¹⁸F-Fluorodeoxyglucose Positron Emission Tomography 10 Days Before Onset of Aortic Dissection

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Figure. (A) Contrast computed tomography (CT) at the onset of aortic dissection on (a,b) August 29, 2016, and at (c) 15 days later (September 13, 2016). Arrows, ulcer-like projection. (B) ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography (¹⁸F-FDG-PET/CT) performed on (a–c) February 3, 2010, (d–f) November 20, 2013, and (g–i) August 18, 2016 (10 days before the aortic dissection), and (j–l) non-contrast CT on August 18, 2016, corresponding to the PET image. Arrow, maximum site of ¹⁸F-FDG uptake in the descending aorta. (C) FDG uptake indices in whole body PET/CT 60 min after injection of 200 MBq FDG. To evaluate the extent and expanse of glycolytic activity, all images were transferred to a dedicated workstation (Syngo.via; Siemens). FDG uptake indices were obtained by setting the volume of interest at the level of the left atrium of the thoracic descending aorta on 3-D FDG-PET/CT. The maximum standardized uptake value (SUV) was divided by the blood-pool SUV, yielding a target-to-background ratio (TBR). Total lesion glycolysis (TLG) was calculated by multiplying the metabolic volume of aortic wall inflammation (MV) with the average SUV within the volume. A threshold of 40% SUVmax was used to delineate the MV.

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Cystic medial degeneration is an important factor for the development of aortic dissection, but recent experimental and clinical investigations have suggested that aortic wall inflammation may also serve as a pathological substrate.^{1,2} ¹⁸F-fluorodeoxyglucose positron emission tomography/computed tomography (¹⁸F-FDG-PET/CT) is a useful modality for visualizing not only metabolically active neoplasm cells but also non-neoplasm inflammatory cells. ¹⁸F-FDG uptake is observed at multiple sites in an atherosclerotic aorta³ and is enhanced after the dissecting aortic wall.^{4,5} We encountered the case of an 87-year-old man who underwent multiple ¹⁸F-FDG-PET/CT, including the last scan performed on August 18, 2016. He complained of chest and back pain at midnight on August 29, 2016, and emergency contrast CT showed a Stanford type B aortic dissection: intimal flap arising from the thoracic descending aorta distal to the left subclavian artery, extending to the level of the diaphragm. In addition, a contrast-material-filled pouch, a so-called ulcer-like projection, was observed at the level of the pulmonary trunk in the thrombosed false lumen of the dissecting aorta (**Figure A-a,b**). ¹⁸F-FDG-PET/CT showed an increase in ¹⁸F-FDG uptake over 6 years (**Figure B-a-c**, February 3, 2010; **d-f**, November 20, 2013; and **g-i**, August 18, 2016), with the maximum uptake being at the level of left atrium on the last scan (arrow, **Figure B-g-i**). **Figure C** summarizes the trend for ¹⁸F-FDG uptake assessed using maximum standardized uptake value (SUVmax), target-to-background ratio (TBR), and total lesion glycolysis (TLG) in the descending aorta. Blood pressure was controlled with β -blockers and angiotensin II blockers, in addition to calcium channel blockers. The follow-up contrast CT 15 days after the aortic dissection showed the elimination of the ulcer-like projection (**Figure A-c**).

¹⁸F-FDG uptake is enhanced following aortic dissection, and its intensity is associated with adverse outcome.⁶ There is little information, however, on whether the aortic wall is inflamed before dissection in humans. We report here that ¹⁸F-FDG-PET captured the latest metabolic status of the aortic wall 10 days prior to dissection. Non-contrast CT performed on the same day did not indicate an impending dissection (**Figure B-j-l**). Serial ¹⁸F-FDG-PET/CT showed that radiotracer activity in the aortic wall was not homogenous. TLG can reflect the volume of metabolically active disease whereas both SUVmax and TBR reflect only single-voxel information.^{7,8} Therefore, we propose TLG as

the monitoring parameter for aortic wall inflammation. Also, intense inflammation may not necessarily initiate aortic wall dissection because maximum ¹⁸F-FDG uptake did not correspond with either ulcer-like projections or the entry site of the aortic dissection.

In conclusion, this study supports the hypothesis that vascular inflammation in atherosclerosis is an important pathological substrate for aortic dissection. Further studies are needed to investigate whether ¹⁸F-FDG-PET might be useful to predict the onset of aortic dissection in patients at high risk of complications for invasive medical procedures, because arterial wall uptake of ¹⁸F-FDG on PET in stable cancer patients indicates higher risk for cardiovascular events.⁹

Disclosures

None.

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