Editorial

Improving risk stratification for hospital mortality using troponin: is it time for a change of heart?

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In a recent issue of the journal *Critical Care*, Docherty *et al.* described their analysis of a strategy of routine cardiac troponin I (cTnI) testing at the Glasgow Royal Infirmary (1). In their study, they analyzed an observational cohort over 4 years at two busy ICUs, with independent derivation and validation cohorts, to determine whether cTnI testing added value to routine clinical evaluation of critically ill patients. What they found should cause some discomfort to all clinicians who take care of critically ill patients. At first blush, they found that cTnI was an independent predictor of hospital mortality across all patients. Full stop. However, after further review, the authors stand down and conclude that they would "not advocate the adoption of routine troponin analysis on admission to ICU." This incredible 180-degree turnaround requires some unpacking, so let's get to it.

If cTnI was such a great independent predictor in Docherty *et al.*'s study, then why isn't the strategy of routine troponin testing for ICU admission indicated? The answer for this specific study is that cTnI was compared with the very robust APACHE score, and the receiver operating curve characteristics did not show added value; additionally, the performance of cTnI lost utility after controls for confounding were performed.

Clinical predictors of in-hospital mortality depend on a variety of factors related to each patient's pathophysiology. Take, for example, the occurrence of myocardial injury following a drug overdose. Our group recently showed that initial cTnI results were highly associated with drug overdose mortality (2). Our group also previously demonstrated that myocardial injury is the most common major adverse cardiovascular event that occurs following acute drug overdose (3). In addition, there are several causes for circulating cTnI elevation in conditions other than acute coronary syndrome or heart failure (4,5). However, it remains extremely difficult to convince clinicians to send cTnI for Emergency Department (ED) patients who present without complaining of any chest pain.

But the greater question is, in real-world clinical practice, why isn't routine cTnI testing accepted yet for all critically ill patients (a la lactate)? The answer there is probably a combination of clinician skepticism, technology creep, and intuitive cost-benefit analysis. Even further, it raises the question regarding how clinicians should manage patients differently if their patient has elevated cTnI not ascribed to acute coronary syndrome. The truth is that more studies are needed to weigh the pros versus the cons in order for widespread practice change to occur. If and when a routine strategy for cTnI testing upon (or in consideration of) ICU admission will cross this line is anyone's guess.

The important work from Docherty *et al.* (1) serves to remind us that we need to continue to improve our efforts for in-hospital risk stratification, irrespective of disease pathophysiology. Perhaps the real issue that should sway our decision to send cTnI or not, is the timing the testing, for example in patients who are being *considered* for an ICU admission. Most ED patients are not risk stratified using APACHE as this is generally not part of clinical decision making from the ED perspective. Therefore, routine cTnI

testing may actually add value for selected patients prior to an APACHE assessment. Clearly, patients with elevated cTnI require telemetry monitoring and consideration of critical care unit admission. Future research should focus on high-risk clinical features to optimize strategies for utilization of cTnI as part of the routine evaluation of patients who are being considered for ICU admission.

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