Abstract—The 12-lead electrocardiogram (EKG), a powerful tool used in evaluating the chest pain patient, has its shortcomings. One such failing is encountered in a patient with one of the following electrocardiographic patterns: left bundle branch block (LBBB), ventricular paced rhythm (VPR), and left ventricular hypertrophy (LVH). These patterns reduce the ability of the EKG to detect acute coronary ischemic change and acute myocardial infarction (AMI). Several strategies are available to assist in the correct interpretation of these complicated electrocardiographic patterns, including a knowledge of the ST segment-T wave changes associated with these confounding patterns, performance of serial EKGs, and comparison with previous EKGs if available. This article suggests guidelines and interpretive tools for diagnosing AMI on EKG in patients with these confounding patterns. © 2000 Elsevier Science Inc.

Keywords—electrocardiogram; EKG; left bundle branch block; ventricular paced rhythms; left ventricular hypertrophy; chest pain; acute myocardial infarction

INTRODUCTION

The 12-lead electrocardiogram (EKG) is a powerful tool in evaluating the chest pain patient and assists in making the diagnosis of acute myocardial infarction (AMI), selecting appropriate therapies, securing adequate inpatient disposition, and predicting risk for cardiovascular complications. The EKG, however, has its shortcomings. One such failing is encountered in a patient with one of the following electrocardiographic patterns: left bundle branch block (LBBB), ventricular paced rhythm (VPR), and left ventricular hypertrophy (LVH). These patterns reduce the utility of the EKG in detecting acute coronary ischemic change. Furthermore, these confounding patterns may mimic pathologic findings associated with acute coronary events, leading the unwary physician to a wrong diagnostic and therapeutic plan. The following cases illustrate the use of the EKG in the chest pain patient with AMI in the presence of these confounding electrocardiographic patterns.

CASE PRESENTATIONS

Case One

A 66-year-old male with a history of myocardial infarction, diabetes mellitus, and hypertension presented to the Emergency Department (ED) with substernal chest pain. The pain, similar to his past angina, had appeared ap-
proximately 4 h before arrival. The physical examination was significant for marked diaphoresis. A 12-lead EKG revealed normal sinus rhythm (NSR) with LBBB morphology (Figure 1). The patient was treated for acute cardiac ischemia but continued to have chest discomfort. Serial EKGs were performed, and after approximately 35 min, the tracing revealed dynamic change with ST segment elevation in the inferior and lateral leads suggestive of inferolateral AMI (Figure 2). The patient received a thrombolytic agent and had an uneventful recovery from the myocardial infarction, which was confirmed by elevated cardiac enzymes.

Case Two

A 74-year-old female with a history of angina and tachy-bradycardia syndrome requiring a permanent ventricular pacemaker presented to the ED with substernal chest discomfort similar to her prior angina. The physical examination was unremarkable. Initial 12-lead EKG showed a VPR with appropriately discordant ST segment waveforms (Figure 3). Despite antianginal therapy, the patient’s pain worsened, with development of electrocardiographic changes seen in the lateral leads (Figure 4). An AMI was confirmed with elevated CPK levels and positive MB fraction.

Case Three

A 64-year-old female with a history of diabetes mellitus and hypertension presented to the ED with chest pain. The chest discomfort was described as left-sided in location, “aching” in quality, and radiating to the left shoulder. The patient also noted diaphoresis and nausea. The physical examination was unremarkable. The initial 12-lead EKG demonstrated ST segment-T wave abnormalities felt to be consistent with LVH (Figure 5). Despite treatment with nitroglycerin, morphine, aspirin, and heparin, the patient continued to have pain. Serial EKGs demonstrated an anterior AMI (Figure 6). The patient underwent emergent cardiac catheterization that revealed a proximal left anterior descending artery lesion, which was successfully treated by angioplasty.

DISCUSSION

ST segment elevation is perhaps the “most demanding” of the electrocardiographic features encountered in the ED. It is “demanding” in that its presence must be explained, and if the etiology is AMI, urgent therapeutic decisions must be made. ST elevation is a common EKG finding in patients with chest pain. Approximately 20% of all adult ED chest pain patients have ST elevation in
two anatomically contiguous leads (1). Of these patients, studies suggest that only 15–25% will have AMI. The remainder will have noninfarction diagnoses such as LBBB, VPR, and LVH (1,2). Even among patients admitted to a coronary care unit, Miller et al. demonstrate that ST elevation is diagnostic for acute infarct

Figure 2. NSR with LBBB demonstrating acute inferolateral AMI. Concordant ST segment elevation is seen in leads V5 and V6 while isoelectric ST segments with upright T waves are found in leads I and aVL. These findings suggest AMI from both the violation of the rule of appropriate discordance and the serial change from Figure 1.

Figure 3. Ventricular paced rhythm. The 12-lead EKG records the altered ventricular activation as it moves from right to left, producing a broad, mainly negative QS or rS complex in leads V1-V6 with QS complexes. A large monophasic R wave is encountered in leads I and aVL and, on occasion, in leads V5 and V6. QS complexes are frequently encountered in leads II, III and aVF. As with LBBB, the appropriate ST segment-T wave configurations are discordant with the QRS complex.
in only half of patients with a history of ischemic heart disease (3).

Syndromes causing ST elevation not related to AMI are frequently misdiagnosed as acute infarction. This may subject patients to unnecessary and potentially dangerous therapies and procedures. Sharkey et al. note that 11% of patients who receive thrombolytic agents are not experiencing AMI (4). The electrocardiographic syndromes producing this pseudo-infarct ST segment elevation include benign early repolarization (30%), LVH (30%), and various intraventricular conduction abnormalities, including LBBB (30%) (4).

**Left Bundle Branch Block Pattern**

With normal atrioventricular (AV) impulse transmission, electrical stimuli from the sinus node proceed through atria and the AV node, entering the well-defined, efficient ventricular conduction system, which results in activation of the ventricular myocardium. With an intact intraventricular conduction tract, depolarization occurs in rapid fashion, resulting in nearly simultaneous electrical discharge of the ventricular myocardium.

In LBBB, the left ventricle cannot be depolarized from the left bundle branch, and the impulse must proceed down the right bundle and across the interventricular septum, eventually reaching the left ventricle via the myocardium. This depolarization pattern is abnormal since activation occurs from right to left, the opposite direction from normal.

On 12-lead EKG, the abnormal right to left ventricular activation produces a broad, mainly negative QS or rS complex in lead V1 (5,6). A positive, monophasic R wave is seen in V6 and frequently in leads I and aVL. Poor R wave progression or QS complexes are noted in the right to mid-precordial leads, rarely extending beyond leads V4 or V5. QS complexes also may be encountered in leads III and aVF. Consistent with intraventricular delay, the QRS complex is widened to at least 0.12 s.

In LBBB, ST segment-T wave configurations are discordant, directed opposite from the terminal portion of the QRS complex. This QRS complex-T wave axes discordance demonstrates the rule of appropriate discordance. According to this rule, leads with either QS or rS complexes (complexes that are partially or entirely negative in deflection) may have markedly elevated ST segments, mimicking AMI (see Figure 1—inferior and anterior leads). Leads with a large monophasic R wave demonstrate ST segment depression (Figure 1—lateral leads). The T wave has a convex upward shape or a tall, vaulting appearance, similar to the hyperacute T wave of early AMI, especially in the right to mid-precordial leads. T waves in leads with the monophasic R wave are frequently inverted.

Loss of this normal QRS complex-T wave axes discordance may imply acute ischemia in patients with

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**Figure 4. Ventricular paced rhythm demonstrating lateral AMI.** Leads I and aVL demonstrate concordant ST segment elevation that is not appropriate for VPR, and also demonstrate serial change from Figure 3.
LBBB (7). Sgarbossa et al. have developed a clinical prediction rule to assist in the EKG diagnosis of AMI in the setting of LBBB using specific electrocardiographic findings (8). They analyzed numerous EKG abnormalities previously reported to be suspicious or diagnostic for AMI in patients with LBBB and identified three criteria suggestive of acute infarction. Investigation of the GUSTO-I trial data on 131 patients with LBBB and enzymatically proven AMI found three specific EKG criteria that are independent predictors of infarction. These criteria, listed below, were then ranked by a scoring system based on the probability of AMI.

1. ST segment elevation > 1 mm concordant with QRS complex (score of 5);
2. ST segment depression > 1 mm in leads V1, V2, or V3 (score of 3); and
3. ST segment elevation > 5 mm discordant with the QRS complex (score of 2).

A score of 3 or more suggests that the patient is experiencing an AMI. A score less than 3 is less helpful. The utility of these criteria is that they allow the clinician to recognize electrocardiographic changes that are suspicious for AMI in the face of LBBB.

Recent studies, however, suggest that the criteria may be less useful than has been reported (9,10). Shapiro et al. report that the criteria have both low interobserver reliability and low sensitivity for AMI in chest pain patients with LBBB in their ED (9). Shlipak also finds that none of the criteria described by Sgarbossa et al. effectively distinguishes AMI from noncoronary diagnoses. The various electrocardiographic criteria (8) indicate AMI in only 3% of cases, with a sensitivity for the diagnosis of only 10%. Based on their findings, the authors suggest that all patients suspected of AMI with LBBB should be considered for thrombolysis (10). Nonetheless, many clinicians believe the criteria developed by Sgarbossa et al. are still useful and have discredited the widely taught belief that the EKG is of no utility in diagnosing AMI in the presence of LBBB.

**Ventricular Paced Rhythm Pattern**

In the patient with VPR, the 12-lead EKG records the altered ventricular activation generated by the pacemaker...
as it moves from right to left, producing a broad, mainly negative QS or rS complex and poor R wave progression in leads V1-V6. A large monophasic R wave is encountered in leads I and aVL and, on occasion, in V5 and V6. QS complexes are frequently encountered in leads II, III, and aVF. As with LBBB, the appropriate ST segment-T wave configurations are discordant, or directed opposite from the terminal portion of the QRS complex (see Figure 3).

Several authors have attempted to characterize diagnostic ischemic changes on EKG in patients with VPR and myocardial infarction. These attempts have included criteria similar to those described above for LBBB, comparison with previous EKGs, and complicated analyses of QRS complex vectors (11–15). These approaches have met with limited success, however, and often are unable to distinguish on EKG between acute infarction changes and more chronic findings associated with ischemic heart disease.

Again, based on GUSTO trial data, Sgarbossa et al. detail the EKG changes encountered in 32 patients with VPR and AMI (16). Three EKG criteria are found to be useful in the early diagnosis of AMI:

1. discordant ST segment elevation > 5 mm;
2. concordant ST segment elevation > 1 mm; and
3. ST segment depression > 1 mm in leads V1, V2, or V3.

The most useful criterion is discordant ST elevation of 5 mm or more. This criterion violates the rule of appropriate discordance because there is an inappropriate degree of discordance. Normally, VPR repolarization changes produce ST elevation of lesser magnitude. Unlike previous studies, the criteria described by Sgarbossa et al. can distinguish between acute and prior infarction, providing the clinician with assistance in making the early diagnosis of AMI in the presence of VPR (see Figure 4—lateral leads) (16,17).

**Left Ventricular Hypertrophy Pattern**

In a study of 5,768 patients with symptoms suggestive of acute ischemia, Larsen et al. report that 8% (413 patients) had an EKG consistent with LVH. However, only one-quarter were found to have acute cardiac ischemia (either unstable angina or AMI). The large majority were ultimately diagnosed with nonischemic syndromes. Ad-
mitting physicians correctly identified LVH on EKG in only 22% of cases. Frequently, physicians attributed ST segment and T wave changes to ischemia or infarction rather than repolarization abnormalities associated with LVH (18).

In LVH, ST segment and T wave changes are encountered in approximately 70% of cases, resulting from altered repolarization of the ventricular myocardium due to hypertrophy. As with other confounding patterns, these LVH-related EKG changes may mask or mimic early findings of acute coronary ischemia, though to a lesser degree than is seen with LBBB or VPR (6,19).

LVH is associated with poor R wave progression and loss of septal R wave, commonly producing a QS pattern most often seen in leads V1 and V2 and rarely extending beyond V3. As predicted by the concept of appropriate discordance, ST elevation is encountered in this same distribution along with prominent, hyperacute T waves. The ST elevation is usually 2 to 4 millimeters in height, though it may reach 5 millimeters or more, and may be difficult to distinguish from that associated with AMI. The initial, upsloping portion of the ST segment-T wave complex is frequently concave in LVH compared with either flattened or convex in AMI, though this morphologic feature is imperfect since early AMI may reveal a concave segment (see Figure 5 for LVH with appropriate ST segment waveforms) (6).

The “strain” pattern, characterized by downsloping ST segment depression with abnormal T waves in leads with prominent R waves (lateral leads I, aVL, V5 and V6), is frequently misinterpreted as acute ischemia (4,6). The ST segment-T wave complex has been described as: initially bowed upward (convex upward) followed by a gradual downward sloping into an inverted, asymmetric T wave with an abrupt return to the baseline. It is important to realize that significant variability may be encountered in this pattern. The T wave may be minimally inverted or greater than 5 millimeters in depth. Abnormal T waves also may be encountered in patients lacking prominent voltage for LVH (4,6). Other features of the ST segment-T wave complex consistent with LVH repolarization changes include:

1. depression of the J point;
2. asymmetric T wave inversion with gradual downsloping and rapid return to baseline;
3. terminal positivity of the T wave, described as “overshoot”;
4. T wave inversion in lead V6 greater than 3 millimeters; and
5. T wave inversion greater in lead V6 than V4.

These criteria suggest that the ST segment-T wave complex is consistent with LVH repolarization as opposed to acute ischemic changes (see Figures 5 and 6) (20).

**CONCLUSION**

Several strategies are available to assist in the correct interpretation of the EKG complicated by the presence of confounding patterns such as LBBB, VPR, and LVH. The rule of appropriate discordance is of considerable use when evaluating the EKG of patients with LBBB, VPR, and LVH. ST segment-T wave complex abnormalities that are not consistent with the anticipated patterns should suggest the possibility of acute ischemia.

Second, the performance of serial EKGs may demonstrate dynamic EKG changes commonly encountered in patients with acute ischemia. Waveform abnormalities associated with LBBB, VPR, and LVH are relatively stable and unlikely to change over the short-term in the ED. Finally, a comparison with previous EKGs, if available, provides an invaluable clue to the patient’s current electrocardiographic status. However, reliance upon the timely availability of past EKGs as a primary clinical tool, rather than a sound knowledge of the electrocardiographic findings associated with these confounding patterns, is not advised. The first strategy, an awareness of the anticipated electrocardiographic changes, is the most important clinical tool in making diagnostic and therapeutic decisions.

**REFERENCES**


