Key Points and Pitfalls in Electrocardiographic Diagnosis of Acute Myocardial Infarction

Toshihisa ANZAI and Satoshi OGAWA

Cardiopulmonary Division, Department of Medicine, Keio University School of Medicine

Abstract: Since electrocardiographic features of acute myocardial infarction vary temporally and spatially among patients, there are many pitfalls in electrocardiographic diagnosis of this condition. In order to avoid overlooking acute myocardial infarction, it is necessary to consider characteristic findings, such as augmentation of T wave amplitude, ST elevation, and the appearance of abnormal Q waves, in light of the time after onset. Observation of time-course changes allows us to noninvasively understand the presence/absence of recanalization and pathological conditions including infarct extension, infarct expansion, and retention of pericardial effusion, as well as to three-dimensionally diagnose the infarct site from changes in waveform in various leads. The latter findings reflect the anatomy of the artery responsible for the infarction. In addition, it is possible to determine the infarct site and the infarct-related artery from the waveforms of premature beats. In recent years, visualized diagnosis of coronary heart disease has become possible by means of improved techniques including coronary angiography and intravascular ultrasound. However, it seems that the well-established qualitative diagnosis of myocardial infarction by electrocardiography will continue to be important.

Key words: Myocardial infarction; Electrocardiogram; Arrhythmia; Reperfusion therapy

Introduction

Although recent years have seen astonishing technological advances in the examination and treatment of cardiovascular diseases, it is still an indisputable fact that electrocardiography (ECG) is the most important examination in diagnosing acute myocardial infarction. If acute myocardial infarction is suspected from symptoms and ECG findings, emergency treatment...
Based on reperfusion therapy should be administered as promptly as possible. However, electrocardiographic diagnosis is often difficult, such that some patients may not receive timely reperfusion therapy during the acute phase of the disease.

The key points and pitfalls of electrocardiographic diagnosis of acute myocardial infarction are described herein.

**Time-course Changes in the Electrocardiogram**

1. **Time after onset of chest pain and the point of ECG reading**

   First of all, obtaining a full history is essential to avoid overlooking acute myocardial infarction. Second, ECG examination should be performed with adequate knowledge of possible ECG findings based on time after onset. If the patient is in the hyperacute phase (within a few hours after onset), ST elevation or depression will not necessarily be observed. It is important to determine whether there is T-wave amplitude augmentation. On the other hand, if there are no ST changes or abnormal Qwaves 6 or more hours after the onset, differentiation from other diseases, particularly fatal disorders such as acute aortic dissection, is necessary.

2. **Importance of observing time-course changes**

   In addition to the importance of comparing the ECG record taken at the emergency department visit with the patient’s previous ECG record, subsequent frequent ECG recordings will also provide important prognostic information. (1) *Spontaneous recanalization and coronary
vasospasm. If ST elevation is restored to the baseline after visiting the outpatient clinic, it is possible that spontaneous recanalization has occurred or that coronary vasospasm was involved in the heart attack. This should be confirmed before thrombolytic therapy or emergency coronary angiography is performed.

(2) **Change from incomplete to complete occlusion of the infarct-related artery.** Further incremental ST elevation suggests progression to complete occlusion due to thrombus formation. In this case, reperfusion therapy should be performed immediately.

(3) **Success or failure of reperfusion therapy.** It is known that ST elevation is promptly reversed (50% reduction within 1 hour) if thrombolytic therapy is successful, i.e., recanalization is achieved. It is also characteristic for negative Twaves to appear in an early phase. Recently, coronary angioplasty has often been performed as an emergency treatment. Even if recanalization is achieved angiographically, microcirculatory disturbances may persist (no-reflow phenomenon), causing prolonged ST elevation.

(4) **Infarct extension.** Infarct extension occurs if an intracoronary thrombus extends proximally, and ST elevation may be recognized in other leads as well. This finding is said to be relatively common in obese women, after thrombolytic therapy, as well as in those who have diabetes or non-Q-wave myocardial infarction.

(5) **Infarct expansion.** ST segment re-elevation occurs if the area of vulnerable and necrotic myocardium is expanded by wall stress following transmural myocardial infarction. ST elevation in this case can be distinguished from ischemia in view of its upward convex form and persistence over time.

(6) **Pericardial effusion.** If infarction is complicated by pericarditis with pericardial effusion, low voltage in all leads will be observed. Subacute cardiac rupture occurring a few or more days after onset often follows infarct expansion or pericardial effusion. Therefore, these findings may facilitate predicting cardiac rupture to some extent (Fig. 1).

**Changes in ECG Waveforms**

1. **Twave augmentation can easily be overlooked**
   As mentioned previously, Twave augmentation is one of the findings that may be overlooked in the acute phase. Since Twave augmentation is also present in early repolarization, hyperkalemia, left ventricular hypertrophy, and left bundle branch block, differentiation from these conditions is necessary. Whereas such Twaves are bilaterally symmetric and narrow, hyperacute Twaves in myocardial infarction are often relatively broad and their peaks are located posteriorly rather than centrally.

2. **Possible diagnoses with ST elevation**
   Acute myocardial infarction is the first diagnosis to be suspected if there is ST elevation together with chest pain. However, other possibilities should also be considered. In cases of pericarditis, ST elevation is said to be relatively slight, 0.5 mV or less, is not accompanied by mirror-image changes, and is observable in most leads.

3. **Diagnosis of the infarction site**
   As shown in Table 1, it is possible to diagnose the infarct site and the infarct-related artery on the basis of which leads show ST elevation and abnormal Qwaves. However, the combination of leads in which ST elevation and abnormal Qwaves are observed varies somewhat according to individual coronary artery anatomy, and does not necessarily agree with the patterns shown in the table. Therefore, it is important to three-dimensionally understand the infarct site reflected in each lead (Fig. 2).

   ST segment changes can often be observed in precordial leads in inferior infarction or in limb leads in anterior infarction. The significance of such changes will be described below.

   1. **ST changes in the precordial leads in inferior infarction**

   **ST depression:** ST depression is seen in the
Table 1 Diagnosis of the Site of Myocardial Infarction

<table>
<thead>
<tr>
<th>Infarct site</th>
<th>Responsible coronary artery</th>
<th>Sites of abnormal Qwaves and ST elevation on ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior septum</td>
<td>Proximal LAD</td>
<td>I      II     III   aV_L  aV_S  aV_T  V_4W  V_1  V_2  V_3  V_4  V_5  V_6</td>
</tr>
<tr>
<td>Apex</td>
<td>LAD, RCA, or LCX</td>
<td></td>
</tr>
<tr>
<td>High lateral wall</td>
<td>First diagonal branch of LAD</td>
<td></td>
</tr>
<tr>
<td>Anterolateral wall</td>
<td>Proximal LAD including diagonal branch</td>
<td></td>
</tr>
<tr>
<td>Extensive anterior wall</td>
<td>Proximal LAD</td>
<td></td>
</tr>
<tr>
<td>Anteroinferior wall</td>
<td>LAD going around the apex extensively</td>
<td></td>
</tr>
<tr>
<td>Lateral wall</td>
<td>Diagonal branch of LAD, extending to the apex</td>
<td></td>
</tr>
<tr>
<td>High posterior wall</td>
<td>LCX</td>
<td></td>
</tr>
<tr>
<td>Posterolateral wall</td>
<td>LCX (including obtuse marginal branch)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LCX (large obtuse marginal branch)</td>
<td></td>
</tr>
<tr>
<td>Posteroinferior wall</td>
<td>LCX or large RCA</td>
<td></td>
</tr>
<tr>
<td>Inferior wall apex</td>
<td>RCA or LCX</td>
<td></td>
</tr>
<tr>
<td>Inferior wall</td>
<td>RCA or LCX</td>
<td></td>
</tr>
<tr>
<td>Inferior wall/right ventricle</td>
<td>RCA, before bifurcation of the anterior branch</td>
<td></td>
</tr>
</tbody>
</table>

Notes: ● denotes R wave augmentation or ST depression
LAD: left anterior descending artery, RCA: right coronary artery, LCX: left circumflex artery

Fig. 2 Site of myocardial infarction and ECG
Leads V_1–V_4 correspond to the anterior wall (AW) and septum (AS, S), and leads II, III, and aV_L correspond to the left ventricular posterior wall (PW) to the posterior septum (PS). Leads I and aV_T correspond to the left ventricular free wall, and have enantiomorphic relations with leads II, III, and aV_L because of their contralateral location. Although there are no available leads directly facing the high posterior wall or lateral wall (LW), changes at these sites appear as mirror-image changes in leads V_1–V_4. Leads V_5–6 correspond to the left ventricular apex. (Original illustration by Ogawa, S.)
precordial leads in about half of all patients with inferior infarction. The prognosis is reportedly poor if ST depression persists. This is probably because most such patients have multi-vessel disease involving the left anterior descending artery or extensive inferior infarction involving the high posterior wall (ST depression occurs in leads V1–V3 as a mirror-image change representing transmural infarction in the lateral wall [LW] of the base of the heart, as shown in Fig. 2).3,4) In particular, when ST depression in the precordial leads is as severe as 3 mm or more, it is strongly suspected that the left anterior descending artery is also affected.5) ST depression in leads I and aVL in inferior infarction represents mirror-image changes against ST elevation in the anterior wall (AW) at the base of the heart in Fig. 2 are enantiomorphic).

**ST elevation:** ST elevation may be found in leads V1 and V2 in less than 10% of patients with inferior infarction. This finding is known to suggest the presence of right ventricular infarction.6) Anterior infarction is different from right ventricular infarction in that the degree of ST elevation is greater in lead V1 than in lead V2 (V1 > V2) in the former whereas it is greater in lead V1 than in lead V2 in the latter (V1 < V2) (Fig. 3). However, the finding associated with the highest sensitivity and specificity in the diagnosis of right ventricular infarction is ST elevation of 1 mm or more in the right precordial lead (V4R).7) In cases of inferior infarction accompanied by a blood pressure decrease, particularly when there is no angina preceding the infarction, right ventricular infarction is also frequently present.8) Therefore, ECG findings in the right precordial lead should always be confirmed.

(2) **ST changes in the limb leads in anterior infarction**

**ST depression:** It is said that, if there is ST depression in the limb leads in anterior infarction, multi-vessel disease is highly probable and the prognosis is thus poor.9) However, ST changes in the inferior wall lead in anterior infarction are defined by the sum total of mirror-image changes (ST depression) in response to ischemia of the anterior wall and changes due to ischemia of the inferior wall (ST elevation). Therefore, if the left anterior descending artery perfuses an extensive area ranging from the apex to the inferior wall, ST changes in the limb leads may be counterbalanced by mirror-image changes in the anterior wall, such that ST changes may appear to be absent or ST elevation may occur. On the contrary, if the left anterior descending artery perfuses a limited area, prominent ST depression is often seen in leads II, III, and aVF.10)

**ST elevation:** In infarction of the left anterior descending artery long enough to go around the apex of the heart as mentioned above, ST elevation may be found in leads II, III, and aVF. When infarction has occurred in an area proximal to the diagonal branch, ST elevation is also seen in leads I and aVL.

(3) **The infarct-related artery in inferior infarction (differentiation between the right coronary artery and the left circumflex artery):**

It is sometimes difficult to electrocardiographically identify the infarct-related artery in
inferior infarction, because the anatomy of the coronary arteries varies among patients. However, a pure high posterior wall infarction showing R wave augmentation alone (mirror-image changes) in lead V1 is a finding characteristic of myocardial infarction in the area of the left circumflex artery, particularly its obtuse marginal branch (Fig. 4). If ST elevation is present in lead I and in at least one of the lateral wall leads (aV_L, V_5, V_6), the lesion is presumed to be located in the left circumflex artery with a sensitivity of 80% and a specificity of 90%.11)

4. Meaning of Q waves

Q waves having a width of 0.04 sec or more and corresponding to one-fourth of the voltage of R waves are regarded as abnormal Q waves. This finding is particularly important in diagnosing the infarct site in transmural myocardial infarction. However, Q waves can be induced by conditions other than infarction; the following findings are useful in differential diagnosis.12)

(1) **Q waves in lead aV_L:** Such Q waves are often found in the heart with a vertical axis. If there are no Q waves with a width of 0.04 sec in lead I or the left precordial lead, or if there is no abnormality in ST-T, myocardial infarction is unlikely.

(2) **Q waves in leads III and aV_R:** If there is QR in lead aV_R, inferior infarction is highly unlikely (the presence of rS in lead aV_R suggests inferior infarction).

(3) **Q waves in leads V_1-V_3:** The presence of Q waves only in leads V_1-V_2 is often seen in normal cases. If Q waves are observed in leads V_1-V_3, anterior infarction should be suspected, but differentiation from left ventricular hypertrophy, left bundle branch block, and chronic obstructive pulmonary disease is necessary.

**Arrhythmia in Myocardial Infarction**

1. **Importance of premature beats**

Ventricular premature beats are often seen in acute myocardial infarction. Observation of the waveform may provide important information for diagnosis. For instance, if there are premature beats with a form of bifascicular block (left anterior hemiblock and right bundle branch block) in inferior infarction, the origin of the premature beats is considered to be the ischemic site of the left posterior branch. However, since the left posterior branch is perfused by both the left anterior descending artery and the right coronary artery, such premature beats are unlikely to occur in inferior infarction alone. In this case, multi-vessel disease involving the left anterior descending artery and right coronary artery or the presence of collateral flow from the right coronary artery to the left anterior descending artery should be suspected.

2. **Tachyarrhythmia**

Ventricular tachycardia and ventricular fibrillation should always be borne in mind as a fatal arrhythmia possibly complicating myocardial infarction. However, accelerated idioventri-
cular rhythm (AIVR) seen in about 50% of reperfused patients is not an indication for antiarrhythmic drug therapy, because the rate is 50–120/min and does not influence hemodynamics. Ventricular tachycardia often begins after a ventricular premature beat, whereas AIVR begins after a long pause. In general, antiarrhythmic medication is given to patients with warning arrhythmia characterized by 6 or more distinct rhythms per minute, RonT, polymorphism, and three or more serial rhythms. To manage potentially fatal arrhythmias such as recurrent sustained ventricular tachycardia or ventricular fibrillation in the chronic phase of myocardial infarction, the use of an implantable defibrillator should also be considered.

A type of supraventricular arrhythmia relatively frequently seen after myocardial infarction is atrial fibrillation. In most cases, such arrhythmia occurs transitorily with hemodynamic worsening, postinfarction pericarditis, or atrial infarction (as represented by elevated PQ segment baseline on ECG) and disappears spontaneously after resolving these problems. If there is hemodynamic disturbance due to atrial fibrillation, cardioversion should be considered promptly. If transient atrial fibrillation is frequent in the chronic phase, the use of antiarrhythmic drugs should also be considered. However, the possibility has been pointed out that the prognosis may be worsened by inhibition of cardiac function or by the proarrhythmic action of antiarrhythmic drugs. Therefore, prudent application of such medications is necessary.

3. Bradyarrhythmia

If atrioventricular block of Mobitz type II or higher, bifascicular block, or symptomatic bradycardia not responding to atropine is found after myocardial infarction, prompt insertion of an intravenous pacemaker is warranted. In these cases of bradycardia, it is necessary to observe the QRS interval of the escape rhythm. If the ventricular escape rhythm has a wide QRS, severe block involving up to the His bundle is suspected, and a permanent pacemaker is often required.

Conclusion

ECG changes in acute myocardial infarction vary among different patients and may cause difficulties in diagnosis. However, it is possible to ascertain the ongoing phenomenon accurately and without difficulty if one copes with waveform changes in each lead in a flexible manner based on a full understanding of the meaning of such changes. Although cardiac catheterization is now frequently used, and visualized diagnosis of myocardial infarction is becoming a common practice, the importance of ECG should never been underestimated in light of the qualitative diagnosis of myocardial infarction.

REFERENCES

7) Klein, H.O., Tordijman, T., Ninio, R. et al.: The


