

ORIGINAL ARTICLES

Utility of Lead aVR for Identifying the Culprit Lesion in Acute Myocardial Infarction

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Background: Lead aVR is a neglected, however, potentially useful tool in electrocardiography. Our aim was to evaluate its value in clinical practice, by reviewing existing literature regarding its utility for identifying the culprit lesion in acute myocardial infarction (AMI).

Methods: Based on a systematic search strategy, 16 studies were assessed with the intent to pool data; diagnostic test rates were calculated as key results.

Results: Five studies investigated if ST-segment elevation (STE) in aVR is valuable for the diagnosis of left main stem stenosis (LMS) in non-ST-segment AMI (NSTEMI). The studies were too heterogeneous to pool, but the individual studies all showed that STE in aVR has a high negative predictive value (NPV) for LMS. Six studies evaluated if STE in aVR is valuable for distinguishing proximal from distal lesions in the left anterior descending artery (LAD) in anterior ST-segment elevation AMI (STEMI). Pooled data showed a sensitivity of 47%, a specificity of 96%, a positive predictive value (PPV) of 91% and a NPV of 69%. Five studies examined if ST-segment depression (STD) in lead aVR is valuable for discerning lesions in the circumflex artery from those in the right coronary artery in inferior STEMI. Pooled data showed a sensitivity of 37%, a specificity of 86%, a PPV of 42%, and an NPV of 83%.

Conclusion: The absence of aVR STE appears to exclude LMS as the underlying cause in NSTEMI; in the context of anterior STEMI, its presence indicates a culprit lesion in the proximal segment of LAD.

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ECG; aVR; culprit lesion; myocardial infarction; ischemia

INTRODUCTION

The electrocardiogram (ECG) predicts the location and size of the injured zone during acute myocardial infarction (AMI).¹ Specifically, ST-segment deviations reflect myocardial ischemia, with important information about first-line treatment and severity of disease. Over the years many criteria have been developed to strengthen the predictive value of the ECG. However, although the augmented unipolar limb lead aVR was developed more than 60 years ago in order to obtain specific information from the right upper side of the heart,² it remains a largely neglected lead for identifying the culprit lesion in clinical practice.^{3–5} This

is somewhat surprising, as lead aVR may contain important information regarding the ischemic myocardium and the location of the culprit lesion. The right upper side of the heart, that is, the out-flow tract of the right ventricle and the basal part of the interventricular septum, is supplied by the main stem of the left coronary artery (LM) and/or branches from the proximal parts of the left anterior descending artery (LAD); hence culprit lesions in these coronary segments cause ST-segment deviations in lead aVR. Due to the dominance of the basal ventricular mass, this should lead to ST-segment elevation (STE) in lead aVR, as the ST-segment vector in the frontal plane points in a superior direction⁶ (Fig. 1).

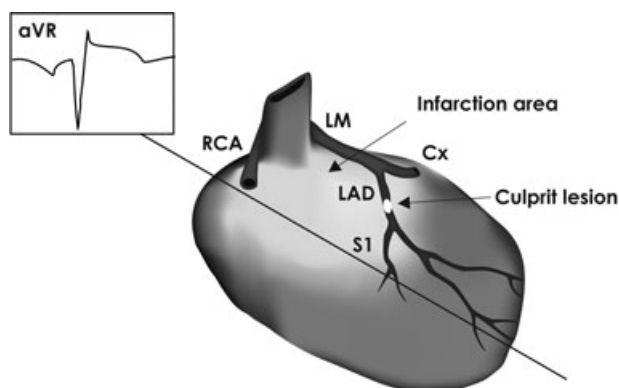


Figure 1. Culprit lesions in the main stem of the left coronary artery or the proximal part of the left anterior descending artery cause ischemia in the right upper part of the heart, that is the outflow tract of the right ventricle and the basal part of the interventricular septum. Due to the dominance of the basal ventricular mass, this may lead to ST-segment elevation in lead aVR. Abbreviations: Cx = left circumflex artery; LAD = left anterior descending artery; LM = main stem of left coronary artery; RCA = right coronary artery.

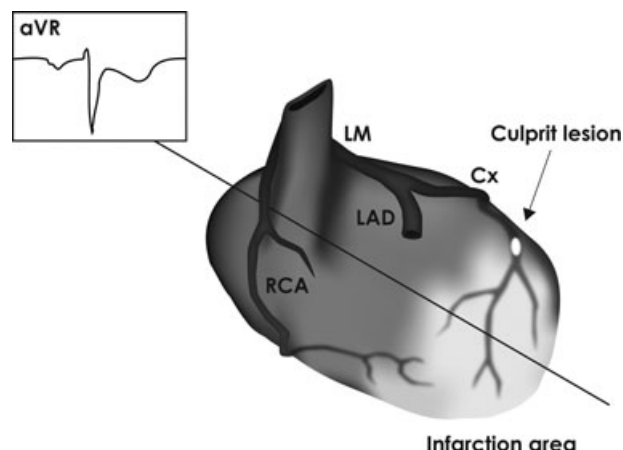


Figure 2. Culprit lesions in the left circumflex artery cause ischemia in the lateral and apical parts of the left ventricle. Hence, the ischemic area is more lateralized than that caused by culprit lesions in the right coronary artery. Hence, ischemia in this region may cause mirroring ST-segment deviations in lead aVR. Abbreviations: Cx = left circumflex artery; LAD = left anterior descending artery; LM = main stem of left coronary artery; RCA = right coronary artery; S1 = first septal branch of the left anterior descending artery.

Moreover, lead aVR conveys reciprocal information from the lateral and apical portions of the left ventricle; hence, ischemia in this region may cause mirroring ST-segment deviations in lead aVR. The myocardium in this region normally receives a bilateral blood supply from the left circumflex artery (Cx) and the right coronary artery (RCA); in this context lead aVR may prove useful to discern inferior AMIs caused by a culprit lesion in Cx from those caused by RCA lesions,⁷ since the area supplied by Cx is typically located more leftward in the frontal plane than that supplied by RCA (Fig. 2).

The aim of the present article was to elucidate the utility of lead aVR in clinical practice, by systematically reviewing the existing literature regarding its implications for identifying the culprit lesion in AMI. Hence, three specific hypotheses were explored: (1) STE in aVR is valuable for the electrocardiographic diagnosis of left main stem stenosis (LMS) in non-ST-segment elevation AMI (NSTEMI), (2) STE in aVR is valuable for the electrocardiographic distinction between proximal and distal LAD lesions as the cause of anterior ST-segment elevation AMI (STEMI), and (3) aVR ST-segment depression (STD) is valuable for the electrocardiographic distinction between Cx and RCA lesions as the cause of inferior STEMI.

METHODS

Literature Search and Data Sources

We searched MEDLINE and Google Scholar. The terms *aVR*, *ischemia*, *myocardial infarction*, and *ST segment elevation and ST segment depression* were applied in our search strategy. The search was restricted to human studies. We searched reference lists of the included papers, and obtained all relevant papers and review articles. There were no language restrictions.

Relevant papers were included if they held results on the role of lead aVR for identifying the culprit lesion in AMI, and explicitly used relevant inclusion criteria; typical chest pain, clinically significant ST deviations, appropriate alterations in coronary enzyme levels, and definite culprit lesion diagnosis by means of coronary arteriography. Moreover, patients with left bundle branch block, electrocardiographic signs of left ventricular hypertrophy according to the Sokolow-Lyon criteria, as well as a previous history of myocardial infarction or cardiac surgery were all excluded.

We obtained information regarding time of ECG in relation to clinical symptoms and coronary arteriography. Furthermore, we registered how

ST-deviations were assessed, as well as which criteria were used for the angiographic diagnosis of the culprit lesion. Studies with more than 25 patients were included, whereas results from smaller studies were only mentioned.

Data Analysis

Data from the various studies were extracted and pooled in 2×2 tables. Fisher's exact test was used to establish significant differences between groups. Diagnostic test rates were calculated as the key results. Two independent observers performed the data extraction and analysis. $P < 0.05$ was considered statistically significant.

RESULTS

Lead aVR in AMI Caused by Left Main Stem Stenosis

We found five relevant papers regarding the implications of lead aVR in AMI caused by LMS. All studies investigated consecutive patients who met the criteria for NSTEMI, two of which specifically assessed patients with a first myocardial infarction,^{8,9} whereas the remaining three studies included patients with acute coronary syndrome, both with and without AMI.^{10–12} In three studies an STE of 0.05 mV was regarded relevant,^{10–12} in one study 0.1 mV was considered,⁹ whereas one study divided patients according to the degree of aVR STE,⁸ hence aVR STEs of 0.05–0.1 mV were considered separately from aVR STEs above 0.1 mV. Lead aVR STE was measured 60 ms after the J-point in three studies,^{9,11,12} 80 ms in one study⁸ and 20 ms in another.¹⁰ Coronary arteriography was performed at different times after the ischemic

event in the various studies, ranging from days^{11,12} to months.^{8,9} Furthermore, the arteriographic definitions of LMS differed between studies; a stenosis of more than 50%⁸ was considered significant in one study, whereas 75% was considered significant in two others,^{11,12} one of which also included the presence of local dissection or thrombosis.¹¹ Two studies did not report any specific criteria.^{9,10} In one study, patients with LM and three-vessel disease were pooled and no differential analyses between the groups were performed.¹² ECG interpreters were blinded in four of the studies,^{8,9,11,12} and none of the studies reported blinding of the angiographer.

The prevalence of LMS varied immensely among the studies (Table 1), probably due to the dissimilar study populations, ECG criteria and culprit definitions applied. Thus, we were unable to pool the data. Nonetheless, it appears consistent that aVR STE yields high negative predictive values, whereas the positive predictive values show large disparities (Table 1). Of the five studies, the findings by Barrabés et al. appear to be the most reliable and accurate when evaluating the implications of lead aVR for identifying the LM as the culprit lesion. This study specifically assesses a population of 775 patients with a first myocardial infarction, of which 435 underwent coronary arteriography; angiographic findings were evaluated in relation to aVR STE between 0.05 and 0.1 mV and aVR STE equal to or above 0.1 mV, respectively. LM was identified as the culprit artery in nine patients, of whom two displayed no aVR STE, none had an aVR STE between 0.05 and 0.1 mV, and seven displayed an aVR STE equal to or above 0.1 mV. When an aVR STE of 0.1 mV is considered significant, a NPV of 99% is attained (Table 1) the highest of all the studies.⁸

Table 1. Lead aVR STE for the Diagnosis of LMS in NSTEMI

	Study	Population	aVR STE (mV)	LMS Cases	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
LMS	Barrabés et al. ⁸	775	0.1	9	77	64	5	99
	Hengussamee et al. ¹¹	26 ^a	0.05	5	80	76	44	94
	Kosuge et al. ¹²	310 ^a	0.05	60 ^b	78	86	57	95
	Rostoff et al. ⁴²	134 ^a	0.05	44	68	73	56	83
	Yu et al. ⁹	91	0.1	9	89	84	38	99

^aStudy populations included patients with acute coronary syndrome, both with and without AMI.

^bPatients with LMS and three-vessel disease were pooled in this study and no differential analyses between groups were performed.

LMS = left main stenosis; NPV = negative predictive value; PPV = positive predictive value.

Lead aVR in Anterior STEMI

We found seven relevant articles,^{13–19} six of which specifically investigated the hypothesis that aVR STE is predictive of proximal LAD lesions,^{13–18} that is before the departure of the first septal branch (S1) from LAD. Engelen was the first to suggest and test different ECG-criteria for discriminating between proximal and distal lesions.¹³ We found that this article had inspired five other studies to test the same hypothesis.^{14–18} All studies included patients with signs of anterior STEMI with STE in leads V₂–V₄. ECGs were evaluated just prior to coronary intervention and if more than one ECG was obtained, the one with the most significant changes was used. Three studies measured STE at the J-point, in the remaining two studies STE was assessed 40¹⁸ and 80¹⁴ ms after the J-point, respectively. Two studies considered any STE in lead aVR relevant,^{13,17} whereas the remaining three studies only considered an STE above 0.05 mV.

The criteria for determining culprit lesion differed between studies, both with regard to time from the insult to coronary arteriography, as well as to the criteria that were used to identify the culprit lesion. Hence, coronary arteriography was performed at various time points, ranging from the acute phase^{13,14,19} and up to 3 weeks after.¹⁸ The culprit lesion was exclusively defined by means of the degree of reduction in diameter of the vessel in two studies,^{15,17} whereas the remaining studies included signs of a residual thrombus, an ulcerated plaque, and local dissection.^{13,14,18}

The pooled data from the six similar studies adds up to 489 patients with anterior STEMI. Of these, 218 (45%) patients had a proximally located culprit lesion, of which 102 had STE in lead aVR, whereas only 10 patients with distally located culprit lesion displayed aVR STE. Fisher's exact test showed significant difference between the groups (2-tailed $P < 0.0001$). The combined studies prove high speci-

ficity and positive predictive value of STE in lead aVR for proximality of the culprit lesion (Table 2). Diagnostic parameters were not different between studies that regarded any aVR STE and those that only regarded STE above 0.05 mV.

Lead aVR in Inferior STEMI

Nair and Glancy were the first to suggest that STD in lead aVR is suggestive of Cx rather than RCA involvement in inferior STEMI.²⁰ We found three additional original articles^{21–23} and one abstract²⁴ that further addressed this hypothesis. Two studies excluded patients if occlusions were found in both Cx and RCA.^{21,23} Three studies assessed STD in aVR 60 ms after the J-point, whereas the remaining study measured STD 20 ms after the J-point.²² All studies except for one explicated 0.1 mV to be the relevant STD.²¹

The description of time of coronary arteriography from the clinical signs of AMI ranged from "median of 3 days after admission"²⁵ to "within 7 days."²⁰ One study did not report anything on the matter.²¹ The culprit lesion was assessed through Thrombolysis in Myocardial Infarction (TIMI) grading in one study,²² whereas the others defined it by the degree of reduction in diameter of the vessel and included signs of ulcerated plaque and residual thrombus.^{20,21,23} Two studies described blinding of both coronary arteriography and ECG observers.^{22,23}

The pooled data from the four original articles added up to 398 patients with inferior STEMI of which Cx was the culprit artery in 89 patients (22%); of these 31 showed STD in aVR. 50 out of 309 RCA-related STEMI showed STE in aVR. Fisher's exact test showed significant difference between the groups (2-tailed $P < 0.0001$). In general, STD in lead aVR appears to yield relatively high specificities and negative predictive values, thus indicating that its absence excludes Cx as the culprit (Table 2).

Table 2. The Utility of Lead aVR for Culprit Diagnostics in STEMI

	Studies	Population	# Relevant Lesion	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Prox. LAD lesions (aVR STE)	Pooled data ^{13–18}	489	218	47	96	91	69
Cx lesions (aVR STD)	Pooled data ^{20–23}	398	89	35	84	38	82

Pooled data for identifying proximal LAD culprit lesions in anterior STEMI (lead aVR STE) and Cx lesions in inferior STEMI (lead aVR STD).

LMS = left main stenosis; NPV = negative predictive value; PPV = positive predictive value; STD = ST-segment depression; STE = ST-segment elevation.

DISCUSSION

It is well established that ST-deviations in lead aVR predict severity of disease,^{8,22,25} infarction volume,^{22,25–27} and prognosis⁸ in patients with AMI. Furthermore, as reviewed in the present article, lead aVR may also be useful for determining the location of the culprit lesion in patients with AMI caused by LMS, LAD lesions, and RCA/Cx lesions.

Acute coronary syndrome due to LMS exhibits higher mortality rates than any other coronary lesion. LMS is a common cause of ischemic heart disease and is found in 3–5% of patients undergoing coronary arteriography for ischemic chest pain, congestive heart failure, and cardiogenic shock.⁸ These patients require specific therapy, including acute coronary artery bypass grafting (CABG).^{28,29} As treatment with platelet inhibitors is routinely used in patients with acute coronary syndrome, but contraindicated prior to CABG, this should not be administered in the case of LMS.³⁰ Accordingly, early recognition or exclusion of LMS in patients with NSTEMI is crucial in order to identify high-risk patients and to implement the optimal therapeutic strategy. In this respect, an aVR STE of 0.1 mV may prove useful, as it is more frequent and pronounced in patients with LMS when compared to other coronary lesions.³⁰ As reviewed in this article, the available evidence proposes that STE in aVR is useful when suspecting LMS in patients with NSTEMI, in the sense that its absence largely excludes LMS as culprit artery; although this notion is based on data from a limited amount of patients, the findings do imply that the clinician should be particularly cautious with regards to platelet inhibitor treatment in patients with acute coronary syndrome when aVR STE is present.

None of the studies reviewed in this article addressed aVR STE in the context of other elec-

trocardiographic signs of LMS. Although initial attempts to identify criteria for the electrocardiographic characterization of LMS failed,³¹ studies conducted through the 1980s and early 1990s conceptualized the presence of diffuse STD in inferior and anterolateral leads in patients with LMS.^{32–34} Thus, the presence of these electrocardiographic signs along with aVR STE may be highly predictive of LMS in patients with acute coronary syndrome, although this needs to be explored further in future studies. Furthermore, it may prove useful to compare STE in leads aVR and V₁, as it has been suggested that STE aVR > V₁ predicts LMS.⁶

The studies that are reviewed in this report uniformly point toward STE in aVR as a useful tool for identifying proximal LAD lesions in the context of other signs of anterior STEMI, that is STE in V₂–V₄, reflected by high specificities and positive predictive values. Discrimination between proximal and distal LAD lesions is important, as the size of the myocardial infarction and prognosis are worsened by the proximity of the occlusion.^{6,35} Conversely, the aVR criterion is not very sensitive, and it has therefore been suggested that it should be linked with other criteria. Hence, Eskola et al. recently tested the combined criteria of “any STE in aVR” and an STE of more than 0.05 mV in aVL. This strengthened the sensitivity, but weakened the specificity and positive predictive value equally.¹⁹

Inferior STEMI amounts to 40–50% of all AMIs and is mostly caused by RCA lesions.^{35–37} As the clinical outcome and treatment differ between inferior STEMI caused by RCA and Cx lesions,^{38–41} it is of interest to discriminate between them at an early stage. Accordingly, a number of electrocardiographic criteria have been developed (Table 3). The studies reviewed in this report show relatively high specificities and negative predictive

Table 3. ECG Criteria for Discerning Cx from RCA Lesions in Inferior STEMI

Culprit Lesion	ECG Criteria	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Cx	aVR STE ^{20–23}	35	84	38	82
	V ₁ –V ₂ STD ^{20,21,23}	75	72	45	91
	I STE ^{20,23,43}	33	99	94	85
RCA	STD aVL > I ^{23,41}	65	92	97	41
	STE III > II ^{20,21,23,41,43,44}	85	72	91	58

Pooled data from studies that examined aVR ST-segment depression along with other electrocardiographic criteria in the context of inferior STEMI.

Cx = left circumflex artery; LMS = left main stenosis; NPV = negative predicative value; PPV = positive predicative value; RCA = right coronary artery; STD = ST-segment depression; STE = ST-segment elevation.

values of aVR STD for Cx lesions in inferior STEMI. Although Nair and Glancy initially suggested that this aVR criterion is highly sensitive,²⁰ it is impossible to establish on basis of the data at hand. When compared to other criteria designed to differentiate between RCA and Cx lesions (Table 3), lead aVR seems to have very little to add. Although aVR STD does not seem to be of much use for identifying the culprit lesion in inferior STEMI, it may nevertheless prove useful, as it has been demonstrated to predict the size of the myocardial infarction.^{26,27}

There are several limitations to this study, as there are a number of potential sources of bias and uncertainty. In fact, the LMS studies were so heterogeneous that pooling of the data was not meaningful, although all studies fulfilled our inclusion criteria. Very few articles explicitly mentioned the use of double blinding. Furthermore, the studies varied much in both angiographic and electrocardiographic definitions.

In conclusion, lead aVR may be useful in clinical practice when assessing patients with acute coronary syndrome as the absence of aVR STE appears to exclude LMS as the underlying cause in NSTEMI, whereas its presence in the context of anterior STEMI indicates a culprit lesion in the LAD, proximal to the first septal branch.

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