

Coronary artery embolization after left ventriculography: A rare cause of myocardial infarction

To the Editor,

Coronary artery occlusion and myocardial infarction (MI) secondary to embolization of intracardiac masses such as thrombi or vegetation is a rare clinical entity (1, 2). Here we present a case of coronary artery embolization (CAE), which occurred after left ventriculography (LVG) in a patient with inferior MI.

A 62-year-old woman was admitted to our emergency department with typical chest pain for 6 h. Twelve-lead ECG showed ST segment elevation in D2, D3, and aVF, which was compatible with acute inferior MI. She received 300 mg acetyl salicylic acid and 600 mg clopidogrel and was transferred to the catheter laboratory for primary percutaneous intervention (PCI). Total occlusion in the middle portion of the right coronary artery (RCA) and a critical lesion (80% stenosis) in the mid left anterior descending (LAD) artery were detected. Intravenous heparin was administered, and direct stent implantation was performed, which restored TIMI 3 flow in RCA. Elective PCI was planned for the LAD lesion. Transthoracic echocardiography showed moderate mitral valve regurgitation (MR) and severe hypokinesia at the mid portion of inferior left ventricular (LV) wall, with LV EF of 45%. An intracardiac mass was not detected. On the third day of hospitalization, she developed dyspnea, and intravenous diuretic treatment was commenced. Control echocardiography was performed, which was consistent with the first

examination. After consultation with the cardiovascular surgeons, we decided to perform control coronary angiography and ventriculography on the fourth day of hospitalization. First, LAD injections were administered, which showed a critical lesion (80% stenosis) in the LAD artery. LVG showed mild MR and hypokinesia of the inferior wall. Just after LVG, the patient described sudden onset chest pain, and ST segment elevation was observed on the monitor. The LAD artery was cannulated, and control angiograms were obtained. The LAD artery was found to be occluded with a huge thrombus at the site of the stenosis. Intravenous heparin and tirofiban infusion were administered, and PCI was performed. A 3 x 20 mm stent was implanted after balloon angioplasty, and distal TIMI 3 flow was restored. The rest of the hospitalization was uneventful.

Even in the modern era of intensive pharmacotherapy, including anticoagulant and antiagregant medications, embolic complications may occur after MI (3). In our case, CAE probably occurred secondary to micro-thrombi formed near the hypokinetic segments of LV during the early phase of MI. Dislodgement of micro-thrombi during catheter manipulation or contrast injection may have caused LAD occlusion. The diagnosis of micro-thrombi is challenging, and as in our case, transthoracic echocardiography may not have enough resolution for the detection of micro-thrombi. Other imaging modalities such as transoesophageal echocardiography may be safer for the evaluation of LV thrombi, aneurysm, and the severity of valvular disease in this patient population.

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