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REVIEW ARTICLE

Microproteinuria to Macroproteinuria as a Dynamic Indicator of Glomerular Injury in Obesity-Related Kidney Disease

Umang Bansal[1,*]

[1] Amity Institute of Pharmacy, Amity University, Noida-201313, Uttar Pradesh, India.

***Corresponding Author:** Umang Bansal
Amity Institute of Pharmacy, Amity University, Noida-201313, Uttar Pradesh, India.
Email: umang26bansal@gmail.com

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Abstract

Obesity-related kidney disease has emerged as a major contributor to the global burden of chronic kidney disease in recent decades. The rapid increase in obesity prevalence worldwide has uncovered renal complications that occur independently of diabetes mellitus and systemic hypertension. Among the earliest manifestations of obesity-induced renal injury is proteinuria, which reflects progressive dysfunction of the glomerular filtration barrier. The progression from microproteinuria to macroproteinuria represents a dynamic continuum of glomerular damage rather than a rigid clinical threshold. Microproteinuria indicates early and potentially reversible alterations in glomerular permeability caused by hemodynamic stress, adipokine imbalance, low-grade inflammation, and oxidative injury. Persistent exposure to these pathological stimuli promotes podocyte dysfunction, basement membrane remodeling, and mesangial expansion, ultimately leading to sustained protein leakage and macroproteinuria. This advanced stage of proteinuria signifies irreversible structural injury and is strongly associated with accelerated renal function decline and increased cardiovascular morbidity. Importantly, proteinuria acts not only as a biomarker of kidney damage but also as an active mediator of tubulointerstitial inflammation and fibrosis, thereby amplifying disease progression. Monitoring the transition from microproteinuria to macroproteinuria provides valuable insight into disease activity, prognosis, and therapeutic response. This review critically examines the mechanisms linking obesity to glomerular injury, with emphasis on proteinuria as a dynamic indicator of renal damage. Understanding this progression may enable earlier diagnosis, improved risk stratification, and timely interventions to mitigate obesity-related kidney disease.

1. Introduction

The global burden of obesity has increased dramatically over the past several decades, transforming it into one of the most significant public health challenges worldwide (Seidell and Halberstadt, 2015). Obesity is no longer viewed solely as a metabolic disorder but is now recognized as a systemic disease with their effects on multiple organ systems. Among these, the kidney has emerged as a particularly vulnerable target. Accumulating clinical and experimental evidence demonstrates that obesity directly contributes to renal injury and chronic kidney disease, even in the absence of diabetes mellitus (Chade and Hall, 2016; Kalaitzidis and Siamopoulos, 2011). This condition is increasingly referred to as obesity-related kidney disease and is characterized by distinct functional and structural renal alterations. The kidney plays a central role in maintaining metabolic homeostasis, fluid balance, and blood pressure regulation. In obesity, excessive caloric intake and increased body mass impose heightened metabolic demands on renal function. To compensate, the kidney undergoes adaptive changes that include increased renal plasma flow and elevated glomerular filtration rate (Phengpol et al., 2023). These early responses initially maintain homeostasis but simultaneously expose the glomerular microcirculation to sustained hemodynamic stress. Over time, these adaptive mechanisms become maladaptive, initiating a cascade of events that culminate in glomerular injury and proteinuria. Proteinuria represents one of the earliest and most sensitive clinical indicators of

renal involvement in obesity. It reflects disruption of the highly selective glomerular filtration barrier, which normally prevents significant loss of plasma proteins into the urine (Praga et al., 2000). In obese individuals, low levels of urinary protein excretion were often classified as microproteinuria. It may be detected before overt reductions in glomerular filtration rate occur (Bhatt et al., 2019; D'Agati et al., 2016). This early stage of proteinuria frequently remains clinically silent, yet it signals ongoing molecular and cellular injury within the glomerulus. Importantly, microproteinuria are potentially reversible and which makes it a critical stage for early intervention.The inflammatory and mechanical increase on the kidney increases due to the condition of obesity.

Microproteinuria gradually progresses to macroproteinuria, reflecting a transition from functional alterations to irreversible structural damage. This progression is not abrupt but occurs along a continuum, mirroring the cumulative impact of sustained glomerular injury (Bertelli et al., 2018). Macroproteinuria was associated with podocyte depletion, mesangial matrix expansion, and focal segmental glomerulosclerosis. At this stage, renal damage accelerates and the risk of progression to end-stage kidney disease rises substantially (Wang and Zhang, 2024). Despite the well-established association between obesity and proteinuria, the dynamic nature of proteinuria progression is often overemphasized in clinical practice. Proteinuria is frequently interpreted as a static marker rather than a reflection of ongoing disease activity (Gorriz and Martinez-Castelao, 2012). However, growing evidence suggests that changes in urinary protein excretion closely parallel underlying pathophysiological processes within the kidney. These include hemodynamic dysregulation, chronic inflammation, oxidative stress, and maladaptive cellular responses. Recognizing proteinuria as a dynamic indicator of glomerular injury may improve early diagnosis and guide therapeutic strategies. This review focuses on the progression from microproteinuria to macroproteinuria as a central feature of obesity-related kidney disease. Emphasis is placed on the mechanisms driving early glomerular dysfunction and the factors that promote irreversible structural injury. By integrating clinical and experimental insights, this review aims to highlight the importance of proteinuria progression as both a marker and mediator of renal disease in obesity.

2. Epidemiology and Clinical Relevance of Obesity-Related Kidney Disease

Obesity-related kidney disease has become increasingly prevalent in parallel with rising obesity rates across both developed and developing nations (Wang et al., 2008). Large epidemiological studies consistently demonstrate a positive association between body mass index and the risk of developing proteinuria and chronic kidney disease (Beddhu, 2004; Kikuchi et al., 2017). This relationship persists even after adjusting for traditional renal risk factors, underscoring the independent contribution of obesity to renal pathology. Obese individuals exhibit a higher prevalence of microproteinuria compared with normal-weight populations (Seo et al., 2016). Longitudinal cohort studies reveal that sustained obesity is associated with progressive increases in urinary protein excretion over time (Mondul et al., 2014; Yoo et al., 2020). Importantly, weight gain during adulthood further amplifies this risk. Whereas weight reduction is associated with improvements in proteinuria (Kittiskulnam et al., 2014). These observations support a causal link between excess adiposity and glomerular injury. The clinical relevance of proteinuria in obesity extends beyond renal outcomes. Even low-grade proteinuria is associated with increased cardiovascular morbidity and mortality (Delles and Currie, 2013). Microproteinuria reflects systemic endothelial dysfunction and heightened inflammatory activity, both of which contribute to atherosclerotic disease (Paisley et al., 2003). As proteinuria progresses to macroproteinuria, the risk of adverse cardiovascular events rises further and it highlights the interconnected nature of renal and cardiovascular pathology in obesity (Shah et al., 2021). Proteinuria often precedes measurable declines in estimated glomerular filtration rate in obese individuals. This temporal relationship underscores its value as an early marker of kidney injury. Microproteinuria identifies a subclinical stage of disease during which structural damage may still be limited. Regular screening for urinary protein excretion in obese populations therefore provides an opportunity for early detection and intervention. Recognizing proteinuria progression allows clinicians to stratify risk and monitor disease evolution more effectively.

3. Renal Hemodynamic Adaptations in Obesity

Obesity induces profound changes in renal hemodynamics that play a central role in the initiation of glomerular injury. Increased metabolic demand leads to enhanced sodium reabsorption in the proximal tubule, which reduces sodium delivery to the macula densa (Vallon et al., 2002). This process activates tubuloglomerular feedback mechanisms, resulting in afferent arteriolar dilation and increased glomerular filtration rate. The consequent state of hyperfiltration represents an

early adaptive response but simultaneously exposes the glomerulus to elevated intraglomerular pressure. Sustained hyperfiltration increases mechanical stress on glomerular capillaries and the filtration barrier. Endothelial cells experience shear stress, while podocytes are subjected to increased tensile forces. These mechanical insults disrupt cellular structure and promote permeability defects. Microproteinuria emerges as a functional manifestation of these hemodynamic disturbances. Over time, persistent hyperfiltration accelerates structural injury and driving the progression toward macroproteinuria (Hang et al., 2025; Sharma and Smyth, 2021).

4. Glomerular Filtration Barrier Dysfunction in Obesity

The integrity of the glomerular filtration barrier is essential for preventing excessive protein loss in urine. This barrier is composed of fenestrated endothelial cells, the glomerular basement membrane, and highly specialized podocytes interconnected by slit diaphragms (Daehn and Duffield, 2021; Menon et al., 2012). In obesity-related kidney disease, each component of this barrier undergoes progressive structural and functional alterations. These changes evolve gradually and closely parallel the transition from microproteinuria to macroproteinuria (Figure 1 & Table 1). Endothelial dysfunction represents an early event in obesity-induced glomerular injury. Excess adiposity is associated with reduced nitric oxide bioavailability and degradation of the endothelial glycocalyx. These changes increase capillary permeability and impair selective filtration. Simultaneously, thickening of the glomerular basement membrane alters its charge and size selectivity. Podocytes respond to mechanical and metabolic stress through cytoskeletal reorganization, leading to foot process effacement.Together, these alterations permit abnormal protein passage into the urinary space and initially manifesting as microproteinuria.

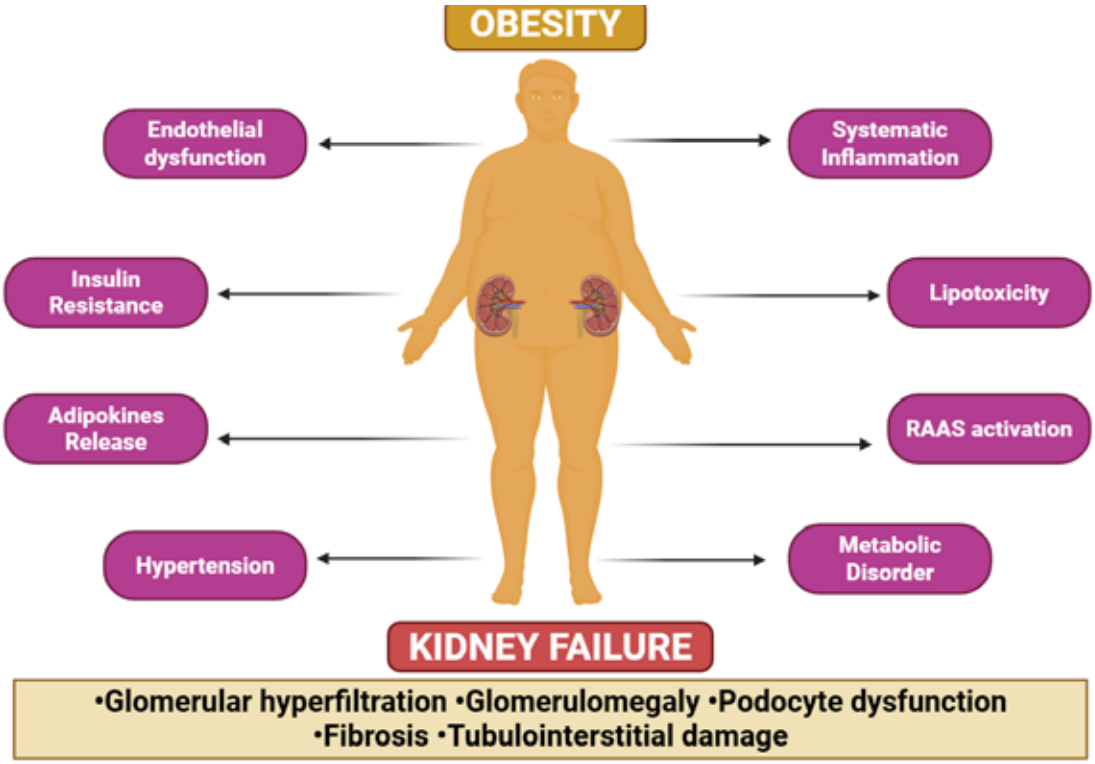

Figure 1: Mechanistic links between obesity and progressive kidney injury leading to renal failure

5. Podocyte Stress, Slit Diaphragm Alterations, and Inflammatory Signaling

Podocytes are highly specialized epithelial cells that play a decisive role in preserving the selective permeability of the glomerular filtration barrier. In obesity-related kidney disease, podocytes are exposed to persistent biomechanical and metabolic stress. Glomerular hypertrophy and sustained intraglomerular hypertension stretch podocyte cell bodies and foot processes, leading to cytoskeletal instability (Du et al., 2025). This mechanical strain disrupts actin filament organization and interferes with intracellular signaling pathways that maintain podocyte architecture. At the molecular level, obesity is associated with altered expression and distribution of key slit diaphragm proteins, including nephrin, podocin, and CD2-associated protein. These proteins are essential for maintaining filtration slit integrity and for transducing survival signals within podocytes. Downregulation or mislocalization of slit diaphragm components compromises barrier selectivity, allowing albumin and other plasma proteins to traverse the filtration barrier. In early stages, these alterations are

predominantly functional and may partially reverse with correction of hemodynamic stress, corresponding clinically to microproteinuria.

Chronic low-grade inflammation further exacerbates podocyte injury in obesity. Adipose tissue functions as an active endocrine organ, and releases adipokines that directly influence renal cells. Elevated leptin levels stimulate mesangial cell proliferation and extracellular matrix production. It significantly increases glomerular size and filtration pressure. In contrast, adiponectin, which normally exerts anti-inflammatory and cytoprotective effects was reduced in obese individuals. Adiponectin deficiency impairs AMP-activated protein kinase signaling and increases vulnerability to oxidative and inflammatory injury. Pro-inflammatory cytokines such as tumor necrosis factor-α and interleukin-6 activate nuclear factor-κB signaling in podocytes and glomerular endothelial cells (Kovalik et al., 2024). This activation disrupts cytoskeletal stability, alters slit diaphragm protein expression, and increases glomerular permeability. Inflammatory mediators also promote immune cell recruitment into renal tissue, sustaining a local pro-injury environment. With persistent exposure to these stimuli, podocyte stress progresses to detachment and apoptosis. Since podocytes have limited regenerative capacity, and their loss produces permanent defects in the filtration barrier. It marks as a critical transition toward irreversible injury and macroproteinuria.

6. Oxidative Stress and Mitochondrial Dysfunction

Oxidative stress represents a central mechanism linking obesity to progressive glomerular injury. Excess caloric intake and lipid accumulation increase mitochondrial workload in renal cells. As mitochondrial electron transport becomes inefficient, reactive oxygen species generation increases substantially. Antioxidant defense systems become overwhelmed and resulted in oxidative damage to proteins, lipids, and nucleic acids within glomerular cells. Podocytes and endothelial cells are particularly vulnerable to oxidative injury due to their high metabolic demands. Reactive oxygen species disrupt cytoskeletal organization and impair signaling pathways essential for cell survival and barrier maintenance. Oxidative stress also amplifies inflammatory signaling, reinforcing nuclear factor-κB activation and cytokine production. This interaction creates a self-perpetuating cycle of oxidative and inflammatory injury. In early stages, oxidative damage contributes to subtle increases in glomerular permeability and manifesting clinically as microproteinuria. With sustained oxidative stress, cumulative cellular injury leads to irreversible structural alterations, including podocyte loss and basement membrane remodeling. These changes promote the transition to macroproteinuria and accelerate renal function decline.

7. Progression from Microproteinuria to Macroproteinuria

The transition from microproteinuria to macroproteinuria reflects the cumulative burden of sustained glomerular injury in obesity-related kidney disease. Microproteinuria represents a disease stage dominated by functional alterations in filtration barrier permeabilitywith relatively preserved structural integrity. At this stage, podocyte was disrupted but not completely lost, and therapeutic interventions targeting hemodynamic stress, inflammation, and oxidative injury may stabilize or reverse protein leakage. Persistent metabolic overload, chronic inflammation, and oxidative stress progressively exhaust compensatory mechanisms. Podocyte detachment and apoptosis reduce podocyte density below a critical threshold which leads to denuded areas of the glomerular basement membrane. Mesangial expansion further compromises capillary architecture (Menon, 2014; Trimarchi, 2020). As a result, protein filtration increases quantitatively and macroproteinuria develops. This stage signifies established structural damage and is strongly associated with accelerated decline in glomerular filtration rate and increased risk of end-stage kidney disease. Proteinuria therefore functions as a dynamic indicator that mirrors the underlying pathological evolution of obesity-related kidney disease, rather than a static marker of renal damage.

Table 1: Progression of Albuminuria as a Dynamic Indicator of Glomerular Injury in Obesity-Related Kidney Disease

Albuminuria Category (KDIGO)	Dominant Pathobiological Changes	Key Renal Structures Affected	Clinical Interpretation	Prognostic Implication
Normal to mildly increased albumin excretion (A1)	Adaptive glomerular hyperfiltration, preserved barrier selectivity	Glomerular endothelium	Subclinical renal stress without overt protein leakage	Potentially reversible with early intervention

Moderately increased albuminuria (A2)	Increased permeability of filtration barrier, early podocyte stress	Podocytes, endothelial glycocalyx	Earliest detectable marker of glomerular injury	Predictor of progressive renal dysfunction
Persistent moderately increased albuminuria (A2)	Podocyte foot process effacement, slit diaphragm instability	Podocytes, mesangial cells	Ongoing glomerular damage	Increased risk of transition to severe disease
Transition from A2 to A3	Amplified inflammation and oxidative stress, loss of compensatory mechanisms	Podocytes, tubular epithelial cells	Shift from functional to structural injury	Critical window for disease-modifying therapy
Severely increased albuminuria (A3)	Basement membrane disruption, podocyte detachment	Glomerular basement membrane	Established glomerular pathology	Rapid decline in renal function
Advanced A3 with fibrosis	Glomerulosclerosis and tubulointerstitial fibrosis		Progressive chronic kidney disease	High risk of end-stage kidney disease and cardiovascular events

8. Structural Remodeling, Glomerulosclerosis, and Tubulointerstitial Injury

Advanced obesity-related kidney disease was characterized by extensive structural remodeling of both glomerular and tubulointerstitial compartments. Glomerulomegaly develops as an adaptive response to chronic hyperfiltration, but this enlargement increases mechanical stress on the filtration barrier (Chagnac and Rozen-zvi, 2019). Mesangial matrix expansion distorts capillary architecture and reduces effective filtration surface area, further impairing glomerular function. Focal segmental glomerulosclerosis emerges as podocyte depletion becomes pronounced. Sclerotic lesions replace functional capillary loops, which leads to persistent macroproteinuria and progressive nephron loss. Once established, glomerulosclerosis is largely irreversible and strongly correlates with poor renal outcomes. Proteinuria also contributes directly to tubulointerstitial injury. Filtered proteins are reabsorbed by proximal tubular epithelial cells, activating inflammatory and profibrotic signaling pathways. Nuclear factor-κB and transforming growth factor-β signaling promote cytokine release, fibroblast activation, and extracellular matrix deposition. Progressive tubulointerstitial fibrosis disrupts nephron structure and reduces functional renal mass.

9. Clinical Implications and Therapeutic Considerations

The degree and progression of proteinuria have important clinical implications in obesity-related kidney disease. Microproteinuria identifies individuals at increased risk for renal and cardiovascular complications, while macroproteinuria predicts rapid progression toward end-stage kidney disease. Serial monitoring of urinary protein excretion provides insight into disease activity, prognosis, and therapeutic response. Weight reduction improves renal hemodynamics, decreases inflammatory burden, and reduces proteinuria. Pharmacological interventions targeting the renin–angiotensin–aldosterone system lower intraglomerular pressure and stabilize podocyte structure. Emerging therapies aimed at reducing oxidative stress and preserving podocyte integrity may further attenuate proteinuria progression. Early intervention during the microproteinuric stage remains critical for preserving renal function.

10. Conclusion

The progression from microproteinuria to macroproteinuria represents a continuous and dynamic indicator of glomerular injury in obesity-related kidney disease. This transition reflects the combined effects of hemodynamic stress, chronic inflammation, oxidative damage, and structural remodeling. Proteinuria functions not only as a marker but also as a mediator of renal disease progression. Early recognition and timely intervention are essential to mitigate obesity-induced renal injury and reduce the long-term burden of chronic kidney disease.

Declarations

Ethics approval statement

Not applicable

Consent to participate

Not applicable

Consent to publish

Not applicable

Data Availability Statement

The data are available from the corresponding author upon reasonable request

Competing Interests

The authors declare that they have no conflict of interest

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Author contribution

U.B solely prepared, written and edited the whole manuscript.

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