Testing the limits: high-sensitivity cardiac troponin in the prediction of non-cardiac complications after major abdominal surgery

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Editor—We read with interest the study performed by Noordzij and colleagues,[1] which evaluates high-sensitivity troponin results as a predictor for non-cardiac complications, in patients after major abdominal surgery. Whilst these data exquisitely demonstrate an association in postoperative concentrations of troponin elevation and adverse non-cardiac events, we would caution against the use of high-sensitivity troponin as a generic risk stratification tool after major non-cardiac surgery.

In the perioperative setting it is important to consider the pathology behind troponin elevations and distinguish between Type 1 and Type 2 myocardial infarction (MI). We suggest the authors have failed to take this into account. Type 1 MI occurs as a result of atherosclerotic coronary artery disease, with thrombotic coronary arterial obstruction secondary to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection.[2] Patients with Type 1 MI may therefore benefit from primary coronary intervention and prevention strategies. Type 2 MI does not usually have atherosclerotic plaque rupture, but rather, myocardial necrosis secondary to an increase in myocardial oxygen demand or a decrease in myocardial blood flow.

It is accepted that distinguishing between Type 1 and 2 MI during the postoperative period is challenging.[3] It is commonly assumed that an alteration in haemodynamic status caused by sepsis or bleeding as described by Noordzij and colleagues,[1] will lead to a Type 2 MI. However, recent data suggest that up to 50% of patients assumed to have Type 2 MI after non-cardiac surgery have evidence of significant coronary lesions and plaque rupture when assessed angiographically.[4] Therefore, using a blanket troponin testing strategy for risk-stratification, may result in patients with clinically important coronary artery lesions being dismissed as having non-cardiac complications. With increasingly sensitive troponin assays becoming available, careful selection of patients for postoperative testing is paramount.

Declaration of interest

None declared.

References

1. Noordzij P, Van Geffen O, Dijkstra I, *et al*. High-sensitive cardiac troponin T measurements in prediction of non-cardiac complications after major abdominal surgery. *Br J Anaesthesia* 2015; **114**: 909–18
2. Thygesen K, Alpert J, Jaffe A, Simoons M, Chaitman B, White H. Third universal definition of myocardial infarction. *Circulation* 2012; **126**: 2020–35
3. Alpert J, Thygesen K, White H, Jaffe A. Diagnostic and therapeutic implications of type 2 myocardial infarction: review and commentary. *Am J Med* 2014; **127**: 105–8
4. Gualandro D, Campos C, Calderaro D, *et al*. Coronary plaque rupture in patients with myocardial infarction after noncardiac surgery: frequent and dangerous. *Atherosclerosis* 2014; **222**: 191–5

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Reply from the authors

Letter in response to 'Testing the limits: high-sensitivity cardiac troponin in the prediction of non-cardiac complications after major abdominal surgery'

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Editor—We thank Dr Williams for the interest in our paper and welcome the opportunity to respond. As Dr Williams correctly points out, both type I and type II myocardial infarction may be involved in the pathophysiology of asymptomatic cardiac troponin (cTn) elevations after non-cardiac surgery. However, the goal of our study was to determine if postoperative cTn

measurements could predict the occurrence of future non- cardiac events, regardless of the pathophysiological mechanism that causes such elevation. Indeed, our main finding was that a two-fold increase in high-sensitive cTnT compared with baseline, is a strong predictor of major non-cardiac 30 day complications, increased hospital stay, and hospital mortality in patients undergoing major abdominal surgery.[1] This finding is relevant because it suggests that cTn measurements may provide important additional information to identify patients at risk for future non-cardiac adverse events.

Whether postoperative cTn elevations in our study patients can solely be explained by the occurrence of myocardial ischaemia (either an imbalance of myocardial oxygen supply and demand or rupture of vulnerable coronary plaques) is unclear. However, acute coronary events seem unlikely as cTn elevations were mild (an absolute increase of 5 ng litre^{-1} in patients with adverse non-cardiac 30 day outcome *vs* 3 ng litre^{-1} (P=0.002) in patients with uneventful recovery), isolated and without clinical symptoms. Also, other mechanisms for postoperative cTn elevation in surgical patients such as preoperative illness (31% of patients in the MICOLON study had a high- sensitive cTnT concentration >14 ng litre^{-1} before surgery) or systemic inflammation as a result of extended surgical trauma must be considered.[2]

We agree with Dr Williams that careful selection of patients for postoperative cTn testing is essential. Patients included in the MICOLON study had at least one major or two minor risk factors for coronary artery disease and were undergoing a surgical procedure with an expected mortality rate >3%. To assure that postoperative cTn elevations were not attributed to the occurrence of a non-cardiac complication, such as sepsis or bleeding, only cTn measurements before a non-cardiac complication were included in the analyses. Therefore, we do not share the concern expressed in the letter by Dr Williams, that patients with postoperative cTn elevations may be dismissed as having non-cardiac complications whilst having important coronary artery lesions.

As has been shown by others, postoperative cTn measurements are of value in risk stratification for adverse outcome after major non-cardiac surgery.[3–5] However, clinicians should be aware that in surgical patients mild asymptomatic postoperative cTn elevation may be associated with other causes than ischaemic heart disease.

Declaration of interest

None declared.

References

1. Noordzij PG, Van Geffen O, Dijkstra I, *et al*. High-sensitive cardiac troponin T measurements in prediction of non-cardiac complications after major abdominal surgery. *Br J Anaesthesia* 2015: **114**: 909–18
2. Tanindi A, Cemri M. Troponin elevation in conditions other than acute coronary syndromes. *Vasc Health Risk Manag* 2011; **7**: 597–603
3. Landesberg G, Shatz V, Akopnik I, *et al*. Association of cardiac troponin, CK-MB, and postoperative myocardial ischemia with long-term survival after major vascular surgery. *J Am Coll Cardiol* 2003; **42**: 1547–54
4. Van Waes JA, Nathoe HM, de Graaff JC, *et al*. Myocardial injury after noncardiac surgery and its association with short-term mortality. *Circulation* 2013; **127**: 2264–71
5. Devereaux PJ, Chan MT, Alonso-Coello P, *et al*. Association between postoperative troponin levels and 30-day mortality among patients undergoing noncardiac surgery. *JAMA* 2012; **307**: 2295–304

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