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SHORT COMMUNICATION

Incidental focal FDG uptake in heart is a lighthouse for considering cardiac screening

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Abstract
Objectives Cardiac FDG uptake is known to show a variety of patterns under clinical fasting conditions. We hypothesized that focal FDG uptake in the heart (FUH) represents a sign of cardiac disease risk, especially in coronary artery disease (CAD).The aim of this study was to clarify the relationship between FUH and cardiac disease.
Methods Cases showing FUH were selected based on comments in diagnostic reports or identification on retrospective review. Quantitative analysis was performed using maximum standardized uptake value (SUV_{max}), with regions of interest drawn over focal uptake areas in the heart as confirmed by PET/CT and in lateral side of the same slice showing focal FDG uptake.
Results For the 20 patients (11 men, 9 women) with confirmed FUH, coronary artery stenosis or history of treatment for coronary disease was present in 11 patients (55.0 %), and 2 patients showed apical hypertrophy. Mean SUV_{max} of FUH did not differ significantly between patients with confirmed cardiac disease and those with no evidence of cardiac disease ($P = 0.78$).
Conclusions FUH suggests a high likelihood of CAD in patients without myocardial symptoms. Cardiac screening or a check of the history of cardiac disease is thus worth considering when FUH is seen incidentally on FDG-PET/CT.

Keywords FDG · PET/CT · Heart · Focal cardiac uptake · Apical hypertrophy

Introduction

2-Fluoro [^{18}F]-2-deoxy-D-glucose positron emission tomography (FDG-PET) is widely used for assessing myocardial viability and evaluating patient prognosis [1, 2]. The glucose–insulin loading method has been considered preferable to optimize FDG uptake in viable myocardium [3]. In addition, administration of heparin, increasing plasma levels of free fatty acids and indirectly stimulating myocardial utilization of fatty acids are available for evaluating heart disease by reducing physiological FDG uptake in the heart [4, 5]. After a sufficient period of fasting, the myocardial metabolism predominantly uses fatty acids as a source of energy [6], so myocardial FDG uptake will remain as low as blood pool tracer activity [3]. However, under the conditions of clinical fasting required for assessment of non-myocardial lesions, distributions remain variable, with reduced associations with age, blood glucose level and duration of fasting [7].

In FDG-PET/CT examinations under clinical fasting conditions aimed at the evaluation of non-myocardial disease, focal FDG uptake in the heart (FUH) is sometimes encountered. Such FDG uptake is usually focal, showing no continuity with FDG uptake at the base of the heart, and is well recognized from the apex to the anterior wall. We hypothesized that FUH represents a sign of cardiac disease risk,

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especially in coronary artery disease (CAD). Cardiac events represent a crucial side effect of several cancer therapies [8, 9], and myocardial disease greatly increases the risks associated with surgery [10, 11]. The prediction of myocardial disease based on PET/CT may thus help reduce the risk of cardiac events. In this study, we retrospectively surveyed the possibility of CAD in patients showing incidental FUH.

Materials and methods

Patients

All protocols in this retrospective observation study were approved by our institutional review board. Patients showing FUH were selected based on comments from diagnostic reports or on retrospective reviews for the existence of these findings. PET/CT images were interpreted as consensus decisions of at least two physicians specializing in nuclear medicine. All FDG-PET/CT scans performed with the specific intention of assessing cardiac disease were excluded from this study. In addition, no history of cardiac disease was provided at the time of diagnosis on FDG-PET/CT, so all cases of FUH were found incidentally. Occurrence of adverse cardiac events was assessed based on the history of cardiac events and/or the results of consultation with a cardiologist.

PET/CT

An in-house cyclotron and automated synthesis system (F100; Sumitomo Heavy Industry, Tokyo, Japan) was used in accordance with the authorized procedure to synthesize FDG. All subjects fasted for 5 h before measuring blood glucose levels and receiving intravenous injection of 370 MBq of FDG. PET/CT images were obtained using two PET/CT systems (Biograph 16; Siemens, Germany, and Discovery PET/CT 600, GE Healthcare, USA), measuring from the vertex to the mid-thigh or knee joint 60 min after intravenous injection of FDG. Low-dose CT was performed first and used for attenuation correction and image fusion. Emission images were acquired in 3-dimensional mode for 2 min per bed position in the Biograph 16 system and for 3 min per bed position in the Discovery PET/CT 600 system. PET data were reconstructed using a Gaussian filter with an ordered-subset expectation maximization algorithm (3 iterations, 8 subsets for Biograph 16, and 3 iterations, 16 subsets for Discovery PET/CT 600).

Data interpretation and image analysis

FUH was interpreted by maximum intensity projection (MIP), axial, sagittal and coronal PET images, and the location of FDG uptake in the myocardium was determined by referring to the CT portion of PET/CT images. FUH was defined as focal FDG uptake that was discontinuous with FDG uptake in the basal area of heart. Quantitative analysis was performed using maximum standardized uptake value (SUV_{max}), with regions of interest (ROIs) drawn over focal uptake areas in the heart as confirmed by PET/CT images. To evaluate the contrast of FUH, reference myocardial FDG uptake was put on the far lateral side from the same slice showing FUH. In addition, the existence of left coronary artery calcification was retrospectively interpreted by a physician specializing in nuclear medicine based on CT images obtained from PET/CT examination.

Further evaluations for cardiac disease were based on decisions made by the attending physician after consulting with a cardiologist. The results of electrocardiography (ECG), cardiac ultrasonography (US), coronary computed tomography angiography (CTA), and coronary angiography (CAG) were diagnosed by a cardiologist and the results of myocardial scintigraphy (Tc99m-tetrofosmin stress myocardial scintigraphy), ^{201}Tl and ^{123}I-β-methyl-*p*-iodophenyl pentadecanoic acid (BMIPP) myocardial scintigraphy were diagnosed by a physician specializing in nuclear medicine. The final diagnosis of CAD was based on the results of CAG with significant (≥50 %) coronary artery stenosis. If coronary CT showed no evidence of coronary artery stenosis, the case was regarded as negative for CAD. Even if CAG or coronary CT was not performed around the time of the FDG-PET/CT test, cases with post-treatment CAD were regarded as CAD-positive.

Statistical analysis

Significant differences in FDG accumulation between the area of focal FDG uptake and lateral wall were compared between patients with and without cardiac disease using the Mann–Whitney *U* test. Values of $P < 0.05$ were considered statistically significant.

Results

Patient characteristics

Of 9,289 FDG-PET/CT scans performed for 5,248 patients between January 2006 and March 2012, cardiac scintigraphy and CAG had been conducted for 194 (3.7 %) and 141 (2.7 %) patients, respectively, due to suspected myocardial ischemia (MI). Of these FDG-PET/CT scans, 30 (18 men, 12 women) cases showed FUH on FDG-PET/CT. At the time of the FDG-PET/CT study, none of the patients had symptoms of angina and consult cardiologist with cardiac awareness. Ten patients did not undergo any further

examinations except for a single ECG which had no abnormal findings. Finally, 20 patients (11 men, 9 women, mean age 75.4 years, range 53–85 years) were analyzed in this retrospective preliminary study. Patient characteristics are shown in Table 1. FDG-PET/CT was performed for staging or restaging of cancer in 10 cases, differentiation between malignancy and non-malignancy in 5 cases, screening of a lesion associated with fever of unknown origin (FUO) in 4 cases and screening of cancer on suspicion of paraneoplastic syndrome in 1 case. The 4 patients with FUO finally diagnosed as scleroderma, pneumonia, Churg–Strauss syndrome and discitis, respectively.

The median follow-up period was 10 months (range 4–36 months) for 5 patients with history of treatment of CAD, and 26 months (range 4–49 months) for 8 patients with no evidence of CAD. No adverse cardiac events occurred in these 15 patients after FDG-PET/CT scan showing FUH. Of the 10 patients performed CAG, 2 patients underwent emergency CAG due to angina attack; the other patients had no cardiac symptoms before CAG.

The final diagnosis of CAD was diagnosed by CAG in 10 patients, history of CAD in 7 patients and CT angiography in 2 patients. CAD was suspected based on ECG in 8 patients, and 2 of these 8 patients were eventually diagnosed with CAD. Two patients were decided not to have CAD based on the results of ECG and US, with no evidence of cardiac disease. Two patients were diagnosed with AHCM by US. CAD was suspected based on ECG in 8 patients, and 2 of these 8 patients were eventually diagnosed with CAD. Heart wall asynergy was identified on US in 3 patients, all of whom were diagnosed with CAD following CAG. Positive findings with tetrofosmin SPECT were obtained in 4 of 5 patients, and 4 patients were diagnosed with CAD. Imaging mismatch between ^{201}Tl

Table 1 Patients characteristics

No.	Age	Sex	BG	Complication	Object for FDG-PET/CT examination	FDG uptake area	FDG uptake value		Calcification at coronary artery	Final diagnosis
							FUH-SUV_{max}	L-SUV_{max}		
1	77	F	99	None	FUO	Apex	4.40	2.23	+	AHCM
2	64	M	142	DM, HT, HL	Lung cancer suspected	Ant	2.42	1.74	+	CAD
3	78	F	178	DM, HT	Lung cancer suspected	Ant-apex	4.07	1.88	+	CAD
4	66	M	107	HT, HL	Restaging of lung cancer	Apex	3.21	2.22	+	CAD
5	80	F	99	None	FUO	Ant-apex	3.20	1.72	−	Normal
6	81	F	93	DM, HT	Unexplained lymphnode swelling	Ant	2.79	1.87	+	CAD
7	81	M	105	DM, HT	Gastric carcinoma staging	Apex	3.64	2.11	−	Normal
8	68	F	289	DM, HL, HT	FUO	Ant	4.30	2.95	+	CAD
9	76	F	88	HT	FUO	Apex	3.64	2.02	−	Normal
10	82	M	129	DM	Lung cancer staging	Apex	3.14	1.62	+	Normal
11	73	M	147	DM, HT	Esophageal cancer staging	Apex	3.60	1.36	+	Normal
12	73	M	112	DM, HL, HT	Restaging for HCC	Apex	6.61	1.69	+	CAD
13	71	M	154	DM, HT, HL	Malignancy unknown origin	Apex	4.46	2.43	−	CAD
14	53	M	138	DM	Multiple myeloma staging	Ant	3.47	2.19	+	CAD
15	82	F	99	HT	Malignancy unknown origin	Apex	5.77	1.94	−	Normal
16	85	M	99	None	Lung cancer staging	Apex	5.82	3.40	−	CAD
17	83	F	98	None	Lung cancer staging	Apex	3.91	2.99	−	Normal
18	77	F	102	DM	Paraneoplastic syndrome suspected	Apex	1.61	1.30	+	CAD
19	84	M	87	HT	Rectal cancer staging	Ant-apex	5.35	2.07	+	AHCM
20	73	M	97	HT, HL	Restaging for malignant lymphoma	Apex	2.80	1.51	+	CAD

BG blood glucose level, *FUH-SUV_{max}* maximum SUV of focal FDG uptake area, *L-SUV_{max}* maximum SUV for lateral area of FUH, *M* male, *F* female, *DM* diabetes mellitus, *HT* hyper tension, *HL* hyper lipidemia, *FUO* fever unknown origin, *HCC* hepatocellular carcinoma, *ant* anterior wall, *FUH* focal heart uptake, *AHCM* apical hypertrophic cardiomyopathy, *OMI* old myocardial infarction, *CABG* coronary artery bypass graft, *CAD* coronary artery disease

Table 2 Findings in cardiac screening

No.	Final diagnosis	Findings				
		ECG	US	Cardiac scintigraphy	CAG[a] or Coronary CT	Others
1	AHCM	–	Apical hypertrophy (19 mm)	Reduction of ^{123}I-BMIPP uptake relative to ^{201}Tl uptake at apex	CAG: negative (2 years before)	–
2	CAD	Negative T wave in lead V1–4	–	Perfusion defect at anterior wall by tetrofosmin SPECT with exercise	–	Post CABG for LAD (#6: 100 %)
3	CAD	Negative	–	–	–	Post coronary stenting #6–7: 75 %, #11: 90 %
4	CAD	Atrial fibrillation	–	Perfusion defect at antero-lateral wall by tetrofosmin SPECT with exercise	–	Post coronary stenting #6–8: 75 %, #12–14: 90 %
5	Normal	ST depression in lead I and aVL	Normal	–	–	–
6	CAD	Negative	Apical asynergy	Perfusion defect at anterior wall by tetrofosmin SPECT with exercise	CAG: #9: 90 % stenosis	Post coronary stenting #8: 90 %
7	Normal	ST segment depression in lead I, II, III, aVL, aVF, V5–6	Mild left ventricular hypertrophy	–	CAG: negative	–
8	CAD	Negative	Septal to anteroseptal asynergy	–	CAG : #7: 99 %, #14: 90 % stenosis	–
9	Normal	CRBBB	–	–	Coronary CT: negative	Disappear of FDG uptake in second PET examination
10	Normal	ST segment depression in lead V4–5 by master exercise test	Normal	Normal perfusion by adenosine stress perfusion imaging	–	–
11	Normal	ST segment depression in lead I, aVL, V4–6	Normal		Normal	–
12	CAD	Negative in several time screening	–	–	–	Post CABG for RCA (#1: 50 %) and LCA (#6–8: 90 %)
13	CAD	Af	Normal	–	CAG: #9: 75 %	–
14	CAD	Negative	Normal	Perfusion defect at anterior wall by tetrofosmin SPECT with exercise	CAG: #6: 50 %, #9: 100 %, intermediate artery 90 % stenosis	–
15	Normal	ST segment depression in lead I, aVL V5	Mild concentric hypertrophy	Reduction of ^{123}I-BMIPP uptake relative to ^{201}Tl uptake at anterior wall	Coronary CT: negative	–
16	CAD	Negative	Normal	–	CAG: #2: 90 %,#5: 75 %, #7: 99 % stenosis	–
17	Normal	Negative	Normal	–	CAG: negative	Disappear of FDG uptake in second PET examination
18	CAD	ST segment elevated in lead V1–4	–	Both ^{201}Tl, ^{123}I-BMIPP uptake decreased in anterior wall to apex	CAG: Stenosis at proximal and distal of stent (#6–7)	Post coronary stenting (#6–7: 75 %)
19	AHCM	ST segment depression in lead V4–6	Apical wall hypertrophy (18 mm)	–	–	–
20	CAD	Negative	Septal to anterowall and apex asynergy	–	CAG: #2: 90 %, #6: 75 %, #15: 75 %, stenosis	Post coronary stenting (#8: 99 %)

ECG electrocardiogram, *US* ultrasonography, *CAG* coronary angiography, *CT* computed tomography, *CAD* coronary artery disease, *CRBBB* complete right bundle branch block, *^{123}I-BMIPP* ^{123}I-β-methyl-p-iodophenyl pentadecanoic acid, *CABG* coronary artery bypass graft

[a] The represented segmental location of coronary stenosis was based on the 15-segment American Heart Association coronary artery model

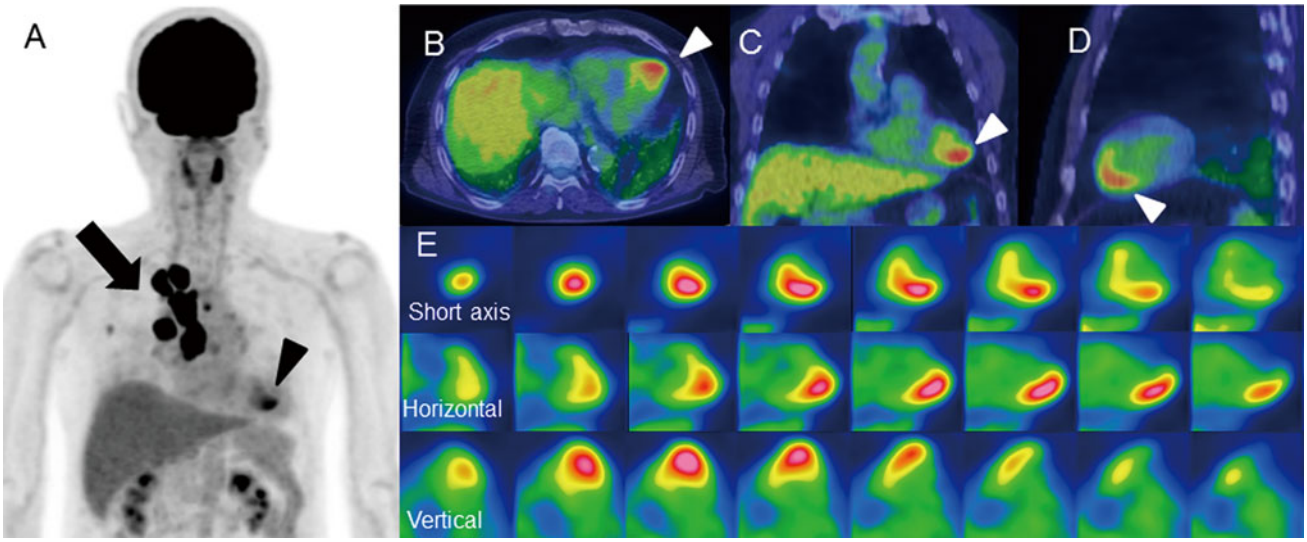

Fig. 1 This figure shows the patient no. 16, who was pointed out focal FDG uptake at anterior wall to apex in FDG-PET/CT scan performed for lung cancer staging. **a** The maximum intensity projection images shows FDG avid mediastina lesion (*arrow*) and focal FDG uptake in heart (FUH) (*arrow head*). **b** Axial image of fused PET/CT, **c** coronal image of fused PET/CT, **d** sagittal image of fused PET/CT, **e** PET image reconstructed for cardiac view

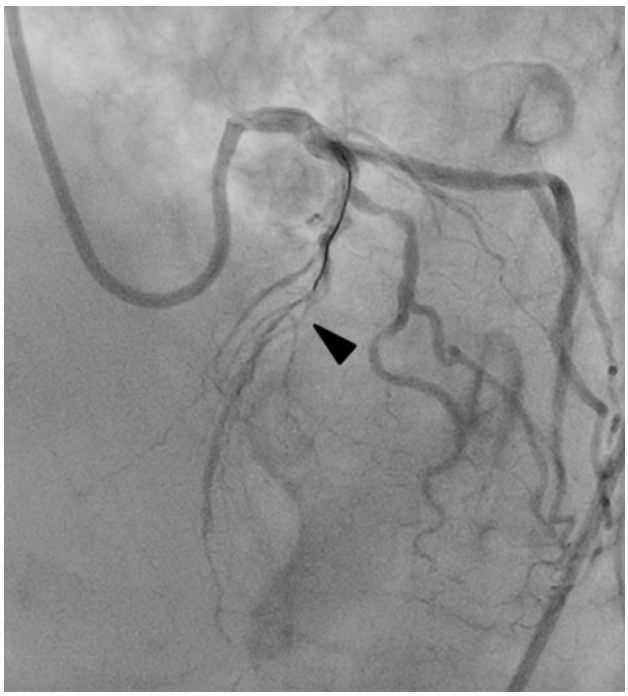

Fig. 2 Left coronary angiogram confirms the flow reduction in LAD due to coronary artery stenosis

and ^{123}I-BMIPP myocardial scintigraphy was confirmed in 3 patients, and just 1 patient had CAD.

Concomitant conditions included diabetes mellitus (DM) in 11 patients (55.5 %), hypertension in 13 patients (65.0 %), hyperlipidemia in 6 patients (30.0 %) and calcification of coronary arteries in 12 patients (60.0 %). CAD was confirmed in 8 patients (72.7 %) with DM, 8 patients (61.5 %) with hypertension, 6 patients (100.0 %) with hyperlipidemia and 9 patients (75.0 %) with calcification of coronary arteries.

Mean blood glucose level in all patients just before FDG injection was 123.1 mg/dl (range 87–289 mg/dl). DM was well controlled by medication in 10 patients, but blood glucose level was unstable in one patient (No. 8) on dialysis. For the 20 patients with FUH, coronary artery stenosis or history of treatment for coronary disease was confirmed in 11 patients (55.5 %), and 2 patients showed apical hypertrophic cardiomyopathy (AHCM). The results of several examinations are shown in Table 2. Representative images of FUH are shown in Figs. 1, 2, 3, 4 and 5.

Mean SUV_{max} for FUH area showed no significant differences between patients with and without evidence of cardiac disease (SUV_{max}, 3.95 vs. 3.84, respectively; $P = 0.78$). The ratio between FUH area and FDG uptake around FUH likewise showed no significant difference between parents with confirmed cardiac disease and those with no evidence of cardiac disease (1.90 vs. 2.04, respectively; $P = 0.26$).

Discussion

The present study showed that incidental findings of FUH of non-cardiology PET studies were associated with a high risk of CAD. Cardiac screening or a check of the history of cardiac disease thus appears to be warranted when FUH is incidentally found on FDG-PET/CT.

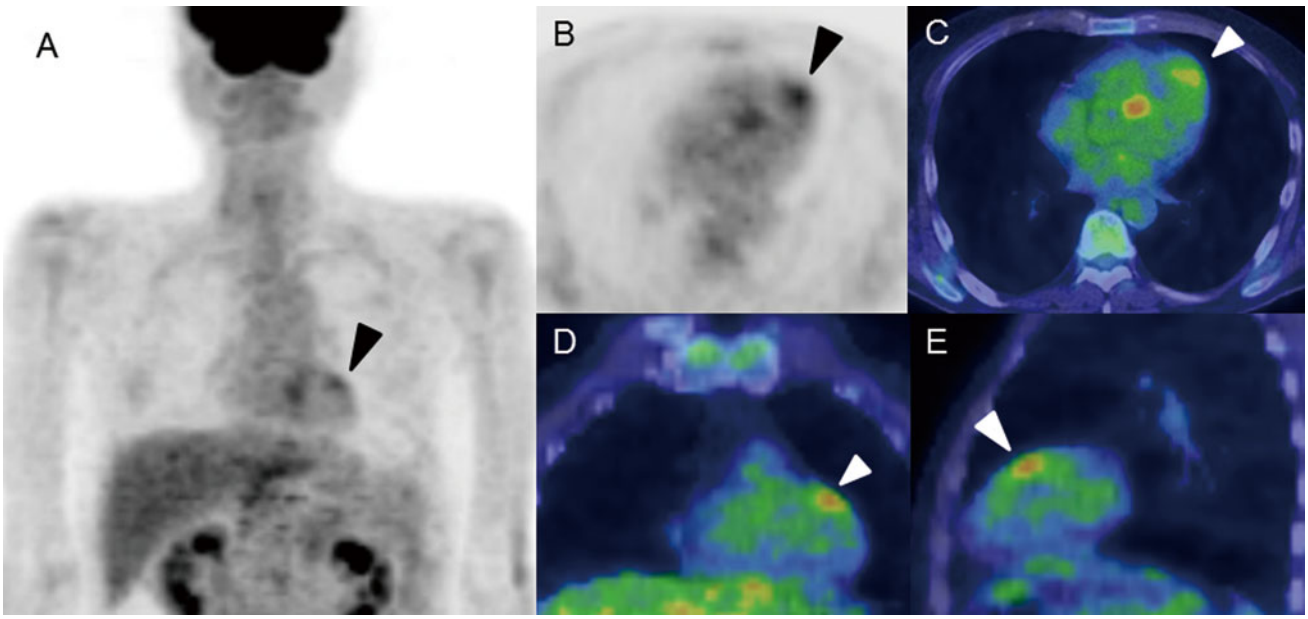

Fig. 3 This figure shows the patient no. 14, who was confirmed focal FDG uptake at anterior wall (*arrow head*) in FDG-PET/CT scan performed for staging of multiple myeloma. **a** The maximum intensity projection PET images, **b** axial image of fused PET/CT, **c** coronal image of fused PET/CT, **d** sagittal image of fused PET/CT

Several patterns of FDG uptake in the heart specific to cardiac disease have been reported. Atrial fibrillation shows as increased uptake in the enlarged right atrium [12], complete left bundle branch block as increased uptake in the lateral wall of the left ventricular and a defect in the septum [13], pulmonary hypertension as apparent FDG uptake in the enlarged right ventricular wall [14] and hypertrophic cardiomyopathy as increased FDG uptake in hypertrophic areas [15]. Regional glucose utilization in myocardium is homogeneously decreased in patients with stable angina examined at rest compared to healthy subjects [16]. In contrast, myocardial glucose utilization is increased in patients with severe myocardial reperfusion injury, unstable angina or ischemic cardiomyopathy. This is because the myocardium shifts from fatty acid oxidation to glucose oxidation [17]. The regional mismatch in myocardial perfusion and metabolism images on perfusion-weighted FDG-PET testing has been regarded as viable myocardium showing preservation of myocardial glucose metabolism, particularly in the presence of resting hypoperfusion [18–20]. A persistent metabolic switch from fatty acids to glucose provides the potential for diagnosing MI in the acute-care setting, and FDG uptake can be regarded as a surrogate marker for antecedent ischemia [21]. Based on the same principles, ^{201}Tl and ^{123}I-BMIPP mismatch is confirmed in ischemic injury persisting in viable myocardial tissues or infarct-related risk areas in patients with preinfarction angina [22].

Metabolic patterns of myocardium basically show large variations by regions and time. According to coronary branch territories, mean myocardial uptake of FDG was higher in LCX territories than in RCA or LAD territories. Similarly, FDG uptake was higher in the lateral wall than in the anterior wall, septum or inferior wall. According to basal–apical gradient analysis, FDG uptake is higher in middle territories than in either proximal or distal areas [23]. FUH was frequently recognized in the apex and/or anterior wall, with these segments tending to show lower physiological FDG uptake than other areas. Therefore, FUH at the anterior wall and apex is quite unlikely to appear as a normal variation in myocardial FDG uptake. However, FUH appears to occur under specific conditions of the myocardium and its surroundings, resulting in a general reduction in FDG uptake by normal myocardium. Therefore, FDG-PET obviously has a limitation for screening all cases suffering from cardiac disease.

Confirmed FUH in patients with no cardiac disease might represent physiological FDG uptake for myocardium, as suggested by 2 cases that showed disappearance of FUH in second PET performed within a relatively short interval. In terms of FDG uptake, no significant differences were confirmed between cases with proven cardiac disease and those with no evidence of cardiac disease. However, patients with FUH frequently showed other risk factors of MI, such as DM, hypertension and calcification of coronary arteries, so further screening for cardiac disease and/or elicitation of past medical history appears warranted if FUH is found in FDG-PET examination. Moreover, cardiac event can hamper ongoing treatment, so the risk of cardiac disease should be avoided before initiating disease

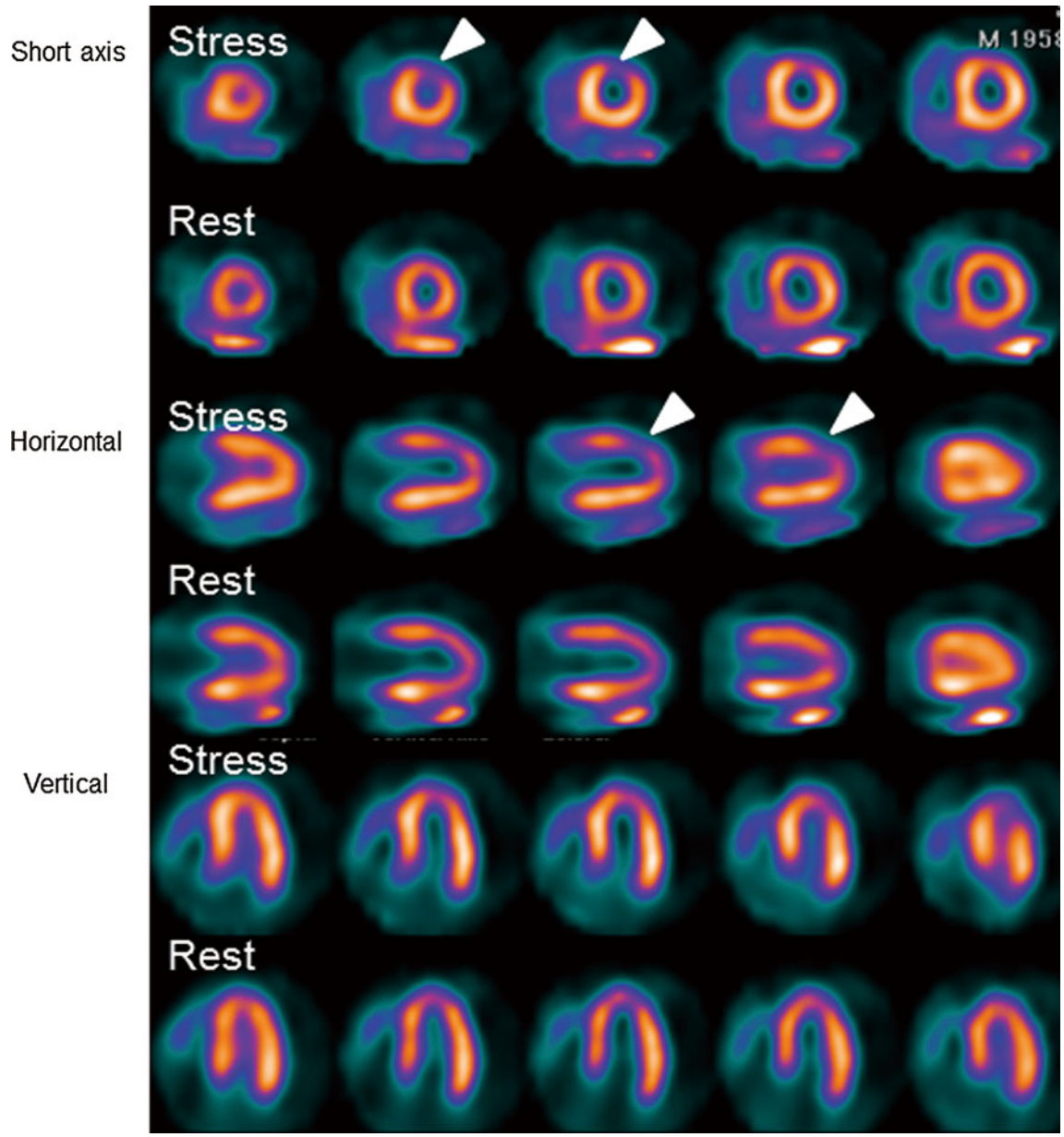

Fig. 4 Tetrofosmin SPECT with exercise shows perfusion defect at middle of anterior wall (*arrow head*)

treatment. The time for screening cardiac disease might be matched to FDG-PET/CT performed for the purpose of selecting the course of treatment.

This study surveyed FUH only in the apex and anterior wall as identified by MIP and/or 3-axis images. We encountered one another case with diffuse FDG uptake at the inferior wall showing decreased uptake in ^{99m}Tc-tetrofosmin stress myocardial scintigraphy. However, evaluation on MIP and axial images to assess FDG uptake of the inferior wall appeared to be difficult because of physiological FDG uptake under the myocardium, such as uptake by the left hepatic lobe, stomach and spleen. More precise information for the localization of FUH may be obtained by reconstruction using advanced cardiac quantification software. This study included the case with wide range of blood glucose before the FDG-PET examination. However, it had no correlation between blood glucose level and SUV_{max} for focal FDG uptake area, and blood glucose level and lateral wall.

The limitations of this retrospective study include the small number of cases, and little evidence for cases diagnosed with no cardiac disease without screening of coronary arteries. In addition, the significance of FUH confirmed in post-treatment cases was not evaluated sufficiently. Further prospective

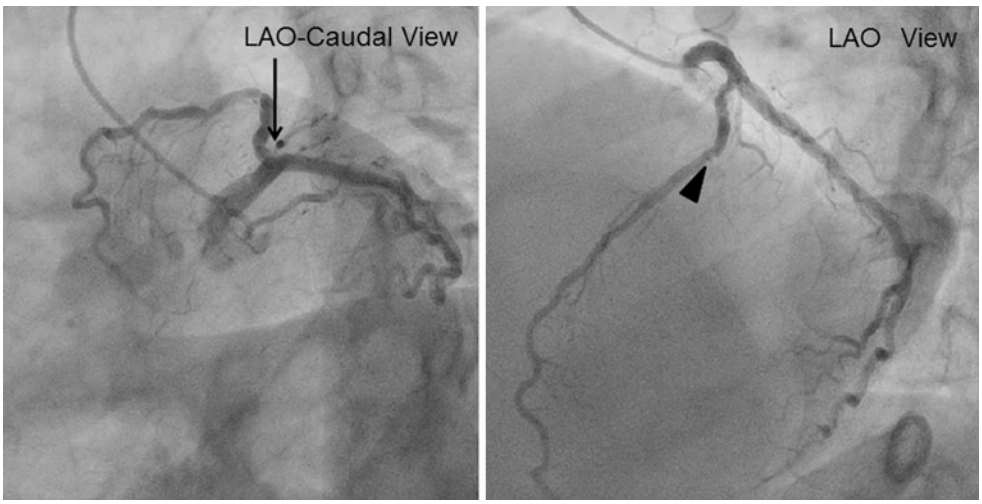

Fig. 5 Left coronary angiogram showing severe stenosis of intermediate artery (*arrow*) and total occlusion of diagonal branch (*arrow head*)

studies are needed to clarify whether our findings are relevant to cardiac disease requiring priority treatment.

Conflict of interest The authors declare no conflict of interest.

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