Early diagnosis of acute myocardial infarction by a newly developed rapid immunoturbidimetric assay for myoglobin

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Abstract

Objective—To evaluate a rapid immunoturbidimetric assay for myoglobin and to investigate its clinical usefulness in the early detection of acute myocardial infarction.

Design—Prospective study. Immunoturbidimetrically determined myoglobin concentrations were compared with radioimmunoassay results obtained with the same blood samples. The diagnostic performance of myoglobin determination was compared with creatine kinase and creatine kinase MB activity (current standard of routine diagnosis).


Patients—Part 1: 30 patients with acute myocardial infarction admitted not later than four hours (median two hours) after the onset of symptoms. Part 2: 126 patients admitted to the emergency room with chest pain not caused by trauma (51 cases of acute myocardial infarction, 51 cases of angina pectoris, and 24 cases of chest pain not related to coronary artery disease).


Main outcome measures—The analytical quality of the immunoturbidimetric myoglobin assay and a comparison between the myoglobin assay and creatine kinase and creatine kinase MB for diagnostic sensitivity and performance.

Results—The immunoturbidimetric myoglobin assay was fast and convenient and gave myoglobin determinations of high analytical quality. The concentration of myoglobin increased, peaked, and returned to the reference range significantly earlier than creatine kinase (p ≤ 0.0001) and creatine kinase MB (p ≤ 0.0002). Before thrombolytic therapy was started, the diagnostic sensitivity of myoglobin was significantly higher than that of creatine kinase MB activity 0–6 h after the onset of chest pain and significantly higher (0.82 v 0.29) than creatine kinase 2–4 h after the onset of chest pain. In almost all patients (92%) plasma myoglobin concentrations were increased 4–6 h after the onset of chest pain.

Conclusion—Myoglobin was more sensitive in detecting early myocardial infarction than creatine kinase and creatine kinase MB activity. Immunoturbidimetric myoglobin measurements could be useful in the early evaluation of patients with suspected myocardial infarction because this assay takes less than two minutes.
non-invasive predictor of the outcome of therapeutic reperfusion, it has not been widely used in the early evaluation of patients with suspected acute myocardial infarction.

We have evaluated a rapid, quantitative immunoturbidimetric myoglobin assay in the early detection of acute myocardial infarction.

**Patients and methods**

**PATIENTS**

The study was carried out in two parts.

**Part 1**

This part of our study was performed to confirm the differences in the time courses of myoglobin, creatine kinase MB, and total creatine kinase activity in plasma after acute myocardial infarction and to establish a correlation between myoglobin concentrations measured by radioimmunoassay and by immunoturbidimetry. Thirty consecutive patients with subsequently confirmed myocardial infarction (22 men and eight women) aged 40–83 years (median 61 years) presented to the hospital coronary care unit within four hours after the onset of symptoms and gave informed consent for extra blood samples to be drawn. Patients presenting to the coronary care unit more than four hours after the onset of symptoms were excluded, because at this stage of acute myocardial infarction there is a high probability of increased creatine kinase and creatine kinase MB activity and an increased myoglobin plasma concentration. The median time to admission was two hours (range 30–240 minutes). All patients subsequently proved to have sustained an acute myocardial infarction (27 Q wave and three non-Q wave myocardial infarctions: 12 anterior and 18 inferior wall infarctions). Myocardial infarction was diagnosed independently by two cardiologists according to the World Health Organisation criteria based on the patient’s clinical history and symptoms, electrocardiographic abnormalities, and serum enzyme findings and without knowledge of the myoglobin results. Treatment was determined by the clinical circumstances. Fifteen patients were given intravenous streptokinase (1.5 million units in 60 minutes), two patients were given intravenous urokinase (2 million units in 10 to 15 minutes), and 11 patients were given intravenous alteplase (initial bolus of 10 mg, followed by 50 mg during the first hour and 20 mg each during the second and third hour after admission). Two patients were not given thrombolytic treatment because of contraindications. All patients additionally received routine coronary care and were treated with intravenous heparin, aspirin, nitrates, occasionally β blockers, and antiarrhythmic drugs as needed. Periheral venous blood samples were collected from an indwelling forearm catheter or by venepuncture before treatment in the coronary care unit. Samples were collected hourly for the first 10 hours, then at 12, 16, 20, 24, 32, 40, 48 hours after admission, and thereafter daily until biochemical markers returned to normal. Patient care requirements occasionally prevented a sample being taken.

**Part 2**

In the second part of the study we evaluated the immunoturbidimetric myoglobin determination as a rapid screening assay for myocardial infarction in patients with chest pain presenting to the emergency room. One hundred and twenty-six consecutive patients in the hospital’s department of internal medicine with chest pain as their major symptom were investigated. The study population did not include patients with chest contusion or injured patients. Fifty one patients subsequently proved to have sustained an acute myocardial infarction (median delay from the onset of chest pain to admission 2.25 h, range 0.5–14.5 h), which was diagnosed by two cardiologists according to the WHO criteria given earlier. Twenty six patients sustained a Q wave acute myocardial infarction (median delay 1.75 h, interquartile range 1–3.5 h) and 25 a non-Q wave acute myocardial infarction (median delay 4 h, interquartile range 1–7.15 hours). Fifty one patients presented with angina pectoris. A further 24 patients had chest pain not related to coronary artery disease (three cases of supraventricular tachycardia, four pneumonia with pleurisy, one obstructive pulmonary disease, four musculoskeletal disorders, three pericarditis, six pulmonary embolism, two oesophagitis, and one case of hypertensive crisis). In these patients a single blood sample was drawn immediately after presentation to the emergency room. All patients received routine emergency treatment. All patients with acute myocardial infarction were transferred to the coronary care unit before the start of thrombolytic therapy. None of these patients with acute myocardial infarction had been given thrombolytic agents before admission to the hospital.

**Laboratory analysis**

**Blood collection**

Blood was collected in tubes coated with EDTA. Creatine kinase and creatine kinase MB activity was measured immediately after collection. Blood samples for the measurement of myoglobin concentrations were centrifuged immediately and the plasma subsequently
frozen and stored at \(-20^\circ\text{C}\) until determination. Under these conditions myoglobin could be assayed within at least four weeks (maximum period of storage) after collection without any decline in concentration.

**Creatine kinase and creatine kinase MB activity**
Total creatine kinase and creatine kinase MB activities were measured (25°C) with N-acetyl-cysteine activated, optimised ultraviolet test kits obtained from Merck (Darmstadt, Germany). Creatine kinase MB activity was measured by means of immunoinhibition based on the presence of inhibiting creatine kinase M antibodies. According to the manufacturer's recommendations, a total creatine kinase activity of 70 U/l for women and 80 U/l for men was used as the upper limit of the reference interval. For creatine kinase MB activity the reference range goes up to 10 U/l. Creatine kinase MB activities of >10 U/l and an increase of >6% in total creatine kinase activity were assumed to indicate myocardial muscle cell damage.

**Myoglobin**
Myoglobin radioimmunoassay—Myoglobin was determined by a commercially available radioimmunoassay (Byk-Sangtec, Dietzenbach, Germany). The manufacturer's upper limit of the reference interval is 80 μg/l.

**Immuno-turbidimetric myoglobin assay**—A myoglobin assay (Turbiquant myoglobin, Behringwerke AG, Marburg, Germany) for use with the Behring Turbitimer analyser was used for rapid immuno-turbidimetric determination of myoglobin concentrations in plasma. This assay is based on polystyrene particles coated with rabbit anti-human myoglobin antibodies. In an immunochemical reaction these particles form agglutinates with the myoglobin contained in serum or plasma. The increase in turbidity is measured photometrically. Quantitative results are available about a minute after the start of the assay. The detection limit of the assay is 50 μg/l and the measurement range 50–650 μg/l. The concentrations of myoglobin in EDTA plasma from 100 apparently healthy subjects (blood donors with no history of cardiovascular diseases) (41 men and 59 women aged 19–65 (median 44 years)) were measured by this new method to obtain a reference interval calculated by non-parametric determination of percentiles.

**DATA ANALYSIS**
Median, interquartile range, and percentiles were calculated to describe continuous variables. The association between continuous variables was analysed by the Spearman rank correlation test. The Wilcoxon signed rank test and \(x^2\) test (or Fisher's exact test where appropriate) were used for between group comparisons. Confidence intervals were calculated according to the method of Gardner and Altman. A \(p\) value of \(<0.05\) was regarded as significant. Bonferroni adjustment was used for comparison of more than two groups. Sensitivity, specificity, efficiency, positive and negative predictive values, likelihood ratio, and Youden index (sensitivity + specificity - 1) were calculated to describe the performance of myoglobin, creatine kinase, and creatine kinase MB activity in the early diagnosis of myocardial infarction in patients in the emergency room.

**Results**
**IMMUNOTURBIDIMETRIC ASSAY FOR MYOLOBIN**
Reference interval of myoglobin concentrations in plasma by the immuno-turbidimetric assay
Figure 1 shows the distribution of myoglobin concentrations in 100 healthy individuals. In most the myoglobin concentration was below the detection limit of the assay. The upper limit of the reference interval (cut off value), calculated as the 97.5% percentile, was 70 μg/l. The age or sex of the reference subjects did not influence the myoglobin concentrations. The correlation coefficient between myoglobin concentrations and age was 0.08. There was no significant (\(p = 0.27\)) difference between myoglobin concentrations in men and women.

**Correlation with radioimmunoassay method**
Myoglobin was simultaneously determined by both methods in serial blood samples taken from 30 patients with acute myocardial infarction. There was a good correlation between the immunoturbidimetric assay and radioimmunoassay for myoglobin time courses of individual patients with myocardial infarction. The median correlation coefficient was 0.96 (interquartile range 0.91–0.97). However, values obtained by the immunoturbidimetric assay were usually lower than those obtained by radioimmunoassay.

**Interference**
Rheumatoid factors (tested for a concentrations up to 1260 IU/ml) did not interfere with the assay. The myoglobin assay is usually not disturbed by icteric, haemolysic, and lipaemic samples. However, if the absorbance of the sample is too high before the reaction starts (for example, because of strong absorption caused by haemolysis or hypertriglyceridaemia), the analyser generates a warning message and the assay cannot be done. Such samples can be...
Immunoturbidimetric measurement of myoglobin

retested after manual predilution. Hypertriglyceridaemic samples can be also clarified by centrifugation (10 min at approximately 15 000 g) and retested.

Reproducibility
The calculation of the coefficients of variation for patients' and control samples was based on 20 measurements each. The intrassay coefficients of variation ranged from 2.2% to 6.8% and the interassay coefficients of variation from 5.4% to 11.7%. The highest coefficients of variation were found for samples with low myoglobin concentrations.

TIME COURSES OF MYOGLOBIN, CREATINE KINASE, AND CREATINE KINASE MB ACTIVITY IN MYOCARDIAL INFARCTION
Figure 2 summarises the time courses of myoglobin concentrations of 30 patients with myocardial infarction. Thrombolytic therapy resulted in early reperfusion in 18 of 25 patients with Q wave infarction. The time courses of myoglobin, creatine kinase, and creatine kinase MB activities in patients with non-Q wave myocardial infarctions resembled those of patients with Q wave myocardial infarctions with early reperfusion. Table 1 shows the criteria describing the release kinetics of myoglobin, creatine kinase, and creatine kinase MB activities. An abnormal increase, the peak concentration, and return into the reference range occurred significantly earlier for myoglobin than for creatine kinase (p ≤ 0.0001) and creatine kinase MB activity (p ≤ 0.0002). The median difference between the first appearances of myoglobin and creatine kinase and the first appearances of myoglobin and creatine kinase MB was one hour each (95% CI 1-2 h). In these patients the diagnostic sensitivity of myoglobin was significantly (p < 0.05) higher than that of both creatine kinase and creatine kinase MB activity at 3-5 and four hours after the onset of symptoms (fig 3). The diagnostic sensitivity of myoglobin was 0.5 (50%) 3-4 hours after the onset of symptoms—a value that creatine kinase activity only reached after 4-8 hours and creatine kinase MB after five hours respectively (table 1). All patients had increased myoglobin concentrations (100% sensitivity) at six hours and increased creatine kinase and creatine kinase MB activities at 12 hours after the onset of chest pain (fig 3). The median peak values of myoglobin, creatine kinase, and creatine kinase MB were attained early after admission because of the high percentage (72%) of patients with Q wave myocardial infarction who achieved early reperfusion. The magnitude of increase (calculated as the peak value divided by the cut off value) of myoglobin was significantly (table 1) greater than for creatine kinase activity (p = 0.0007) and creatine kinase MB activity (0.0001). The period when plasma concentrations of myoglobin were increased after acute myocardial infarction that allowed a diagnosis based on increased biochemical markers was significantly shorter than that of creatine kinase (p = 0.0001) and creatine kinase MB activity (p = 0.0002). The correlation coefficient between peak values of myoglobin and creatine kinase was 0.69 (p = 0.0002) and between myoglobin and creatine kinase MB it was 0.68 (p = 0.0002).

DIAGNOSTIC PERFORMANCE OF MYOGLOBIN, CREATINE KINASE, AND CREATINE KINASE MB ACTIVITY WHEN USED TO DETECT ACUTE MYOCARDIAL INFARCTION IN EMERGENCY ROOM PATIENTS WITH CHEST PAIN
In this group the prevalence of myocardial infarction was 0.4 (pretest odds 0.68). The median creatine kinase activity in patients with acute myocardial infarction (n = 51) was 68 U/l (interquartile range 47-142 U/l), the median creatine kinase MB activity was 10 U/l (interquartile range 4-12 U/l). In these patients myoglobin concentrations ranged from 0.0001 to 2990 µg/l. Eighteen patients with acute myocardial infarction had myoglobin concentrations below the detection limit (50 µg/l) of the immunoturbidimetric assay. The median creatine kinase activity in patients with angina pectoris was 37 U/l (interquartile range 26-68 U/l), the median creatine kinase MB activity was 4 U/l (interquartile range 4-6 U/l). In this group (n = 51) myoglobin concentrations ranged from <50 µg/l to 72.6 µg/l and were <50 µg/l in 43 cases. In patients with chest

Table 1 Release kinetics of myoglobin, creatine kinase (CK), and creatine kinase MB activity in 30 patients with acute myocardial infarction (median 95% CI)

<table>
<thead>
<tr>
<th>Cut off value</th>
<th>CK MB activity</th>
<th>CK activity</th>
<th>Myoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to first increased plasma values (h)</td>
<td>11.3 (11-14.3)</td>
<td>13 (11-15.8)</td>
<td>78 (5-7.5)</td>
</tr>
<tr>
<td>Time to peak (h)</td>
<td>53 (3-76)</td>
<td>78 (50-96)</td>
<td>215 (17-260)</td>
</tr>
<tr>
<td>Return to normal (h)</td>
<td>50 (32-71)</td>
<td>68 (46-76)</td>
<td>17 (12-23-0)</td>
</tr>
<tr>
<td>Period of increased plasma values (h)</td>
<td>43 (3.5-7.7)</td>
<td>7 (4.3-12)</td>
<td>9.5 (5.5-23.6)</td>
</tr>
<tr>
<td>Magnitude of increase*</td>
<td>0.69</td>
<td>68</td>
<td>2990</td>
</tr>
</tbody>
</table>

The onset of symptoms (chest pain) was the reference point for the calculation of first appearance in blood, time to peak value, and time of return to normal.

Peak value divided by cut off value. Twenty five patients with Q wave acute myocardial infarctions were given intravenous thrombolytic treatment, which resulted in early reperfusion in 18.
patients shown in fig 3. Symbols represent the number of increased values (%) including the lowest value of each time classification (30 minute intervals).

Owing to this distribution of delay from the onset of pain to admission only the difference between the sensitivities of myoglobin and creatine kinase MB was statistically significant when all patients were included in the calculation of the sensitivity. Subgroup analysis of the sensitivities of myoglobin and creatine kinase in patients admitted during the 2–4 hour period (limits included), however, also showed a significantly (p = 0.016) higher sensitivity for myoglobin compared with creatine kinase (0.76 v 0.29). Table 3 shows the false positive diagnoses of acute myocardial infarction for creatine kinase, creatine kinase MB activity, or the immunoturbidimetric myoglobin assay. The specificity, positive predictive value, and likelihood ratio of myoglobin were significantly higher than for creatine kinase. The sensitivity and Youden index for myoglobin were significantly higher than for creatine kinase MB activity (table 2).

Fifteen patients with myocardial infarction presented with a non-diagnostic electrocardiogram to the emergency ward within six hours (median two hours) of the onset of chest pain. In these potential candidates for fibrinolytic therapy the sensitivity of myoglobin (0.6, 95% CI 0.35 to 0.85) was significantly higher than that of creatine kinase (0.33, 95% CI 0.09 to 0.57) and creatine kinase MB activity (0.13, 95% CI interval 0 to 3).

**MYOglobin, creatINE kinase, AND CREATINE KINASE MB ACTIVITY SENSITIVITIES BEFORE THE START OF THROMBolytic THERApY**

Table 4 shows the sensitivities of myoglobin, creatine kinase, and creatine kinase MB activity during the early stages of acute myocardial infarction before the start of thrombolytic treatment. This table is based on the combined analysis of the data from emergency room patients and from the blood samples taken from patients with myocardial infarction in part 1 of our study at admission to the coronary care unit before thrombolytic treatment. Patients who had had cardiopulmonary resuscitation or defibrillation therapy were also excluded from data analysis. Myoglobin was significantly more sensitive than creatine kinase MB during the 0–6 hour period and significantly more sensitive than creatine kinase activity during the 2–4 hour period after the onset of chest pain. Almost all patients (92%) had increased plasma myoglobin concentrations during the 4–6 hour period.

**Discussion**

The immunoturbidimetric myoglobin assay allows fast and convenient myoglobin determinations of high analytical quality. Intra and inter assay coefficients of variation are within acceptable limits. Interference by rheumatoid factors, haemoglobin, bilirubin, and triglycerides was negligible under routine conditions. The correlation between immunoturbidimetric results and radioimmunoassay myoglobin results was good. The results of immunoturbidimetric myoglobin assay are available within 1–2 minutes of the start of the assay. These properties make this method...
The prevalence of acute myocardial infarction was 0.4 (pretect odds 0.68). The delay of myocardial infarction patients from the onset of symptoms to admission ranged from 0.5 to 14.5 hours (median 2.25 hours). 95% confidence intervals are given in parentheses.

### Table 3 Patients with false positive myoglobin, creatine kinase (CK), or creatine kinase MB test results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>CK activity</th>
<th>Myoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Angina pectoris</td>
<td>87</td>
<td>65</td>
</tr>
<tr>
<td>2</td>
<td>Angina pectoris</td>
<td>201</td>
<td>&lt;50</td>
</tr>
<tr>
<td>3</td>
<td>Musculoskeletal disorder</td>
<td>77</td>
<td>&lt;50</td>
</tr>
<tr>
<td>4</td>
<td>Pulmonary embolism</td>
<td>108</td>
<td>&lt;50</td>
</tr>
<tr>
<td>5</td>
<td>Angina pectoris</td>
<td>94</td>
<td>&lt;50</td>
</tr>
<tr>
<td>6</td>
<td>DC countershock therapy</td>
<td>123</td>
<td>244</td>
</tr>
<tr>
<td>7</td>
<td>CK increase, unknown origin</td>
<td>136</td>
<td>&lt;50</td>
</tr>
<tr>
<td>8</td>
<td>Angina pectoris</td>
<td>110</td>
<td>&lt;50</td>
</tr>
<tr>
<td>9</td>
<td>Angina pectoris</td>
<td>170</td>
<td>542</td>
</tr>
<tr>
<td>10</td>
<td>Pericarditis</td>
<td>146</td>
<td>&lt;50</td>
</tr>
<tr>
<td>11</td>
<td>Angina pectoris</td>
<td>97</td>
<td>&lt;50</td>
</tr>
<tr>
<td>12</td>
<td>Pericarditis</td>
<td>114</td>
<td>62.8</td>
</tr>
<tr>
<td>13</td>
<td>Seizure</td>
<td>46</td>
<td>162</td>
</tr>
<tr>
<td>14</td>
<td>Angina pectoris</td>
<td>114</td>
<td>72.6</td>
</tr>
<tr>
<td>15</td>
<td>Angina pectoris</td>
<td>123</td>
<td>56.2</td>
</tr>
<tr>
<td>16</td>
<td>Gastritis</td>
<td>87</td>
<td>&lt;50</td>
</tr>
</tbody>
</table>

### Table 4 A comparison of the sensitivity of myoglobin and creatine kinase (CK), and creatine kinase MB in the early stages of myocardial infarction (before thrombotic therapy)

<table>
<thead>
<tr>
<th>Delay between onset of symptoms and blood sampling</th>
<th>Myoglobin</th>
<th>CK</th>
<th>CK MB activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2 h</td>
<td>0.23</td>
<td>0.13</td>
<td>0.05</td>
</tr>
<tr>
<td>2 and ≤4 h</td>
<td>0.82</td>
<td>0.29</td>
<td>0.23</td>
</tr>
<tr>
<td>&gt;4 and ≤6 h</td>
<td>0.75</td>
<td>0.33</td>
<td>(0.77 to 1.0)</td>
</tr>
</tbody>
</table>

Calculations of sensitivities are based on cut off values: 70 µg/l (myoglobin), 70 U/l (creatinine kinase activity), and 10 U/l (creatine kinase MB activity). 95% confidence intervals are given in parentheses. Blood samples obtained from patients after cardiopulmonary resuscitation, defibrillation, or thrombolytic therapy were excluded from data analysis.
a better indicator of further tissue necrosis than
creatine kinase and creatine kinase MB.
Second, myoglobin lacks specificity for cardiac
muscle. When the test result is positive the
clinical setting must be taken into account.
In the absence of concomitant damage to skeletal
muscle or severely impaired renal function a
positive myoglobin test result predicts myocardial
infarction with a very high probability.
The specificities of myoglobin and creatine kinase
activity in patients with chest pain were
surprisingly high because we studied patients
in the emergency room after injured patients
had preselected for treatment elsewhere.
The specificity of myoglobin and creatine kinase
activity in the detection of myocardial
infarction will be lower in a population that includes
cases of acute or chronic skeletal muscle
damage caused by injury, burns, surgery, etc.
A negative myoglobin assay result in the 4–12 h
period after the onset of the infarct related
symptoms on the other hand allows acute
myocardial infarction to be ruled out within a
few minutes with a very high probability.
Finally, immuno turbidimetric myoglobin
measurments do contribute to the diagnosis of
acute myocardial infarction in the subgroup of
infarct patients presenting early with non-
diagnostic electrocardiograms.

We thank Dr Gilbert Reibnegger for statistical advice and Dr
Martin Höninger for providing plasma samples from blood
donors.

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