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EDITORIAL COMMENT

Surgery plays a fundamental role in the care of millions of patients worldwide, each year.1,2 Unfortunately, cardiovascular events occurring after surgery remain as one of the leading cause of morbidity and mortality after major non-cardiac surgery. The results of several large studies demonstrate that a higher than “normal” postoperative troponin concentrations in the absence of a clinical diagnosis of myocardial infarct (also known as myocardial injury after non-cardiac surgery, MINS) is an independent risk factor of short and long term morbidity and mortality.3–6

In this issue of the journal, Nyame et al. elegantly demonstrated in a large cohort of urological patients (n = 8,310) who underwent non-cardiac surgery that those subjects who had abnormal postoperative (30 days) troponin levels (3.6%) also had a significantly worse 5 years overall survival (70.6%) than those individuals with normal troponin concentrations (81.7%) and patients in whom troponins were not measured (90.4%). In my opinion, the reader should carefully consider that the cohort of patients involved in Nyame’s study represents a predominantly cancer population of patients in whom troponin levels were not routinely measured after surgery but as a result of changes in the patients’ medical conditions. In that regards, Nyame’s study’s patients significantly differ from subjects in the VISION trial in whom troponins were routinely measured postoperatively and in 8% of them MINS was detected. This fact does not underscore the relevance of Nyame et al. findings, but again it indicates that routine testing appears appropriate. I think that it is also important to consider that the authors of the manuscript published in this issue of the journal did not provide data on preoperative troponin values which can be increased in up to 40% of the patients undergoing major non-cardiac surgery.7,8 The relevance of an abnormal preoperative troponin resides in the fact that the risk of postoperative mortality is 2two-folds or higher than in patients with normal troponin concentrations.9

The treatment of patients with postoperative troponin elevations appears to be indicated. In the MANAGE trial, patients with MINS who were allocated to receive dabigatran had fewer major vascular than complications than those treated with placebo (hazard ratio: 0.72, 95% confidence interval: 0.55–0.93; P = 0.0115). It is worth considering that not every postoperative elevation in troponin is due to myocardial ischemia (ie, sepsis, pulmonary embolism, myocarditis, or cardioversion). Therefore, it is not clear if patients all with postoperative troponin elevations in Nyame’s study would have benefited from cardiovascular “optimized medical therapy” or dabigatran.

The findings of the study by Nyame et al. gives an insight on the long-term prognostic value of abnormal postoperative troponin measurements. They also open an opportunity to discover new biomarkers or more sensitive techniques (high sensitivity troponin assays) to detect small changes in troponins that could be used in patients “normal” troponins.

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References


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AUTHOR REPLY

If it were tracked by the CDC, mortality after non-cardiac surgery would rank among the top 4 causes of mortality in the United States.1 The aim of our study was to assess the association between postoperative troponin levels, and both short- and intermediate- term mortality in urologic patients undergoing major operations. This baseline information was needed as background information for a quality project to assess the role of routine troponin screening in our department. As such, there are a few inherent biases that were pointed out in this excellent