Bivalirudin for Primary Percutaneous Coronary Interventions

Outcome Assessment in the Ottawa STEMI Registry

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Background—Data from randomized trials has demonstrated the superiority of bivalirudin to glycoprotein IIb/IIIa inhibitors plus heparin in patients undergoing primary percutaneous coronary intervention. Real-world performance of bivalirudin in primary percutaneous coronary intervention and the benefit of bivalirudin over heparin remain unknown in an era of routine dual antiplatelet therapy.

Methods and Results—From July 2004 to December 2010, 2317 consecutive patients were indexed in the University of Ottawa Heart Institute ST-segment–elevation myocardial infarction registry. During this period 748 patients received bivalirudin, 699 patients received glycoprotein IIb/IIIa inhibitors, and 676 patients received unfractionated heparin alone. The primary outcome was the rate of noncoronary artery bypass graft related thrombolysis in myocardial infarction major bleeding. Bivalirudin significantly reduced the primary outcome compared with heparin plus glycoprotein IIb/IIIa inhibitors (2.7% versus 7.3%, adjusted OR 2.96, 95% CI: 1.61–5.45, P<0.001) and the composite end point of death, stroke, reinfarction and major bleed (OR 1.66, 95% CI: 1.12–2.45, P=0.01). Compared with heparin alone, a reduction in major bleeds (OR 1.21, 95% CI: 0.60–2.44, P=0.59) or the composite end point (1.05, 95% CI: 0.68–1.63, P=0.83) with bivalirudin could not be demonstrated. Notably, major bleeding was associated with a 5-fold increase in the risk of mortality both in-hospital (3.5% versus 20.6%) and out to 180 days (5.6% versus 25.8%).

Conclusions—Bivalirudin use compared with glycoprotein IIb/IIIa inhibitors plus heparin as an antithrombotic strategy in primary percutaneous coronary intervention results in less major bleeding in contemporary practice. A benefit of bivalirudin over heparin could not be established with this registry and requires additional investigations to either confirm or refute.  (Circ Cardiovasc Interv. 2012;5:805-812.)

Key Words: bivalirudin ■ bleeding ■ complications ■ primary PCI

Primary percutaneous coronary intervention (PCI) for treatment of ST-segment–elevation myocardial infarction (STEMI) is the preferred treatment strategy in patients in whom revascularization can be achieved within 90 to 120 minutes. Indeed, in the United States nearly 85% of patients with STEMI undergo PCI as a revascularization strategy.1 Adjuvant pharmacotherapy including oral antiplatelet regimens,2 heparins, and glycoprotein IIb/IIIa inhibitors (GPI)3-4 have demonstrated variable efficacy and safety profiles in these patients. However, bleeding remains a potent predictor of prognosis in both STEMI and percutaneous coronary intervention6 where implementation of bleeding avoidance strategies are becoming increasingly important.6 In the Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZONS AMI) trial bivalirudin use resulted in an absolute 2.9% reduction in 30-day net adverse clinical events and 1.0% reduction in 30-day mortality when compared with a GPI plus heparin in STEMI patients undergoing PPCLI.7 More recently, in a comparison of bivalirudin versus abciximab plus heparin in non-ST-segment–elevation myocardial infarction patients, no differences existed in a combined end point of death, recurrent MI, urgent revascularization or major bleeding; however, bivalirudin significantly reduced the risk of major bleeding from 4.6% to 2.6%.8

Clinical trials designed to demonstrate efficacy and safety often exclude high-risk patients or those patients at greatest risk of complications from the studied therapy, thus limiting their generalizability. Data from registries allow real-world evaluation of adjuvant pharmacotherapy permitting a comparison of strategies in a broader population. However, real-world data comparing outcomes with the use of bivalirudin to other antithrombotic strategies in PCI have yet to be reported.

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WHAT IS KNOWN

- The HORIZONS AMI trial demonstrated a reduction in bleeding and mortality with bivalirudin use compared with a glycoprotein IIb/IIIa inhibitor and heparin in primary percutaneous coronary intervention.
- Observational data from the Swedish Coronary Angiography and Angioplasty Registry registry suggest that adding unfractionated heparin to bivalirudin may be beneficial.
- Direct comparisons of bivalirudin to heparin alone in primary percutaneous coronary intervention are lacking.

WHAT THE STUDY ADDS

- The current study confirms the efficacy and safety of bivalirudin compared with glycoprotein IIb/IIIa inhibitor and heparin in a real-world contemporary cohort.
- A benefit of bivalirudin compared with heparin alone could not be demonstrated, highlighting the need for randomized studies between these antithrombotic strategies.

Thus, as a high volume regional STEMI program, we chose to evaluate various antithrombotic strategies in patients undergoing PPCI. Specifically, we set out to evaluate clinical outcomes between bivalirudin, GPI, and heparin alone.

Methods

Study Design, Data Source, and Patients

The University of Ottawa Heart Institute regional STEMI program services a population of ~1.3 million residents in eastern Ontario.9,10 The PPCI program receives patients from 9 referral hospitals as well as a direct transport of STEMIs identified by paramedics evaluating patients in the field. Patients are prospectively indexed in the University of Ottawa Heart Institute–STEMI registry and, demographic data, therapies, and clinical outcomes are recorded.

Inclusion in the study required a confirmed diagnosis of STEMI with the revascularization strategy being PPCI. For the purpose of this study, STEMI was defined as ST elevation of ≥1 mm in 2 contiguous leads on a 12-lead ECG and presentation within 12 hours of onset of symptoms. Patients were excluded if they received thrombolytics, underwent coronary artery bypass graft (CABG) surgery immediately as a revascularization strategy, declined angiography, or received medical therapy alone. Patients were classified based on adjuvant antithrombotic therapy into three groups: bivalirudin with provisional GPI use, GPI, or heparin alone. All patients received aspirin 160 mg to chew, clopidogrel 600 mg load followed by 75 mg daily, and an unfractionated heparin bolus of 60 U/kg at a maximum of 4000 U at time of first medical evaluation by a physician. Notably, this protocol is similar to that in HORIZONS AMI in which 65% of patients received an upfront bolus of heparin followed by bivalirudin in the catheterization laboratory. These therapies are as per our regionalized STEMI protocol and low-molecular weight heparins are not utilized. Subsequent use of bivalirudin, GPIs, or continuing with heparin alone was at the discretion of the interventionalist. This study was reviewed and approved by the University of Ottawa Heart Institute institutional human research ethics board and was deemed not to require informed consent.

Outcome Measures and Definitions

The primary outcome of this study was the rate of non-CABG–related Thrombolysis In Myocardial Infarction (TIMI) major bleeding (defined as a fall in hemoglobin of >5g/dL once adjusted for transfusion or intracranial bleeding).11 Secondary outcomes included a composite end point of in-hospital death, stroke, reinfarction, or non-CABG–related TIMI major bleeding. Stroke was defined as either ischemic or hemorrhagic stroke resulting in a new neurologic deficit of >24 hours’ duration as diagnosed by a treating neurologist. Reinfarction was defined as recurrent chest pain associated with re-elevation of the ST segments in association with either re-elevation of the cardiac enzymes (twice the upper limit of the normal range) or angiographic documentation of reocclusion of the infarct-related artery. Additional secondary outcomes of interest included death at 30-day and 180-day follow-up, bleeding outcomes using the TIMI definitions, need for transfusion, and probable and definite stent thrombosis as defined by the academic research consortium criteria.12 Mortality is reported as both percent of patients with available follow-up and as an adjusted mortality rate calculated by censoring patients lost to follow-up at 180 days.

Statistical Analysis

All continuous variables were described as mean (±SD) or median (and interquartile range) and categorical variables as number (%), as appropriate. For composite end points, all components are reported individually.13 For patient and procedural characteristics, categorical variables were compared by χ² and continuous variables by analysis of variance or Kruskal-Wallis test as appropriate. For adjusted analysis of the primary and composite outcome, multiple logistic regression (MLR) was performed and odds ratio (OR) with 95% CI and adjusted P values are reported. Variables in the MLR model were identified by univariate analysis (online-only Data Supplemental Tables I and II) using bivalirudin as the reference group, with indicator variables for heparin and GPI. Independent variables demonstrating a P<0.15 were retained in the final MLR models. Lastly, to account for baseline differences in the cohorts, propensity scores were calculated using baseline clinical variables in Table 1 and baseline angiographic variables in Table 2. A propensity score-adjusted logistic regression analysis was then performed and adjusted ORs and 95% CI were reported. As there were 3 treatment arms, the outcome in the propensity score model was the treatment group. A logistic regression model with a generalized logit link function was utilized to model 3 treatment arms as the outcome. All analyses were performed using SAS software version 9.2.

Results

Population and Baseline Characteristics

From July 2004 to December 2010, a total of 2317 consecutive patients were indexed in the registry from which a cohort of 2123 patients who underwent PPCI was included in the analysis (Figure 1). Of the patients initially transferred for PPCI, 0.7% received thrombolytics, 2.4% underwent emergent CABG as a revascularization strategy, 0.6% declined angiography, and 4.7% had medical therapy alone. Of patients undergoing PPCI, 748 patients received bivalirudin, 699 patients received GPI, and 676 patients received heparin alone.

Despite the nonrandomized nature of the cohort study, groups were similar in a number of baseline characteristics including age, sex, and cardiac risk factors (Table 1). However, important differences were present between cohorts—namely fewer patients receiving bivalirudin (1.5%) presented in Killip class IV compared with GPI (4.9%) or heparin (3.7%, P<0.001). Procedure-related factors including infarct-related artery, percent of patients receiving stents, and post-percutaneous coronary intervention TIMI flow did not differ significantly among the groups (Table 2). More patients in the heparin group had a radial approach employed (26.2%) compared with GPI (5.7%) or bivalirudin (5.6%, P<0.001).
Antiplatelet regimen, including admission and discharge aspirin and clopidogrel use was similar among the groups.

**Clinical Outcomes**

The incidence of the primary and secondary outcomes are reported in Table 3. The primary outcome, major bleeds, occurred in 2.7% of bivalirudin patients, 7.3% of GPI patients, and in 3.3% of patients treated with heparin alone. The composite end point of in-hospital death, major bleeds, stroke, and reinfarction, occurred in 7.6% of patients receiving bivalirudin compared with 11.4% of patients receiving GPI and in 9.5% of patients receiving heparin. Other than marked differences in the rates of major bleeding, other components of the composite end point (in-hospital mortality, stroke or reinfarction) were similar among the strategies.

Bleeding complications in this cohort of patients was common, with 11.8%, 20.5%, and 12.9% of patients experiencing either a major or minor bleed in the bivalirudin, GPI, and heparin group, respectively. Similarly, patients receiving either bivalirudin (3.1%) or heparin (3.6%) experienced approximately half the number of any major bleeding complication than patients receiving GPI (7.4%). Last, the risk of TIMI minor bleeds was also markedly lower in patients who did not receive GPI (bivalirudin 4.1% versus GPI 13.2% versus heparin 9.5%). Interestingly, the risk of transfusion did not differ between the cohorts.

At 180 days, mortality in the total study population was low at 6.2%. However, in patients in whom a major bleeding complication was identified, mortality increased significantly. Comparatively, major bleeding events increased the incidence of unadjusted mortality ≈5-fold occurring in 3.5% versus 20.6%, 3.8% versus 21.1%, and 5.6% versus 25.8% during the hospitalization, at 30 days, and at 180 days, respectively (P<0.001 for all comparisons, Figure 2). Notably, a numerically higher frequency of stent thrombosis was seen with bivalirudin use (1.9% bivalirudin versus 1.0% GPI versus 0.6% heparin).

**Adjusted Analyses**

Because of the nonrandomized design of this study, univariate logistic regression was first performed to identify predictors of each end point and the results are presented in the online-only Data Supplemental Tables I and II. Variables with significant association with the primary and composite outcome (P<0.15) were retained in the MLR models. In-hospital major bleeds occurred less frequently with bivalirudin compared with GPIs (OR 2.78, 95% CI: 1.53–5.06, P<0.001). In contrast, bivalirudin did not demonstrate significant benefit when compared with heparin alone (OR 1.15, 95% CI: 0.59–2.24, P=0.68). An elevated initial heart rate, anterior MI, Killip class, and renal failure were also independent predictors of the primary outcome. A propensity score-adjusted logistic regression analysis was also performed which yielded results similar to the MLR analysis. In-hospital major bleeds occurred less frequently with bivalirudin compared with GPIs (OR 2.96, 95% CI: 1.60–5.45, P<0.001, Figure 3A). Again, bivalirudin did not
reduce the risk of major bleeds compared with heparin alone (OR 1.21, 95% CI: 0.60–2.43, $P = 0.58$).

The composite end point of death, stroke, reinfarction, and major bleeds did not meet criteria for significance when comparing bivalirudin with either GPI and heparin (OR 1.49, 95% CI: 0.98–2.28, $P = 0.06$) or heparin alone (OR 0.97, 95% CI: 0.62–1.51, $P = 0.88$), although there was a strong trend favoring bivalirudin over GPI. Notably, propensity score adjustment demonstrated that the composite outcome occurred less frequently with bivalirudin compared with GPI and heparin (OR 1.62, 95% CI: 1.07–2.44, $P = 0.02$). However, as with the primary end point, no difference was observed when bivalirudin was compared with heparin alone (OR 1.05, 95% CI: 0.68–1.63, $P = 0.83$).

**Discussion**

The current study is the first to compare bleeding and clinical outcomes between antithrombotic strategies in real-world patients in the context of PPCI. Compared with GPsIs, we found that bivalirudin use resulted in a 4.6% absolute reduction in the number of major bleeds resulting in a reduction in the composite end point of in-hospital death, major bleeding, stroke, and reinfarction. In our study, bivalirudin did not result in significant benefit in bleeding or the composite end point.
point when compared with heparin alone. Finally, though not powered to assess differences in mortality, we noted a 5-fold increase in the risk of death out to 180 days in patients in whom major bleeding occurred confirming the prognostic implication of bleeding seen in data from randomized control trials.\textsuperscript{14,15}

Monitoring real-world performance of antithrombotic pharmacotherapy provides complimentary information to results derived from clinical trials. Specifically, our cohort included all patients presenting with a diagnosis of STEMI in whom PPCI is the revascularization strategy of choice. This allows for evaluation of outcomes in a broader population and validates findings from randomized trials. Despite these important differences, we observed a similar pattern of outcomes compared with the HORIZONS AMI study.\textsuperscript{7} For example, we found a 3.8% decrease in our composite end point (7.6% versus 11.4%, OR 1.66, 95% CI: 1.12–2.45 \( P = 0.01 \)) when comparing bivalirudin with GPI – compared with a 2.9% reduction in net adverse clinical events in HORIZONS (12.1% versus 9.2%). Thus, our findings compliment data from HORIZONS and confirm the efficacy and safety of bivalirudin in a cohort of unselected patients managed by PPCI.

However, our data also permit a comparison of outcomes between patients in whom heparin alone was utilized as an antithrombotic strategy – a comparison not previously performed in PPCI. Most recently, it has been reported that heparin plus bivalirudin compared with bivalirudin alone reduced the rates of death and definite target vessel thrombosis during PPCI in the Swedish Coronary Angiography and Angioplasty Register.\textsuperscript{16} Our study has two important differences. First, all patients (including patients receiving bivalirudin) received unfractionated heparin upfront as part of our standardized protocol. Second, our data provide a cohort of patients in whom heparin alone was utilized for comparison. Interestingly, in the current study we could not detect a reduction in major bleeding (2.7% versus 3.3%, \( P = 0.59 \)) or the combined end point (7.6% versus 9.5%, \( P = 0.83 \)) with bivalirudin when compared with heparin alone. Notably, although this comparison did not meet significance, our bleeding rates and reductions in bleeding are similar to those seen in a randomized comparison of bivalirudin and heparin in patients undergoing elective percutaneous coronary intervention (3.1% versus 4.6%) and the lack of difference may simply reflect the small sample size and resultant lack of statistical power.\textsuperscript{17} Nonetheless, whereas it is evident from the randomized data and now the current study that GPIs increase bleeding events in patients undergoing PPCI, the use of

\begin{table}[h!]
\centering
\caption{Unadjusted Clinical Outcomes}
\begin{tabular}{|c|c|c|c|}
\hline
 & Bivalirudin & GPI & Heparin \\
(n=748) & (n=699) & (n=676) \\
\hline
\textbf{In-hospital outcomes}–(no.), % & & & \\
Bleeding & 20 (2.7) & 50 (7.3) & 22 (3.3) \\
TIMI major non-CABG & 3 (15.8) & 3 (15.8) & 2 (22.2) \\
TIMI major CABG & 23 (3.1) & 52 (7.4) & 24 (3.6) \\
Any major bleed & 65 (8.9) & 91 (13.2) & 63 (9.5) \\
TIMI minor & 88 (11.8) & 143 (20.5) & 87 (12.9) \\
Any major or minor bleed & 31 (4.1) & 36 (5.2) & 32 (4.7) \\
Transfusion & 28 (3.7) & 28 (4.0) & 36 (5.3) \\
Clinical outcomes & 12 (1.6) & 4 (0.6) & 11 (1.6) \\
Death–in hospital & 14 (1.8) & 9 (1.3) & 5 (0.6) \\
Stroke & 57 (7.6) & 80 (11.4) & 64 (9.5) \\
\hline
\textbf{Reinfarction composite end point} & & & \\
Stent Thrombosis–(no.), % & 14 (1.9) & 7 (1.0) & 4 (0.6) \\
ARC definite & 12 (85.7) & 7 (100.0) & 4 (100.0) \\
ARC probable & 2 (14.3) & 0 (0.0) & 0 (0.0) \\
ARC acute (<24 h) & 12 (85.7) & 4 (57.1) & 2 (50.0) \\
ARC subacute (1–30 d) & 2 (14.3) & 3 (42.9) & 2 (50.0) \\
\hline
\textbf{Mortality–(no.) %} & & & \\
30 d & 27* (3.7) & 29 (4.3) & 38 (5.7) \\
180 d & 40 (5.9) & 42 (6.1) & 50 (7.6) \\
\hline
\end{tabular}
\end{table}

\textsuperscript{ARC} indicates academic research consortium; CABG, coronary artery bypass grafting; GPI, glycoprotein IIb/IIIa inhibitor; TIMI, thrombolysis in myocardial infarction.

\textsuperscript{*One patient died after 30 days but in hospital.
of bivalirudin preferentially over heparin in PPCI remains to be confirmed in randomized studies.

Certainly, our data add to the growing evidence that bivalirudin confers equivalent reductions in ischemic end points with an improved safety profile. For example, Marso and colleagues demonstrated marked reductions in bleeding events when bivalirudin was used in patients undergoing percutaneous coronary intervention in their analysis of the National Cardiovascular Data Registry—an effect magnified when used in conjunction with vascular closure devices. Moreover, bivalirudin use has recently been shown in two studies to be strongly associated with reductions in bleeding events and length of stay in STEMI patients, resulting in the additional benefit of a significant reduction in overall cost of care. However, despite the abundance of evidence, a recent analysis of the National Cardiovascular Data Registry database suggests only 14% of STEMI patients receive bivalirudin as an antithrombotic therapy, whereas 65% of patients receive unfractionated heparin of which 80% receive a concomitant GPI. This suggests that the majority of STEMI patients continue to receive GPI plus heparin despite the overwhelming evidence of increased bleeding.

Stent thrombosis in patients undergoing PPCI is an exceedingly rare event although mortality has been reported to be as high as 30% for patients with definite or probable stent thrombosis. In HORIZONS AMI, acute stent thrombosis occurred more frequently with bivalirudin than GPI (1.3% versus 0.3%, P < 0.001). Subsequent analyses of these patients have suggested that an upfront heparin bolus and 600 mg clopidogrel loading could in part mitigate the increased risk seen with bivalirudin. Interestingly, in our study all patients received upfront heparin and clopidogrel 600 mg loading yet we still observed a numerically higher incidence of acute stent thrombosis with bivalirudin compared with GPI or heparin alone (1.6% versus 0.6% versus 0.3%, respectively). Although the low event rates preclude adjustment of our observational data for known risk factors, our study suggests that in a real-world population acute stent thrombosis remains a risk with bivalirudin that remains despite upfront heparin and high dose clopidogrel.

Our study has limitations, both inherent in the observational nature of the cohort study and because of the limited sample sizes achieved. First, our study is not adequately powered to assess differences in hard clinical outcomes such as mortality, stroke, or reinfarction. Accordingly, we did not observe differences among the groups on these individual end points. However, bleeding complications have been strongly associated with higher mortality in patients with MI and thus we elected to use non-CABG–related TIMI major bleeding as our primary end point. Indeed, in our analysis patients in

### Figure 2

Effect of bleeding on in-hospital, 30-day and 180-day mortality. Red bars indicate patients with TIMI major noncoronary artery bypass grafting (CABG) bleeds in-hospital. Gray bars indicate patients without a TIMI major non-CABG bleeding complication. Mortality rates are reported for the total cohort in whom follow-up is available. Adjusted mortality rates are reported by censoring patients lost to follow-up at 180 days. *Mortality is lower at 30 days because of patients who died in-hospital but after 30 days. TIMI indicates thrombolysis in myocardial infarction.

### Figure 3

Outcome of propensity score-adjusted logistic regression analysis for primary and secondary outcomes. A, Effect of antithrombotic regimen on rates of non-CABG–related TIMI major bleeds. B, Effect of antithrombotic regimen on the composite end point of in-hospital major bleeds, death, cerebrovascular accident, or reinfarction. Boxes denote odds ratios (OR) with lines representing 95% CI. CABG indicates coronary artery bypass grafting; GPI, glycoprotein IIb/IIIa inhibitor; TIMI, thrombolysis in myocardial infarction.
whom bleeding complications occurred had a 5-fold increase in risk of mortality during their index hospitalization. Second, the patients were not randomized to antithrombotic strategies. Accordingly, we used accepted statistical modeling to control for differences in risk factors which varied between groups when analyzing our outcomes. However, important differences in the use of radial access existed among the groups (26.2% in heparin versus 5.6% in the bivalirudin group), a magnitude of difference difficult to fully adjust. Indeed in the STEMI cohort of The Radial Versus femorAL (RIVAL) access for coronary intervention trial, radial access reduced a composite outcome of death, MI, stroke, and non-CABG–related major bleeding from 5.2% to 3.1% (P<0.002). Thus, this large difference in the use of radial access in the heparin cohort may minimize the potential benefit of bivalirudin seen in this nonrandomized study. Nonetheless, despite these limitations, the data represent the largest real-world cohort of patients undergoing PPCI in which bivalirudin has been compared with GPI plus heparin and to heparin alone. Finally, our results very closely replicate both the trends and absolute event rates seen from randomized control trials, suggesting the applicability of their findings to clinical practice.

Conclusion

Our analysis confirms that bivalirudin with provisional GPI use compared with upfront GPI plus heparin as an antithrombotic strategy in patients undergoing PPCI results in significantly less bleeding complications. A benefit of bivalirudin over heparin alone could not be demonstrated in our analysis and remains to be established in this population.

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Disclosures

None.

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<td>0.908</td>
<td>0.995</td>
<td>0.030</td>
</tr>
<tr>
<td>Aspiration device used</td>
<td>0.927</td>
<td>0.583</td>
<td>1.474</td>
<td>0.749</td>
</tr>
<tr>
<td>Heparin vs Bivalirudin</td>
<td>1.209</td>
<td>0.654</td>
<td>2.236</td>
<td>0.545</td>
</tr>
<tr>
<td>GPI vs Bivalirudin</td>
<td>2.770</td>
<td>1.631</td>
<td>4.703</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
**Supplemental Table 2.** Univariate analysis composite endpoint endpoint (death, TIMI major non CABG bleed, cerebrovascular accident, and re-infarction)

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR</th>
<th>lower CI</th>
<th>upper CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>0.597</td>
<td>0.441</td>
<td>0.808</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.564</td>
<td>1.158</td>
<td>2.111</td>
<td>0.004</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.640</td>
<td>1.163</td>
<td>2.313</td>
<td>0.005</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>0.918</td>
<td>0.674</td>
<td>1.251</td>
<td>0.589</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.677</td>
<td>0.494</td>
<td>0.926</td>
<td>0.015</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>1.229</td>
<td>0.580</td>
<td>2.603</td>
<td>0.590</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>0.778</td>
<td>0.442</td>
<td>1.371</td>
<td>0.385</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>1.990</td>
<td>1.203</td>
<td>3.290</td>
<td>0.007</td>
</tr>
<tr>
<td>Killip Class</td>
<td>2.449</td>
<td>2.097</td>
<td>2.861</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anterior wall MI</td>
<td>1.591</td>
<td>1.189</td>
<td>2.129</td>
<td>0.002</td>
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<tr>
<td>Left Main IRA</td>
<td>9.968</td>
<td>2.860</td>
<td>34.742</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Initial TIMI flow</td>
<td>0.861</td>
<td>0.760</td>
<td>0.974</td>
<td>0.018</td>
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<tr>
<td>Femoral Access</td>
<td>1.285</td>
<td>0.794</td>
<td>1.285</td>
<td>0.307</td>
</tr>
<tr>
<td>Age</td>
<td>1.043</td>
<td>1.031</td>
<td>1.054</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Initial Systolic BP</td>
<td>0.986</td>
<td>0.981</td>
<td>0.991</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Initial heart rate</td>
<td>1.022</td>
<td>1.015</td>
<td>1.028</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Multivessel CAD</td>
<td>1.980</td>
<td>1.445</td>
<td>2.714</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>GFR (MDRD)</td>
<td>0.961</td>
<td>0.954</td>
<td>0.968</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Door to Balloon Time</td>
<td>1.002</td>
<td>1.000</td>
<td>1.003</td>
<td>0.009</td>
</tr>
<tr>
<td>Body mass index</td>
<td>0.953</td>
<td>0.923</td>
<td>0.983</td>
<td>0.003</td>
</tr>
<tr>
<td>Aspiration device used</td>
<td>1.068</td>
<td>0.779</td>
<td>1.464</td>
<td>0.684</td>
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<tr>
<td>Heparin vs Bivalirudin</td>
<td>1.267</td>
<td>0.873</td>
<td>1.841</td>
<td>0.213</td>
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<tr>
<td>GPI vs Bivalirudin</td>
<td>1.567</td>
<td>1.097</td>
<td>2.238</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>