"A RANDOMISED CONTROL TRIAL TO COMPARE THE ROLE OF INTRAVENOUS IRON SUCROSE VS ORAL FERROUS ASCORBATE FOR PROPHYLAXIS OF ANEMIA IN PREGNANT WOMEN"

BY

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Dissertation submitted to

B.L.D.E (DEEMED TO BE UNIVERSITY) VIJAYAPURA

In partial fulfilment of requirements for the award of the degree of

MASTER OF SURGERY

OBSTETRICS AND GYNAECOLOGY

Under the guidance of

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ABBREVIATIONS

Short form	Full form
ACD	Anaemia of chronic disease
ACT	Advanced Coating Technology
CBC	Complete Blood Count
DMT	Divalent Metal Transporter
Hb	Haemoglobin
HFE	Hereditary Hemochromatosis Protein
HIV	Human Immune Deficiency Virus
I.S	Iron Sucrose
I.V	Intra Venous
ICMR	Indian Council of Medical Research
ID	Iron Deficiency
IDA	Iron deficiency anaemia
IRF	Immature Reticulocyte Fraction
IRP	Iron Regulatory Proteins
IUGR	Intra Uterine Growth Retardation
LBW	Low Birth Weight
LIP	Labile Iron Pool

MCH	Mean Corpuscular Hemoglobin
MCHC	Mean Corpuscular Hemoglobin Concentration
MCV	Mean Corpuscular Volume
Mg	Milligram
MMR	Maternal Mortality Ratio
NNACP	National Nutritional Anaemia Control Programme
O.I	Oral Iron
O_2	Oxygen
OPD	Out Patient Department
PCV	Packed Cell Volume
RBC	Red Blood Cells
RES	Reticulo Endothelial System
RPI	Reticulocyte Production Index
sTfR	Soluble Transferrin Receptor
Tf	Transferrin
TfR	Transferrin receptor
TIBC	Total Iron-Binding Capacity
TSI	Transferrin saturation index
WHO	World Health Organization
ZPP	Zinc Protoporphyrin

ABSTRACT

Background: Anemia in pregnancy is a significant health concern, particularly in low- and middle-income countries like India. Iron deficiency anemia (IDA) is the most common cause, leading to adverse maternal and fetal outcomes. Though oral iron therapy is the standard prophylaxis, poor compliance due to gastrointestinal side effects limits its effectiveness. Intravenous iron sucrose (IVIS) may provide an alternative with better tolerability. The study attempts to assess the efficacy, safety, and tolerability of intravenous iron sucrose vs oral ferrous ascorbate for prophylaxis of anaemia in pregnant women.

Methods: This Randomised controlled trial study was done in the Department of Obstetrics and Gynaecology B.L.D.E (DU) Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura, Karnataka. One hundred antenatal women with confirmed intrauterine pregnancy with hemoglobin $\geq$11g/dL were randomized into IV Iron Sucrose (Group A) and Oral Iron Ascorbate groups (Group B). Group A pregnant women received three doses of Intravenous iron sucrose 200mg in 100ml normal saline as an infusion over 15-20 minutes at 20-24 weeks, 24-28 weeks, and 28-32 weeks of gestation. Group B pregnant women received oral ferrous ascorbate, providing 100 mg of elemental iron daily at bedtime, one hour before meals till delivery. Hb, RBC COUNT, PCV, MCV, MCH, MCHC and Serum Ferritin were assessed at baseline, later at 4 weeks and 12 weeks.

Results: Most cases belonged to 21-25 years in the IV Sucrose group (54%) and Oral Ascorbate group (50%). Most cases belonged to primigravida, i.e., 62% of cases in the IV group and 56% in the Oral group. All the blood indices, such as the mean Hb, RBC, PCV, MCV, MCH, MCHC, and Ferritin, had increased from baseline to 12 weeks in the IV sucrose and in the oral group only mean ferritin was increased while remaining all the parameters were maintained more or less in the same level, but the increment was higher in the IV group than the Oral group. Of the total cases, anaemia was seen in 6.8% of cases of the IV sucrose group and 17% of cases of an oral group that showed improved Hb, which was more with IV sucrose than with an oral group. Regarding side effects, the IV Sucrose group had fewer side effects than the Oral group. Compliance was more in the oral group (94%) than in the IV I.S group (88%). The cost of the treatment in the IV group was lower than in the oral group.

Conclusion: Intravenous iron sucrose is more effective, better tolerated, and improves iron stores significantly with fewer side effects compared to oral ferrous ascorbate for prophylaxis of anemia in pregnancy. The cost of the treatment in the IV group was less than that in the Oral group.

Key words: Antenatal, Prophylaxis, anaemia, IV iron sucrose, Oral iron ascorbate

INTRODUCTION

As per the World Health Organization (WHO), anaemia during pregnancy is characterized by a haemoglobin concentration of less than 11 g/dl.[1] It is categorized as mild, moderate, and severe, according to haemoglobin levels of 10.0 to 10.9, 7.0 to 9.9, and below 7.0 g/dl, respectively.[2,3] As per the WHO, anaemia was present in nearly 37% of pregnant women.[3]

As per WHO, prevalence under 4.9% is a negligible public health issue.[6] Anaemia is categorized as mild, moderate or severe based on prevalence rates of 5.0 to 19.9%, 20.0 to 39.9%, or ≥40.0%, respectively. [7,8]

According to the ICMR, anaemia is categorized as mild, moderate, severe, and very severe, according to haemoglobin levels of 10.0 to 10.9, 7.9-9, 4.6 - 6.9, or below 4.0 g/dl, respectively. [9]

In India, anaemia is a significant factor in approximately 40% of maternal deaths, either directly or indirectly. When haemoglobin (Hb) levels drop below 5 g/dl, the maternal mortality ratio (MMR) increases 8 to 10-fold.[10] Early detection and effective management of anaemia during pregnancy can play a critical role in reducing maternal mortality. Maternal anaemia is linked to poor intrauterine growth, as well as a heightened likelihood of preterm births and LBWs, which lead to higher rates of perinatal morbidity, mortality, and infant mortality.[11] When Hb levels fall below 8 g/dl, there is a 2 times chance of LBWs and a 3-fold rise in perinatal mortality rates.[9] IUGR and LBW often result in poor growth during childhood,

ultimately contributing to reduced adult height.[12] During the pregnancy, the requirement for Iron rises from 0.8 mg/day at the beginning to 7.5 mg/day at the end of pregnancy.[6]

Recognizing the importance of anaemia, under the National Nutritional Anaemia Control Programme (NNACP),1970, the Government of India initiated various methods through government hospitals to prevent and treat anaemia.[9] As per the NNACP, following the first trimester of pregnancy, one Iron tablet daily should be consumed for a minimum of 100 days. Each tablet comprises 60 mg of elemental Iron and 500 mcg of folic acid. The same dosage is appropriate for lactating women. Iron deficiency anaemia (IDA) is still prevalent among pregnant and postpartum women despite the NNACP's diligent initiatives. [9]

Iron therapy, besides dietary modifications, is the sole treatment for iron deficiency anaemia. Government initiatives for the prevention and treatment of anaemia utilize O.I. as it is a cost-effective, safe, and efficacious method for iron replenishment. Vitamin C and 180–200 mg of elemental Iron is generally provided in two or three doses distributed between meals. Nevertheless, their efficacy is frequently impeded by inadequate tolerability, gastrointestinal adverse effects, and reduced adherence in pregnant women.[1]

AIM AND OBJECTIVES

AIM

- To compare the efficacy of prophylactic IV I.S with Oral ferrous ascorbate in Pregnant Women.

OBJECTIVES

- To know the improvement in Hb in both IV I.S and Oral ferrous ascorbate groups.
- To know the compliance and cost effectiveness of parenteral vs O.I supplementation.
- To know the safety of parenteral vs. O.I supplementation.
- To know which is better in increasing iron stores.

REVIEW OF LITERATURE

ANAEMIA

Anaemia has three fundamental causes: dietary deficits, infectious diseases, and genetic problems associated with haemoglobin. Iron deficiency anaemia is especially common in nations with severe poverty. As per the studies, anaemia impacts 27% of the global population,[11] with iron deficiency being the primary cause. Insufficient ingestion of vital nutrients such as iron, vitamin B12, and folic acid and excessive use of tea, coffee, and specific spices may cause nutritional deficiencies that frequently lead to anaemia. Likewise, hereditary abnormalities such as sickle cell disease and glucose-6-phosphate dehydrogenase deficiency, together with diseases including malaria, HIV, and tuberculosis, substantially contribute to the onset of anaemia. This major public health issue impacts almost 500 million women aged 15 to 49.[12] In 2019, the WHO estimated that 30% of non-pregnant women and 37% of pregnant women were impacted by anaemia.[13]

Pregnant women with anaemia experience greater risks for maternal issues, including anaemic heart failure, premature birth, and postpartum haemorrhage, as well as a 3-5fold increased mortality risk in cases of severe anaemia.[14] It corresponds with a higher rate of adverse perinatal outcomes, such as fetal growth restriction, fetal demise, birth asphyxia, and neonatal mortality in 62% anaemic pregnant women versus 28% in non-anaemic pregnant. [3]

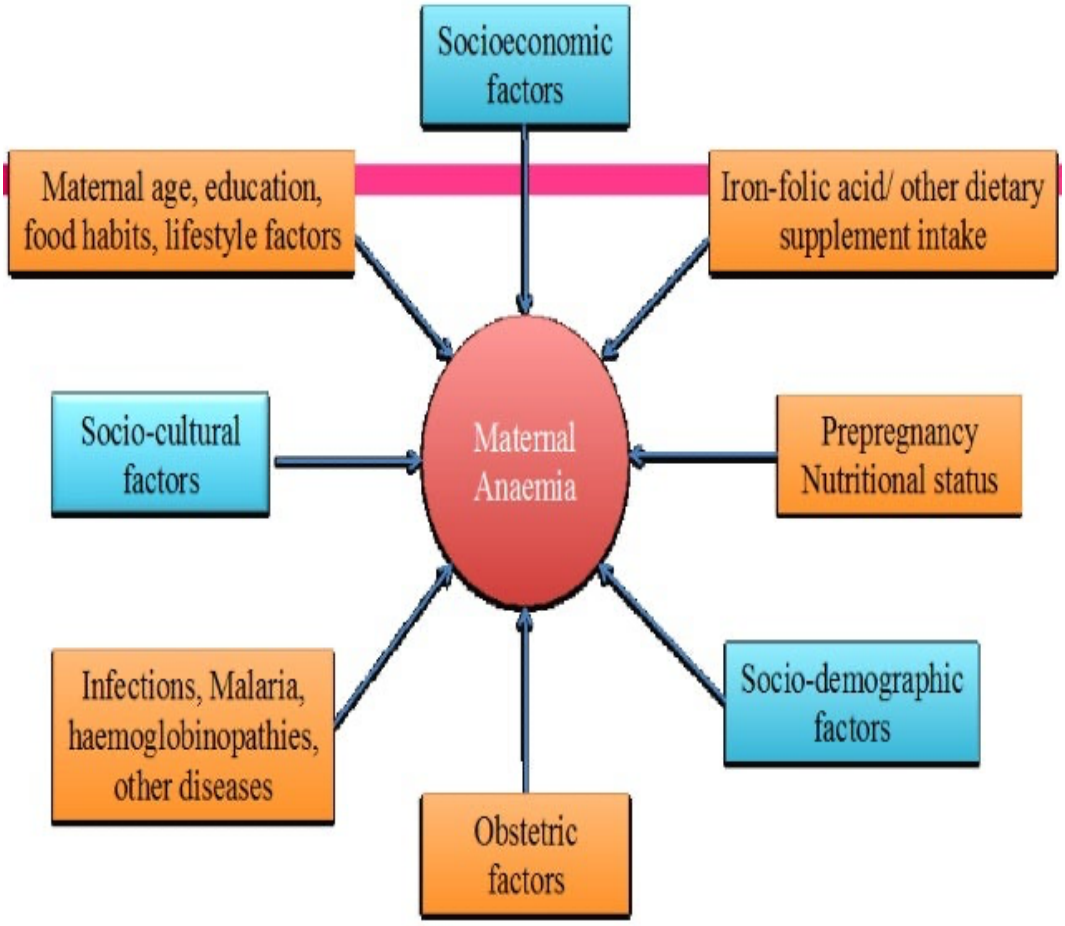


Figure 1: Maternal risk factors of anaemia [15]

CLASSIFICATION OF ANAEMIA:

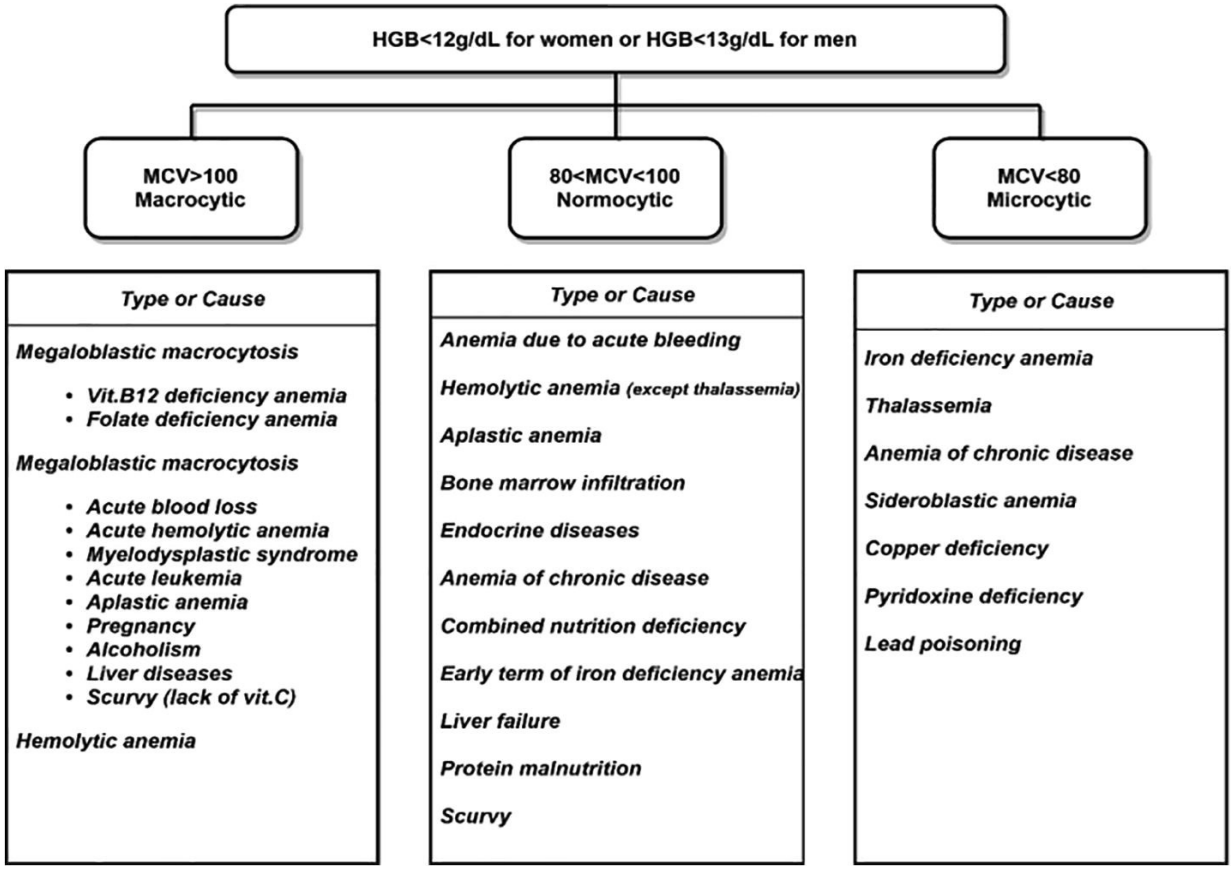


Figure 2: Classification of anaemia [16]

Anaemia remains a significant public health challenge in India, affecting diverse populations across various age groups. The types of anaemia prevalent in India are mainly classified based on their etiologies: nutritional deficiencies, hereditary disorders, and chronic diseases. Each type has distinct causes, manifestations, and implications for health.

Anaemia in India is multifaceted, with various types contributing to the overall burden of the disease. Effective public health interventions, including improved nutrition, screening for hereditary anaemias, and management of chronic diseases, are essential to reduce the prevalence and impact of anaemia in the population.

Numerous studies indicate that iron deficiency is the predominant cause of anaemia during pregnancy, particularly in nations with low or middle incomes. It is linked to poverty and malnutrition, especially among women and girls during menstrual blood loss.[5-8] The demand for iron significantly increases during pregnancy due to the fetoplacental unit's higher requirements and the necessity to balance blood loss during childbirth. This condition typically arises from diminished consumption of iron-rich foods, including red meat, green leafy vegetables, and iron-fortified products like cereals and bread.

Regardless of initiatives to lower the prevalence of anaemia, particularly in countries with lower incomes, it remains prevalent worldwide, leading to significant health effects if not properly addressed.[4,5] Maternal anaemia adversely affects both pregnant mothers and newborns significantly. The mortality risk for pregnant mothers with severe anaemia has been estimated to be double that of women without severe anaemia.[6,7] Significant haemorrhage during birth or postpartum [8,9] and problems arising from anaemia, including heart failure, may lead to this mortality.[8,10]

Anemia adversely affects fetal outcomes by elevating the incidence of perinatal morbidities, including LBW, prematurity resulting from spontaneous preterm birth, and newborn iron deficiency. Inadequate gestational iron consumption correlates with autism, schizophrenia, and atypical brain anatomy in kids.

1.VITAMIN B12 DEFICIENCY ANAEMIA

Vitamin B12 deficiency anaemia is another prevalent type in India, often resulting from inadequate dietary intake or malabsorption. This condition is particularly common among vegetarians due to the limited availability of vitamin B12 in plant-based foods. A study by Yajnik et al. (2019)[17] highlighted that a significant portion of the Indian population, especially vegetarians and the elderly, suffer from vitamin B12 deficiency. Symptoms include weakness, fatigue, numbness, and cognitive disturbances. Severe deficiency can lead to irreversible neurological damage if not treated promptly.[18] Blood peripheral smear shows anisocytosis, poikilocytosis, macro-ovalocytosis.[19]

2.FOLATE DEFICIENCY ANAEMIA

Folate deficiency anaemia, caused by insufficient intake of folate, a type of B vitamin, is also common in India. This deficiency is often seen in pregnant women, as the necessity for folate enhances during pregnancy. A study by Gupta et al.

(2017)[20] indicated that folate deficiency is prevalent among pregnant women in India, contributing to megaloblastic anaemia. Symptoms include fatigue, mouth ulcers, and grey hair. Lack of folate in pregnancy leads to neural tube defects in the newborn.[21]

3.SICKLE CELL ANAEMIA

Sickle cell anaemia is a hereditary disorder that is particularly prevalent among specific tribal populations in India. It occurs due to a mutation in the haemoglobin gene, leading to the generation of abnormal haemoglobin S. This abnormal haemoglobin causes RBCs to be sickle-shaped, leading to chronic hemolysis and organ damage. The prevalence of the sickle cell trait is estimated to be 1-40% in various tribal groups in India,[22] with a higher prevalence in central and western India. Management includes regular monitoring, pain management, and prevention of complications through measures such as vaccination and prophylactic antibiotics.[23]

4.THALASSEMIA

Thalassemia is another significant hereditary anaemia in India, caused by mutations that affect the production of haemoglobin. It is classified into α and β thalassemia. Beta thalassemia major, the most severe form, requires frequent blood transfusions for survival. The prevalence of thalassemia carriers (trait) in India is estimated at 3-4%, translating to nearly 35-45 million nationwide carriers.[24]

Thalassemia major poses significant challenges due to the need for lifelong transfusions and iron chelation therapy to prevent iron overload.[25]

5.ANAEMIA OF CHRONIC DISEASE

Anaemia of chronic disease (ACD) is prevalent among individuals with chronic infections, inflammatory diseases, or malignancies. In India, conditions such as tuberculosis, HIV/AIDS, chronic kidney disease, and cancers contribute significantly to the burden of ACD. This type of anaemia is typically mild to moderate, characterized by impaired iron utilization and a shortened red blood cell lifespan. Treatment focuses on managing the underlying chronic condition and may include erythropoiesis-stimulating agents in severe cases.[26]

6.IRON DEFICIENCY ANEMIA (IDA)

Iron is vital for cellular function due to its involvement in oxygen transport, electron transfer, and enzymatic processes. Cells exhibiting elevated metabolic rates necessitate increased iron and are more susceptible to malfunction in the context of iron deficit. The demand for iron during pregnancy significantly escalates due to the expansion of the mother's blood volume and the growth and development of the fetus.

IDA is the most common type of anaemia globally and in India. It occurs due to inadequate iron intake, poor absorption, or increased iron requirements. In India,

IDA is particularly prevalent among children, adolescent girls, and pregnant women. According to the NFHS-5, 57% of 15-49-year-old women and 67% of children aged 6-59 months are anaemic, primarily due to iron deficiency.[22] Factors contributing to IDA in India include poor dietary intake of iron-rich foods, high prevalence of gastrointestinal infections, and frequent pregnancies. Symptoms of IDA include fatigue, pallor, breathlessness, and impaired cognitive and physical development in children. A few classical signs of IDA are- Koilonychia, pica, glossitis, angular Cheilitis, and brittle nails.[20,21]

It is common in nations where grain is the staple food and meat is in short supply. Regretfully, hookworm infestation is endemic in most of these nations. The risk of getting IDA is increased when prolonged blood loss from parasite infection is combined with low dietary iron availability. Reduced intake of other vital nutrients, such as folate, is linked to malnutrition, in addition to decreased intake of iron. Therefore, a variety of factors might contribute to anaemia linked to malnutrition.

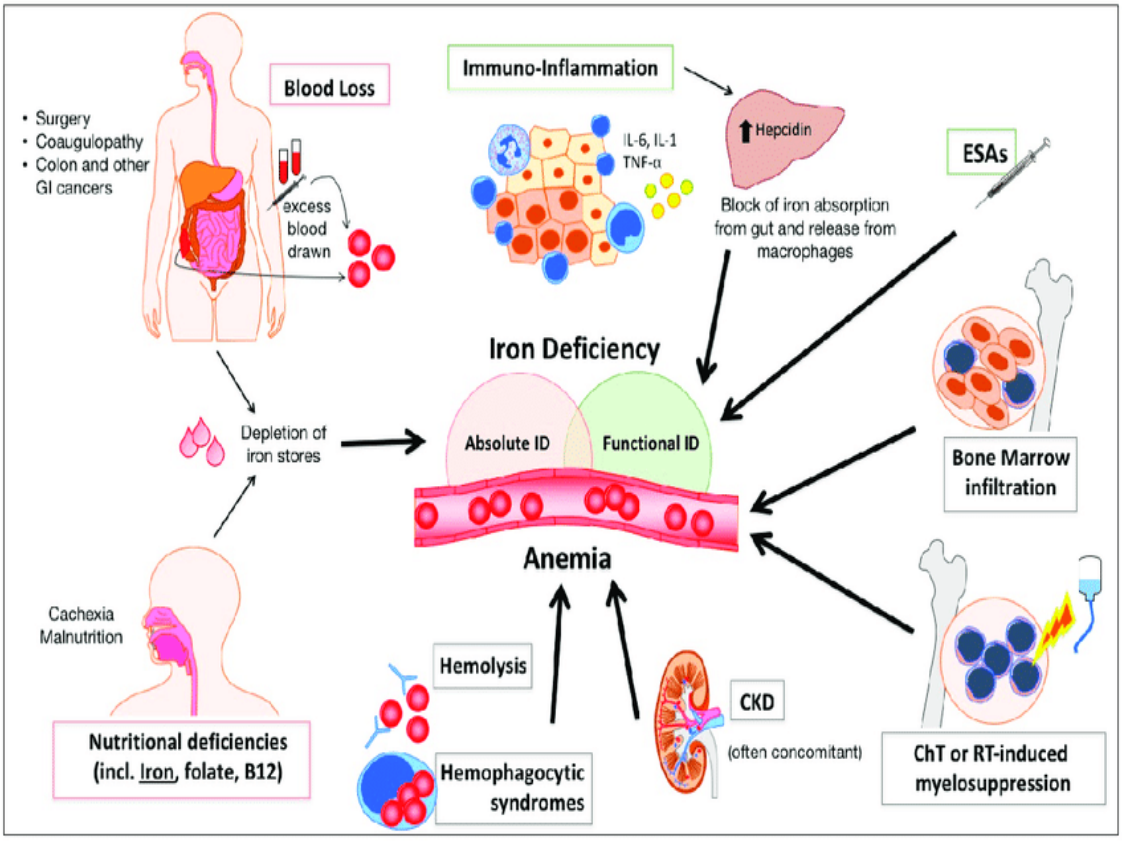


Figure 3: Causes of Iron deficiency [27]

6.1 Iron metabolism [28-30]

The body needs iron for every cell. It is essential for cellular development, proliferation, oxygen delivery and storage, and oxidative metabolism. To do these tasks, iron has to be linked to protein molecules. Iron can be harmful when it is present in inorganic compounds or ionized. Iron toxicity can arise when the body's ability to transport and store iron in its protein-bound state is exceeded. This can lead to damage to cells and a potentially fatal condition. On the other hand, insufficient iron availability limits the production of iron molecules that are physiologically active and inhibits vital metabolic processes.

Iron needs specialized transport involving a range of proteins since it cannot readily diffuse across membranes. Hepatocytes, macrophages, and enterocytes are absorptive cells found at the luminal [apical] surface of the duodenum that can import and export iron.

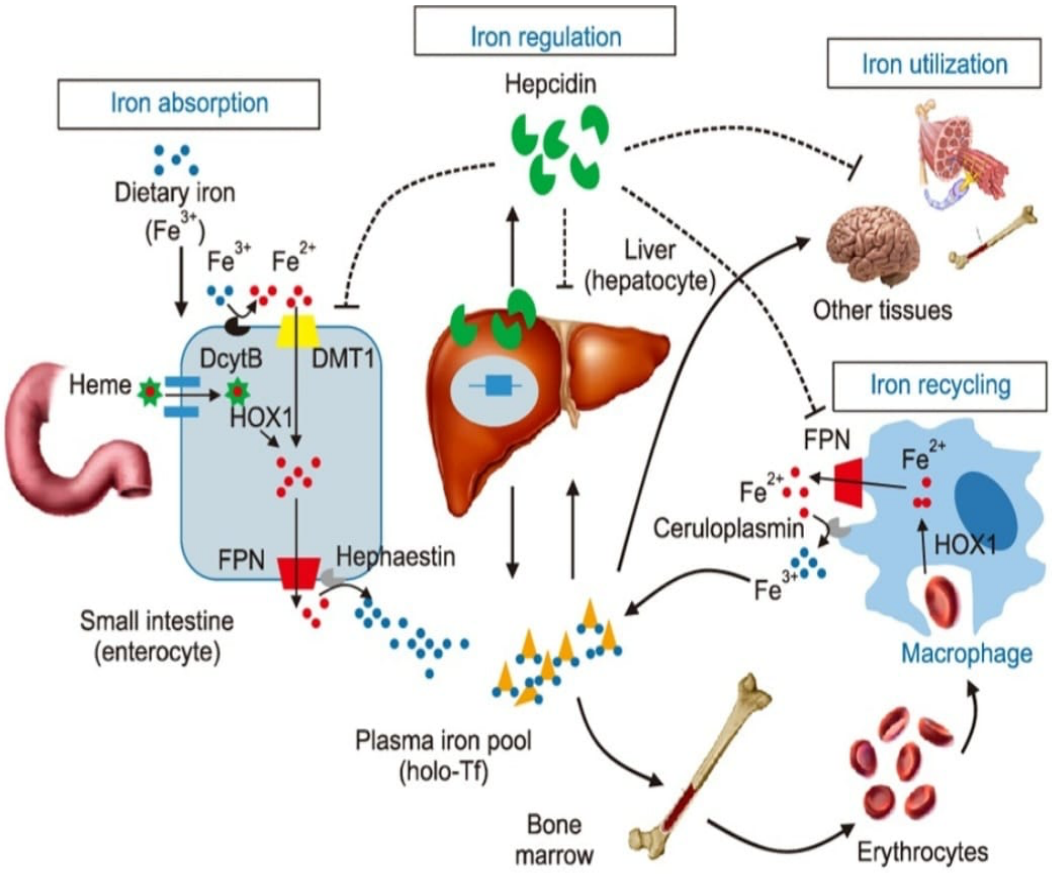


Figure 4: Iron metabolism [31]

6.2 Distribution of Iron

The body contains two different types of substances that contain iron:

(1) Enzymatic (catalase, peroxidase, cytochromes, cytochrome oxidase, and haemoglobin), oxygen transport (haemoglobin, myoglobin, neuroglobin) and

(2) Substances that function as iron storage facilities (ferritin and hemosiderin) or transport proteins (transferrin, transferrin receptor).[31]

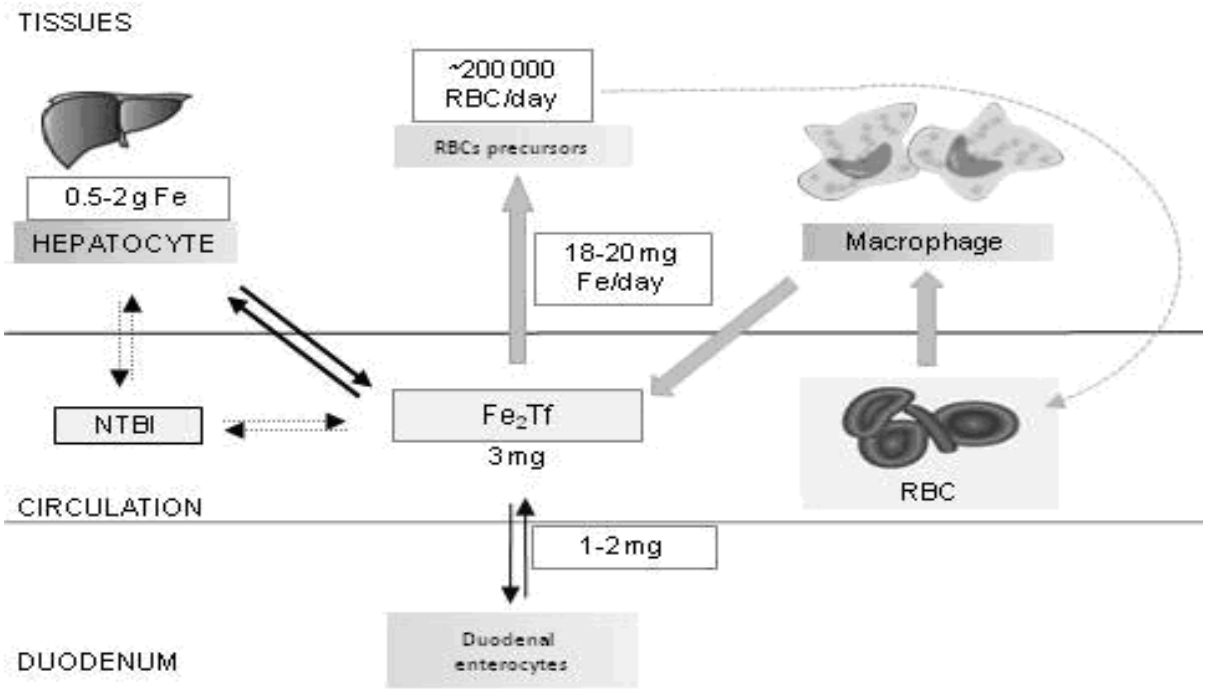


Figure 5: Distribution of iron [32]

A majority (60-70%) of the body's iron is integrated into the Hb of circulating RBCs. Nearly 20-30% of the body's iron is stored as ferritin and hemosiderin within hepatocytes and reticuloendothelial system macrophages as reserve iron.[33]Approximately 3 mg of iron is bound to transferrin;[34] nevertheless, the transferrin compartment serves as a transit compartment through which approximately 20 mg of iron circulates daily.[35] The bone marrow is the primary consumer of circulating iron. Every day, 18-20 mg of predominantly recycled iron

is utilized to synthesize haemoglobin in 200 billion new erythrocytes.[36] Individuals in good health assimilate 1-2 mg of iron daily, which offsets iron depletion.[37]

6.3 Iron absorption

The absorption of iron from food is greatly influenced by the duodenum. By binding to the liver-derived plasma protein transferrin (Tf), the absorbed iron can enter the circulation and be carried throughout the body or retained in the enterocytes. After that, it is absorbed by tissues and used for various functions, including oxidative metabolism in all respiring cells, myoglobin synthesis in muscle, and erythropoiesis in the bone marrow. Iron recycling from senescent erythrocytes is the responsibility of RES-affiliated splenic, hepatic, and bone marrow macrophages. The liver plays a crucial role in regulation and storage. It regulates the release of iron into the bloodstream from enterocytes and macrophages by producing the hormone hepcidin. As a result, plasma iron concentrations can be precisely controlled and kept within physiological ranges. The body loses about 1-2 mg of iron daily due to enterocyte and skin desquamation, haemorrhages, and parasite infestations.[38] There is no active iron excretion mechanism. Therefore, intestinal absorption of iron must be 1-2 mg each day to maintain iron homeostasis.[39] Physiological circumstances, including growth, pregnancy, and menstruation, boost this requirement.

6.4 Systemic iron homeostasis

The primary regulator of systemic iron homeostasis, including intestinal iron intake and recycling in the REC, is presently believed to be hepcidin. A 25-amino acid protein called hepcidin attaches itself to ferroportin, causing internalization and subsequent liposome breakdown. Hepcidin traps iron in enterocytes, macrophages, and hepatocytes since ferroportin is an iron exporter. The level of transferrin receptor (TfR) 1 and 2 in the liver and the degree of transferrin saturation control the hepatic synthesis of hepcidin. Consequently, hepcidin expression is induced by a rise in the ferric Tf/TfR ratio. This inhibits ferroportin-1 activity and, consequently, basolateral iron transfer. However, a drop in the differential Tf/TfR ratio stops the liver's hepcidin synthesis, and iron absorption is restored.[40]

6.4.1 Enterocyte uptake of dietary iron

Heme or nonheme iron can be found in human diets. Meat, poultry, and shellfish contain heme (iron-protoporphyrin IX), primarily derived from haemoglobin and myoglobin. The term "nonheme iron" describes various inorganic iron forms and is typically connected to iron found in plants, vegetables, and whole grains. However, nonheme iron (almost half of the total iron) is also present in foods derived from animals, primarily in the iron storage protein ferritin. However, heme iron significantly contributes to iron nutrition because it is far more accessible than nonheme iron. DMT1 (SLC11A2, solute carrier family 11 member 2) transports dietary nonheme iron across the apical membrane of enterocytes in the acidic

microenvironment at the surface of the proximal small intestine.[36] Mice with intestinal deletion of Slc11a2 exhibit severe iron-deficiency anaemia, reduced enterocyte iron contents, and significantly impaired iron absorption.[38]

Consequently, DMT1 is the sole method of absorbing nonheme iron in the intestinal brush boundary. MT1 operates at acidic pH by linking the movement of protons along an electrochemical gradient to the cellular uptake of iron.[41] The intestinal brush border Na/H exchanger 3 (NHE3) establishes the hydrogen gradient that facilitates DMT1-mediated iron absorption. DMT1 exclusively transports Fe2, but the predominant form of dietary iron is Fe3.[42] This process is facilitated by the ferrireductase duodenal cytochrome B situated in the apical membrane of enterocytes.[43] The fact that Dcytb mRNA expression is significantly up-regulated in the intestines of iron-deficient mice indicates that Cybrd1-null mice display no discernible abnormalities in iron metabolism, even under conditions of iron deficiency.[44]

6.4.2 Export of iron from enterocyte to plasma

Ferroportin (SLC40A1), which is found on the basolateral membrane, transports iron from the enterocyte into the portal blood. Ferroportin is crucial for intestinal iron export. Only Fe2 is transported by ferroportin, while only Fe3 is bound by transferrin in portal blood.[45] It is believed that a ferroxidase-catalyzed oxidation step is necessary for the effective transfer of iron to portal blood transferrin. Hephaestin, a membrane-anchored homologue of ceruloplasmin, the

plasma ferroxidase, is the intestinal ferroxidase with the best characterization.[43] It was discovered that the defective gene in the sex-linked anemia mouse, which exhibits mild iron-deficiency anemia and reduced iron absorption, is hephaestin.[46]

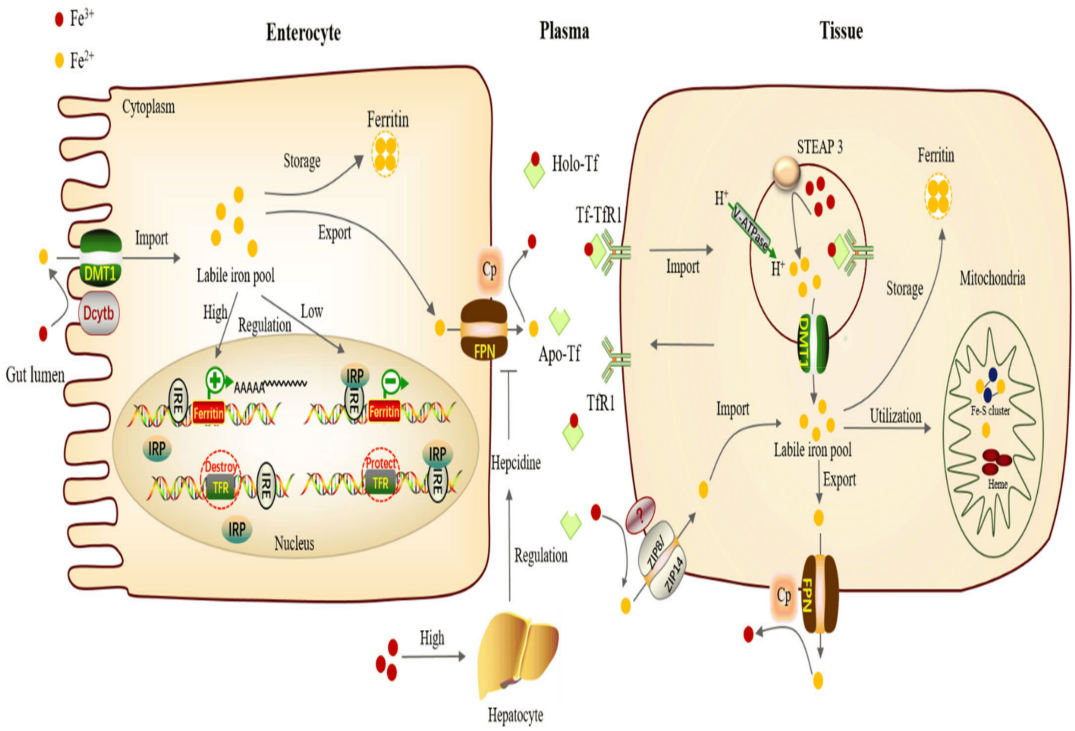


Figure 6: Export of iron from enterocyte to plasma [47]

6.5 Iron distribution

Iron enters the bloodstream after absorption and attaches itself to Tf to facilitate transportation. Because intracellular iron controls the hepatic production of Tf, plasma transferrin levels rise as iron levels fall. The transferrin saturation index (TSI) is typically between 30 and 35 percent because Tf can bind up to two iron atoms.[48] Erythropoiesis is regulated by the TSI, and when the TSI drops below 16%, this is significantly decreased.[49] On the other hand, iron carried by Tf is redirected to the liver and may result in hepatic hemosiderosis when the TSI rises

beyond 90%. Iron regulatory proteins 1 and 2 (IRP1 and IRP2) negatively regulate TfR, DMT-1, and ferritin production in erythroblasts.[50]

6.5.1 Transferrin receptors

TfR1 and TfR2, two varieties of functionally distinct transferrin receptors, are described. All iron-requiring cells express TfR1, although expression levels vary widely. It is highly expressed on placental tissue, quickly dividing cells (both benign and malignant), and immature erythroid cells. The transmembrane glycoprotein TfR1 has a molecular mass of about 90 kDa and is made up of two identical disulfide-bounded subunits.[51] Every subunit has a single transferrin binding site. Compared to iron-free apotransferrin or monoferric form, diferric transferrin exhibits a greater affinity for TfR1. In addition to the membrane-associated TfR1, human serum contains a soluble form of this receptor, which is a soluble portion of the extracellular receptor domain. The C-terminal end of the protein is cleaved by proteases, releasing the soluble transferrin receptor (sTfR).[52] TfR2 shares similarities with hereditary hemochromatosis protein (HFE) and is mostly expressed in the liver, hematopoietic cells, and duodenal crypt cells. Although HFE and transferrin are bound by TfR2, their interaction domains differ from those of TfR1. The transcriptional control of hepcidin synthesis by diferric transferrin is thought to require the TfR2/HFE complex.[53]

6.5.2 Transferrin cycle

Differential transferrin binds to TfR1 at the cell surface, starting the transferrin cycle and causing clathrin-mediated endosome formation. The proton pump's action on the endosome membrane causes the endosome content to become acidic and alters the conformation of both transferrin and the transferrin receptor, which releases iron. Ferrireductase STEAP3 subsequently reduces Fe(III), and DMT1 carries iron across the endosome membrane and into the cytoplasm.[54] After finishing the transferrin cycle and returning to the cell surface, apotransferrin is released and replenished with iron. TfR1 is introduced for a fresh cycle of uptake. Transferrin performs between 100 and 200 cycles of iron transport over its lifetime.[55]

Iron enters the poorly defined "labile iron pool" (LIP) after cellular uptake. Iron complexed with low-affinity ligands is known as a LIP. Iron (II) glutathione was shown to be the predominant component of this pool in a recent study. Less than 5% of the total cellular iron comprises LIP.[56] It provides iron to the mitochondrion to create heme and iron-sulfur clusters, or it may be utilized in the cytosol to synthesize iron-containing proteins, which regulate various metabolic processes. Because LIP is catalytically active and can start free radical reactions, iron in the LIP is more than is needed to synthesize heme and non-heme iron-containing proteins.[57]

Iron enters cells primarily through transferrin-bounded iron entry under normal conditions. Still, pathological iron buildup causes transferrin saturation and the emergence of NTBI, which can enter cells through a transferrin-independent mechanism.

6.6 Hepcidin

Iron metabolism is negatively regulated by hepcidin. It attaches itself to ferroportin, facilitating uptake and, ultimately, the lysosomal breakdown of this iron exporter. Cellular iron retention results from the loss of ferroportin from the cell membrane, which also suppresses iron efflux into the blood from the primary iron flow sites, lowering transferrin saturation and availability of iron.[58]

Iron disorder is caused by a genetic or acquired dysregulation of hepcidin production. A higher body iron level in a healthy person would result in higher hepcidin expression and, consequently, lower iron absorption. Iron absorption persists in HH patients despite a high body iron burden due to insufficient or poor hepcidin-mediated down-regulation of ferroportin. Conversely, hypoferremic microcytic, iron-refractory anemia is linked to overexpression of the hepcidin gene. An 84-amino acid (aa) prepropeptide synthesizes hepcidin, which is then converted into 60–64-aa prohepcidin.[59] Prohormon convertase furin is used to remove the proregion, producing mature and physiologically active 25-aa hepcidin.[60] Four disulfide bonds stabilize the basic hairpin shape that hepcidin produces.[61]

Additionally, it circulates in plasma as the prohormone pro-hepcidin, which has no biological function, and as a 22-aa peptide.[62]

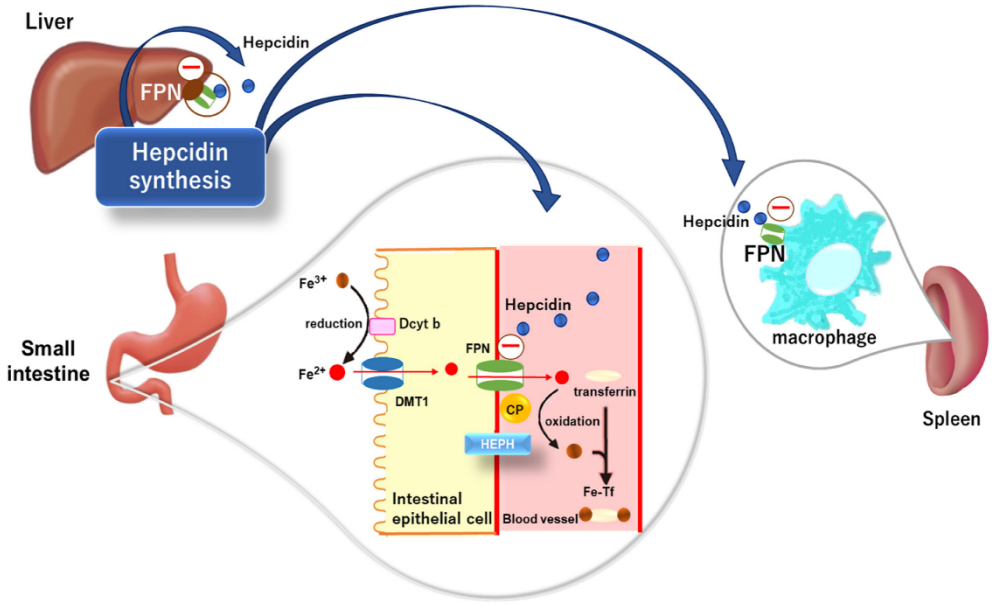


Figure 7: Hepcidin mechanism [61]

6.7 Iron storage and recycling

Senescent erythrocytes are phagocytized by macrophages 120 days after they enter the bloodstream.[62] There, haem oxygenase breaks down the haem group and releases Fe2+, which is then carried into the cytoplasm by Nramp-1. A large amount of this iron is stored as ferritin and hemosiderosis, with the remainder taken by ferroportin1 across the macrophage membrane, oxidized by hephaestin to Fe3+, and integrated into Tf. Since erythron requires 20–30 mg of iron daily, this Fe recycling route is crucial. Iron uptake and storage pathways in the liver differ from enterocytes and macrophages.[63]

Oxidative stress and cardiac alterations brought on by hydrogen peroxide-induced DNA damage during the Fenton reaction can result from an excess of iron in myocytes. Producing proinflammatory cytokines involves lymphocytes in these oxidative stress and iron storage processes.

6.8 Iron loss and excretion

Under normal circumstances, one to two milligrams of iron are eliminated daily. When epithelial cells are shed from the skin and the gastrointestinal tract's lining (main route), normal iron loss takes place. Additionally, it is eliminated through perspiration, menstrual blood, breast milk, and urine. During menstruation, women typically lose about 30 milliliters of blood, while some may lose up to 118 milliliters.[64] About 40–50 mg of iron is present in every 100 millilitres of blood, and increased blood loss during menstruation is a typical cause of IDA in those who do not get enough iron from their diet.[65]

6.9 Iron requirements

Humans typically have a very stable body iron concentration throughout their lives. This is achieved by maintaining an equilibrium between iron absorption and loss in adulthood and building a positive iron balance during the growing years. Since iron cannot be excreted by humans, iron absorption and loss rates must be adjusted to prevent ID or excess.[32]

Factors that increase Fe requirements

Growth and pregnancy and regular physiologic processes like menstruation raise the daily need for iron.

Menstruation

Menstruating women lose an average of 0.6–2.5% more iron per day than non-menstruating women.[35] Menstruating females need to absorb roughly 2 mg of iron per day to maintain the overall iron balance of their bodies.[42]

Pregnancy

The daily iron needed for pregnant women is approximately 3.4 mg, which, when averaged over three trimesters, adds up to nearly 1000 mg of iron per pregnancy.[45] The fetus absorbs roughly 250 mg of iron from the mother's stores through the placenta.[31] This amount is further enhanced by the iron needed for the increased volume of blood in the mother during delivery and iron loss from haemorrhage. Thus, the mother's iron reserves could be depleted within a single pregnancy if no more iron is given.

Infancy/Childhood

More iron is needed in proportion to food intake during infancy due to the fast growth of haemoglobin mass and body size compared to subsequent stages of life. An infant produces roughly 50 g of fresh haemoglobin in the first six months. Iron is also required for the growth of tissue.[27,28] Normal iron levels of 30 mg at birth are

sufficient to last the baby for the first 4-5 months of life,[45] but they can soon run out in a baby whose primary source of nutrition is unfortified cow's milk. Because iron can be extracted from human breast milk so effectively, a significant amount of the iron present can be absorbed by newborns.

Because more iron is transferred from the placenta during the final trimester of pregnancy and premature babies grow more quickly than full-term babies, they are considerably more vulnerable to rapid iron depletion. In one study, 45% of preterm, very low birth weight infants had ID at one year's corrected age.[19] It is advised that LBW infants start taking iron additions no later than 2 months of age and full-term infants no later than four months.

Iron requirements are also high in childhood, especially in 1 to 2 year-olds. Globally, about 25% of preschool children have iron-deficiency anaemia,[31] which affects over 25% of preschool-aged children worldwide.[29]

Promoters of iron absorption: [24]

- Haem iron is present in meat, poultry, fish, and seafood;
- Ascorbic acid, or vitamin C, is present in fruits, juices, and green leafy vegetables.
- Fermented or germinated food.

Preventers of Iron absorption

- Phytates,
- Inositol-containing foods;
- Tannins;
- Tea, coffee, cocoa, and
- Calcium, particularly from milk and milk products.

6.10 Pathophysiology

ID is defined as a lower total body iron content that occurs in phases over a negative iron balance when the body loses more iron than it absorbs in the intestines. These phases are frequently called as IDA; iron deficiency erythropoiesis; loss of iron. As a result, the severity of ID can vary from reduced iron reserves that have little functional impact (Stages 1 and 2) to severe anaemia caused by enzyme shortages in tissue that retain iron (Stage 3). Determining the iron status in the laboratory helps identify these three stages. [41-44]

<u>Stages of Iron Deficiency</u>

It is divided into 3 stages, [6]

Stage 1(pre-latent) is known as Storage Iron Depletion

- Enhanced usage than intake, without Hb drop and alteration in RBC morphology, reduction in ferritin levels without reduced serum iron levels.

- Bone marrow aspirate- decrease or absent iron stain.
- Hb, MCV, Transferrin Saturation-Normal
- No clinical manifestations.

Stage 2(latent) is known as iron-deficient erythropoiesis.

- Iron reserves are diminished, but blood haemoglobin levels remain normal.
- RBC: hypochromic, microcytic
- Increase in TIBC

Stage 3 is iron deficiency anaemia

- Hb reduces with hypochromic and microcytic RBC morphology.
- Clinical manifestations occur

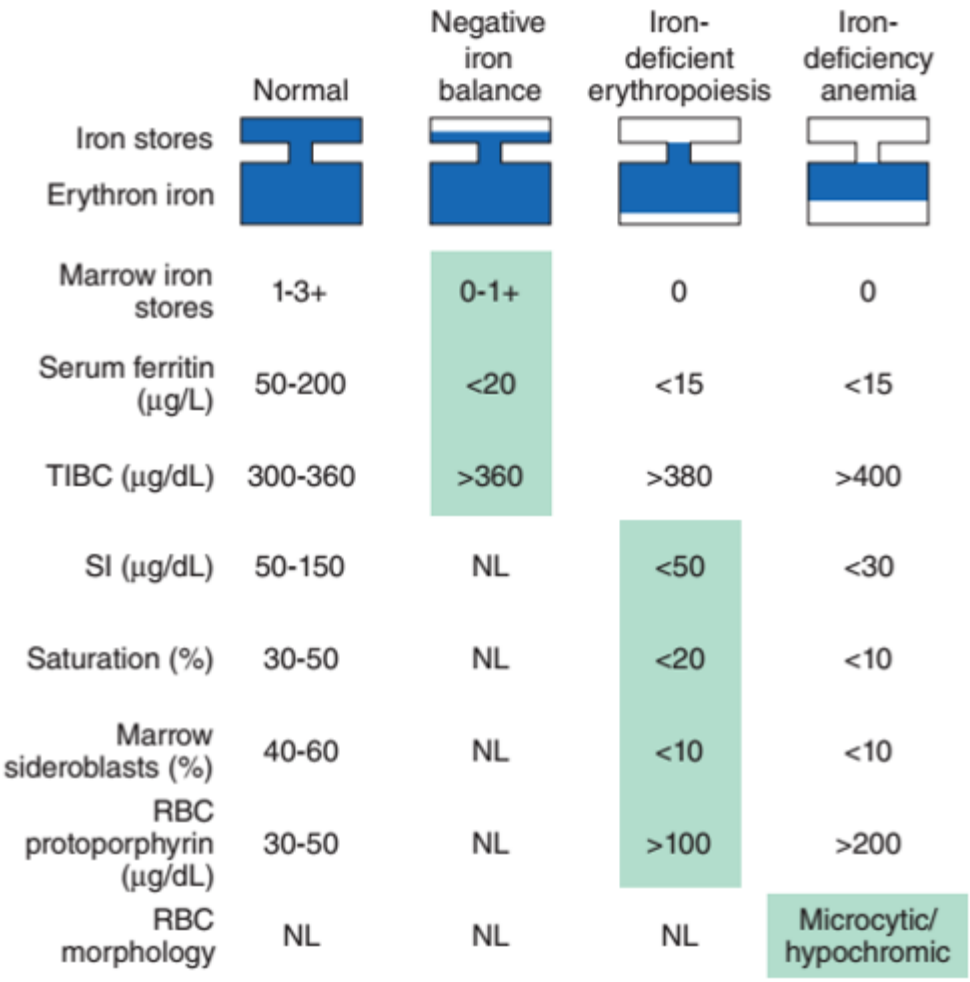


Figure 8: Stages of IDA[28]

6.11 Clinical presentation [30-32]

Symptoms of Mild Anemia: Mostly Asymptomatic

Symptoms of Moderate Anemia:

1**. Fatigue and Weakness:** Fatigue occurs when the body cannot generate enough iron to produce Hb, which is essential for transporting oxygen throughout

the body. Without sufficient Hb, tissues and muscles have minimal O2, leading to fatigue, while your heart works harder to circulate oxygenated blood.

2. Pallor: Pale skin, particularly noticeable on the face, inner eyelids, and nails, is often a symptom of IDA. Haemoglobin is responsible for carrying O2 and contains iron, and its red colour contributes to the reddish hue of blood. When iron levels are low, the production of haemoglobin decreases, and the blood may become less red, leading to a paler appearance in the skin. This is why people with iron deficiency anaemia often have noticeable paleness in these areas.

3. Shortness of Breath: When Hb levels are reduced due to iron deficiency, O2 levels in the body are also generally reduced. Consequently, the breathing rate increases as the body works to take in more O2, leading to a shortage of breath.

4. Dizziness and Lightheadedness: Feeling dizzy or lightheaded is common, especially when standing up quickly or after physical activity.

5. Rapid or Irregular Heartbeat: The heart compensates for the lack of oxygen in the blood by pumping more rapidly, leading to palpitations or an irregular heartbeat.

6. Headaches: Frequent headaches can occur due to insufficient oxygen reaching the brain.

7. Concentration Difficulties: Anemic pregnant women may experience difficulty concentrating or focusing on tasks.

8. Leg Cramps: Cramps, especially in the legs, can be a symptom due to poor oxygenation of the muscles.

9. Changes inside or outside the mouth can sometimes signal IDA. Swollen, inflamed, pale, or smooth mucous membranes, burning sensation on the tongue, dry mouth, burning feeling in the mouth, sore red cracks at the corners of the mouth, and mouth ulcers are the symptoms.

10. Loss of appetite

Symptoms of Severe Anemia: [31]

1. Extreme Fatigue and Weakness: Women with severe anaemia often feel exhausted and extremely weak, with difficulty performing even basic daily activities.

2. Severe Shortness of Breath: Breathing becomes more labored, even at rest, and one can feel like one cannot catch one's breath.

3. Chest Pain: The heart works harder to supply oxygen, which can lead to chest pain or angina, especially in severe cases.

4. Swelling: Edema or swelling, particularly in the legs and ankles, may occur due to poor circulation and fluid buildup.

5. Fainting or Syncope: Severe anaemia can cause fainting spells due to significantly reduced blood and oxygen flow to the brain.

6. Cold Hands and Feet: Poor blood circulation can result in a constant feeling of coldness in the extremities.

7. Pica: An eating condition called pica is characterized by atypical cravings for non-nutritional foods. Eating Ice, dirt/clay, and starch are the most prevalent dysphagias reported in ID patients.

8. Severe Pallor: Pronounced paleness that is easily noticeable and affects not just the face but also the gums, lips, and the inside of the mouth.

9. Rapid, Irregular Heartbeat: A markedly fast and sometimes irregular heartbeat as the heart struggles to supply enough oxygen to the body.

10. Cognitive Disturbances: Severe anaemia can affect mental function, leading to confusion or even signs of cognitive impairment.

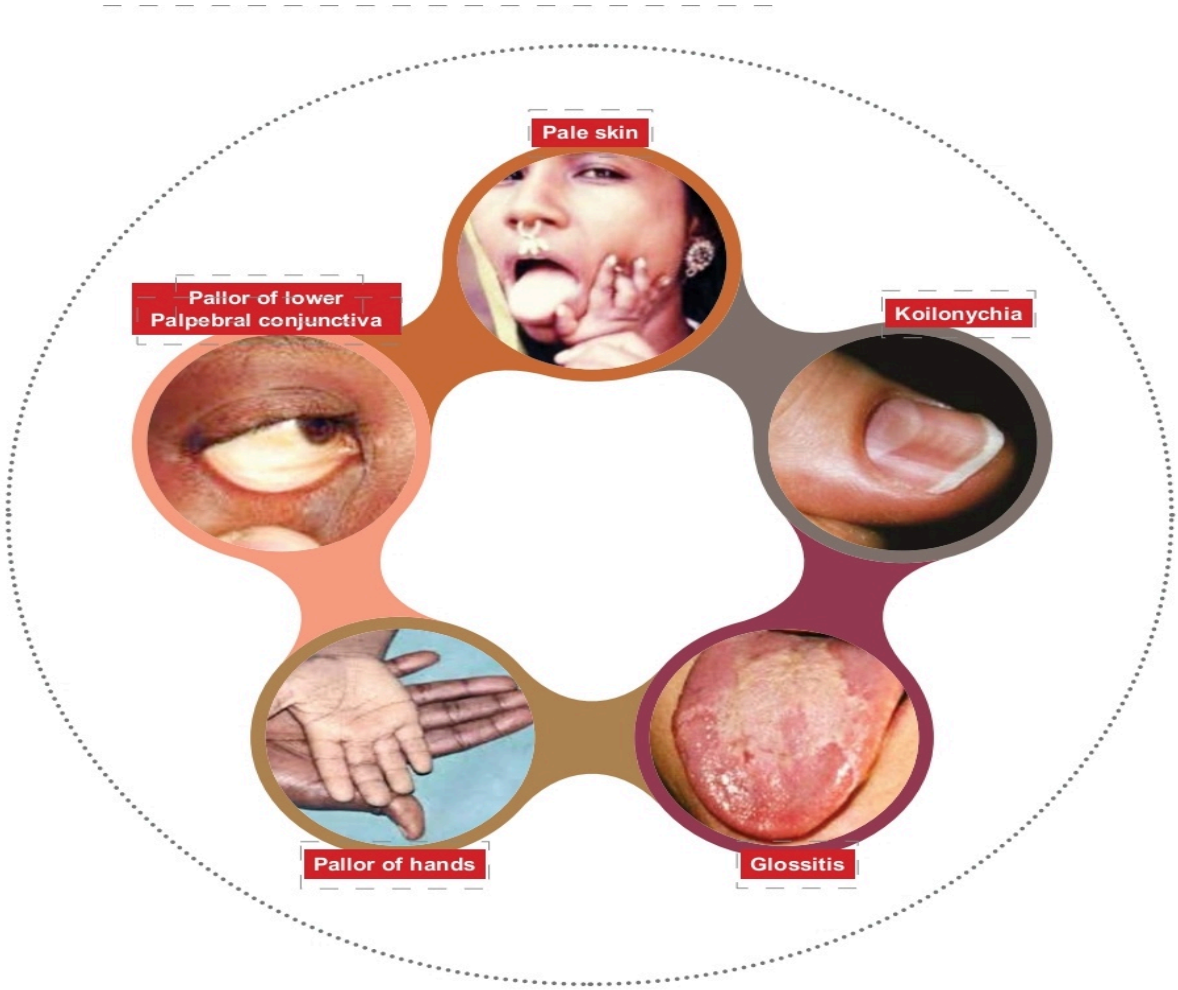


Figure 9: Signs of anaemia [66]

6.12 Laboratory evaluation of Iron deficiency anaemia

When body iron transitions from replete to deficient or excess, there is a corresponding change in clinically meaningful indices of iron status. Coexisting conditions can also have an impact on test outcomes. It is advised to employ numerous metrics to assess iron status because no single signal, or combination of indicators, discloses genuine body iron status in all situations.[21] A total iron-binding capacity (TIBC), serum iron, serum ferritin, and serum transferrin receptor (sTfR) titration, as well as a complete blood count (CBC) with reticulocyte indices, are

among the laboratory tests used to assess iron status. The zinc protoporphyrin (ZPP) assay indirectly measures iron availability.

A) Complete blood count (CBC)

In iron deficiency (ID), microcytic and hypochromic red blood cells gradually replace normocytic and normochromic ones. The rate at which aberrant cells displace the normal population is a function of both the availability and degree of iron demand. As the normocytic cells are replaced by microcytic cells, the RDW rises. Presently available data indicates that the most effective diagnostic test for functional iron shortage is the proportion of hypochromic cells reported by specific haematology analyzers.[23]

B) Transferrin can be quantified as a protein using immunochemical techniques. Still, it is typically evaluated functionally as the highest amount of iron that can be bound in the serum since the per cent saturation (with iron) helps in the differential diagnosis of anaemia (TIBC). The % saturation is determined using the measured serum iron and TIBC. [24]

C) Serum iron and TIBC typically fluctuate in tandem with changes in the amount of total body storage iron. Serum iron rises, and TIBC falls as storage iron increases; on the other hand, serum iron falls, and TIBC increases if storage iron falls or is missing. ID is indicated by a transferrin saturation of less than 15%,[25] while iron overload and perhaps hemochromatosis are suggested by more than 50% saturation.[26]

D) Serum ferritin levels are measurable and correlate approximately with the body's stored iron levels. Serum ferritin levels greater than 1000 mcg/L indicate iron overload, while those less than 2 mcg/L often suggest a reduction in iron storage.[21]

Reduced blood ferritin levels, which are 89% sensitive to identifying ID,[20] maybe the initial sign of developing IDA. Serum ferritin levels decline prior to the depletion of mobilizable iron stores. Because ferritin is an acute phase reactant, serum ferritin levels in viral or inflammatory conditions should be interpreted cautiously. If further iron status tests are not taken into account, concurrent ID may be undetected.

Sensitive immunoassay techniques can detect TfR in serum (sTfR) at negligible levels. The quantity of cellular receptors is reflected in the level of circulating sTfR. Because cellular receptor synthesis rises in low-iron cells, the sTfR is inversely correlated with body iron levels. The bulk of sTfR is produced by the bone marrow's erythroid cells, whose concentration closely correlates with erythroid activity and reticulocyte count. Concurrent illness conditions do not affect serum ferritin levels, but they do on sTfR levels. Circulating sTfR aids in the differential diagnosis of iron-deficiency anaemia but not chronic illness anaemia.[37]

E) Peripheral blood

The blood picture in a well-developed ID is microcytic and hypochromic. Because ID develops progressively, any gradation between the well-

developed microcytic hypochromic iron-deficient blood picture and normal can occur. MCV and MCH are not diagnostic parameters but help monitor trends of ID over some time.[23]

Peripheral blood smears of patients with IDA show microcytic (small) and hypochromic (pale) red blood cells, often accompanied by anisocytosis (variation in cell size) and poikilocytosis (variation in cell shape). Commonly observed shapes include elliptocytes and pencil cells. Additionally, the reticulocyte count may be elevated as the marrow attempts to increase red blood cell production.[48]

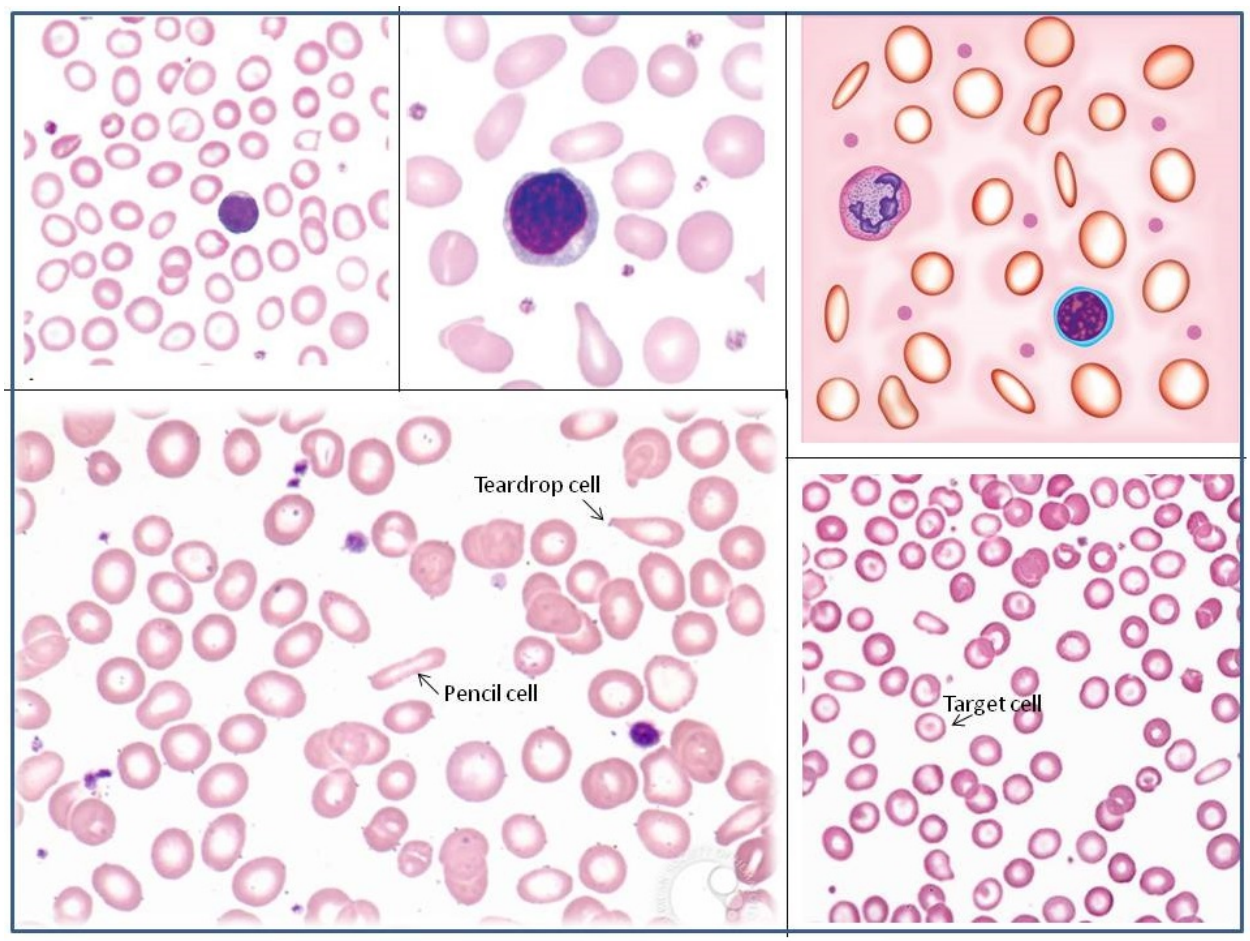


Figure 10: Histopathology report [51]

If the patient is iron deficient and has a coexisting folate or vitamin B12 deficit (causes of macrocytic anaemia), the normal blood picture may be obscured. In some situations, microcytosis might not appear until after starting vitamin B12/folic acid replacement medication. When the reticulocyte production index (RPI) is less than 2, the reticulocyte count is reduced in the severity of the anaemia. The relative and absolute quantity of reticulocytes can be normal or slightly elevated. In those who are not anaemic, a lower CHr and a higher proportion of hypochromic red blood cells are early signs of iron-restricted erythropoiesis.[43]

When evaluating the iron required for erythropoiesis in patients receiving chronic hemodialysis, the CHr has been more accurate than the presence of hypochromic red blood cells. Leukocyte counts are typically normal, but in long-term situations or following bleeding, they may rise due to chronic marrow stimulation. Concomitant hookworm infection may cause eosinophilia.

Normal, elevated, or lowered platelets can occur. Thrombocytosis, caused by prolonged blood loss, has been linked to ID and is commonly observed alongside ID. Patients with severe or chronic anaemia may develop thrombocytopenia, particularly if they also have a folate deficit. Treatment that restores iron stores may be able to repair irregularities in platelet numbers.

F) Bone Marrow Examination

A notable reduction in iron stores was confirmed by Prussian blue staining, which showed decreased or absent iron in macrophages and erythroblasts. The

erythroid precursors are typically more numerous (erythroid hyperplasia), reflecting the bone marrow's attempt to compensate for anaemia by increasing red blood cell production. However, these precursors are smaller and paler than normal, indicating defective haemoglobin synthesis. [51]

Anaemia in India is multifaceted, with various types contributing to the overall burden of the disease. Effective public health interventions, including improved nutrition, screening for hereditary anaemias, and management of chronic diseases, are essential to reduce the prevalence and impact of anaemia in the population.

Therapy

Treatment for anaemia involves treating the underlying illness, giving iron, and monitoring the patient's reaction once the cause of the anaemia has been determined. The usual treatment for anaemia is to take ferrous sulfate orally. Parenteral iron therapy is only seldom needed for exceptional circumstances. For patients with chronic renal illness receiving therapy with recombinant human erythropoietin. When iron is administered to individuals who are iron deficient, their strength, appetite, and overall sense of well-being return in 3 to 5 days. Still, their anaemia does not go away for weeks.

An increase of 1 gm of haemoglobin in a month is considered a response to iron therapy. Reticulocyte counts and the immature reticulocyte fraction (IRF) provide an overview of recent red blood cell synthesis because reticulocytes are

newly produced red blood cells from the bone marrow. The reticulocyte response to iron therapy starts on the third day after the therapy has begun, peaks approximately the ninth or tenth day (4–10% reticulocytes), and then starts to diminish.[49]

After haemoglobin has been restored to normal, prolonged therapy with tiny amounts of iron salts (typically for 6 months) may be necessary to rebuild iron stores. If anaemia does not improve after one month of treatment, more testing is required.

Ferrous Salts for O.I Therapy and Supplementation:

Ferrous salts used to be the preferred choice for O.I. therapy, with various bivalent iron salts frequently utilized for supplementing. These comprise ferrous sulphate, fumarate, gluconate, glutamate, succinate, and lactate. Ferrous ascorbate is distinguished by its solubility at the alkaline pH of the small intestine, providing a superior option compared to other ferrous salts in iron supplements. [67]

Ferrous ascorbate

Ferrous ascorbate is a ferrous form combined with ascorbic acid. Ferrous ascorbate possesses a distinctive chemistry characterized by a substantial iron concentration and the simultaneous presence of ascorbate within the same molecule. Ascorbic acid enhances the absorption of medical iron. Ferrous ascorbate possesses a substantial iron concentration along with ascorbic acid. Ferrous ascorbate elicits a

rapid response, with improvements in haemoglobin levels observable as soon as 15 days following the commencement of treatment.[68] The notable efficacy, superior safety, and tolerability of ferrous ascorbate can be attributed to the benefits of its chemical state, which includes enhanced bioavailability and iron utilization.

Pharmacokinetics

Iron is prone to oxidation by the alkaline environment of the gastrointestinal tract and by dietary components. In the ascorbate formulation, iron absorption is optimized due to:

(i) The suppression of the change of ferrous to ferric iron, enhancing uptake,

(ii) The mitigation of the impact of phytates, phosphates, and oxalates on iron absorption and

(iii) The prevention of the generation of insoluble iron complexes that hinder absorption.[15,19]

Ferrous ascorbate possesses intrinsic characteristics that enhance its absorption. Ferrous ascorbate exhibits a solubility-enhancing action of ascorbate within a pH range of 6 to 8.[67] Twenty Certain unique manufacturing processes, such as advanced coating technology (ACT), enhance the stability of ferrous ascorbate chelate and restrict its dissociation in the stomach, resulting in improved absorption.

Bioavailability

Ferrous ascorbate exhibits increased bioavailability.[68] Numerous investigations have indicated a comparably elevated absorption rate (39–43.7%) of iron from ferrous ascorbate, with absorption rates reaching up to 67% observed in cases of iron deficiency anaemia.[67, 68]

The superior absorption of iron from ferrous ascorbate relative to ferrous sulphate is attributed to ascorbate's ability to inhibit or slow the oxidation of ferrous iron and to the formation of a chelate between ferrous iron and ascorbate.

I.S. is a combination of iron hydroxide and sucrose in an aqueous solution. The molecular weight of I.S. is between 34,000 and 60,000 Daltons.[67] I.S. is delivered either by intravenous bolus injection over 5-10 minutes or as a brief infusion in 100 ml of normal saline over 20-30 minutes.[68] A maximum daily bolus dose of 200 mg may be administered at once, not exceeding three times per week.[69] Common adverse effects include a metallic taste, nausea, disorientation, and localized discomfort.

Iron isomaltoside 1000 (FDI) One of the most recent IV iron formulations on the market is FDI. It is made up of a carbohydrate moiety and iron that is firmly coupled in a matrix structure. It was first introduced in Europe in 2010.[70] The matrix structure prevents possible toxicity from the release of labile iron by allowing a regulated and gradual release of iron to iron-binding proteins. Non-clinical

renotoxicology trials have examined FDI. Rabbits exhibiting fetal abnormalities were given supratherapeutic doses.[67]

At the suggested therapeutic dose, there is thought to be little chance of teratogenic or foetotoxic consequences. Unexpected safety risks have not been found in the few published trials on using FDI to treat iron deficient anaemia. If the benefit is determined to exceed the possible risk to the mother and the fetus, FDI should only be used during the second and third trimesters of pregnancy.

Table 1: Intravenous preparations available[68]

	Iron (III) hydroxide dextran complex	Iron (III) hydroxide sucrose complex	Iron (III) carboxymaltose	Iron (III) isomaltoside
Dose of elemental iron	50mg/ml	20mg/ml	50mg/ml	100mg/ml
Routes of administration	Slow intravenous & IV infusion	Slow intravenous & infusion	Slow intravenous injection & infusion	Slow intravenous & infusion
Able to administer total dose	Yes (up to 20mg/kg body weight over 4-6 hours)	No	Yes (up to 20mg/kg body weight maximum of 1000mg/week over 15mins)	Yes (up to 20mg/Kg body weight over 1 hour)
Half life	5 hours	20 hours	7-12 hours	5 hours
Dosage	100-200mg per IV injection up to 3 times a week. 20mg/kg body weight over 4-6 hours.	Total IV single dose no more than 200mg, can be repeated up to 3 times in 1 week	1000mg by IV injection up to 15mg/kg/week. Total dose infusion up to 20mg/kg body weight. Maximum weekly dose of 1000mg that can be administered over 15mins.	100-200mg per IV injection up to 3 times a week. 20mg/Kg body weight per week.
Use in pregnancy	Not advised in 1st trimester	Not advised in 1st trimester	Not advised in 1st trimester	Not advised in 1st trimester
Adverse drug related events	5% chance	0.5-1.5% chance	3% chance	>1% chance

NEED FOR THE STUDY

The Government of India has implemented comprehensive guidelines to prevent and manage anaemia during pregnancy. These programs emphasize iron and folic acid supplementation, deworming, and dietary diversification to improve maternal health outcomes.

Pregnant women are advised to take daily oral supplements of 60 mg of elemental iron and 500 µg of folic acid. Supplementation should commence after the first trimester (from the fourth month) and continue throughout the pregnancy and for 180 days postpartum. Traditionally, O.I. supplements are recommended for preventing anaemia, but their efficacy is limited by poor absorption, G.I. side effects, and reduced compliance.

In contrast, intravenous (IV) I.S. offers a direct and efficient method to replenish iron stores, bypassing gastrointestinal limitations. IV iron has been increasingly used in cases of moderate to severe anaemia, but its role in prophylaxis remains underexplored. While it provides a rapid haematological improvement, it requires medical supervision and has associated costs.

I.S. is an intravenous iron formulation that enables rapid and direct delivery of iron into the blood, skipping the digestive system. Ferrous ascorbate is a compound of iron and ascorbic acid, improving the absorption of iron and reducing negative impacts.[3-8]

The studies by Rudra S et al.,[70] Deeba S et al.,[71] Gogineni S, Vemulapalli P.,[72] and Sudakshina K et al.[73] were done on the efficacy and safety of the I.S. and Oral ferrous ascorbate. Of these, Gogineni S and Vemulapalli P [72] had done a study on the prophylactic management of anaemia by supplementing I.S. and Oral ferrous ascorbate. They observed only a rise in Hb, but in this randomized control trial, improvement in Hb as well as Serum ferritin was observed.

PREVIOUS STUDIES

Gogineni S and Vemulapalli P (2015) [72] did a prophylactic study among one hundred pregnant women between 20 and 24 weeks of gestation and haemoglobin greater than 10.5 gm%. Group A (50 women) received three doses of I.S., whereas Group B used oral ferrous ascorbate. The mean Hb% difference in Group A was 0.3 with a standard deviation of 0.18, compared to 0.12 with a standard deviation of 0.88 in Group B, indicating no significant difference in Hb% increase between the two groups. Mild anaemia was seen in 6% of Group A and 18% of Group B. In Group A, 2% of subjects experienced a mild rash following the I.S. infusion, while 6% reported pain during the injection. Ninety-two per cent of cases did not exhibit any notable adverse effects. Just 34% of patients in Group B had no symptoms, compared to 12% had constipation, 24% had gastritis, and 30% had nausea and vomiting. Reduced compliance was observed in 4% of Group A and 40%

of Group B. The cost of therapy for Group A was 1600/- for three doses, whereas Group B had a price of 1500/-, indicating comparability.

Bhatt AK et al. (2020) [74] conducted study on 154 women pregnant women between 20-24 weeks and divided into two groups, Group A received 200mg in 100ml N.S. infusion over 15-20mins at 20-24 weeks, 25-28 weeks, 29-32weeks and Group B received oral ferrous sulfate 200mg tablet once daily upto 32 weeks. Hb was estimated in both groups at 20-24 weeks, 25-28 weeks, and 29- 32 weeks. The study concluded that intravenous I.S. can prevent anaemia in the rapidly rising haemoglobin.

Sudakshina K et al. study (2022) [73] among 84 antenatal (less Hb%) found an increase in the mean haemoglobin level from 10.4 ± 0.5 to 11.1 ± 0.6 in the Iron ascorbate group and 8.5 ± 0.3 to 10.9+ 0.6 in I.S group. There was a significant improvement in the mean change in Hb level between the two groups. RBC count increased from 3.7 ± 0.3 to 4.0 ± 0.2 and 3.2 ± 0.1 to 3.8 ± 0.2, respectively, and MCHC from 32 ± 1 to 33 ± 2 and 31 ± 2 to 33 ± 2, respectively. MCV count increased from 73 ± 4 to 84 ± 5, and 63 ± 3 to 78 ± 4, respectively; MCH count increased from 29 ± 1 to 32 ± 1; 27 ±1 to 30 ± 2, respectively; WBC count increased from 6451.42 ± 1446.82 to 6604.42 ± 1502.87 and 6886.19 ± 1444.7 to 7001.8 ±1425.4, respectively. Total adverse effects were seen in 17.4% in the ascorbate

group and 66.7% in the I.S. group. The mean difference within the groups regarding Hb, MCV, MCHC, and MCH is significant.

Agalya M et al.[67] **(2020)** did a study among 50 antenatal anaemic women of 14-34 weeks. The oral group (25 cases) had taken ferrous ascorbate, while the parenteral group (25 cases) received I.S. On the 14th day, the intravenous group exhibited a Hb level of 9.64 ± 0.45 gm/dl, whereas the oral group had 9.19 ± 0.49. On the 28th day, the intravenous group exhibited a haemoglobin level of 11.41 ± 0.51, whereas the oral group displayed 10.76 ± 0.49. This mean difference was statistically significant on the 14th and 28th days. The blood ferritin level in both the intravenous and oral groups exceeded the baseline level, with a greater increase observed in the intravenous iron-sucrose group. The intravenous group exhibited a mean ferritin level of 60.92 ± 6.90, whereas the oral group demonstrated a level of 50.68 ± 2.64, with a statistically significant difference. Only 20% of the IV group experienced side effects, whereas 80% exhibited no negative effects. In the oral group, 32% suffered adverse effects, whereas 68% did not report any side effects. Among the IV group, 4% of cases reported epigastric discomfort, 8% of cases experienced nausea, and 8% had hot flushes; no patients exhibited vomiting, metallic taste, or constipation. In the oral group, 8% of cases experienced nausea, 4% of women reported sickness, and 8% of cases suffered from constipation. Of the

total, 8% experienced gastritis, 8% reported a metallic taste, 4% had vomiting, and none experienced hot flushes. The results were statistically insignificant.

In the **Prajapati SM and Patel MK study (2019),**[75] the weight range was 40-75 kg. The mean weight was 52.06 ± 5.60. The average age was 26.25 ± 2.05. The mean gestational age was 28.32 ± 1.8. In the current study, a total of 35% were primigravidas, 32.5% of cases were second gravidas, and 32.5% of cases were third or more gravidas. The hemoglobin range following IV iron therapy was 10.2-12 g%. The post-treatment range for O.I. was 9.8-10.6 g%. The mean haemoglobin level was 7.9 g% prior to therapy and 10.8 g% post-treatment for intravenous iron. In contrast, it was 7.8 g% before treatment and 10.2 g% after treatment for O.I., resulting in increases of 2.9% and 2.4% over six weeks, respectively. The range of PCV prior to intravenous iron administration was 24-34%, and prior to O.I. supplementation was 25-35%. The range of PCV following intravenous iron administration was 32-39%, while after O.I., it was 30-38%. The mean PCV was 28.36% prior to therapy and increased to 34.06% following intravenous iron administration. The mean PCV was 29.6% prior to treatment and increased to 33.8% following O.I administration. The increase in PCV was 5.7% for intravenous iron and 4.2% for O.I. This increase is markedly substantial. Adverse events occurred in 10% of the research groups. Although the unfavourable effects associated with IV I.S were reduced, this difference was insignificant. The range of PCV prior to

intravenous iron administration was 24-34%, and prior to O.I. supplementation was 25-35%. The range of PCV following intravenous iron administration was 32-39%, while after O.I., it was 30-38%. The mean PCV was 28.36% prior to therapy and increased to 34.06% following intravenous iron administration. The mean PCV was 29.6% prior to treatment and increased to 33.8% following O.I administration. The increase in PCV was 5.7% for intravenous iron and 4.2% for O.I. This increase is markedly substantial. Adverse events occurred in 10% of the research groups. Although the unfavourable effects associated with IV I.S were reduced, this difference was insignificant.

Bhavi SB and Jaju PB (2017) study,[76] among 112 patients, 52% were aged between 21 and 25 years, with the majority being multigravida at 31 to 34 weeks of gestation. A significant elevation in Hemoglobin levels in group A (O.I), increasing from 91.4 ± 11 to 106.5 ± 10.3, and in group B (intravenous iron), rising from 89 ± 10.7 to 106.4 ± 13 after 4 weeks, indicating high significance. Intravenous iron demonstrated a significant difference in serum ferritin levels after 4 weeks of treatment compared to O.I., indicating high relevance. The alteration in Hb % in group B was 22 ± 11.5, greatly surpassing the 12±9.1 observed in group A, indicating statistical significance. The alteration in serum ferritin for group B was 112.17 ± 98.15, which was significantly elevated compared to 22.71 ± 11.32 in group A, indicating statistical significance. Among the 32% of cases who received

O.I., 11 to 20 g/L increases in Hb were seen. In contrast, 55% of cases in the IV I.S group exhibited a more substantial improvement above 20 g/L, while 11% of cases in the O.I group showed such an increase.

Tikkha I and Rai H (2017) [77] conducted a study that included 100 prenatal women (Hb%), indicating that the mean levels of haemoglobin and ferritin in Group II were considerably greater than in Group I. The average haemoglobin level rose significantly over time for both groups. In Group I, there was no difference in mean Hb levels at Day 0 and after 3 weeks; nevertheless, a substantial rise was observed after 8 weeks compared to Day 0. In Group I, there was no statistically significant change in the mean PCV at Day 0 and after 3 weeks; nevertheless, it considerably increased after 8 weeks compared to Day 0. In Group I, there was no significant difference in the mean ferritin levels at Day 0 and after 3 weeks; however, a substantial rise was observed after 8 weeks compared to Day 0.

Rudra S et al. study (2016), [70] among 200 antenatal (less Hb%) women, found that the mean age in the Sucrose group was 25.08 ± 3.32, and in the ascorbate group was 25.12 ± 3.73, and this mean difference was non-significant. Multi gravida were 78% and 80%, respectively. The mean BMI was 22.18 ± 2.42, and 22.13 ± 2.37, respectively, and this mean difference was non-significant. Mean gestational was 27.61 ±2.43 and 27.76 ±2.31, respectively; this mean difference was insignificant. Reticulocyte Count increased in the IV sucrose group from 0.77 ± 0.28

to 3.6 ± 1.07 and increased from 0.76 ± 0.25 to 1.44 ± 0.35 in the Oral group. Mean Hb increased from 7.81 ± 0.44 to 10.93 ± 0.60 and from 7.88 ± 0.45 to 10.30 ± 0.56, respectively. PCV increased from 24.59 ± 0.759 to 33.09 ± 1.15 and from 24.80 ± 0.896 to 32.25 ± 1.24, respectively. MCV increased from 71.88 ± 1.12 to 89.06 ± 1.74 and from 72.19 ± 1.18 to 86.37 ± 4.44, respectively. MCH increased from 23.71 ± 0.41 to 29.47 ± 1.25 and from 23.78 ± 0.44 to 28.27 ± 1.82, respectively. MCHC increased from 29.72 ± 0.41 to 33.89 ± 0.67 and from 29.81 ± 0.44 to 33.31 ± 1.15, respectively. Serum ferritin increased from 10.48 ± 1.46 to 58.26 ± 11.16 and from 10.43 ± 1.86 to 42.10 ± 8.55, respectively. The mean difference between the two groups was significant in all the parameters at all time intervals.

Thobbi VA and Bijapur ZN (2016) [78] conducted a study among 20 to 36 weeks of anaemic pregnant mothers. Group A (n=100) received Ferrous Ascorbate, and Group B (n=100) received I.S. The average change in Hb six weeks after treatment was 2.2 in the intravenous and 1.2 in the oral groups, respectively. At six weeks following medication, serum ferritin levels were 93.0 in the intravenous Group and 35.3 in the oral Group. The increase in haemoglobin every 3 weeks in the intravenous Group was around 1 gram, but in the oral Group, the increase was 0.6 grams. This indicates that the rise in the IV group was much greater than in the oral Group. None of the patients in the orally treated Group experienced increased serum ferritin levels above 150 ng/ml. The oral Group reported a greater incidence

of side effects than the IV group. A maximum of 6% of individuals in the oral Group reported nausea. Three per cent had vomiting, while two per cent each reported diarrhoea and gastritis. In the IV group, 3% reported pain, while 2% and 1% reported burning and swelling.

Rudra S et al. (2016) [70] conducted a study among 24 to 34-week anaemic pregnant women, with 100 cases of Group A received I.S. and 100 cases of Group B received oral ferrous ascorbate. The increase in haemoglobin (Hb) and other indices was greater in the intravenous iron group compared to the O.I group at each point, with a statistically significant difference. The Hb levels at one week were 7.86 ± 0.43 and 7.93 ± 0.41, demonstrating no significant increase after the first week of medication. Nonetheless, haemoglobin levels began to rise consistently from the second week onward in both groups, with this upward tendency persisting until delivery. At each measurement point, the IV group had a significantly higher increase in Hb levels than the oral Group. At 2 weeks, the IV group had a Hb of 8.36 ± 0.43 vs 8.11 ± 0.45; at 4 weeks, it was 9.80 ± 0.36 vs 9.17 ± 0.47; at delivery, it was 11.48 ± 0.61 vs 10.90 ± 0.62. Likewise, all other laboratory measures and serum ferritin assessed showed a more significant increase in group A at each evaluation point.

In the **Abhilashini GD et al. (2014),** [79] study, in the O.I group, 38% of cases had severe anaemia, while 54% in the I.S group. Of the total, 52% of cases in the I.S. group and 42% in the O.I. group exhibited MCV values between 61-70. The mean MCV in the intravenous I.S. group was 71.07, while in the O.I. group, it was 73.07, with no significant significance. The mean difference in haemoglobin between recruitment and the second week was statistically significant when comparing the two groups; however, the mean differences in MCV and PCV were insignificant. The mean differences in haemoglobin and PCV between recruitment and the fourth week were statistically significant. The mean variations in haemoglobin and PCV between recruitment and term were found to be highly important when comparing the two groups. The enhancement in haemoglobin levels in the I.S. group was significantly superior to those of the O.I. group in the 2nd week, 4th week, and at term. The variation in enhancement of MCV and PCV was nearly identical in both groups in the second week. During the fourth week and at term, the enhancement in PCV was significantly more significant in the I.S. group compared to the O.I. group. The mean RBC count in the intravenous I.S. group in the second week was 5.08%, whereas in the O.I. group, it was 4.46%.

No gastrointestinal side effects were observed in women receiving intravenous iron treatment. All patients adhered to intravenous iron therapy and O.I. supplementation. Of the total, 40% of cases in the O.I group experienced gastrointestinal side effects. The majority of patients in both groups underwent vaginal delivery. Only three patients in the intravenous I.S. group and four women

in the O.I. group underwent cesarean sections. 56% of infants in the intravenous I.S. group and 42% in the O.I. group had birth weights ranging from 2.5 to 3.5 kg, respectively. No substantial difference was seen in the birth weights between the two groups. In the sample, 62% exhibited moderate anaemia in the oral Group. The average iron needed for the intravenous I.S. group was 1057 mg, while for the O.I. group, it was 1059 mg. The average iron need in both groups was nearly identical, and the difference was insignificant. The average haemoglobin level at recruitment was 6.89 g/dL in the I.S. group and 7.16 g/dL in the O.I. group, with a statistically significant p-value of 0.039. The mean PCV at recruitment for the intravenous I.S. group was 24.61%, while the O.I. group had a PCV of 25.52%.[79]

Syal Neeru et al. (2012) [80] did a study to compare the efficacy and tolerance of oral and parenteral I.S. [I.V.I.S.] therapy with O.I [O.I.] therapy in pregnant women. Pregnant women between 14-36 weeks of gestation who were treated with I.V.I.S. or O.I. were included in this study. All patients were monitored for lab response and adverse events. They concluded that the increase in reticulocyte count and percentage of Hb was significantly higher in the I.V.I.S. group than in the O.I. group, and serum ferritin was also raised in the parenteral group compared to the O.I group.

Bencaiova G et al. (2009) [81] studied 260 antenatal women with a singleton pregnancy between the 21st and 24th week of gestation and considered into the oral and intravenous groups. Of one hundred thirty women in the intravenous Group, 75 received two doses of 200mg IV I.S, and 55 received three doses of IV I.S group.1st dose was given at 21st and 24th weeks of gestation, 2nd dose was given at 28th and 32 weeks of gestation, 3rd dose was given at 35th and 37th weeks of gestation. Women in the oral Group were given oral tablets of 80mg ferrous sulfate daily from the day of study, and they concluded that the significant difference in increased iron stores before delivery in the intravenous Group compared to the oral Group.

MATERIALS AND METHODS

The current research was conducted to compare the effect of prophylactic I.S with Oral ferrous ascorbate in Pregnant Women.

- **Study design:** Randomization study
- **Study setting**: The current research was conducted at SHRI B.M. Patil Medical College and Hospital, Karnataka.
- **Duration of the study**:18 months
- **Study population**: Antenatal women with confirmed intrauterine pregnancy of gestational age who are attending to the Obstetrics and Gynecology OPD.
- **Sample size -** 100

Calculation of sample size:

Using G*Power 3.1.9.4 software for sample size calculation, the 4th Week MCH IV (Mean=29.47, SD=1.25, and Oral (Mean=28.27, SD=1.82)) and Oral (Mean=28.27, SD=1.82)[70] require a total sample size of 80 (for each group 40, assuming equal group sizes) in order to achieve a power of 92% for detecting an inequality in the Means: Two independent means (two groups)(t-test) with 5% level of significance. A 20% dropout rate was applied to an additional 20 cases, with 50 participants in each group. So, total 100 cases included in the study.

Inclusion criteria:

1. HB ≥ 11 mg/dl
2. Singleton pregnancy
3. Those who gave written informed consent

Exclusion criteria:

1. K/c/o Haemoglobinopathies
2. Hypertensive disorders in pregnancy
3. Diabetes mellitus
4. Chronic bleeding
5. Heart diseases
6. Multiple Pregnancy

Methodology

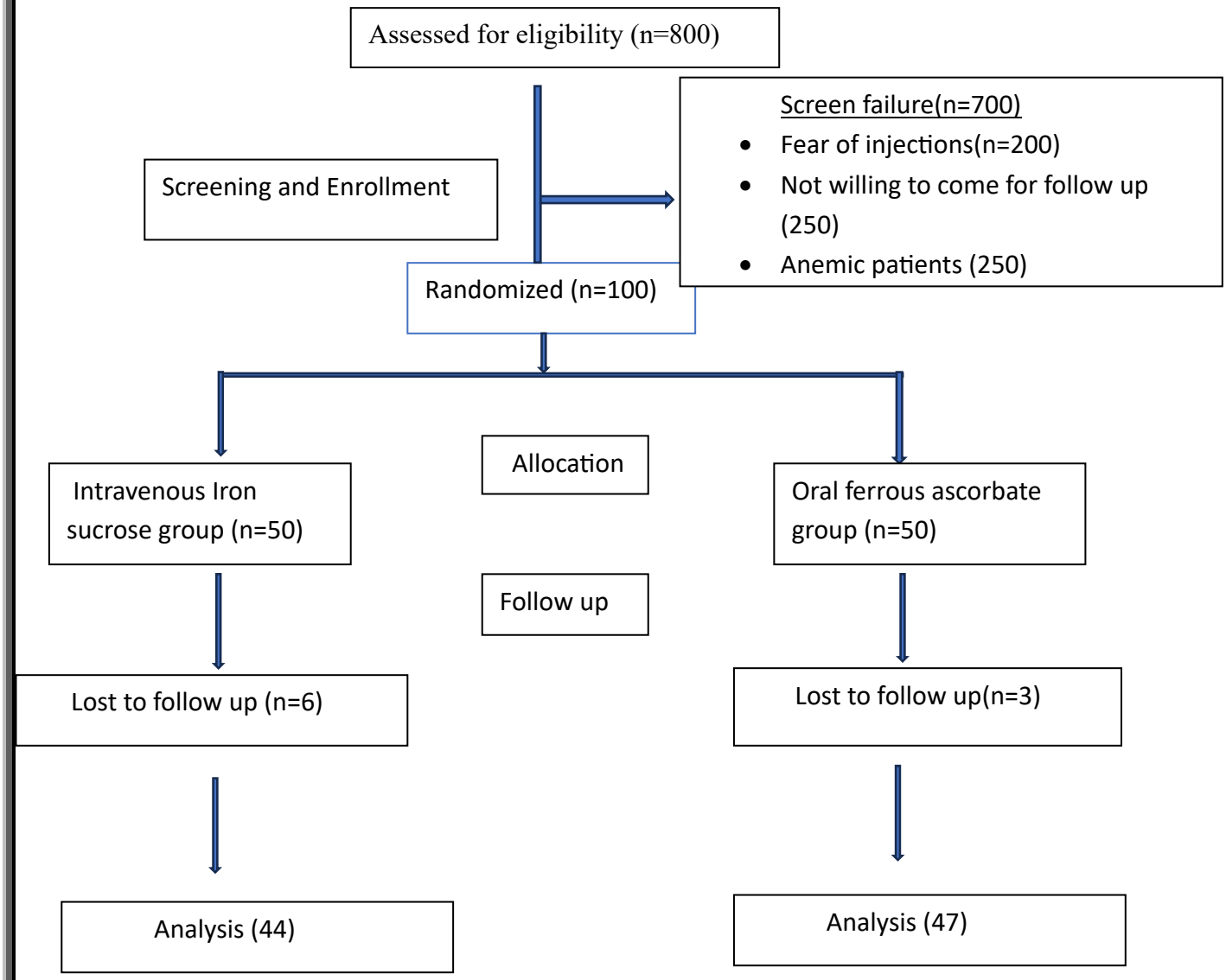
The current research was conducted after obtaining Institutional Ethics Committee permission and the consent from the antenatal women after clearly explaining about the study purpose, and the study procedure. All the antenatal women had undergone a general physical examination, and obstetric evaluation, along with all the routine blood investigations randomized into two groups

Group A: Intravenous Iron Sucrose (IV I.S) group and

Group B: Oral ferrous ascorbate (O.I) group.

Group A pregnant women received three doses of IV I.S 200mg in 100ml normal saline as an infusion over 15-20 minutes at 20-24 weeks, 24-28 weeks, and 28-32 weeks of gestation.

Group B pregnant women were administered oral ferrous ascorbate, providing 100 mg of elemental iron daily at bedtime, one hour before meals till delivery.



Hb, RBC COUNT, PCV, MCV, MCH, MCHC and Serum Ferritin were assessed before starting the prophylactic anaemia treatment, later at 4 weeks and 12 weeks.

Side effects such as Gastrointestinal effects (nausea, vomiting, epigastric pain/acidity, pain abdomen, constipation, and diarrhea), rashes, itching, chills, headache, and local pain at the injection site were observed.

PRIMARY OUTCOME

- To see efficacy of intravenous I.S in maintaining hemoglobin levels as compared to O.I in pregnancy.

SECONDARY OUTCOME

- To know the compliance and cost effectiveness of parenteral vs O.I supplementation.
- To know the safety of parenteral vs. O.I supplementation.
- To know which is better in increasing iron stores.

Statistical analysis

Data was analyzed with Microsoft Excel and Statistical Package for Social Sciences (SPSS) software version 26.0. Mean and standard deviation of the quantitative variables was measured. For categorical variables, association was estimated by using the chi-square test or Fisher's exact test. Difference between two continuous variables was measured by using z test. P value ≤ 0.05 was taken as significant.

RESULTS & ANALYSIS

The current research was conducted to compare the effect of prophylactic I.S with Oral ferrous ascorbate in Pregnant Women. Total of 100 cases were recruited and each 50 cases were allotted to the respective groups, i.e., Intra Venous Sucrose (IV Sucrose) or Group A, and Oral Ferrous Ascorbate or Group B by using Randomization technique.

Table 2: Age distribution

Age (in years)	Group A	Group B	Total
≤20	6 (13.6%)	8 (17%)	14 (15.4%)
21-25	24 (54.5%)	23 (48.9%)	47 (51.6%)
26-30	10 (22.7%)	10 (21.3%)	20 (22%)
31-35	4 (9.2%)	6 (12.8%)	10 (11%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Chi-Square test 0.61; P value 0.89(non-significant)

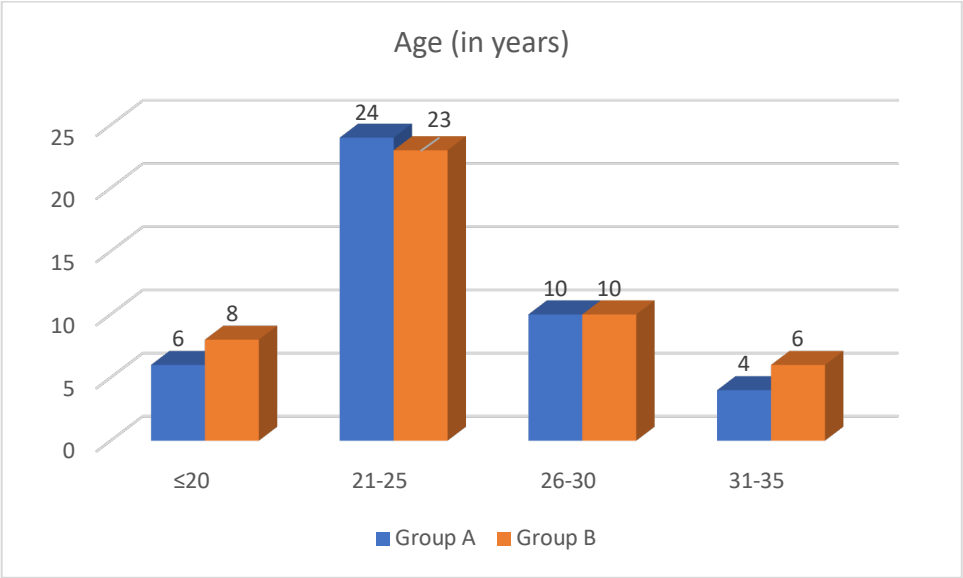


Figure 11: Age distribution

In this current research, among ≤20 years, 6 (13.6%) cases of Group A, and 8 (17%) cases of group B were seen. In the 21-25 years, 24 (54.5%) cases of Group A, and 23 (48.9%) cases of group B were seen. In the 26-30 years, 10 (22.7%) cases in Group A, and 10 (21.3%) cases in Group B. In the 31-35 years, 4 (9.1%) cases of Group A, and 6 (12.8%) cases of group B were seen.

Table 3: Residence

Residence	Group A	Group B	Total
Rural	29 (65.9%)	30 (63.8%)	59 (64.8%)
Urban	15 (34.1%)	17 (36.2%)	32 (35.2%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Chi-Square test 0.04; P value 0.84(non-significant)

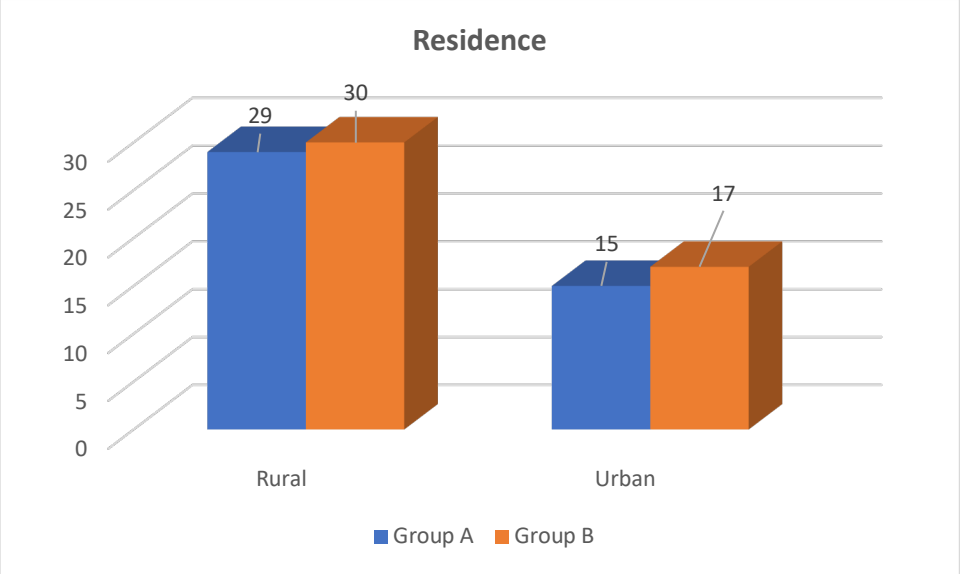


Figure 12: Residence

In the rural areas, 29 (65.9%) cases of group A and 30 (63.8%) cases of group B, and in urban areas, 15 (34.1%) cases of group A and 17 (36.2%) cases of group B were seen.

Table 4: Gravida

Gravida	Group A	Group B	Total
Primi	30 (68.2%)	31 (66%)	61 (67%)
Multi	14 (31.8%)	16 (34%)	30 (33%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Chi-Square test 0.05; P value 0.82(non-significant)

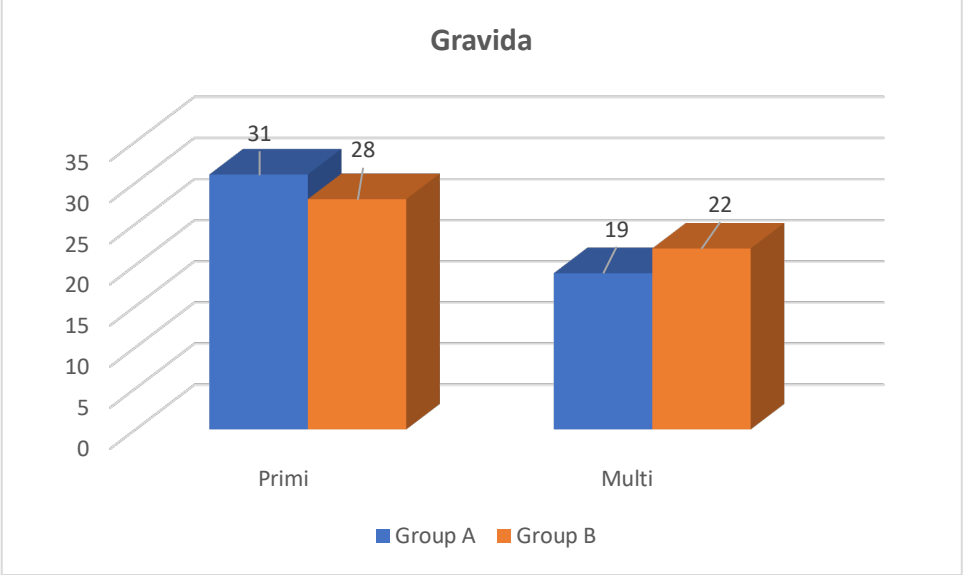


Figure 13: Gravida

Of the total cases, 30 (68.2%) cases of group A and 31 (66%) cases of group B belonged to primigravida, and 14 (31.8%) cases of group A and 16 (34%) cases of group B belonged to multi gravida.

Table 5: Mean age

	Mean age	**S.D**	**P value**
Group A	24.62	4.07	0.75(non-significant)
Group B	24.88	3.86	

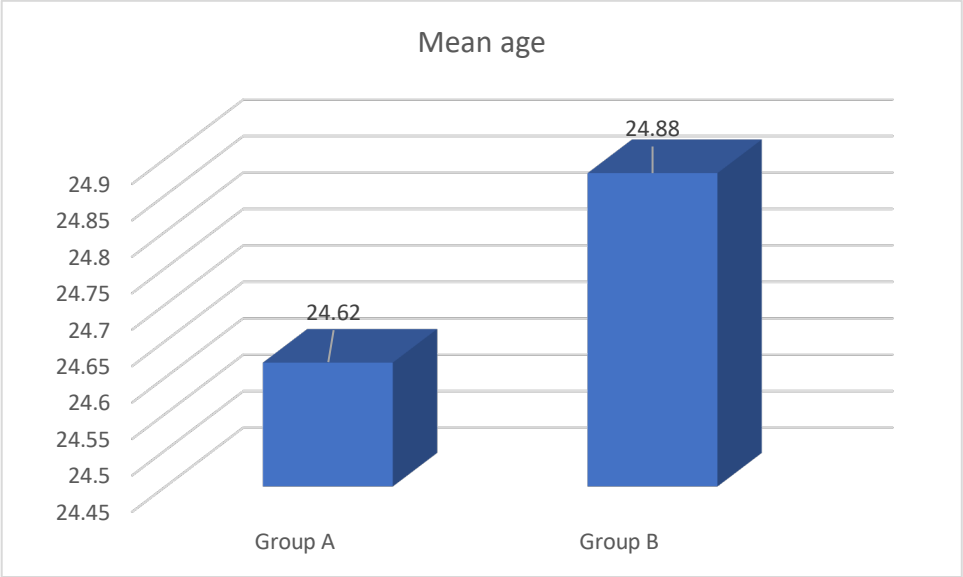


Figure 14: Mean age

The mean age of Group A was 24.62 ± 4.07 years, and Group B was 24.88 ± 3.86 years.

Table 6: Mean period of gestation at enrollment

	Mean period of gestation	S.D	P value
Group A	21.76	1.53	0.61(non-significant)
Group B	20.38	1.47	

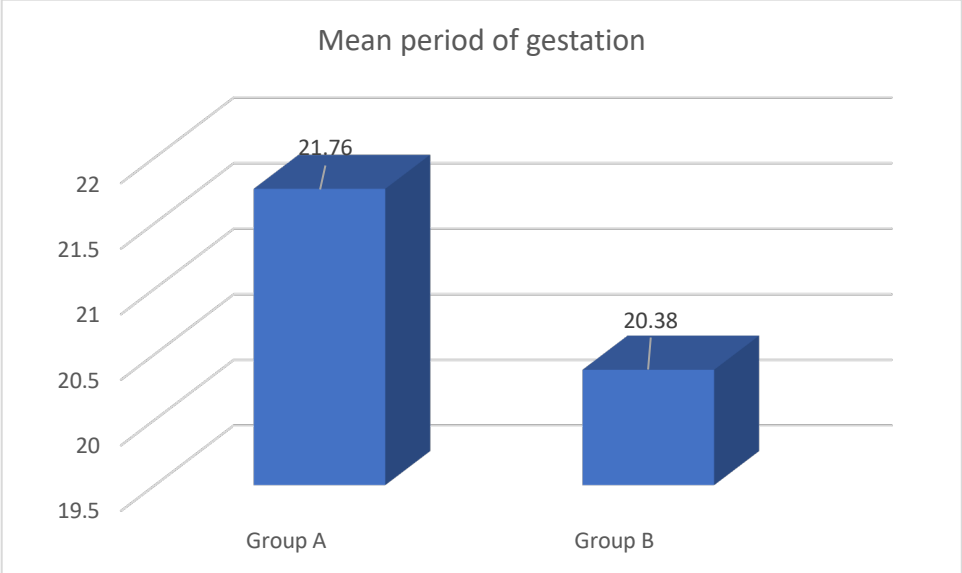


Figure 15: Mean period of gestation at enrollment

The mean period of gestation of Group A was 21.76 ± 1.53 weeks, and Group B was 20.38 ± 1.47 weeks.

Table 7: Comparison of mean Hb

Time	Group A		Group B		P value
	Mean Hb	S.D	Mean Hb	S.D	
At baseline	11.81	0.71	11.74	0.86	0.67(non-significant)
After 4 weeks	11.49	0.84	10.99	0.83	**0.005(significant)**
After 12 weeks	12.19	0.68	11.63	0.74	**0.0001(significant)**

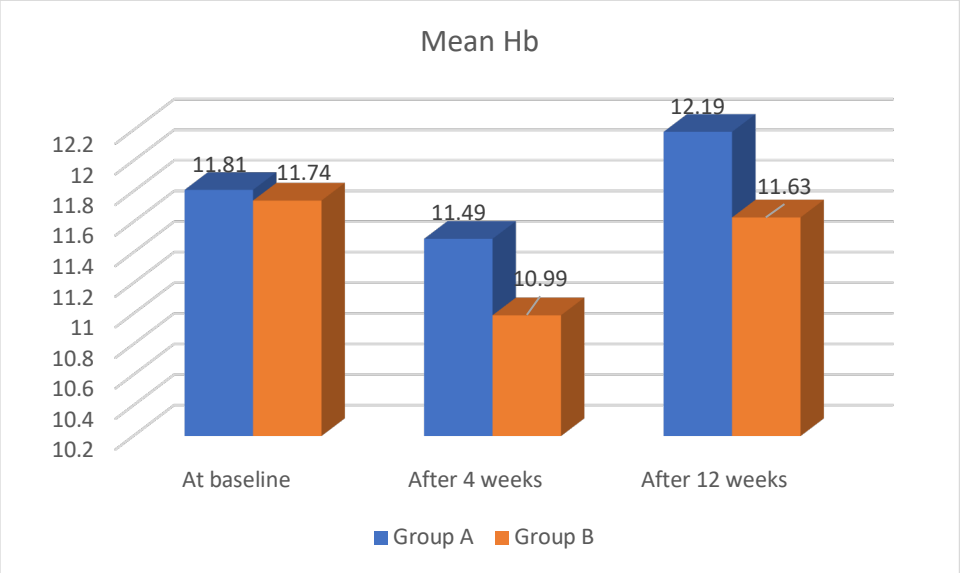


Figure 16: Comparison of mean Hb

At base line, mean Hb in Group A was 11.81± 0.71, and in Group B was 11.74 ± 0.86, after 4 weeks of initiation of treatment, mean Hb in Group A was 11.49 ± 0.84, and in Group B was 10.99 ± 0.83, and after 12 weeks of initiation of treatment, mean Hb in Group A was 12.19 ± 0.68, and in Group B was 11.63 ± 0.74. The mean difference between group A and B was significant after 4 weeks and 12 weeks.

Table 8: Comparison of mean RBC count

Time	**Group A**		**Group B**		**P value**
	Mean RBC	S.D	Mean RBC	S.D	
At baseline	4.12	0.35	4.11	0.41	0.85(non-significant)
After 4 weeks	4.25	0.40	3.87	0.44	0.12(non-significant)
After 12 weeks	5.14	0.84	4.01	0.42	0.19(non-significant)

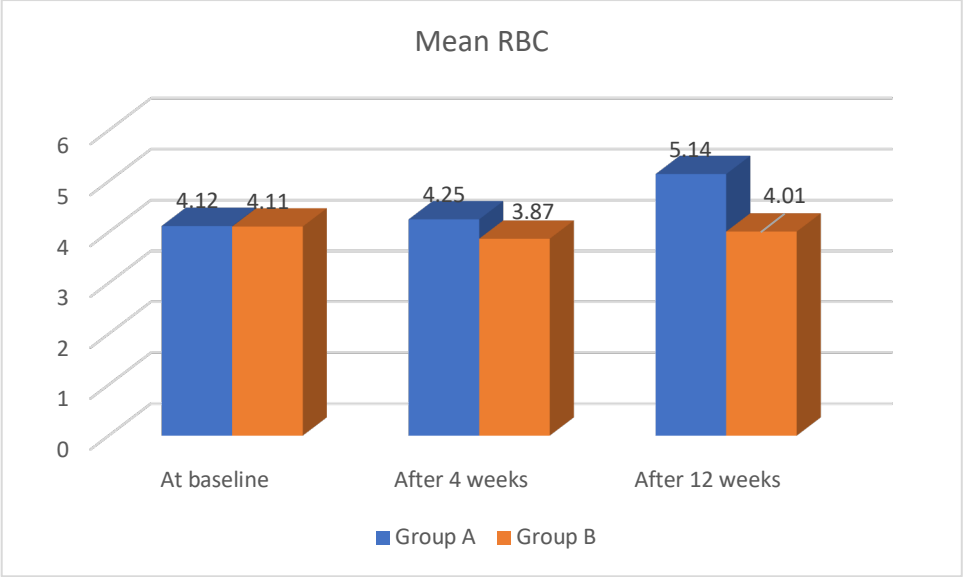


Figure 17: Comparison of mean RBC count

At base line, mean RBC count in Group A was 4.12 ± 0.35, and in Group B was 4.11 ± 0.41, after 4 weeks of initiation of treatment, mean RBC count in Group A was 4.25 ± 0.4, and in Group B was 3.87 ± 0.44, and after 12 weeks of initiation of treatment, mean RBC count in Group A was 5.14 ± 0.84, and in Group B was 4.01 ± 0.42. The mean difference between group A and B was non-significant at all time intervals.

Table 9: Comparison of mean PCV

Time	Group A		Group B		P value
	Mean PCV	**S.D**	**Mean PCV**	**S.D**	
At baseline	34.86	2.59	35.30	2.79	0.42 (non-significant)
After 4 weeks	34.97	2.83	33.39	2.62	**0.04 (significant)**
After 12 weeks	36.12	3.46	33.60	2.51	**0.0001 (significant)**

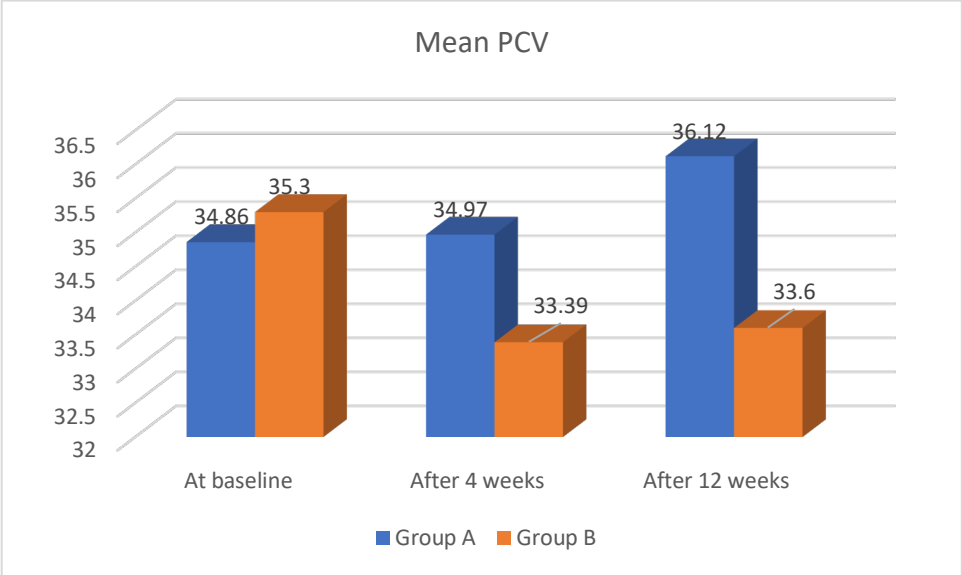


Figure 18: Comparison of mean PCV

At base line, mean PCV in Group A was 34.86 ± 2.59, and in Group B was 35.3 ± 2.79, after 4 weeks of initiation of treatment, mean PCV in Group A was 34.97 ± 2.83, and in Group B was 33.39 ± 2.62, and after 12 weeks of initiation of treatment, mean PCV in Group A was 36.12 ± 3.46, and in Group B was 33.6 ± 2.51. The mean difference between group A and B was significant after 4 weeks and 12 weeks intervals.

Table 10: Comparison of mean MCV

Time	**Group A**		**Group B**		**P value**
	Mean MCV	**S.D**	**Mean MCV**	**S.D**	
At baseline	84.70	6.02	86.35	5.78	0.17(non-significant)
After 4 weeks	85.86	5.94	87.40	7.36	0.28(non-significant)
After 12 weeks	86.80	6.42	84.98	6.99	0.21 (non-significant)

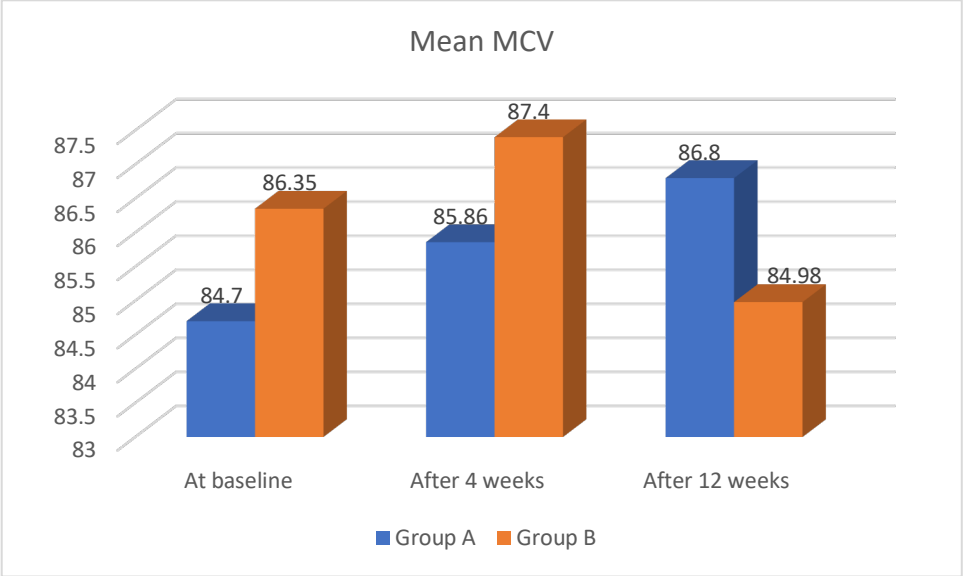


Figure 19: Comparison of mean MCV

At base line, mean MCV in Group A was 84.7 ± 6.02, and in Group B was 86.35 ± 5.78, after 4 weeks of initiation of treatment, mean MCV in Group A was 85.86 ± 5.94, and in Group B was 87.4 ± 7.36, and after 12 weeks of initiation of treatment, mean MCV in Group A was 86.8 ± 6.42, and in Group B was 84.98 ± 6.99. The mean difference between group A and B was non-significant at all time intervals.

Table 11: Comparison of mean MCH

Time	**Group A**		**Group B**		**P value**
	Mean MCH	**S.D**	**Mean MCH**	**S.D**	
At baseline	28.56	2.48	28.55	2.19	0.98(non-significant)
After 4 weeks	28.71	2.28	28.93	3.03	0.71(non-significant)
After 12 weeks	29.26	2.75	27.94	2.73	**0.02(significant)**

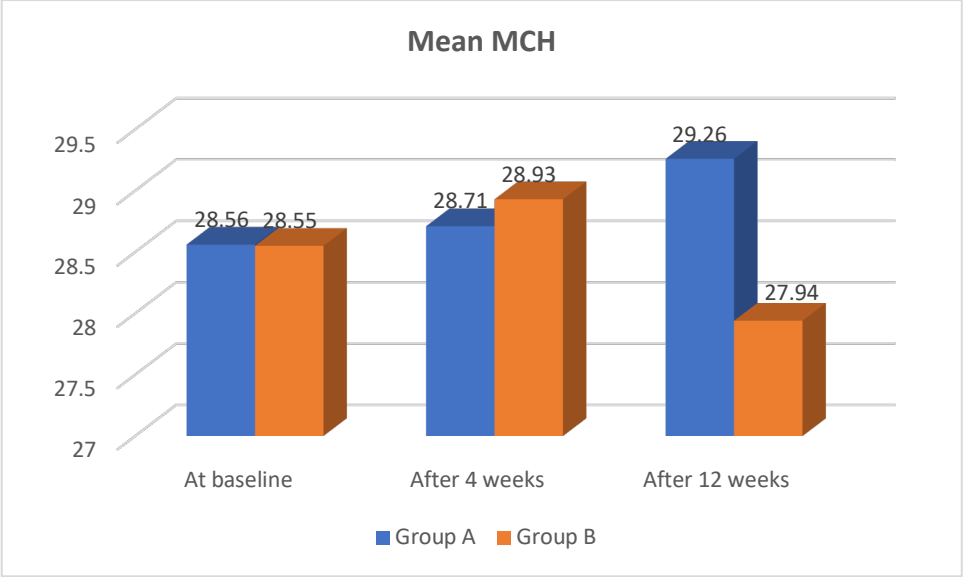


Figure 20: Comparison of mean MCH

At base line, mean MCH in Group A was 28.56 ± 2.48, and in Group B was 28.55 ± 2.19, after 4 weeks of initiation of treatment, mean MCH in Group A was 28.71 ± 2.28, and in Group B was 28.93 ± 3.03, and after 12 weeks of initiation of treatment, mean MCH in Group A was 29.26 ± 2.75, and in Group B was 27.94 ± 2.73. The mean difference between group A and B was significant at 12 weeks interval.

Table 12: Comparison of mean MCHC

Time	Group A		Group B		P value
	Mean MCHC	**S.D**	**Mean MCHC**	**S.D**	
At baseline	33.60	1.64	32.99	1.90	0.09 (non-significant)
After 4 weeks	33.40	1.00	32.93	1.44	0.07(non-significant)
After 12 weeks	33.69	1.95	33.12	1.91	0.16(non-significant)

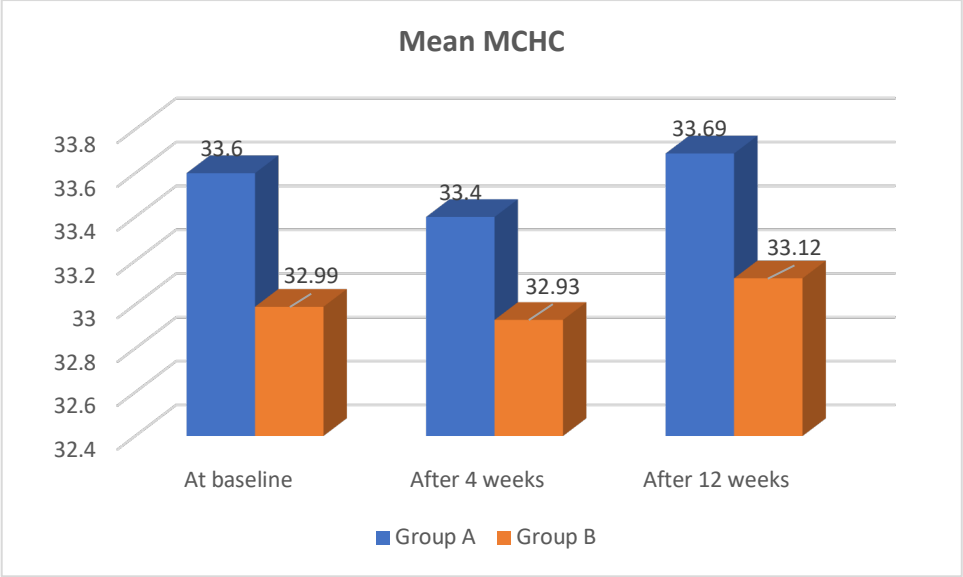


Figure 21: Comparison of mean MCHC

At base line, mean MCHC in Group A was 33.6 ± 1.64, and in Group B was 32.99 ± 1.9, after 4 weeks of initiation of treatment, mean MCHC in Group A was 33.4 ± 1.0, and in Group B was 32.93 ± 1.44, and after 12 weeks of initiation of treatment, mean MCHC in Group A was 33.69 ± 1.95, and in Group B was 33.12 ± 1.91. The mean difference between group A and B was non-significant at all time intervals.

Table 13: Comparison of mean ferritin

Time	Group A		Group B		P value
	Mean ferritin	**S.D**	**Mean ferritin**	**S.D**	
At baseline	51.90	44.92	63.33	33.06	0.35(non-significant)
After 4 weeks	98.36	58.91	70.34	44.75	0.09(non-significant)
After 12 weeks	163.26	76.28	81.28	65.98	**0.0001 (significant)**

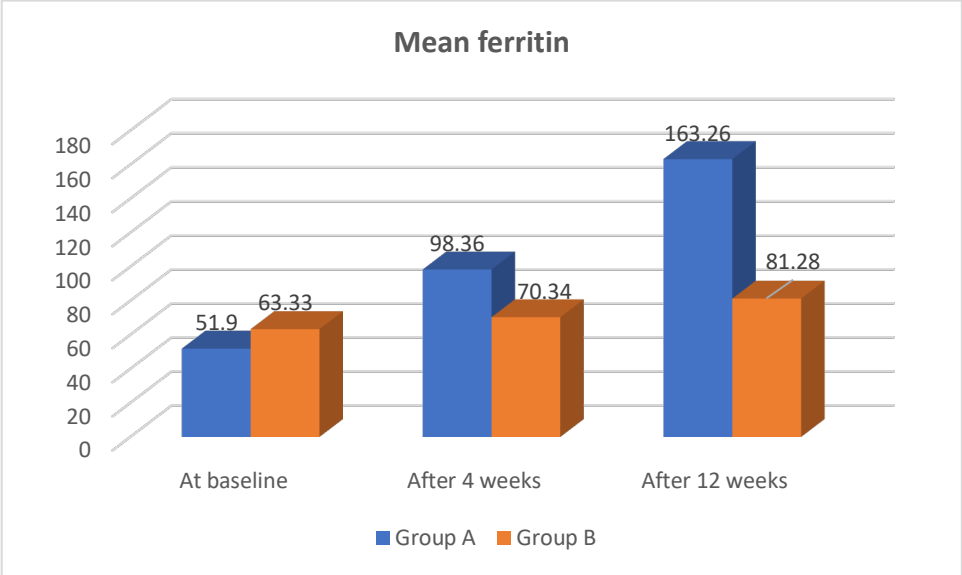


Figure 22: Comparison of mean ferritin

At base line, mean ferritin in Group A was 51.9 ± 44.92, and in Group B was 63.33 ± 33.06, after 4 weeks of initiation of treatment, mean ferritin in Group A was 98.36

± 58.91, and in Group B was 70.34 ± 44.75, and after 12 weeks of initiation of treatment, mean ferritin in Group A was 163.26 ± 76.28, and in Group B was 81.28 ± 65.98. The mean difference between group A and B was significant at 12 weeks-time.

Table 14: Anaemia

Anaemia	Group A	Group B	Total
Present	3 (6.8%)	8 (17%)	11 (12.1%)
Absent	41 (93.2%)	39 (83%)	80 (87.9%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 2.26; P value 0.14(non-significant)

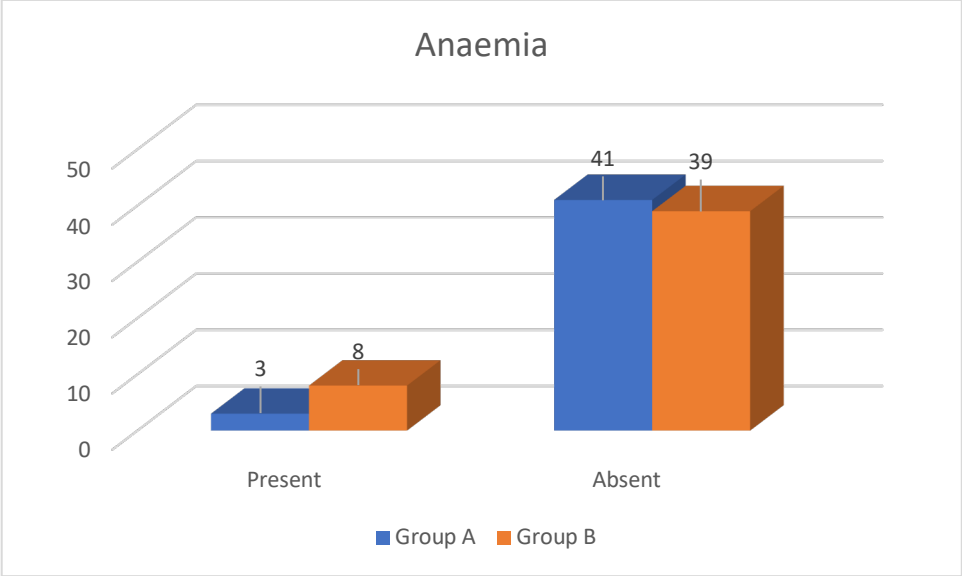


Figure 23: Anaemia

Of the total cases, anaemia was seen in 3 (6.8%) cases of Group A, and 8 (17%) cases of Group B, and this association was non-significant.

Side effects

Table 15: Nausea

Nausea	Group A	Group B	Total
Present	0	8 (17%)	8 (8.8%)
Absent	44 (100%)	39 (83%)	83 (91.2%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 8.54; P **value 0.003 (significant)**

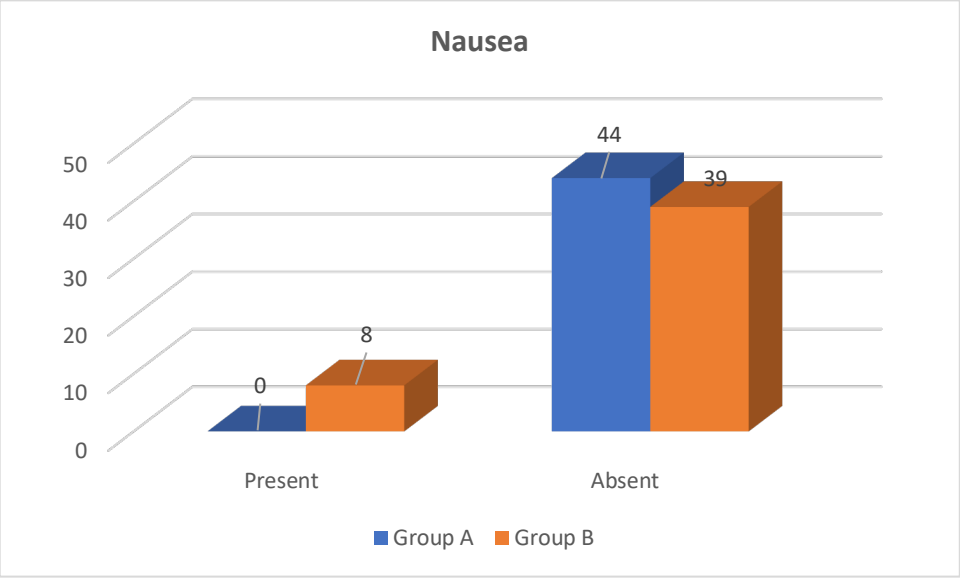


Figure 24: Nausea

In the Group A, none of the cases had nausea and in group B, 8 (17%) cases had nausea. This association was significant.

Table 16: Vomiting

Vomiting	Group A	Group B	Total
Present	0	6 (12.8%)	6 (6.6%)
Absent	44 (100%)	41 (87.2%)	85 (93.4%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 6.01; **P value 0.014 (significant)**

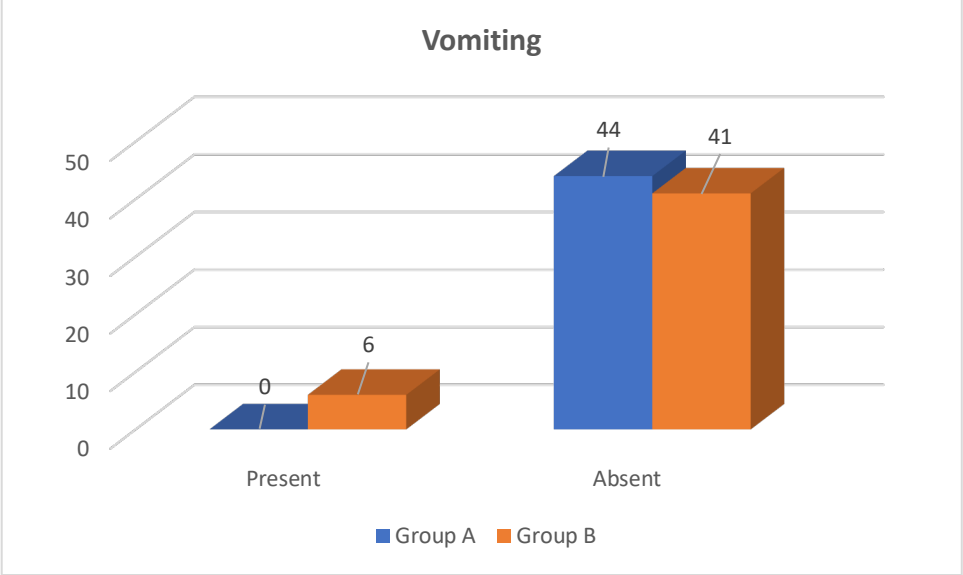


Figure 25: Vomiting

In the Group A, none of the cases had vomiting and in group B, 6 (12.8%) cases had vomiting. This association was significant.

Table 17: Epigastric pain/acidity

Epigastric pain/acidity	Group A	Group B	Total
Present	0	9 (19.1%)	9 (9.9%)
Absent	44 (100%)	38 (80.9%)	82 (90.1%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 9.35; **P value 0.002 (significant)**

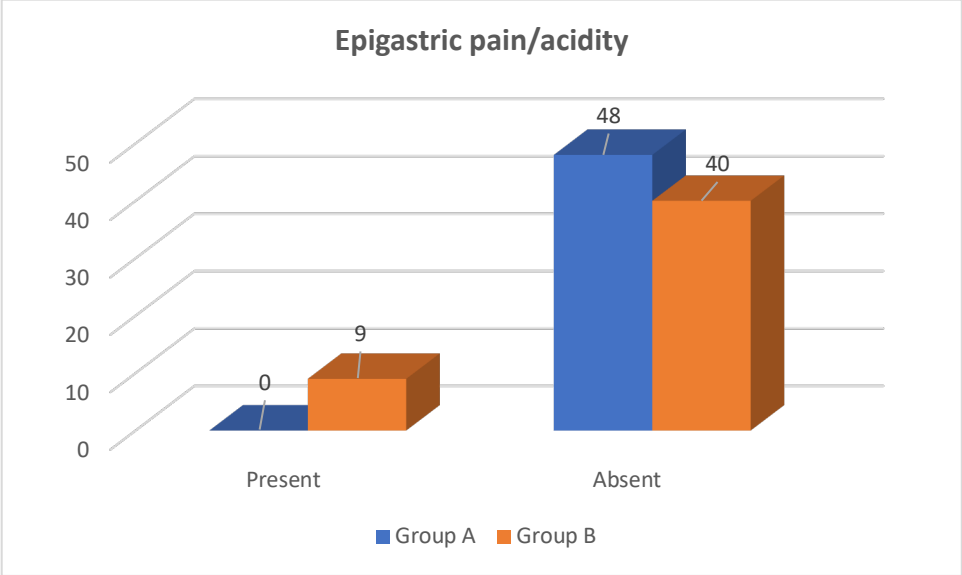


Figure 26: Epigastric pain/acidity

In the Group A, none of the cases had Epigastric pain/acidity and in group B, 9 (19.1%) cases had Epigastric pain/acidity. This association was significant.

Table 18: Pain abdomen

Pain abdomen	Group A	Group B	Total
Present	0	6 (12.8%)	6 (6.6%)
Absent	44 (100%)	41 (87.2%)	85 (93.4%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 6.01; **P value 0.014 (significant)**

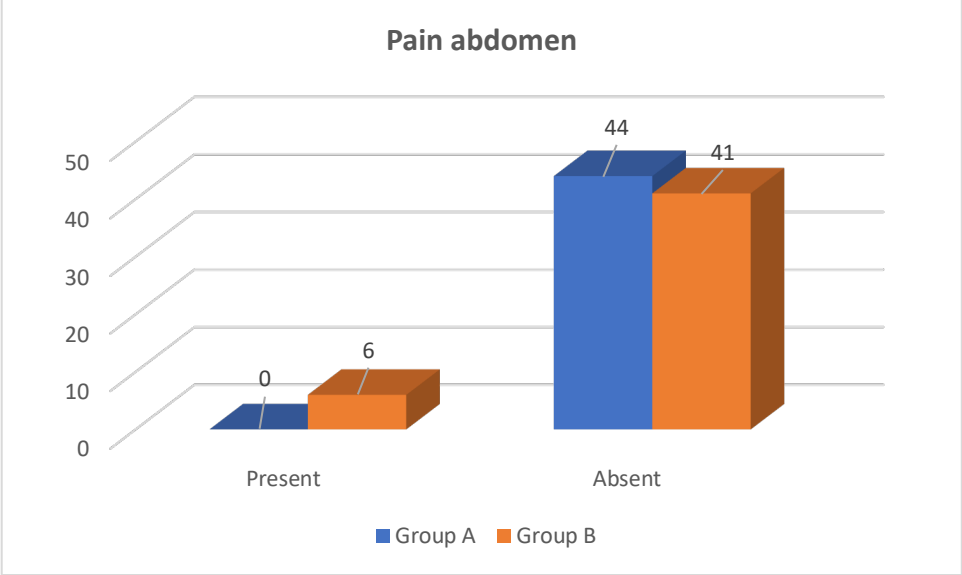


Figure 27: Pain abdomen

In the Group A, none of the cases had Pain abdomen and in group B, 6 (12.8%) cases had Pain abdomen. This association was significant.

Table 19: Constipation

Constipation	Group A	Group B	Total
Present	0	13 (27.7%)	13 (14.3%)
Absent	44 (100%)	34 (72.3%)	78 (85.7%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 14.19; **P value 0.0001 (significant)**

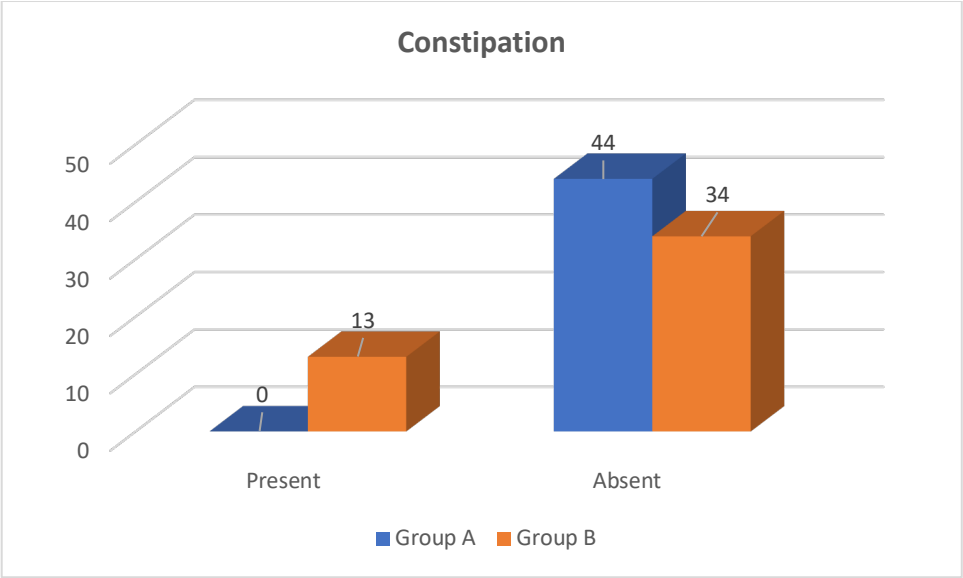


Figure 28: Constipation

In the Group A, none of the cases had Constipation and in group B, 13 (27.7%) cases had Constipation. This association was significant.

Table 20: Diarrhoea

Diarrhoea	Group A	Group B	Total
Present	0	1 (2.1%)	1 (1.1%)
Absent	44 (100%)	46 (97.9%)	90 (98.9%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 0.95; P value 0.33 (non-significant)

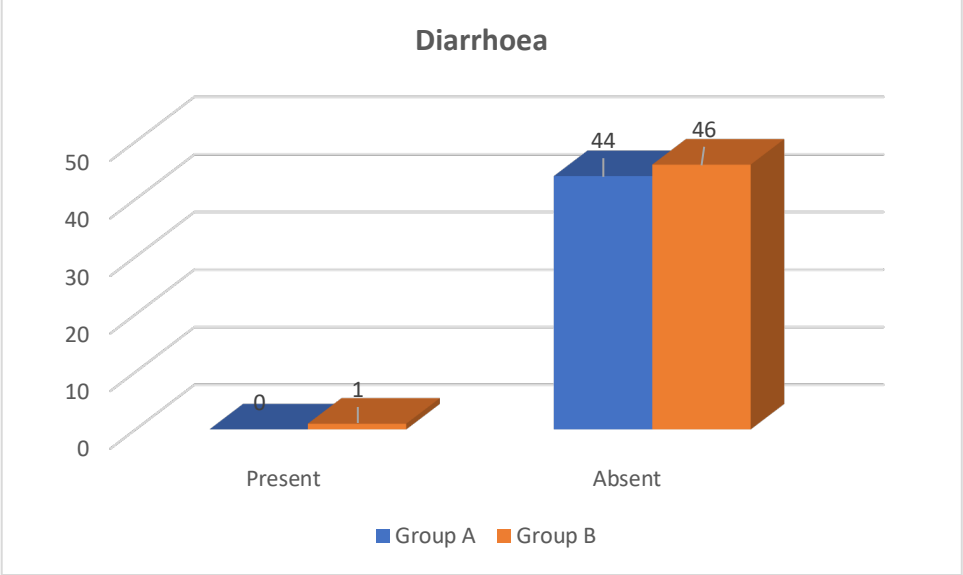


Figure 29: Diarrhoea

In the Group A, none of the cases had Diarrhoea and in group B, 1 (1.1%) case had Diarrhoea. This association was significant.

Table 21: Rashes

Rashes	Group A	Group B	Total
Present	4 (9.1%)	0	4 (4.4%)
Absent	40 (90.9%)	47 (100%)	87 (95.6%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 4.47; **P value 0.035 (significant)**

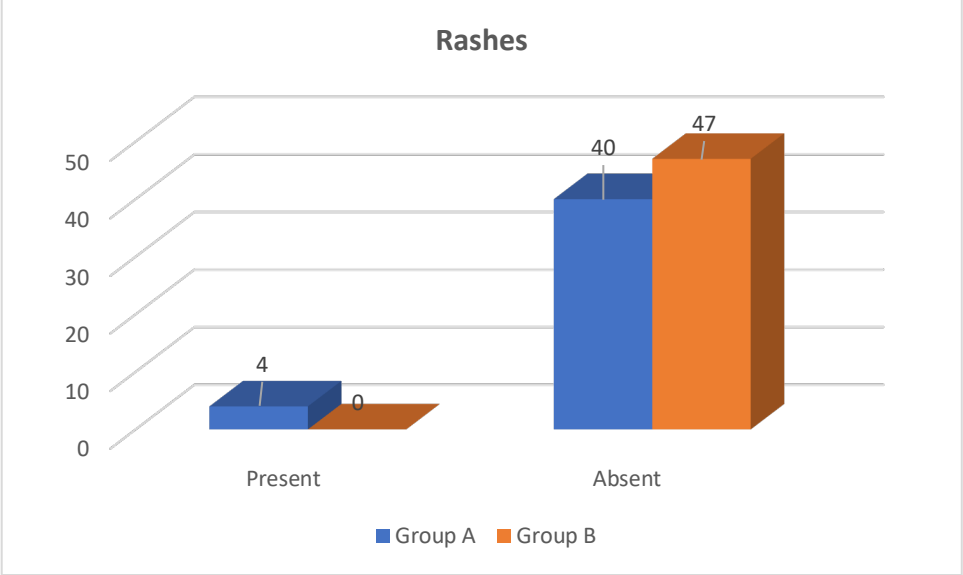


Figure 30: Rashes

In the Group A, 4 (9.1%) cases had rashes and in group B, none of the cases had rashes. This association was significant.

Table 22: Itching

Itching	Group A	Group B	Total
Present	1 (2.3%)	1 (2.1%)	2 (2.2%)
Absent	43 (97.7%)	46 (97.9%)	89 (97.8%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 0.002; P value 0.96 (non-significant)

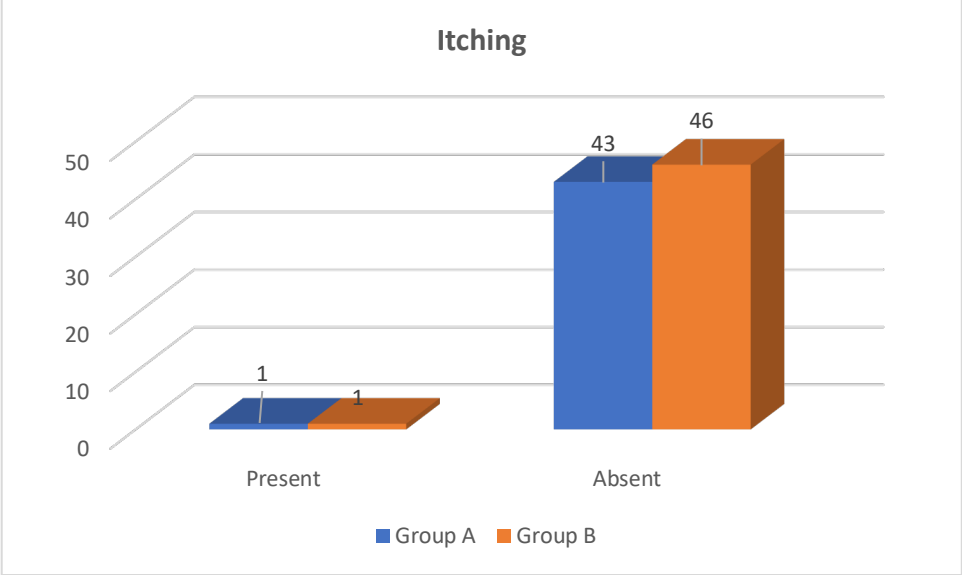


Figure 31: Itching

In the Group A, 1 (2.3%) case had itching and in group B, 1 (2.1%) case had itching.

This association was non-significant.

Table 23: Chills

Chills	Group A	Group B	Total
Present	3 (6.8%)	0	3 (3.3%)
Absent	41 (93.2%)	47 (100%)	88 (96.7%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 3.31; P value 0.067 (non-significant)

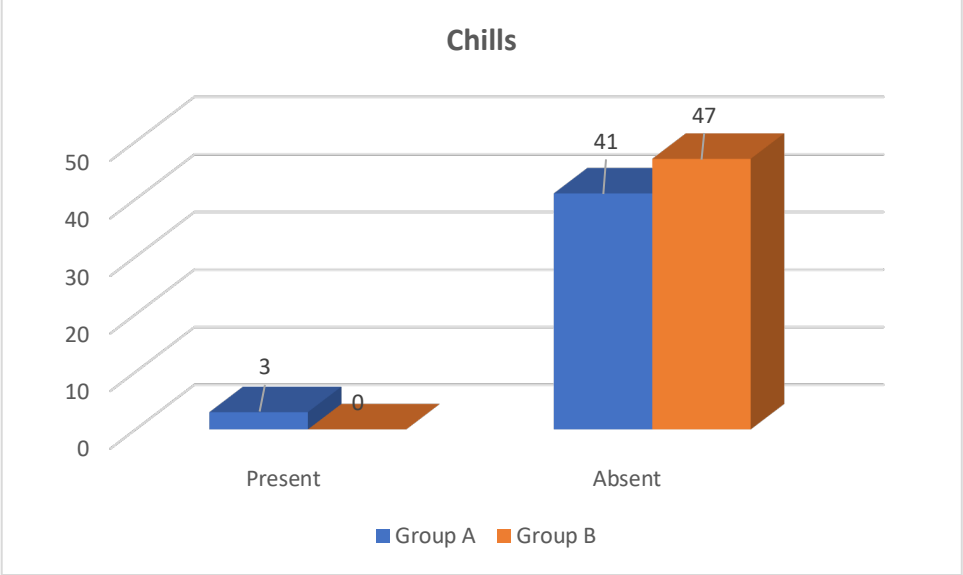


Figure 32: Chills

In the Group A, 3 (6.2%) cases had chills and in group B, none of the cases had chills. This association was non-significant.

Table 24: Headache

Headache	Group A	Group B	Total
Present	2 (4.5%)	0	2 (2.2%)
Absent	42 (95.5%)	47 (100%)	89 (97.8%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 2.18; P value 0.14(non-significant)

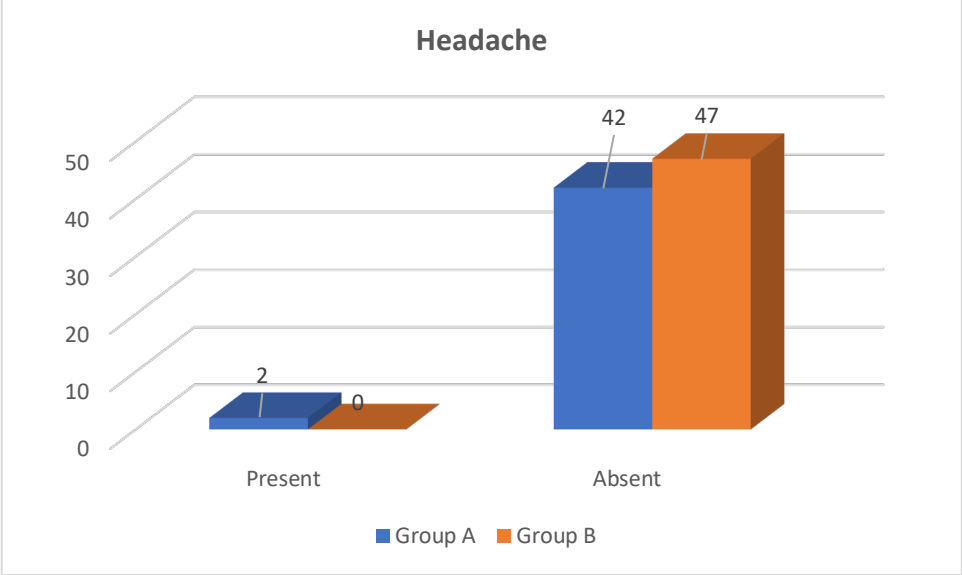


Figure 33: Headache

In the Group A, 2 (4.5%) cases had headache and in group B, none of the cases had headache. This association was non-significant.

Table 25: Local pain

Local pain	Group A	Group B	Total
Present	8 (8.2%)	0	8 (8.8%)
Absent	36 (81.8%)	47 (100%)	83 (91.2%)
Total	44 (48.4%)	47 (51.6%)	91 (100%)

Fishers exact test 9.37; **P value 0.002 (significant)**

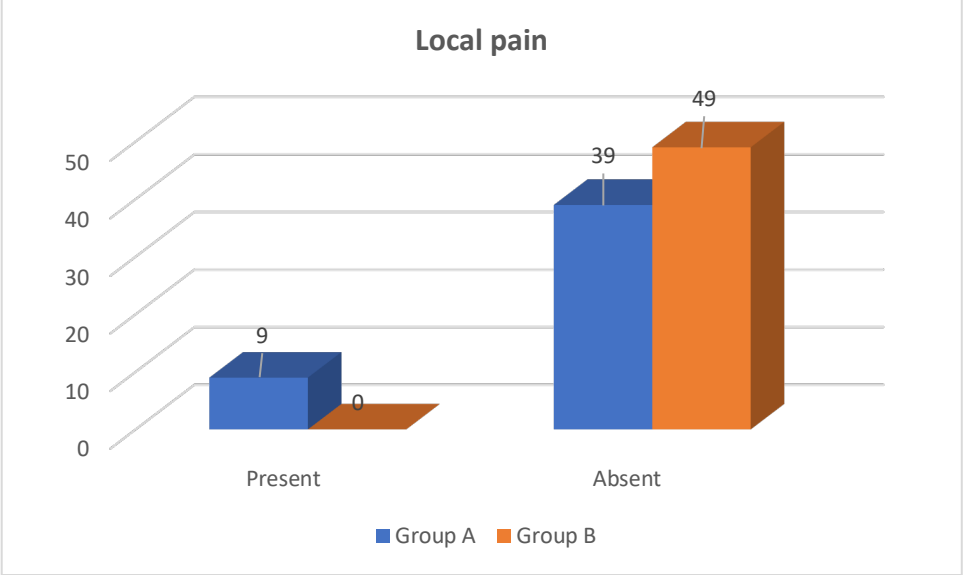


Figure 34: Local pain

In the Group A, 8 (8.2%) cases had local pain and in group B, none of the cases had local pain. This association was significant.

Table 26: Lost to follow up

Lost to follow up	Group A	Group B	Total
Yes	6 (12%)	3 (6%)	9(9%)
No	44 (88%)	47 (94%)	91(91%)
Total	50 (50%)	50 (50%)	100(100%)

Fishers exact test 1.11; P value 0.29 (non-significant)

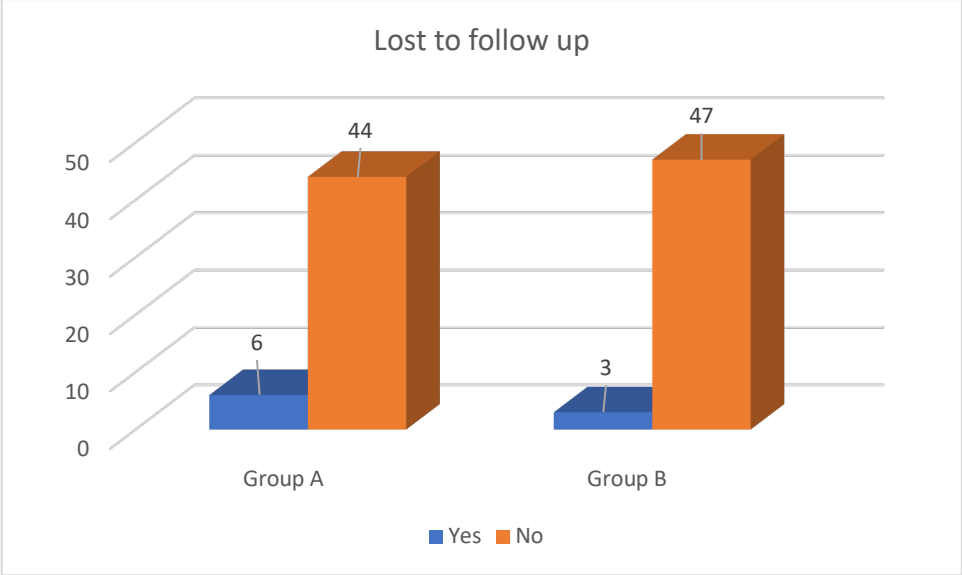


Figure 35: Lost to follow up

In the current research, in IV sucrose group, 12% of cases were lost to follow up, and in the Oral group 6% of cases had lost to follow up.

Table 27: Cost effectiveness

	Mean Cost (in Rupees)		
Group A	2152	35.67	**0.0001(significant)**
Group B	3390	78.5	

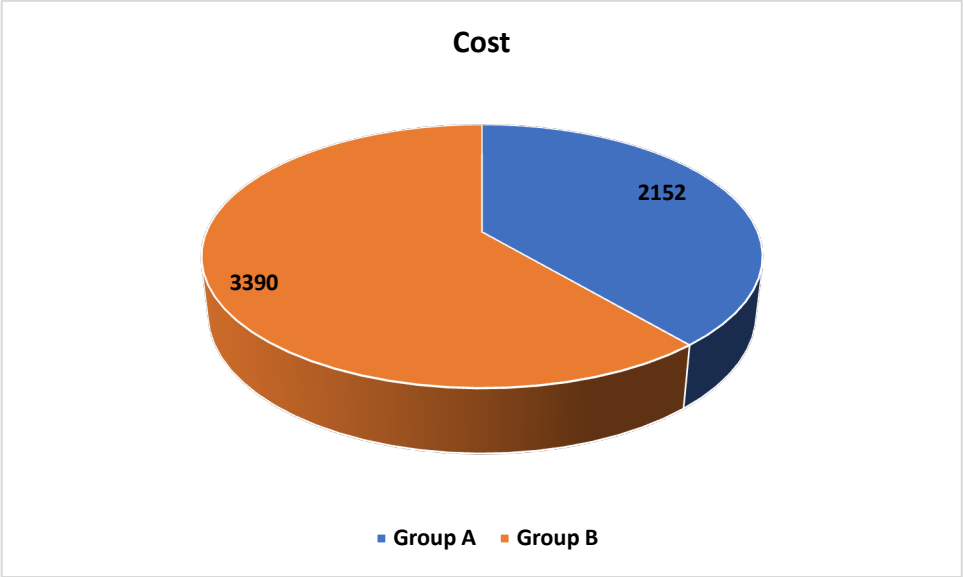


Figure 36: Cost effectiveness

Cost of the Group A, and Group B was 2152 ± 35.67 rupees, 3390 ± 78.5 rupees, respectively.

DISCUSSION

Efficacy

Regarding efficacy of prophylactic anaemia treatment, of the total cases, anaemia was seen in 6.8% of cases of IV sucrose group, and 17% of cases of Oral group, and this association was non-significant. This finding showed that majority of the Antenatal women in IV Sucrose were non-anaemic than O.I Ascorbate group.

Similar finding was mentioned in the study by Gogineni S, and Vemulapalli P, [72] anemia was seen in 6% cases of the IV Sucrose group and 18%of cases of oral group.

Table 28: Efficacy of treatment

Study	Anaemia	
	IV sucrose group	Oral group
Gogineni S, and Vemulapalli P[72]	6%	18%
Current research	6.8%	17%

Compliance

In the current research, in IV sucrose group, 12% of cases were lost to follow up, and in the Oral group 6% of cases had lost to follow up, contrast to the study by Gogineni S, and Vemulapalli P [72] (4% and 40%, respectively).

In the current research, compliance in IV I.S group was 88% and 94% in oral group, contrast to the Dixit R et al. study,[68] i.e., compliance rate was more (93%) in IV sucrose group than O.I group (92%).

Table 29: Lost to follow up

Study	Lost to follow up	
	IV sucrose group	Oral group
Gogineni S, and Vemulapalli P [72]	4%	40%
Dixit R et al. [68]	7%	8%
Current research	12%	6%

Mean period of gestation at enrollment

In the current research, the mean period of gestation of IV group was 21.76 ± 1.53 weeks, and oral group was 20.38 ± 1.47 weeks, that means both groups had more or

less similar period of gestation, which was in accordance with the studies by Rudra S et al. [70] (27.61 ±2.43 and 27.76 ± 2.31, respectively), Deeba S et al. [71] (32 ± 2.46 and 31.93 ± 2.22, respectively), Sudakshina K et al. [73] (24 ± 1.5 and 23.1 ± 1.7, respectively), and Dixit R et al. [68] (20.3 ± 2.1 and 20.2 ± 2.3, respectively).

Table 30: Mean period of gestation at enrollment

Study	**Mean age**	
	IV sucrose group	Oral group
Rudra S et al. [70]	27.61 ±2.43	27.76 ± 2.31
Deeba S et al. [71]	32 ± 2.46	31.93 ± 2.22
Sudakshina K et al. [73]	24 ± 1.5	23.1 ± 1.7
Dixit R et al. [68]	20.3 ± 2.1	20.2 ± 2.3
Current research	21.76 ± 1.53	20.38 ± 1.47

Mean Hb

In the current research, mean Hb in IV sucrose and oral group at baseline was 11.81± 0.71, and 11.74 ± 0.86, respectively, after 4 weeks it was 11.49 ± 0.84, and 10.99 ± 0.83, respectively and after 12 weeks it was 12.19 ± 0.68, and 11.63 ± 0.74, respectively.

Table 31: Mean Hb

Study	IV sucrose		Oral group	
	baseline Hb	At the end of the treatment Hb	baseline Hb	At the end of the treatment Hb
Thobbi VA, and Bijapur ZN [78]	8.6 ± 0.9	10.8 ± 1.3	9.7 ± 1	10.9 ± 1.1
Rudra S et al.[70]	7.81 ± 0.44	10.93 ± 0.60	7.88 ± 0.45	10.30 ± 0.56,
Deeba S et al.[71]	7.9 ± 0.87	10.79 ± 0.84	7.92 ± 0.86	9.91 ± 0.88
Sudakshina K et al. [73]	8.5 ± 0.3	10.9 ± 0.6	10.4 ± 0.5	11.1 ± 0.6
Agalya M et al. [67]	9.64 ± 0.45	11.41 ± 0.51	9.19 ± 0.49	10.76 ± 0.49
Current research	11.81± 0.71	12.19 ± 0.68	11.74 ± 0.86	11.63 ± 0.74.

In the Thobbi VA, and Bijapur ZN study, [78] in IV sucrose group, and Oral Iron Ascorbate group, mean Hb at baseline was 8.6 ± 0.9, and 9.7 ± 1, respectively, after

3weeks, it was 9.8 ± 1, and 10.3 ± 1, respectively and after 6 weeks, it was 10.8 ± 1.3, and 10.9 ± 1.1, respectively.

In the study by Rudra S et al., [70] mean Hb at baseline in IV group and Oral group was 7.81 ± 0.44, and 7.88 ± 0.45, respectively; at 4 weeks, it was 9.80 ± 0.36, and 9.17 ± 0.47, respectively; and at 12 weeks it was 10.93 ± 0.60, and 10.30 ± 0.56, respectively.

In the Deeba S et al. [71] study, mean Hb in IV group and Oral group at baseline was 7.9± 0.87 and 7.92 ± 0.86, respectively; at 4 weeks it was 10.09 ± 0.81 and 9.32 ± 0.87, respectively, and at 6 weeks it was 10.79 ± 0.84 and 9.91 ± 0.88, respectively. The mean difference was significant at 4 weeks and 6 weeks.

In the Sudakshina K et al. study, [73] in the IV sucrose group, the mean Hb before treatment was 8.5 ± 0.3 and after treatment it was 10.9 ± 0.6, respectively, and in the oral group, it was 10.4 ± 0.5, and 11.1 ± 0.6, respectively. This mean Hb difference was significant before and after treatment in both the IV group and Oral groups. The mean Hb increase was more in IV sucrose group than oral group, similar to the current research.

Dixit R et al. study, [68] baseline mean Hb in the IV sucrose group was 9.8 ± 0.9 and in the Oral group was 9.7 ± 0.8, respectively. The change in the mean Hb was more in the IV sucrose group than O.I group, similar to the current research.

In the Agalya M et al. study, [67] the mean Hb 14 days after treatment in the IV sucrose group was 9.64 ± 0.45 and in the oral group was 9.19 ± 0.49, and this mean difference was statistically significant, and after 28 days it was 11.41 ± 0.51, and 10.76 ± 0.49, in both the groups respectively. The mean increase in Hb was more in IV sucrose group than Oral group.

In the Savitha A et al. study,[82] in the IV sucrose group mean Hb increased from 8.26 ± 0.34 to 10.1 ± 0.51 and in the Oral group it increased from 8.35 ± 0.36 to 9.28 ± 0.52. Post treatment mean difference was significant.

In the current research, the mean Hb was increased from baseline to 12 weeks after treatment in IV sucrose group, but it decreased in oral group, contrast to the studies by Rudra S et al., [70] Deeba S et al., [71] Sudakshina K et al., [73] Agalya M et al., [67] and Savitha A et al. [82] This was due to the variation in the duration of treatment in the current research and the above studies.

Mean RBC Count

At base line, mean RBC count in IV group was 4.12 ± 0.35, and in Oral group was 4.11 ± 0.41, after 4 weeks of initiation of treatment, mean RBC count in IV group was 4.25 ± 0.4, and in Oral group was 3.87 ± 0.44, and after 12 weeks of initiation of treatment, mean RBC count in IV group was 5.14 ± 0.84, and in Oral group was 4.01 ± 0.42. The mean difference between IV group and Oral group was non-significant at all time intervals.

Table 32: Mean RBC Count

Study	IV sucrose		Oral group	
	baseline RBC count	At the end of the treatment RBC count	baseline RBC count	At the end of the treatment RBC count
Sudakshina K et al. [73]	3.2 ± 0.1	3.8 ± 0.2	3.7 ± 0.3	4.0 ± 0.2
Current research	4.12 ± 0.35	5.14 ± 0.84	4.11 ± 0.41	4.01 ± 0.42

In the Sudakshina K et al. study, [73] in the IV sucrose group, the mean RBC count before treatment was 3.2 ± 0.1 and after treatment it was 3.8 ± 0.2, respectively, and in the oral group, it was 3.7 ± 0.3, and 4.0 ± 0.2, respectively. This mean RBC count was significant before and after treatment in both the IV group and Oral group. The mean RBC count increase was more in IV sucrose group than oral group, similar to the current research.

In the study by Rudra S et al., [70] mean RBC count at baseline in IV group and Oral group was 0.77 ± 0.28, and 0.76 ± 0.25, respectively; The mean difference between the IV group and oral group was significant at 1 week, but not yet at baseline.

In the current research, the mean RBC count had increased from baseline to 12 weeks after treatment in IV sucrose group, but it decreased in oral group, contrast to the studies by Rudra S et al., [70] and Sudakshina K et al. [73] This was due to the variation in the duration of treatment in the current research and the above studies.

Mean PCV

At base line, mean PCV in IV group was 34.86 ± 2.59, and in Oral group was 35.3 ± 2.79, after 4 weeks of initiation of treatment, mean PCV in IV group was 34.97 ± 2.83, and in Oral group was 33.39 ± 2.62, and after 12 weeks of initiation of treatment, mean PCV in IV group was 36.12 ± 3.46, and in Oral group was 33.6 ±

2.51. The mean difference between IV group and Oral group was significant after 4 weeks and 12 weeks intervals.

Table 33: Mean PCV

Study	IV sucrose		Oral group	
	baseline	At the end	baseline PCV	At the end of the treatment PCV
Rudra S et al. [70]	24.59 ± 0.76	29.91 ± 1.03	24.80 ± 0.90	28.66 ± 0.96
Savitha A et al. [82]	27.21 ± 1.91	33.87 ± 0.96	28.18 ± 1.93	32.07 ± 0.99
Current research	34.86 ± 2.59	36.12 ± 3.46	35.3 ± 2.79	33.6 ± 2.51

In the study by Rudra S et al., [70] mean PCV at baseline in IV group and Oral group was 24.59 ± 0.76, and 24.80 ± 0.90, respectively; and at 4 weeks, it was 29.91 ± 1.03, and 28.66 ± 0.96, respectively.

In the Savitha A et al. study, [82] in the IV sucrose group mean PCV increased from 27.21 ± 1.91 to 33.87 ± 0.96 and in the Oral group it increased from 28.18 ± 1.93 to 32.07 ± 0.99. Post treatment mean difference was significant.

In the current research, the mean PCV difference between IV group and Oral group was significant after 4 weeks and 12 weeks intervals, while significant difference was noticed only at 4 weeks in the study by Rudra S et al. [70]

In the current research, the mean PCV had increased from baseline to 12 weeks after treatment in IV sucrose group, but it decreased in oral group, contrast to the studies by Rudra S et al., [70] and Savitha A et al. [82] This was due to the variation in the duration of treatment in the current research and the above studies.

Mean MCV

In this study, at base line, mean MCV in IV group was 84.7 ± 6.02, and in Oral group was 86.35 ± 5.78, after 4 weeks of initiation of treatment, mean MCV in IV group was 85.86 ± 5.94, and in Oral group was 87.4 ± 7.36, and after 12 weeks of initiation of treatment, mean MCV in IV group was 86.8 ± 6.42, and in Oral group was 84.98 ± 6.99. The mean difference between IV group and Oral group was non-significant at all time intervals.

Table 34: Mean PCV

Study	IV sucrose		Oral group	
	baseline PCV	At the end of the treatment PCV	baseline PCV	At the end of the treatment PCV
Rudra S et al.[70]	71.88 ± 1.12	81.67 ± 2.74	72.19 ± 1.18	77.72 ± 1.97
Sudakshina K et al. [73]	63 ± 3	78 ± 4	73 ± 4	84 ± 5
Savitha A et al.[82]	79.52 ± 1.58	82.08 ± 1.9	80.26 ± 1.46	80.98 ± 1.98
Current research	84.7 ± 6.02	86.8 ± 6.42	86.35 ± 5.78	84.98 ± 6.99

In the study by Rudra S et al., [70] mean MCV at baseline in IV group and Oral group was 71.88 ± 1.12, and 72.19 ± 1.18, respectively; and at 4 weeks, it was 81.67 ± 2.74 and 77.72 ± 1.97, respectively.

In the Sudakshina K et al. study, [73] in the IV sucrose group, the mean MCV before treatment was 63 ± 3 and after treatment it was 78 ± 4, respectively, and in the oral group, it was 73 ± 4, and 84 ± 5, respectively. This mean MCV was significant before

and after treatment in both the IV group and Oral groups. The mean MCV increase was more in IV sucrose group than oral group, similar to the current research.

In the Savitha A et al. study, [82] in the IV sucrose group mean MCV increased from 79.52 ± 1.58 to 82.08 ± 1.9 and in the Oral group it increased from 80.26 ± 1.46 to 80.98 ± 1.98. Post treatment mean difference was significant.

The mean MCV difference between IV group and Oral group was non-significant at all time intervals, while at baseline it was non-significant, but at 4 weeks it was significant in the studies by Rudra S et al. [70]

In the current research, the mean MCV had increased from baseline to 12 weeks after treatment in IV sucrose group, but it decreased in oral group, contrast to the studies by Rudra S et al., [70] Sudakshina K et al. [73] and Savitha A et al. [82] This was due to the variation in the duration of treatment in the current research and the above studies.

Mean MCH

At base line, mean MCH in IV group was 28.56 ± 2.48, and in Oral group was 28.55 ± 2.19, after 4 weeks of initiation of treatment, mean MCH in IV group was 28.71 ±

2.28, and in Oral group was 28.93 ± 3.03, and after 12 weeks of initiation of treatment, mean MCH in IV group was 29.26 ± 2.75, and in Oral group was 27.94 ± 2.73. The mean difference between IV group and Oral group was significant at 12 weeks interval.

Table 35: Mean MCH

Study	IV sucrose		Oral group	
	baseline MCH	At the end of the treatment MCH	baseline MCH	At the end of the treatment MCH
Rudra S et al.[70]	23.71 ± 0.41	29.47 ± 1.25	23.78 ± 0.44	28.27 ± 1.82
Sudakshina K et al. [73]	27 ± 1	30 ± 2	29 ± 1	32 ± 1
Savitha A et al.[82]	24.79 ± 0.87	29.76 ± 0.68	25.21 ± 0.97	27.9 ± 0.57
Current research	28.56 ± 2.48	29.26 ± 2.75	28.55 ± 2.19	27.94 ± 2.73

In the study by Rudra S et al., [70] mean MCH at baseline in IV group and Oral group was 23.71 ± 0.41, and 23.78 ± 0.44, respectively; and at 4 weeks, it was 29.47 ± 1.25 and 28.27 ± 1.82, respectively.

In the Sudakshina K et al. study, [73] in the IV sucrose group, the mean MCH before treatment was 27 ± 1 and after treatment it was 30 ± 2, respectively, and in the oral group, it was 29 ± 1, and 32 ± 1, respectively. This mean MCH was significant before and after treatment in both the IV group and Oral groups. The mean MCH increase was more in IV sucrose group than oral group, similar to the current research.

In the Savitha A et al. study, [82] in the IV sucrose group mean MCH increased from 24.79 ± 0.87 to 29.76 ± 0.68 and in the Oral group it increased from 25.21 ± 0.97 to 27.9 ± 0.57. Post treatment mean difference was significant.

The mean MCH difference between IV group and Oral group was significant at 12 weeks, but it was significant at 4 weeks in the studies by Rudra S et al. [70]

In the current research, the mean MCH had increased from baseline to 12 weeks after treatment in IV sucrose group, but it decreased in oral group, contrast to the studies by Rudra S et al., [70] Sudakshina K et al. [73] and Savitha A et al. [82] This was due to the variation in the duration of treatment in the current research and the above studies.

Mean MCHC

At base line, mean MCHC in IV group was 33.6 ± 1.64, and in Oral group was 32.99 ± 1.9, after 4 weeks of initiation of treatment, mean MCHC in IV group was 33.4 ± 1.0, and in Oral group was 32.93 ± 1.44, and after 12 weeks of initiation of treatment, mean MCHC in IV group was 33.69 ± 1.95, and in Oral group was 33.12 ± 1.91. The mean difference between IV group and Oral group was non-significant at all time intervals.

Table 36: Mean MCHC

Study	IV sucrose		Oral group	
	baseline MCHC	At the end of the treatment MCHC	baseline MCHC	At the end of the treatment MCHC
Rudra S et al.[70]	29.72 ± 0.41	32.33 ± 0.40	29.81 ± 0.44	31.33 ± 0.51
Sudakshina K et al. [73]	31 ± 2	33 ± 2	31 ± 1	33 ± 2
Savitha A et al.[82]	30.40 ± 1.35	33.36 ± 1.05	31.0 ± 1.23	33.41 ± 1.22
Current research	33.6 ± 1.64	33.69 ± 1.95	32.99 ± 1.9	33.12 ± 1.91

In the study by Rudra S et al., [70] mean MCHC at baseline in IV group and Oral group was 29.72 ± 0.41, and 29.81 ± 0.44, respectively; and at 4 weeks, it was 32.33 ± 0.40 and 31.33 ± 0.51, respectively.

In the Sudakshina K et al. study, [73] in the IV sucrose group, the mean MCHC before treatment was 31 ± 2 and after treatment it was 33 ± 2, respectively, and in the oral group, it was 31 ± 1, and 33 ± 2, respectively. This mean MCHC was significant before and after treatment in both the IV group and Oral groups. The mean MCHC increase was more in IV sucrose group than oral group, similar to the current research.

In the Savitha A et al. study, [82] in the IV sucrose group mean MCHC increased from 30.40 ± 1.35 to 33.36 ± 1.05 and in the Oral group it increased from 31.0 ± 1.23 to 33.41 ± 1.22.

The mean MCHC difference between IV group and Oral group was non-significant at all time intervals., but it was significant at 4 weeks in the studies by Rudra S et al.[70]

In the current research, the mean MCHC difference between IV sucrose and oral group was significantly increased from baseline to at the end of the treatment, which

was in concordance with the studies by Rudra S et al., [70] Sudakshina K et al. [73] and Savitha A et al. [82]

Mean ferritin

In the current research, mean serum ferritin in IV sucrose and oral group at baseline was 51.9 ± 44.92, and 63.33 ± 33.06, respectively, after 4 weeks it was 98.36 ± 58.91, and 70.34 ± 44.75, respectively and after 12 weeks it was 163.26 ± 76.28, and 81.28 ± 65.98, respectively. The mean difference between IV sucrose and oral group was significant at 12 weeks.

Table 37: Mean ferritin

Study	IV sucrose		Oral group	
	baseline ferritin	At the end of the treatment ferritin	baseline ferritin	At the end of the treatment ferritin
Thobbi VA, and Bijapur ZN [78]	15.1 ± 22.2	108.2 ± 74.1	9.9 ± 5.9	43.7 ± 25.5
Rudra S et al.[70]	10.48 ± 1.46	58.26 ± 11.16	10.43 ± 1.86	42.10 ± 8.55

Deeba S et al.[71]	8.44 ±1.35	86.98 ±19.94	8.13 ±1.45	34.78 ± 8.79
Current research	51.9 ± 44.92	163.26 ± 76.28	63.33 ± 33.06	81.28 ± 65.98

In the Thobbi VA, and Bijapur ZN study, [78] in IV sucrose group, and Oral group, mean serum ferritin at baseline was 15.1 ± 22.2, 9.9 ± 5.9, respectively, after 3weeks, it was 65 ± 41.3, 27.5 ± 17.9, respectively and after 6 weeks, it was 108.2 ± 74.1, and 43.7 ± 25.5, respectively.

In the study by Rudra S et al., [70] mean ferritin at baseline in IV group and Oral group was 10.48 ± 1.46, and 10.43 ± 1.86, respectively; at 4 weeks, it was 35.47 ± 4.37 and 14.04 ± 1.86, respectively; and at 12 weeks, it was 58.26 ± 11.16 and 42.10 ± 8.55, respectively.

In the Deeba S et al. study, [71] mean ferritin in IV group and Oral group at baseline was 8.44 ±1.35 and 8.13 ±1.45, respectively; at 4 weeks it was 61.1 ± 19.66 and 23.36 ± 8.57, respectively, and at 6 weeks it was 86.98 ±19.94 and 34.78 ± 8.79, respectively. The mean difference was significant at 4 weeks and 6 weeks.

In the Agalya M et al. study, [67] after 28 days the mean ferritin was 60.92 ± 6.9, and 50.68 ± 2.64, in both the groups respectively. The mean increase in ferritin was more in IV sucrose group than Oral group.

The mean ferritin difference between IV group and Oral group was significant at 12 weeks, but it was significant at 4 weeks and 12 weeks in the studies by Rudra S et al., [70] significant at 4 weeks and 6 weeks in Deeba S et al. [71]

In this study, the mean ferritin increase was more in IV group than the oral group, which was in accordance with the studies by Thobbi VA, and Bijapur ZN, [78] Deeba S et al., [71] Agalya M et al. [67] and Rudra S et al. [70]

Side effects

Nausea

In the current research, none of the cases in IV sucrose group had nausea and 17% of cases of the oral group had nausea, similar higher incidence was reported in oral group by the studies by Thobbi VA, and Bijapur ZN [78] (0% and 6%, respectively), Savitha A et al. [82] (0% and 26.7%, respectively), in contrast to the study by Agalya M et al. [67] (8% and 4%, respectively).

Table 38: Nausea

Study	**Nausea**	
	IV sucrose group	Oral group
Thobbi VA, and Bijapur ZN [78]	0%	6%
Savitha A et al. [82]	0%	26.7%
Agalya M et al. [67]	8%	4%

Gogineni S, and Vemulapalli P [72]	0%	30%
Current research	0%	17%

In the study by Gogineni S, and Vemulapalli P, [72] in the IV Sucrose group none of the cases had nausea and vomiting, and 30% of the cases had nausea and vomiting in oral group.

This association regarding nausea was significant in the current research, similar to the studies by Thobbi VA, and Bijapur ZN. [78]

Vomiting

In the current research, in IV sucrose group, none of the cases had vomiting and in the oral group, 12.8% of cases had vomiting, similar higher incidence was reported in oral group by the studies by Thobbi VA, and Bijapur ZN [78] (0% and 3%, respectively), and Agalya M et al. [67] (0% and 4%, respectively).

This association regarding vomiting was significant in the current research, similar to the studies by Thobbi VA, and Bijapur ZN. [78]

Table 39: Vomiting

Study	**Vomiting**	
	IV sucrose group	Oral group
Thobbi VA, and Bijapur ZN [78]	0%	3%
Agalya M et al. [67]	0%	4%
Current research	0%	12.8%

Epigastric pain/acidity

In the current research, in IV sucrose group, none of the cases had epigastric pain/acidity and in the oral group, 19.1% of cases had it, similar higher incidence was reported in oral group by the studies by Thobbi VA, and Bijapur ZN [78] (0% and 2%, respectively), Agalya M et al. [67] (4% and 8%, respectively), Gogineni S, and Vemulapalli P [72] (0% and 24%, respectively), Sudakshina K et al. study [73] (66.7% and 71.4%, respectively).

Table 40: Epigastric pain/acidity

Study	**Epigastric pain/acidity**	
	IV sucrose group	Oral group
Thobbi VA, and Bijapur ZN [78]	0%	2%

Agalya M et al. [67]	4%	8%
Gogineni S, and Vemulapalli P [72]	0%	24%
Sudakshina K et al. [73]	66.7%	71.4%
Current research	0%	19.1%

This association regarding epigastric pain/acidity was significant in the current research, similar to the studies by Thobbi VA, and Bijapur ZN. [78]

Pain abdomen

In the IV Sucrose group, none of the cases had Pain abdomen and in oral group, 12.8% of cases had Pain abdomen. This association was significant.

Constipation

In in the IV Sucrose group, none of the cases had Constipation and in the oral group, 27.7% of cases had Constipation, which was in accordance with the studies by Gogineni S, and Vemulapalli P[72] (0% and 12%), and Agalya M et al. [67] (0% and 8%, respectively), and Savitha A et al. [82] (0% and 20%, respectively).

Table 41: Constipation

Study	**Constipation**	
	IV sucrose group	Oral group
Agalya M et al. [67]	0%	8%
Gogineni S, and Vemulapalli P[72]	0%	12%
Savitha A et al. [82]	0%	20%
Current research	0%	27.7%

Diarrhoea

In the current research, in IV sucrose group, none of the cases had diarrhoea and in the oral group, 1.1% of cases had it, similar higher incidence was reported in oral group by the studies by Thobbi VA, and Bijapur ZN [78] (0% and 2%, respectively).

Table 42: Diarrhoea

Study	**Diarrhoea**	
	IV sucrose group	Oral group
Thobbi VA, and Bijapur ZN [78]	0%	2%
Current research	0%	1.1%

This association regarding diarrhoea was significant in the current research, similar to the studies by Thobbi VA, and Bijapur ZN. [78]

Rashes

In the IV I.S group, 9.1% of cases had rashes and in oral group, none of the cases had rashes. This association was significant.

In the study by Gogineni S, and Vemulapalli P, [72] in the IV Sucrose group 2% of had rashes, and none of the cases had rashes in oral group.

Table 43: Rashes

Study	**Rashes**	
	IV sucrose group	Oral group
Gogineni S, and Vemulapalli P[72]	2%	0%
Current research	9.1%	0%

Itching

In the IV I.S group, 2.3% of cases had itching and in oral group, 2.1% of case had itching. This association was non-significant.

Chills

In the IV I.S group, 6.2% of cases had chills and in oral group, none of the cases had chills. This association was significant.

Headache

In the IV I.S group, 4.5% of cases had headache and in oral group, none of the cases had headache. This association was non-significant.

Local pain

In the current research, in IV sucrose group, 8.2% of the cases had local pain and in the oral group, none of the cases had it, similar higher incidence was reported in IV group by the studies by Thobbi VA, and Bijapur ZN[78] (3% and 0%, respectively), Gogineni S, and Vemulapalli P [72] (6% and 0%, respectively).

Table 44: Local pain

Study	**Local pain**	
	IV sucrose group	Oral group
Thobbi VA, and Bijapur ZN [78]	3%	0%
Gogineni S, and Vemulapalli P[72]	6%	0%
Current research	18.8%	0%

This association regarding local pain was significant in the current research, similar to the studies by Thobbi VA, and Bijapur ZN. [78]

Cost effectiveness

Cost of the treatment in Group A, and Group B was 2152 ± 35.67, and 3390 ± 78.5, respectively.

While, Gogineni S, and Vemulapalli P study[72] had IV group had 1600/- while O.I group had cost of 1500/-

Limitations of the study:

- Small sample size was one of the limitations of the study, with increased sample size, varied results can be found.
- Participant compliance and adherence to the assigned treatments influence the results. With increased compliance better results can be obtained.
- Paucity of the studies is major limitation of the study. In near future, further studies should be conducted to know the better comparison between the study drugs.

Strengths of the study:

- Most of the available studies had focused on comparison of IV Sucrose with Ferrous Fumarate, Ferrous Sulphate, or other available conventional oral formulations, but the current research was done with Ferrous Ascorbate. This is one of the strengths of the study.
- All the blood indices such as Mean Hb, RBC, MCV, MCH, MCHC, and Serum Ferritin was measured, which was one of the strengths of the study.
- Not many studies have been done on IV I.S for prophylaxis of anemia, most of them have been done for treating anemia.

Recommendations

Further studies are needed to conduct with a large sample size in comparison of IV I.S with Oral ferrous Ascorbate for better understanding of the drugs in prevention of anaemia among pregnant mothers.

Conclusion

The current research was done to observe the effect of IV I.S and O.I for the prophylaxis of anaemia during pregnancy. The findings showed significant improvements in Hb levels in the IV I.S than Oral ferrous ascorbate groups. Side effects were also minimal in IV I.S group than O.I group. Hence it can be concluded that IV I.S is a better alternative than O.I group as a prophylactic iron therapy among pregnant mothers with less side effects and better compliance in number of patients who developed anemia, there was significant difference in serum ferritin levels. Cost of the treatment in IV group was less than Oral group.

SUMMARY

The present Randomized control study was done in SHRI. B.M. PATIL Medical College Hospital and Research Centre, Vijayapura to to compare the effect of prophylactic I.S versus Oral ferrous ascorbate in Pregnant Women. Of the total 100 cases, IV I.S group had 50 cases and Oral group had 50 cases. In the IV sucrose group, 12% of cases were lost to follow up, and in the Oral group 6% of cases had lost to follow up, that means compliance in IV I.S group was 88% and 94% in oral group.

The salient features of the study were mentioned below

- Majority of the cases belonged to 21-25 years in the IV Sucrose group (54%), and Oral Ascorbate group (50%).
- The mean age of IV Sucrose group was 24.62 ± 4.07 years, and Oral group was 24.88 ± 3.86 years.
- Majority of the cases belonged to primigravida, i.e., 62% of cases in IV group and 56% of cases in Oral group.
- The mean period of gestation of IV group was 21.76 ± 1.53 weeks, and oral group was 20.38 ± 1.47 weeks.
- The mean Hb increased from baseline to 12 weeks in IV sucrose group (11.81± 0.71 to 12.19 ± 0.68) and but decreased in oral group (11.74 ± 0.86 to 11.63 ± 0.74, respectively). The mean Hb difference between both the

groups was non-significant at baseline (p value 0.67) and significant after 12 weeks **(p value 0.0001).**

- The mean RBC increased from baseline to 12 weeks in IV sucrose group (4.12 ± 0.35 to 5.14 ± 0.84) and but decreased in oral group (4.11 ± 0.41 to 4.01 ± 0.42, respectively). The mean RBC difference between both the groups was non-significant at baseline and after 12 weeks with a p value of 0.85 and 0.19, respectively.
- The mean PCV increased from baseline to 12 weeks in IV sucrose group (34.86 ± 2.59 to 36.12 ± 3.46) and but decreased in oral group (35.3 ± 2.79 to 33.6 ± 2.51, respectively). The mean PCV difference between both the groups was non-significant at baseline (p value 0.42) and significant after 12 weeks (p value 0.0001).
- The mean MCV increased from baseline to 12 weeks in both IV sucrose group (84.7 ± 6.02 to 86.8 ± 6.42) and but decreased in oral group (86.35 ± 5.78 to 84.98 ± 6.99, respectively). The mean MCV difference between both the groups was non-significant at baseline and after 12 weeks with a p value of 0.17 and 0.21, respectively.
- The mean MCH increased from baseline to 12 weeks in both IV sucrose group (28.56 ± 2.48 to 29.26 ± 2.75) and but decreased in oral group (28.55 ± 2.19 to 27.94 ± 2.73, respectively). The mean MCH difference between both the groups was non-significant at baseline (p value 0.98) and significant after 12 weeks (p value 0.02).

- The mean MCHC increased from baseline to 12 weeks in both IV sucrose group (33.6 ± 1.64 to 33.69 ± 1.95) and in oral group (32.99 ± 1.9 to 33.12 ± 1.91, respectively). The mean MCHC difference between both the groups was non-significant at baseline and after 12 weeks with a p value of 0.09 and 0.16, respectively.
- The mean ferritin increased from baseline to 12 weeks in both IV sucrose group (51.9 ± 44.92 to 163.26 ± 76.28) and in oral group (63.33 ± 33.06 to 81.28 ± 65.98, respectively). The mean PCV difference between both the groups was non-significant at baseline (p value 0.35) and significant after 12 weeks (p value 0.0001).
- Of the total cases, anaemia was seen in 6.8% of cases of IV sucrose group, and 17% of cases of Oral group that showed improvement in Hb was more with IV sucrose than with Oral group.
- Regarding side effects, IV Sucrose group had less side effects than Oral group.
- Compliance was more in the oral group (94%) than IV I.S group (88%).
- Cost of the treatment in IV group was lower than oral group.

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CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) S.H.R.I. B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION /RESEARCH

I, the undersigned,_____, D/O W/O_____, aged___years, ordinarily resident of_____do hereby state/declare that **Dr. MAGANTI YAMINI PURNA**. I of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on___at_____(place), and it has been explained to me in my own language that I am suffering from_____ disease (condition). This disease/condition mimics the following diseases. Further **Dr. MAGANTI YAMINI PURNA** informed me that he/she is conducting dissertation/research titled "**A RANDOMISED CONTROL TRIAL TO COMPARE THE ROLE OF INTRAVENOUS IRON SUCROSE VS ORAL FERROUS ASCORBATE FOR PROPHYLAXIS OF ANEMIA IN PREGNANT WOMEN**."

Under the guidance of **Dr. NEELAMMA PATIL**, requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative post-operative, and follow-up observations will be utilized for the study as reference data. The Doctor has also informed me that during the conduct of this procedure like, adverse results may beencountered. Among the above complications, most of them are treatable but are not anticipated; hence there is a chance of aggravation of my condition, and in rare circumstances, it may prove fatal in spite of the anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this

study would help in the evaluation of the results of the study, which is a helpful reference to the treatment of other similar cases in the near future, and also, I may be benefited in getting relieved of suffering or cure of the disease I am suffering. The Doctor has also informed me that information given by me, observations made, photographs and video graphs taken upon me by the investigator will be kept secret and notassessed by anyone other than me or my legal hirer except for academic purposes. The Doctor did inform me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during the course of treatment/study related to diagnosis, the procedure of treatment, the result of treatment or the prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any timeif I want, or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged. After understanding the nature of the dissertation or research, diagnosis made, and mode of treatment, I, the undersigned Smt_____, under my whole conscious state of mind, agree to participate in the said research/dissertation.

Signature of patient: Signature of Doctor:

Date:
Place:

B.L.D.E (DEEMED TO BE UNIVERSITY) ಶ್ರೀ ಬಿ.ಎಂ.ಪಟ್ಟೀಲ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜು,

ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ವಿಜಯಪುರ-586103

ಪ್ರಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಮಾಹಿತಿ ಪಡೆದ ಸಮ್ಮತಿ

ನಾನು, ಕೆಳಗಿನವರು_____ ಸಹಿಯಿಟ್ಟವರು, ಮಗ/ಮಗಳು/ಪತ್ನಿಯ _____ ವಯಸ್ಸು _____ವರ್ಷಗಳು, ಸಾಮಾನ್ಯವಾಗಿ ನಿವಾಸಿಸುವ ಸ್ಥಳದ ಹೆಸರು_____, ಇಲ್ಲಿ ಹೇಳಿದ್ದೇನೆ/ಘೋಷಿಸುತ್ತೇನೆ ಡಾಕ್ಟರ್ ಹೆಸರು_____ ಅವರು ಆಸ್ಪತ್ರೆ ಹೆಸರು_____ ಅವರು ನನ್ನನ್ನು ಪೂರ್ಣವಾಗಿ ಪರೀಕ್ಷಿಸಿದರು ದಿನಾಂಕದಲ್ಲಿ_____ ಸ್ಥಳ ಹೆಸರು_____ ಮತ್ತು ನನಗೆ ನನ್ನ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ ನಾನು ಒಂದು ರೋಗ (ಸ್ಥಿತಿ) ಅನುಭವಿಸುತ್ತಿದ್ದೇನೆ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ಅವರು ಒಂದು ಪದ್ಧತಿ/ಸಂಶೋಧನೆ ನಡೆಸುತ್ತಿದ್ದಾರೆ ಶೀರ್ಷಿಕೆಯುಳ್ಳ_____ ಡಾಕ್ಟರ್_____ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ಕೇಳಿದ್ದಾರೆ ಅಧ್ಯಯನದಲ್ಲಿ.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ಈ ಕ್ರಮದ ನಡೆವಲ್ಲಿ ಪ್ರತಿಕೂಲ ಫಲಿತಾಂಶಗಳನ್ನು ಎದುರಿಸಬಹುದು. ಮೇಲೆ ಹೇಳಿದ ಪ್ರಕಟಣೆಗಳಲ್ಲಿ, ಅಧಿಕಾಂಶವು ಚಿಕಿತ್ಸಿಸಬಹುದಾದರೂ ಅದನ್ನು ನಿರೀಕ್ಷಿಸಲಾಗುತ್ತಿಲ್ಲ ಆದ್ದರಿಂದ ನನ್ನ ಸ್ಥಿತಿಯ ಹಿರಿದಾಗುವ ಅವಕಾಶವಿದೆ ಮತ್ತು ಅಪರೂಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ ಅದು ಮರಣಕಾರಕವಾಗಿ ಪರಿಣಮಿಸಬಹುದು ಹೊಂದಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಯಥಾಶಕ್ತಿ ಚಿಕಿತ್ಸೆ ಮಾಡಲು ಹೊಂದಿದರೂ. ಮುಂದುವರಿದು ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳ ಮೌಲ್ಯಮಾಪನದಲ್ಲಿ ಸಹಾಯಕವಾಗುತತ್ತದೆ ಇತರ ಸಮಾನ ಪ್ರಕರಣಗಳ ಚಿಕಿತ್ಸೆಗೆ ಉಪಯುಕ್ತ ಉಲ್ಲೇಖವಾಗಿದೆ, ಮತ್ತು ನಾನು ಅನುಭವಿಸುವ ರೋಗದಿಂದ ವಿಮುಕ್ತಿ ಅಥವಾ ಗುಣಮುಖಗೊಳ್ಳುವಲ್ಲಿ ನನಗೆ ಪ್ರಯೋಜನವಾಗಬಹುದು.

ಡಾಕ್ಟರ್ ನನಗೆ ಇದನ್ನು ಕೂಡಾ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿ, ಮಾಡಿದ ಪರಿಶೀಲನೆಗಳು / ಫೋಟೋಗ್ರಾಫ್‌ಗಳು / ವೀಡಿಯೋ ಗ್ರಾಫ್‌ಗಳು ನನ್ನ ಮೇಲೆ ತೆಗೆದುಕೊಳ್ಳಲಾಗುವ ಅನ್ವೇಷಕರು ರಹಸ್ಯವಾಗಿ ಇಡುವರು ಮತ್ತು ನಾನು ಅಥವಾ ನನಗೆ ಕಾನೂನು ದೃಷ್ಟಿಯಲ್ಲಿ ಸಂಬಂಧಿತrannu

ಹೊರತುಪಡಿಸಿ ಇತರ ವ್ಯಕ್ತಿಯಿಂದ ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದಿಲ್ಲ. ಡಾಕ್ಟರ್ ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ ಶುದ್ಧವಾಗಿ ಸ್ವೇಚ್ಛಾಯಿತ, ನನ್ನಿಂದ ನೀಡಿದ ಮಾಹಿತಿಯ ಆಧಾರದ ಮೇಲೆ, ಚಿಕಿತ್ಸೆ / ಅಧ್ಯಯನದ ಸಂಬಂಧದಲ್ಲಿ ರೋಗನಿರ್ಧಾರ, ಚಿಕಿತ್ಸೆಯ ವಿಧಾನ, ಚಿಕಿತ್ಸೆಯ ಫಲಿತಾಂಶ ಅಥವ ಆ ಭವಿಷ್ಯದ ಪ್ರವೃತ್ತಿಗಳು ಬಗ್ಗೆ ಯಾವುದೇ ಸ್ಪಷ್ಟತೆ ಕೇಳಬಹುದು. ಅದೇ ಸಮಯದಲ್ಲಿ ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ನಿಲ್ಲಿಸಬಹುದು ನಾನು ಬಯಸಿದರೆ ಅಥವಾ ಅನ್ವೇಷಕರು ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನನ್ನನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಪ್ರಬಂಧ ಅಥವಾ ಸಂಶೋಧನೆಯ ಸ್ವಭಾವ, ಮಾಡಿದ ರೋಗನಿರ್ಧಾರ ಮತ್ತು ಚಿಕಿತ್ಸೆಯ ವಿಧಾನವನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡು, ನಾನು ಕೆಳಗಿನ ಶ್ರೀ / ಶ್ರೀಮತಿ_____ ನನ್ನ ಪೂರ್ಣವಾದ ಪ್ರಜ್ಞೆಯ ಸ್ಥಿತಿಯಲ್ಲಿ ಹೇಳಿದ ಸಂಶೋಧನೆ / ಪ್ರಬಂಧದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ ಡಾಕ್ಟರನ ಸಹಿ

ಸಾಕ್ಷಿಗಳು

1)

2)

PROFORMA

TITLE OF THE STUDY: **"A RANDOMISED CONTROL TRIAL TO COMPARE THE ROLE OF INTRAVENOUS IRON SUCROSE VS ORAL FERROUS ASCORBATE FOR PROPHYLAXIS OF ANEMIA IN PREGNANT WOMEN."**

NAME:	AGE/SEX:	IP/OP NO-
ADDRESS:	PH NO:	

CHIEF COMPLAINTS:

MARITAL HISTORY: OBSTETRIC HISTORY:

LMP: EDD: POG:

EDD[S]: POG:

TEMPERATURE: PULSE: BLOOD PRESSURE:

CVS: R.S.: PALLOR: PER ABDOMEN:

	GROUP A (IV IRON)	**GROUP B (ORAL IRON)**
AT 20-24WEEKS		

AT 24-28WEEKS		
AT 28-32WEEKS		

INVESTIGATIONS [GROUP A IV IRON] / [GROUP B ORAL IRON]

DATE:	**BASELINE**	**>4 WEEKS**	**>12WEEKS**
HB			
RBC COUNT			
PCV			
MCV			
MCH			
MCHC			
Sr. FERRITIN			

SIDE EFFECTS

	GROUP A [IV IRON]	**GROUP B [ORAL IRON]**
NAUSEA		
VOMITING		
CONSTIPATION		
DIARRHOEA		
NONCOMPLIANCE		
ANAPHYLACTIC REACTIONS		
LOCAL PAIN		

ANY OTHERS		

ETHICAL CLEARANCE CERTIFICATE

BLDE

(DEEMED TO BE UNIVERSITY)

Declared as Deemed to be University u/s 3 of UGC Act, 1956

Accredited with 'A' Grade by NAAC (Cycle-2)

The Constituent College

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 959/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: " A RANDOMISED CONTROL TRIAL TO COMPARE THE ROLE OF INTRAVENOUS IRON SUCROSE V/S ORAL FERROUS ASCORBATE FOR PROPHYLAXIS OF ANEMIA IN PREGNANT WOMEN".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.MAGANTI YAMINI PURNA

NAME OF THE GUIDE: DR.NEELAMMA PATIL , PROFESSOR, DEPT. OF OBSTETRICS AND GYNAECOLOGY.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr. Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
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College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

Master Chart

S.No	NAME	AGE	GROUP	RESIDENCE	GRAVIDA	PARITY	LIVING	ABORTION	DEATH
1	ANJAMMA	25	Oral	Rural	1	Nullipara	0	0	0
2	VIDHYA	23	IV	Urban	4	Para 1	1	2	0
3	BHAGYASREE	25	Oral	Rural	4	Para 3	3	0	0
4	SHOBHA	20	IV	Rural	1	Nullipara	0	0	0
5	GOURAMMA	25	IV	Urban	2	Para 1	1	0	0
6	RENUKA	28	Oral	Rural	3	Para 1	1	1	0
7	PRATHIBHA	32	Oral	Rural	2	Para 1	1	0	0
8	VIJAYALAKSHMI	25	Oral	Rural	2	Para 1	1	0	0
9	LAXMI	22	IV	Rural	3	Para 2	2	0	0
10	RAMYA	21	Oral	Urban	1	Nullipara	0	0	0
11	SAHIDA	25	Oral	Rural	1	Nullipara	0	0	0
12	PARVATHI	28	Oral	Urban	3	Para 2	1	0	1
13	PUSPHA	26	IV	Rural	2	Nullipara	0	1	0
14	KAVERI	22	Oral	Rural	2	Para 1	1	0	0
15	KAMALA	28	IV	Urban	6	Para 2	1	3	1
16	NIKITHA	23	IV	Rural	2	Para 1	1	0	0
17	POOJA	20	Oral	Rural	1	Nullipara	0	0	0
18	NIVEDITHA	24	Oral	Rural	3	Nullipara	0	2	0
19	ROOPA	25	IV	Rural	2	Nullipara	0	1	0
20	SANDHYA KUMBAR	25	Oral	Rural	2	Nullipara	0	1	0
21	RAMYA BIRADAR	28	IV	Rural	1	Nullipara	0	0	0
22	SHOBHA K	35	IV	Urban	1	Nullipara	0	0	0
23	ARATI	20	Oral	Rural	1	Nullipara	0	0	0
24	AISHWARYA ANAND	21	IV	Rural	4	Para 1	1	2	0
25	SHAKEELA	31	IV	Rural	2	Para 1	1	0	0
26	SUPRIYA	21	Oral	Rural	3	Nullipara	0	2	0
27	NIRMALA	28	Oral	Urban	3	Para 1	1	1	0
28	CHAITRA	25	IV	Rural	4	Para 2	2	1	0
29	POOJA SHRAVAN	20	IV	Urban	1	Nullipara	0	0	0
30	GURUDEVI	26	IV	Rural	1	Nullipara	0	0	0
31	SRAVYA	22	Oral	Urban	2	Para 1	1	0	0
32	PREETI	28	Oral	Rural	3	Para 2	2	0	0
33	NIRMAALA	20	Oral	Rural	2	Para 1	1	0	0
34	GEETA	30	Oral	Rural	4	Para 2	2	1	0
35	SAVITHRI	28	IV	Urban	4	Para 3	3	0	0
36	SUNITHA	20	Oral	Rural	1	Nullipara	0	0	0
37	PRATHIBA	23	Oral	Rural	2	Para 1	1	0	0
38	CHANDRAVVA	21	IV	Rural	1	Nullipara	0	0	0
39	VIDYA	35	IV	Rural	3	Para 2	2	0	0
40	GOURI	24	IV	Urban	2	Para 1	1	0	0
41	ASHWINI	22	IV	Rural	2	Para 1	1	0	0
42	KHAIRUNBI	23	Oral	Rural	2	Para 1	1	0	0
43	RENUKAA	28	Oral	Rural	3	Para 2	2	0	0
44	SHAKARAMMA	32	IV	Urban	2	Para 1	1	0	0
45	SAKKUBAI	23	IV	Rural	3	Para 2	2	0	0

S.No	POG (weeks)	B-hb	B-rbc	B-pcv	B-mcv	B-mch	B-mchc	B-ferritin	(>4wk) hb	(>4wk) rbc	(4>wk) pcv	(>4wk) mcv	(>4wk) mch
1	20	13	4.13	34.2	82.8	31	37.4	9.2	12.6	4.14	34.8	82.4	30.4
2	20	11	3.75	30.8	82.1	29.6	36	100.4	11.6	4.08	32.6	81.5	29
3	22	11	3.91	32.4	82.9	29.2	35.2	6.1	11	3.72	33.7	90.6	29.8
4	21	12	4.05	31.9	78.8	29.1	37	19.3	11.6	4.32	33.9	78.5	26.9
5	20	11	3.55	30.5	85.9	29.6	34.4	31.2	11	3.83	34.1	89	29.2
6	22	14	4.58	38.3	83.6	29.7	35.5	20.4	9.8	3.94	29.9	75.9	24.9
7	20	12	3.66	33.4	91.3	33.1	36.2	51.3	12.3	4.05	35.6	87.9	30.4
8	23	11	3.73	32	85.8	29	33.8	9.2	9.9	3.92	35.7	91.1	25.3
9	20	11	4.37	32.8	75.1	25.2	33.5	45.6	10.8	4.07	33.3	81.8	26.3
10	21	12	4.34	37.3	85.9	27.9	32.4	95.9	10.8	3.85	33.2	86.2	28.1
11	23	11	4.07	33.3	81.8	26.3	32.1	29.6	9.5	3.54	29.3	82.8	26.8
12	21	12	4.7	36.9	78.5	25.7	32.8	11.8	11.3	3.85	33.3	86.5	29.4
13	22	12	3.94	34.7	88.1	30.5	34.6	91.6	11.8	3.76	31.9	84.8	26.9
14	23	11	3.63	30.9	85.1	29.2	34.3	13.5	10.9	3.59	31.4	87.5	30.4
15	23	11	4.26	29.9	70.2	22.8	32.4	52.5	13.5	4.62	41.8	90.5	29.2
16	24	11	4.06	35.3	88	28.6	32.4	81.8	11.8	4.08	35.1	86	28.9
17	21	12	4.13	35.8	86.7	27.8	32.1	46.1	11.1	4.06	33.9	83.5	27.3
18	24	11	3.61	34.7	96.1	31.6	32.9	35.2	11.4	3.59	33.9	94.4	31.8
19	24	11	3.88	33.8	87.1	29.4	33.7	47.2	10.7	3.96	32.8	82.8	27
20	20	12	3.94	34.7	88.1	29.9	34	68.3	11	3.86	33.5	86.8	28.5
21	22	12	4.34	37.3	85.9	27.9	32.4	95.9	10.8	3.85	33.2	86.2	28.1
22	20	13	4.13	34.2	82.8	31	37.4	48.6	10.8	3.79	33.4	88.1	28.5
23	23	13	4.68	38.3	81.8	27.6	33.7	69.6	11.4	3.98	33.3	83.7	28.6
24	24	11	4.08	33.7	82.6	27.7	33.5	4.6	10.7	3.58	30.7	85.8	29.9
25	20	12	4.25	35.4	83.3	28.2	33.9	21.1	11.6	4.08	35.8	87	28.4
26	20	11	3.59	32.3	90	29.2	32.5	319.7	10.2	3.42	34.1	92.1	28.6
27	22	13	4.57	40.3	88.2	27.4	31	125.4	10.8	3.55	33.3	93.8	30.4
28	21	11	3.82	30	78.5	27.5	35	8.6	10	3.78	28.4	75.1	26.5
29	20	11	3.81	33.2	87.1	29.4	33.7	30.9	10.7	3.65	32.2	88.2	29.3
30	24	12	4.05	38.7	95.6	30.6	32	170.3	9.6	3.4	38.2	82.9	28.2
31	21	11	3.67	35.8	97.5	31.1	31.8	201.5	9.2	2.68	27.7	103.4	34.3
32	22	11	3.85	33.3	86.5	29.4	33.9	86.3	11.8	4.18	36.8	88	28.2
33	20	11	3.64	32.1	88.2	30.5	34.6	116.3	11.4	3.59	33.9	94.4	31.8
34	20	11	3.86	32	82.9	27.5	33.1	32.3	10.5	4.51	33.1	73.4	23.3
35	23	11	3.17	32.5	102.5	35.6	34.8	13.5	12	4.75	37.6	79.2	25.3
36	24	11	4.31	34.6	80.2	25.5	31.8	4.3	10.4	4.41	34.8	78.6	24.2
37	24	11	3.51	29.9	85.2	27.6	32.4	136.5	10.5	3.83	32.3	84.3	27.4
38	22	12	4.16	32.7	78.6	27.9	35.5	19.6	11.9	4.31	36.6	84.9	27.6
39	24	12	4.22	35.2	83.4	27.5	33	65.5	11.3	3.68	33	89.7	30.7
40	22	12	4.27	37.6	88.1	27.4	31.1	45.3	11	3.96	33.4	84.3	27.8
41	21	14	4.58	38.3	83.6	29.7	35.5	48	10.8	3.98	29.9	75.8	24.7
42	23	11	3.88	32.3	83.2	28.9	34.7	19.5	9.9	3.56	31.2	87.6	27.8
43	20	11	3.91	33.6	85.9	27.9	32.4	17	10.4	3.42	31	90.6	30.4
44	21	13	4.24	38.1	89.9	31.1	34.6	65.7	12.6	4.36	37.4	85.8	28.9
45	23	11	3.57	31.2	87.4	30	34.3	4.6	11	3.51	33.4	95.2	31.3

S.No	(>4wk) mchc	(>4wk) ferritin	(12wk) Hb final	(12wk) rbc	(12wk) pcv	(12wk) mcv	(12wk) mch	(12wk) mchc	(12wk) ferritin	nausea	vomiting
1	36.2	9.4	11.5	4.02	32.6	82	31.5	36.7	8.8	0	0
2	35.6	101.7	12.1	3.86	33.5	86.8	31.5	36.1	108.9	0	0
3	32.9	6.8	12	4.7	33.6	81.2	28.1	32.4	16.6	1	0
4	34.2	120.2	11.6	4.88	32.9	78.9	29.4	37.6	122.6	0	0
5	32.8	52.5	12	3.89	31.5	89.7	30.5	34.8	63.8	0	0
6	32.8	18.6	12.8	4.32	32.1	74.3	24.3	32.7	24.1	0	0
7	34.6	63	12.3	4.03	34.7	86.1	29.8	34.6	75.3	0	0
8	27.7	12.7	12	4.71	33.3	70.7	21.9	30.9	70.3	1	0
9	32.7	53.7	11.8	4.46	37.6	84.3	26.9	31.9	69	0	0
10	32.5	69.8	12.5	4.27	38.5	90.2	29.3	32.5	77.5	0	0
11	32.4	6.1	11.8	3.6	31	86.1	28.6	33.2	7.2	0	0
12	33.9	86.3	11	3.52	33.7	95.7	32.1	33.5	75.7	0	0
13	31.7	97.8	12.1	3.98	36.8	103.1	35.5	34.5	184.2	0	0
14	34.7	57.4	12	3.94	35.6	90.4	30.2	33.4	58.2	0	0
15	32.3	127.6	11.6	42.9	36.8	86.8	29	33.4	166.8	0	0
16	33.6	158.2	12	3.97	34.8	89.7	30.2	34.5	257.9	0	0
17	32.7	16.6	12.8	3.6	32.4	90	30.3	33.6	28.5	0	0
18	33.6	62.5	12.3	3.51	33.2	94.6	31.6	33.4	108.3	0	0
19	32.6	213.3	12	4.36	35.3	89	26.1	32.3	219.8	0	0
20	32.8	52.6	11.8	3.82	34.1	89.3	27.5	30.8	94.2	0	0
21	32.5	69.8	12.1	4.27	38.5	90.2	29.3	32.5	177.5	0	0
22	32.3	35	12	4.05	38.5	95.1	28.6	30.1	93.7	0	0
23	34.2	44	11.6	4.37	37.5	85.8	26.1	30.4	216.4	0	0
24	34	7.2	12.1	3.28	28	85.4	29.6	34.6	10.6	0	0
25	32.7	169.7	12	4.18	35.5	84.9	28.5	33.5	182.4	0	0
26	30.1	242.5	11.6	4.28	36.8	86	27.3	31.8	79.8	0	0
27	32.4	102	12	3.49	36.6	104.9	31.2	29.8	121.7	0	0
28	35.2	10.95	12.8	4.37	38.3	88.2	38.9	36.7	151.1	0	0
29	33.2	49	12.3	4.39	40	91.1	30.5	33.5	77.1	0	0
30	34	110.8	12	3.63	38.7	106.6	30.6	28.7	183.9	0	0
31	33.2	74.5	11.8	4.41	34.8	78.9	23.6	29.9	31.2	1	0
32	32.1	51.8	12.5	3.17	31.6	99.7	32.2	32.3	166.3	0	0
33	33.6	625.4	11.8	3.86	33.5	86.8	28.5	32.8	785.8	0	0
34	31.7	48.3	11	4.76	37.6	78.4	24.6	38.4	51.2	0	0
35	31.9	71.2	10.9	4.13	35.2	85.2	29.1	34.1	238.5	0	0
36	32.1	11.2	12.9	3.49	36.6	93.8	30.4	34.2	16.4	1	0
37	32.5	126.4	13.4	4.28	35.7	83.4	27.6	33.1	138.5	0	0
38	32.5	36.8	11.1	4.44	37.7	84.9	27.3	32.1	135.9	0	0
39	34.2	120.4	12	4.65	38.7	95.6	30.6	32.3	186.6	0	0
40	32.9	74	12.7	3.81	33.2	87.1	29.4	33.8	184.4	0	0
41	32.6	69.4	12.8	4.32	32.1	74.3	24.3	32.7	104.8	0	0
42	31.7	60.2	11.8	4.51	33.1	73.4	23.3	31.7	27.58	0	0
43	33.5	21.1	10.5	4.18	35.2	84.2	28.9	34.4	22.6	0	0
44	33.7	68.3	12.8	4.22	38.8	88.6	27.8	35.2	178.9	0	0
45	32.9	163.5	11.8	4.08	33.7	82.6	27.7	33.5	212.8	0	0

S.No	epigastric pain /acidity	pain abdomen	Constipation	diarrhea	Rashes	Itching	chills	headache	local pain	local swelling
1	0	1	0	0	0	0	0	0	0	0
2	0	0	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	1	0	0
5	0	0	0	0	0	0	0	0	0	0
6	1	0	0	0	0	0	0	0	0	0
7	0	0	0	1	0	0	0	0	0	0
8	0	0	1	0	0	0	0	0	0	0
9	0	0	0	0	0	0	0	0	0	0
10	0	0	1	0	0	0	0	0	0	0
11	1	0	1	0	0	0	0	0	0	0
12	0	0	0	0	0	0	0	0	0	0
13	0	0	0	0	0	0	0	0	0	0
14	0	0	0	0	0	0	0	0	0	0
15	0	0	0	0	0	0	0	0	0	0
16	0	0	0	0	0	0	0	0	1	0
17	0	0	0	0	0	0	0	0	0	0
18	0	1	0	0	0	0	0	0	0	0
19	0	0	0	0	0	0	1	1	0	0
20	0	0	0	0	0	0	0	0	0	0
21	0	0	0	0	0	0	0	0	0	0
22	0	0	0	0	0	0	0	0	1	0
23	0	0	0	0	0	0	0	0	0	0
24	0	0	0	0	0	0	0	0	0	0
25	0	0	0	0	0	0	0	0	0	0
26	0	0	0	0	0	0	0	0	0	0
27	0	0	0	0	0	0	0	0	0	0
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32	0	0	0	0	0	0	0	0	0	0
33	0	0	0	0	0	0	0	0	0	0
34	0	0	0	0	0	0	0	0	0	0
35	0	0	0	0	0	0	1	0	1	0
36	1	0	0	0	0	0	0	0	0	0
37	0	0	0	0	0	0	0	0	0	0
38	0	0	0	0	1	0	0	0	0	0
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40	0	0	0	0	0	0	0	0	0	0
41	0	0	0	0	0	0	0	0	0	0
42	0	0	0	0	0	0	0	0	0	0
43	0	0	0	0	0	0	0	0	0	0
44	0	0	0	0	0	0	0	0	0	0
45	0	0	0	0	0	0	0	0	1	0

S.No	NAME	AGE	GROUP	RESIDENCE	GRAVIDA	PARITY	LIVING	ABORTION	DEATH
46	SAVITHA	22	Oral	Rural	1	Nullipara	0	0	0
47	SAROJINI	22	Oral	Rural	2	Nullipara	0	1	0
48	SUJATHA	24	Oral	Rural	3	Para 2	2	0	0
49	SUSHMA	26	Oral	Rural	3	Para 2	2	0	0
50	BHAGHYASHRI	24	IV	Rural	1	Nullipara	0	0	0
51	PRATEEKSHA	22	IV	Urban	2	Nullipara	0	1	0
52	MAHESWARI	20	IV	Rural	1	Nullipara	0	0	0
53	BASAMMA	24	Oral	Rural	1	Nullipara	0	0	0
54	ASHARANI	20	IV	Rural	1	Nullipara	0	0	0
55	HASEENA	25	IV	Rural	4	Para 3	3	0	0
56	SANVI	21	Oral	Urban	1	Nullipara	0	0	0
57	KAVITA	32	Oral	Urban	3	Para 1	1	1	0
58	MUSKAN	20	Oral	Rural	2	Nullipara	0	1	0
59	NAGAMMA	19	IV	Urban	1	Nullipara	0	0	0
60	SAMATHA	25	Oral	Rural	1	Nullipara	0	0	0
61	PALLAVE	22	IV	Urban	2	Para 1	1	0	0
62	PARVEEN	22	IV	Rural	2	Para 1	1	0	0
63	KAVERI	19	Oral	Urban	1	Nullipara	0	0	0
64	DANESWARI	30	Oral	Rural	2	Nullipara	0	1	0
65	SUSHMITHA	25	IV	Rural	2	Para 1	1	0	0
66	ALFIYA	21	IV	Rural	3	Para 2	2	0	0
67	AMBIKA	32	Oral	Rural	3	Nullipara	0	2	0
68	MAREMMA	29	IV	Rural	2	Nullipara	0	1	0
69	SUNANDA	28	Oral	Rural	2	Para 1	1	0	0
70	KAVITA	19	Oral	Urban	1	Nullipara	0	0	0
71	SUNITA	27	IV	Rural	1	Nullipara	0	0	0
72	VANISHREE	21	IV	Urban	2	Nullipara	0	1	0
73	RUBINA	25	Oral	Rural	1	Nullipara	0	0	0
74	KAVERI	19	Oral	Rural	1	Nullipara	0	0	0
75	SANIYA	22	IV	Rural	2	Para 1	1	0	0
76	RENUKA	25	IV	Urban	1	Nullipara	0	0	0
77	ROOPA	34	Oral	Rural	3	Para 2	2	0	0
78	KAVITHA	22	IV	Rural	3	Para 2	2	0	0
79	RAJASHRE	24	Oral	Urban	1	Nullipara	0	0	0
80	LALITHA	20	IV	Rural	2	Nullipara	0	1	0
81	BHAGYASRI	27	IV	Rural	3	Para 2	2	0	0
82	MANISHA	25	Oral	Rural	2	Nullipara	0	1	0
83	VAISHNAVI	22	Oral	Urban	2	Para 1	1	0	0
84	RESHMA	24	Oral	Urban	2	Para 1	1	0	0
85	MALLAMMA	26	IV	Rural	1	Nullipara	0	0	0
86	SANDYA	29	IV	Urban	2	Para 1	1	0	0
87	BHAGYASRI	34	Oral	Rural	3	Para 2	2	0	0
88	AMBIKA	22	IV	Rural	1	Nullipara	0	0	0
89	SWETHA	25	IV	Rural	3	Para 1	1	1	0
90	LEELAVATHI	31	Oral	Urban	1	Para 2	0	0	1
91	RAZIYA	27	Oral	Urban	3	Para 2	1	0	1

S.No	P0G (weeks)	B-hb	B-rbc	B-pcv	B-mcv	B-mch	B-mchc	B-ferritin	(>4wk) hb	(>4wk) rbc	(4>wk) pcv	(>4wk) mcv	(>4wk) mch
46	23	11	3.77	33.3	88.3	29.4	33.3	112	10.5	3.33	31.3	94	31.5
47	24	11	4.17	34.7	83.2	26.2	32	364.3	11.1	3.56	33.8	94.9	31.2
48	20	11	3.56	32.5	92.9	30	32.3	18.6	10.4	4.41	34.8	78.6	24.3
49	24	11	4.73	37.5	79.3	23.6	23.9	36	10.8	3.56	33.7	93.6	30.7
50	21	12	4.01	35.3	88	28.9	32.9	8.6	10.2	3.43	31.8	92.7	29.7
51	20	11	4.11	34.1	83	27	32.6	98.6	13.2	4.45	39.9	89.7	29.7
52	20	11	4.6	34.4	74.8	23.9	32	30	10.8	4.27	33	77.3	25.3
53	21	12	4.16	36.5	87.7	29.1	33.2	88.8	11.3	3.97	32.6	104.3	35.8
54	21	12	3.93	34.2	87	30.3	34.8	137.3	11.3	3.17	32.5	102.5	35.6
55	20	13	4.71	39.2	83.2	27.4	32.9	82.5	12.4	4.05	38.6	95.7	30.8
56	20	11	3.54	34.6	97.7	31.9	32.7	10.5	8.9	2.59	25.3	97.7	34.3
57	21	12	4.3	36.1	84	26.7	31.9	73.1	10.7	3.58	30.7	85.8	29.9
58	23	11	3.96	36.5	92.2	28.8	31.2	39.3	11	3.51	33.4	95.2	31.3
59	20	12	4.67	37.1	79.4	25.9	32.6	38.5	11.6	4.01	35.3	88.1	28.6
60	22	12	4.11	36.5	88.8	29.4	33.2	76.5	11.6	4.01	33.3	88	28.9
61	21	11	4.73	35.3	74.6	23.7	31.7	9.6	11.6	4.84	34.8	78.6	27.9
62	23	12	4.2	36.6	87.4	27.9	32	97.6	11.2	3.81	33.2	87.1	29.4
63	20	11	3.68	36.6	104.7	31.2	29.4	9.4	10.8	3.55	33.3	93.8	30.4
64	20	11	3.99	33.3	83.5	28.6	34.2	12.8	11	3.96	33.4	84.3	27.8
65	20	12	4.22	35.2	83.4	27.5	33	65.5	11.3	3.68	33	89.7	30.7
66	21	13	3.82	36.6	95.8	32.7	34.2	56.4	11.3	3.17	32.5	98.6	34.7
67	20	12	3.99	35.4	88.7	29.3	33.1	12.2	11.4	3.97	32.6	98.2	35.7
68	23	12	4.77	36.9	77.4	25.2	32.5	6.4	11.6	3.95	33.3	84.3	28.6
69	21	13	4.2	37.4	89	30	33.7	48	11.2	3.72	32.8	88.2	29.8
70	20	12	4.52	36.6	81	26.8	33.1	26.2	11.8	3.92	34.6	88.3	30.1
71	24	11	4.36	33.3	76.4	25.2	33	12.8	10.6	3.86	32	82.9	27.5
72	20	11	4.05	33.2	82	25.4	31	64.8	11.9	3.96	35.5	89.6	30.1
73	22	11	4.88	35.3	72.3	23	31.2	21.8	10.4	4.45	33.7	75.7	24.5
74	24	13	4.37	37.4	85.6	30	35	12	12	4.17	38.9	93.3	28.8
75	24	12	4.15	35.1	84.6	29.4	34.8	43.8	11	4.32	35.5	82.2	27.3
76	22	13	4.56	42.1	92.3	32.5	35.2	84.2	13.3	4.1	39.9	92.9	32.4
77	22	14	4.69	39.7	84.6	29	34.3	24.1	12.7	4.41	37.7	85.5	28.8
78	23	11	3.68	33.3	90.5	31	34.2	241	12.1	4.52	36.6	81.2	26.9
79	24	13	4.34	38.1	87.8	30	34.1	74.8	11.2	4.27	30.3	71	22
80	20	12	3.94	35.2	88.7	30	33.8	34.9	12	4.09	33.5	81.9	26.9
81	21	13	4.74	33.3	79.3	27	34	23.1	12.1	4.81	39.5	82.1	26.6
82	23	11	3.95	33.2	84.1	28.4	33.7	141	10.8	3.72	32.6	87.6	29.3
83	23	12	4.82	36.5	75.7	24.5	32.3	23.2	11.2	4.3	35.2	81.9	27.9
84	21	12	4.24	35.7	84.2	27.6	32.8	20.6	11.4	4.34	34.7	80	24.9
85	22	13	4.09	36.2	86.5	30.6	34.5	29.6	12.6	4.12	38.7	86.2	32.1
86	24	12	4.51	37.1	82.3	27.1	32.9	21.4	11.8	3.86	33.7	87.3	29.5
87	20	12	4.52	37.3	82.5	27	32.7	70	11.8	4.82	40.7	80.1	28.6
88	21	13	4.38	38.9	88.8	29	32.6	52.7	12.8	4.39	36.3	82.7	27.6
89	24	13	4.25	37	87.1	30.1	34.6	46.3	11.9	4.6	34.8	75.7	25.2
90	23	13	4.46	40.1	89.9	29.1	32.4	48.4	12.2	4.36	33.3	76.4	25.2
91	22	14	4.24	40.5	95.5	32.5	34.1	157.5	12.4	4.3	36.9	85.8	29.8

S.No	(>4wk) mchc	(>4wk) ferritin	(12wk) Hb final	(12wk) rbc	(12wk) pcv	(12wk) mcv	(12wk) mch	(12wk) mchc	(12wk) ferritin	nausea	vomiting
46	33.5	54	10.9	4.07	33.3	81.8	26.3	32.1	129.8	0	0
47	32.8	118.8	12.8	3.82	34.1	89.3	27.5	30.8	128.6	0	0
48	33.2	21.2	12.2	4.81	34.6	80.2	25.5	31.8	28.9	0	0
49	32.4	42.6	12.4	3.66	32.2	89.3	30.6	35.2	26.8	0	0
50	32.1	153.1	13.2	3.51	31.2	87.4	30	34.3	186.4	0	0
51	33.1	137.5	12.4	4.13	34.2	82.8	31	37.6	186.9	0	0
52	32.7	106.9	12	3.82	33.3	88.2	29.6	33.1	142.6	0	0
53	34.2	94.2	12.2	4.16	32.7	78.6	27.9	35.5	100.4	0	0
54	34.8	142.8	12.6	4.48	39.8	89.9	29.6	33.4	164.4	0	0
55	32.4	98.6	12.8	4.67	42.2	90.4	28.3	31.3	252.2	0	0
56	35.2	86.9	12.4	3.52	31.2	87.4	30	34.3	42.1	1	1
57	34	84.4	11.8	4.08	33.7	82.6	27.7	38.5	86.2	0	0
58	32.9	44.6	12.1	3.52	31.2	87.4	30	34.3	48.8	0	0
59	33.4	21.7	12.4	4.88	38.2	84.6	30.2	34.8	204.6	0	0
60	32.9	78.3	12.4	4.95	37.6	79.2	25.3	31.9	86.6	0	0
61	35.8	36.2	12.6	4.95	37.6	79.2	25.3	31.9	71.8	0	0
62	33.7	119.4	12.8	4.26	38.5	88.7	30.1	34.8	212.8	0	0
63	32.6	12.6	11	3.42	33.1	88.9	30.4	34.2	18.2	0	1
64	32.9	21.7	13	3.41	31.1	89.7	30.4	33.2	18	0	1
65	34.2	72.4	11.6	4.24	38.1	94.2	33.6	38.2	102.6	0	0
66	35.2	89.8	13.2	4.46	39.2	87.9	27.4	31.1	171.3	0	0
67	34.2	18.4	12.8	4.01	34.3	88.6	29.6	35.1	22.2	0	0
68	33.9	80.4	12.4	4.15	36.1	87	30.8	35.5	121.2	0	0
69	33.8	52	11.1	4.08	35.2	86.3	29.4	34.1	60.6	0	0
70	34.1	53.8	11.2	4.08	35	85.8	29.7	34.6	88.6	0	0
71	33.1	8.6	11.8	4.94	36	72.9	23.1	31.7	60.4	0	0
72	33.5	235.6	12.1	4.22	36.8	88.1	30.4	34.2	284.8	0	0
73	32.3	24.2	11.5	3.88	29.3	75.5	24.5	32.4	25.6	1	1
74	30.8	66.4	11	4.15	34.8	83.9	28.4	33.9	23.2	0	0
75	33.2	64.8	12.4	4.56	37	81.1	28.3	34.9	108.6	0	0
76	34.9	148.1	12.4	4.52	37.1	81.2	28.4	34.6	166.6	0	0
77	33.7	18.2	10.3	3.64	31.8	87.4	28.6	32.7	12.4	0	0
78	33.3	262.2	12.6	4.72	37.5	79.4	26.1	32.8	284.4	0	0
79	31	68.1	10.2	3.48	25.9	76.2	24.1	31.7	63	1	1
80	32.8	186.2	12.8	4.68	41.7	89.1	29.7	33.3	242.8	0	0
81	32.4	32	11	3.69	30.6	82.9	26.6	32	28.8	0	0
82	33.4	112.2	10.9	4.12	30.9	75	22.6	30.1	101.6	1	1
83	34.1	24.8	11.7	4.14	31.8	76.8	24.6	32.1	26.8	0	0
84	31.1	22.4	10.2	4.37	33.3	76.2	24.9	32.7	28.6	0	0
85	34.8	112.4	13	4.93	41.6	84.4	30	35.6	406.2	0	0
86	33.8	48.2	11.6	3.12	27.6	92.2	33.1	33.7	76.4	0	0
87	31	76.4	11	3.83	27.8	91.7	31.7	34.5	79.8	0	0
88	33.3	112.6	13.2	4.52	30.3	76.2	26.2	31.4	203.9	0	0
89	33.3	88.2	12.8	4.63	38.5	83.2	28.1	33.8	192.4	0	0
90	33	52	11.1	4.29	35.9	83.7	27.3	32.6	78.4	0	0
91	34.7	188.4	11.5	4.01	34.8	86.8	27.2	31.9	192.8	0	0

S.No	epigastric pain /acidity	pain abdomen	Constipation	diarrhea	Rashes	Itching	chills	headache	local pain	local swelling
46	0	0	0	0	0	0	0	0	0	0
47	0	0	0	0	0	0	0	0	0	0
48	0	0	0	0	0	0	0	0	0	0
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89	0	0	0	0	0	0	0	0	0	0
90	0	0	1	0	0	0	0	0	0	0
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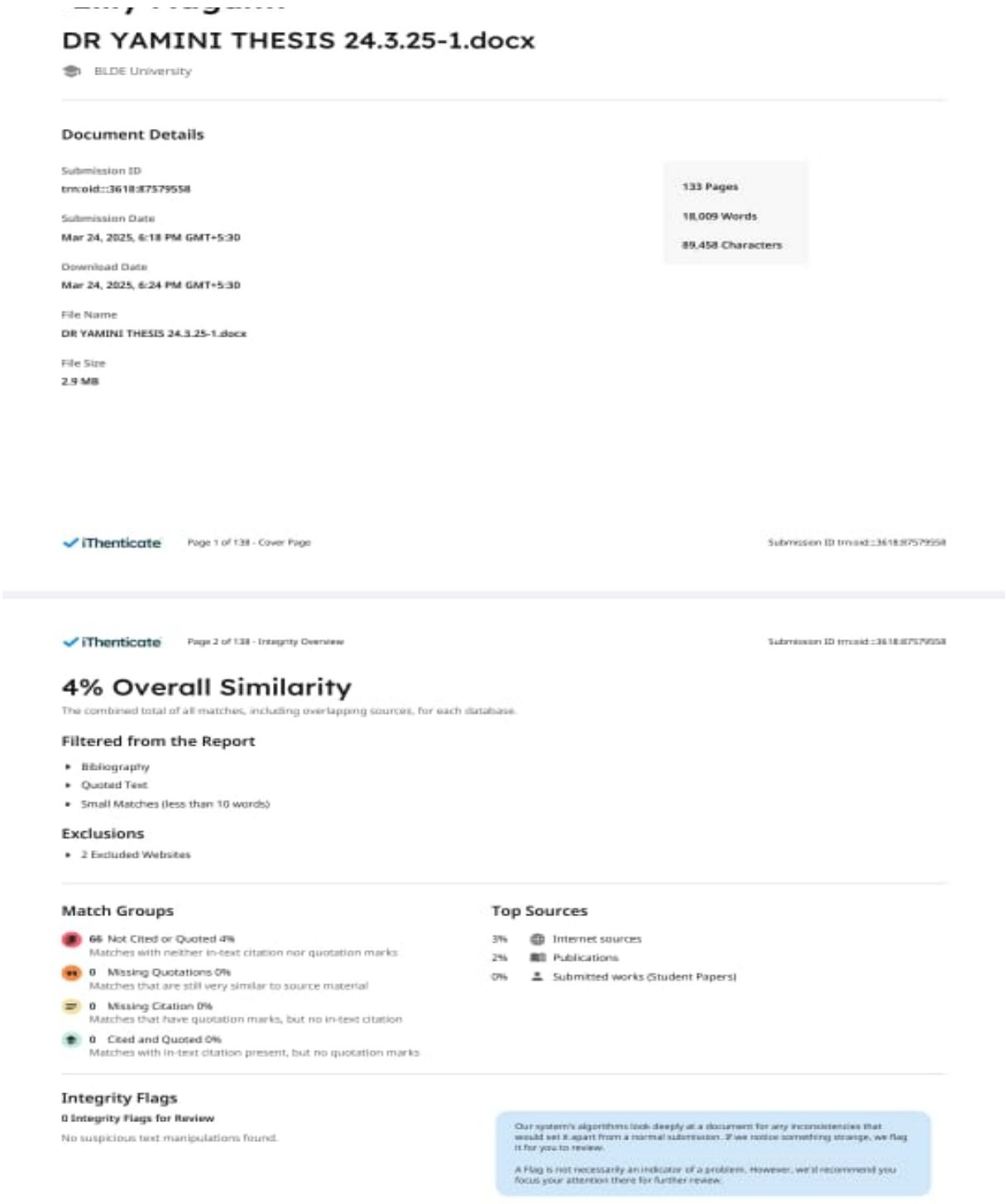

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