Emergency Evaluation of Chest Pain in Patients With Advanced Kidney Disease

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Background: Increased rates of myocardial infarction, heart failure, arrhythmias, and death occur in patients with chronic kidney disease. We sought to evaluate the processes of care and outcomes in patients with chronic kidney disease presenting to an emergency department with chest discomfort.

Methods: We enrolled 817 consecutive patients who underwent evaluation for a possible acute myocardial infarction in a prospective study of cardiac biomarkers. Renal dysfunction did not exclude patients from this study, and baseline renal function and 30-day outcomes were available in 808. Patients were stratified by corrected creatinine clearance rate into quartiles, with those undergoing dialysis (n=51) as a fifth comparison group.

Results: Those patients with advanced renal dysfunction (corrected creatinine clearance rate, <47.0 mL/min [<0.8 mL/s] per 72 kg) or who underwent dialysis had higher rates of diabetes, hypertension, and prior coronary disease. More than 99% of all patients were admitted to a chest pain observation unit or to the hospital. Rates of stress testing were lower as renal dysfunction worsened. Rates of revascularization, however, were similar for all groups. The most frequent in-hospital complication was the development of heart failure, which occurred in 36.5% of those with a corrected creatinine clearance rate of less than 47.0 mL/min per 72 kg. At 30 days, this group had the highest rates of cumulative myocardial infarction, development of heart failure, and death (40.2%).

Conclusion: Chronic kidney disease is a marker for in-hospital and 30-day outcomes in patients presenting to the emergency department with chest discomfort.

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cardiac death in the second group. One additional patient was
excluded because of lost data. Thus, 817 patient encounters were
studied. Of these, 808 had baseline serum creatinine levels and
30-day outcomes available for analysis. The corrected creati-
nine clearance rate (CorrCrCl) was calculated using the fol-
lowing formulas:

\[
\text{CorrCrCl}_{\text{male}} = \frac{(140 - \text{Age}_y)}{\text{Cr}} \\
\text{CorrCrCl}_{\text{female}} = 0.85 \left[ \frac{(140 - \text{Age}_y)}{\text{Cr}} \right]
\]

where Cr indicates the baseline creatinine level in milligrams
decretiller and age, the age in years.

STUDY PROTOCOL

The Institutional Review Board at Henry Ford Hospital
approved the original study. Research nurses and techni-
cians (working 24 hr/d, 7 d/wk) obtained blood samples on patient
arrival and completed a case report form. Five milliliters of hepa-
rinized blood were collected for measurement of CK-MB lev-
els and a biochemistry profile, including serum creatinine level,
in the central laboratory. The CK-MB level was measured us-
ing an immunoassay analyzer (AxSYM; Abbott Laboratories,
Abbott Park, Ill). All central laboratory results, including crea-
tinine levels, were available to the treating clinicians. Disposi-
tion of patients and final discharge diagnoses from the hos-
pital were determined by the treating physicians.

DEFINITIONS

Acute myocardial infarction was defined as (1) at least 1 CK-MB
value above the upper reference limit (9 ng/mL) as measured
in the central laboratory during the 9-hour test period, and (2)
independent agreement between 2 cardiologists (P.A.M. and
J.M.) that an AMI had occurred after reviewing the pattern
of change of CK-MB levels and the medical record. In case of dis-
agreement, a third cardiologist adjudicated the case. The dis-
charging physician determined all other final diagnoses and
documented them in the medical record. Electrocardiograms
were interpreted by a cardiologist masked to the clinical cases
using the classification proposed by the Thrombolysis in Myo-
cardial Infarction Study Group.7

STATISTICAL ANALYSIS

Baseline characteristics are reported in means ± SD with propor-
tions and 95% confidence limits as appropriate. We per-
formed group comparisons using the \( \chi^2 \) test or 1-way analysis
of variance as appropriate. We performed multiple logistic re-
gression for the cumulative combined end point of AMI, CHF,
or death, with the highest-risk group (fourth quartile) vs all
others, controlling for significant risk factors found in the uni-
vatiate analysis. A 2-sided \( \alpha \) level of .05 was used for deter-
mining significance.

RESULTS

PATIENT CHARACTERISTICS

The demographics and medical histories of the study popu-
lation are given in Table 1. The overall mean age of the study
population was 63.8 ± 16.0 years; 54.3% were women;
81.3% were African American; 15.7% were white; and
3.0% were of other races. We found trends for higher rates
of diabetes, hypertension, and prior coronary artery dis-
dease (CAD) in patients with worsened renal dysfunction.
However, we also found lower rates of smoking and fam-

ily histories of premature CAD in patients with CKD. Consist-
ent with higher rates of CAD across the renal strata,
we also found higher rates of expected long-term medica-
tion use, including aspirin and \( \beta \)-blockers. Only 26 pa-
tients (3.2%) were in the predialysis CorrCrCl range of less
than 20 mL/min (<0.3 mL/s) per 72 kg.

PROCESSES OF CARE

Table 2 highlights the initial ECG findings and pro-
cesses of care by the groups studied. Only 2.0% of pa-
tients with ESRD compared with 25.4% of those with nor-
mal renal function (CorrCrCl, >99.4 mL/min [>1.7 mL/s])
per 72 kg) had normal ECG findings. Those with normal
ECG findings tended to be triaged to chest pain units. We
found no significant differences in the rates of ischemic
ST- or T-wave changes among the groups. However, we
found a trend for more left ventricular hypertrophy (\( P = .05 \)),
right bundle-branch block (\( P < .001 \)), and left bundle-
branch block (\( P = .04 \)) in worsening levels of CKD. Over-
all, some form of stress testing was performed in 29.7%
of patients. Decisively fewer regular stress tests and stress
echocardiograms were ordered in patients with advanced
CKD; however, we found no increase in other forms of
testing, including nuclear or pharmacological stress tests.
Overall, only 15.7% of patients with ESRD, compared with
39.7% of patients with normal renal function, underwent
some form of a stress test (\( P < .001 \)). Despite these differ-
ences in stress testing rates, the rates of coronary angiog-
raphy and revascularization were similar across CKD
groups. Overall, more than 99% of patients were admit-
ted to a chest pain observation unit or to a hospital bed.
Patients with worsened CKD tended to be admitted di-
tectly to a telemetry or coronary care unit, with only 13.7%
of patients with ESRD observed in the chest pain unit.

CARDIOVASCULAR OUTCOMES

The most frequent complication in all comparison groups
was CHF, which occurred at the highest rate (36.5%) in
the fourth quartile (Figure 1). Thirty-day outcomes tracked
somewhat differently as depicted in Figure 2. These out-
comes were dominated by all-cause hospitalization, which
occurred in more than 25% of those with ESRD (\( P < .001 \)
for trend). The rates for combined in-hospital and 30-day
outcomes (cumulative rate) for the most frequent compi-
lcations of AMI, CHF, or death are shown in Figure 3. The
rate was highest in the fourth quartile, with 40.2% of all
patients in this group incurring a cardiovascular compli-
cation by 30 days. Multivariate analysis, controlled for age,
sex, diabetes, prior CAD, and aspirin use, found that the
fourth quartile incurred a 58.5% excess risk for the com-
bined cardiovascular end point at 30 days of follow-up.

CAUSES OF DEATH

Of the 29 patients who died during the hospital admis-
sion, 19 deaths (65.5%) were due to cardiovascular causes.
Of these, 11 were due to AMI, 3 were related primarily
to CHF, 4 were due to ventricular tachycardia or ven-
tricular fibrillation without AMI or CHF, and 1 was due
to aortic dissection. Of the 10 noncardiac deaths, 1 pa-
end-stage renal disease; CAD, coronary artery disease; AMI, acute myocardial infarction; PTCA, percutaneous transluminal coronary angioplasty; and CABG, coronary artery bypass grafting.

Excess toxicity of conventionally used therapies, including other established risk factors; (2) therapeutic nihilism; (3) poor outcomes: (1) uncontrolled confounding by age and the following 4 pathways by which CKD may be linked to vascular end points in a group (fourth quartile; CorrCrCl, an unspecified cause of death. The remaining patient had rhabdomyolysis. The remaining patient had an unspecified cause of death.

Table 1. Baseline Characteristics Stratified by Quartile of Corrected Creatinine Clearance*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Quartile 1 (n = 189)</th>
<th>Quartile 2 (n = 189)</th>
<th>Quartile 3 (n = 190)</th>
<th>Quartile 4 (n = 51)</th>
<th>P Value for Trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD creatinine level, mg/dL§</td>
<td>0.65 ± 0.17</td>
<td>0.87 ± 0.18</td>
<td>1.08 ± 0.22</td>
<td>2.21 ± 1.48</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mean ± SD age, y</td>
<td>47.9 ± 13.3</td>
<td>60.9 ± 12.8</td>
<td>70.9 ± 11.2</td>
<td>75.0 ± 12.4</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Men</td>
<td>71 (37.6)</td>
<td>92 (48.7)</td>
<td>93 (48.9)</td>
<td>85 (45.0)</td>
<td>28 (54.9)</td>
</tr>
<tr>
<td>Women</td>
<td>118 (62.4)</td>
<td>97 (51.3)</td>
<td>97 (51.1)</td>
<td>104 (55.0)</td>
<td>23 (45.1)</td>
</tr>
<tr>
<td>African American</td>
<td>148 (78.3)</td>
<td>144 (76.2)</td>
<td>159 (83.7)</td>
<td>162 (85.7)</td>
<td>44 (86.3)</td>
</tr>
<tr>
<td>White</td>
<td>31 (16.4)</td>
<td>38 (20.1)</td>
<td>28 (14.7)</td>
<td>24 (12.7)</td>
<td>6 (11.8)</td>
</tr>
<tr>
<td>Other race</td>
<td>10 (5.3)</td>
<td>7 (3.7)</td>
<td>3 (1.6)</td>
<td>3 (1.6)</td>
<td>1 (2.0)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>36 (19.0)</td>
<td>48 (25.4)</td>
<td>49 (25.8)</td>
<td>68 (36.0)</td>
<td>22 (43.1)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>98 (51.9)</td>
<td>130 (68.8)</td>
<td>145 (76.3)</td>
<td>150 (79.4)</td>
<td>45 (88.2)</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>41 (21.7)</td>
<td>54 (28.6)</td>
<td>57 (30.0)</td>
<td>50 (26.5)</td>
<td>12 (23.5)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>83 (43.9)</td>
<td>50 (26.5)</td>
<td>27 (14.2)</td>
<td>17 (9.0)</td>
<td>11 (21.6)</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>71 (37.6)</td>
<td>73 (38.6)</td>
<td>40 (21.1)</td>
<td>46 (24.3)</td>
<td>14 (27.5)</td>
</tr>
<tr>
<td>Prior CAD</td>
<td>42 (22.2)</td>
<td>51 (27.0)</td>
<td>67 (35.3)</td>
<td>68 (36.0)</td>
<td>25 (49.0)</td>
</tr>
<tr>
<td>Previous AMI</td>
<td>46 (24.3)</td>
<td>64 (33.9)</td>
<td>69 (36.3)</td>
<td>66 (34.9)</td>
<td>19 (37.3)</td>
</tr>
<tr>
<td>Previous PTCA</td>
<td>29 (15.3)</td>
<td>44 (23.3)</td>
<td>40 (21.1)</td>
<td>39 (20.1)</td>
<td>15 (29.4)</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>11 (5.8)</td>
<td>17 (9.0)</td>
<td>29 (15.3)</td>
<td>26 (13.8)</td>
<td>9 (17.6)</td>
</tr>
<tr>
<td>Any previous revascularization</td>
<td>32 (16.9)</td>
<td>49 (25.9)</td>
<td>54 (28.4)</td>
<td>50 (26.5)</td>
<td>17 (33.3)</td>
</tr>
<tr>
<td>Aspirin use</td>
<td>54 (28.6)</td>
<td>70 (37.0)</td>
<td>78 (41.1)</td>
<td>79 (41.8)</td>
<td>27 (52.9)</td>
</tr>
<tr>
<td>β-Blocker use</td>
<td>33 (17.5)</td>
<td>33 (17.5)</td>
<td>53 (27.9)</td>
<td>48 (25.4)</td>
<td>16 (31.4)</td>
</tr>
<tr>
<td>Statin use</td>
<td>22 (11.6)</td>
<td>33 (17.5)</td>
<td>40 (21.1)</td>
<td>37 (19.6)</td>
<td>6 (11.8)</td>
</tr>
<tr>
<td>Calcium channel blocker use</td>
<td>34 (18.0)</td>
<td>46 (24.3)</td>
<td>42 (22.1)</td>
<td>62 (32.8)</td>
<td>20 (39.2)</td>
</tr>
<tr>
<td>Oral hypoglycemic use</td>
<td>22 (11.6)</td>
<td>25 (13.2)</td>
<td>28 (14.7)</td>
<td>25 (13.2)</td>
<td>6 (11.8)</td>
</tr>
<tr>
<td>Insulin use</td>
<td>19 (10.1)</td>
<td>24 (12.7)</td>
<td>23 (12.1)</td>
<td>40 (21.2)</td>
<td>13 (25.5)</td>
</tr>
</tbody>
</table>

*Unless otherwise indicated, data are given as number (percentage) of patients. Percentages have been rounded and may not sum to 100. ESRD indicates end-stage renal disease; CAD, coronary artery disease; AMI, acute myocardial infarction; PTCA, percutaneous transluminal coronary angioplasty; and CABG, coronary artery bypass grafting.
†To convert renal dysfunction to milliliters per second, multiply by 0.0167.
‡Indicates patients were undergoing dialysis.
§To convert creatinine level to micromoles per liter, multiply by 88.4.

Our study has demonstrated a very high rate of cardiovascular end points in a group (fourth quartile; CorrCrCl, <.47.0 mL/min [<.8 mL/s] per 72 kg) presenting to the ED with chest discomfort. This group had a mean age of 75 years, 36.0% rate of diabetes, and mean serum creatinine level of 2.21 mg/dL (195.4 µmol/L); 55.0% were women and 85.7% were African American. Fewer than one third of these patients underwent some form of stress testing or cardiac catheterization. Thus, after hospitalization, these patients underwent conservative medical therapy and were ultimately discharged to home a 40.2% composite end point at 30 days. Patients with ESRD, on the other hand, had the highest individual rates of all-cause rehospitalization (25.5%) and all-cause deaths (5.9%) at 30 days. Most deaths (65.5%) were due to cardiovascular causes, in particular, AMI.

Another study1 from our group previously proposed the following 4 pathways by which CKD may be linked to poor outcomes: (1) uncontrolled confounding by age and other established risk factors; (2) therapeutic nihilism; (3) excess toxicity of conventionally used therapies, including medication-related adverse events; and (4) the special biology of the CKD state with respect to accelerated atherosclerosis, the development of CHF, and arrhythmias. In addition, the simultaneous nature of accelerated atherogenesis, thrombosis, and chronic decreases in cardiac, renal, and cerebral perfusion works to create a state that is quite susceptible to injury due to hemodynamic instability (hypertension or hypotension). We believe we have demonstrated at least the first 2 mechanisms at play. Our univariate and multivariate analyses support the fact that these data are heavily confounded by age. There was a 27.1-year difference between quartiles 1 and 4, and hence, a considerable amount of the variation in processes of care and outcomes could be accounted for by age alone. Most of the older patients with more advanced renal dysfunction had stopped smoking and were using insulin if they were diabetic. However, despite the older age and greater comorbidities, less diagnostic testing occurred in the fourth quartile and there was a very low rate (3.9%) of attempted revascularization. Taken together, we believe these data represent some degree of therapeutic nihilism or at least a strong trend for conservative medical management in this group, which may be the most salvageable with respect to efforts to preserve cardiac and renal function. Unfortunately, the original study design did not call for collection of details on the use of in-hospital and discharge medications or their potential adverse effects. Therefore, we cannot make inferences about
We might infer, however, that there is room for improvement in the diagnostic evaluation and management of chest pain in these patients with a \( \text{CorrCrCI} \lt 47.0 \text{ mL/min per 72 kg} \) (mean creatinine level, 2.21 mg/dL [195.4 µmol/L]), given a rehospitalization rate of 14.0% and composite end point of AMI, CHF, or death of 40.2% at 30 days. We acknowledge that this conclusion should be tempered by the fact that our study included a large proportion of African American patients, and may not be completely transposable to other US populations.

Our findings support some potentially new thinking about the evaluation and management of chest pain in patients with CKD. Renal dysfunction appears to be a clinical marker or warning sign for high event rates. Patients with CKD appear to get a bad deal from the health care system. It is sufficiently unclear why this is so. Perhaps the clinical distraction of being older and sicker and the reluctance to use medical and interventional strategies with proven benefits in healthier populations are at the root of the problem. It is reasonable to think that increased triage to intensive care units, higher rates of evaluation for CAD, higher rates of cardioprotective medication use, and possibly higher rates of revascularization may lead to im-

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**Table 2. Electrocardiographic Findings, Stress Testing, Invasive Management, and Ultimate Disposition Stratified by Level of Renal Dysfunction**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Quartile 1 ( \geq 99.4 ) (n = 51)</th>
<th>Quartile 2 99.3-72.8 (n = 189)</th>
<th>Quartile 3 72.7-47.0 (n = 190)</th>
<th>Quartile 4 (&lt; 47.0 ) (n = 189)</th>
<th>ESRD‡ (n = 51)</th>
<th>( P ) Value for Trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG findings</td>
<td>Normal 48 (25.4) 32 (16.9) 12 (6.3) 10 (5.3) 1 (2.0) &lt;.001</td>
<td>ST elevation 16 (8.5) 15 (7.9) 13 (6.8) 7 (3.7) 3 (5.9) .08</td>
<td>ST depression 18 (9.5) 15 (7.9) 19 (10.0) 18 (9.5) 3 (5.9) .84</td>
<td>Non-specific ST-T changes 20 (10.6) 14 (7.4) 21 (11.1) 19 (10.1) 5 (9.8) .85</td>
<td>LVH 35 (18.5) 44 (23.3) 55 (28.9) 57 (30.2) 15 (29.4) .05</td>
<td>RBBB 2 (1.1) 8 (4.2) 11 (5.8) 17 (9.0) 4 (7.8) &lt;.001</td>
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<tr>
<td></td>
<td>32 (16.9) 12 (6.3) 10 (5.3) 1 (2.0) &lt;.001</td>
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<tr>
<td>Stress testing</td>
<td>Exercise stress without imaging 10 (5.3) 10 (5.3) 1 (0.5) 1 (0.5) 0 . .</td>
<td>Exercise stress with imaging 20 (10.6) 29 (15.3) 27 (14.2) 25 (13.2) 7 (13.7) .82</td>
<td>Pharmacological stress with imaging 20 (10.6) 29 (15.3) 27 (14.2) 25 (13.2) 7 (13.7) .82</td>
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<tr>
<td></td>
<td>Coronary angiogram 28 (14.8) 35 (18.5) 26 (13.7) 19 (10.1) 1 (2.0) &lt;.001</td>
<td>PTCA 9 (4.8) 9 (4.8) 9 (4.7) 3 (1.6) 2 (3.9) .54</td>
<td>CABG 4 (2.1) 2 (1.1) 1 (0.5) 2 (1.1) 0 . .</td>
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<td></td>
<td>Coronary angiogram 28 (14.8) 35 (18.5) 26 (13.7) 19 (10.1) 1 (2.0) &lt;.001</td>
<td>PTCA 9 (4.8) 9 (4.8) 9 (4.7) 3 (1.6) 2 (3.9) .54</td>
<td>CABG 4 (2.1) 2 (1.1) 1 (0.5) 2 (1.1) 0 . .</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disposition from ED</td>
<td>Home 1 (0.5) 2 (1.1) 2 (1.1) 1 (0.5) 0 . .</td>
<td>Chest pain unit 93 (49.2) 60 (31.7) 59 (31.1) 38 (20.1) 7 (13.7) &lt;.001</td>
<td>Telemetry unit 66 (34.9) 96 (50.8) 93 (48.9) 89 (47.1) 28 (54.9) .01</td>
<td>Coronary care unit 19 (10.1) 16 (8.5) 19 (10.0) 29 (15.3) 10 (19.6) .02</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Home 1 (0.5) 2 (1.1) 2 (1.1) 1 (0.5) 0 . .</td>
<td>Chest pain unit 93 (49.2) 60 (31.7) 59 (31.1) 38 (20.1) 7 (13.7) &lt;.001</td>
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<td>Coronary care unit 19 (10.1) 16 (8.5) 19 (10.0) 29 (15.3) 10 (19.6) .02</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Other hospital bed 10 (5.3) 15 (7.9) 17 (8.9) 32 (16.9) 6 (11.8) &lt;.001</td>
<td>Other hospital bed 10 (5.3) 15 (7.9) 17 (8.9) 32 (16.9) 6 (11.8) &lt;.001</td>
<td>Other hospital bed 10 (5.3) 15 (7.9) 17 (8.9) 32 (16.9) 6 (11.8) &lt;.001</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Data are given as number (percentage) of patients. ECG indicates electrocardiographic; LVH, left ventricular hypertrophy; RBBB, right bundle-branch block; LBBB, left bundle-branch block; ED, emergency department; and ellipses, not calculated. Other abbreviations are explained in the first footnote to Table 1.

†To convert to milliliters per second, multiply by 0.0167.

‡Indicates patients were undergoing dialysis.

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**Figure 1.** In-hospital outcomes stratified by renal dysfunction quartile (Q) in the development of congestive heart failure (CHF) (\( P \).001 for trend across Qs 1-4). To convert renal dysfunction values to milliliters per second, multiply by 0.0167. AMI indicates acute myocardial infarction; VT/VF, ventricular tachycardia/ventricular fibrillation; and ESRD, end-stage renal disease.

**Figure 2.** Thirty-day outcomes by renal dysfunction group (\( P \).001 for trend in the rehospitalization; \( P \).05 for recurrent AMI and death). To convert renal dysfunction values to milliliters per second, multiply by 0.0167. Abbreviations are explained in the legend to Figure 1.
proved outcomes over time. These scenarios, however, have to be tempered by the well-understood risks for acute renal failure after coronary angioplasty or coronary artery bypass surgery in this group.19 However, for patients in this group, it seems prudent to have systems in place for close, early follow-up with additional diagnostic testing and medical management on an outpatient basis.10

The results of our study are consistent with a variety of information sources on cardiorenal risk. The recently reported Heart Outcomes Protection Trial demonstrated incrementally higher rates of AMI and death across the strata of baseline serum creatinine levels.11 Perhaps these rates of events were augmented by less vigilant evaluations for chest pain in subjects who reported to the ED with chest discomfort. Although current guidelines for the evaluation of a possible acute coronary syndrome list age greater than 70 years and diabetes as intermediate factors, they do not speak to the risk for CKD that we have observed in this study.12 In patients with ESRD, Herzog and coworkers12 have demonstrated high rates of all-cause mortality after AMI. Perhaps these high rates are due in part to reduced rates of cardioprotective medical therapy, diagnostic testing, and revascularization. Beattie and coworkers have recently published an analysis of 1724 patients with ST-segment elevation AMI, and have demonstrated reduced rates of thrombolysis and primary angioplasty in the patients at the highest CKD risk, including ESRD.

Similar to many retrospective analyses performed on prospectively collected data sets, our study has several limitations. We did not have important information on prior diagnostic evaluations performed. Previous stress tests and angiograms or a well-understood history of CHF might have played into the clinical decision not to order a diagnostic test during the hospitalization we studied. In addition, we did not have data on the rates of cardioprotective medications used by patients during the hospitalization and at discharge. Differential rates of use of aspirin, β-blockers, statins, and angiotensin-converting enzyme inhibitors might have accounted for differences in the composite end point. If so, it makes an even stronger case for more aggressive therapeutic measures in those who are elderly and have impaired renal function. Our division of subjects on the basis of CorrCrCl yielded small subgroups; hence there was some instability of the point estimates and lack of robustness owing to missing data. In addition, CorrCrCl is a crude population tool that overestimates renal function in those who weigh less than 72 kg, and, conversely, underestimates it in those who weigh more than 72 kg. Use of more exact calculations, including the Cockcroft-Gault prediction13 or Modification of Diet in Renal Disease equation,14 would have been desirable if weight was in the database. Also, we did not have serum albumin levels measured in all cases and cannot comment on this important risk factor for death in patients with CKD.

**CONCLUSIONS**

Despite high rates of admission and observation, we found differential rates of diagnostic testing and outcomes when patients with chest pain were stratified by baseline renal function. Renal dysfunction appears to be a clinical signal for high combined end points at 30 days, and hence these patients constitute a high-risk chest pain population.

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