Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 03, June, 2025
Website: usajournals.org

COMPARATIVE ANALYSIS OF ADIPONECTIN AND LEPTIN AS BIOMARKERS OF CHRONIC KIDNEY DISEASE IN INDIVIDUALS WITH EXCESS BODY WEIGHT

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Abstract
Chronic kidney disease (CKD) is increasingly prevalent in the context of rising obesity rates. Adipokines such as adiponectin and leptin play contrasting roles in metabolic regulation and renal pathophysiology. This review compares the diagnostic and prognostic potential of these biomarkers in overweight individuals with CKD. While adiponectin exhibits anti-inflammatory and renoprotective effects, leptin promotes inflammation and fibrosis. Their imbalance—characterized by hypoadiponectinemia and hyperleptinemia—may precede clinical markers of CKD. The leptin/adiponectin ratio may serve as an integrated indicator of metabolic and renal stress, though standardization remains a challenge. Further studies are needed to validate their clinical utility in nephrology.

Keywords: Adiponectin; Leptin; Chronic kidney disease; Obesity; Biomarkers; Adipokines.

Introduction

Chronic kidney disease (CKD) remains one of the leading causes of disability and mortality worldwide. According to KDIGO, 2023, the prevalence of CKD in adults exceeds 10%, and this figure continues to grow, especially among overweight and obese individuals. With increasing body weight, metabolic homeostasis, including lipid metabolism, is disrupted, which leads to an increased risk of renal tissue damage.

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An urgent task of modern nephrology is the search for reliable biomarkers reflecting the early stages of CKD, especially in individuals with metabolic disorders. Adiponectin and leptin, adipokines synthesized by adipose tissue, have attracted attention in recent years as potential indicators of renal dysfunction. Their levels are closely related to insulin resistance, inflammation, and the degree of metabolic imbalance. The present work is aimed at a comparative analysis of the diagnostic and prognostic significance of adiponectin and leptin in patients with overweight and signs of CKD, in order to assess their clinical value in the context of early diagnosis and monitoring of disease progression.

The Role of Obesity in the Pathogenesis of Chronic Kidney Disease

Overweight and obesity are recognized as significant risk factors for the development of chronic kidney disease (CKD), both directly and indirectly through the development of arterial hypertension, type 2 diabetes mellitus, and metabolic syndrome [1,2]. Adipose tissue is not a passive energy depot: it is an active endocrine organ capable of secreting many biologically active substances — adipokines, such as adiponectin, leptin, resistin, visfatin, and others [3].

Low-grade chronic inflammation, characteristic of abdominal obesity, stimulates the production of proinflammatory cytokines such as TNF-α and IL-6, which leads to damage to the endothelium and glomerular filtration membrane [4]. These changes, along with hyperfiltration and increased intraglomerular pressure, contribute to the development of glomerulosclerosis and loss of nephron function.

In obese patients, elevated leptin levels and decreased adiponectin levels are often observed, reflecting an imbalance between proinflammatory and anti-inflammatory signals. Such a hormonal profile contributes to the progression of renal damage even in the absence of other comorbid conditions [5].

Thus, obesity not only accelerates the development and progression of CKD, but also modifies the clinical manifestations of the disease, which makes it necessary to study specific markers of renal damage in this population.

Adiponectin as a Biomarker of Chronic Kidney Disease

Adiponectin is a cytokine produced primarily by white adipose tissue that has potent anti-inflammatory, insulin-sensitizing, and antiatherogenic properties.

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Adiponectin levels are typically inversely related to body mass index and visceral adipose tissue [6]. In healthy individuals, it helps maintain endothelial function, inhibits TNF-α production, and stimulates the synthesis of anti-inflammatory cytokines such as IL-10 [7].

In patients with CKD, circulating adiponectin levels are, paradoxically, often elevated, especially when the glomerular filtration rate (GFR) falls below 60 ml/min/1.73 m². This is not due to increased production, but rather to impaired renal clearance of adiponectin and changes in its molecular structure [8]. In addition, specific forms of adiponectin (high molecular weight) may have different biological activities, which requires further study [9].

Some studies suggest that high adiponectin levels may be a protective factor in the early stages of CKD by reducing inflammation and inhibiting fibrosis [10]. However, as renal failure progresses, its high levels may be associated with a worse prognosis, probably reflecting the overall catabolic status and protein metabolism disorders [11].

In the context of excess body weight, adiponectin demonstrates sensitivity to the patient's metabolic status. Low adiponectin levels are associated with insulin resistance, proteinuria, and glomerular hypertrophy, key links in the pathogenesis of CKD in obese individuals [12].

Thus, despite some contradictory data, adiponectin is considered a promising biomarker of early renal damage in patients with metabolic disorders.

Leptin as a Biomarker of Chronic Kidney Disease

Leptin is a hormone synthesized by adipocytes of white adipose tissue, the main function of which is to regulate energy metabolism and appetite control via hypothalamic centers [13]. In healthy individuals, leptin concentration directly correlates with body weight and percentage of adipose tissue. However, in addition to metabolic effects, leptin has a significant effect on the immune system, endothelium and kidneys [14].

In CKD, the serum leptin level increases proportionally to the degree of decrease in SCF, which is due to its reduced clearance due to impaired renal function [15]. Increased leptin concentration has a nephrotoxic effect: it promotes activation of the sympathetic nervous system, increased production of angiotensin II, induction

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of oxidative stress and activation of fibroblasts [16]. These effects can increase glomerular damage, stimulate sclerosis and accelerate the progression of renal failure.

In obese individuals with metabolic syndrome, chronically elevated leptin levels may precede renal function decline. Some studies show that hyperleptinemia is associated with albuminuria and podocyte dysfunction [17]. It is also suggested that leptin may induce expression of transforming growth factor β (TGF-β) in renal tissue, contributing to the development of interstitial fibrosis [18].

Thus, leptin is considered a prognostically significant biomarker in overweight patients. Its elevated levels reflect not only metabolic imbalance, but also active participation in the pathogenesis of structural kidney damage.

Comparative Analysis of Adiponectin and Leptin as Biomarkers of CKD

Adiponectin and leptin are two key adipokines with opposing metabolic and inflammatory effects. Their ratio may serve as an indicator of the patient's metabolic status and potentially reflect the progression of chronic kidney disease, especially in the context of obesity.

1. Physiological differences

Adiponectin has anti-inflammatory, antifibrotic, and insulin-sensitizing properties, while leptin, on the contrary, has a proinflammatory and proliferative effect. In obese patients, adiponectin levels are reduced and leptin levels are increased, which increases metabolic and vascular damage, including renal damage [19].

2. Dependence on kidney function

Both markers are metabolized partially or completely by the kidneys. An increase in their concentrations with a decrease in SCF reflects not only metabolic disorders, but also a violation of the excretory function of the kidneys [20]. However, adiponectin may have an anti-inflammatory effect, while hyperleptinemia, on the contrary, worsens inflammation and fibrosis [21].

3. Diagnostic and prognostic value

Some studies show that low adiponectin levels in overweight individuals are associated with a higher probability of developing albuminuria and a decrease in

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SCF in the early stages of CKD [22]. At the same time, high leptin levels correlate with a progressive decline in renal function, regardless of body weight, especially in women [23].

In addition, the calculation of the leptin/adiponectin ratio in the last decade has been considered as a potentially more sensitive indicator of metabolic risk and a marker of poor prognosis in CKD [24]. In this case, a low ratio reflects a "protective" profile, and a high ratio reflects an inflammatory and fibrotic potential.

4. Limitations of use

It is worth noting that the interpretation of the levels of these markers depends on many factors: gender, age, stage of CKD, presence of diabetes and concomitant cardiovascular diseases. There are also differences between the methods of quantitative analysis (enzyme immunoassays, chemiluminescent methods, etc.), which may limit the standardization of threshold values [25].

Thus, both markers - adiponectin and leptin - have clinical significance in assessing the risk and progression of CKD in patients with excess body weight. However, they reflect different aspects of the pathogenesis of the disease: adiponectin - rather as an indicator of protective mechanisms, leptin - as a reflection of aggressive metabolic disorders and inflammation.

Discussion

Chronic kidney disease remains one of the leading causes of disability and mortality, especially in the context of increasing prevalence of obesity and metabolic syndrome. In this regard, the search for sensitive and specific markers of early renal damage in overweight individuals is of great clinical importance.

A comparative analysis of adiponectin and leptin demonstrates that these adipokines not only reflect the metabolic state of the patient, but also actively participate in the pathogenesis of renal damage. Despite the fact that both markers increase with decreasing renal function, their biological effects are opposite. Adiponectin, as a rule, has a protective effect, reducing inflammation and

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inhibiting fibrosis, while leptin has a nephrotoxic potential, activating proinflammatory and fibrosing cascades.

In obese and overweight individuals, there is a characteristic imbalance between the levels of these two hormones - hypoadiponectinemia and hyperleptinemia, which increases inflammation, oxidative stress and renal hemodynamic impairment. This hormonal profile may precede the appearance of classical markers of CKD (albuminuria, decreased SCF) and be used for early diagnosis and risk stratification.

In addition, the leptin/adiponectin ratio proposed in the literature seems to be a promising composite marker of metabolic load on the kidneys. An increased value of this ratio correlates with proteinuria, decreased SCF and an increased risk of cardiovascular events in patients with CKD [1].

However, it is important to consider that the level of adipokines can be influenced by many factors - gender, age, level of physical activity, comorbidities, inflammatory markers. In addition, differences in analytical methods complicate the standardization of threshold values \u200b\u200bnecessary for clinical interpretation. Therefore, further prospective studies involving populations stratified by body weight and gender are needed to clarify the prognostic value of these markers and develop clinical algorithms for their use.

Conclusion

Adiponectin and leptin represent two functionally antagonistic adipokines with significant diagnostic and prognostic potential in chronic kidney disease (CKD), particularly in individuals with excess body weight. While adiponectin exhibits anti-inflammatory and renoprotective properties, leptin contributes to pro-inflammatory and profibrotic processes that accelerate renal damage.

heir concentrations are not only reflective of renal clearance but also mirror the underlying metabolic state and pathophysiologic burden on the kidneys. In obese individuals, the characteristic pattern of low adiponectin and high leptin may precede the onset of overt CKD, making these markers valuable for early detection and risk stratification.

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Moreover, the leptin/adiponectin ratio may serve as a composite biomarker that integrates inflammatory and metabolic stress, aiding in personalized prognosis and potentially guiding therapeutic strategies. However, broader clinical application is limited by a lack of standardization and the influence of confounding variables. Future studies are required to validate these findings and integrate them into routine nephrology practice.

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