Are We Able to Prevent Death Due to Postinfarction Cardiac Rupture by Early Diagnosis and Surgical Treatment?

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SUMMARY

Death from postinfarction cardiac rupture (PCR) may occur in two ways. There are patients who die suddenly without symptoms or with symptoms of less than an hour's duration (sudden cardiac death), whereas other patients die several hours after the onset of the rupture. Ninety-six patients who died from AMI and underwent autopsy have been studied. Sixteen patients displayed a rupture of the free wall of the left ventricle at the site of infarction, cardiac hypertrophy and severe coronary alterations. Six of these patients were included among patients who died suddenly. The other 10 showed signs before death that occurred from 240 to 660 min after their appearance. Signs of impending rupture were appearance or increase of chest pain that was not improved by opiates, preterminal sinus rhythm with an unchanged ST-segment and echocardiographic infarct expansion or pericardial effusion. We propose that the emergence of these signs during AMI suggests an impending rupture. Early surgical intervention is essential to save those patients who survive several hours after the initial signs of PCR.

Key Words:
Rupture of the heart Myocardial infarction Surgery

HARVEY[1] described the first case of cardiac rupture in 1647. The frequency of reports of this finding at postmortem examination has increased progressively: approximately 10–20% of the deaths from acute myocardial infarction (AMI) are the result of postinfarction cardiac rupture (PCR).[2)–6)] Clinico-pathologic studies have yielded clues regarding mechanisms that can cause cardiac rupture. During the last decade there has been growing evidence that factors such as transmurality of the first infarction,[2)–8)] cardiac hypertrophy,[3)–7),9)] two-dimensional echocardiographic evidence of infarct expansion,[8)] hypertension[2),7),10),11)] and severe lesions of the coronary arteries[3),4)] are associated with an increased risk of cardiac rupture. In addition, the clinical progression of PCR follows two courses. First, there

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are patients who die suddenly without symptoms or with symptoms of less than an hour's duration; these people die suddenly. By contrast, other patients die several hours after the rupture has formed. Therefore, early markers of impending rupture should permit surgical procedure to save these patients. This study investigated potential diagnostic markers of impending rupture during AMI.

Materials and Methods

Ninety-six autopsy cases (cause of death: AMI, ages: 46–91 years) were included in the study. Sixteen patients (12 males and 4 females) experienced PCR. The other 80 cases (58 males and 22 females) were used as a control group. The age, sex, electrocardiogram, type of chest pain, physical exertion before death, diabetes, hypertension, interval from admission to death and interval from the onset of symptoms before the rupture to death were available for all cases. Two-dimensional echocardiography data were available for only 4 patients with PCR.

The postmortem examination of the heart was conducted with previously described methods.[12] The heart was removed by severing the pulmonary artery and aorta about 5 cm from the free margin of the semilunar valves. After observing the external aspect of the heart to identify the areas of myocardial damage, without opening the coronary vessels, a rubber plug was placed through the aorta into the aortic orifice (Fig. 1) and then the coronary arteries were injected at a pressure of 130 mmHg using a barium-iodine-gelatin radiopaque mass by a cannula tied into the aorta. When a good degree of contrast was noted in x rays, the heart was fixed in 10% formalin solution and cut into 5 to 6 transverse slices (thickness approximately 1 cm) from the apex to the base (Fig. 2). These slices were parallel to the atrioventricular sulcus. The thickness of the left ventricular wall at the level of the third slice was measured, the location and age of the infarct were recorded and its percent size quantified in comparison to the total myocardial area from the photographs by means of a polar planimeter. Every major coronary artery and its branches were cut transversely along their length at intervals of approximately 5 mm. If alterations were seen, their length and degree were recorded.

Microscopic examination of the myocardium was carried out after hematoxylin-eosin staining. Hematoxylin-eosin and Weigert were used to stain the vessels. For the myocardium, both infarcted tissue and the infarct borders were evaluated as described by Mallory.[13] For the coronary arteries, a total of 128 segments were studied from patients with PCR. The internal

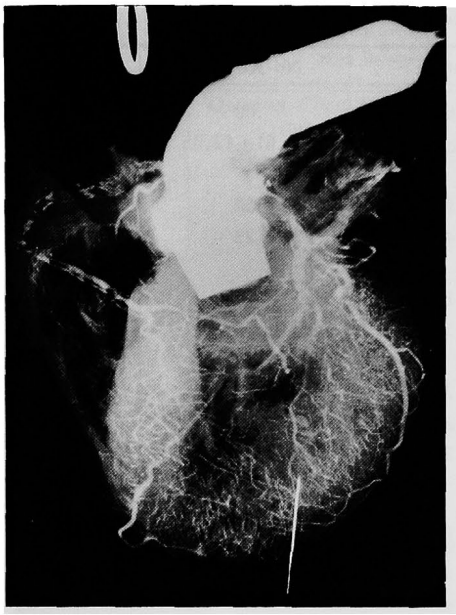

Fig. 1. Illustration of the technique for the postmortem coronary angiography. A rubber plug through the aorta is placed into the aortic orifice and the coronary tree is injected by a radiopaque mass through a cannula tied into the aorta.

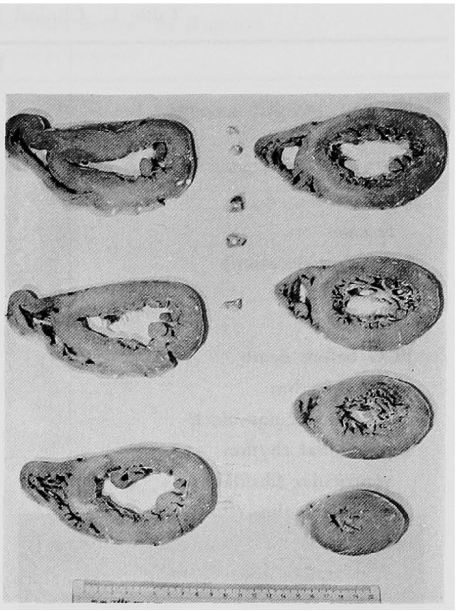

Fig. 2. Method of examination of the heart. Parallel slices of approximately 1 cm thickness.

diameter of the vessel was measured with an ocular micrometer. If stenoses were observed, their degree and length were calculated as the percentage of the normal internal diameter of the vessel. The degree of narrowing was divided into 4 classes: 0–30%, 31–60%, 61–90%, and over 90%.

Clinico-pathologic correlation and statistical method: The age of the patients with and without rupture as well as heart weight, thickness of the left ventricular wall and percent size of the infarction were compared using the t-test. A p value less than 0.01 was taken to denote statistical significance. All data are presented as means$\pm$SD.

Results

Clinical data (Table I)**:** All patients included in the study ranged in age from 46 to 91 years (mean: 66.4$\pm$12.4 years for the people who exhibited PCR and 64.6$\pm$12.4 years for the people without rupture). There was no significant difference between the 2 groups. Eleven of the 16 patients with PCR disclosed an anterior infarction; the remaining 5 cases had inferior

Table I. Clinical Features of the Patients

	Rupture	No Rupture	
Number	16	80	
Mean age	66.4±12.35	64.61±12.35	p=NS
Sex			
male	12	58	
female	4	22	
Infarction (location)			
anterior	11	56	
inferior	5	24	
ECG before death			
sinus rhythm	12		
atrioventricular block	2		
junctional rhythm	2		
ventricular fibrillation		67	
agonal rhythm (standstill)		13	
Severe chest pain before death	10	2	p<0.01
Echocardiography			
infarct expansion	2	0	
pericardial effusion	2	0	
Hypertension	6	9	p<0.01
Diabetes		12	
Physical exertion	0	1	
Death from the onset of AMI	12 hours—7 days	2 hours—25 days	

Table II. Type of Death in Patients with PCR

Death	N° Patients
Sudden death	6
without symptoms	4
with symptoms 1 hour duration	2
Late death	
with premonitory symptoms	10
Total	16

infarctions. Six cases died suddenly (Table II), either without premonitory symptoms or with symptoms of less than an hour's duration. The recorded electrocardiogram showed sinus rhythm in 2 cases, atrioventricular block in 2 cases and junctional rhythm in the remaining 2 cases.

Ten subjects (Table III) had a reappearance of chest pain that was not controlled effectively by treatment with opiates. Their electrocardiograms showed sinus rhythm with unchanged ST-segments before death, which occurred from 240 to 660 min (mean: 368 min) after the onset of chest pain.

Table III. Appearance of Chest Pain Before the Rupture and Recorded Electrocardiogram

N°	Onset of Chest Pain (min)	Recorded Electrocardiogram
1	240	Sinus rhythm with unchanged ST-segment
2	240	"
3	240	"
4	310	"
5	310	"
6	360	"
7	360	"
8	420	"
9	540	"
10	660	"

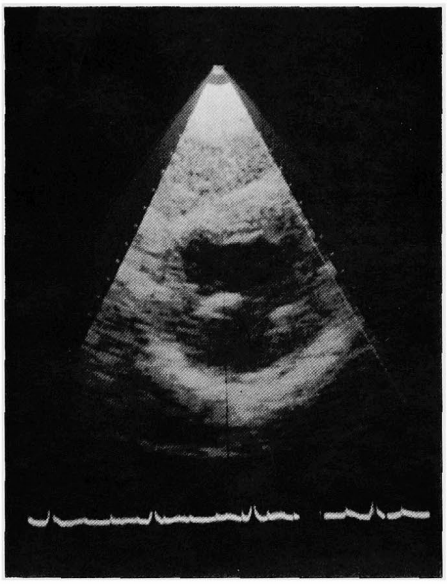

Fig. 3. Two-dimensional echocardiography. Apical dilatation and akinesis (infarct expansion) before the rupture.

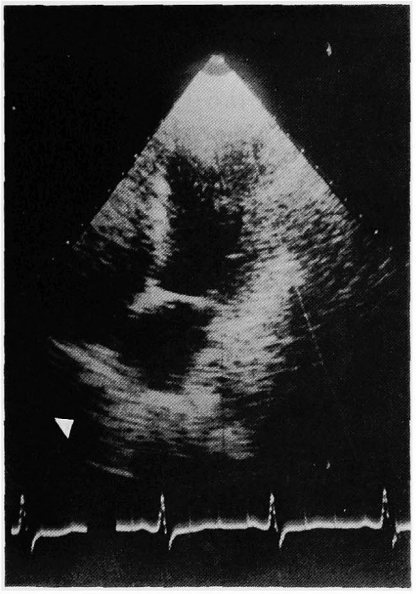

Fig. 4. Two-dimensional echocardiography. Pericardial effusion during rupture (arrow).

Death in the patients with PCR (Table I) occurred from as early as 12 hours to as late as 7 days after infarction.

Only 4 subjects with rupture underwent echocardiography. An area of thinning throughout the cardiac cycle as well as regional dilatation and akinesis at the site of the infarction were seen in 2 patients (Fig. 3). The other 2 cases showed pericardial effusion (Fig. 4). Six patients had hypertension

(systolic blood pressure from 180 to 220 mmHg). No physical exertion before the rupture was found nor was there any evidence of diabetes.

The patients without rupture (Table I) had the same prevalence of anterior and inferior infarction as the PCR cases. Anterior infarction appeared in 56 cases, while inferior infarction was observed in 24 cases. Only 2 cases (2.5%) felt severe chest pain before dying, but their preterminal electrocardiogram showed sinus rhythm with ST elevation as evidence of a recurrent ischemic attack; this was immediately followed by agonal rhythm. Finally, 9 patients had hypertension. These two signs (severe chest pain and hypertension) were compared with a t-test. A p value less than 0.01 was observed between the group with PCR and the group without PCR.

Pathologic data: Of 16 hearts with rupture, 11 showed anterior AMI and 5 inferior AMI. The heart weights ranged from 390–1,020 grams (mean: 627.50±201.1 g). The wall thickness of the left ventricle ranged from 18–30 mm (mean: 25.17±3.61 mm) and the percent size of infarction from 25–55% (mean: 44.2±12.0%). These parameters differed significantly ($p<0.01$) between the PCR and control groups (Table IV). Severe coronary alterations (Table V) were found in the patients with PCR. The left anterior descending artery (LAD) was affected totally by stenoses in 17 instances, the right coronary artery (RC) in 9 instances, the left circumflex artery (LC) and the left main coronary artery (LM) twice. Occlusive thrombi were found in 11 instances. Microscopic examination of the heart showed his-

Table IV. Statistical Correlation of Some Parameters in the Hearts With and Without PCR

Parameters	Rupture	No Rupture	t-test	P
Heart weight	627.50±201.05	400.80±73.40	6.73	<0.01
Wall thickness	25.17±3.61	14.54±2.67	11.74	<0.01
% Infarction	44.17±12.03	30.71±9.79	4.15	<0.01

Table V. Pathologic Features of Coronary Arteries

Coronary Artery	Occlusive Thrombus	% Luminal Narrowing			
		0–30	31–60	61–90	>90
LAD	10	2		4	2
LAD+RC				2	2
LAD+RC+LM				4	
LAD+RC+LC				1	
RC	1			1	

LAD=left anterior descending artery; LM=left main coronary artery; RC=right coronary artery; LC=left circumflex artery.

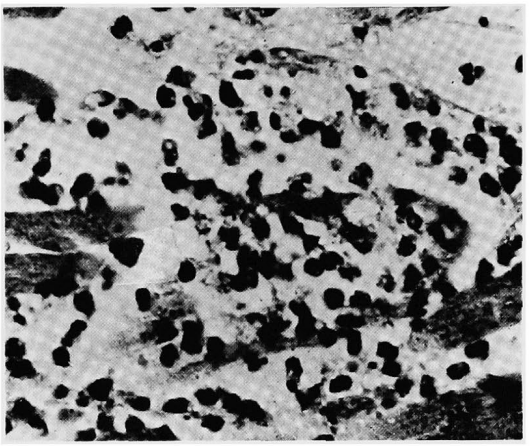

Fig. 5. Myocardial necrosis and massive infiltration of cells around the rupture.

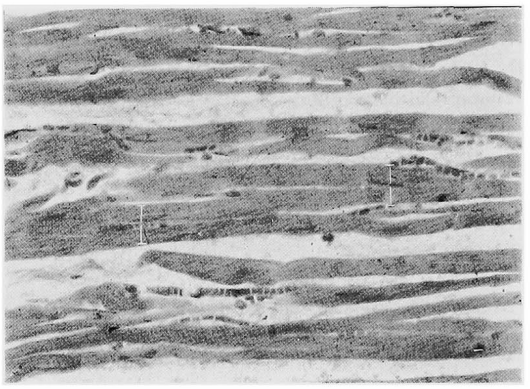

Fig. 6. Hypertrophy of myocardial fibers in a case of cardiac rupture (scale: $-=5\ \mu$).

tologic markers of myocardial necrosis at different stages, massive infiltrates of cells around the rupture (Fig. 5) and hypertrophy of myocardial fibers (Fig. 6).

Discussion

The great majority of patients who develop PCR will die as a function of insufficiency of diagnosis and sudden cardiac death. These factors have precluded a rational surgical approach in all but a few cases. This study and the previous literature[2)-5),8)-17)] raise 3 essential points. First, sudden death occurs in only some patients with PCR. Thus, an appreciable number

Table VI. Markers of PCR

1. Markers intrinsic to anatomoclinical features of the heart
   —Systemic hypertension
   —Cardiac hypertrophy
   —Coronary atherosclerosis
2. Markers correlated to the infarction
   —First infarction
   —Transmurality
   —Sustained hypertension after AMI
3. Markers of impending rupture
   —Appearance or increase of chest pain not improved by opiates
   —Preterminal sinus rhythm with unchanged ST-segment
   —Echocardiographic expansion of AMI

Table VII. Successfully Operated Patients of PCR

References	N° of Patients	Diagnosis
Eisenmann B, 1978, J Thorac Cardiovasc Surg	1	Hemopericardium (Clinical)
Berthoumieu F, 1981, Arch Mal Coeur	1	»
Parr GSV, 1981, J Thorac Cardiovasc Surg	1	Cardiogenic shock
Quereshi SA, 1982, Br Heart J	1	Angiography
Feneley MP, 1983, Br Heart J	2	
Kretz JG, 1984, Coeur	3	
Miyamura H, 1984, Jpn Circ J	1	
Allard-Latour G, 1984, Arch Mal Coeur	1	Hemopericardium (2D Echocardiography)
Commeau P, 1985, Ann Cardiol Angéiol	1	Pericardial clots (2D Echocardiography)
Laurent M, 1985, Coeur	1	»
Pugliese P, 1986, J Cardiovasc Surg	1	Chest pain+Hemopericardium (2D Echocardiography)
Juanema C, 1985, Rev Esp Cardiol	1	Cardiogenic shock (2D Echocardiography)
Denis J, 1987, Arch Mal Coeur	1	»
Ferrini L, 1986, G Ital Cardiol	1	»
Singh RR, 1987, Can J Cardiol	1	»
Stryier D, 1988, Br Heart J	1	»
Araujo A, 1987, Rev Port Cardiol	1	»

of patients are potentially salvageable because they undergo late death.[3),4)] Our observations suggest that there is enough time to plan surgical intervention. The second point is that early signs of impending PCR can be identified. These markers of PCR are listed in Table VI. Finally, the data suggest that only one third of the AMI patients displaying the markers in Table VI die within 1 hour of the onset of symptoms. Thus, a significant propor-

tion of patients are candidates for life-saving surgical intervention.[14),18)-33)]

Clinical, electrocardiographic and echocardiographic patterns have been utilized for the diagnosis of PCR. For successfully operated patients (Table VII), the parameters of diagnosis were electrocardiographic patterns[3),4),34)-36)] of sinus rhythm with unchanged ST-segment, bradycardia and/or junctional rhythm. Echocardiographic patterns of pericardial effusion,[14),25),28)-33)] pericardial clots[24),26)] and infarct expansion[8),37)] have been utilized. Clinical signs of cardiogenic shock[14),18),28)] have also been utilized. These parameters and the factors in Table VI, then, can form a basis for successful surgical treatment of PCR.

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