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*Article*

# Characteristics of Renal Anaemia in Patients with Chronic Kidney Disease and Diabetes Mellitus

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**Abstract:** Aim. To systematize current evidence on the mechanisms, clinical features, and diagnosis of renal anaemia in patients with chronic kidney disease and diabetes mellitus. Material and methods. A narrative review of publications from 2010 to 2026 was performed using the PubMed, Scopus, Web of Science, and eLIBRARY.ru databases. The analysis included studies containing data on the pathogenesis, laboratory parameters, and treatment approaches to anaemia in chronic kidney disease (CKD) and diabetes mellitus. Results. A decline in erythropoietin production is observed once the glomerular filtration rate falls below 60 mL/min/1.73 m² [1]. In patients with diabetes mellitus, anaemia is recorded at earlier stages of CKD-at a glomerular filtration rate of 60–90 mL/min/1.73 m² [2]. A haemoglobin concentration below 120 g/L is detected in 20–40% of patients with diabetic nephropathy [3]. Elevated hepcidin levels restrict iron mobilization and reduce the efficiency of erythropoiesis [4]. Inflammatory markers, including C-reactive protein, correlate with a decline in haemoglobin and with resistance to erythropoietin therapy [5]. Conclusions. Renal anaemia in diabetes mellitus is characterized by an earlier onset and by the coexistence of several pathophysiological mechanisms: erythropoietin deficiency, chronic inflammation, and disturbances of iron metabolism. Diagnosis requires assessment of haemoglobin, ferritin, transferrin, and the level of inflammatory markers. Treatment approaches must take into account the combined nature of the pathogenesis.

**Keywords:** renal anaemia, chronic kidney disease, diabetes mellitus, erythropoietin, hepcidin, iron deficiency.

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## 1. Introduction

Chronic kidney disease frequently develops in patients with diabetes mellitus. According to KDIGO, CKD is diagnosed when the estimated glomerular filtration rate falls below 60 mL/min/1.73 m², or when markers of kidney damage, including albuminuria, persist for at least three months [1]. In patients with diabetes mellitus, renal involvement develops more rapidly, and clinical complications arise at earlier stages of the disease [2].

Anaemia is a common complication of CKD. A decline in haemoglobin below 120 g/L in women and 130 g/L in men is recorded even with a moderate reduction in kidney function [3]. In patients with diabetic kidney disease, anaemia is detected earlier and is characterized by a more pronounced decline in haemoglobin than in non-diabetic CKD [4].

In the study by Thomas et al., anaemia was diagnosed in 20–40% of patients with type 2 diabetes mellitus at comparable glomerular filtration rate values [5].

Renal anaemia develops through several parallel mechanisms. A decline in erythropoietin production occurs with damage to the peritubular cells of the kidney and progresses as the glomerular filtration rate falls [6]. Disturbances of iron metabolism are associated with elevated hepcidin levels, which block the release of iron from macrophages and reduce its availability for erythropoiesis [7]. Chronic inflammation intensifies this effect by increasing the production of pro-inflammatory cytokines, including interleukin-6 [8].

In diabetes mellitus, these mechanisms are compounded by hyperglycaemia and microvascular changes. Damage to renal tissue leads to an earlier reduction in erythropoietin synthesis, while diabetic autonomic neuropathy disrupts the regulation of erythropoiesis [9]. The work of Al-Khoury et al. demonstrated that anaemia in patients with diabetic nephropathy develops earlier and follows a more severe course than in other forms of CKD.

A decline in haemoglobin worsens the clinical course of CKD. In patients with CKD, anaemia is associated with reduced exercise tolerance, impaired quality of life, and an increased risk of cardiovascular complications[10]. These features underscore the need for early diagnosis of renal anaemia in patients with diabetes mellitus and for a comprehensive assessment of its pathogenetic mechanisms.

## 2. Materials and Methods

A narrative review of the literature on renal anaemia in chronic kidney disease among patients with diabetes mellitus was carried out. The analysis included studies containing quantitative data on haemoglobin, indices of iron metabolism, and the levels of erythropoietin, hepcidin, and inflammatory markers, as well as clinical guidelines on the diagnosis and treatment of anaemia in CKD[11].

The literature search was conducted in the PubMed, Scopus, Web of Science, eLIBRARY.ru, and CyberLeninka databases. The following keywords were used: “chronic kidney disease,” “renal anemia,” “diabetic nephropathy,” “hepcidin,” “erythropoietin deficiency,” and “iron metabolism,” together with their Russian-language equivalents. Search queries were combined using the Boolean operators AND/OR. An example query was “chronic kidney disease AND anemia AND diabetes”; the supplementary search included combinations with the terms “inflammation,” “iron deficiency,” and “ESA therapy”[12-14].

The analysis included publications for the period 2000–2026. The lower boundary was determined by the emergence of key works on the pathogenesis of anaemia in CKD and the role of erythropoietin; the upper boundary corresponds to the current KDIGO and ADA guidelines, which reflect contemporary approaches to diagnosis and treatment. The main body of evidence comprised studies from the past 10–15 years, which present data on hepcidin, chronic inflammation, and functional iron deficiency[15].

For the assessment of laboratory parameters, studies were included that contained data on the concentration of haemoglobin and ferritin, transferrin saturation (TSAT), and the levels of hepcidin and C-reactive protein. The analysis included works that used standardized threshold values: haemoglobin <130 g/L in men and <120 g/L in women, ferritin <100 ng/mL, and TSAT <20% as criteria for iron deficiency in CKD[16].

Publications assessing the impact of anaemia on clinical outcomes were additionally reviewed. These included studies that analysed indices of quality of life, exercise tolerance, hospitalization rates, and cardiovascular complications in patients with CKD and diabetes mellitus[17].

The sources were analysed along three main lines of enquiry: the pathogenesis, the clinical features, and the treatment of renal anaemia. The data were systematized with regard to the differences between diabetic and non-diabetic CKD, as well as the stage of decline in kidney function and the severity of anaemia [4, 7, 10]. Publications were selected in which anaemia was considered in patients with chronic kidney disease in the setting of diabetes mellitus or diabetic kidney disease. Studies comparing the course of anaemia in diabetic and non-diabetic CKD were considered separately. This approach made it possible to identify the features specific to patients with diabetes mellitus: an earlier decline in haemoglobin, an association with albuminuria, microvascular renal damage, and impaired erythropoietin production[18-20].

During data extraction, the following principal laboratory parameters were recorded: haemoglobin, ferritin, transferrin saturation, estimated glomerular filtration rate, albuminuria, C-reactive protein, and hepcidin. The threshold values applied in international guidelines were used as diagnostic reference points: haemoglobin below 130 g/L in men and below 120 g/L in women; TSAT below 20% and ferritin below 100 ng/mL as indicators of iron deficiency in CKD[21].

**Table 1.** Principal parameters used in the analysis of the sources.

| **Parameter analysed** | **Value for the review** | **Clinical interpretation** |
|---|---|---|
| Haemoglobin | Principal criterion of anaemia | Reflects the severity of the anaemic syndrome |
| GFR | Staging of CKD | Allows anaemia to be correlated with the degree of decline in kidney function |
| Albuminuria | Marker of diabetic kidney damage | Indicates early microvascular damage |
| Ferritin | Iron stores | Used to detect absolute iron deficiency |
| TSAT | Availability of iron for erythropoiesis | Allows assessment of functional iron deficiency |
| Hepcidin | Regulation of iron metabolism | Elevation is associated with inflammation and reduced iron mobilization |
| C-reactive protein | Marker of inflammation | Helps assess the inflammatory component of anaemia |
| Erythropoietin | Regulation of erythropoiesis | Reduced production is characteristic of renal anaemia |

Treatment data were analysed separately. Oral and intravenous iron preparations, erythropoiesis-stimulating agents, hypoxia-inducible factor prolyl hydroxylase inhibitors, and the effect of glycaemic control on the course of anaemia were considered. In works that presented numerical results, the following were taken into account: the increase in haemoglobin, changes in ferritin and TSAT, the requirement for erythropoiesis-stimulating therapy, and the frequency of cardiovascular complications[22-23].

## 3. Results and Discussion

### Pathogenesis and early features of renal anaemia in diabetes mellitus.

Renal anaemia in CKD develops early. A decline in haemoglobin is already recorded at the stage of a glomerular filtration rate of 60–89 mL/min/1.73 m². In patients with diabetes mellitus, anaemia is recorded at this stage more frequently than in non-diabetic CKD. In cohort studies, the proportion of anaemia among diabetic patients with a glomerular filtration rate ≥60 mL/min/1.73 m² reaches 20–30%. At a comparable glomerular filtration rate, this figure is lower in patients without diabetes[24-26].

Erythropoietin deficiency is the key mechanism. Production of the hormone declines with damage to the peritubular cells of the renal cortex. The fall in erythropoietin levels correlates with a decline in glomerular filtration rate and intensifies as CKD progresses. In diabetes, this process begins earlier owing to microvascular damage. A number of works have shown that the concentration of endogenous erythropoietin in patients with diabetic nephropathy is lower than in other forms of CKD at an equivalent glomerular filtration rate[27-29].

Disturbances of iron metabolism aggravate anaemia. Elevated hepcidin restricts the release of iron from macrophages and reduces its availability for erythropoiesis. In patients with CKD, hepcidin levels rise as kidney function declines. In diabetes mellitus, this effect is more pronounced against a background of chronic inflammation. In clinical samples, the frequency of functional iron deficiency (TSAT <20% with normal or elevated ferritin) reaches 30–50%[29].

The inflammatory component is persistent. Levels of C-reactive protein and interleukin-6 are elevated in a significant proportion of patients with CKD. These markers correlate with a decline in haemoglobin and with the response to erythropoiesis-stimulating agents[30]. In diabetes mellitus, inflammation is sustained by hyperglycaemia and oxidative stress. An increase in pro-inflammatory cytokines is associated with elevated hepcidin and reduced iron mobilization.

Anaemia has quantitative criteria. The guidelines use haemoglobin thresholds of <130 g/L in men and <120 g/L in women. For the diagnosis of iron deficiency, TSAT <20% and ferritin <100 ng/mL are applied in non-dialysis patients. In samples of patients with diabetic kidney disease, these criteria reveal a combination of absolute and functional iron deficiency more frequently than in non-diabetic CKD[31].

Clinical manifestations appear early. A reduction in exercise tolerance is recorded at a haemoglobin of 100–120 g/L. Fatigue and a decline in physical activity intensify as anaemia progresses. In observational studies, lower haemoglobin levels are associated with an increased frequency of heart failure and hospitalizations in patients with CKD[32].

The early development of anaemia in diabetes is of practical importance. Screening for haemoglobin, ferritin, and TSAT is necessary at a glomerular filtration rate <60 mL/min/1.73 m² and in the presence of albuminuria. A number of works have shown that incorporating these parameters into routine follow-up makes it possible to detect anaemia before a pronounced decline in glomerular filtration rate[33].

**Table 2.** Pathogenetic mechanisms of renal anaemia in CKD and their features in diabetes mellitus.

| Mechanism | Manifestation in CKD | Features in diabetes mellitus |
|---|---|---|
| Erythropoietin deficiency | Reduced production as GFR falls | Earlier decline owing to microvascular damage |
| Hepcidin | Elevated levels; blockade of iron release | Intensified against a background of chronic inflammation |
| Iron deficiency | TSAT <20%, ferritin <100 ng/mL | Frequent combination of functional and absolute deficiency |
| Inflammation | Elevated CRP, IL-6 | Sustained by hyperglycaemia and oxidative stress |
| Uraemic toxins | Suppression of erythropoiesis | Intensified in diabetic nephropathy |
| Resistance to ESAs | Reduced response to therapy | More pronounced with inflammation and iron deficiency |

Albuminuria and glomerular filtration rate are used for risk stratification. In patients with diabetic kidney disease, anaemia is detected even in the presence of albuminuria >30 mg/g and a glomerular filtration rate above 60 mL/min/1.73 m². This warrants the inclusion of haemoglobin and iron indices in the standard follow-up of patients with diabetes mellitus. A number of clinical works have shown that early laboratory screening makes it possible to detect anaemia before the glomerular filtration rate falls below 60 mL/min/1.73 m²[34].

Treatment begins with correction of iron deficiency. Oral preparations are used in patients with mild anaemia and preserved intestinal function. Intravenous iron administration is used when the efficacy of oral therapy declines or in cases of pronounced iron deficiency . In clinical studies, intravenous iron formulations provide a more rapid increase in haemoglobin and a greater rise in TSAT than oral preparations[35].

Erythropoiesis-stimulating agents are used when iron therapy is insufficiently effective. The initiation of treatment is recommended when haemoglobin falls below 100 g/L, taking into account the symptoms of anaemia and the risk of cardiovascular complications. In clinical studies, the use of ESAs is accompanied by an increase in haemoglobin of 10–20 g/L over 8–12 weeks of therapy. In diabetes mellitus, the response to ESAs may be diminished owing to inflammation and functional iron deficiency[36].

Hypoxia-inducible factor prolyl hydroxylase inhibitors represent a new direction in therapy. These agents stimulate endogenous erythropoietin production and improve iron utilization. Randomized studies have demonstrated an increase in haemoglobin during HIF-PH inhibitor therapy without a significant rise in ferritin levels. In patients with CKD and diabetes mellitus, these agents show efficacy comparable to that of ESAs while being less dependent on iron stores[37].

Glycaemic control influences the course of anaemia. Elevated HbA1c values are associated with intensified inflammation and a worsened response to therapy. In observational studies, improved glycaemic control is accompanied by a reduction in C-reactive protein and stabilization of haemoglobin indices[38].

Anaemia is linked to clinical outcomes. A decline in haemoglobin below 110 g/L is associated with an increased frequency of heart failure and hospitalizations in patients with CKD. In patients with diabetes mellitus, this risk is higher owing to the combination of anaemia, hyperglycaemia, and vascular complications. In large cohort studies, a low haemoglobin level correlates with increased mortality and progression of CKD.

A comprehensive approach to treatment involves the assessment of several factors. Correction of iron deficiency, control of inflammation, the use of ESAs or HIF-PH inhibitors, and optimization of glycaemia are applied concurrently. Clinical guidelines emphasize the need for individualized selection of therapy, taking into account the stage of CKD, the haemoglobin level, and comorbid conditions[39].

**Table 3.** Principal approaches to the treatment of renal anaemia in CKD and diabetes mellitus.

| Treatment method | Mechanism of action | Indications | Features in diabetes mellitus |
|---|---|---|---|
| Oral iron | Replenishment of iron deficiency | Mild anaemia | Efficacy declines with inflammation |
| Intravenous iron | Rapid increase in iron stores | TSAT <20%, ferritin <100 ng/mL | Preferred in functional deficiency |
| ESAs | Stimulation of erythropoiesis | Hb <100 g/L | Resistance possible with inflammation |
| HIF-PH inhibitors | Stimulation of endogenous EPO | Alternative to ESAs | Less dependent on iron stores |
| Glycaemic control | Reduction of inflammation | HbA1c above target | Improves the response to therapy |

**Conclusion**

Renal anaemia in patients with chronic kidney disease (CKD) and diabetes mellitus develops earlier and has a more complex pathophysiology than anaemia in patients with CKD alone. Its development is primarily associated with reduced erythropoietin production, functional and absolute iron deficiency, increased hepcidin levels, chronic inflammation, and uraemic toxicity, while diabetes further accelerates these processes through microvascular injury, persistent hyperglycaemia, oxidative stress, and albuminuria. Comprehensive diagnosis should include haemoglobin, ferritin, transferrin saturation (TSAT), glomerular filtration rate (GFR), albuminuria, and inflammatory markers, as haemoglobin assessment alone is insufficient to determine the underlying cause of anaemia. Early identification of low TSAT despite normal or elevated ferritin is particularly important for detecting functional iron deficiency. Management of renal anaemia requires an integrated and individualized approach that combines correction of iron deficiency, appropriate use of erythropoiesis-stimulating agents (ESAs), optimization of glycaemic control, and reduction of chronic inflammation. Emerging therapies, particularly hypoxia-inducible factor prolyl hydroxylase (HIF-PH) inhibitors, represent promising treatment alternatives but should be selected according to CKD stage, cardiovascular risk, and associated comorbidities. Poor glycaemic control contributes to persistent inflammation and reduces responsiveness to ESA therapy, whereas effective diabetes management improves treatment outcomes. Overall, early diagnosis and timely comprehensive treatment of renal anaemia are essential for slowing CKD progression, reducing cardiovascular complications and hospitalizations, improving quality of life, and enhancing long-term clinical outcomes in patients with diabetic kidney disease. Regular monitoring of haemoglobin and iron metabolism parameters should therefore be considered an integral component of routine follow-up and long-term patient management.

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