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Anemia of Inflammation vs. Iron-Deficiency Anemia: Diagnostic Challenges and Overlapping Mechanisms

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ABSTRACT

Anemia of inflammation (AI) and iron-deficiency anemia (IDA) are two common causes of anemia that frequently pose diagnostic challenges due to their overlapping clinical and laboratory features. AI, often associated with chronic infections, autoimmune disorders, and malignancies, is primarily driven by the dysregulation of iron homeostasis and inflammatory cytokines, particularly hepcidin. IDA, on the other hand, results from inadequate dietary intake, malabsorption, or chronic blood loss, leading to depleted iron stores. Distinguishing between AI and IDA is critical for effective management but remains complex due to shared laboratory findings, such as low serum iron levels and microcytic hypochromic anemia. This review explores the pathophysiology of AI and IDA, their overlapping mechanisms, and the key diagnostic markers that differentiate them. We also discuss emerging biomarkers, such as soluble transferrin receptor (sTfR) and hepcidin, and the role of therapeutic strategies, including iron supplementation and anti-inflammatory interventions. Improved diagnostic approaches are essential for guiding targeted treatment strategies and optimizing patient outcomes.

Keywords: Anemia of inflammation, Iron-deficiency anemia, Hepcidin, Soluble transferrin receptor, Microcytic anemia, Iron metabolism, Diagnostic biomarkers

INTRODUCTION

Anemia is a major global health burden that affects millions of individuals across diverse demographic groups, with significant implications for public health and clinical practice[1, 2]. Defined as a reduction in the number of red blood cells (RBCs) or hemoglobin concentration below the normal physiological levels required for adequate oxygen transport, anemia presents a wide spectrum of clinical manifestations ranging from fatigue and weakness to severe cardiovascular complications[3–5]. Among the various forms of anemia, iron-deficiency anemia (IDA) and anemia of inflammation (AI) stand out as the most prevalent subtypes, contributing substantially to morbidity and mortality worldwide[1]. Iron-deficiency anemia, as the name suggests, arises due to an absolute deficiency of iron, a critical micronutrient essential for hemoglobin synthesis, cellular respiration, and enzymatic functions[1, 6]. IDA can result from various factors, including inadequate dietary intake, chronic blood loss, malabsorption syndromes, or increased physiological demands such as those seen in pregnancy and rapid growth phases[7]. The hallmark of IDA is a depletion of body iron stores, leading to microcytic, hypochromic red blood cells and reduced oxygen-carrying capacity[7]. In contrast, anemia of inflammation, also known as anemia of chronic disease (ACD), is primarily driven by inflammatory-mediated alterations in iron metabolism. AI is commonly observed in patients suffering from chronic infections, autoimmune disorders, malignancies, and other persistent inflammatory conditions[1]. Unlike IDA, AI is not necessarily due to a lack of iron but rather a dysregulation of iron homeostasis mediated by inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α)[8]. These cytokines upregulate the production of hepcidin, a key regulator of iron metabolism, which inhibits iron absorption from the intestine and promotes iron sequestration within macrophages, thereby reducing iron availability for erythropoiesis[9]. Despite the distinct etiologies of IDA and AI, both conditions share overlapping clinical and hematological features, making differential diagnosis challenging. Patients with either condition may present with similar symptoms, including pallor, fatigue, dyspnea, and cognitive impairment. Traditional hematological parameters such as hemoglobin levels, mean corpuscular volume (MCV), and serum iron concentration may not always provide a clear distinction between IDA and AI[10–12]. The complexity of diagnosing these conditions necessitates a comprehensive approach that includes a detailed patient history, biochemical markers (such as

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ferritin, transferrin saturation, and soluble transferrin receptor), and emerging molecular and imaging techniques[13]. Treatment strategies for IDA and AI also differ significantly due to their distinct underlying mechanisms. The management of IDA primarily involves iron supplementation through oral or intravenous formulations, dietary modifications, and addressing underlying causes such as gastrointestinal bleeding or menstrual losses[14, 15]. Conversely, AI management is more complex, requiring a multifaceted approach that includes controlling the underlying inflammatory condition, modulating hepcidin activity, and, in select cases, considering erythropoiesis-stimulating agents (ESAs) and iron therapy. Recent advancements in therapeutic approaches have explored novel pharmacological agents targeting the hepcidin-ferroportin axis, thereby offering new avenues for treatment[16, 17]. Given the widespread prevalence and significant clinical impact of IDA and AI, there is an urgent need for continued research to enhance our understanding of their pathophysiology, improve diagnostic precision, and develop more effective treatment strategies. This review aims to provide an in-depth analysis of the underlying mechanisms, diagnostic challenges, and therapeutic options for these two prevalent forms of anemia, highlighting recent advancements and future perspectives in anemia research and management.

Pathophysiology of AI and IDA

Iron-Deficiency Anemia (IDA)

Iron deficiency anemia (IDA) is a condition where iron availability is insufficient to support erythropoiesis, the process of red blood cell (RBC) production. This deficiency results in impaired oxygen transport throughout the body and can be categorized into dietary insufficiency, chronic blood loss, malabsorption disorders, and increased physiological demands[1]. Inadequate dietary intake, chronic blood loss, malabsorption syndromes, and increased physiological demands are the primary causes of IDA. Individuals with poor dietary habits, vegetarians, and vegans may have a higher risk due to the lower bioavailability of non-heme iron compared to heme iron[18]. Chronic blood loss can occur due to conditions such as peptic ulcers, gastritis, colorectal cancer, and inflammatory bowel disease. Heavy menstruation, frequent blood donation, and malabsorption syndromes can also contribute to IDA[18]. Malabsorption syndromes include celiac disease, bariatric surgery, and the chronic use of proton pump inhibitors (PPIs). Increased physiological demands during pregnancy, growth spurts, and intense physical activity can lead to iron loss through sweat, hemolysis, and increased metabolic demands[19]. The hallmark of IDA is depleted iron stores, leading to reduced hemoglobin synthesis and impaired erythropoiesis. RBCs become microcytic and hypochromic, resulting in decreased oxygen-carrying capacity and symptoms like fatigue, pallor, shortness of breath, dizziness, and cognitive impairment[19]. Hepcidin, a liver-derived hormone controlling intestinal iron absorption and release from macrophages, is affected in IDA, which decreases to enhance iron absorption from the diet and promote its mobilization from storage sites.[16] Diagnosis and management of IDA typically involve laboratory tests such as complete blood count (CBC), serum ferritin, and serum iron and total iron-binding capacity (TIBC). Management includes addressing the underlying cause and replenishing iron stores through dietary modifications, oral or intravenous iron supplementation, and, in severe cases, blood transfusion.

Anemia of Inflammation (AI)

Anemia of inflammation (AI), also referred to as anemia of chronic disease (ACD), is a common form of anemia associated with chronic medical conditions such as infections, autoimmune diseases (e.g., rheumatoid arthritis, systemic lupus erythematosus), chronic kidney disease, and malignancies[1]. AI is primarily driven by persistent immune activation and systemic inflammation, which disrupts normal iron metabolism and erythropoiesis. AI is a complex disease characterized by increased production of inflammatory cytokines, iron regulation, and bone marrow function[20]. These cytokines include Interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ), which stimulate hepcidin production and inhibit erythropoiesis. Hepcidin, a liver-derived hormone, is upregulated in response to IL-6 and regulates iron homeostasis. It binds to and degrades ferroportin, an iron exporter on macrophages and enterocytes, leading to reduced dietary iron absorption and sequestration of iron within macrophages and the reticuloendothelial system, resulting in functional iron deficiency[21, 22]. Chronic inflammation also affects erythropoiesis and erythropoietin (EPO) resistance. The kidneys' production of EPO is blunted by inflammation, and erythroid progenitor cells in the bone marrow exhibit reduced responsiveness[23]. TNF-α and IFN-γ contribute to ineffective erythropoiesis by inducing apoptosis of erythroid precursors. AI is typically normocytic or microcytic anemia, with low serum iron, total iron-binding capacity, ferritin levels, and transferrin saturation[24]. These mechanisms contribute to the pathophysiology of AI and its impact on bone marrow function.

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Table 1: Distinguishing AI from Iron Deficiency Anemia (IDA)

Parameter	Anemia of Inflammation (AI)	Iron Deficiency Anemia (IDA)
Serum iron	↓ Low	↓ Low
Total iron-binding capacity (TIBC)	↓ Low or normal	↑ Increased
Ferritin	↑ Normal or high	↓ Low
Transferrin saturation	↓ Low	↓ Low
Bone marrow iron stores	↑ Present or increased	↓ Depleted

AI is a complex inflammation-driven anemia characterized by iron sequestration, impaired erythropoiesis, and resistance to erythropoietin. It differs from autoimmune disease (IDA) in that iron stores are preserved or elevated but functionally unavailable for red blood cell production[10]. Effective management involves treating the underlying inflammatory condition and, in some cases, using targeted therapies to correct iron dysregulation and enhance erythropoiesis. Recombinant EPO may be beneficial in patients with chronic kidney disease or cancer. Intravenous iron may be considered in selected cases. Emerging therapies targeting IL-6, hepcidin, or ferroportin may offer novel treatment options in the future.

Overlapping Mechanisms and Diagnostic Challenges

Both AI and IDA present with microcytic anemia and low serum iron levels, complicating diagnosis. However, key differences exist:

Table 2: Overlapping Mechanisms and Diagnostic Challenge

Biomarker	IDA	AI
Serum iron	↓ Decreased	↓ Decreased
Ferritin	↓ Decreased	↑ Normal or increased
Total iron-binding capacity (TIBC)	↑ Increased	↓ Decreased
Soluble transferrin receptor (sTfR)	↑ Increased	↓ Normal or decreased
Hepcidin	↓ Decreased	↑ Increased
C-reactive protein (CRP)	Normal	↑ Increased

Ferritin, a marker of iron stores, is elevated in AI due to inflammation but low in IDA. The soluble transferrin receptor (sTfR) is increased in IDA but normal or reduced in AI. Hepcidin is a distinguishing factor, being suppressed in IDA and elevated in AI. These differences aid in differential diagnosis[25].

Emerging Biomarkers and Diagnostic Approaches

Novel biomarkers and advanced diagnostic tools have been developed to differentiate between iron deficiency anemia (IDA) and anemia of inflammation (AI), conditions with overlapping clinical features[26]. These biomarkers include Reticulocyte Hemoglobin Content (CHr), a direct measure of hemoglobin content in reticulocytes, which is a sensitive indicator of functional iron deficiency in IDA[27]. In contrast, CHr remains normal or mildly reduced in AI because iron is sequestered rather than deficient. Hepcidin assays, a key regulator of iron homeostasis, play a pivotal role in differentiating AI from IDA. In AI, inflammatory cytokines (e.g., IL-6) upregulate hepcidin expression, leading to decreased iron absorption and sequestration in macrophages, whereas in AI, hepcidin levels are typically suppressed, allowing increased iron mobilization from stores and enhanced intestinal absorption[28]. Measuring serum hepcidin levels can help distinguish between iron-restricted erythropoiesis in AI and true iron deficiency, aiding in more targeted management[28]. Iron isotope studies provide a detailed assessment of iron metabolism by tracking iron absorption, utilization, and sequestration dynamics. These insights are valuable in research settings and may have potential for future clinical applications[29]. Magnetic resonance imaging (MRI)-based quantification of iron stores has emerged as a non-invasive and highly accurate tool for assessing iron distribution in the body, particularly in cases where traditional biochemical markers yield ambiguous results[30]. MRI-based hepatic iron quantification helps distinguish between absolute iron deficiency (low hepatic iron stores) and iron sequestration (high or normal hepatic iron stores) in complex cases like chronic kidney disease, chronic infections, and inflammatory disorders. By integrating these novel biomarkers and imaging techniques, clinicians can achieve a more precise diagnosis of anemia subtypes, leading to more personalized and effective treatment approaches[30].

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Treatment Strategies

Management of IDA

Iron deficiency anemia (IDA) is a condition that requires replenishing iron stores, treating the underlying cause, and preventing recurrence. The treatment approach depends on the severity of anemia, the patient's ability to tolerate oral iron, and the presence of any underlying conditions affecting iron absorption or loss[31]. Oral iron therapy is the first-line treatment for IDA in most patients, with common formulations including ferrous sulfate, ferrous gluconate, and ferrous fumarate[31]. To enhance absorption, iron supplements should be taken on an empty stomach with vitamin C or orange juice. Common side effects include gastrointestinal discomfort, nausea, constipation, and dark stools, which can be managed by reducing the dose, switching to a different formulation, or taking iron with food. Slow-release and enteric-coated iron formulations may have fewer gastrointestinal side effects but are less efficiently absorbed.Intravenous iron therapy is an alternative for patients who cannot tolerate oral iron or have severe anemia requiring rapid correction. Common IV iron formulations include iron sucrose, ferric carboxymaltose, and iron dextran[32]. However, these formulations have risks of hypersensitivity reactions, transient flu-like symptoms, and hypotension, and require hospital or outpatient clinic administration, increasing healthcare costs. Successful management of IDA involves identifying and treating the underlying conditions contributing to iron deficiency, such as gastrointestinal disorders, menstrual blood loss, dietary modifications, and parasitic infections[32]. By addressing these factors alongside iron supplementation, the recurrence of IDA can be minimized, ensuring long-term management success.

Management of AI

Anemia of inflammation (AI), also known as anemia of chronic disease (ACD), is a common condition associated with chronic infections, autoimmune diseases, cancer, and chronic kidney disease (CKD)[33]. The management of AI focuses on addressing the underlying inflammation, correcting iron homeostasis, and promoting erythropoiesis. Key therapeutic strategies include anti-inflammatory therapy, biologic agents like TNF inhibitors, IL-6 inhibitors, JAK inhibitors, immunosuppressive therapy, and iron supplementation in select cases. Iron homeostasis is disrupted in AI due to increased hepcidin levels, leading to iron sequestration and reduced availability for erythropoiesis[33]. Intranavenous (IV) iron therapy is preferred over oral iron due to poor gastrointestinal absorption caused by elevated hepcidin levels. Oral iron therapy may be considered in patients with mild anemia and low hepcidin levels. Erythropoiesis-stimulating agents (ESAs) are used in chronic kidney disease (CKD) patients to stimulate erythropoiesis. Recombinant ESAs, such as Epoetin alfa and darbepoetin alfa, stimulate erythropoiesis and are the standard of care for AI in CKD patients[34]. HIF-PHIs, such as roxadustat, vadadustat, and daprodustat, mimic hypoxia, increasing endogenous erythropoietin production and enhancing iron availability by reducing hepcidin levels. Hepcidin-targeting therapies under investigation aim to reduce hepcidin levels and enhance iron availability. Hepcidin antagonists, liver-targeted hepcidin inhibitors, matriptase-2 agonists, and anti-IL-6 therapies may indirectly improve iron metabolism[35]. Future directions include gene therapy approaches targeting iron-regulatory genes, combination therapies involving ESAs, iron supplementation, and anti-inflammatory agents, and artificial intelligence and personalized medicine to tailor therapy based on individual patient profiles.Overall, AI management requires a multifaceted approach tailored to the underlying disease, iron homeostasis, and erythropoietic function. Advances in hepcidin-targeting therapies and HIF-PHIs offer promising alternatives for refractory cases.

CONCLUSION

AI and IDA share several clinical and laboratory features, making differentiation challenging. However, distinct biomarkers, including ferritin, sTfR, and hepcidin, can aid in accurate diagnosis. While iron supplementation remains the cornerstone of IDA treatment, AI management requires addressing the underlying inflammatory state. Advances in diagnostic biomarkers and targeted therapies hold promise for improving patient outcomes. Further research is needed to refine strategies for distinguishing and treating these common forms of anemia.

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