Strategic Utilization of Cardiac Markers for the Diagnosis of Acute Myocardial Infarction*

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ABSTRACT

Current changes of the healthcare environment have promoted the creation of chest pain centers in the emergency departments for rapid triage of patients admitted for cardiac evaluation. Because of the inefficiency of electrocardiogram for the diagnosis of acute myocardial infarction, blood cardiac markers play an important role in the decision making process. Current commercial cardiac tests available include creatine kinase, its MB isoenzyme, MB isoforms, lactate dehydrogenase and its isoenzyme-1, myoglobin, cardiac troponin T and troponin I. The diagnostic efficacy of each of these assays is reviewed. Their appropriate use depends on when the specimens are collected for testing after the onset of myocardial infarction. Since not all patients seek medical attention at the time symptoms appear, the applicability of these markers differs in each case. Based on the time after the onset of chest pain, a utilization strategy of the cardiac markers is proposed. With this protocol, the triage of patients can be optimized resulting in the efficient treatment of patients and large savings in cost.

Introduction

Ischemic heart disease continues to be the leading cause of death in the United States.1 The timely diagnosis of an acute myocardial infarction (AMI) has been of paramount importance to the survival of the patient. With the advent of thrombolytic therapy, early diagnosis of AMI becomes an essential prerequisite for the effectiveness of these drugs.2,3,4 In addition to this medical emergency, the present healthcare environment has forced physicians to shorten the hospital stay of patients. Since only about 30 percent of admissions to a Critical Care Unit actually develop an AMI, chest pain centers have been established in emergency departments solely for the triage of patients admitted for the evaluation of the heart as soon as within 24 h.1,2,3,4,5,6,7 These centers generally have established protocols for the management of patients. However, the clinical laboratory plays a critical role to aid these physicians in the diagnosis of AMI.

According to the recommendation of the World Health Organization,7 the diagnosis of an AMI requires that the
patient presents at least two of the following three criteria: (1) presence of characteristic chest pain; (2) diagnostic electrocardiogram (ECG) changes; and (3) positive blood cardiac marker. Almost all of the patients seeking cardiac evaluation experience some form of chest pain. However, the admission ECG of these patients provides only about 75 percent accuracy in the diagnosis of AMI. In small Q-wave and non-Q-wave infarctions, the sensitivity and specificity of the ECG is only 70 to 80 percent. In over 20 percent of all AMIs, the ECG is indeterminate. In general, the ECG is not of much use in detecting micro infarctions, such as those that may occur in patients with unstable angina pectoris. Because of the lack of diagnostic efficiency of ECG, serum cardiac markers become clinically important diagnostic tools. Although there are numerous markers available, many are either outdated or under development and investigation, such as myosin heavy chain, myosin light chain I and glycogen phosphorylase BB. The tests that are approved by the United States Food and Drug Administration and available commercially are: creatine kinase (CK), creatine kinase isoenzyme MB (CK-MB), MB isoforms, lactate dehydrogenase (LD), lactate dehydrogenase isoenzyme-1 (LD1), cardiac troponin T (cTnT) and I (cTnI), rapid bedside cTnT, myoglobin, and spot CK-MB and myoglobin. The characteristics of these tests and their diagnostic efficacy are described.

MYOGLOBIN

Myoglobin is a low-molecular-weight (17.8 KD), cytoplasmic, heme protein, which binds oxygen in both the cardiac and skeletal muscles. It is cleared by the kidney with a half life of about 3 h. The serum baseline level varies with physical activity and muscle mass and is in the range of 30 to 90 ng/mL. Concentrations greater than 100 ng/mL are considered elevated. As it is a small molecule, it may be leaked into the serum within 1 h after myocardial cell death, with peak levels reached in 5 to 12 h and returned to normal levels generally within 16 to 36 h. In patients with AMI, the serum myoglobin level may be elevated by a factor of 5 to 20, with an initial increase rate of greater than 50 percent every 2 h. Like other markers, the sensitivity and specificity for myoglobin depend on when the test is performed. At 4 h post-AMI, the sensitivity and specificity reach 100 percent and 76 percent, respectively, with a predicative value of 75 percent. By 6 h, the sensitivity and specificity reach 90 percent and 100 percent, respectively. Woo et al. reported a sensitivity of 90 percent and specificity of 74 percent using release rate of 20 ng/mL/hr as criteria. For the diagnosis of AMI, myoglobin should be tested serially. If a repeated myoglobin level doubles within 1 to 2 h after initial value, it is highly specific for AMI. The serial testing will also alleviate the false elevation of myoglobin owing to renal diseases, since it is cleared by the kidneys.

Although myoglobin appears to be more sensitive than CK-MB for early diagnosis of AMI, it is not specific to the cardiac muscle and can be released as a result of skeletal muscle damage. Carbonic anhydrase isoenzyme III (CA III) has been suggested to remedy this deficiency. The CA III is found to be present in the skeletal muscle but not in the cardiac muscle. Thus, in skeletal muscle damage, both CA III and myoglobin will be increased. On the other hand, only myoglobin will be elevated in AMI. By measuring the ratio of myoglobin to CA III, the source of myoglobin can be distinguished. Only in AMI will the ratio increase. Unfortunately, CA III assay is not currently available commer-
cally. Thus, the interpretation of myoglobin results for AMI should take into account all the possible false positives.

Myoglobin can be quantitated by immunoassays and requires less than 20 minutes; thus, stat testing is possible. A qualitative spot test is also available which takes only 15 minutes and can be performed at the point-of-care.

**Creatine Kinase, Its Isoenzymes and Isoforms**

Of the three cytosolic isoenzymes of CK, only CK-MB has been shown to be more specific for the myocardium, which contains up to 50 percent of the total CK activity as CK-MB, compared to less than 2 percent in the skeletal muscle. Determinations of CK-MB are generally regarded as the reference standard of diagnostic tests for AMI. Elevated CK-MB levels can be found within 4 h after infarction, with peak levels occurring at 9 to 30 h and returning to normal between 48 to 72 h. The sensitivity and specificity of a single-test result in patients admitted in emergency departments vary considerably based on the time after the onset of symptoms, with a range of sensitivity from 17 to 63 percent at 0 h and 92 to 100 percent at 3 h after presentation. The specificity at 3 h after admission is 92.6 percent. After AMI, MB2 is rapidly released from the damaged heart tissue. This causes a dramatic increase in the ratio of MB2 to MB1 while the total CK-MB level remains within reference range. The MB2 activity and the MB2/MB1 ratio increase within 2 h after the onset of chest pain. A ratio of greater than 1.5 is indicative of myocardial cell damage. The ratio peaks by 4 to 6 h. Reperfusion causes even earlier peaking, from 0.75 to 2.25 h. In general, more than half of AMI patients show a MB2/MB1 ratio $\geq 1.7$ and MB2 $\geq 2.6$ U/L within 2 to 4 h from onset of pain. Within 4 to 6 h, 93 percent of AMI patients show a MB2/MB1 ratio above 1.7 and MB2 greater than 2.6 U/L. Thus, the determination of the MB2/MB1 ratio is a very important determinant of early myocardial infarction. The percent of CK-MB should also be used as a criterion since skeletal muscle damage may result in an elevated MB2/MB1, but the percent of CK-MB will be less than 4. The sensitivity and specificity of MB isoforms at 4 h post-AMI is 92 to 94 percent and 94 to 100 percent, respectively, with a positive
predictive value of 95 percent.\textsuperscript{9,35} Measurement of MB isoforms is available commercially by electrophoresis. An automated electrophoresis system can perform the test within 20 minutes.\textsuperscript{26,35}

**Lactate Dehydrogenase and Its Isoenzymes**

Lactate dehydrogenase (LD) is present in numerous organ systems, and many different conditions can cause an increase in total LD.\textsuperscript{39} It has five isoenzymes, LD1 to LD5. Serum LD1 exceeds LD2 in the myocardium and erythrocytes.\textsuperscript{40} Serum LD2 is normally higher than LD1; however, in cases of AMI, there is a reversal in the LD1/LD2 ratio which is referred to as the LD “flip.” Increased LD1 and the so-called “flip” have sensitivities and specificities of about 75 to 90 percent in patients suspected of AMI.\textsuperscript{31,39} The serial LD1 levels are believed to be more specific for AMI than the LD “flip,” with the most sensitive and specific indicator being the LD1/total LD ratio. Because of its high molecular weight (135 KD), LD is slowly released into the blood. Elevations of serum LD occur 8 to 18 h after the onset of symptoms, peak at 24 to 72 h, and return to normal after 6 to 10 days.\textsuperscript{39,40}

Thus, LD1/total LD ratio is best used as a confirmatory test for AMI. The optimum interval for analysis of LD isoenzymes is the 24 to 48 h period after the onset of chest pain.\textsuperscript{28,39} Since LD1 is present in high concentrations in the erythrocytes, hemolysis is a major cause of false positives. Both LD1 and total LD can be assayed enzymatically providing results in less than 10 minutes.\textsuperscript{39}

**Troponin T and I**

The regulatory troponin complex of the myofibril is composed of TnT, TnI, and troponin C. Cardiac troponin T is the tropomyosin-binding subunit located on the thin myofilament of the contractile apparatus. Although most of the troponin T is bound, about 3 to 5 percent are present in the cytosol.\textsuperscript{41,42} Cardiac troponin I regulates actomyosin adenosine triphosphase (ATPase) and is a smaller protein (22.5 KD vs. 39.7 KD). Cardiac specific isoforms of both TnT and TnI have been identified which can be differentiated immunologically.\textsuperscript{42,43,44,45,46,47,48}

While cTnI has a similar degree of sequence heterogeneity when compared with skeletal muscle isoforms, it has an additional 26 amino acid residue at the N-terminus allowing for the development of antibodies.\textsuperscript{45,46,47}

Cardiac TnT is elevated in AMI patients within 3.5 h of onset of chest pain, slightly earlier than CK, and remains elevated for more than 14 days.\textsuperscript{22,42,48,49,50} There is a plateau from the second to the fifth day.\textsuperscript{50} In early reperfusion, cTnT reaches a peak within 24 h after admission. The kinetics of cTnT released into the circulation are similar for patients with Q wave and non-Q wave AMI.\textsuperscript{44,49,50} Cardiac TnT shows an average of 30 to 40 fold increase above the upper limit of the reference interval, significantly greater than the relative increases of other markers.\textsuperscript{44} The clinical sensitivity of cTnT measurements reaches 100 percent within 4 to 10 h and remains at a high level until day five or six.\textsuperscript{48,49,50,51,52,53,54} Although more sensitive, cTnT seems to have a lower specificity than CK-MB for myocardial damage, although it has been reported to be diagnostically similar.\textsuperscript{22,52,53,54} At 4 to 12 h after AMI, it has a specificity of 74 percent.\textsuperscript{55} The diagnostic efficiency is about 98 percent. Ninety-nine percent of healthy subjects have serum troponin T concentrations below 0.06 µg/L.\textsuperscript{49}

Troponin T has not been proven to be an early marker for acute myocardial infarction. Within 0 to 2 h after onset of
chest pain, cTnT has only a sensitivity of 33 percent.\textsuperscript{56} Only 50 percent of AMI patients have increased TnT concentrations at 4 h.\textsuperscript{50}

Troponin T may be more accurate in quantifying infarct size.\textsuperscript{42,57} Comparison of early and late rises in troponin T concentrations following AMI may provide valuable information about the infarct size, reperfusion, and response to thrombolytic therapy.

Increased troponin T in serum can detect a subgroup of ischemic heart disease patients in whom AMI has been ruled out, such as those with unstable angina.\textsuperscript{44,53,58} Studies indicate that about 70 percent of patients with unstable angina had cTnT concentrations between 0.2 to 6.9 µg/L.\textsuperscript{58} This is an important aspect since it is known that 10 to 20 percent of patients with unstable angina have a poor prognosis, with progression to AMI or cardiac death within the first year.\textsuperscript{59,60} It is, therefore, important to stratify those patients with a small but significant increase of cTnT who have no traditional evidence of AMI.\textsuperscript{61}

Cardiac TnT may be a useful marker for cardiac transplantation rejection,\textsuperscript{62} but its cardiospecificity is uncertain in patients with chronic renal disease, chronic muscle disease, and noncardiac trauma.\textsuperscript{63,64,65,66} These false positives possibly could be due to cross-reactivity of the assay with skeletal TnT or skeletal muscle expressed cTnT. For renal patients, it is possible that the patients may have minor myocardial damage.\textsuperscript{67} Cardiac TnT is also elevated in confirmed myocarditis,\textsuperscript{44} patients with perimyocarditis,\textsuperscript{50} and heart contusions in blunt heart trauma.\textsuperscript{50,54,68} Because of these complications, it may be difficult to discriminate with certainty those patients with an acute coronary syndrome who are experiencing unstable angina without myocardial necrosis from those with focal non-Q wave MI.\textsuperscript{39,53}

Cardiac TnT has also been reported to be more sensitive and specific than CK-MB for the detection of perioperative non-Q-wave infarctions.\textsuperscript{69,70} The application in this area is important as the incidence of MI in patients undergoing noncardiac surgery varies widely from 1 to 26 percent.\textsuperscript{71} There is no accepted standard criterion for the diagnosis of small, non-Q-wave infarctions in patients undergoing aortocoronary bypass surgery.

Currently, cTnT is quantitated by a sandwich immunoassay which takes about 90 minutes. Because of the long assay time, use of cTnT for stat diagnosis of AMI is impractical and cost prohibitive. However, new format of the test is forthcoming which will shorten the assay time to less than 20 mins. In addition a rapid qualitative cTnT spot assay is available for point-of-care testing. With a detection limit between 0.1 and 0.2 µg/L, results are obtainable in 3 to 20 minutes depending on the cTnT concentration in the sample.\textsuperscript{51,56,72} The rapid cTnT test has a sensitivity of 33 percent at 0 to 2 h from onset of chest pain and increases to 75 to 100 percent after 4 h. It has a specificity of 86 to 100 percent.\textsuperscript{51,56} Thus, rapid cTnT assay is as efficient as the quantitative assay but provides a simple and faster test for point-of-care evaluation of patients with chest pain.\textsuperscript{51}

Previous clinical data on cTnI suggest that all benefits for cTnT are also valid for cTnI. However, cTnI is absolutely cardiospecific.\textsuperscript{73} There is no increase in cTnI in patients with acute or chronic skeletal muscle disease, trauma, chronic renal disease, vascular surgery, or cocaine-induced chest pain who do not have documented AMI.\textsuperscript{63,73,74,75,76,77,78} Measurement of cTnI would clarify the diagnosis in patients with concomitant myocardial and skeletal muscle injury.\textsuperscript{79} This distinction arises from the fact that cTnI is not expressed in fetal, diseased, or regenerating skeletal muscle.\textsuperscript{80,81,82}
Cardiac TnI is present only in the myocardium throughout ontogeny and has a unique amino acid sequence.\(^{47}\) Like cTnT, it exhibits appearance kinetics after AMI similar to that of CK-MB, becoming detectable in the serum within 4 h after infarction, peaks at about 14 to 18 h, and remains increased for up to 5 to 7 days.\(^{45,46,73,74}\) Thus, cTnT provides mid- to late detection but lacks sensitivity at 0 to 4 h.\(^{74,76,77,83,84}\) At a cutoff value of 3.1 \(\mu\)g/L, the sensitivity of cTnI was 96.6 percent and specificity was 94.9 percent.\(^{79}\) With a different assay, Wu et al\(^{76}\) have reported a sensitivity of 100 percent by 6 h after infarction with an average specificity of 96 percent. While some investigators showed that cTnI and CK-MB have statistically indistinguishable diagnostic accuracies for the detection of AMI, others think cTnI is superior.\(^{45,51,74,76,79,86}\)

Similar to cTnT, cTnI has also been found to be sensitive and specific for the diagnosis of perioperative MI for patients undergoing vascular surgery and spinal surgery.\(^{83,84}\) It is devoid of surgical and clinical interferences from skeletal muscle cross-reactivity.\(^{73,75,76,82}\) In addition, cTnI is increased in unstable angina patients.\(^{76,77,78,79}\)

**Discussion**

Owing to the difference in molecular size of the cardiac markers, various lengths of time will be required for these proteins to be released into the circulation. Infarct size and location of infarction are other factors that affect the interval when the marker becomes increased above the upper reference limit after an AMI. Thus, different time windows of elevation of these markers have been reported. The important intervals of events for these markers are summarized in table I. Conceivably, a massive infarct will result in early elevation of proteins after cardiac necrosis. Thus, the time landmarks in table I must be taken as a guide and not as a rule. Graphically, the kinetics of evolution of these markers in the blood are represented in figure 1. From this figure, it can be realized that myoglobin and MB isoforms evolve earliest after an AMI and are the best choices for early diagnosis.\(^{15,16,17,18,19,20,21,36,37,38}\) A single serum myoglobin measurement has diagnostic utility at 3 h after the onset of symptoms.\(^{23}\) The BIOMACS study has shown that myoglobin can be used to exclude 64 percent of patients within 3 h of admission and that the combination of myoglobin and MB is superior for diagnosis of AMI with 98 percent sensitivity and 93 percent specificity after 6 h.\(^{85}\)

Combining ECG and myoglobin measurement substantially improved the sensitivity and predictive accuracy for early diagnosis of AMI.\(^{11}\) Bakker\(^{55}\) showed that the best predictors of AMI within 4 h after onset of chest pain were ECG and myoglobin. Puleo et al\(^{26,35,36}\) have demonstrated that MB isoforms have better sensitivity and specificity within 6 h of infarct. In contrast, CK-MB activity does not achieve the diagnostic accuracy that isoforms offer at 6 h until 10 to 12 h post-infarct.\(^{26}\) Its variable normal serum levels, its presence in noncardiac muscular tissues, and its brief elevation in serum during course of an AMI limit the diagnostic value of CK/CK-MB determinations.\(^{40,44}\) After 48 h, the measurement of CK-MB is frequently of little diagnostic utility.\(^{43}\) In this regard, cTnT and cTnI are far superior. They also remain elevated longer than total LD and LD1 and are highly sensitive and specific.\(^{44}\) In comparison between the two, cTnI seems to be exceptional.\(^{76,86}\)

Based on the arguments set forth previously, a strategic utilization scheme is proposed of the cardiac markers for triage of patients admitted to the hospital for cardiac evaluation. As depicted in figure 2, patients with chest pain are first evaluated with ECG. If there are characteris-
<table>
<thead>
<tr>
<th></th>
<th>Myoglobin</th>
<th>CK</th>
<th>CK-MB</th>
<th>MB Isoform</th>
<th>LD</th>
<th>LD1</th>
<th>cTnT</th>
<th>cTnI</th>
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<tbody>
<tr>
<td>Mol. wt.</td>
<td>17,800</td>
<td>86,000</td>
<td>86,000</td>
<td>86,000</td>
<td>135,000</td>
<td>135,000</td>
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<tr>
<td>Reference range</td>
<td>&lt;90 ng/mL</td>
<td>24–195 U/L</td>
<td>10–25 U/L (&lt;5%)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>MB2&lt;2.6 U/L</td>
<td>35–88 U/L (L→P)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>14–36% of total LD</td>
<td>&lt;0.1 ng/mL</td>
<td>&lt;0.5 ng/mL&lt;sup&gt;d&lt;/sup&gt;</td>
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<td></td>
<td>0–9 ng/mL (&lt;2.5)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>MB2/MB1&lt;1.4</td>
<td>83–100 U/L (P→L)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.45–0.74</td>
<td>&lt;3 ng/mL&lt;sup&gt;d&lt;/sup&gt;</td>
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<td>Cutoff</td>
<td>100 ng/mL</td>
<td>200 U/L</td>
<td>&gt;25 U/L (&gt;5%)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>MB2&gt;2.6 U/L</td>
<td>&gt;100 U/L (L→P)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>&gt;40% of total LD</td>
<td>0.1 ng/mL</td>
<td>1.5–3.1 ng/mL</td>
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<td></td>
<td>10 ng/mL (&lt;2.5)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>MB2/MB1 &gt;1.5</td>
<td>&gt;200 U/L (P→L)&lt;sup&gt;b&lt;/sup&gt;</td>
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<td>Time to above reference</td>
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<td>3–8 hr</td>
<td>3–8 hr</td>
<td>1–4 hr</td>
<td>8–18 hr</td>
<td>8–18 hr</td>
<td>3–6 hr</td>
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<td>Time to peak</td>
<td>5–12 hr</td>
<td>10–36 hr</td>
<td>9–30 hr</td>
<td>4–8 hr</td>
<td>24–72 hr</td>
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<td>10–24 hr</td>
<td>14–20 hr</td>
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<td>Time to normal</td>
<td>18–30 hr</td>
<td>72–96 hr</td>
<td>48–72 hr</td>
<td>12–24 hr</td>
<td>6–10 days</td>
<td>6–10 days</td>
<td>10–15 days</td>
<td>5–7 days</td>
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<tr>
<td>Sensitivity 0–6 hr</td>
<td>50–100%</td>
<td>NR&lt;sup&gt;c&lt;/sup&gt;</td>
<td>17–62%</td>
<td>92–96%</td>
<td>NR&lt;sup&gt;c&lt;/sup&gt;</td>
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<tr>
<td>Specificity 0–6 hr</td>
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<td>92–100%</td>
<td>94–100%</td>
<td>NR&lt;sup&gt;c&lt;/sup&gt;</td>
<td>NR&lt;sup&gt;c&lt;/sup&gt;</td>
<td>74–96%</td>
<td>93–99%</td>
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<sup>a</sup> Percent or index of creatinine kinase-MB/total creatinine kinase.

<sup>b</sup> Assay direction: P → L, pyruvate to lactate; L → P, lactate to pyruvate.

<sup>c</sup> NR = not reported.

<sup>d</sup> Method dependent.
tic diagnostic changes of the ECG pattern, the patient can be diagnosed as having an AMI which can be further confirmed with cTnI. Since diagnostic ECG changes are 100 percent specific, serum markers may not be necessary for confirmation. However, cTnI can be used to monitor the effect of thrombolytic therapy. Patients with non-diagnostic ECGs will be tested for the cardiac markers. The choice of the markers depends on the time interval from onset of symptoms. Considering the diagnostic windows of the markers, a cutoff of 8 h after infarction will effectively distinguish the usefulness of the early and other tests. If the onset of symptoms is within 8 h, myoglobin or MB isoforms should be tested. However, if the patient has any skeletal muscle injury, myoglobin assay should not be used since it is not specific to the myocardium. If the result is positive, AMI is possible. The diagnosis can be confirmed at least 2 h later with cTnI. If the result is negative, the early marker should be retested after 2 h. If the result remains negative, MI may be ruled out, which can be confirmed later with cTnI. If a patient is admitted later than 8 h after chest pain, either cTnT or cTnI may be used for diagnosis, although cTnI is superior and cheaper. If the result is negative, AMI may be ruled out. However, if the result is positive, AMI is possible, and the test may be repeated to confirmed the diagnosis. The results of cTnT or cTnI may be used to stratify patients with unstable angina. Point-of-care rapid
cTnT or CK-MB spot test may be used if the patient is admitted after 5 h of symptoms. Although the turnaround time may be shortened, the tests are not cost-effective.

In this protocol, LD1/LD and CK-MB/CK are not utilized. The use of cTnT or cTnI efficiently covers the time window of these classical markers.39,76 By combining the usage of ECG, myoglobin (MB isoform if skeletal injury is present) and cTnI, any suspected AMI may be diagnosed from the onset of symptoms to 7 days after. Use of cTnT will further extend that period to 14 days.

The proposed protocol will be economical in the triage of patients. Early diagnosis with myoglobin or MB isoform will increase the efficiency of thrombolytic therapy. Replacing LD, CK, and its isoenzymes with cTnI or cTnT eliminates serial testing, thus saving time in the triage of the patients. In addition, the cost of the cTnI is lower than all the enzyme assays and is anticipated to continue to decrease in the future.

References