Pseudoaneurysm Following Lateral Wall Myocardial Infarction


Abstract

Pseudoaneurysm (PA) formation of left ventricle (LV) following acute myocardial infarction (AMI) is uncommon and is usually believed to be associated with a grave prognosis. We describe a case of 55 year old male patient presented with AMI and heart failure with a systolic murmur later diagnosed to have PA of the lateral wall of LV on echocardiography (transthoracic and transesophageal, TTE and TEE). Cardiac MRI and coronary angiogram (CAG) were performed. CAG showed 60% lesion at origin of major obtuse marginal artery (OM). The patient was advised surgical treatment, but he refused and took discharge against medical advice on 27th day of admission on stable condition.

INTRODUCTION

Rupture of the free wall of LV following AMI is an uncommon but dramatic mechanical complication of AMI, which constitutes up to 10 % of hospital deaths due to AMI. Rupture of the free wall of LV leads to hemopericardium and death from cardiac tamponade, but in some patients the course is subacute with nausea, hypotension, persistent chest pain of pericardial type and persistent gallops. In this second group of patients the rupture is incomplete and organizing thrombus and hematoma together with pericardium seal the rupture of the LV and thus prevents development of hemopericardium and tamponade. With time this area of organized thrombus and pericardium can become a PA (false aneurysm) that maintains a communication with LV through a narrow neck. Myocardial infarction is the most common cause that leads to cardiac rupture and formation of PA. Other less common causes are cardiosurgical operations, penetrating cardiac trauma, use of left ventricular apical venting during cardiosurgical procedures and infective endocarditis. Cardiac rupture and formation of PA can occur within few days (3-6 days) after AMI but there are reports of much later presentation even up to 2 years. PA formation has also been reported following silent MI. The most frequently reported clinical symptoms of PA are heart failure (36%), chest pain (30%), dyspnea (25%) and sudden death (3%).

Myocardial rupture and formation of PA usually occurs in anterior or lateral walls of LV, in the area of terminal distribution of left anterior descending artery.

Echocardiography (TTE and TEE) is the gold standard in establishing the diagnosis of PA. But in some cases where the location of PA is unusual, cardiac MRI is a useful tool for early diagnosis. Under most favorable conditions cardiac catheterizations can be carried out not necessarily to confirm the diagnosis of PA but to delineate the coronary anatomy. This is helpful so that, in addition to ventricular repair, CABG can be performed in appropriate patients.

When diagnosis of PA is confirmed prompt elective surgery is indicated because rupture of the PA occurs relatively frequently. However there are some recent reports of long term survival of patients with post-infarction left ventricular PA. 2

CASE REPORT

A 55 years old male, smoker, nondiabetic, normotensive patient presented with left sided pain in chest of more than 12 hours duration and dyspnea. On admission his electrocardiogram showed elevation of ST segment in leads I, aVL (Fig. 1) and bedside Trop T test read positive. Physical examination revealed sinus tachycardia, low volume pulse, blood pressure 80/ 60 mm Hg on admission, and a short mid systolic murmur along left sternal edge. Cardiac palpation was unremarkable. On auscultation of chest there were fine crepitations at bases extending up to angle of scapula. He was given antianginal management along with low molecular weight heparin and frusemide injection, IV nitroglycerine, and aspirin. Because of his late presentation he did not qualify for thrombolytic therapy. His anginal symptoms improved, he was partially relieved of dyspnea but the systolic murmur persisted.

His other biochemical parameters are: FBS - 88mg %, blood urea- 23mg %, serum creatinine- 0.8 mg %, serum CK 252 mg% and serum CK - MB 53mg%. On 4th day of admission
echocardiographic examination was done to exclude papillary muscle dysfunction/post-infarction ventricular septal defect. It revealed borderline LV systolic dysfunction (LVEF - 46%) and rupture of the myocardial free wall of lateral aspect of LV and small pericardial effusion but no feature of cardiac tamponade. One remarkable finding was pressure gradient of 36 mm Hg at the neck of the rupture (Fig. 2). The patient was subjected to TEE on the following day where no added information was recorded. The patient was given optimal antifailure treatment and he showed symptomatic improvement. Cardiac MRI was done which clearly demonstrated the PA and its neck and hemopericardium (Fig. 3). CAG was done on 12th day of admission. CAG showed 60% lesion at origin of OM1 (Fig. 4). Other coronary arteries were normal. Other wall motion showed no abnormality. Surgery was planned but the patient refused surgery and he took discharge against medical advice.

**DISCUSSION**

LV pseudoaneurysm is an uncommon complication of AMI in which a free-wall rupture is contained by overlying adherent pericardium. The timely diagnosis of pseudo-aneurysm and its immediate surgical treatment are of great clinical importance because of the increased danger of acute rupture of aneurysm and sudden death. In a recent review article, Frances et al described the etiology, clinical picture, diagnostic value of various techniques, prognosis and the treatment of this serious post-infarction complication in detail. The probable events that leads to the creation of PA are: transmural myocardial infarction causes local reactive pericarditis at the site of infarction and consequently the growth of fibrous symphysis between the visceral and parietal layer of pericardium. Rupture of the myocardial wall at the necrosis site and escape of blood, which is entrenched by the symphysis of pericardium shaping the aneurysmatic sac. This sac progressively enlarges and its internal surface is covered by thrombus.

Slatre et al reported in the SHOCK trial registry that most cases of cardiac rupture and PA occur within 3 - 6 days of AMI. In our case it was detected on the 4th day of AMI by echocardiography. Frances et al described that most cases (36%) of PA presented with symptoms of heart failure. In our case persistent signs and symptoms of left heart failure made
us suspicious of some mechanical complication of AMI which was diagnosed as a case of PA.

Echocardiography is the method of choice for prompt diagnosis of PA and immediate clinical decisions for patient management. Echocardiographic features of PA include - small diameter of rupture compared to length of the sac (ratio<0.5) and the presence of thrombus in the internal wall of aneurysmal sac. Color flow Doppler may demonstrate systolic flow velocity towards the cavity of the aneurysmal sac and diastolic flow velocity towards the cavity of the LV. In our case also we noted systolic flow gradient at the neck of the aneurysm.

There are certain points of interest in this patient. Firstly this patient had a very small infarction as evidenced by ECG (ST-T changes on I and a VL only), echocardiogram and coronary angiogram. On echocardiogram only the basal aspect of lateral wall of LV was dyskinetic where rupture occurred. On coronary angiogram, a branch of left circumflex (OM1) was diseased and only 60 % of that artery was occluded. Other major vessels were normal. Second point is the demonstration of pressure gradient at the neck of the PA on echocardiogram along with a systolic murmur. Systolic murmur originating at the neck of PA had not been documented in literature. Third point of interest is the relatively benign course of the patient. The patient was almost symptom-free at the time of discharge without operation.

PA may lead to fatal rupture a long time after MI and most clinicians recommend surgical repair as soon as possible when the diagnosis is established. However two variables should be taken into consideration, postoperative mortality and the risk of fatal rupture following conservative treatment. Postoperative mortality after surgical treatment of LV PA ranges from 13 to29 %.5 Most clinicians accept this relatively high mortality because of risk of fatal rupture. In a recent review, Natarajan et al5 concluded that medical treatment of chronic PA is not associated with an increased risk of cardiac rupture. Recently a report published by Moreno et al5 showed that long term outcome of patients (10 in number) with post-infarction LV PA is relatively benign with very low risk of fatal rupture(none in this series with 4 years of follow up). Chronic anticoagulation should be considered in these patients because of the relatively high risk of ischemic stroke (32.5%). The study conducted by Natarajan et al5 showed13% of patients with LV PA had systemic embolism as the clinical presentation.

References


Announcement

Journal of Postgraduate Medicine, a publication of Staff Society of Seth GS Medical College and KEM Hospital, India, has the distinction of completing 50 years of publication in 2004. As a part of the Golden Jubilee celebrations of the Journal, a conference on writing, editing, e-publishing and open access is being organised in September, 2004. This will be first of its kind of event in a developing country and is being supported by Open Society Institute (OSI), Internation Network for Availability of Scientific Publications (INASP) and World Association of Medical Editors (WAME).

Title: JPGM GOLD COIN: 50 years of medical writing - International conference on medical writing, editing and publishing.

Organisers: Journal of Postgraduate Medicine

Dates: 23rd - 26th September 2004

Venue: Mumbai, India

URL: http://www.jpgmonline.com/goldcon.asp (provides facility of online registration and payment through credit card and internet banking)

Main topics: Medical writing, editing, journal publishing, electronic publishing, open access, publication ethics

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