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REVIEW

Emergent diagnosis of acute coronary syndromes: Today's challenges and tomorrow's possibilities☆

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Summary Prompt diagnosis and effective early management of acute coronary syndromes within the Emergency Department are imperative. Arguably the most important step in the management of the acute coronary syndromes is identifying the problem in the first place. This narrative review explores the significant but under-recognised limitations to current diagnostic strategies and addresses both contemporary and possible future solutions in a rapidly evolving field.

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☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2008.02.006.

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Background

Coronary heart disease remains the single biggest killer in the United Kingdom, accounting for around one in five deaths in men and one in six deaths in women.[1] Approximately 3% of patients who attend the ED have chest pain that we suspect may be cardiac in origin.[2] 74—88% of these patients are admitted to hospital, making up one in five of all medical admissions.[2—4] Ultimately only a quarter of these patients will be diagnosed with an acute coronary syndrome (ACS), which implies that we adopt a very cautious approach to the problem. Despite this fact, up to 6% of the patients who are discharged from the ED actually have myocardial damage that is of prognostic significance.[5] This poses a question: why do we admit so many patients who do not have ACS but still miss the diagnosis in so many patients who do? In order to answer this question, this review aims to summarise the use and limitations of diagnostic strategies that are currently employed in the ED.

The ECG

American Heart Association and European Society of Cardiology guidelines recommend that all patients who present to the ED with chest pain should have a 12-lead ECG recorded within 10 min of arrival.[6,7] This is based upon evidence that longer delays are associated with adverse prognosis.[8] The high specificity (at least 94%) of ECG criteria makes it the diagnostic test of first choice for establishing a diagnosis of STEMI and enables confident early institution of measures to achieve revascularisation.[9]

In patients who do not have STEMI but are suspected to have NSTE-ACS, the ECG is still an important diagnostic tool. 32% of patients with T wave inversion and 48% of patients with ST depression will have AMI, as diagnosed using serum creatine kinase (CK). Regardless of whether these patients have AMI, T wave inversion and ST depression are powerful prognostic markers (Figure 1). ST depression is an independent predictor of 30-day mortality, even among troponin-negative patients.[10—14] These patients should have aggressive initial management and further investigation should be strongly considered regardless of troponin levels.

The ECG is, however, an insensitive tool and cannot be used to exclude the diagnosis of AMI. Among patients who present to the ED with suspected cardiac chest pain and have a normal ECG, 6% will have AMI.[15] In fact, the sensitivity of the ECG for establishing a diagnosis of AMI is as low as 25—50%.[16,17] Further, for establishing a diagnosis of acute cardiac ischaemia the ECG is less sensitive still, even using serial ECG's (sensitivity 21—25%).[18] Even during episodes of myocardial ischaemia as demonstrated on thallium scanning, 25% of patients with known left main stem or triple vessel disease will have a normal ECG.[19]

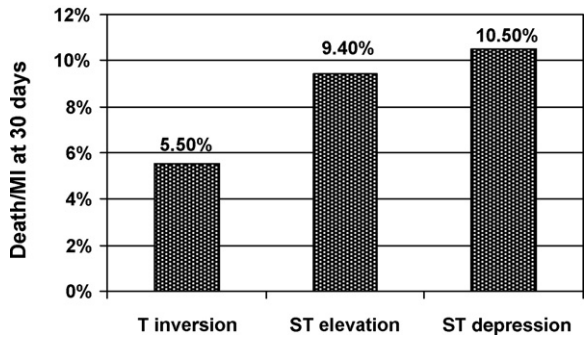

Figure 1 Prognostic value of the admission electrocardiogram.[10]

Clinical features

Traditional teaching that 90% of diagnoses can be established by history and examination alone does not apply to ED patients with suspected ACS. Clinical features are notoriously unreliable for establishing this diagnosis. Over half of patients with unstable angina and a third of patients with AMI will report atypical symptoms.[20,21] Up to one-third of patients with ACS do not experience any chest pain. They may present with dyspnoea, syncope, diaphoresis, pain in the epigastrium, arms or neck or they may report no symptoms at all. As a result, up to one-third of AMI's are initially unrecognised.[22,23] The prognosis for these patients is no better than patients with AMI that is initially recognised.

Given the high prevalence of atypical symptoms in patients with ACS, it is no surprise that systematic review has failed to identify any atypical features that help to exclude the diagnosis of ACS. On multivariate analysis, pleuritic pain carries an odds ratio of 0.6 (95% confidence intervals 0.2—1.7) for the diagnosis of ACS, while a tender chest wall also has an odds ratio of 0.6 (0.3—1.2) far from excluding the diagnosis.[24]

Many atypical clinical features that physicians often believe help to 'exclude' the diagnosis of ACS may actually be positive predictors of the diagnosis. Among ED patients with an 18% prevalence of AMI, pain that radiates to the right shoulder may shift the post-test probability of AMI to 39%.[15] Burning or indigestion-like pain may yield a post-test probability of 43%.[24]

Despite these statistics, a careful history remains an important diagnostic tool for the prudent physician who has a good appreciation of Bayesian principles. While no combination of clinical features has shown to accurately exclude ACS, combinations of typical or atypical features will shift the probability of the diagnosis. Among patients with suspected stable angina, combinations of typical symptoms give a very high probability of significant coronary artery disease (at least 90%) whereas combinations of decidedly atypical symptoms in low-risk groups such as young women are associated with low probability (about 5%) of disease.[25,26]

Cardiac troponins

The troponins are subunits of the thin filament associated troponin—tropomyosin complex, which helps to regulate muscle contraction. Genetic differences between skeletal and cardiac muscle have enabled the development of monoclonal antibodies to the cardiac troponins, which enables their quantification in peripheral blood.[27,28] Contemporary assays are available for troponins T and I, which are essentially equivalent.[29] Troponins represent the most sensitive