Hospital Performance and Differences by Kidney Function in the Use of Recommended Therapies After Non–ST-Elevation Acute Coronary Syndromes

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Abstract

Background—Chronic kidney disease (CKD) is associated with an increased risk of cardiac events and death, yet underuse of guideline-recommended therapies is widespread. The extent to which hospital performance affects the care of patients with CKD and non–ST-segment elevation acute coronary syndromes (NSTE ACS) is unknown.

Study Design—Observational cohort.

Setting & Participants—81,374 patients with NSTE ACS treated at 327 US hospitals.

Predictor—Hospital performance, as measured by quartiles of composite adherence to American Heart Association class I guidelines for therapy acutely (aspirin, beta-blockers, clopidogrel, heparin, glycoprotein IIb/IIIa inhibitors) and at discharge (aspirin, clopidogrel, angiotensin-converting enzyme inhibitors, lipid-lowering agents) in eligible patients.

Outcomes & Measurements—Use of each American Heart Association class I acute and discharge therapy, stratified by continuous estimated glomerular filtration rate (eGFR). Multivariable models were adjusted for demographics, clinical factors, and hospital features.

Results—Better performing hospitals had lower prescribing rates for most therapies (5 of 9) with lower levels of kidney function while lower performing hospitals were more likely to have similar prescribing rates across the eGFR spectrum, suggesting that prescribing patterns at these hospitals were insensitive to differences in eGFR.

Limitations—Observational design, selection bias of study cohort.

Conclusion—Patients with lower levels of kidney function admitted with NSTE ACS are less likely to receive evidence-based therapies. Treatment disparities related to CKD are most evident at top-performing hospitals.

Introduction

Chronic kidney disease (CKD) is associated with increased all-cause mortality, largely attributable to cardiovascular disease.[1] Because patients with CKD have particularly high cardiovascular risk, several guidelines recommend aggressive therapy with risk-reductive medications.[2–5] Yet, studies demonstrate that risk-reducing medications are dramatically underused, particularly during acute coronary syndromes. In fact, prescribing actually declines with lower levels of kidney function despite increasing risk for cardiovascular events.[6–14] Underuse of recommended therapies is a problem that is also found among several other populations including high-risk patients[14] and vulnerable groups including the elderly and women.[15]

In order to address such quality problems, several national health quality improvement initiatives have been launched.[9,16–19] These initiatives have demonstrated significant associations between selected care processes and outcomes,[16] supporting the use of broad, guideline-based performance metrics as a means of assessing and helping improve hospital quality. Among high-risk patient subgroups, improving the alignment between treatment intensity and patient risk remains an important area of investigation. However, limited data exist on the impact of hospital performance on the care of patients with CKD and cardiovascular disease. Thus, we sought to determine whether higher performing hospitals have better rates of prescribing at all levels of kidney function, and thus mitigate the paradoxical underuse of therapies among patients with non-ST-segment elevation acute coronary syndromes (NSTE ACS) at highest-risk for poor outcomes.

Methods

Data Source

We analyzed treatment patterns among eligible patients included in the CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Implementation of the American College of Cardiology/American Heart Association Guidelines) National Quality Improvement Initiative in the United States. Details of data collection methods and data validation have been previously described.[14,16,47] Between October 1, 2003, and June 30, 2006, a total of 101,946 patients with NSTE ACS (chest pain equivalent with ischemic ST-segment changes and/or positive cardiac markers) were treated at 472 hospitals throughout the United States. Participating hospitals collected data in an anonymous fashion for consecutive patients who met the inclusion criteria during the initial hospitalization. Next, 12,264 patients who were transferred out from participating hospitals were excluded because discharge medication data could not be collected after transfer as a result of current privacy laws. In order to define adequately site-level performance assessment and insure data quality, we further restricted the study cohort to those treated at sites with at least 40 patients during the study time period (n=2,341 excluded) and with at least 1 death recorded (n=1,468 excluded). We also excluded 4,499 patients with missing data on age, sex, race, or creatinine because these data were necessary for estimating kidney function. Thus, our study population consisted of 81,374 patients from 327 hospitals. Estimated glomerular filtration rates (eGFR) were calculated using admission serum creatinine values and the 4-variable Modification of Diet in Renal Disease (MDRD) Study equation[20] with values truncated at 100 (i.e., GFR values >100 were set at 100).

Outcomes

We evaluated hospitals' average use of 9 class I evidence-based therapies for patients with NSTE ACS endorsed by The American College of Cardiology (ACC)/American Heart Association (AHA) guidelines.21 For patients without contraindications, acute therapies upon hospital admission included aspirin, β-blockers, clopidogrel, heparin (unfractionated or low-molecular weight), and glycoprotein (GP) IIb/IIIa inhibitors. Discharge therapies were evaluated for patients without contraindications and who were discharged alive. Discharge therapies included aspirin; clopidogrel; lipid-lowering agents for patients with a history of dyslipidemia or low-density lipoprotein cholesterol levels >100 mg/dL; and angiotensin-converting enzyme (ACE) inhibitors for patients with a history of hypertension, history of heart failure, signs of heart failure, an in-hospital heart failure event, ejection fraction <40%, moderate or severe left ventricular dysfunction, or treated diabetes mellitus. Although angiotensin II receptor blockers may have been used as therapeutically equivalent substitutes for ACE inhibitors, they was not included in the ACE inhibitor quality measure because the prevailing guidelines during the study period did not specify it as an alternative.21 Contraindications included pre-specified clinical conditions as well as contraindications documented by clinicians.15 Missing outcomes were excluded when analyzing each specific outcome and were less then 1% for most outcomes except for acute clopidogrel (1.19%) and discharge ACE inhibitor (1.26%).

Hospital Ranking

Hospital performance was calculated by aggregating the number of times a therapy was administered divided by the sum of total eligible opportunities for all patients at a hospital. 16 Patient eligibility for each measure was determined according to defined ACC/AHA guideline indications and reported contraindications.21 Patients who died anytime during the hospital stay were considered ineligible for discharge medications. In order to assess the impact of hospital ranking on medication usage among CKD patients, the hospital performance was calculated based on patients without significant CKD (eGFR ≥60 mL/min/1.73 m2, n = 47,796) to establish a measure that was not confounded by the proportion of CKD patients within each hospital. After the performance score was calculated, the hospitals were separated into 4 ranks based on their performance scores: leading (>75%), 50–75%, 25–50%, and lagging (≤25%).16

Statistical Analysis

For descriptive analyses, comparisons between patients who were in hospitals of different ranks were made factoring in baseline characteristics, presenting features, and primary attending physician. Continuous variables are presented as median (25th, 75th percentile) and categorical variables are expressed as percentages. To test for the independence of a patient's baseline characteristics, presenting features, and primary attending physician with respect to hospitals of different ranks, Kruskal-Wallis nonparametric tests were used for continuous variables and Pearson chi-square tests were used for categorical variables.

Patients from hospitals within the same rank were divided into deciles based on their eGFR values. For each decile within each hospital group the proportion of patients who received each therapy as well as average eGFR values were calculated. The associations between eGFR and medication usage were then graphically displayed by plotting the proportion of patients who received each therapy by average eGFR stratified by hospital ranks.

Multivariable logistic regression models were used to estimate the marginal effects of eGFR in different ranks. The variables of interest in the model were eGFR (as a continuous variable), hospital rank (4-level categorical variable), and the interaction between eGFR and hospital rank. The relationship between eGFR and medication adherence of leading and

lagging quality ranks hospitals were compared in the regression models. Generalized estimating equations were used to account for within-hospital clustering,[22] because patients at the same hospital are more likely to be treated similarly relative to patients in other hospitals (i.e. considers within-center correlation for response). The method produces estimates similar to those from ordinary logistic regression, but the estimated variances of the estimates are adjusted for the correlation of outcomes within each hospital. The variables in adjusted models were sex, white race, BMI, age, heart rate at presentation, systolic blood pressure, family history of coronary artery disease, hypertension, diabetes mellitus, current/recent smoker, dyslipidemia, prior myocardial infarction, prior percutaneous coronary intervention, prior coronary artery bypass graft surgery, prior congestive heart failure, prior stroke, ischemic electrocardiogram abnormalities, heart failure at presentation, positive cardiac marker, primary attending, insurance status, number of hospital beds, region, hospital capabilities, and teaching hospital status. Odds ratios and 95% confidence intervals were presented per 10 ml/min/1.73m^2 increase in eGFR within each hospital quality rank to examine the variation of the strength of its influence on outcomes. Formal comparisons between leading and lagging hospitals for each outcome were carried out in the multivariable logistic regression models. A significant p-value for the comparison means the eGFR effects are different across leading and lagging hospital quality ranks.

A *P* value <0.05 was considered significant for all tests. All analyses were performed using SAS software (version 8.2, SAS Institute, Cary, NC).

Results

Hospital Characteristics

Among the study patients (N=81,374) who were enrolled from 327 hospitals, nearly one third of patients were in the leading hospitals (rank >75% for composite adherence) while nearly one in seven patients were in the lagging hospitals (rank ≤ 25% for composite adherence) (Table 1). Substantial differences of hospital features existed between the two groups with higher total hospital bed number and a greater proportion in the leading rank group having hospitals with cardiac catheterization, percutaneous coronary intervention, and coronary artery bypass grafting capabilities as well as teaching hospital status designations.

Patient Characteristics

The median age was 68 years, 40% were women, and almost one in nine patients was African American, although there was substantial variability across hospital rank groups (Table 2). The proportions of patients with various comorbid conditions also varied by hospital rank group: the leading group had higher proportions of patients with a history of hyperlipidemia and recent or current smoking status, while the lagging group had higher proportions of patients with hypertension, diabetes mellitus, prior heart failure and prior stroke. Average estimations of kidney function and the proportion of patients primarily cared by cardiology were also lower among those in the lagging group.

Estimated GFR Effect and Hospital Performance Groups

As expected, when compared to lower performing hospitals, higher performing hospitals were found to have higher rates of prescribing for all acute (Figure 1) and discharge (Figure 2) medications. Associations were explored between prescribing rates and level of kidney function across hospital performance groups. A significant eGFR effect demonstrated that prescribing rates varied across the eGFR spectrum such that prescribing rates for several acute (Table 3) and discharge (Table 4) medications declined with decreasing levels of kidney function.

Four patterns of this eGFR effect emerged when comparing the highest and lowest performing hospital rank groups. First, an eGFR effect was present in both the leading and lagging hospital rank groups for acute GP IIb/IIIa inhibitors, indicating that there was a decrease in the use of this medication across the spectrum of eGFR for both leading and lagging quality ranks. However, the absence of significance when comparing leading and lagging quality ranks indicated that there were no significant differences across the highest and lowest hospital rank groups in the observed decrease in use of this medication. Second, an eGFR effect was present in the leading but not the lagging hospital rank groups for the majority (5 of 9) of the guideline-recommended therapies, including several acute (aspirin, clopidogrel) and discharge (aspirin, clopidogrel, lipid-lowering agent) therapies. In this case, several comparisons between leading and lagging quality ranks were significant (4 out of 5) indicating that the relationship between eGFR and decrease in use of these medications were different between leading and lagging hospital ranks. In the third pattern, we found the opposite of what was observed in the previous pattern: an eGFR effect was present in the lagging but not the leading hospital rank groups for a single discharge therapy—use of an ACE inhibitor. This pattern was consistent with the initial study hypothesis that when compared to lower performing hospitals, higher performing hospitals would have consistent rates of prescribing at all levels of eGFR, mitigating the pattern of underuse commonly found at lower levels of eGFR. Unexpectedly, we also found a fourth pattern in which no eGFR effect was present in either the leading or lagging hospital rank groups for acute β-blocker and heparin therapies, indicating that prescribing rates for these medications did not vary according to eGFR across hospitals of both the leading and lagging quality ranks.

Discussion

In a large contemporary cohort of patients admitted with NSTE ACS in all regions of the United States, we confirmed that administration of evidence-based therapies declines with lower levels of kidney function across hospitals of all performance levels. Despite better rates of prescribing at all levels of kidney function among higher performing hospitals, for most therapies the paradoxical underuse among the patients at highest-risk is not diminished by their overall higher guideline performance.

Our findings of greater underuse of medications for cardiovascular disease as kidney function declines are consistent with numerous prior studies.[6–14] Underuse of recommended therapies is a problem that has long been recognized among patients with coronary artery disease.[12] Paradoxical under-utilization of evidence-based therapies has also been described in other high-risk and under-represented patient subgroups such as the elderly,[23] African-Americans,[24] women,[15] and patients with peripheral arterial disease,[25] diabetes mellitus,[26,27] or multiple chronic conditions.[14,28] Overall, underuse of appropriate therapies is responsible for the majority of the quality gap throughout all of healthcare.[29,30]

However, the extent to which quality improvement initiatives have reduced existing gaps in quality has not been well characterized. We sought to determine the impact upon quality gaps in therapies among patients with CKD admitted with NSTE ACS. Despite a higher risk for poor outcomes, patients with lower levels of kidney function were less likely to receive nearly all guideline-based therapies except for acute β-blockers, even among higher performing hospitals. But when we compared higher performing and lower performing hospitals, four patterns emerged of how prescribing rates declined with decreasing levels of kidney function.

The first pattern demonstrated decreased use of GP IIb/IIIa inhibitors across the spectrum of eGFR without significant differences between leading and lagging quality ranks, suggesting that there may be a very strong reason for underuse with lower levels of kidney function.

Because GP IIb/IIIa inhibitors are recommended for high-risk patients or those with ongoing and recurrent ischemia, overall use of this acute therapy was considerably lower than the others evaluated here. Nonetheless, patients with lower levels of kidney function were not more likely to be low-risk or have less ongoing and recurrent ischemia. The most likely explanation for the pattern observed is that the risk of bleeding increases substantially with lower levels of kidney function.[31,32] The risk of major bleeding has been found to be about 1.5 times higher among patients with creatinine clearances between 30–60 mL/min and 2– 2.5 times higher among patients with creatinine clearances <30 mL/min. Further, some episodes of bleeding after therapy with GP IIb/IIIa inhibitors may result in death.[33] However, overdosing of GP IIb/IIIa inhibitor therapy increases in frequency among patients with lower levels of kidney function and may be responsible for many of these adverse bleeding events. Given the higher potential to overdose patients with lower eGFR, physicians may simply be avoiding such risks by not prescribing GP IIb/IIIa inhibitor therapy. Hopefully, future studies will provide better dosing algorithms that maximize the benefits of anti-platelet therapy while minimizing the adverse risks of bleeding.[34]

The majority of the therapies that we evaluated were characterized by the second pattern in which there were significant differences between leading and lagging quality ranks. Specifically, a decrease in use of these medications was observed in the leading but not the lagging hospital rank groups for several acute (aspirin, clopidogrel) and discharge (aspirin, clopidogrel, lipid-lowering agent) therapies. It is not clear why these treatment disparities by eGFR were highest among the highest performing hospitals. Inadequate experience due to low volumes of patients with lower eGFR is not likely to account for these differences because the leading hospitals provided care for almost one-third of patients and were likely to have more experience with ACS care as a result. Although the potential risks associated with these medications are not as severe as those for GP IIb/IIIa inhibitors, each one has a relatively higher rate of side effects in the setting of lower levels of kidney function.[32,35] As a result, physicians at higher quality hospitals may have been more cautious in using these medications in patients with lower levels of kidney function while those at lower quality hospitals were less sensitive to these potential side effects and did not alter their prescribing pattern. Alternatively, prescribing rates at lower quality hospitals were much lower overall and as a result, may have obscured the impact of lower eGFR on treatment patterns. In addition to various local practices at higher quality hospitals, the presence of computer-based decision support systems may have guided medication dosing for patients with lower levels of kidney function.[36] Nonetheless, it remains unclear whether this under prescribing is beneficial since several of these agents have been demonstrated to be effective and safe among patients with mildly to moderately decreased kidney function.[37,38]

Only one therapy demonstrated the pattern consistent with the initial study hypothesis that when compared to lower performing hospitals, higher performing hospitals would have higher rates of prescribing at all levels of eGFR. Unlike the patterns found among lower quality hospitals, underuse of ACE inhibitors as eGFR declined was not as prevalent at higher quality hospitals. Many physicians become concerned when the serum creatinine rises after initiating therapy, however, these patients may be the ones who benefit the most. [39] Use of ACE inhibitors often declines with lower levels of kidney function,[13,40] however, the benefits after myocardial infarction seen in the general population likely applies to those with mildly to moderately decreased kidney function as well.[41] Because ACE inhibitor therapy can precipitate acute kidney failure in the presence of severe bilateral renal artery stenosis, many physicians withdraw ACE inhibitors when kidney function declines.[42] Renal artery stenosis has long been recognized among patients who undergo coronary angiography and can be relatively common.[43] Perhaps physicians at the higher performing hospitals were more likely to recognize the benefits of therapy, and in turn were more willing to treat with ACE inhibitors despite the potential of precipitating acute kidney failure.

Finally, the last pattern that we observed was unexpected: prescribing rates for acute β-blocker and heparin therapies did not vary according to eGFR across hospitals of leading and lagging quality ranks. Although a ceiling effect was likely present at the higher performing hospitals where rates of use were above 90–95%, there may still be some room for improvement among lower performing hospitals where rates of use were 70–80%. For β-blocker therapy, the consistency of prescribing rates across the entire range of kidney function is likely related to several factors. First, β-blocker therapy has long been known to be effective in reducing mortality after acute myocardial infarctions.44 Second, it is generally well tolerated and does not require renal dosage adjustments. Third, it has long been targeted in quality measurement efforts. In fact, improving rates of acute β-blocker therapy has been so successful that it was recently "put to rest" as a quality measure.[45] Our findings are consistent with this decision that this measure no longer reflects significant variability in quality, particularly with respect to gaps among patients with lower levels of kidney function. Although prescribing rates did not vary according to eGFR across hospitals of leading and lagging quality ranks for heparin therapy, they did vary significantly for the middle 2 quality rank groups and the effect in the leading quality group was moderate, but not significant (OR 1.030; p=0.08). Thus, the pattern observed for acute heparin therapy was mixed and reflected a varied pattern of underuse according to eGFR rather than the truly flat response observed for β-blocker therapy.

The findings in our study must be considered in light of several limitations. First, CRUSADE hospitals were self-selected for those interested in this national quality improvement initiative and may not be fully representative of all community hospitals in the US. In addition, our study cohort excluded patients who were transferred to other facilities which likely biased the study sample at facilities with limited cardiac services by excluding lower-risk patients.[46] As a result, patients who remained at hospitals without diagnostic catheterization capabilities may have been less likely to receive some acute therapies.[47] Variability in creatinine measurements and the known imprecision of the MDRD Study equation among patients with normal kidney function and mild CKD may have lead to some inaccuracy in our eGFR estimates.[20] Outcomes related to the underuse of the therapies evaluated in this study were not ascertained because of our limited ability to fully account for the selection bias among those who receive the various guideline-recommended treatments. We could not account for undocumented contraindications that may have significantly influenced treatment patterns, including possible site-level variation in listed contraindications for GP IIb/IIIa inhibitor therapy. In addition, we could not account for informed risk-benefit decisions between clinicians and patients that influenced lower use of these therapies. Because we only assessed medication use until discharge, we were not able to examine adherence after discharge when other studies have observed fewer differences in medication use among patients with lower levels of kidney function.[48,49] Finally, we were not able to assess changes in medication use over time to determine whether there may have been improvements that reduced the underuse gap.

Despite a higher risk for poor outcomes, patients with lower levels of kidney function admitted with NSTE ACS were less likely to receive evidence-based therapies. Although higher performing hospitals had better rates of prescribing at all levels of kidney function, for most therapies the underuse among patients at highest-risk is not diminished by their higher overall performance according to established guidelines. In fact, treatment disparities by eGFR were highest among the highest performing hospitals, even among patients with mildly to moderately decreased kidney function. The reasons for persistently low prescribing rates of most therapies at lower levels of kidney function need to be explored, even among higher performing hospitals. Subsequent efforts to promote adherence to NSTE ACS guidelines may result in more favorable prescribing rates of recommended therapies among patients with CKD who are at particularly high-risk for adverse outcomes.

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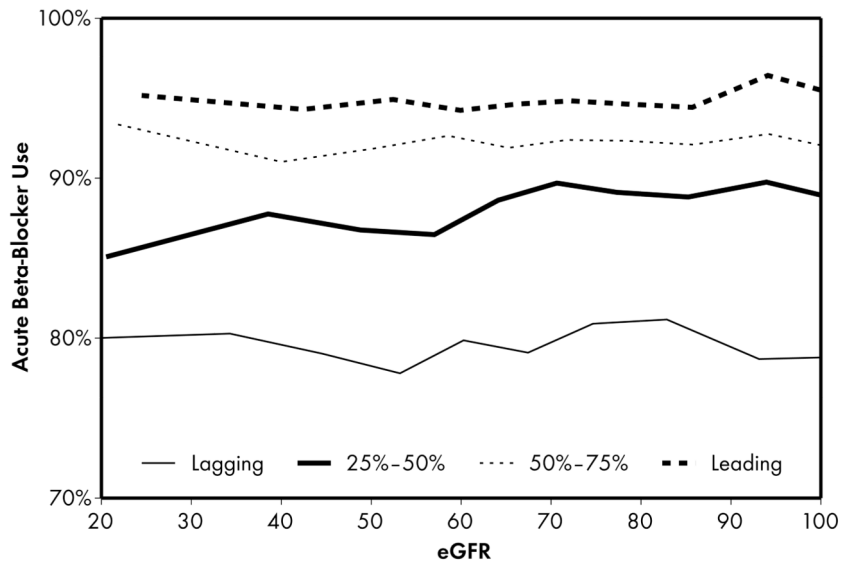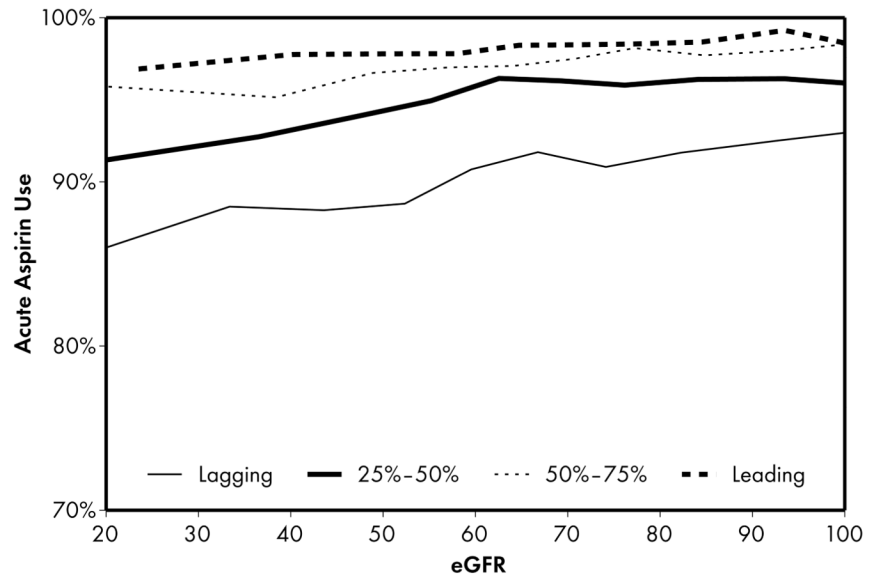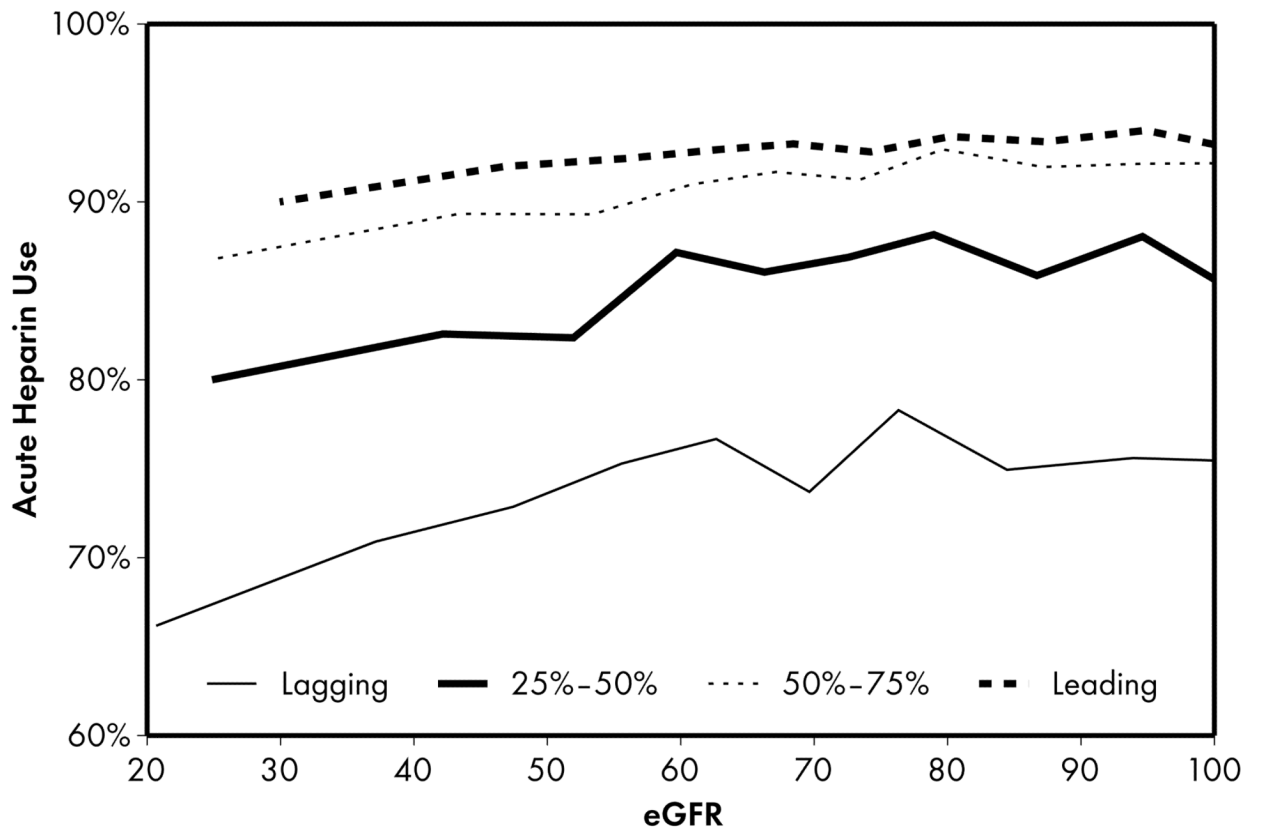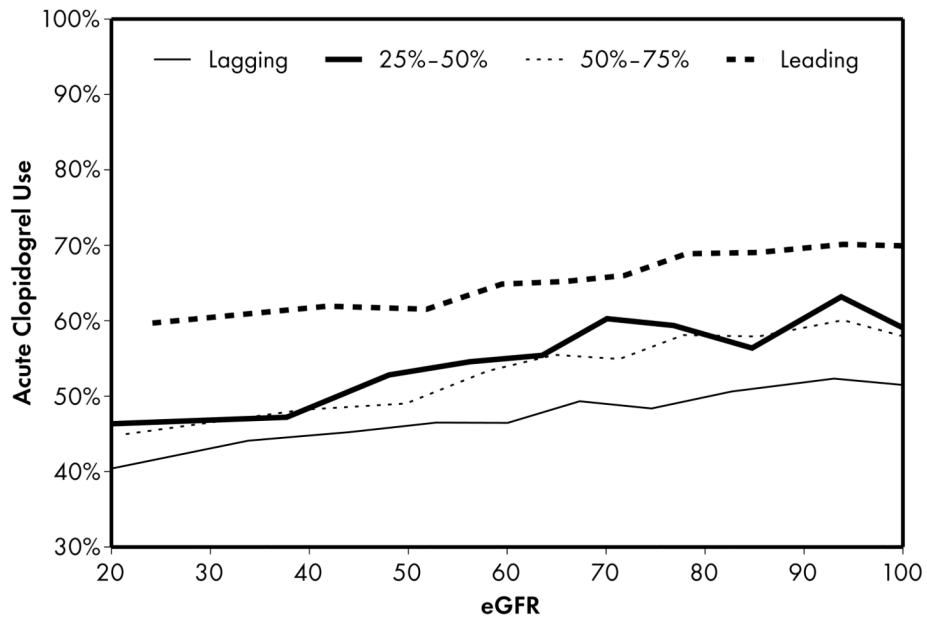
Figure 1.
Higher performing hospitals were found to have higher rates of prescribing for each of the 5 guideline-recommended acute medications: aspirin (1a), β-blocker (1b), clopidogrel (1c), heparin (1d), and GP IIb/IIIa inhibitors (1e). An eGFR effect was present in both the leading and lagging hospital rank groups for GPIIb/IIIa inhibitors (1e) indicating that there was a decrease in use of this medication across the spectrum of eGFR while no eGFR effect was present in either rank groups for β-blocker and heparin therapies (1b, 1d), indicating that prescribing rates for this medication do not vary according to eGFR across hospitals of all quality ranks. For the other therapies (1a, 1c), an eGFR effect was present in the leading but not the lagging hospital rank groups.
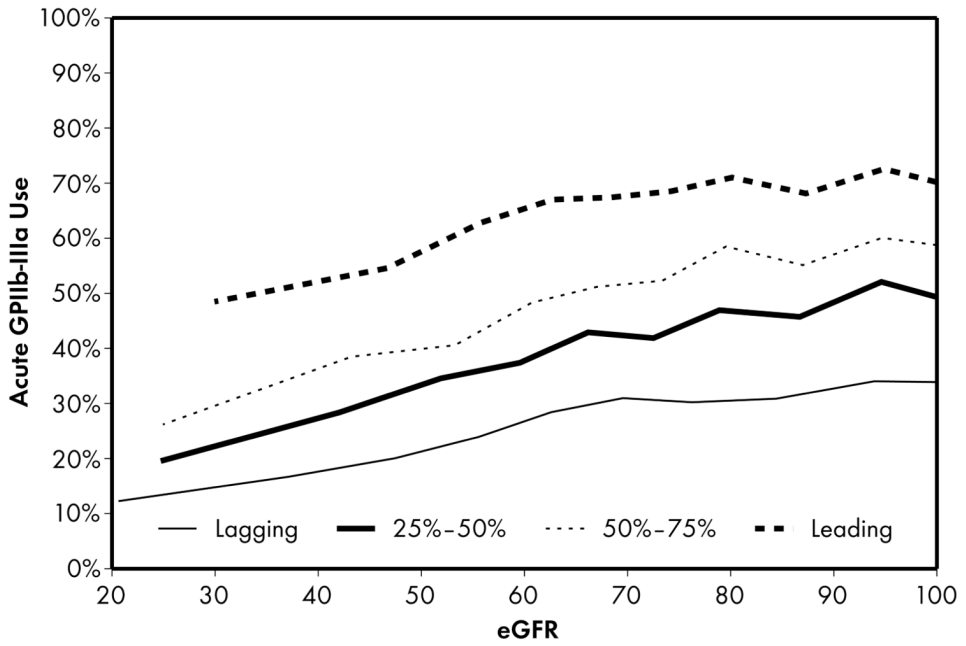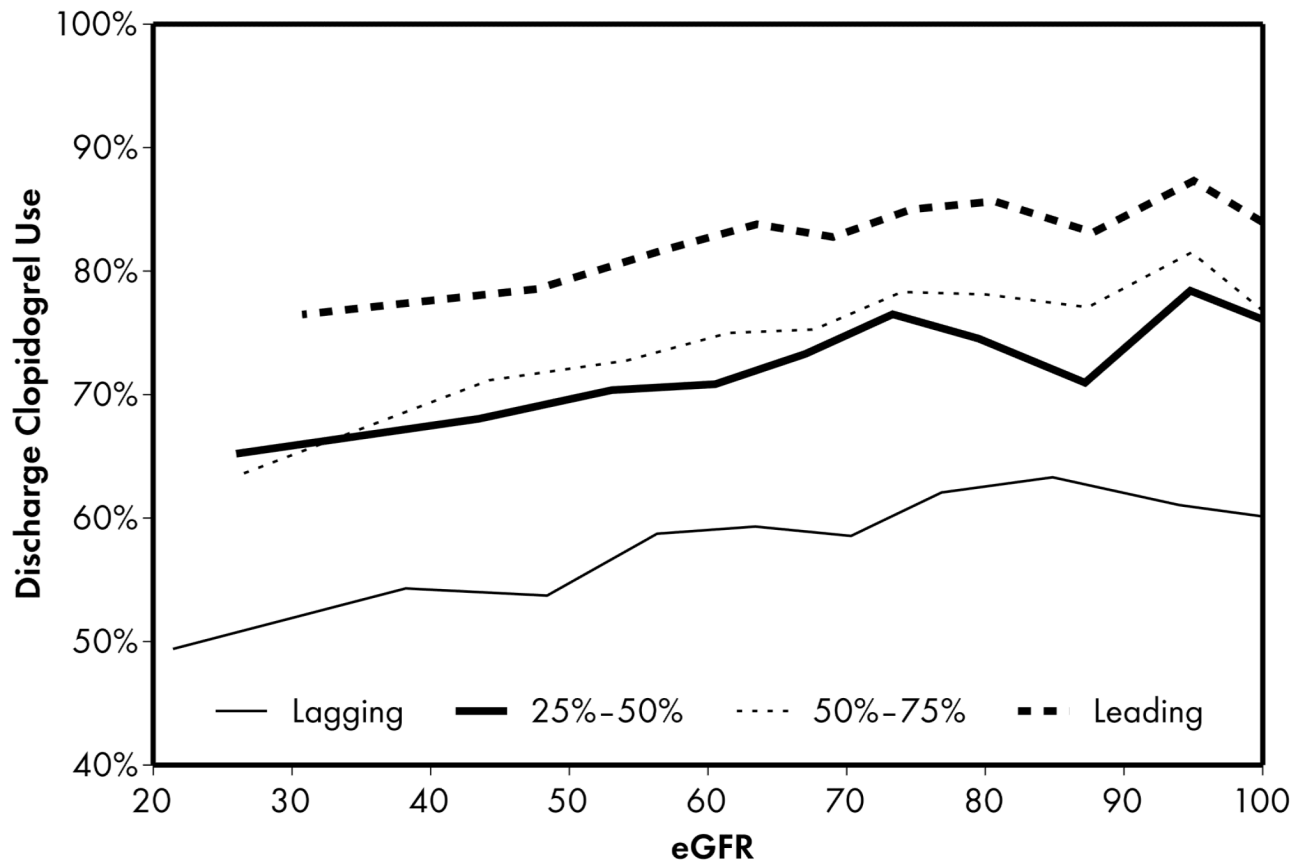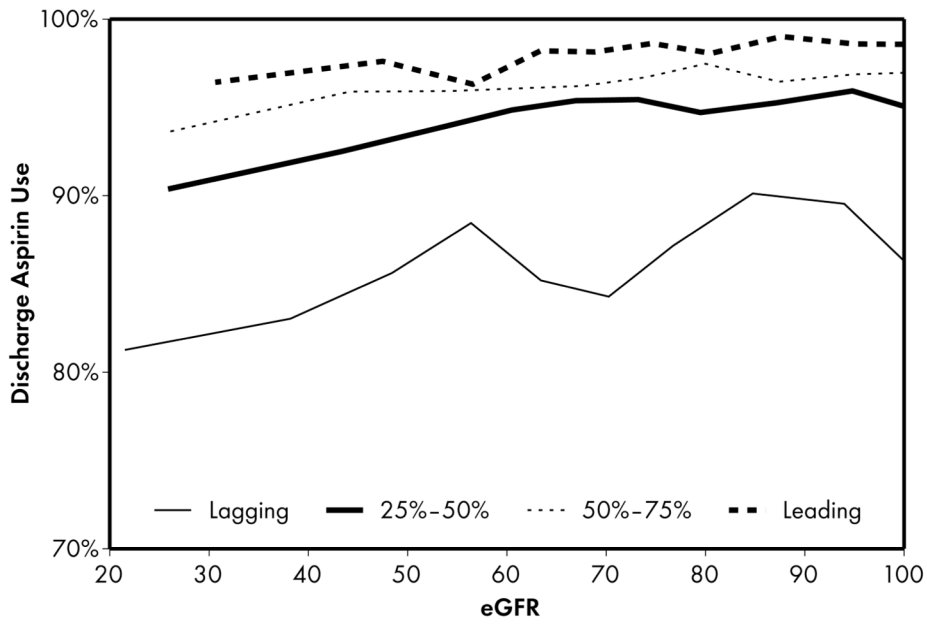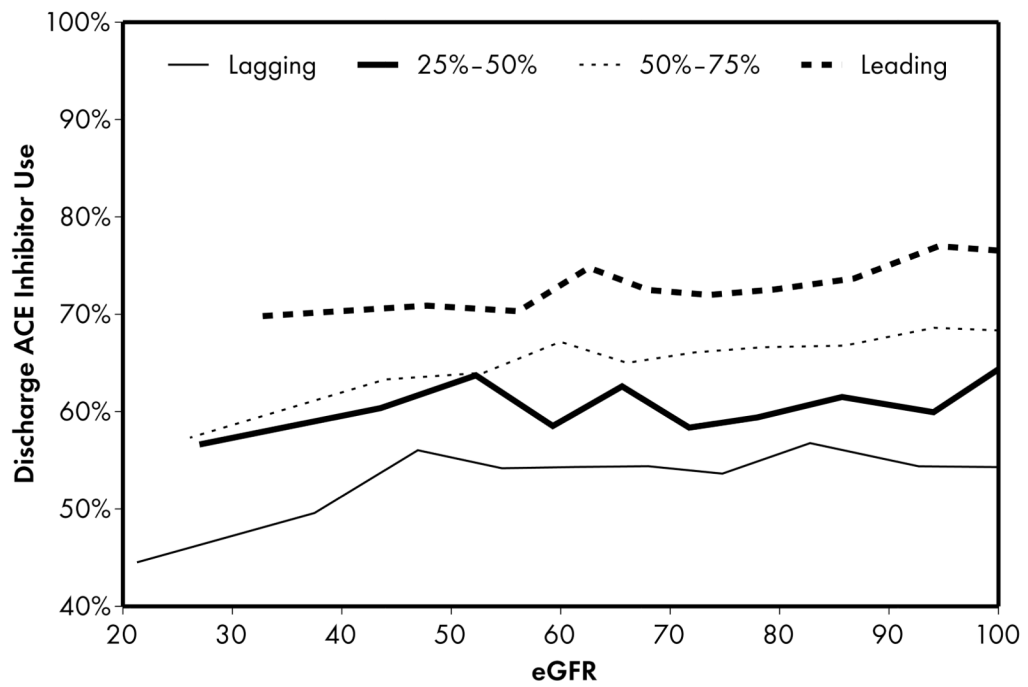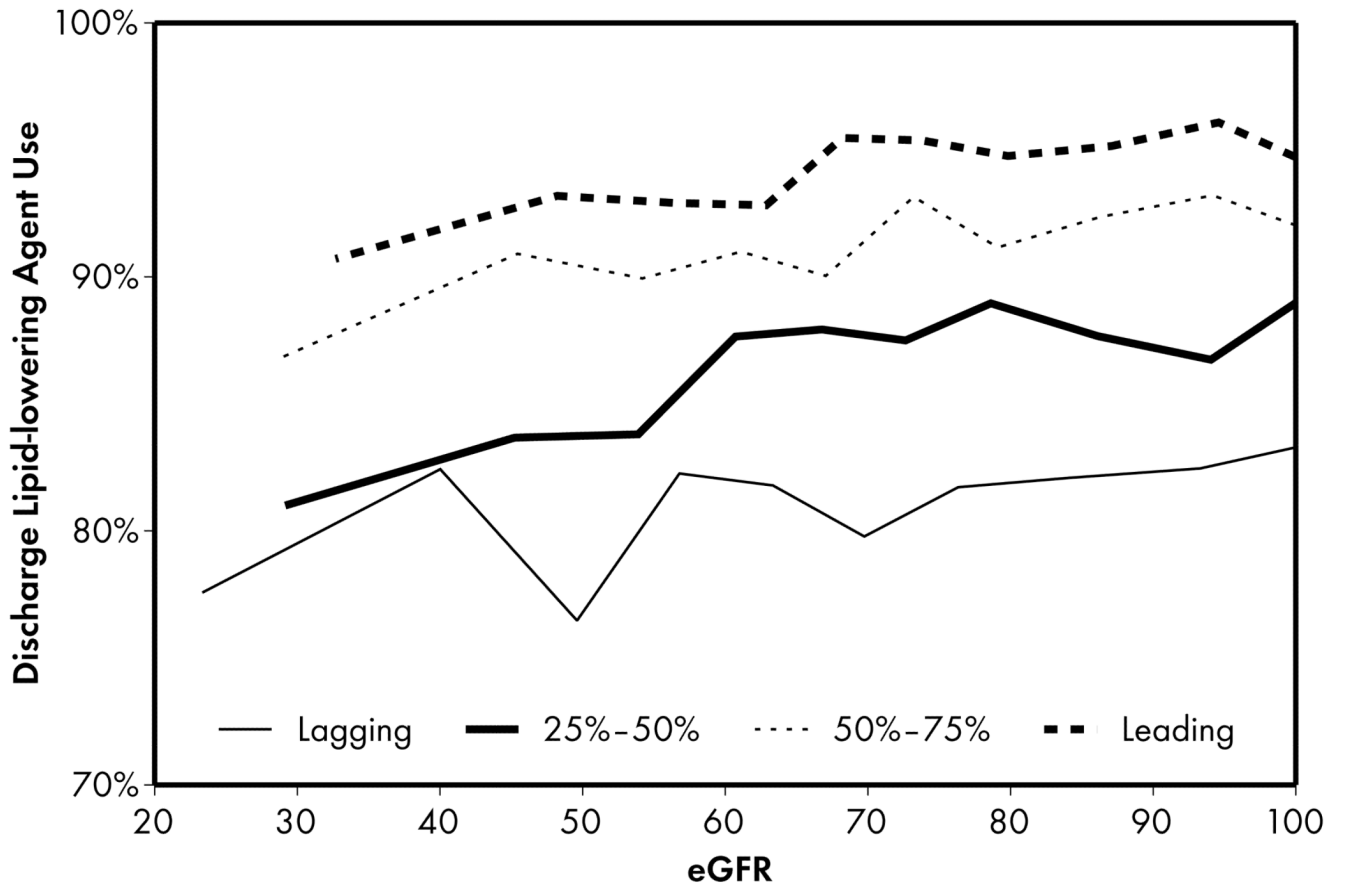
Figure 2.

Higher performing hospitals were also found to have higher rates of prescribing for each of the 4 guideline-recommended discharge medications: aspirin (2a), clopidogrel (2b), lipid-lowering agent (2c), and ACE inhibitor (2d). For most therapies (2a, 2b, 2c), an eGFR effect was present in the leading but not the lagging hospital rank groups. An eGFR effect was present in the lagging but not the leading hospital rank groups for ACE inhibitor use (Fig 2d), a pattern consistent with the initial study hypothesis that higher performing hospitals would have higher rates of prescribing at all levels of eGFR, mitigating the pattern of underuse commonly found as eGFR declines.

Table 1

Baseline Hospital Characteristics by Hospital Performance Group

Variable	Overall	Lagging (≤25%)	25–50%	50–75%	Leading (>75%)	P-value
	N=327 (100%)	n=81 (24.8%)	n=82 (25.1%)	n=82 (25.1%)	n=82 (25.1%)	
Hospital Features						
Total hospital beds[a]	325 (217, 473)	258 (183, 375)	337 (210, 460)	386 (250, 567)	329 (242, 482)	0.0008
Hospital services						0.0001
No services	6.4	11.1	9.8	1.2	3.7	
Cardiac cath only	9.2	18.5	11.0	1.2	6.1	
Cardiac cath and PCI	8.0	12.4	4.9	6.1	8.5	
Cardiac cath, PCI and CABG	76.5	58.0	74.4	91.5	81.7	
Teaching hospital[b]	26.0	14.8	26.8	39.0	23.2	0.005

Abbreviations: CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention.

Data are expressed as percentages except for continuous variables

[a]Expressed as medians (25th, 75th percentile).

[b]Member of the Council of Teaching Hospitals.

Table 2

Baseline Patient Characteristics by Hospital Performance Group

Variable	Overall N=81,374 (100%)	Lagging (≤25%) n=12,226 (15.0%)	25–50% n=18,201 (22.4%)	50–75% n=26,123 (32.1%)	Leading (>75%) n=24,824 (30.5%)	P-value
Demographics						
Age, y[a]	68 (56, 79)	71 (58, 81)	69 (58, 80)	68 (56, 78)	67 (56, 78)	<0.0001
Men	60.1	55.9	60.0	60.6	61.7	<0.0001
African-American	10.8	13.6	8.8	12.1	9.4	<0.0001
Past Medical History						
Hypertension	71.3	71.9	71.8	72.0	70.0	<0.0001
Diabetes mellitus	33.3	36.0	33.3	33.8	31.3	<0.0001
Hyperlipidemia	51.8	43.5	50.2	54.2	54.6	<0.0001
Current/recent smoking	27.2	24.2	25.3	28.2	29.0	<0.0001
Prior MI	29.2	27.3	26.3	31.6	29.8	<0.0001
Prior heart failure	17.9	21.4	18.5	17.8	15.8	<0.0001
Prior PCI	21.4	19.2	20.9	22.2	22.1	<0.0001
Prior CABG	19.5	19.6	19.7	19.7	19.2	0.3
Prior stroke	10.4	11.7	10.2	10.7	9.6	<0.0001
Presenting Features						
+ Cardiac markers	91.4	84.4	90.8	93.4	93.3	<0.0001
ST depression	30.6	28.8	31.6	30.5	30.9	<0.0001
Signs of heart failure	24.3	25.9	26.2	23.5	23.1	<0.0001
Heart rate (bpm)[a]	83 (70, 99)	85 (72, 101)	83 (70, 100)	83 (70, 98)	82 (70, 97)	<0.0001
Systolic BP (mm Hg)[a]	144 (123, 165)	143 (122, 164)	142 (122, 163)	144 (124, 165)	145 (125, 166)	<0.0001
SCr (mg/dL)[b]	1.39 (1.53)	1.51 (1.62)	1.43 (2.00)	1.38 (1.43)	1.32 (1.16)	<0.0001
eGFR (mL/min/1.73m)^{2c}	65.7 (47.0, 83.7)	62.3 (42.1, 81.2)	65.0 (45.9, 83.5)	66.7 (48.2, 84.3)	67.2 (49.8, 84.1)	<0.0001
CCr (mL/min)[a,d]	53.2 (34.1, 75.6)	47.1 (29.5, 69.8)	51.6 (33.0, 75.0)	54.3 (34.9, 76.4)	56.2 (36.9, 77.6)	<0.0001
Primary Attending						
Care by cardiologist	59.0	45.3	58.0	59.6	65.9	<0.0001

Abbreviations: BP, blood pressure; CABG, coronary artery bypass grafting; CCr, creatinine clearance; eGFR, estimated glomerular filtration rate; HF, heart failure; MI, myocardial infarction; PCI, percutaneous coronary intervention; SCr, serum creatinine.

Data are expressed as percentages except for continuous variables

[a] Expressed as medians (25th, 75th percentile)

[b] Expressed as mean (SD)

Conversion factors: serum creatinine in mg/dL to mmol/L, x88.4; eGFR in mL/min/1.73 m^2 to mL/s/1.73 m^2, x0.01667.

[c] Determined by using MDRD Study equation.

[d] Determined with Cockcroft-Gault formula.

Table 3

Associations between eGFR Effect (per 10 ml/min/1.73m^2 increase) and 5 Guideline-recommended Acute Therapies according to Hospital Performance Groups

Category	Adjusted OR (95% CI)^a	P-value
Acute Aspirin		0.2^b
eGFR effect in leading (>75%) hospitals	1.06 (1.02, 1.10)	0.007
eGFR effect in 50–75% hospitals	1.06 (1.03, 1.10)	<0.001
eGFR effect in 25–50% hospitals	1.07 (1.03, 1.10)	<0.001
eGFR effect in lagging (≤25%) hospitals	1.02 (0.99, 1.06)	0.2
Acute β-Blockers		0.7^b
eGFR effect in leading (>75%) hospitals	1.01 (0.98, 1.04)	0.8
eGFR effect in 50–75% hospitals	0.99 (0.98, 1.01)	0.5
eGFR effect in 25–50% hospitals	1.03 (1.01, 1.06)	0.005
eGFR effect in lagging (≤25%) hospitals	1.00 (0.98, 1.02)	0.7
Acute clopidogrel		0.006^b
eGFR effect in leading (>75%) hospitals	1.03 (1.01, 1.05)	0.001
eGFR effect in 50–75% hospitals	1.03, (1.02, 1.05)	<0.001
eGFR effect in 25–50% hospitals	1.03, (1.02, 1.05)	<0.001
eGFR effect in lagging (≤25%) hospitals	0.99, (0.97, 1.01)	0.3
Acute Heparin		0.4^b
eGFR effect in leading (>75%) hospitals	1.03 (0.97, 1.07)	0.08
eGFR effect in 50–75% hospitals	1.05, (1.02, 1.07)	<0.001
eGFR effect in 25–50% hospitals	1.05, (1.02, 1.07)	<0.001
eGFR effect in lagging (≤25%) hospitals	1.01 (0.99, 1.03)	0.2
Acute GPIIb/IIIa inhibitors		0.7^b
eGFR effect in leading (>75%) hospitals	1.04 (1.02, 1.06)	<0.001
eGFR effect in 50–75% hospitals	1.08 (1.06, 1.10)	<0.001
eGFR effect in 25–50% hospitals	1.06 (1.06, 1.08)	<0.001
eGFR effect in lagging (≤25%) hospitals	1.05 (1.02, 1.07)	<0.001

Abbreviations: eGFR, estimated glomerular filtration rate; GP, glycoprotein.

[a] Variables in the model – sex, white race, body mass index, age, heart rate, systolic blood pressure, family history of coronary artery disease, hypertension, diabetes, current/recent smoker, dyslipidemia, prior myocardial infarction, prior percutaneous coronary intervention, prior coronary artery bypass surgery, prior heart failure, prior stroke, ischemic electrocardiogram abnormalities, heart failure, positive cardiac markers; primary attending, insurance type, number of hospital beds, region, hospital capabilities, teaching/nonteaching hospital.

[b] P-value for comparison of Odds Ratios between leading and lagging hospital quality ranks.

Table 4

Associations between eGFR Effect (per 10 ml/min/1.73m^2 increase) and 4 Guideline-recommended Discharge Therapies According to Hospital Performance Groups

Category	Adjusted OR (95% CI)[a]	P-value
Discharge Aspirin		**0.02**[b]
eGFR effect in leading (>75%) hospitals	1.06 (1.02, 1.11)	0.003
eGFR effect in 50–75% hospitals	1.05 (1.02, 1.08)	0.002
eGFR effect in 25–50% hospitals	1.06 (1.03, 1.09)	<0.001
eGFR effect in lagging (≤25%) hospitals	1.00 (0.98, 1.02)	0.9
Discharge clopidogrel		**0.02**[b]
eGFR effect in leading (>75%) hospitals	1.03 (1.01, 1.048)	0.002
eGFR effect in 50–75% hospitals	1.04 (1.03, 1.058)	<0.001
eGFR effect in 25–50% hospitals	1.02 (1.00, 1.03)	0.08
eGFR effect in lagging (≤25%) hospitals	0.99 (0.97, 1.02)	0.5
Discharge lipid-lowering agent[c]		**0.01**[b]
eGFR effect in leading (>75%) hospitals	1.050 (1.02, 1.08)	<0.001
eGFR effect in 50–75% hospitals	1.051 (1.02, 1.08)	0.001
eGFR effect in 25–50% hospitals	1.04 (1.01, 1.07)	0.007
eGFR effect in lagging (≤25%) hospitals	1.00 (0.96, 1.03)	0.8
Discharge ACE inhibitors[d]		**0.05**[b]
eGFR effect in leading (>75%) hospitals	1.02 (0.99, 1.04)	0.2
eGFR effect in 50–75% hospitals	1.04 (1.01, 1.07)	0.02
eGFR effect in 25–50% hospitals	1.00 (0.98, 1.03)	0.9
eGFR effect in lagging (≤25%) hospitals	1.05 (1.03, 1.07)	<0.001

Abbreviations: eGFR, estimated glomerular filtration rate; ACE, angiotensin-converting enzyme.

[a] Variables in the model – sex, white race, body mass index, age, heart rate, systolic blood pressure, family history of coronary artery disease, hypertension, diabetes, current/recent smoker, dyslipidemia, prior myocardial infarction, prior percutaneous coronary intervention, prior coronary artery bypass surgery, prior heart failure, prior stroke, ischemic electrocardiogram abnormalities, heart failure, positive cardiac markers; primary attending, insurance type, number of hospital beds, region, hospital capabilities, teaching/nonteaching hospital.

[b] P-value for comparison of Odds Ratios between leading and lagging hospital rank groups

[c] Among patients with history of dyslipidemia or low-density lipoprotein cholesterol levels >100 mg/dL

[d] Among patients with history of, signs of, or in-hospital congestive heart failure, ejection fraction <40%, severe or moderate left ventricle dysfunction, diabetes mellitus, or hypertension