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CASE IN POINT

A PET/CT-follow-up imaging study to differentiate takotsubo cardiomyopathy from acute myocardial infarction

Jelena R. Ghadri · Svetlana Dougoud · Willibald Maier · Philipp A. Kaufmann · Oliver Gaemperli · Abhiram Prasad · Thomas F. Lüscher · Christian Templin

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Abstract Takotsubo cardiomyopathy (TTC) is still an under-recognized disease and little data exists on the coexistence of TTC and obstructive coronary artery disease. Our patient case of an 80-year-old female lady highlights the impact of a positron emission tomography/computed tomography (PET/CT) follow-up imaging study to delineate this unique entity from acute coronary syndrome (ACS). Furthermore, we show for the first time that coronary flow reserve and myocardial blood flow is globally impaired in TTC and not only restricted to the non-contracting parts. This indicates a global microcirculatory impairment effect of the heart in the acute stage of TTC. Our case also demonstrates that a transient metabolic defect is also involved in this disease. Follow-up imaging by PET/CT in our patient case unmasked TTC and facilitated to exclude the differential diagnosis of ACS.

J. R. Ghadri · W. Maier · O. Gaemperli · T. F. Lüscher · C. Templin (✉)
Department of Cardiology, Cardiovascular Center, University Hospital Zurich, Ramistrasse 100, D-Rämi, 8091 Zurich, Switzerland
e-mail: christian.templin@usz.ch

J. R. Ghadri · S. Dougoud · P. A. Kaufmann · O. Gaemperli
Cardiac Imaging Divison, Department of Radiology, University Hospital Zurich, Zurich, Switzerland

P. A. Kaufmann · T. F. Lüscher
Center for Integrative Human Physiology (ZIHP), University of Zurich, Zurich, Switzerland

A. Prasad
Division of Cardiovascular Diseases, Mayo Clinic Rochester, Rochester, MN, USA

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Case report

An 80-year-old female patient was admitted to the emergency room with acute chest pain and ST-segment elevation. Emergency coronary angiography revealed a single 70–90 % stenosis of the mid left anterior descending (LAD) artery with TIMI III flow (Fig. 1a). The left ventricular (LV) angiogram showed a large akinesia of the apex involving the LV mid segments extending way beyond the myocardial territory supplied by the LAD and a hyperdynamic contraction of the LV base (Fig. 1a). LV-function was markedly reduced (35 %). The patient underwent coronary stenting of the LAD lesion. Echocardiography study on the next day revealed a fast recovery of LV ejection fraction (52 %); however, regional wall motion abnormalities were still present. An in-depth history of the patient's emotional state disclosed that a sudden illness of her husband's with the diagnosis of a carcinoma was identified as her emotional stressor. To differentiate ACS from takotsubo cardiomyopathy (TTC) a combined perfusion/metabolism positron emission tomography/computed tomography (PET/CT) study using ^{13}N-ammonia (^{13}N-NH_3) and ^{18}F-fluorodeoxyglucose (FDG) was performed on the 7th post-interventional day and at follow-up 3 months later. At baseline, the ^{13}N-ammonia-PET/CT study demonstrated an extensive area of decreased tracer uptake in the apex and midventricular segments without reversibility during stress (adenosine) and a congruent defect on FDG PET study (Fig. 1b). Hyperemic myocardial blood flow (MBF) and coronary flow reserve (CFR) were globally decreased in the apical, midventricular and basal

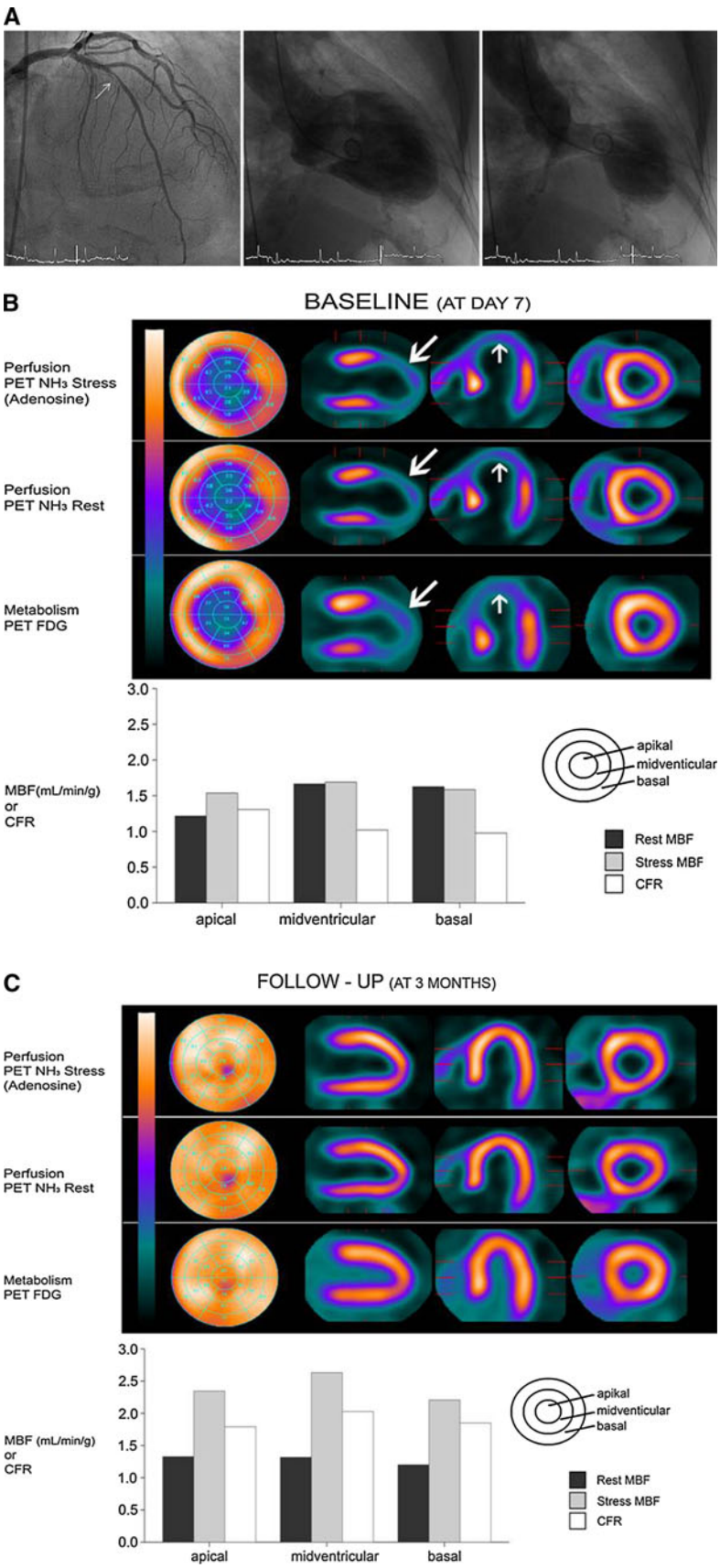

Fig. 1 **a** Coronary angiography demonstrating a 70–90 % stenosis of the mid left anterior descending artery (LAD) (*arrow*) and left ventricular angiography showing apical ballooning pattern. **b** ^{13}N-ammonia-positron-emission-tomography/computed-tomography (PET/CT) during pharmacological stress (*upper row*), at rest (*middle row*), and ^{18}F-fluorodeoxyglucose (FDG) PET/CT (*lower row*) Baseline Studies performed 7 days after index event demonstrate a large perfusion defect involving the apex and midventricular segments without reversibility during stress (*arrows*) and a matching defect on metabolic study with FDG (*arrows*). Total Myocardial blood flow (MBF) at rest was elevated (1.5 mL/min/g) with minimal increase during pharmacological stress (1.6 mL/min/g) and severely reduced CFR. MBF abnormalities affected similarly apical, midventricular and basal segments. **c** At 3 months follow-up, the combined PET/CT studies showed full recovery of the perfusion defect on ^{13}N-NH_3-PET (*upper and middle row*) and metabolic defect on FDG-PET (*lower row*). Resting MBF (1.3 mL/min/g) was lower and hyperemic MBF (2.3 mL/min/g) returned to normal values. Similarly, CFR was increased significantly compared to baseline values

segments of the LV myocardium (Fig. 1B). The matched FDG-PET/CT finding, as well as the globally impaired hyperemic MBF and CFR, did not correspond to the territory of a single coronary artery distribution. The PET/CT-follow-up-study at 3 month documented full recovery (Fig. 1c). Therefore, complete reversibility of the perfusion, microcirculatory and metabolic defect suggests that an acute anterior myocardial infarction due to the LAD stenosis was unlikely the underlying cause of the large akinesia of the apical and midventricular segments, but that it rather represents TTC.

One proposed mechanism in TTC encompasses the catecholamine spillover in which a direct cell toxic effect is suggested through the cyclic-adenosine-monophosphate-mediated calcium overload which decreases myocyte viability [1]. The reduced FDG tracer uptake in the acute phase of TTC has been referred to as the "metabolic trapping effect" by which glucose is transiently trapped in the cardiac tissue without further metabolism [2]. In this regard, it has been demonstrated that catecholamines stimulates the expression of cardiac glucose transporters through an alpha-1adrenergic receptor mediated mechanism in isolated rat cardiomyocytes [3]. However, the exact mechanism remains unclear, although a, catecholamine-induced microcirculatory dysfunction has also been suggested as a potential cause [4]. In fact in our study we observed a perfusion and matched metabolic defect in the concordant non contracting myocardial parts at baseline, while CFR and MBF were globally impaired and not solely restricted to the dysfunctional LV myocardium. Our findings indicate that microcirculatory dysfunction is a global phenomenon in TTC, though more severe in regions with contractile dysfunction. In contrast, the glucose dysmetabolism was limited to the morphologic dysfunctional myocardium.

Whether this contributes to the pathophysiology or whether this is a secondary cause of TTC, this needs to be elucidated.

Our study highlights the utility of PET/CT follow-up imaging to delineate the unique entity of TTC from myocardial infarction in patients with obstructive coronary artery disease. Furthermore, we would like to emphasize that—contrary to popular belief—TTC can infrequently co-exist with obstructive CAD, as acknowledged in the revised Mayo Clinic diagnostic criteria. Thus, we speculate that myocardial infarction may be misdiagnosed in patients with TTC who have concomitant obstructive CAD stenosis in the LAD.

Conflict of interest None.

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