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CASE REPORTS

Cardiac Tamponade Early After Thrombolysis for Acute Myocardial Infarction: A Rare but Not Reported Hemorrhagic Complication

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Among 392 consecutive patients admitted for acute myocardial infarction and treated with thrombolytic drugs, 4 patients (1%) developed an early hemorrhagic pericardial effusion (without ventricular wall rupture) evolving within 24 h to cardiogenic shock consequent to cardiac tamponade. They all suffered from a large anterior myocardial infarction treated within 4 h after onset of symptoms with intravenous anisoylated plasminogen streptokinase activator complex (one case), recombinant tissue-type plasminogen activator (rt-PA) (two cases) or streptokinase (one case), anticoagulation with heparin (all cases) and aspirin (three cases).

As soon as pericardial effusion was established by echocardiography, emergency percutaneous pericardiocentesis was performed at the bedside 20 ± 6 h after thrombolytic therapy was started. This corrected immediately the clinical and hemodynamic status of each patient and a catheter was left in the pericardial space for 34 ± 18 h. Thus, in the presence of unexplained clinical and hemodynamic deterioration occurring during the first 24 h after thrombolytic treatment of a large myocardial infarction, cardiac tamponade should be suspected. Immediate percutaneous pericardiocentesis followed by continuous drainage is a simple and definitive treatment for this complication.

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Many large controlled trials (1–8) have demonstrated that thrombolysis performed during the first hours after the onset of symptoms in patients with acute myocardial infarction decreases infarct size, limits left ventricular dysfunction and reduces hospital mortality irrespective of the type of thrombolytic agent used. Accordingly, the rate of the most common infarct-related complications has been favorably affected by thrombolysis. Hemorrhage is the major specific side effect of thrombolytic agents (9–11). With an incidence of <1% in most reported studies, cerebral hemorrhage represents the most serious bleeding. On the other hand, pericardial bleeding (primary hemopericardium) represents a well-known complication of chronic anticoagulation but was very uncommonly reported as an early complication after thrombolytic therapy for acute myocardial infarction except in case of rupture (secondary hemopericardium).

We report the clinical, hemodynamic and angiographic data of four patients who presented with cardiac tamponade due to pericardial hemorrhage during the first 24 h after intravenous thrombolysis for acute myocardial infarction in the absence of ventricular wall rupture.

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Methods

Study patients. We reviewed the clinical records of 392 consecutive patients admitted to the coronary care unit between 1984 and June 1989 for acute myocardial infarction and treated with various thrombolytic drugs. We excluded from this study three cases of hemopericardium observed after thrombolysis for myocardial infarction. One occurred at pacemaker removal 2 days after thrombolysis and was obviously related to a right ventricular perforation. The two remaining cases were observed late after thrombolysis, 16 and 15 days respectively, in patients treated with oral anticoagulants. Successful pericardiocentesis was performed in each case.

Treatment protocol. Different thrombolysis protocols were applied to these patients: 250,000 IU streptokinase intracoronary (n = 42), 500,000 IU streptokinase intravenously (n = 249), 1,500,000 IU streptokinase intravenously (n = 40), 30 mg anisoylated plasminogen streptokinase activator complex (APSAC) intravenously (n = 11) and 100 mg recombinant tissue-type plasminogen activator (rt-PA) intravenously (n = 50). Detailed protocols have been previously published using streptokinase (11), APSAC (11,12) or rt-PA (4,13). All patients were given heparin before and during thrombolysis (intravenous bolus of 5,000 to 10,000 IU) followed by an infusion of 600 to 1,200 IU per h for 3 to 4 days. Concomitant therapy was usually limited to analgesics and nitroglycerin or isosorbide dinitrate infusions. In case streptokinase or APSAC was given, treatment with aspirin (100 to 300 mg per day orally) was started after

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Table 1. Clinical Data in Four Patients

	Patient			
	1	2	3	4
Age (yr)	60	67	67	69
Gender	M	M	M	F
MI location	ANT	ANT	ANT	ANT
Time delay from symptom onset to thrombolysis (min)	145	275	190	250
IV Thrombolytic agent used	APSAC (30 mg)	rt-PA (100 mg)	rt-PA (100 mg)	SK (1×10^6 IU)
Concomitant therapy	IV HEP IV ASA	IV HEP IV ASA	IV HEP IV ASA	IV HEP —
Time delay from symptom onset to peak CK (h)	5.5	9	10.5	11
Peak CK (IU/liter)	2,180	10,370	7,220	5,740

ANT = anterior; ASA = aspirin; CK = serum creatine kinase; HEP = heparin; IV = intravenous; MI = myocardial infarction; SK = streptokinase.

heparin infusion was discontinued. For patients treated with rt-PA, aspirin therapy was started on admission.

Patient monitoring. In the coronary care unit all patients were continuously monitored by electrocardiogram (ECG) and invasive arterial pressure measurements during 3 to 4 days. Right heart catheterization was performed when clinically indicated in case of extensive infarction or heart failure. Serial creatine kinase (CK) determinations (normal <160 IU) were performed every 3 h during the first 24 h and every 6 h during the next 2 days. Echocardiography (ADR 4,000 S-LC unit with a medium focus 3 MHz transducer) was available 24 h a day but was not routinely recorded on admission except for specific clinical reasons. Coagulation studies were performed in some subgroups of patients, as reported in detail elsewhere (11).

Diagnosis and treatment of cardiac tamponade. The diagnosis of cardiac tamponade was made in the presence of the following criteria: 1) new and unexplained development of clinical signs of shock: hypotension, tachycardia and poor peripheral perfusion; 2) hemodynamic data compatible with tamponade: diastolic elevation and equalization of right atrial (>10 mm Hg), pulmonary artery and pulmonary capillary wedge pressures; 3) presence of a paradoxical pulse exceeding 10% of the systolic pressure during normal respiration; and 4) echocardiographic evidence of pericardial effusion.

For pericardiocentesis after local anesthesia, a 21 gauge needle was advanced in the subxiphoid space directed toward the left midclavicle under pressure control. Once blood could be withdrawn, recording the intrapericardial pressure insured the proper location of the needle. Then a 0.024 in. and 30 cm long guidewire was inserted and a 20 cm long 4 F catheter (Seldicath, Plastimed, France) was advanced by the Seldinger technique and left in the pericardial space.

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Among the 392 patients with acute myocardial infarction treated by thrombolysis, 4 patients (1%) developed a documented hemorrhagic pericardial effusion, evolving in <24 h to a clinical and hemodynamic syndrome of cardiac tamponade.

Clinical and hemodynamic data (Tables 1 and 2). All four patients were >60 years old and were admitted while developing a large anterior myocardial infarction. The mean time delay between onset of symptoms and thrombolysis was 215 ± 59 min. Tamponade occurred with each of the three thrombolytic compounds used. Early serum CK peaking after symptom onset (9 ± 2.5 h) was consistent with a successful reperfusion in each case. Excessively high peak CK levels were observed in Patients 2, 3 and 4 (>5,500 IU) suggesting a large infarct size.

Heart rate and arterial systolic and diastolic pressures were normal at the beginning of the thrombolytic infusion (Fig. 1). Chest pain was present at the initiation of treatment and disappeared or decreased significantly during or after thrombolytic therapy. Only Patient 4 had no real improve-

Table 2. Characteristics of the Pericardial Effusion in Four Patients

	Patient			
	1	2	3	4
Time delay from thrombolysis to P.P. (h)	21	12.5	26	20
Intrapericardial pressure (mm Hg)				
At P.P.	12	12	17	15
At CATH removal	5	5	7	6
Pericardial fluid hematocrit (%)	37	40	35	43
Pericardial drainage duration (h)	26	13	45	54
Total pericardial fluid removed (ml)	625	410	280	370

CATH = intrapericardial catheter; P.P. = pericardial puncture.

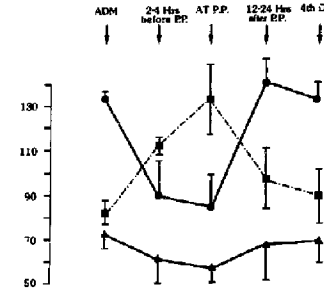

Figure 1. Hemodynamic pattern (mean values ± 1 SD) during the first 4 days after admission and thrombolysis in four patients with cardiac tamponade. Square = heart rate (beats/min); circle = systolic blood pressure (mm Hg); triangle = diastolic blood pressure (mm Hg); ADM = on admission; P.P. = pericardial puncture.

ment of her chest pain immediately after thrombolysis. Patient 3 complained of reappearance of chest discomfort 12 h after thrombolysis and this was associated with the presence of a typical pericardial friction rub.

After a time delay of 14 ± 6 h, we observed in each case a progressive, but significant, hemodynamic deterioration. The clinical syndrome was characterized by tachycardia, hypotension, tachypnea and signs of serious reduction of peripheral perfusion suggestive of cardiogenic shock. Hemodynamic data were compatible with cardiac tamponade and echocardiography confirmed in each case the presence of a significant pericardial effusion. Emergency pericardiocentesis was successfully performed at the bedside in the four cases. In Patient 3 respiratory arrest required the use of endotracheal intubation simultaneously with pericardial puncture and mechanical ventilation during 4 h. The mean time delay between thrombolysis and puncture was 20 ± 6 h. The mean intrapericardial pressure was 14 ± 3 mm Hg (Table 2). After rapid removal of about 100 ml of pericardial blood, a dramatic clinical and hemodynamic improvement was observed and maintained in each patient. The intrapericardial catheter was left in place for continuous drainage during 34 ± 18 h and the mean volume removed during this period was 421 ± 126 ml.

Twenty four hours after the pericardial drainage the clinical status of each patient remained significantly improved, as confirmed by hemodynamic data that had returned to baseline values (Fig. 1). Nevertheless, each patient suffered from pericardial chest pain during 12 to 24 h after pericardial fluid removal associated with a typical pericardial rub. Echocardiograms were performed daily during 4 days and confirmed the disappearance of pericardial effusion. No other major complication occurred and patients were dismissed from the coronary care unit after 7 ± 3 days.

Coagulation data (Table 3). Baseline values of fibrinogen and thrombin clotting time were within the normal range before treatment. The fibrinogen level was significantly depressed during the first 12 h after APSAC infusion as opposed to rt-PA infusion where a <50% decrease was observed. In each case the thrombin time was highly prolonged (>120 s) as a consequence of full heparinization before cardiac tamponade and pericardial puncture. At that time heparin was stopped and neutralized using protamine sulfate in two patients. No plasmin inhibitors were given. In Patient 4 low-dose heparin was started again after pericardial puncture, which explains the persistent prolonged thrombin time.

Angiographic data (Table 4). In Patients 1 and 2 early coronary patency was confirmed by angiography 5 and 2 h, respectively, after thrombolysis and for Patient 2 immediate coronary angioplasty was successfully performed on the infarct-related stenosis located in the proximal left anterior descending artery. These procedures were part of specific protocols (12,13).

Angiographic evaluation was performed after a few days for Patient 3 (11 days) and Patient 4 (3 days). The absence of high-grade residual stenosis in the left anterior descending artery was noted in each patient. The residual stenosis was

Table 3. Coagulation Data in Four Patients

Patient		Before Thrombolysis	After Thrombolysis 6 h	12 h		24 h		48 h
1	Fg	230	0	0	*	155		290
	TT	20	>120	>120		33		21
2	Fg	280	170	175	*	230		350
	TT	21	>120	>120		38		19
3	Fg	224	167	210		337	*	539
	TT	26	120	120		120		23
4	Fg	NA	NA	244	*	NA		387
	TT	20	>120	>120		>120		>120

*Time interval during which pericardial puncture was performed (between 12 and 24 h in Patients 1, 2 and 4 and between 24 and 48 h in Patient 3). Fg = fibrinogen (mg/100 ml; normal range 150 to 350); NA = not available; TT = thrombin time (seconds; normal range 18 to 22).

Table 4. Angiographic Data in Four Patients

	Patient			
	1	2	3	4
Time delay between thrombolysis and angiography	5 h	2 h	11 days	3 days
Infarct-related vessel	LAD	LAD	LAD	LAD
LAD residual stenosis (%)	50 to 75	50 to 75 (<50)*	<50	<50
Number of diseased vessels (>50%)	3	1 (0)*	0	0
Ejection fraction (%)	34	44	29	43

*Immediate and successful angioplasty was performed immediately after diagnostic coronary angiography. LAD = left anterior descending artery.

estimated as 50% to 75% in luminal diameter in Patient 1 (three-vessel disease) and Patient 2 (one-vessel disease treated by angioplasty). In patients 3 and 4 the residual stenosis was estimated as <50%, so that no significant coronary stenosis could be demonstrated in these patients. However, severe left ventricular dysfunction was observed in each case confirming the extensive myocardial damage in spite of reperfusion. Anterior systolic dyskinesis was present except in patient 2 in whom only anterior akinesis was observed.

Follow-up data. Patients 1, 2 and 4 had an uneventful in-hospital evolution after the CCU period. They performed a maximal bicycle exercise test associated with thallium 201 scintigraphy at a mean of 14 ± 4 days after admission. In each case there were no clinical or ECG signs of myocardial ischemia. Thallium scintigraphy showed a large nonreversible anterior perfusion defect in each case. After a mean follow-up of 33 months (range 5 to 54), these three patients were alive and free of cardiac symptoms.

In patient 3 subacute myocardial rupture was suspected because he showed the most severe left ventricular dysfunction with extensive anterior dyskinesis. Therefore, aneurysmectomy was contemplated and performed 18 days after admission. No macroscopic evidence of previous myocardial rupture could be observed, as well as no abnormal feature in the pericardial cavity. However, the patient died 3 days after the operation because of intractable ventricular arrhythmias.

Discussion

Hemorrhage is the major specific side effect of thrombolytic therapy. According to the data of the major clinical trials evaluating thrombolysis in acute myocardial infarction (1–10), the most severe complication is related to cerebral hemorrhage with an incidence of 0% to 1.7% usually occurring within the first 48 h after treatment and with a mortality rate of 20% to 50%. In our experience of 392 consecutive patients treated with various thrombolytic agents, only one fatal cerebral accident was observed (0.3%). However, during the same period we noted in four cases the rapid development of hemopericardium with cardiac tamponade occurring in the first hours after thrombolytic therapy. Because these cases were not related to cardiac rupture and secondary hemopericardium, this complication must be considered as a major primary hemorrhagic complication of thrombolytic treatment associated with anticoagulation. Early recognition of this life threatening complication allowed proper treatment using percutaneous pericardial drainage.

Previous reports. In a large review by Yusuf et al. (14) of 33 earlier studies using intracoronary or intravenous thrombolysis in acute myocardial infarction, no case of early hemopericardium was observed in >7,000 patients. Similarly, no case of primary hemopericardium or cardiac tamponade was documented in the major randomized clinical trials (1–10), published since 1986, that used different thrombolytic drugs and included more than 23,000 patients.

To the best of our knowledge, only three cases of late hemopericardium have been documented and reported (15,16), all three cases occurring after intracoronary streptokinase infusion. Surgical pericardial drainage was performed several days after thrombolytic treatment (3rd, 9th and 10th day, respectively). However, in our patients an emergency pericardiocentesis followed by prolonged continuous catheter drainage was performed within the first 26 h after intravenous thrombolysis because of early cardiac tamponade with cardiogenic shock.

Several reasons may explain the absence of similar reports to date. The first one could be related to a clustering phenomenon for this exceptional complication in our institution during this particular time period. On the other hand, the diagnosis of tamponade may have been missed if hemodynamic or echocardiographic monitoring, or both, were not available or used. The clinical pattern of early and evolving cardiogenic shock can be regarded as infarct-related left ventricular failure or as subacute cardiac rupture. In addition, even when the diagnosis of tamponade is suspected, the physician may hesitate to perform pericardiocentesis in the presence of coagulation defects. With these considerations in mind, it is possible that some early deaths reported as "cardiogenic shock" or "suspected myocardial rupture" in the different studies previously reported (1–10) could have been related to unrecognized cardiac tamponade. This suspicion is reinforced by data from the Gissi trial (17) where a surprising and unexplained excess of death was observed on the first day in treated patients as compared with the placebo group (31.8% of the 628 versus 20.2% of the 758 deaths, respectively, that occurred during the 1st week).

Pathophysiology. In the absence of cardiac rupture, some cases of hemopericardium were reported before the thrombolysis era either as spontaneous complications or more frequently associated with anticoagulant therapy after infarc-

tion (18). These cases were reported as occurring a few days after infarction but not during the first 24 h.

Pericardial bleeding is also observed in absence of myocardial infarction in nonspecific pericarditis or during anticoagulation therapy (18,19). Another case was reported (20) after intravenous thrombolytic treatment of acute pulmonary embolism. Accordingly, the hypothesis that could explain the early cardiac tamponade in our patients implies the association of two triggering factors. The first is the occurrence of a large anterior transmural infarction with an early form of pericarditis, as clinically evidenced in one of our patients. It is noteworthy that the three previously reported cases (15,16) also involved a large anterior infarction. The second factor is the major alteration in coagulation related to the combination of thrombolytic agents, heparin and aspirin. Such drug association is not unusual and was used in most recent large scale trials of thrombolysis in acute myocardial infarction (3,4,13). In addition, the coagulation data demonstrate that a profound systemic lytic state was not present in our two patients treated with rt-PA. In this hypothesis the drug regimen used to obtain sustained coronary recanalization cannot, in itself, be the only triggering factor. It is surprising to note that in each of our patients coronary angiography showed good patency of the infarct-related left anterior descending artery without severe residual stenosis. A relatively brisk myocardial reperfusion after prolonged acute ischemia in a large area at risk can lead to an hemorrhagic infarction, as demonstrated by experimental studies (21,22). However, none of these studies reported the association of hemorrhagic myocardial infarction and hemopericardium. Similarly, a few postmortem studies (23–26) documented myocardial hemorrhagic necrosis in patients treated with intracoronary or intravenous thrombolysis but no associated hemopericardium was reported. This suggests the absence of a direct relation between early myocardial reperfusion and pericardial bleeding within the first 24 h.

Treatment. According to the unclear pathophysiology of early hemopericardium occurring within the first hours after thrombolysis for myocardial infarction, optimal treatment—percutaneous or surgical pericardial drainage—remains questionable. Some authors (27–29) have reported dramatic clinical and hemodynamic improvement after pericardiocentesis performed for a documented (postmortem or postoperative) subacute myocardial rupture. In some patients only a temporary improvement was observed because a fatal rupture eventually occurred; in others this improvement allowed a successful surgical repair of a subacute rupture. However, no case of prolonged percutaneous pericardial drainage without surgical intervention was reported.

In our four patients, because of their precarious clinical status, immediate pericardiocentesis was performed and continuous pericardial drainage maintained for more than 24 h, allowing the removal of more than 300 ml in each case. These blood volumes are comparable with those in previously reported cases (600, 450 and 400 ml) (15,16). This technique appears more rapid and simple for acute patients with evolving cardiac tamponade and cardiogenic shock, as compared with emergency surgery. In addition, pericardiocentesis allows the patient's condition to improve and stabilize in case surgery should be eventually performed.

Conclusions. Early pericardial hemorrhage occurring after thrombolysis for acute myocardial infarction should be considered as a major complication whose incidence may be comparable with that of cerebral hemorrhage. Therefore, in the presence of unexplained clinical and hemodynamic deterioration occurring during the first hours of thrombolytic therapy of a large acute myocardial infarction, echocardiographic examination must be rapidly performed to exclude this potentially lethal, but treatable, complication. Bedside pericardiocentesis followed by prolonged pericardial drainage is a simple, immediate and safe treatment yielding a good prognosis. Early hemorrhagic pericarditis may occur during the first hours after a large acute transmural myocardial infarction in patients treated with anticoagulant, antiplatelet and thrombolytic agents.

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