

18F-FDG PET finding of an inflammatory abdominal aortic aneurysm leading to fatal rupture: case report

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Introduction

Fluorine-18-fluorodeoxyglucose positron emission tomography (18F-FDG PET) is routinely used mainly for diagnostics and monitoring of many oncological diseases. However, fluorine-18-fluorodeoxyglucose as a radiopharmaceutical is not an exclusively tumour-specific tracer and numerous inflammatory diseases may be evaluated as well. An inflammatory lesion showing high 18F-FDG accumulation is likely to have high metabolic activity.

Herein we report a case of a man, who presented with low-back pain and increased acute-phase proteins due to inflammatory abdominal aortic aneurysm.

Case report

We present the case of a polymorbid 55-year-old male with medical history of arterial hypertension, chronic obstructive bron-

chopulmonary disease, ischemic disease of the lower extremities, peptic ulcer disease, status post left femoral popliteal bypass 3 years ago and status post benign tumour (chondroid hamartoma) resection of the left lung. The patient complained of low-back pain and weight loss of 15 kg in the past year. Laboratory tests showed C-reactive protein elevation (81 mg/l) as well as elevated erythrocyte sedimentation rate (45 mm/h) and CEA (carcinoembryonic antigen) levels (3.7 µg/l), without further biochemical or haematological pathologies. Previously performed examinations failed to reveal a clear aetiology (gastroscopy and colonoscopy showed bulbitis and internal haemorrhoids, respectively).

Therefore, the patient was indicated for 18F-FDG PET examination to exclude occult malignancy (elevation of inflammatory markers, CEA tumour marker and presence of cachectization). We performed 18F-FDG PET scanning in euglycaemia after fasting for 6 hours and oral hydration. 18F-FDG PET scan images of the body, at the extent from the proximal thirds of femurs to the cranial base, were obtained 60 minutes after intravenous injection of 18F-FDG (341 MBq). Data acquisition was performed on a PET scanner ECAT ACCEL SIEMENS in a 3D mode. Emission and transmission scans were reconstructed using an iterative reconstruction algorithm. On acquired data, attenuation correction was applied. For semiquantitative analysis, maximum standardized uptake values (SUV_{max}) were measured. Finally, off-line image fusion of PET and computed tomography (CT) data sets was performed.

The 18F-FDG PET scan detected high 18F-FDG uptake (SUV_{max} = 4.3) circularly around the distal abdominal aorta and left common iliac artery (Figure 1). Further sites of pathological 18F-FDG uptake were not found. On the fusion PET/CT scan these sites were precisely localised into the abdominal aorta wall, left common iliac artery and also paraortic and parailiacally. The central photopenic (non-active) area corresponded with the artery lumen (Figure 2). Thus, the finding proved a metabolic active inflammation — periaortitis.

Unfortunately, soon after the PET examination the patient died unexpectedly. Autopsy determined the cause of death as fatal aneurysm rupture of the distal part of abdominal aorta leading to a haemorrhagic shock. No malignancy was revealed.

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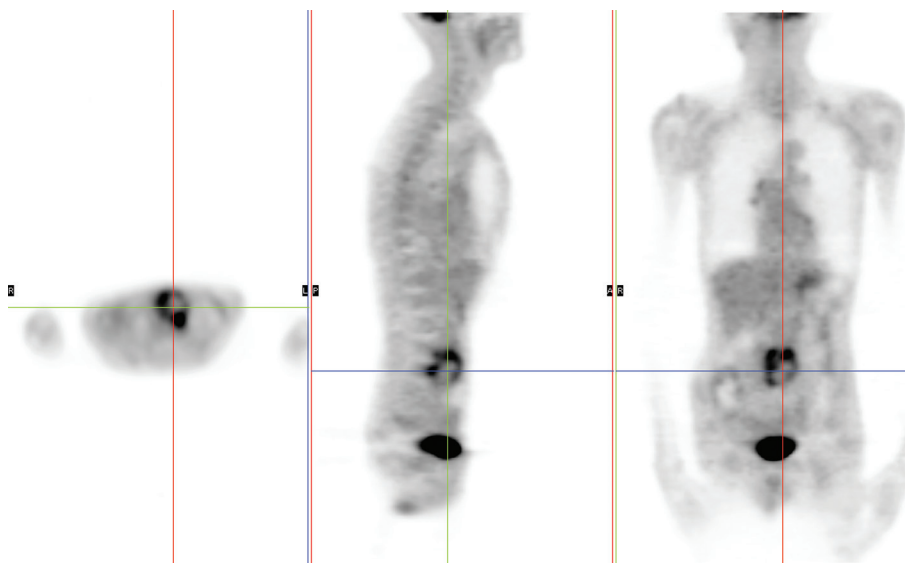


Figure 1. 18F-FDG PET: High FDG uptake in the distal abdominal aorta wall, aortic bifurcation, paraaortically and left parailiacally, without further pathological FDG uptake



Figure 2. CT scan: aneurysm of the abdominal aorta at its bifurcation (the black arrow) extending up to the common iliac artery. Around there is an inflammatory abscess formation with pyogenic membrane (the white arrows) propagating in the vicinity

Discussion

Not only malignancies may be the cause of pathological 18F-FDG PET finding, but also some inflammatory diseases [1]. 18F-FDG uptake is not specific for tumours. For instance, in large vessel vasculitis (LVV) PET examination is used not only in primary diagnostics, but for inflammation activity monitoring during therapy as well. Concerning LVV, increased glucose metabolism proves active inflammation [2]. Sometimes even inflammatory infiltrate of an atherosclerotic plaque may lead to PET positivity; it is supposed that activated macrophages are mainly responsible for increased 18F-FDG uptake [3, 4].

Chronic periaortitis (including 3 entities: idiopathic retroperitoneal fibrosis - IRF, inflammatory abdominal aortic aneurysms — IAAA and perianeurysmal retroperitoneal fibrosis — PRF) may also be manifested in an increased 18F-FDG uptake in concentric wall thickening with a perivascular cuff around the abdominal aorta.

This disease is frequently accompanied by low-back pain, weight loss, anaemia, fatigue, high erythrocyte sedimentation rate and elevated C-reactive protein levels [5–7]. In patients with IRF, decrease in metabolic activity of the inflammation in correlation with erythrocyte sedimentation rate, C-reactive protein and a CT documented thickening was described and documented in a series of 18F-FDG PET scans during therapy with corticosteroids or tamoxifen [8, 9].

In some patients with IAAA, correlation between periaortic 18F-FDG uptake and intensity of inflammatory cell infiltration was found [10]. The process seems to depend upon the release of matrix metalloproteinases focally produced and/or activated by inflammatory cells, which leads to degradation of elastin and collagen in the aneurysm wall. Increased metabolic activity in the wall of the IAAA on PET scan reflects increased rupture risk better than aneurysm size [11, 12]. Also another case report found a potential link between high wall stress and accelerated metabolism in aortic aneurysm wall [13].

Unfortunately, in our patient the inflammation of artery wall aneurysm, accompanied by increased 18F-FDG uptake, led as far as to fatal rupture.

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