Unruptured giant left ventricular pseudoaneurysm after silent myocardial infarction

Rajeev Bhardwaj,1 Sachin Sondhi,1 Ayushi Mehta2

DESCRIPTION
An 88-year-old woman presented with complaints of class 2 dyspnoea for the last 6 months with episodes of paroxysmal nocturnal dyspnoea. Except for her age, she had no other risk factor for coronary artery disease. On cardiovascular examination, she had double apical impulse with a pan-systolic murmur at the apex and her ECG showed Q waves in inferior leads. She had one episode of resting typical chest pain 2 years back for which she did not seek medical consultation; afterwards, she never experienced angina pain.

Transthoracic echocardiography revealed giant aneurysm involving the lateral wall of the left ventricle (figure 1, videos 1 and 2). The ratio of the maximum diameter of the orifice to the maximum internal dimensions of the aneurysmal cavity was less than 0.5 (figure 2). There was large clot in the aneurysm involving the posterior wall of the left ventricle (figures 3 and video 2). The colour flow signals across the neck of aneurysm showed bidirectional flow (figure 4) and moderate mitral regurgitation (figure 5). The basal septum was akinetic and the posterior wall was dyskinetic (video 2) and left ventricular ejection fraction was 20%–25%. She was started on ecosprin, rosuvastatin, metoprolol, remipril, spironolactone and torsemide and was advised to undergo surgery.

Left ventricular pseudoaneurysm is a rare complication seen after myocardial infarction (MI). The left ventricular free wall rupture (LV FWR) in this thrombolytic era is seen only in 0.5% of cases and carries a substantial mortality of 20%. The FWR is seen either within 48 hours or within 2 weeks of acute MI. The LV FWR causes sudden cardiac death, but sometimes rupture of a ventricular free wall is contained by overlying adherent pericardium resulting in pseudoaneurysm formation. The pseudoaneurysm lacks myocardial tissue and communicates with the cavity of the left ventricle by a narrow neck, whose diameter is less than 50% of the maximum internal dimensions of an aneurysm with more ragged edges and turbulent bidirectional flow. The risk factors for the development of pseudoaneurysm after MI include advanced age, female gender, hypertension, first transmural MI and lack of collateral circulation. In contrast to pseudoaneurysm, true LV aneurysm develops after MI due to scar thinning and infarct expansion and contains epicardium, myocardium and endocardium and has a wide neck, smooth margins and requires conservative treatment.

Figure 1 Transthoracic echocardiogram, apical four-chamber view showing a large aneurysm in the lateral wall of the left ventricle (LV), the neck of the aneurysm (blue arrowhead) and clot in the cavity of the aneurysm (white arrowhead). LA (left atrium), LV (left ventricle), RA (right atrium), RV (right ventricle).
The most common location of LV pseudoaneurysm is posterolateral, in contrast to a true aneurysm, which is mostly located in the apicoanterior wall. The usual presentation of LV pseudoaneurysm is heart failure, embolism, arrhythmias and sometimes sudden cardiac death due to rupture. Some pseudoaneurysms are surprisingly stable and go undetected for years. Besides 2D echocardiography, contrast echo, CMR or even LV ventriculography are other diagnostic modalities for differentiation of true and false aneurysms. Cardiac CT is another important modality as it is easily accessible, quick and offers high spatial resolution. Because the tendency of LV pseudoaneurysm to rupture is high, early surgical intervention by patch closure or Dor’s procedure is recommended.

Learning points

► Left ventricular (LV) pseudoaneurysm appears more commonly after inferior myocardial infarction.
► Although heart failure, embolism and arrhythmias are common presentations, some aneurysms are surprisingly stable and go undetected for years.
► LV pseudoaneurysm requires differentiation from a true aneurysm; because of the tendency of pseudoaneurysm to rupture, early surgical intervention is recommended.

Contributors RB and SS diagnosed the case. Echocardiography was performed by RB. The manuscript is written by SS and AM. The manuscript is approved by all the authors.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES
