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CASE REPORT

Unusual [^{18}F]FDG Avidity in multiple major arteries and pulmonary trunk: A case report of active large-vessel vasculitis

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ABSTRACT

Large-vessel vasculitis (LVV), including Takayasu arteritis and giant cell arteritis, is a rare inflammatory condition affecting the aorta and its major branches. This case report describes a 33-year-old male with fever of unknown origin (FUO), anemia, and elevated inflammatory markers. Initial imaging was inconclusive, but Fluorodeoxyglucose Positron Emission Tomography - Computed Tomography ([^{18}F]FDG PET-CT) revealed diffuse uptake in major arteries (carotid, subclavian, brachiocephalic, aorta, and pulmonary trunk) with an SUVmax of 6.91, indicating active vasculitis. The absence of [^{18}F]FDG-avid lymphadenopathy ruled out malignancy or infection, while faint uptake in bilateral femoral arteries suggested systemic involvement. Physiological uptake in organs like the gastrointestinal tract and adrenal glands showed no malignancy, and axial skeleton activity was linked to anemia. This case highlights [^{18}F]FDG PET-CT's role in early LVV detection, stressing the need for timely diagnosis to prevent irreversible vascular damage and the importance of integrating imaging with clinical evaluation for effective management.

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INTRODUCTION

Vasculitis refers to a diverse group of disorders characterized by blood vessel inflammation, classified by vessel size: large-vessel vasculitis (LVV), such as Takayasu arteritis (TAK) and giant cell arteritis (GCA); medium-vessel vasculitis, like polyarteritis nodosa; and small-vessel vasculitis, such as ANCA-associated vasculitis [1]. Treatment typically involves corticosteroids and immunosuppressants, tailored to the type and severity of the condition. LVV, including TAK and GCA, affects the aorta and its major branches. TAK typically occurs in younger individuals, while GCA is more common in older adults and often involves extracranial branches [2]. [18F]FDG PET-CT in diagnosing and managing LVV, particularly in patients presenting with FUO [3]. It enables early detection of vascular inflammation before irreversible structural changes occur. This imaging modality has demonstrated high sensitivity for identifying active inflammation, making it a powerful tool for both diagnosis and monitoring therapeutic response [4]. The aim of this case report is to present a rare instance of increased [18F]FDG avidity in multiple major arteries and the pulmonary trunk, indicating active vasculitis, to contribute to the understanding and management of such vascular conditions.

CASE PRESENTATION

A 33-year-old male presenting with a FUO and a history of fatigue was evaluated. Laboratory investigations revealed anemia (Hb = 9.4 g/dL), elevated inflammatory markers (ESR: 72 mm/hr, CRP: 8 mg/L), and these findings could be suggestive of chronic inflammation or infection. Imaging studies prior to PET-CT included MRI of the lumbar spine, which demonstrated disc bulging at T12-L1 and L3-L4 levels, and a whole-body bone scan (WBBS) that showed degenerative joint disease (DJD). CT imaging of the chest, CT scans of the head, neck, brain, and abdominal-pelvic regions were unremarkable. Bone marrow aspiration (BMA) and biopsy (BMB) showed no abnormal findings.

The patient underwent PET/CT imaging following a standardized preparation protocol to ensure reproducibility and optimal image quality. He was confirmed non-diabetic and had fasted for at least six hours prior to tracer administration, with a measured blood glucose level of 88 mg/dL at the time of injection. A dose of 7 mCi (259 MBq) of [18F]FDG was administered intravenously, and imaging was initiated exactly 60 minutes post-injection, which aligns with current procedural guidelines for oncologic PET/CT. The scan was performed in 3D high-definition acquisition mode, from the vertex to mid-thigh, with an acquisition time of 3 minutes per bed position.

PET-CT imaging revealed diffuse [18F]FDG uptake in major large vessels, including the carotid, subclavian, and brachiocephalic arteries, as well as the aorta and pulmonary trunk (Figure 1), raising suspicion for large-vessel vasculitis or another systemic inflammatory condition. The SUVmax values recorded during the PET scan were notable, with a mediastinal blood pool SUVmax of 2.25 and a hepatic background SUVmax of 3.68. These values suggest a baseline metabolic activity that is crucial for interpreting the results in the context of the patient's clinical presentation.

The [18F]FDG PET/CT scan demonstrated diffuse physiologic brain uptake, which complicated the assessment of potential abnormal lesions due to high background activity, underscoring the need for correlation with brain MRI for definitive evaluation. No significant [18F]FDG-avid lymphadenopathy or abnormal uptake was observed in cervical regions, effectively ruling out malignancy or infectious processes in these areas. The thoracic region revealed markedly increased [18F]FDG activity in major arteries, including the bilateral carotid and subclavian arteries as well as the aorta and pulmonary trunk, with a maximum SUV of 6.91 - a finding highly suggestive of active vasculitis. This vascular inflammation pattern was further supported by faint [18F]FDG avidity in bilateral femoral arteries (SUVmax=2.74), indicating likely systemic vascular involvement. In the abdominopelvic region, imaging showed no remarkable abnormal uptake in the liver or spleen apart from small benign calcifications, while the gastrointestinal tract displayed only physiological uptake without evidence of significant lymphadenopathy or lesions in the intraperitoneal and retroperitoneal spaces. Physiological uptake was also noted in the urinary system and other abdominal viscera, including both adrenal glands where the observed activity may represent benign adrenal hyperplasia. The musculoskeletal evaluation demonstrated no focal abnormal uptake, though increased [18F]FDG activity throughout the axial and proximal appendicular skeleton was likely related to the patient's underlying anemia, reflecting systemic influences on metabolic imaging patterns.

DISCUSSION

LVV, including TAK and GCA, is a rare but significant cause of systemic inflammation and FUO. In the presented case, the diffuse [^{18}F]FDG uptake involving the carotid, subclavian, brachiocephalic arteries, aorta, pulmonary trunk, and femoral arteries, with a maximum standardized uptake value (SUVmax) of 6.91, raised strong suspicion for active vasculitis. The diagnostic role of [^{18}F]FDG PET/CT in LVV is well supported in the literature. It provides sensitive detection of vascular inflammation even before anatomical changes occur and is particularly useful when other imaging modalities are inconclusive [3, 4].

To interpret [^{18}F]FDG avidity, semi-quantitative thresholds are essential. Studies have shown that an SUVmax >2.0–2.5 relative to background liver uptake, or a vascular-to-liver SUV ratio >1.0, is suggestive of inflammation [5, 6]. In our patient, the aortic SUVmax (6.91) was notably higher than both the liver (SUVmax 3.68) and mediastinal blood pool (SUVmax 2.25), fulfilling these criteria. Moreover, the observed pattern of diffuse vascular uptake, including the pulmonary trunk, suggests widespread disease activity. While pulmonary artery involvement is rare, it has been reported in cases of TAK and granulomatous vasculitis [7]. Diffuse vascular involvement has been observed in up to 80% of patients with active LVV and is frequently associated with elevated inflammatory markers, such as ESR and CRP [6].

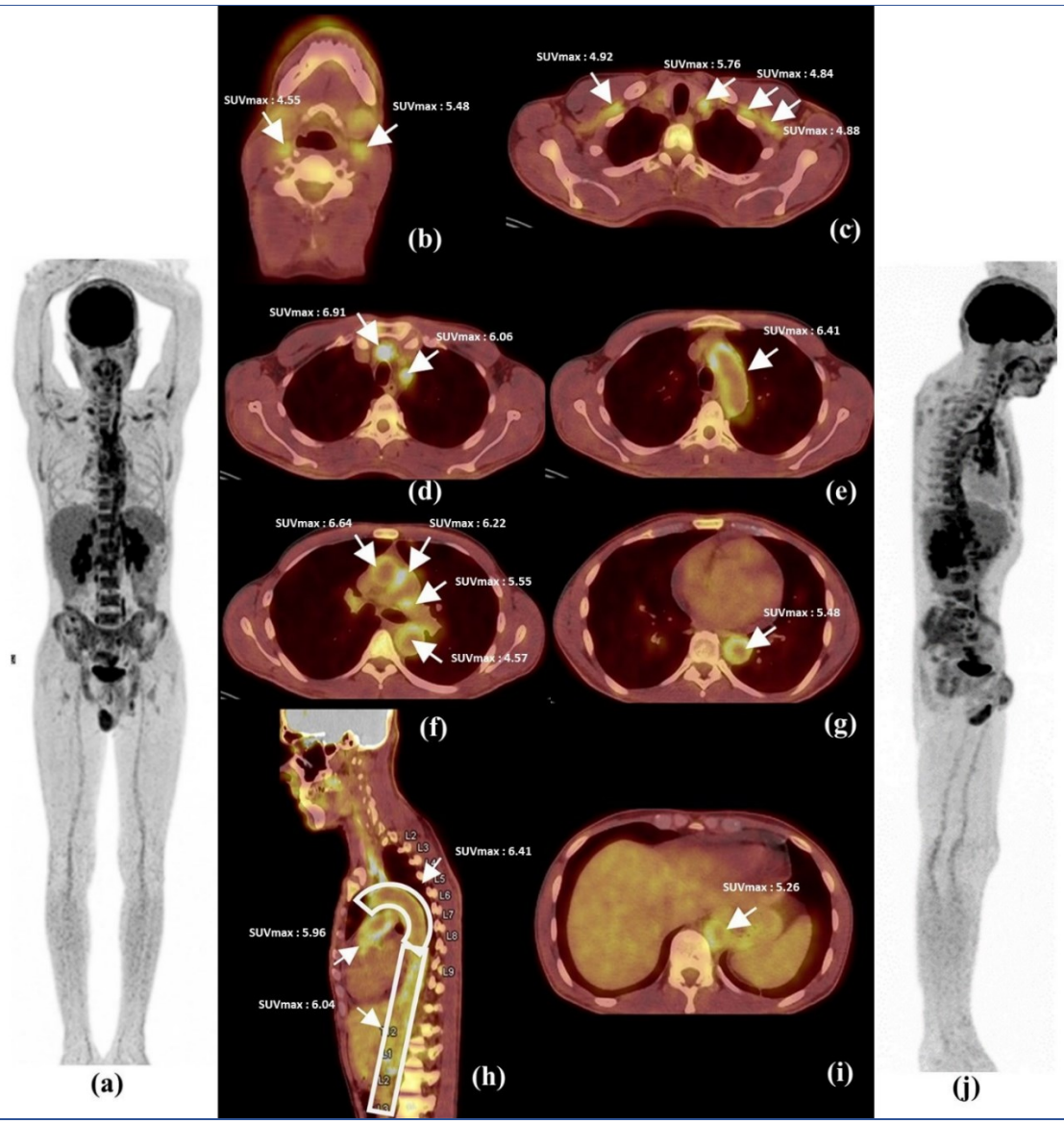

Figure 1. A widespread [^{18}F]FDG uptake in (a): Whole-body Maximum Intensity Projection (MIP), (b): Axial PET/CT-Upper neck, (c): Axial PET/CT-Thoracic inlet, (d): Axial PET/CT-Upper mediastinum, (e): Axial PET/CT-Aortic arch, (f): Axial PET/CT-Pulmonary trunk, (g): PET/CT-Upper abdomen, (h): Sagittal PET/CT-Abdominal aorta, (i): Axial PET/CT-Abdominal aorta, (j): 90-degree lateral view MIP

International guidelines from EULAR (European League Against Rheumatism) and SNMMI (the Society of Nuclear Medicine and Molecular Imaging) - EANM (the European Association of Nuclear Medicine) recommend [18F]FDG PET/CT as an appropriate imaging modality in patients with suspected LVV, particularly when other diagnostic methods yield inconclusive results [8]. The diagnostic hallmarks of LVV on [18F]FDG PET/CT include visual criteria such as increased uptake along the vessel wall that is equal to or greater than hepatic activity, typically classified as grade 2-3 [9]. Semiquantitatively, a vessel-to-liver SUV ratio of 1 or higher supports the diagnosis [10]. The pattern of uptake is also critical: smooth, linear, circumferential, and multicentric distribution strongly favors LVV, whereas patchy or focal uptake is more suggestive of atherosclerosis [11]. Importantly, PET findings must always be interpreted in conjunction with the patient's clinical presentation and laboratory markers of inflammation to ensure accurate diagnosis and appropriate management [8]. This case clearly met these criteria, reinforcing PET/CT's pivotal role when conventional imaging was inconclusive.

Differential diagnoses of increased vascular [18F]FDG uptake must be considered. Atherosclerosis often presents as focal or heterogeneous uptake of lower intensity and usually occurs without systemic inflammatory markers [12]. Infectious aortitis typically shows focal uptake with adjacent periaortic soft tissue changes and is associated with positive blood cultures [13]. Other mimics include sarcoidosis and Behçet's disease, which generally show additional systemic [14] or mucocutaneous findings, and paraneoplastic vasculitis, which often coincides with [18F]FDG-avid malignancies [6]. Therefore, [18F]FDG PET/CT findings must be interpreted alongside clinical data and laboratory results to ensure diagnostic accuracy.

This case has several limitations. Histopathological confirmation via biopsy was not obtained, and while [18F]FDG PET/CT is highly sensitive, its specificity relies on clinical context. No follow-up imaging was available to assess treatment response, and corticosteroid therapy prior to scanning may have reduced [18F]FDG uptake, potentially underestimating disease extent. Additionally, the absence of vessel wall thickening on CT angiography limited anatomical correlation. Despite these constraints, the imaging and clinical presentation strongly support LVV, demonstrating PET/CT's utility in diagnosing complex cases of systemic inflammation.

CONCLUSION

In conclusion, our case highlights the significance of [18F]FDG PET imaging in detecting early inflammatory changes associated with large-vessel vasculitis before irreversible vascular damage takes place. These findings emphasize the need for prompt diagnosis and intervention to prevent complications. Furthermore, integrating these results with other imaging techniques and clinical assessments is crucial for confirming the diagnosis and optimizing management strategies.

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