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CASE REPORT

EXTENSIVE AORTITIS WITH STANFORD TYPE B INTRAMURAL HEMATOMA AND RENAL ARTERITIS

INFECTIOUS VERSUS INFLAMMATORY AORTITIS

MOHAMMED AMINE BIYI · ZAKARIA OUASSAFRAR · AMAL GUENSI
MOROCCO

LARGE-VESSEL INFLAMMATION
Extensive aortic and renal artery FDG uptake

DIAGNOSTIC OVERLAP
Inflammatory and infectious patterns may overlap

STANFORD TYPE B IMH
Intramural hematoma and aortic wall ulcerations

ETIOLOGIC CAUTION
PET/CT findings require clinical correlation

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Extensive Aortitis with Stanford Type B Intramural Hematoma and Renal Arteritis: A Nuclear Medicine Perspective on Infectious versus Inflammatory Aortitis

Mohammed Amine Biyi[1]*, Zakaria Ouassafrar[1], Amal Guensi[1]

[1]Nuclear Medicine Department, Ibn Rochd University Hospital Center, Casablanca, Morocco

***Corresponding author:** biyiamine@gmail.com

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Abstract

Aortitis associated with a Stanford type B intramural hematoma and aortic wall ulcerations requires prompt assessment because both inflammatory and infectious etiologies may produce overlapping imaging findings. We report a 78-year-old woman with poorly controlled hypertension who presented with pulsatile abdominal pain, dyspnea, and vomiting. Marked inflammatory activity was present. 18F-FDG PET/CT demonstrated intense large-vessel uptake, and subsequent CT angiography demonstrated post-isthmic descending aortitis with a Stanford type B intramural hematoma, aortic wall ulcerations, and right renal artery involvement. Treponemal serology was reactive, whereas RPR/VDRL was nonreactive, raising concern for a possible infectious contribution without establishing syphilitic aortitis. The patient subsequently deteriorated, required intensive care, and died shortly thereafter. This case illustrates that inflammatory and infectious aortitis may show overlapping patterns of vascular FDG uptake.

Keywords: 18F-FDG PET/CT; aortitis; Stanford type B intramural hematoma; giant cell arteritis; infectious aortitis; syphilis

Introduction

18F-FDG PET/CT is an established imaging modality for assessing suspected large-vessel vasculitis and can demonstrate metabolically active inflammation in the aorta and its major branches. Visual assessment commonly compares vascular FDG uptake with hepatic background activity; uptake greater than liver is considered strongly suggestive of active large-vessel inflammation in the appropriate clinical setting [3,4].

However, FDG uptake is not etiologically specific. Infectious aortitis, including syphilitic aortic disease, may overlap with inflammatory large-vessel vasculitis on PET/CT [1,5–8]. Consequently, a vasculitic distribution of FDG uptake should be interpreted in the context of clinical presentation, laboratory findings, microbiological or serological investigations, and anatomic imaging. The present case highlights this diagnostic overlap in a patient with extensive aortic involvement and a Stanford type B intramural hematoma.

Case Presentation

A 78-year-old woman with poorly controlled hypertension was admitted on 29 January 2026 with a 10-day history of pulsatile abdominal pain accompanied by dyspnea and vomiting. Initial abdominal-pelvic CT demonstrated an infrarenal abdominal aortic aneurysm and an intramural hematoma. Laboratory testing showed a marked inflammatory syndrome, with C-reactive protein 337 mg/L, erythrocyte sedimentation rate 108 mm/h, and a leukocyte count of 9,760/µL.

18F-FDG PET/CT was performed for suspected large-vessel vasculitis after administration of 232 MBq 18F-FDG, with image acquisition 60 minutes after injection. Blood glucose was 1.24 g/L and body weight was 80 kg. The examination demonstrated intense FDG uptake involving the aorta and the right renal artery. The principal reported quantitative findings are summarized in Table 1.

Table 1. Reported vascular FDG uptake values.

Vascular site	SUVmax	Hepatic background	Visual uptake grade
Aortic arch	6.08	5.11	Grade 3
Thoracic descending aorta	5.48	5.11	Grade 3 (reported)
Abdominal aorta	5.30	5.11	Grade 3
Right renal artery	7.46	5.11	Grade 3
Femoral arteries	2.52–2.61	5.11	Grade 1

Additional reported findings included bilateral femoral/popliteal artery uptake (SUVmax 2.15–2.61), splenic uptake (SUVmax 5.77), and bone-marrow uptake (SUVmax 8.71). Subsequent CT angiography demonstrated post-isthmic descending aortitis with a Stanford type B intramural hematoma, aortic wall ulcerations, and right renal artery involvement. Serological testing performed after PET/CT showed reactive syphilis IgG/IgM serology (index 4.99 S/CO) and reactive TPHA (titer 1:80), with nonreactive RPR/VDRL. No further infectious-disease assessment was available because of rapid clinical deterioration. The documented acute management consisted of bisoprolol 5 mg/day, perindopril

5 mg/day with a target systolic blood pressure of ≤120 mmHg, and therapeutic heparin. After admission to the intensive care unit, the patient received broad-spectrum antibiotics and vasoactive treatment. No specific antisyphilitic treatment was administered. The planned temporal artery biopsy was not performed, and the patient died shortly after intensive-care admission from hemodynamic instability and heart failure. No follow-up PET/CT was available.

Imaging Findings

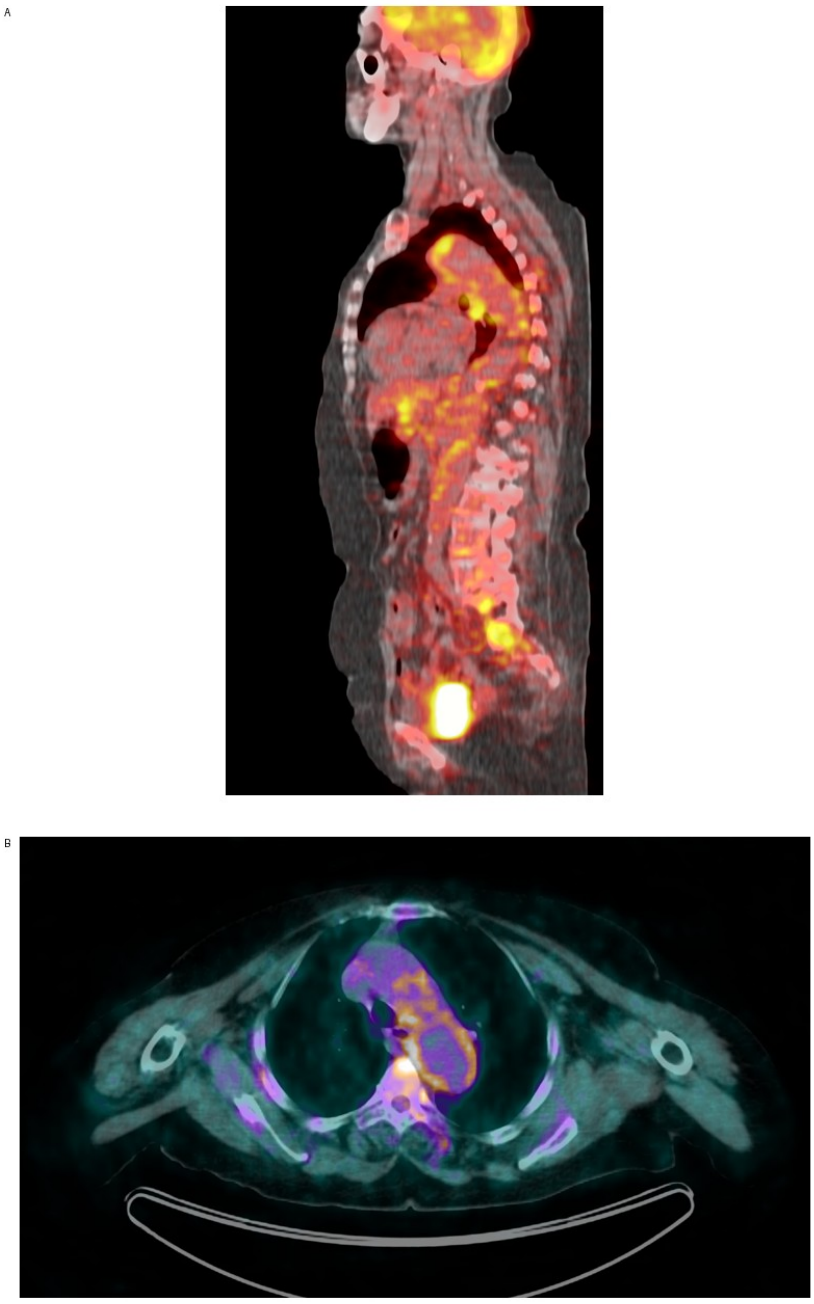

Figure 1. 18F-FDG PET/CT. (A) Sagittal fused PET/CT image demonstrating increased FDG uptake along the thoracic and abdominal aorta. (B) Axial fused PET/CT image at the level of the thoracic aorta demonstrating prominent mural FDG uptake.

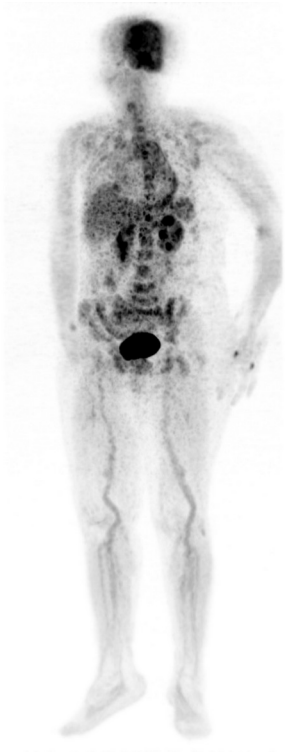

Figure 2. Whole-body maximum-intensity-projection 18F-FDG PET image from the submitted image set.

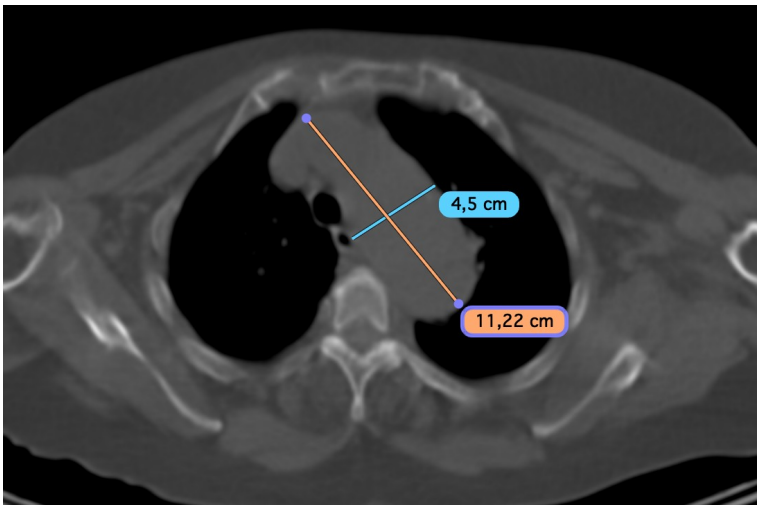

Figure 3. Axial CT angiographic image of the thoracic aorta showing the reported aortic wall abnormality associated with the Stanford type B intramural hematoma. Source-image caliper measurements are shown.

Discussion

The principal educational message of this case is not that PET/CT can distinguish inflammatory from infectious aortitis, but rather that it may identify the extent and metabolic activity of vascular inflammation while leaving the underlying etiology unresolved. In this patient, intense aortic and right renal artery FDG uptake supported active large-vessel inflammation (Figs. 1 and 2), whereas the subsequently identified reactive syphilis serology introduced an important infectious differential diagnosis [1,5–8].

The imaging appearance should therefore not be described as “identical” between infectious and inflammatory aortitis. There is substantial overlap in FDG uptake, but diagnostic interpretation depends on the pattern and distribution of vascular involvement, the accompanying CT angiographic abnormalities (Fig. 3), the clinical phenotype, inflammatory markers, and microbiological or serological evidence. In an older patient, giant cell arteritis remains an important inflammatory consideration [2,3], but reactive syphilis serology requires dedicated infectious-disease assessment before causality can be assigned.

This distinction is clinically important because treatment strategies differ fundamentally according to etiology. Immunosuppressive treatment for inflammatory large-vessel vasculitis may be inappropriate or potentially harmful when an active vascular infection has not been adequately excluded. Conversely, positive serology alone does not establish that syphilis is the cause of the aortic inflammation. Therapeutic decisions should therefore follow multidisciplinary assessment and a sufficiently complete etiological workup rather than PET/CT findings in isolation.

This case has important limitations. The definitive etiology remained unresolved because rapid clinical deterioration precluded temporal artery biopsy, further infectious-disease assessment, and longitudinal clinical or imaging follow-up. Although two treponemal tests were reactive, the nonreactive RPR/VDRL and the absence of treatment history or additional confirmatory assessment prevent attribution of the vascular inflammation to active syphilis. The imaging findings therefore support active large-vessel inflammation but not a specific infectious or inflammatory cause.

Conclusion

18F-FDG PET/CT can demonstrate the extent and activity of large-vessel inflammation but cannot reliably determine whether aortitis is inflammatory or infectious on the basis of uptake pattern alone. In this case, reactive syphilis serology discovered after PET/CT broadened the differential diagnosis and underscored the need for etiological correlation before disease-specific treatment. The key practical message is therefore one of diagnostic caution: marked vascular FDG uptake should prompt integration of imaging with clinical, serological, microbiological, and complementary anatomic findings, particularly before immunosuppressive therapy is initiated.

Author Contributions

- Mohammed Amine Biyi: Conceptualization, investigation, data curation, writing – original draft.
- Zakaria Ouassafrar: Methodology, supervision, formal analysis, visualization, writing – review and editing.
- Amal Guensi: Supervision, project administration, validation.

Declarations

Ethics and Declaration of Helsinki: The authors state that this case report was prepared in accordance with the ethical principles of the World Medical Association Declaration of Helsinki (2024 revision), including respect for the patient's dignity, privacy, and confidentiality, and with applicable local and institutional requirements.

ICMJE statement: The authors state that they meet the applicable authorship criteria of the International Committee of Medical Journal Editors (ICMJE), approved the final version for publication, and agree to be accountable for all aspects of the work, including the accuracy and integrity of its content.

Consent for publication: Written informed consent was obtained for publication of the patient's clinical information and images. Patient anonymity has been protected.

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Data availability: All data relevant to this case report are contained within the article. No separate research dataset was generated.

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