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## CLOPIDOGREL LOADING WITH TIROFIBAN TO ARREST THE REACTIVITY OF PLATELETS IN PATIENTS WITH ACUTE

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**Objectives** we performed a prospective randomised pharmacodynamic investigation of four antiplatelet regimens to compare different effects in arresting the reactivity of platelets by assessed the

P-selectin expression on platelet membrane and the plasma levels of sCD40L and PDGF-BB. Meanwhile, we compared end events between groups during 6-months follow-up.

**Methods** We enrolled eighty patients with acute ST segment elevated MI according to the American Heart Association/American College of Cardiology criteria. Patients undergoing primary percutaneous coronary intervention were enrolled in a 2×2 factorial study (group A: clopidogrel 300 mg; group B: clopidogrel 600 mg; group C: clopidogrel 300 mg plus tirofiban; and group D: clopidogrel 600 mg plus tirofiban). All patients were aged >18 years and pretreated with 300-mg aspirin loading dose. The clopidogrel loading dose was given to all patients immediately after diagnosed and was followed by 75 mg daily. Tirofiban was administered as a bolus (10 μg/kg) followed by an infusion (0.15 μg/kg per minute) for 36 h after the procedure. We initiated cardiovascular intervention within 2 h after clopidogrel pretreatment. In addition, all patients had received β-blockers coadministrated with 300 mg aspirin before catheterisation and 100 mg was administered daily thereafter. Enzyme linked immunosorbent assay was used to assess circulating levels of sCD40L and PDGF-BB. Flow cytometry were used to assess platelet reactivity. We obtained the venous blood sample at three point of time: before pretreatment, 24 h and 5 days after intervention, respectively.

**Results** (1) Posttreatment P-selectin expression was significantly reduced in all groups compared with baseline expression, whereas treatment with 300-mg clopidogrel alone had the least effect in P-selectin expression. In the groups not treated with tirofiban, a 600-mg loading dose of clopidogrel provided greater platelet inhibition throughout the first 24 h after stenting, whereas this effect to some extent was attenuated 5 days after intervention. Inhibition of P-selectin positive platelet was higher in patients treated with tirofiban plus clopidogrel compared with clopidogrel alone ($p<0.05$).

**Conclusions** A strategy of parenteral GPIIb/IIIa inhibitor plus high-dose (600-mg) clopidogrel pretreatment administration is associated with superior platelet inhibition and lower MACE occurring compared with a strategy of high-dose (600-mg) or standard-dose (300-mg) clopidogrel loading alone. In the absence of a GPIIb/IIIa inhibitor, 600-mg clopidogrel pretreatment provides better platelet inhibition than standard 300-mg dose. Administration with clopidogrel of more than 75 mg daily after loading dose will provide surperior inhibition of the proliferative response of vascular smooth muscle cells after PTCA. These results require confirmation in a large-scale clinical trial.