The Comparison of Intracoronary Versus Intravenous Eptifibatide Administration during Primary Percutaneous Coronary Intervention of Acute ST-Segment Elevation Myocardial Infarction

Morteza Safi MD[1], Mohammad Hasan Namazi MD[1], Hosein Vakili MD[1], Habibollah Saadat MD[1], Ramin Khameneh Bagheri MD[2*], Javad Ramezani MD[2], Mostafa Ahmadi MD[3], Amin Sahebi MD[4]

[1]Cardiovascular Department, Shahid Beheshti Cardiovascular Research Center, Shahid Beheshti University of Medical Science, Tehran, Iran.
[2] Cardiovascular Department, Emam Reza Educational, Research and Treatment Center, Mashhad University of Medical Sciences, Mashhad, Iran.
[3] Cardiovascular Department, Ghaem Educational, Research and Treatment Center,Mashhad University of Medical Sciences, Mashhad, Iran.
[4]Cardiovascular surgery Department, Emam Reza Educational, Research and Treatment Center, Mashhad University of Medical Sciences, Mashhad, Iran.
drramin2004@yahoo.com

Abstract: Background: Administration of the glycoprotein IIb/IIIa inhibitors, including eptifibatide is an effective adjunctive treatment strategy during primary percutaneous coronary intervention (PPCI) for ST-segment elevation myocardial infarction. Recent data suggest that the intracoronary administration of these drugs during PPCI may increase the efficacy of them. **Methods:** A total of 40 ST-segment elevation myocardial infarction patients undergoing PPCI within 12 hours of symptom onset were randomized to either intracoronary or intravenous two boluses of eptifibatide (0.180 µg/kg) each 10 minutes. The primary endpoints of the trial were enzymatic infarct size, myocardial reperfusion measured as ST-segment resolution (STR), and post-procedural Thrombolysis in Myocardial Infarction (TIMI) grade flow of infarct related artery. The secondary endpoints were intra-procedural adverse effect (arrhythmia) and no-reflow phenomenon, in-hospital mortality, reinfarction , hemorrhage and post-procedural global systolic function. **Results:** Post-procedural TIMI grade 3 flow was achieved in 95% and 90% of the intracoronary (IC) and intravenous (IV) groups (P=0.61). The enzymatic infarct size assessed by the area under the curve of creatine phosphokinase-mb (CPK-mb) in the first 48 hours after PPCI ($\mu mol.L^{-1}.h^{-1}$) was similar in the IC and IV groups with 7206 (IQR, 5346.75 to 10384.50) versus 7294 (IQR , 10384.50 to 10384.50), P=0.87. Complete STR was achieved in 55% and 40% of the IC and IV groups (P=0.27). No deaths, urgent revascularizations, reinfarctions, or TIMI major bleeding events were observed among the both groups. **Conclusion:** Although, the IC administration of eptifibatide is safe, but does not add a benefit in comparison to the standard IV route.

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Introduction

Primary percutaneous coronary intervention (PCI) is the treatment of choice in the management of acute ST-segment elevation myocardial infarction (STEMI). It has been constantly observed that, despite restoring good epicardial flow with PCI, myocardial perfusion at the cellular level remains impaired in nearly 50 % of STEMI patients.[1] This is attributable to embolisation of the coronary thrombus into the distal vasculature, producing microvascular plugging, vasospasm, interstitial oedema and cellular injury. With Doppler guidewire technology, it was estimated that an average of 25 embolic events occurred during primary PCI for ST-segment elevation myocardial infarction. [2,3,4] There is consequently less salvage of infarct size, reduced left ventricular function and poorer clinical outcomes. There have been efforts to identify mechanical and pharmacological strategies to improve myocardial perfusion after primary PCI. Compared with systemic administration of intravenous pharmacotherapies, highly localized administration of intracoronary pharmacotherapy may be associated with a several-hundred-fold increase in the local concentration of an agent in the epicardial artery and microcirculation. A number of pharmacotherapies, including adenosine[5,6], calcium channel blockers[7], vasodilators[8,9],