



Review Article

A SCITECHNOL JOURNAL

Anemia: A Brief Overview Regards the Pregnant State

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Abstract

Anemia in pregnancy has been a long talked about problem. In India, anemia is the second most common cause of maternal mortality, affecting around 84% of pregnant women and accounting for about 20% of the total maternal deaths. Despite massive efforts, it continues to be a major cause of morbidity as well, especially in pregnant females, which in turn bears consequences on the physical and mental development of the fetus and the pregnancy outcome. Anemia may well be a simple physiological aggravation, during pregnancy, or may be pathological. Timely diagnosis and intervention can go a long way in improving both the maternal and the fetal outcome.

Keywords

Pregnancy; Anemia; Morbidity; Fetus

Introduction

The red blood cell and its associated milieu undergo certain physiological changes during the pregnant state. Health of both fetus and mother rests largely on health and nutritional status of mother. Consequently, maternal health and associated co-morbidities if any are to be taken special care of during pregnancy.

According to WHO (World Health Organization), in developing countries, prevalence of anemia in pregnant females is 56% on an average (ranging between 35-100% in different regions of the world) [1]. In India, anemia is the second most common cause of maternal mortality, affecting around 84% of pregnant women [2] and accounting for about 20% of the total maternal deaths. Amongst lactating females prevalence is found to be as high as 92.2% [1,2].

The CDC (Centers for Disease Control) in 1990 [3] recommended cut off values of Hb and Hct to label anemia in pregnancy are mentioned in Table 1.

Hemoglobin thresholds used to define anemia by the World Health Organization [4] are mentioned in Table 2.

NFHS (National Family Health Survey)-3 data [2005-2006] gives the severity wise prevalence of anemia in Indian women [5]. Mild anemia was seen in 38.6%, moderate in 15.0% and severe anemia was seen in 1.8% cases.

Physiologic anemia of pregnancy, results from reduction in concentration of hemoglobin as a result of dilution because the

Table 1: CDC* Hemoglobin and Hematocrit cut-offs for defining anemia in pregnancy.

Trimester	Hemoglobin (g/dL)	Hematocrit (%)
First	<11	<33
Second	<10.5	<32
Third	<11	<33

*CDC-Center for Disease Control

Table 2: WHO** cut offs for labelling anemia.

Age/ Gender group	Hb threshold(g/l)
Children(6-59 months)	≤ 110
Children(5-11 yrs)	≤ 115
Children(12.00-14.99 yrs)	≤ 120
Non-pregnant women(>14.99 yrs)	≤ 120
Pregnant women	≤ 110
Men(>14.99 yrs)	≤ 130

**WHO-World Health Organization

plasma volume expands more (40-50%) than the red blood cell volume (20-30%). However, since pure physiological anemia does not satisfy the CDC cut-offs mentioned above, a few people consider the term 'physiological anemia of pregnancy' to be an oxymoron.

In pathologic anemia of pregnancy, the oxygen-carrying capacity of the blood is diminished because of umpteen reasons which might have pre-existed or might have been aggravated by the pregnant state itself. Pre-existing causes may lead to decreased production, increased destruction or blood loss, resulting in a fall of hemoglobin to extents as would satisfy the CDC definition.

It is essential to reach to an etiologic diagnosis of anemia during pregnancy, especially to prevent effects on the fetus as also that on future pregnancies and make timely interventions if possible. Maternal anemia influences placental vascularization by altering angiogenesis during early pregnancy

Principal causes of anemia discussed here are;

Acquired

- Anemia due to hematinic deficiencies
- Anemia due to acute blood loss
- Anemia of inflammation
- Acquired hemolytic anemia
- Aplastic anemia

Hereditary

- Hemoglobinopathies
- Hereditary hemolytic anemias

Hematinic Deficiencies

Hematinics are vitamins and minerals essential for normal erythropoiesis and deficiencies of which lead to dyserythropoiesis and anemia. These include iron, copper, cobalt, vitamins A, B₁₂, B₆, C, E, folic acid, riboflavin, and nicotinic acid. Deficiencies of these are

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Received: August 11, 2013 Accepted: January 08, 2014 Published: January 13, 2014

more likely to occur in pregnancy because of the increased demands. Most common hematinic deficiencies during pregnancy follow the same incidence as in non-pregnant state, reflecting their dependency principally on nutritional status again [6]. Thus, iron, folate, and vitamin B₁₂ deficiencies are the most common hematinic deficiencies.

Transfusions of red blood cells or whole blood is seldom indicated for anemia attributable to hematinic deficiencies, unless there is associated hypovolemia from acute blood loss or an emergency operative procedure has to be performed on a severely anemic woman.

Iron Deficiency

An Indian study [7] reported iron deficiency anemia in 65% of the study population. Iron deficiency mainly reflects poor nutrition principally attributed to economic status worldwide, and is consequently more rampant in the developing countries. Moreover, females are especially prone to develop iron deficiency due to menstrual losses. The risk is even more in multiparous females due to poorly furnished demands of previous pregnancies.

The recommended daily allowance for iron in pregnancy is 30 mg/day. Iron requirements in pregnancy rise sharply from 1–2 mg/day in the first trimester to 4 mg/day in the second trimester and peaking at 6mg/day in the third trimester. Lactation requires 0.5–1.0 mg/day of iron. More than 500 mg of stored iron is required to avoid iron deficiency in pregnancy.

O'Brien [8] believes that transfer of iron to the fetus is regulated in response to maternal iron status at the level of gut. Iron deficiency anemia in pregnancy is also a risk factor for preterm delivery and subsequent low birth weight and poor neonatal health as well [9]. Maternal iron deficiency may affect iron status in their babies and predispose them to iron deficiency [10], while others believe that the amount of iron diverted to the fetus is similar in a normal and in an iron-deficient mother, and hence the newborn infant of a severely anemic mother usually does not suffer from iron-deficiency anemia [6].

There is a physiological increase in levels of serum transferrin, total iron binding capacity (TIBC) and plasma bound iron, especially during the second half of pregnancy which is attributable to the increased synthesis of transferrin by the liver [6].

At least 200mg of elemental iron should be supplemented for correction of anemia. Orally absorbable iron preparations viz ferrous sulphate, fumarate or gluconate may be used. If the woman cannot take or tolerate oral iron preparations, parenteral therapy is instituted [11]. Oral iron therapy should be continued for 3 months after anemia has been corrected, to replenish the depleted iron stores.

Folate/Vit B₁₂ Deficiency

There has been an increasing evidence of megaloblastic erythropoiesis as one of the major etiologies of anemia in pregnancy [12]. Folate deficiency has a low prevalence especially in developed countries. It also reflects the maternal nutritional status. Vitamin B₁₂ deficiency is rare in pregnancy as it is usually associated with infertility [10]. B₁₂ plays a key role in the development of new tissue; thus women who are deficient may not ovulate, or a fertilized egg may not develop, resulting in miscarriage.

Low levels of vitamin B₁₂ have also been found in women with children with neural tube defects, though a direct causal association has not been proved yet [10].

Treatment of clinical vitamin B₁₂ deficiency has traditionally been accomplished by intramuscular injection of crystalline vitamin B₁₂ at a dosage of 1 mg weekly for eight weeks, followed by 1 mg monthly for life. Vitamin B₁₂ has been found to be safe in pregnancy [13].

The principal factors operating in the pathogenesis of megaloblastic anaemia in pregnancy and the puerperium are dietary deficiency and an increased demand by the developing fetus. The maternal stores of folic acid are used up by the growing fetus, and this process is accelerated in multiple pregnancies, after hemorrhage, or in women with hemolytic anemia. Maternal folate deficiency may also predispose to prematurity [14].

The treatment of pregnancy-induced megaloblastic anemia should include folic acid, a nutritious diet, and iron. The American College of Obstetricians and Gynecologists [15] recommends a periconceptual folic acid supplementation (0.4 mg) for all women of the childbearing age. Additional folic acid is given in circumstances in which folate requirements are increased, such as in multi fetal pregnancy, hemolytic anemia, Crohn's disease, alcoholism, and inflammatory skin disorders.

Anemia From Acute Blood Loss

Anemia due to acute blood loss is less common in early pregnancy and maybe seen in cases of abortion, ectopic pregnancy and hydatidiform mole. Vaginal bleeding in late pregnancy maybe due to potentially serious conditions, including placenta praevia, placental abruption, and vasa praevia [16]. More commonly, anemia from obstetrical hemorrhage is encountered postpartum. Greater blood loss is associated with longer duration of the first stage of labour, increased placental weight, receipt of oxytocin, preterm birth, and grand multiparity [17]. Moreover, women with pre-existing anemia are suspected to be at a greater risk of having post partum hemorrhage, though this needs confirmation [17]. Massive hemorrhage demands immediate treatment with volume replenishment and supportive care. Once hemostasis has been achieved and euvoemia has been established, the residual anemia is treated with iron. In a moderately anemic woman (hemoglobin greater than 7 g/dL) who is hemodynamically stable, can ambulate without adverse symptoms, and is not septic, iron therapy is given for at least 3 months.

Anemia Associated with Chronic Disease

Chronic inflammatory states including both infections and malignancies, result in moderate and occasionally severe anemia, usually of the normocytic-normochromic type and sometimes microcytic hypochromic type. Chronic renal failure, cancer and chemotherapy, TB infection and tropical diseases like kala-azar and hyperreactive malarial spleen syndrome are the most common causes. In fact anemia is the most common hematological abnormality in HIV seropositive patients [18]. Also, inflammatory bowel disease and autoimmune states like systemic lupus erythematosus and rheumatoid arthritis are other well known etiologies.

As in the non pregnant state, anemia of chronic disease in pregnancy requires treatment of the underlying disease per se. For instance, highly active antiretroviral therapy (HAART) treatment avoiding zidovudine and treatment of associated tuberculosis (if present) are extremely important both to prevent and treat anemia in HIV cases [19]. Persistent improvement of anemia in tuberculosis needs general control of tuberculosis by anti-tubercular drugs, improving appetite, reducing hemoptysis episodes, and improving

intestinal absorption of iron (intestinal tuberculosis) [19].

Acquired Hemolytic Anemias

Autoimmune hemolytic anemia

It follows the same course as in the non pregnant states. However, there may be a marked acceleration in hemolysis during pregnancy. Both the direct and indirect antiglobulin (Coombs') tests are typically positive. IgM antibodies are large and do not cross the placenta. Hence, fetal RBCs are not affected in cold agglutinin disease. Glucocorticoids usually are effective, and treatment is with prednisone, 1 mg/kg per day, or its equivalent [10]. Variable fetal outcomes have been reported [20].

Drug-Induced hemolytic anemia

Hemolysis typically is mild, resolves on withdrawing the drug, and can be prevented by avoidance of the drug. Drug-induced hemolysis is much more often related to a congenital RBC enzyme defect, such as glucose-6-phosphate dehydrogenase (G6PD) deficiency.

Historically, alpha methyl dopa and high dose penicillin were considered to be the main causes of drug induced hemolytic anemias. Alpha-methyl dopa has been demonstrated to be safe for use during pregnancy and is now used to treat gestational hypertension but it should be used with caution [21]. While the incidence of drug induced immune hemolytic anemia has decreased, since then the second and third generation cephalosporins, especially, cefotetan and ceftriaxone have been associated increasingly with its incidence [22].

Pregnancy-Induced hemolytic anemia

Unexplained hemolytic anemia during pregnancy is a very rare but distinct entity in which severe hemolysis develops early in pregnancy and resolves within months after delivery [23]. There is no evidence of an immune mechanism or for any intraerythrocytic or extraerythrocytic defects [11] but because the infant also may demonstrate transient hemolysis, an immunological cause is suspected. Of late some case reports have also supported this theory [23]. Maternal corticosteroid treatment usually is effective.

Paroxysmal nocturnal hemoglobinuria

It is a serious and unpredictable disease and its course during pregnancy may be dangerous. Increased maternal mortality may occur due to post partum venous thromboses, life threatening hemolysis and haemorrhage. General principles of anti coagulation during pregnancy should be followed, with heparin being the anticoagulant of choice. Warfarin should typically be withheld, specially in the first trimester (due to risk of fetal malformations) and be re-started only 4-6 weeks post partum.

Other Acquired Anemias

Microangiopathic hemolytic anemia

Microangiopathic hemolysis with visible hemoglobinemia is seen with severe preeclampsia and eclampsia. Mild degrees are likely present in most cases of severe disease and may be referred to as HELLP syndrome-hemolysis, elevated liver enzymes, and low platelets. It occurs in 0.5 to 0.9% of all pregnancies and in 10-20% of cases with severe preeclampsia [24].

It is suggested that the variable response to treatment with heparin of patients with microangiopathic haemolytic anaemia may be due to

relative roles of intravascular coagulation and primary vascular disease in the pathogenesis of the microangiopathy, and to the development of irreversible vascular damage if treatment is delayed [25].

Hereditary spherocytosis

In general, women with hereditary spherocytosis do well during pregnancy. When complications develop, they are not serious as a rule. Anemia deteriorates in only a few patients and still little in those who are already splenectomized [26]. Folic acid supplementation is recommended. The newborn who has inherited hereditary spherocytosis (Autosomal Dominant inheritance) may have hyperbilirubinemia and anemia.

Among the hereditary RBC enzyme defects, G6PD deficiency is by far the most common. It is X-linked. Pyruvate kinase deficiency, although uncommon, is probably the next most common enzyme deficiency. It is inherited as an autosomal recessive trait. During pregnancy, iron and folic acid are given, oxidant drugs are avoided, and bacterial infections are treated promptly.

Aplastic and Hypoplastic Anemia

Aplastic anemia is rarely encountered during pregnancy. Association usually occurs by chance. It has been postulated that pregnancy induces erythroid hyperplasia [27]. The major risks to the pregnant woman with aplastic anemia are hemorrhage and infection which are also the two major causes of death due to it. Maternal and fetal outcomes are invariably poor in severe aplastic anemia [28]. Management is usually supportive. Even when thrombocytopenia is intense, the risk of severe hemorrhage can be minimized using vaginal delivery.

Hemoglobinopathies

Sickle-cell hemoglobinopathies

Major sickle disorders with severe clinical symptoms include sickle cell anemia (HbSS), sickle cell hemoglobin C (HbSC) disease, and sickle cell-beta thalassemia (HbS beta-Thal). Minor disorders include hemoglobin SE (HbSE) and hemoglobin SD (HbSD).

Deoxygenation of the abnormal red blood cells results in sickling. These permanently damaged red blood cells are then removed by the reticuloendothelial system, with the average red blood cell lifespan reduced to 17 days. The result is a chronic compensated anemia.

A woman who is pregnant is at risk of developing sickle cell crisis. These crises typically are vasoocclusive and may be precipitated by infection, though may also occur in preeclampsia. There is a high incidence of fetal growth restriction and perinatal mortality associated with sickle cell anemias which is likely due to sickling in the decidual vessels and due to poor placental perfusion [29]. Intrauterine growth restriction is uncommon in women with sickle cell trait [29]. Women suffering from sickle cell disease are more prone to develop complications like cerebral venous thrombosis, pneumonia, pyelonephritis, deep venous thrombosis, transfusion dependency, postpartum infection, sepsis and systemic inflammatory response syndrome. They are more likely to undergo caesarean section and experience pregnancy related complications (like abruptio, pre-eclampsia and preterm labour). Pregnant women with sickle cell trait are, however, not at an increased risk for developing pre-eclampsia [30].

In general, treating a pregnant woman who has sickle cell

disease requires close observation. Folic acid supplementation is recommended because of the quick turnover of erythrocytes. Pregnancy should be monitored with serial sonograms for fetal growth and the patient should be administered pneumococcal and meningococcal vaccines before pregnancy, if possible. Therapeutic measures for sickle cell crises are primarily supportive like initiation of intravenous fluid administration to decrease blood viscosity, pain management and treatment of any underlying infection. Supplemental oxygen and prophylactic transfusion may help when there is a sudden drop in hematocrit.

Prenatal diagnosis in the newborn is done using polymerase chain reaction, in which the DNA (deoxy ribonucleic acid) is obtained by amniocentesis or chorionic villus sampling.

Alpha-thalassemias

There are two major phenotypes. The deletion of all four α -globin chain genes (----) characterizes homozygous α -thalassemia. Because chains make up fetal hemoglobin, the fetus is affected in utero. Without α -globin chains, hemoglobin Bart and hemoglobin H are formed as abnormal tetramers. Fetal death occurs either in-utero or immediately after birth. Homozygous alpha thalassemia is the most common cause of hydrops fetalis in south east Asia [31].

HbH (β_4) disease is compatible with extrauterine life. The abnormal red cells at birth contain a mixture of hemoglobin Bart, hemoglobin H, and hemoglobin A. The neonate appears well at birth but soon develops hemolytic anemia. Most hemoglobin Bart present at birth is replaced postnatally by hemoglobin H. The disease is characterized by hemolytic anemia, which may be severe and similar to α -thalassemia major. Anemia in these women usually is worsened during pregnancy.

A deletion of two genes results clinically in α -thalassemia minor, which is characterized by minimal to moderate hypochromic microcytic anemia. Thus, genotypes may be $-/-$ or $-/.$ Differentiation is only by DNA analysis because there is no associated clinical abnormality with α -thalassemia minor, it often goes unrecognized. Red cells are hypochromic and microcytic, and the hemoglobin concentration is normal to slightly depressed. Women with α -thalassemia minor tolerate pregnancy quite well.

The single gene deletion ($-/$) is the silent carrier state. No clinical abnormality is evident in the individual with a single gene deletion.

Individuals with mild forms of alpha thalassemia may not require specific treatment except as needed for management of low hemoglobin levels. In some patients, supplementation of iron or folic acid may be useful. Patients with more severe anemia may require lifelong transfusion therapy [32].

Beta-thalassemias

In the typical case of thalassemia major, the neonate is healthy at birth, but as the hemoglobin F level falls, the infant becomes severely anemic and fails to thrive. A child who is entered into an adequate transfusion program develops normally until the end of the first decade, when effects of iron loading become apparent. A female who survives beyond childhood usually is sterile, and life expectancy even with transfusion therapy is shortened.

Patients of β thalassemia intermedia are at an increased risk for complications during pregnancy, which include, worsening anemia and thrombocytopenia, miscarriage, intra-uterine fetal demise, pre-

term labour, caesarean section and intrauterine growth restriction [33].

There is no specific therapy for β -thalassemia minor during pregnancy. Prophylactic iron and folic acid are given. Prenatal diagnosis of β -thalassemia using chorionic villus sampling can be carried out at 9 to 13 weeks. The course of pregnancy of patients with thalassemia minor, including perinatal outcomes, is favorable [34]. Karimi M et al. [35] interviewed 510 parents who had children suffering from β thalassemia major and 254 patients themselves, to determine their attitude towards prenatal diagnosis and subsequent medical termination of pregnancy. He found high compliance high compliance between the two. The decision not to have termination of pregnancy solely correlated with religious beliefs.

A study of pregnancy outcomes in 48 E- β thalassaemic women showed that 72.9% patients were transfusion dependent throughout the pregnancy, 18.75% landed into preterm labour, 14.5% patients underwent caesarean delivery, 45.8% patients had fetal growth restriction and 8.3% pregnancies ended in stillbirth [36].

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