Incidence, angiographic features and outcomes of patients presenting with subtle ST-elevation myocardial infarction

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Background Borderline electrocardiograms represent a challenge in ST-segment elevation myocardial infarction (STEMI) management and are associated with inappropriate discharges and delays to intervention.

Objectives To assess angiographic characteristics and outcomes of patients presenting with subtle ST-elevation (STE) myocardial infarction.

Methods A total of 504 consecutive patients with suspected STEMI treated by systematic primary percutaneous coronary intervention were prospectively included. Subtle STE was defined as a maximal preinterventional STE of 0.1 to 1 mm. Angiograms were interpreted by investigators unaware of the electrocardiographic data.

Results The proportion of patients with subtle STE was 18.3%, 86% of them presented with Thrombolysis In Myocardial Infarction flow grade 0/1 and 91% underwent percutaneous coronary intervention. Despite having smaller infarcts, subtle STE patients associated more frequent multivessel disease (57% vs 44%, $P = .02$) and larger delays to reperfusion. During a follow-up of 19.0 ± 4.9 months, the rates of death or reinfarction were similar among groups (10.0% vs 12.6%, $P = .467$). Subtle STE was not associated with better outcomes neither in univariate nor after adjustment in a multivariate analysis (adjusted hazard ratio 0.79, 95% CI 0.37-1.69, $P = .546$).

Conclusions Subtle STEMI is frequent in clinical practice and is usually associated with acute total coronary occlusion. Therefore, it should be diagnosed and treated in the same expeditiously manner as marked STEMI. (Am Heart J 2014;168:884-90.)

Electrocardiogram (ECG) remains a cornerstone in the management of acute myocardial infarction. New ST-segment elevation (STE) usually reflects transmural ischemia due to a complete thrombotic occlusion of an epicardial coronary artery. In this setting, urgent reperfusion by primary percutaneous coronary intervention (pPCI) significantly improves survival and reduces morbidity and costs.[1]

Current guidelines have set the cut-points for STE at 0.1 to 0.2 mV, depending on age, sex, and location.[2] The criterion for reperfusion has also been established as STE of ≥1 mm in ≥2 contiguous leads in recent trials.[3] However, STE criteria have limited sensitivity in diagnosis of myocardial infarction.[4,5] In fact, many patients only develop minor ECG anomalies and, unfortunately, do not receive prompt reperfusion.[6]

The reported incidence of nonobvious ECG varies widely, from 9% to 31% of myocardial infarctions, depending on STE criteria and the affected territory.[7-9] Observational studies have shown that less extensive ECG changes are a common cause of inappropriate discharges and larger delays to pPCI.[7,8,10]

Among nonobvious ECG, development of subtle STE (≤0.1 mV) may be particularly frequent. No study to date has specifically analyzed the frequency of subtle STE, and the clinical profile of these patients is largely unknown. Accordingly, the goals of the present study were to determine the angiographic features and clinical outcomes of patients presenting with subtle STE treated in a systematic pPCI program.

Methods

Study patients

We conducted a prospective registry of all STE myocardial infarction (STEMI) patients presenting to a tertiary care center with a 24/7 pPCI program between

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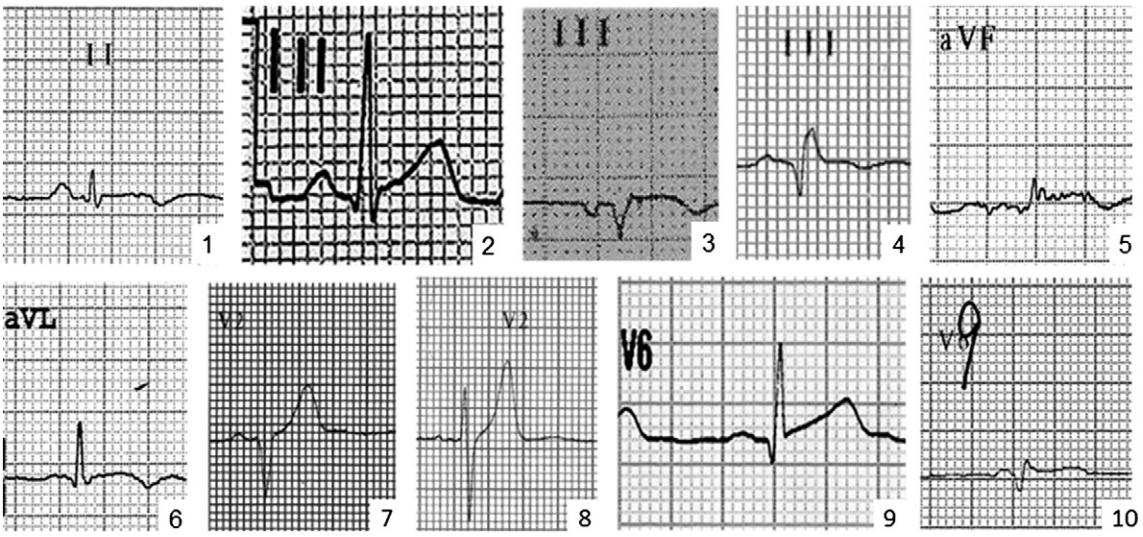
Figure 1



Examples of ten different subtle STE patients included in the study. All of them presented with culprit artery TFG 0/1, and all but cases 5 and 6 reached peak CPK levels above 1000 U/L. Examples include small vessel disease (case 6), LCx occlusion (cases 1, 4, 8-10), late presentation (cases 3, 4, 6, 9) and hyperacute proximal LAD occlusion (case 7). All but case 6 were treated by stent implantation.

June 2008 and October 2011. During the study period, 504 patients with persistent ischemic symptoms not responding to nitrates plus any STE were admitted for emergent coronary angiography. Patients with cardiogenic shock and out-of-hospital cardiac arrest were also included.

Twenty-four patients were excluded from analysis because the final diagnosis was not an acute myocardial infarction (5 patients with myopericarditis, 4 with Takotsubo syndrome, 6 with coronary spasm, 2 with old myocardial infarction, 2 with massive pulmonary embolism, and 1 each with aortic coarctation, hypertrophic cardiomyopathy, ventricular tachycardia, cardiac metastasis, and acute pulmonary edema). In addition, 33 patients were excluded because they had nonevaluable STE due to wide QRS complexes, artifacts, or technical reasons. The final study population comprised 447 consecutive patients treated by systematic pPCI. The study was approved by the ethics committee of Ramón y Cajal University Hospital, and all patients proved written informed consent.

Electrocardiographic analysis

Electrocardiograms were recorded in technical paper at standard calibration. A minimum of 2 serial 12-lead tracings plus posterior and right precordial leads were obtained in each case prior to catheterization. ST-segment elevation was measured at the J-point, in the trace and lead with maximal elevation above the TP segment. Patients were grouped at admission into 2 categories based on the amplitude of STE: (1) subtle STE defined as STE of 0.1 to 1 mm (0.01-0.1 mV) and (2) marked STE defined as STE of >1 mm (>0.1 mV). ST-segment elevation may be associated with reciprocal changes, or hyperacute, negative, or domed T waves (Figure 1). Infarct location was classified as inferior (main STE in leads II, III, and aVF), anterior (main STE in precordial leads V_1-V_4), and lateral or inferobasal (main STE leads V_5-V_9, I, or aVL, without STE in inferior leads).

Interventional procedure and clinical management

Catheterization laboratory was activated by cardiology staff in 24/7 onsite service. In case of diagnostic dilemma, urgent bedside echocardiography revealing wall motion anomaly was required prior to angiography. Patients were pretreated with aspirin and clopidogrel and received weight-adjusted heparin at the beginning of the procedure. Adjunctive abciximab and thrombectomy were used at the operator's discretion. Primary percutaneous coronary intervention was performed according standard practice, exclusively at the infarct-related artery (IRA), with the exception of cardiogenic shock. Biochemistry samples for troponin I and total creatine kinase were extracted at every 6 hours until peak value and daily thereafter. Follow-up was obtained in every patient by office visits or telephone calls.

Definitions and outcomes

The primary end point was the preprocedural frequency of Thrombolysis In Myocardial Infarction flow grade (TFG) of 0 or 1.[11] Secondary end points were death from any cause, reinfarction, repeat revascularization, definite

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