Uncommon complication of myocardial infarction revealed by sustained ventricular tachycardia

Mathieu Montoy 1, Pierre-Yves Courand 1,2, Samir Fareh 1, Fadi Farhat 3, Pierre Lantelme 1,2, Brahim Harbaoui 1,2

Available online: 12 January 2016

A 75-year-old woman presented to the emergency department because she was suffering from moderate shortness of breath, cough and asthenia for 2 weeks. Her past medical history included paroxysmal atrial fibrillation related to hyperthyroidism (Basedow’s disease) and CREST syndrome. Cardiovascular risk factors included untreated hypertension, dyslipidemia and age. She denied any symptoms suggestive of angina or myocardial infarction. Physical examination was unremarkable except for the presence of bilateral moderate lung crackles. First laboratory investigations did not find any abnormalities. The presenting ECG showed a regular monomorphic wide complex tachycardia at 170 bpm, with atrioventricular dissociation (figure 1, panel A). Intravenous adenosine did not have any effect on the tachycardia. A diagnosis of ventricular tachycardia exiting from the interoseptal wall was made and the patient was subsequently transferred to our intensive care unit. A first transthoracic echocardiography (TTE) seemed normal with a normal left ventricular ejection fraction at 55% and no mitral regurgitation. The ventricular tachycardia was terminated by a right ventricular overdrive pacing performed through a femoral venous access. ECG showed small Q wave and T wave inversion in the inferolateral leads. Coronary angiography via a right transradial approach demonstrated a chronic total occlusion of the right coronary artery and no significant stenosis on the left one. Angioplasty of the right coronary artery was not attempted in emergency without proof of viability. Chest X-ray did not show significant abnormality. Six hours after admission, cardiac biomarkers significantly increased: NT-proBNP 3862 pg/mL (normal value < 334) and troponin 490 ng/L (normal value < 45). A more detailed TTE
revealed a large echo-free space adjacent to the inferior wall without any detectable thrombus. This large echo-free space communicated with the left ventricle through a narrow neck (figure 1, panel B).

What is your diagnosis?
A cardiac computed tomography confirmed the diagnosis of Left ventricular pseudoaneurysm (LVPSA) of the inferobasal wall (38 mm in antero-posterior axis, 28 mm in transversal axis, 34 mm in infero-superior axis, figure 1, panel C). The following day, the patient was fitted with an implantable cardioverter defibrillator (ICD) and referred to cardiac surgery. A few days later, the patient underwent left ventricular aneurysmectomy and direct closing of the left ventricular wall defect without patch insertion (figure 1, panels D, E, F and G). After 6 months of follow-up, the patient was still asymptomatic and did not suffered from any ventricular tachyarrhythmias recurrences.

LVPSA is an uncommon complication of myocardial infarction, especially at the time of early myocardial revascularization by primary angioplasty [1]. Diagnosis of LVPSA is still challenging. Indeed, clinical presentation includes a broad range of unspecific signs and symptoms. However, this diagnosis remains crucial because of high risk of fatal secondary rupture [2]. LVPSA occurred when myocardial rupture is contained by pericardial adhesions or thrombi. The diagnosis is still challenging because of unspecific symptoms [2]. TTE is usually the first exam to suggest the diagnosis. Cardiac MRI and CT are useful to confirm the diagnosis and differentiate LVPSA from true left ventricular aneurysm [3]. An orifice-to-pseudoaneurysm diameter ratio < 0.5 (narrow orifice) has been described to characterize LVPSA, whereas the range for true aneurysms was between 0.9 and 1.0. Cardiac MRI can also demonstrate loss of epicardial fat at the orifice of the LVPSA [2]. Moreover, the location of LVPSA is mainly in the posterior or inferior wall, while aneurysms are more frequently described on the anterior wall [3]. This distinction is crucial because the treatment strategy is not the same. LVPSA is a surgical emergency most of the time whereas left ventricular aneurysm required only a medical treatment. Secondary rupture of LVPSA is the main cause of mortality but its frequency is difficult to appreciate. The decision of surgery remains sometimes difficult especially for high-risk patients and those with little LVPSA. In these cases, a conservative attitude may be discussed [4]. However, the literature review shows a lack of recent data to decide of a consensual strategy. We illustrated with the present report, an unusual case of LVPSA related to an asymptomatic myocardial infarction and revealed by a sustained ventricular tachycardia. To our knowledge, we described the first case of LVPSA revealed by a sustained ventricular tachycardia after a silent myocardial infarction. Numerous cases of asymptomatic LVPSA have been previously reported in other clinical settings for example: after a mitral surgery for an endocarditis or ischemic atrial fibrillation [5,6].

This case demonstrates the importance of TTE as a first imaging tool in the presence of sustained ventricular tachycardia. Distinguish LVPSA and true aneurysm is still challenging. These two complications following myocardial infarction required specific therapeutic approach.

**Disclosure of interest**: the authors declare that they have no competing interest.

References


