Original Article

Electrocardiograph Changes, Troponin Levels and Cardiac Complications After Orthopaedic Surgery

Carol P Chong, [1,4]*MBBS, FRACP, MD*, William J van Gaal, [2,4]*FRACP, FCSANZ, FESC*, Konstantinos Profitis, [2]*MBBS*, Julie E Ryan, [3]*BApp Sci, MAACB*, Judy Savige, [4]*MBBS, FRACP, PhD*, Wen Kwang Lim, [1,4]*MBBS, FRACP, MD*

Abstract

Introduction: The relationship between electrocardiograph (ECG) changes and troponin levels after the emergency orthopaedic surgery are not well characterised. The aim of this study was to determine the correlation between ECG changes (ischaemia or arrhythmia), troponin elevations perioperatively and cardiac complications. Materials and Methods: One hundred and eighty-seven orthopaedic patients over 60 years of age were prospectively tested for troponin I and ECGs were performed on the first 3 postoperative mornings or until discharge. Results: The incidences of pre- and postoperative troponin elevation were 15.5% and 37.4% respectively, the majority were asymptomatically detected. Most of the patients who sustained a troponin rise did not have any concomitant ECG changes (51/70 or 72.9%). Postoperative ECG changes were noted in 18.4% (34/185) and of those with ECG changes, slightly more than half (55.9%) had a troponin elevation. Most ECG changes occurred on postoperative day 1 and were non-ST elevation in type. ECG changes occurred more frequently with higher troponin levels. Postoperative troponin elevation (*P* = 0.018) and not preoperative troponin level (*P* = 0.060) was associated with ECG changes on univariate analysis. Two premorbid factors were predictors of postoperative ECG changes using multivariate logistical regression; age [odds ratio (OR), 1.05; 95% CI, 1.005 to 1.100, *P* = 0.029) and sex OR, 2.4; 95% CI, 1.069 to 5.446, *P* = 0.034). Twenty patients sustained postoperative cardiac complications; 9 (45%) were associated with ECG changes and 16 (80%) with postoperative troponin elevation. Pre- or postoperative troponin elevation better predicted cardiac complications compared with preoperative ECG changes. Conclusion: Electrocardiograph changes do not necessarily accompany troponin elevations after the emergency orthopaedic surgery but are more likely to have higher troponin levels. The best predictor of postoperative cardiac complications is troponin elevation.

Ann Acad Med Singapore 2013;42:24-32

Key words: Cardiovascular, Fracture, Myocardial ischaemia, Myocardial infarction, Surgery

Introduction

Following orthopaedic surgery, approximately 5% of patients sustain cardiac complications which leads to increased morbidity and mortality.[1,2] The mortality rate after hip fracture is as high as 10% to 26% at 6 months[3] and cardiac-related death are common.[4,5] Cardiac injury is potentially treatable and therefore early detection of myocardial ischaemia is essential. This has led to studies conducted to determine predictors of postoperative cardiac events including perioperative cardiac troponin and electrocardiograph (ECG) monitoring.[6-11] Interestingly, patients who are at risk of postoperative cardiac complications may be missed if the standard criteria for the diagnosis of myocardial infarction are applied. For example, recent studies have shown that troponin elevations, a surrogate marker for cardiac injury, occur frequently (up to 53% in 1 cohort) and usually present without symptoms.[7,12] Troponin elevations are associated with higher mortality at 1 year.[7,12] In the past, non-cardiac surgical studies have focussed mainly on the vascular population as they have

[1]Department of Aged Care, The Northern Hospital, Epping, Victoria, Australia
[2]Department of Cardiology, The Northern Hospital, Epping, Victoria, Australia
[3]Department of Biochemistry The Northern Hospital, Epping, Victoria, Australia
[4]Northern Clinical Research Centre, Northern Health, The University of Melbourne, Victoria, Australia
Address for Correspondence: Dr Carol P Chong, Department of Aged Care, The Northern Hospital, 185 Cooper Street, Epping, Victoria, Australia.
Email: carol.chong@nh.org.au

the traditional risk factors for cardiovascular disease.[13-16] Few orthopaedic studies correlate ECG changes with troponin levels.[10,17-19] These studies include a small cohort of orthopaedic patients and have found that postoperative myocardial ischaemia is often silent, occur early after surgery and ECG changes are usually non-Q wave in origin.[18,19]

The diagnosis of perioperative myocardial infarction (PMI) can be difficult to make following orthopaedic surgery and not fitting with the traditional myocardial infarction definition outside of the surgical setting.[20] The main difficulties with the diagnosis is that PMI often occurs silently, and with limited or subtle ECG changes in the setting of a troponin elevation.[21] Therefore, the aim of this study was to characterise pre- and postoperative ECG changes and correlate them with troponin levels and cardiac complications.

Materials and Methods

This prospective observational study was performed as a substudy of the randomised controlled trial which is currently being conducted, randomising patients who sustain a troponin elevation to usual (standard) care or cardiology care (Australian New Zealand Clinical Trials Registry trial number ACTRN12608000165381). Ethics approval was given by the Northern Hospital Human Research and Ethics Committee and patients were recruited between April 2008 and February 2009.

Subjects

Patients were recruited consecutively from the orthopaedic-geriatric unit at The Northern Hospital, Epping, Victoria, Australia. The inclusion criteria for admission to this study was age over 60 years undergoing emergency (non-elective) orthopaedic surgery, mainly for fractures, or an acute condition such as septic arthritis requiring an operative procedure. These patients were jointly managed by the orthopaedic and geriatric unit of the hospital. Patients were excluded if they did not undergo surgery, died before surgery, came from a nursing home or were receiving high level care at home or patients with an estimated mortality of less than 1 year (e.g. terminal cancer), and patients with severe dementia.

Collection of Blood for Troponin

Consenting patients had blood drawn for troponin I on the first 3 postoperative days in addition to blood tests ordered by the treating unit. Troponin was tested immediately after blood collection postoperatively. However, preoperative troponin was only tested and assayed after the operation. The preoperative specimen was stored in a refrigerator at 4˚C for 7 to 10 days. It has previously been shown that troponin results remain stable during this time.[12] This serum was assayed at the same time the first postoperative troponin blood test was ordered. This ensured that the study did not interfere with preoperative management since the preoperative troponin results were not known before the operation. The Beckmann-Coulter Troponin I assay was used with the 99th percentile of a healthy reference population being <0.04 mcg/L. Levels of 0.05 mcg/L and above were considered abnormal for the purpose of this study. Results of troponin were known to both the researchers and the treating team at the time of testing.

Electrocardiographs

All patients received routine preoperative management including preoperative electrocardiographs. However, in the postoperative period in addition to standard orthopaedic-geriatric management by the treating unit, ECGs were obtained on the first 3 postoperative mornings or until discharge by the study team and the treating unit were also aware of the results.

Patient Care and Management

Patients continued to receive the usual care from the orthopaedic-geriatric unit during this study including the additional ordering of troponin or ECGs if clinically indicated. As part of the randomised controlled trial, patients with elevated postoperative troponin were randomised that day to either routine care from the treating orthopaedic-geriatric unit or cardiology care (the intervention being studied). Description of the cardiology management is beyond the scope of this particular article but in brief included evaluation for the cause of the troponin elevation, continuous holter monitoring for at at least 24 hours and treatment of myocardial ischaemia if warranted (e.g. with aspirin, beta blockers or other cardiac medications).

Statistical Analyses

A physician associated with this study and independent of the treating unit obtained medical and demographic data from medical records and additionally interviewed patients and their carers for information if it was not clear. Cardiac events and other complications were determined clinically by the treating team with review by the research team to confirm events. Patients were reviewed daily during the inpatient stay for symptoms of myocardial ischaemia such as chest pain, dyspnoea, hypotension. Postoperative cardiac complications or cardiac events included acute myocardial infarction, congestive cardiac failure, new onset or rapid atrial fibrillation, major arrhythmia or cardiac arrest. For this study, postoperative acute myocardial infarction was

diagnosed by an increase in troponin I above the reference range accompanied by either ischaemic symptoms, ischaemic ECG changes or new pathological Q waves. This definition is based on the recent Task Force definition of acute myocardial infarction.[22] Renal failure was defined as an acute increase in creatinine above the normal reference range (or above the baseline creatinine level in cases of known chronic elevations).

At the conclusion of the recruitment period, all ECGs were interpreted by a cardiologist blinded to the troponin results. ECG parameters deemed abnormal and suggestive of ischaemia were ST segment elevation or depression of 1 mm or more, T wave inversion or flattening, new Q waves, new bundle branch block or conduction abnormality. New onset arrhythmias were also recorded (e.g. atrial fibrillation).

Data were analysed using SPSS (Version 17.0; SPSS, Chicago, IL, USA). Patient characteristics were divided into patients with and without postoperative ECG changes to determine univariate then multivariate predictors of postoperative ECG changes using logistic regression with the enter or simultaneous method. For multivariate predictors of postoperative ECG changes premorbid patient characteristics with a univariate $P < 0.05$ were included in the analysis except for premorbid renal failure due to small patient numbers. For multivariate predictors of postoperative cardiac complications, only preoperative ECG changes and either pre- or postoperative troponin rises were included in the model as they were the predictors of interest, given the small number of outcomes. Odds ratios and 95% confidence intervals are reported with $P < 0.05$ considered statistically significant. The independent samples t test was used to determine the means for continuous variables.

Results

In total, 383 patients were screened for this study. There were 187 eligible patients included in the randomised controlled trial. The reason for exclusion for the majority of patients was receiving high level care, undergoing elective surgery, terminal illness or coexisting severe dementia. Fifteen eligible patients declined involvement in this study. Two out of 187 did not have any ECGs performed during the perioperative period (1.1%) and were excluded from the ECG analysis. Out of 187, 6 (3.2%) did not have any postoperative ECGs but did have a preoperative ECG, 38/187 (20.3%) had 1 postoperative ECG, 48/187 (25.7%) had 2 postoperative ECGs and 95/187 (50.8%) had 3 ECGs performed postoperatively and 77/187 (41.2%) were discharged within 3 days of surgery.

Demographic and clinical data relating to the patients are shown in Table 1. The mean age of the patients was 76.7 years (standard deviation [SD] of 9.3 years) and for fractured neck of femur 80.9 years (standard deviation of 7.7 years). The most common operation was for fractured neck of femur (49.2%). Intraoperative hypotension occurred in a similar proportion of patients with and without ECG changes, 58.8% vs 55.6% respectively, $P = 0.735$. Neither general anaesthetic ($P = 0.782$) or spinal and regional anaesthesia predicted intraoperative hypotension.

Table 1. Univariate Predictors of postoperative ECG Changes

	No postoperative ECG changes (n = 151)	New postoperative ECG changes (n = 34)	Odds Ratio (95% CI)	*P* value*
Age, mean ± SD	76.1 ± 9.3	80.1 ± 8.7	1.1 (1.0 – 1.1)	**0.024**
Male, n (%)	39 (25.8)	15 (44.1)	2.3 (1.05 – 4.9)	**0.037**
Barthels score, mean ±SD	98.0 ± 7	98.0 ± 6	1.0 (0.95 – 1.1)	0.919
Number of Comorbidities, mean ± SD	2.1 ± 1.2	2.6 ± 1.8	1.3 (1.0 – 1.7)	**0.045**
Type of comorbidity, n (%)				
Ischaemic heart disease	22 (14.6)	9 (26.5)	2.1 (0.9 – 5.1)	0.098
Congestive cardiac failure	8 (5.3)	5 (14.7)	3.1 (0.9 – 10.1)	0.063
Hypertension	93 (61.6)	23 (67.6)	1.4 (0.6 – 2.9)	0.510
AF†	9 (6.0)	5 (14.7)	2.7 (0.9 – 8.7)	0.092
Renal Failure	2 (1.3)	4 (11.8)	9.9 (1.7 – 56.7)	**0.010**
Dementia	11 (7.3)	3 (8.8)	1.2 (0.3 – 4.7)	0.76
Diabetes	35 (23.2)	8 (23.5)	1.0 (0.4 – 2.5)	0.965
COPD‡	12 (7.9)	3 (8.8)	1.1 (0.3 – 4.2)	0.866
Stroke	17 (11.3)	2 (5.9)	0.5 (0.1 – 2.2)	0.36
Smoker	15 (9.9)	5 (14.7)	1.5 (0.5 – 4.6)	0.412
Malignancy	3 (2.0)	0 (0)	0.8 (0.07 – 9.4)	Nil

Table 1. Univariate Predictors of postoperative ECG Changes (con't)

	No postoperative ECG changes (n = 151)	New postoperative ECG changes (n = 34)	Odds Ratio (95% CI)	*P* value*
Type of medication, n (%)				
Beta Blocker	31 (20.5)	11 (32.4)	1.9 (0.8 – 4.2)	0.141
Antiplatelet agent	49 (32.7)	12 (25.3)	1.1 (0.5 – 2.5)	0.769
Warfarin	8 (5.3)	4 (11.8)	2.4 (0.7 – 8.4)	0.178
ACEI or ATII§	74 (49.0)	11 (32.4)	0.5 (0.2 – 1.1)	0.082
Calcium Channel Blocker	29 (19.2)	9 (26.5)	1.5 (0.6 – 3.6)	0.346
Nitrate	5 (3.3)	1 (3.0)	0.9 (0.1 – 8.1)	0.934
Diuretic	37 (24.7)	12 (35.3)	1.7 (0.7 – 3.7)	0.209
Statin	46 (30.5)	10 (29.4)	1.0 (0.4 – 2.1)	0.904
NSAID‖	9 (6.0)	1 (2.9)	0.48 (0.1 – 3.8)	0.475
Steroids	6 (4.0)	1 (2.9)	0.73 (0.1 – 6.3)	0.776
Cholinesterase Inhibitor	3 (2.0)	1 (2.9)	1.5 (0.2 – 14.7)	0.736
Antipsychotic Agent	5 (3.3)	2 (5.9)	1.8 (0.3 – 9.8)	0.484
Oral hypoglycaemic Agent	24 (15.9)	4 (11.8)	0.7 (0.2 – 2.2)	0.546
Insulin	6 (4.0)	3 (8.8)	2.3 (0.5 – 9.8)	0.251
No. of medications ±SD	4.7±3.2	5.1±3.7	1.0 (0.9 – 1.2)	0.419
Fracture Type, n (%)				
Neck of femur	67 (44.4)	25 (73.5)	3.5 (1.5 – 8.0)	**0.003**
Lower limb	20 (13.2)	3 (8.8)	0.4 (0.1 – 1.4)	0.140
Wrist	24 (15.9)	3 (8.8)	0.3 (0.1 – 1.8)	0.512
Upper limb	19 (12.6)	2 (5.9)	0.3 (0.5 – 10.4)	0.278
Other	8 (5.3)	0 (0)	0.4 (0.05 – 3.1)	0.370
Time to surgery, mean days ± SD	1.9 ± 2.2	2.4 ± 2.3	1.1 (0.95 – 1.3)	0.215
Anaesthetic type, n (%)				
General	101 (66.9)	18 (52.9)	0.5 (0.2 – 1.1)	0.094
Spinal or Regional	44 (29.1)	15 (44.1)	1.9 (0.9 – 4.1)	0.094
Length of surgery, mean hours ± SD	1.5 ± 0.8	1.6 ± 0.9	1.2 (0.8 – 1.8)	0.421
Intraoperative hypotension	84 (55.6)	20 (58.8)	1.1 (0.5 – 2.4)	0.735
Average dose of postoperative metaraminol (mg)	1.1 ± 1.5	1.3 ± 1.9	1.1 (0.8 – 1.3)	0.608
Postoperative complications, n (%)				
Acute myocardial infarction	6 (4)	6 (17.6)	5.2 (1.6 – 17.2)	**0.007**
Congestive cardiac failure	5 (3.3)	4 (11.8)	3.9 (1.0 – 15.4)	0.052
Any cardiac complication	11 (7.3)	9 (26.5)	4.6 (1.7 – 12.2)	**0.002**
Pneumonia	9 (6.0)	5 (14.7)	2.7 (0.8 – 8.7)	0.092
Renal Failure	3 (2.0)	5 (14.7)	8.5 (2.0 – 37.6)	**0.005**
Blood transfusion	26 (17.2)	7 (20.6)	1.2 (0.5 – 3.2)	0.643
Preoperative pathology, mean ± SD				
Troponin elevation, n (%)	20 (13.2)	9 (26.5)	2.4 (1.0 – 5.8)	0.060
Haemoglobin (g/L)	12.1 ± 1.8	12.2 ± 2.2	1.0 (0.8 – 1.2)	0.965
Creatinine (umol/L)	80.2 ± 31.2	109.8 ± 84.0	1.01 (1.003 – 1.02)	**0.010**
Estimated Glomerular Filtration Rate	70.3 ± 19.7	61.8 ± 23.6	0.98 (0.96 – 0.99)	**0.039**

Table 1. Univariate Predictors of postoperative ECG Changes (con't)

	No postoperative ECG changes (n = 151)	New postoperative ECG changes (n = 34)	Odds Ratio (95% CI)	*P* value*
Postoperative pathology, mean ± SD				
Troponin elevation, n (%)	51 (33.8)	19 (55.9)	2.5 (1.2 – 5.3)	**0.018**
Haemoglobin (g/L)	10.7 ± 1.9	10.7 ± 1.8	1.0 (0.8 – 1.2)	0.890
Creatinine (umol/L)	81.9 ± 33.2	113.9 ± 100.6	1.01 (1.002 – 1.02)	**0.016**
Estimated Glomerular Filtration Rate	68.8 ± 20.0	60.2 ± 24.0	0.981 (0.96 – 0.99)	**0.035**

* Unadjusted *P* value for predictors of postoperative ECG changes
† Atrial Fibrillation
‡ Chronic Obstructive Airways Disease
§ Angiotension Converting Enzyme Inhibitor or Angiotension II Receptor Blocker
‖ Non Steroidal Anti-Inflammatory Drugs

The incidence of a troponin elevation postoperatively was 37.4% (70/187), the majority of these rises were asymptomatically detected. Only 5.7% (4/70) had symptoms suggestive of myocardial ischaemia and 2 later developed symptoms after the troponin results were known to be elevated. Twenty-nine patients (29/187 or 15.5%) sustained a preoperative troponin elevation.

The most common postoperative day for troponin elevation was day 1 with an incidence of 47/70 or 67.1% followed by day 2, 15/70 (21.4%) and day 3, 8/70 or 11.4%.

Postoperative ECG changes

During the inpatient admission, 34/185 (18.4%) developed ECG changes. Of those with an ECG change, 19/34 (55.9%) had a troponin elevation whereas 15/34 (44.1%) did not have a troponin elevation. The majority of patients who sustained a troponin rise did not have any ECG changes, 51/70 (72.9%). The majority of ECG changes (64.7%) occurred on postoperative day 1, 26.5% on day 2, 5.9% on day 3 and 2.9% on day 5. Thirty-eight new ECG abnormalities were detected postoperatively in 34 patients. These were ST elevation of 2 mm (1 patient), ST elevation 1 mm (2 patients), ST depression 1 mm (11 patients), T wave changes (14 patients), new left bundle branch block (2 patients), new right bundle branch block (1 patient), poor R wave progression (1 patient), ventricular trigeminy (1 patient), new onset atrial fibrillation (5 patients). Resolution of these abnormalities in subsequent ECGs were seen in 14/38 (36.8%) patients in subsequent ECGs. Resolution of ST elevation occurred in 2/3 (66.7%) patients, T wave inversion 5/14 (35.7%), ST depression 6/11 (54.5%) and first degree AV block 1/1 (100%).

ECG changes were more likely with higher troponin levels (Table 2). Patients with troponin levels above 0.10 mcg/L had at least a 40% incidence of an abnormal ECG. A normal postoperative troponin level was associated with 13.0% of abnormal postoperative ECGs.

Univariate and Multivariate Predictors of Postoperative ECG Changes

Univariate predictors of postoperative ECG changes were age, male sex, number of comorbidities, premorbid renal failure and fractured neck of femur (Table 1). The type of medications did not predict postoperative ECG changes. There were 54 males in total (out of 185) or 29.2%, 12 (22.2%) had concomitant ischaemic heart disease (IHD) in comparison to women 19/133 or 14.3% which was not significantly different using chi-square test, $P = 0.186$. Preoperative troponin elevation ($P = 0.06$) did not predict postoperative ECG changes whereas postoperative troponin elevations did ($P = 0.018$). Postoperative complications associated with ECG changes on univariate analysis were acute myocardial infarction, any cardiac complications (including acute myocardial infarction, atrial fibrillation, congestive cardiac failure, cardiac arrest) and postoperative renal failure.

Two premorbid factors were predictors of postoperative ECG changes using multivariate logistical regression were age OR, 1.05; 95% CI, 1.005 to 1.100, $P = 0.029$) and sex OR, 2.4; 95% CI, 1.069 to 5.446, $P = 0.034$) adjusting for the number of comorbidities OR, 1.2; 95% CI, 0.896 to 1.576, $P = 0.231$.

Table 2. ECG Changes and Correlation with Troponin Levels

Postoperative peak troponin	No ECG changes	Postoperative ECG changes detected	% with new postoperative ECG changes
≤0.04	100	15	15/115 (13.0%)
0.05 to 0.09	37	8	8/45 (17.8%)
0.10 to 0.4	9	6	6/15 (40%)
>0.4	5	5	5/10 (50%)
TOTAL	151	34	

Perioperative Cardiac Intervention

Of the 70 patients included in the randomised controlled trial, 35 received cardiology care or intervention and 35 received standard care. Of these 35 patients who received cardiology care, 9 had postoperative ECG changes. Twenty-nine patients (82.9%) received changes to their medications, 6 did not have any medications added; 16 patients started aspirin, 9 started a beta blocker and 1 had their dose increased, 1 reduced and 1 ceased, 2 started an ACE inhibitor and 8 started a statin.

In comparison, of the other 35 patients who received standard care, 10 had postoperative ECG changes. Twenty-seven patients (77.1%) continued with standard care. Eight patients received a change to their management including 3 who were referred for cardiology review. Two patients started aspirin, 1 started a beta blocker and 1 started a statin.

Therefore, a total of 35 patients (35/187 or 18.7%) received changes to their medications or care as a result of the intervention.

Receiver Operator Curve for Postoperative ECG Changes Using Peak Troponin as Predictor

A Receiver Operative Curve (ROC) curve was drawn for postoperative ECG changes using peak troponin as a predictor in Figure 1. The area under the curve was 0.712 (95% CI, 0.574 to 0.845, P <0.007) at a cut-off troponin level of 0.065 mcg/L with a sensitivity of 79% and specificity of 57% for postoperative ECG changes. For a cut-off troponin level of 0.105 mcg/L, the sensitivity was 58% and specificity was 74.5%.

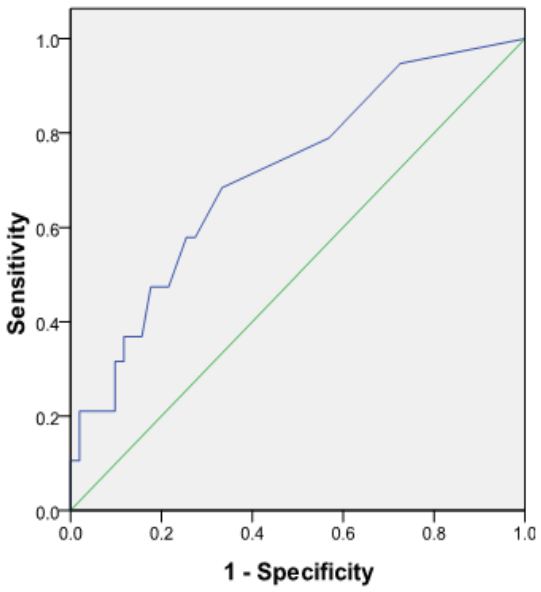

Fig. 1. Receiver operator curve for postoperative ECG changes with peak troponin as a predictor.

Total Inpatient Mortality

There were 3 inpatient deaths (1.6% mortality), all died after the hip surgery. One patient had normal ECGs and troponin levels but died on day 3 of a sudden cardiac arrest found in electromechanical dissociation followed by ventricular fibrillation during failed resuscitation. The two other patients died of acute myocardial infarction with peak troponins of 1.14 mcg/L and 62.04 mcg/L , both having preoperative ECG abnormalities. The first patient had left bundle branch block (which remained unchanged postoperatively) and the second patient had preoperative ST depression V1 and V2 with subsequent T wave changes postoperatively in leads II, III, V3 and V4.

Postoperative Cardiac Complications

Twenty patients sustained any postoperative cardiac complication, of which 9 (45%) were associated with ECG changes and 16 (80%) associated with a postoperative troponin elevation. Predictors of postoperative cardiac complications are shown in Table 3. When included in a multivariate model together both preoperative troponin elevation OR, 7.4 ; 95% CI, 2.3 to 24.2, P = 0.001 and preoperative ECG changes OR, 4.1; 95% CI, 1.3 to 13.4, P = 0.017 predicted postoperative cardiac complications. Similarly, when postoperative troponin elevation was included instead of preoperative troponin elevation , this was significantly associated with cardiac complications

Table 3. Predictors of Postoperative Cardiac Complications

	Unadjusted OR (95% CI)	**Unadjusted *P* value**
Age	1.0 (1.0 to 1.1)	0.079
Sex	1.8 (0.7 to 4.6)	0.250
Number of comorbidities	2.0 (1.3 to 2.9)	0.001
Premorbid comorbidities		
Ischaemic heart disease	4.2 (1.5 to 11.3)	0.005
Congestive cardiac failure	2.8 (0.7 to 11.1)	0.149
Atrial fibrillation	3.9 (1.1 to 14.0)	0.035
Renal failure	4.5 (0.8 to 26.5)	0.094
Aspirin use	2.3 (0.9 to 5.8)	0.089
Beta blocker use	2.0 (0.8 to 5.5)	0.160
Preoperative ECG abnormalities	4.5(1.5 to 13.4)	0.007
Preoperative eGFR	0.96 (0.95 to 0.99)	0.004
Fractured neck of femur	2.7 (0.98 to 7.3)	0.056
Preoperative Troponin rise	7.8 (2.9 to 21.1)	<0.001
Postoperative ECG changes	4.6 (1.7 to 12.2)	0.002
Postoperative Troponin rise	8.4 (2.7 to 26.2)	<0.001

eGFR: estimated Glomerular Filtration Rate

OR, 11.3; 95% CI, 2.4 to 53.1, $P = 0.002$ adjusted for preoperative ECG changes OR, 3.9; 95% CI, 1.2 to 12.5, $P = 0.023$.

Twelve patients had a postoperative acute myocardial infarction. All of these patients sustained a troponin rise, 12/187 or 6.4%. Of these 12 patients, 6 had new postoperative ECG changes. The 6 new ECG changes associated with acute myocardial infarction were T wave inversion in 1 patient, ST depression in 3 patients and 2 new left bundle branch blocks. Out of 34patients with new ECG changes, 28 were not diagnosed with an acute myocardial infarction (AMI). The mean troponin for those who had a postoperative AMI was 7.0 mcg/mL (SD 17.7) compared with 0.14 mcg/mL (SD 0.27) for those not diagnosed with an AMI.

Preoperative ECG changes

The majority of patients were in sinus rhythm preoperatively. Only 6 patients were in atrial fibrillation (3.2%) on ECG. Preoperative ECG abnormalities were seen in 29 patients or 15.7% of the cohort. Of the 29 patients, 9 (31.0%) had a concurrent preoperative troponin elevation. Of the 29 patients with a preoperative ECG abnormality, 16 went on to sustain a troponin rise (55.2%). The following preoperative abnormalities were detected: ST depression (5 patients), Q waves (5 patients), T wave inversion (7 patients), paced rhythm (2 patients), frequent premature ventricular complexes (2 patients), frequent premature atrial complexes (1 patients), other including first degree AV block (7 patients). Preoperatively, 14/187 (7.5%) had left bundle branch block and 9/187 (4.8%) had right bundle branch block.

Preoperative ECG abnormalities associated with postoperative myocardial infarction were seen in 6/12 (50%) of patients. Preoperative abnormalities were partial left bundle branch block (2 patients), first degree AV-block and left axis deviation (1 patient), ST depression and frequent atrial ectopics (1 patient), anterior Q waves (1 patient), ST depression antero-laterally and Q waves inferiorly (1 patient). Four of these patients with preoperative abnormalities who sustained a postoperative AMI had additional ECG changes on postoperative ECGs .

Discussion

This study found that troponin elevations occur commonly after emergency orthopaedic-geriatric surgery and are not always associated with ECG changes. Notably, the majority of patients (73%) who sustained a postoperative troponin elevation in this series did not have any postoperative ECG changes. Patients with a troponin elevation were also likely to be asymptomatic. These patients may have isolated troponin elevations without any evidence of ECG changes nor cardiac symptoms. Using the universal definition of myocardial infarction, these patients with an isolated troponin elevation do not fit the criteria for a postoperative myocardial infarction using ECG criteria.[22] In patients without ECG changes, continuous monitoring may be helpful in finding episodes of ischaemia and similarly imaging with echocardiography. With more intensive pre- or postoperative monitoring there may be an increase in the detection of myocardial ischaemia. However, this was beyond the scope of this present study.

We evaluated the relationship between electrocardiograph changes and troponin levels after emergency surgery. Other studies have focused mainly on an elective population or those with risk factors for ischaemic heart disease.[10,18,19] Overall in this study, electrocardiograph changes occurred in approximately 1 in 5 patients in total but only half of these changes were associated with troponin elevation. It is not surprising that electrocardiograph changes were more likely with higher troponin levels which may reflect a larger burden of myocardial ischaemia and infarction.

In this study, the majority of ECG changes occurred on postoperative days 1 and 2 with the yield from subsequent ECGs being low. Troponin was a more sensitive marker to detect cardiac injury than the use of an ECG. In patients with preoperative ECG abnormalities, postoperative troponin measurement would be a helpful adjunct as postoperative ECG changes may be harder to distinguish. Patients with ECG changes in general were weaker postoperatively with more complications sustained compared with those without ECG changes. These patients benefit from a multidisciplinary team including orthopaedic surgeons, anaesthetics, physicians and allied health staff given the problems that can arise in the perioperative period. In some hospitals orthopaedic-geriatric units have been established to manage these patients with close collaboration with the anaesthetic unit to promote early fixation of a fracture.[23]

Only half of the patients who were diagnosed with an acute myocardial infarction in this study had new postoperative ECG changes suggestive of ischaemia which is similar to previous studies.[10,19] Therefore, monitoring for ECG changes alone without troponin testing would not be sensitive enough to detect myocardial infarction. Similar to our study, previous surgical studies have found perioperative myocardial infarction to be non-ST elevation, non-Q wave in origin and usually accompanied by subtle ECG changes or ST depression.[18,19,21] There is growing evidence to suggest that Type 2 PMI, where there is an imbalance between oxygen supply and demand, is the mechanism leading to myocardial ischaemia in the majority of patients. In this study, approximately half the patients with and without ECG changes were hypotensive during the operation. Our study further supports this theory given that none of the patients

had postoperative ST elevation on ECG which is more suggestive of Type 1 perioperative myocardial infarction. Type 1 perioperative myocardial infarction is typically due to acute coronary thrombosis which is the classic mechanism for infarction in the non-operative setting.[21]

The only multivariate predictors of postoperative ECG changes using multivariate analysis were age and sex. Interestingly, these 2 factors did not predict postoperative cardiac complications. Instead, postoperative cardiac complications were best predicted by pre- and postoperative troponin elevations. The first postoperative day was when the majority of troponin elevations occurred. Therefore, troponin testing in the perioperative period could alert clinicians early to patients who may later develop cardiac complications, particularly during their inpatient stay. The question remains how these patients should be treated as there are no interventional studies to date to address this dilemma. This study is part of a randomised controlled trial where patients with a troponin elevation are randomised to receiving comprehensive cardiology care including cardiac monitoring, medications and cardiologist review compared with no specific medical intervention and standard orthopaedic-geriatric care. The study is still being evaluated and patients continue to be followed-up.

Lower troponin elevations were less likely to lead to ECG changes whereas higher troponin elevations were often associated with ECG features of myocardial ischaemia. Low level troponin elevations are therefore less likely to suggest myocardial ischaemia but may be due to various other causes of troponin elevations. Causes of a "troponinitis" include sepsis, hypovolaemia, renal failure, congestive cardiac failure, pulmonary embolism which are less likely to invoke ECG changes.[24] Nevertheless, an abnormal troponin level is indicative of myocardial injury and likely worse prognosis as demonstrated by 2 recent studies which found that mortality is higher at one year with raised postoperative troponin.[7,12]

Different ECG abnormalities were considered in this study including ischaemic changes and arrhythmias. Arrhythmias may not necessarily be due to myocardial ischaemia perioperatively and other causes such as sympathetic overactivity or noxious stimuli eliciting a hyperdynamic cardiovascular response is a phenomenon that can occur.[25]

Further research should correlate troponin levels with prognosis to try and elucidate more concerning troponin cut-offs in the absence of symptoms or electrocardiograph changes which is the majority of patients. One recent elective orthopaedic series who studied patients with risk factors for ischaemic heart disease endeavoured to define perioperative myocardial infarction more precisely by determining a troponin cut-off for their population.[10] Urban et al[10] found that in an elective orthopaedic population with risk factors for ischaemic heart disease and mainly undergoing arthroplasties, patients with a low positive troponin reading and without symptoms or ECG changes were at lower risk of cardiac complications as none of these patients had postoperative cardiac problems compared with those with higher troponin levels accompanied by symptoms or ECG changes.

There was a low inpatient mortality of 1.6% in our study. This could be because of the combined nature of orthopaedic-geriatric care at the hospital studied which has been postulated to improve outcomes.[23] Nevertheless, all the 3 patients who died of a cardiac cause highlighted the importance of detecting and treating postoperative myocardial infarction as outcome can be poor with a high mortality rate.[21]

Limitations

The cohort of patients selected were more robust than the typical emergency cohort who presented with fractures as patients requiring nursing home level of care were excluded from this study as it was felt they were not appropriate for randomisation to the cardiology arm of the study which included monitoring in a coronary care unit. A subset of patients studied with troponin elevations were randomised to receive cardiology care which may influence their outcome and possibly decrease the incidence of ECG changes and cardiac complications. However, only a minority (18.7%) received any intervention. Given the relatively small number of patients outcomes, multivariate analysis was limited to a few variables. Future research should enrol greater number of orthopaedic patients to increase the power of the study and to detect more cardiac complications. Also, longer term follow-up would be of interest for this cohort as only in-hospital cardiac complications were evaluated.

Conclusion

Troponin elevations occur commonly after emergency orthopaedic surgery. Most are asymptomatically detected. Postoperative electrocardiograph changes do not necessarily accompany troponin elevations but are more likely with higher troponin levels indicating a postoperative myocardial infarction. Troponin elevation is the best predictor of postoperative cardiac complications rather than ECG changes or other variables. Future research should look for causes of troponin elevations and treatment options to improve prognosis.

Acknowledgments

Our thanks to Austin Pathology nurses and staff at The Northern Hospital and to Beckmann Coulter and Austin Pathology for complimentary testing of Troponin I.

Funding: This study was sponsored by a Cardiovascular Lipid grant 2009 from Pfizer Australia. Dr Carol Chong is a recipient of a National Health and Medical Research Council (Australia) postgraduate research scholarship and has received research stipends from The University of Melbourne and the Northern Clinical Research Centre, Northern Health.

REFERENCES

1. Modesti PA, Simonetti I, Olivo G. Perioperative myocardial infarction in non-cardiac surgery. Pathophysiology and clinical implications. Intern Emerg Med 2006;1:177-86.
2. Devereaux PJ, Goldman L, Cook DJ, Gilbert K, Leslie K, Guyatt GH. Perioperative cardiac events in patients undergoing noncardiac surgery: a review of the magnitude of the problem, the pathophysiology of the events and methods to estimate and communicate risk. CMAJ 2005;173:627-34.
3. Naglie G, Tansey C, Kirkland JL, Ogilvie-Harris DJ, Detsky AS, Etchells E et al. Interdisciplinary inpatient care for elderly people with hip fracture: a randomized controlled trial. CMAJ 2002;167:25-32.
4. Perez JV, Warwick CP, Case CP, Bannister GC. Death after proximal femoral fracture - an autopsy study. Injury 1995;26:237-40.
5. Rosencher N, Vielpeau C, Emmerich J, Fagnanis F, Samama M. Venous thromboembolism and mortality after hip fracture surgery: the ESCORTE study. J Thromb Haemost 2005;3:2006-14.
6. McHugh P, Gill NP, Wyld R, Nimmo WS, Reilly CS. Continuous ambulatory ECG monitoring in the perioperative period: relationship of preoperative status and outcome. British Journal of Anaesthesia 1991;66:285-91.
7. Ausset S, Auroy Y, Lambert E, Vest P, Plotton C, Rigal S, et al. Cardiac troponin I release after hip surgery correlates with poor long-term cardiac outcome. Eur J Anaesthesiol 2008;25:158-64.
8. Mouzopoulos G, Kouvaris C, Antonopoulos D, Stamatakos M, Tsembeli A, Mouratis G, et al. Perioperative creatine phosphokinase (CPK) and troponin I trends after elective hip surgery. J Trauma2007;63:388-93.
9. Jules-Elysee K, Urban MK, Urquhart B, Milman S. Troponin I as a diagnostic marker of a perioperative myocardial infarction in the orthopedic population. J Clin Anesth 2001;13:556-60.
10. Urban MK, Jules-Elysee K, Loughlin C, Kelsey W, Flynn E. The One Year Incidence of Postoperative Myocardial Infarction in an Orthopedic Population. HSSJ 2008;4:76-80.
11. Muir AD, Reeder M, Foex P, Ormerod OJM, Sear JW, Johnson C. Preoperative silent myocardial ischaemia: incidence and predictors in a general surgical population. Br J Anaesth 1991;67:373-7.
12. Chong CP, Lam Q, Ryan JE, Sinnappu RN, Lim WK. Incidence of post-operative Troponin I rises and one year mortality after emergency orthopaedic surgery in older patients. Age and Ageing 2009;38:168-74.
13. Blecha MJ, Clark ET, Worley TA, Salazar MR, Podbielski FJ. Predictors of electrocardiographic change, cardiac troponin elevation, and survival after major vascular surgery: a community hospital experience. Am Surg 2007;73:697-702.
14. Landesberg G, Shatz V, Akopnik I, Wolf YG, Mayer M, Berlatzky Y, et al. Association of cardiac troponin, CK-MB, and postoperative myocardial ischemia with long-term survival after major vascular surgery.[see comment]. J Am Coll Cardiol 2003;42:1547-54.
15. Landesberg G, Mosseri M, Shatz V, Akopnik I, Bocher M, Mayer M, et al. Cardiac troponin after major vascular surgery: the role of perioperative ischemia, preoperative thallium scanning, and coronary revascularization. [see comment]. J Am Coll Cardiol 2004;44:569-75.
16. Landesberg G, Mosseri M, Zahger D, Wolf Y, Perouansky M, Anner H et al. Myocardial infarction after vascular surgery: the role of prolonged stress-induced, ST depression-type ischemia. J Am Coll Cardiol 2001;37:1839-45.
17. Adams JE, 3rd, Sicard GA, Allen BT, Bridwell KH, Lenke LG, Dávila-Román VG, et al. Diagnosis of perioperative myocardial infarction with measurement of cardiac troponin I.[see comment]. N Engl J Med 1994;330:670-4.
18. Neill F, Sear JW, French G, Lam H, Kemp M, Hooper RJ, et al. Increases in serum concentrations of cardiac proteins and the prediction of early postoperative cardiovascular complications in noncardiac surgery patients. [see comment]. Anaesthesia 2000;55:641-7.
19. Badner NH, Knill RL, Brown JE, Novick TV, Gelb AW. Myocardial infarction after noncardiac surgery.[see comment][erratum appears in Anesthesiology. 1999 Feb;90(2):644; PMID: 9952182]. Anesthesiology 1998;88:572-8.
20. Thygesen K, Alpert JS, Jaffe AS, White HD. Intraoperative myocardial infarction in non-cardiac surgery: is a universal definition feasible? reply. European Heart Journal 2008;29:567-8.
21. Landesberg G, Beattie WS, Mosseri M, Jaffe AS, Alpert JS. Perioperative Myocardial Infarction. Circulation 2009;119:2936-44.
22. Thygesen K, Alpert JS, White HD, on behalf of the Joint EAAWTFftRoMI. Universal Definition of Myocardial Infarction. J Am Coll Cardiol 2007;50:2173-95.
23. Chong CP, Christou J, Fitzpatrick K, Wee R, Lim WK. Description of an orthopaedic-geriatric model of care in Australia with 3 years data. Geriatr Gerontol Int 2008;8:86-92.
24. Jeremias A, Gibson CM. Narrative Review: Alternative Causes for Elevated Cardiac Troponin Levels when Acute Coronary Syndromes Are Excluded. Ann Intern Med 2005;142:786-91.
25. Lessa MA, Rodrigues E, Tibirica E. Cardioprotective action of fentanyl in a model of central sympathetic overactivity in rabbits: antiarrhythmic and anti-ischemic effects. Acta Anaesthesiol Scand 2004;48:1115-22.