

Cystatin-C and Urinary Albumin-to-Creatinine Ratio as Complementary Markers of Renal Involvement in Adults with Metabolic Syndrome: A Hospital-Based Comparative Study

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Abstract

Background: Metabolic syndrome (MetS) is associated with an increased risk of renal dysfunction, but conventional measures of kidney function may not fully characterize early renal involvement. Cystatin-C provides an alternative marker of glomerular filtration, while urinary albumin and the urinary albumin-to-creatinine ratio (UACR) provide information on glomerular permeability and albumin leakage. Evaluation of these complementary markers may provide a broader assessment of renal involvement in individuals with MetS. **Material and Methods:** We conducted an analytical cross-sectional comparative study among 360 adults, including 180 participants with MetS and 180 age- and sex-matched controls. We defined MetS according to the International Diabetes Federation 2006 criteria. We assessed serum cystatin-C, urinary albumin, and UACR alongside clinical and anthropometric parameters. We evaluated associations between waist circumference (WC), waist-to-hip ratio (WHR), and renal parameters using correlation analysis. We considered statistical significance at $p < 0.05$. **Results:** Serum cystatin-C was significantly higher among participants with MetS than controls (1.08 ± 0.22 vs 0.92 ± 0.16 mg/L; $t = 8.29$, $p < 0.001$). Urinary albumin was also significantly higher in the MetS group (24.53 ± 16.86 vs 13.04 ± 8.38 mg/L; $t = 8.19$, $p < 0.001$). Similarly, UACR was significantly elevated among participants with MetS (38.45 ± 25.47 vs 18.72 ± 10.58 mg/g; $t = 9.60$, $p < 0.001$). Most correlations between anthropometric indices and renal parameters were not statistically significant. We observed a weak inverse correlation between WHR and cystatin-C ($r = -0.193$, $p = 0.009$). Urinary albumin and UACR did not differ significantly across the 2-, 3-, and 4-component MetS subgroups. **Conclusion:** Adults with metabolic syndrome demonstrated significantly higher cystatin-C, urinary albumin and UACR than controls. These findings suggest that renal involvement associated with MetS may involve both altered filtration and increased urinary albumin excretion. Cystatin-C and urinary albumin assessment may therefore provide complementary information alongside conventional renal function testing. However, the cross-sectional design prevents inference of causality or chronicity.

Keywords: Metabolic syndrome; cystatin-C; albuminuria; urinary albumin; UACR; renal involvement; kidney function; glomerular filtration; chronic kidney disease.

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INTRODUCTION

Metabolic syndrome (MetS) is a cluster of interrelated metabolic abnormalities that includes central obesity, hyperglycaemia, hypertension and dyslipidaemia. Although its traditional clinical significance has been related primarily to cardiovascular disease and type 2 diabetes mellitus, increasing evidence indicates that MetS is also associated with renal abnormalities.^[1-3]

The kidney is particularly vulnerable to the haemodynamic, metabolic and inflammatory consequences of obesity, insulin resistance, hypertension and dysglycaemia. The mechanisms linking MetS with renal dysfunction are multifactorial. They may include altered intraglomerular haemodynamics, endothelial dysfunction, oxidative stress, chronic low-grade inflammation, activation of the renin-angiotensin-aldosterone system and abnormal adipokine signalling.^[4,5] Renal involvement may occur before overt clinical manifestations of CKD become apparent. Consequently, relying on a single conventional renal marker may not fully

capture early renal abnormalities.

Serum creatinine remains the most commonly used biochemical marker for estimation of GFR. However, creatinine concentration is affected by muscle mass, age, dietary intake and other non-renal factors. Cystatin-C has attracted considerable interest as an alternative filtration marker because it is less dependent on muscle mass than creatinine. Importantly, current KDIGO guidance recommends considering cystatin-C in

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appropriate circumstances and supports combined creatinine- and cystatin-C-based GFR estimation when greater accuracy is required.^[6]

Researchers have previously investigated the potential usefulness of cystatin-C in MetS. Kadam et al. studied 100 individuals with MetS and 50 controls and reported significantly higher cystatin-C concentrations among participants with MetS. They also reported that cystatin-C levels increased with increasing numbers of MetS components and correlated with waist circumference, fasting plasma glucose, triglycerides, and blood pressure.^[7] Their findings led the authors to suggest that cystatin-C may have utility as an early marker of renal dysfunction in MetS.

Similarly, Mao et al. reported an association between cystatin-C and MetS in patients with non-ST-segment elevation acute coronary syndrome who had preserved renal function, suggesting that cystatin-C may reflect metabolic and cardiovascular risk even when conventional renal impairment is not pronounced.^[8]

Albuminuria represents a different aspect of renal involvement. Urinary albumin reflects abnormal glomerular permeability and is an established marker used in CKD risk assessment. The urinary albumin-to-creatinine ratio corrects, to some extent, for variations in urine concentration and is therefore widely used for quantifying albuminuria.

Evidence linking MetS to albuminuria is substantial. Palaniappan et al. reported an association between microalbuminuria and MetS in participants from NHANES III.^[9] Thomas et al. subsequently showed in a systematic review and meta-analysis that MetS was associated with the development of microalbuminuria or proteinuria, as well as reduced eGFR.^[2]

More specifically, Rashidbeygi et al. conducted a meta-analysis comprising 57 studies and 10,603,067 participants. They reported that MetS was associated with increased risks of albuminuria and proteinuria and found evidence of a dose-response relationship between the number of MetS components and albuminuria risk.^[10] These findings provide strong epidemiological support for the concept that renal involvement in MetS may extend beyond changes in filtration.

Nand et al., in an Asian Indian cohort, also reported renal abnormalities and microalbuminuria among individuals with MetS.^[11] Anand and Vidya subsequently reported significant renal-function abnormalities and microalbuminuria among obese Indian individuals with MetS compared with obese individuals without MetS.^[12]

Assessing both cystatin-C and urinary albumin/UACR is therefore potentially valuable because these markers represent related but distinct aspects of renal involvement. Cystatin-C primarily reflects filtration, whereas urinary albumin reflects glomerular permeability and urinary protein leakage.

This study therefore compared serum cystatin-C, urinary albumin, and UACR between adults with and without MetS and investigated their relationship with waist circumference and waist-to-hip ratio.

MATERIALS AND METHODS

Study design and setting: This was an analytical cross-sectional comparative study conducted in the Department of Biochemistry and Department of Medicine at Index Medical College, Hospital & Research Centre.

Participants were recruited from the Medicine outpatient and inpatient services.

Study population

A total of 360 adult participants were included:

- 180 participants with metabolic syndrome.
- 180 age- and sex-matched controls.

Definition of metabolic syndrome

MetS was defined according to the International Diabetes Federation 2006 criteria.^[1]

We additionally classified participants with MetS by the number of metabolic syndrome components present.

Inclusion criteria

Participants were eligible if they were adults aged ≥ 18 years, fulfilled the relevant study criteria, were willing to participate and provided written informed consent.

Exclusion criteria

We excluded participants with conditions that could independently affect renal function. These included:

- Previously diagnosed CKD.
- Dialysis.
- Renal transplantation.
- Acute kidney injury.
- Pregnancy.
- Chronic liver disease.
- Malignancy.
- Serious acute or chronic infection.
- Autoimmune disease associated with renal involvement.

Clinical and anthropometric assessment

The following variables were recorded:

- Age
- Sex
- Height
- Weight
- BMI
- Waist circumference
- Hip circumference
- Waist-to-hip ratio
- Systolic blood pressure
- Diastolic blood pressure

Laboratory investigations

We collected blood and urine samples according to the study protocol.

The renal assessment included:

- Serum creatinine
- Blood urea
- BUN
- eGFR
- Serum uric acid
- Serum electrolytes
- Cystatin-C
- Urinary albumin
- Urinary creatinine
- UACR

This manuscript focuses primarily on cystatin-C, urinary albumin, and UACR.

Statistical analysis

We summarized continuous variables as mean $\pm$ SD. Comparisons between the MetS and control groups were performed using an independent-samples t-test. We used correlation analysis to assess relationships between anthropometric parameters and renal markers. A p-value <0.05 was considered statistically significant. Because this was a cross-sectional study, associations were interpreted without implying causality or chronicity.

Ethical considerations

The study followed institutional ethical requirements. Written informed consent was obtained from participants

before enrolment.

RESULTS

Comparison of cystatin-C and urinary renal injury markers
Serum cystatin-C was significantly elevated among participants with MetS compared with controls.

The mean cystatin-C concentration was 1.08 ± 0.22 mg/L in the MetS group compared with 0.92 ± 0.16 mg/L in controls.

Urinary albumin concentration was also significantly higher in participants with MetS.

Similarly, UACR was substantially higher in the MetS group than in controls.

Table 1: Comparison of cystatin-C, urinary albumin and UACR between study groups

Parameter	MetS (n=180) Mean $\pm$ SD	Control (n=180) Mean $\pm$ SD	t-value	p-value
Cystatin-C (mg/L)	1.08 ± 0.22	0.92 ± 0.16	8.29	<0.001
Urinary albumin (mg/L)	24.53 ± 16.86	13.04 ± 8.38	8.19	<0.001
UACR (mg/g)	38.45 ± 25.47	18.72 ± 10.58	9.60	<0.001

UACR, urinary albumin-to-creatinine ratio.

The thesis dataset demonstrates significantly higher cystatin-C, urinary albumin and UACR in participants with MetS.

Serum electrolyte and cystatin-C profile

Although this manuscript primarily focused on cystatin-C

and urinary markers, we also evaluated serum electrolytes.

Sodium and chloride did not differ significantly between the groups. Potassium was significantly higher and bicarbonate significantly lower among participants with MetS.

Table 2: Comparison of serum electrolytes and cystatin-C between study groups

Parameter	MetS (n=180) Mean $\pm$ SD	Control (n=180) Mean $\pm$ SD	t-value	p-value
Sodium (mmol/L)	139.31 ± 2.91	139.22 ± 2.90	0.29	0.772
Potassium (mmol/L)	4.32 ± 0.44	4.16 ± 0.37	3.66	<0.001
Chloride (mmol/L)	101.75 ± 2.93	102.02 ± 2.94	-0.88	0.380
Bicarbonate (mmol/L)	24.06 ± 3.06	25.08 ± 2.89	-3.27	0.001
Cystatin-C (mg/L)	1.08 ± 0.22	0.92 ± 0.16	8.29	<0.001

The electrolyte findings are secondary to the principal objectives of this manuscript. The major finding remains the significantly higher cystatin-C concentration in the MetS group.

Correlation between anthropometric parameters and renal markers

We examined the relationship between waist circumference,

waist-to-hip ratio, and renal parameters among study participants.

Most associations were weak and statistically non-significant.

WHR showed a weak inverse correlation with cystatin-C ($r=-0.193$, $p=0.009$).

Table 3: Correlation of waist circumference and waist-to-hip ratio with renal parameters

Renal parameter	Waist circumference r	p-value	Waist-to-hip ratio r	p-value
BUN	-0.028	0.707	-0.028	0.707
Blood urea	-0.028	0.710	-0.028	0.707
Serum creatinine	-0.112	0.135	-0.137	0.067
eGFR	0.103	0.170	0.121	0.107
Serum uric acid	0.029	0.699	0.060	0.427
Cystatin-C	-0.094	0.210	-0.193	0.009
Urinary albumin	0.033	0.656	0.071	0.342
UACR	-0.066	0.377	-0.044	0.559

The inverse association between WHR and cystatin-C was statistically significant but weak. Given its unexpected direction and modest correlation coefficient, interpret it cautiously rather than as evidence of a protective relationship.

Metabolic syndrome component burden

Among the 180 participants with MetS:

- 37 (20.6%) had two components.
- 70 (38.9%) had three components.
- 73 (40.6%) had four components.

Table 4: Distribution of MetS participants according to number of components

Number of MetS components	n	Percentage
2 components	37	20.6
3 components	70	38.9
4 components	73	40.6
Total	180	100.0

DISCUSSION

The present study demonstrated significantly higher serum cystatin-C, urinary albumin and UACR among adults with MetS compared with age- and sex-matched controls. These findings are important because they suggest that renal involvement in MetS may encompass both altered filtration-related biomarkers and increased urinary albumin excretion. The study's principal finding was elevated cystatin-C in the MetS group. Mean serum cystatin-C was 1.08 ± 0.22 mg/L in participants with MetS compared with 0.92 ± 0.16 mg/L in controls, representing a statistically significant difference.

Cystatin-C and metabolic syndrome

Cystatin-C is a low-molecular-weight cysteine protease inhibitor produced by nucleated cells and is freely filtered through the glomerulus. Its clinical use has expanded because it provides additional information about GFR, particularly when creatinine-based estimation may be less accurate.

This finding is strongly consistent with the study by Kadam, Bavikar, Shendye, and Aghade.^[7] In their cross-sectional study of 100 MetS cases and 50 controls, serum cystatin-C was significantly increased among individuals with MetS. They also reported that cystatin-C concentrations increased with increasing numbers of MetS components and were positively correlated with waist circumference, fasting plasma glucose, triglycerides, systolic blood pressure, and diastolic blood pressure.

The present study therefore supports the observation that cystatin-C may be associated with the metabolic syndrome phenotype.

However, the two studies differ in an important way. Kadam et al. reported a progressive increase in cystatin-C with increasing MetS components. In contrast, the present study did not demonstrate a statistically significant difference in renal parameters across the 2-, 3- and 4-component groups. This discrepancy may reflect differences in sample size, population characteristics, statistical power and study design. Mao et al.^[8] also reported an association between cystatin-C and MetS in patients with preserved renal function. Their findings support the possibility that cystatin-C may capture renal or metabolic risk that is not necessarily apparent from creatinine alone.

The present results should therefore be considered supportive of cystatin-C as a complementary renal biomarker rather than evidence that cystatin-C is specific for MetS-associated kidney disease.

Cystatin-C compared with conventional renal markers

An important rationale for evaluating cystatin-C is that several non-renal factors influence creatinine.

The present study demonstrated higher cystatin-C in the MetS group, while the companion analysis also demonstrated higher serum creatinine and lower eGFR in these participants. The concordance between these findings

strengthens the overall evidence of an altered renal profile.

The study by Kadam et al. is particularly interesting in this context because they reported increased cystatin-C despite no significant differences in serum creatinine, urea or eGFR between their MetS and control groups.^[7] Their findings suggest that cystatin-C may sometimes demonstrate differences before conventional markers become clearly abnormal.

This study differs because both cystatin-C and conventional renal parameters showed significant between-group differences.

Current KDIGO 2024 guidance recommends serum creatinine-based eGFR as the initial assessment of GFR and recommends use of combined creatinine- and cystatin-C-based eGFR when cystatin-C is available and greater accuracy is needed.^[6] KDIGO also emphasizes that an isolated abnormal eGFR or ACR should not automatically be interpreted as evidence of chronic kidney disease without confirmation of chronicity.

Thus, the elevated cystatin-C observed in the present study should be regarded as evidence of an altered renal biomarker profile rather than definitive proof of CKD.

Urinary albumin and metabolic syndrome

The second major finding was a significant increase in urinary albumin concentration among participants with MetS.

Urinary albumin was approximately higher in the MetS group than in controls, with mean values of 24.53 ± 16.86 mg/L and 13.04 ± 8.38 mg/L, respectively.

Albuminuria represents an important marker of glomerular permeability and is increasingly recognized as an indicator of kidney and cardiovascular risk.

This finding is consistent with the systematic review and meta-analysis by Thomas et al.^[2] which demonstrated an association between MetS and the development of microalbuminuria/proteinuria, as well as reduced eGFR.

The large meta-analysis by Rashidbeygi et al.^[10] provides particularly strong supporting evidence. Their analysis included 57 studies involving more than 10.6 million participants. They reported increased risks of albuminuria and proteinuria among individuals with MetS. They also found that obesity, impaired fasting glucose, elevated blood pressure and hypertriglyceridaemia were associated with increased risk of albuminuria or proteinuria.

These findings provide biological and epidemiological context for the increased urinary albumin observed in the present study.

UACR and early renal involvement

UACR was significantly higher among participants with MetS than controls, with mean values of 38.45 ± 25.47 mg/g and 18.72 ± 10.58 mg/g, respectively.

UACR is clinically useful because it relates urinary albumin concentration to urinary creatinine concentration, reducing the influence of urine dilution.

Palaniappan et al.^[9] using NHANES III data, reported an association between microalbuminuria and MetS. Their findings contributed to the recognition that renal involvement in MetS

may occur even before advanced renal dysfunction becomes clinically evident.

Anand and Vidya,^[12] also reported microalbuminuria among obese individuals with MetS and found that renal dysfunction was more common among MetS cases than controls. Their Indian study is especially relevant to the present investigation because both studies were conducted in hospital-based Indian populations.

The present findings are therefore consistent with the broader literature indicating that albuminuria may represent an important renal manifestation of metabolic syndrome.

Cystatin-C and urinary albumin represent complementary processes

One important conceptual implication of this study is that cystatin-C and urinary albumin should not be regarded as interchangeable markers.

Cystatin-C primarily provides information related to filtration, whereas urinary albumin provides information about glomerular permeability and albumin leakage.

The simultaneous elevation of both markers in the MetS group suggests that renal involvement may involve more than a simple reduction in filtration.

The combination of:

- elevated cystatin-C,
- elevated urinary albumin,
- elevated UACR,

along with the lower eGFR and higher creatinine observed in the overall study, provides a multidimensional picture of renal involvement.

This interpretation is consistent with the modern CKD framework, in which both GFR and albuminuria contribute to kidney disease assessment and risk stratification.^[6]

Importantly, however, a single cross-sectional measurement of elevated albuminuria or cystatin-C cannot establish chronic kidney disease. KDIGO recommends confirmation of abnormalities and assessment of chronicity over time.^[6]

Relationship between central adiposity and cystatin-C

The study examined the relationship between waist circumference and WHR and renal parameters.

Most correlations were not statistically significant.

There was no significant association between waist circumference and cystatin-C ($r=-0.094$, $p=0.210$). Similarly, waist circumference was not significantly associated with urinary albumin or UACR.

A weak inverse association was observed between WHR and cystatin-C ($r=-0.193$, $p=0.009$).

The latter finding requires cautious interpretation.

The correlation coefficient was small, and the direction of association was unexpected given the established relationship between central adiposity and metabolic risk. Moreover, the study design does not permit determination of whether the association is biologically meaningful.

This differs from the findings of Kadam et al,^[7] who reported a positive correlation between cystatin-C and waist circumference. It also differs from the findings of studies examining cystatin-C in relation to obesity and metabolic risk factors.

The discrepancy may reflect differences in population characteristics, sample composition or unmeasured

confounding.

Therefore, the present WHR-cystatin-C association should be reported as a weak statistical association, rather than interpreted as evidence of a protective effect of greater WHR.

Urinary albumin, UACR and the number of MetS components

An important finding was the absence of statistically significant differences in urinary albumin and UACR across participants with two, three or four MetS components.

This result contrasts somewhat with the large meta-analysis by Rashidbeygi et al,^[10] which reported an escalating dose-response association between the number of MetS components and albuminuria risk.

There are several possible explanations for this discrepancy.

First, the present study was cross-sectional, whereas the meta-analysis included multiple study designs and populations. Second, the present subgroup sizes were relatively modest, particularly the two-component group. Third, the study population was recruited from a tertiary care hospital, which may differ substantially from community-based populations. Finally, biological heterogeneity among individuals with the same number of MetS components may be considerable.

Thus, the absence of a significant gradient in this dataset should not be interpreted as evidence that increasing metabolic burden has no renal consequences.

Rather, the appropriate interpretation is that the present study demonstrated significantly higher urinary albumin and UACR in participants with MetS overall, but did not demonstrate a statistically significant difference according to the number of MetS components.

Comparison with Indian evidence

The findings of the present study are particularly relevant in the context of Indian research.

Nand et al,^[11] studied 300 individuals in an Asian Indian cohort and investigated renal function and microalbuminuria in relation to MetS. Their work reported an association between MetS and renal impairment.

Anand and Vidya,^[12] examined 100 obese individuals, including 50 with MetS and 50 without MetS, in a university hospital setting. They reported significant renal-function derangements among participants with MetS, including reduced eGFR and microalbuminuria. Their study also reported associations between altered renal function and fasting glucose, blood pressure and triglyceride concentrations.

The present study extends this literature by demonstrating significant differences in cystatin-C, urinary albumin and UACR in a larger cohort of 360 participants.

The larger sample and simultaneous assessment of a filtration-related marker and urinary injury markers provide a broader characterization of renal involvement.

Potential mechanisms linking MetS with albuminuria

Several mechanisms may explain the association between MetS and increased urinary albumin.

Central adiposity is associated with increased inflammatory activity, insulin resistance and altered adipokine signalling. Hypertension may increase intraglomerular pressure, while dysglycaemia may contribute to endothelial dysfunction and alterations in glomerular permeability.

Dyslipidaemia may additionally contribute to endothelial injury and vascular dysfunction.

These mechanisms may act simultaneously, resulting in increased albumin leakage even before substantial reductions in filtration become evident.

The meta-analysis by Rashidbeygi et al.^[10] provides epidemiological support for this interpretation because several individual components of MetS—including obesity, impaired fasting glucose, elevated blood pressure and hypertriglyceridaemia—were independently associated with increased albuminuria risk.

Thus, albuminuria may represent one manifestation of the cumulative vascular and metabolic consequences of MetS.

Clinical implications

The findings have potential implications for clinical assessment.

Traditional renal assessment often emphasizes serum creatinine and eGFR. However, the present findings indicate that individuals with MetS may also demonstrate significant changes in cystatin-C and urinary albumin/UACR.

The KDIGO 2024 guideline recommends assessment of both GFR and urine albumin in people at risk for CKD. It also recommends combined creatinine- and cystatin-C-based estimation of GFR where cystatin-C is available and greater accuracy is clinically important.^[6]

Consequently, combined evaluation may be particularly useful in individuals with MetS who have multiple renal risk factors but do not yet demonstrate overt CKD.

Nevertheless, elevated urinary albumin or cystatin-C should not be interpreted as definitive evidence of chronic kidney disease based on a single measurement. Repeat testing and assessment of chronicity remain important.^[6]

Strengths of the Study

The major strengths of the present study include:

- Inclusion of 360 participants.
- Equal representation of MetS and control groups.
- Age- and sex-matched controls.
- Use of standardized IDF criteria for MetS.
- Assessment of cystatin-C in addition to conventional renal parameters.
- Direct evaluation of urinary albumin and UACR.
- Assessment of anthropometric–renal relationships.
- Analysis of renal markers according to MetS component burden.

CONCLUSION

The present study demonstrated significantly higher serum cystatin-C, urinary albumin and UACR among adults with metabolic syndrome compared with age- and sex-matched controls.

The elevation of cystatin-C suggests an alteration in filtration-related renal biomarkers, while the increase in urinary albumin and UACR indicates greater urinary albumin excretion and may reflect altered glomerular permeability.

Taken together with the conventional renal findings from the overall study, these results suggest that renal involvement associated with MetS may encompass both filtration-related changes and urinary albumin abnormalities.

The findings are consistent with previous reports by Kadam et al., Nand et al., Anand and Vidya, Thomas et al., and

Rashidbeygi et al., which have linked MetS with renal dysfunction and albuminuria.^[2,7,10-12]

However, the present study did not demonstrate a statistically significant relationship between renal parameters and increasing MetS component count, nor did it demonstrate significant correlations between waist circumference and most renal markers.

Cystatin-C and urinary albumin/UACR may therefore be useful complementary markers in the renal evaluation of individuals with metabolic syndrome, but longitudinal studies with repeated measurements are required to establish whether these abnormalities predict subsequent CKD development or progression.

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Conflicts of interest

There are no conflicts of interest.

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