Research Square

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Early Cardiopulmonary Adaptation Following Arteriovenous Fistula Creation in Pre- Dialysis Chronic Kidney Disease Patients: A Prospective Echocardiographic Study

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Abstract

Background

Arteriovenous fistula (AVF) creation induces substantial haemodynamic alterations through increased venous return and reduced systemic vascular resistance. Although cardiovascular remodelling after AVF creation has been described in haemodialysis populations, prospective echocardiographic data in pre-dialysis chronic kidney disease (CKD) patients remain limited.

Methods

This prospective observational study enrolled adult pre-dialysis CKD patients undergoing elective AVF creation at the University of Malaya Medical Centre between March 2021 and December 2022. Transthoracic echocardiography was performed before AVF creation, at early follow-up (4–12 weeks), and at late follow-up (≥ ≥ 6 months). Parameters assessed included left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), left ventricular ejection fraction (LVEF), stroke volume (SV), left ventricular mass index (LVMI), cardiac index (CI), pulmonary artery systolic pressure (PASP), and right ventricular functional indices. Paired analyses were performed using paired t-tests or Wilcoxon signed-rank tests according to normality of paired differences.

Results

Thirty-four patients underwent baseline echocardiography, with 27 completing early follow-up and 14 completing late follow-up. Mean age was 65.2 ± 8.9 years, and 52.9% were male. At early follow-up, LVEDV increased from 79.8 ± 21.8 mL to 104.8 ± 32.0 mL (mean difference + 25.0 mL, 95% CI 11.1–39.0; p = 0.001), and LVESV increased from 29.4 ± 10.5 mL to 37.5 ± 15.5 mL (mean difference + 8.1 mL, 95% CI 2.1–14.1; p = 0.012). SV increased from 50.5 ± 13.5 mL to 66.2 ± 21.0 mL (mean difference + 15.8 mL, 95% CI 6.5–24.9; p = 0.002), accompanied by an increase in CI from 2.18 ± 0.65 to 3.05 ± 1.15 L/min/m² (mean difference + 0.87 L/min/m², 95% CI 0.28–1.46; p = 0.007). LVEF remained preserved throughout follow-up. PASP increased from 28.0 ± 8.6 mmHg to 34.2 ± 9.9 mmHg (mean difference + 6.2 mmHg, 95% CI 0.7–11.7; p = 0.032), while TAPSE also increased. Patients with proximal AVFs demonstrated greater PASP increases than those with distal AVFs.

Conclusions

AVF creation induces early cardiopulmonary adaptation in pre-dialysis CKD patients, characterised by increased LV volume loading, augmented cardiac output, and increased pulmonary haemodynamic burden while preserving systolic function. Proximal AVFs may confer greater pulmonary circulatory burden, supporting selective cardiovascular surveillance in higher-risk patients, although larger studies are needed to confirm these findings.

1. Background

Chronic kidney disease affects an estimated 850 million people globally and carries a substantially elevated risk of premature cardiovascular mortality [1,2,3]. Arteriovenous fistulae (AVFs) remain the preferred vascular access modality for maintenance haemodialysis because of superior long-term patency and lower infection risk compared

with central venous catheters [6]. Nevertheless, AVF creation induces substantial systemic haemodynamic alterations that may affect cardiovascular structure and function, particularly in patients with advanced chronic kidney disease (CKD), who frequently already exhibit pre-existing cardiac abnormalities [4,5,7–11].

By creating a low-resistance conduit between the arterial and venous circulation, AVFs increase venous return, augment cardiac preload, and elevate cardiac output [7–10]. Sustained high-flow states may subsequently contribute to ventricular remodelling, pulmonary hypertension, and high-output cardiac failure [14,15,16]. These haemodynamic effects are thought to be more pronounced in proximal or high-flow AVFs because of greater shunt flow volumes [18–20].

Several studies have demonstrated increases in cardiac output, ventricular dimensions, and left ventricular mass following AVF creation [7–13,21–23]. However, much of the existing literature has focused on established haemodialysis populations or renal transplant recipients, limiting applicability to pre-dialysis CKD patients [11–13,21–23]. Furthermore, many studies utilised cardiac magnetic resonance imaging or retrospective analyses, while prospective serial echocardiographic data in pre-dialysis CKD remain relatively limited [11–13].

Understanding the early cardiovascular response to AVF creation before dialysis initiation may be clinically important, particularly in patients with baseline cardiac dysfunction or elevated cardiopulmonary risk [4–5,14–20]. Early identification of maladaptive haemodynamic responses may inform vascular access planning and post-operative surveillance strategies [6,17–20].

The present study therefore aimed to evaluate serial echocardiographic changes following AVF creation in pre-dialysis CKD patients, with particular emphasis on cardiopulmonary adaptation, pulmonary haemodynamics, and right ventricular function. Secondary objectives were to explore whether these changes differed according to AVF location and access flow rate [18–20].

2. Methods

2.1 Study design and setting

This was a single-centre prospective observational study conducted at the University of Malaya Medical Centre, Malaysia, between March 2021 and December 2022.

2.2 Study population

Adult patients aged 18 years or older with advanced CKD planned for elective AVF creation were eligible for inclusion. Exclusion criteria included atrial fibrillation, significant valvular heart disease, baseline New York Heart Association class III or IV symptoms, baseline left ventricular ejection fraction below 40%, and urgent haemodialysis initiation before AVF creation.

A total of 91 patients were screened. Thirty-four patients fulfilled eligibility criteria and completed baseline echocardiographic assessment.

2.3 Echocardiographic assessment

Transthoracic echocardiography was performed at three predefined timepoints: (1) baseline before AVF creation, (2) early follow-up at 4–12 weeks, and (3) late follow-up at ≥ 6 months. To capture the acute phase of

haemodynamic adaptation, the earliest echocardiographic study was preferentially selected for paired analysis when multiple timepoints fell within the early follow-up window.

Echocardiographic parameters included:

- Left ventricular end-diastolic volume (LVEDV)
- Left ventricular end-systolic volume (LVESV)
- Left ventricular ejection fraction (LVEF)
- Stroke volume (SV)
- Left ventricular mass (LVM)
- Left ventricular mass index (LVMI)
- Mitral inflow parameters
- E/A ratio
- Isovolumic relaxation time (IVRT)
- Deceleration time
- Pulmonary artery systolic pressure (PASP)
- Tricuspid annular plane systolic excursion (TAPSE)
- Right ventricular S' velocity [25]

SV was defined using the Simpson biplane-derived stroke volume (MOD-sp4) variable from the raw dataset. Cardiac index was recalculated as cardiac output divided by body surface area, where cardiac output was derived from SV × heart rate and body surface area was calculated using the DuBois formula [25].

2.4 AVF characteristics

AVF location and access flow were assessed using duplex ultrasonography during post-operative follow-up [6]. AVFs were classified as proximal if created at or above the antecubital fossa and distal if created below the antecubital fossa [6,18–20].

Access flow was categorised as low-flow if below 600 mL/min and high-flow if 600 mL/min or greater [6,19,20].

2.5 Statistical analysis

Continuous variables are presented as mean ± standard deviation, while categorical variables are presented as frequency and percentage.

Normality of paired differences was assessed using the Shapiro–Wilk test. Normally distributed paired data were analysed using paired t-tests, while non-normally distributed paired data were analysed using Wilcoxon signed-rank tests.

Mean differences with corresponding 95% confidence intervals (CI) were calculated for principal paired analyses.

Owing to incomplete follow-up and varying paired sample sizes across timepoints, pairwise paired analyses were chosen rather than repeated-measures modelling.

Missing echocardiographic parameters were handled using available-case analysis without imputation because missingness primarily reflected technical acquisition limitations (e.g., inadequate acoustic windows or

unmeasurable tricuspid regurgitant envelopes).Given the exploratory physiological nature of this study and the relatively small cohort size, formal correction for multiple comparisons was not performed. Findings should therefore be interpreted as hypothesis-generating. A two-tailed p-value below 0.05 was considered statistically significant.

3. Results

3.1 Baseline characteristics and follow-up

A total of 34 patients underwent AVF creation and completed baseline echocardiography. Mean age was 65.2 ± 8.9 years, and 18 patients (52.9%) were male. Diabetes mellitus and hypertension were present in 32 (94.1%) and 28 (82.4%) patients, respectively, while 26 patients (76.5%) had stage V CKD.

Early echocardiographic follow-up was available for 27 patients overall, while 14 patients completed late follow-up echocardiography. Analysable sample size varied according to parameter availability. Sample sizes varied due to acoustic shadowing, unmeasurable tricuspid regurgitant jet envelopes for PASP calculation, or poor endocardial border visualization.

Twenty patients underwent proximal AVF creation and 14 underwent distal AVF creation within the early follow-up cohort.

[Figure 1]

[Table 1]

Table 1
Baseline Characteristics

Characteristic	Value
n	34
Age (years), mean ± SD	65.2 ± 8.9
Male sex, n (%)	18 (52.9%)
Race, n (%)	
— Malay	20 (58.8%)
— Chinese	9 (26.5%)
— Indian	5 (14.7%)
Diabetes mellitus, n (%)	32 (94.1%)
Hypertension, n (%)	28 (82.4%)
Cause of ESRD, n (%)	
— Diabetic nephropathy	29 (85.3%)
— Hypertensive nephropathy	3 (8.8%)
— Other	2 (5.9%)
eGFR (mL/min/1.73m²), mean ± SD	12.9 ± 4.4
Haemoglobin (g/L), mean ± SD	106.3 ± 14.5
CKD Stage V, n (%)	26 (76.5%)
Baseline echocardiography	
LVEF (%), mean ± SD	65.2 ± 7.3
LV enlarged, n (%)	14 (41.2%)
LV systolic dysfunction, n (%)	1 (2.9%)
LV diastolic dysfunction, n (%)	26 (76.5%)
AVF characteristics	
Proximal AVF, n (%)	20 (58.8%)
Distal AVF, n (%)	14 (41.2%)
High-flow (≥ 600 mL/min), n (%)	20/29 (69.0%)

3.2 Left ventricular structural changes

LVEDV increased significantly at early follow-up from 79.8 ± 21.8 mL to 104.8 ± 32.0 mL (mean difference + 25.0 mL, 95% CI 11.1–39.0; p = 0.001; n = 25). This increase remained evident at late follow-up, rising from 77.3 ± 26.7 mL to 98.0 ± 26.8 mL (p = 0.038; n = 13).

LVESV similarly increased at early follow-up from 29.4 ± 10.5 mL to 37.5 ± 15.2 mL (mean difference + 8.1 mL, 95% CI 1.9–14.3; p = 0.012; n = 25). A significant increase also persisted at late follow-up (34.0 ± 7.9 mL, p = 0.039).

No statistically significant changes in LVM or LVMI were observed at either follow-up timepoint.

[Table 2]

Table 2
Serial Echocardiographic Changes (primary outcomes)

Parameter	Baseline	Early FU (4–12 wk)	MD (95% CI)	Δ%	p	n	Late FU (≥ 6 mo)	MD (95% CI)	Δ%	p	n
LV Volumes & Systolic Function											
LVEDV (mL)	79.8 ± 21.8	104.8 ± 32.0	+ 25.0 (+ 11.1, + 39.0)	+ 31.4%	**0.001**	25	98.0 ± 25.8	+ 20.7 (+ 1.4, + 40.0)	+ 26.8%	**0.038**	13
LVESV (mL)	29.4 ± 10.5	37.5 ± 15.2	+ 8.1 (+ 1.9, + 14.3)	+ 27.5%	**0.012**	25	34.0 ± 7.6	+ 6.4 (+ 0.4, + 12.3)	+ 23.0%	**0.039**	13
LVEF (%)	64.6 ± 7.1	63.6 ± 6.4	−1.0 (− 3.8, + 1.8)	−1.5%	0.463	27	64.5 ± 5.4	−0.6 (− 5.2, + 4.0)	−1.0%	0.767	14
SV (mL)	50.5 ± 13.5	66.2 ± 20.6	+ 15.8 (+ 6.6, + 25.0)	+ 31.2%	**0.002**	25	64.0 ± 20.5	+ 14.3 (− 1.5, + 30.1)	+ 28.9%	0.072	13
Diastolic Function											
E/A ratio	0.94 ± 0.49	1.10 ± 0.53	+ 0.06 (− 0.02, + 0.24)	+ 17.4%	0.070	23	1.00 ± 0.48	−0.05 (− 0.10, + 0.14)	+ 6.5%	1.000	13
IVRT (s)	0.083 ± 0.021	0.099 ± 0.023	+ 0.016 (+ 0.003, + 0.030)	+ 19.3%	**0.023**	18	0.083 ± 0.022	+ 0.005 (− 0.015, + 0.024)	+ 5.5%	0.629	7
LV Structure											
LVM (g)	233.6 ± 62.7	235.9 ± 63.0	+ 2.3 (− 25.3, + 29.9)	+ 1.0%	0.864	23	234.1 ± 61.3	−7.6 (− 22.9, + 7.8)	−3.1%	0.317	22
LVMI (g/m²)	130.3 ± 29.8	132.5 ± 33.7	+ 2.2 (− 13.6, + 18.0)	+ 1.7%	0.772	23	130.2 ± 31.6	−1.3 (− 10.0, + 7.4)	−1.0%	0.764	22

Parameter	Baseline	Early FU (4–12 wk)	MD (95% CI)	Δ%	p	n	Late FU (≥ 6 mo)	MD (95% CI)	Δ%	p	n
LV Volumes & Systolic Function											
Pulmonary & RV											
PASP (mmHg)	28.0 ± 8.6	34.2 ± 9.9	+ 6.2 (+ 0.6, + 11.8)	+ 22.2%	**0.032**	14	33.0 ± 10.2	+ 4.5 (− 7.3, + 16.3)	+ 15.8%	0.372	6
TAPSE (mm)	21.9 ± 4.5	24.5 ± 5.2	+ 2.6 (+ 0.9, + 4.4)	+ 12.0%	**0.005**	27	22.6 ± 3.2	0.0 (− 3.5, + 3.5)	0.0%	1.000	14

[Figure 2]

3.3 Left ventricular systolic function and cardiac output

LVEF remained preserved throughout follow-up and did not change significantly.

At early follow-up, SV increased significantly from 50.5 ± 13.5 mL to 66.2 ± 21.0 mL (mean difference + 15.8 mL, 95% CI 6.5–24.9; p = 0.002; n = 25).

CI also increased significantly at early follow-up from 2.18 ± 0.65 L/min/m² to 3.05 ± 1.15 L/min/m² (mean difference + 0.87 L/min/m², 95% CI 0.28–1.46; p = 0.007; n = 21).

At late follow-up, SV and CI remained numerically higher than baseline values but no longer reached statistical significance.

3.4 Diastolic function

The E/A ratio demonstrated a non-significant trend toward increase at early follow-up, while mitral deceleration time did not change significantly.

IVRT increased significantly at early follow-up from 0.083 ± 0.021 seconds to 0.099 ± 0.023 seconds (mean difference + 0.016 seconds, 95% CI 0.003–0.030; p = 0.023; n = 18).

No significant IVRT change was observed at late follow-up.

3.5 Pulmonary haemodynamics and right ventricular function

PASP increased significantly at early follow-up from 28.0 ± 8.6 mmHg to 34.2 ± 9.9 mmHg (mean difference + 6.2 mmHg, 95% CI 0.7–11.7; p = 0.032; n = 14).

At late follow-up, PASP remained numerically elevated but did not reach statistical significance.

TAPSE increased significantly at early follow-up from 21.9 ± 4.5 mm to 24.5 ± 5.3 mm (mean difference + 2.6 mm, 95% CI 0.9–4.3; p = 0.005; n = 27).

RV S' velocity did not change significantly at either follow-up timepoint.

The increase in TAPSE, in the absence of RV S' change, may reflect augmented RV preload rather than impaired RV contractility.

3.6 Subgroup analysis according to AVF location

Subgroup analyses were restricted to patients with paired PASP measurements (n = 14).Among patients with paired PASP data, proximal AVFs demonstrated a numerically larger PASP increase than distal AVFs. The between-group difference in ΔPASP was statistically significant (+ 11.4 ± 9.6 mmHg vs + 1.0 ± 6.8 mmHg; p = 0.030), although findings should be interpreted cautiously given the small subgroup size.

[Table 3]

Table 3
PASP Subgroup – Distal vs Proximal AVF group

PASP baseline (mmHg)	**Proximal AVF (n = 7)**	**Distal AVF (n = 7)**	**Between-group p**
	24.7 ± 4.5	**31.3 ± 10.7**	**—**
PASP early FU (mmHg)	36.1 ± 12.2	32.3 ± 8.5	—
ΔPASP (mmHg)	+ 11.4 ± 9.6	+ 1.0 ± 6.8	**0.030**
Within-group p	**0.020**	0.710	—

3.7 Subgroup analysis according to AVF flow rate

Flow-rate data were available for 29 patients at early follow-up, with mean AVF flow of 793 ± 318 mL/min. Formal between-group statistical comparisons were not performed due to insufficient paired data.

3.8 Correlation between AVF characteristics and pulmonary haemodynamic response

AVF proximity was significantly correlated with the magnitude of early PASP change (Spearman ρ = −0.621, p = 0.018, n = 14), with proximal AVFs associated with greater PASP increases. A positive trend was observed between AVF flow rate and ΔPASP (Spearman ρ = +0.561, p = 0.073, n = 11), although this did not reach statistical significance, likely reflecting limited sample size in the paired PASP dataset. No significant correlations were identified between AVF flow rate and changes in LVEDV, SV, or CI.

[Figure 3]

4. Discussion

This prospective study demonstrates that AVF creation in pre-dialysis CKD patients is associated with significant early cardiopulmonary adaptation characterised by increased LV volume loading, augmented cardiac output, and increased pulmonary haemodynamic stress while preserving left ventricular systolic function [7–12].

The observed increase in LVEDV and LVESV supports the concept that AVF creation induces substantial preload augmentation through increased venous return and reduced systemic vascular resistance [7–10]. By diverting arterial blood directly into the venous circulation, AVFs create a chronic low-resistance state that increases cardiac preload and necessitates compensatory increases in cardiac output [7–10].

Importantly, despite significant increases in LV volumes, LVEF remained preserved throughout follow-up. This finding suggests that the haemodynamic response observed in this cohort primarily reflects an adaptive high-output physiological state rather than early systolic decompensation [7–13]. The concomitant increase in stroke volume and cardiac index further supports compensatory augmentation of forward flow via the Frank–Starling mechanism [7–12].

Our findings are broadly consistent with previous studies demonstrating increased cardiac output and ventricular dimensions following AVF creation [7–13]. However, most previous literature has focused on established haemodialysis populations or renal transplant recipients, in whom cardiovascular remodelling may already be substantially advanced [11–13,21–23]. In contrast, the present study specifically evaluated pre-dialysis CKD patients using serial echocardiographic assessment, thereby providing insight into the earlier phases of cardiovascular adaptation before dialysis initiation [9,10,12].

Interestingly, no significant increase in LV mass or LVMI was observed. Several explanations may account for this finding. First, the duration of haemodynamic exposure may have been insufficient for detectable hypertrophic remodelling to occur [11,21–24]. Second, the relatively small sample size may have limited statistical power to detect gradual structural changes. Third, pre-dialysis CKD patients may experience different haemodynamic adaptation trajectories compared with long-term haemodialysis populations [10–13,24].

The physiological significance of the observed IVRT increase remains uncertain and may reflect preload-related alterations or measurement variability rather than clinically meaningful diastolic impairment.

One of the more clinically relevant findings of the present study was the significant increase in pulmonary artery systolic pressure following AVF creation [14–20]. Increased pulmonary blood flow secondary to AVF-mediated preload augmentation may contribute to elevated pulmonary pressures, particularly in patients with impaired pulmonary vascular reserve or baseline diastolic dysfunction [14–18].

The concomitant increase in TAPSE may represent an adaptive right ventricular response to increased preload and pulmonary flow rather than overt right ventricular dysfunction [13–18]. Nevertheless, TAPSE is load-dependent and should therefore be interpreted cautiously in the context of altered haemodynamics following AVF creation. Importantly, proximal AVFs were associated with numerically larger and statistically significant increases in PASP, although interpretation is limited by small subgroup size [18–20]. This observation is biologically plausible because proximal accesses typically generate larger shunt volumes and higher access flows, thereby imposing greater cardiopulmonary burden [18–20].

The significant correlation between AVF proximity and ΔPASP ($\rho = -0.621$, $p = 0.018$) provides mechanistic support for the hypothesis that proximal AVFs impose disproportionate pulmonary haemodynamic burden through greater shunt flow volumes [18–20]. Notably, AVF flow rate and location did not correlate with changes in LV volumes or cardiac index, suggesting that the degree of LV volume loading after AVF creation may be less flow-dependent than the pulmonary vascular response [10–13,18–20].

These findings may have potential clinical implications. Although AVFs remain the preferred vascular access modality, selective cardiovascular surveillance may be reasonable in higher-risk patients, particularly those with pre-existing cardiopulmonary disease or proximal high-flow AVFs [6,16–20]. Early identification of patients who develop substantial pulmonary haemodynamic stress may potentially inform vascular access planning and longitudinal monitoring strategies [6,17–20].

5. Limitations

This study has several limitations.

First, the overall cohort size and particularly the late follow-up subgroup were relatively small, thereby reducing statistical power and limiting generalisability.

Second, incomplete echocardiographic follow-up led to variable analysable sample sizes across parameters. Patients completing follow-up echocardiography may have differed systematically from those lost to follow-up, introducing potential attrition bias.

Third, subgroup analyses according to AVF location and flow rate were exploratory and based on limited sample sizes.

Fourth, the observational study design precludes causal inference.

In addition, paired PASP measurements were available only in a subset of patients, which may limit precision of pulmonary haemodynamic analyses.

Finally, given the exploratory physiological nature of this hypothesis-generating study and the limited sample size, formal adjustment for multiple comparisons was not performed to avoid excessive type II error. Findings should therefore be interpreted cautiously and considered exploratory.

6. Conclusions

AVF creation in pre-dialysis CKD patients induces early and measurable cardiopulmonary adaptation characterised by increased LV volume loading, augmented cardiac output, and increased pulmonary haemodynamic stress while preserving LV systolic function [7–15].

Patients with proximal AVFs may experience greater pulmonary haemodynamic burden, supporting consideration of selective cardiovascular surveillance in higher-risk individuals [18–20].

Further prospective multicentre studies are required to better define the long-term cardiovascular implications of AVF-mediated haemodynamic remodelling in advanced CKD.

Declarations

Conflict of Interest: The authors declare no conflict of interest.

Ethics Approval: Approved by the University Malaya Medical Centre Medical Research Ethics Committee, MREC ID NO: 2021118-9726.

Ethics approval and consent to participate
This study was approved by the Medical Research Ethics Committee, University of Malaya Medical Centre, Kuala Lumpur, Malaysia (MREC ID No: 2021118-9726), and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Consent for publication
Not applicable.

Authors' contributions
KVP: Conceptualization, methodology, data collection, data curation, formal analysis, writing of the original draft, and review and editing of the manuscript.
MJ: Conceptualization, supervision, and review and editing of the manuscript.
SKL: Supervision and review and editing of the manuscript.
MDI: Echocardiographic assessment, supervision, and review and editing of the manuscript.
ARH: Fistula assessment, supervision, and review and editing of the manuscript.

Competing interests
The authors declare that they have no competing interests.

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Availability of data and materials
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request, subject to institutional ethical requirements.

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Figures

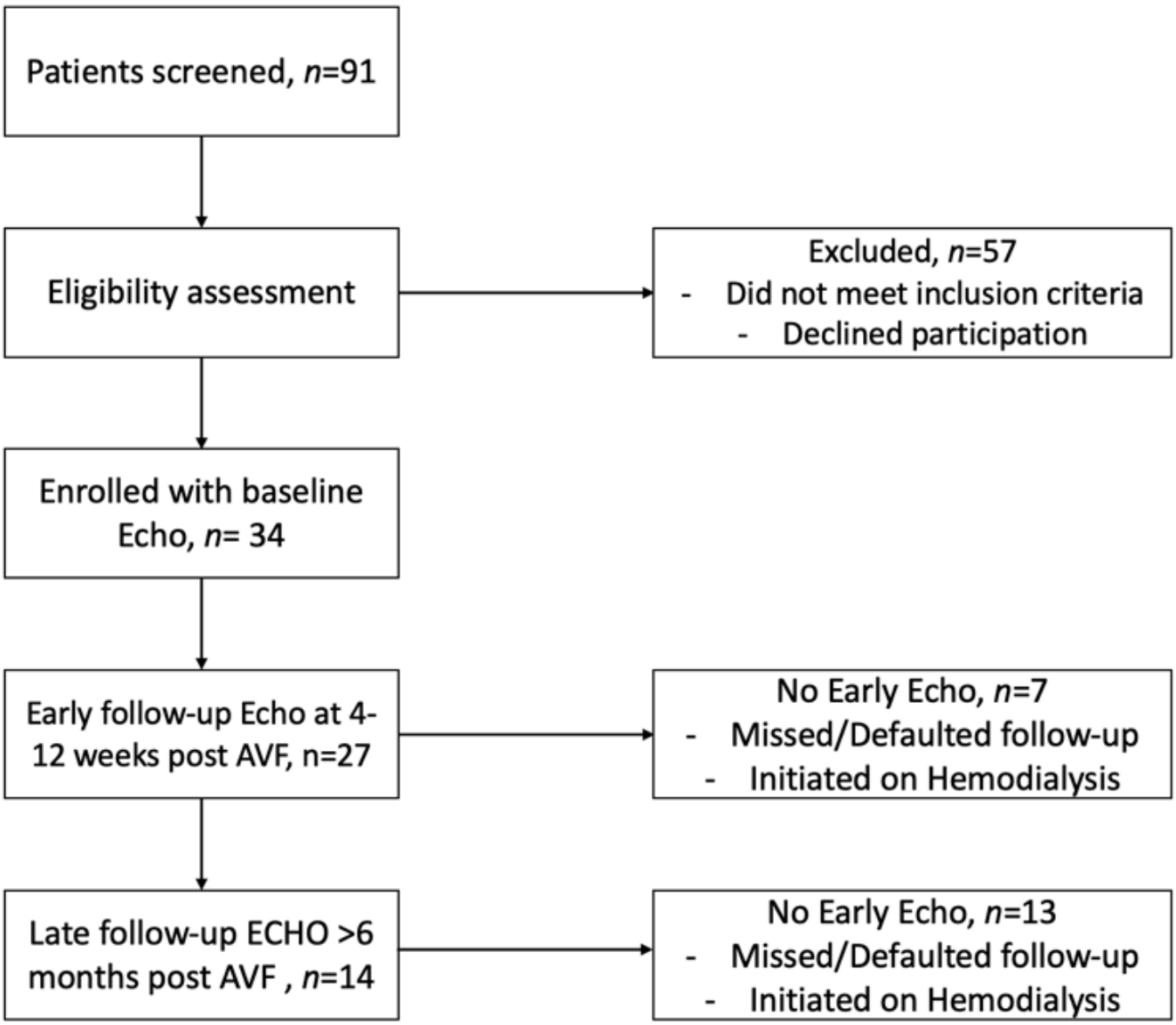


Figure 1

Flow diagram of patient screening, enrolment, and echocardiographic follow-up.

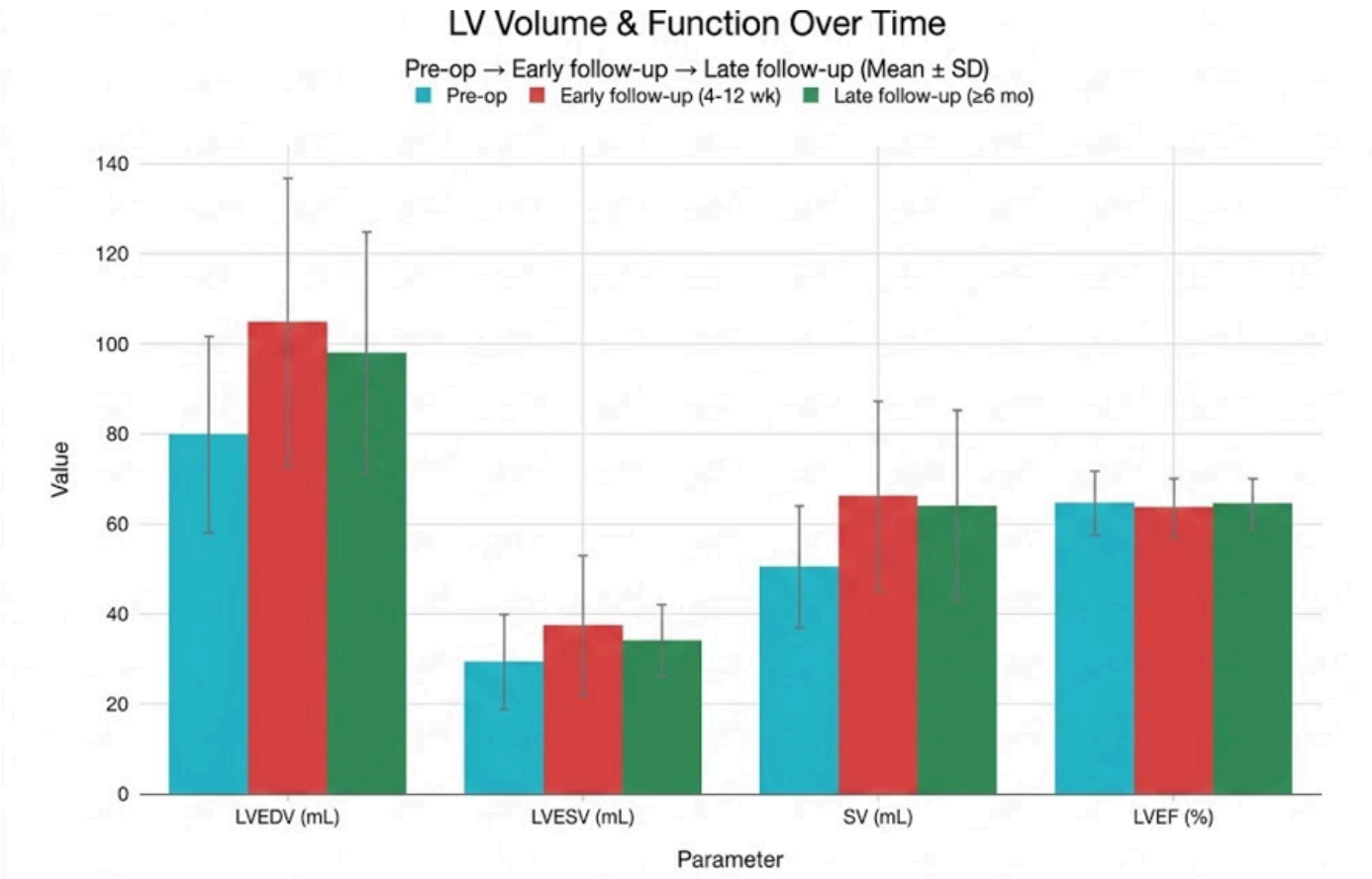
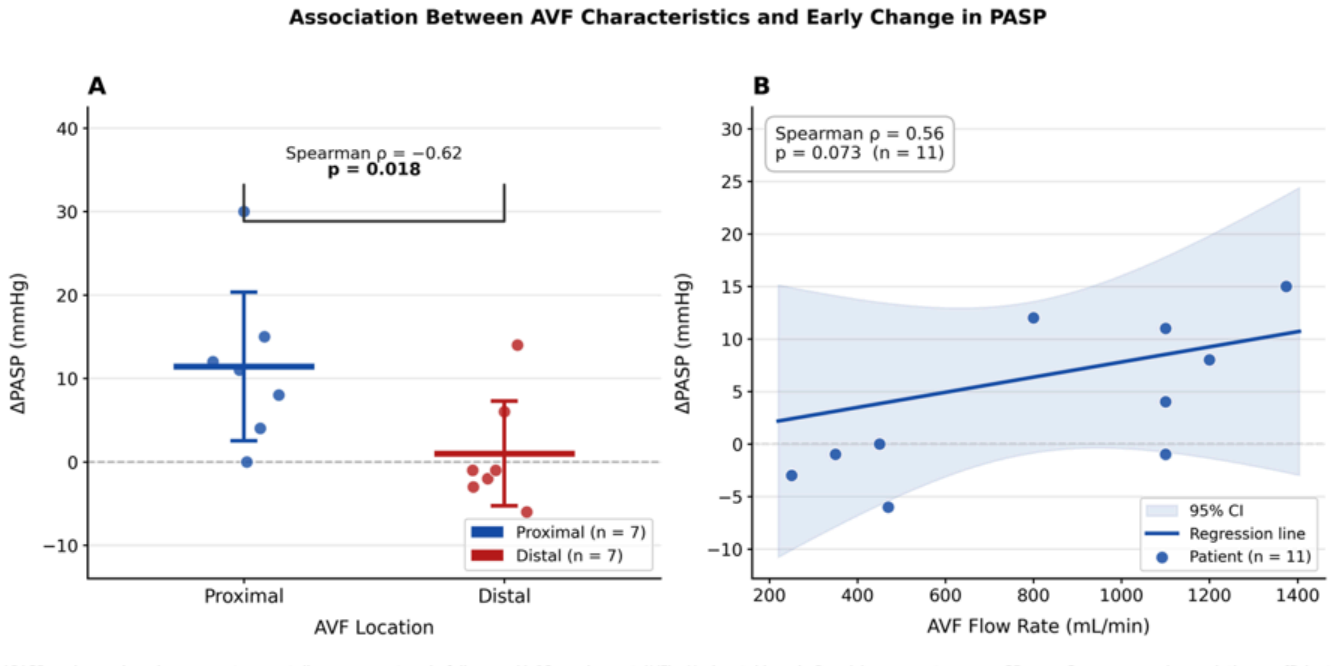


Figure 2

LV Volume and Function at baseline, and following AVF creation



ΔPASP = change in pulmonary artery systolic pressure at early follow-up (4–12 weeks post-AVF). Horizontal bars in Panel A represent mean ± SD. ρ = Spearman rank correlation coefficient.

Figure 3

Association between AVF characteristics and early change in pulmonary artery systolic pressure (PASP).

(A) Individual patient ΔPASP values at early follow-up (4–12 weeks) stratified by AVF proximity. Horizontal bars represent mean ± SD. Proximal AVFs were associated with greater increases in PASP, although interpretation is limited by small subgroup size (Spearman ρ = −0.62, p = 0.018, n = 14).
(B) Scatter plot of AVF access flow rate against ΔPASP, with linear regression line and 95% confidence interval. A positive trend was observed but did not reach statistical significance (Spearman ρ = 0.56, p = 0.073, n = 11).
ΔPASP = change in pulmonary artery systolic pressure from baseline. ρ = Spearman rank correlation coefficient.