In-hospital and Long-term Prognosis of Acute Inferior Myocardial Infarction with Cardiogenic Shock: Clinical Implication of Right Ventricular Infarction

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Abstract

Cardiogenic shock in the setting of an acute myocardial infarction (MI) with left ventricular (LV) failure is associated with an extensive infarct and a poor prognosis. It also occurs due to hemodynamic deterioration from severe right ventricular (RV) dysfunction in RVMI, and adversely affects the prognosis of acute inferior MI. We evaluated the outcomes of patients with acute inferior MI and cardiogenic shock with respect to the presence or absence of RVMI. Subjects consisted of 504 consecutive patients with acute inferior MI. On admission, 159 of the 504 patients were diagnosed as having an RVMI. Of these, 69 developed cardiogenic shock in the acute phase (RVMI (+)/shock (+)), while 90 did not (RVMI (+)/shock (-)). Of the 345 patients without RVMI, 99 developed cardiogenic shock in the acute phase (RVMI (-)/shock (+)), while 246 did not (RVMI (-)/shock (-)). Compared with RVMI (+)/shock (+) patients, patients in the RVMI (-)/shock (+) group had a significantly increased LV dimension (p<0.01) and total wall motion index (p<0.01) and significantly reduced values for LV ejection fraction, RV dimension, right atrial pressure, and the rate of primary coronary angioplasty (all p<0.05). RVMI (-)/shock (+) patients exhibited increased in-hospital mortality (p<0.01) and reduced long-term survival (p<0.01) compared to the RVMI (+)/shock (+) group. Multiple logistic-regression analysis revealed cardiogenic shock, but not RVMI, to be a significant, independent marker for the prediction of long-term prognosis (odds ratio, 3.57; 95% confidence interval 2.05-6.22; p<0.01). In acute inferior MI, cardiogenic shock is an important predictor of worse in-hospital and long-term outcomes. Development of cardiogenic shock following acute inferior MI is associated with a poor prognosis, even in the absence of RVMI.

Key words: inferior myocardial infarction, right ventricular infarction, cardiogenic shock.
Introduction

Cardiogenic shock occurs in 5 to 15% of patients with acute myocardial infarction (MI), and the mortality rates for these patients, when treated with conservative therapy only, are as high as 80 to 100%\(^{8,9}\). Postmortem studies in patients with MI who developed cardiogenic shock have revealed the presence of tissue necrosis involving greater than 40% of the left ventricular (LV) myocardium\(^{49}\). The major cause of cardiogenic shock in these cases appears to be severe LV dysfunction due to extensive LVMI. Acute inferior MI accompanied by right ventricular (RV) MI may also lead to cardiogenic shock, since RV dysfunction leads to a substantial reduction in cardiac output\(^{45}\). Both cardiogenic shock and RVMI associated with acute inferior MI have been reported to be significant, independent predictors of outcome\(^{9,20}\). Thus, it seems clinically important to study the prognosis of patients with acute inferior MI who develop cardiogenic shock with respect to the absence or presence of RVMI. Since no such studies have been reported to our knowledge, we evaluated the effects of cardiogenic shock and RVMI on cardiac function and outcome in patients with acute inferior MI.

Methods

**Study patients**

The subjects consisted of 504 consecutive patients admitted to our coronary care unit with a diagnosis of acute inferior MI between 1980 and 1997. The diagnosis of acute inferior MI was made according to the following criteria: 1) typical chest pain lasting for more than 30 minutes, 2) an increase in the serum creatine phosphokinase level greater than the normal value, and 3) ST-segment elevation of $\geq 0.1$ mV in at least 2 of the 3 inferior leads II, III, or aVF. Blood samples for the measurement of creatine phosphokinase activity were collected every 3 hours until the peak value was recorded. The diagnosis of acute RVMI was made if ST-segment elevation $\geq 0.1$ mV was present in V3R and/or V4R\(^{11,12}\). The diagnosis of cardiogenic shock was based on two specific findings: (1) systolic blood pressure $\leq 80$ mmHg without response to volume loading, (2) signs of acute circulatory insufficiency. Patients presenting in Killip class II or greater were diagnosed as having a complication of congestive heart failure. Hypertension was defined as systolic blood pressure $>160$ mmHg and/or diastolic blood pressure $>90$ mmHg. Hyperlipidemia was considered as a serum total cholesterol level $>240$ mg/dl and/or serum triglycerides $>150$ mg/dl on admission.

**Echocardiography**

Two-dimensional echocardiography was performed within 48 hours of the onset of acute MI. Images were acquired with the patient in the left decubitus position using a Toshiba SSH 65A or 160A echocardiographic system (Toshiba, Tokyo, Japan) or a Hewlett-Packard SONOS 1000 (Andover, MA) with a 2.5-MHz transducer. Left atrial dimension, LV end-diastolic dimension, LV end-systolic dimension, and RV dimension were calculated from the long-axis tomographic images\(^{33}\). The LV wall was divided into 11 segments, and the wall motion index of each segment was scored on a 4-point scale (0=normal, 1=hypokinesis, 2=akinesis, and 3=dyskinesis). The scores for the 11 segments were then summed to obtain the total wall motion index\(^{40}\).

Abbreviations: LV, left ventricular; MI, myocardial infarction; RV, right ventricular.
**Multigated radionuclide angiocardiography**

A total of 378 patients underwent electrocardiographic gated equilibrium blood pool scintigraphy in the subacute period (14 to 30 days after admission, mean 20 ± 5 days), in order to determine the LV and RV ejection fractions. Following intravenous injection of 740 MBq of Tc-99m labeled human serum albumin, data acquisition was performed in list mode with a single crystal gamma camera (model GCA-90B; Toshiba, Tokyo, Japan) fitted with a low energy all purpose parallel hole collimator and interfaced to a dedicated minicomputer (model GMS-55U; Toshiba). The LV and RV ejection fractions were determined from the time-activity curve as measures of global ventricular function.

**Hemodynamic measurements and coronary angiography**

Immediately upon admission to the coronary care unit, a 7.5 F thermodilution catheter (Spectrametto, Liberty Corner, NJ) was introduced to measure the right atrial pressure and the pulmonary capillary wedge pressure, and the cardiac index was determined by thermodilution technique using a Computer Monitor IM-1000 (Spectrametto).

Coronary angiography was performed within 30 days of the onset of acute MI. The angiographic findings were graded according to the American Heart Association classification\(^\text{[50]}\), in which a significant coronary stenosis is defined as a >75% narrowing. Patients were further classified as having 1-, 2-, or 3-vessel disease. Informed consent was obtained from all subjects. Patients who underwent percutaneous transluminal coronary angioplasty within 6 hours from onset of MI were included in the angioplasty group. Patients receiving tissue type plasminogen activator or urokinase within 6 hours from onset of MI were included in the thrombolytic therapy group.

**Statistical analysis**

Values are expressed as means ± standard deviation. Significant differences between two groups were assessed using the Student's t test. To compare 3 or more groups, data were first examined analysis of variance (ANOVA). If significant differences were observed, Bonferroni's correction was used. The Kaplan-Meier method was used to analyze the timing of cardiac death during the 6-year follow-up period. Differences between curves were analyzed using the log-rank test.

Stepwise logistic regression analysis was performed to identify the independent contribution of clinical variables that predict cardiac death. The covariates considered were age, peak serum creatine kinase level, LV ejection fraction, RV ejection fraction, and the presence or absence of cigarette smoking (>10 cigarettes/day), thrombolysis, coronary angioplasty, RVMI, and cardiogenic shock. A Cox proportional-hazards model was used to determine if a significant difference existed in the time to cardiac death in patients stratified by the presence or absence of chronic RVMI.

**Results**

**Clinical characteristics (Table 1)**

Of the 504 patients with acute inferior MI, 159 (32%) had RVMI and 168 (33%) developed cardiogenic shock. Of the 159 patients with RVMI, 69 (43%) developed cardiogenic shock (RVMI (+)/shock (+) group) and 90 (57%) did not (RVMI (+)/shock (−) group). Of the 345 patients without RVMI, 99 (29%) developed cardiogenic shock (RVMI (−)/shock (+) group) and 246 (71%) did
Table 1 Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>RVMI(−)shock(−)</th>
<th>RVMI(−)shock(+)</th>
<th>RVMI(+)shock(−)</th>
<th>RVMI(+)shock(+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>246</td>
<td>99</td>
<td>90</td>
<td>69</td>
</tr>
<tr>
<td>Age (years)</td>
<td>63±12</td>
<td>69±11</td>
<td>66±11</td>
<td>67±13</td>
</tr>
<tr>
<td>Gender (F/M)</td>
<td>58/188</td>
<td>30/69</td>
<td>26/64</td>
<td>22/47</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>130(54)</td>
<td>48(50)</td>
<td>36(42)</td>
<td>29(43)</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>53(22)</td>
<td>22(23)</td>
<td>14(17)</td>
<td>19(29)</td>
</tr>
<tr>
<td>Hyperlipidemia, n (%)</td>
<td>93(39)</td>
<td>23(26)</td>
<td>33(39)</td>
<td>22(35)</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>150(61)</td>
<td>48(48)</td>
<td>57(63)</td>
<td>44(64)</td>
</tr>
<tr>
<td>Peak creatine kinase (IU/L)</td>
<td>2215±2481</td>
<td>3476±2789</td>
<td>2821±2003</td>
<td>4858±3964*</td>
</tr>
<tr>
<td>In-hospital complication</td>
<td></td>
<td></td>
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<tr>
<td>Cardiac death, n (%)</td>
<td>6(4)</td>
<td>51(53)*</td>
<td>2(2)</td>
<td>15(25)††</td>
</tr>
<tr>
<td>Congestive heart failure, n (%)</td>
<td>37(15)</td>
<td>56(58)*</td>
<td>26(29)</td>
<td>31(46)††</td>
</tr>
<tr>
<td>Atrio-ventricular block, n (%)</td>
<td>44(18)</td>
<td>40(41)</td>
<td>32(36)*</td>
<td>43(63)*</td>
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<tr>
<td>Treatment in acute phase</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>t-PA, n (%)</td>
<td>21(9)</td>
<td>14(16)†</td>
<td>3(3)</td>
<td>14(21)††</td>
</tr>
<tr>
<td>Urokinase, n (%)</td>
<td>76(31)</td>
<td>22(22)</td>
<td>26(30)</td>
<td>17(25)</td>
</tr>
<tr>
<td>PTCA, n (%)</td>
<td>25(10)</td>
<td>17(17)</td>
<td>14(16)</td>
<td>16(24)</td>
</tr>
<tr>
<td>Catecholamine, n (%)</td>
<td>43(18)*</td>
<td>76(85)††</td>
<td>38(44)</td>
<td>60(88)††</td>
</tr>
<tr>
<td>CABG, n (%)</td>
<td>51(2)</td>
<td>9(9)‡‡</td>
<td>2(2)</td>
<td>2(3)</td>
</tr>
<tr>
<td>IABP, n (%)</td>
<td>0(0)</td>
<td>21(21)††</td>
<td>0(0)</td>
<td>12(18)††</td>
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</table>

PTCR, percutaneous transluminal coronary recanalization; PTCA, percutaneous transluminal coronary angioplasty; t-PA, tissue-plasminogen activator; CABG, coronary artery bypass graft; IABP, intra-aortic balloon pumping; RVMI, right ventricular myocardial infarction.

*P<0.01 vs. other groups. **P<0.05 vs. other groups. †P<0.01 vs. RVMI(−)shock(−) group. ††P<0.05 vs. RVMI(−)shock(−) group. †††P<0.01 vs. RVMI(+)shock(−) group. ††††P<0.05 vs. RVMI(+)shock(−) group.

not (RVMI(−)/shock(−) group). The incidence of cardiogenic shock was greater in patients with RVMI than in those without RVMI (P<0.01).

The RVMI(−)/shock(+) patients were older than those in the RVMI(−)/shock(−) group (P<0.01). No significant difference was observed with respect to the prevalence of hypertension, hyperlipidemia or diabetes mellitus between the four groups. However, the proportion of smokers was higher in the RVMI(+)shock(+) group than in the RVMI(−)/shock(+) group (P<0.05). The peak level of creatine kinase was greater in RVMI(+)shock(+) patients than in the other groups (P<0.01), and was greater in the RVMI(−)/shock(+) patients than in the RVMI(−)/shock(−) group (P<0.01).

The proportion of patients receiving thrombolytic therapy with tissue-plasminogen activator was greater in the RVMI(+)shock(+) group than in the RVMI(+)shock(−) and RVMI(−)/shock(−) groups (P<0.01); no significant difference was noted between the RVMI(+)shock(+) and RVMI(−)/shock(+) groups. More patients underwent percutaneous transluminal coronary angiography in the RVMI(+)shock(+) group.
than in the other three groups \((p<0.01)\), and
the rate of coronary artery bypass grafting
was greater in the RVMI \((-)/shock (+)\)
group than in the other three groups
\((p<0.05)\). The use of catecholamines and
intra-aortic balloon pumps was higher in the
RVMI \((+)/shock (+)\) and RVMI \((-)/shock (+)\) groups than in the RVMI \((+)/shock (-)\)
and RVMI \((-)/shock (-)\) groups \((p<0.01)\).

**Echocardiographic findings** (Table 2)

Two-dimensional echocardiography was
performed in 466 of the 504 patients within 48
hours of onset of MI. The LV end-diastolic
and end-systolic dimensions were greater in
the RVMI \((-)/shock (+)\) group than in the
RVMI \((+)/shock (+)\) group \((p<0.01\) and
\(p<0.01\), respectively). There was no signifi-
cant difference in left atrial dimension be-
tween the four groups. The RV dimension
was greater in the RVMI \((+)/shock (+)\) and
RVMI \((+)/shock (-)\) patients than in the
RVMI \((-)/shock (-)\) and RVMI \((-)/shock (+)\)
patients \((p<0.01)\). The total wall
motion index was greater in the RVMI \((-) /
shock (+)\) patients than in the other three
groups \((p<0.01)\); and was also greater in the
RVMI \((+)/shock (+)\) group than in the
RVMI \((-)/shock (-)\) group \((p<0.01)\).

**Radionuclide angiographic data** (Table 2)

Electrocardiographic gated equilibrium pool
scintigraphy was conducted in 379 of 504
patients 20.2 ± 5.2 days (mean ± SD) after
admission. The number of patients under-
going scintigraphy was lower in the RVMI \((-) /
shock (+)\) and RVMI \((+)/shock (+)\) groups
than in the other groups because of
the higher mortality rates in the acute stage.
The LV ejection fraction was lower in the
RVMI \((-)/shock (+)\) patients than in other
three groups \((p<0.01)\), but was greater in the
RVMI \((-)/shock (-)\) patients than in the
other groups \((p<0.01)\). The RV ejection
fraction was greater in the RVMI \((-)/shock (-)\)
patients than in the RVMI \((+)/shock (-)\)
and RVMI \((+)/shock (+)\) groups \((p<0.01\)
and \(p<0.01\), respectively).

**Hemodynamic data** (Table 2)

The pulmonary capillary wedge pressure was
greater in the RVMI \((+)/shock (+)\) patients
than in the RVMI \((-)/shock (-)\) group
\((p<0.01)\). The right atrial pressure was
higher in the RVMI \((+)/shock (+)\) and
RVMI \((+)/shock (-)\) groups than in the
RVMI \((-)/shock (-)\) group \((p<0.01)\). It
was also higher in the RVMI \((+)/shock (+)\)
patients than in the RVMI \((-)/shock (+)\)
patients \((p<0.01)\).

**Coronary angiographic data** (Table 2)

Coronary angiography was performed in 263
of the 504 patients immediately after ad-
mission. The proportion of patients with 1-vessel
disease was similar among the four groups,
and no significant differences existed in the
rate of residual stenosis in the right coronary
artery. However, the proportion of patients
with 3-vessel disease was greater in the RVMI
\((-)/shock (+)\) patients than in the RVMI
\((+)/shock (+)\) and RVMI \((+)/shock (-)\)
and RVMI \((+)/shock (+)\) groups \((p<0.05)\).

**In-hospital complications and long-term prog-
nosis** (Table 1)

The incidence of congestive heart failure
was greater in the RVMI \((+)/shock (+)\) and
RVMI \((-)/shock (+)\) groups than in the
RVMI \((+)/shock (-)\) and RVMI \((-)/shock
\(-)\) groups \((p<0.01)\). However, no signifi-
cant difference existed in the incidence of
complications between the RVMI \((+)/shock
(+)) and RVMI \((-)/shock (+)\) groups. The
incidence of atrio-ventricular block was
greater in the RVMI \((+)/shock (+)\) patients
than in the other groups \((p<0.01)\).
In-hospital mortality was increased in patients with cardiogenic shock compared to those without shock [66/168 (40%) vs. 8/236 (2%); *p<0.01]. The in-hospital mortality rate for the RVMI (−)/shock (+) patients was 53%, higher than for the other groups (p<0.01), and in-hospital mortality was found to occur more often in the RVMI (+)/shock (+) group than in the RVMI (−)/shock (−) and RVMI (+)/shock (−) groups (p<0.01).

Kaplan-Meier analysis of the cumulative survival rates 6 years after the onset of MI revealed lower long-term survival rates in the RVMI (−)/shock (+) group than in the RVMI (−)/shock (−) group (p<0.01) and in the RVMI (+)/shock (+) group compared to the RVMI (+)/shock (−) group (p<0.01) (Fig. 1). When cardiogenic shock was present, the long-term survival rate was lower in the RVMI (−)/shock (−) patients than in the RVMI (+)/shock (+) patients (p<0.01).

A multivariate analysis of the clinical indicators of long-term prognosis identified cardiogenic shock (odds ratio, 3.57; 95% confidence interval, 2.05-6.22; p<0.01) and

<table>
<thead>
<tr>
<th>Table 2 Cardiac function and coronary angiographic data</th>
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<tbody>
<tr>
<td>RVM (-) shock (-)</td>
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<td>--------------------------------------------------------</td>
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<tr>
<td><strong>Echocardiographic data</strong></td>
</tr>
<tr>
<td>Number of patients, n (%)</td>
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<tr>
<td>LV end-diastolic dimension (mm)</td>
</tr>
<tr>
<td>LV end-systolic dimension (mm)</td>
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<tr>
<td>Left atrial dimension (mm)</td>
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<tr>
<td>RV dimension (mm)</td>
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<tr>
<td>Total wall motion index</td>
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<td>--------------------------------------------------------</td>
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<tr>
<td><strong>Radionuclide angiographic data</strong></td>
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<tr>
<td>Number of patients, n (%)</td>
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<tr>
<td>LV ejection fraction (%)</td>
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<tr>
<td>RV ejection fraction (%)</td>
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<td>--------------------------------------------------------</td>
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<tr>
<td><strong>Hemodynamic data</strong></td>
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<tr>
<td>Pulmonary capillary wedge pressure (mmHg)</td>
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<tr>
<td>Right atrial pressure (mmHg)</td>
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<tr>
<td>Cardiac index (L/min/m²)</td>
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<tr>
<td><strong>Coronary angiographic data</strong></td>
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<tr>
<td>Total number of patients, n (%)</td>
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<tr>
<td>1-vessel disease, n (%)</td>
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<tr>
<td>2-vessel disease, n (%)</td>
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<tr>
<td>3-vessel disease, n (%)</td>
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<tr>
<td>Multivessel, n (%)</td>
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<tr>
<td>RCA residual stenosis (%)</td>
</tr>
</tbody>
</table>

LV, left ventricular; RV, right ventricular; RCA, right coronary artery; LAD, left anterior descending artery; LCX, left circumflex artery; RVMI, right ventricular myocardial infarction. *p<0.01 vs. other groups. **p<0.05 vs. other groups. †p<0.01 vs. RVMI(−)/shock(−) group. ‡p<0.05 vs. RVMI(−)/shock(−) group. ††p<0.01 vs. RVMI(+) shock(−) group. †‡p<0.01 vs. RVMI(−) shock(+) group.
Prognosis of Acute Inferior Myocardial Infarction

Fig. 1 Kaplan-Meier analysis of the cumulative survival rates for patients with myocardial infarction during a 6-year follow-up. Survival rates are stratified by presence or absence of right ventricular infarction and cardiogenic shock. p=probability.

0, RVMI (-) shock (-) group; □, RVMI (-) shock (+) group; △, RVMI (+) shock (-) group; ○, RVMI (+) shock (+) group.

RVMI=right ventricular myocardial infarction; shock=cardiogenic shock.

age (odds ratio, 1.03; 95% confidence interval, 1.01-1.06; p<0.01) as independent prognostic parameters. RVMI was not an independent predictor of worse outcome.

Discussion

Cardiogenic shock in inferior myocardial infarction

The mortality for patients with acute inferior MI presenting with cardiogenic shock is as high as 40 to 70%, which is similar to patients with anteroseptal MI who develop cardiogenic shock. Our consecutive series of patients with acute inferior MI had an in-hospital mortality of 40% in the presence of cardiogenic shock, which was markedly higher than the rate of 2% for those without shock. The incidence of cardiogenic shock was greater in patients presenting with RVMI than in those without RVMI. The in-hospital mortality rate for patients with acute inferior MI was significantly increased in patients presenting with cardiogenic shock without RVMI than in those having both cardiogenic shock and RVMI, and the long-term outcome also was worse in patients presenting with
cardiogenic shock and without RVMI than in those having both. These results indicate the need to consider the presence or absence of RVMI in evaluating the prognosis of patients with acute inferior MI presenting in cardiogenic shock.

**Cardiogenic shock without right ventricular infarction**

Alonzo et al. reported that the extent of LV damage averaged 51% in patients presenting with shock who died after MI, but only 23% in control patients who died without shock. Shock was also associated with the presence of recent MI and infarct extension. They observed that the total mass of infarction was greater in patients with shock than in those without shock. The SPRINT Study Group also found that the infarct extension was associated with the presence of cardiogenic shock and suggested the utility of early reperfusion therapy for the management of shock to prevent infarct extension. In the present study, patients having cardiogenic shock in the absence of RVMI had a greater total wall motion index, a lower LV ejection fraction, and an increased incidence of congestive heart failure during the acute stage of MI than the other three groups. In addition, their in-hospital and post-discharge mortality rates were greater than any other group. These results indicate that cardiogenic shock in RVMI (-)/shock (+) patients may be attributable to severe LV dysfunction associated with LV infarct extension. Extensive, irreversible injury may occur in the LV myocardium during cardiogenic shock since affected myocardial cells enter into a self-perpetuating cycle of progressive ischemic damage. The worse long-term prognosis in the RVMI (-)/shock (+) appears to be due to a severe reduction in LV function associated with extensive infarction. Furthermore, a significantly greater proportion of patients in the RVMI (-)/shock (+) group had three-vessel disease and required coronary artery bypass grafting during the acute stage. Similarly, the increased presence of multiple-vessel disease was also responsible for the poor prognosis in the RVMI (-)/shock (+) group.

**Cardiogenic shock with right ventricular infarction**

Patients presenting with both RVMI and cardiogenic shock exhibited increased RV dimensions, more elevated right atrial pressures, and an increased incidence of atrioventricular block than patients without RVMI. Patients in the RVMI (+)/shock (+) group exhibited signs of RV dysfunction due to RVMI. The severity of their LV dysfunction was noted to be mild, and their clinical outcome was more favorable than that of RVMI (-)/shock(+). patients.

Cardiogenic shock associated with RVMI has been reported to be reversible in most cases. Most patients with RVMI experience improvement in their clinical signs and RV function during the chronic stage of the disease. Several theories have been advanced to explain these observations. The right ventricle has a more favorable oxygen supply-demand ratio than the left ventricle. This difference has been attributed to the reduced oxygen requirement resulting from its smaller muscle mass as well as to the improved oxygen delivery from the biphasic nature of coronary blood flow during both systole and diastole. In addition, the right ventricle receives a more extensive collateral blood supply. The RV myocardium is perfused directly from the RV cavity by the Thebesian veins. Due to these factors most patients
with RVMI recover RV function over a period of weeks to months\(^6\).

While cardiogenic shock associated with LV myocardial injury often is fatal, cardiogenic shock due to RVMI often is reversible, and cardiac function may improve significantly. Thus, the in-hospital and long-term outcome of patients with RVMI (+)/shock (+) is more favorable than for patients with RVMI (-)/shock (+), LV dysfunction is less severe, and impairment of cardiac function is reversible.

More patients in the RVMI (+)/shock (+) group than the RVMI (-)/shock (+) group required thrombolytic therapy and mechanical revascularization with coronary angioplasty during the acute phase. Early reperfusion therapy with intracoronary thrombolysis can improve prognosis after acute inferior MI\(^{14,18-20}\), and can also improve the course of patients with cardiogenic shock\(^{17,19}\). It seems that the availability of these effective treatments contributed to the better outcomes in the RVMI (+)/shock (+) patients.

**Study limitations**

The present study is a retrospective investigation from 1980 to 1997. During this period, several changes in the treatment for MI occurred which may have affected the mortality and long-term outcome. Treatment modalities were selected for each subject according to the best available therapy at each time-point. However, multivariate analysis demonstrated that treatment was not a significant marker affecting prognosis. Thus, we conclude that changes in treatment modality, if any, during the study period did not significantly affect our results.

Hemodynamic measurements obtained using echocardiography or Swan-Ganz catheterization were performed when possible in the acute phase prior to the start of pharmacotherapy. However, some patients in a state of severe shock received catecholamines prior to hemodynamic assessment, and the effect of these drugs on these determinations could not be excluded.

**Conclusions**

Cardiogenic shock secondary to acute inferior MI has a poor in-hospital prognosis and long-term outcome when LV, but not RV function is impaired. This difference is attributable to extensive LV infarction in the absence of RVMI.

**References**


7) Zehender M, Kasper W, Kauder E, Geibel A, Schonthaler M, Geibel A, Olschewski M