Comparison of Left Ventricular Function and Myocardial Infarct Size Determined by 2-Dimensional Speckle Tracking Echocardiography in Patients With and Without Chronic Obstructive Pulmonary Disease After ST-Segment Elevation Myocardial Infarction

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Patients with chronic obstructive pulmonary disease (COPD) have a high risk of mortality after acute ST-segment elevation myocardial infarction (STEMI). We compared STEMI patients with versus without COPD in terms of infarct size and left ventricular (LV) systolic function using advanced 2-dimensional speckle tracking echocardiography. Of 1,750 patients with STEMI (mean age 61 ± 12 years, 76% male), 133 (7.6%) had COPD. With transthoracic echocardiography, left ventricular ejection fraction (LVEF) and wall motion score index were measured. Infarct size was assessed using biomarkers (creatine kinase and troponin T). LV global longitudinal strain (GLS), reflecting active LV myocardial deformation, was measured with 2-dimensional speckle tracking echocardiography to estimate LV systolic function and infarct size. STEMI patients with COPD were significantly older, more likely to be former smokers, and had worse renal function compared with patients without COPD. There were no differences in infarct size based on peak levels of creatine kinase (1315 [613 to 2181] vs 1477 [682 to 3047] U/l, p = 0.106) and troponin T (3.3 [1.4 to 7.3] vs 3.9 [1.5 to 7.8] μg/l, p = 0.489). Left ventricular ejection fraction (46% vs 47%, p = 0.591) and wall motion score index (1.38 [1.25 to 1.66] vs 1.38 [1.19 to 1.69], p = 0.690) were comparable. In contrast, LV GLS was significantly more impaired in patients with COPD compared with patients without COPD (−13.9 ± 3.0% vs −14.7 ± 3.9%, p = 0.034). In conclusion, despite comparable myocardial infarct size and LV systolic function as assessed with biomarkers and conventional echocardiography, patients with COPD exhibit more impaired LV GLS on advanced echocardiography than patients without COPD, suggesting a greater functional impairment at an early stage after STEMI. **(Am J Cardiol 2017;120:734–739)**

Patients with chronic obstructive pulmonary disease (COPD) who present with ST-segment elevation myocardial infarction (STEMI) have higher in-hospital and 6-month mortality rates compared with patients without COPD.[1–3] Possible mechanisms underlying this worse prognosis in patients with COPD include poor recognition and management of myocardial infarction, underutilization of secondary prevention therapies, and pathophysiological factors related to COPD (i.e. chronic inflammation).[3,4] These factors may result in larger infarct size and worse left ventricular (LV) systolic function. However, it remains unknown whether STEMI patients with COPD differ from patients without COPD in terms of infarct size and LV systolic function acutely after STEMI. LV ejection fraction (EF) and wall motion score index (WMSI) are commonly used in clinical practice to estimate LV systolic function and for risk stratification of patients with STEMI.[5,6] However, these conventional echocardiographic parameters may not be sensitive enough to characterize the extent of myocardial damage after STEMI.[7,8] Two-dimensional (2D) speckle tracking echocardiography global longitudinal strain (GLS), reflecting active deformation of the LV myocardium, has emerged as a valuable index of LV systolic function and infarct size.[9] The present study aimed at evaluating the differences in infarct size and systolic function in STEMI patients with versus without COPD by measuring biomarkers (creatine kinase [CK] and troponin T) as well as conventional and advanced 2D speckle tracking echocardiography.

Methods

Of an ongoing registry of patients admitted with acute STEMI and treated with primary percutaneous coronary intervention (PCI) at the Leiden University Medical Centre (Leiden, The

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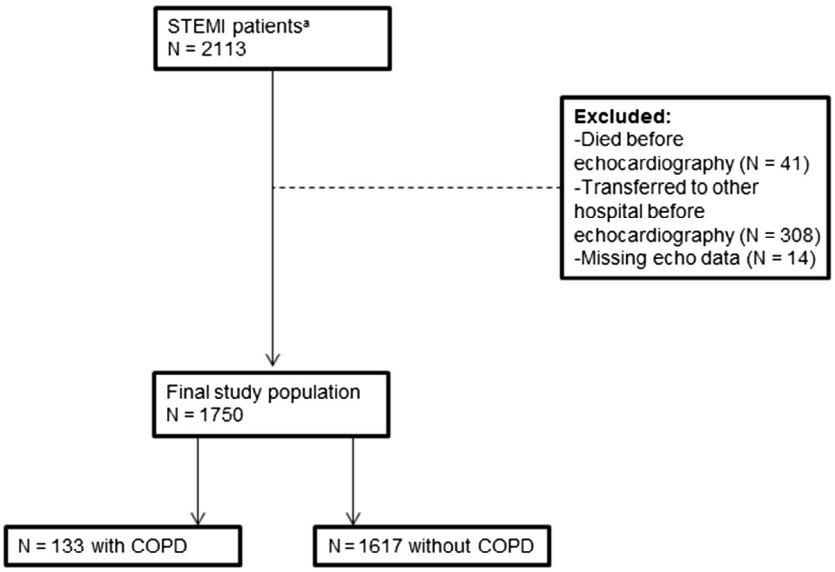

Figure 1. Flowchart of patient selection and enrollment for analysis. [a]Patients with STEMI were admitted between February 2004 and May 2013.

Netherlands), patients with complete echocardiographic data at baseline (within 48 hours of admission) were included (Figure 1).[10] Patients were managed according to the most recent American College of Cardiology/American Heart Association and European Society of Cardiology guidelines.[6,11] This includes systematic measurements of CK and troponin T and transthoracic echocardiography within 48 hours of admission. Clinical and echocardiographic data were prospectively collected at the departmental Cardiology Information System (EPDvision, Leiden University Medical Centre) and echocardiography database, respectively, and analyzed retrospectively. For this retrospective analysis of clinically acquired data, the institutional review board waived the need for patient written informed consent.

Patient demographic and clinical characteristics were recorded. Hypertension was defined as a systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg, or previous use of antihypertensive drugs. Hypercholesterolemia was defined as having a recorded history of hypercholesterolemia and/or use of statins. Diabetes mellitus was defined as having a history of diabetes and subsequent treatment with a diet, oral glucose-lowering agents, or insulin. Killip class was registered at admission.[12] During invasive coronary angiography, the culprit vessel was identified and multivessel disease was defined as more than 1 vessel with >50% luminal stenosis.

COPD was defined as having a documented history of COPD at admission, after reviewing the entire medical record. When available, pulmonary function tests were considered, and COPD was diagnosed if the ratio of forced expiratory volume in one second to forced vital capacity ratio was <0.7.[13] The use of this definition of COPD, when systematic pulmonary function tests are not available, is in line with previous studies.[1,2,14–16] Patients with a recorded history of asthma were listed as non-COPD, given the different pathophysiology.[13]

Images were obtained with the patient at rest, in left lateral decubitus position using a commercially available system (Vivid 7 and E9, GE Healthcare, Horten, Norway). Standard 2D images were obtained from the parasternal (long- and short-axis) and apical (long-axis, 2- and 4-chamber) views, using 3.5-MHz or M5S transducers, and digitally stored in cine-loop format. Furthermore, color, pulsed, and continuous wave Doppler images were acquired. Offline analysis of the obtained images was performed using EchoPAC (version BT13, GE Medical Systems, Horten, Norway).

Left ventricular diameters and wall thickness were measured in the parasternal long-axis view. Subsequently, LV mass was calculated using the Devereux's formula and indexed for body surface area.[5] Left ventricular end-systolic and end-diastolic volumes were measured in apical 4- and 2-chamber views and left ventricular ejection fraction (LVEF) was calculated using the biplane Simpson's method.[5] For calculation of the WMSI, the LV was divided into 16 segments. Each segment was assessed and scored based on its motion and systolic thickening (1 = normokinesia, 2 = hypokinesia, 3 = akinesia, 4 = dyskinesia). The WMSI was calculated as the sum of the individual segment scores divided by the number of segments.[5]

Left ventricular diastolic function was assessed based on peak velocity of the early diastolic filling (E-wave) and late filling by atrial contraction (A-wave), together with E-wave deceleration time obtained on the pulsed-wave Doppler recordings of the transmitral flow, the left atrial volume, and pulmonary systolic pressures estimated from the tricuspid regurgitation peak velocity on continuous wave Doppler recordings.[17]

For evaluation of LV GLS, 2D speckle tracking analysis was performed as previously described.[18] In brief, the LV endocardial border was traced at an end-systolic frame in the apical long-axis, 2-and 4-chamber views. The automatically created region of interest was manually adjusted to the thickness of the myocardium. Thereafter, the myocardium was automatically tracked throughout the cardiac cycle. LV GLS was calculated by the software as the average of the peak systolic strain of the three apical views, and presented in a 17-segment "bull's-eye" plot. The inter- and intraobserver variability of LV GLS measurements have been previously reported as mean absolute difference of $1.2 \pm 0.5\%$ and $0.9 \pm 1.0\%$, respectively.[19]

Statistical analyses were performed using SPSS software (version 23, IBM SPSS statistics for Windows, Armonk, New York). Continuous variables are presented as mean ± standard

Table 1
Clinical characteristics

Variable	COPD		p value
	Yes (N = 133)	No (N = 1617)	
Age (years)	69 ± 11	60 ± 12	<0.001
Men	97 (73%)	1229 (76%)	0.427
Systolic blood pressure (mmHg)	140 [120–160]	135 [120–150]	0.096
Diastolic blood pressure (mmHg)	84 ± 18	81 ± 16	0.071
Heart rate (beats/min)	73 ± 16	74 ± 18	0.709
Body mass index (kg/m^2)	25.7 [24–29]	26.0 [24–28]	0.502
Killip class			0.045
<2	121 (92%)	1539 (96%)	
≥2	11 (8%)	72 (4%)	
Diabetes mellitus	19 (14%)	168 (10%)	0.163
Hypertension	54 (41%)	569 (35%)	0.226
Hypercholesterolemia	27 (21%)	325 (20%)	0.879
Family history of cardiovascular disease	47 (37%)	705 (44%)	0.133
Current smoker	68 (51%)	764 (47%)	0.396
Ex-smoker	23 (17%)	173 (11%)	0.021
Previous myocardial infarction	12 (9%)	47 (3%)	<0.001
Culprit coronary artery			0.389
Left anterior descending	49 (37%)	721 (45%)	
Right coronary artery	55 (42%)	618 (38%)	
Circumflex coronary artery	26 (20%)	258 (16%)	
Left main	1 (1%)	10 (1%)	
Number of diseased vessels			0.225
1	61 (46%)	751 (47%)	
2	31 (24%)	555 (35%)	
3	39 (30%)	304 (19%)	
Peak creatinine kinase (U/L)	1316 [613–2183]	1477 [682–3047]	0.106
Peak troponin T (μg/L)	3.3 [1.4–7.3]	3.9 [1.5–7.8]	0.489
Glucose (mmol/L)	7.5 [6.3–9.8]	8.0 [6.6–9.7]	0.223
Creatinine clearance (ml/min/1.73 m^2)	84 ± 33	99 ± 37	<0.001
Medication at discharge			
β-blocker	114 (89%)	1507 (95%)	0.011
ACE inhibitor or angiotensin-II receptor blocker	124 (98%)	1555 (98%)	1.000
Aspirin	124 (97%)	1530 (96%)	0.814
Thienopyridines*	127 (99%)	1587 (100%)	0.462
Statin	128 (100%)	1581 (99%)	0.616

COPD = chronic obstructive pulmonary disease. Continuous variables are presented as mean ± standard deviation or median [25th to 75th percentile]. *Clopidogrel or prasugrel.

deviation when normally distributed, and median and interquartile range otherwise. Categorical variables are presented as frequencies and percentages. Differences in continuous variables between patients with and without COPD were analyzed using the unpaired Student's *t* test or Mann-Whitney U-test as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test as appropriate. A 2-tailed p value of <0.05 was considered statistically significant.

Results

Of 1,750 patients with STEMI, 133 patients (7.6%) had a history of COPD. The clinical characteristics of the patients with and without COPD are presented in Table 1. Compared with patients without COPD, patients with COPD were significantly older, more likely to be former smokers, and had a higher prevalence of previous myocardial infarction. In addition, patients with COPD had a significantly worse kidney function and presented more frequently with heart failure. Guideline-based medical therapy at discharge was similar in groups, except for β-blockers, which were less frequently prescribed in patients with COPD compared with patients without COPD (89% and 95%, p = 0.011).

There were no statistically significant differences in patients with and without COPD in terms of infarct size based on peak levels of CK (1315 [613 to 2181] vs 1477 [682 to 3047] U/l, p = 0.106, respectively) and troponin T (3.3 [1.4 to 7.3] vs 3.9 [1.5 to 7.8] μg/l, p = 0.489, respectively).

Echocardiographic characteristics of the patients with and without COPD are presented in Table 2. Compared with patients without COPD, patients with COPD had significantly smaller LV dimensions. LVEF was similar in both patients with and without COPD (46 ± 10% and 47 ± 9%, respectively, p = 0.591). In addition, the WMSI was 1.38 [1.25 to 1.66] for patients with COPD and 1.38 [1.19 to 1.69] for patients without COPD, p = 0.690. However, LV GLS was significantly more impaired in patients with COPD compared with patients without COPD (−13.9 ± 3.0% and −14.7 ± 3.9%, p = 0.034), indicating more reduced systolic LV function and larger area of infarction in the patients with

Table 2
Echocardiographic characteristics

Variable	COPD		p value
	Yes (n = 133)	No (n = 1617)	
LV end-diastolic diameter (mm)	46 ± 7	48 ± 6	0.001
LV end-systolic diameter (mm)	30 [26–37]	32 [27–37]	0.101
Posterior wall thickness (mm)	12 ± 2.2	11 ± 2.1	0.031
Interventricular septum thickness (mm)	11 [10–13]	11 [10–13]	0.181
LV mass (g)	203 ± 75	212 ± 66	0.134
Indexed LV mass (g/m^2)	104 ± 34	108 ± 30	0.171
LV end-systolic volume (mL)	48 [39–61]	53 [42–68]	0.012
LV end-diastolic volume (mL)	92 [74–116]	101 [82–123]	0.009
Wall motion score index	1.38 [1.25–1.66]	1.38 [1.19–1.69]	0.690
LV ejection fraction (%)	46 ± 10	47 ± 9	0.591
LV global longitudinal strain (%)	−13.9 ± 3.9	−14.7 ± 3.9	0.034
E-wave peak velocity (cm/s)	66 ± 18	66 ± 19	0.885
A-wave peak velocity (cm/s)	76 ± 21	71 ± 24	0.051
E/A ratio	0.84 [0.69–1.06]	0.91 [0.72–1.14]	0.039
E-wave deceleration time (ms)	215 ± 85	210 ± 72	0.638

COPD = chronic obstructive pulmonary disease; LV = left ventricle. Continuous variables are presented as mean ± standard deviation or median [25th to 75th percentile].

COPD despite similar infarct size according to enzyme release (biomarkers). Figure 2 presents a more impaired LV GLS in a STEMI patient with COPD compared with a patient without COPD, with comparable LVEF, WMSI, and infarct size based on peak levels of CK and troponin T.

Discussion

The present study demonstrates that STEMI patients with concomitant COPD have more impaired LV systolic function and larger infarct area based on LV GLS compared with patients without COPD, despite having similar infarct size as assessed with cardiac biomarkers. These findings suggest that STEMI patients with COPD have greater impairment of LV systolic function at an early stage after STEMI. LVEF and WMSI may not be sensitive enough to detect this impairment of LV systolic function.

Infarct size is an important prognostic marker after STEMI.[20] Creatine kinase and troponin levels and imaging modalities, including echocardiography, nuclear imaging techniques, and late gadolinium contrast-enhanced cardiac magnetic resonance, are well-established methods to estimate the infarct size.[21] Whether the presence of COPD has influence on infarct size has not been extensively evaluated.[22,23] In 818 patients with STEMI (8.6% with COPD), Lazzeri et al showed no significant differences in peak troponin I in patients with and without COPD (54.1 [28.7 to 212] vs 92.2 [44.1 to 186.0] ng/ml, p = 0.084, respectively).[22] Similar results were demonstrated by Wakabayashi et al in 365 STEMI patients with COPD compared with 2,884 patients without COPD (peak CK-MB 59.0 ± 93.2 ng/ml vs 63.5 ± 97.2 ng/ml, p = 0.4, respectively).[23] The present study provides additional information by providing echocardiographic estimates of infarct size. LVEF and WMSI measured on echocardiography have been shown to correlate with infarct size.[20,24] Particularly, WMSI may reflect infarct size better than LVEF.[25] In this large cohort of patients, LVEF and WMSI were not significantly different between STEMI patients with COPD and patients without COPD, suggesting that both groups of patients have comparable myocardial damage based on these conventional echocardiographic parameters.

Although LVEF and WMSI are routine echocardiographic parameters measured in patients with STEMI to estimate LV systolic function and for risk stratification, the advent of speckle tracking echocardiography (which measures LV strain, deformation of the LV myocardium) has permitted obtaining more sensitive parameters of LV systolic function and better reflectors of myocardial damage.[24,26] Both global and regional longitudinal strain have shown to be accurate in evaluating global LV function and the presence of segments with transmural necrosis using cardiac magnetic resonance as a reference.[27] In addition, using single-photon emission computed tomography myocardial perfusion imaging as reference, LV GLS has demonstrated to be superior to LVEF in predicting the infarct size at 30-day follow-up.[26]

In the present study, STEMI patients with COPD showed more impaired LV GLS compared with patients without COPD, despite having similar levels of biomarkers and comparable values of conventional echocardiographic parameters of LV systolic function. These results suggest that STEMI patients with COPD have larger myocardial damage than patients without COPD. Therefore, measuring LV GLS in the acute phase of STEMI may be a better marker of true myocardial damage in patients with COPD than conventional echocardiographic parameters and may be a more sensitive parameter for risk stratification of these patients. The underlying mechanisms explaining worse LV GLS as a reflection of larger infarct size in patients with COPD may relate to the chronic inflammatory status of patients with COPD. The severity of the airflow obstruction has been shown to influence the inflammatory status, and patients with severe COPD have shown higher levels of C-reactive protein and higher cardiac infarction injury score compared with patients without airflow obstruction.[28] In addition, systemic inflammation has been linked to reperfusion injury, causing larger myocardial infarct size.[29] Future research is needed to elaborate this physiopathology.

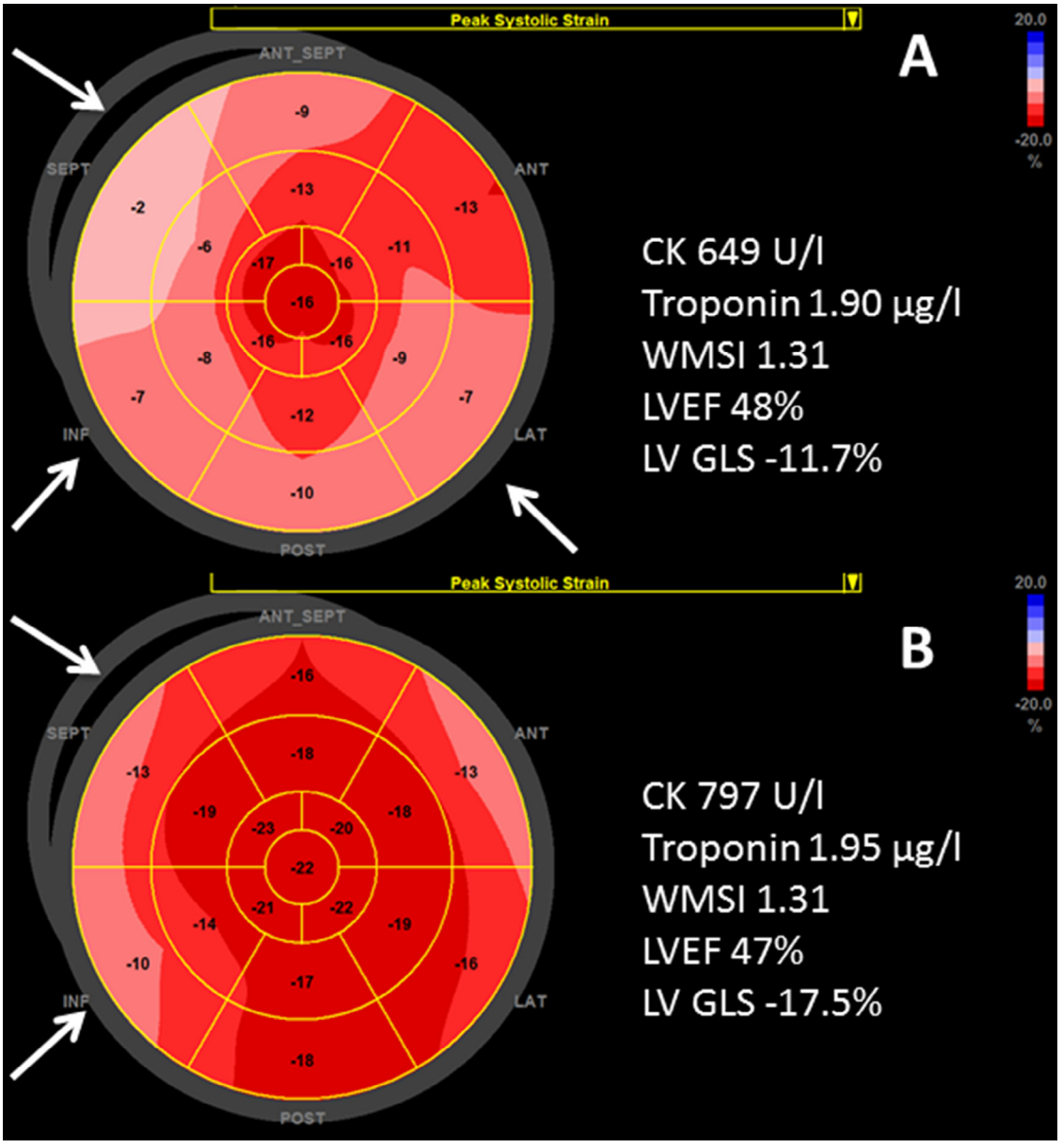

Figure 2. Bull's-eye plot of left ventricular (LV) global longitudinal strain (GLS) assessed by speckle tracking echocardiography. (*A*) STEMI patient with COPD (culprit vessel RCA), with LVEF 48% (comparable with patient B without COPD). LV GLS was more impaired (less negative) than in patient B without COPD, −11.7% vs −17.5%, respectively. (*B*) STEMI patient without COPD (culprit vessel RCA) with LVEF 47%. Biomarkers for infarct size (CK and troponin) were similar in both patients. Arrows indicate the infarct location. ANT = anterior; ANT_SEPT = anteroseptal; CK = creatine phosphokinase; INF = inferior; LAT = lateral; LVEF = left ventricular ejection fraction; POST = posterior; RCA = ramus descendens anterior; SEPT = septal; WMSI = wall motion score index.

Several study limitations should be acknowledged. First, this is a single centre, retrospective study that cannot provide causal relations. Second, the presence or absence of COPD in the study population was solely based on patients' medical records as pulmonary functional tests were not systematically available. However, we did find a similar prevalence of COPD in our population compared with the existing studies in the literature, which used the same definition of COPD.[1,14] In addition, without pulmonary functional tests, we were not able to classify the severity of COPD.

In conclusion, STEMI patients with and without COPD have comparable myocardial infarct size and LV systolic function as assessed with biomarkers and conventional echocardiography, respectively. However, patients with COPD exhibit more impaired LV GLS than their counterparts, suggesting more myocardial damage and worse LV systolic function in the early phase after STEMI in patients with COPD.

Disclosures

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1. Enriquez JR, Parikh SV, Selzer F, Jacobs AK, Marroquin O, Mulukutla S, Srinivas V, Holper EM. Increased adverse events after percutaneous coronary intervention in patients with COPD: insights from the National Heart, Lung, and Blood Institute dynamic registry. *Chest* 2011;140:604–610.
2. Campo G, Guastaroba P, Marzocchi A, Santarelli A, Varani E, Vignali L, Sangiorgio P, Tondi S, Serenelli C, De Palma R, Saia F. Impact of COPD on long-term outcome after ST-segment elevation myocardial infarction receiving primary percutaneous coronary intervention. *Chest* 2013;144:750–757.
3. Rothnie KJ, Quint JK. COPD and acute myocardial infarction: effects on presentation, management and outcomes. *Eur Heart J Qual Care Clin Outcomes* 2016;2:81–90.
4. Rothnie KJ, Smeeth L, Herrett E, Pearce N, Hemingway H, Wedzicha J, Timmis A, Quint JK. Closing the mortality gap after a myocardial infarction in people with and without chronic obstructive pulmonary disease. *Heart* 2015;101:1103–1110.
5. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt JU. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015;28:1–39, e14.
6. Steg PG, James SK, Atar D, Badano LP, Lundqvist CB, Borger MA, Di Mario C, Dickstein K, Ducrocq G, Fernandez-Aviles F, Gershlick AH, Giannuzzi P, Halvorsen S, Huber K, Juni P, Kastrati A, Knuuti J, Lenzen MJ, Mahaffey KW, Valgimigli M, van't Hof A, Widimsky P, Zahger D, Bax JJ, Baumgartner H, Ceconi C, Dean V, Deaton C, Fagard R, Funck-Brentano C, Hasdai D, Hoes A, Kirchhof P, Knuuti J, Kolh P, McDonagh T, Moulin C, Popescu BA, Reiner Ž, Sechtem U, Sirnes PA, Tendera M, Torbicki A, Vahanian A, Windecker S, Hasdai D, Astin F, Åström-Olsson K, Budaj A, Clemmensen P, Collet J-P, Fox KA, Fuat A, Gustiene O, Hamm CW, Kala P, Lancellotti P, Maggioni AP, Merkely B, Neumann F-J, Piepoli MF, Van de Werf F, Verheugt F, Wallentin L. ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Eur Heart J* 2012;33:2569–2619.
7. Antoni ML, Mollema SA, Delgado V, Atary JZ, Borleffs CJ, Boersma E, Holman ER, van der Wall EE, Schalij MJ, Bax JJ. Prognostic importance of strain and strain rate after acute myocardial infarction. *Eur Heart J* 2010;31:1640–1647.
8. Joyce E, Hoogslag GE, Leong DP, Debonnaire P, Katsanos S, Boden H, Schalij MJ, Marsan NA, Bax JJ, Delgado V. Association between left ventricular global longitudinal strain and adverse left ventricular dilatation after ST-segment-elevation myocardial infarction. *Circ Cardiovasc Imaging* 2014;7:74–81.
9. Dimitriu-Leen AC, Scholte AJ, Katsanos S, Hoogslag GE, van Rosendael AR, van Zwet EW, Bax JJ, Delgado V. Influence of myocardial ischemia extent on left ventricular global longitudinal strain in patients after ST-segment elevation myocardial infarction. *Am J Cardiol* 2017;119:1–6.
10. Liem S-S, van der Hoeven BL, Oemrawsingh PV, Bax JJ, van der Bom JG, Bosch J, Viergever EP, van Rees C, Padmos I, Sedney MI. MISSION!: optimization of acute and chronic care for patients with acute myocardial infarction. *Am Heart J* 2007;153:14, e1-11.
11. O'Gara PT, Kushner FG, Ascheim DD, Casey DE, Chung MK, de Lemos JA, Ettinger SM, Fang JC, Fesmire FM, Franklin BA, Granger CB, Krumholz HM, Linderbaum JA, Morrow DA, Newby LK, Ornato JP, Ou N, Radford MJ, Tamis-Holland JE, Tommaso CL, Tracy CM, Woo YJ, Zhao DX. 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. *J Am Coll Cardiol* 2013;61:e78–e140.
12. Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit. *Am J Cardiol* 1967;20:457–464.
13. Gómez FP, Rodriguez-Roisin R. Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines for chronic obstructive pulmonary disease. *Curr Opin Pulm Med* 2002;8:81–86.
14. Bursi F, Vassallo R, Weston SA, Killian JM, Roger VL. Chronic obstructive pulmonary disease after myocardial infarction in the community. *Am Heart J* 2010;160:95–101.
15. Salisbury AC, Reid KJ, Spertus JA. Impact of chronic obstructive pulmonary disease on post-myocardial infarction outcomes. *Am J Cardiol* 2007;99:636–641.
16. Stefan MS, Bannuru RR, Lessard D, Gore JM, Lindenauer PK, Goldberg RJ. The impact of COPD on management and outcomes of patients hospitalized with acute myocardial infarction: a 10-year retrospective observational study. *Chest* 2012;141:1441–1448.
17. Nagueh SF, Smiseth OA, Appleton CP, Byrd BF 3rd, Dokainish H, Edvardsen T, Flachskampf FA, Gillebert TC, Klein AL, Lancellotti P, Marino P, Oh JK, Alexandru Popescu B, Waggoner AD. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging* 2016;17:1321–1360.
18. Negishi K, Negishi T, Kurosawa K, Hristova K, Popescu BA, Vinereanu D, Yuda S, Marwick TH. Practical guidance in echocardiographic assessment of global longitudinal strain. *JACC Cardiovasc Imaging* 2015;8:489–492.
19. Ng AC, Delgado V, Bertini M, van der Meer RW, Rijzewijk LJ, Shanks M, Nucifora G, Smit JW, Diamant M, Romijn JA, de Roos A, Leung DY, Lamb HJ, Bax JJ. Findings from left ventricular strain and strain rate imaging in asymptomatic patients with type 2 diabetes mellitus. *Am J Cardiol* 2009;104:1398–1401.
20. Burns RJ, Gibbons RJ, Yi Q, Roberts RS, Miller TD, Schaer GL, Anderson JL, Yusuf S. The relationships of left ventricular ejection fraction, end-systolic volume index and infarct size to six-month mortality after hospital discharge following myocardial infarction treated by thrombolysis. *J Am Coll Cardiol* 2002;39:30–36.
21. Giannitsis E, Steen H, Kurz K, Ivandic B, Simon AC, Futterer S, Schild C, Isfort P, Jaffe AS, Katus HA. Cardiac magnetic resonance imaging study for quantification of infarct size comparing directly serial versus single time-point measurements of cardiac troponin T. *J Am Coll Cardiol* 2008;51:307–314.
22. Lazzeri C, Valente S, Attana P, Chiostri M, Picariello C, Gensini GF. The prognostic role of chronic obstructive pulmonary disease in ST-elevation myocardial infarction after primary angioplasty. *Eur J Prev Cardiol* 2013;20:392–398.
23. Wakabayashi K, Gonzalez MA, Delhaye C, Ben-Dor I, Maluenda G, Collins SD, Syed AI, Gaglia MA Jr, Torguson R, Xue Z, Suddath WO, Satler LF, Kent KM, Lindsay J, Pichard AD, Waksman R. Impact of chronic obstructive pulmonary disease on acute-phase outcome of myocardial infarction. *Am J Cardiol* 2010;106:305–309.
24. Eek C, Grenne B, Brunvand H, Aakhus S, Endresen K, Hol PK, Smith HJ, Smiseth OA, Edvardsen T, Skulstad H. Strain echocardiography and wall motion score index predicts final infarct size in patients with non-ST-segment-elevation myocardial infarction. *Circ Cardiovasc Imaging* 2010;3:187–194.
25. Baron T, Flachskampf FA, Johansson K, Hedin EM, Christersson C. Usefulness of traditional echocardiographic parameters in assessment of left ventricular function in patients with normal ejection fraction early after acute myocardial infarction: results from a large consecutive cohort. *Eur Heart J Cardiovasc Imaging* 2016;17:413–420.
26. Munk K, Andersen NH, Nielsen SS, Bibby BM, Botker HE, Nielsen TT, Poulsen SH. Global longitudinal strain by speckle tracking for infarct size estimation. *Eur J Echocardiogr* 2011;12:156–165.
27. Cimino S, Canali E, Petronilli V, Cicogna F, De Luca L, Francone M, Sardella G, Iacoboni C, Agati L. Global and regional longitudinal strain assessed by two-dimensional speckle tracking echocardiography identifies early myocardial dysfunction and transmural extent of myocardial scar in patients with acute ST elevation myocardial infarction and relatively preserved LV function. *Eur Heart J Cardiovasc Imaging* 2013;14:805–811.
28. Sin DD, Man SFP. Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular diseases? The potential role of systemic inflammation in chronic obstructive pulmonary disease. *Circulation* 2003;107:1514–1519.
29. Blancke F, Claeys MJ, Jorens P, Vermeiren G, Bosmans J, Wuyts FL, Vrints CJ. Systemic inflammation and reperfusion injury in patients with acute myocardial infarction. *Mediators Inflamm* 2005;6:385–389.