The Comparison of Clinical Outcomes Between Inferior ST-Elevation Myocardial Infarction with Right Ventricular Infarction Versus Without Right Ventricular Infarction

Masamitsu Noguchi,1 MD, Kenichi Sakakura,1 MD, Naoyuki Akashi,1 MD, Yusuke Adachi,1 MD, Yusuke Watanabe,1 MD, Yousuke Taniguchi,1 MD, Tatsuro Ibe,1 MD, Kei Yamamoto,1 MD, Hiroshi Wada,1 MD, Shin-ichi Momomura,1 MD and Hideo Fujita,1 MD

Summary

Right ventricular infarction (RVI) is a complication following inferior ST-elevation myocardial infarction (STEMI). The aim of the present study was to investigate the clinical outcomes of RVI in the contemporary primary percutaneous coronary intervention (PCI) era. The primary endpoint was in-hospital death, and the secondary endpoint was major adverse cardiac events (MACE), defined as the composite of cardiovascular death, re-hospitalization for heart failure, and non-fatal acute myocardial infarction (AMI). Event-free survival curves for MACE were constructed using the Kaplan-Meier method, and statistical differences between curves were assessed using the log-rank test. A total of 1354 patients with AMI were screened from January 2010 to December 2016. The final study population involved 315 patients with STEMI whose infarct related artery (IRA) was the right coronary artery (RCA). We categorized these 315 patients into the RVI group (n = 85) and the non-RVI group (n = 230). Median follow-up duration was 358 (IQR: 208-987) days. In-hospital deaths were more frequently observed in the RVI group (9.4%) than in the non-RVI group (3.0%) (P = 0.018). However, the incidence of MACE was not different between the groups (P = 0.537). In conclusion, in-hospital clinical outcomes were poorer in the RVI group than in the non-RVI group. However, mid-term MACE was not different between the two groups, suggesting the importance of aggressive acute treatment for STEMI patients with RVI.

Key words: Acute myocardial infarction, MACE, STEMI

Method

Study patients: We identified patients with AMI from hospital records of our medical center from January 2010 to December 2016. The diagnosis of AMI required the following criteria: symptoms consistent with AMI; elevated cardiac enzymes including troponin T, troponin I, and/or creatinine kinase (at least two-fold increase from the normal upper limit); and ST-segment elevation or depression in electrocardiograms compatible with AMI.10 RVI criteria were defined as ST-segment elevation in V4R (> 1 mm) or abnormal right ventricular wall motion in the echocardiogram, accompanying clinical symptoms such as hypotension.11 We included only inferior STEMI whose infarct related artery (IRA) was the right coronary artery (RCA). We categorized those study patients into the RVI group and the non-RVI group. The primary endpoint was in-hospital death, and the secondary endpoint was major adverse cardiac events (MACE), defined as the composite of cardiovascular death, re-hospitalization for heart failure, and non-fatal AMI. In-hospital and mid-term clinical events were acquired from our hospital records. Patients were followed up until meeting a study endpoint (cardiovascular death, re-hospitalization for heart failure, or non-fatal AMI) or until the study end-date (June 2017). This
study was approved by the institutional review board, and written informed consent was waived because of the retrospective study design.

Primary PCI was performed using standard techniques via the radial artery, femoral artery, or rarely brachial artery. First, we advanced a conventional guidewire across the lesion, and used a small (2.0-mm diameter) balloon or thrombus aspiration catheter. The choice of devices was at the discretion of the interventional cardiologist. Activated coagulation time was maintained for > 250 seconds during PCI.

**Definition:** Hypertension was defined as medical treatment for hypertension and/or a history of hypertension before admission. Dyslipidemia was defined as a total cholesterol level of ≥ 220 mg/dL or low-density lipoprotein cholesterol level of ≥ 140 mg/dL, or medical treatment for dyslipidemia or a history of dyslipidemia. Diabetes mellitus was defined as a hemoglobin A1c level of ≥ 6.5% (as NGSP value) or medical treatment for diabetes mellitus or a history of diabetes mellitus. We also calculated the estimated glomerular filtration rate (eGFR) from the serum creatinine level, age, weight, and gender using the following formula: eGFR = 194 × Cr−1.094 × age−0.287 (male), eGFR = 194 × Cr−1.094 × age−0.287 × 0.739 (female). Peripheral arterial disease (PAD) was defined as an ankle brachial index of < 0.9 or medical treatment for PAD. Chronic total occlusion (CTO) was defined as a 100% luminal narrowing in a non-IRA before PCI without antegrade flow and with antegrade or retrograde filling by collateral vessels. Shock was defined as systolic blood pressure < 90 mmHg or vasopressors required to maintain blood pressure or a history of attempted cardiopulmonary resuscitation.

**Statistical analysis:** Data are presented as the mean ± SD or percentage. Categorical variables are presented as numbers (percentage), and were compared using the Pearson chi-square test. The Kolmogorov-Smirnov test was performed to determine if the continuous variables were normally distributed. Normally distributed continuous variables were compared between the groups using the unpaired Student t test. Otherwise, continuous variables were compared using the Mann-Whitney U-test. Event-free survival curves for MACE were constructed with the Kaplan-Meier method, and statistical differences between curves were assessed using the log-rank test. A P value of < 0.05 was considered statistically significant. We analyzed all data using the SPSS ver. 25 for Windows (SPSS, Inc., Chicago, Illinois).

**Results**
A total of 1354 patients with AMI were screened from January 2010 to December 2016. We excluded patients with non-ST-elevation myocardial infarction (NSTEMI) (n = 428) and patients with STEMI whose IRA was not RCA (n = 611). The final study population comprised 315 patients with STEMI whose IRA was RCA. We categorized those patients into the RVI group (n = 85) and the non-RVI group (n = 230). The study flowchart is shown in Figure 1.

The comparison of patient characteristics between the RVI and non-RVI groups is shown in Table I. The systolic blood pressure on admission was significantly less in the RVI group (111 ± 29 mmHg) than in the non-RVI group (129 ± 31 mmHg) (P < 0.001). Shock status was more frequently observed in the RVI group (36.5%) than in the non-RVI group (18.3%) (P = 0.001). In laboratory data, aspartate transaminase, alanine transaminase, and serum total bilirubin were significantly greater in the RVI group than in the non-RVI group.

The comparison of lesion and procedural characteristics between the RVI and non-RVI groups is shown in Table II. The prevalence of bradycardia was significantly higher in the RVI group (35.3%) than in the non-RVI group (19.1%) (P = 0.003). The incidence of catecholamine use before CAG was significantly higher in the RVI group (34.1%) than in the non-RVI group (8.3%) (P < 0.001). The culprit lesion was more proximal in the RVI group than in the non-RVI group (P < 0.001). The initial TIMI was significantly less in the RVI group than in the non-RVI group (P = 0.016). Aspiration catheter was more frequently used in the RVI group (78.8%) than in the non-RVI group (58.3%) (P = 0.001).

The comparison of clinical outcomes between the RVI and non-RVI groups is shown in Table III. Peak creatinine phosphokinase (CK) and peak CK-muscle and brain (CK-MB) were significantly greater in the RVI group than in the non-RVI group (P < 0.001). In-hospital death and 30-day mortality were more frequently observed in the RVI group than in the non-RVI group. A total of 15 in-hospital deaths were observed, of which 9 (60%) were cardiac deaths. Figure 2 shows Kaplan-Meier curves for MACE between the groups. The median follow-up duration was 358 (IQR: 208–987) days. The incidence of MACE was not different between the groups (P = 0.537).

We also categorized the RVI group into the in-hospital death group (n = 8) and in-hospital survivor group (n = 77) to compare the clinical characteristics of RVI between the in-hospital death and in-hospital survivor groups. The comparison of patient characteristics of the RVI group between the in-hospital death and in-hospital survivor groups.
|                           | All (n = 315) | RVI group (n = 85) | non-RVI group (n = 230) | P value |
|---------------------------|--------------|-------------------|------------------------|---------|
| Age, years (n)            | 69 ± 12      | 71 ± 11           | 69 ± 13                | 0.125   |
| Female sex, n (%)         | 64 (20.3)    | 22 (25.9)         | 42 (18.3)              | 0.136   |
| Body mass index, kg/m² (n)| 24.2 ± 5.0   | 25.2 ± 7.6        | 23.9 ± 3.6             | 0.15    |
| Hypertension, n (%)       | 216 (68.6)   | 62 (72.9)         | 154 (67.0)             | 0.31    |
| Dyslipidemia, n (%)       | 188 (59.7)   | 47 (55.3)         | 141 (61.3)             | 0.334   |
| Diabetes mellitus, n (%)  | 113 (35.9)   | 34 (40.0)         | 79 (34.3)              | 0.553   |
| Current smoker, n (%)     | 111 (35.4)   | 30 (35.7)         | 81 (35.2)              | 0.935   |
| Peripheral arterial disease, n (%) | 3 (1.0) | 1 (1.2) | 2 (0.9) | 0.803 |
| Previous myocardial infarction, n (%) | 30 (9.6) | 2 (2.4) | 28 (12.2) | 0.008 |
| Previous PCI, n (%)       | 49 (15.6)    | 9 (10.6)          | 40 (17.4)              | 0.139   |
| Previous CABG, n (%)      | 2 (0.6)      | 1 (1.2)           | 1 (0.4)                | 0.462   |
| Previous atrial fibrillation, n (%) | 10 (3.2) | 4 (4.8) | 6 (2.6) | 0.336 |
| Previous stroke, n (%)    | 28 (8.9)     | 11 (12.9)         | 17 (7.4)               | 0.124   |
| Killip classification     |              |                   |                       |         |
| 1 or 2, n (%)             | 274 (87.0)   | 68 (80.0)         | 206 (89.6)             |         |
| 3 or 4, n (%)             | 41 (13.0)    | 17 (20.0)         | 24 (10.4)              |         |
| Systolic blood pressure on admission, mmHg (n) | 124 ± 31 | 111 ± 29 | 129 ± 31 (n = 227) | < 0.001 |
| Diastolic blood pressure on admission, mmHg (n) | 71 ± 18 | 66 ± 17 | 74 ± 18 (n = 226) | 0.001 |
| Hemodialysis, n (%)       | 15 (4.8)     | 2 (2.4)           | 13 (5.7)               | 0.222   |
| Left ventricular ejection fraction on admission, (n) | 48 ± 8 | 48 ± 7 | 48 ± 8 (n = 80) | 0.728 |
| Left ventricular ejection fraction before discharge, (n) | 57 ± 11 | 56 ± 11 | 58 ± 11 (n = 194) | 0.312 |
| Shock status, n (%)       | 73 (23.2)    | 31 (36.5)         | 42 (18.3)              | 0.001   |
| Cardio-pulmonary arrest, n (%) | 11 (3.5) | 2 (2.4) | 9 (3.9) | 0.503 |
| Ventricular tachycardia/ventricular fibrillation, n (%) | 16 (5.1) | 5 (5.9) | 11 (4.8) | 0.693 |
| Bradycardia (HR < 50 bpm or temporary pacing before CAG), n (%) | 74 (23.5) | 30 (35.3) | 44 (19.1) | 0.003 |
| ≥24 h from onset to hospital arrival, n (%) | 59 (19.2) | 18 (21.4) | 41 (18.3) | 0.535 |
| Intubation at admission, n (%) | 13 (4.1) | 5 (5.9) | 8 (3.5) | 0.341 |
| Catecholamine use before CAG, n (%) | 48 (15.2) | 29 (34.1) | 19 (8.3) | < 0.001 |
| Laboratory Data           |              |                   |                       |         |
| Aspartate transaminase, U/L (n) | 83 ± 216 | 141 ± 389 | 62 ± 80 (n = 228) | < 0.001 |
| Alanine transaminase, U/L (n) | 42 ± 93 | 66 ± 169 | 33 ± 32 (n = 228) | < 0.001 |
| Serum total bilirubin, mg/dL (n) | 0.6 ± 0.4 | 0.7 ± 0.4 | 0.6 ± 0.3 (n = 224) | 0.004 |
| Serum creatinine, mg/dL (n) | 1.7 ± 4.8 | 1.5 ± 2.1 | 1.7 ± 5.5 | 0.009 |
| eGFR (mL/min/1.73 m²)      | 64 ± 28      | 58 ± 26           | 66 ± 28                | 0.011   |
| Serum albumin, mg/dL (n)   | 3.9 ± 0.5    | 3.9 ± 0.5         | 3.9 ± 0.5              | 0.814   |
| Hemoglobin, g/dL (n)       | 13.7 ± 8.7   | 13.1 ± 2.0        | 13.9 ± 10.1            | 0.379   |
| C-reactive protein, mg/dL (n) | 1.6 ± 3.8 | 2.0 ± 3.7 | 1.5 ± 3.8 (n = 223) | 0.382 |
| Total cholesterol, mg/dL (n) | 191 ± 45 | 189 ± 53 | 191 ± 42 (n = 200) | 0.419 |
| LDL cholesterol, mg/dL (n) | 108 ± 40 | 104 ± 45 | 109 ± 38 (n = 221) | 0.11 |
| HDL cholesterol, mg/dL (n) | 43 ± 12 | 42 ± 11 | 43 ± 12 (n = 218) | 0.794 |
| Triglyceride, mg/dL (n)    | 134 ± 105    | 142 ± 138         | 130 ± 90               | 0.938   |
| HbA1c (NGSP), % (n)        | 6.4 ± 1.3    | 6.2 ± 1.0         | 6.4 ± 1.4              | 0.748   |
| BNP, (n)                   | 242 ± 489    | 248 ± 362         | 240 ± 530              | 0.461   |
| Medications at admission   |              |                   |                       |         |
| ACE-I and/or ARB, n (%)    | 113 (37.0)   | 33 (41.8)         | 80 (36.5)              | 0.41    |
| Calcium channel blocker, n (%) | 102 (34.2) | 29 (36.7) | 73 (33.3) | 0.588 |
| β-Blocker, n (%)           | 36 (12.1)    | 6 (7.6)           | 30 (13.7)              | 0.154   |
| α-Blocker, n (%)           | 2 (0.7)      | 1 (1.3)           | 1 (0.5)                | 0.45    |
| Diuretic, n (%)            | 24 (8.1)     | 10 (10.9)         | 16 (7.3)               | 0.43    |
| Oral antidiabetic agents, n (%) | 53 (17.4) | 17 (21.0) | 36 (16.1) | 0.317 |
| Insulin, n (%)             | 12 (3.9)     | 2 (2.4)           | 10 (4.5)               | 0.419   |
| Statin, n (%)              | 80 (26.2)    | 19 (23.2)         | 61 (27.4)              | 0.461   |
| Aspirin, n (%)             | 56 (18.4)    | 9 (11.1)          | 47 (21.0)              | 0.049   |
| Clopidogrel, n (%)         | 17 (5.6)     | 4 (4.9)           | 13 (5.8)               | 0.771   |
| Ticlopidine, n (%)         | 3 (1.0)      | 1 (1.2)           | 2 (0.9)                | 0.789   |
| Prasugrel, n (%)           | 2 (0.6)      | 0 (0.8)           | 2 (0.9)                | 0.387   |

RVI indicates right ventricular infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting; HR, heart rate; CAG, coronary angiography; eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein; HDL, high-density lipoprotein; HbA1c, hemoglobin A1c; NGSP, National Glycohemoglobin Standardization Program; BNP, brain natriuretic peptide; ACE-I, angiotensin converting enzyme-inhibitor; and ARB, angiotensin receptor blockers.
The present study included 315 consecutive patients STEMI whose IRA was RCA, and compared clinical characteristics and outcomes between the RVI group (n = 85) and the non-RVI group (n = 230). The incidence of hypotension, shock status, or need for catecholamine at admission was more frequently observed in the RVI group than in the non-RVI group. The culprit lesion was more proximal in the RVI group than in the non-RVI group. Primary PCI was performed in most cases, and thrombus aspiration was more frequently performed in the RVI group as compared to the non-RVI group. In-hospital clinical outcomes were poorer in the RVI group than in the non-RVI group, especially in-hospital deaths were more frequent in the RVI group. However, the incidence of mid-term MACE was not different between the two groups. Our results may suggest the importance of acute
management for STEMI patients with RVI during the index hospitalization including primary PCI, because midterm clinical outcomes for STEMI patients with RVI may not be worse as compared to STEMI patients without RVI.

Clinical characteristics of RVI such as hypotension, shock, or bradycardia in our study were similar to those in previous studies. Although RVI sometimes occurs following STEMI whose culprit is the distal RCA, the incidence of RVI in our study was more frequent in the proximal RCA than in the mid or distal RCA, which was consistent with the previous studies. In interventional procedure, thrombus aspiration was more frequently performed in the RVI group than in the non-RVI group. Indeed, thrombus aspiration was performed in approximately 80% of RVI. Although the ACC/AHA/SCAI guideline downgraded thrombus aspiration from class IIa to IIb (selective and bailout aspiration thrombectomy) or III (routine aspiration thrombectomy), most of our study patients received primary PCI before the issue of that guideline. Higher incidence of thrombus aspiration group may suggest the presence of visible large thrombus in the RVI group.

In-hospital outcomes were worse in the RVI group.

Table III. The Comparison of Clinical Outcomes Between the RVI and Non-RVI Groups

|                          | All (n = 315) | RVI group (n = 85) | non-RVI group (n = 230) | P value |
|--------------------------|--------------|-------------------|-------------------------|---------|
| Peak CK, IU/L            | 1983 ± 1815  | 2701 ± 1913       | 1718 ± 1707             | <0.001  |
| Peak CK-MB, IU/L         | 187 ± 243    | 274 ± 396         | 154 ± 140               | <0.001  |
| Duration of CCU stay, days (median) | 4 (3-5) | 4 (3-6) | 3 (3-5) | <0.001 |
| Duration of hospitalization, days (median) | 11 (8-14) | 12 (IQR: 10-16) | 10 (8-13) | 0.003 |
| In-hospital death, n (%) | 15 (4.8)     | 8 (9.4)           | 7 (3.0)                 | 0.018   |
| 30-days mortality, n (%) | 15 (5.2) (n = 291) | 8 (9.8) (n = 82) | 7 (3.4) (n = 209) | 0.026 |
| Cardiovascular death, n (%) | 13 (4.1) | 6 (7.1) | 7 (3.0) | 0.112 |
| Re-hospitalization due to heart failure, n (%) | 10 (3.2) | 4 (4.7) | 6 (2.6) | 0.346 |
| Non-fatal AMI, n (%)     | 9 (2.9)      | 1 (1.2)           | 8 (3.5)                 | 0.276   |

RVI indicates right ventricular infarction; CK, creatinine phosphokinase; CK-MB, creatinine phosphokinase, muscle and brain; CCU, cardiac care unit; IQR, interquartile range; and AMI, acute myocardial infarction.

Figure 2. Kaplan-Meier curves for MACE stratified between the RVI and non-RVI groups. MACE indicates major adverse cardiovascular events; and RVI, right ventricular infarction.
than in the non-RVI group. Especially, in-hospital deaths were more frequently observed in the RVI group than in the non-RVI group, which is consistent with previous studies in 1990s\textsuperscript{2} or 2000s.\textsuperscript{3} A contemporary strategy toward STEMI may not be sufficient to reduce the mortality of STEMI with RVI, because some RVIs complicated with shock were refractory to any aggressive treatment.\textsuperscript{22) Moreover, longer duration of CCU stay and hospital stay
In the RVI group would reflect that RVI would require more medical resources than non-RVI. On the contrary, mid-term clinical outcomes were not different between the RVI group and the non-RVI group. The risk of MACE was attenuated in the RVI group after the patient discharged from the index admission, which was consistent to early studies.23) Although RV injury may be irreversible for more than 12 months, RV function such as RV ejection fraction is considered to recover in late phase.17) Moreover, the number of diseased vessels, left main involvement, and the presence of CTO in non-IRA arteries, which would affect the incidence of long-term MACE, were not different between the groups. Therefore, the hemodynamic effect caused by RVI may be minimal in late phase, which can be a reason for the attenuation of the MACE risk after the patients were discharged in the RVI group.

Clinical implications of the present study should be noted. Because in-hospital outcomes were poorer, but mid-term outcomes were not worse in inferior STEMI with RVI, our goal for inferior STEMI with RVI may be the successful discharge from the index hospitalization. Aggressive acute treatment including primary PCI, fluid resuscitation, temporary pac- ing, catecholamine support, and mechanical support if needed should be considered for inferior STEMI with RVI,24) because the hemodynamic effect by RVI would be much less in the late phase than in the acute phase.17) Furthermore, impaired renal function may be associated with in-hospital death in patients with RVI. Special attention...
should be reserved for patients with RVI and impaired renal function. Successful primary PCI alone would not be sufficient for the improvement of in-hospital outcomes even in contemporary primary PCI era. If a patient with RVI is successfully discharged from the index hospitalization, we may not need to pay a special attention to RVI, which would be a different approach in anterior myocardial infarction with LV dysfunction.

**Study limitations:** First, as the present study was a single-center retrospective study, there may be a patient selection bias. Second, we could not perform a statistical power analysis, which may have resulted in a possibility of beta error in the comparisons between the RVI and non-RVI groups. Third, although we compared the clinical characteristics of RVI between the in-hospital death and in-hospital survivor groups, the number of in-hospital death group was significantly small, which requires careful interpretation. Fourth, there were several missing values in the left ventricular ejection fraction before discharge and on admission, which are important prognostic markers. Finally, the present study was observational in nature, and our findings need to be confirmed prospectively in a well-organized trial.

**Conclusions**

In-hospital clinical outcomes were poorer in the RVI group than in the non-RVI group, especially in-hospital deaths were more frequent in the RVI group. However, the incidence of mid-term MACE was not different between the two groups, suggesting the importance of aggressive acute treatment for STEMI patients with RVI.

**Acknowledgments**

The authors acknowledge all staff in the catheter laboratory in Saitama Medical Center, Jichi Medical University, for their technical support in this study.

**Disclosure**

**Conflicts of interest:** Dr. Sakakura has received speaking honoraria from Abbott Vascular, Boston Scientific, Medtronic Cardiovascular, Terumo, OrbisNeich, Japan Lifeline, and NIPRO. He has served as a proctor for Rotablator for Boston Scientific and has served as a consultant for Abbott Vascular and Boston Scientific. Prof. Fujita served as a consultant for Mehergen Group Holdings, Inc.

**References**

1. Obradovic S, Dzudovic B, Djuric I, Jovic Z, Djenic N. Women have right ventricular infarction more frequently than men. Acta Cardiol 2015; 70: 343-9.
2. Zehender M, Kasper W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med 1993; 328: 981-8.
3. Gumina RJ, Wright RS, Kopecky SL, et al. Strong predictive value of TIMI risk score analysis for in-hospital and long-term survival of patients with right ventricular infarction. Eur Heart J 2002; 23: 1678-83.
4. Mehta SR, Eikelboom JW, Natarajan MK, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. J Am Coll Cardiol 2001; 37: 37-43.
5. Ozaki Y, Katagiri Y, O numa Y, et al. CVIT expert consensus document on primary percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) in 2018. Cardiovasc Interv Ther 2018; 33: 178-203.
6. Watanabe Y, Sakakura K, Taniguchi Y, et al. Determinants of in-hospital death in acute myocardial infarction with triple vessel disease. Int Heart J 2016; 57: 697-704.
7. Yamamoto K, Sakakura K, Akashi N, et al. Clinical outcomes after acute myocardial infarction according to a novel stratification system linked to a rehabilitation program. J Cardiol 2018; 72: 227-33.
8. Ito M, Wada H, Sakakura K, et al. Clinical characteristics and mid-term outcomes of non-elderly obese patients with acute decompensated heart failure in Japan. Int Heart J 2018; 59: 766-71.
9. Matsuo S, Imai E, Horio M, et al. Revised equations for estimated GFR from serum creatinine in Japan. Am J Kidney Dis 2009; 53: 982-92.
10. Chaudru S, de Mullenheim PY, Le Faucheur A, Kaladjii A, Jaqunindl V, Mahé G. Training to perform ankle-brachial index: systematic review and perspectives to improve teaching and learning. Eur J Vasc Endovasc Surg 2016; 51: 240-7.
11. Mizuguchi Y, Takahashi A, Hashimoto S, et al. Impact of the presence of chronic total occlusion in a non-infarct-related coronary artery in acute myocardial infarction patients. Int Heart J 2015; 56: 592-6.
12. Ryu KS, Park HW, Park SH, et al. Comparison of clinical outcomes between culprit vessel only and multivessel percutaneous coronary intervention for ST-segment elevation myocardial infarction patients with multivessel coronary diseases. J Geriatr Cardiol 2015; 12: 208-17.
13. Hoebers LP, Vis MM, Claessen BE, et al. The impact of multivessel disease with and without a co-existing chronic total occlusion on short- and long-term mortality in ST-elevation myocardial infarction patients with and without cardiogenic shock. Eur J Heart Fail 2013; 15: 425-32.
14. Yamamoto S, Sakakura K, Taniguchi Y, et al. Safety of reversing anticoagulation by protamine following elective transfermoral percutaneous coronary intervention in the drug-eluting stent era. Int Heart J 2018; 59: 482-8.
15. Chockalingam A, Gnanavelu G, Subramaniam T, Dorairajan S, Chockalingam V. Right ventricular myocardial infarction: presentation and acute outcomes. Angiology 2005; 56: 371-6.
16. Shiraki H, Yokozuka H, Negishi K, et al. Acute impact of right ventricular infarction on early hemodynamic course after inferior myocardial infarction. Circ J 2010; 74: 148-55.
17. Kumar A, Abdel-Aty H, Kriedemann I, Schröder R. Effects of thrombolytic therapy in acute inferior myocardial infarction with or without right ventricular involvement. J Am Coll Cardiol 2005; 46: 1969-76.
18. Andersen HR, Falk E, Nielsen D. Right ventricular infarction: frequency, size and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. J Am Coll Cardiol 1987; 10: 1223-32.
19. Goldstein JA, Barzilai B, Rosamond TL, Eisenberg PR, Jaffe AS. Determinants of hemodynamic compromise with severe right ventricular infarction. Circulation 1990; 82: 359-68.
20. Zeyer M, Neuhaus KL, Wegscheider K, Tzempelik N, Holmskov P, Schröder R. Effects of thrombolytic therapy in acute inferior myocardial infarction with or without right ventricular involvement. HFT-4 Trial Group. Hirudin for Improvement of Thrombolysis. J Am Coll Cardiol 1998; 32: 876-81.
21. Levine GN, Bates ER, Blankenship JC, et al. 2015 ACC/AHA/SCAI focused update on primary percutaneous coronary intervention for patients with ST-elevation myocardial infarction: an update of the 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention and the 2013 ACCF/AHA guideline for
for the management of ST-elevation myocardial infarction. J Am Coll Cardiol 2016; 67: 1235-50.
22. Taniguchi Y, Sakakura K, Adachi Y, et al. In-hospital outcomes of acute myocardial infarction with cardiogenic shock caused by right coronary artery occlusion vs. left coronary artery occlusion. Cardiovasc Interv Ther 2018; 33: 338-44.
23. Gumina RJ, Murphy JG, Rihal CS, Lennon RJ, Wright RS. Long-term survival after right ventricular infarction. Am J Cardiol 2006; 98: 1571-3.
24. Inohara T, Kohsaka S, Fukuda K, Menon V. The challenges in the management of right ventricular infarction. Eur Heart J Acute Cardiovasc Care 2013; 2: 226-34.