The Adverse Long-Term Impact of Renal Impairment in Patients Undergoing Percutaneous Coronary Intervention in the Drug-Eluting Stent Era

Clare E. Appleby, MRCP, PhD; Joan Ivanov, PhD; Shahar Lavi, MD; Karen Mackie, RN; Eric M. Horlick, MD; Douglas Ing, MD; Christopher B. Overgaard, MD; Peter H. Seidelin, MD, MB; Rūdiger von Harsdorf, MD; Vladimír Džavík, MD, FAHA

Background—An observational study determining the long-term impact of chronic kidney disease (CKD) on patients undergoing percutaneous coronary intervention at a tertiary cardiac referral center. CKD is associated with poor in-hospital outcomes after percutaneous coronary intervention, but its effect beyond 1 year, particularly in the drug-eluting stent (DES) era, has not been reported.

Methods and Results—Baseline creatinine was available for 11,953 patients entered into a prospective registry (April 2000 to September 2007). Patients were stratified: those with or without at least moderate CKD (creatinine clearance, <60 mL/min). Follow-up data were obtained through linkage to a provincial registry. Kaplan–Meier analysis was performed. Cox multiple-regression analysis identified independent predictors of late mortality and major adverse cardiac events (MACE) and examined the association between DES use and late outcomes in the presence or absence of CKD. CKD was present in 3070 patients (25.7%). In-hospital mortality and MACE were significantly increased in CKD (3.34% versus 0.44%, \(P<0.001\) and 5.73% versus 2.2%, \(P<0.001\)). Survival and MACE-free survival at 7 years were reduced (64.5±1.4% versus 89.4±0.5%, \(P<0.001\); 44.0±1.4% versus 63.4±0.8%, \(P<0.001\)). CKD was an independent predictor of late mortality and MACE (hazard ratio [HR]: 2.18, CI: 1.90 to 2.49, \(P<0.001\); HR: 1.37, CI: 1.25 to 1.49, \(P<0.0001\)). DES use was associated with a significant reduction in both (HR: 0.71, CI: 0.60 to 0.83, \(P<0.0001\); HR: 0.70, CI: 0.63 to 0.78, \(P<0.0001\)). In patients with CKD, DES use was associated with reduced revascularization (HR: 0.68, CI: 0.53 to 0.88, \(P=0.004\)) and reduced MACE (HR: 0.81, CI: 0.69 to 0.95, \(P=0.011\)) but not reduced mortality (HR: 0.85, CI: 0.69 to 1.05, \(P=0.01\)).

Conclusion—In a large registry of “all comers” for percutaneous coronary intervention, CKD was an independent predictor of adverse late outcomes. DES use may be associated with improved long-term outcomes in this high-risk cohort, but further prospective studies are required.

Key Words: coronary disease ■ stents ■ survival ■ chronic kidney disease ■ drug-eluting stents

Preexisting renal impairment (RI) or chronic kidney disease (CKD) in patients undergoing percutaneous coronary intervention (PCI) is associated with worse in-hospital outcomes and reduced procedural success, probably as a reflection of more complex and calcified anatomy.4 Mortality, myocardial infarction (MI), repeat revascularization, and bleeding complication rates are all increased after PCI compared with that of patients with normal renal function. The severity of CKD predicts clinical outcome at 1 year, with adverse clinical events occurring despite routine stenting and use of glycoprotein IIb/IIIa inhibitors.6 However, the effect of RI on late outcomes, particularly in the era of drug-eluting stent (DES) use has not been reported.
need for repeat revascularization remains high, although the restenosis rate, in mild to moderate CKD, is not increased at 9 months. Medium and long-term outcomes are improved compared with balloon angioplasty alone. Although patients with CKD have usually been excluded from DES trials, registry data suggest that the benefits associated with their use may extend to this population. Retrospective analyses have demonstrated that, in the intermediate term, despite a higher overall mortality and morbidity after PCI, patients with end-stage renal disease as well as those with milder degrees of CKD benefit from lower rates of target vessel revascularization with DES versus BMS placement.

Therefore, we sought to investigate the long-term clinical outcomes of patients with CKD undergoing PCI at a large, high-volume, tertiary center. The objective was to determine, through the use of our prospective PCI registry database, the impact of moderate to severe CKD on the late outcomes of unselected patients in modern routine PCI practice.

Methods

The University Health Network PCI Registry

All patients undergoing PCI at the Peter Munk Cardiac Centre in Toronto, Canada, from clinical, angiographic, procedural, and outcome variables collected prospectively and entered into a data registry by dedicated specially trained nursing personnel, as described previously. All patients undergoing their first PCI procedure between April 2000 and September 2007, with available baseline creatinine and weight, were included in the current study. Estimated creatinine clearance (CrCl) was calculated using the Cockroft-Gault formula: 

\[
\text{CrCl} (\text{mL/min}) = \left( \frac{140 - \text{age} \times \text{weight} \, [\text{kg}]}{\text{serum creatinine} \, [\text{mg/dL}] \times 72} \right) \times 0.85 \times 1000
\]

A CrCl < 60 mL/min, consistent with CKD class \(\geq 3\), was considered at least moderate RI. DES use is at the discretion of the operator and is generally reserved for bifurcations or 2 of the following: diabetics, small vessels, or long lesions. Clopidogrel was given as indicated by guidelines current at the time; loading was 300 to 600 mg, maintenance for 6 weeks to 12 months dependent on stent type and clinical indication.

In-Hospital Outcomes and Definitions

In-hospital mortality and morbidity rates were calculated for each cohort. Complications were defined according to the American College of Cardiology/American Heart Association cardiac catheterization standards. The definition of peri-procedural MI was more inclusive than that of the American College of Cardiology/American Heart Association criteria, including all patients with creatine kinase elevations \(\geq 2\) times the upper limit of normal. Indications for PCI were defined as follows: emergent PCI included primary, facilitated, or rescue PCI (failure to reperfuse or reinfarction after thrombolytic therapy in the setting of ST-elevation MI); urgent acute coronary syndrome PCI referred to any procedure for non-ST-segment elevation acute coronary syndromes or \(\geq 48\) hours after ST-elevation MI; salvage PCI was defined as no surgical revascularization option. Lesion type (A/B1/B2/C) was defined according to the American College of Cardiology/American Heart Association classification.

Late Outcomes

Long-term follow-up data were obtained through linkage of the clinical database to the Discharge Abstract Database of the Canadian Institute for Health Information and the Registered Persons Database. All time-to-event analyses were conducted at the Institute for Clinical Evaluative Sciences, Ontario. The Institute for Clinical Evaluative Sciences is supported by an operating grant from the Ontario Ministry of Health and Long-Term Care. The results and conclusion are those of the authors and should not be attributed to the Institute for Clinical Evaluative Sciences or the Ontario Ministry of Health and Long-Term Care.

Data were available on time to event for all-cause mortality, repeat PCI, or subsequent CABG, as well as hospitalization for MI, heart failure, or stroke (defined by primary hospital coding for admission). Late major adverse cardiac events (MACE) were defined as death, repeat PCI, or CABG; and cardiovascular event-free survival was defined as freedom from death or any readmission to hospital for revascularization, MI, heart failure, or stroke.

Statistical Analysis

All data analysis was performed using SAS, version 8.2 for Windows (SAS Institute, Cary, NC) statistical software. Descriptive statistics were used for continuous variables including mean, median, SD, and SE. Frequencies were used for categorical variables. Univariate comparisons included unpaired \(t\) tests and ANOVA for continuous variables and contingency table analysis for categorical variables. Kaplan-Meier analysis was used to calculate cumulative probabilities for time-to-event outcomes. Cox multiple regression analysis was performed to identify independent predictors of late mortality, late MACE, and cardiovascular event by entering all variables that had a univariate association with either outcome at \(P<0.25\) or those of known clinical significance but failing to meet the critical \(\alpha\) level for submission to the model. Variables entered are listed in Appendix A. The \(\alpha\) for variable retention in multivariable models was 0.10.

The evaluation of DES versus BMS in the entire data set was truncated to 4 years of follow-up. The fully adjusted risk ratios for DES and renal dysfunction (CrCl < 60 mL/min) were determined by a nonparsimonious Cox regression analysis by forcing all variables listed in Appendix A into the model, including the interaction term DES \(\times\) CrCl < 60 mL/min. In propensity-matched cohorts, patients with DES were matched with those with BMS at a ratio of 1:2 using propensity score methods. Patients were matched on the following variables: age, gender, urgent priority, primary PCI, shock, failed thrombolysis, left ventricular grade, New York Heart Association, recent MI within 30 days before PCI, diabetes, hypertension, peripheral vascular disease, left main coronary artery disease, multivessel disease (MVD), Type C lesion, glycoprotein inhibitor use, and a preprocedural thrombolysis in MI flow \(<3\) (Appendix B). Late outcomes for DES versus BMS stratified by renal function were evaluated by Kaplan-Meier analysis.

This study was approved by the Institutional Research Ethics Boards of the University Health Network and the Institute of Clinical Evaluative Sciences. The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

Between April 2000 and September 2007, the clinical, angiographic, procedural, and outcome variables of 12 662 consecutive patients who underwent their first PCI procedure at the Peter Munk Cardiac Centre were entered into a prospective database. Baseline creatinine was available for 11 969 patients who were stratified into groups with or without at least moderate CKD, defined as a CrCl < 60 mL/min (CKD class \(\geq 3\)). Long-term follow-up was available for 11 953 patients (99.9%) who constituted the total study population. CKD was present in 3070 patients (25.7%).

Clinical, Procedural, and Angiographic Characteristics

Baseline patient characteristics are shown in Table 1. Patients with CKD were older, more likely to be female, hypertensive, or diabetic. They had a greater prevalence of other comorbidities, particularly other arteriopathies such as peripheral vascular disease, carotid stenosis, and cerebrovascualr disease, were more likely to have had a previous CABG and a
lower ejection fraction. They were more likely to present emergently (acute coronary syndrome, rescue, facilitated, or salvage PCI) and had a significantly greater occurrence of cardiogenic shock. Procedural and angiographic characteristics are shown in Table 2. Patients with lower CrCI more frequently had MVD and required treatment of a greater number of vessels and lesions. They underwent more left main stem, saphenous vein graft, and complex lesion interventions. Their baseline thrombolysis in MI flow grade was lower, and they required more intra-aortic balloon pump support. The complexity of their procedures was reflected in longer fluoroscopy times. The proportion of patients treated with at least 1 stent and the proportion treated with DES did not differ between groups.

### Late Outcomes

The mean follow-up period was 3.6±2.2 years (range, 0 to 7.75 years). Survival estimated by Kaplan–Meier analysis was 82.7±0.6 for the group as a whole over the 7-year follow-up period. Survival rates decreased with increasing level of CKD (P<0.0001) (Figure 1). Patients with normal renal function (CrCI≥90 mL/min) or only mild CKD (CrCI,
60 to 89 mL/min) had 91.9±0.6% and 84.5±1.1% survival, respectively. As renal function deteriorated, survival rates were lower at 7 years: 73.3±1.7% in patients with moderate CKD (CrCl, 30–59 mL/min). Patients with at least moderate CKD had reduced survival at 7 years (64.5±1.1% survival, 84.5±1.1% survival, respectively). As renal function deteriorated, survival rates were lower at 7 years: 73.3±1.7% versus 68.1±0.8%, P<0.001, and reduced cardiovascular event-free survival (44.0±1.4% versus 63.4±0.8%, P<0.001).

By multivariate analysis (Tables 4 and 5), preexisting moderate or severe CKD was a significant independent predictor of late mortality, associated with a more than 2-fold risk of death (hazard ratio [HR]: 3.24, P<0.0001). This was more significant than the adverse effect of diabetes (HR: 1.82, P<0.0001). Only the presence of cardiogenic shock (HR: 3.24, P<0.0001) and/or a Grade IV ventricle (HR: 2.85, P<0.0001) carried a greater risk. Emergent procedures, left
DES Use and Late Outcomes in Patients With CKD

To investigate further the relationship between DES use and late outcomes in the presence or absence of CKD, the evaluation of DES versus BMS in the entire data set was truncated to 4 years of follow-up. Fully adjusted Cox regression analysis in this subset showed that CKD remained independently associated with increased mortality (HR: 2.23, \( P<0.001 \)), increased MACE (HR: 1.32, \( P<0.001 \)), and increased cardiovascular events (HR: 1.32, \( P<0.001 \)). DES use was independently associated with reduced mortality (HR: 0.51, \( P<0.001 \)), reduced revascularization (HR: 0.67, \( P<0.001 \)), reduced MACE (HR: 0.66, \( P<0.001 \)), and reduced CV events (HR: 0.69, \( P<0.001 \)). The interaction term (DES × CrCl < 60) was significant for mortality (\( P=0.005 \)), MACE (\( P=0.041 \)), and CV events (\( P=0.024 \)).

Kaplan–Meier survival curves are shown in Figure 2. Patients with normal or mild CKD (CrCl ≥ 60 mL/min) who received DES rather than BMS showed a clear survival advantage (96.8% versus 93.8%, \( P<0.001 \), Figure 2a) and a significantly greater late MACE-free survival (82.8% versus 74.5%, \( P<0.001 \)). Multivariate analysis suggested that DES use in patients with normal renal function was associated with a lower mortality (HR: 0.52, \( P<0.001 \)), as well as a reduced need for CABG (HR: 0.06, \( P=0.006 \)) and repeat PCI (HR: 0.78, \( P=0.002 \)) (Table 6). After propensity matching, a survival advantage remained (96.7% versus 94.4%, \( P=0.005 \); Figure 2b).

In patients with moderate to severe CKD (CrCl < 60 mL/min) who received DES, there was no overall survival benefit demonstrated at 4 years (75.6% versus 75.2%, \( P=0.3 \); Figure

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**Figure 2.** Kaplan–Meier survival curves for patients treated within DES period, according to stent type and the presence or absence of CKD. (a) Unadjusted survival in patients with normal or only mild renal impairment (CrCl ≥ 60 mL/min). (b) Adjusted survival in patients with normal or only mild renal impairment (CrCl ≥ 60 mL/min) (after propensity matching). (c) Unadjusted survival in patients with CKD (CrCl < 60 mL/min). (d) Adjusted survival in patients with CKD (CrCl < 60 mL/min) (after propensity matching).
Table 6. Fully Adjusted Risk Ratios for DES in Stratified Cox Regressions for Normal and Abnormal Renal Function (2000–2007, Data Truncated to 4 y)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>CrCl, mL/min</th>
<th>&lt;60</th>
<th>≥60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td></td>
<td>0.85 (0.69 to 1.05)</td>
<td>0.52 (0.38 to 0.71)</td>
</tr>
<tr>
<td>Combined late MACE</td>
<td></td>
<td>0.81 (0.69 to 0.95)</td>
<td>0.66 (0.60 to 0.81)</td>
</tr>
<tr>
<td>PCI</td>
<td></td>
<td>0.78 (0.60 to 1.03)</td>
<td>0.78 (0.68 to 0.91)</td>
</tr>
<tr>
<td>CABG</td>
<td></td>
<td>0.35 (0.05 to 2.69)</td>
<td>0.06 (0.01 to 0.46)</td>
</tr>
<tr>
<td>Any revascularization</td>
<td></td>
<td>0.68 (0.53 to 0.88)</td>
<td>0.67 (0.59 to 0.79)</td>
</tr>
<tr>
<td>CV event</td>
<td></td>
<td>0.83 (0.72 to 0.96)</td>
<td>0.68 (0.61 to 0.77)</td>
</tr>
</tbody>
</table>

Data are displayed as HR (95% CI). Late MACE, late major adverse cardiac events (death or repeat revascularization); CV event, late MACE and hospital admissions for heart failure, myocardial infarction, and stroke.

2c), although significant divergence of the curves was seen in the first year (P=0.002), with catch-up at 2 years (P=0.057), suggesting an early but transient survival advantage. MACE-free survival differed significantly (64.7% versus 61.8%, P=0.02). By multivariate analysis, however, DES use in patients with CKD was associated with reduced subsequent revascularization (HR: 0.68, CI: 0.53 to 0.88, P=0.004), reduced MACE (HR: 0.81, CI: 0.69 to 0.95, P=0.011), and reduced CV events (HR: 0.83, CI: 0.72 to 0.96, P=0.014) but was not associated with lower mortality (HR: 0.85, CI: 0.69 to 1.05, P=0.1; Table 6). When patients were propensity matched, early divergence of the survival curves was again demonstrated, but at 4 years no survival benefit was observed (Figure 2d).

Discussion

We have demonstrated that the adverse impact of RI on unselected patients undergoing PCI continues to influence late outcomes even in the modern era of DES, dual antiplatelet therapy, and glycoprotein IIb/IIIa inhibitors. Patients with CKD have reduced survival and reduced MACE-free survival at long-term follow-up, and CKD is shown to be an independent predictor of both mortality and MACE. DES use in this high-risk cohort is associated with improved outcomes.

Our study identified preexisting CKD of at least moderate severity in just over 25% of all-comers for PCI between 2000 and 2007 at a single institution. The definition excluded those with mild CKD (CrCl, 60 to 89 mL/min) who comprise another 36% of the population in whom outcome might also be unfavorably affected.3 Prolonged survival of patients with renal dysfunction and the aging of the general population suggest that this proportion will continue to increase. The PCI as a strategy remains suboptimal in this group despite a limited.

We anticipated that patients with renal disease would be overrepresented in these subgroups, overall, the clinically driven revascularization rates in our registry were lower than the protocol-driven repeat PCI rates reported in DES randomized controlled trials. Lemos et al showed that DES reduced TVR rates only in certain subgroups (those patients with diabetes, long lesions, or small vessels who were at increased risk of restenosis) and although it might be anticipated that patients with renal disease would be overrepresented in these subgroups, overall, the clinically driven revascularization rates in our registry were lower than the protocol-driven repeat PCI rates reported in DES randomized controlled trials. Lemos et al observed that in patients with renal dysfunction undergoing PCI, RI itself was not an independent predictor of TVR after DES versus BMS use, although they did demonstrate an antirestenotic effect of sirolimus eluting stents versus BMS in patients with (HR:...
0.39, \( P=0.001 \)) and without CKD (HR: 0.31, \( P=0.03 \)) at 1 year.\(^6\) That CABG-free survival might also be increased by DES use in patients with CKD was perhaps unexpected, although this observation did not persist after multivariate analysis (HR: 0.35, \( P=0.31 \)). This may, however, reflect the higher prevalence of complex and/or MVD in patients with CKD, which can now be considered for PCI in DES era. Although our group has demonstrated previously that patients with CKD undergoing PCI have lower procedural success rates, more frequent failure rates of stent delivery, and greater post-PCI residual stenosis compared with those with normal renal function,\(^4\) DES compared with BMS use may allow the operator to achieve more complete revascularization. The tendency to attempt more complex lesions in more vessels may contribute to lower rates of subsequent CABG. In addition, restenosis in DES compared with that in BMS is more focal.\(^5\) Those patients with CKD in whom revascularization is incomplete, or in whom restenosis occurs, are more likely to be treated by repeat PCI rather than CABG given the high morbidity and mortality associated with CABG in this population.

Despite reporting outcomes from a large prospective registry, our study suffers from the limitations of retrospective, observational data. Specifically, we cannot account for all possible confounding factors and, because follow-up data were collected from linkage to a provincial administrative database, we cannot report on certain types of data such as causes of death or types of clinical presentation for subsequent revascularization (acute coronary syndrome, elective, etc.). In addition, acute renal failure patients were not excluded from the database, although the numbers of patients requiring immediate dialysis were small (0.08%, Table 3).

In summary, our data represent the longest reported follow-up of patients with RI undergoing PCI in the modern era. It is also the largest CKD cohort studied. We demonstrate that the adverse effect of CKD on both mortality and morbidity early postprocedure has a lasting impact. The use of DES in this cohort may in part mitigate the poorer late outcomes, but patients remain at high risk compared with those with normal renal function. Further, prospective studies are required. As this population continues to increase as a proportion of all comers for PCI, RI will have a growing influence on the long-term clinical outcomes of our patients.

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**Disclosures**

Dr V. Dzavik received honoraria from Cordis J&J, Abbott Vascular, and Boston Scientific.

**References**


21. Smith SC, Dove JT, Jacobs AK, Ward Kennedy J, Kereiakes D, Kern MJ. Prospective registry, we found the presence of moderate to severe CKD (creatinine clearance reduced and in-hospital and 1-year outcomes are poor. Little is known about outcomes in this cohort beyond 1 year, and high-risk patient cohort.

**CLINICAL PERSPECTIVE**

In patients with chronic kidney disease (CKD) undergoing percutaneous coronary intervention, procedural success is reduced and in-hospital and 1-year outcomes are poor. Little is known about outcomes in this cohort beyond 1 year, and the effect of CKD on late outcomes in contemporary practice with drug-eluting stents remain unknown. In a large prospective registry, we found the presence of moderate to severe CKD (creatinine clearance <60/mL/min), present in a quarter of all patients undergoing percutaneous coronary intervention, to be associated with significantly lower survival and major adverse cardiac events-free survival out to 7 years. Only cardiogenic shock and severe left ventricular dysfunction were more predictive of worse long-term outcomes. In contrast to patients with more normal renal function, in those with CKD, drug-eluting stents use was not associated with a lower late mortality. An association with a lower rate of major adverse cardiac events was primarily a function of a decreased need for repeat revascularization. Poor late outcome after percutaneous coronary intervention in patients with CKD likely occurs as a result of a constellation of unfavorable anatomic, inflammatory, biochemical, and rheological factors. Although the use of drug-eluting stents may in part mitigate poorer late outcomes, patients with CKD remain at high risk. Clinicians must use all strategies possible to optimize percutaneous coronary intervention results and to ensure optimal use of proven therapies in this increasingly important high-risk patient cohort.
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SUPPLEMENTAL MATERIAL
Appendix A.

Variables entered into multivariate analysis model.

1. Renal Impairment defined as creatinine clearance <60ml/min
2. Age
3. Gender
4. Urgent Indication for PCI
5. Primary PCI
6. Presence of cardiogenic shock
7. Failed Thrombolysis
8. LV grade 3 – left ventricular ejection fraction grade 3 (20-39%)
9. LV grade 4 - left ventricular ejection fraction grade 4 (<20%)
10. NYHA grade 4 – New York Heart Association grade 4 heart failure symptoms
11. Recent MI within 30 days prior to PCI
12. Diabetes – clinical history of insulin- or non-insulin-treated diabetes
13. Hypertension
14. Vascular disease - clinical history of cerebrovascular disease or peripheral vascular disease
15. LMS disease
16. Multivessel disease
17. Prior revascularisation – Coronary bypass surgery or PCI predating index procedure
18. DES use – drug eluting stent use
19. LMS – left main stem disease (>50% stenosis)
20. MVD – multivessel disease (> 1 major epicardial vessel diseased)
21. GPI use – Glycoprotein IIbIIIa inhibitors
22. C type lesion – ACC/AHA lesion type

23. Pre-procedure TIMI flow<3

24. Interaction of DES and CrCl<60

Appendix B  Clinical Characteristics of Propensity-Matched Cohorts*

<table>
<thead>
<tr>
<th>Variable</th>
<th>BMS</th>
<th>DES</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>4599</td>
<td>2299</td>
<td></td>
</tr>
<tr>
<td>Age (mean ± SD)</td>
<td>62.8 ± 11.8</td>
<td>63.3 ± 11.6</td>
<td>0.06</td>
</tr>
<tr>
<td>No. diseased vessels</td>
<td>1.9 ± 0.9</td>
<td>1.9 ± 0.9</td>
<td>0.55</td>
</tr>
<tr>
<td>Creatinine pre-PCI</td>
<td>101 ± 75</td>
<td>100 ± 66</td>
<td>0.57</td>
</tr>
<tr>
<td>Female (%)</td>
<td>27.2</td>
<td>28.1</td>
<td>0.44</td>
</tr>
<tr>
<td>Urgent</td>
<td>48.3</td>
<td>48.7</td>
<td>0.74</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>1.3</td>
<td>1.4</td>
<td>0.60</td>
</tr>
<tr>
<td>Failed thrombolysis</td>
<td>2.7</td>
<td>2.6</td>
<td>0.60</td>
</tr>
<tr>
<td>Shock</td>
<td>0.7</td>
<td>0.9</td>
<td>0.44</td>
</tr>
<tr>
<td>LV Grade 3</td>
<td>8.9</td>
<td>10.3</td>
<td>0.07</td>
</tr>
<tr>
<td>LV Grade 4</td>
<td>1.7</td>
<td>1.8</td>
<td>0.84</td>
</tr>
<tr>
<td>NYHA Class IV</td>
<td>2.0</td>
<td>2.0</td>
<td>0.9</td>
</tr>
<tr>
<td>MI &lt;30 days</td>
<td>28.4</td>
<td>29.1</td>
<td>0.54</td>
</tr>
<tr>
<td>Diabetes</td>
<td>28.6</td>
<td>28.5</td>
<td>0.93</td>
</tr>
<tr>
<td>Hypertension</td>
<td>65.6</td>
<td>63.6</td>
<td>0.10</td>
</tr>
<tr>
<td>PVD</td>
<td>8.6</td>
<td>9.4</td>
<td>0.25</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>7.4</td>
<td>7.4</td>
<td>1.0</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>62.5</td>
<td>61.7</td>
<td>0.51</td>
</tr>
<tr>
<td>Left main disease</td>
<td>3.4</td>
<td>4.1</td>
<td>0.10</td>
</tr>
<tr>
<td>Type C Lesion</td>
<td>47.6</td>
<td>47.9</td>
<td>0.79</td>
</tr>
<tr>
<td>GPI use</td>
<td>80.8</td>
<td>80.9</td>
<td>0.88</td>
</tr>
<tr>
<td>TIMI flow &lt;3</td>
<td>20.2</td>
<td>20.1</td>
<td>0.87</td>
</tr>
</tbody>
</table>
Results reported as proportions except where indicated as means ± 1 standard deviation (SD).