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Case Report

Occasionally increased ^{18}F-fluorodeoxyglucose uptake in apical hypertrophic cardiomyopathy with mid-ventricular obstruction

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ABSTRACT

We report on a 77-year-old man who was diagnosed with apical hypertrophic cardiomyopathy (HCM) with mid-ventricular obstruction. He had a cured hepatitis C infection. We detected occasionally increased ^{18}F-fluorodeoxyglucose (^{18}F-FDG) uptake on whole-body positron emission tomography (PET)/computed tomography (CT) performed to examine the involvement of bladder carcinoma. ^{18}F-FDG-PET was restudied following specific preparation involving an 18-h low-carbohydrate diet and following 15-h fasting plus heparin pre-administration. Increased uptake of ^{18}F-FDG was observed reproducibly in the hypertrophic apical to mid left ventricular myocardium, with a maximum standardized uptake value of 6.2. In contrast, relatively lower ^{18}F-FDG-uptake areas tended to match areas of late gadolinium enhancement on cardiac magnetic resonance (CMR). Histopathological examination of myocardial biopsy showed disarried hypertrophic myocytes with cellular infiltration. Increased uptake of ^{18}F-FDG may reflect the phenomenon of increased glucose utilization in hypertrophied myocardium. The increasing clinical utility of whole-body PET/CT for evaluating malignancies may increase the detection of occasional abnormal ^{18}F-FDG uptake in the heart. It is necessary to clarify that ^{18}F-FDG myocardial PET in combination with CMR may provide a more detailed risk assessment in patients with HCM.
<**Learning objective:** Increased ^{18}F-fluorodeoxyglucose (^{18}F-FDG) uptake was occasionally found in the apex of the heart on whole-body positron emission tomography performed to evaluate bladder cancer involvement. Increased uptake of ^{18}F-FDG was observed in the hypertrophic apical to mid left ventricular myocardium, while relatively lower ^{18}F-FDG-uptake areas tended to match areas of late gadolinium enhancement.>

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Introduction

^{18}F-Fluorodeoxyglucose (^{18}F-FDG)-positron emission tomography (PET) is used clinically to diagnose the systemic involvement of malignancies or inflammatory diseases by virtue of their ability to metabolize glucose [1]. Increased uptake of ^{18}F-FDG in the myocardium may indicate the presence of cardiac tumors or inflammatory diseases, such as cardiac sarcoidosis [2]. Herein, we report a patient with apical hypertrophic cardiomyopathy (HCM) with mid-ventricular obstruction, with occasionally increased ^{18}F-FDG uptake in the apex of the heart on whole-body PET/computed tomography (CT).

Case report

A 77-year-old man had been diagnosed with apical to mid-ventricular-type HCM at Hiroshima University Hospital. He had experienced dyspnea with chest discomfort since his 60s. This case was complicated by interstitial pneumonitis and a history of cerebral infarction due to paroxysmal atrial fibrillation. He had a history of hepatitis C viral infection but the viral antigen disappeared after antiviral agents. He had no remarkable family history. His blood pressure was 116/70 mmHg and his pulse was regular at 60 bpm. Auscultation revealed systolic ejection murmur

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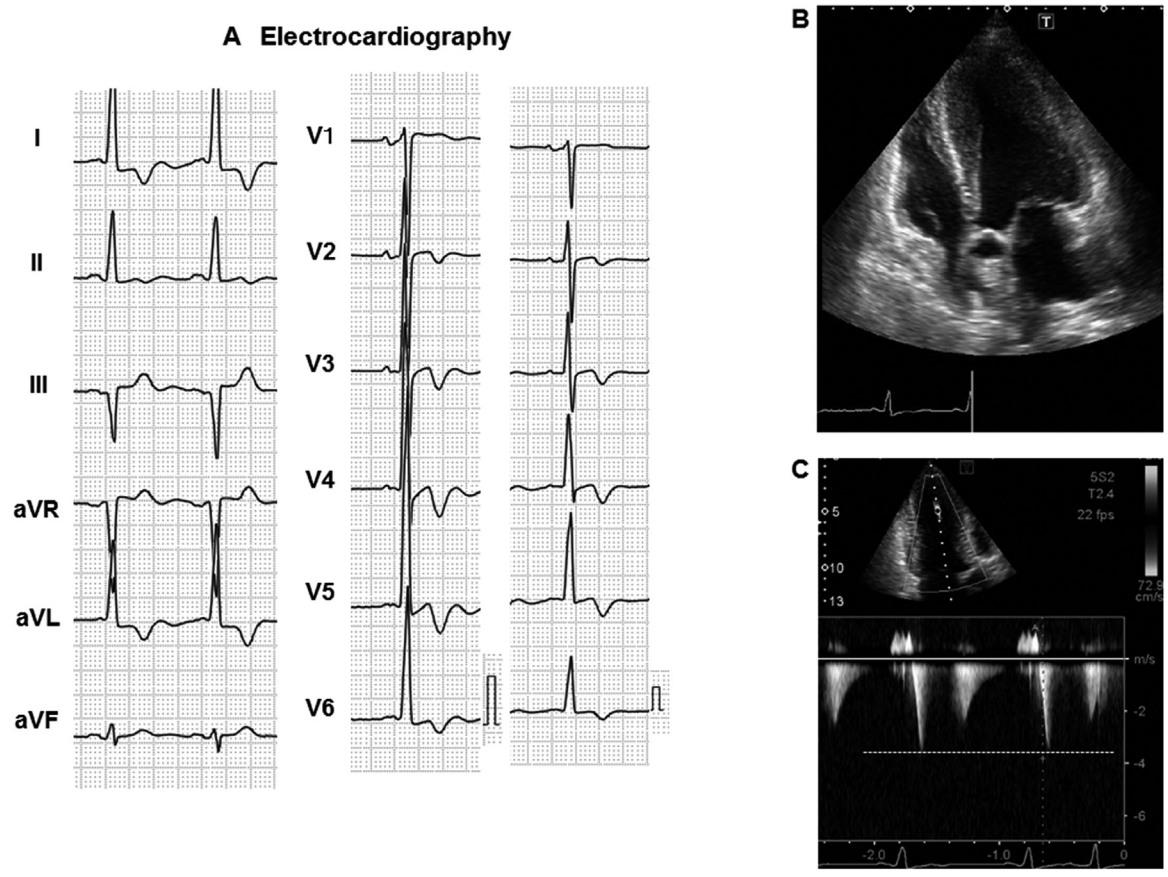

Fig. 1. Electrocardiography (A) showed deep negative T waves in I, aV_L, and precordial leads (V_{2-6}) (7 mm depth in V_5). Echocardiography (B) showed marked mid to apical left ventricular hypertrophy (maximum thickness 19 mm) with a left ventricular ejection fraction of 71%. Continuous Doppler examination showed accelerated late systolic peak flow (3.8 m/s) in the left ventricular cavity of the mid-obliterans portion during the Valsalva maneuver (C).

(Levine II/VI) at the apex area, and fine crackle in bilateral lung fields.

Electrocardiography showed deep negative T waves in I, aV_L, and V_{2-6} leads (7 mm depth in V_5), while Doppler echocardiography showed marked left ventricular hypertrophy in the mid to apical portion, and accelerated late systolic peak flow (3.8 m/s) in the left ventricular cavity of the mid-obliterans portion during the Valsalva maneuver (Fig. 1). His serum creatinine kinase-MB level was slightly elevated (9 U/L) and plasma N-terminal B-type natriuretic peptide level was elevated (1,074 pg/mL), but serum C-reactive protein and lysozyme levels were normal. Cine cardiac magnetic resonance (CMR) performed in June 2016 revealed normal myocardial blood flow and a patchy pattern of late gadolinium enhancement in the inner layer of the mid to apical hypertrophic myocardium. There was no obvious high-intensity signal in the T2-weighted image. He received bisoprolol 2.5 mg/day, cibenzoline 300 mg/day, and apixaban 10 mg/day.

The patient visited a urological clinic in October 2016 because of hematuria. He was diagnosed with bladder carcinoma and scheduled to receive surgical treatment. Prior to surgery, he underwent whole-body ^{18}F-FDG-PET/CT at Chugoku Electronic Hospital to evaluate the involvement of the bladder carcinoma. PET/CT was performed following the usual 12-h fasting preparation. A marked increase in ^{18}F-FDG uptake was occasionally found in the apex of the heart, but no other abnormal uptake was detected (Fig. 2A and B).

^{18}F-FDG-PET was repeated at Hiroshima University Hospital after resection of the bladder cancer, following specific preparation involving an 18-h low-carbohydrate diet and following 15-h fasting plus heparin pre-administration 15 min prior to ^{18}F-FDG injection (50 IU/kg) [3]. The CMR and ^{18}F-FDG/CT images are compared in Fig. 2C–H. Increased uptake of ^{18}F-FDG was observed reproducibly in the hypertrophic apical left ventricular myocardium, with a maximum standardized uptake value of 6.2. In contrast, relatively lower ^{18}F-FDG-uptake areas tended to match the areas with late gadolinium enhancement. Histopathological examination of the myocardial biopsy obtained from the endocardium of the right ventricular apex showed disarray of hypertrophic myocytes with mononuclear cell aggregation and mild fibrosis, but no signs of granuloma or malignancies (Fig. 3).

Discussion

In the present case with apical HCM with mid-ventricular obstruction, increased ^{18}F-FDG uptake was occasionally found in the apex of the heart on whole-body PET/CT performed to evaluate bladder cancer involvement.

Abnormal ^{18}F-FDG uptake in HCM has been sporadically reported, and they speculated that the increased uptake of ^{18}F-FDG in HCM is caused by increased glucose utilization, complementary to impaired fatty acid metabolism [4,5]. In addition, Takeishi et al. demonstrated increased uptake of ^{18}F-FDG and relatively low uptake in the late gadolinium enhancement area in the apex [5]. However, the current patient showed mild to moderate mononuclear cell aggregation in the myocardial biopsy. Then, increased uptake of ^{18}F-FDG may reflect the phenomenon that inflammation caused the increased glucose utilization in hypertrophied myocardium. In contrast, a previous study reported

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