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Postoperative troponin release is associated with major adverse cardiovascular events in the first year after noncardiac surgery☆,☆☆

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ABSTRACT

Introduction: Troponin elevations after intermediate-to-high risk noncardiac surgery are common and can predict mortality. However, the prognostic value for early and late major adverse cardiovascular events (MACE) is less well investigated. The authors evaluated the relationship between postoperative troponin release and MACE in the first year after noncardiac surgery.

Methods: This observational cohort registry comprised data of patients aged ≥60 years undergoing intermediate-to-high risk noncardiac surgery between July 2012 and 2015, at the Erasmus University Medical Center, Rotterdam, the Netherlands. High-sensitivity troponin T was measured on day 1 to 3 after surgery. Peak troponin values were divided into four categories: <14 ng·L^{-1}, 14–49 ng·L^{-1}, 50–149 ng·L^{-1} and ≥150 ng·L^{-1}. The primary endpoint MACE was defined as the occurrence of myocardial infarction, angina, revascularization therapy or cerebrovascular accident in the first year after surgery. The incidence of MACE and all-cause mortality was calculated using Kaplan-Meier estimates. Cox regression was used to estimate risks for both endpoints.

Results: In total, 3085 patients were included for analyses and peak troponin elevation above 14 ng·L^{-1} was present in 1678 (54.4%) patients. The overall incidence for one-year MACE was 5.8% (3.4%, 6.1%, 10.4% and 40.6% per increasing troponin category) with adjusted HR (95% CI) 1.32 (0.85–2.06), 2.53 (1.42–4.53) and 10.24 (5.91–17.75) for the consecutive increasing categories. One-year mortality occurred in 14.6% and showed a similar stepwise increase with adjusted HR (95% CI) 1.25 (0.98–1.60), 2.39 (1.72–3.32) and 3.79 (2.60–5.54).

Conclusion: Our dataset demonstrates a graded relationship between postoperative troponin release and occurrence of MACE in the first year after intermediate-to-high risk noncardiac surgery.

1. Introduction

Over the years, many strategies have been explored to identify patients at risk for adverse cardiac outcomes after surgery. In 1990 Mangano et al. showed that perioperative myocardial ischemia is associated with an increased risk for cardiovascular events following noncardiac surgery [1]. To this day, focus on cardiac ischemia and its consequences remain a topic of interest, with troponin being the most sensitive marker for cardiac injury at this moment [2].

Routine troponin surveillance after intermediate-to-high risk noncardiac surgery is an accepted and upcoming tool to identify patients at risk for postoperative mortality [3]. However, proportions of patients with elevated troponin vary within the range of 6–73% in literature [4]. The pathophysiology and thereby therapeutic consequence of troponin elevations is still under debate in both literature and in daily clinical practice. In the general population, troponin release is related to stable coronary artery disease [5,6] and elevation is recognized as an independent predictor of adverse cardiovascular outcome [6]. In the surgical population, attention has been focused predominantly on mortality, and troponin release has been shown to be an independent predictor for early and late onset mortality after surgery [3,7,8]. However, the prognostic value for early and late major adverse cardiovascular events is less well investigated. Ekeloef et al. investigated the association between postoperative troponin release and major adverse cardiac events through a systematic review and meta-analysis, but was only able to include 3 articles on one-year follow up and all were in an orthopaedic surgical population [4]. In this cohort study, we evaluated the relationship between postoperative troponin release and the occurrence of major adverse cardiovascular events in a large cohort in the first year after surgery.

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☆☆ All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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2. Methods

2.1. Patient selection

Between July 2012 and July 2015, all consecutive patients aged 60 years and older, undergoing intermediate-to-high risk noncardiac surgery at the Erasmus University Medical Center (Erasmus MC), were registered in an ongoing clinical routine troponin registry [9,10]. Intermediate-to-high risk noncardiac surgery included the following operations: major abdominal, genitourinary, vascular, orthopaedic and neurologic surgery [11]. Expected length of surgery had to exceed at least 1 h and an expected postoperative in-hospital stay ≥24 h. This observational study was reviewed by the Medical Ethical Committee of Erasmus University, Rotterdam, the Netherlands who approved the non-interventional character of the present study. Patients were not subjected to acts, neither were any mode of behaviour imposed, otherwise than as part of their regular treatment and there was no need for extra blood sampling. Therefore, according to Dutch law, written informed consent for a patient to be enrolled in this study was not required. The study was conducted in compliance with the Helsinki declaration [12].

2.2. Postoperative troponin measurements

In this cohort registry, routine troponin measurement on day 1 to 3 after surgery were performed as standard clinical care in patients aged 60 years and older undergoing intermediate-to-high risk noncardiac surgery at the Erasmus University Medical Center. Fifth generation high-sensitivity troponin T assay was used (Elecsys® Troponin T hs (TnT-hs), Roche Diagnostics, Basel, Switzerland).

Myocardial injury was defined as a peak troponin elevation above the 99th percentile of 14 $ng \cdot L^{-1}$ in the first 3 days after surgery. For analysis, troponin levels were divided into four categories, as previously published [9,10]. The hsTnT thresholds were based on the manufacturers and previous determined postoperative prognostic values of the fourth generation assay [8]. The lowest threshold was the manufacturer's 99th percentile of a normal population, i.e. 14 $ng \cdot L^{-1}$ [13]. The second was based on the 0.03 $ng \cdot mL^{-1}$ threshold of the fourth generation assay's abnormal elevations, which is equivalent to 50 $ng \cdot L^{-1}$ of the fifth assay [13]. The highest threshold was extrapolated from the highest threshold in the Vascular Events In Noncardiac Surgery Patients Cohort Evaluation (VISION) study [8], which was 10 times the threshold of an abnormal troponin elevations, i.e. 140 $ng \cdot L^{-1}$ for the 5th generation, rounding off to 150 $ng \cdot L^{-1}$. The first category (0–13 $ng \cdot L^{-1}$) is considered normal, the second (14–49 $ng \cdot L^{-1}$) and third (50–149 $ng \cdot L^{-1}$) being minor and moderate myocardial injury respectively, and the fourth (≥150 $ng \cdot L^{-1}$) being approximately ten times the upper limit of the normal reference value of 14 $ng \cdot L^{-1}$.

2.3. Data collection and follow-up

Data were extracted from the electronic hospital patient information system. Patient characteristics include age, sex, type of surgery, past medical history, use of medication, length of hospital admission and discharge location. Past medical history focused on cardiovascular risk profile, including previous myocardial infarction, coronary artery disease, lower extremity arterial disease, renal failure (as reported in medical file or creatinine values >177 $\mu mol \cdot L^{-1}$), chronic heart failure and cerebrovascular accident. Follow-up data for the primary outcome were acquired from patients' own report on cardiovascular events either through questionnaires sent out by post 1 year after their initial surgery or, in case of a non-response, by telephone interview. In case of a total non-response, the electronic hospital patient information system was used as best alternative possible, and the date of last review or consultation was used to calculate the clinical follow-up time. All patient-reported events were checked in the electronic hospital patient information system if possible or (if permission was given) through contacting one's general practitioner or other hospitals. Information on survival status was obtained from the civil registries.

2.4. Outcome

The primary outcome was the occurrence of major adverse cardiovascular events (MACE) within one year after surgery, obtained as written above. MACE was defined as the composite endpoint of myocardial infarction, angina, coronary revascularization (both percutaneous coronary intervention (PCI) and coronary artery bypass graft (CABG)) or cerebrovascular accident. Hospital admission was required for all events. The secondary endpoint was all-cause mortality during one-year follow-up.

2.5. Statistical analysis

Categorical data are presented as numbers and percentages. Continuous data are described as mean with standard deviation or median with interquartile range if distribution was not normal. Baseline characteristics were compared between all four categories of troponins, using chi-square analyses for categorical data and Kruskal Wallis test for continuous data. Incidence of MACE and all-cause mortality at one year after surgery was

Table 1
Baseline characteristics.

hsTnT values ($ng \cdot L^{-1}$)					
	<14	14–49	50–149	≥150	P-value
	N = 1407	N = 1312	N = 267	N = 99	
Age (IQR)	66 (63–71)	71 (66–77)	73 (67–78)	73 (66–77)	<0.001
Gender (M)	694 (49.3)	864 (65.9)	181 (67.8)	70 (70.7)	<0.001
Hypertension	658 (46.8)	778 (59.3)	192 (71.9)	70 (70.7)	<0.001
Coronary artery disease	138 (9.8)	262 (20.0)	80 (30.0)	47 (47.5)	<0.001
Myocardial infarction	86 (6.1)	198 (15.1)	69 (25.8)	34 (34.3)	<0.001
Cerebrovascular accident	172 (12.2)	232 (17.7)	67 (25.1)	25 (25.3)	<0.001
PAD	105 (7.5)	183 (13.9)	46 (17.2)	21 (21.2)	<0.001
COPD	151 (10.7)	209 (15.9)	58 (21.7)	18 (18.2)	<0.001
Diabetes mellitus	248 (17.6)	341 (26.0)	115 (43.1)	38 (38.4)	<0.001
Current or history of heart failure	22 (1.6)	97 (7.4)	41 (15.4)	15 (15.2)	<0.001
Renal failure	23 (1.6)	145 (11.1)	88 (33.0)	27 (27.3)	<0.001
Revised cardiac risk index					<0.001
0 risk factors	758 (53.9)	470 (35.8)	38 (14.2)	19 (19.2)	
1 risk factor	459 (32.6)	445 (33.9)	83 (31.1)	25 (25.3)	
2 risk factors	150 (10.7)	236 (18.0)	75 (28.1)	23 (23.2)	
≥3 risk factors	40 (2.8)	161 (12.3)	71 (26.6)	32 (32.3)	
Medication use					
Beta blockers	418 (29.7)	552 (42.1)	145 (54.3)	53 (53.5)	<0.001
Statins	515 (36.6)	646 (49.2)	153 (57.3)	65 (65.7)	<0.001
ACE inhibitor	267 (19.0)	350 (26.7)	68 (25.5)	40 (40.4)	<0.001
Aspirin	337 (24.0)	473 (36.1)	123 (46.1)	51 (51.5)	<0.001
Oral anticoagulants	93 (6.6)	205 (15.6)	49 (18.4)	27 (27.3)	<0.001
Diuretics	316 (22.5)	419 (31.9)	127 (47.6)	41 (41.4)	<0.001
Insulin	71 (5.0)	149 (11.4)	71 (26.6)	20 (20.2)	<0.001
Type of surgery					
General	193 (13.7)	180 (13.7)	29 (10.9)	15 (15.2)	0.587
Orthopedics	205 (14.6)	190 (14.5)	35 (13.1)	5 (5.1)	0.062
Urology/gynecology	265 (18.8)	183 (13.9)	28 (10.5)	8 (8.1)	<0.001
Vascular	283 (20.1)	368 (28.0)	88 (33.0)	30 (30.3)	<0.001
Other	461 (32.8)	391 (29.8)	87 (32.6)	41 (41.4)	0.062
Emergency surgery	47 (3.3)	101 (7.7)	61 (22.8)	31 (31.3)	<0.001
Length of surgery (IQR)	211 (155–290)	202 (150–274)	196 (141–275)	179 (110–292)	0.003

Table 1 describes numbers of patients (%). PAD = peripheral arterial disease. COPD = chronic obstructive pulmonary disease. Renal failure is defined as creatinine values >177 $\mu mol \cdot L^{-1}$. Length of surgery is represented in minutes.

calculated using Kaplan-Meier estimates and compared using the Log-rank test. Time-dependent ROC curve and area under the curve (AUC) was calculated through the Kaplan Meier method for MACE at one-year follow-up [14,15]. Both univariate and multivariate COX regression hazard ratios were calculated for the occurrence of all-cause mortality and MACE for all different categories of troponins. Peak troponin levels below 14 ng·L^{-1} was used as the reference category. Multivariable analyses included sex, age, coronary heart disease, diabetes, cerebrovascular accident, renal failure, chronic heart failure and emergency surgery. Sensitivity analyses were performed between patients who could be reached through questionnaires by post or telephone, and patients whose follow-up data was obtained from the electronic hospital patient information system. A two-sided P value of <0.05 was considered significant. Analyses were performed using IBM SPSS 21.0 statistical software (SPSS Inc., Chicago, Illinois, USA). R Studio Version 1.0.153 was used for survival analysis and time-dependent AUC analysis [15,16].

3. Results

3.1. Study population

A total of 3085 (72.5%) of 4253 consecutive patients undergoing surgery fulfilled the inclusion criteria of at least one postoperative troponin measurement (see online appendix A). Baseline characteristics are presented in Table 1. In total, 1678 (54.4%) patients had an elevated peak troponin measurement of 14 ng·L^{-1} and above during the first 3 days after surgery. Of these patients, 78.2%, 15.9%, 5.9% had peak troponin levels of 14 to 49 ng·L^{-1}, 50 to 149 ng···L^{-1} and ≥150 ng·L^{-1}, respectively. With increasing levels of peak troponin, patients were of older age, male sex was predominant and more patients had an extensive history of cardiovascular disease.

3.2. Major adverse cardiac event

At one year after surgery 2634 patients were still alive and at least one year of follow-up was obtained for 2448 patients, of whom 2135 (87.2%) were contacted through written questionnaire or by phone. Of the remaining patients and patients who died, information on follow-up was extracted from the hospital's electronic patient data management system.

The primary outcome one-year MACE occurred in 178 (5.8%) patients of whom 135 (75.8%) with troponin values of 14 ng/L and above. In total, 76 patients experienced a myocardial infarction, 68 patients had angina requiring hospitalization, 54 patients had a cerebrovascular accident and 49 patients underwent revascularization therapy (see online appendix B). MACE was more common in patients with higher troponin levels after surgery (3.4%, 6.1%, 10.4% and 40,6% for increasing levels of troponin (P < 0.01, Fig. 1, Table 2A)). Cumulative incidence of MACE showed the same relationship across the different types of surgery (online appendix C). The AUC was 0.70. Time to event was inversely related to peak troponin after surgery (Table 2). The majority of the events occurred in the first few months after surgery with approximately 50% of all events within index hospitalization (Table 2). Adjusted hazard ratios for occurrence of 1-year MACE were: aHR 1.32 (95% CI, 0.85–2.06), 2.53 (95% CI, 1.42–4.53) and 10.24 (95% CI, 5.91–17.75) for increasing levels of peak troponin (Table 2).

Sensitivity analyses were performed to assess the effect of patients who responded through questionnaire compared to follow-up through the electronic patient record system: aHR 0.78 (95% CI, 0.40–1.55), 2.00 (95% CI, 0.78–5.07) and 5.43 (95% CI, 2.01–14.66) in the responders group compared to 1.87 (95% CI, 1.02–3.45), 2.50 (95% CI, 1.16–5.40) and 14.95 (95% CI, 7.21–31.02) for non-responders throughout increasing levels of peak troponin elevation.

3.3. All–cause mortality

In total, 451 (14.6%) patients died within the first year after surgery and median time to death was 99 (IQR, 35 - 222) days. Cumulative incidence for mortality is shown by Kaplan Meier estimates at 6 months and 1-year follow-up (Fig. 2, Table 2A). In the reference category of peak troponin values <14 ng···L^{-1}, cumulative incidence was 10,3% which

Table 2
Incidence, hazard ratios, median time to event and discharge in days (IQR, 25th–75th percentile, Tukey's Hinges) for MACE and all-cause mortality in the first year postoperative.

A

hsTnT values (ng·L^{-1})	<14	14–49	50–149	≥150	P-value
	N = 1407	N = 1312	N = 267	N = 99	
MACE at 1 year (%)	44 (3.4)	73 (6.1)	25 (10.4)	36 (40.6)	<0.001
HR (95% CI)[1]	1	1.70 (1.12–2.58)	3.42 (2.04–5.75)	14.83 (9.13–24.10)	
HR (95% CI)[2]	1	1.32 (0.85–2.06)	2.53 (1.42–4.53)	10.24 (5.91–17.75)	
Median time to event (IQR)	160 (48–254)	76 (13–251)	10 (3—22)	3 (1—11)	
In-hospital MACE (%)	26 (1.8)	32 (2.4)	17 (6.4)	26 (26.3)	<0.001
Median time to event (IQR)	3 (2–5)	9 (4–14)	8 (3–18)	2 (1–4)	
Mortality at 1 year (%)	144 (10.3)	187 (14.3)	74 (27.9)	46 (46.5)	<0.001
HR (95% CI)[1]	1	1.34 (1.07–1.68)	2.62 (1.96–3.49)	4.50 (3.19–6.35)	
HR (95% CI)[2]	1	1.25 (0.98–1.60)	2.39 (1.72–3.32)	3.79 (2.60–5.54)	
Median time to event (IQR)	162 (73–255)	101 (42–224)	54 (14–183)	27 (7–63)	

Panel A describes events and one-minus Kaplan-Meier estimates (%) of MACE and all-cause mortality in patients. P-values are calculated through Log-Rank analyses.
HR[1]: hazard ratios are calculated with a Univariate Cox regression model.
HR[2]: hazard ratios are calculated with a Multivariate Cox regression model.
Median time to event is represented in days (IQR).
For in hospital MACE and discharge location absolute numbers (%) are presented.

B

hsTnT values (ng·L^{-1})	<14	14–49	50–149	≥150	P-value
	N = 1407	N = 1312	N = 267	N = 99	
Length hospital stay (median(IQR))	6 (4–9)	7 (4–13)	11 (6–19)	11 (6–23)	<0.001
Discharge location (%)					<0.001
Home	1262 (89.7)	1064 (81.1)	177 (66.3)	55 (55.6)	
Nursing home	81 (5.8)	155 (11.8)	53 (19.9)	13 (13.1)	
Peripheral hospital	41 (2.9)	37 (2.8)	11 (4.1)	6 (6.1)	
In hospital death	9 (0.6)	29 (2.2)	23 (8.6)	25 (25.3)	
Unknown	14 (1.0)	27 (2.1)	3 (1.1)	0 (0.0)	

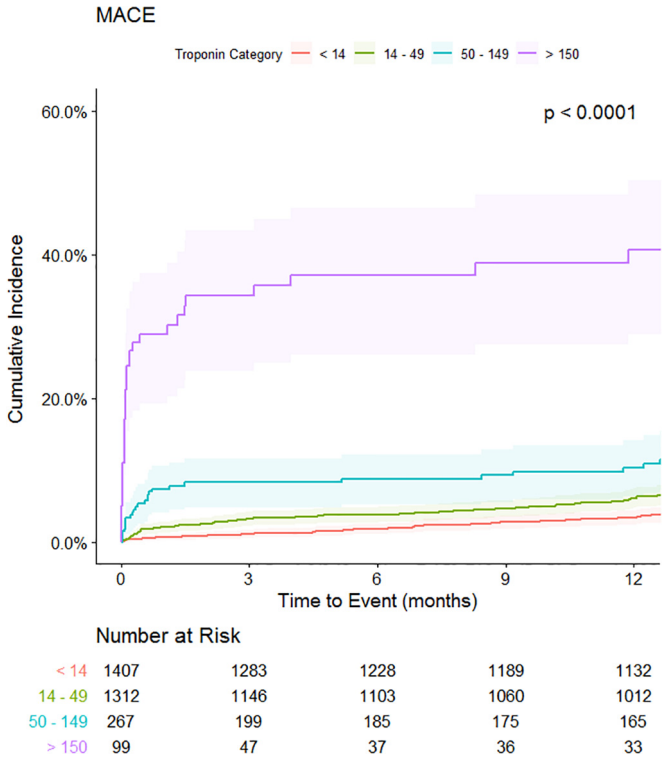

Fig. 1. Kaplan Meier of MACE. Estimates calculated using the one-minus Kaplan-Meier approach. The lines represent the four different peak troponin categories: <14 ng·L^{-1} [coral], 14–49 ng·L^{-1} [green], 50–149 ng·L^{-1} [blue] and >150 ng·L^{-1} [purple].

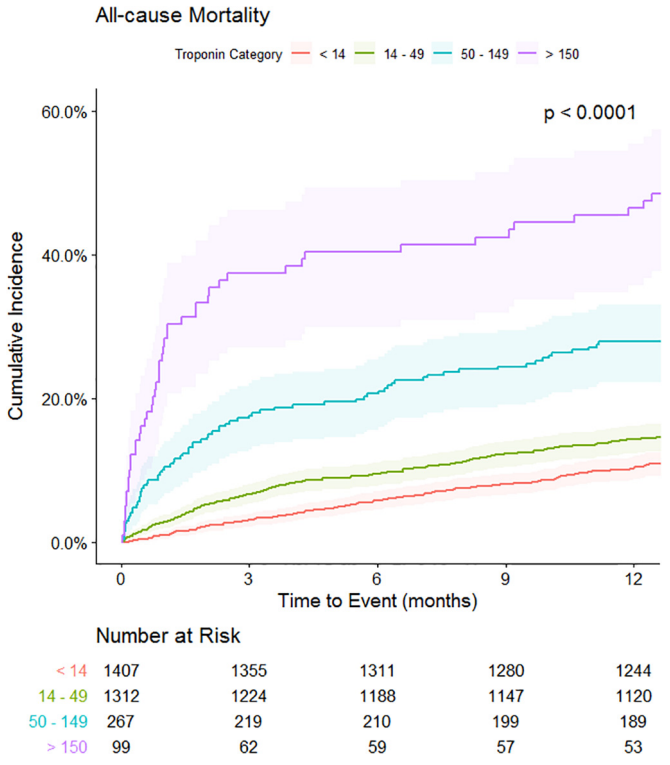

Fig. 2. Kaplan Meier of all-cause Mortality. Estimates calculated using the one-minus Kaplan-Meier approach. The lines represent the four different peak troponin categories: <14 ng·L^{-1} [coral], 14–49 ng·L^{-1} [green], 50–149 ng·L^{-1} [blue] and >150 ng·L^{-1} [purple].

increased to 14.3%, 27.9% and 46.5% for higher troponin values (P < 0.01, Table 2). After adjustment, hazard ratios remained significant: aHR 1.25 (95% CI, 0.98–1.60), 2.39 (95% CI, 1.72–3.32) and 3.79 (95% CI, 2.60–5.54) for increasing levels of peak troponin elevation (Table 2).

3.4. Discharge location

The majority of patients were sent home after discharge across all categories of peak troponin. An inverse relationship with increasing peak troponin can be seen, with 89.7% for values <14 ng·L^{-1} and 55.6% for values >150 ng·L^{-1} (Table 2B). In-hospital death and discharge to a nursing home was more often seen in patients within the higher troponin categories.

4. Discussion

This study shows that patients with troponin elevation after intermediate-to-high risk non-cardiac surgery have an increased risk for MACE and all-cause mortality in the first year following surgery. There is a graded association between peak troponin elevation and incidence of both MACE and death. Furthermore, time to event is inversely related to increasing levels of troponin with approximately half of all cardiovascular events occurring after discharge.

More than half of the patients in our study had an elevated peak troponin measurement above the 99th percentile. The therapeutic consequences of postoperative troponin elevations are frequently debated amongst clinicians. A systematic review and meta-analysis on postoperative troponin release published in 2016 concluded that higher troponin concentrations were associated with increased incidence in death and major adverse cardiac events within one-year after surgery [4]. These increased risks are in line with our findings regarding both adverse outcomes. The distinctive feature of our study is the addition of the graded association for MACE with increasing levels of peak troponin in contrast to troponin elevation as a dichotomized value below or above the 99th percentile threshold for the specific assay used. The three studies that reported on the association between postoperative troponin elevation and 1-year MACE, with varying outcome definitions used for adverse cardiac events, were limited to patients undergoing orthopaedic surgery [17–19]. In our study, we included all types of intermediate-to-high risk noncardiac surgery to generate results for a general surgical population. Furthermore, different cut off-values of different troponin assays are used which limit direct comparisons. In our overall cohort, patients had a 5.8% risk of developing MACE. In the literature, the proportion of patients experiencing MACE at one year after noncardiac surgery varies between 4% and 32% [4]. Our results show a clear stepwise increase in incidence with increased levels of troponin.

Association between troponin elevation and occurrence of all-cause mortality within one year after surgery showed the same relationship with increasing levels of peak troponin expected based upon previous work [4,20–23]. Similar to our results, investigators of the VISION study group and van Waes and colleagues found increasing hazards for moderate and major peak troponin elevation on early postoperative complications [20,22]. The VISION study group primarily focused on the association between different levels of troponin elevation and 30-day mortality. Our study adds that troponin shows to be an independent predictor for long-term cardiovascular events for troponin elevations above 50 ng/L. In a more recent paper, the VISION trial showed that only 11% of subjects had troponin elevation of a non-ischemic etiology [22], suggesting an underlying cardiovascular disease. Van Waes reported that in 38% of patients with postoperative myocardial injury receiving cardiac consultation postoperatively, an intervention was initiated (e.g. change in medication, coronary angiography). In our data, approximately half of all events occurred within index hospitalization. Median time to event in the highest troponin category (≥150 ng·L^{-1}) was 3 days. Thence, troponin elevation itself is likely to be part of these events rather than being a predictor for the occurrence of MACE

in the future. In this group it could therefore be suggested to consider troponin elevations as a symptom rather than a prognostic factor. For patients who experience MACE with peak troponin elevation between 50 and 149 $ng \cdot L^{-1}$, median time to event was 10 days after surgery and hazards remained throughout multivariate analysis. In these patients, troponin elevations could be assumed to be of prognostic value. If so, this particular group, for whom cardiac consultations are frequently requested, can be a target group for additional preventive strategies [4,20]. Recently, investigators of the MANAGE trial published on management of patients with postoperative troponin elevation. [24] They were able to show a relative reduction in incidence of major vascular events with 27% by adding dabigatran to patients' medical treatment. The results of this study as well as both of the VISION and MANAGE trial, suggest a relationship between troponin elevation and atherosclerotic cardiovascular diseases, albeit in opposite ways.

Troponin has been well identified as a diagnostic and prognostic tool for adverse cardiovascular events in patients with established stable coronary artery disease [6,25,26]. Current coronary arteriosclerotic research suggests that the pathophysiological process underlying troponin elevation in stable coronary artery disease are repetitive cycles of subclinical rupture and/or erosion with subsequent microembolisation of non-calcified plaques and repair and that 60% of ischemic coronary events are precipitated by plaque rupture followed by thrombosis [6,27].

Additionally, adding troponin to variables of established risk score improves prediction of cardiovascular death and cardiovascular disease. We endorse the suggestion that postoperative troponin release can be used in risk stratification [3,4,28,29].

4.1. Limitations

There are several limitations to this study. First, while monitoring troponin elevations, preoperative troponin values were not measured routinely, so delta troponin could not be calculated.

Secondly, 922 (35%) patients could not be reached through written or telephone questionnaire, and their information on follow-up was only limited to our University Hospital. Third, patients' events were self-reported. However, we asked for events for which a hospital admission was required to improve the quality of the reported events and moreover, self-reported events have been checked. Furthermore, we did not have information on cause of death, which could have added valuable information. We also had to exclude approximately one-fourth of patients who enrolled in this study due to poor protocol adherence for routine troponin measurement in the beginning. After implementing electronic laboratory requisitions, protocol adherence improved. Sensitivity analyses on peak troponin levels showed similar results after implementation of electronic orders. Last, the present study is a single center cohort registration in a University Hospital with a corresponding patient population, which may limit generalizability.

5. Conclusion

This paper describes a set of data providing information on long-term follow-up in patients with myocardial injury after intermediate-to-high risk noncardiac surgery. While many studies have demonstrated a relation between troponin elevation and mortality, little is known about the effect on cardiovascular morbidity. Our results showed a significant association between postoperative troponin release and MACE in the first year after intermediate-to-high risk noncardiac surgery and a higher risk for (future) cardiovascular events and consequent morbidity in patients with moderate and high troponin elevations.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2019.01.035.

Abbreviations

MACE	Major Adverse Cardiovascular Events
hsTnT	High-sensitivity troponin T

Competing interests

All authors declare no competing of interests.

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