Brief Communication

Free Wall Rupture and Ventricular Septal Defect Post Acute Anterior Myocardial Infarction

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Abstract

Myocardial free wall rupture is a catastrophic complication of acute myocardial infarction, and prognosis will depend on the prompt diagnosis by echocardiography, extension of infarct size, and prompt surgical treatment. Free wall rupture concomitant with ventricular septal defect (VSD) may be more complicated for management. A case of a 69-year-old man with myocardial free wall rupture and VSD following acute anterior myocardial infarction is presented.

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Introduction

Myocardial free wall rupture is the second most common cause of in-hospital mortality among patients with an acute myocardial infarction; and based upon several large studies, it accounts for 7-17% of all deaths.[1,2] Free wall rupture may present with a constellation of symptoms that, if recognized early and diagnosed accurately, may allow for emergent successful treatment. These include chest pain, hypotension, nausea, vomiting, agitation, and signs of increased adrenergic drive. Autopsy studies suggest that a subset of free wall ruptures, up to 40%, follow a sub acute course.[3] Numerous pathologic observations have confirmed that rupture is an ongoing stuttering process characterized by progressive tears and hemorrhage into the pericardial space, which may seal itself with an overlying clot or with the formation of a pseudoaneurysm.[4] Ventricular septal defect (VSD) is a rare and serious complication. The incidence is 1-2% of all myocardial infarctions.[5,6]

Case report

A 69-year-old man with a history of hypertension, diabetes mellitus, and acute renal failure with creatinine=3.1 mg/dl was admitted in our hospital with an acute anterior myocardial infarction. His electrocardiogram showed Q wave, elevation of ST segments, terminal T invert in the leads V1 through V6, and a rise in serum levels of the myocardial- specific isoenzyme of creatine kinase and of troponine.

Transthoracic echocardiography (TTE) suggested: normal left ventricular (LV) size; concentric left ventricular hypertrophy (LVH); evidence of myocardial infarction in 6 segments in the left anterior descending artery (LAD) territory: relatively thin with akinesia of apical segments, anteroseptal mid portion, and septal mid portion with aneurismal formation in septal apical and inferoapical; large immobile LV apical clot; global left ventricular ejection fraction (LVEF) about 30%; a large ventricular septal defect (VSD) (Figure 1) in septal apical with peak systolic gradient

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about 52 mmHg; and severe pericardial effusion up to 30 mm around right ventricular (RV) and up to 15 mm around left ventricular (LV) with right atrium (RA) and right ventricular (RV) collapse. Color Doppler showed evidence of active flow into the pericardium and rupture of septal apical into the pericardium. Flow within the pericardium was non turbulent (Figure 2), but in the site of rupture of septal apical to pericardium, there was a turbulent flow (Figure 3).

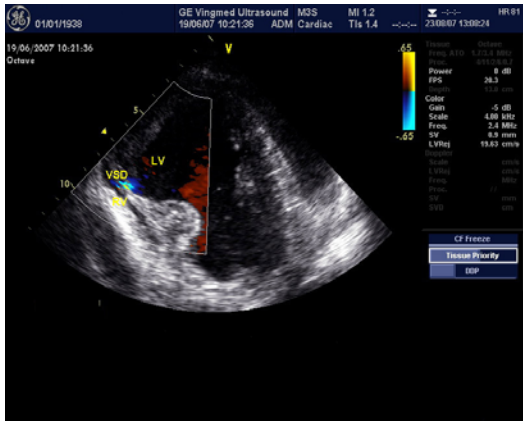

Figure 1. Transthoracic echocardiography showed a large VSD
VSD, Ventricular septal defect; LV, Left ventricle

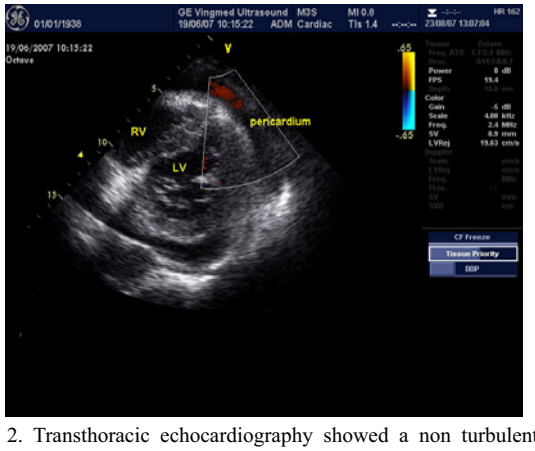

Figure 2. Transthoracic echocardiography showed a non turbulent flow within the pericardium
RV, Right ventricle; LV, Left ventricle

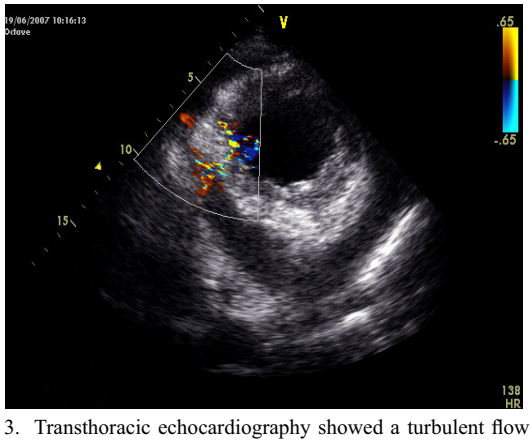

Figure 3. Transthoracic echocardiography showed a turbulent flow in the site of myocardial rupture into the pericardium

Also, there was mild tricuspid valve regurgitation (TR), estimated pulmonary artery pressure (PAP)=42 mmHg, and mild RV enlargement with mild RV systolic dysfunction. Cardiac catheterization showed three-vessel coronary artery disease with an ejection fraction of 30%.

Because of hemodynamical instability, intra-aortic balloon pump (IABP) was used before surgery. The patient underwent surgery for repair of VSD and myocardial free wall rupture concomitant with coronary artery bypass grafting (CABG). At surgery, blood was present in the pericardium, and a localized rupture site in the anterolateral wall of LV was conceded. When the rupture of LV was opened, large apical and mid septal VSD was seen, and necrosis had progressed close to the base of the heart. The surgical procedure included ventriculotomy throughout necrotic and ruptured LV, debridement of necrotic free wall and septum, extensive septal patch with cortex 0.6, and closure of LV by Teflon felt and prolene. Because of the necrosis of the LAD artery, we did not use the left internal mammary artery (LIMA) for grafting. CABG with two saphenous vein bypass grafts was performed. Postoperative echocardiography findings were: LVEF=35%, no myocardial rupture or VSD, mild pericardial effusion, and mild TR and PAP= 24 mmHg.

About 4 weeks after surgery, the patient was discharged uneventfully.

Discussion

Free wall rupture complicates 4-6% of all infarctions. It is the most common cause of hemopericardium, exceeding even that of aortic dissection.[3,7] With advances in echocardiography, it is possible to diagnose free wall rupture prior to the development of tamponade and hemodynamic collapse. The absence of pulse and heart sounds despite normal rhythm on the electrocardiogram suggests that cardiac rupture after acute myocardial infarction produces tamponade and electromechanical dissociation. Two-dimensional echocardiography is the most sensitive and expeditious test for the diagnosis of cardiac rupture, as demonstrated from numerous studies. The most common findings of rupture on echocardiography are that of a pericardial effusion and layered echogenic pericardial thrombus.[8,9] Finding regional dilatation and an abnormally thin myocardium with akinesis, which may indicate infarct expansion, increases the specificity.

Lopez-Seadan et al. prospectively looked at the sensitivity and specificity of echocardiography and found that echocardiograms were 100% sensitive, as all patients with rupture had at least a 5-mm pericardial effusion during diastole and 97% had evidence of an intrapericardial thrombus.[10]

The incidence of ventricular septal rupture following a myocardial infarct is approximately 1%.[11] Clinical features associated with an increased risk of rupture of the interventricular septum include lack of development of a

collateral network, advanced age, hypertension, anterior location of infarction, and possibly Thrombolysis. The timing of the rupture varies between several hours and several weeks following the infarct. The site of rupture depends on the coronary anatomy and the infracted vessel. Two large studies have reported that concomitant CABG is beneficial. First, Muehrcke et al.[12] found that patients who had coronary artery disease outside of the infracted region of myocardium fared significantly better, long term, if they were grafted. Cox et al.[13] reported similar findings.

Management of hemodynamically unstable patients with a large septal rupture consists of inotropic support, left ventricular afterload reduction by medical therapy, as well as placement of an intra-aortic balloon pump. Urgent surgical closure of such VSDs is recommended.[14] We presented one case of anterior MI complicated by both VSD and free wall rupture in which transthoracic echocardiography clearly assisted in the correct diagnosis.

Conclusion

Our patient had VSD concomitant with myocardial rupture post acute anterior MI. Echocardiography, which has already been proven to be the most effective diagnostic tool in the acute setting, led to the correct and immediate diagnosis of both lesions.

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