TOPICAL REVIEW

The role of the initial 12-lead ECG in risk stratification of patients with acute coronary syndrome

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Abstract

This article reviews the prognostic significance of the initial 12 lead ECG in acute coronary syndrome (ACS). In patients with non ST segment elevation ACS, the initial ECG may vary from a normal one to an ECG which demonstrates T wave inversion and ST segment deviation. Patients, who present with either a normal ECG or T wave inversion in less than 5 leads, are considered to be at low risk. Those patients who have ST segment depression or a combination of ST segment depression and elevation have the highest incidence of cardiac death, re-infarction and recurrent chest pain.

In patients with ST segment elevation ACS, the mortality and morbidity is mostly influenced by infarct size. The ECG findings which correlate with infarct size are 1) the degree and extent of ST segment elevation, 2) the infarct related coronary artery and 3) distortion of the terminal portion of the QRS complex. Patients with acute anterior wall myocardial infarction due to a proximal occlusion of the left anterior descending (LAD) coronary artery have the worst short and long-term prognosis. The ECG manifestation of a proximal left anterior descending artery occlusion is ST segment elevation in lead aVL and the precordial leads, combined with ST segment depression in the inferior leads.

With regard to patients who have an acute inferior wall myocardial infarction, the patients at highest risk are those with a proximal right coronary artery (RCA) occlusion artery and posterolateral extension. The ECG findings in proximal right coronary artery occlusion are ST segment in the inferior leads and in V4R. Less frequently ST segment elevations may be present in lead V1and V2. Patients with posterolateral wall extension can be identified by the presence of ST segment depression in the right precordial leads.

Finally, distortion of the terminal portion of the QRS complex is an important indicator of poor outcome. ***(Fig. 3, Ref. 42.)***

Key words: initial 12-lead ECG, risk stratification of patients with acute coronary syndrome.

An acute coronary syndrome (ACS) is caused by disruption of an atheromatous plaque with superimposed thrombosis and distal embolization (1). An incomplete and transient occlusion causes unstable angina (UA) or non-Q wave myocardial infarction (NQMI) (1—3). Because of common pathogenesis and clinical presentations, patients with UA and NQMI are also classified as non-ST segment elevation ACS (3). These patients are treated with heparin, beta-blockers, aspirin and glycoprotein IIb/IIIa antagonists (2, 3). Complete interruption of coronary artery blood flow for more than 30 minutes causes an ST segment elevation ACS which usually result in a Q wave myocardial infarction (2, 4). However, less frequently, the ST segment elevation is transient and not followed by the development of Q waves. Patients with ST segment elevations are candidates for reperfusion therapy (4). Because these interventions, specifically, thrombolysis, glycoprotein IIb/IIIa antagonists or percutaneous coronary intervention, are expensive and risky, it is important to identify

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This work is dedicated to RNDr. Vavrinec Szathmary.

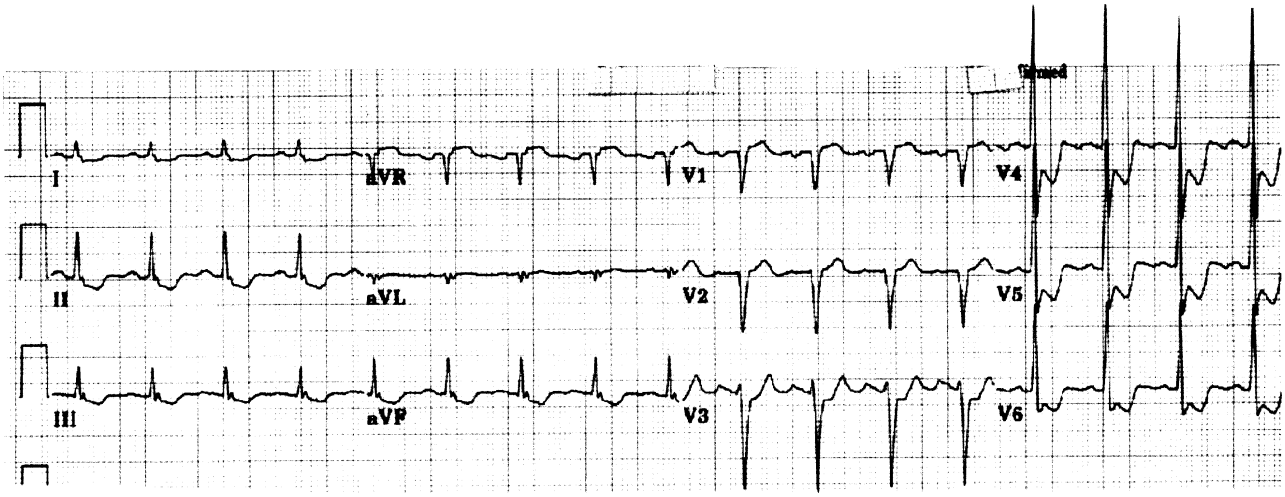

Fig. 1. The 12-lead during chest pain in a patient with documented severe proximal three-vessel disease. There is ST segment depression in all limbs and chest leads except lead V1, V2. The ST segment in lead aVR is elevated.

patients in whom the benefits of such a strategy will outweigh the risk. The 12 lead ECG is crucial for making that determination. The aim of this article is to review the role of the initial 12 lead ECG in the risk stratification of ACS.

Non-ST segment acute coronary syndrome

Patients with non-ST segment elevation ACS are a heterogeneous group. Using both clinical factors as well as ECG criteria they can be stratified into high, intermediate and low-risk groups (3, 5). The high risk patients are those with ST segment deviations, intermediate risk patients with T wave inversion, and low risk patients have normal ECG or nonspecific changes.

The non-ECG criteria for high risk are: age over 65, documented coronary artery disease, chest pain in the preceding 24 hours and abnormal biochemical markers for myocardial necrosis (2, 5).

Normal or non-diagnostic ECG. An initially normal or non-diagnostic ECG can be seen in patients with a small area of ischemia, necrosis due to branch disease or occlusion of the left circumflex coronary artery (LCF) (6, 7). Additionally a normal initial ECG may be the result of delayed evolution of the ST-T and Q wave changes. The majority of patients with a normal or non-diagnostic initial ECG have a good short and long-term prognosis. According to Cannon et al. (8), 60 % of patients with a non ST elevation ACS will have a normal ECG and the one-year mortality rate in these patients is 8.2 %. Nyman et al. (9) reported similar results. In patients who have a delayed ECG evolution of an acute myocardial infarction (AMI), the prognosis depends on the infarct location and size (6, 7). Kontos et al. (6) using myocardial perfusion imaging with technetium-^{99m} sestamibi suggested that some patients with a non-diagnostic initial ECG have the same amount of myocardium at risk as those with abnormal ECG.

Isolated T wave inversion. This group comprises patients with isolated 1 or 2 mm T wave inversions or pseudo normalized T waves. The prognostic significance of this ECG finding is not completely clear. According to Cannon et al. (8), the incidence of isolated T wave changes in patients with ACS is 20.5 % with one-year mortality of 6.8 % which is similar to those patients with a normal ECG. On the other hand, Holmvang et al. (1) suggested that patients with T wave inversions in five or more leads have the same outcome as those patients who have ST segment depression (see below). In a subsequent study, reported by the same authors (11), the 30 day outcome in patients with isolated T wave inversions was similar to that of patients with a normal ECG. That study however did not report the number of leads, which had T wave inversions. In the study of Nyman et al. (9), the incidence of isolated T wave inversions in ACS patients was 31 % and the one year rate of death and non fatal myocardial infarction almost twice that of patients who had a normal ECG. Finally, Savonitto et al. (12) compared the prognostic significance of T wave inversion, ST segment depression and ST segment elevation in a cohort of 12,142 patients who were enrolled in the GUSTO II study. When compared to patients with ST segment deviation, patients with isolated T wave inversions had the highest incidence of non-obstructive coronary artery disease, and the lowest incidence of three vessel disease They also had the lowest 30 day and 180-day mortality and re-infarction rates. However, there was no difference in the need for revascularization, the incidence of previous cardiac events and cardiac risk factors between patients with isolated T wave inversions and those with ST segment depression or elevation.

ST segment depression. Patients with ST segment depression have the highest incidence of death, re-infarction and recurrent myocardial ischemia (8—17). The ST segment deviation is measured 60 or 80 ms after the J point. Depending on the criteria (0.5 or 1.0 mm), the incidence of ST segment depression in ACS

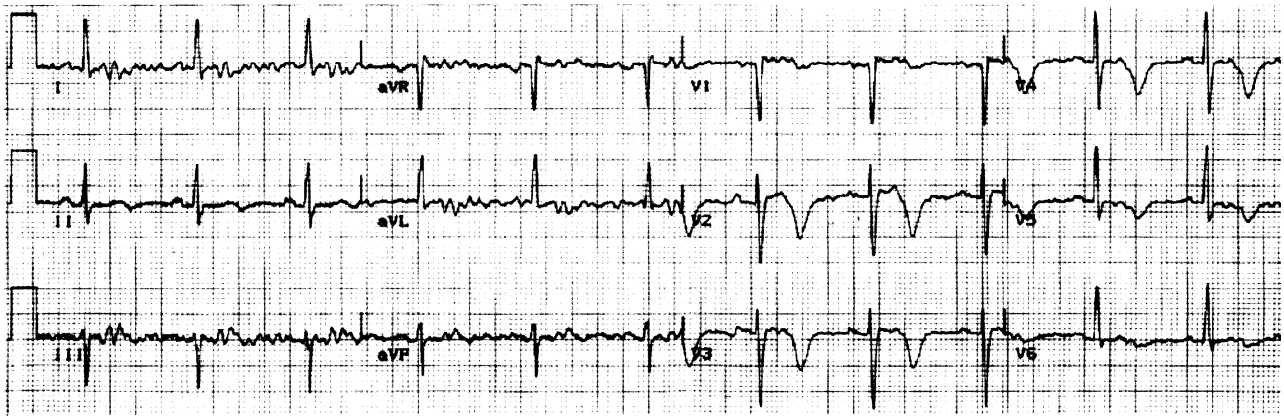

Fig. 2. The 12-lead ECG during chest pain showing mild ST segment elevation in lead V1—3 and deep T wave inversion in lead V2—V5. Coronary angiography revealed 90 % proximal LAD disease and 85 to 90 % RCA and LCF obstruction respectively.

varies from 12.4 % to 46 % (8, 16). The 30-day rate of mortality and non-fatal myocardial infarction ranges between 3.6 % to 16 %. At 60 days the mortality rate was 15 % and at one year it was 9.8—22 %. According to Hyde et al. (16), the one year mortality or AMI rate in patients with 0.5 mm, 1 mm and >2 mm ST segment depression was 12 %, 15 % and 41 % respectively. Holmvang et al. (10) found a similar correlation between the degree of ST segment depression and clinical outcome. In addition, the prognosis is the worst in patients with combined ST segment depression and T wave inversion (18). In comparison, the ST-segment elevation in the lateral leads has similar outcome as patients with normal ECG (9).

About 50 % of patients with ST segment depression have elevated serum cardiac markers. According to Savonitto et al. (12) the 30 day mortality or re-infarction rate in patients with ST segment depression who have normal CPK was 9.7 % whereas the rate among those who had an increased CPK 16.7 %. Similar differences were also observed at six months (14.6 % and 21.7 %). As suggested by Cannon et al. (19), the diagnosis of NQMI should be suspected in patients with new ST segment deviation (elevation or depression >1 mm in two contiguous leads), chest pain for 60 minutes or more, and absence of prior percutaneous coronary intervention and recent onset of angina pectoris.

The ECG is also helpful to identify patients with significant left main or three-vessel disease (20). As seen in figure 1 there is diffuse ST segment depression and ST elevation in lead aVR due to extensive proximal three vessel disease. These patients are candidates for early coronary angiopgraphy and revascularization.

In summary, ACS patients with ST segment depression have the worst short and long term outcome because of the higher incidence of NQMI, and three vessel coronary artery disease. They also tend to have worse left ventricular function, a history of previous myocardial infarction, and a longer history of angina pectoris. These patients are also more likely to have had prior angioplasty or bypass surgery (6—17).

ST segment elevation acute coronary syndrome

Patients with ST segment elevation on the initial ECG can be divided into two groups. The first one is those patients with transient ST segment elevation who does not develop abnormal Q waves. They are classified as having UA or NQMI. The incidence of transient ST segment elevation in patients with ACS is approximately 10 %. Their outcome is similar to patients with ST segment depression. The patients with the worst prognosis have a combination of ST segment elevation and depression (9). An important subgroup are patients who have ischemic type chest pain, mild ST segment elevation and deep T wave inversion in lead V2—4. This is often due to severe LAD obstruction (21) (Fig. 2). These patients should undergo early coronary arteriography to identify those patients with proximal LAD disease.

The majority of patients with ST segment elevation develop a Q wave myocardial infarction. The main determinant of short and long term prognosis is the amount of myocardium at risk and final infarct size (22, 23). The concept of myocardium at risk and final infarct size is important for risk stratification as well as for the assessment of various therapeutic interventions. According to Christian et al. (22) the final infarct size depends on 1) the ischemic area, 2) collateral circulation, 3) myocardial metabolic demand and 4) the duration of complete occlusion. The following ECG findings are useful in assessing infarct size.

The ST segment score. The degree and extent of ST segment elevation is one of the oldest noninvasive techniques for estimating infarct size. Aldrich et al. (23) developed a simple ST segment score for predicting myocardium risk. They suggested that in patients with an anterior wall acute myocardial infarction the number of leads involved correlated with the amount of myocardium at risk and final infarct size. On the other hand, for inferior wall acute myocardial infarction, it was the sum of the voltage of ST segment elevation, which was predictive of myocardium at risk and final infarct size. However, the prognostic value of the ST segment score has certain limitations. As reviewed by Holland and Arnsdorf (24) the degree and extent of ST segment ele-

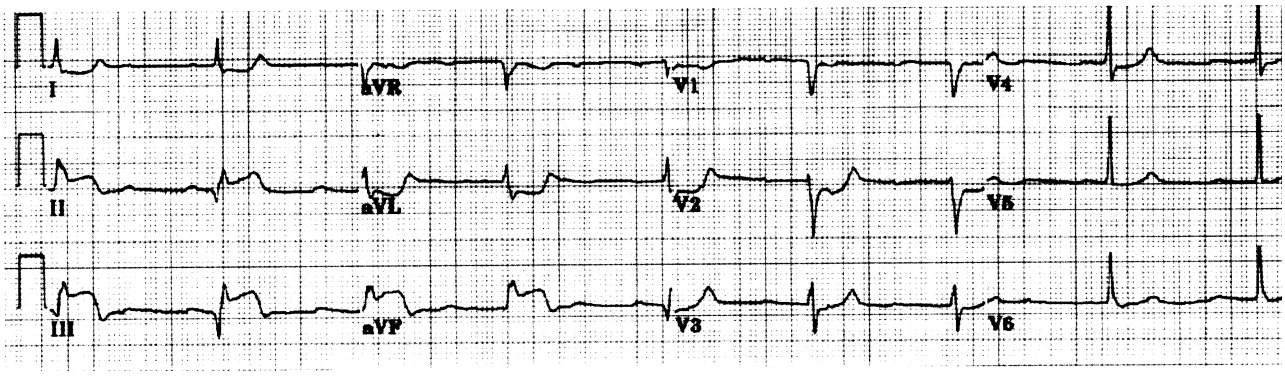

Fig. 3. The 12-lead ECG in a patient with inferior wall acute myocardial infarction. The underlying rhythm is sinus with third degree AV block and AV junctional escape rhythm. Note prominent ST segment elevation in leads II, III and aVF starting immediately after the peak of the R wave. In addition there is ST segment depression in leads I, aVL, V2—4. Coronary angiography showed severe three vessel disease with complete occlusion of the mid RCA and 85 and 90 % proximal LAD and LCF obstruction.

vation is influenced not only by the ischemic area itself but also by: 1) the boundary between ischemic and nonischemic myocardium, 2) the distance between the leads and myocardial necrosis and 3) the non-spatial effects of transmembrane potential.

The results of studies correlating the ST segment score with myocardium at risk and final infarct size were not uniform. Aldrich et al. (23) using the Selvester QRS score for infarct size found good correlation between ST segment score and final infarct size. However, this was not confirmed by Birnbaum et al. (25) using the same approach. Christian et al. (22) compared the ST segment score with technetium^{99} Sestamiby imaging and found only a weak association between myocardium at risk and the ST segment score. There was however better correlation between the ST segment score and collateral flow.

Infarct related artery. The identification of the infarct related artery and the occlusion site is another approach used to estimate the extent of myocardial necrosis. In the case of an acute anterior wall myocardial infarction a proximal LAD occlusion causes larger infarcts than distal one. The ECG criteria for proximal LAD occlusion (before the first septal or diagonal branch) are: 1) ST elevation in leads I, aVL and the precordial leads and 2) ST segment depression in the inferior leads (26). In patients with a distal LAD occlusion, ST segment elevation is limited to the precordial leads with isoelectric ST segments in the inferior leads (26). In addition, ST elevation in both the anterior and inferior leads usually indicates a mid LAD occlusion (27).

In patients with an inferior wall acute myocardial infarction the culprit coronary artery is either the RCA or the LCF. RCA occlusion usually causes larger infarcts than LCF occlusion. The following findings are helpful in the differential diagnosis between RCA and LCF occlusion. First , ST segment depression in lead aVL and I is consistent with RCA occlusion, while isoelectric ST in lead I and ST elevation in aVL suggest LCF occlusion (28). Second, a ratio of ST elevation in leads II/II <1 is more frequent in RCA than LCF occlusion (28, 29). Third, the ratio of ST depression in lead V2 to ST elevation in lead III of <1:2 supports an RCA occlusion whereas a ratio of >1:2 suggests LCF disease (30). Fourth a tall R wave in leads V1 and V2 is seen in LCF occlusion (31).

In patients with an RCA infarct the infarct size depends on the level of the occlusion. A proximal RCA occlusion is associated with right ventricular infarction and an increased mortality. The ECG criteria for proximal RCA occlusion are ST elevation in lead V3R and V4R (less frequently in V1, 2) (32, 33), and second and third degree AV block (34). Another group with an RCA infarct who have a worse short and long-term prognosis are patients with ST segment depression in the right precordial leads. This is believed to be due to extension of myocardial necrosis into the posterolateral wall and posterior septum (35, 36). In addition, ST segment depressions in lead V4—6, which are more prominent than ST depressions in V1—3 suggest multivessel disease and worse outcome (37).

The next question is what is the prognostic significance of the 12-lead ECG in patients with LCF disease. In general, occlusion of the LCF causes smaller myocardial infarcts. However this may not always be true. O'Keefe et al. (38) showed that the size of an inferior wall acute myocardial infarction caused by LCF occlusion could be similar to those infarcts caused by an RCA occlusion. In addition, some patients with LCF occlusion develop hemodynamically significant mitral regurgitation (39). The diagnostic accuracy of the 12-lead ECG in LCF disease is not as good as it is in patients with LAD or RCA disease. As shown by Huey et al. (31) the ECG is only abnormal in 50 % of patients with LCF occlusion. One approach to improve the diagnostic accuracy of LCF occlusion, is the recording of lead V7—9 (40). In addition, some patients with LCF occlusion can present with ST segment depression in the right precordial leads imitating anterior NQMI.

Distortion of the terminal QRS. One of the newer ECG findings used in identifying high-risk patients is distortion of the terminal portion of the QRS complex (25, 41). In leads with QR

morphology, the main criteria for this abnormality are tall T waves with ST segment elevation and the J point above the lower half of the R wave. On the other hand, in leads with RS morphology this abnormalities causes disappearance of the S waves (25, 41) (Fig. 3). These ECG changes are similar to those reported by Wimalarta (42) who introduced the term „Tombstoning" of the ST segment. The criteria of Wimalarta were recently modified by Guo et al. (43) and are the following: a) absent R wave or an R wave duration <0.04 s with minimal amplitude, b) convex upward ST segment merging with the descending R or the ascending QS/QR, c) the peak of the ST segment is higher than the R, and d) the ST segment merges with the T wave.

The ECG is helpful in differentiating between various types of ACS and to identify high, intermediate and low risk patients. In non-ST segment elevation ACS, the high-risk patients are those with ST segment depression alone or in combination with ST segment elevation. Furthermore, marked and diffuse ST segment depression is consistent with significant left main or severe three vessel disease. In patients with either a normal ECG or T wave inversion in less that 5 leads, the rates of mortality, AMI and recurrent chest pain are low. In patients with ST segment elevation ACS, the prognostic indicators include the degree and extent of ST segment elevation, the identification of the infarct related artery and the presence of distortion of the terminal portion of the QRS complex. There are however some important limitations of the ECG, including its failure to detect small infarcts and LCF occlusion. In other words, the best approach for risk stratification of patients with ACS is to combine the clinical data, ECG findings and biochemical markers of myocardial necrosis. In addition, the initial ECG has to be supplemented with follow up tracings to identify patients who have a delayed evolution of ischemic changes.

References

1. Theroux P, Fuster V: Acute coronary syndrome: Unstable angina and non-Q-wave myocardial infarction. Circulation 1998; 97: 1195—1206.

2. Savonitto S, Fusco R, Granger CB et al: Clinical, electrocardiographic, and biochemical data for immediate risk stratification in acute coronary syndrome. Ann. Noninvasive Electrocardiology 2001; 6: 64—77.

3. O'Rourke RA, Hochman JS, Cohen MC et al: New approaches to diagnosis and management of unstable angina and non-ST-segment elevation myocardial infarction. Arch Intern Med 2001; 161: 674—682.

4. Fibrinolytic therapy trialists (FTT) collaborative group. Collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. Lancet 1994; 343: 311—322.

5. Antman EM, Cohen M, Bernink PJLM et al: The TIMI risk score for unstable angina/non-ST elevation MI. JAMA 2000; 284: 835—842.

6. Kontos MC, Kurdziel KA, Ornato JP et al: A nonischemic electrocardiogram does not always predict a small myocardial infarction: Results with acute myocardial perfusion imaging. Am Heart J 2001; 141: 360—366.

7. Caceres L, Cookie D, Zalenski R et al: Myocardial infarction with an initially normal electrocardiogram-angiographic findings. Clin Cardiol 1995; 18: 563—568.

8. Cannon CP, McCabe CH, Stone PH et al: The electrocardiogram predicts one-year outcome of patients with unstable angina and non-q wave myocardial infarction: Results of TIMI III registry ECG ancillary study. J Am Coll Cardiol 1997; 30: 133—140.

9. Nyman I, Areskog M, Areskog NH et al: Very early risk stratification by electrocardiogram at rest in man with suspected unstable coronary artery heart disease. J Intern Med 1993; 234: 293—301.

10. Holmvang L, Luscher MS, Clemmensen P et al: Very early risk stratification using combined ECG nd biochemical assessment in patients with unstable coronary artery disease (A thrombin inhibition in myocardial ischemia) (TRIM) substudy). Circulation 1998; 98: 2004—2009.

11. Holmvang L, Clemmensen P, Wagner G et al: Admission standard electro-cardiogram for early risk stratification in patients with unstable coronary artery disease not eligible for acute revascularization therapy: A TRIM substudy. Am Heart J 1999; 137: 24—33.

12. Savonitto S, Ardissino D, Granger CB et al: Prognostic value of the admission electrocadiogram in acute coronary syndrome JAMA 1999; 281: 707—713.

13. Mahon NG, Codd MB, McKenna CJ et al: Characteristics and outcomes in patients with acute myocardial infarction with ST-segment depression on initial electrocardiogram. Am Heart J 2000; 139: 311—319.

14. Willich SN, Stone PH, Muller JE et al: High-risk subgroup of patients with non-W wave myocardial infarction based on direction and severity of ST segment deviation. Am Heart J 1987; 114: 1110—1119.

15. Lee HS, Cross SJ, Rawles JM et al: Patients with suspected myocardial infarction who present with ST depression. Lancet 1993; 342: 1204—1207.

16. Hyde TA, French JK, Wong CK et al: Four-year survival of patients with acute coronary syndrome without ST-segment elevation and prognostic significance of 0.5-mm ST-segment depression. J Am Cardiol 1999; 84: 379—385.

17. Schechtman KB, Capone RJ, Kleiger RE et al: Risk stratification of patients with non-Q wave myocardial infarction; The critical role of ST segment depression. Circulation 1989; 80: 1148—1158.

18. 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—941.

19. Cannon CP, Thompson B, McCabe CH et al: Predictors of non-q-wave acute myocardial infarction in patients with acute ischemic syndrome: An analysis from the thrombolysis in myocardial ischemia (TIMI) III trials. Am J Cardiol 1995; 75: 977—981.

20. Gorgels AP, Vos MA, Mulleneers R et al: Value of the electrocardiogram in diagnosing the number of severely narrowed coronary arteries in rest angina pectoris. Am J Cardiol 1993; 72: 999—1003.

21. de Zwaan C, Bar FW, Janssen JHA et al: Angiographic and clinical characteristics of patients with unstable angina showing an ECG pattern indicating critical narrowing of the proximal LAD coronary artery. Amer Heart J 1989; 117: 657—665.

22. Christian TF, Gibbons RJ, Clements IP et al: Estimation of myocardium at risk and collateral flow in acute myocardial infarction using

electrocardiographic indexes with comparison to radionuclide and angiographic measures. J Amer Coll Cardiol 1995; 26: 388—393.

23. Aldrich HR, Wagner NB, Boswick J et al: Use of initial ST segment deviation for prediction of final electrocardiographic size of acute myocardial infarctions. Amer J Cardiol 1988; 61: 745—753.

24. Holland RP, Arnsdorf MF: Solid angle theory and the electrocardiogram: Physiologic and quantitative interpretation. Prog Cardiovasc Dis 1977; 19: 431—457.

25. Birnbaum Y, Maynard C, Wolfe S et al: Terminal QRS distortion on admission ECG is better than ST-segment measurements in predicting final infarct size and assessing the potential effect of thrombolytic therapy in anterior wall acute myocardial infarction. Amer J Cardiol 1999; 84: 530—534.

26. Birnbaun Y, Sclarovsky S, Solodky A et al: Prediction of the level of anterior descending coronary artery obstruction during anterior wall acute myocardial infarction by the admission electrocardiogram. Amer J Cardiol 1993; 72: 823—826.

27. Tamura A, Kataoka H, Nagase K et al: Clinical significance of inferior ST elevation during acute anterior myocardial infarction. Brit Heart J 1995; 74: 611—614.

28. Cia BL, Yip JWL, Tan HC et al: Usefulness of ST elevation II/III ratio and ST deviation in lead I for identification the culprit artery in inferior wall acute myocardial infarction. Amer J cardiol 2000; 86: 341—343.

29. Herz I, Assali AR, Adler Y et al: New electrocardiographic criteria for predicting either right or left circumflex artery as the culprit coronary artery in inferior wall acute myocardial infarction. Amer J Cardiol 1997; 80: 1343—1345.

30. Kosude M, Kimura K, Ishikawa T et al: New ectrocardiographic criteria for predicting the site of coronary artery occlusion in inferior mall acute myocardial infarction. Amer J Cardiol 1998; 82: 1318—1322

31. Huey BL, Beller GA, Kaiser DL et al: A comprehensive analysis of myocardial infarction due to left circumflex artery occlusion: comparison with infarction due to right coronary and left anterior descending artery occlusion. J Amer Coll Cardiol 1988; 12: 1156—1166.

32. Robalino BD, Whitlow PL, Underwood DA et al: Electrocardiographic manifestations of right ventricular infarction. Amer Heart J 1988; 11: 138—144.

33. Brat SH, Brugada P, Zwann C et al: Value of electrocardiogram in diagnosis of right ventricular involvement in patients with an acute inferior wall myocardial infarction. Brit Heart J 1983; 49: 368—372.

34. Brat SH, de Zwaan C, Brugada P et al: Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of development atrioventricular conduction disturbance. Amer Heart J 1984; 107: 1183—1187.

35. Shah PK, Pichler M, Bergman DS et al: Non-invasive identification of a high risk subset of patients with acute inferior myocardial infarction. Amer J Cardiol 1980; 46: 915—921.

36. Peterson ED, Hathway WR, Zabel KM et al: Prognostic significance of precordial ST segment depression during inferior myocardial infarction in the thrombolytic era: results in 16521 patients. J Amer Coll Cardiol 1996; 28: 305—312.

37. Birnbaum Y, Wagner GS, Barbash GI et al: Correlation of angiographic findings in right (V1 to V3) versus left (V4 to V6) precordial ST segment depression in inferior wall acute myocardial infarction. Amer J Cardiol 1999; 83: 143—148.

38. O'Keefe JH Jr, Sayed-Taga K, Gibson W et al: Do patients with left circumflex coronary artery-related acute myocardial infarction without ST-segment elevation benefit from reperfusion therapy? Amer J Cardiol 1995; 75: 718—720.

39. Matetzky S, Freimark D, Feinberg MS et al: Acute myocardial infarction with isolated ST-segment elevation in posterior chest leads V7-9. „Hidden" St segment elevation revealing acute posterior infarction. J Amer Coll Cardiol 1999; 34: 748—753.

40. Birnbaum Y, Sclarovsky S, Blum A et al: Prognostic significance of the initial electrocardiographic pattern in a first acute anterior wall myocardial infarction. Chest 1993; 103: 1681—1687.

41. Wimalarata HSK: „Tombstoning" of St segment in acute myocardial infarction. Lancet 1993; 342: 496 (letter).

42. Guo XH, Yap YG, Chen LJ et al: Correlation of coronary angiography with „Tombstoning" electrocardiographic pattern in patients with after acute myocardial infarction. Clin Cardiol 2000; 23: 347—353.

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