Fall 12-14-2018

Diagnostic effectiveness of lead aVR as a STEMI equivalent

Phung Vu
James Madison University

Karen Hayes
James Madison University

12-12-2018

Diagnostic Effectiveness of Lead aVR as a STEMI Equivalent

Karen Hayes, PA-S
James Madison University

Phung Vu, PA-S
James Madison University

Diagnostic Effectiveness of Lead aVR as a STEMI Equivalent

Karen Hayes & Phung Vu

Abstract

Background: Acute coronary syndrome (ACS) encompasses a collection of three acute processes related to myocardial ischemia. These include: unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI). The 12-lead electrocardiogram (ECG) is a crucial tool in the diagnosis and risk stratification of ACS. Unlike the other 11 leads, lead augmented Vector Right (aVR) has been long neglected until recent years. Recent investigations have shown that an analysis of ST-segment shift in lead aVR provides useful information on the coronary angiographic anatomy and risk stratification in ACS. This implies that the use of lead aVR can be effective in the early detection and extent of tissue ischemia, increasing the chances of acute myocardial infarction (AMI) survival. **Objective:** The purpose of this review is to determine whether the use of lead aVR can be used as a STEMI equivalent compared to the standard STEMI criteria as defined by the American Heart Association to predict proximal left anterior descending (LAD) or left main coronary artery (LMCA) occlusion in order to decrease door to balloon time and overall mortality. **Methods:** A PubMed search was conducted using the following search terms and filters: aVR, STEMI, and myocardial infarction articles in the last 10 years, English language, randomized control trials, meta-analysis reviews. Articles were excluded if not specific to lead aVR, emphasis on treatment rather than diagnosis, no full text of the article was available, and low participant numbers. **Conclusion:** While the use of lead aVR in insolation as a STEMI equivalent remains unclear, there is evidence supporting that ST elevation in lead aVR is associated with higher mortality in the presence of a recognized STEMI. It is also suggested that ST elevation (STE) in lead aVR may involve the LAD or all three main coronary arteries (triple vessel disease). This is promising in early recognition of tissue ischemia and can be used as a potential marker of disease severity.

Keywords: *Left Anterior Descending Coronary Artery (LAD); Left Main Coronary Artery (LMCA); Left Bundle Branch Block (LBBB); Right Bundle Branch Block (RBBB); Percutaneous Coronary Intervention (PCI); Coronary artery disease (CAD); ST segment elevation myocardial infarction (STEMI); Non-ST segment elevation myocardial infarction (NSTEMI), ST segment elevation (STE).*

Introduction

The 12 lead ECG is a diagnostic tool that aids in the evaluation of coronary artery disease (CAD). A ST segment elevation myocardial infarction (STEMI) is a distinct pattern detected on 12-lead ECG and is a type of myocardial infarction that indicates occlusion of one of the main coronary arteries supplying the heart muscle.[1] Clinicians are provided a strict set of criteria as to what qualifies as a STEMI and subsequent emergent activation of the cardiac catheterization lab for reperfusion therapy. The American Heart Association defines a STEMI as ST elevation at the J point in at least two contiguous leads. Contiguous leads view the same anatomical portion of the left ventricle. The 11 leads are divided based on the portion of the left ventricle they are viewing; inferior leads (II, III, and aVF), septal leads (V1 and V2), anterior leads (V3 and V4), and lateral leads (I, aVL, V5, and V6). In this functional categorization, the remaining lead, aVR, is frequently disregarded. However, lead aVR may provide the clinician with valuable clinical

information due to its different directional orientation than all other leads.[2] It has been proposed that ST elevation in aVR should be treated as a STEMI equivalent given the appropriate clinical context. With clinical symptoms of myocardial ischemia, ST segment elevation in aVR greater than or equal to 1 mm is suggested to signify significant proximal LAD or LMCA occlusion. Just as reciprocal changes add to the validity of a classic STEMI, reciprocal changes represented as widespread ST depression and ST elevation in aVR greater than V1, adds to the validity of significant left main disease. The left main coronary artery (LMCA) arises from the aorta just above the aortic valve and is responsible for supplying a significant portion of myocardium. LMCA occlusion can lead to devastating consequences, including cardiogenic shock and death, especially if unrecognized. Lead aVR may have the ability to identify these high-risk patients and activate the cardiac catheterization lab earlier for intervention, decreasing ischemic complications.[3]

While the mechanism behind ST segment elevation in lead aVR is unclear, it is suggested that it may be the result of being electrically opposite of the left sided leads (I, aVL, V4-V6). Occlusion of the LMCA would result in ST depression in the left sided leads with reciprocal changes in aVR manifested by ST elevation. Another proposed mechanism involves the fact that aVR looks directly at the right side of the heart (along with V1), unlike the other 10 leads. The basal portion of the interventricular septum is located in the right upper portion of the heart and is supplied by the first branch of the left anterior descending coronary artery (LAD). It would therefore suggest that the culprit lesion would be located in the proximal LAD or LMCA causing insufficient coronary artery blood flow.[4]

Many studies have been performed looking at the diagnostic ability of aVR as well as potential limitations. The presence of ST elevation in lead aVR may not be entirely specific to LMCA or proximal LAD occlusion. It may also suggest the presence of triple vessel disease or diffuse subendocardial ischemia. Furthermore, some studies argue that it is unable to distinguish between LMCA occlusion versus insufficiency, indicating some blood flow is still present. This study aims to investigate if the predictive value of ST elevation in lead aVR is high enough to be used as a diagnostic STEMI equivalent.

PICO

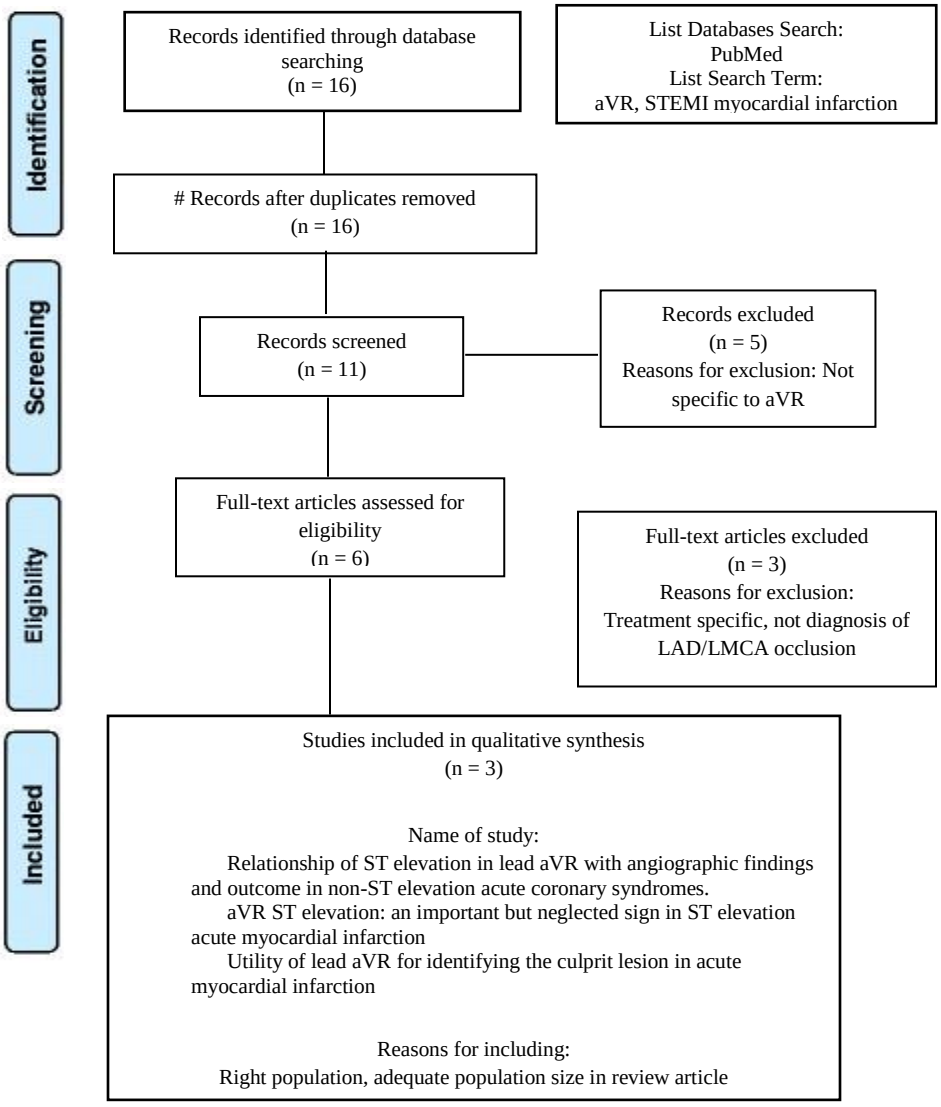
P: Patients aged 40 and above presenting with acute coronary syndrome (ACS) as defined by the presentation of STEMI, NSTEMI, or unstable angina.
I: Evaluation of lead aVR as a STEMI equivalent
C: Standard STEMI criteria as defined by the American Heart Association
O: To predict proximal LAD or LMCA occlusion in order to decrease door to balloon time and overall mortality

Clinical Question

Patients aged 40 and above presenting with acute coronary syndrome as defined by the presentation of STEMI, NSTEMI, or unstable angina, does the use of Lead aVR as a STEMI equivalent provide an earlier diagnosis of proximal LAD or LMCA occlusion in order to decrease door to balloon time and overall mortality compared to standard STEMI criteria.

Methods

An initial literature search of PubMed using the search terms “aVR” and “STEMI” and “myocardial infarction” yielded 16 results. Inclusion criteria included randomized control trials and meta-analyses with publication dates within the last 10 years. No duplicate articles were removed. 11 studies were screened for relevance to the research subject matter. For example, some studies were removed because they were not specific to lead aVR, and some focused more on treatment rather than diagnosis. A total of three articles were selected to be included in this analysis based on relevance and population size.



This PRISMA flow chart helped identify the studies involved in the analysis. The articles that made it to the screening phase were evaluated by two reviewers based on criteria highlighted in the chart and the outcome measurements used by the studies that addressed the clinical question.
http://prisma-statement.org/prismastatement/flowdiagram.aspx

Results

Study #1:
Relationship of ST elevation in lead aVR with angiographic findings and outcome in non-ST elevation acute coronary syndromes.[5]

Objective:
In the setting of a NSTEMI, the goal of this study was to evaluate the connection between ST elevation in lead aVR with coronary artery angiographic findings on cardiac catheterization, as well as with mortality rates.

Study Design:
This study is an ongoing prospective electrocardiographic sub-study of the Global Registry of Acute Coronary Events (GRACE). The GRACE registry analyzes patient populations with ACS in 13 countries. The electrocardiographic sub-study used 39 sites in 11 countries, with 8,202 patients initially enrolled. Patient inclusion and exclusion criteria for the prospective study were based on clinical symptoms and ECG findings (Table 1).

Table 1. Inclusion and exclusion criteria for study participation in the electrographic sub-study of the GRACE registry.[5]

Inclusion Criteria	**Exclusion Criteria**
At least 18 years old Admitted with non-ST segment elevation myocardial infarction	Serious comorbidity Poor quality ECG Ventricular or paced rhythm STEMI on ECG Left bundle branch block on ECG

A total of 5,064 patients met inclusion and exclusion criteria and formed the sub-study cohort. The patients were divided into three groups based on ST segment elevation in lead aVR: no ST elevation, minor ST elevation (0.5-1mm), and major ST segment elevation (>1mm). The GRACE risk score evaluates for age, gender, amount of ST segment deviation, vital signs, biomarkers, and Killip class. A logistic regression was therefore utilized to adjust for these confounding variables to be able to evaluate ST aVR ST elevation independently. Reading of the ECGs were all performed at the Canadian Heart Research Centre. The clinical data and outcomes were blinded to the interpreter. ST segment deviation was measured at the J point and rounded to the nearest 0.5mm.

Patients were followed up with six months after hospital discharge via telephone call. 88.2% of those entering the study were able to be followed up with. The Kaplan-Meier method was utilized to produce survival curves, and hazard ratios were calculated.

Study Results:

Of all the patients in the substudy diagnosed with a NSTEMI, 5.8% were found to have 0.5-1mm of ST elevation in lead aVR. 1.5% had greater than 1mm of ST segment elevation in aVR. Overall, patients with ST segment elevation in aVR were found to have concurrent diffuse ST depression in other leads. These patients in general were also older, presented with more tachycardia, and had a worse Killip class. It was found that patients with greater than 1mm of ST elevation in lead aVR had higher in hospital mortality rates compared to those with no or minor aVR ST elevation (P=0.03). Mortality rate was also evaluated six months after hospital discharge. There was a 7.6% mortality rate for patients that presented with no STE in aVR, 12.7% for those with minor elevation, and 18.3% for those with major STE (Figure 1).

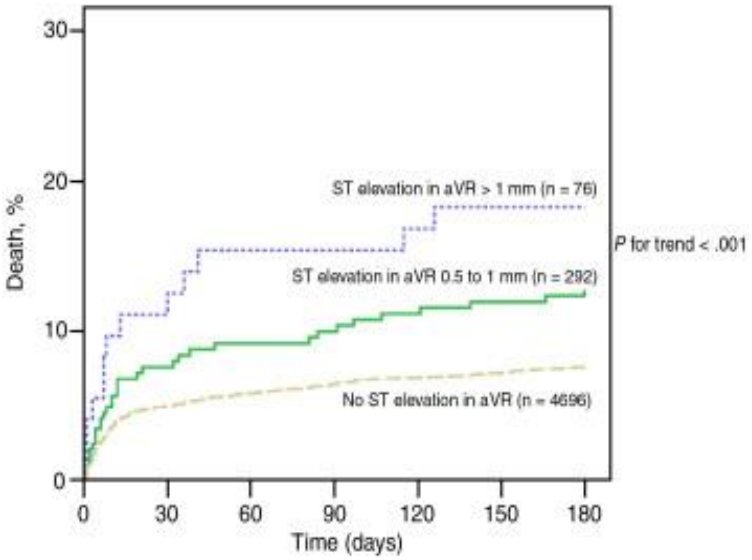


Figure 1. Kaplan-Meier curves analyzing relationship between ST elevation in aVR with mortality rates.[5]

It appears as if minor or major STE in aVR correlates with an increased mortality rate when compared to those with no ST deviation in aVR. However, when analysis was performed with the multivariable Cox regression tool analyzing components of the GRACE risk score, it was shown that aVR STE was not an independent predictor of mortality. Cox regression tool determined that the effects of the predictor variables listed in the GRACE risk score were maintained over time with the addition that it had no effect on the survival time of patients.

The study also looked at the potential of STE in aVR to predict the culprit lesion in an acute myocardial infarction. It was found that the presence of STE in aVR was significantly more common in left main coronary artery occlusion or triple vessel disease than any other location (Table 2). The authors concluded that the presence of STE greater than 1mm in lead aVR was predictive of left main coronary artery disease and/or triple vessel disease.

Table 2. Angiographic findings comparing patients with no, minor, or major STE in lead aVR.[5]

% of patients	Overall (N=2416)	No STE in aVR (n=2252)	0.5-1mm STE in aVR (n=30)	>1mm STE in aVR (n=34)	P value for trend
LAD stenosis	59.6	59.2	66.2	61.8	0.20
LCx Stenosis	50.0	48.6	70.8	64.7	<.001
RCA stenosis	55.0	54.4	61.5	70.6	0.02
LM coronary stenosis	5.4	5.1	9.2	14.7	0.002
3-vd	24.0	23.3	32.3	44.1	<0.001
LM/3 VD	27.0	26.1	36.2	55.9	<0.001

Overall, this study concluded that while STE in aVR alone may not have diagnostic potential, it was a predictor of severity and extent of disease. The unadjusted in-hospital and 6-month mortality were significantly higher in those with major STE in aVR (>1mm) as opposed to those with no or minor STE (0.5-1mm) in lead aVR. While these findings were not supported in isolation, it was suggested that in combination with widespread ST depression, major STE in lead aVR (>1mm) may be used to identify patients with severe coronary artery disease.

Study Critique:

There were many strengths to this study. The patient population consisted of ACS patients diagnosed with an NSTEMI. This produced a patient population that eliminated variables such as ST elevation in other leads besides aVR, but still necessitated a cardiac catheterization in order to verify results. The study population was also from 11 different countries in varying settings, which may add to the generalizability of the results. The study also adjusted for multiple confounding variables by utilizing the GRACE risk model. The GRACE risk model allowed the study to look at lead aVR in isolation to determine its prognostic value. Bias were minimized by utilizing a blinded method in interpreting the ECGs.

Some limitations of this study include the population size. While there were 5,064 patients overall, only 368 presented with STE in lead aVR. Of those 368 patients, there were 76 with STE in lead aVR greater than 1mm. Of note, not all patients received cardiac catheterizations depending on their clinical presentation and the physician discretion. This could have skewed the overall data on ability to predict severity and location of coronary disease. There was also an 11.3% loss to follow up at six months for undisclosed reasons. It was also suggested that the prognostic value of STE in lead aVR might have been miscalculated given that the ECGs analyzed were those performed on admission to the hospital. This does not allow for trending of ST segment deviation throughout the course of hospitalization and disease progression.

Study #2:
aVR ST elevation: an important but neglected sign in ST elevation acute myocardial infarction.[6]

Objective:
The goal of this study was to determine the prognostic value of ST elevation in lead aVR as an indicator of mortality risk with an acute myocardial infarction.

Study Design:
This study utilized data from the Hirulog and Early Reperfusion/Occlusion-2 (HERO-2) trial to determine if there was a relationship between ST segment elevation in lead aVR and 30-day mortality. The goal of the initial HERO-2 trial was to analyze the effect of different anticoagulation agents on 30-day mortality in patients receiving fibrinolytic therapy during an acute myocardial infarction. In the HERO-2 study, 17,073 patients were considered for evaluation. Patient inclusion and exclusion criteria for the trial was based on clinical symptoms and ECG findings (Table 3).

Table 3. Inclusion and exclusion criteria for study participation in HERO-2 trial.[6]

Inclusion Criteria	**Exclusion Criteria**
>30 minutes of ischemic chest pain with ST segment MI or new onset LBBB	Chest pain not consistent with ischemia Chest pain for greater than 6 hours
Within 6 hours of symptom onset	No ECG evidence of myocardial infarct

Patients were randomized to receive either bivalirudin or heparin, as well as streptokinase and aspirin. During the protocol, the patients received ECG tracings at random and at 60 minutes post administration of streptokinase, which was utilized as a fibrinolytic agent. The ECGs were interpreted by eight trained technicians at the Green Lane Hospital. Technicians were blinded to study participants and treatment groups. The HERO-2 trial concluded that there was no statistical difference between the two groups.

This sub-study obtained the information from the HERO-2 trial and analyzed the ECGs. The ECGs were all consistent with an acute MI and were included/excluded based on technical findings (Table 4). Of the 17,073 patients that were studied in the HERO-2 trial, 325 were excluded due to the presence of a left ventricle conduction delay (LBBB) obscuring the value of the ST segment, 691 due to RBBB, and 717 due to ventricular rhythm, paced rhythm, evidence of preexcitation syndrome, or poor-quality ECG. After exclusion criteria were applied, 15,315 patients remained in the study.

Table 4. Inclusion and exclusion criteria for lead aVR study participation.[6]

Inclusion Criteria	**Exclusion Criteria**
Sinus or atrial rhythm	Interventricular conduction delay (LBBB)
Good quality ECG tracing	Ventricular or paced rhythm

	Pre-excitation syndrome Poor quality ECG with artifact

The ST segment was analyzed in all 12 leads ST segment deviation was measured 60ms past the J point, and rounded to the nearest 0.5mm. This information was utilized to perform statistical analysis via logistic regression to determine if ST elevation in lead aVR greater than or equal to 1 mm was prognostic of 30-day mortality. The logistic regression model is a predictive analysis tool that looks at the relationship between two variables. In this case, the model was adjusted for total amount of baseline ST segment elevation/depression in other 11 leads, with the addition of age, sex, vital signs, time of symptom onset, and prior AMI.

Study Results:

Of the 15,315 patients studied with an acute MI, it was found that 7,299 were anterior in location, and 8,016 with inferior in location. There were 779 patients presenting with an anterior MI with ST segment elevation in lead aVR (greater than 1mm), and 365 of the patients with an inferior MI had ST elevation in aVR (greater than 1mm). In comparison to the patients without ST segment elevation in aVR, it was generally noted that patients with elevation tended to be older and had a history of prior MI.

The study concluded that patients with ST segment elevation in aVR greater than or equal to 1mm, regardless of infarct location, had a higher 30-day mortality rate. Those with an anterior MI and ST elevation in aVR had a 15.5% mortality rate compared to those without aVR findings at 12.2% (P=0.0069). Those with an inferior MI had a 15.9% mortality rate compared to 6.5% without aVR involvement (P value less than 0.0001). It was concluded that ST elevation in lead aVR provided important information on prognosis after an acute myocardial infarction.

Study Critique:

The limitations of this study include the population utilized and location. The study participants were initially chosen based on criteria for the HERO-2 trial, which included those patients who qualified for both anticoagulation and fibrinolytic therapy. This may differ from clinical populations, in that not all patients are candidates for both anticoagulation and fibrinolytic therapies. The HERO-2 trial was also performed in non-Western countries, which could have contributed to overall mortality if there was no access to primary PCI. Other risk factors may have also played a contributory role in mortality to include age and previous history of AMI. It was stated that there was a general trend (although not analyzed statistically) that the older individuals with previous history tended to have ST elevation in aVR. Therefore, aVR may be used as a simple way to quickly analyze risk factors in a clinical situation where you may not be able to obtain clinical information.

A strength of this study was the population size. It looked at a large cohort of individuals diagnosed with an acute MI based on ECG findings. While the study looked at aVR STE in the context of a previously recognized STEMI based on standard criteria, rather than in isolation, it still provided valuable information of the utility of aVR. Establishing the prognostic value of aVR will hopefully lead to further studies about the implementation of aVR as a STEMI equivalent.

Study #3:

Utility of lead aVR for identifying the culprit lesion in acute myocardial infarction.[7]

Objective:

This literature review aimed to evaluate lead aVR as a tool to identify the vessel involved in an acute myocardial infarction.

Study Design

A systematic search strategy was utilized, and data pooled in order to analyze lead aVR as a diagnostic tool. MEDLINE and Google Scholar were searched for relevant data, with key term "aVR," "ischemia," "myocardial infarction," and "ST segment elevation and depression." Studies were evaluated for relevance and confounding factors for inclusion and exclusion criteria (Table 5).

Table 5. Inclusion and exclusion criteria for study participation.[7]

Inclusion Criteria	**Exclusion Criteria**
Typical Chest Pain	Left bundle branch block
Clinically significant ST deviations	Left ventricular hypertrophy
Elevation of coronary enzymes	Previous history of MI
Coronary angiography with known culprit lesion	Cardiac surgery

The information obtained from the literature review was placed in 2 x 2 contingency tables and the Fisher Exact test was utilized. The Fisher Exact test is used to determine statistical significance when analyzing the association between two variables. The data collection and statistical analysis was performed by two independent researchers. Lead aVR was evaluated in the context of predicting left main stenosis (LMS) and proximal LAD occlusion. In the assessment of LMS, five studies were investigated with patients meeting NSTEMI criteria. ST segment elevation was measured 60ms beyond the J point in lead aVR. A cutoff of 0.05-0.1mV was established for J point elevation significance. Seven articles were analyzed looking at the predictive value of ST elevation of aVR in diagnosing a proximal LAD lesion above the first septal branch. ECGs were evaluated with evidence of ST elevation in anterior leads V2-V4.

Study Results

The summary of results from the five studies analyzing the ability of STE in lead aVR to predict LMS is shown in Table 6. There is inconsistent positive predictive values but relatively reliable negative predictive values. The high negative predictive value suggests that in the absence of STE in lead aVR, LMS is unlikely the culprit coronary artery.

Table 6: Lead aVR STE for Diagnosis of LMS in NSTEMI.[7]

Studies	Population	aVR STE (mV)	LMS Cases	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Barrabes et al	775	0.1	9	77	64	5	99
Henguss amee et al	26	0.05	5	80	76	44	94
Kosuge et al	310	0.05	60	78	86	57	95
Rostoff et al	134	0.05	44	68	73	56	83
Yu et al	91	0.1	9	89	84	38	99

Table 7 summarizes the data collected from the seven studies evaluating the ability of STE in aVR to predict proximal LAD lesions. With concurrent STE in V2-V4 (anterior STEMI), STE in lead aVR is shown to be beneficial in predicting proximal LAD involvement, with a high positive predictive value and specificity.

Table 7: Lead aVR STE for Diagnosis of proximal LAD lesion.[7]

Studies	Population	# Relevant Lesions	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Pooled Data	489	218	47	96	91	69

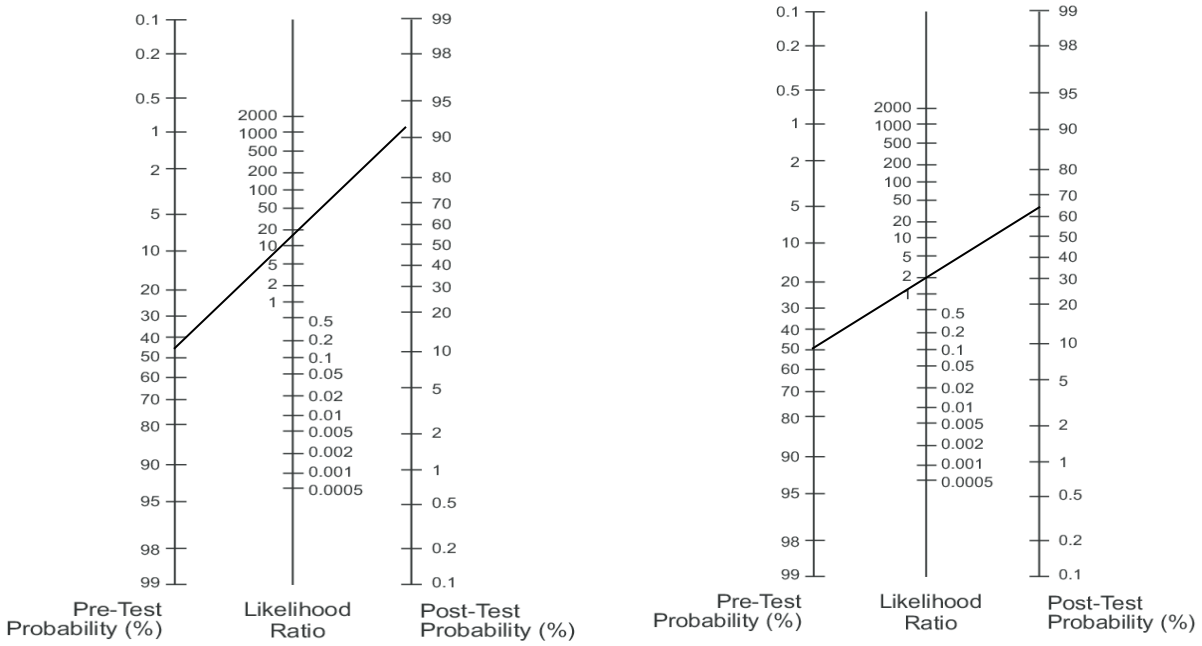
Study Critique

This review acknowledged aVR as an indicator of disease severity but aimed to determine if STE in aVR could determine location of the culprit lesion in an acute myocardial infarction. There were many limitations in this review. The studies used all had different cut offs as far as what was considered significant lesions (anywhere from 50 to 75%), as well as different cut-off values for what mV criteria was considered significant ST elevation. Lead aVR was also not analyzed in isolation from the presence of an anterior myocardial infarction (STE in leads V2-V4). Other potential limitations leading to bias were the inconsistency of population demographics, as well as non-consistent use of blinding in the studies. A positive of this literature review was its organization and consolidation of information in easy to follow tables. It presents the information in a way that the conclusion can clearly be drawn that lead aVR is not sensitive enough to be used in isolation, but rather should be looked in context with a coexisting STEMI.

Discussion

It is well established that earlier intervention with an acute MI with fibrinolytics or primary coronary intervention with ballooning/stenting produces better outcomes for patients.[1] A strict set of guidelines have been established as to what qualifies as a STEMI in order to recognize these patients with acute coronary artery occlusion and therefore emergently activate the cardiac catheterization lab without the need for biomarkers.[2] The purpose of this investigation was to determine if there was strong enough evidence to support the use of STE in lead aVR as a STEMI equivalent, suggesting LMCA or proximal LAD occlusion. Recognition of these patients with significant LMCA/LAD disease should be of high priority due to the large amount of myocardium they supply and subsequent ischemic consequences.

According to table 7, the sensitivity is 47% and specificity is 96% for lead aVR STE for diagnosis of proximal LAD lesion. The calculated positive likelihood ratio is 11.75 vs the negative likelihood ratio is 0.55. According to the nomogram, when a patient has a positive test for lead aVR STE, there is a 91% that proximal LAD lesion is involved. On the other hand, when there is a negative test result for lead aVR STE, there is a 69% that the proximal LAD is not involved. Since both PPV and NPV values are high, it indicates that ECG is a powerful assessment tool to determine whether proximal LAD is involved in lead aVR STE.



While lead aVR is often referred to as the "forgotten lead," it has gained popularity over the last 10 years. The 2013 ACC/AHA STEMI guidelines even incorporate lead aVR in the decision to administer fibrinolytics. They state that if there is ST elevation in lead aVR with associated ST depression, fibrinolytics are indicated.[2] However, there is debate on the evidence used to formulate this statement. The debate stems on how to define coronary artery "occlusion." While some assume that the word occlusion means there is complete blockage of the left main, some studies reference any stenosis greater than 50%. At 50% stenosis, fibrinolytics and PCI may not be indicated. It is therefore suggested that ST elevation in lead aVR should signify left main insufficiency rather than occlusion, and not be classified as a STEMI equivalent.[8] The three studies analyzed in this investigation support these suggestions. Yan et al reported that there was an increased mortality rate in hospital and at 6 months follow up in individuals with STE in lead aVR greater than 1mm. It also showed the predictive value of STE to show significant left main

stenosis or triple vessel disease. However, these results proved to be insignificant when aVR was viewed in isolation using a regression analysis model.[5] Gao et al was a meta-analysis that concluded that there was a significant increase in 30-day mortality in patients with STE in lead aVR greater than 1mm, but no conclusion was drawn about its predictive ability in isolation.[6] Kuhl et al concluded that in the absence of STE in lead aVR, left main stenosis was statistically unlikely. However, with highly variable positive predictive values, the authors are unable to make a recommendation on the diagnostic ability of lead aVR.[7]

Conclusion

There is evidence to suggest that ST elevation in lead aVR is associated with higher mortality in the presence of a recognized STEMI, as well as an association with subendocardial ischemia in a patient with an NSTEMI and/or diffuse ST depression. It is also suggested that in the setting of a patient with ACS and STE in lead aVR, the culprit lesion is not limited to the left main coronary artery, but rather may also indicate the LAD or involvement of all three main coronary arteries (triple vessel disease). The clinical use of lead aVR in isolation as a STEMI equivalent remains unclear. It is therefore concluded that without further studies to evaluate its prognostic ability, there is not strong enough evidence to support STE in lead aVR as a STEMI equivalent, but rather a potential marker of disease severity.

Acknowledgements

We would like to thank Dr. Erika Kancler, Carolyn Schubert, James Madison University's Physician Assistant Program staff, and James Madison University's Writing Center for the time and assistance in completing our Capstone Project - from the brainstorming of ideas to the final poster.

References

1. IJkema BB, Bonnier JJ, Schoors D, Schalij MJ, Swenne CA. Role of the ECG in initial acute coronary syndrome triage: Primary PCI regardless presence of ST elevation or of non-ST elevation. *Neth Heart J*. 2014;22(11):484-490. doi: 10.1007/s12471-014-0598-9 [doi].
2. Knotts RJ, Wilson JM, Kim E, Huang HD, Birnbaum Y. Diffuse ST depression with ST elevation in aVR: Is this pattern specific for global ischemia due to left main coronary artery disease? *Journal of Electrocardiology*. 2013;46(3):240-248. doi: https://doi.org/10.1016/j.jelectrocard.2012.12.016.
3. Kossaify A. ST segment elevation in aVR: Clinical significance in acute coronary syndrome. *Clinical Medicine Insights: Case Reports*. 2013;6:CCRep.S11261. https://doi.org/10.4137/CCRep.S11261. doi: 10.4137/CCRep.S11261.
4. Kühl JT, Berg RMG. Utility of lead aVR for identifying the culprit lesion in acute myocardial infarction. *Annals of Noninvasive Electrocardiology*. 2009;14(3):219-225. doi: 10.1111/j.1542-474X.2009.00300.x.
5. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: A report of the american college of cardiology Foundation/American heart association task force on practice

guidelines. *Journal of the American College of Cardiology*. 2013;61(4):e78-e140. doi: https://doi.org/10.1016/j.jacc.2012.11.019 ".

6. Wong CK, Gao W, Stewart RA, et al. aVR ST elevation: An important but neglected sign in ST elevation acute myocardial infarction. *Eur Heart J*. 2010;31(15):1845-1853. doi: 10.1093/eurheartj/ehq161 [doi].
7. Yamaji H, Iwasaki K, Kusachi S, et al. Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography: ST segment elevation in lead aVR with less ST segment elevation in lead V1. *Journal of the American College of Cardiology*. 2001;38(5):1348-1354. doi: https://doi.org/10.1016/S0735-1097(01)01563-7.
8. Yan AT, Yan RT, Kennelly BM, et al. Relationship of ST elevation in lead aVR with angiographic findings and outcome in non–ST elevation acute coronary syndromes. *American Heart Journal*. 2007;154(1):71-78. doi: https://doi.org/10.1016/j.ahj.2007.03.037.