Coronary Interventions

Association Between Acute Kidney Injury and In-Hospital Mortality in Patients Undergoing Percutaneous Coronary Interventions

Judith Kooiman, MSc; Milan Seth, MS; Brahmajee K. Nallamothu, MD; Michael Heung, MD; David Humes, MD; Hitinder S. Gurm, MD

Background—Acute kidney injury (AKI) post percutaneous coronary intervention (PCI) is associated with increased mortality but both death and AKI share common risk factors. Moreover, the effect of a high contrast dose, a known modifiable risk factor for AKI, on mortality is unknown. The aim of our study was to analyze the association between AKI and in-hospital mortality post PCI after adjustment for confounding by common risk factors.

Methods and Results—This study was performed using a regional registry of all patients undergoing PCI in Michigan. Primary end points were AKI (serum creatinine increase >0.5 mg/dL) and all-cause in-hospital mortality. Propensity matching was performed, with each AKI patient matched to 4 controls. Attributable risk fraction and the exposed index number of AKI for mortality were calculated within the propensity-matched cohort. Between 2010 and 2013, 92,317 patients underwent PCI, of whom 2141 (2.3%) developed AKI. We matched 1371/2141 patients with AKI to 5484 controls. AKI was strongly associated with mortality (odds ratio=12.52, 95% confidence interval 9.29–16.86) in the propensity-matched cohort. The attributable risk fraction for mortality of AKI was 31.4% (95% confidence interval 26.8%–37.5%), and one death could be prevented for every 9 cases of AKI successfully avoided. The independent impact of a high contrast dose at time of PCI on in-hospital mortality risk was weak (adjusted odds ratio 1.19, 95% confidence interval 0.97–1.45).

Conclusions—Nearly one-third of the in-hospital mortality post PCI is attributable to AKI. Preventing 9 cases of AKI could potentially prevent one death. These study findings stress the need for developing effective AKI preventive strategies beyond minimization of contrast dose. (Circ Cardiovasc Interv. 2015;8:e002212. DOI: 10.1161/CIRCINTERVENTIONS.114.002212.)

Key Words: contrast media ■ infarction ■ kidney ■ mortality ■ revascularization

Acute kidney injury (AKI) is a common complication of percutaneous coronary intervention (PCI) and is associated with increased morbidity, mortality, duration of hospital stay, and healthcare cost.1–3 Although AKI is generally a reversible condition, patients developing this complication post PCI may experience up to 20-fold increase in their in-hospital mortality risk.4 Interpretation of results reported by previous studies on the association between AKI and mortality post PCI is hampered by the fact that both outcomes share common risk factors, such as hemodynamic instability, heart failure, diabetes mellitus, and preexisting chronic kidney disease. It therefore remains unclear whether the occurrence of AKI is independently associated with mortality and if the mortality risk associated with AKI is clinically relevant in relation to the broader population of patients undergoing PCI. Our study seeks to address this gap in knowledge by studying the association between AKI and in-hospital mortality after adjustment for confounding by common risk factors among a large population undergoing contemporary PCI. Second, we analyzed the attributable risk fraction of AKI-related in-hospital mortality.

Methods
This study was performed using data from the Blue Cross Blue Shield of Michigan cardiovascular consortium (BMC2), a regional registry of all patients undergoing PCI at nonfederal hospitals in Michigan. A detailed outline of the BMC2 registry has previously been described.4–7 Briefly, consecutive patients undergoing PCI at the 47 participating centers were included. Data on procedural and demographic patient characteristics, medication, laboratory values, renal...
WHAT IS KNOWN

- Acute kidney injury (AKI) is a common complication of percutaneous coronary intervention (PCI) and is associated with increased morbidity and mortality.
- Understanding the association between AKI and mortality post PCI is hampered by common risk factors for these outcomes, such as diabetes mellitus and congestive heart failure.

WHAT THE STUDY ADDS

- Our analysis demonstrates that nearly one-third of the in-hospital mortality risk post PCI is attributable to AKI.
- Avoiding nine cases of AKI post PCI could potentially save one life.
- Although the use of a high contrast dose at time of PCI significantly increases the risk of AKI, contrast dosing is only a minor contributor to the overall burden of AKI in this population.

Propensity-Matching Methods

Propensity-matching was used to select a population from which to estimate the exposure impact number (EIN) for AKI-related in-hospital mortality. A logistic regression model was used to estimate the propensity to develop AKI based on hospital, patient, and procedural characteristics and preprocedural AKI and mortality risks (Appendix Table I in the Data Supplement).

Propensity-matching was then performed using nearest neighbor–matching without replacement, with each AKI patient matched to 4 control patients using a caliper width of 0.25 of the standard deviation of the logit of the propensity score. In addition, we exact matched for presentation with ST-segment elevation myocardial infarction (STEMI), and occurrence of cardiogenic shock. Further, we used a 365-day caliper on procedure date to minimize the impact of temporal trends in outcome. Absolute standardized differences were computed to evaluate matching effectiveness and displayed graphically (Appendix Figure I in the Data Supplement); values below 10% indicate similarity with respect to baseline covariates in the cohort.

Study End Points

The primary end points of this study were AKI and all-cause in-hospital mortality. AKI was defined by an absolute increase in serum creatinine >0.5 mg/dL because this definition was demonstrated to be superior to alternate definitions in patients undergoing PCI.13,14 Post procedural adverse clinical outcomes, including CVA or stroke, myocardial infarction, major bleeding complications, and in-hospital mortality, were defined using the CathPCI Registry versions 3.0 and 4.0 data collection form definitions.

Statistical Analyses

A logistic regression model was fitted in the propensity-matched cohort with in-hospital mortality as the dependent variable and adjusting for all variables included in the match.

After observation of a statistically significant association between AKI and in-hospital mortality, estimated coefficients from the fitted model adjusting for all baseline covariates (Appendix Table I in the Data Supplement) were used to predict, based on patient-level baseline characteristics, the following: (a) the expected number of deaths in the entire cohort and (b) the expected number of deaths if none of the patients had experienced AKI post PCI. The attributable risk fraction, or the proportion of the risk of mortality attributable to AKI, was calculated as one minus the ratio of these estimates (b/a). A 95% confidence interval (CI) for the attributable risk fraction estimate was obtained using bootstrap resampling. The 95% CI was estimated by the 2.5th and 97.5th percentiles of the bootstrap attributable risk fraction estimates.

The EIN was defined as the number of individuals with the exposure among whom one excess case is because of the exposure.14 In this case, the number of cases of AKI that if eliminated would prevent one death. EIN point estimates and 95% CIs for the overall matched cohort and by subgroups were constructed from the fitted regression model described earlier.13,18

Three supplemental analyses were performed to assess the extent to which the association between AKI and in-hospital mortality and the resultant estimated proportion of mortality risk attributable fraction to AKI were explained or affected by either (a) hospital-level variability in the process or quality of care, or (b) variability in the effect of AKI on mortality risk across various baseline or procedural covariates (effect modification), or (c) the use of a high contrast dose during PCI.

The impact of hospital variability was assessed using a hierarchical generalized linear mixed effects regression model fitted to the matched cohort.19 The hierarchical model adjusted for sex, age, predicted baseline AKI and mortality risks, and eGFR as fixed effect covariates, as well as a normally distributed site-level random effect term to allow for hospital clustering, and attributable risk fraction was estimated using the fixed effect coefficients from the model as described previously.

The impact of potential effect modification was evaluated graphically using forest plots presenting the univariate association between
AKI and in-hospital mortality (odd ratios and Fisher’s exact 95% CIs) in subgroups within the propensity-matched cohort based on baseline clinical covariates, including cardiogenic shock, cardiac arrest, diabetes mellitus, heart failure, primary PCI indication, and eGFR (<30, 30–60, 60–90, >90 mL/min). For these same subgroups, likelihood ratio tests were used to test for interactions between AKI and subgroups. The attributable risk fraction was then estimated based on a regression model, including all significant interactions in addition to all other baseline covariates.

Interaction between the occurrence of post-PCI bleeding and the association between AKI and in-hospital mortality post PCI was studied in a multivariate regression analysis in the entire study cohort. Effect of administration of a high contrast dose at time of PCI on mortality risk was assessed in the entire population using a multivariate regression model adjusted for baseline covariates. High contrast dose was defined as a contrast volume (in ml) ≥3× eGFR.20

Two-sided statistical significance was defined as $P \leq 0.05$. Demographic data were described across treatment groups as mean (standard deviation) for continuous variables and number (%) for categorical variables. Univariate analysis of the association between AKI and mortality was performed using the Fisher's Exact test and CIs for the odds ratio (OR). Fisher's exact and Pearson's Chi-squared tests were used for comparisons of other categorical variables and Student t-tests for continuous variables. All statistical analyses were performed using R version 2.15.21

**Results**

Between January 2010 and June 2013, 112 687 PCI procedures were performed in 109 929 patients. In total 20 370 procedures in 19 546 patients were excluded from analysis because of the predefined exclusion criteria (Figure 1). As a result, the cohort used for analysis consisted of 92 317 (81.9%) procedures in 90 383 (82.2%) patients. The clinical, demographic, and procedural characteristics of patients in the total cohort (N=92 317) are represented in Table 1, stratified by the presence or absence of postprocedural AKI. As expected, patients developing AKI had a greater burden of comorbidity at baseline. Information on the indication for PCI is provided by Appendix Table II in the Data Supplement.

AKI occurred after 2156 (2.3%) PCI procedures in 2141 (2.4%) unique patients. As a result, 271/90 838 (0.30%) patients had a need for dialysis post PCI. Patients with AKI were more likely to have adverse in-hospital outcomes (Table 2), including in-hospital mortality, cardiogenic shock, and new-onset dialysis.

**Propensity-Matched Cohort**

We successfully matched 1371/2141 (63.6%) patients with AKI to 5484 patients without AKI possessing similar patient characteristics at baseline, with standardized differences of <10% between groups for all assessed characteristics (Table 1; Appendix Figure I in the Data Supplement). Patients included in the propensity-matched cohort generally had a higher burden of acute illness than the patients in the overall cohort. After matching, rates of postprocedural adverse clinical outcomes remained significantly higher in AKI compared with non-AKI patients (Table 2).

AKI was strongly associated with in-hospital mortality (OR=12.52, 95% CI 9.29–16.86, $P<0.0001$) after adjustment for baseline covariates in a logistic regression model fitted to the propensity-matched cohort. High baseline predicted risk of AKI (>3%), cardiogenic shock, cardiac arrest, and STEMI in AKI patients were associated with high in-hospital mortality rates (Figure 2). The estimated in-hospital mortality risk attributable fraction to AKI was 31.4% (95% CI 26.8%–37.5%). EIN estimates are provided in Figure 3 for the entire propensity-matched cohort and for subgroups defined by patient characteristics at baseline. Among matched patients with AKI, we estimate one death could be potentially prevented if 9 cases of AKI were to be successfully obviated. Within the specific subgroups, this estimate varied from <3 cases in patients developing shock post PCI to 15 among patients at low baseline AKI risk (<3%).

**Sensitivity Analyses**

The association between AKI and mortality persisted after allowing for hospital-level variability through a hierarchical generalized linear mixed effect model (OR=9.78, 95% CI 7.48–12.79, $P<0.0001$, attributable risk fraction=29.3% [95% CI 24.6%–34.9%]). In the effect modification analysis, inclusion of potential 2-way AKI by subgroup interaction terms in modeling did not materially impact the estimated
attributable risk fraction (data not shown). Additionally, multivariate regression analysis fitted to the entire cohort demonstrated a strong association between post-PCI bleeding and both AKI (OR 3.14, 95% CI 2.68–3.68) and death (OR 2.39, 95% CI 1.92–2.97). However, the occurrence of post-PCI bleeding did not significantly change the association between AKI and in-hospital mortality (P=0.35 for AKI by bleeding interaction). We also performed a sensitivity analysis using a 1:1 propensity score match allowing for replacement of controls but otherwise following the same procedure as specified. Although allowing replacement and selecting only one control match for each AKI patient succeeded in matching more high-risk AKI patients, resulting in the higher mortality rates for both groups in the matched cohort, the risk differences (and thus EIN values) were similar to the initial match (data not shown).

### Impact of High Contrast Dose

Using a contrast dose ≥3× baseline eGFR at time of PCI was associated with an increased risk of AKI (adjusted OR 1.35, 95% CI 1.19–1.53) in the entire population. High contrast dose, however, was only a marginal predictor of AKI with an estimated attributable risk fraction to AKI of a high contrast dose of 10.6% (95% CI 5.96–15.25) and an EIN of 97.9 (95% CI 69.7–164.4). Accordingly, the independent impact of a high-contrast dose at time of PCI on in-hospital mortality risk was weak (adjusted OR 1.19, 95% CI 0.97–1.45) after adjusting for other baseline clinical covariates.

### Discussion

The key finding of our study is that nearly one-third of the in-hospital mortality risk post PCI is attributable to AKI. Additionally, our data suggest that avoiding 9 cases of AKI post PCI could potentially save one life. Mortality risk associated with AKI was highest in those with cardiogenic shock, cardiac arrest, or presenting with STEMI, but was also clinically relevant in patients with a more stable presentation.

Our study findings corroborate and significantly extend prior work in this field. Previous studies have shown an association between AKI post PCI and in-hospital mortality, but were limited by collinearity because of the presence of similar risk factors. Our analytic techniques attempt to overcome...
these challenges and provide an estimate of the clinical impact of AKI and also highlight the potential life-saving impact if AKI could reliably be prevented.

AKI post PCI has several potential causes, including acute tubular necrosis from poor perfusion, nephrotoxicity from contrast media, cholesterol embolization, the use of nephrotoxic drugs, procedure-related factors, such as route of vascular access for PCI, or a combination of the above. The differentiation between these etiologies is often difficult because they all result in an increase in serum creatinine within a few days post PCI, frequently in the absence of other clinical findings suggestive of a cause of injury. The clinical significance of different underlying causes of post-PCI AKI remains uncertain because previous studies have not been able to make these distinctions. Therefore, future prospective studies should seek to differentiate among the different causes.

That said, almost all research in the prevention of AKI has been on contrast media–induced AKI, for which avoidance of high-osmolar contrast agents, renal function–based contrast dosing, and intravenous volume expansion with either saline or sodium bicarbonate are effective. However, our study results indicate that although the use of a high contrast dose at time of PCI significantly increases the risk of AKI, contrast dosing is only a minor contributor to the overall burden of AKI in this population. Thus, efforts to reduce contrast dose, although worthwhile, would only moderately impact

Table 2. In-Hospital Adverse Clinical Outcomes Post-PCI

<table>
<thead>
<tr>
<th></th>
<th>Before Propensity-Matching</th>
<th></th>
<th></th>
<th></th>
<th>After Propensity-Matching</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AKI</td>
<td>No AKI</td>
<td>OR</td>
<td>P Value, (95% CI)</td>
<td>AKI</td>
<td>No AKI</td>
<td>OR</td>
</tr>
<tr>
<td>Length of hospital stay, mean (SD), in days*</td>
<td>9.7 (8.7)</td>
<td>2.8 (7.0)</td>
<td>NA</td>
<td></td>
<td>9.0 (8.2)</td>
<td>4.8 (4.5)</td>
<td>NA</td>
</tr>
<tr>
<td>In-hospital mortality</td>
<td>450/2156 (20.9%)</td>
<td>526/2156 (24.9%)</td>
<td>44.9</td>
<td>P &lt; 0.001, (39.2–51.5)</td>
<td>189/1371 (13.8%)</td>
<td>112/1371 (8.2%)</td>
<td>7.7</td>
</tr>
<tr>
<td>Cardiogenic shock (among patients not in shock at presentation)</td>
<td>270/1759 (15.3%)</td>
<td>890/1759 (50.4%)</td>
<td>17.8</td>
<td>P &lt; 0.001, (15.4–20.7)</td>
<td>171/1290 (13.3%)</td>
<td>167/1290 (12.8%)</td>
<td>4.6</td>
</tr>
<tr>
<td>Heart failure</td>
<td>502/2156 (23.3%)</td>
<td>1574/90108 (17.1)</td>
<td>17.1</td>
<td>P &lt; 0.001, (15.2–19.1)</td>
<td>299/1371 (21.8%)</td>
<td>322/1371 (23.4%)</td>
<td>4.5</td>
</tr>
<tr>
<td>Stroke</td>
<td>44/2156 (2.0%)</td>
<td>195/90109 (9.6)</td>
<td>9.6</td>
<td>P &lt; 0.001, (6.7–13.4)</td>
<td>28/1371 (2.0%)</td>
<td>24/1371 (1.7%)</td>
<td>4.7</td>
</tr>
<tr>
<td>RBC/whole blood transfusion</td>
<td>609/2156 (28.2%)</td>
<td>1760/90109 (19.8)</td>
<td>19.8</td>
<td>P &lt; 0.001, (17.8–22.0)</td>
<td>306/1371 (22.3%)</td>
<td>428/1371 (31.4%)</td>
<td>3.4</td>
</tr>
<tr>
<td>Bleeding w/in 72 h</td>
<td>329/2153 (15.3%)</td>
<td>1892/90113 (8.4)</td>
<td>8.4</td>
<td>P &lt; 0.0001, (7.4–9.5)</td>
<td>196/1369 (14.3%)</td>
<td>278/1369 (20.1%)</td>
<td>3.1</td>
</tr>
<tr>
<td>New requirement for dialysis (N RD)</td>
<td>209/2156 (9.7%)</td>
<td>62/90113 (0.1)</td>
<td>156.5</td>
<td>P &lt; 0.001, (116.2–211.7)</td>
<td>85/1371 (6.2%)</td>
<td>19/5482 (0.4%)</td>
<td>19.0</td>
</tr>
</tbody>
</table>

Data are presented as n, %. OR represent outcomes of a crude analysis. AKI indicates acute kidney injury; CI, confidence interval; OR, odds ratio; PCI, percutaneous coronary intervention; and RBC, red blood count.

*Standard difference between AKI and non-AKI group before propensity-matching 88.3%, afterward 64.2%.

Figure 2. In-hospital mortality in the propensity-matched cohort. Risk of in-hospital mortality was calculated using the CathPCI National Cardiovascular Data Registry risk model. This clinical model includes 8 variables: age, cardiogenic shock, previous congestive heart failure, peripheral vascular disease, chronic lung disease, estimated glomerular filtration rate, New York Heart Association class IV heart failure, and PCI status. AKI indicates acute kidney injury; PCI, percutaneous coronary intervention; and STEMI, ST-segment elevation myocardial infarction.
in-hospital mortality rates. This suggests that efforts to reduce the incidence and impact of AKI need to move beyond contrast media choice and dosing and be targeted at other mechanistic pathways of AKI, such as inflammation and the potential role for statin preloading. Further, these results suggest that it would be inaccurate to invoke contrast media as the major mediator of AKI in this population and terms, such as contrast-induced nephropathy or contrast-induced-AKI, should not be used to denote all post-PCI AKI.

There are several possible mechanisms via which AKI can increase risk of death. First, AKI is frequently complicated by altered immune function, increased propensity to infection, bleeding, and thrombosis.25,26 Second, AKI can result in acute volume overload, acidemia, and hyperkalemia contributing to increased cardiac stress and risk of arrhythmias.27 Third, cross-talk between the kidney and other organs involving inflammatory and proapoptotic pathways may result in multiorgan failure eventually causing death.28,29 Fourth, AKI alters the kinetics of most pharmacotherapies used in this setting and may potentially increase the risk of toxicity. Many drugs that have been demonstrated to improve outcomes of patients with acute cardiac disease (ie, modulators of the angiotensin–aldosterone-pathway) are relatively contraindicated and typically withheld in patients with AKI.30,31 Finally, AKI alters tissue healing and may potentially interfere with myocardial recovery after infarction.32–34

Currently, patients with AKI are managed with supportive therapy because of the absence of specific strategies for established AKI. Almost all therapeutic agents that have been previously tested for this indication have failed to reach clinical practice, although some promising approaches in the prevention of AKI are being explored. These include ischemic preconditioning, use of Na/K citrate, selective adenosine A1-receptor antagonists, GLP-1 analogues (Exendin-4 or liraglutide), and losartan preloading.35–38 Future research along these lines is warranted to identify agents that can either prevent or ameliorate the clinical impact of AKI in this population.

The results of our study must be interpreted with certain caveats. First of all, despite the use of propensity-matching, the effect of residual confounding on study findings cannot be excluded. Second, the timing of AKI and other clinical outcomes during hospitalisation was not registered. It therefore remains unknown whether these complications were more common in patients with AKI as a result or were caused by the acute reduction in renal function. However, this should not impact the association between death and AKI, our primary study interest. Third, our study reflects routine clinical practice, and there was no standardization of preprocedural care, choice or volume of contrast media, or in use of adjuvant therapies. However, this reflects contemporary practice across an entire state and makes our findings more generalizable. Fourth, we were unable to adjust our analysis for in-hospital withdrawal of medication that could possibly have influenced the risk of both AKI and in-hospital mortality. Fifth, information
on the incidence of hyperkalemia and use of drugs or initiation of dialysis specifically to correct for this condition post PCI is unavailable. Furthermore, our study demonstrates an association between in-hospital development of AKI and in-hospital mortality because data on post discharge renal function or mortality were not available. Finally, the specific results of our analysis are likely contingent on the definition of AKI and use of a more sensitive definition as proposed by consensus groups, such as AKIN or RIFLE may give different results. These consensus-based definitions have not been extensively validated for their clinical impact in the post-PCI population unlike the traditional AKI definition that has been previously demonstrated to be superior to alternate definitions.\(^\text{13,14}\)

In conclusion, AKI post PCI is a major predictor of in-hospital mortality post-PCI, and nearly one-third of these deaths may be attributable to it. The association between AKI and in-hospital mortality was present in all subgroups and strata of baseline AKI-risk, although most profound in those with cardiogenic shock, cardiac arrest, or STEMI. Potentially preventing 9 cases of AKI could possibly prevent one death after PCI. Our study findings highlight the need to identify highly effective strategies for prevention and treatment of AKI in patients undergoing PCI.

Acknowledgments

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Disclosures

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References


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Data Supplement (unedited) at:
http://circinterventions.ahajournals.org/content/suppl/2015/06/08/CIRCINTERVENTIONS.114.002212.DC1

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Appendix Figure 1. Absolute standardized differences in baseline covariates before and after matching.
### Appendix Table 1. Variables used in propensity-match equation

<table>
<thead>
<tr>
<th>Patient characteristics at baseline and procedural variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Race</td>
</tr>
<tr>
<td>Height</td>
</tr>
<tr>
<td>Weight</td>
</tr>
<tr>
<td>Current/recent smoker (w/in 1 yr)</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Dyslipidemia</td>
</tr>
<tr>
<td>Family history of premature CAD</td>
</tr>
<tr>
<td>Prior MI</td>
</tr>
<tr>
<td>Prior heart failure</td>
</tr>
<tr>
<td>Prior valve surgery/procedure</td>
</tr>
<tr>
<td>Prior PCI</td>
</tr>
<tr>
<td>Prior CABG</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

### Treatment variables

| Fondaparinux | Aspirin | GP IIb/IIIa |
| Prasugrel | LMWH |
| Bivalirudin | Clopidogrel | Ticagrelor |
| UFH | Direct thrombin inhibitor (other) | Ticlopidine |

Abbreviations: CAD = coronary artery disease, MI = myocardial infarction, PCI = percutaneous coronary intervention, CAGB = coronary artery bypass graft, LV = left ventricular, STEMI = ST-elevation myocardial infarction, NSTEMI = non-ST-elevation myocardial infarction, UA = unstable angina pectoris, NCDR = national cardiovascular data registry, AKI = acute kidney injury, IABP = intra-aortic balloon pump, LAD = left anterior descending artery, TIMI = thrombolysis in myocardial infarction, LMWH = low-molecular-weight-heparin, UFH = unfractionated heparin

* Emergency department, home or nursing care, ** as performed within the last 6 months, *** STEMI stable >12 hours, STEMI unstable >12hrs, STEMI stable after successful thrombolysis, STEMI after failed full dose thrombolytics, high risk NSTEMI/unstable angina, staged PCI, or other.
**Appendix Table 2.** Pre-procedural presentation of coronary artery disease

<table>
<thead>
<tr>
<th>Category</th>
<th>Before propensity-matching</th>
<th>After propensity-matching</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AKI</td>
<td>No AKI</td>
</tr>
<tr>
<td>No symptom, no angina</td>
<td>82 (3.80%)</td>
<td>5,806 (6.44%)</td>
</tr>
<tr>
<td>Symptom unlikely to be ischemic</td>
<td>33 (1.53%)</td>
<td>2,034 (2.26%)</td>
</tr>
<tr>
<td>Stable angina</td>
<td>80 (3.71%)</td>
<td>13,718 (15.22%)</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>512 (23.75%)</td>
<td>36,673 (40.68%)</td>
</tr>
<tr>
<td>Non-STEMI</td>
<td>733 (34.00%)</td>
<td>18,445 (20.46%)</td>
</tr>
<tr>
<td>ST-Elevation MI (STEMI) or equivalent</td>
<td>716 (33.21%)</td>
<td>13,485 (14.96%)</td>
</tr>
<tr>
<td>P-value</td>
<td>( P &lt; 0.001 )</td>
<td></td>
</tr>
</tbody>
</table>
**Appendix Table 3. PCI status**

<table>
<thead>
<tr>
<th></th>
<th>Before propensity-matching</th>
<th>After propensity-matching</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective</td>
<td>261 (12.11%)</td>
<td>222 (16.19%)</td>
</tr>
<tr>
<td></td>
<td>35,501 (39.38%)</td>
<td>875 (15.96%)</td>
</tr>
<tr>
<td>Urgent</td>
<td>1,075 (49.86%)</td>
<td>775 (56.53%)</td>
</tr>
<tr>
<td></td>
<td>40,540 (44.96%)</td>
<td>3,155 (57.53%)</td>
</tr>
<tr>
<td>Emergency</td>
<td>789 (36.60%)</td>
<td>372 (27.13%)</td>
</tr>
<tr>
<td></td>
<td>14,049 (15.58%)</td>
<td>1,436 (26.19%)</td>
</tr>
<tr>
<td>Salvage</td>
<td>31 (1.44%)</td>
<td>2 (0.15%)</td>
</tr>
<tr>
<td></td>
<td>71 (0.08%)</td>
<td>18 (0.33%)</td>
</tr>
<tr>
<td>P-value</td>
<td>P&lt;0.001</td>
<td>P = 0.603</td>
</tr>
</tbody>
</table>