openheart

The relevance of periprocedural troponin rise: the never ending story!

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To cite: Fröhlich GM, Leistner DM. The relevance of periprocedural troponin rise: the never ending story!. *Open Heart* 2017;**4**:e000590. doi:10.1136/openhrt-2017-000590

Accepted 29 March 2017

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High-sensitivity troponin is—per definition—highly sensitive to detect all sorts of myocardial injury. This does not necessarily mean that permanent damage has been done to the myocyte. It is known that the troponin level may well be elevated after exceptional physical exercise, like in marathon runners.[1] Of course, long-term prognosis will not be compromised in these athletes, but also in non-coronary conditions like aortic valve or mitral valve disease, elevated troponin values were detected, with different implications on prognosis (table 1).

A variety of studies addressed the frequent finding of elevated biomarker values following coronary angiography and percutaneous interventions with or without stent deployment in patients with stable coronary artery disease.[2–4] Potential mechanism of periprocedural infarcts are (1) side branch occlusion, (2) distal embolisation, (3) prolonged or multiple balloon inflation, (4) coronary dissection with slow flow or (5) microthrombi and no reflow.[5]

However, the definition of periprocedural myocardial injury varies among different authors and the interpretation of these data may proof difficult. In particular, as an isolated troponin elevation might have less prognostic impact if compared with true myocardial necrosis with a creatine kinase MB (CK-MB) rise.[5] Tricoci and collegues compared the prognostic impact of Creain-kinase-MB (CK-MB) and troponin rise. Interestingly enough, the mortality risk of a CK-MB rise >3× upper limit of normal (ULN) was comparable to a cTroponin rise >60× ULN.[5]

In the interventional community, it is widely accepted that an isolated minor troponin rise following percutaneous coronary procedures will not affect prognosis. Therefore, no guidelines recommend routine evaluation of biomarkers in patients with an uneventful postinterventional course. However, the European Society of Cardiology defined the percutaneous coronary intervention (PCI)-associated myocardial ischaemia as a *Type 4a* infarct.[4] The Type 4a infarct is characterised by an elevation of troponin values >5×99th percentile ULN in patients with normal baseline values and (1) symptoms suggestive of myocardial ischaemia, (2) new ischaemic ECG changes or new left bundle branch block, (3) angiographic loss of patency of a major coronary artery or a side branch or persistent flow or no flow or embolisation or (4) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality.[4]

In the present issue of 'Open Heart', Hamaya and collegues investigate the impact of high-sensitivity I troponin elevation. Their study included 538 stable patients who underwent a diagnostic coronary angiogram. The authors identified patients with minor procedure-related myocardial necrosis and those with major procedure-related myocardial necrosis with troponin elevation >3–5x ULN. The troponin was measured just before the angiogram and 18–24 hours postprocedure. Importantly, in patients with significant coronary artery disease, any revascularisation procedure was rescheduled for a second session.

The main findings of this study were that patients with troponin elevation were older, female, had previous coronary interventions and a longer procedural time. Patients with major elevations of troponin had higher levels of N-terminal -Brain Natriuertic Protein (NT-proBNP) and a higher left ventricular enddiastolic pressure. Moreover, aortic stenosis or pressure wire measurements were associated with a troponin rise. In addition, the authors conclude that a major troponin rise was associated with a worse long-term outcome.

Indeed, it is not surprising that older and sicker patients will experience a more pronounced troponin rise. If this troponin rise does translate into a worse outcome remains somehow speculative. Unfortunately, the patient number in the present study is too small to elucidate this research question.

In general, the interpretation of the presented data is impeded by several potential unmeasured confounders. In particular, the outcome of the revascularisation procedure

Table 1 Troponin as a prognostic marker

In aortic valve disease	
Saito *et al*[6]	Preoperative levels of hs-troponin T predict cardiac death and fatal arrhythmia after aortic valve replacement in patients with aortic stenosis without concomitant coronary artery disease
Rosjo *et al*[7]	Increased hs-troponin-T levels are detectable in patients with moderate and severe aortic stenosis and are associated with poor prognosis
Chin *et al*[8]	Plasma troponin-I concentrations are associated with the need for aortic valve replacement and cardiovascular death
In mitral valve disease	
Oshima *et al*[9]	Length of cardiopulmonal bypass time influences troponin-I levels; higher troponin-I levels on postoperative days 1 and 2 significantly correlated with increased intensive care unit and hospital day
Monaco *et al*[10]	cTroponin I higher after surgical mitral valve replacement than after mitral valve repair and cTroponin I concentrations strongly associated with risk of impending postoperative complications
Wöhrle *et al*[11]	Higher baseline concentrations of hs-troponin strongly predict cardiovascular death and rehospitalisation after percutaneous mitral valve repair
In patients with HF	
Peacock *et al*[12]	Troponin above the upper reference limit is associated with more severe heart failure, more severe heart failure symptoms, need for more aggressive supportive measures and worse outcome
Pascual-Figal *et al*[13]	Nearly all patients with acutely decompensated heart failure have highly sensitive troponin-I or troponin-T value above the 99th percentile
Masson *et al*[14]	5284 patients with chronic heart failure (pooled analysis from Val-HeFT and GISSI-HF): Increases in high sensitivity troponin over 3–4 months of follow-up are associated with all-cause mortality and improve prognostic discrimination beyond baseline high sensitivity troponin values only
In heart transplant recipients	
Bocceciampe *et al*[15]	Elevated troponin levels in the pretransplant period are correlated with reduced LV-EF and regional wall motion abnormalities in the donor heart without correlation to early or late post-transplant outcome
Marasco *et al*[16]	Peak troponins are correlated with postoperative primary graft failure
De Santo *et al*[17]	Total ischaemic time and postoperative troponin elevation >10 µg/L are markers for increased postoperative morbidity and mortality

AS, aortic stenosis; cTnI, cardiac troponin I; ICU, intensive care unit; HF, heart failure.

during a second session is not reflected in the statistical analysis. Was full revascularisation achieved in all patients or not, for example? Did the patient solely experience a minor troponin rise or was it a true type 4a myocardial infarct following the percutaneous intervention?

It is hard to believe that a troponin rise following a diagnostic procedure should impact on survival, while a minor isolated troponin elevation after percutaneous intervention is considered to be negligible?

In conclusion, it is unlikely that this study will change current clinical practice.

Contributors GMF: Idea, draft of the manuscript. DML: Table and proof reading.

Competing interests None declared.

Provenance and peer review Commissioned; internally peer reviewed.

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*Open Heart*2017 4:
doi: 10.1136/openhrt-2017-000590