Childhood Anaemia

Anaemia in childhood is defined as a haemoglobin (Hb) concentration below established cut-off levels. These levels vary depending on the age of the child and on the laboratory in which the blood sample is tested. Reference ranges for specific laboratories and age groups should always be referred to. The World Health Organization (WHO) has suggested levels of Hb below which anaemia is said to be present. These levels are <11 g/dL in children aged 6-59 months, <11.5 g/dL in children aged 5-11 years and 12 g/dL in older children (aged 12-14). [1]

Epidemiology

Childhood anaemia poses a major public health issue leading to an increased risk of child mortality, as well as the negative consequences of iron-deficiency anaemia on cognitive and physical development. [2] At its special session on children in 2003, the United Nations General Assembly set a goal to reduce the prevalence of anaemia by one third by 2010. [2] Despite this, the incidence of anaemia in children aged under 5 between 1990-2010 has actually increased. [3]

Aetiology[4]

The likely cause of childhood anaemia varies depending on the area of the world that the child lives in. Overall, iron deficiency (usually because of diet) is the most common cause but, in the developing world, infectious diseases such as malaria, helminth infections, HIV and tuberculosis are also important. [3]

Inherited forms of anaemia are occasionally encountered. Diamond-Blackfan anaemia is a congenital hypoplastic anaemia that usually presents in infancy (an average of seven babies born each year). [5] Certain racial groups are more likely to have inherited anaemias than others; for example, sickle cell disease is more common in people of Central African origin whilst beta thalassaemias are more common in Mediterranean, Middle Eastern and Southeast Asian populations. [6]

Anaemia can be classified as:

Anaemia due to reduced red blood cell/haemoglobin production

- Bone marrow aplasia - Fanconi's anaemia (congenital aplastic anaemia), acquired aplastic anaemia, Diamond-Blackfan anaemia (red blood cell aplasia).
- Bone marrow replacement by tumour cells - leukaemias, secondary metastases.
- Bone marrow replacement by fibrous tissue or granulomas - granulomas can occur in the congenital toxoplasmosis, other, rubella, cytomegalovirus, herpes simplex (TORCH) infections or in tuberculosis infection.
- Deficiency of iron - there appears to be a current epidemic of iron deficiency in Western societies, particularly in the first two years of life in the inner cities. It is more common in Asian communities and is related to a poor food intake, the early introduction of whole cow’s milk and a high intake of fruit juice, often seen as a consequence of late weaning. [7] Iron deficiency is also prevalent in infants brought up in disadvantaged circumstances, who are predominantly breast-fed. [8] Always consider and screen for coeliac disease.
- Deficiency of folic acid - megaloblastic anaemia of infancy can develop due to folic acid deficiency during rapid growth. Folate deficiency can also occur in malabsorption syndromes such as coeliac disease, in inflammatory bowel disease and in children taking anticonvulsants.
- Deficiency of vitamin B12 - this can occur in infants who are breast-fed by a vegetarian mother, due to malabsorption or worm infestation. It can also (rarely) result from congenital pernicious anaemia where there is inability to secrete gastric intrinsic factor. [9]
- Thalassaemias - normal Hb is underproduced due to mutations in the alpha or beta globin chains.
• Anaemia of chronic disease (e.g., chronic pyelonephritis, chronic kidney disease, bacterial endocarditis, osteomyelitis) - due to impaired erythropoietin production. Anaemia can also be associated with hypothyroidism.

• Sideroblastic anaemia - hereditary sideroblastic anaemia is very rare.

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Anaemia due to increased red blood cell destruction (haemolysis)

• Genetic:
  • Red cell membrane defects - including hereditary spherocytosis.
  • Red cell enzyme abnormalities - including glucose-6-phosphate dehydrogenase (G6PD) deficiency, pyruvate kinase deficiency.
  • Haemoglobinopathies - including sickle cell disease, thalassaemias.

• Acquired:
  • Autoimmune haemolysis.
  • Isoimmune haemolysis (haemolytic disease of the newborn, blood transfusion reactions).
  • Infections (including malaria, septicaemia).
  • Drug- and toxin-induced.
  • Disseminated intravascular coagulation.
  • Hypersplenism.

Anaemia due to blood loss

• Including gastrointestinal blood loss and heavy menstruation in girls.

Presentation[4]

Symptoms

• May be asymptomatic.
• Fatigue.
• Shortness of breath.
• Left upper quadrant pain (if there is associated splenomegaly).
• Right upper quadrant pain (secondary to cholelithiasis in haemolytic anaemia).
• Failure to thrive.
• Symptoms related to underlying disease pathology - e.g., acute pain in sickle cell crises and chronic/recurrent diarrhoea, which can suggest a malabsorption syndrome such as coeliac disease.

Signs

• Cardiovascular system - look for exertional tachycardia, a systolic flow murmur. Gallop rhythm, cardiomegaly and hepatomegaly are signs of congestive cardiac failure.
• Plot weight, height and head circumference - in chronic anaemia, growth can be affected, usually with preservation of head circumference.
• Pallor - examine conjunctivae, nail beds, palmar creases.
• Petechiae and bruising - may be a sign of thrombocytopenia secondary to marrow aplasia or malignancy or of vasculitis.
• Splenomegaly - this occurs in chronic haemolysis and malignancy.
• Papulovesicular lesions on the feet - this may indicate hookworm infestation.
• Dysmorphic features of Fanconi's anaemia - small stature, small head, frontal bossing, absent thumbs, hyperpigmented skin.
• Jaundice - this may be present in haemolytic anaemias.

Points to consider in history taking[4]

• Ethnic origin.
• Evidence of blood loss - haemoptysis, melaena, haematuria, menorrhagia.
• Diet.
• Chronic infections - infections such as infective endocarditis and osteomyelitis can lead to anaemia of chronic disease.
• Drug history - certain drugs can trigger haemolysis in G6PD deficiency.
• Family history - for inherited anaemias.
Child's sex - eg, haemolytic anaemias are more likely in girls, G6PD deficiency is more common in boys.

Past medical history/neonatal history.

Recent travel.

Investigations\[^{10}\]

- **FBC** - including Hb and haematocrit levels, mean corpuscular volume (MCV), mean corpuscular Hb (MCH) and mean corpuscular Hb concentration (MCHC). A low MCV suggests iron deficiency, thalassaemia or lead poisoning. A high MCV suggests B12 or folate deficiency or reactive reticulocytosis which may be secondary to haemolysis. If the MCV is normal, anaemia may be due to: marrow failure, anaemia of chronic disease, haemolysis or mixed anaemia (a combination of B12 and iron deficiency).\[^{11}\] If there is reduced white cell and/or platelet count, this suggests bone marrow failure (aplasia or replacement).

- **Reticulocyte count** - these are immature red blood cells. If the reticulocyte count is high, it suggests active red blood cell production. A high reticulocyte count occurs in anaemia due to blood loss or haemolysis. The count varies with age, with particular variation in neonates. Normal ranges should be verified with your local laboratory. Care must be taken when interpreting reticulocyte count results. The normal reference value for reticulocytes is approximately 1%. However, the percentage reticulocyte count should be interpreted in conjunction with the Hb level and red cell count. For example, a result of 2-3% reticulocytes in a child with an Hb level half of the reference range does not indicate a reticulocyte response. The percentage either needs to be corrected for the degree of anaemia or the absolute reticulocyte cell count used.\[^{12}\]

- **Blood film** - this can show specific blood cell abnormalities - eg, sickle-shaped cells in sickle cell disease, spherocytes in hereditary spherocytosis, ghost or bite cells in G6PD deficiency. Thick films may show malaria parasites.

- **Haemoglobin electrophoresis** - this confirms haemoglobinopathies.

- **Red cell enzyme studies** - show G6PD and pyruvate kinase deficiency.

- **Coombs' test** - identifies autoimmune haemolytic anaemia.

- **Folate, vitamin B12 levels** - may highlight these deficiencies in macrocytic anaemia.

- **Iron, ferritin and total iron binding capacity levels** - can confirm iron-deficiency anaemia.

- **Endomysial antibodies** - if coeliac disease is suspected.

- **Bilirubin and lactate dehydrogenase levels** - are raised in haemolytic anaemia.

- **TFTs** - excludes hypothyroidism.

- **Other diagnostic tests** - tests, such as bone marrow biopsy, can show specific causes - for example, marrow infiltration by tumour cells.

Management

This depends on the underlying cause. Transfusion is only required if the child is compromised and on the verge of high-output cardiac failure. Care needs to be taken to avoid iron overload.

Prognosis

Most children have a good capacity for tolerating low Hb levels and the morbidity of childhood anaemia is usually related to the primary disease process rather than the anaemia itself.

Prevention\[^{13}\]

As iron deficiency is the most common cause of childhood anaemia in the Western world, the main focus for prevention of this needs to be on education around childhood nutrition. The Child Health Subgroup of the National Screening Committee undertook a review in 2013 and concluded that screening of iron deficiency in children aged under 5 should not be recommended. They found no causal relationship between iron deficiency and adverse developmental outcomes. Moreover, they considered the evidence supporting iron supplementation in asymptomatic children to be conflicting. They recommended that emphasis should continue to be placed on primary prevention through dietary advice.

Further reading & references

12. Reticulocyte Count; Lab Tests Online, 2013
13. The UK NSC policy on Iron Deficiency Anaemia screening in children under 5 years of age; UK National Screening Committee, 2013

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