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β Fibrinogen gene -455 G/A polymorphisms as Determinants of Ischemic Stroke Outcome Severity in Response to Aspirin Treatment

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Abstract : Stroke is a leading cause of disability and death. For secondary treatment, aspirin is the agent of choice. Polymorphisms in the promoter region of β fibrinogen gene -455 G/A are associated with increased plasma fibrinogen levels. We evaluated the correlation between β fibrinogen gene-455 G/A promoter polymorphisms and ischemic stroke (IS) outcome on modified Rankin Scale (mRS) in patients treated with aspirin by age groups. A Cohort study design was adopted comprising 136 patients of younger (<55) and older ages (>55). Patients used aspirin for 3 months after IS and completed a detailed stroke-outcome questionnaire. Overall, genotype distribution was 66.2% for GG, 27.2% for GA and 6.6% for AA. The young-age group showed genotypes GG, GA and AA as 30.1%, 15.4% and 6.6%, respectively. After aspirin administration, plasma fibrinogen levels dropped to 248.65±100.71 mg/dl and 235.75±82.01 mg/dl in young and old age groups, respectively. Low and high plasma fibrinogen levels were determined on mRS with a cut-off value of 268.05 mg/dl. In a logistic regression model, the -455 G/A locus genotype showed a significant correlation with age and plasma fibrinogen levels on mRS at day 0 (P<0.25). Poisson Confidence Intervals (PCI) were calculated. Age relative risk was 0.67 (PCI: 0.30-1.49), while fibrinogen-mRS relative risk was 0.78 (PCI: 0.37 – 1.63) at day 0. Aspirin significantly (P<0.05) decreased plasma fibrinogen levels in correlation with promoter polymorphisms in an age-dependent fashion. Significant differences between high and low plasma fibrinogen levels before and after the use of aspirin coincided with substantially reduced mRS scores.
Keywords: β fibrinogen -455 FGB; aspirin; stroke outcome; ischemic stroke risk.

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