Thromboelastography for the Assessment for Clopidogrel /Aspirin Resistance in Patients Following Primary PCI for STEMI - Preliminary Results

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Background: There is growing evidence that substantial portion of STEMI patients successfully treated by primary PCI will not respond adequately to Aspirin and/or Clopidogrel in standard doses, and will suffer recurrent events.

Thromboelastography (TEG) may be a valuable tool to assess the response to antiplatelet therapy. However, there is little data on its use in STEMI patients undergoing PCI. Our study is designed to assess platelet response to aspirin and clopidogrel by TEG in this group. Patients in the 3rd and 4th quartile according MA-thrombin >69mm (High MA), were at greater risk of recurrent ischemic events, the 4th quartile (MA >72mm) imposed the greatest risk. On-treatment platelet reactivity after stimulation with ADP or ASA is utilized to assess proper response to therapy (<70% for clopidogrel; <50% for aspirin). Based on these data we considered reasonable to suggest the goal for on-treatment platelet inhibition of MA-ADP <50.4mm and MA-AA <36mm in these high risk patients.

Methods: Fifteen patients with STEMI treated by PCI with BMS were enrolled so far. All received ASA 200 mg/d for 1 month followed then by 100mg/d, and Clopidogrel 75mg/d after loading dose, all medications on a fixed time-point. TEG analysis was performed just before the next ASA and Clopidogrel dose ("trough"), on the day of discharge (or 5th day of hospitalization) – Visit 1, on day 14 – Visit 2, and is planned on day 90 – Visit 3. On every visit, parallel to TEG, serum was separated and frozen for further pharmacokinetic analysis.

Results: Visit 1: 7 of 15 patients (46.7%) were in High MA group. Four (27%) were clopidogrel non-responders (MA-ADP >50.4mm), three of them in High MA group, all presented normal (<70%) on-treatment reactivity. One clopidogrel non-responder was in the 1st quartile (MA-thrombin 62.1mm, 81.3% on-treatment reactivity). There were two cases of aspirin non-responsiveness (13%) one - also clopidogrel non-responder.

Visit 2: Data on nine patients (one refused follow up). No change in medications or dosage was reported. Seven patients were in High MA group, six of them remained in High MA from their first visit. However, of three clopidogrel non-responders (30%) found on Visit 2, only one preserved his pattern of response, two others either lost or gained responsiveness to clopidogrel compared to previous visit. Two previously good aspirin responders on visit 1 appeared non-responders on visit 2.

Within 3 months two events were observed. One, who is combined aspirin-clopidogrel non-responder, suffered recurrent ischemic event (hospitalization for UAP). Additional patient, who was good responder initially, suffered ischemic CVA. At the point of the event he shifted from 3rd to 4th quartile and turned combined aspirin-clopidogrel non-respoder.

Conclusions: These preliminary results show that a large portion of patients with STEMI (47%) are in substantial risk of recurrent ischemic events. Longer observation in perspective of clinical outcome is needed to select the candidates for dose modification and to estimate if the change in treatment regiment will prevent recurrent ischemia.