Right vs Left Bundle Branch Block in Acute MI

When your patient's signs and symptoms indicate possible myocardial ischemia or infarction and the ECG shows a bundle branch block, side matters. The authors explain.

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The diagnosis of ST-segment elevation myocardial infarction (STEMI) in the setting of bundle branch block represents a particular challenge for the emergency physician. In evaluating such a patient, the following issues merit consideration:

- Is the conduction disturbance new or old?
- Does the bundle branch block mask any electrocardiographic features of STEMI?
- Is it possible to assess the size or location of the infarction?
- Does the presence of the bundle branch block have prognostic importance?

PATIENT PRESENTATION

A 60-year-old woman is brought to the emergency department with chest pain that started about an hour earlier, after dinner. The pain is crushing, substernal, left sided, nonradiating, and associated with diaphoresis. She denies shortness of breath, palpitations, or fever, and claims she has never experienced chest pain before. The review of systems is otherwise negative.

She has been smoking a pack of cigarettes a day for the past 30 years, she says, and drinks five beers daily. She denies illicit drug use. Her medical history is significant for hypertension, dyslipidemia, stroke, and seizure disorder. The family history is unremarkable.

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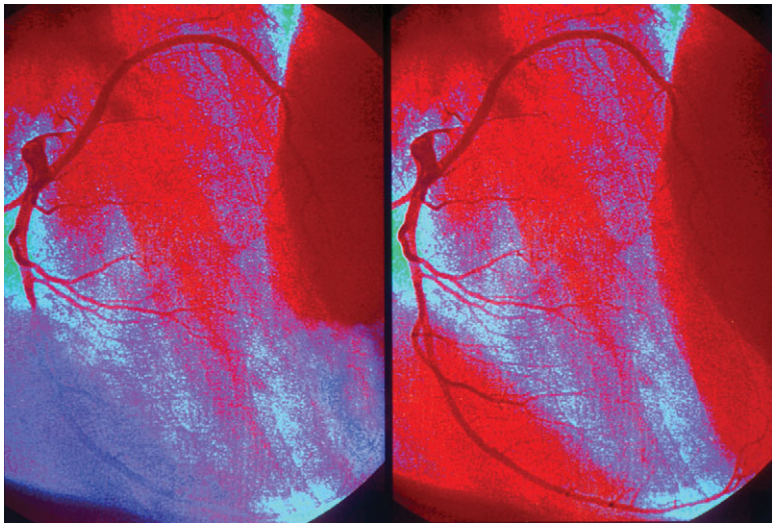

Coronary angiography of a STEMI patient with a mid-LAD lesion, before and after reperfusion.

On examination, she is afebrile, with a blood pressure of 137/63 mm Hg, a heart rate of 60 bpm, and a respiratory rate of 16 breaths/min. Her oxygen saturation is 100% on 3 L/min of oxygen via nasal cannula. No jugular vein distension is evident. She is pale and diaphoretic with cold, clammy extremities. The cardiac rhythm is regular, with normal S1 and S2 sounds, and no murmur or gallop is appreciable. A chest x-ray is obtained and is negative. The ECG obtained on her arrival (Figure 1) is interpreted as showing normal sinus rhythm at 66 bpm, right bundle branch block, and an acute anteroseptal MI. A baseline ECG from 6 months earlier (Figure 2) demonstrates that the right bundle branch block is new.

The catheterization laboratory is activated immediately while the patient receives 325 mg aspirin, 600 mg clopidogrel, and a 5000-unit heparin bolus.

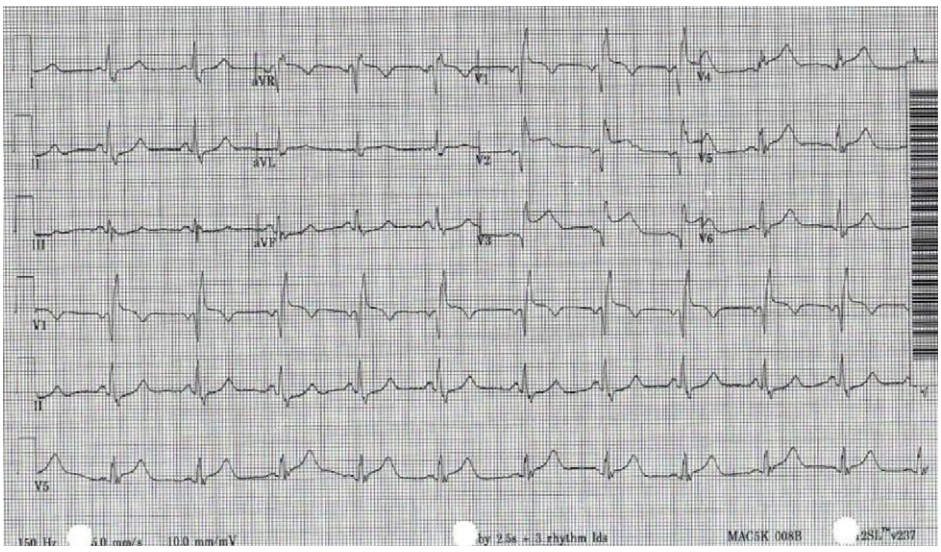

FIGURE 1. ECG on Arrival

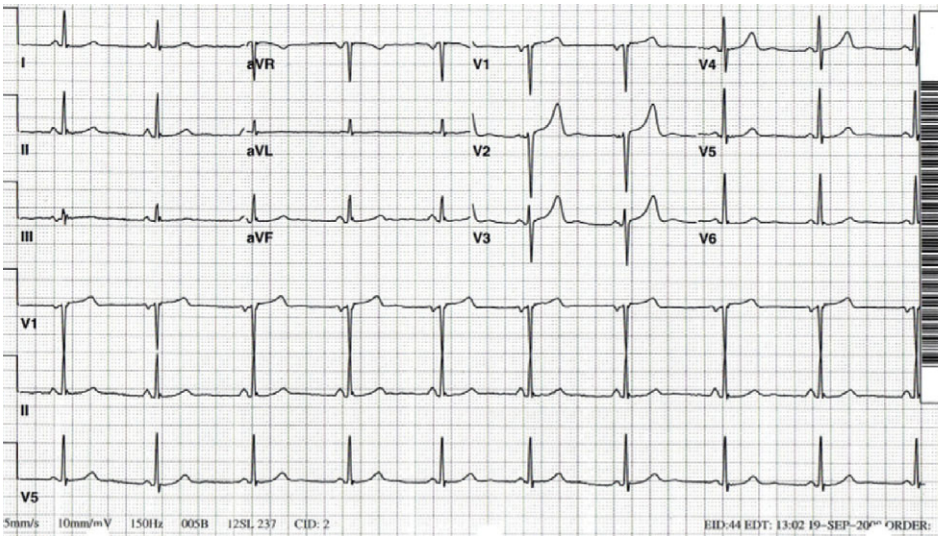

FIGURE 2. Baseline ECG From 6 Months Earlier

Laboratory tests show nothing of note in her complete blood count or chemistry panel. Her cardiac troponin level is 0.034 µg/L (reference range, 0-0.034). In the lab, she develops respiratory distress and hypertension and is intubated for acute pulmonary edema. Catheterization reveals 100% occlusion of the proximal left anterior descending (LAD) artery and severely reduced left ventricular function. A bare metal stent is successfully placed.

Afterward in the coronary care unit, the patient requires treatment for cardiogenic shock with dopamine and dobutamine. The relevant cardiac markers by now have risen sharply out of normal range, peaking at 594 µg/L for troponin and 16,247 U/L for creatine kinase. Once stabilized, she is extubated on hospital day 4. The remainder of her hospital course is uneventful.

The ECG done after return from the catheterization laboratory (Figure 3) demonstrates resolution of the RBBB, along with improvement in the degree of ST-segment elevation in the precordial leads, consistent with reperfusion. Persistent Q waves and T-wave inversions (both typical of STEMI evolution) are also seen.

DISCUSSION

Normal conduction proceeds from the atrioventricular node to the bundle of His and then to the left and right bundle branches, which run from the base to the apex of the heart on either side of the ventricular septum. Activation of the bundle branches, and therefore of the left and right ventricles, occurs nearly simultaneously, and thus the normal QRS complexes and ST segments represent combined left and right ventricular activity. Because the mass of the left ventricle is much greater, however, under normal circumstances its activity overshadows that of the superimposed right ventricle.

In the case of left bundle branch block, depolarization of the right ventricle occurs normally, but left ventricular conduction occurs via slow cell-to-cell conduction from right to left. Normally, depolarization of the interventricular septum is activated from left

to right, thus causing small "septal Q waves" in leads I and aVL. Since the septum is activated from the right side in left bundle branch block, the absence of these septal Q waves is a hallmark of left bundle branch block. The entire left ventricle is activated in a leftward-moving wave of slow cell-to-cell conduction, causing the broad monophasic positive deflection in leads I and aVL that is also typical.

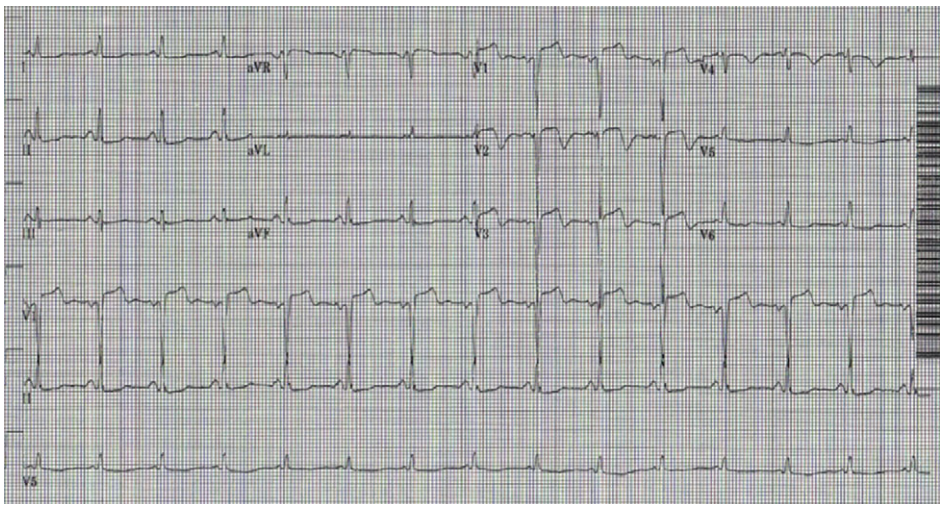

FIGURE 3. ECG After Catheterization and Stent Placement

Because the activation sequence of the left ventricle is markedly abnormal, Q waves cannot reliably be detected in the setting of left bundle branch block. Similarly, repolarization, represented by the ST segments and T waves, follows the same abnormal sequence, and thus ST-segment changes cannot reliably indicate acute ischemia or injury. While criteria for the detection of acute MI in the presence of left bundle branch block have been described, they are neither sensitive nor specific and for all practical purposes the ECG should be considered unreadable with regard to the presence of STEMI in the setting of left bundle branch block.

Left bundle branch block usually indicates some form of underlying hypertensive, ischemic, or degenerative heart disease and in a general population is associated with a substantially increased long-term mortality risk.

In contrast, left ventricular activation is normal in a patient with right bundle branch block. There is normal left-to-right septal depolarization, and then rapid depolarization of the left ventricle in the usual sequence. The first 80 ms or so of the QRS complex therefore appear nearly normal. After completion of left ventricular depolarization in the first 80 ms, however, the unopposed slow right ventricular activation is visible and is manifested in the typical findings of right bundle branch block: a broad S wave in I and aVL and an R-prime wave in the right precordial leads. Similarly, repolarization of the left ventricle is normal and thus ST-segment changes should occur as they do normally. The electrocardiographic evidence of STEMI therefore should not be obscured by the presence of right bundle branch block.

While right bundle branch block may also occur in the setting of structural heart disease (or pulmonary disease), it can occur in normal individuals, and is therefore more common than left bundle branch block. In the absence of underlying disease, it is not associated with an adverse prognosis.

INNOCENT OR OMINOUS?

Any discussion of prognosis and bundle branch block in the setting of acute MI must distinguish between a preexisting bundle branch block that is a "bystander" in the MI and a new bundle branch block caused by the infarction itself. If either left or right bundle branch block is preexisting, then it is prognostically relevant only to the extent that it is a marker for comorbidities.

Although a baseline was available in the case presented here, in practice, whether or not bundle branch block is truly new often cannot be immediately determined. If new right or left bundle branch block does occur in the setting of acute MI, it most commonly reflects septal injury and therefore is a marker of large infarctions, generally involving the proximal LAD artery. In the case of right bundle branch block, the ST-segment eleva-

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tions would be expected to be in the anteroseptal distribution (V1-V4); in left bundle branch block, the ST-segment elevations cannot be meaningfully localized. Whether a patient with a septal infarction develops left bundle branch block, right bundle branch block, complete heart block, or no conduction disturbance at all depends on the particular anatomy of the individual patient's conduction system and the size of the infarction. The left bundle branch divides early into anterior and posterior fascicles, while the right bundle is more compact and thus more susceptible to complete disruption. Right bundle branch block is therefore a more common sequela of anteroseptal MI.[1]

Since the advent of thrombolytic agents, several studies have demonstrated that right bundle branch block in the presence of anteroseptal STEMI is associated with a very high 30-day mortality risk, on the order of 30%. This represents a three- to four-fold risk increase when compared with STEMI in patients with normal conduction.[1-4]

Melgarejo-Moreno and colleagues demonstrated that new onset of right bundle branch block in the setting of STEMI is associated with large LAD-distribution infarctions, increased mortality, and an increased incidence of in-hospital complications such as heart failure, atrial fibrillation, complete heart block, and ventricular fibrillation.[1] Wong and colleagues confirmed this mortality risk and demonstrated a relationship between QRS duration and mortality risk in right bundle branch block, presumably because the degree of prolongation relates to the completeness of the block and thus the size of septal injury.[2,3] Kleemann and colleagues demonstrated that right bundle branch block was associated with increased hospital and long-term mortality in STEMI but not independently associated with a worse outcome in NSTEMI.[4]

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Each of these studies supports the concept that in the setting of anteroseptal STEMI, right bundle branch block often portends a high-risk proximal LAD occlusion, whereas in other distributions or settings, including NSTEMI, it is more likely to be an incidental finding that reflects chronic comorbidities rather than an inherently high-risk coronary insult.[4]

PRACTICAL APPROACH

High-risk proximal LAD lesions as seen with new right or left bundle branch block signify the likelihood of cardiogenic shock and death if perfusion is not quickly reestablished. Recognition of such a lesion should therefore lead to either immediate percutaneous coronary intervention (PCI) or contingency plans for rescue PCI (and other supportive measures) in case thrombolysis is given and fails.

The presence of left bundle branch block precludes electrocardiographic detection or localization of STEMI or ischemia, making the diagnosis of STEMI dependent on other criteria. If a patient has a left bundle branch block that is known to be new, then a large septal infarct, and therefore a high-risk, proximal LAD lesion, should be suspected. Of note, such a patient would be expected to have ongoing chest pain or equivalent symptoms consistent with a large MI. If the patient is pain-free and comfortable, then it is unlikely that the presence of left bundle branch block represents a major transmural infarction.

Since right bundle branch block does not alter the appearance of Q waves or ischemic ST-segment changes, concurrent anterior ST-segment elevations should raise suspicion for a high-risk proximal LAD lesion, especially if the right bundle branch block is known to be new. However, if ST-segment elevations are noted in an inferior or lateral distribution, the right bundle branch block is likely to be preexisting, unrelated to the infarction, and insignificant to patient management. □

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