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The central role of conventional 12-lead ECG for the assessment of microvascular obstruction after percutaneous myocardial revascularization

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Abstract Guidelines report that the optimal treatment for ST-elevation myocardial infarction (STEMI) is a primary percutaneous coronary intervention (PPCI) when performed timely by trained operators. Yet, the reopening of the infarct-related artery (IRA) is not always followed by myocardial reperfusion. This phenomenon is most commonly called "no-reflow", is caused by microvascular obstruction (MVO) and is associated to a worse outcome. Electrocardiogram (ECG) is crucial for the diagnosis of STEMI, but is also useful for the assessment of MVO. In this review we summarize ECG-derived parameters associated to MVO and their prognostic relevance.

Keywords: Microvascular obstruction; Electrocardiogram; Acute myocardial infarction

Introduction

The most effective treatment for ST segment elevation acute myocardial infarction (STEMI) includes timely myocardial revascularization by pharmacological (fibrinolysis) or mechanical (primary percutaneous coronary intervention, or PPCI) interventions, which allow salvage of myocardium at risk [1,2]. Yet recanalization of the infarct-related artery (IRA) is not followed by myocardial reperfusion in a definite proportion of patients. This phenomenon, commonly known as "no-reflow", is caused by microvascular obstruction (MVO) [3]. MVO has been documented in about 30%–50% of patients undergoing successful PPCI [4–6] and, occasionally, also during elective PCI, particularly when performed on vein grafts [7] or in the setting of unstable angina [8]. The pathogenesis of MVO is complex, and can be caused by a variable combination of: 1) ischemic injury, 2) reperfusion injury [9], and 3) distal embolization of debris from plaque and/or thrombus during PCI [10,11]. Furthermore, individual predisposing factors are likely to play a relevant role in its occurrence [12]. In particular, patients with MVO present more frequently the 1976 T > C polymorphism of the adenosine 2A receptors gene [13] and a more compact fibrin network [14]. Moreover, several studies carried out in humans and in animal models [15,16] suggest that acquired risk factors such as diabetes and hypercholesterolemia might predispose to MVO. Patients presenting MVO have a worse outcome, including a higher rate of death and re-infarction [12]. Accordingly, it is important to identify these patients as early as possible, as they might benefit from a more aggressive pharmacological treatment, although no therapy has hitherto been shown consistently effective in improving or preventing MVO [17]. Notably, MVO has been reported to spontaneously improve in a few days after revascularization in a proportion of STEMI patients, and this improvement is associated with a better left ventricular remodelling [17–20].

MVO can be diagnosed by coronary angiography or by non invasive imaging techniques, including myocardial contrast echocardiography (MCE) and cardiovascular magnetic resonance (CMR). MCE uses ultrasound to visualize contrast microbubbles that freely flow within patent microcirculation and MVO is detected as lack of intramyocardial contrast opacification. CMR, using gadolinium to assess regional cardiac perfusion, diagnoses MVO as: 1) lack of gadolinium enhancement during first pass; and 2) lack of gadolinium enhancement within a necrotic region, identified

Abbreviations: CMR, cardiovascular magnetic resonance; ECG, electrocardiogram; HRT, heart rate turbulence; HRV, heart rate variability; IRA, infarct-related artery; MBG, myocardial blush grade; MCE, myocardial contrast echocardiography; MVO, microvascular obstruction; PPCI, primary percutaneous coronary intervention; PTCA, percutaneous transluminal coronary angioplasty; STE, ST segment elevation; STEMI, ST segment elevation myocardial infarction; STER, rate of ST segment elevation resolution; STR, ST segment elevation resolution; TIMI, Thrombolysis In Myocardial Infarction

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by late gadolinium hyper-enhancement [21]. These techniques, however, are usually not readily available, require specific expertise and are very expensive [21–25].

Several studies have suggested that a simple serial electrocardiogram (ECG) analysis can be very helpful to identify patients with persistent MVO within STEMI patients treated by PPCI [26–29]. Furthermore, some ECG abnormalities that suggest the presence of MVO have been shown to be helpful for risk stratification.

Fig. 1 shows the most important diagnostic features of patients presenting MVO detectable by invasive and non-invasive imaging techniques.

In this report we review the evidence for the accuracy of 12-lead ECG in the detection of MVO and its impact on patient's management.

ECG and diagnosis of MVO (Fig. 2)

Changes in ST-segment elevation

In the thrombolytic era, a large number of studies clearly demonstrated that, in patients admitted with acute STEMI, a rapid ST segment elevation resolution (STR) after fibrinolytic therapy strongly suggested effective reperfusion of the occluded IRA [30,31]. In contrast, persistent ST segment elevation (STE) or incomplete STR after treatment was in several cases associated with a failure of fibrinolysis in saving the myocardial area at risk of necrosis, suggesting a failure in restoring epicardial coronary blood flow.

In the PPCI era it has become clear, however, that the lack of rapid STR does not necessarily indicate failure to recanalize the IRA, but rather treatment's inability to restore myocardial perfusion due to MVO.

Different criteria and indexes have, however, been proposed and applied to define STR:

1) *sum STR*, expressed as the percent reduction after PPCI of the sum of STE, defined as the sum of the magnitude of STE, measured 60 ms after J point, in all leads related to infarct area [32]; referring to this index, STR is assumed as complete when $\geq 70\%$, partial when 30% to 70%, and absent when $<30\%$ [27];
2) *single-lead STR*, expressed as the percent reduction after PPCI of ST deviation in the single ECG lead with the most prominent ST segment deviation at baseline and at a given time point after reperfusion therapy; referring to this specific ECG index, it is necessary to take into account only STE in patients presenting with anterior STEMI, whereas both STE in inferior leads and ST segment depression in anterior precordial leads should be considered in patients presenting with inferior STEMI;
3) *maximal STR*, representing the absolute maximal STR at a given time of assessment, independent of ST segment changes at the baseline ECG [33]; once again, it is necessary to take into account only STE in patients presenting with anterior STEMI, whereas both inferior STE and anterior ST segment

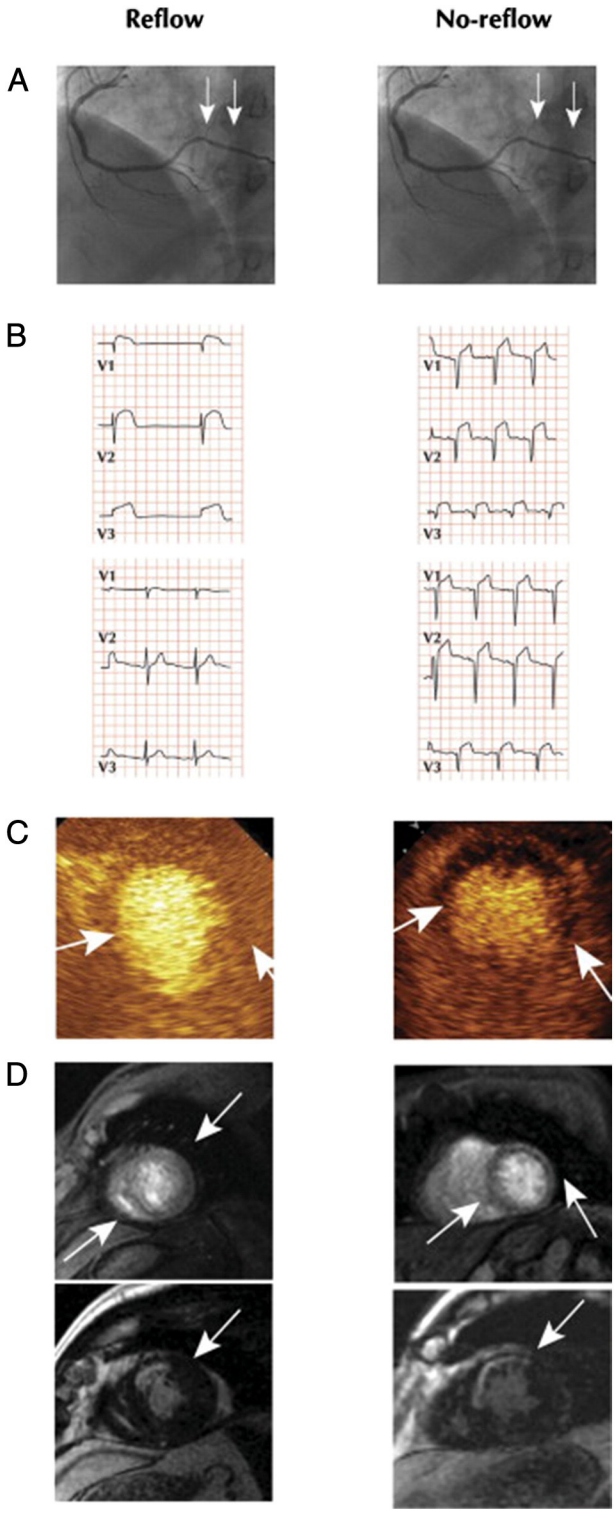

Fig. 1. (A) Right: MBG 0 (white arrows); left: MBG 3 (white arrows). (B) Right: lack of STR; left: complete STR. (C) Right: no-reflow assessed by MCE (white arrows); left: reflow assessed by MCE (white arrows). (D) Right: no-reflow assessed by magnetic resonance imaging with first-pass of gadolinium (top) or the delayed enhancement (bottom) (white arrows); left: reflow assessed by magnetic resonance imaging with first-pass of gadolinium (top) or the delayed enhancement (bottom) (white arrows). MBG = myocardial blush grade; STR = ST elevation resolution; MCE = myocardial contrast echocardiography. Color illustration online.