

IMAGES AND CASE REPORTS IN HEART FAILURE

Serial Quantitative Assessment of Myocardial Blood Flow With ¹³N-Ammonia Positron Emission Tomography in a Symptomatic Patient With Tachycardia-Induced Cardiomyopathy

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Tachycardia-induced cardiomyopathy (TIC) is a systolic or diastolic ventricular dysfunction resulting from prolonged elevated heart rate, often leading to heart failure. TIC was considered reversible in achieving heart rate control; however, reversal may not occur or be complete.¹ TIC causes coronary microvascular dysfunction and is associated with significant structural alterations suggestive of myocyte injury.² However, evidence of the time-course changes of microcirculation from acute to recovery phases of TIC is limited. We report the serial quantitative assessments of myocardial blood flow (MBF) and myocardial flow reserve (MFR), assessed using myocardial perfusion positron emission tomography (PET) in a patient with TIC.

CASE PRESENTATION

A previously healthy 74-year-old man presented with recurrent palpitations and progressive exertional dyspnea for 3 months. On admission, his blood pressure was 131/81 mmHg, heart rate 122 bpm, body temperature 36.7 °C, and respiratory rate 14/min, with 99% oxygen saturation on ambient air. A 12-lead ECG showed regular narrow supraventricular tachycardia (Figure 1A). Chest radiography revealed slight cardiac enlargement. Transthoracic echocardiogram revealed left ventricular (LV) ejection fraction (LVEF) of 35%

to 40% with a mildly enlarged LV cavity (LV end-diastolic dimension, 53 mm), no evident LV hypertrophy, and normal valve function. The complete blood count and metabolic profile were normal, except for increased NT-pro-BNP (N-terminal pro-B-type natriuretic peptide) levels (2078 pg/mL; normal <125). After careful ECG analysis, atrial tachycardia was suspected. He was hospitalized for worsening heart failure 10 days after beta blocker and oral anticoagulant administration. On day 2 of hospitalization, electrical cardioversion was successfully performed for sinus rhythm restoration (Figure 1B). Coronary computed tomography angiography showed no stenotic lesions. Cardiac magnetic resonance showed reduced LVEF of 39% with significant LV enlargement where a mid-wall late gadolinium enhancement was present in the basal septum (Figure 2A). On day 4 of hospitalization, pharmacological stress/rest ¹³N-ammonia PET using ATP infusion showed normal findings on myocardial perfusion imaging (Figure 3A) but severely reduced MFR (0.89) and stress MBF (0.62 mL/[g·min]; Figure 4A). The patient became asymptomatic after electrical cardioversion and was administered oral amiodarone at discharge. Two months after the initial presentation, he was asymptomatic with ECG showing sinus rhythm, transthoracic echocardiogram showing mild LV hypertrophy, and improved LVEF of 58%. The clinical course confirmed

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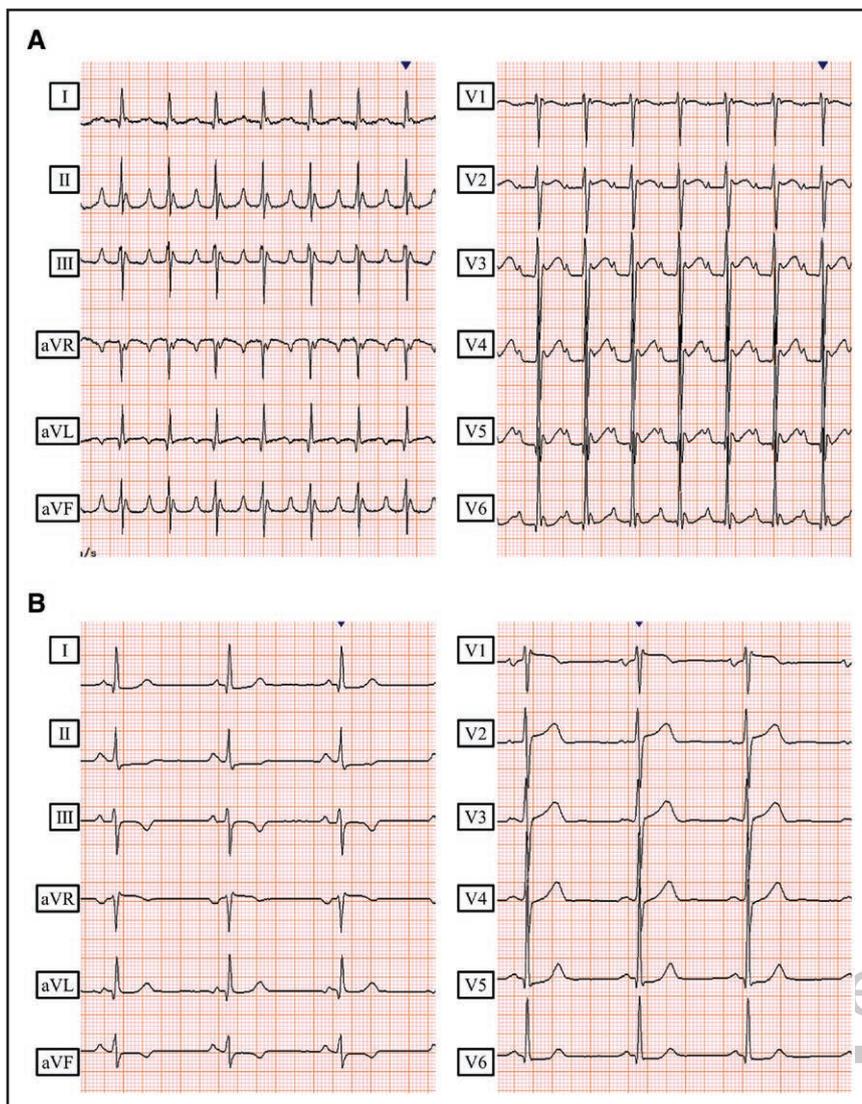


Figure 1. ECG findings.

Initial 12-lead ECG before (A) and after (B) successful electrical cardioversion.



the definitive diagnosis of TIC per a common definition.³ At the 12-month follow-up, he was asymptomatic with sinus rhythm, mild LV hypertrophy, and preserved LVEF at 70%, assessed by transthoracic echocardiogram. However, repeated PET (Figure 4B) revealed moderately reduced stress MBF (1.33 mL/[g·min]) and MFR (1.97) with normal findings on myocardial perfusion imaging (Figure 3B). In contrast, cardiac magnetic resonance (Figure 2B) demonstrated marked LVEF improvement at 69% with a normalized LV size and late gadolinium enhancement in the same basal septum. At the 18-month follow-up, he was asymptomatic; ECG showed sinus rhythm, and transthoracic echocardiogram demonstrated a preserved LVEF at 76% with mild LV hypertrophy. Follow-up PET (Figure 4C) revealed further improvement in stress MBF and MFR values (2.16 mL/[g·min] and 2.48, respectively), with near-normal myocardial perfusion imaging findings (Figure 3C). The patient was recommended to undergo genetic testing; however, the proposal was declined for economic reasons.

DISCUSSION

This case illustrates that coronary circulation impairment could be crucial in TIC development. Abnormal tachycardia increases myocardial oxygen consumption and decreases diastolic duration, increasing the susceptibility to myocardial ischemia.⁴ Chronic supraventricular tachycardia could lead to myocardial structure and function changes through reduced myocardial capillaries and MBF, increased capillary-myocyte distance and coronary vascular resistance, and impaired MFR, resulting in myocardial injury and LV dysfunction.^{1,5} These findings were consistent with ours, where stress MBF and MFR were critically reduced in the acute phase. Additionally, the patient showed evidence of fibrotic changes in the myocardium on cardiac magnetic resonance images, which is uncommon and suggestive of a worse prognosis in TIC.⁶ One of the TIC hallmarks is the reversibility of LV dysfunction. The patient showed moderately reduced stress MBF and MFR at the 12-month follow-up PET, and both parameters were almost normal

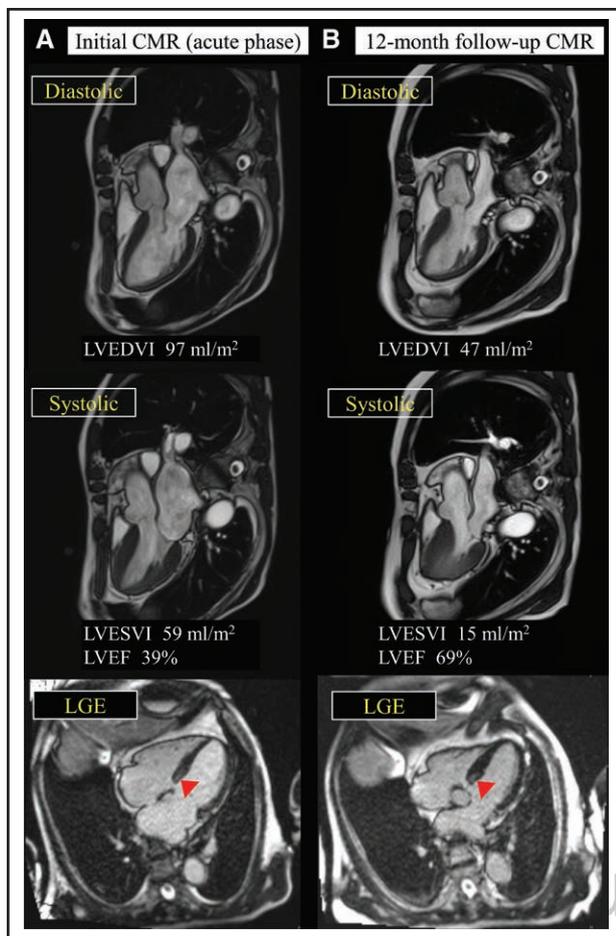


Figure 2. Cardiovascular magnetic resonance findings. Vertical long axis in diastolic (top) and systolic (middle) cine cardiovascular magnetic resonance (CMR) images with late gadolinium enhancement (LGE; bottom) 16 days (A) and 12 months (B) after initial presentation. In both cases, LGE (red arrowheads) is present in the basal septum. LVEDVI indicates indexed left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; and LVESVI, indexed left ventricular end-systolic volume.

at the 18-month follow-up PET. Accordingly, our case suggested that microscopic abnormalities, reflected by reduced stress MBF and MFR, may reverse more slowly than improvement at the clinical level. Similarly, MBF at rest increased from the initial PET to the 18-month follow-up PET. Therefore, we could speculate that the serial changes in rest MBF might reflect the long-term improvement in coronary microvascular function and the myocardial changes in hemodynamic, structural, cellular, and neurohormonal levels.¹ Supporting these notions of slow or incomplete recovery, a previous study using

echocardiographic assessments suggested that negative LV remodeling in patients with TIC persists long after tachycardia resolution at a mean follow-up of 14 months.⁷ Furthermore, recurrent tachycardia in patients with a prior TIC history could lead to recurrent cardiomyopathy at a faster and more severe rate, alarming a potential risk of sudden death.⁸

This is the first report outlining the serial PET and cardiac magnetic resonance findings from acute to recovery phases beyond 1 year in a patient developing TIC. Our findings may elucidate the potential mechanism of TIC and serial changes in micro and macro levels associated with coronary circulation.

ARTICLE INFORMATION

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Disclosures

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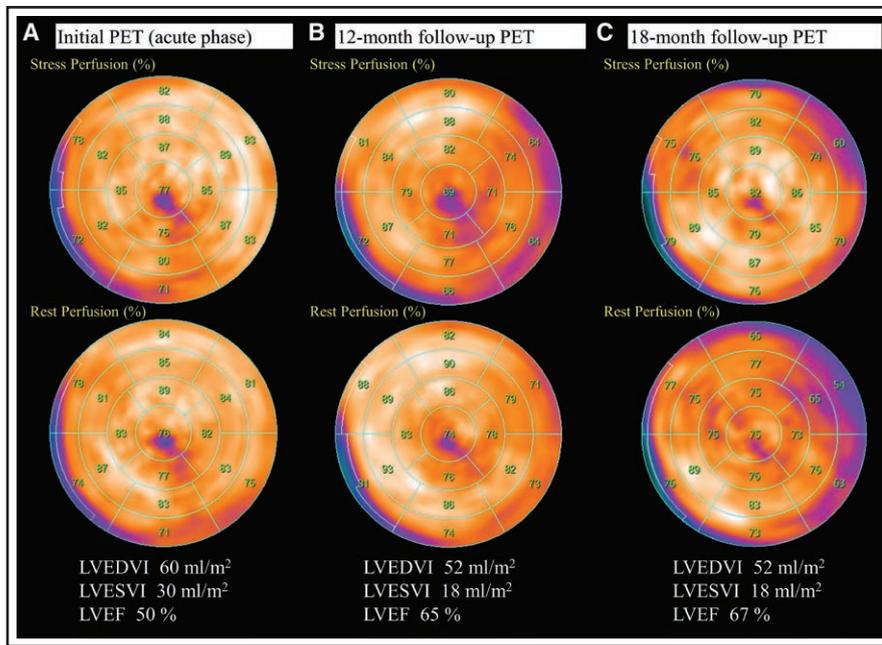


Figure 3. Myocardial perfusion imaging assessed using positron emission tomography.

Polar maps of qualitative myocardial perfusion imaging from stress/rest ¹³N-ammonia positron emission tomography (PET) with parameters (rest) on left ventricular function 13 days (A), 12 months (B), and 18 months (C) after initial presentation. LVEDVI indicates indexed left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; and LVESVI, indexed left ventricular end-systolic volume.

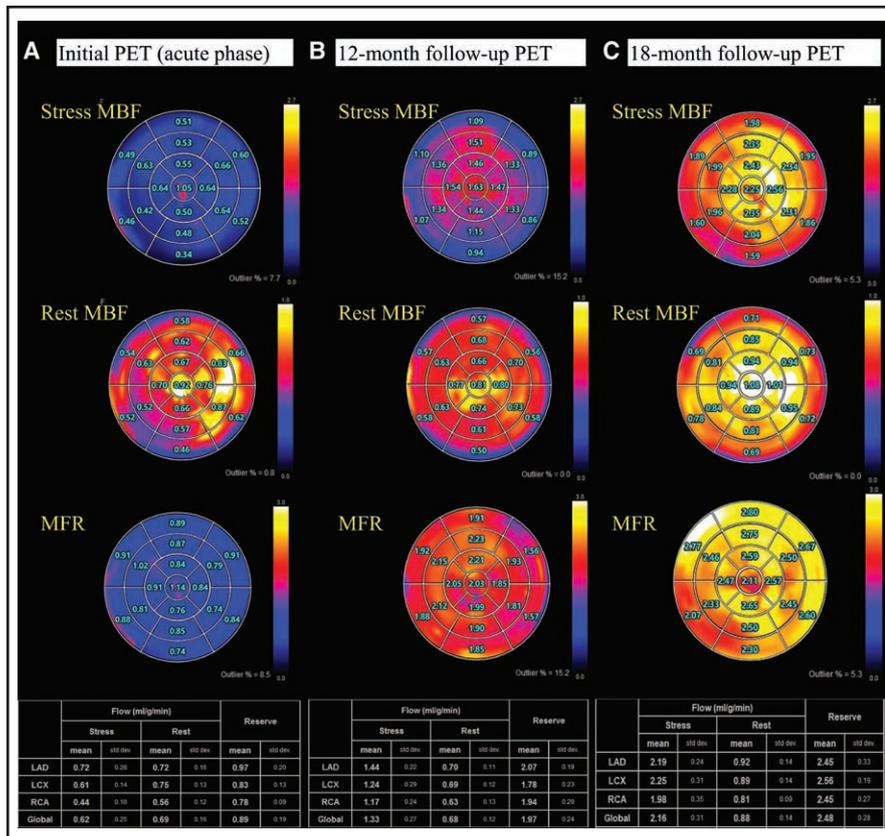


Figure 4. Myocardial blood flow assessed using positron emission tomography.

Quantitative assessment of myocardial perfusion with ¹³N-ammonia positron emission tomography (PET) 13 days (A), 12 months (B), and 18 months (C) after initial presentation. Significant improvement is observed for stress myocardial blood flow (MBF) and myocardial flow reserve (MFR) from the acute phase to the 12-month follow-up, with further improvement in both parameters at the 18-month follow-up PET. LAD indicates left anterior descending artery; LCX, left circumflex artery; and RCA, right coronary artery.