Coexistence of atrial myxoma and lung cancer on fluorodeoxyglucose positron emission tomography/computed tomography: The impact of distinct fluorodeoxyglucose uptake pattern on differential diagnosis

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ABSTRACT

The information regarding fluorodeoxyglucose (FDG) uptake in benign and malignant cardiac tumors is limited in the literature and most of the currrently available data were derived from single case reports. Herein we reported coexistence of atrial myxoma and lung cancer on FDG positron emission tomography/computed tomography with the aim of emphasizing the importance of distinct FDG uptake pattern on differential diagnosis.

Keywords: Fluorodeoxyglucose, lung cancer, myxoma, positron emission tomography/computed tomography

A 64-year-old male patient, who experienced acute myocardial infarction 2 days ago, underwent transthoracic echocardiogram to evaluate post-infarction left ventricular systolic function, intracavitary thrombus formation, etc., Besides regional wall motion abnormality, it showed a mass lesion in the left atrium that prolapsed to the base of the left ventricle suggestive of myxoma and accompanying mild mitral regurgitation (not shown). In order to further defining the characteristics of that lesion, intravenous-contrast enhanced thorax computed tomography (CT) was performed. It revealed an intracavitary, heterogeneous, hypoattenuated mass in the left atrium and a nodular lesion (3 cm in diameter) in the left lung upper lobe apicoposterior segment (not shown). Transthoracic fine needle aspiration biopsy of the lung lesion was reported as non-small cell lung cancer. The patient was referred to fluorodeoxyglucose (FDG) positron emission tomography/CT (PET/CT) for initial staging. Although intense hypermetabolism was seen in the lung nodule (standardized uptake value [SUV] max = 10.3) (Figure 1a: Maximum intensity projection image, b: Axial PET, CT and fusion PET/CT images from top to bottom), the lesion in the left atrium showed only mild to moderate heterogeneous FDG uptake (SUVmax = 4.2) (Figure 1c). Mild hypermetabolic lymph nodes (SUVmax = 3.5) were also detected in the mediastinum (Figure 1d). Mediastinoscopic sampling of these nodes was reported as reactive lymphoid hyperplasia. Since the resection of the atrial lesion was important in terms of both treatment option of the lung cancer and preventing possible complications it was surgically removed. The pathology supported our initial diagnosis of myxoma. Then the patient underwent left upper lobectomy. Primary cardiac tumors are very rare with an incidence of less than 0.1% in autopsy series.[1] Over 75% of these tumors are benign.[1] Myxomas are the most common benign primary cardiac neoplasm in adults. Metastatic involvement of the heart is encountered up to 20-40 fold more frequently than primary tumors.[2] The most common secondary tumors of the heart include malignant melanoma, lung cancer, breast cancer, soft tissue sarcoma, renal cell carcinoma, esophageal cancer, hepatocellular cancer and thyroid cancer.[3] The estimated metastasis rate of lung cancer to the heart is nearly 30%.[2] The information regarding FDG uptake in benign and malignant cardiac tumors is limited in the literature and most of the currently available data were derived from a single case reports.[4-6] Although surgical excision of the lesion is required for the

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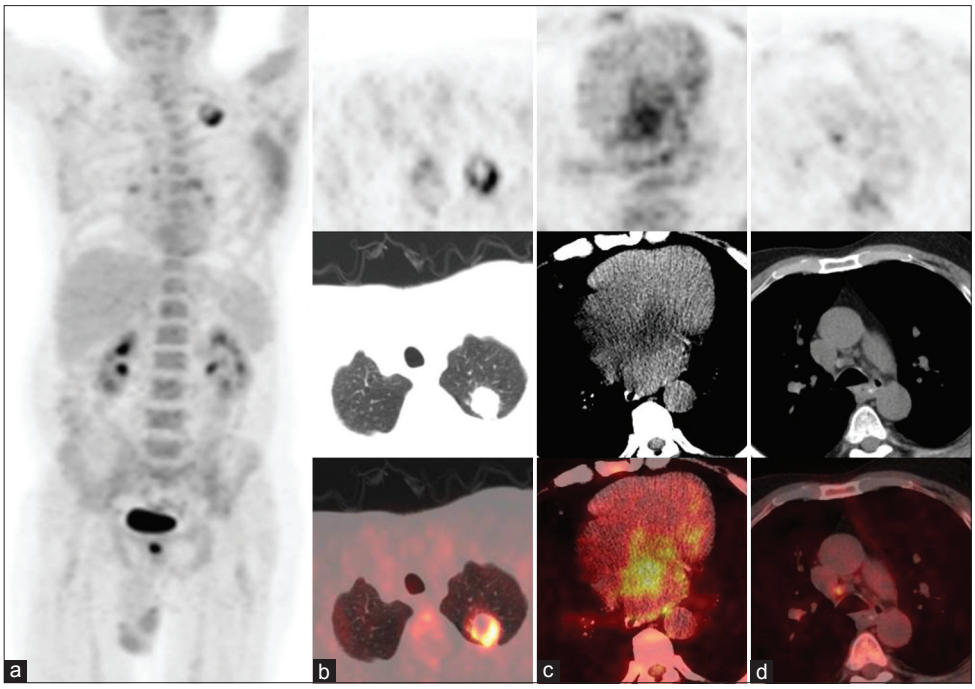

Figure 1: Although intense hypermetabolism was seen in the lung nodule (standardized uptake value [SUV]max = 10.3) (a: Maximum intensity projection image, b: axial PET, CT and fusion PET/CT images from top to bottom), the lesion in the left atrium showed only mild to moderate heterogeneous FDG uptake (SUVmax = 4.2) (c). Mild hypermetabolic lymph nodes (SUVmax = 3.5) were also detected in the mediastinum (d)

definitive diagnosis of the cardiac tumors, it was reported that FDG PET/CT can aid in benign and malignant differentiation [Figure 1b] with malignant tumors (both primary and secondary) had significantly higher SUVmax than benign ones as this was the case in our patient.[1] The increased glucose transporter expression, hexokinase activity, tumor perfusion and viable cancer cell number may be responsible for this differential FDG uptake.

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