MAGNETIC RESONANCE

Four-year cardiac magnetic resonance (CMR) follow-up of patients treated with percutaneous pulmonary valve stent implantation

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Abstract

Objectives To investigate follow-up after percutaneous pulmonary valve implantation (PPVI).

Methods Forty patients with pulmonary conduit dysfunction (males/females 24/16; 21±08 years; 12 tetralogy of Fallot, 11 aortic valve disease, 17 other congenital heart disease) were planned for CMR before PPVI and repeated 7 times up to 48 months. CMR prospective results regarded: pressure gradient (PG) and regurgitation fraction (RF); end-diastolic volume, end-systolic volume, and stroke volume indexed to body surface area (EDVI, ESVI, and SVI); ejection fraction (EF) of right and left ventricles (RV, LV). A Friedman test was used for comparisons.

Results Overall, PG (31 ± 06 to 16 ± 4 mmHg), RF (16 ± 17 to 0.3 ± 1 %), RVEDVI (82 ± 38 to 58 ± 12 ml/m^2), and RVESVI (44 ± 12 to 30 ± 13 ml/m^2) declined ($p<0.001$), RVEF (49 ± 13 to 58 ± 12 %) and RVSVI (from 38 ± 14 to 40 ± 8 ml/m^2) increased ($p<0.001$), LVEDVI (67 ± 17 to 73 ± 18 ml/m^2) and LVSVI (37 ± 11 to 43 ± 10 ml/m^2) increased ($p=0.034$ and $p<0.001$). Two patients had valve fracture at 24 and 36 months and underwent surgery. One patient had stent restenosis at 24 months and underwent percutaneous retreatment. Baseline/follow-up CMR did not predict PPVI failure.

Conclusions CMR demonstrated restored pulmonary conduit function, reduced RV volumes and increased RV and LV function but did not predict valve fracture/restenosis.

Key points
- *A CMR 4-year follow-up after PPVI showed restored pulmonary conduit function*
- *RV volumes were significantly reduced*
- *RV function was significantly better in terms of increased EF and SVI*
- *LV function was significantly better in terms of increased EDVI and SVI*
- *Baseline/follow-up CMR did not predict three cases of PPVI failure*

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Introduction

Percutaneous pulmonary valve implantation (PPVI) is a non-surgical option for treating right ventricular (RV) outflow tract and/or pulmonary trunk dysfunction, representing one of the most exciting approaches of interventional cardiology as the first in-human application of this technique [1]. In particular, Bonhoeffer et. al. [2] developed a new approach to treat RV conduit dysfunction using a bovine valve sutured on a platinum-iridium alloy stent, with welded joints of gold (Melody, Medtronic, Inc., Minneapolis, MN, US) [2].

Being less invasive than surgical pulmonary valve replacement, PPVI avoids the risks of ventriculotomy and cardiopulmonary bypass as well as the risks of bleeding and infections associated with reoperation, avoiding postoperative intensive care reduces costs. The procedure would have to provide feasibility and safety comparable to those of surgical procedures, which are burdened by conduit failures and require multiple re-interventions [3]. The most common indications for surgical pulmonary valve replacement are pulmonary regurgitation with or without stenosis, RV dilation and hemodynamically significant residual lesions [1].

The majority of patients referred for surgical pulmonary valve replacement or PPVI have a diagnosis of tetralogy of Fallot with pulmonary stenosis or pulmonary atresia or a previous surgical treatment for critical pulmonary valve stenosis [4–6]. In these patients, after repairing, the chronic RV volume overload leads to ventricular dilation and impairment of systolic and diastolic function. In the long term, this causes atrial and ventricular arrhythmia as well as heart failure and increases the risk of sudden death [7]. Restoration of pulmonary valve competence at an appropriate time has resulted in improvement in RV function and effort tolerance and reduces the incidence of arrhythmias [8, 9]. Thus, patient selection and correct timing for PPVI plays a key role in the success of this technique.

Follow-up after PPVI may be performed using echocardiography as a first-line technique [10]. This modality is relatively inexpensive, widely available, and is not associated with ionizing radiation exposure. However, its limitations include poor acoustic windows in assessing RV morphology, function and valve regurgitation [11]. In recent years, studies have been reporting the use of cardiac magnetic resonance (CMR) to evaluate patients before and after PPVI [12–14]. CMR has been demonstrated to outperform echocardiography in the evaluation of cardiac chambers, especially the RV, and it is considered as a first-choice technique in this setting [15–17].

Our aim was to report the 4-year CMR follow-up of patients treated with PPVI.

Materials and methods

Study design and population

After institutional review board approval waived specific patient consent for data analysis, we retrospectively retrieved prospective reports of CMR examinations performed before and after PPVI from January 2008 to June 2014 in 40 consecutive patients treated with a Melody valve stent (Medtronic,

Table 1 Patient characteristics, disease spectrum, and conduit type

Sex and age	Number	%
Males (age 21±09 years)	24	60
Females (age 22±07 years)	16	40
Total (age 21±08 years)	**40**	**100**
Disease spectrum		
Tetralogy of Fallot	12	30
Aortic valve disease (Ross procedure)	11	28
Transposition of the great arteries	5	13
Pulmonary atresia	4	10
Double outlet right ventricle	3	7
Truncus arteriosus	3	7
Pulmonary stenosis	2	5
Total	**40**	**100**
Right ventricle outflow tract conduit type		
Homograft	20	50
Shelhigh	5	13
Matrix	4	10
Monocuspid Homograft	3	7
Hancock	3	7
Contegra	1	3
Jonescu-Shiley	1	3
Conduit not implanted	3	7
Total	**40**	**100**

Data for age (at baseline CMR) are presented as mean ± standard deviation.

Inc., Minneapolis, MN, US). Demographics, age, and disease spectrum are shown in Table 1. CMR had been planned at least 1 month before PPVI, and 1, 3, 6, 12, 24, 36, and 48 months after PPVI. Paediatric cardiologists obtained informed consent for the intervention and use of this type of valve for PPVI. Informed consent was obtained by each patient for the injection of gadolinium (Gd)-based contrast material, when administered.

CMR protocol

We used a 1.5-T unit with 40-mT/m gradient power (Magnetom Sonata Maestro Class, Siemens, Erlangen, Germany) and a four-channel cardio-thoracic phased-array coil. The same imaging protocol was used before and after PPVI. The mean CMR duration was 45± 10 min.

Electrocardiographically-triggered short-axis, four-chamber and long-axis images were obtained with a breath-hold steady-state free precession (SSFP) sequence (true fast imaging with SSFP, true-FISP) acquired with the following

technical parameters: TR/TE, 4.0/1.5 ms; FA, 80°; slice thickness, 8 mm; temporal resolution, 45 ms; mean acquisition time, 14±4 s. A breath-hold turbo spoiled gradient echo sequence (fast low-angle shot, FLASH) was performed for phase-velocity mapping of pulmonary flow with the following technical parameters: TR/TE, 4.0/3.2 ms; slice thickness, 5 mm; velocity encoding (VENC) from 150 to 350 ms; temporal resolution, 41 ms; mean acquisition time, 15±4 s. Firstly we acquired the phase-contrast sequence with a VENC of

150 ms; in the presence of aliasing, we modified the VENC adding 50 ms for each new sequence, step-by-step up to the complete disappearance of the aliasing artefact.

Before PPVI, the slice of the phase-contrast sequence was located on the pulmonary conduit, perpendicular to the vessel long axis, immediately distal to the valve; after PPVI, the slice was located at least 4 mm distal to the stent artefact to avoid contamination of images from the metallic artefact. Mr angiography with a

Fig. 1 Example of RV volume analysis using CMR cine sequences in a short-axis view of the basis (**a, b**), the mid-ventricle (**c, d**) and the apex (**e, f**). In the left column are shown images of the short axis at the end-diastolic phase (**a, c, e**) and in the right column at the end-systolic phase (**b, d, f**)
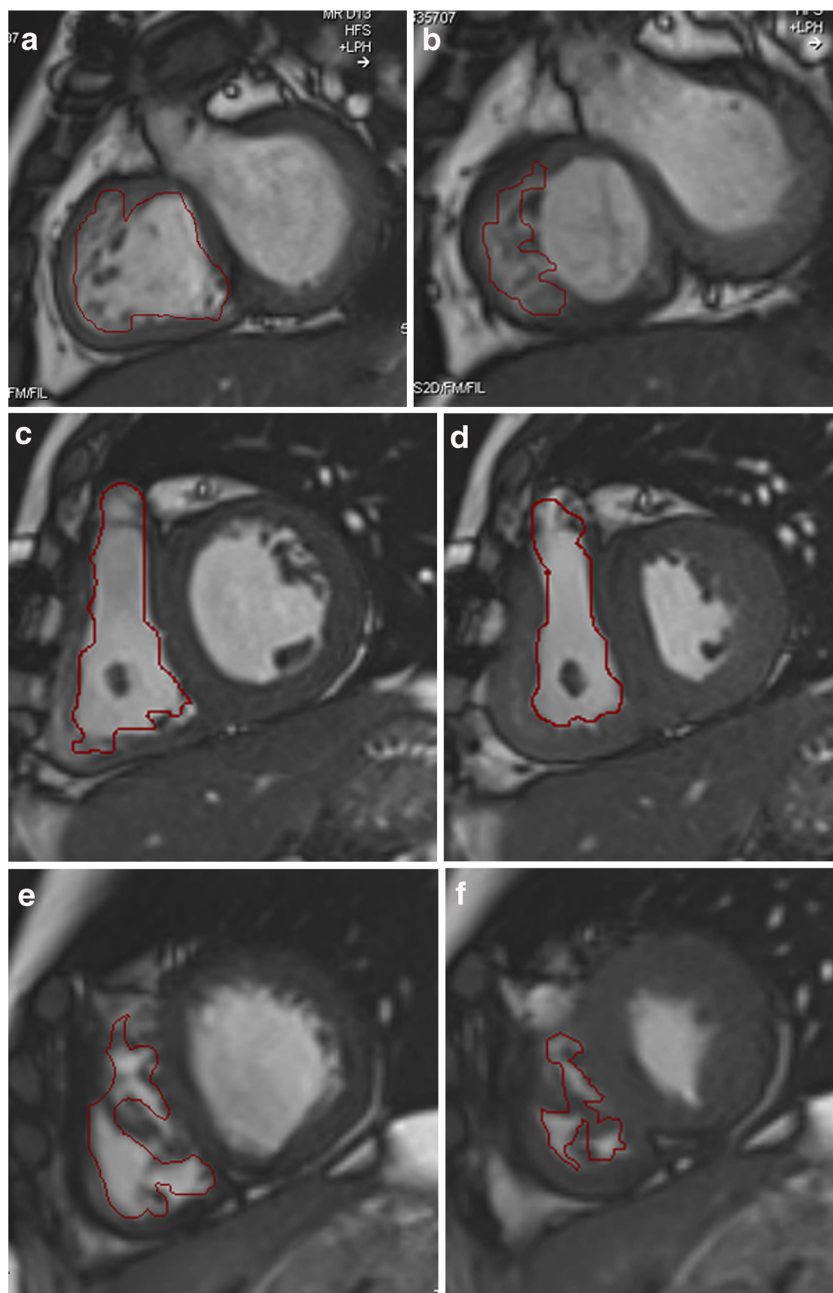
three-dimensional gradient-echo fast low-angle shot sequence (FLASH, TR=3.32 ms, TE=1.21 ms, flip angle 25°, slice thickness=1.5 mm, 72 partitions, acquisition time 21 s) after injection of 0.1 mmol/kg of gadobenate dimeglumine (MultiHance, Bracco Imaging SpA, Milan, Italy) was performed only at the baseline CMR before PPVI [18]. MR angiography was not performed after PPVI due to the presence of the metal artefact in the pulmonary conduit.

Image analysis

Cine true-FISP images in the short-axis plane, from heart base to apex, were segmented semiautomatically (Syngo-Argus, version VE32B, Siemens Medical Solutions, Erlangen, Germany) by a reader with 7 years of experience in CMR imaging, to obtain end-diastolic volume (EDV), normalized to body surface area (EDV index, EDVI), end-systolic volume index (ESVI), stroke volume (SVI), and ejection fraction (EF) of the RV and left ventricle (LV) [19]. In Fig. 1 is shown an example of RV segmentation. Papillary muscles were excluded from the volume. The basis was chosen with the references of a four-chamber view [20].

Reverse flow, forward flow and peak velocity data of the pulmonary valve were obtained by segmenting phase contrast images with the same software. We estimated pulmonary regurgitation fraction (RF) by flow data [21]. The pressure gradient (PG) was estimated from peak flow velocities using Bernoulli's equation as described [22].

Statistical analysis

A Friedman test was used to compare EDVI, ESVI, SVI, EF, RF and PG before and after 1, 3, 6, 12, 24, 36, and 48 months from PPVI. All testing was performed using SPSS statistical software for Windows, release 14.0 (SPSS Inc., Chicago, IL, US). P values lower than 0.05 were considered statistically significant.

Results

Overall results of CMR before and during the follow-up after PPVI are shown in Table 2. Overall, PG (31±06 to 16±4 mmHg) and RF (16±17 to 0.3±1 %) declined significantly ($p<0.001$). A significant reduction of RV volumes (EDVI from 82±38 to 58±12 ml/m^2, $p<0.001$; ESVI from 44±12 to 30±13 ml/m^2, $p<0.001$), as well as a significant increase of RVEF (from 49±13 to 58±12 %, $p<0.001$) and SVI (from 38±14 to 40±8 ml/m^2, $p<0.001$) were observed; RF and PG were significantly reduced at the last

Table 2 CMR results before and after percutaneous pulmonary valve implantation

Months after PPVI	Number of Patients	Pulmonary conduit		RV				LV			
		RF (%)	PG (mmHg)	EDVI (ml/m^2)	ESVI (ml/m^2)	SVI (ml/m^2)	EF (%)	EDVI (ml/m^2)	ESVI (ml/m^2)	SVI (ml/m^2)	EF (%)
Baseline	40	16±17	32±16	82±38	44±12	38±14	49±13	67±17	30±13	37±11	56±09
1	19	0.9±03	12±12	67±21	33±15	37±11	54±11	66±18	29±35	37±12	57±10
3	29	1.5±04	13±13	52±15	34±19	36±12	53±15	74±21	32±11	42±13	57±08
6	31	2.0±05	14±12	65±23	30±16	35±12	55±12	75±20	32±13	43±13	59±10
12	35	1.8±05	12±05	53±10	32±12	36±10	53±10	78±18	33±12	45±10	58±09
24	32	0.3±01	13±04	56±12	30±14	38±12	56±12	74±23	30±12	49±15	58±08
36	18	0.3±01	14±05	69±17	30±16	39±06	57±09	74±17	31±11	43±09	58±07
48	20	0.2±01	14±04	58±12	30±13	40±8	58±12	73±18	30±11	43±10	59±06
p		**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**<0.001**	**0.034**	**0.508**	**<0.001**	**0.147**

PPVI: percutaneous pulmonary valve replacement. RV: right ventricle. LV: left ventricle. RF: regurgitation fraction. PG: pressure gradient. EDVI: end-diastolic volume index. ESVI: end-systolic volume index. SVI: stroke volume index. EF: ejection fraction. Data are presented as mean ± standard deviation.
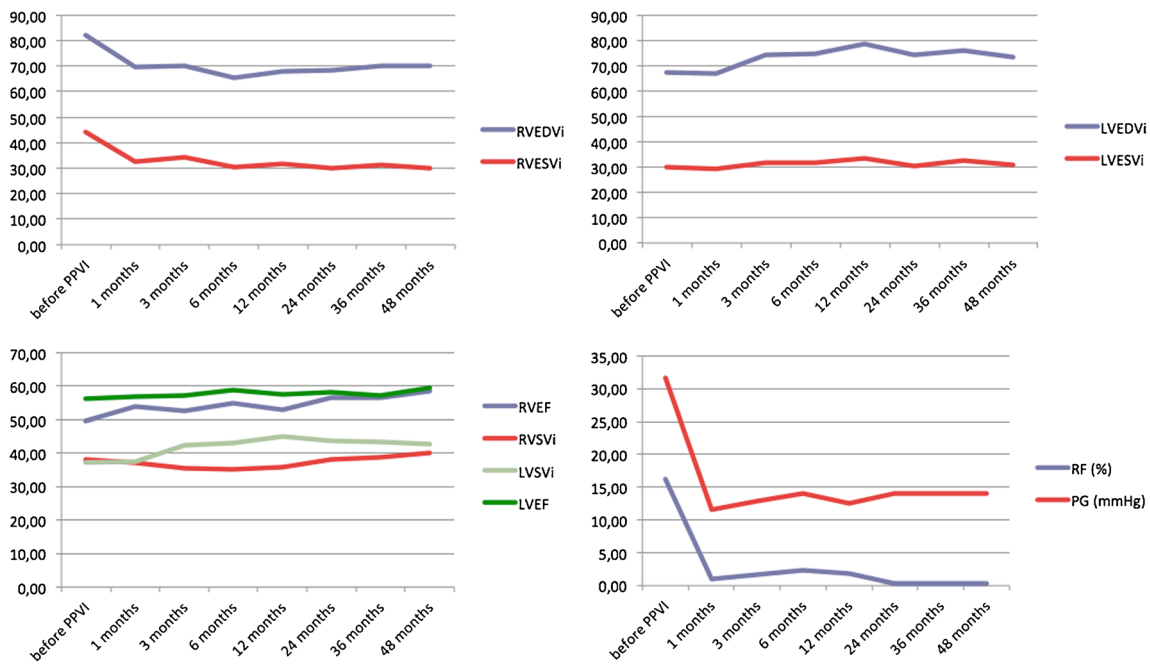
Fig. 2 Graphs showing ventricle volumes, ejection fraction, regurgitation fraction and pressure gradient as plotted versus time. PPVI: percutaneous pulmonary valve replacement. RV: right ventricle.

LV: left ventricle. EDVI: end-diastolic volume index. ESVI: end-systolic volume index. SVI: stroke volume index. EF: ejection fraction. RF: regurgitation fraction. PG: pressure gradient

follow-up ($p<0.001$). LV analysis showed a significant increase in EDVI (from 67 ± 17 to 73 ± 18 ml/m^2, $p=0.034$) and SVI (from 37 ± 11 to 43 ± 10 ml/m^2, $p<0.001$) while no significant changes were observed for ESVI and EF. In

Fig. 2, RV and LV volumes, EF, RF and PG are represented during the follow-up as plotted versus time. In Figs. 3 and 4, CMR images before PPVI and during the follow-up are shown.
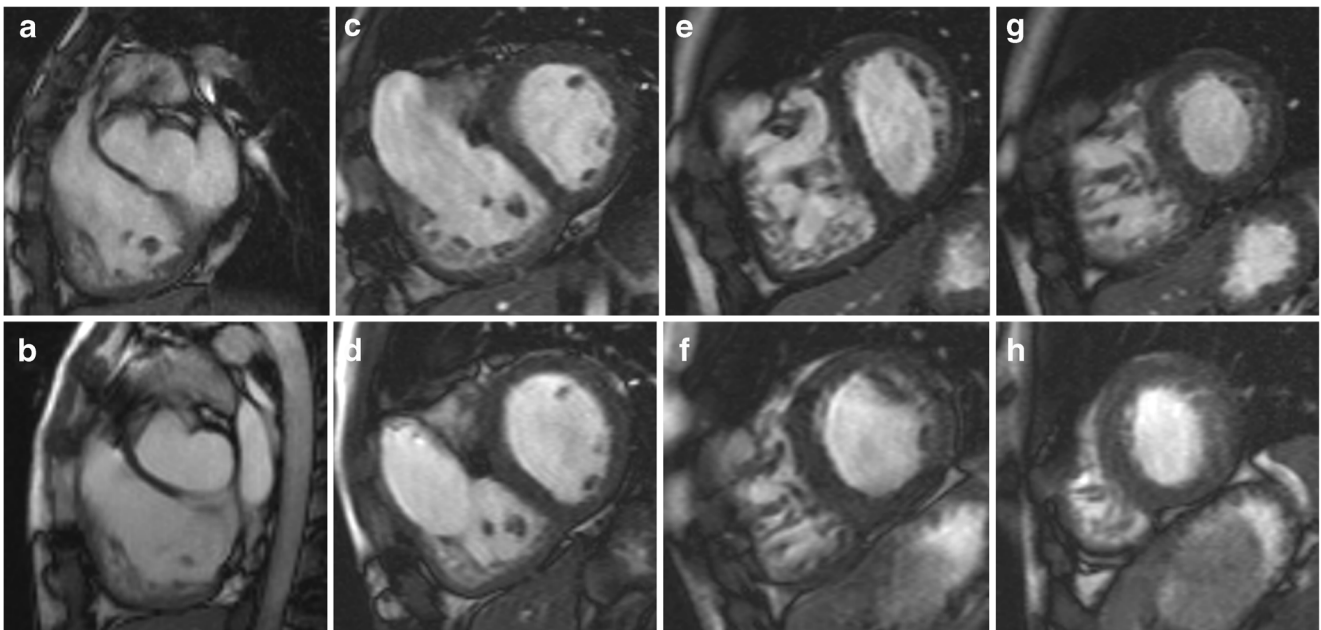

Fig. 3 CMR images of a 14-year-old patient with congenital pulmonary atresia treated with a homograft. Due to homograft stenosis, a percutaneous pulmonary valve (PPVI) was implanted. In the figure are shown images of cine true-FISP sequences at the end-diastolic phase. Right ventricle outflow tract (**a, b**) and three short axis planes (**c, d, e, f, g** and **h**) from the basal portion of the heart to the apex before (upper line)

and after 48 months from PPVI (lower line) are shown. Note the artefact of pulmonary valve stent after PPVI in **b** and the reduction of the right ventricle end-diastolic volume (from 80 ml/m^2 to 55 ml/m^2) and septal flattening. Septal flattening is visible on **c** and **e**. Reduction of the volume abd of septal flattenig is evident when comparing images **e** and **f**

Fig. 4 CMR of a 24-year-old patient with tetralogy of Fallot treated with a homograft. Due to homograft stenosis, a percutaneous pulmonary valve was implanted (PPVI). Cine true-FISP images at the end-diastolic phase of the right ventricle outflow tract before (**a**) and after PPVI (**b**) are shown. Reference lines (white lines) of phase-contrast sequences before (**a**) and after PPVI (**b**) are also shown
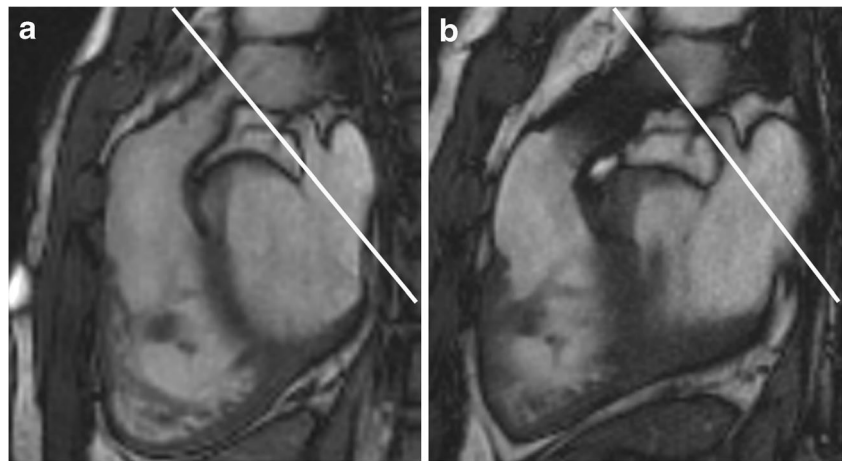
One patient performed CMR with an intracardiac defibrillator with the housing implanted on the right anterior side of the chest (Fig. 5). Before CMR examinations, a dedicated cardiologist deactivated the device to avoid a wrong response during the examination.

Two patients needed a surgical conduit replacement 24 and 36 months after the PPVI due to a fracture of the pulmonary stents. One patient needed a second implantation of a stent in the pulmonary conduit after 24 months of PPVI due to a restenosis of the valve. Patient characteristics as well as baseline and follow-up CMR results of these three patients up to the adverse event are shown in Table 3. We did not find any signs or parameters of CMR that could predict the adverse events.

Discussion

In the last decade, PPVI has emerged as the preferred treatment in pulmonary conduit dysfunction in CHD patients, being less invasive than surgical repair. We investigate with CMR a consecutive series of patients before and after PPVI to evaluate the immediate and mid-term changes in cardiac function values.

We observed a significant and progressive reduction of RV volumes as well as a significant increase of RVEF and RVSVI during the investigated follow-up. The most important remodelling occurred 3 months after the PPVI. However, it was important to show what happened during the following four years, when a further progressive remodelling was observed with a continuous increase of RVEF. Regarding flow analysis, both RF and PG were strongly and immediately reduced (at 1 month follow-up) and were thereafter stable at lowered values. These results were in agreement with the literature [12–14, 16], showing a good effect of PPVI on the RV function. However, to our knowledge, there are no studies showing the follow-up of these patients up to 4 years after PPVI.
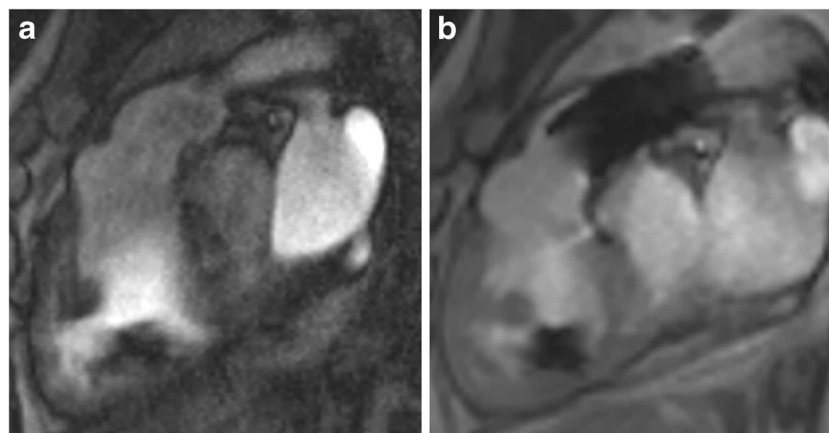

Fig. 5 CMR of a 31-year-old patient with tetralogy of Fallot with an implantable cardiac defibrillator (ICD), previously treated with a homograft. Cine FLASH sequences at the end-diastolic phase of the right ventricle outflow tract before (**a**) and after PPVI (**b**) are shown. Note the artefact created by the catheter in the right ventricle and that created by the pulmonary valve stent after PPVI. The ICD in the right position of the chest allowed for a diagnostic CMR without artefacts impairing heart evaluation

Table 3 CMR results before and after percutaneous pulmonary valve implantation in three cases of valve failure

Number, Patient sex, CHD, Conduit Type, Age at baseline	Time	Pulmonary conduit		RV					LV			
		RF (%)	PG (mmHg)	EDVI (ml/m²)	ESVI (ml/m²)	SVI (ml/m²)	EF (%)		EDVI (ml/m²)	ESVI (ml/m²)	SVI (ml/m²)	EF (%)
1. Female, Truncus arteriosus, Hancock, 10 years	Baseline	0	57	80	32	48	62		68	27	41	61
	F/U to 24 months	0.4±1	27±01	75±26	30±17	45±15	60±12		82±08	33±04	48±10	59±9
2. Female, Double outlet, RV Monocuspid Homograft, 12 years	Baseline	28	51	87	42	45	51		79	31	48	61
	F/U to 36 months	0±0	24±17	57±10	33±08	24±06	42±09		65±13	32±08	34±07	52±05
3. Female, Fallot tetralogy, Jonescu-Shiley, 27 years	Baseline	67	33	45	26	19	42		34	19	15	44
	F/U to 24 months	8±4	26±18	47±04	30±05	17±04	41±07		60±03	12±03	18±02	60±03

CHD: congenital heart disease. RV: right ventricle. LV: left ventricle. RF: regurgitation fraction. PG: pressure gradient. EDVI: end-diastolic volume index. ESVI: end-systolic volume index. SVI: stroke volume index. EF: ejection fraction. F/U: follow-up. Data are single values for baseline CMR, mean ± standard deviation for F/U.

Patients 1 and 2 had valve fracture and underwent open surgery; patient 3 had endocarditis and valve stenosis and underwent a second percutaneous stent positioning (see text).

Interestingly, follow-up of LV volumes showed a significant increase in EDVI and SVI. The former result can be explained considering that the reduction of RVEDVI allows a better diastolic compliance of LV, in turn determining an increased LVSVI. The improvement of LVEDVI likely reflects a decrease in RV volume and pressure overload, and hence a reduction in interventricular dependence from paradoxical septal motion. Conversely, the LVEF remained unchanged due to the insignificant increase in LVESVI.

Of note, one patient with an implantable cardiac defibrillator was fully studied with CMR with a comprehensive evaluation of RV and RV outflow tract function. The reason for this good result is that the housing of the device was implanted on the right side of the chest, causing artefacts to not influence heart imaging. This CMR was performed because the patient had been enrolled in an IRB-approved prospective study aimed at evaluating the MR safety in patients having pacemakers or implantable cardiac defibrillators.

PPVI implies some possible complications as stent fracture [23] or stent restenosis [23, 24]. In our population, two patients had stent fracture and one had stent restenosis. In the two cases of stent fracture, a surgical implantation of a conduit was performed; in the case of restenosis, a new stent was implanted. We did not find any CMR sign or parameter that could predict the adverse events, the baseline and follow-up results (see Table 3) not being different from those patients who did not have any adverse events. Thus, while endocarditis is the most probable cause of restenosis, the only possible explanation for the fracture is a suboptimal correspondence between the stent geometry and the individual outflow tract anatomy.

This study had some limitations. First, we retrospectively evaluated a heterogeneous sample of patients with congenital heart diseases originally treated with different surgical procedures. However, the series of patients was consecutive and all patients presented the same condition of pulmonary stenosis/insufficiency treated with PPVI. Second, we did not estimate the inter- and intra-observer reproducibility of CMR data. However, CMR is widely considered more reproducible than echocardiography in the evaluation of the right chambers and pulmonary artery in a multiplanar and comprehensive evaluation [15, 21]. Finally, only one type of pulmonary valve stent was involved, as described in the Methods section, so that our conclusion cannot be applied to other pulmonary valve stents.

In conclusion, a CMR four-year follow-up after PPVI demonstrated restored pulmonary conduit function, reduced RV volumes and increased RV and LV function. However, it did not predict valve fracture/restenosis.

References

1. Khambadkone S, Bonhoeffer P (2004) Nonsurgical pulmonary valve replacement: why, when, and how? Catheter Cardiovasc Interv 62:401–408

2. Bonhoeffer P, Boudjemline Y, Saliba Z et al (2000) Percutaneous replacement of pulmonary valve in a right-ventricle to pulmonary-artery prosthetic conduit with valve dysfunction. Lancet 356:1403–1405

3. Stark J (1998) The use of valved conduits in pediatric cardiac surgery. Pediatr Cardiol 19:282–288

4. Lurz P, Bonhoeffer P, Taylor AM (2009) Percutaneous pulmonary valve implantation: an update. Expert Rev Cardiovasc Ther 7:823–833

5. Eyskens B, Reybrouck T, Bogaert J et al (2000) Homograft insertion for pulmonary regurgitation after repair of tetralogy of fallot improves cardiorespiratory exercise performance. Am J Cardiol 85:221–225

6. Kanter KR, Budde JM, Parks WJ et al (2002) One hundred pulmonary valve replacements in children after relief of right ventricular outflow tract obstruction. Ann Thorac Surg 73:1801–1806

7. Gatzoulis MA, Elliott JT, Guru V et al (2000) Right and left ventricular systolic function late after repair of tetralogy of Fallot. Am J Cardiol 86:1352–1357

8. Conte S, Jashari R, Eyskens B et al (1999) Homograft valve insertion for pulmonary regurgitation late after valveless repair of right ventricular outflow tract obstruction. Eur J Cardiothorac Surg 15:143–149

9. Therrien J, Siu SC, Harris L et al (2001) Impact of pulmonary valve replacement on arrhythmia propensity late after repair of tetralogy of Fallot. Circulation 103:2489–2494

10. Li W, Davlouros PA, Kilner PJ et al (2004) Doppler-echocardiographic assessment of pulmonary regurgitation in adults with repaired tetralogy of Fallot: comparison with cardiovascular magnetic resonance imaging. Am Heart J 147:165–172

11. Valente AM, Cook S, Festa P et al (2014) Multimodality imaging guidelines for patients with repaired tetralogy of fallot: a report from the american society of echocardiography: developed in collaboration with the society for cardiovascular magnetic resonance and the society for pediatric radiology. J Am Soc Echocardiogr 27:111–141

12. McElhinney DB, Hellenbrand WE, Zahn EM et al (2010) Short- and medium-term outcomes after transcatheter pulmonary valve placement in the expanded multicenter US melody valve trial. Circulation 122:507–516

13. Eicken A, Ewert P, Hager A et al (2011) Percutaneous pulmonary valve implantation: two-centre experience with more than 100 patients. Eur Heart J 32:1260–1265

14. Lurz P, Nordmeyer J, Giardini A et al (2011) Early versus late functional outcome after successful percutaneous pulmonary valve implantation: are the acute effects of altered right ventricular loading all we can expect? J Am Coll Cardiol 57:724–731

15. Hendel RC, Patel MR, Kramer CM et al (2006) ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 appropriateness criteria for cardiac computed tomography and cardiac magnetic resonance imaging: a report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology. J Am Coll Cardiol 48:1475–1497

16. Secchi F, Resta EC, Piazza L et al (2014) Cardiac magnetic resonance before and after percutaneous pulmonary valve implantation. Radiol Med 119:400–407

17. Secchi F, Di Leo G, Papini GD et al (2011) Cardiac magnetic resonance: impact on diagnosis and management of patients with congenital cardiovascular disease. Clin Radiol 66:720–725

18. Vogt FM, Theysohn JM, Michna D et al (2013) Contrast-enhanced time-resolved 4D MRA of congenital heart and vessel anomalies: image quality and diagnostic value compared with 3D MRA. Eur Radiol 23:2392–2404

19. Van Dijck I, Budts W, Cools B et al (2014) Infective endocarditis of a transcatheter pulmonary valve in comparison with surgical implants. Heart 0:1–6

20. Kramer CM, Barkhausen J, Flamm SD, Kim RJ, Nagel E (2013) Society for Cardiovascular Magnetic Resonance Board of Trustees Task Force on Standardized Protocols. Standardized cardiovascular magnetic resonance (CMR) protocols 2013 update. J Cardiovasc Magn Reson 15:91

21. Caudron J, Fares J, Lefebvre V, Vivier PH, Petitjean C, Dacher JN (2012) Cardiac MRI, assessment of right ventricular function in acquired heart disease: factors of variability. Acad Radiol 19(8):991–1002

22. Kilner PJ, Gatehouse PD, Firmin DN (2007) Flow measurement by magnetic resonance: a unique asset worth optimising. J Cardiovasc Magn Reson 9:723–728

23. Secchi F, Iozzelli A, Papini GD et al (2009) MR imaging of aortic coarctation. Radiol Med 114:524–537

24. Butera G, Milanesi O, Spadoni I et al (2013) Melody transcatheter pulmonary valve implantation. Results from the registry of the Italian Society of Pediatric Cardiology. Catheter Cardiovasc Interv 81:310–316