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ANEMIA OF CHRONIC DISEASE: PATHOGENESIS, DIAGNOSTIC CHALLENGES, AND TREATMENT APPROACHES

LINGUISTIC INTERPRETATION OF MYTHONYMS AND THEIR PLACE IN THE SYSTEM OF FOLKLORE: LEXICO-SEMANTIC AND ETHNOLINGUISTIC ANALYSIS

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ANNOTATION. This article studies the linguistic nature of the concept of mythonym, their cross-linguistic semantic development and their place in folklore linguistics. The process of transformation of mythonyms into anthroponyms and cognate nouns, as well as their lexical-semantic and pragmatic features on the example of Uzbek and Khorezm folklore, is highlighted. The article provides a comparative analysis of the role of mythonyms in word formation and their etymological roots.

Keywords: Mythonym, folklore linguistics, semantics, Uzbek folklore, Khorezm mythology, anthroponym, lexical layer, cultural context, etymology.

INTRODUCTION. Myths, which are an integral part of human culture, encompass extremely rich and multifaceted concepts in linguistics. Although the term "myth" originally meant "legend" or "narrative", in the modern linguistic context its range of meanings has expanded significantly. Mythonyms - names representing mythological images, characters and symbolic concepts - occupy a special place in the language system. They reflect the worldview, beliefs and convictions of the people through language, enriching the lexical layer of the language.

In different languages, the word "myth" has undergone its own semantic development. For example, the word myth in English means "an imagination that contradicts reality", in German Mythe and Mythos "an unfounded imagination", and in French mythe also includes negative connotations such as "slander". This indicates that myths are perceived as "false" over time, as a result of their contradiction with historical truths.

Mythonyms (Greek μῦθος - myth and ὄνομα - name) are proper nouns that represent mythical concepts. Their emergence is directly related to semantic features, and over time these images become universal qualities. For example, the name Penelope in Greek mythology became a common noun as a symbol of fidelity, and Hercules became a common noun as a symbol of physical strength.

Mythonyms are used not only as ready-made units, but also serve as a derivational basis for the formation of new words. This can be seen in the following examples:

Hypnos (Hypnos): Terms such as hypnosis (hypnosis), hypnotherapy (hypnotherapy), hypnophobia were formed based on the name of the god of sleep.

Bacchus (Bacchus): The word bacchanalia, associated with the name of the god of wine, is used today in the meaning of "disorderly gathering".

Tantalum: The name of the hero condemned to suffering gave rise not only to the metaphor of "unattainable", but also to the name of the chemical element - Tantalum (Ta).

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Many names in Uzbek mythology are associated with ancient Indo-Iranian languages, and their etymology indicates the development of the language:

Dev (Daeva): In the Avestan language, "daeva" meant evil spirits. Interestingly, in Indo-European languages, this root (deiwos) means "divine", "enlightened". In Uzbek, this word has undergone semantic transformation and has become a huge, muscular character.

Alvasti: The root of this mythological figure may have come from the words "al" (red) and "basdi" (to press). This explains its mythological function associated with night terrors and women.

Folk linguistics studies the language features in folklore texts. Mythologies in Uzbek folklore are mainly a mixture of pre-Islamic and Islamic concepts.

Classification of Good and Evil

Mythonyms in the Uzbek language are divided into two poles according to their semantic orientation:

Mythonyms of goodness: Khizr, chilton, maloyika, pari. "Khizr" is a symbol of wisdom and patronage, while "pari" is a symbol of beauty and grace.

Mythonyms of evil: Dev, alvasti, ajina, yalmogiz. "Alvasti" reflects danger and fear in the minds of the people and creates a negative coloring in speech.

Functional comparison of world and Uzbek mythonyms

Traces of ancient Zoroastrianism and shamanism have been preserved in the system of mythological representations of the Khorezm region. In the folklore of this region, images such as Torso pari and Sumyon pari are of particular importance. They are mentioned as auxiliary spirits in the ritual songs of shamans. Chilton and childukhtaron (forty girls) are considered protective spirits. All these mythonyms are closely related to the elements of nature (water, fire, earth).

Mythonyms are a powerful poetic tool not only in oral speech, but also in written literature. In the works of Alisher Navoi, mythological images carry symbolic meaning. For example, through the image of "Ozar" (a mythological hero associated with fire), the poet illuminates the theme of love. In classical poetry, mythonyms such as "Parivash" or "Huri paykar" provide expressiveness of speech when describing the beauty of a lover.

CONCLUSION. Mythonyms are not just legendary names, but are a linguistic reflection of folk thinking. Along with enriching the lexical fund of the language, they serve as a bridge in transmitting cultural and historical heritage from generation to generation. The example of Uzbek and Khorezm folklore shows that mythonyms have become an integral part of our modern speech through semantic expansion and pragmatic loading.

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Abstract

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Anemia of chronic disease (ACD), also known as anemia of inflammation, is one of the most common forms of anemia observed in patients with chronic infections, autoimmune disorders, malignancies, chronic kidney disease, and other long-standing inflammatory conditions. This type of anemia develops through complex interactions between the immune system, iron metabolism, and erythropoiesis. The main mechanisms include increased hepcidin production, reduced intestinal iron absorption, impaired release of stored iron, decreased erythropoietin activity, and suppressed bone marrow response. As a result, patients develop iron-restricted erythropoiesis despite normal or increased iron stores. The present article reviews the current understanding of the pathogenesis, laboratory characteristics, differential diagnosis, and treatment strategies of anemia of chronic disease. Special attention is given to the distinction between ACD and iron deficiency anemia, since these conditions may coexist and create diagnostic difficulty. The reviewed evidence shows that accurate diagnosis requires combined interpretation of hemoglobin levels, ferritin, transferrin saturation, inflammatory markers, and the clinical background of the patient. Management of ACD is primarily based on treatment of the underlying disease, while iron therapy and erythropoiesis-stimulating agents may be used in selected cases. A better understanding of inflammation-driven iron dysregulation, especially the role of the hepcidin-ferroportin axis, may improve future diagnostic and therapeutic approaches.

Keywords

Anemia of chronic disease; anemia of inflammation; hepcidin; iron metabolism; erythropoiesis; chronic inflammation; diagnosis; treatment

Introduction

Anemia of chronic disease (ACD), also referred to as anemia of inflammation, is one of the most common forms of anemia encountered in hospitalized patients and individuals with long-standing inflammatory, infectious, autoimmune, neoplastic, or chronic organ diseases. It develops as part of a complex host response in which immune activation alters iron metabolism, suppresses erythropoiesis, and shortens red blood cell survival. Unlike iron deficiency anemia, ACD is characterized not only by reduced circulating iron but also by impaired iron utilization despite preserved or increased iron stores. This distinction makes ACD both diagnostically challenging and clinically significant [1,2].

The pathophysiology of ACD is strongly linked to chronic inflammation. Pro-inflammatory cytokines, particularly interleukin-6, stimulate hepatic production of hepcidin, the principal regulator of systemic iron homeostasis. Hepcidin decreases intestinal iron absorption and blocks iron release from macrophages and hepatocytes by inducing degradation of ferroportin, thereby causing hypoferremia and iron-restricted erythropoiesis. In addition, inflammatory mediators can reduce erythropoietin production and impair bone marrow responsiveness, further contributing to anemia. These mechanisms explain why ACD is frequently observed in patients with chronic kidney disease, malignancy, rheumatoid arthritis, chronic infections, heart failure, and other persistent inflammatory states [4].

From a clinical perspective, ACD is important because it contributes to fatigue, reduced physical capacity, impaired quality of life, longer hospitalization, and worse outcomes in patients with chronic illness. Accurate diagnosis is essential, yet differentiation between ACD and absolute iron deficiency may be difficult because both conditions may coexist. Hemoglobin-based recognition of anemia remains the first step, and current World Health Organization guidance provides updated recommendations for defining anemia using hemoglobin thresholds in clinical and population settings [6]. However, establishing the specific cause of anemia

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requires integration of inflammatory markers, iron studies, and the clinical background of the patient.Given its high prevalence and multifactorial mechanism, ACD remains a major diagnostic and therapeutic challenge in modern medicine . Better understanding of the hepcidin-ferroportin axis and inflammatory regulation of erythropoiesis has improved insight into disease mechanisms and opened new possibilities for targeted treatment. Therefore, this article aims to review the current understanding of anemia of chronic disease with emphasis on its pathogenesis, diagnostic approach, and treatment strategies [5].

Methods

This article was prepared as a narrative literature review focusing on anemia of chronic disease (ACD), also known as anemia of inflammation. The purpose of the review was to summarize and synthesize current scientific understanding of the epidemiology, pathophysiology, diagnosis, and treatment of ACD based on published medical literature and international guidance documents. Relevant sources were selected from peer-reviewed journals, hematology reviews, and global health guidelines addressing anemia, iron metabolism, inflammation, and chronic disease-associated hematologic changes. Particular attention was given to publications describing the role of inflammatory cytokines, hepcidin-mediated iron restriction, erythropoietin suppression, and the clinical distinction between ACD and iron deficiency anemia [3]. The review process involved identification of key concepts related to the pathogenesis and clinical assessment of ACD. Major thematic areas included inflammatory regulation of iron homeostasis, mechanisms of impaired erythropoiesis, laboratory features used in differential diagnosis, and currently recommended therapeutic approaches in patients with chronic inflammatory states. Sources were chosen according to their scientific relevance, citation frequency, clinical applicability, and contribution to contemporary understanding of the disorder. International recommendations, including World Health Organization guidance on anemia definition and classification, were also incorporated in order to align the discussion with current diagnostic standards [6].

During data extraction, information from the selected publications was organized into several analytical categories: definition and terminology of ACD, etiologic conditions associated with chronic inflammation, molecular mechanisms of disease development, characteristic laboratory findings, diagnostic challenges, and available management strategies. Evidence describing the central role of interleukin-6 and hepcidin in iron sequestration and iron-restricted erythropoiesis was reviewed in detail because these mechanisms form the biological basis of ACD and explain many of its hematologic and biochemical manifestations. Additional emphasis was placed on studies discussing the overlap between ACD and true iron deficiency, since coexistence of these conditions often complicates clinical interpretation of ferritin, transferrin saturation, and other iron indices [5].

The collected material was analyzed descriptively rather than statistically, because the objective of the article was not to perform a meta-analysis but to provide an integrated and clinically meaningful overview of the topic. Information from the included references was compared and synthesized in a structured manner to identify common findings and major conclusions regarding disease mechanisms, diagnosis, and treatment. Priority was given to well-established evidence from authoritative hematology literature and widely recognized clinical sources. Through this method, the article aimed to present a coherent summary of current knowledge on anemia of chronic disease for academic and educational purposes .

Results

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The reviewed literature consistently showed that anemia of chronic disease (ACD) is a frequent hematologic complication in patients with persistent inflammatory conditions, including chronic infections, autoimmune disorders, malignancy, chronic kidney disease, heart failure, and chronic pulmonary disease. Across the analyzed sources, ACD was described as a predominantly mild to moderate anemia that most often presents as normocytic and normochromic, although microcytic features may develop in prolonged or more severe cases. The overall findings demonstrated that the central biological pattern of ACD is iron-restricted erythropoiesis caused by inflammation-driven disturbances in iron handling rather than absolute depletion of total body iron stores [1,3]. A major result observed throughout the reviewed studies was the consistent role of inflammatory mediators in suppressing effective erythropoiesis. Interleukin-6 was repeatedly identified as a key cytokine stimulating hepatic hepcidin synthesis, which in turn reduces intestinal iron absorption and prevents iron export from macrophages and hepatocytes through ferroportin degradation. This process leads to decreased circulating serum iron despite normal or increased ferritin stores, producing a state of functional iron deficiency. In addition, chronic inflammation was found to blunt erythropoietin production and impair bone marrow responsiveness, while also modestly shortening erythrocyte survival, further worsening anemia severity [1].

The laboratory profile described across the selected references was relatively uniform. Hemoglobin levels were reduced according to standard anemia definitions, while reticulocyte counts were typically low or inappropriately normal for the degree of anemia, indicating insufficient marrow response. Serum iron and transferrin saturation were commonly decreased, whereas transferrin or total iron-binding capacity tended to be low because transferrin behaves as a negative acute-phase reactant in inflammatory states [2]. Ferritin was usually normal or elevated, reflecting preserved iron stores and the acute-phase response, which helps differentiate ACD from pure iron deficiency anemia, where ferritin is generally reduced. However, the results also emphasized that mixed patterns are common, especially when ACD coexists with true iron deficiency, making isolated interpretation of ferritin potentially misleading [3,5].

Another important result from the reviewed literature was the diagnostic difficulty of distinguishing ACD from iron deficiency anemia in patients with chronic illness. Several authors noted that no single biomarker is sufficient in all cases, and interpretation must be based on the combined pattern of hemoglobin, ferritin, transferrin saturation, inflammatory markers, and clinical context. Inflammatory conditions may artificially raise ferritin concentrations, masking depleted iron stores, while low transferrin saturation can be present in both conditions. This overlap was reported as one of the main reasons why ACD remains underrecognized or misclassified in daily clinical practice.

With regard to clinical impact, the reviewed evidence showed that ACD is associated with fatigue, weakness, decreased exercise tolerance, impaired quality of life, and, in many chronic diseases, poorer clinical outcomes . The presence of anemia was linked to greater functional limitation and increased burden of disease, especially in patients with chronic kidney disease, cancer, or systemic inflammatory disorders. Although anemia severity is often not extreme, the persistent and multifactorial nature of ACD was found to contribute substantially to morbidity.

Therapeutic findings across the literature indicated that treatment of the underlying inflammatory disease remains the cornerstone of management. Correction of the primary disorder may improve iron mobilization and erythropoiesis, but many patients require additional supportive strategies depending on the underlying cause and degree of anemia. Erythropoiesis-stimulating agents were reported to be useful in selected settings such as chronic kidney disease, while iron therapy may be considered in carefully evaluated patients, especially when absolute

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iron deficiency coexists. The literature also highlighted increasing scientific interest in therapies targeting the hepcidin–ferroportin pathway, although these approaches remain more relevant to emerging research than routine practice .

Table 1. Main findings of the reviewed literature on anemia of chronic disease

Parameter	Typical finding in ACD	Clinical interpretation
Hemoglobin	Decreased	Confirms anemia based on current diagnostic thresholds
MCV	Usually normal; sometimes low in prolonged disease	ACD is commonly normocytic, but may become microcytic
Reticulocyte count	Low or inappropriately normal	Suggests reduced marrow response
Serum iron	Low	Reflects reduced circulating available iron
Transferrin / TIBC	Low	Decreases in inflammatory states as a negative acute-phase reactant
Transferrin saturation	Low	Indicates restricted iron availability for erythropoiesis
Ferritin	Normal or elevated	Suggests preserved/increased iron stores and inflammation effect
Hepcidin	Increased	Central mediator of iron sequestration and reduced absorption
Bone marrow iron stores	Usually preserved	Helps distinguish ACD from pure iron deficiency
Common associated diseases	CKD, cancer, autoimmune disease, chronic infection, heart failure	Indicates strong association with persistent inflammation

Overall, the results of this review indicate that ACD is a multifactorial anemia driven primarily by inflammation-induced iron sequestration, impaired erythropoietin activity, and reduced erythroid response. The reviewed evidence supports the view that diagnosis should rely on integrated clinical and biochemical assessment rather than a single laboratory parameter, especially in patients with possible coexisting iron deficiency . These findings reinforce the importance of recognizing ACD as a distinct but overlapping clinical entity that significantly influences the course and quality of life of patients with chronic disease [1,4].

Discussion

The findings of this review confirm that anemia of chronic disease (ACD), or anemia of inflammation, should be understood as a multifactorial disorder in which chronic immune activation disrupts iron homeostasis and erythropoiesis rather than simply reducing total body

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iron stores . This distinction is clinically important because many patients with chronic inflammatory disorders present with low serum iron, yet the underlying mechanism is primarily iron sequestration and impaired iron availability caused by elevated hepcidin and inflammatory cytokine activity. As a result, ACD differs fundamentally from pure iron deficiency anemia, even though the two conditions may produce overlapping laboratory findings and may coexist in the same patient [1,4].

The reviewed evidence highlights the central role of the hepcidin–ferroportin axis in explaining the laboratory and clinical profile of ACD. Inflammatory signaling, particularly through interleukin-6, increases hepcidin synthesis, which reduces intestinal iron absorption and blocks release of stored iron from macrophages and hepatocytes. This mechanism explains why serum iron and transferrin saturation are typically low despite normal or elevated ferritin concentrations. From a pathophysiologic perspective, these findings support the concept that ACD is best viewed as a state of functional iron deficiency superimposed on chronic inflammatory disease, with additional contribution from reduced erythropoietin activity, impaired marrow responsiveness, and shortened erythrocyte survival [1,2].

An important implication of these results is the persistent diagnostic challenge of distinguishing ACD from absolute iron deficiency in real clinical settings. Ferritin, although widely used as a marker of iron stores, behaves as an acute-phase reactant and may therefore appear normal or increased even when true iron deficiency is present in patients with inflammation. Likewise, low transferrin saturation can be seen in both iron deficiency anemia and ACD, limiting its specificity when used alone . For this reason, the reviewed literature consistently supports an integrated diagnostic approach based on hemoglobin concentration, iron studies, inflammatory markers, reticulocyte response, and the patient's overall clinical context rather than reliance on a single laboratory parameter. The updated WHO guidance remains important for defining anemia by hemoglobin thresholds, but identification of ACD specifically requires etiologic and biochemical interpretation beyond anemia screening alone [3].

The clinical relevance of ACD extends beyond laboratory abnormality. Even when anemia is mild or moderate, it contributes to fatigue, reduced exercise tolerance, impaired functional capacity, and poorer quality of life in patients who are already burdened by chronic disease. In disorders such as chronic kidney disease, cancer, autoimmune disease, and chronic infection, anemia may worsen overall prognosis and complicate management. Therefore, recognition of ACD is not merely descriptive; it has direct implications for symptom burden, disease monitoring, and therapeutic decision-making.

The reviewed evidence also suggests that management of ACD should remain centered on treatment of the underlying inflammatory condition whenever possible. Because inflammation is the major driver of disordered iron trafficking and ineffective erythropoiesis, successful control of the primary disease may partially or substantially improve the anemia. However, many patients do not experience full correction with treatment of the underlying illness alone, especially when inflammation persists or when chronic kidney disease, malignancy, or mixed iron deficiency is present. In these settings, erythropoiesis-stimulating agents may be appropriate in selected patients, particularly in chronic kidney disease, while iron supplementation may be useful when absolute iron deficiency coexists or when functional iron restriction is carefully documented. This point is especially important because indiscriminate iron treatment without proper differentiation between iron deficiency and ACD may lead to suboptimal results.

Another significant issue raised by the reviewed literature is the growing interest in targeted therapies that interfere with the hepcidin pathway or improve iron mobilization [1]. These

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emerging approaches reflect a major shift from supportive management toward mechanism-based treatment. Although such therapies are not yet standard in most routine settings, they demonstrate how improved understanding of inflammatory iron regulation may reshape future care of ACD. From an academic standpoint, this also confirms that ACD is no longer viewed as a passive byproduct of chronic illness, but as a biologically active and potentially modifiable component of disease.

This review has several limitations. Because it was designed as a narrative review rather than a systematic review or meta-analysis, the findings are descriptive and integrative rather than quantitative. The article synthesizes current concepts from authoritative reviews, clinical references, and guideline documents, but it does not estimate pooled prevalence, treatment effect size, or outcome risk statistically. In addition, diagnostic criteria and therapeutic strategies may vary depending on the underlying disease population, meaning that conclusions about ACD should always be interpreted within specific clinical contexts. Overall, the discussion supports the view that ACD is a distinct but overlapping form of anemia characterized by inflammation-driven iron restriction, inadequate erythropoietic response, and substantial clinical impact. Early recognition and careful differentiation from iron deficiency are essential for rational management. Future progress will likely depend on improved biomarkers, better identification of mixed anemia states, and wider clinical translation of therapies targeting hepcidin-mediated iron dysregulation [1,4].

Conclusion

In conclusion, anemia of chronic disease is a common and clinically important form of anemia that develops in association with chronic inflammation, infection, malignancy, autoimmune disorders, and chronic organ failure. Its pathogenesis is complex and involves inflammation-induced disturbances in iron homeostasis, increased hepcidin activity, reduced erythropoietin response, and impaired erythropoiesis. Unlike pure iron deficiency anemia, ACD is characterized by restricted iron availability despite normal or increased iron stores, which makes diagnosis more challenging and requires careful interpretation of laboratory findings in relation to the patient's clinical condition [1,2]. The evidence reviewed in this article shows that ACD has a significant negative effect on patient well-being, physical capacity, and overall disease burden. Accurate differentiation between ACD and iron deficiency anemia, particularly in mixed cases, is essential for appropriate management. Treatment should primarily focus on the underlying disease process, while supportive strategies such as iron therapy or erythropoiesis-stimulating agents may be considered in selected patients depending on the cause and severity of anemia .

Overall, a better understanding of the inflammatory mechanisms underlying ACD, especially the role of the hepcidin-ferroportin axis, has improved diagnostic and therapeutic approaches. Continued research into more specific biomarkers and targeted therapies may further enhance clinical outcomes and provide more effective management of this common hematologic complication.

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