VIA MEDICA

EDITORIAL

Folia Cardiol.
2006, Vol. 13, No. 7, pp. 543–546

ISSN 1507–4145

# Can we use the electrocardiogram to refine the estimation of the size and location of ischemia in anterior ST elevation myocardial infarction?

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## Introduction

Based on the results of several clinical randomized trials the current guidelines recommend that every patient with suggestive symptoms and ST elevation (> 0.2 mV ST elevation at the J-point in leads V1–V3 and > 0.1 mV in all other leads) should be immediately subjected to reperfusion therapy, preferentially by primary percutaneous intervention (PCI) [1, 2]. Many cardiologists developed an approach that since every patient with ST elevation is triaged for immediate heart catheterization; there is no need to obtain any other information from the electrocardiogram (ECG). However, in systems with more limited resources there is an increased need for improving our ability for early diagnosis and risk stratification in order to decide whether the patient will receive thrombolytic therapy or be referred for PCI. When this decision is made, the clinician can rely only on the clinical data (history and physical examination) and the presenting ECG.

Many studies have been published trying to correlate different parameters in the presenting ECG with coronary anatomy, ischemic area at risk, final infarct size, left ventricular function and/or prognosis in patients with ST elevation myocardial infarction (STEMI). For a recent review the readers are referred to [3].

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Received: 16.09.2006 Accepted: 2.10.2006

## Correlations with coronary anatomy

The left anterior descending coronary artery (LAD) is located above the anterior interventricular septum, supplying septal branches to the septum and diagonal branches to the anterolateral zones of the heart. However, there are considerable variations in the anatomy of the coronary arteries. The left anterior descending coronary artery can be short, not reaching the apex; intermediate size; or long, wrapping the cardiac apex and supplying various portions of the apical, distal lateral and distal inferoposterior segments. In addition, various portions of the anterior free wall of the right ventricle also get blood supply from LAD branches. The basal, mid and distal anterolateral and inferolateral segments may get blood supply from the diagonal branches of the LAD, marginal branches of the left circumflex artery or from a ramus intermedius branch. In patients with multivessel disease, the variations in regional blood supply can be even greater due to the presence of collaterals. Thus, the ECG findings in a patient with an acute occluded short LAD and proximal chronic occlusion of a right coronary artery with collaterals from the LAD to the right posterior descending artery actually should be similar to those of a patient with occlusion of a long wrapping LAD. It has to be remembered that the surface 12-lead ECG shows the summation of the electrical activity of the heart rather than reflecting the actual coronary anatomy.

We have also a problem with the "goal standard" of the coronary angiogram, especially in studies using angiograms of patients after thrombolytic therapy or predischarge angiograms, as most of the

older studies did. The site of the most severe coronary narrowing does not necessarily reflect the size and location of the ischemic bed. In many cases the plaque ruptures at bifurcation site. The occlusive thrombus may progress proximally and distally in the vessel, so during acute ischemia side branches could have been occluded and later on, after (partial) dissolution of the clot, the side branches appear patent. Moreover, coronary angiography is not sensitive for depicting small collateral vessels that may (at least partially) nourish the segments assumed to be supplied by the infarct related artery. Even visible collaterals may disappear shortly after recanalization of the infarct-related artery.

Nevertheless, studies have repeatedly shown several patterns. Ischemia of the lateral zones causes ST elevation in leads I and aVL [4, 5] and ST depression in the inferior leads [6]. This pattern is detected whether the first acute marginal branch of the left circumflex is involved (usually accompanied by ST depression in leads V2–V3); or when the first diagonal branch is involved (usually with concomitant ST elevation in the anterior precordial leads) [4]. However, when there is concomitant ischemia of the inferior segments (as may occur with proximal occlusion of a long wrapping LAD or a proximal occlusion of a dominant left circumflex artery), the injury vector of the inferior zone and that of the lateral zone cancel each other and no ST elevation is detected. Sasaki et al. [7] has shown that indeed, proximal occlusion of a short LAD causes ST elevation in leads I and aVL. However, proximal occlusion of a wrapping LAD is not associated with ST elevation in leads I and aVL or reciprocal ST depression in the inferior leads [7]. Thus, ST elevation in leads I and aVL or ST depression in the inferior leads is a specific, but insensitive sign for proximal occlusion of the LAD. The findings of Czechowska et al. [8], presented in the present issue, reflect these facts. Because lack of ST elevation in leads I and aVL in a patient with anterior STEMI may reflect either LAD occlusion distal to the first diagonal branch, or proximal occlusion of a long wrapping LAD, it is not expected that ST elevation in these leads would be associated with lower left ventricular ejection fraction or more segments with regional dysfunction.

Czechowska et al. [8] have confirmed our previous observation that ST elevation in lead V1 does not reflect LAD occlusion before the first septal branch, or lower ejection fraction [9], although Engelen et al suggested that ST elevation > 2.5 mm in lead V1 can predict LAD occlusion proximal to the first septal branch [10].

## Correlations with regional and global left ventricular systolic function

Final infarct size is determined by the size of the ischemia area at risk, the severity of ischemia (ischemic preconditioning, various drugs, residual flow by collaterals or partial or intermittent occlusion of the infarct related artery), the duration of ischemia and by events occurring during reperfusion (reperfusion injury) [3, 11]. The presenting ECG may reflect the size of the ischemic area at risk and/or the severity of ischemia and may show signs that irreversible damage has already occurred (presence of Q waves); however, it cannot predict the total time of ischemia (especially the time period between the ECG and the actual recanalization of the artery) and the severity of "reperfusion injury". Therefore, the expected correlation between the presenting ECG and final infarct size can be only fair, especially in patients undergoing reperfusion therapy.

Currently we have difficulties in demonstrating the size of the ischemic area at risk. Echocardiograms obtained before reperfusion may show the extent of ischemia. However, in most cases the quality of such echocardiogram is low due to the fact that in the acute stages of STEMI the patient may be dyspneic, cannot lay on his left side, and during off hours the examinations are done by less experienced technicians. Furthermore, complete study may be long and delay transfer to the catheterization laboratory, although echocardiogram can be done in patients receiving intravenous thrombolytic therapy. The echocardiograms in many of the studies were obtained the next day [12–14]. Although, due to the effect of stunning (delayed recovery of regional function after reperfusion), regional function at the time of the examination may still reflect the pattern during acute ischemia, it is less than ideal. Pretreatment technetium 99m-sestamibi perfusion defect size has been used by some investigators [15, 16]; however, they are technically difficult to obtain and there is no evidence that the correlation between the perfusion defect size and the actual size of the ischemic area at risk is good. Obviously, in patients with previous myocardial infarction (and assuming that ~30% of the infarcts are subclinical) the correlation cannot be good. Cardiac magnetic resonance imaging is a promising evolving technique that may help in the future depicting the ischemic area at risk [17]. However, this imaging modality is expensive and still time consuming, thus may delay the initiation of primary PCI. Czechowska et al. [8] used two parameters

for estimation of infarct size: echocardiogram and peak CK-MB. From the methods it is unclear whether the patients were treated with reperfusion therapy upon arrival. Although it is stated that all patients underwent coronary angiography, it is unclear whether it was part of primary PCI or later risk stratification. Accordingly, the timing of the echocardiogram is unclear. Most of them underwent it before angiography, whereas 3 underwent it within 12 hours of admission. Peak CK-MB is determined by the size of the ischemic area at risk, the severity and duration of ischemia and the quality of reperfusion. Prompt reperfusion causes a high early peak of CK-MB due to wash out from the previously ischemic zone with rapid decline in levels over time. In the past it has been suggested that the area under the curve of CK levels over the first 24 hours correlates with final infarct size. Although for convenience reasons many investigators have used the value of peak CK or peak CK-MB as a surrogate endpoint of infarct size, this methods has not been validated. Nevertheless, Czechowska et al. [8] have found correlation between the sum of ST elevation in all leads and peak CK-MB. Interestingly, Aldrich et al. [18] and Clemmensen et al. [19] reported that in anterior STEMI, the number of leads with ST elevation better correlated with the final QRS Sylvester score (a measure of final infarct size) in patients not subjected to reperfusion therapy. However, Christian et al. [16] reported no correlation between the sum of ST elevation or the number of leads with ST elevation and the perfusion defect size before reperfusion therapy. As previously mentioned, due to the effect of opposing vectors (for example inferior and lateral) and the fact that not all myocardial segments are equally represented by the 12-lead ECG, a simple linear correlation between the number of leads with ST elevation (or deviation), or the sum of ST elevation (or deviation) and infarct size cannot be expected. The sum of ST elevation is affected by both the extent (the size of the ischemic area at risk) and the severity of ischemia (in addition to other non-ischemic variable such as myocardial mass, the width of the chest wall and the distance between the electrode and the ischemic zone). This explains therefore, the fact the sum of ST elevation correlated with peak CK-MB, but probably not with left ventricular ejection fraction, or regional wall motion score (data not reported) [8]. The study by Sadanandan et al. [20] illustrates these non-linear relations. Patients with ST elevation in both the anterior and inferior leads have actually smaller apical infarction caused by a distal occlusion of a wrapping LAD, despite having greater sum of ST elevation in larger number of leads than patients with pure anterior ST elevation [20]. Moreover, as stated by Czechowska et al. [8], patients with anterior STEMI and ST depression, rather than elevation in leads V5–V6 had more extensive regional wall motion abnormalities and more severe regional dysfunction.

In conclusion, the correlations between the various ECG patterns and the size and localization of the ischemic area at risk in patients with anterior STEMI are complex. The situation with non-anterior STEMI may be even more complicated [3]. Further studies are needed to better understand the information that can be obtained from the presenting ECG of STEMI and how to use it for diagnosis, risk stratification and triage for therapy.

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