PET/CT Case Series: Unmasking the Mystery of Cardiac Sarcoidosis

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The annual incidence of cardiac sarcoidosis is low, but increased awareness of this inflammatory condition has led to earlier disease recognition. High mortality rates compel prompt diagnosis and intervention to reduce the risk of major adverse cardiac events. This cardiac sarcoid PET/CT case series provides information on cardiac sarcoid features, reviews advanced imaging and its role in treatment monitoring, examines the importance of dietary modifications required for ^{18}F-FDG cardiac sarcoid imaging and the associated consequences of noncompliant patient preparation, and provides an in-depth review of a patient's cardiac sarcoid journey.

Key Words: cardiac sarcoidosis; immunosuppressive; metabolic shift; inflammation; HFHPVLC diet

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Have you ever had a patient plead, bargain, or speak untruths when questioning if they followed their patient preparation instructions? "It was just one bite" or "it was only a sip or two" are phrases capable of crushing the heart of any nuclear medicine technologist interviewing a patient to verify patient prep was followed for an ^{18}F-FDG cardiac sarcoid scan. This case series will explore the effects of patient prep compliance for ^{18}F-FDG PET cardiac sarcoid scans. We will review and discuss a patient's systemic sarcoidosis diagnosis and their cardiac sarcoidosis imaging journey. In addition to patient prep woes, we will also report the impact of high baseline blood glucose levels, improper ^{18}F-FDG cardiac intensity display, and the key role serial ^{18}F-FDG PET scans have in evaluating and monitoring sarcoidosis. Let us explore how PET impacts cardiac sarcoidosis staging and treatment monitoring!

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BACKGROUND

The incidence and prevalence of systemic sarcoidosis are likely underestimated and vary on the basis of race, sex, and region. Data show prevalence rates ranging from 100 cases per 100,000 up to 330 cases per 100,000, with heavy influence stemming from environmental and genetic factors (*1*). The disease is more prevalent in women than in men, with Black Americans facing a higher prevalence and incidence (*2*). Individuals aged 20–40 y face the highest incidence, yet the mortality rate associated with systemic sarcoidosis remains low. Since sarcoidosis is a systemic disease, it can affect any organ, and 25% of patients will have other organ involvement during the first 2 y after their initial diagnosis (*1*). Up to 90% of sarcoidosis cases involve the lung, making it the most often involved organ. Clinically silent or asymptomatic cardiac sarcoidosis is present in 20%–25% of sarcoidosis patients, and only 5% of patients display clinically evident cardiac involvement (*2–5*).

What red flags prompt clinicians to further evaluate for cardiac sarcoidosis? Diagnosis is challenging because cardiac sarcoidosis is known as the great masquerader (*2*). Clinical presentations will vary on the basis of the extent and location of cardiac inflammation, and a diagnosis relies on tissue sampling positive for noncaseating granulomas, suspicious clinical presentation, inclusion of specific cardiac sarcoidosis criteria, and exclusion of other causes, all paired with a suspicious clinical appearance (*6*). Electrocardiography may show a variety of nonspecific findings (*2*). Echocardiography is a readily available resource and helps follow left ventricle (LV) function; despite this, it is insensitive to early changes, yielding 10%–47% sensitivity and 82%–99% specificity (*4*). Suspicious echocardiography findings may include reduced LV ejection fraction (LVEF), regional wall aneurysm, or basal septal thinning without coronary artery disease (CAD). Additionally, hypercalcemia, high-grade atrioventricular block (age less than 60 y), or unexplained ventricular arrhythmia can prompt further evaluation. To further complicate clinical workup, electrocardiography and echocardiography can yield normal findings in cardiac sarcoid patients. The stakes are high in detecting cardiac sarcoidosis as early as possible

because of its impact on LV function. When risk stratification includes LV function, patients with an LVEF greater than 50% at diagnosis have an 80% 10-y survival rate. In contrast, 10-y survival rates plummet to 19% if the LVEF is less than 30%. Because of the considerable risk of cardiac sarcoidosis affecting LV systolic function (*1*), advanced imaging techniques such as cardiac MRI (CMR) and ^{18}F-FDG PET are used (*2*).

Early diagnosis with CMR and PET in tandem with prompt treatment can help potentially prevent cardiac complications (*7*). In CMR, normal myocardial tissue rapidly washes out gadolinium contrast; slow gadolinium washout occurs in areas of fibrosis or scarring and inflammation caused by sarcoidosis. These areas are said to have late gadolinium enhancement (LGE), and when used alongside reference clinical criteria, CMR can reach 95% sensitivity and 85% specificity. CMR provides a high negative predictive value and is the initial advanced imaging test if clinical suspicion for cardiac sarcoidosis is low. However, CMR is unable to completely exclude cardiac sarcoidosis since early stages of disease involvement may not cause LGE; early disease may go undetected. ^{18}F-FDG PET's high diagnostic accuracy may help catch inflammatory changes sooner than CMR because the actively inflamed noncaseating granulomas present in cardiac sarcoidosis can be localized on metabolic ^{18}F-FDG PET before LGE is appreciated on CMR. PET is also helpful when a high pretest probability remains after a nondiagnostic, equivocal, or negative CMR or if CMR is contraindicated (*2*).

Before imaging with ^{18}F-FDG PET, the presence of obstructive CAD or a history of prior myocardial infarction should first be evaluated using rest/stress myocardial perfusion imaging (MPI), CT coronary angiography, or invasive coronary angiography. Based on risk factors and patient age, coronary angiography may be more appropriate for excluding anatomic stenosis in patients with suspected cardiac sarcoidosis (*1*). Once CAD and myocardial infarction are excluded, rest perfusion data are acquired to evaluate perfusion defects triggered by scarring or the inflammatory compression of the myocardial microvasculature. Rest MPI is used in conjunction with ^{18}F-FDG PET for myocardial colocalization and can be performed on the same day or on a different day as ^{18}F-FDG PET (*1,4*). PET rest MPI is preferential over SPECT because of PET's ability to detect small perfusion defects commonly present in patients with cardiac sarcoidosis. If PET is unavailable, SPECT data can be acquired but should include gated acquisition data and the use of attenuation correction (AC) to help circumvent attenuation artifacts (*4*).

With an ^{18}F-FDG PET cardiac sarcoid imaging protocol, it is crucial first to suppress physiologic myocardial glucose consumption. Under normal dietary conditions, myocardial cells use a mixture of fatty acids and glucose for their primary energy needs. Therefore, normal myocardial metabolism must be switched to only fatty acid metabolism (*1,4,7*). A combination of strategies can induce this metabolic switch, including diet modification, prolonged fasting, or administration of unfractionated heparin (UFH). Diet modification is the most used strategy (*8*). Through strict patient adherence to a high-fat, high-protein, very low-carb (HFHPVLC) diet, myocardial metabolism can be shifted to fatty acid metabolism. Examples of permitted food include plain meat (e.g., beef, pork, poultry, or fish) cooked without breading, real butter or olive oil, plain eggs, salt and pepper, plain water, plain black coffee, or plain black tea. Unacceptable foods include all processed foods (lunch meat, hot dogs), vegetables, grains, baked goods, candy, fruits, sweeteners, alcohol, condiments, etc. Further reading regarding specific dietary recommendations is provided by Chareonthaitawee et al. (*1*). If followed for 24–48 h, a strict HFHPVLC diet, in combination with a prolonged 12- to 18-h (overnight) fast will increase normal myocardial fatty acid metabolism, minimize normal myocardial glucose utilization, and improve the ^{18}F-FDG myocardial target-to-background ratio needed to help identify active cardiac sarcoidosis (*1,8*). Complying with a HFHPVLC diet for 24 h can suppress myocardial glucose utilization in up to 80% of patients, and extending the diet to 72 h can yield a 95% suppression rate (*2*). If a patient cannot follow the recommended diet, fasting for more than 18 h is recommended. The adjunct use of intravenous administration of UFH before ^{18}F-FDG injection may also increase myocardial fatty acid metabolism and suppress myocardial glucose uptake, but its impact may be lower than initially understood. Consequently, the use of UFH continues to present ambiguous value and is a topic of debate (*1,2,8*).

Once dietary compliance is confirmed and a rest MPI scan is completed, the patient is injected with ^{18}F-FDG and undergoes the site-specific uptake time, usually 60–90 min. A 1–field-of-view (FOV) ^{18}F-FDG cardiac PET acquisition is acquired and followed by a limited ^{18}F-FDG whole-body (WB) PET scan. A workflow example for a 1-d cardiac sarcoid PET/CT protocol is detailed in Figure 1.

The limited WB scan can assess extracardiac disease, evaluate potential biopsy sites, determine the potential role of systemic immunosuppressive treatment, and evaluate treatment response (*1,4*). Minor deviations from the HFHPVLC diet—even the "it was only one bite" deviation—can result in poor myocardial glucose suppression, resulting in diffuse homogeneous ^{18}F-FDG myocardial uptake, yielding a nondiagnostic scan. Adherence to the HFHPVLC diet is imperative for a successful examination, so detailed patient education and diligent patient compliance are critical to the examination's diagnostic accuracy. Patients are encouraged to document all food and drink consumed during the diet duration and bring their food journal to their PET appointment. Variations from the HFHPVLC diet require consultation with the interpreting physician because rescheduling the examination, restarting the diet, or extending the diet's duration may be needed.

How are diabetic patients managed? Each diabetic patient is unique; patient safety is paramount, and detailed communication with the patient before the diet begins and during the diet's duration is imperative. According to cardiac sarcoid consensus information shared by Chareonthaitawee et al., diabetic and nondiabetic patients should follow the same dietary preparation for cardiac sarcoid imaging but change their

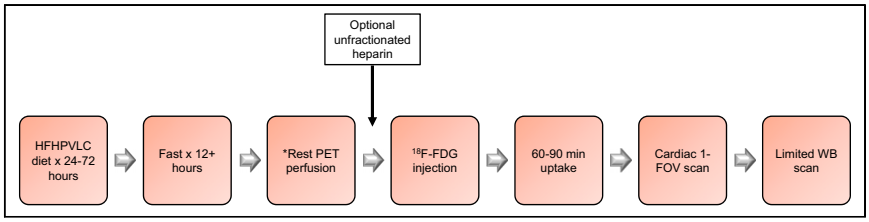

FIGURE 1. Workflow example for 1-d cardiac sarcoid PET/CT imaging. *One-day PET/CT protocols currently use ^{82}Rb-chloride or ^{13}N-ammonia for rest PET perfusion imaging. Use of ^{18}F perfusion radiopharmaceuticals may require increased time between rest PET perfusion and ^{18}F-FDG scans.

medications based on diabetes type. Type 1 diabetics should continue any basal insulin during dietary preparation and try to minimize fast-acting insulin products during the diet if it is believed safe for the patient. Sliding-scale, fast-acting insulin may be needed in the days before the ^{18}F-FDG study and should ideally be avoided on the day of the PET scan. Type 2 diabetics should avoid oral diabetic medications or noninsulin injections during the prolonged fast and on the morning of the PET scan (*1*). Each diabetic patient has unique blood glucose management requirements, and consultation with the ordering health care provider or interpreting physician may be needed before PET scan completion.

CASE SERIES JOURNEY

A 59-y-old woman with a history of biopsy-proven nonnecrotizing granulomatous inflammation presented for chest radiography and CT angiography for chest pain and suspected pulmonary embolism. Chest radiography was unremarkable, but CT angiography showed multiple enlarged hilar and mediastinal lymph nodes requiring further workup. A lymph node aspiration and bronchoscopy yielded noncaseating granulomas indicative of sarcoidosis. Echocardiography showed LV hypertrophy; a rest/stress PET MPI was obtained to evaluate chest pain, dyspnea, bradycardia, bigeminy, and frequent premature ventricular contractions. PET MPI showed no fixed or reversible perfusion abnormalities, diffusely decreased myocardial flow reserve—likely due to microvascular disease—and dilated LV with 46% ejection fraction. After the MPI, a CMR was obtained which showed a transmural scar in the basal to mid inferolateral wall worrisome for cardiac sarcoidosis. The patient was referred to PET for ^{18}F-FDG cardiac sarcoidosis imaging. A total of 4 ^{18}F-FDG PET/CT scans were performed on the patient throughout their cardiac sarcoid journey.

PET/CT Scan 1

The patient presented to the PET clinic for cardiac sarcoidosis imaging with a body mass index (BMI) of 41 and an elevated fasting blood glucose of 131 mg/dL. Patient compliance with HFHPVLC dietary prep requirements was confirmed, and standard institutional clinical ^{18}F-FDG patient preparation about long- and short-acting insulin-containing medication was verified, if applicable. Per department protocol, the patient underwent routine ^{82}Rb-chloride rest perfusion imaging to evaluate perfusion defects. The patient received weight-based UFH 15 min before the ^{18}F-FDG injection. Uptake times for ^{18}F-FDG 1-FOV cardiac and limited WB scans were 60 min and 78 min, respectively (Figs. 2–4).

Scan 1 Discussion

An accurate comparison of ^{18}F-FDG cardiac metabolism with rest perfusion images is imperative to help accurately stratify patient risk. It is important for technologists to understand that rest perfusion defects caused by cardiac sarcoidosis can look different than defects caused by CAD. Cardiac sarcoid perfusion defects can result from altered coronary microcirculation, and these defects may not align with coronary territories (*1*), yielding perfusion defects in odd locations. ^{18}F-FDG uptake in the right ventricle, a less common finding in routine MPI imaging, places patients at an extremely high risk for cardiac events. Technologists should also be aware of the potential for hardware-induced image artifacts. For example, cardiac device insertion sites can create CT AC errors, so AC and non-AC ^{18}F-FDG images should be reviewed to overcome AC-induced artifacts. Additional information discussing perfusion-metabolism uptake patterns is available in multiple publications (*1,4,6,7*). Positive PET findings have huge implications for patients; when PET imaging is positive for rest perfusion defects and abnormal glucose metabolism, patients have a 4 times higher risk of ventricular arrhythmias or death (*1,4*).

Why was ^{18}F-FDG cardiac uptake absent when the MRI showed concern for the basal to the mid inferolateral wall? Recall the patient's fasting blood glucose was 131 mg/dL. Hyperglycemia can affect the diagnostic accuracy of detecting cardiac sarcoidosis on ^{18}F-FDG PET. Even though the patient denied a personal history of diabetes, ensuing labs led to a diabetes diagnosis, and the patient started oral diabetic medication for blood glucose management. The patient's initial baseline hyperglycemia was likely caused by undiagnosed diabetes. Patient blood glucose levels greater than 120–200 mg/dL may warrant examination rescheduling (*7,9*). This proposed lower 120 mg/dL glucose threshold for cardiac sarcoidosis may be more stringent than the recommended 150–200 mg/dL threshold used for many ^{18}F-FDG oncology examinations. Glucose levels for cardiac sarcoid examinations may vary on the basis of the interpreting physician's preference. Because of the depressed LVEF seen on both CMR and PET/CT, the lingering presence of intermittent bradycardia, and enduring cardiac arrhythmias, the patient was prescribed prednisone and methotrexate as a treatment for presumed cardiac sarcoidosis.

Immunosuppressive drugs are commonly used to treat cardiac sarcoidosis, but with the high toxic side-effect profile of these medications, advanced imaging plays a key role in decision-making regarding treatment initiation and modification (*1,4*). Corticosteroids such as prednisone are a first-line

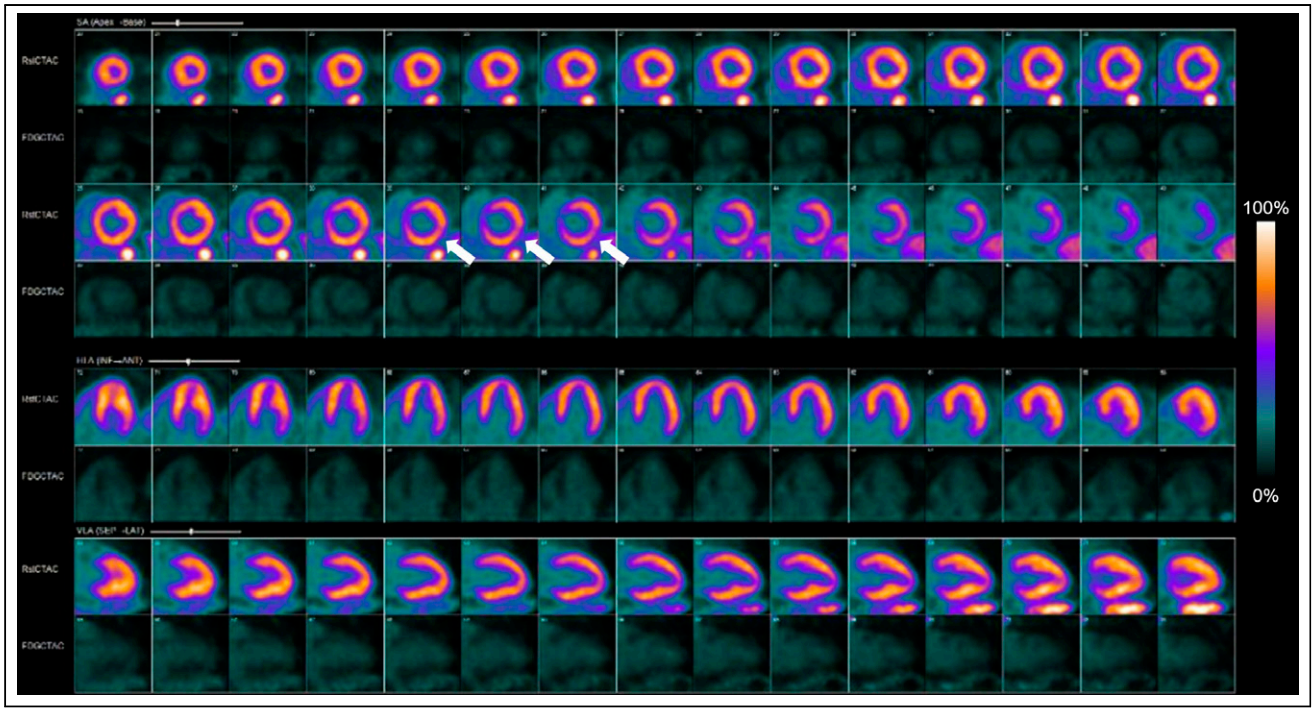

FIGURE 2. Scan 1 ^{82}Rb-chloride rest perfusion (top rows) and ^{18}F-FDG cardiac metabolism (bottom rows). Rest perfusion images show small mid inferolateral wall defect (white arrows) and normal distribution of activity throughout remaining LV myocardium. Gated rest LVEF is 32%. ^{18}F-FDG cardiac slices show suboptimal display intensity for ^{18}F-FDG cardiac sarcoidosis imaging, as neither ^{18}F-FDG myocardial blood-pool activity nor activity in LV myocardial wall can be appreciated in current display.

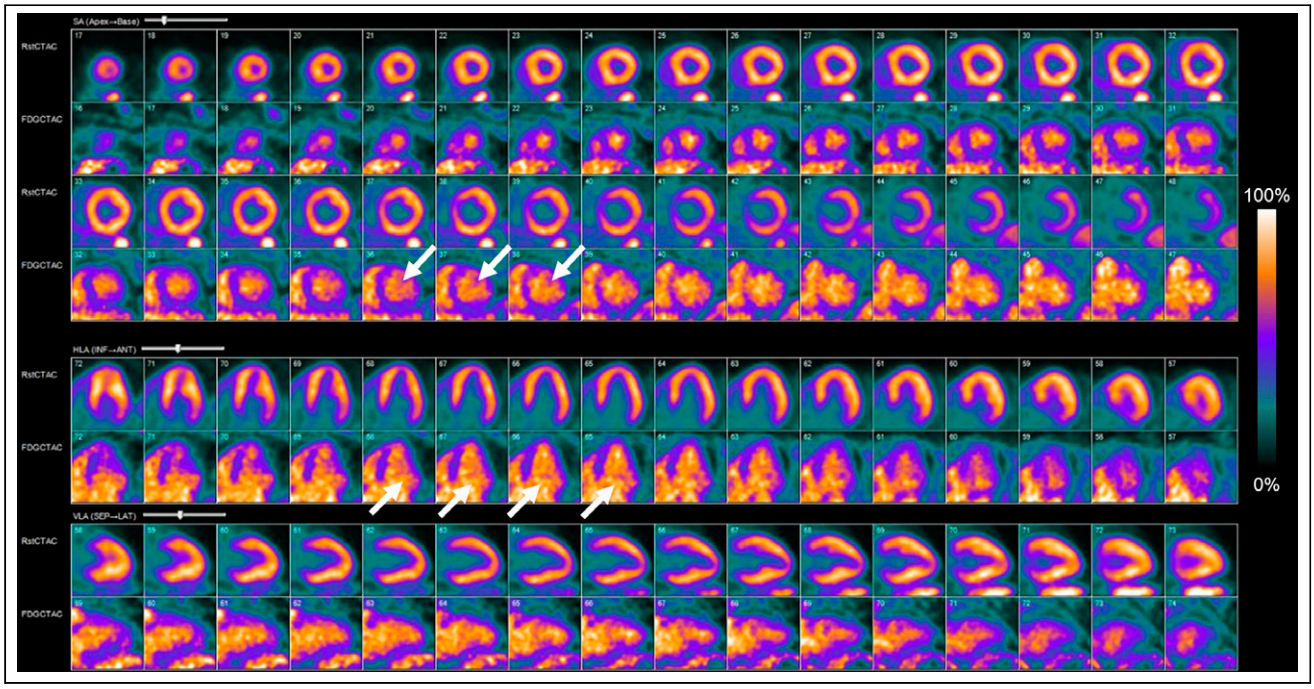

FIGURE 3. Scan 1 ^{82}Rb-chloride rest perfusion (top rows) and rescaled ^{18}F-FDG cardiac metabolism (bottom rows). Images show appropriate ^{18}F-FDG display intensity with blood-pool activity (white arrows) brighter than LV wall, indicating normal FDG uptake pattern. No evidence of any focally increased ^{18}F-FDG uptake in LV myocardial wall is seen, which indicates negative examination for active cardiac sarcoidosis.

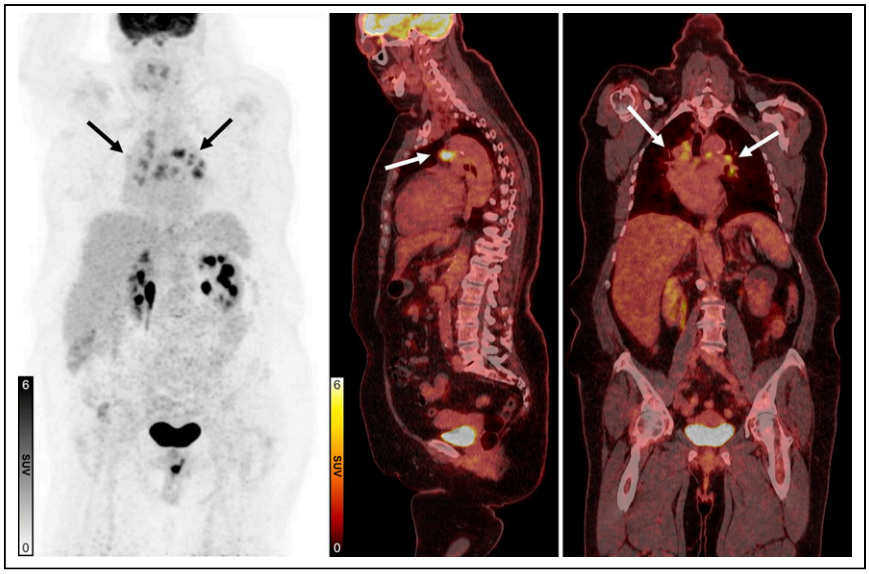

FIGURE 4. Scan 1 ^{18}F-FDG maximum-intensity projection (left), fused ^{18}F-FDG PET/CT sagittal (middle), and fused ^{18}F-FDG PET/CT coronal (right) views. Images show diffuse hypermetabolic uptake (arrows) in mediastinal and hilar lymphadenopathy biopsy-proven noncaseating granulomatous disease. ^{18}F-FDG PET/CT scan 1 is negative for cardiac sarcoidosis but positive for extracardiac sarcoidosis.

treatment option and can improve atrioventricular block. Another treatment choice includes combination therapy of a steroid-sparing agent (methotrexate) and a corticosteroid if the patient is not able to tolerate moderate to higher corticosteroid doses. This combination therapy can also serve as a second-line treatment if corticosteroids alone fail. If second-line therapy fails, tumor necrosis factor-α–targeted therapy (infliximab or adalimumab) can be used as a third-line treatment but requires cautious use and diligent monitoring at regular intervals (*2*).

Despite combination therapy, over the next 3.5 y—which so happened to coincide with the onset of the COVID-19 pandemic—the patient continued to experience cardiac arrhythmias, chest pain, dyspnea, generalized aches, and fatigue. Because of the length of time elapsed since the last MPI, a repeat rest/stress PET MPI test was performed to rule out occlusive CAD; this showed LV enlargement, normal LV myocardial perfusion, no significant perfusion defects, normal myocardial flow reserve values, and depressed rest and stress LVEFs of 37% and 38%, respectively.

PET/CT Scan 2

Two months after repeat rest/stress PET MPI, the patient presented for repeat ^{18}F-FDG cardiac sarcoidosis imaging to evaluate combination therapy response; BMI was 42, and fasting blood glucose was 110 mg/dL. Patient compliance with HFHPVLC dietary prep requirements was confirmed, and standard institutional clinical ^{18}F-FDG patient prep covering long- and short-acting insulin-containing medication was followed. The patient did not receive UFH for scan 2 because of staff physicians' request for the removal of this adjunct medication from the clinical protocol. ^{18}F-FDG cardiac 1-FOV and limited WB scan uptake times were 60 min and 75 min, respectively (Figs. 5 and 6).

Scan 2 Discussion

Scan 2 is an example of an excellent, diagnostic quality scan resulting from adequately controlled fasting blood glucose levels and strict patient compliance with a HFHPVLC diet. When myocardial glucose metabolism is adequately suppressed, ^{18}F-FDG PET has high diagnostic accuracy (*6*) and serves as a robust imaging tool to help monitor disease activity and assess treatment response (*5*). Like oncology examinations, serial ^{18}F-FDG PET scans for cardiac sarcoidosis should be performed with consistent protocol parameters: similar dietary prep and fasting requirements, comparable uptake times, similar injected activity, and analogous display parameters. Serial ^{18}F-FDG–limited WB images should be assessed qualitatively and quantitatively to prevent errors caused by software normalization (*1*). Posttreatment CMR and PET provide valuable guidance for treatment decisions; if LGE persists in CMR postimmunosuppressive treatment in patients with an LVEF of 35%–49% or if the patient's LVEF is less than 35%, as it was on scan 2, pacemaker or implantable cardioverter defibrillator placement may be considered (*4*).

Positive myocardial ^{18}F-FDG uptake on scan 2 indicates the patient failed first- and second-line treatments. Uptake in the abdominal lymph node was worrisome for an increase in extracardiac sarcoid involvement. Because ^{18}F-FDG PET showed a lack of treatment response, the patient changed to a third-line treatment of infliximab infusions. Due to the continued low LVEF present on serial ^{18}F-FDG PET imaging, the patient was referred for automatic implantable cardioverter defibrillator placement.

The next 10 mo presented challenges for the patient and health care team. For various personal reasons, the patient was delayed in starting infliximab infusions and then became noncompliant with receiving scheduled infliximab infusions. The patient did have an automatic implantable cardioverter defibrillator placed after scan 2, but the patient continued to struggle with dyspnea, cough, and weight gain.

PET/CT Scan 3

The patient returned 10 mo later for a third ^{18}F-FDG PET cardiac sarcoidosis scan; BMI was 40, and fasting blood glucose was 103 mg/dL. Patient compliance with HFHPVLC prep requirements was confirmed, and standard institutional clinical ^{18}F-FDG patient prep regarding long- and short-acting insulin-containing medication was followed. ^{18}F-FDG cardiac

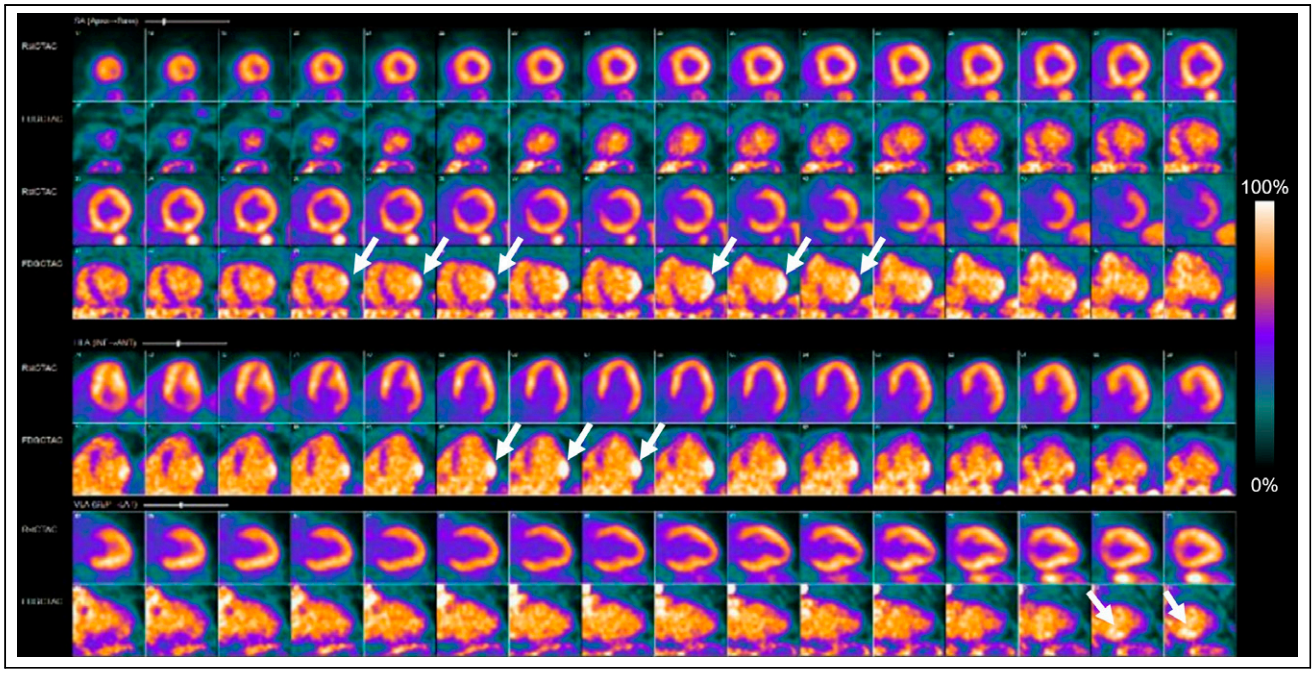

FIGURE 5. Scan 2 ^{82}Rb-chloride rest perfusion (top rows) and ^{18}F-FDG cardiac metabolism (bottom rows). Images show diagnostic evaluation with ^{18}F-FDG blood-pool activity indicating adequate dietary preparation. Rest images show borderline hypoperfusion in basal to mid inferolateral wall and moderate LV enlargement. Rest gated images showed 31% LVEF. ^{18}F-FDG images show suspected early-to-progressive stable cardiac sarcoidosis with moderate inflammatory ^{18}F-FDG uptake in basal to mid inferolateral wall (arrows).

1-FOV and limited WB scan uptake times were 69 min and 84 min, respectively (Figs. 7 and 8).

Scan 3 Discussion

Scan 3 provided an extremely limited evaluation of cardiac sarcoidosis due to high baseline cardiac ^{18}F-FDG uptake. ^{18}F-FDG blood-pool activity was not visualized, indicating inadequate myocardial glucose suppression.

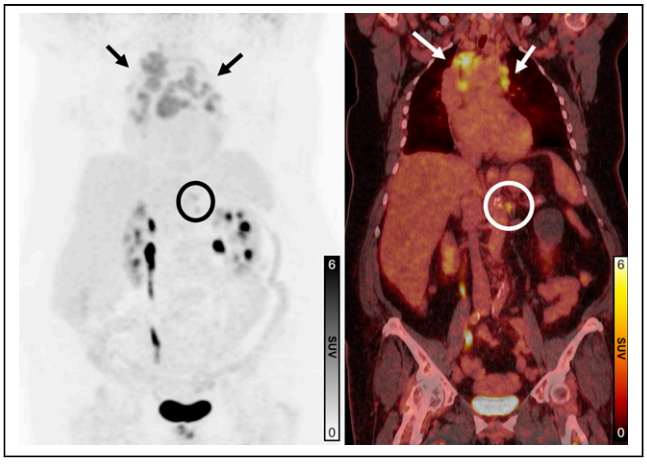

FIGURE 6. Scan 2 ^{18}F-FDG maximum-intensity projection (left) and ^{18}F-FDG PET/CT fused coronal (right) images. Persistent ^{18}F-FDG–avid bilateral mediastinal and hilar/perihilar adenopathy is visualized from known extracardiac sarcoidosis (arrows). Solitary ^{18}F-FDG–avid left upper abdominal node is suspicious for additional sarcoid involvement (circles).

Extensive mediastinal, supraclavicular, and retroperitoneal hypermetabolic lymphadenopathy was appreciated with interval development of new or enlarged lymph nodes since scan 1. These findings were consistent with extracardiac sarcoidosis. Examination findings were also worrisome for bilateral rib involvement.

What caused the high baseline ^{18}F-FDG cardiac uptake in this scan? Even though the patient told PET clinic staff they followed the HFHPVLC diet, the patient was not truthful. During a post-PET scan follow-up appointment, the patient confessed to eating "a couple of grapes" during the 1–2 d before the PET scan. The consumption of grapes had likely prevented the myocardial cells from shifting over to fatty acid metabolism. The grapes reinforced the heart's normal resting metabolic conditions, which ensured active cardiac glucose metabolism at the time of ^{18}F-FDG injection. Had the patient been honest about their grape indulgences, the examination could have been rescheduled, thus preventing a nondiagnostic examination. Regrettably, scan 3 could not provide accurate diagnostic results about cardiac sarcoidosis treatment response.

PET/CT Scan 4

The patient continued noncompliance with infliximab infusions for cardiac sarcoidosis treatment over the next 11 mo. Infusions finally resumed at the patient's request, and a repeat PET/CT scan was ordered. The patient presented 14 mo after scan 3 for ^{18}F-FDG cardiac sarcoidosis imaging; BMI was 43, and fasting blood glucose was 106 mg/dL.

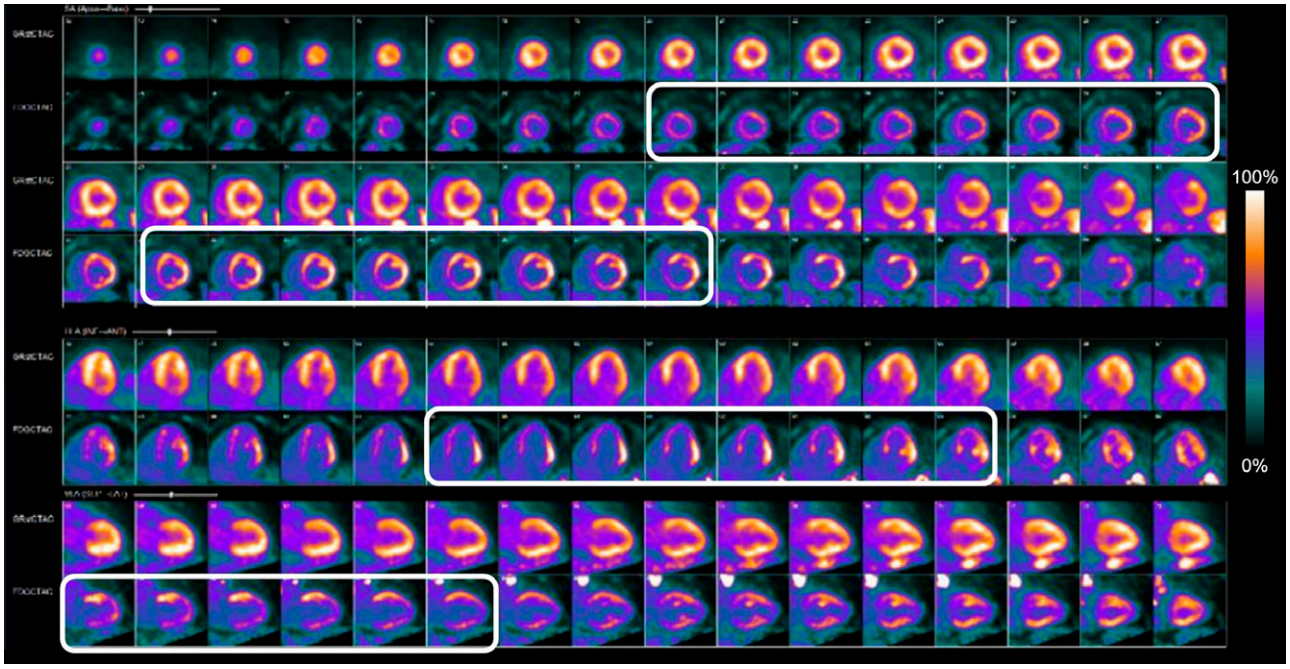

FIGURE 7. Scan 3 ^{82}Rb-chloride rest perfusion (top rows) and ^{18}F-FDG cardiac metabolism (bottom rows). Rest MPI shows no significant perfusion defects in LV. ^{18}F-FDG PET images show diffusely increased myocardial uptake (rectangles), more prominent in lateral wall. This pattern of diffusely increased ^{18}F-FDG cardiac uptake indicates hyperinsulinemic state, suggesting possible inadequate dietary compliance, and limits evaluation of ^{18}FDG-PET images for cardiac sarcoidosis. Gated rest images show estimated LVEF of 24%.

Patient compliance with HFHPVLC prep requirements was confirmed, with no delectable grapes being included in the diet, and standard institutional clinical ^{18}F-FDG patient prep regarding long- and short-acting insulin-containing medication was followed. ^{18}F-FDG cardiac 1-FOV and limited WB scan uptake times were 62 min and 80 min, respectively (Figs. 9 and 10).

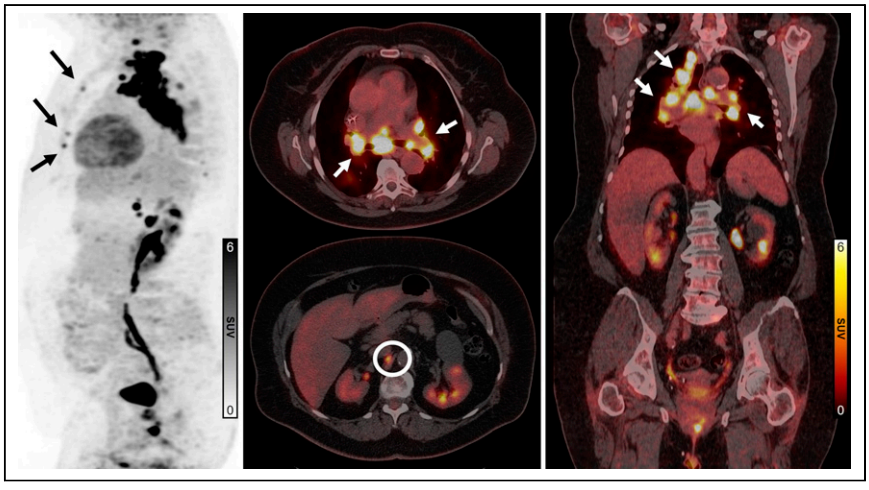

FIGURE 8. Scan 3 ^{18}F-FDG sagittal maximum-intensity projection (left), ^{18}F-FDG PET/CT fused axial (middle), and ^{18}F-FDG PET/CT fused coronal (right) views. Images show mediastinal, supraclavicular (white arrows), and retroperitoneal (circle) hypermetabolic lymphadenopathy with new or enlarged lymph nodes since scan 1, consistent with sarcoidosis with possible involvement of bilateral ribs (black arrows).

Scan 4 Discussion

Scan 4 showed adequate dietary preparation. Borderline rest hypoperfusion and inflammatory ^{18}F-FDG uptake remain in the basal to mid lateral inferolateral wall, similar to prior examinations. A new area of mild inflammatory ^{18}F-FDG uptake was seen in the basal anteroseptal wall and indicated disease progression since the prior scan. No other sites of cardiac sarcoidosis were noted. Marked improvement in extracardiac sarcoid disease was seen and represented by a decreased burden of mediastinal and bilateral hilar/perihilar lymphadenopathy. Previous examinations showed progressively worsening disease. The retroperitoneal paraaortic adenopathy also improved since the prior examination. Unfortunately, an incidental renal cell carcinoma was suspected because of an ^{18}F-FDG–avid exophytic 1.7-cm isodense nodule in the mid left renal pole. This isodense nodule was new since scan 1 and had slowly enlarged since scan 2. Ultrasound was recommended for further evaluation.

The importance of obtaining the limited WB ^{18}F-FDG scan in all sarcoidosis patients extends beyond monitoring extracardiac disease extent and treatment

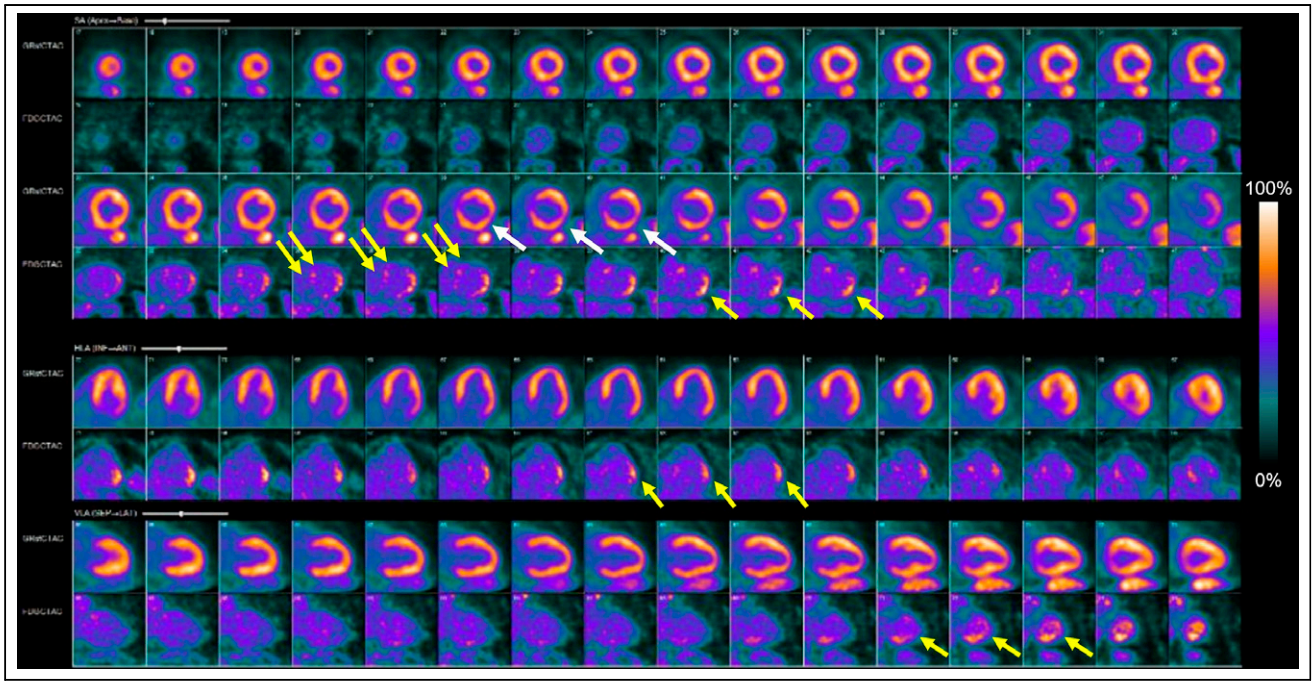

FIGURE 9. Scan 4 ^{82}Rb-chloride rest perfusion (top rows) and ^{18}F-FDG cardiac metabolism (bottom rows). Rest perfusion images show medium-sized mild defect in basal to mid lateral and inferolateral wall (white arrows). Gated rest LVEF was 31%. ^{18}F-FDG images show diffusely suppressed myocardial uptake, indicating adequate dietary preparation. Medium-sized area of increased ^{18}F-FDG uptake is visualized in basal to mid lateral and inferolateral wall (single yellow arrows) similar to scan 2. New small-sized area of mildly increased ^{18}F-FDG uptake is visualized in basal anteroseptal wall (double yellow arrows).

response because patients with sarcoidosis have a higher risk of malignancy (*7,10*). Due to common lung involvement, sarcoidosis often affects thoracic lymph nodes, especially the hilar and mediastinal regions; these areas are amenable to biopsy and help differentiate sarcoidosis-related lesions from pathologic ^{18}F-FDG uptake attributed to malignancy or infection (*1,4*). The sarcoidosis inflammatory process significantly affects certain organs such as the kidneys and skin, thereby strengthening the association between sarcoidosis and malignancy in these organs (*10*). The patient was referred to urology for follow-up of the renal lesion.

For patients with cardiac sarcoidosis, serial ^{18}F-FDG PET is a robust tool for monitoring treatment response (*11*), but what is the ideal timing interval between serial PET scans? Published data are currently limited, and although optimal frequency schedules are still being established, 3- to 6-mo intervals are generally used (*2*). New data from a small, retrospective, observational study showed patients on moderate-dose prednisone therapy had similar treatment response rates regardless of follow-up time between PET scans; approximate median times between follow-up scans were 3.1, 5.9, and 9.8 mo. No significant differences were seen in short-term major adverse cardiovascular events. In summary, a treatment strategy with early surveillance using ^{18}F-FDG PET imaging yields results comparable to a similar approach with delayed ^{18}F-FDG PET imaging (*11*). Prolonged corticosteroid therapy can alter serum insulin and glucose levels, potentially affecting normal myocardial cell uptake (*5,7*); the exact influence of steroid-included glucose metabolic changes on myocardial cells is unknown (*4*). Other

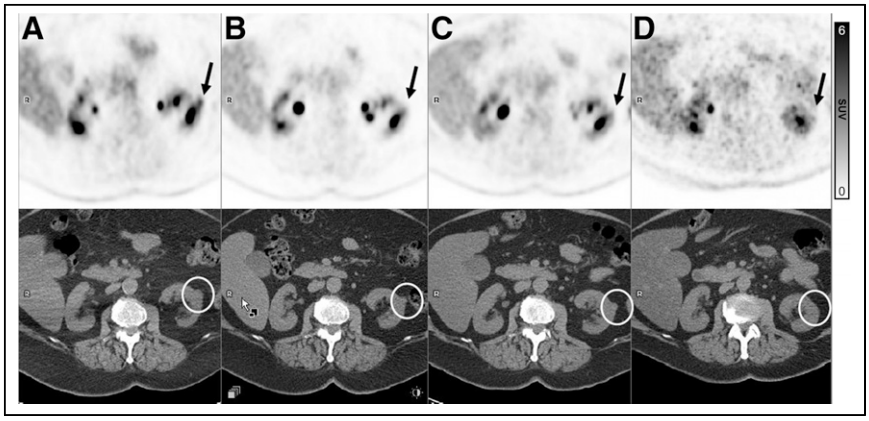

FIGURE 10. ^{18}F-FDG PET axial (top row) and CT axial (bottom row) images from scan 4 (A), scan 3 (B), scan 2 (C), and scan 1 (D). CT axial images from limited WB ^{18}F-FDG PET/CT scan show incidental ^{18}F-FDG–avid exophytic 1.7-cm isodense nodule in mid left renal pole (circles). This lesion is new since scan 1, has slowly enlarged since most recent examination, and displays increased ^{18}F-FDG activity on scan 4 (arrows).

information discussing serial ^{18}F-FDG PET imaging is likely forthcoming.

CONCLUSION

Whether it is grapes, granola, guacamole, or gummy bears, the phrase "it was just a few bites" may indeed be enough to compromise the diagnostic accuracy of an ^{18}F-FDG cardiac sarcoid scan. The importance of proper dietary modifications, in combination with prolonged fasting, provides the ideal metabolic environment to shift myocardial cells from glucose metabolism to fatty acid metabolism. Incorrect patient prep and baseline hyperglycemia can alter ^{18}F-FDG uptake, and 5%–20% of patients may exhibit suboptimal myocardial glucose suppression following adherence to cardiac sarcoid preparation protocols (*6*).

How can glucose suppression be confirmed? New data show measuring β-hydroxybutyrate (BHB) levels via point-of-care ketone testing before ^{18}F-FDG injection may help verify myocardial glucose suppression (*6,8*). Data show a linear relationship between BHB levels and improved myocardial glucose suppression. The longer a HFHPVLC diet's duration, the higher the BHB levels. Improved rates of glucose suppression and higher rates of diagnostic examinations correlate with incremental increases in BHB levels (*12*). Although these correlations are promising, additional research is needed to define thresholds and understand patient-specific variations (*8*).

Cardiac sarcoidosis is the great masquerader and presents a substantial risk to LV function. Early diagnosis with advanced imaging techniques such as CMR and ^{18}F-FDG PET can help thwart cardiac complications. Nuclear medicine technologists must recognize the implications of dietary compliance, patient honesty, and technical factors on ^{18}F-FDG PET's diagnostic accuracy. As cardiac sarcoid diagnoses increase because of enhanced awareness and advanced imaging techniques, so does the significance of the nuclear medicine technologist's role in ensuring high-quality ^{18}F-FDG cardiac sarcoid imaging.

DISCLOSURE

No potential conflict of interest relevant to this article was reported.

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