Cardiac Troponin I Predicts Short-Term Mortality in Vascular Surgery Patients

To the Editor:

We have read with interest the study by Kim and colleagues1 on the relationship between cardiac troponin I (cTnI) concentration and perioperative morbidity and 6-month mortality of patients undergoing vascular surgery. We remain unsure as to whether the authors when stating “postoperative surveillance with cardiac enzymes is not routinely performed in these patients” refer to creatinine kinase and its MB isoenzyme or to cTnI, a contractile protein, reported to be more specific for the diagnosis of myocardial infarction during surgery.2

We agree that screening with cardiac enzymes is not routinely performed after vascular surgery. However, it has been our practice to measure cTnI levels routinely, in all high- and intermediate-risk surgery groups, immediately after surgery and in the postoperative days 1 to 3 beginning in September 1995. The surveillance of this marker is not unique to our center and has been reported by other groups in France.3

Our unit has already published the results of a study consisting of 329 consecutive patients, undergoing infrarenal aortic surgery, included in a prospective manner and followed up for 1 year.4 We have limited our patient number to a specific surgical risk group with a standardized anesthetic protocol and 1-year follow-up visit. We have used the same immunoassay technique (Stratus fluorometric enzyme immunoassay; Dade Pharmaceuticals) as the authors used. Our study aim was to determine whether there is a cutoff value for cTnI that may predict cardiac complications perioperatively and at 1 year postoperatively, and we employed a receiver-operator characteristics curve for evaluation of the ideal discrimination value between the complicated and uncomplicated patient groups. We have found cTnI to correlate with immediate but not with short- to medium-term mortality.

From our experience, we find the perioperative incidence of 12% of cTnI >1.5 ng/mL much greater than ours, which was 8.2% (27 of 329 patients). It is not entirely clear whether the authors have considered all 9 perioperative deaths to be of cardiovascular origin, as this too, would contrast with our findings of only 5 patients having a clear cardiac cause of death. From our follow-up, mortality at 1 year is 1.2%, with a comparable cardiac morbidity of 12.6%. This may be related to the difference in perioperative incidence of cTnI >1.5 ng/mL.

Our last comment regards the aim of the study and the authors’ recognition “that the primary outcome of the main study has not yet been reviewed.” Kim et al1 used multivariate analysis with cTnI as a dichotomous variable and considered 6-month survival in patients with cTnI above and below 1.5 ng/mL. However, all the cause-specific mortality data were not available (7 of 18 deaths, not including those lost to follow-up) to them.

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Reply

We thank Doctors Godet and Arhanghelchi for their thoughtful comments; we can learn much from our French colleagues. While we did not formally evaluate the degree to which cardiac troponin I (cTnI) surveillance is routinely performed in hospitals in the United States, our informal discussions with several US hospitals revealed that cTnI surveillance is not routine. We should have made clear in our manuscript that we were referring to this informal evaluation. Further research is needed to learn whom to screen, and subsequently, how to use the results to improve patient outcomes.

Our higher incidence of cTnI >1.5 ng/mL may reflect our varied patient populations.1,2 Our study included patients who had abdominal and thoracic surgery. Indeed, 9 of 28 patients (32%) with elevated cTnI and 6 of 18 deaths (33%) occurred in patients who had thoracic aorta aneurysm repair. We agree that it would be helpful to have more detailed information on cause-specific mortality. Nonetheless, our findings using all-cause mortality are striking and may help inform clinical practice.

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