

Hematological Disorders In Pregnancy

BY

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Hematological disorders in pregnancy include :

****Anemias**

****Platelet disorders**

****Thrombophilias**

Anemias

in

pregnancy

Physiological changes during pregnancy:

--Intravascular volume expansion begins at 8-10 weeks & reaches its maximum in the 2nd trimester.

--Plasma volume expansion is greater than the increase in the red cell volume resulting in physiological drop in hemoglobin & hematocrite concentrations (*physiological hemodilution*).

Definition of anemia during pregnancy:

--Hemoglobin concentration less than 12% g/dl & hematocrite value less than 36% which define anemia in general population are not applicable in pregnancy because of the physiological hemodilution .

--Drop of hemoglobin concentration below than 10% g/dl & hematocrite value less than 30% define anemia in pregnancy.

Classification of anemia during pregnancy:

--Iron deficiency anemia (80%) .

--Megaloblastic anemia.

--Hemolytic anemia.

--Aplastic anemia

Iron deficiency anemia :

- Many women when become pregnant have no adequate iron stores
- The iron content of unsupplemented diet provides only one to two thirds of pregnancy requirement.

Iron deficiency anemia :

The iron requirement in normal pregnancy:

--Expansion of red cell mass	450 mg
--Fetal iron	270 mg
--Placental iron	90 mg
--External losses	170 mg
Total	980

mg

The average diet provides about 1.5 mg per day

Iron deficiency anemia :

Pathophysiology (3 stages):

*****Depletion of Iron stores:*** The production of erythrocytes is maintained & the need of pregnancy are fulfilled

*****Deficient erythropoiesis:*** Transferrin saturation is less than 15%, erythropoiesis is impaired resulting in microcytosis & hypochromia

Iron deficiency anemia :

Pathophysiology 3 stages:

***Frank Iron deficiency Anemia:* The production of the red cells by the bone marrow is decreased

Ideally iron deficiency anemia should be detected and treated in the first stage

Iron deficiency anemia :

Diagnosis:

***Serum ferritin level by radioimmunoassay (n=155 ng/ml): values < 20 ng/ml indicate deficient iron stores*

***Serum transferrin level (n= 200-360 mg/dl): level increases with iron deficient erythropoiesis before frank signs of Iron deficiency anemia can be diagnosed*

Iron deficiency anemia :

Diagnosis:

***Decreased serum iron concentration (n=60-175 ug/dl)*

***Decreased transferrin saturation level (n= 25%-60%):*

***Microcytosis & hypochromia, low MCV & MCHC*

Iron deficiency anemia :

Prevention:

****Every pregnant woman needs iron supplementation which should be started as early as possible usually early in the second trimester**

****One tablet per day is enough to fulfil the needs of pregnancy provided it is taken for the remaining two trimesters and there is no pre-existing anemia.**

Iron deficiency anemia :

Treatment:

****Oral iron preparation 300 mg t.d.s. The response is rapid and the hemoglobin level rises by 0.3-1 gm per week, but GIT upsets may be a significant problem which may necessitate decreasing the dose.**

Iron deficiency anemia :

Treatment:

****Parenteral Iron therapy is indicate in :**

- patients with severe anemia (hemoglobin level < 8 g/dl) few weeks before their expected date of delivery**
- patients with malabsorption syndromes**
- patients who develop incapacitating symptoms with oral iron**

Megaloblastic anemia :

****Represents 3-4 % of anemias in pregnancy. The vast majority is due to folic acid deficiency. Very rarely due to vitamin B₁₂ deficiency.**

****Folic acid deficiency results from inadequate intake, poor absorption or increased utilization.**

****Both folic acid deficiency and vitamin B₁₂ deficiency cause megaloblastic anemia by affecting DNA replication**

Megaloblastic anemia :

****Vitamin B₁₂ deficiency causes progressive demyelination while folic acid deficiency does not. Therefore the differentiation between these two conditions is important.**

****Both folic acid deficiency and vitamin B₁₂ deficiency may mask iron deficiency because red cell synthesis is inhibited and iron is not utilized and once corrected red cell synthesis is resumed and iron deficiency becomes apparent**

Megaloblastic anemia :

Diagnosis:

****Increased MCV & presence of hypersigmented neutrophils.**

****Reticulocytic count (elevated in folic acid deficiency and normal in vitamin B₁₂ deficiency).**

**** serum levels of vitamin B₁₂ deficiency and folic acid.**

Megaloblastic anemia :

Treatment:

****Treatment of folic acid deficiency anemia requires 1 mg/day of folic acid.**

****Treatment of vitamin B₁₂ deficiency anemia requires 250 ug of cyanocobalamine every month.**

****An underlying iron deficiency anemia may be detected few days after initiation of therapy and should be treated.**

Hemolytic anemia :

****Normally RBCs live for about 120 days. In hemolytic anemia the life span of the RBCS is shortened due to premature destruction of the RBCs.**

****Destruction of the RBCs may occur intravascularly as in microangiopathic hemolytic anemia of preeclampsia or extravascularly as in acquired immune hemolytic anemia.**

Hemolytic anemia :

****Indicators of hemolysis are:**

- presence of free hemoglobin**
- decreased or absent haptoglobin**
- reticulocytosis**
- increased bilirubin & its metabolism**

Hemolytic anemia due to hemoglobinopathies :

**** *Sickle cell anemia:***

--Hemoglobin S is produced as a result of abnormality in β globin chains. In hypo-oxygenic states the red cells resume sickle shape with a rigid cell membrane which are finally cleared by the RES with an average life span of 17 days.

--Repeated transfusions, adequate oxygenation, intravenous hydration and intravenous methylprednisolone are treatment options.

Hemolytic anemia due to hemoglobinopathies :

**** β Thalassemia minor:**

--There is decreased synthesis of β globin chains. There is microcytic hypochromic anemia which may be wrongly treated by iron with no response which may be dangerous as they may develop cardiac and hepatic hemosiderosis.

Hemolytic anemia due to hemoglobinopathies :

--hemoglobin A2 & serum iron determinations should be ordered for every patient with microcytic hypochromic anemia not responding to iron therapy by elevation of the reticulocytic count and hemoglobin concentration.

--Patients with β Thalassemia minor do not require iron supplementation during pregnancy and if there is a need to raise their red cell concentration blood transfusion is needed.

Aplastic anemia :

****Very rare during pregnancy and has a serious prognosis since the mortality rate reaches 30%. However recent treatment option such as bone marrow transplantation, high dose corticosteroids and cyclosporine may improve the maternal outcome.**

**Thank
you**

