

Poster presentation

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## Diagnosis of acute cardiac allograft rejection using cardiovascular magnetic resonance - preliminary findings

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### Study objective

To assess the ability of cardiovascular magnetic resonance (CMR) to detect acute cardiac allograft rejection.

### Background

Acute cardiac allograft transplant rejection (ACAR) remains a common and significant complication of cardiac transplantation (HTX). Endomyocardial biopsy (EMB) is the current gold standard for its diagnosis and monitoring. However EMB is invasive, prone to sampling error and relatively insensitive for detecting antibody-mediated rejection. CMR is an attractive potential non-invasive alternative.

### Methods

Preliminary data was collected on transplant recipients scheduled for EMB. Same-week pre-biopsy CMR imaging was performed on a 1.5 Tesla scanner (Avanto, Siemens, Erlangen, Germany) using a 32 channel coil system. Myocardial signal intensity was measured by drawing regions of interest (ROI) within the myocardium and was normalised to skeletal muscle signal intensity with a ROI placed within skeletal muscle on the same image. CMR image analysis was correlated with histological findings obtained with EMB.

### Results

Nine patients were scanned, with a mean time from transplantation of 7+4 months. One patient was experiencing severe rejection (EMB grade 3). Eight patients had no evidence of acute rejection on biopsy (grade 0). On T2-weighted (T2w) turbospin echo and on T2-weighted

short-tau inversion recovery imaging (STIR), the patient with grade 3 rejection had a higher mean myocardial:skeletal muscle signal intensity (SI) ratio than patients with grade 0 (Table 1). The patient with grade 3 rejection had an SI ratio of 1.95 on T2w imaging which is above the diagnostic SI ratio used in myocarditis (1.9) [1]. Two patients with grade 0 rejection had heterogeneous myocardial signal on T2w and STIR imaging, one with high signal intensity in ventricular septum (Figure 1A) and one with high signal intensity in the lateral left ventricular wall (1B). No patients had evidence of late gadolinium enhancement.

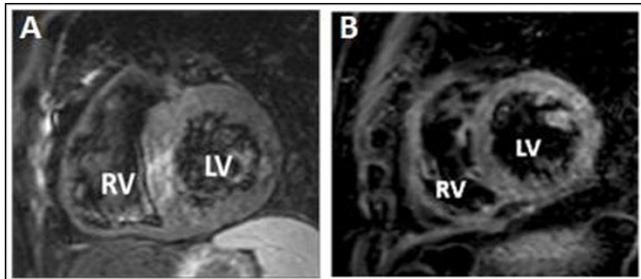
### Conclusion

In these preliminary findings, severe ACAR was associated with a higher normalised myocardial signal intensity on T2w and STIR imaging, possibly reflecting myocardial oedema. Heterogeneous high signal was observed in patients with no evidence of rejection on EMB, the significance of which is not clear, but may reflect patchy myo-

**Table 1: Signal intensity ratios in different rejection grades**

	Rejection grade	
	0 n = 8 mean + SD	3 n = 1
<b>T2w TSE</b>		
Mean myocardial:muscle SI ratio	1.60+0.24	1.95
<b>STIR</b>		
Mean myocardial:muscle SI ratio	1.28+0.10	1.82

cardial oedema, with EMB sampling error. With optimised oedema sequences it may be possible to define thresholds for the presence and grade of ACAR.



**Figure 1**

## References

1. Friedrich, et al.: **Cardiovascular magnetic resonance in myocarditis: A JACC White Paper.** *JACC* 2009, **53**:1475-87.

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