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Arrhythmogenic right ventricular cardiomyopathy (ARVC) mimics: the knot unravelled by cardiovascular MRI

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AIM: To assess the role of cardiovascular magnetic resonance imaging (CMRI) in patients referred for suspected arrhythmogenic right ventricular cardiomyopathy (ARVC), its ability to identify ARVC mimics, and subsequent clinical impact.

MATERIALS AND METHODS: The CMRI registry of the year 2014 was analysed to identify all consecutive patients referred for suspected ARVC. A comprehensive CMRI protocol that included anatomy, bi-ventricular function modules, and late gadolinium enhancement (LGE) was performed in all patients.

RESULTS: Out of 2,481 CMRI performed, 124 patients (5%) were referred for suspected ARVC. A pathological substrate was identified at CMRI in 36 patients (29%): five patients (4%) had ischaemic heart disease (IHD) and 10 (8%) non-IHD; five patients (4%) met CMRI criteria for ARVC and 16 (13%) were ARVC mimics. right ventricular end-diastolic volume (RVEDV) and right ventricular stroke volume (RVSV) were significantly higher in patients with ARVC mimics (RVEDV p=0.007, RVSV p=0.012) and ARVC (RVEDV p=0.013, RVSV p=0.013), as compared to those with structurally normal hearts. CMRI was superior to echocardiography in the identification of ARVC mimics (13% versus 1%, p=0.01).

CONCLUSIONS: CMRI was able to identify 16 (13%) ARVC mimics, from congenital abnormalities to acquired heart disease. CMRI was superior in identifying ARVC mimics compared to echocardiography, and overall provided a change in diagnosis in 22% of patients.

Introduction

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a rare genetic disease, with variable penetrance.[1] First described in 1736 by Giovanni Maria Lancisi in "De Motu Cordis et Aneurysmatibus",[2] it was initially thought to involve primarily the right ventricle (RV), with partial or

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total absence of the RV musculature and fibro-fatty replacement,[3,4] but recent evidence showed that in up to 70% of cases there is also left ventricular (LV) involvement.[5–7] Clinical symptoms are often heterogeneous and non-specific, including palpitations, syncope, and atypical chest pain; hence representing a diagnostic challenge. ARVC can lead to biventricular heart failure and sudden cardiac death (SCD), which represents the first manifestation of the disease in up to 20% of cases.[8] An implantable cardioverter defibrillator (ICD) decreases the risk of SCD, so the correct diagnosis is crucial. The diagnosis of ARVC is based on the 2010 Task Force Criteria (TFC),[9] which recommended a multiparametric approach that takes into account echocardiography, electrocardiography (ECG), and histological abnormalities, and documented ventricular arrhythmia and family history. Imaging criteria for ARVC subtend potential diagnostic pitfalls of which the clinician needs to be aware: normal variants mischaracterised as ARVC, such as chest wall deformity and non-ARVC fatty infiltration (obesity, post-myocardial infarction), and pathological conditions mimicking ARVC, such as myocarditis, sarcoidosis, and pre-tricuspid shunts, which are commonly referred to as ARVC mimics.[10] Cardiovascular magnetic resonance imaging (CMRI) as part of the 2010 TFC is increasingly used in clinical practice in patients with suspected ARVC in the context of a multimodality imaging assessment. The aim of the present study was to assess the diagnostic role of CMRI in patients referred for suspected ARVC and its ability to identify ARVC mimics, and to explore its additional clinical impact.

Materials and methods

The CMRI registry data from the year 2014 (January to December) of a UK tertiary centre was analysed retrospectively to identify consecutive patients referred for suspected ARVC. Clinical, ECG and echocardiography data were collected from the clinical records. CMRI was performed on a 1.5 T MRI system (Magnetom Avanto, Siemens Medical Solutions, Enlargen, Germany) and all patients underwent a CMRI protocol including the LV and RV anatomy, cine and late gadolinium enhancement (LGE) images. Cine images were performed using a steady-state free-precession sequence in the four-chamber, three-chamber, and two-chamber long-axis view, followed by a stack of short-axis sections from base to apex; typical image parameters were 38 ms repetition time (TR), 1.07 ms echo time (TE), 80° flip angle, 930 Hz/Px bandwidth, 2×2×8 mm voxel size, 8 mm section thickness, 0 mm inter-section gap. Additional RVOT cine images were obtained, followed by a stack of axial views (5 mm section thickness, 5 mm inter-section gap) through the RVOT from the pulmonary valve to the RV diaphragmatic wall. LGE images were obtained 15–20 minutes after intravenous administration of 0.1 mmol/kg of gadobutrol (Gadovist 1 mmol/ml, Bayer-Schering, Berlin, Germany) in identical planes to the long- and short-axis cine images, using an inversion recovery segmented gradient echo sequence. Typical image parameters were 700 ms TR, 3.15 ms TE, 25° flip angle, 8 mm section thickness, 0 mm intersection gap, 140 Hx/Px bandwidth, 2×1.5×8 mm voxel size. The inversion time was optimised progressively to null normal myocardium (typical values, 250–350 ms). Each section was obtained during a breathhold of 10–15 seconds depending on the patient's heart rate. According to 2010 TFC,[9] CMRI criteria were defined as *major* in the presence of regional RV akinesia/dyskinesia/dyssynchronous contraction, associated with a ratio of RV end-diastolic volume (RVEDV) to body surface area (BSA) of $\geq$110 ml/m^2 (male) or $\geq$100 ml/m^2 (female), or RV ejection fraction (RVEF) $\leq$40%; a *minor* criterion was defined as the presence of regional RV akinesia/dyskinesia/dyssynchronous contraction, associated with ratio of RVEDV to BSA of 100–109 ml/m^2 (male) or 90–99 ml/m^2 (female), or RVEF 40–45%. BSA was calculated using the Du Bois method. The study was reviewed by the local Institutional Research and Innovation Department and in view of its retrospective design a formal ethical approval was waived.

Statistical analysis

Continuous and categorical variables were expressed as mean±SD and n (%), respectively. Continuous data were compared by using the 2-tailed unpaired t test or by using the Mann–Whitney U test. Categorical variables were compared by using the chi-square test or Fisher exact test, as appropriate. A p-value of <0.05 was considered statistically significant; a Bonferroni-corrected p-value was used for comparison of less than two groups. Comparisons between more than two groups were assessed using the Kruskall–Wallis test, using Dunn's test for *post hoc* comparison. Data were analysed with SPSS version 23 (IBM, Bristol, United Kingdom).

Results

Out of 2,481 examinations performed in the CMRI centre between January to December 2014, 124 patients (5%; 56% male, mean age 41±16 years, age range 17–78 years) were identified as having been referred for suspected ARVC. Patients were referred with suspected ARVC/D on the basis of symptoms, family history of ARVC and/or SCD, abnormal ECG, or abnormal transthoracic echocardiogram (TTE). Eighty-five patients (69%) were symptomatic: history of palpitations/arrhythmias was reported in 53 patients (43%), syncope with no documented arrhythmia in 26 (21%), and both history of arrhythmia and syncope in six patients (5%), whereas 39 patients (31%) were asymptomatic, with an abnormal ECG and/or TTE found incidentally during school or competitive sport pre-participation screening or preoperatively. ECG data were available in 65 patients (52%): 53/65 patients (82%) had abnormal ECG, most commonly T-wave inversion in leads V1–V3. Echocardiographic data were available in 96 patients (77%): 26/96 patients (27%) had evidence of abnormal RV on echocardiogram. Family history of SCD was reported in 16 patients (13%), five patients (4%) had family history of ARVC and one patient (1%) had family history of both SCD and ARVC (Table 1).

Table 1
Demographic and clinical characteristics.

Demographic and clinical characteristics	Suspected ARVC n=124	Structurally normal heart n=82 (1)	ARVC mimics n=16 (2)	ARVC by CMRI criteria n=5 (3)	p-Value	p-Value (1 versus 2+3)
Gender, male, n (%)	69 (56)	44 (54)	11 (68)	3 (66)	0.55	0.27
Age, years, mean±SD	41±16	38±16	40±17	44±11	0.48	0.88
Family history of SCD, n (%)	16 (13)	9 (11)	1 (6)	2 (40)	0.14	0.93
Family history of ARVC, n (%)	5 (4)	4 (5)	1 (6)	0 (0)	1.00	1.00
Symptoms, n (%)	85 (69)	55 (67)	10 (63)	4 (80)	0.85	0.80
Documented arrhythmias, n (%)	53 (43)	32 (39)	5 (31)	3 (60)		
Syncope, n (%)	26 (21)	20 (24)	4 (26)	1 (20)		
Arrhythmias + syncope, n (%)	6 (5)	3 (4)	1 (6)	0 (0)		
Abnormal ECG, n (%)	53/65 (82)	36/44 (82) Twi 18 (50) Lbbb 2 (6)	7/9 (78) Twi 2 (29) Lbbb 2 (29) Rbbb 2 (29)	4/4 (100) Twi 2 (50)	1.00	1.00
Abnormal RV on TTE, n (%)	26/96 (27)	21/62 (33)	4/12 (33)	2/4 (50)	0.48	0.65

ARVC, arrhythmogenic right ventricular cardiomyopathy; ECG, electrocardiogram; RV, right ventricle; SCD, sudden cardiac death; TTE, trans-thoracic echocardiogram; TWI, T wave inversion; LBBB, left bundle branch block; RBBB, right bundle branch block.

CMRI findings

Biventricular volumes and function were overall preserved: mean LV ejection fraction (LVEF) was 61±8%, mean LV end-diastolic volume (LVEDV) was 83±24 ml/m^2, and mean LV end-systolic volume (LVESV) was 34±19 ml/m^2; mean RVEF was 58±8%, mean RVEDV 84±23 ml/m^2, and mean RV end-systolic volume (RVESV) was 36±15 ml/m^2. Thirteen patients (10%) had evidence of LGE. Based on CMRI findings, a pathological substrate was found in 36 patients (29%): ischaemic heart disease (IHD) was found in five patients (4%) and non-ischaemic heart disease in 10 (8%); five patients (4%) met CMRI criteria for ARVC (Fig 1), of which one had findings consistent with ALVC, and 16 patients (13%) were ARVC mimics. A structurally normal heart was found in 82 patients (66%) and non-specific findings (mild non-specific regional wall motion abnormalities) in six (5%). CMRI findings are listed in Table 2. Echocardiographic data were available in 96 patients (77%). TTE and CMRI findings agreed in 49 patients (51%); CMRI provided an entirely new diagnosis in 22 patients (22%) and found a structurally normal heart in 20 patients (21%) who had abnormal findings on TTE. One patient (1%) was identified to have an ARVC mimic on TTE, as compared to 12 (13%) identified on CMRI (p=0.01).

ARVC mimics

Sixteen patients (13%) were found to have ARVC mimics on CMR. Six patients had normal variant mischaracterised as ARVC: one patient had a pectus excavatum (Fig 2a and b) and five had findings consistent with athlete's heart. Ten patients had pathological conditions mimicking ARVC: cardiac sarcoidosis (n=1), myocarditis (n=1), RV myocardial infarction (n=1), partial congenital absence of pericardium (n=1; Fig 2c and d); three patients were diagnosed with LV non-compaction (LVNC) and three with pre-tricuspid left-to-right shunting (two atrioventricular septal defect [ASD] and one partial anomalous venous return; Fig 3). There was no significant difference in clinical, ECG, and TTE characteristics between patients with structurally normal hearts on CMRI and those with ARVC and ARVC mimics, and between ARVC and ARVC mimics and the remaining population (Table 1). RVEDV and RV stroke volume (SV) were significantly higher in patients with ARVC (RVEDV p=0.013, RVSV p=0.013) and ARVC mimics (RVEDV p=0.007, RVSV p=0.012), as compared to those with structurally normal hearts. There was no significant difference in RV volumes and function in patients with ARVC and ARVC mimics, while LVESV was significantly larger in patients with ARVC. When comparing patients with ARVC and ARVC mimics (n=21) and the remaining population (n=103), there was no significant difference in clinical, ECG, and TTE characteristics whereas biventricular volumes and RV stroke volume were significantly higher in patients with ARVC and ARVC mimics (RVEDV 79 versus 103 ml/m^2, p=0.001; RVESV 34 versus 47 ml/m^2, p=0.02, RVSV 46 versus 56, p=0.001; Table 2).

Discussion

Arrhythmogenic right ventricular cardiomyopathy is a rare disease, with variable penetrance and prognosis. Given the implications of such a diagnosis, the 2010 TFC recommended a multiparametric approach, comprehensive of imaging findings, family history, arrhythmias, ECG, and histological abnormalities.[9] The symptoms of the disease are non-specific (chest pain, palpitations) and overlap with other cardiomyopathies, thus not being helpful for a definite diagnosis.[3,4,7,8] It is well established that the diagnosis of ARVC cannot rely on imaging findings alone, as imaging is subject to diagnostic pitfalls, such as normal variants mischaracterised for ARVC (i.e., athlete's heart) or pathological conditions mimicking it.[10] Bomma *et al.*[11] showed that <30% of patients referred for ARVC actually met the TFC after a comprehensive clinical, invasive, and non-invasive reassessment. The advent of CMRI offered a new insight into ARVC[12–18]: due to its superior spatial resolution, unique tissue characterisation, increased contrast between blood pool and endomyocardium, and multiplanarity, CMRI is considered the reference standard for the assessment of RV

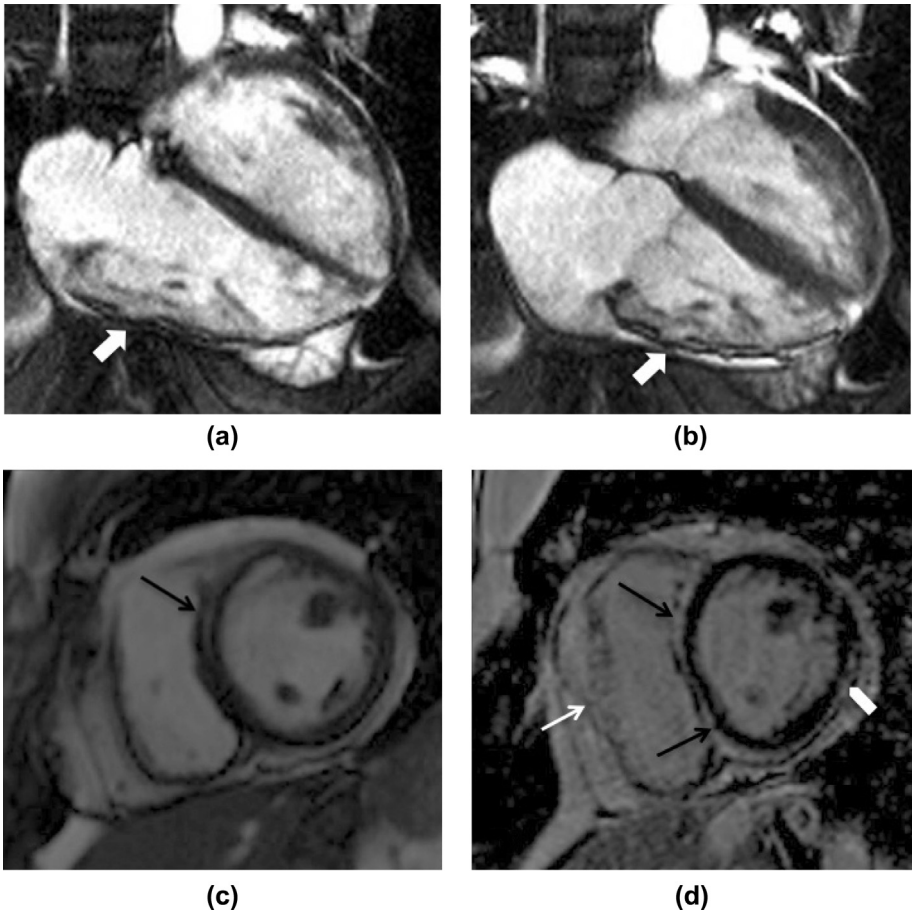

Figure 1 Right and left-dominant arrhythmogenic cardiomyopathy. Top panel: Diastolic (a) and systolic (b) four-chamber view showing dilated RV with bulging of the free wall (solid arrows) in a patient meeting one major CMRI criterion for ARVC. Bottom panel: Mid-cavity short axis cine sequence (c) with evidence of right ventricular free wall late gadolinium enhancement (LGE; d, white arrow) and extensive LGE of the interventricular septum (c and d, black arrows) and left ventricular inferolateral wall (d, white pentagon).

Table 2
Cardiac magnetic resonance imaging (CMRI) findings.

CMRI findings	Suspected ARVC n=124	Structurally normal heart n=82 (1)	ARVC mimics n=16 (2)	ARVC by CMRI criteria n=5 (3)	p-Value All	p-Value (1 versus 2)	p-Value (1 versus 3)	p-Value (2 versus 3)	p-Value (1 versus 2+3)
LVEF, %, mean±SD	61±8	64±6	60±11	56±9	NS				NS
LVEDV, ml/m^2, mean±SD	83±24	79±19	93±40	101±12	NS	NS	0.004	NS	0.03
LVESV, ml/m^2, mean±SD	34±19	29±9	41±35	44±8	0.004	0.04	0.001	0.04	0.02
LVSV, ml, mean±SD	50±12	50±13	52±12	57±14	NS				NS
RVEF, %, mean±SD	58±8	59±6	57±12	55±7	NS				NS
RVEDV, ml/m^2, mean±SD	84±23	80±18	100±29	112±36	0.007	0.007	0.013	NS	0.001
RVESV, ml/m^2, mean±SD	36±15	33±11	45±24	52±22	NS	0.028	0.029	NS	0.02
RVSV, ml, mean±SD	48±12	47±11	54±11	60±15	0.011	0.012	0.013	NS	0.001

LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVSV, left ventricular stroke volume; RVEF, right ventricular ejection fraction; RVEDV, right ventricular end-diastolic volume; RVESV, right ventricular end-systolic volume; RVSV, right ventricular stroke volume; LGE, late gadolinium enhancement; IHD, ischaemic heart disease; NIHD, non-ischaemic heart disease; ARVC, arrhythmogenic right ventricular cardiomyopathy; CMR, cardiovascular magnetic resonance.

volumes and function. The implementation of the new TFC led to a significant reduction in the number of patients confirmed with the diagnosis: Sen-Chowdhry reported an excellent sensitivity but low specificity (29%) of CMRI in relation to the TFC.[19] Similar findings were confirmed by the study of Vermes *et al.*,[20,21] which showed a reduction in the prevalence of major and minor CMRI criteria after the revised TFC. In the present study, only 5/124 patients (4%) referred for suspected ARVC actually met the TFC, in keeping with findings from Quarta *et al.*[22] in a similar cohort. Normal and pathological conditions mimicking ARVC make the diagnosis even more challenging. Chest wall deformity and non-ARVC-related fatty infiltration (obesity, lipomatous metaplasia post-myocardial infarction) could be

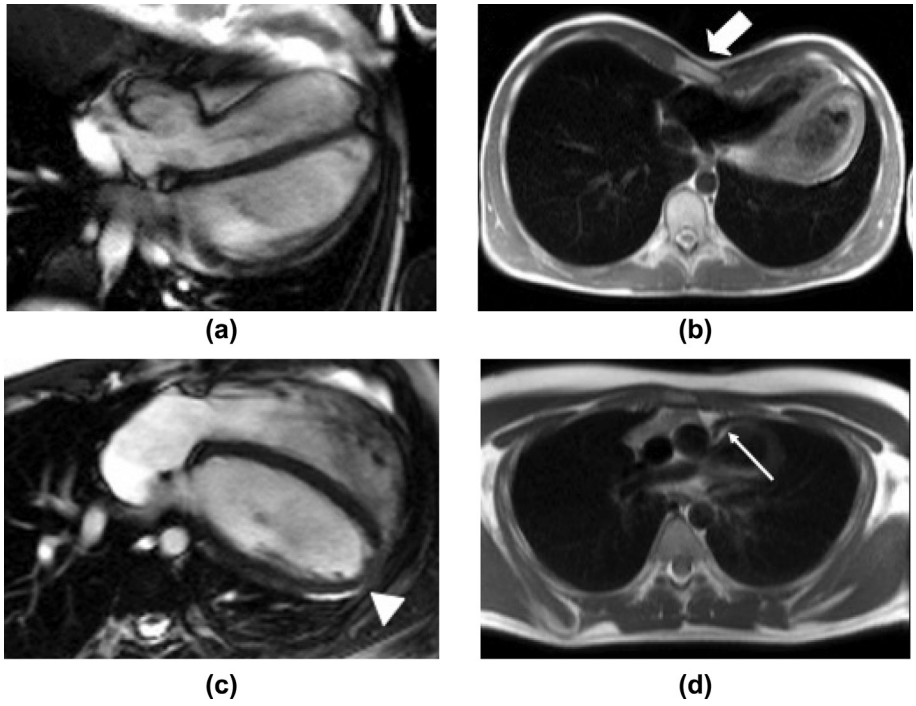

Figure 2 Abnormal right ventricular features mimicking ARVC. Four chamber long axis cine view showing a distorted RV (a) in a patient with pectus excavatum (b, solid white arrow). Four chamber long axis cine view showing heart displacement towards the left with cardiac apex pointing posteriorly (c, white arrowhead) and evidence of lung interposition between the aorta and the pulmonary artery (d, white arrow) in a patient with partial congenital absence of the pericardium.

misinterpreted as ARVC. Moreover, increased RV volumes in athlete's heart or pre-tricuspid shunting often lead to misdiagnosis.[22–27] In the present study, 16/124 patients (13%) were found to have ARVC mimics, which were mainly represented by pathological conditions rather than normal variants mimicking the disease, leading to important clinical implications. In the present cohort, the prevalence of ARVC mimics was slightly higher compared with those previously reported in literature: Quarta *et al.*[28] reported a 5% prevalence of ARVC mimics among patients referred for CMRI for suspected ARVC, with similar findings confirmed by Ting *et al.*,[29] which showed a 4.4% prevalence of ARVC mimics. As CMRI is part of the multimodality assessment in patients with suspected ARVC, it is increasingly used in clinical practice, especially due to the potential clinical and prognostic implications that such a diagnosis would carry, and sometimes it is performed to definitely rule out ARVC also in cases where pre-test likelihood is low; this might, at least in part, explain the higher prevalence of ARVC mimics in the present cohort. The ability of TTE to identify ARVC mimics was also assessed, and it was found that CMRI was significantly superior (13% by CMRI versus 1% by TTE, $p=0.01$). Although RV volumes were bigger in patients with ARVC and ARVC mimics, as compared to the remaining population, the lack of difference among clinical, ECG, TTE, and CMRI characteristics between ARVC and ARVC mimics, makes it challenging to identify ARVC mimics in the early differential diagnosis. Interestingly, 82/124 patients (66%) with suspected ARVC based on clinical assessment showed a structurally normal heart on CMR. The present study confirms and extends previous findings and highlights the limitations of the TFC that do not consider the occurrence of ARVC mimics. Tissue characterisation by CMRI, including LGE, might help in the differential diagnosis; however, to date, tissue characterisation is not currently included among the TFC. The main limitation of the present study was the retrospective design; moreover, neither endomyocardial biopsy (given its little access at Bristol Heart Institute) nor genetic testing was available in the present cohort. As ARVC is a rare disease, prospective multicentre studies are needed to confirm and expand the present findings, with the aim of improving the generalisability of the results.

In conclusion, out of 2,481 CMRI examinations performed at Bristol Heart Institute over a year, 124 (5%) were performed for suspected ARVC. Based on CMRI findings, a pathological substrate was found in 29% of patients and a structurally normal heart in 66%. ARVC imaging criteria were met in only 4% of patients, while 13% of patients showed findings consistent with ARVC mimics. CMRI was superior to TTE in the identification of ARVC mimics (13% versus 1%, $p=0.01$) and, overall, provided a change in diagnosis in 22% of patients. Accurate identification of the underlying pathology in patients with suspected ARVC is pivotal given the impact on clinical management and prognosis. The present study shows the incremental role of CMRI in the identification of ARVC mimics, over and above TTE.

Conflicts of interest

The authors declare no conflict of interests.

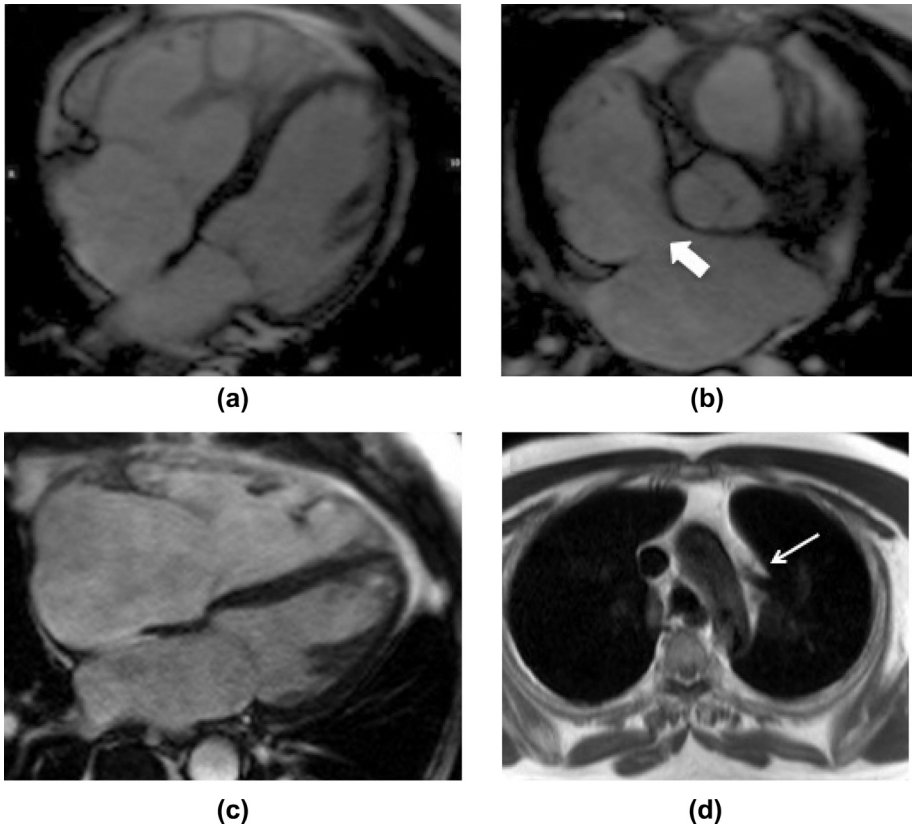

Figure 3 Pre-tricuspid shunting mimicking ARVC. Four-chamber long axis view showing dilated RV (a) in a patient with evidence of atrial septal defect and left-to-right shunting on the short axis view (b, solid arrow). Four-chamber long axis cine view showing dilated RV with septal flattening, in keeping with RV overload (c) in a patient with left upper pulmonary vein (d, white arrow) draining into the brachiocephalic trunk.

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