Risk Stratification for Patients with Non-ST-Segment Elevation Acute Coronary Syndromes in the Emergency Department

Dear Colleagues:

Emergency physicians routinely risk stratify multiple patients simultaneously in the emergency setting. Identifying the critically ill or injured patient rapidly allows for appropriate therapy to be delivered in a timely fashion. For patients with possible acute coronary syndrome, the 12-lead electrocardiogram obtained within 10 minutes after arrival to the ED quickly identifies patients with ST-segment elevation myocardial infarction (STEMI) requiring rapid reperfusion by percutaneous coronary intervention in the cardiac catheterization laboratory or the administration of fibrinolytic therapy if cardiac catheterization will be delayed. For patients with unstable angina and non-ST-segment elevation myocardial infarction (NSTEMI), other diagnostic modalities help to define the critically ill patient.

Cardiac biomarkers, particularly the troponins, are perhaps the best indicators of a patient with non-ST-segment elevation acute coronary syndrome (NSTE-ACS) at risk for ischemic complications. Over the last 2 decades, serial cardiac biomarkers have proved extremely effective in establishing the diagnosis of myocardial necrosis in the NSTE-ACS patient. Troponin also identifies the high risk NSTEMI patient who will benefit from administration of anti-thrombin and anti-platelet therapy. In multiple NSTE-ACS trials, patients with elevated troponin levels benefit while those with normal troponin levels do not benefit from these important therapies. Other diagnostic modalities such as radionuclide myocardial perfusion imaging, contrast echocardiography, and CT coronary angiography also provide important diagnostic information for patients with possible ACS presenting to the ED.

In this EMCREG-International Newsletter, Dr. Gerry Brogan explores this important topic of risk stratification of patients with possible ACS in the ED. Comprehensive in nature, we hope this guide helps provide you with the background necessary to confidently approach this difficult clinical problem. It is our hope this information helps you to continue to provide outstanding care for your patients with possible ACS.

Sincerely,

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Objectives:

1. Understanding the positive and negative predictive value of the various components of the history and physical examination of the chest pain patient.
2. Understand the use of cardiac biomarkers and their value in both the diagnosis and risk stratification of patients with possible NSTE-ACS.
3. Evaluate the utility of provocative testing in the low risk chest pain patient.
4. Understanding the process of risk stratification and the appropriate disposition of the chest pain patient with NSTE-ACS.
5. Review cardiac biomarkers for both ischemia and infarction that are in development and have potential diagnostic utility in the emergency department setting.

Introduction

Ischemic heart disease remains one of the leading causes of mortality in the United States, accounting for more than 20% of the 2.4 million total deaths in 2002.1 Unstable angina (UA) and non-ST-segment elevation myocardial infarction (NSTEMI) accounted for 1,295,000 hospital admissions in the US in 2001, almost 60% of those patients were 65 years of age or older, and 41% were women.2 The National Center for Health Statistics recently reported 5,637,000 US emergency department (ED) chest-pain syndrome visits, accounting for approximately 5% of total visits.3 Accurate diagnosis and risk stratification of the UA/NSTEMI patient is essential to identify patients at risk and to initiate appropriate treatment. Underscoring this challenge was a recent multi-center study that demonstrated approximately 2% of patients with myocardial infarction (MI) and 2.3% of UA patients were inadvertently discharged from the ED.4

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History and Physical Examination

Angina is characterized as a deep, poorly localized chest or arm discomfort associated with physical exertion or emotional stress, relieved promptly by rest or sublingual nitroglycerin (NTG). Patients with UA may have discomfort that has all of the qualities of typical angina, except the episodes are more severe or prolonged, may occur at rest or may be precipitated by less exertion. Jaw, neck, ear, arm, epigastric discomfort or unexplained dyspnea may be the sole manifestations of UA. Isolated unexplained new onset or worsening exertional dyspnea is the most common anginal equivalent symptom. Other symptoms include nausea, vomiting, diaphoresis, and unexplained fatigue.

While typical characteristics of the chest discomfort raise the probability of coronary artery disease (CAD), atypical features, such as sharp stabbing pain or reproduction of the pain with palpation, do not exclude acute coronary syndrome.

In patients with symptoms of possible ACS, some traditional risk factors for CAD including hypertension, dyslipidemia, or cigarette smoking, are only weakly predictive of the likelihood of acute ischemia and are less important than the history, electrocardiogram (ECG), and cardiac biomarkers. The presence or absence of these traditional risk factors should not be used to determine whether an individual patient should be admitted or treated for ACS. Diabetes and presence of extra cardiac (peripheral or carotid) arterial disease are major risk factors for poor outcomes in patients with known ACS, reflected in higher mortality and risk of acute heart failure.

Recent guidelines recommend that a 12-lead ECG be obtained within 10 minutes of arrival to the ED in patients with ongoing chest pain, and as soon as possible in all other patients. The initial 12-lead ECG obtained in the ED in patients with suspected ACS is diagnostic of injury in only 24% to 60% of patients with the final diagnosis of acute myocardial infarction (AMI). Initial ECG findings that have prognostic and diagnostic significance include ST-segment elevation, ST-segment depression, left bundle branch block (LBBB) and T-wave inversion. Findings of
ST-segment elevation or depression, pathologic Q waves or T wave inversion on the ECG correlated with increased odds of percutaneous coronary intervention (PCI), MI or coronary artery bypass grafting (CABG). A recent study reported that the one year mortality/MI rate doubled among patients with significant ST-segment depression compared to patients with minor or no ST-segment changes. Serial ECGs increase sensitivity for AMI.

In addition, ST-segment ischemic changes identified by continuous monitoring predict cardiac related death or MI in patients with UA and a non-diagnostic ECG, but are most useful in patients at high risk for ACS. Additional posterior and right-sided leads can increase the sensitivity of the ECG for ST-segment changes, from 56% up to 78%. Incorporating V3R, V8 and V9 leads, Zalesinski et al. demonstrated a 15-lead electrocardiography sensitivity for ST-segment elevation myocardial infarction (STEMI) of 59% versus standard 12-lead sensitivity of 48%. Body surface mapping electrocardiography has been shown to have increased sensitivity with no decline in specificity compared to traditional 12-lead ECG. Although audioelectric ECG data can help risk stratify heart failure patients, abnormal heart sounds have not correlated well with the diagnosis of ACS in an undifferentiated, low-risk ED population.

A recent ED study compared patients who normal or non-diagnostic ECGs taken during symptoms to those asymptomatic patients with similar ECG findings. Patients who were symptomatic during acquisition of a normal or non-diagnostic ECG had rates of adverse cardiovascular events similar to those of patients without symptoms.

**Risk Scores**

Antman et al., developed a 7-point risk score: age greater than 65 years, more than 3 coronary risk factors, prior angiographic coronary obstruction, ST-segment deviation, more than 2 angina events within 24 hours, use of aspirin within 7 days, and elevated cardiac biomarkers. The risk of developing an adverse outcome such as death, re-infarction or recurrent severe ischemia requiring revascularization, ranged from 5% to 41% with the TIMI (Thrombolysis In Myocardial Infarction) risk score, defined as the sum of the individual prognostic variables. The score was derived from data of the TIMI IIb trial and validated in 3 additional trials – ESSENCE, TACTICS-TIMI-18 and PRISM-PLUS. Among patients with UA/NSTEMI there is a progressively greater benefit from newer therapies such as low molecular weight heparin (LMWH), glycoprotein (GP) IIb/IIIa inhibition, and an invasive strategy with increasing risk score.

Applicability in the ED setting was confirmed in a study of 1,458 consecutive ED chest pain patients. However, the 30-day death, AMI and revascularization rate for TIMI risk score of zero was still 1.7% (95% CI 0.42-2.95) indicating that the TIMI risk score should not be used in isolation to determine disposition of ED chest pain patients. Pollack et al. also demonstrated that although TIMI risk score correlated with 30 day outcomes, patients with a score of zero still experienced a 2.1% event rate.

**Cardiac Biomarkers**

Myoglobin, a low-molecular weight protein found in cardiac and skeletal muscle, is not cardiac specific, but is released more rapidly from myocardium than CK-MB or troponin and is detectable as early as 2 hours and peaks at 4-6 hours after the onset of myocardial necrosis. Utilizing a single cut-off value or a delta value of 40 ng/ml between a presentation sample and a sample one hour later, myoglobin has a sensitivity of 91%, specificity of 87%, and a 99% negative predictive value (NPV) to rule out AMI in an ED chest pain population. The average time from symptom onset to presentation in this study group was 3.2 hours. Due to lower specificity than other cardiac biomarkers, myoglobin is best utilized as an early “rule out” myocardial necrosis biomarker and should be sampled only when combined with more specific cardiac biomarkers such as CK-MB or troponin.

CK-MB until recently has been a commonly used cardiac biomarker for myocardial cell necrosis, rising within 3-4 hours after the onset of symptoms, reaching twice the upper limit of normal at 6 hours, with peak values at 12-18 hours after symptom onset. Skeletal muscle injury and increasing levels of renal failure can reduce the specificity of CK-MB. CK-MB can provide important prognostic information and predict mortality risk. Troponin I or T has replaced CK-MB in many institutions consistent with recently revised diagnostic criteria for AMI published by the American College of Cardiology (ACC) and European Society of Cardiology.

Cardiac troponin T (cTnT) and cardiac troponin I (cTnl) levels rise in the blood similar to CK-MB, but can remain elevated for up to 10-14 days. If the initial troponin sample is elevated, CK-MB may be needed to define the temporal course of the infarct. As a result of these release kinetics, patient blood should be sampled at least 6 to 9 hours after presentation to provide adequate clinical sensitivity for AMI.
Elevated levels of cTnT or cTnl convey prognostic information beyond that supplied by the clinical characteristics of the patient, the ECG at presentation, and a predischarge exercise test. Furthermore, among patients without ST-segment elevation and normal CK-MB levels, elevated cTnI or cTnT concentrations identify those at an increased risk of death. Finally, there is a quantitative relationship between the quantity of cTnI or cTnT that is measured and the risk of death in patients who present with an ACS. However, troponins should not be relied on as the sole cardiac marker for risk, because patients without troponin elevations may still exhibit substantial risk of an adverse outcome. Neither marker is totally sensitive and specific in this regard. With currently available assays, cTnI and cTnT are of equal sensitivity and specificity in the detection of cardiac injury.

Patients who present without ST-segment elevation and have elevated troponin levels receive a greater treatment benefit from platelet GP IIb/IIIa inhibitors and LMWH. For example, in the c7E3 Fab Antiplatelet Therapy in Unstable Refractory Angina (CAPTURE) trial, patients with an elevated cTnT level on presentation had a rate of death or nonfatal MI of 23.9% when treated with placebo vs. 9.5% when treated with abciximab (p=0.002) whereas among patients with a normal cTnT level, the rate of death or MI was 7.5% in the placebo group vs. 9.4% in the abciximab group (p=NS). Similar results have been reported for cTnI and cTnT with use of the GP IIb/IIIa inhibitor tirofiban and also in the Fragmin during Instability in Coronary Artery Disease (FRISC) trial of UA patients randomized to dalteparin.

Newby et al. confirmed enhanced efficacy of GP IIb/IIIa inhibitors in the treatment of high risk UA/NSTEMI patients with a positive troponin level at the time of presentation (Figure 1). The prognostic value of troponin was again demonstrated in the recently published 5-year outcome data from FRISC-II, which demonstrated that the troponin positive patients experienced sustained benefit of an early invasive strategy. In a TACTICS-TIMI-18 sub-study, Dokainish et al showed an elevated troponin in ACS was associated with a higher risk for 6-month death or re-infarction, even among patients who did not have significant angiographic CAD (Figure 2). Only patients with elevated troponin levels demonstrated benefit with an early invasive strategy (Figure 3). In summary, patients with ACS and elevated troponins derive greater benefit from treatment with GP IIb/IIIa inhibitors, LMWHs and early PCI.

Other Cardiac Biomarkers

B-type natriuretic peptide (BNP) is increased as a result of non-cardiac ventricular wall stress and tissue hypoxia rather than
cell injury, and has been shown to be a prognostic indicator independent of hemodynamic variables and other biochemical markers in patients with known ACS. In a study evaluating BNP in an ED setting, patients were sampled at presentation and 90 minutes later for BNP in addition to myoglobin, CK-MB and troponin. The BNP cutoff was derived from receiver operating curves with the upper limit of normal cutoff being 51, 31 and 31 pg/ml for AMI, ACS and 30 day events, respectively. When BNP was added to cTnI, CK-MB and myoglobin, an increase in sensitivity and trends towards increased NPV and decreased specificity were demonstrated, along with a trend towards decreased positive predictive value (PPV). In a sub-study of the ICTUS trial, 1,148 patients with NSTEMI ACS and an elevated cTnI had baseline N-terminal proBNP (NT-proBNP) levels analyzed. Mortality at 1 year was 7.3% in the highest quartile NT-proBNP group compared with 1.1% of patients in the lower three groups (p <0.0001). In this study, NT-proBNP levels were not associated with the increased incidence of recurrent MI by 1 year, nor could increased levels identify patients who would benefit by early invasive strategy compared to a selected invasive strategy.

As destabilization of an atherosclerotic plaque and rupture result in part from an inflammatory process, there has been interest in markers of inflammation, such as C-reactive protein (CRP), serum amyloid A, and interleukin-6 levels in ACS. Patients without biochemical evidence of acute myocardial necrosis, but who have an elevated CRP, are at increased risk of an adverse outcome especially in those whose CRP levels are markedly elevated in the highest quintile. This biomarker has not been shown to be useful in the acute evaluation of patients in the ED.

Myeloperoxidase (MPO) is a hemoprotein released into the extracellular fluid and general circulation during inflammatory conditions, such as a destabilization of coronary artery plaques leading to ACS. In the TACTICS-TIMI 18 trial, MPO mass concentration was measured in 1090 patients with ACS. Death and MI rates were determined at 6 month follow up. With a cutoff of 350 µg/ml, the adjusted hazard ratio was 2.25 (95% CI 1.32 to 3.82). The effects were particularly impressive in patients with an undetectable troponin level where the hazard ratio was 7.48 (95% CI 1.98 to 28.29). Of note, only an admission troponin T was used to define this group. Metalloproteinases (MMP) are physiologic regulators of the extracellular matrix found in most tissues. In the heart, they participate in vascular remodeling, plaque instability and ventricular remodeling after cardiac injury. In a study from the AtheroGene investigators evaluating 1,127 patients with either stable (n= 795) or unstable (n=332) CAD, values of MMP-9 were related to future cardiovascular deaths. Although there was some association with other markers, MMP-9 after correction for CRP, fibrinogen,IL-6 and IL-18 levels maintained prognostic significance. Circulating soluble CD 40 ligand (sCD40L) is derived largely from activated platelets and can trigger an inflammatory reaction in vascular endothelial cells by secretion of cytokines and chemokines. In a study of patients presenting to the ED with acute chest pain, sCD40L concentration > 5 µg/L reliably identified patients with increased cardiovascular risk. Pregnancy-associated plasma protein A (PAPP-A) is an insulin like growth factor IGF-dependent binding protein-4-specific metalloproteinase and potentially a pro-atherosclerotic molecule through its role in disrupting integrity of the atheroma’s protective cap. In a series of 136 consecutive patients presenting to the ED with suspected ACS and cTnI negative during the first 24 hours after admission, an increase in circulating PAPP-A appeared to be an independent predictor of future ischemic cardiac events, as well as the need for PCI or coronary artery bypass surgery. An adjusted risk ratio of 4.6 (95% CI 1.8 to 11.8) was obtained.

Whole blood choline (WBCHO) and plasma choline (PLCHO) concentrations increase rapidly in coronary plaque destabilization and tissue ischemia. The combination of WBCHO and cardiac troponins allowed a superior risk assessment compared with each test alone in a recent ACS study. In this study, WBCHO was not a marker for myocardial necrosis but indicated high risk unstable angina in patients without AMI (sensitivity 86.4%, specificity 86.2%). The albumin cobalt binding (ACB) test is a quantitative assay that measures ischemia-modified albumin (IMA) in human cells. In a study evaluating IMA for the diagnosis of cardiac ischemia in patients presenting to the ED with symptoms of ACS, the sensitivity of IMA at presentation for chest pain of ischemic origin was 82% (95% CI 74-84%), specificity 46% (95% CI 34-57%).
Risk Stratification for Patients with Non-ST-Segment Elevation Acute Coronary Syndromes in the Emergency Department

the NPV was 59% and the PPV was 72%. In this trial, IMA, ECG and cTnT in combination identified 95% of patients whose chest pain was attributable to ischemic heart disease.52 In patients presenting with ischemic symptoms of ACS, plasma free fatty acids (FFA) monitoring may provide an early indication of cardiac ischemia. In myocardial infarction patients, FFA was increased in 100% of patients on presentation, whereas only 22% of these patients had increased cardiac Troponin I at presentation.53

Glycogen phosphorylase isoenzyme BB (GPBB) also increases early in patients with ACS and reversible ST-segment alterations in the resting ECG.54 Placent al growth factor (PIGF) has been implicated as a principle instigator of plaque instability, which is the physiologic common denominator for coronary artery thrombus formation and ACS. In an ED cohort from the CAPTURE trial, PIGF was also an independent predictor of important outcomes (p<0.001). In particular, patients who were negative for cTnT, sCD40L, and PIGF had very low cardiac risk for complications showing no events by 7 days after presentation, and a 2.1% event rate at 30 days after presentation.55

Provocative Testing

If patients are not found to have NSTEMI or high risk UA in the ED, including a non-diagnostic ECG and negative cardiac biomarkers they should have a stress test and/or imaging study performed. Further tests and options include exercise stress testing with or without intravenous contrast, resting nuclear scan, stress echocardiogram with or without contrast, computer tomographic (CT) coronary angiography or cardiac catheterization based on the patient’s clinical presentation and course during evaluation.

Stress testing is determined by the patient’s resting ECG, ability to perform exercise, and local availability and expertise of the operator. The standard treadmill exercise test is utilized in most patients, except for those who have baseline ST-segment abnormalities on their 12-lead ECG such as left ventricular hypertrophy (LVH), intraventricular conduction defect, paced rhythm, pre-excitation syndrome or digoxin effect. Patients with these conditions should have a radionuclide imaging modality included in the exercise stress test. Stress test features that are markers of adverse outcomes include a Duke treadmill score of -11 or less, extensive ischemia on imaging, stress induced left ventricular dilatation or severe dysfunction defined as ejection fraction <35%, and increased lung intake of radionuclide (thallium-201). Patients who have a high risk stress test, recurrent chest pain, abnormal ECG features, or cardiac biomarker elevations during observation period should also be admitted and undergo early coronary angiography. Exercise stress testing, based on data from an analysis of 147 consecutively published reports involving 24,074 patients, has an overall sensitivity of 68% and a specificity of 77%.56

Standard 2-dimensional echocardiography is not sensitive for AMI, especially with small myocardial infarcts. It is also not sensitive for ischemia. In a study comparing the prognostic value of myocardial contrast echocardiography (MCE) in patients presenting with acute chest pain and negative troponin levels, stress MCE was superior to TIMI risk score and exercise electrocardiography in the assessment of risk in suspected ACS patients. Although the study was not designed to test the accuracy of MCE for the detection of significant CAD, MCE demonstrated a sensitivity of 80% and a specificity of 56% in 34 patients who underwent coronary arteriography compared with the sensitivity of 45% for exercise stress electrocardiography.57

In a study of 158 consecutive patients with chest pain and possible ACS who underwent dobutamine myocardial contrast echocardiography stress testing, a sensitivity, specificity and accuracy for detecting a greater than 50% coronary artery stenosis was 92%, 77% and 88% respectively. This compared favorably to 62%, 85% and 67% for wall motion analysis.58

The use of myocardial perfusion imaging (SPECT with technetium Tc 99m sestamibi) for evaluation and treatment of chest pain patients suspected of acute coronary ischemia was evaluated in a trial of patients presenting with suspected acute ischemia but no initial ECG changes. Patients were included only if symptoms were ongoing or resolved no longer than 3 hours prior to consent. Patients were excluded with a history of previous MI. Patients were normally assigned to receive either the usual ED evaluation strategy (n=1,260) or the usual strategy supplemented with results from acute resting myocardial perfusion imaging using single photon computerized tomography with injection of 20-30 mCi Tc-99m sestamibi (n=1,215). Results were interpreted in real time and provided to the ED physician for decision-making. Among patients without cardiac ischemia, hospitalization was 52% in the group with usual care vs. 42% when the sestamibi scan was performed and results returned to the treating physician. Among the 1,215 patients randomized to the scan strategy, the imaging results were related to the risk of an adverse outcome. Among patients with normal, equivocal or abnormal scan results, the risk of AMI was 0.6%, 0.8% and 10.3% respectively. A similar relationship was observed between scan findings and any cardiovascular event at 30 days defined as AMI, death or revascularization. Among patients with normal, equivocal or abnormal scan results, the risk a cardiac event as defined previously was 3.0%, 6.1% and 20.5% respectively (relative risk equivocal or abnormal vs. normal scan, 3.8; 95% CI 2.36-6.21, p<0.001).59
Computed tomographic angiography evaluation of the coronary arteries is a newer methodology for the evaluation of patients with potential ACS (Figure 4). Calcium scoring performed using electron beam CT (EBCT) detects coronary calcium but does not detect plaque without calcification. While studies in ED patients have demonstrated the safety of using the absence of coronary calcifications as a criteria for risk stratification, the addition of multi-detector 64-slice CT (MDCT) coronary angiography to calcium scoring further enhances this diagnostic test, allowing the detection of coronary calcification, non-calcified atherosclerotic plaque, as well as coronary luminal stenosis.

Goldstein et al. compared the accuracy of CT coronary angiography with stress myocardial perfusion imaging for the detection of an ACS or 30-day major adverse cardiac events in low-risk chest pain patients following a “rule out” in an observation unit. All patients had both rest and stress myocardial perfusion imaging and CT coronary angiography. Patients with abnormal myocardial perfusion imaging defined as reversible perfusion defects or positive CT coronary angiography results, defined as stenosis >50% or calcium score > 400, were considered for cardiac catheterization. Those with discordant results had a 30-day re-evaluation. Of 85 study patients, 7 (8%) were found to have significant coronary stenosis and none had MI or an adverse cardiovascular event during 30 days follow-up. The sensitivity of myocardial perfusion imaging was 71% (95% CI 36-92%) and CT coronary angiography was 86% (95% CI 49-97%). The specificity was 90% (95% CI 81-95%) for myocardial perfusion imaging and 92% (95% CI 84-96%) for CT coronary angiography. The NPV of myocardial perfusion imaging and CT coronary angiography was 97% (95% CI 90-99%) and 99% (95% CI 93-100%), respectively and the positive predictive value was 38% (95% CI 18-64%) and 50% (95% CI 23-75%). These data suggest that the performance of CT coronary angiography is at least as good as that of stress myocardial perfusion imaging for detection or exclusion of ACS in low risk chest pain patients.

Hollander et al. reported 30-day cardiovascular outcomes in a cohort of patients discharged from the ED on the basis of CT coronary angiography. This study evaluated low risk patients including TIMI risk score less than or equal to 2, and a non-ischemic ECG, who underwent CT coronary angiography in the ED, rather than being admitted for a rule-out protocol. Patients with a negative CT angiogram, defined as calcium score of less than 100 and less than 50% stenosis in coronary artery, were discharged home. The primary outcome was 30-day death or MI. Ninety-three patients were enrolled and 12% had positive findings on CT coronary angiography. After CT coronary angiography, 69 patients (75%) were discharged from the ED. Eighty six patients (92%) were followed for 30 days and none had a adverse event during index hospitalization or at 30-day followup (0%; 95% CI 0-3.5%).

Integrated Protocols

Fesmire et al. demonstrated the utility of an integrated accelerated chest pain protocol to identify and exclude ACS in ED patients with chest pain. They evaluated 2,074 potential ACS patients with serial 12-lead ECGs, cardiac biomarker testing using 2 hour delta values, and selective nuclear stress testing. Patients were followed for 30 day ACS complications including AMI, PCI, CABG, significant CAD, life threatening complication or cardiac death. At completion of the full ED evaluation protocol, sensitivity and specificity for 30 day ACS events were 99.1% (95% CI 97.3 – 99.8) and 87.4% (95% CI 85.8 – 88.9) respectively (positive LR 7.9, negative LR 0.01).
Risk Stratification for Patients with Non-ST-Segment Elevation Acute Coronary Syndromes in the Emergency Department

Figure 5: Integration of the 2002 ACC/AHA guidelines for diagnostic and treatment strategies in the emergency department for patients with ACS. Reprinted with permission from Gibler WB, Cannon CP, Blomkalns AL, et al.: Circulation 2005;111:2699-2710.63

Conclusion

In summary, the history, including cardiac risk factors, physical examination, 12-lead ECG, and cardiac biomarkers can help identify low versus high risk patients with possible ACS. Validated risk stratification algorithms, such as the TIMI Risk score, can be used to integrate these multiple findings into a practical tool in the emergency setting. Low risk patients can then be further risk-stratified using provocative testing, such as stress testing, echocardiography, resting perfusion imaging, and CT coronary angiography to identify individuals safe for discharge from the ED. Therapy will be driven by findings of patients at high risk for ACS (Figure 5). Appropriate follow-up by a cardiologist or primary care physician will help provide an optimal outcome for these patients.

REFERENCES


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CME Post Test

After you have read the monograph carefully, record your answers by circling the appropriate letter answer for each question.

1. What percent of Medicare patients with UA can present with typical symptoms?
   a. 0%    c. 25%
   b. 10%   d. 50%

2. The initial ED 12-lead ECG, has an approximate sensitivity of ____ for AMI?
   a. 0%    d. 75%
   b. 20%   e. 90%
   c. 50%

3. Which of the following is true regarding cardiac troponins (troponin I or troponin T)?
   a. Release kinetics similar to CK-MB
   b. Can be elevated up to 10 to 14 days
   c. Is useful for risk stratification of patients who would benefit from aggressive medical management
   d. A, B and C

4) The addition of a negative resting sestimibi scan in low risk chest pain patients, along with the history, physical examination and cardiac biomarker testing, helps to further identify patients at very low risk for ACS.
   a. True
   b. False

5) Which of the following are true regarding CT coronary angiography?
   a. Newer 64-slice scanners demonstrate higher diagnostic accuracy than older 16-slice scanners.
   b. The performance of CT coronary angiography has been demonstrated to be at least as good as that of stress myocardial perfusion imaging for detection or exclusion of acute coronary syndromes in low-risk chest pain patients.
   c. CT coronary angiography is an attractive alternative from a cost perspective to traditional standard evaluation in a chest pain observation unit.
   d. The negative CT coronary angiogram is defined as no stenosis, or stenosis <50% with a calcium score of <100.
   e. All of the above

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