



CLINICAL UTILITY OF AVR – THE NEGLECTED ECG LEAD: HIDDEN CLUES IN ACUTE INFERIOR WALL MYOCARDIAL INFARCTION

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ABSTRACT Lead aVR, often regarded as a neglected ECG lead, can provide additional clues to the extent and distribution of myocardial ischaemia in acute coronary syndromes. We present a 47-year-old man with diabetes mellitus and hypertension who presented with acute chest pain associated with backache and shoulder pain. Electrocardiography showed ST-segment elevation predominantly in the inferior leads, greater in lead II than III, with associated lateral-lead changes and ST-segment depression in aVR. The ECG pattern suggested an acute inferior myocardial infarction, with features raising the possibility of left circumflex coronary artery involvement. However, coronary angiography demonstrated an occluded right coronary artery, which was subsequently revascularised. This apparent discordance between electrocardiographic localisation and angiographic findings highlights the importance of careful interpretation of aVR in conjunction with the complete 12-lead ECG. Rather than serving as an isolated marker of culprit-vessel localisation, changes in aVR may provide an additional perspective on the underlying ischaemic pattern and contribute to clinical decision-making in acute myocardial infarction. This case underscores the diagnostic value of systematically evaluating aVR while recognising the limitations of ECG-based prediction of the culprit coronary artery.

KEYWORDS : Lead aVR; Electrocardiogram; Inferior Myocardial Infarction; Acute Coronary Syndrome; Coronary Angiography Culprit Vessel; ST-segment Elevation; Right Coronary Artery; Left Circumflex Artery

INTRODUCTION

The 12-lead electrocardiogram remains one of the most important initial investigations in patients presenting with suspected acute coronary syndrome. While considerable attention is routinely given to the precordial and inferior leads, lead aVR is frequently neglected during ECG interpretation.

Lead aVR provides information regarding electrical activity directed toward the right shoulder and may demonstrate reciprocal ST-segment changes in a variety of ischemic conditions. Widespread ST-segment depression accompanied by ST-segment elevation in aVR has been described in association with diffuse subendocardial ischemia and may be seen in patients with significant coronary artery disease. In acute inferior wall myocardial infarction, careful evaluation of the pattern of ST-segment changes, including changes in aVR, may provide additional information regarding the likely culprit coronary artery. Previous literature has examined the relationship between ST-segment changes in aVR and coronary anatomy, including differentiation between right coronary artery and left circumflex artery-related infarction.

We present a case of acute inferior wall myocardial infarction in which detailed ECG interpretation, including assessment of lead aVR, provided additional diagnostic clues regarding the coronary territory involved.

Case Study

A 47-year-old man with diabetes mellitus and hypertension presented to the emergency department with acute-onset chest pain associated with backache and shoulder pain. His vital signs were stable at presentation. The initial 12-lead ECG showed sinus rhythm with ST-segment elevation in the inferior leads, more prominent in lead II than III, with associated ST-segment changes in leads V5 and V6 and ST-segment depression in aVR.

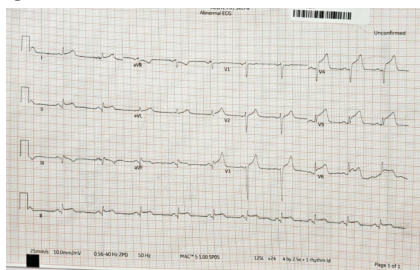


Figure 1: ECG of this Patient with Features Suggestive of LCX Territory ACS

The ECG findings were consistent with an acute inferior wall myocardial infarction. The pattern of ST-segment changes, particularly the inferior-to-lateral distribution and changes in aVR, raised the possibility of left circumflex coronary artery involvement. The ECG therefore provided an important initial clue for anatomical localisation of the infarct.

However, coronary angiography revealed an occluded right coronary artery (RCA). The RCA was subsequently revascularised, with post-intervention angiography demonstrating restoration of coronary flow.

This case highlights an important diagnostic paradox: the ECG pattern suggested one coronary territory, while angiography demonstrated another as the culprit vessel. It illustrates that changes in aVR can provide valuable additional information during ECG interpretation, but their significance must be considered alongside the complete pattern of ST-segment changes rather than used in isolation to identify the culprit artery.

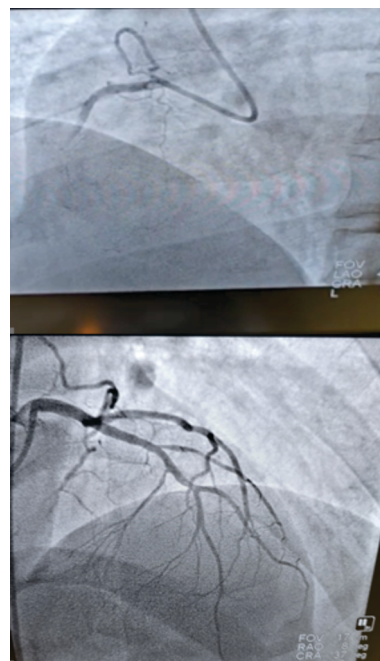
CAG



Figure 2 (a-c): Diagnostic CAG Showing Occluded RCA with Patent LAD and Post Revascularization of RCA

DISCUSSION

Lead aVR, although frequently given less attention during routine ECG interpretation, can provide useful information about the direction and extent of myocardial ischaemia. Because of its electrical orientation, ST-segment changes in aVR are often interpreted in relation to reciprocal changes occurring elsewhere on the 12-lead ECG. In particular, ST-segment elevation in aVR accompanying widespread ST-segment depression is classically described in the setting of diffuse subendocardial ischaemia and may be associated with significant left-main or multivessel coronary disease. The presentation also highlights the recognised distinction between reciprocal aVR changes in diffuse subendocardial ischaemia and primary ST-segment elevation involving the basal septum.

In the present case, the ECG demonstrated inferior ST-segment elevation, greater in lead II than III, with lateral-lead involvement and ST-segment depression in aVR. This pattern suggested an inferior myocardial infarction, with the distribution of changes raising the possibility of left circumflex coronary artery involvement. However, coronary angiography demonstrated RCA occlusion, which was subsequently revascularised.

This ECG–angiographic discordance highlights an important limitation of surface ECG-based culprit-vessel localisation. Although the direction and distribution of ST-segment changes can provide valuable anatomical clues, their relationship with the culprit coronary artery is influenced by factors including coronary anatomy, myocardial territory and the extent of ischaemia. Accordingly, changes in aVR should be interpreted as part of the overall electrocardiographic pattern rather than as an isolated determinant of the culprit vessel.

Previous studies have evaluated ST-segment depression in aVR as a potential clue to culprit-vessel anatomy in inferior STEMI. Although aVR depression has been associated with left circumflex involvement, its sensitivity is limited and it may also occur in right coronary artery infarction. [2] Several ECG criteria and algorithms have subsequently been evaluated for differentiating RCA from LCX occlusion, with variable diagnostic performance. [3-5] The present case illustrates the importance of maintaining this finding within its appropriate clinical context: an ECG pattern may suggest a particular coronary territory, but angiography may reveal a different culprit vessel.

Thus, the value of aVR lies not in providing a definitive anatomical diagnosis, but in adding another layer to systematic ECG interpretation. Its careful assessment can refine the initial diagnostic hypothesis while recognising that ECG-based localisation remains probabilistic and should be correlated with the clinical presentation and, when indicated, coronary angiography.

Clinical Significance

aVR—the neglected lead—may provide important hidden clues in acute myocardial infarction.

1. Assess aVR as part of every systematic 12-lead ECG interpretation.
2. Interpret aVR in conjunction with the overall ST-segment pattern, not in isolation.
3. In inferior STEMI, aVR may provide additional clues to the suspected culprit coronary territory.
4. ECG-based culprit-vessel localisation remains suggestive rather than definitive, as illustrated by the discordance between the ECG pattern and angiographic RCA occlusion in this case.
5. Correlate ECG findings with coronary angiography when invasive assessment is clinically indicated.

CONCLUSION

Lead aVR, the often-neglected ECG lead, can provide additional clues to the distribution of myocardial ischaemia when interpreted within the complete 12-lead ECG. In this case, the ECG pattern suggested possible left circumflex involvement, whereas coronary angiography demonstrated RCA occlusion, highlighting the limitations of ECG-based culprit-vessel localisation.

Careful interpretation of aVR can enrich ECG-based clinical reasoning, but its findings should be integrated with the overall ECG pattern, clinical context and, when indicated, coronary angiography.

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