5	11	90	338.35	12	91	152.25	_
17	PRIYANKA	19	7198	7	1	48	961	8	78	M.H.A	18	9.8	85	179.2	11	87	123	_
18	VIJI	26	7707	6	2	59	967	8.7	84	M.H.A	36	10.7	88	307	11.2	90	136.8	_
19	YASODHA	22	8122	2	2	40	836	8.5	80	M.H.A	34	10.3	84	283.8	10.9	88	142.8	_
20	RAJESWARI	19	8230	2	2	47	816	9.2	86	M.H.A	42.7	10	88	329.2	11.7	88	149.45	_
21	PRIYA	22	8359	5	1	52	837	9.3	87	M.H.A	48.2	11.2	90	317	12	90	112.65	_
22	SARITHA	28	8661	1	2	42	823	8.8	79	M.H.A	39.5	10.9	87	306.05	12.3	89	150.1	_
23	BHAVANI	20	8728	1	2	61	1086	8	74	M.H.A	16.9	10.2	85	182.85	10.7	87	101.82	_
24	SUMATHY	36	9041	7	1	56	997	8.3	78	M.H.A	25.3	10.5	86	229.86	11.9	88	143.8	_
25	KALPANA	25	9110	5	1	64	1022	8.6	77	M.H.A	32.7	10.7	86	269.2	11.2	87	130.8	_
26	DHATCHYANI	24	9152	6	1	57	924	8.9	80	M.H.A	43.5	10.8	87	338.35	12.1	88	165.3	_
27	EZHILARASI	23	9255	6	2	55	1015	8	76	M.H.A	19.3	9.9	86	186.87	10.6	87	132.3	_
28	GOMATHY	19	9282	2	1	60	1047	8.1	75	M.H.A	18.9	10.3	88	195.85	11	88	124.9	_
29	SARITHA	35	9301	7	2	72	1191	8.2	77	M.H.A	20.2	10.4	84	198.24	11.3	87	119.7	_
30	NAGAMMA	18	9318	6	2	53	856	8	73	M.H.A	21.1	10.3	86	199.6	10.9	87	108.3	_
31	BHARANI	24	9362	5	1	48	926	9.2	82	M.H.A	46.7	11.4	87	357.87	12.3	88	116.72	_
32	NALINI	24	9387	3	2	52	937	8.3	75	M.H.A	21.7	10.8	84	201.03	11.4	86	148.21	_
33	RAJESWARI	28	9391	2	3	51	892	8.5	77	M.H.A	34.6	10.3	85	297.9	11.6	88	138.4	_
34	SURYAKALA	22	9481	7	2	55	1015	8.8	80	M.H.A	42.2	11.3	87	334.64	11.8	87	140.06	_
35	POONGODI	24	9520	5	3	49	818	8.1	72	M.H.A	19.5	10.6	85	190	11	86	129.9	_
36	SUMATHI	21	9532	2	1	52	837	9.3	85	M.H.A	43.5	11.2	88	338.35	11.6	88	152.25	_
37	REGINA	29	9823	1	1	55	975	8.4	78	M.H.A	31.8	10.6	86	260.62	11	87	147	P
38	SAVITHRI	26	9895	4	2	48	949	8.1	73	M.H.A	16.4	10.3	85	179.6	11	88	133.8	_
39	LAKSHMI	23	9926	1	2	53	945	8.5	77	M.H.A	34.7	10.4	84	288.14	10.7	86	145.74	_
40	BHARANI	22	10117	2	2	50	956	8.2	78	M.H.A	18.6	10.4	88	184.6	11	89	129.64	_
41	PRIYADHARSHINI	19	10123	2	1	49	794	9.5	86	M.H.A	49.4	11.7	87	374.34	12.5	90	138.7	P
42	KOMALA	23	10164	2	1	47	771	9.6	85	M.H.A	50.6	10.8	86	371.54	12.2	89	147.6	_
43	JEYANTHI	25	10192	7	2	56	997	8.3	78	M.H.A	17.8	10.6	86	188.7	11.4	87	134.3	_
44	SEETHA	29	10392	5	1	55	922	8.8	79	M.H.A	37	11	89	302.4	12.3	91	129.5	_
45	VANITHA	18	10495	2	2	57	1047	8	74	M.H.A	15.5	10.5	85	166	10.8	89	143	_
46	YASMIN	26	10533	2	2	56	1024	8.1	73	M.H.A	16.4	10.8	84	173.04	12.1	86	112.8	_
47	RANI	25	10552	3	3	55	1028	8	74	M.H.A	15.8	10.6	82	166.22	11.3	87	106.3	_
48	GEETHA	21	11033	2	2	50	956	8.2	72	M.H.A	18.9	10.8	80	195.85	12.5	88	111.4	_
49	AMULU	22	11597	7	1	47	895	8.5	76	M.H.A	31.3	10.6	83	267.06	11.9	87	155	_
50	NEELAVATHY	26	11731	1	3	58	945	8.8	80	M.H.A	43	10.4	84	331	11.2	88	153.9	_
	AVG	23.10					937.94	8.54	78.94		31.50	10.58	86.28	266.02	11.37	88.18	130.86	