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Minimized myocardial infarction despite left main coronary artery thrombotic occlusion in a patient with left ventricular assist device

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A 53-year-old man was implanted with a left ventricular assist device (LVAD, HeartMate 3) for refractory heart failure due to dilated cardiomyopathy. He had a history of implantable cardioverter-defibrillator implantation for symptomatic ventricular tachycardia 2 years earlier. On post-operative day 7, he complained of malaise without chest pain or dyspnoea. Although electrocardiogram

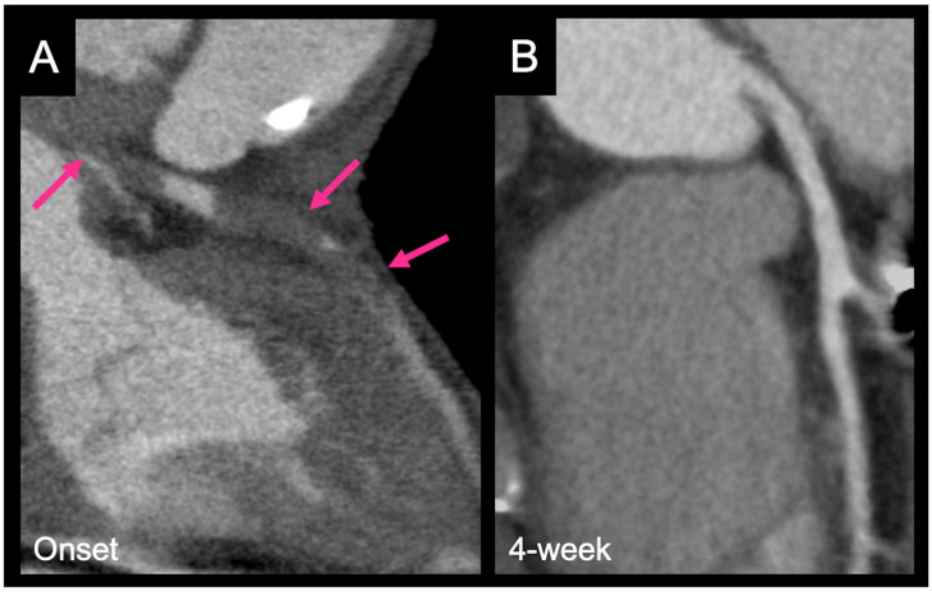

Figure 1 Serial images of coronary computed tomography angiography. (*A*) A large aortic root thrombus extending into the left main coronary artery and its anterior descending branch is noticeable (arrows). (*B*) The thrombi were completely disappeared 4 weeks after the onset.

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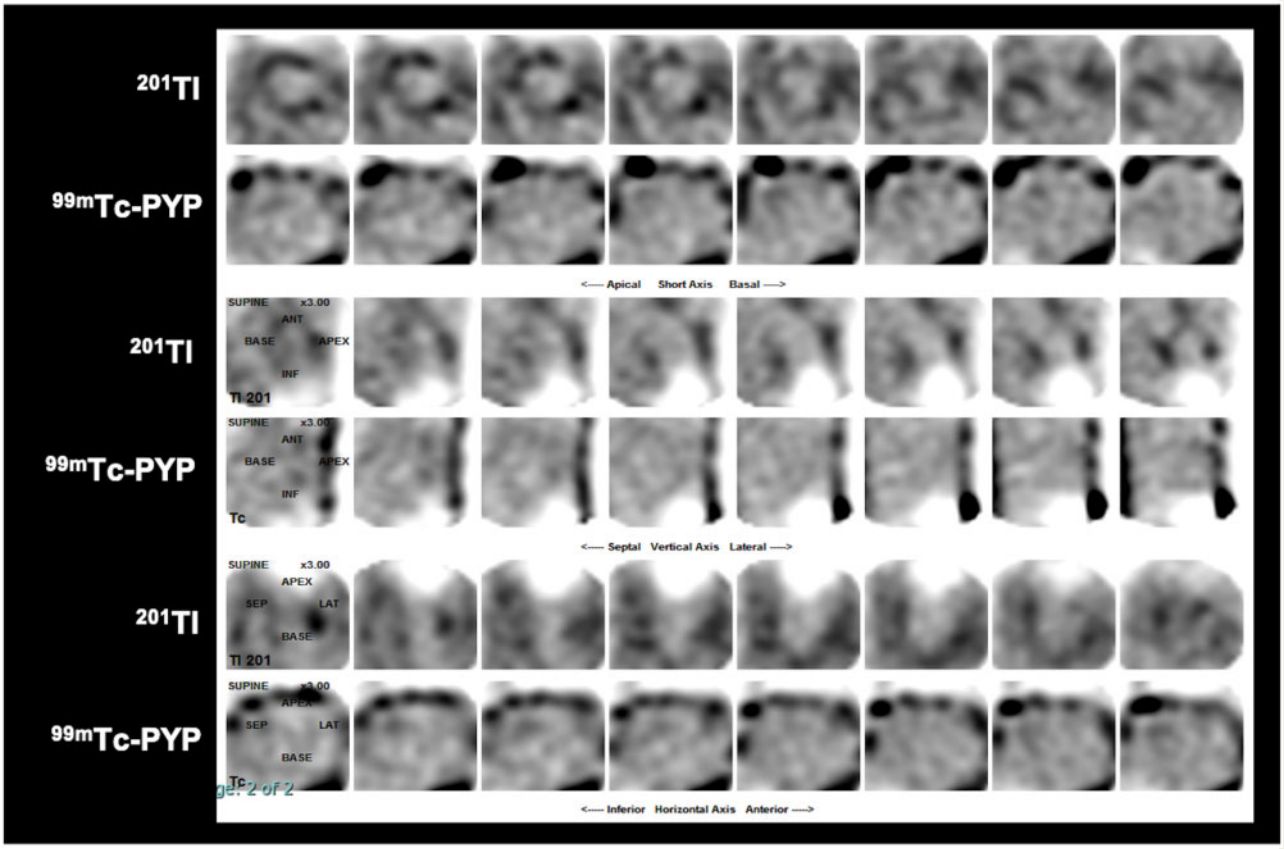
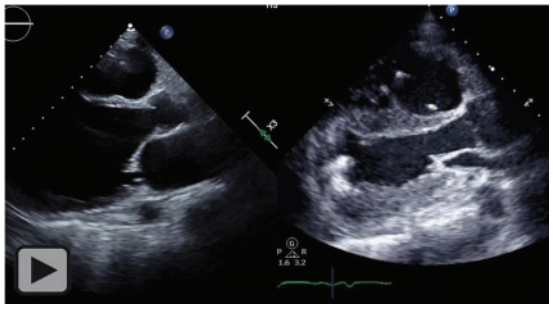
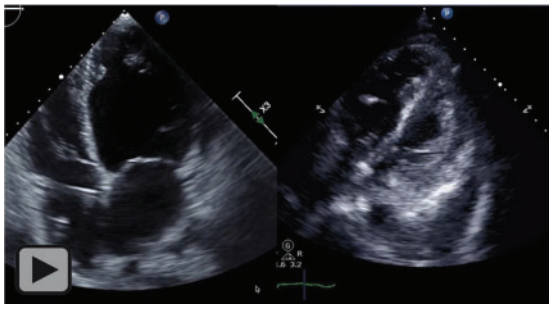


Figure 2 Myocardial single-photon emission computed tomography scans with ^{201}TlCl and technetium-99m pyrophosphate tracers. No apparent uptake of pyrophosphate suggesting infarct area was detected.



Video 1 The parasternal long-axis views of transthoracic echocardiography before (left) and after (right) implantation of LVAD.



Video 2 The apical four-chamber views of transthoracic echocardiography before (left) and after (right) implantation of LVAD.

showed no apparent ischaemic changes (Supplementary material online, *Figure S1*), myocardial biomarkers mildly rose; creatine kinase (CK) up to 1205 U/L, CK-MB 236 U/mL, troponin T 3.921 ng/mL. Echocardiography demonstrated thrombus formation (17 mm × 21 mm × 30 mm) in the left coronary cusp of the aortic valve (Supplementary material online, *Figure S2*) despite therapeutic anticoagulation with heparin (activated partial thromboplastin time, aPTT, 50–60 s) and antiplatelet therapy with aspirin. Coronary computed tomography angiography revealed a large thrombus in the aortic root occluding the left main coronary artery (LMCA) and anterior descending branch (*Figure 1*). There were no signs of LVAD pump thrombosis. Considering the haemodynamic stability without signs of heart failure under LVAD support and bleeding risk, conservative strategy by intensification of anticoagulation regimen (target aPTT of

80 s) was adopted instead of surgical thrombectomy with coronary artery bypass grafting, or thrombolytic therapy. The percutaneous coronary intervention was also deferred because of thromboembolic risk during the procedure. The LVAD settings were not changed considering a risk of thromboembolism due to abrupt resumption of aortic valve leaflet motion. Since LVAD did not allow cardiac magnetic resonance imaging, the infarct area was evaluated by technetium-99m pyrophosphate myocardial scintigraphy, which detected no significant uptake (*Figure 2*). The thrombi diminished with time and finally disappeared in 4 weeks without any evidence of peripheral thromboembolism. In the present case, lacking in aortic valve opening and subsequent blood stasis likely led to aortic root thrombosis and myocardial infarction.[1,2] Despite total occlusion of LMCA, which is potentially lethal, the resultant myocardial infarction was minimal by effective left ventricular unloading with LVAD.[3] In fact, LVAD implantation reduced left ventricular internal dimension at diastole (LVIDd) from 71 mm to 41 mm, and left ventricular end-diastolic volume (LVEDV) from 261 mL to 75 mL, suggesting sufficient LV unloading (*Videos 1* and 2). Although the implantation of LVAD was a trigger of myocardial infarction, it paradoxically saved the patient with its haemodynamic effect.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidelines.

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