Novel dietary protocol prior to 18F-fluorodeoxyglucose positron emission tomography to evaluate for cardiac sarcoidosis

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The diagnosis of cardiac sarcoidosis (CS) is challenging. Recently, guidelines incorporated cardiac positron emission tomography (PET) with 18F-Fluorodeoxyglucose (F18-FDG) as a non-invasive diagnostic modality for the detection and follow-up of CS. However, this technique is dependent of patient dietary preparation to suppress physiological myocardial F18-FDG uptake. We present a case of possible CS which highlights a novel preparation protocol that facilitated appropriate myocardial suppression.

Key Words: Inflammation • diseases/processes • PET • modalities • metabolism • imaging agents • tracers • Image guided application • outcomes • sarcoid heart disease

INTRODUCTION

The diagnosis of cardiac sarcoidosis (CS) is challenging, and histological confirmation with endomyocardial biopsy is limited due to patchy myocardial involvement and consequent poor sensitivity. More recently, guidelines have incorporated imaging criteria to aid in establishing a clinical diagnosis of CS.[1-3] Cardiac positron emission tomography (PET) with rubidium-82 (Rb^{82}) or N13-Ammonia for perfusion and ^{18}F-Fluorodeoxyglucose (F18-FDG) for metabolism has become an important non-invasive diagnostic modality for the detection and follow-up of CS.[4] Unlike cardiac magnetic resonance imaging (CMR), PET is more rapidly acquired, less dependent on patient cooperation during the exam, and not subject to interference by intracardiac devices. However, this technique is dependent on patient dietary preparation to suppress physiological myocardial F18-FDG uptake in the myocardium. Several protocols have been utilized to suppress myocardial F18-FDG uptake including prolonged fasting, dietary modification (low carbohydrate and/or high fat diet) and intravenous administration of unfractionated heparin.[5]

We present a case of possible cardiac sarcoidosis which highlights a novel preparation protocol that facilitated appropriate suppression of physiological myocardial glucose utilization.

A 36-year-old woman presented for evaluation of shortness of breath and recent diagnosis of heart failure with reduced ejection fraction of 31% by echocardiogram and 25% by CMR. CMR revealed diffuse multifocal delayed hyperenhancement in a nonischemic patchy mid-myocardial to subepicardial distribution (Figure 1).

Given the above findings, a cardiac monitor was ordered to evaluate presence of arrhythmias, and a cardiac PET was ordered to evaluate for active inflammation possibly related to CS. Sarcoid PET with 15.5 h of fasting (resting glucose of 83 mg/dl) was non-diagnostic for inflammation due to failure to suppress myocardial glucose utilization (Figure 2). She had followed our standard dietary protocol which consists of 24 h high fat, no carbohydrate, no sugar diet followed by $>$ 12 h fast. To assure dietary compliance, the patient

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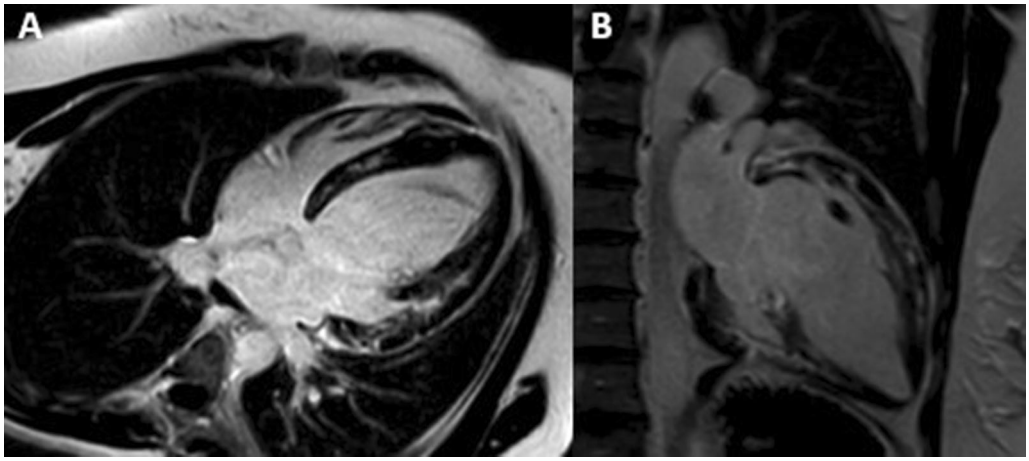

Figure 1. Four chamber long axis (**A**) and three chamber long axis (**B**) showing multiple areas of delayed gadolinium enhancement in a mid-myocardial and subepicardial pattern.

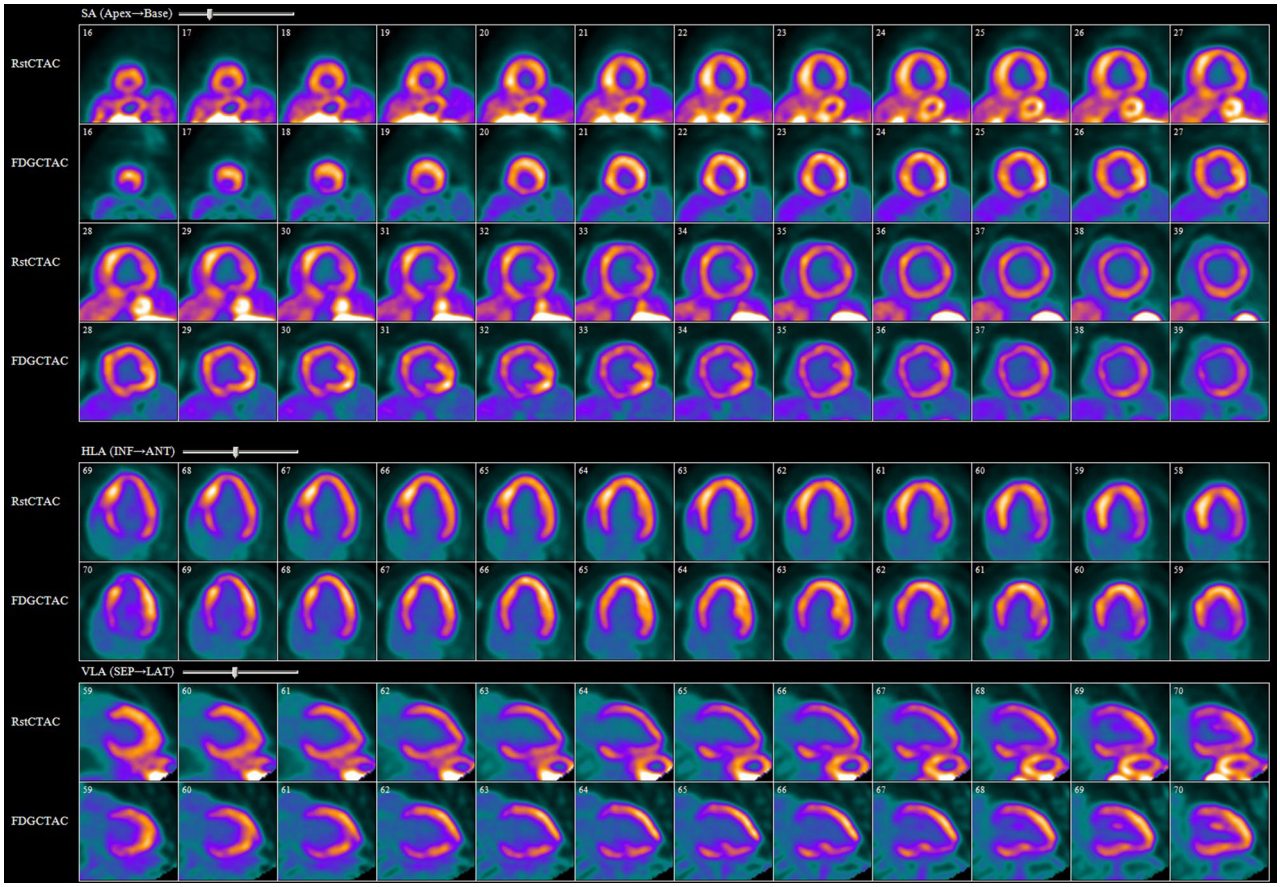

Figure 2. Non-diagnostic cardiac PET F18-FDG due to failure to suppress myocardial FDG uptake evidenced by global FDG uptake of the myocardium (above: resting Rb82 perfusion; below: F18-FDG images which show diffuse myocardial uptake of F18-FDG).

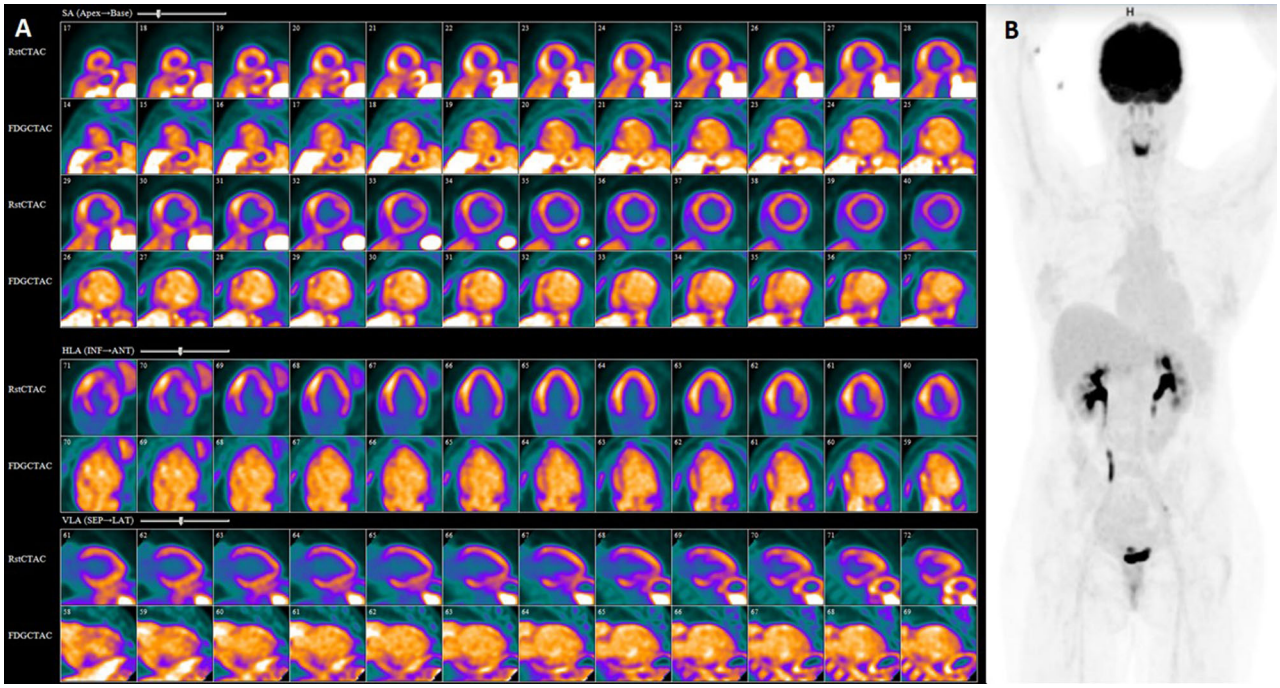

Figure 3. Cardiac PET F18-FDG showing lack of F18-FDG uptake by the myocardium suggesting absence of active inflammation (**A** resting Rb-82 perfusion and F18-FDG images which show myocardial blood pool signal; **B** time-of-flight images with no increased myocardial F18-FDG signal).

is given a list of ''allowed food'' and ''not allowed food'', and a food diary is reviewed prior to F18-FDG PET.

A repeat F18-FDG PET was pursued following a novel dietary protocol 9 days after the initial F18-FDG PET. This protocol consists of 24 h low carbohydrate, high fat diet with two meals (lunch and dinner of the day before the test) replaced with a ketone-based infant formula (Ross Carbohydrate Free by Abbott) followed by 12 h fasting. This formula is a soy based formula that contains 384 ml, 399 kJ, 4 g of protein, 0.007 g carbohydrate, and 7.2 g of fat per can. This time, she presented following a 15 h fast with a blood glucose of 71 mg/dl and a serum β-Hydroxybutyrate was elevated at 1.69 mmol/L (ref $<$ 0.28 mmol/L). Her F18-FDG PET revealed no evidence of active inflammation with only myocardial blood pool signal (Figure 3).

A major obstacle with F18-FDG PET in the diagnosis and follow up of CS is the unpredictable physiologic myocardial FDG uptake, which can result in a low diagnostic accuracy. A metaanalysis and systemic review revealed patient preparation significantly impacts diagnostic performance of F18-FDG PET for the diagnosis of CS, with sensitivity and specificity ranging between 33 and 100%.[6] A more recent study by Lu et al. showed that a prolonged 72 h instead of a 24 h high fat low carbohydrate diet followed by 12 h fasting was more successful in suppressing myocardial FDG uptake.[7] Currently, there is no gold standard preparation for F18-FDG cardiac PET as myocardial FDG uptake is highly variable between individuals. However, the protocol we present standardizes the composition of caloric intake and may lead to more consistent suppression of physiologic myocardial glucose utilization.

Disclosures

The authors have no financial relationships to disclose.

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