

The Difference in Endothelial Nitric Oxide Synthase Levels in STEMI Patients With Comorbid Diabetes Mellitus And Those Without Comorbid Diabetes Mellitus at RSUP. Dr. M. Djamil Padang

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ABSTRACT

Background: Cardiovascular disease is a leading cause of global mortality, with Indonesia ranking third in the highest cardiovascular deaths. Acute myocardial infarction, divided into STEMI and NSTEMI, is part of the acute coronary syndrome due to total occlusion of coronary arteries requiring immediate revascularization. Diabetes Mellitus reduces endothelial nitric oxide synthase (eNOS) and nitric oxide (NO) production, leading to endothelial dysfunction and an increased risk of vascular disease. DM-related endothelial dysfunction affects vascular relaxation and the formation of atherosclerotic plaques as well as thrombi in ST-elevation myocardial infarction (STEMI). Methods: This research was an analytical observational study with a cross-sectional approach, using normality tests and the Mann-Whitney U Test. The research sample was collected using consecutive sampling techniques and obtained from the medical records of 72 confirmed STEMI patients, with and without comorbid Diabetes Mellitus. Results: The research results indicated that the majority of patients were in the age range of 60-69 years (52.7%), male (86.1%), with a Body Mass Index (BMI) of 23-24.9 (45.8%), comorbid hypertension (52.7%), and a history of smoking (72.3%). The mean eNOS level in the control group was Median eNOS 38,839 U/mL with maximum level 584,762 U/ml and minimum 17,024 U/ml. Median eNOS level in the test group was 36,077 U/mL with the maximum level 679,754 U/ml and minimum 1,836 U/ml. Conclusions: There was no significant difference in eNOS levels between the two groups (Sig (2-Tailed) > 0.05).

INTRODUCTION

Data from the World Health Organization (WHO) in 2012 states that cardiovascular diseases are the leading cause of death globally, with Indonesia ranking third. Approximately 7,200,000 (12.2%) deaths worldwide are attributed to this disease (Tsao et al., 2023).

Acute Myocardial Infarction (AMI) has the highest case fatality rate (CFR) compared to other heart diseases (Puymirat et al., 2017). According to national epidemiological reports, several provinces in Indonesia demonstrate a relatively high prevalence of cardiovascular disease (Alhabib et al., 2019).

Acute Myocardial Infarction (AMI) is part of the Acute Coronary Syndrome (ACS). Acute Coronary Syndrome (ACS) is a cardiac emergency condition characterized by clinical manifestations such as discomfort in the chest or other symptoms caused by myocardial ischemia. Acute myocardial infarction is myocardial necrosis resulting from inadequate blood supply due to acute obstruction in the coronary arteries (Go et al., 2013). The occlusion typically occurs due to the rupture of an atheromatous plaque in the coronary arteries, followed by thrombosis, vasoconstriction, inflammatory reactions, and distal microembolization (Muttaqin, 2014).

Endothelial nitric oxide synthase (eNOS, NOS3) plays a crucial role in the production of nitric oxide (NO), an essential molecule that can act directly or indirectly as a vasodilator and anti-inflammatory mediator. Nitric oxide is a vasoactive enzyme produced by endothelial cells with antiproliferative effects on vascular smooth muscle cells. Optimal availability of NO for artery dilation supports normal endothelial repair, but an excess of NO can also lead to the production of free radicals. Nitric oxide is not only a potent vasodilator but also a strong anti-thrombotic agent, playing biological roles in digestion, respiration, the nervous system, and the immune system. The loss of endothelial ability to produce and release NO can trigger the atherogenic process (Darwin et al., 2018).

Diabetes mellitus (DM) is a metabolic disorder characterized by chronic hyperglycemia resulting from impaired carbohydrate, lipid, and protein metabolism due to inadequate insulin secretion, insulin action, or both. Patients with DM have a higher risk of acute myocardial infarction compared to non-DM patients (Romes, 2023). Diabetes mellitus can reduce endothelial nitric oxide synthase (eNOS). Nitric oxide is synthesized from L-arginine through eNOS. Reduced eNOS affects the effects of NO, leading to increased activity of the proinflammatory transcription. This enhances the migration of monocytes and smooth muscle cells into the intima, forming foam cells within macrophages, representing early morphological signs of atherosclerosis (Paneni et al., 2013). Major complications of DM include abnormalities in the function and structure of blood vessel walls, which can occur due to macro- and microvascular disturbances. Vascular disease is more common in patients with DM than in those without diabetes, and it remains a leading cause of death (Romes, 2023).

METHODS

Study Design and Population

This type of study is an observational study with a cross-sectional design, where independent and dependent variables are analyzed simultaneously. This research is a subgroup analysis of a previous study conducted by Dr. dr. Eka Fithra Elfi, Sp.JP(K)-FIHA, regarding the role of endothelial nitric oxide synthase and several other markers of endothelial dysfunction in AMI. The population in this study consists of patients with acute ST-segment elevation myocardial infarction (STEMI) and diabetes

mellitus who were treated at Dr. M. Djamil Padang Teaching Hospital. The comparative group consists of STEMI patients without diabetes mellitus. The population was extracted from the medical records data of the research who met the inclusion and exclusion criteria.

Procedure

Samples that meet the inclusion and exclusion criteria are consecutively taken as the test group and control group. Basic data, including demographics, cardiovascular risk factors, and diagnoses, are obtained from patient history and medical records. Other Laboratory examinations, such as electrocardiography and cardiac biomarker testing, are conducted upon patient admission. Blood samples for routine laboratory tests (random blood sugar, HbA1C, hematology, and lipid profile) as baseline data are taken at the time of admission and examined at the central laboratory of Dr. M. Djamil Padang Hospital. Blood samples for testing eNOS concentration are taken from patients within a 48-hour window after admission and analyzed in the Biomedical Laboratory of the Faculty of Medicine, Universitas Andalas.

Data Collection

After determining the independent and dependent variables, a data distribution analysis was conducted using a normality test to ascertain whether the data is normally distributed or not, employing the Kolmogorov-Smirnov test. The data was found to be not normally distributed ($\text{sig} > 0.05$) even after two rounds of data transformation. Subsequently, bivariate analysis was carried out to determine the relationship between the two groups, comparing patients with STEMI and DM with the control group of STEMI patients without DM using the Mann-Whitney U Test through the SPSS v26.

RESULTS AND DISCUSSION

Results

This study examines the differences in endothelial dysfunction markers, specifically eNOS, in patients with ST-segment elevation myocardial infarction (STEMI) with or without diabetes mellitus (DM). The research was conducted at Dr. M. Djamil Padang Teaching Hospital since November 2022, involving 36 research subjects with STEMI as the control group and 36 subjects with STEMI and DM as the test group, all meeting the inclusion and exclusion criteria. The total sample size for this study was 72 samples.

Table 1. Characteristics of Samples

	Total N = 72 (%)	STEMI without DM n = 36 (%)	STEMI with DM n = 36 (%)
Age (years)			
<40	1 (1,4)	1 (2,7)	0 (0)
40-49	8 (11,3)	1 (2,7)	7 (19,5)
50-59	20 (27,7)	9 (25)	11 (30,5)
60-69	37 (52,7)	21 (58,3)	16 (44,5)
≥70	6 (6,9)	4 (11,3)	2 (5,5)
Sex			
Male	62 (86,1)	34 (94,4)	28 (77,77)
Female	10 (14,9)	2 (5,6)	8 (22,23)
BMI (kg/m²)			
<18,5	0 (0)	0 (0)	0 (0)
18,5-22,9	28 (39)	15 (41,6)	13 (36,1)
23-24,9	33 (45,8)	20 (55,6)	13 (36,1)
25-29,9	9 (12,4)	1 (2,8)	8 (22,2)
≥30	2 (2,8)	0 (0)	2 (5,6)
Random Blood Sugar (mg/dl)			
<200	43 (59,7)	35 (97,3)	8 (22,2)
≥200	29 (40,3)	1 (2,7)	28 (77,8)
Other Risk Factors			
Smoking	52 (72,3)	30 (83,3)	22 (61,1)
Hypertension	38 (52,7)	20 (55,5)	18 (50)
Dyslipidemia			
LDL (mg/dl)			
<100	20 (27,8)	10 (27,8)	10 (27,8)
100-129	22 (30,4)	14 (38,9)	8 (22,2)
130-159	17 (23,7)	8 (22,2)	9 (25)
160-189	12 (16,7)	4(11,1)	8 (22,2)
>190	1 (1,4)	0 (0)	1 (2,8)
Triglycerides (mg/dl)			
<150	47 (65,3)	25 (69,4)	22 (61,1)
150-199	16 (22,2)	7 (19,4)	9 (25)
200-499	9 (12,5)	4 (11,2)	5 (13,9)
>500	0 (0)	0 (0)	0 (0)

Table description: Data is presented in number n (percentage).

BMI, Body Mass Index; LDL, low density lipoprotein; STEMI, ST- Segment Elevation Myocardial Infarction

Table 2. ENOS level (U/mL) STEMI Patients with Diabetes Mellitus

	Total (n)	Minimum	Maximum	Median
STEMI with DM	36	1,836	679,754	36,077

Table 2 shows the levels of eNOS (U/mL) in STEMI patients with comorbid DM. The samples obtained for the control group test are 36 samples. Based the table, The median of eNOS level in the subjects of this group is 36,077 U/mL.

Table 3. ENOS level (U/mL) STEMI Patients without Diabetes Mellitus

	Total (n)	Minimum	Maximum	Median
STEMI without DM	36	17,042	584,762	38,839

Table 3 shows the levels of eNOS (U/mL) in STEMI patients without comorbid DM. The samples obtained for the control group test are 36 samples. Based on the table 3, the median of eNOS level in the subjects of this group is 38,839 U/mL.

Table 4. Difference in eNOS Levels in STEMI Patients With and Without DM

	n	eNOS Level (U/ml)	
		Median	Sig (2-Tailed)
STEMI with DM	36	36,077	0,408
STEMI without DM	36	38,839	

Based on Table 4, the median eNOS level (U/mL) in patients with STEMI and DM is 36.077 U/mL, which is lower than in patients with STEMI without DM, which is 38.839 U/mL. According to the Mann-Whitney test, there is no significant difference in eNOS levels between patients with STEMI with and without comorbid DM because the Sig (2-Tailed) value is greater than 0,05.

Discussion

Basic Characteristic of Samples

The basic characteristics of the study sample include age, gender, body mass index, and dominant risk factors such as smoking and hypertension. These basic characteristics also encompass the history of STEMI, diabetes mellitus, and laboratory tests as markers of dyslipidemia, namely triglycerides and LDL. In the study, the majority of patients were in the age range of 60–69 years (52.7%), with an average age of 60 years, where the minimum age was 39 years and the maximum age was 83 years. This is consistent with research conducted by Susilo C, indicating that the risk of coronary heart disease increases with age, with the peak incidence occurring in the 50–60 age range (Susilo, 2015).

In this study, the majority of patients were male, with 62 individuals (86.1%), while there were 10 female patients (13.9%). This aligns with research conducted by Gayatri et al., where 87% of STEMI patients were male, and 72% were under the age of 65 (Gayatri et al., 2016). As mentioned by Susilo C, the peak occurrence of heart disease in men is between the ages of 50–60, while in women, it occurs between the ages of 60–70 (Susilo, 2015). This is attributed to sex hormones estrogen and

progesterone being produced from cholesterol, which protects the endothelium. Women experience heart disease with a delay of around 10–15 years compared to men, and their risk increases after menopause. Susilo C also notes that although the incidence in women is delayed by 10–15 years, heart attacks and sudden deaths in women tend to be more serious (Susilo, 2015).

Based on the research results in both groups, findings related to overweight or obesity concerning the body mass index (BMI) ratio were obtained. In this study, out of 72 patients, 45.8% (33 individuals) had a BMI ratio of 23–24.9, indicating overweight, followed by normal weight (BMI 18.5–22.9) in 39% (28 individuals). Haryuni explains that in patients with acute myocardial infarction, overweight or obesity is more commonly found than in those with normal weight. This may be attributed to excessive calorie intake and an unhealthy lifestyle (Haryuni, 2015). According to Cercato, obesity increases the risk of atherosclerosis due to excessive growth of adipose tissue, which can worsen inflammation and elevate levels of free radicals. Additionally, obesity is often associated with the occurrence of type 2 diabetes due to imbalances in the function of adipose cells and cell infiltration that can disrupt sensitivity, insulin secretion, and lead to increased blood glucose levels (Cercato & Fonseca, 2019).

Another discussed risk factor in STEMI in the study is dyslipidemia, and one of the studied markers of dyslipidemia is LDL (low-density lipoprotein) and triglyceride levels. The LDL levels in both groups are still considerably above the normal range recommended by NCEP ATP III 2001, with 22 individuals (30.5%) having LDL levels in the range of 100–129 mg/dl, while those with normal LDL levels/within the recommended range (<100 mg/dl) are 20 individuals (27.7%). Research by Hajar R on the risk factors for coronary heart disease explains the same, indicating a connection between the risk of heart disease and high LDL levels in the blood (Hajar, 2017). A different trend is observed in another dyslipidemia marker, namely triglycerides. Among the 72 samples studied, the majority of patients (47 samples) have normal/recommended triglyceride levels (<150 mg/dl, 65.3%), followed by borderline levels according to the National Institute of Health 2001 (150–199 mg/dl) in 16 individuals (22.2%). Supported by research conducted by Hussain MA on modifiable risk factors for coronary heart disease, hyperlipidemia is the second most prevalent risk factor after smoking that leads to coronary heart disease in Indonesia (Hussain et al., 2016).

In this study, it was also found that 52 individuals (72.3%) were smokers, while the remaining 27.7% did not have a history of smoking. This aligns with the results of research conducted by Song et al., where the percentage of STEMI patients with a smoking factor was 61.5% (Song et al., 2016). According to Ibanez, the nicotine content in cigarettes can disrupt the sympathetic nervous system, increasing the oxygen demand of the myocardium. Nicotine also stimulates the release of adrenaline, increases heart rate, and causes cardiac rhythm disturbances. Carbon monoxide (CO) leads to hemoglobin desaturation, reducing oxygen supply throughout the body, including the heart, and can accelerate the formation of atherosclerosis (Ibanez et al., 2018).

Another discussed risk factor in this study is the history of hypertension. According to Hanratty, hypertension is a condition when systolic blood pressure is higher than 140 mmHg and/or diastolic pressure is higher than 90 mmHg (Hanratty, 2000). In this study, it was found that 38 out of 72 individuals (52.7%) had a history of hypertension, while the remaining 47.3% did not have such a

history. This is consistent with the research by Konstantinou, where 52.1% of STEMI patients had a history of hypertension. According to Konstantinou, the relationship between a history of hypertension and the occurrence of STEMI is related to atherosclerosis and specific vasoactive effects that may lead to myocardial necrosis (Konstantinou et al., 2019).

ENOS level of STEMI Patients with Diabetes Mellitus

In this study, the median eNOS level in STEMI patients without comorbid diabetes mellitus is 38,839 U/ml, based on 36 patient samples. This level is slightly different from the research conducted by Elfi et al., regarding coronary heart disease risk factors where the mean eNOS level in this group was 71,316 U/ml. Research by Förstermann on the role of NO in oxidative stress leading to vascular diseases explains that chronic exposure to cardiovascular disease risk factors disrupts the vascular endothelium's defense mechanisms, leading to endothelial dysfunction. The long-term impact of endothelial dysfunction results in vascular rhythm disturbances due to an imbalance in the production and inactivation of endothelial nitric oxide (eNOS) (Elfi et al., 2021; Förstermann, 2010).

ENOS level of STEMI Patients without Diabetes Mellitus

The results obtained indicate a decrease in eNOS levels in the research subjects compared to the control subjects. The median eNOS level in STEMI patients with comorbid diabetes mellitus is 36,077 U/ml, based on 36 patient samples. The average random blood sugar level from 36 samples is 202.43 mg/dl, with a minimum value of 77 mg/dl and a maximum of 425 mg/dl. Research conducted by Adela et al. found that serum NO levels in hyperglycemic patients were significantly smaller at 81.7 μM compared to those without hyperglycemia at 111.8 μM (Adela et al., 2015). According to Ren et al., eNOS levels and activity decrease in diabetes and coronary heart disease conditions, often associated with insulin resistance and hyperglycemia. This condition is also related to excessive production of AGEs (advanced glycation end products), which subsequently leads to endothelial dysfunction. AGEs are produced at higher levels in diabetic patients due to insulin resistance and hyperglycemia, triggering the production of proinflammatory cytokines, oxidative stress, and a decrease in eNOS expression (Ren et al., 2017).

Difference in eNOS Levels in STEMI Patients With and Without DM

Based on the data obtained from the 72 samples, the median eNOS level (U/ml) in STEMI patients with DM is 36,077 U/ml, which is lower than in STEMI patients without DM, which is 38,839 U/ml. Looking at the maximum and minimum values in the STEMI group with DM, they are 679,754 U/ml and 1,836 U/ml, respectively, while in the STEMI group without DM, they are 584,762 U/ml and 17,042 U/ml. Although the Mann-Whitney test shows no significant difference in eNOS levels between STEMI patients with and without comorbid DM (sig > 0.05). Similar results were found in a study by Elfi EF, which compared eNOS levels in DM patients with and without acute myocardial infarction. Despite differences in eNOS levels, STEMI patients had lower levels than those without STEMI (Elfi et al., 2021). According to Wang M, hyperglycemia, hyperinsulinemia, and insulin resistance cause damage to endothelial function, including barrier dysfunction, impaired nitric oxide (NO) activity, excessive reactive oxygen species (ROS) production, oxidative stress, and dysregulated inflammation.

This also serves as a trigger for endothelial dysfunction leading to mortality in patients with coronary heart disease. The study also states that in uncontrolled DM patients, vascular damage develops in relation to hyperglycemia. Therefore, it is important to evaluate the duration of treatment, blood sugar control, and treatment history in patients with comorbid DM to determine more significant eNOS levels (Wang et al., 2022).

Study Limitations

The study has limitations, as evidenced by the non-normal distribution of data even after transformation and analysis through Kolmogorov-Smirnov. This may be due to the wide distribution of eNOS levels and risk factors in both groups. The study did not further analyze the effects of therapy, duration of treatment, and the length of the disease history in patients with STEMI and DM, preventing the researchers from eliminating the therapy's influence on eNOS between the two groups. The lack of a more in-depth comparison of IMT levels between the control and test groups might contribute to the increased eNOS levels in the samples. Further research with a larger population is still needed to generate more accurate data.

CONCLUSION

Based on the characteristics, most of the samples were male (86.1%), aged between 60 to 69 years (52.7%), with an overweight classification in terms of BMI (45.8%). The most prevalent additional risk factors included comorbid hypertension (52.7%), a history of smoking (72.3%), and dyslipidemia, with the highest LDL levels falling in the range of 100-129 mg/dl (30.4%), and the highest triglyceride levels below 150 mg/dl (65.3%).

Lower eNOS levels were found in STEMI patients with DM compared to those without DM. Although there was no significant difference in the mean eNOS levels between STEMI patients with DM and those without DM (sig >0.05).

REFERENCES

- Adela, R., Nethi, S. K., Bagul, P. K., Barui, A. K., Mattapally, S., Kuncha, M., et al. (2015). Hyperglycaemia enhances nitric oxide production in diabetes: A study from South Indian patients. *PLoS ONE*, *10*(4), e0125270.
- Alhabib, K. F., Kinsara, A. J., Alghamdi, S., Al-Murayeh, M., Hussein, G. A., AlSaif, S., et al. (2019). The first survey of the Saudi Acute Myocardial Infarction Registry Program: Main results and long-term outcomes (STARS-1 Program). *PLoS ONE*, *14*(5), e0216551.
- Cercato, C., & Fonseca, F. A. (2019). Cardiovascular risk and obesity. *Diabetology & Metabolic Syndrome*, *11*(1), 74. <https://doi.org/10.1186/s13098-019-0468-0>
- Darwin, E., Elfi, E. F., & Elvira, D. (2018). *Endotel: Fungsi dan disfungsi*. Andalas University Press.
- Elfi, E. F., Decroli, E., Nasrul, E., Yanwirasti, Y., & Darwin, E. (2021). The risk factors of coronary heart

- disease and its relationship with endothelial nitric oxide synthase. *Open Access Macedonian Journal of Medical Sciences*, 9(B), 451–456. <https://doi.org/10.3889/oamjms.2021.5994>
- Förstermann, U. (2010). Nitric oxide and oxidative stress in vascular disease. *Pflügers Archiv - European Journal of Physiology*, 459(6), 923–939. <https://doi.org/10.1007/s00424-010-0808-2>
- Gayatri, N. I., Firmansyah, S., Hidayat, S., & Rudiktyo, E. (2016). *Prediktor mortalitas dalam-rumah-sakit pasien infark miokard ST elevation (STEMI) akut di RSUD dr. Dradjat Prawiranegara Serang, Indonesia* (Vol. 43, No. 3).
- Go, A. S., Mozaffarian, D., Roger, V. L., Benjamin, E. J., Berry, J. D., Borden, W. B., et al. (2013). Heart disease and stroke statistics—2013 update: A report from the American Heart Association. *Circulation*, 127(1), e6–e245. <https://doi.org/10.1161/CIR.0b013e31828124ad>
- Hajar, R. (2017). Risk factors for coronary artery disease: Historical perspectives. *Heart Views*, 18(3), 109–114. https://doi.org/10.4103/HEARTVIEWS.HEARTVIEWS_106_17
- Hanratty, B. (2000). Sex differences in risk factors, treatment and mortality after acute myocardial infarction: An observational study. *Journal of Epidemiology and Community Health*, 54(12), 912–916. <https://doi.org/10.1136/jech.54.12.912>
- Haryuni, S. (2015). Hubungan antara berat badan dengan kejadian infark miokard akut pada pasien di ruang Intensive Coronary Care Unit RSUD Dr. Iskak Kabupaten Tulungagung. *Journal Care*, 3, 36–44.
- Hussain, M. A., Al Mamun, A., Peters, S. A., Woodward, M., & Huxley, R. R. (2016). The burden of cardiovascular disease attributable to major modifiable risk factors in Indonesia. *Journal of Epidemiology*, 26(10), 515–521. <https://doi.org/10.2188/jea.JE20150186>
- Ibanez, B., James, S., Agewall, S., Antunes, M. J., Bucciarelli-Ducci, C., Bueno, H., et al. (2018). 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *European Heart Journal*, 39(2), 119–177. <https://doi.org/10.1093/eurheartj/ehx393>
- Konstantinou, K., Tsioufis, C., Koumelli, A., Mantzouranis, M., Kasiakogias, A., Doumas, M., et al. (2019). Hypertension and patients with acute coronary syndrome: Putting blood pressure levels into perspective. *The Journal of Clinical Hypertension*, 21(8), 1135–1143. <https://doi.org/10.1111/jch.13631>
- Muttaqin, A. (2014). *PERKI dalam*. Salemba Medika.
- Paneni, F., Beckman, J. A., Creager, M. A., & Cosentino, F. (2013). Diabetes and vascular disease: Pathophysiology, clinical consequences, and medical therapy: Part I. *European Heart Journal*, 34(31), 2436–2446. <https://doi.org/10.1093/eurheartj/eh149>
- Puymirat, E., Simon, T., Cayla, G., Cottin, Y., Elbaz, M., Coste, P., et al. (2017). Acute myocardial infarction. *Circulation*, 136(20), 1908–1919. <https://doi.org/10.1161/CIRCULATIONAHA.117.030798>
- Ren, X., Ren, L., Wei, Q., Shao, H., Chen, L., & Liu, N. (2017). Advanced glycation end-products decreases

expression of endothelial nitric oxide synthase through oxidative stress in human coronary artery endothelial cells. *Cardiovascular Diabetology*, 16(1), 52. <https://doi.org/10.1186/s12933-017-0531-9>

Romesh, K. (2023). *Type 2 diabetes mellitus: Practice essentials, background, pathophysiology*. Medscape. <https://emedicine.medscape.com/article/117853-overview?reg=1>

Song, F., Yu, M., Yang, J., Xu, H., Zhao, Y., Li, W., et al. (2016). Symptom-onset-to-balloon time, ST-segment resolution and in-hospital mortality in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention in China: From China Acute Myocardial Infarction Registry. *The American Journal of Cardiology*, 118(9), 1334–1339. <https://doi.org/10.1016/j.amjcard.2016.07.055>

Susilo, C. (2015). Identifikasi faktor usia, jenis kelamin dengan luas infark miokard pada penyakit jantung koroner (PJK) di ruang ICCU RSD Dr. Soebandi Jember. *The Indonesian Journal of Health Science*, 6.

Tsao, C. W., Aday, A. W., Almarzooq, Z. I., Anderson, C. A. M., Arora, P., Avery, C. L., et al. (2023). Heart disease and stroke statistics—2023 update: A report from the American Heart Association. *Circulation*, 147(8), e93–e621. <https://doi.org/10.1161/CIR.0000000000001123>

Wang, M., Li, Y., Li, S., & Lv, J. (2022). Endothelial dysfunction and diabetic cardiomyopathy. *Frontiers in Endocrinology*, 13, Article 851941. <https://doi.org/10.3389/fendo.2022.851941>