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Original Article

ST elevation: Differential diagnosis and caveats. A comprehensive review to help distinguish ST elevation myocardial infarction from nonischemic etiologies of ST elevation

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A B S T R A C T

Prompt diagnosis of acute ST segment elevation myocardial infarction (STEMI) by the initial ECG is important in order to perform an urgent coronary angiography as soon as possible and achieve successful revascularization, therewith improving mortality and morbidity. Several diseases and conditions can mimic an acute myocardial infarction (AMI) but may not benefit from a (percutaneous) revascularization strategy. This narrative clinical review will discuss the ECG features of some of the causes of non-ischemic ST segment elevation to facilitate early recognition, prevent wrongful diagnosis and improve treatment outcomes.

1. Introduction

The diagnosis of STEMI should be made by a 12-lead ECG.[1] Presence of ST segment elevation (STE) in a patient with acute chest pain should be considered as STEMI. If uncertainty about the diagnosis exists, repetitive ECG recordings, together with additional information from echocardiography and laboratory testing, can be included as long as it does not cause any delay in reperfusion therapy.

Since only a minority of patients with chest pain and STE seen in the emergency department have a final diagnosis of AMI, also alternative non-ischemic causes may be considered, especially if less typical symptoms are present.[2,3] Although these patients would not directly benefit from revascularization therapy, an urgent coronary angiography (CAG) might still be indicated according to the guidelines to rule out obstructive coronary artery disease in the first place, especially when uncertainty exists.

This paper provides an updated comprehensive review of some non-ischemic causes of STE.

2. Electrophysiology of ST elevation

In a typical dome shaped action potential (AP) the ST segment starts with the J point at the onset of the plateau phase (phase 2) when the majority of myocardial cells have gone through rapid depolarization (phase 0) and early repolarization (phase 1). It ends with the T wave when repolarization (phase 3) returns the myocardial cells back into the negative charged resting phase

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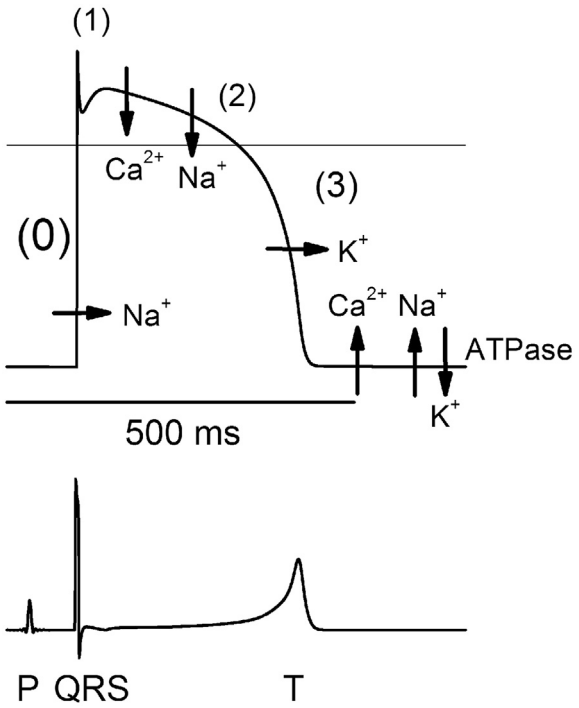

Fig. 1. Schematic illustration of the action potential on the ECG. How the different electrophysiological phases of the action potential correspond with the surface ECG. With permission from: Timour Q, Frassati D, Descotes J, et al. Sudden death of cardiac origin and psychotropic drugs. Front Pharmacol. 2012;3:76.

(phase 4) (see Fig. 1). In normal situations the ST segment will be reflected as an isoelectric horizontal line at the 'baseline' because all cells have the same membrane potential during plateau phase 2 and no net voltage gradient is present in the myocardium.[4–6]

Any (local) delay in activation or distortion in duration, height and/or shape of the AP causes a voltage gradient ('current of injury') between injured myocardial cells and surrounding unaffected cells that influences the ST segment. Depending on the timing and location of the lesion, the ST segment can become elevated or depressed with many possible morphologies (eg. strait horizontal or oblique, upsloping or downsloping, concave upward or convex upward, dome shaped, etc.). In general, the ST segment vector points towards a lesion causing STE in transmural or epicardial lesions, and depression of the ST segment if only (sub)endocardial damage is present (see Fig. 2).

3. ST elevation myocardial infarction

In STEMI transmural ischemia is present due to abrupt blockage of a coronary artery, both organic or functional (see Fig. 3).

Ischemia counteracts the working of ion channels causing a change in AP by loss of voltage gradient, both in repolarization (diastole) and in depolarization (systole).[4,7] Leads 'overseeing' an ischemic region record the current of injury in systole as an elevation of the ST segment. During diastole this actually would cause a depression of the TP and PR segment, but since the baseline is (artificially) depicted isoelectric all other segments have an upward shift recognized as additional STE (see Fig. 2).

Opposing leads can show STE inverted as *reciprocal* ST segment depression.[4,5,7] The presence of concomitant reciprocal ST-segment depression in opposing leads has a high specificity for a STEMI and is therefore always important to look for, sometimes by using additional recordings of the right precordial leads (V3R and V4R) or posterior leads (V7-V9).

An important remark must be made that simultaneous ST abnormalities can interfere, known as ST segment 'counterpoise'.[8] This can lead to 'cancellation' if ST elevation (or depression) is present at the same time in two opposing sides of the heart. Contrary, ST segment abnormalities might also be enhanced in case of both (minor) ST elevation on one side and (major) ST depression on the other side, causing the risk of overlooking STE due to remarkable ST depression in the opposing leads.

Upsloping convex STE during the acute phase of AMI, as described by Harold E.B. Pardee in his original paper in 1920,[9] is considered the typical morphology of a STEMI ("*Pardee's sign*") and represents a high positive predictive value. Nevertheless, mostly a concave morphology of STE is seen which is less specific for STEMI and can be found as well in many other non-ischemic causes.

Weakening of the electrical activity in ischemic myocardium leads to a decreased R wave amplitude. If myocardial cells are no longer active, as is the case in a more extensive infarction, only the activity of opposing areas appear on the ECG as Q waves. Small Q waves may be present in healthy individuals as the normal left-to-right depolarization of the interventricular septum ('septal' Q waves) seen in the left-sided leads (I, aVL, V5 and V6) or in leads III and aVR as a normal variant. Q waves may also (temporarily) exist in circumstances where the vector of depolarization has changed; e.g. intraventricular conduction defect, ventricular overload and myocardial hypertrophy.

Following an infarction the weakened transmural necrotic myocardium may develop an aneurysm, most frequent of the apex and anterior wall. An aneurysm may be recognized as persistent STE, T wave inversion and loss of R wave in the precordial leads. The morphology of the ST segment ranges from subtle concave to ample convex. It must be noted that a more extensive acute myocardial infarction may still develop in or near a former infarcted region, resulting in new or more obvious STE with the pre-existent loss of R wave amplitude. The appearance of distinct Q waves, absence of reciprocal ST depression and ECG abnormalities that remain unchanged over time can help distinguish an aneurysm from STEMI. Of course, and especially in this situation, the ECG has to be interpreted in conjunction with the clinical presentation and possible information from other diagnostic tools (e.g. echocardiography).

4. Pericarditis/Myocarditis

Although the pericardial sac itself has no electrical activity, inflammation of the pericardium (pericarditis) can disrupt the AP in the epicardium.[5] As a result of the diffuse process the current of injury may be recognized without correspondence to a specific coronary territory. STE will be present in most leads, except for aVR and V1 where ST segment depression can be seen due to its distant and opposite position of the normal heart axis (see Fig. 4). Involvement of the atria is responsible for depression of the PR segment and is highly suggestive for pericarditis although it may be sporadically seen in cases where myocardial infarction or early repolarization extended to the atria.

The repolarization abnormalities undergo different chronological phases that might not all be seen in every patient.[10] In the first phase, up to two weeks after the onset of symptoms, an upward concave STE with positive T wave and PR depression can be appreciated (with opposite findings in lead aVR and V1). After the first days to several weeks, PR and ST segments normalize and the T wave may become flattened. The last phase consists of a symmetrical T wave inversion (mostly in leads with former STE) that

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