RESEARCH PAPER

Anemia During Pregnancy: An Comprehensive Review

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Abstract:

Anemia during pregnancy remains a significant global public health concern and a major contributor to maternal and perinatal morbidity, particularly in low- and middle-income countries. Despite advances in antenatal care, its prevalence remains high because of nutritional deficiencies, physiological changes of pregnancy, infectious diseases, hereditary disorders, chronic illnesses, and socioeconomic inequalities. Iron deficiency is the leading cause, although folate and vitamin B_{12} deficiencies, hemoglobinopathies, and inflammatory conditions also contribute substantially. Maternal anemia is associated with increased risks of fatigue, infections, postpartum hemorrhage, hypertensive disorders, cardiac complications, and maternal mortality, while fetal consequences include preterm birth, low birth weight, fetal growth restriction, neonatal anemia, and impaired neurodevelopment. Early diagnosis using appropriate laboratory investigations, followed by individualized nutritional and pharmacological management, is essential to improve pregnancy outcomes. Recent advances in iron metabolism, particularly the understanding of hepcidin and ferritin, along with newer intravenous iron formulations, have enhanced diagnostic accuracy and treatment strategies. This review summarizes recent evidence on the epidemiology, pathophysiology, diagnosis, complications, management, prevention, and emerging advances in anemia during pregnancy to support evidence-based clinical practice.[1–6]

Keywords: Anemia; Pregnancy; Iron deficiency anemia; Maternal health; Ferritin; Hepcidin; Iron supplementation; Obstetric complications.

How to cite this article: Latha.P1 Prof.Dr.Nar Singh Malav2. Anemia During Pregnancy: An Comprehensive Review. Int J Drug Deliv Technol. 2026;16(80s):1641-1654. DOI: 10.25258/ijddt.16.80s.204.

Introduction

Anemia during pregnancy remains one of the most prevalent medical disorders encountered in obstetric practice and continues to represent a significant challenge for maternal and neonatal healthcare worldwide. Although considerable progress has been achieved in antenatal screening, nutritional supplementation, and public health interventions over the past several decades, anemia continues to affect a substantial proportion of pregnant women, particularly in resource-limited settings where nutritional deficiencies, recurrent infections, limited healthcare access, and socioeconomic disparities frequently coexist. Consequently, maternal anemia is increasingly recognized not merely as an isolated hematological abnormality but as a multifactorial condition reflecting the interaction between biological, nutritional, environmental, and social determinants of health.[1–4]Pregnancy is characterized by profound physiological adaptations designed to support fetal growth and prepare the maternal body for childbirth. Expansion of plasma volume, increased erythropoiesis, enhanced placental blood flow, and rising metabolic demands collectively increase maternal requirements for iron and other micronutrients. These normal physiological changes frequently reduce hemoglobin concentration through dilutional effects, making the distinction between physiological adaptation and pathological anemia particularly challenging. Women entering pregnancy with inadequate nutritional reserves or chronic medical conditions are therefore at substantially greater risk of developing clinically significant anemia as gestation progresses.[2–5]

Iron deficiency remains the principal cause of anemia during pregnancy and is estimated to account for approximately half of all cases worldwide. Nevertheless, contemporary research has demonstrated that maternal anemia is frequently multifactorial, with deficiencies of folate and vitamin B_{12}, chronic inflammatory disorders, hereditary hemoglobinopathies, infectious diseases, renal disorders, and autoimmune conditions contributing significantly to disease development. The relative contribution of these factors varies according to geographical location, socioeconomic status, dietary habits, healthcare accessibility, and the prevalence of endemic infectious diseases. This diversity emphasizes the importance of comprehensive clinical assessment rather than empirical treatment based solely on hemoglobin concentration.[3–6]The consequences of maternal anemia extend well beyond hematological abnormalities. Persistent reductions in oxygen-carrying capacity impair maternal functional status and may compromise placental perfusion, thereby increasing the risk of adverse pregnancy outcomes. Moderate to severe anemia has been associated with fatigue, reduced physical performance, cardiovascular strain, postpartum hemorrhage, infections, prolonged hospitalization, and increased maternal mortality. Simultaneously, fetal complications include preterm birth, low birth weight, intrauterine growth restriction, neonatal anemia, impaired neurodevelopment, and increased perinatal mortality. The severity of these complications depends on the underlying etiology, degree of anemia, gestational age at onset, and the timeliness of therapeutic intervention.[1–6]

Over the past decade, significant advances have transformed the understanding and management of anemia during pregnancy. Improved knowledge of iron metabolism, particularly the regulatory role of hepcidin, has enhanced the interpretation of iron biomarkers and facilitated differentiation between absolute and functional iron deficiency. In parallel, the introduction of safer intravenous iron preparations, improved laboratory diagnostics, and updated international guidelines has shifted clinical practice toward individualized management strategies based on etiology rather than uniform supplementation. Such developments highlight the importance of integrating contemporary evidence into routine obstetric care.

This review critically evaluates recent evidence concerning the epidemiology, physiological adaptations, etiological factors, pathophysiological mechanisms, classification, diagnosis, clinical manifestations, complications, management, nutritional interventions, preventive strategies, and emerging therapeutic advances in anemia during pregnancy. By synthesizing current literature and international recommendations, the review aims to provide an evidence-based resource for clinicians, researchers, and healthcare professionals involved in maternal health.

Global public health concern

Anemia during pregnancy remains a major global public health concern, affecting maternal and neonatal health worldwide. Despite improvements in antenatal care and nutritional supplementation, approximately 37% of pregnant women (around 32 million) are affected globally, with the highest burden observed in low- and middle-income countries.[1–3] Nutritional deficiencies, infectious diseases, limited healthcare access, and socioeconomic inequalities continue to be the primary determinants of its high prevalence.

The greatest burden is reported in the WHO African and South-East Asia Regions, where iron deficiency commonly coexists with malaria, parasitic infestations, poor dietary intake, and frequent pregnancies. Although prevalence is comparatively lowerin high-income countries due to better nutrition and healthcare services, vulnerable populations remain at increased risk. India continues to contribute substantially to the global burden despite national iron–folic acid supplementation and maternal health programs, with rural and socioeconomically disadvantaged women being disproportionately affected.Iron deficiency remains the leading cause of maternal anemia; however, folate and vitamin B_{12} deficiencies, chronic inflammatory disorders, inherited hemoglobinopathies, infections, and recurrent blood loss also contribute significantly. The condition not only increases maternal and neonatal morbidity but also imposes considerable socioeconomic and healthcare burdens. Therefore, strengthening antenatal screening, improving maternal nutrition, and addressing broader social determinants of health remain essential strategies for reducing the global burden of anemia during pregnancy.[1–6]

Physiological Changes in Pregnancy and Their Relationship with Anemia

Pregnancy is associated with significant physiological adaptations that support fetal growth and meet the increased metabolic demands of gestation. Hematological changes are among the most important adaptations, as they directly influence oxygen transport and nutrient delivery. However, these normal physiological changes also alter hematological parameters, making it challenging to distinguish physiological adaptations from pathological anemia.[1–4]

One of the major hematological changes during pregnancy is the expansion of maternal blood volume. Plasma volume increases by approximately 40–50%, whereas red blood cell (RBC) mass increases by only 20–30%, resulting in a physiological decline in hemoglobin and hematocrit due to hemodilution. This phenomenon, known as the physiological anemia of pregnancy, is a normal adaptive response that improves uteroplacental perfusion, reduces blood viscosity, and provides a circulatory reserve against blood loss during delivery. Therefore, a mild reduction in hemoglobin concentration should be interpreted cautiously and differentiated from pathological anemia.[2–5]

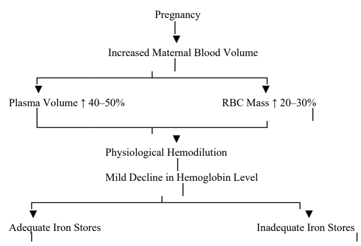
Pregnancy also stimulates erythropoiesis through increased secretion of erythropoietin (EPO), promoting expansion of maternal RBC mass. Effective erythropoiesis, however, depends on adequate iron, folate, vitamin B_{12}, and other micronutrients. Deficiencies of these nutrients impair hemoglobin synthesis and predispose women to nutritional anemia. Total maternal iron requirements increase to nearly 1,000 mg during pregnancy, with iron required for maternal erythropoiesis, fetal growth, placental development, and compensation for physiological losses. Women entering pregnancy with depleted iron stores are therefore at greater risk of developing iron deficiency anemia.[2–6]

Recent advances have identified hepcidin as the key regulator of iron metabolism during pregnancy. Physiological suppression of hepcidin enhances intestinal iron absorption and mobilization of stored iron to meet increasing maternal and fetal demands. In contrast, chronic inflammation, infections, obesity, and autoimmune disorders increase hepcidin production, reducing iron absorption and causing functional iron deficiency, in which iron stores are adequate but unavailable for erythropoiesis. This mechanism partly explains the poor response to oral iron therapy observed in some pregnant women.[3–6]

The placenta plays a crucial role in maternal-fetal iron transfer through transferrin receptors and ferroportin-mediated transport. Although fetal iron requirements are preferentially maintained during mild maternal deficiency, prolonged or severe anemia may impair placental iron transfer, increasing the risk of fetal iron deficiency, intrauterine growth restriction, neonatal anemia, and impaired neurodevelopment. Pregnancy is also associated with increased cardiac output and oxygen consumption, and severe anemia may overwhelm these compensatory mechanisms, resulting in cardiovascular stress and placental insufficiency.[2–6]

Overall, understanding these physiological adaptations is essential for accurate interpretation of hematological investigations during pregnancy. Clinical assessment should integrate gestational age, hemoglobin concentration, red cell indices, serum ferritin, and inflammatory markers to distinguish physiological hemodilution from pathological anemia and facilitate timely, individualized management.[1–6]

Fig-1: Physiological hematological adaptations during pregnancy and progression to anemia



Maternal and Fetal Complications

Etiology and Risk Factors

Anemia during pregnancy is a multifactorial condition resulting from the interaction of nutritional, physiological, infectious, genetic, and socioeconomic factors. Although iron deficiency remains the leading cause globally, accounting for nearly half of all cases, deficiencies of folate and vitamin B_{12}, chronic inflammatory disorders, inherited hemoglobinopathies, infectious diseases such as malaria and hookworm infestation, chronic kidney disease, and recurrent blood loss also contribute significantly to its development. Pregnancy itself increases the demand for iron and other micronutrients, making women with inadequate nutritional reserves particularly susceptible to anemia. Inflammatory conditions may further impair iron utilization through hepcidin-mediated functional iron deficiency, reducing the response to oral iron therapy despite adequate iron stores. Maternal factors including adolescent pregnancy, multiparity, multiple gestation, short interpregnancy intervals, chronic illnesses, gastrointestinal disorders, and previous obstetric blood loss increase the risk of anemia. Additionally, poverty, food insecurity, poor dietary diversity, limited education, delayed antenatal care, and inadequate adherence to supplementation remain important socioeconomic determinants, particularly in low- and middle-income countries. As multiple etiological factors often coexist, accurate diagnosis requires a comprehensive evaluation of nutritional status, hematological parameters, and underlying medical conditions to facilitate individualized management and improve maternal and fetal outcomes.[1–6]

Table 2. Major etiological factors and associated risk factors for anemia during pregnancy

Etiological factor	Pathophysiological mechanism	Major risk factors
Iron deficiency	Depletion of iron stores leading to impaired hemoglobin synthesis	Poor dietary intake, recurrent pregnancies, chronic blood loss, poor compliance with supplementation
Folate deficiency	Defective DNA synthesis and ineffective erythropoiesis	Inadequate dietary intake, increased fetal demand, malabsorption
Vitamin B_{12} deficiency	Impaired erythrocyte maturation and neurological dysfunction	Vegetarian diet, pernicious anemia, gastrointestinal disorders
Anemia of inflammation	Hepcidin-mediated reduction in iron availability	Chronic infections, autoimmune diseases, obesity, chronic kidney disease
Hemoglobinopathies	Abnormal globin synthesis or erythrocyte destruction	Family history, endemic geographical regions
Infectious diseases	Hemolysis, chronic blood loss, bone marrow suppression	Malaria, hookworm infestation, HIV, tuberculosis
Socioeconomic factors	Poor nutrition and inadequate healthcare access	Poverty, food insecurity, low educational status, delayed antenatal care

Pathophysiology

The pathophysiology of anemia during pregnancy is a complex process involving increased physiological demands, altered iron homeostasis, nutritional deficiencies, inflammation, and impaired erythropoiesis. Pregnancy requires a substantial increase in iron to support maternal erythrocyte expansion, placental development, fetal growth, and blood loss during delivery. When maternal iron intake or stores are inadequate, iron depletion progresses from reduced iron reserves to impaired hemoglobin synthesis and ultimately iron deficiency anemia. A key regulator of this process is hepcidin, the principal hormone controlling systemic iron metabolism. During normal pregnancy, physiological suppression of hepcidin enhances intestinal iron absorption and mobilization of stored iron to meet increasing maternal and fetal requirements. However, chronic inflammatory conditions, infections, obesity, and autoimmune disorders increase hepcidin production, reducing ferroportin-mediated iron release and resulting in functional iron deficiency, where iron stores are adequate but unavailable for effective erythropoiesis. This mechanism explains the poor response to oral iron therapy observed in some pregnant women despite normal or elevated serum ferritin levels.[1–6]

Reduced hemoglobin concentration ultimately compromises oxygen transport, leading to maternal tissue hypoxia and placental dysfunction. Although compensatory mechanisms such as increased erythropoietin secretion, cardiac output, and oxygen extraction initially maintain tissue perfusion, persistent anemia overwhelms these adaptations and contributes to endothelial dysfunction, oxidative stress, impaired placental angiogenesis, and reduced uteroplacental blood flow. Consequently, chronic anemia increases the risk of fetal growth restriction, preterm birth, low birth weight, neonatal anemia, and maternal cardiovascular complications. Emerging evidence also suggests that oxidative stress and inflammatory cytokines further suppress erythropoiesis, creating a vicious cycle that aggravates anemia. These findings emphasize that evaluation of anemia during pregnancy should extend beyond hemoglobin concentration alone and incorporate assessment of iron biomarkers, nutritional status, inflammatory conditions, and underlying medical disorders to facilitate individualized diagnosis and management.[1–6]

Figure 2. Patho-physiological mechanisms involved in anemia during pregnancy

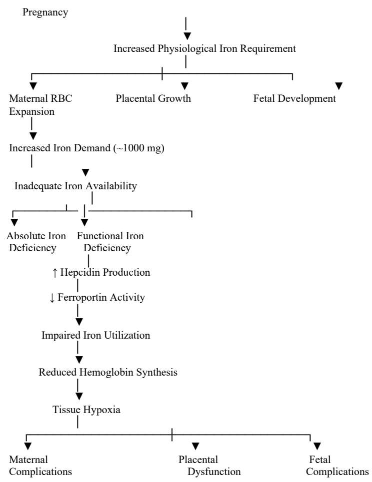


Figure 2.*Schematic representation of the major mechanisms contributing to anemia during pregnancy. Both absolute iron deficiency and inflammation-mediated functional iron deficiency impair hemoglobin synthesis, leading to tissue hypoxia and adverse maternal and fetal outcomes.*

Critical Insight

One aspect that distinguishes recent research from traditional understanding is the growing recognition that not all anemia in pregnancy is caused by depleted iron stores. The identification of hepcidin as a key regulator of iron metabolism has shifted clinical practice toward differentiating absolute iron deficiency from functional iron deficiency, particularly in women with obesity, chronic inflammation, or autoimmune disorders. This distinction has important therapeutic implications because women with functional iron deficiency may respond poorly to oral iron supplementation despite normal ferritin concentrations, highlighting the need for individualized diagnostic evaluation and treatment strategies.[3–6]

Classification and Diagnostic Criteria

Accurate classification and timely diagnosis of anemia during pregnancy are fundamental to reducing maternal and fetal morbidity. Although hemoglobin concentration remains the primary screening parameter, contemporary evidence emphasizes that anemia should be evaluated as a clinical syndrome rather than a laboratory abnormality alone. Pregnancy is associated with physiological hemodilution resulting from disproportionate plasma volume expansion, making interpretation of hematological indices more complex than in non-pregnant women. Consequently, current international guidelines recommend integrating gestational age, clinical findings, red blood cell indices, and biochemical markers of iron status to distinguish physiological adaptation from pathological anemia and to identify the underlying etiology.[1–6]

The World Health Organization (WHO) defines anemia during pregnancy as a hemoglobin concentration below 11.0 g/dL, irrespective of gestational age. This threshold has been widely adopted because it provides a standardized approach for epidemiological surveillance and public health reporting. However, physiological plasma volume expansion reaches its maximum during the second trimester, resulting in a modest decline in hemoglobin concentration even among healthy pregnant women. Recognizing this normal physiological adaptation, organizations such as the American College of Obstetricians and Gynecologists (ACOG), the British Society for Haematology (BSH), and several national obstetric guidelines recommend trimester-specific diagnostic thresholds to minimize overdiagnosis of physiological hemodilution. According to these recommendations, anemia is generally defined as hemoglobin <11.0 g/dL during the first and third trimesters and <10.5 g/dL during the second trimester.[2–5] Although these differences appear small, they have important clinical implications because inappropriate labeling of physiological hemodilution as pathological anemia may result in unnecessary investigations or treatment.

Once anemia has been identified, classification according to severity becomes essential because maternal and fetal risks increase progressively as hemoglobin concentration declines. Mild anemia is frequently asymptomatic and is often detected during routine antenatal screening, whereas moderate and severe anemia are associated with a substantially greater likelihood of cardiovascular stress, postpartum hemorrhage, fetal growth restriction, preterm birth, and maternal mortality. Classification based on severity therefore guides therapeutic decisions, including the choice between oral iron supplementation, intravenous iron therapy, hospitalization, or blood transfusion. Moreover, severity assessment facilitates risk stratification during antenatal follow-up and enables timely referral of women requiring specialized obstetric care.

Table-3. Classification of anemia during pregnancy according to hemoglobin concentration

Classification	Hemoglobin (g/dL)	Clinical implication
Normal	≥11.0	Normal hematological status
Mild anemia	10.0–10.9	Usually asymptomatic or mildly symptomatic; requires evaluation and iron replacement where indicated
Moderate anemia	7.0–9.9	Increased maternal and fetal risk; active therapeutic intervention required
Severe anemia	<7.0	High-risk pregnancy requiring urgent evaluation and intensive management
Very severe anemia*	<4.0	Medical emergency with life-threatening maternal and fetal consequences

*Recognized in several national guidelines for emergency clinical management.

Although hemoglobin concentration establishes the presence of anemia, it provides limited information regarding its underlying cause. Consequently, evaluation of red blood cell indices obtained from the complete blood count (CBC) represents the next step in diagnostic assessment. The mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) assist in categorizing anemia into microcytic, normocytic, or macrocytic patterns, thereby narrowing the differential diagnosis and guiding subsequent laboratory investigations. While this morphological classification remains clinically valuable, it should always be interpreted alongside biochemical markers because mixed nutritional deficiencies may obscure classical hematological patterns.

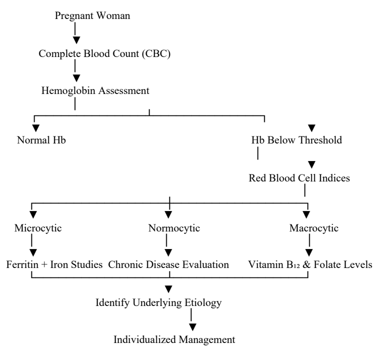
Table 4. Morphological classification of anemia during pregnancy

Morphological type	Mean corpuscular volume (MCV)	Common causes
Microcytic	<80 fL	Iron deficiency anemia, thalassemia, chronic blood loss
Normocytic	80–100 fL	Acute hemorrhage, anemia of inflammation, chronic kidney disease, mixed deficiencies
Macrocytic	>100 fL	Folate deficiency, vitamin B_{12} deficiency, liver disease, hypothyroidism

Among laboratory investigations, serum ferritin is considered the most reliable indicator of iron stores and is recommended for the early diagnosis of iron deficiency during pregnancy. A serum ferritin level <30 µg/L is generally suggestive of iron deficiency in the absence of inflammation. However, because ferritin is an acute-phase reactant, its levels may be elevated in inflammatory or infectious conditions, potentially masking iron deficiency. Therefore, additional biomarkers such as transferrin saturation (TSAT), soluble transferrin receptor (sTfR), reticulocyte hemoglobin content, and hepcidin may improve diagnostic accuracy, particularly in complex cases, although their routine clinical use remains limited.[1–6]

Peripheral blood smear examination and reticulocyte count provide valuable information regarding red cell morphology and bone marrow response, while targeted investigations, including serum folate, vitamin B_{12}, hemoglobin electrophoresis, and evaluation for infections or chronic diseases, should be performed when clinically indicated. Overall, diagnosis should integrate hemoglobin concentration with iron biomarkers, red blood cell indices, inflammatory status, and clinical findings to accurately identify the underlying etiology and guide individualized management.[1–6]

Figure 3. Diagnostic algorithm for anemia during pregnancy



Clinical Manifestations

The clinical manifestations of anemia during pregnancy vary according to its severity, underlying cause, rate of onset, and maternal physiological reserve. Women with mild anemia are often asymptomatic because gradual reductions in hemoglobin are partially compensated by physiological adaptations. As anemia progresses, impaired oxygen delivery leads to symptoms such as fatigue, generalized weakness, reduced exercise tolerance, dyspnea on exertion, palpitations, dizziness, and diminished physical performance. On clinical examination, pallor of the conjunctivae, nail beds, oral mucosa, and palmar creases remains the most common finding, while severe anemia may present with tachycardia, tachypnea, systolic flow murmurs, orthostatic hypotension, or signs of cardiac decompensation.[1–6]

Certain clinical features may suggest specific etiologies. Iron deficiency is commonly associated with pica, brittle nails, hair loss, glossitis, and angular cheilitis, whereas folate and vitamin B_{12} deficiencies may present with macrocytic anemia, glossitis, and neurological manifestations such as paresthesia and gait disturbances. Persistent maternal anemia may also compromise fetal well-being, leading to reduced fetal movements, placental insufficiency, fetal growth restriction, and abnormal fetal surveillance findings. However, because many symptoms overlap with normal physiological changes of pregnancy, clinical findings should always be interpreted alongside laboratory investigations for accurate diagnosis and timely management.[1–6]

Table 5. Clinical manifestations of anemia during pregnancy according to severity

Severity	Common maternal manifestations	Potential fetal manifestations
Mild anemia	Often asymptomatic, mild fatigue, reduced exercise tolerance	Usually none; fetal growth generally preserved
Moderate anemia	Marked fatigue, weakness, dyspnea on exertion, dizziness, palpitations, pallor	Reduced fetal growth, decreased fetal movements, increased risk of preterm birth
Severe anemia	Resting dyspnea, tachycardia, syncope, chest discomfort, heart failure, marked pallor	Fetal growth restriction, placental insufficiency, fetal distress, low birth weight, increased perinatal mortality

Clinical Pearl

An important clinical consideration is that symptom severity does not always correlate directly with hemoglobin concentration. Women with slowly developing chronic anemia may remain relatively asymptomatic despite markedly reduced hemoglobin levels because of gradual cardiovascular adaptation, whereas acute anemia resulting from hemorrhage may produce profound symptoms even at comparatively higher hemoglobin concentrations. Consequently, treatment decisions should be based on the overall clinical condition, laboratory findings, gestational age, and underlying etiology rather than hemoglobin concentration alone.[3–6]

Maternal and Fetal Complications

Maternal anemia is associated with a broad spectrum of adverse maternal, fetal, and neonatal outcomes, with the risk increasing according to the severity, duration, and underlying cause of anemia. Chronic reductions in hemoglobin impair oxygen transport and placental perfusion, leading to tissue hypoxia and compromised maternal and fetal health. Consequently, anemia is now recognized not only as a hematological disorder but also as an independent predictor of adverse pregnancy outcomes.[1–6]

Maternal complications include fatigue, reduced physical capacity, impaired immunity, increased susceptibility to infections, postpartum hemorrhage, prolonged hospitalization, and, in severe cases, congestive cardiac failure, pulmonary edema, and maternal mortality. Chronic anemia is also associated with hypertensive disorders, placental abruption, prolonged labor, increased operative delivery, and greater need for blood transfusion, particularly when complicated by obstetric hemorrhage. Early correction of maternal anemia has been shown to improve maternal outcomes and reduce peripartum complications.[2–6]

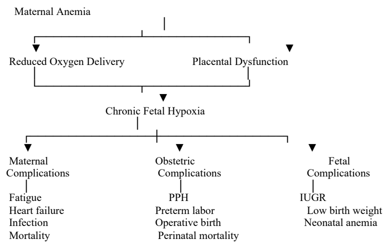
For the fetus, impaired maternal oxygen delivery and placental dysfunction increase the risk of intrauterine growth restriction (IUGR), low birth weight, preterm birth, fetal distress, neonatal anemia, and perinatal mortality. Severe or prolonged maternal iron deficiency may also reduce fetal iron stores and adversely affect neurodevelopment, resulting in long-term cognitive and behavioral impairment. These complications highlight the importance of early antenatal screening, timely diagnosis, and individualized management to optimize both maternal and neonatal outcomes.[1–6]

Table 6. Major maternal and fetal complications associated with anemia during pregnancy

Maternal complications	Fetal and neonatal complications
Fatigue and reduced physical capacity	Intrauterine growth restriction (IUGR)
Reduced work productivity	Low birth weight
Increased susceptibility to infections	Preterm birth
Postpartum hemorrhage	Neonatal anemia
Congestive cardiac failure (severe anemia)	Fetal distress
Prolonged hospitalization	Placental insufficiency
Increased blood transfusion requirement	Impaired neurodevelopment
Maternal mortality (severe untreated cases)	Stillbirth and increased perinatal mortality

Table 6.*Major maternal, fetal, and neonatal complications associated with anemia during pregnancy. The likelihood and severity of these outcomes increase with worsening anemia and delayed therapeutic intervention.*

Figure 4. Relationship between maternal anemia and adverse pregnancy outcomes



Critical Perspective

Recent systematic reviews suggest that the timing of anemia may be as important as its severity. Anemia developing during the first trimester appears to have a stronger association with abnormal placental development and fetal growth restriction, whereas anemia identified during the third trimester more commonly influences maternal tolerance to labor, postpartum hemorrhage, and neonatal iron status. These observations support the growing emphasis on preconception optimization of iron stores and first-trimester screening, rather than relying solely on treatment initiated later in pregnancy.[1–6]

Management Strategies

The management of anemia during pregnancy aims to restore hemoglobin concentration, replenish iron stores, improve maternal well-being, and optimize fetal outcomes. Effective treatment requires identification of the underlying etiology, assessment of disease severity, gestational age, and associated maternal comorbidities. Contemporary guidelines advocate an individualized approach based on laboratory findings and clinical presentation rather than empirical iron supplementation alone. Initial evaluation includes a complete blood count, assessment of iron status, and investigation for nutritional deficiencies or underlying medical disorders.[1–6]

Oral iron supplementation remains the first-line treatment for uncomplicated iron deficiency anemia. Ferrous sulfate, ferrous fumarate, and ferrous gluconate are commonly prescribed formulations. The WHO recommends 30–60 mg of elemental iron with 400 µg of folic acid daily for prevention during pregnancy, while higher therapeutic doses are used for confirmed iron deficiency anemia. Gastrointestinal adverse effects such as nausea, constipation, and abdominal discomfort may reduce adherence, although recent evidence suggests that alternate-day dosing may improve tolerability in selected patients.[2–6]

Intravenous iron therapy is recommended for women with moderate-to-severe anemia, intolerance or poor response to oral iron, malabsorption, or late gestation when rapid correction is required. Newer preparations, particularly ferric carboxymaltose, provide faster replenishment of iron stores with fewer treatment sessions than iron sucrose, although the choice of preparation should consider safety, cost, and local availability. Blood transfusion is reserved for severe symptomatic anemia, acute hemorrhage, or hemodynamic instability and should be considered a life-saving intervention rather than routine therapy.[1–6]

Management of non-iron deficiency anemia should target the underlying cause. Folate and vitamin B_{12} deficiencies require appropriate supplementation, while inflammatory anemia, chronic diseases, and hemoglobinopathies necessitate disease-specific management. Treatment response should be monitored by reassessing hemoglobin after 2–4 weeks, with serum ferritin used to confirm restoration of iron stores. Failure to respond should prompt evaluation of treatment adherence, persistent blood loss, malabsorption, ongoing inflammation, or an alternative diagnosis. Early diagnosis and timely individualized therapy remain essential for reducing maternal and fetal complications associated with anemia during pregnancy.[1–6]

Table 7. Comparison of therapeutic approaches for anemia during pregnancy

Treatment	Primary indication	Advantages	Limitations
Oral iron	Mild iron deficiency anemia	Inexpensive, widely available, effective in uncomplicated cases	Gastrointestinal adverse effects, slower response, poor compliance
Intravenous iron sucrose	Moderate anemia, intolerance to oral iron	Good safety profile, effective replenishment	Multiple infusions required
Ferric carboxymaltose	Moderate-to-severe anemia, late pregnancy	Rapid correction, larger single doses, fewer visits	Higher cost
Blood transfusion	Severe symptomatic anemia, hemorrhage, hemodynamic instability	Immediate improvement in oxygen-carrying capacity	Transfusion-related risks; not first-line therapy
Folate/Vitamin B_{12} supplementation	Confirmed deficiency states	Corrects underlying nutritional deficiency	Ineffective in isolated iron deficiency

Critical Perspective

Recent evidence has shifted clinical emphasis from simply treating anemia to preventing iron deficiency before anemia develops. Early identification of depleted iron stores through ferritin assessment, individualized use of intravenous iron in appropriate patients, and optimization of maternal iron status before delivery are increasingly regarded as more effective strategies than correcting severe anemia during late pregnancy. This proactive approach is reflected in recent WHO and FIGO recommendations and represents one of the most important developments in contemporary obstetric hematology.

Nutritional Management and Dietary Recommendations

Nutritional management is an essential component of both the prevention and treatment of anemia during pregnancy and should complement pharmacological therapy. Pregnancy increases the requirements for iron, folate, vitamin B_{12}, protein, and other micronutrients necessary for erythropoiesis, placental development, and fetal growth. Although dietary modification alone is insufficient to correct moderate or severe anemia, it enhances the effectiveness of iron supplementation and helps restore maternal iron stores.[1–6]

Iron-rich foods, particularly those with high bioavailability, should be encouraged. Heme iron from animal sources is absorbed more efficiently than non-heme iron from plant-based foods. For vegetarian women, iron-rich foods such as green leafy vegetables, legumes, soy products, whole grains, and fortified cereals should be combined with vitamin C-rich foods to enhance iron absorption. Conversely, tea, coffee, and calcium-rich foods may reduce iron absorption and should be avoided around the time of iron supplementation. Adequate intake of folate, vitamin B_{12}, protein, and other micronutrients is also essential for normal erythropoiesis, with supplementation recommended when dietary intake is inadequate or deficiencies are confirmed.[2–6]

Individualized dietary counseling, considering cultural practices, socioeconomic status, and nutritional habits, should form an integral part of antenatal care. Current international guidelines recommend combining nutritional optimization with iron supplementation rather than relying on dietary measures alone, as this integrated approach improves maternal hematological status, replenishes iron stores, and reduces the risk of adverse maternal and neonatal outcomes.[1–6]

Table 8. Dietary recommendations for prevention and management of anemia during pregnancy

Nutrient	Major food sources	Clinical importance
Iron	Green leafy vegetables, legumes, soybeans, sesame seeds, fortified cereals, lean meat	Hemoglobin synthesis and erythropoiesis
Vitamin C	Guava, amla, citrus fruits, tomatoes, bell peppers	Enhances non-heme iron absorption
Folate	Spinach, broccoli, legumes, fortified cereals	DNA synthesis and fetal neural tube development
Vitamin B_{12}	Milk, eggs, dairy products, meat, fish	Erythrocyte maturation and neurological function
Protein	Pulses, paneer, soy products, dairy, lean meat	Globin synthesis and tissue growth

Prevention and Public Health Strategies

Although effective treatment options are available, prevention remains the most cost-effective strategy for reducing the burden of anemia during pregnancy. Contemporary public health approaches increasingly recognize that maternal anemia originates long before conception and is influenced by nutritional status during adolescence, reproductive health practices, socioeconomic conditions, and access to quality healthcare. Consequently, prevention requires a life-course approach extending from adolescence through pregnancy and the postpartum period rather than focusing exclusively on antenatal supplementation.

Preconception counseling represents the first opportunity to optimize maternal nutritional status. Women planning pregnancy should undergo assessment for anemia, receive nutritional counseling, and initiate iron and folic acid supplementation where appropriate. Early correction of depleted iron stores substantially reduces the likelihood of developing clinically significant anemia during pregnancy and improves maternal physiological adaptation to increasing gestational demands. Recent evidence suggests that women entering pregnancy with adequate ferritin concentrations experience fewer obstetric complications and require less intensive therapeutic intervention later in pregnancy.[1–6]

Routine antenatal screening remains the cornerstone of secondary prevention. Measurement of hemoglobin concentration during the first antenatal visit, followed by repeat assessment during the second and third trimesters, facilitates early detection of declining hematological status before severe complications develop. Women identified as being at high risk—including adolescents, women with multiple gestations, previous anemia, closely spaced pregnancies, chronic diseases, or poor nutritional status—should undergo closer surveillance and individualized management.

Population-level interventions have also demonstrated substantial effectiveness. Universal iron–folic acid supplementation, food fortification programs, deworming in endemic regions, malaria prevention, delayed adolescent pregnancy, birth spacing, female education, and improved sanitation collectively contribute to reducing the incidence of maternal anemia. However, recent evidence emphasizes that supplementation programs alone cannot eliminate anemia unless accompanied by broader improvements in nutrition, women's education, healthcare accessibility, and socioeconomic development. Therefore, successful public health strategies should integrate clinical interventions with multisectoral policies addressing the underlying determinants of maternal health.

Conclusion

Anemia during pregnancy continues to represent one of the most prevalent and preventable complications affecting maternal and fetal health worldwide. Despite significant advances in antenatal care, nutritional supplementation, and evidence-based clinical guidelines, its burden remains disproportionately high in low- and middle-income countries because of the combined influence of nutritional deficiencies, infectious diseases, chronic medical conditions, socioeconomic disparities, and inadequate healthcare access. Although iron deficiency remains the predominant etiology, contemporary evidence highlights that maternal anemia is a multifactorial disorder requiring a comprehensive diagnostic approach that extends beyond measurement of hemoglobin concentration alone. Evaluation of iron biomarkers, particularly serum ferritin, together with assessment of nutritional status, inflammatory conditions, and underlying systemic disorders, facilitates accurate diagnosis and individualized treatment.

The physiological adaptations of pregnancy, including plasma volume expansion and increased iron requirements, further complicate clinical interpretation and emphasize the importance of trimester-specific assessment. Untreated anemia adversely affects maternal health by increasing the risk of fatigue, reduced functional capacity, postpartum hemorrhage, infections, cardiovascular complications, and maternal mortality, while simultaneously compromising fetal growth, increasing the incidence of preterm birth, low birth weight, neonatal anemia, and impaired neurodevelopment. These findings reinforce the need for early antenatal screening and timely intervention before severe anemia develops.

Current management strategies increasingly emphasize personalized care based on disease severity, gestational age, underlying etiology, and maternal risk factors. While oral iron supplementation remains the cornerstone of treatment for uncomplicated iron deficiency anemia, intravenous iron preparations have emerged as safe and effective alternatives for women with moderate-to-severe anemia, poor treatment tolerance, malabsorption, or inadequate response to oral therapy. Nutritional optimization, patient education, and management of associated medical conditions remain essential adjuncts to pharmacological treatment and contribute significantly to long-term maternal health.

Recent advances in the understanding of iron metabolism, particularly the role of hepcidin, have transformed the conceptual framework of maternal anemia by distinguishing absolute iron deficiency from functional iron deficiency. Emerging biomarkers, improved intravenous iron formulations, and increasingly individualized treatment strategies offer promising opportunities to enhance diagnostic precision and therapeutic outcomes. Nevertheless, further high-quality multicenter studies are required to establish standardized diagnostic thresholds, evaluate novel biomarkers, and optimize evidence-based management protocols across diverse populations.

Ultimately, reducing the global burden of anemia during pregnancy will require an integrated approach combining preconception nutritional optimization, routine antenatal screening, timely diagnosis, individualized treatment, and comprehensive public health interventions addressing the underlying social and nutritional determinants of maternal health. Strengthening maternal nutrition programs, improving healthcare accessibility, promoting health education, and implementing evidence-based clinical practices will be essential for achieving sustained reductions in maternal anemia and improving pregnancy outcomes worldwide.

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