

Dynamic Left Ventricular Mid-Cavitary Obstruction Complicating Anteroseptal Myocardial Infarction

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Abstract

Dynamic left ventricular outflow tract (LVOT) and mid-cavitary obstruction has been associated with hypertrophic obstructive cardiomyopathy. Acute LVOT and mid-cavitary obstruction has been described as a complication of myocardial infarction (MI). In the latter situation, this unusual form of dynamic obstruction occurs as a result of compensated hyperdynamic basal wall motion in patients with apical infarction. We describe a patient who developed mid-cavitary obstruction following silent anteroseptal MI (*Iranian Heart Journal 2003; 4 (4):72-74*).

Case report

A 60-year-old woman with a history of hypertension and hyperlipidemia presented with low-threshold angina following a recent neglected anteroseptal myocardial infarction. At the time, echocardiogram revealed no evidence of LVOT or mid-cavitary obstruction. It was claimed two months before coronary angiography that the patient had severe resting angina, but she refused to present to the hospital because of remission of pain. Since then she developed progressively worsening chest pain and dyspnea on exertion. Spect myocardial perfusion imaging showed a partially-reversible defect in the inferior wall apex, mid-anterior and anteroseptal wall (Fig. 1).

Electro-cardiogram (ECG) showed a new Q-wave in inferior & V₁-V₃ leads compared to the previous ECG (Fig. 2).

Physical examination revealed a new grade II/VI systolic ejection murmur in the left parasternal area and apex without radiation to the carotid arteries. Transesophageal echocardiography revealed mid-ventricular dynamic obstruction, apical true aneurysm

and a 30 mmHg gradient at the mid-left ventricular level (Fig. 3).

Coronary angiography revealed 3-vessel coronary artery disease with total occlusion of LAD in the distal part, antero-apical aneurysm and ejection fraction of 60% with 30 mmHg gradient at the mid-left ventricle (Fig. 4).

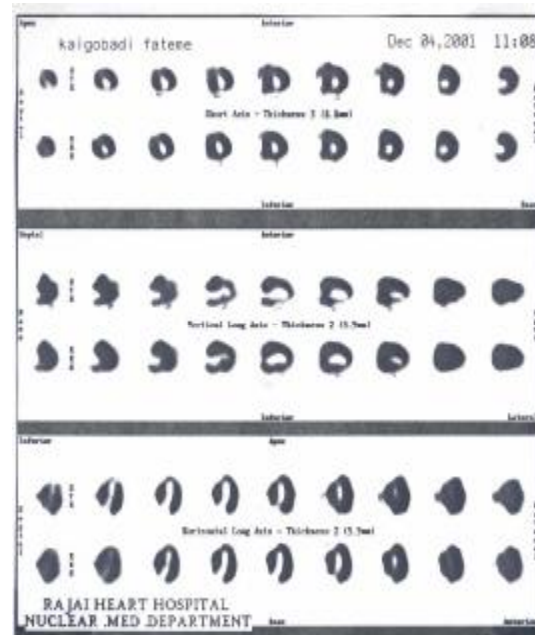


Fig. 1. Spect MPI by Tc^{99m} – sestamibi depicting ischemia in the apex, inferior wall, anteroseptal and mid-anterior wall.



Fig. 2. ECG showed NDR, LAD, ST-T charges in I, AVL, V₆ and Q-S pattern in II,III, AVF and V₁ and poor R-wave progression.

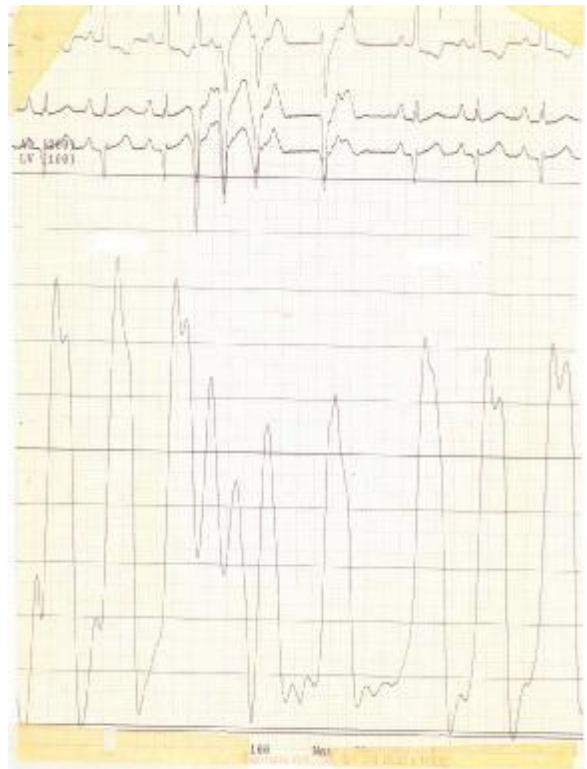
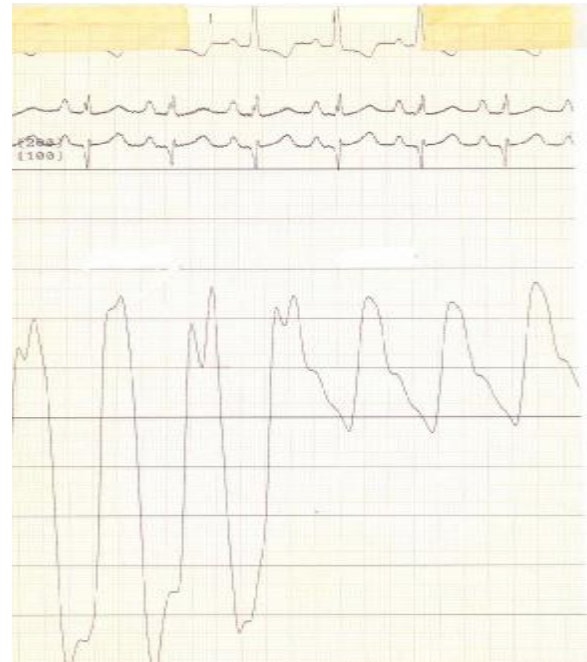


Fig. 4. Pull-back of catheter from LV apex to aorta showed a 30mmHg gradient in mid-LV cavity (A, B).



Fig. 3. Transesophageal echocardiogram. A, mid-esophageal view 135⁰ showing basal septal hypertrophy and normal aortic valve. B, Transgastric view 90⁰ revealed mid-cavity obstruction in systole.

Discussion

The occurrence of dynamic LV obstruction in the setting of an acute coronary syndrome is usually due to an antero-apical infarction with compensating basal hyperkinesis, resulting in a decreased mid-LV cross-sectional area. The treatment of AMI complicated by the occurrence of a dynamic mid-LV gradient is different from the standard treatment. The use of vasodilators and inotropic agents should be avoided in this setting. Our patient improved medically with discontinuation of nitrates and administration of propranolol and diltiazem. Doppler echocardiography should be considered the diagnostic modality of choice to exclude the presence of dynamic mid-cavitary obstruction.

References

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