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

Insights From the National Cardiovascular Data ACTION Registry

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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.\(^1,10\) 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,\(^5,11\) 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.\(^1,11\)

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
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. 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=19 512, 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, pulmonary edema) (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, pulmonary edema) 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 \( \chi^2 \) 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 with-in-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. \( \chi^2 \) 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 \( \beta \)-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. \( \chi^2 \) 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.

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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 \( \beta \)-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 \( \beta \)-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>
<thead>
<tr>
<th>Characteristic</th>
<th>NSTEMI</th>
<th>STEMI</th>
<th>P</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, (median, IQR)</td>
<td>64 (54–75)</td>
<td>74 (63–82)</td>
<td>&lt;0.0001</td>
<td>59 (51–69)</td>
</tr>
<tr>
<td>Female sex</td>
<td>37,740 (36%)</td>
<td>2014 (46%)</td>
<td>&lt;0.0001</td>
<td>20,546 (27%)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>29.7±6.8</td>
<td>28.9±7.2</td>
<td>&lt;0.0001</td>
<td>29.2±6.2</td>
</tr>
<tr>
<td>Race</td>
<td>88,071 (83%)</td>
<td>3639 (84%)</td>
<td>63,245 (84%)</td>
<td>2371 (84%)</td>
</tr>
<tr>
<td>White – no., %</td>
<td>10,629 (10%)</td>
<td>444 (10%)</td>
<td>6115 (8%)</td>
<td>233 (8%)</td>
</tr>
<tr>
<td>Black/African-American – no., %</td>
<td>1241 (4%)</td>
<td>151 (3%)</td>
<td>3082 (4%)</td>
<td>103 (4%)</td>
</tr>
<tr>
<td>Asian – no., %</td>
<td>4131 (1%)</td>
<td>60 (1%)</td>
<td>56 (2%)</td>
<td></td>
</tr>
<tr>
<td>Hispanic – no., %</td>
<td>10,141 (1%)</td>
<td>25 (1%)</td>
<td>30 (1%)</td>
<td></td>
</tr>
<tr>
<td>Other – no., %</td>
<td>61,671 (58%)</td>
<td>2325 (53%)</td>
<td>43,464 (58%)</td>
<td>1543 (55%)</td>
</tr>
<tr>
<td>History of HF – no., %</td>
<td>2,323 (22%)</td>
<td>1828 (42%)</td>
<td>15,907 (21%)</td>
<td>829 (29%)</td>
</tr>
<tr>
<td>Hypertension – no., %</td>
<td>4,5586 (73%)</td>
<td>3557 (82%)</td>
<td>45,862 (61%)</td>
<td>1992 (71%)</td>
</tr>
<tr>
<td>Dyslipidemia – no., %</td>
<td>65,430 (62%)</td>
<td>2763 (64%)</td>
<td>38,839 (52%)</td>
<td>1494 (53%)</td>
</tr>
<tr>
<td>Diabetes mellitus – no., %</td>
<td>32,432 (31%)</td>
<td>1828 (42%)</td>
<td>15,907 (21%)</td>
<td>829 (29%)</td>
</tr>
<tr>
<td>Prior MI – no., %</td>
<td>26,526 (25%)</td>
<td>1397 (32%)</td>
<td>13,513 (18%)</td>
<td>587 (21%)</td>
</tr>
<tr>
<td>Prior PCI – no., %</td>
<td>25,625 (24%)</td>
<td>1103 (25%)</td>
<td>14,388 (19%)</td>
<td>546 (19%)</td>
</tr>
<tr>
<td>Prior CABG – no., %</td>
<td>16,940 (16%)</td>
<td>986 (23%)</td>
<td>4613 (6%)</td>
<td>270 (10%)</td>
</tr>
<tr>
<td>Prior atrial fibrillation or flutter – no., %</td>
<td>5262 (7%)</td>
<td>410 (13%)</td>
<td>1643 (3%)</td>
<td>155 (8%)</td>
</tr>
<tr>
<td>ECG findings – NSTEMI</td>
<td>&lt;0.0001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presumed or new ST depressions – no., %</td>
<td>23,213 (22%)</td>
<td>1212 (28%)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>T wave inversions – no., %</td>
<td>15,521 (15%)</td>
<td>616 (14%)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Transient ST-elevations – no., %</td>
<td>3181 (3%)</td>
<td>82 (2%)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>None – no., %</td>
<td>63,196 (60%)</td>
<td>2394 (55%)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>ECG findings – STEMI</td>
<td>&lt;0.0001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST-elevation – no., %</td>
<td>N/A</td>
<td>73,199 (98%)</td>
<td>2668 (95%)</td>
<td></td>
</tr>
<tr>
<td>LBBB – no., %</td>
<td>N/A</td>
<td>1228 (2%)</td>
<td>128 (5%)</td>
<td></td>
</tr>
<tr>
<td>Isolated posterior MI – no., %</td>
<td>N/A</td>
<td>364 (0.5%)</td>
<td>15 (0.5%)</td>
<td></td>
</tr>
<tr>
<td>Vital signs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission heart rate, bpm</td>
<td>80 (69–94)</td>
<td>89 (74–105)</td>
<td>&lt;0.0001</td>
<td>77 (65–90)</td>
</tr>
<tr>
<td>Admission systolic blood pressure, mm Hg</td>
<td>147 (129–167)</td>
<td>141 (120–162)</td>
<td>&lt;0.0001</td>
<td>142 (123–162)</td>
</tr>
<tr>
<td>Laboratories</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Estimated glomerular filtration rate (Cockcroft-Gault, nondialysis patients), ml/min</td>
<td>83.3 (57.2–113.0)</td>
<td>55.5 (36.6–82.9)</td>
<td>&lt;0.0001</td>
<td>91.0 (67.8–117.4)</td>
</tr>
<tr>
<td>Hemoglobin, mg/dL</td>
<td>14.0 (12.7–15.2)</td>
<td>12.8 (11.3–14.2)</td>
<td>&lt;0.0001</td>
<td>14.6 (13.4–15.6)</td>
</tr>
<tr>
<td>Baseline troponin T or I ratio (×ULN)</td>
<td>2.0 (0.5–9.7)</td>
<td>3.2 (0.8–17.8)</td>
<td>&lt;0.0001</td>
<td>1 (0.2–9)</td>
</tr>
<tr>
<td>Peak troponin T or I ratio (×ULN)</td>
<td>24.2 (6.3–96.7)</td>
<td>38.2 (9.5–148.4)</td>
<td>&lt;0.0001</td>
<td>133.3 (32.0–504.7)</td>
</tr>
<tr>
<td>Home medications</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE inhibitor – no., %</td>
<td>31,147 (29%)</td>
<td>1479 (34%)</td>
<td>&lt;0.0001</td>
<td>15,731 (21%)</td>
</tr>
<tr>
<td>ARB – no., %</td>
<td>11,960 (11%)</td>
<td>588 (14%)</td>
<td>&lt;0.0001</td>
<td>5905 (8%)</td>
</tr>
<tr>
<td>Aldosterone inhibitor – no., %</td>
<td>1751 (2%)</td>
<td>149 (3%)</td>
<td>&lt;0.0001</td>
<td>615 (0.8%)</td>
</tr>
<tr>
<td>β-blocker – no., %</td>
<td>41,039 (39%)</td>
<td>2178 (50%)</td>
<td>&lt;0.0001</td>
<td>18,766 (25%)</td>
</tr>
<tr>
<td>Statin – no., %</td>
<td>43,015 (41%)</td>
<td>2052 (47%)</td>
<td>&lt;0.0001</td>
<td>21,689 (29%)</td>
</tr>
</tbody>
</table>

(Continued)
(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 1. Continued

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No HF (n=4348)</th>
<th>HF (n=7482)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF≥50% – no., %</td>
<td>69721 (66%)</td>
<td>1507 (35%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF 40–50% – no., %</td>
<td>20792 (20%)</td>
<td>993 (23%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF 25–40% – no., %</td>
<td>12523 (12%)</td>
<td>1383 (32%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF&lt;25% – no., %</td>
<td>2744 (3%)</td>
<td>465 (11%)</td>
<td>0.0004</td>
</tr>
</tbody>
</table>

HF indicates heart failure; MI, myocardial infarction; STEMI, ST-elevation MI; NSTEMI, non-ST-elevation MI; LVEF, left ventricular ejection fraction.

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

<table>
<thead>
<tr>
<th>Medication During First 24 h</th>
<th>No HF (n=4348)</th>
<th>HF (n=7482)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin – no., %</td>
<td>100660 (98%)</td>
<td>3956 (96%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Clopidogrel – no., %</td>
<td>59502 (60%)</td>
<td>1951 (50%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Any anticoagulant (subcutaneous or IV)</td>
<td>96669 (93%)</td>
<td>3802 (92%)</td>
<td>0.0004</td>
</tr>
<tr>
<td>GPIIb/IIIa inhibitor – no., %</td>
<td>39300 (39%)</td>
<td>1136 (30%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ACE inhibitor – no., %</td>
<td>41017 (42%)</td>
<td>1433 (41%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ARB – no., %</td>
<td>8446 (8%)</td>
<td>348 (9%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Aldosterone inhibitor – no., %</td>
<td>1438 (11%)</td>
<td>142 (3%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker – no., %</td>
<td>86354 (90%)</td>
<td>3415 (80%)</td>
<td>0.1514</td>
</tr>
<tr>
<td>Statin – no., %</td>
<td>63981 (62%)</td>
<td>2377 (57%)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

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.

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

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Management strategy: NSTEMI</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noninvasive stress testing</td>
<td>5868 (6%)</td>
<td>306 (7%)</td>
</tr>
<tr>
<td>Intervention</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical management without any catheterization – no., %</td>
<td>13 302 (13%)</td>
<td>1318 (30%)</td>
</tr>
<tr>
<td>Medical management with diagnostic catheterization only – no., %</td>
<td>25 864 (24%)</td>
<td>923 (21%)</td>
</tr>
<tr>
<td>PCI – no., %</td>
<td>54 520 (52%)</td>
<td>1256 (29%)</td>
</tr>
<tr>
<td>CABG – no., %</td>
<td>11 990 (11%)</td>
<td>841 (19%)</td>
</tr>
<tr>
<td>Any revascularization – no., %</td>
<td>66 510 (63%)</td>
<td>2097 (48%)</td>
</tr>
<tr>
<td><strong>Management strategy: STEMI</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>CABG</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>No reperfusion therapy – no., %</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Any revascularization – no., %</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td><strong>No. of diseased vessels</strong></td>
<td></td>
<td>N/A</td>
</tr>
<tr>
<td>None – no., %</td>
<td>7492 (8%)</td>
<td>191 (6%)</td>
</tr>
<tr>
<td>One vessel disease – no., %</td>
<td>27 083 (29%)</td>
<td>500 (17%)</td>
</tr>
<tr>
<td>Two vessel disease – no., %</td>
<td>26 874 (29%)</td>
<td>795 (26%)</td>
</tr>
<tr>
<td>Three vessel disease – no, %</td>
<td>29 756 (32%)</td>
<td>1496 (50%)</td>
</tr>
<tr>
<td>Proximal LAD ≥70% – no, %</td>
<td>18 796 (31%)</td>
<td>839 (44%)</td>
</tr>
<tr>
<td>Left main coronary disease ≥50%</td>
<td>8226 (9%)</td>
<td>574 (19%)</td>
</tr>
<tr>
<td><strong>Time to procedure</strong></td>
<td></td>
<td>N/A</td>
</tr>
<tr>
<td>Hospital arrival to any diagnostic catheterization, median (IQR), h</td>
<td>20.0 (6.6–37.1)</td>
<td>23.5 (9.6–52.0)</td>
</tr>
<tr>
<td>Hospital arrival to PCI</td>
<td>18.7 (6.6–33.6)</td>
<td>22.1 (6.8–58.6)</td>
</tr>
<tr>
<td>Hospital arrival to CABG</td>
<td>72.0 (42.4–114.9)</td>
<td>89.5 (45.4–143.2)</td>
</tr>
<tr>
<td><strong>Median time from arrival to primary PCI, min (IQR)</strong></td>
<td>N/A</td>
<td>63 (49–79)</td>
</tr>
<tr>
<td><strong>In-hospital outcomes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-hospital mortality, no., %</td>
<td>958 (0.9%)</td>
<td>278 (6.7%)</td>
</tr>
<tr>
<td>Stroke, no., %</td>
<td>518 (0.5%)</td>
<td>97 (2.3%)</td>
</tr>
<tr>
<td>GWGTG major bleeding, no., %</td>
<td>6373 (6%)</td>
<td>846 (20%)</td>
</tr>
<tr>
<td>Length of stay, days (median, IQR)</td>
<td>3 (2–5)</td>
<td>7 (4–11)</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Discharge Interventions, no. (%)</th>
<th>NSTEMI No HF (n=105 780)</th>
<th>NSTEMI HF (n=4348)</th>
<th>( P )</th>
<th>STEMI No HF (n=74 862)</th>
<th>STEMI HF (n=2813)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac rehabilitation referral</td>
<td>66 495 (74%)</td>
<td>2122 (72%)</td>
<td>0.0114</td>
<td>56 609 (83%)</td>
<td>1854 (84%)</td>
<td>0.3252</td>
</tr>
<tr>
<td>Smoking cessation</td>
<td>32 343 (97%)</td>
<td>889 (96%)</td>
<td>0.4146</td>
<td>32 522 (88%)</td>
<td>852 (88%)</td>
<td>0.3487</td>
</tr>
<tr>
<td>Aspirin</td>
<td>93 934 (98%)</td>
<td>3370 (97%)</td>
<td>0.0009</td>
<td>70 040 (99%)</td>
<td>2378 (98%)</td>
<td>0.3769</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>69 129 (74%)</td>
<td>2017 (61%)</td>
<td>&lt;0.0001</td>
<td>60 246 (86%)</td>
<td>1923 (81%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker</td>
<td>89 546 (95%)</td>
<td>3341 (96%)</td>
<td>0.02010</td>
<td>67 375 (98%)</td>
<td>2327 (97%)</td>
<td>0.8445</td>
</tr>
<tr>
<td>ACE inhibitor or ARB</td>
<td>65 069 (69%)</td>
<td>2387 (73%)</td>
<td>&lt;0.0001</td>
<td>54 053 (79%)</td>
<td>1879 (83%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Aldosterone blocking agent</td>
<td>2471 (3%)</td>
<td>360 (10%)</td>
<td>&lt;0.0001</td>
<td>2030 (3%)</td>
<td>370 (15%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Statins</td>
<td>87 080 (90%)</td>
<td>3043 (86%)</td>
<td>&lt;0.0001</td>
<td>67 007 (95%)</td>
<td>2266 (93%)</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

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

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

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

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.
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. Specific therapies that minimize the risk of adverse LV remodeling and HF, such as renin-angiotensin aldosterone system antagonists or β-blockers, 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. In a landmark study of patients from the Global Registry of Acute Coronary Events (GRACE) Registry, Steg et al 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.

### 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

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

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.
(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 (NRMII-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 2,347 patients with HF or LV systolic dysfunction on admission or during hospitalization after STEMI, 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 intraaortic 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 poor23 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

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References


In-Hospital HF Post-MI

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Shah et al

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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

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