

Prognostic Value of Right Ventricular Function Assessment by Echocardiography in End-Stage Renal Disease Patients on Maintenance Hemodialysis in Myanmar: A Prospective Observational Cohort Study

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ABSTRACT

Background

Cardiovascular disease is the leading cause of mortality among End-Stage Renal Disease (ESRD) patients in Southeast Asia. While left ventricular function is routinely assessed, the prognostic significance of right ventricular (RV) dysfunction remains critically understudied in Myanmar.

Objectives

To determine the prevalence of RV dysfunction in maintenance hemodialysis patients and evaluate its independent association with a composite risk of one-year all-cause mortality and cardiovascular hospitalization.

Methods

This prospective observational cohort study enrolled 200 adult ESRD patients. To minimize volume loading artifacts, comprehensive post-dialysis transthoracic echocardiography was performed. RV function was assessed using Tricuspid Annular Plane Systolic Excursion (TAPSE), RV Fractional Area Change, and Tissue Doppler Systolic Velocity. The primary composite endpoint evaluated 12-month all-cause mortality and decompensated heart failure hospitalization.

Results

The cohort (mean age 53.1±12.4 years; 62% male) exhibited a 41.3% prevalence of RV dysfunction (TAPSE < 17 mm). Over a 12-month median follow-up, the primary endpoint occurred in 26.7% of patients. Non-survivors presented with significantly lower TAPSE (13.8±3.1 vs. 18.9±4.2 mm, $p<0.001$) and serum albumin. Kaplan-Meier analysis indicated significantly reduced event-free survival for patients with RV dysfunction ($p<0.001$). Following multivariable Cox regression adjustment for age, LVEF, dialysis vintage, and nutritional status, TAPSE < 17 mm remained a strong independent predictor of the composite outcome (HR: 2.38; 95% CI: 1.55-3.72; $p<0.001$).

Conclusions

Despite overall mortality being lower than historical national averages, RV dysfunction was an independent predictor of adverse cardiovascular outcomes in this optimized hemodialysis cohort.

KEYWORDS: End-Stage Renal Disease, Right Ventricle, Echocardiography, Prognosis, Myanmar.

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INTRODUCTION

End-Stage Renal Disease (ESRD) represents a critical public health challenge throughout Southeast Asia. This burden is aggravated by the region's surge in non-communicable diseases [1]. Myanmar, specifically, faces a rising prevalence of Chronic Kidney Disease (CKD) fueled by a 'double burden': the simultaneous pressure of emerging lifestyle conditions—like hypertension and diabetes mellitus and persistent infectious risks [2]. While renal replacement therapy (RRT) has advanced locally, cardiovascular disease (CVD) remains the leading cause of mortality, responsible for more than half of all deaths among dialysis patients [3].

Traditional cardiovascular risk stratification in dialysis patients has focused predominantly on Left Ventricular (LV) parameters, specifically LV hypertrophy (LVH) and ejection fraction (LVEF) [4]. This exclusively 'LV-centric' view overlooks the unique susceptibility of the right heart within the hemodynamic stress of dialysis. The Right Ventricle (RV), often termed the "forgotten ventricle," is structurally distinct from the LV; it is a thin-walled, compliant chamber poorly adapted to acute changes in pressure or volume [5].

In the hemodialysis setting, the RV is subjected to a relentless 'double hit' of hemodynamic instability. This burden begins with chronic preload excess, driven by high-flow arteriovenous fistulas (AVF). This process can elevate the cardiac output by 15-20% and precipitate volume overload [6]. Simultaneously, the ventricle must overcome heightened afterload caused by pulmonary hypertension (PH) and the arterial stiffness inherent to CKD [5]. Recent data indicates this mechanical vulnerability is compounded by 'uremic cardiomyopathy.' In uremic cardiomyopathy, toxins like Indoxyl Sulfate cause myocardial fibrosis and capillary rarefaction. These biochemical insults trigger oxidative stress and inflammation, degrading cardiomyocyte function independently of the hemodynamic load [7, 8].

In developing regions like Myanmar, these physiological challenges are frequently aggravated by the 'Malnutrition-Inflammation Complex Syndrome' (MICS) [9]. This syndrome is pervasive in resource-limited settings and is a known cause of adverse myocardial remodeling and reduced survival [10]. Despite these compounding risks, the specific prognostic role of the RV remains largely unexplored in local literature. This data gap is most pronounced within restricted-access healthcare systems.

This study aims to bridge this knowledge gap by determining the prevalence of RV dysfunction. We assessed the independent prognostic value of RV dysfunction in predicting a composite of one-year mortality and cardiovascular hospitalization among ESRD patients in this specific population.

MATERIALS & METHODS

Study Design and Population

This prospective observational cohort study was conducted at the Department of Nephrology and Cardiology at 700 Bedded Military Hospital, Pyin Oo Lwin, Myanmar. The sample size was calculated based on the anticipated incidence of the primary composite outcome (all-cause mortality or cardiovascular hospitalization) among patients with end-stage renal disease on maintenance hemodialysis. Assuming an event rate of 25%, a hazard ratio of 2.0 for patients with right ventricular dysfunction, a two-sided α level of 0.05, and 80% statistical power, a minimum sample size of 182 participants was required. To compensate for potential loss to follow-up and incomplete data, 200 consecutive eligible patients were enrolled. We recruited 200 consecutive adult patients (age ≥ 18 years) undergoing maintenance hemodialysis (dialysis vintage ≥ 3 months) between January 2024 and June 2025. Exclusion criteria included congenital heart disease, primary valvular heart disease, and acute coronary syndrome within 3 months. Patients were followed for a median duration of 12 months after initial enrollment to track the primary composite endpoint.

Ethical Considerations

The study protocol was approved by the Institutional Review Board and written informed consent was obtained from all participants [11].

Clinical and Laboratory Assessment

A focused nutritional assessment was performed using the Malnutrition-Inflammation Score (MIS) [12]. Routine laboratory parameters were measured pre-dialysis, including hemoglobin, serum albumin, creatinine, calcium and phosphate.

Echocardiography Protocol

Transthoracic echocardiography was performed post-dialysis to minimize volume artifacts. RV function was assessed using TAPSE (Dysfunction < 17 mm), RV S' (Dysfunction < 9.5 cm/s), and RVFAC (Dysfunction $< 35\%$). Pulmonary Hypertension (PH) was defined as PASP > 35 mmHg [13, 14].

Statistical Analysis

Continuous variables are presented as mean $\pm$ SD. Comparisons between groups were made using the independent samples t-test. Survival curves were generated using the Kaplan-Meier method. Multivariable Cox proportional hazards regression models identified independent predictors of the primary endpoint.

RESULTS

Baseline Characteristics

The study cohort comprised 200 patients undergoing maintenance hemodialysis, predominantly middle-aged

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males (62%), with hypertension (80%) identified as the primary etiology of ESRD in the majority of cases (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	Total Cohort (N=200)	RV dysfunction (n=83)	Preserved RV (n=117)	P-value
Age (years)	53.1 ± 12.4	55.2 ± 11.8	51.6 ± 12.7	0.09
Male Sex	124 (62.0%)	51 (61.4%)	73 (62.4%)	0.88
Dialysis Vintage (months)*	20.0 (9–45)	28.5 (12–50)	16.0 (8–38)	0.02
Hypertension	160 (80.0%)	70 (84.3%)	90 (76.9%)	0.32
Diabetes Mellitus	71 (35.5%)	38 (45.8%)	33 (28.2%)	0.03
Chr. Glomerulonephritis	24 (12.0%)	7 (8.4%)	17 (14.5%)	0.21
AV Fistula	140 (70.0%)	60 (72.3%)	80 (68.4%)	0.56
Catheter (Perm/Temp)	60 (30.0%)	23 (27.7%)	37 (31.6%)	-
MIS Score (0–30)	6.2 ± 2.9	8.1 ± 3.2	4.9 ± 2.1	<0.01

Dialysis Vintage is presented as Median (IQR). P-values calculated using Independent Samples t-test (t), Chi-square test (χ^2), or Mann-Whitney U test (Z).

After grouping patients according to RV function, clear differences in clinical characteristics were observed. Patients with RV dysfunction had a significantly longer dialysis vintage compared to those with preserved function, suggesting a cumulative effect of uremic exposure and hemodynamic stress over time. Additionally, the prevalence of Diabetes Mellitus (35.5%) was notably higher in the RV dysfunction group, likely contributing to a more adverse cardiovascular substrate.

Nutritional assessment revealed a significant disparity; patients with RV dysfunction exhibited higher MIS, indicating a worse nutritional status. Interestingly, while the use of AVF was high across the entire cohort, there was no significant difference in vascular access type between the two groups, suggesting that the presence of an AVF alone does not dictate RV dysfunction, but rather the individual ventricular response to the volume load may be the differentiating factor.

Biochemical and Nutritional Profile

The biochemical analysis further corroborated the link between RV impairment and the MICS (Table 2). Patients with reduced TAPSE exhibited a distinct metabolic profile characterized by significantly lower serum albumin and hemoglobin levels compared to those with preserved RV

function. Furthermore, markers of mineral bone disease differed significantly; serum phosphate levels were elevated in the RV dysfunction group. This hyperphosphatemia, in conjunction with the observed cardiac dysfunction, supports the hypothesis that vascular calcification and uremic toxicity may contribute to increased right ventricular afterload and myocardial stiffness in this population.

Table 2: Biochemical and Nutritional Parameters Stratified by RV Function

Parameter	Total (N=200)	RV Dysfunction (TAPSE <17mm)	Preserved RV (TAPSE ≥17mm)	P-Value
Hemoglobin (g/dL)	9.1 ± 1.5	8.6 ± 1.2	9.5 ± 1.6	0.01
Serum Albumin (g/dL)	3.5 ± 0.6	3.2 ± 0.4	3.7 ± 0.5	<0.01
Serum Creatinine (mg/dL)	8.4 ± 2.2	7.8 ± 2.0	8.9 ± 2.3	0.03
Calcium (mg/dL)	8.8 ± 0.9	8.6 ± 1.1	9.0 ± 0.8	0.15
Phosphate (mg/dL)	5.4 ± 1.6	5.9 ± 1.8	5.1 ± 1.4	0.04
Ca x P Product	47.5 ± 14.2	50.7 ± 15.5	45.9 ± 12.8	0.06

Data are presented as Mean ± SD. Comparisons performed using Independent Samples t-test.

Echocardiographic Analysis

RV dysfunction was common in this cohort, affecting 41.5% of the study population (Table 3).

Table 3: Echocardiographic & Hemodynamic Parameters

Parameter	Total cohort (N=200)	RV dysfunction (n=83)	Preserved RV (n=117)	P-value
TAPSE (mm)	18.2±4.1	14.1±2.2	21.3±3.1	<0.001
RV S' (cm/s)	10.8±2.4	8.2±1.5	12.6±2.1	<0.001
RV FAC (%)	39±8	31±6	45±7	<0.001
PASP (mmHg)	38±12	46±14	31±9	<0.001
LVEF (%)	52±11	45±12	56±9	0.02
Left Atrial Vol. Index (ml/m ²)	34±10	39±11	30±8	0.01
Pulmonary Hypertension*	92 (46.0%)	56 (67.5%)	36 (30.8%)	<0.001

Data are presented as Mean ± SD for continuous variables and n (%) for categorical variables. P-values derived from Independent Samples t-test (t) or Chi-square test (χ^2).

*Pulmonary Hypertension defined as PASP > 35 mmHg.

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Hemodynamic assessment revealed a strong coupling between pulmonary pressures and right heart function; Pulmonary Artery Systolic Pressure (PASP) was significantly elevated in patients with RV dysfunction, with a markedly higher prevalence of pulmonary hypertension observed in this subgroup.

Prevalence of Structural Abnormalities

Structurally, the impact of pressure overload was evident across both ventricles (Table 4). While LVH was ubiquitous, the concentric pattern of remodeling was significantly more common in patients with RV dysfunction. Notably, while LVEF was lower in the RV dysfunction group, it remained relatively preserved in many patients who nonetheless had severe RV impairment. This "uncoupling" highlights that a preserved LVEF can mask significant right-sided heart failure. In addition, valvular abnormalities were more common in patients with RV dysfunction, exhibiting a significantly higher prevalence of moderate-to-severe tricuspid and mitral regurgitation.

Table 4: Associated Cardiac Structural Abnormalities

Echo Finding	Total prevalence (N=200)	RV dysfunction (n=83)	Preserved RV (n=117)	P-value
LVH (Any)	149 (74.5%)	72 (86.7%)	77 (65.8%)	<0.01
Concentric LVH	93 (46.5%)	51 (61.4%)	42 (35.9%)	<0.01
Eccentric LVH	56 (28.0%)	21 (25.3%)	35 (29.9%)	0.62
Diastolic Dysfunction				
Grade I (Impaired Relaxation)	60 (30.0%)	13 (15.7%)	47 (40.2%)	<0.01
Grade II/III (Pseudonormal)	73 (36.5%)	47 (56.6%)	26 (22.2%)	<0.001
Mod/Severe TR	51 (25.5%)	43 (51.8%)	8 (6.8%)	<0.001
Mod/Severe MR	33 (16.5%)	24 (28.9%)	9 (7.7%)	<0.001

Data are presented as n (%). Comparisons performed using Chi-square test (χ^2). TR: Tricuspid Regurgitation; MR: Mitral Regurgitation.

Survival Analysis and Prognosis

Over the 12-month follow-up period, the composite endpoint of all-cause mortality and heart failure hospitalization occurred in 26.5% of the cohort. A comparative analysis of survivors versus non-survivors demonstrated that RV systolic function (TAPSE) was a more potent discriminator of mortality than LVEF. Non-survivors were characterized by a triad of profound RV dysfunction, elevated pulmonary pressures, and severe

malnutrition (lower albumin and higher MIS scores) (Table 5).

Table 5: Comparison of Survivors vs. Non-Survivors at 12 Months

Parameter	Survivors (n=147)	Non-Survivors (n=53)	P-Value
Age (years)	52.4 $\pm$ 12.1	58.1 $\pm$ 11.5	0.06
TAPSE (mm)	18.9 $\pm$ 4.2	13.8 $\pm$ 3.1	<0.001
LVEF (%)	53.2 $\pm$ 10.5	47.5 $\pm$ 12.8	0.04
PASP (mmHg)	36 $\pm$ 11	48 $\pm$ 15	0.001
Serum Albumin (g/dL)	3.6 $\pm$ 0.5	3.1 $\pm$ 0.4	<0.001
MIS Score	5.8 $\pm$ 2.6	8.9 $\pm$ 3.4	<0.001
Hemoglobin (g/dL)	9.3 $\pm$ 1.4	8.2 $\pm$ 1.3	0.003

Data are presented as Mean $\pm$ SD. Differences analyzed using Independent Samples t-test.

Correlations

Correlation analysis reinforced these physiological links, showing that TAPSE was positively correlated with nutritional markers (albumin, hemoglobin) and negatively correlated with inflammatory scores and pulmonary pressures (Table 6).

Table 6: Pearson Correlation Coefficients of RV Function (TAPSE) with Clinical Parameters

Parameter	Correlation Coefficient (r)	P-Value
LVEF (%)	0.35	<0.01
Serum Albumin	0.42	<0.01
Hemoglobin	0.28	0.02
MIS Score	-0.48	<0.001
Dialysis Vintage	-0.22	0.06
PASP	-0.55	<0.001

Data represent the strength and direction of the linear relationship between TAPSE and various clinical parameters. The Pearson Correlation Coefficient (r) was used for analysis. The Test Statistic (t) represents the t-value calculated to determine the statistical significance of the correlation ($df = 198$). Abbreviations: LVEF, Left Ventricular Ejection Fraction; MIS, Malnutrition-Inflammation Score; PASP, Pulmonary Artery Systolic Pressure.

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Predictors of the Composite Endpoint

We constructed a multivariable Cox regression model to determine independent predictors. After adjusting for Age, LVEF, and Nutritional Status (MIS), TAPSE < 17 mm remained a potent independent predictor of the composite endpoint (Hazard Ratio: 2.38; 95% CI: 1.55-3.72; $p < 0.001$). The MIS score also remained a significant predictor (HR: 1.15 per point increase) (Figure 1).

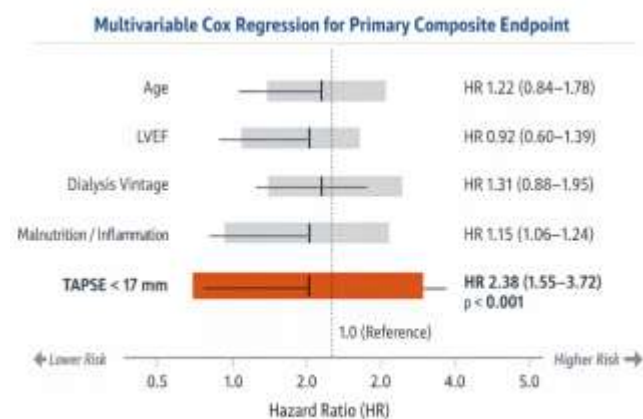


Figure 1: Multivariable Cox Regression Analysis for the Primary Composite Endpoint

This forest-style bar plot displays adjusted hazard ratios (HRs) with 95% confidence intervals (CIs) for variables included in the multivariable Cox proportional hazards model predicting the primary composite endpoint of all-cause mortality and heart failure hospitalization at 12 months. The vertical dashed line represents the reference value (HR = 1.0). Bars extending to the right indicate increased risk, whereas bars to the left indicate reduced risk. RV systolic dysfunction, defined as TAPSE < 17 mm, emerged as the strongest independent predictor of adverse outcomes (HR 2.38, 95% CI 1.55-3.72; $p < 0.001$) after adjustment for age, LVEF, dialysis vintage, and malnutrition-inflammation status.

DISCUSSION

We performed the first comprehensive assessment of RV prognostic utility in a restricted Myanmar dialysis facility. Our data reveals a high burden of RV dysfunction (41.5%). More importantly, TAPSE served as a potent, independent predictor of adverse one-year mortality and heart failure admission. This demonstrated stronger prognostic performance than traditional risk factors such as LVEF and vascular access type in predicting survival and hospitalization.

Pathophysiology of Hemodynamic Burden of AV Fistulas

The independent predictive value of TAPSE underscores a fundamental physiological reality: the Right Ventricle is ill-equipped for the hemodynamic demands of dialysis.

Structurally, the RV has thin walls and low elastance, creating a chamber that is highly compliant yet vulnerable to acute volume overload [15]. In ESRD, this vulnerability is tested by the AVF, which functions as a continuous left-to-right shunt. In our cohort, AVF utilization was notably high (70%). While these high-flow fistulas improve access longevity, they can elevate cardiac output by 15-20%, placing a disproportionate volume load on the right heart [16]. When the RV fails to accommodate this load (evidenced by reduced TAPSE), the resulting systemic venous congestion transmits backward to the gut and residual kidney tissue, worsening malnutrition [5]. Crucially, the fact that non-survivors exhibited lower TAPSE—despite having similar vascular access types to survivors (Table 5)—suggests that mortality is driven not by the presence of a fistula, but by the maladaptation of the right ventricle to the fistula-induced load.

Comparison with Recent Local Data

These results corroborate recent cross-sectional analyses from our center. Myo et al. (2025) previously established the high baseline burden of structural heart disease in this population, documenting LVH in 58.3% and diastolic dysfunction in 66.7% of patients [4]. Their study focused on the cross-sectional prevalence of abnormalities. But our current longitudinal study extends these findings by demonstrating the direct prognostic implication of echocardiographic parameters, specifically RV dysfunction, on mortality and hospitalization.

Furthermore, the high rate of arrhythmias was observed by Oo et al. (2025), such as premature atrial and ventricular contractions. This aligns with our observation of structural remodeling. This link suggests that the RV dysfunction serves as a trigger for electrical instability [17]. The consistent observation of high prevalence rates of cardiac remodeling across both studies underscores the urgent need for routine echocardiographic screening in this population.

The Uremic-Inflammatory Axis

Our data showed a correlation between RV dysfunction and higher serum phosphate levels (Table 2). This finding supports the role of uremic cardiomyopathy, a process driven not just by hemodynamics but by toxicity [8]. Central to this process are protein-bound uremic toxins like Indoxyl Sulfate, which are notoriously difficult to clear via standard dialysis. These toxins exert a direct pro-fibrotic effect on the myocardium. They also promote oxidative stress in the RV, which has lower antioxidant reserves compared to the Left Ventricle [7]. Furthermore, the hyperphosphatemia noted in our RV dysfunction group likely accelerates vascular calcification. This metabolic derangement increases pulmonary vascular stiffness, effectively raising RV afterload and perpetuating a vicious cycle of ventricular failure [18, 19].

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The Malnutrition-Inflammation Link

Our analysis reveals a robust triad of RV dysfunction, hypoalbuminemia, and elevated Malnutrition-Inflammation Scores (Tables 2 & 6). This strongly corroborates the presence of 'Malnutrition-Inflammation-Atherosclerosis' (MIA) syndrome in this cohort. This relationship is not merely associative but causative. Systemic inflammation, a hallmark of uremia, drives myocardial fibrosis and capillary rarefaction, thereby increasing RV stiffness [9]. This creates a vicious cycle: RV failure causes gut edema/congestion, leading to poor nutrient absorption and anorexia, which worsens malnutrition and inflammation, further damaging the heart. The clinical weight of this cycle is confirmed by our multivariate model, where the MIS score emerged as a significant independent predictor (HR 1.15). This confirms that nutritional status is as vital as cardiac mechanics in predicting survival [5].

Limitations

This study has several limitations. It was conducted at a single center with a relatively modest sample size, which may limit the generalizability of the findings. Right ventricular function was assessed using conventional echocardiographic parameters without advanced imaging techniques such as right ventricular strain or cardiac magnetic resonance imaging. In addition, echocardiography was performed only at baseline, and residual confounding from unmeasured clinical and dialysis-related factors cannot be completely excluded. Larger multicenter studies with longer follow-up and advanced imaging are needed to validate these findings.

Clinical Implications

In resource-limited settings where Cardiac MRI is unavailable, our results validate TAPSE as a robust, high-yield biomarker [20]. Based on these findings, we advocate a change in standard practice at government and regional hospitals:

Routine Screening:

TAPSE should be a mandatory component of the 6-monthly dialysis echo protocol.

Red Flag Protocol:

A TAPSE < 17 mm should trigger a review of "dry weight" to minimize RV preload and an assessment of AV fistula flow to rule out high-output failure.

Nutritional Support:

Given the link with MIS, patients with RV dysfunction should be prioritized for nutritional counselling.

CLINICAL PERSPECTIVES

Competency in Medical Knowledge

Right ventricular (RV) dysfunction represents a pervasive but frequently overlooked pathology in ESRD. Our data indicates that significant RV systolic dysfunction

(TAPSE < 17 mm) is endemic, affecting over 40% of the maintenance hemodialysis population even within well-resourced facilities. Crucially, this study establishes TAPSE as a superior prognostic marker compared to traditional parameters like LVEF, offering a more accurate prediction of one-year mortality and cardiovascular admission. Consequently, the RV must be recognized not as a bystander, but as a critical determinant of systemic health, where failure leads to venous congestion that actively worsens malnutrition and inflammation.

Translational Outlook

These findings advocate for a paradigm shift in the routine cardiovascular assessment of dialysis patients. Standard echocardiographic protocols should mandate the inclusion of RV functional parameters, specifically TAPSE, due to its reproducibility and prognostic power. The identification of RV dysfunction should trigger a specific management algorithm focused on careful volume management to optimize preload, careful evaluation of vascular access flow to rule out high-output failure, and targeted nutritional support to mitigate the malnutrition-inflammation-atherosclerosis syndrome. Future research should explore the utility of advanced imaging modalities, such as RV longitudinal strain, in detecting subclinical dysfunction earlier in the disease course, and investigate whether RV-targeted pharmacological interventions can reverse remodelling and improve survival in this high-risk population.

CONCLUSIONS

In this ESRD cohort, the Right Ventricle has emerged as a critical, independent prognostic factor. While the center's optimized multidisciplinary dialysis care has achieved favorable mortality rates relative to the region, RV dysfunction continues to effectively stratify risk within this population. Consequently, the adoption of routine TAPSE assessment represents a pragmatic evolution in standard care. This simple addition would allow clinicians to pinpoint high-risk patients who require more aggressive cardiorenal and nutritional intervention.

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Conflicts of interest

The author declares no conflict of interest.

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