Pericardial Effusion in Acute Myocardial Infarction: Frequency and In-Hospital Course

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Objective: To determine the frequency and in-hospital course of pericardial effusion in acute myocardial infarction in our population. **Design:** Prospective observational study. **Place of Study**: The study was conducted in the Department of Cardiology and Medicine, Mayo Hospital, Lahore. **Patients and methods:** One hundred consecutive patients presented within first 24 hours of first episode of ST elevation myocardial infarction (STEMI) were studied. Patients with known coronary artery disease (CAD), chronic renal failure, collagen vascular disease, cardiac surgery and metastatic disease were excluded from the study. 2-D and M-mode echocardiographic examination was carried out daily and pericardial effusion (PE) was considered to be present when separation between two pericardial layers persisted throughout the cardiac cycle. **Results:** Among 100 patients who were enrolled, 27 developed PE. Frequency of baseline variable like age, gender, and risk factors for coronary artery disease including hypertension, smoking, and diabetes mellitus were same in patients who developed PE when compared to those who did not. Most of the PE was detected on day 5 of the admission. About 82 % patients had mild PE (only posteriorly and <10 mm). Moderate PE was detected in 18% (present all around and between 10-20 mm). None of the patients developed large PE (>20 mm). About 15 % patients who were thrombolysed and 40% who could not be thrombolysed developed PE ($p < 0.01$). Frequency of PE was statistically highly significant ($p < 0.001$) among patients with higher Killip class and lower ejection fraction at the time of presentation. Patients who developed PE had statistically significant longer hospital stay ($p < 0.001$) and higher in-hospital mortality ($p < 0.05$). **Conclusion:** Thrombolysis decreased the frequency of PE in acute STEMI. Development of PE during the course of acute STEMI has prognostic implications and early invasive strategy may be offered for patients who develop this complication.
Key words: Pericardial effusion; acute myocardial infarction; streptokinase

Acute myocardial infarction (AMI) due to coronary artery disease (CAD) is the commonest cause of death in western world. Pericardial effusion (PE) is one of the complications occurring during the course of AMI, but there is marked variability in its reported incidence, mechanisms, and clinical significance[1-5]. Echocardiography is the procedure of choice for the detection of PE, and although PE demonstrated by echocardiography may indicate fluid retention, this is not diagnostic of pericardial injury. A pericardial rub is not rare after AMI and has been reported in patients with pericardial involvement near the infarct[6,7]. Therefore, it is clinically important to distinguish between irritant and hemodynamic factors contributing to the occurrence of PE in the early phase of AMI

An increase in the production of myocardial interstitial fluid or interference of fluid drainage is reported to cause PE.[3] Patients with PE during AMI had larger infarcts and significantly higher hospital mortality rates[4,5]. PE is a reliable bedside marker of myocardial infarct size and poor outcome. Frequency and day of onset of PE vary widely as stated in many previous studies ranging from 5.6 to 43%. This study was done to determine the frequency and in-hospital course of PE in acute ST elevation myocardial infarction (STEMI).

Patients and methods

This prospective observational study was conducted in Department of Cardiology and Medicine, Mayo Hospital, Lahore. One hundred consecutive patients presenting within 24 hours of first episode of acute STEMI were included after informed consent. Those with known history of coronary artery disease, pericardial disease, chronic renal failure, collagen vascular disorder, previous cardiac surgery and metastatic disease were excluded from the study. Diagnosis of acute STEMI was made by 12 lead ECG showing J point elevation of more than 1 mm in 2 contiguous leads. Acute myocardial infarction was confirmed by two-fold rise in creatine kinase (CK). Two dimensional (2-D) and M-mode echocardiography was done to detect PE. First examination was performed within 24 hours of admission and then daily till the day of discharge. PE was considered to be present when separation between two pericardial layers persisted throughout the cardiac cycle. A single operator performed the echocardiogram and left ventricular ejection fraction (LVEF %) of every patient was measured by eyeballing technique. PE was considered as mild if effusion was less than 10mm, or if present only posteriorly; moderate if between 10-20mm and large if it was more than 20mm and present all around. A preset proforma was filled on the day of admission and subsequently daily till the day of discharge from the hospital. Data banking in the second phase was computerized and all the results were generated using the software SPSS version 10.

Results

A total number of 100 patients were included, 81 were male and 19 were female. Age of the patients ranged from 32 to 69 years (mean $\pm$ SD 53.42 $\pm$ 7.94). PE was detected

in 27 (27%) of the patients, 18 among them were male and 9 were female. Frequency of baseline variable like age, gender, and risk factors for coronary artery disease including hypertension, smoking, and diabetes mellitus were same in patients who developed PE when compared to those who did not. Most of the PE was detected on day 5 of the admission. In 22 out of 27 (81%) patients, PE was mild. Moderate PE was detected in 5 (19%). None of our patients developed large PE. Age and gender was not a significant factor for development of PE. Streptokinase was used as a thrombolytic agent. The effect of thrombolysis on subsequent development of PE was assessed. Eight of 53 (15%) patients who were thrombolysed and 19 of 47 (40%) who could not be thrombolysed developed PE ($p < 0.01$). Twenty-four out 27 who later developed PE were in cardiac failure at the time of presentation. Only three among those who were not in cardiac failure at the time of presentation developed PE ($p < 0.001$). Mean $\pm$ SD LVEF of patients who developed PE was 45 $\pm$ 7 % (range 35-55%) as compared to 53 $\pm$ 7 % (range 40-70%) in those who did not developed PE ($p < 0.001$). Forty-four patients suffered anterior MI, 38 inferior MI and 4 presented with chest pain and left bundle branch block (LBBB). Location of MI was not a significant factor in development of PE. The duration (Mean $\pm$ SD) of hospital stay among patients who developed PE was 9 $\pm$ 3 days (range 5-16) and 7 $\pm$ 1 days (range 3-15) that did not ($p < 0.001$). Five patients died during hospital stay and four of these had significant PE ($p < 0.05$).

Discussion

Pericardial effusion is a relatively common early finding after acute myocardial infarction, but its incidence is variable. In this study PE was found in 27% of the patients. Most of it was detected on day 5 of the admission. Galve et al [5] found PE in 28% and Takarda et al [6] in 33 % patients with AMI. Sigiura et al [7] found 24% incidence of PE. Wunderik [8] studied 100 patients with first Q-wave AMI and found PE in only 5.6% [9] The apparent difference of incidence can be explained on the fact that in most of the studies including ours, PE was found on day 5, while Wunderik performed one echocardiogram within 24 hours and another on the day of discharge, so he might had missed significant number of effusions. Correale et al[9] found 6.7% incidence and attributed low incidence to administration of thrombolytic therapy. Widimisky et al [10] found 43% incidence of PE in AMI. This high incidence in his observation can be explained on the basis that he used different echocardiographic criteria (minimal separation of 3 mm of 2 layers).

Decrease in the frequency of PE in those who received thrombolytic treatment may be explained on the basis that thrombolytics restore coronary blood flow leading to smaller infarct size and better LVEF. Correale et al [9] analyzed data from GISSI-1 trial and confirmed that who received thrombolytics within 6 hours had statistically significant reduction in the development of PE (49% risk reduction, $2p < 0.0001$) and had significant favorable results on mortality. (30%, $2p < 0.001$).

Takarada et al[6] stated that PE was found more frequently in those whose infarct related artery was non-canalized than in those patients in whom it was canalized. Improvement of regional wall motion abnormality in patients with PE was less regardless of the successful early recanalization. They concluded that PE was important for predicting prognosis. In contrast to other studies, Widmisky et al[10] found slight increase in the number of PE in patients who were thrombolysed. This may be due to overall very high incidence of PE in their study. Berger at al [11] and Kessler at al[12] stated that heart failure was the single most important etiological factor in 29% and 22% patients respectively. Similarly Wunderik[8] found that PE was more common in patients of AMI who were in heart failure. He failed to show any positive correlation between the two. This may also be due to low incidence of PE in his study. Both Correale et al[9] and Galve et al [5] in their separate studies found a direct correlation of heart failure and PE. Myocardial lymph drains to the subepicardium and ultimately to the mediastinum and right heart cavities. Thus, PE appears to result from an increase in the production of myocardial interstitial fluid or from interference of myocardial venous and lymph drainage by elevated central venous pressure[2, 7].

Patients with AMI who subsequently developed PE had statistically significant longer in-hospital stay. The subject has not been touched by any previous studies. Sigiura et al [7] analyzed 8 variables and demonstrated significant increase in in-hospital mortality associated with occurrence of PE in AMI. By reviewing the data from GISSI-1 and GISSI-2 trial Correlae et al [9] found out that although pericardial involvement was a good indicator of size of infarct, he failed to show any significant short-term increase in mortality associated with it. He however stated that it was significantly associated with long-term mortality. It was difficult to explain his observation.

The present study demonstrates that thrombolysis decreases the frequency of pericardial effusion in acute STEMI. Development of pericardial effusion during the course of acute STEMI has prognostic implications and early invasive strategy may be offered for patients who develop this complication.

References

1. Krainin FM, Flessas AP, Spodick DH: Infarction-associated pericarditis: Rarity of diagnostic electrocardiogram. N Engl J Med 1984; 311:1211-1214
2. Spodick DH: The normal and diseased pericardium: Current concepts of pericardial physiology, diagnosis and treatment. J Am Coll Cardiol 1983; 1:240-251.
3. Kaplan K, Davison R, Parker M, Przybylek J, Light A, Bresnahan D, Ribner H, Talano JV: Frequency of pericardial

effusion as determined by M-mode echocardiography in acute myocardial infarction. Am J Cardiol 1985; 55:335-337
4. Pierard LA, Albert A, Henrard L, Lempereur P, Sprynger M, Carlier J, Kulbertus HE: Incidence and significance of pericardial effusion in acute myocardial infarction as determined by two-dimensional echocardiography. J Am Coll Cardiol 1986; 8:517-520
5. Galve E, Garcia-Del-Castillo H, Evangelista A, Batlle J, Permanyer-Miralda G, Soler-Soler J: Pericardial effusion in the course of myocardial infarction: Incidence, natural history, and clinical relevance. Circulation 1986; 73:294-299
6. Takarada A, Kurogane H: Pericardial effusion in acute myocardial infarction: Clinical features and significance. Am J Cardiol 1991; 21 (3): 517-25.
7. Sugiura T, Iwasaka T, Takayama Y et al. Factors associated with pericardial effusion in acute Q-wave myocardial infarction. Circulation 1990; 81:477-481.
8. Wunderik R G: Incidence of pericardial effusion in acute myocardial infarction. Chest 1984; 85:494-496
9. Correale E, Maggioni Ap: (GISSI-2) Comparison of frequency, diagnostic and prognostic significance of pericardial involvement in AMI treated with and without Thrombolysis. Am J Cardiol 1993; Jun 15, 71 (16): 1377-1381.
10. Widimisky P, Gregor P: Pericardial involvement during the course of myocardial infarction: A long-term clinical and echocardiographic study. Chest 1995; 108:89-93.
11. Berger M, bobak L, Jelveh M, Goldberg E: Pericardial effusion diagnosis by echocardiography. Chest 1978; 74:174-9.
12. Kessler KM, Rodriquez D, Rahim A, Dheen M, Samet P: Echocardiographic observation regarding pericardial effusion with cardiac disease. Chest 1980; 78:736-40.