Predictive value of ^{18}F-FDG PET/CT in patients with acute type B aortic intramural hematoma

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Background. **The clinical course and predictors of adverse aortic events (AAE) in patients with acute Stanford type B intramural hematoma (IMH) remain controversial. This study aimed to investigate whether ^{18}F-FDG PET/CT can predict risk in patients with acute type B IMH.**

Methods and Results. **This study included 34 patients with acute type B IMH who underwent PET/CT within 14 days from the onset of symptoms. The maximal standardized uptake values (SUVmax) of ^{18}F-FDG uptake was significantly different between patients with or without AAE (4.3 ± 0.6 vs 3.7 ± 1.0, $P = 0.020$), but not the target to blood ratio (TBR, SUVmax divided by SUV in the superior vena cava) (1.6 ± 0.2 vs 1.5 ± 0.5, $P = 0.064$). In patients with initial ulcer-like projection (ULP), a blood-filled pouch protruding into the IMH, which was seen in 25 patients(74%), both the SUVmax and TBR were significantly higher in patients who developed AAE, (4.3 ± 0.6 vs 3.3 ± 0.5, $P = 0.001$; 1.6 ± 0.2 vs 1.4 ± 0.2, $P = 0.01$); the TBR >1.5, which is determined from receiver-operating-characteristic curve, had a sensitivity of 73% and a specificity of 80% in predicting AAE.**

Conclusion. **Patients with ULP and high ^{18}F-FDG uptake were more likely to develop AAE and may require closer surveillance with serial imaging. (J Nucl Cardiol 2019;26:633–41.)**

Key Words: Fluorodeoxyglucose (FDG) • PET/CT imaging • peripheral artery disease • outcomes research

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Abbreviations

AAS	Acute aortic syndrome
CTA	Computed tomography angiography
^{18}F-FDG	^{18}F-fluorodeoxyglucose
IMH	Intramural hematoma
PAU	Penetrating aortic ulcer
PET/CT	Positron emission tomography/computed tomography
SUV	Standardized uptake value
TBR	Target to blood ratio
TEVAR	Thoracic endovascular repair
ULP	Ulcer-like projection

INTRODUCTION

Acute intramural hematoma (IMH) is an important disease entity of acute aortic syndrome (AAS).[1] Although IMH has been widely studied, the natural history and pathogenesis remain unclear.[2–5] In the International Registry of Aortic Dissection (IRAD), 58% of IMH were classified as Stanford type B and abrupt chest and back pains were the most common presenting symptoms in 78% and 62% of patients, respectively. The in-hospital mortality was 4% in conservatively treated group.[6] Previous studies have investigated the predictors of adverse aortic events (AAE) in patients with type B IMH, including factors such as persistent pain, maximum aortic diameter ≥50 mm, sustained elevation of C-reactive protein (CRP) level, and organ ischemia.[7–9] Given the favorable short-term and long-term prognosis, patients with distal IMH involving the descending aorta are recommended to have conservative treatment.[4,5,10,11]

Ulcer-like projection (ULP), defined as a focal, blood-filled pouch protruding into the thrombosed lumen of the aorta (Figure 1), has been considered as a prognostic factor of progression of type B IMH. The presence of ULP, whether detected initially or newly developed in the follow-up period, seems to increase the rate of AAE such as overt aortic dissection and aortic rupture.[12–15] Based on these results, some investigators recommend that in addition to optimal medication therapy, type B IMH patients should receive endovascular treatment to eliminate ULP.[16–19] However, others advocate a more conservative approach since most patients are asymptomatic.[13] It is therefore important to investigate methods for more accurate risk stratification of patients with type B IMH. It has been suggested that ^{18}F-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography/computed tomography (PET/CT) could be used to assess aortic involvement in inflammatory vascular disease (e.g., Takayasu arteritis) and to detect endovascular graft infection.[20–23] However, there are limited data on the application of PET/CT to assess AAE in AAS.

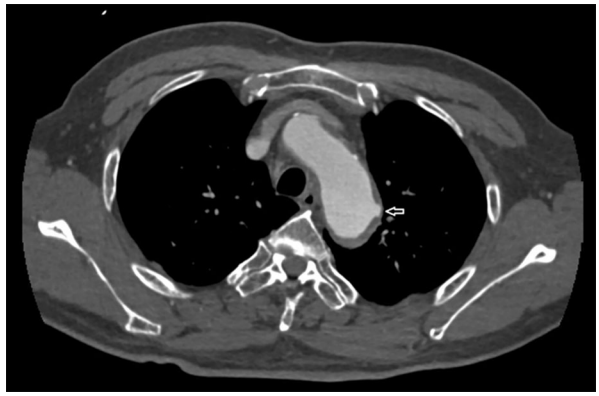

Figure 1. Intramural hematoma with ulcer-like projection (*arrow*).

The aim of this study therefore was to determine the prognostic value of ^{18}F-FDG PET/CT imaging in patients with type B IMH.

METHODS

Patient Characteristics

This study included a total of 36 consecutive patients admitted to our institute with a diagnosis of acute type B IMH between March 2015 and March 2016. The diagnosis was confirmed on the basis of clinical symptoms and results of contrast-enhanced computed tomography angiography (CTA) performed within 14 days from the onset of symptoms. Type B IMH was diagnosed by the presence of a circular or crescent-shaped thickening along the descending aortic wall and absence of any detectable blood flow in the contrast-enhanced CT scan. IMH was classified according to the Stanford Classification.[1]

ULP was defined as one of these, 1- a focal, blood-filled pouch protruding into the thrombosed lumen of the aorta on the initial CT images, 2- an intramural blood pool (which was defined as small concentration of contrast agent within the IMH), 3- a penetrating aortic ulcer without calcified plaques.

Patients with sub-acute and chronic type B IMH or Type A IMH or aortic dissection with typical double-barreled false lumen were excluded from this study. Written informed consent was obtained from all patients. This study was approved by the Institutional Review Board of Guangdong General Hospital.

Management Protocol

Patients with acute type B IMH were initially treated with medications for pain relief and blood pressure control (systolic blood pressure ≤100 to 120 mmHg). Patients with persistent pain despite receiving adequate medications, patients with

signs of organ ischemia, and patients who exhibited progression of IMH during the follow-up period received thoracic endovascular repair (TEVAR) based on current clinical guidelines.[1]

CTA Imaging Evaluation

All study patients underwent CT scans with contrast enhanced, thin-sliced (range 1 to 2.5 mm) spiral CT (64-slice multidetector LightSpeed VCT; General Electric Fairfield, CT). Multiplanar reconstruction was achieved for image evaluation. Morphologic parameters like initial ULP and maximum aortic diameter on initial CTA, interval changes of initial ULP, maximum aortic diameter, and newly developed ULP on follow-up CTA scans were evaluated independently by two experienced radiologists and all decisions were made by consensus. The readers were blinded to information obtained from other imaging modalities.

PET/CT Imaging Evaluation

The procedure was explained to all patients prior to undergoing the PET/CT imaging in order to minimize their concerns and anxiety. All images were obtained by using Sensation Biograph Somatom 16 HR PET/CT (SIEMENS, Germany). Patients were instructed to fast at least 6 hours prior to the scan to achieve a blood glucose level between 4.0 and 8.0 mmol/L (72.0 to 144.0 mg/dL) before injection of the tracer. Patients were administered 0.16 mCi/kg of ^{18}F-FDG intravenously. Whole body PET/CT imaging was performed approximately 60 ± 5 minutes after injection of the tracer, starting at the base of the skull and scanning in the cranio-caudal direction down to the mid-thigh in the supine position. The CT acquisition parameters were as follows: 120 kV, 50 mA, 16 helical slices, 0.5 s/rotation, a pitch of 0.75, and a slice thickness of 5 mm. The PET images were acquired sequentially over a 50 cm field of view in 2 minutes in 3-dimensional mode. The matched PET and CT were reconstructed using iterative ordered-subset expectation maximization. After the PET scan was completed, a volume of non-ionic contrast medium (Iopamido 370, Bracco, Italy) was administered using an automated injector at a flow rate of 3.5 to 4 mL/s and a dose of 1.5 mL/kg of body weight. The CT scan delay was set by an automated bolus tracking technique with the triggering region of interest (ROI) at the aortic arch. The scan was triggered when the CT value of the ROI reached 100 Hounsfield Unit (HU).

Metabolic Definitions

Based on visual analysis of fused PET/CT images, the increased ^{18}F-FDG focal uptake in the vascular wall, which was found to be above levels of the surrounding vascular wall, was recorded to be positive site. Semi-quantitative analysis was performed using circular ROI over the positive site and superior vena cava (Figure 2). The maximum standardized uptake value of aortic wall (SUVmax), maximum standardized uptake value of superior vena cava (SUV_{SVC}), and the target to blood ratio (TBR, SUVmax divided by SUV_{SVC}) were calculated. The corresponding PET/CT images, displayed in multiplanar reconstruction, were interpreted by consensus of

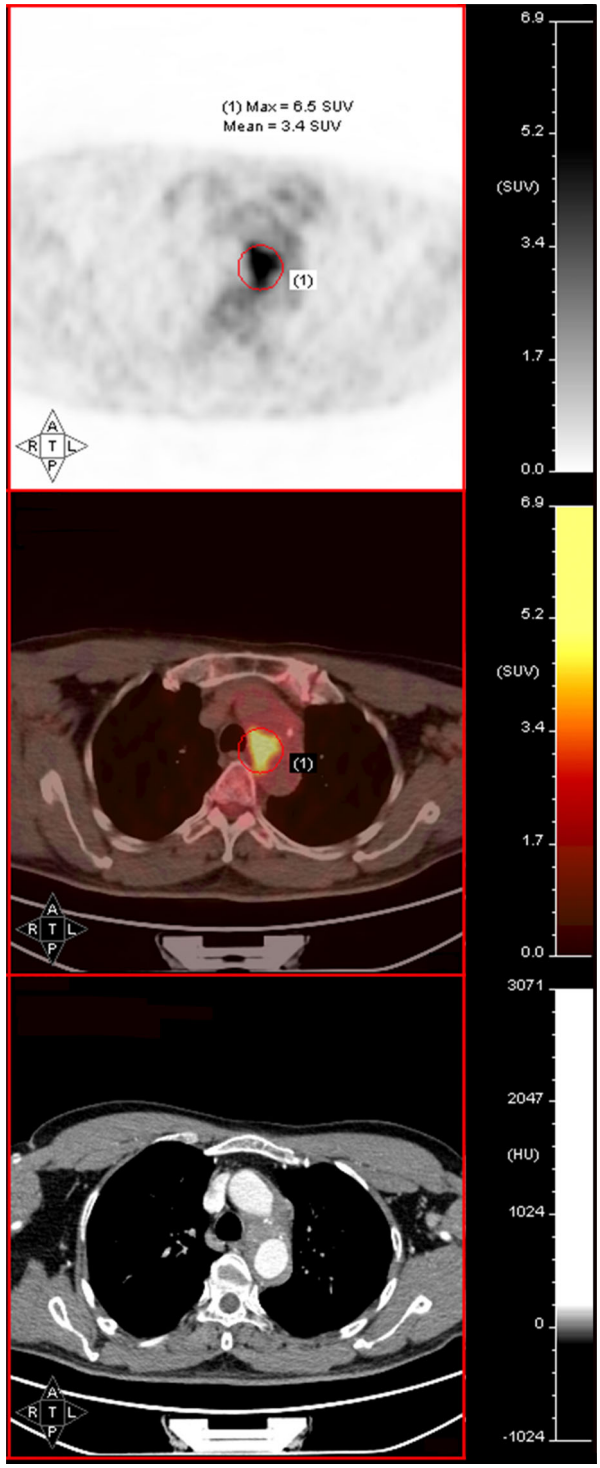

Figure 2. Three images represented PET, PET/CT, and contrast-enhanced CT ,respectively (from *top* to *bottom*). A circular ROI was placed over the positive site and the maximum SUV were measured.

two experienced nuclear medicine physicians who were blinded to reports from other imaging modalities.

Follow-up Protocol

Contrast-enhanced CTA scans were performed within 3 months of onset of IMH. These follow-up CTA scans were compared to the initial CTA. The interval changes of ULP and aortic diameter were measured on axial and, if available, on coronal and sagittal CT images.

Adverse aorta-related events were defined by a composite of conversion to TEVAR, development of aortic dissection with intimal flap (classical or localized), enlargement of initial ULP, newly developed ULP, and aortic enlargement (≥60 mm or increased by 25%). A patient having multiple events was considered as having only one AAE.

Statistical Analysis

Continuous variables were expressed as means and standard deviation, and compared using *t* test for independent variables. Data which did not present a normal distribution were expressed as median (25th–75th percentile) and calculated by Mann–Whitney test. Categorical variables were expressed as percentages and compared using Chi squared test. Fisher's exact test was used when necessary. The receiver-operating-characteristic curve was applied to estimate the predictive accuracy of ^{18}F-FDG uptake with calculation of the area under these curves. The optimal SUV value was determined by Maximum Youden's index. Differences were considered significant if the two sided *P* value was <0.05. The adjusted P values were controlled by the Benjamini–Hochberg critical value for a false discovery rate of 0.05. Data analyses were performed with SPSS 17.0 (SPSS, Inc, Chicago, IL) statistical software.

RESULTS

The study population comprised 28 males and 6 females, and the mean age was 57.9 ± 9.2 years. All patients underwent PET/CT scans within 2 weeks of onset of IMH. Two of the 36 patients refused to receive follow-up CTA and were therefore excluded. Of the 34 patients, 18 (53%) had AAE. The patient demographics and clinical features were not significantly different at baseline between patients with or without AAE (Table 1). The average time between disease onset and admission was 4.06 ± 2.89 days. Among the life-threatening complications observed during the hospitalized period were visceral ischemia (n = 2), acute kidney injury (n = 4), and pleural effusion (n = 6). Most of the study patients received β-blockers (n = 33, 97%) and there was no significant difference in medication use between the two groups.

Inflammation Markers, CTA and PET/CT Imaging

There were no significant differences in D-dimers or CRP levels between patients with or without adverse aortic events (Table 2). In 25 of the 34 study patients (74%), an initial ULP was seen, but this was not associated with AAE. There was no significant difference in the mean value of maximal aorta diameter (36.5 ± 4.95 mm) between the two groups. Patients with adverse aortic events had a significantly higher level of SUVmax (4.3 ± 0.6 vs 3.7 ± 1.0, $P = 0.02$) compared to patients without AAE, although there was no significant difference in TBR (1.6 ± 0.2 vs 1.5 ± 0.5, $P = 0.064$).

Follow-up Data

The median follow-up time was 42 (33, 63) days. No patients died during the follow-up period. A total of 18 (53%) patients had AAE in the 3 months period after onset of IMH (Table 3). Of the three patients (8.8%) who underwent TEVAR, one patient suffered recurrent back pain and emergent CTA demonstrated increased hematoma. Follow-up CTA showed development of overt dissection in the other two patients. Despite adequate risk counseling, one patient with classic dissection refused interventional therapy.

ULP, PET/CT Imaging, and Adverse Aortic Events

Based on the appearance of ULP, patients with or without AAE were further divided into two categories for subgroup analysis (Table 4). Of the patients with ULP, both the SUVmax and TBR were significantly higher in the AAE group (4.3 ± 0.6 vs 3.3 ± 0.5, $P = 0.001$; 1.6 ± 0.2 vs 1.4 ± 0.2, $P = 0.01$). There was no significant association between ^{18}F-FDG uptake and adverse events in patients without ULP (SUVmax, $P = 0.548$; TBR, $P = 0.905$). Patient outcome was not associated with baseline characteristics, or therapy in patients with or without ULP. The SUV values in relation to each adverse event are demonstrated in Supplement Table 1.

Receiver-operating-characteristic (ROC) curves were generated to evaluate the predictive accuracy of TBR for AAE. In patients with ULP, the mean area under the curve (AUC) was 0.807 ± 0.090. The optimal TBR cutoff point was 1.5, with a sensitivity of 74% and a specificity of 80%. In patients without ULP, the mean area under the curve was 0.44 ± 0.19 (P = NS). The

Table 1. Baseline characteristics between patients with or without adverse aortic events

	Total (n = 34)	Adverse aortic events (n = 18)	NO adverse aortic events (n = 16)	*P*
Basic characteristics				
Age, years	57.97 ± 9.18	57.56 ± 10.07	58.44 ± 8.37	0.986
Male/female	28/6	15/3	13/3	>0.999
BMI (kg/m^2)	25.4 ± 4.5	25.2 ± 4.7	25.6 ± 4.5	0.697
Heart rate	75.91 ± 10.6	76.72 ± 11.74	75 ± 9.45	0.670
Onset time	4.06 ± 2.89	3.61 ± 2.55	4.56 ± 3.25	0.484
Smoking, n (%)	12 (35.3)	7 (38.9)	5 (31.3)	0.729
Hypertension, n (%)	31 (91.2)	18 (100.0)	13 (81.3)	0.094
Hyperlipidemia, n (%)	5 (14.7)	4 (22.2)	1 (6.3)	0.340
Diabetes mellitus, n (%)	2 (5.9)	1 (5.6)	1 (6.3)	>0.999
Coronary artery disease, n (%)	4 (11.8)	2 (11.1)	2 (12.5)	>0.999
Complication				
Renal insufficiency, n (%)*	4 (11.8)	4 (22.2)	0 (0)	0.105
Pneumonia, n (%)	2 (5.9)	1 (5.6)	1 (6.3)	>0.999
Visceral ischemia, n (%)	2 (5.9)	1 (5.6)	1 (6.3)	>0.999
Pleural effusion, n (%)	6 (17.6)	5 (29.4)	1 (6.3)	0.175
Medical therapy				
β-blocker, n (%)	33 (97.1)	18 (100.0)	15 (93.8)	0.471
ACEI, n (%)	1 (2.9)	0 (0)	1 (6.3)	0.471
ARB, n (%)	25 (73.5)	14 (77.8)	11 (68.8)	0.703
α-Blocker, n (%)	8 (23.5)	5 (27.8)	3 (18.8)	0.693
CCB, n (%)	26 (76.5)	13 (72.2)	13 (81.3)	0.693
Statin, n (%)	18 (52.9)	11 (61.1)	7 (43.8)	0.492

Values are presented as n (%) or mean ± SD
ACEI, angiotensin-converting enzyme inhibitor; *ARB*, angiotensin receptor blocker; *BMI*, body mass index; *CCB*, calcium channel blocker
* Clearance of creatinine <60 mL/min

prognostic accuracy of TBR for AAE in patients with or without ULP is demonstrated in Figure 3.

Representative cases of IMH patients with ULP are shown in Figure 4. The first patient was a 55-year-old man with high ^{18}F-FDG uptake in the aortic wall, whereby the 1-month follow-up CTA indicated that the ULP at the middle descending aorta had progressed to classic aortic dissection. The second patient was a 52-year-old man with a low ^{18}F-FDG uptake in the aortic wall whereby the 1-month follow-up CTA indicated no change in the ULP at the proximal descending aorta compared to the initial scan.

DISCUSSION

The present study is a prospective study investigating the use of PET/CT to predict clinical and morphologic outcomes in patients with acute type B IMH. Our data provide new insights into the application of PET/CT in type B IMH. A number of previous studies have reported that the evolution of hematoma in the early phase of IMH could be a complex process where it could be absorbed, progress to classic aortic dissection, or even rupture.[4,5,8,12,24] However, due to limitations of current examination methods and lack of histological evidence, the etiology and prognosis of IMH remain controversial.

In this study, over half of IMH patients (n = 18, 53%) encountered AAE within 3 months of onset. These data are consistent with previous studies which reported that 16% to 47% patients with IMH progressed to dissection.[25] In this study, 12 (35%) patients progressed to classic or localized aortic dissection. A previous study of 44 medically treated patients with acute uncomplicated type B IMH, the 8-year freedom from dissection-related mortality was 40%.[26] In our present study, most of the AAE were morphologic events and there was no short-term mortality during a follow-up of up to

Table 2. Laboratory, morphologic, and metabolic findings between patients with or without adverse aortic events

	Total (n = 34)	Adverse aortic events (n = 18)	NO adverse aortic events (n = 16)	*P*
D2-dimer, μg/L	1460 (650–3135)	1855 (690–3105)	1110 (540–3140)	0.630
C-reactive protein, mg/L	87.5 (35–123.8)	86.3 (53.8–128.5)	87.5 (9.8–110.5)	0.377
Maximum aortic diameter, mm	36.5 ± 4.95	36.83 ± 4.23	36.4 ± 5.87	0.772
Initial ULP, n (%)	25 (73.5)	15 (83.3)	10 (62.5)	0.250
SUVmax	4.0 ± 0.8	4.3 ± 0.6	3.7 ± 1.0	0.020
SUV_{SVC}	2.6 ± 0.5	2.7 ± 0.43	2.5 ± 0.4	0.484
TBR	1.6 ± 0.4	1.6 ± 0.2	1.5 ± 0.5	0.064

Values are presented as n (%), mean ± SD or median (25th–75th percentile)
TBR, target to blood ratio; *ULP*, ulcer-like projection; *SUV*, standardized uptake value; *SVC*, superior vena cava

Table 3. Early progression of IMH

	n = 34
Follow-up period (day)	42 (33,63)
Composite endpoint	18 (53%)
TEVAR	3 (8.8%)
Development to aortic dissection (classic and localized)	12 (35%)
Enlargement of initial ULP	14 (41%)
New appearance of ULP	7 (21%)
Aortic dilatation	4 (12%)

Values are presented as n (%) or median (25th–75th percentile)
IMH, intramural hematoma; *TEVAR*, thoracic endovascular repair; *ULP*, ulcer-like projection

3 months. Unfavorable aortic remodeling has been regarded as a marker of poor prognosis of aortic disease,[27] and may be useful in patient management.

The present study demonstrated that 14 (41%) patients had enlargement of the initial ULP, and 7 (21%) patients exhibited development of a new ULP during the follow-up period. ULP represents the site of an intimal disruption, and it has been shown that both initial ULP as well as ULP which develops in the follow-up period predict progression.[1,4,7,8,13–17,24,28] However, due to the variable remodeling process of ULP, a generally accepted therapeutic strategy has not been established. Most patients are initially treated with a conservative approach, although a more aggressive therapy is recommended in the high-risk patients. For example, patients with a new ULP in the proximal descending aorta might benefit from endovascular treatment.[15,29] Sueyoshi

Table 4. Comparison of metabolic findings related to adverse aortic events in patients with or without ULP

	With ULP			Without ULP		
	Adverse aortic events (n = 15)	NO adverse aortic events (n = 10)	*P*	Adverse aortic events (n = 3)	NO adverse aortic events (n = 6)	*P*
SUVmax	4.3 ± 0.6	3.3 ± 0.5	0.001*	4.1 ± 0.1	4.4 ± 1.2	0.548
SUV_{SVC}	2.7 ± 0.5	2.3 ± 0.4	0.160	2.5 ± 0.3	2.7 ± 0.4	0.381
TBR	1.6 ± 0.2	1.4 ± 0.2	0.01*	1.7 ± 0.2	1.7 ± 0.8	0.905

Values are presented as n (%) or mean ± SD
TBR, target to blood ratio; *ULP*, ulcer-like projection; *SUV*, standardized uptake value; *SVC*, superior vena cava
* *P* values are significant after controlling by the Benjamini–Hochberg critical value for a false discovery rate of 0.05

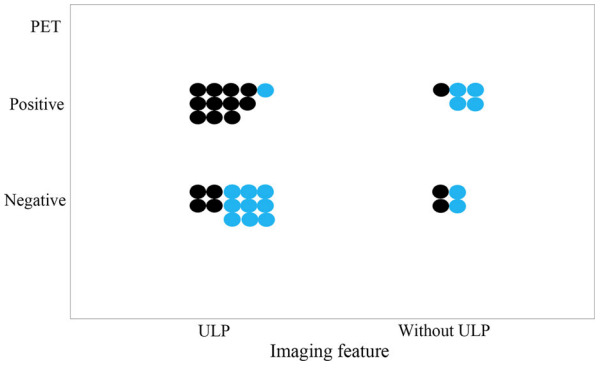

Figure 3. Prognostic accuracy of cutoff point (TBR = 1.5) for the occurrence of adverse aortic events. (positive: TBR > 1.5; negative: TBR ≤ 1.5; *black circle* adverse aortic event; *blue circle* no adverse aortic event; *ULP*, ulcer-like projection; *TBR*, target to blood ratio).

et al[17] reported that endovascular treatment could close complicated ULP possibly via hematoma resorption of the affected aorta. Consequently, differentiating high-risk patients may have direct influence on the treatment strategies.

Previous studies showed that ^{18}F-FDG uptake was associated with unfavorable prognosis in AAS.[30–32] In our study, patients with ULP had a higher risk of adverse aorta events when associated with increased SUVmax. Due to circulating ^{18}F-FDG activity, TBR was introduced to eliminate the individual variability in metabolism. TBR of 1.5 had an acceptable predictive value for differentiating high-risk from low-risk patients. The potential pathologic mechanism leading to increased ^{18}F-FDG uptake along the vessel wall is not clear. A previous study demonstrated that regions with ^{18}F-FDG uptake were enriched with leukocytes.[33] It seems plausible that acute inflammation could result in the accumulation of hypermetabolic cells such as macrophages, and enhanced ^{18}F-FDG uptake. The higher ^{18}F-FDG uptake may therefore indicate instability of the aortic wall and poorer outcomes. Based on previous reports showing the strong prognostic value of ^{18}F-FDG uptake in patients with ULP, we classified patients with ULP and with active ^{18}F-FDG uptake as high-risk group who could qualify for early intervention though more studies are needed to confirm this observation. The maximal standardized uptake values (SUVmax) reached the level of significant difference between patients with or without AAE (4.3 ± 0.6 vs 3.7 ± 1.0, $P = 0.020$), but not the TBR (1.6 ± 0.2 vs

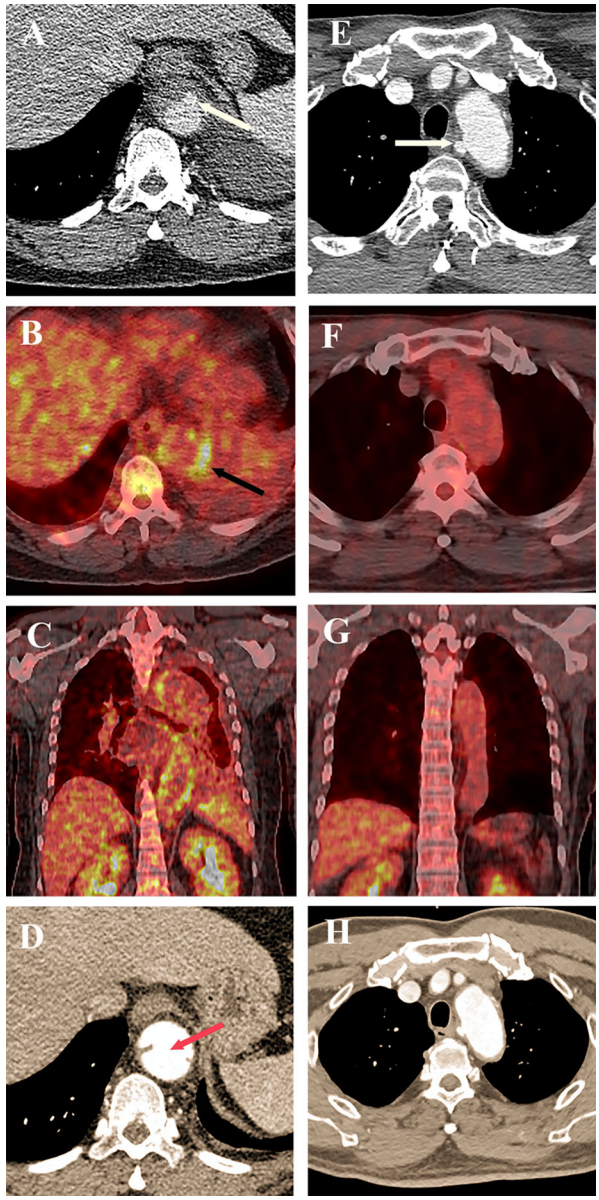

Figure 4. Two representative cases of type B IMH patients with ULP. (*ULP*, ulcer-like projection; *TBR*, target to blood ratio). *Left panel*, a 55-year-old man with greater uptake of ^{18}F-FDG in the aortic wall: A, ULP (*arrow*) was detected at the middle descending aorta in initial CTA; B-C, accumulation of ^{18}F-FDG in the aortic wall in PET/CT (*arrow*), the SUVmax and TBR were 4.8 and 1.3, respectively; D, ULP progressed to classic aortic dissection (*arrow*) 1 mo after onset. *Right panel*, a 52-year-old man with low uptake of ^{18}F-FDG in the aortic wall: E, ULP (*arrow*) was detected at the proximal descending aorta in initial CTA; F-G, no obvious accumulation of ^{18}F-FDG in the aortic wall on PET/CT, the SUVmax and TBRwere 2.7 and 1.1, respectively; H, ULP had no change 35 days after onset.

1.5 ± 0.5, $P = 0.064$). Conceivably due to excessive inflammation, patients with ULP were more likely to have AAE in the acute phase compared to patients without ULP. However, the underlying mechanisms still need further investigation and verification.

LIMITATIONS

Being a single center study, our present investigation has inherent limitations. The small sample size and short follow-up period were the major limitations of this study. It is important to verify our results in a larger sample size and from multiple institutions and with longer durations of observation. Additionally, although the PET/CT examinations were all performed during the acute phase, severely ill patients had to wait until their condition improved. These patients therefore did not receive the scans at the onset of the disease. However, the proliferative and exudative repair mechanism in the injured aortic wall which resulted in accumulation of hypermetabolic cells might be more obvious several days after the onset.[34] The PET/CT results could therefore possibly reflect, to some extent, the metabolic status of patients. Lack of biopsy collection would be a barrier while exploring the relationship between pathology and metabolic status.

NEW KNOWLEDGE GAINED

To the best of our knowledge, the present study is the first prospective study investigating the use of PET/CT to predict clinical and morphologic outcomes in patients with acute type B IMH. The results indicate that^{18}F-PET/CT imaging in patients with ulcer-like projection could identify high-risk patients for future adverse aortic events.

CONCLUSION

^{18}F-FDG PET/CT provided a new approach for predicting risk in patients with type B IMH. Patients with ULP who exhibited a greater uptake of ^{18}F-FDG in the aortic wall were more likely to develop adverse aortic events. More careful surveillance with serial imaging is recommended for high-risk patients. Further long-term studies involving a larger sample size are required to validate our data. It is also important to conduct studies investigating the efficacy of endovascular treatment for high-risk patients with ULP.

Disclosure

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