Postcardiac surgery myocardial ischemia: Why, when, and how to intervene

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Feature Editor's Introduction—The diagnosis of postoperative myocardial ischemia in the context of cardiac surgery remains challenging, and its management is a topic of ongoing debate. The electrocardiographic changes after cardiac surgery are common and difficult to interpret, whereas the troponin levels are expectedly elevated. Because patients are rarely capable of reporting the classic ischemic symptoms in the early period after cardiac surgery, it is difficult for clinicians in the intensive care units to discern between trivial and benign changes from clinically important myocardial ischemia, which is reversible with appropriate intervention. Clinicians primarily rely on cardiac biomarkers, hemodynamic changes, and imaging to detect myocardial injury. Whether to rush the patient to the catheterization laboratory, take the patient back to the operating room for revision, or wait patiently and monitor the course is a matter of debate and uncertainty, and this decision requires a stepwise investigation.

In this data-oriented review, Robinson and colleagues describe the incidence and causes of postmyocardial ischemia, review the role and the limitations of diagnostic tests, and propose an algorithm for expeditious intervention in case myocardial ischemia is suspected. They stress on *EARLY*, and they stress on *INTERVENTION*.

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Although relatively uncommon, perioperative myocardial ischemia (PMI) is associated with significantly worse outcomes in patients undergoing cardiac surgery both in terms of mortality and morbidity.¹⁻⁴ The causes of PMI are heterogeneous but can be broadly classified as bypass graft or nongraft-related issues.⁵ In milder forms, PMI can be difficult to differentiate from other expected postoperative metabolic and physiologic responses to surgery.

Algorithm for the treatment of suspected PMI.

CENTRAL MESSAGE

Although the identification of PMI is not always obvious or manifest, when suspected its appropriate management dictates prompt immediate repeat angiography.

See Commentaries on pages 696 and 697.

Electrocardiogram (ECG) and echocardiogram abnormalities are the simplest and most useful initial confirmatory tools, and abnormalities should prompt immediate consideration of urgent angiography.

Once identified, management of PMI with percutaneous coronary intervention (PCI), reoperation, or conservative strategies can be challenging with several clinical factors to consider. Furthermore, because the impact of PMI on short- and long-term outcomes is highly significant, expeditious management is paramount to ameliorate many of these risks.

In this review, we describe the incidence, causes, and presentation of PMI after cardiac surgery and outline treatment options. Although the origin of PMI is usually traceable to intraoperative events, this review deals particularly with the management of PMI that is only detected after the patient has left the operating room (OR).

INCIDENCE OF PERIOPERATIVE MYOCARDIAL ISCHEMIA

Because of the lack of a consistently agreed on definition, the incidence of PMI varies widely in different studies. The exact incidence and precise diagnosis are ultimately dependent on angiographic confirmation, which, in itself, requires

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decisiveness in taking the patient back to the catheterization laboratory. For the purposes of this review, PMI is broadly considered as any patient who experiences new ECG signs of ischemia or new regional wall motion abnormalities (RWMA) on echocardiography, intractable arrhythmias that cannot be explained by the preoperative condition of the patient, or events that occurred during the operation or cardiogenic shock. It is outside the scope of this review to delve into the granular details of this heavily debated topic.

The incidence is also dependent on whether it is isolated coronary artery bypass grafting (CABG) (average, 3.9%, range, 0.3%-9.8%) or a combined operation (average, 2.0%, range, 0.7%-12%). The majority of published series with a sample size of more than 500 patients identify a PMI rate of 2% to 5% (Table 1). In the largest retrospective analysis published to date, which included 554,987 patients who underwent CABG with or without concomitant valve surgery, Alqahtani and colleagues¹⁵ reported that coronary ischemia was suspected in 4.4%. In a meta-analysis of 39,266 patients who underwent repeat angiography after CABG, the pooled PMI rate was 3.5% (95% confidence interval [CI], 0.2-6.8%).²

It is also noteworthy that the incidence of early graft failure is higher than that of PMI, because early graft failure is frequently asymptomatic. However, Laflamme and colleagues¹³ reported that more than 80% of all PMI is caused by early graft failure. Early graft failure rates are related to the graft type and invariably lower for arterial grafts, such as the left internal thoracic artery (1.0%-2.5%) and radial artery (3.0%-4.0%), and higher for saphenous vein grafts (3.0%-12%).²³

DETECTING POTENTIAL PROBLEMS IN THE OPERATING ROOM

Because most cases of PMI have their origin in the OR, a strong case can be made for attempting to detect potential graft problems before the chest is closed. In a study of 366 patients (769 grafts) who underwent routine post-CABG angiography in the OR, an angiographic defect was detected in 12% of grafts, with 22 grafts (2.8%) requiring minor revision (ie, adjustment of conduit position or unroofing of fascia) and 27 grafts (3.4%) requiring traditional surgical revision.²⁴ Although conventional angiography remains the gold standard for assessing graft patency, in the absence of a hybrid OR, more immediate and accessible strategies include transesophageal echocardiography (TEE, to be discussed later under the “Diagnostic Tests” section), intraoperative transit time flow measurement (TTFM),²⁵ or intraoperative fluorescence imaging (IFI), which offer a more practical method for the assessment of intraoperative graft patency.^{26,27}

TTFM and IFI have proven to be safe, efficient, and reproducible.²⁷ Using IFI, authors have reported graft revision rates from 1.4% to 5.0%.^{28,29} Likewise, using a combination of TTFM and high-frequency ultrasound, the prospective 1016 patient REQUEST Registry reported surgical changes

to grafts in 7.8% of patients.³⁰ A pooled analysis of 9 studies investigating TTFM reported a slightly higher graft failure rate at 12%; however, the study definitions varied with graft patency rates ranging from 4% to 20%.³¹

Both IFI and TTFM have some limitations. First, neither technique can be used after chest closure. Second, abnormal angiographic findings have been reported in up to 55% of patients with satisfactory intraoperative TTFM flows but who subsequently developed PMI.²⁰

CAUSES OF PERIOPERATIVE MYOCARDIAL ISCHEMIA

PMI can result from graft-related or nongraft-related complications. The causes of complications due to graft-related and nongraft-related causes are summarized in Table 2.

Graft-related complications represent the most common cause of PMI, accounting for approximately two-thirds of cases. This broad group includes technical errors leading to direct graft injury and subsequent thrombosis, graft kinking, and anastomotic problems such as stenosis, competitive flow, poor distal runoff, and graft spasm. In a recent meta-analysis of 9 studies including 1104 patients with PMI after CABG, Biancari and colleagues² found that 62% of patients had acute graft failure, 6% had incomplete revascularization, and 3.5% had developed a new native coronary artery lesion. Other analyses have reported similar findings.^{7,21}

Nongraft-related causes account for up to 33% of cases of PMI. This includes new native coronary artery problems (ie, plaque rupture, thrombus), distal coronary microembolism, incomplete myocardial revascularization, inadequate myocardial protection, hypercoagulability, and preoperative severe ischemic injury.^{4,7,9,11,16,19,20} In a prospective angiographic study of 1604 consecutive patients who underwent off-pump CABG, incomplete revascularization was reported in 13% and was associated with lower 5-year survival (75% vs 85%).³² Notably, the incidence of nongraft-related causes of PMI is higher in patients undergoing combined surgical procedures where new native coronary artery occlusion was found in 20% and coronary artery spasm in 13% (5/40) of patients.¹⁹

PRESENTATION

PMI can manifest across a wide spectrum from asymptomatic ECG changes to hemodynamic instability to sudden cardiovascular collapse. Early arterial graft failure can cause a more severe clinical presentation than vein graft failure, particularly when supplying the left anterior descending coronary artery territory.⁶

PATIENT CHARACTERISTICS

Although certain preoperative characteristics have been identified epidemiologically with an increased risk of PMI after CABG, including female sex, advanced age,

TABLE 1. Incidence of perioperative ischemia

Author, year	Study period	Type of study	Procedure	No. of patients	Perioperative ischemia	Early mortality*	Long-term mortality
Rasmussen, 1997 ⁵	1990-1995	Prospective	Isolated CABG	2003	3.5%	12.7%	- 23% mortality at median 3-y follow-up
Thielmann, 2006 ⁶	1999-2006	Prospective	Isolated CABG	5427	2.2%	14.9%	- No difference in survival at 6.5 y among PCI, redo CABG, and conservative treatment
Szavits-Nossan, 2012 ⁷	1999-2009	Prospective	Isolated CABG	3000	1.8%	30.0%	- NR
Davierwala, 2013 ⁸	2004-2010	Prospective	Isolated CABG	7461	5.3%	7.3%	- 75% $\pm$ 2.9% 5-y survival (PMI) - 86% $\pm$ 0.5% 5-y survival (no PMI)
Fabricius, 2001 ⁹	1999	Retrospective	Isolated CABG	2052	6.4%	14.5%	- NR
Tan, 2008 ¹⁰	1999-2005	Retrospective	Isolated CABG	4301	1.4%†	18.4%	- 8.1% death within 4 y after PCI
Yau, 2008 ¹	2002-2003	Retrospective	Isolated CABG	3014	9.8%	6.8%	- 2-y mortality was highest in patients with CK-MB >15 $\times$ ULN (9.5%)
Virani, 2009 ¹¹	1997-2002	Retrospective	Isolated CABG	1731	1.7%	13.0%	- NR
Karhunen, 2010 ¹²	1988-2007	Retrospective	Isolated CABG	5251	2.1%	2.8%	- NR
Laflamme, 2012 ¹³	2003-2009	Retrospective	Isolated CABG	5598	0.7%	15.8%	- NR
Narayan, 2014 ¹⁴	2011-2012	Retrospective	Isolated CABG	2312	1.1%	11.1%	- NR
Preußner, 2017 ³	2006-2013	Retrospective	Isolated CABG	4028	4.2%	10.7%	- 63% 7-y survival (PMI) - 81% 7-y survival (no PMI)
Alqahtani, 2019 ¹⁵	2003-2014	Retrospective	Isolated CABG	554,987	4.4%	5.1%	- NR
Rupprecht, 2019 ¹⁶	2008-2015	Retrospective	Isolated CABG	4825	2.2%	13.0%	- NR
Sef, 2019 ⁴	2011-2015	Retrospective	Isolated CABG	1119	3.8%	9.0%	- NR
Sharma, 2020 ¹⁷	1996-2017	Retrospective	Isolated CABG	53,287	0.3%	15.6%	- 15.4% (PCI) - 29.8% (Redo-CABG) - 45.5% (Conservative)
Javierre, 2012 ¹⁸	2004-2009	Prospective	Valve surgery and CABG	2038	Men 11.8% Women 5.6%	5.1%	- NR
Gaudino, 2015 ¹⁹	2005-2014	Prospective	Valve surgery and CABG	5275	0.7%	10.0%	- At a mean follow-up of 29 $\pm$ 26 mo, 6 patients died
Hultgren, 2016 ²⁰	2007-2012	Retrospective	Valve surgery and CABG	4446	2.0%	7.0%	- 77% 5-y survival (PMI) - 87% 5-y survival (no PMI)
De Mey, 2012 ²¹	2003-2009	Retrospective	Valve surgery and CABG	5988	0.7%	NR	- NR
Laimoud, 2020 ²²	2016-2019	Retrospective	Valve surgery and CABG	1869	3.3%	36.0%	- NR

CABG, Coronary artery bypass grafting; PCI, percutaneous coronary intervention; NR, not reported; PMI, perioperative myocardial ischemia; CK-MB, creatinine kinase MB; ULN, upper limit of normal. *Defined as in-hospital or 30-d mortality among patients diagnosed with PMI. †Includes only patients who underwent PCI or re-do CABG within 72 h of index procedure.

TABLE 2. Incidence of graft-related versus nongraft-related complications

Cause	Complication rate (%) ^{4,7,8,12,13,15,16}
Graft related	43-82
Graft stenosis	22-64
Graft occlusion	12-65
Anastomosis stenosis	5.7-49
Graft thrombosis	8.5-35
Graft kinking	2.9-9.9
Graft spasm	7.0-11
Graft displacement	5.0-9.5
Graft dissection	4.0-5.6
Nongraft related	18-37
New native coronary artery stenosis	3.0-20
Incomplete revascularization	3.1-14
Coronary artery spasm	13
Poor distal runoff	7.9

All values represent a range, when applicable.

hyperglycemia, left main or triple vessel disease, unstable angina, peripheral vascular disease, and low body surface area,^{1,33-35} they are too generic to be useful in the individual patient.

In contrast, a high index of suspicion may result from a preoperative angiogram demonstrating multiple small or diffusely diseased and calcified coronary vessels or additional intraoperative difficulties, including incomplete revascularization (where an anatomically important intramyocardial native coronary artery cannot be located) or the need for endarterectomy. Intraoperative variables, such as prolonged aortic crossclamp and cardiopulmonary bypass times, concomitant valve surgery, and inadequate myocardial protection, are also associated with a higher incidence of PMI (Table 3).^{36,37}

However, predisposition to PMI is not isolated to coronary surgery. Calcified aortic stenosis can predispose to calcified fragments occluding the coronary ostia or distal embolization, whereas aortic valve replacement in a right coronary artery particularly close to the annulus can be susceptible to distortion or compression.³⁸ Patients undergoing mitral valve surgery are uniquely at risk of iatrogenic injury to the circumflex coronary artery due its close proximity to the posterior segment of the mitral annulus.³⁹ In type A aortic dissection, aortic hematoma can compress the coronary arteries or the dissection flap can extend into the coronary ostia, significantly increasing the risk of PMI.⁴⁰

DIAGNOSTIC TESTS

ECG abnormalities are common in patients with PMI. It is axiomatic that, as a consequence of coronary artery disease, many patients will have existing abnormalities in the baseline ECG; therefore, new ECG changes are critical in the diagnosis of perioperative ischemia. Although ST-segment and T-wave changes are common in the

TABLE 3. Characteristics of patients at elevated risk of perioperative myocardial ischemia

Operation	Characteristics
CABG	<ul style="list-style-type: none"> • Angiographic features <ul style="list-style-type: none"> ◦ Small coronary artery diameter (ie, <1.5 mm) ◦ Diffuse coronary artery disease • Intraoperative <ul style="list-style-type: none"> ◦ Prolonged cardiopulmonary bypass time ◦ Prolonged aortic crossclamp time ◦ Poor-quality coronary anatomy ◦ Incomplete revascularization ◦ Coronary endarterectomy
Valve surgery	<ul style="list-style-type: none"> • Calcific aortic stenosis in AVR • Low origin of the coronary arteries in AVR • Circumflex coronary artery course close to the posterior mitral annulus in mitral valve surgery • Injury to right coronary artery during tricuspid repair • Anomalous circumflex coronary artery that travels immediately behind the aortic annulus during AVR • Flap extension into the coronary arteries or hematoma adjacent to coronary ostia in aortic dissection

CABG, Coronary artery bypass grafting; AVR, aortic valve replacement.

postoperative period secondary to epicardial injury, ST-segment elevation with reciprocal ST-segment depression may represent a more reliable indicator of true myocardial ischemia and should prompt further investigation.⁴¹

In the SWEDEHEART national registry, Hultgren and colleagues²⁰ reported that 92% of patients with PMI had ECG changes, of whom 83% had isolated ST elevation. However, a wide variety of ECG changes can occur immediately postoperatively, ranging from overt to subtle and including ST-segment elevation or depression, new Q-waves, new left bundle branch block, new ventricular arrhythmias, or subtle and nonspecific T-wave changes. Crescenzi and colleagues⁴² reported that isolated new Q-waves, in conjunction with cardiac enzyme elevation, are predictive of postoperative cardiac ischemia. Furthermore, hemodynamic instability accompanied by ECG changes is more predictive of positive findings on angiography than isolated ECG changes²⁰ and should prompt early repeat angiography.

Echocardiography is invaluable for the detection or confirmation of PMI, and the identification of new changes compared with a preoperative echocardiogram is crucial. New abnormal echocardiographic findings in PMI include paradoxical motion of the interventricular septum, new or worsening wall motion abnormalities, and worsening left ventricular ejection fraction. Some of these findings, such as new paradoxical motion of the septum, can be seen after cardioplegic arrest in the absence of overt ischemia. Intraoperative TEE is especially helpful in identifying/confirming

PMI, and deteriorating TEE wall motion scores is associated with myocardial infarction and subsequent mortality.⁴³ Equally as important is differentiating global ischemia from RWMA, because the latter should raise suspicion of early graft failure. However, in a study of more than 5000 patients, Laflamme and colleagues¹³ reported that intraoperative TEE identified new RWMA in only 20% of patients ultimately diagnosed with PMI after CABG, but remained normal in 54% of patients. Likewise, in a small study of only 32 patients, De Mey and colleagues²¹ found that new TEE RWMA had a specificity of only 57%.

Cardiac biomarkers of myocardial necrosis such as creatinine kinase MB (CK-MB) and cardiac troponin (cTn) can identify and confirm PMI and eventual myocardial infarction and its severity. In general, cTn is the preferred biomarker because of its higher degree of sensitivity and prognostic significance.⁴⁴ However, differentiating expected transient myocardial enzyme release and temporary ECG changes from true PMI can be particularly difficult in the very early postoperative period. Recently, the joint American College of Cardiology/American Heart Association/European Society of Cardiology/World Health Federation task force defined procedure-related myocardial injury as an increase from a normal baseline cTn to more than 10 times the 99th percentile of the upper reference limit within the first 48 hours accompanied by ECG, angiographic, or imaging evidence of new myocardial ischemia (now called “type 5 myocardial ischemia”).⁴¹

There is still no consensus on what cTn level differentiates myocardial procedural injury from myocardial infarction.⁴⁴ Although prior studies have concluded that high levels of enzyme release (ie, cTn >20 × the upper reference limit) may be indicative of PMI, a recent post hoc analysis of SYNTAXES trial demonstrated that in the absence of ECG changes, enzyme release alone as a marker for PMI was not predictive of worse mortality after CABG.⁴⁵ Results from the ongoing Vascular Events in Surgery Patients Cohort Evaluation Cardiac Surgery Study (VISION, NCT01842568) will provide more clarity on this topic.

Using delayed enhanced cardiac magnetic resonance imaging, Pegg and colleagues⁴⁶ found that both CK-MB and cTn peaked at 24 hours in patients with evidence of myocardial necrosis on imaging. Most significantly, cTn was superior to CK-MB in the detection of new myocardial injury after CABG ($r = 0.62$, $P < .01$). Increases in CK-MB levels after internal thoracic artery harvesting that damages the surrounding chest wall muscle is more marked after bilateral internal thoracic artery harvesting.^{47,48}

CONFIRMATORY DIAGNOSIS

Currently, conventional coronary angiography is the only expeditious way to identify the precise cause of PMI. Immediate repeat angiography is mandatory in any case of suspected PMI. Although it is preferable that this is expeditiously agreed by the Heart Team, composed of the

cardiac surgeon, anesthetist, or intensive care unit physician and interventional cardiologist (Figure 1), this must not cause delays.^{7,12,13} Reluctance to formally investigate potential PMI in favor of inotropic or mechanical support (eg, intra-aortic balloon pump) should be strongly resisted.

TREATMENT OPTIONS

A careful, but urgent, decision by the Heart Team should be made between conservative management, PCI or redo CABG, in accordance with the clinical status of the patient and angiographic findings.^{4,6-8,13,19,49} Factors to consider include the hemodynamic status of the patient, coronary anatomy, timing and extent of the myocardial tissue at risk for ischemia, and existing graft configuration.⁵⁰ A summary of treatment strategy rates in the published literature is available in Table 4.

Most cases of PMI that require formal intervention can be treated with PCI, and this is reflected in the most recent European Society of Cardiology guidelines.⁵¹ In the setting of acute graft failure specifically, immediate PCI is the preferred treatment option to limit the extent of infarcted tissue, with several studies reporting success in up to 97%.^{4,7,8,52,53}

A unique consideration with PCI is whether to intervene on the coronary bypass graft or the native coronary artery. A recent meta-analysis of clinical outcomes of PCI after saphenous vein graft failure demonstrated that native artery PCI was associated with lower major adverse cardiac events (odds ratio, 0.51; 95% CI, 0.45-0.57; $P < .001$) and lower all-cause death (odds ratio, 0.65; 95% CI, 0.49-0.87; $P = .004$).⁵³ When anastomotic stenosis is identified, the culprit bypass graft is commonly the primary PCI target (although caution should be exercised when performing PCI on a recent anastomosis).^{4,11} In cases of detected graft spasm during angiography, a catheter intervention with intragraft nitroglycerin injection can also be performed.¹²

Recent guidelines recommend that redo CABG should be considered if the anatomy is unsuitable for PCI, if several important grafts are occluded, or in the case of obvious technical errors.⁴⁹ Patients with severe hemodynamic instability (cardiogenic shock, ventricular arrhythmias) or sudden hemodynamic collapse may need to be taken immediately to the OR. It should be noted that although early postoperative mortality in patients who develop early graft failure and undergo redo CABG is increased in comparison with patients treated with PCI, there is an obvious treatment allocation bias in that redo CABG is usually indicative of a more complex and difficult clinical scenario than that which could not be resolved by PCI.^{4,52,54}

Conservative treatment may be indicated if angiography has excluded any mechanical or other overtly treatable cause.^{49,55} Conservative treatment should be considered in patients who have a patent graft in the setting of PMI or in cases where baseline myocardial viability is already known to be limited.⁵⁰

FIGURE 1. An algorithm for management of patients with suspected PMI after cardiac surgery. Potential reintervention should be immediate and depend on the precise clinical picture. If clinically stable, then immediate angiography is required. If unstable hemodynamics/collapse, then initiation of MCS, if feasible and available, or immediate reexploration in the OR or intensive care unit needs to be considered. ^aSigns and symptoms that should trigger the algorithm include clinical features such as low cardiac output, hypotension, sudden hemodynamic collapse, arrhythmias or chest pain, new ECG changes, or new transthoracic or transesophageal echocardiogram changes. *HD*, Hemodynamically; *MCS*, mechanical circulatory support (ie, intra-aortic balloon pump, extracorporeal membrane oxygenation); *ICU*, intensive care unit; *OR*, operating room; *PCI*, percutaneous coronary intervention; *CABG*, coronary artery bypass grafting.

Mechanical circulatory support should only be used as an adjunct rather than as a primary treatment. Intra-aortic balloon pump or peripheral extracorporeal membrane oxygenation can be considered in rescuing patients who develop significant hemodynamic instability before transfer to the OR or catheterization laboratory, or in the case of hemodynamic instability despite proven patent grafts and maximum inotropic support.⁵⁵

Irrespective of the strategy used, expeditious treatment is of paramount importance to minimize myocardial damage and to improve clinical outcomes.^{8,13} Karhunen and colleagues¹² have reported that in comparison with patients with PMI who did not undergo repeat angiography, angiography was associated with a significant decrease in in-hospital mortality (22% vs 46%, $P = .015$) and a lower rate of emergency CABG reoperations. Davierwala and

TABLE 4. Management strategies in patients undergoing angiography for suspected perioperative myocardial ischemia*

Study	No. of patients undergoing angiography		Treatment strategy			Outcomes
	Total	Positive findings	PCI	Redo CABG	Conservative management	
Daviera, 2013	399	255 (64)	34 (8.5)	130 (33)	91 (23)	- There was no association between type of treatment strategy and in-hospital mortality - No difference in long-term mortality was identified ($P = .01$)
De Mey, 2012	39	32 (82)	15 (38)	4 (10)	13 (33)	- NR
Fabricsius, 2001	108	63 (58)	34 (31)	9 (8.3)	20 (19)	- NR
Gaudino, 2015	40	33 (83)	15 (38)	3 (7.5)	15 (38)	- All 3 patients in the reoperation group died - Long-term survival was highest in the PCI group ($P < .001$)
Hultgren, 2016	87	60 (69)	14 (15)	25 (30)	48 (55)	- 30-d mortality was not significantly different in patients who received a new revascularization and in those who were treated conservatively (10% vs 4%, $P = .27$)
Laimoud, 2020	61	NR	35 (57)	15 (25)	NR	- In-hospital mortality was 25% for PCI group and 87% for reoperation group ($P < .001$)
Narayan, 2014	26	18 (69)	1 (3.8)	17 (65)	NR	- NR
Preußner, 2017	168	130 (77)	60 (36)	30 (18)	78 (46)	- CABG revision in-hospital mortality = 13.3% - PCI in-hospital mortality = 11.7% - Conservative treatment in-hospital mortality = 9.0%
Rupprecht, 2019	108	79 (73)	52 (48)	10 (9.3)	17 (15)	- CABG revision 30-d mortality = 25% - PCI 30-d mortality = 10.2% - Conservative treatment 30-d mortality = 2.2%
Sharma, 2020	180	111 (62)	38 (21)	43 (24)	98 (55)	- NR
Szavits-Nossan, 2012	55	33 (60)	30 (55)	8 (15)	17 (30)	- NR
Thielmann, 2006	118	67 (57)	25 (21)	15 (13)	27 (23)	- PCI group in-hospital mortality was 12% and 1-y mortality was 20% - CABG revision group in-hospital mortality was 20% and 1-y mortality was 27% - Conservative management in-hospital mortality was 15% and 1-y mortality was 19%

PCI, Percutaneous coronary intervention; CABG, coronary artery bypass grafting; NR, not reported. *All values expressed as n (%). Percentages are calculated as a reflection of all patients undergoing angiography.

colleagues⁸ found that a delay in diagnosis of more than 30 hours was an independent predictor of late mortality in patients with PMI.

SHORT-TERM OUTCOMES

PMI after cardiac surgery is associated with significantly increased postoperative morbidity and mortality.²⁻⁴ In a 2019 analysis of patients undergoing CABG, overall 30-day mortality was 1.4% in patients without PMI and 9.0% in those who developed PMI. Sef and colleagues⁴ noted higher rates of stroke (1.6% vs 2.1%, $P < .001$) and acute kidney injury (12.3% vs 16.0%, $P < .001$) in patients with versus those

without PMI. Likewise, in Biancari and colleagues' meta-analysis,² the pooled early mortality rate of patients with PMI was 8.9% (95% CI, 6.7-11) with individual studies reporting a range of 5.1% to 24%. By comparison, the Society of Thoracic Surgeons' reported mortality rate for patients undergoing isolated CABG is 1.8%.⁵⁶

LONG-TERM OUTCOMES

Although the association of PMI with short-term mortality is clear, the impact of PMI on long-term outcomes is less well documented. However, several analyses have demonstrated higher long-term mortality in patients diagnosed with PMI

versus those without.^{3,57,58} Davierwala and colleagues⁸ reported lower 5-year survival in patients with PMI when compared with those without PMI (75% vs 86%, $P < .001$). However, after excluding in-hospital mortality, the difference in midterm survival was no longer apparent ($P = .09$).

Conversely, in a cohort of 501 patients from Finland of whom 80 had PMI after CABG as diagnosed by cardiac enzyme or ECG criteria, Järvinen and colleagues⁵⁹ found that 1- and 10-year survivals were similar (93% PMI vs 98% no PMI and 73% PMI vs 80% no PMI, respectively).

Most recently, Sharma and colleagues¹⁷ found that long-term mortality in patients with PMI who underwent PCI was significantly lower compared with patients who underwent redo CABG or conservative medical treatment, but they did not evaluate the difference between patients with and without PMI.

CONCLUSIONS

Given the heterogeneity of presentation and etiology, PMI can be difficult to manage, and consequently the need for teamwork in these scenarios is paramount. Although collaboration by the heart team, intuitively and practically, can lead to better outcomes, it must not lead to delays in reintervention.

Overall, although the incidence of PMI is low, it is not insignificant, ranging from 2% to 5% depending on the study and definition used. Because most cases of PMI originate in the OR, the use of adjunctive tests such as TTFM and IFI can provide an opportunity for early detection of graft failure while allowing for corrective operative strategies. There are a number of angiographic and patient characteristics that should raise clinical suspicion of PMI, including those with small and diffusely disease coronary arteries or cases with prolonged crossclamp and cardiopulmonary bypass times. PMI can manifest a wide spectrum of clinical presentations from asymptomatic to sudden hemodynamic collapse. For patients in whom PMI is suspected, conventional coronary angiography offers the most expeditious modality not only for diagnosis but also for potential reintervention. Although inotropic and mechanical support can offer support, they must not be used to avoid urgent angiography. In patients with sudden hemodynamic collapse, immediate return to the OR (or re-sternotomy in the intensive care unit) may be necessary. Likewise, where repeat angiography excludes feasible percutaneous intervention options, redo surgery should be considered.

Conflict of Interest Statement

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