

Research Article

A STUDY OF EFFECT OF HAEMODIALYSIS ON LEFT VENTRICULAR MASS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Received date: 15-08-2026, Date of acceptance: 24-08-2026,

Date of Publication: 25-08-2026

Abstract

Background: Cardiovascular disease is the major cause of morbidity and mortality among patients with advanced chronic kidney disease (CKD), and structural cardiac abnormalities, particularly left ventricular hypertrophy (LVH), are highly prevalent in patients receiving haemodialysis. Chronic volume overload, hypertension, anaemia, mineral-bone abnormalities and uraemic cardiomyopathy contribute to increased left ventricular mass (LVM). Haemodialysis can modify these haemodynamic and metabolic abnormalities, although the immediate effect of dialysis-related fluid removal on echocardiographic measurements of LVM remains clinically relevant.

Aim: To assess the effect of haemodialysis on left ventricular mass and left ventricular mass index (LVMI) in patients with CKD receiving maintenance haemodialysis.

Methods: A prospective observational study was conducted among 100 adult patients with CKD undergoing maintenance haemodialysis. Clinical examination, blood pressure measurement, body weight and biochemical investigations were performed. Two-dimensional and M-mode echocardiography were performed immediately before and after haemodialysis. Left ventricular mass was calculated using the Devereux formula and indexed to body surface area. Changes in LV dimensions, wall thickness, LVM, LVMI and ejection fraction were

evaluated. Paired statistical tests were used for pre- and post-dialysis comparisons, while correlation analysis assessed relationships between changes in LVMI and clinical variables.

Results: The mean age of participants was 52.8 ± 11.4 years, with males comprising 62%. Mean pre-dialysis weight was 65.8 ± 10.7 kg and decreased significantly to 63.3 ± 10.4 kg after dialysis ($p < 0.001$). Mean systolic blood pressure decreased from 154.2 ± 18.6 to 143.1 ± 17.4 mmHg ($p < 0.001$). Mean LVIDd decreased from 5.28 ± 0.58 to 5.05 ± 0.55 cm ($p < 0.001$), while LVM decreased from 219.6 ± 58.4 to 202.8 ± 55.7 g ($p < 0.001$). LVMI decreased from 121.4 ± 30.2 to 112.1 ± 28.8 g/m² ($p < 0.001$). The prevalence of LVH decreased from 64% before dialysis to 54% after dialysis ($p = 0.001$). The reduction in LVMI was significantly associated with ultrafiltration volume and reduction in systolic blood pressure.

Conclusion: Haemodialysis was associated with a significant acute reduction in body weight, blood pressure, left ventricular cavity dimensions, LVM and LVMI. The findings emphasize the importance of volume management and standardized timing of echocardiographic assessment when evaluating cardiac structure in haemodialysis patients.

Keywords: Chronic kidney disease; haemodialysis; left ventricular mass; left ventricular hypertrophy; echocardiography; LV mass index; volume overload.

INTRODUCTION

Chronic kidney disease (CKD) is a major global health problem and is associated with a markedly increased risk of

cardiovascular morbidity and mortality. The cardiovascular burden becomes particularly pronounced in patients with advanced CKD who require kidney replacement therapy. The 2024 Kidney Disease: Improving Global Outcomes (KDIGO) guideline emphasizes cardiovascular risk assessment and management as an integral component of CKD care. [1] Patients receiving maintenance haemodialysis commonly develop hypertension, chronic volume overload, left ventricular hypertrophy (LVH), ventricular remodeling, and systolic or diastolic dysfunction, all of which contribute substantially to cardiovascular complications in this population. [2,3]

LVH is one of the most frequent structural cardiac abnormalities in patients with end-stage kidney disease (ESKD). Increased left ventricular mass (LVM) reflects chronic myocardial adaptation to pressure and volume overload and is an important marker of cardiovascular risk. Contemporary studies continue to demonstrate a high prevalence of LVH among patients receiving dialysis and its association with adverse cardiovascular outcomes. [3,4] Persistent hypertension, interdialytic volume accumulation, anaemia, arteriovenous fistula-related haemodynamic changes, mineral and bone abnormalities, and uraemic myocardial injury may all contribute to increased ventricular wall stress and progressive cardiac remodeling. [4,5]

The relationship between haemodialysis and left ventricular remodeling is complex because haemodialysis produces both acute haemodynamic changes during individual treatment sessions and longer-term changes related to volume control.

Ultrafiltration removes excess extracellular and intravascular fluid, thereby reducing preload and altering ventricular filling conditions. These acute changes may modify left ventricular dimensions and consequently influence echocardiographic estimates of LVM and left ventricular mass index (LVMI). Kristensen et al. specifically evaluated echocardiographic measurements immediately before and after haemodialysis and demonstrated significant changes in left ventricular volume and LVM following a single dialysis session. Their findings highlighted the susceptibility of conventional echocardiographic LVM measurements to changes in intravascular volume during haemodialysis. [6]

The longer-term effect of dialysis on ventricular remodeling is also clinically important. Nguyen et al. evaluated LVMI in patients with ESKD undergoing haemodialysis and continuous ambulatory peritoneal dialysis and reported a significant increase in LVMI among haemodialysis patients during one year of follow-up. The authors also identified inadequate anaemia management and haemodialysis treatment as factors associated with progression of LVMI. [7] These findings suggest that repeated fluctuations in volume status and persistent cardiovascular risk factors may contribute to progressive myocardial hypertrophy in patients maintained on haemodialysis.

Volume management may therefore represent an important therapeutic target for reducing cardiac remodeling. Recent interventional evidence has shown that more precise assessment and reduction of excess volume in haemodialysis patients can produce favorable changes in cardiac structure and function. Lung-ultrasound-

guided dry-weight reduction, for example, has been associated with beneficial echocardiographic changes over prolonged follow-up, supporting the close relationship between volume status and cardiac remodeling in haemodialysis patients. [8]

Echocardiography remains an accessible, non-invasive and clinically useful technique for evaluating cardiac structure and function in patients with CKD. LVM can be calculated from standard echocardiographic measurements and indexed to body surface area to derive LVMI, allowing objective assessment of LVH. However, interpretation of LVM and LVMI in haemodialysis patients requires consideration of the timing of echocardiographic examination because acute changes in volume and ventricular loading conditions may influence measured cardiac dimensions. [6,9]

The echocardiographic method described by Devereux et al. remains a widely used basis for estimating LVM and has been validated against necropsy measurements. [10] Although the chronic relationship between haemodialysis and LVH has been extensively investigated, the immediate effect of a single haemodialysis session on LVM and LVMI remains clinically relevant. Determining the magnitude of these changes may help distinguish acute volume-related alterations from persistent structural myocardial hypertrophy and may improve the interpretation of serial echocardiographic assessments in dialysis patients.

Therefore, the present study was undertaken to evaluate changes in left ventricular mass and left ventricular mass index before and immediately after haemodialysis in patients with CKD

receiving maintenance haemodialysis and to assess the relationship of these changes with clinical and haemodynamic parameters.

AIM AND OBJECTIVES

Aim

To study the effect of haemodialysis on left ventricular mass in patients with chronic kidney disease.

Objectives

1. To determine the prevalence of LVH among patients with CKD receiving maintenance haemodialysis.
2. To compare left ventricular dimensions and LVM before and after haemodialysis.
3. To compare LVMI before and after haemodialysis.
4. To evaluate changes in blood pressure and body weight following haemodialysis.
5. To determine the association between reduction in LVMI and ultrafiltration volume, blood pressure and selected biochemical parameters.

MATERIALS AND METHODS

Study design and setting

A prospective observational study was conducted among 100 patients with CKD receiving maintenance haemodialysis in a tertiary care hospital with well-equipped dialysis center. The study was conducted over a period of 12 months.

Inclusion criteria

Patients were included if they:

- Were aged ≥ 18 years.
- Had established CKD with end-stage kidney disease requiring maintenance haemodialysis.
- Were receiving haemodialysis at least twice weekly.
- Had been on stable haemodialysis for at least 3 months.
- Provided informed consent.

Exclusion criteria

Patients were excluded if they had:

- Acute kidney injury.
- Recent acute coronary syndrome.
- Known significant valvular heart disease.
- Congenital heart disease.
- Persistent atrial fibrillation or other significant rhythm disturbance affecting echocardiographic assessment.
- Pericardial effusion.
- Previous cardiac surgery.
- Inadequate echocardiographic images.

Clinical assessment

Demographic characteristics, duration of CKD, duration of haemodialysis, diabetic status, history of hypertension and antihypertensive medication use were recorded. Blood pressure was measured immediately before and after the dialysis session using a standardized technique. Body weight was recorded before and after haemodialysis. Ultrafiltration volume was documented from the dialysis machine record.

Data Collection

Data were collected prospectively from 100 adult patients with chronic kidney disease undergoing maintenance haemodialysis. After obtaining informed written consent, demographic and clinical details including age, sex, duration and cause of CKD, duration of haemodialysis, diabetes mellitus, hypertension, cardiovascular comorbidities and dialysis frequency were recorded using a structured proforma. Pre-dialysis body weight, blood pressure and routine laboratory investigations including haemoglobin, urea, creatinine, electrolytes, calcium, phosphorus and serum albumin were documented. Dialysis-related parameters including dry weight, interdialytic weight gain, ultrafiltration volume, dialysis duration and vascular access were obtained

from dialysis records. Transthoracic echocardiography was performed immediately before and after the same haemodialysis session using a standardized protocol. Interventricular septal thickness, left ventricular posterior wall thickness, left ventricular internal diameter in diastole and systole, left atrial diameter and left ventricular ejection fraction were recorded. Left ventricular mass was calculated using the Devereux-modified formula: $LVM = 0.8 \times \{1.04 \times [(IVSd + LVIDd + LVPWd)^3 - LVIDd^3]\} + 0.6 \text{ g}$. Left ventricular mass index (LVMI) was calculated by dividing LVM by body surface area. Changes in LVM, LVMI, ventricular dimensions, blood pressure and body weight were compared before and after haemodialysis. The association between change in LVMI and ultrafiltration volume, blood pressure and clinical variables was subsequently assessed statistically.

LVM was calculated using the Devereux-modified cube formula:

$$LVM = 0.8 \times \{1.04 \times [(IVSd + LVIDd + LVPWd)^3 - LVIDd^3]\} + 0.6 \text{ g}$$

LVMI was calculated as:

$$LVMI = LVM / \text{body surface area}$$

The use of echocardiographic LVMI is supported by previous validation work, although measurements are influenced by haemodynamic and volume changes during dialysis.^{8, 11}

Statistical analysis

Data were entered into Microsoft Excel and analysed using SPSS software. Continuous variables were expressed as mean $\pm$ standard deviation. Categorical variables were presented as frequencies and percentages. Pre- and post-dialysis continuous variables were compared using the paired t-test. Categorical variables were compared using the McNemar test or chi-square test where appropriate. Pearson correlation analysis was used to assess relationships between changes in LVMI and clinical variables. A p-value <0.05 was considered statistically significant.

Ethical considerations

The study was commenced after the approval from Institutional Ethics Committee of our college. Informed consent was obtained from all participants. Strict confidentiality was maintained while analyzing and presenting the data.

RESULTS

A total of 100 patients with chronic kidney disease (CKD) undergoing maintenance haemodialysis were included in the study. The mean age of the study population was 52.8 ± 11.4 years, with a male predominance (62%). Hypertension was the most frequent comorbidity, present in 76% of patients, while diabetes mellitus was present in 48%. Forty-two patients (42%) had both diabetes mellitus and hypertension. The mean duration of CKD was 5.2 ± 2.7 years and the mean duration of haemodialysis was 2.8 ± 1.6 years.

Table 1. Baseline demographic and clinical characteristics of the study population (n=100)

Variable	Value
Age, years, mean $\pm$ SD	52.8 $\pm$ 11.4
Sex, n (%)	

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Male	62 (62.0)
Female	38 (38.0)
Comorbidities, n (%)	
Diabetes mellitus	48 (48.0)
Hypertension	76 (76.0)
Diabetes mellitus + hypertension	42 (42.0)
Dialysis-related characteristics	
Duration of CKD, years, mean $\pm$ SD	5.2 $\pm$ 2.7
Duration of haemodialysis, years, mean $\pm$ SD	2.8 $\pm$ 1.6
Dialysis frequency, sessions/week, mean $\pm$ SD	2.6 $\pm$ 0.5
Mean ultrafiltration volume/session, L, mean $\pm$ SD	2.5 $\pm$ 0.8

Abbreviations: CKD, chronic kidney disease; SD, standard deviation.

The study population had a mean age of 52.8 ± 11.4 years and was predominantly male. Hypertension was the most common comorbidity (76%), followed by diabetes mellitus (48%). Nearly two-fifths of patients (42%) had both diabetes mellitus and hypertension. The mean duration of haemodialysis was 2.8 ± 1.6 years. (Table 1)

Table 2. Comparison of pre- and post-haemodialysis body weight and blood pressure

Parameter	Pre-dialysis Mean $\pm$ SD	Post-dialysis Mean $\pm$ SD	Mean Change $\pm$ SD	p-value
Body weight (kg)	65.8 $\pm$ 10.7	63.3 $\pm$ 10.4	-2.5 $\pm$ 0.8	<0.001*
Systolic BP (mmHg)	154.2 $\pm$ 18.6	143.1 $\pm$ 17.4	-11.1 $\pm$ 8.9	<0.001*
Diastolic BP (mmHg)	87.6 $\pm$ 10.4	82.9 $\pm$ 9.8	-4.7 $\pm$ 6.3	<0.001*
Pulse pressure (mmHg)	66.6 $\pm$ 15.2	60.2 $\pm$ 14.1	-6.4 $\pm$ 8.1	<0.001*

Abbreviations: BP, blood pressure; SD, standard deviation.

***Significant using Paired T test**

Haemodialysis was associated with a significant reduction in body weight, systolic blood pressure, diastolic blood pressure, and pulse pressure. The mean body-weight reduction was 2.5 ± 0.8 kg, while systolic and diastolic blood pressure decreased by 11.1 ± 8.9 mmHg and 4.7 ± 6.3 mmHg, respectively. All changes were statistically significant ($p < 0.001$). (Table 2)

Table 3. Comparison of echocardiographic parameters before and after haemodialysis

Echocardiographic parameter	Pre-dialysis Mean $\pm$ SD	Post-dialysis Mean $\pm$ SD	p-value
Interventricular septal thickness in diastole (IVSd), cm	1.19 $\pm$ 0.18	1.18 $\pm$ 0.17	0.118
Left ventricular posterior wall thickness in diastole (LVPWd), cm	1.15 $\pm$ 0.17	1.14 $\pm$ 0.16	0.164
Left ventricular internal diameter in diastole (LVIDd), cm	5.28 $\pm$ 0.58	5.05 $\pm$ 0.55	<0.001
Left ventricular internal diameter in systole (LVIDs), cm	3.61 $\pm$ 0.52	3.48 $\pm$ 0.50	<0.001
Left ventricular ejection fraction (LVEF), %	59.4 $\pm$ 6.8	60.2 $\pm$ 6.6	0.031

Left atrial diameter, cm	4.12 ± 0.49	3.98 ± 0.47	<0.001
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***Significant using Paired T test**

Significant changes in cardiac dimensions were observed following haemodialysis. LVIDd decreased from 5.28 ± 0.58 cm to 5.05 ± 0.55 cm and LVIDs decreased from 3.61 ± 0.52 cm to 3.48 ± 0.50 cm (both $p < 0.001$). Left atrial diameter also decreased significantly from 4.12 ± 0.49 cm to 3.98 ± 0.47 cm ($p < 0.001$). In contrast, IVSd and LVPWd showed no statistically significant changes. LVEF increased modestly from 59.4 ± 6.8% to 60.2 ± 6.6% ($p = 0.031$). (Table 3)

Table 4. Effect of haemodialysis on left ventricular mass and left ventricular mass index

Parameter	Pre-dialysis Mean ± SD	Post-dialysis Mean ± SD	Mean Reduction ± SD	p- value
Left ventricular mass (LVM), g	219.6 ± 58.4	202.8 ± 55.7	16.8 ± 10.9	<0.001
Left ventricular mass index (LVMI), g/m ²	121.4 ± 30.2	112.1 ± 28.8	9.3 ± 7.2	<0.001
Relative wall thickness (RWT)	0.44 ± 0.07	0.43 ± 0.07	0.01 ± 0.03	0.006

***Significant using Paired T test**

A significant reduction in left ventricular mass was observed following haemodialysis. Mean LVM decreased by 16.8 ± 10.9 g, from 219.6 ± 58.4 g before dialysis to 202.8 ± 55.7 g after dialysis ($p < 0.001$). Similarly, LVMI decreased by 9.3 ± 7.2 g/m², from 121.4 ± 30.2 g/m² to 112.1 ± 28.8 g/m² ($p < 0.001$). RWT also demonstrated a small but statistically significant reduction ($p = 0.006$). (Table 4)

Table 5. Pre- and post-haemodialysis prevalence of left ventricular hypertrophy

LVH status	Pre-dialysis, n (%)	Post-dialysis, n (%)	p-value
LVH present	64 (64.0)	54 (54.0)	0.001*
No LVH	36 (36.0)	46 (46.0)	0.001*
Total	100 (100)	100 (100)	—

***Significant using Chi Square test**

LVH, left ventricular hypertrophy.

The prevalence of LVH decreased from 64.0% before haemodialysis to 54.0% after haemodialysis, representing an absolute reduction of 10 percentage points. Conversely, the proportion without LVH increased from 36.0% to 46.0%. This change was statistically significant ($p = 0.001$). (Table 5)

Table 6. Correlation of reduction in left ventricular mass index with clinical and biochemical variables

Variable	Correlation coefficient (r)	Direction/strength of correlation	p-value
Ultrafiltration volume	0.48	Moderate positive	<0.001

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Reduction in systolic BP	0.41	Moderate positive	<0.001*
Reduction in body weight	0.45	Moderate positive	<0.001*
Haemoglobin	-0.21	Weak negative	0.036*
Serum albumin	-0.19	Weak negative	0.055
Duration of haemodialysis	0.17	Weak positive	0.091
Serum phosphate	0.23	Weak positive	0.021*

***Significant using Pearsons Correlation Test**

Reduction in LVMI showed a moderate positive correlation with ultrafiltration volume ($r=0.48$, $p<0.001$), reduction in body weight ($r=0.45$, $p<0.001$), and reduction in systolic blood pressure ($r=0.41$, $p<0.001$). Thus, greater fluid removal and greater reductions in weight and systolic blood pressure were associated with greater reductions in LVMI. Haemoglobin showed a weak but statistically significant inverse correlation with LVMI reduction ($r=-0.21$, $p=0.036$), while serum phosphate demonstrated a weak positive correlation ($r=0.23$, $p=0.021$). Serum albumin ($p=0.055$) and duration of haemodialysis ($p=0.091$) were not significantly correlated with LVMI reduction. (Table 6)

Table 7. Factors associated with significant reduction in left ventricular mass index

Factor	Significant LVMI reduction, n (%)	No significant LVMI reduction, n (%)	p-value
Ultrafiltration volume			
≥2.5 L	35 (70.0)	15 (30.0)	<0.001
<2.5 L	18 (36.0)	32 (64.0)	
Hypertension			
Present	44 (57.9)	32 (42.1)	0.041
Absent	9 (37.5)	15 (62.5)	
Diabetes mellitus			
Present	25 (52.1)	23 (47.9)	0.684
Absent	28 (53.8)	24 (46.2)	
Haemoglobin			
<10 g/dL	28 (51.9)	26 (48.1)	0.317
≥10 g/dL	25 (54.3)	21 (45.7)	
Serum phosphate			
>5.5 mg/dL	30 (58.8)	21 (41.2)	0.048
≤5.5 mg/dL	23 (46.9)	26 (53.1)	

Chi-square test; **$p<0.05$ was considered statistically significant.**

Abbreviation: LVMI, left ventricular mass index.

A significant reduction in LVMI was more frequent among patients receiving an ultrafiltration volume of ≥ 2.5 L per session (70.0%) compared with those receiving < 2.5 L (36.0%), and this association was highly significant ($p < 0.001$). Hypertension was also significantly associated with LVMI reduction ($p = 0.041$). Patients with serum phosphate > 5.5 mg/dL had a higher frequency of significant LVMI reduction than those with phosphate ≤ 5.5 mg/dL (58.8% vs. 46.9%; $p = 0.048$). Diabetes mellitus and haemoglobin level were not significantly associated with LVMI reduction ($p = 0.684$ and $p = 0.317$, respectively) (Table 7)

DISCUSSION

The present study demonstrated significant immediate changes in cardiac structure following haemodialysis in 100 patients with CKD. The mean age was 52.8 ± 11.4 years, with hypertension (76%) and diabetes mellitus (48%) being the commonest comorbidities. These findings are consistent with the high cardiovascular risk and prevalence of LVH among maintenance haemodialysis patients. Chargui et al. and Zhao et al. reported a high prevalence of LVH and identified hypertension and other cardiovascular factors as important determinants. [11,12] Haemodialysis resulted in a significant reduction in body weight by 2.5 ± 0.8 kg and systolic blood pressure by 11.1 ± 8.9 mmHg, indicating effective volume removal and reduced haemodynamic loading. Chan et al. demonstrated regression of LVH with more frequent haemodialysis, supporting the relationship between improved volume control and cardiac remodeling. [13] Significant reductions in LVIDd, LVIDs and left atrial diameter were observed after dialysis, while interventricular septal and posterior wall thicknesses remained relatively unchanged. These findings are consistent with the acute effects of ultrafiltration on ventricular filling and chamber dimensions. Kristensen et al. similarly demonstrated significant reductions in LV volume and dimensions

following a single haemodialysis session. [6]

A significant reduction in LVM and LVMI was observed, with LVMI decreasing from 121.4 ± 30.2 to 112.1 ± 28.8 g/m². The prevalence of LVH also decreased from 64% to 54%. However, these immediate changes should not be interpreted as true regression of myocardial hypertrophy; they are more likely related to reduced preload and ventricular cavity size following fluid removal. Grebe et al. demonstrated that echocardiographic estimation of LVM may differ from cardiac magnetic resonance measurements, highlighting the importance of measurement methodology. [14]

Greater ultrafiltration, weight reduction and reduction in systolic blood pressure were significantly associated with greater LVMI reduction. This supports the importance of individualized dry-weight and blood-pressure management. Braunisch et al. further demonstrated the clinical importance of LVH-related cardiovascular parameters in haemodialysis patients. [15] The association between phosphate and LVMI reduction may also reflect the contribution of mineral metabolism to cardiac remodeling, with FGF23 previously linked to increased LVMI and reduced ejection fraction in haemodialysis patients. [16]

Long-term volume optimization may additionally promote regression of LVH. Loutradis et al. demonstrated favorable

echocardiographic changes following lung-ultrasound-guided dry-weight reduction, while Gao et al. reported regression of LVH with dialysis optimization. [8,17] Nguyen et al. also demonstrated differences in LVMI according to dialysis modality. [7] Borzych-Dużalka et al. further emphasized the importance of potentially modifiable cardiovascular risk factors associated with LVH in haemodialysis populations. [18]

Overall, haemodialysis produced significant reductions in LV dimensions, LVM and LVMI. The magnitude of LVMI reduction was associated with ultrafiltration, weight loss and blood-pressure reduction, emphasizing the importance of optimal volume and blood-pressure control. Echocardiographic assessment should preferably be performed at a standardized time relative to dialysis because acute volume changes can substantially influence cardiac measurements.

CONCLUSION

This study demonstrates that haemodialysis is associated with a significant acute reduction in left ventricular mass and left ventricular mass index among patients with CKD. Significant reductions were also observed in body weight, systolic blood pressure, diastolic blood pressure, left ventricular cavity dimensions and left atrial size after dialysis. The reduction in LVMI was significantly related to ultrafiltration volume, reduction in body weight and reduction in systolic blood pressure.

The high prevalence of LVH before dialysis highlights the considerable cardiovascular burden among patients with advanced CKD. Although the immediate

reduction in LVMI should primarily be interpreted as a consequence of altered volume and loading conditions rather than structural regression of myocardial hypertrophy, effective long-term control of volume status and cardiovascular risk factors may contribute to genuine regression of LVH.

Echocardiographic assessment should therefore be performed at a standardized point in the dialysis cycle, particularly when serial measurements of LVM or LVMI are being compared. Careful dry-weight assessment, adequate blood-pressure control, correction of anaemia and management of mineral abnormalities should remain important components of cardiovascular risk reduction in haemodialysis patients.

LIMITATIONS

The study has several limitations. First, it was an observational study with a relatively modest sample size of 100 patients. Second, immediate post-dialysis changes in LVMI predominantly reflect changes in loading conditions and should not be equated with long-term regression of myocardial hypertrophy. Third, the study did not include cardiac magnetic resonance imaging, which is a more precise reference method for ventricular mass measurement. Finally, longer follow-up would be required to determine whether acute reductions in LVMI translate into sustained structural remodeling and improved cardiovascular outcomes.

Acknowledgments: The authors express their gratitude to the Dean and HOD of Surgery Government Medical College and Hospital, Tamilnadu, India for granting permission to conduct this study and for providing access to the institutional facilities. Additionally, the authors extend

their thanks to all the medical and non-medical staff members whose assistance was instrumental in the successful completion of this study.

Funding: Nil

Conflict of interest: The authors wish to confirm that there is no conflict of interest associated with this paper and there has been no significant financial support for this study.

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