LETTERS TO THE EDITOR

Comparison of Troponin T and Creatine Kinase-MB Fraction in Evaluating Cardiac Patients Postoperatively

Dr. Januzzi and colleagues in a recent study (1) published in the Journal suggest replacement of creatine kinase-MB fraction (CK-MB) with serum troponin testing for postoperative evaluation of the cardiac surgical patient. An important point to remember is the release pattern of these markers. Although the rise (CK-MB 4 to 8 h; CK-MB isoform 2 to 6 h; cardiac troponin I [cTnI] 4 to 6 h; cardiac troponin T [cTnT] 4 to 8 h) and peak (CK-MB 12 to 24 h; CK-MB isoform 18 h; cTnI 12 h; cTnT 12 to 28 h) are similar, the markers differ greatly in their return to normal (CK-MB 72 to 96 h; CK-MB isoform <24 h; cTnI 3 to 10 days; cTnT 7 to 10 days) (2).

Because of their prolonged elevation in the blood (up to 10 days), cardiac troponins may reflect a summation of preoperative, perioperative, and postoperative events, thereby limiting the ability to detect perioperative injury exclusively. In contrast, CK-MB and its isoforms have a more rapid clearance and return to normal more quickly, thus facilitating better timing of myocardial injury (3). The long circulating half-lives of the cardiac troponins make it difficult to distinguish new episodes of myocardial necrosis from earlier episodes (4,5). Hence, reinfarctions may be difficult to diagnose with the sole use of cardiac troponin assays, if the initial myocardial infarction (MI) occurred within a week of cardiac surgery. Subsequently, both CK-MB and cardiac troponins are concurrently useful and not mutually exclusive in the diagnosis of postoperative MI and reinfarction after coronary artery bypass graft surgery.

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REFERENCES

REPLY

We appreciate the comments of Drs. Chen-Scarabelli and Scarabelli. We agree that the distinctly different release kinetics of cardiac troponin MB (CK-MB) and troponin T (TnT) allow for complementary information in certain settings, particularly that of reinfarction. Indeed, it has been suggested that CK-MB be considered the marker of choice for the detection of reinfarction (1).

Though it was not the purpose of our study (2) to compare the prognostic role of cardiac markers among patients undergoing cardiac surgery soon after acute cardiac myonecrosis, among our cohort of 224 patients, 58 had TnT data available preoperatively. Of these, 26 had a level ≥0.10 ng/ml (at the time the conventional upper limit of normal for myocardial infarction [MI]). No clear association between elevated preoperative TnT and adverse postoperative outcomes was noted, nor did the elevation of preoperative TnT obscure the markedly powerful prognostic ramifications of marked elevations of TnT in the postoperative setting, which were independently prognostic, irrespective of preoperative TnT levels. This may be because the magnitude of TnT release identifying patients at risk for impending postoperative complications was so significant at each time point (with mean levels among complicated patients reaching nearly 9 ng/ml), compared to the generally lower levels of TnT released in the setting of most acute coronary syndromes. Given the small number of patients with elevated preoperative TnT in our study, however, it is impossible to characterize conclusively the comparative value of cardiac markers in this situation.

We agree that for the unusual circumstance of urgent cardiac surgery in the setting of a recent large acute MI, a cardiac marker with shorter serum existence such as CK-MB might be preferable for postoperative biochemical risk stratification. Finally, as we and others have demonstrated, the “expected” magnitude of TnT release following cardiac surgery varies between different procedures, and not all patients undergoing cardiac surgical procedures are expected to release large amounts of TnT (2–4). An example of this would be coronary artery bypass grafting without cardiopulmonary bypass, which is associated with significantly lower amounts of postoperative TnT release (3,4). In such patients, significant elevations of preoperative TnT might obviate the use of this marker for postoperative risk stratification. Nonetheless, for many, if not most patients undergoing cardiac surgical procedures, we believe that postoperative measurement of cardiac TnT affords superior prognostic information.

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Quantitative Relationship Between Severity of Pulmonary Hypertension and LV Diastolic Function Has Been Established

We read with interest the article entitled “Correlation of Left Ventricular Filling Characteristics With Right Ventricular Overload and Pulmonary Artery Pressure in Chronic Thromboembolic Pulmonary Hypertension” published in the July 17, 2002, issue of the Journal (1). Dr. Mahmud and his colleagues identified an impaired relaxation pattern of left ventricular (LV) filling in 39 patients with chronic thromboembolic pulmonary hypertension (CTEPH), with a mean pulmonary artery pressure >30 mm Hg, which normalized after successful pulmonary thromboendarterectomy. The objective (e.g., finding a quantitative relationship between the right ventricular [RV] pressure overload and the type of LV diastolic dysfunction) and some of the results of this study (only patients with severe pulmonary hypertension have an altered LV filling) resemble those published by our group in 2001 (2). We were disappointed by the investigators’ claim in the Objectives section of their Abstract that “a quantitative relationship between RV pressure overload and LV diastolic function has not been established.”

We examined by Doppler-echocardiography 120 patients with chronic pulmonary hypertension (of whom 12 patients had CTEPH), and we found that only in patients with a systolic pulmonary artery pressure (SPAP) ≥60 mm Hg is LV diastolic filling altered in the form of impaired relaxation pattern. In addition, we also found that the late systolic and early diastolic interventricular septum flattening occurs in 70% of patients with SPAP ≥60 mm Hg and only in 6% of those with SPAP <60 mm Hg. Therefore, a quantitative relationship between the severity of pulmonary hypertension and LV diastolic function was already established.

We would like to add that, in addition to abnormal geometrical configuration and motion of interventricular septum mentioned by Dr. Mahmud, other possible mechanisms of LV diastolic dysfunction in patients with severe pulmonary hypertension might be related to the presence of some degree of LV interstitial edema, which increases the LV wall stiffness and alters its normal diastolic filling (3) and the diastolic asynchrony found in the apical and lateral walls (4).

Finally, we believe that the findings of both reports complement each other and enlarge our understanding of the mechanisms of dyspnea in patients with severe pulmonary hypertension.

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REPLY

We would like to respond to the comments by Barasch et al. regarding our study “Correlation of Left Ventricular Filling Characteristics With Right Ventricular Overload and Pulmonary Artery Pressure in Chronic Thromboembolic Pulmonary Hypertension” recently published in the Journal (1). The study by Moustapha, Barasch, and colleagues (2) was published only a short time before we submitted our manuscript and so, regrettably, was not included in our references.

The objective of our study was to define a quantitative relationship between right ventricular (RV) pressure overload and left ventricular (LV) diastolic filling characteristics, measuring both as continuous variables (i.e., mean pulmonary artery pressure and E/A ratio). Furthermore, we only studied patients with chronic thromboembolic pulmonary hypertension (CTEPH) who were undergoing pulmonary thromboendarterectomy (PTE) so that we could assess the change in LV diastolic filling with resolution of an RV pressure overload state.

In the study by Moustapha et al. (2), the degree of pulmonary hypertension was defined only as a dichotomous variable (mild-moderate vs. severe). Pulmonary artery (PA) pressure was estimated by Doppler-echocardiography, not measured directly as in our study. Moustapha and colleagues reported that the group of patients with severe (systolic PA pressure >60 mm Hg) had lower E/A ratios than did the group with mild-moderate pulmonary hypertension (systolic PA pressure 40 to 60 mm Hg) (0.96 ± 0.37 vs. 1.34 ± 0.54, respectively [p < 0.05]). This relationship of low E/A ratio and severe pulmonary hypertension with associated interventricular septal distortion is, however, well recognized (3,4).