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Renal Biomarkers and Early Kidney Injury in Women with Polycystic Ovary Syndrome: A Narrative Review

Abstract
Polycystic ovary syndrome (PCOS) has been a highly studied endocrine-metabolic condition in women of reproductive age, which is traditionally known to have reproductive and metabolic effects. There is also emerging evidence that PCOS may also have early renal involvement that may put the affected women at risk of having chronic kidney disease in them later in life. This narrative review will synthesize the existing evidence on pathophysiological connections between PCOS and renal dysfunction, critically assess the limitations of traditional renal biomarkers, and the use of emerging biomarkers in the early detection of kidney damage in women with PCOS. The synergistic effects on the development of subtle glomerulonephritis and tubular damage occur as a result of insulin resistance in PCOS, hyperandrogenism, obesity, persistent low-grade inflammation, oxidative stress, endothelial dysfunction, and renin-angiotensin-aldosterone system activation. The traditional renal markers, serum creatinine, blood urea nitrogen, and estimated glomerular filtration rate using creatinine, are commonly insensitive to such early alterations and can be observed to be within normal ranges despite continued stress to the renal system. New biomarkers, such as urinary albumin -to -creatinine ratio, cystatin C, neutrophil gelatinase-associated lipocalin, and kidney injury molecule-1, are more sensitive in detecting early glomerular permeability changes, tubular injury, and microvascular dysfunction, in contrast. Risk stratification and targeted interventions in women with PCOS could be achieved by early detection of renal involvement by sensitive biomarkers. The introduction of renal biomarker evaluation as part of the routine clinical evaluation, in combination with lifestyle modification and metabolic optimization, could aid in avoiding the development of overt renal disease. More longitudinal and mechanistic research is needed to confirm these biomarkers and determine their prognostic potential in determining renal outcomes in the long term in PCOS.

1. Introduction

Polycystic ovary syndrome (PCOS) is an extremely widespread endocrine-metabolic syndrome among women of reproductive age and is linked with hyperandrogenism, ovulatory impairment, and polycystic ultrasonic appearance of the ovary (1). Besides the reproduction manifestations, PCOS is also becoming increasingly relevant as an overall metabolic disease that is associated with insulin resistance, obesity, dyslipidemia, chronic low-grade inflammation and endothelial dysfunction (2). Such comorbid deformities expose women with PCOS to the risk of cardiometabolic conditions, including hypertension and type 2 diabetes mellitus (3). The renal involvement of PCOS has become a major interest over the last few years, particularly, the danger of early kidney damage as a predisposing factor to the overt chronic kidney disease (CKD) (4). Traditional renal biomarkers, such as serum creatinine and the estimated glomerular filtration rate (eGFR) are not likely to detect the subtle nature of renal impairment (5). Hence, novel renal biomarkers have taken a leading role in the screening of early

kidney injuries in PCOS women (6).

Serum creatinine, blood urea nitrogen, and creatinine-based estimated glomerular filtration rate (eGFR) are conventional renal function tests that are commonly used in clinical practice to determine the health of the kidney (7). Nevertheless, these are mainly associated with global filtration capacity and are not very sensitive to early or subclinical renal injury (8). Renal impairment in young women with PCOS might firstly appear as either functional or microvascular changes and not as a decrease in filtration resulting in the normalization or even elevation of eGFR values as the result of glomerular hyperfiltration (9). Therefore, the use of traditional markers might not adequately calculate the risk of renal in this population and act as a delaying factor (10). Innovations in the field of biomarkers have presented new renal biomarkers that can indicate early glomerular and tubular damage before irreversible structural damage is manifested (11). Urinary albumin- to-creatinine ratio, cystatin C, neutrophil gelatinase-associated lipocalin, and kidney injury molecule-1 are biomarkers that are used to give a mechanistic understanding of early renal stress, endothelial dysfunction, and tubular injury (12). Such indicators can be especially applicable in PCOS when the metabolic and inflammation processes can selectively target the renal microvasculature and tubular spaces (13).

Due to the fact that PCOS has high prevalence and is related to metabolic morbidity over a long period of time, the renal implication of this disease is clinically important (14). Early renal susceptibility detection presents a chance to implement preventive measures that help to narrow the development of overt CKD and minimize the risks of cardiovascular complications in the long term (15). It is against this backdrop that the current narrative review is expected to summarize existing evidence regarding the pathophysiological connections between PCOS and renal dysfunction, critically discuss the shortcomings of traditional renal markers, and identify the growing importance of new renal biomarkers in the early detection and risk stratification of kidney injury in women with PCOS (16).

Table 1: Clinical and Metabolic Characteristics of Polycystic Ovary Syndrome Relevant to Renal Risk

PCOS Characteristic	Key Clinical Features	Underlying Pathophysiology	Potential Impact on Renal Function
Insulin resistance	Hyperinsulinemia, impaired glucose tolerance, increased HOMA-IR	Reduced insulin sensitivity in peripheral tissues; compensatory hyperinsulinemia	Glomerular hyperfiltration, increased intraglomerular pressure, early albumin leakage (17)
Hyperandrogenism	Elevated serum androgens, hirsutism, acne	Androgen excess–induced oxidative stress and sympathetic activation	Renal microvascular injury, glomerular hypertrophy, pro-fibrotic signaling (18)
Obesity (central/visceral)	Increased waist circumference, elevated BMI	Adipose tissue dysfunction, lipotoxicity, altered adipokine secretion	Renal inflammation, tubular lipid accumulation, progression toward CKD (19)
Dyslipidemia	Elevated triglycerides, reduced HDL-cholesterol	Atherogenic lipid profile, oxidative lipid modification	Endothelial dysfunction, renal microvascular damage (20)
Chronic low-grade inflammation	Elevated hs-CRP, IL-6, TNF-α	Persistent inflammatory cytokine release	Tubular epithelial injury, interstitial fibrosis (21)
Oxidative stress	Increased reactive oxygen species, reduced antioxidant capacity	Mitochondrial dysfunction and redox imbalance	Damage to glomerular basement membrane and tubular cells (22)
Endothelial dysfunction	Reduced nitric oxide bioavailability, increased endothelin-1	Impaired vasodilation and microcirculatory regulation	Reduced renal perfusion, microalbuminuria (23)
RAAS activation	Increased angiotensin II and aldosterone activity	Insulin- and adiposity-driven RAAS upregulation	Glomerulosclerosis, tubular inflammation, renal fibrosis (24)
Hypertension (early or masked)	Elevated systolic or diastolic blood pressure	Volume expansion, vascular stiffness	Accelerated renal injury and nephron loss (25)

2. Pathophysiological Links Between PCOS and Renal Dysfunction

The correlation between polycystic ovary syndrome (PCOS) and renal dysfunction is also becoming known

to be a meeting point between endocrine, metabolic, vascular and inflammatory processes (26). Instead of single causative pathway, the renal involvement in PCOS indicates the cumulative effect of chronically acted stressors of metabolism on the glomerular and tubular systems (27).

2.1. Hyperinsulinemia and Insulin Resistance

The main characteristic that PCOS has is insulin resistance, and it is the key determinant of renal pathophysiology. Compensatory hyperinsulinemia favors increased sodium reabsorption of the proximal tubules resulting into volume expansion and increased intraglomerular pressure (28). Constant hyperfiltration is a primary adaptive reaction, but ultimately leads to injury of the glomerulus, mesangial proliferation, and albumin exudation. Insulin resistance too affects the bioavailability of nitric oxide, which is a factor in renal endothelial dysfunction and the modulating effect of autoregulation of renal blood flow (29).

2.2. Hyperandrogenism and Renal Hemodynamics

Excess androgens which are a characteristic of PCOS have both direct and indirect impact on the kidney. Androgens activate oxidative stress signaling and augment the activity of the sympathetic nervous system, therefore, having an effect on renal vascular tone. There is experimental evidence to support that androgen excess increases glomerular hypertrophy and facilitates pro-fibrotic signalling in renal tissues. Chronic hyperandrogenism in women with PCOS, therefore, may be a major contributor to vulnerability to renal microvascular damage, especially in the presence of metabolic stress (30).

2.3.Renin-Angiotensin-Aldosterone System Activation

PCOS is often linked with inappropriate renin-angiotensin-aldosterone system (RAAS) even in normotensive patients. Adipose tissue-derived angiotensinogen and hyperinsulinemia are involved in the the process of RAAS upregulation leading to efferent arteriolar vasoconstriction and glomerular capillary pressure increase. Persistent RAAS leads to hastening glomerulosclerosis, tubular inflammation, and increases oxidative stress, and hence connects PCOS with progressive renal dysfunction (31).

2.4. Malignant Low-Grade Inflammation

A low-grade inflammatory condition of PCOS women is sustained by high levels of circulating cytokines and acute phase proteins. These inflammatory mediators cause endothelial stimulation and raise vascular permeability, as well as in the renal microcirculation (32). Tubular epithelial cell injury and apoptosis is also caused by inflammatory signaling which preconditions dysfunction of the tubules early. With time, remodeling caused by inflammation process leads to interstitial fibrosis and nephron integrity (33).

2.5. Oxidative Stress/Mitochondrial Dysfunction

There is a common process linking insulin resistance, hyperandrogenism, and inflammation in PCOS which is oxidative stress. Overproduction of reactive oxygen species destroys glomerular basement membranes and tubular epithelial cells, causing impairment of the processes of filtration and reabsorption. The dysfunction of the mitochondria of renal cells increases the energy imbalance and exposure to damage. Such redox abnormalities are also of special interest in early, asymptomatic phases of renal damages, when structural alterations are not evident to date (34).

2.6. Obesity, Lipotoxicity, and Adipokines

A large percentage of women having PCOS bear central obesity, which itself causes renal damage. The dysfunction of the adipose tissue can result in a change of adipokines secretions of leptin and adiponectin. High levels of leptin use sympathetic stimulation and kidney fibrosis, whereas decreased adiponectin interferes with anti-inflammatory and renal protective responses. Moreover, the lipotoxicity caused by the deposition of ectopic lipids in renal tissue worsens the performance of glomeruli and tubules (35).

2.7. Endothelial Dysfunction and Microvascular Injury

The dysfunction of the endothelium is a very crucial mediating factor connecting the metabolic disorders of the whole body with the renal injury in PCOS (36). High levels of endothelin-1, low levels of nitric oxide, and elevated levels of oxidative stress all damage the renal microvascular perfusion. This microvascular damage is preceded by the actual loss of nephrons and can be clinically revealed as microalbuminuria and minor loss of renal reserve (37).

2.8. Combined Pathophysiological Viewpoint

There is synergistic interaction between insulin resistance, androgen excess, RAAS activation, inflammation, oxidative stress, and obesity in the establishment of a pro-injury renal milieu in PCOS. These processes give reasons why early stages of kidney damage may be observed in young women who do not have the usual traditional kidney risk factors like diabetes or chronic high blood pressure. This combined pathophysiology provides a clear insight into the significance of early renal monitoring and specific metabolic treatment of women with PCOS (38).

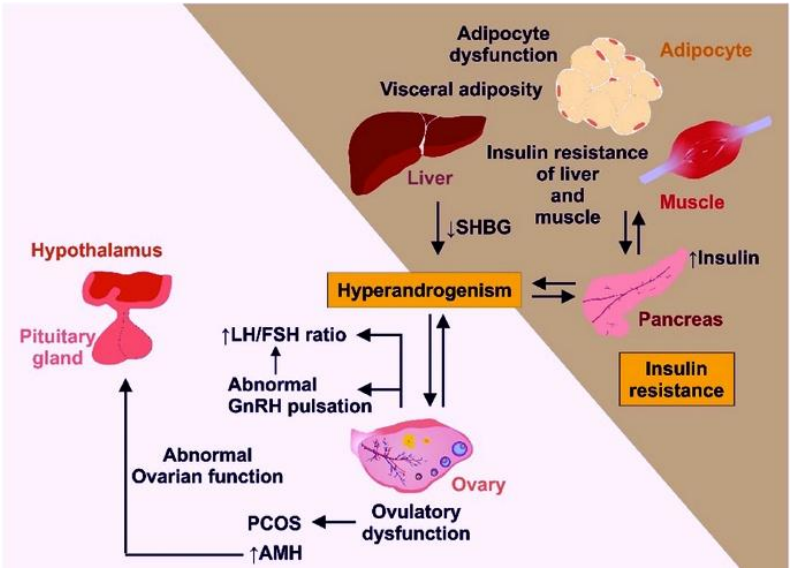

Figure 1: Integrated pathophysiological mechanisms linking PCOS to early renal dysfunction

The schematic **Figure 1** shows how the endocrine and metabolic abnormalities typical of PCOS such as insulin resistance, hyperandrogenism, and obesity are interrelated and lead to the secondary impact on the structure and function of the kidneys. The resistance to insulin and the consequent hyperinsulinemia stimulates sodium retention by the kidneys and hyperfiltration at the glomerulus and the renin-angiotensin-aldosterone system (RAAS) (39). Hyperandrogenism, persistent low-grade inflammation, and imbalance of adipokines augment oxidative stress and dysfunction of the endothelium in the renal microvasculature. These pathways synergistically cause glomerular changes in permeability, tubular epithelial pressure, and premature nephron damage, which in the absence of chronic kidney disease is clinically characterized by microalbuminuria and subclinical renal failure (40).

3. Conventional Renal Markers and Their Limitations

Traditional renal biomarkers continue to be the basis of the common evaluation of kidney functioning in clinical and research applications. These parameters mostly indicate the global glomerular filtration capacity and excretion of nitrogenous wastes. Nevertheless, in the context of polycystic ovary syndrome (PCOS), in which a renal involvement is so subtle, functional, and early onset, conventional markers exhibit critical limitations of diagnostic and interpretative use (41).

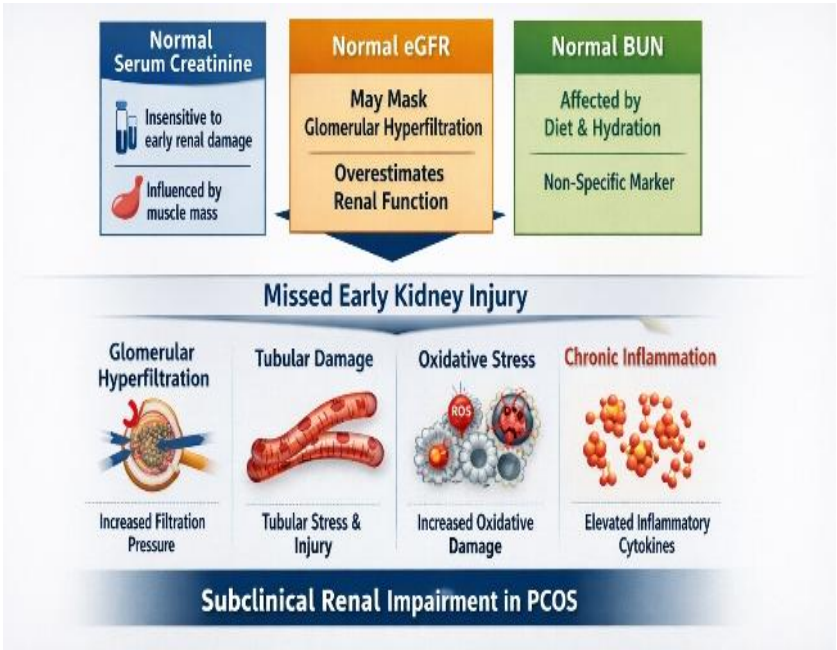

Figure 2: Limitations of Renal Markers in PCOS

3.1 Serum Creatinine

The most commonly used measures of renal functioning are serum creatinine which are used to estimate glomerular filtration rate. Creatinine does not increase until after significant nephron loss, which is usually more than 4050% loss in functional renal mass. The serum creatinine level in young women with PCOS tends to stay within the normal reference range

even in the case of glomerular hyperfiltration, endothelial dysfunction, or initial tubular damage. Besides, non-renal factors, including muscle mass, age, diet, and physical activity, have a strong impact on the creatinine concentration. Serum creatinine should not be relied upon to diagnose early renal failure because most women with PCOS, due to relatively low muscle mass or shifting body composition, have serum creatinine that is low (42).

3.2. Blood Urea Nitrogen (BUN)

Blood urea nitrogen indicates the liver production of urea and excretion by the kidneys. BUN is a nonspecific indicator of renal function although it is frequently measured. Protein consumption, hydration, hepatic functioning and catabolic conditions influence its levels. Metabolic changes in PCOS, including insulin resistance and obesity, have the potential to alter protein metabolism, and this decreases the specificity of BUN in the detection of early kidney damage. The level of BUN is generally normal until renal dysfunction takes place in the advanced stages as is the case with creatinine (43).

3.3. Estimated Glomerular Filtration Rate (eGFR)

Central to the diagnosis and staging of chronic kidney disease is estimated GFR which is estimated using equations based on creatinine. Nevertheless, eGFR lacks some limitations in PCOS populations. Early renal contribution to PCOS can be glomerular hyperfiltration instead of low filtration leading to normal or even higher eGFR levels (44). This effect may conceal intrinsic stress on the kidney and postpone the detection of foci of progressive damage (**Figure 2**). Also, the use of creatinine-based equations had not been explicitly tested in young and hormonally active women with metabolic disorders, which diminished their sensitivity in this group (45).

3.4. Creatinine Clearance

Timed urine collections are used to determine creatinine clearance, which is a more accurate assessment of the actual GFR as opposed to serum creatinine. However, it can be susceptible to gathering mistakes and overevaluation because of tubular secretion of creatinine. Its variability and complexity do not allow its routine application in clinical practice, especially in screening early renal changes in PCOS (46).

3.5. Failure to identify Tubular Injury.

One of the fundamental drawbacks of the traditional renal markers is that they fail to show tubular or interstitial damage. Kidney injury in PCOS can be early, and it is possible that tubular epithelial stress, oxidative damage and microvascular dysfunction are the main problems, as opposed to an apparent loss of glomerular filtration. Conventional markers are not sensitive in terms of detecting subclinical renal injury because they fail to capture these processes (47).

3.6. Low Correlation with Metabolic and Inflammatory Stress.

Traditional markers of renal offer a little information on the metabolic and inflammatory environment that is leading to renal damage in PCOS. They fail to reflect the input of insulin resistance, hyperandrogenism, chronic inflammation, oxidative stress, or endothelial dysfunction, which are major pathophysiological mechanisms involved in the early renal damage. This means that the use of these markers can inadequately estimate the renal risk among women with PCOS (48).

3.7. Clinical Implications

These drawbacks of traditional renal markers highlight the necessity of biomarkers to supplement traditional ones in order to identify early glomerular and tubular damage in PCOS. Combining classic parameters with delicate signs like urinary albumin-to-creatinine ratio, cystatin C, and tubular injury parameters will be able to enhance the first-time risk stratification, inform the right moment to act, and possibly eliminate the progression to chronic kidney disease.

Table 2: Conventional Renal Markers and Their Diagnostic Limitations in PCOS

Conventional Marker	Physiological Basis	Strengths	Key Limitations in PCOS
Serum creatinine	Product of muscle metabolism, filtered by glomeruli	Widely available, inexpensive	Insensitive to early renal injury; influenced by muscle mass, diet, age; remains normal despite hyperfiltration (49)
Blood urea nitrogen (BUN)	Hepatic urea production and renal excretion	Simple indicator of nitrogen balance	Affected by hydration, protein intake, liver function; poor specificity for early kidney injury (19)
Estimated GFR (eGFR)	Creatinine-based filtration estimation	Standard CKD staging tool	Early PCOS may show hyperfiltration; equations not validated in young metabolically active women (50)
Creatinine clearance	Timed urinary creatinine excretion	Closer approximation of true GFR	Collection errors; overestimation due to tubular secretion; impractical for routine screening (51)
Routine urinalysis	Detection of gross proteinuria or hematuria	Useful for advanced disease	Unable to detect microalbuminuria or tubular stress (52)
Global limitation	Focus on filtration capacity	Useful in advanced CKD	Fails to detect tubular injury, inflammation, oxidative stress, or microvascular damage (53)

4. Emerging Renal Biomarkers in PCOS

4.2.Microalbuminuria and Albumin-to-Creatinine Ratio

Microalbuminuria is a long-proven indicator of renal tissue injury and endothelial diseases. Other studies have documented increased urinary albumin excretion in women with PCOS relative to age matched controls, despite absence of apparent diabetes of hypertension. High albumin-to-creatinine ratio (ACR) in PCOS is indicative of the initial alterations in glomerular permeability, and strongly correlates with insulin resistance, hypertension and inflammatory condition (54).

4.3 Cystatin C

Cystatin C is a protein of low molecular weight that is freely filtered at the glomerulus and it is not as dependent on muscle mass as creatinine. Women with PCOS have been observed to have high levels of serum cystatin C indicating a premature deterioration of the renal filtration capacity (55). Significantly, the cardiometabolic risk factors are also correlated with cystatin C, which supports the dual use of cystatin C as a renal and cardiovascular biomarker in PCOS.

4.4.Neutrophil Gelatinase-Associated lipocalin (NGAL)

NGAL is a novel tubular injury and oxidative stress biomarker. High urinary and serum NGAL levels in patients with PCOS suggest the presence of early initial tubular stress despite eGFR being maintained. NGAL expression is induced during inflammatory and metabolic conditions, which is especially important in conditions of vulnerability of the kidney associated with PCOS (56).

4.5. Kidney Injury Molecule-1 (KIM-1)

After injury KIM-1 is a transmembrane protein expressed in proximal tubular cells. Metabolic disorders have been associated with subclinical tubular damage at increased levels of urinary KIM-1. Raised KIM-1 is suggestive of early tubular damage in PCOS that may be mediated by insulin resistance, oxidative stress and androgen overload (57).

4.6.Markers of Oxidative Stress and Inflammatory Stress.

Red flags like high-sensitivity C-reactive protein (hs-CRP), interleukin- 6, tumor necrosis factor-a and indicators of oxidative stress are indirectly related to the risk of renal injury. Their increased levels of PCOS justify the idea of the role of systemic inflammation and redox imbalance in causing early renal injury (58).

5. Conclusion

The polycystic ovary syndrome is being viewed as a systemic disorder with significant renal implications that are not confined to the reproductive and metabolic manifestations. An emerging body of evidence suggests that women with PCOS are prone to premature kidney damage as a result of a complicated interaction between insulin resistance, hyperandrogenism, obesity, chronic, low-grade inflammatory status, oxidative stress, endothelial dysfunction, and renin-angiotensin-aldosterone system. These pathophysiological processes have the potential to cause subtle injury to the glomeruli and tubules even before the more traditional signs of renal dysfunction vary (59).

Conventional renal biomarkers such as serum creatinine, blood urea nitrogen and creatinine-based estimated glomerular filtration rate are not sensitive to identify early and subclinical renal injury in this group. On the contrary, the new renal biomarkers, including urinary albumin-to-creatinine ratio, cystatin C, neutrophil gelatinase-associated lipocalin, and kidney injury molecule-1, provide useful information on early alterations of glomerular permeability, tubular strain, and microvascular damage among women with PCOS (60).

By including sensitive renal biomarkers in the clinical assessment of PCOS, it is possible to identify the vulnerability of the renal condition in time, more precisely stratify risks, and prevent and treat it in a timely manner. Changes in lifestyle, treatment aimed at metabolic optimization, and specific pharmacological changes can contribute to the prevention of organ risks and the enhancement of the long-term results (61). These biomarkers should be validated in future longitudinal and mechanistic studies to determine their clinical relevance thresholds as well as the prognostic power of these biomarkers in chronic kidney disease progression. An interdisciplinary strategy based on biomarkers can eventually improve renal and cardiometabolic outcomes in PCOS women (62).

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