Postoperative myocardial injury in patients undergoing elective hip and knee arthroplasty operations

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Globally, approximately 5% of patients undergoing non-cardiac surgery have cardiac complications.1 There is a growing body of research looking at the use of cardiac biomarkers to both stratify patient risk and guide pre-, intra- and postoperative management of patients undergoing surgery. Within this topic, the implications of elevated high sensitivity Troponin I (hsTnI) levels postoperatively in both patients with cardiac symptoms and those who are asymptomatic are not fully understood. Elevated hsTnI levels signify myocardial injury, which may be due to cardiac causes, such as infarction and arrhythmia, or non-cardiac conditions, such as sepsis or pulmonary embolism.1

While hsTnI testing is commonly used to diagnose myocardial injury in the context of acute cardiac signs or symptoms, perioperative baseline TnI testing is not routinely performed as a screening measure at Burwood Hospital, Christchurch. A survey of postoperative hsTnI measurements was undertaken in patients undergoing elective total-hip and total/hemi-knee arthroplasty operations at Burwood Hospital to assess the results of the tests and how abnormal results were managed.

Prior to collecting information, the Health and Disability Ethics Committees reviewed the plan for the survey and deemed formal application to the ethics committee was not required. The National Health Identifiers of 3,722 patients who underwent elective surgery at Burwood hospital between 1 March 2018 and 28 June 2019 were submitted to Canterbury Health Laboratories (CHL). CHL provided the biomarker levels of all patients who had hsTnI levels measured within the two months before or after their hip or knee arthroplasty surgery. This included patients undergoing bilateral and revision arthroplasties. The electronic medical record system Health Connect South was accessed to ascertain any relevant diagnosis documented for patients with a raised postoperative hsTnI and management details. CHL’s hsTnI reference ranges (0–34 ng/L in males and 0–16 ng/L in females) were used in this audit to differentiate normal and abnormal results.

Over the period of the survey, 1,692 patients underwent elective hip or knee arthroplasty surgery at Burwood Hospital. Eighty one patients (4.8%) had a postoperative hsTnI measured, and 25 (1.5%) were raised. All patients who had raised hsTnI concentrations had postoperative cardiac symptoms or signs (chest pain, dyspnea, syncope, tachycardia, hypoxia). Twelve of the 25 patients with a raised hsTnI postoperatively (and 23 of the total 81 patients) had chest pain recorded as a reason (or one of multiple reasons) for hsTnI testing, and all patients diagnosed with a non-ST segment elevation myocardial infarction (NSTEMI) had chest pain postoperatively. Further analysis into symptoms preceding hsTnI testing was not performed as most patients had multiple, interlinked signs/symptoms documented.

Six patients (0.35%) were diagnosed with NSTEMI. In four patients (0.24%), hsTnI leak secondary to atrial fibrillation (AF) was diagnosed. Two patients (0.12%) had an hsTnI rise associated with congestive heart failure
(CHF), and two others were diagnosed with a ‘postoperative [hs]TnI leak’. One patient (0.06%) was diagnosed with a raised hsTnI secondary to postoperative systemic inflammatory response syndrome (SIRS). One patient was diagnosed with unstable angina, one with a type II myocardial infarction, one with a pericardial effusion and one with a spontaneous coronary artery dissection (SCAD). Five patients (0.30%) did not have a documented diagnosis for their raised hsTnI. The hsTnl of one patient (0.06%) was raised but was diagnosed by the treating doctor(s) as being likely reflective of a ‘high baseline’ level, as there was no dynamic change in the hsTnl result on serial testing. This patient did not have a previously recorded hsTnI level on Health Connect South. Additionally, none of the other patients with an abnormal postoperative hsTnI had (within two months preoperatively) had an hsTnI measured either for screening or investigation of acute cardiac signs/symptoms.

Five out of the six patients diagnosed with a NSTEMI, plus four other patients (one with SCAD, one with a type two myocardial infarction, one with SIRS and one with AF), had dynamic hsTnI changes in the hundreds to thousands of ng/L. Three patients (one with AF and two with no diagnosis for their raised hsTnI level) did not have serial hsTnI testing performed postoperatively. All other patients had a dynamic TnI change of less than 100ng/L.

Understandably, management of patients with a raised postoperative hsTnI level was mainly influenced by the diagnosis of the cause for the hsTnI rise. One of the patients diagnosed with atrial fibrillation (who also had chest pain and an ischaemic ECG change of anterior S-T segment depression) as the cause for their hsTnI rise was commenced on lifelong aspirin. None of the other patients in this group were prescribed long-term antiplatelet therapy.

All patients diagnosed with NSTEMIs were prescribed dual antiplatelet therapy for at least three months (some were already on a single antiplatelet). Two of these patients proceeded to have angiograms both showing severe triple vessel disease; one had a coronary artery bypass graft and the other was continued on medical management. The patient who had a SCAD (diagnosed on angiogram) was also prescribed dual antiplatelet therapy—three months of clopidogrel and lifelong aspirin. The one patient who was diagnosed with unstable angina had no new medications started, but they continued aspirin and anti-anginal medications.

None of the other patients with postoperatively abnormal hsTnI levels had new antiplatelet medications started, other than six weeks of aspirin 100mg daily for venous thromboembolism prophylaxis.

This survey found that 1.5% of patients who underwent elective hip or knee arthro-
plasty and had postoperative cardiac signs or symptoms had a raised postoperative hsTnI. However, it is likely that this number underrepresents the true total number of patients (both symptomatic and asymptomatic) with postoperative myocardial injury. The VISION study (which included both acute and elective non-cardiac surgery) reported that 93.1% of patients who had postoperative myocardial injury and 68.0% of those who had postoperative myocardial infarctions did not have postoperative cardiac symptoms. Myocardial injury was defined as “elevated TnI after non-cardiac surgery irrespective of the presence of a feature of an ischaemic feature, judged as resulting from myocardial ischaemia.” Thus it is likely that more patients would have elevated hsTnI levels if asymptomatic patients were screened perioperatively.

There are several more limitations to this survey. Firstly, the small sample size resulted in few abnormal postoperative hsTnI results. Additionally, the lack of preoperative hsTnI levels makes it impossible to know whether a raised hsTnI could have been a patient’s baseline level and not a change in level reflecting acute myocardial injury. This is especially applicable to the cases where there was a smaller dynamic change in hsTnI level or serial testing was not performed. Lastly, diagnosing the cause of a raised hsTnI postoperatively is difficult, as simultaneous processes may elevate hsTnI levels. The true cause may be multifactorial, as opposed to a single documented diagnosis.

Current practice at Burwood Hospital is to use acute cardiovascular signs and symptoms to prompt measurement of hsTnI pre- and post-hip and knee arthroplasty and not to employ measurement as a screening tool. It is likely the incidence of perioperative hsTnI elevation is much higher than currently observed. Any perioperative hsTnI elevations are associated with an increase in postoperative mortality, and this increase is doubled if associated with a myocardial infarction. Further work is required to elucidate the mechanisms of troponin leak associated with different clinical scenarios and the appropriate management strategies. Until then, perioperative hsTnI screening outside a research environment may raise more questions than provide answers.

Competing interests: Nil.

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