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High-Sensitivity Troponin Release Profile After Cardiac Surgery

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Background

Postoperative serum troponin levels and perioperative myocardial infarction (MI) rates correlate with mortality and morbidity following cardiac surgery. The objective of this study was to document the release profile of high sensitivity troponin T (hsTnT) following different cardiac operations.

Methods

Patients undergoing one of five different isolated cardiac surgical procedures (eligible preoperative hsTnT <29ng/L, serum creatinine < 0.2mmol/L) were recruited prospectively. Serum hsTnT was measured at 0, 4, 6, 8, 10, 12, 24 and 72 hours after the first surgical insult to myocardium, together with daily electrocardiographs.

Results

There were 10 patients in the on-pump coronary artery bypass group and 5 each in the remaining groups (off-pump coronary artery bypass, open aortic valve replacement, transcatheter aortic valve implantation and mitral valve replacement). Five additional patients were excluded due to perioperative MI or renal failure. Median [range] of peak hsTnT was 241[99–566], 64[50–136], 353[307–902], 115[112–275], and 918[604–1166] ng/L, respectively. Operations with the lowest peak hsTnT values peaked earliest (four hours) while those with highest values peaked latest (eight hours).

Conclusion

After cardiac surgery, the hsTnT profile peaks four to eight hours after the initial surgical insult. The magnitude and timing of the peak correlates to the expected degree of surgically-induced myocardial injury.

Keywords

Troponin • Troponin T • Perioperative period • Cardiac surgical procedures • Myocardial revascularisation • Heart valve prosthesis implantation

Introduction

Q3 Postoperative serum cardiac troponin levels have important prognostic significance with regards to mortality, length of intensive care unit stay, length of hospital stay and other complications following adult cardiac surgery [1–7]. A procedure-dependent level of myocardial cell injury and necrosis is inevitable in cardiac procedures and elevation of cardiac biomarkers is universal, but a higher amount of troponin release indicates a larger myocardial insult. In

addition to the myocardial injury, there may be vascular associated events relating to native or graft vessels, leading to myocardial infarction (MI). Morbidity, early and long-term survival and hospital resource utilisation after CABG are negatively impacted by the occurrence of a perioperative MI [8–12]. Therefore, it is worthwhile to differentiate the “expected background” myocardial injury from the “unexpected complication” of perioperative MI.

The challenge of defining perioperative MI following cardiac surgery has been well recognised and efforts to develop

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consensus definitions have undergone several revisions [13]. Familiarity with the normal postoperative troponin release profile following various cardiac operations would facilitate the recognition of higher than expected troponin levels, thus aiding clinicians in the detection of perioperative MI.

We have previously reported that following coronary artery surgery the release profile of conventional (lower sensitivity) troponin peaks at six to eight hours post initiation of ischaemia in the absence of perioperative MI [14,15]. The release profile of (lower sensitivity) troponin following coronary bypass surgery has been documented by Jorgensen using the Abbott c16000 cardiac troponin I assay (Abbott Diagnostics, Chicago, IL, USA) [16]. Contemporary troponin assays have increased sensitivity and analytical performance, improving the detection of myocardial injury [17-19]. Few studies have documented serial troponins following cardiac surgery in the era of high sensitivity troponin [20].

The objective of the present study was to characterise the release profile of high-sensitivity troponin T (hsTnT) following different coronary and valvular cardiac surgical procedures.

Patients and Methods

Study Design

This was an observational study with prospective data collection. Records were held in the local cardiac surgery database. Data definitions were according to the Australia and New Zealand Society of Cardiac and Thoracic Surgeons (ANZSCTS) national database dataset data definitions 2009.

Patient Selection

Consecutive patients with preoperative hsTnT < 29ng/L and normal renal function (serum creatinine < 0.2 mmol/L, no history of dialysis or renal transplantation) undergoing either on-pump coronary artery bypass grafting (CABG), off-pump coronary artery bypass (OPCAB), open aortic valve replacement (AVR), transfemoral aortic valve implantation (TAVI), or mitral valve repair/replacement (MVR) without any concomitant procedures. Recruitment continued until a predetermined number of patients was reached in each group. Those with MI or renal failure were excluded.

Patients were excluded from the main analysis if they met criteria for MI as defined by The Third Universal Definition of Myocardial Infarction [13]. By arbitrary convention, this expert consensus statement defines myocardial infarction after CABG as the presence of postoperative troponin greater than 10 times the 99th percentile upper reference limit during the first 48 hours following surgery, occurring from a normal baseline troponin, in addition to one of the following criteria: occurrence of angina or equivalent symptoms; ECG changes (new pathological Q wave or new left bundle branch block); angiographically documented new graft or native coronary artery occlusion; or imaging evidence of new loss of viable myocardium [13]. For non-coronary cardiac procedures the

expert consensus statement suggests that it is reasonable to apply the same criteria for procedure-related MI as stated for CABG, despite there being scant evidence.

Patients were excluded if they developed new postoperative renal failure (two or more of serum creatinine ≥ 0.2 mmol/L; double the preoperative value; new requirement for dialysis).

Surgical Techniques and Myocardial Protection Strategies

Five different surgical procedures were studied, each with different degrees of expected surgical-induced myocardial injury.

On-pump CABG used pedicled left internal mammary artery conduits to bypass left anterior descending coronary artery disease, plus saphenous vein conduits to other coronary territories. An arterial cannula and a two-stage venous cannula were inserted into the ascending aorta and right atrium respectively, after the administration of intravenous heparin (300 IU/kg), target activated clotting time (ACT) over 400 seconds. The patient's temperature was allowed to drift to 34°C. Antegrade hyperkalaemic tepid blood cardioplegia (500 mL blood-to-crystalloid ratio 4:1) was delivered to achieve cardiac arrest, followed by maintenance doses (250 mL) every 20-30 minutes.

OPCAB was performed on beating hearts using the Octopus® Evolution AS tissue stabiliser system (Model TS2500, Medtronic, Minnesota, USA) to immobilise a small area of the beating heart to perform coronary grafting, and intra-coronary shunts (Flo-Thru Intraluminal Shunt, Synovis, Minnesota, USA) were used for all grafts to maintain distal perfusion. Procedures were conducted at normothermia, with 10000 IU heparin used for all patients (target ACT over 200 seconds).

The technique for AVR was as for CABG. Maintenance doses of cardioplegia were achieved via direct coronary ostial perfusion when required. Retrograde cardioplegia was not used routinely.

TAVI involves the delivery of a prosthetic aortic valve via a catheter inserted into the femoral artery and passed retrograde along the aorta to replace the diseased native aortic valve. A separate catheter in the femoral vein permits the passage of a pacing wire along the inferior vena cava to the right ventricular apex via which rapid pacing can be activated to achieve an intermittently non-pulsatile environment conducive to the safe deployment of prosthetic valves. Rapid pacing was activated twice per case, first for balloon valvuloplasty to open up the narrowed aperture of the diseased native valve, then for deployment of the Edwards SAPIEN transcatheter heart valve (Edwards Lifesciences Corporation, California, USA).

Mitral valve surgery (MVR) was as for CABG, with bi-caval venous cannulation (one cannula for superior vena cava and a separate cannula for inferior vena cava). Cardioplegic arrest was achieved by combined antegrade and retrograde (via the coronary sinus) routes. Access was routinely gained to the left atrium via standard approach

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