ORIGINAL CLINICAL RESEARCH REPORT

Association Between Hospital Postoperative Troponin Use and Patient Outcomes After Vascular Surgery

Paymon M. Azizi, MSc,*† Duminda N. Wijeysundera, MD, PhD,*†‡ Harindra C. Wijeysundera, MD, PhD,*†§ Peter C. Austin, PhD,*† Angela Jerath, MD, MSc,*†‡ Ahmed Kayssi, MD, MSc, MPH,*† and Dennis T. Ko, MD, MSc*†§

BACKGROUND: Acute myocardial injury after noncardiac surgery, which is most often symptomatically silent, is associated with increased mortality and morbidity. However, it is not known if routine postoperative troponin testing will affect patient outcomes.

METHODS: We assembled a cohort of patients who underwent carotid endarterectomy or abdominal aortic aneurysm repair in Ontario, Canada, from 2010 to 2017. Hospitals were categorized into high, medium, and low troponin testing intensity based on the proportion of patients who received postoperative troponin testing. Cox proportional hazards modeling was used to assess the association between hospital-specific testing intensity and 30-day and 1-year major adverse cardiovascular events (MACEs) while adjusting for patient-, surgery-, and hospital-level factors.

RESULTS: The cohort consisted of 18,467 patients from 17 hospitals. Mean age was 72 years, and 74.0% were men. Rates of postoperative troponin testing were 77.5%, 35.8%, and 21.6% in the high-, medium-, and low-testing intensity hospitals, respectively. At 30 days, 5.3%, 5.3%, and 6.5% of patients in high-, medium-, and low-testing intensity hospitals experienced MACE, respectively. Higher troponin testing rate was associated with lower adjusted hazard ratios (HRs) for MACE at 30 days (0.94; 95% confidence interval [CI], 0.89–0.98) and at 1 year (0.97; 95% CI, 0.94–0.99) for each 10% increase in hospital troponin rate. Hospitals with high-testing intensity had higher rates of postoperative cardiology referrals, cardiovascular testing, and rates of new cardiovascular prescriptions.

CONCLUSIONS: Patients undergoing vascular surgery at hospitals with higher postoperative troponin testing intensity experienced fewer adverse outcomes than patients who had surgery at hospitals with lower testing intensity. (Anesth Analg 2023;137:629–37)

KEY POINTS

- **Question:** Is increased postoperative troponin surveillance after vascular surgery associated with better patient outcomes?
- **Findings:** Patients who had vascular surgery at high-intensity troponin testing hospitals had fewer major adverse cardiovascular events (MACEs) after surgery.
- **Meaning:** Increased postoperative troponin testing practices may be associated with reduced adverse, possibly mediated through increased physician referrals and medication changes.

GLOSSARY

AAA = abdominal aortic aneurysm; **ACC** = American College of Cardiology; **ACEi** = angiotensin-converting enzyme inhibitor; **AHA** = American Heart Association; **ARB** = angiotensin receptor blocker; **CABG** = coronary artery bypass grafting; **CCS** = Canadian Cardiovascular Society; **CI** = confidence interval; **CIHI-DAD** = Canadian Institute for Health Information Discharge Abstract Database; **ESA** = European Society of Anaesthesiology; **ESC** = European Society of Cardiology; **HR** = hazard ratio; **ICD-10** = International Classification of Diseases Tenth Revision; **IQR** = interquartile range; **MACE** = major adverse cardiovascular event; **MANAGE** = Management of myocardial injury After NoncArdiac

From the *Institute of Health Policy, Management and Evaluation, University of Toronto, Toronto, Ontario, Canada; †ICES, Toronto, Ontario, Canada; ‡Department of Anesthesiology and Pain Medicine, University of Toronto, Toronto, Ontario, Canada; and §Schulich Heart Centre, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada.

Accepted for publication January 9, 2023.

Funding: Supported in part by a Canadian Institutes of Health Research (CIHR). Additional funding was provided by Foundation Grants (FDN 154333). D.T.K. is supported by the Jack Tu Chair in Cardiovascular Outcomes Research. P.C.A. is supported by a Mid-Career Award from the Heart and Stroke Foundation of Canada (HSFC). D.N.W. is supported in by the Endowed Chair in Translational Anesthesiology Research at St. Michael's Hospital and University of Toronto. D.N.W. and A.J. are supported by the Merit Award from the Department of Anesthesia at the University of Toronto. H.C.W. is supported by a phase 2 Clinician Scientist Award from the HSFC.

The authors declare no conflicts of interest.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's website (www.anesthesia-analgesia.org).

Reprints will not be available from the authors.

Address correspondence to Dennis T. Ko, MD, MSc, ICES, G1-06, 2075 Bayview Ave, Toronto, ON M4N 3M5, Canada. Address e-mail to dennis.ko@ices.on.ca.

DOI: 10.1213/ANE.0000000000006411

surGEry; **MI** = myocardial infarction; **MINS** = myocardial injury after noncardiac surgery; **ODB** = Ontario drug benefit; **OLIS** = Ontario Laboratory Information System; **PCI** = percutaneous coronary intervention; **POISE** = Perioperative Ischemic Evaluation Study; **RCRI** = Revised Cardiac Risk Index; **RPDB** = Registered Persons Database; **SD** = standard deviation; **TIA** = transient ischemic attack

Studies have suggested that 10% of patients undergoing major noncardiac surgery experience myocardial injury after noncardiac surgery (MINS), which is associated with increased morbidity and mortality.[1–4] These events are often asymptomatic and missed without routine postoperative troponin testing. In vascular surgery, perioperative myocardial infarction (MI) and myocardial injury after noncardiac surgery (MINS) occur more frequently as patients often have more cardiac risk factors. It is estimated that up to a fifth of vascular surgery patients will experience MINS, which is consequently associated with up to a 10-fold increase in mortality.[5–8] International perioperative guidelines have had different recommendations on how routine postoperative troponin testing should be used to detect postoperative troponin levels after noncardiac surgery. The Canadian Cardiovascular Society (CCS) guidelines and the joint European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA) guidelines recommend routine testing of patients using a risk-based approach.[9,10] American guidelines via the American College of Cardiology (ACC) and the American Heart Association (AHA), previously, specifically discouraged troponin screening; however, more recently, the AHA published a scientific statement recommending screening for high-risk patients.[11,12] These recommendations, when published, were mainly based on expert opinions; while there was substantial evidence that troponin elevations are associated with poor prognosis, the evidence for how to best manage patients with perioperative MI and MINS was more limited.[13,14] Despite more recent suggestions to increase perioperative testing, it has not yet been established if increased testing is actually associated with improving patient outcomes. This study focused on addressing this gap in knowledge by evaluating the association between the intensity of postoperative troponin testing at a hospital level, and patient outcomes of patients undergoing vascular surgery in Ontario, Canada. We hypothesized that patients managed at institutions that have clinical practices of more liberal postoperative troponin testing will experience fewer major adverse cardiovascular events (MACEs) (death, MI, or revascularization) compared to patients managed at institutions that use postoperative troponin testing less regularly.

METHODS

Design and Data Sources

We conducted a retrospective cohort study using routinely collected administrative and laboratory data in Ontario, Canada. The analytical and statistical plan was written and filed with the ICES institutional review board before data were accessed. The data are housed, linked using unique encoded identifiers, and analyzed at ICES, formally known as the Institute for Clinical Evaluative Sciences, in Toronto, Canada. Projects conducted within section 45 of Ontario's Personal Health Information Protection Act do not require review by a research ethics board and allow for collection, analysis, and reporting without the need for patient consent. Consequently, given that this body of work was conducted wholly under section 45, research ethics review was exempted, and informed consent was not required.

The primary databases accessed included: (1) Ontario Laboratory Information System (OLIS), which is an Ontario-wide repository of hospital and community laboratory information in Ontario; (2) Canadian Institute for Health Information Discharge Abstract Database (CIHI-DAD): a database containing administrative and clinical data on patients admitted to hospital, which was used to identify hospitalizations, surgeries, and comorbidities[15–17]; (3) Ontario Health Insurance Plan: a database containing all provincial physician fee-for-service billing; (4) Statistics Canada: census data used to identify neighborhood income data; and (5) Ontario Drug Benefit (ODB): a database of prescription claims for patients aged 65 and older.

Study Population

Eligible patients were aged 40 to 105 years undergoing carotid endarterectomies or abdominal aortic aneurysm (AAA) surgeries' repair (open and endovascular approach) from January 1, 2010, to December 31, 2017. We chose these surgeries because they represent relatively common vascular procedures that are performed across many hospitals in Ontario and allow for comparisons to previous studies that used the same cohort.[18] We limited our study date to 2017 in accordance with when CCS guidelines were published to minimize bias from changes in testing practices. We previously showed that troponin testing rates did not substantially change during the study period.[18] Surgeries were identified using Canadian Classification of Health Interventions codes in the CIHI-DAD. Patients who died intraoperatively were excluded as they would not have the opportunity to receive testing. We also excluded non-Ontario

residents and patients with missing information (postal code and surgery duration).

Exposure

We defined hospital-level troponin testing intensity as the proportion of patients who received postoperative troponin testing within 2 days after surgery (ie, the index procedure of carotid endarterectomy, endovascular AAA repair, or open AAA repair). Hospital-level troponin testing intensity was defined using time periods, where OLIS data were available at each site and applied throughout the study period. We assumed that hospital testing practices remained consistent throughout the study period. We excluded institutions with fewer than 50 procedures for each type of surgery during the OLIS reporting period. We used a 2-day threshold for testing as a surrogate for routine testing as CCS guidelines recommend daily testing for up to 48 to 72 hours after surgery. In the primary analysis, we categorized patients into low-, medium-, and high-testing intensity groups in accordance with the hospital they had their procedure, such that each group reflected roughly a third of all patients (Supplemental Digital Content 1, Figure 1, http://links.lww.com/AA/E224). In sensitivity analyses, hospital testing intensity was considered as a continuous variable (see Statistical Analyses).

Outcomes

The primary outcome was MACEs, which was defined as the composite of all-cause mortality, MI, and coronary revascularization (which included percutaneous coronary intervention and coronary artery bypass grafting [CABG]). Death was abstracted from the Registered Persons Database (RPDB). MI was defined as International Classification of Diseases Tenth Revision (ICD-10) codes I21 or I22. Stroke and transient ischemic attack (TIA) were defined as ICD-10 G45. CABG and percutaneous coronary intervention (PCI) were obtained from the CCI database. We assessed MACE at 30 days and 1 year after surgery. The secondary outcomes were all-cause mortality assessed at 30 days and 1 year after surgery. We also assessed rates of stroke and TIA at 30 days and 1 year after surgery. MI and stroke represented events that happened as a complication of the vascular surgery and rehospitalization of these events.

Processes of Care

We also evaluated the proportion of patients who received cardiovascular testing (eg, electrocardiogram, echocardiography, and cardiac stress test) and referrals (eg, cardiology, general internal medicine, and medical) within 60 days after surgery. Cardiology and general internal medicine referrals were defined as referrals from physicians for whom their main specialty was cardiology or general internal medicine, respectively. Medical referrals included physicians with a listed specialization of cardiology, internal medicine, geriatric medicine, nephrology, or endocrinology. Medication use was only available for patients aged 65 years of age or older. New prescriptions were defined as patients who filled a prescription for a medication within 90 days after the surgery and had not filled a prescription for the same category of medications in the 90 days before surgery.

Statistical Analyses

Baseline characteristics were described for each tertile of hospital testing intensity and were compared using the Kruskal-Wallis test and χ^2 test, as appropriate. Differences in cardiovascular testing, referrals, and medication changes were assessed using the χ^2 test. The Revised Cardiac Risk Index (RCRI) was computed as described previously.[18,19] Briefly, the RCRI consists of 6 criteria, and 1 point is received for each criterion met: high-risk surgery, history of ischemic heart disease, history of congestive heart failure, history of cerebrovascular disease, diabetes requiring preoperative insulin, and preoperative creatinine >2 mg/dL (176.8 μmol/L). We modeled the relationship between the outcome and predictor variables using a Cox proportional hazard model. We used a robust or sandwich estimator[20] to account for clustering within hospitals, and confidence intervals (CIs) were derived using Wald tests. The model adjusted for patient (age, sex, rural living status, income quintile, and RCRI), surgical (urgent surgery admission status, surgery duration, and surgery type), and hospital factors (hospital size and teaching status).

As a sensitivity analysis, we modeled the primary exposure, hospital testing intensity as a continuous variable. We first modeled association between hospital testing intensity and log hazard of MACE outcomes using restricted cubic splines with 3 knots and assessed if the relationship could be modeled linearly using a Wald χ^2 test. Accordingly, after confirming a linear relationship, we modeled the hospital testing intensity linearly. Additional sensitivity analyses were also performed where the cohort was restricted to one of the following subsets: AAA repair procedures (open and endovascular approach); hospitals during OLIS reporting dates; elective procedures; and procedures from 2015 onward. We restricted to AAA repair procedures (open and endovascular approach) as carotid endarterectomies represent a different population of patients. We restricted to only patients who had procedures when hospitals were reporting to OLIS. We also restricted to procedures from 2015 onward to assess a more contemporary cohort. All analyses were performed using SAS statistical software, version 9.4 (SAS Institute). For all analyses, a 2-sided P value <.05 was considered statistically significant.

RESULTS

Cohort Assembly

A total of 17 hospitals were included in this study. Of the hospitals included, 16 hospitals contributed to carotid endarterectomies, 15 to endovascular AAA repair, and all 17 to open AAA repair. We identified 25,237 patients who underwent vascular surgery in Ontario from January 1, 2010, to December 31, 2017. We excluded patients at hospitals that never reported laboratory information to OLIS (n = 6537, 25.9%) or patients at hospitals where <50 eligible procedures were performed during the study period (n = 394, 1.6%). We also excluded patients based on age <40 or >105 (n = 76, 0.3%), who died during surgery (n = 75, 0.3%), residency outside Ontario, and missing information (n = 213, 0.8%). After all exclusions, we identified 18,467 eligible patients for the study.

Baseline Characteristics

Patients' baseline characteristics stratified by high, medium, and low hospital testing intensity are represented in Table 1. Hospitals' testing intensity for 1 vascular surgery type generally correlated with testing intensity for other vascular surgery types (Supplemental Digital Content 1, Figure 1 and Table 1, http://links.lww.com/AA/E224). The proportion of patients who received postoperative troponin testing at high-, medium-, and low-testing hospitals was 77.5%, 35.8%, and 21.6%, respectively. Mean age was 71.7 (standard deviation [SD] 9.3), 71.2 (SD 9.3), and 72.0 (SD 9.0) years for patients at high-, medium-, and low-testing hospitals, respectively. A higher proportion of patients at high-testing hospitals had coronary artery disease (high 19.5%, medium 16.4%, and low 16.4%; $P < .001$). Overall, patients at high-testing hospitals had slightly higher RCRI scores. Among

Table 1. Baseline Characteristics

Characteristic	Low intensity n = 6090	Medium intensity n = 6054	High intensity n = 6323	*P* value
Demographics				
Age (y; mean ± SD)	72.0 ± 9.0	71.2 ± 9.3	71.7 ± 9.3	<.001
Male (%)	72.7	73.8	75.6	<.001
Rural resident (%)	32.0	19.2	9.3	<.001
Cardiovascular comorbidities (%)				
Hypertension	84.1	84.7	86.8	<.001
Coronary artery disease	16.6	16.4	19.5	<.001
Heart failure	14.2	13.7	14.4	.528
Stroke/transient ischemic attack	9.6	14.0	10.7	<.001
Atrial fibrillation	7.8	8.3	8.4	.395
Medical comorbidities (%)				
Chronic obstructive pulmonary disease	43.4	36.7	37.7	<.001
Diabetes	30.8	33.0	33.3	.007
Peripheral vascular disease	20.0	17.1	21.8	<.001
Anemia/blood disease	5.4	6.5	5.0	.001
Chronic kidney disease	3.6	3.5	4.0	.347
Revised Cardiac Risk Index (%)				
0	10.5	12.6	11.2	<.001
1	48.0	45.9	44.7	
2	28.0	28.9	29.4	
3+	13.5	12.7	14.8	
Surgical factors				
Urgent admission (%)	15.8	22.4	21.6	<.001
Duration of index surgery (min; IQR)	121–226	143–248	150–249	<.001
Surgery type (%)				
Carotid endarterectomy	33.1	40.7	36.3	<.001
Endovascular AAA repair	28.9	28.3	29.9	
Open AAA repair	38.0	30.9	33.8	
Hospital factors				
Proportion of patients tested (%)	21.6	35.8	77.5	<.001
Teaching hospital (%)	57.6	56.4	64.9	<.001
Hospital size (beds; IQR)	288–360	327–501	294–555	<.001
Prescription medication use before surgery (90 d; %)[a]	n = 4660	n = 4446	n = 4730	
Statin	57.8	58.2	59.3	.001
ACEi/ARB	49.8	48.9	50.0	.005
Beta-blocker	30.2	30.6	32.4	<.001
Clopidogrel/ticagrelor	20.8	14.2	16.2	<.001
Direct oral anticoagulants	3.4	3.7	3.5	.500

Abbreviations: AAA, abdominal aortic aneurysm; ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; IQR, interquartile range; SD, standard deviation.

[a]Data for prescription information were only available for patients aged ≥65 y.

patients aged ≥65 years of age, slightly more patients at high-testing hospitals were on cardiovascular medications, including statins, renin angiotensin aldosterone inhibitors, and beta-blockers. Over half of patients had their procedures performed at teaching hospitals (high 64.9%, medium 56.4%, low 57.6%; $P < .001$). There were small differences in the distribution of surgery type, as shown in Table 1. Carotid endarterectomies were most common in medium-testing intensity hospitals (40.7%), endovascular AAA repairs in high-intensity hospitals (29.9%), and open AAA repairs in low-intensity hospitals (38.0%).

Postoperative Referrals, Cardiovascular Testing, and Medication Use

Table 2 shows the proportion of patients who received postoperative referrals, cardiovascular testing, and new medication prescriptions by hospital intensity of troponin testing. Patients managed at high-testing intensity hospitals had more physician referrals within 30 days after their surgery. For example, more patients at high-testing intensity hospitals had postoperative cardiologist referrals (high 8.9%, medium 4.5%, low 4.9%; $P < .001$). There were also higher rates of internal medicine and referrals from other medical specialties at high-testing intensity hospitals. Rates of electrocardiogram and transthoracic echocardiography were higher in high-testing intensity hospitals (electrocardiogram: high 35.8%, medium 23.8%, low 23.3%; $P < .001$; echocardiography: high 11.6%, medium 10.5%, low 10.0%; $P = .012$); however, we noted no significant difference in the use of cardiac stress testing.

We assessed prescription medication in a subgroup of patients over 65 years of age. New prescription medications filled after surgery; in general, a higher proportion of patients managed at high-testing intensity hospitals were started on new medications. For example, a high proportion of patients were started on statin therapy (high 20.0%, medium 18.4%, low 16.9%; $P < .001$). We also observed higher rates of new prescriptions of renin angiotensin aldosterone inhibitors, beta-blockers, and antiplatelet agents (clopidogrel and ticagrelor) (Table 2).

Association Between Hospital-Level Troponin Intensity and MACE

Over 30 days after surgery, MACE occurred in 5.3%, 5.3%, and 6.5% of patients at high-, medium-, and low-testing intensity hospitals, respectively ($P = .004$; Table 3). After adjusting for patient-, surgical-, and hospital-level factors, there was a reduced hazard for MACE at 30 days for patients at high-intensity hospitals compared to low-intensity hospitals (hazard ratio [HR] 0.71; 95% CI, 0.57–0.89; Figure). Over 1-year follow-up, the effects persisted in a similar fashion. After adjustment, high-testing hospitals were associated with a lower hazard of 1-year MACE compared to low-testing hospitals (HR, 0.83; 0.72–0.94). Modeling hospital-level testing intensity linearly (as opposed to categorization of hospitals into tertiles) also yielded a significant association between testing intensity and 30-day MACE (HR for 10% increment in testing 0.94; 95% CI, 0.89–0.98) and over 1-year MACE (HR, 0.97; 95% CI, 0.94–0.99; Supplemental Digital Content 1, Table 2, http://links.lww.com/AA/E224). The full regression models can be found in Supplemental Digital Content 1, Table 3, http://links.lww.com/AA/E224.

Rates of 30-day all-cause mortality were 2.9%, 3.2%, and 2.7% in patients managed at high-, medium-, and low-intensity testing hospitals,

Table 2. Rates of Postoperative Testing, Referrals, and New Prescriptions

Outcome	Low intensity	Medium intensity	High intensity	*P* value
	n = 6090	n = 6054	n = 6323	
Postoperative testing (30 d; %)				
Electrocardiogram	23.3	23.8	35.8	<.001
Echocardiography	10.0	10.5	11.6	.012
Cardiac stress test	1.5	1.4	1.5	.863
Postoperative referrals[a] (30 d; %)				
Cardiology	4.9	4.5	8.9	<.001
Internal medicine	4.2	6.2	6.9	<.001
Medicine	10.1	10.8	14.7	<.001
	n = 4660	n = 4446	n = 4730	
New prescription[b] (90 d; %)				
Statin	16.9	18.4	20.0	<.001
ACEi/ARB	13.6	14.1	14.8	.005
Beta-blocker	13.3	11.2	15.5	<.001
Clopidogrel/ticagrelor	7.5	7.9	8.6	.035
Direct oral anticoagulants	2.4	2.2	2.7	.702

Abbreviations: ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.

[a]Cardiology and internal medicine referrals were defined as referrals from physicians for whom their main specialty was cardiology or internal medicine, respectively. Medical referrals included physicians with a listed specialization of cardiology, internal medicine, geriatric medicine, nephrology, or endocrinology.

[b]Medication data were only available for patients aged ≥65 y.

Table 3. 30-Day and 1-Year Outcome Counts

Outcome	Low intensity n = 6090	Medium intensity n = 6054	High intensity n = 6323	*P* value
30-d outcomes (%)				
MACE	6.5	5.3	5.3	.004
All-cause mortality	2.7	3.2	2.9	.266
Myocardial infarction	4.2	2.3	2.5	<.001
Percutaneous coronary intervention	1.3	0.7	1.0	<.001
Coronary artery bypass graft surgery	0.2	0.1	0.1	.554
Stroke/transient ischemic attack	1.4	1.6	1.5	.504
1-y outcomes (%)				
MACE	12.9	11.0	11.8	.004
All-cause mortality	7.4	7.2	7.4	.938
Myocardial infarction	6.9	4.3	4.4	<.001
Percutaneous coronary intervention	3.2	2.1	2.7	<.001
Coronary artery bypass graft surgery	0.8	0.9	0.6	.23
Stroke/transient ischemic attack	3.2	3.3	3.3	.838

Abbreviation: MACE, major adverse cardiac event.

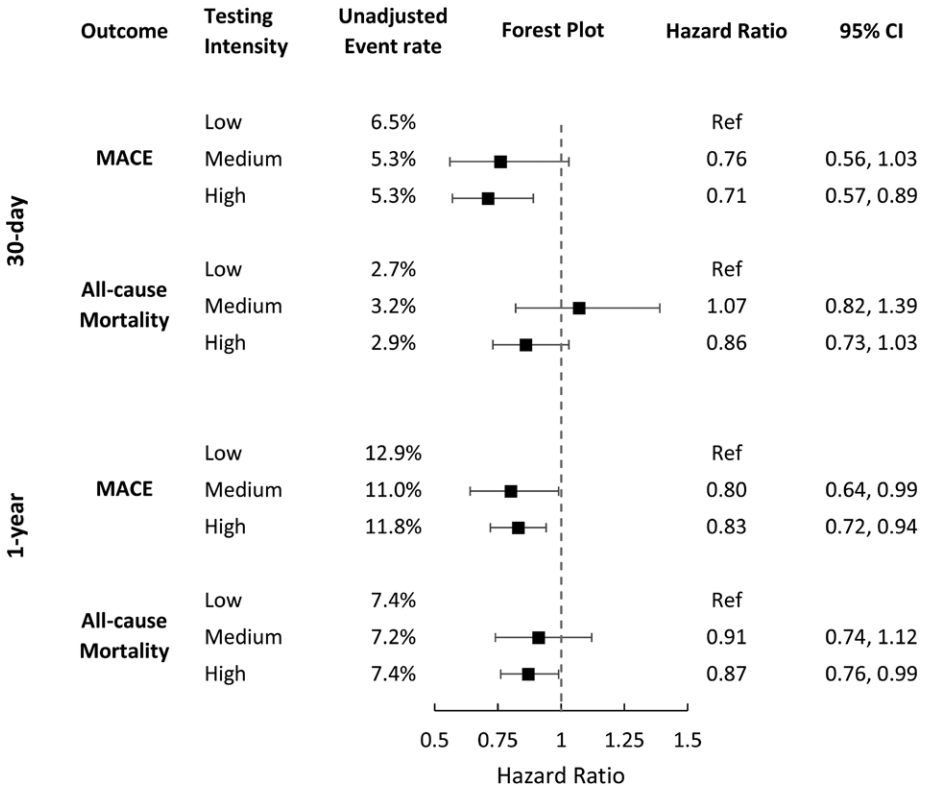

Figure. Association between hospital troponin testing intensity and patient outcomes. CI indicates confidence interval; MACE major adverse cardiac event.

respectively. After adjustment, there was no significant association between hospital-level testing intensity and 30-day all-cause mortality. However, at 1 year, high-testing intensity hospitals were associated with a reduction in all-cause mortality compared to low-testing intensity hospitals (HR, 0.87; 95% CI, 0.76–0.99).

Sensitivity Analyses

We conducted several sensitivity analyses for the composite MACE outcome (Supplemental Digital Content 1, Figures 2 and 3, http://links.lww.com/AA/E224). We limited analyses to AAA repair procedures (open and endovascular approach), excluded patients who had procedures during non-laboratory reporting periods, excluded patients undergoing urgent surgery, and restricted to a more recent sample (2015–2017). In general, for these analyses, we also saw similar reductions in MACE 30 days and 1 year for high-testing hospitals compared to low-testing hospitals.

DISCUSSION

In this study, we show that for vascular surgeries, high troponin testing intensity practices are associated with small improvements in postoperative

patient outcomes. In a cohort of patients undergoing vascular surgeries, we found that patients managed at hospitals with higher intensity of postoperative troponin testing had lower likelihood of MACE compared to patients managed at lower testing intensity institutions. At higher testing hospitals, we found increased cardiology and internal medicine referrals, cardiac testing with electrocardiograms, and increased prescriptions of evidence-based medications, potentially offering an explanation why improved outcomes were seen. It is important to recognize, however, that the effect size we observed was only modest; with a small absolute risk reduction, the clinical impact of increased testing practices is not clear. Our findings should stimulate future studies in improving our understanding on the role of routine postoperative troponin screening in noncardiac surgeries.

Given the observational study design, we cannot exclude the possibility that benefits observed in high-testing intensity hospitals were due to residual confounding due to difference in patient and hospital factors. However, we found that patients at high-testing intensity hospitals generally had a higher burden of measured cardiovascular comorbidities and had slightly higher RCRI scores compared with low-testing intensity hospitals. Furthermore, lower likelihood of MACE remained significant even after adjustment for patient, surgical, and hospital factors. Another reason to suggest our results were not due to confounding was in the examination of the individual MACE outcomes. If risks of patients were substantially different among the categories of testing intensity hospitals, one would expect to see a difference between outcomes (ie, stroke, mortality, and MI). However, our results were primally driven by differences in MI, which is the intended role of routine postoperative troponin that could lead early diagnosis and management. Moreover, the rates of MI at low-intensity hospitals may be an underestimate since troponin testing is required to make a diagnosis. Another contributing factor to the effect observed may be that hospital testing intensity may be in part also reflecting quality of care. For instance, despite that our study sample reflects relatively high-risk patients (via age and RCRI), only 21.6% of patients at low-testing intensity hospitals had postoperative troponin testing. This may indicate undertreatment of patients at high risk for MINS and perioperative MI, which may, in turn, result in poorer patient outcomes.

Nevertheless, given that we evaluated the intensity of troponin testing at an ecologic level, our study was not designed to elucidate the specific mechanism by which high-testing intensity hospitals were associated with better outcomes. At high-testing-intensity hospitals, we saw higher rates of physician referrals and cardiovascular testing. We also saw that a larger proportion of patients who were managed at high-testing institutions received newly prescribed cardiovascular medications, including statins, antiplatelet agents (clopidogrel and ticagrelor), angiotensin receptor blockers, and beta-blockers. In fact, previous studies have shown that early cardiology assessments and intensification of medication use in MINS patients are associated with reduction of adverse outcomes.[21,22] The difference in process of care changes (ie, referrals, testing, and medication changes) is also likely to represent a larger proportion of patients who had elevated postoperative troponin levels. Unfortunately, we did not have access to troponin values and were not able to assess this relationship.

Although elevated cardiac troponins after noncardiac surgery have been shown to be prognostically important for vascular and other noncardiac surgeries, it was previously unclear if increased testing or increased identification of patients with MINS is beneficial. International guidelines have differed in recommendation for routine postoperative testing because there is limited evidence that specifically shows that increased testing is beneficial. This is, in part, because it is not yet been clearly established how patients with MINS should be managed. Secondary evidence from the Perioperative Ischemic Evaluation Study (POISE) suggested that the use of statin and acetylsalicylic acid therapy is beneficial for patients with MINS.[23,24] The benefit of statin therapy in MINS patients has also been confirmed in another observational study. More recently, the Management of myocardial injury After NoncArdiac surGEry (MANAGE) study showed the use of the anticoagulant dabigatran conferred reduction in MACE events in patients with MINS.[25] However, the use of dabigatran in a noncardiac surgery cohort remains uncertain as the trial was stopped early and the primary outcome changed part-way through the trial. Additional research in this field is necessary.

Limitations

Several potential limitations of this study merit discussion. First, even though we found a relationship between increase troponin testing and improve MACE outcomes, we believe additional studies are needed to confirm our finding and to understand the incremental cost of this strategy. Second, we did not have laboratory data for the entire study duration and thus, calculated hospital testing rate based on available data. To address if this created substantial bias, we ran sensitivity analyses in which we restricted our sample to time periods when laboratory data were available and saw similar results to the primary analyses. Third, while our dataset captured troponin testing, we did not have the troponin values

available to us and, therefore, were not able to assess if patients met criteria for MINS and whether changes in care were made only among patients with MINS or perioperative MI. For example, we were unable to determine medication difference by hospital was due to troponin testing results or for other reasons. Fourth, despite conducting a hospital-level analysis and adjusting for a variety of patient, surgical, and hospital factors, observational study despite is still potential subjected to unmeasured confounding. We believe that future results in other setting would be important to confirm these findings. Finally, while we observed an association between hospital testing intensity and patient outcomes in a cohort of patients undergoing carotid endarterectomies and AAA repair procedures, it is not clear if this effect will also be observed in all vascular surgeries or in other noncardiac surgeries. Patients undergoing vascular surgeries often have more cardiac risk factors and have a higher incidence of perioperative MI and MINS. The benefit conferred by increased testing would thus be expected to be greater and easier to detect differences in outcomes.

CONCLUSIONS

In this study of vascular surgery patients, we showed that patients managed at high-intensity troponin testing hospitals experienced fewer adverse outcomes compared to patients managed at low-intensity troponin testing hospitals. This effect may in part be mediated through increased physician referrals and medication changes but may also reflect unmeasured factors. These results lend support toward a standardized approach to postoperative troponin testing for patients undergoing vascular surgery.

ACKNOWLEDGMENTS

This study was supported by ICES, which is funded by an annual grant from the Ontario Ministry of Health (MOH) and the Ministry of Long-Term Care (MLTC). ICES is an independent, non-profit research institute. Its legal status under Ontario's health information privacy law allows it to collect and analyze health care and demographic data, without consent, for health system evaluation and improvement. This document used data adapted from the Statistics Canada Postal Code Conversion File, which is based on data licensed from Canada Post Corporation, and/or data adapted from the Ontario Ministry of Health Postal Code Conversion File, which contains data copied under license from ©Canada Post Corporation and Statistics Canada. Parts of this material are based on data and/or information compiled and provided by the MOH and CIHI. The analyses, conclusions, opinions and statements expressed herein are solely those of the authors and do not reflect those of the funding or data sources. We thank IQVIA Solutions Canada Inc. for use of their Drug Information File.

DISCLOSURES

Name: Paymon M. Azizi, MSc.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
Name: Duminda N. Wijeysundera, MD, PhD.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
Name: Harindra C. Wijeysundera, MD, PhD.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
Name: Peter C. Austin, PhD.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
Name: Angela Jerath, MD, MSc.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
Name: Ahmed Kayssi, MD, MSc, MPH.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
Name: Dennis T. Ko, MD, MSc.
Contribution: This author provided substantial contributions to the conception or design of the work; and helped with analysis and interpretation of data for the work, drafting and revising the work critically for important intellectual content, and final approval of the version to be published.
This manuscript was handled by: Tong J. Gan, MD.

REFERENCES

1. Devereaux PJ, Chan MV, Alonso-Coello P, et al; Vascular Events In Noncardiac Surgery Patients Cohort Evaluation (VISION) Study Investigators. Association between postoperative troponin levels and 30-day mortality among patients undergoing noncardiac surgery. *JAMA*. 2012;307:2295–2304.
2. Devereaux PJ, Biccard BM, Sigamani A, et al; Writing Committee for the VISION Study Investigators. Association of postoperative high-sensitivity troponin levels with myocardial injury and 30-day mortality among patients undergoing noncardiac surgery. *JAMA*. 2017;317:1642–1651.
3. Botto F, Alonso-Coello P, Chan MV, et al. Myocardial injury after noncardiac surgery: a large, international, prospective cohort study establishing diagnostic criteria, characteristics, predictors, and 30-day outcomes. *Anesthesiology*. 2014;120:564–578.
4. Smilowitz NR, Redel-Traub G, Hausvater A, et al. Myocardial injury after noncardiac surgery: a systematic review and meta-analysis. *Cardiol Rev*.27:267–273.
5. Biccard BM, Scott DJA, Chan MV, et al. Myocardial injury after noncardiac surgery (MINS) in vascular surgical

patients: a prospective observational cohort study. *Ann Surg*. 2018;268:357–363.
6. Reed GW, Horr S, Young L, et al. Associations between cardiac troponin, mechanism of myocardial injury, and long-term mortality after noncardiac vascular surgery. *J Am Heart Assoc*. 2017;6:e005672.
7. Górka J, Polok K, Fronczek J, et al. Myocardial injury is more common than deep venous thrombosis after vascular surgery and is associated with a high one year mortality risk. *Eur J Vasc Endovasc Surg*. 2018;56:264–270.
8. Thomas KN, Cotter JD, Williams MJA, van Rij AM. Diagnosis, incidence, and clinical implications of perioperative myocardial injury in vascular surgery. *Vasc Endovascular Surg*. 2016;50:247–255.
9. Duceppe E, Parlow J, MacDonald P, et al. Canadian Cardiovascular Society guidelines on perioperative cardiac risk assessment and management for patients who undergo noncardiac surgery. *Can J Cardiol*. 2017;33:17–32.
10. Kristensen SD, Knuuti J, Saraste A, et al. ESC/ESA guidelines on non-cardiac surgery: cardiovascular assessment and management: the joint task force on non-cardiac surgery: cardiovascular assessment and management of the European Society of Cardiology (ESC) and the European Society of Anaesth. *Eur Heart J*. 2014;201435:2383–2431.
11. Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130:e278–e333.
12. Ruetzler K, Smilowitz NR, Berger JS, et al. Diagnosis and management of patients with myocardial injury after noncardiac surgery: a scientific statement from the American Heart Association. *Circulation*. 2021;144:e287–e305.
13. Horr S, Reed G, Menon V. Troponin elevation after noncardiac surgery: significance and management. *Cleve Clin J Med*. 2015;82:595–602.
14. Puelacher C, Lurati-Buse G, Singeisen H, Dang M, Cuculi F, Müller C. Perioperative myocardial infarction/injury after noncardiac surgery. *Swiss Med Wkly*. 2015;145:w14219.
15. Austin PC, Daly PA, Tu JV. A multicenter study of the coding accuracy of hospital discharge administrative data for patients admitted to cardiac care units in Ontario. *Am Heart J*. 2002;144:290–296.
16. Lee DS, Stitt A, Wang X, et al. Administrative hospitalization database validation of cardiac procedure codes. *Med Care*. 2013;51:e22–e26.
17. Porter J, Mondor L, Kapral MK, Fang J, Hall RE. How reliable are administrative data for capturing stroke patients and their care. *Cerebrovasc Dis Extra*. 2016;6:96–106.
18. Azizi PM, Wijeysundera DN, Wijeysundera HC, et al. Troponin testing after non-cardiac surgery in Ontario: an observational study. *CJC Open*. 2021;3:904–912.
19. Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999;100:1043–1049.
20. Lee EW, Wei LJ, Amato DA, Leurgans S. Cox-Type regression analysis for large numbers of small groups of correlated failure time observations. *Surviv Anal State Art*. 1992:237–247.
21. Hua A, Pattenden H, Leung M, et al. Early cardiology assessment and intervention reduces mortality following myocardial injury after non-cardiac surgery (MINS). *J Thorac Dis*. 2016;8:920–924.
22. Park J, Kim J, Lee S-H, et al. Postoperative statin treatment may be associated with improved mortality in patients with myocardial injury after noncardiac surgery. *Sci Rep.* [Internet] 2020;10:11616.
23. Foucrier A, Rodseth R, Aissaoui M, et al. The long-term impact of early cardiovascular therapy intensification for postoperative troponin elevation after major vascular surgery. *Anesth Analg*. 2014;119:1053–1063.
24. Devereaux PJ, Xavier D, Pogue J, et al. Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing noncardiac surgery: a cohort study. *Ann Intern Med*. 2011;154:523–528.
25. Devereaux PJ, Duceppe E, Guyatt G, et al. Dabigatran in patients with myocardial injury after non-cardiac surgery (MANAGE): an international, randomised, placebo-controlled trial. *Lancet*. 2018;391:2325–2334.