Sources of diagnostic inaccuracy of conventional versus new diagnostic criteria for myocardial infarction in an unselected UK population with suspected cardiac chest pain, and investigation of independent prognostic variables

J Trevelyan, E W A Needham, S C H Smith, R K Mattu

Objective: To assess the degree and sources of current diagnostic inaccuracy of serial conventional cardiac markers and ECGs compared with the new diagnostic criteria for myocardial infarction, with specific reference to physician specialty and the prognostic value of troponin T.

Design: Prospective, blinded observational study.

Setting: University hospital.

Patients and interventions: All suspected cardiac chest pain admissions for six months, with additional blinded measurement of CK-MB mass and troponin T. World Health Organization and new criteria myocardial infarction diagnoses were made by an expert panel.

Main outcome measures: Diagnostic adjustment by expert panel; completeness of serial measurements; six months prognosis.

Results: A complete set of serial cardiac markers was not taken in 38.7% of patients, this being twice as likely when managed by non-cardiologists than by cardiologists (p = 0.0001). The WHO myocardial infarction diagnosis was adjusted by the expert panel in 4% of cases, this being 90% more likely in patients admitted under non-cardiologists (p = 0.026). The new criteria for myocardial infarction identified an additional 27.3% of infarcts, with a diagnostic alteration in 12.0% of the cohort; 45.2% of these cases had an additional cause for diagnostic adjustment. Only troponin T (p = 0.0004), ST depression (p = 0.0003), and heart failure (p = 0.016) were independently predictive of prognosis.

Conclusions: Chest pain patients appear less likely to be fully and accurately assessed by non-cardiologists than by cardiologists. The new criteria for myocardial infarction identify ~25% of additional patients as MI, with potential additional advantages related to simplicity of diagnostic protocols. Troponin T was the most potent predictor of six month prognosis in an unselected cohort of chest pain admissions.
with a blinded expert panel using the new diagnostic criteria for myocardial infarction for comparison. The specialist status of the attending physician was documented, and the care of the patient was free from interference or influence by the review panel. We also examined the impact of troponin T on the six months outcome in relation to major adverse cardiovascular events compared with other clinical and biochemical variables in our unselected cohort.

METHODS

Patients

All patients over the age of 18 years admitted with suspected cardiac chest pain to University Hospitals of Coventry and Warwickshire over a six month period were enrolled. The patients were divided into those admitted under a cardiologist (coronary care unit or specialist cardiology ward) or general physician (medical ward). Patient management by the admitting team was based upon conventional criteria using serial ECGs and daily CK/AST. Extra blood samples were drawn at 12 hours after symptom onset for the measurement of CK-MB mass and cTnT. At no stage was the admitting team aware of these results.

Following venesection, blood samples were stored at 4°C for 30 minutes, centrifuged at 3000g for 10 minutes, and the serum stored at −80°C before batch analysis. The serum concentration of myoglobin, CK-MB mass, and cTnT were measured quantitatively with the Elecsys 1010 machine, using nine minute STAT assays (Roche Diagnostics UK, Lewes, East Sussex, UK). The reference range for the cTnT assay in our laboratory was validated and underwent appropriate quality control against samples of known concentration. Our data were therefore analysed according to the manufacturer’s recommended discriminator at that time, with an abnormal result defined as cTnT > 0.1 µg/l.

Sensitivity of the cTnT test is 0.01 µg/l. All analyses were done blind at a single laboratory, without knowledge of the patient’s diagnosis. Coefficients of variation for the assays were: cTnT, 4.8% at 0.15 µg/l and 3.9% at 5.7 µg/l; CK-MB mass, 4.0% at 6.3 µg/l and 4.0% at 55.8 µg/l. Investigation of our CK-MB mass assay in a sample population of 1388 individuals generated median values of 0.97 µg/l in women and 1.35 µg/l in men, yielding 97.5 centile values of 2.88 µg/l and 4.94 µg/l, respectively (manufacturer’s data). The recommended discriminator value for the diagnosis of myocardial necrosis by this assay is therefore 5 µg/l.

Standard 12 lead ECGs were obtained at presentation in all patients. Follow up ECGs were obtained daily, with additional ECGs if considered necessary by the physician in charge. ECG changes were categorised as follows: myocardial infarction (new Q waves or ST elevation > 1 mm in two limb leads or > 2 mm in two precordial leads), ischaemia (ST depression > 1 mm in two or more leads, T wave inversion, bundle branch block), other cardiac (for example, left ventricular hypertrophy, arrhythmia), or non-diagnostic/normal.

Diagnosis

Following discharge, the patient’s case notes were examined by a blinded, independent, expert panel comprising a data presenter (JT), a consultant cardiologist (RKM), a cardiology registrar, and a non-cardiologist. Three diagnoses were recorded. First, the “physician diagnosis”, recorded on discharge by the physician in charge of the patient’s care, was taken from the case notes. Second, the expert panel reached a “panel diagnosis” after reviewing the case notes, ECGs, and conventional cardiac markers. The physician and panel diagnoses were categorised as myocardial infarction, unstable angina pectoris (UAP), other cardiac (for example, stable angina, arrhythmia), or non-cardiac. The panel diagnosis of myocardial infarction was made by strict interpretation of the World Health Organization criteria, requiring two of the following three features: typical chest pain of > 30 minutes’ duration, serum conventional cardiac marker of more than twice the upper limit of normal, and definite ECG changes. Third, a “cTnT diagnosis” was made in accordance with the new recommended diagnostic criteria for myocardial infarction, as follows: myocardial infarction was diagnosed if cTnT was > 0.1 µg/l with either typical chest pain, new Q waves, ECG indicating ischaemia (ST elevation or depression) myocardial infarction was excluded if cTnT was < 0.1 µg/l.

The panel also examined whether the patient underwent a complete evaluation with respect to serial measurements over three days, and the influence of alterations in the patient’s ward and attending physician.

Prognosis

Patients were monitored for six months after discharge from the index admission by examination of the case notes in all cases, in conjunction with telephone contact with the patient and the primary care physician if necessary. Follow up was 100% complete. All events were classified by an independent panel according to standard criteria for major adverse cardiovascular events (MACE): cardiac death, non-fatal myocardial infarction, percutaneous transluminal coronary angioplasty, coronary artery bypass grafting, or readmission with UAP.

Statistics

Categorical variables were analysed with χ² Fisher’s exact test and normally distributed continuous variables (age) with Student’s t test. Normality was tested with the Kolmogorov-Smirnov test. To assess the impact of clinical and biochemical markers on MACE-free survival, variables were entered in a Cox binary logistic regression analysis, using a forward regression technique. All analyses were done using SPSS version 10.0, and were two tailed, with p < 0.05 considered significant.

Ethics

The study was approved by the local research and ethics committee, and all subjects gave informed consent.

RESULTS

We enrolled 401 consecutive patients into the study, with a median time for presentation from symptom onset of 193 minutes. CK-MB mass and cTnT samples were drawn at a median time of 750 minutes after symptom onset, consistent with our predetermined protocol for collection after 12 hours. There were important demographic differences between patients admitted under a cardiologist compared with a medical physician (table 1): 235 (58.6%) of the cohort were admitted under a cardiologist compared with a medical physician (table 1): 235 (58.6%) of the cohort were younger, 40% more likely to have diabetes (p = 0.001), but 20% less likely to be hypertensive (p = 0.015).

There were 155 patients (38.7%) who did not have three full sets of conventional cardiac enzymes taken, with 137 (34.2%) having an incomplete CK series and 136 (33.9%) an incomplete AST series. However, 90 (22.4%) of these had two complete sets of conventional markers, showing either a typical pattern of myocardial infarction within these (n = 11) or having two negative sets (n = 79), which is commonly, but sometimes mistakenly, accepted as excluding myocardial infarction. Among the 65 patients (16.2%) with more significant gaps in conventional enzyme sampling, 45 (11.2%) had two samples missing, with the remaining
Patients admitted through medical wards were therefore 90% more likely to have the discharge diagnosis adjusted by the panel (RR 1.9, 95% CI 1.3 to 2.7; p = 0.026).

Table 3 shows the impact of implementing the new diagnostic criteria upon the physician diagnosis. Forty four of 161 myocardial infarction cases (27.3%) diagnosed by the new criteria had been discharged as not myocardial infarction. These 44 cases comprised 33.8% of those discharged as UAP, 15.2% of those discharged as “other cardiac”, and 7.0% of those classified as “non-cardiac”. Additionally, the physician diagnosis of myocardial infarction was rejected in four cases (3.3%) where elevation of conventional markers was from non-cardiac sources. Overall, a significant diagnostic alteration occurred in 48 of 401 patients (12.0%) when applying the new criteria. Further examination of these 48 cases (table 4) shows that in 26 (54.2%) the diagnostic adjustment was because of inaccuracy of the conventional cardiac markers, while in 22 cases (45.8%) the cause may have been preventable. Fourteen (29.2%) of these cases did not have a complete set of conventional markers for analysis, while eight cases (16.7%) were related to inappropriate interpretation of data by the physician in charge.

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### Table 1 Demographics by medical specialty

<table>
<thead>
<tr>
<th></th>
<th>Whole cohort</th>
<th>Cardiology</th>
<th>Non-cardiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>401</td>
<td>235</td>
<td>166</td>
</tr>
<tr>
<td>Male</td>
<td>259 (64.6%)</td>
<td>164 (69.8%)</td>
<td>95 (57.2%)</td>
</tr>
<tr>
<td>Age (median, range)</td>
<td>68 (29 to 99)</td>
<td>66 (29 to 94)</td>
<td>68 (36 to 99)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>362 (90.3%)</td>
<td>213 (90.6%)</td>
<td>149 (89.8%)</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>182 (45.4%)</td>
<td>135 (57.4%)</td>
<td>47 (28.3%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>157 (39.2%)</td>
<td>81 (34.5%)</td>
<td>76 (45.8%)</td>
</tr>
<tr>
<td>Current or ex-smoker</td>
<td>268 (66.8%)</td>
<td>163 (78.0%)</td>
<td>105 (75.0%)</td>
</tr>
<tr>
<td>Cerebrovascular dise</td>
<td>37 (9.2%)</td>
<td>20 (8.5%)</td>
<td>17 (10.2%)</td>
</tr>
<tr>
<td>MI (n = 65)</td>
<td>120 (29.9%)</td>
<td>74 (31.5%)</td>
<td>46 (27.7%)</td>
</tr>
<tr>
<td>Previous revascularisation</td>
<td>64 (16.0%)</td>
<td>43 (18.3%)</td>
<td>21 (12.7%)</td>
</tr>
</tbody>
</table>

### Table 2 Physician diagnosis and retrospective adjustment by expert panel

<table>
<thead>
<tr>
<th>Physician diagnosis</th>
<th>MI (n = 123)</th>
<th>UAP (n = 77)</th>
<th>Other cardiac (n = 64)</th>
<th>Non-cardiac (n = 137)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI (n = 121)</td>
<td>114 (94.2%)</td>
<td>6 (5.0%)</td>
<td>1 (0.8%)</td>
<td>0</td>
</tr>
<tr>
<td>UAP (n = 71)</td>
<td>0</td>
<td>71 (100%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Other cardiac (n = 66)</td>
<td>3 (4.5%)</td>
<td>0</td>
<td>63 (95.5%)</td>
<td>0</td>
</tr>
<tr>
<td>Non-cardiac (n = 143)</td>
<td>6 (4.2%)</td>
<td>0</td>
<td>0</td>
<td>137 (95.8%)</td>
</tr>
</tbody>
</table>

Values are n (%).

### Table 3 Physician diagnosis and reclassification according to the new diagnostic criteria for myocardial infarction

<table>
<thead>
<tr>
<th>Physician diagnosis</th>
<th>cTnT diagnosis</th>
<th>MI (n = 161)</th>
<th>No MI (n = 240)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI (n = 121)</td>
<td>117 (96.7%)</td>
<td>4 (3.3%)</td>
<td></td>
</tr>
<tr>
<td>UAP (n = 71)</td>
<td>24 (33.8%)</td>
<td>47 (66.2%)</td>
<td></td>
</tr>
<tr>
<td>Other cardiac (n = 66)</td>
<td>10 (15.2%)</td>
<td>56 (84.8%)</td>
<td></td>
</tr>
<tr>
<td>Non-cardiac (n = 143)</td>
<td>10 (7.0%)</td>
<td>133 (93.0%)</td>
<td></td>
</tr>
</tbody>
</table>

Values are n (%).

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**Table 1** Demographics by medical specialty

**Table 2** Physician diagnosis and retrospective adjustment by expert panel

**Table 3** Physician diagnosis and reclassification according to the new diagnostic criteria for myocardial infarction

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These 48 cases who underwent diagnostic adjustment by the new criteria were subsequently studied for any correlation between potential causes of diagnostic adjustment with either human error or errors-related to assay sensitivity/specificity. We evaluated two suspected causes of human error that commonly occur—namely, a change in the patient’s ward and an alteration in the physician in charge during the first three days (table 4). Examination of these potentially avoidable factors shows that these occurred with similar frequency where marker sensitivity/specificity was to blame and where there might have been human error. While this does not prove that such factors are not causative, they do not seem to occur more often in those cases with a potentially avoidable reason for diagnostic inaccuracy.

The six month composite end point of any MACE occurred in 61 patients (15.2%). To determine if cTnT provided prognostic information beyond accepted clinical markers of high risk (age, sex, diabetes, ST depression > 1 mm on the ECG, presence of heart failure, and post-myocardial infarction defined by WHO criteria), these variables, together with cTnT, were entered in a Cox multiple logistic regression analysis (table 5). When CK-MB mass of > 5 µg/l alone is entered as the biochemical risk marker (model 1), it is strongly predictive of MACE outcome (p = 0.002), together with ST depression > 1 mm (p = 0.001) and the presence of heart failure (p = 0.018). In contrast, when cTnT is added to the analysis (model 2), CK-MB mass loses this independent predictive value, with cTnT > 0.1 µg/l (p = 0.0004), ST depression > 1 mm (p = 0.003), and the presence of heart failure (p = 0.016) emerging as the independent variables. Thus CK-MB mass appears to track the prognostic ability of cTnT, albeit as a less potent risk stratifier, and is not independently predictive when entered in a model with cTnT. Finally, we evaluated the status of the physician in charge of the patient as a clinical risk marker (model 3). We found that consultant specialty did not independently influence patient prognosis, and had no impact on the other risk variables in this model, although it was noted that cardiologists care for the higher risk cardiac patients (data not shown).

**DISCUSSION**

In an unselected cohort of patients with suspected cardiac chest pain attending a UK hospital, 27.3% of myocardial infarcts (44/161) diagnosed by the new criteria using cTnT are missed by physicians employing the current diagnostic strategies of serial CK/AST and ECGs. The current strategy also resulted in 3.3% of myocardial infarcts (4/121) being incorrectly assigned this diagnosis. Over half these diagnostic alterations (54.2%, 26/48) appeared to be caused by lack of sensitivity/specificity of the conventional markers. However, in 45.8% of cases (22/48), the diagnostic inaccuracy was found to be potentially avoidable and was related to inappropriate interpretation of diagnostic results and failure to complete the current three day sampling strategy. Indeed, 38.7% of the cohort (155/401) did not undergo a full evaluation with serial conventional cardiac markers.

In our cohort, we have found that patients cared for by a cardiologist are consistently more likely to be assessed with a full set of conventional cardiac markers and to have an accurate diagnosis made by the physician responsible for patient care. The cardiology cases where there was a conflict of diagnoses between our panel and the physician were predominantly “grey” cases in which the correct diagnosis was unclear. In contrast, patients admitted through medical wards were more likely to have an incomplete series of conventional markers taken, and 6.6% of cases (11/166) were discharged with an incorrect diagnosis owing to misinterpretation of available data. The underlying causes are unclear, but inadequate levels of staffing, training, or supervision of junior staff may contribute, although we did not evaluate these factors. We suspected that patient relocation between wards and between consultant teams could be responsible, but were unable to demonstrate any clear relation with diagnostic alteration. While we have not tested whether the introduction of the new diagnostic criteria with simpler sampling requirements would have resolved these issues, the adoption of more straightforward and rapid diagnostic protocols may assist clinicians in reaching more certain diagnoses.

Previous studies have shown that in selected cohorts with acute coronary syndromes, the cardiac troponins aid distinction of high and low risk individuals. We found that troponin T provided prognostic information superior to clinical and other biochemical variables in our unselected cohort with suspected cardiac chest pain, and support the earlier observations in other acute coronary syndromes. CK-MB mass provides similar prognostic prediction, but appears to track with cTnT as it offers no independent prognostic information when cTnT is used. The ability of the new markers to distinguish reliably the 3.3% of myocardial infarcts diagnosed by the new criteria was subsequently studied for any correlation between potential causes of diagnostic adjustment with either human error or issues related to assay sensitivity/specificity. We evaluated two suspected causes of human error that commonly occur—namely, a change in the patient’s ward and an alteration in the physician in charge during the first three days. Examination of these potentially avoidable factors shows that these occurred with similar frequency where marker sensitivity/specificity was to blame and where there might have been human error. While this does not prove that such factors are not causative, they do not seem to occur more often in those cases with a potentially avoidable reason for diagnostic inaccuracy.

| Table 4 | Sources of diagnostic adjustment by new criteria for myocardial infarction |
|-----------------------------------------------|
| Potential source of diagnostic adjustment | Total (n = 48) | Ward adjustment days 1–3 | Physician adjustment days 1–3 |
| Incomplete serial cardiac markers | 14 (29.2%) | 4 (28.6%) | 10 (71.4%) |
| Inappropriate physician diagnosis | 8 (16.7%) | 2 (25.0%) | 6 (50.0%) |
| Sensitivity/specificity of conventional markers | 26 (54.2%) | 10 (38.5%) | 16 (69.2%) |

Values are n (%).

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| Table 5 | Multiple logistic regression models for major adverse cardiovascular events |
|---------------------------------------------------------------|
| Risk variable | Model 1 | Model 2 | Model 3 |
| Age | 0.137 | 0.71 | 0.187 | 0.67 | 0.278 | 0.20 |
| Sex | 0.086 | 0.77 | 0.069 | 0.92 | 0.120 | 0.73 |
| Diabetes mellitus | 0.516 | 0.47 | 0.396 | 0.53 | 0.175 | 0.68 |
| WHO MI | 0.008 | 0.93 | 0.189 | 0.66 | 0.131 | 0.72 |
| ST depression >1 mm | -1.088 | 0.001 | -0.983 | 0.003 | 1.077 | 0.003 |
| Heart failure | -0.752 | 0.018 | -0.763 | 0.016 | 0.934 | 0.017 |
| CK-MB >5 µg/l | 0.925 | 0.002 | 0.046 | 0.83 | 0.029 | 0.87 |
| cTnT >0.1 µg/l | -1.119 | 0.0004 | 1.26 | 0.0003 |
| Consultant specialty | 0.141 | 0.71 |

CK-MB, creatine kinase MB isoenzyme; cTnT, cardiac troponin T; MI, myocardial infarction.
infarcts incorrectly diagnosed by CK/AST elevations of non-myocardial origin will help avoid psychological, social, and physical detriment to these patients and their families, and inappropriate use of health care resources.

The new diagnostic criteria for myocardial infarction have an important impact among patients admitted with suspected cardiac chest pain, and consideration should be given to the adoption of these guidelines. The limitations of current diagnostic strategies are not only related to performance of the markers themselves, but also to the time consuming nature of the associated diagnostic protocols, which are subject to more opportunities for human error in data collection. The introduction of simpler diagnostic strategies using more sensitive and specific markers should allow more accurate diagnosis, provide prognostic value beyond that afforded by current risk factors, and may also shorten the hospital stay. Our observed differences in management between general medical and cardiological care support the recommendations of the National Service Framework for assessment of the majority of these patients in a specialist setting.

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Authors’ affiliations

J Trevelyan, R K Mattu, Department of Cardiology, University Hospitals of Coventry and Warwickshire NHS Trust, Coventry, UK
S C H Smith, Department of Clinical Biochemistry, University Hospitals of Coventry and Warwickshire NHS Trust
E W A Needham, Department of Biological Sciences, University of Warwick, Coventry

REFERENCES


From BMJ Journals

Defining clinical features of amiodarone induced optic neuropathy

Amiodarone is known to cause optic neuropathy that can result in visual loss. It may present similarly to that of non-arteritic anterior optic neuropathy (NAION). Three cases are described in an attempt to distinguish the conditions.

All three patients (aged 59–72) presented with mild visual loss and bilateral optic disc swelling. Two had unilateral subjective visual complaints while one was asymptomatic. All had been taking 200mg of amiodarone daily for 3–12 months. Visual acuity improved in all after discontinuing the drug and their dyschromatopsia and visual field defects resolved. In general optic disc swelling lasted for more than six months, a finding which would be unusual in NAION. Swelling lasted up to four months after discontinuing the drug.

Compared with NAION, amiodarone induced neuropathy has an insidious onset, less visual loss, longer duration of disc oedema and is more commonly bilateral. The diagnosis can be confirmed only by observing slow resolution after stopping the intensely cationic amphiphilic medication which accumulates by interaction with polar lipids, allowing it to be deposited as lysosomal inclusion bodies in the optic nerve.

The authors suggest periodic ophthalmic screening could be useful in patients taking amiodarone (who have a risk of optic neuropathy of 1.79% in one series), while visual symptoms should provoke expeditious fundoscopy.

Other ocular side effects previously reported with amiodarone include corneal microdeposits, anterior subcapsular lens opacities, multiple chalazia and dry eye syndrome.

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Supplementary material can be found at:
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