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Electrocardiogram dynamics for risk stratification in ST-segment elevation myocardial infarction—immediate and serially updated information on outcome

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Abstract Early and serially updated predictions of final infarct-size and clinical outcome - before, during and after reperfusion treatment of ST-elevation myocardial infarction might allow a more individualized treatment: High-risk patients with a predicted major loss of viable myocardium can be identified immediately or during therapy, at a stage when treatment may still be modified; and low-risk patients with predictions of small infarcts and good outcome already after standard primary reperfusion therapy can be identified and thereby avoid a possibly harmful intensified treatment. The necessary information for such predictions seem to be available from the standard 12-lead ECG and from ST-segment monitoring. Today this information, however, is not readily available in clinical practice. Automated algorithms need to be engineered for a broader use and for possibilities of a refined triage and thus for a more individualized strategy of reperfusion therapy.

Keywords: ECG; Myocardial infarction; Triage

Introduction

The clinical syndrome of ST-segment elevation myocardial infarction (STEMI) has literally been defined by specific changes observed on the electrocardiogram (ECG) after an acute coronary occlusion. In most of such clinical presentations, ST-segment elevation is associated with coronary occlusion that has persisted long enough to produce myocardial necrosis. We know, however, from the laboratory findings of Reimer et al[1] that myocardial necrosis spreads over time, like a wave front downstream from the occluded coronary artery. These findings have set the stage for a broad therapeutic concept: Urgent restoration of myocardial flow could interrupt the infarction process, salvage myocardium at risk, and result in an improvement of mechanical and electrical function, ultimately translating into a reduction of clinically evident outcomes such as heart failure and death. This concept has been supported by several reports showing that lives are saved[2] and infarctions are "aborted"[3] by timely reperfusion intervention.

The standard 12-lead ECG is the most frequently used tool for diagnosis and immediate decision support in the choice of treatment and care of a patient with chest pain suggestive of myocardial ischemia. In this situation, analyses of the first-contact ECG, either prehospitally or in the emergency department, are often very pragmatic, aimed at answering whether the patient has ST-segment elevation, ST-segment depression, or even a normal ST-segment level.

Typical ST-segment elevation or a presumably new left bundle branch block should, in clinical practice, result in consideration of the decision to urgently let the patient undergo reperfusion therapy. In the Western world, this would in many cases mean transportation to a catheter laboratory for an urgent percutaneous coronary intervention (PCI). This could take time, which might be costly with respect to "grams of necrotic myocardium," but it is generally accepted that the delay for such treatment may take up to 90 minutes from first contact, and data support that this probably is safe—at least for the majority.

In the hands of a dedicated ECG researcher, the very same first-contact ECG might reveal more information than is usually assessable in the clinical situation described above. Scores can be calculated from the ECG to estimate the area at risk for infarction, how far into the process of

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infarction the patient is at the particular time, how well protected the myocardium is to ischemia, or how severe the current ischemia really is.

Following the ECG development by either serial ECG recordings or even continuous monitoring will further increase assessable information on the evolution of ischemia and on the effect of any given therapy. By merging the data accessed by the different scores with data on ECG development, we would probably have a tool for a more individualized initial risk stratification and triage and a possibility of updating information on risk and on treatment efficacy when therapy has been initiated.

There is, however, (at least) one major issue to resolve before any of this can be tested and readily implemented clinically: The information "hidden" in the ECG must become available outside the hands of the dedicated ECG researcher.

The "STEMI weather forecast"

In an attempt to test the feasibility and possible value of a serially updated assessment of final infarct size, we recently reported a study[4] in which we used various scores from the first-contact 12-lead ECG together with patient-reported symptom delay time and then followed up the patients with ST-segment monitoring. We hypothesized that combinations of clock time and previously evaluated ECG variables could be useful for serially updated forecasts of final infarct size and that these forecasts would change with ECG signs of either successful or unsuccessful reperfusion treatment.

To estimate the amount of myocardium initially at risk for infarction, ST-segment analyses from the admission ECG were made according to the Aldrich score,[5,6] which previously has been shown to correlate with final infarct size in patients not receiving reperfusion therapy. This first ECG was also used to estimate the "acuteness" of the infarction process by the Anderson-Wilkins score,[7] which has been shown to be complementary to information from patient-reported symptom delay.[8] Patients underwent reperfusion therapy, in this study, by fibrinolysis, and were continuously monitored for ST-segment recovery analyses to assess treatment efficacy, that is, speed and quality of restoration of epicardial flow[9-11] and myocardial perfusion.[12,13] Final infarct size was estimated from a predischarge ECG by the Selvester score.[14]

We knew from previous studies that rapid, high-grade, and stable ST-segment recovery consistently had been associated with better clinical outcome[15-18] and that early and complete ST-segment recovery is indicative of smaller infarct size,[19,20] all consistent with the findings that faster restoration of myocardial perfusion results in greater myocardial salvage and smaller infarct size.[1]

The hypothesis of being able to forecast final infarct size and to serially update this, turned out to be fulfilled only for the patients who were early by both their initial ECG and delay time. In these patients, favorable ST-segment resolution resulted in excellent outcome with really small infarct sizes (median, 3% of the left ventricle), whereas an unfavorable ST-segment resolution was indicative of a large final infarct size. Among patients that were late by both the ECG score and clock time, however, the outcome did not change with signs of favorable/unfavorable ST-segment resolution. Instead, all these patients, identified at first contact by the Anderson-Wilkins acuteness score and patient-reported delay, were shown to have large final infarct sizes (median, 20% of the left ventricle) irrespective of their ST-segment resolution pattern.

Next steps

In the study above, we included initial estimations of acuteness and area at risk. To improve the first-contact prediction of outcome, we would need an estimation of the severity of the ischemia or the predicted rate of necrosis of the ischemic myocardium. The Sclarovsky-Birnbaum grading of ischemia[21] has been shown to yield such an estimation. Terminal QRS distortion, grade III ischemia, has been associated with less myocardial salvage[22] and worse prognosis,[23] indicating that patients showing these signs on their first-contact ECG could be at higher risk for treatment delay. Together with the scores mentioned above, the Sclarovsky-Birnbaum grading would give an estimation of the extent, the acuteness, and the severity of the ongoing ischemia. Thus, by combining these scores, we might have a very valuable and early decision support in the initial care and triage of patients with STEMI, but this, of course, needs to be studied.

Clinical implications

Primary PCI has been shown superior to *in-hospital* fibrinolysis[24] and is today, when available, the reperfusion treatment of choice. Because of the concern of increased infarct size as a result of treatment delay, the concept of facilitated PCI, that is, prehospital/first-contact administration of fibrinolytics or highly effective platelet inhibitors, is under investigation. So far though, this theoretically promising idea has shown discouraging results, at least when full-dose fibrinolysis is used for "facilitation" and routinely followed by an urgent PCI.[25]

The urgency and great importance of time when initially treating patients with STEMI leads, by necessity, to fast and pragmatic decisions and a tendency to lump STEMI patients together. If, however, we could individualize this first decision, based on the information that actually is in the ECG and basic clinical variables, we might be able to improve outcomes and avoid therapy-related adverse events.[26,27] There is probably a subpopulation among ST-segment elevation infarctions in which the patients are more vulnerable for transport delay than others. Patients who have their ischemic myocardium "unprotected," that is, they have had no opportunity to produce a collateral circulation, or their myocardium has had no opportunity to be preconditioned and thereby "used to" an ischemic situation, would seem more vulnerable to reperfusion delay. These patients might benefit the most from very urgent and intensive prehospital treatment, that is, prehospital fibrinolysis or facilitated PCI.