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Correlation of Midday and Morning Urine Protein-Creatinine Ratio with 2 4-Hour Urine Protein

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ABSTRACT

Background: Proteinuria is a key marker for diagnosing and monitoring chronic kidney disease (CKD) and is associated with disease progression and cardiovascular risk. Spot urine protein-to-creatinine (P/C) ratio offers a simpler alternative to 24-hour urine collection, but its accuracy remains debated. **Objective:** To evaluate the correlation between morning and midday spot P/C ratios and 24-hour urinary protein excretion and assess their diagnostic reliability.

Patients and Methods: In this cross-sectional study, 200 participants were scheduled for 24-hour urine protein estimation provided morning and midday spot urine samples for P/C ratio measurement. Demographic, anthropometric, biochemical, lipid profile, and urinalysis data were collected. Pearson correlation tested agreement between methods, and associations with lipid parameters were analyzed. **Results:** Mean ± standard deviation (SD) 24-hour urinary protein was 1.65 ± 1.38 mg, while morning and midday P/C ratios were 1.83 ± 2.2 mg/g and 1.72 ± 2.08 mg/g, respectively. Both morning ($r = 0.783$, $P < 0.0001$) and midday ($r = 0.780$, $P < 0.001$) P/C ratios showed strong positive correlations with 24-hour proteinuria, with closer agreement ($P^2 = 0.9220$) was according to multiple regression analysis. Significant positive correlations were, also, observed between 24-hour proteinuria and total cholesterol ($P = 0.002$), triglycerides ($P = 0.006$), and LDL cholesterol ($P < 0.0001$); similar correlations were found for morning and midday P/C ratios.

Conclusion: Morning and midday spot P/C ratios were strongly correlated with 24-hour urinary protein excretion. Midday sampling showed marginally superior agreement and, given its convenience, represents a practical and reliable alternative to 24-hour urine collection for assessing proteinuria and improving patient compliance without loss of diagnostic accuracy.

Keywords: Midday, Morning Urine Protein-Creatinine Ratio, 24-Hour Urine Protein.

INTRODUCTION

Chronic kidney disease (CKD) represents a long-standing clinical condition in which the kidneys undergo a steady decline in their ability to maintain normal function and preserve structural integrity. Rather than occurring abruptly, this deterioration evolves gradually, reflecting ongoing injury to renal tissue that compromises filtration, metabolic balance, and overall homeostasis with time. Globally, its prevalence is estimated to be between 11% and 13%, with severity determined primarily by the glomerular filtration rate (GFR) and the degree of proteinuria or albuminuria. In older adults, CKD is often multifactorial, as cumulative episodes of kidney injury and comorbidities contribute to its development [(1)].

Proteinuria remains one of the most sensitive markers of renal injury. Rapid qualitative and semi-quantitative assessment in random urine samples has improved the early detection and monitoring of both primary kidney disorders and systemic diseases affecting the kidneys [(2)]. Despite these advances, CKD-related mortality has been rising steadily, driven in large part by reduced GFR and higher proteinuria levels [(3)].

Proteinuria should be viewed not merely as a passive indicator of kidney damage but as a pathogenic driver that accelerates renal decline. The abnormal passage of proteins across the glomerular barrier exposes proximal tubular epithelial cells to a sustained toxic load. This exposure initiates cellular stress responses, activates inflammatory cascades, and promotes tubular cell loss with subsequent interstitial scarring. Over time, these maladaptive processes converge to advance chronic injury, culminating in progressive loss of renal function and, in many patients, transition to end-stage kidney disease [(4)]. The underlying mechanisms may include increased circulating low-molecular-weight proteins (prerenal), glomerular filtration barrier damage (glomerular), defective tubular reabsorption (tubular), or urinary tract protein shedding (post-renal) [(5)].

Persistent proteinuria is strongly associated with adverse renal and cardiovascular outcomes, making early detection and treatment like blood pressure control and management of the underlying cause critical for improving prognosis [(6)]. In nephrotic syndrome, severe protein loss into the urine is accompanied by hypoalbuminemia, hyperlipidemia, and edema, resulting from glomerular injury caused by primary or secondary kidney diseases [(7)].

Measurement of proteinuria using the urine protein-to-creatinine (P/C) ratio in spot samples provides a convenient and cost-effective alternative to 24-hour urine collection. This method has been shown to correlate with CKD severity and progression risk [(8)]. However, 24-hour urine collection remains the gold standard for quantifying proteinuria, despite its limitations, including incomplete collection, inaccurate timing, and bacterial contamination [(2,9)].

Although the spot urine P/C ratio is widely used for proteinuria assessment, its precision compared with 24-hour urine protein measurement remains uncertain. Therefore, this study aimed to compare different methods of proteinuria measurement and determine the

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diagnostic accuracy of the spot urine P/C ratio against the standard 24-hour urine protein test.

PATIENTS AND METHODS

We performed this cross-sectional study at the Internal Medicine Department, Faculty of Medicine, Zagazig University, including both inpatients and outpatients referred for 24-hour urine protein measurement over a period of from June 2024 to June 2025

A total of 200 subjects were included, with a mean age of 49.62 ± 21.56 years and a median age of 47, ranging from 19 to 90 years. This cross-sectional study consisted of 160 males (80%) and 40 females (20%).

Ethical approval:

Approval for the study protocol was granted by the Zagazig University Institutional Review Board (ZU-IRB#26-15-Jan-2024). Prior to enrollment, written informed consent was secured from every participant. All procedures were carried out in strict accordance with the ethical principles outlined in the Declaration of Helsinki, ensuring the protection of human subjects throughout the research process. The selected patients were above 18 years of both sexes who were advised to undergo 24-hour urine protein estimation.

A detailed history was obtained from each participant, including demographic data, presenting complaints, relevant comorbidities, past surgical history, and family history. Anthropometric measurements included weight (kg) and calculation of body mass index (BMI) as weight (kg) divided by height squared (m^2).

Laboratory investigations:

Hematological parameters, including total leukocytic count, hemoglobin concentration, and platelet count, were assessed using the Sysmex XN-1000 automated hematology analyzer (Sysmex Corporation, Japan). Liver function evaluation comprised measurement of serum albumin via the bromocresol green method and total protein using the Biuret method, both quantified by spectrophotometry to ensure precision and reproducibility. Renal function was assessed by determining serum creatinine, blood urea nitrogen, and uric acid levels using the Roche Cobas 6000 automated analyzer (Roche Diagnostics, Switzerland) [10]. Lipid profile assessment included measurement of serum triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), performed on the Cobas b 101 system (Roche Diagnostics, Switzerland), ensuring standardized and reliable results.

Urinalysis and proteinuria assessment:

Midstream urine samples (15–30 mL) were collected for microscopic examination of red and white blood cells. Twenty-four-hour urine protein measurement: Urine was collected over a 24-hour period, beginning after the first void in the morning and continuing until the same time the following day. Samples were stored at +4 °C during collection. Protein concentration was determined by the immunoturbidimetric method using the Cobas C111 device (Roche Diagnostics, Switzerland), following centrifugation at 1500–2000 ×g for 10 minutes to remove debris. Spot urine protein-to-creatinine ratio: Midday and early morning urine samples were analyzed for protein and creatinine content, and the ratio was calculated.

Statistical analysis

Statistical analyses were carried out with SPSS version 23.0. Qualitative variables were expressed as numbers and percentages, whereas quantitative data were summarized using mean ± SD or median with range. Group differences were assessed using one-way ANOVA, and associations between continuous measures were tested with Pearson's correlation. Logistic regression was applied to determine independent predictors. A p-value below 0.05 denoted statistical significance.

RESULTS

The study enrolled 200 subjects (mean age 49.6 ± 21.6 years; range 19–90), comprising 160 males (80%) and 40 females (20%). This study demonstrated a notable prevalence of chronic conditions, particularly no history of medical diseases (40%) and HTN (30%), mean body weight was 73.95 ± 8.42 kg, and mean BMI was 25.52 ± 3.36 kg/m^2 (Table **1**).

Table (1): Demographic, Medical History, and Anthropometric Data of the Included Subjects (N = 200)

Parameter	Statistic	Result
Age (Years)	Mean ± SD	49.62 ± 21.56
	Median (Range)	47 (19 - 90)
Sex	Male	160 (80%)
	Female	40 (20%)
Medical History	HCV	20 (10%)
	DM	20 (10%)
	HTN	60 (30%)
	no history of medical diseases	80 (40%)
	CKD	20 (10%)
	BPH	20 (10%)
	HTN and CKD	20 (10%)
Body Weight (Kg)	Mean ± SD	73.95 ± 8.42
	Median (Range)	74 (56 - 87)
BMI (Kg/m^2)	Mean ± SD	25.52 ± 3.36
	Median (Range)	25 (21 - 33)

The mean total leukocyte count was 7.43 × 10^3/µL, mean hemoglobin was 10.11 g/dL, and mean serum albumin was 2.9 g/dL. Mean serum creatinine was 1.54 mg/dL and BUN was 42.46 mg/dL. The mean total cholesterol was markedly elevated at 289.7 mg/dL. Triglycerides and HDL were within acceptable ranges (132.6 mg/dL and 43.3 mg/dL, respectively), while LDL showed wide variability with a mean of 113.7 mg/dL. Mean urine pus cells were 5.79/high power field, and RBCs were 2.68/high power field, (Table **2**).

Table (2): Hematological, Kidney Function, Lipid Profile, and Urine Analysis Data of the Included Subjects (N = 200)

Parameter	Statistic	Result
CBC Data	TLC (×10³) Mean ± SD	7.43 ± 1.38
	TLC (×10³) Median (Range)	7.55 (3.6 - 11.2)
	Hb (g) Mean ± SD	10.11 ± 2.17
	Hb (g) Median (Range)	8.85 (7.8 - 15.5)
	PLT (×10³) Mean ± SD	224.79 ± 9.82
	PLT (×10³) Median (Range)	224 (111 - 405)
Kidney Function Tests	S. Albumin (g) Mean ± SD	2.9 ± 0.72
	S. Albumin (g) Median (Range)	3.05 (1.6 - 4)
	Total Protein (g) Mean ± SD	6.03 ± 1.26
	Total Protein (g) Median (Range)	6.3 (3.6 - 7.9)
	S. Creat. (mg/dl) Mean ± SD	1.54 ± 0.76
	S. Creat. (mg/dl) Median (Range)	1.1 (0.3 - 8)
	BUN (mg/dl) Mean ± SD	42.46 ± 8.6
	BUN (mg/dl) Median (Range)	36.05 (11.4 - 103)
Lipid Profile	Total Cholesterol (mg/dl) Mean ± SD	289.69 ± 52.91
	Total Cholesterol (mg/dl) Median (Range)	227 (145 - 680)
	TG (mg/dl) Mean ± SD	132.64 ± 5.03
	TG (mg/dl) Median (Range)	122.5 (79 - 234)
	LDL (mg/dl) Mean ± SD	113.72 ± 27.91
	LDL (mg/dl) Median (Range)	65.5 (16 - 480)
	HDL (mg/dl) Mean ± SD	43.34 ± 7.9
	HDL (mg/dl) Median (Range)	39 (23 - 93)
Urine Analysis	Urine Pus Mean ± SD	5.79 ± 1.35
	Urine Pus Median (Range)	5 (0 - 15)
	Urine RBCs Mean ± SD	2.68 ± 0.2
	Urine RBCs Median (Range)	2 (0 - 9)

The mean 24-hour urinary protein was elevated at 1650.2 mg, and both morning and midday (P/C) ratio values were markedly raised, indicating significant proteinuria. No statistically significant variations were detected between the 24-hour protein values and the protein-to-creatinine ratio (p = 0.6572). Similarly, post hoc pairwise testing demonstrated no significant variations, with all p-values greater than 0.05 (Table **3**).

Table (3): 24hr Urine Protein and Urine Protein-Creatinine Ratio data among included subjects (N = 200)

		Value (N = 200)
24hr protein in urine (mg)	Mean ± SD	1650.2 ± 377.9
	Median (Range)	1300 (100-4400)
PCR		
Morning (mg/g)	Mean ± SD	1825.24 ± 197.03
	Median (Range)	887.5 (60-12000)
Midday (mg/g)	Mean ± SD	1723.84 ± 275.62
	Median (Range)	833.34 (53.33-11400)
P. Value		0.6572
Post Hoc analysis		P1 = 0.6325, P2 = 0.9220, P3 = 0.8573

P1 = 24 hr urine protein vs morning PCR, P2 = 24 hr urine protein vs midday PCR, P3 = morning PCR vs midday PCR.

There were significant positive correlations between 24 hr urine protein and morning (P/C) ratio (r = 0.783, P < 0.0001) and between 24 hr urine protein and midday (P/C) ratio (r = 0.78, P < 0.001) (Table **4**).

Table (4): Correlation between 24hr Urine Protein and Urine Protein-Creatinine Ratio (N = 200)

Variables	**24hr Urine Protein**	
	r	**P. Value**
Morning Urine Protein-Creatinine Ratio (mg/g)	0.783	<0.0001
Midday Urine Protein - Creatinine Ratio (mg/g)	0.78	<0.0001

Significant positive correlations were observed between 24-hour urinary protein with total cholesterol (r = 0.214, p = 0.002), triglycerides (r = 0.193, p = 0.006), and LDL cholesterol (r = 0.433, p < 0.0001). Morning urinary protein also showed significant positive correlations with total cholesterol (r = 0.187, p = 0.008), triglycerides (r = 0.165, p = 0.019), and HDL cholesterol (r = 0.436, p < 0.0001). Similarly, midday urinary protein correlated positively with total cholesterol (r = 0.187, p = 0.008), triglycerides (r = 0.163, p = 0.021), and LDL cholesterol (r = 0.434, p < 0.0001) (Table **5**).

Table (5): Correlation between protein in Urine and lipid profile (N = 200)

	24hr Urine Protein		Morning protein		Midday protein	
	r	P. Value	r	P. Value	r	P. Value
Total cholesterol	0.214	0.002*	0.187	0.008*	0.187	0.008*
TG	0.193	0.006*	0.165	0.019*	0.163	0.021*
LDL	0.127	0.074	0.104	0.143	0.105	0.138
HDL	0.433	<0.0001*	0.436	<0.0001*	0.434	<0.0001*

Triglycerides, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein.

DISCUSSION

In clinical practice, accurate 24-hour urine collection is often difficult to achieve due to patient nonadherence, incomplete collection, and associated costs. As an alternative, the spot urine protein-to-creatinine ratio urinary (P/C) ratio is frequently used as a surrogate measure. To improve accuracy, urine specific gravity should be considered, as extreme dilution or concentration can respectively underestimate or overestimate urinary P/C ratio values, a point emphasized by **Karrar *et al.*** [11].

When measured using automated biochemical analyzers, the U(P/C) ratio has shown potential as a primary screening tool for proteinuria, offering good accuracy, sensitivity, and reproducibility while accommodating variations in hydration status. Furthermore, this method facilitates simplified point-of-care testing, as highlighted by **Alao *et al.*** [12].

In the present study, we included 200 participants with a mean age of 49.6 ± 21.6 years (median 47, range 19–90). Males constituted the majority of the study (80%), with females representing 20%. This demographic profile is clinically significant, as age and sex are known to influence renal physiology, glomerular filtration, and urinary protein excretion. Factors such as hormonal variations, muscle mass differences, and underlying comorbidities can alter proteinuria patterns, a consideration also noted by **Alao *et al.*** [12].

Our findings align with those of **Raza *et al.*** [13] who evaluated 157 patients with a mean age of 30.45 ± 12.11 years, of whom 59.8% were male and 40.2% were female. Although their study population was younger on average, both studies shared the observation that males predominated in proteinuria assessment.

Regarding comorbidities, the most prevalent condition in our study was hypertension (30%), followed by diabetes mellitus (10%), chronic kidney disease (10%), benign prostatic hyperplasia (10%), and hepatitis C virus infection (10%). Notably, 40% of participants reported no prior medical conditions. This distribution reflects the multifactorial nature of proteinuria, where systemic diseases such as hypertension and diabetes are well-established contributors to renal injury, while CKD represents a direct manifestation of progressive nephron loss. BPH may contribute to lower urinary tract symptoms and urinary retention, potentially affecting urinalysis, whereas hepatitis C is recognized for its extrahepatic manifestations, including glomerular disease. These patterns are consistent with the epidemiological observations reported by **MacRae *et al.*** [14].

Our findings are supported by **Gutiérrez-Peredo *et al.*** [15] who observed that hypertension was the most common comorbidity in their study, affecting 51.3% of participants. Diabetes mellitus was present in 14.5%, while heart failure and cerebrovascular disease were less frequent, occurring in 8.6% and 0.7% of individuals, respectively. Peripheral vascular disease was reported in 3.3%, and chronic liver disease in 7.9%. These results, similar to our own, emphasize the diversity of systemic conditions influencing kidney health. In the present study, the mean body weight was 75.2 ± 10.6 kg (median 74, range 56–87), with a mean BMI of 25.5 ± 3.4 kg/m² (median 25, range 21–33). Most participants fell within the overweight range, which is a recognized risk factor for hypertension, diabetes, and CKD. This observation aligns with the metabolic and cardiovascular risk patterns reported by **Visram *et al.*** [16].

Comparable to our results, **Raza *et al.*** [13] found that their study population had a mean BMI of 22.3 ± 5.65 kg/m², placing most participants in the normal weight category. While their study tended to be leaner, both studies underline the importance of considering body composition in the assessment of proteinuria risk and renal outcomes.

In our study, the mean total leukocyte count (TLC) was 7.43 ± 2.38 × 10^3, hemoglobin averaged 10.11 ± 2.17 g/dL, and platelet count (PLT) was 224.8 ± 79.8 × 10^3. Mean serum cholesterol was elevated at 289.7 ± 152.9 mg/dL, with notable variability. These hematological parameters indicate that mild anemia was common, consistent with the pathophysiology of CKD and other chronic illnesses. This trend coincides with the observations of **Teimoury *et al.*** [17] who highlighted anemia as a frequent finding in patients with renal impairment. Similarly, **Gutiérrez-Peredo *et al.*** [15] reported a mean hemoglobin level of 10.5 ± 3 g/dL, also suggestive of mild anemia. They attributed this to factors such as chronic disease burden, nutritional deficiencies, and impaired erythropoiesis, mechanisms likely relevant to our study population as well.

In our study, the mean serum albumin was 2.9 ± 0.72 g/dL (median 3.05, range 1.6–4), and mean total protein was 6.03 ± 1.26 g/dL (median 6.3, range 3.6–7.9). Mean serum creatinine was 1.54 ± 1.76 mg/dL (median 1.1, range 0.6–8), while the mean blood urea nitrogen (BUN) was 42.46 ± 28.6 mg/dL (median 36.05, range 11.4–103) [10]. These findings reflect a proportion of patients with hypoalbuminemia, which may be attributed to urinary protein loss, liver disease, or malnutrition. The variability in serum creatinine and BUN levels indicates a spectrum from preserved renal function to significant impairment. Elevated creatinine levels suggest reduced glomerular filtration, whereas

high BUN values may reflect impaired kidney function, dehydration, or high protein intake. These patterns are consistent with the pathophysiological considerations discussed by **Rovin *et al.*** [18].

Our results agree with **Gutiérrez-Peredo *et al.*** [15] who reported a mean serum albumin of 3.1 ± 1.1 g/dL in their cohort, reflecting variable protein status influenced by nutritional and disease factors. They observed median creatinine levels of 1.2 mg/dL (IQR 0.8–2.2) and median BUN values of 26.0 mg/dL (IQR 15.4–43.4), also indicating a wide range of renal function among participants.

In our population, mean triglyceride levels were 132.64 ± 45.03 mg/dL (median 122.5, range 79–234), and total cholesterol averaged 289.69 ± 152.91 mg/dL (median 227, range 145–680). Mean LDL cholesterol was 113.72 ± 127.91 mg/dL (median 65.5, range 16–480), and mean HDL cholesterol was 43.34 ± 17.9 mg/dL (median 39, range 23–93). These results show that triglycerides were generally within the normal to mildly elevated range, LDL values were highly variable, and HDL levels ranged from suboptimal to protective. Low HDL cholesterol, in particular, is a recognized risk factor for atherosclerotic disease, especially in patients with CKD, who already face heightened cardiovascular risk.

Zhang *et al.* [19] have similarly emphasized the prognostic importance of lipid abnormalities in kidney disease, particularly the contribution of dyslipidemia to accelerated atherosclerosis. Furthermore, our findings parallel those of **Gutiérrez-Peredo *et al.*** [15] who found mean total cholesterol levels of 199.7 ± 55.3 mg/dL in their cohort, reflecting interindividual differences in lipid metabolism and associated cardiovascular risk profiles.

In our study, the mean urine pus cell count was 5.79 ± 4.35 (median 5, range 0–15), while the mean urine red blood cell (RBC) count was 2.68 ± 2.2 (median 2, range 0–9). These values suggest leukocyturia within normal to mildly elevated limits, indicative of minimal urinary tract inflammation in most participants. Hematuria was generally minimal, with only a few cases showing higher counts that could warrant further evaluation for urinary tract infection, nephrolithiasis, or glomerular pathology. Similar patterns were described by **Akin *et al.*** [20] who reported that most patients exhibited normal to mildly elevated urine pus and RBC counts.

Our observations align with **Medina-Rosas *et al.*** [21] who also found that urine microscopy results in their cohort generally fell within the normal range, with minimal inflammatory or hematuric findings.

For urinary protein excretion, the 24-hour measurements averaged 1.65 ± 1.38 mg, with comparable values recorded in morning (1.51 ± 1.22 mg) and midday (1.43 ± 1.16 mg) samples. Similarly, the protein-to-creatinine ratio (P/C) ratio showed close agreement between 24-hour (2.06 ± 2.52 mg/g), morning (1.83 ± 2.2 mg/g), and midday (1.72 ± 2.08 mg/g) measurements, with midday (P/C) ratio demonstrating the strongest correlation to the 24-hour standard ($P^2 = 0.9220$). This supports the use of midday spot samples as a practical and statistically robust alternative to 24-hour urine collections, a conclusion also emphasized by **Wahbeh *et al.*** [22].

Mendelson *et al.* [23] similarly reported high concordance between U(P/C) ratio and 24-hour urine collections, particularly in patients with proteinuria <500 mg/day. Montero *et al.* (24) also demonstrated strong agreement between morning and midday (P/C) ratio with 24-hour values in nephrology patients, with midday (P/C) ratio showing slightly better alignment—findings that closely match our own.

Within our study population, strong positive correlations were identified between 24-hour urinary protein and the protein-to-creatinine ratio measured in the morning ($r = 0.783$, $p < 0.0001$) and at midday ($r = 0.78$, $p < 0.001$). These findings are consistent with the observations of **Raza *et al.*** [13] who demonstrated a significant association between spot U(P/C) ratios and 24-hour protein excretion. **Nischintha *et al.*** [27] further confirmed a positive Pearson correlation between the two methods, reinforcing the validity of spot testing in clinical practice.

Additionally, our study found significant positive correlations between proteinuria (whether measured over 24 hours, in the morning, or at midday) and lipid parameters, including total cholesterol, triglycerides, and LDL. These associations reflect the established link between proteinuria and dyslipidemia in kidney disease, where altered lipid metabolism may contribute to both renal and cardiovascular pathology. Similar relationships were reported by **Hu *et al.*** [28] in pediatric patients with nephrotic syndrome, where proteinuria correlated significantly with lipid parameters across different urine collection times.

This study had some limitations, first, although the sample size was adequate, it may not fully reflect variations in patients with advanced renal disease or differing demographic characteristics. Second, being a single-center study may limit the generalizability of the results. Third, while spot urine samples offer convenience over 24-hour collections, factors such as hydration status, dietary intake, and circadian variation in protein excretion could have influenced measurements. Finally, we did not evaluate the effects of comorbidities, medication use, or disease progression on proteinuria patterns, which warrants further investigation.

CONCLUSION

This study found a strong positive correlation between morning and midday urine protein-to-creatinine ratios (P/C) ratio and 24-hour urinary protein excretion, supporting the use of spot urine testing as a reliable alternative for proteinuria assessment. Given the practical challenges and potential errors of 24-hour urine collection, spot P/C ratio particularly midday sampling offers a convenient, accurate, and patient-friendly method for routine clinical evaluation.

Adoption of this approach could enhance patient compliance and efficiency in renal function monitoring without compromising diagnostic accuracy.

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REFERENCES

1. **Hill N, Fatoba S, Oke J *et al.* (2016):** Global prevalence of chronic kidney disease – a systematic review and meta-analysis. PLoS One, 11(7): e0158765. doi: 10.1371/journal.pone.0158765.
2. **Amin S, Illipilla S, Hebbar S *et al.* (2014):** Quantifying proteinuria in hypertensive disorders of pregnancy. International Journal of Hypertension, 14: 941408. doi: 10.1155/2014/941408.
3. **Nitsch D, Grams M, Sang Y *et al.* (2013):** Associations of estimated glomerular filtration rate and albuminuria with mortality and renal failure by sex: A meta-analysis. BMJ., 346: f324. doi: 10.1136/bmj.f324.
4. **Liu D, Lv L (2019):** New understanding on the role of proteinuria in progression of chronic kidney disease. Adv Exp Med Biol., 1165:487-500.
5. **Makhammajanov Z, Dauyey Z, Markhametova Z *et al.*** (**2022**): Urinary protein profiling for potential biomarkers of chronic kidney disease: A pilot study. Diagnostics, 12(11):2583. doi: 10.3390/diagnostics12112583.
6. **Vallon V, Thomson S** (**2020**): The tubular hypothesis of nephron filtration and diabetic kidney disease. Nature Reviews Nephrology, 16(6): 317–336.
7. **Leslie S, Rathi B, Sharif S *et al.* (2024):** 24-Hour Urine Collection and Analysis. Treasure Island (FL): StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK482482/
8. **Makhammajanov Z, Gaipov A, Myngbay A *et al.* (2024):** Tubular toxicity of proteinuria and the progression of chronic kidney disease. Nephrology Dialysis Transplantation, 39(4): 589–599.
9. **Cheung H, Leung K, Choi C (2016):** Diagnostic accuracy of spot urine protein-to-creatinine ratio for proteinuria and its association with adverse pregnancy outcomes in Chinese pregnant patients with pre-eclampsia. Hong Kong Med J., 22(3): 249-55.
10. **Kabunda J, Gabela L, Kalinda C *et al.*** (**2021**): Comparing 99mTc-PSMA to 99mTc-MDP in prostate cancer staging of the skeletal system. Clinical Nuclear Medicine, 46(7): 562–568.
11. **Karrar S, Fogel J, Hong P** (**2023**): Withstanding the test of time: Morning versus afternoon/evening urine protein-to-creatinine ratios in preeclampsia. Pregnancy Hypertension, 34: 90–94.
12. **Alao M, Asinobi O, Ibrahim O *et al.*** (**2021**): Spot urine protein-to-creatinine ratio compared with dipstick proteinuria as a primary screening tool for renal disease in a community setting. Saudi Journal of Kidney Diseases and Transplantation, 32(6): 1655–1665.
13. **Raza, A, Nawaz, S. H, Rashid, R *et al.*** (**2023**): The correlation of spot urinary protein-to-creatinine ratio with 24-h urinary protein excretion in various glomerulopathies. World J Nephrol., 12(5): 159–167.
14. **MacRae C, Mercer S, Guthrie B *et al.*** (**2021**): Comorbidity in chronic kidney disease: A large cross-sectional study of prevalence in Scottish primary care. British Journal of General Practice, 71(704): 243–249.
15. **Gutiérrez-Peredo G, Montaño-Castellón I, Gutiérrez-Peredo A *et al.*** (**2024**): The urine protein/creatinine ratio as a reliable indicator of 24-h urine protein excretion across different levels of renal function and proteinuria: The TUNARI prospective study. BMC Nephrology, 25(1): 418. doi: 10.1186/s12882-024-03804-7.
16. **Visram A, Al Saleh A, Parmar H *et al.*** (**2020**): Correlation between urine ACR and 24-h proteinuria in a real-world cohort of systemic AL amyloidosis patients. Blood Cancer Journal, 10(12): 124. doi: 10.1038/s41408-020-00391-2.
17. **Teimoury A, Iraj B, Heidari-Beni M *et al.*** (**2014**): Why 24-h urine albumin excretion rate method still is used for screening of diabetic nephropathy in Isfahan laboratories? International Journal of Preventive Medicine, 5(3): 341-47.
18. **Rovin B, Adler S, Barratt J *et al.*** (**2021**): KDIGO 2021 clinical practice guideline for the management of glomerular diseases. Kidney International, 100(4): 1. doi: 10.1016/j.kint.2021.05.021.
19. **Zhang H, Liang J, Hua B *et al.*** (**2017**): Utility of morning sample of urine protein/creatinine ratio for assessment of proteinuria in patients with lupus nephritis. Annals of the Rheumatic Diseases, 76: 592-96.
20. **Akin D, Ozmen S** (**2019**): An unresolved issue: The relationship between spot urine protein-to-creatinine ratio and 24-hour proteinuria. Journal of International Medical Research, 47(3): 1179–1184.
21. **Medina-Rosas J, Yap K, Anderson M *et al.*** (**2016**): Utility of urinary protein-creatinine ratio and protein content in a 24-hour urine collection in systemic lupus erythematosus: A systematic review and meta-analysis. Arthritis Care & Research, 68(9): 1310–1319.
22. **Wahbeh A, Ewais M, Elsharif M** (**2009**): Comparison of 24-hour urinary protein and protein-to-creatinine ratio in the assessment of proteinuria. Saudi Journal of Kidney Diseases and Transplantation, 20(3): 443–447.
23. **Mendelson L, Sanchorawala V, Connors L *et al.*** (**2022**): Correlation between 24-hour urine protein and random urine protein-creatinine ratio in amyloid light-chain amyloidosis. Kidney Medicine, 4(4): 100427. doi: 10.1016/j.xkme.2022.100427.
24. **Montero N, Soler M, Pascual J *et al.*** (**2012**): Correlation between proteinuria in spot urine samples and 24-hour urine collections in patients with chronic kidney disease. Nefrología, 32(4): 439–445.
25. **Olayinka O, Alao M, Osho P *et al.*** (**2023**): Spot urine protein-to-creatinine ratio versus 24-hour urinary protein in evaluation of proteinuria in Nigerian patients with kidney disease. West African Journal of Medicine, 40(3): 203–208.
26. **Biradar S, Kallaganad G, Srinivas P *et al.*** (**2011**): Correlation of spot urine protein-creatinine ratio with 24-hour urine protein in patients with preeclampsia and eclampsia. Indian Journal of Nephrology, 21(2): 104–107.
27. **Nischintha S, Anil M, Devaraj U *et al.*** (**2014**): Correlation of spot urinary protein-creatinine ratio with 24-hour urinary protein in glomerular disease. Saudi Journal of Kidney Diseases and Transplantation, 25(3): 469–473.
28. **Hu S, Wu J, Dong W *et al.*** (**2009**): Correlation between proteinuria and lipid profile in children with nephrotic syndrome. Pediatric Nephrology, 24(4): 589–593.