

Summary

Analysing of different schemes of clopidogrel therapy after intracoronary stent implantation

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Based on data of the Clopidogrel Registry, pretreatment using a loading dose of 300 mg and having been administered 6-24 hours prior to the planned PCI has minimal effectivity over loading treatment having been applied at time of the procedure, this effectivity is reached at the expense of the increase of drug-induced bleeding events. Our results match with the current therapeutic recommendations. Both in vitro data and marked inhibition of platelet aggregation experienced 4 hours after loading dose administration and observed during optical aggregometry support the relative efficacy of „ad hoc” treatment. This efficacy, however, on examination of combined endpoints lags slightly behind the efficacy of pretreatment. Based on all these data, clopidogrel pretreatment at least 6 hours pre-PCI is mainly suggested for patients having stable angina, while it is no sense postponing the PCI for a later date after termination of diagnostic coronary angiography due to the satisfactory relative efficacy of „ad hoc” loading.

The results of platelet marker tests clearly proved that in patients with stable angina or after stabilization of those ACS patients, who have undergone successful intracoronary stent implantation, the administration of a 300 mg clopidogrel loading dose at the time of intervention effectively and promptly inhibits platelet activation markers and is as effective as the pretreatment. P-selectin and LAMP-3 expression were significantly inhibited by the clopidogrel loading dose of 300 mg after 1 hour, the number of GPIIb/IIIa receptors decreased after 2 hours, and the initially depressed platelet count normalized after 4 hours. We also found that our results were not biased by aspirin non-responsiveness. Our results emphasize the variable sensitivity of different platelet activation markers toward ADP receptor blockers, primarily inhibition of expression of cell surface markers (p-selectin, LAMP-3) could be observed having been released from intracellular storing granules during platelet activation. Our data also point to the beneficial effect of clopidogrel treatment in reducing platelet reactivity during stenting. The minimal advantage observed in our clinical trial for the favour of pretreatment and thus being consistent with the current therapeutic recommendations call attention to the emphatic role of other factors bearing a part in developing stent thrombosis.