Updated Electrocardiographic Classification of Acute Coronary Syndromes

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Abstract: The electrocardiogram (ECG) findings in acute coronary syndrome should always be interpreted in the context of the clinical findings and symptoms of the patient, when these data are available. It is important to acknowledge the dynamic nature of ECG changes in acute coronary syndrome. The ECG pattern changes over time and may be different if recorded when the patient is symptomatic or after symptoms have resolved. Temporal changes are most striking in cases of ST-elevation myocardial infarction. With the emerging concept of acute reperfusion therapy, the concept ST-elevation/non-ST elevation has replaced the traditional division into Q-wave/non-Q wave in the classification of acute coronary syndrome in the acute phase.

Keypoints: In acute coronary syndrome, in addition to the traditional electrocardiographic risk markers, such as ST depression, the 12-lead ECG contains additional, important diagnostic and prognostic information. Clinical guidelines need to acknowledge certain high-risk ECG patterns to improve patient care.

Keywords: Acute coronary syndrome, classification, ECG, myocardial infarction, ST-segment elevation, ST-segment depression.

INTRODUCTION

In patients with myocardial ischemia caused by decreased blood supply, the initial 12-lead electrocardiogram (ECG) typically shows 1) predominant ST-segment elevation (STE) as part of STE acute coronary syndrome (STE-ACS), or 2) no predominant STE, i.e. non–STE ACS (NSTE-ACS). Patients with predominant STE are classified as having either aborted myocardial infarction (MI) or ST-elevation MI (STEMI) based on the absence or presence of biomarkers of myocardial necrosis. NSTE-ACS patients are classified as having either unstable angina or NSTEMI, based also on the absence or presence of biomarkers of myocardial necrosis.

ECG interpretation is an essential part of the initial evaluation of patients with symptoms suspected to be related to myocardial ischemia (pain, shortness of breath, dizziness, etc), along with focused history and physical examination. Availability of prior ECG tracings and repeated ECG enhances the accuracy of reading. Patients with typical symptoms and ST elevation should be referred for emergent reperfusion therapy (preferentially by primary percutaneous coronary intervention). On the other hand, certain patterns of NSTE-ACS may indicate high risk, and a more aggressive approach should be considered. In a minority of patients with acute total coronary occlusion, no ST elevations are present

in the 12-lead ECG - the importance of recognizing STEMI equivalent ECG patterns has been pointed out [1, 2] (Fig 1). It should be stressed that the ECG findings should always be interpreted in the context of the clinical findings and the symptoms reported by the individual patient, when these data are available.

Classifying ECG changes in ACS may aid in risk stratification of individual patients, but also in the planning of epidemiological and clinical studies to produce comparable data. Different classifications have been proposed by many working groups and committees. Also in textbooks, ACS changes have been classified by the authors. In national, international and universal definitions of MI, ECG classification plays a central role. Classifications of ECG changes in ACS can be based strictly on the changes present in the various ECG leads, but aspects of temporal evolution and location of the disease process within the walls of the heart may be included. Some classifications include aspects of the ECG as a risk indicator. In the following, both well-established and newer, probably less well known ECG classifications will be discussed.

CODING SYSTEMS

The Minnesota Code was developed in the late 1950s in response to the need for reporting ECG findings in uniform, clearly defined, and objective terms and is the most widely used ECG classification system for clinical trials and epidemiologic studies [3]. The Minnesota coding system classifies

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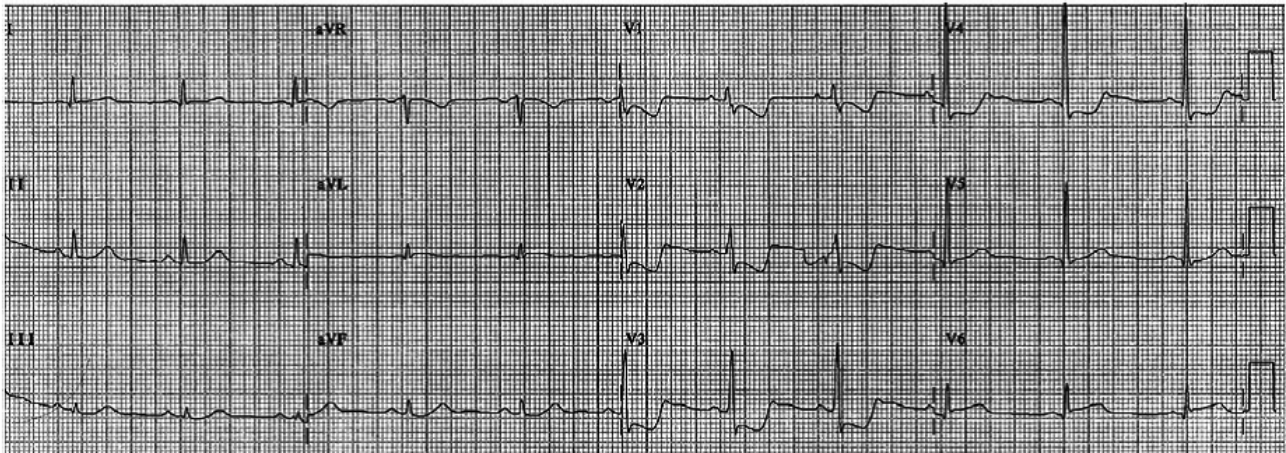
Fig. (1). 12-Lead ECG recorded during chest pain in a patient with acute occlusion of the left circumflex coronary artery. No significant lesions were present in the other coronary arteries. The ECG shows ST depression in leads V1 to V4 and only minor ST elevation, not fulfilling ST elevation myocardial infarction criteria, in leads I, aVL, and V6. (With permission from Elsevier).

changes in the QRS complex, the ST segment and the T wave - those parts of the ECG that are typically involved in ACS. The Selvester QRS scoring system was developed as a method to estimate the total percentage of the left ventricle that is infarcted by using a weighted scoring system [4]. There has been good agreement between the Selvester QRS score and MI size determined by postmortem histopathology in patients with nonreperfused MI, while the agreement with myocardial resonance imaging- (MRI-) determined MI size in reperfused STEMI has been poorer [5].

STANDARD ECG CRITERIA IN THE DIAGNOSIS OF MYOCARDIAL INFARCTION

The World Health Organization (WHO) has played a leading role in the formulation and application of standard criteria for the diagnosis of MI. The WHO Expert Committee in 1959 established the ECG criteria for "very probable" MI, which was mainly based on Q waves with concomitant T-wave changes [6]. In the Joint International Society and Federation of Cardiology/WHO task force statement of 1979, unequivocal ECG changes were the development of abnormal, persistent Q or QS waves and evolving "injury current" lasting longer than one day [7].

In the joint European Society of Cardiology/American College of Cardiology (ESC/ACC) committee consensus document, published in 2000, certain improvements regarding ECG classification were introduced [8]. The J point was specified as the preferred measurement point of ST elevation. Until that, various measurement points had been used in publications and in clinical practice, while in recent years, ST elevations often are measured from the J point. The document also highlighted the need for classification into STEMI and NSTEMI instead of Q-wave and non-Q wave MI in the acute phase, because of the importance of reperfusion therapy in STEMI.

In the Universal definition of MI, published in 2007, the introduction of different cut points for ST elevation in men and women in leads V2-V3 was a clear improvement [9]. Population studies have clearly shown gender differences especially in these leads [10]. Also, cut points for ST depression and T-wave changes were introduced in the document.

In the most recent universal definition of MI, an additional factor – the patient´s age – was introduced [1]. In leads V2-V3 the cut point for J-point elevation is ≥0.2mV in men ≥40 years, ≥0.25 mV in men <40 years and ≥0.15 mV in women, in the absence of left bundle branch block of left ventricular hypertrophy. Importantly, the authors pointed out that lesser degrees of ST displacement or T-wave inversion do not exclude acute myocardial ischemia or evolving MI, since a single static recording may miss the more dynamic ECG changes that might be detected with serial recordings. It was recommended to use supplemental leads such as V3R and V4R (reflecting the free wall of the right ventricle) and V7–V9 (reflecting the inferobasal wall), as well as serial ECG recordings, in patients who present with ischemic chest pain and a non-diagnostic initial ECG.

CLASSIFICATION BASED ON MYOCARDIAL INFARCTION LOCATION

Q-wave Myocardial Infarction

Monumental work in the 1930's and 1940's established the theoretical and empirical basis for association between myocardial necrosis and Q-wave abnormality [11, 12]. Based on anatomical studies, a relationship between the location of infarcted areas and Q waves was accepted and, with minor modifications, implemented in scientific statements and textbooks. According to these studies, Q waves in individual ECG leads correspond to the following myocardial segments: leads V1-V2 to the septal wall, leads V3-V4 to the anterior wall, leads I and aVL to the lateral wall, and leads II, III and aVF to the inferior wall. The mirror pattern (high R wave) in leads V1-V3 was considered as corresponding to the basal part of the inferoposterior wall. Later on, the correlation between Q waves in the right precordial leads and septal MI was questioned, as patients with ST elevation in leads V1 to V3 proved to have an anteroapical MI and a normal septum [13].

The traditional view that Q waves represent transmural MI, while subendocardial MI is reflected by ECG changes other than Q waves, was questioned in the 1980's [14]. Autopsy studies showed that about half of subendocardial MIs generated pathological Q waves, while about half of transmural MIs did not [15]. The terms Q-wave and non-Q wave MI replaced the former designations. MRI studies have shown that the Q-wave/non-Q wave distinction is determined rather by the total size than by the transmural extent of the underlying MI [16].

In a committee statement, Bayés de Luna *et al.* proposed a new terminology for left ventricular walls and location of Q-wave MIs based on the cardiac MRI [(17]. According to the authors, the concordance between the ECG patterns and the location of MI by MRI shows that abnormally increased R waves, the Q-wave equivalent, in leads V1 and V2 indicate a lateral MI and that abnormal Q waves in leads aVL and I without a Q wave in lead V6 indicate a mid-anterior MI. Therefore, it was recommended that the terms posterior and high lateral MI be replaced by lateral wall MI and mid-anterior wall MI, respectively (Fig. **2**).

ST Elevation and Non-St Elevation Myocardial Infarction

Since the introduction of coronary angiography, echocardiography, myocardial scintigraphy, cardiac MRI, and the

Type of MI		Infarction area (CMR)	ECG pattern	Name given to MI	Most probable place of occlusion
A N T E R O S E P T A L Z O N E	A1		Q in V1–2 SE: 86% ES: 98%	Septal	LAD
	A2		Q in V1–2 to V4–V6 SE: 86% ES: 98%	Apical anteroseptal	LAD
	A3		Q in V1–2 to V4–V6 VL and sometimes I SE: 83% ES: 98%	Extensive anterior	LAD
	A4		Q (qs or r) in VL and sometimes I, V2–3 SE: 70% ES: 100%	Limited anterior	LAD
I N F E R O L A T E R A L Z O N E	B1		Q (qr or r) in I, VL, V5–6 and/or RS in V1 SE: 50% ES: 98%	Lateral	LCX
	B2		Q in II, III, VF SE: 87.5% ES: 98%	Inferior	RCA LCX
	B3		Q in II, III, VF (B2) + Q in I, VL, V5, 6 and / or RS in V1 (B1) SE: 70% ES: 100%	Inferolateral	

Fig. (2). ECG patterns of Q-wave/Q-wave equivalent myocardial infarction on the 17-segment model of the left ventricle, with the names given to the infarcted left ventricular regions documented by cardiac myocardial resonance imaging. The 4 MI regions in the anteroseptal zone are designated as A1, A2, A3, and A4; the 3 MI regions in the inferolateral zone are designated as B1, B2, and B3. (With permission from Elsevier).

emerging concept of acute reperfusion therapy, many studies have dealt with the issue of the correlation of the ECG changes with coronary anatomy and the size and location of the ischemic region in the acute phase of ACS. Especially in STE-ACS, the ECG recorded during the symptomatic phase contains clinically important information regarding the culprit artery, the size of the ischemic area, and the "severity" of myocardial ischemia [18, 19]. On the other hand, in NSTE-ACS, the information is more limited. The algorithms developed to identify the culprit artery and the level of occlusion with respect to side branches are based on the injury vector induced by transmural myocardial ischemia. The direction and displacement of the ST segment is determined by the sum of direction and magnitude of all ST vectors at a certain time point, for example at the J point. The resulting main vector will point in the direction of the most pronounced ischemia resulting in ST elevation in the ECG lead(s) reflecting that particular area. The anatomically opposite area(s) will record ST depression. By correlating the ST elevations and depressions in the different leads with the myocardial segments reflected by the leads, anatomical information can be gained. This vectorial concept is particularly useful when analysing the frontal plane leads [20]. The anatomical "information" of the ECG in STE-ACS is especially useful in geographical regions with long transport distances and limited access to primary PCI.

Grades of Ischemia

The concept of "grades of ischemia" was introduced by Sclarovsky and co-workers in the 1990's [21]. According to this concept, severe transmural ischemia affects the terminal portion of the QRS complex, which is manifested as a decrease in S-wave amplitude in leads with a terminal S wave and an elevation of the J point >50% the height of the R wave amplitude in leads with qR configuration. Patients with significant terminal QRS distortion (Sclarovsky-Birnbaum grade 3 of ischemia) have more severe ischemia, will have less myocardial salvage despite successful recanalization of the epicardial coronary arteries, and have poorer prognosis, compared to patients with STE-ACS without terminal QRS distortion (Sclarovsky-Birnbaum grade 2) [22, 23]. It was recently shown that grade 3 ischemia on the admission ECG during STEMI, is closely associated with the development of severe microvascular damage on cardiac MRI [24].

ECG CLASSIFICATION BASED ON TEMPORARY EVOLUTION

The duration of the ischemic process is an important factor that determines the ECG pattern observed in an individual patient with acute MI. Anderson, Wilkins and colleagues developed an "ECG acuteness score" to augment the historical timing of acute symptom onset in guiding the clinician about the potential for using reperfusion therapy to achieve myocardial salvage [25]. In one study, the Anderson-Wilkins acuteness score, combined with the historical estimation of symptom duration, seemed to provide a more accurate basis for predicting the potential for limitation of final MI size than either method alone [26].

Another classification based on simple ECG parameters – the Q wave, the ST segment and the T wave – uses the concepts of preinfarction syndrome and evolving MI [18] (Fig. 3). The preinfarction syndrome - ST elevation accompanied by positive T waves - is the initial manifestation of acute regional transmural myocardial ischemia, which occurs before the development of MI. This stage of the disease process represents the window of opportunity for reperfusion therapy before irreversible myocardial damage develops. In evolving MI, myocardial necrosis has occurred and the ECG
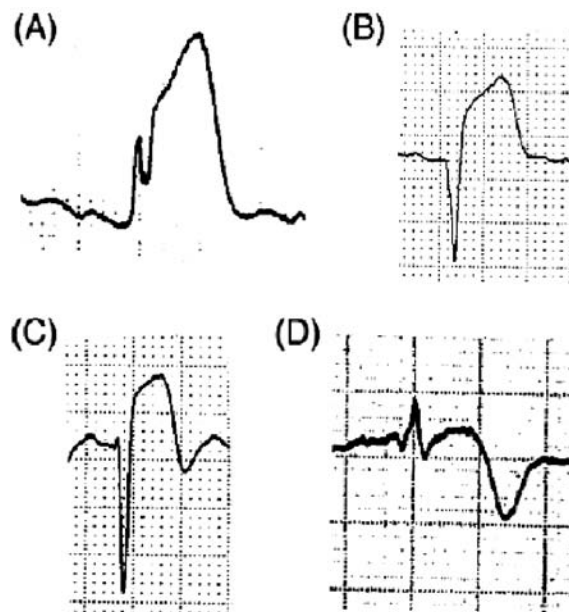
Fig. (3). The ECG patterns of the preinfarction syndrome and evolving myocardial infarction. (**A**) The preinfarction syndrome: an elevated ST segment and a peaked T wave; (**B**) Evolving myocardial infarction without ECG signs of reperfusion: a deep Q wave, an elevated ST segment and a positive T wave; (**C**) Evolving myocardial infarction with incomplete reperfusion: ST elevation, a biphasic T wave (negative terminal portion); (**D**) Evolving myocardial infarction with complete reperfusion: minor ST elevation, negative T wave. (With permission from Oxford University Press).

shows Q waves and/or inverted T waves. In a large Danish Trial in Acute Myocardial Infarction-2 (DANAMI-2) study, the evolving MI pattern was independently predictive of adverse outcome in multivariable analysis at median 2.7 year follow-up [27]. In patients with anterior evolving MI without ECG signs of reperfusion (no inverted T waves), superiority of primary PCI compared with thrombolytic therapy was driven by a 51% reduction in the relative risk of composite endpoint.

NEED FOR RE-CLASSFICATION OF ST-ELEVATION- AND NON-ST ELEVATION ACUTE CORONARY SYNDROME?

A recent working-group statement questioned the definitions behind the prevailing ECG classification of STE- and non-STE-ACS (2). A classification based on the pathophysiologic processes involved was recommended instead of strict classification based on the ECG findings per se. The authors pointed out that the ECG pattern in ACS changes over time and may be different if recorded when the patient is symptomatic or after symptoms have resolved. An early normal ECG recorded when the patient is asymptomatic usually becomes abnormal during pain, and on the contrary, a very abnormal ECG, recorded during an asymptomatic period, may "pseudonormalize" during pain (Fig. **4**). Temporal changes are most striking in cases of STEMI. The ECG pattern will be different if the ECG is recorded within minutes to hours from the onset of the ischemic process in comparison to several hours to days later. Accordingly, in case of strict reliance on standard MI criteria, patients with identical pathophysiologic processes may be classified into different operational clinical ACS categories. For example, if a patient has sudden thrombotic occlusion of the left anterior descending coronary artery (LAD), and the ECG is recorded during the occlusive phase, the ECG will show ST elevations in the precordial leads and the patient is classified as having STE-ACS. However, if the coronary flow is spontaneously or therapeutically reestablished before the first ECG is recorded, a deep inverted T wave may be present in leads V1-V3/V4 and, according to standard ECG classification the

patient will have an initial diagnosis of NSTE-ACS, although the ECG pattern represents an evolutionary phase of STE-ACS (Fig. **4**).

ST-ELEVATION ACUTE CORONARY SYNDROME

In the working group statement, apart from ST elevation, also two other ECG patterns were regarded as STE-ACS [2]. In transmural ischemia of ACS, ST elevation is usually followed by T-wave inversion. In a patient, who presents with inverted T waves in the precordial leads V1/V2-V3/V4 (leads with predominantly rS-configuration), standard criteria recommend using NSTE-ACS as the initial diagnosis. However, T-wave inversion without concomitant ST depression is almost never recorded during the symptomatic ischemic phase, but instead during the "post-ischemic" reperfusion phase—spontaneous or induced by reperfusion therapy—of an anterior STE-ACS. It seems clinically relevant to classify these cases as STE-ACS, because in case of spontaneous reperfusion, there is a potential for impending reocclusion of the artery. After thrombolysis or primary PCI, this ECG pattern indicates successful reperfusion. The working group also recommended classifying "hyperacute" prominent T waves, which usually persist for only a brief period in acute coronary artery occlusion, as STE-ACS. However, as pointed out in the statement, there are many clinical situations, other than ischemic heart disease, such as hyperkalaemia, early repolarization, and ventricular hypertrophy, which may present tall and peaked positive T waves. Also, persistent tall T waves may be the expression of a mirror pattern of chronic lateral wall myocardial ischemia.

NON-ST ELEVATION ACUTE CORONARY SYNDROME

Acute subendocardial ischemia due to partial reduction of flow and/or increase of demand causes ST depression in leads facing the involved zone. ST depression that is typical for NSTE-ACS may be caused by circumferential (global) or regional subendocardial ischemia [2, 18]. ST depression during anginal pain present in six or more leads, often with in-
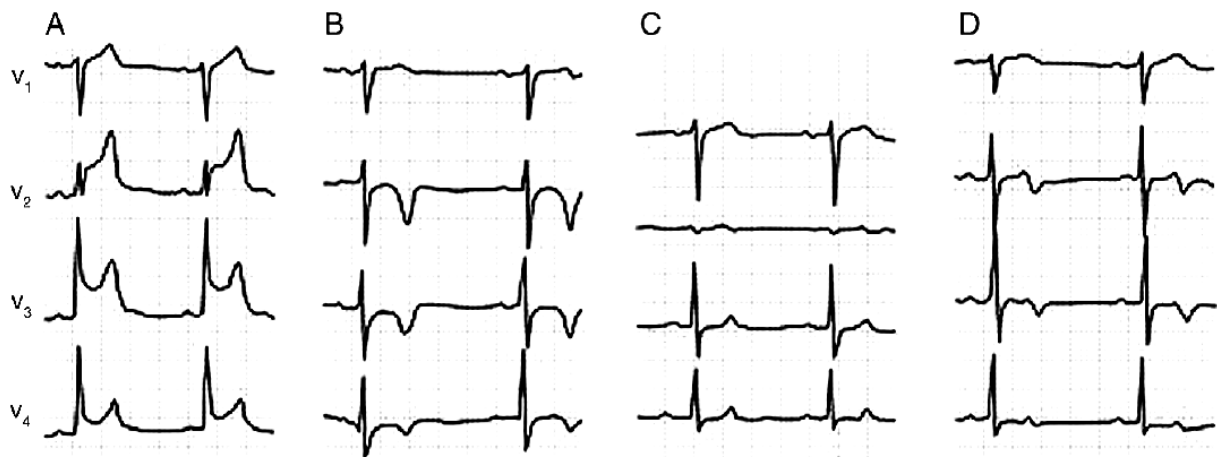

Fig. (4). Four consecutive ECGs (precordial leads V1 to V4) of a patient with an acutely occluded left anterior descending coronary artery. (**A**) Typical ST elevation during the occlusive phase; (**B**) ST resolution with ("post-ischemic") T-wave inversion representing myocardial reperfusion; (**C**) Re-occlusion manifests as "pseudonormalization" of the T waves; (**D**) Reappearance of inverted T waves when myocardial ischemia has subsided. (Technical error in lead V2 in "C"). (With permission from Elsevier).

verted T waves, has been associated with autopsy-proven subendocardial MI and with left main-, left main equivalent-, or severe 3-vessel disease. This ECG pattern of circumferential subendocardial ischemia also encompasses ST elevation in lead aVR and was present in 8% of consecutive patients with ACS admitted to a university hospital [28]. ACS patients with ST depression in six or more leads, maximal in leads V4 to V6, especially when associated with inverted T waves and ST elevation in lead aVR, should have high priority for urgent invasive evaluation because of high probability of severe angiographic coronary artery disease (Fig. **5**). The probability for severe coronary artery disease is higher if the patient's baseline ECG is normal and the changes are dynamic. However, it must be pointed out that the ECG pattern of diffuse ST depression with inverted T waves in the lateral precordial leads and concomitant ST elevation in lead aVR can be seen in various other clinical situations associated with an increased left ventricular end-diastolic pressure, such as rest angina with sinus tachycardia and chronic MI with restrictive remodeling. The same ECG pattern may also be present in structural heart disease with left ventricular remodeling, such as valve disease and cardiomyopathy.

A recent paper highlighted the obvious risks for false prediction of severe coronary artery disease by the ECG when interpretation is done without access to clinical data [29]. The authors questioned recommendations by the American Heart Association/ACC Foundation/Heart Rhythm Society, which state that in "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 V1 but are otherwise unremarkable", the automated interpretation should suggest "ischemia due to multivessel or left main coronary artery obstruction" [30]; the recommendations do not mention symptoms during ECG recording.

In general, the ECG manifestations of regional subendocardial ischemia are less well defined than those of circumferential subendocardial ischemia or of transmural ischemia. Up-sloping ST depression with positive T waves is commonly seen during tachycardia, even in persons without coronary artery disease and is usually not considered a pattern indicated ischemia. However, the same pattern may be seen in patients with NSTE-ACS at slower heart rates and is increasingly recognized as a sign of regional subendocardial ischemia [18, 31] (Fig. **6**). It has been suggested that when present in the precordial leads, this pattern points to subtotal occlusion of the LAD or total occlusion of a coronary artery side branch with resulting subendocardial ischemia. However, ST depression with positive T waves may be present in regional transmural ischemia caused by total occlusion of the left circumflex or right coronary artery at the evolving MI stage with inverted T waves (mirror-image of ST elevation and a negative T
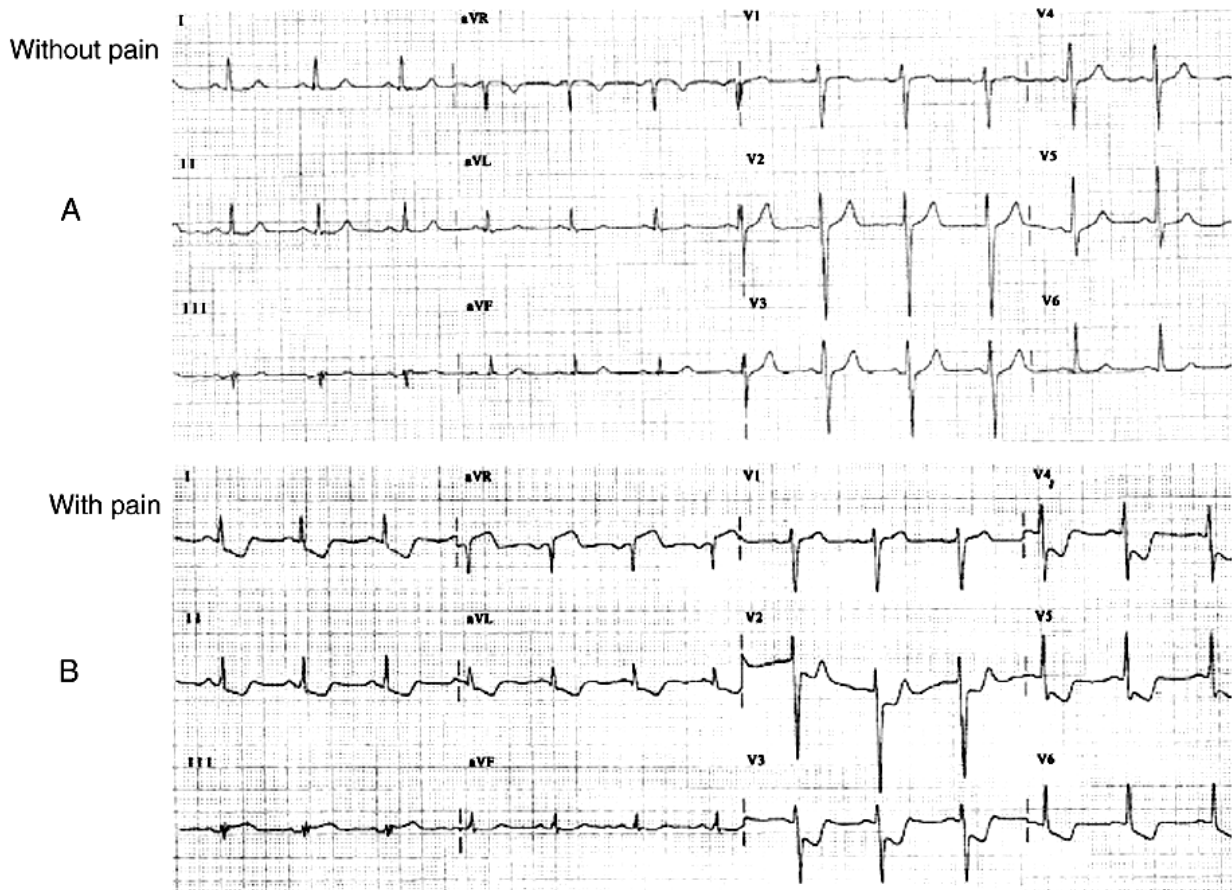
Fig. (5). In a patient with left main coronary artery stenosis the ECG is normal when the patient is asymptomatic (**A**). During chest pain, the ECG pattern of circumferential subendocardial ischemia – widespread ST depression with inverted T waves and lead aVR ST elevation – is present (**B**). (With permission from Elsevier).
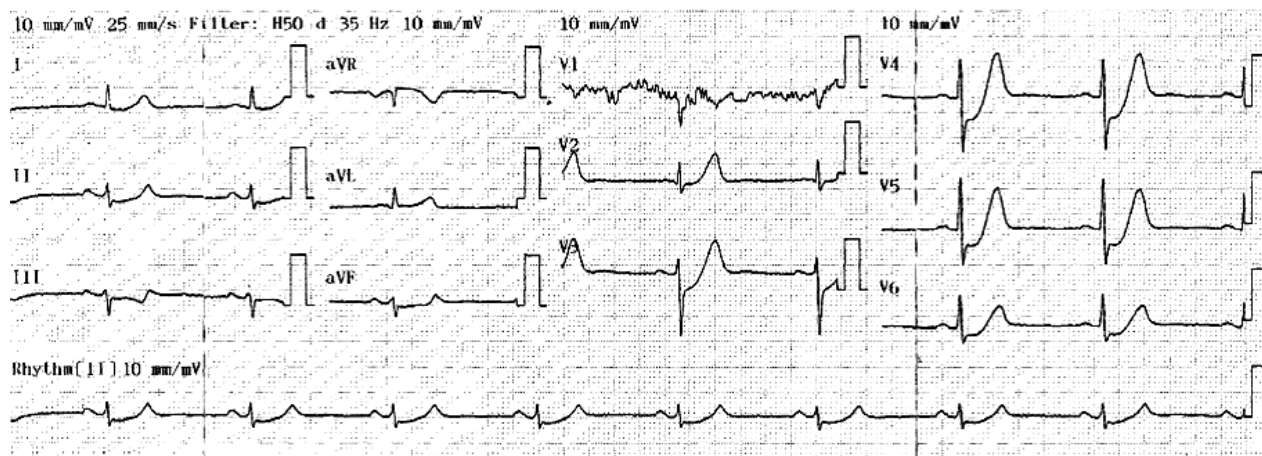
Fig. (6). In a patient with acute coronary syndrome, 12-lead ECG shows diffuse ST depression with positive T waves in the inferior leads II, III and aVF, and in leads V2-V6 besides ST elevation in lead aVR. Coronary angiography showed a subtotal occlusion with impaired flow of the left anterior descending coronary artery (Technical artifact in lead V1). (With permission from Elsevier).

wave of reperfusion). It should be remembered that this ECG pattern may be unstable and may evolve to ST segment elevation if the culprit artery progresses to total occlusion or there are changes in collateral circulation.

CONCLUSION

ECG findings in ACS should always be interpreted in the context of the clinical findings and the symptoms reported by the individual patient, when these data are available. It is also extremely important to acknowledge the dynamic nature of ECG changes in ACS. The ECG pattern changes over time and may be different if recorded when the patient is symptomatic or after symptoms have resolved. An early normal ECG recorded when the patient is asymptomatic usually becomes abnormal during pain, and on the contrary, a very abnormal ECG, recorded during an asymptomatic period, may "pseudonormalize" during pain.

In a minority of patients with acute total coronary occlusion, no ST elevations are present in the 12-lead ECG. ST depression in leads V1-V3/V4 may represent a STEMI-equivalent ECG pattern, indicating need for immediate reperfusion therapy. In patients who present with ischemic chest pain and a non-diagnostic initial ECG, it is recommended to use supplemental leads such as V3R and V4R (reflecting the free wall of the right ventricle) and V7–V9 (reflecting the inferobasal wall).

T-wave inversion without concomitant ST depression is almost never recorded during the symptomatic ischemic phase, but instead during the "post-ischemic" reperfusion phase—spontaneous or induced by reperfusion therapy—of an anterior STE-ACS. It seems clinically relevant to classify these cases as STE-ACS, because in case of spontaneous reperfusion, there is a potential for impending reocclusion of the artery.

Patients with a clinical picture compatible with ACS, who have ST depression in six or more leads, maximal in leads V4 to V6, especially when associated with inverted T waves and ST elevation in lead aVR, should have high priority for urgent invasive evaluation because of high probability of severe angiographic coronary artery disease. The prob-

ability for severe coronary artery disease is higher if the patient's baseline ECG is normal and the changes are dynamic.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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