Risk of Heart Failure Complication During Hospitalization for Acute Myocardial Infarction in a Contemporary Population

Insights From the National Cardiovascular Data ACTION Registry

Ravi V. Shah, MD; DaJuanicia Holmes, MS; Monique Anderson, MD; Tracy Y. Wang, MD, MHS, MSc; Michael C. Kontos, MD; Stephen D. Wiviott, MD; Benjamin M. Scirica, MD, MPH

Background—Patients with acute myocardial infarction (MI) complicated by heart failure (HF) are subject to higher mortality during the index hospitalization. Early risk prediction and intervention may help prevent HF-related morbidity and mortality.

Methods and Results—We examined 77 675 ST-elevation MI and 110 128 non-ST-elevation patients with MI without cardiogenic shock or HF at presentation treated at 609 hospitals in Acute Coronary Treatment and Intervention Outcomes Network Registry (ACTION) Registry-Get With The Guidelines between January 1, 2007, and March 31, 2011. Logistic regression identified patient characteristics associated with development of in-hospital HF. Overall, 3.8% of patients with MI developed in-hospital HF, which was associated with higher mortality in both ST-elevation MI and non-ST elevation MI. In multivariable logistic regression, left ventricular ejection fraction ≤30%, prior HF, diabetes mellitus, female sex, ST-elevation MI, and hypertension (all $P<0.005$) were independently associated with in-hospital HF. Patients who developed HF during non-ST-elevation MI were more likely to be medically managed without catheterization (30% versus 13% with HF, $P<0.0001$) or had longer delays to surgical or percutaneous revascularization. Patients with ST-elevation MI and HF were less likely to receive primary percutaneous coronary revascularization (84% versus 79% with HF, $P<0.0001$), and more likely to receive thrombolytic therapy (14% versus 11%; $P=0.0001$).

Conclusions—Patients with MI who develop HF during hospitalization have a higher risk clinical profile and greater mortality, but may be less likely to receive revascularization in a timely fashion. Targeting these highest risk patients may improve outcome post-MI. **(*Circ Heart Fail*. 2012;5:693-702.)**

Key Words: heart failure ■ myocardial infarction ■ outcomes

Heart failure (HF) during admission for acute myocardial infarction (MI) is an important predictor of short- and long-term clinical outcomes, and various studies have attempted to examine predictors of subsequent HF hospitalization after index MI.[1–9] Despite greater use of early coronary revascularization, which has improved clinical outcomes during index hospitalization for MI,[10] the incidence of HF during index MI has been reported as high as 20% to 30% in contemporary registries,[6,11] and represents a major source of in-hospital morbidity, length of stay, readmission, and downstream costs.[4,12] Given the rapidly expanding therapeutic options for patients with HF, identification of contemporary predictors of HF during index hospitalization for acute MI may allow early targeting of anti-remodeling therapies to this high-risk cohort.[13]

Clinical Perspective on p 702

Although prior work in randomized clinical trials[2] and registry data[4,11,14–16] have suggested several key clinical markers of HF risk, there is limited contemporary evidence on the distribution of in-hospital therapies and outcomes in patients with HF by MI type. Accordingly, we sought to evaluate the incidence of HF development in patients presenting with MI (either non-ST-elevation [NSTEMI] and ST-elevation MI [STEMI]), as well as describe differences in in-hospital therapies and correlates of incident HF. The National Cardiovascular Data Registry (NCDR) ACTION Registry-Get With The Guidelines (GWTG) provides a unique real-world cohort of well-characterized patients with acute MI, an ideal population in which to study the contemporary phenotype of

Received March 20, 2012; accepted September 24, 2012.

From the Cardiology Division, Department of Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA (R.V.S.); Duke Clinical Research Institute, Duke University School of Medicine, Durham, NC (D.H., M.A., T.Y.W.); Virginia Commonwealth University, Department of Cardiology, Richmond, VA (M.C.K.); TIMI, Brigham and Women's Hospital, Harvard Medical School, Boston, MA (S.D.W.); TIMI Study Group, Cardiovascular Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston MA (B.M.S.).

The online-only Data Supplement is available in this article at http://circheartfailure.ahajournals.org/lookup/suppl/doi:10.1161/CIRCHEARTFAILURE.112.968180/-/DC1.

Correspondence to Benjamin Scirica, MD MPH, 350 Longwood Avenue, 1st Floor, Boston, MA 02115. E-mail: bscirica@partners.org

Circ Heart Fail **is available at http://circheartfailure.ahajournals.org** **DOI: 10.1161/CIRCHEARTFAILURE.112.968180**

post-MI HF. Using data from ACTION Registry-GWTG, we identified NSTEMI and STEMI patients who developed signs or symptoms of HF during index hospitalization, compared in-hospital management (including revascularization), and modeled differences in patient characteristics associated with post-MI development of HF.

Methods

Study Population

The ACTION Registry-GWTG is a database containing records from >600 participating hospitals. The registry uses a standardized data set with written definitions, has requirements in place to ensure uniform data entry and transmission, and is subject to data quality checks. The general methodology of the registry has been previously reported.[17] For the purposes of establishing a contemporary cohort of patients with MI, we studied 267 605 patients admitted using long data collection form with NSTEMI or STEMI from January 1, 2007, to March 31, 2011. Given the interest in characterizing the development of HF during admission, we excluded patients with cardiogenic shock on presentation or during hospitalization (n=19 312, 7% of total cohort), missing left ventricular ejection fraction (LVEF) assessment during index hospitalization (n=28 412, 11%), HF on admission (n=31 665, 12%), and missing information regarding HF during admission (n=413, 0.2%). The final population included 187 803 patients (STEMI, n=77 675; NSTEMI, n=110 128; Table 1).

Data Collection and Study Definitions

Trained data collectors obtained abstract clinical and biochemical correlates and clinical outcomes from chart review according to standard definitions and data collection forms prescribed by the registry http://www.ncdr.com/WebNCDR/ACTION/Elements.aspx Collected data included demographics (age, sex, weight and height, race, insurance status), medical history (smoking, hypertension, dyslipidemia, dialysis, chronic lung disease, presence of diabetes mellitus, prior MI or revascularization, prior HF, peripheral arterial disease, stroke), signs and symptoms at presentation (electrocardiographic patterns, vital signs), and selected laboratory data. In addition, time to revascularization (surgical or percutaneous) as well as details of coronary angiography were collected. Medication use on admission, during first 24 hours, and at discharge, as well as details on in-hospital diagnostic and therapeutic cardiac procedures (eg, stress testing) and outcomes were also collected. HF during index hospitalization was defined as the documentation of clinical symptoms of HF (dyspnea on exertion or fluid retention) or signs of HF (rales, jugular venous distension, pulmonary edema) by a physician. In an internal but unpublished 2011 audit of NCDR, there was a 91% agreement between this NCDR definition of HF and a hospital chart audit, suggesting that the NCDR-adjudicated HF is a reasonable reflection of a clinical HF diagnosis. Preserved left ventricular (LV) function was defined as LVEF≥50% for the purposes of this analysis.

Statistical Analysis

Covariates were compared between HF and no HF in each group (STEMI, NSTEMI) using the Wilcoxon rank-sum test to compare continuous variables and the Pearson χ^2 test for categorical variables. Continuous variables were presented as medians with interquartile ranges, and categorical variables were expressed as frequencies and percentages. A P value <0.05 was considered significant. To evaluate the relationship between baseline risk factors and the development of in-hospital HF, logistic generalized estimating equations method with exchangeable working correlation matrix was used to account for within-hospital clustering as patients within hospitals tend to have more similar responses relative to patients at other hospitals. This method produces estimates similar to those from ordinary logistic regression, but variances are adjusted for the correlation of outcomes within a hospital.[18] Adjusted associations for outcomes were displayed as odds ratios (OR) (95% CI). The empirical standard errors were used in the calculation of the P values. LVEF, age, heart rate, systolic blood pressure, creatinine clearance (by the Cockcroft-Gault formula), baseline troponin concentration, prior HF, prior coronary artery bypass surgery (CABG), prior percutaneous coronary revascularization (PCI), prior MI, history of hypertension, history of diabetes mellitus, sex, MI type (STEMI/NSTEMI) at presentation, weight, race, home β-blocker, use, home angiotensin converting enzyme inhibitor or angiotensin receptor blockers, use and home aldosterone blocking agent use were included as covariates. Continuous variables that became nonlinear or flat in relationship to development of in-hospital HF were fit with linear splines or truncated, respectively. For all covariates included in the model, the percent of missing data was <3%. Missing variables were imputed to the most frequently occurring group for categorical variables and imputed to the median for continuous variables.[19] Missing weight and creatinine clearance were imputed to the sex-specific median of the nonmissing values. Given the exploratory and descriptive nature of this study, no adjustments were made for multiple testing. All analyses were performed using SAS software (version 9.2, SAS Institute, Cary, NC). All P values were 2-sided.

This research was supported by the American College of Cardiology Foundation's NCDR. The views expressed in this article represent those of the author(s), and do not necessarily represent the official views of the NCDR or its associated professional societies identified at www.ncdr.com.

Results

Correlates of HF Development in Patients With NSTEMI

The overall demographic and clinical characteristics of patients stratified by presence or absence of HF during index admission with NSTEMI are shown in Tables 1 and 2. Of the 110 128 patients with NSTEMI without HF on admission, 4348 (4%) developed post-MI HF during the index hospitalization. Patients who developed HF were generally older (median 74 versus 64 years, P<0.0001) with a higher female proportion. Patients who developed HF were more likely to have a history of HF, MI, hypertension, CABG surgery, and diabetes mellitus. There were statistically significant differences in admission vital signs (blood pressure and heart rate), and patients who developed HF were more likely to have ST depression on initial ECG. Notably, patients who developed HF had a markedly reduced renal function on admission, and as expected had a higher frequency of LV dysfunction. Interestingly, however, 35% of patients with HF during admission had a preserved LVEF, suggesting that normal LVEF during admission may not exclude the possibility of developing HF during admission for MI. Preadmission use of β-blockade and renin-angiotensin system inhibition was higher in patients who developed HF, likely representing more frequent prior cardiac disease or HF. Despite a high use of β-blocker, aspirin, and anticoagulant therapy during the first 24 hours of hospitalization, patients who developed HF were less likely to receive clopidogrel or statin therapy (Table 2).

Compared with patients who did not develop HF during admission, patients who developed HF were more likely to undergo medical management without any catheterization during admission (30% versus 13%, P<0.0001, Table 3), and had a higher incidence of contraindication to catheterization

Table 1. Baseline Characteristics of Patients Stratified by Development of HF During Index Hospitalization and Type of MI

	NSTEMI			STEMI		
Characteristic	No HF (n=105 780)	HF (n=4348)	*P*	No HF (n=74 862)	HF (n=2813)	*P*
Age, y, (median, IQR)	64 (54–75)	74 (63–82)	<0.0001	59 (51–69)	68 (57–79)	<0.0001
Female sex	37 740 (36%)	2014 (46%)	<0.0001	20 546 (27%)	1169 (42%)	<0.0001
Body mass index, kg/m^2	29.7±6.8	28.9±7.2	<0.0001	29.2±6.2	28.4±6.5	<0.0001
Race			0.0271			0.1466
White – no., %	88 071 (83%)	3639 (84%)		63 245 (84%)	2371 (84%)	
Black/African-American – no., %	10 629 (10%)	444 (10%)		6115 (8%)	233 (8%)	
Asian – no., %	1241 (1%)	60 (1%)		1091 (1%)	56 (2%)	
Hispanic – no., %	4131 (4%)	151 (3%)		3082 (4%)	103 (4%)	
Other – no., %	1041 (1%)	25 (1%)		858 (1%)	30 (1%)	
Insurance			<0.0001			<0.0001
Private insurance – no., %	61 671 (58%)	2325 (53%)		43 464 (58%)	1543 (55%)	
Medicare or Medicaid – no., %	31 552 (30%)	1725 (40%)		17 511 (23%)	906 (32%)	
Self or none – no., %	10 125 (10%)	222 (5%)		11 898 (16%)	311 (11%)	
Other and military – no., %	2293 (2%)	67 (2%)		1897 (2.5%)	51 (1.8%)	
Current dialysis – no., %	2151 (2%)	158 (4%)	<0.0001	493 (0.7%)	32 (1%)	0.0023
History of HF – no., %	8450 (8%)	1065 (24%)	<0.0001	2094 (3%)	253 (9%)	<0.0001
Hypertension – no., %	77 586 (73%)	3557 (82%)	<0.0001	45 862 (61%)	1992 (71%)	<0.0001
Dyslipidemia – no., %	65 430 (62%)	2763 (64%)	0.0243	38 839 (52%)	1494 (53%)	0.1995
Diabetes mellitus – no., %	32 432 (31%)	1828 (42%)	<0.0001	15 907 (21%)	829 (29%)	<0.0001
Prior MI – no., %	26 526 (25%)	1397 (32%)	<0.0001	13 513 (18%)	587 (21%)	0.0001
Prior PCI – no., %	25 625 (24%)	1103 (25%)	0.0859	14 388 (19%)	546 (19%)	0.8109
Prior CABG – no., %	16 940 (16%)	986 (23%)	<0.0001	4613 (6%)	270 (10%)	<0.0001
Prior atrial fibrillation or flutter – no., %	5262 (7%)	410 (13%)	<0.0001	1643 (3%)	155 (8%)	<0.0001
ECG findings – NSTEMI			<0.0001			
Presumed or new ST depressions – no., %	23 213 (22%)	1212 (28%)		N/A	N/A	
T wave inversions – no., %	15 521 (15%)	616 (14%)		N/A	N/A	
Transient ST-elevations – no., %	3181 (3%)	82 (2%)		N/A	N/A	
None – no., %	63 196 (60%)	2394 (55%)		N/A	N/A	
ECG findings – STEMI						<0.0001
ST-elevation – no., %	N/A	N/A		73 199 (98%)	2668 (95%)	
LBBB – no., %	N/A	N/A		1228 (2%)	128 (5%)	
Isolated posterior MI – no., %	N/A	N/A		364 (0.5%)	15 (0.5%)	
Vital signs						
Admission heart rate, bpm	80 (69–94)	89 (74–105)	<0.0001	77 (65–90)	83 (70–100)	<0.0001
Admission systolic blood pressure, mm Hg	147 (129–167)	141 (120–162)	<0.0001	142 (123–162)	136 (117–156)	<0.0001
Laboratories						
Estimated glomerular filtration rate (Cockcroft-Gault, nondialysis patients), ml/min	83.3 (57.2–113.0)	55.5 (36.6–82.9)	<0.0001	91.0 (67.8–117.4)	67.8 (46.5–95.4)	<0.0001
Hemoglobin, mg/dL	14.0 12.7–15.2)	12.8 (11.3–14.2)	<0.0001	14.6 (13.4–15.6)	13.8 (12.4–15.1)	<0.0001
Baseline troponin T or I ratio (×ULN)	2.0 (0.5–9.7)	3.2 (0.8–17.8)	<0.0001	1 (0.2–9)	2.5 (0.4–29.5)	<0.0001
Peak troponin T or I ratio (×ULN)	24.2 (6.3–96.7)	38.2 (9.5–148.4)	<0.0001	133.3 (32.0–504.7)	237.0 (55–847.5)	<0.0001
Home medications						
ACE inhibitor – no., %	31 147 (29%)	1479 (34%)	<0.0001	15 731 (21%)	704 (25%)	<0.0001
ARB – no., %	11 960 (11%)	588 (14%)	<0.0001	5905 (8%)	305 (11%)	<0.0001
Aldosterone inhibitor – no., %	1751 (2%)	149 (3%)	<0.0001	615 (0.8%)	55 (2%)	<0.0001
β-blocker – no., %	41 039 (39%)	2178 (50%)	<0.0001	18 766 (25%)	974 (35%)	<0.0001
Statin – no., %	43 015 (41%)	2052 (47%)	<0.0001	21 689 (29%)	920 (33%)	<0.0001

(Continued)

Table 1. Continued

Characteristic	NSTEMI			STEMI		
	No HF (n=105 780)	HF (n=4348)	*P*	No HF (n=74 862)	HF (n=2813)	*P*
LVEF≥50% – no., %	69 721 (66%)	1507 (35%)	<0.0001	40 929 (55%)	616 (22%)	<0.0001
LVEF 40–50% – no., %	20 792 (20%)	993 (23%)		20 113 (27%)	735 (26%)	
LVEF 25–40% – no., %	12 523 (12%)	1383 (32%)		12 116 (16%)	1145 (41%)	
LVEF<25% – no., %	2744 (3%)	465 (11%)		1704 (2%)	317 (11%)	

HF indicates heart failure; MI, myocardial infarction; STEMI, ST-elevation MI; NSTEMI, non-ST-elevation MI; IQR, interquartile range; PCI, percutaneous coronary revascularization; CABG, coronary artery bypass surgery; LBBB, left bundle branch block; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker; LVEF, left ventricular ejection fraction.

(24% versus 9%, $P<0.0001$). Among patients undergoing cardiac catheterization, those who developed HF after NSTEMI frequently had severe proximal left anterior descending artery stenosis (44% versus 31%), left main coronary disease (19% versus 9%), or 3-vessel disease (50% versus 32%, all $P<0.0001$). Although rates of PCI were lower and CABG surgery higher in patients who developed HF, the overall rate of revascularization was markedly lower in patients who developed HF (63% versus 48%, $P<0.0001$). Furthermore, there was a 3.5 hours additional delay to diagnostic angiography in patients who developed HF, and an additional 3.4 hours and 17.5 hours delay to PCI and CABG (both $P<0.0001$). Accordingly, the baseline and peak troponin values for patients with in-hospital HF were significantly higher than patients without HF. The corresponding in-hospital mortality was higher in patients with HF (6.7% versus 0.9%, $P<0.0001$), as was the length of stay. Optimal discharge interventions (aspirin, clopidogrel, renin-angiotensin system inhibition, statins, referrals to cardiac rehabilitation and smoking cessation) occurred in the majority of patients, though patients who developed HF were less likely to receive clopidogrel or statin therapy (Table 4). Of note, exclusion of patients with contraindications to catheterization did not affect these observations regarding treatments applied or correlates of HF. In general, similar relationships were observed when patients with a prior history of HF before admission were excluded.

Correlates of HF Development in Patients With STEMI

The overall demographic and clinical characteristics of patients stratified by presence or absence of HF during index admission with STEMI are shown in Tables 1 and 2. Of the 77 675 patients with STEMI included in this analysis, 2813 (3.6%) developed HF during admission. Similar to patients with NSTEMI, patients with STEMI developing in-hospital HF were more likely to be significantly older (median age 68 versus 59, $P<0.0001$), with nearly 36% of patients with HF >75 years. Patients who developed HF were more likely to have a history of HF, MI, CABG surgery, hypertension, and diabetes mellitus. There were statistically significant differences in admission vital signs in patients who subsequently developed HF as compared with patients who did not develop HF. Similar to patients with NSTEMI, STEMI patients who developed HF had a lower admission renal function and were more likely to be on anti-hypertensive therapy on admission. At 24 hours after admission, there was a slightly lower use of clopidogrel in STEMI patients who developed HF, but no other clinically significant differences in HF or MI pharmacotherapies (eg, aspirin, β-blockade, anticoagulant therapy). Specifically, early β-blocker use was not different between patients who did and did not develop in-hospital HF. As expected, patients who developed HF had a lower LVEF than patients without HF (Table 1), though (as observed with

Table 2. Medications During First 24 h of Hospitalization Stratified by Development of HF During Index Hospitalization

Medication During First 24 h	NSTEMI			STEMI		
	No HF (n=105 780)	HF (n=4348)	*P*	No HF (n=74 862)	HF (n=2813)	*P*
Aspirin – no., %	100 660 (98%)	3956 (96%)	<0.0001	73 089 (99%)	2730 (99%)	0.5288
Clopidogrel – no., %	59 502 (60%)	1951 (50%)	<0.0001	62 564 (86%)	2148 (81%)	<0.0001
Any anticoagulant (subcutaneous or IV)	96 669 (93%)	3802 (92%)	0.0004	71 139 (96%)	2685 (97%)	0.0101
GPIIb/IIIa inhibitor – no., %	39 300 (39%)	1136 (30%)	<0.0001	50 066 (69%)	1710 (64%)	<0.0001
ACE inhibitor – no., %	41 017 (42%)	1433 (41%)	0.0651	38 762 (57%)	1230 (54%)	0.0013
ARB – no., %	8446 (8%)	348 (9%)	<0.0001	3420 (5%)	141 (6%)	0.0562
Aldosterone inhibitor – no., %	1438 (1%)	142 (3%)	<0.0001	1169 (2%)	123 (5%)	<0.0001
β-blocker – no., %	86 354 (90%)	3415 (90%)	0.1514	64 093 (94%)	2319 (94%)	0.4731
Statin – no., %	63 981 (62%)	2377 (57%)	<0.0001	54 585 (75%)	1764 (65%)	<0.0001

HF indicates heart failure; NSTEMI, non-ST-elevation MI; STEMI, ST-elevation MI; GPIIb/IIIa, glycoprotein IIb/IIIa receptor blocker; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker.

Table 3. Management Strategy Stratified by Development of HF During Index Hospitalization

Characteristic	NSTEMI			STEMI		
	No HF (n=105 780)	HF (n=4348)	*P*	No HF (n=74 862)	HF (n=2813)	*P*
Management strategy: NSTEMI						
Noninvasive stress testing	5868 (6%)	306 (7%)	<0.0001	N/A	N/A	N/A
Intervention			<0.0001			
Medical management without any catheterization – no., %	13 302 (13%)	1318 (30%)		N/A	N/A	N/A
Medical management with diagnostic catheterization only – no., %	25 864 (24%)	923 (21%)		N/A	N/A	N/A
PCI – no., %	54 520 (52%)	1256 (29%)		N/A	N/A	N/A
CABG – no., %	11 990 (11%)	841 (19%)		N/A	N/A	N/A
Any revascularization – no., %	66 510 (63%)	2097 (48%)	<0.0001	N/A	N/A	N/A
Management strategy: STEMI						
Thrombolytic therapy	N/A	N/A	N/A	7536 (11%)	311 (14%)	0.0001
Primary PCI	N/A	N/A	N/A	56 654 (84%)	1807 (79%)	<0.0001
CABG	N/A	N/A	N/A	4257 (6%)	347 (12%)	<0.0001
No reperfusion therapy – no., %	N/A	N/A	N/A	3497 (5%)	173 (8%)	<0.0001
Any revascularization – no., %	N/A	N/A	N/A	67 416 (93%)	2351 (90%)	<0.0001
No. of diseased vessels			<0.0001			<0.0001
None – no., %	7492 (8%)	191 (6%)		1869 (3%)	48 (2%)	
One vessel disease – no., %	27 083 (29%)	500 (17%)		28 191 (39%)	762 (29%)	
Two vessel disease – no., %	26 874 (29%)	795 (26%)		23 639 (32%)	860 (33%)	
Three vessel disease – no %	29 756 (32%)	1496 (50%)		18 799 (26%)	944 (36%)	
Proximal LAD ≥70% – no., %	18 796 (31%)	839 (44%)	<0.0001	15 390 (33%)	909 (54%)	<0.0001
Left main coronary disease ≥ 50%	8226 (9%)	574 (19%)	<0.0001	3390 (5%)	256 (10%)	<0.0001
Time to procedure						
Hospital arrival to any diagnostic catheterization, median (IQR), h	20.0 (8.6–37.1)	23.5 (9.6–52.0)	<0.0001	N/A	N/A	
Hospital arrival to PCI	18.7 (6.6–33.6)	22.1 (6.8–58.6)	<0.0001	N/A	N/A	
Hospital arrival to CABG	72.0 (42.4–114.9)	89.5 (45.4–143.2)	<0.0001	N/A	N/A	
Median time from arrival to primary PCI, min (IQR)	N/A	N/A	<0.0001	63 (49–79)	67.5 (51–84)	<0.0001
In-hospital outcomes						
In-hospital mortality, no., %	958 (0.9%)	278 (6.7%)	<0.0001	803 (1.1%)	161 (5.8%)	<0.0001
Stroke, no., %	518 (0.5%)	97 (2.3%)	<0.0001	343 (0.5%)	54 (2%)	<0.0001
GWTG major bleeding, no., %	6373 (6%)	846 (20%)	<0.0001	5201 (7%)	609 (22%)	<0.0001
Length of stay, days (median, IQR)	3 (2–5)	7 (4–11)	<0.0001	3 (2–4)	6 (4–10)	<0.0001

HF indicates heart failure; NSTEMI, non-ST-elevation MI; STEMI, ST-elevation MI; NSTEMI, non-ST-elevation MI; PCI, percutaneous coronary revascularization; CABG, coronary artery bypass; IQR, interquartile range; LAD, left anterior descending artery; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker; GWTG, Get With The Guidelines.

NSTEMI), 22% of patients with in-hospital HF had a preserved LVEF.

Notably, as compared with patients who did not develop HF, patients who developed HF were less likely to receive primary PCI (79% versus 84%, *P*<0.0001, Table 3), and more likely to receive thrombolytic therapy (14% versus 11%, *P*<0.0001). A slightly higher proportion of patients who developed HF were considered to have a contraindication to catheterization (5% versus 2%, *P*<0.0001). For patients undergoing primary PCI after direct arrival to the ACTION-GWTG hospital, there was a statistically but not clinically significant difference in time from arrival to balloon inflation between patients who did and did not develop HF during admission (median 63 minute in no-HF versus 67.5 minute in patients with HF, *P*<0.0001). Accordingly, the baseline and peak troponin values for patients with in-hospital HF were significantly higher than patients without HF. Development of HF during index admission was associated with a higher risk of in-hospital mortality (5.8% versus 1.1% in patients without HF, *P*<0.0001) and longer length of stay. On discharge, optimal discharge interventions occurred in a similar proportion of HF and non-HF patients (Table 4). Excluding patients with contraindication to catheterization did not affect treatments applied or correlates of HF. In general, similar relationships

Table 4. Discharge Interventions in Patients Stratified by Development of HF During Index Hospitalization

	NSTEMI			STEMI		
Discharge Interventions, no.(%)	No HF (n=105 780)	HF (n=4348)	*P*	No HF (n=74 862)	HF (n=2813)	*P*
Cardiac rehabilitation referral	66 495 (74%)	2122 (72%)	0.0114	56 609 (83%)	1854 (84%)	0.3252
Smoking cessation	32 343 (97%)	889 (96%)	0.4146	32 522 (98%)	852 (98%)	0.3487
Aspirin	93 934 (98%)	3370 (97%)	0.0009	70 040 (99%)	2378 (98%)	0.3769
Clopidogrel	69 129 (74%)	2017 (61%)	<0.0001	60 246 (86%)	1923 (81%)	<0.0001
β-blocker	89 546 (95%)	3341 (96%)	0.02010	67 375 (98%)	2327 (97%)	0.8445
ACE inhibitor or ARB	65 069 (69%)	2387 (73%)	<0.0001	54 053 (79%)	1879 (83%)	<0.0001
Aldosterone blocking agent	2471 (3%)	360 (10%)	<0.0001	2030 (3%)	370 (15%)	<0.0001
Statins	87 080 (90%)	3043 (86%)	<0.0001	67 007 (95%)	2266 (93%)	0.0001

HF indicates heart failure; NSTEMI, non-ST-elevation MI; STEMI, ST-elevation MI; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker.

were observed when patients with a prior history of HF before admission were excluded.

Multivariable Regression Model

In an adjusted multivariable logistic regression model in the overall population for association with in-hospital HF (Table 5), LVEF ≤30% (OR=2.99, 95% CI=2.78–3.20; *P*<0.0001), age (per 5 years OR=1.03, 95% CI 1.02–1.03; *P*<0.0001), prior HF (OR=1.66, 95% CI=1.48–1.86; *P*<0.0001), history of diabetes mellitus (OR=1.23, 95% CI=1.16–1.29; *P*<0.0001), female sex (OR=1.25, 95% CI=1.18–1.32; *P*<0.0001), STEMI (OR=1.22, 95% CI=1.14–1.31; *P*<0.0001), and history of hypertension (OR=1.08, 95% CI=1.02–1.15; *P*=0.005) were independently associated with in-hospital HF for both types of MI combined. LVEF alone carried a C-statistic of 0.60, which improved to 0.76 for the overall model (Table 5), suggesting that though LVEF was the strongest contributor to the model, the full model was substantially more robust in the discrimination of risk. In addition, the C-statistic for LVEF, age, and sex alone was 0.73, suggesting significant contributions of age and sex to discrimination of risk. In addition, covariates associated with in-hospital development of HF were similar when

Table 5. Adjusted Multivariate Logistic Regression Analysis to Identify Predictors of In-Hospital HF Among Overall Population

	Adjusted				
Variable	OR	95% CI for OR (Lower)	95% CI for OR (Upper)	*P* (Individual)	*P* (Global)
LVEF ≤30% (vs LVEF >30%)	2.99	2.78	3.20	<0.0001	—
Age (per 5 y increase)	1.03	1.02	1.03	<0.0001	—
HR (per 10 bpm increase, HR <70 bpm)	0.98	0.93	1.03	0.4038	<0.0001
HR (per 10 bpm increase, 70–100 bpm)	1.21	1.18	1.25	<0.0001	
HR (per 10 bpm increase, HR <100 bpm)	1.04	1.02	1.06	0.0002	
Creatinine clearance (per 10 ml/min decrease, <100)	1.09	1.07	1.10	<0.0001	<0.0001
Creatinine clearance (per 10U increase, ≥100)	0.97	0.95	0.98	<0.0001	
SBP (per 10 mm Hg decrease, SBP <170 mm Hg)	1.06	1.05	1.07	<0.0001	<0.0001
SBP (per 10 mm Hg increase, SBP ≥170 mm Hg)	1.00	0.98	1.03	0.7211	
Baseline troponin ratio (per 1×ULN increase)	1.00	1.00	1.00	<0.0001	—
Prior CHF	1.66	1.48	1.86	<0.0001	—
Female (vs male)	1.25	1.18	1.32	<0.0001	—
Weight (per 10 kg decrease, weight <80 kg)	0.99	0.96	1.02	0.3854	<0.0001
Weight (per 10 kg increase, weight ≥80 kg)	1.08	1.06	1.10	<0.0001	
History of diabetes mellitus	1.23	1.16	1.29	<0.0001	—
STEMI	1.22	1.14	1.31	<0.0001	—
Prior PCI	0.90	0.85	0.94	<0.0001	—
β-blocker (on admission)	1.10	1.05	1.16	0.0001	—
History of hypertension	1.08	1.02	1.15	0.0054	—
ACE or ARB (on admission)	0.95	0.90	0.99	0.0203	—

HF indicates heart failure; OR, odds ratios; LVEF, left ventricular ejection fraction; HR, hazard ratios; SBP, systolic blood pressure; ULN, upper limit of normal; CHF, congestive heart failure; STEMI, ST-elevation MI; PCI, percutaneous coronary revascularization; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker.

Table 6. Adjusted Multivariate Logistic Regression Analysis to Identify Predictors of In-Hospital HF When Patients With a Prior History of HF Were Excluded From the Overall Population

	Adjusted				
Variable	OR	95% CI for OR (Lower)	95% CI for OR (Upper)	*P* (Individual)	*P* (Global)
LVEF ≤30% (vs LVEF >30%)	3.54	3.27	3.84	<0.0001	—
Age (per 5 y increase)	1.03	1.02	1.03	<0.0001	—
HR (per 10 bpm increase, HR <70 bpm)	1.00	0.95	1.06	0.8857	<0.0001
HR (per 10 bpm increase, 70–100 bpm)	1.21	1.17	1.25	<0.0001	
HR (per 10 bpm increase, HR >100 bpm)	1.05	1.02	1.07	0.0002	
Creatinine clearance (per 10 ml/min decrease, <100)	1.10	1.08	1.11	<0.0001	<0.0001
Creatinine clearance (per 10U increase, ≥100)	0.97	0.96	0.99	0.0037	
SBP (per 10 mm Hg decrease, SBP <170 mm Hg)	1.07	1.06	1.08	<0.0001	<0.0001
SBP (per 10 mm Hg increase, SBP ≥170 mm Hg)	1.01	0.98	1.04	0.5074	
Baseline troponin ratio (per 1×ULN increase)	1.00	1.00	1.00	<0.0001	—
Female (vs male)	1.26	1.19	1.33	<0.0001	—
History of diabetes mellitus	1.27	1.19	1.34	<0.0001	—
Weight (per 10 kg decrease, weight <80 kg)	0.97	0.94	1.01	0.1517	<0.0001
Weight (per 10 kg increase, weight ≥80 kg)	1.07	1.05	1.10	<0.0001	
STEMI	1.25	1.16	1.34	<0.0001	—
β-blocker (on admission)	1.13	1.06	1.19	<0.0001	—
Prior PCI	0.91	0.86	0.97	0.0020	—
History of hypertension	1.08	1.02	1.15	0.0101	—
ACE or ARB (on admission)	0.94	0.89	0.99	0.0224	—

HF indicates heart failure; OR, odds ratios; LVEF, left ventricular ejection fraction; HR, hazard ratios; SBP, systolic blood pressure; ULN, upper limit of normal; STEMI, ST-elevation MI; PCI, percutaneous coronary revascularization; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker.

patients with prior history of HF were excluded (Table 6). Relationships were similar after excluding patients (n=11 568) determined by the site to have an exclusion for catheterization (data not shown).

Discussion

In this large observational study, we found that 4% of STEMI and NSTEMI patients without HF or cardiogenic shock on admission developed new signs or symptoms of HF during their index hospitalization. Patients who developed HF during index hospitalization had similar characteristics regardless of type of MI, including older age, a higher frequency of prevalent cardiovascular risk factors, and prior cardiac events (including HF), worse renal function, modest differences in admission vital signs, and a lower LVEF. Importantly, we found that patients who developed HF in both NSTEMI and STEMI cohorts were more likely to have medical management without percutaneous or surgical revascularization. Moreover, in those who develop HF who undergo revascularization, there seems to be a greater delay to definitive revascularization in patients with NSTEMI, but not STEMI. In addition, a lower catheterization rate in patients with in-hospital HF was not accompanied by a clinically significant higher rate of noninvasive stress testing (7% versus 6%), usually used in high-risk patients (eg, renal dysfunction) for risk stratification. Ultimately, these differences in comorbid conditions and management may in part explain the markedly higher cardiac mortality rates and longer length of stay in patients with HF.

The identification of patients early in disease progression is of paramount importance because the development of HF in the post-MI setting portends a significant risk of poor in-hospital and long-term outcomes.[4,12] Specific therapies that minimize the risk of adverse LV remodeling and HF, such as renin-angiotensin aldosterone system antagonists or β-blockers,[20,21] may modulate these outcomes. Thus, targeting at-risk patients before clinical HF represents an opportunity for early intervention that may prevent HF onset and improve post-MI outcomes. In this regard, characterizing the phenotype of at-risk patients who eventually manifest clinically apparent HF is of major clinical interest.

Our study represents the most recent analysis of correlates of HF during hospitalization for acute MI, and some of our results are consistent with prior work.[4,11,15,22] In a landmark study of patients from the Global Registry of Acute Coronary Events (GRACE) Registry, Steg et al[4] identified 1778 patients (13% of the overall population) with HF on admission for acute coronary syndrome between 1999 and 2001, and found a lower use of percutaneous revascularization, optimal medical therapy, and higher in-hospital and 6 month postdischarge mortality. Most relevant to our analyses, patients who developed HF during the index hospitalization (869 patients in their overall cohort) had a higher in-hospital mortality rate (18%) as compared with patients with HF on presentation

(12%) and a longer length of hospital stay. In comparison, the in-hospital mortality rate for the 31 665 patients with HF on presentation in our study was 5.01%. Patients who developed in-hospital HF in GRACE were less likely to have a prior history of MI and more likely to receive revascularization therapy as compared with patients with HF on admission, suggesting that features other than delayed therapy (eg, higher clinical risk) may mediate the worse outcomes seen for in-hospital HF in this cohort as compared with ACTION. Indeed, nearly 19% of patients with HF on admission and 32% of patients with in-hospital HF required inotropes, suggesting a more advanced, decompensated HF phenotype.

In an analysis from the National Registry of Myocardial Infarction-2 (NRMI-2; 606 500 patients), between 1994 and 2000,[15] patients with HF on admission were less likely to undergo percutaneous revascularization or be administered optimal medical therapy (only 21% on β-blockade); HF on admission was associated with a nearly 70% increased risk of in-hospital mortality. However, as the comparison between patients who did or did not develop HF during hospitalization was not presented, our data suggesting a more adverse risk profile and more infrequent revascularization in a more clinically stable population with in-hospital HF extend these observations. Moreover, in a study of 2347 patients with HF or LV systolic dysfunction on admission or during hospitalization after STEMI,[22] Velasquez et al demonstrated that patients with HF were less likely to have percutaneous revascularization (31%) or angiography (55%), β-blockade therapy (58%), and more likely to require mechanical support (7% with intra-aortic balloon pump), associated with a 4-fold higher risk of in-hospital death (13% versus 2% without HF). Relative to these studies, patients with in-hospital HF in ACTION had a higher rate of revascularization and use of optimal medical therapy (above 90% β-blocker use regardless of MI type), possibly accounting for the lower observed in-hospital mortality. In addition, our relatively low rate of occurrence of in-hospital HF in comparison with prior studies (up to 37% in-hospital HF in an older Medicare analysis) may also be referable to higher use of medical therapy and revascularization in our contemporary cohort (only 50% β-blocker use and 17% revascularization in the Medicare data).

Our analysis significantly extends the work from prior analyses from NRMI, VALsartan In Acute myocardial iNfarcTion (VALIANT), and GRACE to a more contemporary population, with more extensive phenotyping of angiographic results and delay times to angiography or CABG surgery, both of which were not explicitly analyzed in these prior reports. A clear and consistent finding from our study and these prior results is that patients who develop HF during index admission comprise a high-risk cohort, who may benefit from early and aggressive care to improve outcome. Although we did not examine long-term outcome in this study, the finding that patients with HF during hospitalization had a more advanced clinical profile, worse renal function, and more severe, multivessel coronary artery disease with reduced LV systolic function—all markers of poorer prognosis in post-MI HF—is striking. Moreover, even with continued guideline support of directed earlier revascularization strategies for NSTEMI complicated by HF, our work demonstrates that these patients have a decreased rate of referral for diagnostic and therapeutic percutaneous and surgical revascularization, and when referred, a longer delay to any definitive procedure. In turn, these delays may play a role in reinforcing the poorer outcomes in this extremely high-risk population.

The limitations of our work must be viewed with the context of the study design and ACTION Registry data collection process. Although this analysis is retrospective, the implication that patients with HF represent a distinct cohort potentially subject to different management patterns in contemporary practice is consistent with prior work. Although the factors we observed more frequently in patients with in-hospital HF cannot be interpreted as causative of HF, lower rates of revascularization in patients with HF are a physiologically plausible explanation to our observations. In addition, the diagnosis of HF in this population was determined by treating physicians according to predefined criteria and therefore susceptible to differential interpretation, though this would tend to bias toward a null association. Although we excluded and therefore cannot characterize patients with cardiogenic shock in this study, our results are likely generalizable to a larger population of patients with clinically and prognostically relevant degrees of HF. We did not directly assess the impact of excluding patients without an LVEF assessment on our results. Although it is possible that systematic differences in care between patients with and without LVEF assessment may influence in-hospital development of HF, only 11% of our cohort did not have an LVEF assessed. However, when excluding patients with a prior history of HF clinically, the associations we observed in the overall cohort remained significant (Table 6). Finally, the timing of HF occurrence during hospitalization cannot be assessed with the collected data from the ACTION Registry.

An additional limitation of our work is the inability to clarify the specific contraindications for catheterization in this cohort, though poorer renal function, age, and ongoing HF are likely factors included in these decisions. Unfortunately, myocardial salvage during acute MI becomes even more significant in older patients with significant comorbidities. Furthermore, with contrast-induced nephropathy of significant concern, outcomes during acute MI in patients with HF and renal dysfunction remains poor[23] and PCI use is lower in comparison to patients without renal dysfunction.[24] However, our results suggest that patients with MI and HF are more likely to have more severe coronary disease and more impaired LV function, suggesting that low-contrast diagnostic angiography may facilitate early identification and revascularization of this especially high-risk subgroup. Moreover, with an increasing focus on renal protection,[25] renal dysfunction may not necessarily be a strict contraindication to angiography and PCI in this population. In addition, B-type natriuretic peptide was not available in a significant proportion of patients, and was therefore not included in our models.

Because of the enrollment in ACTION only during acute MI, we cannot with clarity exclude patients with prior LV dysfunction or prior HF with LV dysfunction (a potentially high-risk group even prior to acute MI). Although confirmatory to prior work in large registries, the emergence of prior HF as an

independent predictor of HF during hospitalization during MI suggests closer surveillance of this population, regardless of LV ejection fraction.

In conclusion, in a large cohort of patients enrolled in a contemporary setting, we found that patients who develop HF during index hospitalization for acute MI had a more adverse clinical and biochemical profile. Despite these clear indications of higher cardiovascular risk during MI, we found decreased use of revascularization and medical therapies in these patients, with a correspondingly higher in-hospital mortality. Despite increased focus on early revascularization in acute MI, patients with in-hospital development of HF represent an ongoing subgroup with poorer outcomes, and should be specifically considered for prompt coronary reperfusion and optimal medical therapies before discharge.

Disclosures

R.V.S. is supported by an American Heart Association Post-Doctoral Fellowship Award (11POST000002) and a training grant from the Heart Failure National Institutes for Health - National Heart, Lung and Blood Institute Clinical Research Network (U01-HL084877). T.Y.W reports research grants to the Duke Clinical Research Institute from Bristol-Myers Squibb/Sanofi Aventis Partnership; Schering Plough/ Merck & Co., Inc, The Medicines Co, Heartscape Technologies, Inc., Canyon Pharmaceuticals, and Eli Lilly/Daiichi Sankyo Alliance, as well as consulting or honoraria from Medco Health Solutions, Inc., Astra Zeneca, and the American College of Cardiology Foundation (all conflicts of interest are listed at www.dcri.org.). B.M.S. reports research grants from Merck, AstraZeneca, Johnson and Johnson, Bayer Healthcare, Daiichi Sankyo, Bristol-Myers Squibb, Gilead Sciences, Novartis; Consultant: Gilead Sciences, Arena Pharmaceuticals, Lexicon. M.C.K reports speaker's bureau and consultancy from Astellas and AstraZeneca. S.D.W reports research grants from AstraZeneca, Daiichi Sankyo, Eli Lilly, Merck, and consultancy from AstraZeneca, Bayer, Arena, Ortho McNeil, Johnson and Johnson, Norvartis, Merck Schering Plough. R.V.S., M.A., and D.H. have no disclosures to report. The authors would like to acknowledge the support of the Duke Clinical Research Institute and ACTION Registry-Get With The Guidelines for statistical support and critical review of this article. ACTION Registry-GWTG is an initiative of the American College of Cardiology Foundation and the American Heart Association with partnering support from the Society of Chest Pain Centers, the American College of Emergency Physicians, and the Society of Hospital Medicine. The registry is funded in part by an independent grant from Merck and by Bristol-Myers Squibb.

References

1. Hellermann JP, Goraya TY, Jacobsen SJ, Weston SA, Reeder GS, Gersh BJ, Redfield MM, Rodeheffer RJ, Yawn BP, Roger VL. Incidence of heart failure after myocardial infarction: is it changing over time? *Am J Epidemiol*. 2003;157:1101–1107.
2. Lewis EF, Velazquez EJ, Solomon SD, Hellkamp AS, McMurray JJ, Mathias J, Rouleau JL, Maggioni AP, Swedberg K, Kober L, White H, Dalby AJ, Francis GS, Zannad F, Califf RM, Pfeffer MA. Predictors of the first heart failure hospitalization in patients who are stable survivors of myocardial infarction complicated by pulmonary congestion and/or left ventricular dysfunction: a VALIANT study. *Eur Heart J*. 2008;29:748–756.
3. Anavekar NS, Anavekar NS. Clinical modifiers for heart failure following myocardial infarction. *Curr Heart Fail Rep*. 2005;2:165–173.
4. Steg PG, Dabbous OH, Feldman LJ, Cohen-Solal A, Aumont MC, López-Sendón J, Budaj A, Goldberg RJ, Klein W, Anderson FA Jr; Global Registry of Acute Coronary Events Investigators. Determinants and prognostic impact of heart failure complicating acute coronary syndromes: observations from the Global Registry of Acute Coronary Events (GRACE). *Circulation*. 2004;109:494–499.
5. Najafi F, Dobson AJ, Hobbs M, Jamrozik K. Temporal trends in the frequency and longer-term outcome of heart failure complicating myocardial infarction. *Eur J Heart Fail*. 2007;9:879–885.
6. Ezekowitz JA, Kaul P, Bakal JA, Armstrong PW, Welsh RC, McAlister FA. Declining in-hospital mortality and increasing heart failure incidence in elderly patients with first myocardial infarction. *J Am Coll Cardiol*. 2009;53:13–20.
7. Hasdai D, Topol EJ, Kilaru R, Battler A, Harrington RA, Vahanian A, Ohman EM, Granger CB, Van de Werf F, Simoons ML, O'connor CM, Holmes DR Jr. Frequency, patient characteristics, and outcomes of mild-to-moderate heart failure complicating ST-segment elevation acute myocardial infarction: lessons from 4 international fibrinolytic therapy trials. *Am Heart J*. 2003;145:73–79.
8. Emanuelsson H, Karlson BW, Herlitz J. Characteristics and prognosis of patients with acute myocardial infarction in relation to occurrence of congestive heart failure. *Eur Heart J*. 1994;15:761–768.
9. Ali AS, Rybicki BA, Alam M, Wulbrecht N, Richer-Cornish K, Khaja F, Sabbah HN, Goldstein S. Clinical predictors of heart failure in patients with first acute myocardial infarction. *Am Heart J*. 1999;138(6 Pt 1):1133–1139.
10. Yeh RW, Sidney S, Chandra M, Sorel M, Selby JV, Go AS. Population trends in the incidence and outcomes of acute myocardial infarction. *N Engl J Med*. 2010;362:2155–2165.
11. Bennett KM, Hernandez AF, Chen AY, Mulgund J, Newby LK, Rumsfeld JS, Hochman JS, Hoekstra JW, Ohman EM, Gibler WB, Roe MT, Peterson ED. Heart failure with preserved left ventricular systolic function among patients with non-ST-segment elevation acute coronary syndromes. *Am J Cardiol*. 2007;99:1351–1356.
12. Lacey L, Tabberer M. Economic burden of post-acute myocardial infarction heart failure in the United Kingdom. *Eur J Heart Fail*. 2005;7:677–683.
13. Thomas KL, Velazquez EJ. Therapies to prevent heart failure post-myocardial infarction. *Curr Heart Fail Rep*. 2005;2:174–182.
14. Steg PG, Goldberg RJ, Gore JM, Fox KA, Eagle KA, Flather MD, Sadiq I, Kasper R, Rushton-Mellor SK, Anderson FA; GRACE Investigators. Baseline characteristics, management practices, and in-hospital outcomes of patients hospitalized with acute coronary syndromes in the Global Registry of Acute Coronary Events (GRACE). *Am J Cardiol*. 2002;90:358–363.
15. Spencer FA, Meyer TE, Gore JM, Goldberg RJ. Heterogeneity in the management and outcomes of patients with acute myocardial infarction complicated by heart failure: the National Registry of Myocardial Infarction. *Circulation*. 2002;105:2605–2610.
16. Wu AH, Parsons L, Every NR, Bates ER; Second National Registry of Myocardial Infarction. Hospital outcomes in patients presenting with congestive heart failure complicating acute myocardial infarction: a report from the Second National Registry of Myocardial Infarction (NRMI-2). *J Am Coll Cardiol*. 2002;40:1389–1394.
17. Peterson ED, Roe MT, Rumsfeld JS, Shaw RE, Brindis RG, Fonarow GC, Cannon CP. A call to ACTION (acute coronary treatment and intervention outcomes network): a national effort to promote timely clinical feedback and support continuous quality improvement for acute myocardial infarction. *Circ Cardiovasc Qual Outcomes*. 2009;2:491–499.
18. Zeger SL, Liang KY. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics*. 1986;42:121–130.
19. Harrell Jr, F, Regression Modeling Strategies: With Applications to Linear Models, Logistic Regression, and Survival Analysis2001, New York: Springer-Verlag.
20. Solomon SD, Skali H, Anavekar NS, Bourgoun M, Barvik S, Ghali JK, Warnica JW, Khrakovskaya M, Arnold JM, Schwartz Y, Velazquez EJ, Califf RM, McMurray JV, Pfeffer MA. Changes in ventricular size and function in patients treated with valsartan, captopril, or both after myocardial infarction. *Circulation*. 2005;111:3411–3419.
21. Solomon SD, Shin SH, Shah A, Skali H, Desai A, Kober L, Maggioni AP, Rouleau JL, Kelly RY, Hester A, McMurray JJ, Pfeffer MA; Aliskiren Study in Post-MI Patients to Reduce Remodeling (ASPIRE) Investigators. Effect of the direct renin inhibitor aliskiren on left ventricular remodelling following myocardial infarction with systolic dysfunction. *Eur Heart J*. 2011;32:1227–1234.
22. Velazquez EJ, Francis GS, Armstrong PW, Aylward PE, Diaz R, O'Connor CM, White HD, Henis M, Rittenhouse LM, Kilaru R, van Gilst W, Ertl G, Maggioni AP, Spac J, Weaver WD, Rouleau JL, McMurray JJ, Pfeffer MA, Califf RM; VALIANT registry. An international perspective on heart failure and left ventricular systolic dysfunction complicating myocardial infarction: the VALIANT registry. *Eur Heart J*. 2004;25:1911–1919.

23. Hanna EB, Chen AY, Roe MT, Wiviott SD, Fox CS, Saucedo JF. Characteristics and in-hospital outcomes of patients with non-ST-segment elevation myocardial infarction and chronic kidney disease undergoing percutaneous coronary intervention. *JACC Cardiovasc Interv*. 2011;4:1002–1008.
24. Fox CS, Muntner P, Chen AY, Alexander KP, Roe MT, Cannon CP, Saucedo JF, Kontos MC, Wiviott SD; Acute Coronary Treatment and Intervention Outcomes Network registry. Use of evidence-based therapies in short-term outcomes of ST-segment elevation myocardial infarction and non-ST-segment elevation myocardial infarction in patients with chronic kidney disease: a report from the National Cardiovascular Data Acute Coronary Treatment and Intervention Outcomes Network registry. *Circulation*. 2010;121:357–365.
25. Hogan S, Gurm HS. Preventing contrast-induced nephropathy in patients undergoing primary PCI. *J Invasive Cardiol*. 2011;23:451–452.

CLINICAL PERSPECTIVE

The identification of patients at risk for development of HF postmyocardial infarction (MI) is critical, given a significant risk of associated poor long-term outcome and progression to chronic HF. Therefore, targeting at-risk patients before clinical HF represents an opportunity for early intervention that may prevent HF onset and improve post-MI outcomes. In this regard, characterizing the phenotype of at-risk patients who eventually manifest clinically apparent HF is of major clinical interest. However, data on the impact of contemporary, real-world practice on incidence of in-hospital development of HF and predictors of HF development are lacking. In this context, Shah et al report results from the National Cardiovascular Data Registry-ACTION Registry from 77 675 patients with ST-elevation and 110 128 with non-ST-elevation MI patients without cardiogenic shock or HF at presentation treated between 2007 and 2011, demonstrating a nearly 4% incidence of HF development during index hospitalization, far lower than previous studies. Patients who developed HF during non-ST-elevation were more likely to be medically managed without catheterization or had longer delays to surgical or percutaneous revascularization. Patients with ST-elevation and HF were less likely to receive primary percutaneous coronary revascularization and more likely to receive thrombolytic therapy. These results suggest that patients with MI who develop HF during hospitalization have a higher risk clinical profile and greater mortality, but may be less likely to receive revascularization in a timely fashion, and targeting these highest risk patients may improve outcome post-MI.

Risk of Heart Failure Complication During Hospitalization for Acute Myocardial Infarction in a Contemporary Population: Insights From the National Cardiovascular Data ACTION Registry

Ravi V. Shah, DaJuanicia Holmes, Monique Anderson, Tracy Y. Wang, Michael C. Kontos, Stephen D. Wiviott and Benjamin M. Scirica

Circ Heart Fail. 2012;5:693-702; originally published online October 9, 2012;
doi: 10.1161/CIRCHEARTFAILURE.112.968180
Circulation: Heart Failure is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 1941-3289. Online ISSN: 1941-3297