PREDICTORS OF HIGH THROMBIN-INDUCED PLATELET-FIBRIN CLOT STRENGTH IN PATIENTS TREATED WITH PCI

Background: High thrombin-induced platelet-fibrin clot strength (MATHROMBIN) measured by thrombelastography is associated with the risk of adverse clinical event occurrence. However, predictors and correlation with “HPR to ADP” of high MATHROMBIN are not undetermined.

Methods: Among PCI patients (n=197), we evaluated 5 μM ADP-platelet aggregation (PA) measured by conventional aggregometry and MATHROMBIN during clopidogrel treatment. Genotyping of CYP2C19*2, ABCB1 C3435T, and PON1 Q192R was performed. “HPR to ADP” was defined as 5μM ADP-PA ≥ 46% (consensus criteria).

Results: In ROC curve analysis, MAKH ≥ 68 mm was identified as the corresponding point to “HPR to ADP” (AUC, 0.709; 95% CI, 0.628-0.789; p < 0.001). Combination of high MATHROMBIN (≥ 68 mm) + CYP2C19*2 allele was associated with an increased risk of HPR (OR, 13.89; 95% CI, 3.41 to 55.56; p<0.001). In multivariate analysis, female (OR, 3.92; 95% CI, 1.94 to 7.87; p < 0.001), platelet count (per 104/mm3: OR, 1.08; 95% CI, 1.02 to 1.15; p = 0.009), anemia (OR, 0.37; 95% CI, 0.17 to 0.80; p = 0.011), LV ejection fraction ≤ 45% (OR, 0.28; 95% CI, 0.09 to 0.82; p = 0.020), and “HPR to ADP” (OR, 2.58; 95% CI, 1.14 to 5.81; p = 0.022) determined the risk of high MATHROMBIN.

Conclusions: This is the first study to demonstrate the determinants of thrombin-induced platelet-fibrin interaction in PCI-treated patients. The utility of combining measures of “HPR to ADP” and high MATHROMBIN may cover multiple aspect of hemostasis and bleeding.