ORIGINAL ARTICLE

Factors associated with the need for pulmonary valve replacement in asymptomatic patients with isolated pulmonary regurgitation after repair of tetralogy of Fallot

A cardiac magnetic resonance study

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ABSTRACT

BACKGROUND Pulmonary regurgitation (PR) is the most common late complication in patients after repair of tetralogy of Fallot (TOF). Most patients remain asymptomatic over years, but eventually, the compensatory mechanisms fail, leading to right ventricular (RV) dilation and dysfunction, limited exercise capacity, ventricular arrhythmia, and sudden death.

AIMS We aimed to evaluate associations between cardiac magnetic resonance (CMR) parameters and the need for either surgical or percutaneous pulmonary valve replacement (PVR) in asymptomatic patients with significant PR after repair of TOF.

METHODS Of 209 patients with repaired TOF who had undergone a CMR study, we selected 61 asymptomatic patients with moderate-to-severe PR and followed them for up to 4 years (mean [SD], 21.4 [13.7] months). We excluded patients with residual ventricular septal defect, a peak RV outflow tract gradient of 30 mm Hg or higher, or at least moderate tricuspid regurgitation.

RESULTS Receiver operating characteristic curve analyses revealed that the ratio of RV to left ventricular (LV) volume (RV / LV ratio; threshold >2.4) and PR fraction (PRF; threshold >33%) had acceptable discriminatory capacity to differentiate between patients requiring PVR and those treated conservatively. The Cox proportional hazards regression and the Kaplan–Meier curves revealed that the RV / LV ratio and PRF was significantly associated with the need for PVR. The combination of the RV / LV ratio and PRF provided significant discrimination in terms of survival without PVR (P <0.001; log-rank test for trend).

CONCLUSIONS The RV / LV ratio and PRF were significantly associated with the need for PVR in asymptomatic patients with isolated moderate-to-severe PR after repair of TOF.

INTRODUCTION Pulmonary regurgitation (PR) is the most common late complication in patients after repair of tetralogy of Fallot (TOF). Although the majority of patients remain asymptomatic for many years, the compensatory mechanisms eventually fail, leading to right ventricular (RV)

WHAT'S NEW?

We demonstrated that the ratio of right ventricular to left ventricular volume (RV/LV ratio) and pulmonary regurgitation fraction (PRF) are significantly associated with the need for pulmonary valve replacement in patients with isolated moderate-to-severe pulmonary regurgitation (PR) after the repair of tetralogy of Fallot during the mean follow-up of 21 months. The combination of the RV/LV ratio and PRF provided improved discrimination ability when compared with either of the parameters alone. Thus, patients can be stratified according to the threshold of the RV/LV ratio (>2.4) and PRF (>33%) into clinically useful groups, determining further management (conservative treatment or the need for pulmonary valve replacement or percutaneous pulmonary valve implantation).

dilation and dysfunction, limited exercise capacity, ventricular arrhythmia, and sudden death.1,2

The timing of pulmonary valve replacement (PVR), either percutaneous or surgical, in patients with repaired TOF remains controversial.1,2 While the need for PVR in symptomatic patients is not questionable, performing PVR in asymptomatic individuals raises concerns about the risk-to-benefit ratio. Since an ideal valve substitute is yet to be found, an early surgery exposes a patient to the need for reintervention in about 5 to 10 years after the index procedure, with a considerable number of patients experiencing early homograft failure.3-5 On the other hand, there are concerns that too long a delay leads to the point when RV dysfunction is irreversible and thus the benefits of late PVR become fairly limited.6-9

Cardiac magnetic resonance (CMR) imaging is considered the reference standard for the quantification of PR and the assessment of RV size and function.1,2,10 It would be of a particular clinical value to identify factors associated with the need for future PVR in asymptomatic patients with significant PR after the TOF repair. Accordingly, we aimed to evaluate an association between CMR parameters and the need for future PVR in this population.

METHODS

Study population Data from a high-volume tertiary adult congenital heart disease center were retrospectively analyzed. All patients after repair of TOF who had undergone CMR were identified. Patients with pulmonary atresia and ventricular septal defect were excluded to ensure homogeneity. Individuals with moderate-to-severe PR, as assessed by echocardiography, were selected. We also excluded symptomatic patients, individuals with a peak instantaneous RV outflow tract (RVOT) gradient of 30 mm Hg or higher, or those with residual ventricular septal defect. Additionally, patients with at least moderate tricuspid, aortic, or mitral regurgitation were excluded.

Patients were followed for up to 49 months. Each patient gave a written informed consent for the CMR imaging. The local Ethics Committee approved the analysis of medical records and computer databases. The study complied with the Declaration of Helsinki.

Cardiac magnetic resonance Cardiac magnetic resonance imaging was performed with the use of a 1.5T scanner (Avanto, Siemens, Erlangen, Germany). The size and function of the ventricles (RV and left ventricular [LV] end-diastolic volume [RVEDV and LVEDV, respectively], RV and LV end-systolic volume, RV and LV ejection fraction, as well as RV and LV mass) were calculated by the dedicated software (Mass 6.2.1, Medis, Leiden, the Netherlands) on the basis of a stack of balanced steady-state free precession cine images acquired from the base to the apex. All volume and mass parameters were indexed to the body surface area and expressed either in ml/m^2 or g/m^2. By dividing RVEDV by LVEDV, the ratio of RV to LV volume (RV/LV ratio) was calculated.11 The corrected RV ejection fraction was calculated by dividing the net pulmonary flow by RVEDV.12

Pulmonary flow was derived from a phase-sensitive gradient echo sequence with an imaging plane located in the mid-point of the main pulmonary artery or conduit perpendicularly to the vessel wall. The PR fraction (PRF = regurgitant volume / forward volume × 100%) and PR volume (PRV = regurgitant volume / body surface area) were also calculated.

Echocardiography Echocardiography imaging was performed with commercially available systems by physicians with experience in congenital heart diseases. The severity of PR was assessed by integrating the indices of severity.13 The maximum velocity across the RVOT was determined with continuous-wave Doppler imaging, and the peak instantaneous RVOT gradient was calculated using the Bernoulli equation.

Indications for pulmonary valve replacement The decision about performing PVR was made by consensus between a treating physician or physicians, a cardiac surgeon, and an interventional cardiologist, based on a comprehensive evaluation of patients, including clinical assessment, echocardiography, as well as CMR imaging, and in selected cases, also cardiopulmonary exercise testing and Holter monitoring. The following situations were considered as an indication for PVR: 1) development of symptoms; 2) reduced exercise capacity; 3) progressive or severe RV dilation; 4) moderate-to-severe RV dysfunction; and 5) sustained ventricular or atrial arrhythmias.1,2,14 Since the universal definitions of severe RV dilation and moderate-to-severe RV dysfunction are lacking, we did not use prespecified criteria for RV dilation and RV dysfunction. These were judged arbitrarily on the basis of available findings from imaging studies, and the judgement considered the cotemporary guidelines, recommendations, and our own experience.

Percutaneous treatment was offered to patients with an indication or indications for PVR and fulfilling criteria for percutaneous pulmonary valve implantation (PPVI), namely, those with a high probability of a suitable landing zone for the valve (based on noninvasive imaging, cardiac catheterization, or both) and no risk of coronary artery compression.15,16 The remaining individuals were referred for surgery. The presence of an RVOT patch per se was not a contraindication for PPVI.17

Statistical analysis All variables were presented as mean (SD) or as median (interquartile range), as appropriate. Normality was tested with the use of the Kolmogorov–Smirnov test. Continuous variables were compared using the *t* test or the Mann–Whitney test, where appropriate. Categorical variables were compared with the χ^2 test or Fisher exact test, as appropriate.

A receiver operating characteristic curve (ROC) analysis was used to test the ability of various parameters to identify patients requiring PVR. We selected parameters with acceptable discrimination (area under the curve [AUC] ≥0.7) and applied Cox proportional hazards regression to determine whether any of these parameters were independent predictors. Additionally, binary analyses were performed using the cutoff values derived from the ROC analysis.

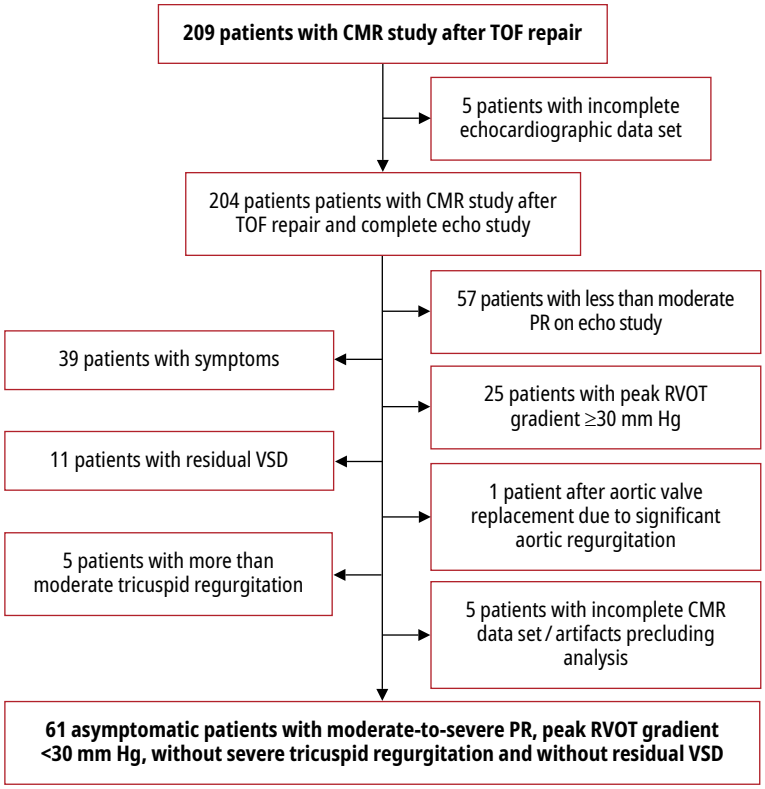

FIGURE 1 Flow chart of patient selection for the study.
Abbreviations: CMR, cardiac magnetic resonance; PR, pulmonary regurgitation; RVOT, right ventricular outflow tract; TOF, tetralogy of Fallot; VSD, ventricular septal defect

The Kaplan–Meier curves were constructed to compare the groups above and below the optimal ROC analysis–based threshold (parameters with acceptable discrimination in ROC analyses were used). Additionally, considering the previously published data indicating that the low likelihood of RV size normalization as soon as the RVEDV exceeded a certain threshold (>170 ml/m^2, >168 ml/m^2, >163 ml/m^2, and >160 ml/m^2),6-8 we generated the survival curves for the RVEDV at these threshold levels.

A 2-sided *P* value of less than 0.05 was considered significant. All analyses were performed with the MedCalc 12.1.4.0 software (MedCalc, Mariakerke, Belgium).

RESULTS A total of 209 patients after TOF repair underwent CMR during the study period. A flow chart of patient selection for the study is presented in FIGURE 1. The final analysis included 61 patients. The baseline characteristics of patients who were treated conservatively and those referred for PPVI or surgical PVR are presented in TABLE 1.

Pulmonary valve replacement or percutaneous pulmonary valve implantation During the follow-up, 25 patients (41%) were referred for either PPVI or surgical PVR due to the following reasons: development of symptoms (n = 7), reduced exercise capacity on cardiopulmonary exercise test (n = 13), progressive or severe RV dilation with impaired RV systolic function (n = 4), and sustained ventricular arrhythmias (n = 1). The PPVI was attempted in 14 patients: 1) in 9 patients, balloon sizing and cardiac catheterization revealed no suitable anchoring site and the valve implantation was abandoned; 2) in 2 patients, a bare metal stent was implanted with subsequent implantation of an Edwards SAPIEN valve (a 2-stage procedure); 3) 1 patient underwent stent implantation and Edwards SAPIEN valve implantation in a single procedure; 4) 1 patient underwent a Melody valve implantation with prestenting performed in a single procedure; 5) in 1 patient, prestenting was performed but the stent migrated into the branch pulmonary artery during the introduction of the valve into the RVOT, and the patient underwent surgical removal with a simultaneous homograft implantation. The remaining patients (n = 11) were referred for surgical treatment with homograft implantation. The mean (SD) time from CMR to the intervention was 21.4 (13.7) months.

Factors associated with the need for pulmonary valve replacement or percutaneous pulmonary valve implantation The ROC analyses revealed that the RV / LV ratio and PRF demonstrated an acceptable discriminatory capacity to differentiate between patients with the need for PVR or PPVI and those treated conservatively (TABLE 2). Neither RVEDV nor RVEF showed any

TABLE 1 Baseline characteristics of the study population

Parameter		All patients (n = 61)	Conservative treatment (n = 36; 59%)	PVR or PPVI (n = 25; 41%)	*P* value[a]
Male sex, n (%)		47 (77)	29 (81)	18 (72)	0.78
Age at CMR study, y, median (IQR)		22.5 (18.8–25.3)	24.3 (7.9)	22 (3.2)	0.11
Age at TOF repair, y, median (IQR)		3.3 (2.2–4.6)	3.6 (2.3–6)	3 (1.9–3.9)	0.09
Time between TOF repair and CMR study, y		18.7 (3.7)	18.5 (4)	19.0 (3.2)	0.57
Previous palliative shunt, n (%)		19 (31)	11 (31)	8 (32)	0.87
Type of TOF repair, n (%)	Patch	43 (71)	20 (55)	23 (92)	0.003
	Conduit	2 (3)	2 (6)	0	0.5
	Details unknown	16 (26)	14 (39)	2 (8)	0.01
RVEDV, ml/m^2		172.8 (38.6)	163.7 (30.5)	186.1 (45.3)	0.04
RVESV, ml/m^2		93.6 (26.8)	87.4 (23.4)	102.6 (29.3)	0.03
RVSV, ml/m^2		79.2 (16.2)	76.3 (13.7)	83.5 (18.8)	0.09
RVEF, %		46.3 (6)	47.2 (6.7)	45.2 (4.9)	0.22
Corrected RVEF, %		27.5 (6.3)	29.2 (6.1)	24.9 (5.8)	0.01
RVM, g/m^2		30.1 (7.2)	29.1 (7.5)	31.4 (6.8)	0.22
LVEDV, ml/m^2		86.3 (15.6)	89.5 (16.7)	81.8 (12.9)	0.06
LVESV, ml/m^2		38.7 (10.3)	40.1 (11.2)	36.8 (8.6)	0.22
LVSV, ml/m^2		47.6 (7.9)	49.4 (8.2)	45.0 (6.7)	0.03
LVEF, %		55.6 (5.9)	55.8 (6.3)	55.3 (5.2)	0.77
LVM, g/m^2		54.5 (12.3)	55.1 (12)	52.4 (11.4)	0.38
RV / LV ratio		2.04 (0.48)	1.86 (0.32)	2.30 (0.54)	<0.001
PRF, %		36.6 (11.9)	32.8 (11.5)	41.8 (10.6)	0.003
PRV, ml/m^2		28.2 (13.7)	24.2 (11.8)	33.9 (14.5)	0.01

Data are presented as mean (SD) unless otherwise indicated.

a Conservative treatment vs PVR / PPVI

Abbreviations: IQR, interquartile range; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; LVM, left ventricular mass; LVSV, left ventricular stroke volume; PPVI, percutaneous pulmonary valve implantation; PRF, pulmonary regurgitation fraction; PRV, pulmonary regurgitation volume; PVR, pulmonary valve replacement; RVEDV, right ventricular end-diastolic volume; RVEF, right ventricular ejection fraction; RVESV, right ventricular end-systolic volume; RVM, right ventricular mass; RVSV, right ventricular stroke volume; RV / LV ratio, ratio of right ventricular to left ventricular volume; others, see FIGURE 1

association with the need for PVR (AUC, 0.63; 95% CI, 0.49–0.75; *P* = 0.08 and AUC, 0.61; 95% CI, 0.48–0.72; *P* = 0.14; respectively). The remaining parameters had either poor or no discriminatory ability and were not included in further analyses.

The results of the Cox proportional hazards regression are presented in TABLE 3. Univariate analyses revealed that the RV / LV ratio and PRF were associated with the outcome. In the multivariate analysis, PRF did not prove to be an independent predictor of the need for PVR or PPVI either as a continuous or as a binary variable. The only independent predictor was the RV / LV ratio.

Freedom from pulmonary valve replacement or percutaneous pulmonary valve implantation The Kaplan–Meier analysis showed that the ROC analysis–based cutoff values of both the RV/LV ratio and PRF provided good separation of survival curves (FIGURE 3A-3D). We combined these 2 parameters and created the following subgroups: 1) patients with an RV / LV ratio ≤2.4 and a PRF ≤33% (n = 25); 2) patients with an RV / LV ratio ≤2.4 and a PRF >33% (n = 26); and 3) patients with an RV / LV ratio >2.4 and a PRF >33% (n = 10). There were no patients with an RV / LV ratio higher than 2.4 and a PRF of 33% or lower (ie, all patients with an RV / LV

TABLE 2 Receiver operating characteristic curve analyses: discriminatory ability of cardiac magnetic resonance parameters to identify patients requiring pulmonary valve replacement or percutaneous pulmonary valve implantation

Parameter	AUC	Threshold	*P* value	Sensitivity, %	Specificity, %	PPV, %	NPV, %
RV / LV ratio	0.74 (0.61–0.85)	>2.4	<0.001	36 (18–58)	100 (90–100)	100 (66–100)	70 (55–81)
PRF, %	0.71 (0.58–0.82)	>33	0.002	80 (59–93)	56 (38–72)	56 (38–72)	80 (59–93)

Data in parentheses are 95% CIs.

Abbreviations: AUC, area under the curve; NPV, negative predictive value; PPV, positive predictive value; others, see TABLE 1

ratio >2.4 had a PRF >33%). The combination of the RV / LV ratio and PRF had significant discriminatory ability (FIGURE 3C, *P* <0.001 by log-rank test for trend). Only 5 patients with an RV / LV ratio of 2.4 or lower and a PRF of 33% or lower underwent PVR or PPVI (20%). On the other hand, 9 patients (90%) with an RV / LV ratio exceeding 2.4 and a PRF exceeding 33% demonstrated the need for PVR or PPVI.

To avoid the potential bias caused by referring patients in whom PVR or PPVI had already been planned for CMR, we only analyzed patients with PVR or PPVI performed at least 2 months after CMR (n = 54) and obtained similar results (survival proportion: 0.79, 0.37, and 0.13 for subgroup 1, 2, and 3 described above; *P* <0.001 by log-rank test for trend, FIGURE 3D).

None of the Kaplan–Meier analyses using the previously published RVEDV thresholds indicating the likelihood of irreversible RV dilation revealed any association with the outcome (by log-rank test: *P* = 0.97 for a threshold >170 ml/m^2; *P* = 0.53 for a threshold >168 ml/m^2; *P* = 0.31 for a threshold >163 ml/m^2; and *P* = 0.17 for a threshold >160 ml/m^2).

DISCUSSION

Optimal timing for PVR in asymptomatic patients after repair of TOF remains unknown, although some studies indicate certain thresholds of RV parameters beyond which PVR might be suboptimal in terms of normalizing RV size and preserving RV function.1,2,6-9,18-20 The results of our study demonstrate the potential clinical usefulness of CMR variables, namely the RV / LV ratio and PRF, in asymptomatic patients with isolated PR after TOF repair. These parameters showed a significant association with the need for future PVR or PPVI in this population. Therefore, they might be used as a guide for optimal timing for PVR. This, however, needs to be confirmed in prospective studies.

By using the Cox proportional hazards regression, we proved that the RV / LV ratio was the only independent predictor of the need for PVR or PPVI in this population. Additionally, since the Kaplan–Meier curves are better for time-dependent events, we investigated the association with the future PVR or PPVI using survival curves and demonstrated that also PRF had a predictive value.

The RV / LV ratio provides a broad spectrum of information on the impact of the PR on the heart. It reflects not only RV dilation but also adverse consequences for the LV (compression).11 Thus, since the decision about performing PVR in patients after TOF repair is based on numerous factors, the RV / LV ratio seems to be a useful parameter. Normal values of the RV / LV ratio are about 1.15,21-22 and the value of 2.0 has been proposed as a cutoff for severe RV dilation.23-24 In our

TABLE 3 Cox proportional hazards regression analyses including variables with acceptable discriminatory ability in the receiver-operating characteristic curve analyses

Variable	Univariate analysis		Multivariate analysis	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Continuous variables				
RV / LV ratio	5 (2.3–11.1)	<0.001	5 (2.3–11.1)	<0.001
PRF	1.05 (1.01–1.09)	0.01	–	–
Binary variables				
RV / LV ratio	4.3 (1.9–9.9)	0.001	4.3 (1.9–9.9)	0.001
PRF	2.8 (1.07–7.6)	0.02	–	–

Binary analyses compared patients above and below the optimal ROC analysis–based threshold.

Abbreviations: HR, hazard ratio; ROC, receiver operating characteristic curve; others, see TABLE 1

study, the RV/LV ratio higher than 2.4 showed a specificity and positive predictive value of 100% for identifying patients who required PVR or PPVI during follow-up, that is, all patients with the RV/LV ratio exceeding 2.4 underwent the intervention (FIGURE 2). However, the RV/LV ratio had poor sensitivity. This limitation could be at least partially reduced by combining the RV/LV ratio with PRF, which showed a sensitivity of 80% at a cutoff value of 33%. The combination of the RV/LV ratio and PRF provided improved discrimination when compared with either of the parameters alone (FIGURE 3C and 3D).

The universal definition of the severity of PR derived from CMR is lacking. Although numerous investigators use the threshold of 20% as a cutoff distinguishing significant from insignificant PR, higher thresholds (eg, >25%, >35%, and >40%) are used for defining severe PR.11,12,24-29 Additionally, it has been suggested that PRV indexed for the body surface area may better reflect the impact of volume load on the RV than PRF does, which makes the determination of PR severity on the basis of CMR parameters even more confusing.26 We demonstrated that the PRF exceeding 33% provided an acceptable discriminatory ability to distinguish between patients who require and do not require PVR or PPVI. This may help guide therapeutic decisions in this population.

Our study has several limitations. First, the sample size was relatively small. However, the strength of our study is the fact that we included a highly selected population. To avoid changing the decision on performing PVR or PPVI by the factors other than PR and its consequences, we excluded patients with residual ventricular septal defect, a peak RVOT gradient of 30 mm Hg or higher, or at least moderate tricuspid regurgitation. Secondly, like all observational nonrandomized studies, our findings need to be interpreted with a certain degree of caution. Ideally, the potential benefits of incorporating CMR-based parameters into clinical decision making should be confirmed in a randomized prospective study. However, performing such a study in patients with repaired TOF is challenging due to various reasons, and only large multicenter trials have a potential to clearly elucidate this

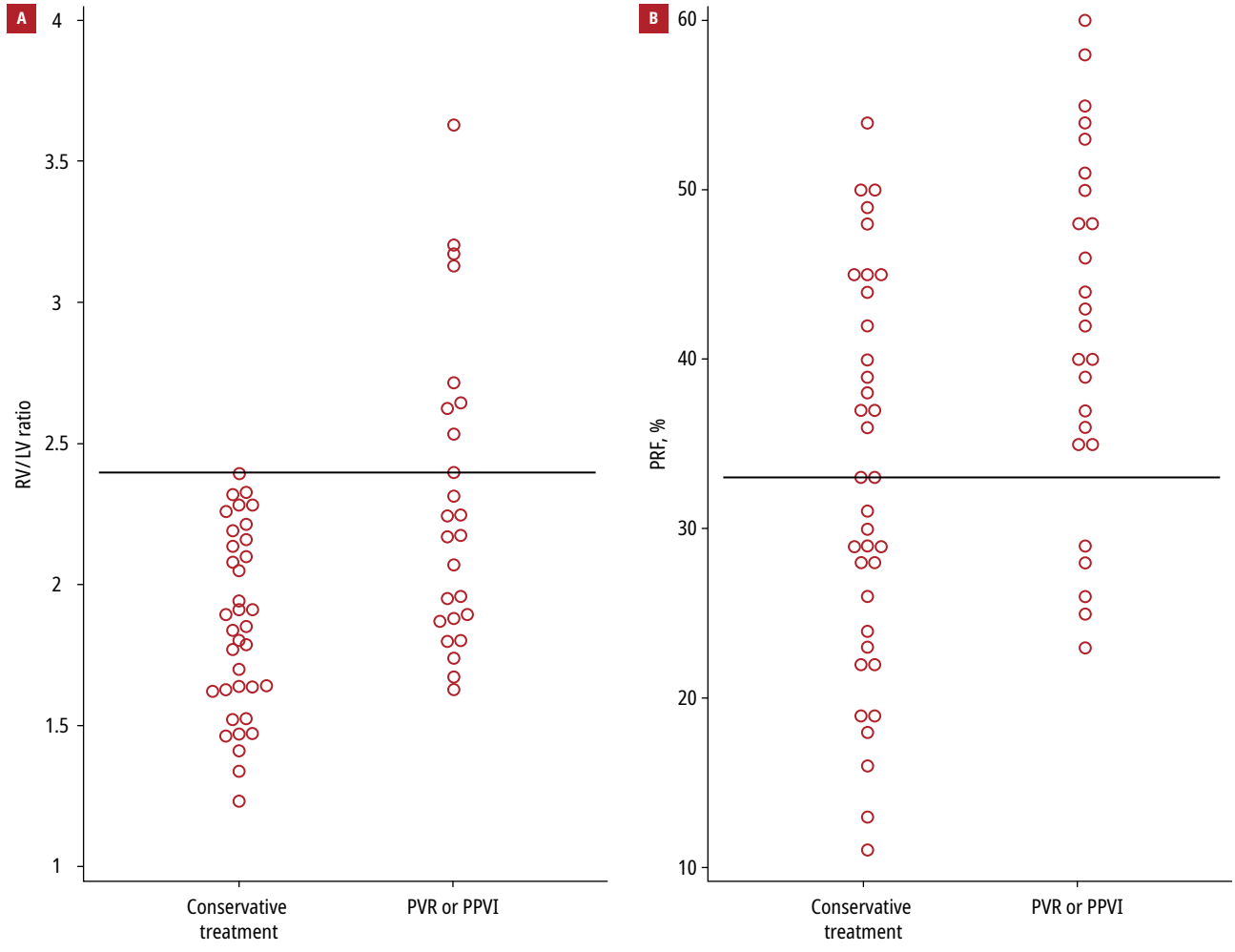

FIGURE 2 Scatterplots of the ratio of right ventricular to left ventricular volume (RV/LV ratio) (**A**) and pulmonary regurgitation factor (PRF) (**B**) in patients on conservative treatment and those requiring pulmonary valve replacement (PVR) or percutaneous pulmonary valve implantation (PPVI). Solid lines indicate the optimal thresholds for identifying patients with the need for PVR or PPVI.

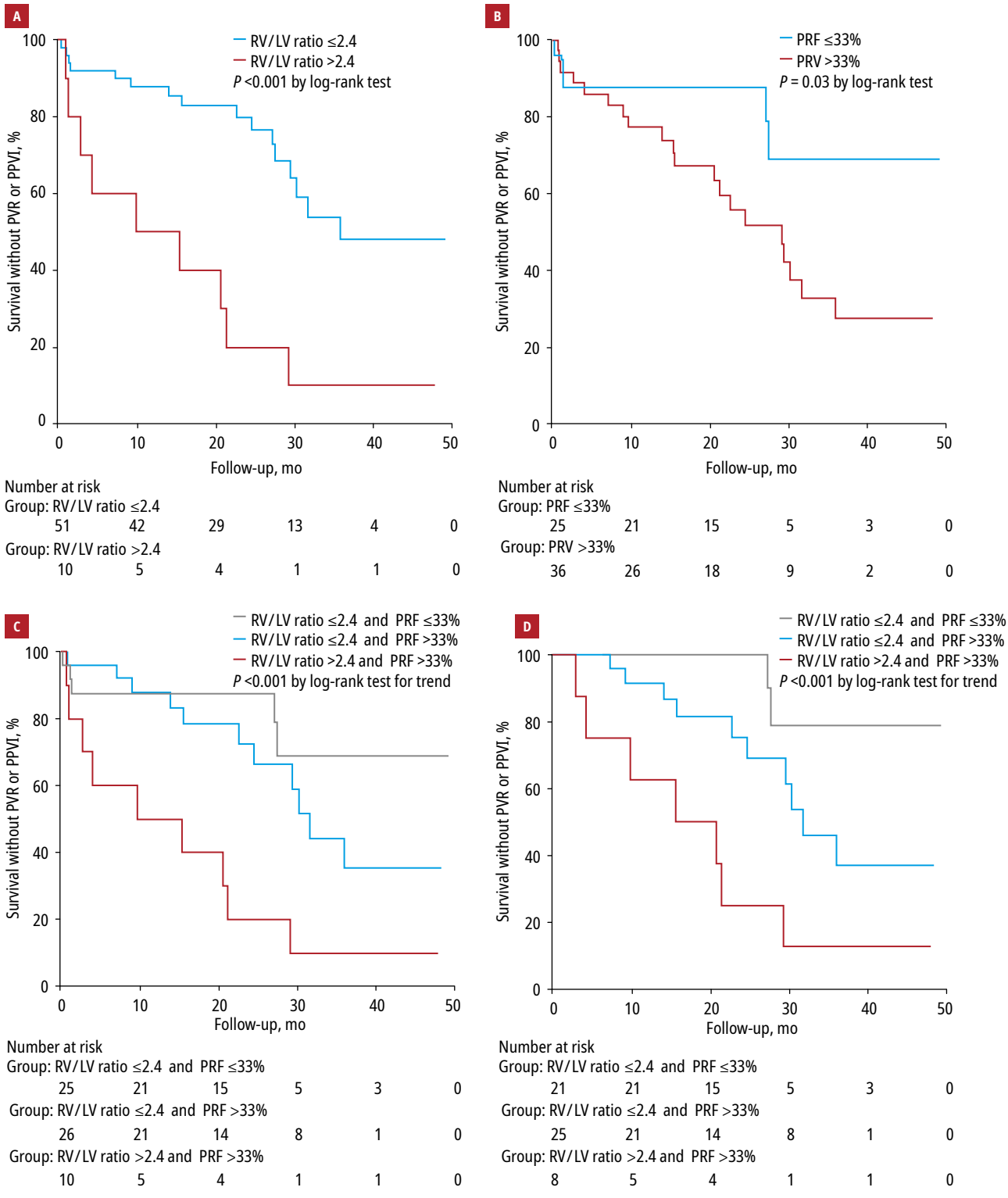

FIGURE 3 The Kaplan-Meier curves for survival without pulmonary valve replacement (PVR) or percutaneous pulmonary valve implantation (PPVI): **A** – stratified by the right ventricular to left ventricular volume (RV/LV ratio); **B** – stratified by pulmonary regurgitation factor (PRF); **C** – stratified by both the RV/LV ratio and PRF; **D** – stratified by both the RV/LV ratio and PRF in a population restricted to patients with PVR or PPVI performed at least 2 months after cardiac magnetic resonance

issue. Finally, restrictive RV physiology affects the RV size in patients with PR. Thus, we cannot exclude the effect of this factor on the RV size.

In contrast to asymptomatic patients with aortic regurgitation in whom precise indications for surgery are established in terms of excessive LV dilation and dysfunction with exact cutoff values provided for these parameters, in asymptomatic patients with PR, such a categorical approach is limited and mainly descriptive indications are given (ie, moderate-to-severe or progressive RV dilation, moderate-to-severe RV

dysfunction).1,2 Current European recommendations for PVR in asymptomatic patients after TOF repair do not provide any cutoff value indicating the need for intervention due to RV dilation or dysfunction, leaving place for arbitrary decisions of a treating physician.1 Thus, we did not use any specific thresholds for RV dilation and impaired RV function. In our study, as in a typical clinical scenario, all heart team members had full access to CMR data. This might have caused some bias and might have resulted in referring a patient for PVR or PPVI as soon as the RVEDV exceeded a certain threshold of the RV volume when no normalization of the RV size should be expected.6-8 However, the Kaplan–Meier curves did not show differences in freedom from PVR or PPVI in groups stratified according to these thresholds. Additionally, the bias was limited by making the decision on performing PVR or PPVI by a consensus of heart team members with experience in congenital heart diseases. Nevertheless, some bias cannot be excluded.

In conclusion, we demonstrated that the RV / LV ratio and PRF are significantly associated with the need for PVR or PPVI in asymptomatic patients with isolated moderate-to-severe PR after repair of TOF. However, our findings may be hampered by the retrospective design of the study and the limited number of patients included. Further studies are needed to elucidate the potential clinical value of these parameters in deciding about early intervention for pulmonary incompetence in this population.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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