Response to Letter Regarding Article, “Bivalirudin for Primary Percutaneous Coronary Interventions: Outcome Assessment in the Ottawa STEMI Registry”

We welcome the insightful and constructive comments by our colleagues, Dr Thomas Johnson et al, with regard to our recent report on bleeding outcomes with various antithrombotic regimens used in the Ottawa ST-segment–elevation myocardial infarction registry.1 As highlighted in our article, patients were recruited between 2006 and 2010, during which the publication of the Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI)2 study occurred, resulting in an increased use of bivalirudin by operators in our center. Moreover, as a nonrandomized study, important differences in bleeding reduction strategies, such as radial access,3 existed between the groups. Interestingly, despite these potential limitations, our event rates closely reproduced those in the HORIZONS-AMI study, and neither aspiration device use nor the time of enrollment was significantly related to our primary outcome. Thus, although we fully agree that cautious interpretation of the data are needed, we are buoyed by the fidelity with which our findings closely parallel to those from randomized clinical studies.

Our colleagues raise 2 interesting points with regard to complementary pharmacotherapy. First, in our registry, patients receiving bivalirudin received a bolus of heparin at the point of medical contact, which Johnson et al suggest may limit the reductions in bleeding. However, in our report,1 a significant reduction in bleeding was maintained over glycoprotein IIb/IIIa inhibitor and heparin, despite the upfront heparin bolus. Second, data from the Swedish Coronary Angiography and Angioplasty Registry4 compared bivalirudin with bivalirudin and heparin, demonstrating a reduction in death or definite target lesion thrombosis with addition of heparin but no significant change in bleeding (1.2% versus 1.6%; P=0.41). Thus, we contend that current evidence does not suggest that addition of heparin negates important reductions in bleeding outcomes.

Perhaps, most concerning was the high rate of acute stent thrombosis seen with bivalirudin in our study (1.6% bivalirudin versus 0.6% glycoprotein IIb/IIIa inhibitor plus heparin versus 0.3% heparin). We are, however, encouraged by real-world data, which suggests that newer more potent thienopyridines may attenuate the clear increased risk of stent thrombosis associated with bivalirudin use in primary percutaneous coronary intervention.5 Indeed, the more rapid onset of P2Y12 inhibition achieved with newer agents may result in important reductions in this critical end point.

In conclusion, we unequivocally agree with our colleagues that contemporary trials using the newest pharmacotherapy and techniques are needed to achieve the best possible outcomes definitively for patients undergoing primary percutaneous coronary intervention.

Disclosures

None.

References


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