Right Ventricular Free Wall Pseudoaneurysm Following an Inferior Myocardial Reinfarction Cured by Late Mechanical Reperfusion

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Abstract

We present a 63-year male patient with an inferior-wall myocardial reinfarction and a recent episode of gastrointerstinal bleeding, who was treated with mechanical reperfusion sixteen hours after the onset of pain, and who experienced right ventricular pseudoaneurism as a serious postinfarction mechanical complication. Elective surgical revascularisation was the definite treatment.

INTRODUCTION

In-hospital reccurent acute myocardial infarction (AMI) is associated with an inverse prognosis, primarily because of an increase in early mortality during the index hospitalization [1]. Revascularization during the index hospital period, even out to 36 hours post MI [2], is associated with a lower rate of early and long-term mortality up to two years.

Mechanical complications after AMI strongly influence the prognosis. There are very few reported cases of right ventricular (RV) free wall pseudoaneurysm after AMI, resembling the wall rupture contained into the myopericardial space [3]. Because of a high risk of rupture, prophylactic repair of this serious complication is indicated soon after an exact echocardiographic diagnosis is assessed [4].

CASE REPORT

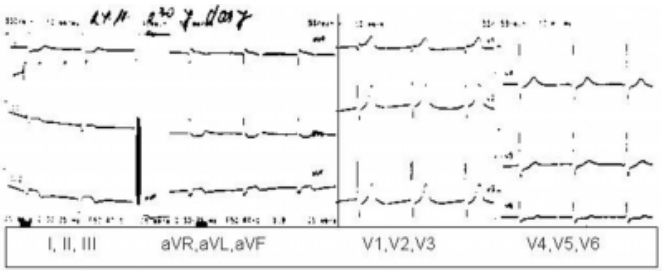
A 63-year male patient was admitted into the coronary care unit because of precordial pain lasting for 2 hours before presentation. He suffered several attacks of gastrointestinal bleeding since 1961 when duodenal ulcer was diagnosed; the last attack occurred eight days before admission.

His physical presentation was normal. The electrocardiogram (ECG) showed atrial fibrillation, monomorphic ventricular premature beats, ST segment elevation in leads II, III, aVF and V4R-V6R, and ST segment depression in leads I, aVL, V2-V6.

The diagnosis of acute inferoposterior myocardial reinfarction was established, and a parenteral therapy with analgetics, H2 blockers, and anticoagulants was started. Twenty-four hours after admission, the patient was painless. Unfortunately, on the fourth day of hospitalization he experienced intensive retrosternal pain, and ECG showed nodal rhythm (Figure 1).

Figure 1

Figure 1: ECG showing inferior reinfarction with nodal rhythm and 56 bpm heart rate. ST elevation in lead V1 points to the right ventricular infarction.



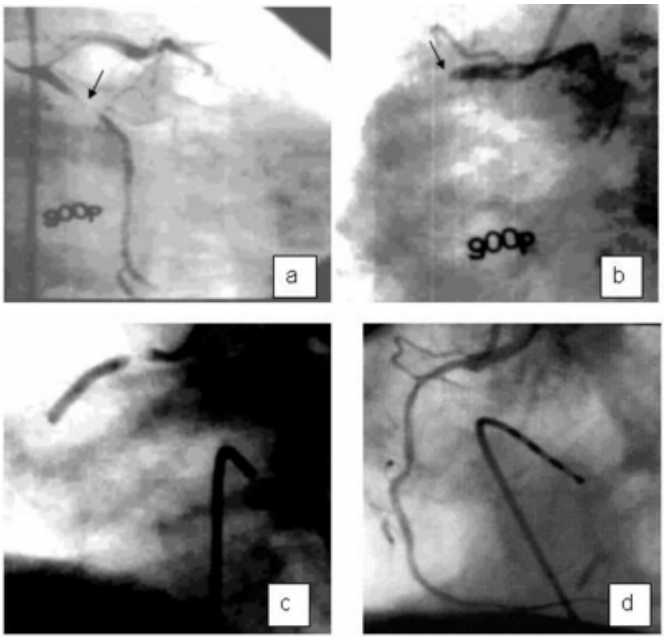
Because of the persistence of pain, ST elevation and contraindication for thrombolysis (recent gastrointestinal bleeding), coronary angioplasty was started 16 hours after the onset of pain. The left coronary artery was visualised first, and a 75% stenosis was noticed on the circumflex branch before the onset of the first marginal artery (figure 2a). Angiography of the right coronary artery (RCA) revealed its proximal occlusion (figure 2b). RCA was reopened using coronary guidewire, and TIMI I-II flow was

established.

Because of the irregular atherosclerotic lesions in the proximal and medial parts of the artery, coronary angioplasty (figure 2c) was performed, and resulted in RCA proximal and medial TIMI II -III flow. The posterior descending branch remained occluded (figure 2d).

Figure 2

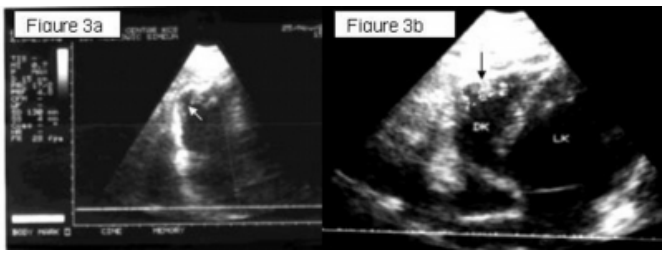
Figure 2: a) 75% stenosis on the circumflex branch (arrow) before the onset of the first marginal artery, b) proximal occlusion of the right coronary artery (arrow), c) inflated baloon in the proximal right coronary artery during the first transluminal angioplasty, d) final result.



After the procedure, repeated attacks of dyspnea and hypotension were observed; early echocardiographic examination showed right ventricular enlargement, with medial free-wall systolic dyskinesia. There was an interruption in the continuity of the endocardium in this area (figure 3a - arrow), with saccular formation which communicated with the right ventricle through its narrow neck (figure 3b - arrow). Dense pericardial effusion was noticed around the right ventricle, measuring 8 mm in thickness.

Figure 3

Figure 3: a) interruption in the continuity of the endocardium in the area of free wall systolic dyskinesia, b) saccular formation that communicates with right ventricle through its narrow neck (arrow), DK=right ventricle, LK=left ventricle



Sinus rhythm was recovered three days later and the patient was transported to cardiac surgery. On reviewed echocardiography, small fibrous echo and altered wall motion localized between medial and apical segments of the right ventricle free wall were detected, but without evident signs of right ventricle pseudoaneurysm. No liquid was present in the pericardial space.

The patient underwent an elective surgical revascularisation one month later. Autovenous grafts were placed from aorta to the right coronary artery and to the left circumflex branch, periaortic sympatectomy and mediastinal drainage being performed within the same procedure. No pseudoaneurysm was observed during surgery, and postoperative course was uncomplicated.

The patient came a year later to our hospital for clinical evaluation. His physical examination was normal. ECG showed sinus rhythm, low r waves in leads II, III, aVF, and high R wave in lead V2. Exercise stress test found no significant ST/T segment changes.

Echocardiography revealed thin, fibrous and hypocontractile basal segment of the left ventricular inferior wall. Right ventricular dimensions were normal. Mild tricuspidal regurgitation was present. Pericardium was hyperechogenic, with adhesions along the right ventricle free wall.

DISCUSSION

Primary coronary angioplasty (PCA), performed out to 36 hours from the onset of ST elevation or new bundle branch block, improved survival in patients with AMI and cardiogenic shock [2]. In another study, PCA was associated with reduced mortality after 20 days (5.2% vs. 9.9%) and reduced combined rate of death and heart failure (6.5% vs. 16.4%); the rate of reocclusion was 18% [5]. The TAMI-6 study also evaluated late coronary angioplasty or

thrombolysis, 7 to 48 hours after the onset of symptoms. After six months there were no late benefits on angiographic patency, rate of reinfarction and systolic function [6]. Conclusions concerning late reperfusion will require data from large-scale trials designed to assess mortality reduction.

In exceptional cases, postinfarction rupture is not transmural, but remains circumscribed within the wall itself as a cavity joined to the left ventricle through a narrow neck, called pseudo-pseudoaneurysm. A new classification of aneurysms is proposed: true, false, pseudo-false and mixed [7].

Of 2600 studied consecutive patients with AMI , six had left ventricular pseudoaneurysm [8]. Coronary angiography demonstrated multivessel disease and occlusion of the infarction-related artery (TIMI-0) without adequate collateral circulation (grade 0-1). Pseudoaneurysm of the right ventricle after myocardial infarction may be assocciated with ventricular septal rupture [3]. The prognosis of pseudoaneurysm might be determinated by organised thrombus, serpentine-like structure, coronary revascularisation during surgical repair, and vigorous medical management [9]. Ventricular pseudoaneurysms and pseudo-pseudoaneurysms after myocardial infarction need urgent surgical repair [10]. Prior to surgery, exact diagnosis, anatomical relations and size can be assessed with echocardiography [4].

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