Myocardial Injury and Cardiac Troponin I Release After Off-Pump Versus On-Pump Coronary Surgery

In a recent study by Selvanayagam and colleagues aimed at evaluating reversible and irreversible myocardial injury in patients undergoing off-pump (OP-) versus on-pump (P-) coronary artery bypass surgery (CABG), the authors documented a similar incidence and magnitude of new irreversible myocardial injury in both groups, despite the greater release of cardiac troponin I (cTnI) after P-CABG. These findings raise questions about not only the clinical significance of increased cTnI levels after P-CABG, but also the higher degree of cardioprotection allegedly supplied by OP-CABG. The extent of cTnI release does not necessarily correlate with occurrence or magnitude of irreproducible myocardial injury. Although the inaccuracy of cTnI levels in the “quantification” of postsurgical myocardial infarction seems to be mainly related to the “washout phenomenon,” the lack of correlation between cTnI release and occurrence of irreversible myocardial injury may be ascribed to iatrogenic stressors, intrinsic to the P-CABG surgical approach, and not typically associated with enduring myocardial injury. Release of cTnI after P-CABG was detected in bypass patients in the absence of ischemic conditions, as well as associated with myocardial stunning (a common occurrence after P-CABG), and elevated preload, independently of cardiac ischemia. This release, potentially occurring in cardiopulmonary bypass together with that inevitably associated with surgical manipulations, may possibly explain higher, though benign, cTnI levels, detected in P-CABG versus OP-CABG. Reperfusion, whether pharmacologically or mechanically achieved, results in earlier and augmented cTnI release versus no reperfusion. This was substantiated by a recent study showing that P-CABG had graft patency rates significantly greater than OP-CABG, despite higher cTnI levels at 6 to 12 hours postoperatively. Additional studies are therefore needed to confirm whether an earlier and greater cTnI release in P-CABG patients should be paradoxically welcomed as a marker of earlier and greater reperfusion.

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References

Response
We welcome the interest of Dr Chen-Scarabelli et al in our article. They state, correctly, that release of cardiac troponin I (cTnI) after on-pump cardiac surgery does not necessarily imply irreversible myocardial injury. Indeed, as discussed in our article, this was a fundamental rationale for using cardiac magnetic resonance imaging to distinguish between reversible and irreversible myocardial injury in our randomized trial of on- and off-pump surgery. We previously reported in a series of 150 patients that the predominantly early pattern of cTnI release after on-pump cardiac surgery, with peak values observed within 6 to 12 hours after cardiopulmonary bypass, was inconsistent with true myocardial necrosis (in which case peak levels would not be seen for 48 to 72 hours) but was more likely to represent a cytoplasmic “washout” phenomenon. This phenomenon is likely to be exaggerated in on-pump surgery because of the increased cell wall permeability that accompanies the systemic inflammatory response of cardiopulmonary bypass and is, by far, the most likely explanation for the higher cTnI levels reported early after on-pump surgery by Khan and colleagues.

The suggestion by Dr Chen-Scarabelli et al that “an earlier and greater release of cTnI in on-pump surgery patients should be paradoxically welcomed as a marker of earlier and greater reperfusion” is not only counterintuitive but potentially harmful in the setting of coronary artery surgery, where strenuous efforts are made to minimize cardiac ischemia. Elevation of cardiac enzymes is already known to be associated with both a higher mortality rate and a significantly increased risk of subsequent adverse cardiac events after percutaneous coronary intervention, and more recently, a 5-fold increase in creatine kinase–MB early after coronary artery bypass surgery was reported to be associated with a significantly greater 1-year risk of death and myocardial infarction.

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