Cardiac complications following non-cardiac surgery: A call for interdisciplinary and interprofessional collaboration

Christian Mueller, Mirjam Pargger

Cardiovascular Research Institute Basel (CRIB) and Department of Cardiology, University Hospital Basel, University of Basel, Switzerland

Related article
by Polok et al.

Correspondence to:
Prof. Christian Mueller, MD, Cardiovascular Research Institute Basel and Department of Cardiology, University Hospital Basel, Petersgraben 4, CH-4031 Basel, Switzerland,
phone: +41 61 328 65 49,
e-mail: christian.mueller@usb.ch

DOI: 10.33963/v.phj.101016

Received:
May 30, 2024

Accepted:
May 30, 2024

Early publication date:
June 5, 2024

We wish to congratulate Polok and colleagues [1] for their important work on self-reported functional capacity in the MET-REPAIR trial, a large multicenter prospective cohort study, which included patients aged ≥45 years and undergoing elective, elevated-risk non-cardiac surgery from June 2017 to April 2020. In that study, the authors demonstrated that the predictive value of self-reported functional capacity was restricted to the sites that could reliably detect patients with elevated cardiac troponin (cTn) concentration postoperatively due to their institutional active surveillance program. Accordingly, self-reported functional capacity should continue to complement other clinical information and high-sensitivity cTn (hs-cTn) concentration in preoperative risk assessment in high-risk patients undergoing major non-cardiac surgery [1–5].

Based on the accumulating evidence generated from large prospective cohort studies with central adjudication of cardiac complications following major non-cardiac surgery, the European Society of Cardiology (ESC) developed guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery. These recent guidelines, which were endorsed by the European Society of Anaesthesiology and Intensive Care (ESAIC), summarize the key messages for clinicians [1–10]. First, at least in part due to intra- and postoperative analgesia and anesthesia, the vast majority of patients developing acute myocardial injury associated with non-cardiac surgery do NOT report typical ischemic symptoms [1–5]. Therefore, these events are missed in hospitals that restrict the measurement of hs-cTn to patients experiencing acute chest discomfort. Unfortunately, this is still common practice in the majority of hospitals worldwide. The perioperative community needs to massively increase its efforts to heighten the awareness among physicians, including cardiologists, that the reliable detection of these prognostically highly relevant (deadly) events requires active surveillance using serial hs-cTn measurements [1–10]. Second, a preoperative measurement of hs-cTn is mandatory to reliably detect acute myocardial injury as a result of surgery and differentiate it from chronic myocardial injury that preexisted preoperatively and has nothing to do with surgery (Figure 1). Accordingly, perioperative myocardial infarction/injury (PMI) should only be diagnosed, if the postoperative hs-cTn concentration has shown a relevant increase above the preoperative concentration. Third, the magnitude of the increase in hs-cTn above the preoperative concentration required is a matter of ongoing debate and research [1–10]. The current ESC guidelines recommend an increase of at least the upper limit of normal above the preoperative concentration of the individual hs-cTnT/I assay used. This definition is simple, easy to use, and scientifically preferable to this employed previously as it incorporates intraindividual differences in preoperative hs-cTnT/I concentrations as well as analytical differences between hs-cTnT/I assays [10]. Fourth, PMI is not a homogenous disease but a working diagnosis with several different possible underlying etiologies. These include myocardial infarction, but also acute heart failure, tachyarrhythmias, takotsubo cardiomyopathy, and even primarily non-cardiac disorders like

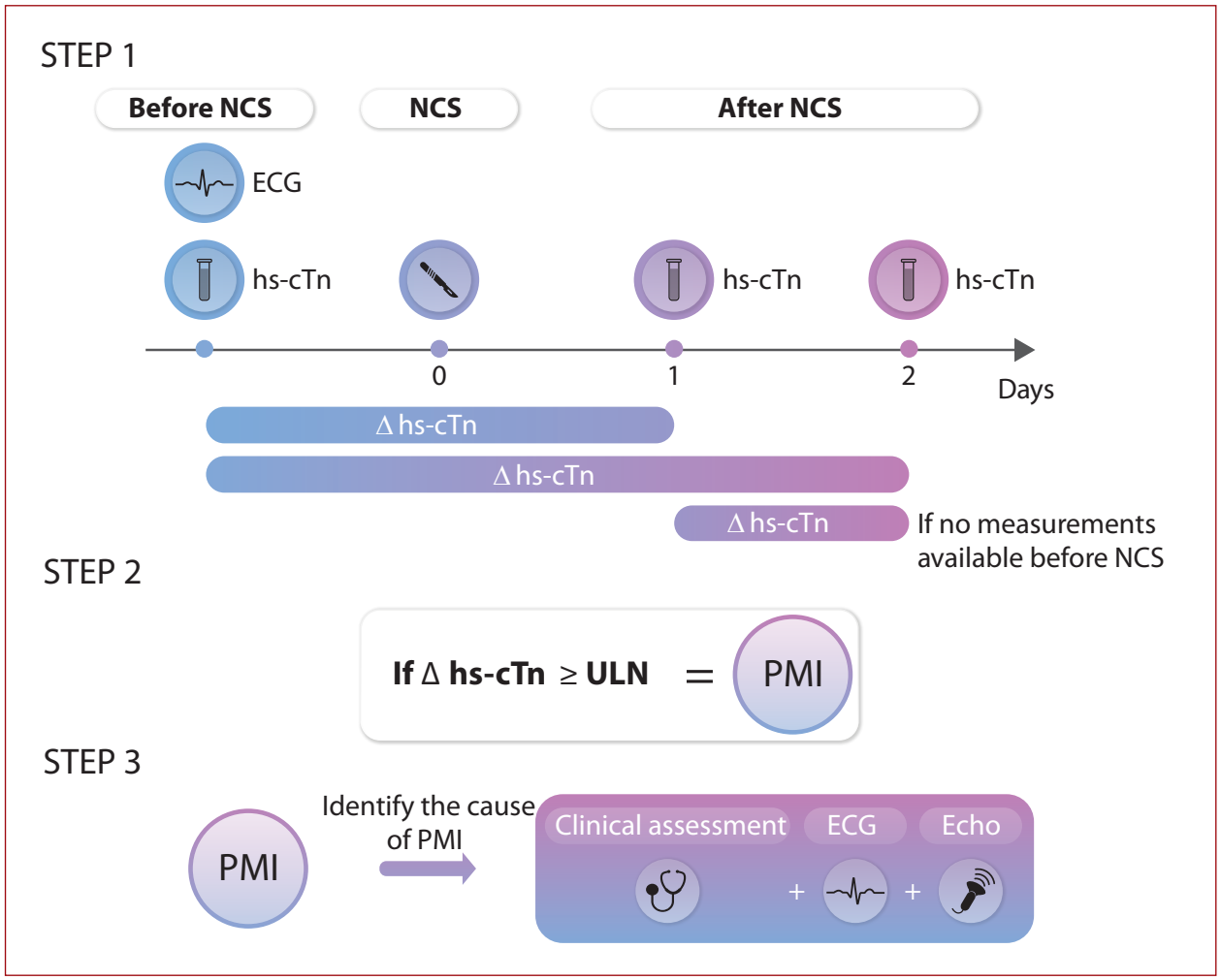

Figure 1. Current recommendation for the timing of hs-cTn measurement to detect PMI in high-risk patients undergoing non-cardiac surgery [10]

Abbreviations: ECG, electrocardiogram; hs-cTn, high-sensitivity cardiac troponin; NCS, non-cardiac surgery; PMI, perioperative myocardial infarction/injury; ULN, upper limit of normal

severe sepsis, stroke, and pulmonary embolism that result in acute myocardial injury and thereby cause an acute rise in hs-cTnT/I concentration. Therefore, it is important to use the generic term "perioperative myocardial infarction/injury" initially to remind colleagues and ourselves that at this time point, we usually have not yet fully understood the cause of cardiomyocyte damage. The detection of PMI must, therefore, be followed by a systematic PMI workup (Figure 2) to allow cause-specific therapy.

Implementing active surveillance of PMI and the systematic PMI workup, as indicated by the current ESC guidelines (class I recommendation), is a major undertaking for institutions. It requires close interdisciplinary collaboration between anesthesia, surgery, cardiology, and laboratory medicine, but also close interprofessional collaboration between nurses, physicians, and IT specialists [10]. This investment, however, is crucial to address the major problem of perioperative cardiac complications including death. Sites that have successfully implemented active surveillance of PMI and a systematic PMI workup should serve as role models and help other sites learn from their experience.

At the same time, we need to continue investing in large prospective high-quality studies in the perioperative setting to gradually overcome our ignorance of the real driving forces and exact mechanisms that cause perioperative cardiac damage, PMI, and its corresponding clinical etiologies. The fact that most randomized controlled intervention studies in the perioperative setting have shown inconclusive results should help us acknowledge how much we still have to learn to finally better serve the needs of our patients.

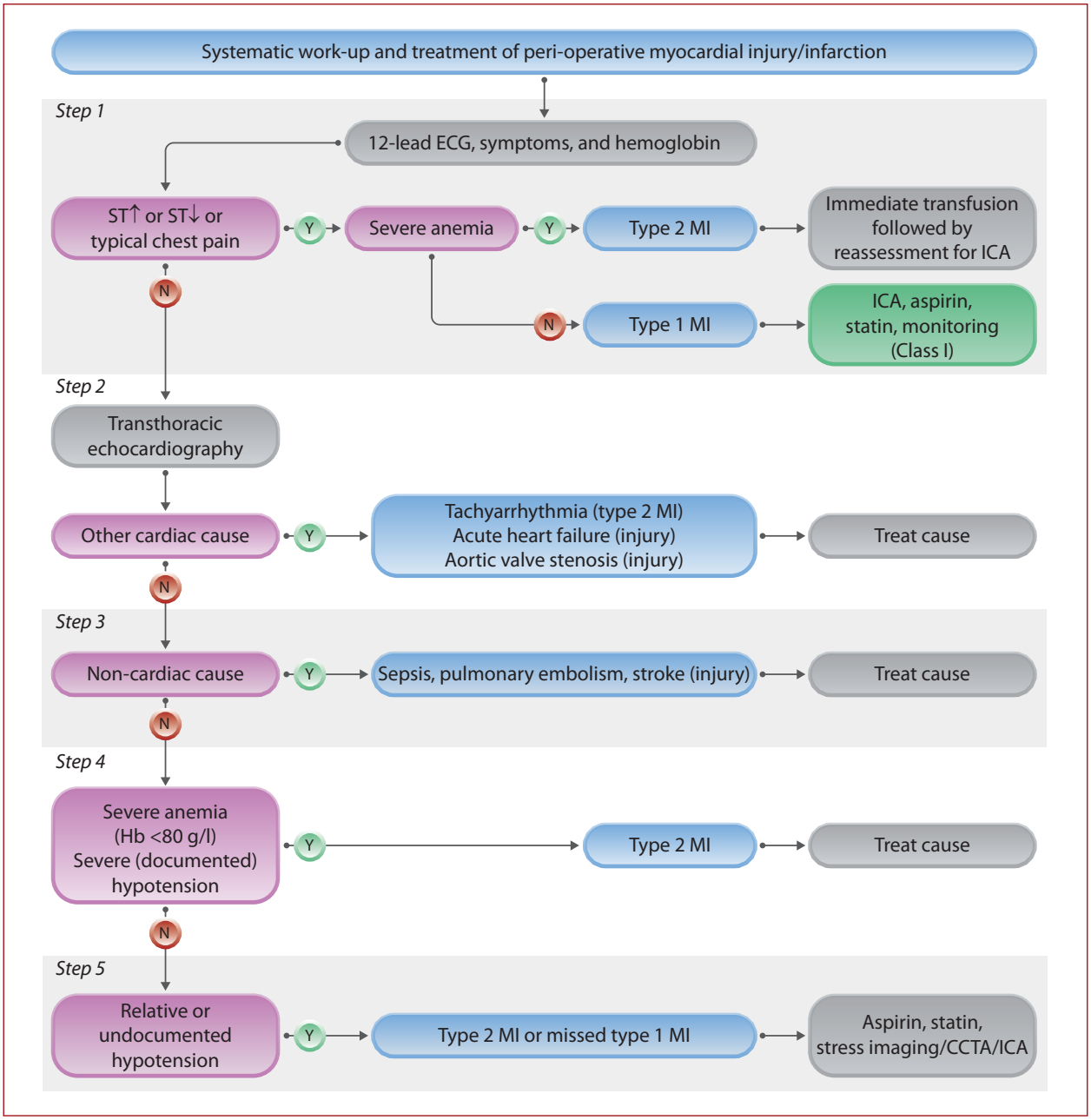

Figure 2. Systematic workup and treatment of patients suffering from perioperative myocardial infarction/injury [10]

Abbreviations: CCTA, coronary computed tomography angiography; Hb, hemoglobin; ICA, invasive coronary angiography; MI, myocardial infarction; N, no; ST, ST-segment; Y, yes; other — see Figure 1

Article information

Conflict of interest: CM has received research support from the Swiss National Science Foundation, the Swiss Heart Foundation, the University Hospital Basel, the University of Basel, Abbott, Astra Zeneca, Beckman Coulter, Boehringer Ingelheim, Brahms, Idorsia, Novartis, LSI Medience Corporation, Ortho Clinical Diagnostics, Quidel, Roche, Siemens, Singulex, and SpinChip, as well as speaker honoraria/consulting honoraria from Abbott, Amgen, Astra Zeneca, Bayer, Boehringer Ingelheim, BMS, Idorsia, Novartis, Osler, Roche, Sanofi, and SpinChip, all outside the current work and paid to the institution. MP declared no conflict of interest.

Funding: None.

REFERENCES

1. Polok K, Buse GL, Mauermann E, et al. Association between self-reported functional capacity measures and postoperative myocardial injury in patients undergoing noncardiac surgeries. Pol Heart J. 2024: 82(7–8): 716–726, doi: 10.33963/v.phj.100567, indexed in Pubmed: 38767162.

2. Weber M, Luchner A, Manfred S, et al. Incremental value of high-sensitive troponin T in addition to the revised cardiac index for peri-operative risk stratification in non-cardiac surgery. Eur Heart J. 2013; 34(11): 853–862, doi: 10.1093/eurheartj/ehs445, indexed in Pubmed: 23257946.
3. Duceppe E, Patel A, Chan MTV, et al. Preoperative N-terminal pro-B-type natriuretic peptide and cardiovascular events after noncardiac surgery: A cohort study. Ann Intern Med. 2020; 172(2): 96–104, doi: 10.7326/M19-2501, indexed in Pubmed: 31869834.
4. Puelacher C, Mueller C, Puelacher C, et al. Perioperative myocardial injury after noncardiac surgery: incidence, mortality, and characterization. Circulation. 2018; 137(12): 1221–1232, doi: 10.1161/CIRCULATIONAHA.117.030114, indexed in Pubmed: 29203498.
5. Gualandro DM, Puelacher C, Lurati Buse G, et al. Incidence and outcomes of perioperative myocardial infarction/injury diagnosed by high-sensitivity cardiac troponin I. Clin Res Cardiol. 2021; 110(9): 1450–1463, doi: 10.1007/s00392-021-01827-w, indexed in Pubmed: 33768367.
6. Puelacher C, Gualandro DM, Lurati Buse G, et al. Etiology of peri-operative myocardial infarction/injury after noncardiac surgery and associated outcome. J Am Coll Cardiol. 2020; 76(16): 1910–1912, doi: 10.1016/j.jacc.2020.08.043, indexed in Pubmed: 33059838.
7. Puelacher C, Gualandro DM, Glarner N, et al. Long-term outcomes of perioperative myocardial infarction/injury after non-cardiac surgery. Eur Heart J. 2023; 44(19): 1690–1701, doi: 10.1093/eurheartj/ehac798, indexed in Pubmed: 36705050.
8. Gualandro DM, Puelacher C, Mueller C, et al. Acute heart failure after non-cardiac surgery: incidence, phenotypes, determinants and outcomes. Eur J Heart Fail. 2023; 25(3): 347–357, doi: 10.1002/ejhf.2773, indexed in Pubmed: 36644890.
9. Devereaux PJ, Biccard BM, Sigamani A, et al. Association of postoperative high-sensitivity troponin levels with myocardial injury and 30-day mortality among patients undergoing noncardiac surgery. JAMA. 2017; 317(16): 1642–1651, doi: 10.1001/jama.2017.4360, indexed in Pubmed: 28444280.
10. Halvorsen S, Mehilli J, Choorapoikayil S, et al. 2022 ESC Guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery. Eur Heart J. 2022; 43(39): 3826–3924, doi: 10.1093/eurheartj/ehac270, indexed in Pubmed: 36017553.