Myocardial Metastasis or Benign Brown Fat?

Peter M. Rao, MD; Pamela K. Woodard, MD; G. Alexander Patterson, MD; Linda R. Peterson, MD

Noninvasive positron emission tomography (PET) imaging of cell glucose metabolism has become an essential tool for assessing tissue viability in cardiac (hibernating myocardium) and malignant disease (tumor regression or recurrence). However, PET studies alone provide information regarding tissue and organ metabolism but may not provide enough spatial resolution for accurate assessment of critical structural changes that are the cause or consequence of metabolic perturbations. The following case illustrates the pitfalls of relying on a single cardiac imaging modality and demonstrates how integration of metabolic data from PET with structural data from echocardiography, computed tomography (CT), or MRI studies can be used to correctly identify and manage a diagnostic dilemma.

A 63-year-old woman with a medical history of chronic obstructive pulmonary disease and tobacco use was diagnosed with a left lung mass on a chest x-ray examination. A combined CT/fluorodeoxyglucose (FDG)-PET scan at another institution revealed the left lung mass (Figure 1) as well as a cardiac mass with increased uptake of radiotracer concerning for a cardiac metastasis (Figure 2). The CT Hounsfield unit (HU) density in the cardiac mass was −57 HU. She was referred for transesophageal echocardiography in the case presented (Figure 3). She had marked thickening of the interatrial septum with sparing of the fossa ovalis. A cardiac MRI (Figure 4) also demonstrated a dumbbell-shaped thickening of the interatrial septum that spared the fossa ovalis. The signal intensity of the interatrial mass was the same as that of the subcutaneous fat on all imaging sequences without evidence of internal enhancement. On steady-state free precession (SSFP) cine sequences (not shown) there was etching at the border of the mass consistent with fat/water cancellation artifact. Additionally, there was no contrast enhancement. Based on the classic dumbbell-shaped appearance of the mass, the CT density of LHIS is −57 HU, which is consistent with fat (−10 to −100 HU). On MRI, LHIS has a signal intensity that generally follows the epicardial fat signal (although LHIS may have a lower signal on SSFP imaging, possibly because brown fat has a longer T1), and there is no internal or contrast enhancement. There is characteristic etching of the border as a result of the fat/water cancellation artifact. Additionally, MRI with spin-echo imaging can confirm that a mass is composed of benign fat because with inversion-recovery fat suppression, a mass composed of fat tissue will almost disappear. Thus, although this subject underwent different imaging modalities for further evaluation of the intracardiac mass, either the transesophageal echocardiography or MRI should be sufficient for making a diagnosis of LHIS, based on the classic dumbbell-shaped appearance of the mass. MRI, however, provides additional information regarding the tissue composition of intracardiac masses that transesophageal echocardiography does not; MRI can more conclusively determine whether a mass is composed of fat, regardless of whether the mass is caused by classically appearing LHIS or a lipoma, which does not usually appear dumbbell-shaped. Alternatively, chest CT alone may be used to determine that a mass is composed of fat because of its characteristic low attenuation, similar to subcutaneous fat. Histologically, LHIS appears as nonencapsulated circumscribed fatty tissue that can extend in continuity with epicardial fat. In contrast to a lipoma, LHIS lacks encapsulation and is composed of both white fat cells and brown fat cells, interspersed with hypertrophied myocardial cells, fibrosis, and occasionally chronic inflammatory cells.

Brown fat, which can be a constituent of LHIS, is the likely reason for the increased radiotracer uptake in the interatrial septum during PET imaging in the case presented. Brown fat is metabolically very active because it is involved in adrenergic thermogenesis, via brown fat–specific uncoupling protein-1. Although brown fat was...
once thought to only occur in humans when they are infants and to be lost postnatally, brown fat has recently been recognized to also occur in the upper body (and not only in LHIS) in a subset of adult humans. In an era when the use of PET/CT in oncology for diagnosis, staging, restaging, and tumor monitoring continues to proliferate, LHIS and other upper body locations of brown fat (eg, symmetrically distributed: supraclavicular, neck, mediastinal, para-aortic, and suprarenal regions) may increasingly be mistaken for metastasis. Interestingly, because brown fat is thermogenic (and so its metabolic function is increased in cold temperatures), and because it is stimulated by sympathetic drive, PET imaging at warm temperatures or after β-adrenergic blockade can decrease brown fat’s uptake of FDG to the point that it is invisible on PET images under these conditions.

Important for prognosis and the avoidance of inappropriate therapeutic interventions/strategies are (1) awareness of the potential for brown fat to exist in adult humans and for FDG-PET scans to demonstrate it, (2) recognition of the distinctive appearance of LHIS and the typical symmetrical distribution of brown fat, and (3) recognition of the need to
use techniques aimed at both metabolic and structural imaging to correctly distinguish between brown fat and metastases.

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References
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