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Critical Care Update

Chest Pain

David J. Dries, MSE, MD

Nonspecific presentation of chest pain is a significant drain on emergency department (ED) and emergency medical service (EMS) resources. Although EMS is not often touched by the need to complete evaluation for patients with this problem, a review of recent articles in this area is appropriate. The recent trend in publications reflects a less invasive approach to the evaluation of patients with potentially significant coronary artery disease. It appears that evaluation can be faster and simpler than before, perhaps with less need for transport.

Carlton EW, Khattab A, Greaves K. Identifying patients suitable for discharge after a single-presentation high-sensitivity troponin result: a comparison of five established risk scores and two high-sensitivity assays. *Ann Emerg Med*. 2015;66:635-645.

Mahler SA, Riley RF, Hiestand BC, et al. The HEART Pathway randomized trial: identifying emergency department patients with acute chest pain for early discharge. *Circ Cardiovasc Qual Outcomes*. 2015;8:195-203.

Reichlin T, Twerenbold R, Wildi K, et al. Prospective validation of a 1-hour algorithm to rule-out and rule-in acute myocardial infarction using a high-sensitivity cardiac troponin T assay. *CMAJ*. 2015;187:E243-E252.

The presentation of patients with nonspecific chest pain, possibly cardiac in origin, occurs in millions of ED visits annually in the United States. Various estimates include numbers as high as 6 million visits or higher. Because of the obvious implications of misdiagnosis, the evaluation of these patients can entail significant use of medical resources. Guideline recommendations include serial measurements of cardiac troponin up to 6 or 12 hours after presentation to the ED, leading to the creation of a variety of cardiac observation unit strategies. Many patients require prolonged assessment before safe discharge, despite the fact that only 15% to 25% of these patients receive a final diagnosis of acute coronary syndrome. Excessive use of ED resources contributes to crowding and may contribute to adverse events in patients with both acute and nonacute coronary syndrome—related chest pain.

The first of the 3 articles cited uses high-sensitivity troponin assays as a means to reduce door-to-discharge times with serial assay testing during the first 3 hours after presentation. For these authors, a high-sensitivity troponin assay is defined as one that has a coefficient of variation less than 10% at the 99th percentile value and is able to detect cardiac troponin in greater than 50% of a reference population. The diagnostic performance of high-sensitivity assays when used in isolation is thought to be too low to allow immediate discharge of patients presenting to the ED after a single blood draw on arrival. To reduce door-to-discharge times, the authors of the first article cited combined troponin assessment with clinical chest pain risk scores to give an estimate of pretest probability in combination with high-sensitivity troponin testing to improve overall diagnostic effectiveness. Combination strategies are being investigated at multiple sites. However, the optimum choice of a rapid rule-out protocol remains unclear because the majority of existing clinical risk scores are untested in combination with high-sensitivity troponin assays.

These authors selected clinical risk scores for study if they had been previously evaluated in large ED cohort studies designed to improve throughput of chest pain patients. Risk scores evaluated include the time-honored Goldman Score, the Thrombolysis in Myocardial Infarction Score, the Global Registry of Acute Cardiac Events Score, the HEART Score (which incorporates History, ECG, Age, Risk factors, and Troponin), and, finally, the Vancouver Chest Pain Scale. Authors combine these clinical profiles with 2 high-sensitivity troponin assays. Consistent with many studies of this kind, the primary end point was fatal or nonfatal acute myocardial infarction occurring within 30 days of hospital presentation.

Although these data must be considered preliminary, Carlton et al identified the potential for the introduction of clinical risk scoring in combination with a single-presentation high-sensitivity troponin assay to reduce the length of stay for low-risk patients and allow discharge of approximately 30% of chest pain presenters after a single blood draw on arrival in the ED. The authors have a high standard of performance for this strategy. They seek a negative predictive value of 99.5% for the diagnosis of acute myocardial infarction with the combination of a single troponin draw with an established risk score.

The second contemporary article examines the HEART Pathway in combination with troponin measures at 0 to 3 hours to trend biochemical change. The HEART Pathway plus troponin strategy reduced objective cardiac testing during 30 days, reduced the length of stay, and increased early discharges. The HEART (plus troponin) Pathway patients were compared with subjects receiving usual care in this trial. Positive troponins led to admission as well as individuals screened as high

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risk using the HEART Pathway clinical parameters. It is interesting to note which parameters appeared commonly. For example, a large number of patients were identified at higher risk simply on the basis of age. Over 40% of electrocardiograms had nonspecific change. However, troponins on presentation and at 3 hours were negative in approximately 93% of patients, contributing to 46.8% of patients who could be discharged after the 3-hour protocol. The other 53% of patients either ruled in based on troponins or were believed to be at high risk based on other clinical parameters.

The most impressive use of troponin assays is seen in the third article in this group from the *Canadian Medical Association Journal*. These authors use high-sensitivity troponin assessment on presentation and again within 1 hour to evaluate biochemical changes indicative of myocardial infarction. The algorithm used incorporates baseline high-sensitivity cardiac troponin levels and absolute changes of the levels within the first hour. This reflects the very high diagnostic accuracy of 2 samples obtained such a short time apart. Specific criteria were established for positive and negative troponin level trends. Patients who did not rule in or rule out based on biochemical criteria were placed in an observation status.

Over 1,300 patients were studied. With the 1-hour high sensitivity troponin testing, 786 patients or 59.5% of the study population ruled out for myocardial infarction with a sensitivity of 99.6% and a negative predictive value of 99.9%; 16.4% of patients ruled in for myocardial infarction based on this 1-hour protocol, whereas 24.1% of patients were triaged for further evaluation, and the ultimate prevalence of acute myocardial infarction in the observational group was 18.6%. I should note that acute myocardial infarction in the group that had ruled out based on the 1-hour protocol occurred on 1 occasion. Specificity for patients ruling in with this protocol was 95.7% with a positive predictive value of 78.2%.

Obviously, work in this area is evolving rapidly. However, it appears that new assays and technologies are becoming available that will enhance our ability to provide appropriate resources to adequately screen patients without referral to cardiac centers.

Weinstock MB, Weingart S, Orth F, et al. Risk for clinically relevant adverse cardiac events in patients with chest pain at hospital admission. *JAMA Intern Med*. 2015;175:1207-1212.

Foy AJ, Liu G, Davidson WR Jr, et al. Comparative effectiveness of diagnostic testing strategies in emergency department patients with chest pain: an analysis of downstream testing, interventions, and outcomes. *JAMA Intern Med*. 2015;175:428-436.

Annual hospital charges of more than $11 billion for nonspecific chest pain were identified in the United States in 2006. Most patients admitted are eventually identified with a noncardiac diagnosis, suggesting a very low short-term risk for serious adverse events. Wide variability exists in the treatment and disposition of these patients based in part on risk aversion by the patient and the health care professional.

Hospital admission or extended observation after negative ED evaluation is assumed to confer a safety benefit for the patient with chest pain. However, this assumption remains untested. The potential for this benefit depends largely on the rate of treatable or avoidable adverse events during hospitalization. Previous studies have examined the primary rates of cardiac events at 30 days or longer, including adverse events after hospitalization and after outpatient follow-up. ED-based randomized trials of patients with chest pain that compared clinical pathways and disposition decisions have been unable to identify outcome benefits with any particular approach.

Inpatient admission or observation for managing potential adverse events constitutes a considerable burden to the health care system and the patient. In addition, risks of hospital admission including false-positive testing consequences, hospital-acquired infections, venous thromboembolism, pneumonia, falls, and other iatrogenic events may be significant.

Weinstock et al quantify the incidence of short-term, life-threatening events among patients with chest pain admitted to the hospital after ED evaluation is negative for ischemia including negative serial biomarker findings, normal vital signs, and nonischemic electrocardiogram findings. Adults presenting to the ED with chest pain, chest tightness, chest burning, or chest pressure who were subsequently admitted to the hospital after serial negative troponin testing in the ED were reviewed. A number of key events were identified including inpatient myocardial infarction, cardiac or respiratory arrest, death during hospitalization, and life-threatening arrhythmias. Screening for these events revealed these serious outcomes in only 20 of over 11,000 patients. When patients with serious events and other findings that justified hospitalization but did not qualify as serious adverse cardiac outcomes were screened out, important adverse events occurred in only 4 of 7,266 patients. What is more remarkable is that these data support the "notion" that adverse iatrogenic events, because of hospital admission, may cancel out potential benefits of hospitalization in low-risk patients. Contemporary estimates, cited by these authors, are that 1 in 164 hospitalized patients have a preventable adverse event contributing to death with serious harm in 10- to 20-fold more individuals. In this study, using conservative estimates, the risk of adverse events after a negative ED evaluation was 1 in 1,817, suggesting that this rate is the maximum potential rate of clinical benefit from hospitalization. Given the established risks of hospitalization, the authors suggest that current practice favoring admission, observation, or further provocative testing performed routinely on patients after ED evaluation for chest pain has negative implications and should be reconsidered.

The article by Foy et al examines the use of noninvasive cardiac imaging conducted early after discharge from the ED. Remarkably, there is no strong evidence that noninvasive testing reduces the risk of future cardiac events compared with a more conservative approach. This study examined patients receiving exercise electrocardiography, stress echocardiography, myocardial perfusion scintigraphy, and coronary computed tomographic imaging. The outcomes of patients subjected to initial noninvasive testing using 1 of these 4 modalities or no noninvasive testing were compared.

The authors gathered data from patients seen in the ED using health insurance claims data for a national sample of privately insured patients. Individuals screened had a primary or secondary diagnosis of chest pain in the ED. Outcomes were identified in association with 1 of 5 testing strategies including the 4 modalities described earlier and a strategy that excluded this noninvasive testing. In patients who underwent early noninvasive testing after ED evaluation, myocardial perfusion scintigraphy was used most often followed by stress echocardiography, exercise electrocardiography, and coronary computed tomographic imaging. The mean length of follow-up after index testing was 190 days. Only 464 patients (0.11%) and 1,396 patients (0.33%) were hospitalized with an acute myocardial infarction during 7 and 190 days of follow-up, respectively. Compared with the no-testing cohort, there was no significant difference in