

ANEMIA IN CHRONIC DISEASES: PATHOGENESIS, DIAGNOSIS AND CLINICAL RELEVANCE

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Abstract. *Anemia that occurs in infectious and inflammatory processes, non-infectious inflammatory diseases, and tumors is termed anemia of chronic disease (ACD). Despite the diverse mechanisms underlying the development of anemia in these conditions, a central factor is the redistribution of iron into macrophages activated during inflammatory or neoplastic processes. ACD ranks second in prevalence after iron deficiency anemia (IDA). Distinguishing ACD from IDA and sideroachrestic anemias is crucial due to their similar clinical-laboratory features, but differing pathophysiological mechanisms and treatment strategies.*

Keywords: *anemia of chronic disease; iron deficiency anemia; prevalence; diagnosis; inflammation; cytokines; erythropoietin.*

Anemia affects approximately 4% of men and 8% of women in the general population, increasing to 8–44% in middle-aged and elderly individuals. One of the most common types is *anemia of chronic disease* (ACD), also known as anemia of chronic inflammation or iron-restrictive anemia. ACD typically arises in patients with prolonged activation of cellular immunity, persisting for more than 1–2 months. Among all anemia types, ACD ranks second in frequency, following IDA.

In older adults, comorbidities contribute significantly to the overall disease burden, and anemia often remains underrecognized. ACD frequently develops in the setting of chronic inflammatory, infectious, or malignant diseases. The primary pathogenic mechanism is impaired iron metabolism, with iron sequestration in activated macrophages of the reticuloendothelial system (RES), limiting iron availability for erythropoiesis despite adequate or increased total body iron stores.

Pathogenesis and Morphological Features

Anemia is a multifactorial condition, with diverse etiologies and manifestations. ACD is classified morphologically as normocytic anemia (MCV 81–100 fL), and according to regeneration levels, as regenerative anemia, although the reticulocyte response may vary.

Clinical signs depend both on the degree of anemia and the severity of the underlying disease. Tissue hypoxia plays a major role in symptom expression. ACD may mask or mimic other disease processes, making timely diagnosis essential.

In ACD, hemoglobin (Hb) levels typically range between 90–110 g/L, and symptoms may be subtle due to the body's adaptive mechanisms. Clinical manifestations become more apparent with Hb levels below 80 g/L, and when Hb drops under 40 g/L, the risk of anemic coma increases.

Diagnostic Evaluation

Laboratory diagnostics should include the following iron metabolism parameters:

- **Serum iron (SI):** decreased in ACD.
- **Ferritin (SF):** normal or increased (acute-phase reactant).
- **Transferrin saturation (TSAT):** reduced.
- **Soluble transferrin receptor (sTfR):** typically normal or mildly increased (unlike in IDA, where it's elevated).

Ferritin is the most specific indicator of iron stores in the absence of inflammation. However, its levels may be misleading in ACD due to its role as an acute-phase protein. The concentration of iron in reticulocytes (>28 pg) can indicate sufficient functional iron. Inflammatory processes downregulate transferrin production (a negative acute-phase reactant), reducing serum iron delivery to erythroid precursors.

Further diagnostic steps include evaluating erythropoietin (EPO) levels and assessing erythrocyte hypochromia. Bone marrow examination is reserved for cases with diagnostic uncertainty or suspicion of marrow infiltration.

Differential Diagnosis: ACD vs IDA

A key challenge is distinguishing ACD from IDA or mixed forms. In IDA:

- Ferritin is low.
- sTfR is elevated.
- Transferrin is elevated, with low TSAT.

In contrast, ACD presents with low SI, normal/increased SF, and normal/mildly increased sTfR. Mixed forms of IDA and ACD often exhibit microcytosis and more pronounced hypochromia.

ACD in Specific Clinical Contexts

1. Infectious Diseases

ACD commonly occurs in patients with chronic infections such as lung abscesses, bronchiectasis, or kidney infections. It can appear a month or more after disease onset, often as a secondary complication. Treating the infection typically leads to anemia improvement without the need for targeted anemia therapy.

2. HIV Infection

HIV-related anemia stems from direct viral effects on bone marrow, opportunistic infections, cytokine dysregulation, and antiretroviral drug toxicity. Anemia correlates with higher viral load and significantly impacts patient quality of life and survival.

3. Chronic Kidney Disease (CKD)

ACD in CKD arises from inadequate EPO production, iron-restricted erythropoiesis, and chronic inflammation. Additional factors include hyperparathyroidism, uremic toxins, and reduced red cell lifespan. Anemia severity correlates with CKD progression and contributes to cardiovascular complications.

4. Connective Tissue Disorders

Rheumatoid arthritis, systemic lupus erythematosus (SLE), and vasculitides frequently present with ACD. Inflammatory cytokines (IL-6, TNF- α , IFN- γ) inhibit erythroid progenitor cells and promote hepcidin synthesis, a key hormone in iron sequestration. In RA, anemia occurs in up to 65% of patients; in SLE, around 50%.

5. Liver Disease

Anemia in chronic liver disease may result from hypersplenism, hemolysis, nutritional deficiencies, and folate depletion. ACD in liver pathology is typically macrocytic or normocytic, often secondary to impaired EPO production and bone marrow suppression.

6. Endocrine Disorders

Endocrinopathies may cause normo-, hypo-, or macrocytic anemia. Hypothyroidism, adrenal insufficiency, hypogonadism, and diabetes mellitus are frequent contributors. In hypothyroidism, anemia results from decreased oxygen demand and reduced EPO levels. Diabetes-related anemia is often related to nephropathy and reduced renal EPO production.

6. Malignancies

Anemia is highly prevalent in cancer patients—observed in up to 90% during treatment.

The causes include chronic inflammation, chemotherapy-induced marrow suppression, impaired iron utilization, and nutritional deficiencies. ACD in cancer is associated with worsened prognosis, reduced functional status, and poor response to chemotherapy. Erythropoiesis-stimulating agents (ESAs) and intravenous iron can improve Hb levels and quality of life, though response rates are variable.

Diagnostic and Therapeutic Algorithm

The approach to normocytic anemia includes:

1. **Reticulocyte Count:** High count suggests blood loss or hemolysis. Low or normal count indicates impaired production.
2. **Iron Studies:** To distinguish between IDA, ACD, and mixed states.
3. **Kidney and Liver Function Tests:** To identify organ-related anemia.
4. **Bone Marrow Examination:** Reserved for unclear etiology or suspected marrow disease.
5. **Inflammatory Markers (CRP, ESR):** Helpful in identifying ACD.

Treatment of ACD centers on managing the underlying disease. Iron supplementation is generally ineffective unless true deficiency coexists. ESA therapy may be indicated in CKD or oncology patients, often in combination with IV iron.

Conclusion

Anemia of chronic disease is a common and multifactorial syndrome occurring in diverse clinical conditions. Its diagnosis requires careful evaluation of iron metabolism, erythropoiesis, and inflammation markers. Differentiating ACD from IDA is critical due to differing treatment strategies. In all cases, addressing the underlying disease remains the cornerstone of effective management.

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