INTRODUCTION

Myocardial infarction (MI) is the leading cause of morbidity and mortality worldwide.1,2 MI is the most common form of coronary heart disease (CHD). MI occurs when a coronary artery is completely or partially stenotic and causes a significant reduction in coronary blood flow. Furthermore, the myocardium supplied by the artery can undergo an infarct. It can be recognized by clinical features and characteristic changes on Electrocardiography (ECG). ECG has a fundamental role in diagnosing ST-Elevation Myocardial Infarction (STEMI), both in determining the location of myocardial infarction and predicting the location of the culprit lesion in the coronary arteries.3,4 STEMI occurs when transmural myocardial ischemia causes necrosis or injury to the myocardium, which also causes deviation of the ST segment vector on the ECG. It is an indicator of the location of the blockage in the coronary arteries.5

Inferior STEMI caused by complete occlusion of the right coronary artery/RCA (80% of cases) and left circumflex artery/LCx (20% of cases) can be predicted from the ECG during the acute phase. Inferior STEMI usually has a better prognosis than anterior STEMI. Inferior STEMI accounts for approximately 40% to 50% of all STEMI events.6 Inferior STEMI has a low mortality rate of less than 10%, but complicating factors such as cardiogenic shock, heart block bradycardia, right ventricular infarction, and hypotension can increase the risk of mortality.7

The previous studies used ECG information to predict the culprit lesion in STEMI patients before future angiography, thereby reducing the risk of misclassification in determining infarct...
related artery (IRA). In this era, primary percutaneous coronary intervention (PCI) procedures are the main revascularization method in STEMI. There is an opportunity to review the ECG algorithm because it is important for early predicting the culprit lesion because the prognosis and management strategy can vary between occlusion in RCA and LCx and Under certain conditions, angiography of the culprit lesion is more difficult to determine and an ECG can be helpful in those conditions. Based on those mentioned above, this case report aims to overview ECG Algorithm to diagnose Inferior STEMI and predict the artery’s culprit.

CASE DESCRIPTION

A 48-year-old man came to the Emergency Unit (ER) of Sanjiwani Regional Hospital Gianyar with a complaint of typical chest pain for 2 hours before coming to the hospital. Chest pain was said to be dull, like pressure from a heavy object and radiates to the back, accompanied by nausea, vomiting, and cold sweats. Patients also complain of shortness of breath at rest or activity. Swollen legs and palpitations were denied. The patient had a history of being a heavy smoker from adolescence until now and had dyslipidemia. History of hypertension, diabetes mellitus, stroke, and kidney disease was denied.

On arrival, the patient was in a very serious condition. GCS E4V5M6 (agitation) blood pressure 110/70mmhg, pulse 129 beats per minute, respirations 22 beats per minute with 95% oxygen saturation, the nasal cannula 3 liters per minute. The general status examination was within normal limits. Electrocardiographic (ECG) examination showed atrial fibrillation rhythm with ST elevation in II, III, aVF, V5, and V6, and ST depression in I, aVL, and V1 to V4 and ST elevation in the posterior leads V7-V9. Thus, from the ECG, it is concluded that inferoposterolateral STEMI with Rhythm Atrial Fibrillation (AF). Cardiac markers showed an increase in troponin I and creatine kinase-MB, namely 81.8ng/L (reference value <19ng/L) and 36.0 U/L (reference value <24 U/L), and other laboratory tests are within normal limits. Shortly after the ECG examination, the patient became unconscious, with blood pressure dropping to 60/palpation and bradycardia at 48 beats/minute. The patient’s extremities were cold.

The patient’s initial diagnosis was Killip IV inferoposterolateral STEMI with 2 hours onset and atrial fibrillation, planned to undergo fibrinolytic because there were no PCI facilities.

The patient received IVFD therapy with NaCl 0.9%, oxygen 3 liters per minute nasal cannula, Aspilet 160mg continued 1x80 mg, Clopidogrel 300mg followed by a maintenance dose of 1x75mg, Atorvastatin 1x40mg, Diazepam 3x5mg, and dobutamine starting at 5 mcg/kg/minute and increased quickly to reach the target. After that, blood pressure became 100/60 mmHg. After blood pressure stabilized, the patient was immediately given fibrinolytic therapy with Streptokinase 1,500,000 U for 30 minutes. Yet, not long after Streptokinase was administered, the patient became unconscious, with the ECG monitor showing signs of Ventricular fibrillation (VF). Thus, immediately cardiopulmonary resuscitation was performed and the patient received 360 Joules of defibrillation 2 times, then the ECG returned to sinus rhythm (Figure 1). The patient’s blood pressure dropped to 70/50 mmHg. The patient was administered additional norepinephrine starting at 0.1 mcg/kg/min. Blood pressure managed to rise to 100/60 mmHg; after hemodynamically stable the patient was immediately referred to the PCI Center for rescue PCI with the results of Coronary Angiography: LM: normal, LAD: Normal, LCx: Dominant, Subtotal Stenosis proximal to distal, thrombus

![Figure 1](image1.png) The electrocardiogram on admission shows ST-segment elevation of 1.0, 2.0, and 1.0 mm, in inferior leads II, III, aVF, 1.0 mm, and 2.0 mm in lateral leads V5, and V6, respectively. ST-segment depression is observed in leads I, aVL, and V1 to V4. Modified posterior leads V7 to V9 show an ST elevation of 3.0 mm.

![Figure 2](image2.png) (A) Angiography on LCx shows Dominant, Subtotal Stenosis proximal to distal, thrombus (+) 80% OMI stenosis, (B) RCA angiography shows small, non-dominant.
However, the specificity of ECG in ACS is influenced by several factors, such as variations in coronary anatomy, previous history of myocardial infarction or history of coronary heart disease (CHD), and the presence of collateral circulation. Determining the location of the culprit lesion is important to assist in selecting management and anticipating complications. According to Sohrabi et al., ECG with occlusion in LCx has a higher risk with a worse 30 days prognostic outcome than occlusion in RCA. One of the important factors in determining the location of the blockage on the ECG is the direction of deviation of the ST segment vector, which always leads to the infarct area. The vector deviation in RCA occlusion will point to the inferior and right sides. In contrast, in LCx occlusion, the vector deviation will point more posteriorly to the left or lateral.

Therefore, there will be more ST elevation in leads III > II in RCA occlusion and LCx ST-elevation occlusion in leads II > III. ST depression leads to aVL > I and is also highly predictive of occlusion in RCA. Tierala et al. describe three steps in identifying the culprit lesion. The first step in the algorithm is to assess ST elevation in leads II and III. If ST elevation in the lead II ≥III, the occlusion lies in LCx. If not, continue to the second step, assess the presence of ST-Elevation in V1 or isoelectric ST segment in V1 and ST depression in V2. If yes, that indicates occlusion in RCA, if not, proceed to step three, that is, ST depression in lead aVR>aVL indicates occlusion in LCx and vice versa.

There are various ECG and angiographic features that have a significant effect on the sensitivity of the ECG criteria for determining the culprit lesion. ECG changes in Inferior STEMI can be understood by knowing the anatomy of the coronary arteries. Mohanty A and Saran RK pointed out the importance of aVR segment depression as a sign of LCx occlusion. The specificity and sensitivity for lead aVR to predict LCx involvement were 94% and 70%, respectively. However, changes in lead aVR have lower sensitivity than other ECG criteria for determining the culprit lesion in Inferior STEMI. In this case, the ECG showed (1) ST elevation in leads III>II, (2) ST depression in leads aVL>I and no ST depression in aVR, indicating occlusion in the RCA, although the results of angiography showed LCx as the culprit lesion. In the study, Chia B et al reported that the presence of ST elevation in leads III ≥ II which is a diagnostic criterion for RCA occlusion with a sensitivity value of 97% and a specificity of 90%, but the results of this study found 2 cases that met in the ECG criteria but the results of angiography showed LCx as the culprit lesion. In our case, LCx occlusion was the presence of ST elevation in leads II>III, ST elevation in leads I>aVL, and ST depression in aVL was in STEMI with increased suspicion of LCx occlusion. Similar results were also reported in the study of Sahi R et al that assessed the role of ST depression in limb leads aVR and aVL for diagnosing Inferior STEMI and identified infarct-related artery (IRA) with results demonstrating that ST depression in lead aVR and aVL helps diagnose STEMI Inferior with LCx occlusion as the cause of IRA.

LCx branch occlusion occurs when the ST vector moves backward, resulting in ST-elevation II > III. When the LCx branch is occluded, the ST vector will move to the left at an angle with lead aVL. As a result, the range of ST-segment changes is smaller than that of RCA occlusion and LCx branch occlusion can cause ischemic damage to the lateral wall leading to lateral wall STEMI. However, the ECG results showing occlusion of the LCx were not found in this case. This result was in line with the case of Gul EE et al., who found RCA results on ECG results, but angiography results found LCx occlusion.

Using all these ECG criteria, we could predict the presence of RCA occlusion in our case before coronary angiography. However, from angiography, unexpected results were obtained, namely the presence of subtotal stenosis in proximal to distal, thrombus (+), 80% OMI stenosis in LCx, and dominant LCx. Based on previous statistical studies, the possibility of this finding can explain that LCx can provide a more important blood supply to several segments of the myocardium, including the inferior wall and even the right ventricle. Some populations can have this left dominant condition as much as 20%.

The limitations of this study because this is a case study, these results cannot be assumed for the entire general population. Further investigations with a larger sample size were required to identify the clinical significance of ECG in determining Infarct Related Artery in Inferior STEMI patients.

**CONCLUSION**

In conclusion, from our case, the use of the ECG criteria in predicting occlusion in LCx, namely the presence of ST elevation in leads II>III, ST elevation in leads I>aVL, and ST depression in aVL was in STEMI with increased suspicion of LCx occlusion.
the culprit lesion is very useful. It is also important in determining the culprit lesion's location in most cases. However, it has limitations, as in our case where the angiographic results did not match the predicted results according to the existing ECG algorithm, due to some situations such as if a person has left dominant. Therefore, further research is needed for conditions like this case.

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CONFLICT OF INTEREST

The authors stated that there is no conflict of interest in writing this article.

ETHICAL CONSIDERATION

The authors had gained consent from the patients to publish his case in an academic journal without revealing any personal identity and solely for academic purposes.

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AUTHOR CONTRIBUTIONS

NLSEW was responsible for the patient management and oversight of the patient's pharmacotherapy. FSMP was responsible for writing the report and conducting a follow-up examination in collaboration with NLSEW.

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