Hospital Outcome of Moderate to Severe Pericardial Effusion Complicating ST-Elevation Acute Myocardial Infarction

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**Background**—Hospital prognosis of moderate to severe pericardial effusion (MPE; ≥10 mm) in ST-elevation myocardial infarction is largely unknown.

**Methods and Results**—Data from 446 ST-elevation myocardial infarction patients, 228 with MPE—88 with cardiac tamponade (CT) and electromechanical dissociation (EMD), 44 with CT without EMD (w/oEMD), and 96 without initial CT—and 218 with small PE (5 to 9 mm), were compared. Patients with MPE without initial CT were also compared with 96 patients without PE. CT patients showed larger PE (P<0.001) than those without initial CT; 85% of those with CT+EMD and 86% with CTw/oEMD were treated with pericardiocentesis and 10% and 21% were treated with a surgical repair, respectively. Among MPE patients, 30-day mortality was 43% and was higher in those with CT+EMD (operated, 89%; and nonoperated, 85%) than in those with CTw/oEMD (22% and 11%, respectively; P<0.001) and those without initial CT (17%; P<0.001). It was also higher than in patients with small PE (10%; P<0.001) or those without PE (6%; P=0.001). Death was attributable to cardiac rupture in 83% of patients with CT+EMD, 7% with CTw/oEMD, and 8% with MPE without initial CT and occurred late (≥7 days) in 14%, 67%, and 100%, respectively.

**Conclusions**—MPE carries an increased mortality that is highest in patients with CT+EMD. In those with CTw/oEMD, however, mortality is considerably low after pericardiocentesis, and subsequent management may be individualized because a conservative approach is often successful. Importantly, MPE patients without initial CT are not free from late rupture and deserve further investigation. 

**Key Words:** myocardial infarction ■ cardiac rupture ■ cardiac tamponade ■ death, sudden ■ pericardial effusion

Moderate to severe pericardial effusion (MPE; ≥10 mm) in patients with ST-elevation myocardial infarction (STEMI) may lead to cardiac tamponade (CT), which presents as electromechanical dissociation (EMD) or arterial hypotension.1-5 Although it is often associated with left ventricular free-wall rupture (FWR),3,5 it can also be produced by heart failure,1-2 myocardial hemorrage caused by anticoagulant or antiaggregant therapies,6-8 or pericarditis.9 Moreover, in view of the high rate of underlying FWR, CT often requires emergency surgery,2,5,6 particularly when associated with EMD.10,11 Pericardiocentesis and blood pressure control may also be life-saving.12,13 Because of the lack of large clinical series in which different therapeutic alternatives have been contemplated, it is unclear whether open heart surgery should be performed routinely in all STEMI patients with CT and hypotension. Also unknown is the risk of CT in those with MPE without initial CT.

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Thus, we prospectively evaluated the prognosis of STEMI patients with MPE, seeking to identify those at high risk of mortality. To that effect, we compared 4 groups with a potentially different outcome, namely patients with CT+EMD, CT without EMD (CTw/oEMD), MPE without initial CT, or small PE (5 to 9 mm). We also assessed whether management of MPE in a progressive fashion from medical treatment to surgical intervention may be an appropriate alternative to systematic surgery.

**Methods**

**Patients**

From January 1990 to December 2008, 4446 consecutive STEMI patients were admitted to our institution, a tertiary referral where >80% of patients are admitted directly. Diagnosis of STEMI was based on concurrence of ST elevation and chest pain or symptoms of acute or subacute heart failure. For patients admitted within 48 hours, elevated creatine kinase-MB was an additional criterion.

A small (5 to 9 mm) PE was demonstrated echocardiographically in 257 patients shortly after admission or during hospitalization; 39 evolved to MPE and 218 did not, and a predischarge echocardiogram performed in 92 patients showed no increase in PE size. An MPE was already detected in the first echocardiogram in 218 other patient, but 33 were excluded: 13 because MPE was secondary to right ventricular perforation by a pacing catheter and 20 because of an associated septal (n=19) or papillary (n=1) muscle rupture. Sixteen
additional patients without echocardiogram were suspected to have died of FWR because they died of EMD, and the 4 with a FWR confirmed at autopsy were included. Thus, 228 patients with MPE were included (Figure 1): 132 with CT (88 with and 44 without EMD) and 96 without CT. Eleven of these 228 were referred from other centers because of MPE (4.8%): 7 with CT and 4 without. MPE was detected within 48 hours in 55 patients with CT + EMD (62.5%), 26 with CTw/oEMD (59.1%), and 72 without CT (75%). In addition, in 24 CT patients (18.2%) and in 15 with MPE without initial CT (15.6%), MPE was preceded by small PE. In 56 of the 224 patients with echocardiographic evidence of MPE, a thoracotomy (n = 24) or an autopsy study (n = 32) was performed, and an unsealed or sealed FWR was confirmed. Thirty-eight of these 56 patients had presented CT+EMD (11 at surgery [9 for FWR repair, 1 for pericardial toilette, and 1 for coronary bypass grafting] and 27 at autopsy), 14 CTw/oEMD (12 at surgery [8 for FWR repair, 3 for pericardial toilette, and 1 for coronary bypass grafting] and 2 at autopsy), and 4 MPE without initial CT (1 at surgery and 3 at necropsy).

Hospital mortality was available for the 3955 patients without PE who did not die suddenly of EMD. Individual data, however, could not be retrieved except for the 96 matched individually for age and calendar year with the 96 with MPE without initial CT (control group). Age was the only variable independently associated with MPE in a previous study with 1149 STEMI patients.5

**Protocol and Management**

A 12-lead ECG was performed on admission, daily thereafter, and during episodes of chest pain or hemodynamic deterioration. Creatine kinase-MB levels were measured every 4 to 6 hours for 48 hours. From January 1990 to December 1991, a 2-dimensional echocardiogram was performed in patients who became hypotensive; from January 1992 on, it was performed in all patients admitted to our cardiac care unit within the first 12 hours. It was often repeated before discharge, at least in those patients who developed arterial hypotension or had a small or MPE. PE was measured at end diastole at the parasternal transversal plane and was graded in millimeters as the summation of anterior and posterior effusions. CT was diagnosed in patients who developed EMD or hypotension without preceding symptoms of overt heart failure in whom MPE was associated with right atrial compression, jugular venous distention, arterial hypotension (≥90 mm Hg), and pulsus paradoxus >15 mm Hg.

Aspirin, intravenous nitroglycerin, and analgesia were routinely administered. Propranolol was given orally if systolic blood pressure ≥110 mm Hg and heart rate was >50 bpm in the absence of heart failure, atrioventricular conduction abnormalities, or chronic lung disease. In patients with heart failure, angiotensin-converting enzyme inhibitor was given as tolerated, and heparin was administered during at least 48 hours to patients treated with fibrinolytics or without reperfusion therapy. Treatment of CT+EMD included cardiac massage, intravenous volume, inotropic agents, mechanical ventilation if needed, and pericardiocentesis. In patients who recovered hemodynamically, a thoracotomy with repair of FWR was considered, considering the risk of associated comorbidity (eg, advanced age, chronic pulmonary disease, extensive myocardial damage, or chronic renal failure). Patients with CTw/oEMD were initially managed also with intravenous volume, inotropic agents, and small-volume pericardiocentesis whenever feasible. In the absence of hemodynamic recovery, surgical repair of the possible FWR was also entertained. However, if rapid hemodynamic recovery was attained, patients were managed conservatively as those with EMD in whom surgery was excluded, including maintenance of inotropic support. This was followed by institution or reinstitution of β-blockers to keep systolic blood pressure at 100 to 120 mm Hg. Heparin was withdrawn and patients were kept on bed rest for at least 5 days.

Hospital mortality and 30-day mortality were analyzed, and 1-year follow-up was available in patients with CT. In these patients, ≥1 echocardiograms were carried out to assess the recurrence of PE or the development of left ventricular pseudoaneurysm.

**Statistical Analysis**

Comparisons across the 4 groups with PE of decreasing clinical severity (CT + EMD, CT w/oEMD, MPE without initial CT, and small PE) were performed by the Jonckheere-Terpstra test for continuous factors and the χ² test for trend. Comparisons among patients with uncomplicated MPE and their individually matched control subjects were performed by means of paired t tests for continuous variables or by the Mantel-Haenszel odds ratio with the corresponding χ² test for discrete variables. In addition, the association between uncomplicated MPE and hospital mortality was assessed by conditional logistic regression analysis in the age-matched pairs with adjustment for other clinical predictors (sex, diabetes mellitus, Killip class, extent of ST elevation, and reperfusion therapy). Analysis was performed with SPSS 13.0 (SPSS Inc, Chicago, Ill). Values of P < 0.05 were considered significant.

**Results**

**Demographics, ECG, and Angiographic Data**

Patients with CT + EMD, CTw/oEMD, MPE without initial CT, and small PE showed similar gender distribution and risk factors, although patients with CT + EMD were older. Moreover, patients with small PE showed a higher incidence of previous angina and a shorter admission delay (from pain onset to hospital admission) than the other groups and a lower incidence of lateral infarction, together with patients with MPE without initial CT, than those with CT (Table 1).

Admission ST elevation was similar in the 4 groups with PE, whereas patients with MPE showed a higher ST elevation at 24 and 48 hours than those with small PE (Table 1). Incidence of new-onset atrial fibrillation (before surgery if performed), pericarditic pain, and friction rub were significantly higher in patients with CTw/oEMD than in the other 3 groups (Figure 2). Patients with MPE without initial CT showed a lower incidence of previous infarction and previous angina and a longer admission delay than control patients. ST elevation at 24 and 48 hours was higher and ejection fraction was lower in the former group (Table 2).

Patients with MPE without initial CT had smaller PE (15.5 ± 6.5 mm) than those with CT + EMD (24.0 ± 4.4 mm) or CTw/oEMD (24.2 ± 6.2 mm; P < 0.001) and a lower incidence of right atrial (27.7% versus 98.7% and 93.4%; P < 0.001) or right ventricular (13.8% versus 96.1% and 78.6%; P < 0.001, respectively) compression. In most patients...
with CT who had ≥2 echocardiograms (116 of 128, 90.6%), PE was first localized in the anterior pericardial space but became more evenly distributed over time.

**Management and Outcome**

β-Blockers, nitrates, angiotensin-converting enzyme inhibitors, and statins were used less frequently in patients with CT than in those with MPE without initial CT or with small PE, in part because of their overall more impaired hemodynamic condition and the shorter survival time in those with CT (Table 3). There was also a significant trend toward an increased rate of reperfusion therapy in the last 2 groups (Table 3). A pericardiocentesis performed in patients with CT+EMD or CTw/oEMD showed a bloody fluid in all, and the hematocrit measured in 40 instances was comparable in the 2 groups (Figure 3). A diagnostic pericardiocentesis was performed in 8 patients with MPE without initial CT, and a bloody pericardial fluid was also observed although the hematocrit was lower (Figure 3). It is our contention that most patients with CT presented an acute or subacute FWR as the underlying cause, whereas its frequency among those with MPE without initial CT was more uncertain.

A surgical repair of FWR was performed in 19 patients: 9 with CT+EMD, 9 with CTw/oEMD, and 1 with PE without CT.

### Table 1. Characteristics of the 4 Groups of STEMI Patients With PE

<table>
<thead>
<tr>
<th></th>
<th>CT+EMD (n=88)</th>
<th>CTw/oEMD (n=44)</th>
<th>MPE no CT (n=96)</th>
<th>Small PE (n=218)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>72.8±7.9</td>
<td>68.7±7.9</td>
<td>69.4±9.5</td>
<td>69.4±9.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female, %</td>
<td>36.4</td>
<td>34.1</td>
<td>31.3</td>
<td>28.9</td>
<td>0.130</td>
</tr>
<tr>
<td>Active smoker, %</td>
<td>25.0</td>
<td>45.5</td>
<td>28.1</td>
<td>33.5</td>
<td>0.404</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>50.0</td>
<td>63.6</td>
<td>58.3</td>
<td>54.1</td>
<td>0.813</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>35.2</td>
<td>36.4</td>
<td>35.4</td>
<td>28.9</td>
<td>0.216</td>
</tr>
<tr>
<td>Cholesterol &gt;230 mg/dL, %</td>
<td>41.2</td>
<td>52.4</td>
<td>48.4</td>
<td>50.9</td>
<td>0.189</td>
</tr>
<tr>
<td>COPD, %</td>
<td>36.6</td>
<td>38.6</td>
<td>28.0</td>
<td>22.9</td>
<td>0.006</td>
</tr>
<tr>
<td>CVA, %</td>
<td>9.2</td>
<td>6.8</td>
<td>8.3</td>
<td>6.4</td>
<td>0.440</td>
</tr>
<tr>
<td>PVD, %</td>
<td>17.2</td>
<td>13.6</td>
<td>25.0</td>
<td>13.8</td>
<td>0.517</td>
</tr>
<tr>
<td>Old MI, %</td>
<td>5.7</td>
<td>9.1</td>
<td>4.2</td>
<td>7.3</td>
<td>0.728</td>
</tr>
<tr>
<td>Previous angina, %</td>
<td>18.2</td>
<td>13.6</td>
<td>17.7</td>
<td>34.4</td>
<td>0.001</td>
</tr>
</tbody>
</table>

**Infarct site, %**

- Inferior: 42.0 vs. 31.8 vs. 38.9 vs. 43.6
- Anterior: 33.0 vs. 45.5 vs. 53.7 vs. 53.7
- Lateral: 22.7 vs. 18.2 vs. 5.3 vs. 2.8
- Other: 2.3 vs. 4.5 vs. 2.1 vs. 0

**Admission delay, h**

- 18.8±33.9 vs. 17.9±31.3 vs. 21.2±31.9 vs. 10.8±18.9

**Killip class I–II, %**

- 87.5 vs. 77.3 vs. 93.7 vs. 88.5

**CAVB, %**

- 3.4 vs. 2.3 vs. 6.3 vs. 9.2

**RBBB, %**

- 11.8 vs. 9.1 vs. 14.6 vs. 6.0

**VF/VT, %**

- 9.1 vs. 11.4 vs. 13.5 vs. 17.0

**Maximum STE, mm**

- 3.3±2.2 vs. 3.7±3.3 vs. 3.8±2.9 vs. 3.2±1.9

**Leads with STE, n**

- 5.1±2.1 vs. 5.3±2.4 vs. 5.2±1.9 vs. 5.1±1.6

**STE 24 h, mm (n)**

- 1.8±1.1 (59) vs. 2.1±1.4 (43) vs. 1.8±1.6 (90) vs. 1.3±1.1 (158)

**STE 48 h, mm (n)**

- 2.0±1.5 (48) vs. 2.3±1.6 (41) vs. 1.8±1.6 (90) vs. 1.4±1.2 (158)

**Peak CK-MB, µg/L**

- 282±279 vs. 274±265 vs. 244±232 vs. 297±244

**Ejection fraction, % (n)**

- 47.4±9.6 (60) vs. 42.8±10.6 (43) vs. 45.4±9.4 vs. 46.9±10.8 (214)

**Vessels ≥70%, n**

- 39 vs. 24 vs. 50 vs. 124

**0, %**

- 0.0 vs. 0.0 vs. 4.0 vs. 0.8

**1, %**

- 48.7 vs. 54.2 vs. 40.0 vs. 41.9

**2, %**

- 33.3 vs. 25.0 vs. 36.0 vs. 30.6

**3, %**

- 17.9 vs. 20.8 vs. 18.0 vs. 20.2

**Left main**

- 0.0 vs. 0.0 vs. 2.0 vs. 6.5

**3 Vessel/left main, %**

- 17.9 vs. 20.8 vs. 20.0 vs. 26.6

**100% occlusion, %**

- 75.0 vs. 66.7 vs. 52.2 vs. 58.2

COPD indicates chronic obstructive pulmonary disease; CVA, cerebrovascular accident; PVD, peripheral vascular disease; MI, myocardial infarction; CAVB, complete atrioventricular block; RBBB, right bundle-branch block; VF, ventricular fibrillation; VT, ventricular tachycardia; STE, ST elevation; and CK-MB, creatine kinase-MB.
Suture of the ruptured zone with or without infarctectomy was carried out in 9 patients, and a Dacron patch was glued to the fissured site in 10 patients (4 with CT + EMD, 5 with CTw/oEMD, and 1 with MPE without initial CT). Patients with MPE without initial CT were more frequently treated with nitrates and heparin than control subjects, in part because of the lesser use of primary percutaneous coronary intervention (Table 4).

Thirty-day mortality in MPE patients was 42.5% (97 of 228) and was higher in those with CT + EMD (operated, 8 of 9, 89%; nonoperated, 67 of 79, 84.8%) than in those with CTw/oEMD (2 of 9, 22.2%; and 4 of 35, 11.4%, respectively; \( P < 0.001 \)), those with MPE without initial CT (16, 16.7%; \( P < 0.001 \)), or those with small PE (21, 9.6%; \( P = 0.001 \); Figure 2). Importantly, 8 patients with MPE without initial CT evolved to CT, and all died of EMD. Mortality was lower in patients with CT + EMD (83%), and it occurred late (≥7 days) in 10 of 73 with CT + EMD (13.7%), 2 of 3 with CTw/oEMD (66.6%), and 8 of 8 with PE without initial CT (100%; \( P < 0.001 \)). The main reason why patients with CT + EMD were not operated on was lack of hemodynamic recovery after cardiac arrest.

Hospital mortality in patients with MPE without initial CT was higher than in the 96 matched control subjects (Table 4) and the overall group of STEMI patients without PE not dying of sudden EMD (378 of 3,955, 9.6%; \( P = 0.001 \)), and the last 2 subsets showed similar mortality (\( P = 0.376 \)). Multivariable conditional logistic regression analysis showed that initially uncomplicated MPE was independently associated with in-hospital mortality after adjustment for other clinical predictors (odds ratio, 4.35; 95% confidence interval, 1.12 to 16.99; \( P = 0.034 \)).

One-year mortality in operated and nonoperated patients with CT + EMD was 86% (68 of 79) and 88.9% (8 of 9), respectively, and 31.4% (11 of 35) and 22.2% (2 of 9) among those with CTw/oEMD. Three nonoperated patients showed a cardiac event: 2 cardiac deaths and 1 asymptomatic left ventricular false aneurysm.

**Discussion**

Our study indicated that STEMI patients with an MPE had a 5-fold increased mortality than those with small PE or without PE, and in 80% of instances, death was attributable to FWR. Importantly, however, mortality was lower in patients with CTw/oEMD than in those with CT + EMD and was comparable to that in patients with MPE without initial CT. Furthermore, the last subset presented an increased proportion of late deaths caused by FWR.

**Causes of MPE in STEMI**

It is generally accepted that bleeding from the infarcted wall is the most frequent cause of MPE in STEMI patients.\(^3\,\,5\,\,6\,\,14\)

Accordingly, pericardial fluid in our MPE patients was...
consistently bloody and its hematocrit was comparable in the 2 groups with CT, suggesting similarities in their anatomic substrate. Indeed, patients with CTw/oEMD showed features similar to those with CT/H11001EMD, a condition highly associated with FWR,10,11—including demographics, persistence of STE at 24 to 48 hours, and time from pain onset to CT. Unfortunately, no other large series have routinely investigated the content of PE. In the largest series, pericardiocenteses was hemorrhagic in 16 of 26 patients with a hematocrit/H1102225%,3 thereby hampering diagnosis of self-limited bleeding with subsequent hemodilution.5

Sources of MPE in CT patients other than FWR such as heart failure2,4 or fibrinous pericarditis with bleeding enhanced by anticoagulants or thrombolytic agents6–8 could not be ruled out. However, similarities between their clinical, ECG, and angiographic features and those in patients in whom an FWR was documented would suggest a similar mechanism of MPE. Heart failure, on the other hand, was unlikely because 89% of patients with CT were in Killip class I to II.

Table 3. Treatment Before Detection of MPE and In-Hospital Events in the 4 Groups of STEMI Patients With PE

<table>
<thead>
<tr>
<th>Treatment, %</th>
<th>CT+EMD (n=88)</th>
<th>CTw/oEMD (n=44)</th>
<th>MPE no CT (n=96)</th>
<th>Small PE (n=218)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-blockers</td>
<td>38.6</td>
<td>34.9</td>
<td>53.1</td>
<td>64.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Nitrates</td>
<td>81.6</td>
<td>77.3</td>
<td>99.0</td>
<td>95.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>16.3</td>
<td>27.9</td>
<td>45.2</td>
<td>46.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Statins</td>
<td>6.8</td>
<td>13.6</td>
<td>23.7</td>
<td>27.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Aspirin</td>
<td>93.0</td>
<td>95.5</td>
<td>96.8</td>
<td>99.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Heparin</td>
<td>92.0</td>
<td>95.5</td>
<td>96.8</td>
<td>80.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>75.0</td>
<td>61.4</td>
<td>18.1</td>
<td>19.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reperfusion, %</td>
<td>45.5</td>
<td>45.5</td>
<td>52.1</td>
<td>66.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>38.6</td>
<td>29.5</td>
<td>37.5</td>
<td>40.4</td>
<td></td>
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<tr>
<td>PCI</td>
<td>6.8</td>
<td>15.9</td>
<td>14.6</td>
<td>25.7</td>
<td></td>
</tr>
<tr>
<td>STEMI onset to MPE, d</td>
<td>3.4±2.7</td>
<td>4.4±5.6</td>
<td>2.7±3.2</td>
<td>...</td>
<td>0.009</td>
</tr>
<tr>
<td>STEMI onset to CT, d</td>
<td>3.5±2.8</td>
<td>5.0±4.6</td>
<td>7.3±6.6</td>
<td>...</td>
<td>0.028</td>
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<tr>
<td>Pericardiocentesis, %</td>
<td>85.4</td>
<td>86.4</td>
<td>15.3</td>
<td>...</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mechanical ventilation, %</td>
<td>70.5</td>
<td>20.5</td>
<td>10.5</td>
<td>10.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Surgery, %</td>
<td>10.2</td>
<td>20.5</td>
<td>1.1</td>
<td>...</td>
<td>0.062</td>
</tr>
<tr>
<td>Postinfarction angina, %</td>
<td>35.6</td>
<td>36.4</td>
<td>25.3</td>
<td>17.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Reinfarction, %</td>
<td>15.1</td>
<td>10.0</td>
<td>4.2</td>
<td>2.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>30-d Mortality, %</td>
<td>85.2</td>
<td>13.6</td>
<td>16.7</td>
<td>9.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hospital mortality, %</td>
<td>86.4</td>
<td>22.7</td>
<td>20.8</td>
<td>11.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>STEMI onset to death, d</td>
<td>4.4±4.7</td>
<td>31.7±25.4</td>
<td>14.0±10.5</td>
<td>14.3±21.0</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

ACE indicates angiotensin-converting enzyme; PCI, primary coronary intervention.

Table 4. In-Hospital Treatment and Cardiac Events in STEMI Patients With MPE Without Initial CT and Their Matched Control Subjects Without PE

<table>
<thead>
<tr>
<th>MPE no CT (n=96)</th>
<th>PE (n=96)</th>
<th>Unadjusted Odds Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-blockers</td>
<td>53.1</td>
<td>68.8</td>
<td>0.52 (0.28–0.94)</td>
</tr>
<tr>
<td>Nitrates</td>
<td>99.0</td>
<td>87.1</td>
<td>12.00 (1.56–92.29)</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>45.2</td>
<td>36.5</td>
<td>1.36 (0.79–2.36)</td>
</tr>
<tr>
<td>Statins</td>
<td>23.7</td>
<td>27.1</td>
<td>0.82 (0.41–1.67)</td>
</tr>
<tr>
<td>Aspirin</td>
<td>96.8</td>
<td>99.0</td>
<td>0.33 (0.03–3.19)</td>
</tr>
<tr>
<td>Heparin</td>
<td>96.8</td>
<td>78.1</td>
<td>7.00 (2.09–23.47)</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>18.1</td>
<td>16.7</td>
<td>0.93 (0.44–1.98)</td>
</tr>
<tr>
<td>Reperfusion, %</td>
<td>52.1</td>
<td>65.6</td>
<td>0.55 (0.30–1.02)</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>37.5</td>
<td>43.8</td>
<td></td>
</tr>
<tr>
<td>PCI</td>
<td>14.6</td>
<td>21.9</td>
<td></td>
</tr>
<tr>
<td>Mechanical ventilation, %</td>
<td>10.5</td>
<td>4.2</td>
<td>2.25 (0.69–7.31)</td>
</tr>
<tr>
<td>Postinfarction angina, %</td>
<td>25.3</td>
<td>17.7</td>
<td>1.64 (0.77–3.46)</td>
</tr>
<tr>
<td>Reinfarction, %</td>
<td>4.2</td>
<td>5.2</td>
<td>0.75 (0.17–3.35)</td>
</tr>
<tr>
<td>30-d Mortality, %</td>
<td>16.7</td>
<td>6.3</td>
<td>3.50 (1.15–10.63)</td>
</tr>
<tr>
<td>Hospital mortality, %</td>
<td>20.8</td>
<td>6.3</td>
<td>5.33 (1.55–18.30)</td>
</tr>
<tr>
<td>STEMI onset to death, d</td>
<td>14.0±10.5</td>
<td>1.3±0.6</td>
<td>0.237</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 3, plus CI indicates confidence interval.

Figure 3. Hematocrit (Hct) in systemic blood (syst) and pericardial fluid (peric) in the 3 groups with MPE. *P<0.001 for differences with respect to MPE and no CT (mean±SEM).
Pericarditis and Atrial Fibrillation

Patients with CTw/oEMD had a higher incidence of pericarditis and new-onset atrial fibrillation than the other 3 groups with PE. Although patients with CT+EMD died much earlier than those with CTw/oEMD, 4 versus 32 days, and thus had less chance to develop pericarditis, this finding is consistent with the high incidence of pericarditis (86%) reported by Oliva et al\cite{16} in 70 patients who died beyond the first 48 hours of an autopsy-documented FWR. We hypothesize that fibrous pericarditis was related to increased transmurality of STEMI but also, at least in some patients, to the episodic bleeding from the infarcted tissue through fissures of different severity. A smaller transmurality of STEMI was probably consequence to pericarditis because increases in atrial pressure\cite{17} were infrequent since most patients were in Killip class I to II. These 2 complications are usually late and hence are of limited help for clinical guidance. However, they may alert to the presence of MPE.

Management and Outcome

Patients with CT+EMD presented a high mortality, and only a few survived with surgical or medical management. Hence, attempts to significantly reduce mortality clearly require a highly motivated and coordinated medical and surgical team eager to initiate resuscitative maneuvers at any time and to rush the patient to the operating room because these are mostly cases of blowout FWR.\cite{18,19} Unfortunately, this is often not the case; mortality has been steadily high in patients with CT+EMD, even in the reperfusion era.\cite{3,15,20–25}

Patients with CTw/oEMD, generally considered cases of subacute FWR, showed a manifest lower mortality. Indeed, a substantial number of them had prompt hemodynamic recovery with pericardiocentesis, and in contrast to other reports,\cite{1,14,27–29} they were initially managed medically on the basis of a previous positive experience.\cite{13} Nevertheless, surgery was indicated in those patients who remained hypertensive or had recurrence of CT, although it was not always performed for a number of reasons (comorbidity, persistence of shock, etc). Despite this approach, 30-day mortality with conservative management was not higher than in operated patients, a finding that, in turn, was comparable to existing series.\cite{3,14,20}

Moreover, 1-year mortality was also similar in these 2 subsets but tended to be higher in nonoperated patients because of 3 new deaths, 2 cardiac. Therefore, these results would lend support to the feasibility of medical management in some selected patients,\cite{13} also reported by others.\cite{12,15,26,30}

Our results, however, are subject to the concept of “confounding by indications” because surgery versus medical therapy was selected in part on the basis of the patient’s clinical profile and prognosis. Hence, it is difficult to distinguish how much of the treatment difference in outcomes was due to treatment per se as opposed to the patient profile. Furthermore, although only a few (7%) nonoperated patients died of FWR, the mechanisms of the 2 cardiac deaths in the follow-up remained unclear.

Patients with MPE without initial CT showed a higher mortality than those with small PE or the control group. Importantly, 8% died suddenly of a likely FWR (CT+EMD); similar cases have been previously documented.\cite{1,15,31} This incidence of CT was higher than that reported for STEMI patients in general (0.9% to 2%).\cite{20,22–23} Unfortunately, however, no specific features allowed identification of those patients who finally developed CT other than it was a late event. Thus, we believe that more refinement is needed in the diagnostic tools aimed to clarify the source of PE and to identify those patients at high risk of EMD. Preliminary observations would suggest that magnetic resonance imaging may help recognize impending FWR.\cite{32}

Limitations

Although this is the largest series of STEMI patients with MPE, the total number of operated patients with CT was modest. However, and as indicated, our mortality was similar to that of large multicentric STEMI series,\cite{3,15,19–25} as was the
proportion of operated patients. Among patients with CT w/o EMD, the number of operated cases was also low, and surgical mortality was comparable to existing series. Nevertheless, it was not lower than in medically treated patients. It is therefore unclear whether mortality could have been different had we operated on all our patients. Indeed, a lack of randomized studies prevents us from extracting more solid conclusions as to whether the surgical or the medical approach is best suited after stabilization with pericardiocentesis. Thus, even though it is undisputable that surgical treatment of stabilized patients is generally successful, it is also true that in most instances of CT w/o EMD there is no active bleeding at surgery, as in the present series, which may explain the potential benefit of a conservative management.

**Implications**

We have shown that MPE was associated with an increased mortality ascribable to FWR in 80% of patients and was an unexpected prognostic marker of mortality in patients without initial CT. Accordingly, practice of an early conservative approach as a valid alternative to surgery in patients with clinicopathologic correlations. The presence of MPE should warrant echocardiographically because it may eventually evolve to mortality ascribable to FWR in 80% of patients and was an unexpected prognostic marker of mortality in patients without initial CT. Accordingly, practice of an early conservative approach as a valid alternative to surgery in patients with CT w/o EMD or without CT at risk of a late EMD who could benefit from a surgical intervention. Our results also underscore the value of a conservative approach as a valid alternative to surgery in some patients with CT w/o EMD who regain hemodynamic stability after pericardiocentesis.

**Sources of Funding**

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**Disclosures**

None.

**References**


**CLINICAL PERSPECTIVE**

Hospital prognosis of moderate to severe pericardial effusion (MPE; ≥10 mm) in ST-elevation myocardial infarction is largely unknown, and its management poses a therapeutic dilemma because not all patients benefit from emergency surgery aimed at treating an underlying free-wall rupture. Data from 446 ST-elevation myocardial infarction patients, 228 with MPE—88 with cardiac tamponade (CT) and electromechanical dissociation (EMD), 44 with CT and hypotension, and 96 without initial CT—and 218 with small PE (5 to 9 mm), were compared. CT patients showed larger PE ($P<0.001$) than those without initial CT; 85% of those with CT+EMD and 86% with CT plus hypotension were treated with pericardiocentesis, and 10% and 21% were treated with a surgical repair, respectively. Among MPE patients, 30-day mortality was 43% and was higher in those with CT+EMD (operated, 89%; nonoperated, 85%) than in those with CT plus hypotension (22% and 11%, respectively; $P<0.001$) and those without initial CT (17%; $P<0.001$). It was also higher than in patients with small PE (10%; $P<0.001$). Death was attributable to cardiac rupture in 83% of patients with CT+EMD, 7% with CT plus hypotension, and 8% with MPE without initial CT, and it occurred late (≥7 days) in 14%, 67%, and 100%, respectively. Thus, MPE carries an increased mortality, which is highest in patients with CT+EMD. In those with CT plus hypotension, however, mortality is considerably low after pericardiocentesis, and subsequent management may be individualized because a conservative approach is often successful. Importantly, MPE patients without initial CT are not free from late rupture and deserve further investigation.
급성 심근경색 후 중등도 심낭 삽출: 좌심실 파열의 전조

김 덕현 교수 서울아산병원 심혈관과

Summary

대형
급성 심근경색증에 좌심실 파열이 발생하면 심낭에 급속하게 혈액이 차서 전기기계 페리(electromechanical dissociation, EMD) 및 심낭압전(cardiac tamponade)을 유발하게 되고, 적절한 응급 조치가 없으면 사망하게 된다. 그러나 급성 심근경색증이 중등도 심낭 삽출(pericardial effusion, PE)이 동반되는 경우는 좌심실 파열 외에도 심부전, 심방염, 항응고제에 의한 심근 줄럼 등의 원인에 의해서도 발생할 수 있어 항생 및 응급 처치가 필요

방법 및 결과
1965년부터 1987년까지 내원한 4,446명의 연속적인 5년 간의 급성 심근경색증 환자를 대상으로 내원 12시간 내에 심초음파 검사가 시행되었고, parasternal short-axis view에서 이완기말에 축적된 심낭 삽출의 두께가 10mm 이상인 환자 및 중등도 심낭 삽출(moderate to severe PE, MPE) 환자 228명을 대상으로 동반 환자 132명 중 MPE가 동반된 환자가 68명. EMD는 동반되지 않은 환자 중 44명이었고, 85%에서 심낭원착질(pericardiocentesis)이 시행되었는데 EMD가 동반된 환자의 86%에서 동반된 환자의 23%가 사망하였다. EMD가 동반된 환자 9명(10%), 심낭원착질 동반된 환자 9명(23%)에서 동반된 환자는 후 수술이 시행되었는데 EMD 환자 8명(89%)과 심낭원착질 환자 2명(22%)이 수술 후 사망하였다. 특기할 사항으로는 외과 진단 시 경종 PE 소견을 보인 환자 257명 중 38명에서 MPE로 진단하였고, 최초 진단 시 심낭원착 질환이 없었던 MPE 환자 96명 중 88명(89%)에서는 민원 7일 이상 경과하여 심낭원착 및 EMD가 자연 발생하였고 모두 사망하였다. 결과적으로 초기에 심낭원착이 동반되지 않은 MPE 환자의 입원 사망률(20%) 또한 경종 PE 환자의 입원 사망률(11%)에 비해 유의하게 높았다.

결론
EMD를 동반한 MPE의 사망률이 가장 높고, 심낭원착이 동반된 환자들이에서는 심낭원착질 후 폐쇄적적으로 회복되는 경우 보존적인 접근 방법도 효과적일 수 있다. 초기에는 심낭원착이 동반되지 않은 MPE도 양주로 최심실 파열 및 심낭원착으로 진단할 수 있으므로 주의를 요한다.
심근경색증 동반된 대부분의 PE는 혈역학적 영향이 없어서 경증 및 중증도 PE는 심근경색증에 대한 양상 반응(benign reaction)으로 간주되기도 한다. 1986년, Galvão 등이 138예의 연속적인 급성 심근경색증 환자를 대상으로 Circulation에 보고한 연구 결과로 심초음파 일반상의 상 한자들의 28%에서 PE가 관찰되었지만, 심낭압전의 보인 환자는 없었다. 그러나 1986년 이후로 급성 심근경색 증상의 치료는 재관류 요법 중심으로 변화하였고, 그 결과 PE의 반도 및 임상 경과가 변화하였다.

본 연구의 가장 큰 특징은 급성 심근경색증 후 발생한 PE에 대한 임상 연구 중 가장 대규모로 진행된 연구로 PE 환자의 다양한 임상 경과를 살펴볼 수 있다는 점이다. 본 연구에서 PE의 반도가 약 10%로 과거 연구에 비해 감소된 결과를 보였는데, 재관류 치료가 도입된 이후 시행된 다른 연구들에서도 PE의 반도는 10% 이하로 보고되어 재관류 치료가 PE의 발생 반도가 감소한 것으로 판단된다. 본 연구 결과 심낭압전 및 EMD의 소견을 통한 MPE 환자는 늑골isyndrome이 발생한 것으로 판단하여 헬리만 환자를 시행 시 심낭 압전의 hemorrhoid을 중재할 수 있으며 심낭선전환술 시행 시 심낭압전의 hemorrhoid을 중재함으로써 이질중을 나타낼 수 있었다. 심낭압전의 소견이 없어도 중등도 이상의 PE가 관찰되는 경우는 애매한 피로 증상이 중증시에서 염증 모니터링, 심초음파 감시 등을 해야 하고 심낭선전환술 시행을 고려해야 한다.

본 연구의 문헌점으로는 517명 상승 급심경색증 환자를 만한 대상으로 하였음에도 불구하고 재관류 요법의 시행 반도가 50% 이하였으며, 특히 일차적 관통맥 종업이의 시행 반도는 20% 미만으로 경색과 관련된 동맥에서 신속하고 완전한 재관류가 이루어지지 않아서 PE의 발생 반도 및 치유시의 완료의 위험이 증가하였을 것이다. 또한, 심낭압전을 동반한 MPE 환자들에서의 수술 시행 반도가 매우 낮아 심낭하혈출 후 보존적인 치료법과 수술적 치료 방법의 선택을 비교할 수 없었다.

본 연구의 임상적 의미를 요약하면, 첫째, 혈액학적으로