Association of Lipoprotein(a) levels with intrinsic and on-clopidogrel platelet reactivity

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Background: Lipoprotein(a) [Lp(a)] is an independent, genetic, and causal risk factor for premature cardiovascular disease (CVD). Laboratory data have suggested an interaction of Lp(a) with platelet function, potentially caused by its structural similarity to plasminogen. So far, the potential association of Lp(a) with platelet activation and reactivity has not been well established in larger clinical cohorts.

Methods: This secondary analysis of the EXCELSIOR study analyzed intrinsic platelet reactivity before loading with clopidogrel 600mg and on-treatment platelet reactivity tested 24 hours following loading in patients undergoing elective coronary angiography. Platelet reactivity was tested by optical aggregometry as final aggregation after 5 min following stimulation with 5μM ADP. Platelet reactivity was also assessed by flow cytometry (expression of CD62P and PAC1) following stimulation with ADP and TRAP. Levels of Lp(a) on admission of each patient were immediately measured from fresh samples in a central laboratory.

Results: The present analysis included 2046 patients. Levels of Lp(a) ranged between 0 and 332 mg/dl. Results for intrinsic (p=0.80) and on-clopidogrel platelet reactivity (p=0.81) did not differ between quartiles of Lp(a) levels (Figure). Flow cytometry analyses confirmed these findings.

Conclusion: The present data do not support the hypothesis of an interaction of Lp(a) with platelet function. This finding might be important to define the safety of evolving therapeutic options for lowering Lp(a).