In-hospital Prognosis of Patients with First Nontransmural and Transmural Infarctions

Samer Thanavaro, M.D., Ronald J. Krone, M.D., Robert E. Kleiger, M.D., Michael A. Province, A.M., J. Philip Miller, A.B., Vincent R. deMello, M.D., and G. Charles Oliver, M.D.

SUMMARY We studied the in-hospital mortality and morbidity of 745 patients who had suffered a first myocardial infarction. One hundred twenty-four patients (16.6%) had nontransmural infarction and 621 (83.4%) had transmural infarction. Both groups of patients were similar in the distribution of age, sex, and coronary risk factors. Patients with nontransmural infarction had a significantly lower mortality (3% vs 11%, p < 0.01) and a lower prevalence of premature ventricular complexes (81% vs 88%, p < 0.05). The patients with transmural infarction were distributed evenly among the three subgroups with peak SGOT levels less than 120 units, 120–240 units, and more than 240 units (31%, 34% and 35%, respectively), while most patients with nontransmural infarction (60%) had peak SGOT levels less than 120 units (p < 0.0001). When the in-hospital mortality and morbidity were compared between the parallel subgroups, the prognosis of patients with the two types of infarctions was similar. This study shows that the peak SGOT level is more important than the type of infarction in determining the acute mortality and morbidity of first myocardial infarction.

PREVIOUS STUDIES comparing the clinical course of patients with nontransmural and transmural infarction have reported conflicting results. Mahony and associates found a more favorable prognosis in patients with nontransmural infarction, but Madias et al. and Rigo et al. found no difference in the in-hospital mortality and morbidity between the two types of infarctions. Patients with nontransmural infarction usually have lower serum enzyme elevations than those with transmural infarction, and Scheinman and Abbott reported a better prognosis in patients with nontransmural infarction who had lower enzyme levels than patients with nontransmural infarction and higher enzyme levels or those with transmural infarction.

Peak serum glutamic oxaloacetic transaminase (SGOT) level, which is readily available in most hospitals, has been shown to correlate well with prognosis during acute myocardial infarction (MI). Though the discrepancy in the in-hospital prognosis between the two infarctions in previous reports can be explained in part by different populations studied (some with previous infarctions) or discrepant criteria for the diagnosis of nontransmural infarction, a full explanation has not been made. The present study on 745 patients suffering their first MI was designed to clarify the importance of the type of infarction (transmural vs nontransmural) and the magnitude of myocardial damage, reflected by peak SGOT level, in determining the prognosis of acute MI.

Materials and Methods

The population was selected from 1152 consecutive admissions for acute MI of patients hospitalized within 48 hours of infarction to the Jewish or Barnes hospital coronary care units (CCU) between November 1971 and June 1975. The diagnosis of acute MI was made if two of the following three criteria were present: 1) compatible clinical history, 2) typical pattern of serum enzyme changes and 3) classic ECG changes of transmural infarction.

Only the 773 patients who had no previous documented MI by review of available clinical data and preadmission ECG were considered for this study. Twenty-one patients with persistent left bundle branch block (LBBB), three patients with an inadequate number of serial ECGs for diagnosis of MI and four patients with inadequate SGOT measurement were subsequently excluded from the analysis. The ECG diagnosis of transmural infarction was based on pathologic Q waves (0.04-second duration and amplitude greater than 25% of following R wave) associated with evolving ST-segment and T-wave changes in the same leads or, in the case of posterior infarction, on the presence of pathologic R waves (0.04-second duration with \( RV_1/SV_1 \) or \( RV_2/SV_2 \) equal to or greater than 1) associated with evolving ST-segment and T-wave changes in leads \( V_1 \) and \( V_2 \). The diagnosis of nontransmural infarction was made when the clinical history and serum enzyme elevations were diagnostic of acute MI but the ECG showed no evidence of transmural infarction.

All patients were admitted to the CCU for close surveillance and continuous cardiac monitoring for at least 3 days. Patients were treated according to generally accepted methods for CCUs. Standard 12-lead ECG and serum enzymes were obtained for at least the first 3 consecutive days. Demographic and clinical information was prospectively abstracted from the patients' charts as well as by direct patient interview by trained personnel according to a well-defined protocol. All records were subsequently
transferred to a computerized SAS data base.12 The information was subjected to rigorous checks of consistency and when questions arose, the patients’ charts were reviewed by a senior cardiologist. The patients were divided into two groups, those with transmural and those with nontransmural infarctions. Each group was further divided into three subgroups according to the peak serum SGOT levels: less than 120 IU (low), 120–240 IU (medium) and more than 240 IU (high). The upper limit of normal for serum SGOT at our institution is 40 IU. These peak SGOT levels were chosen because they divided patients with MI into subgroups of approximately equal numbers of patients and because they were used in our previous analyses.13, 14

The in-hospital mortality and morbidity of patients in the two groups as well as in the respective subgroups were compared. We selected the following demographic and clinical features for analysis because we had previously examined their association with the mortality of acute MI: 1) smoking at least one pack of cigarettes per day in the last 6 months before acute MI; 2) any diagnosis of previous diabetes mellitus, regardless of its severity; 3) history of hypertension, ascertained by the patient’s knowledge of previous diagnosis by a physician; 4) cardiac arrhythmias and intraventricular conduction defects while in the CCU, including premature ventricular complexes (PVCs), supraventricular premature complexes (SPCs), paroxysmal atrial tachycardia (PAT), sinus tachycardia, atrial flutter or fibrillation, atrioventricular (AV) blocks (first-degree, second-degree or complete), right bundle branch block (RBBB), left anterior or posterior hemiblocks (hemiblocks), and nonspecific intraventricular conduction defect (IVCD); 5) congestive heart failure during the CCU admission, diagnosed by the presence of vascular redistribution or pulmonary congestion (mild or moderate) on chest x-ray with clinical findings of failure (rales and/or ventricular gallop) or marked pulmonary congestion or pulmonary edema with or without clinical findings of failure; and 6) cardiogenic shock, defined as a systolic blood pressure less than 90 mm Hg with evidence of peripheral vasoconstriction, including sweating, cold extremities, confused sensorium and low urinary output (less than 25 ml/hour).

Statistical analyses were performed with either a chi-square or Fisher’s exact test when cell expectation fell below five. The test for difference in peak SGOT levels of the two infarction types was performed with a t test on the log transform of the peak SGOT levels. A two-tailed probability value of 0.05 or less was considered statistically significant. In addition, a loglinear model14 was fit with the computer program BMDP3F13 in order to determine whether the relationship between the type of MI and the clinical features (mortality and morbidity) remained after the patients were stratified by peak SGOT level.

Results

The inclusion criteria were met by 745 of the 1152 patients admitted with acute MI during the study period. Six hundred twenty-one patients (83.4%) had acute transmural infarction and 124 patients (16.6%) had nontransmural infarction. As shown in table 1, both groups were similar in the distribution of age, sex, and coronary risk factors (smoking, diabetes mellitus and hypertension). However, patients with transmural infarction had significantly higher average peak SGOT levels (231 vs 129 IU, p < 0.001).

When each group was divided into three subgroups according to peak SGOT level, the distribution of age, sex, and coronary risk factors in the parallel subgroups of both types of infarction remained similar. Patients with transmural infarction were distributed evenly between the subgroups with low, medium and high peak SGOT levels (31%, 34% and 35%, respectively). In contrast, the majority of patients with nontransmural infarction (60%) had low peak SGOT levels (table 2). The difference between the two groups in subgroup distribution according to peak SGOT levels was highly significant statistically (p < 0.0001).

The clinical courses of both groups of patients are compared in table 3. Although patients with transmural infarction had a higher prevalence of congestive heart failure and cardiogenic shock, the differences did not reach the significance level. Patients with transmural infarction had a significantly higher mortality (11% vs 3%; p < 0.01). The prevalence of PVCs was higher in patients with transmural infarction (88% vs 81%; p < 0.05). In addition, patients with transmural infarction also had significantly higher prevalences of SPCs, PAT, sinus tachycardia, RBBB and AV blocks. However, when subgroups with comparable peak SGOT levels were compared, these differences were diminished.

| TABLE 1. Demographic History of Patients by Type of Infarction |
|-----------------------------|------------------|------------------|------------------|
|                             | Transmural       | Nontransmural    | p                |
| Mean age (years)            | 61               | 61               | NS               |
| Sex (male)                  | 432              | 428              | NS               |
| History of smoking          | 248              | 248              | NS               |
| History of hypertension     | 242              | 242              | NS               |
| History of diabetes mellitus| 111              | 111              | NS               |

| TABLE 2. Peak SGOT Levels by Type of Infarction |
|---------------------------|-----------------|-----------------|-----------------|
| Peak SGOT levels          | Transmural      | Nontransmural   |                |
| Low (< 120 IU)            | 191             | 74              | 60              |
| Medium (120-240 IU)       | 210             | 36              | 29              |
| High (> 240 IU)           | 220             | 14              | 11              |
| Total                     | 621             | 124             | 100             |

The probability value of a chi-square test for the difference in subgroup distribution of the two infarction types is less than 0.0001.
TABLE 3. Clinical Features By Type of Infarction

<table>
<thead>
<tr>
<th>Feature</th>
<th>Transmural</th>
<th>Nontransmural</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>69 (11)</td>
<td>4 (3)</td>
<td>0.007</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>260 (42)</td>
<td>51 (41)</td>
<td>NS</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>336 (54)</td>
<td>61 (49)</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>46 (7)</td>
<td>4 (3)</td>
<td>NS</td>
</tr>
<tr>
<td>Premature ventricular complexes</td>
<td>545 (88)</td>
<td>100 (81)</td>
<td>0.05</td>
</tr>
<tr>
<td>Supraventricular premature complexes</td>
<td>387 (62)</td>
<td>62 (50)</td>
<td>0.01</td>
</tr>
<tr>
<td>Paroxysmal atrial tachycardia</td>
<td>38 (6)</td>
<td>1 (1)</td>
<td>0.02</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>285 (46)</td>
<td>41 (33)</td>
<td>0.009</td>
</tr>
<tr>
<td>Atrial flutter-fibrillation</td>
<td>71 (11)</td>
<td>8 (6)</td>
<td>NS</td>
</tr>
<tr>
<td>AV blocks</td>
<td>126 (20)</td>
<td>8 (6)</td>
<td>0.0002</td>
</tr>
<tr>
<td>Right bundle branch block</td>
<td>44 (7)</td>
<td>2 (2)</td>
<td>0.02</td>
</tr>
<tr>
<td>Hemiblocks</td>
<td>58 (9)</td>
<td>10 (8)</td>
<td>NS</td>
</tr>
<tr>
<td>Nonspecific IVCD</td>
<td>39 (6)</td>
<td>7 (6)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: AV = atrioventricular; IVCD = intraventricular conduction defect.

The respective subgroups, the mortality and prevalence of PVCs (table 4) as well as the prevalence of SPCs, sinus tachycardia and RBBB associated with the two types of infarction were statistically similar. Furthermore, regardless of the peak SGOT level, patients with either type of infarction showed no difference in the prevalence of cardiomegaly, atrial flutter or fibrillation, hemiblocks or IVCD.

Table 5 shows the partial likelihood ratio chi-square test from the loglinear analysis. This analysis allows a determination of the independent contribution of one or more variables to the specified outcomes. The chi-square values indicate the influence of that particular independent variable on mortality and morbidity when controlling for the other. For example, after adjusting for whether or not an infarction is transmural, peak SGOT level is significantly related to the mortality (chi-square 22.70; p < 0.001). In contrast, after adjusting for peak SGOT level, whether or not an infarction is transmural does not have a significant relationship to the mortality (chi-square 2.34; p > 0.15). The results of this analysis show that the peak SGOT level had substantially more influence on the acute mortality and morbidity than the type of MI.

**Discussion**

Clinicians have attached considerable importance to the differentiation between nontransmural and transmural infarctions, based on the presence or absence of pathologic Q waves on the ECG. A non-transmural infarction has generally been thought to have a better prognosis and a more benign clinical course. However, recent studies comparing the prognosis of patients with the two types of infarction have reported conflicting results. Madias et al.2 and Rigo et al.3 reported a similar mortality and prevalence of cardiogenic shock and cardiac arrhythmias in patients who had either type of infarction. Madigan and associates,4 who included patients with previous infarction in their study, found no mortality or cardiogenic shock in 50 patients with non-transmural infarction. Mahony and associates5 evaluated patients without previous infarction, bundle branch block or left ventricular hypertrophy and

TABLE 4. Clinical Features by Type of Infarction and Peak SGOT Level

<table>
<thead>
<tr>
<th>Feature</th>
<th>Low peak SGOT level</th>
<th>Medium peak SGOT level</th>
<th>High peak SGOT level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Transmural n (%)</td>
<td>Nontransmural n (%)</td>
<td>Transmural n (%)</td>
</tr>
<tr>
<td>No. patients</td>
<td>191 (74)</td>
<td>79 (75)</td>
<td>210 (36)</td>
</tr>
<tr>
<td>Average peak SGOT level</td>
<td>79 (5)</td>
<td>3 (4)</td>
<td>176 (170)</td>
</tr>
<tr>
<td>Death</td>
<td>9 (5)</td>
<td>17 (8)</td>
<td>17 (3)</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>68 (36)</td>
<td>78 (37)</td>
<td>100 (48)</td>
</tr>
<tr>
<td>CHF</td>
<td>95 (50)</td>
<td>84 (46)</td>
<td>18 (50)</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>10 (5)</td>
<td>7 (3)</td>
<td>29 (13)</td>
</tr>
<tr>
<td>PVCs</td>
<td>156 (82)</td>
<td>184 (88)</td>
<td>31 (86)</td>
</tr>
</tbody>
</table>

Abbreviations: CHF = congestive heart failure; PVCs = premature ventricular complexes.
reported a lower mortality and morbidity in patients
without transmural infarction.
Because the patient’s prognosis has been shown to
be related to the total amount of myocardium lost
from all the infarctions rather than to the extent of the
most recent myocardial damage,22 the inclusion of
patients with previous infarction in some of the above
studies may have contributed to the different con-
ductions drawn.4 Therefore, patients with previous
infarction and LBBB were excluded from our study.
The amount of myocardial damage exerts an im-
portant influence on the clinical course and prognosis
of patients after MI.23,24 While many studies have
relied upon elevations of serum creatine kinase and its
isoenzymes to indicate the extent of myocardial
damage,25 peak SGOT level has also been corre-
lated with the amount of myocardial necrosis.27
Furthermore, the peak SGOT level, which is almost
universally available for clinical use, has been shown
to be a good predictor for the mortality and morbidity
of patients with MI.7,9
We found that patients whose first MI is non-
transmural usually have significantly lower serum en-
zeyme levels than those with transmural infarction.
Although several authors2,3,4 have also reported the
difference in enzyme elevations between the two types
of infarction, very few of them used this observation to
explain their findings. Scheinman and Abbott4 found a
better prognosis in patients with nontransmural
infarction and lower enzyme (SGOT or lactic
dehydrogenase) levels when compared with those with
nontransmural MI and higher enzyme elevations or
patients with transmural infarction. However, they
did not indicate the exact ranges of the enzymes used
for their division and, in addition, they also included
patients with previous infarctions in their study. Our
present prospective study on a large number of
patients allowed a systematic analysis for the indepen-
dent contribution of the enzyme (peak SGOT) level
and the type of infarction to the prognosis of the first
MI. Regardless of the peak SGOT levels, patients
with nontransmural infarction had a better prognosis
than those with transmural infarction. However,
regardless of the electrocardiographic changes of non-
transmural or transmural infarction, we found a
similar mortality and morbidity in the group with
comparable peak SGOT levels.
We were concerned that by dividing our patients
into subgroups, the smaller numbers in each subgroup
might prevent us from detecting a true relationship
between the type of infarction and the clinical features,
especially the mortality, independent of the peak SGOT level. The loglinear model is a statistical
 technique that addresses this issue. The results of this
analysis (table 5) show that the mortality and mor-
bidity are highly related to the peak SGOT level.
Although there may be an association between the
type of MI and mortality, the association we found is
weak ($p > 0.15$) and could have arisen by chance.
Furthermore, recent evidence casts doubt on the
ability of the clinician to determine from the ECG
whether an infarction is transmural.29,30 Because most
patients with nontransmural infarction generally have
lower serum enzyme levels,2,3,4 the presence or
absence of abnormal Q waves may be only a con-
vienent way of indicating the extent of myocardial
damage and may not reflect the true pathologic type
of infarction.
In conclusion, the better prognosis of electrocardi-
ographically diagnosed nontransmural infarction is
attributed to the lesser myocardial damage in this
group of patients. The acute mortality and morbidity
of first MI are more closely related to the peak SGOT
level than to the electrocardiographic type of infarc-
tion.

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