

Diverse Presentations of Cardiac Rupture Following Acute Myocardial Infarction

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Abstract

Background: Cardiac rupture is a rare but ominous complication of myocardial infarction.

Objectives: To study the clinical presentation, medical course, outcome and echocardiographic predictors of patients with myocardial rupture.

Methods: We evaluated 15 consecutive patients with cardiac rupture during a 4 year period in our department. The current report explores the presence of potential risk factors, timing, relation to the thrombolysis, coronary interventions and outcome.

Results: The index event in all patients was first ST elevation myocardial infarction. In seven patients rupture occurred in the first 24 hours. Pericardial effusion on admission with a clot was present in three patients. Five patients received thrombolytic therapy. Only three patients underwent coronary angioplasty, but in one case it was performed late and in two patients the culprit artery could not be opened. Six patients reached the operating room, of whom three survived.

Conclusions: The lack of early mechanical reperfusion in acute myocardial infarction and thrombolytic therapy are risk factors for cardiac rupture. Pericardial effusion on admission and evidence of a clot are echocardiographic indicators of cardiac rupture and should alert the medical team to further assess the possibility of cardiac rupture.

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Cardiac rupture is a rare complication of myocardial infarction (1–4%) [1] and has a poor prognosis if not operated on immediately. The early diagnosis of this life-threatening complication is crucial. In this report we summarize the clinical course in 15 patients with cardiac rupture and try to establish the most common clinical and echocardiographic predictors of this serious complication.

Patients and Methods

During the period 2000–2003 we encountered 15 cases of cardiac rupture following acute myocardial infarction. During the same period 1,600 patients with acute ST elevation MI were admitted to our institute. One patient had recurrent leak from the site of operation and was re-operated successfully. The patients' clinical presentation, medical course, relation to the thrombolytic therapy or primary angioplasty and the echocardiographic findings were analyzed.

MI = myocardial infarction

Results

The mean age of the 15 patients (6 males and 9 females) was 73 (range 52–94 years). Their mean left ventricular ejection fraction was 37% (range 25–50%) [Tables 1 and 2]. The incidence of rupture was 0.93%. The index event in all patients was first ST elevation myocardial infarction, which was inferior in eight, anterior in six and lateral in one. Pericardial effusion on admission and evidence of intrapericardial clot were present in three patients, none of whom had received thrombolytic therapy. The time from the onset of chest pain to rupture was 48 hours (range 3–144 hours). In seven patients rupture occurred in the first 24 hours: six of them had anterior MI; three of the seven were treated with thrombolytic therapy. Four patients received thrombolytic therapy (tissue plasminogen activator or streptokinase). The time from onset of pain to lytic therapy was 6 hours (range 3–11 hours). The incidence of thrombolytic therapy in the general cohort of MI patients was 40% and in the patients with rupture 33%.

Six patients underwent coronary angiography; in three of them angioplasty was performed, but in one patient it was late (18 hours after the beginning of chest pain) and in two patients it was not performed to the culprit artery. The incidence of primary angioplasty in the general cohort was 31%; none of the patients with rupture had undergone adequate primary angioplasty.

Pericardiocentesis was attempted in three patients but was unsuccessful in two of them. Six patients were transferred to another hospital for immediate operation and reached the operating room; three of them survived. One patient had a recurrent rupture 2 months after the initial urgent surgical closure of a tear in the inferior wall with a patch. He was admitted because of dyspnea, atypical chest pain, pleural effusion and elevation of the cardiac enzymes. Echocardiography revealed large bloody pericardial effusion with compression of the right heart chambers [Figure 1]. The patient underwent successful re-closure of the rupture. Figure 2 demonstrates rare documentation of pericardial rupture in a 77 year old woman with extensive anterior MI that occurred during echocardiographic examination. In spite of immediate aggressive therapy and pericardial drainage the patient could not be saved.

Discussion

Cardiac rupture is a rare but frequently fatal complication of myocardial infarction. Cardiac rupture may occur early, often within 24 hours, due to the initial evolution of MI. Late peak occurs between day 4 and 7 and is related to the expansion of left

Table 1. Individual data of the patients

Patient	Age (yrs)	Gender	MI	Time to rupture (hrs)	Angio-plasty	Thrombolysis	Time to thrombolysis (hrs)	Hypertension	Diabetes	Effusion admission	EF (%)	Pericardiocentesis	Surgery	Outcome
1	69	M	I-P	48	No	No	NA	Yes	No	Small	38	No	No	Died
2	71	M	I-P	144	Yes, no PTCA	No	NA	Yes	No	No	42	No	CABG+patch	Alive
3	78	F	L	48	Yes, no PTCA	TPA	6	Yes	Yes	Moderate	40	No	Patch	Alive
4	78	F	I-P	96	Yes, no PTCA	No	NA	Yes	No	No	40	No	No	Died
5	72	M	A-L	9	PTCA*	TPA	3.5	No	No	No	38	Yes	No	Died
6	72	F	A	13.6	No	TPA	11.3	No	No	No	37	No	No	Died
7	73	F	I-P	112.5	PTCA**	No	NA	Yes	No	No	38	No	No	Died
8	94	F	A	14	No	TPA	3	No	No	No	30	No	No	Died
9	65	M	A	13	No	No	NA	Yes	No	No	27	No	No	Died
10	80	M	A	13	No	SK	10	Yes	No	No	30	No	No	Died
11	74	F	I-P	24	PTCA***		NA	Yes	No	No	30	No	CABG+patch	Died
12	76	F	I-P	48	No	No	NA	Yes	No	Small	35	No	Patch	Died
13	77	F	A	9	No	No	NA	Yes	Yes	No	30	Yes	No	Died
14	52	F	I	3	No	No	NA	No	Yes	No	50	Yes	Patch	Died
15	68	M	I	120	No	No	NA	Yes	No	No	43	No	Patch	Alive

* Incomplete revascularization: Rescue PTCA to diagonal, occluded LAD.

** Late PTCA to right coronary artery.

*** Late PTCA to non-culprit LAD, triple-vessel coronary artery disease

Time to rupture = time from beginning of the pain to rupture, Time to thrombolysis = time from beginning of the pain to thrombolysis, TPA = tissue plasminogen activator, SK = streptokinase, CABG = coronary artery bypass grafting, PTCA = percutaneous coronary angioplasty, LAD = left anterior descending artery, A = anterior, I = lateral, A-L = anterolateral, I = inferior, I-P = inferoposterior.

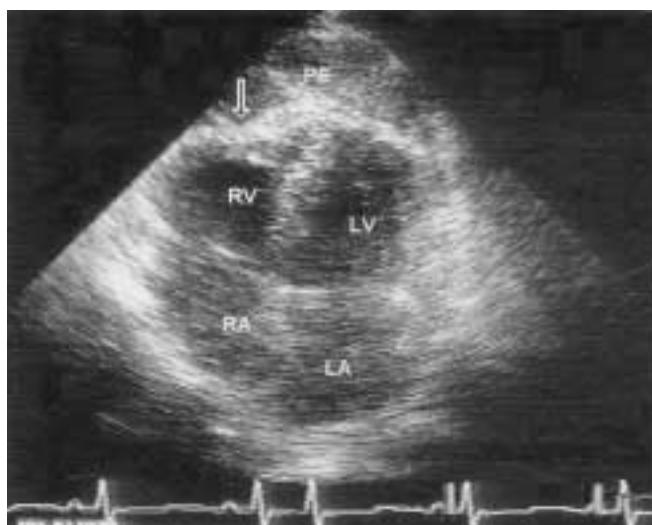


Figure 1. Four-chamber view of the patient with recurrent cardiac rupture. The arrow depicts diastolic compression of the right ventricle by the pericardial effusion. LV = left ventricle, RV = right ventricle, LA = left atrium, RA = right atrium, PE = large pericardial effusion with clot.

ventricular wall remodeling [1]. Rupture occurs more frequently in elderly patients, in women, following the first myocardial infarction, especially anterior, without previous angina and often without evidence of collateral flow. Late thrombolysis (more than 14 hours) may be another risk factor for rupture [1]. All the patients in this series were admitted with a first myocardial infarction.

According to Topol et al. [2] there are three types of cardiac rupture. Type I occurs on the first day of anterior MI due to single-vessel occlusion and can be potentiated by thrombolytic therapy. The proposed mechanism is coronary occlusion followed by reperfusion with proteolytic enzymes and metalloproteinase activation.

Table 2. Clinical characteristics of the patients

Age (yrs)	73 (52–94)
Male/Female	6/9
Hypertension	11
Diabetes mellitus	3
Left ventricular ejection fraction	37% (25–50)
First ST elevation MI	All
Time from the beginning of the pain to rupture	48 hrs (3–144)
Thrombolytic therapy	5
Time from the beginning of the pain to thrombolysis	6 hrs (3 – 11.3)
Adequate primary PTCA*	0
Pericardial effusion on admission	3
Pericardiocentesis	3
Surgery	6
Alive	3

* PTCA was performed in three patients, in one after 18 hours, and in two patients the culprit artery was not opened.

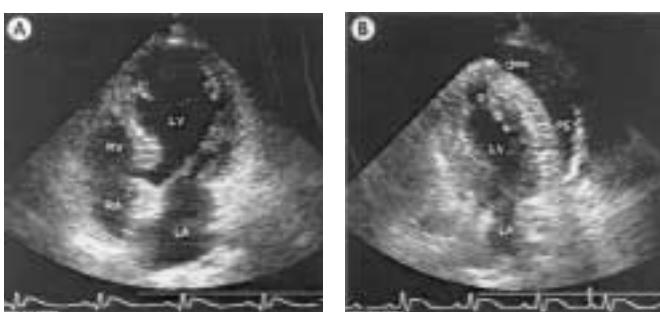


Figure 2. [A] Four-chamber view of a 77 year old woman with acute anterior MI. No pericardial effusion is seen. [B] Four-chamber view of the same patient a few seconds later. Large pericardial effusion (PE) around the left ventricle is seen. Arrow depicts the suspected site of rupture at the apex of the left ventricle. The right heart chambers are not clearly visualized due to collapse. LV = left ventricle, LA = left atrium, RV = right ventricle, RA = right atrium.

tion, subsequent myocardial matrix degradation, and myocardial slit. This type of rupture possibly occurred in six of our patients; four of them had received thrombolytic therapy. The second type of rupture occurs due to a small to moderate, usually posterior myocardial infarction in patients with multivessel coronary artery disease during the first days of acute MI, and may also be associated with thrombolysis. The third type of rupture is late, is usually associated with large anterior myocardial infarction, and may be prevented by early revascularization. Among 1,375 patients with acute ST elevation [3], MI primary angioplasty was an independent protective factor with overall frequency of free wall rupture of 1.8% compared with 3.3% following thrombolytic therapy. Age over 70 years, anterior location, female gender, and administration of treatment more than 2 hours after the onset of symptoms were associated with a higher rate of free wall rupture [3]. None of our patients had appropriate revascularization; angioplasty was performed in three patients, but in one it was late and in two it did not include the culprit artery. The clinical presentation of the patient with recurrent rupture, occurring not in the setting of an ST elevation MI was highly unusual. The proposed mechanism was probably related to ischemia and stretch of the ischemic myocardium in the absence of revascularization before the first rupture and inadequate operation.

Pericardial effusion due to rupture must be differentiated from pericardial involvement after a large myocardial infarction. As demonstrated by Figueras and colleagues [4], mild pericardial effusion within the first 2 days in patients over the age of 60 with a first ST elevation acute MI usually propagates into late moderate to severe pericardial effusion, while in the setting of an acute MI moderate to severe pericardial effusion is most frequently associated with hemopericardium, and many of these patients have already developed free wall rupture.

Echocardiographic predictors of cardiac rupture are early pericardial effusion – even relatively small – during the first 2 days of MI [4], echocardiographic signs of right atrial/ventricular compression, and evidence of a clot, namely high density intrapericardial echoes [5]. The clot is usually anterior, independent of the site of the rupture, and is usually not visible. In our series pericardial effusion on admission was found in 3 of the 15 patients.

In up to 40% of cases with cardiac rupture death occurs subacutely over a matter of hours, not minutes [6]. In fact, early pericardial effusion with subsequent propagation to a large effusion is a manifestation of subacute rupture with slower hemorrhagic leakage into the pericardium and thrombus formation at the site of rupture. In these cases a subacute course allows correct diagnosis and successful treatment. Among 1,247 consecutive patients with acute MI, subacute rupture occurred in 33 [7]. Pericardial effusion greater than 5 mm, high density intrapericardial echoes or right atrial/right ventricle wall compression had a sensitivity greater than 70% and specificity greater than 90% for the diagnosis of rupture [7]. The identification of intrapericardial echo densities suggesting clots, in the absence of cardiac tamponade, allowed a diagnosis of subacute rupture [8]. In these cases contrast echocardiography may

be useful for diagnosis of the active bleeding into the pericardium [9]. When subacute cardiac rupture is suspected, prolonged bedrest and blood pressure control to systolic values lower than 120 mmHg with beta-blockers are favorable [10].

In patients with cardiac tamponade, immediate pericardiocentesis should be performed as soon as the diagnosis is made and the patients should be taken to the operating room. Emergency thoracotomy with surgical repair is the definitive treatment and is the only chance for survival.

Conclusions

Early pericardial effusion, right atrial/ventricle compression and intrapericardial clot in the setting of ST elevation MI should alert the clinician to the possibility of cardiac rupture. Adequate and prompt revascularization can decrease the occurrence of cardiac rupture. All patients with ST elevation myocardial infarction should undergo echocardiography prior to thrombolytic therapy or primary angioplasty.

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