LETTER TO THE EDITOR

Deblurring the significance of ST-elevation in aVL in anterolateral myocardial infarction

To the Editor,

I read with great interest the article entitled "The significance of ST-elevation in aVL in anterolateral myocardial infarction: An assessment by cardiac magnetic resonance imaging" by Allencherril et al. (2018). I want to thank the authors for the influential investigation of the importance of electrocardiographic changes in lead aVL in patients with first acute anterolateral myocardial infarction (MI).

Acute anterolateral MI is basically diagnosed with the electrocardiogram by ST segment elevation (STE) in D1, aVL, and the precordial leads. In acute anterolateral MI, ST segment changes in reciprocal derivations are the milestone of several investigations in the literature (Sasaki, Yotsukura, Sakata, Yoshino, & Ishikawa, 2001; Birnbaum et al., 1993). Several hypotheses have aimed to explain the ST segment changes with a practical theory. Benign reciprocal alterations are merely charged with underlying mechanism by Little, Rogers, and Sodums (1984). On the other hand, Schuster et al. have defended the occurrence of ST segment depression secondary to ischemia of those adjacent areas due to lack of collateral blood flow from the occluded coronary artery (Schuster & Bulkley, 1980). The well-accepted theory has been put forward by Sasaki et al. which is a combination of electrical alterations and remote myocardial ischemia as a pathophysiological mechanism of ST segment depression in acute MI (Sasaki et al., 2001). This is once again validated in patients with acute anterior MI treated with primary percutaneous intervention (Hayiroğlu et al., 2018). Nevertheless, another important contribution is very valuable with regard to electrocardiographic changes in aVL during MI by the help of cardiac magnetic resonance (CMR) imaging. For this reason, I am very grateful to Allencherril et al. for their considerable contribution. CMR imaging has even been recommended as an alternative imaging method to echocardiography because of its precise results (Kim et al., 2000). Interpretation of the ECG changes in aVL with the aid of CMR brings this study to almost perfection. On the other hand, I have minor comments and questions about some issues in this trial. The data were limited with regard to occlusion site of the left anterior descending (LAD) artery, multivessel disease especially comprising right coronary artery and the wrapping feature of the LAD artery. The distal right coronary artery lesions may either have a role in ST segment changes in lead aVL or enhancing myocardium at risk in apical inferior segment. Moreover, the wrapping feature of the LAD artery has a great influence on the myocardium at risk. In other words, patients with STE in lead aVL may mostly include patients with proximally occluded nonwrapped LAD artery or patients without ST segment elevation in lead aVL may mostly include patients with distally occluded wrapped LAD artery.

Therefore, we will probably make a mistake to conclude patients with anterior MI and concomitant STE in aVL have less myocardium at risk in the apical inferior segment or more in the basal anterior segment. Therefore, the additional data will greatly increase the value of the article. Nevertheless, this trial is enlightening work about the significance of STE in aVL.

CONFLICT OF INTEREST

None declared.

ORCID

Mert İlker Hayiroğlu <http://orcid.org/0000-0001-6515-7349>

Mert İlker Hayiroğlu MD

Department of Cardiology, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey

Correspondence

Mert İlker Hayiroğlu, Department of Cardiology, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey.
Email: mertilkerh@yahoo.com

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