Anaemia in pregnancy

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**types**

There are many types of anaemia, based on characteristics and causes they can be listed as below:

1. Aplastic anaemia
2. Blood loss anaemia
3. Auto immune haemolytic anaemia
4. Diamond-Blackfan anaemia
5. Folate deficiency anaemia
6. Hemolytic anaemia (due to severe infections)
7. Pernicious anaemia
8. Iron deficiency anaemia
9. Sickle cell anaemia
10. Thalassemia

**Definition**

is a decrease in number of red blood cells (RBCs) or less than the normal quantity of hemoglobin in the blood. However, it can include decreased oxygen-binding ability of each hemoglobin molecule due to deformity or lack in numerical development as in some other types of hemoglobin deficiency.

Anaemia in pregnancy is a HB conc. of less than 11 gm/dl, or 6.8 mmol/l & a haematocrit of less than 33%

**Anaemia in pregnancy**

- a condition of pregnancy characterized by a reduction in the concentration of hemoglobin in the blood. It may be physiologic or pathologic.
- In physiologic anemia of pregnancy, the reduction in concentration results from dilution because the plasma volume expands more than the erythrocyte volume. The hematocrit in pregnancy normally drops several points below its pregnancy level.
- In pathologic anemia of pregnancy, the oxygen-carrying capacity of the blood is deficient because of disordered erythrocyte production or excessive loss of erythrocytes through destruction or bleeding. Pathologic anemia is a common complication of pregnancy, occurring in approximately half of all pregnancies.
- Disordered production of erythrocytes may result from nutritional deficiency of iron, folic acid, or vitamin B_{12} or from sickle cell or another chronic disease, malignancy, chronic malnutrition, or exposure to toxins. Destruction of erythrocytes may result from inflammation, chronic infection, sepsis, autoimmune diseases, microangiopathy, or a hematologic disease in which the erythrocytes are abnormal. Excessive loss of erythrocytes through bleeding may result from abortion, bleeding hemorrhoids, intestinal parasites such as hookworm, placental abnormalities such as placenta previa and abruptio placentae, or postpartum uterine atony.
### HB measure

<table>
<thead>
<tr>
<th>Age or gender group</th>
<th>Hb threshold (g/dl)</th>
<th>Hb threshold (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children (0.5–5.0 yrs)</td>
<td>11.0</td>
<td>6.8</td>
</tr>
<tr>
<td>Children (5–12 yrs)</td>
<td>11.5</td>
<td>7.1</td>
</tr>
<tr>
<td>Teens (12–15 yrs)</td>
<td>12.0</td>
<td>7.4</td>
</tr>
<tr>
<td>Women, non-pregnant (&gt;15yrs)</td>
<td>12.0</td>
<td>7.4</td>
</tr>
<tr>
<td>Women, pregnant</td>
<td>11.0</td>
<td>6.8</td>
</tr>
<tr>
<td>Men (&gt;15yrs)</td>
<td>13.0</td>
<td>8.1</td>
</tr>
</tbody>
</table>

### causes

- Impaired production
- Increased destruction
- Blood loss
- Fluid overload
- Causes
  - Acquired
    - Iron deficiency anaemia
    - Megaloblastic anaemia
    - Pernicious anaemia
    - Active bleeding
    - Aplastic anaemia
    - Acquired haemolytic anaemia
    - Anaemia associated with chronic disease
  - Hereditary
    - Thalassaemias
    - Sickle-cell haemoglobinopathies
    - Hereditary haemolytic anaemia (hereditary spherocytosis, hereditary elliptocytosis, and glucose-6-phosphate dehydrogenase or G6GD deficiency)

### Iron deficiency anaemia

It is the commonest type of anaemia in pregnancy. 42% of pregnant women have anemia worldwide. Almost 90% of anemic women reside in Africa or Asia. Most countries have policies and programs for prenatal iron-folic acid supplementation.

Iron deficiency anemia
Factors affect iron absorption
- Diatary iron
- Enhancers of absorption like proteins, ascorbic acid, gastric acidity, alcohol, low iron stores
- Inhibitors of iron absorption like: Ca, tea, coffee

Factors causing iron loss:
- Physiological factors like: basal losses from desquamation from intestines and skin, menstruation, delivery, lactation
- Pathological factors like: hookworm, haemorrhage from GIT, allergies, occult blood losses

Iron absorption in normal women is 15-30%, but it can increase to 50% in iron def. state and can reduce to 5-8% with an excessive haem diet.

Iron requirements in pregnancy vary with the body wt. of the mother and the size/maturity of the fetus
Pregnant who do not take supplementary iron during pregnancy show a reduction in iron in the bone marrow as well as a progressive reduction in mean red cell volume and serum ferritin levels

Prevention
Prophylaxis of non-pregnant women
Iron supplementation during pregnancy
Treatment of hookworm infestation
Improvement of dietary habits
Social services
Food fortification

Clinical features of IDA
symptoms:.
- Mild, no symptoms
  feeling of weakness, exhaustion, lassitude, indigestion, loss of appetite, palpitation, dyspnoea, giddiness, oedema or even congestive heart failure

Signs:
- Mild no signs
  pallor, glossitis, stomatitis, oedema, soft systolic murmur in the mitral area, fine crepitations at bases of the lungs

Investigations
- CBC (comple blood count)
  Low HB
  Low hematocrit
  Low MCV
  low serum ferritin, a low serum iron level, an elevated serum transferrin
  high red blood cell distribution width (RDW)
  reflecting an increased variability in the size of red blood cells (RBCs)
  A low mean corpuscular hemoglobin (MCH) and/or Mean corpuscular hemoglobin concentration (MCHC)
  increase total iron binding capacity (TIBC)
• HB electrophoresis
• Bone marrow examination
• Gastric system examination
• Urin examination
• Sputum examination
• Peripheral blood film film for malarial parasites
• Chest X-ray for TB
• S. protein

Treatment

• Oral iron supplementation like
  Ferrous sulphate 300 mg/tab (60mg elemental iron/tab)
  Ferrous gluconate 300 mg/tab (36mg/tab)
  Ferrous fumarate 200mg/tab (66/tab)
• For prophylaxis 100 mg of elemental iron + 0.5 mg folic acid
• For treatment 180 mg of elemental iron/day (3tab/day)
• Parenteral iron therapy like
  iron dextran (imferon)IM or IV
  Jectofer plus (folic acid+vit B12 +iron)IM
  Sorbitol citrate IM
• Blood transfusion
• A slight increase in vitamin A intake can lead to a significant rise in hemoglobin levels
• Copper is necessary for iron uptake

Megaloblastic Anaemia

Is an anemia results from inhibition of DNA synthesis in red blood cell production. When DNA synthesis is impaired the defect in red cell DNA synthesis is most often due to hypovitaminosis, specifically a deficiency of vitamin B\textsubscript{12} and/or folic acid. The pathological state of megaloblastosis is characterized by many large immature and dysfunctional red blood cells (megaloblasts) in the bone marrow and also by hypersegmented neutrophils

Folate deficiency megaloblastic anemia

Folic Acid -- also called folate is change to dihydrofolic acid and then to tetrahydrofolic acid (folinic acid) which is required for cell growth and division. essential for the production, repair, and functioning of DNA, production of red blood cells to meet the needs of the fetus, the placenta, uterine hypertrophy

More common in multiple pregnancies

Causes

Dietary lack & together with prolonged cooking which destroys it
Pregnancy/lactation
Intake of goat’s milk
Malabsorption syndrome & gastrointestinal disease
Abnormally high demands (multiple pregnancy, hookworm infestations, bleeding & others)
Drugs like anti-epileptic
Iron therapy in IDA
**Clinical features**
- May be asymptomatic
- A feeling of weakness
- General malaise
- Unwell with loss of appetite
- Dyspnea on exertion
- Palpitation & ↑ cardiac output
- Intermittent claudication of the legs
- Pallor
- Pica
- Hepatosplenomegaly
- Bleeding spots in the skin

**Effect on pregnancy**
↑ incidence of abortion
Growth retardation
Infections & sepsis
Abruptio placentae
Pre-eclampsia in some of the patients but not all

**Effects on fetus**
Neural tube defects
Abortion
Premature babies
Small for date
Perinatal mortality

**Investigations**
HB <11g/dl, ↑ MCV & MCHC
Peripheral blood film (macrocytic anaemia with hypersegmentation of neutrophils, neutopenia, thrombocytopenia)
Low S.folat & low red cell folate
S.iron is normal or high
↑ urin formiminoglutamic acid, S.lactic dehydrogenase, S.homocysteine levels
Bone marrow exam. Will show megaloblastic picture

**Treatment**
Prophylaxis
300-500 microg/day (0.5mg/day) with iron is enough
More green vegetables, liver, food fortification

Treatment
Oral folate 5mg/day
Parenteral folate is indicated in gastric intolerance or in late pregnancy
Vit.C is helpful
Associated iron def. should be corrected by iron therapy
Megaloblastic anemia due to B12 deficiency

Is a low blood level of vitamin B12, it can cause permanent damage to nervous tissue as a long term effect.

Vitamin B12 was discovered from its relationship to the disease pernicious anemia, which is an autoimmune disease that destroys parietal cells in the stomach that secrete intrinsic factor.

Total amount of vitamin B12 stored in the body is about 2–5 mg in adults.

Clinical features

- Anemia with bone marrow promegaloblastosis (megaloblastic anemia)
- Gastrointestinal symptoms (pernicious anaemia)
- Neurological symptoms (ataxia, paresthesias, fatigue, depression, and poor memory)

Causes

- Inadequate dietary intake of vitamin B12. As the vitamin B12 occurs naturally only in animal products (eggs, meat, milk)
- Malabsorption or maldigestion syndrome
- Pernicious anaemia
- Gastrectomy
- Ileal disease & resection
- Infestations by the fish tapeworm *Diphyllobothrium*

Investigation

- Vit.B12 are lower (<90microg/l)
- Homocysteine increased leading to hyperhomocysteinemia
- Methylmalonic Acid is increased

Treatment

The average daily diet contains 5-30 microg of vit.B12 of which 1-5microg, is absorbed. Parenteral cyanocobalamin (250microg) IM. every month.

Acquired haemolytic anaemia

Autoimmune haemolytic an. It is uncommon condition

It may be due to warm-active autoantibodies or cold-active antibodies or a combination (primary or idiopathic)

Secondary type is caused by underlying diseases like lymphoma, leukaemia, connective tissue disease, some infections, chronic inflammatory disease, drug induced factors or HELLP syndrome.

Treatment

- Glucocorticoids (prednisone 1mg/kg/day)
- Treat the underlying causes
Aplastic or hypoplastic anaemia

Aplastic anemia is a condition where bone marrow does not produce sufficient new cells to replenish blood cells. The condition, per its name, involves both aplasia and anemia. Typically, anemia refers to low red blood cell counts, but aplastic anemia patients have lower counts of all three blood cell types: red blood cells, white blood cells, and platelets, termed pancytopenia.

Signs & symptoms

Anemia with malaise, pallor and associated symptoms such as palpitations
Thrombocytopenia (low platelet counts), leading to increased risk of hemorrhage, bruising and petechiae
Leukopenia (low white blood cell count), leading to increased risk of infection
Reticulocytopenia (low reticulocyte counts)

Causes

Idiopathic
Autoimmune disorder
exposure to toxins such as benzene, or with the use of certain drugs, including chloramphenicol, carbamazepine, phenytoin, quinine
Irradiation
Leukaemia
Parovirus

Diagnosis

- Bone marrow aspirate and biopsy: to rule out other causes of pancytopenia.
- History of iatrogenic exposure to cytotoxic chemotherapy: can cause transient bone marrow suppression
- X-rays, computed tomography (CT) scans, or ultrasound imaging tests: enlarged lymph nodes (sign of lymphoma), kidneys and bones in arms and hands (abnormal in Fanconi anemia)
- Chest X-ray: infections
- Liver tests: liver diseases
- Viral studies: viral infections
- Vitamin B₁₂ and folate levels: vitamin deficiency
- Test for antibodies: immune competency

Treatment

Bone marrow transplantation
Corticosteroids
Cyclosporine
Search for infection
Red cell transfusion
Platelet transfusion
Granulocyte transfusion
Sickle-cell anaemia
Sickle HB S results from a single B-chain substitution of glutamic acid by valine (because of a mutation in the haemoglobin gene) characterized by red blood cells that assume an abnormal, rigid, sickle shape. Sickle-cell disease, usually presenting in childhood, occurs more commonly in people from parts of tropical and sub-tropical regions where malaria is or was common. ↑ maternal morbidity and mortality, abortion, perinatal mortality

Sickle cell anaemia

Pathophysiology
Red cells with S undergo sickling when they deoxygenated & the HB aggregates, causing ischaemia & infarction within various organs. These changes produce clinical symptoms, predominantly pain, called (sickle crisis)

Chronic and acute changes from sickling include bony abnormalities, renal medullary damage, autosplenectomy, splenomegaly, hepatomegaly, ventricular hypertrophy, pulmonary infarction, leg ulcers, infection, and sepsis.

Management
Close observation with careful evaluation of all symptoms

The term sickle cell crisis should be applied only after all other possible causes of pain or fever or reduction HB concentration have been excluded

Folic acid
Eradication of any bacteriuria
Treatment of crisis
Assessment of fetal health
Management of labour

Thalassaemia
is an inherited autosomal recessive blood disease that originated in the Mediterranean region

They are characterized by impaired production of one or more of the normal globin peptide chains

Alpha-thala: impaired production of alpha peptide chain
Beta-thala: impaired production of beta globin chain

Management
- Mild thalassemia: patients with thalassemia do not require medical or follow-up care after the initial diagnosis is made. Patients with β-thalassemia trait should be warned that their blood picture resembles iron deficiency and can be misdiagnosed.
- They should eschew empirical use of iron therapy; yet iron deficiency can develop during pregnancy or from chronic bleeding.
- Severe thalassemia: patients with severe thalassemia require medical treatment, and a blood transfusion regimen was the first measure effective in prolonging life
- Folic acid 1mg/day
- Iron 60mg/day
- Blood transfusion