Sequential FDG-PET in the management of multiorgan sarcoidosis

Alan Kelly,[1] Michael P Keane,[1,2] David Keane,[3] Jonathan D Dodd,[4] David J Murphy,[4] Cormac McCarthy[1,2]

[1]Respiratory Medicine, St. Vincent's University Hospital, Dublin, Ireland
[2]School of Medicine and Medical Science, University College Dublin, Dublin, Ireland
[3]Cardiology, St. Vincent's Uiversity Hospital, Dublin, Ireland
[4]Radiology, St. Vincent's University Hospital, Dublin, Ireland

Correspondence to
Dr Cormac McCarthy, Medicine, University College Dublin, Dublin 4, Ireland; cormac.mccarthy@ucd.ie

DJM and CM are joint senior authors.

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A 32-year-old man presented with palpitations and central chest pain, examination was unremarkable and chest radiograph was normal. ECG demonstrated right axis deviation and inferolateral T-wave inversion; cardiac enzymes were normal. Cardiac MRI demonstrated multifocal, patchy subepicardial and mesocardial left ventricular late gadolinium enhancement, suggestive of cardiac sarcoidosis. Whole body and dedicated cardiac fluorodeoxyglucose (FDG)-positron emission tomography (PET) was performed 60 min after radiotracer injection following a special patient preparation to suppress physiological myocardial uptake of glucose. This consists of a high-fat, low-carbohydrate diet the day before scanning followed by a 15 hours fast. The PET revealed multifocal metabolically active cardiac inflammation, corresponding to the areas of late gadolinium enhancement on MRI and extensive sites of nodal, pulmonary, hepatic and splenic metabolic activity (figures 1A and 2A). Endobronchial ultrasound-guided biopsy of station 7 and 11R lymph nodes did not identify any granuloma. A myocardial biopsy demonstrated only very focal lymphocytic inflammation and no granuloma. Despite a lack of histological confirmation, a diagnosis of cardiac sarcoidosis was determined by multidisciplinary discussion and the patient was commenced on high-dose corticosteroids. A comprehensive electrophysiological study was positive for inducible sustained ventricular tachycardia, and an implantable cardioverter-defibrillator was inserted.

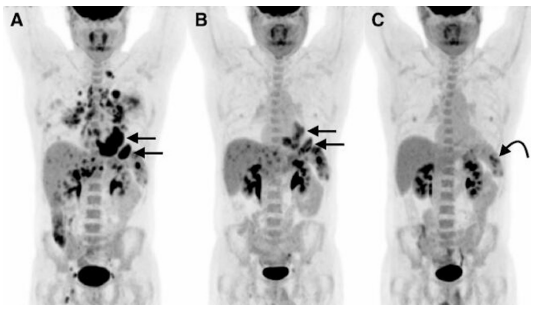

Figure 1 MIP FDG-PETs images. At diagnosis (A), there is extensive metabolically active cardiac disease (arrows), as well as pulmonary, nodal, hepatic and splenic disease. Follow-up FDG-PET post 3 months of treatment (B) demonstrates significant reduction in extent and avidity of cardiac uptake (arrows), along with near resolution of the extra-cardiac disease. Subsequent FDG-PET (C) shows no residual cardiac activity, with minor splenic uptake the only residual focus (curved arrow). FDG, fluorodeoxyglucose; MIP, maximum intensity projection; PET, positron emission tomography.

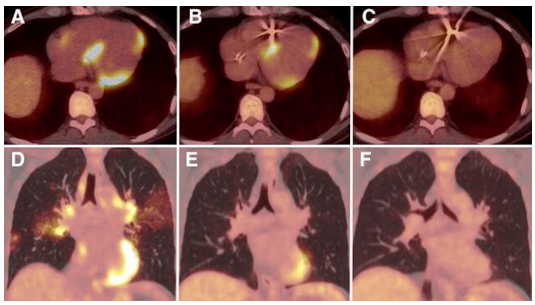

Figure 2 Axial and coronal fused FDG-PET/CT images. Initially at diagnosis there are multiple focal areas of intense left ventricular myocardial uptake (A, myocardial SUV_{max} 8.9), along with active mediastinal lymphadenopathy and peribronchovascular pulmonary nodularity (D). Interim follow-up FDG-PET shows an implantable cardiac defibrillator in situ, reduction in the avidity of focal cardiac uptake (B, myocardial SUV_{max} 6.3) and resolution of the nodal and pulmonary disease (E). The cardiac uptake has completely resolved on the final follow-up FDG-PET (C, myocardial SUV_{max} 1.8), with no residual nodal or pulmonary disease (F). FDG, fluorodeoxyglucose; PET, positron emission tomography; SUV, standardised uptake value.

FDG-PET can be used to guide both diagnosis and management of sarcoidosis,[1 2] and may be useful for assessing treatment response, disease extent, occult disease,[3] and may have utility in stratifying prognosis[4] and FDG-PET and cardiac MRI can be complementary,[5] but there is significant radiation exposure associated with repeated PET imaging. Follow-up FDG-PET after 6 months of therapy showed interval reduction in cardiac metabolic activity and improvement in the pulmonary and nodal disease (figures 1B and 2B). Immunosuppression was continued for a further 3 months and follow-up imaging demonstrated almost complete resolution of disease (figures 1C and 2C). This report highlights the utility of FDG-PET in diagnostically challenging cases, and its applicability to direct therapy based on metabolic activity in sarcoidosis.

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REFERENCES

1 Akaike G, Itani M, Shah H, *et al*. PET/CT in the diagnosis and workup of sarcoidosis: focus on atypical manifestations. *Radiographics* 2018;38:1536–49.
2 Chareonthaitawee P, Beanlands RS, Chen W, *et al*. Joint SNMMI-ASNC Expert Consensus Document on the Role of ^{18}F-FDG PET/CT in Cardiac Sarcoid Detection and Therapy Monitoring. *J Nucl Med* 2017;58:1341–53.
3 Kersey CB, Flaherty KR, Goldenthal IL, *et al*. The use of serial cardiac ^{18}F-fluorodeoxyglucose- positron emission tomography imaging to diagnose, monitor, and tailor treatment of cardiac sarcoidosis patients with arrhythmias: a case series and review. *Eur Heart J Case Rep* 2019;3:1–7.
4 Flores RJ, Flaherty KR, Jin Z, *et al*. The prognostic value of quantitating and localizing F-18 FDG uptake in cardiac sarcoidosis. *J Nucl Cardiol* 2018. doi:10.1007/s12350-018-01504-y. [Epub ahead of print: 12 Nov 2018].
5 Vita T, Okada DR, Veillet-Chowdhury M, *et al*. Complementary value of cardiac magnetic resonance imaging and positron emission tomography/computed tomography in the assessment of cardiac sarcoidosis. *Circ Cardiovasc Imaging* 2018;11:e007030.