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TITLE: Obesity-Related Chronic Kidney Disease: Current State of Knowledge

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Abstract

Introduction: Obesity and chronic kidney disease are a growing global health problem that often coexist[1]. Obesity is an independent risk factor for CKD, promoting kidney damage through hemodynamic and metabolic mechanisms[2].

Aim of the study: This article aims to summarize current evidence on chronic kidney disease in the context of obesity, focusing on key pathophysiological pathways and contemporary strategies for prevention and treatment.

Material and methods: A narrative review of the literature was performed using PubMed, Google Scholar, MedRxiv, and Scopus to identify human and selected experimental studies addressing epidemiology, mechanisms, diagnosis, and treatment of obesity-related chronic kidney disease.

Summary of current knowledge: Obesity-related CKD arises from interconnected hemodynamic, metabolic, inflammatory, and hormonal mechanisms, leading to hyperfiltration, proteinuria, and progressive renal damage[34]. The disease is often clinically silent, with albuminuria as an early marker[56]. Management requires early detection, weight reduction, RAAS blockade, and novel therapies such as SGLT2 inhibitors and GLP-1 receptor agonists, which provide renal protection beyond weight loss alone[7].

Conclusion: Obesity is an independent driver of chronic kidney disease[89]. Obesity-related CKD results from hemodynamic and metabolic injury and is often underdiagnosed[5]. Early detection and modern therapies are key to slowing progression and reducing risk.

Keywords: Obesity; Chronic Kidney Disease; Insulin Resistance; Inflammation; Proteinuria; Adipokines;

1. Introduction and purpose

Obesity and chronic kidney disease (CKD) are rapidly emerging as major global public health challenges, with prevalence rates increasing in parallel over recent decades[101112]. According to World Health Organization estimates, obesity has reached epidemic proportions worldwide, affecting a growing proportion of both adult and pediatric populations[2]. At the same time, CKD represents a leading cause of morbidity and mortality, currently affecting approximately 10–12% of the global population and contributing substantially to cardiovascular disease burden and premature death[1113] .

The coexistence of obesity and CKD is increasingly common[14]. Large population-based studies and cohort analyses have consistently demonstrated that individuals with overweight and obesity have a significantly higher lifetime risk of developing CKD compared with normal-weight individuals[1516]. Epidemiological data indicate that obesity may contribute to the development of CKD in up to 30% of affected patients, with both general and central adiposity being strongly associated with reduced estimated glomerular filtration rate (eGFR) and albuminuria[917] . Importantly, this association persists even after adjustment for traditional risk factors such as type 2 diabetes mellitus and hypertension[16].

Accumulating evidence now supports obesity as an independent risk factor for CKD. Excess adipose tissue promotes kidney injury through multiple interrelated mechanisms, including glomerular hyperfiltration, activation of the renin–angiotensin–aldosterone system, chronic low-grade inflammation, insulin resistance, oxidative stress, and ectopic lipid accumulation within renal tissue[7218]. These processes may ultimately lead to structural and functional kidney damage, even in the absence of overt metabolic disease[192021].

A distinct clinicopathological entity, termed obesity-related glomerulopathy (ORG), has been described as a characteristic renal phenotype associated with obesity[22]. ORG is typically defined by glomerulomegaly, focal segmental glomerulosclerosis, and progressive proteinuria, reflecting adaptive and maladaptive responses to sustained hemodynamic and metabolic stress[2324]. Notably, ORG often develops insidiously and may remain clinically silent for years before significant kidney dysfunction becomes apparent[25] .

Early recognition of CKD in patients with obesity is therefore of critical clinical importance. Delayed diagnosis is associated with accelerated disease progression, increased cardiovascular risk, and markedly higher all-cause mortality[26]. Given the projected rise in both obesity and CKD prevalence[1], improving awareness, early detection, and targeted management of obesity-related kidney disease represents an urgent priority for contemporary nephrology and public health practice.

The aim of this article is to provide an overview of the current state of knowledge regarding chronic kidney disease in the context of obesity, with particular emphasis on the underlying pathophysiological mechanisms and contemporary therapeutic approaches.

2. Material and methods

A comprehensive narrative literature review was conducted to summarize current evidence on chronic kidney disease in the context of obesity. The PubMed, Google Scholar, MedRxiv, and Scopus databases were systematically searched to identify relevant publications. The search

strategy included combinations of the following keywords: “obesity,” “chronic kidney disease,” “obesity-related kidney disease,” “obesity-related glomerulopathy,” “renal hyperfiltration,” “albuminuria,”. The review focused primarily on studies involving human participants, while selected experimental and preclinical studies were also considered when they provided important mechanistic insights. Eligible publications included meta-analyses, randomized controlled trials, observational and cohort studies, and case reports. The selected studies were analyzed and grouped according to their relevance to epidemiology, pathophysiological mechanisms, clinical presentation, diagnostic approaches, and therapeutic strategies for obesity-related chronic kidney disease.

3. Current state of knowledge

3.1.Pathophysiology of Kidney Injury in Obesity

Kidney damage associated with obesity results from a complex interplay of hemodynamic, metabolic, inflammatory, and hormonal mechanisms that act synergistically over time[18]. Rather than a single causal pathway, obesity-related kidney disease reflects the cumulative burden of overlapping insults that progressively impair renal structure and function[2].

One of the earliest and central mechanisms is obesity-induced hemodynamic alteration, particularly glomerular hyperfiltration[27]. Increased body mass leads to higher metabolic demands and enhanced renal plasma flow, resulting in afferent arteriolar dilation and elevated intraglomerular pressure[2820]. This adaptive response initially increases the glomerular filtration rate but chronically exposes the glomerular filtration barrier to mechanical stress[2930]. Sustained hyperfiltration promotes glomerulomegaly, podocyte stretch and detachment, and ultimately focal segmental glomerulosclerosis, thereby accelerating nephron loss and disease progression[28].

Activation of the renin–angiotensin–aldosterone system (RAAS) plays a pivotal role in amplifying these hemodynamic disturbances. In obesity, RAAS overactivity arises not only from systemic mechanisms but also from local production of RAAS components by adipose tissue[1928]. Elevated angiotensin II levels induce efferent arteriolar vasoconstriction, further increasing intraglomerular pressure[2931], while aldosterone promotes sodium retention, volume expansion, and fibrosis[28]. The combined activation of RAAS and the sympathetic nervous system perpetuates hypertension, proteinuria, and progressive renal injury[219].

Chronic low-grade inflammation represents another key contributor to obesity-related kidney damage[3233]. Excess adipose tissue functions as an active endocrine organ, secreting pro-inflammatory cytokines and adipokines such as leptin, resistin, and tumor necrosis factor-α,

while protective adipokines like adiponectin are reduced[234]. This pro-inflammatory milieu induces endothelial dysfunction, promotes fibrotic signaling pathways, and exacerbates podocyte injury[29]. Leptin, in particular, has been shown to stimulate transforming growth factor-β expression, contributing to glomerulosclerosis and interstitial fibrosis[35].

Insulin resistance, frequently present in obesity even in the absence of overt diabetes[20], further aggravates renal injury[18]. Insulin signaling is essential for maintaining podocyte structure and survival[19]; impaired signaling leads to cytoskeletal disruption, podocyte apoptosis, and increased glomerular permeability[22]. Additionally, insulin resistance contributes to altered renal sodium handling and afferent arteriolar dilation, reinforcing hyperfiltration and intraglomerular hypertension[2136].

Oxidative stress and lipotoxicity represent closely interconnected mechanisms in obesity-related kidney disease[27]. Expansion of adipose tissue increases the production of reactive oxygen species, which damage cellular proteins, lipids, and organelles within renal cells[197]. Simultaneously, ectopic lipid accumulation in podocytes, mesangial cells, and tubular epithelial cells induces lipotoxic injury, mitochondrial dysfunction, and activation of inflammatory pathways[22]. Perirenal and renal sinus fat may also exert direct mechanical and paracrine effects, further compromising renal perfusion and function[728].

Emerging evidence suggests that alterations in the gut microbiota and genetic susceptibility may modulate the risk and progression of obesity-related kidney disease[7]. Obesity-associated dysbiosis leads to increased production of gut-derived uremic toxins, such as indoxyl sulfate and p-cresyl sulfate, which exert pro-inflammatory and profibrotic effects on the kidneys[2028]. In parallel, genetic polymorphisms related to adiposity, inflammation, and metabolic regulation may predispose certain individuals to more severe renal manifestations[3719].

Collectively, these mechanisms highlight the multifactorial and self-reinforcing nature of kidney injury in obesity, underscoring the need for early recognition and comprehensive therapeutic strategies targeting both metabolic and renal pathways.

3.2.Clinical Presentation and Diagnosis of Obesity-Related Chronic Kidney Disease

Chronic kidney disease associated with obesity typically follows an indolent and slowly progressive course, often remaining clinically silent for many years[20]. In its early stages, patients are usually asymptomatic, and kidney involvement may go unrecognized until routine laboratory testing reveals abnormalities[5]. The most common initial clinical manifestation is the gradual development of subnephrotic proteinuria, frequently accompanied by a bland urinary sediment and preserved serum albumin levels[193822]. As the disease progresses, protein excretion

may increase and kidney function may decline, eventually leading to the typical signs and symptoms of established CKD. Importantly, obesity-related kidney disease can progress even in the absence of diabetes mellitus or overt hypertension, which may delay clinical suspicion and diagnosis[1639].

Obesity itself is formally defined by the World Health Organization as a body mass index (BMI) ≥30 kg/m² [40] and is recognized as a chronic disease associated with substantial metabolic and organ-specific complications, including kidney disease. In this context, excess adiposity exerts both direct and indirect effects on renal structure and function, contributing to glomerular hyperfiltration, albuminuria, and progressive nephron injury even before overt clinical manifestations become apparent[27].

Assessment of kidney function in patients with obesity poses several challenges. Serum creatinine, the most widely used marker of renal function, is influenced by muscle mass, dietary intake, and body composition[4142]. In individuals with obesity, increased muscle mass and creatinine production may mask early declines in glomerular filtration rate, leading to underestimation or misclassification of kidney impairment[43]. Similarly, estimated glomerular filtration rate (eGFR) equations based on creatinine or cystatin C have limitations in this population, particularly in those with severe obesity or glomerular hyperfiltration[44]. Body surface area indexing may further distort eGFR estimates, complicating accurate staging of CKD[43].

Despite these limitations, albuminuria remains a cornerstone of diagnosis and risk stratification in obesity-related CKD. Even low-grade increases in urinary albumin excretion reflect glomerular injury and are strongly associated with disease progression and cardiovascular risk[284519]. Albuminuria often precedes measurable declines in eGFR and serves as a sensitive marker of early kidney damage[369]. Regular assessment of urinary albumin excretion is therefore essential in individuals with obesity, particularly those with additional metabolic risk factors[2].

The official diagnosis of CKD follows established international guidelines, as defined by the Kidney Disease: Improving Global Outcomes (KDIGO) organization, and is based on the presence of reduced eGFR (<60 mL/min/1.73 m²) and/or markers of kidney damage, such as albuminuria, persisting for at least three months[46]. In patients with obesity, careful longitudinal assessment is required to distinguish transient functional changes from sustained structural kidney injury[41].

Growing interest has focused on the development of novel biomarkers and imaging modalities to improve early detection of obesity-related kidney disease[25]. Emerging urinary and serum biomarkers reflecting tubular injury, inflammation, and fibrosis may provide additional insight

into subclinical renal damage[4748]. Imaging techniques, including ultrasonography [49] and advanced methods to assess renal and perirenal fat deposition[19], may further enhance diagnostic accuracy. Although these approaches are not yet routinely implemented, they represent promising tools for future risk assessment and personalized management of obesity-related chronic kidney disease[20].

3.3.Therapeutic Strategies in Chronic Kidney Disease in Patients with Obesity

Management of chronic kidney disease (CKD) in patients with obesity requires a multifaceted approach targeting both excess adiposity and obesity-specific mechanisms of renal injury. Given the complex and overlapping pathways involved in obesity-related kidney disease, therapeutic strategies should combine lifestyle modification with pharmacological and, in selected cases, surgical interventions.

Reduction of body weight remains the cornerstone of therapy. Lifestyle interventions based on caloric restriction, balanced nutrition, and regular physical activity have been shown to reduce proteinuria and slow CKD progression in patients with obesity[5051]. Even modest weight loss may lead to improvements in glomerular hyperfiltration, blood pressure control, and metabolic parameters[52]. However, sustained weight reduction is difficult to achieve with lifestyle measures alone, and their long-term effectiveness is often limited. Nevertheless, lifestyle modification remains an essential foundation of care and should accompany all other treatment modalities.

Pharmacological therapy plays a central role in renal protection in obese patients with CKD. Blockade of the renin–angiotensin–aldosterone system (RAAS) with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers is a well-established strategy to reduce intraglomerular pressure, proteinuria, and fibrosis[2028]. These agents are recommended in patients with albuminuria and provide nephroprotective and cardioprotective benefits that extend beyond blood pressure reduction[46]. Mineralocorticoid receptor antagonists, particularly newer non-steroidal agents, may offer additional reductions in proteinuria with a lower risk of adverse effects, although careful monitoring is required[19].

Sodium–glucose cotransporter 2 (SGLT2) inhibitors have emerged as a key therapeutic option in CKD, including in patients with obesity[2]. Initially developed as glucose-lowering agents, SGLT2 inhibitors exert renal benefits through hemodynamic mechanisms, such as restoration of tubuloglomerular feedback and reduction of glomerular hyperfiltration[53]. Large clinical trials including CREDENCE (canagliflozin in diabetic kidney disease) and EMPA-REG OUTCOME (empagliflozin in type 2 diabetes with high cardiovascular risk), have demonstrated that these

agents slow eGFR decline, reduce albuminuria, and delay kidney failure in patients with and without diabetes[5455]. Importantly, their nephroprotective effects appear largely independent of weight loss, which is typically modest[20].

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) represent another promising therapeutic class. In addition to promoting significant and sustained weight reduction, GLP-1 RAs improve insulin sensitivity, lower blood pressure, and reduce systemic inflammation[5657]. Secondary renal outcomes from cardiovascular outcome trials suggest beneficial effects on albuminuria and renal disease progression. These findings derive from large trials such as LEADER (liraglutide in high cardiovascular risk type 2 diabetes)[58], SUSTAIN-6 (semaglutide cardiovascular safety)[59], and SELECT (semaglutide in obesity with cardiovascular disease)[60], in which analyses of secondary renal endpoints demonstrated reductions in albuminuria and a slower decline in eGFR. Collectively, these results suggest a class-wide nephroprotective effect[2]. These agents may therefore address both metabolic and renal components of obesity-related CKD, making them particularly attractive in this population.

Bariatric surgery should be considered in selected patients with severe obesity who fail to achieve adequate weight loss with conservative measures[61]. Surgical interventions can result in substantial and durable reductions in body weight, improvements in metabolic comorbidities, and, in some cases, normalization of glomerular hyperfiltration[220]. Evidence suggests that bariatric surgery may reduce proteinuria and slow CKD progression, although perioperative risks and patient selection remain critical considerations[27].

Overall, many therapeutic interventions in obesity-related CKD confer kidney protection through mechanisms that extend beyond simple weight reduction. Targeting hemodynamic stress, inflammation, and metabolic dysregulation is essential for slowing disease progression and reducing the high cardiovascular risk associated with obesity and chronic kidney disease.

3.4.Clinical Significance and Practical Implications

The growing recognition of obesity as an independent driver of chronic kidney disease has important clinical and public health implications. Given the largely asymptomatic nature of early obesity-related kidney damage, timely identification of individuals at increased risk is essential[9]. Patients with obesity, particularly those with central adiposity, metabolic syndrome, or a family history of kidney disease, should be considered a high-risk population and undergo regular screening for albuminuria and renal function abnormalities. Early detection allows for the implementation of targeted interventions at a stage when kidney damage may still be reversible or its progression significantly slowed[46].

Effective management of obesity-related CKD requires an interdisciplinary approach that addresses the complex metabolic and renal aspects of the disease. Collaboration between nephrologists, diabetologists or endocrinologists, dietitians, and primary care physicians is crucial for optimizing patient outcomes[1]. Such coordinated care enables comprehensive risk factor control, including weight management, glycemic optimization, blood pressure regulation, and cardiovascular risk reduction. Individualized nutritional counseling and lifestyle support are particularly important, as dietary habits and physical activity strongly influence both obesity and kidney disease progression[46].

From a broader perspective, the increasing prevalence of obesity-related CKD poses a significant challenge to healthcare systems worldwide. Preventive strategies targeting obesity at the population level are likely to have a substantial impact on reducing the future burden of kidney disease. Public health initiatives promoting healthy nutrition, physical activity, and early metabolic screening may help mitigate the rising incidence of CKD and its associated complications. Furthermore, increased awareness among healthcare professionals regarding the renal consequences of obesity may improve early diagnosis and appropriate referral to specialized care.

Incorporating obesity-related kidney disease into preventive frameworks emphasizes the need to move beyond traditional risk factors such as diabetes and hypertension alone. Recognizing obesity as a modifiable determinant of kidney health provides an opportunity to intervene earlier and more effectively[28]. Ultimately, integrating clinical care with preventive strategies and public health policies is essential to address the growing impact of obesity-related chronic kidney disease on individual patients and society as a whole.

4. Conclusions

Obesity has emerged as a major and often underestimated determinant of chronic kidney disease, contributing significantly to the growing global burden of renal morbidity and mortality. Beyond its well-established associations with diabetes mellitus and hypertension, excess adiposity exerts direct and independent effects on kidney structure and function[2]. As a result, obesity-related chronic kidney disease represents a distinct and increasingly prevalent clinical entity that warrants greater recognition in both nephrology practice and public health strategies[46].

The pathogenesis of kidney injury in obesity is complex and multifactorial, involving the interplay of hemodynamic stress, hormonal dysregulation, chronic low-grade inflammation, insulin resistance, oxidative stress, and lipotoxicity[20]. These mechanisms act in a synergistic

and self-perpetuating manner, ultimately leading to progressive glomerular and tubular damage. The concept of obesity-related glomerulopathy highlights the existence of a characteristic renal phenotype that may develop insidiously and remain undiagnosed until advanced stages of disease, underscoring the importance of heightened clinical awareness[46].

Recent advances in pharmacotherapy have substantially expanded the therapeutic landscape for patients with obesity and chronic kidney disease. In addition to established renin–angiotensin–aldosterone system blockade, novel agents such as SGLT2 inhibitors and GLP-1 receptor agonists have demonstrated robust nephroprotective and cardioprotective effects, many of which appear to be independent of weight reduction alone[6020]. These therapies offer new opportunities to slow disease progression and reduce cardiovascular risk in a population historically associated with poor outcomes[46].

Despite these advances, significant gaps in knowledge remain. Further research is needed to refine risk stratification, validate emerging biomarkers, and establish optimal screening strategies tailored to patients with obesity. Moreover, the development of clear, evidence-based guidelines for the early diagnosis and integrated management of obesity-related chronic kidney disease is essential. Addressing this growing clinical challenge will require a coordinated effort that combines early detection, targeted treatment, and population-level prevention aimed at reducing the impact of obesity on kidney health.

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Conceptualization: Martyna Grabowska; Karolina Grabowska; Jagoda Kubicka;

Methodology: Karolina Grabowska;

Software, Martyna Grabowska;

Check, Karolina Grabowska; Martyna Grabowska; Jagoda Kubicka;

Formal analysis, Jagoda Kubicka;

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