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The evaluation of cardiac sarcoidosis with ^{18}F-FDG PET scan

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Absctract:

Cardiac involvement in sarcoidosis is associated with poor prognosis. ^{18}F-FDG PET can detect the presence of Cardiac sarcoidosis, assesses disease activity, and serves as a mean to monitor treatment response in patients with cardiac sarcoidosis.

Introduction:

Cardiac involvement in patients who had systemic sarcoidosis occurs in 20-30% as shown in pathology examinations and is associated with poor prognosis (1,2). Although myocardial granulomas can be identified in almost 25%–79% of autopsy examinations, only 25 % of all patients with sarcoidosis have clinical manifestations of cardiac involvement (1). In this report, ^{18}F-FDG PET scan helped in diagnosing a patient with suspected Cardiac sarcoidosis (CS), classifying the disease activity, and monitoring the response to treatment.

Case report:

A 68-year-old male with history of cutaneous sarcoidosis presented to the cardiology clinic with progressive dyspnea. Physical examination revealed +4-pitting edema. ECG showed complete heart block. A Cardiac MRI was suggestive for infiltrative process. With a baseline blood glucose of 93 mg/dL, the patient was injected with 10.67 mCi (3.94 Bq) of F-18-fluorodeoxyglucose (FDG), and the patient was instructed to take a high-fat low-carbohydrate diet the day prior without additional overnight fast. After one hour delay, multiple metabolic tomographic images of the myocardium were obtained. The N-13 ammonia perfusion images (bottom row of image A) demonstrated defects in the mid to basal inferior wall ,basal anterior walls,anteroseptal and inferoseptal areaswith corresponding increased FDG uptake in these regions; consistent with active sarcoid infiltration of the myocardium. The calculated ejection fraction was 24%. The patient completed a 6 month trial of high dose steroids. The follow-up PET myocardial imaging study (image B) demonstrated moderate improvement of the perfusion defects (while the FDG portion demonstrated only blood pool activity without myocardial trapping. The ejection fraction improved to 37%. These findings indicate excellent response to treatment of exisiting inflammation although the clinical improvement depends on the extent of previous damage.

Discussion:

Cardiac sarcoidosis represents the cause of death in 13 % to 25 % of fatal cases of sarcoidosis (1). A definite diagnosis of cardiac sarcoidosis can be made by endomyocardial biopsy with sensitivity less than 20% (2). For a definitive diagnosis and management all patients will likely benefit from an echocardiogram and either cardiac MRI(CMR) or ^{18}F-FDG PET(1). CMR

usually performed as initial test in patients with suspected CS. Gadolinium enhanced CMR with delayed imaging has the benefit of high sensitivity and spatial resolution without radiation exposure. If negative, ^{18}F-FDG PET can be avoided. If there is a suggestion of cardiac involvement ,^{18}F-FDG PET imaging could be performed to establish baseline disease activity, assess the need for initiation of medical therapy and monitor response to treatment over time. In patients with contraindication to performing CMR, ^{18}F-FDG PET could be used as first line imaging (1). Usually, CMR shows scar, and thus chronic disease, whereas FDG PET identifies active involvement. Typical radionuclide protocols for imaging cardiac sarcoidosis include ^{18}F-FDG PET for imaging inflammation combined with SPECT or PET myocardial perfusion imaging (1).

The myocardial perfusion assessment can be performed with Tc-99m, Tl-201, N-13 ammonia, or Rb-82 based radiotracers using standard protocols. Attenuation correction should be used with SPECT myocardial perfusion imaging whenever possible. In the absence of coronary arteries abnormalities, the perfusion defects on PET scanning in a patient with sarcoidosis strongly lead toward the involvement of cardiac sarcoidosis (2). One way to describe the pattern in CS on PET scan is based on a comparison of the degree of perfusion abnormality and ^{18}F-FDG uptake: normal (normal perfusion/normal^{18}F-FDG), early stage (mild perfusion defect/increased ^{18}F-FDG), progressive stage (moderate perfusion defect/increased ^{18}F-FDG), progressive myocardial impairment stage (severe perfusion defect/increased ^{18}F-FDG), and fibrosis stage (severe perfusion defect/minimal or no^{18}F-FDG uptake) (1). In our case we used this method, and the results were described in the progressive myocardial impairment stage (image A). Our patient showed clinical improvement after 6 months of treatment, and the PET scan findings of resolved FDG uptake were consistent with subsided inflammation though clinical improvement (image 2) is ultimately limited by the previous damage.

Conclusion:

^{18}F-FDG PET can detect the presence of CS, assess disease activity, and monitor treatment response in patients with cardiac sarcoidosis.

References:

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Images:

Image A. The perfusion images demonstrated defects in the mid to basal inferior wall ,basal anterior walls,anteroseptal and inferoseptal areaswith corresponding increased FDG uptake in these regions

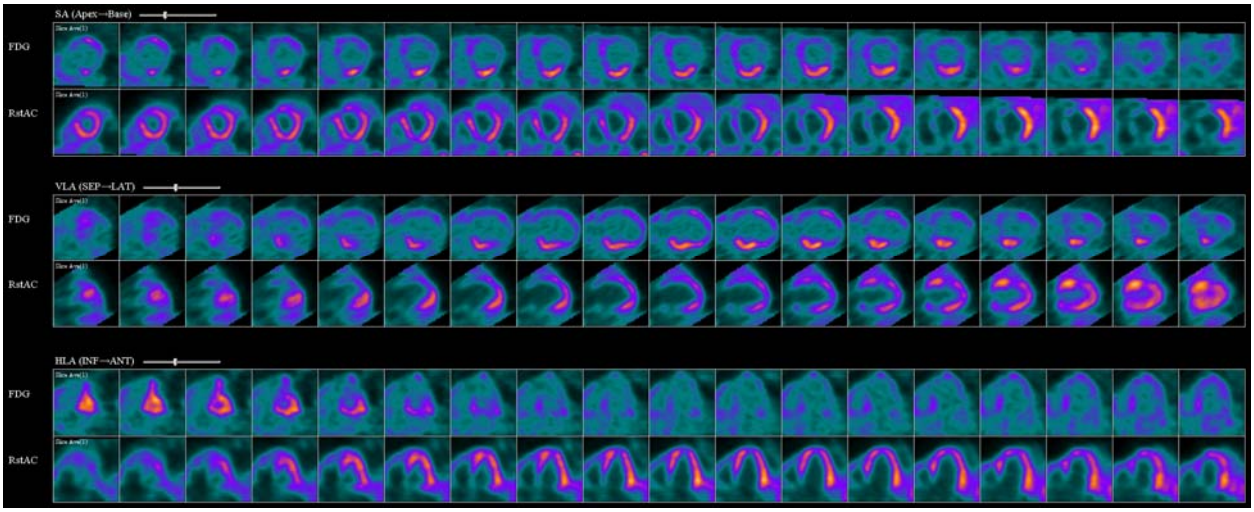

Image B. Improvement of the perfusion defects () with only blood pool activity on the FDG images.