Childhood Anemia
A Practical Approach

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Useful facts of childhood anemia

1) In interpretation of laboratory haematological values, age and sex has to be taken into consideration.

2) Reticulocyte count is very often not included in the CBC.

3) Macrocytic anemia is rare in childhood.
Useful facts of childhood anemia

4) In macrocytosis, it is prudent to determine if the $\uparrow$MCV is due to reticulocytosis.

5) Many childhood anemias have a hereditary basis.

6) Nutritional deficiency is extremely rare in infants who are fed on commercial formula or breastfed by mothers with an adequate diet or taking supplement.
Useful facts of childhood anemia

7) Worldwide, Fe deficiency is probably the most common cause of isolated anemia especially in children aged 1-5 years.

8) In a patient with $\beta$ thalassaemia trait & concomitant Fe deficiency, HbA$_2$ level can be normal as Fe deficiency depresses $\delta$ globin synthesis; Hb electrophoresis should be repeated after Fe deficiency is corrected.

9) A group of anemias unique to childhood is aregenerative anemia and constitutional aplastic anemias.
Useful facts of childhood anemia

10) Parvovirus is unique in its erythrotropic nature & striking affinity for erythroid precursors and produces transient erythroid marrow aplasia. It may cause a dramatic fall in Hb in patients who have chronic haemolysis with a shortened red cell survival – “aplastic crisis”.
Pathophysiology of childhood anemia

Bone marrow

- Nutrients (Fe, vit B12, folates)
  - e.g. Thalassaemias
  - e.g. Nutritional deficiency

- Intrinsic ability (normal RBC precursors)
  - e.g. Aplastic anemia
  - Transient erythroblastopenia of childhood (TEC)
  - Diamond-Blackfan anemia
  - Fanconi anemia
  - Infiltrative disorders

- Protein/globin chains
  - e.g. Thalassaemias

Effective erythropoiesis

- Intact compartment
  - e.g. External bleeding
  - e.g. G6PD / PK deficiency

- Intact enzymes
  - e.g. G6PD / PK deficiency

- Intact extravascular
  - e.g. Hereditary spherocytosis

- Hormone (Erythropoietin)
  - e.g. Kidney disease

RBC

- Intact extracellular environment
  - e.g. Antibodies
  - Drugs
  - Toxins
  - Splenomegaly
  - Microangiopathy

- Intact enzymes
  - e.g. G6PD / PK deficiency

- Intact membrane
  - e.g. Hereditary spherocytosis

- Intact compartment
  - e.g. External bleeding
Physiologic classification of anemia

A. Blood loss

B. Reduced production
   1. Marrow failure
      a. Hereditary/constitutional
         Fanconi anemia
         Diamond-Blackfan anemia
         Dyskeratosis congenita
         Osteopetrosis……etc
      b. Acquired
         Idiopathic, infection, drug, malignant infiltration, myelofibrosis, chronic renal disease
   2. Impaired erythropoietin production
      Chronic renal failure,
      Hypothyroidism,
      Hypopituitarism,
      Chronic inflammation,
      Malnutrition

C. Disorder of maturation & ineffective erythropoiesis
   1. Abnormal cytoplasmic maturation
      Iron deficiency, thalassemia, sideroblastic anemia, lead poisoning
   2. Abnormal nuclear maturation
      B12 or folate deficiency, inborn or drug induced disorders of DNA synthesis
   3. Hereditary dyserythropoietic anemia

D. Hemolytic anemia
   1. Defects of Hb, RBC membrane or metabolism
      Thalassemia, spherocytosis, G6PD or pyruvate kinase deficiency
   2. Antibody mediated
   3. Mechanical, thermal, oxidant injury
   4. Infection induced
   5. Paroxysmal nocturnal hemoglobinuria
Symptomatology and etiology of anemia in children
• Non-specific symptoms
  – Irritability
  – Poor sleep quality
  – Anorexia
  – Poor concentration, school work
  – Failure to thrive
• Dizziness / syncope
• Malaise, easy fatigue, impaired exercise tolerance
• Palpitation
Staged algorithm of evaluation of anemia

1) Is the child truly anemic?
2) History
3) Physical examination
4) RBC indices & peripheral blood smear
5) Reticulocyte count
6) Other appropriate investigations
Is the child truly anemic?

- “Pallor” can be due to
  - True anemia
  - Vasoconstriction
  - Fair color
- Compare child’s Hb/Hct with normal values for age & sex
- Venous blood preferred
- Adequate volume of blood
Definition of anemia in children
• Anemia is functionally defined as an insufficiency in RBC mass to adequately deliver oxygen to peripheral tissue.

• For practical purpose anemia is defined by a laboratory value 2 SD below the mean for normal population in any 1 of the 3 red cell indices:
  – haemoglobin level (Hb), haematocrit (Hct) or red cell count (RBC)
Physiological changes in red cell indices (Hb, Hct, RBC) with age

- Very high level at birth
  - Relative hypoxemia in fetal life
- Physiological trough at 2 – 3 month of age
  - Dramatic reduction in erythropoiesis after birth
  - Rapid growth in early infancy
- Gradual rise from childhood to adolescent
- Higher level in male vs female in adulthood
  - Effect of androgens vs estrogen, menstruation
<table>
<thead>
<tr>
<th>Age</th>
<th>Haemoglobin (g/dL)</th>
<th>Haematocrit (%)</th>
<th>Red cell count (10^{12}/L)</th>
<th>MCV (fl)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>± 2 SD</td>
<td>Mean</td>
<td>± 2 SD</td>
</tr>
<tr>
<td>Birth (cord blood)</td>
<td>16.5</td>
<td>13.5-19.5</td>
<td>51</td>
<td>42-60</td>
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<tr>
<td>1-3 days (capillary)</td>
<td>18.5</td>
<td>14.5-22.5</td>
<td>56</td>
<td>45-67</td>
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<tr>
<td>1 week</td>
<td>17.5</td>
<td>13.5-21.5</td>
<td>54</td>
<td>42-66</td>
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<tr>
<td>2 weeks</td>
<td>16.5</td>
<td>12.5-20.5</td>
<td>51</td>
<td>39-63</td>
</tr>
<tr>
<td>1 month</td>
<td>14.0</td>
<td>10.0-18.0</td>
<td>43</td>
<td>31-55</td>
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<tr>
<td>2 months</td>
<td>11.5</td>
<td>9.0-14.0</td>
<td>35</td>
<td>28-42</td>
</tr>
<tr>
<td>3-6 months</td>
<td>11.5</td>
<td>9.5-13.5</td>
<td>35</td>
<td>29-41</td>
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<tr>
<td>0.5-2 years</td>
<td>12.0</td>
<td>10.5-13.5</td>
<td>36</td>
<td>33-39</td>
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<tr>
<td>2-6 years</td>
<td>12.5</td>
<td>11.5-13.5</td>
<td>37</td>
<td>34-40</td>
</tr>
<tr>
<td>6-12 years</td>
<td>13.5</td>
<td>11.5-15.5</td>
<td>40</td>
<td>35-45</td>
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<tr>
<td>12-18 years</td>
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<tr>
<td>Female</td>
<td>14.0</td>
<td>12.0-16.0</td>
<td>41</td>
<td>36-46</td>
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<tr>
<td>Male</td>
<td>14.5</td>
<td>13.0-16.0</td>
<td>43</td>
<td>37-49</td>
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<tr>
<td>18-49 years</td>
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<tr>
<td>Female</td>
<td>14.0</td>
<td>12.0-16.0</td>
<td>41</td>
<td>36-46</td>
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<tr>
<td>Male</td>
<td>15.5</td>
<td>13.5-17.5</td>
<td>47</td>
<td>41-53</td>
</tr>
</tbody>
</table>
Historical factors of importance in evaluating anemia in children
Historical factors of importance in evaluating anemia in children

<table>
<thead>
<tr>
<th>Age</th>
<th>Anemia manifesting in neonatal period is usually the result of recent blood loss, isoimmunization, congenital hemolytic anemia or congenital infection</th>
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<tbody>
<tr>
<td></td>
<td>Anemia 1st detected at 3 – 6 months suggests a diagnosis of haemoglobinopathy</td>
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<tr>
<td></td>
<td>Nutritional iron deficiency is seldom responsible for anemia before 6 months of age in term infants (earlier in preterm infants)</td>
</tr>
<tr>
<td>Gender</td>
<td>Consider X-linked disorders in male: G6PD deficiency</td>
</tr>
</tbody>
</table>
| Ethnicity          | Thalassemia syndrome more common in South East Asians and Mediterraneans  
|                   | Sickle cell disease more common in Africans                          |
| Inheritance       | Consanguinity, family Hx of anemia, jaundice, gallstone, splenomegaly / splenectomy |
| Neonatal          | Significant jaundice suggest congenital hemolytic anemia (e.g. hereditary spherocytosis, G6PD, pyruvate kinase deficiency)  
|                   | Prematurity predispose to early development of iron deficiency anemia |
| Diet              | Assess for dietary sources of iron, folic acid and vitamin B12  
<p>|                   | Pica, geophagia, pagophagia suggest iron deficiency                |</p>
<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastro-intestinal</td>
<td>Chronic diarrhea suggest small bowel disease with malabsorption (folate, B12, iron) or occult blood loss. Epigastric pain suggest occult upper GI bleeding</td>
</tr>
<tr>
<td>Infection</td>
<td>Hepatitis-induced aplastic anemia, infection induced hemolytic anemia or red cell aplasia. Suspect associated WBC abnormalities in recurrent infection</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Suspected associated platelet abnormalities</td>
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<tr>
<td>Drugs</td>
<td>Oxidant induced hemolytic anemia, phenytoin induced megaloblastic anemia, drug induced aplastic anemia</td>
</tr>
<tr>
<td>MR</td>
<td>Suspect congenital marrow failure or IEM</td>
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</tbody>
</table>
Physical findings as clues to the cause of anemia in children
<table>
<thead>
<tr>
<th>Physical Findings as Clues to the Etiology of Anemia</th>
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<tbody>
<tr>
<td>Skin</td>
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<tr>
<td>Keratoderma</td>
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<td>Jaundice</td>
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<tr>
<td>Large hemangioma</td>
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<tr>
<td>Ulcers on lower extremities</td>
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<tr>
<td>Facies</td>
</tr>
<tr>
<td>Eyes</td>
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<td>--------------------------</td>
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<tr>
<td>Micro-ophthalmia</td>
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<tr>
<td>Physical Findings as Clues to the Etiology of Anemia</td>
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<tr>
<td>-----------------------------------------------</td>
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<tr>
<td><strong>Mouth</strong></td>
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<tr>
<td><strong>Chest</strong></td>
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<td><strong>Hands</strong></td>
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<tr>
<td><strong>Spleen</strong></td>
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The red cell indices

- MCV (mean corpuscular volume)
  - The only red cell index directly measured by the electronic counter
  - Reflects a quantitative defect in the production of Hb due to ↓ haem or globin synthesis
  - Categorise anemias into microcytic, normocytic and macrocytic types
  - Value must be interpreted with age
• **Rule of thumb:**

  – In children < 10 years age, lower limit of MCV = 70 fl + age in years; if < 72 fl, usually abnormal.

  – > 6 months of age, upper limit for MCV is 84 + 0.6 fl per year till upper limit of 96 fl; MCV > 98 beyond the immediate neonatal period is very rare.
• MCHC & MCH are calculate values & therefore less accurate.

• MCHC is a measurement of cellular hydration status; an increase is characteristic of spherocytosis.
• **RDW (Red cell volume distribution width)**
  - Reflects the variability in cell size and measures the degree of anisocytosis
  - Normal < 14.5%
  - ↑ in Fe deficiency anemia
  - Normal in thalassemia trait
Reticulocyte count (RC)

- Reflects the rate at which new RBC are produced; normal < 1% after 3 months; at birth up to 10%.

\[
\text{Reticulocytes} = \frac{\text{RC as percentage}}{\text{RBC count}} \times 100\%
\]

- RC as in CBC may not reflect the true marrow response i.e. the raw RC may be misleading in anaemic patients.

- In anemic patients, the reticulocyte life span ↑ from 1 to 2-2.5 days
• Absolute reticulocyte count or reticulocyte index more accurately reflect the rate of erythropoiesis.

• **Absolute reticulocyte count**
  \[ \text{Absolute reticulocyte count} = \text{Reported reticulocyte }\% \times \text{RBC count} \text{ (N: 50-100 x 10}^9/\text{L}) \]

• **Reticulocyte index (RI)**
  \[ \text{Reticulocyte index} = \frac{\text{Reported reticulocyte count} \times \text{patient’s hematocrit}}{\text{normal hematocrit (0.45)}} \]

- Normal < 3%
• Reticulocyte production index (RPI)
  – To correct the longer life span of prematurely released reticulocytes

<table>
<thead>
<tr>
<th>Ht (%)</th>
<th>Reticulocyte survival (days)/maturation correction</th>
</tr>
</thead>
<tbody>
<tr>
<td>36-45</td>
<td>1.0</td>
</tr>
<tr>
<td>26-35</td>
<td>1.5</td>
</tr>
<tr>
<td>16-25</td>
<td>2.0</td>
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<tr>
<td>15 or below</td>
<td>2.5</td>
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</tbody>
</table>

\[
\text{RI} \\
\text{RPI} = \frac{\text{Reticulocyte survival (days)}}{\text{Maturation correction}}
\]
Example:

Retic count 6%; Hb 7g/dL; Hct 25%

\[ RI = \frac{6 \times 25}{45} = 3.33\% \]

\[ RPI = \frac{6 \times 25 \times 1}{45 \times 2.0} = 1.7\% \]

Normal RPI 1.0 – 2.0

\(< 2 \rightarrow \downarrow \text{ production} \)
\(> 2 \rightarrow \uparrow \text{ production} \)
Differential of anemia (Normal WBC/platelet)

Complete blood count: Hgb, indices, retic count & smear

Appropriate reticulocyte response to anemia (RPI > 2)

Yes

Evidence of haemolysis (↑bilirubin, ↑LDH, ↓haptoglobin hemoglobinuria)

Yes

Haemolysis

Haemoglobinopathy
RBC enzymopathy
RBC membranopathy
Extrinsic factors

No

Haemorrhage

No

Macrocytic
B12 deficiency
Folate deficiency
Medication
Liver disease

Normocytic
Chronic disease
Transient erythroblastopenia of childhood (TEC)
Renal disease
Endocrine disease

Microcytic
Fe deficiency
β-thalassaemias
α-thalassaemias
Lead poisoning
Differential of anemia (Abnormal WBC and/or platelet)

Complete blood count & peripheral blood smear

Abnormal WBC and/or platelet

Bone Marrow Failure

Peripheral consumption

infection

Haematological malignancy
Aplastic anemia (hereditary/acquired)
Aregenerative anemias (TEC, Diamond-Blackfan anemia, Parvovirus)
Infiltration

Microangiopathy
DIC
Splenomegaly
Autoimmune

EBV
Hepatitis virus
Tuberculosis
HIV
Microcytic anemia

- ↓ Serum iron
  - ↑ TIBC
  - ↓ Ferritin
  - Iron deficiency

- ↓ Serum iron
  - Normal or ↑ TIBC
  - ↓ Ferritin
  - Anemia of chronic disease (inflammation)

- Normal Serum iron
  - Normal TIBC
  - ↑ Ferritin
  - Thalassemia trait
    - Hemoglobin E
    - Hemoglobin C
    - Lead poisoning

- ↑ Serum iron
  - Normal TIBC
  - ↑ Ferritin
  - Congenital sideroblastic anemia
Iron deficiency anemia

- Most common haematological abnormality of childhood
- Total body Fe content 3-5G
- 2.5G in Hb
Causes of Fe deficiency

- Dietary deficiency
- Increased demand (growth)
- Defective absorption
- Blood loss
Who is at risk of Fe deficiency?

• Infants
  – Preterm babies
  – Infants after 6 months of age
    • Fe store depleted after 6 months
    • Rapid growth
    • Rapid increase in blood volume
    • Especially wholly breast fed
Who is at risk of Fe deficiency?

- **Toddlers**
  - Too early & too much cow’s milk
    - Max 16 oz/day
    - Interferes with food absorption – delay gastric emptying
    - Colitis
    - Decreased appetite – high satiation value
  - Picky food

- **Teenagers**
  - Increased growth at puberty
  - Menstrual loss
Fe deficiency anemia

Laboratory findings

In sequence
- BM haemosiderin
- RDW
- Ferritin
- ↓Fe, ↑TIBC
- ↓ Hb
- PBS – hypochromic, microcytic
- Reticulocyte response - insufficient
Fe deficiency anemia

Prevention

• Preterm – Fe supplement after birth
• Breast fed infants – Fe-fortified cereals at 6 months
• Formula-fed infants – Fe fortification
• No cow’s milk until 12 months
• Limit cow’s milk intake to < 16 oz / day in toddlers
Fe deficiency anemia

Treatment

• **Diet**
  – Decrease cow’s milk
  – Fe fortified foods
  – Iron rich food with high bioavailability e.g. fish, poultry, meat
  – Avoid phytates (bran, oats, rye, fiber) and tea

• **Fe therapy**
  Elemental Fe 4-6 mg/kg/day for at least 3 months
Transient erythroblastopenia of childhood (TEC)

- Relatively rare
- Unknown etiology
- Acquired erythroid marrow failure
- Often follows viral infection
- Age: 18 month to 2 years
- Child otherwise normal
TEC

- \(N^cN^c\) anemia
- Hb 5-7 g/dL
- Retic count: low
- Other cell lines normal
- Resolves in weeks to months
- Px: Supportive
  - Transfusion if indicated
Constitutional aplastic anemias

- Inherited bone marrow failure syndromes
- Impaired hematopoiesis + congenital anomalies + cancer predisposition
- Genomic instability or ribosomal dysfunction
- Fanconi’s anemia (FA)
  Dyskeratosis congenita (DC)
  Diamond-Blackfan anemia (DBA)
  Schwachman-Diamond syndrome (SDS)
<table>
<thead>
<tr>
<th>Disease</th>
<th>Haematological abnormalities</th>
<th>Age of onset</th>
<th>Congenital anomalies</th>
<th>Cancer predisposition</th>
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</thead>
<tbody>
<tr>
<td>FA</td>
<td>Aplastic anemia</td>
<td>6-7 years</td>
<td>Short stature</td>
<td>Acute leukaemia (AML)</td>
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<td></td>
<td></td>
<td></td>
<td>Radial &amp; thumb</td>
<td>Solid tumours</td>
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<td></td>
<td>anomalies</td>
<td>- squamous cell CA</td>
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<td></td>
<td>Pigmentation</td>
<td>of head &amp; neck &amp;</td>
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<td></td>
<td></td>
<td></td>
<td>Microcephaly</td>
<td>female genital tract</td>
</tr>
<tr>
<td>DC</td>
<td>Aplastic anemia</td>
<td>8-9 years</td>
<td>Dystrophic nails</td>
<td>Acute leukaemia</td>
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<td></td>
<td></td>
<td></td>
<td>Mucosal leukoplakia</td>
<td>Solid tumours</td>
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<td></td>
<td>Reticular rash</td>
<td>- squamous cell CA</td>
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<td>Short stature</td>
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<tr>
<td>DBA</td>
<td>Reticulocytopenic anemia</td>
<td>Infancy</td>
<td>Craniofacial anomaly</td>
<td>AML</td>
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<td>Radial ray</td>
<td>Osteosarcoma</td>
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<td></td>
<td>abnormality</td>
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<td>Cardiac defect</td>
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<td>SDS</td>
<td>Neutropenia</td>
<td>Infancy</td>
<td>Exocrine pancreatic</td>
<td>AML</td>
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<td></td>
<td>Aplastic anemia</td>
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<td>insufficiency</td>
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Bring home messages
• Symptoms of anemia in children may be non-specific
• Nutritional history is important, especially in infants and young children for possible iron deficiency
• Family and neonatal history are important for possible inherited causes of anemia (congenital marrow failure, intrinsic RBC defects, inborn error of metabolism)
• Associated physical and developmental abnormalities may provide useful clues for diagnosis (growth parameters, cutaneous or skeletal abnormalities, etc)
• A systemic approach should be adopted in laboratory investigations for the cause of anemia
• Red cell indices and reticulocyte response remain the most useful tools for evaluation of anemia
**Scenario**

- 20 month old child, noticed to be a bit pale
- Drinks a lot of whole cow’s milk since 9 months of age
- Hb 7, WBC 7.5, platelet 650
- Retic count 0.7%; MCV 60; RDW 21%
- PBS: microcytic, hypochromic
- Ferritin ↓

**DX:** Fe deficiency anemia
**Scenario**

- 8 year old girl, short, multiple café-au-lait spots and short thumbs
- Hb 8.5  MCV 105  WBC 3.5  Platelet 105
- Bone marrow: hypoplastic

DX: Fanconi’s anemia
Scenario

- 20 month old girl in good health; suffered from a cold 10 days ago; now pale
- Hb 7.0  MCV 82  WBC & Platelet - normal
- Retic count 0.2%

DX: Transient erythroblastopenia of childhood
Scenario

- 6 month old boy with pallor and poor appetite: both parents are China residents.
- P/E: pallor +ve; hepatosplenomegaly +ve
- Hb 6.0  Retic 10%  MCV 55  WBC/Platelet - normal
- PBS: target cells +++; hypochromia, microcytosis, polychromasia; basophilic stippling

DX: β-thalassaemia major
Scenario

- 5 month old boy with fever 39.5°C and lethargy
- P/E: pallor +++ & jaundice+; liver° spleen°
- Hb 5.5  MCV 83     Retic count 15%
- WBC 20     platelet 150
- Urine haemstix ++; RBC°
- PBS – spherocyte +; blister cells ++; bite cell+

DX: G6PD deficiency
**Scenario**

- 4 year old girl has pallor or/and mild jaundice on routine examination
- +ve family history of anemia in father who has a splenectomy and cholecystectomy done at 12 years of age
- P/E: pallor +ve, Jaundice +ve, spleen 4cm
- Hb 9.5  MCV 83  MCHC 39  WBC/Platelet - normal
- Retic count 9%
- PBS: microspherocytes ++; mild anisocytosis

DX: Hereditary spherocytosis
**Scenario**

- 8 year old girl with URTI; otherwise asymptomatic
- Routine CBC: Hb 11.5    MCV 68
  WBC/Platelet - normal
- Retic count 0.9%
- Hb pattern: HbA predominant; HbA₂ 4.5%
- Fe, TIBC, ferritin: normal

**DX:** β-thalassaemia trait
Scenario

- 7 year old boy with fever x 2 days
- CBC: Hb 11.5    MCV 66    WBC/Platelet - normal
- Retic count 0.8%
- Hb pattern: HbA; HbA$_2$ 2.8%
- Fe, TIBC, ferritin: normal

DX: $\alpha$-thalassaemia trait
Scenario

- 3 month old baby with thumb abnormalities seen for progressive pallor for 2 months
- Hb 7 MCV 120 WBC/Platelets - normal
- Retic count 0.1%
- Bone marrow: marked erythroid hypoplasia

DX: Pure red cell aplasia (Diamond-Blackfan anaemia)
Scenario

- 8 year old boy, known hereditary spherocytosis with baseline Hb of 9.5 & retic count ~ 12%
- Fever & mild rash x 2 days with increasing pallor and fatigue
- Hb 5, Retic count 2%, MCV 82
  WBC 2.5, Platelet 350

DX: Parvovirus infection with transient erythroid marrow aplasia