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CLINICAL CASE REPORT

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Multiple ^{18}F-Fluorodeoxyglucose Positron Emission Tomography Scans Showing Progression of Abdominal Aortic Aneurysm

A Case Report

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Abstract: Although the precise mechanisms underlying the pathogenesis of abdominal aortic aneurysm (AAA) remain unclear, aortic wall inflammation has been implicated in AAA development. Several studies have reported the use of ^{18}fluoro-deoxyglucose (^{18}F-FDG)/positron emission tomography (PET) to assess the nature of AAA.

We present a case of 77-year-old Japanese male with juxta-anastomotic AAA who was followed up with multiple ^{18}F-FDG-PET/CT scans over 7 years. The scans revealed chronological changes in aortic wall inflammation leading to progress and eventual rupture.

This case supports a notion that aortic wall inflammation plays a role in AAA progression and rupture.

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Abbreviations: ^{18}F-FDG = ^{18}fluoro-deoxyglucose, AAA = abdominal aortic aneurysm, CT = computed tomography, MMP = matrix metalloproteinase, PET = positron emission tomography, SUVmax = maximum standardized uptake value, VOI = volume of interest.

INTRODUCTION

Abdominal aortic aneurysm (AAA) is a degenerative disease of the aorta, occurring primarily in the elderly. The aneurysmal wall comprises intimal atherosclerosis, disruption of elastic media, and inflammatory cell infiltration in the adventitial layer. Although the underlying mechanisms remain to be elucidated, aortic inflammation, particularly at the adventitial layer, is suggested to be the important pathogenesis of AAA.[1–3]

We previously reported that adventitial mast cells play a role in the development of AAA, and we showed that mast cells stimulate the synthesis of matrix metalloproteinase (MMP)-9 in macrophages, leading to the degradation of extracellular matrix in aortic wall.[4]

As the aneurysm grows, the rupture rate increases.[1] Patients are regularly followed up with computed tomography (CT); however, vulnerability of aneurysmal wall cannot be adequately evaluated using CT scans. Other studies, as well as our previous study, have shown that ^{18}fluoro-deoxyglucose (^{18}F-FDG) positron emission tomography (PET) imaging may be an alternative method to assess the nature of AAA wall[5–10]; we found that the amount of ^{18}F-FDG uptake is associated with MMP-9 activity.[10] Here, we present a case of a patient with juxta-anastomotic AAA who was followed up with multiple ^{18}F-FDG-PET/CT scans, which revealed chronological changes in aortic wall inflammation leading to progress and eventual rupture.

CASE PRESENTATION

In 2004, a 77-year-old Japanese man with a vascular prosthesis (open repair) for infrarenal abdominal aortic aneurysm, received in 2001, was admitted to our hospital for evaluation of a re-dilating abdominal aorta (41 × 42 mm in maximal diameter) at just below the bifurcation of the renal arteries and anastomosis. He never stopped smoking since the previous surgery, and he was on antihypertensive drugs (amlodipine 5 mg/day and losartan 50 mg/day). He refused the re-operation; thus, we decided to follow-up with CT on a regular basis. In 2007, he was re-admitted to our hospital because of acute pneumonia. On admission, we performed ^{18}F-FDG-PET/CT for the first time to exclude the possible coexistence of lung cancer. Whole body PET/CT images were obtained 60 minutes after injection of 200 MBq FDG using the 3-dimensional method. We reconstructed all PET images using iterative algorithms (Fourier rebinning plus attenuation-weighted ordered-subset expectation maximization, 2 iterations, 8 subsets, 5-mm Gaussian filter) with CT-based attenuation correction, as previously described.[11] Then, the data were further reconstructed with a 256 × 256 matrix and 3-mm-thick slices using the ESOFT4.5 workstation (Siemens/CTI, Knoxville, TN). A volume of interest (VOI) of the AAA was manually drawn on the 3-dimensional whole body images encompassing the entire lesion. The maximum VOI was defined as the maximum standardized uptake value (SUVmax). To monitor the lesion quantitatively, SUVmax of the aneurysm was recorded in each study. Figure 1A (October 2007) showed that the maximum size of the aneurysm was 44 × 53 mm and

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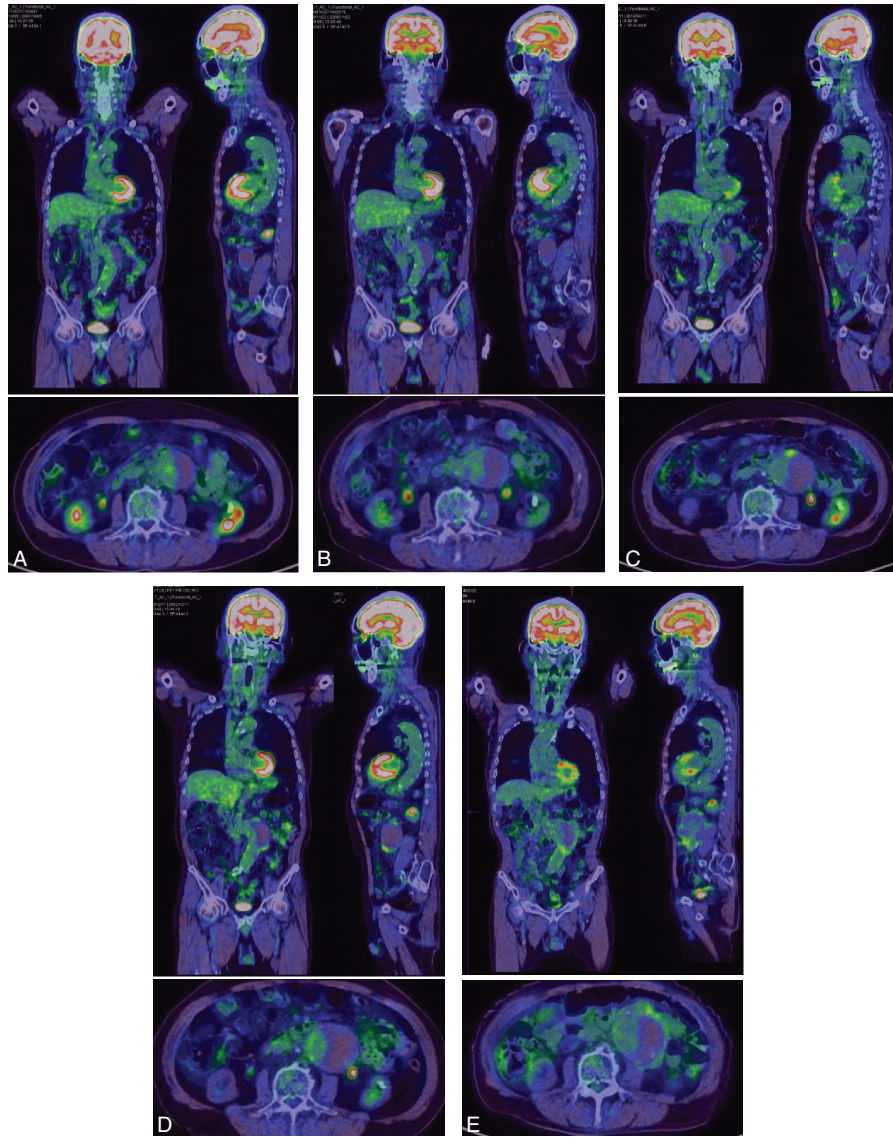

FIGURE 1. Coronal, sagittal, and axial sections of ^{18}F-FDG-PET scans fused with CT scans of a patient with a juxta-anastomotic abdominal aortic aneurysm over a 7-year follow-up period obtained in October 2007 (A), November 2009 (B), April 2012 (C), December 2012 (D), and January 2014 (E). High focal ^{18}F-FDG uptake along with the implanted graft (A–C) shifted to the bottom of the aneurysm sac (D), and it extended to the entire aneurysmal wall before the rupture and patient's death (E). ^{18}F-FDG = ^{18}fluoro-deoxyglucose, CT = computed tomography, PET = positron emission tomography.

SUVmax was 3.92. In 2009, the patient complained of hoarseness and underwent laryngeal microsurgery for squamous cell carcinoma of the vocal cords. We performed ^{18}F-FDG-PET/CT to evaluate metastases preoperatively and determined that the maximal aneurysm diameter was 49 × 52 mm with SUVmax of 3.68 (Figure 1B; November 2009). Thereafter, he agreed to join our research program (permission number 985, University of Miyazaki) for follow-up evaluation of the aneurysm with ^{18}F-FDG-PET/CT under administration of amlodipine 5 mg/day, losartan 50 mg/day, and mast cell stabilizer tranilast 300 mg/day (the product is not labeled for use) on the following dates: April 2012 (Figure 1C; 55 × 62 mm in maximal diameter; SUVmax = 4.25); December 2012 (Figure 1D; 58 × 61 mm; SUVmax = 5.14); and January 2014 (Figure 1E; 65 × 82 mm, SUVmax = 5.18). Figure 2 summarizes the chronological changes of SUVmax over 7 years. Nine months after the last scan, he was brought to the emergency room due to syncope accompanied by massive upper gastrointestinal bleeding. A CT scan suggested further growth of the aneurysm (70 × 83 mm) with an aortojejunal fistula. He died the following day.

This patient provided written informed consent to participate in the study, and his family approved the publication of this case details. This study was approved by the Human Investigation Review Committee of the University of Miyazaki (No. 985) and conformed to the principles outlined in the Declaration of Helsinki (2013).

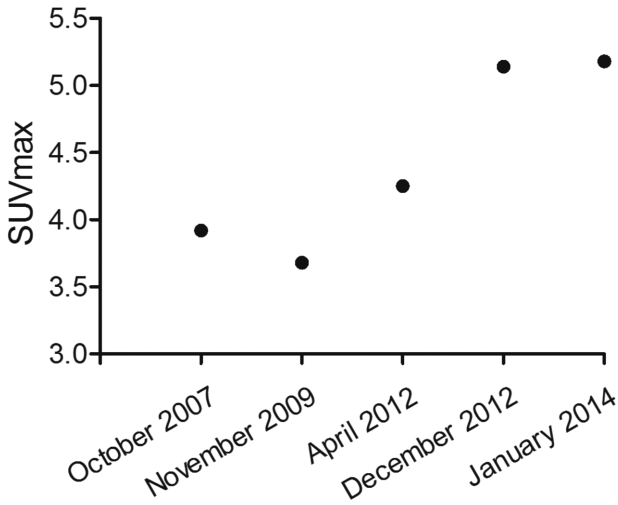

FIGURE 2. Chronological changes of maximum standardized uptake value (SUVmax) of ^{18}F-FDG in aneurysmal wall over 7 years. ^{18}F-FDG = ^{18}fluoro-deoxyglucose, SUVmax = maximum standardized uptake value.

DISCUSSION

This report describes the clinical course of a juxta-anastomotic AAA during progression and eventual rupture, as assessed by multiple ^{18}F-FDG-PET/CT scans over the 7-year follow-up period. To the best of our knowledge, this is the first case to follow the AAA size and its nature by ^{18}F-FDG-PET/CT scan for a long period. In addition, these images support the mechanistic insight into the significant role of glycolytic activity in chronological progression and rupture of the aneurysmal wall.[10,12,13] The first 3 scans revealed high focal ^{18}F-FDG uptake along with the implanted graft in the neck over 5 years. The fourth scan showed that the most intensified site shifted to the bottom of the aneurysm sac, and the final scan before the rupture and patient's death indicated that it extended to the entire aneurysmal wall. We previously reported that the ^{18}F-FDG-PET signal was correlated with the expression of glucose transporter-3, which is mainly distributed in macrophages, and the activity of MMP-9 in human AAA.[10] The multiple ^{18}F-FDG-PET/CT scans suggest that the extent of inflammation of the aneurysmal wall was not uniform and that the maximal inflammatory site varied over time. We speculate that the high focal ^{18}F-FDG uptake might have reflected ongoing extracellular matrix degradation, leading to the continued enlargement of the aneurysm and the resultant rupture. Future studies are designed to image ^{18}F-FDG-PET/CT scan to assess if any medical treatment affects the aortic inflammation and the resultant expansion.

CONCLUSION

This case supports that active inflammation is associated with the progression and rupture of AAA, and that multiple periodic ^{18}F-FDG-PET/CT scans indicate sequential changes in size and potential active inflammation of the aneurysmal wall.

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