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The relationship of Soluble Suppression of Tumorigenicity 2 (sST2) and Troponin T (TnT) levels in Acute Myocardial Infarction (AMI) patients at Sanglah General Hospital, Bali, Indonesia

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ABSTRACT

Background: Acute Myocardial Infarction (AMI) as cardiovascular disease is a significant cause of death worldwide and requires examining cardiac biomarkers such as Troponin T (TnT) for diagnosis. However, increased troponin levels are often found in non-AMI patients. Soluble Suppression of Tumorigenicity 2 (sST2) as a novel biomarker has known will increase after the occurrence of AMI. This study aims to evaluate the relationship of sST2 and TnT levels in AMI patients at Sanglah General Hospital, Bali, Indonesia.

Methods: The study was conducted among 61 participants during August-December 2020 by consecutive sampling. The samples were examined for TnT levels using a Roche Cobass h232 and sST2 with the Human sST2 Elabscience© Enzyme-linked Immunosorbent Assay (ELISA) Kit. Normality and correlation tests were performed between the two parameters. Data were analyzed using SPSS version 17 for Windows.

Results: The average age of participants was 59.5±10.0 years old. Most of the participants were male and had hypertension history in STEMI (71.0% and 67.7%) and NSTEMI (70.0% and 79.0%) groups. There is no significant difference in BMI between STEMI (23.5±3.1 kg/m^2) and NSTEMI (24.6±3.8 kg/m^2) (p=0.319). However, there was a significant moderate positive correlation between TnT and sST2 levels (r=0.394, p=0.002).

Conclusion: There was an increase in troponin T and sST2 levels in AMI patients at Sanglah Hospital. There is a significant positive correlation between sST2 levels and troponin T levels in AMI patients at Sanglah Hospital.

Keywords: AMI, Sanglah General Hospital, sST2, Troponin T.
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INTRODUCTION

Cardiovascular disease is a significant cause of death worldwide. The incidence of this disease is still increasing in developing countries and has a major impact on reducing the quality of life and a person's life expectancy.[1] Cardiovascular disease is the leading cause of morbidity and mortality, occurring in 1 in 6 deaths in the United States.[1,2]

Acute myocardial infarction (AMI) is one of the complications of coronary heart disease characterized by chest pain that most patients complain about. Based on data from the World Heart Organization (WHO), as many as 7.3 million deaths worldwide each year are caused by ischemic heart disease, as the leading cause of death in developing countries.[3,4]

Examination of cardiac biomarkers is important in diagnosing AMI, the degree of risk factors, monitoring of medical care and adjusting therapy.[5,6] As the biomarker of choice, an increase or decrease in troponin levels helps diagnose AMI. However, an increase in troponin levels in non-AMI patients is often found, as well as the development of a more sensitive cardiac troponin (cTn) test. Troponin can be detected in all people and levels will increase above the 99th percentile in a stable chronic condition.[7,8]

Weinberg et al. introduced Soluble Suppression of Tumorigenicity 2 (sST2) as a novel biomarker that increases its levels after AMI. Weir et al. found an association of sST2 and infarct magnitude/evolution in AMI patients with left ventricular systolic dysfunction for 24 weeks.[9] The use of biomarkers, such as sST2 as a marker of death in AMI, is associated with cardiac remodeling due to the overproduction of fibrous tissue.[10] Studies examining the role of sST2 and its relationship with TnT as a diagnosis of AMI have not been widely performed. This study aims to determine the role of sST2 and its relationship with troponin T in AMI patients at Sanglah Hospital Denpasar so that it is expected to assist in diagnosis and assist clinicians in

providing further patient care.

Based on those mentioned above, this study aims to describe the levels of sST2 and troponin T and their relationship in AMI patients at Sanglah Hospital Denpasar.

METHODS

This study is an observational analytic study with a cross sectional method conducted at Sanglah Hospital from July to December 2020. A total of 61 patients were selected by consecutive sampling as study participants after meeting the inclusion criteria, namely at least 18 years of age with a diagnosis of AMI no more than 2 times based on a heart and blood vessel specialist and met the exclusion criteria, namely, the patient had a history of more than 2 AMI attacks, heart failure, stroke, inflammation, Systemic Lupus Erythematosus (SLE) disease. Blood samples were taken as much as 6 ml in the first 24 hours after the subject was diagnosed with AMI, each 3 mL for troponin T examination and 3 mL for sST2 examination.

Examination of troponin levels was measured by the double antibody sandwich method.[11] The sample used was 3 mL of venous blood plasma, which was accommodated in a heparin tube and then immediately examined using a Roche Cobas h232 device. The sST2 level as measured by the Human sST2 Elabscience© Enzyme-linked Immunosorbent Assay (ELISA) Kit using the double sandwich antibody ELISA method.[12] The sample used was venous blood plasma using a tube with 3 mL Ethylene Diamine Tetraacetic Acid (EDTA) anticoagulant. The recommended cut-off point for sST2 levels is 35 ng/mL; in normal individuals, 90 - 95% of sST2 levels are lower than 35 ng/mL.[13] Quality assurance of ELISA examination is carried out by testing precision and accuracy. The precision test method is the within run method and the accuracy test is the spike and recovery method.

The data of this study were statistically analyzed with SPSS for Windows version 17.0. All data obtained in this study were analyzed descriptively and the results will be presented in the form of mean±standard deviation (SD) and median (minimum-maximum). The respondent's characteristic data is displayed in the form of absolute numbers and percentages. The normality test of the data used the Saphiro-Wilk test followed by the Spearman correlation test to determine the relationship between sST2 levels and troponin T levels with a significance level of $p < 0.05$.

Table 1. Baseline characteristics of respondents

Variable	STEMI (n=31)	NSTEMI (n=30)	Total (n=61)	p
Age (Years) (mean±SD)	60.0±10.6	59.1±9.6	59.5±10.0	0.720
Gender, n (%)				
Male	22 (71.0)	21 (70.0)	43 (70.5)	0.934
Female	9 (29.0)	9 (30.0)	18 (29.5)	
BMI (kg/m^2) (mean±SD)	23.5±3.1	24.6±3.8	24.1±3.5	0.319
History of Disease, n (%)				
Hypertension	21 (67.7)	21(79.0)	42(68.9)	0.849
DM	7 (22.6)	15(50.0)	22(36.1)	0.026*

STEMI: ST-*Elevation Myocardial Infarction;* NSTEMI: *Non-ST-Elevation Myocardial Infarction*; SD: Standard Deviation; BMI: Body Mass Index; DM: Diabetes Melitus; *Statistically significant if p-value less than 0.05.

Table 2. The TnT and sST2 levels between groups

Variable	STEMI (n=31)	NSTEMI (n=30)	Total (n=61)	p
TnT (ng/L)				
Median	557	334	468	0.051
IQR *range* (min-max)	113-1866	101-1831	101-1866	
sST2 (ng/mL)				
Median	52.2	55.5	53.4	0.902
IQR *range* (min-max)	22.5-67.4	25.6-63.9	22.5-67.4	

STEMI: ST-*Elevation Myocardial Infarction;* NSTEMI: *Non-ST-Elevation Myocardial Infarction*; TnT: Troponin T; sST2: *Soluble Suppression of Tumorigenicity* 2; IQR: *Interquartile range;* *Statistically significant if p-value less than 0.05.

Table 3. Spearman correlation test between TnT and sST2

Variable	sST2		
	n	r	p
TnT	61	0.394	0.002*

TnT: Troponin T; sST2: *Soluble Suppression of Tumorigenicity* 2; *Statistically significant if p-value less than 0.05.

RESULTS

The characteristics of the study sample based on age, gender, BMI, history of hypertension and DM did not show any significant difference between the STEMI and NSTEMI groups (p>0.05) (Table 1). There was no significant difference in age between the two groups, with the average age of the study participants being 59.5±10.0 years old. There was no difference in the prevalence of sex in the two groups (p=0.934). Most of the participants were male (70.5%). The highest BMI was 34.72 kg/m2 and the lowest was 17.48 kg/m2 with an average BMI of 24.1 kg/m2±3.5 SD. A total of 42 people (68.9%) had a history of hypertension and 22 people (36.1%) had a history of DM. The characteristics of the research participants are shown in Table 1.

The results of the examination of TnT and sST2 levels in 61 participants with a diagnosis of AMI are shown in Table 2. The median value of TnT levels was 468 ng/L. The study data showed that the highest troponin levels were found in patients with a diagnosis of STEMI. The median value of sST2 levels was 53.4 ng/mL, with the lowest value of 22.5 ng/mL

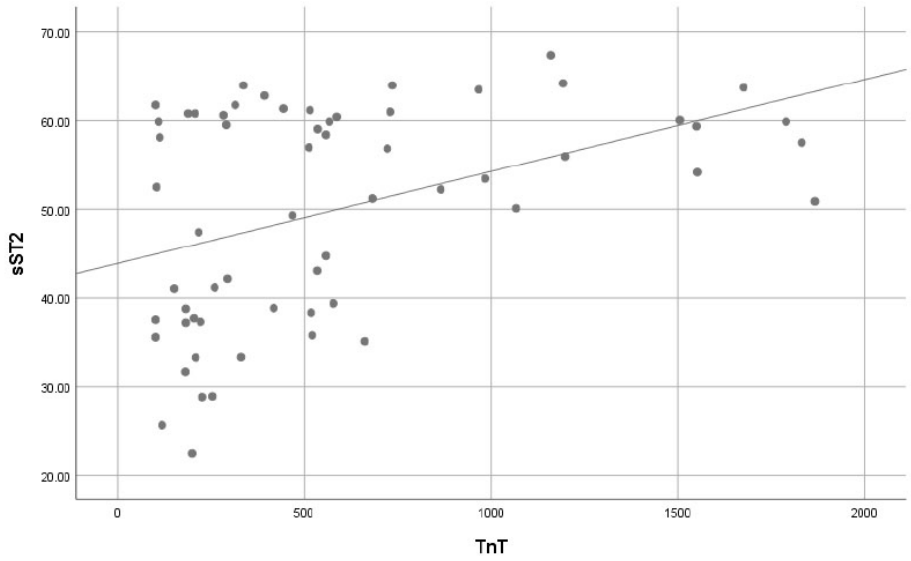

Figure 1. Scatter plot graph of sST2 and TnT correlation on AMI.

and the highest value of 67.4 ng/mL. With the cut-off value recommended by several previous researchers, namely >35 ng/mL, this study found an increase in sST2 levels in 51 people (83.6%) (Table 1).

The Spearman correlation analysis test results to determine the relationship between sST2 and TnT levels in AMI patients are shown in Table 3. Spearman correlation test showed that there was a significant moderate positive correlation between levels of TnT and sST2 (r=0.394, p=0.002). This means that the higher the TnT level, the higher the sST2 level in AMI patients (Table 3). The scatter plot graph in Figure 1 shows a moderate positive correlation between TnT and sST2 levels in AMI patients.

DISCUSSION

The mean age in this study was 59.5 ± 10.0 years, most of whom were male (70.5%). According to previous studies, advanced age and male gender are important factors that determine will affect the prognosis of death.[14,15] Gender has a strong influence on sST2 levels, with lower levels in women than men. This difference in levels is because androgen hormones influence the synthesis or secretion of sST2. Data provided by a previous study also showed that the lowest levels of sST2 were found in female patients receiving estrogen therapy.[16]

The relationship between overweight and obesity with AMI is still controversial. In recent years, obesity and overweight rates have increased and are associated with Diabetes Mellitus (DM), metabolic syndrome, hypertension and cardiovascular disease.[17] The average BMI in this study is not in accordance with previous studies conducted by Stepien M et al. in 65 obese patients.[18] The study found a high mean BMI, but no significant relationship between BMI and morbidity and mortality in AMI.[18] Male patients with normal BMI, overweight, and obesity have the same risk of death, while overweight women have a lower risk than women with normal BMI based on a study conducted by Danish.[19,20]

Hypertension is a significant risk factor as a cause of atherosclerosis which is the etiology of AMI. Hypertension will increase myocardial pressure and cardiac myocyte sST2 levels due to acute or chronic mechanical strain on blood vessels.[21,22]

In this study, 36.1% of patients with a history of DM were found. Several studies have shown an association between AMI, sST2 and glucose. Higher levels of sST2 have been found in patients with DM, although the mechanism remains unclear. This may occur because of the long-term inflammatory and remodeling processes in DM patients. Conditions of hyperglycemia, insulin resistance, high production of fatty acids will cause oxidative stress and increase the inflammatory response. This mechanism will lead to coronary atherosclerosis and microvascular dysfunction, which are risk factors for AMI.[23,24]

Troponins are protein complexes found in skeletal muscle and cardiac myocytes, whose function is to regulate or inhibit the contraction of these muscles. Troponin T is found in cardiac muscle and is immediately released into the circulation during myocyte damage.[7,25] Based on the European Society of Cardiology (ESC) definition of acute myocardial infarction, all Unstable Angina (UA) patients with elevated cTn levels will be diagnosed with acute NSTEMI (30% of cases). UA will change to NSTEMI). After the onset of AMI, TnI and TnT levels will increase (starting with the free cytosolic fraction). In the following 4-6 hours, they are detected in the blood, reach a peak in 1-2 days and decrease after 10 days. Patients with high cTn levels have a higher mortality rate.[25-28]

Cardiac and myocardial fibroblasts are the primary sources of sST2 production in response to injury or stress. Non myocardial sources are endothelial cells from the aorta and coronary arteries.[27,28] Myocytes will immediately secrete sST2 at the time of decreased myocardial function due to AMI. This is related to the production of pro-inflammatory cytokines due to the mechanical stretching of myocytes. ST2 is a gene responsive to mechanical stress on the myocardium during in vitro experiments in AMI and heart failure conditions.[27-29] sST2 is closely involved in left ventricular hypertrophy, fibrosis and remodeling through its interaction with IL-33. sST2 levels in several reports have been reported to increase significantly after an AMI attack and in patients with chronic heart failure. Blood samples for sST2 examination were serum, lithium heparin plasma, and EDTA plasma.[30-32]

Elevated sST2 levels are associated with ongoing myocardial injury or mechanical strain to the left ventricle and associated release of sST2 into the circulation after an AMI.[33-35] Higher sST2 levels have been found in patients with a diagnosis of STEMI than in NSTEMI. In experimental

studies, sST2 levels increase immediately after infarction, with maximum levels detected 12-18 hours after onset.[35,36]

Spearman correlation test between levels of sST2 and TnT obtained significant positive results. Myocardial injury in AMI will cause activation of pro-inflammatory receptors resulting in impaired myocardial oxygenation. Oxygen reserves will decrease due to increased ventricular wall stress and neurohormonal activity to improve contraction and heart rate. This results in myocardial ischemia, myocyte necrosis and apoptosis followed by the release of TnT into the circulation.[37,38] When cardiac ischemia occurs, pro-inflammatory cytokines are secreted by myocytes. In this condition, ST2 in the form of ST2L and sST2, which are pro-inflammatory mediators, will be involved in the occurrence of myocardial injury. The soluble form of ST2 is rapidly secreted by cardiac muscle cells when subjected to excessive loads, such as myocardial infarction. The binding of IL-33 to the ST2 membrane is due to an increase in myocardial biomechanical forces with anti-hypertrophic and anti-fibrotic responses. The cardiac protective effect will be inhibited by sST2, which prevents the increase of IL-33. This results in myocardial death and tissue fibrosis. Mechanical strain as myocardial compensation to meet oxygen demand will stimulate fibroblasts to produce sST2. If the strain or damage to the myocardium is prolonged, the secretion of sST2 by the myocardium will increase the level of sST2 in the blood.[2,38]

One of this study's limitations was that other inflammatory biomarkers were not examined, such as Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), or IL-6, to determine the occurrence of a systemic inflammatory process that could lead to an increase in blood levels of sST2.

CONCLUSION

In this study, there was an increase in TnT and sST2 levels in AMI patients at Sanglah Hospital. There is a significant positive correlation between sST2 levels and TnT levels in AMI patients at Sanglah Hospital.]

CONFLICT OF INTEREST

There is no competing interest regarding the manuscript.

ETHICS CONSIDERATION

Ethics approval has been obtained from the Ethics Committee, Faculty of Medicine, Universitas Udayana, Sanglah General Hospital, Bali, Indonesia, prior to the study being conducted.

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None.

AUTHOR CONTRIBUTION

All authors equally contribute to the study from the conceptual framework, data gathering, data analysis until reporting the study results through publication.

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