Bradycardia in a Man With Acute Chest Pain

July 01, 2006
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A 56-year-old man presents with substernal chest pain, diaphoresis, and weakness of 1 hour's duration. He had taken a sublingual nitroglycerin tablet that had been prescribed for his wife. Afterwards, he did not notice any change in the chest discomfort but did note increased weakness. The patient has hypertension and diabetes mellitus; his medications include metoprolol and metformin.

Blood pressure is 80/60 mm Hg; heart rate, 50 beats per minute; and respiration rate, 26 breaths per minute. Oxygen saturation is normal, and the lung fields are clear. An ECG rhythm strip in lead II (A) shows a regular narrow QRS-complex rhythm; in addition, in this rhythm strip, the ST segment is elevated with a concave morphology. The patient is given a normal saline bolus and 1 mg of atropine intravenously; he is also given a single 325-mg oral aspirin. His blood pressure and pulse improve, yet his chest pain continues. A 12-lead ECG (B) shows significant ST-segment changes and sinus bradycardia with first-degree atrioventricular (AV) block. Rapid consultation with an interventional cardiologist is requested. The patient suddenly experiences extreme dizziness, which is accompanied by the return of hypotension and bradycardia (C).

What is the prognostic significance of the findings on the 12-lead ECG?

WHAT THE ECG SHOWS

About 25% to 30% of patients with acute coronary syndromes (ACS) have bradydysrhythmias. These dysrhythmias include bradycardia (sinus, junctional, and idioventricular) and AV block (first-, second-, and third-degree [complete heart block]).\(^1\,^2\) Sinus bradycardia and complete heart block are the bradydysrhythmias most often seen in patients with ACS, particularly in those with inferior wall and inferior-variant ST-segment myocardial infarction (STEMI) presentations.\(^1\,^2\) These 2 rhythm disturbances—as well as a first-degree AV block—are seen in this patient: a compromising **sinus bradycardia** (Figure 1) is noted on presentation, and **complete heart block** with a ventricular rate of about 20 beats per minute (Figure 2) develops soon after his arrival at the hospital.
PATHOPHYSIOLOGY OF BRADYDYSRHYTHMIAS IN ACS
The basic lesion that generates these dysrhythmias is an abnormality of either impulse formation or impulse conduction. However, the underlying pathophysiology usually results from multiple factors, including:

- Reversible ischemic injury to the conduction system.
- Irreversible necrosis of the conduction system.
- Altered autonomic function (particularly heightened parasympathetic tone).
- Metabolic acidosis.
- Electrolyte disorder.
- Systemic hypoxia.
- Adverse effects resulting from cardioactive medications (eg, b-blockers and calcium channel blockers).

Sinus bradycardia. Sinus bradycardia (see Figure 1) is characterized by a ventricular rate of less than 60 beats per minute, regular rhythm, a narrow QRS complex, and an association between each P wave and a QRS complex; in addition, the P-wave morphology and the PR interval are normal and consistent.

In the setting of an ACS, either ischemic injury to the sino-atrial node and/or heightened parasympathetic tone is responsible for this bradyarrhythmia. Patients with right coronary artery-related infarctions (inferior, posterior, lateral, and right ventricular) are at risk for the development of sinus node dysfunction, manifested primarily by sinus bradycardia. In cases of sinus arrest, the resulting rhythm originates from either the AV node (which produces a junctional escape rhythm at rates less than 60 beats per minute, with a narrow QRS complex), or from the ventricle (which produces an idioventricular escape rhythm at rates less than 45 beats per minute, with a widened QRS complex).

Complete heart block. The most ominous dysrhythmia seen in this patient is third-degree AV block, also known as complete heart block. The term "third-degree AV block" describes a disturbance in the conduction of the electrical impulse in and around the AV node. In third-degree AV block, there is a complete lack of electrical communication between the atria and the ventricles--ie, the atria and ventricles work independently of one another. Consequently, the P wave is dissociated from the QRS complex. Both the atrial and ventricular electrical rhythms are most often regular; however, the atrial rate is greater than the ventricular rate. (Note that if the atrial rate were less than the ventricular rate, the patient would be experiencing AV dissociation.)

Third-degree AV block usually results from dysfunction in 1 of 3 areas of the cardiac conduction system:

- Within the AV node (above the His bundle).
- Within the His bundle (intra-Hisian).
- Beyond the His bundle or the bundle branches (infra-Hisian).3,4

"Proximal" third-degree AV block usually develops in patients with inferior wall STEMI--as a result of ischemic damage to the AV node and/or increased parasympathetic influence on the AV node. In contrast, patients with anterior wall infarctions suffer "distal" third- degree AV block as a result of dysfunction of the trifascicular intraventricular conduction system. The site of the escape rhythm is usually immediately below the level of the block. When the ventricular escape rhythm is located near the His bundle, the rate is greater than 40 beats per minute and QRS complexes tend to be narrow. When the site of escape is distal to the His bundle, the rate tends to be less than 40 beats per minute and QRS complexes tend to be wide. The second scenario is seen in this patient (see Figure 2).

PREDICTION OF BRADYDYSRHYTHMIAS
The ability to predict the development of bradyarrhythmias in patients with ACS is vital; prophylactic management can lessen associated morbidity and mortality. The likelihood of bradyarrhythmia development can be estimated and the important clinical features of an anticipated rhythm disturbance predicted based on the anatomic location and size of the infarction. The clinical variables that can be predicted include:

- Expected time course for the dysrhythmia.
- Potential for hemodynamic instability.
- Degree of response to resuscitative therapy.
Predicting risk and prognosis. Acute inferior and inferior-variant STEMI\textsuperscript{s} are frequently complicated by bradyarrhythmias. As infarct size increases, the risks of arrhythmia, left ventricular dysfunction, and death increase proportionally.\textsuperscript{5,6} Patients with an inferior wall STEMI accompanied by right ventricular infarction have a markedly worse prognosis--with respect to both acute cardiovascular complications and death--than do patients with an isolated inferior wall STEMI.\textsuperscript{7}

What the 12-lead ECG shows. This patient's 12-lead ECG (Figure 3) reveals an inferior wall STEMI--and probably right ventricular infarction as well. Briefly, the ECG findings seen in right ventricular infarction include ST-segment elevation in the right precordial chest leads (particularly lead V\textsubscript{1}), in additional ECG lead RV\textsubscript{4}, and in the inferior leads (II, III, and aVF). A finding of ST-segment elevation that is greater in lead III than in the other inferior leads in the setting of inferior STEMI also suggests right ventricular myocardial infarction.\textsuperscript{8} In this man's ECG, the ST-segment elevation is more pronounced in lead III than in the other inferior leads, and there is ST-segment elevation in lead V\textsubscript{1} as well. This large inferior-variant STEMI carries a high risk of acute cardiovascular complications.

Calculating the risk of complete heart block. A useful method for predicting the development of third-degree AV block in any patient with a STEMI is based on ECG evidence of the following risk factors:

- AV block (first-degree or second-degree [type I or type II]).
- Bundle-branch block (right or left).
- Hemiblock (left anterior or left posterior).

Each ECG risk factor counts for 1 point. The risk of third-degree AV block is calculated based on the total score. A score of zero carries a 1.2% risk; scores of 1, 2, and 3 carry risks of 7.8%, 25.0%, and 36.4%, respectively. (This patient had 1 ECG "lesion" in his conduction system--first-degree AV block--which made his risk for the development of third-degree AV block 7.8%.)

Predicting clinical features of a dysrhythmia. Compromising rhythm disturbances (including second- and third-degree AV blocks) in patients with acute inferior and inferior-variant STEMI\textsuperscript{s} can begin abruptly within 6 hours of the onset of infarction; they usually have relatively slow ventricular escape rates and respond readily to atropine therapy. These "early" bradydysrhythmias usually result from heightened parasympathetic tone.

Patients in whom bradyarrhythmias develop more than 6 hours after the onset of an inferior STEMI have reversible ischemia of the conduction system. The block progresses gradually, and the return to sinus rhythm is equally slow. The escape rhythm is usually of ventricular origin, has a relatively fast rate, and responds poorly to medical therapy. Necrosis of the AV node is rare because of the presence of both extensive collateral circulation and a low metabolic rate with high glycogen reserves.

OUTCOME OF THIS CASE

The third-degree heart block returned to sinus bradycardia--and systemic perfusion improved--a short time after administration of an additional fluid bolus and 1 mg of atropine. Transcutaneous pacing pads were applied; however, the unit was not activated. The patient was quickly transferred to the cardiac catheterization laboratory for percutaneous coronary intervention and placement of a transcutaneous pacing wire.

The catheterization revealed inferior and right ventricular hypokinesis, as well as single-vessel occlusion of the proximal right coronary artery; this occlusion was successfully opened with an intracoronary stent. Serum marker analysis confirmed the diagnosis of acute myocardial infarction, and the patient was admitted to the coronary care unit. No further dysrhythmia was noted. He was discharged on hospital day 4 and was well at follow-up.

References: REFERENCES:

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