Rupture of the myocardium

Occurrence and risk factors

MIKAEL DELLBORG, PETER HELD, KARL SWEDBERG, ANDERS VEDIN

From the Cardiac Unit, Department of Medicine, Östra Hospital, Gothenburg, Sweden

SUMMARY The occurrence of myocardial rupture was studied in a well defined unselected population of patients with acute myocardial infarction, and the group of patients who died of rupture of the heart were compared with two control groups. Of a total of 3960 patients, 1746 (44%) fulfilled the diagnostic criteria for acute myocardial infarction. Rupture was defined solely on the basis of the presence of a pathological passage through part of the myocardium, either the free wall of the left ventricle or the septum, found at necropsy or during operation. Two controls were selected for each patient and matched for age and sex, one (control group A) with acute myocardial infarction having died in hospital but not of rupture (non-rupture cardiac death) and one (control group B) with acute myocardial infarction having survived the hospital stay. Necropsy was performed in 75% of all fatal cases with acute myocardial infarction. The total hospital mortality was 19%, the highest mortality being among women over 70 years (29%). Ruptures (n=56) were found in 17% of the hospital deaths, or 3-2% of all cases of acute myocardial infarction. Women aged <70 had the highest incidence of rupture, 42% of deaths being due to rupture. The mean age for patients with rupture and controls was 70-5 years. The median time after admission to death was approximately 50 hours for patients and control group A. Thirty per cent of the patients with rupture occurred within 24 hours of the initial symptoms occurring. Angina and previous acute myocardial infarction were more common among control group A. Patients with rupture and control group B were mostly relatively free of previous cardiovascular or other diseases (chronic angina pectoris (>2 months) and previous myocardial infarction). Sustained hypertension during admission to the coronary care unit was more common in patients than in control group A. Hypotension and shock were more common among control group A. Most (79%) of the patients who subsequently ruptured did not receive any corticosteroids at all during the hospital stay. Severe heart failure and antiarrhythmic treatment were more uncommon among patients than among control group A. Patients with rupture received analgesics approximately three times a day throughout their stay. Control group B received analgesics mostly during the first 24 hours.

Thus female patients, patients with first infarcts, and patients with sustained chest pain should be investigated for the possibility of rupture. As many as one third (32%) of ruptures may be subacute, and therefore time is available for diagnosis and surgery.

Rupture of the myocardium after acute myocardial infarction is at present the second most important cause of death after pump failure during hospital admission.1 Rupture may involve the interventricular septum, the papillary muscles, or the free wall, the last site being by far the most common. Previous studies have indicated that increasing age and female sex are important risk factors but have yielded conflicting results regarding the importance of hypertension, steroid medication, prehospital morbidity, and time and frequency of rupture.2-9 Most studies lack adequate controls, are based on necropsy findings, or represent highly selected patient groups.
most are now out of date.

We studied a well defined unselected population of patients with acute myocardial infarction in order to estimate the frequency of rupture and identify the group of patients who die of rupture of the heart. We also compared them with matched controls in order to define groups of patients at increased risk of myocardial rupture.

Patients and methods

A series a consecutive patients admitted to the coronary care unit of this hospital with symptoms occurring within the previous 48 hours and suggesting acute myocardial infarction was studied. The hospital serves a geographically and demographically well defined area with a population of 260,000, from which all cases of suspected acute myocardial infarction are admitted without any preselection regarding age, previous disease, or clinical condition. After admission all patients routinely remain in the unit until they have been without chest pain or arrhythmias for 48 hours. Patients are kept in bed for the initial 24 hours and then mobilised.

From 1 January 1979 to 31 December 1982 a total of 3960 patients were admitted to the unit. Of these, 1746 (44%) fulfilled a diagnosis of acute myocardial infarction based on two of the following criteria: clinical history, typically increased enzyme activity above the normal value, and typical evolution of the electrocardiogram.

DEFINITIONS

Myocardial rupture was defined solely on the basis of the presence of a pathological passage through part of the myocardium, either the free wall of the left ventricle or the septum, found at necropsy or during operation. Thus patients who died with a clinical picture suggesting rupture but who were not examined at necropsy were not included. Five patients with isolated rupture of the papillary muscles were also excluded.

C linical history

Angina—The presence of angina was based on a history of angina on exertion as stated by the patient on admission to the hospital. Patients with angina for less than two months were studied separately.

Previous myocardial infarction—This was defined as a previous admission to hospital with a diagnosis of acute myocardial infarction according to the above criteria.

Hypertension was defined as a history of treatment with antihypertensive drugs because of high blood pressure.

Sustained hypotension was defined as a systolic blood pressure of ≤90 mm Hg on more than two occasions. Hypotension due to any obvious cause (arrhythmia, drug side effect) was excluded as was hypotension occurring during the final two hours before death. Since blood pressure was routinely recorded four times an hour at most hypotension had to be present for at least 30 minutes.

Shock was defined as hypotension (according to definition above) occurring in combination with signs of poor peripheral circulation (coldness, pallor), or oliguria, or mental confusion or all three.

Sustained hypertension was defined as a systolic blood pressure of ≥170 mm Hg or diastolic pressure of >105 mm Hg on more than two occasions during the entire hospital stay. Since blood pressure was routinely recorded four times an hour at most hypertension had to be present for at least 30 minutes.

Heart failure was defined as the presence of basal pulmonary rales or dyspnoea requiring treatment with oral diuretics or intravenous injections of diuretics or increased doses of diuretics in patients already treated with diuretics on admission. Modest heart failure was defined as failure requiring oral or intravenous diuretic treatment (or increased doses) or both twice a day at most. Severe heart failure was defined as that requiring intravenous injections of diuretics three or more times a day.

Inpatient corticosteroid treatment consisted of a single administration of prednisolone (5–10 mg orally) in patients with persistent chest pain of suspected

<p>| Table 1 Distribution of myocardial infarcts and ruptures by age (years) and sex |
|---------------------------------|---------------------------------|-------------------|-------------------|</p>
<table>
<thead>
<tr>
<th>Total No (% of patients with AMI)</th>
<th>Total No (% of deaths)</th>
<th>Myocardial rupture No</th>
<th>% Of AMI</th>
<th>% Of deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;70</td>
<td>815 (46)</td>
<td>105 (13)</td>
<td>15</td>
<td>1-8</td>
</tr>
<tr>
<td>≥70</td>
<td>439 (25)</td>
<td>112 (26)</td>
<td>13</td>
<td>2-9</td>
</tr>
<tr>
<td>All</td>
<td>1254 (72)</td>
<td>217 (17)</td>
<td>28</td>
<td>2-2</td>
</tr>
<tr>
<td>Women:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;70</td>
<td>215 (12)</td>
<td>31 (14)</td>
<td>13</td>
<td>6-0</td>
</tr>
<tr>
<td>≥70</td>
<td>277 (16)</td>
<td>81 (29)</td>
<td>15</td>
<td>5-4</td>
</tr>
<tr>
<td>All</td>
<td>492 (28)</td>
<td>112 (23)</td>
<td>28</td>
<td>5-7</td>
</tr>
<tr>
<td>Men and women (all ages)</td>
<td>1746 100</td>
<td>329 (19)</td>
<td>56</td>
<td>3-2</td>
</tr>
</tbody>
</table>

*Of patients with acute myocardial infarction (AMI).*
pericardial origin not responding to other treatment.

STATISTICAL METHODS
Two controls with acute myocardial infarction were selected and matched for age and sex for each patient with myocardial rupture, one who had died in hospital but not from rupture (non-rupture cardiac death) (control group A) and one who had survived the hospital stay (control group B). The controls were selected from the next two patients in the consecutive patient register after the patient with rupture who fulfilled the matching criteria. The three groups were compared by Fisher’s two tail exact test. Statistical significance was defined as p<0.05.

Results
MORTALITY
A necropsy was performed in 75% of all fatal cases with acute myocardial infarction. Table 1 shows the numbers of patients with acute myocardial infarction, deaths, and myocardial ruptures. The total hospital mortality was 19% and was highest among women over 70 years (29%). Rupture through the free wall (n=46) or the septum (n=5) was found at necropsy in a total of 51 patients and at operation in another five (28 men and 28 women). Rupture was found in 17% of the hospital deaths, or 3.2% of all patients with acute myocardial infarction. Women aged <70 had the highest incidence of rupture, 42% of hospital deaths being due to rupture. The mean age of patients with rupture and the controls was 70.5 years. Figure 1 shows the time from the onset of symptoms to death for patients and controls. The median time from

![Image](https://example.com/image.png)

**Fig. 1** Time from onset of symptoms to death for patients with myocardial rupture (●) and control group A (non-rupture cardiac death) (□).
admission to death was approximately 50 hours for patients and control group A (non-rupture cardiac death). Thirty per cent of the ruptures occurred within 24 hours after the onset of the initial symptoms.

CLINICAL HISTORY
Angina and previous acute myocardial infarction were more common among control group A (Table 2). Patients and control group B (survivors) were in most cases relatively free of previous cardiovascular or other disease—for example, chronic angina pectoris (>2 months) and previous myocardial infarction. On the other hand, angina of recent onset (<2 months before infarction) was more common among patients and control group B than among control group A.

CLINICAL FINDINGS DURING ADMISSION
Table 2 also shows some clinical findings recorded during hospital admission for the three groups. Sustained hypertension while in the coronary care unit was more common among patients than among control group A. Generally, hypertension was associated with survival, whereas hypotension and shock were associated with death. The majority of patients and control group B had normal blood pressure during their hospital stay. During the time in the unit more patients received moderate amounts of corticosteroids intravenously than the two control groups. The vast majority (79%) of the patients who subsequently ruptured did not receive any corticosteroids at all during the hospital stay.

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Severe heart failure and antiarrhythmic treatment were uncommon among patients compared with control group A. No clear cut differences between the three groups as to the site of the infarction could be seen. The controls in group A had large infarcts as estimated by the peak serum aspartate aminotransferase activity, but even higher peak values were obtained in the patient group, being significantly higher than those in control group B (p<0.05).

The degree and duration of chest pain was estimated by determining the cumulated individual number of analgesic injections during the hospital stay (Fig. 2). Patients received analgesics approximately three times a day without any tendency towards a decrease throughout their stay. Controls in group B received analgesics during the first 24 hours but very few after that. The differences after three days were significant for all three groups (p<0.05).

Discussion
The introduction of coronary care units has decreased hospital mortality in cases of acute myocardial infarction. This residual mortality is mainly due to infarctions affecting more than 40% of the myocardium, which lead to severe pump failure and refractory arrhythmias. The second most common cause of inhospital death is myocardial rupture. Primary ventricular fibrillation is no longer an important cause of inhospital deaths.1

In published reports myocardial rupture is commonly divided into four groups: rupture of the papillary muscles, the interventricular septum, and the free wall (acute and subacute rupture). Rupture of the papillary muscles leading to acute mitral incompetence with >90% mortality10 was very rare in our study, the incidence being only 1.5% of inhospital deaths or 8% of all ruptures. Similar figures have been reported by others (Table 3). Rupture of the interventricular septum is apparently more common, between 10% and 20% of all ruptures occurring through the septum.2-5 In our study the figure was 16%. Without surgical treatment 50% of these patients have been reported to die within the first week and 85% within two months.13 14 Surgical correction of this condition is often possible, and patients undergoing successful operation seem to have a relatively good prognosis.13 There is a great deal of controversy as to the optimal time of surgical intervention, although recent studies suggest a more favourable outcome if surgery is performed within 24 hours.12 Acute rupture of the free wall is by far the most common type of rupture, comprising about 50% of all ruptures. Successful surgical intervention has been reported, always including rapid pericardiocentesis, volume expansion, and
Rupture of the myocardium

Table 3 Incidence of myocardial rupture at different sites

<table>
<thead>
<tr>
<th>Study</th>
<th>% Of all ruptures involving:</th>
<th>Total No</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Papillary muscles</td>
<td>Septum</td>
</tr>
<tr>
<td>Zeman and Rodstein11</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>van Tassel and Edwards2</td>
<td>17/5</td>
<td>20</td>
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<tr>
<td>Feneley et al12</td>
<td>8</td>
<td>67</td>
</tr>
<tr>
<td>Present study</td>
<td>8</td>
<td>16</td>
</tr>
</tbody>
</table>

Immediate subsequent surgery using cardiopulmonary bypass.15-17 Subacute rupture of the free wall was described in 1973 by O'Rourke, who reported two cases of rupture of the free wall in which severe hypotension and shock were present for several hours after diagnosis but before death or surgical intervention.18

According to our findings as well as those of others, patients suffering a myocardial rupture are previously healthy compared with other victims of in-hospital deaths.19 20 In the present study the prevalence of hypertension, diabetes, angina, and previous myocardial infarction was as high among patients with rupture as among survivors but lower than that among the control group with non-rupture cardiac death. Thus we cannot agree with previous investigators who have claimed that the absence of previous cardiovascular disease would predispose to heart rupture.20 21 Such a statement seems valid only when other in-hospital deaths rather than survivors served as controls.

The incidence of myocardial rupture as well as the age and sex distribution in our study are consistent with those in other studies.3 8 We also agree with others that women have a higher risk of rupture of the free wall. Prolonged and recurrent chest pain has often been considered to be the most frequent and consistent clinical characteristic of myocardial rupture,8 19 which agrees with our findings.

Corticosteroid treatment has been held responsible for defective healing and scar formation predisposing to cardiac rupture. The basis for this seems to be a case report22 of a single patient who developed a ventricular aneurysm after receiving corticosteroids for 53 days. Roberts et al have reported a trial in which very large doses of methylprednisolone were given repeatedly in accordance with a non-randomised protocol to 12 patients, of whom five died. Two of the victims had rupture of the ventricular septum.7 In our study patients who subsequently ruptured received steroids more often than controls. We believe that this was due to their persistent or recurrent chest pain often being interpreted as a sign of pericarditis and therefore treated with small doses of prednisolone (5-10 mg/day). Nevertheless, since 79% of patients who ruptured did not receive any steroid treatment at all defective healing due to corticosteroid treatment seems to play only a minor role, if any, in the pathogenesis of myocardial rupture.

Hypertension has been considered by several authors as an important pathogenetic factor.5 9 20 We believe that this is a misconception due to the lack of adequate controls. When cardiac death due to rupture is compared with non-rupture cardiac death, hypertension will indeed be more common among the victims with rupture. But the highest frequency of hypertension occurs among patients who survive their infarction. Sustained hypertension may thus be regarded as a sign of better preserved left ventricular function.

It is important to remember that because of the time course of myocardial rupture and the methods used the present study is subject to some bias. Because of the short period of in-hospital observation for cases of rupture the true prevalence of most clinical findings among these cases tends to be underestimated, not only compared with those among survivors. Therefore the observed differences between patients with rupture and survivors underestimate the importance of such differences with regard to sustained hypotension, shock, in-hospital treatment with steroid, antiarrhythmic medication, and enzyme activity. In view of the details of the findings the importance of any of the analysed differences is less likely to be overestimated.

Since 1973, when O'Rourke introduced the term subacute heart rupture,18 several reports of successful surgical treatment of patients with apparently subacute heart rupture have been published.23 Clinically, these patients have generally been severely hypoten-
sive, often in shock, with severe right sided heart failure and sometimes, but not very often, showing classical signs of pericardial tamponade. It has been suggested that this condition might be more readily diagnosed at an early stage by echocardiography, since this technique can detect very small amounts of fluid (15–20 ml) in the pericardial sack.

Table 4 shows previous studies together with the present series in which data indicate the presence of subacute heart rupture. Sustained hypotension, shock, sudden fall in blood pressure, or thrombus formation at the site of rupture have been regarded as indicating subacute rupture. As many as 30% of all ruptures may be subacute.

There is a need for further studies directed towards the treatment and, if possible, prevention of myocardial rupture. These should aim at evaluating techniques for diagnosis before death and at investigating patients with the highest risk of rupture—that is, women <70 years of age with sustained chest pain suffering their first myocardial infarction. Therapeutic interventions should also be evaluated. Because of the nature of myocardial rupture of the free wall, however, treatment has to be directed towards prevention—that is, reduction in infarct size.

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References