Identifying the risk of death following hospital discharge in patients admitted with a suspected acute myocardial infarction in whom the diagnosis is not confirmed

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Aims To describe clinical factors, available at the time of discharge, that predicted survival of patients admitted with a suspected acute myocardial infarction in whom the diagnosis was not confirmed.

Methods and Results A cohort study based on the Nottingham Heart Attack Register of 1716 sequential patients discharged alive from two acute teaching hospitals following admission in 1992. The main outcome was identification of high and low mortality risk groups over 5 years of follow-up. Overall 5-year survival was 58% (95% CI 56 to 60%). Having abnormal cardiac enzyme changes or an abnormal ECG that was insufficient to meet established diagnostic criteria of myocardial infarction, or both, identified three groups with a 5 year survival of 77%, 60% or 51%. Multivariate methods were used to develop a risk score from seven variables available at the time of discharge (age, sex, past history of myocardial infarction, ECG abnormalities, cardiac enzyme abnormalities, Killip score of 2 or 3 on admission and being discharged on a diuretic). Quartiles of this risk score then identified four groups with 5 year survival ranging from 89% to 25%.

Conclusion Among the study cohort, it was possible to identify subgroups with a markedly different risk of subsequent mortality from clinical indicators that were readily available at the time of hospital discharge. Risk stratification has the potential to improve targeting of subsequent secondary preventive efforts, but further work is required to ascertain whether cardiovascular risk can be modified through a more intensive approach to management.


Key Words: Suspected myocardial infarction, survival, risk stratification.

See page 180 for the Editorial comment on this article


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Introduction

Up to 35% of all acute emergency admissions to hospital are attributable to patients in whom an acute myocardial infarction is suspected[1]. Using established WHO criteria based on the clinical history, serial cardiac enzyme and ECG changes[2,3], patients will develop either major changes sufficient to diagnose an acute myocardial infarction or minor changes suggestive of some other acute coronary syndrome such as a non-Q-wave myocardial infarction or unstable angina, while in some the lack of ECG or enzyme changes may suggest a diagnosis of non-cardiac chest pain. Widely differing case definitions exist[1,4] but in the U.K., one definition within which many of these patients with an unconfirmed myocardial infarction have been grouped is ‘possible myocardial infarction’[5,6]. These patients have some non-diagnostic ECG abnormality or a slight rise in cardiac enzymes. Patients presenting in this way are less well investigated[5,7], with very little published work on identification of future high and low risk of subsequent death. The mortality in this group of patients, following hospital discharge, is worse than that experienced by patients with a confirmed myocardial infarction[8]. However, patients in the ‘possible myocardial infarction’ category comprise a heterogeneous group which includes acute coronary syndromes as well as non-cardiac chest pain. Identifying those at differing levels of risk could help distinguish those at low risk who could
safely be discharged, from those at high risk who might benefit from closer monitoring, investigation and follow-up.

The aims of this study were to explore clinical factors, available at the time of hospital discharge, which were associated with survival following discharge in a cohort of patients admitted with a suspected myocardial infarction, where the diagnosis was not confirmed and patients were given the diagnosis of ‘possible myocardial infarction’, and to identify high and low risk groups as an aid to subsequent management.

**Methods**

The study cohort and method of assembly has been described in detail elsewhere\(^8\). The cohort was assembled using the existing Nottingham Heart Attack Register (NHAR), which has collected data on all patients admitted to Nottingham’s two acute hospitals with suspected myocardial infarction since 1973. The operation and case definitions used in the register have been reported elsewhere\(^6,8\). Patients in this study fulfilled the case definition of ‘possible myocardial infarction’ as follows:

*Possible myocardial infarction*

Good history for acute myocardial infarction and either ECG changes other than new Q waves or a rise in cardiac enzymes (creatine phosphokinase and lactate dehydrogenase) but to less than twice the upper limit of normal, or both.

*Tracing patients listed on the NHAR*

For the purposes of this study, the index admission was the first admission with the diagnosis of ‘possible myocardial infarction’ during the year 1992 and patients had to be resident in the Nottingham District Health Authority (DHA) area. Patients were traced using the DHA computer database of patients registered with local general practitioners (GPs). Tracing and identification of cause of death data as at 31 December 1997 on the study cohort have been reported\(^3\).

*Identification of cause of death*

Death registration data from the Office of National Statistics (ONS) containing International Classification of Disease revision 9 (ICD-9) coded information was used to determine the cause of death\(^10\). Details of the methods used to describe the Cause of Death have been reported previously\(^3\).

*Clinical variables*

These were directly available in the patient records at the time of discharge. The variables under study were chosen in advance using an evidence-based approach combined with information known to be routinely clinically available in the majority of settings in the U.K. The Townsend\(^9\) score of the enumeration district of the patients’ home address at the time of admission (derived from the patients full postcode) was used as a proxy for deprivation\(^10,11\). The number of variables chosen was also influenced by the need to ensure there was a reasonable ratio of deaths to parameters being investigated.

**Statistical analysis**

Survival times were calculated by counting the time (in days) from hospital discharge to death or last date known to be alive, with termination of follow-up for all subjects on 31 December 1997. Kaplan–Meier survival curves\(^12\) were constructed for all members of the cohort who had been discharged alive. Cox’s proportional hazards model\(^13\) was used to explore associations between defined clinical variables available at the time of discharge and death. The main outcome measure was 5 year survival. All statistical analyses were carried out using SPSS for Windows version 8.0.0.\(^14\). To derive a risk stratification model, the cases were split (in the ratio 3:1) into a training set to develop a model and a validation set to explore its performance, using Harrell’s ‘c’\(^15\). Harrell’s ‘c’ is a rank-based measure of the concordance between a prediction and an outcome: if a high prognostic index denotes a bad prognosis then when two subjects are compared, the subject with a higher index should have a worse prognosis. The values of ‘c’ range from 0 to 1. A value of 0.5 indicates that concordance is no better than chance and a value of 1 indicates perfect concordance. A value of 0 indicates perfect disagreement. If the outcome is binary (as in dead or not dead) then the value of ‘c’ is interpretable as the area under a receiver operating characteristic curve. A separate validation dataset was used because prognostic indexes are tailored to fit the data they were derived from, and so their performance in practice is often less good. The use of the validation set also provides confidence limits for the discriminating power, on which to base sample size for studies designed to check the validity of the risk score.

The validity of the proportional hazards assumptions for the risk scores were assessed by informally inspecting plots of cumulative hazard on a logarithmic/logarithmic scale for the four categories of risk, with parallel plots indicating proportionality.

*Assessment of risk of subsequent mortality following admission: deriving risk scores*

Two approaches were used to explore the association between clinical variables available at discharge and subsequent mortality.
ENZYMES.

had ECG abnormalities and 54% abnormal cardiac enzymes. Eighty seven percent of patients with cardiac enzyme abnormalities (n=915) were no more likely to die during follow-up than those with normal cardiac enzymes (57% vs 60%, P=0.367). Patients with ECG abnormalities in hospital (n=1486) had a worse 5 year survival than those with normal ECGs (n=220) (56% vs 77%, P<0.0001). Patients with cardiac enzyme abnormalities (n=915) were no more likely to die during follow-up than those with normal cardiac enzymes (57% vs 60%, P=0.39).

The extended clinical approach

ECG and cardiac enzyme findings (as defined in the case definition) were used to identify three risk groups in the study patients:

(a) any abnormality of ECGs and any rise in cardiac enzymes during the index admission;

(b) abnormal ECG with normal cardiac enzymes; and

(c) abnormal cardiac enzymes with normal ECGs.

The standard clinical approach

Extended clinical approach

Coefficients derived from the Cox regression analysis were used to construct an individual’s risk score based on the presence or absence or value of each variable. These were then used to assign each individual a quartile of risk from lowest (group 1) to highest (group 4) risk.

Results

The characteristics of the cohort have been reported elsewhere[8]. Briefly, the 1716 patients comprised 1005 males (mean age 65·2 years) and 711 females (mean age 70·7 years). Thirty four percent gave a past history of myocardial infarction (n=1128) was greater than those who gave a history of myocardial infarction (63% vs 50%, P<0.0001).

Patients with ECG abnormalities in hospital (n=1486) had a worse 5 year survival than those with normal ECGs (n=220) (56% vs 77%, P<0.0001). Patients with cardiac enzyme abnormalities (n=915) were no more likely to die during follow-up than those with normal cardiac enzymes (57% vs 60%, P=0.39).

Table 2 lists the variables associated with subsequent 5 year mortality in multivariate analysis, and which were included in the model used to derive the prognostic risk assessment. The results presented are those derived from the training set (1299 cases). The variables used in the model were: age at the time of the index admission, presence of heart failure on admission (Killip grades 2 or 3), the presence of abnormal ECG or cardiac enzyme findings during admission, a history of previous myocardial infarction, male sex and being discharged on a diuretic. There was no significant interaction between abnormal ECG findings and a past history of myocardial infarction in the Cox multivariate model. The beta coefficients in the multivariate model were used to construct the ‘extended model’.

Risk scores

Clinical variables associated with subsequent mortality

Table 1 lists significant univariate associations between individual clinical variables present at the time of the index admission and subsequent mortality. All variables chosen for analysis are reported in the Table. There was a highly significant association between cumulative mortality and increasing age with 5-year survival ranging from 93% in those under 45, to 25% in those over 85. After adjusting for age, women had a better survival (69% after 5 years) than men (59% after 5 years, P<0.0001). Five-year survival in those with no previous history of myocardial infarction (n=1128) was greater than those who gave a history of myocardial infarction (63% vs 50%, P<0.0001).

Table 1 Clinical variables associated with subsequent survival in the 1992 cohort: univariate analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>P value</th>
<th>Relative risk (exp(B))</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0·054</td>
<td>&lt;0·0001</td>
<td>1·06*</td>
<td>1·05 to 1·06</td>
</tr>
<tr>
<td>Deprivation (Townsend score)</td>
<td>0·013</td>
<td>0·292</td>
<td>1·01</td>
<td>0·99 to 1·04</td>
</tr>
<tr>
<td>Male sex</td>
<td>0·035</td>
<td>0·688</td>
<td>1·04</td>
<td>0·87 to 1·23</td>
</tr>
<tr>
<td>Abnormal cardiac enzymes</td>
<td>0·080</td>
<td>0·349</td>
<td>1·08</td>
<td>0·92 to 1·28</td>
</tr>
<tr>
<td>Previous documented MI</td>
<td>0·367</td>
<td>&lt;0·0001</td>
<td>1·44</td>
<td>1·22 to 1·71</td>
</tr>
<tr>
<td>Killip class 2 or 3</td>
<td>0·775</td>
<td>&lt;0·0001</td>
<td>2·17</td>
<td>1·74 to 2·70</td>
</tr>
<tr>
<td>Abnormal ECG</td>
<td>0·787</td>
<td>&lt;0·0001</td>
<td>2·20</td>
<td>1·58 to 3·06</td>
</tr>
<tr>
<td>Discharged on a diuretic</td>
<td>1·226</td>
<td>&lt;0·0001</td>
<td>3·41</td>
<td>2·85 to 4·07</td>
</tr>
</tbody>
</table>

*per unit change in variable

Variables significant in the training set (n=1299) reported

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<th>Variable</th>
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Risk scores

Standard clinical approach

Figure 1 shows the Kaplan–Meier curves for survival for the three risk categories based on ECG and cardiac enzyme results alone. These risk categories had highly significantly different survival figures after 1 year of 89%, 79% and 70% (P<0.0001) and 5 year survivals of 77%, 60% and 51% respectively (P<0.0001). For the training set only, the one year survival was 93%, 84% and 79% at one year and 75%, 59% and 50% at 5 years respectively.

Extended clinical approach

The clinical variables detailed in Table 2 were used to develop a simplified ‘bedside’ version of a risk score.
and the distribution of these risk scores is shown in Fig. 2. The extended approach was more discriminating than the forecast based on the ECG and cardiac enzyme results alone. This risk scoring system identified four groups and survival by quartiles is shown for the training set on a Kaplan–Meier plot (Fig. 3), plotted alongside survival of patients with a confirmed myocardial infarction for the NHAR during the same year (1992). For the training set, the 1 year survival for each extended risk score category were 96%, 90%, 79% and 69% respectively ($P<0.00001$), with 5 year survivals of 89%, 68%, 47% and 25% respectively ($P<0.0001$). The

### Table 2 Clinical variables associated with subsequent survival in the 1992 cohort: multivariate analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
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<th>RR (95% CI)</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.047</td>
<td>$&lt;0.001$</td>
<td>1.05*</td>
<td>1.04 to 1.06</td>
</tr>
<tr>
<td>Male sex</td>
<td>0.410</td>
<td>$&lt;0.001$</td>
<td>1.51</td>
<td>1.26 to 1.80</td>
</tr>
<tr>
<td>Abnormal cardiac enzymes</td>
<td>0.225</td>
<td>0.011</td>
<td>1.25</td>
<td>1.05 to 1.50</td>
</tr>
<tr>
<td>Previous documented MI</td>
<td>0.192</td>
<td>0.030</td>
<td>1.21</td>
<td>1.02 to 1.44</td>
</tr>
<tr>
<td>Killip class 2 or 3</td>
<td>0.241</td>
<td>0.037</td>
<td>1.27</td>
<td>1.01 to 1.60</td>
</tr>
<tr>
<td>Abnormal ECG</td>
<td>0.387</td>
<td>0.028</td>
<td>1.47</td>
<td>1.04 to 2.08</td>
</tr>
<tr>
<td>Diuretic on discharge</td>
<td>0.749</td>
<td>$&lt;0.0001$</td>
<td>2.11</td>
<td>1.74 to 2.57</td>
</tr>
</tbody>
</table>

Variables significant in multivariate model applied to the training set (n=1299) reported.

*per unit change in variable.

### Figure 1 Survival based on ECG and cardiac enzyme results.

### Figure 2 Distribution of extended risk scores.
overall performance of the risk score was shown to be moderately good: Harrell’s 'c' was 0.715 (training set) and 0.721 (validation set, n=425). The standard error of 'c' in the validation set was estimated (by bootstrapping) to be 0.01746, which also confirmed its normal distribution (Kolmogorov-Smirnov One Sample Test P=0.903).

The standard clinical approach using three groups based on ECG and cardiac enzyme results performed less well on a similar training and validation set exercise with Harrell’s 'c' of 0.5657 and 0.5708 respectively.

Secondary prevention

Data were available on drug treatment (other than lipid lowering agents) on discharge. The use of aspirin and angiotensin converting enzyme inhibitors (ACEI) was compared across the four ‘extended risk’ groups. The use of aspirin was similar in all four risk groups (range 44% to 53%). The use of beta-blockers was much higher in the ‘best’ compared with the ‘poorest’ survival group (32% v 10% respectively) but ACEI use was highest in the ‘poorest’ survival group (20% vs 8% respectively). The proportion who received either ACEI or beta-blocker on discharge varied between 31% and 45%, but there was no significant difference across risk groups.

Cause of death

Overall, 53% of deaths during follow-up were attributable to a cardiovascular cause (55% in the training set). For the training set, there were no significant differences in the proportion of patients assigned a cardiovascular Cause of Death, depending on the presence or absence of different methods of risk stratification. This was the case for groups based on the presence or absence of ECG changes at the time of the index admission (56% vs 50% respectively; difference 6%, 95% CI –10 to 22%, ECG/cardiac enzyme groupings (57%, 54% and 47%) or the extended risk score (56%, 50%, 52% and 59%).

Data from a subgroup (dying during 1994 or subsequently) with full cause of death available from ONS (n=222) showed that a designated cause of ‘cardiovascular’ in this subgroup was 53%, compared with 43% for cause of death designated as ‘cardiovascular’ based on 1a alone. For the whole cohort, there was a non-significant fall in the proportion of cases dying from cardiovascular causes at the end of each completed year of follow-up, from 61% during year one to 47% during year 5.

Discussion

The management of patients admitted with a suspected acute myocardial infarction which is later confirmed is guided by the results of randomized clinical trials[16] or expert panels[17]. A few patients in whom an infarction has been ruled out may be recalled to clinic because an underlying cardiac cause is considered likely, but the majority will be discharged to the general practitioner’s care with no formal hospital follow-up to establish the reason for hospital admission. Nevertheless, the mortality of this group of patients may be greater than those who have sustained and survived an acute myocardial infarction[8].

In the construction of the model, the clinical variables chosen a priori were fitted simultaneously and non-significant terms subsequently rejected. No other interaction terms were included to allow the construction of a clinical risk score, for which simplicity was important. No models were amended following validation. In fact, no diminution in discriminating power (shrinkage) was observed. If there had been, the prognostic index
(extended risk score) would have been reported as originally defined together with the degree of shrinkage. This validation step confirmed that there had been no overfitting of the model to the data, but not the extent to which the discriminating power would be degraded if the index were applied to other, possibly different patient populations.

The risk stratification identified in the extended model described here is strongly influenced by age and by proxy measures for heart failure (diuretic prescription on discharge), as indicated by the relative risk values in Table 2. However, the model does provide significant additional prognostic information in the situation where a clinician may have two patients of similar age or heart failure status. We feel that such an approach may improve the identification of high-risk individuals and justifies the modest extra time required.

Ideally, all patients would be screened to establish the cause of symptoms leading to hospital admission and given some estimate of the likelihood of coronary disease. The principle of risk stratification is well established following a confirmed infarction, where, for instance, sophisticated cardiac measurements have identified subgroups with between 3% and 60% subsequent 2-year mortality. Recognizing high-risk individuals following an unconfirmed infarction has also been described. Launbjerg et al. reported that an abnormal ECG recorded during the index admission was associated with an increase in 7-year mortality from 10% to 50%, with other non-invasive investigations having similar predictive value. Millaine et al. have proposed that an unchanging ECG during admission is a simple and useful way of identifying those who may be discharged safely within 24 h. Our study supports the concept that a normal ECG is of practical value because it identifies a patient at low risk who might be discharged, confident that the subsequent prognosis is good. Even so, as our study shows, the sensitivity of risk stratification can be improved further using additional clinical information that is readily available at the time of discharge. Simplification of the risk calculation did not lead to a reduction in the predictive value and simple addition based on the presence or absence of seven clinical variables provided a rapid bedside indication of likely subsequent survival. The choice of factors considered for analysis was limited to those readily available and accepted in clinical practice, and to keep the risk-stratification model simple.

Of particular importance, our proposed model can identify first subgroups of patients with 5-year mortality rates greatly in excess of their age- and sex-matched peers (and higher than after a confirmed myocardial infarction); and second subgroups of patients with mortality rates similar to those expected in the general population. The high proportion of cases whose death certification mentioned a cardiovascular cause is important, although possible bias (in the completion of death certificates of patients recently admitted with a suspected myocardial infarction) means that this data should be viewed with caution.

Because death from coronary disease is common in this group of patients, providing a model with greater powers of discrimination increases the options available to clinicians. The model allows clinicians additional information to aid management decisions at the time of discharge: patients at ‘very low risk’ can be informed that, whatever the cause of symptoms, their prognosis is likely to be excellent; patients at a pre-specified ‘high risk’ could be followed-up and investigated more intensively for coronary disease. We do not know what effect the practical application of such a risk stratification policy would be on subsequent health service utilization, mortality or morbidity. Although we did not demonstrate statistically significant differences in the uptake of effective interventions between the risk groups, the power of this observational study to detect such differences was low and should encourage further work to address this a priori. There is evidence that psychological morbidity is high following discharge in some patients where a diagnosis of myocardial infarction is not confirmed. Being able to offer reassurance or a clear management plan for those at higher risk at the time of discharge may reduce such psychological distress, but this too requires further exploration.

Our observational study has shown that high-risk patients discharged after an unconfirmed infarction can be identified using simple, readily available clinical information. While establishing risk status is important, what is really needed is evidence that there are potential health gains of a policy targeted at high-risk individuals, involving regular monitoring, appropriate investigation and treatment that may influence outcome. This can only be achieved by conducting a randomized clinical trial.

The Nottingham Heart Attack Register (NHAR) was established by J.R.H. in 1972. We would like to thank all the staff of the NHAR for continued help in gathering the data, the GPs and hospital clinicians for allowing access to patient information used in the report, to Jean Robinson and Kathy Scriven for help in tracing the patients, and to Dr Sarah Wilson, all of Nottingham Health Authority, for additional support.

References

Appendix

**Full extended model**

The calculation of risk score was derived from the B values listed in Table 2. The final score was calculated as:

$$\text{risk score} = (0.047 \times \text{age at MI}) + (0.41 \text{ if male}) + (0.192 \text{ if documented previous MI}) + (0.241 \text{ if Killip 2 or 3 on admission}) + (0.387 \text{ if any ECG abnormality during admission}) + (0.225 \text{ if any cardiac enzyme abnormality during admission}) + (0.749 \text{ if discharged on a diuretic}).$$

The risk score provided a means of assessing subsequent survival. For the cohort under study, the results were divided into quartiles:

<table>
<thead>
<tr>
<th>Risk scores</th>
<th>Average subsequent 5 year survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 3·88</td>
<td>89%</td>
</tr>
<tr>
<td>3·88 to less than 4·63</td>
<td>68%</td>
</tr>
<tr>
<td>4·63 to less than 5·23</td>
<td>47%</td>
</tr>
<tr>
<td>5·23 or more</td>
<td>25%</td>
</tr>
</tbody>
</table>

Simple risk score (for clinical 'bedside' use). A simplified 'bedside' clinical scoring system derived from the above extended approach:

$$\text{Age in years divided by 20} + 0.75 \text{ if discharged on a diuretic} + 0.25 \text{ if Killip 2 or 3 on admission} + 0.2 \text{ if previous history of MI} + 0.5 \text{ if male} + 0.3 \text{ if any ECG abnormality during admission}.$$

This gives a score which indicates subsequent 5 year survival within the following groups:

<table>
<thead>
<tr>
<th>5 year survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 3·93</td>
</tr>
<tr>
<td>3·93 to less than 4·58</td>
</tr>
<tr>
<td>4·58 to less than 5·23</td>
</tr>
<tr>
<td>5·23 or more</td>
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