Letter to the Editor

Hospital Admission and Myocardial Injury Prevalence after the Clinical Introduction of a High-Sensitivity Cardiac Troponin I Assay

To the Editor:

A cardiac troponin concentration above the 99th percentile is an indication of myocardial injury, and only a subset of all increased concentrations are caused by acute myocardial infarction (AMI). Interestingly, Sandoval et al. suggest that fewer AMI diagnoses may occur when using a high-sensitivity cardiac troponin I (hs-cTnI) assay with sex-specific 99th percentile cutoffs (1). This may well be a result of the superior analytical characteristics of the hs-cTnI assay, particularly at low concentrations near the decision levels (2). An important metric that was not reported by Sandoval et al. was the prevalence of myocardial injury using the hs-cTnI assay with sex-specific cutoffs. By extrapolating data from Tables 1 and 2 in their publication (1), we have estimated the prevalence of myocardial injury in the female population to be 46% (61/133) compared with 37% (66/177) in the male population. In another UK population, using the same hs-cTnI assay (Abbott Architect) with sex-specific cutoffs, the proportion of women with myocardial injury was >30% compared with <30% for men in the same population (3). Both of these studies suggest an increased prevalence of myocardial injury in the female population when using sex-specific cutoffs. The application of sex-specific cutoffs for high-sensitivity cardiac troponin is an evolving area; gaps still exist regarding the transition from a sensitive cTnI assay to a hs-cTnI assay when using an overall 99th percentile cutoff. Studies reported in Clinical Chemistry have indicated an increased prevalence of myocardial injury with an overall 99th percentile cutoff when centers have transitioned from cTnT to hs-cTnT (4) or between 2 sensitive cTnI assays (5). As our hospital transitioned from a sensitive cTnI assay to a hs-cTnI assay, we assessed both the prevalence of myocardial injury and hospital admissions from the emergency department (ED) in the 2 months before and 2 months after implementation of the hs-cTnI test.

For this analysis, data were obtained from all adult ED patients who presented from September 24 to November 24, 2014, at either the Hamilton General Hospital or the Juravinski Hospital and Cancer Centre and who had cTnI ordered, results given, and a final ED disposition recorded in the hospital information system (cTnI ED cohort, group 1). On November 25, 2014, both the Hamilton General Hospital and Juravinski Hospital and Cancer Centre replaced the Abbott Architect cTnI assay using the overall 99th percentile of 0.03 μg/L with the Abbott Architect hs-cTnI assay using the overall 99th percentile of 30 ng/L [obtained from the Canadian Laboratory Initiative on Pediatric Reference Intervals (CALIPER) Study and the Gutenberg Health Study]. From November 26, 2014, to January 26, 2015, data from all ED patients at both hospital sites who had hs-cTnI ordered, results given, and a final ED disposition recorded in the hospital information system were also collected (hs-cTnI ED cohort, group 2). After ethics approval, we compared the proportion of hospital admissions or ED deaths and the proportion of patients with myocardial injury (assessed using the peak cTnI concentration per ED patient) of the 2 time frames and, additionally, performed ROC curve analyses for each assay for hospital admission/ED death. We conducted our analyses using StatsDirect software and Analyse-it software.
For the 2 months before transitioning to the hs-cTnl assay, there were 5533 cTnl test results on 3204 patients [median age 68 years (interquartile range 55–81)] vs 6141 hs-cTnl test results on 3355 patients [median age 71 years (interquartile range 57–82)] after transitioning to the hs-cTnl assay (P < 0.01, Mann–Whitney test between ages). There was no significant difference in the prevalence of myocardial injury in group 1 (20.0%) vs group 2 (21.7%) (P = 0.09) using the overall 99th percentile cutoff. Furthermore, there was no significant difference in the proportion of hospital admission/ED death in group 1 (52.8%) vs group 2 (53.3%) (P = 0.73). The area under the curve was significantly higher for hs-cTnl (0.78; 95% CI 0.77–0.80) compared with cTnl (0.71; 95% CI 0.70–0.73) for hospital admission/ED death (Fig. 1).

Applying the overall 99th percentile cutoff, there was no difference in the prevalence of myocardial injury in females (group 1, 17.0% vs group 2, 19.3%; P = 0.08) or males (group 1, 23.1% vs group 2, 24.2%; P = 0.50). However, applying sex-specific cutoffs yielded a significantly higher proportion of myocardial injury in females (30.7%) compared with males (22.4%; P < 0.01) in group 2, which was also significantly higher than the prevalence of myocardial injury in females within group 1 (17.0%; P < 0.01).

Using an overall 99th percentile for hs-cTnl does not appear to increase the prevalence of myocardial injury or lead to further hospital admissions from the ED. However, implementation of sex-specific cutoffs will increase the prevalence of myocardial injury in the female population. Additional health outcome studies are needed to assess the impact of using sex-specific cutoffs in a general North American ED population.

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