Pharmacogn J. 2024; 16(3): 538-544
A Multifaceted Journal in the field of Natural Products and Pharmacognosy
www.phcogj.com

Original Article

Renal Resistive Index and Estimated Glomerular Filtration Rate in Nondiabetic Chronic Kidney Disease Stage 3: A Cross-Sectional Analytical Study

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ABSTRACT

Background: Renal resistive index (RRI) and estimated glomerular filtration rate (eGFR) are predictive markers for chronic kidney disease (CKD) progression. **Aim:** To evaluate RRI value, eGFRcr-cys and renal biomarker in nondiabetic patients with CKD stage 3 in Bangkok, Thailand. **Methods:** A cross-sectional analytical analysis was conducted involving nondiabetic patients with CKD stage 3, aged 35-85 years. Ultrasound was used to assess the RRI of arteries in both kidneys. Patients underwent assessments of serum and urine 24-hour. **Results:** Among the 61 participants (67.2% male; mean age 69.03 ± 12.59 years), the mean eGFRcr-cys was 41.63 ± 8.64 mL/min/1.73 m², and the mean RRI was 0.65 ± 0.06. Patients were categorized into three RRI groups: low (<0.65, n=35), intermediate (0.65-0.70, n=14), and high (>0.70, n=12). The high RRI group showed a mean RRI of 0.73 ± 0.05 ($p < 0.01$). Among those with high RRI group were significant decreased right kidney size ($p<0.05$) and they had a lower BMI, averaging 22.49 ± 3.48. An increase in PP (59.66 ± 13.84, p=0.04) was also significant in this group. The correlations coefficient of RRI value showed a significant positive correlation with age ($p<0.05$) and significant negative with BMI ($p<0.05$). In addition, eGFRcr-cys displayed a significant negative correlation with UAGT and 24hUP ($p<0.05$) and a significant while eGFRcr-cys positive correlation with both kidney size and urine iNOS($p<0.01$). **Conclusion:** An increase in RRI is inversely linked to age, BMI, and PP. Lower eGFR is correlated with factors that cause CKD progression.

Keywords: Renal ultrasonography, Renal resistive index, Chronic kidney disease, Estimated glomerular filtration rate.

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History

- Submission Date: 12-04-2024;
- Review completed: 18-05-2024;
- Accepted Date: 01-06-2024

DOI : 10.5530/pj.2024.16.85

Article Available online

http://www.phcogj.com/v16/i3

INTRODUCTION

Chronic kidney disease (CKD) is characterized by a persistent alteration in kidney function and structure for a period exceeding three months, representing a significant global health challenge. Often a silent precursor to severe renal impairment, renal fibrosis marks an irreversible[1] and critical phase in the progression of CKD, signaling advanced structural and functional kidney damage.[2] The renal resistive index (RRI), assessed through Doppler sonography, has become increasingly important in nephrology and general medicine. Initially employed for diagnosing renal disorders, RRI has evolved into a crucial prognostic marker in chronic kidney disease, playing a key role in evaluating the success of revascularization after renal artery stenosis treatment and predicting outcomes in renal transplantation and acute kidney injury among critically ill patients.[3,4,5] Furthermore, RRI acts as an indicator of flow resistance in renal parenchymal vessels, playing a pivotal role in assessing morphological and vascular changes, thereby aiding in the early diagnosis and continuous monitoring of CKD.[6] Vascular abnormalities in CKD, often related to atherosclerosis, contribute to the pathological changes detected by RRI measurements atherosclerosis.[7-8] Additionally, studies have demonstrated a significant correlation between RRI values and renal dysfunction in both diabetic and non diabetic kidney disease patients, underscoring RRI's utility in tracking renal health deterioration and providing a comprehensive view of CKD.[9] In hypertensive patients, an elevated RRI has been linked with retinopathy and proteinuria, highlighting its predictive value for end-organ damage and the broader impact of systemic hypertension on renal function.[10] Moreover, RRI has been associated with renal arteriolosclerosis, underscoring its potential in reflecting the extent of vascular pathology in CKD.[11] The assessment of RRI also extends to evaluating renal endothelial function, where its response to nitric oxide synthase inhibition has been examined, offering insights into the vascular components of kidney health.[12] Evidence suggests that RRI could serve as an effective prognostic tool for anticipating the course of kidney disease progression, thus becoming a focal point in nephrological research.[13] Considering the multifaceted role of RRI in diagnosing and prognosticating CKD, understanding its determinants is essential, especially in non diabetic CKD patients, where the etiological factors and disease progression may differ from those in diabetic individuals. Therefore, this study aimed to assess RRI value, eGFRcr-cys and renal biomarker in nondiabetic patients with CKD stage 3 in Bangkok, Thailand.

MATERIALS AND METHODS

Study Design and Patients Characteristics

This cross-sectional analytical study involved 61 consecutive nondiabetic CKD stage 3 patients from

Cite this article: Wongmanit P, Sriyakul K, Tungsukruthai P, Supasyndh O, Tungsukruthai S, Phetkate P. Renal Resistive Index and Estimated Glomerular Filtration Rate in Nondiabetic Chronic Kidney Disease Stage 3: A Cross-Sectional Analytical Study. Pharmacogn J. 2024;16(3): 538-544.

Thailand, conducted over three months (October-December 2021) in Bangkok, Thailand. Ethical approval was granted by the Human Ethics Committee of Thammasat University (Medicine) under protocol No. MTU-EC-OO-0-278/63, COA 041/2021, in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants. Patients were recruited from the Nephrology outpatient department at Phramongkutklao Hospital. The inclusion criteria were CKD stage 3 patients, of any gender, aged over 35 years, with an eGFR ranging from 30-59 mL/min/1.73 m^2. Patients with active cancer, heart disease, liver disease, diabetes, pregnancy, breastfeeding, alcoholism, or autoimmune disease were excluded.

Blood and Urine Examination

Each patient underwent assessments including age, gender, weight, height, body mass index (BMI), and blood pressure. Within 24 hours before the ultrasonographic assessment, biochemical serum tests were conducted for hemoglobin (Hb), phosphorus (PO_4), albumin (Alb), and estimated glomerular filtration rate calculated using creatinine and cystatin C (eGFRcr-cys). Additionally, a 24-hour urine collection was analyzed for urinary angiotensinogen (UAGT), 24-hour urine potassium (24hUK), inducible nitric oxide (iNOS), and 24-hour urine protein (24hUP).

Ultrasonographic Determination of the Renal Resistive Index Assessment

Renal duplex ultrasonography to measure the RRI was performed using a Toshiba Aplio™ 500 ultrasound machine. This procedure involved examining the kidney size, parenchymal thickness, and RRI, employing the pulse wave mode to assess blood vessel function. The examination targeted the interlobar arteries, divided into upper, middle, and lower parts. The machine measured peak systolic velocity (PSV) and end diastolic velocity (EDV), calculating the Doppler RRI as ([PSV - EDV] / PSV). An average of three readings from different sections of both kidneys was recorded (Figure 1).[14]

Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) version 25. A two-tailed p-value of less than 0.05 was considered statistically significant for all analyses. Continuous variables were reported as mean ± standard deviation (SD). Comparisons among RRI risk categories were assessed using one-way ANOVA or Kruskal-Wallis and Mann-Whitney U tests, as appropriate. For descriptive purposes, clinical and demographic variables were presented by RRI categories, with cutoffs at 0.65 and 0.70. Correlations between the renal resistive index, eGFRcr-cys and selected demographic and laboratory variables were evaluated through Spearman's rank correlation.

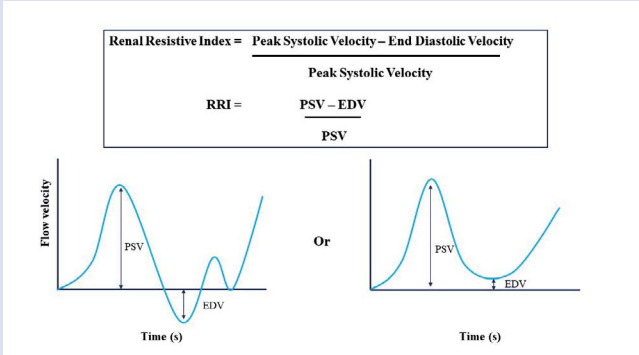

Figure 1: Calculation the renal resistive value index (RRI), where PSV is the peak systolic velocity and EDV is the end diastolic velocity.

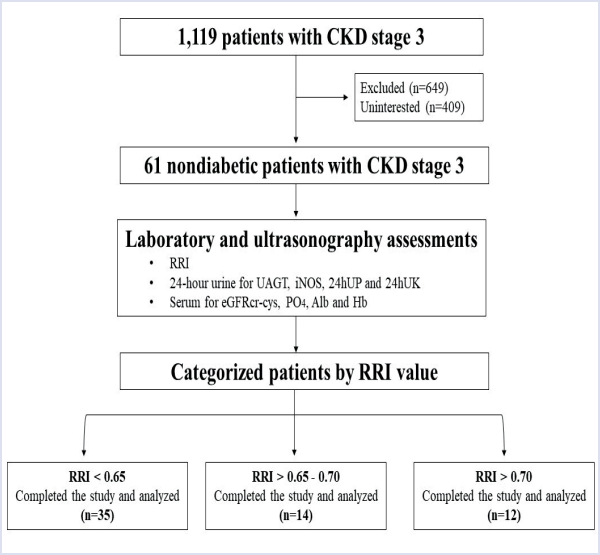

Figure 2: Study flow chart.

RESULTS AND DISCUSSION

Baseline Demographic and Characteristic of Participants

A total of 61 patients with nondiabetic chronic kidney disease stage 3 in the study. There were 41 males and 20 females (males 67.2%). Average RRI was detected equal to 0.65 ± 0.06, categorized patients into 3 groups based on their RRI values into: < 0.65 (n = 35), >0.65-0.70 (n = 14), and >0.70 (n =12). The mean age was 69.03 ± 12.59 years, with the oldest average age observed in the highest RRI group (75.16 ± 6.01 years), although this difference was not statistically significant (p = 0.08). BMI differences neared significance (p =0.05), with the lowest BMI found in the highest RRI group (22.49 ± 3.48 kg/m^2). Pulse pressure differed significantly (p=0.04), with higher values in the >0.70 RRI group (59.66 ± 13.84 mmHg). There were no significant differences in smoking status, eGFRcr-cys, hemoglobin levels, serum phosphorus, albumin, urinary indicators such as iNOS, UAGT, potassium (K^+), and protein excretion, along with kidney size and parenchymal thickness across RRI categories. However, the right kidney size was significantly smaller in the > 0.70 RRI group (8.58 ± 0.92 cm, p = 0.03), and the mean RRI values were significantly higher in the > 0.70 group for both kidneys (p < 0.01), indicating potential structural and functional implications associated with higher RRI values in nondiabetic patients with CKD stage 3, as shown in Table 1.

The Correlations Between Renal Resistive Index with Demographic and Laboratory Parameters in Nondiabetic Patients with CKD stage 3

The analysis of the correlation coefficients (rho) of various factors with the RRI values in patients revealed a significant correlation between RRI value and age (r = 0.277, p = 0.031). Additionally, increased RRI levels were associated with a decrease in BMI (r = -0.261, p = 0.043), as shown in Table 2.

The Correlations Between Estimate Glomerular Filtration Rate with Demographic and Clinical Laboratory Parameters in Nondiabetic Patients with CKD stage 3

The correlation coefficients (rho) between eGFRcr-cys and various demographic and clinical laboratory parameters in nondiabetic patients with CKD stage 3 were analyzed using Spearman's rank

Table 1: Comparison demographic characteristics and clinical of nondiabetic patients with CKD stage 3: classified by renal resistive index (RRI) value.

	Renal resistive index (RRI)				p-value
	Overall (n=61)	< 0.65 (n=35)	> 0.65-0.70 (n=14)	> 0.70 (n=12)	
Sex (n, %)					
Female	20 (32.8)	12(34.3)	4(28.6)	4(33.3)	0.92
Male	41 (67.2)	23(65.7)	10(71.4)	8(66.7)	
Mean age (yr)	69.03±12.59	68.85±11.83	64.21±16.61	75.16±6.01	0.08
Age (n, %)					
< 60 yr.	13 (21.3)	9(25.7)	4(28.6)	0 (0)	0.12
≥60 yr.	48 (78.7)	26(74.3)	10(71.4)	12(100.0)	
Mean BMI (kg/m^2)	25.15±12.59	25.69±3.74	26.07±6.68	22.49±3.48	0.05*
BMI (kg/m^2), (n, %)					
< 18.5	3 (4.9)	1(2.2)	0 (0)	2(16.7)	0.32
18.5-24.9	19 (31.1)	8(22.9)	5(35.7)	6(50.0)	
25-29.9	14 (23)	9(25.7)	3(21.4)	3(16.7)	
30-39.9	16 (26.2)	11(31.4)	4(28.6)	1(8.3)	
> 40	9 (14.8)	6(17.1)	2(14.3)	1(8.3)	
MAP (mmHg)	94.2±11.3	95.53±11.98	92.69±10.43	92.22±9.54	0.57
PP (mmHg)	51.93±15.37	52.25±16.72	44.5±9.08	59.66±13.84	0.04*
Smoke (%)					
Current	1(1.96)	1(2.9)	0 (0)	0 (0)	0.83
Never	57(93.4)	33(94.3)	13(92.9)	11(91.7)	
Ever	3(4.9)	1(2.9)	1(7.1)	1(8.3)	
eGFR$_c$r-cys (mL/min/1.73 m^2)	41.63±8.64	42.57±8.29	41.50±8.89	30.08±9.55	0.49
Hemoglobin (g/dL)	12.95±1.59	13.12±1.69	13.02±1.49	12.38±1.36	0.37
Serum phosphorus (mg/dL)	3.30±0.55	3.28±0.56	3.23±0.50	3.45±0.60	0.57
Serum albumin (mg/dL)	4.36±0.31	4.33±0.31	4.39±0.35	4.40±0.24	0.73
Urine 24 hr					
iNOS (µmol/L)	319.69±225.70	328.09±218.64	323.13±280.39	291.19±189.88	0.88
UAGT (ng/mL)	14.29±7.68	12.69±4.99	17.97±10.30	14.67±6.61	0.09
24hUK (mmol/24hr)	28.77±17.92	31.24±17.95	25.65±21.13	25.20±13.51	0.46
24hUP(mg/24hr)	167.05±78.91	163.71±78.18	163.57±72.38	180.83±92.87	0.80
Kidney size (cm)					
Right	9.10±0.91	9.12±0.85	9.52±0.90	8.58±0.92	0.03*
Left	9.09±1.04	9.13±0.97	9.28±0.86	8.75±1.40	0.42
Kidney Parenchymal thickness (cm)					
Right	1.06±0.32	1.05±0.34	1.13±0.35	1.02±0.27	0.70
Left	1.09±0.35	1.04±0.31	1.21±0.37	1.09±0.41	0.35
Mean RRI	0.65±0.06	0.60±0.04	0.68±0.01	0.73±0.05	<0.01*

Note: Data are presented as number (%), or mean ± standard deviation (Mean±SD). BMI; Body mass index, MAP; Mean arterial pressure, PP; Pulse pressure, RRI; renal resistive index. iNOS; Inducible nitric oxide synthase, UAGT; urinary angiotensinogen, eGFRcr-cys; estimate glomerular filtration rate creatinine-based and Cystatin C-based, 24hUK; 24-hour urine potassium, 24hUP; 24-hour urine protein. The One-way ANOVA test was used to assess differences among the three groups, p-value was significant difference (≤ 0.05).

Table 2: Correlations of renal resistive index with demographic and laboratory in nondiabetic patients with chronic kidney disease stage 3

		RRI in nondiabetic patients with CKD stage3 (n = 61)	
		rho	p-value
Age		0.277	0.031*
BMI (kg/m^2)		-0.261	0.043*
MAP (mmHg)		-0.251	0.051
PP (mmHg)		0.179	0.168
eGFRcr-cys (mL/min/1.73 m^2)		-0.211	0.103
24hUP (mg/24 hr)		-0.103	0.430
UAGT (ng/mL)		0.237	0.066
Urine iNOS (µmol/L)		-0.191	0.140
Kidney size (cm)			
	Right	-0.153	0.241
	Left	-0.135	0.302
Kidney parenchymal thickness (cm)			
	Right	0.019	0.884
	Left	0.106	0.429
Renal resistive index (RRI)		-0.211	0.103

Note: BMI; Body mass index, MAP; Mean arterial pressure, PP; Pulse pressure, iNOS; Inducible nitric oxide synthase, UAGT; Urinary angiotensinogen, eGFRcr-cys; estimate glomerular filtration rate creatinine-based and Cystatin C-based, RRI: Renal resistive index. 24hUP; 24-hour urine protein. Spearman's rank test, *. Correlation was significant at the < 0.05 level.

Table 3: Correlations of estimate glomerular filtration rate creatinine-based and Cystatin C-based with demographic and laboratory in nondiabetic patients with chronic kidney disease stage 3

		eGFRcr-cys (mL/min/1.73 m²) in nondiabetic patients with CKD stage3 (n = 61)	
		rho	p-value
Age		-0.186	0.151
BMI (kg/m²)		-0.011	0.933
MAP (mmHg)		0.199	0.124
PP (mmHg)		-0.178	0.170
24hUP (mg/24 hr)		-0.259	0.044*
UAGT (ng/mL)		-0.259	0.044*
Urine iNOS (µmol/L)		0.414	0.001**
Kidney size (cm)			
	Right	0.299	0.019*
	Left	0.337	0.008**
Kidney parenchymal thickness (cm)			
	Right	0.198	0.133
	Left	0.201	0.129
Mean RRI		-0.211	0.103

Note: BMI; Body mass index, MAP; Mean arterial pressure, PP; Pulse pressure, iNOS; Inducible nitric oxide synthase, UAGT; Urinary angiotensinogen, eGFRcr-cys; estimate glomerular filtration rate creatinine-based and Cystatin C-based ,RRI; renal resistive index, 24hUP; 24-hour urine protein. Spearman's rank test, *. Correlation was significant at the < 0.05 level, **. Correlation was significant at the 0.01 level.

correlation test. The results indicated a significant negative correlation between eGFRcr-cys and 24-hour urine protein (24hUP) (r = -0.259, p = 0.044) as well as urinary angiotensinogen (UAGT) (r = -0.259, p = 0.044). Conversely, there was a positive correlation between eGFRcr-cys and urinary inducible nitric oxide synthase (iNOS) (r = 0.414, p = 0.001) and kidney size (r = 0.337, p = 0.008), as shown in Table 3.

DISCUSSION

The RRI assessed by Doppler ultrasonography, examining its relationship with kidney arteries and various laboratory parameters, such as eGFRcr-cys, phosphorous, albumin, and hemoglobin. In addition, 24-hour urine tests for UAGT, iNOS, 24hUP, and 24hUK were conducted alongside the RRI assessment in patients with stage 3 CKD, predominantly attributed to hypertension and non diabetic conditions.

This study marks the first instance of exploring these clinical parameters specifically in non diabetic patients with stage 3 CKD, including hypertension, without considering the influence of antihypertensive drugs, which implies that the results from this cohort might not be directly generalizable. In non diabetic CKD patients, the RRI plays a crucial role in predicting renal outcomes and mortality.[9]

Renal duplex ultrasonography for RRI determination is recognized as a reliable method for evaluating CKD severity. Its advantages include the detection of macrovascular abnormalities in the kidneys and providing essential diagnostic and prognostic information.[15] Furthermore, a higher RRI value is considered a predictor of adverse outcomes in CKD patients, signifying a decline in eGFR and potentially indicating a deterioration in ultrasonography metrics. Studies have shown that an intrarenal RRI ≥ 0.80 in nonproteinuric CKD patients of unknown etiology is associated with a faster decline in renal function and increased long-term mortality.[16] Furthermore, changes in RRI values between 1 month and 3 months post-transplantation have been linked to mortality in renal transplant recipients, with different impacts observed in diabetic and non diabetic patients.[17]

The current study revealed that elevated RRI is associated with older age and diabetes but not directly with CKD or a decrease in eGFR.[9,18] In patients with essential hypertension, an increased RRI predicts renal function deterioration, with hemodynamic changes significantly influencing RRI.[19] Factors such as post-surgery renal resistance, intrarenal arterial impedance, and alterations in blood pressure ratios affect the decline in eGFR and contribute to kidney function deterioration alongside an increased RRI.[9,18]

An increase in RRI has been linked to a reduction in kidney size, with studies indicating that higher RRI values are associated with older age, diabetes, and elevated serum creatinine levels. The relationship between eGFR-to-kidney size ratios and kidney function decline suggests that high ratios, indicative of glomerular hyperfiltration, correlate with a more significant reduction in kidney function. These findings imply that an elevated RRI reflects both intra-renal arterial impedance and systemic vascular properties, affecting kidney size and function.[18,20,21,22,23]

In our study, a lower BMI was observed in patients with an RRI exceeding 0.70. Although RRI increases with various factors, a decrease in BMI does not consistently correlate with RRI changes.[9,24] RRI increases parallel with carotid intima-media thickness (CIMT) in diabetic patients with microalbuminuria, indicating a link between renal and vascular changes.[25] Conversely, post-bariatric surgery improvements in renal function and vascular parameters, with reduced renal intravascular resistance, were noted, especially in younger patients,[19] suggesting that while BMI changes may indirectly affect renal function via vascular mechanisms, the direct correlation between BMI decrease and RRI increase is not consistently observed across patient groups.

The rise in RRI positively correlates with advancing age with older individuals more likely to exhibit higher RRI values.[28-29]Additionally, a positive correlation exists between age above 60 years and higher RRI values, emphasizing age as a significant factor influencing RRI.[18, 20-23]

Elevated RRI values are associated with the progression of cardiorenal syndrome, renal function deterioration in essential hypertension, and increased renal blood flow resistance, potentially indicating kidney injury and severity. High RRI levels have also been associated with systemic inflammatory responses in COVID-19 patients. However, there is no direct evidence correlating RRI to blood phosphorus levels, despite the fact that RRI has been associated with renal and cardiovascular outcomes, reflecting renal hemodynamics and function rather than directly influencing blood phosphorus levels.[18,19,26,28,30]

The increase in RRI also correlates with lower hemoglobin levels in CKD patients,[31] with significant associations found in univariate

analysis.[32] While diabetic nephropathy studies showed RRI correlation with systolic blood pressure, microalbuminuria, and glomerular filtration rate, no association with hemoglobin levels was noted.[33] However, in children and adolescents with type 1 diabetes mellitus, increased RRI was significantly linked with higher serum HbA1c levels, suggesting a connection between RRI and glycemic control rather than hemoglobin levels.[18] Higher RRI in hypertensive patients is associated with an increased risk of developing proteinuria, indicating its predictive value for nephropathy. Monitoring RRI can offer valuable insights into proteinuria risk and renal disease progression in various patient populations.[10,16,34]

In this our study, eGFRcr-cys, significant negative correlations with UAGT and 24hUP. levels, while it exhibited positive correlations with urinary iNOS and bilateral kidney sizes. According to a previous study, UAGT levels are positively correlated with blood pressure in hypertensive patients and negatively correlated with eGFR, indicating its association with renal function. Additionally, eGFR has been shown to correlate with kidney size in children aged 1-10 years, where renal dimensions—including length, width, and thickness—positively correlate with eGFR, alongside age, weight, height, BMI, and BSA.[21,35,36,37] Studies indicated that in CKD patients, as the disease progresses, eGFR levels decreased, while proteinuria levels increased.[38] Furthermore, a study on renal hyperfiltration (RHF) in Korean adults found that higher eGFR levels were linked to an increased risk of incident proteinuria, especially in men.[39] GFR also shows a correlation with iNOS activity in various studies. For instance, in diabetic rats, iNOS inhibition led to a decrease in GFR and renal plasma flow, highlighting a role for iNOS in glomerular hyperfiltration.[40] Clinical studies Supported this relationship, showing that basal NOS activity, particularly iNOS, is a major determinant of resting GFR levels.[41] Additionally, in pregnant rats, iNOS inhibition attenuated the increase in GFR, suggesting iNOS involvement in renal hemodynamic changes during pregnancy.[42] Moreover, serum nitric oxide metabolites were associated with an increased risk of chronic kidney disease, underscoring the significance of nitric oxide in renal function.[43] Overall, these findings collectively suggest a significant correlation between iNOS activity and GFR under various physiological and pathological conditions.

CONCLUSION

This study could be concluded that higher RRI values are linked to older age, smaller kidney size, and increased pulse pressure, but not directly correlated with overall CKD severity or eGFR decline. These findings underscore RRI's potential as an indicator of macrovascular changes and its prognostic value in this demographic. The variability in RRI, influenced by factors such as BMI and kidney size, points to the need for further research to understand the complex dynamics affecting RRI in CKD.

LIMITATION

This study has some limitations due to its cross-sectional design, focusing exclusively on nondiabetic patients with stage 3 CKD, It lacks comprehensive data on the underlying causes of CKD and types of antihypertensive drugs administered, which may influence the RRI. Consequently, the findings should be interpreted with caution. Future research should be included a more diverse patient demographic across various stages of CKD and a larger sample size to enhance the generalizability of the results. Additionally, a detailed evaluation of etiological factors and the impact of different antihypertensive medications on the RRI is essential to further understand its clinical relevance and application. Further longitudinal studies with additional variables are required to provide an improved comprehension of the factors influencing RRI.

ACKNOWLEDGEMENTS

This study was support by funding from the Thai Traditional Medicine Knowledge Fund, the Department of Thai Traditional and Alternative Medicine, Ministry of Public Health, Thailand.

CONFLICTS OF INTEREST

There is no competing interest among the authors.

AUTHOR'S CONTRIBUTION

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Cite this article: Wongmanit P, Sriyakul K, Tungsukruthai P, Supasyndh O, Tungsukruthai S, Phetkate P. Renal Resistive Index and Estimated Glomerular Filtration Rate in Nondiabetic Chronic Kidney Disease Stage 3: A Cross-Sectional Analytical Study. Pharmacogn J. 2024;16(3): 538-544.