

# Specific electrocardiographic findings due to occlusion of the first diagonal artery

## *Birinci diyagonal arter tıkanıklığının neden olduğu spesifik elektrokardiyografik bulgular*

Okan Gülel, Hülya Çiçekçioğlu\*, Meltem Tekin\*, Sinan Aydoğdu\*, Erdem Diker\*

Department of Cardiology, Faculty of Medicine, 19 Mayıs University, Samsun, Turkey

\*Department of Cardiology, Numune Education and Research Hospital, Ankara, Turkey

### Introduction

The prediction of exact site of occlusion of the infarct-related artery by a noninvasive method immediately after admission to the hospital may help clinicians to estimate myocardial area at risk and to plan therapeutic interventions. The electrocardiography (ECG) is an effective noninvasive tool for rapid diagnosis of acute myocardial infarction. Some ECG criteria, which are usually underutilized by clinicians are presented to estimate precise location of culprit lesion for occlusions of both coronary arteries and their side branches. Below we report a case with specific ECGs due to occlusion of the first diagonal artery.

### Case Report

Fifty eight-year-old woman was admitted to the emergency department due to angina pectoris lasting for about 10 hours. She had been using antihypertensive medication (enalapril 20 mg once a day) for 5 years and had been smoking for 10 years. At admission, systolic blood pressure was 140 mmHg, diastolic blood pressure was 80 mmHg, pulse rate was 90/minute, respiration rate was 20/minute and body temperature was 37.0 °C. Her physical examination was normal. Serum creatine kinase (CK), CK-MB and troponin-I levels were 390 U/L (35-195 U/L), 70 U/L (5-25 U/L) and 5 ng/mL (<0.1 ng/ml) respectively. Other blood tests were normal. On her electrocardiography, interestingly, ST segment elevations in noncontiguous leads of aVL and V2 together with ST segment depressions in leads of III and aVF were detected (Fig. 1A). In coronary intensive care unit, treatment with aspirin, heparin, nitroglycerin,  $\beta$ -blocker (metoprolol), angiotensin-converting enzyme inhibitor (captopril) and statin (atorvastatin) was given to the patient. After that treatment, her chest pain relieved and ST segment elevations returned to isoelectric line. During follow-up, cardiac enzymes (CK, CK-MB) reached the peak levels and then decreased, and no complication has occurred. Two days later, Q waves developed in the ECG leads aVL and V2 (Figure 1B). Transthoracic echocardiography displayed hypokinesia in mid and basal portions of anterior wall. Other regional wall

motions were normal and ejection fraction was 55% by modified Simpson's rule. Coronary angiography revealed only obstructive lesion at the first diagonal branch of left anterior descending (LAD) coronary artery (Fig. 2). She did not accept percutaneous coronary intervention to the diagonal artery and was discharged from hospital on medical treatment.

### Discussion

The most frequently seen infarct-related arteries among patients with myocardial infarction are the LAD coronary artery (44%-56%), followed by the right coronary artery (27%-39%) and the circumflex artery (17%) (1). Almost all anterior infarctions are due to occlusions somewhere in LAD coronary artery or in one of its branches. At this point, the 12-lead ECG has a special importance for the diagnosis and triage of patients with infarction by detecting myocardial injury and by assessing myocardial area at risk.

Some underestimated ECG criteria help us to predict precise location of the occlusion site. For example, a few ECG signs were found to be specific for the occlusions of the LAD coronary artery at the level of 1st septal perforator. These are ST elevation in aVR, ST depression in V5, disappearance of pre-existent septal Q waves in lateral leads and right bundle branch block (2-4). Lesion of the LAD coronary artery at the level of the first diagonal can cause ST elevation in both leads I and aVL or can cause association of precordial ST elevation with ST elevation in lead aVL (5, 6). Also when ST elevation in leads I and aVL is together with ST depression in lead V2, the culprit lesion is usually in the first marginal branch of circumflex artery (7).

The first diagonal branch of the LAD coronary artery supplies large area of the anterolateral wall of the left ventricle. As in our case, the occlusion of this branch can cause distinct electrocardiographic pattern due to the affected myocardial area. In many reports, the specific electrocardiographic features are defined as follows (7-9): 1) ST segment elevation in noncontiguous leads of aVL and V2; 2) ST segment depression in leads of III and aVF or V4-V5. This pattern represents a special subtype of anterior myocardial infarction, so called mid-



Figure 1. Electrocardiograms of the patient at admission (A) and two days later (B)



Figure 2. Coronary angiography findings

anterior myocardial infarction (8). Most of the anterior myocardial infarctions affect septal and apical regions of left ventricle. Anterior myocardial infarctions without apical or septal wall involvement are rare. Since blood supply by LAD coronary artery is not blocked in the case of the first diagonal branch occlusion, septal and apical regions are not affected.

Our case shows us that it is possible to predict exact site of culprit lesion in the clinical setting only by characterizing unique ECG patterns.

## References

1. Blanke H, Cohen M, Schlueter GU, Karsch KR, Rentrop KP. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarction. *Am J Cardiol* 1984; 54: 249-55.
2. Yotsukura M, Toyofuku M, Tajino K, Yoshino H, Ishikawa K. Clinical significance of the disappearance of septal Q waves after the onset of myocardial infarction: correlation with location of responsible coronary lesions. *J Electrocardiol* 1999; 32: 15-20.
3. Tamura A, Kataoka H, Mikuriya Y. Electrocardiographic findings in a patient with pure septal infarction. *Br Heart J* 1991; 65: 166-7.
4. Engelen DJ, Gorgels AP, Cheriex EC, De Muinck ED, Ophius AJ, Dassen WR, 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: 389-95.
5. Birnbaum Y, Sclarovsky S, Solodky A, Tschori J, Herz I, Sulkes J, et al. Prediction of the level of left anterior descending coronary artery obstruction during anterior wall acute myocardial infarction by the admission electrocardiogram. *Am J Cardiol* 1993; 72: 823-6.
6. Kim TY, Alturk N, Shaikh N, Kelen G, Salazar M, Grodman R. An electrocardiographic algorithm for the prediction of the culprit lesion site in acute anterior myocardial infarction. *Clin Cardiol* 1999; 22: 77-83.
7. 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: 38-42.
8. Sclarovsky S, Birnbaum Y, Solodky A, Zafrir N, Wurzel M, Rechavia E. Isolated mid-anterior myocardial infarction: a special electrocardiographic sub-type of acute myocardial infarction consisting of ST-elevation in non-consecutive leads and two different morphologic types of ST-depression. *Int J Cardiol* 1994; 46: 37-47.
9. Sgarbossa EB, Birnbaum Y, Parrillo JE. Electrocardiographic diagnosis of acute myocardial infarction: current concepts for the clinician. *Am Heart J* 2001; 141: 507-17.