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# Utility of Ischemic Signs from Initial ECG in Detecting Culprit Vessels in NSTE–ACS Patients.

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**Abstract**

**Background:** Non-ST-Elevation ACS (NSTE-ACS) is a part of ACS that requires some special attention. Multivessel coronary disease (MVD) is common in patients with NSTE-ACS and is associated with difficulties in determining the main target of revascularization. ECG is the first-line diagnostic tool in the assessment of patients with suspected ACS. However, the utility of the ECG in localizing coronary culprit lesions in NSTE-ACS is not well established. This study was conducted to evaluate whether the pattern of the ischemic signs in ECG can be used to identify the coronary culprit vessel in patients with NSTE-ACS.

**Methods:** This is a single-centered cross-sectional study using secondary data. The data of all 101 patients with NSTE-ACS who were planned for revascularization procedures between January 2021 and December 2021 were collected from medical records. ECG with ischemic signs was classified into three locations of suspected coronary vessels with culprit lesions and it will be compared to its corresponding angiographic data. The accuracy data will be presented including both sensitivity and specificity.

**Results:** This study involved 75 men (74.3%) and 26 women (25.7%) with a mean age of 61.2±9.1 years old. There were 72 patients presented with ischemic signs from ECG with identifiable culprit vessels to be suspected. The sensitivity and specificity of ischemic signs ECG in localizing culprit vessels from angiography were 37.0% and 85.5% in LAD distribution, 38.1% and 81.3% in LCX distribution, and 41.1% and 85.1% in RCA distribution, respectively.

**Conclusion:** Overall ischemic signs in ECG gave the impression of modest accuracy with conspicuous key points that ECG distribution has high specificity in detecting culprit vessels but with low sensitivity. Thus, ischemic signs from initial ECG can be used to detect culprit vessels in NSTE-ACS patients.

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**Keywords:** Culprit Lesion, ECG, NSTEMI, UAP, PCI.

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## Introduction

Acute Coronary Syndrome (ACS) is a leading cause of death worldwide, including Indonesia.[1] Non-ST-Elevation ACS (NSTE-ACS) is a part of ACS that requires some special attention. The proportion of patients with NSTE-ACS increased from one-third in 1995 to more than half in 2015.[2] One of the most important pillars of NSTE-ACS therapy is the revascularization procedure. An initial revascularization approach is associated with a lower rate of the combined endpoint of death, myocardial infarction (MI), or refractory angina at 4 to 6 months follow-up. The benefits of this approach are most pronounced among patients with high and very high risk of cardiovascular events.[3-4] In the largest study to date of 21.857 patients with non-ST-elevation myocardial infarction (NSTEMI) and multivessel coronary disease (MVD), 53.7% of these patients underwent complete revascularization during Percutaneous Coronary Intervention (PCI) for NSTEMI, while the rest had PCI to infarct-related artery (IRA) only.[5]

The term culprit vessel or culprit lesion is used to define the coronary stenosis considered to be responsible for the ACS and its early recognition enables appropriate treatment.[6] Inability to correctly identify the culprit lesion may affect the clinical outcome.[7] In addition, MVD is common in patients with NSTE-ACS ranging in 40-80% of patients, which is associated with poor clinical outcomes and difficulties in determining the main target of revascularization.[4,8-9] There have only been a few studies of revascularization strategy in patients with NSTE-ACS which makes it challenging to assign the main revascularization target.[10]

The resting 12-lead ECG is the first-line diagnostic tool in the assessment of patients with suspected ACS.[11] ST-segment elevation seen on ECG accurately localizes the culprit vessel in ST-Elevation myocardial infarction (STEMI).[12] However, the utility of the ECG in localizing coronary culprit lesions in patients with NSTE-ACS is not well established and current literature is sparse.[8] This study was conducted to evaluate whether the pattern of the ischemic signs in ECG can be used to identify the coronary culprit vessel in patients with NSTE-ACS.

## Materials and Methods

This is a single-centered cross-sectional study using secondary data which was designed to answer whether specific ischemic signs from ECG in NSTE-ACS patients are related to culprit lesions in its corresponding angiographic data. The data of all patients with NSTE-ACS who were planned for revascularization procedure prior to discharge between January 2021 and December 2021 were consecutively collected from medical records. Exclusion criteria were STEMI evolution during hospitalization, non-obstructive coronary lesion, history of Coronary Artery Bypass Graft (CABG), and any severe valvular lesion. 101 patients were finally accounted in this research to be analyzed. The detailed process of patient selection and recruitment may be observed in **Figure 1**.

The diagnosis of NSTE-ACS adheres to the criteria determined by ESC guideline in 2020.4,11 Patients with clinical symptoms of myocardial ischemia which were suspected to be ACS were examined by both ECG and cardiac enzyme marker (troponin-I) at first point contact in the emergency unit. The diagnosis of UAP and NSTEMI was established based on the value of troponin-I.[11] A-standard-twelve-lead ECG was performed in the Emergency Room (ER) within 30 minutes of the patient's arrival. Ischemic signs included in the ECG assessments were T wave inversion ≥ 1 mm and ST segment depression ≥ 0.5 mm in two contiguous leads with a prominent R wave and R/S ratio > 1.[8] The ECG results were assessed by a cardiologist blinded to patients' angiography results. Troponin I measurement is based on a blood vein sample processed by PATHFAST® cardiac biomarker analyzer. The upper limit value for troponin-I with such measurement is 0,02 ng/mL. Revascularization procedures varied in time based on the clinical condition of the patient and the availability of the catheterization lab facility. It ranged from as early as within 5 hours until 4 days of hospitalization. Echocardiographic data was also obtained within hospitalization by Transthoracic Echocardiogram (TTE). All measurements of echocardiography adhere to the guideline of echocardiography published by the American Society of Echocardiography (ASE) in 2019.[13]

Invasive coronary angiography of all patients was performed prior to discharge. The degree of stenosis lesions was analyzed visually by a team of cardiovascular

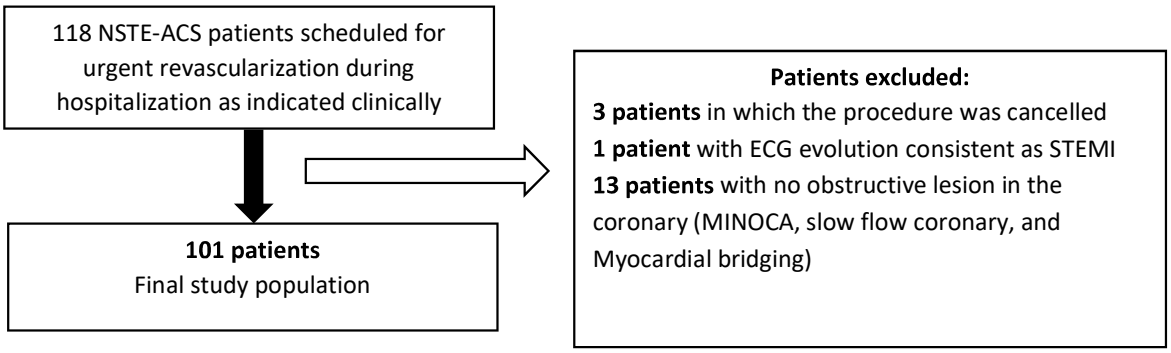


**Figure 1.** Patient Selection and Recruitment.

**Table 1.** Electrocardiographic (ECG) criteria used for localization and nomenclature in non-ST elevation acute coronary syndrome (NSTE-ACS)[8,9].

| Involved artery | Location | Involved ECG lead |
|---|---|---|
| LAD | Anterior or Septal | • [V1–V2] = Septal or mid-distal LAD |
| | | • [aVL ± I ± V2–V3] = First diagonal |
| | | • [V1–V3 with extension to V4–V6 without aVL/I = Mid LAD |
| | | • [V1–V6 + aVL ± I] = proximal LAD |
| LCX or OM | Lateral | • Q-wave equivalents of abnormally prominent R waves in [V1–V2] |
| | | • [I, aVL] and/or [V5-V6] |
| RCA or LCX | Inferior | • [II, III, aVF] |

LAD - Left Anterior Descending Artery, LCX - Left Circumflex Artery, OM - Obtuse Marginal Artery, RCA - Right Coronary Artery..

intervention cardiologists who were blinded to the ECG results. The culprit lesion in each patient was determined by the team based on the criteria specified by ESC guideline in 2015. Culprit lesion was determined when there were morphological features suggestive of acute plaque rupture. These features should fulfill at least two of the following features: 1) intraluminal filling defects consistent with thrombus, 2) plaque ulceration, 3) plaque irregularity, 4) dissection, or 5) impaired flow.[4] When there was more than one lesion which fulfilled those features, the one with more severe features was chosen to be the culprit subjectively by the team.

ECG with ischemic signs was classified into three locations of suspected coronary vessels with culprit lesions. This classification is modified from previous recommendations for Q-wave myocardial infarction by the International Society for Holter and Non-invasive Electrocardiography.[8,12] Since the dominancy of coronary vessels anatomically could not be determined at initial ECG, We could not differ whether ischemic signs at the inferior lead were derived from RCA or LCX. Therefore, we presume any ischemic inferior lead was derived from RCA. This modified method of classification can be observed in **Table 1**. Patients with no ischemic signs in ECG was specific to any locations of suspected coronary vessels with culprit lesions will be marked as unidentified culprit vessels by ECG and will also be accounted in the statistical analysis. This classification will be compared and analyzed statistically with its corresponding culprit vessel from the angiographic data. This statistical analysis will result in sensitivity, specificity, Positive Predictive Value (PPV), and Negative Predictive Value (NPV) in each ECG ischemic finding.

Normally distributed continuous variables are presented in mean ± standard deviation. Non-normally distributed continuous variables are presented in the

**Table 2.** Baseline characteristic.

| Variables | Value (n=101) |
|---|---|
| Gender | |
| Male, n(%) | 75 (74.3) |
| Female, n(%) | 26 (25.7) |
| Age (years) | 61.2 ± 9.1 |
| BMI (kg/m2) | 25.7 ± 5.0 |
| Risk Factors | |
| Smoking, n(%) | 49 (48.5) |
| Hypertension, n(%) | 58 (57.4) |
| Diabetes, n(%) | 43 (42.6) |
| Dyslipidemia, n(%) | 48 (47.5) |
| Obesity, n(%) | 53 (52.5) |
| Diagnosis | |
| NSTEMI, n(%) | 54 (53.5) |
| UAP, n(%) | 47 (46.5) |
| Laboratory Parameter | |
| Creatinine (mg/dL) | 1.07 (0.90 – 1.32) |
| Troponin I (ng/mL) | 0.023 (0.004 – 0.236) |
| Echocardiographic data | |
| LVEDD (mm) | 48.8 ± 9.7 |
| LVESD (mm) | 32 (28 – 41) |
| LVEF (%) | 58 (41.5 – 65) |
| TAPSE (mm) | 22 (18 – 25) |

BMI, Body Mass Index; NSTEMI, Non-ST Elevation Myocardial Infarction; UAP, Unstable Angina Pectoris; eGFR, Estimated Glomerular Filtration Rate; LVEDD, Left Ventricular End Diastolic Diameter; LVESD, Left Ventricular End Systolic Diameter; LVEF, Left Ventricular Ejection Fraction; TAPSE, Tricuspid Annular Plane Systolic Excursion.

median and Interquartile Range (IQR). Categorical data are presented as frequencies and percentages. The main statistical analysis is an X2 test. Statistical analysis was performed using IBM SPSS statistics 25 (SPSS Inc, Chicago, Illinois). The p-value less than 0.05 was considered statistically significant.

## Results

The initial data identified 118 NSTE-ACS patients. The final population included 101 patients of which 74.3% were males and 25.7% were females. The mean age in this research was 61.2 ± 9.1. The proportion of NSTEMI diagnoses in this population was 53.5%. The average Body Mass Index (BMI) was 25.7 ± 5.0. Risk factors varied from smoking, hypertension, diabetes, dyslipidemia, and obesity. Echocardiography data showed modest Left Ventricular Ejection Fraction (LVEF) and Tricuspid Annular Plane Systolic Excursion (TAPSE). These detailed baseline characteristics can be observed in **Table 2**.

Electrocardiographic analysis revealed ECG with ischemic signs in 71.3% cases. Axis distribution among ECG analysis of the patients showed 59.4% normoaxis, 30.7% left axis deviation, and 9.9% right axis deviation. Based on ECG criteria, the culprit lesion was located in the Left Anterior Descending (LAD) coronary artery in 24.8% cases, in Left Circumflex artery (LCX) in 22.8%, in Right Coronary Artery (RCA) in 23.8%, and 28.7%

**Table 3.** ECG and Angiographic analysis of the patients.

| Variables | Value (n=101) |
|---|---|
| Sinus rhythm, n(%) | 99 (98) |
| Ischemic signs from ECG, n(%) | 72 (71.3) |
| Presence of T wave inversion, n(%) | 69 (68.3) |
| Presence of ST depression, n(%) | 21 (20.8) |
| ECG Axis | |
| Normoaxis, n(%) | 60 (59.4) |
| Left axis deviation, n(%) | 31 (30.7) |
| Right axis deviation, n(%) | 10 (9.9) |
| Culprit coronary vessel based on ECG criteria | |
| LAD, n(%) | 25 (24.8) |
| LCX, n(%) | 23 (22.8) |
| RCA, n(%) | 24 (23.8) |
| Unknown, n(%) | 29 (28.7) |
| Vessels involved with stenosis | |
| 1 vessel disease, n(%) | 15 (14.9) |
| 2 vessels disease, n(%) | 24 (23.8) |
| 3 vessels disease, n(%) | 62 (61.4) |
| Culprit coronary vessel angiographically | |
| LAD, n(%) | 46 (45.5) |
| LCX, n(%) | 21 (20.8) |
| RCA, n(%) | 34 (33.7) |
| Revascularization procedure | |
| Stent, n(%) | 80 (79.2) |
| Multiple stents, n(%) | 12 (11.9) |
| Planned for urgent CABG, n(%) | 17 (16.8) |
| No cathlab intervention, n(%) | 4 (4.0) |

LAD, Left Anterior Descending coronary artery; LCX, Left Circumflex coronary artery; RCA, Right Coronary Artery.

with the unknown location of the culprit lesion.

Coronary angiography as a part of revascularization procedure was performed to detect and analyze culprit lesions. There were 45.5% cases in which the culprit lesion was found at LAD. Furthermore, the culprit lesion was identified at LCX in 20.8% cases and at RCA in 33.7% cases respectively. Single vessel disease was identified in 14.9% patients, while 85.1% presented with MVD. Out of all 101 patients, 79.2% underwent stenting procedures, 16.8% were planned for urgent CABG, while 4% received a conservative medical treatment. Comprehensive data regarding ECG analysis and coronary angiography of the patients is presented in **Table 3**.

The culprit vessel suspected by ECG criteria was then compared to the angiography result. Out of 25 patients classified as LAD culprit vessel based on ECG criteria, 17 of them were identified with LAD culprit vessel based on their angiography result, 3 of them with LCX culprit vessel, and 5 of them with RCA culprit vessel. Out of 23 patients presented with LCX culprit vessels based on their ECG analysis, 8 of them have their culprit lesion located in LCX by angiography. Out of 24 patients with RCA culprit vessels based on the

**Table 4.** Comparison of ECG distribution of culprit vessel with angiography (p=0.023).

| ECG distribution of culprit vessel | Culprit vessel (angiography) | | | Total |
|---|---|---|---|---|
| | LAD | LCX | RCA | |
| LAD | 17 | 3 | 5 | 25 |
| LCX | 9 | 8 | 6 | 23 |
| RCA | 6 | 4 | 14 | 24 |
| Unknown | 14 | 6 | 9 | 29 |
| Total | 46 | 21 | 34 | 101 |

LAD, Left Anterior Descending coronary artery; LCX, Left Circumflex coronary artery; RCA, Right Coronary Artery.

**Table 5.** Accuracy of ECG distribution in localizing culprit vessels from angiography.

| ECG distribution | Sensitivity, % | Specificity, % | PPV, % | NPV, % |
|---|---|---|---|---|
| LAD | 37.0 | 85.5 | 68.0 | 61.8 |
| LCX | 38.1 | 81.3 | 34.8 | 83.3 |
| RCA | 41.1 | 85.1 | 58.3 | 74.0 |

LAD, Left Anterior Descending coronary artery; LCX, Left Circumflex coronary artery; RCA, Right Coronary Artery; PPV, Positive Predictive Value; NPV; Negative Predictive Value.

ECG, 14 of them were confirmed to have the culprit lesion located in RCA by angiography. Meanwhile, out of 29 patients with unknown culprit vessels based on the ECG, 14 of them were identified with LAD culprit vessels, 6 with LCX culprit vessels and 9 with RCA culprit vessels. Detailed distribution of this comparison is presented in **Table 4**.

Accuracy data of ischemic signs in detecting culprit vessels are presented in **Table 5.** The sensitivity and specificity of ECG on localizing culprit vessels from angiography were 37.0% and 85.5% in LAD distribution, 38.1% and 81.3% in LCX distribution, and 41.1% and 85.1% in RCA distribution, respectively.

## Discussion

Out of 101 patients assessed in this study, 74.3% of them were males and 25.7% were females with a mean age of 61.2 ± 9.1. The average Body Mass Index (BMI) was 25.7 ± 5.0. These characteristics resemble NSTE-ACS population in general which was dominated by male.[14] Risk factors of Coronary Artery Disease (CAD) are divided into traditional/classic risk factors and novel/emerging risk factors. Classic risk factors include age with a mean of 62.[7] (being the strongest risk factor), male sex, prior MI, hypertension, tobacco use, diabetes mellitus, dyslipidemia, and family history of CAD. Emerging risk factors include a sedentary lifestyle, overweight/obesity, psychosocial factors (anxiety/depression), chronic kidney disease (CKD), obstructive sleep apnea, and environmental pollutants.[14-16] Risk factors recorded among patients in this study were smoking (48.5%), hypertension (57.4%), diabetes (42.6%), dyslipidemia (47.5%), and obesity (52.5%). Hence, the population of this study is similar to previous studies regarding NSTE-ACS.

Ischemic signs from ECG examination were found in 71.3% patients, meaning that almost 30% of the population in this study had normal ECG. This finding might seem unlikely, but 46.5% of patients in this study were diagnosed with UAP. This profile elucidates the result. Moreover, based on the ECG criteria we were able to identify suspected culprit vessels in 71.3% of the population, leaving the rest to be categorized as unknown culprits due to either normal ECG findings or no ischemic signs in the ECG that could be categorized based on the criteria in **Table 1**. This is in line with a previous study which showed that identifying culprit vessels based on ECG is challenging and less likely to be precise.[6]

The culprit coronary vessel identified by angiography in this study was mostly the LAD, for as many as 45.5% of the population. This result is similar to a previous study which identified culprit lesions in NSTE-ACS patients to be mostly in the LAD, which was in 40.9% of their patients.[6] Moreover, three-vessel disease in our study was found in 61.4% of patients. This corresponds to a previous study which found that MVD is common in patients with NSTE-ACS.[8] Furthermore,

revascularization procedure (PCI) in our population was done in 79.2% patients. It was recorded that 17 patients (16.8%) needed urgent CABG. A previous publication from the Acute Coronary Treatment and Intervention Outcomes Network Registry – Get With The Guidelines, reported that 11.5% of patients presenting with NSTEMI underwent CABG.[17]

The main result found in this study showed that the culprit vessels identified based on ECG criteria and those identified through angiography analysis were significantly related statistically. Moreover, we were able to present the accuracy value including sensitivity, specificity, PPV, and NPV for all culprit vessels based on ECG analysis. Our study revealed that ischemic signs from ECG had low sensitivity (37.0% for LAD, 38.1% for LCX, and 41.4% for RCA) but high specificity to detect the culprit vessels (85.5% for LAD, 81.3% for LCX and 85.1% for RCA). These findings are in line with previous studies that showed low sensitivity (28.4% for–41.5%) and good specificity (88.7%–96.5%) in NSTE-ACS.[6,18-20] In addition, a previous study which was conducted in patients with PCI-treated NSTEMI successfully showed that ECG changes before and after revascularization have modest accuracy in localizing culprit vessels.[8]

The specificity of ischemic signs from ECG to identify culprit vessels in this study was quite high for all coronary arteries even though the sensitivity was low. This might happen because most NSTE-ACS patients have MVD. Our study presented 86 patients (85.2%) with MVD and 15 patients (14.9%) with single coronary artery disease. In patients with NSTE-ACS and MVD, the relationship between ischemic signs from ECG and culprit vessel is less clear. One previous study found that higher lesion complexity such as MVD was associated with more difficulties to identify the culprit from NSTE-ACS.[6] Ischemic signs from ECG, especially in MVD, could be less accurate for culprit detection because the ischemic process could involve multiple areas of localization. On the other hand, these changes do not necessarily represent culprit vessels that require urgent revascularization.

This study included both NSTEMI and UAP cases to be analyzed statistically. This might seem uncommon because previous studies included only NSTEMI patients.[8,18,20] Around 47 patients (46.5%) were diagnosed with UAP in this study. Much effort is needed to diagnose UAP based on clinical assessment alone, it is even more challenging with normal ECG and normal cardiac enzymes.[21] UAP was still included in our research to fulfill all NSTE-ACS spectrum. Patients with unknown culprit lesions based on ECG criteria were also included in this study. However, this study excluded patients with non-obstructive coronary blood flow as well as myocardial bridging, coronary artery ectasia, and slow-flow coronary vessels. These conditions might explain why the sensitivity number in our study is quite low.

Different from previous study, no dynamic changes in ECG were accounted in this study as the sole ECG to be analyzed was the one recorded on patients' admission in the ER. The repercussion of this method is that we could not distinguish the ischemic signs we identified to be an acute or chronic process. Previous studies utilize these dynamic changes in ECG examination by analyzing two sets of serial ECGs which were conducted before and after revascularization procedure. It showed modest accuracy in localizing culprit vessels.[8] We do not adopt such a method because the essence of predicting the culprit vessel prior to angiography could not be achieved.

This research contains some limitations. Our study does not take LCX/RCA coronary dominance to be accounted in coronary vessel analysis. This drawback is being accepted because coronary dominancy anatomically could not be determined at initial ECG and can only be determined by angiogram. We did not reassess the patients' ECG after the dominant vessels were determined through angiography data. However, since the right dominant coronary system is a lot more common than the left one, the result of this research is still representative.[22] Another limitation of our study is that we did not compute detailed anatomical characteristics of the culprit lesion. Rather than presented in a 16-segment-coronary assessment on the vessel's diameter, the culprit lesion identification based on angiography in this study was presented according to the culprit location in 3 coronary arteries (LAD, LCX, RCA). This could affect or even lessen the specificity of the ECG in determining involved coronary artery. Finally, the number of samples in this research is 101 patients collected in one year period. Increasing the sample size by adding more periods might give valuable data that represents better value to the population.

## Conclusion

This study shows that overall ischemic signs in ECG gave the impression of modest accuracy with conspicuous key points that ECG distribution has high specificity in detecting culprit vessels but with low sensitivity. Thus, ischemic signs from initial ECG can be used to detect culprit vessels in NSTE-ACS patients.

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