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Original Paper

Right Ventricular Dysfunction Across Chronic Kidney Disease Stages: An Echocardiographic Cross-Sectional Study

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ABSTRACT

Background: Chronic kidney disease (CKD) is accompanied by a substantial cardiovascular burden, yet right ventricular (RV) involvement is less consistently characterized than left ventricular disease. The RV is particularly vulnerable to changes in preload, pulmonary vascular load, anemia, volume expansion, and hemodialysis-related hemodynamic stress. A stage-focused assessment may clarify whether conventional echocardiographic RV abnormalities track directly with declining kidney function or reflect a more complex cardiorenal-pulmonary interaction.

Objective: To describe the prevalence and stage-wise distribution of RV systolic dysfunction in adults with CKD and to examine associations among estimated glomerular filtration rate (eGFR), tricuspid annular plane systolic excursion (TAPSE), right ventricular ejection fraction (RV-EF), pulmonary artery systolic pressure (PASP), and left ventricular ejection fraction (LV-EF).

Methods: This stage-focused secondary analysis used a cross-sectional observational cohort of 100 adults with CKD evaluated at the echocardiography clinic of Baquba Teaching Hospital, Diyala, Iraq, between August 2025 and February 2026. Clinical assessment, serum creatinine/urea testing, eGFR estimation, and transthoracic echocardiography were performed. RV systolic function was assessed using TAPSE and RV-EF; PASP was estimated from the tricuspid regurgitation jet. CKD stage and reported normal/abnormal RV categories were retained from the source study. Stage-wise proportions were recalculated from the reported aggregate counts, and chi-square tests were recomputed for the stage-by-RV-category tables.

Results: The cohort included 100 participants; 53% were older than 60 years and 58% were receiving hemodialysis. CKD stage distribution was stage 2, 1%; stage 3, 18%; stage 4, 33%; and stage 5, 48%. Abnormal RV-EF was present in 51%, while abnormal TAPSE was present in 35%. The proportion with abnormal TAPSE was 0.0%, 44.4%, 42.4%, and 27.1% in stages 2–5, respectively (recomputed chi-square $p=0.339$). Abnormal RV-EF occurred in 0.0%, 50.0%, 45.5%, and 56.3% across stages 2–5 ($p=0.576$). Mean eGFR did not differ significantly between normal and abnormal TAPSE groups or between normal and abnormal RV-EF groups. In the reported correlation analysis, PASP correlated inversely with eGFR ($r=-0.207$, $p=0.039$) and TAPSE ($r=-0.256$, $p=0.010$), while LV-EF correlated positively with RV-EF ($r=0.367$, $p<0.001$).

Conclusions: RV systolic abnormalities were common in this CKD cohort but did not show a simple monotonic relationship with CKD stage. The observed associations of PASP with both eGFR and TAPSE suggest that pulmonary vascular load and volume/hemodynamic factors may be clinically important links between renal impairment and RV performance. Comprehensive right-heart assessment may therefore provide information not captured by CKD stage alone. Larger prospective studies using standardized RV measurements and longitudinal outcomes are warranted.

KEYWORDS ***Safety;*** *chronic kidney disease; right ventricle; TAPSE; right ventricular ejection fraction; pulmonary artery systolic pressure; echocardiography; hemodialysis*

1-INTRODUCTION

Chronic kidney disease (CKD) is a persistent disorder of kidney structure or function with broad systemic consequences. Current KDIGO guidance emphasizes both glomerular filtration rate and markers of kidney damage because cardiovascular risk rises progressively as kidney disease advances [1]. In clinical practice, the cardiovascular phenotype of CKD is heterogeneous: hypertension, chronic volume expansion, anemia, arterial stiffness, neurohormonal activation, metabolic disturbance, endothelial dysfunction, and uremic toxin accumulation can act simultaneously. These mechanisms promote structural remodeling of the myocardium and vasculature and help explain why cardiovascular morbidity and mortality remain major concerns in advanced CKD and kidney failure.

The traditional cardiovascular focus in CKD has centered on the left ventricle, particularly left ventricular hypertrophy, systolic dysfunction, diastolic dysfunction, and heart failure. The right ventricle, however, is not simply a passive chamber. It is a thin-walled, highly compliant pump that is exquisitely sensitive to changes in

loading conditions and pulmonary vascular resistance. Even modest increases in RV afterload can impair systolic shortening, increase wall stress, and alter ventricular interdependence. Contemporary echocardiography guidance therefore recommends a systematic right-heart assessment rather than reliance on a single measurement [2]. This is especially relevant in CKD, where pulmonary pressures, volume status, anemia, vascular access flow, and left-sided filling pressures can change over time and may not be reflected by eGFR alone.

Several mechanisms may connect kidney dysfunction to RV abnormalities. Chronic sodium and water retention increases venous return and can raise right-sided filling pressures. Anemia may drive a high-output state, while arteriovenous fistulas used for hemodialysis can further increase cardiac output and RV preload. Pulmonary hypertension may emerge through a combination of volume overload, elevated left-sided filling pressures, endothelial dysfunction, vascular calcification, sleep-disordered breathing, and high-flow vascular access. In addition, the chronic inflammatory and neurohormonal milieu of CKD may contribute to myocardial fibrosis and impaired contractile reserve. These pathways make it plausible that RV dysfunction in CKD is multifactorial and that its relationship to conventional CKD stage may be nonlinear.

Clinical studies support the relevance of this problem but demonstrate considerable variation in prevalence, definitions, and methods. In stage III CKD, Floccari and colleagues reported early right-heart abnormalities detectable by echocardiography [10]. Among patients receiving maintenance hemodialysis, Zhao et al. found pulmonary hypertension in approximately two-fifths of participants and RV dysfunction in nearly half, with pulmonary pressure associated with RV abnormalities [9]. López-Quijano et al. likewise identified RV systolic dysfunction in a subset of hemodialysis patients and examined clinical and echocardiographic factors associated with reduced TAPSE [7]. High-flow arteriovenous access has also been associated with impaired RV function in some cohorts, illustrating how dialysis-related hemodynamics can modify the cardiac phenotype independently of eGFR [8].

More recent work has expanded the emphasis from isolated measures of RV shortening to RV-pulmonary arterial coupling and clinical outcomes. Wang et al. evaluated the TAPSE/PASP ratio in maintenance hemodialysis and reported that impaired RV-pulmonary arterial coupling was associated with excess cardiovascular risk [4]. Nobayashi et al. subsequently showed that RV dysfunction defined by reduced TAPSE was independently associated with mortality in a maintenance hemodialysis cohort [5]. A recent echocardiographic study of chronic renal disease also reinforces the high frequency of RV functional abnormalities and the value of combining conventional and tissue-based measures rather than relying on left ventricular indices alone [3]. These observations underscore the potential clinical importance of RV assessment in nephrology populations.

A key unanswered question is whether RV dysfunction follows a straightforward stage-dependent gradient across CKD. One might expect progressively worse RV performance with lower eGFR, but several competing influences complicate that assumption. Stage 5 patients may be more likely to receive dialysis with active volume management, while patients at earlier stages may differ in blood pressure, anemia, diabetes, volume status, and access type. Conventional TAPSE is also load dependent and reflects longitudinal shortening rather than global RV contractility. RV-EF may capture a broader systolic phenotype but can be difficult to quantify accurately using two-dimensional methods. Therefore, a lack of simple stage-by-stage progression does not necessarily indicate absence of clinically important cardiorenal interaction.

The source cohort for the present manuscript consisted of adult CKD patients referred for cardiac assessment at Baquba Teaching Hospital in Diyala, Iraq. The original study evaluated standard echocardiographic RV measures and renal function. The present manuscript reframes those data as a stage-focused secondary analysis. Rather than reproducing the broad original thesis structure, it concentrates on three questions: (1) How common are abnormal TAPSE and RV-EF in the cohort? (2) Do the proportions of abnormal RV indices differ across the observed CKD stages? and (3) Are renal function and RV performance more closely related to pulmonary pressure than to CKD stage itself? This approach is relevant to settings in which advanced strain imaging or cardiac magnetic resonance may not be routinely available.

Accordingly, the primary objective was to characterize the distribution of conventional RV systolic abnormalities across CKD stages in this Iraqi cohort. Secondary objectives were to examine differences in eGFR according to TAPSE and RV-EF categories, assess the relationship of hemodialysis status to RV categories, and interpret the reported correlation pattern among eGFR, PASP, TAPSE, LV-EF, and RV-EF. We hypothesized that RV abnormalities would be common in advanced CKD but that CKD stage alone would not fully explain the observed right-heart phenotype.

2. MATERIALS AND METHODS

2.1 Study design and setting

This manuscript presents a stage-focused secondary analysis of a cross-sectional observational study performed at the coronary care unit echocardiography clinic of Baquba Teaching Hospital, Diyala, Iraq. The source study recruited patients with CKD between August 2025 and February 2026 and assessed right ventricular function using transthoracic echocardiography. No additional patients were recruited for the present manuscript, and no outcome data beyond those reported in the source dataset were invented or imputed.

The secondary-analysis framing was selected to provide a distinct clinical question centered on CKD stage and RV phenotype. Aggregate counts in the source tables were used to recalculate stage-specific percentages of abnormal TAPSE and abnormal RV-EF. Because patient-level raw data were not available in the supplied document, no multivariable regression, time-to-event analysis, or reclassification analysis was attempted. The reported continuous-variable means and correlation coefficients were retained as documented in the source study.

2.2 Study population

The analytic cohort comprised 100 adults diagnosed with CKD. Participants were recruited from nephrology and internal medicine clinics and referred for cardiac functional assessment. Eligible patients were 18 years of age or older and had CKD according to the source study criteria. Both men and women were eligible. Although the original inclusion framework allowed CKD stages 1–5, the reported analytic cohort contained participants in stages 2–5; no stage 1 participant appeared in the source results table.

The source study excluded patients with known congenital or clinically important valvular heart disease, previous pulmonary embolism, chronic lung disease or cor pulmonale, uncontrolled hypertension or diabetes with acute complications, temporary dialysis, and acute kidney injury. These criteria were intended to reduce major nonrenal causes of RV dysfunction and to create a cohort in which the cardiorenal-pulmonary relationship could be examined more directly.

2.3 Clinical and laboratory assessment

All participants underwent clinical history and physical examination with emphasis on dyspnea, fatigue, edema, cardiovascular history, and CKD-related manifestations. Blood pressure, heart rate, and body mass index were recorded in the source protocol. Laboratory testing included serum creatinine and urea, with estimated glomerular filtration rate derived from a creatinine-based equation documented in the study file. The equation shown in the source document corresponds to the 2021 CKD-EPI creatinine equation form. For the present manuscript, eGFR values and CKD categories are reported exactly as available from the supplied study tables, subject to the data-verification note at the end of this draft.

CKD stage categories used in the source protocol were: stage 1, eGFR ≥90 mL/min/1.73 m²; stage 2, 60–89; stage 3, 30–59; stage 4, 15–29; and stage 5, <15 mL/min/1.73 m² or dialysis. These categories are consistent with the broad G-stage framework used in KDIGO classification [1], although current KDIGO practice also integrates albuminuria categories for prognosis. Albuminuria data were not reported in the supplied study document and therefore were not introduced into this analysis.

2.4 Echocardiographic assessment

Transthoracic echocardiography was performed with a Vivid GE E9 system using a 2.5–3.5 MHz transducer. Standard M-mode, two-dimensional, and Doppler views were acquired. TAPSE was measured in the apical four-chamber view with an M-mode cursor positioned at the lateral tricuspid annulus, reflecting longitudinal RV systolic excursion. PASP was estimated from the tricuspid regurgitation jet using the modified Bernoulli relationship described in routine echocardiographic practice. The source protocol also recorded RV-EF using a Simpson-based approach and LV-EF by M-mode assessment.

The supplied study file classifies TAPSE and RV-EF as normal or abnormal but does not state the operative dichotomization threshold in the Methods section. Elsewhere in the source narrative, a TAPSE value of approximately 14 mm is discussed as abnormal and an RV-EF >45% is described as normal. Because exact laboratory cutoffs must be verified against the original echocardiography worksheets before journal submission, the present manuscript retains the source categories without asserting a new cutoff. This is particularly important because contemporary right-heart guidance emphasizes multiparametric assessment and provides updated reference values [2].

2.5 Outcomes and analytical focus

The primary outcome was the stage-specific proportion of participants categorized as having abnormal TAPSE. A co-primary descriptive outcome was the stage-specific proportion with abnormal RV-EF. Secondary outcomes included overall prevalence of abnormal TAPSE and RV-EF, differences in mean eGFR according to

RV category, hemodialysis subgroup distributions, and the reported correlation coefficients among eGFR, PASP, TAPSE, LV-EF, and RV-EF.

To ensure that the secondary manuscript addressed a clinically distinct question, emphasis was placed on the absence or presence of a stage gradient and on the potential role of pulmonary vascular load. Findings were interpreted as associative rather than causal because of the cross-sectional design.

2.6 Statistical analysis

The source study analyzed data using SPSS version 21.0. Quantitative variables were summarized as mean ± standard deviation, and categorical variables as frequency and percentage. The source analysis used chi-square tests for categorical comparisons, t tests for two-group numerical comparisons, analysis of variance for comparisons involving more than two numerical groups, and correlation coefficients (r) for relationships between numerical variables. A two-sided p value <0.05 was considered statistically significant.

For this stage-focused manuscript, the stage-by-TAPSE and stage-by-RV-EF contingency tables were reconstructed directly from the aggregate counts printed in the source results. Pearson chi-square tests were recomputed from those counts, yielding $p=0.339$ for TAPSE category across stages and $p=0.576$ for RV-EF category across stages. These recalculations are compatible with the source report of $p>0.05$ and provide exact values for the secondary stage-focused presentation. No other statistics were recalculated beyond simple percentages and these two chi-square tests.

2.7 Ethical considerations

The source study protocol was reviewed and approved by the Scientific Committee of the Department of Internal Medicine, College of Medicine, University of Diyala (approval code 2025OAA933; 9 August 2025). Informed consent was obtained from all participants before inclusion. The present manuscript uses only data from that approved cohort. Any journal submission should also disclose prior presentations or publications arising from the same dataset, if applicable, in accordance with the target journal's publication-ethics requirements.

3. RESULTS

3.1 Cohort characteristics

The study included 100 participants with CKD. The age distribution was weighted toward older adults: 53 participants (53%) were older than 60 years, 19% were 51–60 years, 17% were 41–50 years, 7% were 31–40 years, and 4% were 30 years or younger. Fifty-eight participants (58%) were receiving hemodialysis at the time of assessment. The mean eGFR for the cohort was reported as 19.29 ± 7.67 mL/min/1.73 m², indicating that the sample was predominantly composed of advanced CKD.

The distribution of CKD stage was markedly skewed toward stages 4 and 5. One participant was categorized as stage 2, 18 as stage 3, 33 as stage 4, and 48 as stage 5. Thus, 81% of the cohort had stage 4 or 5 CKD. This composition is important when interpreting stage comparisons because stage 2 contributed only one observation and stage 1 was absent from the reported analytic table.

3.2 Prevalence of RV systolic abnormalities

Abnormal RV-EF was reported in 51 of 100 participants, indicating that approximately one-half of the cohort met the source study's criterion for impaired global RV systolic performance. Abnormal TAPSE was present in 35 participants, while 65 had a normal TAPSE category. The difference in prevalence between the two metrics illustrates that conventional RV parameters do not identify identical patient subsets. TAPSE reflects longitudinal tricuspid annular excursion and is influenced by loading conditions, while an ejection-fraction measure attempts to capture a more global volumetric change. Without patient-level cross-tabulation of TAPSE and RV-EF categories, the degree of overlap between these abnormal groups cannot be determined from the supplied document.

The high frequency of an abnormal RV-EF category is clinically notable in a cohort dominated by CKD stages 4–5. However, prevalence alone does not establish that renal stage is the primary determinant. The stage-stratified analysis below addresses that question directly.

3.3 Stage-wise distribution of abnormal TAPSE and RV-EF

Among the 18 participants with stage 3 CKD, 8 had abnormal TAPSE (44.4%). In stage 4, 14 of 33 participants had abnormal TAPSE (42.4%). In stage 5, 13 of 48 participants had abnormal TAPSE (27.1%). The single stage 2 participant had normal TAPSE. The stage distribution was not statistically significant in the reconstructed contingency table (chi-square $p=0.339$). Importantly, the pattern was not monotonic: the highest observed abnormal TAPSE proportions occurred in stages 3 and 4 rather than stage 5.

For RV-EF, 9 of 18 stage 3 participants (50.0%), 15 of 33 stage 4 participants (45.5%), and 27 of 48 stage 5 participants (56.3%) were categorized as abnormal. The single stage 2 participant had normal RV-EF. Again, there was no significant association between CKD stage and RV-EF category (chi-square $p=0.576$). The absolute percentage in stage 5 was somewhat higher than stages 3–4, but the differences were modest and statistically nonsignificant.

Table 1. Baseline demographic and echocardiographic profile of the cohort

Characteristic	n	%
Age ≤20 years	2	2.0
Age 21–30 years	2	2.0
Age 31–40 years	7	7.0
Age 41–50 years	17	17.0
Age 51–60 years	19	19.0
Age >60 years	53	53.0
On hemodialysis	58	58.0
Not on hemodialysis	42	42.0
Abnormal LV-EF	77	77.0
Abnormal RV-EF	51	51.0
Abnormal TAPSE	35	35.0
CKD stage 2	1	1.0
CKD stage 3	18	18.0
CKD stage 4	33	33.0
CKD stage 5	48	48.0

Table 2. Stage-wise distribution of abnormal right ventricular indices

CKD stage	Total n	Abnormal TAPSE, n (%)	Abnormal RV-EF, n (%)
2	1	0 (0.0%)	0 (0.0%)
3	18	8 (44.4%)	9 (50.0%)
4	33	14 (42.4%)	15 (45.5%)
5	48	13 (27.1%)	27 (56.3%)

Stage-by-TAPSE chi-square p=0.339; stage-by-RV-EF chi-square p=0.576 (recomputed from reported aggregate counts).

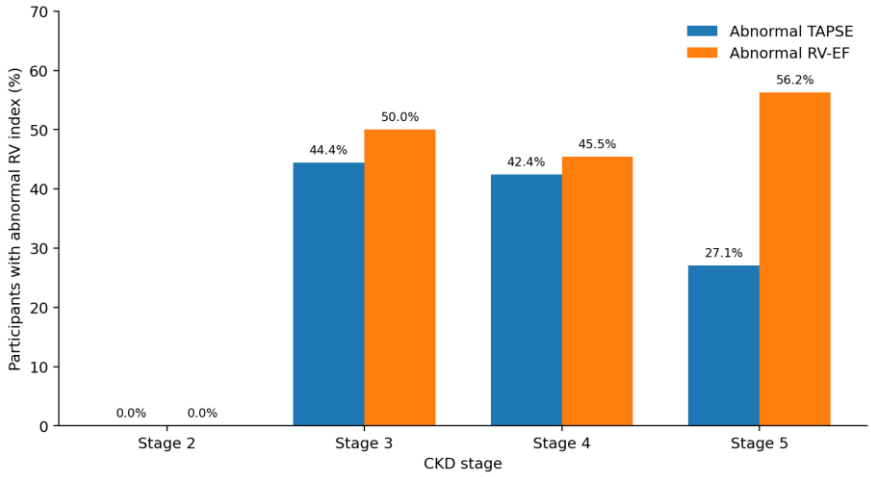

Figure 1. Proportion of participants with abnormal TAPSE and abnormal RV-EF across the observed CKD stages. Percentages were recalculated from the aggregate counts reported in the source tables.

3.4 eGFR according to RV category

Mean eGFR was 17.97 ± 8.883 mL/min/1.73 m² among participants with normal TAPSE and 21.73 ± 9.816 mL/min/1.73 m² among those with abnormal TAPSE. The difference was reported as not statistically significant (p>0.05). Thus, lower eGFR was not directly associated with the abnormal TAPSE category in this cross-sectional cohort.

Similarly, mean eGFR was 20.56 ± 12.107 mL/min/1.73 m² in participants with normal RV-EF and 18.06 ± 11.219 mL/min/1.73 m² in those with abnormal RV-EF, with no statistically significant difference (p>0.05). The direction of the mean difference was more intuitive for RV-EF than for TAPSE, but the overlap was substantial and statistical significance was not reached. These findings reinforce the stage analysis by showing that kidney filtration level alone did not separate participants with normal from abnormal conventional RV systolic indices.

Table 3. Mean eGFR according to right ventricular functional category

Group	n	eGFR, mean ± SD (mL/min/1.73 m²)	p value
TAPSE: normal	65	17.97 ± 8.883	>0.05
TAPSE: abnormal	35	21.73 ± 9.816	
RV-EF: normal	49	20.56 ± 12.107	>0.05
RV-EF: abnormal	51	18.06 ± 11.219	

3.5 Hemodialysis status and RV categories

Of the 58 participants on hemodialysis, 18 (31.0%) had abnormal TAPSE and 40 (69.0%) had normal TAPSE. Among the 42 participants not on hemodialysis, 17 (40.5%) had abnormal TAPSE and 25 (59.5%) had normal TAPSE. The source study reported no significant association between hemodialysis status and TAPSE category. This finding suggests that dialysis exposure alone did not identify the subgroup with impaired longitudinal RV motion in this sample.

For RV-EF, 33 of 58 hemodialysis participants (56.9%) had abnormal RV-EF compared with 18 of 42 non-hemodialysis participants (42.9%). Although the numerical difference was larger than for TAPSE, the source analysis again reported no statistically significant association. Because information on dialysis vintage, ultrafiltration volume, access type, access flow, timing of echocardiography relative to dialysis, and dry weight was not available in the supplied dataset, a more granular assessment of dialysis-related RV loading was not possible.

3.6 Correlation pattern: renal function, pulmonary pressure, and ventricular performance

The most clinically informative correlations involved pulmonary artery systolic pressure. eGFR correlated inversely with PASP (r=-0.207, p=0.039), indicating that lower renal filtration was associated with higher estimated pulmonary pressure. Although the magnitude of this correlation was modest, it reached statistical significance and is compatible with a model in which advancing renal impairment is accompanied by greater pulmonary vascular or left-heart loading stress.

TAPSE also correlated inversely with PASP (r=-0.256, p=0.010). In physiologic terms, higher pulmonary pressure was associated with lower tricuspid annular systolic excursion, supporting the concept that RV longitudinal performance is sensitive to afterload. By contrast, eGFR was not significantly correlated with TAPSE in the source matrix (r=0.154, p=0.126), further suggesting that pulmonary hemodynamic load may be a more proximate correlate of RV longitudinal function than kidney filtration level alone.

LV-EF correlated positively with RV-EF (r=0.367, p<0.001), indicating moderate ventricular interdependence within the cohort. This association may reflect shared myocardial disease, loading conditions, or the mechanical interaction of the two ventricles within the pericardium. The source matrix also reported an inverse correlation between TAPSE and RV-EF; because this direction is physiologically unexpected if both are coded as continuous measures of better systolic function, the value should be checked against the original SPSS coding before it is interpreted or emphasized in a submitted paper.

Table 4. Selected reported correlations relevant to the stage-focused analysis

Variables	r	p value	Interpretive summary
eGFR vs PASP	-0.207	0.039	Lower eGFR associated with higher PASP
TAPSE vs PASP	-0.256	0.010	Higher PASP associated with lower TAPSE
LV-EF vs RV-EF	0.367	<0.001	Positive biventricular functional association
eGFR vs TAPSE	0.154	0.126	Not statistically significant

4. DISCUSSION

4.1 Principal findings

This stage-focused analysis yielded four principal observations. First, conventional RV systolic abnormalities were common: 51% of participants had an abnormal RV-EF category and 35% had an abnormal TAPSE category. Second, neither TAPSE nor RV-EF showed a statistically significant stage-by-stage association across the observed CKD stages. Third, eGFR did not significantly distinguish participants with normal versus abnormal TAPSE or RV-EF. Fourth, the strongest mechanistically coherent relationships involved pulmonary pressure: PASP correlated inversely with both eGFR

and TAPSE, while LV-EF correlated positively with RV-EF. Taken together, these findings argue against a simple model in which CKD stage alone determines RV systolic dysfunction and instead support a more complex cardiorenal-pulmonary interaction.

This interpretation is clinically important. In everyday practice, CKD stage is often used as a shorthand for disease burden, but cardiovascular remodeling reflects cumulative exposure to multiple processes that are only partly represented by eGFR. Volume status, blood pressure, anemia, arteriovenous access flow, left-sided filling pressure, pulmonary vascular resistance, diabetes, and duration of kidney disease may vary substantially among patients within the same CKD stage. A stage 5 patient receiving regular dialysis and careful volume management may have a different right-heart load than a stage 4 patient with persistent congestion. The nonmonotonic TAPSE pattern in the present data is therefore plausible, although it must be interpreted cautiously because of the cross-sectional design and uneven stage sizes.

4.2 RV dysfunction across CKD stages

The absence of a statistically significant stage gradient does not imply that the RV is unaffected by CKD. Earlier echocardiographic work has shown detectable RV abnormalities even in moderate CKD. Floccari et al. evaluated stage III CKD and reported right-heart changes that supported a role for echocardiography before kidney failure develops [10]. In patients with more advanced disease, multiple hemodialysis studies have documented RV dysfunction using TAPSE, tissue Doppler, myocardial performance indices, or strain-based measures [7–9]. These studies collectively indicate that RV involvement can occur throughout the CKD spectrum but is influenced by the method used to define dysfunction.

The stage-specific abnormal TAPSE proportions in this cohort were 44.4% in stage 3, 42.4% in stage 4, and 27.1% in stage 5. A purely stage-driven hypothesis would predict a higher stage 5 prevalence. Several explanations may account for the observed pattern. First, TAPSE is load dependent; measurement timing relative to dialysis could alter values by changing preload and pulmonary pressures. Second, stage 5 encompasses patients with potentially different dialysis status, vascular access, and volume control. Third, the stage 5 group was larger and may have contained more heterogeneous clinical phenotypes. Fourth, the precise TAPSE abnormality threshold used in the source analysis must be verified, and any threshold near 14 mm will identify a more severely impaired subgroup than contemporary cutoffs used in some guidelines and studies. Finally, the stage 2 group had only one participant, limiting inference at the mild end of the spectrum.

The RV-EF pattern differed from TAPSE: abnormal RV-EF was present in 50.0% of stage 3, 45.5% of stage 4, and 56.3% of stage 5 participants. This suggests that a global systolic measure may detect a different aspect of RV impairment than longitudinal annular motion. However, the method used in the source study to derive RV-EF should be reviewed carefully before submission because two-dimensional RV volumetry is technically challenging due to the chamber's complex geometry. Contemporary ASE recommendations favor a multiparametric approach that integrates TAPSE, tissue Doppler S', fractional area change, three-dimensional RV-EF when feasible, strain, chamber size, and hemodynamic context [2]. The present data therefore support the clinical concept of multimodal assessment even though the study itself relied on conventional measurements.

4.3 Renal function and the pulmonary circulation

The inverse association between eGFR and PASP was one of the clearest findings in the source correlation matrix. Although modest in magnitude (r=-0.207), it provides a plausible link between declining kidney function and the RV because PASP is an important component of RV afterload. Pulmonary hypertension is well described in CKD and is particularly common in dialysis populations. Zhao et al. reported a high prevalence of pulmonary hypertension among hemodialysis patients and demonstrated its association with RV dysfunction [9]. Edmonston et al., using invasive hemodynamic data, further showed that pulmonary hypertension subtypes are common in CKD and carry prognostic implications, emphasizing that pulmonary vascular and postcapillary mechanisms may coexist [15].

Several CKD-specific mechanisms may raise pulmonary pressure. Chronic volume expansion can elevate left atrial and pulmonary venous pressures. Arteriovenous access can increase cardiac output and pulmonary blood flow, especially when access flow is high. Endothelial dysfunction, vascular calcification, anemia, sleep-disordered breathing, and uremic milieu may contribute additional pulmonary vascular stress. Sathiavageesan et al. reported that vascular access location influenced pulmonary arterial pressure in hemodialysis patients, supporting the relevance of access-related hemodynamics [12]. Yilmaz et al. found that high access flow was independently associated with impaired RV function, plausibly through increased preload [8]. These observations provide a mechanistic context for the eGFR-PASP relationship in the present cohort.

The inverse relationship between PASP and TAPSE (r=-0.256) strengthens this interpretation. The RV is particularly sensitive to afterload; as pulmonary pressure rises, longitudinal systolic excursion may fall. This relationship also suggests why CKD stage alone may be an incomplete predictor of RV performance. Two patients with the same eGFR can have markedly different pulmonary pressures depending on volume status, LV diastolic function, vascular access, lung disease, anemia, and treatment. A clinically useful approach may therefore be to combine kidney stage with a targeted right-heart and pulmonary-pressure assessment rather than assuming that RV risk tracks directly with eGFR.

4.4 Hemodialysis and right-heart loading

Hemodialysis introduces acute and chronic hemodynamic changes that can affect RV function in opposite directions. Ultrafiltration reduces circulating volume and may decrease pulmonary pressure and RV preload during or after a treatment session. Conversely, the creation and long-term presence of an arteriovenous fistula can increase venous return and cardiac

output, potentially raising RV preload and pulmonary flow. The net echocardiographic effect therefore depends on timing, dry-weight achievement, access flow, dialysis vintage, myocardial reserve, and pulmonary vascular response.

In the present cohort, 58% were on hemodialysis. Abnormal TAPSE occurred in 31.0% of dialysis participants versus 40.5% of non-dialysis participants, while abnormal RV-EF occurred in 56.9% versus 42.9%, respectively. Neither comparison was reported as significant. The divergent directions of TAPSE and RV-EF again indicate that no single conventional measurement fully captures dialysis-related RV physiology. Tanasa et al. prospectively evaluated RV function around initiation of hemodialysis and demonstrated that RV indices can change with dialysis-related loading conditions [6]. Akyüz et al. similarly assessed RV systolic function before and after hemodialysis using multiple echocardiographic modalities and reported measurable acute changes [14].

Access flow may be particularly important. Yilmaz et al. found that patients with impaired TAPSE had higher arteriovenous fistula flow and that high access flow independently predicted impaired RV function [8]. López-Quijano et al. identified additional clinical and echocardiographic determinants of RV systolic dysfunction in hemodialysis [7]. Because the source dataset did not provide access flow, access location, time from dialysis, or ultrafiltration volume, the present study cannot distinguish the effects of dialysis treatment from the effects of kidney failure itself. This is a major target for future prospective work.

4.5 Ventricular interdependence

The positive correlation between LV-EF and RV-EF ($r=0.367$, $p<0.001$) suggests that the two ventricles were functionally linked in this cohort. Ventricular interdependence arises from the shared interventricular septum, common myocardial fibers, pericardial constraint, and common loading conditions. CKD-related myocardial fibrosis, ischemic disease, hypertension, and volume overload can therefore affect both ventricles simultaneously. This relationship also argues against interpreting RV abnormalities in isolation from left-sided function and filling pressures.

Dini et al. previously demonstrated an association between RV dysfunction and CKD in patients with chronic systolic heart failure and showed that RV dysfunction carried prognostic significance [11]. Although the present cohort was not defined as a heart-failure cohort, the LV-RV association points toward a shared cardiac substrate. Importantly, the source results text occasionally uses the phrase “patients with heart failure” despite the Methods defining enrollment by CKD rather than heart failure. The present manuscript avoids assuming a heart-failure diagnosis and treats the cohort as CKD patients; this wording should be verified against the original clinical records before submission.

4.6 Comparison with contemporary evidence

Recent studies have moved beyond binary TAPSE abnormality toward integrated RV-pulmonary arterial coupling. Wang et al. examined the TAPSE/PASP ratio in maintenance hemodialysis and found that impaired coupling was associated with increased cardiovascular risk [4]. This is directly relevant to the present observation that PASP correlated with both eGFR and TAPSE. Even without a calculated TAPSE/PASP ratio in the supplied dataset, the two component relationships point toward the same physiologic axis: the ability of the RV to maintain systolic shortening in the face of pulmonary load.

Nobayashi et al. reported that RV dysfunction defined by reduced TAPSE was independently associated with mortality among patients on maintenance hemodialysis [5]. Their findings underline why a non-significant relationship between CKD stage and TAPSE should not lead clinicians to dismiss RV assessment. Stage may fail to discriminate risk even when the RV measure itself has prognostic value. Similarly, Abuelfotoh et al. recently demonstrated the usefulness of conventional and tissue Doppler echocardiography for identifying RV systolic dysfunction in chronic renal disease [3]. These contemporary data reinforce the value of evaluating the right heart as a distinct target in CKD.

Current ASE guidance also supports a comprehensive approach to the right heart and pulmonary hypertension [2]. The guideline emphasizes systematic measurement of RV size, systolic function, right atrial parameters, tricuspid regurgitation velocity, estimated pulmonary pressures, and signs of RV-pulmonary vascular interaction. For resource-limited environments, conventional indices such as TAPSE and PASP remain attractive because they are widely available and quickly obtained. Their limitations, however, require interpretation in conjunction with clinical volume status and other echo parameters. The present study is therefore best viewed as a pragmatic conventional-echocardiography dataset that identifies a signal worthy of more advanced prospective evaluation.

4.7 Clinical implications

The practical message is not that every CKD patient requires advanced cardiac imaging. Rather, the findings support deliberate right-heart evaluation when echocardiography is already being performed, especially in advanced CKD, hemodialysis, unexplained dyspnea, recurrent volume overload, elevated estimated pulmonary pressure, or evidence of biventricular dysfunction. A normal LV-EF should not automatically end the cardiac assessment if symptoms or pulmonary pressure suggest right-heart stress.

The inverse PASP-TAPSE relationship also highlights the importance of identifying potentially modifiable contributors to RV afterload and preload. These may include volume excess, uncontrolled systemic blood pressure, anemia, sleep-disordered breathing, excessive arteriovenous access flow, and left-sided filling pressure. The study does not test whether treating these factors improves RV function; therefore, the data should not be used to recommend a specific therapy. They do, however, support a multidisciplinary nephrology-cardiology approach when right-heart abnormalities are detected.

From a risk-stratification perspective, a stage-only model may be insufficient. Two participants in the same CKD stage can have different pulmonary pressures and RV reserve. Incorporating PASP and RV systolic measurements may help identify patients who deserve closer follow-up, especially if future studies confirm prognostic value in Iraqi and other regional populations.

4.8 Strengths and limitations

This analysis has several strengths. It addresses an underemphasized component of CKD cardiovascular disease using routinely available echocardiographic measures. The cohort includes a substantial proportion of advanced CKD and dialysis patients, a population in whom right-heart loading abnormalities are clinically plausible. The stage-focused reanalysis also avoids overstating a renal-stage gradient and instead presents the actual non-significant stage comparisons. Finally, the observed eGFR-PASP and PASP-TAPSE correlations provide a coherent physiologic signal that can guide future hypothesis-driven studies.

The limitations are substantial and should be explicit. First, the study is cross-sectional, so temporal or causal relationships cannot be established. Second, the sample size is modest and stage distribution is highly uneven, with only one stage 2 participant and no stage 1 participants in the reported analytic table. Third, key confounders were not available in the supplied results, including hemoglobin, diabetes status, blood pressure level, volume markers, dialysis vintage, ultrafiltration volume, vascular access location and flow, and detailed LV diastolic indices. Fourth, timing of echocardiography relative to dialysis was not specified, even though RV preload and PASP can change acutely with ultrafiltration.

Fifth, the exact operational cutoffs used to classify TAPSE and RV-EF as normal or abnormal need to be confirmed from the original echocardiography records. Sixth, the use of a Simpson-based method for RV-EF should be reviewed against the original acquisition protocol, because RV geometry complicates two-dimensional volumetric estimation. Seventh, no strain imaging, three-dimensional RV-EF, cardiac magnetic resonance, or invasive pulmonary hemodynamics were available. Eighth, the source table contains at least one value requiring data verification: the reported mean eGFR for the single stage 2 participant is 31.00 mL/min/1.73 m², which is inconsistent with the stated stage 2 eGFR range of 60–89. This discrepancy should be resolved before submission and the relevant table corrected from the raw dataset.

Finally, if another article has already been published from the same 100-patient cohort, the present secondary analysis must cite and disclose that publication, explain the distinct research question, and comply with the target journal's rules on secondary analyses and overlapping datasets. Rewording alone is not sufficient to create a distinct publication; the scientific question, analysis, and interpretation must be transparently differentiated.

5. CONCLUSIONS

Right ventricular systolic abnormalities were frequent in this cohort of adults with CKD, with abnormal RV-EF reported in approximately half and abnormal TAPSE in approximately one-third. Despite the advanced renal disease burden, neither RV-EF nor TAPSE showed a statistically significant monotonic association with CKD stage, and mean eGFR did not differ significantly according to these RV categories.

The most coherent cardiorenal signal involved pulmonary hemodynamics: lower eGFR was associated with higher PASP, and higher PASP was associated with lower TAPSE. These findings suggest that pulmonary vascular and volume-related load may be an important intermediate pathway linking renal impairment to RV dysfunction. Conventional CKD stage alone may therefore be insufficient to characterize right-heart risk.

Prospective studies should incorporate standardized multiparametric RV assessment, dialysis timing, objective volume measures, access flow, anemia and comorbidity profiles, and longitudinal cardiovascular outcomes. In clinical settings where advanced imaging is unavailable, systematic use of TAPSE and estimated pulmonary pressure as part of a complete echocardiographic examination may still provide valuable information when interpreted within the broader hemodynamic context.

Declarations

Ethics approval and consent to participate: Approved by the Scientific Committee of the Department of Internal Medicine, College of Medicine, University of Diyala (Code 2025OAA933; 9 August 2025). Written informed consent was obtained from participants according to the source study.

Consent for publication: Not applicable to individually identifiable material in this manuscript.

Availability of data and materials: The underlying patient-level dataset is not embedded in this manuscript. Data availability should be stated according to the authors' institutional approval and the target journal's policy.

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