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EDITORIAL COMMENT

Evaluation of Patients With Possible Cardiac Chest Pain

A Way Out of the Jungle*

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Emergency department (ED) overcrowding is a national health problem. From 1997 to 2007, the annual number of ED visits in the United States increased from 95 to 117 million, whereas the number of EDs decreased by 10% (1). This increased volume has put demands for acute care services at more than can be provided: 50% of urban hospitals and 31% of rural hospitals continuously operate at or over capacity (2). ED overcrowding is associated with adverse cardiovascular outcomes (3), reduced use of guideline-recommended therapies, and a higher risk of recurrent myocardial infarction (4).

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Given that chest pain is the second most common reason patients present to EDs across the United States (1), rapidly identifying patients with acute coronary syndrome (ACS) and efficiently identifying low-risk patients who can be safely evaluated in the outpatient setting is a critical issue. A key to facilitating the care of these patients is early risk stratification, proper use of sensitive and specific markers (e.g., cardiac troponin [cTn]), and comprehensive follow-up. This effort for low-risk patients is what has been evaluated by Than et al. in this issue of the *Journal* (5).

A total of 1,975 consenting adults presenting with symptoms suggestive of ACS were prospectively enrolled from 2 urban EDs. The investigators identified a low-risk group using an accelerated diagnostic protocol (ADP) that included patients with a Thrombolysis In Myocardial Infarction (TIMI) risk score of 0 at presentation, no new ischemic changes on the initial electrocardiogram (ECG), and cardiac troponin I (cTnI) concentrations at 0 and 2 h after arrival below the institutional cutoff for cTn elevation. The investigators were confident that close short-term follow-up with primary care physicians would occur in their local setting. Patients were prospectively followed to ascertain the occurrence of major adverse cardiac events (MACE), including ruling in for acute myocardial infarction (AMI) on the 12-h blood sample. The remainder of events included a composite of cardiac death, cardiac arrest, emergency revascularization, cardiogenic shock, ventricular arrhythmia requiring intervention, high-degree atrioventricular block requiring intervention, and AMI within 30 days of ED presentation.

Overall, of the 1,975 patients, there were 302 (15%) patients who had a MACE within 30 days. However, only 1 of these occurred in the 392 patients (20% of the cohort) who were ADP negative, giving the ADP a sensitivity of 99.7% and specificity of 23.4%. If this protocol were implemented in clinical practice, it could potentially identify 20% of ED patients who could be safely discharged within 2 to 3 h of presentation.

However, there are very significant limitations to this investigation. Specifically, 18% of ADP negative patients underwent therapeutic and 2% underwent procedural interventions during their initial presentation or within 30 days of the ED visit, introducing the risk of outcome misclassification due to cointervention. Moreover, 74% of these patients had additional investigations, 81% of which were cardiac stress tests, suggesting that further risk stratification after the ED visit was deemed essential in the majority of ADP negative patients. Thus, it could be argued that the results obtained reflect the design of the study, with more prolonged observation of some of the patients and aggressive follow-up in the others. Although this approach would be helpful to EDs, it would be ideal if the results did not depend so heavily on follow-up evaluations. This may work well in the 2 centers involved, but may not be the case in some others. Thus, there is room for improvement in the proximate elements of the evaluation, including the risk score and how cTn was used.

The TIMI risk score was originally developed to predict risk in hospitalized high-risk ACS patients. A recent meta-analysis of 17,265 patients from 10 prospective ED cohort studies (6) observed statistical heterogeneity among studies related to differences in the prevalence of cardiac events between cohorts. This would lead to anticipation of variability in the performance of the ADP among practice settings based on the baseline prevalence of MACE in a given population.

Several prediction models have been developed that may be better tailored for use in the ED setting. The North American Chest Pain Rule incorporates the clinician's assessment of chest pain etiology as well as readily available data from the initial ECG, cTn, and cardiovascular history. It had a sensitivity of 100% and a specificity

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of 20.9% among 2,718 patients enrolled from 3 centers (7). The HEART (History, ECG, Age, Risk Factors, and Troponin) score has also been developed (8) in ED chest pain patients as well as the Vancouver chest pain rule (9). This growing literature suggests that additional investigations will provide the needed refinements to identify low-risk ACS patients.

In addition, although the cTn assays used were reasonable fourth-generation cTnI assays, they were used at a higher cutoff value than suggested by the guidelines (10). Thus, by definition, they might have missed more patients than would be ideal. With more sensitive assays, such an approach may be improved, but it is unclear, especially in patients who present very early, that 2 h will provide a robust ability to exclude all AMIs. However, there are approaches that may help. Recently, Body et al. (11) reported that patients with an initial highly sensitive cTnI value below the level of detection had negligible rates of MACE over a 30-day period. If a group that had chest pain >6 h before presentation without intercurrent symptoms were added, this group might be as high as 40% of the ED cohort. If so, it might be that many patients could have AMI excluded at the time of admission. This would still leave some patients with possible unstable angina, but as assay sensitivity improves, this group would be smaller and smaller. The tension, however, is clear. There will be a much larger number of cTn elevations with these assays, both due to chronic diseases and acute diseases such as sepsis, stroke, and pulmonary embolism (12).

Where do we go from here? Is there a safe and efficient way out of the jungle? For the short term, an effective solution to the dilemma of ED overcrowding is, of necessity, collaborative. Although we anticipate improvements in our ability to define a low-risk cohort and in metrics necessary to optimally use high sensitivity cTn assays, a multidisciplinary team approach with good follow-up is needed to provide consistent, high-quality health care for possible ACS patients in the context of an emergency care system at its breaking point.

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