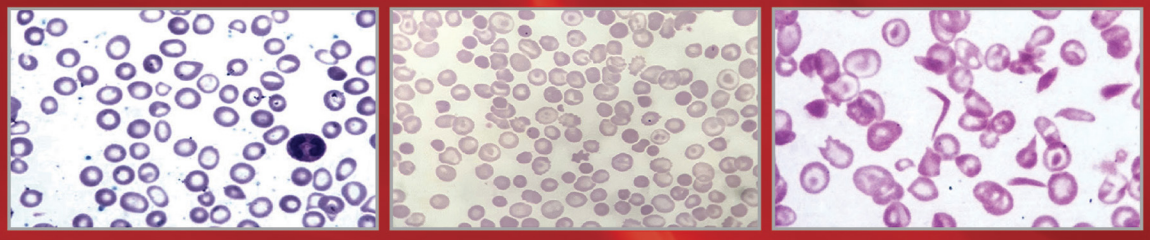
ISSN 2248-9517
2019 Volume 7 Number 1

World Clinics

OBSTETRICS & GYNECOLOGY

Anemia



Editor-in-Chief
Mala Arora

Guest Editors
Sabaratnam Arulkumaran
Manju Puri

JAYPEE

Contents

Editorial xi
Mala Arora

Abbreviations xv

Overview of Anemia **1**
Sadia Mansoor, Manju Puri

Applied Clinical Pathology for the Diagnosis of Anemia **11**
Sunita Sharma, Shivali Sehgal

Iron Deficiency Anemia in Pregnancy—A Review **27**
Swati Agrawal, Manju Puri

Folic Acid and Vitamin B12 Deficiency Anemia **42**
Anju Bhatia, Kazila Bhutia

Hemolytic Anemias **58**
Kanwal Gujral, Sakshi Nayar

Anemia Due to Chronic Diseases in Pregnancy **74**
Muralidhar V Pai, Rekha Upadhya

Thalassemia in Pregnancy: From Screening to Obstetric Management **80**
Tak Yeung Leung

Aplastic Anemia and Myelodysplastic Syndrome **95**
Shipra Gandhi, Aman Gupta

Blood Component Therapy and Transfusion Practice in Obstetrics **112**
Raj N Makroo, Gargi Agarwal

Sickle Cell Anemia **126**
Samip R Master

Pregnancy and Anemia: Special Situations, Severe Anemia in Labor, Decompensated Antepartum Patient **138**
K Aparna Sharma, Kavita Khoiwal

World Clin Obstet Gynecol. 2019;7(1):27-41.

Iron Deficiency Anemia in Pregnancy—A Review

*Swati Agrawal MD, Manju Puri MD

Department of Obstetrics and Gynaecology, Lady Hardinge Medical College
New Delhi, India

ABSTRACT

Iron deficiency anemia (IDA) is the most common nutritional disorder in the world and pregnant women are particularly susceptible to this. Worldwide more than 20% women are iron deficient in their reproductive years, more in developing compared to developed nations. During pregnancy, there is a 2–3 fold increase in requirement of iron, and usual diet is often insufficient to fulfill this demand. Iron, deficiency anemia in pregnancy is associated with an increased maternal and perinatal morbidity and mortality. The interventions to prevent iron deficiency anemia in pregnancy include iron supplementation, food fortification, control of helminthic infections, dietary education and improvement in hygiene and sanitation. Both oral and parenteral iron therapies are effective in the treatment of iron deficiency anemia. The newer parenteral preparations are safe and effective in rapid repletion of iron stores. Blood transfusion remains the last option in the management of severe anemia.

INTRODUCTION

Anemia in pregnancy is a major cause of maternal morbidity and mortality especially in developing nations.[1] According to World Health Organization (WHO) estimates, 41.8% of pregnant women globally are anemic.[2] Iron deficiency is responsible for more than half of these while other micronutrient deficiencies such as folic acid, Vitamin B12 and Vitamin A deficiency, chronic infections, helminthic infestations and inherited disorders of erythropoiesis account for the rest.

*Corresponding author
Email: drswatimail@gmail.com

© 2019 Jaypee Brothers Medical Publishers. All rights reserved.

JAYPEE BROTHERS

World Health Organization estimates that about 5,91,000 perinatal deaths and 11,500 maternal deaths occur per annum worldwide due to iron deficiency anemia either directly or indirectly.[3] India contributes to approximately 80% of maternal deaths due to anemia in South East Asia. Despite more than four decades of public health interventions in India, nearly half of pregnant women still suffer from anemia and it contributes to 20-40% of maternal mortality either directly or indirectly. According to National Family Health Survey 2015-2016 (NFHS-4), the prevalence of anemia is 50.3% in pregnant women in India. In developed nations like UK, anemia continues to be a major health concern affecting approximately 24% of all pregnant women, there has been no reduction in the burden of the disease in the last decade even though national recommendations and guidelines are in place.[4]

This review aims at providing up date information to clinicians to effectively prevent and manage iron deficiency anemia.

IRON METABOLISM IN PREGNANCY

During pregnancy there is an increase in iron requirement, approximately 1000 mg for the entire pregnancy. Of these 500–600 mg red blood cell (RBC) expansion 300 mg for fetus and placenta and rest for the growing uterus. A state of amenorrhea during pregnancy saves about 150 mg of iron hence, pregnancy imposes an additional requirement of 850 mg of iron. The recommended daily dietary allowance for iron in pregnancy is 27 mg which is roughly 3 times the requirement in a nonpregnant woman. A daily dietary allowance of 10 mg is required during lactation.[5]

Absorption of iron usually decreases during the first trimester of pregnancy but increases in the second and third trimesters. The extent of iron absorption from the gut is affected by the type of diet; absorption from heme iron being 15–30% and nonheme iron being 5–8%.[6] The absorption increases by 50% in iron deficiency state.

DEFINITIONS

WHO defines anemia as hemoglobin (Hb) level of less than 11 g/dL and a hematocrit of less than 33%.

The centre for disease control and prevention (CDC) defines anemia as less than 11 g/dL in the first and third trimesters and less than 10.5 g/dL in the second trimester.

The Indian Council of Medical Research (ICMR) categorizes anemia during pregnancy as mild for Hb levels between 10-10.9 g/dL, moderate between 7-9.9 g/dL, severe between 4-6.9 g/dL and very severe if less than 4 g/dL.

JAYPEE BROTHERS

Iron deficiency anemia usually develops over a period of time and has three well defined stages. Stage 1 is the stage of iron depletion where the iron stores get depleted but the hemoglobin level is unaffected. In stage 2 or the stage of iron restricted erythropoiesis, the supply of iron to bone marrow is affected which is reflected by a significant fall in the levels of serum ferritin, plasma iron and transferrin saturation but Hb levels remain unaltered. Stage 3 is the stage of iron deficiency anemia, where Hb levels fall and all the hematological parameters are abnormal.[7]

ETIOLOGICAL FACTORS

There are myriad reasons for iron deficiency anemia in pregnancy especially in developing countries. These include decreased iron intake, chronic blood loss and excess iron demand. Vegetarian diet consisting mainly of cereals rich in phytates, inhibitors of iron absorption and hose having very low bioavailable iron.[8] Excess consumption of tea and coffee reduce the absorption of iron.

High prevalence of worm infestation especially hookworm and heavy menstrual bleeding are important causes chronic blood loss.[9] This translates into negative iron balance and poor iron stores during pregnancy.

Repeated pregnancies at short interval and prolonged periods of lactation deplete women of their iron stores.

CLINICAL PRESENTATION

Clinical features of iron deficiency anemia depend upon the severity of the disease. Women with mild anemia may be asymptomatic however, moderately anemic women present with generalized weakness, easy fatigability, breathlessness on exertion, and poor work performance. Women with severe anemia may have palpitations, breathlessness at rest and generalized anasarca. Sometimes, they may present with features of decompensation and cardiac failure. Other presenting symptoms may be pica or restless leg syndrome or pagophagia which is a symptom specific to iron deficiency. Subtle signs of iron deficiency without clinical signs of anemia include alopecia, atrophy of lingual papillae and dry mouth due to decreased salivation.[10] Women may have angular cheilosis, atrophic glossitis, stomatitis and koilonychia.

DIAGNOSIS AND WORKUP

Early diagnosis of iron deficiency anemia (IDA) is imperative for initiation of timely intervention and optimization of fetomaternal outcome. Most guidelines recommend that pregnant women should be screened for anemia at booking and at 28 weeks of gestation.

JAYPEE BROTHERS

Complete blood count is a simple and inexpensive screening tool for diagnosing IDA. Low hemoglobin and reduced mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) are suggestive of IDA. Red cell distribution width (RDW) measures the change in the width of red blood cells and hence, measures variability in the red blood cell volume distribution. Anisocytosis in iron deficiency anemia leads to increased RDW. Its sensitivity is higher than MCV in the diagnosis of IDA. A sharp rise in RDW following initiation of iron therapy due to marked reticulocytosis strongly supports the diagnosis of IDA.

Peripheral blood film can further help in differentiating iron deficiency anemia from megaloblastic and hemolytic anemia. In iron deficiency anemia, peripheral blood film shows presence of microcytosis, anisocytosis, poikilocytosis and hypochromia. On the basis of the results of complete blood count with peripheral smear, a trial of oral iron may be given which can be both diagnostic and therapeutic. If the Hb fails to rise within 2–3 weeks of the therapy, additional tests should be carried out to make a definitive diagnosis such as serum ferritin and hemoglobin electrophoresis to rule out thalassemia.[11]

Diagnosis of iron deficiency can be made in the presence of normal hemoglobin with reduced MCV and MCHC levels by measuring serum ferritin levels. A level of less than 30 μg/L warrants iron therapy in the dose of 30–60 mg/day.

Serum iron studies are indicated when the Hb fails to rise despite adequate iron supplementation by a compliant patient. These studies include assessment of iron stores, measurement of iron heme form and assessment of iron uptake status.

ASSESSMENT OF IRON STORES

Serum ferritin is a sensitive marker of iron deficiency in pregnancy which is stable and unaffected by recent iron intake. It represents depletion of iron stores accurately and is the first laboratory test to be altered in iron deficiency. Serum ferritin level below 15 μg/L indicates iron deficiency but treatment needs to be started when the levels fall below 30 μg/L as it indicates early depletion of iron stores and impending anemia.[12] Serum iron and total iron binding capacity (TIBC) are other indicators reflecting iron stores. TIBC measures the availability of iron binding sites on transferrin which is the carrier protein for extracellular iron. As the iron stores are depleted, the levels of serum iron decrease (<50 μg/dL) and TIBC increases (>400 μg/dL). TIBC also helps in differentiating IDA from other causes of anemia as the levels decrease with chronic infection, inflammation and cancer. Transferrin saturation is a test which measures the quantity of bound serum iron. A level of less than 20% indicates iron deficiency while high transferrin saturation reflects iron overload. Estimation of bone marrow iron is the gold standard for the diagnosis of IDA, but has limited applicability due to invasive nature of the test.

JAYPEE BROTHERS

MEASUREMENT OF IRON HEME FORM

Free erythrocyte protoporphyrin (FEP) measures the concentration of nonheme protoporphyrin in red blood cells and its levels are increased in conditions interfering with heme synthesis. It has good sensitivity but limited specificity as the levels increase in other conditions such as chronic disease.[6]

ASSESSMENT OF IRON UPTAKE STATUS

Soluble transferrin receptor (sTfR) depicts tissue iron deficiency. It is a transmembrane protein which is responsible for incorporation of circulating iron into the RBCs and is directly proportional to total transferrin levels. Its levels are increased in iron deficiency to more than 8 mg/L.[13]

Zinc protoporphyrin (ZPP) is a compound formed by incorporation of zinc into protoporphyrin instead of iron during heme biosynthesis. The levels are elevated in iron deficiency (> 40 mmol/mole heme) as well as number of other conditions such as sickle cell anemia, anemia of chronic disease and some cancers.[14] Owing to high cost involved, the above tests are rarely used in the diagnosis of IDA.

Besides the above mentioned tests, certain other tests are done in patients with anemia to evaluate the cause. These include kidney function tests, routine examination of stool, and urine to detect occult blood and infections; and chest X-ray in suspected cases of pulmonary tuberculosis.

COMPLICATIONS

Maternal

Higher incidence of preeclampsia, preterm labor and infections has been reported in pregnant women with anemia. It leads to reduced work performance and may lead to congestive cardiac failure in severe cases. Pregnant women with anemia are most prone to develop cardiac failure between 32–34 weeks, during labor and in the immediate postpartum period when the cardiac load is increased. In the postpartum period, the women may develop puerperal sepsis, subinvolution of uterus, failure of lactation and thromboembolism.[6]

Fetal

Babies born to iron deficient mothers are not usually anemic as they obtain iron from maternal transferrin trapped in placenta. However, they have decreased iron stores and are at higher risk of developing anemia later in life. Risk of prematurity,

JAYPEE BROTHERS

fetal growth restriction, and perinatal mortality is higher in these babies.[6] A recent systematic review of 6 Randomized controlled trials (RCTs) and 13 observational studies, concluded that both iron deficiency as well as its excess during pregnancy are detrimental for the mental and psychomotor development of the child and hence, the iron supplementation during pregnancy should be individualized for each woman taking into account their dietary habits and status of iron stores.[15]

PREVENTION

The prevention of iron deficiency anemia should commence before the woman embarks on pregnancy. Prompt treatment of the underlying causes as well as replenishment of iron stores is important. To tackle the problem of anemia during adolescence, a 12 by 12 initiative was launched by the Government of India in 2007. The aim of the program was to achieve target hemoglobin of at least 12g/dL for all children by the age of 12 years. The key components of the initiative were weekly prophylactic iron and folic acid supplementation, periodic deworming and health education.

India was also the first country to launch a National Nutritional Anemia Prophylaxis Program in 1970 to prevent nutritional anemia in mother and children. Under this program, all pregnant and lactating women contraceptive acceptors and children between the ages of 1–11 years are provided with iron and folic acid supplementation.

During pregnancy, all women should be screened for anemia at the first antenatal visit for early detection

Most developed countries do not recommend routine administration of iron supplementation for prophylaxis of iron deficiency. WHO recommends 30–60 mg of elemental iron and 400 μg of folic acid daily for at least 6 months during pregnancy to prevent IDA. An additional 3 months intake is recommended in the postpartum period in countries where the prevalence of anemia in pregnancy is more than 40%.

According to Government of India recommendations, 100 mg of elemental iron and 500 μg of folic acid should be given to all pregnant women for at least 100 days in the second half of pregnancy for prevention of IDA. In addition, all pregnant women should be dewormed with single dose albendazole (400mg) or 100 mg mebendazole given twice daily for 3 days to treat worm infestation.

A recent Cochrane systematic review concluded that routine daily iron supplementation reduces the risk of IDA in pregnancy but does not show any clear benefit in terms of maternal and fetal outcomes.[16] Another Cochrane review conducted in 2015 concluded that intermittent iron supplementation produces similar fetomaternal outcomes as daily supplementation. Further, it was associated with fewer side effects and had a reduced risk of high levels of Hb in mid and late

JAYPEE BROTHERS

pregnancy. Hence, it was concluded that intermittent iron supplementation may be better than daily iron supplementation in prevention of IDA, in adequately supervised antenatal women having a normal Hb.[17] Irrespective of the debate on the role of prophylactic iron supplementation, all the guidelines recommend that every antenatal woman must be given dietary advice and counseling regarding consumption of iron rich food (e.g., jaggery, green leafy vegetables, meat, fish etc.). Education regarding inhibitors of iron absorption (tannins, tea, coffee, calcium, chocolate etc.) and facilitators (proteins, ascorbic acid etc.) should be imparted to all. Interventions such as improvement in sanitation and food fortification are long term approaches in reducing the incidence of IDA at the national level.

MANAGEMENT

Antenatal

Oral iron supplementation is the first line treatment for most cases of IDA. The therapeutic dose of elemental iron for treatment of IDA is 180–200 mg per day. There are many oral iron preparations available commercially. Most preparations contain iron in the form of ferrous salts which are effective and inexpensive. Preparations containing ferric compounds have also been introduced but they are less soluble and bioavailable.

While prescribing oral iron therapy, the woman should be instructed to take the drug either empty stomach or at least 1 hour prior to meals to maximize absorption. She is advised to avoid concomitant intake of inhibitors like tannins and calcium and should be encouraged to consume it with iron enhancers like citrus fruits and juices.

The main drawbacks of oral iron supplementation are poor tolerability and gastrointestinal side effects which affect compliance. The gastrointestinal side effects associated with oral iron therapy include nausea, vomiting, constipation, abdominal cramping and diarrhea. Most of these side effects are dose related and may be alleviated by reducing the dose of oral iron or prescribing it with meals. If these interventions fail other preparations such as modified release tablets may be prescribed.

Most commonly used iron preparations are listed in table 1.[18]

Conventional Preparations

Ferrous sulfate is the most commonly used iron preparation throughout the world. It is inexpensive and rapidly absorbed but may be associated with undesirable gastrointestinal side effects. Ferrous fumarate has lesser gastrointestinal (GI) side effects and its absorption is comparable to ferrous sulfate. Ferrous ascorbate has

JAYPEE BROTHERS

Table 1: Available Preparations and Bioavailability

Preparation	Dose of salt in mg	Elemental iron in mg
Ferrous sulfate	100 mg	30 mg
Ferrous fumarate	100 mg	30 mg
Ferrous gluconate	100 mg	10 mg
Ferrous succinate	100 mg	35 mg
Ferrous ascorbate	100 mg	12 mg
Ferrous glycine sulfate	100 mg	15 mg

a distinct advantage of enhancing iron absorption and increase bioavailability of iron.

Iron Amino Acid Chelates

These are chelates of ferrous or ferric ions with amino acids namely ferrous glycine sulfate and iron polymaltose complex (IPC) which prevent binding and precipitation of iron in the gut. These ensure a high absorption even in the presence of iron inhibitors in diet. IPC is a relatively new preparation which is commercially available in United Kingdom but not in India. It has the dual benefit of fewer GI side effects and high rate of absorption even when taken with food.[1]

Modified Release Preparations

The dissolution rates of various iron salts vary, and fast dissolving tablets release the active form of free iron, rapidly leading to excessive accumulation and consequent irritation of the GI mucosa and related side effects. Modified release preparations release the active ingredient gradually over a period of time in the gut. They usually consist of an outer coating which dissolves slowly releasing the active ingredient or entrap the active ingredient in a polymeric matrix causing its release in a slow and phased manner. Thus, these preparations avoid free iron localization and GI irritation and hence, are better tolerated. However, these are more expensive and a large fraction of the drug is delivered beyond the absorbent part of the gut which is eventually excreted. It is for this reason, many regulatory bodies such as the UK National Health Service (NHS) advise against the use of modified release preparations.[19]

Choice of Oral Supplementation

There is no consensus regarding the most effective and well tolerated oral iron preparation for use in pregnant women. A RCT comparing various iron preparations

JAYPEE BROTHERS

in antenatal women, ferrous bisglycinate and ferrous ascorbate were found to be better tolerated and more effective than ferrous sulfate.[20] Another multi centric RCT, the perfect trial, concluded that ferrous fumarate is more efficacious and tolerable than carbonyl iron for the treatment of IDA in pregnancy.[21] A double blind RCT conducted in India demonstrated comparable rise in hemoglobin in pregnant women treated with low doses of ferrous fumarate versus ferrous sulfate.[22] Better safety profile and high absorption rate of IPC compared to ferrous sulfate has been reported in some studies.[23]

An innovative study conducted in 2013 in UK compared iron absorption rates for various solid and liquid iron preparations using in vitro model of human intestine using human intestinal Caco-2 cells. The various supplements compared were ferrous sulfate, ferrous gluconate, sustained release tablet of ferrous fumarate plus minerals, and controlled released tablet of ferrous sulfate plus ascorbic acid. The study revealed that iron absorption was maximum from ferrous sulfate conventional release tablets and lowest for sustained and controlled release tablets containing minerals and vitamins.[24]

In the absence of available evidence regarding the best oral iron preparation during pregnancy, the treating physician has to individualize the treatment which is efficacious, well tolerated and affordable. The authors are of the opinion that conventional preparations like ferrous sulfate, ferrous fumarate and ferrous gluconate should be the first choice as they are cost effective and have high bioavailability.

Parenteral Therapy

Although, the preferred route of administration of iron is oral, parenteral therapy is indicated in certain cases.

Advantages of parenteral therapy are the certainty of iron administration and rapid restoration of iron stores. However, the disadvantages include high cost and severe anaphylactic reactions. Administration by intramuscular route can result in pain at injection site, abscess formation, skin staining, arthralgia, fever and painful lymphadenopathy but is cost effective. Newer intravenous parenteral preparations are safer in pregnancy and lactation. Intravenous parenteral iron use may sometimes cause self limiting nonallergic infusion reactions such as urticaria, palpitations, dizziness and neck and back spasms. These reactions are rare and usually not serious. Severe anaphylactic reactions with intravenous iron are extremely rare.

The diagnosis of IDA should be confirmed and the total dose needs to be calculated carefully before starting parenteral therapy by the following formulae.

- Weight of patient in kg x (normal Hb– patient's Hb in g/dL) x 2.21 + 1000 mg for stores

JAYPEE BROTHERS

- Weight of patient in pounds x Hb deficit in percentage x 0.3. Add 50% of the calculated dose for stores
- Hemoglobin deficit is calculated and total dose is derived by giving 200 mg of elemental iron for every 1 gm desired rise in Hb.

The indications and contraindications of parenteral iron therapy are given in table 2.

Oral iron supplementation should also be stopped one day prior to start of parenteral therapy to avoid toxic reactions. It is also recommended that parenteral iron preparations should only be administered at a health facility where adequate monitoring and facilities for resuscitation are available. The woman should be observed for at least 30 minutes after administration of the drug to look for any evidence of anaphylactic reactions.

The route of administration of parenteral iron can be intramuscular or intravenous. Different preparations with their elemental iron content and route of administration are given in table 3.

High molecular weight iron dextran and iron sorbitol citric acid have long been the most commonly used parenteral agents. However, frequent side effects and the threat of unpredictable anaphylactic reactions with these preparations have replaced them with newer safer options.

Iron sucrose is the most extensively researched intravenous iron preparation for use in pregnancy and has been found to have an excellent safety profile with good efficacy. The rate of adverse drug reaction (ADR) is 0.5–1.5% and anaphylaxis less than 0.1%. It can be administered either as an intravenous infusion with 200 mg (4 mL) diluted in 200 mL of normal saline given over 15-20 minutes or by slow intravenous injection of undiluted dose of 200 mg (4 mL) given at the rate of 20 mg/min. The total dose given should not exceed 600 mg per week.

Iron carboxymaltose or ferric carboxymaltose (FCM) is usually given in the dose of 20 mg/kg body wt as intravenous infusion. For preparing intravenous

Table 2: Indications and Contraindications of Parenteral Iron Therapy

Indications	Contraindications
• Noncompliance	• First trimester of pregnancy
• Poor tolerability due to side effects of oral iron	• History of anaphylactic reaction to parenteral iron therapy
• Malabsorption syndromes leading to poor iron absorption from gut	• Iron overload
• Women needing rapid repletion of iron stores such as those with moderate to severe anemia near term	• Presence of chronic liver disease or active infection
• Women on recombinant erythropoietin	• Asthma
	• Acute renal failure
	• Active rheumatoid arthritis

JAYPEE BROTHERS

Table 3: Various Available Parenteral Iron Preparations[18]

Preparation	FDA category	Strength	Route of administration
Iron dextran complex	C	2 mL ampoule 50 mg/mL 10 mL multi dose vial	IM or IV
Iron sorbitol citric acid complex	B	1.5 mL ampoule 50 mg/mL	IM
Iron sucrose	B	5 mL ampoule 20 mg/mL	IV
Iron gluconate	N	5 mL ampoule 12.5 mg/mL	IV
Iron carboxymaltose	C	2 mL and 5 mL vials 50 mg/mL	IV

infusion, the concentration should not be less than 2 mg iron/mL, and usually a 1,000 mg FCM infusion is prepared in 250 mL of normal saline and given over 15 minutes. The total dose should not exceed 1000 mg of iron per week. It is an extremely effective therapy for management of postpartum IDA, and many studies have validated the safety and efficacy of FCM in pregnancy as well.[25]

Low molecular weight iron dextran (LMW ID) is one of the inexpensive options which is safe for use in pregnancy and can be given intravenously in multiple doses (100 mg/day) or administered in a single total dose infusion, up to a maximum of 1,000 mg. Treatment with LMW ID has shown to be highly efficacious, safe and cost effective in resolving IDA in pregnancy in a recent study conducted in 2016.[26]

Other intravenous preparations such as iron isomaltoside, ferric gluconate and ferumoxytol have been used for treatment of IDA in pregnancy with good results. However, due to the dearth of sufficient evidence to support their use in pregnancy, their routine use in pregnancy is not justified.

The decision to choose between oral and intravenous iron depends on a number of factors including the severity of anemia, period of gestation, ability of patient to tolerate oral iron, patient compliance, and lastly cost and availability of iron preparation.

Oral iron is the undisputed first choice for treatment of IDA in pregnancy because it is safe, inexpensive, efficacious and readily available. However, GI side effects are reported in more than 50% of patients consuming oral iron thus, significantly affecting patient acceptability and compliance. Parenteral iron preparations are prescribed when even dose modification fails to relieve the side effects. Many studies have shown that intravenous parenteral preparations treat IDA, and replenish iron stores faster and more effectively than oral preparations,

JAYPEE BROTHERS

without any serious adverse effects.[27] Studies comparing intramuscular and intravenous preparations have shown similar efficacy on pregnancy but with higher rate of adverse reactions in the intramuscular group.[28] A Cochrane review conducted in 2011 regarding treatment for IDA in pregnancy concluded that there is insufficient evidence to say when and how anemia in pregnancy should be treated.[29] In selected cases of IDA blood transfusion is indicated. This includes pregnant women with severe anemia beyond 36 weeks of gestation, anemia with presence of infections or heart disease to replace antepartum and postpartum hemorrhage and nonresponders to oral or parenteral therapy.

Packed red cell transfusion is preferred to whole blood transfusion to avoid volume overload. Each unit of packed cell transfusion should raise the HB by 1 g/dL. Major drawbacks of blood transfusion are fluid overload leading to congestive heart failure, transfusion reactions, precipitation of preterm labor and rarely life threatening condition like transfusion related acute lung injury (TRALI).

Management of Labor

Women with mild to moderate anemia are usually managed in the same way as nonanemic women with special focus on prevention of infection and minimization of blood loss. Women with severe anemia are at high risk of developing complications such as congestive heart failure during labor. Adequate pain relief should be given in the first stage and the woman is nursed in a propped up position with administration of intermittent oxygen by mask. Antibiotic prophylaxis may be given as these women are prone to infections. Adequate blood should be arranged and intravenous access established. However, intravenous fluids are restricted and the woman is encouraged to take oral fluids to maintain hydration. Digitalization may be needed if the patient is at a very high risk of developing congestive cardiac failure. Maternal vitals are strictly monitored to detect early signs of cardiac decompensation. The second stage should be cut short by application of outlet forceps or vacuum if there is a tendency for prolongation. Active management of third stage of labor is done and any blood loss should be promptly replaced by packed cell transfusion. Diuretics in the form of 40 mg furosemide may be given intravenously immediately after delivery to reduce cardiac load. Finally, the woman should be observed in the immediate post natal period carefully for any evidence of decompensation.

Management in Postpartum Period

Women with severe IDA during pregnancy are predisposed to complications like subinvolution of uterus, congestive cardiac failure, thromboembolism, puerperal sepsis and lactation failure in the postpartum period. So a careful watch should

JAYPEE BROTHERS

Available at all medical bookstores
or buy online at www.jaypeebrothers.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers

ISBN 978-93-5270-393-7