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Platelet ADP receptor blocking effect of high dose clopidogel in patients with acute coronary syndrome

Mohamed Omar El Ghonemi

Cairo, Egypt

The rationale for using a 300 mg clopidogrel LD in clinical practice derives from dose finding studies performed mainly in healthy volunteers. Platelet reactivity in patients with coronary atherosclerosis may be significantly different from that of healthy individuals. In fact, in this clinical setting there is an increased thrombotic *milieu* not observed in healthy individuals due to several factors, such as the presence of activated platelets or increased platelet reactivity induced by coronary stenting and/or heparin administration.16 In addition, drug-drug interactions inhibiting clopidogrel activation by the hepatic cytochrome P450 (CYP) 3A4, genetic polymorphisms (GPIIb/IIIa receptor, GPIa receptor, P2Y12 receptor),and signalling defects may also modulate clopidogrel-induced antiplatelet effects**.** Recent data have demonstrated an interindividual variability of platelet inhibition in acute coronary syndrome patients receiving a standard 300 mg clopidogrel LD. In particular, approximately one-third of patients may have a suboptimal antiplatelet response early after intervention using this treatment regimen. Although clopidogrel response improves over time a high clopidogrel LD regimen has been suggested to optimise this response more rapidly***)*** However, the effect of a high clopidogrel LD on platelet reactivity, interindividual variability of platelet inhibition and number of clopidogrel responders has still not been well defined. Thus, the aim of this study was to compare platelet function profiles in patients receiving a standard 300 mg clopidogrel LD with that obtained following a 900mg LD and to assess variability in platelet inhibition as well as the clinical outcome and occurrence of complications.