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Identification of F2RL3 Gene Methylation Induced by Cigarette Smoking in Acute Myocardial Infarction

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Abstract : Acute Myocardial Infarction (AMI) has complex pathophysiology and influenced by multiple factors. Several recent studies at an epigenetic level conclude that AMI has a strong correlation with changes in methylation patterns of genes associated with IMA, one of which is the F2RL3 gene. This gene encoding for PAR-4, of the receptor for thrombin and widely involved in the several mechanisms of platelet activation, coagulation and has a strong correlation with atherosclerosis. The aim of this study is to investigate the correlation of methylation level on F2RL3 gene caused by cigarette smoking on AMI events. Sixteen AMI patients were collected from Saiful Anwar Hospital, Malang, Indonesia. The whole genome was extracted from patient's blood, then converted with bisulfite conversion method. CpG island in cg03636183 from F2RL3 gene was amplified with a pair of specific primers to obtain specific amplicon containing CpG island. This amplicon then performed with direct sequencing and analyzed by Sequence Scanner v1.0 and BioEdit software. Our result shows that eight AMI patients (50%) having a smoker while eight others (50%) nonsmoker. Three CpG sites can be analyzed which is CpG site 2, CpG site 3, and CpG site 4. There are no differences between methylation level and pattern of F2RL3 gene on AMI patients that induced by cigarette smoking which each group has same methylation levels up to 91,67% respectively. DNA methylation is a complex epigenetic mechanism which is influenced by various factors, not cigarette smoking alone.
Keywords: Acute Myocardial Infarction, cigarette smoking, methylation level of F2RL3 gene.

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