POSTINFARCTION CARDIAC RUPTURE IN THE NINETIES : DO WE KNOW DETERMINATING FACTORS?

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ABSTRACT

Postinfarction cardiac rupture (PCR) up to the present accounts for approximately 20 percent of autopsy infarcted cases, ranking only behind arrhythmias and cardiac failure in the frequency of AMI complications.

We re-examined our observations of a previous anatomo-clinical study of 96 patients who underwent autopsy after death from AMI. Sixteen patients had rupture of the free wall of the left ventricle at the site of infarction. All the patients with rupture showed the following statistically significant characteristics ($p < 0.01$) if compared to those without rupture : cardiac hypertrophy (heart weight 390 to 1020 gm; mean: 627.5±201 gm; left ventricular wall thickness 18 mm to 29 mm; mean: 25.17± 3.6 mm), sudden death (6 cases) without premonitory symptoms or with symptoms of less than an hour's duration or reappearance of chest pain not improved by opiates before late death, that occurred 240 to 660 minutes from chest pain, recorded electrocardiograms showing sinus rhythm with unchanged ST-segment (12 cases), atrioventricular block (2 cases) and junctional rhythm (2 cases). Hypertension pre-existing to the infarction was seen in 6 cases with rupture versus 9 cases without rupture ($p < 0.01$).

Blood pressure, heart weight and wall thickness of the left ventricle are the most increased parameters in the patients with PCR. Preventive measures against these factors can reduce PCR.

Keywords: heart rupture, infarct, factors.

SINGAPORE MED J 1992; Vol 33: 282-286

INTRODUCTION

Postinfarction cardiac rupture (PCR) up to the present accounts for approximately 20 percent of autopsy infarcted cases, ranking only behind arrhythmias and cardiac failure in the frequency of AMI complications[1-8].

Despite major advances in the surgical techniques over the past ten years [8-13], PCR is still a common cause of in-hospital death. It may involve the free wall of the left ventricle, the interventricular septum, the papillary muscle and, more rarely, the right heart [14,15]. Our experience [7,8] seems to show that the great majority of patients, who develop PCR, undergo rupture of the free wall of the left ventricle at the site of the infarction.

Early markers of impending rupture during AMI[16] can also be identified.

We have re-analysed our previous anatomo-clinical observations to assess whether there had been peculiar characteristics that pre-disposed to the rupture in an attempt to prevent this dramatic occurrence.

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MATERIALS AND METHODS

This study included ninety-six patients who underwent autopsy after in-hospital death from AMI.

Of these patients, sixteen (16.6%), twelve male and four female, aged from 52 years to 83 years (mean: 66.4 ± 7.85 years), disclosed PCR whereas the other eighty (83.4%), 58 male and 22 female, aged from 46 years to 91 years (mean: 64.61 ± 12.35), were used as a control group.

Anatomo-clinical correlation was performed for all patients as follows:

Clinical examination

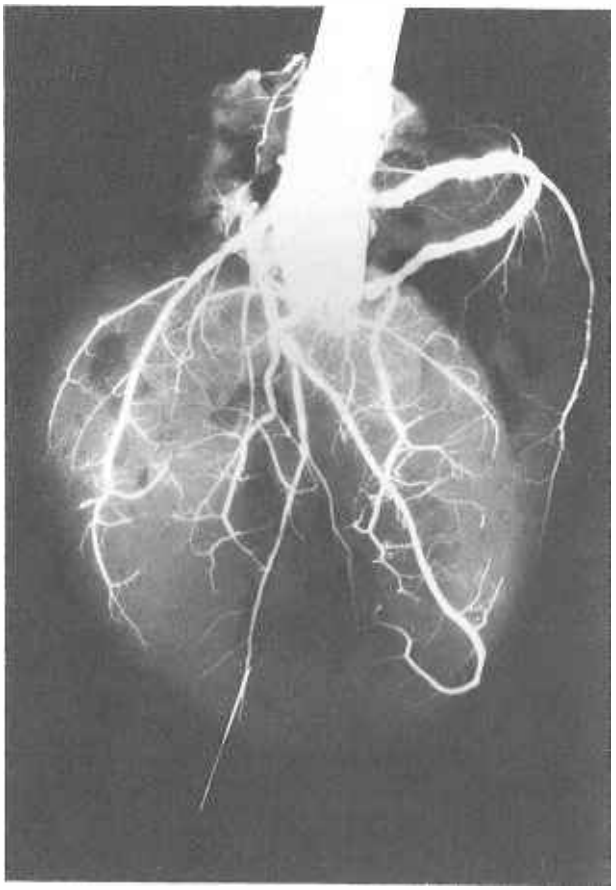
Recorded data in admitted patients were: age, sex, electrocardiogram, type of chest pain, physical exertion before the death, diabetes, hypertension, interval from the admission to the death, interval from the onset of symptoms before the rupture to the death, two dimensional echocardiography in only four patients with PCR.

Postmortem examination of the heart

It was carried out by using a method previously described [17]. In each patient, the heart was removed, 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 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 by means of X-rays, all the hearts were fixed in ten percent water-formalin and then five to six transverse slices of thickness approximately 1 cm were cut from the apex to the base (Fig 2). These slices were parallel to each other and 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 transversely cut 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

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



Weigert were used to stain the vessels. For the myocardium, pieces were taken into the infarcted area as well as around its borders. The histologic patterns of infarction were evaluated as described by Mallory[18]. For the coronary arteries, every removed segment was studied. There were a total of 128 segments for the patients with PCR. The internal diameter of the vessel was measured with an ocular micrometer. If stenoses were observed, their degree and length were calculated in percentage as compared to the normal internal diameter of the vessel. The degree of narrowing was divided into four classes: 0-30%, 31-60%, 61-90%, and over 90%.

Clinico-pathologic correlation and statistical method

All the data obtained from clinical and pathologic examination were compared. The age of the patients with and without rupture as well as heart weight, thickness of the left ventricular wall and percentage size of the infarction were tested statistically. Standard statistical methods (t-test) were used to compare the aforesaid parameters. P less than 0.01 was taken to denote statistical significance. All data were presented as means ± SD.

RESULTS

Clinical data (Table I).

All patients included in the study were in the age range from 46 years to 91 years (mean: 66.4±12.35 years for the people who showed PCR and 64.6 ± 12.35 years for the people without rupture). There was no statistically significant difference among studied people (p=NS). The sixteen patients with PCR disclosed anterior infarction in 11 observations (68.75%) and inferior infarction in 5 cases (31.25%). Death occurred from as early as twelve hours to as late as seven days after infarction. Six patients (37.5%) died suddenly (Table II) without premonitory symptoms or with symptoms of less than an hour's duration in a time interval 12 hours to seven days from the onset of AMI. The recorded electrocardiograms showed sinus rhythm in two cases, atrioventricular block in two cases and junctional rhythm in the remaining two cases. By contrast, ten subjects (62.5%) (Table III) had a reappearance of chest pain that was only slightly or not at all improved by opiates. Their electrocardiograms showed sinus rhythm with unchanged ST-segment before death, that occurred in a time interval from 240 minutes to 660 minutes (mean: 368 minutes) after the onset of chest pain. We defined this death as late death. It occurred from 3 to 7 days after the onset of myocardial

Fig 2 - Method of examination of the heart. The heart is cut transversely in slices parallel to each other of thickness approximately 1 cm.

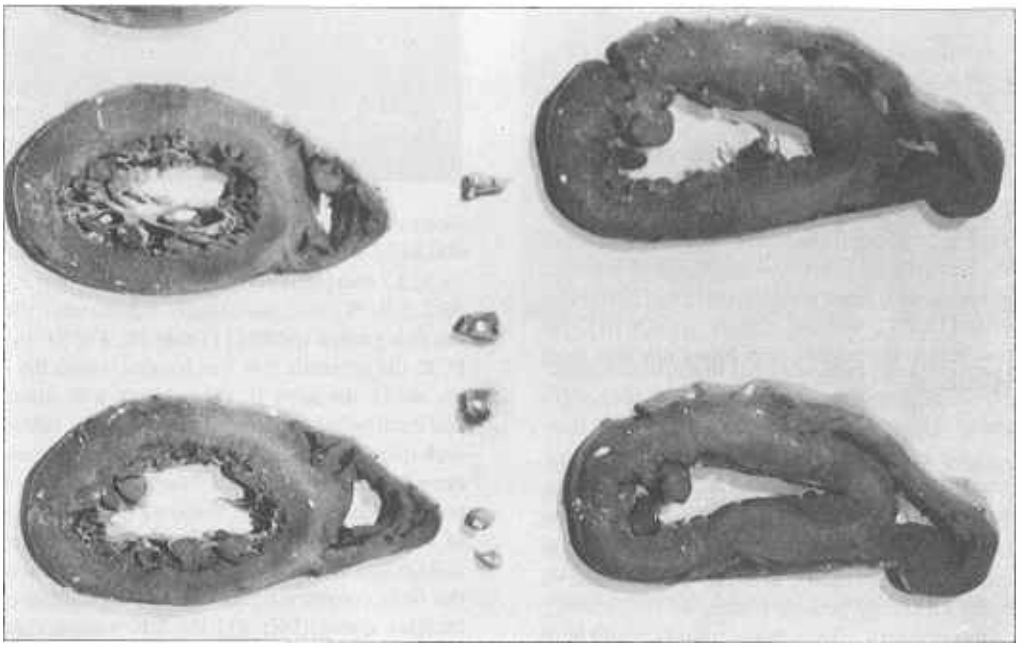


Table I - Clinical Features of the Patients

	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		
Echocardiography			
infarct expansion	2		
pericardial effusion	2		
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 the patients with PCR

Type of death	No. of Patients	Days from AMI onset						
		1	2	3	4	5	6	7
Sudden death	6 (37.5%)	2	1	2				1
without symptoms	4							
with chest pain of < 1 hour's duration	2							
Late death	10 (62.5%)			6	2	1		1
with premonitory symptoms	10							
- severe chest pain	10							
- infarct expansion	2							
- pericardial effusion	2							
Total	16 (100%)	2	1	8	2	1		2

infarction. Only four subjects with rupture underwent echocardiography. An area of thinning throughout the cardiac cycle and akinesis at the site of infarction were seen in the two patients (Fig 3). The remaining two cases showed pericardial effusion (Fig 4). Six patients (37.5%) with PCR had previous hypertension (systolic blood pressure 180 mmHg to 220 mmHg) versus 9 patients (11.2%) without rupture ($p < 0.01$). No physical exertion before the rupture was found nor was there any evidence of diabetes.

Pathologic data

Of the sixteen hearts with rupture, 11 showed transmural anterior infarctions and 5 transmural inferior infarctions. The heart weights were between 390 and 1020 gm (mean: 627.5±201.05 gm), the wall thickness of the left ventricle between 18mm and 30mm (mean: 25.7±3.61mm), and the percent size of infarction between 25 and 65 percent (mean: 44.17±12.03%). The same parameters in the people without

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

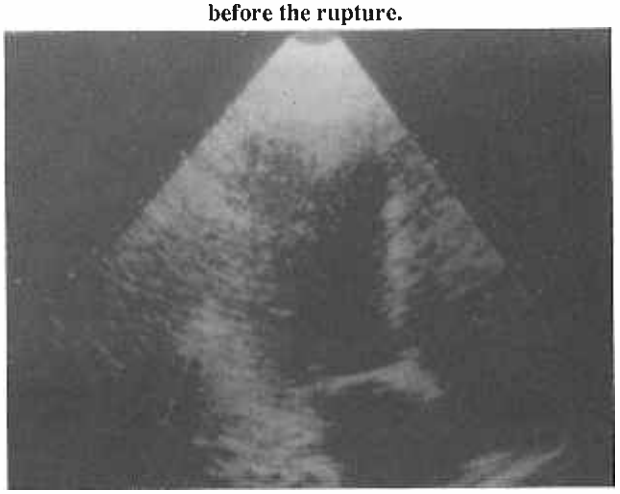
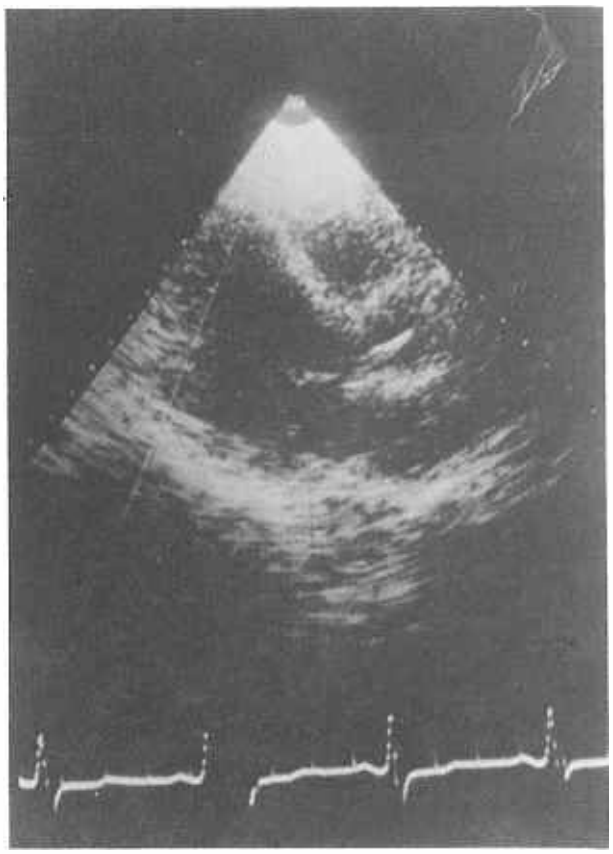


Fig 4 - Two dimensional echocardiography. Pericardial effusion before the rupture



rupture were as follows: heart weight 290 to 650 gm (mean: 400.8±73.4 gm), left ventricular thickness 11 to 23 mm (mean: 14.5±2.7 mm), and percent size of infarction 20 to 65% (mean: 30.2 ± 9.7%) with statistically significant difference between the two groups ($p<0.01$) (Table IV; Fig 5). In the group with PCR, the eccentric tear was located within the infarcted area 2 cm above the apex in the subjects with anterior infarctions, and 2 cm below posterior atrioventricular sulcus in the patients with inferior infarctions. No right ventricular and/or septal ruptures were seen nor were there any incomplete rupture or pseudoaneurysm. Table V shows the type of coronary lesions of the patients with PCR. The left anterior descending artery (LAD) was affected totally by stenoses in seventeen instances, the right coronary artery (RC) in nine instances, the left circumflex artery (LC) and the left main coronary artery(LM)

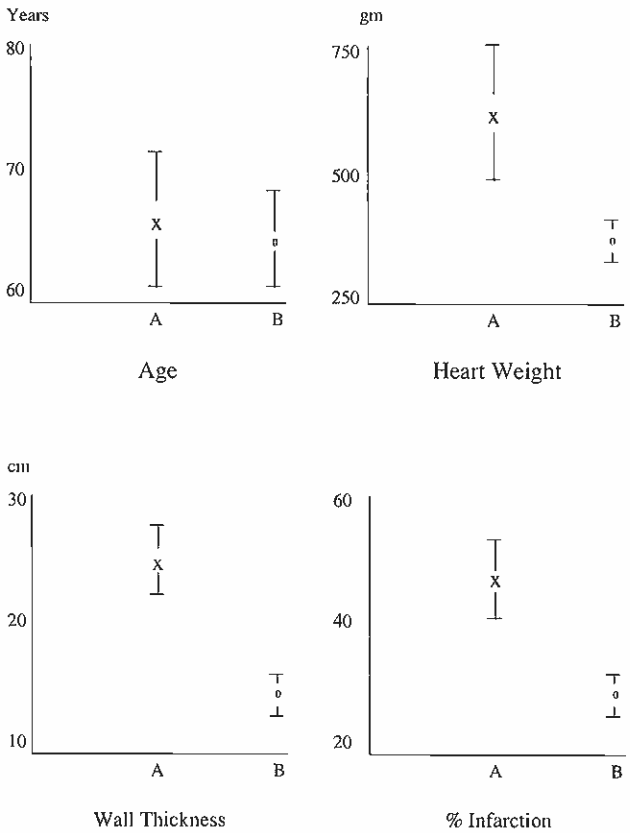
Table III - Appearance of chest pain before the rupture and recorded electrocardiogram

No.	Onset of Chest Pain (minutes)	Recorded Electro-cardiogram
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	"
mean:	368	

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

Fig 5 - Graphic showing statistically significant difference of some parameters in patients with and without PCR



Mean and Confidence interval of the cardiac parameters tested statistically

A = Group with rupture

B = Group without rupture

Table V - Coronary lesions in the patients with PCR

Coronary Artery	Occlusive Thrombi		% Luminal Narrowing			
	PL	Hyal	0-30	31-60	61-90	over 90
LAD	0	6	2		4*	2*
LAD + RC	0	3			2(1*)	2*
LAD+RC+LM	0	2			4(2*)	
LAD+RC+LC	0	1			1*	
RC	0	1			1*	

LAD = Left anterior descending artery

LM = Left main coronary artery

RC = Right coronary artery

LC = Left circumflex artery

PL = Platelet

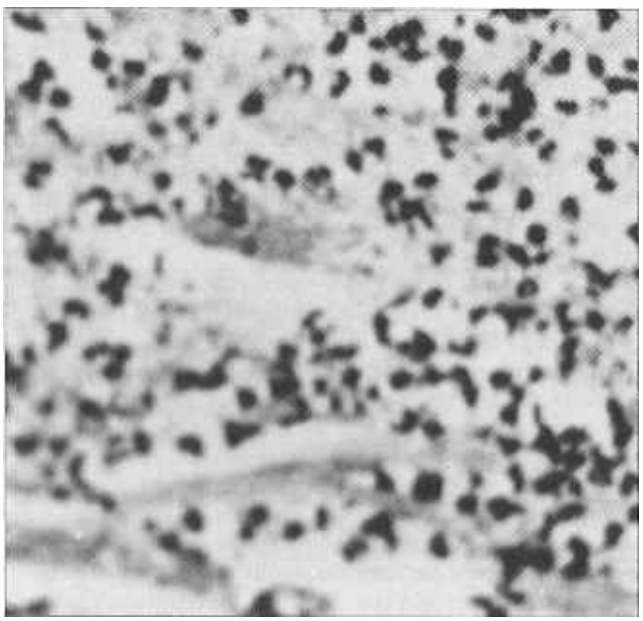
Hyal = Hyalin

* = Pre-existing thrombosed stenoses.

twice. In the great majority of cases the stenoses were over 60 percent. Occlusive thrombi were found in thirteen instances. They were located in the epicardial segment of coronary arteries and affected LAD ten times and RC three times. All observed thrombi were hyalin thrombi, found at the site of old stenoses greater than 60%.

Microscopic examination of the hearts displayed myocardial necrosis, massive infiltrates of cells around the rupture (Fig 6) and cardiac hypertrophy (Fig 7).

Fig 6 - Myocardial necrosis and massive infiltrates of cells around the rupture



Characteristics of the increased parameters (Table VI)

Of the analysed parameters, anatomically the heart weight and wall thickness of the left ventricle were showing the greatest increase (over 55% of their maximum normal value) in the patients with PCR ($p < 0.01$). Clinically, hypertension, which pre-existed to the infarction, was seen to have statistical difference ($p < 0.01$) between the group with rupture and the group without rupture. However, hypertensive attacks during admission were not recorded in the previously hypertensive patients who died from PCR.

Fig 7 - Cardiac hypertrophy. One can see evident amount in size of myocardial fibres

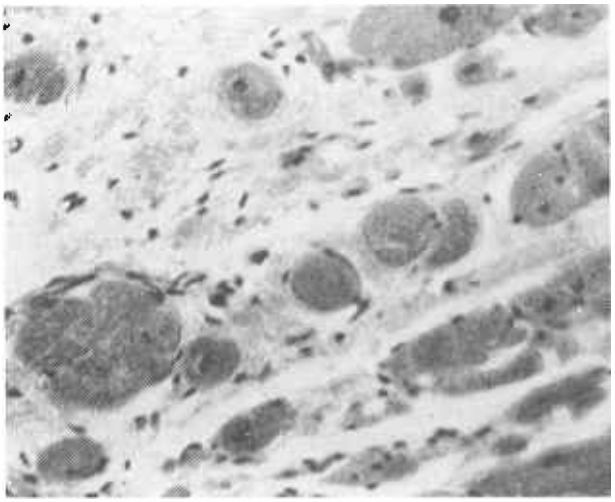


Table VI - Parameters significantly increased in the hearts with postinfarction cardiac rupture

Clinical Parameters	
- Hypertension (compared to the patients without rupture)	$P < 0.01$
Pathologic Parameters	
– Heart Weight	over 55% of normal value
– Wall Thickness	over 55% of normal value

DISCUSSION

Although the age in the patients with and without rupture of this study was similar, there are reports [19,20] that the patients of highest risk of free wall rupture appear to be women older than 60 years. Yet, there are different reports about infarct size of the hearts with PCR.The infarcts may be small to moderate[20] as well as involving more than 20 percent of the left ventricle[19]. Our study shows large infarcts in the patients with PCR.

The mortality of the patients with PCR is still extremely high. One-third of subjects die suddenly, whereas two-thirds will undergo death several hours after the appearance of some premonitory symptoms[7,8,16]. These people, who could be potentially salvageable by surgical intervention, often do not undergo this approach. Thus, we must face the problem of PCR by preventive measures against such an occurrence.

Markers of PCR have been described [6 8, 16] in a great number of patients who develop heart rupture. Some markers are markers of impending rupture, but there are factors that seem to predispose the heart chronically to the rupture in case of appearance of infarction. Systemic hypertension, cardiac hypertrophy and severe coronary atherosclerosis belong to this group[16].

With regard to cardiac hypertrophy, it may occur in a great variety of cardiovascular conditions that place burdens on the myocardium. In patients with hypertension, acute myocardial infarction, coronary atherosclerosis, especially if combined with hypertension, the heart may increase its weight and hypertrophies.

In systemic hypertension, common disease up to the present, the weight of the heart and wall thickness of the left ventricle show the greatest increase and myocardial fibres usually tend to hypertrophy chronically. The heart also enhances left ventricular contractile performance. All these factors cause both increased work capacity and oxygen demand. As the degree of hypertrophy increases, the number of blood capillaries per unit of myocardial mass becomes insufficient and that predisposes myocardial cells to a chronic hypoxia. Further blood impairment caused by myocardial infarction may be a serious factor leading to the rupture. Our observations found hypertension with a frequency statistically significant ($p < 0.01$) in the patients with PCR if they were compared with infarcted people without rupture. However, several data seem to show that the percentage of deaths due to coronary events has risen since the introduction of antihypertensive drug treatment, leaving it as the major cause of death in treated hypertension[21]. In these patients PCR is rarely observed. That would seem to show even more the role that pre-existing stable hypertension plays in causing rupture during AMI. Systemic hypertension hypertrophies the heart and that leads to coronary atherosclerosis. Jointly, these factors are evident in almost all cases with PCR.

Therefore, a careful removal of those factors that may cause cardiac hypertrophy chronically might reduce the occurrence of PCR.

CONCLUSION

Blood pressure, heart weight and wall thickness of the left ventricle were the most increased parameters in this study.

They correlate well a chronic myocardial damage either metabolic damage or loading damage pre-existing to the infarction.

Preventive measures against such factors can reduce PCR.

ACKNOWLEDGEMENT

This work has been presented orally at the 8th Asean Congress of Cardiology-1990, 7-11 December 1990, Singapore.

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