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Incidence and Significance of Pericardial Effusion in Acute Myocardial Infarction as Determined by Two-Dimensional Echocardiography

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To determine the incidence and clinical significance of pericardial effusion after acute myocardial infarction, two-dimensional echocardiography was serially performed in 66 consecutive patients. Pericardial effusion was observed in 17 (26%); the effusion was small in 13 patients, moderate in 3 and large with signs of cardiac tamponade in 1. In this patient, two-dimensional echocardiography strongly suggested myocardial rupture. The observation of pericardial effusion was not associated with age, sex, previous myocardial infarction, atrial fibrillation or treatment with heparin. It was more often a complication of anterior than of inferior acute infarction. Patients with pericardial effusion had higher peak levels of creatine kinase and lactic dehydrogenase and a higher wall motion score index. More patients with pericardial effusion had congestive heart failure or ventricular arrhythmias, developed a ventricular aneurysm or died within 1 year after their infarction.

In conclusion, pericardial effusion is frequently visualized by two-dimensional echocardiography after acute myocardial infarction and its presence is associated with an increased occurrence of complications and cardiac death.

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The significance of pericardial effusion after acute myocardial infarction is unknown. M-mode echocardiography was used in two recent studies (1,2) to determine the incidence of pericardial effusion after acute myocardial infarction. The frequencies found in the two studies were quite different: 5.6% (1) and 37% (2). M-mode echocardiography has been shown to be accurate for the detection of pericardial effusion (3); however, the narrow field of this technique leads to pitfalls that can be obviated by two-dimensional echocardiography. Therefore, the aim of this study was to determine the incidence and significance of pericardial effusion detected by two-dimensional echocardiography after acute myocardial infarction.

Methods

Study patients. Seventy consecutive patients admitted for definite acute myocardial infarction were studied by two-dimensional echocardiography. Acute myocardial infarction was diagnosed by a rise in creatine kinase to at least twice the normal level including a creatine kinase MB rise of greater than 5% in the clinical setting of prolonged angina and serial electrocardiographic changes. Four patients (6%) were excluded because their echocardiograms were technically inadequate. None of the 66 remaining patients had had acute myocardial infarction or cardiac surgery within 3 months of admission or had a potential cause of pericardial effusion.

Clinical evaluation. Serum creatine kinase levels and lactic dehydrogenase levels were analyzed every fourth hour during the first 3 days and daily thereafter. The auscultation of pulmonary rales at admission was codified according to Killip's classification (4). Congestive heart failure was defined by the observation during the acute phase of infarction of a third heart sound or Killip class greater than 1, or both. Rhythm abnormalities were observed through electrocardiographic monitoring. Ventricular fibrillation, ventricular tachycardia and ventricular premature beats of Lown classes 3 to 5 (5) were considered to be significant ventricular arrhythmias.

Echocardiography. Two-dimensional echocardiography was performed with a Diasonics V3400R phased-array sector scanner. All classic views were recorded on videotape for subsequent analysis by observers who were

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unaware of other patient data. Echocardiograms were performed on the first, third and seventh days. In 10 patients, the initial echocardiogram could not be obtained within 24 hours of admission; these patients were not excluded from analysis, because no pericardial effusion was resolved before hospital discharge in the study of Kaplan et al. (2). No patient died before an echocardiogram was obtained. The presence of pericardial effusion was independently assessed by two observers using the method described by Martin et al. (6). The amount was estimated as small, moderate or large. Signs of cardiac tamponade (7) were searched for when pericardial effusion was present.

Regional ventricular performance was analyzed. A ventricular segmentation of 16 left and 5 right ventricular segments was used (8). For each segment, abnormal wall motion was graded as: 0= hyperkinetic, 1 = normal, 2 = hypokinetic, 3 = akinetic, 4 = dyskinetic, and 5 = aneurysmal. Aneurysmal motion was diagnosed as thinned myocardium that was dyskinetic in systole and demonstrated persistent bulging in diastole. From the echocardiogram obtained on the third day, a wall motion score index was calculated by dividing the sum of the segmental scores by the number of segments visualized.

Statistical analysis. The mean values and standard deviation were computed for all quantitative variables, and proportions (%) were computed for binary or discrete clinical findings. Means and proportions for patients with and without pericardial effusion were compared using the classic Student's *t* test or the chi-square test, respectively. Results were considered significant at the 5% critical level.

Results

Incidence of pericardial effusion. Pericardial effusion was detected by two-dimensional echocardiography in 17 (26%) of the 66 patients. It was detected within the first 24 hours in 8 of the 17 patients, and was found on the third day in 4 patients, 2 of whom had not had an echocardiogram on day 1. In the other five, pericardial effusion was identified on the seventh day. The size of pericardial effusion was small in 13 patients, moderate in 3 and large in 1. This last patient presented clinical end echocardiographic signs of cardiac tamponade on the first day after a recurrent posterior infarct; the two-dimensional echocardiogram revealed rupture of the basal posterolateral free wall and the patient was operated on, but died in cardiogenic shock. Permission to perform an autopsy was refused.

Pericardial effusion had cleared on hospital discharge in only one patient. No patient in this series developed clinical features consistent with a diagnosis of postmyocardial infarction syndrome (9).

Clinical characteristics (Table 1). There were no differences between the two groups (pericardial effusion versus no effusion) in age, sex, incidence of previous acute myocardial infarction, occurrence of atrial fibrillation or treatment with intravenous heparin. Thrombolytic therapy was not used in this series of patients. Pericardial effusion was observed more frequently with an anterior than with an inferior infarct. There was no significant difference in the proportion of non-Q wave infarction in our two study groups.

Patients with pericardial effusion had significantly higher peak creatine kinase and higher peak lactic dehydrogenase levels than did those without pericardial effusion. More patients with pericardial effusion showed signs of congestive heart failure and significant ventricular arrhythmias. The Killip class at admission was higher in patients with pericardial effusion. The wall motion score index calculated from the echocardiogram obtained on the third day was higher in patients with pericardial effusion (2.15 ± 0.59) than in those without effusion (1.67 ± 0.42) ($p < 0.001$). In the subgroup of patients with a first acute myocardial infarction, left ventricular aneurysm developed more frequently in patients with pericardial effusion.

Total mortality at 1 year was higher in patients with than in patients without pericardial effusion. The three patients with pericardial effusion who died during the acute phase of infarction were in cardiogenic shock; one of them developed myocardial rupture. Five of the six patients with pericardial effusion who died during the first year after hospital discharge were dead within the first month; the sixth patient died later of noncardiac causes. Three of these five patients died suddenly, one developed a recurrent infarct and one had subsequent intractable heart failure.

Discussion

Incidence of pericardial effusion. The results of this study show a 26% incidence of pericardial effusion after acute myocardial infarction as determined by two-dimensional echocardiography. Two previous prospective echocardiographic surveys made with the M-mode technique showed a marked discrepancy in results (1,2). M-mode echocardiography may fail to detect pericardial effusion because it visualizes a limited area of the heart; its specificity is not perfect, because subepicardial adipose tissue may produce the M-mode echocardiographic appearance of pericardial effusion (10). The incidence found in our study is more in agreement with that reported by Kaplan et al. (2) (36%) than with the low incidence (6%) reported by Wunderink (1).

Significance of pericardial effusion. Pericarditis after acute myocardial infarction was previously diagnosed by the occurrence of pericardial chest pain or pericardial rub, or both (11–15). Because there is no correlation between a pericardial rub and the presence and amount of pericardial effusion (2,16), the prognostic significance of pericarditis

Table 1. Patient Characteristics

	No Pericardial Effusion	Pericardial Effusion	p Value
No. of patients	49 (74%)	17 (26%)	
Age (yr)	58 ± 9	59 ± 10	NS
Sex			
Male	40	11	
Female	9	6	NS
First AMI	39	12	NS
Site			
Anterior	19	10	<0.05
Inferior	28	6	<0.05
Lateral	2	1	NS
Non-Q wave infarction	8	1	NS
Peak CK	1,257 ± 867	1,853 ± 1,122	<0.05
Peak LDH	1,596 ± 834	2,165 ± 1,023	<0.05
Killip class on admission	1.45 ± 0.73	2 ± 1.03	<0.02
Congestive heart failure	16	11	<0.02
Ventricular arrhythmias	13	9	<0.02
Atrial fibrillation	9	2	NS
Echographic score index	1.67 ± 0.42	2.15 ± 0.59	<0.001
Aneurysm (first AMI)	5/39	7/12	<0.001
Heparin therapy	31	10	NS
Mortality			
In hospital			
(at 15 days)	5	3	NS
From 15 days to 1 year	3	6	<0.01
Total at 1 year	8	9	<0.01

AMI = acute myocardial infarction; CK = creatine kinase; LDH = lactic dehydrogenase; NS = not significant.

or pericardial effusion after acute myocardial infarction may be different.

Our results show that pericardial effusion is associated with a larger infarct, frequently complicated by congestive heart failure and cardiac death. In this series, 8 (53%) of 15 patients who died within 1 year of infarction had pericardial effusion. This confirms the clinicopathologic correlations reported by Roeske et al. (17), who found 40% of 85 necropsy patients to have pericarditis.

All of our patients with pericardial effusion presented segments without myocardial thickening as detected by two-dimensional echocardiography. However, this does not necessarily imply the transmural nature of their infarct (18). Only one patient with pericardial effusion had a non-Q wave infarct, but there was no significant difference between the two groups in this respect. Our findings do not permit us to distinguish between the irritant versus the hemodynamic theory as the major factor contributing to effusion in acute myocardial infarction (19).

Ventricular arrhythmia. We observed a higher incidence of significant ventricular arrhythmias among patients with pericardial effusion as compared with the series of Spodick (20), who studied patients with acute pericarditis by 24 hour electrocardiographic monitoring. In our series, arrhythmias were noted whenever they occurred during hospitalization; they are probably related to the amount of myocardial necrosis and do not imply an arrhythmogenic influence of pericardial effusion.

Ventricular aneurysm. The association found in our study between pericardial effusion and left ventricular aneurysm may be of clinical importance. It is unknown whether the development of early regional cardiac dilation may be a cause of pericardial effusion. The thinned area of myocardium in the aneurysm is at increased risk of rupture (21). Pericardial effusion was present in 4 of the 20 cases of myocardial rupture in the series of Norris and Sammel (22). Cardiac rupture occurred in one patient in our study group; the large pericardial effusion was detected on the first echocardiogram soon after admission. There is no evidence that effusion preceded the rupture in this case.

Clinical implications. Our results indicate that pericardial effusion is frequently visualized by two-dimensional echocardiography after acute myocardial infarction and suggest that its presence is associated with an increased occurrence of complications and cardiac death. Therefore patients presenting with pericardial effusion after acute

myocardial infarction should be closely followed up. Further studies are needed to determine whether the time of hospital discharge must be delayed in these patients and whether serial two-dimensional echocardiograms may lead to improved management.

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