Role of cardiac magnetic resonance imaging in heart failure

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Heart failure (HF) is approaching global pandemic proportions, with at least 26 million people currently affected worldwide. Irrespective of HF phenotype, stratified according to left ventricular ejection fraction (LVEF), i.e. "reduced" — HFrEF, "mildly reduced" — HFmrEF, and "preserved" — HFpEF, and despite an expanding array of effective treatments including renin-angiotensin-aldosterone-system inhibitors, angiotensin-receptor-neprilysin-inhibitors, β-blockers, sodium-glucose-co-transporter-2 inhibitors and cardiac resynchronization (CRT) therapy, morbidity, and mortality remains high [1]. Worryingly, with an aging population, coupled with rising HF incidence and an increasing prevalence of comorbidities, it is projected that HF hospitalizations will rise by 50% over the next 25 years, significantly impacting global healthcare systems [1].

Prompt diagnosis of HF and assessing underlying etiology is therefore imperative. Whilst echocardiography is the traditional workhorse in the HF diagnostic pathway, cardiac magnetic resonance imaging (CMR) is a class 1C recommendation [1] in those with poor acoustic windows and in whom myocardial tissue characterization is paramount e.g. myocardial infarction (MI), infiltration (e.g. amyloidosis). Compared to echocardiography, CMR offers unlimited imaging planes, superior spatial resolution, and unrivaled tissue characterization properties, enabling further sub-categorization of HF into differing clinical etiologies with a high degree of accuracy [2].

In the recent issue of *Kardiologia Polska* (*Kardiol Pol*, *Polish Heart Journal*), Ojrzyńska-Witek et al. [3] sought to evaluate the role of CMR in identifying the underlying etiology of HF patients in whom preceding diagnostic workup (including clinical evaluation, echocardiography, and coronary angiography in those with cardiac risk factors for coronary artery disease [CAD]) had failed to elicit this. The authors reported the findings from a single-center, retrospective analysis of 243 CMR scans performed over 10 years. In summary, a new clinical diagnosis following CMR was observed in 38.7%. Such new CMR findings included dilated cardiomyopathy (DCM, 58.8%), myocarditis (7%), restrictive cardiomyopathy in 5.3% of which amyloidosis accounted for just over half, left ventricular (LV) non-compaction (2.9%), hypertrophic cardiomyopathy (1.2%), and Takotsubo cardiomyopathy (0.8%). Overall, CMR resulted in subsequently modified patient management in 16.9% of patients. While this study strongly advocates the value of CMR in HF and the authors should be applauded for their elegant work and study conduct, the findings also raise a number of key points for further consideration:

- The single-center study setting is not truly representative of the general HF population and is prone to referral bias. The baseline characteristics reveal a marked male preponderance (73%), and the mean age was only 44 years, which is in stark contrast to published epidemiological

data from HF populations whereby females typically account for more than 50% and the average age is a few decades older [1].

- The main HF phenotype studied was HFrEF (in 81%), again contrasting with the published literature [1], whereby HFmrEF/HFpEF account for nearly half of all HF cases. Furthermore, given the predominance of HFrEF and since subjects with CAD on angiography were excluded, a new finding of non-ischemic DCM is highly unsurprising.
- The CMR yield of new diagnoses (38.7%) is higher in comparison to another albeit, smaller (n = 150), prospective study [4] of HF patients comprising only HFrEF or HFmrEF, in which 30% had newly identified clinical pathologies. In both studies, the most common finding again was of non-ischemic DCM (58.8% vs. 24%). In our own single-center experience confined to HFpEF (n = 154; mean age, 72 years), similar new CMR findings were observed in 27% of cases [5]. We speculate that the overall CMR diagnostic yield would likely have been even greater if the proportion of HFpEF studied was higher because this particular HF phenotype is associated with increasing age and a heavy co-morbidity burden, including conditions such as obesity, atrial fibrillation, and lung disease, rendering traditional echocardiographic assessment challenging and increasing the influence of CMR in diagnosis [1].
- It remains unclear from the study description whether the same protocol was used throughout. For example, consistent use of parametric T1/T2/T2* mapping may have further enhanced the sensitivity to detect conditions such as amyloidosis, myocarditis, and iron-loading [2].
- Although the study did not report clinical outcome data related to the new clinical pathologies identified by CMR, a strong signal further supporting CMR usage in HF has recently been shown in a multi-center trial (OUTSMART-HF) [6] of 500 patients (mean age 59 years; HFpEF proportion 7%) who were randomized to either routine i.e. echocardiography plus CMR vs. selective CMR (only at the physician's discretion) in non-ischemic HF. While the study did not reveal any significant differences between the imaging arms in terms of specific HF diagnoses, CMR yet again increased the overall yield of such diagnoses (44%) above baseline echocardiography. Furthermore, patients with specific HF causes identified by CMR suffered more clinical events compared to non-specific HF causes (19% vs. 12% at 12 months; $P = 0.02$), demonstrating the potential prognostic value of CMR-guided HF assessment.
- Evidence on the cost-effectiveness of CMR in HF is still lacking.

Beyond the refinement of underlying clinical diagnosis, CMR may play a useful role as we enter an era of precision-based, personalized medicine. CMR has high diagnostic accuracy for detecting significant CAD following stress perfusion and is also the imaging gold standard for detection of both left atrial (LA) and right ventricular (RV) volumes and function, as well as focal (late gadolinium enhancement [LGE]) and diffuse fibrosis (T1 mapping/extracellular volume [ECV]). CMR allows further refinement of HF into key underlying pathophysiological substrates, enabling targeted therapies and further risk stratification. The first step in such prognostication is the detection of CAD with a high degree of accuracy by stress perfusion CMR and LGE [7, 8]. Ischemic HF confers a worse prognosis compared to non-ischemic HF (5-year survival 45% vs. 62%) [9]. Furthermore, MI is readily detectable by CMR with accurate information about localization and transmural extent, as well as predicting response to revascularization, LV recovery of dysfunctional myocardium, and ultimately survival [8]. Across the spectrum of HF, irrespective of LVEF, worse outcomes have been observed with CMR-derived RV dysfunction [10, 11], LA volumes and LAEF [12], focal fibrosis detected by LGE (MI [8] and non-MI [2,13]), and diffuse fibrosis (ECV) [14]. The presence of mid-wall focal fibrosis may further impact decision-making regarding device selection (CRT-P vs. CRT-D), guide LV ventricular lead placement in HFrEF, and predict responsiveness to CRT [15].

Article information

Conflict of interest: None declared.

REFERENCES

1. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021; 42(36): 3599–3726, doi: 10.1093/eurheartj/ehab368, indexed in Pubmed: 34447992.
2. Karamitsos TD, Francis JM, Myerson S, et al. The role of cardiovascular magnetic resonance imaging in heart failure. J Am Coll Cardiol. 2009; 54(15): 1407–1424, doi: 10.1016/j.jacc.2009.04.094, indexed in Pubmed: 19796734.
3. Ojrzyńska-Witek N, Marczak M, Mazurkiewicz Ł, et al. Role of cardiac magnetic resonance in heart failure of initially unknown etiology: A 10-year observational study. Kardiol Pol. 2022; 80(3): 278–285, doi: 10.33963/KP.a2021.0186, indexed in Pubmed: 34936084.
4. Abbasi SA, Ertel A, Shah RV, et al. Impact of cardiovascular magnetic resonance on management and clinical decision-making in heart failure patients. J Cardiovasc Magn Reson. 2013; 15: 89, doi: 10.1186/1532-429X-15-89, indexed in Pubmed: 24083836.
5. Kanagala P, Cheng ASH, Singh A, et al. Diagnostic and prognostic utility of cardiovascular magnetic resonance imaging in heart failure with preserved ejection fraction — implications for clinical trials. J Cardiovasc Magn Reson. 2018; 20(1): 4, doi: 10.1186/s12968-017-0424-9, indexed in Pubmed: 29321034.
6. Paterson DI, Wells G, Erthal F, et al. OUTSMART HF: A Randomized Controlled Trial of Routine Versus Selective Cardiac Magnetic Resonance for Patients With Nonischemic Heart Failure (IMAGE-HF 1B). Circulation. 2020; 141(10): 818–827, doi: 10.1161/CIRCULATIONAHA.119.043964, indexed in Pubmed: 31910649.
7. Jaarsma C, Leiner T, Bekkers SC, et al. Diagnostic performance of noninvasive myocardial perfusion imaging using single-photon emission com-

puted tomography, cardiac magnetic resonance, and positron emission tomography imaging for the detection of obstructive coronary artery disease: a meta-analysis. J Am Coll Cardiol. 2012; 59(19): 1719–1728, doi: 10.1016/j.jacc.2011.12.040, indexed in Pubmed: 22554604.
8. Khan JN, McCann GP. Cardiovascular magnetic resonance imaging assessment of outcomes in acute myocardial infarction. World J Cardiol. 2017; 9(2): 109–133, doi: 10.4330/wjc.v9.i2.109, indexed in Pubmed: 28289525.
9. Felker GM, Shaw LK, O'Connor CM. A standardized definition of ischemic cardiomyopathy for use in clinical research. J Am Coll Cardiol. 2002; 39(2): 210–218, doi: 10.1016/s0735-1097(01)01738-7, indexed in Pubmed: 11788209.
10. Gulati A, Ismail TF, Jabbour A, et al. The prevalence and prognostic significance of right ventricular systolic dysfunction in nonischemic dilated cardiomyopathy. Circulation. 2013; 128(15): 1623–1633, doi: 10.1161/CIRCULATIONAHA.113.002518, indexed in Pubmed: 23965488.
11. Kanagala P, Arnold JR, Singh A, et al. Prevalence of right ventricular dysfunction and prognostic significance in heart failure with preserved ejection fraction. Int J Cardiovasc Imaging. 2021; 37(1): 255–266, doi: 10.1007/s10554-020-01953-y, indexed in Pubmed: 32737707.
12. Pellicori P, Zhang J, Lukaschuk E, et al. Left atrial function measured by cardiac magnetic resonance imaging in patients with heart failure: clinical associations and prognostic value. Eur Heart J. 2015; 36(12): 733–742, doi: 10.1093/eurheartj/ehu405, indexed in Pubmed: 25336215.
13. Kato S, Saito N, Kirigaya H, et al. Prognostic significance of quantitative assessment of focal myocardial fibrosis in patients with heart failure with preserved ejection fraction. Int J Cardiol. 2015; 191: 314–319, doi: 10.1016/j.ijcard.2015.05.048, indexed in Pubmed: 26005800.
14. Schelbert EB, Piehler KM, Zareba KM, et al. Myocardial fibrosis quantified by extracellular volume is associated with subsequent hospitalization for heart failure, death, or both across the spectrum of ejection fraction and heart failure stage. J Am Heart Assoc. 2015; 4(12), doi: 10.1161/JAHA.115.002613, indexed in Pubmed: 26683218.
15. Leyva F, Leyva F, Leyva F, et al. Cardiac resynchronization therapy guided by cardiovascular magnetic resonance. J Cardiovasc Magn Reson. 2010; 12(3): 64–262, doi: 10.1186/1532-429X-12-64, indexed in Pubmed: 21062491.