Mechanical Complications of Myocardial Infarction

Retrospective Analysis Focusing on Frailty, Nutrition, and Clinical Implication of Surgery

Riku Arai,¹ MD, Daisuke Fukamachi,¹ MD, Yasunari Ebuchi,¹ MD, Suguru Migita,¹ MD, Tomoyuki Morikawa,¹ MD, Masaki Monden,¹ MD, Takehiro Tamaki,¹ MD, Keisuke Kojima,¹ MD, Naotaka Akutsu,¹ MD, Nobuhiro Murata,¹ MD, Daisuke Kitano,¹ MD and Yasuo Okumura,¹ MD

Summary

In this retrospective observational study, we have examined the incidence, characteristics, and treatment of serious myocardial infarction (MI)-associated mechanical complications (MCs) occurring in Japanese patients in this era of percutaneous coronary intervention (PCI), focusing on frailty, nutrition, and clinical implication of surgery. Included were 883 patients who, having suffered an MI, had been admitted to Nihon University Hospital between January 2013 and April 2020. Fifteen (1.70%) of these patients had suffered a potentially catastrophic MC—ventricular free wall rupture (VFWR, $n = 8$), ventricular septal rupture (VSR, $n = 6$), or papillary muscle rupture (PMR, $n = 1$). Factors associated with the MCs were age, poor nutritional status, a high Killip class, delayed diagnosis of MI, a high lactate concentration, a low thrombolysis in myocardial infarction flow grade, and single-vessel disease. Thirty-day mortality among MC patients was 60% (9/15): 87.5% associated with VFWR, 33.3% associated with VSR and 0% associated with PMR. On adjusted multivariate analysis, occurrence of an MC was independently associated with 30-day mortality. Despite a high surgical risk (EuroSCORE II: 11.8 ± 4.7) with less frailty, 30-day mortality was lower among patients whose MC was treated surgically than among those whose MC was treated conservatively (40.0% versus 100.0%, respectively; $P = 0.044$).

Our data suggest that surgical intervention can save patients with a life-threatening MI-associated MC and should be considered, if they are not particularly frail.

(Int Heart J 2021; 62: 499-509)

Key words: Papillary muscle rupture, Ventricular free wall rupture, Ventricular septal rupture

Myocardial infarction (MI) can give rise to serious mechanical complications (MCs), the most catastrophic of which are ventricular free wall rupture (VFWR), ventricular septal rupture (VSR), and papillary muscle rupture (PMR). Although the incidence of MCs has decreased due to the development of optimal medical therapies and reperfusion therapy, associated mortality rates have remained high. Several studies from Western countries¹⁻⁵⁾ and Japan⁶⁻¹⁰⁾ have shown the MCs complicating MI in this era of percutaneous coronary intervention (PCI), but most of these studies only indicated the descriptive results of its incidence, treatment, and poor outcomes. In the superaged society in Japan, the proportion of older patients with MI is dramatically increasing.¹¹⁾ It is thus clinically important for the management in the elderly patients to consider specific physiologic condition such as sarcopenia, frailty, or nutrition. That is because those conditions worsen morbidity and mortality in cardiovascular disease.¹²⁾ Nonetheless, there was lacking data regarding the clinical significance of frailty or nutritional status on outcome in MCs associated with MI. With this

in mind, we conducted a comparative, retrospective observational study to investigate the incidence, treatment, and outcomes of patients with an MI-associated MC treated at a single center in Japan; in particular, we focused on frailty, nutrition level, and clinical implication of surgery in those patients.

Methods

Study patients: In total, we have included 883 patients in this study after meeting the inclusion criteria as follows: those who have suffered an MI and had been admitted to Nihon University Hospital, Tokyo, Japan, between January 2013 and April 2020. All patients included in this study had been admitted within 30 days of MI symptom onset. Patients included were identified through a review of the records of consecutive patients admitted for MI, and all had consented, by the opt-out method, to use their data for study purposes.

The study protocol was approved by the Ethics Committee of Nihon University Itabashi Hospital (RK-200714-

From the ¹Division of Cardiology, Department of Medicine, Nihon University School of Medicine, Tokyo, Japan.
Address for correspondence: Yasuo Okumura, MD, Division of Cardiology, Department of Medicine, Nihon University School of Medicine, Oyaguchijomachi 30-1, Itabashi-ku, Tokyo 1738610, Japan. E-mail: okumura.yasuo@nihon-u.ac.jp
Received for publication August 23, 2020. Revised and accepted November 12, 2020.
Released in advance online on J-STAGE May 15, 2021.
doi: 10.1536/ihj.20-595
All rights reserved by the International Heart Journal Association.

10) and was in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Data collection and definitions: Information from patients' clinical records had been entered anonymously into an Excel spreadsheet by physicians or the clinical research coordinator at Nihon University Hospital. For the purpose of the study, we have obtained the following data: patients' clinical characteristics; features of the MI, including MI class, thrombolysis in myocardial infarction (TIMI) flow grade; information pertaining to any MC, including type of MC, means by which the MC was diagnosed, treatment of the MC (whether surgical or conservative treatment), and treatment outcomes.

The definition of TIMI flow was graded as follows: TIMI 0 = no perfusion, TIMI 1 = penetration without perfusion, TIMI 2 = partial perfusion, or TIMI 3 = complete perfusion (as has been described for the Phase I TIMI Trial).¹³⁾

MI had been diagnosed on the basis of patients' symptoms and a serum troponin-I concentration > 0.014 ng/mL with or without > 2 mm ST segment elevation in the precordial leads or > 1 mm ST segment elevation in the limb leads on electrocardiography (ECG). The infarction was classified as ST segment elevation MI (STEMI) or non-ST segment elevation MI (NSTEMI), depending on the presence or absence of ST segment elevation.¹⁴⁾ The MI was also classified as acute (AMI), recent (RMI), or old (OMI), depending on the time from symptom onset to diagnosis, whether < 24 hours, 24 hours to 30 days, or > 30 days, respectively. MC was defined as any spontaneous rupture of the myocardium following MI and was classified by type as VFWR, VSR, or PMR, according to the area where the rupture has reportedly occurred.¹⁵⁾ VFWR was defined, in some cases, as the occurrence of electromechanical dissociation or sudden hemodynamic compromise associated with pericardial effusion (> 1 cm) and cardiac tamponade (identified echocardiographically) or hemopericardium (also identified echocardiographically) requiring pericardiocentesis. Otherwise, VFWR was identified upon surgery or at autopsy. VSR was defined as Doppler echocardiography depiction of left to right shunt flow across the septal defect after MI, oximetric detection of a step-up of > 5% from the right atrium to the right ventricle, or surgical closure of the septal defect. PMR was diagnosed on Doppler echocardiography or identified during the surgery. Echocardiography was performed at the time of admission for MI and when an MC was suspected at the discretion of the attending physician.

The following cardiovascular risk factors were assessed: hypertension (defined as use of an antihypertensive agent, systolic blood pressure $\geq$ 140 mmHg, or diastolic blood pressure $\geq$ 90 mmHg), diabetes mellitus (defined as use of an oral hypoglycemic agent or insulin or a glycosylated hemoglobin level of $\geq$ 6.5%), and dyslipidemia (defined as use of a statin or triglyceride-lowering drug, a low-density lipoprotein concentration of $\geq$ 140 mg/dL, a high-density lipoprotein concentration of < 40 mg/dL, or a triglyceride concentration of $\geq$ 150 mg/dL). Body mass index (BMI), calculated as weight in kilograms divided by

height in meters squared, was also assessed.¹⁶⁾ Heart failure, if present at the time of initial presentation, was classified by severity according to the Killip system.³⁾ Patients' nutrition-related risk was also assessed on the basis of the Geriatric Nutritional Risk Index (GNRI), which is a simple tool often used in elderly patients; the lower the GNRI, the worse the patient's nutritional status.¹⁷⁾ Nutritional risk was classified as major-moderate, low, or none, depending on whether the GNRI was < 92, 92-98, or > 98, respectively, as previously reported.¹⁸⁾

Coronary angiography (CAG) was performed in consenting patients during their hospitalization for MI. If PCI was performed during the same session as the index CAG, it was defined as ad hoc PCI. When the culprit coronary vessel showed TIMI grade 0 flow, the degree of collateral circulation (assessed by contrast echocardiography) was then graded according to the Rentrop system as follows: grade 0 = no collateral circulation, grade 1 = filling of side branches of the artery to be dilated via collateral channels without visualization of the epicardial segment, grade 2 = partial filling of the epicardial segment via collateral channels, or grade 3 = complete filling of the epicardial segment of the artery being dilated via collateral channels.¹⁹⁾

EuroSCORE II²⁰⁾ and JapanSCORE²¹⁾ scores obtained at the time of MC diagnosis were also assessed, as were Canadian Study of Health and Aging Clinical Frailty Scale (CSHA-CFS) scores.²²⁾ Possible CSHA-CFS scores are 1 (very fit), 2 (well without active disease), 3 (well with treated comorbid disease), 4 (apparently vulnerable), 5 (mildly frail), 6 (moderately frail), 7 (severely frail), 8 (very severely frail), and 9 (terminally ill). A CSHA-CFS score $\geq$ 6 indicates that help is needed with both instrumental and non-instrumental activities of daily living.¹¹⁾

Evaluations and study endpoint: We first divided the 883 study patients into 2 groups: those who had suffered an MC (MC group, $n = 15$) and those who had not (No MC group, $n = 868$). We then compared the patient characteristics between the two groups. We then assessed the characteristics of patients in the MC group. Once we had done that, we divided patients in the MC group between those whose MC was treated surgically ($n = 10$) and those whose MC was treated by conservative therapy ($n = 5$); we then compared characteristics and treatment outcomes between these two groups of patients.

The main study endpoint was 30-day mortality, which has been defined as in-hospital death from any cause within 30 days after admission, which was ascertained through our review of the patient data. Thus, we compared 30-day mortality between patients in the MC group and those in the No MC group and then between patients with an MC treated surgically and those with an MC treated conservatively.

Statistical analysis: Continuous variables are shown as mean $\pm$ SD values, while differences between groups were analyzed via Student's *t*-test or Mann-Whitney *U* test. Categorical variables are presented as the number and percentage of patients, and between-group differences in these variables were analyzed using chi-square or Fisher's exact test. Thirty-day mortality among patients with and without an MC and among patients with an MC treated

surgically and those with an MC treated conservatively was described via a bar graph, and between-group differences were analyzed using Fisher's exact test. Independent association between MC and 30-day mortality was tested by means of univariate and multivariate logistic regression analyses by forced entry method in each multivariate model. Five multivariate models were then created, with male sex included in each one. The other factors included were all shown by univariate analysis to be significant. The models were as follows: Model 1, adjusted for presence of an MC, age, male sex, GNRI, hypertension, and dyslipidemia; Model 2, adjusted for presence of an MC, age, male sex, GNRI, systolic blood pressure, and heart rate; Model 3, adjusted for presence of an MC, age, male sex, GNRI, Killip class, and left ventricular ejection fraction (LVEF); Model 4, adjusted for presence of an MC, age, male sex, GNRI, hemoglobin, and lactate concentrations; and Model 5, adjusted for presence of an MC, age, male sex, GNRI, left main trunk as the culprit vessel and treatment by PCI. All analyses were performed with SPSS Statistics 19.0 (SPSS Inc., Chicago, IL, USA), and $P < 0.05$ was considered significant.

Results

Comparison of patients in the MC group and patients in the No MC group: Clinical characteristics, CAG findings, and therapeutic interventions of patients in the MC group and of those in the No MC group are shown in Table I. Patients in the MC group were significantly older than those in the No MC group (76.1 ± 8.3 years versus 67.8 ± 13.6 years, respectively; $P = 0.002$), and the GNRI was found to be lower in patients in the MC group than in patients in the No MC group (92.8 ± 12.9 versus 101.6 ± 13.2 , $P = 0.015$). Five patients (33.3%) in the MC group presented in cardiogenic shock (Killip class IV), whereas 679 (79.3%) patients in the No MC group had no sign of heart failure (Killip class I) ($P < 0.001$). Time from symptom onset to diagnosis of MI of > 24 hours was significantly more prevalent in the MC group than in the No MC group (53.3% versus 19.2%, $P = 0.004$). No significant between-group difference was noted in LVEF, and the only laboratory values that differed were the lactate and albumin concentrations (Table I).

There was no significant difference in the occluded (culprit) vessel between the two groups, although the right coronary artery was less frequently involved in the MC group (13.3% versus 36.5%, $P = 0.064$). TIMI flow 0 on initial CAG was significantly more common in the MC group than in the No MC group (93.3% versus 53.1%, $P = 0.018$), and single-vessel disease was also significantly more common in this group (60.0% versus 21.5%, $P = 0.002$). Ad hoc PCI was performed less frequently in the MC group than in the No MC group (60.0% versus 93.8%, $P < 0.001$), and a drug eluting stent was used less frequently in this group (60.0% versus 83.5%, $P = 0.025$). TIMI grade 3 flow was achieved after PCI in a significantly smaller percentage of patients in the MC group than in the No MC group (33.3% versus 96.9%, $P < 0.001$). Intra-aortic balloon pumping (IABP) and VA-ECMO (veno-arterial extracorporeal membrane oxygena-

tion) were used significantly more frequently in the MC group than in the No MC group (IABP: 80.0% versus 31.8%, $P < 0.001$; VA-ECMO: 53.3% versus 6.1%, $P < 0.001$).

Clinical characteristics of patients in the MC group and details of the MCs: With 15 of the 883 patients having suffered an MI-associated MC, the incidence of such MCs was calculated to be 1.70%. Clinical characteristics of the 15 MC patients are shown in Tables II, III. Of the 15 patients who suffered an MC, 8 (0.91%) suffered a VFWR, 6 (0.68%) suffered a VSR, and 1 (0.11%) suffered a PMR. For each patient with an MC and for whom frailty was evaluated ($n = 9$), the CSHA-CFS score was < 6 . GNRI was available for 14 of the 15 patients: 8 (57.1%) patients had a GNRI < 92 , 3 (21.4%) had a GNRI of 92–98, and 3 (21.4%) had a GNRI > 98 . Eight patients (53.3%) suffered the MC in association with an RMI. In 8 (53.3%) of these 15 patients, the MC reportedly occurred within 48 hours (on Day 1 or Day 2) after MI symptom onset, whereas in 4 patients (26.6%), the MC occurred 1 week later, and in 1 (Patient 10), the MC occurred on Day 17.

Although echocardiography was the most effective means for MC diagnosis, the clinical manifestations that led to a suspicion of MC were specific to the type of MC. Typically, the initial manifestation of VFWR was sudden pulseless electrical activity (PEA), i.e., electromechanical dissociation on arterial blood pressure and ECG monitoring. The typical manifestation of VSR was a new pansystolic murmur heard during a patient round and/or exacerbation of heart failure despite intensive treatment. In our single case of PMR, sudden-onset pulmonary edema was observed during PCI, leading to cardiogenic shock that could not be explained from the culprit lesion, i.e., from occlusion of the distal left circumflex artery.

In some cases, not only was the main coronary artery occluded; a side branch, i.e., the diagonal, obtuse marginal, or posterolateral branch, was also determined to be occluded. In all but one case (Patient 12), there was no flow through the culprit lesion (TIMI grade 0 or 1 flow). TIMI grade 3 flow was achieved by PCI in 3 (33.3%) cases.

EuroSCORE II scores at the time of MC occurrence were high at 13.4 ± 6.3 . The MC was treated surgically within 24 hours after its diagnosis in 4 of 4 patients (100%) with LVFWR, in 3 of 5 patients (60.0%) with VSR, and in the patient (100%) with PMR. Thirty-day mortality of patients with an MC was 60% (9/15)—87.5% (7/8) for those with VFWR, 33.3% (2/6) for those with VSR, and 0% (0/1) for the patient with PMR.

Association between MI-related MC and 30-day mortality: Follow-up time was 28.3 ± 6.3 days. Overall 30-day mortality was 7.5% (i.e., 66 patients died within 30 days of admission for MI). Nine of the deaths (13.6%) were determined to be MC-related deaths. Thirty-day mortality of patients in the MC group was 60% (9/15), which was deemed to be significantly higher than that of patients in the No MC group (60.0% versus 6.6%, $P < 0.001$) (Figure 1), with an odds ratio (OR) of 19.53 (95% confidence interval [CI]: 6.71–56.79; $P < 0.001$) for 30-day mortality in the MC group (versus No MC group) (Table

Table I. Characteristics, CAG Findings, and Therapeutic Interventions of Patients in the MC Group and No MC Group

	MC (<i>n</i> = 15)	No MC (<i>n</i> = 868)	<i>P</i> -value
Age (years)	76.1 ± 8.3	67.8 ± 13.6	0.002
Male sex	13 (86.7%)	685 (78.9%)	0.749
Body mass index (kg/m ²)	23.1 ± 4.4	23.7 ± 3.8	0.571
GNRI	92.8 ± 12.9	101.6 ± 13.2	0.015
Coronary risk factors			
Hypertension	7 (46.7%)	547 (63.5%)	0.179
Diabetes mellitus	4 (26.7%)	288 (33.4%)	0.581
Dyslipidemia	5 (33.3%)	422 (49.0%)	0.230
Current smoking	9 (64.3%)	540 (64.2%)	0.995
History of stroke	2 (13.3%)	70 (8.1%)	0.354
History of PCI	2 (13.3%)	125 (14.5%)	1.000
History of CABG	0	14 (1.6%)	1.000
Clinical presentation			
Systolic blood pressure (mmHg)	123.4 ± 32.4	126.8 ± 47.3	0.789
Diastolic blood pressure (mmHg)	72.1 ± 16.9	79.0 ± 24.8	0.290
Heart rate (bpm)	93.8 ± 20.5	83.5 ± 24.8	0.106
Body temperature (°C)	36.4 ± 0.7	36.1 ± 0.9	0.273
Respiratory rate (breaths/minute)	21.9 ± 7.7	20.8 ± 8.4	0.617
Killip class			< 0.001
I	6 (40.0%)	679 (79.3%)	
II	1 (6.7%)	85 (9.9%)	
III	3 (20.0%)	29 (3.4%)	
IV	5 (33.3%)	63 (7.4%)	
Time from symptom onset to diagnosis of MI			
0–6 hours	4 (26.7%)	510 (58.8%)	0.012
6–12 hours	1 (6.7%)	100 (11.5%)	1.000
12–24 hours	2 (13.3%)	85 (9.8%)	0.652
> 24 hours	8 (53.3%)	167 (19.2%)	0.004
Unknown	0	13 (1.5%)	1.000
Electrocardiographic diagnosis			
ST-segment elevation MI	15 (100.0%)	643 (74.1%)	0.016
Non-ST-segment elevation MI	0	225 (25.9%)	0.016
Left ventricular ejection fraction (%)	51.8 ± 15.5	48.9 ± 13.7	0.500
Results of initial laboratory tests			
Lactate (mmol/L)	5.3 ± 3.9	2.8 ± 3.4	0.010
Hemoglobin (g/dL)	12.3 ± 1.9	13.4 ± 2.4	0.085
Albumin (mg/dL)	3.3 ± 0.6	3.8 ± 0.6	0.004
Creatinine (mg/dL)	1.1 ± 0.3	1.4 ± 1.8	0.483
CAG findings			
CAG performed	15 (100.0%)	851 (98.0%)	1.000
Occluded (culprit) vessel			
Left main trunk	0	25 (2.9%)	1.000
Left anterior descending coronary artery	9 (60.0%)	408 (47.0%)	0.318
Left circumflex coronary artery	4 (26.7%)	95 (10.9%)	0.077
Right coronary artery	2 (13.3%)	317 (36.5%)	0.064
Graft	0	5 (0.6%)	1.000
Pre-interventional TIMI flow grade			0.018
0	14 (93.3%)	451 (53.1%)	
1	0	208 (24.5%)	
2	0	93 (10.9%)	
3	1 (6.7%)	98 (11.5%)	
Therapeutic Interventions			
Single-vessel disease	9 (60.0%)	187 (21.5%)	0.002
Ad hoc PCI	9 (60.0%)	814 (93.8%)	< 0.001
POBA	3 (27.3%)	68 (8.5%)	0.064
Bare-metal stent	0	62 (7.8%)	1.000
Drug-eluting stent	6 (60.0%)	665 (83.5%)	0.025

Table I. Characteristics, CAG Findings, and Therapeutic Interventions of Patients in the MC Group and No MC Group (continued)

	MC (<i>n</i> = 15)	No MC (<i>n</i> = 868)	<i>P</i> -value
Post-interventional TIMI flow grade			< 0.001
0	0	10 (1.2%)	
1	3 (33.3%)	3 (0.4%)	
2	3 (33.3%)	12 (1.5%)	
3	3 (33.3%)	780 (96.9%)	
Cardiac surgery	10 (66.7%)	23 (2.7%)	< 0.001
Mechanical circulatory support			
IABP	12 (80.0%)	276 (31.8%)	< 0.001
VA-ECMO	8 (53.3%)	53 (6.1%)	< 0.001
Impella support	0	12 (1.4%)	1.000

Mean $\pm$ SD values or number (%) of patients are shown by Student's *t*-test, Mann-Whitney *U* test, chi-square test, or Fisher's exact test. CABG indicates coronary artery bypass graft; CAG, coronary angiography; GNRI, Geriatric Nutritional Risk Index; IABP, intra-aortic balloon pump; MC, mechanical complication; MI, myocardial infarction; PCI, percutaneous coronary intervention; POBA, plain old balloon angioplasty; TIMI, thrombolysis in myocardial infarction; and VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

Table II. Clinical Details of the 15 Cases of MI-Associated MC

Patient	Age (years)	Sex	CSHA-CFS score	GNRI	Type of MC	Year of occurrence	Time between symptom onset and diagnosis of MI (hours)	Day of MC detection (after symptom onset)	Manifestation(s) of the MC
1	85	Female	NA	89	VFWR	2013	2.9	Day 2	Sudden PEA
2	70	Male	NA	85	VFWR	2014	24.5	Day 2	Sudden PEA bradycardia during CT examination on admission
3	76	Male	NA	101	VFWR	2014	28.8	Day 2	Sudden PEA bradycardia
4	73	Male	2	97	VFWR	2014	25.3	Day 9	Sudden PEA bradycardia
5	61	Male	1	86	VSR	2015	179.1	Day 8	New pansystolic murmur
6	71	Male	NA	89	VSR	2016	56.2	Day 8	New pansystolic murmur
7	74	Male	NA	74	VFWR	2016	2.0	Day 1	Sudden PEA
8	82	Male	NA	NA	VFWR	2016	130.5	Day 6	OHCA with an initial non-shockable rhythm, and PEA bradycardia on admission ¹⁸⁾
9	80	Female	3	116	VFWR	2017	12.3	Day 17	Sudden PEA bradycardia
10	70	Male	1	94	VSR	2018	4.6	Day 1	New pansystolic murmur after PCI, and worsening HF despite intensive treatment
11	71	Male	2	89	VSR	2019	26.4	Day 2	New pansystolic murmur and worsening HF despite intensive treatment
12	76	Male	1	119	VFWR	2019	7.23	Day 1	Sudden BP drop without any change in HR or 12-lead ECG; after a few minutes, reduction in HR and asystole occurred during ad hoc PCI ³⁰⁾
13	93	Male	3	98	VSR	2019	12.2	Day 5	New pansystolic murmur and worsening HF despite intensive treatment ¹⁹⁾
14	89	Male	4	77	VSR	2019	75.3	Day 6	New pansystolic murmur ¹⁹⁾
15	71	Male	2	84	PMR	2020	2.7	Day 1	Rapidly progressive pulmonary edema and shock without systolic murmur during ad hoc PCI

BP indicates blood pressure; CABG, coronary artery bypass graft; CSHA-CFS, Canadian Study of Health and Aging Clinical Frailty Scale; CT, computed tomography; ECG, electrocardiogram; GNRI, Geriatric Nutritional Risk Index; HF, heart failure; HR, heart rate; MC, mechanical complication; MI, myocardial infarction; NA, not available; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; PEA, pulseless electrical activity; PMR, papillary muscle rupture; VFWR, ventricular free wall rupture; and VSR, ventricular septal rupture.

Table III. Further Clinical Details of the 15 Cases of MI-Associated MC

Patient	Culprit lesion	Rentrop grade	Ad hoc PCI	TIMI grade after PCI	MCS	Euro-SCORE II score	Japan SCORE (%)		CVS	Time between diagnosis of MC and admission to the operating room (hours)	30-day mortality
							30-day operative mortality (CABG only/valve) (%)	Plus major complications (CABG only/valve) (%)			
1	Diagonal branch	0	Yes	3	VA-ECMO	27.2	26.1/77.5	60.3/76.3	No [†]	-	Yes
2	LCX-OM	0	No	-	IABP, VA-ECMO	11.2	14.2/59.0	55.8/77.5	Yes	3.37	Yes
3	Proximal LAD	0	No	-	VA-ECMO	9.9	41.8/43.0	57.4/69.6	Yes	1.85	Yes
4	Proximal RCA	1	Yes	2	IABP	9.2	59.2/24.9	59.7/67.2	Yes	1.72	Yes
5	Middle LAD	0	No	-	IABP	9.5	24.0/24.1	49.9/62.5	Yes	6.85	No
6	Proximal LAD	1	No	-	IABP	10.0	29.3/17.0	51.1/56.2	Yes	26.00	No
7	Middle LAD	0	Yes	1	-	10.2	28.6/19.9	57.3/68.2	No [†]	-	Yes
8	LCX-OM	0	No	-	IABP, VA-ECMO	8.3	34.5/41.9	50.8/76.8	No [‡]	-	Yes
9	Proximal LAD	-*	Yes	3	IABP, VA-ECMO	14.7	57.5/54.8	80.5/82.8	No [†]	-	Yes
10	Middle RCA	1	Yes	1	IABP, VA-ECMO	11.8	25.9/34.7	47.6/73.7	Yes	13.75	Yes
11	Middle LAD	1	Yes	2	IABP	19.2	26.5/82.1	72.0/90.3	Yes	23.93	No
12	Proximal LCX	0	No	-	IABP, VA-ECMO	6.2	41.8/34.6	61.5/73.3	Yes	3.32	No
13	Distal LAD	0	Yes	2	IABP	23.4	56.2/23.9	58.5/61.6	No	-	Yes
14	Middle LAD	1	Yes	3	IABP	21.3	13.4/68.0	52.6/88.3	Yes	48.55	No
15	LCX-PL	0	Yes	1	IABP, VA-ECMO	9.3	37.7/26.8	48.3/71.8	Yes	6.23	No

*There was no collateral flow; before intervention, coronary blood flow was TIMI grade 3. [†]CVS not scheduled because sudden death resulted from abrupt VFWR. [‡]CVS not scheduled because of hypoxic encephalopathy complicating the cardiac arrest. BP indicates blood pressure; CABG, coronary artery bypass graft; CSHA-CFS, Canadian Study of Health and Aging Clinical Frailty Scale; CT, computed tomography; CVS, cardiovascular surgery; ECG, electrocardiogram; GNRI, Geriatric Nutritional Risk Index; HF, heart failure; HR, heart rate; IABP, intra-aortic balloon pump; LAD, left anterior descending coronary artery; LCX-OM, left circumflex artery-obtuse marginal branch; LCX-PL, left circumflex artery-posterolateral branch; MC, mechanical complication, MCS, mechanical circulatory support; MI, myocardial infarction; NA, not available; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; PEA, pulseless electrical activity; PMR, papillary muscle rupture; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction; VA-ECMO, veno-arterial extracorporeal membrane oxygenation, VFWR, ventricular free wall rupture; and VSR, ventricular septal rupture.

IV). Age, a relatively low GNRI, absence of hypertension and dyslipidemia, various vital signs, Killip class, a relatively low LVEF, relatively high lactate, relatively low hemoglobin, left main trunk as the culprit vessel, and PCI were all significantly associated with 30-day mortality (Table IV). Multivariate analysis showed MC to be independently associated with 30-day mortality (Model 1—OR: 11.75, 95% CI: 3.35-41.24; $P < 0.001$) (Model 2—OR: 19.01, 95% CI: 5.40-67.49; $P < 0.001$) (Model 3—OR: 36.59, 95% CI: 5.88-227.68; $P < 0.001$) (Model 4—OR: 13.02, 95% CI: 3.25-52.10; $P < 0.001$) (Model 5—OR: 13.26, 95% CI: 3.88-45.30; $P < 0.001$) (Table IV).

Characteristics of patients with an MC treated surgically and of patients with an MC treated conservatively: Ten patients who underwent surgery for MC represent 66.7% of the 15 patients with an MC. Four of these

10 patients had a VFWR, 5 had a VSR, and 1 had a PMR. Clinical characteristics, CAG findings, and therapeutic interventions have been determined for the patients whose MC was treated surgically and for those whose MC was treated conservatively, as shown in Table V. There was no significant between-group difference in sex, BMI, GNRI, coronary risk factors, vital signs, LVEF, laboratory test results, or CAG findings, but patients treated surgically were significantly younger than those treated conservatively (72.8 ± 7.1 years versus 82.8 ± 7.0 years, respectively; $P = 0.022$). They were also less frail than those treated conservatively (CSHA-CFS 1: 42.9% versus 0%, 2: 42.9% versus 0%, 3: 0% versus 100.0%, 4: 14.3% versus 0%; $P = 0.029$), and they had lower EuroSCORE II scores (11.8 ± 4.7 versus 16.8 ± 8.2 , respectively; $P = 0.154$). During the mean follow-up time of

Figure 1. Thirty-day mortality among patients with and without mechanical complications. Thirty-day mortality has been determined to be significantly higher among patients with mechanical complications than among those without (60.0% versus 6.3%, $P < 0.001$).

16.7 $\pm$ 12.3 days, 9 (60.0%) patients died (7/8 [87.5%] from VFWR, 2/6 [33.3%] from VSR and 0/1 [0.0%] from PMR), for an overall 30-day mortality rate of 60.0%. Thirty-day mortality in the surgical treatment group was 40% (4/10), but 100% (5/5) in the conservative therapy group ($P = 0.044$) (Figure 2).

Discussion

Incidence and characteristics of MCs in Japan versus MCs reported outside of Japan: The 1.70% incidence of MCs among our study patients (VFWR, 0.91%; VSR, 0.68%; PMR, 0.11%) was well in line with incidences (all $< 3\%$) reported data of other Japanese⁶⁾ and outside of Japan in this PCI era.¹⁻⁵⁾ Although previous studies showed increased age, female sex, a first transmural MI,²³⁾ and single-vessel disease²⁴⁾ to be more common among patients with an MC than among those without, the proportion of female patients did not differ between our study groups. Important to our study was the observation that frailty at the onset of an MC was not severe (CSHA-CFS score of < 6 for all patients), but nutritional status, per the GNRI, was not good. About half of the MCs (53.3%) occurred in association with an RMI, i.e., following an MI that was diagnosed between 24 hours and 30 days after symptom onset, which suggests that delayed treatment contributed to the occurrence of MC. Similarly, several studies conducted in either the pre-PCI era^{23,25)} or PCI era⁵⁾ have shown association between a prolonged interval between symptom onset and hospitalization and occurrence of an MC. Approximately half of our patients' MCs (53.3%) occurred within 48 hours after MI symptom onset, whereas 26.6% occurred after 1 week, and 1 occurred nearly 3 weeks after MI symptom onset. The reported median time between symptom onset and occurrence of an MC is 23.5 hours (interquartile range: 5.0-76.8 hours),²⁾ and there are reports of occurrence after 30 days.^{26,27)} We have also characterized the CAG findings in cases of MC. The culprit lesion was not only the main coronary artery but also a side branch, either the diagonal, obtuse marginal, or posterolateral branch, as reported previously in case 8.²⁸⁾ Patients who suffered an MC showed low TIMI flow both before and after PCI and poor collateral flow in the setting of transmural infarction, like patients outside of

Japan.^{2,5)}

Mortality in cases of an MC and its implications in terms of treatment: In our study, the prognosis of MC was determined to be poor, as indicated by the 30-day mortality rate of 60% (87.5% for VFWR, 33.3% for VSR, and 0% for PMR). One of our most important findings is that all 5 patients for whom the MC was treated conservatively did not survive, whereas 60.0% (6/10) of patients treated surgically did survive, even though their surgical risk was high and their nutritional status (per GNRI) was deemed not good. Surgical treatment might have been chosen for these patients because they were, in general, younger than those treated conservatively (72.8 versus 82.8 years of age, respectively), and they were less frail, but the clinical course in these individual cases should be carefully interpreted. For example, as we reported previously,²⁹⁾ cases 13 and 14 were both cases of VSR in very elderly patients, who both been considered to be at high surgical risk, but they were not particularly frail. For Patient 13, who, along with family members, was informed of the risks of surgery, conservative treatment was chosen, but the patient did not survive. For Patient 14, whose general condition was worse than that of Patient 13, surgery was performed, and his life was saved. Typically, MCs are treated surgically.³⁰⁻³³⁾ Moreover, there are reports that surgical intervention improves one's survival, but it is likely that the improved survival represents a selection bias, i.e., that surgery is chosen for healthier patients,²⁾ e.g., for patients who are hemodynamically stable. Many patients in whom an MC develops are at high surgical risk and are thus unavoidably assigned to conservative treatment. It has been reported that hypoalbuminemia, marked frailty (assessed by the CSHA-CFS), and a high BMI could be early prognostic factors in very elderly (≥ 85 years of age) patients admitted for MI, but that the prognosis is not generally poor for high-risk patients with a CSHA-CFS < 6 .¹¹⁾ Furthermore, for patients who suffer MI-associated PMR and are considered to be at high surgical risk, the initial high-risk phase is often overcome, and even though surgery for PMR is associated with notable operative mortality, mortality of these patients could be similar to that of patients without PMR.³⁴⁾ Surgical repair, regardless of the timing of the surgery, has been reported to be a significant factor associated with survival of patients with MI-associated VSR.⁸⁾ Our data, together with reported data, imply that because MCs are potentially fatal complications for which conservative treatment is ineffective, even in this PCI era, surgical intervention should be considered even if a patient's surgical risk is high, as long as the patient is not particularly frail.

Clues suggesting occurrence of an MC: Our study revealed that age, severity of heart failure, and time from symptom onset to MI recognition are associated with MCs, as has been noted in previous studies,^{2,3)} and that surgical treatment is significantly associated with reduced 30-day mortality. As per other studies, it was determined that early diagnosis of MI, correction of hemodynamics by means of mechanical circulatory support to decrease afterload, and subsequent surgical treatment are important for preventing MCs and improving the patients' prognosis.^{35,36)} Therefore, it is essential that physicians understand

Table IV. Results of Logistic Regression Analysis for 30-Day Mortality

	OR (95%CI)	P-value
Univariate models		
MC	19.53 (6.71–56.79)	< 0.001
Age (years)	1.04 (1.02–1.06)	0.001
Male sex	0.77 (0.43–1.37)	0.366
Body mass index (kg/m ²)	0.96 (0.89–1.03)	0.252
GNRI	0.94 (0.92–0.97)	< 0.001
Hypertension	0.41 (0.25–0.69)	0.001
Diabetes mellitus	0.85 (0.49–1.48)	0.556
Dyslipidemia	0.30 (0.17–0.55)	< 0.001
Systolic blood pressure (mmHg)	0.990 (0.985–0.995)	< 0.001
Heart rate (bpm)	1.02 (1.01–1.03)	0.001
Killip class	2.99 (2.41–3.71)	< 0.001
Left ventricular ejection fraction (%)	0.92 (0.90–0.94)	< 0.001
Lactate (mmol/L)	1.26 (1.15–1.38)	< 0.001
Hemoglobin (g/dL)	0.80 (0.72–0.89)	< 0.001
Left main trunk as the culprit vessel	7.22 (3.06–17.07)	< 0.001
Ad hoc PCI	0.23 (0.12–0.45)	< 0.001
Multivariate models		
Model 1		
MC	11.75 (3.35–41.24)	< 0.001
GNRI	0.965 (0.933–0.999)	0.043
Dyslipidemia	0.330 (0.129–0.844)	0.021
Model 2		
MC	19.01 (5.40–67.49)	< 0.001
Age (years)	1.06 (1.01–1.12)	0.032
Systolic blood pressure (mmHg)	0.990 (0.981–0.999)	0.036
Model 3		
MC	36.59 (5.88–227.68)	< 0.001
Killip class	1.67 (1.10–2.54)	0.016
Left ventricular ejection fraction (%)	0.92 (0.89–0.96)	< 0.001
Model 4		
MC	13.02 (3.25–52.10)	< 0.001
Lactate (mmol/L)	1.23 (1.11–1.37)	< 0.001
Model 5		
MC	13.26 (3.88–45.30)	< 0.001
GNRI	0.953 (0.922–0.984)	0.003
Left main trunk	7.20 (2.09–24.80)	0.002

Model 1: Adjusted for MC, age, male sex, GNRI, hypertension, and dyslipidemia. Model 2: Adjusted for MC, age, male sex, GNRI, systolic blood pressure, and heart rate. Model 3: Adjusted for MC, age, male sex, GNRI, Killip classification, and left ventricular ejection fraction. Model 4: Adjusted for MC, age, male sex, GNRI, hemoglobin, and lactate level. Model 5: Adjusted for MC, age, male sex, GNRI, left main trunk, and PCI. CI indicates confidence interval; GNRI, Geriatric Nutritional Risk Index; OR, odds ratio; MC, mechanical complication; and PCI, percutaneous coronary intervention.

the clinical manifestations of the various MI-associated MCs and to rapidly examine the patient echocardiographically to diagnose the MC. It is very important to immediately perform echocardiography when an MI-associated MC is suspected.

To aid clinicians, we provide the following list of clues to the occurrence of an MI-associated VFWR, VSR, or PMR. These are clues encountered not only by us, as described above, but also by other groups of investigators, as noted.

VFWR

- Sudden-onset hypotension with bradycardia, with or without premonitory symptoms^{37,38)}
- Occurrence of electromechanical dissociation during PCI³⁹⁾

VSR

- A new pansystolic murmur with a palpable thrill^{40,41)}

- Exacerbation of heart failure and prolongation of the shock state despite PCI and intensive care²⁹⁾

PMR

- Sudden-onset, progressive pulmonary edema and/or cardiogenic shock (with or without systolic murmur) that cannot be explained by the culprit lesion. Rather, the change is due to a rapid increase in left atrial pressure and a decrease in cardiac output resulting from mitral regurgitation. Absence of systolic murmur is due to concomitant left ventricular dysfunction leading to a decrease in regurgitation jet volume and rapid equalization of pressure in the ventricle and atria^{34,42)}

Study limitations: Our study data should be interpreted in light of our study limitations. It was carried out as a

Table V. Clinical Characteristics, CAG Findings, and Therapeutic Interventions of Patients with an MC Treated Surgically and Patients with an MC Treated Conservatively

	Surgical therapy (n = 10)	Conservative therapy (n = 5)	P-value
Age (years)	72.8 ± 7.1	82.8 ± 7.0	0.022
Male sex	10 (100.0%)	3 (60.0%)	0.095
Body mass index (kg/m ²)	22.4 ± 3.4	22.4 ± 7.0	0.817
GNRI	92.2 ± 11.7	94.5 ± 17.6	0.801
CSHA-CFS			0.029
1	3 (42.9%)	0	
2	3 (42.9%)	0	
3	0	2 (100.0%)	
4	1 (14.3%)	0	
Type of MC			
Ventricular free wall rupture	4 (40.0%)	4 (80.0%)	0.282
Ventricular septal rupture	5 (50.0%)	1 (20.0%)	0.580
Papillary muscle rupture	1 (10.0%)	0 (0.0%)	1.000
Coronary risk factors			
Hypertension	5 (50.0%)	2 (40.0%)	1.000
Diabetes mellitus	3 (30.0%)	1 (20.0%)	1.000
Dyslipidemia	3 (30.0%)	2 (40.0%)	1.000
Current smoking	7 (70.0%)	2 (50.0%)	0.580
Clinical presentation			
Systolic blood pressure (mmHg)	116.2 ± 34.1	141.3 ± 21.2	0.202
Diastolic blood pressure (mmHg)	68.0 ± 18.4	82.3 ± 5.9	0.162
Heart rate (bpm)	98.1 ± 21.7	85.4 ± 16.7	0.273
Body temperature (°C)	36.4 ± 0.8	36.5 ± 0.5	0.778
Respiratory rate (breaths/minute)	22.4 ± 7.3	21.2 ± 9.0	0.801
Left ventricular ejection fraction (%)	48.8 ± 14.3	59.0 ± 19.0	0.371
Results of initial laboratory tests			
Lactate (mmol/L)	5.6 ± 4.2	4.6 ± 3.6	0.670
Hemoglobin (g/dL)	12.9 ± 1.8	11.1 ± 1.5	0.063
Albumin (mg/dL)	3.2 ± 0.6	3.4 ± 0.5	0.542
Creatinine (mg/dL)	1.1 ± 0.3	1.0 ± 0.4	0.895
Occluded (culprit) vessel			
Left main trunk	0	0	–
Left anterior descending coronary artery	5 (50.0%)	4 (80.0%)	0.580
Left circumflex coronary artery	3 (30.0%)	1 (20.0%)	1.000
Right coronary artery	2 (20.0%)	0	0.524
Graft	0	0	–
Therapeutic intervention			
Ad hoc PCI	5 (50.0%)	4 (80.0%)	0.580
Cardiac surgery	10	0	–
CABG	3 (30.0%)	0	–
Endoventricular circular patch plasty	4 (40.0%)	0	–
VSD surgery	5 (50.0%)	0	–
MV surgery	1 (10.0%)	0	–
EuroSCORE II score (%)	11.8 ± 4.7	16.8 ± 8.2	0.154
Mechanical circulatory support			
IABP	9 (90.0%)	3 (60.0%)	0.242
VA-ECMO	5 (50.0%)	3 (60.0%)	1.000

Mean ± SD values or number (%) of patients are shown. *by Student's *t*-test, Mann-Whitney *U* test, chi-square test, or Fisher's exact test. CABG indicates coronary artery bypass graft; CAG, coronary angiography; CSHA-CFS, Canadian Study of Health and Aging Clinical Frailty Scale; GNRI, Geriatric Nutritional Risk Index; IABP, intra-aortic balloon pump; MI, myocardial infarction; MV surgery, mitral valve surgery; PCI, percutaneous coronary intervention; VA-ECMO, veno-arterial extracorporeal membrane oxygenation; and VSD surgery, ventricular septal defect surgery.

single-center retrospective observational study, and its sample size was rather small. In addition, the incidence of MC might have been underestimated as MCs could have occurred before admission or after discharge. VFWR can also result in sudden out-of-hospital death, which would

preclude identification of the underlying cause in the absence of autopsy.⁴³⁾ Further, the observation period was as short as 1 month; thus, long-term outcomes were not evaluated. Finally, because of the small number of MCs, we were unable to perform logistic analysis for predictors

Figure 2. Thirty-day mortality among patients with a mechanical complication (MC) treated surgically and those with MC treated conservatively. Thirty-day mortality is significantly lower for patients treated surgically than for those treated conservatively (40.0 versus 100.0%, $P = 0.044$).

of MI-associated MCs. We acknowledge that a long-term prospective study that compensates for these shortcomings is needed to support our findings.

Conclusion

In summary, our patients who suffered an MI-associated MC, in comparison to those who did not, were determined to be older, were of somewhat poor nutrition status, had suffered an RMI, were of poorer hemodynamic status, had lower TIMI flow grades, and were at higher surgical risk. None were particularly frail. Although MI-related MCs remain strongly associated with high mortality even in this PCI era, more than half of our MC patients survived after surgical treatment. These findings suggest that even if patients who suffer an MC are at high surgical risk, if they are not particularly frail, surgical intervention should be taken into consideration as a life-saving measure.

Acknowledgments

We thank Ms. Wendy Alexander-Adams for her help in reporting our findings in English.

Disclosure

Conflicts of interest: Yasuo Okumura has received research funding from Boston Scientific Japan.

IRB information: The study protocol was approved by the Ethics Committee of Nihon University Itabashi Hospital (RK-200714-10).

References

1. Moreno R, López-Sendón J, García E, *et al.* Primary angioplasty reduces the risk of left ventricular free wall rupture compared with thrombolysis in patients with acute myocardial infarction. *J Am Coll Cardiol* 2002; 39: 598-603.
2. French JK, Hellkamp AS, Armstrong PW, *et al.* Mechanical

complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI). *Am J Cardiol* 2010; 105: 59-63.

3. Qian G, Jin RJ, Fu ZH, *et al.* Development and validation of clinical risk score to predict the cardiac rupture in patients with STEMI. *Am J Emerg Med* 2017; 35: 589-93.
4. Figueras J, Alcalde O, Barrabés JA, *et al.* Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. *Circulation* 2008; 118: 2783-9.
5. Lanz J, Wyss D, Räber L, *et al.* Mechanical complications in patients with ST-segment elevation myocardial infarction: A single centre experience. *PLOS ONE* 2019; 14: e0209502.
6. Koeda Y, Itoh T, Ishikawa Y, *et al.* A multicenter study on the clinical characteristics and risk factors of in-hospital mortality in patients with mechanical complications following acute myocardial infarction. *Heart Vessels* 2020; 35: 1060-9.
7. Fujita T, Yamamoto H, Kobayashi J, *et al.* Mitral valve surgery for ischemic papillary muscle rupture: outcomes from the Japan cardiovascular surgery database. *Gen Thorac Cardiovasc Surg* 2020; 68: 1439-46.
8. Kageyama S, Nakanishi Y, Murata K, *et al.* Mortality and predictors of survival in patients with recent ventricular septal rupture. *Heart Vessels* 2020; 35: 1672-80.
9. Nozoe M, Sakamoto T, Taguchi E, Miyamoto S, Fukunaga T, Nakao K. Clinical manifestation of early phase left ventricular rupture complicating acute myocardial infarction in the primary PCI era. *J Cardiol* 2014; 63: 14-8.
10. Okino S, Nishiyama K, Ando K, Nobuyoshi M. Thrombolysis increases the risk of free wall rupture in patients with acute myocardial infarction undergoing percutaneous coronary intervention. *J Interv Cardiol* 2005; 18: 167-72.
11. Sujino Y, Tanno J, Nakano S, *et al.* Impact of hypoalbuminemia, frailty, and body mass index on early prognosis in older patients (≥ 85 years) with ST-elevation myocardial infarction. *J Cardiol* 2015; 66: 263-8.
12. Ekerstad N, Swahn E, Janzon M, *et al.* Frailty is independently associated with short-term outcomes for elderly patients with non-ST-segment elevation myocardial infarction. *Circulation* 2011; 124: 2397-404.
13. Chesebro JH, Knatterud G, Roberts R, *et al.* Thrombolysis in myocardial infarction (TIMI) Trial, Phase I: A comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. *Circulation* 1987; 76: 142-54.
14. Kimura K, Kimura T, Ishihara M, *et al.* JCS 2018 guideline on diagnosis and treatment of acute coronary syndrome. *Circ J* 2019; 83: 1085-196.
15. Puerto E, Viana-Tejedor A, Martínez-Sellés M, *et al.* Temporal trends in mechanical complications of acute myocardial infarction in the elderly. *J Am Coll Cardiol* 2018; 72: 959-66.
16. Arai R, Suzuki S, Semba H, *et al.* The predictive role of E/e' on ischemic stroke and atrial fibrillation in Japanese patients without atrial fibrillation. *J Cardiol* 2018; 72: 33-41.
17. Kunimura A, Ishii H, Uetani T, *et al.* Impact of Geriatric Nutritional Risk Index on cardiovascular outcomes in patients with stable coronary artery disease. *J Cardiol* 2017; 69: 383-8.
18. Cereda E, Pedrololi C. The geriatric nutritional risk index. *Curr Opin Clin Nutr Metab Care* 2009; 12: 1-7.
19. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. *J Am Coll Cardiol* 1985; 5: 587-92.
20. Nashef SA, Roques F, Sharples LD, *et al.* EuroSCORE II. *Eur J Cardiothorac Surg* 2012; 41: 734-44, discussion 744-5.
21. Motomura N, Miyata H, Tsukihara H, Takamoto S, Japan Cardiovascular Surgery Database Organization. Risk model of thoracic aortic surgery in 4707 cases from a nationwide single-race population through a web-based data entry system: the first report of 30-day and 30-day operative outcome risk models for

- thoracic aortic surgery. *Circulation* 2008; 118: S153-9.
22. Rockwood K, Song X, MacKnight C, *et al.* A global clinical measure of fitness and frailty in elderly people. *CMAJ* 2005; 173: 489-95.
23. Figueras J, Cortadellas J, Calvo F, Soler-Soler J. Relevance of delayed hospital admission on development of cardiac rupture during acute myocardial infarction: study in 225 patients with free wall, septal or papillary muscle rupture. *J Am Coll Cardiol* 1998; 32: 135-9.
24. Bajaj A, Sethi A, Rathor P, Suppogu N, Sethi A. Acute complications of myocardial infarction in the current era: diagnosis and management. *J Investig Med* 2015; 63: 844-55.
25. Honan MB, Harrell FE Jr, Reimer KA, *et al.* Cardiac rupture, mortality and the timing of thrombolytic therapy: a meta-analysis. *J Am Coll Cardiol* 1990; 16: 359-67.
26. Crenshaw BS, Granger CB, Birnbaum Y, *et al.* Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (global utilization of streptokinase and TPA for occluded coronary arteries) Trial Investigators. *Circulation* 2000; 101: 27-32.
27. Yip HK, Fang CY, Tsai KT, *et al.* The potential impact of primary percutaneous coronary intervention on ventricular septal rupture complicating acute myocardial infarction. *Chest* 2004; 125: 1622-8.
28. Asakura K, Homma T, Akutsu N, *et al.* Cardiac rupture due to side branch occlusion after stent implantation- the crime of jailed stent. *Circ J* 2020; 84: 295.
29. Arai R, Fukamachi D, Akutsu N, *et al.* Ventricular Septal Rupture after Recent myocardial infarction in the Very Elderly: clinical implication for surgery. *Int Heart J* 2020; 61: 831-7.
30. Massad MG, Geha AS. Surgical repair of mechanical complications of myocardial infarction. *World J Surg* 2004; 28: 847-56.
31. O'Gara PT, Kushner FG, Ascheim DD, *et al.* 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation* 2013; 127: e362-425.
32. Ibanez B, James S, Agewall S, *et al.* 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J* 2018; 39: 119-77.
33. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. *Ann Thorac Surg* 2012; 94: 436-43; discussion 443-4.
34. Russo A, Suri RM, Grigioni F, *et al.* Clinical outcome after surgical correction of mitral regurgitation in the reperfusion era: a papillary muscle rupture. *Circulation* 2008; 118: 1528-34.
35. Gueret P, Khalife K, Jobic Y, *et al.* Echocardiographic assessment of the incidence of mechanical complications during the early phase of myocardial infarction in the reperfusion era: a French multicentre prospective registry. *Arch Cardiovasc Dis* 2008; 101: 41-7.
36. Ternus BW, Mankad S, Edwards WD, Mankad R. Clinical presentation and echocardiographic diagnosis of postinfarction papillary muscle rupture: a review of 22 cases. *Echocardiography* 2017; 34: 973-7.
37. López-Sendón J, González A, López de Sá E, *et al.* Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. *J Am Coll Cardiol* 1992; 19: 1145-53.
38. Yang D, Xiang M, Liu X, He A. Left ventricular free-wall rupture in acute myocardial infarction: a blow-out type case. *High Blood Press Cardiovasc Prev* 2009; 16: 201-4.
39. Arai R, Fukamachi D, Akutsu N, Tanaka M, Okumura Y. Surviving case of a blowout-type left ventricular free wall rupture during percutaneous coronary intervention for a lateral acute myocardial infarction. *Int Heart J* 2020; 61: 606-10.
40. Hayashi T, Hirano Y, Takai H, *et al.* Usefulness of ST-segment elevation in the inferior leads in predicting ventricular septal rupture in patients with anterior wall acute myocardial infarction. *Am J Cardiol* 2005; 96: 1037-41.
41. Menon V, White H, LeJemtel T, Webb JG, Sleeper LA, Hochman JS. The clinical profile of patients with suspected cardiogenic shock due to predominant left ventricular failure: a report from the SHOCK Trial Registry. Should we emergently revascularize Occluded Coronaries in cardiogenic shock? *J Am Coll Cardiol* 2000; 36(Suppl A): 1071-6.
42. Bursi F, Enriquez-Sarano M, Nkomo VT, *et al.* Heart failure and death after myocardial infarction in the community: the emerging role of mitral regurgitation. *Circulation* 2005; 111: 295-301.
43. Batts KP, Ackermann DM, Edwards WD. Postinfarction rupture of the left ventricular free wall: clinicopathologic correlates in 100 consecutive autopsy cases. *Hum Pathol* 1990; 21: 530-5.