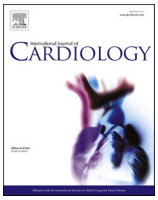
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A new Dawn: Advances and innovation in heart transplantation part I - cardiac magnetic resonance imaging in heart transplantation

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ABSTRACT

Heart transplantation (HTx) remains the treatment of choice for patients with advanced heart failure. HTx recipients require intensive surveillance over time. Major complications that should be monitored, prevented, and treated in a timely manner include graft dysfunction secondary to cellular rejection or antibody-mediated rejection (AMR) and coronary allograft vasculopathy (CAV). While echocardiographic surveillance plays a key role in detecting major changes in graft function and endomyocardial biopsy remains the most widely used invasive technique for the surveillance of graft rejection, the role of cardiac magnetic resonance (CMR) imaging is expanding as a complementary non-invasive imaging modality capable of identifying structural, functional, and tissue characterization abnormalities associated with several graft complications. Providing detailed characterization of myocardial tissue, CMR may facilitate early detection of abnormalities suggestive of graft dysfunction and rejection through advanced techniques such as parametric mapping to assess edema, while also enabling longitudinal surveillance of fibrosis, adverse remodeling, and treatment response. Combining CMR with novel blood-based assays, such as donor-derived cell-free DNA, has the potential to improve non-invasive surveillance of late cellular rejection or AMR. Inevitable ischemic injury occurring during transplantation may affect CMR findings, even in the absence of other significant conditions, with late gadolinium enhancement (LGE) detection in 62% of rejection-free HTx recipients in the first year after HTx, with a low LGE extent of 2% on average, making the interpretation of pathological findings more difficult or uncertain compared with native hearts. In this review, we summarize the current evidence on the role of CMR in the follow-up of HTx recipients, highlighting its strengths, limitations, and complementary role in the assessment of graft dysfunction, rejection, and CAV.

1. Introduction

Heart transplantation (HTx) remains the treatment of choice for patients with advanced heart failure (HF), although its application is limited by the donor scarcity and contraindications in potential candidates [1,2]. Survival after HTx has significantly improved, with 1-year survival rates ranging from 80 to 90% and 10-year survival rates from 50 to 60% [3]. In parallel, the number of HTx continues to increase, with >7000 performed worldwide every year, the majority in Europe and the US [3]. Therefore, a significant number of HTx patients require follow-up evaluations over time.

Current guidelines suggest intensive follow-up during the first year after HTx, followed by a gradual reduction in monitoring frequency [4]. Long-term success of HTx relies on adequate follow-up programs at

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specialized centers, aimed at detecting graft dysfunction (GD), acute cellular rejection (ACR, a T-cell–mediated immune response against the graft), antibody-mediated rejection (AMR, caused by donor-specific antibodies (DSA) targeting the graft and associated with late GD), coronary allograft vasculopathy (CAV), and non-cardiovascular complications [4].

Among the diagnostic tools, routine echocardiographic surveillance plays a key role in detecting major changes in graft function [4]. Invasive coronary angiography is also recommended annually or biannually as the current invasive reference method for early detection of CAV, while the latest guidelines suggest complementary coronary computed tomography as a non-invasive alternative for CAV detection based on its high diagnostic accuracy [4,5]. Eventually, endomyocardial biopsy (EMB), which is frequently performed during the first year after HTx, remains the most widely used invasive technique for the histopathological assessment of graft rejection [4]. However, it is invasive, resource-intensive, and associated with procedural risks. Moreover, it has well-recognized limitations, including sampling error due to the focal and heterogeneous distribution of rejection, interobserver variability in histopathological grading, and its inability to comprehensively assess the entire myocardium. Therefore, interest in non-invasive assessment has recently increased, with multiple blood-based assays under study [4,6]. *Donor-derived cell-free DNA (dd-cfDNA)* is released by the allograft and is distinguishable from the recipient cfDNA, with results representing a fraction of the total circulating cfDNA. High levels of dd-cfDNA have been shown to correlate with graft rejection and have high negative predictive values (NPV) as early as 14 days after HTx [6,7]. *Gene expression profiling*, on the other hand, evaluates the expression of 11 genes correlated with the immune response and, when elevated, has been shown to distinguish between high-grade ACR and immune system quiescence starting on day 55 after HTx [6]. Both methods have been proposed as adjuncts to non-invasive rejection surveillance, although more evidence is needed to better elucidate their combined routine use and their ability to distinguish between ACR and AMR [4,6]. Monitoring of *anti-HLA antibodies* starting from month 1 after HTx has also been recommended as a non-invasive tool for rejection surveillance, since up to 30% of patients may develop DSA, which are linked to AMR and graft loss [4,8]. However, the significance of DSA detection in the absence of GD still needs to be elucidated, with no definitive recommendation available regarding the need for treatment [8]. Eventually, the use of high-sensitivity troponin and natriuretic peptides has also been suggested as complementary tools for identifying patients at higher risk of rejection during periodic surveillance, with greater emphasis on within-individual changes than on absolute values [4].

In addition to blood-based assessments, cardiac magnetic resonance (CMR) has emerged as a useful non-invasive tool for detecting functional and structural changes in the transplanted heart [6]. Providing detailed characterization of myocardial tissue, CMR may facilitate the early diagnosis of myocardial abnormalities associated with GD and allograft rejection, using advanced tissue characterization techniques, including parametric mapping for edema assessment and longitudinal surveillance of fibrosis, adverse remodeling, and treatment response. Nevertheless, the peculiar setting of HTx makes CMR interpretation more difficult. Inevitable ischemic injury occurring during transplantation may affect CMR findings, even in the absence of clinically significant graft pathology. For example, late gadolinium enhancement (LGE) may be detected in 62% of HTx recipients at a median of 6.4 months after HTx, with a low LGE extent of 2% on average and an interquartile range (IQR) of 0–7% in rejection-free patients [9]. Moreover, transplanted hearts show higher T1 and T2 mapping values in the immediate post-transplant period, with a progressive decrease over 1–2 years of follow-up [9–12].

In this review, we summarize the current evidence on the role of CMR in the follow-up of HTx recipients, focusing on its applications for detecting rejection, CAV, GD, and other post-transplant complications, while highlighting its strengths and limitations (Fig. 1).

2. Acute cellular and antibody-mediated rejection

ACR is a T-cell-mediated process that affects about 30% of patients in the first year after HTx. It is typically reversible with intensification of immunosuppressive therapy and is graded based on histological findings

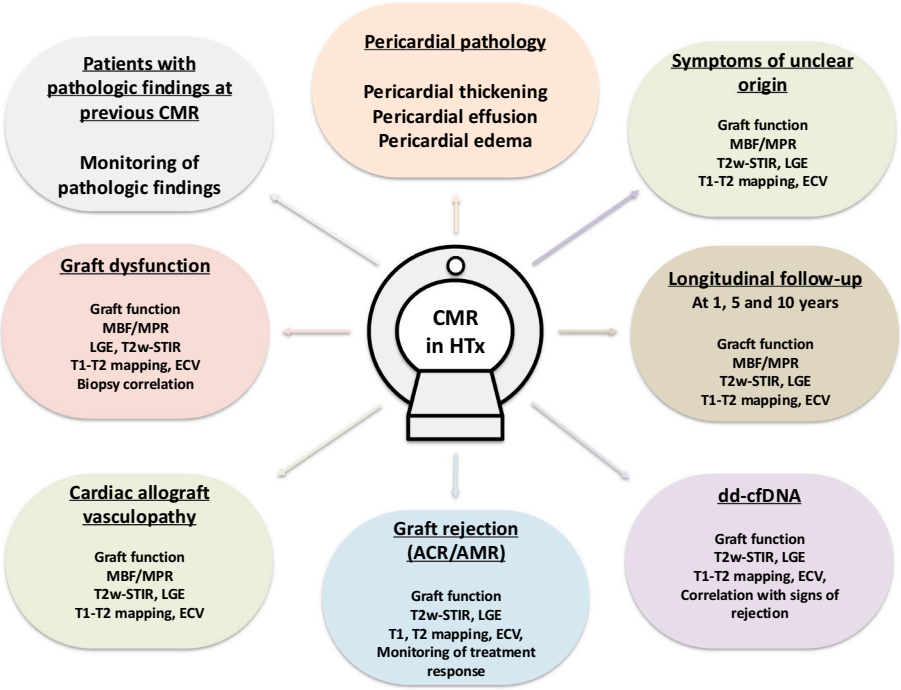


Fig. 1. Role of cardiac magnetic resonance (CMR) in heart transplant (HTx) surveillance. ACR indicates acute cellular rejection; AMR, antibody-mediated rejection; dd-cfDNA, donor-derived cell-free DNA; ECV, extracellular volume; LGE, late gadolinium enhancement; MBF, myocardial blood flow; MPR, myocardial perfusion reserve; T2-STIR, T2-weighted short Tau inversion recovery.

on EMB. Grading follows the 2004 International Society for Heart and Lung Transplantation (ISHLT) classification and includes 4 stages: 0R, no rejection; 1R, mild rejection with a maximum of one focus of myocardial damage; 2R, moderate rejection with two or more foci of myocardial damage; 3R, severe rejection with diffuse infiltrates and multifocal myocyte damage [13].

AMR, which may coexist with ACR, involves humoral immune mechanisms and may rarely present as hyperacute AMR in cases of preformed antibodies against the donor's heart or as acute symptomatic AMR. More frequently, AMR may present as asymptomatic with histopathologic (e.g., intravascular macrophage activation, endothelial cell edema) and/or immunopathologic (e.g., antibody deposition or complement activation) findings at EMB, even in the absence of GD [4,14]. AMR grading relies on the 2013 ISHLT classification and includes 4 stages: pAMR 0, negative histopathologic and immunopathologic findings; pAMR 1, positive histopathologic (H+) or immunopathologic findings (I+); pAMR 2, positive histopathologic and immunopathologic findings; pAMR3, severe pathologic AMR with interstitial hemorrhage [14]. This condition may become chronic and contribute to CAV development and late GD. Its diagnosis is also supported by non-invasive methods, including dd-cfDNA and DSA [4,8].

Although EMB remains the most widely used invasive technique, CMR has also been investigated as a non-invasive alternative for rejection surveillance, with most studies on CMR after HTx focusing on this topic (Table 1) [15]. In addition to providing information on structural and functional measures of the transplanted heart, CMR's diagnostic value mainly relies on multiparametric tissue characterization. Notably, this approach parallels the 2018 updated Lake Louise Criteria for myocarditis, as the combined assessment of T1- and T2-based markers enables detection of immune-mediated injury and inflammation, which are similar to the pathophysiological processes underlying rejection (Fig. 2) [16]. The diagnosis of myocarditis is based on at least one T2-based criterion (global or regional increase of myocardial T2 relaxation time or an increased signal intensity in T2-weighted CMR images, with at least one T1-based criterion, including increased myocardial T1, extracellular volume (ECV), or LGE. While having both a positive T2-based marker and a T1-based marker increases specificity for diagnosing acute myocardial inflammation, having only one (i.e., T2-based or T1-based) marker may still support a diagnosis of acute myocardial inflammation in an appropriate clinical scenario, albeit with reduced specificity [16]. However, evidence remains limited, and CMR cannot reliably assess rejection severity or differentiate between AMR and ACR; histopathological evaluation is still required [4], although segmental CMR strain analysis has recently been shown to improve diagnostic accuracy for detecting high-grade AR in an observational study [17].

Currently, only one randomized controlled trial comparing a CMR-based approach to standard EMB-based surveillance in the first year after HTx has been conducted, with similar clinical results, lower infection and hospitalization rates in the CMR group, and a 94% reduction in the need for EMB performance [18]. In this study, a CMR-based multiparametric assessment was implemented, including both T1 and T2 mapping, which demonstrated good reproducibility and promising diagnostic performance to identify patients with ACR-related myocardial abnormalities, with greater specificity and negative predictive value than either parameter alone [18]. Similarly, the combination of T2 mapping and ECV has been suggested as a noninvasive tool to evaluate AR; although up to 100% sensitivity and specificity have been reported in an observational study, these findings require validation in larger populations and are limited by small sample size [19].

2.1. Single CMR parameters to evaluate suspected AR

High *native T1 mapping values*, suggestive of interstitial fibrosis, edema, and inflammation, have been shown to be useful parameters for detecting myocardial abnormalities associated with graft rejection, and T1 mapping has a high NPV for excluding high-grade ACR, especially during the first year after HTx [9,17,18,20,21]. In the study by Sade et al., T1 mapping times also showed a significant decrease after rejection treatment, from 1105 ms (1054 to 1141 ms) to 1085 ms (1051 to 1096 ms; $p = 0.02$), confirming a correlation with ACR [21].

Elevated T2 mapping values correlate with the presence of edema in AR and have high sensitivity and negative predictive value for AR [17,18,22–24], reflecting myocardial edema and water content, although thresholds vary among scanners, sequences, and acquisition protocols. Some studies have also shown that T2 mapping values can be used to monitor treatment response during AR [18].

Elevated *ECV values*, with varying cut-offs across cohorts, have also been associated with AR, although they perform worse than other parameters when used alone [19,21,22,25].

The role of *LGE*, a typical marker of fibrosis, in AR [26] remains unclear. Preliminary studies have shown no direct correlation between the presence of LGE and AR [27,28], although recent evidence has found higher LGE extent, with a typical patchy appearance, among HTx recipients with a history of ACR, possibly reflecting cumulative injury [9]. Moreover, extensive LGE may guide further investigations to detect causes of myocardial injury, as it is a strong predictor of prognosis [29].

Overall, as demonstrated in a meta-analysis from Han et al. [25] T2 mapping has the highest sensitivity and specificity for AR diagnosis (86.5 with 95% confidence interval [CI] of 72.1–94.1 and 85.9 with 95% CI of 65.2–94.6, respectively, area under the curve (AUC) 0.92 [95%CI: 0.89–0.94]), followed by T1 mapping (84.6 [95%CI: 65.6–94.0] and 70.1 [95%CI: 54.2–82.2], AUC 0.84 [95%CI: 0.81–0.87]) and ECV assessment (91.3 [95%CI: 63.9–98.4] and 67.6 [95% CI: 56.1–77.4], AUC 0.78 [95%CI: 0.74–0.81]), with LGE having the lowest accuracy (50.1 [95%CI: 31.2–68.9] and 60.2 [95%CI: 36.7–79.7], AUC 0.56 [95% CI: 0.51–0.60]). However, these findings should be interpreted cautiously because of heterogeneity in imaging protocols, patient populations, rejection definitions, and study designs.

Regarding the differential diagnosis between AMR and ACR, CMR still has to demonstrate the ability to discriminate between the two, with no definitive imaging recommendation available [30]. Studies to detect specific CMR markers of AMR are currently ongoing and aim to integrate T1 and T2 mapping with clinical characteristics, dd-cfDNA measurement, and DSA monitoring (CARdiac Imaging and BIomarkers in the Diagnosis of Antibody Mediated Rejection in Heart Transplantation [CARIBIAM], clinicaltrials.gov identifier NCT06774365).

Overall, these findings provide encouraging evidence supporting the potential role of CMR in non-invasive assessment of HTx rejection. However, most studies suffer from limited sample sizes, observational designs, variability in CMR techniques, and the presence of multiple rejection phenotypes, leading to a lack of unanimity on which single technique would be most useful. Multiparametric evaluation, including T1 and T2 mapping, may optimize the use of CMR in the evaluation of rejection and provide additional information, especially in cases with negative findings at EMB and concomitant alterations in other parameters (e.g., DSA and dd-cf-DNA), which correlate with worse clinical outcomes [31]. Importantly, many studies have evaluated CMR parameters against EMB findings; therefore, reported sensitivity, specificity, positive predictive value, NPV, and AUC values should be interpreted as measures of concordance with EMB-defined rejection rather than as independent validation of CMR for the detection of rejection. Conversely, studies evaluating associations between CMR parameters and clinical outcomes provide complementary evidence of their potential prognostic value.

2.2. Cardiac allograft vasculopathy

CAV is a major determinant of long-term survival after HTx, with a prevalence of approximately 30% at 5 years and 50% at 10 years [32]. It is characterized by diffuse, rapidly progressive involvement of the coronary vasculature, driven by interactions among multiple immune and metabolic factors and donor characteristics. CAV affects both arterial

Table 1
Main studies on the role of CMR in HTx recipients.

Author, Year	Study type	N° patients	Timing post-transplant	CMR parameters	Results
Acute cellular and antibody-mediated rejection					
Anthony et al., 2022 [18]	Randomized control trial	40	Baseline CMR 4 weeks after HTx and CMR follow-up 52 weeks after HTx.	T2, RVEDVi, T1, ECV, LGE	CMR multiparametric assessment achieved 93% sensitivity, 92% specificity and 99% NPV for detecting AR at EMB, with greater specificity and NPV than either T1 or T2 mapping alone.
Han et al., 2021 [25]	Meta-analysis	564 (10 studies)	Various stages post-HTx	T1, T2, ECV, LGE	T2 mapping: sensitivity 86.5%, specificity 85.9%, AUC 0.92. T1 mapping: sensitivity 84.6%, specificity 70.1%, AUC 0.84. ECV: sensitivity 91.3%, specificity 67.6%, AUC 0.78. LGE: sensitivity 50.1%, specificity 60.2%, AUC 0.56. T2 demonstrated highest diagnostic accuracy for AR at EMB.
Taylor AJ et al., 2010 [27]	Prospective study	50	Various stages post-HTx	T2-weighted STIR imaging, early relative myocardial contrast enhancement, RVEDVi	Multisequential CMR combining early relative myocardial contrast enhancement or increased T2 STIR signal intensity yielded sensitivity and specificity of 100% and 73% for AR at EMB/echocardiography.
Butler et al., 2015 [24]	Prospective study	60	Mean 41 ± 47 months (~3.4 years)	T2, RVEDVi, LV function	T2 and RVEDVi were independent predictors of positive EMB. Combined threshold values predicted positive EMB with sensitivity 93%, specificity 78%, PPV 52%, NPV 98%. CMR more sensitive than EMB for clinical rejection (67% vs 58%).
Şimşek E et al., 2016 [28]	Prospective study	41	No LGE group: 31.8 ± 22.5 months; LGE group: 43.2 ± 27.4 months	LGE	LGE showed limited diagnostic performance for acute cellular rejection at EMB (grade ≥ 1R). Sensitivity 50.1%, specificity 60.2%.
Pedrotti et al., 2017 [29]	Prospective study	48	Various stages post-HTx	LGE	LGE detected in 54%. Independent predictors of MACE: CAV (HR 3.63, $p = 0.0039$), LGE mass (HR 1.04, $p = 0.0007$), LGE % of LV mass (HR 1.083, $p = 0.0002$). Independent predictors of death: CAV (HR 6.33, $p = 0.0201$), LGE mass (HR 1.04, $p = 0.005$), LGE % (HR 1.075, $p = 0.007$)
Vermes et al., 2018 [22]	Prospective study	20	Median 80 days (range 56–156 days)	T2, T1, ECV	Basal T2 > 57.7 ms: sensitivity 71%, specificity 96%, accuracy 90% for AR detection at EMB. Basal ECV >32%: sensitivity 86%, specificity 85%. Combined T2 + ECV: 100% sensitivity, 100% NPV.
Dolan et al., 2019 [15]	Prospective study	61	5.5 ± 5.7 yrs	T1, T2, ECV	T2 (50.5 ± 3.4 vs 45.2 ± 2.3 ms, $p = 0.01$) and T1 (1037.8 ± 48.0 vs 993.8 ± 34.1 ms, p = 0.01) elevated vs controls. Impaired systolic longitudinal velocities (2.9 ± 1.1 vs 5.1 ± 1.2 cm/s, p = 0.01). T2 correlated with ECV ($r = 0.45$, p = 0.01) and inversely with peak systolic velocities ($r = -0.29$, $p = 0.02$)
Dolan et al., 2019 [19]	Prospective study	58	Mean 3.8 ± 4.6 years (45% CMR scans performed <1 yr, 39% 1-6 yrs., 17% ≥7 yrs)	T1, T2, ECV, strain	Global ECV highest AUC (0.80) with 100% sensitivity at 26.8% cutoff for diagnosis AR at EMB. Combined model (age + T2 + ECV): AUC 0.84.
Sade et al., 2019 [21]	Prospective study	49	Rejection: 3 months (IQR 3–36); No rejection: 12 months (IQR 36–84)	T1, ECV	T1 ≥ 1090 ms: 100% sensitivity, 100% NPV for ≥2R ACR at EMB.
Imran et al., 2019 [20]	Prospective study	112	89% CMR scans performed <1 yr, mean time 2.5 ± 1.3 months; 11% CMR scans performed >12 months	T1	T1 significantly higher in grade 2R/3R/AMR (1066 ± 78 ms) vs grade 0 ACR (984 ± 42 ms, $p = 0.0001$) vs grade 1R ACR (1001 ± 54 ms, $p = 0.001$) at EMB. T1 cutoff 1029 ms: sensitivity 93%, specificity 79%, NPV 99%
Taipale et al., 2025 [9]	Prospective study	58 (244 scans)	Serial scans: 1, 3, 6, 12, 24 months post-transplant	T1, T2	T1 elevated during ACR at EMB in children (1188 vs 1079 ms, $p = 0.001$) and adults (1087 vs 1016 ms, $p = 0.007$). T1 and T2 decreased over 24 months post-transplant.
Zhou et al., 2025 [23]	Prospective study	51	Mean 3.9 ± 3.3 years	T1, T2, GLS	T2 (50.9 ± 2.7 vs 45.7 ± 4.3 ms, p<0.001) and GLS (−9.7 ± 3.1% vs −13.1 ± 2.9%, p<0.001) independently associated with ≥2R ACR at EMB.
Pouliopoulos et al., 2025 [17]	Prospective study	54	Mean 9.2 ± 15.9 months	Strain (GLS, GCS, GRS), T1, T2	GCS (AUC 0.852, NPV 98.3%) and GRS (AUC 0.826, NPV 95.8%) superior to GLS for ≥2R ACR detection at EMB. Multiparametric models: T2 + GRS (AUC 0.931, NPV 98.2%), T1 + T2 + GCS (AUC 0.943, NPV 97.5%).
Cardiac allograft vasculopathy					
Nunoda et al., 2010 [33]	Prospective Study	22 (13 MDCT only, 16 MRCA only, 7 both)	Various stages post-HTx	Whole-heart MRCA	MRCA: sensitivity 60%, specificity 100%, PPV 100%, NPV 92.2% for CAV detection.
Miller et al., 2014 [37]	Prospective Study	48	Median 7.1 years (IQR 4.6–10.3 years)	MPR, LV function, tissue characterization	MPR was the only independent predictor of both epicardial (β = −0.57, p = 0.001) and microvascular disease (β = −0.60, p = 0.001).

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Table 1 (*continued*)

Author, Year	Study type	N° patients	Timing post-transplant	CMR parameters	Results
					MPR outperformed angiography for detecting moderate CAV (AUC 0.89 vs 0.59, $p = 0.01$) and severe CAV (AUC 0.88 vs 0.67, $p = 0.05$)
Erbel et al., 2016 [35]	Prospective Study	63	Various stages post-HTx	MPRI, strain-encoded CMR (systolic strain, diastolic strain rate)	Patients with cardiac allograft microvasculopathy had significantly reduced MPRI (1.3 ± 0.3 vs 2.6 ± 0.6 in controls, p 0.001) and impaired diastolic strain rate (55 ± 18/s vs 95 ± 25/s, p 0.001). Systolic strain was preserved (−18 ± 5% vs −21 ± 4%). MPRI 1.5 was independent predictor of microvasculopathy (AUC 0.92). MPRI correlated with adverse outcomes during follow-up.
van Heeswijk et al., 2020 [42]	Prospective Study	27	Various stages post-HTx	T1, ECV	T1 and ECV increased in XRCA-CAV+ patients ($p = 0.03$ each). ECV correlated with EMB interstitial fibrosis ($\rho = 0.46$, $p = 0.02$).
Duran et al., 2021 [62]	Prospective Study	16	Mean 84.7 ± 40.0 months	MPR	MPR in CAV patients lower vs no CAV (1.43 vs 1.48, $p = 0.49$), non statistically significant. Patients within 6 yrs. since HTx non-significant trend toward a higher MPR (median: 1.57) vs patients with older HTx (median: 1.47; $P = 0.46$)
Husain et al., 2021 [34]	Retrospective Study	26 HTx + 18 controls	Median 5.1 years (IQR 7.5 years)	MPRI, T1, T2, ECV, strain	MPRI lower in PHT vs controls (0.69 ± 0.21 vs 0.94 ± 0.22, p<0.001). Low-grade CAV associated with higher T1, ECV, and T2.
Patel et al., 2022 [63]	Retrospective Study	38 (16 s/p HTx)	Various stages post-HTx	MPRI	Global MPRI of the 16 HTx patients was significantly lower than the non-transplant patients (0.75 ± 0.22 vs. 0.92 ± 0.23, $p = 0.03$)
Kikano et al., 2024 [43]	Prospective Study	59	Various stages post-HTx	GCS, T1, T2, ECV	CAV patients had worse GCS vs healthy (− 26.6% vs - 30-9%, $p = 0.019$) with normal global function. T1, T2, ECV no significantly different compared to healthy PHT.
Quinn et al., 2025 [41]	Retrospective Study	51	Mean 3.2 ± 1.5 years	T1, T2, ECV, feature-tracking strain	CAV+ patients: higher T2 (54.5 ± 7.7 vs 50.2 ± 3.3 ms), higher ECV (31.3 ± 5.3% vs 27.4 ± 4.1%), attenuated strain rates. Combined model (ECV + strain rates): AUC 0.85 for CAV detection
Jablonowski et al., 2025 [36]	Retrospective Study	110	Various stages post-HTx	Quantitative stress perfusion, MPR	Gradual decrease of stress MP and MPR with increased CAV grade. MPR discriminated CAV 2–3 with AUC 0.88. MPR cutoff 2.2: sensitivity 100%, specificity 68%, NPV 100%
Graft dysfunction					
Chaikriangkrai et al., 2020 [12]	Prospective Study	99	T1 cohort: median 5.1 years (IQR 2.0–8.0); T2 cohort: median 6.3 years (IQR 3.9–8.7)	Native T1, post-contrast T1, ECV, T2 mapping, LGE	Mean global native T1: 1030 ± 56 ms, ECV: 27 ± 4%, T2: 50 ± 4 ms. ECV >29% independently predicted adverse outcomes (HR 2.87, $p = 0.04$). T2 ≥ 50.2 ms independently predicted adverse outcomes (HR 3.01, $p = 0.005$) and cardiac events (HR 4.92, p = 0.005).
Yang Y et al., 2023 [10]	Prospective Study	21 HTx, 25 controls	3, 6, and 12 months	T1, T2, ECV, strain (GLS, GCS, GRS)	At 3 months: decline in LVGRS ($p = 0.02$, RVGLS (p<0.001); elevated native T1, T2, ECV (all $p = 0.001$). At 12 months: native T1, ECV, LVGLS remained abnormal vs controls. ECV correlated with LVGLS, LVGCS, LVGRS. T2 normalized at 6 months.
Hughes et al., 2019 [40]	Retrospective Study	152	Mean 5.0 ± 5.4 years	LGE (presence and extent)	Myocardial fibrosis present in 18% (37% infarct pattern, 41% non-infarct, 22% both). Fibrosis independently associated with death or MACE (HR 2.88, 95% CI 1.59–5.23, p<0.001). Every 1% increase in fibrosis associated with 6% higher hazard (HR 1.06, p = 0.001).
Shenoy et al., 2020 [45]	Retrospective Study	152	Mean 5.0 years	CMR-FT GLS	Median GLS: −11.6% (IQR −13.6% to −9.2%). GLS independently associated with death/MACE: HR 1.15 per 1% worsening (95% CI 1.06–1.24, p = 0.001) after adjustment for CAV, rejection history, LVEF, RVEF, and myocardial fibrosis.
Barrett CM et al., 2024 [46]	Retrospective Study	257	Median 4.3 yrs. (IQR 1.1–10.0)	RVEF, RVGLS (feature-tracking)	RVGLS independently associated with death/MACE: HR 1.08 per 1% worsening (95% CI 1.00–1.17, $p = 0.046$). RVEF not independently associated. RVGLS provided incremental prognostic value over RVEF and other variables.
Anand S et al., 2024 [44]	Retrospective Study	34	Median time from HTx to GD: 1.3 (IQR 0.8–4.7) years	LGE, T1 mapping, T2 mapping, ECV	LGE present in 46% (16/34). LGE+ patients developed GD later after HTx: 4 (1.4–6.8) vs 0.8 (0.3–1.2) years (p<0.001). LGE+ more often symptomatic: 88% vs 56% ($p = 0.06$). LGE+ had greater hemodynamic derangement: PCWP 19 ± 7 vs 13 ± 3 mmHg ($p = 0.002$). No difference in primary composite outcome: 50% vs 38% events ($p = 0.52$). At median 3.8-year of follow up, LVEF improved similarly in LGE- (37 → 55%) and LGE+ (32 → 55%) groups ($p = 0.16$)

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Table 1 (*continued*)

Author, Year	Study type	N° patients	Timing post-transplant	CMR parameters	Results
CMR and dd-cfDNA					
Taipale MA et al., 2025 [9]	Prospective Study	58	Serial scans: 1, 3, 6, 12, 24 months post-transplant	T1 mapping, T2 mapping, GLS	T1 positively associated with dd-cfDNA (p<0.001). T2 positively associated with dd-cfDNA ($p = 0.014$). T2 correlated with worse GLS (p<0.001). During rejection: T1 elevated in children (1188 vs 1079 ms, p0.001) and adults (1087 vs 1016 ms, $p = 0.007$).
Post-transplant pericardial disease					
Lloyd JW et al., 2021 [52]	Retrospective surgical series	8	Median 1.7 years (range 0.8–18.1) to symptomatic CP	T1	Cross-sectional imaging (CMR or CT) performed in 6/8: ≥mild pericardial thickening in 100% (6/6).
Pediatric recipients					
Feingold et al., 2017 [61]	Prospective study	25	Mean 10.7 ± 6.5 years	ECV, histologic CVF	Mean ECV: 25.1 ± 3.0% (vs 23.7 ± 2.0% in controls, $p = 0.09$). ECV moderately correlated with histologic CVF ($r = 0.47$, $p = 0.019$). Log-transformed BNP correlated with ECV ($r = 0.46$, $p = 0.02$). No associations with hemodynamics, ischemic time, or time since transplant.
Sethi N et al., 2020 [56]	Prospective study	11	Mean 8.18 years	Quantitative T2 mapping	Increase in the T2-relaxation time observed at higher EMB ACR grades. T2 values in cases of histologic or clinical rejection were statistically significantly higher than in non-rejection cases. Mean T2 value: 58 ± 4 ms in rejection group vs 53 ± 2 ms in non-rejection group ($p = 0.001$)
Soslow JH et al., 2022 [57]	Prospective Study	30	AR group: median 5.7 yrs. (IQR 2.6,12.5). NoAR group: median 2.1 yrs. (IQR 1.0, 4.0)	T1, T2, ECV, LGE	T2 Z scores and ECV were increased in AR versus NoAR (Global T2 Z score 3.7 vs 0.6, $p = 0.009$; Global ECV 34.9% vs 27.2%, $p = 0.004$). No significant difference in native T1 Z score (Global native T1 Z score 3.3 vs 2.3, $p = 0.18$)
Ceneri NM et al., 2024 [55]	Prospective Study	21	Median 6.3 years (IQR 5.2–7.9) [Treated Rejection] vs 7.7 years (IQR 4–11.3) [No Rejection]	T1. T2	Peak T1 higher in treated AR vs noAR patients (1086 ms vs 1066 ms, $p = 0.021$). Peak T1 in trated AR decreased with treatment (1086 vs 1052 ms, $p = 0.028$). After treatment, no significant difference between the groups (1052 vs 1066, $p = 0.7$). No significant difference in the mean or peak T2 values (mean: 48 ms vs 50 ms, $p = 0.398$; peak 52 ms vs 53 ms, $p = 0.866$)
Richmann DP et al., 2022 [59]	Retrospective study	24	Mean 5.3 ± 4.1 yrs	T1	Peak and mean T1 significantly associated with AR with monotonic trend with increased rejection grade.
Lawson AA et al., 2023 [58]	Retrospective study	59	Mean 7.3 ± 5.0 yrs	LGE, functional parameters	LGE present in 36% (29/81). LGE+ patients: older (16.7 ± 2.9 vs 12.8 ± 4.6 years, $p = 0.001$), longer time since HTx (8.3 ± 5.4 vs 5.7 ± 3.9 years, $p = 0.041$), higher LVESVI (34.7 ± 11.7 vs 28.7 ± 6.1 ml/m^2, $p = 0.011$), lower LVEF (56.2 ± 8.1 vs 60.6 ± 5.3%, $p = 0.015$). No significant difference in rejection history ($p = 0.196$) or CAV ($p = 0.709$).
Lawson AA et al., 2025 [60]	Retrospective study	113	Mean 6.0 ± 4.0 yrs	T1, T2, ECV, LGE	Cardiac death 2%, re-transplantation 4%, cardiac hospitalization 22%. High T1 (≥1061 ms): HR 4.0 (95% CI 1.7–9.2, p = 0.001). High T2 (≥50.0 ms): HR 2.8 (95% CI 1.1–7.1, $p = 0.026$). High ECV (≥31.4%): HR 3.5 (95% CI 1.5–8.1, $p = 0.004$).
Lawson AA et al., 2026 [54]	Retrospective study	28 (+20 healthy controls)	Median 4.6 (3.9–6.0) yrs	T1, T2, ECV	Higher native T1 (1020 ms (1002−1033) vs 980 ms (942–995), $p < 0.001$), and ECV values (27.7% (25.0–30.2) vs 23.8% (22.2–25.9), $p = 0.003$) than healthy controls. Patient age negatively correlated with T1, T2, ECV values.

Studies are ordered by clinical setting and study type. List of abbreviations in order of appearance: RVEDVi = RV end-diastolic volume index, AR = Acute Rejection, LV = Left Ventricle, EMB = Endomyocardial Biopsy, PPV = Positive Predictive Value, NPV = Negative Predictive Value, LGE = Late Gadolinium Enhancement, MACE = Major Adverse Cardiovascular Events, CAV = Cardiac Allograft Vasculopathy, ECV = Extracellular Volume, AMR = Antibody-mediated Rejection, ACR = Acute Cellular Rejection, GLS = Global Longitudinal Strain, GCS = Global Circumferential Strain, GRS = Global Radial Strain, MRCA = Magnetic Resonance Coronary Angiography, MPR = Myocardial Perfusion Reserve, MPRI = Myocardial Perfusion Reserve Index, XRCA = X-Ray Coronary Angiography, CMR-FT = Cardiac Magnetic Resonance Feature-Tracking, LVGRS = Left Ventricle GRS, RVGLS = Right Ventricle GLS, RVEF = Right Ventricle Ejection Fraction, GD = Graft Dysfunction, PCWP = Pulmonary Capillary Wedge Pressure, dd-cfDNA = donor-derived cell-free DNA, CVF = Collagen Volume Fraction, LVESVI = Left Ventricle End-Systolic Volume Index.

and venous vessels of the transplanted heart, from the microvasculature to the epicardial vessels, and is characterized by diffuse myo-intimal hyperplasia with perivascular infiltrate. Angiographically, it is evidenced by a progressive reduction in caliber from the main branches to the more distal ones or by diffuse parietal irregularities with absent distal peripheral branches [32]. Although these features may limit revascularization, CAV detection and treatment may benefit long-term patient survival and are currently recommended by international guidelines, with percutaneous revascularization using drug-eluting stents as the first-line treatment [4].

Despite initial studies suggesting limited sensitivity of CMR perfusion imaging for detecting CAV, modern CMR assessment may detect CAV even in patients with subclinical pathology [33,34]. Studies have estimated that up to 30% of patients with no angiographically significant coronary artery disease have abnormal perfusion, as measured by a low myocardial perfusion reserve (MPR), and demonstrate worse event-free survival [35–37]. An MPR < 2.2 was found to have a 100% NPV for the exclusion of significant CAV in a single observational study,

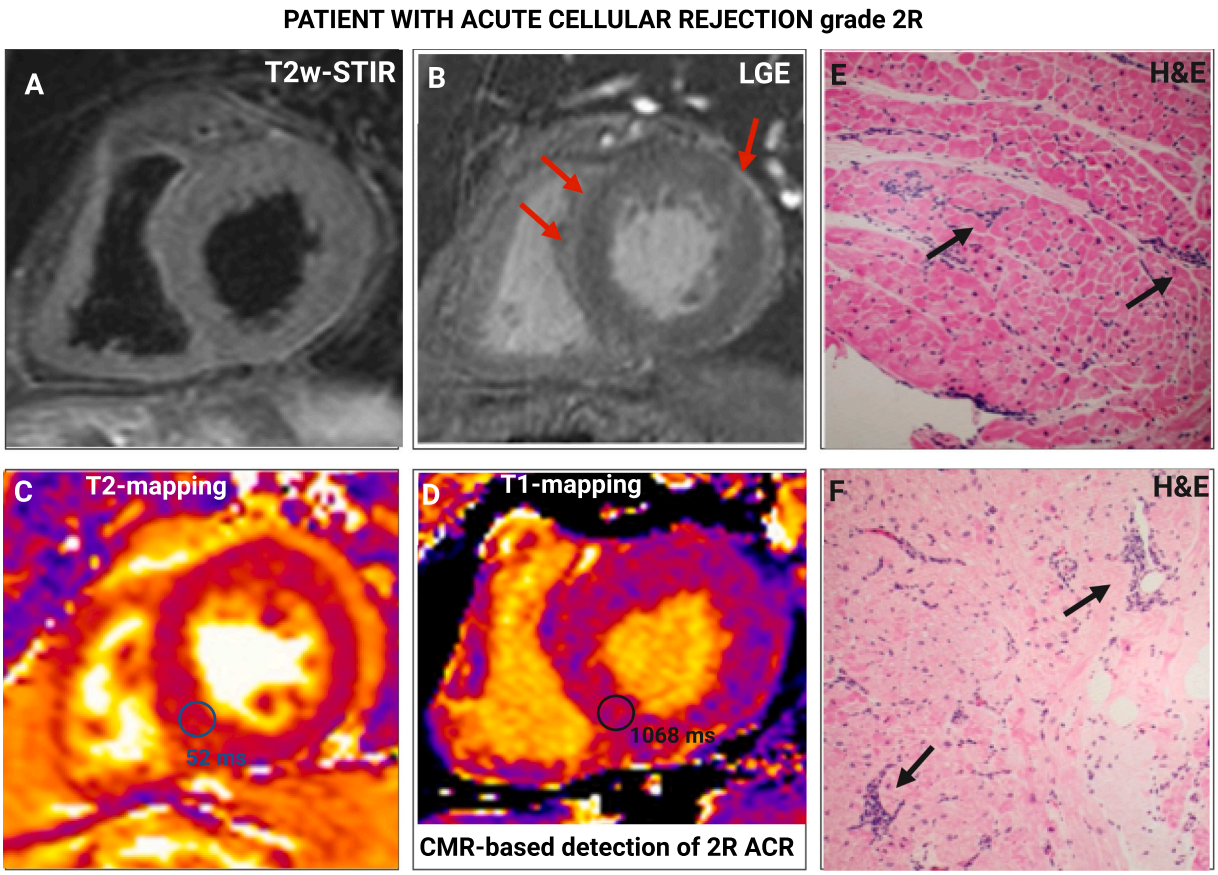


Fig. 2. Patient with acute cellular rejection grade 2R. (A-D) Same short-axis slice analyzed using different sequences. (A) STIR T2-weighted image showing diffuse signal hyperintensity, with a myocardium-to-skeletal muscle signal ratio > 2. (B) Post-contrast image showing diffuse LGE with a non-ischemic pattern and areas of prominent contrast accumulation (red arrows). (C) T2 mapping (True-FISP sequence) demonstrating an increased T2 value of 52 ms (laboratory reference range: 41–49 ms). (D) T1 mapping (MOLLI sequence) showing an elevated T1 value of 1068 ms (laboratory reference range: 923–1043 ms). Notably, both left and right ventricular walls appear thickened. Taken together, these findings indicate diffuse myocardial edema with areas of more pronounced myocardial injury. (*E*-F) The histological images show moderate rejection with two or more foci of myocardial damage, graded as 2R according to the 2004 International Society for Heart and Lung Transplantation classification. H&E indicates hematoxylin and eosin staining; LGE, late gadolinium enhancement; STIR, short Tau inversion recovery.

although broader validation is requested [36]. Moreover, multiparametric assessment with CMR, incorporating myocardial blood flow (MBF $\leq$ 1.95 ml/min per g), coronary flow reserve ($\leq$1.97), native T1 ($\geq$1030 ms), and T2 mapping ($\geq$51 ms) demonstrated good sensitivity (>80%) for significant CAV detection in a selected cohort [38]. In addition, late post-contrast sequences may reveal an ischemic (transmural and/or subendocardial) LGE pattern (Fig. 3) [39,40]. Although a non-infarct pattern may also be present in patients with CAV, the prevalence of the infarct pattern increases with increasing CAV severity, adds prognostic value, and may help to discriminate between underlying graft pathologies [40]. Additional modifications of relevant CMR parameters may occur in the presence of CAV. Higher levels of T2 mapping (54.5 $\pm$ 7.7 ms vs. 50.2 $\pm$ 3.3 ms, $p < 0.05$) and ECV (31.3 $\pm$ 5.3% vs. 27.4 $\pm$ 4.1%, $p < 0.05$) were found in CAV vs. non-CAV recipients [41]. In addition, radial and circumferential peak systolic strain rates, as well as circumferential and longitudinal peak diastolic strain rates, were attenuated in CAV patients, suggesting that subtle myocardial dysfunction may be present when CAV develops [41]. CMR-based ECV also showed a significant association with the extent of interstitial fibrosis on biopsy, suggesting ECV as a potential biomarker for early-stage CAV interstitial fibrosis and aiding in its detection [42]. Nevertheless, differentiating between CAV and AR remains difficult, with no definitive parameter demonstrating sufficient ability to discriminate between the two diagnoses, although in a pediatric population of 59 HTx patients, Kikano et al. suggested that identifiable patterns of CMR abnormalities belong to the two populations, with elevated T1, T2, and ECV predominating in AR [43]. However, additional invasive evaluations are still needed to confirm the diagnosis, and thresholds varied across studies and should not be considered universally applicable.

2.3. Graft dysfunction

CMR parameters in HTx recipients typically differ from those in healthy controls, but progressive normalization over time has been described [9,15]. In general, HTx recipients may present with a variety of findings, including higher T1 and T2 mapping values, elevated ECV, and low-extent LGE. Higher donor age and longer ischemic time correlate with these findings, and the *normal* transplanted heart may vary among patients depending on the organ's background [9]. Nevertheless, chronic GD may happen, even in the absence of significant CAV, ACR, or AMR [31,44]. While echocardiography remains the most common method for prospectively evaluating graft function, CMR can provide additional information on ventricular remodeling and dysfunction. For example, modest changes in global longitudinal strain (GLS) have been associated with a higher risk of death or major adverse events, highlighting the sensitivity of CMR in detecting early dysfunction [45].

CMR is also particularly valuable when evaluating EMB-negative GD, as it may identify both interstitial fibrosis (through elevated T1 mapping and ECV values) and replacement fibrosis (based on LGE presence) (Fig. 4), and monitor its evolution over time. In a single-center study by Anand et al. [44] among 34 HTx recipients with biopsy-negative GD, LGE was present in approximately 50%, and 47% of patients had DSA,

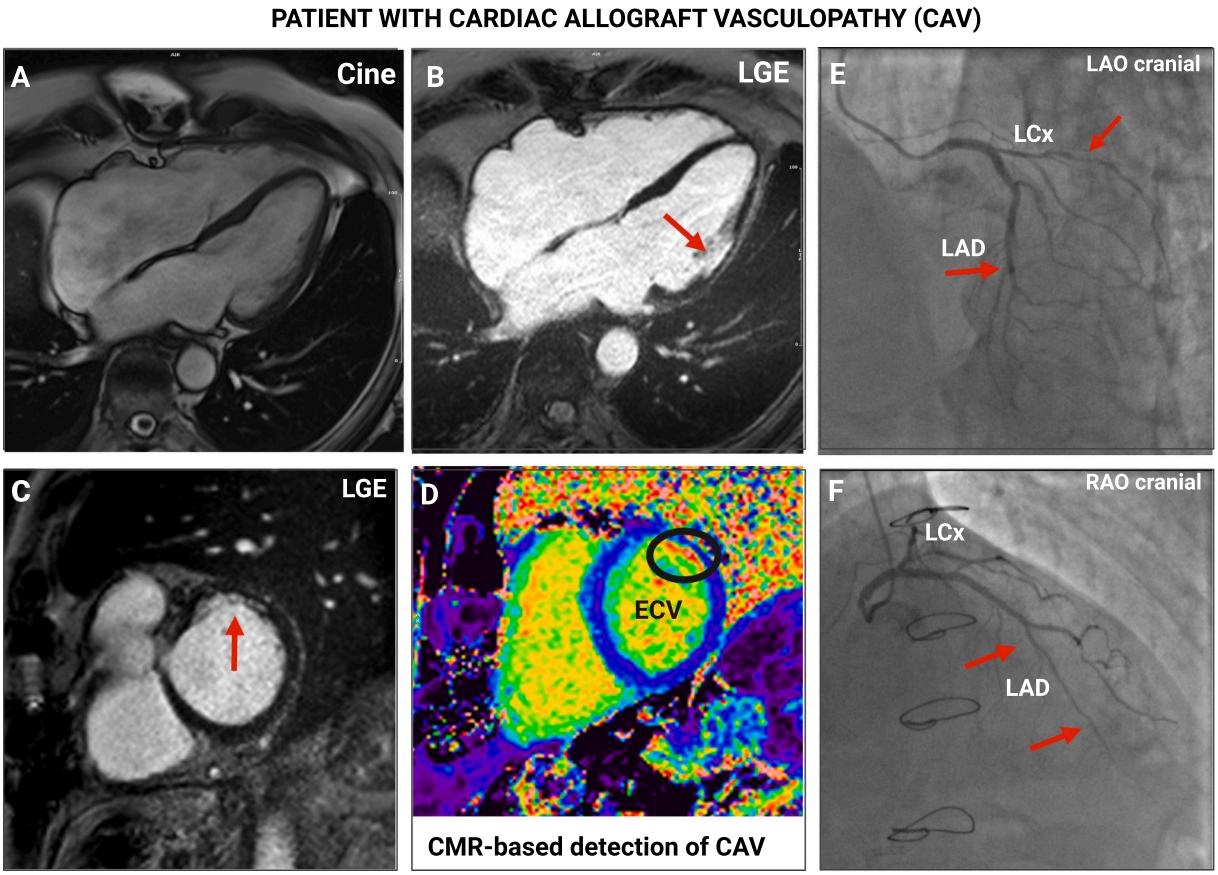


Fig. 3. Patient with cardiac allograft vasculopathy (CAV). (A) Four-chamber cine view showing a mild reduction in wall thickness of the mid-anterolateral wall associated with a regional wall motion abnormality in this region. (B-C) Subendocardial LGE, consistent with an ischemic pattern, is present in the same area on post-contrast images (B, four-chamber view and C, short-axis view). (D) ECV map in the short-axis view demonstrating significantly elevated ECV in the sub-endocardium of the mid-anterolateral wall (black circle), consistent with the area of ischemic LGE. (*E*-F) Coronary angiograms showing diffuse CAV involving the left circumflex coronary (LCx) artery and left anterior descending (LAD) coronary artery in 2 views. The LCx coronary lesion can explain the detected subendocardial LGE. ECV indicates extracellular volume; LAO, left anterior oblique view; LGE, late gadolinium enhancement; RAO, right anterior oblique view.

with signs of edema in 18%. LGE was either patchy or diffuse subepicardial, and most patients were treated empirically with a combination of steroids, plasmapheresis, and rituximab. Interestingly, irrespective of LGE, LVEF improved in most patients [44]. Paired with evidence of higher mortality in EMB-negative patients with elevated dd-cfDNA [31], CMR may help identify signs of dysfunction and/or rejection when EMB fails, especially when supported by the integration of DSA and dd-cfDNA. Interestingly, CMR imaging, paired with changes in non-invasive blood assessments, might also guide immunosuppressive treatment de-escalation in the presence of imaging response. In addition, as the reference imaging method for right ventricular (RV) assessment, CMR may also be useful in HTx patients. By analyzing RV ejection fraction and RV GLS >1 year after HTx, Barrett et al. demonstrated a significant association between the latter and mortality and major cardiovascular events, in the absence of significant interobserver variability [46].

2.4. *Pericardial disorders and other findings*

Pericardial effusion following HTx affects up to one-third of patients [47–49]. Risk factors include the use of sirolimus, prolonged graft ischemia time, and weight mismatch between donor and recipient [48–50]. Moreover, RV perforation during EMB should be considered among the causes of hemorrhagic pericardial effusion. Most effusions are reported early after surgery and are benign, resolving in the first months after HTx [48]. However, complications may occur, such as tamponade and pericardial constriction (Fig. 5) [51,52]. CMR allows accurate quantification of pericardial effusion, inflammation, and assessment of pathological ventricular interdependence.

CMR can also detect mediastinal masses or cardiac involvement from post-transplant lymphoproliferative disease (PTLD) [53], although its role remains adjunctive and nonspecific. PTLD encompasses a spectrum of lymphoproliferative disorders associated with immunosuppressive therapy, Epstein-Barr virus (EBV) infection, and age. Clinical presentation is often non-specific, making early diagnosis difficult. Multimodality imaging and histological confirmation are typically required for diagnosis.

2.5. *Pediatric recipients of HTx and CMR-based surveillance*

Despite similar complications as adults, unique challenges are present when monitoring pediatric HTx recipients, including limited symptom reporting, higher biopsy-related risks due to smaller anatomy, need for sedation during procedures. CMR use may help to minimize some of the risks associated with periodic surveillance, including the absence of radiation exposure. Data from a small retrospective study demonstrated higher native T1 and ECV in pediatric HTx recipients with no prior history of rejection than in healthy children, posing difficult challenges in CMR interpretation [54]. Moreover, as in adults, elevated native T1, T2, and ECV, and their combinations, consistently reflect myocardial edema and inflammation compatible with AR, with T1 showing sensitivity to treatment response and T2 levels correlating with biopsy grades [55–57]. Increased T1 has also been associated with ACR and a markedly higher risk of adverse outcomes during follow-up, alone

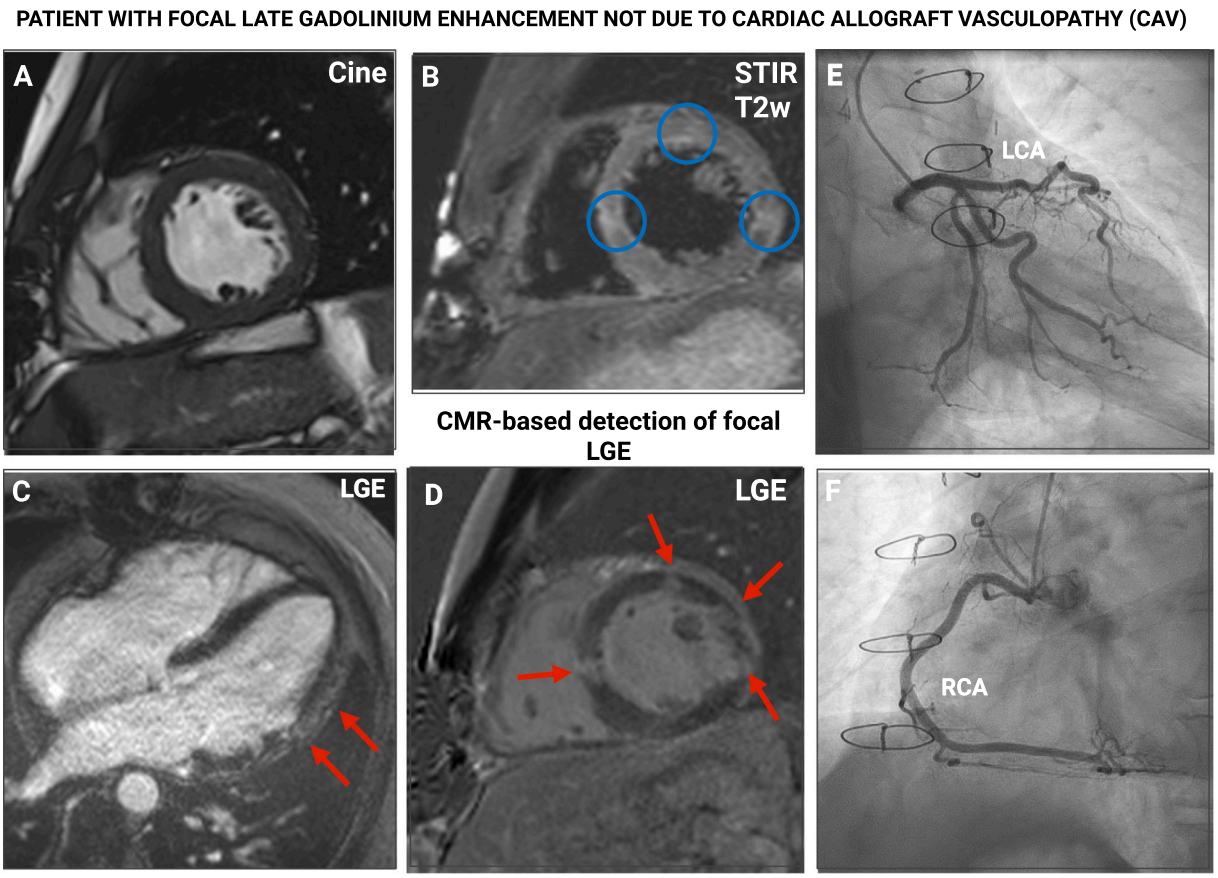


Fig. 4. Patient with focal late gadolinium enhancement (LGE) (A) Mid-ventricular cine short-axis view, with normal wall motion. (B) STIR T2-weighted image showing multiple focal areas of hyperintense signal indicative of myocardial edema (blue circles), with corresponding LGE that is focally transmural, as indicated by arrows. in C and D. Taken together, these findings suggest a possible embolic origin; however, as some lesions appear more subepicardial, a dual unclear etiology (or double component) may be present. (*E*-F) Coronary angiograms showing no coronary lesions involving the left coronary artery (LCA) and right coronary artery (RCA). Histology was negative for rejection (images not shown). STIR indicates short Tau inversion recovery.

or in combination with T2 and ECV [58–60]. ECV has also been shown to correlate with the extent of fibrosis in an observational study >10 years after HTx [61]. Moreover, perfusion CMR may also be used in pediatric recipients, with or without anesthesia, and shows signs of microvascular dysfunction with respect to healthy controls, but definitive evidence on the role of CAV assessment is still lacking [62,63]. The main limitations of CMR imaging in pediatric recipients stem from the need for anesthesia, especially in younger patients, and from the presence of metallic remnants and implantable devices.

2.6. Limitations of CMR in HTx

Several limitations affect the use of CMR in HTx. Technical challenges include difficulties with electrocardiographic gating due to potential sinus tachycardia in the denervated heart, as well as artifacts from surgical materials. Moreover, given the high prevalence of chronic kidney disease associated with the use of calcineurin inhibitors (cyclosporine and/or tacrolimus) among post-HTx patients [64], the administration of gadolinium-based contrast agents must be carefully weighed. CMR is also contraindicated in patients with metallic remnants from previous surgeries or non-CMR-conditional implantable devices, and image quality is limited in patients with CMR-conditional devices. In addition, generalizability of reference values and cut-offs obtained from different studies is influenced by many different factors, including vendor, magnetic field strength, pulse sequence, contrast administration protocol, and post-processing methodology. Finally, logistical constraints (including cost, examination time, limited availability, and the requirement for specialized expertise) may hinder widespread adoption, particularly in acute settings, and may limit implementation outside specialized transplant centers [65]. An additional limitation concerns that CMR findings have been widely validated using EMB. Concordance between CMR and EMB should not necessarily be interpreted as definitive evidence of biological or clinical validity, while discordance between the two modalities should not automatically be interpreted as failure of CMR.

2.7. Proposal for a post-HTx surveillance plan

Considering these limitations, while multicenter studies continue to refine the role of CMR in HTx surveillance, current literature and clinical practice offer valuable insights. The post-HTx scenario is unique for imaging specialists, as certain findings (e.g., LGE, functional or structural abnormalities) may have been pre-existing in the donor heart. Although donor screening is thorough, it can be hampered by the challenging clinical environment (e.g., intensive care, mechanical ventilation, forced supine position), potentially leading to subtle alterations being overlooked. Furthermore, young donors may carry subclinical pathologies (e.g., cardiomyopathy) that are not yet phenotypically expressed. The donor heart may also suffer injury during the clinical phase, leading to death; moreover, ischemia time and organ preservation represent additional critical phases where myocardial damage can occur.

Taking these factors into account, alongside the physiologic response of the graft in the early post-HTx phase [9–11], a proposed surveillance framework could involve a baseline CMR at approximately 1 year post-transplant, performed in conjunction with EMB, coronary angiography,

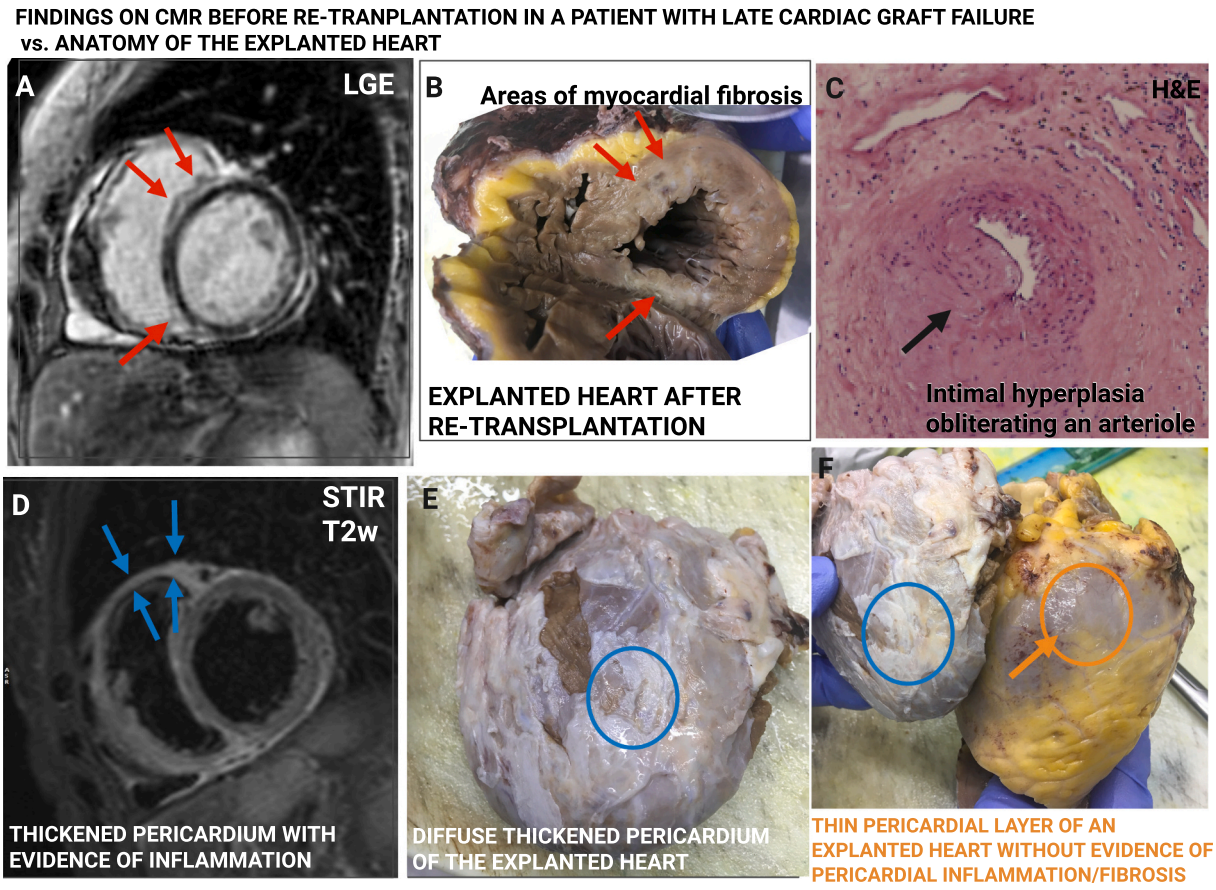


Fig. 5. Comparison between cardiac magnetic resonance (CMR) findings and pathological findings in a patient who underwent retransplantation due to severe graft dysfunction. (A) Post-contrast short-axis image showing extensive myocardial late gadolinium enhancement (LGE) with a non-ischemic pattern (red arrows). (B) The pathological findings from the explanted heart after retransplantation showed areas of myocardial fibrosis (red arrows) detected by CMR. (C) Histological findings showed intimal hyperplasia obliterating arterioles in a patient, where diffuse distal cardiac allograft vasculopathy was documented before retransplantation. In addition, a constrictive physiology was observed and documented by CMR. (D) Short-axis STIR T2-weighted image demonstrating a hyperintense signal indicative of myocardial edema, particularly involving the right ventricular side of the interventricular septum. Additionally, intense pericardial edema and thickening are visible (blue arrows). (E) Pathological evidence of diffuse pericardial thickening was observed (blue circle), consistent with CMR findings. (F) Comparisons between the explanted heart with evidence of diffuse thickening of the pericardium (blue circle) and an explanted heart without thickening of the pericardium (orange circle). STIR indicates short Tau inversion recovery.

and DSA screening. This approach would provide a comprehensive multidimensional *snapshot* of the graft after the acute phase, serving as a baseline for future longitudinal comparisons. The CMR protocol should ideally incorporate stress perfusion, preferably with quantitative assessment of myocardial blood flow [34–37]. As part of the proposed long-term surveillance program for patients with an uneventful post-HTx clinical course, follow-up CMR scans, including stress perfusion, could be considered at 5 and 10 years, aligning with the timing of routine surveillance coronary angiography.

Conversely, patients who develop complications (such as GD, CAV, suspected ACR/AMR, or the detection of elevated dd-cfDNA, as well as those presenting with symptoms of unclear origin) could significantly benefit from the comprehensive multiparametric assessment provided by CMR, in addition to standard reference tests. Similarly, abnormal CMR findings should prompt further clinical evaluation and be interpreted in conjunction with clinical presentation, EMB findings when available, DSA, dd-cfDNA, and other laboratory parameters. Depending on the overall clinical context, subsequent management may include closer surveillance, repeat CMR or EMB, adjustment of immunosuppressive therapy, or further invasive evaluation. Furthermore, CMR may serve as a valuable tool for monitoring the therapeutic efficacy of specific clinical interventions (Fig. 1).

3. Conclusions

CMR represents a powerful non-invasive tool for the comprehensive assessment of HTx recipients. Its ability to combine functional evaluation with detailed tissue characterization makes it particularly attractive for detecting AR, CAV, and GD. In a field in which currently available surveillance strategies have important limitations, CMR may provide complementary information that is not readily captured by conventional approaches. While current evidence supports its growing role in clinical practice, further standardization and large-scale studies are required to define its optimal integration into surveillance strategies.

CRediT authorship contribution statement

Paola Sormani: Writing – original draft, Conceptualization. **Enrico Ammirati:** Writing – original draft, Conceptualization. **Alessandro Villaschi:** Writing – original draft. **Francesca Daus:** Writing – original draft. **Andrea Garascia:** Writing – review & editing. **Gabriella Masciocco:** Writing – review & editing. **Luciano Potena:** Writing – original draft. **Cristina Giannattasio:** Writing – review & editing. **Angelo Vanzulli:** Writing – review & editing. **Patrizia Pedrotti:** Writing – original draft, Conceptualization.

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Declaration of competing interest

E. Ammirati is a consultant for Lexeo and ICON and previously served as a consultant for Hotgen Health Inc.

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