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CORRELATION BETWEEN URINE PROTEIN CREATININE RATIO AND GLOMERULAR FILTRATION RATE IN PRE-DIALYSIS CHRONIC KIDNEY DISEASE PATIENTS AT NGOERAH GENERAL HOSPITAL DENPASAR

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ABSTRACT

Background: Chronic Kidney Disease (CKD) is a global health problem with increasing prevalence and high mortality. By definition, CKD is characterized by abnormalities in kidney structure or function lasting for more than three months. Presence of protein in the urine (proteinuria) is one of the key indicators for assessing intrinsic structural damage in CKD. On the other hand, index for evaluating kidney function is Glomerular Filtration Rate (GFR). The degree of proteinuria influences renal function, with higher levels leading to faster deterioration of renal function.

Objective: To investigate correlation between UPCR and GFR in pre-dialysis CKD patients at Ngoerah General Hospital Denpasar

Methods: Analytical observation study with a cross-sectional design involving 40 pre-dialysis CKD patients at Ngoerah General Hospital Denpasar during the period 2023–2024 who met the inclusion and exclusion criteria. The correlation between UPCR and GFR was assessed using Spearman's correlation test.

Results: The study population consisted predominantly of females (57.5%), with a mean age of 46.28 ± 14.49 years, most of whom were in stage 5 (32,5%). The median UPCR was 1.45 (0.05–13.15) mg/mg, while the median GFR was 30.00 (2.74–114.90) mL/min/1.73 m². Spearman's correlation analysis demonstrated a strong negative correlation between UPCR and GFR ($r = -0.615$; $p < 0.001$).

Conclusion: A strong and significant negative correlation was found between UPCR and GFR in pre-dialysis CKD patients. These findings suggest that higher levels of proteinuria are associated with lower renal function in pre-dialysis CKD patients.

Keywords: Chronic Kidney Disease, Urine Protein Creatinine Ratio, Glomerular Filtration Rate

INTRODUCTION

Chronic Kidney Disease (CKD) is a global health problem with increasing prevalence and high mortality.[1] According to Kidney Disease: Improving Global Outcomes (KDIGO), CKD is defined as a clinical syndrome characterized by abnormalities in kidney structure or function that lasting for more than three months and have health implications.[2] The incidence of CKD increases with age and the presence of comorbidities, with the most common causes of CKD are type II diabetes mellitus (30–50%) and hypertension (27.2%).[3]

Data from the Global Burden of Disease (GBD) in 2017 showed that the global prevalence of CKD was 9.1%, accounting for approximately 1.2 million deaths. Mortality due to CKD has continued to rise, reaching 1.43 million deaths worldwide in 2019.[4] It is estimated that by 2040, CKD will become the fifth leading cause of death globally.[5] In 2018, the prevalence of CKD in Indonesia was 0.38%, with 19.33% of cases undergoing hemodialysis therapy. In the province of Bali, the prevalence was slightly higher at 0.44%.[6] Although these numbers appear low, only 0.1% of CKD cases in Indonesia are estimated to be detected.[7]

The overall burden of CKD is considerably higher compared to other diseases with similar prevalence rates. The cost of CKD management increases exponentially with disease progression.[8] Treatment costs under the Indonesian National Health Insurance program showed that CKD was the second-largest healthcare expenditure after cardiovascular diseases, with approximately 2.68 trillion rupiah spent on kidney failure treatment in 2015.[9] In the advanced stages of CKD, Renal Replacement Therapy (RRT) such as dialysis or kidney transplantation becomes necessary. However, these treatments are costly, and only about 10% of those requiring RRT have access to it, making CKD and RRT accessibility major global health challenges.[10]

The best index currently used to assess kidney function is Glomerular Filtration Rate (GFR). The GFR represents the

volume of plasma filtered from the glomerulus into Bowman's capsule over a specific period of time and is normalized to body surface area, expressed in mL/min/1.73 m².[11] In healthy individuals, the GFR is estimated around 120 mL/min/1.73 m².[12] The calculation of GFR can be obtained using measured clearance of an exogenous filtration marker or by using an endogenous filtration marker. The calculation of GFR using exogenous filtration markers, such as injection of iohexol, provides higher accuracy. Nevertheless, in clinical practice, the use of endogenous filtration markers, such as creatinine, estimated through the CKD-EPI equation, is more commonly applied.[13,14] Based on the glomerular filtration rate (GFR), CKD is classified into six stages: stage 1 (GFR ≥ 90 mL/min/1.73m²), stage 2 (GFR 60–89 mL/min/1.73m²), stage 3a (GFR 45–59 mL/min/1.73m²), stage 3b (GFR 30–44 mL/min/1.73m²), stage 4 (GFR 15–29 mL/min/1.73m²), and stage 5 (GFR < 15 mL/min/1.73m²).[2]

Proteinuria is an important marker which reflects intrinsic renal pathology and a strong predictor of CKD prognosis. Proteinuria refers to the presence of proteins such as albumin, Bence-Jones protein, and mucoproteins in the urine. Proteinuria may result from increased glomerular permeability in filtering blood (glomerular proteinuria) or from tubular dysfunction leading to impaired reabsorption of proteins that pass into the glomerular filtrate (tubular proteinuria). In glomerular proteinuria, larger proteins with higher molecular weights such as albumin (MW = 69,000 Da) are able to pass through the filtration barrier and are subsequently excreted in the urine. On the other hand, tubular proteinuria results in a more severe degree of proteinuria, characterized by the massive excretion of positively charged, low molecular weight proteins in the urine.[15]

Methods for measuring urinary protein can be categorized into semiquantitative and quantitative methods. The semiquantitative method utilizes urine dipsticks to detect the presence of proteins but cannot accurately measure concentrations, particularly at levels above 1 g/day.[15] Quantitative methods, while requiring more complex laboratory procedures, provide precise numerical data. The gold standard for quantitative proteinuria assessment is a 24-hour urine collection, which captures daily protein fluctuations. However, this method is time-consuming and may reduce patient compliance, potentially affecting accuracy.[16] As an alternative, the Urine Protein Creatinine Ratio (UPCR) can be used. The UPCR is calculated by dividing the protein concentration by the creatinine concentration in a single urine specimen, typically a morning sample.[15] This method is based on the assumption that creatinine excretion remains relatively constant throughout the day, thus serving as a reference for urinary concentration. Consequently, the ratio of protein to creatinine in a single specimen reflects total daily protein excretion, regardless of whether the urine sample is concentrated or dilute.[17] The normal value of the UPCR is less than 0.2 mg/mg.[16]

The progression of CKD leads to an increased amount of protein entering the renal tubules. As a result, oxygen consumption in the proximal tubules rises to compensate for the enhanced reabsorption process, ultimately leading to tubular hypoxia and tubulointerstitial fibrosis.[18] When proteins are absorbed and reabsorbed in the proximal tubules, their interaction with proximal tubular epithelial cells triggers the release of exosomes containing messenger RNA (mRNA) and transforming growth factor-β (TGF-β). These molecules stimulate tubular cell proliferation, apoptosis, and gene transcription, resulting in tissue fibrosis and a decline in kidney function.[19]

Patients with CKD often remain asymptomatic in the early stages, yet the kidney damage that occurs is irreversible.[20,21] The health implications of CKD are highly diverse, affecting multiple organ systems.[3] Based on the background of the problem, the objective of this study is to determine the correlation between UPCR and GFR in pre-dialysis CKD patients. It is expected that this research will provide data on the relationship between UPCR and GFR in pre-dialysis CKD patients, which can serve as a basis for further studies and provide education on the importance of evaluating UPCR in minimizing CKD complications.

METHODS

This study is an analytical observational study with a cross-sectional design, which has been approved [2025.01.1.0318] by the Research Ethics Committee of the Faculty of Medicine, Udayana University. The research samples were obtained from pre-dialysis CKD patients who received treatment at Ngoerah General Hospital, Denpasar during the period of 2023–2024 and met the inclusion and exclusion criteria. The inclusion criteria of this study were patients who had been diagnosed with CKD by an internist and had not yet undergone dialysis or kidney transplantation, male or female patients aged 21–70 years, and those with recorded UPCR and GFR examinations performed at the same time to minimize the influence of time-related factors on the differences between UPCR and GFR values. The exclusion criteria were incomplete medical records and pregnant patient.

Samples were obtained using a consecutive sampling method. The sample size was determined using the formula for a correlational analytical study, with $Z\alpha = 1.96$, $Z\beta = 0.84$, and a correlation coefficient of –0.441, resulting in a minimum required sample size of 38.[22] Based on medical record data, a total of 414 patients diagnosed with CKD by internist were identified. Of these, 64 patients were excluded for not meeting the age criteria (21–70 years), leaving 350 eligible subjects. From this group, 56 patients who had undergone dialysis therapy and 11 patients who had received kidney transplantation were excluded, resulting in 283 patients who had not received Renal Replacement Therapy (RRT). Among these 283 subjects, 243 were excluded due to UPCR and GFR examinations were not performed at the same time. Consequently, 40 subjects remained for analysis. These subjects were then assessed according to the exclusion criteria, and no incomplete medical records or pregnancy were found. Therefore, the total number of samples that met both the inclusion and exclusion criteria was 40 subjects.

Data obtained from the medical records included age, sex, CKD stage, UPCR value, and GFR value. The data obtained from this study were analyzed and processed statistically using the Statistical Package for the Social Sciences (SPSS) version 26. All data obtained in this study were analyzed descriptively. Descriptive analysis began with normality test using the Shapiro–

Wilk test. The age variable was found to be normally distributed (p = 0,064) and therefore presented as mean ± standard deviation (SD). In contrast, the UPCR and GFR variables were not normally distributed (p < 0,001 and p = 0,002) and were thus presented as median (minimum–maximum). Since the data were not normally distributed, the correlation analysis was performed using the Spearman correlation test.

RESULTS

The characteristics of the study population are presented in **Table 1**. Based on the statistical analysis, the mean ± standard deviation (SD) of the participants age was 46.28 ± 14.49 years, with the youngest being 23 years old and the oldest 70 years old. The majority of the study samples were female (n = 23; 57,5%). Most of the study participants were in stage V CKD (n = 13; 32,5%). The median UPCR value was 1.45 mg/mg, with the lowest value being 0.05 mg/mg and the highest 13.15 mg/mg. The median GFR value was 30.00 mL/min/1.73 m², with the lowest value being 2.74 mL/min/1.73 m² and the highest 114.90 mL/min/1.73 m².

The results of the Spearman correlation test showed a strong and significant negative correlation between UPCR and GFR values, with a correlation coefficient of r = –0.615 (p < 0.001). This result indicates that an increase in UPCR is associated with a decrease in GFR.

Table 1. Characteristics of the study population

Characteristics	n (%)	Mean ± SD	Median (minimum–maximum)
Age (years)		46.28 ± 14.49	
Sex			
Male	17 (42.5)		
Female	23 (57.5)		
Staging of CKD			
Stage I	2 (5)		
Stage II	6 (15)		
Stage IIIa	4 (10)		
Stage IIIb	8 (20)		
Stage IV	7 (17.5)		
Stage V	13 (32.5)		
UPCR (mg/mg)			1.45 (0.05–13.15)
GFR (mL/minute/1,73m^2)			30.00 (2.74–114.90)

CKD: Chronic Kidney Disease; UPCR: Urine Protein Creatinine Ratio; GFR: Glomerular Filtration Rate

DISCUSSION

This study involved 40 pre-dialysis CKD patients who met the inclusion and exclusion criteria as research subjects. The sample size was similar to that of a study by Surya et al. (2018) which also included 40 patients.[9] In several other studies, the sample size was larger for example, Puspita et al. (2019) included 50 samples, while Rosdiana et al. (2020) included 54 samples.[23,24]

The subjects in this study ranged in age from 23 to 70 years, with a mean ± SD of 46.28 ± 14.49 years. This finding is consistent with the results of Puspita et al. (2019), who reported a mean age of 49 years among pre-dialysis CKD patients.[23] These results indicate that most pre-dialysis CKD patients belong to the middle-aged group, in which kidney function begins to decline by approximately 0.75 mL/min/1.73 m² per year after the age of 40, and this decline can be exacerbated by conditions such as hypertension, diabetes mellitus, and other comorbidities.[3] Nonetheless, these findings differ from the Basic Health Research Indonesia (Riskesdas, 2018) data, which showed the highest prevalence of CKD in the 65–74-year age group. Sun et al. (2019) reported that the incidence of CKD among younger individuals has been increasing, with an Estimated Annual Percentage Change (EAPC) in the CKD Age-Standardized Incidence Rate (ASIR) of 0.98% per year.[25] Similarly, Wang et al. (2025) stated that unhealthy lifestyle changes, obesity, and poor dietary patterns contribute to an increased risk of CKD in younger populations.[26]

The majority of subjects in this study were female (57.5%). This proportion is consistent with the findings of Rosdiana et al. (2020), who reported a female percentage of 55.6%.[24] Similarly, Wyld et al. (2022) stated that the prevalence of CKD is 1.3 times higher in females than in males.[27] The higher proportion of females among CKD patients may be explained by biological and sex-specific risk factors, such as increased susceptibility to kidney damage due to pregnancy-induced hypertension, gestational diabetes, and shorter urinary tract structure that associated with higher risk of recurrent urinary tract infections (UTIs).[28]

The distribution of patients according to the Kidney Disease: Improving Global Outcomes (KDIGO, 2024) classification showed an uneven pattern across CKD stages. The highest proportion was observed in stage 5 (GFR < 15 mL/min/1.73 m²), accounting for 32.5% of subjects, with a median GFR of 30.00 mL/min/1.73 m² and a range of 2.74–114.90 mL/min/1.73 m². These findings differ from those reported by Hill et al. (2016) and the Global Burden of Disease Study (2017), which indicate that, globally, the majority of CKD patients are in stage 3.[4,29] This difference may be attributed to the characteristics of the study setting, Ngoerah General Hospital,

which is a tertiary referral center in Denpasar City and the Province of Bali, which consequently manages a higher proportion of advanced CKD cases, particularly stage 5, compared to earlier stages. Moreover, the results of this study are consistent with the findings of Kampmann et al. (2023), who reported that age stratification across CKD stages showed that younger patients were more likely to present as incident CKD stage 5 or ESKD cases. This may reflect a more rapid progression of kidney disease in younger individuals, or the fact that serum creatinine testing is less frequently performed in younger populations, leading to delayed diagnosis until the onset of more severe clinical manifestations.[30] This consistency supports that the middle-aged population in this study tended to be incidentally diagnosed at advanced CKD stages, with most subjects not yet undergoing dialysis therapy, aligning with the characteristics of the pre-dialysis CKD group included in the present research.

Most of the 40 pre-dialysis CKD subjects in this study exhibited elevated UPCR values, with a median of 1.45 and a range of 0.05–13.15 mg/mg. Six subjects (15%) had normal UPCR values (< 0.2 mg/mg), while 34 subjects (85%) showed a significant increase in UPCR (> 0.2 mg/mg). These findings are consistent with the results of Qin et al. (2020), who reported a mean ± SD UPCR value of 2.09 ± 3.22 among CKD patients. Nevertheless, proteinuria does not always occur in all CKD patients, particularly in the early stages, due to certain etiologies such as Diabetic Kidney Disease (DKD) and Autosomal Dominant Polycystic Kidney Disease (ADPKD).[31] In addition, a normal UPCR value may be attributed to the use of antihypertensive therapy, such as angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blocker (ARB). The ACEi/ARB therapy induces dilation of the glomerular efferent arteriole, thereby reducing intraglomerular filtration pressure and subsequently decreasing protein leakage from the glomerular capillaries into the urine.[32]

The present study demonstrated a strong and significant negative correlation between UPCR and GFR, with a correlation coefficient of r = –0.615 (p = 0.000). This finding suggests that higher degrees of proteinuria, as reflected by increased UPCR values, are associated with lower kidney function as indicated by reduced GFR levels. The strong and significant negative correlation observed in this study is consistent with findings from several previous studies.. Rosdiana et al. (2020), in a multicenter study across five hospitals in Riau Province, reported a significant association between proteinuria (measured semi-quantitatively) and decreased GFR (p = 0.016).[24] However, Surya et al. (2018) found no significant correlation between urinary protein (assessed by semi-quantitative urine dipstick) and eGFR among CKD patients at Dr. M. Djamil General Hospital, Padang (p = 0.118).[9] This discrepancy may be explained by differences in measurement methods, as semi-quantitative urine dipstick tests have lower sensitivity and specificity compared to quantitative assessments such as UPCR.[33]

Furthermore, Qin et al. (2022) reported a positive association between UPCR levels and CKD progression [HR = 1.210, 95% CI: 1.760–1.903, p < 0.00001], indicating that each one-unit increase in UPCR was associated with a 12.1% higher risk of CKD progression. These findings further support the predictive value of UPCR in assessing renal function decline.[34]

This finding also supports the KDIGO (2024) guidelines, which recommend that UPCR and GFR assessments be performed at least once annually. These evaluations play a key role in risk stratification, monitoring disease progression, and preventing further complications. Therefore, the findings of this study underscore the importance of routine UPCR monitoring as a clinical parameter for evaluating patient condition and guiding early intervention strategies in CKD management.[2]

From the pathophysiology of proteinuria, proteinuria serves not only as a marker of renal injury but also as an active contributor to kidney damage. Proteinuria reflects increased glomerular membrane permeability or impaired tubular reabsorption of filtered proteins. Proteins that pass into the glomerular filtrate are reabsorbed in the proximal tubule through binding with the Neonatal Fc Receptor (FcRn), apical endocytic receptors, megalin, and cubilin. The binding of albumin to FcRn triggers transcytosis, allowing it to be transported back into systemic circulation. Conversely, the binding of proteins to megalin and cubilin induces endocytosis through the formation of clathrin-coated pits, followed by lysosomal proteolysis, after which the resulting amino acids are released into the bloodstream. Excessive protein reabsorption by proximal tubular cells triggers inflammatory responses, oxidative stress, and the activation of profibrotic cytokines such as transforming growth factor-beta (TGF-β), leading to interstitial fibrosis, which further exacerbate renal injury and functional decline.[18,19,35]

The limitation of this study is that the analysis did not categorize samples based on the etiology of CKD and did not differentiate patients who had received treatments such as ACE inhibitors or ARBs, which could potentially influence UPCR and GFR values.

CONCLUSION

In conclusion, there was a strong and significant negative correlation between UPCR and eGFR among pre-dialysis CKD patients, indicating that an increase in UPCR levels is associated with a decrease in eGFR. These findings support the use of UPCR measurement as part of the management and evaluation of CKD patients. Future studies are recommended to further investigate the influence of CKD etiological factors and administered therapies on the values and correlation between UPCR and GFR.

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