Mortality After Acute Myocardial Infarction in Hospitals That Disproportionately Treat Black Patients

Jonathan Skinner, PhD; Amitabh Chandra, PhD; Douglas Staiger, PhD; Julie Lee, PhD; Mark McClellan, MD, MPA, PhD

Background—African Americans are more likely to be seen by physicians with less clinical training or to be treated at hospitals with longer average times to acute reperfusion therapies. Less is known about differences in health outcomes. This report compares risk-adjusted mortality after acute myocardial infarction (AMI) between US hospitals with high and low fractions of elderly black AMI patients.

Methods and Results—A prospective cohort study was performed for fee-for-service Medicare patients hospitalized for AMI during 1997 to 2001 (n = 1 136 736). Hospitals (n = 4289) were classified into approximate deciles depending on the extent to which the hospital served the black population. Decile 1 (12.5% of AMI patients) included hospitals without any black AMI admissions during 1997 to 2001. Decile 10 (10% of AMI patients) included hospitals with the highest fraction of black AMI patients (33.6%). The main outcome measures were 90-day and 30-day mortality after AMI. Patients admitted to hospitals disproportionately serving blacks experienced no greater level of morbidities or severity of the infarction, yet hospitals in decile 10 experienced a risk-adjusted 90-day mortality rate of 23.7% (95% CI 23.2% to 24.2%) compared with 20.1% (95% CI 19.7% to 20.4%) in decile 1 hospitals. Differences in outcomes between hospitals were not explained by income, hospital ownership status, hospital volume, census region, urban status, or hospital surgical treatment intensity.

Conclusions—Risk-adjusted mortality after AMI is significantly higher in US hospitals that disproportionately serve blacks. A reduction in overall mortality at these hospitals could dramatically reduce black-white disparities in healthcare outcomes. (Circulation. 2005;112:2634-2641.)

Key Words: death, sudden ■ myocardial infarction ■ hospitals ■ outcomes ■ race
been described elsewhere. As noted by previous studies, measures of risk-adjusted AMI mortality have been shown to be biased by differences across hospitals in the average severity of disease. However, measures of risk-adjusted AMI mortality have been shown to be valid indicators of hospital quality and have been incorporated into hospital profiling efforts, for example, those developed by the Agency for Healthcare Research and Quality. Flexible quadratic age and gender interaction terms (age, age², sex, age×sex, and age²×sex) were included in all analyses. As well, the following disease categories were entered separately as categorical variables: vascular disease, dementia, renal disease, pulmonary disease, diabetes (with and without complications), liver disease (with and without complications), and cancer (nonmetastatic and metastatic). Also included were year categorical variables (with the year 2001 being the reference year) and categorical variables that indicated the severity of the AMI, whether anterior, inferior, subendocardial, or a reference “other” category.

**Methods**

**Data**

The primary data set was a longitudinal sample from the 100% Medicare fee-for-service population hospitalized for AMI between January 1997 and September 2001. The criterion for determining the presence of AMI from the claims was a primary diagnosis code of AMI (I2100–I2109) without evidence of an old myocardial infarction. Federal hospitals were excluded. The initial sample with valid provider and location identification comprised 1,254,786 individuals. Patients were assigned to their hospital of initial admission for heart disease treatment, even if the patient was later treated at another hospital. Using information from the Medicare Denominator File, the race of each patient was determined as black, other (which includes Hispanic identification), or the residual group, which we denote as “white.” Because of concerns about the statistical power required to discern outcome differences and the low sensitivity of Hispanic responses, we exclude respondents in the “other” category (n=42,200), which left 2 groups, black and white. There is a very strong correlation between black racial measures in the Medicare claims data and self-reported racial identity.

Observations were excluded if there was evidence from claims data of a previous myocardial infarction (n=54,357) or if patients enrolled in a health maintenance organization during the calendar year after the AMI index event (n=17,160). (Patients enrolled in a risk-bearing health maintenance organization at the time of the AMI were not in this sample because there was no record of the AMI on the claims data.) Additional criteria for exclusion were the inability to match the patient’s zip code to the patient’s region of residence (n=1,141), lack of valid income data for that zip code (n=2,019), and hospitals with fewer than 10 AMIs over the entire period of analysis (n=1,400), which left a sample of 1,136,736.

This sample was used to calculate the percentage of all AMI patients in a hospital who were black. We then created approximate deciles of this measure to provide a summary measure of the extent to which a hospital serves the black community. The lowest “decile” comprised the 12.5% of patients admitted to hospitals without any black AMI patients during the period 1997 to 2001. The use of this slightly larger grouping avoided the need to split the sample in an arbitrary way. Decile 2 is attenuated as a result, so that the bottom 2 groups constitute one fifth of the sample. The remaining deciles are defined conventionally. Patient counts in each of these higher deciles were not exactly 10% because patients in a given hospital were retained in the same decile category.

**Measuring Healthcare Outcomes**

The primary measure of outcomes was risk-adjusted 90-day mortality rate. Although risk-adjusted 30-day mortality rates are also presented, we favor 90-day rates because they are less likely to penalize hospitals with high rates of revascularization and subsequent operator mortality. Previous uses of these outcome data have been described elsewhere. As noted by previous studies, measures of hospital performance that use patient outcome data can be biased by differences across hospitals in the average severity of disease. However, measures of risk-adjusted AMI mortality have been shown to be valid indicators of hospital quality and have been incorporated into hospital profiling efforts, for example, those developed by the Agency for Healthcare Research and Quality.

Flexible quadratic age and gender interaction terms (age, age², sex, age×sex, and age²×sex) were included in all analyses. As well, the following disease categories were entered separately as categorical variables: vascular disease, dementia, renal disease, pulmonary disease, diabetes (with and without complications), liver disease (with and without complications), and cancer (nonmetastatic and metastatic). Also included were year categorical variables (with the year 2001 being the reference year) and categorical variables that indicated the severity of the AMI, whether anterior, inferior, subendocardial, or a reference “other” category.

**Analysis**

Multivariable logistic regression models were estimated for risk-adjusted 90-day (and 30-day) mortality across the deciles of the percentage of black patients in each hospital. In each model reported here, standard errors were clustered at the hospital level, and all statistical analysis was performed with STATA version 8.0. We wish to facilitate exposition and to avoid misinterpreting ORs as relative risks when the underlying event is not rare, and so we report expected probabilities rather than ORs. We used the ADJUST command in STATA, which sets all covariates to their mean values and then “turns on” each of the decile categorical variables in turn. For each decile, an estimate and CI was calculated in log-ORs; these were then converted into probability units. This was the expected mortality rate (and CI) for a representative patient, one with average risk characteristics.

**Potential Explanations for Differences in Risk-Adjusted Mortality Outcome Measures**

We examined the role of 6 observable factors that might explain differences in hospital-level mortality outcomes. The first was that the different racial composition of the deciles could lead to unmeasured confounding if black patients exhibited higher rates of mortality even after adjustment for risk factors. We addressed this hypothesis in 2 ways. The first was to estimate the logistic model with race-decile interaction terms, which allowed for 2 separate mortality gradients, one for black and the other for white AMI patients. The disadvantage of this approach is that there were very few black AMI patients in the lower deciles, with a corresponding deficiency in statistical power. We therefore combined deciles 2 to 6 into 1 group that comprised 11.4% of black patients and 39.8% of white patients. The second approach was to estimate a logistic model separately for black and white patients, but with a single variable, the percentage of black AMI patients in the hospital (a hospital-level variable), to test for a linear race-specific gradient in the logistic regression.

Second, hospitals that admitted black AMI patients have been shown to be less likely to perform surgical interventions. To capture these effects, hospital-specific rates of CABG and percutaneous coronary interventions in the sample were included in 1 specification of the regression. Third, hospitals may differ with respect to average volume of treatment. The hospital-specific AMI volume was therefore included as an additional explanatory variable. Fourth, the ownership status of hospitals could confound racial effects if black patients were more likely to be admitted to government hospitals. We therefore used indicator variables to adjust for the teaching and ownership status of the hospital (government [non-Federal], not-for-profit, and for-profit). As noted by others, these variables are markers for multiple competing factors and should not be interpreted as measuring the effect of ownership, volume, or treatment intensity per se.

Fifth, there may be systematic differences in income levels across regions, and so we adjusted by median household income by zip code from the 2000 US census. Finally, we adjusted for location of residence using the 4 US census regions and whether the individual lived in an urban area. We note that adjusting for geography can lead to underestimates (or overestimates) of true racial disparities. If a large fraction of blacks live in the South, then adjusting for Southern residence automatically removes 1 factor (average mortality differences between Southern and non-Southern hospitals) that can explain overall racial disparities in outcomes.

Unmeasured confounding factors could bias estimates. Specifically, if patients seen in hospitals that disproportionately treated blacks experienced a higher prevalence of comorbidities not observed in the data, we would spuriously attribute elevated mortality rates to such hospitals. To examine this hypothesis, we constructed an index of disease severity, as proxied by observed comorbidities and the location of the infarct. This index was estimated with a
logistic regression that predicts 90-day mortality and included race, age, sex, all measured comorbidities, and the severity of the AMI. Differences in the severity of the comorbidities and location of the AMI generate variation in the index of predicted mortality and are presented by hospital racial decile in terms of predicted 90-day mortality. When there are no differences across hospital deciles in the comorbidity index, the role for unmeasured confounding variables to bias estimates is circumscribed, because confounding variables tend to be correlated with one another; smokers (an unmeasured variable), for example, tend to have lower incomes and are more likely to present with chronic obstructive pulmonary disease or cancer (measured variables). The finding that the measured variables are unassociated with hospital deciles therefore reduces the likelihood that the unmeasured (correlated) covariates are positively associated with hospital deciles.48

Results

Table 1 presents summary statistics for fee-for-service Medicare beneficiaries who were treated for AMI between January 1, 1997, and September 30, 2001. The table illustrates the construction of the deciles used in the analysis. The average Medicare AMI patient was treated in a hospital at which 6.9% of the patients were black. The bottom “decile” accounted for 12.5% of the population who were admitted to 1369 hospitals (comprising 32% of all hospitals) that saw no black AMI patients over the duration of the study period. These hospitals constitute decile 1 (the lowest decile) of percentage of black patients in the hospital. On the other end of the spectrum, 33.6% of patients in decile 10 hospitals were black. Patients admitted to hospitals with the highest fraction of black patients were more likely to live in the South and less likely to live in an urban setting. Differences across the deciles were significant statistically ($P<0.001$).

There was large variation in ownership status and treatment intensity between hospitals based on the extent to which they treated the black population (Table 2). Relative to the hospital at which the average AMI patient was treated, hospitals that disproportionately treat blacks were more likely to be teaching hospitals, more likely to be government (non-Federal), and less likely to be not for profit. These hospitals were similar in terms of CABG and PTCA intensity but have lower AMI volume. All differences across the deciles were highly significant statistically ($P<0.001$).

With the exception of hospitals that treated no blacks, the distribution of comorbidities and severity of the AMI across hospitals (adjusted for age, race, and sex) was similar (Figure 1). In decile 2 hospitals (where only 0.3% of patients were black), the index of predicted 90-day mortality based solely on comorbidities and severity of the AMI was 22.2% (95% CI 22.1% to 22.3%). It was 22.1% (95% CI 22.0% to 22.2%) and 22.0% (95% CI 21.9% to 22.1%) for hospitals in deciles 9 and 10, respectively. The noticeable exception to the similarity in comorbidities across deciles of percentage of black patients was seen for patients in decile 1 hospitals. These patients, all of whom were white, had predicted 90-day mortality of 23.7% (95% CI 23.6% to 23.8%), 7% higher than the expected mortality in the other deciles. Although not reported in Table 1, the elevated mortality in decile 1 was attributable largely to the elevated prevalence of renal failure (2.9% in this decile compared with 2.2% for other deciles; $P<0.001$) and a lower likelihood of being diagnosed with a subendocardial infarction (39.0% versus 49.0% for other deciles; $P<0.001$).

Figure 2 illustrates risk-adjusted 90-day mortality across hospital deciles. Hospitals that had a greater share of black AMI patients had substantially higher risk-adjusted mortality. Even though patients in decile 1 hospitals were the sickest (as measured by the index of comorbidities), they experienced the lowest risk-adjusted mortality after AMI. Figure 2 presents results from 2 models. In the first, outcomes were adjusted for age, race, sex, and comorbidities. In the second, we further adjusted for income, hospital ownership, region, and treatment characteristics. The 2 models yielded similar results, which suggests that the hospital characteristics and income were not significant explanatory variables once we

<table>
<thead>
<tr>
<th>Deciles of Percentage of Black Patients in Hospital</th>
<th>Average Percentage of Patients Who Are Black, %</th>
<th>Range (Minimum–Maximum), %</th>
<th>No. of Hospitals</th>
<th>Percent of Patients in an Urban Area, %</th>
<th>Percent of Patients in the South, %</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lowest decile</td>
<td>0.0</td>
<td>0–0</td>
<td>1369</td>
<td>24.0</td>
<td>12.5</td>
<td>142,666</td>
</tr>
<tr>
<td>2nd</td>
<td>0.3</td>
<td>0.0–0.4</td>
<td>162</td>
<td>40.1</td>
<td>9.7</td>
<td>84,971</td>
</tr>
<tr>
<td>3rd</td>
<td>0.7</td>
<td>0.4–0.9</td>
<td>276</td>
<td>47.8</td>
<td>21.3</td>
<td>113,853</td>
</tr>
<tr>
<td>4th</td>
<td>1.4</td>
<td>0.9–1.8</td>
<td>342</td>
<td>43.9</td>
<td>29.8</td>
<td>113,526</td>
</tr>
<tr>
<td>5th</td>
<td>2.2</td>
<td>1.8–2.8</td>
<td>287</td>
<td>48.4</td>
<td>36.9</td>
<td>113,769</td>
</tr>
<tr>
<td>6th</td>
<td>3.4</td>
<td>2.8–4.2</td>
<td>291</td>
<td>43.9</td>
<td>38.3</td>
<td>112,265</td>
</tr>
<tr>
<td>7th</td>
<td>5.2</td>
<td>4.2–6.4</td>
<td>326</td>
<td>41.2</td>
<td>39.9</td>
<td>114,056</td>
</tr>
<tr>
<td>8th</td>
<td>8.2</td>
<td>6.4–10.4</td>
<td>337</td>
<td>43.6</td>
<td>51.2</td>
<td>114,348</td>
</tr>
<tr>
<td>9th</td>
<td>14.2</td>
<td>10.4–19.4</td>
<td>358</td>
<td>43.8</td>
<td>71.6</td>
<td>114,686</td>
</tr>
<tr>
<td>Highest decile</td>
<td>33.6</td>
<td>19.5–98.6</td>
<td>541</td>
<td>35.2</td>
<td>68.9</td>
<td>115,596</td>
</tr>
<tr>
<td>Total</td>
<td>6.9</td>
<td>0.0–98.6</td>
<td>4289</td>
<td>40.8</td>
<td>38.0</td>
<td>1,136,736</td>
</tr>
</tbody>
</table>

Based on AMI index events from January 1, 1997, to September 30, 2001, for beneficiaries enrolled in fee-for-service Medicare. In calculating averages, each hospital is weighted by the number of patients treated over the study period. Urban areas are defined as counties that are classified as being part of a Metropolitan Statistical Area. States are classified as being in the South or West using US Census Bureau definitions for these regions. All differences across deciles are jointly significant at the $P<0.001$ level.
had adjusted for comorbidities. The area under the receiver operator curve was 0.679 for the first model and 0.681 for the second.

Figure 3 presents estimated adjusted mortality separately by race. Because of the small number of black AMI patients in deciles 2 to 6, these deciles were combined to improve statistical power in estimating race-specific adjusted mortality. Estimated mortality for blacks in decile 10 hospitals was significantly higher than for decile 2 to 6 hospitals ($P<0.04$). The difference between black and white adjusted mortality rates was not significant within each hospital decile, but a joint test of significance rejected the null hypothesis of equality ($P<0.001$). For the logistic regressions estimated separately for white and black AMI patients, the mortality gradient (by fraction of black admissions to the hospital) was significant for both white ($P<0.001$) and black ($P=0.007$) patients.

**Table 2.** Hospital Ownership Characteristics and Hospital Treatment Characteristics by Average Percentage of Black AMI Patients in the Admitting Hospital

<table>
<thead>
<tr>
<th>Percentage of Black Patients in Hospital (Deciles)</th>
<th>Hospital Teaching and Ownership Status, %</th>
<th>Average CABG Rate After AMI, %</th>
<th>Average PTCA Rate After AMI, %</th>
<th>Annual AMI Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Teaching</td>
<td>Government Non-Federal</td>
<td>Not For Profit</td>
<td>For Profit</td>
</tr>
<tr>
<td>Lowest</td>
<td>3</td>
<td>18</td>
<td>76</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>6</td>
<td>91</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>7</td>
<td>77</td>
<td>15</td>
</tr>
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<td>4</td>
<td>8</td>
<td>9</td>
<td>82</td>
<td>9</td>
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<tr>
<td>5</td>
<td>16</td>
<td>8</td>
<td>81</td>
<td>11</td>
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<tr>
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<td>15</td>
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<td>83</td>
<td>11</td>
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<td>7</td>
<td>23</td>
<td>8</td>
<td>80</td>
<td>13</td>
</tr>
<tr>
<td>8</td>
<td>28</td>
<td>14</td>
<td>74</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>20</td>
<td>17</td>
<td>70</td>
<td>13</td>
</tr>
<tr>
<td>Highest</td>
<td>30</td>
<td>17</td>
<td>70</td>
<td>13</td>
</tr>
<tr>
<td>Average</td>
<td>16</td>
<td>11</td>
<td>78</td>
<td>11</td>
</tr>
</tbody>
</table>

Based on AMI index events from January 1, 1997, to September 30, 2001, for beneficiaries enrolled in fee-for-service Medicare. Each hospital is weighted by the number of patients treated over the study period. Annual AMI Volume is the average number of patients aged at least 65 years in the Medicare program admitted to the hospital for AMI. All differences across deciles are jointly significant at the $P<0.001$ level.

Figure 1. Index of comorbidity and AMI severity by average percentage of black AMI patients admitted to the hospital. The graph reports the average index of comorbidity and AMI severity by hospital decile according to the average percentage of black AMI patients admitted to that hospital. Multiple indicators for severity were used: the presence of vascular disease, pulmonary disease, dementia, diabetes, renal failure, or cancer, and the location of the infarct (anterior, inferior, subendocardial, or other). These indicators were combined into 1 index with the coefficients from a prediction model for 90-day mortality used as weights. Thus, the index predicts 90-day mortality based on comorbidities and severity of the AMI, after adjustment for age, gender, and race. This index was intended to test the hypothesis that AMI patients are sicker in hospitals that disproportionately admit blacks. The graph indicates that this hypothesis was rejected; indeed those patients admitted to the lowest decile (no black admissions) experienced elevated risk factors.
We obtained similar results regarding the association between hospital deciles and mortality using 30-day mortality. In these models, which also adjusted for age, race, gender, comorbidities, hospital teaching status, region, ownership, and treatment intensity, 30-day mortality in decile 1 hospitals was 14.9% (95% CI 14.6% to 15.2%), in decile 2 15.6% (95% CI 15.2% to 16.1%), and in decile 10 17.6% (95% CI 17.2% to 18.0%). With these estimates, hospitals in decile 10 experienced 18% higher mortality relative to decile 1 hospitals; however, 30-day mortality within hospitals was not significantly higher among black patients.

Any potential burdens of higher mortality risk in hospitals that serve blacks was borne disproportionately by black patients, because a large fraction of this population was seen in the hospitals that comprised decile 9 and decile 10 hospitals. As Figure 4 shows, nearly half of blacks were seen in decile 10 hospitals, which were those with among the highest risk-adjusted mortality. Sixty-nine percent of black patients were seen in the 21% of hospitals that constituted decile 9 and 10 hospitals.

**Discussion**

Risk-adjusted mortality after AMI was significantly higher in hospitals that disproportionately served blacks, and this result held even after adjustment for a variety of potential confounding factors. The results of the present study may appear inconsistent with those in the study by Kahn et al., who found that blacks with a variety of clinical conditions were more likely to be admitted to higher-quality urban teaching hospitals. However, their study used a different time period (1981–1986), and their sample was limited to 5 states. More recently, several studies have noted that black patients are treated by physicians with less clinical training, referred to lower-quality cardiac surgeons, and treated at hospitals with higher risk-adjusted surgical mortality. Other studies have also found a negative association between the fraction of blacks admitted to the hospital and the use of emerging medical technologies and favorable birth outcomes.

Within hospitals, 90-day mortality rates for blacks were somewhat higher than for whites. These results contrast with most studies using data from earlier periods that generally have not found elevated mortality risks among black AMI patients. More recent studies, however, have
found higher rates of mortality and functional disability among black AMI patients.\textsuperscript{18,19}

The most important limitation of the present study is the possibility that the unobservable health status of AMI patients in neighborhoods served by hospitals with a disproportionate number of black AMI admissions is systematically different from the average. If so, the higher mortality rates observed in these hospitals could be the result of unmeasured confounding factors, rather than hospital performance per se. One obvious difference across hospital deciles is simply that there are more black patients in the higher deciles, and if they are systematically sicker, conditional on covariates, then the estimates could be biased. However, even if outcomes are measured using white mortality rates or black mortality rates separately, a significant mortality gradient is obtained.

Another limitation arises if risk adjustment does not adjust adequately for underlying illness. If the categorical comorbidity variables do not measure the severity of the disease (for example, if diabetes is more severe among black AMI patients in decile 2 hospitals than among black AMI patients in decile 10 hospitals), then the results could be biased. It could also be the case that unmeasured confounding factors (for example, smoking or exercise behavior) play a role in the elevated rates of mortality in the high-decile hospitals. But the role for unmeasured confounding factors is constrained. For unmeasured confounding factors to bias the results, they would need to be unassociated with the measured confounders, which, as shown in Figure 1, explain none of the observed mortality gradient.

In addition, hospitals serving a disproportionate number of black AMI patients tend to be located in low-income neighborhoods, so the race variable could reflect socioeconomic status. A parallel analysis (not reported) assigning hospitals to deciles of zip code income (rather than deciles of the proportion of black AMI patients) failed to show any consistent patterns: risk-adjusted mortality in decile 10 (high-income) hospitals was not significantly different from risk-adjusted mortality in decile 1 (low-income) hospitals. Similarly, accounting for the broad region of residence or urban status did not alter the results of the present study.

Why is risk-adjusted mortality, for both blacks and whites, associated with a higher fraction of black hospital admissions? One hypothesis is that black AMI patients are more likely to be admitted to hospitals with lower volume, and lower volume has predicted worse outcomes in other studies.\textsuperscript{44–46} Similarly, it could be the case that overall revascularization rates in hospitals with a large fraction of black AMI patients could be lower, as suggested by previous research documenting racial gaps in surgical treatment of cardiovascular disease.\textsuperscript{3–19,43} These explanations alone cannot explain the gradient, because the regression analysis adjusted for such factors. A plausible explanation is that differences in hospital-level quality not adequately adjusted for in the present analysis but highlighted in recent studies, such as time to reperfusion, the prescription of β-blockers, postsurgical mortality, or the quality of physicians, could explain observed differences in outcomes.\textsuperscript{28,30–32,55,56}

Another limitation of the study is that it does not address racial disparities that take place within the hospital because of differences in the use of effective treatments or lack of communication between black patients and a largely white clinical staff. For example, Barnato et al\textsuperscript{12} documented substantially lower rates of PTCA and CABG for black AMI patients even after adjusting for the hospital to which they were admitted. Statistical analysis that distinguishes between these 2 explanations—disparities within hospitals and disparities that occur because blacks go to different hospitals than whites—is therefore critical for future research on disparities.\textsuperscript{57}

The potential benefits that come from increasing quality of care are well understood.\textsuperscript{58–61} One implication of the present study is that reducing mortality rates in high-mortality hospitals can have implications for reducing racial disparities in health outcomes. Because 21% of hospitals treat 69% of elderly black AMI patients, targeting quality improvements at hospitals that disproportionately serve blacks could dramatically reduce black-white disparities in care. In addition,
because many black Medicare beneficiaries live in urban areas with more than 1 hospital, efforts to better direct patients toward high-quality hospitals may also be an effective means of reducing disparities.

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References


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