The electrocardiographic evidence of injury appears in the presence of evident diastolic depolarization due to an important reduction in blood supply or other non-ischemic causal factors. This in turn leads to a "defective" transmembrane action potential reflected upon the ECG tracing as an ST-segment deviation. When diastolic depolarization occurs at subendocardial level, an ST-segment descent appears on the ECG, while depolarization at subepicardial (or transmural) level gives rise to ST-segment elevation.

In patients with acute coronary syndrome (ACS), the ST-segment elevation pattern is associated to rupture or fissuring of an unstable atheroma plaque, with acute thrombotic occlusion of an epicardial artery in a patient without previous important ischemia (ACS with ST-segment elevation [ACSSTE]). When occlusion is incomplete or distal flow is present thanks to collateral circulation, ST-segment elevation is not observed (ACS without ST-segment elevation [ACSWSTE]). The initial management of ACSTTE involves emergency aperture of permeation of the affected artery through pharmacological (thrombolysis) or mechanical means (percutaneous revascularization). In contrast, thrombolysis is not effective in ACSWSTE, and coronary revascularization in such cases is not an immediate emergency concern.

In a small group of patients ST-segment elevation is transient (less than 20 min). According to the current criteria of the International Society for Holter and Noninvasive Electrocardiology, such cases correspond to patients with a "predominance of ST-segment elevation" that are in turn subclassified according to the degree of elevation of the myocardial necrosis markers as either "aborted infarctions" or "infarctions without ST-segment elevation". The main ACS management guides have not defined a specific designation for these patients, and include ACS with transient ST-segment elevation within the group of ACSWSTE.

In this number of Medicina Intensiva, Arroyo-Úcar et al. report that patients with ACS and transient ST-segment elevation exhibit a characteristic profile that differentiates them from the rest of patients with ACSWSTE: younger age, a greater prevalence of males and smokers, a lesser elevation of the necrosis markers, a greater ejection fraction and a higher prevalence of single-vessel disease.

These results are consistent with those of the previous studies. In this context, Drew et al., in a study involving continuous ST-segment monitorization, found patients with episodes of ST-segment elevation to have a greater frequency of single-vessel disease, while patients with ST-segment depression showed more frequent multivessel disease.
In turn, Meisel et al. found that in comparison with the group of patients with persistent ST-segment elevation, those with ACS and transient ST-segment elevation showed a lesser creatine kinase peak, a greater ejection fraction, less extensive coronary disease, a greater prevalence of TIMI III flow, and fewer coronary events over the course of follow-up.

This profile of limited myocardial damage with normal systolic function refers to the known good prognosis of patients with ACSSTSTE that normalize their ST-segment after thrombolysis, or to the patients subjected to primary percutaneous coronary revascularization with initial TIMI III flow. However, in the recent metaanalysis of individual patients included in the FRISC-II, RITA and ICTUS trials, the cardiovascular mortality or infarction risk was found to be practically identical in the patients with transient elevation of the ST-segment (19.2% versus 19.4%, respectively).

Some authors have suggested that initial conservative management may be indicated in these cases, considering their supposedly good prognosis. However, as correctly commented by Arroyo-Úcar et al., the treatment of ACS with transient ST-segment elevation has not been well established, and the ACSSTSTE management guidelines offer no specific recommendations for such cases. Therefore, an important gap in knowledge is found in this field that must be filled by future research work.

It should be mentioned that the study of ACS with transient ST-segment elevation poses some specific difficulties. Firstly, transient ST-segment elevation is not a nosologic entity as such but rather a clinical sign that may be due to causes other than coronary thrombosis, such as coronary vasospasm (variant angina) or transient apical dysfunction syndrome (tako-tsubo syndrome), among other anecdotal conditions with different therapeutic connotations. On the other hand, the dividing or differentiating lines between some of these disease conditions have not been well defined. The section of the ARIAM study corresponding to 2010 identified 64 patients with transient ST-segment elevation out of a total of 1379 patients with an initial diagnosis of ACS (4.6%); of these, 3 were classified as corresponding to Prinzmetal angina, 12 as unstable angina (“aborted infarctions”), 45 as infarctions, and 4 as other conditions/causes (unpublished data).

Secondly, the dynamic nature of ACS must be stressed, with frequent episodes of myocardial ischemia due to thrombus formation and lysis, intermittent vasoconstriction and platelet thrombus embolization. As an example, we can consider the case of a patient with persistent subepicardial damage secondary to thrombotic occlusion of an artery, exhibiting spontaneous reperfusion. Depending on the moment of the ECG recording, the patient may be classified as presenting ACSSTSTE (early ECG), ACS with transient ST-segment elevation (ECG tracing immediately before reperfusion), or ACSSTSTE (late ECG tracing).

In principle, continuous ST-segment monitoring with multiple leads could obviate the low sensitivity associated with surface ECG recordings in detecting transient ST-segment elevation. Caution is required, however, since studies with conventional ECG and those based on continuous ECG monitoring differ not only in the diagnostic tool used but also in the population under evaluation. In the former case, subjects with transient ST-segment elevation are patients who have probably experienced spontaneous reperfusion. The latter case in turn corresponds to individuals who continue to suffer ischemic episodes, and is therefore at an increased risk of developing coronary events.

In any case, the scientific evidence regarding the management of these patients should be derived from randomized clinical trials involving patients with ACS with transient ST-segment elevation. Until such information becomes available, non-randomized studies fitting for treatment tendencies or systematic reviews of randomized trials (analyses of subgroups according to the initial ECG pattern) could contribute useful evidence for decision taking in this important group of patients.

References


