ABSTRACT

EFFECT OF STREPTOKINASE ON sST2 LEVEL IN PATIENT WITH ACUTE MYOCARDIAL INFARCTION

BACKGROUND: Acute myocardial infarction is an ischemic heart disease, in which there is imbalance between oxygen supply and demand to cardiac muscle, due to thrombus, atherosclerosis, or another condition that disrupt coronary blood perfusion. Ischemic condition will eventually result in myocytes death, aggravating workload of remaining myocytes. Mechanical tension will stimulate release of markers. Suppression of Tumorigenicity 2 (ST2) is a receptor for IL-33 that is expressed by cardiomyocyte as a celullar damage response. Soluble form of ST2 (sST2) can be measured to predict the condition. Streptokinase is an widely used fibrinolytic agent that is aimed to reperfuse cardiac muscle.

OBJECTIVES: This study was aimed to analyze the effect of streptokinase on sST2 level, as a cardiac marker, in patients with acute myocardial infarct.

METHODS: This was a prospective observational study with one group pre-post test design. Conducted in ICCU of Dr. Soetomo Teaching Hospital from July-November 2017 after recieving ethical clearance from hospital's committee. Patients colleted using consecutive sampling. sST2 were measured before and 6 hours after streptokinase administration using using Quantikine® ELISA: Human ST2/IL-33 R Immunoassay. Paired t test then used to compare sST2 pre and post concentration

RESULT: 12 patients were included (11 male, 1 female, with average age 53 years old). sST2 concentration 6 hours after streptokinase significantly increase, with mean concentration of 94.7 7 ± 77.5 ng/mL became 534,8 ± 526,8 ng/mL (p=0,011). As a prognostic marker, sST2 has a cut off point that predict mortality and developing heart failure risk in 30 days and one year, that is 35 ng/mL. In this study, 4 patients had sST2 concentration (at admission) lower that 35 ng/mL.

CONCLUSION: There was an increase between sST2 concentration before and after streptokinase.

KEYWORDS: Suppression of Tumorigenicity 2; soluble ST2; Acute Myocardial Infarct; Streptokinase; Reperfusion