

Extensive Anterior Myocardial Infarction and Ventricular Septal Rupture Caused by Myocardial Bridge

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Abstract

A 45-year-old man was referred to our hospital for angina pectoris 2 days after an anterolateral myocardial infarction. On admission, he had a blood pressure of 100/70 mm Hg and holosystolic murmur at the left sternal border. Transthoracic echocardiography showed akinesia in the apicoseptal, apicoinferior, and apicoposterior regions of the left ventricle, with large apical aneurysm and clot. There was also a large apical ventricular septal defect (VSD) with a significant left-to-right shunt. During cardiac catheterization, the only abnormality found beside the VSD was myocardial bridging in the mid part of the left anterior descending coronary artery and ostial lesion in the first diagonal artery. The patient was scheduled for bypass surgery and VSD closure (*Iranian Heart Journal 2012; 13 (1):49 -51*).

Keywords : VSD ■ Muscle bridge ■ MI

Case Report

A 45-year-old man was referred to our hospital for angina pectoris 2 days after an anterolateral myocardial infarction. On admission, he had a blood pressure of 100/70 mm Hg and holosystolic murmur at the left sternal border. The electrocardiogram at referral showed small R-waves in leads V1 and V2, pathologic Q waves in leads V3 and V4, and negative T-waves in the lateral leads. Transthoracic echocardiography showed akinesia in the apicoseptal, apicoinferior, and apicoposterior regions of the left ventricle, with large apical aneurysm and clot (Fig1). Left ventricular function was about 20%. There was also a large apical ventricular septal defect (VSD) with a significant left-to-right shunt with a 55 mm Hg gradient.

The patient was scheduled for coronary angiography on the admission day (Fig 2).



Fig 1- Apical off- axis view of ventriculoseptal defect, apical

Received Mar. 10, 2012; Accepted for publication May. 2, 2012

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During cardiac catheterization, the only abnormality found beside the VSD was myocardial bridging in the mid part of the left anterior descending coronary artery and an ostial lesion in the first diagonal artery.

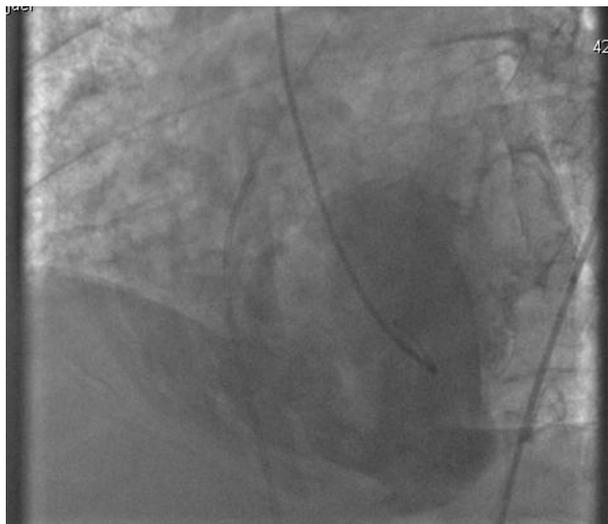


Fig 2. Left ventricular injection in LAO showing muscular VSD

Right heart catheterization showed a central venous pressure of 18 mm Hg, pulmonary pressure of 62/30 mm Hg, and pulmonary wedge pressure of 30 mm Hg. Saturation study showed 92% (aorta), 72% (pulmonary artery), and 60% (mixed venous). The ratio of pulmonary-to-systemic circulation was thus 1.77:1. He was scheduled for surgery after two weeks. At operation, the infarcted area extended from the mid-left anterior descending artery to the apex. A pericardial patch was inserted on the left ventricular aspect of the rupture, and the right ventricular free wall was used to stabilize the rupture. On follow-up echocardiography, a residual VSD (4 mm in size) with no significant shunt was evident. He was discharged 10 days after the operation and is doing well now 8 weeks postoperatively.

Discussion

The prevalence of myocardial bridges in various anatomic studies varies from 5.4% to

85.7%¹ During angiography, systolic narrowing of a coronary artery segment is seen in 0.5% to 4.5% of patients, and the most frequent site reported is the middle segment of the left anterior descending coronary artery. Presumably, most myocardial bridges do not induce any symptoms but sometimes they can be the origin of angina pectoris, myocardial infarction, life-threatening ventricular arrhythmias, or even death. Myocardial perfusion occurs mainly during diastole; therefore, systolic narrowing is probably not the only cause of ischemia. However, ischemia can be caused by other mechanisms. Endothelial cell damage might occur because of the continuous mechanical stress, which predisposes the vessel segment to vasospasm. Another mechanism might be premature arteriosclerosis at the site of the myocardial bridge.

Although there was a 1% to 2% incidence of ventricular septal rupture in the pre thrombolytic era, as a complication of myocardial infarction treated with thrombolysis, it has recently been reported to be as low as 0.2%². In recent studies, advanced age, anterior location of infarction, female sex, and history of non-smoking are the most important predictors of ventricular septal rupture.³ In the present case, the ventricular septal rupture occurred in the presence of only myocardial bridging of the mid-left anterior descending artery. There was stenosis in the diagonal artery; however, its size was not large enough to be the reason for extensive infarction. In addition, a left ventriculogram showed contrast in the right ventricle. Although the failure of reperfusion of the infarct-related coronary artery is thought to be an important contributing factor in ventricular septal rupture, we found the left anterior descending artery to be patent in our patient. Recurrent ischemia caused by the bridging, presented as post-infarct angina, could have contributed to the rupture.^{4,5,6}

References

1. Ishii T, Asuwa N, Masuda S, Ishikawa Y. The effect of a myocardial bridge on coronary atherosclerosis and ischaemia. *J Pathol.*1998;185: 4 –9.
2. Ishikawa Y, Akasaka Y, Ito K, Akishima Y, Kimura M, Kiguchi H, Fujimoto A, Ishii T. Significance of anatomical properties of myocardial bridge on atherosclerosis evolution in the left anterior descending coronary artery. *Atherosclerosis.* 2006;186:380 –389
3. Channer, KS, Bukis, E, Hartnell, G, et al (1989) Myocardial bridging of the coronary arteries. *Clin Radiol* 40,355-359.
4. Juillièrè, Y, Berder, V, Sutti-Selton, CH. Isolated myocardial bridges with angiographic milking of the left anterior descending coronary artery: a long-term follow-up study. *Am Heart J* 1995;129,663-665.
5. Wymore, P, Yedlicka, JW, Garcia-Medina, V. The incidence of myocardial bridges in heart transplants. *Cardiovasc Intervent Radiol* 1989;12,202-206
6. Bezerra, AJ, Prates, JC, DiDio, LJ Incidence and clinical significance of bridges of myocardium over the coronary arteries and their branches. *Surg Radiol Anat* 1987;9,273-280