Cardiovascular Imaging Following Perioperative Myocardial Infarction/Injury

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Research Article

Keywords: Cardiovascular Imaging, Perioperative Myocardial Infarction/injury, Patients, mortality, non-cardiac surgery

Posted Date: October 6th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-923505/v1>

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Version of Record: A version of this preprint was published at Scientific Reports on March 15th, 2022. See the published version at <https://doi.org/10.1038/s41598-022-08261-6>.

Abstract

Background: Patients developing perioperative myocardial infarction/injury (PMI) have high mortality. PMI work-up and therapy remain poorly defined.

Methods: In a prospective multicenter study enrolling high-risk patients undergoing major non-cardiac surgery within a systematic PMI screening and clinical response program, the frequency of cardiovascular imaging during PMI work-up and its yield for possible type 1 myocardial infarction (T1MI) was assessed. Automated PMI detection triggered evaluation by the treating physician/ cardiologist on service, who determined selection/timing of cardiovascular imaging. In transthoracic echocardiography (TTE) a new wall motion abnormality within 30 days, in myocardial perfusion imaging (MPI) a new scar or ischemia within 90 days, and in coronary angiography (CA) Ambrose-Type II or complex lesions within 7 days of PMI detection were considered indicative of T1MI.

Results: In patients with PMI, 21% (268/1269) underwent at least one cardiac imaging modality. TTE was used in 13% (163/1269), MPI in 3% (37/1269), and CA in 5% (68/1269). Consultation by a cardiologist was associated with higher use of cardiovascular imaging (27% versus 13%). Signs indicative of T1MI were found in 8% of TTE, 46% of MPI, and in 63% of CA.

Conclusion: Most patients with PMI did not receive any cardiovascular imaging within their PMI work-up. If performed, MPI and CA have high yield for signs indicative of T1MI.

Study registration: <https://clinicaltrials.gov/ct2/show/NCT02573532>

Introduction

With over 300 million surgeries performed annually, perioperative complications are of major medical and economic relevance [1-4]. Perioperative myocardial infarction/injury (PMI) has recently been identified as the most common cardiac complication following non-cardiac surgery and an important contributor to postoperative mortality [1-4]. PMI occurs most often with patients under anaesthesia and high-dose analgesia. Therefore, more than 85% of patients with PMI do not experience typical ischemic symptoms [1-3, 5, 6]. In order to identify these patients at increased risk of death, systematic screening for PMI by measuring cardiac troponin pre- and postoperatively has been endorsed in patients with a high cardiovascular risk [7-9]. Several different pathophysiological processes including type 1 myocardial infarction (T1MI) [7] may lead to PMI. Therefore, detailed clinical assessment, 12-lead ECG, and cardiovascular imaging are required during PMI work-up for identification of the most likely underlying pathophysiology [1-3, 10-14]. This is mandatory for the selection of the most appropriate therapy.

As PMI has only recently been identified as an important clinical entity [1-4], the frequency of different cardiovascular imaging modalities applied after the detection of PMI within a clinical PMI screening program, the impact of cardiology consultation on the selection of cardiovascular imaging modalities

and their yield for possible T1MI are largely unknown. Therefore, we addressed these major uncertainties within a prospective international multicenter study.

Methods

Study Design and Population

This was a secondary analysis within the prospective multicenter observational study Basel-PMI (NCT02573532). The study was carried out according to the principles of the Declaration of Helsinki and was approved by the Ethics committee of Northwestern Switzerland (EKNZ). Between September 2012 and September 2018, we included consecutive patients undergoing non-cardiac surgery at three hospitals (University Hospital Basel, Cantonal Hospital Aarau, both in Switzerland, and the Heart Institute (InCor), University Sao Paulo, Brazil) which were eligible for the institutional routine PMI screening and response program and provided written informed consent to registration into the BASEL-PMI database. We adhered to the STROBE reporting guidelines for observational studies (Supplement Table 1)[15].

The screening and response system for PMI was part of the standard of care for high-risk patients undergoing non-cardiac surgery at the participating hospitals in selected surgical departments. Patients were screened if they had a planned hospital stay exceeding 24h after surgery and were considered at increased cardiovascular risk defined as 65 to 85 years of age, OR 45 to 64 years of age with a history of coronary artery disease (CAD), peripheral arterial disease (PAD), or cerebrovascular disease. For this analysis, patients with PMI adjudicated to be due to primarily extra-cardiac cause such as sepsis, cardiac trauma or pulmonary embolism, as well as PMI due to heart failure and tachyarrhythmia were excluded (supplemental methods).

PMI definition

Plasma concentrations of high-sensitivity cardiac Troponin T (hs-cTnT assay [Elecsys, Roche diagnostics, Mannheim, Germany] in Basel and in Sao Paulo) and sensitive cardiac Troponin I (s-cTnI assay [Siemens Dimension Vista; Siemens Health Care Diagnostics, Tarrytown, NY] in Aarau) were measured before surgery, and on postoperative days 1 and 2 as well as later if clinically indicated. Patients who did not have at least two measurements of the respective cTn assay were excluded. PMI was defined as an absolute increase from preoperative (or between two postoperative measurements if the preoperative measurement was missing) of $\geq 14\text{ng/L}$ for hs-cTnT and $\geq 45\text{ng/L}$ for s-cTnI (the respective 99th percentile of both assays) within three days following surgery.

PMI response and consultation by a cardiologist

On regular working days, PMI patients underwent a 12-lead ECG and a clinical evaluation by the cardiologist on service, unless patients were already under close supervision in the intensive care unit,

where no consultation by a cardiologist was deemed necessary. Based on the evidence available at the time of the study, presence of ST-segment elevation, ST-segment depression, or typical chest pain and consequently elevated troponin-levels were considered suggestive of T1MI and an indication for early coronary angiography after interdisciplinary discussion with the treating surgeon was given. Presence of perioperative hemodynamic instability with hypotension, tachyarrhythmia or severe postoperative anemia were considered suggestive of type 2 myocardial infarction and an indication for the correction of the respective trigger of myocardial supply-demand mismatch [1-3, 5, 6] was given. With the exception of patients who clearly fulfilled the criteria for the Universal definition of Myocardial Infarction for T1MI according to the ESC guidelines[7], given the lack of evidence, no explicit guidance on the selection and/or timing of cardiovascular imaging was provided for the cardiologist on service.

Cardiovascular imaging findings suggestive of T1MI

The cardiovascular imaging modalities were analyzed separately, as one individual patient may have undergone several tests.

Transthoracic echocardiography

New regional wall motion abnormality was interpreted as suggestive of possibly occurred T1MI[7, 16]. Left ventricular ejection fraction (LVEF), using the modified Simpson's method, valve dysfunction and wall motion abnormalities were prospectively recorded. In order to assess a new regional wall motion abnormality, the preoperative echocardiography up to one year before surgery and the postoperative transthoracic echocardiography (TTE) up to 30 days after surgery were evaluated. A new regional wall motion abnormality (dyskinesia/akinesia/hypokinesia) was documented if it was clearly detectable compared to the preoperative echocardiography. In case no preoperative echocardiography was available, a regional wall motion abnormality was assumed to be new in the absence of a history of MI or other medical records indicating reduced LVEF.

Myocardial perfusion imaging

Using 99Tc-labelled single-photon emission computed tomography (SPECT) or ⁸²Rb perfusion positron emission tomography (PET) up to 90 days after surgery, any new scar or new postoperative ischemia was interpreted as suggestive of possibly occurred T1MI[7, 17]. The summed stress scores (SSS) quantified overall perfusion abnormality (scar plus ischemia), whilst the summed difference score (SDS) represented the extent of ischemia[18, 19]. An SSS of 4 or greater was considered as abnormal (minimum of 5% of myocardium affected) and a reversible defect with an SDS of greater than 2 was defined as ischemia (minimum 3% of myocardium is ischemic)[20]. In order to assess new scarring in the postoperative myocardial perfusion imaging (MPI), direct comparison with preoperative imaging up to

one year before surgery was performed. In case no preoperative MPI was available, a scar was assumed new in the absence of a history of MI or other medical records indicating reduced LVEF.

Coronary angiography

Using coronary angiography, the presence of Ambrose's type II lesion or a complex lesion as adjudicated by an experienced interventional cardiologist was interpreted as an indication of a T1MI[10]. Lesions with an obstruction of over 50% were documented. Each lesion was classified based on Ambrose's classification[21, 22]. According to this classifications the lesions were divided into 4 types: Concentric (symmetric and smooth borders and a broad neck), type I eccentric (asymmetric stenosis with smooth narrowing), type II eccentric (asymmetric stenosis with a convex intraluminal obstruction and narrow neck due to overhanging edges or irregular borders, or both) and multiple irregularities (three or more serial, closely spaced narrowing or diffuse irregularities)[21-23]. The complexity of the lesions was also examined. Lesions with a stenosis of more than 50% and at least one of the following morphologies were identified as complex lesions[10, 24]: intraluminal filling defect consistent with thrombus, plaque ulceration, plaque irregularity (haziness, defined by irregular margins or overhanging edges), impaired flow (TIMI flow <3, except chronic total occlusion), Supplement Figure 1 and 2.

Statistical analysis

The data are expressed as medians and interquartile range (IQR) for continuous variables, and for categorical variables as numbers and percentages (%). All variables were compared by Mann Whitney U test for continuous variables and Pearson chi-square or Fisher's exact test for categorical variables, as appropriate.

Analyses were performed at imaging modality level and at patient level. For the patient level analysis, only the most invasive/sophisticated imaging modality per patient was considered (coronary angiography over MPI, MPI over echocardiography). All hypothesis testing was two-tailed, and P values of less than 0.05 were considered to indicate statistical significance. Statistical analyses were performed using IBM SPSS Statistics for Mac, version 26.0 (IBM Corp., Armonk, NY) and R version 3.6.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

From 2012 until 2018, 11'871 consecutive patients were enrolled in the Basel-PMI study, of which 1599 patients developed a PMI. Among these patients, 1269 patients were eligible for this analysis (Figure 1). The median age was 75 years, 39% (497/1269) were women, 45% (565/1269) had known CAD, 12% (156/1269) known stroke/TIA, 36% (459/1269) PAD, 30% (383/1269) diabetes mellitus, and 59% (755/1269) chronic kidney disease (Table1). Accordingly, 53% (661/1269) were on aspirin treatment and 56% (705/1269) on a statin at baseline.

PMI characteristics

Postoperatively, median peak hs-cTnT concentration was 59ng/L (IQR 40, 109), median peak s-cTnI concentration 154ng/L (IQR 97, 429), 12% (136/1269) of patients had ischemic symptoms, 14% (180/1269) ischemic ECG changes.

Cardiac consultation and cardiovascular imaging

Most PMI patients (60%, 757/1269) were consulted by a cardiologist. Patients receiving cardiac consultation had higher peak cTn concentration, more often ischemic symptoms and/or ECG findings compared to patients without cardiac consultation.

Overall, 21% (268/1269) of patients with PMI received at least one cardiovascular imaging modality. Echocardiography was performed in 13% (163/1269) of patients within 30 days, MPI in 3% (37/1269) within 90 days and coronary angiography in 5% (68/1269) of patients within 7 days after surgery (Figure 2.A). Patients receiving cardiac consultation more often received cardiovascular imaging (27% versus 13%, $p<0.001$) versus patients without cardiac consultation (Figure 2.B and 2.C).

Cardiovascular imaging evidence suggestive of T1MI

Echocardiography

Among 198 TTE, a new wall motion abnormality was identified in 15 (8%). These patients had significantly higher hs-cTnT concentrations (median 240 ng/L, IQR [109-504] versus 85 ng/L, IQR [49-162], $p=0.005$) and significantly lower postoperative LVEF (median 45%, IQR [39-55%] vs 55% IQR [45-60%], $p=0.043$) than patients without a new wall motion abnormality (Table 2 and Figure 3.A).

Myocardial perfusion imaging

Among 39 myocardial perfusion scans, 18 (46%) had either evidence of a new scar or evidence of ischemia or both (median SDS 6 [IQR 5-9.5], median SSS 11 [IQR 8-12]). These patients had higher hs-cTnT concentrations and more often a history of heart failure versus patients without scar/ischemia (Table 2 and Figure 3.B).

Coronary angiography

Among 68 coronary angiographies, 43 (63%) had Ambrose's type II or complex lesions (Figure 3.C). Overall, 28 (41%) showed a multi-vessel-disease, 13 (19%) showed a significant stenosis of the left main and in 14 (21%) there was no evidence of a significant CAD (Figure 4). The median time from surgery to angiography was 4 days (IQR 2-6) and median time from detection of PMI to angiography was 2 days (IQR 1-4). Myocardial revascularization was performed in 33 patients (47%). After a positive MPI 3 (8%) patients received coronary angiography within 7 days and 8 patients within 90 days postoperatively.

Discussion

In this large, international, multicenter study prospectively enrolling high-risk patients undergoing non-cardiac surgery in a PMI-screening program, we evaluated the frequency of cardiovascular imaging and its yield for findings suggestive of possibly occurred T1MI, the PMI phenotype with the best documented therapeutic consequences [10, 11, 13, 25, 26]. We report five major findings.

First, patients developing PMI were older patients (median age 75 years) with extensive pre-existing cardiovascular comorbidity, including CAD in 45%, PAD in 36%, diabetes mellitus in 30% and chronic kidney disease in 59%. Second, despite the well documented association between PMI and death within 30-days following non-cardiac surgery,[1-3, 10-14] the vast majority (79%) of patients with detected PMI did not receive any cardiovascular imaging postoperatively. Third, cardiac consultation and large PMI size as quantified by peak cTn seemed to modify the frequency of cardiovascular imaging being performed. Fourth, echocardiography was the most commonly used cardiovascular imaging modality and performed in 13% of patients with PMI, followed by coronary angiography in 5% and MPI in 3%. Fifth, when performed as part of the PMI work-up, the incidence of findings suggestive of possibly occurred T1MI varied widely among the three imaging modalities. The incidence of new wall motion abnormalities with echocardiography was low (8%), of a new scar/ischemia with MPI moderate-to-high (46%), and of Ambrose's type II or complex lesions high (63%) with coronary angiography.

These findings extend and corroborate prior studies on the detection and management of PMI, a silent and neglected killer following non-cardiac surgery [1-3, 5, 6]. PMI predominately affects patients burdened by four factors: high age, high prevalence of end organ damage from atherosclerosis including CAD, stroke, and PAD, but also chronic kidney disease, wounds and impaired mobility following major surgery, and the often-severe underlying non-cardiac disorder that has required major surgery. These complexities substantially increase the challenges associated with the early diagnosis of T1MI. In addition, these complexities also increase the risk of complications associated with cardiovascular imaging modalities, particularly those using contrast agents, but likely decrease the likelihood of benefit from therapeutic interventions specific to T1MI on health-related quality of life and long-term mortality. These considerations may explain at least in part the very low use of cardiovascular imaging following PMI. Underestimation of the substantial risk of 30-day mortality associated with PMI (about 10%) among the treating clinicians as well as the cardiologist on service may also have played a role [1-3, 12]. On the other hand, early diagnosis of T1MI would be essential for the early initiation for evidence-based and ultimately potentially life-saving therapy[16].

This systematic assessment of different cardiovascular imaging modalities following PMI detection within a systematic PMI-screening and clinical response program may help institutions to estimate the imaging work load and cost associated with the implementation of a PMI screening program. It also extends and complements important single-center pilot studies of patients identified clinically with PMI undergoing coronary angiography due to suspected T1MI[10, 11, 13]. Among 120 patients enrolled in Sao Paolo, 45% had Ambrose type 2 lesions versus 57% in a control cohort with spontaneous MI[10]. Among

66 patients enrolled in New York, 26% had evidence of a thrombotic process suggesting T1MI[11]. Among 30 patients enrolled in Hamilton, 13% had evidence of thrombus on optical coherence tomography versus 67% in a control cohort with spontaneous MI[13].

The different frequencies of the cardiovascular imaging modalities investigated seemed to mainly reflect their different availability and ease of use. Appropriate clinical selection of the cardiovascular imaging modalities was further documented by the increasing yield regarding possibly occurred T1MI with increasing invasiveness and cost of the procedures. Given the much higher sensitivity for detecting ischemic myocardium versus echocardiography, MPI seemed underused in this study.

Some limitations warrant consideration when interpreting these findings. First, as it is a retrospective assessment of cardiovascular imaging performed according to the clinical decision of the treating physician, referral bias rather than method-inherent characteristics should be considered the main determinant for the different yields for signs of T1MI observed, as only a subgroup of patients (only patients who were referred for any type of cardiovascular imaging from the attending cardiologist) were examined. Second, while clinically well-established and useful, all investigated cardiovascular imaging techniques have inherent limitations regarding the detection of acute atherothrombosis as the pathophysiological signature underlying T1MI. However, complex lesions morphology and Ambrose's type II lesions were found to be strongly correlated with plaque rupture[10, 27, 28]. Moreover, the 4th universal definition of MI lists new wall motion abnormalities and loss of viable myocardium as definite criteria in conjunction with an increase in cTn[7] as associated with T1MI. Third, the average time from PMI to angiography was 2 days (IQR 1,4). Possible thrombus formation with autolysis could have occurred, possibly leading to an underestimation of T1MI signs.

In conclusion, only about one out of five patients developing PMI received cardiovascular imaging for PMI work-up. If performed, MPI and CA have high yield for signs indicative of possibly occurred T1MI.

Declarations

Funding and role of funders

This study was funded by the University Basel, the University Hospital Basel, the Swiss Heart Foundation, Roche Diagnostics, Abbott, Astra Zeneca, the PhD Educational Platform for Health Sciences, the Forschungsfond Kantonsspital Aarau, and the Cardiovascular Research Foundation Basel. Fundação de Amparo à Pesquisa do Estado de São Paulo, Brazil [FAPESP grant number 2015/ 23731-6];

The funders had no role in the design, data collection, statistical analysis, writing of this manuscript, or decision to publish.

Conflict of interest

Dr. Arslani has received a research grant from the Swiss Academy of Medical Sciences and the Bangerter Foundation (YTCR 09/19). Dr. Puelacher reports grants from PhD Educational Platform for Health Sciences, Roche Diagnostics and the University Hospital Basel during the conduct of the study. Dr. Gualandro has received research grants from FAPESP (Sao Paulo Research Foundation) for the submitted work, grants from the Swiss Heart Foundation and consulting honoraria from Roche, outside the submitted work. Dr. Mueller reports grants from the Swiss Heart Foundation and grants and non-financial support from several diagnostic companies during the conduct of the study, as well as grants, personal fees and non-financial support from several diagnostic companies outside the submitted work. Dr. Kindler reports grants from the Forschungsfond Kantonsspital Aarau during the conduct of the study. Dr. Blum reports a grant from the Mach-Gaensslen Foundation outside the submitted work. All other authors report no conflicts of interest.

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Tables

Table 1: Baseline characteristics in patients with cardiac PMI				
	All cardiac PMI* n =1269	With cardiology consultation n=757	Without cardiology consultation n=512	p-value
Age - median [IQR[†]]	75 [69, 81]	75 [69, 80]	76 [70, 81]	0.003
Women - no. (%)	497 (39)	286 (38)	211 (41)	0.242
Risk factors and History - no. (%)				
Hypertension	947 (75)	583 (77)	364 (71)	0.021
Diabetes				0.339
NIDDM [‡]	203 (16)	130 (17)	73 (14)	
IDDM [§]	180 (14)	103 (14)	77 (15)	
Coronary artery disease	565 (45)	340 (45)	225 (44)	0.777
History of myocardial infarction	281 (25)	152 (25)	129 (25)	0.895
Previous stroke/TIA	156 (12)	99 (13)	57 (11)	0.343
Atrial Fibrillation	277 (25)	133 (22)	144 (28)	0.014
Chronic heart failure	245 (19)	150 (20)	95 (19)	0.627
Peripheral artery disease	459 (36)	331 (44)	128 (25)	<0.001
COPD [¶]	221 (18)	102 (14)	119 (23)	<0.001
Chronic kidney disease	755 (59)	430 (57)	325 (63)	0.020
Preoperative Medication - no. (%)				
ASA [#]	661 (53)	433 (59)	228 (45)	<0.001
P2Y12 Inhibitors	112 (10)	59 (10)	53 (10)	0.964
β- Blocker	621 (49)	390 (52)	231 (45)	0.029
Statin	705 (56)	454 (60)	251 (49)	<0.001
ACEI/ARB ^{**}	589 (46)	366 (48)	223 (44)	0.105
Surgery - no. (%)				<0.001
Orthopedic/Traumatology	382 (30)	233 (31)	149 (29)	
Visceral	104 (8)	44 (6)	60 (12)	
Vascular	379 (30)	275 (36)	104 (20)	
Thoracic	114 (9)	46 (6)	68 (13)	
Spinal	162 (13)	97 (13)	65 (13)	
Urologic	102 (8)	49 (6)	53 (10)	
Other	26 (2)	13 (2)	13 (3)	
ESC/ESA risk^{††} - no. (%)				0.947
low	230 (18)	135 (18)	95 (19)	
medium	778 (61)	466 (62)	312 (61)	
high	261 (21)	156 (21)	105 (21)	
Revised cardiac risk score - no. (%)				0.346
I	331 (26)	203 (27)	128 (25)	
II	424 (33)	239 (32)	185 (36)	
III	290 (23)	174 (23)	116 (23)	

IV	224 (18)	141 (19)	83 (16)	
Standard evaluation for PMI - no. (%)				
Ischemic symptoms	136 (12)	102 (16)	34 (7)	<0.001
Ischemic ECG findings	180 (14)	153 (26)	27 (18)	0.045
Peak Troponin - median [IQR]				
S-cTnI ^{††}	154.0 [97, 429]	179.5 [107, 730]	120.0 [81, 227]	0.004
Hs-cTnT Troponin T ^{§§}	59.0 [40, 109]	61.0 [41, 124]	56.0 [38, 93]	0.001

Table legend: Hs-cTnT was only measured in patients from the University Hospital Basel, Switzerland and INCOR Heart institute, Sao Paulo, Brasil; S-cTnI was only measured in patients from the Cantonal Hospital Aarau, Switzerland;

*PMI: perioperative myocardial infarction/injury; [†] IQR: interquartile range; [‡] NIDDM: Non-insulin-dependent diabetes mellitus, [§] IDDM: insulin-dependent diabetes mellitus; ^{||} TIA: transient ischemic attack; [¶] COPD: chronic obstructive pulmonary disease; [#] ASA: acetylsalicylic acid; ^{**} ACEI/ARB: angiotensin-converting enzyme inhibitor/ angiotensin receptor blocker; ^{††} ESC/ESA: European society of cardiology/European society of anesthesiology; ^{††} S-cTnI: sensitive cardiac troponin I; ^{§§} Hs-cTnT: high-sensitivity cardiac troponin T

Table Legend: Hs-cTnT was only measured in patients from the University Hospital Basel, Switzerland and INCOR Heart institute, Sao Paulo, Brasil; S-cTnI was only measured in patients from the Cantonal Hospital Aarau, Switzerland;

*T1MI: Type 1 myocardial infarction; [†] IQR: interquartile range; [‡] NIDDM: Non-insulin-dependent diabetes mellitus, [§] IDDM: insulin-dependent diabetes mellitus; ^{||} TIA: transient ischemic attack; [¶] COPD: chronic obstructive pulmonary disease; [#] ASA: acetylsalicylic acid; ^{**} ACEI/ARB: angiotensin-converting enzyme inhibitor/ angiotensin receptor blocker; ^{††} ESC/ESA: European society of cardiology/European society of anesthesiology; ^{††} S-cTnI: sensitive cardiac troponin I; ^{§§} Hs-cTnT: high-sensitivity cardiac troponin T; ^{||||} NA: not available

Figures

Table 2: Evidence suggestive of T1MI* in different cardiac imaging modalities									
	Coronary angiography n = 68			Myocardial perfusion imaging/ Positron emission tomography n = 39			Transthoracic echocardiography n = 198		
	T1MI criteria present	T1MI criteria absent	p- value	T1MI criteria present	T1MI criteria absent	p- value	T1MI criteria present	T1MI criteria absent	p- value
	(n = 43)	(n = 25)		(n = 18)	(n = 21)		(n = 15)	(n = 184)	
Age - median [IQR [†]]	74.0 [70.5, 77.5]	71.0 [68.0, 74.0]	0.056	72.5 [71.0, 74.8]	72.0 [69.0, 79.0]	0.366	79.0 [74.5, 82.5]	76.0 [70.0, 81.0]	0.236
Female gender - no. (%)	9 (21)	8 (32)	0.468	5 (28)	5 (24)	1.000	7 (47)	67 (36)	0.608
Risk factors and History - no. (%)									
Hypertension	33 (77)	19 (76)	1.000	16 (89)	18 (86)	1.000	9 (60)	137 (74)	0.361
Diabetes	17 (39)	8 (30)	0.605	9 (50)	7 (33)	0.615			0.553
1. NIDDM [‡]	11 (26)	5 (20)		5 (28)	4 (19)		1 (7)	27 (15)	
1. IDDM [§]	6 (14)	2 (8)		4 (22)	3 (14)		4 (27)	33 (18)	
Coronary artery disease	26 (60)	13 (52)	0.670	10 (56)	8 (38)	0.442	6 (40)	90 (49)	0.692
History of myocardial infarction	10 (28)	8 (38)	0.608	5 (28)	6 (29)	1.000	1 (7)	53 (29)	0.074
Previous stroke/TIA	8 (19)	2 (8)	0.304	1 (6)	5 (24)	0.190	0 (0)	21 (11)	0.376
Atrial Fibrillation	4 (11)	2 (10)	1.000	4 (22)	4 (19)	1.000	3 (20)	41 (22)	1.000
Chronic heart failure	9 (21)	4 (16)	0.754	6 (33)	1 (5)	0.035	3 (20)	47 (26)	0.765

Peripheral artery disease	23 (53)	12 (48)	0.853	7 (39)	5 (24)	0.488	6 (40)	58 (32)	0.698
COPD [†]	4 (9)	3 (12)	0.702	3 (17)	1 (5)	0.318	2 (13)	32 (17)	1.000
Renal failure	23 (53)	12 (48)	0.853	12 (67)	13 (62)	1.000	9 (60)	115 (62)	1.000
Preoperative Medication – no. (%)									
ASA [#]	30 (75)	17 (68)	0.742	9 (50)	12 (57)	0.901	8 (53)	95 (52)	1.000
P2Y12 Inhibitors	2 (7)	2 (9)	1.000	1 (6)	2 (10)	1.000	1 (7)	15 (8)	1.000
β- Blocker	27 (63)	12 (48)	0.350	8 (44)	9 (43)	1.000	8 (53)	84 (46)	0.761
Statin	23 (53)	15 (60)	0.789	11 (61)	9 (43)	0.415	4 (27)	90 (49)	0.113
ACEI/ARB**	18 (42)	12 (48)	0.812	15 (83)	11 (52)	0.051	7 (47)	85 (46)	1.000
Surgery – no. (%)			0.304			0.403			0.284
Orthopedic/Traumatology	12 (28)	5 (20)		5 (28)	5 (24)		4 (27)	72 (39)	
Visceral	0 (0)	2 (8)		NA	NA		4 (27)	13 (7)	
Vascular	24 (5)	10 (40)		4 (22)	3 (14)		4 (27)	42 (23)	
Thoracic	2 (5)	2 (8)		1 (6)	3 (14)		1 (7)	15 (8)	
Spinal	3 (7)	3 (12)		7 (39)	4 (19)		2 (13)	24 (13)	
Urologic	1 (2)	2 (8)		1 (6)	5 (24)		0 (0)	15 (8)	
Other	1 (2)	1 (4)		0 (0)	1 (5)		0 (0)	3 (2)	
ESC/ESA risk^{††} – no. (%)			0.892			0.908			0.389
low	7 (16)	5 (20)		4 (22)	6 (29)		1 (7)	39 (21)	
medium	23 (53)	12 (48)		11 (61)	12 (57)		10 (67)	111 (60)	
high	13 (30)	8 (32)		3 (17)	3 (14)		4 (27)	34 (18)	
Revised cardiac risk score – no. (%)			0.639			0.907			0.682
I	8 (19)	8 (32)		4 (22)	7 (33)		5 (33)	46 (25)	
II	12 (28)	7 (28)		6 (33)	6 (29)		4 (27)	64 (35)	
III	14 (33)	6 (24)		6 (33)	6 (29)		2 (13)	40 (22)	
IV	9 (21)	4 (16)		2 (11)	2 (10)		4 (27)	34 (18)	
Standard evaluation for									

PMI – no. (%)									
Ischemic symptoms	14 (33)	9 (36)	0.981	4 (22)	3 (14)	0.682	4 (27)	36 (20)	0.508
Ischemic ECG findings	26 (60)	8 (33)	0.061	3 (18)	3 (15)	1.000	5 (42)	45 (31)	0.521
Peak Troponin – median (IQR)									
1. S-cTnI ^{††}	745.0 [192, 2001]	539.5 [125, 1262]	0.561	NA ^{lll}	NA		NA	183.0 [127.0, 803.0]	NA
1. Hs-cTnT Troponin T ^{§§}	238.5 [108, 738]	124.0 [86, 518]	0.299	108.5 [77, 201]	66.0 [46, 137]	0.071	240.0 [109, 504]	85.0 [49, 162]	0.003

Figure 1

Patient Flowchart Patient flowchart showing number of patients assessed, number of patient excluded and reasons for exclusion. PMI: perioperative myocardial infarction/injury; T1MI: Type 1 myocardial infarction

Figure 2

Patient level analysis: Cardiac imaging modalities in cardiac PMI. A. All patients. B. Patients with no cardiology consultation. C. Patients with cardiology consultation. Pie chart showing the frequency of cardiac imaging modalities in general and in patients with and without cardiology consultation. MPI: myocardial perfusion imaging. TTE: transthoracic echocardiography.

Figure 3

Imaging modality level analysis. A. Transthoracic echocardiography. B. Myocardial perfusion imaging. C. Coronary angiography. Pie chart showing the yield for signs of type 1 MI in different cardiac imaging modalities. MI: Myocardial infarction.

68 patients receiving coronary angiography

Coronary artery disease

4 patients with 3-vessel CAD
24 patients with 2-vessel CAD
25 patients with 1-vessel CAD
14 patients with no CAD
13 patients with left main

129 Lesions were analyzed

Ambrose Score

40 (31%) concentric lesions
24 (19%) eccentric type 1 lesions
34 (26%) eccentric type 2 lesions
31 (24%) multiple irregularities lesions

Complex lesions

29 (22%) lesions with totally occlusion
7 (5%) lesions with thrombus
27 (21%) lesions with ulceration
40 (31%) lesions with irregularities
8 (6%) lesions with impaired flow (TIMI <3)

Figure 4

Details of coronary angiography. Details and summary of information collected from coronary angiography records. CAD: coronary artery disease. TIMI: Thrombolysis in Myocardial Infarction.

Supplementary Files

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- [SupplementMaterialCardiacimagingfinal.docx](#)