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ST peak during primary percutaneous coronary intervention predicts final infarct size, left ventricular function, and clinical outcome

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Abstract

Background and Purpose: One third of patients treated with primary percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction develop a secondary increase in electrocardiographic ST segment (ST peak) during reperfusion. The purpose was to determine the clinical importance of ST peak during primary PCI.

Methods: A total of 363 patients with ST-elevation myocardial infarction were stratified to no ST peak or ST peak. Final infarct size and ejection fraction (EF) were assessed by cardiovascular magnetic resonance.

Results: Patients with ST peak had a larger infarct size (14% vs 10%; $P = .003$) and lower EF (53% vs 57%; $P = .022$). Rates of cardiac mortality (8% vs 3%; $P = .047$) and cardiac events (cardiac mortality and admission for heart failure; 19% vs 10%; $P = .018$) were higher among patients with ST peak, but not all-cause mortality (8% vs 5%; $P = .46$). In a multivariable Cox regression analysis, ST peak remained significantly associated with cardiac events (adjusted hazard ratio, 2.03 [1.08-3.82]).

Conclusion: ST peak during primary PCI is related to larger final infarct size, a reduced EF, and adverse cardiac clinical outcome.

Keywords: ST peak; ST-elevation myocardial infarction; Magnetic resonance; Reperfusion injury; Primary percutaneous coronary intervention; ST resolution

Introduction

The recommended therapy for patients with ST-segment elevation myocardial infarction (STEMI) is restoration of the epicardial coronary blood flow by primary percutaneous coronary intervention (PCI) or fibrinolysis to minimize myocardial damage and improve clinical outcome.[1,2] However, patients achieving a normal epicardial blood flow (thrombolysis in myocardial infarction [TIMI] flow 3) may still have impaired microvascular perfusion and, hence, adverse outcomes.[3,4] The ST resolution is related to the extent of microvascular perfusion[5-8] and also toclinical outcome.[7-13] ST resolution is traditionally measured after the PCI procedure and may therefore be of limited clinical use. In contrast, the assessment of ST changes during reperfusion would potentially predict prognosis. In the era of thrombolysis, an increase in ST-segment elevation early after treatment seemed to be related to improved outcome and thus considered a marker of successful reperfusion.[14-17] However, ST patterns during reperfusion after thrombolysis and primary PCI differ.[17] Increased ST elevation (ST peak) during reperfusion with primary PCI is observed in approximately one third of the patients (Fig. 1). Despite final ST resolution, the ST-peak phenomenon has been

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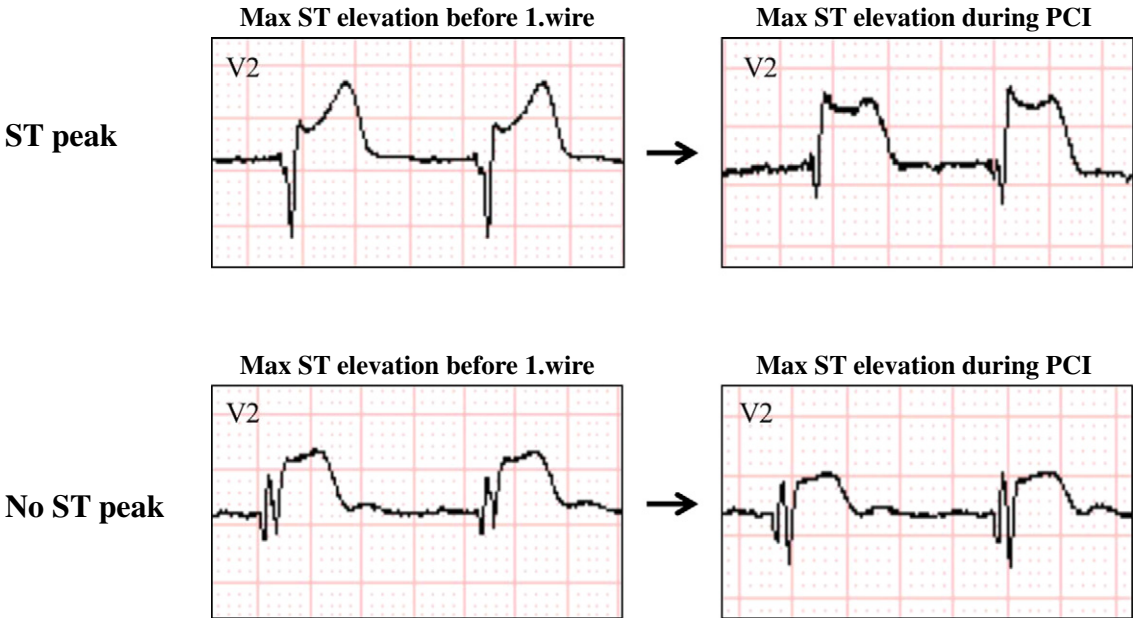

Fig. 1. Electrocardiographic examples of ST peak and no ST peak. Electrocardiographic examples (lead V_2) in 2 patients both with anterior infarction and culprit lesion in left anterior descending artery are shown. The level of ST elevation was measured at the J-point + 1/16 of the R-R interval (STM) using automatic computer software. The upper panel shows that ST peak, ST elevation before intervention is 0.385 mV (left) and 0.693 mV (right) during reperfusion. The lower panel shows that a no-ST-peak, ST elevation before intervention is 0.517 mV (left) and 0.416 mV (right) during reperfusion.

related to an increased myocardial infarct size measured by release of cardiac biomarkers and single-photon emission computed tomography, as well as to a poorer left ventricular (LV) ejection fraction (EF).[18-24] Most of these studies are, however, small, and no evidence exist regarding the association between ST peak during primary PCI and long-term clinical outcome. Furthermore, the adverse consequences of ST-peak episodes during reperfusion therapy has not yet been evaluated by cardiovascular magnetic resonance (CMR), which is considered the current method of choice for measuring myocardial infarct size.[25] Thus, the aim of the present study was to determine the association of ST peak during reperfusion in patients undergoing primary PCI with final infarct size measured by CMR and with clinical outcome.

Materials and methods

Study population and treatment

This is a retrospective substudy from 2 randomized studies in patients with STEMI.[26,27] One study compared ischemic postconditioning (IPost) to conventional primary PCI,[26] and the other study compared intravenous administration of the glucagon-like-peptide-1 analogue exenatide to placebo in primary PCI.[27] In the latter study, some patients were randomized but excluded from the parent study because they met the angiographic-based exclusion criteria. These patients were still followed up for safety reasons, and data from all randomized patients were thus available for inclusion in the present study. The present study included patients with a first STEMI and symptom duration of 12 hours or less who were field triaged directly to the PCI center or transferred from a local hospital. *ST-segment elevation myocardial infarction* was defined as ST-segment elevation measured in the ST J-point in 2 contiguous electrocardiogram (ECG) leads of more than 0.1 mV in V_4 to V_6 or limb leads II, III, and aVF, or more than 0.2 mV in lead V_1 to V_3.[28] Patients were not considered for enrollment if they presented with cardiogenic shock, unconsciousness, acute stent thrombosis, known renal insufficiency, previous myocardial infarction assessed by patient history or scarring on the CMR in the non–infarct-related territory, or previous coronary artery bypass graft surgery. All patients eligible for primary PCI were pretreated with aspirin 300 mg orally or 500 mg intravenously, clopidogrel 600 mg orally, and unfractionated heparin 10 000 units intravenously, usually administered in the prehospital setting or at the referring hospital. On arrival at the catheterization laboratory, a coronary angiography was performed to identify the culprit lesion, and primary PCI was performed as previously described.[26,27] A glycoprotein IIb/IIIa receptor antagonist was administered at the discretion of the operator if no contraindications were present. All patients were treated with clopidogrel 75 mg daily for 12 months and aspirin 75 mg daily indefinitely. Cardiac biomarkers (troponin T) were obtained before intervention and immediately after, 6 hours after, and 12 to 18 hours after intervention. All patients were informed verbally and in writing, and all gave their written consent before inclusion. The study was performed according to the Helsinki Declaration of good clinical practice, and The Danish National Committee on Biomedical Research Ethics approved the protocol.

ST peak and resolution

At admission, a 12-lead ST-monitoring ECG system was put on all patients for continuous ST-segment changes