The spectrum of pericardial effusion in acute myocardial infarction: an echocardiographic study

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Key words:
Pericardial effusion; Textural patterns; Cardiac tamponade.

Background. **The aim of this study was to assess the prevalence of pericardial effusion in acute myocardial infarction and the different prognosis associated with distinct patterns of pericardial effusion (anechoic/hypoechoic vs hyperechoic effusion).**

Methods. **Five hundred eighty-five consecutive patients admitted to the Coronary Care Unit for acute myocardial infarction were initially considered. Forty of them were excluded due to a technically poor acoustic window. The remaining 545 patients were studied by two-dimensional echocardiography at admission, before discharge (after an average of 9 days in the Coronary Care Unit) and whenever there was an important change in the clinical status (chest pain, lipothymia or syncope, hemodynamic deterioration with systolic blood pressure < 90 mmHg, cardiac arrest).**

Results. **Pericardial effusion was found in 51 patients (9%). Three distinct textural patterns of pericardial effusion were noted on the basis of the echogenic properties: 1) anechoic or hypoechoic pericardial effusion was frequent (30 patients), mild or moderate and generally benign; 2) hyperechoic type A effusion pattern was rare (2 patients) and associated with fever, leukocytosis and pericardial rubs; 3) hyperechoic type B was frequent (19 patients), large and always associated with major complications (all cases cardiac tamponade and/or death).**

Conclusions. **Pericardial effusion is not an uncommon finding in serial echo evaluation of patients with acute myocardial infarction, especially when infarction is anterior, extensive and Q wave. Echocardiographically detected pericardial effusion shows different textural patterns with hypoanechoic effusion more frequent, limited and prognostically benign than hyperechoic effusion larger and often associated with adverse events.**

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Introduction

Two-dimensional echocardiography plays a pivotal role in the initial diagnostic evaluation and follow-up monitoring of the patients admitted to the Coronary Care Unit for an acute myocardial infarction. The analysis of myocardial wall motion is certainly the main information in the echocardiographic assessment of acute myocardial infarction. However two-dimensional echocardiography can give a lot more important information and is particularly capable of evaluating a possible pericardial involvement that is rather frequent[1-3], not necessarily clinically meaningless and can on occasion become the main protagonist of an unfavorable outcome.

Moreover there are several studies reporting the possibility of distinguishing the nature of pericardial effusion on the basis of echo texture[4-11].

The aim of this study was: 1) to assess the prevalence of pericardial effusion in acute myocardial infarction in the present thrombolytic era; 2) to establish the relative incidence of different echo textural patterns of pericardial effusion; 3) to evaluate whether different echo patterns are associated with a different short-term prognosis.

Methods

Study patients. The study population consisted of 585 consecutive patients admitted to our Coronary Care Unit from January 1992 to March 1996 for an acute myocardial infarction.

The diagnosis of acute myocardial infarction was defined by standard clinical, electrocardiographic and laboratory criteria[12]. In the Coronary Care Unit all patients were closely observed with continuous ECG monitoring (Hewlett-Packard 78560A System). Blood pressure was assessed at least every 3 hours. Systemic thrombolysis was performed according to the GISSI-2 study[13].

Of the 585 initial patients, 40 were excluded due to a technically poor acoustic window. The remaining 545 patients (167 females, 378 males, mean age 67 – 13 years) were enrolled in the study and underwent two-dimensional echo evaluation at admission, before discharge (occurring 9 – 5 days after admission), and whenever there was an important change in the clinical status (chest pain, lipothymia or syncope, hemodynamic deterioration with systolic blood pressure < 90 mmHg, cardiac arrest).

Echocardiographic study. Serial echocardiograms were obtained employing commercially available ultrasound systems with 3.5 MHz probes (Sonos 1000, Hewlett-Packard). Images were obtained in the standard parasternal long- and short-axis, apical and subcostal 4-chamber views in the two-dimensional and M-mode techniques and all images were recorded on videotape for subsequent analysis. Echo recordings were interpreted by two experienced observers who did not know the patient s clinical features. Discrepancies were resolved by a consensus.

The following variables were investigated:

• the diagnosis of pericardial effusion was made only in the presence of an echo-free space without end-diastolic obliteration (Horowitz s D pattern). According to Galve et al.[14] we assumed that this finding assures an amount of fluid > 50 ml, when the separation between the pericardial layers was present at M-mode on the whole cardiac cycle;

• the amount of pericardial effusion was arbitrarily considered as mild (< 5 mm separation), moderate (10 to 20 mm), severe (> 20 mm);

• the presence of intrapericardial echoes of high acoustic density;

• the presence of cardiac tamponade (diastolic invagination of right ventricular and/or right atrial during diastole both with two-dimensional and M-mode examination).

On the basis of qualitative textural appearance of pericardial fluid, effusions were considered:

- anechoic: echo-free pericardial effusion;
- hypoechoic: mildly echoic pericardial effusion;
- hyperechoic: with strongly reflective pericardial effusion. Two different kinds of hyperechoic effusion were considered: type A with strands connecting parietal and visceral pericardial layers, and type B with high acoustic density comparable to liver or clot density.

Statistical analysis. Continuous data are described as mean – 1 standard deviation. Differences between continuous data were compared with the unpaired Student s t-test. Discontinuous variables were compared by χ^2 analysis. Differences between groups were considered statistically significant when p values were < 0.05.

Results

Prevalence of pericardial effusion. Pericardial effusion was found in 51 patients (9%) showing a unimodal distribution of frequency peaking on the third day. A consensus was needed in 6 cases (moderate/mild pericardial effusion). There were no differences, between the two groups, in so far as sex, creatine kinase peak, and thrombolysis were concerned, but patients with effusion were younger, and more frequently had anterior Q wave infarction associated with heart failure, and suffered from a higher in-hospital mortality (Table I).

Table I. Main demographic and clinical findings of patients.

	PE	Non PE	p
No. patients	51 (9%)	494 (91%)	
Age	66 – 12.9	70.5 – 8.9	< 0.005
Male	30 (59%)	341 (69%)	NS
Female	21 (41%)	153 (31%)	NS
Site of infarction			
Anterior	29 (57%)	201 (41%)	< 0.05
Inferior	20 (39%)	248 (59%)	NS
Lateral	1 (2%)	13 (2.5%)	NS
Undefinable	1 (2%)	32 (6.5%)	NS
Q wave infarction	48 (94%)	322 (65%)	< 0.0001
Non-Q wave infarction	3 (6%)	172 (35%)	< 0.0001
CK peak	1233	1052	NS
Thrombolysis	24 (47%)	198 (40%)	NS
Heart failure	19 (37%)	62 (15%)	< 0.0001
Angina	8 (16%)	49 (10%)	NS
Pericardial rub	4 (8%)	16 (3%)	NS
In-hospital mortality	21 (41%)	42 (8.5%)	< 0.0001

CK = creatine kinase; PE = pericardial effusion.

Quantitative and qualitative aspects of pericardial effusion. Pericardial effusion was mild in 20 patients, moderate in 11, and large in 20. The textural appearance of the effusion was anechoic in 25 (Fig. 1), hypoechoic in 5 (Fig. 2) and hyperechoic in 21 patients: 2 A type (Fig. 3) and 19 B type (Figs. 4 and 5).

Mild to moderate effusions were more frequently associated with anechoic/hypoechoic and type A pericardial effusion (30/31 patients), whereas large effusions were much more frequently found in type B pericardial effusion (19/20 patients) (Fig. 6).

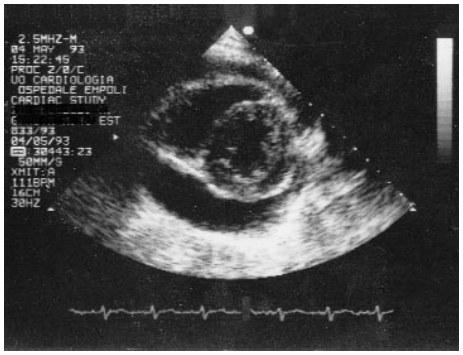

Figure 1. *Typical echo appearance of an anechoic pericardial effusion.*

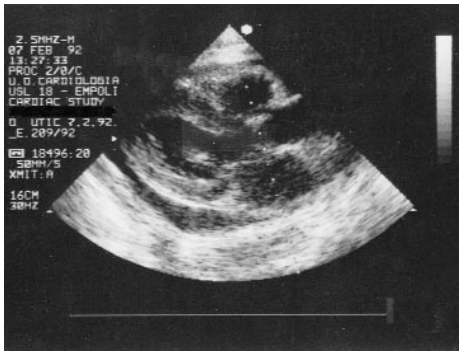

Figure 2. *Typical echo appearance of a hypoechoic pericardial effusion.*

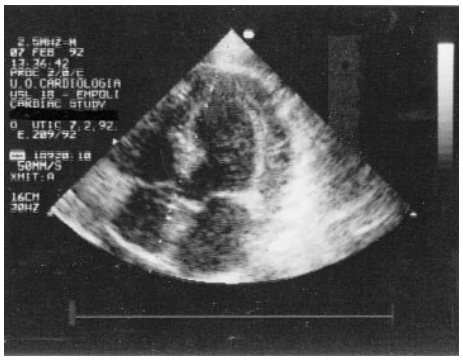

Figure 3. *Typical hyperechoic appearance of pericardial effusion with fibrinous band.*

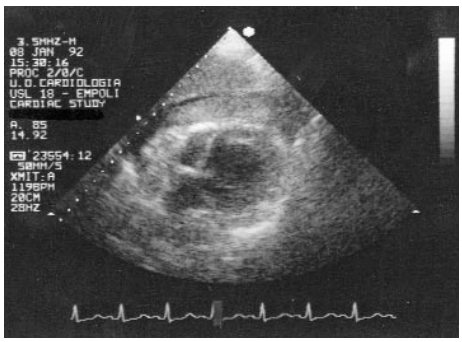

Figure 4. *Typical hyperechoic appearance of pericardial effusion with acoustic density comparable to liver: type B hyperechoic pericardial effusion.*

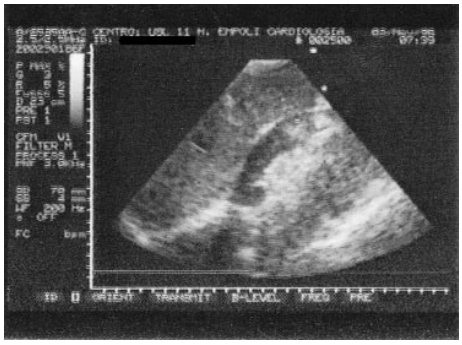

Figure 5. *Typical echo appearance of pericardial effusion with acoustic density comparable to clot: type B hyperechoic pericardial effusion.*

In-hospital course. No clinical differences were found into the subgroups of patients (Table I).

The clinical picture underlying the echocardiographic evidence of pericardial effusion could be categorized into three main conditions:

- asymptomatic or with minor changes in the clinical status not attributable to pericardial effusion (n = 29, 57%);
- inflammatory syndrome with systemic (fever, leukocytosis, erythrosedimentation rate) and local signs (chest pain) (n = 2; 4%);
- sudden, life-threatening manifestations (hemodynamic instability, cardiac arrest, shock) (n = 20, 39%).

The asymptomatic picture was much more frequent with anechoic/hypoechoic effusion and inflammatory syndrome with type A pericardial effusion, whereas cataclysmic onset was more frequent with type B pericardial effusion.

Sixty-three in-hospital deaths (11.5%) occurred. The in-hospital mortality was significantly higher in patients with pericardial effusion (41 vs 8.5%). In patients with pericardial effusion the causes of death were left ventricular free wall rupture in 17 and shock in 4. In patients without pericardial effusion the causes of death were cardiogenic shock in 31, major arrhythmias in 8, and 3 due to other causes.

Cardiac tamponade and/or in-hospital death occurred in all patients with type B hyperechoic effusion, and

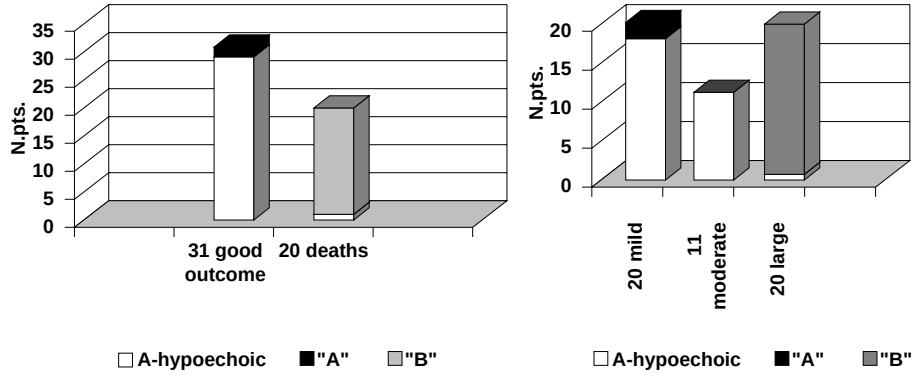

Figure 6. *Bar histograms representing the prevalence of the different categories of pericardial effusion, separated according to the entity* (right panel) *and prognosis* (left panel).

only in 1 of those with anechoic/hypoechoic pericardial effusion (Fig. 6). According to these data, in-hospital mortality was greater in the subgroup with type B pericardial effusion.

In all patients with large pericardial effusion and electromechanical dissociation, an ultrasonically-guided pericardiocentesis was performed: in 6/20 patients we observed a decreasing of echo-free space with temporary improvement of the clinical status.

Systemic thrombolysis. Forty percent of patients (222/545) received systemic thrombolysis at hospital admission. The prevalence of pericardial effusion in these patients was 10% (24/222) vs 9% (27/323) of patients not treated (p = NS). No significant differences were found as regards quantitative and qualitative aspects of pericardial effusion. Late thrombolysis (> 6 hours) was performed in 38/222 patients (17%): 4 of them had type B pericardial effusion (11 vs 8% of patients with early thrombolysis, p = NS).

Discussion

Pericardial effusion is not an uncommon finding in patients with acute myocardial infarction also during the thrombolytic era. Nevertheless, in our study the frequency of pericardial effusion was lower than reported by others[2,14], probably due to the more restrictive criteria adopted for the diagnosis of pericardial effusion. The amount of the effusion ranges over a broad spectrum, from small to large. The textural appearance of pericardial effusion is even more important than its amount as far as the associated clinical syndrome and the prognostic outcome are concerned, with the worst evolution associated with hyperechoic effusions. Anechoic pericardial effusion is more frequent in patients with heart failure, whereas pericardial effusion is usually absent during early post-infarction pericarditis.

The biological basis of different textural echo patterns. The variety of textural appearances of pericardial effusion identifies a likely morphological substrate in the different histological nature of the pericardial effusion.

A serous exudate is known to be echo-free, and in fact serous pericarditis is a benign cause of effusion or a frequent pattern in patients with heart failure. Patients with this kind of pericardial effusion show, according to this hypothesis, a fairly good outcome and a progressive resolution of pericardial effusion over a period of time. On some occasions, the amorphous intrapericardial echo-free space was associated with discrete, echogenic structure bridging, in an irregular way, parietal and visceral pericardium. This pattern might identify a plausible histological substrate in a serofibrinous exudate[14,15]. Interestingly, this echocardiographic pattern was more frequently associated with an inflammatory systemic syndrome, chest pain and progressive slow resolution consistent with the hypothesized serofibrinous nature of the effusion[9].

The most likely substrate of type B hyperechoic effusion is hemopericardium, subsequent to a subacute or acute free wall rupture. Experimental studies[5] have demonstrated that blood and clots in the pericardial cavity produce high acoustic echoes easily identifiable by echocardiography. Clinical experiences have confirmed that this acoustic pattern is frequently associated with hemopericardium[5,7,8,10]. In association with the clinical status, this echocardiographic finding is highly suggestive of cardiac rupture with obvious therapeutic and prognostic implications.

Role of systemic thrombolysis. As regards thrombolytic therapy, no significant difference was found between two groups of patients with or without treatment, either prevalence and entity or the textural appearance of pericardial effusion. The reason could be found in the relatively small number of patients of this study. However, regarding the timing of thrombolysis (late thrombolysis) and its possible effect on the risk of cardiac rup-

ture[16], our study seems to confirm this possible role but our data have not statistical power.

Comparison with previous studies. The prevalence of echocardiographically detected pericardial effusion has been reported in various studies to be in the range of 5 to 64% of cases. In comparison with previous studies, our has several peculiarities:

- the study population is the largest studied to date, with 585 consecutive patients initially evaluated and 545 finally enrolled;
- particular attention was paid not only to the presence and entity, but also to the textural appearance of pericardial effusion. In fact, in recent years there has been increasing attention to tissue characterization studies, with the possibility even of simple visual assessment of echo texture to offer some clues to the underlying histology of the tissue.

In conclusion, our data confirm the fairly high prevalence of pericardial effusion, demonstrate the textural heterogeneity of the echocardiographically detected pericardial effusion, and outline the clinical and prognostic implications associated with the different textural appearances.

References

1. Wunderink RG. Incidence of pericardial effusion in acute myocardial infarction. Chest 1984; 85: 494-6.
2. PiØrard LA, Albert A, Henrard L, et al. Incidence and significance of pericardial effusion in acute myocardial infarction as determined by two-dimensional echocardiography. J Am Coll Cardiol 1986; 8: 517-20.
3. Kaplan K, Davison R, Parker M, et al. Frequency of pericardial effusion as determined by M-mode echocardiography in acute myocardial infarction. Am J Cardiol 1985; 55: 335-40.
4. Pollak H, Diez W, Spiel R, Enenkel W, Mlczoch J. Early diagnosis of subacute free wall rupture complicating acute myocardial infarction. Eur Heart J 1993; 14: 640-8.
5. Mikell FL, Asinger RW, Elsperger KJ, et al. Regional stasis of blood in dysfunctional left ventricle: echocardiographic detection and differentiation from early thrombosis. Circulation 1982; 66: 755-63.
6. Chandraratna PAN. Echocardiography and Doppler ultrasound in the evaluation of pericardial disease. Circulation 1991; 84 (Suppl 1): 303-10.
7. Mazzoni V, Bartoletti A, Monopoli A, Taiti A, Bini A. Studio ecocardiografico dell interessamento pericardico in corso di infarto miocardico acuto. Giornale Italiano di Ecocardiografia 1994; 2: 43-7.
8. Brack M, Asinger RW, Sharkey SW, Herzog CA, Hodges M. Two-dimensional echocardiographic characteristics of pericardial hematoma secondary to left ventricular free wall rupture complicating acute myocardial infarction. Am J Cardiol 1991; 68: 961-4.
9. Martin RP, Bowden R, Filly K, Popp RL. Intrapericardial abnormalities in patients with pericardial effusion. Circulation 1980; 61: 586-72.
10. Knopf WD, Talley JD, Murphy DA. An echo-dense mass in the pericardial space as a sign of left ventricular free wall rupture during acute myocardial infarction. Am J Cardiol 1987; 59: 1202.
11. Garcia-Fernandez MA, Moreno M, Rossi PN, Lopez-Sendon JL, Banuelos F. Echocardiographic features of hemopericardium. Am Heart J 1984; 107: 1035-6.
12. WHO Task Force on Standardization of Clinical Nomenclature and Criteria for diagnosis of ischemic heart disease. Circulation 1979; 59: 607-8.
13. GISSI-2: A factorial randomised trial of alteplase versus streptokinase and heparin versus no heparin among 12 490 patients with acute myocardial infarction. Gruppo Italiano per lo Studio della Sopravvivenza nell Infarto Miocardico. Lancet 1990; 336: 65-71.
14. Galve E, Garcia-Del Castillo H, Evangelista A, et al. Pericardial effusion in the course of myocardial infarction: incidence, natural history and clinical relevance. Circulation 1985; 73: 294-9.
15. Sugiura T, Iwasaka T, Takayama Y, et al. Factors associated with pericardial effusion in acute Q wave myocardial infarction. Circulation 1990; 81: 477-81.
16. Honan MB, Harrel FE, Reimer KA, et al. Cardiac rupture, mortality and the timing of thrombolytic therapy: a meta-analysis. J Am Coll Cardiol 1990; 16: 359-67.