Available online at www.sciencedirect.com

SciVerse ScienceDirect

Journal of Electrocardiology 45 (2012) 463–475

JOURNAL OF Electrocardiology

www.jecgonline.com

Common pitfalls in the interpretation of electrocardiograms from patients with acute coronary syndromes with narrow QRS: a consensus report☆

Yochai Birnbaum, MD, FACC, FAHA,[a,*] Antoni Bayés de Luna, MD, PhD,[b] Miquel Fiol, MD,[c] Kjell Nikus, MD,[d] Peter Macfarlane, MD,[e] Anton Gorgels, MD,[f] Alessandro Sionis, MD,[g] Juan Cinca, MD,[g] Jose A. Barrabes, MD,[h] Olle Pahlm, MD,[i] Samuel Sclarovsky, MD,[j] Hein Wellens, MD,[k] Leonard Gettes[l]

[a] *Department of Medicine, Section of Cardiology, Baylor College of Medicine, Houston, TX, USA*
[b] *Institut Catala Ciencies Cardiovasculars, Hospital Sant Pau, Barcelona, Spain*
[c] *Hospital Son Espases, Palma de Mallorca, Spain*
[d] *Heart Center, Tampere University Hospital, Finland*
[e] *University Clinic of Cardiovascular Disease, Glasgow, UK*
[f] *Maastrich University Medical Center, The Netherlands*
[g] *Hospital Sant Pau, Barcelona, Spain*
[h] *Hospital Vall d'Hebron, Barcelona, Spain*
[i] *University Hospital Lund, Sweden*
[j] *Emeritus Professor, Tel-Aviv University, Israel*
[k] *Emeritus Professor of Cardiology, Maastricht University. The Netherlands*
[l] *University of North Carolina, Chapel Hill, NC, USA*

Received 29 March 2012

Abstract Acute coronary syndromes (ACS) with narrow QRS are divided into 2 groups: ST-elevation ACS that requires emergency percutaneous coronary intervention, and non–ST elevation ACS. The classification of ACS into these 2 groups is not always straightforward. In this document, we discuss several electrocardiogram patterns of acute ischemia that are often misinterpreted. We suggest that any new recommendations or guidelines from the Scientific Societies should acknowledge these aspects of electrocardiogram interpretation by including appropriate diagnostic criteria that should prove helpful for the optimal management of patients with ACS.

Keywords: Electrocardiogram; Acute coronary syndromes; ST elevation; ST depression; T wave

Introduction

Acute coronary syndromes (ACS) are divided into 2 groups, for the purpose of diagnosis, risk stratification, and management: ST-segment elevation ACS (STE-ACS) and non–ST-segment elevation ACS (NSTE-ACS).[1] It is commonly accepted that ischemic ST-segment elevation is the electrocardiographic manifestation of complete or near-complete thrombotic occlusion of an epicardial coronary artery, resulting in a sudden homogeneous transmural ongoing ischemia of an area of the left ventricle (LV) that was previously relatively, or totally, free of ischemia. In these cases, the risk of myocardial necrosis is imminent if the coronary artery remains occluded ("time is muscle"). On the other hand, NSTE-ACS is usually not associated with ongoing transmural ischemia.[1]

As the term implies, the STE-ACS group includes cases of ACS with ST elevation. In addition, it includes cases with an occlusion of the left circumflex (LCX) or distal right coronary artery (RCA) with transmural involvement of the inferolateral segments, manifested as ST depression in V_1 to V_4 (a mirror pattern of the ST elevation in leads facing the ischemic zone; Fig. 1A). This gives rise to the term *STE-ACS equivalent*.

☆ This work was sponsored by the International Society for Holter and Noninvasive Electrocardiology.

* Corresponding author. John S. Dunn Chair in Cardiology Research and Education, the Department of Medicine, Section of Cardiology, Baylor College of Medicine, Houston, TX 77030, USA.
E-mail address: ybirnbau@bcm.edu

0022-0736/$ – see front matter © 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2012.06.011

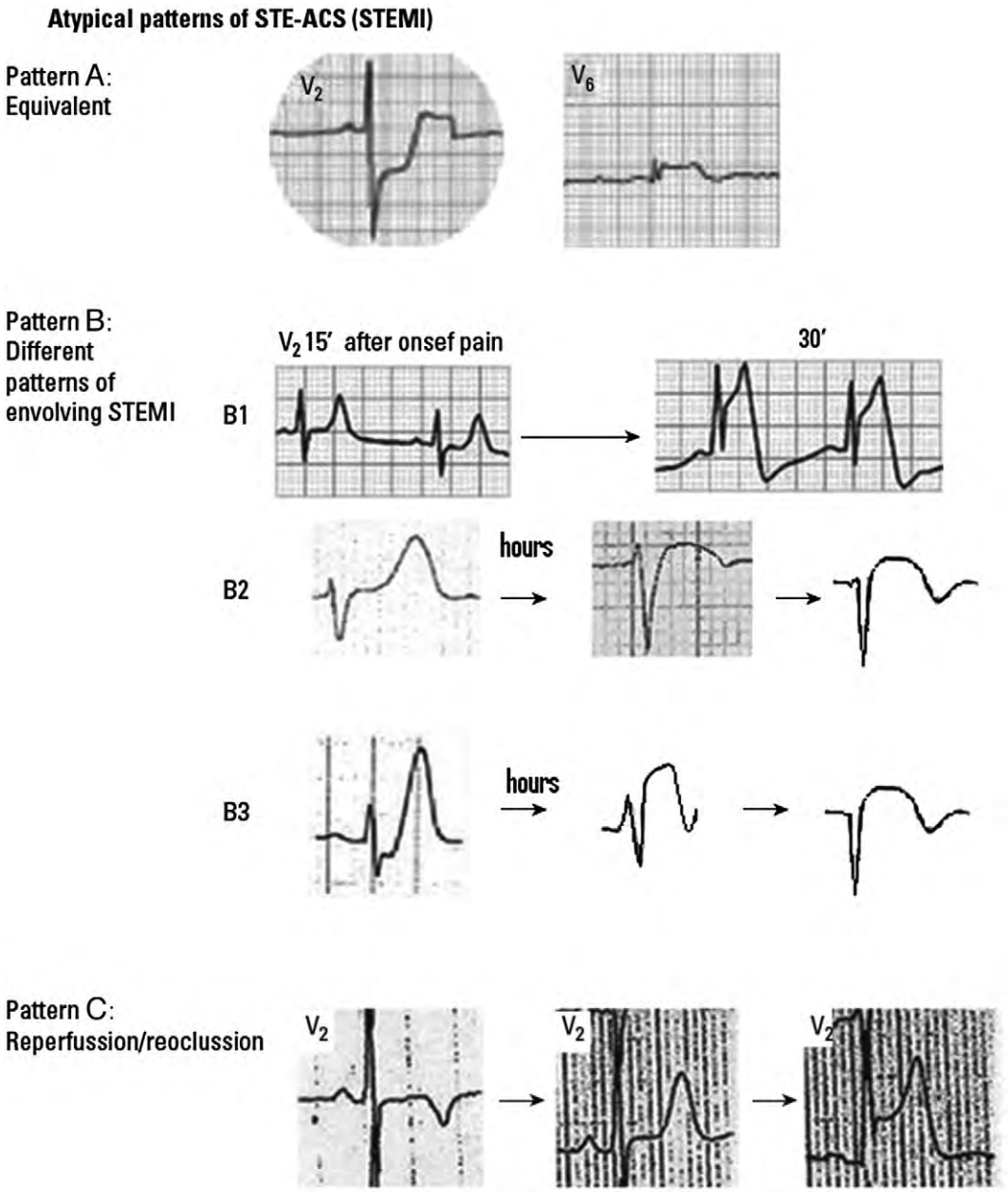

Fig. 1. Atypical patterns of STE-ACS. Pattern A: STE-ACS equivalent in LCX occlusion (involvement of LCX). Pattern B: 3 patterns of positive peaked and symmetric T waves as a first sign of ischemia. B1: transient pattern followed by ST elevation a few minutes later. B2 and B3: examples of persistent tall positive T waves (B3 with ST depression) lasting several hours, before complete occlusion and transmural involvement evolving to Q wave MI (see text). Pattern C: a deep negative T wave when the pain disappears in STEMI (postischemic change). However, the pain may reappear (new occlusion due to new thrombus or coronary spasm), with pseudonormalization of the T wave, and later ST elevation evolves.

Two atypical electrocardiogram (ECG) patterns may be seen during the evolving course of STE-ACS, especially in cases of left anterior descending coronary artery (LAD) occlusion: (i) peaked and wide, positive T waves without significant ST deviation in the hyperacute phase of ACS (this pattern is usually transient and may evolve to ST elevation within a short time) (Fig. 1B1) and (ii) deep negative T waves, as a postischemic ECG pattern, if the ACS is (spontaneously) aborted (Fig. 1C). However, it should also be remembered that nonischemic ST elevation is prevalent, and patients may present with NSTE-ACS physiology, but their ECG may show ST elevation ("pseudo STE-ACS").[2]

In NSTE-ACS, the culprit artery is usually not totally occluded. In the case of a total occlusion, the myocardial segment involved still receives blood through collateral circulation or is protected by preconditioning. Thus, the left ventricular wall involvement may not be transmural, and although the risk of extensive necrosis may exist, it is usually not imminent. Many cases with NSTE-ACS present after symptoms have ameliorated, and the ECG may not reflect active ischemia but postischemic changes. Non–ST-segment elevation ACS encompasses all non–ST-elevation patterns including: (i) ST depression with or without a final positive T wave, (ii) flat or mildly negative T waves, (iii) changes in

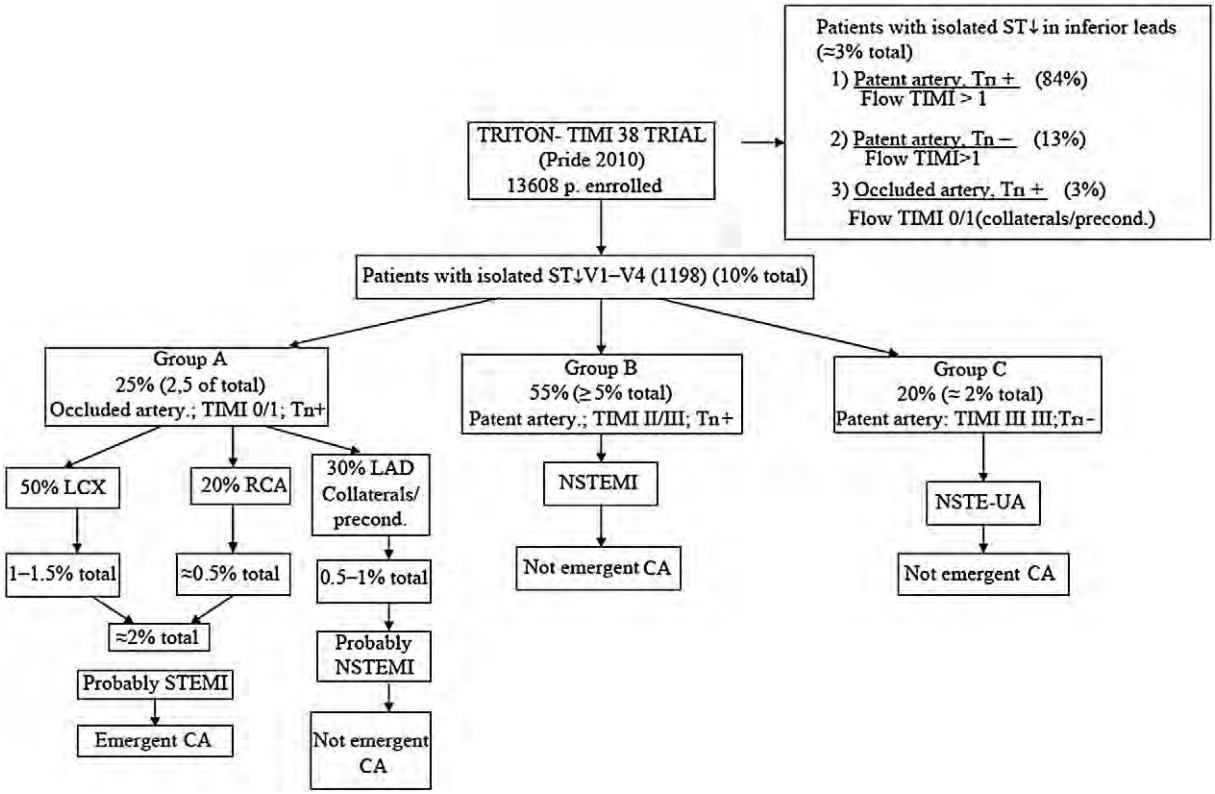

Fig. 2. Patients of the TRITON-TIMI 38 trial who presented with isolated ST depression in V_1 to V_4. Note the type of occluded artery, TIMI flow, troponin levels (positive or negative), and probable type of ACS.[6] The presence or absence of a final positive T wave in leads with ST depression and in leads in which ST depression is more evident is not indicated. The cases of LCX or RCA occlusion (group A) are most likely true STE-ACS. CA, coronary angiography. Tn, troponin.

the U wave, and (iv) a normal ECG or no dynamic changes as compared with "baseline" abnormal ECG pattern.

The classification of ACS as STE-ACS or NSTE-ACS is not always straightforward; misclassification commonly occurs. Acute coronary syndrome classification can be affected by many factors: the cancellation of electrical forces, the presence of multiple stenoses, the presence of collaterals, the severity of ischemia while ECG was recorded, and heart rate. In cases of NSTE-ACS, it is often difficult, if not impossible, to correlate the ECG changes with the occluded artery.[3,4] On the other hand, the severity of ischemia, the culprit artery, and even the exact location of the occlusion can usually be determined in patients with STE-ACS.[5]

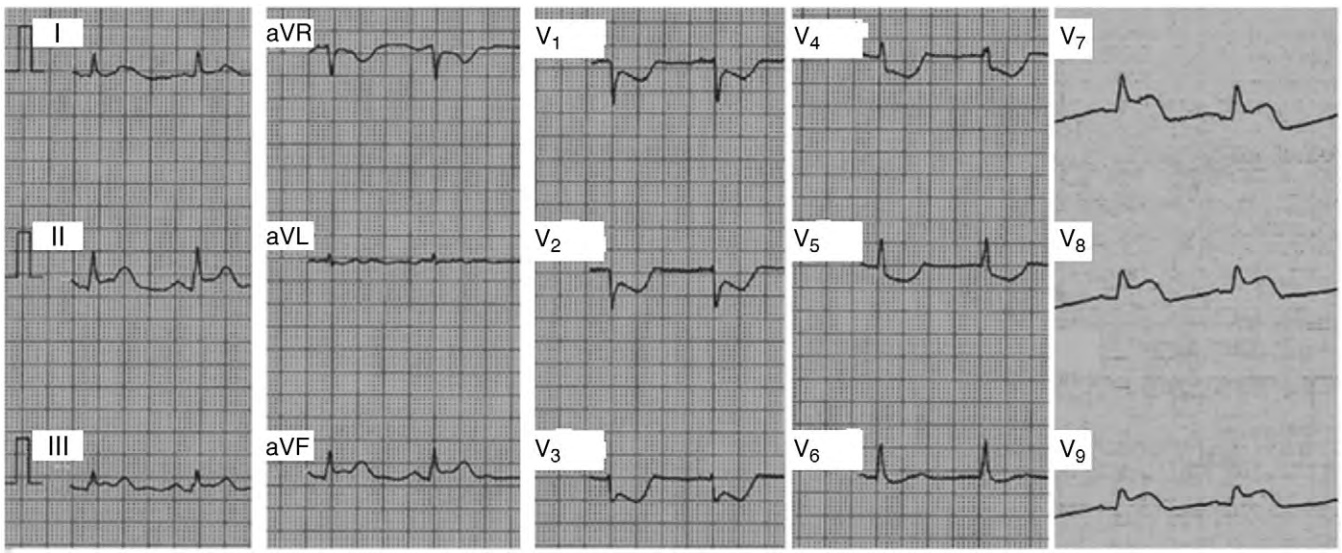

Fig. 3. A case of hyperacute STE-ACS due to LCX occlusion, in which ST depression in many precordial leads is seen (V_1-V_5) without a final positive T wave and with ST elevation in II, III, and VF. The recording of V_7 to V_9 visualizes the STE-ACS pattern better. However, leads V_7-V_9 are often not recorded and the ECG is interpreted as NSTE-ACS.

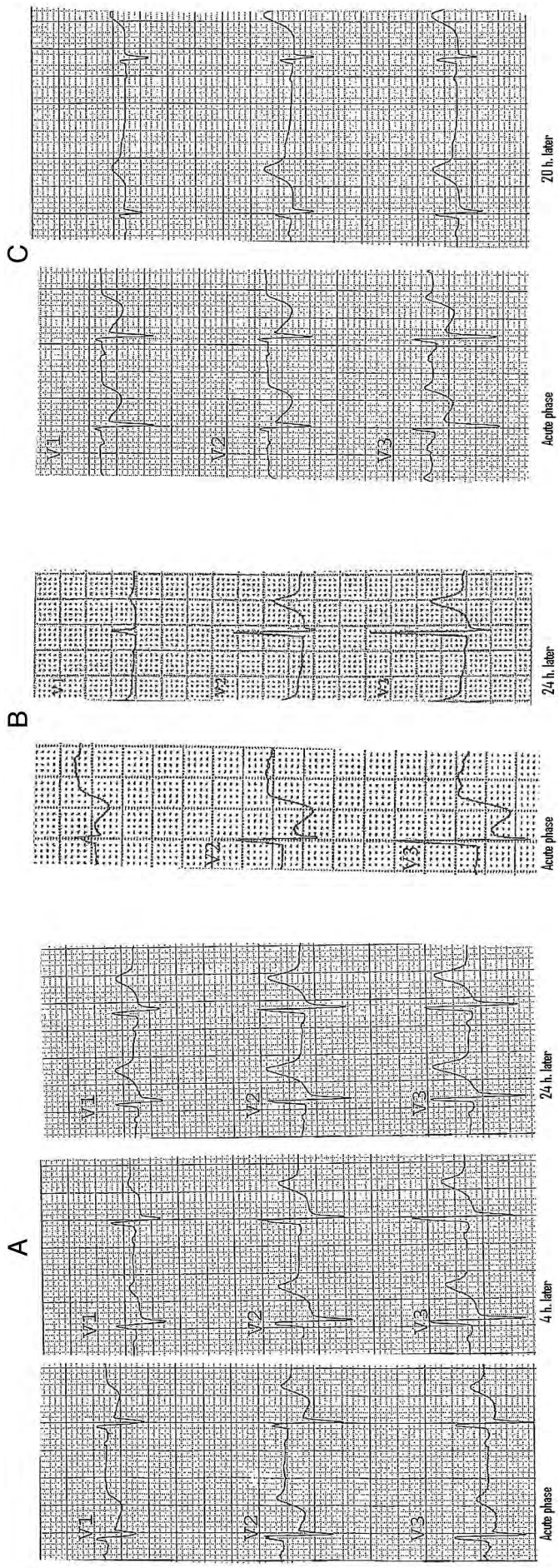

Fig. 4. Three examples of patients with ACS with ST depression in V_1 to V_3 evolving to lateral MI due to LCX occlusion (STE-ACS equivalent—Fig. 1). In the acute phase, the terminal part of the T wave in V_1 is not positive, and in the evolving phase, there is a decrease in ST depression and the terminal part of the T wave becomes positive (mirror pattern).

The aim of the present document is to present a guide to interpretation of selected ECG patterns in patients with ACS that are often misinterpreted.

A. ST depression in the precordial leads V_1 to V_3/V_4

This pattern was shown to appear as an isolated ECG change in 10% of ACS cases in the TRial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet InhibitioN with Prasugrel-Thrombolysis In Myocardial Infarction-38 trial (TRITON-TIMI 38)[6] (Fig. 2). Frequently, these cases are labeled NSTE-ACS caused by LAD subocclusion, when, in fact, they may represent STE-ACS equivalent due to LCX or distal RCA occlusion (Fig. 3). Numerous studies have shown that a large percentage of patients presenting with ST depression in V_1 to V_4 (either limited to these leads or, if more diffuse, maximal ST depression is in these leads) have LCX occlusion (STE-ACS equivalent) and not NSTE-ACS (due to proximal LAD subocclusion). In cases of anterior subendocardial ischemia, most often, maximal ST depression is seen in leads V_4 to V_5. However, in a case of predominant ST depression in V_1 to V_4, it is important to identify whether the ECG pattern is due to transmural ischemia of the inferolateral segments caused by complete occlusion of the LCX or distal RCA, or anterior subendocardial ischemia caused by proximal subocclusion of the LAD. In many cases of LCX occlusion, there is no, or only minimal, ST elevation in the inferior or lateral leads, although ST elevation may be detected in leads V_7 to V_9 (Fig. 3). A correct diagnosis is vital because LCX occlusion requires emergency percutaneous coronary intervention (PCI), whereas the immediate management of NSTE-ACS (LAD subocclusion) may not.

In the hyperacute phase of ischemia caused by LCX occlusion, typically there is ST depression in V_1 and the T waves in V_1 to V_2 are mostly negative (Figs. 3 and 4). On the other hand, in LAD subocclusion (or occlusion with collaterals), ST depression in lead V_1 is less prominent, and typically, the terminal portion of the T waves is positive, especially in V_2 to V_4 (Figs. 5 [right], 6, and 7B). The amplitude of the positive T-wave component is often greater than the amplitude of the ST depression.[7] In the course of evolving myocardial infarction (MI) caused by LCX occlusion, after a number of hours, the amplitude of the R wave increases, the magnitude of the ST depression decreases, and the terminal component of the T wave becomes positive, corresponding to a mirror pattern of evolved lateral MI (Q waves, ST elevation with terminal T-wave inversion)[8] (Fig. 4). At this stage, the differentiation from LAD subocclusion is more difficult but is probably less important than that in the hyperacute phase. The changes observed in leads positioned on the back (V_7-V_9) may help to clarify the diagnosis, but these leads are frequently not recorded. It is therefore important to make a diagnosis based on a 12-lead ECG and to always consider the clinical presentation, especially the presence of ongoing symptoms. However, the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines for the management of patients with STE-ACS have made completely opposite recommendations.[1] The guidelines recommend that reperfusion therapy may be appropriate in patients with marked ST depression confined to leads V_1 to V_4 and accompanied by tall R waves in the right precordial leads (V_1-V_4) and upright T waves indicative of a true posterior (inferolateral) injury current and circumflex coronary occlusion. We believe that this pattern is the late "mirror image" of fully evolved ST-segment MI (STEMI) (Q waves with terminal T-wave inversion) and not the acute phase of STEMI. Likewise, the "Universal Definition

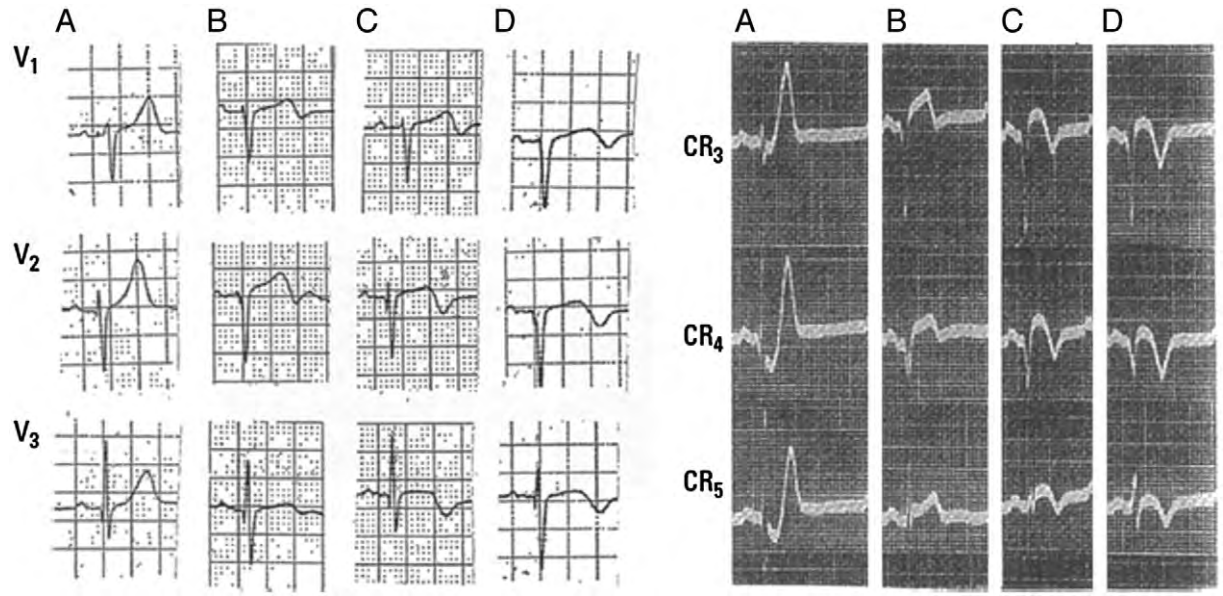

Fig. 5. Left: a patient with acute chest pain and evolving anterior wall MI. A, During prolonged chest pain showing positive peaked T waves without ST-segment elevation. B and C, ECG recorded 12 hours (ST elevation) and 24 hours later. D, ECG 1 week later with QS and deep negative T wave.[12] Right: patient with acute chest pain. A, Three hours after the onset of chest pain. B, Changes in QRS (QS and ST elevation) were not present until 18 hours after the onset of symptoms. C and D, Progressive inversion of the previously high T waves.[16]

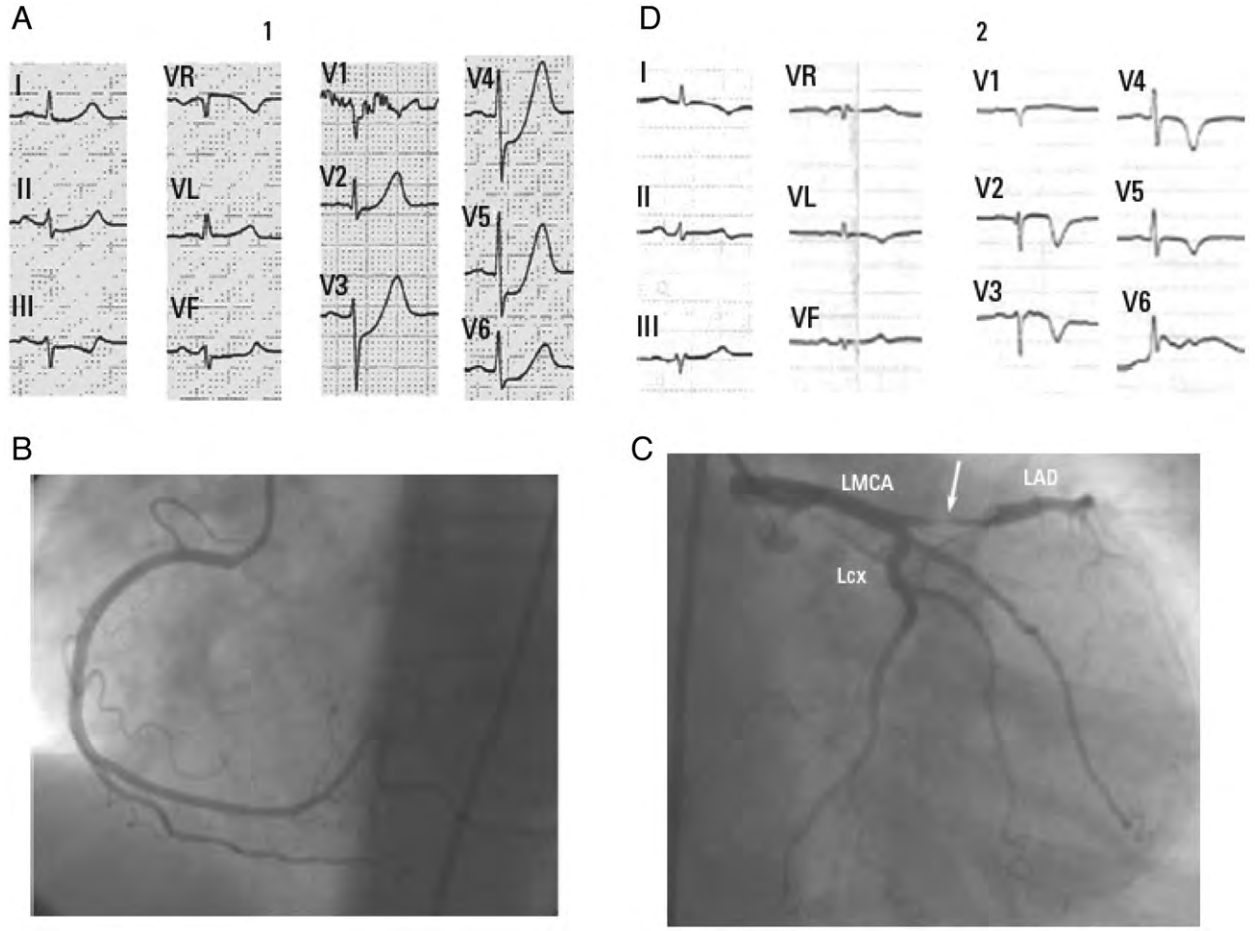

Fig. 6. A, ECG showing diffuse ST-segment depression with positive T waves in the inferior leads II, III, and aVF and in leads V_2 to V_6, along with an ST-segment elevation of 0.1 mV in lead aVR. The ST segment in lead V_1 cannot be reliably analyzed because of technical artifacts. B, Right coronary artery with a large posterior descending branch. No significant stenoses are present. C, Left anterior oblique caudal angiographic view shows a very tight stenosis in the proximal LAD (arrow). D, Discharge ECG of the patient showing Q wave in V_1 and V_2 and inverted T waves in leads V_2 to V_6 and in leads I and aVL. Probably, the patient evolved to STE-ACS with final residual "q" in V_1 to V_2 and a deep negative T wave in V_2 to V_5 because of final transmural involvement (adapted from Gul and Nikus[34]).

of Myocardial Infarction" document also states that ST depression in leads V_1 to V_3 suggests myocardial ischemia and STE-ACS equivalent, especially when the terminal T wave is positive (ST-elevation equivalent).[9] More studies are needed to assess the accuracy of these patterns to differentiate between acute inferolateral STEMI equivalent and anterior subendocardial ischemia in the clinical setting.

According to From et al[10] in cases of acute MI due to single-vessel acute coronary occlusion requiring PCI, the most frequently found culprit artery was the LAD (≈45%), followed by the RCA (≈35%) and the LCX (≈20%). ST-segment elevation ACS due to occlusion of the left main trunk (LMT) or side branches is rare. However, only 10% of all types of STE-ACS are caused by LCX occlusion, suggesting that in many cases with LCX occlusion present as NSTE-ACS, although there is no evidence to support this. It is plausible that many cases with LCX ACS, STE-ACS equivalent pattern (ST depression in V_1-V_4 with mild or even no ST elevation in the inferior or lateral leads), could be misdiagnosed as NSTE-ACS (Fig. 3). As previously mentioned, in the TRITON-TIMI 38 study[6] (Fig. 2), approximately 10% of the patients presented with isolated ST depression in V_1 to V_4. Of these, 25% had a complete artery occlusion with TIMI 0/1 flow and elevated cardiac troponins, with LCX or RCA as the culprit artery in 70%. It seems probable that these cases represent true STE-ACS equivalent (Fig. 2) requiring emergency reperfusion therapy. The remaining 30% had an LAD occlusion but did not show an ST-elevation pattern probably because of residual perfusion via collaterals or partial spontaneous reperfusion.

B. Prominent positive T waves

The only ECG change in the hyperacute phase of STE-ACS may be a symmetric, wide, and usually tall T wave that is often transient (Fig. 1B1).[5,11] Occasionally, the pattern of tall T waves is more persistent[12] (Fig. 1B2) possibly because the ischemia is not severe (Fig. 5, left). This pattern is caused by subendocardial ischemia (prolonged action potential [AP])[13,14] (Fig. 8A).

C. ST depression with prominent positive T waves

Occasionally, there is no transmural ischemia of the left ventricular wall, but because of significant subendocardial

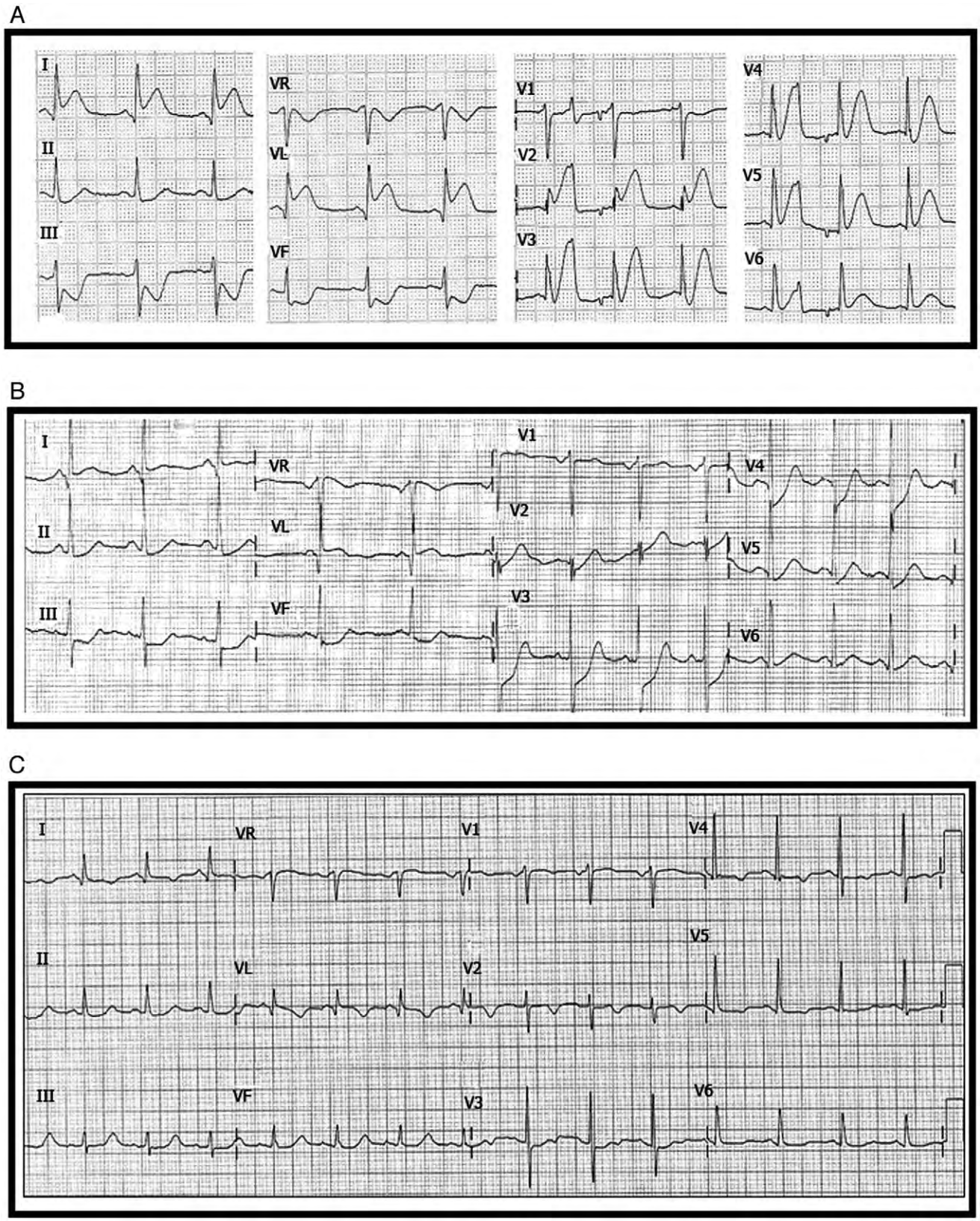

Fig. 7. A, A 59-year-old patient with chest pain that lasted for more than 1 hour. The ECG corresponds to a first-stage STE-ACS (ST elevation in I and VL and tall wide T wave from V_2 to V_5 of the hyperacute phase). ST depression in aVR and V_1 (the latter with flat T wave) is consistent with an occlusion distal from the first septal branch. The patient received antithrombotic treatment and was sent to a reference hospital for PCI. B, On arrival, the patient still presented with precordial pain of lower intensity, and the ECG has changed significantly. Now, the ST segment in I and VL is only mildly elevated, and in the precordial leads, there is a pattern of NSTE similar to the ECG pattern that may be seen in isolated tight subocclusion of the LAD (ST depression V_2-V_5). In fact, the patient presents with a tight subocclusion of a big first diagonal with LAD involvement that had opened after thrombolytic treatment. The ST depression in the precordial leads indicates that at this moment, there is no any part of the left ventricular walls with transmural involvement, there being predominantly subendocardial involvement (NSTE-ACS). C, Finally, after PCI, the ECG shows negative T waves only.

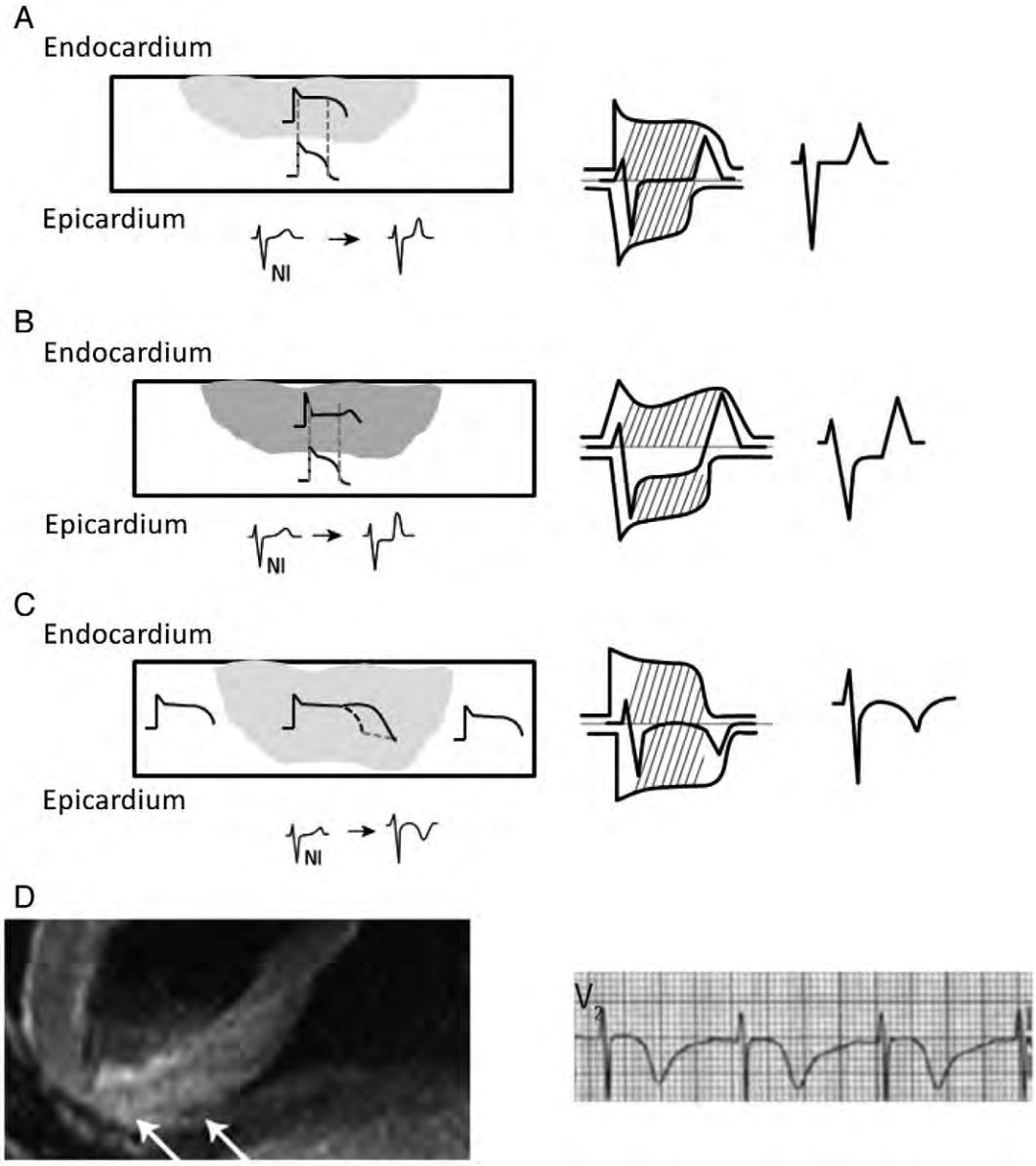

Fig. 8. A, In subendocardial ischemia, the presence of a longer action potential (AP) in the subendocardial area explains the development of peaked and wide T waves. B, If more pronounced subendocardial ischemia exists, the AP of the ischemic zone will exhibit not only a longer duration but also a smaller area. The sum of the 2 APs explains the ECG pattern (ST depression + tall T wave) (see Gorgels et al[15] and Fig. 5B). C, The AP of the involved transmural area is probably due to edema, in view of the deep negative T waves, which occur sometimes in the evolving phase of STE-ACS. The AP is longer but similar in shape to that of the rest of normal left ventricular wall, but the recording electrode faces the tail of the vector of this area and records a negative T wave. The negative T wave may be also explained by the sum of the APs of the damaged zone and the APs of the rest of the LV. D, The ECG pattern in a case of STE-ACS resolution, with the evidence of transmural edema detected by cardiac magnetic resonance.[19] The negative T waves may be explained by the sum of APs of the damaged zone and the APs of the rest of the LV. NI indicates nonischemic. Color illustration online.

ischemia, the AP in the subendocardial area is not only prolonged, but also, its amplitude decreases and its area is smaller[11,15] (Fig. 8B). Consequently, there might be upsloping ST depression and tall T waves (subendocardial injury pattern).[7] This pattern, already described in the 1940s by Dressler and Roesler,[16] evolves without treatment to transmural involvement and Q-wave MI (NSTE-ACS converts to STE-ACS pattern—see later)[7,16] (Figs. 5 and 6). As De Winter et al[7] suggested that these cases that represent 2% of STE-ACS due to LAD occlusion likely require emergency PCI, a measure that is currently not included in the guidelines. In fact, this pattern (ST depression + tall T wave) as a sign of a predominant, very significant LAD subocclusion (NSTE-ACS) is the same pattern previously described in the differential diagnosis of LCX occlusion (STE-ACS equivalent). The degree of ST depression may vary (Figs. 1B3, 5 [right], and 6).

Sclarovsky et al[17] described that the prognosis of patients with horizontal or downslope precordial ST depression and negative T waves (n = 11) was worse than that of patients with positive T waves (n = 21).

Upsloping ST depression has not been considered a sign of ischemia in the past because upsloping ST

depression is commonly seen with tachycardia.[9] In exercise stress tests, we do not consider upsloping ST depression as a sign of ischemia. Nevertheless, when seen with slower heart rates, upsloping ST depression associated with tall T waves may be a manifestation of localized subendocardial ischemia.

D. Small inverted T waves in V_1 to V_3

Small symmetric negative T waves, especially in V_1 to V_3, in the absence of pain, during the course of an ACS, represent a risk for new cardiac events. In fact, flat or negative T waves (≤ 0.2 mV) may be found in patients with a critical LAD stenosis, representing a postischemic ECG change (Fig. 9). An urgent (not emergent, ie, within several hours of presentation) PCI may be recommended in these cases to prevent reischemia/reinfarction. However, T-wave inversion in leads V_1 to V_3 is a normal finding in children and young women, especially African American women.[18] This juvenile pattern (asymmetric negative T wave) should not be confused with post ischemic changes, which usually presents with symmetric negative T waves.

E. Deep negative T waves in the precordial leads

In patients with suspected ACS presenting after resolution of symptoms, deep negative T waves in the precordial leads ($V_{1\text{-}2}$ to V_4-V_5) represent an evolving phase of STE-ACS. This should be considered an atypical transient pattern of STE-ACS (Fig. 1C). It also represents a postischemic change. Because the artery is usually already open or because collateral flow exists, the patient is asymptomatic when the ECG is recorded. However, the ECG may suddenly evolve into pseudonormalization of the negative T waves and even

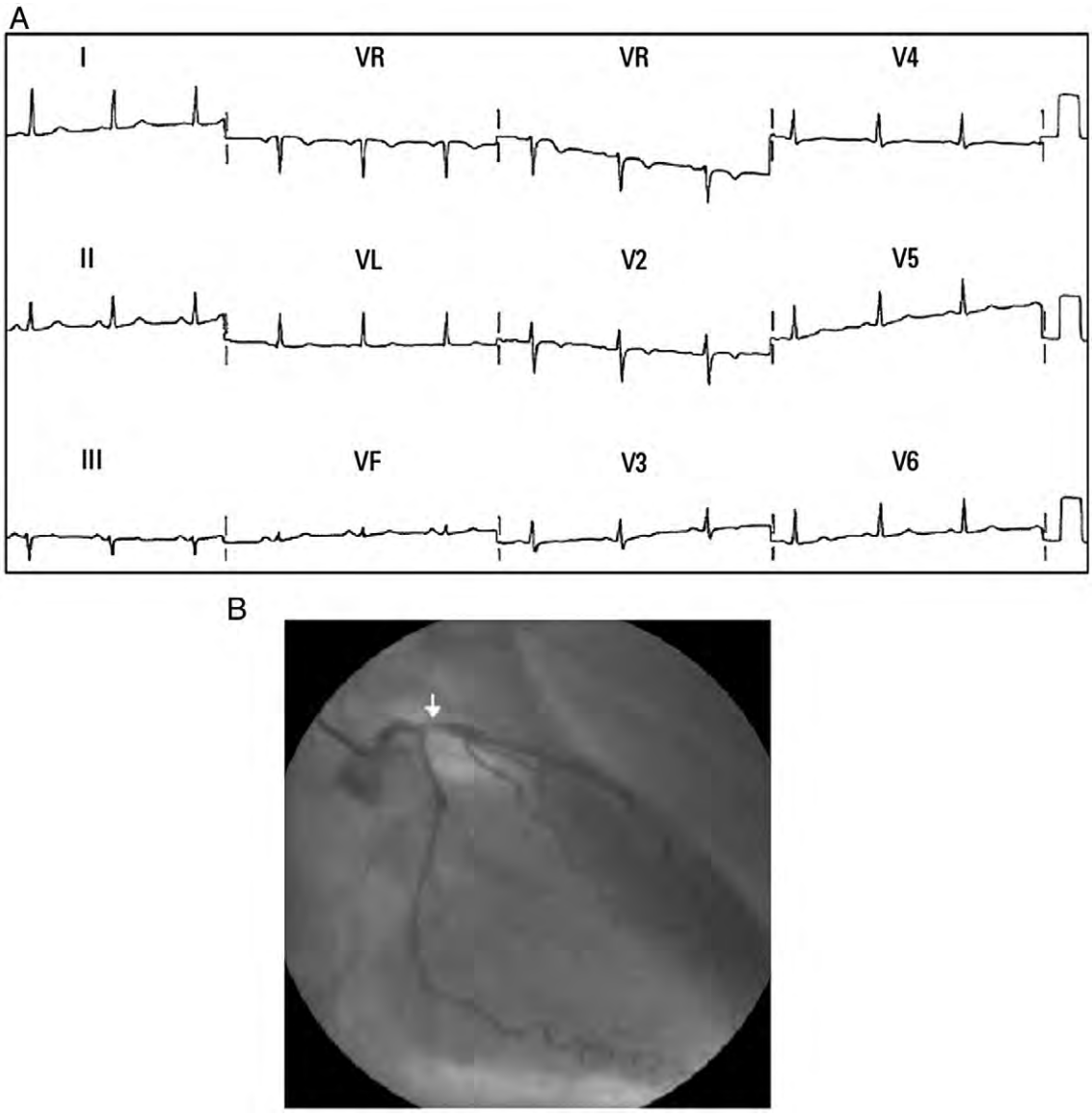

Fig. 9. Electrocardiogram of a 55-year-old man with NSTE-ACS showing symmetric and mild negative T waves from V_1 to V_3. The coronary angiogram shows a significant LAD proximal occlusion.

ST elevation (Fig. 1C). It was recently demonstrated that at least in some cases, there is a transmural edema (transmural area with longer AP—Fig. 8C, D) that disappears when the ECG pattern is normalized.[19]

F. ST elevation in I and aVL

Although this finding traditionally was considered a pattern of high lateral involvement because of occlusion of the LCX, ST elevation in leads I and aVL, when not associated with ST depression in V_1 to V_2, is a manifestation of occlusion of a first diagonal with/without LAD subocclusion that perfuses the mid low anterolateral wall and not the high lateral wall[20] (Fig. 7A). The ST elevation in I and aVL that is caused by LCX occlusion presents with ST depression in leads V_1 to V_2 to leads V_3 to V_4.[21]

G. The diagnosis of LMT occlusion

It has been suggested that ST depression in 6 to 7 or more leads with an ST elevation of 0.1 mV or greater in leads aVR and V_1 (aVR > V_1) is a typical manifestation of ischemia induced by LMT subocclusion or severe 3-vessel disease.[4,22-25] However, conditions other than left main stenosis resulting in diffuse subendocardial ischemia may induce similar changes (eg, left ventricular hypertrophy and aortic stenosis, especially in conditions associated with increase of demand, eg, tachycardia, anemia, fever, elevated blood pressure, etc). Thus, the term *circumferential subendocardial ischemia*, which does not imply specific coronary anatomy, may be more appropriate. In some series of patients with left main stenosis, the prevalence of circumferential subendocardial ischemia ECG pattern is relatively low. For example, Atie et al[26] reported that during episodes of pain, only 60% of the patients had ST elevation in lead aVR and 67% had maximal ST depression in lead V_4. The average number of leads with ST-T changes was only 6.4. Yamaji et al[27] reported that in most of their 16 patients with acute occlusion of the LMT, there was ST elevation in the precordial leads. More recently, Taglieri et al[28] reported that only 24 (42%) of 57 patients with NSTE-ACS due to left main stenosis had ST elevation in lead aVR with ST depression in other leads. It should be remembered that during pain-free episodes, the ECG may be completely normal.[26] The possibility that, in some cases, the ECG was not recorded during active ischemia cannot be excluded because it often happens in patients with NSTE-ACS, and therefore, the ECG may not show the typical pattern.[26] It is important to recognize that this pattern may represent active ischemia due to LMT subocclusion (especially when accompanied by typical clinical symptoms); however, when presentation is atypical, the predictive value for LMT stenosis is lower even if cardiac markers are positive. Moreover, some patients presenting with ACS induced by LMT occlusion have ST elevation and not diffuse ST depression.[4,27,28]

Fiol et al present in this issue of the journal a series of 8 cases of LMT complete occlusion that arrive at the hospital alive with the following clinical and ECG patterns: (*a*) cardiogenic shock/cardiac arrest and (*b*) STE-ACS pattern similar to the one seen in LAD occlusion proximal to the first septal and diagonal branches. However, in LMT infarction with ST elevation, there may not be ST elevation in leads aVR and V_1 (because concomitant ischemia due to LCX involvement counteracts the ST elevation in leads V_1 and aVR induced by the proximal LAD involvement). Another pattern is (*c*) right bundle-branch block and left anterior hemiblock. On the other hand, Yamaji et al[27] reported that ST-segment elevation in aVR with less ST-segment elevation in lead V_1 is an important predictor of acute LMCA obstruction. Thus, the importance of the various signs (ST elevation in aVR, ST elevation in aVR more than in V_1, ST depression in the inferior and anterolateral leads) alone or in combination has yet to be determined. Some investigators consider ST elevation in aVR as a specific and independent predictor of LMT-related ischemia,[27,29] whereas others believe that changes in the ST segments seen in lead aVR are always accompanied by opposite ST deviation in leads II or V_5 to V_6 because these leads are reciprocal.[30]

The previous review by the Committee of the International Society for Holter and Non-Invasive Electrocardiology in 2010 suggested that the pattern of diffuse ST-segment depression in 6 or more leads, often with inverted T waves, is associated with LMT or severe 3-vessel disease–induced ischemia.[4] The current recommendations by the AHA/American College of Cardiology Foundation/Heart Rhythm Society for "resting ECGs that reveal ST-segment depression greater than 0.1mV in 8 or more body surface leads coupled with ST-segment elevation in aVR and/or V_1 but are otherwise unremarkable," is that the automated interpretation should suggest "Ischemia due to multivessel or left main coronary artery obstruction."[31] Both documents do not specify lower accuracy in patients with intraventricular conduction delays, ECG signs of left ventricular hypertrophy with repolarization changes, and the importance of the presenting symptoms.

H. Recognizing changes from NSTE-ACS to STE-ACS ECG patterns and vice versa[11]

The ECG changes of STE-ACS are caused by ongoing transmural homogeneous involvement, usually with a completely occluded epicardial artery, whereas the ECG findings of NSTE-ACS are caused by incomplete transmural homogeneous involvement due to incomplete coronary artery occlusion. If, for any reason, a patient with predominant ST depression (often with a positive terminal T wave) due to NSTE-ACS develops total occlusion (total thrombotic occlusion, coronary spasm, or occlusion of a stent) and there is no collateral circulation/preconditioning, then transmural involvement will develop and the ECG pattern will change from NSTE-ACS to STE-ACS (Figs. 5 and 6).

In contrast, sometimes STE-ACS spontaneously evolves to NSTE-ACS. For example, a patient with ACS presented

with an ECG pattern of STE-ACS (Fig. 7A) (tall T waves in V_1-V_5 and ST elevation in I, aVL, and V_2) with ST depression in aVR and V_1, consistent with an occlusion distal to the first septal branch.[32] Antithrombotic treatment was given, and the patient was transferred to a referral center for emergent coronary angiography. On arrival, partial reperfusion has probably occurred because symptoms abated and the tall T waves in the precordial leads were replaced by ST depression with positive terminal T waves (NSTE-ACS pattern) (Fig. 7B). After PCI of the culprit artery, the ECG normalized, showing only a mild negative T wave, as a sign of complete reperfusion (Fig. 7C).

Conclusions

1. Classification of ACS into 2 groups, STE-ACS and NSTE-ACS, according to the ECG pattern in the acute phase is crucial for proper management. The patients with ACS and ST elevation (STE-ACS) need emergency PCI, whereas in those with NSTE-ACS, the therapeutic strategy depends on the clinical circumstances.
2. ST depression in V_1 to V_4 may be caused by LAD subocclusion (NSTE-ACS) or LCX or distal RCA occlusion (STE-ACS equivalent). There are some subtle ECG changes that may allow differentiation between them.
3. On some occasions, the ECG pattern of evolving STE-ACS in the hyperacute phase shows only peaked positive T waves with or without ST deviation, and in other cases with STE-ACS, spontaneous reperfusion occurs and the ECG may then change to showing negative T waves (reperfusion pattern).
4. The evolving ACS is a dynamic process and, sometimes, may present as an NSTE-ACS pattern but evolves to an STE-ACS pattern or vice versa. As discussed above, if the dynamic nature of the disease is not taken into account, the ECG changes may be misinterpreted.
5. When there are changes in the severity of symptoms (either worsening or resolution), repeated ECG recording may help to detect subtle changes that otherwise could be missed.
6. It is also important, especially in cases with NSTE-ACS, to note whether the ECG was recorded while the

Table 1
More common pitfalls in the ECG interpretation: see ECG pattern, type of ACS and involved artery, zone involved, and recommended management

	ECG patterns	Type of ACS and involved artery	Zone and characteristics of involvement	Management
A	ST depression in V_1-V_4: in the acute phase, prominent ST depression inV_1 without significant terminal positive T wave in V_1-V_2 See Figs. 1A, 3, and 4.	Probably, it is true STE-ACS (STE-ACS equivalent) due to LCX occlusion (rarely distal RCA). Patients have ongoing active symptoms.	Transmural lateral involvement	Emergent PCI
B	Leads V_1-V_4: isoelectric ST segment with tall, wide, positive T wave. Often a transient pattern See Fig. 1B1	Hyperacute phase of STE-ACS. Patient with angina Repeat ECG in few minutes Evolving to LAD total occlusion. Patients usually have ongoing active symptoms.	Anterior subendocardial involvement evolving to transmural involvement	Probably emergent PCI
C	Leads V_1-V_4: ST↓ plus tall positive T wave that evolves to Q wave MI. The change occurs in hours. See Figs. 1B3, 5 (right), 6, and 9B	NSTE-ACS evolving to STE-ACS usually in hours Usually LAD subocclusion evolving to total occlusion Patients usually have ongoing symptom.	Nontransmural involved wall evolving to transmural involvement.	Emergent PCI
D.	Leads V_1-V_3: isoelectric ST segment with mild negative T wave in V_1-V_3 See Fig. 8.	Resolution phase of NSTE-ACS. LAD subocclusion Patients usually have resolution of symptoms.	No transmural involvement	Most probably urgent PCI
E	LeadsV_1 to V_4-V_5: isoelectric ST segment with deep negative T wave. Subacute phase of STEMI once the artery is open without evolving to Q-wave MI. May reocclude if treatment is not started See Fig. 1C.	Resolution phase of STE-ACS due to LAD subocclusion (or occlusion with collaterals) Patients usually have resolution of symptoms.	At least in some cases, transmural edema that disappears if pattern normalizes	Urgent PCI
F	ST elevation in I and VL without ST depression in V_1 V_2 See Fig. 9A.	True STE-ACS usually due to first diagonal occlusion Patients usually have ongoing active symptoms.	Transmural involvement mid/low anterolateral wall	Emergent PCI
G	ST depression in ≥7 leads + ST elevation a VR-V_1	NSTE-ACS LMT (3-vessel/disease) subocclusion	No transmural involvement	Urgent or emergent PCI according to clinical situation
H	ST elevation in I, VL, V_2 to $V_{3\text{-}6}$ No ST elevation a[illegible]nd V_1 Frequent RBBB + superoanterior hemiblock	STE-ACS due to LMT occlusion	Transmural involvement	Emergent PCI

RBBB indicates right bundle-block branch.

patient had active symptoms or after symptoms had already resolved.

7. The presence of small negative T wave, especially in V_1 to V_3, in the course of NSTE-ACS may represent a risk for new cardiac events.
8. Complete occlusion of LMT may produce a typical pattern of STE-ACS, and although the clinical state at arrival is usually critical, some patients may survive after emergent PCI.
9. Finally, having in mind the lack of clear recognition of these possible pitfalls in the current guidelines, we suggest that any new guidelines for treatment of ACS published by Scientific Societies include the recommendations presented in this article, especially in relation to how to differentiate between LCX occlusion and LAD subocclusion in the presence of ST depression in V_1 to V_4. In addition, in cases of STE-ACS equivalent without an ECG pattern of ST elevation, but in the presence of tall positive T waves, especially with ST depression (LAD subocclusion evolving to occlusion), it is recommended that emergent PCI should be considered (see Table 1). Also, currently only horizontal or downsloping ST depression is considered a sign of ischemia, whereas upsloping ST depression is not. We would like to emphasize that upsloping ST depression, especially when associated with tall positive T waves in patients without tachycardia and within the appropriate clinical setting, has to be considered a manifestation of localized subendocardial ischemia.

Limitations

Although some of the ECG patterns and their correlations with coronary anatomy and physiology have been validated by studies (mostly retrospective and post hoc analyses of existing data, collected for different purposes), many of the opinions expressed here are based on clinical experience and electrocardiographic expertise of the authors participated in writing the manuscript. It has been reported that only the 314 of the 2711 recommendations (median, 11%), published in the 16 current ACC/AHA guidelines reporting levels of evidence, are classified as level of evidence A (recommendation based on evidence from multiple randomized trials or meta-analyses), whereas 1246 (median, 48%) are classified as level of evidence C (recommendation based on expert opinion, case studies, or standards of care).[33] Most of the recommendations presented in the current document should be considered as based on level of evidence C. Thus, further studied are needed to directly test these hypotheses and, especially, the predictive value of all these ECG patterns in unselected patients from various ethnicities and origins.

This document is limited to ECGs with narrow QRS (<120 ms). QRS widening due to left ventricular hypertrophy or conduction abnormalities may cause secondary changes in the ST segment and T waves that are not discussed in the present study. Moreover, it is unclear how left ventricular hypertrophy, even without QRS widening), affects the interpretations of these particular ECG patterns.

References

1. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction; a report of the American College Of Cardiology/American Heart Association Task Force on Practice Guidelines (committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). J Am Coll Cardiol 2004;44:E1.
2. Huang HD, Birnbaum Y. ST elevation: differentiation between ST elevation myocardial infarction and nonischemic ST elevation. J Electrocardiol 2011;44:494e1.
3. Birnbaum Y, Zhou S, Wagner GS. New considerations of ST segment "Elevation" and "Depression" and accompanying T wave configuration in acute coronary syndromes. J Electrocardiol 2011;44:1.
4. Nikus K, Pahlm O, Wagner G, et al. Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology. J Electrocardiol 2010;43:91.
5. Bayes de Luna A, MF. The electrocardiography in ischemic heart disease: clinical and imaging correlations and prognostic implications. Oxford, UK: Futura-Blackwell; 2008.
6. Pride YB, Tung P, Mohanavelu S, et al. Angiographic and clinical outcomes among patients with acute coronary syndromes presenting with isolated anterior ST-segment depression: a TRITON-TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition With Prasugrel-Thrombolysis in Myocardial Infarction 38) substudy. JACC Cardiovasc Interv 2010;3:806.
7. de Winter RJ, Verouden NJ, Wellens HJ, Wilde AA. A new ECG sign of proximal lad occlusion. N Engl J Med 2008;359:2071.
8. Porter A, Vaturi M, Adler Y, et al. Are there differences among patients with inferior acute myocardial infarction with ST depression in leads V_2 and V_3 and positive versus negative T waves in these leads on admission? Cardiology 1998;90:295.
9. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. J Am Coll Cardiol 2007;50:2173.
10. From AM, Best PJ, Lennon RJ, Rihal CS, Prasad A. Acute myocardial infarction due to left circumflex artery occlusion and significance of ST-segment elevation. Am J Cardiol 2010;106:1081.
11. Bayes de Luna A. Clinical electrocardiography: a textbook. Chichester, West Sussex, UK: Wiley-Blackwell; 2012.
12. Sagie A, Sclarovsky S, Strasberg B, et al. Acute anterior wall myocardial infarction presenting with positive T waves and without ST segment shift. Electrocardiographic features and angiographic correlation. Chest 1989;95:1211.
13. Franz MR. Long-term recording of monophasic action potentials from human endocardium. Am J Cardiol 1983;51:1629.
14. Burnes JE, Ghanem RN, Waldo AL, Rudy Y. Imaging dispersion of myocardial repolarization, I: comparison of body-surface and epicardial measures. Circulation 2001;104:1299.
15. Gorgels AP. Explanation for the electrocardiogram in subendocardial ischemia of the anterior wall of the left ventricle. J Electrocardiol 2009;42:248.
16. Dressler W, Roesler H. High T waves in the earliest stage of myocardial infarction. Am Heart J 1947;34:627.
17. Sclarovsky S, Rechavia E, Strasberg B, et al. Unstable angina: ST segment depression with positive versus negative T wave deflections—clinical course, ECG evolution, and angiographic correlation. Am Heart J 1988;116:933.
18. Assali AR, Khamaysi N, Birnbaum Y. Juvenile ECG pattern in adult black Arabs. J Electrocardiol 1997;30:87.
19. Migliore F, Zorzi A, Marra MP, et al. Myocardial edema underlies dynamic T-wave inversion (Wellens' ECG pattern) in patients with reversible left ventricular dysfunction. Heart Rhythm 2011;8:1629.
20. Bayes de Luna A, Wagner G, Birnbaum Y, et al. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee

appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation 2006;114:1755.
21. Birnbaum Y, Hasdai D, Sclarovsky S, Herz I, Strasberg B, Rechavia E. Acute myocardial infarction entailing ST-segment elevation in lead aVL: electrocardiographic differentiation among occlusion of the left anterior descending, first diagonal, and first obtuse marginal coronary arteries. Am Heart J 1996;131:38.
22. Kosuge M, Kimura K, Ishikawa T, et al. Predictors of left main or three-vessel disease in patients who have acute coronary syndromes with non–ST-segment elevation. Am J Cardiol 2005;95:1366.
23. Nikus K, Jarvinen O, Sclarovsky S, Huhtala H, Tarkka M, Eskola M. Electrocardiographic presentation of left main disease in patients undergoing urgent or emergent coronary artery bypass grafting. Postgrad Med 2011;123:42.
24. Kosuge M, Ebina T, Hibi K, et al. An early and simple predictor of severe left main and/or three-vessel disease in patients with non–ST-segment elevation acute coronary syndrome. Am J Cardiol 2011;107:495.
25. Nikus K, Pahlm O, Wagner G, Terkelsen CJ, Gettes L. Report of the Third International Society for Holter and Noninvasive Electrocardiology Working Group on improved electrocardiographic criteria for acute and chronic ischemic heart disease—Lund, Sweden: June 2010. J Electrocardiol 2011;44:84.
26. Atie J, Brugada P, Brugada J, et al. Clinical presentation and prognosis of left main coronary artery disease in the 1980s. Eur Heart J 1991;12:495.
27. Yamaji H, Iwasaki K, Kusachi S, et al. Prediction of acute left main coronary artery obstruction by 12-lead electrocardiography. ST segment elevation in lead aVR with less ST segment elevation in lead V(1). J Am Coll Cardiol 2001;38:1348.
28. Taglieri N, Marzocchi A, Saia F, et al. Short- and long-term prognostic significance of ST-segment elevation in lead aVR in patients with non–ST-segment elevation acute coronary syndrome. Am J Cardiol 2011;108:21.
29. Gorgels AP, Engelen DJ, Wellens HJ. Lead aVR, a mostly ignored but very valuable lead in clinical electrocardiography. J Am Coll Cardiol 2001;38:1355.
30. Sclarovsky S, Nikus KC, Birnbaum Y. Manifestation of left main coronary artery stenosis is diffuse ST depression in inferior and precordial leads on ECG. J Am Coll Cardiol 2002;40:575.
31. Wagner GS, Macfarlane P, Wellens H, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part VI: acute ischemia/infarction: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol 2009;53:1003.
32. Engelen DJ, Gorgels AP, Cheriex EC, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. J Am Coll Cardiol 1999;34:389.
33. Tricoci P, Allen JM, Kramer JM, Califf RM, Smith Jr SC. Scientific evidence underlying the acc/aha clinical practice guidelines. JAMA 2009;301:831.
34. Gul EE, Nikus KC. An unusual presentation of left anterior descending artery occlusion: significance of lead aVR and T-wave direction. J Electrocardiol 2011;44:27.