

Cardiac magnetic resonance for the risk stratification of heart transplant recipients: ready for prime time?

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With great interest we read the study of Butler *et al.* (1) entitled 'Cardiovascular MRI predicts 5-year adverse clinical outcome in heart transplant recipients', which was recently published in the *American Journal of Transplantation*. Over a long follow-up duration (4.9 yrs) the authors demonstrated that the evaluation of cardiac magnetic resonance imaging (CMR) parameters is useful for the prediction of outcomes in heart transplant (HT) recipients. In this regard, myocardial scar assessed by late gadolinium enhancement (LGE) and right ventricular end-diastolic volume index (RVEDVI) were independently associated with cardiac death and hospitalization due to cardiac symptoms. Based on these results, the authors suggest that prognostic models should not only include clinical and demographic variables as recommended by the international society of heart and lung transplantation (ISHLT) (2), but also consider CMR imaging parameters. In this regard, the versatility of CMR allows for the noninvasive and reproducible assessment of cardiac anatomy, deformation and function, perfusion, viability and if required metabolism and tissue characterization without radiation exposure for the patients (3).

Despite the comprehensive methodology the present study has some limitations. Thus, the study cohort (n=56 patients who had LGE, n=4 excluded due to renal dysfunction) and the number of hard cardiac events were relatively small (n=7). In this regard, 2 patients who died during the study period had severely impaired renal function, which precluded gadolinium administration. LGE was present in 32 of 56 HT recipients (57%), the majority (88%) exhibiting non-ischemic LGE patterns. In contrast to previous findings (4), both ischemic and non-ischemic LGE patterns were related to outcomes, which may indicate

that further mechanisms apart from silent infarction may be involved in such patients, including myocardial hypertrophy and fibrosis due to pressure overload and prior rejections or systemic inflammatory response, respectively. In addition, although LGE was primarily located in the left ventricle, RVEDVI rather than left ventricular (LV)-parameters was independently related to cardiac outcomes. This finding merits further investigation, because RVEDVI may depend on non-CAV related factors like prior to transplant pulmonary hypertension or tricuspid regurgitation after HT. Furthermore, quantitative parameters of regional myocardial deformation and perfusion reserve during pharmacologic hyperemia were not evaluated.

Based on our current work to be published in the *American Journal of Transplantation* (5), we also identified CMR based parameter, which serve as predictors of cardiac outcomes in HT recipients. In our cohort (n=108 patients with n=18 cardiac events over a follow-up period of 4.2 yrs), LV-ejection, septal wall thickness, CAV by ISHLT criteria and LGE were not independently associated with cardiac outcomes, when myocardial perfusion reserve was added in our multivariable model. We consider the latter as a surrogate marker of both epicardial and microvascular components of CAV, which may therefore surpass the value of surveillance coronary angiography for early CAV detection. Ischemic LGE and increased cardiac volume indexes on the other hand may represent rather markers of advanced CAV and subclinical transplant heart failure, respectively when pharmacologic interventions may be less effective.

We would like to congratulate Butler *et al.* for their work, which represents an important cornerstone towards

the CMR risk assessment for future cardiac events in HT recipients. Future multi-center studies are now warranted in order to shed more light on the value of different CMR parameters for the risk stratification of HT recipients.

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