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Tania G Singh

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CLINICS IN OBSTETRICS

Containing Long and Short Cases in Obstetrics

LONG CASES

• Antenatal Care • Anemia in Pregnancy • Recurrent Pregnancy Loss • Antepartum Hemorrhage in Early Pregnancy • Antepartum Hemorrhage in Late Gestation • Uterine Size More than Expected • Uterine Size Less than Expected • Diabetes in Pregnancy • Hypertensive Disorders in Pregnancy • Previous Cesarean Section • Pain Abdomen during Pregnancy • Breech Presentation • Cardiac Disease in Pregnancy • Pyrexia in Pregnancy • Postpartum Hemorrhage • Puerperium

SHORT CASES

• Postterm Pregnancy • Convulsions in Pregnancy • Rh Negative Pregnancy • Jaundice in Pregnancy • HIV in Pregnancy • Thyroid Disorders in Pregnancy

Foreword
Nutan Jain

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CHAPTER 2

Anemia in Pregnancy

Tania G Singh

HISTORY IN A CASE OF ANEMIA AND SIGNIFICANCE OF EACH CONTENT

General Information

- Name
- Age → <17 years, additional demand ≈ 270 mg during the course of pregnancy
- Address → resident of endemic or tropic areas
- Marital status
- Education level
- Economic resources (Income and category according to Kuppu Swamy Scale)
- Employment (type of work and position of the patient) → if agriculture → ↑ sweat → iron loss 15 μg/month
- Housing (type, size, number of occupants, electricity, cooking facilities) → unemployment, poor housing and overcrowding ↑the risk of TB, malnutrition and IUGR.

HISTORY OF PRESENT ILLNESS (HOPI)

- Asymptomatic — iron deficiency in the absence of anemia. It can manifest as:
  - Mild anemia (hemoglobin > 10 g/dL)
  - Gradual onset of anemia
  - In active, well-conditioned persons
- Excessive vomiting, nausea, loss of appetite
- Leg cramps, which occur on climbing stairs, also are common in patients deficient in iron.

Nonspecific Symptoms

- Fatigue
- Generalized weakness
- Lassitude
- Dyspnea on exertion
- Light-headedness, giddiness
- Palpitations
- Pruritus (Iron deficiency anemia).

Dietary/Personal History

- Diet (veg/nonveg/mixed)
- *Inadequate iron or protein intake:* Iron deficiency

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- *Strict vegetarian:* Vitamin B_{12} deficiency
- *Diet lacking in fruits and vegetables:* folate deficiency
- Dysphagia or loss of appetite
- Calculate kcal/day
- Smoking — IUGR
- Alcohol — IUGR
- Malabsorption with hypochlorhydria → ↓ absorption
- A fundamental concept is that after age of 1 year, dietary deficiency alone is not sufficient to cause clinically significant iron deficiency, so a source of blood loss should always be sought
- Pica — compulsive eating of nonfood substances
  - Pica is not a cause of iron deficiency anemia; it is a symptom of iron deficiency anemia
  - It is the link between iron deficiency anemia and lead poisoning, which is why iron deficiency anemia should always be sought when a child is diagnosed with lead poisoning. Both substances decrease the absorption of dietary iron
  - Pagophagia (ice eating) — most common form of pica in patients with iron deficiency anemia
  - Usually, they crave for ice to suck or chew
  - Occasionally, patients are seen who prefer cold celery or other cold vegetables in lieu of ice
  - Clay and dirt eating (geophagia) also occurs
  - Starch (amylophagia)
  - Hair (trichophagia).
- Sleep
- *Bladder:* UTI/dysuria
- *Bowel habits:* Constipation/diarrhea/dysentery/hemorrhoids.

History of Hemorrhage

- Two-thirds of body iron is present in circulating red blood cells as hemoglobin
- Each gram of hemoglobin contains 3.47 mg of iron; thus, each mL of blood lost from the body (hemoglobin 15 g/dL) results in a loss of 0.5 mg of iron
- Causes:
  - Bleeding is the most common cause of iron deficiency from:
    - History of passing worms in stools
      - Blood depletion (0.5–2 mg daily)
      - Each worm extracts ≈ 0.05 mL/day
      - There is intestinal hurry → ↓ iron absorption
    - With hematuria, hematemesis, hemoptysis, patients will present before they develop chronic iron deficiency anemia
    - But GI bleed (e.g. melena) may go unnoticed: *Blood must remain in GI tract for at least 14 hours before melena develops*
  - Increased menstrual bleed. Ask the patient about history of passage of clots, number of days of bleeding, use of multiple pads
  - IUCD in situ just prior to index pregnancy (IUCD users tend to bleed more)
  - Any obstetric hemorrhage in present pregnancy.

Obstetric History

- Repeated pregnancy at short intervals with prolonged lactation (≈ 2 years are required to replenish ≈ 1000 mg iron lost in pregnancy in healthy females with adequate diet)

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- Pregnancy interferes with maternal erythropoiesis by competing for folic acid, vitamin B, proteins, iron → hence explains polymorphism of anemia in pregnancy.

Past History

- Blood transfusion
- Allergy to medication or food
- Current use of any medications:
  - Antacids or any other medications which need to be changed or interfere with iron absorption
  - Oxidant medications provoke G6PD.

Family History

- Hereditary spherocytosis
- Sickle cell anemia (Black patients)
- Thalassemia (Asian, Black or Mediterranean patients)
- Other hemoglobinopathies
- Glucose-6-phosphate dehydrogenase deficiency (G6PD).

History of Chronic Illness (Anemia of Chronic Disease)

- Chronic infection
- Collagen vascular disease
- Malignancy
- Hypothyroidism
- Hypoadrenalism
- Renal disease
- Hypopituitarism
- History of gastrectomy or malabsorption (e.g. diarrhea)
  - Vitamin B_{12} deficiency
  - Iron deficiency anemia
  - Folate deficiency anemia
- Recent infection (Infection↑↑ interference with erythropoiesis)
  - Urinary tract (UTI, asymptomatic bacteriuria, pyelonephritis)
  - Malaria, Tuberculosis } recently or in index pregnancy
  - Unexplained fever
  - *Parvovirus:* Erythroblastopenia
  - *Hepatitis:* Aplastic anemia
- Abdominal pain
  - Peptic ulcer disease
  - Lead toxicity
  - Porphyria

General/Local Examination

- Built
- PICCLE (pallor, icterus, cyanosis, clubbing, lymphadenopathy, edema)

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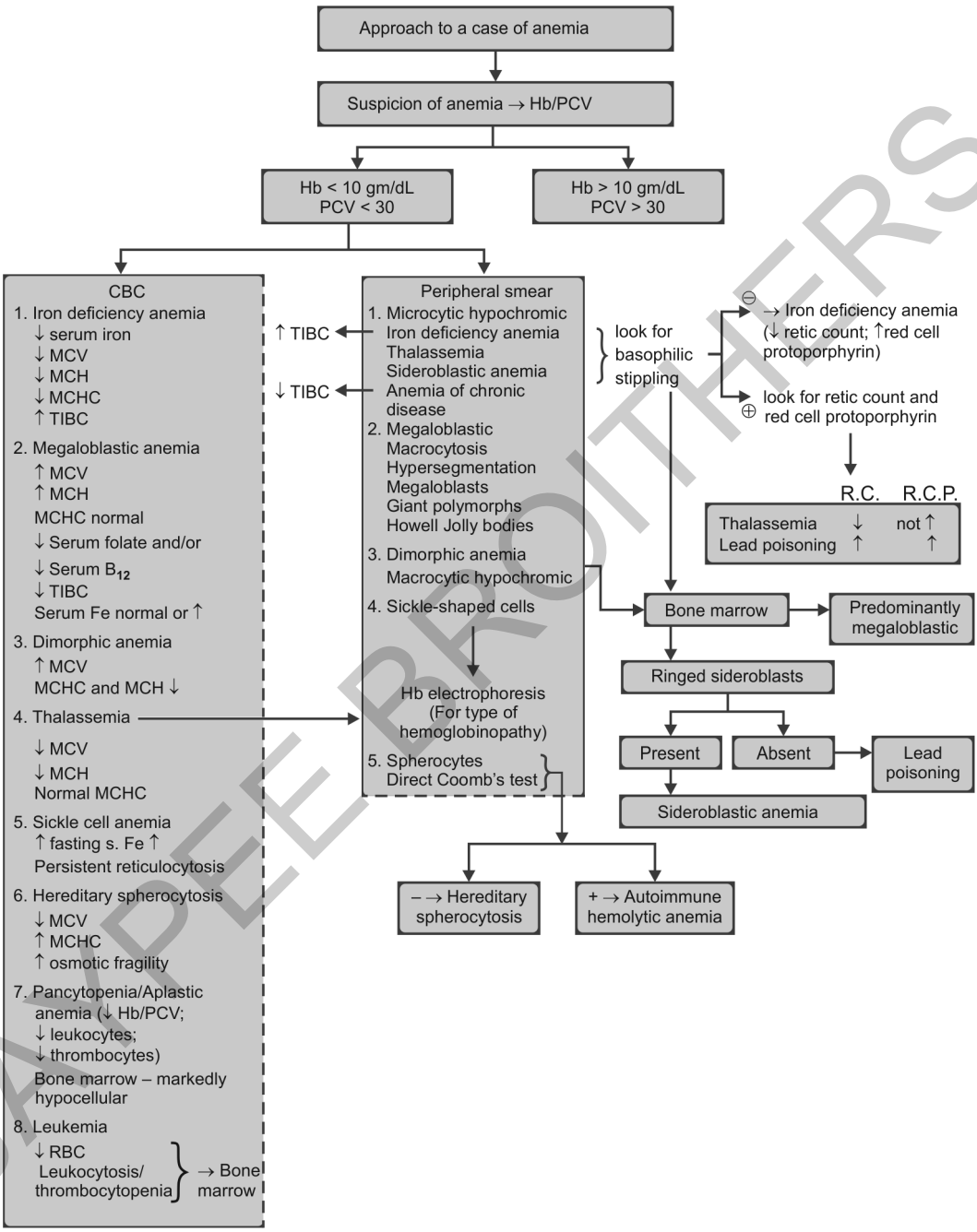

Abbreviations: * RC-Retic count; ** RCP-Red cell protoporphyrin

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- PR
- BP
- RR (look for dyspnea)

RS
CVS
CNS

Weight
Height
BMI

Thyroid
Breasts

- Lymph nodes in neck, axillae, inguinal areas
- Ankle swelling or swelling of legs.

Signs (Clinical Clues)

- Pallor in severe anemia (hematocrit<25%, hemoglobin<7 g/dL)
  - Pale conjunctiva or mucous membranes
  - Pallor at the nail beds or palmar creases
- Jaundice
  - Hemolytic anemia
  - Hepatitis
- Angular cheilitis
  - Iron deficiency anemia
- Glossitis
  - Iron deficiency anemia
  - Vitamin B_{12} deficiency
  - Folate deficiency anemia
- Splenomegaly
  - Chronic hemolytic anemia
  - Acute infection
  - Leukemia
  - Lymphoma
  - Portal hypertension
- Frontal bossing with prominent malar and maxillary bone
  - Chronic hemolytic anemia
- Neurologic changes (dementia, ataxia, paresthesia)
  - Vitamin B_{12} deficiency
- Symphysis fundal height – to look for IUGR secondary to anemia.

1. Define anemia

Definition

- Any condition in which the number of red cells, the amount of hemoglobin and the volume of packed red blood cells per unit volume is less than normal
- A pathophysiological condition in which the body cannot meet its demands for oxygen.

WHO

- No anemia ≥ 11 g/dL
- Mild 10–10.9 g/dL
- Moderate 7–9.9 g/dL
- Severe 4–6.9 g/dL
- Very severe < 4 g/dL

CDC

- Hb <11 g/dL in 1st and 3rd trimesters
- Hb <10.5 g/dL in 2nd trimester (Packed cell volume ≈ 32%)

Pregnancy

- For practical purposes, Hb <10 g/dL at any time during pregnancy is considered anemia, as plasma volume expands resulting in Hb dilution
- Hb ≤ 9 g/dL → need to investigate.

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Prevalence

- High prevalence ≥ 40%
- Medium prevalence 15–39%
- Low prevalence 5–14.9%
- Not a problem <5%.

Global scenario

- India has the highest prevalence of iron deficiency anemia among women in the world, including adolescents: 60-70% of Indian adolescent girls are anemic (2010)
- Worldwide—2 billion people affected (25% of the population)
- Out of this 40–75% are in developing world
- 58% of pregnant women in India are anemic
- Maternal mortality because of anemia — 20–40% (in India)

} Guidelines for control of IDA, NRHM, 2013

2. RBC 'Rule of 3'

For normal erythrocytes:

- Hemoglobin (g/dL) ≈ 3 × RBC count (millions)
- Hematocrit (%) ≈ 3 × hemoglobin (g/dL) ± 3%.
  Failure to obey this rule suggests an abnormality in erythrocytes (e.g. sickle cells, etc.)

3. Iron distribution in the body

Distribution

Total quantity of iron in the body ≈ 4 g. Out of this,

- 65% — present as hemoglobin
- 15–30% — mainly in RES and liver parenchymal cells in the form of ferritin (iron stores)
- 4% — myoglobin
- 1% — various heme compounds that promote intracellular oxidation
- 0.1% — combined with protein transferrin in blood plasma.
  Absorption of 1.4 mg/day is required from the diet in women to maintain hemostasis.
  To maintain adequate supplies of iron for heme synthesis, 20 mg of iron is recycled daily, going from senescent red cells that are removed from the circulation to new cells in the bone marrow.

Transport and Metabolism

Three stages–

1. Secretion

Liver → secretes apotransferrin into bile $\xrightarrow{\text{through}}$ bile duct is absorbed in small intestine.

2. Absorption

In duodenum (2nd part) and jejunum → apotransferrin + iron → transferrin (loosely bound, can be released to any tissue cell at any point in the body) → stimulates receptor on enterocytes → enters it and is released as plasma transferrin.

3. Actual metabolism

Fe^{3+} (in food) $\xrightarrow{\text{Fe reductase present in luminal border of enterocytes}}$ Fe^{2+} (from duodenal lumen to inside erythrocyte)

$\xrightarrow{\text{DCT 1 (Transporter protein)}}$ Fe^{2+} enters in enterocyte where it is stimulated by ferroxidase and is converted into Fe^{3+}.

This ferric iron has three forms:

1. Within the enterocyte, it forms a complex with ferritin → when intestinal epithelial cells are shed, ferritin is also shed with it.

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2. In plasma, combines with transferrin → this will be excreted.
3. It is stored in combined form with apoferritin.

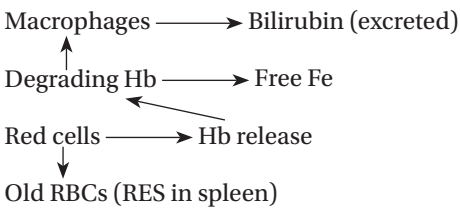
Tissues → free iron is stored as ferritin and hemosiderin.

Ferritin

- Storage form
- Deposited especially in reticuloendothelial cells and liver hepatocytes
- Apoferritin + Fe → ferritin (in cell cytoplasm).

Hemosiderin

- Small quantities are stored as this
- Extremely insoluble
- When total quantity of iron in body is more than apoferritin storage pool



- Fe excreted — 0.6 mg daily
- Fe lost in menses — 0.7 mg/day

4. Components of blood

Red blood cells

- Shape
  - Biconcave disc
- Size
  - Diameter
    - 7.5 μm
  - Thickness
    - Center →<1 μm
    - Periphery – 2.5 μm
- Average life span → 120 days
- Concentration in blood → 4.7–5.0 mill/cmm in women
- Daily RBC production requires ≈ 30–35 mg of iron/day (from recycling of storage pool iron)
- Production (Erythropoiesis → "erythro" — red and "poiesis" — to make)
  - Embryonic life
    - From stem cells in yolk sac
  - Fetal life
    - RBCs in liver, spleen and bone marrow
  - Newborn — childhood
    - Bone marrow of all bones and liver
  - Adult life
    - Bone marrow present in vertebra, sternum, ribs, iliac crest and proximal ends of long bones

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Differentiation of Erythrocyte

Hemocytoblast (a multipotent hematopoietic stem cell) → Common Myeloid Progenitor (a multipotent stem cell) → Unipotent Stem Cell → Proerythroblasts → Basophil erythroblast → Polychromatophilic erythroblast → Orthochromatic erythroblast → Reticulocyte → Erythrocyte.

These different appearances can be seen under light microscope after staining with James Homer Wright stain.

Regulatory Factors

- Erythropoietin
  - Produced by renal tubules (90%) and liver (10%)
  - Secretion stimulated by:
    - Decrease in tissue oxygenation
      - Low blood volume
      - Anemia
      - Low hemoglobin
      - Poor blood flow
      - Chronic lung disease
      - High altitudes
    - Placental lactogen
    - Progesterone
  - Stimulates stem cells in marrow → increases red cell production
  - Molecular weight: 55,000 kD
- Hepcidin **(recent advances)**
  - Peptide hormone
  - Composed of 25 amino acids
  - Synthesized in the liver and detectable in blood and urine
  - Plays a role in the regulation of hemoglobin production and thus affects erythropoiesis.

Role of Hepcidin in Iron Deficiency Anemia

- It acts as a coordination between use and storage of iron in the body
- It acts on the inhibition of intestinal iron absorption and iron release by macrophages and enterocytes and is a mediator in the cycle of iron absorption between the liver and intestine
- Measurement of hepcidin concentrations can be used for different diagnoses of anemia, such as iron deficiency anemia, which is characterized by low levels of this hormone
- Iron homeostasis is regulated by two main mechanisms:
  1. An intracellular mechanism, dependent on the amount of iron available for the cell
  2. A systemic mechanism, in which hepcidin plays a crucial role
- Inadequate oxygen (blood loss/hypoxia) →↑ production of erythrocytes →↓ Hepcidin and its inhibitory effects → more iron is made available from diet and from storage pool in macrophages and hepatocytes
- Hepcidin binds to ferroportin (protein required for ferrous transport into plasma) → regulates iron export into plasma
  - ↓ hepcidin → ferroportin exports iron → iron released in bloodstream
  - ↑ hepcidin → internalization and degradation of ferroportin molecules → iron release decreases progressively →↓ iron absorption in enterocytes

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- Hepcidin ↑ in anemia of chronic disease but the classic iron deficiency anemia in humans is associated with low hepcidin expression, which makes this hormone a potential marker for detection of iron deficiency anemia coexisting with anemia of chronic disease
- Diagnosis and therapy for anemia based on hepcidin may provide a more effective approach to prevent toxicity associated with iron overload.

Maturation

Vitamins

- RNA → vitamin B12 (early stages)
- DNA → folic acid (late stage)
- Folic acid requires vitamin C for folinic acid synthesis
- Deficiency of vitamin B_{12} → defective synthesis of both RNA and DNA
- Deficiency of folic acid → defective synthesis of DNA only

} Maturation failure of RBCs ↓

Result in megaloblast production having very little O_2 carrying capacity and have short life span

Minerals

- Iron (mainly)
- Traces of copper and cobalt

} Hemoglobin synthesis

Proteins

- Supply amino acid for synthesis of globin moiety
- *In nonpregnant state, iron flows mainly towards cells in marrow*
- *In pregnancy, iron flows towards trophoblastic cells of placenta and delivered to fetus*

Hemoglobin

- Globular proteins, comprising 1/3rd of the weight of red cell
- Molecular weight 68000
- Concentration in whole blood
  - Females — 13.5–14 gm% per mL
  - Each gram of hemoglobin can combine with 1.3 mL of O_2
- Consists of heme and globin
- Each red cell contains ≈ 640 million Hb molecules
- Synthesis
  - 65% in erythroblasts
  - 35% in reticulocyte stage
  - Heme synthesis occurs in mitochondria
  - Globin synthesis occurs in polyribosome
- It has eight functional globin chains
- Globin synthesis is first detected in primitive erythroid precursors of yolk sac at ≈ 3 weeks gestation
- Embryonic
  - Hb Gower I
  - Hb Portland
  - Hb Gower II

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- Fetal
  - HbF ($\alpha_2\ \gamma_2$)
  - HbA ($\alpha_2\ \beta_2$)

  

  change from fetal to adult hemoglobin occurs at 3–6 months after birth
- Adult
  - HbA ($\alpha_2\ \beta_2$) → > 95%, consists of 4 polypeptide chains, each with its own heme group
  - HbA2 ($\alpha_2\ \delta_2$) → 1.5–3.2%
  - HbF ($\alpha_2\ \gamma_2$) → 0.5–0.8% (<1%)

Classification of Anemia

Can be classified as:
- Physiological
- Pathological
  - Acquired
  - Inherited

Acquired

- Deficiency anemia
  - Iron deficiency
  - Folic acid deficiency
  - Vitamin B_{12} deficiency
  - Protein deficiency
- Hemorrhagic
  - Acute
    - Antepartum hemorrhage
  - Chronic
    - Hookworm infestation
    - Bleeding piles
- Bone marrow insufficiency
  - Hypoplasia or aplasia due to radiation, drugs (aspirin, indomethacin)
- Anemia of infection
  - Malaria, tuberculosis
- Anemia due to chronic disease
  - Renal disease or neoplasm.

Inherited

- Thalassemia
- Hereditary hemolytic anemia (RBC membrane defects, spherocytosis)
- Sickle cell hemoglobinopathy.

Concept of Physiological Anemia in Pregnancy

- RBC increases by (350 mL) 20–30%
- Plasma volume increases by (250 mL) 40–50%
- Erythrocyte dilution by 5–15%
- Decrease in hemoglobin concentration by ≈ 2 g/dL
- Total blood volume, increases by (1500 mL) 30–40%

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- Because of hemodilution → physiological anemia occurs → hemoglobin is brought down to 11 g%
- Blood picture of physiological anemia in pregnancy: normocytic, normochromic.

Benefits

- Decreases blood viscosity → decreases load on heart
- Facilitates blood flow through placenta
- Increases blood volume → acts as protective barrier against blood loss in third stage of labor.

Harmful Effects

- In case of cardiac disease, the increased circulatory burden can be dangerous, especially in the presence of anemia and fetus may be less efficiently oxygenated because of decreased oxygen carrying capacity of diluted blood.

Daily Intake of Dietary Iron

- 15 mg can replenish daily loss of 1.5 mg of iron
- Absorption rate → 10%.

Factors Leading to Anemia during Pregnancy

- *Increased demand*
  - Increased absorption by presence of HCl in stomach
  - Multiparity (increased iron demand by 2-folds)
  - Women with rapidly occurring pregnancy within 2 years following last delivery
  - Teenage pregnancy (e.g. at the age of 17, additional demand is ≈ 270 mg during pregnancy) → becomes nil by age 21
  - Parturition
  - Lactation
    - Adequate balanced diet → 18–20 mg of iron
    - Absorption rate increased by 20%, therefore, demand is hardly fulfilled.
- *Diminished intake of iron*
  - Low socioeconomic status
  - Vegetarian diet
  - Lack of balanced diet or poor intake (e.g. diet rich only in carbohydrates)
  - Alcoholism
  - High-risk ethnic groups
  - Drugs — alphadopa/levodopa/ciprofloxacin/cimetidine → interfere with iron absorption
  - Achlorhydria and copious intake of antacids decreases absorption
  - Loss of appetite and vomiting
  - Indian tradition — housewife eats last, after all male members and children have eaten and, in many families, the women eat only the leftovers. Hence, even though the food prepared for the family is the same, women are more prone to develop IDA than other members of the family.
- *Disturbed metabolism: presence of infection*
  - Asymptomatic bacteriuria
  - Chronic and latent pyelonephritis
  - UTI

  Therefore, antibacterial therapy is necessary before response to hematinics is expected.

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- *Decreased absorption*
  - Dietary factors (tannins, phytates in fiber, calcium in milk, tea, coffee, carbonated drinks)
  - Upper GI pathology
    - Chronic gastritis
    - Chronic diarrhea
    - Gastric lymphoma
    - Celiac disease
    - Crohn's disease
  - Medications that decrease gastric acidity or bind iron
  - Gastrectomy or intestinal bypass
  - Duodenal pathology
  - Chronic renal failure patients.
- *Iron loss*
  - Loss by sweat ≈ 15 mg/month
  - Repeated pregnancy at short intervals (along with prolonged lactation)
    - It requires ≈ 2 years to replenish ≈ 1000 mg of iron lost during delivery and lactation
  - Increased blood loss during menstruation
  - Hookworm infestation
    - 0.5–2 mg of iron daily (each worm extracts up to 0.05 mL of blood/day)
    - A hookworm burden of 40–160 worms (depending on the iron status of the host) is associated with iron deficiency anemia
  - Malaria (falciparum and vivax), chronic blood loss due to bleeding piles/dysentery
  - Sickle cell anemia and thalassemia
    - About 10% of the world's thalassemia patients belong to the Indian subcontinent and 3.4% of them are carriers
    - In India, about 32,400 infants are born with hemoglobinopathies every year
  - GI bleeding
  - Regular blood donors
  - Hematuria
  - Intravascular hemolysis: hemoglobinuria
  - Extreme physical exercise (endurance athletes)
  - Pathological (hemolytic)
- *Prepregnancy health status: anemic or inadequate state of stored iron*
- *Polymorphism*
  - Another important problem posed by anemia in pregnancy is its polymorphism. Pregnancy interferes with maternal erythropoiesis by competing for available raw materials.

5. How does *H. pylori* infection lead to iron deficiency anemia?

Iron deficiency occurs due to the following:

- *H. pylori* infection is intraluminal in proximal part of small intestine where iron absorption takes place and any disruption in metabolism and absorption would lead to iron deficiency anemia
- *H. pylori* infection may be caused via the decreased expression of DMT1 (divalent metal ion transporter 1)
- Ferric iron must be reduced to ferrous iron before absorption. Gastric acid and low gastric juice pH are needed for this reduction. Atrophic gastritis caused by *H. pylori* infection can decrease the excretion of gastric acid, which can decrease the absorption of iron

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CLINICS IN OBSTETRICS

Clinics in Obstetrics, as the name suggests, is basically meant for the preparation of practical and viva voce. Not only for the postgraduate and undergraduate students, this book is very useful for the practicing clinicians as well.

It is divided into two sections—one section for *Long Cases* and another section for *Short Cases*. All the chapters have been formulated keeping the latest trends in view. Each chapter contains a hypothetical case, followed by stepwise discussion starting with history, differential diagnosis and how to reach the final diagnosis. Management of each case is covered in complete detail.

It is an examination-friendly book, in a very comprehensive manner, with very original and authentic information starting from the basics and covering the recent advances. It is need-based and I hope it will well fulfill its purpose.



Tania G Singh MS (Obstetrics and Gynecology) has been working extensively in the field of obstetrics for the last seven years and is well-known for her analytical approach and a great attentiveness and concern towards high-risk pregnancies. She has finished her postgraduation from Jawaharlal Nehru Medical College, Belgaum, Karnataka. She is an outstanding medical writer and teacher par excellence and, employing her concepts and perceptions, she has made sincere efforts to simplify each case in this book for easy readability.

Dr Tania has also done her Fellowship in Gynae Endoscopy. She has been a speaker at various national conferences. She is a life member of Association of Obstetricians and Gynaecologists of Delhi (AOGD), The Federation of Obstetric and Gynaecological Societies of India (FOGSI), Indian Association of Gynaecological Endoscopist (IAGE) and various other national and international associations. She is a Senior Consultant in well-reputed private hospitals in New Delhi. She is also running her own center—Bodyline Trauma and Maternity Center—in New Delhi, India.

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