



Association of Type IV Mosseri Lesion with Permanent Advance Atrioventricular Block in Inferior ST-Segment Elevation Myocardial Infarction Patients after Percutaneous Coronary Intervention

Hafizha Herman*, Erika Maharani, Budi Yuli Setianto

Departemen Kardiologi dan Kedokteran Vaskular, Fakultas Kedokteran, Kesehatan Masyarakat, dan Keperawatan, Universitas Gadjah Mada

ARTICLE INFO

*Corresponding author

Email:
hafizha.herman@mail.ugm.ac.id

Address:
Jl. Laut Banda Blok D1/10A Kav. AL, Duren Sawit,
Duren Sawit, Jakarta Timur, DKI Jakarta, 13440

Keywords:
STEMI; frontal QRS-T angle; myocardial perfusion defect.

Manuscript submitted: 22 Des 2022
Revised and accepted: 2 Jun 2022

ABSTRACT

Background: Advance atrioventricular (AV) block is one of the ST-segment Elevation Myocardial Infarction (STEMI) complications related to mortality, primarily in inferior STEMI. Mosseri classification divides the severity of coronary supply to the AV node. Mosseri type IV is the most common finding in general populations with chronic conduction disturbance in STEMI populations.

Aim: To determine the type IV Mosseri lesion association with permanent advance AV block in inferior STEMI patients after percutaneous coronary intervention (PCI).

Research Methods: Analytical observations were conducted with a cross-sectional design, using secondary data from medical records in RSUP Dr. Sardjito within August 2018-August 2021 on inferior STEMI with advance AV block population underwent PCI, then we obtained baseline characteristic, electrocardiogram, and coronary angiography.

Results: Within observation period 192 patients had advance AV block in inferior STEMI, 138 patients included as research subject. Mean age of permanent advance AV block group was $62,77 \pm 10,78$ years old ($p= 0,739$), and female gender 30,8% ($p= 0,303$). There was statistically significant difference such as lower systolic 91 (70-147) mmHg & diastolic 54 (25-104) mmHg blood pressure ($p= 0,019$ & $0,000$), and bigger proportion in thrombolysis in myocardial infarction (TIMI) flow <3 61,5% ($p= 0,024$) in group with permanent advance AV block. Type IV Mosseri lesion had bigger proportion in permanent advance AV block (61,5%) (OR 1,79; IK 95% 0,56 – 5,77), but not statistically significant ($p= 0,325$). Multivariate analysis showed low diastolic blood pressure ($p= 0,008$, OR 0,897; 0,828-0,971) and TIMI flow <3 ($p= 0,042$, OR 3,935; 1,048-14,770) were independent predictor that associated with permanent advance AV block.

Conclusions: Permanent advance AV block with Type IV Mosseri lesion had a more considerable proportion in patients with inferior STEMI after PCI, but the association is not statistically significant.

INTISARI

Latar Belakang: Blok atrioventrikular (AV) lanjut merupakan salah satu komplikasi pada infark miokard akut elevasi segmen-ST (IMA-EST) yang berkaitan dengan mortalitas, khususnya IMA-EST inferior. Klasifikasi Mosseri membagi keparahan lesi koroner yang menyuplai arteri nodus AV. Lesi Mosseri tipe IV paling sering ditemukan pada populasi pasien yang mengalami gangguan konduksi kronis dan populasi IMA-EST secara umum.

Tujuan: Mengetahui hubungan lesi Mosseri tipe IV dengan blok AV lanjut menetap pada pasien IMA-EST inferior pasca intervensi koroner perkutan (IKP).

Metode Penelitian: Observasi analisis ini dilakukan secara potong lintang menggunakan data sekunder catatan medis di RSUP Dr. Sardjito sejak periode Agustus 2018-Agustus 2021, pada populasi IMA-EST inferior yang mengalami blok AV lanjut dan dilakukan IKP, kemudian dilakukan penelusuran terhadap karakteristik dasar, elektrokardiogram, dan hasil angiografi koroner.

Hasil: Selama periode observasi sebanyak 192 pasien mengalami blok AV lanjut pada IMA-EST inferior, 138 pasien memenuhi kriteria inklusi dan eksklusi. Rerata usia pada kelompok blok AV lanjut menetap $62,77 \pm 10,78$ tahun ($p = 0,739$), dan jenis kelamin perempuan 30,8% ($p = 0,303$). Terdapat perbedaan yang signifikan pada kelompok dengan blok AV lanjut menetap didapatkan tekanan darah yang lebih rendah dengan median tekanan darah (TD) sistolik 91 (70-147) mmHg dan diastolik 54 (25-104) mmHg ($p = 0,019$ & $0,000$), dan proporsi aliran thrombolysis in myocardial infarction (TIMI) <3 sebesar 61,5% ($p = 0,024$). Lesi Mosseri tipe IV pada blok AV lanjut menetap memiliki proporsi yang lebih besar (61,5%) (OR 1,79; IK 95% 0,56 - 5,77), namun hubungan tersebut tidak signifikan secara statistik ($p = 0,325$). Analisis multivariat menunjukkan TD diastolik rendah ($p = 0,008$, OR 0,897; 0,828-0,971) dan aliran TIMI <3 ($p = 0,042$, OR 3,935; 1,048-14,770) merupakan faktor prediktor independen yang berhubungan dengan kejadian blok AV lanjut menetap.

Simpulan: Kejadian blok AV lanjut menetap dengan lesi Mosseri tipe IV memiliki proporsi yang lebih besar pada IMA-EST inferior pasca IKP, namun hubungan tersebut tidak signifikan secara statistik.

Introduction

Acute myocardial infarction (AMI) is one of the most common causes of morbidity and mortality across the globe. Around 15% of mortality each year is caused by AMI.¹ In developing countries, the prevalence of AMI has varied since the revascularization era.² Data from healthcare facilities in Indonesian STEMI (iSTEMI) from 2014 to 2017 showed an increment of number ST-Elevation Myocardial Infarction (STEMI) every year with a prevalence of 48.9% from all acute coronary syndrome (ACS) in 2017.³

An advanced atrioventricular (AV) block is one of the complications of AMI. High degree AV block (HDAVB) is defined as conduction disturbance in the AV node when there were >2 consecutive P waves within physiologic heart rate that do not conduct to ventricle with the evidence of AV conduction. Meanwhile, total AV block is no evidence of AV conduction.⁴ Traditionally, AV block 2:1 and HDAVB are grouped as second-degree AV blocks, but the location of conduction disturbance cannot solely be seen from a surface electrocardiogram (ECG)⁵ Several studies included second-degree Mobitz II to total AV block into the spectrum of advanced AV block.⁶⁻⁸

Incidence of AV block range between 2-14% and reach up to 28% in inferior STEMI. After the reperfusion era with primary percutaneous coronary intervention (PCI), AV block incidence in AMI decreased to 1,9%, but 79% of AV blocks still happen on inferior STEMI⁹ Inferior STEMI 2-4 times more vulnerable to advanced AV block than anterior infarct location¹⁰ the latest study showed

inferior-lateral/lateral STEMI was an independent risk factor for advanced AV block and the need for a temporary pacemaker. It is related to coronary dominance of blood vessel that supplies the AV nodal artery, which derives from the right coronary artery (RCA) or left circumflex (LCx) artery.¹¹

Thirty-day mortality of STEMI with total AV block was 33%, and long-term mortality is still significant even though the incidence of advanced AV block decreases after the primary PCI era.^{6,10} Another study showed 30 days mortality of inferior STEMI with AV block higher than the population without AV block with an odds ratio (OR) 2,2.¹² Recent guidelines related to PCI on STEMI late presentation more than 12 hours onset only recommended to a patient with unstable hemodynamic and electrical instability or ongoing ischemia. Total AV block did not explicitly mention as one of electrical instability.¹³ Recommendation of latest guideline PCI only conducted in the infarct-related artery.¹⁴

After revascularization, 70% of AV block cases recovered <12 hours. Meanwhile, 30% of patients recovered within various times from 12 hours to 12 days.¹⁵ After PCI, delayed AV block recovery was estimated to be caused by a microvascular obstruction in the artery that supplies the AV node.¹⁶ Most STEMI patients with AV block complications that had disruption of coronary flow to the bundle branch have a higher risk of permanent AV block in the future and are considered candidates for permanent pacemaker (PPM) implantation.^{17,18}

Mosseri et al. (1997) made pathological classifications from the coronary artery, which supplies the AV node conduction system to four types in patients with chronic AV block with PPM. In Mosseri type I, there is no significant lesion in the septal branch or AV nodal artery; in type II, there is a significant lesion (>50% stenosis) interrupting the septal branch from the left descending artery (LAD); in type III, there is a significant lesion interrupting the flow to AV node artery; and in type IV is the combination of type II and III lesion.¹⁹ Mosseri classification has the highest proportion, around 40% among AMI patients complicated with advanced AV block. Generally, reversibility of AV block in AMI patients is higher (86,7%) than in non-AMI presentation populations.²⁰ Patients with stenosis in RCA and LAD that supplies septal branch (type IV Mosseri lesion) are commonly found in patients with conduction disturbance using PPM.^{19,21}

Advanced AV block is caused by decreased coronary blood flow that supplies the AV node. Around 9% of patients with high degree AV block need implantation of PPM because permanent damage to the conduction system causes an inadequate flow of coronary artery and thrombolysis in myocardial infarction (TIMI) before or after revascularization.²² Besides the coronary supplies from the AV nodal artery, most supplies are derived from the collateral or contralateral system of the septal perforator branch from proximal LAD.²³ After revascularization, the patency of collateral arteries is influenced by vasoconstrictor drugs and nitrate release.²⁴

In several cases, conduction disturbance can be permanent, possibly caused by disruption of flow on proximal LAD that supplies ventricular septal. The latest recommendation for STEMI patient interventions needs to be conducted only in the infarct-related artery. This study aims to find Mosseri type IV lesion association with permanent advanced AV block in the inferior STEMI population. The result from this study is expected to be a consideration for clinical judgment in inferior STEMI complicated with AV block underwent revascularization and had type IV Mosseri lesion. To this extent, there is no further study related to coronary pathology classification on inferior STEMI.

Methods

This study is an analytical observational with a cross-sectional method conducted in RSUP Dr. Sardjito after receiving permission from FKKMK UGM medical ethical committee. Study subject data was taken from Sardjito Cardiovascular intENSive CarE (SCIENCE) registry and RSUP Dr. Sardjito medical records that have fulfilled inclusion and exclusion criteria since 2018. The study subject is inferior STEMI complicated with block AV and had successful PCI, defined as recovery flow in infarct-related artery, then hospitalized in ICCU RSUP Dr. Sardjito Yogyakarta and registered in SCIENCE registry since August 2021 taken retrospectively to August 2018.

Inclusion criteria in this study consist of 1) patient's age > 18 years old, 2) diagnosed with inferior/inferior with

the right, or posterior, or lateral infarct, 3) advanced AV block before PCI, 4) underwent primary or rescued PCI and had recovery of blood flow in an infarct-related artery (RCA/LCx depends on coronary dominance), 5) hospitalized in ICCU RSUP Dr. Sardjito, and 6) has been evaluated for ECG after PCI. Exclusion criteria in this study are: 1) patients with a history of coronary artery by-pass surgery, 2) patients with congenital heart disease, 3) patients with PPM implantation before, 4) patients with advanced AV block which has recovered to second degree AV block Mobitz I, first degree AV block, or normal sinus rhythm before PCI, 5) patient had intervention in LAD and, 6) study subject without complete medical record data.

Research Protocol

The study subjects were patients diagnosed with inferior STEMI who experienced an advanced AV block before PCI. We collected 12-leads before and after PCI from medical records. After PCI, the reversibility of AV block was decided from the ECG evaluation showing transformation to sinus rhythm or at least to second-degree Mobitz I or first-degree AV block. Permanent advanced AV block is defined if there's no recovery of advanced AV block from the ECG evaluation before implantation of PPM or death. Duration of AV block is defined as the time of patient underwent revascularization at PCI until recovery of AV conduction delay, death, or implantation of PPM.

Data related to coronary angiography taken from the patient's procedure log record include coronary profile, wire crossing time, and TIMI flow after PCI. Mosseri classification will be classified from the coronary angiography profile by the researcher from the operational definition of Mosseri classification. Mosseri classification will be grouped into type IV and non-type IV; in this study, non-type IV is type III. Data of TIMI flow taken from each interventionist conducting the procedure has been calculated for inter-observer study with mean kappa 0,87.²⁵

The analyzed parameter is patients' clinical characteristics (age, sex, diabetes mellitus, hypertension, smoking history, history of ACS, clinical conditions), systolic and diastolic blood pressure, coronary angiography profile such as Mosseri classifications, TIMI flow after PCI, and total ischemic time. If there were incomplete data from medical records, the subject would be excluded from the study.

Statistical Analysis

Data were analyzed with Statistical Package for the Social Science (SPSS) International Business Machine (IBM) software version 23. Univariate analysis numeric variable using Kolmogorov-Smirnov/ Shapiro-Wilk test where $p > 0,05$ showed normal data distribution. Numeric variables with normal distribution will be shown as mean + standard deviation (SD). Meanwhile, data without normal distribution will be shown as median (minimum-maximum).²⁶

Numeric variables with normal distribution will be bivariate analyzed with an independent T-test. Meanwhile, without normal distribution will be tested with a non-parametric test Mann-Whitney. The categorical variable will be analyzed with the Chi-Square test; if there were less than five expected counts from one cell Fisher test will be used. All independent variables will be analyzed with multivariate analysis to determine confounding factors from Mosseri lesion with the Chunk test.^{26,27}

Result

Baseline Characteristics of Study Subjects

Table 1.
Patients' baseline characteristic

	Advanced AV block		P-value
	Permanent (n=13)	Transient (n=125)	
Age, years	62,77 ± 10,78	61,69 ± 11,17	0,739
Sex, %			
Female	4 (30,8)	26 (20,8)	0,303**
Risk Factors, %			
Hypertension, %	8 (61,5)	77 (61,6)	0,609**
Diabetes mellitus, %	5 (38,5)	57 (45,6)	0,622
Smoker, %	9 (69,2)	83 (66,4)	0,552
History of ACS, %	0 (0)	4 (3,2)	0,670**
BP after PCI			
Systolic, mmHg	91 (70-147)	111(70-180)	0,019*
Diastolic, mmHg	54 (25-104)	65 (38-99)	0,000*
TIMI after PCI<3**, %	8 (61,5)	37 (29,6)	0,024*
Total ischemic time, minute	1277 (302-7455)	853(165-7444)	0,129

*Baseline characteristic study subject had statistically significant difference between 2 groups with p-value <0,05
**Fisher test is used because there were cells with an expected count of less than 5

Information: ACS, acute coronary syndrome; AV, Atrioventricular; BP, blood pressure; PCI, percutaneous coronary interventions; TIMI, Thrombolysis in Myocardial Infarction

From August 2018 to August 2021, 192 patients (27,04%) had advanced AV block from 710 patients diagnosed with inferior STEMI in RSUP Dr. Sardjito. A total of 138 patients fulfilled inclusion and exclusion criteria and were included as a study subject, 13 patients (9,4%) had permanent, and 125 patients (90,6%) had transient advanced AV block (Table 1).

Hypothesis Analysis of Type IV Mosseri Lesion with Permanent Advanced AV Block

Table 2.
Hypothesis analysis of type IV Mosseri lesion with permanent Advanced AV block

	Advanced AV Block		p-value	OR	CI 95%	
	Permanent (n=13)	Transient (n=125)			min	max
Type of Mosseri, %			0,325	1,79	0,56	5,77
IV	8 (61,5)	59 (47,2)				
Non-type IV	5 (38,5)	66 (52,8)				

Information: AV, atrioventricular; CI, confidence interval; OR, Odds Ratio

Analysis of association of advanced AV block with type IV of Mosseri lesion shows the difference in proportion between permanent (61,5%) and transient (38,5%) advanced AV block. Value of OR 1,79 (CI 0,56 - 5,77), but the difference is not statistically significant (p= 0,325) (Table 2).

Multivariate Analysis Mosseri Lesion with Confounding Factors

This multivariate analysis tests several variables such as age, sex, risk factors of hypertension, diabetes mellitus, smoker, history of ACS, systolic and diastolic blood pressure, TIMI flow after PCI, and total ischemic time (Table 3). That analysis showed that type IV Mosseri lesion is not statistically significant in association with permanent advanced AV block (p= 0,788). Low diastolic blood pressure and TIMI flow <3 are independent risk factors to the permanent advanced AV block. At the final multivariate analysis, diastolic blood pressure and TIMI flow <3 after PCI had statistically significant value in a row p= 0,008 (OR 0,897, CI 95% 0,828-0,971) and p= 0,042 (OR 3,935, CI 95% 1,048-14,770).

Table 3.
Multivariate analysis of type Mosseri lesion with confounding factors of reversibility Advanced AV block

	p-value	OR (CI 95%)
Type IV Mosseri Lesion	0,788	0,829 (0,211-3,259)
Systolic BP	0,445	1,017 (0,974-1,063)
Diastolic BP	0,008*	0,897 (0,828-0,971)
TIMI flow <3 after PCI	0,042*	3,935 (1,048-14,770)

Information: BP, blood pressure; CI, confidence interval; OR, Odds Ratio; TIMI, thrombolysis in myocardial infarction

Sub-analysis of Transient Advanced AV Block Group with Duration of AV Block after PCI

Sub-analysis was conducted in transient advanced AV block compared to type IV and non-type IV Mosseri lesion to find out Advanced AV block recovery duration after PCI (Figure 1). This study showed that 74,6% of patients with type IV and 69,7% of patients with non-type IV Mosseri lesions experience reversibility of advanced AV block less than the first 24 hours after PCI. Bivariate analysis between Mosseri lesion and each group of transient advanced AV block (>24-48 hours, >48-72 hours, and >72 hours group) compared to advanced AV block <24 hours

group showed a difference which is not statistically significant ($p = 0,753$; $0,535$; and $0,404$).

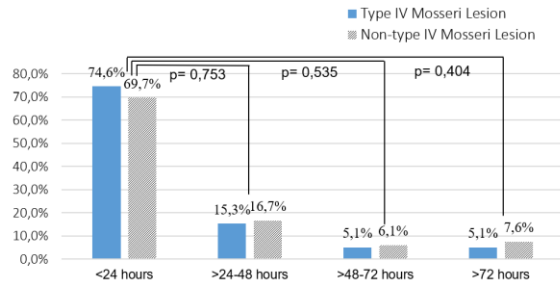


Figure 1. Bar chart of transient Advanced AV block after PCI which grouped based on type IV and non-type IV Mosseri lesion

Discussion

There was no statistically significant difference in age between the group of patients with permanent and transient advanced AV block in this study. The degenerative process of the conduction system starts at the age of 40-75 years old.²⁸ That result aligns with the previous case report in geriatric patients with ACS who underwent PCI; advanced AV block was transient. This result showed that the chronic degenerative process in the conduction system had no significant difference in the occurrence of permanent advanced AV block in acute STEMI conditions after PCI.²⁹

Theoretically, the existing comorbid conditions such as hypertension, diabetes mellitus, older age, and smoking may be caused by chronic damage to the conduction system. In STEMI with advanced AV block, complications will add severity to the occurrence of permanent advanced AV block.³⁰⁻³³ The result from the bivariate analysis did not show those theories. Chronic risk factors did not add severity to conduction disturbance in acute conditions.

In this study, no patient had a history of ACS that had permanent advanced AV block, and there was no difference between the group with transient and permanent AV block. Conclusion This result supports the previous data that a patient with a history of ACS is less likely to have advanced AV block.⁷ The probability that may explain this phenomenon was the previous ischemic condition already triggered the development of collateral flow. When acute ischemic happens after the existing collaterals will be increased and prevent the occurrence of permanent advanced AV block.^{34,35}

On the ECG evaluation, most study subjects had recovery of AV block into sinus rhythm, first-degree AV block, or second-degree Mobitz I after the patient had PCI. There was 61,5% patient with type IV, and 38,5% with non-type IV lesion had permanent advanced AV block with OR 1,79 (CI 95% 0,55-5,77), but its statistically not significant. This result differed from the previous study, where a higher proportion of permanent advanced AV blocks had type IV Mosseri lesions (58,3%) even though the result was not statistically significant. That study may have

different results because that population included anterior STEMI with advanced AV block.²⁰

The possibilities were that anterior infarction frequently involved conduction disturbance in His bundle because of flow obstruction at the proximal septal perforator branch. The damage that happened to His bundle was more likely to be permanent and needed PPM implantation.^{4,22} This result shows that in the inferior STEMI population, there was a better possibility of advanced AV block reversibility compared to anterior STEMI after PCI. Unlike patients with coronary artery disease, a chronic condition with type IV Mosseri lesion had a higher possibility of conduction disturbance that needed the implantation of PPM than the AMI population after revascularization.^{18,19,36} Patients with permanent advanced AV block prone had lower systolic and diastolic blood pressure than transient advanced AV block. This result is in line with a previous study that showed lower systolic and diastolic blood pressure in a group with permanent advanced AV block (median 94 mmHg and 64 mmHg in a row) and an independent predictor of advanced AV block in inferior STEMI.¹¹

There was a statistical difference in proportion with permanent and transient AV blocks in TIMI flow <3 after PCI. A decrease in TIMI flow indicates non-fully reversible reperfusion. A similar result showed from a study in Hong Kong on inferior STEMI patients who did not undergo PCI; the reversibility of advanced AV block is longer up to the implantation of PPM.¹⁵ Decrease flow to RCA od septal branch caused by blood pressure or TIMI flow factors supports the ischemia theory related to the variance of conduction disturbance.²² In multivariate analysis, diastolic blood pressure and TIMI flow <3 showed that the higher value of diastolic blood pressure will be protective meanwhile TIMI flow <3 after PCI will be the risk factors for permanent advanced AV block. Lower blood pressure may be related to larger infarct size and right ventricular infarction.¹⁵ Diastolic blood pressure is considered a triggering factor for developing collateral flow to the AV node to fasten the recovery of conduction disturbance.^{22,35,37} Lower blood pressure and recurrent hypotension were related to permanent Advanced AV block.²²

Recovery from advanced AV block with good TIMI flow and high diastolic blood pressure cannot be separated from the theory of good collaterals to AV node and branches of conduction systems. The growth and patency of collaterals are mentioned depending on balloons inflation at PCI and the release of nitrates. After revascularization on the infarct-related artery or additional drug injection with vasoconstrictors properties, the collateral flow will regress, even though the regression functionally will not happen in the first 24 hours after successful PCI.^{24,38,39}

In this study subjects, there was no significant difference between the total ischemic time of patients with permanent and transient Advanced AV block, with median total ischemic time in consecutively 853 and 1277 minutes, and 90,6% of subject populations in this

study recovered from Advanced AV block. The result supports a previous study in China with a similar baseline characteristic of late presentation inferior STEMI (>12 hours); there was recovery from total AV block after PCI.¹³ Theoretically, permanent damage to the myocardium can be caused by microvascular obstruction resulting from prolonged ischemic time >120 minutes, even though the epicardial coronary artery is already patented.⁴⁰

This study's result showed the difference from the common theory with long ischemic time after PCI the reversibility of Advanced AV block may still happen. Microvascular obstruction is one of the reperfusion injuries suspected as one of the mechanisms of sustained ischemia on the conduction system after successful PCI.¹⁶ Fast recovery after reperfusion may be influenced by protective factors on the AV node that prevents infarction or permanent damage from the AV node. Those characteristics are not owned by the myocardium, such as the ability to absorb oxygen and nutrition from the sinusoid vein, collateral flow, and high content of intracellular glycogen.^{6,16,41} Development of good collateral flow in patients with significant chronic lesions on more than one coronary artery, as seen on type IV Mosseri lesion, might be a better trigger of acute recanalization of the collateral vessel than non-type IV lesion.³⁵

Conclusions

Permanent advanced AV block with Type IV Mosseri lesion had a more considerable proportion in patients with inferior STEMI after PCI, but the association is not statistically significant.

Acknowledgements

The authors would like to thank all staff and residents of the Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada, for the discussion and suggestions regarding this research.

Funding Sources

This research was funded by RSUP Dr. Sardjito research grant 2021.

Disclosures and Ethics

The authors have no conflicts of interest to declare. This study has been approved by the medical ethics committee of the Faculty of Medicine, Nursing, and Public Health, Gadjah Mada University, number KE/FK/1065/EC/2021

References

- Jayaraj JC, Davatyan K, Subramanian SS, Priya J. Epidemiology of Myocardial Infarction. In: Myocardial Infarction. IntechOpen; 2019. p. 2083–7.
- Ralapanawa U, Sivakanesan R. Epidemiology and the Magnitude of Coronary Artery Disease and Acute Coronary Syndrome: A Narrative Review. 2020;0(CVD).
- Sunjaya AP, Sunjaya AF, Priyana A. Insights and challenges of Indonesia's acute coronary syndrome telecardiology network: Three year experience from a single center and in west Jakarta, Indonesia. IOP Conf Ser Mater Sci Eng. 2019;508(1).
- Kusumoto FM, Committee W, Schoenfeld MH, Committee VW, Barrett C, Member WC, et al. 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay. Hear Rhythm. 2018;(October).
- Josephson ME. Josephson's clinical cardiac electrophysiology: Techniques and interpretations. 5th ed. Wolters Kluwer. Philadelphia: Wolters Kluwer; 2016. 109–112 p.
- Shacham Y, Leshem-Rubinow E, Steinvil A, Keren G, Roth A, Arbel Y. High degree atrioventricular block complicating acute myocardial infarction treated with primary percutaneous coronary intervention: Incidence, predictors and outcomes. Isr Med Assoc J. 2015;17(5):298–301.
- Misumida N, Ogunbayo GO, Catanzaro J, Etaaee F, Kim SM, Abdel-Latif A, et al. Contemporary practice pattern of permanent pacing for conduction disorders in inferior ST-elevation myocardial infarction. Clin Cardiol. 2019;42(8):728–34.
- Singh SM, FitzGerald G, Yan AT, Brieger D, Fox KAA, López-Sendón J, et al. High-grade atrioventricular block in acute coronary syndromes: Insights from the Global Registry of Acute Coronary Events. Eur Heart J. 2015;36(16):976–83.
- Rosa SA, Timóteo AT, Ferreira L, Carvalho R, Oliveira M, Cunha P, et al. Complete atrioventricular block in acute coronary syndrome: prevalence, characterisation and implication on outcome. Eur Hear journal Acute Cardiovasc care. 2018;7(3):218–23.
- Gang UJO, Hvelplund A, Pedersen S, Iversen A, Jons C, Abildstrom SZ, et al. High-degree atrioventricular block complicating ST-segment elevation myocardial infarction in the era of primary percutaneous coronary intervention. Europace. 2012;14(11):1639–45.
- Chera HH, Mitre CA, Nealis J, Mironov A, Budzikowski AS. Frequency of complete atrioventricular block complicating ST-Elevation myocardial infarction in patients undergoing primary percutaneous coronary intervention. Cardiol. 2018;140(3):146–51.
- Meine TJ, Al-Khatib SM, Alexander JH, Granger CB, White HD, Kilaru R, et al. Incidence, predictors, and outcomes of high-degree atrioventricular block complicating acute myocardial infarction treated with thrombolytic therapy. Am Heart J. 2005;149(4):670–4.
- Fan X, Maharjan P, Liu P, Bai L. Effect of primary PCI on the recovery of atrioventricular block in inferior STEMI patients with late presentation (>12 hours): Insights from a single-center 10-year experience. J Investig Med. 2020;68(5):1011–4.

14. PERKI. Pedoman Tata Laksana Sindrom Koroner Akut 2018. Perhimpunan Dokter Spesialis Kardiovaskular Indonesia. 2018. p. 76.
15. Jim MH, Chan AOO, Tse HF, Barold SS, Lau CP. Clinical and angiographic findings of complete atrioventricular block in acute inferior myocardial infarction. *Ann Acad Med Singapore*. 2010;39(3):185-90.
16. John TJ, Kyriakakis C, Zachariah D, Doubell A. Inferior ST-elevation myocardial infarction managed with a pharmacoinvasive strategy and conservative management of delayed atrioventricular block: Classical case report. *Eur Hear J - Case Reports*. 2020;4(6):1-7.
17. Sutton R, Davies M. The conduction system in acute myocardial infarction complicated by heart block. *Circulation*. 1968;38(5):987-92.
18. Tandoğan I, Yetkin E, Güray Y, Aksoy Y, Sezgin AT, Özdemir R, et al. Distribution of coronary artery lesions in patients with permanent pacemakers. *Anadolu Kardiyol Derg*. 2002;2(4):279-83.
19. Mosseri M, Izak T, Rosenheck S, Lotan C, Rozenman Y, Zolti E, et al. Coronary angiographic characteristics of patients with permanent artificial pacemakers. *Circulation*. 1997;96(3):809-15.
20. Hwang IC, Seo WW, Oh IY, Choi EK, Oh S. Reversibility of atrioventricular block according to coronary artery disease: Results of a retrospective study. *Korean Circ J*. 2012;42(12):816-22.
21. Wei S, Zhong L, Chen S, Li X. The status of coronary artery lesions in patients with conduction disturbance. *J Cardiovasc Med*. 2011;12(10):709-13.
22. Cardoso R, Alfonso CE, Coffey JO. Reversibility of High-Grade Atrioventricular Block with Revascularization in Coronary Artery Disease without Infarction: A Literature Review. *Case Reports Cardiol*. 2016;2016:1-6.
23. Simons GR, Sgarbossa E, Wagner G, Califf RM, Topol EJ, Natale A. Atrioventricular and intraventricular conduction disorders in acute myocardial infarction: A reappraisal in the thrombolytic era. *PACE - Pacing Clin Electrophysiol*. 1998;21(12):2651-63.
24. Fujita M, Sasayama S. Reappraisal of functional importance of coronary collateral circulation. *Cardiology*. 2011;117(4):246-52.
25. uryantoro M, Taufiq N, Bagaswoto HP. Perbedaan Derajat Aliran Koroner Thrombolysis in Myocardial Infarction 3 antara Pasien Infark Miokard Akut dengan Elevasi Segmen ST DE yang Dilakukan Strategi Farmako-invasif Alteplase Dibandingkan dengan Strategi Intervensi Koroner Perkutan Primer di RSU. Universitas Gadjah Mada; 2021.
26. Siswosudarmo R. Pendekatan Praktis Penelitian Epidemiologi Klinis Dan Aplikasi SPSS untuk Analisis Statistika. *J Chem Inf Model*. 2015;53(9):1689-99.
27. Kleinbaum DG, Klein M. Modeling Strategy for Assessing Interaction and Confounding. In: *Modeling Strategy for Assessing Interaction and Confounding*. 3rd ed. New York, NY: Springer New York; 2010. p. 203-39.
28. Waller BF, Orr CM, Slack JD, Pinkerton CA, Van Tassel J, Peters T. Anatomy, histology, and pathology of coronary arteries: A review relevant to new interventional and imaging techniques—Part II. *Clin Cardiol*. 1992;15(7):535-40.
29. Sueyoshi H, Akita Y, Oishi Y, Mukai Y, Hagino T, Yutaka K, et al. Consecutive electrocardiographic changes during percutaneous coronary intervention for acute coronary syndrome with high-grade atrioventricular block: a case report. 2020;6:1-5.
30. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*. 2011 Jan 1;34(Supplement_1):S62-9.
31. Kerola T, Eranti A, Aro AL, Haukilahti MA, Holkeri A, Junttila MJ, et al. Risk Factors Associated With Atrioventricular Block. *JAMA Netw open*. 2019;2(5):e194176.
32. Movahed MR, Hashemzadeh M, Jamal MM. Increased prevalence of third-degree atrioventricular block in patients with type II diabetes mellitus. *Chest*. 2005;128(4):2611-4.
33. Gosse P, Coulon P, Papaioannou G, Litalien J, Lemetayer P. Atrioventricular conduction in the hypertensive patient: Influence of aging, pulse pressure, and arterial stiffness. *Rejuvenation Res*. 2011;14(4):405-10.
34. Bassan R, Maia IG, Bozza A, Amino JGC, Santos M. Atrioventricular block in acute inferior wall myocardial infarction: Harbinger of associated obstruction of the left anterior descending coronary artery. *J Am Coll Cardiol*. 1986;8(4):773-8.
35. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. *J Am Coll Cardiol*. 1985;5(3):587-92.
36. Yesil M, Arikan E, Postaci N, Bayata S, Yilmaz R. Locations of coronary artery lesions in patients with severe conduction disturbance. *Int Heart J*. 2008;49(5):525-31.
37. Shu W, Jing J, Fu LC, Min JT, Bo YX, Ying Z, et al. The relationship between diastolic pressure and coronary collateral circulation in patients with stable angina pectoris and chronic total occlusion. *Am J Hypertens*. 2013;26(5):630-5.
38. Yetgin T, Magro M, Manintveld OC, Nauta ST, Cheng JM, Den Uil CA, et al. Impact of multiple balloon inflations during primary percutaneous coronary intervention on infarct size and long-term clinical outcomes in ST-segment elevation myocardial infarction: Real-world postconditioning. *Basic Res Cardiol*. 2014;109(2).
39. Perera D, Kanaganayagam GS, Saha M, Rashid R, Marber MS, Redwood SR. Coronary collaterals remain recruitable after percutaneous intervention. *Circulation*. 2007;115(15):2015-21.
40. Henderson M, Carberry J, Berry C. Targeting an Ischemic Time <120 Minutes in ST-Segment-

- Elevation Myocardial Infarction. J Am Heart Assoc. 2019 Jun 18;8(12).
41. Jedeikin LA. Regional Distribution of Glycogen and Phosphorylase in the Ventricles. Circ Res. 1964;14(March):202-11.