**Abstract.** Cardiogenic shock is the most severe form of acute heart failure. The aim of the current study was to investigate correlations between diagnostic parameters and the estimated ejection fraction in patients with cardiogenic shock. A total of 2,445 patients with acute myocardial infarction were subjected to coronary angiograms and standard 2D transthoracic echocardiography. Information for culprit vessel(s) and mitral regurgitation were collected. The Spearman's correlation test was used to assess the correlation between diagnostic parameters and estimated ejection fractions at a 95% confidence level. The angiographically-derived numbers of culprit vessels had a significant correlation with mitral regurgitation (r=0.907; P=0.034). The echocardiographically-derived mitral regurgitation was significantly correlated with the numbers of culprit vessels (r=0.896; P=0.04). Positive correlation was established between angiographically- and echocardiographically-measured left ventricular ejection fraction (r=0.356; P=0.045). The numbers of culprit vessels (P=0.058) and mitral regurgitation (r=0.99; P=0.001) were similar for angiography and echocardiography. Echocardiography- and angiography-derived results were correlated with the estimated ejection fractions of patients with cardiogenic shock. However, there is a substantial difference in the procedures of the two operational techniques.

**Introduction**

Cardiogenic shock is the most severe form of acute heart disease (1). Optimal revascularization strategies for patients with cardiogenic shock are complex (2) as the majority of patients with cardiogenic shock have multi-vessel disease (3). Echocardiographic and angiographic assessments aid the diagnosis and guide revascularization strategies for patients with cardiogenic shock associated with myocardial infarction. For such patients, coronary angiography or echocardiography are used as diagnostic techniques (4). The severity of mitral annular calcification (5), aortic valve sclerosis (6), the ejection fraction of the left ventricle (7) evaluated by echocardiography (4), and the ejection fraction of the left ventricle and severity of disease evaluated by angiography are predictors of survival (4). To the best of our knowledge, no previously published studies have compared transthoracic echocardiography and coronary angiography assessments in cardiogenic shock-associated myocardial infarction in a large population.

The primary objective of the current prospective cohort study was to assess correlations between the diagnostic parameters (derived by coronary angiography and acute transthoracic echocardiography) and the estimated ejection fraction in patients with cardiogenic shock. The secondary objective of the study was to provide further insight into the pathogenesis of acute myocardial infarction in the management of cardiogenic shock in a Chinese setting.

**Materials and methods**

**Patient selection.** A total of 3,908 patients aged ≥18 years with acute myocardial infarction were admitted to the Department of Cardiology at PLA General Hospital, Chongqing Traditional Chinese Medicine Hospital (Chongqing, China) and the Hospital of Beijing the Hui People (Beijing, China) between February 2014 and July 2018 due to medical emergency (i.e. requiring immediate medical attention) were subjected to a 12-lead electrocardiogram (ECG) diagnosis (8) prior to revascularization. The male to female ratio was 2.5:1 and mean age...
of patients was 64.55±10.45 years (age range, 55-82 years). Patients with acute myocardial infarction (diagnosed as a new left bundle branch block, new Q waves and ST elevations on the ECG) who exhibited signs of end-organ hypoperfusion (not due to sustained ventricular arrhythmia and/or bradycardia), including chest pain, back pain, dyspnea, warm and/or flushed skin, raised respiratory rate, confusion and hypotension (including signs of dizziness, lightheadedness, blurred vision, nausea, fainting, syncope, fatigue, and/or lack of concentration) were included in the current study. Patients with a history of revascularization, systemic illness (for example, anoxic brain damage may adversely affect the outcome of coronary bypass surgery), heparin allergy, anaphylaxis, sepsis, hypovolemia and/or those receiving the revascularization treatment were excluded from the study. Additionally, patients who had not provided consent or were not willing to undergo diagnosis were excluded.

Coronary angiography. Coronary angiograms were digitally recorded by seven cardiologists with three years' experience prior to revascularization, and coronary parameters were analyzed per the Thrombolysis In Myocardial Infarction Criteria (3). Based on the ejection of contrast media into the left atrium, mitral regurgitation was graded as follows: i) 0, absent (no ejection of contrast media); ii) 1, mild (contrast refuxes into the left atrium but cleared on each beat); iii) 2, moderate (left atrial contrast density gradually increased but did not equal left ventricle density); iv) 3, severe (the density of contrast in the atrium and ventricle equalized after several beats); and v) 4, extreme (the left atrium had become as dense as the left ventricle on the first beat and contrast was seen refluxing into the pulmonary veins) (4).

Image analysis for angiography. The type of lesion was graded per the American Heart Association/American College of Cardiology guidelines (9) as follows: i) A, <10 mm in size, concentric, readily accessible, discrete, <45° smooth contours, absent or little calcification, no involvement of side branches, not completely occluded, not ostial and no thrombus; ii) B1, one characteristic from the following: 10-20 mm in size, eccentric, proximal segment had moderate tortuosity, irregular contour, thrombus present, moderate or heavy calcification, total occlusion was <3 months old and an ostial/bifurcation lesion which required two guide wires; iii) B2, ≥2 of the characteristics described for the B1 type; and iv) C, ≥20 mm, diffuse, proximal segment had excessive tortuosity, total occlusion was >3 months old, side branches were involved, friable lesions and a degenerated vein graft (10). The culprit vessel collateral score was graded per the Rentrop classification (11) as follows: i) 0, collateral flow was absent; ii) 1, collateral flow was present in secondary branches only; iii) 2, collateral flow was present in the major vessels but had not reached the culprit lesion; and iv) 3, collateral flow reached the infarct-associated artery and the culprit lesion. The culprit lesion thrombus score was graded as follows: i) 0, no thrombus; ii) 1, possible thrombus; iii) 2, the dimension of the thrombus was less than one-half of the diameter of the culprit vessel diameter; iv) 3, the dimension of the thrombus was greater than one-half but less than two-thirds of the culprit vessel diameter; v) 4, the dimension of the thrombus was greater than two-thirds of the culprit vessel diameter but did not result in total culprit vessel occlusion; and vi) 5, total culprit vessel occlusion (12).

Transthoracic echocardiography. All patients were subjected to standard 2D transthoracic echocardiography using a Sonimage HS1 ultrasound system (Konica Minolta Medical & Graphic Imaging Europe B.V.) and S4-2 phased transducers performed by cardiac ultra-sonographers (with 3 years' experience) prior to revascularization. Images were blindly analyzed by cardiothoracic surgeons (with 3 years' experience) at the Chongqing Traditional Chinese Medicine Hospital (Chongqing, China) (5).

Image analysis for echocardiography. Mitral regurgitation was calculated according to the following formula for the regurgitant fraction of the mitral valve: Regurgitant fraction (%)=(mitral valve regurgitant volume)/(trans-mitral volume) x100. Grading was performed as follows: i) 0, absent (regurgitant fraction, 0); ii) 1, mild (regurgitant fraction, <30%); iii) 2, moderate (regurgitant fraction, 30-39%); iv) 3, severe (regurgitant fraction, 40-49%); and v) 4, extreme (regurgitant fraction, ≥50%) (4). The relative wall thickness and shortening fraction were measured from the short-axis view using 3-MHz transducers. The four-chambered view was taken for the mitral flow velocity and was calculated according to the following equation: Mitral flow velocity=(peak early diastolic flow velocity)/(peak atrial flow velocity) (13). The left ventricular diastolic dysfunction was graded as follows: i) 1, isolated early relaxation abnormality (initial stage); ii) II, increased filling pressure in the atrium (moderate condition); and iii) III, restrictive filling of the heart (severe condition) (14).

Statistical analysis. Categorical data are presented as frequency (percentage) whilst continuous data are presented as mean ± standard deviation. InStat (version 3.1; GraphPad Software, Inc.) was used to analyze the data. Categorical data were analyzed by the χ² test and continuous data were analyzed by the Wilcoxon test (15). The Spearman's correlation test was used to analyze correlations between the diagnostic parameters (echocardiographically- and angiographically-measured data) and estimated ejection fractions. A Spearman's correlation coefficient (r) of 0.123-0.994 was considered significant (4). P<0.05 was considered to indicate a statistically significant difference.

Results

Patient demographic characteristics and medical history. Among the enrolled patients, 515 had undergone previous revascularization, 14 had systemic illness, 212 had septic shock, 512 had hypovolemic shock, 27 had been admitted to hospital due to a side effect (shock) of the medication they were administered, 145 had a normal ECG and 35 were not willing to undergo further diagnostic testing following admission to the emergency department or the cardiology department at the Chongqing Traditional Chinese Medicine Hospital, (Chongqing, China), the Fourth Medical Center of PLA General Hospital (Beijing, China) and the Hospital of Beijing The Hui People (Beijing, China) where three patients were afflicted with shock due to anaphylaxis. Therefore, they were excluded from the analysis. A total of 2,445 patients with
cardiogenic shock were included in the current prospective cohort study, where 165 patients died in the emergency department and during follow-up period (main cause of death was cardiogenic shock). A schematic representation of the study design is presented in Fig. 1.

Among the enrolled patients, 115 had end-organ hypoperfusion, 629 had a new left bundle branch block on the ECG, 715 patients had new Q waves on the ECG, 986 patients had ST elevations on the ECG and 101 patients had a history of coronary artery bypass graft. All the patients received oral treatment (including β-blockers, angiotensin converting enzyme inhibitors, angiotensin receptor blocker, calcium channel blocker, and statins) for cardiogenic shock. The other demographic characteristics of the enrolled patients are presented in Table I.

Coronary angiographic evaluation. Coronary angiography-derived mitral regurgitation grade had a significant correlation with the number of culprit vessels ($r=0.907$; $P=0.034$). The coronary angiography-derived number of culprit vessels had no significant correlation with the location of the culprit vessels ($r=-0.21$; $P=0.735$). The coronary angiography-derived culprit lesion type had no significant correlation with the culprit vessel collateral score ($r=-0.054$; $P=0.947$) and culprit lesion thrombus score ($r=-0.406$; $P=0.594$). The other coronary angiography assessments of the enrolled patients are presented in Table II.

Transthoracic echocardiographic evaluation. The echocardiographically-derived mitral regurgitation had a significant strong correlation with the number of culprit vessel ($r=0.896$; $P=0.04$). The echocardiographically-derived number of culprit vessels had no significant correlation with the degree of left ventricular diastolic dysfunction ($r=-0.693$; $P=0.513$). The other echocardiographic assessments of the enrolled patients are presented in Table III.

Correlations between imaging modalities for diagnostic parameters. The left ventricular ejection fractions derived by coronary angiography ($31.12\pm12.45\%$; 28% as the lowest
and 42% as the highest ejection fractions detected; Fig. 2) were similar to values obtained by transthoracic echocardiography (31.9±17.15%; lower limit, 29%; upper limit, 46%; Fig. 3). There was a significant positive correlation between angiographically- and echocardiographically-measured left ventricular ejection fractions (r=0.356; P=0.045). There was a significant difference between angiographically and echocardiographically measured low ejection fractions (P=0.041; Fig. 3). Using a cut-off of <36%, the sensitivity of echocardiography was found to be 75% (mean range, 55-95%) and specificity was found to be 68% (mean range, 35-95%).

Echocardiographically-measured left ventricular function was correlated with angiographically-calculated disease severity (r=0.656; P=0.012). Echocardiographically-measured
Table II. Angiographic findings of the enrolled patients.

| Characteristic                  | No. patients (n=2,445) | Spearman's correlation |
|--------------------------------|------------------------|------------------------|
|                                |                        | P-value | r-value |
| Mitral regurgitation<sup>a</sup> |                        |         |         |
| 0                              | 5 (0.2)                | 0.034   | 0.907   |
| 1                              | 885 (36.3)             |          |         |
| 2                              | 745 (30.5)             |          |         |
| 3                              | 468 (19)               |          |         |
| 4                              | 342 (14)               |          |         |
| Number of culprit vessel       |                        |         |         |
| 0                              | 5 (0.2)                | P-value<sup>e</sup>   | r-value<sup>e</sup> |
| 1                              | 1,358 (55.5)           |          |         |
| 2                              | 758 (31)               |          |         |
| 3                              | 311 (12.7)             |          |         |
| 4                              | 13 (0.6)               |          |         |
| Total                          | 3,859                  |          |         |
| Location of the culprit vessels|                        |         |         |
| Right coronary artery          | 1,485 (38)             | 0.735   | 0.21    |
| Left coronary artery           | 641 (17)               |          |         |
| Left anterior descending artery | 815 (21)               |          |         |
| Left circumflex artery         | 573 (15)               |          |         |
| Saphenous vein graft           | 345 (9)                |          |         |
| Culprit lesion type<sup>b</sup> |                        | P-value<sup>f</sup>   | r-value<sup>f</sup> |
| A                              | 556 (14)               |          |         |
| B1                             | 1,341 (35)             |          |         |
| B2                             | 1,152 (30)             |          |         |
| C                              | 810 (21)               |          |         |
| Culprit vessel collateral score<sup>c</sup> |                  | 0.947   |         |
| 0                              | 1,313 (34)             |          |         |
| 1                              | 1,245 (32)             |          |         |
| 2                              | 854 (22)               |          | 0.054   |
| 3                              | 447 (12)               |          |         |
| Culprit lesion thrombus score<sup>d</sup> |                    | 0.594   |         |
| 0                              | 1,341 (36)             |          |         |
| 1                              | 845 (22)               |          |         |
| 2                              | 741 (19)               |          | 0.406   |
| 3                              | 441 (11)               |          |         |
| 4                              | 289 (7)                |          |         |
| 5                              | 202 (5)                |          |         |

Data are presented as number (percentage). <sup>a</sup>0, absent; 1, mild; 2, moderate; 3, severe; 4, extreme. <sup>b</sup>A, <10 mm in size, concentric readily accessible, discrete, ≤45° smooth contour, absent or little calcification, side branches not involved, incomplete occlusion, not ostial and no thrombus present; B1, only one characteristic from the following: 10-20 mm in size, eccentric, proximal segment had moderate tortuosity, irregular contour, thrombus was present, moderate to heavy calcification, total occlusion >3 months old, and ostial/bifurcation lesion requiring two guide wires; B2, two or more from the characteristics of the B1 type; and C, >20 mm diffuse, proximal segment had excessive tortuosity, total occlusion >3 months old, involvement of side branches, friable lesions and degenerated vein graft. <sup>c</sup>The Rentrop classification: 0, the collateral flow was absent; 1, collateral flow only in secondary branches; 2, the collateral flow was present in the major vessel but did not reach the culprit lesion; 3, collateral flow reached the infarct-associated artery and the culprit lesion. <sup>d</sup>0, no thrombus; 1, possible thrombus; 2, dimension of the thrombus less than or equal to one-half of the diameter of the culprit vessel diameter; 3, dimension of the thrombus greater than one-half but less than or equal to two-thirds of the diameter of the culprit vessel diameter; 4, dimension of the thrombus greater than two-thirds of the diameter of the culprit vessel diameter but no total occlusion; 5, total culprit vessel occlusion. <sup>e</sup>Reference 1, number of culprit vessel; <sup>f</sup>Reference 2, culprit lesion type. P<0.05 was considered to indicate a statistically significant difference. Mitral regurgitation was compared with the number of culprit vessels. Location of the culprit vessels, Culprit vessel collateral score, and Culprit lesion thrombus score were compared with Culprit lesion type.
ejection fractions were significantly negatively correlated with angiographic jeopardy score \((r=-0.290; P=0.032)\). The left ventricular main obstruction measured by coronary angiography was correlated with left ventricular ejection fraction measured echocardiographically \((27.12\pm2.12\% vs. 27.31\pm5.05\%; r=0.71; P=0.016)\). The lower median left ventricular end-systolic volumes measured echocardiographically were correlated with those measured angiographically \((r=0.73; P=0.029; \text{Fig. 4})\). There were no correlations between the number of diseased vessels, collateral flow to the culprit vessel derived angiographically and worst left ventricular function evaluated echocardiographically \((P>0.05 \text{ for all comparisons}; \text{Table IV})\). There was no significant difference between the number of culprit vessels derived angiographically and echocardiographically \((P=0.058; \text{Fig. 5})\). Echocardiographically-derived mitral regurgitation on the same day as that derived angiographically. The \(r\) and \(P\)-values indicated that there was strong correlation between mitral regurgitation derived echocardiographically and mitral regurgitation derived angiographically \((1-2 \text{ vs. } 1-2; r=0.99; P=0.001; \text{Fig. 6})\).

**Discussion**

The current study investigated Chinese patients with acute myocardial infarction using angiography and transthoracic echocardiography. Myocardial infarction may be caused by a number of factors and several treatment options are available (11). The results obtained in the current study may aid the selection of optimal revascularization strategies and the identification of mortality-associated factors in patients with cardiogenic shock.
The current prospective cohort study suggested that transthoracic echocardiography and coronary angiography findings in patients with cardiogenic shock prior to revascularization were correlated with each other. The left ventricular
ejection fractions and mitral regurgitation grades were positively correlated among the enrolled patients. The results of the present study were in line with those of the SHOCK trial performed in Caucasian patients (4,15). However, certain differences in echocardiographically-derived results compared with coronary angiographically-derived results were observed in the present study. This is because echocardiograms are technically more difficult to perform in critically ill patients (including those requiring mechanical ventilation) compared with angiography (4). Perfusion imaging with contrast echocardiography (16) may improve accuracy and reduce the variability of echocardiography images.

The present study demonstrated that mitral regurgitation quantified echocardiographically was correlated with mitral regurgitation quantified angiographically. This result was similar to results obtained in the SHOCK trial (4,15). However, the present results differed from those of previously published studies (1,17-19) suggesting that mitral regurgitation is an independent factor for mortality in patients (derived from institutional records) with cardiogenic shock. Collectively, the present study suggests that mitral regurgitation is a marker of worse prognosis for patients with cardiogenic shock.

The numbers of culprit vessels derived angiographically and echocardiographically were correlated with the severity of mitral regurgitation. The results obtained in the current study were in line with those of the SHOCK trial (4,15), the SHORTWAVE study (7) and the STEMI study (18) performed in Caucasian patients. However, the aforementioned trials enrolled a limited number of the patients (127, 386 and 147, respectively). Mitral regurgitation is affected by presumed distortion in left ventricular geometry (4,20) and left ventricular dysfunction (21). A previous study (9) suggested that mitral regurgitation should be evaluated in patients with cardiogenic shock to assess cardiac risk.

In the present study, angiographic findings revealed that 1,082 patients had >1 culprit vessel while echocardiographic results showed that 1,161 patients had >1 culprit vessel. A possible reason for this could be plaque ruptures leading to thrombotic occlusion in multiple coronary arteries (22).

The present study had a number of limitations. As the current study enrolled a large population for angiography, echocardiography and revascularization procedures, different cardiologists, ultra-sonographers and medical and non-medical staff were involved and inter-and intra-evaluator reliabilities were not evaluated.

In addition, several aspects of the present study were not in line with those of the SHOCK trial (15). In particular, coronary angiography failed to derive a correlation between the number of culprit vessels and the location of the culprit vessels, between the culprit lesion type and the culprit vessel collateral score and between the culprit lesion type and culprit lesion thrombus score. Echocardiography failed to derive a correlation between the number of culprit vessels and degree of left ventricular diastolic dysfunction. A possible explanation is that the ejection fraction of the left ventricle is decreased in patients with cardiogenic shock (23). Therefore, a broad spectrum of left ventricular function is not available. Furthermore, excessive nitric oxide production has been reported in patients with cardiogenic shock and this inhibits myocardial contractility (24). Compensatory hyperkinesis is a normal manifestation of patients with cardiogenic shock (25) and this affects the ejection fraction of the heart. A further randomized trial is required to investigate such correlations.

Important parameters, including vitamin D, calcium, parathormone and phosphorous levels, were not evaluated in the present study. The degree of deposition of calcium on the mitral valve may help to predict the pathophysiology of patients with cardiogenic shock (26) as calcium deposition may cause elevation of the mitral leaflets (5). Exercise-induced electrocardiography has a better prediction of risk stratification of mitral regurgitation compared with electrocardiography (27) but was not performed in the current study. The present study did not evaluate epicardial fat thickness by electrocardiography and did not derive a correlation with mitral regurgitation. A possible justification is that, unlike magnetic resonance imaging and computed tomography, electrocardiography is not an exact method for the quantification of epicardial fat thickness (28). Follow-up data after percutaneous coronary intervention regarding the event-free survival and overall survival were not discussed.

In conclusion, the worst coronary artery outcomes are associated with more severe mitral regurgitation. Transthoracic echocardiography and coronary angiography results were correlated with the estimated ejection fractions in the current large population study, despite the vast operational difference between these two techniques in patients with cardiogenic shock.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors’ contributions

JC and YaH contributed equally to the conceptualization, literature review, data curation, funding and analysis of the study. YuH administrated the study, contributed to data curation, analysis, the literature review and methodology development. XY provided software and contributed to the literature review and methodology development. GZ contributed to resources, formal analysis and literature review of the study. JZ provided software and contributed to the literature review, data curation and formal analysis of the study. YY provided software, contributed to the literature review and conceptualization of the study and drafted, reviewed and edited the manuscript for intellectual content. All authors read and approved the manuscript for publication.
Ethics approval and consent to participate

The present study is registered in Research Registry (www.researchregistry.com; UID no. researchregistry4512; dated February 15, 2014). The protocol (CTH/CL/2/14; dated February 11, 2014) was approved by the Chongqing Traditional Chinese Medicine Hospital review board. All patients provided informed consent prior to enrollment in the study.

Patient consent for publication

All patients provided informed consent for publication prior to enrollment in the study.

Competing interests

The authors declare that they have no competing interests.

References

1. Rudiger A: Understanding cardiogenic shock. Eur J Heart Fail 17: 466-467, 2015.
2. Aissouli N, Puyrasset E, Tabone X, Charbonnier B, Schiele F, Lefèvre T, Durand E, Blanchard D, Simon T, Cambou JP and Danchin N: Improved outcome of cardiogenic shock at the acute stage of myocardial infarction: A report from the USIK 1995, USIC 2000, and FAST-MI French nationwide registries. Eur Heart J 33: 2535-2543, 2012.
3. Thiele H, Desch S, Pick JI, Stepińska J, Oldroyd K, Serpyts P, Montalescot G, Noc M, Huber K, Fuernau G, et al: CULPRIT-SHOCK Investigators. Multivessel versus culprit lesion only percutaneous revascularization plus potential staged revascularization in patients with acute myocardial infarction complicated by cardiogenic shock: Design and rationale of CULPRIT-SHOCK trial. Am Heart J 172: 160-169, 2016.
4. Berkowitz MJ, Picard MH, Harkness S, Sanborn TA, Hochman JS and Slater JN: Echocardiographic and angiographic correlations in patients with cardiogenic shock secondary to acute myocardial infarction. Am J Cardiol 98: 1004-1008, 2006.
5. Zemer WN, Shapira Y, Weisenberg D, Monakier D, Bental T, Sagie A and Vaturi M: Association between mitral annular calcification and flail mitral leafllet in degenerative mitral valve disease. Am J Cardiol 116: 124-125, 2015.
6. Saha SA, Beatty AL, Mishra RK, Wholey MA and Schiller NB: Usefulness of an echocardiographic composite cardiac calcium score to predict death in patients with stable coronary artery disease (from the Heart and Soul Study). Am J Cardiol 116: 1258-1259, 2015.
7. Paganello G, Faggiano P, Candido R, Tarantini L, Di Lenarda A, De Feo S and Cioffi G: The worrisome liaison between left ventricular systolic dysfunction and mitral annulus calcification in type 2 diabetes without coronary artery disease: Data from the SHORTWAVE study. Nutr Metab Cardiovasc Dis 23: 1188-1194, 2013.
8. Newby DE, Williams MC, Flapan AD, Forbes JF, Hargreaves AD, Leslie SJ, Lewis SC, McKillop G, McLean S, Reid JH, et al: Role of multidetector computed tomography in the diagnosis and management of patients attending the rapid access chest pain clinic. The Scottish computed tomography of the heart (SCOT-HEART) trial: Study protocol for randomized controlled trial. Trials 13: 184, 2012.
9. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP III, Guyton RA, O’Gara PT, Ruiz CE, Skubas NJ, Sorajja P, et al: 2014 AHA/ACC guideline for the management of patients with valvular heart disease: Executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 63: 2438-2488, 2014.
10. Hoe J: CT coronary angiography of chronic total occlusions of the coronary arteries: How to recognize and evaluate and usefulness for planning percutaneous coronary interventions. Int J Cardiovasc Imaging 25: 43-54, 2009.
11. Figueiras J, Otasegui I, Marti G, Domingo E, Baneras J, Barrabés JA, Del Blanco BG and Garcia-Dorado D: Area at risk and collateral circulation in a first acute myocardial infarction with occluded culprit artery. STEMIs vs. non-STEMIs patients. Int J Cardiol 258: 14-19, 2019.
12. Duman H, Cetin M, Durakoglugil ME, Degirmenci H, Hamur H, Bostan M, Karadag Z and Cicek Y: Relation of angiographic thrombus burden with severity of coronary artery disease in patients with ST-segment elevation myocardial infarction. Med Sci Monit 17: 3540-3546, 2011.
13. Suzue M, Mori K, Inoue M, Hayabuchi Y, Nakagawa R and Kagami S: Developmental changes in the left ventricular diastolic wall strain on M-mode echocardiography. J Echocardiogr Diag 12: 98-105, 2014.
14. Gent AD, Negishi K, Negishi T, Collier P, Kapadia SR, Thomas JD, Marwick TH, Griffin BP and Popovic ZB: Grading diastolic function by echocardiography: Hemodynamic validation of existing guidelines. Cardiovasc Ultrasound 13: 28, 2015.
15. Sanborn TA, Sleeper LA, Webb JG, French JK, Bergman G, Parikh M, Wong SC, Boland J, Pfisterer SL, Slater JN, et al: Correlates of one-year survival in patients with cardiogenic shock complicating acute myocardial infarction: Angiographic findings from the SHOCK trial. J Am Coll Cardiol 42: 1373-1379, 2003.
16. Eskandari M and Monaghan M: Contrast echocardiography in daily clinical practice. Herz 42: 271-278, 2017.
17. Song JM, Kang SH, Lee EJ, Shin MJ, Lee JW, Chang CH, Kim DH, Kang DH and Song KJ: Echocardiographic predictors of left ventricular function and clinical outcomes after successful mitral valve repair: Conventional two-dimensional versus speckle-tracking parameters. Ann Thorac Surg 91: 1816-1823, 2011.
18. Engström AE, Vis MM, Bouma BJ, Claessen BE, Sjauw KD, Baan J, Meeuwissen M, Koch KT, de Winter RJ, Tijssen JG, et al: Mitral regurgitation is an independent predictor of 1-year mortality in ST-elevation myocardial infarction patients presenting in cardiogenic shock on admission. Acute Care Card 12: 51-57, 2010.
19. Harjola VP, Lassus J, Sionis A, Kober L, Tarvainen M, Spinar J, Parissis J, Banaszewski M, Silva-Cardoso J, Carubelli V, et al: Clinical picture and risk prediction of short-term mortality in cardiogenic shock. Eur J Heart Fail 17: 501-509, 2015.
20. Timek TA, Lai DT, Bothe W, Liang D, Daughters GT, Ingels NB and Miller DC: Geometric perturbations in multiheaded papillary tip positions associated with acute ovine ischemic mitral regurgitation. J Thorac Cardiovasc Surg 150: 232-237, 2015.
21. Tu Y, Zeng QC, Huang Y and Li JY: Percutaneous coronary intervention for acute myocardial infarction with mitral regurgitation. J Geriatr Cardiol 13: 521-527, 2016.
22. Ananthakrishna R, Wang LJ, Zhao LP and Tan HC: Double jeopardy in acute ST-segment elevation myocardial infarction. Singapore Med J 58: 225-227, 2017.
23. Rogers PA, Daye J, Huang H, Blaustein A, Virani S, Alam M, Kumar A, Paniagua D, Kar B, Bozkurt B, et al: Revascularization improves mortality in elderly patients with acute myocardial infarction complicated by cardiogenic shock. Int J Cardiol 172: 239-241, 2014.
24. Wong VW and Lerner E: Nitric oxide inhibition strategies. Future Sci OA 1: pii: FSO35, 2015.
25. Wu HM and Tzeng BH: Dynamic left ventricular outflow tract obstruction with cardiogenic shock in apical ballooning syndrome. Acta Cardiol Sin 29: 370-373, 2013.
26. Pandit A, Moosafad M, Boddou S, Pandit A, Tandar A, Chaliki H, Cha S and Lee HR: Vitamin D levels and left ventricular diastolic function. Open Heart 1: e000111, 2014.
27. Dulgheru R, Marchetta S, Sugimoto T, Go YY, Girbea A, Oury C and Lancellotti P: Exercise testing in mitral regurgitation. Prog Cardiovasc Dis 60: 342-350, 2017.
28. Park JS, Lee YH, Seo KW, Choi BJ, Choi SY, Hwang GS, Taek JH and Shin JH: Echocardiographic epicardial fat thickness is a predictor for target vessel revascularization in patients with ST-elevation myocardial infarction. Lipids Health Dis 15: 194, 2016.

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