Periprocedural Myocardial Injury: Not a Benign Entity

Prasad et al. (1) present valuable insights into the prognostic influence of spontaneous myocardial infarction (SMI) and periprocedural myocardial infarction (PMI) following stent insertion in the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) trial. They concluded that PMI is of limited prognostic significance as it is predominantly a marker for procedural and baseline risk factors, and that, in contrast to SMI, it does not have independent prognostic significance. Given the differences in the pathophysiology, it is unsurprising that PMI and SMI have differential influence on prognosis at 1 year, but we wish to question some of the investigators’ interpretation of their data, as we feel that the significance of PMI has been understated.

Their principal conclusion about the lack of prognostic influence is based on the 1-year mortality hazard ratio for PMI being found to be not significant after adjustment for procedural and baseline characteristics. In this study, both PMI (3.2% vs. 0.8%, p < 0.0001) and SMI (5.0% vs. 0.8%, p < 0.0001) are associated with higher 30-day mortality without any significant difference between the 2 types of myocardial infarction (p = 0.27). No hazard ratio for 30-day mortality after the time-updated covariate-adjusted multivariate analysis is quoted, but the Kaplan–Meier graphs show similar curves for SMI and PMI in the early post-procedural period, suggesting that both types of myocardial infarction predict an adverse short-term outcome. Interestingly, stent thrombosis in patients that sustained PMI appears to have a worse prognosis than SMI without stent thrombosis.

The relevance of periprocedural enzyme elevation has been the topic of considerable debate, but it is increasingly clear that even small periprocedural troponin rises reflect new areas of myocardial necrosis on magnetic resonance imaging (2,3). Debate persists about the influence on prognosis of these small/moderate enzyme rises, but there is little doubt about the influence of large periprocedural enzyme rises—even in this study, the 1-year mortality for Q-wave PMI is extremely high at 27%, versus 17.3% for Q-wave SMI (p = 0.22).

Previous studies from patients undergoing nonemergency percutaneous coronary intervention, whether using creatine kinase-myocardial band or troponin definitions of PMI, have shown that PMI is an adverse long-term prognosticator (4–6). It is notable in this study of patients with acute coronary syndromes that a definition combining new elevation of >3× creatine phosphokinase or creatine kinase-myocardial band and electrocardiography criteria has been used for PMI. Troponin, a more sensitive and specific biomarker, is not used for the diagnosis of PMI but is used for the diagnosis of SMI.

We agree that PMI and SMI are different entities, and inevitably SMI would portend a worse prognosis. However, we feel that PMI with imaging evidence of myocardial necrosis represents an adverse event with long-term outcome implications. The long-term consequences of minor periprocedural enzyme rises are more uncertain and need more investigation, especially with the advent of the new universal definition of PMI using a troponin cutoff of >3× the 99th percentile (7).

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colleagues remain concerned that we have underestimated the importance of PMI. We agree with many of the suppositions in their communication (as clearly stated throughout our report [1]), namely that “even small periprocedural troponin rises reflect new areas of myocardial necrosis on MRI imaging,” and “there is little doubt about the influence of large periprocedural enzyme rises . . . .” We disagree, however, that “debate persists about the influence on prognosis of these small/moderate enzyme rises . . . .” Whereas the literature is replete with prior studies examining the influence of PMI, the largest studies in the stent era have shown that only truly large PMI as evidenced by elevation of peak creatine kinase-myocardial band to >8× or >10× normal or the development of new Q-waves significantly influences subsequent survival (2,3). Other studies that have used a low threshold for PMI have found that such infarctions have no long-term prognostic significance (4). Smaller enzyme elevations are a marker for more diffuse atherosclerosis (5), but do not have per se a great enough influence on left ventricular function or arrhythmogenesis to directly impair survival. Other studies have suggested that biomarker elevations after percutaneous coronary intervention are of no clinical relevance unless associated with a failed procedure or angiographic complications (6). We also agree with Dr. Lim and colleagues that the prognostic implications of troponin elevations after percutaneous coronary intervention are uncertain, with questionable clinical relevance (7).

The data from the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) trial are consistent with these previous studies; when considering all PMI (defined as creatine kinase-myocardial band >3× normal, the most widely used definition) in a time-updated, covariate-adjusted multivariable model, PMI was not an independent correlate of 1-year survival. However, large PMI (such as Q-wave myocardial infarction), was a predictor. Our study was not meant to minimize the role of large PMI, which clearly reduce early and late survival. Rather, most small PMI may be disregarded as clinically inconsequential, whereas SMI, even using a sensitive threshold for detection (any troponin elevation greater than the local laboratory normal), is a powerful independent predictor of subsequent mortality. These data emphasize the fact that SMI and PMI deserve different thresholds for diagnosis and should prompt suitably different therapeutic responses.

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Aortic Stiffness in Hypertrophic Cardiomyopathy

In the paper by Boonyasirinant et al. (1) in a recent issue of the Journal, there are serious problems in the use of pulse wave velocity (PWV) data as an index of aortic stiffness in hypertrophic cardiomyopathy (HCM).

The major technical problem is that whereas magnetic resonance imaging (MRI) can measure dimensions accurately, it can only measure flow at intervals of around 30 ms (Fig. 1 from Boonyasirinant et al. [1]), so that PWV, as distance travelled divided by time between wave feet, is likely to be less accurate, especially over very short distances (typically 12 to 13 cm in this report [1]) than when measured invasively or noninvasively by high fidelity manometry (2,3). This may account for unusually high variability of PWV measurements, especially in the HCM groups.

The most curious issue in this MRI report (1) is that normal subjects had lower values of aortic PWV (3.7 m/s, SD 0.9 m/s) than previously published for any normal group using invasive or noninvasive techniques (2,3). A previous MRI study (4) in normal (but obese) subjects of similar age gave PWV values of 6.8 m/s (SD 2.2 m/s), which is similar to that reported for HCM patients in the recent MRI report (1). Further, normal subjects (Fig. 5 from Boonyasirinant et al. [1]) had a blunted wave foot for the distal aortic wave, which is quite different from normal flow and pressure waves, from which PWV is usually calculated. High variability of PWV in HCM (mean 9.66 m/s, SD 6.43 m/s in 1 group and 6.51 m/s, SD 3.2 m/s in another) is not correctly represented in Figure 2 of Boonyasirinant et al. (1), nor are the confidence intervals, whose long “whiskers” do not appear at all. Data in the text and legend of Figure 2 (1) do not correspond to that shown in the figure.

These issues ought to be considered before the confident assertions of Boonyasirinant et al. (1) or Kuhl (5) are accepted that the MRI technique reveals “novel insights in vascular function from MRI” (1) or “an unidentified association unravelled by MRI” (5).