Utility of $^{18}$F-Fluorodeoxyglucose Positron Emission Tomography Computed Tomography in the Diagnosis and Management of Aortitis

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A 68-year-old woman was referred to the Vasculitis Service following several specialty reviews. She reported 6 months of back pain, a 10-kg weight loss, and lethargy. She was a current smoker with a 26 pack-year history. She had no prior illness and the results of her physical examination were normal. Blood pressure was 132/82 mm Hg. She was prescribed no regular medications but had taken intermittent acetaminophen. Initial blood investigations revealed normal kidney function, a hemoglobin of 9.2 g/dL (normal, 11.5–16.0), platelet count of 449×10^9/L (150–400), C-reactive protein of 78 mg/L (0–10), and an erythrocyte sedimentation rate of 102 mm/h (<40). Urinalysis was clear, and full infection and immunology screens were negative. Serum immunoglobulins including IgG, were all within normal limits, and a temporal artery biopsy revealed no evidence of giant cell arteritis.

Initially, the patient underwent a computed tomography (CT) scan of chest, abdomen, and pelvis with contrast. This demonstrated widespread noncalcific mural thickening of the thoracic and abdominal aorta (Figure 1A, red arrow). No other vascular abnormalities were noted. An $^{18}$F-fluorodeoxyglucose positron emission tomography ($^{18}$F-FDG PET) CT scan was then performed; an uptake time of 1 h was used between injection of the tracer and scanning. The scan demonstrated diffuse uptake in relation to the aortic wall corresponding to the mural thickening seen on CT (Figure 1B: sagittal attenuated corrected image and fused $^{18}$F-FDG-PET CT). Quantification of mural uptake was based on the maximal standardized uptake value; a value of <4.9 represents low uptake, 5 to 9.9 indicates a moderate degree of uptake, and >10 indicates high uptake. The patient had a maximal standardized uptake value >10. Based on these findings and the clinical picture, she was diagnosed with aortitis. Following assessment of carotid-femoral pulse wave velocity, as a measure of arterial stiffness, the patient was started on immunosuppressive treatment with prednisone and azathioprine. Prednisone was commenced at a dose of 60 mg daily (=1 mg/kg), gradually reduced to 10 mg daily over a period of 12 weeks and then to a maintenance dose of 5 mg daily after a further 12 weeks. Azathioprine was initially commenced at 100 mg daily and then increased to a maintenance dose of 150 mg daily once it was established that there was no evidence of hepatotoxicity.

There was a rapid improvement in both the patient’s symptoms and blood parameters. At 1 year the patient was considered to be in established clinical disease remission. Whereas her blood pressure was similar to that at disease presentation (137/78 mm Hg), carotid-femoral pulse wave velocity had fallen from 8.9 m/s before treatment to 6.8 m/s in keeping with an unstiffening of the vasculature and an improved cardiovascular prognosis. $^{18}$F-FDG-PET CT scan was repeated at this point. This showed complete resolution of the previously seen changes with no detectable $^{18}$F-FDG uptake (Figure 2A and 2B, maximal standardized uptake value <4.9). The patient’s immunosuppression was tapered and stopped 3 months later (after a total treatment period of 18 months). She remains well at last follow-up 5 years after the initial presentation.

This report highlights the use of $^{18}$F-FDG-PET CT in assessing the response to therapy in large-vessel vasculitides. Where biopsy or biochemical diagnosis is not possible, $^{18}$F-FDG-PET CT may also offer a valuable diagnostic tool over other imaging modalities. Neumann et al previously reported on the use of $^{18}$F-FDG-PET CT to diagnose large-vessel vasculitides where standard CT and MRI had proven inconclusive. The scan in that case demonstrated $^{18}$F-FDG uptake in the wall of the thoracic and abdominal aorta and also in the major branches. The $^{18}$F-FDG-PET CT scan was repeated after 6 weeks of immunosuppression and demonstrated reduced but persistent $^{18}$F-FDG uptake. It remains unclear whether persistently increased $^{18}$F-FDG uptake by a vessel is attributable to chronic inflammation or a reflection of vascular remodeling, fibrosis, or atherosclerosis following treatment. Maximum standardized uptake value has been shown to correlate positively with circulating measures of inflammation. For the case described here, we repeated the $^{18}$F-FDG-PET CT scan at 12 months, following clinical improvement alongside a resolution of the inflammatory response and a fall in arterial stiffness (which was unaccounted for by a fall in blood pressure). The current report takes forward the use of $^{18}$F-FDG-PET CT in autoimmune inflammatory conditions to demonstrate complete resolution of...
18F-FDG uptake following immunosuppressive treatment in keeping with disease remission.

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Disclosures
None

References

Figure 1. A, Contrast-enhanced sagittal CT scan demonstrating widespread noncalcific mural thickening of the thoracic and abdominal aorta. B, Sagittal attenuated corrected image and fused 18F-FDG-PET CT demonstrating diffuse uptake in relation to the aortic wall. CT indicates computed tomography; and 18F-FDG PET, 18F-fluorodeoxyglucose positron emission tomography.

Figure 2. A, Sagittal CT scan and (B) sagittal attenuated corrected image and fused 18F-FDG-PET CT demonstrating resolution of the FDG uptake in relation to the aortic wall. CT indicates computed tomography; and 18F-FDG PET, 18F-fluorodeoxyglucose positron emission tomography.
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