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Review Article

Categorization of different forms of Anemia (*Faqr al-Dam*) among children with an emphasis on iron deficiency anemia: A review

* Dr. Safder Husain[1], Dr. Chand Bibi [2], Dr. Arish Mohammad Khan Sherwani [3], Dr. Ziaul Haq Siddiqui [4]

*1. Research Associate, Regional Research Institute of Unani Medicine under CCRUM- New Delhi, Ministry of AYUSH, Govt. of India

2. MD, Dept. of Tahaffuzi Wa SamajiTib, National Institute of Unani Medicine, Bengaluru, India

3. Professor and HoD, Department of Tahaffuzi Wa Samaji Tib, National Institute of Unani Medicine. Bengaluru, Karnataka, India

4. Deputy Director In charge, Regional Research Institute of Unani Medicine, under CCRUM- New Delhi, Ministry of AYUSH, Govt. of India

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*For Correspondence:

Dr. Safder Husain, Research Associate, Regional Research Institute of Unani Medicine under CCRUM- New Delhi, Ministry of AYUSH, Govt. of India

Abstract

Background and Objectives: Anemia, characterized by decreased hemoglobin (Hb) levels in red blood cells (RBCs), remains a significant public health issue. Globally, the prevalence of anemia among children under five years has decreased from 48% to 39.8% but has stagnated since 2010. In traditional Unani Medicine, anemia is referred to as *Faqr al-Dam*, literally meaning "shortage of blood." Anemia classifications depend on morphological, functional, and etiological factors, with iron deficiency anemia (IDA) recognized as the most common nutritional anemia among children. This review aims to highlight the importance of iron, its dietary sources, and nutritional requirements to prevent iron deficiency anemia across various age groups, alongside providing guidance on identification and diagnosis.

Methods: Relevant data were gathered through a comprehensive literature search of databases including Web of Science, PubMed, Google Scholar, and Scopus.

Results: Anemia significantly impacts children, adults, pregnant women, and women of childbearing age. Given the slow progress in anemia reduction, especially among women, the World Health Assembly (WHA) has set a global target of halving anemia prevalence by 2030. Effective clinical management and public health interventions addressing IDA can substantially improve health outcomes and quality of life.

Conclusion: This review provides insights into various anemia types, emphasizing iron deficiency anemia, its diagnosis, and preventive strategies. Despite extensive global research on anemia, critical gaps remain, necessitating future studies on iron absorption mechanisms, the long-term cognitive impacts of iron deficiency, and dietary influences on iron homeostasis.

Keywords: Anemia, *Faqr al-Dam*, Iron deficiency, Iron homeostasis, Unani Medicine

Introduction

Anemia is a hematological disorder characterized by diminished capacity of the blood to transport oxygen.[1] its prevalence is notably high among school-aged children, adults, women of childbearing age, and pregnant women. The World Health Organization (WHO) defines anemia as hemoglobin (Hb) levels below 12.0 g/dL in women and below 13.0 g/dL in men.[2]Anemia exerts a substantial adverse effect on the developmental processes of children and increases the likelihood of child mortality. Iron deficiency anemia ranks as the third most prevalent cause of disability globally and is the 13th most significant risk factor for disability-adjusted life years (DALY).[3]Vitamins thiamine (B1), riboflavin (B2), pyridoxine (B6), folate (B9), and cyanocobalamin (B12) play crucial roles in hemoglobin production and iron metabolism. Along with iron and vitamin B12, folate (folic acid) is a vital component of human erythropoiesis. Despite its abundance in foods, particularly in green leafy vegetables, dietary folate deficiency remains the leading cause of megaloblastic anemia globally. A deficiency in folate prolongs the synthesis phase of cell division and slows germ cell maturation, resulting in abnormal red cell precursors (megaloblasts) characterized by larger than normal cell and nuclear diameters in the bone marrow.[4] Iron deficiency anemia (IDA) represents one of the most prevalent causes of morbidity and mortality across both industrialized and developing nations, impacting individuals of all age groups.[5]The global prevalence of anemia among children aged 6–59 months was 39.8%, corresponding to 269 million children diagnosed with anemia in 2019. At the age of five, the highest incidence of anemia has been observed in Africa, with a prevalence of 60.2% since 2000. Iron deficiency is the most prevalent cause of anemia and iron supplementation is the most common treatment. Vitamin C is an effective reducing agent that enhances ferrous iron absorption. For the treatment of iron deficiency anemia (IDA), a combination of iron supplements and vitamin C is strongly recommended.[6] Vitamin C supplementation enhances iron absorption; however, its impact is minimal in individuals who consume a healthy, balanced diet.[7] Vitamin D deficiency is among the most prevalent nutritional deficiencies and undiagnosed medical conditions globally. In the Indian subcontinent, this deficiency affects 50-90% of young children. Numerous studies have indicated that a deficiency in 25 hydroxyvitamin (OH) D is associated with an elevated risk of anemia, a significant public health concern affecting up to 50% of Indian children.[8] Anemia in both children and adults can be categorized based on several criteria, including morphological, functional, and pathophysiological aspects.[9]

Morphological classification of Anemia

Table 1: Anemia is categorized as follows based on RBC morphology.

Microcytic hypochromic	**Macrocytic and dimorphic Anemia**	**Normocytic normochromic**
Iron deficiency	Megaloblastic Anemia	Acute blood loss
Anemia of chronic disease	Pernicious Anemia	Hemolytic Anemia
Thalassemia (more common in certain groups)	Vitamin B_{12} deficiency	Hereditary spherocytosis
	Folate deficiency	Sickle cell disease (most common in sub-Saharan Africa)
	Liver disease	G6PD deficiency (varies with ethnicity)
	Marrow infiltration	Autoimmune hemolytic anemia
		Malaria
		Bone marrow failure (e.g., aplastic anemia, leukemia)
		Pregnancy.[4, 8]

Pathophysiological classification of anemia

Table 2: Shows pathophysiological classification of anemia.

Anemia due to Blood loss	**Increased red cell destruction (Hemolysis)**	**Decreased red cell production**
Acute blood loss (e.g., trauma)	Inherited genetic defects	Nutritional deficiencies
Chronic blood loss (e.g., GI tract lesions, gynecological disturbances)	Red cell membrane disorders (e.g., hereditary spherocytosis)	Deficiencies affecting DNA synthesis (e.g., B12and folate deficiencies)
	Enzyme deficiency (e.g., G6PD deficiency)	Deficiencies affecting hemoglobin synthesis (e.g., iron deficiency anemia).
	Hemoglobin abnormalities	Erythropoietin deficiencies (e.g., renal failure, anemia of chronic disease)
	Deficient globin synthesis (e.g., Thalassemia syndromes)	Immune-mediated injury of progenitors (e.g., Aplastic anemia, pure red cell aplasia)
	Structurally abnormal globin (e.g., sickle cell anemia)	Inflammation-mediated iron sequestration (e.g., anemia of chronic disease).[4]
	Infections of red cell (e.g., Malaria, babesiosis)	
	Toxic or chemical injury (e.g., lead poisoning)	

According to Unani concepts, anemia or *Sū'al-Qinya* occurs with either change in bodily *mijaz* (temperament) or when there is an abnormal humeral imbalance. The phrase *Faqr al-Dam* is a term that was coined in the 20 centuries. It means "shortage of blood". The Arabic word *"Sū 'al-Qinya"* refers to a shortage, change or deficiency of iron. According to the Unani system of medicine, *Sū'al-Qinya* is either caused by *"Ḍu'f al-jigar"* (weakness of the liver) or by "Sū'-i-Mizāj" (a change in an individual's temperament or temperament of the liver). [10]

Types of Anaemia in the Unani System of Medicine

- *Sawiul kurriyatee* (Normocytic anemia)
- *Kurria kibriya* (Macrocytic anemia)
- *Fauladi* (Hypochromic anemia) [11]

Etiological Factors *(Asbab)*

The following causes of anemia have been described in the Unani ancient literature:

- *Amrāḍ-i-kabid* (Liver Disorders)
- *Amrāḍ-i-ṭiḥāl* (Splenic Disorders)
- *Amrāḍ-i-mi'da-o-am'ā* (Gastrointestinal Disorders)
- *Amrāḍ-e-gurda* (kidney diseases)
- *Amrāḍ-i-Sadr* (Cardio-pulmonary diseases) [11]

Etiology of Anemia in Children

Anemia is characterized by a multitude of complex etiologies. Among the various factors contributing to anemia within a population, iron deficiency is the most prevalent, affecting children and individuals across all age groups.[4] Iron deficiency anemia is the most prevalent nutritional form of anemia in children. Red blood cells, characterized by low mean corpuscular volume (MCV), low mean corpuscular hemoglobin (MCH), and low reticulocyte hemoglobin content, are classified as hypochromic and microcytic. Iron content in the blood decreases.[12] Iron, folic acid, and vitamin B12 are essential nutrients for hemoglobin synthesis. Insufficient dietary iron intake is the predominant cause of iron deficiency anemia in infants and children.[13] Iron is an essential nutrient for the growth and optimal functioning of various organs and systems, particularly during erythropoiesis. Consequently, they must be ingested and absorbed by the upper gastrointestinal tract. Iron deficiency arises when the body's iron requirements are unmet, such as when the equilibrium between iron intake, storage, and loss is inadequate to support erythrocyte formation. This condition is known as iron deficiency.[14]

Iron

Iron plays a crucial role in various physiological processes, particularly in hemoglobin production. It is an essential component of hemoglobin in red blood cells and myoglobin in muscle tissues, underscoring its importance in human nutrition. The adult human body typically contains 3–4 g of iron, with approximately 60-70% found in the blood as hemoglobin-bound iron, while the remaining 1–1.5 grams is stored within the body. Notably, each gram of hemoglobin contains approximately 3.34 milligrams of iron. As individuals transition from pre-adolescence to adolescence, their iron requirements increase significantly, rising from 0.7-0.9 milligrams per day to 1.4-1.9 milligrams per day for boys and 1.4-3.3 milligrams per day for girls. Table 1 shows the daily iron requirements of children and adolescents of various ages.[15]

Table 3: Shows the Daily Iron requirement

Age Groups		Daily iron need (mg/dl)
Children		
1-3 years		0.45
4-6 years		0.63
7-9 years		0.77
Adolescence		
Boys	10-12 years	1.05
Girls	10-12 years	1.33
Boys	13-15 year	1.60
Girls	13-15 year	1.36
Boys	16-17 years	1.37
Girls	16-17 years	1.30

Sources of Iron

Iron exists in two forms: haem and non- haem. Heme iron is absorbed more efficiently than non-heme iron is. Foods rich in heme iron include the liver, meat, poultry, and fish, which not only serve as significant sources of readily available iron but also enhance the absorption of non-heme iron from plant-based foods consumed concurrently. Non-heme iron is found in foods of vegetable origin, such as cereals, green leafy vegetables, legumes, nuts, oilseeds, jaggery, and dried fruits.[13] Notably, non-heme iron constitutes approximately 90% of the iron in the diets of infants and young children. The absorption of non-heme iron is facilitated by ascorbic acid as well as by beef, fish, and poultry.[16]

Table 4: Iron sources (heme-iron and non-hemeiron).

Non-heme iron (vegetable Sources)	Heme-iron (Non-vegetable Sources)
Cereals,	Liver
Green leafy vegetables	Meat
Legumes	Poultry
Nuts, and dried fruits	Fish
Jaggery	Other

Iron metabolism

Absorption: Mature villus enterocytes located in the duodenum and proximal jejunum are responsible for the absorption of dietary iron, which exists in both heme and non-heme forms.[17] Intestinal mucosal absorptive cells, known as enterocytes, are derived from undifferentiated progenitor cells situated in the crypts of Lieberkühn. These cells then migrate upwards along the villi over a period of 2 to 3 days, eventually being shed through exfoliation and undergoing apoptosis.[18] Divalent metal transporter 1, a member of the solute carrier family of membrane transport proteins, is responsible for the absorption of iron in enterocytes. This process primarily occurs in the duodenum and upper jejunum.

Regulation of iron homeostasis: Maintaining iron homeostasis is crucial, because iron is essential for various cellular activities. Achieving this balance requires careful regulation of iron intake, transport, storage, and utilization.[20] Hepcidin is a peptide hormone secreted by the liver that plays a crucial role in the regulation of iron homeostasis. It serves as the primary regulator of systemic iron homeostasis by coordinating the uptake, utilization, and storage of iron. Hepatocytes produce this hormone, which acts as a negative regulator of plasma iron absorption. [21]

Iron transport: Transferrin, a plasma iron-binding glycoprotein, is a crucial iron-specific transport protein that facilitates the movement of iron between various sites. It is predominantly present in plasma at a concentration of approximately 200 mg/dl. While transferrin is primarily synthesized by parenchymal cells in the liver, it is also produced by macrophages within the lymphoid tissue. The measurement of transferrin is typically expressed in terms of its iron-binding capacity, known as the "total iron binding capacity" (TIBC). Transferrin plays a vital role in the transport of iron within the plasma and in the transfer of iron from the plasma to developing erythrocytes.[22]

Iron storage: Ferritin and hemosiderin concentrations serve as indicators of iron storage within the body. These proteins are predominantly located in the liver, spleen, and bone marrow, where they store iron in an insoluble form.[23] The predominant portion of iron is stored in ferritin, an iron-binding protein that is both widely distributed and highly conserved across species.[24] Hemosiderin is an iron storage complex that releases iron at a slower rate than the body requires. Under steady-state conditions, serum ferritin levels exhibit a strong correlation with total body iron stores.[25] Consequently, serum ferritin is the most convenient laboratory test for estimating iron stores.

Excretion of iron: Beyond iron loss associated with menstruation, various forms of bleeding, or pregnancy, the body effectively conserves iron, making its loss uncommon. The physiological shedding of cells from epithelial surfaces contributes to necessary iron loss from the body, including the skin, genitourinary tract, and gastrointestinal tract.[26, 27] During periods of rapid growth, such as infancy and puberty, iron levels increase and may surpass the body's absorption capacity. During pregnancy, iron is transferred to the fetus and placenta, and the maternal red blood cell mass increases. Additionally, iron is lost during childbirth because of hemorrhage.[28]

Diagnostic Approach

Anemia in children is often asymptomatic and is typically identified through laboratory screening evaluations. Screening is advised exclusively in high-risk children. Laboratory tests employed in the diagnosis of anemia

Subsequently, iron is transferred across the duodenal mucosa into the bloodstream, where it is transported to cells or the bone marrow for erythropoiesis (production of red blood cells [RBCs]) via transferrin.[19]

include the measurement of ferritin, which indicates iron storage, and transferrin or total iron-binding capacity (TIBC), which reflects the body's capacity to transport iron for red blood cell production.[29] The assessment of iron status was conducted using the following parameters.

Hemoglobin concentration is a relatively insensitive indicator of nutrient depletion. Its value tends to be lower in population groups in which anemia is not severe. This is because anemia is a late manifestation of iron deficiency that often occurs without anemia.[30]

Serum iron concentration serves as a more informative index than hemoglobin concentration, with a normal range of 0.80 to 1.80 mg/L. Values below 0.50 mg/L suggest probable iron deficiency.[31]

Serum ferritin is the most sensitive measure for assessing iron status, reflecting the size of iron stores in the body. In populations with a low iron insufficiency prevalence, it is the most effective indicator of iron status. Values below 10-12 μg/L likely indicate the absence of stored iron.[32] Serum transferrin saturation should exceed 16%, with a normal value of 30%.[15] **Hematocrit and red blood cell indices** such as hemoglobin or hematocrit concentration are the most commonly employed screening methods for detecting iron deficiency anemia in populations.[33] The hematocrit (PCV-packed cell volume) cut-off value is (40±3%).[34] Various tests, including hemoglobin concentration, mean corpuscular hemoglobin concentration (MCHC), MCV, RDW, TIBC, serum ferritin, serum transferrin receptor (TFR), and bone marrow biopsy, can diagnose iron deficiency, although they have limitations. Chronic iron deficiency may result in low hemoglobin levels, which are influenced by health status, and ferritin levels can increase for various reasons. Erythrocyte indices (MCV, MCHC, and RDW) assess iron availability in mature blood cells, although they are slow and insensitive early indicators. The serum TFR test is often prohibitively expensive, and bone marrow biopsy is too invasive for routine testing.[35] The size of RBCs, indicated by MCV, classifies anemia: microcytic anemia (MCV < 80 FL), normocytic anemia (MCV 80-100 FL), and macrocytic anemia (MCV > 100 FL). The RBC distribution width measures the size variance, with a low width indicating a uniform cell size, and a wide width (>14%) indicating varied sizes.[36]

Leukocyte count is typically normal, although slight absolute granulocytopenia may occur in long-standing cases.[37] Anemia and eosinophilia are common in hookworm infestations.[38] Platelet count may also be affected by iron deficiency, with most patients exhibiting normal or elevated counts, sometimes exceeding > 1000 × 109/L at diagnosis.[39] Bone marrow aspiration is necessary to assess normoblastic, megaloblastic, or sideroblastic erythropoiesis and to rule out marrow pathology, examining erythroid, myeloid, and megakaryocytic morphology (e.g., aplastic anemia, leukemia, and benign or malignant marrow infiltration).[40]

Method: Data were sourced from a range of books and journals, including the Web of Science, PubMed, Google Scholar, and Scopus.

Conclusion

Anemia is a significant clinical condition affecting a substantial proportion of children, adults, pregnant women, and women of childbearing age. The global prevalence of anemia among children aged 6–59 months was 39.8%, corresponding to 269 million children diagnosed with anemia in 2019.[41] The World Health Assembly's global nutrition target to reduce the prevalence of anemia by 50% by 2030 is not being achieved, primarily because of insufficient progress among women aged 15–49 years globally, regionally, and in nearly all countries.[42]Additionally, the incidence of anemia in children remains elevated. Various diagnostic tests, including mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and serum ferritin levels, are available in modern medicine. However, a significant portion of the Indian population belongs to low socioeconomic groups, which may limit their access to affordable diagnostic tests.[11] Considering the prevalence of anemia across different age groups, particularly iron-deficiency anemia, further research is needed in areas such as the mechanisms of iron absorption and utilization, long-term effects of iron deficiency on cognitive development, and influence of diet on iron status. Future studies should focus on understanding the molecular mechanisms of iron metabolism, long-term consequences of iron deficiencies, and development of more effective public health prevention and treatment strategies. Addressing iron deficiency anemia through clinical and public health interventions has the potential to significantly enhance overall health outcomes and quality of life.

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