Coulden et al. Journal of Cardiovascular Magnetic Resonance 2014, 16(Suppl 1):P299
http://www.jcmr-online.com/content/16/S1/P299

Journal of Cardiovascular Magnetic Resonance

POSTER PRESENTATION Open Access

Cardiac MRI and FDG-PET in the diagnosis of cardiac sarcoidosis

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From 17th Annual SCMR Scientific Sessions
New Orleans, LA, USA. 16-19 January 2014

Background

Sarcoidosis is a multisystem disorder with cardiac involvement in 25% of cases [1]. Diagnosis of cardiac sarcoidosis is challenging with FDG-PET and cardiac MRI (CMRI) proving most reliable. We compare FDG-PET and CMRI with delayed enhancement (LGE) in patients with biopsy proven extra-cardiac sarcoidosis being investigated for cardiac involvement.

Methods

30 patients meeting Japanese Ministry of Health & Welfare guidelines [2] for clinical cardiac sarcoidosis were investigated with FDG-PET CT (Gemini TF Philips) and CMRI (Aera 1.5T Siemens) on the same day. Patients undergoing FDG-PET followed a 24 hour low-carbohydrate diet and overnight fast [3]. CMRI examination included SSFP assessment of left ventricular (LV) function, short axis T2-weighted STIR and PSIR-LGE 10 minutes post 0.2 mmol/kg GdDTPA. Images were reviewed by experienced readers blinded to the results of the other examination. FDG-PET was considered positive if any segment (AHA 17 segment model) had an SUVmax > 3.6 (3). CMRI was considered positive if any segment showed 'sarcoid-type' LGE. In no case was edema present on STIR imaging without LGE in the same segment on subsequent PSIR.

Results

FDG-PET and CMRI were positive in 10 patients; FDG-PET +ve and CMRI -ve in 3; FDG-PET -ve and CMRI +ve in 4; both -ve in 13. Distributions of FDG and LGE throughout the myocardium are summarized in Figure 1. In 2 cases where FDG and CMRI were +ve, LGE was in an ischemic pattern and both patients had known

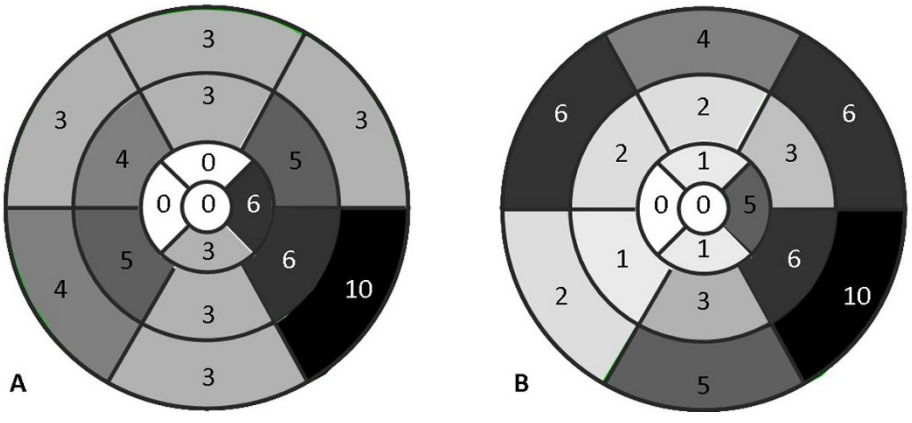

Figure 1 LV distribution of LGE (A) and significant myocardial FDG uptake (B) for all 30 patients. By both modalities, cardiac sarcoidosis predominantly involves the lateral wall with relative sparing of the distal anterior wall, distal septum and apex.

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recent iscemic events (sub-endocardial). 6 of the remaining 8 had impaired LV function (EF < 50%). All FDG +ve/CMRI -ve cases had intense mediastinal/lung FDG uptake. All 4 FDG -ve/CMRI +ve had either long-standing sarcoidosis (> 2 years) or absent node/lung FDG activity. Seven of the cases, -ve by both modalities, had no significant or minimal node/lung FDG uptake (SUVmax < 2.5).

Conclusions

Previous single modality studies have suggested sensitivity and specificity for FDG-PET of 89% and 78% and CMRI of 75% and 77% respectively. In our study, 43% showed no cardiac sarcoidosis by either modality and half of these had minimal or no FDG-PET evidence of active sarcoidosis elsewhere. Are these false -ve FDG-PET/CMRI studies or are the clinical criteria too sensitive? Our findings suggest FDG-PET and CMRI-LGE show different degrees of cardiac sarcoid involvement: FDG-PET indicating active inflammatory disease, LGE showing severe edema and scar. Those patients with LGE and no myocardial FDG uptake appear to have 'burnt-out' disease.

Funding

None.

Published: 16 January 2014

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doi:10.1186/1532-429X-16-S1-P299
Cite this article as: Coulden *et al.*: Cardiac MRI and FDG-PET in the diagnosis of cardiac sarcoidosis. *Journal of Cardiovascular Magnetic Resonance* 2014 **16**(Suppl 1):P299.