

Cardiovascular Magnetic Resonance Pulmonary Artery Peak Flow Velocity as a Marker of Pulmonary Vascular Resistance in Fibrotic Interstitial Lung Disease

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Abstract

Background. Pulmonary hypertension (PH) is a common and severe complication of interstitial lung disease (ILD), yet early diagnosis remains challenging. Cardiovascular magnetic resonance (CMR) offers non-invasive assessment of pulmonary vascular hemodynamics, but evidence in PH associated with ILD is limited. This study aimed to evaluate the relationship between CMR-derived parameters and invasive hemodynamics in patients with fibrotic ILD.

Methods. In this prospective observational study, 41 patients with fibrotic ILD underwent pulmonary function testing, cardiopulmonary exercise testing, transthoracic echocardiography, and CMR. Twenty patients additionally underwent RHC. CMR-derived measurements included ventricular function parameters, heart chamber and great vessel dimensions, and pulmonary artery flow hemodynamic metrics. Associations between CMR parameters, invasive hemodynamics, exercise capacity and symptom burden were assessed using correlation, regression, and receiver operating characteristic (ROC) analyses.

Results. Among 20 patients who underwent RHC, PH was confirmed in all cases, including 15 with isolated precapillary PH and 5 with combined PH. Pulmonary vascular resistance (PVR) of over 5 Wood units defined severe PH in 6 patients who met the criteria. Pulmonary artery peak flow velocity (PA-Vmax) measured on CMR was significantly higher in the non-severe PH group (81.15 ± 19.27 cm/s vs. 52.33 ± 5.13 cm/s, $p < 0.001$) and demonstrated promising discriminatory performance for identifying severe PH (AUC 0.923, 95% CI 0.778-1.000, $p = 0.004$), with a threshold of < 62.5 cm/s yielding 100% sensitivity and 92.3% specificity. An inverse-transformed PA-Vmax model demonstrated a strong association with invasive PVR ($R^2 = 0.651$, $F = 31.65$, $p < 0.001$). Higher PA-Vmax correlated with greater exercise capacity, including higher anaerobic threshold ($r = 0.419$, $p = 0.015$) and maximal workload ($r = 0.329$, $p = 0.050$). Lower CMR-derived TAPSE was independently associated with greater dyspnea severity ($\beta = -0.426$, $p = 0.025$), while CMR-TAPSE/RHC-mPAP ratio demonstrated an even stronger association with symptom burden ($r_s = -0.570$, $p = 0.021$).

Conclusions. In patients with fibrotic ILD, CMR-derived PA-Vmax was significantly associated with PVR measured by RHC and demonstrated promising discrimination for severe PH in this exploratory cohort. CMR measurements of right ventricular adaptation were associated with functional capacity and dyspnea severity. These findings support further evaluation of CMR as a promising non-invasive modality for pulmonary vascular assessment in fibrotic ILD patients undergoing evaluation for suspected PH.

Background

Pulmonary hypertension (PH) is a frequent complication of fibrotic interstitial lung disease (ILD) and is associated with reduced exercise capacity, impaired quality of life, and increased mortality (1, 2, 3).

However, early recognition remains challenging because symptoms of both conditions frequently overlap, limiting diagnostic discrimination based on symptoms alone (4).

Transthoracic echocardiography (TTE) remains the routine initial non-invasive screening modality for PH (5). Nonetheless, its diagnostic accuracy is often limited in patients with ILD, where fibrotic parenchymal changes and pulmonary hyperinflation degrade acoustic windows (6). Consequently, although right heart catheterization (RHC) remains the gold standard for confirming PH, its invasive nature, procedural burden, and restricted applicability in frail respiratory populations limit its use as a universal screening or monitoring strategy.

Cardiovascular magnetic resonance (CMR) has emerged as a precise, reproducible, operator-independent imaging modality capable of detailed hemodynamic profiling. Phase-contrast techniques permit quantitative evaluation of pulmonary artery (PA) flow characteristics that may reflect pulmonary vascular remodeling and altered vascular compliance. Previous studies have demonstrated correlations between CMR-derived flow parameters and invasive hemodynamic measurements in PH populations (7). However, data specifically addressing fibrotic PH associated with ILD remain limited. We therefore conducted a prospective observational study in patients with fibrotic ILD to evaluate the relationship between CMR-derived pulmonary vascular parameters and invasive hemodynamics. We hypothesized that CMR PA flow metrics would correlate with RHC-measured PVR.

Methods

We conducted a prospective observational study at our Pulmonary hypertension referral center in Vilnius between 2023 and 2025. Forty-one consecutive patients with fibrotic ILD were enrolled. The diagnosis of ILD was established by a multidisciplinary team (MDT) based on clinical evaluation, chest computed tomography (CT) findings, and when available, histopathological findings. Despite differing ILD subtypes, all participants demonstrated a broadly comparable extent of pulmonary fibrosis on chest CT.

At study inclusion, 30 patients (73.2%) were receiving antifibrotic therapy, and none were treated with pulmonary vasodilators.

The study was approved by our ethics committee, and all participants provided written informed consent.

Patients underwent the following testing:

- Symptom assessment: Dyspnea severity was evaluated using the Modified Medical Research Council (mMRC) dyspnea scale, a validated 5-point scale (grades 0–4) rating breathlessness during daily physical activities, with higher scores indicating greater functional impairment.
- Pulmonary function tests (PFTs): Performed on room air and interpreted in accordance with the ERS/ATS standards (8). Recorded parameters included: forced vital capacity (FVC), total lung

capacity (TLC), vital capacity (VC), residual volume (RV), and single-breath diffusing capacity of the lung for carbon monoxide (DLCO).

- Cardiopulmonary exercise test (CPET): CPET was conducted on a cycle ergometer with breath-by-breath gas analysis. Recorded metrics included peak oxygen uptake ($\text{VO}_{2\text{peak}}$), anaerobic threshold (AT), peak work rate (W_{peak}), and VE/VCO_2 slope.
- TTE: Performed by a single expert using a comprehensive protocol. Echocardiographic markers of PH included tricuspid regurgitation velocity (TRV), left and right atrial and ventricular dimensions, left and right ventricular systolic function, inferior vena cava diameter and collapsibility. An echocardiographic suspicion of PH was defined as $\text{TRV} > 2.8 \text{ m/s}$ and/or presence of secondary echocardiographic signs suggestive of elevated pulmonary pressure (1).
- CMR. Examinations were performed on a 1.5-T Philips Ingenia Ambition scanner (Philips Healthcare, Best, the Netherlands). The protocol comprised breath-hold cine imaging (28 phases per cardiac cycle) in two-, three- and four-chamber, right ventricular outflow tract and short-axis orientations, and breath-hold through-plane phase-contrast imaging of the main PA (velocity encoding 160 cm/s) (Figs. 1 and 2). Gadobutrol (Gadovist, Bayer, Leverkusen, Germany) was administered at a dose of 0.15 mmol/kg body weight, and native and post-contrast T1 mapping, T2 mapping, contrast-enhanced pulmonary MR angiography and late gadolinium enhancement imaging were additionally acquired. Images were analyzed using Philips IntelliSpace Portal.

The following CMR parameters were recorded:

- Ventricular and atrial function and volumes: right (RV) and left ventricular (LV) ejection fraction (EF), stroke volume (SV), cardiac output (CO), end-diastolic volume (EDV) and end-systolic volume (ESV). Volumes were indexed to body surface area (EDVI, ESVI, SVI).
- Longitudinal and structural metrics: tricuspid annular plane systolic excursion (TAPSE), LV and RV diameters, left (LA) and right atrial (RA) diameters and areas, and the interventricular septal angle. TAPSE was measured on four-chamber cine images as the point-to-point displacement of the lateral tricuspid annulus between end-diastole and end-systole; because CMR-derived and echocardiographic TAPSE are not interchangeable, echocardiographic cut-off values were not applied.
- Vascular and flow hemodynamics: PA peak flow velocity (PA-Vmax) and PA antegrade and retrograde flow volumes.
- Great vessel dimensions: aortic diameter at multiple levels and main PA trunk diameter.
- Some CMR measurements were unavailable in individual patients because of suboptimal image quality or technical limitations during post-processing.
- RHC: Performed only in a part of our patients, selected by multidisciplinary PH team consensus. The decision to proceed with invasive testing was reserved for cases in which clinical suspicion of PH was high and the hemodynamic data had the potential to influence therapeutic management. Only clinically stable (non-frail) patients who provided informed consent underwent RHC. Precapillary PH

was confirmed with RHC using 2022 ESC/ERS guidelines (1). The diagnostic criteria were defined as: mean pulmonary arterial pressure (mPAP) > 20 mmHg and pulmonary vascular resistance (PVR) > 2 Wood units (WU). To capture the full hemodynamic spectrum of this population, both isolated precapillary PH and combined pre- and post-capillary PH phenotypes were included in the final analysis. Severe PH was defined by a PVR > 5 WU (1) (9). The average interval between CMR and RHC was 2–3 months; no major clinical deterioration, hospitalization, or treatment escalation occurred during this interval.

Our primary endpoint was the association between CMR-derived parameters and RHC findings. The secondary endpoint was the association between CMR-derived metrics and symptoms: dyspnea severity and functional limitation.

Statistical analysis. Analyses were performed using IBM SPSS version 27. Normality of the data was assessed using the Shapiro-Wilk test. Continuous variables are presented as mean ± SD, and categorical variables are presented as frequencies and percentages. Group comparisons were conducted using Student's t-test or Mann-Whitney U test, as appropriate. Correlational analyses were performed using Pearson's r or Spearman's rho (r_s) coefficients. Due to occasional missing values across CMR, TTE and functional tests, sample sizes vary across specific analyses; a pairwise deletion approach was used for bivariate correlations, while multivariate regression models utilized complete-case analysis. Receiver operating characteristic (ROC) curve analysis was used to assess diagnostic performance and determine the optimal clinical thresholds of PA-Vmax for detecting severe PH. Linear and logarithmic regression models were constructed to characterize the mathematical relationship between CMR PA-Vmax and invasive PVR. Multivariate linear regression was performed to assess whether CMR parameters independently predicted invasive hemodynamics when controlling for baseline lung function and age. Collinearity was evaluated using the variance inflation factor (VIF). Statistical outliers identified during exploratory analyses were intentionally retained in the final analysis to represent the full clinical spectrum and real-world heterogeneity of this ILD cohort. Statistical significance was defined as a two-sided p-value < 0.05.

Results

1. Baseline cohort characteristics.

The baseline clinical and functional characteristics of the study cohort (n = 41) are detailed in Table 1. Among the clinically selected subset of patients who underwent invasive assessment (n = 20), PH was confirmed by RHC in all catheterized patients (Table 2). 15 patients had isolated precapillary PH, while 5 had combined pre- and post-capillary PH (CpcPH). Within this catheterized sub-cohort, six patients met criteria for severe PH, defined as PVR > 5 Wood units.

Table 1. Patient population, baseline characteristics.

Patients, n = 41	
Age, years	70.90±8.94
Sex, male (%)	27 (66)
BMI, kg/m ²	28.56±5.09
NT-proBNP, ng/L	289.41±295.01
PFT measurements	
· FVC, %	89.05±18.60
· TLC, %	74.78±14.60
· VC, %	89.13±18.49
· DLCO, %	50.68±13.94
CPET measurements	
· Max performance, Watts	74.56±33.66
· Peak VO ₂ , % pred.	61.36±16.27
· VE/VCO ₂ slope	43.87±10.41
· Anaerobic threshold, L/min	0.91±0.27
ILD subtypes	
· IPF, n (%)	26 (63.4)
· Fibrotic sarcoidosis, n (%)	4 (9.8)
· CTD-associated-ILD, n (%)	4 (9.8)
· Hypersensitivity pneumonitis, n (%)	3 (7.3)
· NSIP, n (%)	2 (4.9)
· IPAF, n (%)	2 (4.9)
Dyspnea severity (mMRC)	
· Low-to-moderate burden (grades 0-2), n (%)	29 (70.7)
· Severe burden (grades 3-4), n (%)	12 (29.3)

Abbreviations: BMI – body mass index, CPET – cardiopulmonary exercise testing, CTD-associated-ILD – connective tissue disease-associated interstitial lung disease, DLCO – diffusing capacity of the lung for carbon monoxide, FVC – forced vital capacity, ILD – interstitial lung disease, IPAF – interstitial pneumonia with autoimmune features, IPF – idiopathic pulmonary fibrosis, mMRC – modified Medical

Research Council (dyspnea scale), NSIP – nonspecific interstitial pneumonia, NT-proBNP – N-terminal pro-B-type natriuretic peptide, PFT – pulmonary function test, TLC – total lung capacity, VC – vital capacity, VE/VCO2 – ventilator equivalent for carbon dioxide, VO2 – volume of oxygen uptake.

Table 2. Hemodynamic parameters derived from RHC.

RHC performed, n=20	
mPAP, mmHg	31.00±7.73
PAWP, mmHg	13.05±3.87
PVR, Wood units	4.60±1.89
CO, L/min.	4.40±1.18

Abbreviations: CO – cardiac output, mPAP – mean pulmonary artery pressure, PAWP – pulmonary artery wedge pressure, PVR – pulmonary vascular resistance, RHC – right heart catheterization.

Table 3. Comparison of CMR parameters between RHC confirmed non-severe and severe PH groups.

	Non-severe PH	Severe PH	P value
RV function and volumes			
TAPSE, mm	23.40±4.30 (n=10)	19.83±6.05 (n=6)	0.188
RV ejection fraction, %	51.69±8.13 (n=13)	48.69±13.53 (n=6)	0.486
RV end-diastolic volume, ml	166.23±49.77 (n=13)	150.50±25.85 (n=6)	0.480
RV end-systolic volume, ml	79.62±27.87 (n=13)	78.17±34.33 (n=6)	0.923
RV stroke volume, ml	86.23±29.84 (n=13)	71.00±18.14 (n=6)	0.268
RV cardiac output index, L/min/m ²	3.50±1.85 (n=11)	2.88±1.63 (n=6)	0.529
RV end-diastolic volume index, ml/m ²	83.77±26.01 (n=13)	78.67±12.55 (n=6)	0.657
RV end-systolic volume index, ml/m ²	38.69±16.53 (n=13)	40.67±13.09 (n=5)	0.801
RV stroke volume index, ml/m ²	44.15±14.82 (n=13)	42.92±14.82 (n=6)	0.868
Pulmonary artery flow hemodynamics			
PA-Vmax, cm/s	81.15±19.27 (n=13)	52.33±5.13 (n=6)	< 0.001
PA antegrade flow, ml	79.64±20.48 (n=14)	55.17±8.75 (n=6)	0.012
PA retrograde flow, ml	1.83±1.44 (n=14)	2.92±3.10 (n=6)	0.289
Pulmonary valve pressure gradient, mmHg	2.12±1.13 (n=8)	0.97±0.29 (n=4)	0.078
Cardiac chamber and great vessel dimensions			
RA area, cm ²	22.08±4.50 (n=13)	20.80±1.64 (n=5)	0.551
LA area, cm ²	21.69±6.06 (n=13)	20.50±4.93 (n=6)	0.680
RV diameter, mm	34.43±9.52 (n=14)	33.40±4.62 (n=5)	0.822
LV end-systolic diameter, mm	29.31±6.52 (n=13)	32.17±8.23 (n=6)	0.424
LV end-diastolic diameter, mm	46.79±6.54 (n=14)	46.33±5.75 (n=6)	0.885
Interventricular septal thickness, mm	10.81±1.93 (n=14)	9.67±1.21 (n=6)	0.198
Main PA trunk diameter, mm	33.25±7.02 (n=12)	33.58±5.68 (n=6)	0.921
LV function and volumes			
LV ejection fraction, %	67.21±8.68 (n=14)	58.83±6.59 (n=6)	0.049
LV end-diastolic volume, ml	131.21±42.23 (n=14)	135.00±27.51 (n=6)	0.843

LV end-systolic volume, ml	45.29±26.59 (n=14)	57.83±19.58 (n=6)	0.314
LV stroke volume, ml	87.08±20.19 (n=13)	73.67±14.50 (n=6)	0.164
LV end-diastolic volume index, ml/m ²	66.43±22.18 (n=14)	70.50±12.47 (n=6)	0.681
LV end-systolic volume index, ml/m ²	23.22±13.93 (n=14)	30.00±7.92 (n=6)	0.276
LV stroke volume index, ml/m ²	41.00±13.18 (n=14)	40.40±7.29 (n=6)	0.932
LV cardiac output index	3.27±1.03 (n=14)	3.50±1.62 (n=6)	0.705
Septal angle, degrees	152.86±9.42 (n=7)	150.40±4.34 (n=5)	0.602

Note: Data are presented as mean ± standard deviation. Sample sizes (n) vary per row parameter due to variable missing tracking values resulting from technical artifacts or poor geometric alignment during post-processing.

Abbreviations: LA – left atrium, LV – left ventricle, PA – pulmonary artery, PA-Vmax – pulmonary artery peak flow velocity, PH – pulmonary hypertension, RA – right atrium, RV – right ventricle, TAPSE – tricuspid annular plane systolic excursion.

2. Associations between CMR pulmonary vascular measurements and invasive hemodynamics

Differences in CMR measurements between RHC confirmed PH severity groups are outlined in Table 3. CMR-derived PA-Vmax was significantly higher in the non-severe PH group (81.15±19.27 cm/s, n=13) compared to the severe PH group (52.33±5.13 cm/s, n=6; $p < 0.001$). ROC analysis showed that CMR-derived PA-Vmax had promising discriminatory performance for identifying severe PH, yielding an AUC of 0.923 (95% CI: 0.778-1.000; $p=0.004$) (Figure 3). In this exploratory cohort, a threshold of <62.5 cm/s provided an optimal balance of 100% sensitivity and 92.3% specificity for severe PH detection.

To further evaluate the relationship between CMR and invasive hemodynamics, linear regression analysis was performed. Given the expected inverse physiological relationship between pulmonary flow velocity and vascular resistance, inverse-transformed PA-Vmax was additionally modeled to characterize its relationship with PVR. The transformed model demonstrated a significant association with RHC-measured PVR ($R^2=0.651$, $F=31.65$, $p < 0.001$) (Figure 4).

A multiple linear regression model was constructed using complete-case analysis (n=19) to evaluate whether the relationship between CMR PA-Vmax and RHC-measured PVR was independent of baseline lung function and age (Supplementary table S1). One patient was excluded from this model due to missing CMR flow parameter. The overall model was significant ($F(3, 15) = 9.246$, $p=0.001$) and accounted for 64.9% of the variance in invasive PVR ($R^2=0.649$). When adjusting for DLCO and age, CMR-derived PA-Vmax emerged as the only significant independent predictor of PVR ($\beta=-0.976$, $t=-3.884$, $p=0.001$). Multicollinearity was within acceptable limits, with all predictor VIF values remaining below 2.7.

Notably, LV function was modestly but significantly lower in the severe PH group, with CMR-derived LVEF being significantly lower in severe PH compared to the non-severe PH patients ($58.83 \pm 6.59\%$ vs. $67.21 \pm 8.68\%$, $p = 0.049$).

Bivariate correlation analysis additionally demonstrated a moderate positive association between CMR-derived PA-Vmax and TTE-measured peak pulmonary valve gradient (PAV-Gmax) ($r=0.426$, $n=26$, $p=0.030$).

PA-Vmax did not differ significantly between isolated precapillary PH (72.21 ± 21.60 cm/s, $n=14$) and CpcPH phenotype (71.60 ± 21.96 cm/s, $n=5$, $p=0.957$).

3. CMR measurements and symptoms – functional limitation

Bivariate correlation analysis across the entire study population ($n=41$, including the patients who did not undergo RHC) showed that higher PA flow velocities were consistently linked to better functional outcomes. Specifically, higher CMR-derived PA-Vmax demonstrated a significant moderate positive correlation with a patient's gas exchange capacity (DLCO % pred., $r=0.395$, $n=37$, $p=0.015$). Furthermore, higher PA-Vmax values correlated significantly with greater exercise capacity on CPET, including higher anaerobic threshold ($r=0.419$, $n=33$, $p=0.015$) and a higher maximal workload performance ($r=0.329$, $n=36$, $p=0.050$).

A moderate negative correlation was found between CMR-derived TAPSE and mMRC scores ($r_s = -0.412$, $p=0.017$) (Figure 5). In a multivariate linear regression model utilizing complete-case analysis ($n=33$), the combined clinical model significantly predicted dyspnea severity ($R^2=0.301$, $p=0.035$). TAPSE remained significantly associated with dyspnea severity ($\beta=-0.426$, $p=0.025$) after adjusting for pulmonary gas exchange (DLCO% pred.), age, and BMI.

When integrated with RHC data, the RV-arterial coupling index (CMR-TAPSE/RHC-mPAP ratio) demonstrated a superior correlation with symptomatic burden ($r_s=-0.570$, $p=0.021$) compared to TAPSE alone.

Discussion

In this prospective cohort of patients with fibrotic ILD, CMR-derived pulmonary vascular flow parameters demonstrated significant associations with invasive hemodynamic measurements and clinical symptom burden. Among the evaluated imaging metrics, PA-Vmax showed the strongest relationship with invasive PVR and demonstrated promising discriminatory performance for identifying severe PH.

Pulmonary vascular remodeling in fibrotic ILD is characterized by destruction of the pulmonary capillary bed, vascular pruning, endothelial dysfunction, and progressive loss of arterial compliance (10). These processes increase downstream resistance and impair forward pulmonary arterial flow, resulting in

reduced peak systolic PA velocity. Our findings suggest that PA-Vmax may reflect these hemodynamic alterations non-invasively and may therefore provide indirect insight into PVR in this population.

This physiological mechanism is supported by recent 4D-flow CMR data from Kim et al. (11), who demonstrated that elevated PVR reduces main PA peak velocity and alters vascular wall stress. Our findings suggest that a similar relationship may exist in fibrotic ILD. Beyond peak flow velocity, CMR-derived PA antegrade stroke volume was also significantly reduced in patients with severe PH. This parallel reduction is physiologically expected: as downstream PVR increases, elevated RV afterload restricts total forward stroke volume per cardiac cycle alongside lowering peak systolic flow velocity.

Importantly, PA-Vmax also demonstrated promising discriminatory performance for severe PH. Although identified threshold values should be interpreted cautiously due to the limited sample size, the observed reduction in PA-Vmax among patients with severe PH supports the physiological plausibility of this parameter. Furthermore, the inverse-transformed PA-Vmax model showed a strong association with invasive PVR, supporting the expected relationship between pulmonary flow velocity and vascular resistance. Crucially, the relationship between PA-Vmax and invasive PVR remained significant after adjusting for age and lung function (DLCO). Together, these findings suggest that phase-contrast CMR may provide clinically relevant pulmonary vascular information that complements conventional markers of RV remodeling.

Our findings are consistent with previous CMR flow-imaging literature in PH populations. A comprehensive systematic review by Alghamdi et al. demonstrated that phase-contrast CMR-derived velocity metrics consistently correlate with RHC-derived measurements (12). However, fibrotic ILD populations remain underrepresented in existing imaging studies despite presenting particular diagnostic challenges for TTE assessment. In this context, our study extends the existing literature by focusing on pulmonary vascular disease associated with fibrotic ILD.

Conventional RV macro-structural dimensions and volumes did not differ significantly between groups. Although LV systolic function was lower in the severe PH group, mean values remained within normal limits in both groups. The clinical significance of this finding is uncertain and should be interpreted with caution given the small sample size. It may reflect the clinical reality of multifactorial PH in the elderly ILD population: patients with advanced fibrotic ILD often present with a combined PH phenotype, where precapillary pulmonary vascular disease coexists with chronic, age-related left-heart pathologies. Nevertheless, PA-Vmax did not differ significantly between isolated precapillary PH and combined PH phenotypes in our cohort.

Beyond pulmonary vascular flow metrics, our data additionally emphasize the clinical relevance of RV functional adaptation (13). We observed that reduced CMR-derived TAPSE was associated with greater dyspnea severity independently of DLCO, age, and BMI. This finding supports the concept that symptomatic limitation in fibrotic ILD may not solely reflect restrictive ventilatory impairment, but may also be influenced by progressive RV-pulmonary vascular uncoupling (14). As pulmonary vascular resistance increases, the RV may progressively lose the ability to maintain efficient ventriculo-arterial

interaction, resulting in reduced forward cardiac reserve during exertion and worsening functional limitation (15). The clinical consequence of this hemodynamic failure is underscored by a study from Japan, demonstrating that alterations in pulmonary hemodynamics quantified by CMR are independent predictors of short-term mortality in ILD patients (16).

Furthermore, the combined RV-arterial coupling index (CMR-TAPSE/RHC-mPAP ratio) demonstrated a stronger association with symptom burden than CMR-TAPSE alone in our cohort. This observation aligns with emerging evidence suggesting that load-adjusted RV functional metrics may better reflect cardiopulmonary adaptation than isolated ventricular performance (17).

Our findings suggest that CMR may provide additional value in fibrotic ILD patients. It should be noted that current expert consensus and standard clinical algorithms do not include CMR as part of routine screening for PH associated with ILD (18, 19). However, unlike TTE, CMR allows reproducible assessment of pulmonary flow dynamics, right heart adaptation, and vascular morphology without reliance on acoustic window quality. Nevertheless, CMR should currently be regarded as complementary to rather than a replacement for RHC, because invasive hemodynamic assessment remains essential for definitive diagnosis and treatment stratification.

Limitations

Our study has several limitations. First, this was a single-center study with a relatively small sample size, limiting statistical power and external generalizability. Second, RHC was performed only in clinically selected patients. Consequently, all catheterized patients fulfilled hemodynamic criteria for PH, and the present study was designed to evaluate associations between CMR parameters and PH severity rather than the diagnostic performance of CMR for detecting PH itself. Therefore, the findings should not be interpreted as evidence supporting CMR as a screening test for PH in unselected fibrotic ILD population. Third, the study population included a heterogeneous group of fibrotic lung diseases, and disease-specific analyses were not feasible due to the sample size. Fourth, no internal or external validation cohort was available. Fifth, the average interval between CMR and RHC was 2-3 months, which may have introduced temporal variability in measurements despite overall clinical stability during follow-up. Sixth, interobserver and intra-observer variability analyses of CMR-derived measurements were not performed, limiting assessment of measurement reliability and reproducibility within this cohort. Seventh, only six patients met criteria for severe PH, therefore, estimates of diagnostic performance and optimal threshold values are subject to substantial statistical uncertainty and should be interpreted cautiously. Finally, because of the observational and exploratory design, causality cannot be inferred and findings should be considered hypothesis-generating. The proposed PA-Vmax threshold of 62.5 cm/s was derived from the same exploratory cohort and was not externally validated, therefore it should not be considered a clinically established cut-off.

Conclusions

In patients with fibrotic ILD, CMR-derived PA-Vmax was associated with invasive PVR and demonstrated promising discriminatory performance for severe PH. Additional CMR metrics were associated with symptom burden. These findings support CMR as a promising non-invasive tool for pulmonary vascular and right ventricular assessment in patients with fibrotic ILD and warrant validation in larger cohorts. In particular, PA-Vmax showed a potential to differentiate severe from non-severe PH in fibrotic ILD, suggesting its possible value as a non-invasive marker of disease severity. These findings require validation in larger, independent cohorts.

Declarations

Ethics approval and consent to participate. The study was conducted in accordance with the World Medical Association Declaration of Helsinki. Ethical approval was received from the Vilnius Regional Biomedical Research Ethics Committee (registered internally under the study identifier RL-PC-1.4 at the Vilnius university Hospital Santaros Klinikos, Biomedical Research Department). All the participants provided written informed consent prior to their inclusion in the study.

- Consent for publication. Not applicable.
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References

1. Humbert M, Kovacs G, Hoeper MM, Badagliacca R, Berger RMF, Brida M, et al. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J*. 2022;43(38):3618–731. 10.1093/eurheartj/ehac237.
2. Ang HL, Schulte M, Chan RK, Tan HH, Harrison A, Ryerson CJ, et al. Pulmonary Hypertension in Interstitial Lung Disease. *Chest*. 2024;166(4):778–92. 10.1016/j.chest.2024.04.025.

3. Nathan SD, Barbera JA, Gaine SP, Harari S, Martinez FJ, Olschewski H, et al. Pulmonary hypertension in chronic lung disease and hypoxia. *Eur Respir J*. 2019;53(1):1801914. 10.1183/13993003.01914-2018.
4. Haynes ZA, Chandel A, King CS. Pulmonary Hypertension in Interstitial Lung Disease: Updates in Disease, Diagnosis, and Therapeutics. *Cells*. 2023;12(19):2394. 10.3390/cells12192394.
5. Arvanitaki A, Diller GP, Gatzoulis MA, McCabe C, Price LC, Wort SJ. Noninvasive diagnostic modalities and prediction models for detecting pulmonary hypertension associated with interstitial lung disease: a narrative review. *Eur Respir Rev*. 2024;33(174):240092. 10.1183/16000617.0092-2024.
6. Jose A, Sood N, Elwing JM, Akkanti B, Bajwa A, Bernardo R, et al. Pulmonary Hypertension Associated With Interstitial Lung Diseases. *Chest*. 2026;169(1):220–9. 10.1016/j.chest.2025.07.4107.
7. Bradlow WM, Gibbs R, Mohiaddin JS. Cardiovascular magnetic resonance in pulmonary hypertension. *J Cardiovasc Magn Reson*. 2012;14(1):6. 10.1186/1532-429. X-14-6 PubMed PMID: 22257586; PubMed Central PMCID: PMC3305675.
8. Stanojevic S, Kaminsky DA, Miller MR, Thompson B, Aliverti A, Barjaktarevic I, et al. ERS/ATS technical standard on interpretive strategies for routine lung function tests. *Eur Respir J*. 2022;60(1):2101499. 10.1183/13993003.01499-2021.
9. Zeder K, Avian A, Bachmaier G, Douschan P, Foris V, Sassmann T, et al. Elevated pulmonary vascular resistance predicts mortality in COPD patients. *Eur Respir J*. 2021;58(2):2100944. 10.1183/13993003.00944-2021.
10. Patel NM, Lederer DJ, Borczuk AC, Kawut SM. Pulmonary Hypertension in Idiopathic Pulmonary Fibrosis. *Chest*. 2007;132(3):998–1006. 10.1378/chest.06-3087.
11. Kim B, Lee J, Berhane H, Freed BH, Shah SJ, Thomas JD. Differences in Pulmonary Artery Flow Hemodynamics Between PAH and PH-HFpEF: Insights From 4D-Flow CMR. *Pulm circ*. 2025;15(1):e70022. 10.1002/pul2.70022.
12. Alghamdi KS, Maiter A, Hyde GA, Alnasser T, Sharkey M, Salehi M, et al. Pulmonary hypertension and the role of MRI flow assessment: a systematic review. *Br J Radiol*. 2025;98(1175):1938–46. 10.1093/bjr/tqaf182 . PubMed PMID: 40711861; PubMed Central PMCID: PMC12659743.
13. Wessels JN, Celant LR, de Man FS, Vonk Noordegraaf A. The Right Ventricle in Pulmonary Hypertension. *Semin Respir Crit Care Med*. 2023;44(6):738–45. 10.1055/s-0043-1770117 . PubMed PMID: 37487527.
14. Vonk-Noordegraaf A, Haddad F, Chin KM, Forfia PR, Kawut SM, Lumens J, et al. Right heart adaptation to pulmonary arterial hypertension: physiology and pathobiology. *J Am Coll Cardiol*. 2013;62(25 Suppl):D22–33. 10.1016/j.jacc.2013.10.027 . PubMed PMID: 24355638.
15. Tello K, Gall H, Richter M, Ghofrani A, Schermuly R. Right ventricular function in pulmonary (arterial) hypertension. *Herz*. 2019;44(6):509–16. 10.1007/s00059-019-4815-6 . PubMed PMID: 31101945.

16. Tsuchiya N, Iwasawa T, Ogura T, Yamashiro T, Yara S, Fujita J, et al. Pulmonary flow assessment by phase-contrast MRI can predict short-term mortality of fibrosing interstitial lung diseases. *Acta Radiol.* 2020;61(10):1350–8. doi:10.1177/0284185120901503 PubMed PMID: 32028775.
17. Tello K, Wan J, Dalmer A, Vanderpool R, Ghofrani HA, Naeije R, et al. Validation of the Tricuspid Annular Plane Systolic Excursion/Systolic Pulmonary Artery Pressure Ratio for the Assessment of Right Ventricular-Arterial Coupling in Severe Pulmonary Hypertension. *Circ Cardiovasc Imaging.* 2019;12(9):e009047. 10.1161/CIRCIMAGING.119.009047 . PubMed PMID: 31500448; PubMed Central PMCID: PMC7099862.
18. Haynes ZA, Chandel A, King CS. Pulmonary Hypertension in Interstitial Lung Disease: Updates in Disease, Diagnosis, and Therapeutics. *Cells.* 2023;12(19):2394. doi:10.3390/cells12192394 PubMed PMID: 37830608; PubMed Central PMCID: PMC10572438.
19. Rahaghi FF, Kolaitis NA, Adegunsoye A, de Andrade JA, Flaherty KR, Lancaster LH, et al. Screening Strategies for Pulmonary Hypertension in Patients With Interstitial Lung Disease. *Chest.* 2022;162(1):145–55. 10.1016/j.chest.2022.02.012 . PubMed PMID: 35176276; PubMed Central PMCID: PMC9993339.
20. Supplementary. table S1. Multiple linear regression for predicting invasive PVR (n = 19).

Figures

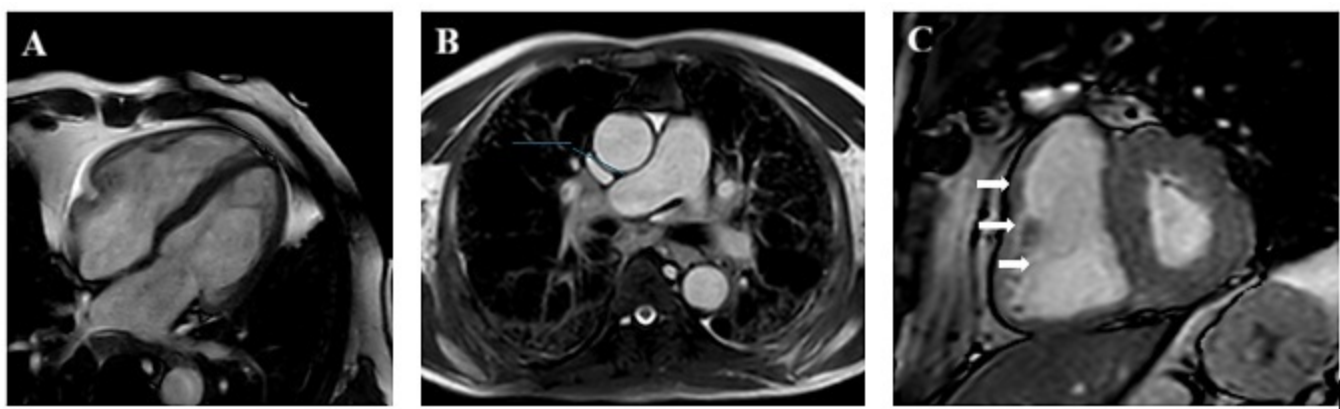


Figure 1

72-year-old man with histologically confirmed fibrotic hypersensitivity pneumonitis. (A) Four-chamber cine image showing moderate right ventricular (RV) enlargement, with the RV exceeding the left ventricle (LV) in size. (B) Axial single-shot image showing a dilated main pulmonary artery (PA, 36 mm) with a PA diameter exceeding that of the ascending aorta (35 mm; PA:Ao ratio > 1). (C) Short-axis cine image in systole showing flattening of the interventricular septum (arrows) and a resulting D-shaped left ventricle.

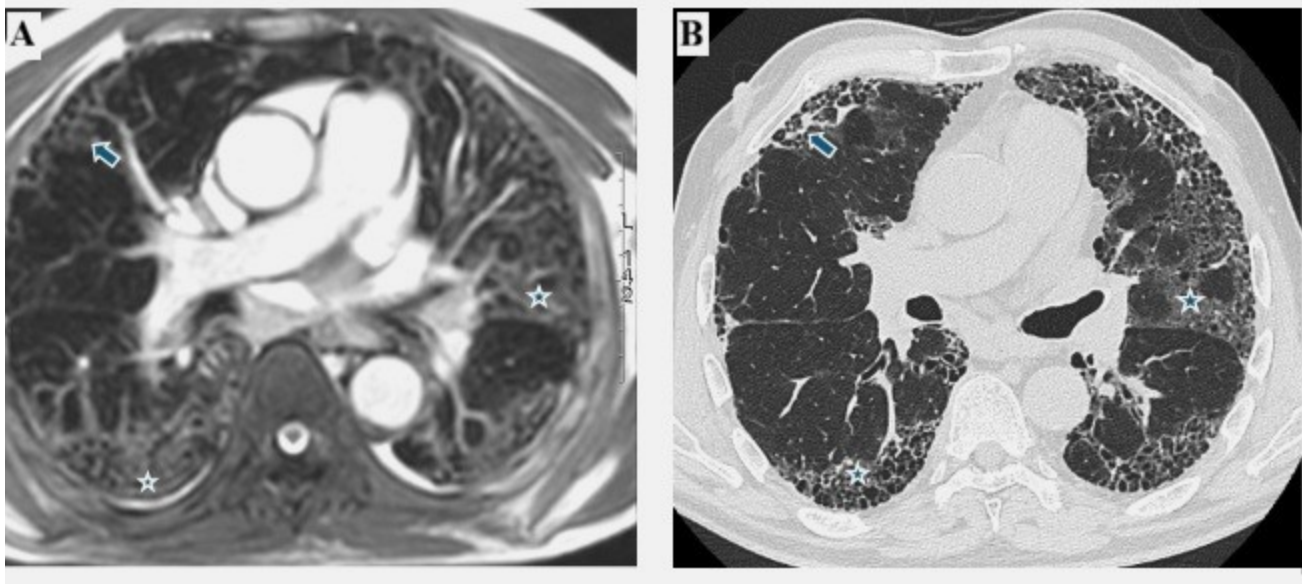


Figure 2

Same patient as in Figure 1. (A) Axial single-shot MR image and (B) axial unenhanced chest CT image (lung window) showing findings consistent with fibrotic hypersensitivity pneumonitis: ground-glass opacity with superimposed reticulation (asterisk in A and B) and honeycombing (arrow in A and B). The extent of parenchymal fibrosis is also appreciable on CMR, illustrating the context in which pulmonary vascular assessment was performed.

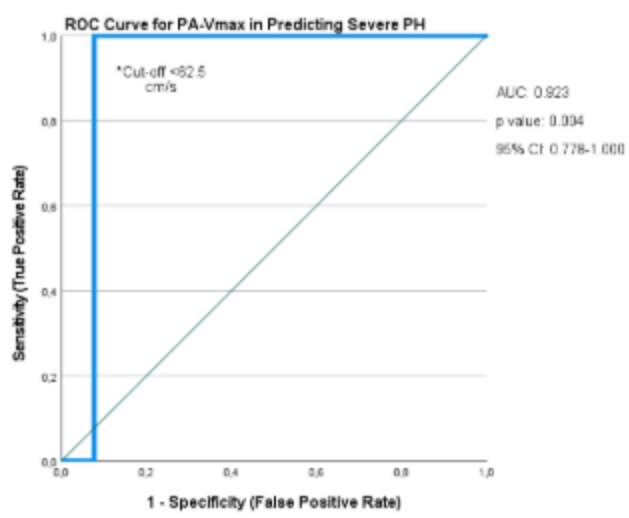


Figure 3

Diagnostic performance of CMR-derived PA-Vmax in identifying severe PH associated with ILD. ROC curve illustrating the ability of PA-Vmax to distinguish between non-severe and severe PH in patients with ILD. The AUC is 0.923 (95% CI: 0.778-1.000, $p=0.004$), demonstrating promising discriminatory performance. A PA-Vmax threshold of <62.5 cm/s provides an optimal balance of sensitivity and specificity for severe PH detection.

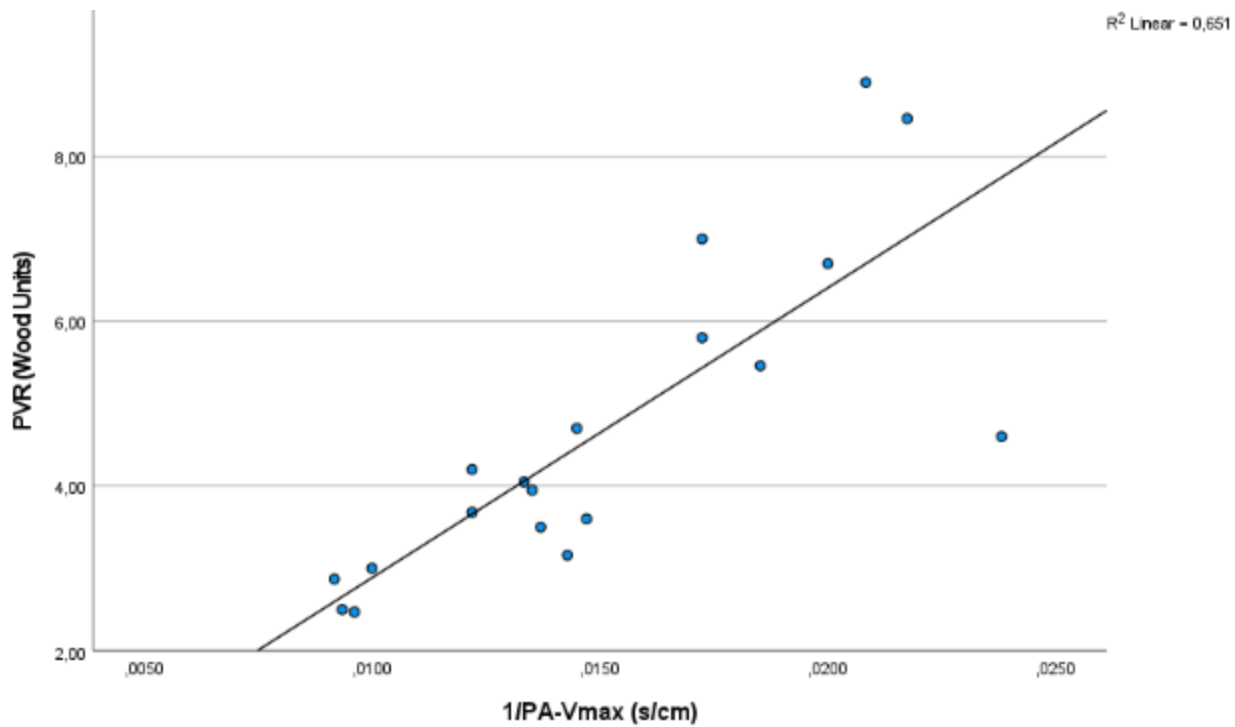


Figure 4

Scatter plot demonstrating the relationship between the inverse of CMR-derived PA-Vmax in cm/s (1/PA-Vmax, x-axis) and RHC-measured PVR in Wood units (y-axis). The linear model (solid line, $R^2=0.651$, $p < 0.001$) reveals that decreasing peak forward velocities closely track increasing PVR.

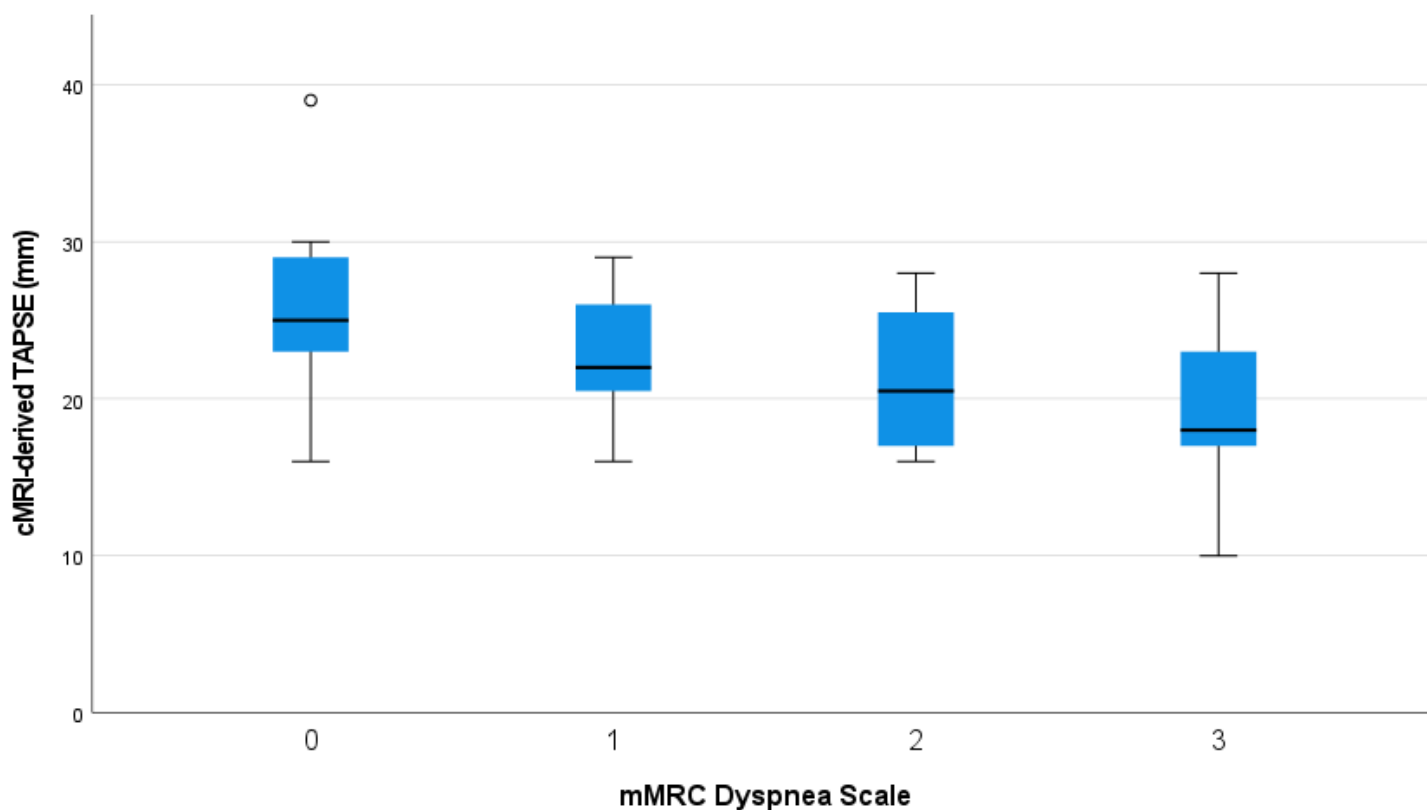


Figure 5

Boxplot representing the distribution of CMR-derived TAPSE values across mMRC dyspnea grades. A progressive decline in RV longitudinal function is observed with increasing symptomatic severity ($r_s = -0.412$, $p = 0.017$), highlighting the contribution of right heart hemodynamics to the functional limitations of ILD patients.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [SupplementarytableS1.docx](#)