The treatment of perioperative myocardial infarctions following noncardiac surgery

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Keywords: surgery, morbidity, myocardial infarction

Abstract

Background: Perioperative myocardial infarction (PMI) is a common complication following noncardiac surgery, with a 30-day mortality of 10-20%. Effective therapeutic interventions are of public health importance.

Method: This is a systematic review, aimed to determine the evidence for therapies following PMI.

Results: A PubMed Central search up to May 2011 identified 20 case series and reports (89 patients). We extracted data on the type and timing of treatment and short-term mortality. Short-term mortality differed significantly between haemodynamically stable and unstable patients (0% and 32.2% respectively, p-value = 0.015). Significantly more haemodynamically unstable patients received acute coronary interventions (75.8% vs. 23.1%, p-value = 0.0006). Acute coronary intervention in haemodynamically unstable patients was not associated with improved short-term survival (p-value = 0.53). The high proportion of symptomatic and haemodynamically unstable patients suggests publication bias ($\chi 2$ = 16.29, p-value = 0 < 0001 and $\chi 2$ = 154.41, p-value < 0.0001, respectively).

Conclusion: This systematic review highlights the paucity of evidence for PMI management, and the need for future prospective trials.

Ⓟ **Peer reviewed. (Submitted: 2011-11-04. Accepted: 2012-03-12.)** **South Afr J Anaesth Analg 2012;18(2):86-93**

Introduction

Recent studies suggest that perioperative myocardial infarction (PMI) is a common complication of noncardiac surgery, with an incidence of 5% in patients who are 45 years or older, with cardiovascular risk factors.[1] This carries a significant health burden. Therefore, efforts to accurately document the incidence of perioperative cardiovascular complications and associated clinical risk predictors,[2] as well as to study preventative strategies to decrease perioperative cardiovascular complications,[3-5] are appropriate.

We are of the opinion that there have been few, if any, studies examining therapeutic interventions for patients who have had a PMI. This is despite a reported 30-day mortality of between 11.6%[1] and 21.6%.[6] Medical (nonsurgical) trials of patients with myocardial infarction (MI) have highlighted the importance of both the timing and the choice of therapeutic intervention in patients with MI.[7] Thus, through appropriate perioperative therapeutic interventions, the potential may exist for an enormous impact on both the short- and long-term survival of patients following a PMI.

The aim of this systematic review is to determine the evidence for therapeutic interventions following PMI.

Method

We conducted a systematic review of the treatment received, and associated outcomes following PMI in noncardiac surgical patients.

Study end-points

The intention was to extract data on the following:

- The treatment of PMI (medical therapy, invasive coronary intervention, or coronary artery surgical intervention)
- The timing of the intervention (acute, as part of resuscitation associated with the PMI, or delayed, following successful acute therapy for the PMI
- The short-term (30-day or in-hospital) mortality associated with PMI in relation to the received intervention.

Study identification and selection

On 5 May 2011, a PubMed search was conducted for the period 1966-2011. The terms used in the search strategy were “perioperative myocardial infarction” and “treatment”.

The abstracted data were screened and excluded non-eligible studies. All studies that reported treatment modalities used in patients suffering PMI after noncardiac surgery were included. Non-human studies, cardiac surgical studies, paediatric studies, reviews, comments, and letters to the editor, were excluded. Studies listing PMI or raised troponin levels as outcomes, but not detailing treatment, were also excluded, as were studies that reported on treatment of MI in the nonsurgical (medical) population, or outside of the perioperative period. Within eligible studies, individual patients were excluded from the analysis if they did not experience a PMI, e.g. postoperative angina or preoperative MI.

Data extraction

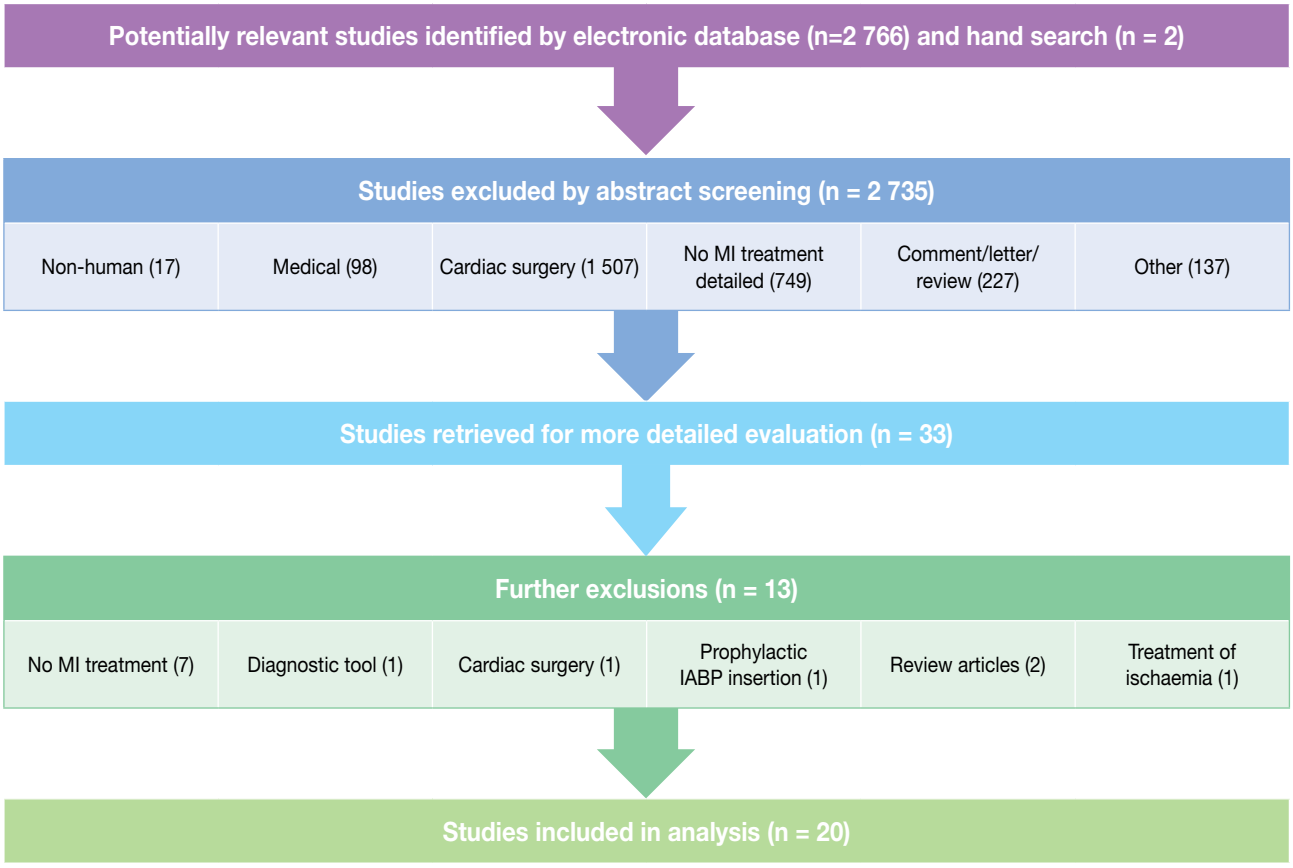
Data on the treatment modality administered to patients with PMI, the timing of the intervention (acute or delayed), haemodynamic stability of the patients following PMI, and the short-term (30-day or in-hospital) mortality, were extracted. Where possible, demographic data, including age, gender, known cardiovascular risk factors, and preoperative cardiovascular medications, were extracted. Citations were independently screened, data abstracted, and methodological quality assessed, using a standardised data extraction sheet. Any disagreements were resolved. In cases where data required clarification, or were not presented in the publication, an attempt was made to contact the original authors.

The extracted data only allowed comparison of conservative and invasive coronary therapies and associated outcomes using χ-square testing. Publication bias regarding outcomes was assessed by comparing observed vs. expected frequencies using χ-square testing. All statistical analyses were conducted using GraphPad® software online calculators.

Results

The PubMed search identified 2 766 studies between 1966-2011, and an additional two potentially eligible studies were identified from one of the reviewer's own records.[8,9] Initial abstract screening eliminated 2 735 studies. The remaining 33 studies were extracted for more detailed evaluation, following which a further 13 studies were deemed unsuitable for inclusion. Twenty publications[8,10-28] fulfilled our criteria for analysis (see Figure 1).

From the 20 publications finally selected, 89 patients with PMI were identified, as included in eight case series[8,10,11,14,18,20-22] and 12 case reports.[12,13,15-17,19,23-28] The type of surgery, patient demographics, co-morbidities and preoperative medication are tabulated in Table I.



MI = myocardial infarction, IABP = intra-aortic balloon pump

Figure 1: Flowchart of systematic review process

Table I: Characteristics of the included studies

References	Type of surgery	PMI[a] (n)	Age	Sex	Co-morbidities	Preoperative medications
Medina-Polo et al[22]	Simultaneous pancreas-kidney transplantation	1	66	Not reported	DM,[b] HT[c]	Not reported
Lee et al[18]	Neurosurgery (lumbar fusion)	6	62	Male	DM, HT	Not reported
			70	Male	HT, CVA[d]	Not reported
			67	Female	HT	Not reported
			66	Male	HT	Not reported
			62	Female	HT	Not reported
			64	Male	DM, HT	Not reported
Chang et al[10]	Vascular	2	Not reported	Not reported	Not reported	Not reported
Chiang et al[11]	Head and neck	7	66	Male	Not reported	Not reported
			73	Male	Not reported	Not reported
			69	Male	Not reported	Not reported
			81	Female	Not reported	Not reported
			67	Female	Not reported	Not reported
			85	Female	Not reported	Not reported
			64	Male	Not reported	Not reported
Berger et al[8]	Abdominal (14), orthopaedic (11), vascular (11), urology (5), neurosurgical (3), other (4)	41	70 (± 7.7)	Male (65)%	HT (73%), CAD[f] (48%), DM (29%)	CCB[e] (29%), aspirin (27%)
Malek et al[20]	Urology	1	Not reported	Not reported	Not reported	Not reported
Mangano et al[21]	Thoracic (1), vascular (7), neurosurgical (1), orthopaedic (1)	10	69 ± 9	Male	CAD (all)	Not reported
Gewertz et al[14]	Vascular	2	Not reported		HT (64%), previous MI[g] (28%), CCF[h] (14%)	Not reported
Ito et al[16]	Vascular	1	66	Male	HT, IGT,[i] no CAD	Intravenous heparin (stopped 12 hours prior to surgery)
Uchida et al[27]	Neurosurgery	1	80	Female	PVD[j]	Not reported
Mottard et al[23]	Orthopaedic	1	72	Male	PVD	Statin, warfarin (changed to LMWH[k])
Schmitto et al[25]	Obstetric	1	22	Female	None	Not reported
Iwashita et al[17]	Neurosurgery	1	68	Female	None	Not reported
Fippel et al[13]	Orthopaedic	1	21	Male	None	None
Takahashi et al[26]	Vascular	1	67	Male	Aortic valve replacement	Warfarin until 3 days preoperatively
Corda et al[12]	Vascular	1	84	Female	HT, RAS,[l] PVD	CCB
Lim et al[19]	General surgery	1	70	Male	None	Not reported
Winship et al[28]	Bilateral adrenalectomy	1	64	Male	Conn's syndrome, HT, CAD, previous CABG[m]	Spironolactone, captopril, amlodipine, terazosin, steroids
Ishiyama, Tsujitou[15]	Vascular	1	73	Male	HT, CAD, renal dysfunction	Dialysis
Roth et al[24]	Orthopaedic	1	47	Male	DM, HT	Propranolol, chlorpropamide, enalapril

a = perioperative myocardial infarction, b = diabetes mellitus, c = hypertension, d = cerebrovascular accident, e = calcium-channel blocker, f = coronary artery disease, g = myocardial infarction, h = congestive cardiac failure, i = impaired glucose tolerance, j = peripheral vascular disease, k = low-molecular-weight heparins, l = renal artery stenosis, m = coronary artery bypass graft

Demographic data, co-morbidities and preoperative medical therapy were not reported for a number of the patients. Of the 89 patients, the most commonly performed surgeries were vascular in 29.2% (n = 26), orthopaedic in 16.8% (n = 15), abdominal in 15.7% (n = 14), and neurosurgical in 13.4% (n = 12). Other surgeries included head and neck (n = 7), urological (n = 6), "other" (n = 5), transplant (n = 1), general surgery (n = 1), obstetric (n = 1) and thoracic (n = 1).

The presentation of the PMI, haemodynamic stability, time to intervention, type of therapy (medical and haemodynamic support), coronary revascularisation, and patient outcomes, are tabulated in Table II.

The presentation of the PMI, the presence of haemodynamic instability, the short-term mortality of the patients in the included studies, and the expected 30-day mortalities from a previous meta-analysis and randomised controlled trial,[1,6] are tabulated in Table III. A single study[10] is not included in this table as we had insufficient data to classify outcomes, hence the two patients from this study were excluded, leaving 87 patients for analysis. Of these 87 patients, PMI presented as asymptomatic or unspecified in 12 patients (13.8%), while 75 patients were symptomatic. Of these 75 patients, 13 (14.9%) were haemodynamically stable, with no mortality in this group. The remaining 62 patients (69.7%) were haemodynamically unstable, and had a short-term mortality of 32.2%. Short-term mortality differed significantly between haemodynamically stable and unstable patients (0% and 32.2% respectively, p-value = 0.015).

There were four patients in this series, two with preoperative myocardial infarction, one with intraoperative myocardial infarction, and one with postoperative myocardial infarction. Three of the four patients demised. The fourth patient was left severely disabled. We could not determine which of the patients demised.

Patient management differed significantly according to haemodynamic presentation (see Table IV). Haemodynamically unstable patients received significantly more acute coronary interventions than haemodynamically stable patients [47/62 (75.8%) vs. 3/13 (23.1%) respectively, p-value = 0.0006]. However, within the haemodynamically unstable patient group, the short-term mortality rates did not differ between those who received acute coronary intervention vs. those who did not, namely [14/47 (29.8%) and 6/15(40%), respectively (p-value = 0.53]. The case series of Chang et al[10] was excluded from this analysis as we could not determine which of the patients with PMI had died, thus the analysis included 87 of the 89 identified patients for this review.

We found evidence of potential publication bias. The proportion of asymptomatic patients presented in this review is significantly less than the expected 35%[1] ($\chi 2$ = 16.29, p-value = 0 < 0001). The proportion of haemodynamically unstable patients is also significantly more than the expected 19%[1] ($\chi 2$ = 154.41, p-value < 0.0001).

Discussion

We found no completed randomised controlled trials of therapeutic interventions for PMI, despite the fact that in nonsurgical patients, randomised controlled trials for MI date back nearly 30 years.[29] This would be understandable if PMI treatment was considered to be similar to that of a nonsurgical MI, and hence therapies would be expected to have similar efficacies between medical and surgical patients. However, significant differences clearly exist between these two patient cohorts. In particular, the postoperative patient is exposed to an environment associated with haemodynamic instability, procoagulation, sympathetic stress, and potential bleeding and hypoxia.[30] The pathophysiology of the PMI may also be slightly different to the nonsurgical MI.[31] These factors may explain why the majority of PMIs present with ST segment depression, rather than ST segment elevation that is characteristic of medical patients.[1,32] Finally, while anticoagulants are used extensively in managing nonsurgical MI, in the perioperative patient, this raises concerns of significant bleeding. Therefore, it is likely that the management of PMI requires specific therapeutic investigation and therapies.

We believe that the studies identified in this systematic review should not guide therapeutic management of PMI patients, as it is predominantly retrospective, and appears to be heavily influenced by both publication and patient selection bias. Therefore, the bias in these data would seriously affect the reported outcomes associated with any of the interventions reported. Secondly, the majority of patients identified in this systematic review presented with symptomatic PMI, either through patient cardiac symptoms, or associated haemodynamic instability. Therefore, this review does not reflect the majority of patients with a PMI. The PeriOperative ISchemic Evaluation (POISE) trial, with high quality observational data with respect to PMI, showed that > 60% of patients with a PMI are asymptomatic.[1] Thirdly, in the POISE trial, only 19% of the patients with a PMI developed congestive cardiac failure.

In our systematic review, > 80% of patients with a PMI had haemodynamic instability (see Table III). This suggests that data reported in the literature is biased towards critically ill PMI patients. It is likely that identified publications are also biased towards patients who had positive outcomes. We

Table II: Presentation, diagnosis and management of perioperative myocardial infarctions

References	PMI[a] presentation			Time of first intervention	Medical therapy	Haemodynamic therapy	CR[b] and timing	Outcome
	Time	Diagnosis	Haemodynamics					
Case series								
Medina-Polo et al[22]	Perioperative	Not reported	Not reported	Not reported	Not reported	Not reported	Not reported	Not reported
Lee et al[18]	Day 3	Abdominal pain	Stable	Delayed	Conservative	Not reported	No	Survived
	Day 1	Typical symptoms[c]	Stable		Conservative	Not reported	No	Survived
	Day 0	Typical symptoms[c]	Stable		Conservative	Not reported	No	Survived
	Day 1	Typical symptoms[c]	Stable		Conservative	Not reported	PTCA[d] after failed conservative therapy	Survived
	Day 0	Typical symptoms[c]	Stable		Conservative	Not reported	No	Survived
	Day 7	Dyspnoea, cyanosis, diaphoresis	Stable	Delayed	Conservative	Not reported	CABG[e] after failed conservative therapy	Survived
Chang et al[10]	Intraoperatively (1), postoperatively (1)	Abrupt onset of shock	Unstable	Emergent	Not reported	IABP[f] or percutaneous pacing	Emergency PCI[g] in both	Death or severe disability
Chiang et al[11]	Day 1	ECG,[h] troponins	Unstable	Undetermined	Aspirin		CABG day 2	Discharged
	Day 3	ECG, troponins	Unstable	Undetermined	Aspirin		CABG day 7	Discharged
	Day 1	ECG, troponins	Unstable	Undetermined	Aspirin		CABG day 17	Discharged
	Day 1	ECG, troponins	Stable	Undetermined	Aspirin, digoxin, diuretics, antihypertensives		None	Discharged
	Day 3	ECG troponins	Stable	Undetermined	Aspirin, digoxin, diuretics		None	Discharged
	Days 3 and 15	ECG, troponins	Stable	Undetermined	Aspirin, digoxin, heparin, antihypertensives		None	Died day 99
	Day 8	ECG, troponins	Unstable	Undetermined	Aspirin, heparin, diuretics, antihypertensives		PCI	Died day 74
Berger et al[8]	1.6 (± 1.9)	Typical symptoms,[c] ECG	Shock (21/48)	11.1 h (± 17.4) for angiography	Not reported	21/48 IABP, 16/48 pacing	PTCA 41, CABG 2	31/48 survived
Malek et al[20]	Perioperatively	Not reported	Not reported	Not reported	Conservative	Not reported	No	Not reported
Mangano et al[21]	Day 3	ECG, CKMB[i]	Not reported	Not reported	Not reported	Not reported	No	Died day 8
	Day 5	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	CABG day 75	Not reported
	Day 15	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	No	Died day 16
	Day 4	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	CABG day 478	Not reported
	Day 2	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	PTCA day 43	Not reported
	Not reported	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	No	Died day 26
	Day 2	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	No	Survived
	Day 29	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	No	Noncardiac death day 69
	Day 1	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	No	Survived
	Day 2	ECG, CKMB	Not reported	Not reported	Not reported	Not reported	No	Died day 73

Table II: Presentation, diagnosis and management of perioperative myocardial infarctions

References	PMI[a] presentation			Time of first intervention	Medical therapy	Haemodynamic therapy	CR[b] and timing	Outcome
	Time	Diagnosis	Haemodynamics					
Case reports								
Ito et al[16]	Intraoperatively	ECG changes, RWMAs	Unstable	Immediate	ISDN,[k] lignocaine for VT[l]	Ephedrine, noradrenaline, dopamine infusion	No	Survived
Uchida et al[27]	Intraoperatively	VF[m]	Unstable	Immediate	Nicorandil, nitrates, calcium antagonists, heparin	Cardioversion, catecholamines	No	Discharged
Mottard et al[23]	On completion of surgery	ECG changes	Unstable	Immediate	Aspirin, clopidogrel, ACE-I (later)	Ephedrine, atropine, adrenaline, IABP	PCI	Survived
Schmitto et al[25]	Intraoperatively, on administration of oxytocin	Chest pain, troponins	Unstable	Immediate haemodynamic support. Later, medical	Metoprolol, midazolam	Ephedrine, colloids	No	Survived
Iwashita et al[17]	2 hours postoperatively	ECG changes, CNS[o] symptoms	Unstable	Immediate	Not reported	Not reported	Emergency PCI	Survived
Fippel et al (abstract)[13]	Day 1, day 6	Not reported	Unstable	Immediate	Thrombolysis	IABP	PTCA 'after resuscitation'	Not reported
Takahashi et al[26]	6 hours postoperatively	VF, angiography	Unstable	Immediate	Not reported	Not reported	PTCA	Not reported
Corda et al[12]	Intraoperatively (post-induction)	RWMA	Stable	Immediate	Nitroglycerin, metoprolol, milrinone	No	No	Survived
Lim et al[19]	Day 2	ECG, echo, cardiac enzymes	Unstable	Preoperatively	Not reported	Dopamine, dobutamine, noradrenaline, IABP, Finally, vasopressin	No	Survived
Winship et al[28]	Day 4	Chest pain and cardiac arrest	Unstable	Immediate	Not reported	Not reported	No	Died 24 hours post-MI
Ishiyama, Tsujitou[15]	Preoperatively	Chest pain, ECG	Unstable	Immediate	Not reported	Adrenaline, dopamine, dobutamine, lignocaine	No	Died day 29
Roth et al[24]	Intraoperatively	ECG changes, echo	Stable	Immediate	Nitroglycerin, verapamil, esmolol, morphine	Neosynephrine, lignocaine for VPCs	PTCA within 30 min	

a = perioperative myocardial infarctions, b = coronary revascularisation, c = typical symptoms were defined as chest pain, dyspnoea, diaphoresis, and palpitations, d = percutaneous transluminal coronary angioplasty, e = coronary artery bypass grafting, f = intra-aortic balloon pump, g = percutaneous transluminal intervention, h = echo echocardiography, i = creatine kinase MB fraction, j = regional wall motion abnormalities, k = isosorbide dinitrate, l = ventricular tachycardia, m = ventricular fibrillation, o = central neverous system, p = post myocardial infaction, q = ventricular premature contractions

Table III: Short-term (in-hospital and 30-day) mortality associated with the type of presentation of perioperative myocardial infarctions (PMI

Category	Presentation n (%)	Observed short-term mortality n (%)	Expected 30-day mortality (%)
Unspecified or asymptomatic PMI	12 (13.8)	3 (25)	11.6[1]-21.6[6]
Haemodynamically stable symptomatic PMI	13 (14.9)	0 (0)	11.6[1]-21.[6]6
Haemodynamically unstable PMI	62 (69.7)	20 (32.2)	No known reports

Chang et al[10] is excluded from this analysis.

Table IV: Acute invasive coronary interventions associated with the presentation of a perioperative myocardial infarction

Presentation	Medical therapy only	Invasive coronary intervention
Haemodynamically stable	10	3
Haemodynamically unstable	15	47

p-value = 0.0006
Chang et al[10] is excluded from this analysis

are unaware of any publications that accurately document the 30-day mortality associated with haemodynamically unstable patients, following PMI. A recent meta-analysis and the POISE cohort, which have reported outcomes of both haemodynamically stable and unstable patients, revealed a mortality of 11-22%.[1,6] Thus, we would expect haemodynamically unstable patients to have a mortality rate well in excess of this, yet in this review, the mortality rate was only 32%.

This publication bias has important implications for some of the therapeutic options presented in these case series. For example, the study published by Berger et al,[8] in which they make use of an acute invasive coronary strategy for PMI, is difficult to interpret. Notwithstanding their high survival rate of 65%, despite haemodynamic instability, there is no indication of the number of PMI patients who presented with haemodynamic instability at their hospital. Furthermore, no indication is given of the number of patients who were not referred for an acute invasive coronary strategy following PMI, and the associated outcome. This would suggest a selection bias to these data. Furthermore, when examining all the studies of acute coronary interventions in unstable patients following PMI in this review, the data suggest no difference in survival between patients who had acute coronary interventions and those who had medical therapy alone. All that these studies suggest is that, at best, an acute invasive coronary strategy for PMI needs prospective evaluation. It is important to remember that studies without a comparison group do not allow for any inferences to be made about association or causation.[33]

This systematic review highlights the urgency with which we need to embark on prospective randomised controlled trials of therapeutic interventions for PMI. This is particularly important when we consider that interventional studies are likely to show greater clinical benefit than preventative studies.[34]

Conflict of interest

No external funding and no competing interests are declared.

Acknowledgements

Dr Biccard is supported by a Medical Research Council self-initiated research grant. Dr Rodseth is supported by a Canadian Institutes of Health Research Scholarship (the Canada-HOPE Scholarship).

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