

Myocardial Injury Among Postoperative Patients: Where Is the Wisdom in Our Knowledge?

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The ability to detect myocardial injury has never been more advanced. With the availability of high-sensitivity troponin testing, microscopic evidence of myocyte death can now be detected, often within an hour or so of the inciting event. This, in turn, has facilitated quicker and more accurate identification and treatment of affected patients. However, these advances in detection have, in some cases, outstripped our understanding of the etiology and appropriate management of troponin elevation.

This dilemma is particularly apparent among patients undergoing noncardiac surgery. Annually, over 200 million of these surgeries occur worldwide, many in patients with elevated cardiac risk or overt cardiac disease. Naturally, physicians treating these patients are concerned that the stress of surgery will provoke myocardial injury. Since symptoms are often masked in the immediate postoperative period because of sedating or analgesic medications, many physicians rely on troponin testing to detect signs of myocardial injury. With the increased sensitivity of these assays, the prevalence of troponin elevation has increased, which currently affects nearly one in five postoperative patients. This knowledge, however, doesn't lend itself to a clear management strategy, particularly in those patients with no other objective evidence of infarction. To paraphrase T.S. Eliot, have we lost the wisdom in our knowledge?

In this journal issue, Cohn and colleagues summarize the current information around this phenomenon of myocardial injury after noncardiac surgery, or MINS.¹ Consistent with the literature, they define MINS as an acute rise and/or fall in troponin (above the assay's upper limit of normal) at any point in the 30 days following noncardiac surgery. Importantly, MINS is an umbrella term that can indicate either a myocardial infarction (MI) or nonischemic myocardial injury (NIMI). An MI exists if there are clinical signs of ischemia and/or objective evidence of infarction on imaging.

The authors found that MINS is highly prevalent (19.6%) and associated with both cardiac disease and perioperative hemodynamic stress. Between 2.9% and 13.5% of MINS patients experienced 30-day adverse cardiac events, with higher rates in patients with higher troponin elevations and/or accompanying ischemic symptoms. The authors suggested MINS man-

agement with standard cardio-protective medications, such as statins, beta-blockers, and angiotensin-converting enzyme inhibitors, or angiotensin receptor blockers. For those patients at low bleeding risk, they also suggested dabigatran based on the recent MANAGE trial. Finally, they noted that US cardiac society guidelines suggested no screening for MINS, while the European and Canadian guidelines advocated for screening in patients at high risk for cardiac complications.

The authors are to be congratulated for highlighting an important and vexing area of postoperative management. To date, it has been difficult to chart the best path forward for these patients because we could "see" the issue, thanks to increasingly sensitive troponin assays, but we didn't know what to do once we found it.

So what rationale exists to justify screening? Some advocate that the presence of MINS suggests a need for further imaging and closer monitoring of these patients to identify those with an MI. Indeed, several recent MINS registry studies have found that 20% to 40% of MINS patients had definitive evidence of MI.^{2,4} But what about those patients with troponin elevation and no evidence of MI? A small, propensity-matched, observational study of MINS patients, including those without MI, noted positive associations between cardioprotective medications, such as aspirin and statins, and cardiac outcomes.⁵ In addition, the MANAGE trial suggested that MINS patients, with or without evidence of an MI, receiving dabigatran had reduced vascular events without increased bleeding complications.⁶ With this growing base of evidence, the rationale for systematic screening for MINS appears to be standing on stronger footing.

As noted by the authors, the recommendations for MINS screening differ across three major cardiovascular societies. How does the practicing clinician make sense of this discordant advice? Differences often occur when the evidence is of moderate or low quality, which means guideline committees must make their own interpretations of equivocal findings. Another driver of discordant recommendations is the timing of the guidelines. Both the US and European guidelines were published in 2014, while the Canadian guidelines were published in 2017. Over time, experience with postoperative troponin testing increased, which may have influenced the Canadian guidelines. Finally, many members of the Canadian guideline writing committee were the ones conducting the various studies identifying management options for MINS patients, which may have guided their ultimate recommendation. Regardless, practicing physicians should collectively view the guidelines as acceptable "guardrails" to guide their practice.

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Received: May 14, 2020; **Revised:** May 14, 2020; **Accepted:** May 15, 2020

© 2020 Society of Hospital Medicine DOI 10.12788/jhm.3468

Selection of the appropriate strategy can then be tailored to the individual patient's risks and benefits, as well as available management options.

In this era of high-sensitivity troponin testing, we now possess an exquisite opportunity to “see” minute levels of myocardial injury among postoperative patients. Our growing ability to effectively act on this knowledge will enable us to make wise decisions with our patients to optimize their cardiac outcomes during the vulnerable postoperative period.

Disclosures: Dr Maddox disclosed current grant funding from the National Institutes of Health National Center for Advancing Translational Sciences (1U24TR002306-01: A National Center for Digital Health Informatics Innovation), current consulting for Creative Educational Concepts, Inc., and Atheneum Partners, and honoraria and/or expense reimbursement in the past 3 years from the University of Utah (May 2017), New York Presbyterian (September 2017), Westchester Medical Center (October 2017), Sentara Heart Hospital (Dec 2018), the Henry Ford Health System (March 2019), and the University of California San Diego (January 2020). He is currently employed as a cardiologist and the executive director of the Healthcare Innovation Lab at BJC HealthCare/ Washington University School of Medicine. In this capacity, he is advising Myia Labs, for which his employer is receiving equity compensation in the company. He is receiving no individual compensation from the company. He is also a

compensated director for a New Mexico–based foundation, the J.F. Maddox Foundation.

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