

CLINICAL VIGNETTE

doi:10.1093/eurheartj/ehm539

Visualization of pericarditis with fluoro-deoxy-glucose-positron emission tomography/computed tomography

Klaus Strobel^{1*}, Roland Schuler², and Michele Genoni³

¹Division of Nuclear Medicine, Department of Medical Radiology, University Hospital Zurich, Raemistr. 100, 8091 Zurich, Switzerland; ²Institute of Clinical Pathology, Department of Pathology, University Hospital Zurich, Switzerland; and ³Department of Cardiovascular Surgery, University Hospital Zurich, Switzerland

* Corresponding author. Tel: +41 44 255 28 50, Fax: +41 44 255 44 14. Email: klaus.strobel@usz.ch

A 38-year-old man presented with recurrent fever, night sweat, retrosternal pain, and vomiting. Laboratory test failed to identify a viral or bacterial agent. The patient did not respond to antibiotics. Transoesophageal echocardiography showed pericardial effusion and no valvular vegetations. The patient was referred to fluoro-deoxy-glucose-positron emission tomography/computed tomography (FDG-PET/CT) to identify an infectious focus. Whole-body FDG-PET/CT was performed after fasting and 1 hour after intravenous administration of 350 MBq FDG. Coronal PET image (Panel D) demonstrates increased uptake in the whole pericardium, pronounced on the right side (long arrows). PET/CT technique provides cross-sectional axial CT- (Panel A), PET- (Panel B), and fused PET/CT- (Panel C) images. The pericardial layers were thickened and took up FDG. Between the pericardial layers effusion was visible. Additionally, small pleural effusion was detected on the right side (short arrows). Pericardial biopsy was performed. The histopathological examination showed fibrosis with chronic sclerosing and lymphoplasmacellular inflammation consistent with chronic sclerosing pericarditis (Panel E). A specific aetiological lesion (e.g. granulomas, rheumatoid nodules) could not be identified. Furthermore, no causing agent could be identified on specially stained histological sections, in microbiology or with PCR. A therapy with corticosteroids was not successful and 9 months after the biopsy, a pericardectomy was necessary because of the pericardial constriction.

Although the majority of patients imaged with FDG-PET/CT suffer from malignant diseases, this radionuclide is not tumour-specific. It has been proven experimentally that inflammatory cells also take up FDG. The most experience with FDG in infection and inflammation imaging exists in patients with fever of unknown origin and osteomyelitis. FDG-PET/(CT) has been used in the cardiovascular area for imaging of large vessel vasculitis (Takayasu or giant cell arteritis) and graft infections. Because there can be markedly physiological uptake of FDG in the heart, especially in the left ventricle myocardium, it is crucial to differentiate pathological FDG-uptake due to pericarditis from physiological uptake. That is why patient preparation for imaging of inflammatory or infectious heart diseases with FDG differs from preparation for myocardial viability studies. In infection indications fasting for at least 4 h should guarantee energy supply of the myocardium by fatty acids. In FDG-viability studies, the combination of glucose and insulin administration before FDG-injection leads to FDG-uptake by switching the energy supply from fatty acids to glucose. Our case demonstrates that it is possible to visualize a chronically active pericarditis with FDG-PET/CT. Of course, FDG-PET/CT should not be the first imaging tool for patients with the suspicion for pericarditis but in unclear cases it may help prove the diagnosis, to show the extent of the disease, to exclude other infectious foci, to guide the biopsy, and it might be useful for therapy response assessment.

Panel A. Axial low-dose CT image showing the thickened pericardial layers (long arrow) with pericardial effusion in-between and pleural effusion (short arrow).

Panel B. Corresponding axial positron emission tomography image with increased FDG-uptake in the pericardium (arrow)

Panel C. Fusion of corresponding CT and positron emission tomography image illustrating that the increased FDG-uptake matches with the pericardium.

Panel D. Coronal MIP (maximum intensity projection) image with increased FDG-uptake in the pericardium (arrow).

Panel E. Haematoxylin-eosin stained histologic section (200 ×) showing fibrotic soft tissue with a chronic lympho-plasmacellular inflammatory infiltrate interspersed between thick collagenous bundles.

