

Original Article

Beyond eGFR and proteinuria: Noninvasive ultrasonographic markers of renal impairment in diabetic kidney disease

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Abstract

Current literature regarding the combined associations of structural renal ultrasonographic parameters and renal resistive index (RRI) with diabetic kidney disease (DKD) severity remains limited, particularly in Southeast Asian populations. The aim of this study was to explore the associations of renal ultrasonographic parameters with estimated glomerular filtration rate (eGFR) and proteinuria among patients with DKD. A cross-sectional study was conducted involving 51 patients with DKD. Bilateral renal longitudinal diameter, cortical thickness, parenchymal echogenicity, and RRI were assessed using ultrasonography. Renal function was evaluated using the 2021 Chronic Kidney Disease Epidemiology Collaboration creatinine-based eGFR equation, and proteinuria was categorized from urine dipstick results as absent-to-mild (negative, trace, or 1+) or moderate-to-severe (2+ to 4+). The present study found that higher eGFR correlated with greater right renal longitudinal diameter ($r=0.347$, $p=0.013$), but not with left renal diameter ($r=0.253$, $p=0.073$). Cortical thickness was not correlated with eGFR in either kidney. RRI was inversely correlated with eGFR bilaterally ($r=-0.491$ for both; $p<0.001$). Mean eGFR was numerically lower in patients with increased parenchymal echogenicity than in those with normal echogenicity, but the differences were not significant. Renal longitudinal diameter, cortical thickness, and parenchymal echogenicity did not differ significantly between proteinuria groups. The mean right RRI was lower in patients with moderate-to-severe proteinuria than in those with absent-to-mild proteinuria (0.66 ± 0.11 vs 0.75 ± 0.08 , $p=0.038$), whereas left RRI did not differ significantly ($p=0.231$). In conclusion, bilateral RRI was consistently associated with lower eGFR, whereas associations involving structural ultrasonographic parameters and proteinuria were less consistent, suggesting that RRI may complement conventional assessment of kidney function in DKD; however, its clinical and prognostic value requires confirmation in larger prospective studies.

Keywords: Diabetic kidney disease, renal ultrasonography, renal resistive index, estimated glomerular filtration rate, proteinuria

Introduction

Diabetic kidney disease (DKD) affects up to 40% of individuals with diabetes and is a leading cause of chronic kidney disease (CKD) and kidney failure worldwide [1]. Prolonged hyperglycemia initiates interacting metabolic, hemodynamic, inflammatory, and profibrotic pathways that promote glomerular hyperfiltration, endothelial dysfunction, albumin leakage, extracellular



matrix accumulation, glomerulosclerosis, and tubulointerstitial fibrosis [2,3]. Progressive nephron loss subsequently reduces glomerular filtration and may culminate in kidney failure requiring dialysis or transplantation [4]. Nevertheless, early DKD is frequently asymptomatic, and clinically apparent manifestations may not emerge until substantial structural damage has occurred [5]. Delayed recognition therefore limits opportunities to initiate kidney-protective therapy before irreversible functional decline.

Current guidelines recommend screening individuals with diabetes using urinary albumin measurement and estimated glomerular filtration rate (eGFR) [3,4,6]. Although these measures are accessible and prognostically informative, each has important limitations. Albuminuria may fluctuate and can be influenced by acute illness, physical activity, glycemic control, and blood pressure [7], whereas serum creatinine-based eGFR may remain within the reference range during early structural injury [8]. Moreover, some patients develop progressive loss of kidney function without marked albuminuria [9]. Kidney biopsy provides the most definitive assessment of glomerular, vascular, and tubulointerstitial lesions and may distinguish DKD from nondiabetic kidney disease [10]. However, its invasive nature, risk of complications, cost, sampling variability, and limited availability preclude routine use, particularly in patients with a typical clinical presentation.

Renal ultrasonography offers a noninvasive, widely available, and relatively inexpensive method for evaluating renal structure and intrarenal hemodynamics [11]. Conventional parameters—including renal length, cortical thickness, and parenchymal echogenicity—may reflect chronic parenchymal damage, whereas the renal resistive index (RRI), derived from Doppler ultrasonography, provides an indirect measure of intrarenal vascular resistance [12]. Reduced cortical thickness, increased parenchymal echogenicity, and elevated RRI have been associated with lower eGFR, more advanced CKD, and histopathological evidence of glomerular, vascular, and tubulointerstitial injury [13]. In patients with diabetic and nondiabetic kidney disease, RRI has shown moderate-to-strong correlations with histological injury, particularly tubulointerstitial damage, and tends to increase as renal function declines [14].

Studies regarding the combined relationship of structural ultrasonographic parameters and RRI with DKD severity remain limited [15-19], particularly in Southeast Asian populations. Previous studies have commonly evaluated individual parameters, included heterogeneous CKD etiologies, or focused on comparisons between patients with and without nephropathy [20-22]. Therefore, the aim of this study was to explore the associations of renal ultrasonographic parameters with eGFR and proteinuria in patients with DKD at a tertiary referral center in Indonesia.

Methods

Study design and setting

A cross-sectional study was conducted at Endocrinology Outpatient Clinic and Department of Radiology, Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia, from October 2025 to March 2026. Renal ultrasonography was performed bilaterally using a standardized protocol to measure renal longitudinal diameter and cortical thickness, assess parenchymal echogenicity relative to the adjacent liver or spleen, and determine the renal resistive index from spectral Doppler waveforms of the interlobar arteries. Renal impairment was assessed using eGFR, calculated from serum creatinine with the 2021 Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation without a race coefficient [23], and proteinuria, evaluated semiquantitatively by urine dipstick grading from negative to 4+.

Sample size, sampling method, and eligibility criteria

The minimum sample size was calculated using the formula for an analytical cross-sectional study, with a two-sided significance level of 5% and a statistical power of 80%, yielding a minimum requirement of 40 patients. Eligible patients were recruited consecutively until the required sample size was achieved. The inclusion criteria were: (1) a confirmed diagnosis of diabetes mellitus based on documented medical history, current use of glucose-lowering medication, and/or laboratory findings meeting established diagnostic criteria; and (2)

fulfillment of the clinical definition of DKD, indicated by persistent proteinuria and/or an eGFR <60 mL/min/1.73 m² for ≥ 3 months, without clinical evidence suggestive of nondiabetic kidney disease; (3) availability of bilateral renal ultrasonographic measurements, including renal longitudinal diameter, cortical thickness, parenchymal echogenicity, and RRI; and (4) availability of serum creatinine, HbA1c, and urine dipstick protein results obtained within 1 month of renal ultrasonography. The exclusion criteria were: (1) maintenance hemodialysis; (2) acute kidney injury at the time of assessment; (3) sepsis; (4) acute coronary syndrome; (5) an acute cerebrovascular event; or (6) incomplete or technically inadequate renal ultrasonographic data.

Data collection

Demographic and clinical data were obtained from medical records using a standardized data-collection form. The variables collected included age, sex, duration of diabetes, blood pressure, comorbidities, HbA1c, serum creatinine, eGFR, proteinuria, and renal ultrasonographic findings. Laboratory measurements performed within 1 month before or after renal ultrasonography were recorded. When multiple laboratory results were available within this interval, the measurement obtained closest to the date of ultrasonography was used.

Renal ultrasonographic assessment

Renal ultrasonography was performed in the Department of Radiology, Dr. Zainoel Abidin Hospital, Banda Aceh, Indonesia, using a Voluson S8 system (GE HealthCare, Chicago, IL, USA) or an Aloka ultrasound system (Hitachi Aloka Medical, Tokyo, Japan), each equipped with a curvilinear abdominal transducer. All examinations were performed by a fourth-year radiology resident (R.A.S) according to a standardized renal ultrasonography protocol, and the images and measurements were subsequently reviewed and verified by a consultant abdominal radiologist with more than 10 years of experience (N.D.S). Each patient was initially examined in the supine position and repositioned into the right or left lateral decubitus position when required to optimize renal visualization. Both kidneys were evaluated separately in longitudinal and transverse planes, with image settings adjusted to clearly delineate the renal margins, cortex, medullary pyramids, and intrarenal vessels.

The evaluated ultrasonographic parameters were renal longitudinal diameter, cortical thickness, parenchymal echogenicity, and RRI. Renal longitudinal diameter was measured in the longitudinal plane as the maximum distance between the upper and lower renal poles and recorded in centimeters. Cortical thickness was measured at the mid-pole in the longitudinal plane, from the base of a medullary pyramid to the renal capsule, and recorded in millimeters. Measurements were obtained from both kidneys, and the bilateral mean was used in the analysis. Parenchymal echogenicity was assessed by comparing the renal cortex with the adjacent liver on the right and spleen on the left. Echogenicity was classified as: (1) grade 0, cortical echogenicity lower than that of the liver or spleen, with preserved corticomedullary differentiation; (2) grade 1, cortical echogenicity equal to that of the liver or spleen, with preserved corticomedullary differentiation; (3) grade 2, cortical echogenicity greater than that of the liver or spleen, with reduced corticomedullary differentiation; and (4) grade 3, markedly increased cortical echogenicity with poor or absent corticomedullary differentiation. If the echogenicity grades differed between kidneys, the higher grade was used in the analysis. Increased parenchymal echogenicity was defined as cortical echogenicity equal to or greater than that of the reference organ, corresponding to echogenicity grades 1–3.

RRI was measured using spectral Doppler ultrasonography at the interlobar arteries of the upper, middle, and lower poles of each kidney. Peak systolic velocity (PSV) and end-diastolic velocity (EDV) were measured in centimeters per second (cm/s). RRI was calculated using the following equation: $RRI = (PSV - EDV) / PSV$. Three measurements were obtained from each kidney, resulting in six measurements per patient. The mean of these six measurements was used as the patient-level RRI value in the analysis.

Renal impairment severity: Estimated glomerular filtration rate (eGFR) and proteinuria

Renal impairment severity was assessed using eGFR and proteinuria. Serum creatinine measured within 1 month of renal ultrasonography was used to calculate eGFR according to the 2021 CKD-

EPI equation without a race coefficient. The eGFR was expressed in mL/min/1.73 m² and categorized as G1 (≥ 90), G2 (60–89), G3a (45–59), G3b (30–44), G4 (15–29), or G5 (< 15). Proteinuria was assessed semiquantitatively by routine urine dipstick examination according to the hospital laboratory protocol and categorized as negative, trace, 1+, 2+, 3+, or 4+. For inferential analysis, proteinuria was classified as absent-to-mild (negative, trace, or 1+) or moderate-to-severe (2+ to 4+).

Statistical analysis

Normality was evaluated using the Shapiro–Wilk test. Associations involving categorical variables were assessed using the Chi-squared test or Fisher’s exact test. Pearson or Spearman’s correlation tests were used to evaluate associations of renal longitudinal diameter, cortical thickness, parenchymal echogenicity grade, and RRI with continuous eGFR values. Ultrasonographic parameters were compared between patients with absent-to-mild and moderate-to-severe proteinuria using the independent-samples *t*-test or Mann–Whitney *U* test, as appropriate. All analyses were two-sided, with $p < 0.05$ indicating statistical significance, and were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA).

Results

Baseline characteristics of the included patients

Among 51 patients with DKD, the mean age was 61.69 years, 26 (51.0%) were male, and the mean duration of diabetes was 9.67 $\pm$ 5.66 years (**Table 1**). The mean HbA1c level was 8.37 $\pm$ 2.73%, the mean serum creatinine level was 1.97 $\pm$ 2.36 mg/dL, and the mean eGFR was 60.04 $\pm$ 32.93 mL/min/1.73 m². Urine dipstick testing was negative for proteinuria in 39 patients (76.5%); proteinuria grades of 1+ and 2+ were observed in 8 (15.7%) and 4 (7.8%), respectively. The most common eGFR category was G2 (33.3%), followed by G3b (19.6%), G1 (17.6%), G3a (11.8%), G5 (11.8%), and G4 (5.9%). The mean longitudinal diameter was 9.73 $\pm$ 1.61 cm in the right kidney and 9.43 $\pm$ 1.17 cm in the left kidney, and the corresponding mean cortical thicknesses were 13.89 $\pm$ 4.16 mm and 14.04 $\pm$ 4.39 mm. Increased parenchymal echogenicity was identified in 10 right kidneys (19.6%) and 9 left kidneys (17.6%). The mean RRI was 0.74 $\pm$ 0.08 in the right kidney and 0.76 $\pm$ 0.08 in the left kidney.

Table 1. Characteristics of the included patients (n=51)

Characteristic	Frequency (%)
Age (years), mean $\pm$ SD	61.69 $\pm$ 12.24
Sex	
Male	26 (51.0)
Female	25 (49.0)
Diabetes duration (years), mean $\pm$ SD	9.67 $\pm$ 5.66
HbA1c (%), mean $\pm$ SD	8.37 $\pm$ 2.73
Urine dipstick protein grade	
Negative	39 (76.5)
1+	8 (15.7)
2+	4 (7.8)
3+	0 (0.0)
4+	0 (0.0)
Serum creatinine (mg/dL), mean $\pm$ SD	1.97 $\pm$ 2.36
eGFR (mL/min/1.73 m ²), mean $\pm$ SD	60.04 $\pm$ 32.93
KDIGO eGFR category	
G1 (≥ 90 mL/min/1.73 m ²)	9 (17.6)
G2 (60–89 mL/min/1.73 m ²)	17 (33.3)
G3a (45–59 mL/min/1.73 m ²)	6 (11.8)
G3b (30–44 mL/min/1.73 m ²)	10 (19.6)
G4 (15–29 mL/min/1.73 m ²)	3 (5.9)
G5 (< 15 mL/min/1.73 m ²)	6 (11.8)
Renal longitudinal diameter (cm), mean $\pm$ SD	
Right kidney	9.73 $\pm$ 1.61
Left kidney	9.43 $\pm$ 1.17
Cortical thickness (mm), mean $\pm$ SD	
Right kidney	13.89 $\pm$ 4.16
Left kidney	14.04 $\pm$ 4.39

Characteristic	Frequency (%)
Increased parenchymal echogenicity	
Right kidney	10 (19.6)
Left kidney	9 (17.6)
Renal resistive index (RRI), mean±SD	
Right kidney	0.74±0.08
Left kidney	0.76±0.08

eGFR: estimated glomerular filtration rate; HbA1c: glycated hemoglobin; KDIGO: Kidney Disease Improving Global Outcomes; SD: standard deviation

Association of renal ultrasonographic parameters with estimated glomerular filtration rate (eGFR) and proteinuria in patients with diabetic kidney disease

Higher eGFR was positively correlated with the longitudinal diameter of the right kidney ($r=0.347$, $p=0.013$), whereas the correlation with the left kidney diameter did not reach statistical significance ($r=0.253$, $p=0.073$) (**Table 2**). Cortical thickness was not correlated with eGFR in either the right ($r=0.070$, $p=0.627$) or left kidney ($r=-0.020$, $p=0.887$). By contrast, the RRI was inversely correlated with eGFR in both the right and left kidneys ($r=-0.491$ for both; $p<0.001$), indicating that a higher RRI was associated with poorer kidney function.

Table 2. Association of renal ultrasonographic parameters (renal longitudinal diameter, cortical thickness, and renal resistive index) with estimated glomerular filtration rate (eGFR) in patients with diabetic kidney disease

Variable	eGFR (mL/min/1.73 m ²)	
	<i>r</i>	<i>p</i> -value
Renal longitudinal diameter (cm)		
Right kidney	0.347	0.013 ^{a*}
Left kidney	0.253	0.073 ^a
Cortical thickness (mm)		
Right kidney	0.070	0.627 ^b
Left kidney	-0.020	0.887 ^b
Renal resistive index (RRI)		
Right kidney	-0.491	<0.001 ^{b*}
Left kidney	-0.491	<0.001 ^{b*}

eGFR: estimated glomerular filtration rate

^a Analyzed using Spearman correlation test

^b Analyzed using Pearson correlation test

* Statistically significant at $p<0.05$

Mean eGFR was lower among patients with increased parenchymal echogenicity than among those with normal echogenicity in both the right kidney (47.66 ± 34.07 vs 62.69 ± 32.48 mL/min/1.73 m², $p=0.218$) and left kidney (45.44 ± 33.51 vs 63.16 ± 32.35 mL/min/1.73 m², $p=0.145$) (**Table 3**). However, neither difference reached statistical significance.

Table 3. Association between renal echogenicity and estimated glomerular filtration rate (eGFR) in patients with diabetic kidney disease

eGFR (mL/min/1.73 m ²)	Parenchymal echogenicity		<i>p</i> -value ^a
	Normal	Increased	
Right kidney, mean±SD	62.69±32.48	47.66±34.07	0.218
Left kidney, mean±SD	63.16±32.35	45.44±33.51	0.145

eGFR: estimated glomerular filtration rate

^a Analyzed using independent t-test

Renal longitudinal diameter and cortical thickness did not differ significantly between patients with absent-to-mild proteinuria and those with moderate-to-severe proteinuria (all $p>0.05$) (**Table 4**). Increased parenchymal echogenicity was more frequent in the absent-to-mild proteinuria group than in the moderate-to-severe group for both kidneys, although the differences were not statistically significant (13.7% vs 3.9%; $p=0.077$ for both). The mean right RRI was significantly lower in patients with moderate-to-severe proteinuria than in those with absent-to-mild proteinuria (0.66 ± 0.11 vs 0.75 ± 0.08 , $p=0.038$). The left RRI did not differ significantly between the groups (0.71 ± 0.10 vs 0.76 ± 0.08 , $p=0.231$).

Table 4. Association between renal ultrasonographic parameters and proteinuria in patients with diabetic kidney disease

Variable	Proteinuria		p-value
	Absent-to-mild	Moderate-to-severe	
Renal longitudinal diameter (cm)			
Right kidney, median (min–max)	9.59 (7.21–15.00)	9.79 (8.15–12.50)	0.687 ^a
Left kidney, median (min–max)	9.56 (5.61–11.70)	9.44 (9.09–12.00)	0.661 ^a
Cortical thickness (mm)			
Right kidney, mean±SD	1.37±0.42	1.55±0.17	0.404 ^b
Left kidney, mean±SD	1.40±0.45	1.33±0.15	0.758 ^b
Increased parenchymal echogenicity			
Right kidney, n (%)	7 (13.7)	2 (3.9)	0.077 ^c
Left kidney, n (%)	7 (13.7)	2 (3.9)	0.077 ^c
Renal resistive index (RRI)			
Right kidney, mean±SD	0.75±0.08	0.66±0.11	0.038 ^{b*}
Left kidney, mean±SD	0.76±0.08	0.71±0.10	0.231 ^b

^a Analyzed using Mann-Whitney U test^b Analyzed using independent t-test^c Analyzed using Chi-squared test* Statistically significant at $p < 0.05$

Discussion

In the present study, higher eGFR was associated with a greater longitudinal diameter of the right kidney, whereas lower eGFR was associated with higher RRI in both kidneys. No significant association was observed between eGFR and renal parenchymal echogenicity. The positive correlation between right renal longitudinal diameter and eGFR suggests that structural reduction in kidney size accompanies impaired renal function. This finding is consistent with previous CKD studies demonstrating positive bilateral correlations between renal length and eGFR [21,22,24]. In a cohort of 235 patients, renal length correlated positively with eGFR in both the right ($r=0.470$) and left kidneys ($r=0.454$) [22]. Biopsy-linked analyses have similarly shown direct associations of bipolar kidney diameter and parenchymal thickness with eGFR, although kidney length explained only a small proportion of functional variability and its association differed according to the underlying histopathological diagnosis [21]. A computed tomography (CT)-based study also demonstrated that conventional longitudinal diameter correlated less strongly with eGFR than kidney volume or minor-axis length, indicating that renal length provides useful but incomplete information regarding structural disease severity [24]. The absence of a significant association for the left kidney in the present study should therefore not be interpreted as a true unilateral biological difference, but may reflect modest effect size, sample variability, or measurement-related variation.

The association between renal size and function may be particularly complex in diabetic kidney disease because renal hypertrophy can occur during early disease, whereas parenchymal loss and renal contraction generally emerge during advanced kidney failure. CT-derived morphological measurements have shown slightly weaker correlations with eGFR in patients with diabetes than in patients without diabetes [24]. Similarly, renal length in type 1 diabetes was greater than healthy reference values, varied with body size, and declined primarily in advanced renal failure, limiting its value for predicting earlier functional deterioration [17]. In diabetic CKD cohorts, poorer kidney function has also been observed despite renal lengths comparable to those of patients without diabetes and relatively preserved or increased cortical and parenchymal thickness [15]. However, renal length alone did not predict disease progression, whereas a composite ultrasonographic score provided greater prognostic value [15].

Differences were also observed for parenchymal measures. Cortical thickness was not correlated with eGFR in either kidney in the present study, in contrast to the weak associations reported by Habtegiorgis *et al.* and the modest positive correlation between parenchymal thickness and eGFR reported by Getaneh *et al.* ($r=0.25$, $p=0.010$) [18,22]. Both previous studies identified a significant inverse association between renal echogenicity and eGFR ($p<0.001$) [18,22], whereas the present study found only numerically lower eGFR among patients with increased echogenicity, without statistical significance.

Echogenicity and corticomedullary differentiation may reflect renal dysfunction more closely than renal length in patients with CKD [19,22]. Parenchymal thickness has also shown correlations with eGFR that are comparable to or stronger than those observed for renal length [18,19]. Furthermore, renal length varies according to height, weight, body mass index, age, sex, and ethnicity, emphasizing the importance of body-size normalization when interpreting this measurement [15,19]. Accordingly, the significant right-sided association in the present study should be interpreted cautiously and not regarded as evidence of a true biological asymmetry between the kidneys.

By contrast, the present study demonstrated a consistent inverse correlation between eGFR and RRI in both kidneys ($r=-0.491$ for both; $p<0.001$). This finding is comparable to the correlation reported by a previous study in which baseline eGFR was inversely correlated with RRI ($r=-0.521$, $p<0.001$) [25]. That study also found a progressive increase in RRI across advancing CKD stages, with significant differences between patients with and without diabetes in stages I–III [25]. Similarly, Qian *et al.* demonstrated that RRI was independently associated with subsequent eGFR decline ($r=-0.245$, $p<0.001$), supporting its potential value beyond cross-sectional assessment [26]. The present findings are further supported by a previous study demonstrating inverse correlations of eGFR with RRI ($r=-0.712$), and in multivariable analysis, RRI remained a significant predictor of eGFR ($p=0.030$), suggesting that RRI provides information on renal dysfunction beyond conventional biochemical markers [16].

The inverse association may reflect progressive changes in renal microcirculation and parenchymal architecture. During early CKD, predominantly glomerular involvement may coexist with a near-normal RRI. With disease progression, glomerulosclerosis, tubular atrophy, interstitial fibrosis, and intrarenal arteriosclerosis increase vascular resistance and consequently elevate RRI [20]. RRI may therefore detect altered intrarenal hemodynamics even when renal dimensions remain relatively preserved. In contrast, longitudinal diameter, cortical thickness, and echogenicity predominantly reflect accumulated structural remodeling, which may develop later, vary according to the underlying disease, or remain obscured by early diabetic renal hypertrophy [18,19].

Nevertheless, RRI should not be interpreted as a kidney-specific marker. Age, body mass index, pulse pressure, arterial stiffness, and other systemic vascular factors can influence RRI independently of intrinsic renal injury [27,28]. Furthermore, absolute RRI values may remain within the conventionally normal range during stages I–IV CKD despite progressive functional deterioration, suggesting that serial changes may be more informative than a single cutoff value [19]. Structural–functional relationships may be particularly variable in diabetes, and Doppler-based screening has shown limited prognostic performance in some adults with type 1 diabetes [17].

The significantly lower right RRI among patients with moderate-to-severe proteinuria was unexpected and contrasts with most previous evidence. In broader CKD populations, elevated RRI and proteinuria have emerged as independent markers of disease progression, with higher RRI associated with poorer outcomes in both proteinuric and non-proteinuric phenotypes [29]. Accordingly, the present finding should not be interpreted as evidence that greater proteinuria reduces intrarenal vascular resistance. The absence of a corresponding difference in the left kidney suggests that the right-sided association may reflect sampling variability, measurement sensitivity, transient hemodynamic conditions, or multiple testing rather than true biological asymmetry. Physiological right–left differences in RRI are generally small, and larger discrepancies may indicate acquisition variability, renovascular abnormalities, or unilateral renal injury [29]. Standard Doppler protocols therefore obtain measurements from multiple poles of both kidneys and use an averaged value. Nevertheless, dissociation between proteinuria and RRI may occur because these measures reflect different pathological processes. Proteinuria primarily reflects glomerular barrier injury, whereas RRI is influenced by intrarenal vascular resistance, arterial compliance, tubulointerstitial damage, age, systemic blood pressure, and other hemodynamic factors [30]. Proteinuria may therefore increase while RRI remains within the normal range, particularly when serum creatinine and eGFR are relatively preserved. However, this dissociation does not support a consistent inverse relationship. Given the bilateral inverse correlations between RRI and eGFR in the present study, the isolated association between lower

right RRI and greater proteinuria may reflect sample-specific or measurement-related factors. Confirmation requires larger longitudinal studies using quantitative Urine Albumin-Creatinine Ratio (UACR), averaged bilateral RRI measurements, standardized Doppler acquisition, and adjustment for eGFR, systemic vascular factors, medication use, and glycemic control.

Several limitations require consideration. Potential determinants of RRI, including age, blood pressure, pulse pressure, arterial stiffness, body mass index, hydration status, cardiovascular disease, and antihypertensive treatment, were not included in an adjusted analysis. Proteinuria was categorized using semiquantitative urine dipstick results rather than UACR or 24-hour urinary protein excretion, potentially introducing exposure misclassification. Bilateral RRI measurements may also have been affected by operator-dependent acquisition, and interobserver reproducibility was not evaluated. In addition, the absence of kidney biopsy prevented direct comparison of ultrasonographic parameters with glomerulosclerosis, tubulointerstitial fibrosis, and intrarenal vascular injury. Multicenter prospective studies should evaluate whether serial, averaged bilateral RRI measurements independently predict eGFR decline and albuminuria progression after adjustment for established clinical and vascular risk factors.

Conclusion

Bilateral RRI was consistently associated with lower eGFR, whereas associations involving structural ultrasonographic parameters and proteinuria were less consistent, suggesting that RRI may complement conventional assessment of kidney function in DKD. However, its clinical and prognostic value requires confirmation in larger prospective studies.

Ethics approval

Protocol of the present study was reviewed and approved by Ethical Committee for Health Research, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia (Approval No. 280/ETIK-RSUDZA/2025). Written informed consent was obtained from all participants prior to study enrolment.

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None to declare.

Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

This study used artificial intelligence (AI) tool, ChatGPT, for language refinement (improving grammar, sentence structure, and readability of the manuscript). Authors confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the results. The final decisions and interpretations presented in this article were solely made by the authors.

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